ATHERO UPDATE

October 2026

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(An Official Publication of Indian Society for Atherosclerosis Research Delhi Chapter)
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CHIEF PATRON

Message from the Vice-Chancellor

It gives me great pleasure to welcome all delegates, distinguished speakers and young researchers to the 11th Annual Conference of the Indian Society for Atherosclerosis Research – Delhi Chapter (ISAR–DCCON 2026), hosted at Jamia Millia Islamia.

Since its founding in 1920, Jamia has stood for the pursuit of knowledge in the service of society. Cardiovascular disease, and atherosclerosis in particular, remains one of the gravest health challenges facing our nation, affecting people at a younger age and in greater numbers than ever before. Meeting this challenge demands exactly what this conference sets out to do: to bring the laboratory and the clinic into closer conversation.

The theme, “Bridging the Gap: From Molecular Mechanisms to Precision Management of Atherosclerosis”, reflects the interdisciplinary spirit that Jamia values deeply. I am particularly pleased that the programme gives young investigators a platform to present their work alongside leading clinicians and scientists from India and abroad.

I congratulate the Indian Society for Atherosclerosis Research – Delhi Chapter, the DDU Kaushal Kendra and the Faculty of Life Sciences for organising this conference. I wish all participants fruitful deliberations and a memorable stay at Jamia Millia Islamia.

Prof. Mazhar Asif
Vice-Chancellor
Jamia Millia Islamia, New Delhi

PATRON

Message from the Registrar

On behalf of Jamia Millia Islamia, I extend a warm welcome to all participants of ISAR–DCCON 2026, the 11th Annual Conference of the Indian Society for Atherosclerosis Research – Delhi Chapter.

Academic gatherings of this kind are central to the life of a university. They create opportunities for our faculty and students to engage with eminent clinicians and researchers, to exchange ideas across institutions, and to build collaborations that extend well beyond a single day.

The University is happy to support this conference and to host it at the Faculty of Engineering & Technology Auditorium. I hope that every delegate finds the arrangements comfortable and the hospitality of Jamia memorable.

I appreciate the dedicated efforts of the organising team, the Indian Society for Atherosclerosis Research – Delhi Chapter and the DDU Kaushal Kendra in bringing together such a distinguished scientific programme. I wish the conference every success and look forward to its outcomes contributing to better cardiovascular health in our country.

Prof. Md. Mahtab Alam Rizvi
Registrar
Jamia Millia Islamia, New Delhi

CO-PATRON

Message from the Dean, Faculty of Life Sciences

It is a privilege for the Faculty of Life Sciences to be associated with ISAR–DCCON 2026, and I welcome all speakers, chairpersons and delegates to Jamia Millia Islamia.

Atherosclerosis is a disease that begins at the level of molecules and cells, and ends in the clinic as heart attack and stroke. Understanding it fully requires biochemists, geneticists, structural biologists, pharmacologists and clinicians to work together. This conference, with its emphasis on biomarkers, molecular mechanisms, translational research and precision management, captures that spirit of collaboration well.

I am especially glad to see the space given to young investigators through oral and poster presentations. Our students and research scholars will benefit greatly from the opportunity to present their work and to learn directly from leaders in the field.

I compliment the organising committee for their hard work and wish all participants an enriching and productive conference.

Prof. Luqman A. Khan
Dean, Faculty of Life Sciences
Jamia Millia Islamia, New Delhi

CONVENER

Message from the Convener

It is my great pleasure to welcome you to ISAR–DCCON 2026 at Jamia Millia Islamia. The DDU Kaushal Kendra is honoured to host the 11th Annual Conference of the Indian Society for Atherosclerosis Research – Delhi Chapter.

The Kendra is committed to building skills and capacity that connect education with real-world needs. Organising a conference that links fundamental science with patient care is a natural extension of that mission. Over the course of one day, the programme brings together a plenary lecture, keynote addresses, invited talks, vision lectures by senior members of the Society, and presentations by young investigators.

I am deeply grateful to our Chief Patron, Patron and Co-Patron for their guidance and support, to the office bearers of ISAR – Delhi Chapter for their partnership, and to every speaker and chairperson who has generously given their time. My sincere thanks also go to the faculty, staff and student volunteers whose efforts have made this conference possible.

I wish you a stimulating and rewarding conference.

Prof. Md. Imtaiyaz Hassan
Convener, ISAR–DCCON 2026
Director, DDU Kaushal Kendra, Jamia Millia Islamia

COORDINATOR

Message from the Coordinator

Dear colleagues and friends, a very warm welcome to ISAR–DCCON 2026.

When we chose the theme “Bridging the Gap: From Molecular Mechanisms to Precision Management of Atherosclerosis”, our aim was simple: to bring basic scientists and clinicians into the same room, and to let each learn from the other. The scientific programme has been built around that idea. It moves from genomics, proteomics and structural biology to biomarkers, interventional cardiology and preventive care, and closes with the Society's vision for the future and the work of our young investigators.

I am grateful to our plenary and keynote speakers, invited faculty and session chairs for accepting our invitation, and to the poster and oral presentation judges for their time. I thank the leadership of Jamia Millia Islamia, the office bearers of ISAR – Delhi Chapter and the entire organising team for their unstinting support.

To the students and early-career researchers in particular: please ask questions, share your ideas and make the most of the day. I look forward to meeting each of you and hope ISAR–DCCON 2026 proves both rewarding and memorable.

Prof. Mohammad Zahid Ashraf
Coordinator, ISAR–DCCON 2026
Department of Biotechnology, Jamia Millia Islamia

PATRON

Message from the Patron

Dear Esteemed Members and Colleagues,

I am proud to have been associated with ISAR as -

- FOUNDER MEMBER
- Past President.
- PATRON,ISAR-DC

Honoured with Life Time Achievement awards **-** both by ISAR as well as ISAR-DC.

Bestowed with the honour to conduct 'BIENNIAL ELECTIONS OF ISAR-DC' **in the capacity of** Chief Election Officer.

I congratulate the New Team of office bearers, to be inducted during the conference on 10th October 2026. .

