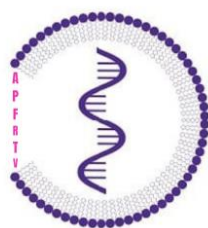




APFRTV 2026



ASIA-PACIFIC FORUM ON RNA THERAPEUTICS AND VACCINES

THEME:

"ADVANCING RNA SCIENCE FROM BENCH TO POPULATION IMPACT"

VISIT US: WWW.ONEHEALTH-IN.ORG/APFRTV-CONFERENCE-2

DATE:

25 SEPTEMBER 2026

VENUE:

**CS- AUDITORIUM,
FACULTY OF ENGINEERING & TECHNOLOGY,
SOUTH ASIAN UNIVERSITY,
RAJPUR ROAD, MAIDAN GARHI
NEW DELHI**

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Welcome to

APFRTV 2026

(Asia-Pacific Forum on RNA Therapeutics and Vaccines)

organized by

One Health India Organization, New Delhi

in collaboration with

FLSB, South Asian University, New Delhi

Theme:

“Advancing RNA Science from Bench to Population Impact”

Scheduled on:

September 25, 2026

At:

CS- Auditorium,

**Faculty of Engineering & Technology, South Asian University,
Rajpur Road, Maidan Garhi, New Delhi**

A one-day conference consisting of a scientific program, comprehensive talks, special sessions, oral & poster presentations of peer-reviewed contributed papers and exciting and innovative research products.

For In Person Participants:

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- Conference kit including Nametag, Program & Abstract booklet
- Coffee breaks and lunch for all
- Certificate accreditation from Organizing Committee

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Welcome to the Premier Gathering of RNA Therapeutics and Vaccines Experts in the Asia-Pacific Region!

We are delighted to welcome you to the Asia-Pacific Forum on RNA Therapeutics and Vaccines (APFRTV), taking place in the vibrant city of New Delhi, India. This premier event brings together leading experts, researchers, and industry professionals from around the world to share knowledge, foster collaboration, and drive innovation in the field of RNA therapeutics and vaccines.

Our Mission:

The APFRTV aims to accelerate the development and deployment of RNA-based therapies and vaccines, addressing some of the most pressing health challenges of our time. Through this forum, we strive to create an inclusive platform for the exchange of groundbreaking research, regulatory updates, and technological advancements.

What to Expect:

- **Engaging Keynotes:** Hear from renowned leaders and visionaries in RNA research and development.
- **Interactive Panels:** Participate in discussions addressing the latest trends, challenges, and opportunities in the industry.
- **Networking Opportunities:** Connect with over 250 professionals, including C-Suite executives, VPs, Directors, and Heads of RNA, Vaccine, CMC, and Drug Delivery.
- **Cutting-Edge Research:** Explore the latest data and insights from top-tier biopharmaceutical companies and research institutions.
- **Exhibition and Sponsorship:** Discover innovative solutions and services from our esteemed exhibitors and sponsors.

Special Thanks:

We extend our gratitude to our speakers, sponsors, exhibitors, and attendees for their invaluable contributions and support. Your participation is key to the success of this forum and to the advancement of RNA therapeutics and vaccines.

MESSAGE FROM THE ORGANIZING COMMITTEE

It is our great pleasure to welcome you to the Asia-Pacific Forum on RNA Therapeutics and Vaccines (APFRTV 2026) at South Asian University, New Delhi.

With this year's theme, "Advancing RNA Science from Bench to Population Impact," APFRTV 2026 brings together leading scientists, clinicians, researchers, academicians, industry experts, and young researchers to explore the rapidly evolving landscape of RNA biology, therapeutics, vaccines, and precision medicine.

The forum provides a platform for scientific exchange, innovation, collaboration, and translation, featuring keynote lectures, expert sessions, young researcher presentations, posters, exhibitions, and a panel discussion on the future of RNA-based vaccines and therapeutics in India.

Beyond science, the programme also embraces health awareness and community engagement through the BBIF Cancer Care Awareness Walkathon and Run Competition.

We sincerely thank all our speakers, delegates, partners, committees, volunteers, and supporters for joining us in advancing RNA science and fostering a shared vision for innovation, collaboration, and impact.

Welcome to APFRTV 2026 — where science moves from the bench towards population impact.

Organizing Committee
APFRTV 2026



Dr. Birendra Kumar Yadav
(BBIF)



Dr. Yuba Raj Pokheral
(SAU)



Dr. Sonam Dhamija
(SAU)

Scientific Program

25th September, 2026 (Friday)

Theme: “Advancing RNA Science from Bench to Population Impact”

6:00-07.00	BBIFCC CANCER AWARENESS WALKATHON	Organized by BBIF Cance Care & South Asian University
7:00-07.30	200 METER RUN COMPETITION	
8.30-09.30	Registration	
09:30-09:35	Welcome address, about the conference	Dr. Birendra Kr. Yadav, BBIFCC (Convener and Chief Organizing Secretary) Dr Yuba Raj Pokheral, SAU (Co-convener and Organizing Secretary) Dr. Sonam Dhamija (Convenor)
09:35-09:40	lamp lightening	Dr. Birendra Kr. Yadav, BBIFCC (Convener and Chief Organizing Secretary) Dr Yuba Raj Pokharel, SAU (Co-convener and Organizing Secretary) Dr. Sonam Dhamija (Convenor) Prof. Rinkoo Devi Gupta, Dean, FLSB, SAU Prof. Ritu Gaur, Vice President, SAU Prof. Kapil Sharma, Vice President, SAU Prof. Sudhanshu Vratl, Former Executive Director at Regional Centre for Biotechnology Prof. (Dr) K.K. Pandey, Ex. Sr. Prof. & HOD FoAy, IMS BHU, BBIFCC Fellow & Advisor

09:40-09:50	Inaugural Address	Prof. Kapil Sharma, Vice President, SAU
	Address by Guest of Honor	Prof. Ritu Gaur, Vice President, SAU
	Address by Guest of Honor	Prof. Rinkoo Devi Gupta, Dean, FLSB, SAU
09:50-10:00	Vote of thanks and abstract book release	Prof. (Dr) K.K. Pandey, Ex. Sr. Prof. & HOD FoAy, IMS BHU, BBIFCC Fellow & Advisor
Session	Special Talk & Keynote	Chairpersons : Prof. (Dr) K.K. Pandey Dr. Birendra Kr. Yadav
10:00–10:25	From Bench to Public Health: What Can We Learn from India's Vaccine Development Journey?	Prof. Sudhanshu Vrat, Former Executive Director at Regional Centre for Biotechnology
10:25–10:45	<i>CpG Depletion in RNA Viruses: A Strategy to Hide in Plain Sight</i>	Prof. Vivekanandan Perumal, IIT Delhi
10:45–11:15 ☕ Tea & Networking Break		
SESSION I: 11:15–12:00	<i>RNA Biology, Infection & Immunity</i>	Chairpersons: Prof. Sudhanshu Vrat Dr. Bhupendra Kumar Verma
11:15-11:35	Reading and Rewriting the Functional Code of RNA: From Structure to Mechanism	Dr. Niyati Jain Ashoka University
11:35-11:55	Rapid and Sensitive RNA-Based Detection of Measles and Rubella Using LAMP-CRISPR/Cas Technology: A Novel Diagnostic Approach	Dr. Yuba Raj Pokharel, Prof. South Asian University
12:00-12:20	Unravelling the Role of tRNA Fragments in Dengue Virus Pathogenesis	Dr. Bhupendra Kumar Verma AIIMS, New Delhi
SESSION 2: 12:20–13:10	RNA Biology, Infection & Immunity	Dr. Yuba Raj Pokharel Dr. Ajay K Yadav
12:20-12:40	Revolutionizing Pandemic Response: Harnessing the Power of mRNA Technology to Achieve the 100-Day Mission for Vaccine Development	Dr. Arun Kumar Coalition for Epidemic Preparedness Innovations (CEPI), Oslo, Norway

12:40-13:00	Synergistic Silencing of Influenza A Virus Matrix Protein Expression Using a Dual-Function siRNA–Ribozyme Chimeric RNA	Dr. Madhu Khanna VPCI, University of Delhi, India
13:00-13:20	Role of Ayurveda in RNA Science	Prof. (Vd.) Parul Anil Kumar Joshi, MRIAS/Parul University , Gujarat
13:20-14:20	Lunch, Poster Presentation & Exhibition	Poster Viewing • Poster Judging • Networking • Exhibition
SESSION 3: 14:20–15:30 RNA Therapeutics & Precision Medicine		Chairpersons: Dr. Mukesh Nandave Prof. Rinkoo Devi Gupta,
14:20-14:40	Targeting Deubiquitinating Enzymes with siRNA to Decipher Glioma Resistance	Dr. Ajay K. Yadav ACBR, University of Delhi
14:40 –15:00	RNA Therapeutics in Rheumatology	Dr. Niket Verma AIIMS-CAPFIMS, New Delhi
15:00-15:20	Graph-Theoretic and Petri Net Based Modeling of RNA–Protein Interaction Networks	Dr. Gajendra Singh, JNU
15:20-15:40	RNA-Binding Protein HuR Drives Glycolytic Reprogramming in Triple-Negative Breast Cancer: A Novel Therapeutic Avenue	Dr. Chandra Prakash Prasad , AIIMS , New Delhi
15:40- 16:10	Young Researchers: Oral Presentations	Chairpersons: Dr. Kavita Arrora Dr. Sonam Dhamija
15:40-15:48	Dengue Virus-Induced lncRNA Landscape Reveals Molecular Signatures of Impaired Megakaryopoiesis	Preeti Sohkhla, AIIMS, New Delhi
15:48-15:56	Inhibition of HepG2, DU-145, and MCF-7 Cell Lines by Thiosemicarbazone Derivatives (A3, A5, and A8)	Amit Sarder, FLSB, SAU
15:56-16:04	Differential tRNA Fragmentation During Dengue Virus Infection: functional investigation of tRFs that modulate DENV replication and PI3K/Akt signaling	Sushant Phadnis, AIIMS, New Delhi