I am delighted to be associated with the theme of the conference: BRIDGING THE GAP: FROM MOLECULAR MECHANISMS TO PRECISION MANAGEMENT OF ATHERO-SCLEROSIS being conducted under the leadership of Prof Zahid Ashraf at Jamia Milia Islamia, New Delhi, Premier Central University.

I wish the conference a great success with highly academic feast.

Dr. D.K. Srivastava

Patron, ISAR-DC

Founder Member, ISAR

ADVISOR

Message from the Advisor

Dear Esteemed Members, Colleagues, Researchers and Students,

It is a matter of great satisfaction to witness the remarkable progress of ISAR Delhi Chapter in promoting scientific learning, academic excellence, and professional interaction.

I would like to particularly commend Dr. Jagrati, President; Dr Parul Goyal, Vice President; Dr Rajeev Goyal, Treasurer; and Dr. Harsh Vardhan Singh, Secretary, ISAR-DC, for their exemplary leadership and dedicated efforts throughout their tenure. Their vision, commitment, and enthusiasm have elevated the Association to new heights, creating a vibrant academic environment and encouraging meaningful scientific engagement.

The forthcoming conference on “Bridging the Gap: From Molecular Mechanisms to Precision Management of Atherosclerosis” is yet another reflection of the Association's commitment to advancing knowledge and research.

I also extend my heartfelt appreciation to Prof. Zahid Ashraf and the entire organizing team at Jamia Millia Islamia, New Delhi, whose dedicated efforts and academic vision have made this conference a valuable platform for bringing together eminent scientists, researchers, and academicians to exchange knowledge and explore new frontiers in medical science.

I congratulate everyone associated with this academic initiative and convey my best wishes for a highly successful, inspiring, and intellectually enriching conference.

Dr. Ritu Singh

Advisor, ISAR-DC

Former ISAR National and ISAR Delhi Chapter President

PRRSIDENT

Message from President

Dear Esteemed Members and Colleagues,

It is my utmost honor to connect with all of you through our newsletter, *Athero Update*. It was indeed my privilege, as well as that of my entire Executive committee, to serve our esteemed society. It is with great pleasure that I welcome the newly elected Executive team under the astute and charismatic leadership of Dr Seema Bhargava, our new President.

We are a multi-disciplinary society with a common goal to promote research and awareness in atherosclerosis by promoting academic activities. I am happy to note that our '11th Annual Conference of ISAR Delhi Chapter' is being organized on 10th October, 2026 at Faculty of Engineering & Technology Auditorium, DDU Kaushal Kendra, Faculty of Life Sciences, Jamia Millia Islamia, New Delhi under the able and experienced leadership of Dr Md Zahid Ashraf, Co-ordinator, ISAR-DCCON 2026 & Professor, Department of Biotechnology, JMI. We thankfully acknowledge your support, commitment and execution for a great ISAR-DCCON 2026 at JMI. My heartfelt thanks to Prof Mazhar Asif, Vice-Chancellor, JMI; Prof Md Mahtab Alam Rizvi, Registrar, JMI; Prof Luqman Ahmad Khan, Co-Patron & Dean, Faculty of Life Sciences, JMI; Prof Imtaiyaz Hassan, Convenor & Director, DDU Kaushal Kendra, JMI and all the organizing committee team members for extending whole-hearted support and working with dedication and passion for ensuring a brilliant ISAR-DCCON 2026. The theme of the conference is 'Bridging the gap: from Molecular Mechanisms to Precision Management of Atherosclerosis' and indeed we hope to bring together students, teachers, researchers, scientists and clinicians from Delhi-NCR region to a truly inspiring and immersive day of learning and building collaboration to foster growth in both research and clinical practice.

Lastly, I take this opportunity to convey my thanks to all of you for your unwavering commitment and support for ISAR Delhi Chapter! Let us work together towards our commitment for promoting good science and advancing research in the field of atherosclerosis and promoting holistic health!

Thanks, and warm regards,

Dr Jagriti Bhatia,

President,

ISAR Delhi Chapter & Professor,

Department of Pharmacology,

AIIMS New Delhi

PRESIDENT – ELECT

Message from President – Elect

Dear Colleagues and Members,

It gives me immense pleasure to extend my warm greetings and best wishes to all the distinguished speakers, delegates, researchers, and participants attending the ISAR-DC Conference on 10th October.

The conference theme, “Bridging the Gap: From Molecular Mechanisms to Precision Management of Atherosclerosis,” reflects the growing importance of translating scientific discoveries into meaningful advances in clinical practice.

I would like to place on record my sincere appreciation for Dr. Jagrati, President, ISAR-DC, and Dr. Harsh Vardhan Singh, Secretary, ISAR Delhi Chapter and Executive Committee Members for their outstanding contributions during their tenure. Their dedication, dynamic leadership, and tireless efforts have taken the Association to newer heights, fostering a vibrant academic environment and strengthening the spirit of scientific collaboration.

I am confident that this academic gathering will provide an excellent platform for the exchange of innovative ideas, scientific knowledge, and collaborative research.

I extend my sincere appreciation to Prof. Zahid Ashraf and the organizing team at Jamia Millia Islamia, New Delhi, for their dedicated efforts in bringing together the scientific community.

I wish the conference a grand success and look forward to continued academic excellence and meaningful contributions from ISAR-DC.

Dr. Seema Bhargava

President-Elect, ISAR-DC

VICE PRESIDENT

Message from Vice President

Dear Members,

It gives me immense pleasure to connect with all members through this issue of DC-ISAR Newsletter.

Our Society has grown over the years through the collective efforts, enthusiasm and commitment of its members. Beyond academic activities and scientific exchange, it has provided us with a platform to connect, collaborate and grow together as a professional community.

As we move forward, it is important that we continue to encourage young colleagues, promote meaningful academic and research initiatives, and create opportunities for interaction and collaboration.

I sincerely appreciate the efforts of the editorial team in bringing together this newsletter and thank all members who have contributed to it. I hope this issue provides an opportunity to share our achievements, ideas and experiences.

I congratulate Dr Zahid Ashraf and his team for organizing the upcoming CME on 10th October 2026 at Jamia Millia Islamia. I

Let us continue to work together with enthusiasm, friendship and a shared commitment to taking our Society to greater heights.