16:10-17:00	Panel Discussion: The Future of RNA-Based Vaccines & Therapeutics in India	Moderator Dr. Yuba Raj Pokharel, Prof. South Asian University Panelist: Dr. Ajay Yadav, ACBR Dr. Madhu Khanna, VPCI Dr. Arun Kumar, CEPI, Norway Dr. Gajendra Singh, JNU Dr. Bhupendra Kumar Verma
17:00–17:30	★ VALEDICTORY FUNCTION & AWARDS	Award, Reel competition award • Vote of Thanks
17:30	BBIF CANCER CARE: RANG-E-ZINDAGI <i>An Evening of Culture, Hope & Cancer Awareness</i> Celebrating Life • Spreading Awareness • Inspiring Hope	Open to All

APFRTV 2026 Abstracts



Title: From bench to public health: What can we learn from India's rotavirus vaccine development journey

Author: Dr. Sudhanshu Vrati, Regional Centre for Biotechnology, Faridabad

Co Author: N/A

Abstract ID: APFRTV 2026 - 20

Abstract Body: Rotavirus is the leading cause of diarrhoea-associated hospitalisations and deaths in developing countries, estimated to account for approximately 610,000 deaths annually. It is a particularly important problem in India, where nearly one-fourth of these deaths occur and it is estimated that about 1 child in 250 in India will die of this disease. Accordingly, development of vaccines for rotavirus infections has been accorded high priority

As part of the Indo-US Vaccine Action Program, a Department of Biotechnology (DBT), Government of India sponsored activity to promote new vaccine development, two rotavirus strains were identified that demonstrated interesting properties as potential candidate vaccines. These strains were obtained from outbreaks of asymptotically infected newborns in Delhi (116E) and Bangalore (I321). Having undergone phase I and II, safety and immunogenicity studies, a randomized, double-blind, placebo-controlled, multicentre trial of the 116E vaccine was conducted at three sites in Delhi (urban), Pune (rural), and Vellore (urban and rural) that enrolled 6799 infants aged 6-7 weeks. The incidence of severe rotavirus gastroenteritis per 100 person-years was 1·5 in the vaccine group and 3·2 in the placebo group.

Prevalence of immediate, solicited, and serious adverse events was similar in both groups. These studies demonstrated that the monovalent human-bovine (116E) rotavirus vaccine was effective and well tolerated in Indian infants. Having established its safety and efficacy, the 116E rotavirus vaccine 'Rotavac' produced by BBIL, Hyderabad was launched for commercial use. The vaccine has since been prequalified by the World Health Organization.

The development of this vaccine serves as a profound demonstration of the synergy between precise clinical observation and rigorous laboratory science. This milestone underscores the critical role of international collaboration and cross-disciplinary teamwork in accelerating translation from bench to bedside, ultimately establishing a new paradigm for rapid, evidence-based public health interventions. Crucially, this rapid advancement was made possible by the robust financial backing and strategic support of national and international organizations, highlighting that sustained public and private funding is indispensable for mobilizing global scientific infrastructure during public health crises.



Title: CpG depletion in RNA viruses: a strategy to hide in plain sight.

Author: Prof. Vivekanandan Perumal, IIT, Delhi

Co Author: N/A

Abstract ID: APFRTV 2026 - 21

Abstract Body: The depletion of CpG dinucleotides is observed universally in most RNA viruses infecting vertebrates, but not in those infecting invertebrates, suggesting selective pressures in vertebrate hosts acting against CpG motifs in this group of viruses.

The zinc-finger antiviral protein (ZAP) encoded by vertebrate hosts binds and recruits cofactors that degrade CpG-rich viral RNA.

The CpG dinucleotide content of HIV-1 may help predict long-term disease outcomes at early stages of infection. Over the past decade, numerous studies have focused on recoding virus genomes to increase their CpG content without altering their amino acid sequences.

These recoded viruses have been linked to lower virus replication and reduced pathogenesis without compromising the host immune responses elicited in vertebrate hosts, thus providing a novel platform technology for live attenuated vaccines



Title: Reading and Rewriting the Functional Code of RNA: From Structure to Mechanism

Author: Dr. Niyati Jain, Ashoka University

Co Author: N/A

Abstract ID: APFRTV 2026 - 22

Abstract Body: Hepatitis C virus (HCV) is a major global health burden, associated with chronic liver diseases, including cirrhosis and hepatocellular carcinoma. Viral replication critically depends on conserved cis-acting replication elements (CREs), such as the 5BSL3.2 stem-loop near the 3' end of the open reading frame. This element forms a long-range kissing-loop interaction with the SL2 domain of the 3'X tail, essential for efficient genome replication.

However, the role of host RNA-binding proteins (RBPs) in regulating this RNA-RNA interaction remains poorly understood. To explore this, we investigated whether the host RBP hnRNPA1 modulates HCV replication by targeting the 5BSL3.2 element.

Using an integrated approach combining structural biology, biophysics, and biochemical assays, we identify the terminal loop of 5BSL3.2 as a high-affinity binding site for the tandem RNA recognition motifs (RRMs) of hnRNPA1.

Our data reveal that adenine-rich residues within the loop are critical for binding specificity. Our results uncover a structural mechanism by which hnRNPA1 binding perturbs the kissing-loop interaction between 5BSL3.2 and the SL2 element of the viral 3'X-tail, which impacts viral replication. This study highlights a previously unrecognized role of hnRNPA1 in modulating viral RNA structure and suggests a novel interface for host-directed antiviral intervention.



Title: Unravelling the Role of tRNA Fragments in Dengue Virus Pathogenesis

Author: Dr. Bhupendra Kumar Verma, AIIMS, New Delhi

Co Author: N/A

Abstract ID: APFRTV 2026 - 23

Abstract Body: Dengue virus infection is a major global health concern, with DENV pathophysiology heavily reliant on host cellular machinery for its replication. Currently, no effective treatment exists for DENV infection, making it crucial to understand the molecular basis of its pathogenesis. Dengue is an arboviral disease that presents a wide range of therapeutic outcomes, from mild to life-threatening conditions. The absence of effective treatments underscores the need for a deeper understanding of DENV pathogenesis, along with the development of reliable biomarkers for rapid detection, severity assessment, and prognostic measures. tRNA-derived small RNAs (tsRNAs), is an emerging class of sncRNAs which play an important role in regulating transcription and translation machinery thus holding the immense diagnostic and therapeutic potential during DENV infection.

In our study, we demonstrated that during DENV infection, RNase levels are modulated, with angiogenin significantly upregulated as infection progresses. RNA sequencing revealed increased tRNA fragmentation with increase in DENV pathogenesis. Analysis indicated modulation of specific tsRNA subtypes: 3'tRFs were upregulated, while i-tRFs were downregulated. Notably, transfection with antisense oligonucleotides (ASO) targeting the upregulated i-tRF-40, derived from tRNA-Tyr-GTA, resulted in over 70% reduction in DENV infection. PI3K-Akt pathway was found in the top list from KEGG pathway analysis of RNA seq data. Akt activation regulates various critical functions involved in cellular maintenance. We therefore observed the effects of ASOs on Akt modulation and thus in regulating DENV infection. i-tRF 40 TyrGTA and i-tRF 25 LysCTT were found to show pro-viral property that facilitates Akt activation during DENV infection.

While i-tRF 28 LysCTT was found to act as a negative regulator of Akt phosphorylation, which showed anti-viral properties. This study aims to give insights about the potential regulatory roles of tRFs in DENV pathophysiology, contributing to the better understanding about host-virus interaction.

References:

Madhry, D., Kumari, K., Meena, V., Roy, R., & Verma, B. (2024). Unravelling tRNA fragments in DENV pathogenesis: Insights from RNA sequencing. *Scientific Reports*, 14(1), 1–10.

Madhry, D., Roy, R., & Verma, B. (2024). Biotin-Based Northern Blotting (BiNoB): A Cost-Efficient Alternative for Detection of Small RNAs. *Current Protocols*, 4(12), 1–15.

Madhry, D., Malvankar, S., Phadnis, S., Srivastava, R. K., Bhattacharyya, S., & Verma, B. (2023). Synergistic correlation between host angiogenin and dengue virus replication. *RNA Biology*, 20(1), 805–816.



Title: Role of Ayurveda in RNA Science

Author: Prof. (Vd.) Parul Anil Kumar Joshi, MRIAS/Parul University, Gujarat

Co Author: N/A

Category: RNA Therapeutics (siRNA, miRNA, ASO, mRNA)

Abstract ID: APFRTV 2026 - 24

Abstract Body: Recent advances in RNA-based therapeutics have transformed modern medicine through technologies such as messenger RNA (mRNA), self-amplifying RNA (saRNA), circular RNA (circRNA), and RNA interference (RNAi), enabling novel approaches to disease prevention and treatment. At the same time, increasing concerns regarding congenital disorders, infertility, and declining overall health have renewed interest in traditional systems of medicine. Ayurveda, the ancient Indian system of medicine, emphasizes preventive healthcare, reproductive health, and the promotion of healthy progeny through comprehensive lifestyle, dietary, and therapeutic interventions. This paper explores the conceptual relevance of Ayurvedic principles in the context of contemporary RNA science.