With warm regards and best wishes,

Dr. Parul Goyal

Vice President

DC-ISAR

SECRETARY

Message from Secretary

Dear Esteemed Members, Colleagues, Researchers and Students,

It is a pleasure to reach out to you through this edition of the ISAR Delhi Chapter Newsletter 'Athero update'. Each passing year brings new opportunities to learn, collaborate and contribute, and it is encouraging to witness the continued enthusiasm and involvement of our members in strengthening our scientific community.

ISAR Delhi Chapter represents a unique confluence of diverse disciplines, united by a shared commitment to advancing research, fostering awareness and improving our understanding of atherosclerosis. Our strength lies not only in scientific accomplishments but also in the exchange of ideas, meaningful professional relationships and encouragement we extend to the next generation of researchers and clinicians.

We eagerly look forward to the **11th Annual Conference of ISAR Delhi Chapter (ISAR-DCCON 2026)**, scheduled for **10th October 2026 at Jamia Millia Islamia, New Delhi**. The theme, **"Bridging the Gap: From Molecular Mechanisms to Precision Management of Atherosclerosis,"** highlights the growing importance of translating scientific discoveries into effective clinical strategies. I am confident that the conference will offer stimulating discussions, fresh insights and valuable opportunities for collaboration.

My warm congratulations to **Prof. Md Zahid Ashraf**, Conference Coordinator, and his organizing team for their dedication and efforts. I also sincerely acknowledge the invaluable support of **Prof. Mazhar Asif, Vice-Chancellor; Prof. Md Mahtab Alam Rizvi, Registrar; and Prof. Imtaiyaz Hassan, Convenor and Director, DDU Kaushal Kendra, Jamia Millia Islamia**, in making this academic gathering possible.

I extend my appreciation to the editorial team and all contributors for bringing this newsletter to our members. As we move ahead, let us continue to inspire young minds, embrace new scientific perspectives and strengthen the collaborative spirit that defines our Chapter. Our greatest progress will come from the knowledge we share, the partnerships we nurture and the impact we create together. Looking forward to meeting you all at ISAR-DCCON 2026.

With warm regards and best wishes,

Dr. Harsh Vardhan Singh

Indian Society for Atherosclerosis Research - Delhi Chapter (ISAR-DC)

Department of Biochemistry, North Delhi Municipal Corporation Medical College & Hindu Rao Hospital, Delhi-110007

TREASURER

Message from Treasurer

Dear Friends,

It gives me great pleasure to welcome you to the 11th Annual Conference of the Indian Society for Atherosclerosis Research, Delhi Chapter (ISAR-DCCON 2026), to be held on October 10, 2026, organized by Jamia Millia Islamia, New Delhi.

The theme this year, & quot; Bridging the Gap: From Molecular Mechanisms to Precision Management of Atherosclerosis, & quot; reflects our shared commitment to bringing laboratory discoveries to the bedside.

With cardiovascular diseases placing a growing burden on our country, forums like this, where basic scientists, clinicians, and industry come together, matter more than ever.

I sincerely thank the entire Organizing Committee for their initiative and dedication, which is working tirelessly to ensure that event is beneficial to all researchers working in the domain of atherosclerosis, especially the students and young researchers.

With warm regards,

Rajeev Goyal

Dr Rajeev Goyal

Treasurer, ISAR-DC

EDITOR-IN-CHIEF

Message from Editor – In – Chief

Dear Esteemed Members and Colleagues,

It gives me immense pleasure to note the progress of the Indian Society for Atherosclerosis Research, Delhi Chapter during the last few years, the way Delhi Chapter has grown at such a fast pace is commendable. Thanks to the younger generation working hard to take the Society higher. One example of their perseverance is the regular publication of Athero Update for the last two years. Though currently its contents are quite informative and useful for the readers. There is a definite need to make it a half yearly publication and quarterly in the near future so that more young members of society are able to publish their observations, work and reviews in their own journal. As a Patron and Chief Editor of Athero Update I wish to put on record my deepest sense of appreciation for Dr Jagriti, President ISAR DC and Dr Harsh Vardhan Singh, the dynamic Secretary and many others who have been working behind relentlessly to make this happen. I will fail in my duty if I don't mention the hard way Prof. Zahid Ashraf has been working day and night to make the Annual Conference of 2026 academically and professionally a rich experience. My heartiest compliments to the entire team of Athero Update 2026 and Annual Conference DC ISAR 2026. I wish the Society a future full of productivity, high-quality research and constructive output. Long Live DC ISAR and Long Live India.

Dr Shridhar Dwivedi, MD, PhD

Patron and Chief Editor -in -Chief

Athero Update, DC ISAR

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R1

Beyond Barker's Hypothesis to Precision Prevention

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Ancient Indian literature is rich in empirical observations that a child's physical, intellectual and emotional development is shaped by the mother's physical, nutritional, intellectual and emotional health during pregnancy. The best known is the story of Abhimanyu, son of Arjuna and Subhadra. While still in the womb, he heard Arjuna describe how to enter the Chakravyuha, the formidable battle formation of the Mahabharata war. Subhadra fell asleep before Arjuna could explain how to break out of it. Abhimanyu was therefore born knowing how to enter the formation but not how to exit, and he lost his life inside it.

The idea that parental qualities pass to the next generation appears again in Tulsidas's Ramcharitmanas. Tulsidas describes Sita, daughter of King Janaka, and Rama, son of King Dasharatha, as the living embodiment of their fathers' virtues (**जनक सुकृत मूरति बैदेही। दशरथ सुकृत रामु धरें देही॥**).

A rationalist may ask where the hard-scientific evidence for such narratives lies. Yet history offers its own testimony. Great personalities such as Chhatrapati Shivaji Maharaj, Mahamana Madan Mohan Malaviya, Swami Vivekananda, Dr B.C. Roy and Dr A.P.J. Abdul Kalam were deeply shaped by their mothers' convictions and early upbringing and each credited much of their success to that grooming. The mother's dominant role in building a child's character and way of life is beyond dispute.