Materials and Methods: A narrative review of classical Ayurvedic literature, including the major Samhitas, was undertaken alongside an examination of current scientific literature on RNA-based technologies. Conceptual comparisons were made between Ayurvedic principles of health promotion, maternal and paternal preconception care, Beeja (hereditary factors), Garbha (embryonic development), and Rasayana (rejuvenation), and emerging knowledge in molecular genetics and RNA therapeutics.

Observations: RNA-based technologies have demonstrated significant potential in vaccine development, cancer immunotherapy, protein replacement, and the management of selected genetic disorders. Simultaneously, Ayurveda provides a holistic framework for optimizing reproductive health, strengthening physiological resilience, and promoting healthy foetal implantation and development through individualized preventive strategies. Although Ayurvedic concepts do not describe RNA mechanisms in modern molecular terms, they offer a preventive healthcare model that may complement ongoing research in genomics, epigenetics, and precision medicine.

Conclusion: Modern RNA therapeutics represent a major advancement in the treatment of infectious, genetic, and neoplastic diseases but remain disease-specific and resource-intensive. Ayurveda offers a complementary, preventive approach focused on improving overall health, reproductive wellness, and the quality of future generations through evidence-informed lifestyle and healthcare practices.

Further interdisciplinary research integrating Ayurvedic concepts with contemporary molecular biology, genomics, and RNA science may provide valuable insights into preventive and personalized cost-effective healthcare while maintaining rigorous scientific validation.

Key words: RNA science, Genomics, Indian System of Medicine Ayurveda, Healthy progeny.



Title: Targeting Deubiquitinating Enzymes with siRNA to Decipher Glioma Resistance

Author: Dr. Ajay K. Yadav, ACBR, University of Delhi

Co Author: N/A

Abstract ID: APFRTV 2026 - 25

Abstract Body: Glioblastoma (GBM) treatment is limited by resistance to Temozolomide (TMZ), the standard alkylating chemotherapy. TMZ kills tumor cells by alkylating DNA, but resistant cells reactivate MGMT, an enzyme that reverses this damage. Normally, spent MGMT is degraded via ubiquitination and proteasomal turnover; however, deubiquitinating enzymes (DUBs) can reverse this tag, stabilizing MGMT and sustaining resistance.

Using siRNA-mediated knockdown as a functional probe, this analysis shows that TMZ-resistant U87MG and LN229 glioma cells markedly upregulate DUBs (USP5, USP8, USP10, TNFAIP3), and that silencing USP5 or USP8 alone (rather than requiring co-knockdown, as in naïve cells) is sufficient to induce 95–98% apoptosis. USP5 was found to directly stabilize MGMT through the proteasomal pathway, while USP8 sustains MGMT via an independent mechanism.

This USP5–MGMT relationship was further validated at both transcript and protein levels in patient-derived Grade IV GBM tumor tissue, and confirmed functionally through USP5 overexpression and proteasome-inhibitor rescue experiments. Together, these findings identify USP5 as a central node conferring TMZ resistance and a promising therapeutic target.

Beyond its use as a research tool, siRNA itself holds direct therapeutic promise: packaging USP5/USP8-targeting siRNA into lipid nanoparticles (liposomes) or exosomes could enable targeted, stable delivery across the blood–brain barrier, offering a translational route from this mechanistic knockdown data toward clinical DUB-directed gene-silencing therapy in resistant glioma



Title: RNA Therapeutics in Rheumatology

Author: Dr. Niket Verma, AIIMS-CAPFIMS, New Delhi

Co Author: N/A

Abstract ID: APFRTV 2026 - 26

Abstract Body: Background - Immune-mediated rheumatological diseases are complex disorders involving multiple and often interconnected pathogenic mechanisms, with a chronic disease course and considerable variability in clinical manifestations and treatment response. Among these, rheumatoid arthritis (RA) and systemic lupus erythematosus (SLE) are two major systemic rheumatological diseases that impose a substantial burden on patients and healthcare systems.

Rheumatoid arthritis (RA) is a chronic systemic inflammatory disorder characterized by synovial inflammation and joint destruction. Its pathogenesis involves B-cell and CD4⁺ T-cell infiltration of the synovium, with synovitis driven by TNF- α , IL-1, and IL-6. Anti-citrullinated protein antibodies (ACPA) are a hallmark, and extra-articular involvement may affect the eyes, lungs, skin, heart, kidney, and vasculature. Treatment includes conventional therapy, DMARDs and biological agents.

Systemic lupus erythematosus (SLE) is a chronic systemic autoimmune disease characterized by loss of immune tolerance, autoantibody production, immune-complex formation, and multi-organ inflammation and tissue injury. Clinical manifestations include constitutional symptoms, fatigue, fever, arthralgia, photosensitivity, malar rash, oral ulcers, alopecia, and serositis.

Dysregulated innate and adaptive immunity, abnormal activation of B cells and CD4⁺ T cells, and prominent type I interferon (IFN-I) signalling contribute to its pathogenesis. Antinuclear antibodies (ANA), anti-dsDNA, and anti-Sm antibodies, with hypocomplementemia, are important serological features. Treatments include conventional therapy and targeted/advanced therapy.

Unmet Need and Rationale for miRNAs

Conventional therapies for rheumatological diseases include hydroxychloroquine, glucocorticoids, methotrexate, azathioprine, mycophenolate mofetil, cyclophosphamide, calcineurin inhibitors, and NSAIDs. Targeted and advanced therapies include belimumab, rituximab, and investigational CAR-T cell therapy. Long-term treatment is limited by non-response and toxicity, including gastrointestinal ulcers, perforation and bleeding; bone and metabolic complications, ocular, dermatologic, cardiovascular, and neuropsychiatric effects; increased infection and malignancy risk; reproductive and sexual health impairment; limited treatment monitoring; and lack of reliable biomarkers to assess treatment response. miRNAs may serve as minimally invasive biomarkers for disease activity and therapeutic response, creating a need for targeted, mechanism-based alternatives such as miRNA-based therapeutics.

miRNA Biology and Mechanism

MicroRNAs (miRNAs) are small, highly conserved, single-stranded non-coding RNA molecules, that act as regulators of gene expression at the post-transcriptional level. miRNAs regulate the expression of over 60% of all human protein-coding genes. A single miRNA can target hundreds of different mRNAs, while a single mRNA can be targeted by multiple distinct miRNAs. Perfect or near-perfect matching with the target mRNA can result in mRNA degradation/cleavage, whereas imperfect base pairing, results primarily in translational repression and mRNA destabilization. Circulating miRNAs are detected in serum, plasma, urine and synovial fluid, expanding their role beyond intracellular regulation. They are protected from RNases by extracellular vesicles or by binding to Argonaute proteins and lipoproteins. Extracellular miRNAs can be transferred to recipient cells and modulate gene expression. Their stability and accessibility in body fluids make them promising minimally invasive biomarkers of disease activity, organ involvement, and treatment response.

The delivery process for miRNA/siRNA involves three key stages: Stabilize, Transport and Optional targeting add-on) allows a targeting ligand (such as folate) to be added to the outer surface of the chosen nanocarrier for active targeting.

miRNAs in Autoimmune Pathogenesis and Immunology

Dysregulated miRNAs may contribute to autoimmune pathogenesis through apoptotic imbalance, chronic inflammation, autoantigen exposure, and tissue-specific inflammation. Dysregulated miRNAs can alter apoptotic pathways, promoting survival of autoreactive T and B cells. Loss of regulatory miRNAs such as miR-146a weakens negative feedback on NF- κ B/IFN signalling, contributing to persistent inflammation. Defective cell-death and clearance pathways may increase exposure of nuclear autoantigens and promote loss of tolerance. Dysregulated miRNAs also modulate adhesion molecules and chemokines, promoting immune-cell recruitment to target tissues such as the synovium in RA and kidneys in SLE.

Clinical Implications

Rheumatological diseases have heterogeneous and often unpredictable clinical courses. Despite this heterogeneity, several common pathological features, particularly systemic inflammation, are shared across diseases. MicroRNAs (miRNAs) may play an important role in regulating these shared inflammatory pathways. miRNAs (e.g., miR-155, miR-146a) tightly regulate toll-like receptor (TLR) signalling in macrophages and dendritic cells, preventing excessive acute inflammatory responses. miRNAs directly target the 3' UTRs of critical cytokine mRNAs (like TNF- α , IL-6, IFN- γ), acting as epigenetic brakes on potential cytokine storms.

The emerging diagnostic microRNAs in rheumatoid arthritis (RA) include miR-145-5p (found in plasma/synovium, upregulated, and aggravates cartilage erosions through a strong link to increased MMP activity); miR-1276 (found in mononuclear cells, upregulated, and acting as a novel biomarker that regulates extracellular matrix organization and immune activation); miR-186 (found in whole blood, downregulated, and highly predictive of acute RA exacerbation and flares when combined with ESR); and miR-155 (found in synovial fluid, upregulated, and serving as a key driver of TNF- α -mediated inflammation while promoting osteoclastogenesis).