It is equally true that the development of the foetus in the womb is affected by the mother's physical, mental and nutritional health. The heart and brain are among the first organs to form during embryogenesis, so the mother's emotional and physical wellbeing matters from the earliest weeks. The father is an equally important contributor. His genetic profile shapes the foetus, and his habits, such as smoking and alcohol use around conception and during his partner's pregnancy, also play a part. Beyond healthy parents, the developing foetus needs a cool, calm and serene environment throughout its nine months in the womb.

Figure 1: Mother's womb – baby's first nursery, Nurture it, culture this sanctuary. (माँ की कोख – शिशु की पहली पाठशाला ,इसे आहार दें , संस्कार दें न बनाएं धूम्रशाला -मधुशाला)[1](Picture Courtesy : Mrs Bhavna Pandey , Delhi based artist)

CARDIAC EMBRYOGENESIS: Cardiac embryogenesis can be affected by genetic factors, maternal factors, environmental factors & drugs / infections.[2]

Genetic factors:

Changes in genes or chromosomes can affect heart development, Chromosomal abnormalities eg:- Down syndrome (trisomy 21), Other inherited deletion syndrome, Family history of congenital heart disease (CHD).

Maternal disease:

Diseases in the mother during pregnancy can increase the risk, Diabetes Mellitus, especially if poorly controlled, Phenylketonuria (PKU) - It is inherited metabolic disorder where the body cannot break down the amino acids to phenylalanine leading to toxic buildup & amp; potential brain damage, Certain autoimmune diseases, Maternal nutritional deficiencies.

Maternal infections:

Some infections during early pregnancy can interfere with cardiac development, Rubella- It is associated with CHD, especially PDA, Other significant maternal infections may also contribute.

Drugs & chemical:

- Some medicine or chemical exposure during pregnancy can affect embryogenic development. Ex:- thalidomide, retinoic acid.

Nutritional factors:

- Poor maternal nutrients can interfere with normal fetal development, Folate deficiency is particularly important for fetal development, Severe nutritional deficiency may increases the development risks.

Conflict at home during pregnancy:

Frequent or severe stress , anxiety, emotional conflict or domestic violence during pregnancy can affect the mother health and may interfere fetal development through the change's in stress hormones (such as cortisol), sleep, nutrition and overall well-being's. However ordinary arguments or occasional stress are not proven to directly cause CHD. Heart defects are usually related to a combination of genetic & environment factors.

Smoking:

Chemical such as nicotine & carbon monoxide can reduce oxygen deliver to the foetus & may interfere with normal CVS development. It is an associated with an increased risks of some congenital heart defects.

Alcohol drinking:

Alcohol can cross the placenta & directly affect the developing embryo. Heavy alcohol exposure, especially in the early pregnancy can interfere with the normal heart development and is associated with CHD.

Hypertension:

Maternal HTN is associated with the increased risk of adverse pregnancy outcome & has been associated with some congenital heart defects. It may affect the placental blood flow & fetal development.

Obesity:

Maternal obesity is associated with higher risks of coronary artery disease (CAD). It may act through metabolic changes inflammation, insulin resistance & nutritional factors.

Increased triglycerides:

Elevated maternal triglycerides are mainly a marker of metabolic disturbance. Abnormal maternal lipid metabolism may be associated with adverse fetal development, but high triglycerides alone not well being established direct cause of CAD.

Barker's Hypothesis

The Barker hypothesis, developed from epidemiological observations in the late twentieth century, proposed that fetal undernutrition and disproportionate fetal growth can programme susceptibility to coronary heart disease later in life. Barker described associations between lower birth weight and later coronary disease and linked fetal growth patterns with subsequent blood pressure, glucose regulation, lipid metabolism and other physiological pathways. The modern Developmental Origins of Health and Disease (DOHaD) framework has broadened this concept to include interactions among genetic susceptibility, the intrauterine environment, placental function, childhood growth, family environment and later-life exposures.[3]

Our proposition goes one step beyond the original fetal-origin formulation: the clinical question is not only what happened in the womb, but how early-life biology and family context can be recognised early enough to change the subsequent trajectory of atherosclerotic risk.

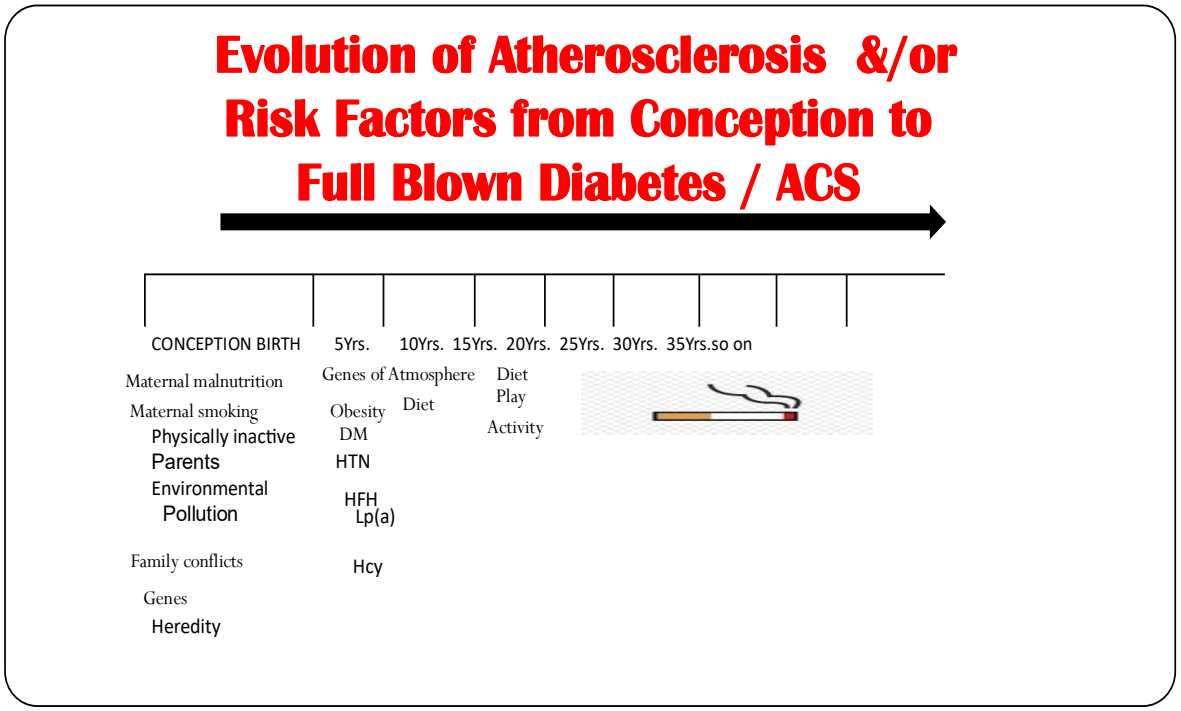

Figure 2: (Evolution of Atherosclerosis)[4]