The key dysregulated microRNAs in systemic lupus erythematosus (SLE) include miR-146a (which is downregulated, targets IRAK1 and TRAF6 while failing to suppress the IFN pathway, and shows a strong negative correlation with SLEDAI scores); miR-155 (which is upregulated, drives T-cell activation and pathogenic B-cell survival, and serves as a marker of overall systemic inflammatory burden); miR-21 (which is upregulated, regulates PDCD4 and alters T-cell/B-cell subset ratios, and positively correlates with anti-dsDNA titers); and miR-124 (which is downregulated, regulates inflammatory cytokine networks, and acts as a newly validated diagnostic marker in active SLE).

Therapeutic Modalities

miRNA mimics: Utilized when a protective, anti-inflammatory miRNA is downregulated in the disease state (e.g., miR-146a in SLE or miR-124 in RA). miRNA mimics are double-stranded RNA molecules that imitate endogenous mature miRNA and re-establish the physiological 'brakes' on the immune system.

AntagomiRs (inhibitors): Utilized when a pathogenic, pro-inflammatory miRNA is upregulated in the disease state (e.g., miR-155 in RA or miR-203 in LN). They are designed to complement and bind to the pathogenic miRNA, blocking its ability to bind to mRNA and remove the pathological suppression of beneficial genes. However, currently no miRNA based therapeutic modalities have received FDA approval for rheumatological disorders.

Looking Ahead: The Clinical Future of miRNA

Predictive liquid biopsies: Forecasting SLE/RA flares before clinical symptoms emerge.

Targeted delivery: Directing LNPs or exosomes straight to the synovium or glomerulus.

Precision prescribing: Using baseline miRNA profiles to predict patient response to specific biologics.

Network pharmacology: Suppressing multiple pathogenic pathways simultaneously with a single therapeutic.

Overcoming resistance: Pairing miRNA adjuncts with standard biologics for deeper remission.

AI-driven discovery: Using machine learning to map novel targets for treatment-resistant cases.

Conclusion

miRNAs have potential applications across rheumatology, including prognosis, diagnosis, assessment of inflammatory activity, evaluation of end-stage organ damage, and therapeutic intervention. Their ability to regulate multiple immune and inflammatory pathways, together with their stability and accessibility in body fluids, supports their potential as minimally invasive biomarkers and as targets for mechanism-based therapeutics. However, no miRNA-based therapeutic has yet been approved for the treatment of rheumatological diseases, and these approaches remain investigational.



Title: Graph-Theoretic and Petri Net Based Modeling of RNA–Protein Interaction Networks

Author: Dr. Gajendra P. Singh, JNU, New Delhi

Co Author: N/A

Abstract ID: APFRTV 2026 - 27

Abstract Body: RNA–protein interactions (RPIs) play a crucial role in gene regulation, RNA processing, stability, translation, and cellular function. This talk presents a computational framework integrating Graph Theory and Petri Nets for modeling and analysing RPI networks. RNA and protein molecules are represented as nodes in a bipartite graph, with interactions represented as edges. Graph-theoretic measures such as degree, centrality, connectivity, and community structure can be used to identify important interaction patterns and functional modules.

Petri Nets are further employed to represent the dynamic behaviour of RNA–protein interactions, including binding, regulation, synthesis, and degradation. The framework also provides opportunities for integrating Boolean Petri Nets, machine learning, and graph-based AI for interaction prediction and biological network analysis. The approach has potential applications in computational biology, disease modeling, RNA therapeutics, and vaccine research.

Keywords: RNA–protein interactions; Graph Theory; Petri Nets; Bipartite Graphs; Biological Networks; Computational Biology; Boolean Petri Nets; Machine Learning; RNA Therapeutics.



Title: RNA binding protein HuR drives glycolytic reprogramming in Triple negative breast cancer: A novel therapeutic avenue

Author: Dr. Chandra Prakash Prasad, AIIMS, New Delhi

Co Author: N/A

Abstract ID: APFRTV 2026 - 28

Abstract Body: Human Antigen R (HuR/ELAVL1), an RNA-binding protein, stabilizes and translates mRNAs with U/AU-rich elements in their 3' UTR, driving cell proliferation, survival, metastasis, tumor growth, and therapy resistance, hence, positioning it as a key prognostic and therapeutic target in cancer. This study investigates HuR's role in triple-negative breast cancer (TNBC), the most aggressive breast cancer subtype. In silico analysis and immunohistochemistry assessed HuR and MMP9 expression, and survival correlation.

CMLD2-treated TNBC (MDA-MB-231, MDA-MB-468) cells were investigated for proliferation, migration, invasion, clonogenicity (2D/3D), lactate/acidification assays, immunofluorescence/cell fractionation for HuR induced physiological changes, and molecular changes were investigated using NanoString transcriptomics, RNA-IP, qPCR/Western for glycolytic proteins (PFKP/LDHA/MCT4).

In vivo tumor formation in BALB/c mice was also validated using CMLD2. IHC showed HuR (65%) and MMP9 (55%) overexpression in TNBC tissues (n=70); and in silico analysis linked high HuR to poor survival ($p=0.028$). CMLD2 downregulated HuR and its target (i.e., CDK2, MMP9, β -catenin) and EMT markers, in parallel reducing proliferation, migration, invasion, clonogenicity, lactate, and acidification.

Furthermore, transcriptomics revealed HuR inhibition induced changes in processes like glycolysis, cell cycle, DNA repair, amino-acid synthesis etc. HuR inhibition significantly downregulated glycolytic proteins i.e., PFKP, LDHA, and MCT4 at RNA and protein levels. In vivo, CMLD2 treatment impaired tumorigenesis in BALB/c mice via glycolysis modulation.

Overall, HuR overexpression in TNBC tissues correlated with poor survival. CMLD2 inhibition suppressed HuR targets, EMT, proliferation, invasion, glycolysis, and tumorigenesis in vivo suggesting HuR blockade as a strategy to halt TNBC progression and metastasis.

Oral Abstract Presentation

Title: Dengue Virus-Induced lncRNA Landscape Reveals Molecular Signatures of Impaired Megakaryopoiesis

Author: Preeti Sokhal, AIIMS, New Delhi

Co Author: Dr. Bhupendra Verma, AIIMS, New Delhi

Abstract ID: APFRTV 2026 - 02

Abstract Body: Dengue virus infection is linked with various hematological abnormalities majorly including thrombocytopenia resulting from impaired megakaryopoiesis. Viral infection triggers extensive reprogramming of host cellular networks as viruses exploit host regulatory machinery to establish successful infection.

Among the host factors involved in these virus-host interactions, long noncoding RNAs (lncRNAs) have emerged as important regulators of cellular homeostasis, influencing diverse biological processes through their ability to modulate gene expression at the epigenetic, transcriptional, post-transcriptional, and post-translational levels.

Various evidence highlights the regulatory role of long non-coding RNAs in hematopoietic differentiation and antiviral responses. However, their involvement in dengue-mediated megakaryocyte dysfunction remains poorly understood. In this study, we performed transcriptomic profiling using RNA sequencing to identify differentially expressed lncRNAs during DENV infection in a megakaryocytic model system.

Bioinformatic analysis revealed a distinct lncRNA expression signature associated with infection, with several candidates significantly dysregulated, suggesting their potential involvement in cellular stress, immune modulation, and platelet formation.

Functional enrichment analysis of lncRNA-associated target genes indicated their involvement in key biological pathways, including immune response modulation, cell cycle regulation, and megakaryocyte differentiation. Selected candidate lncRNAs were further validated using quantitative PCR, confirming their differential expression patterns.

Hence, this study provides new insights into the molecular alterations in lncRNA-mediated regulatory landscape that occur in megakaryocytes during DENV infection, revealing potential lncRNA biomarkers or therapeutic targets for managing Dengue-induced thrombocytopenia.

Title: Differential tRNA Fragmentation During Dengue Virus Infection: functional investigation of tRFs that modulate DENV replication and PI3K/Akt signaling

Author: Sushant Phadnis, AIIMS, New Delhi

Co Author: Dr. Bhupendra Verma, AIIMS, New Delhi

Abstract ID: APFRTV 2026 - 05

Abstract Body: Introduction: tRNA-derived RNA fragments (tRFs) are an abundant, conserved class of small non-coding RNAs produced by specific RNases (e.g., angiogenin, Dicer, ELAC2) under stress. Emerging evidence indicates tRFs regulate translation, stress responses and signaling pathways, but their roles in dengue infection are poorly understood. We used small non-coding RNA sequencing and targeted silencing to define how DENV infection alters tRNA fragmentation and to test whether selected tRFs influence viral replication and host PI3K/Akt signaling.

Aim: To characterize DENV-induced tRNA fragmentation and determine the functional impact of selected differentially expressed tRFs on viral replication and Akt phosphorylation.

Objectives

1. Identify differentially expressed tRFs during DENV infection by small-RNA sequencing.
2. Validation of selected tRFs using in-house optimized biotin-based Northern blot (BiNoB) Approach.
3. Functional characterisation of candidate tRFs and assess effects on DENV replication and PI3K/Akt signaling.

Methodology: Huh7 cells were profiled by small-RNA sequencing to identify DENV-induced tRFs. Selected tRFs were validated using our in-house Biotin-based Northern blotting (BiNoB). Candidate tRFs were selectively silenced using complementary antisense oligonucleotides. Functional effects were assessed by MTT, qPCR, Western blot (NS5, pan- and phospho-Akt), and TCID50 to quantify viral replication and host signaling.