Our Initial Observations

Our earlier work on familial aggregation of coronary risk factors and coronary artery disease in the young, together with the clinical examples assembled for this update, repeatedly draws attention to clustering of obesity, diabetes, hypertension, dyslipidaemia, tobacco exposure and premature CAD within families. The present compilation extends these observations with 17 illustrative clinical

scenarios collated during period 2012 to 2026 including families with two, three and, in some cases, four generations of cardiometabolic disease.[5,6]

Our Proposition

Atherosclerosis should be viewed as a life-course process rather than an event that begins when an adult develops angina or acute coronary syndrome. Genetic susceptibility, fetal and early-life conditions, family habits, childhood adiposity, tobacco exposure and accumulation of conventional risk factors may interact over decades. Therefore, the family pedigree and the child's or young adult's phenotype can become practical opportunities for primordial and precision prevention.

These observations are hypothesis-generating and clinically useful, but they should not be interpreted as proof that any single maternal, familial or environmental exposure directly causes coronary disease in an individual. Their practical value is in recognising risk earlier and acting on modifiable factors.

Pedigree Study: A Rapid Family Cardiovascular Risk Check

A simple pedigree can reveal patterns that may be missed during a routine consultation. Ask about premature CAD/ACS, stroke, diabetes, hypertension, dyslipidaemia, obesity and tobacco exposure in parents, siblings, children and grandparents. When several generations are affected, consider earlier family-based risk assessment and cascade screening rather than waiting for symptoms.[5,6]

Collating Clinical Evidence (2012–2026)

The following 17 cases are illustrative clinical observations rather than a formal epidemiological cohort. Their purpose is to show how family history, early-life exposures and the evolution of cardiometabolic risk can be connected across the life course—and where prevention can intervene before premature CAD/ACS becomes the presenting event.[3,8,9]

CLINICAL SCENARIOS: FROM EARLY-LIFE EXPOSURES TO PREMATURE ATHEROSCLEROSIS

These scenarios illustrate the life-course concept of cardiovascular risk, linking genetic susceptibility, intrauterine and early-life exposures, accumulation of conventional risk factors, and opportunities for primordial/precision prevention.

No.	Clinical scenario	Key observation	Learning point / prevention opportunity
1.	**Fetal papyraceous / adverse intrauterine event**	Early developmental environment may influence the lifelong cardiovascular and metabolic trajectory.	Optimise maternal health, nutrition, BP and diabetes before and during pregnancy.
2.	**27-year-old man with MI at 24 years**	Severe inherited hypercholesterolaemia with very premature CAD illustrates cumulative LDL exposure and genetic susceptibility.	Early lipid testing, cascade screening and intensive LDL-C lowering are opportunities for prevention.
3.	**Mother with CAD and four sons with CAD and central obesity**	Genes, shared family environment and lifestyle may interact to produce premature CAD.	Family-based risk assessment should begin early.
4.	**Offspring of a mother with poorly controlled diabetes/obesity**	Maternal metabolic health may influence the offspring cardiometabolic phenotype.	Preconception weight optimisation and good glycaemic control are key preventive opportunities.
5.	**Low birth weight followed by rapid catch-up growth**	Illustrates the developmental mismatch / 'thrifty phenotype' concept.[3]	Monitor growth trajectory and prevent excessive catch-up adiposity.
6.	**Child exposed to maternal/parental**	Environmental exposure can adversely influence fetal	Smoking cessation before conception and a smoke-free

No.	Clinical scenario	Key observation	Learning point / prevention opportunity
	tobacco smoke	development and later cardiovascular risk.	home are important.[7]
7.	**Adolescent with obesity, hypertension and dyslipidaemia**	Atherosclerotic risk accumulates long before the first clinical coronary event.	Early identification and treatment of obesity, BP and dyslipidaemia can alter the trajectory.[8]
8.	**Young adult with strong family history of premature CAD**	Silent inherited susceptibility may precede clinical atherosclerosis by decades.	Early lipid/Lp(a) assessment where appropriate and cascade screening can identify high-risk relatives.
9.	**Young adult with worsening cardiovascular health from 20–35 years**	Cardiovascular risk reflects cumulative life-course exposure rather than a single risk factor.	Repeated risk assessment and maintenance of ideal cardiovascular health are central.[9]
10.	**35–40-year-old with premature ACS despite modest conventional risk**	ACS may represent the endpoint of decades of interacting genetic, developmental and lifestyle factors.	This supports a shift from reactive treatment to life-course precision prevention.
11.	**Two brothers, S (38year) and R (34 year), with obesity & diabetes**	Four-generation family history of obesity, diabetes and CAD.	Strong multigenerational clustering; early family screening and primordial prevention.
12.	**S, 13-year-old, with obesity & dyslipidaemia**	Four-generation family history; great-grandmother had diabetes, CAD, CVD and diffuse atherosclerosis.	Cardiometabolic risk evident in childhood; early screening and lifestyle intervention.
13.	**U N, 29-year-old, with morbid obesity & hypertension**	Mother has hypothyroidism/CAD; patient is a heavy smoker.	Familial risk compounded by obesity, hypertension and tobacco; aggressive risk-factor modification.
14.	**S, 32-year-old, with progressive obesity & dyslipidaemia**	Mother has morbid obesity, severe knee OA, CAD and hypothyroidism.	Familial obesity and reduced mobility may reinforce risk; early weight and lipid management.
15.	**D B, 28-year-old, developing obesity & diabetes**	Both parents diabetic; father had ACS and stroke.	Strong familial clustering of diabetes and premature vascular disease; early metabolic prevention.
16.	**D G (45 year), obese with tobacco/alcohol habits; younger brother (42 year) has ideal weight, regular gym, no tobacco**	Contrasting lifestyle profiles; their sons show similar contrasting phenotypes. Bothe parents suffering from hypertension and CAD. Mother also had diabetes and CKD. Father was smoker	Illustrates heredity–lifestyle interaction and the potential for primordial prevention.
17.	**45-year-old man with morbid obesity, diabetes & dyslipidaemia and alopecia totalis. Stressful job**	Both parents have diabetes and hypertension;	Multiple cardiometabolic risks with familial clustering; comprehensive early risk assessment and prevention.

Conception → Placenta/Fetus → Childhood → Adolescence → Young adulthood → Premature CAD/ACS → Precision prevention

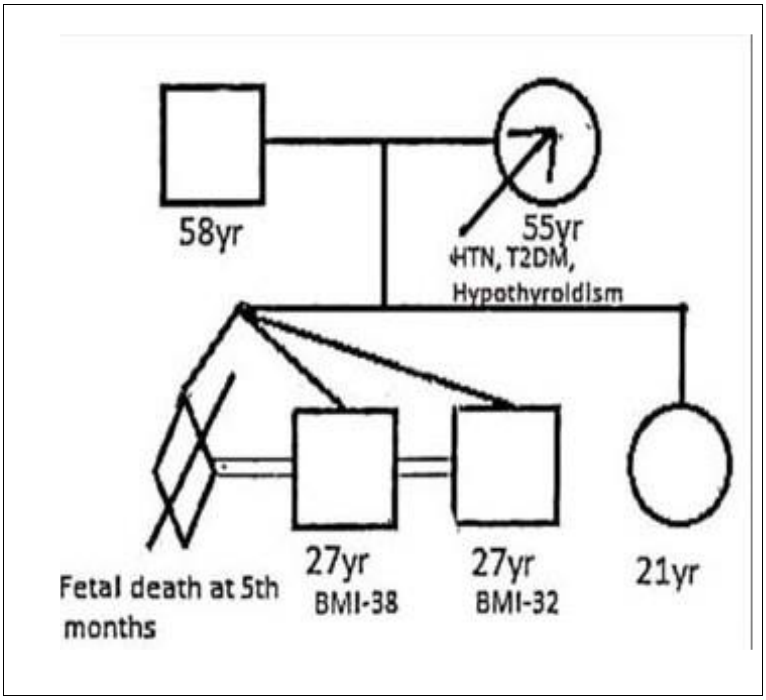

Figure 3: Family pedigree of fetal papyraceous[2]

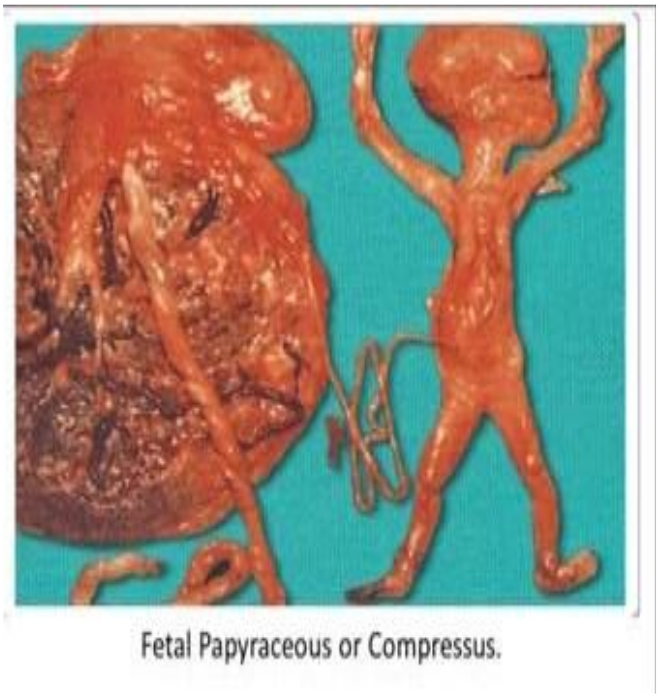

Figure 4: Fetal papyraceous[2]

A 27-year-old man was being investigated for a possible diagnosis of familial hypercholesterolemia (total cholesterol, 480 mg/dL; low-density lipoprotein, 440 mg/dL; high-density lipoprotein, 25 mg/dL; triglycerides, 76 mg/dL on treatment). He had been diagnosed with Acute Inf MI at 24 years of age, and angiography had revealed extensive TVD.

Figure 5: Giant gluteal xanthoma [10]

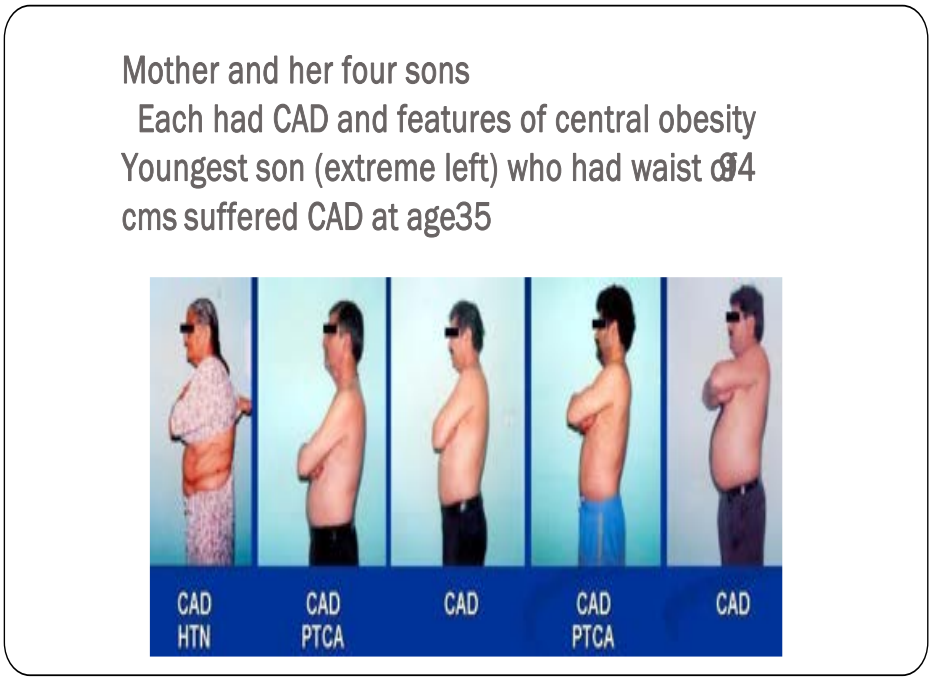

Figure 6: Mother and her four sons [11]