Results: DENV infection induced widespread tRNA fragmentation: small-RNA profiling identified 733 significantly modulated tRFs, with 3'-tRFs broadly upregulated and many i-tRFs downregulated. BiNoB validated selective parental tRNA cleavage like tRNA_GluTTC and tRNA_ValTAC remained intact while tRNA_TyrGTA was depleted and confirmed differential expression of 3'tRF_48_GluTTC, 3'tRF_45_ValTAC, i-tRF_40_TyrGTA, i-tRF_25_LysCTT and i-tRF_28_LysCTT. 100 nM ASO used for functional assays.

Functionally, ASO-i-tRF_40 reduced replication $\approx 70\%$, ASO-i-tRF_25 reduced $\approx 40\%$, ASO-i-tRF_28 increased replication $\approx 50\%$, and ASO-3'tRF_45 had no effect. These results matched across Western blot (NS5), qPCR and TCID50. Silencing showed tRF-specific PI3K/Akt effects like i-tRF_40 and i-tRF_25 promote Akt phosphorylation, whereas i-tRF_28 negatively regulates it. These findings indicate distinct pro- and anti-viral roles for specific tRFs during DENV infection and host signaling.

Conclusion: DENV infection drives selective tRNA fragmentation, producing tRFs that have distinct functional consequences for viral replication and host signaling. Using BiNoB validation and ASO-mediated silencing, we show that individual tRFs can be pro- or anti-viral and modulate the PI3K/Akt pathway. i-tRF_40_TyrGTA and i-tRF_25_LysCTT promote DENV replication and Akt activation, while i-tRF_28_LysCTT acts as a negative regulator of both. These findings identify specific tRFs as mechanistic regulators and potential biomarkers or therapeutic targets for dengue virus infection.

Title: Inhibition of HepG2, DU-145, and MCF-7 Cell Lines by Thiosemicarbazone Derivatives (A3, A5, and A8)

Author: Amit Sarder, SAU, New Delhi

Co Author: Dr. Yuba Raj Pokharel, SAU, New Delhi

Abstract ID: APFRTV 2026 - 07

Abstract Body: **Abstract:** **Background:** Thiosemicarbazones (TSCs) are a group of active compounds synthesised by the condensation of thiosemicarbazide with aldehydes or ketones. TSCs inhibit DNA synthesis by targeting the enzyme ribonucleotide reductase, and their anticancer activity has already been reported. This study was designed to investigate the effects of TSC derivatives (A3, A5, and A8) on liver cancer cells (HepG2), prostate cancer cells (DU-145), and breast cancer cells (MCF-7).

Methods: The effects of A3, A5, and A8 derivatives on cell viability were studied by the crystal violet staining assay. The inhibitory effects of these derivatives on colony-forming ability were studied using the clonogenic assay. The anti-migratory effects and the effects on cell growth and division were assessed using the wound-healing assay and cell-cycle assay, respectively. The effects of these derivatives on cell death were studied by the propidium iodide staining assay.

Results: Both the crystal violet staining assay and clonogenic assay revealed dose-dependent cytotoxic effects of A3, A5, and A8 derivatives on liver, prostate, and breast cancer cells. The wound-healing assay demonstrated the anti-migratory potential of the derivatives. Cell cycle assay showed that the derivatives cause the cell cycle arrest in S-phase. Again, the propidium iodide staining assay suggested the apoptotic potentials of the derivatives.

Conclusion: Our experimental data suggest that thiosemicarbazone derivatives (A3, A5, and A8) exhibit significant cytotoxic and anti-migratory effects on liver, prostate, and breast cancer cells, and cell death in these cancer cells proceeds via apoptosis.

Keywords: Cancer, Thiosemicarbazones, HepG2, DU-145, MCF-7, Apoptosis.

Poster Abstract Presentation

Title: Dengue Virus-Induced lncRNA Landscape Reveals Molecular Signatures of Impaired Megakaryopoiesis

Author: Preeti Sokhal, AIIMS, New Delhi

Co Author: Dr. Bhupendra Verma, AIIMS, New Delhi

Abstract ID: APFRTV 2026 - 02

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Functional enrichment analysis of lncRNA-associated target genes indicated their involvement in key biological pathways, including immune response modulation, cell cycle regulation, and megakaryocyte differentiation. Selected candidate lncRNAs were further validated using quantitative PCR, confirming their differential expression patterns.

Hence, this study provides new insights into the molecular alterations in lncRNA-mediated regulatory landscape that occur in megakaryocytes during DENV infection, revealing potential lncRNA biomarkers or therapeutic targets for managing Dengue-induced thrombocytopenia.

Title: In Silico Characterization, Property Identification, and Functional Analysis of the Stress Response Protein 3 (SRP 3) with Accession Number AT1G01310, in *Arabidopsis thaliana*

Author: Debraj Saha, SAU, New Delhi

Co Author: Dr. Siddharth Dutta

Abstract ID: APFRTV 2026 - 03

Abstract Body: *Arabidopsis thaliana* is a well-established model organism extensively employed to investigate the molecular basis of plant growth, development, and adaptation to environmental challenges. Despite the availability of genomic information, the protein encoded by AT1G01310 (SRP3) remains insufficiently characterized, and its structural and functional attributes are yet to be fully elucidated. The present study was undertaken to provide a comprehensive in silico characterization of SRP3, with particular emphasis on its potential function, regulatory mechanisms, expression profile, and structural properties. A combination of bioinformatics tools, databases, and computational approaches was employed to investigate the molecular characteristics of SRP3.

The BAR database was utilized to identify putative protein–protein interactions and infer potential functional associations. Gene Ontology analysis provided insights into the probable biological processes, cellular components, and molecular functions associated with the protein. Co-expression analysis using ATTED-II revealed associations with genes involved in stress-responsive mechanisms, metabolic activities, biosynthetic processes, and phytohormone-mediated signalling, particularly pathways associated with abscisic acid and gibberellin. These observations indicate a possible involvement of SRP3 in the regulation of plant responses to environmental stress.

Furthermore, PLACE-based promoter analysis of the 1253 bp upstream regulatory region identified several putative cis-regulatory elements that may contribute to the transcriptional regulation of SRP3 under diverse developmental and environmental conditions. Expression profiling through ePlant and the Plant eFP Viewer demonstrated differential expression across tissues, developmental stages, and various biotic and abiotic stress conditions. Structural modelling using SWISS-MODEL, complemented by physicochemical and Kyte–Doolittle hydropathy analyses, suggested that SRP3 is likely to possess predominantly hydrophilic and soluble characteristics. Collectively, the findings provide a comprehensive computational framework for understanding the potential biological significance of SRP3 and establish a foundation for future experimental validation and functional characterization of this poorly characterized protein.

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Author: Debraj Saha, SAU, New Delhi

Co Author: Dr. Siddharth Dutta

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Title: In Silico Design and Optimization of a Single-Domain Antibody for Bispecific Cancer Immunotherapy Against the C-Terminal Region of Mesothelin

Author: Nitika Samanta, SAU, New Delhi

Co Author: Dr. Rinkoo D. Gupta, SAU, New Delhi

Abstract ID: APFRTV 2026 - 06

Abstract Body: Background: Mesothelin (MSLN) is a tumor-associated antigen overexpressed in several solid tumors, including pancreatic ductal adenocarcinoma, mesothelioma, and lung cancer, while having restricted expression in normal tissues, making it an attractive cancer target. However, MSLN shedding and poor T-cell infiltration into solid tumors make effective MSLN-targeted immunotherapy challenging. Therefore, the development of a single-domain antibody (sdAb) targeting the C-terminal region of Mesothelin could provide a promising therapeutic strategy for incorporation into a bispecific format, thus targeting MSLN and simultaneously recruiting T-cells.

Methods: A previously established humanized RO4 antibody (hRO4) generated against a C-terminal peptide of MSLN was used as the parental sequence. The CDR regions of the hRO4 scFv were retained, while multiple IGKV framework sequences were analyzed through Multiple Sequence Alignment to design different humanized VL constructs as sdAbs.

Structural modeling of these VL constructs was done through AlphaFold and the predicted structures were docked against the C-terminal region of MSLN using HADDOCK.

Further visualization and protein-protein interactions were studied using Discovery Studio and LigPlot. The lead construct was further optimized through in silico affinity maturation to improve its predicted binding with MSLN.

Results: The parental RO4 construct when initially docked on the C-terminal region of MSLN showed a HADDOCK score of -59.7 ± 2.9 . After screening the different engineered VL constructs, the best candidate achieved a HADDOCK score of -101.0 ± 1.7 , representing an approximately 41-unit shift towards a more favorable predicted interaction with the C-terminal region of MSLN.

Conclusion: Computational design and optimization generated a single-domain antibody construct with a favorable predicted interaction with the C-terminal region of MSLN. This construct, if made into a bispecific format targeting both MSLN and CD3, could potentially facilitate better tumor penetration due to its small size and promote T-cell-mediated cytotoxicity against MSLN-positive solid tumors. These findings highlight its potential for solid-tumor cancer immunotherapy.

Title: Unveiling the Theranostic Potential of Aurora A-TPX2 signalling in Gallbladder Cancer: A Synergistic In-Silico and Cell-based Evaluation.

Author: Nisha Manav, AIIMS, New Delhi

Co Author: Dr. Chandra Prakash Prasad, AIIMS, New Delhi

Abstract ID: APFRTV 2026 - 08

Abstract Body: Introduction: Gallbladder cancer (GBC) is the most common malignancy of the biliary tract and remains a major clinical challenge due to late presentation and aggressive behavior. Median survival is under one year and overall survival ranges from approximately 17.8% to 21.7%. These poor outcomes are driven by a high propensity for local invasion and distant metastasis. The molecular drivers of GBC progression and dissemination remain incompletely characterized, limiting development of effective targeted therapies and sensitive diagnostic biomarkers. The Aurora A-TPX2 signaling axis is a key regulator of mitotic spindle assembly and cell cycle progression, frequently dysregulated in multiple malignancies and implicated in genomic instability, proliferation, and therapy resistance. Integrated computational and cellular interrogation of this axis in GBC may reveal mechanistic insights and identify candidate theranostic targets to improve diagnosis, prognostication, and treatment.

Materials and methods: Public cancer transcriptome repositories were mined to quantify AURKA and TPX2 expression, analyze co expression relationships, and evaluate associations with clinical features and survival. Established gallbladder cancer cell lines were subjected to pharmacologic inhibition of the AURKA-TPX2 axis through AURKIN A. Functional assays included cell proliferation assays (MTT), wound healing and cell migration/invasion assays, and colony formation assays. Also, the expression of downstream targets of this signaling axis were analysed using western blotting. Statistical significance was set at $p < 0.05$

Results: AURKA and TPX2 were significantly overexpressed across multiple tumor types, including GBC, and their expression levels showed a strong positive correlation. Elevated expression correlated with adverse clinicopathological features.

Pharmacological disruption of the AURKA-TPX2 axis with AURKIN A markedly reduced GBC cell proliferation, colony formation, migration, and invasion in vitro, supporting a functional oncogenic role for this signaling module. It also reduced the expression of its downstream targets.

Conclusion: Our integrated computational and experimental study demonstrates that the Aurora A-TPX2 axis is upregulated and functionally oncogenic in gallbladder cancer. Pharmacologic targeting of this axis with AURKIN A suppresses core malignant phenotypes in cell-based models, supporting AURKA-TPX2 as a promising theranostic target for GBC. These findings justify further preclinical evaluation and translational development to assess therapeutic potential and biomarker utility in clinical settings.

Title: Integrated Bioinformatics and Network-Based Identification of a Potential miRNA Mimic Therapeutic Axis for Pancreatic Ductal Adenocarcinoma

Author: Nishi Sharma, Omnicell Research Facility, New Delhi

Co Author: Dr. Manish Sharma, Omnicell Research Facility, New Delhi

Abstract ID: APFRTV 2026 - 09

Abstract Body: Background: Pancreatic ductal adenocarcinoma (PDAC) is an aggressive cancer with poor prognosis and limited treatment options. MicroRNAs (miRNAs) regulate gene expression after transcription and may offer opportunities for developing RNA-based therapies. This study aimed to identify a PDAC-associated miRNA–mRNA regulatory axis with potential for miRNA mimic-based therapeutic development.

Methods: TCGA-PAAD miRNA and mRNA expression data were analyzed following sample matching, expression filtering, and differential expression analysis using DESeq2. miRNA–gene interaction data were integrated with the differentially expressed molecules, and candidate genes were screened against the KEGG pancreatic cancer pathway (hsa05212). Gene Ontology (GO) and KEGG enrichment analyses were performed, followed by Cytoscape-based network analysis and STRING protein–protein interaction analysis. Potential therapeutic candidates were further evaluated using expression correlation, survival analysis, literature screening, and independent target-prediction resources.

Results: The analysis identified 21 differentially expressed miRNAs, including 13 upregulated and 8 downregulated miRNAs. Integration of target information produced 51,034 unique miRNA–gene–regulation records, including 9,851 genes targeted by multiple miRNAs. Fifty-six genes overlapped with the KEGG pancreatic cancer pathway. Network analysis identified AKT1, TP53, KRAS, STAT3, EGFR, CCND1, CDC42, MAPK3, MTOR, and NFKB1 as major hub genes, all showing increased expression in PDAC. Among the candidate interactions, hsa-miR-342-5p–CCND1 showed the strongest overall support. Precursor hsa-miR-342 was downregulated ($\log_2\text{FC} -1.02$; adjusted $p=0.0471$), while CCND1 was significantly higher in tumors ($p=0.02832$). An inverse correlation was observed between their expression (Spearman $\rho=-0.291$; $p=6.611\times 10^{-5}$).

Conclusion: The miR-342-5p–CCND1 axis represents a potentially novel therapeutic candidate in PDAC. Its biological and therapeutic relevance should be established through experimental validation of miRNA mimic activity, CCND1 regulation, and associated cellular effects.

Keywords: Pancreatic ductal adenocarcinoma; miR-342-5p; CCND1; miRNA mimic; RNA therapeutics; TCGA-PAAD; bioinformatics; miRNA–mRNA regulation.

Title: Exploration of Exosomal Proteins as a Non-invasive Biomarker for the Diagnosis of Gallbladder Cancer

Author: Akshya Mishra, SAU, New Delhi

Co Author: Girijesh Kumar Patel & Ujla Minhas

Abstract ID: APFRTV 2026 - 10

Abstract Body: Background: - Gallbladder cancer (GBC) is an aggressive malignancy with a poor prognosis, mainly due to late diagnosis and early spread to distant organs. The lack of specific biomarkers for early detection makes diagnosis challenging. Exosomes are small extracellular vesicles that contain proteins, lipids, mRNA, microRNA, and DNA fragments and may serve as a promising source of non-invasive biomarkers. This study focused on identifying exosomal proteins that could be useful for the early detection and progression of GBC.

Methods: - Different expression analyses were performed to identify exosomal proteins associated with GBC. Serum-derived exosomes were obtained from GBC patients and age-matched healthy controls. Mass spectrometry-based proteomic analysis was used to identify proteins that were differentially expressed between the two groups.

Results: - Among the identified proteins, NME2 (NME/NM23 Nucleoside Diphosphate Kinase 2) showed significant overexpression in serum-derived exosomes from GBC patients compared with healthy controls. NME2 has been reported to play important roles in tumor progression, metastasis, and cellular signaling pathways.

Conclusion: - Exosomal proteins, particularly NME2, may serve as promising non-invasive biomarkers for the diagnosis and progression of gallbladder cancer. However, further experimental validation and clinical studies with larger sample sizes are required to confirm their diagnostic and therapeutic potential.

Title: Enhanced immunogenicity of the tandem-repeat Fubc epitopes designed from the highly conserved regions of the dengue virus envelope protein

Author: Nidhi Dhama, SAU, New Delhi

Co Author: Barnali Halder, Satyajit Podder, Md Fahim Khalid, Dr. Rinkoo Devi Gupta

Abstract ID: APFRTV 2026 - 11

Abstract Body: Dengue virus (DENV) infection remains a major global health concern, and developing a safe and broadly effective vaccine is challenging due to its four serotypes and the risk of antibody-dependent enhancement (ADE). We previously identified the highly conserved fusion (Fu) and bc loop regions of the DENV envelope protein as potential immunogenic epitopes and developed the recombinant Fubc antigen. Here, we evaluated two strategies to enhance its immunogenicity: C-terminal solubility-controlling peptide (SCP) tagging and tandem-repeat multimerization (TR2, TR3 and TR4). Recombinant proteins were evaluated for immunogenicity in BALB/c mice by measuring anti-Fubc IgG responses and dose-normalized molar potency.

Fubc-SCP showed 1.41-fold higher endpoint IgG titers than Fubc, although the difference was not statistically significant. In contrast, tandem-repeat constructs significantly enhanced antibody responses, with TR3 showing a 16-fold increase in endpoint titer and a 34.8-fold increase in molar potency compared with monomeric Fubc. SPR analysis further supported enhanced antibody binding, while preliminary DENV2 neutralization assays demonstrated reduced plaque formation in sera from immunized groups. Overall, tandem-repeat presentation substantially enhanced the immunogenicity of the conserved Fubc epitope, supporting its further evaluation as a potential peptide-based dengue vaccine candidate.

Title: c-MYC Transcriptionally Regulates DUBs to Sustain the MGMT–Mediated Temozolomide Resistance Axis in Glioblastoma

Author: Devansh Sharma, Dr. B.R. Ambedkar Center for Biomedical Research, University of Delhi

Co Author: Prof. Ajay Kumar Yadav, ACBR, New Delhi

Abstract ID: APFRTV 2026 - 12

Abstract Body: Background: Glioblastoma (GBM) is a fatal disease, with resistance to temozolomide (TMZ) driven predominantly by expression of the DNA repair enzyme MGMT. The oncogenic transcription factor c-MYC, stabilised via serine-62 phosphorylation, has been implicated in chemoresistance, but the transcriptional targets sustaining TMZ resistance in GBM remain incompletely defined. We investigated whether c-MYC directly drives transcription of the deubiquitinases USP5 and USP10 to sustain MGMT-mediated resistance.

Methods: Pan-cancer and GBM-specific expression of MYC, USP5, USP10, and HNRNPA1 was profiled using GEPIA across 33 TCGA tumour types. MYC/MAX E-box motifs within the USP5 proximal promoter were mapped using EPD and validated against the JASPAR position weight matrix. Direct c-MYC chromatin occupancy at the USP5 and USP10 promoters was assessed by ChIP-PCR using phospho-(S62)- and total-c-MYC antibodies in parental and TMZ-resistant U87 MG cells. Protein and transcript changes were quantified by western blot, semiquantitative PCR, and real-time qPCR. USP5 and MGMT localisation following c-MYC inhibition with 10058-F4 (20–50 μ M, 48h) was assessed by immunofluorescence microscopy.