Recommendations: From Quick Pedigree to Precision Prevention

As regards precision prevention with reference to preventive cardiology is an approach to cardiovascular disease prevention that uses an individual's genetic, biological, environmental, behavioural and clinical risk factors to identify their specific risk of developing disease and then delivers tailored preventive interventions. Therefore the practical message of our observations is to move prevention upstream—from treating established CAD to identifying risk in families and children before atherosclerotic disease becomes clinically apparent. These steps constitute: [12]

1. Quick pedigree study

At routine clinical encounters, ask briefly about premature CAD/ACS, stroke, diabetes, hypertension, dyslipidaemia and obesity in parents, siblings and offspring. A striking multigenerational pattern should trigger earlier assessment.

2. Look for obesity, tobacco exposure and cardiovascular disease in accompanying siblings or progeny

When one family member presents with premature CAD, obesity or diabetes, look for obesity, tobacco exposure, diabetes, hypertension, dyslipidaemia or cardiovascular disease in accompanying siblings and progeny. The family may represent shared risk environment as well as inherited susceptibility.

3. If the child is obese, measure BP and evaluate cardiometabolic risk

Measure blood pressure and evaluate cardiometabolic risk, particularly the lipid profile and HbA1c when clinically appropriate. Consider earlier lipid/Lp(a) assessment and family-based evaluation when there is premature CAD or a strong family history.

4. Act before the first event

Promote healthy pregnancy and preconception care, appropriate maternal weight and glycaemic control, avoidance of tobacco exposure, healthy childhood growth, physical activity and a smoke-free home. Prevent excessive catch-up adiposity after low birth weight rather than waiting for adult obesity.

No.	Life's Essential 8	Practical focus
1	Eat better	Healthy overall dietary pattern
2	Be more active	Regular physical activity
3	Quit tobacco / avoid nicotine	Include vaping and second-hand exposure
4	Get healthy sleep	Adults: generally 7–9 hours/night
5	Manage weight	Healthy body weight and trajectory
6	Manage blood lipids	Assess and treat dyslipidaemia appropriately
7	Manage blood sugar	Use glucose/HbA1c as appropriate
8	Manage blood pressure	Detect and control elevated BP

The central message is therefore simple: a pedigree can be a preventive test. When familial clustering is evident, risk-factor assessment can begin in childhood or young adulthood, and prevention can be tailored to the individual's biological, behavioural, environmental and clinical risk profile.

Finally, it is worthwhile to remember the farsighted words of World's First Cardiac Transplant Surgeon Dr Christian Barnard at this juncture:

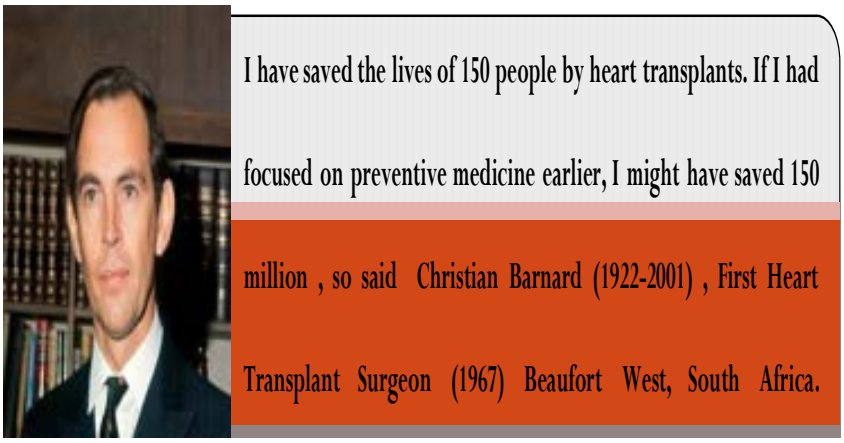

Figure 7: S Dwivedi, Harsh Arora, National Heart Institute

References

1. Dwivedi S. Maa ki kokh shishu ki phli pathshala. In: Heart Aphorisms (हृदय सूक्तियां). Compiled by Dr S Dwivedi. New Delhi: Scientific International Private Limited; 2013.

2. Dwivedi S, Udita. Embryogenesis of the heart and factors affecting premature atherogenesis during pregnancy. Heart News. 2026;65:6-9.

3. Barker DJP. Fetal origins of coronary heart disease. BMJ. 1995;311(6998):171-174.

4. Dwivedi S. Pictorial reflections of coronary atherosclerosis: Indian scenario. In: Proceedings of the 15th International CME on Atherosclerosis, Hypertension and Coronary Artery Disease and 15th Annual Conference of Indian Society of Hypertension; 2005 Nov 12-13; p.164.

5. Dwivedi S, Awasthy N. Familial aggregation of coronary risk factors—an illustrative pedigree. South Asian J Prev Cardiol. 1999;3:108-111.

6. Dwivedi S, Dwivedi G, Chaturvedi A, Sharma S. Coronary artery disease in the young: heredofamilial or faulty lifestyle or both. J Indian Acad Clin Med. 2000;1(3):222-229.

7. Dwivedi S. Smoking initiating coronary artery disease. In: Tobacco Atlas (तम्बाकू चित्रावली). New Delhi: Health and Family Welfare Directorate, Government of Delhi; 2012. p.33.

8. Jacobs DR Jr, Woo JG, Sinaiko AR, Daniels SR, Ikonen J, Juonala M, et al. Childhood cardiovascular risk factors and adult cardiovascular events. N Engl J Med. 2022;386(20):1877-1888.