Results: MYC, HNRNPA1, and USP10 were significantly upregulated in GBM relative to normal brain tissue ($p < 0.05$). Three MYC/MAX E-box motifs were identified within 900 bp of the USP5 transcription start site. Phospho-c-MYC(S62), but not total c-MYC, was selectively elevated (1.2–2.0-fold) in TMZ-resistant cells, and ChIP-PCR confirmed preferential c-MYC occupancy at both promoters exclusively in resistant cells. 10058-F4 reduced USP5 mRNA from a 2.07-fold baseline to 0.56-fold and 0.43-fold at 20 and 50 μ M respectively, with concordant reductions in USP10, hnRNPA1 Var2, and MGMT at both transcript and protein levels.

Conclusion: c-MYC, stabilised post-translationally through S62 phosphorylation directly upregulates USP5 and USP10 to sustain MGMT-dependent TMZ resistance in GBM. Pharmacological disruption of c-MYC/MAX dimerisation reverses this resistance axis and hence the c-MYC-USP5-MGMT can be a druggable therapeutic target in glioblastoma

Title: The Non-Coding Transcriptome in Autophagy

Author: Bhawana Kadyan, KSBS, IIT, Delhi

Co Author: N/A

Abstract ID: APFRTV 2026 - 13

Abstract Body: Autophagy or “self-eating” is an evolutionarily conserved process that helps in the degradation of various intracellular components by targeting them to the lysosomes for recycling metabolites. Autophagy and lysosomal biogenesis are essential for cellular homeostasis, particularly under stress conditions such as nutrient deprivation and hypoxia. It is well known that autophagy inhibitor Bafilomycin A1 (BafA1) inhibits autophagosome–lysosome fusion and disrupts autophagic flux leading to autophagosome accumulation and metabolic stress.

We hypothesized that this blockade may trigger a transcriptional feedback loop involving long non-coding RNAs (lncRNAs) and protein-coding genes. To test this hypothesis, we analyzed publicly available GEO transcriptomic datasets generated from control and BafA1 treated cell lines. We identified over 100 lncRNAs and several protein coding genes to be differentially expressed ($p \leq 0.05$) upon Bafilomycin A1 treatment.

The candidate lncRNAs were further sorted based on basal expression levels and BafA1 dependent altered expression in more than 10 cell lines. Experimental validation of these transcripts through qPCR is underway and we further aim to investigate these lncRNAs, which may act as transcriptional feedback mediators to fine-tune autophagy related pathways.

Title: Next-Generation Sequencing-Based Profiling of Plasma miRNAs for Identification of Potential Early Diagnostic Biomarkers in Coronary Artery Disease

Author: Fayez M Salik, Jamia Hamdard University, New Delhi

Co Author: Sanjali Pramanik, Vidhi Oza, Dr. Pratibha Sharma

Abstract ID: APFRTV 2026 - 14

Abstract Body: Background: Coronary artery disease (CAD) is a major cardiovascular disorder characterized by atherosclerotic plaque formation and progressive impairment of coronary blood flow, contributing substantially to global morbidity and mortality. The identification of sensitive molecular biomarkers capable of detecting disease-associated molecular alterations at an early stage remains an important clinical need. Circulating microRNAs (miRNAs), small non-coding RNAs that regulate gene expression post-transcriptionally, have emerged as promising candidates for cardiovascular biomarker discovery from exosomes or small extracellular vesicles. Next-generation sequencing (NGS)-based small-RNA profiling enables comprehensive characterization of circulating miRNAs and facilitates the identification of differentially expressed miRNAs associated with disease phenotypes. The present study aimed to characterize the plasma exosome- miRNA profile of individuals with CAD using NGS and identify differentially expressed miRNAs with potential utility as early diagnostic biomarkers.

Methods: Plasma samples were collected from 16 participants comprising patients with CAD (n=8) and age- and sex-matched healthy controls (n=8). Exosomes were isolated using density-gradient ultracentrifugation and characterized by nanoparticle tracking analysis, transmission electron microscopy, immuno-electron microscopy, scanning electron microscopy, and Western blotting. Total RNA was individually extracted from isolated exosomes, and RNA quantity and quality were assessed using NanoDrop and Agilent Bioanalyzer. Small-RNA libraries were prepared using the QIAseq miRNA Library Kit and subjected to single-end 50-bp sequencing on an Illumina NovaSeq 6000 platform. Following adapter removal and quality filtering, reads of 16–40 nucleotides were retained for downstream analysis. Known miRNAs were identified based on sequence homology with miRBase, while putative novel miRNAs were predicted based on genomic alignment and precursor stem-loop structures. Differential miRNA expression was assessed using DESeq, followed by target prediction, pathway analysis, and Gene Ontology enrichment analysis.

Results: Approximately 17 million raw reads were generated per sample, of which an average of 13 million high-quality reads per sample were retained following length and quality filtering. Across the 16 samples, an average of 748 known miRNAs representing 371 miRNA families and approximately 392 putative novel miRNAs were identified. A total of 1,086 known miRNAs were detected in both study groups. Differential expression analysis identified 205 upregulated and 207 downregulated miRNAs in CAD compared with healthy controls, based on the predefined log₂ fold-change threshold. In addition, 149 miRNAs were detected exclusively in controls, whereas 120 miRNAs were detected exclusively in CAD samples. Following correction for multiple testing, three miRNAs—hsa-miR-107, hsa-miR-3184-5p, and hsa-miR-486-3p—demonstrated statistically significant differential expression, with all three showing reduced expression in CAD. Hierarchical clustering and heatmap visualization further demonstrated distinct miRNA expression patterns between the CAD and control groups, suggesting the presence of disease-associated circulating miRNA signatures.

Conclusion: NGS-based plasma miRNA profiling identified distinct circulating miRNA expression patterns associated with CAD and highlighted hsa-miR-107, hsa-miR-3184-5p, and hsa-miR-486-3p as candidate biomarkers. These findings provide a basis for further investigation of circulating miRNAs as minimally invasive molecular biomarkers for CAD. Further validation of these miRNAs using RT-qPCR in larger Indian population for CAD will present diagnostic performance and potential utility for early CAD detection.

Title: Unveiling Host Nucleoporins as Regulators of Influenza A Virus Infection: Implications for Host-Directed Antiviral Therapy

Author: Kajal Sharma, Vallabhbhai Patel Chest Institute, University of Delhi

Co Author: Prakash Jha, Binod Kumar, Madhu Chopra, Jai Gopal Sharma, Dr. Madhu Khanna*

Abstract ID: APFRTV 2026 - 15

Abstract Body: Background: Influenza A virus (IAV) is a negative-sense single-stranded RNA virus that uses the host nucleocytoplasmic transport machinery to transport viral ribonucleoprotein (vRNP) complexes. Viruses exploit the nuclear pore complex (NPC), composed of nucleoporins (NUPs), as a critical interface for molecular exchange between the nucleus and cytoplasm. In this study, we examined interactions between human nucleoporins and IAV using *in silico* and experimental approaches.

Methods: Molecular docking was used to predict binding affinities at the protein-protein interface, and molecular dynamics simulations were used to evaluate the conformation and structural stability of the protein complexes under physiological conditions. Subsequently, *in vitro* studies were performed to validate the functional relevance of the identified interactions, including expression analysis during infection and siRNA-mediated silencing of nucleoporins.

Results: Computational analysis showed crucial interactions between nucleoporins NUP54 and NUP62 and the IAV vRNP protein throughout the 100 ns simulation time. NUPs engaged with influenza nucleoprotein through distinct mechanisms. The NUP54-NP complex showed dynamic rearrangements, while the NUP62-NP complex was stable. The early increase in NUP54 expression and viral NP accumulation in the infected cells suggests an active role of NUP54 in viral replication. siRNA-mediated silencing of NUP54 reduced viral infection by about 60–70%, suggesting that NUP54 contributes significantly to vRNP trafficking.

Conclusion: The study provides novel insights into the role of NPC-associated proteins in replication of influenza A virus. Identification of host factors such as NUP54 and NUP62 in viral infection highlights their potential as antiviral therapeutic targets to overcome rapid antigenic drift and antiviral resistance and provides a framework for the functional characterization of host-virus mechanisms that could inform host-directed vaccine strategies.

Title: Liposome-Based Delivery Platform for Influenza Antigenic RNA: Toward Stable and Efficient mRNA Vaccine Development

Author: Archana, Vallabhbhai Patel Chest Institute, University of Delhi

Co Author: Sanjesh Saini, Madhu Khanna, Adib Khan, Mairaj Ahmed Ansari

Abstract ID: APFRTV 2026 - 16

Abstract Body: Background: Messenger RNA (mRNA) vaccines have redefined the landscape of modern immunization; however, achieving a stable and efficient RNA delivery system remains a major hurdle. Overcoming this limitation is crucial for rapidly mutating RNA viruses such as influenza, where adaptable vaccine platforms are essential. In this study, we developed a liposome-based RNA delivery system to transport influenza virus NS1 RNA to antigen-presenting cells effectively.

Methods: The influenza antigen (NS1) gene was cloned and transcribed in vitro to produce capped and polyadenylated RNA constructs. Egg phosphatidylcholine (PC) was isolated and utilized to prepare PC-based liposomes through thin-film hydration method, followed by characterization for particle size, uniformity, and stability. The transcribed RNA was then encapsulated within the liposomes, and the encapsulation efficiency was assessed using spectrophotometric and gel electrophoresis analyses. To evaluate delivery performance, GFP-tagged RNA was incorporated into the liposomes and introduced into macrophage cells, enabling visualization and assessment of targeted intracellular delivery and expression efficiency.

Results: The liposomal formulation yielded intact and uniform vesicles with over 80% RNA loading efficiency and a stable nanoparticle size distribution, confirming its suitability for RNA transport. The encapsulated mRNA maintained structural integrity, a prerequisite for effective intracellular delivery and antigen expression.

Conclusion: This study presents a novel approach that merges RNA construct with optimized lipid encapsulation, emphasizing delivery precision and molecular protection as critical parameters. The findings outline a promising platform for RNA-based influenza vaccines and offer adaptability for other emerging viral pathogens.

Title: Therapeutic potential of Insulin-like growth factor-1 (IGF-1) in Polyglutamine (polyQ) neurotoxicity

Author: Kaushiki & Shovan Kundu, SAU, New Delhi

Co Author: Md. Mostafizur Rahman, Rishabh Rai, Dr. Ravi Shankar Akundi

Abstract ID: APFRTV 2026 - 17

Abstract Body: Polyglutamine (polyQ) neurotoxicity is a group of neurodegenerative disorders caused by polyglutamine expansion in the host protein, which confers the protein a dominant gain of function and leads to cellular dysfunction as well as death. Among the polyQ disorders, spinocerebellar ataxia type 3 (SCA3) is highly prevalent worldwide, and is caused by CAG trinucleotide tandem repeat expansion in the coding region of ataxin-3, which leads to abnormal protein aggregation, inclusion body formation and neuronal death.

There currently is no cure for SCA3. In this research, it was aimed to test the cytoprotective role of insulin-like growth factor-1 (IGF-1) against poly(Q)-mediated cell death. To test this objective, an in vitro model was used, wherein HEK293T cells were transfected with pEGFP-C1-ataxin3Q84 (SCA3) plasmid in the presence or absence of IGF-1. The results show that, treatment with IGF-1 shifted the location of aggregates to the cytosol instead of the more prominent and toxic nuclear aggregates observed in the absence of IGF-1.

Furthermore, to analyze the molecular and mechanistic property, p38 MAPK activation was checked, and it was found that, the level of Phospho-p38 was reduced with IGF-1 treatment, which in turn decreases cell death. Additionally, higher Hsp70 (cellular stress marker and molecular chaperone) in SCA3 stress condition, was also decreased with IGF-1 treatment.

The results of ataxin3Q84-mediated effects were normalized with the effects seen in cells transfected with the wildtype protein-ataxin3Q28. This study shows the potential cytoprotective role of IGF-1 in polyQ disorders, and has implications for future research in this area.

Title: Lipid Nanoparticle-Mediated Delivery of Plumbagin Enhances Anticancer Responses in A431 Skin Cancer Cells

Author: Lam Dorji Tamang, SAU, New Delhi

Co Author: Dr. Yuba Raj Pokharel and Madhav Dhakal, SAU, New Delhi

Abstract ID: APFRTV 2026 - 18

Abstract Body: Plumbagin, a naturally occurring naphthoquinone, exhibits promising anticancer activity but is limited by poor water solubility, low bioavailability, rapid metabolism, and potential toxicity. Lipid nanoparticle-based delivery may improve the biological effectiveness of plumbagin by enhancing its delivery to cancer cells. This study investigated and compared the anticancer effects of free plumbagin (FP) and plumbagin-loaded lipid nanoparticles (LP) in A431 human epidermoid carcinoma cells.

Cytotoxicity was evaluated using a crystal violet assay, while colony formation, wound healing, apoptosis, cell cycle progression, DNA fragmentation, three-dimensional (3D) spheroid formation, and western blotting were used to assess the effects of treatment on cellular growth and apoptotic responses. LP demonstrated greater cytotoxicity than FP, with IC values of 2.3 μ M and 5.8 μ M, respectively. LP also showed stronger inhibition of colony formation, cell migration, and 3D spheroid growth compared with FP.

Flow cytometric analysis revealed increased apoptosis following treatment, with $29.77 \pm 0.34\%$ apoptotic cells after LP treatment at 3 μ M compared with $27.55 \pm 0.26\%$ following FP treatment at 10 μ M. Cell cycle analysis showed distinct responses, with FP at 10 μ M inducing marked S-phase accumulation ($71.45 \pm 2.06\%$), whereas LP at 3 μ M increased the G2/M population ($27.13 \pm 1.01\%$).

DNA fragmentation was more pronounced following LP treatment, and western blot analysis showed increased p53 and reduced Bcl-xL expression. Overall, LP exhibited enhanced anticancer activity compared with free plumbagin across multiple cellular and molecular endpoints, highlighting the potential of lipid nanoparticle-mediated delivery for improving plumbagin-based cancer therapy.

Keywords: Plumbagin; Lipid nanoparticles; A431 cells; Skin cancer; Apoptosis; Cell cycle; Nanomedicine

Title: Identification and Validation of NONO as a Molecular Target of CuHDm in Lung Cancer

Author: Madhav Dhakal, SAU, New Delhi

Co Author: Dr. Yuba Raj Pokharel, SAU, New Delhi

Abstract ID: APFRTV 2026 - 19

Abstract Body: Lung cancer is a leading cause of cancer related mortality worldwide. There is a need for identification of novel molecular targets and therapeutic agents. In our study, an integrative bioinformatics and experimental approach was used to identify and validate key molecular targets of CuHDm, a coumarin-thiosemicarbazone copper complex with anticancer activity.

We in our study used transcriptomic data from three independent GEO datasets (GSE81089, GSE19188, and GSE19804) and identified 408 commonly dysregulated genes in lung adenocarcinoma (LUAD). In the analysis, functional enrichment analysis revealed significant association with RNA processing, mRNA splicing, and post-transcriptional regulation pathways. Protein-protein interaction network analysis using STRING and Cytoscape identified NONO (Non-POU Domain-Containing Octamer-Binding Protein) as a top-ranked hub gene.

Clinical validation using the UALCAN database confirmed significant upregulation of NONO in LUAD tumor tissues compared to normal tissues. Molecular docking of CuHDm with NONO using CB-Dock2 revealed a strong binding affinity of -8.2 kcal/mol. Experimental validation demonstrated that CuHDm treatment significantly downregulated NONO expression at both protein and mRNA levels in A549 and NCI-H460 lung cancer cell lines.

Western blot analysis showed concentration-dependent reduction of NONO protein, while qPCR confirmed downregulation of NONO mRNA expression.

Collectively, these findings identify NONO as a novel molecular target of CuHDm in lung cancer, providing a mechanistic basis for its anticancer activity. The integration of computational prediction with experimental validation establishes a framework for target identification and supports further investigation of CuHDm as a promising therapeutic candidate.

POSTER SESSIONS

FINAL ID	TITLE	PRESENTER NAME	STATE/COUNTRY
APFRTV 2026 - 02	Dengue Virus-Induced lncRNA Landscape Reveals Molecular Signatures of Impaired Megakaryopoiesis	Preeti Sokhal	India
APFRTV 2026 – 03	In Silico Characterization, Property Identification, and Functional Analysis of the Stress Response Protein 3 (SRP 3) with Accession Number AT1G01310, in <i>Arabidopsis thaliana</i>	Debraj Saha	India
APFRTV 2026 - 04	In Silico Characterization, Property Identification, and Functional Analysis of the Stress Response Protein 3 (SRP 3) with Accession Number AT1G01310, in <i>Arabidopsis thaliana</i>	Debatri karmakar	India
APFRTV 2026 - 06	In Silico Design and Optimization of a Single-Domain Antibody for Bispecific Cancer Immunotherapy Against the C-Terminal Region of Mesothelin	Nitika Samanta	India
APFRTV 2026 - 08	Unveiling the Theranostic Potential of Aurora A-TPX2 signalling in Gallbladder Cancer: A Synergistic In-Silico and Cell-based Evaluation.	Nisha Manav	India
APFRTV 2026 - 09	Integrated Bioinformatics and Network-Based Identification of a Potential miRNA Mimic Therapeutic Axis for Pancreatic Ductal Adenocarcinoma	Manish Sharma	India
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APFRTV 2026 - 15	Unveiling Host Nucleoporins as Regulators of Influenza A Virus Infection: Implications for Host-Directed Antiviral Therapy	Kajal Sharma	India
APFRTV 2026 - 16	Liposome-Based Delivery Platform for Influenza Antigenic RNA: Toward Stable and Efficient mRNA Vaccine Development	Archana	India
APFRTV 2026 - 17	Therapeutic potential of Insulin-like growth factor-1 (IGF-1) in Polyglutamine (polyQ) neurotoxicity	Kaushiki	India
APFRTV 2026 - 18	Lipid Nanoparticle-Mediated Delivery of Plumbagin Enhances Anticancer Responses in A431 Skin Cancer Cells	Lam Dorji Tamang	India
APFRTV 2026 - 19	Identification and Validation of NONO as a Molecular Target of CuHDM in Lung Cancer	Madhav Dhakal	India



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Start Time
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