9. Guo JW, Ning H, Allen NB, Perak AM, Walker JM, Gabriel KP, et al. Cardiovascular health changes in young adults and risk of later-life cardiovascular disease. JAMA Netw Open. 2025;8(10):e2535573.

10. Dwivedi S, Aggarwal A, Sharma V, Dwivedi G. Giant gluteal xanthomas. Circulation. 2010;121(16):1866-1867.

11. Dwivedi S. Diabetes in three generations. In: Diabetes in Pictures. Compiled by Dr S Dwivedi. New Delhi: Scientific International Private Limited; 2013.

12. Lloyd-Jones DM, Allen NB, Anderson CAM, Black T, Brewer LC, Foraker RE, et al. Life's Essential 8: updating and enhancing the American Heart Association's construct of cardiovascular health: a presidential advisory from the American Heart Association. Circulation. 2022;146(5):e18-e43.

R2

Matrix Metalloproteinases and their Role in Cardiovascular Disease

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Introduction

Cardiovascular disease (CVD) remains one of the leading causes of morbidity and mortality worldwide, accounting for more than 30% of global deaths [1]. Cardiovascular disorders include coronary artery disease (CAD), myocardial infarction (MI), stroke, heart failure and hypertension. Although conventional risk factors such as heart failure, myocardial infarction (MI), stroke, coronary artery disease (CAD), and hypertension play an important role in cardiovascular diseases. Inflammation, oxidative stress, and extracellular matrix (ECM) remodelling are significant contributors to cardiovascular disease, despite the well-established conventional risk factors of hypertension, diabetes, dyslipidaemia, smoking, and obesity [2]. Matrix metalloproteinases (MMPs) have attracted a lot of interest among the molecular mediators implicated in these processes. The zinc-dependent endopeptidases known as MMPs control the remodelling and breakdown of extracellular matrix constituents [3]. MMPs are involved in regular ECM turnover, cell migration, tissue formation, and repair under physiological conditions. Tissue inhibitors of metalloproteinases (TIMPs) strictly regulate their activity, maintaining equilibrium between ECM synthesis and degradation [3,4]. A disturbance of this equilibrium may lead to abnormal remodelling of the heart and blood vessels.

MMPs and Cardiovascular Disorders

MMPs are involved in diverse cardiovascular disorders through their effects on **ECM turnover, inflammation and tissue remodeling**. MMPs produced by vascular cells and macrophages in atherosclerosis may affect fibrous-cap integrity and plaque stability in addition to contributing to the ECM breakdown within plaques [5]. MMP-mediated matrix degradation aids in wound healing and tissue repair in myocardial infarction, but excessive activity can cause negative ventricular remodelling and change the shape of the heart [6]. Dysregulated MMP activity in heart failure contributes to structural remodelling and cardiac dysfunction by influencing collagen turnover and myocardial extracellular matrix organization [6].

By altering the extracellular environment, promoting vascular smooth muscle cell migration, and altering the architecture of the vascular wall, MMPs also contribute to vascular remodelling [5]. In hypertension, altered MMP activity may play a role in arterial stiffness and vascular ECM remodelling. Increased proteolytic activity in ischaemic stroke, especially MMP-9, can lead to cerebral tissue damage by promoting ECM degradation and rupture of the blood–brain barrier [7]. Therefore, depending on the particular MMP, cellular source, illness stage, and degree of enzymatic activity, MMPs can have both physiologic and pathogenic roles.

MMP-2, MMP-9 and Their Regulation

MMP-2 and MMP-9, or gelatinases A and B, are two members of the MMP family that are particularly relevant for cardiovascular disease. Both participate in cardiac and vascular remodeling and break down gelatin and other ECM-associated proteins [3]. While MMP-9 is closely linked to inflammatory vascular processes and atherosclerotic plaque remodeling, MMP-2 plays a role in ECM turnover in cardiovascular cells [5,6].
Proteolytic activation is necessary for both enzymes to become functionally active because they are produced as inactive proenzymes (zymogens). Membrane-type MMPs, especially MMP-14, are involved in MMP-2 activation, and TIMP-2 regulates it [3, 4]. MMP activity is further controlled by TIMPs, substrate availability and the local tissue environment, maintaining balanced ECM turnover.

Several growth factors, oxidative stress, and inflammatory cytokines control MMP expression and activity. MMP production can be influenced by TNF-α and IL-1β, and MMP regulation may be further modified by redox-sensitive signalling pathways including transcription factors such AP-1 and NF-κB

[8]. It's important to remember that more MMP gene or protein expression does not always translate into higher enzymatic activity. For this reason, it's important to take into account gene expression, protein abundance, activation state, and functional activity all at once. MMP expression and activity may also be influenced by genetic factors; variations in MMP2 and MMP9 have been linked to cardiovascular disease [8].

Conclusion

Matrix metalloproteinases are important regulators of extracellular matrix remodeling and participate in multiple cardiovascular processes, including **atherosclerosis, vascular remodeling, myocardial infarction and heart failure.** Because of their functions in tissue remodeling, inflammation, and ECM breakdown, MMP-2 and MMP-9 are especially important. But MMPs are more than just enzymes that lead to disease. Their physiological roles in tissue remodeling and repair are crucial, and the balance between MMP activation, TIMP-mediated inhibition, substrate availability, and cellular environment determines how they function. Therefore, a deeper comprehension of the MMP-2/MMP-9–TIMP–ECM network may offer important insights into the mechanisms underlying cardiovascular disease and future therapeutic research.

References

1. Roth GA, Mensah GA, Johnson CO, Addo IY, Ammirati E, Baddour LM, et al. Global burden of cardiovascular diseases and risk factors, 1990–2019: update from the GBD 2019 study. Journal of the American College of Cardiology. 2020;76(25):2982–3021.

2. Frangogiannis NG. The extracellular matrix in myocardial injury, repair, and remodeling. Journal of Clinical Investigation. 2017;127(5):1600–1612.

3. Nagase H, Visse R, Murphy G. Structure and function of matrix metalloproteinases and TIMPs. Cardiovascular Research. 2006;69(3):562–573.

4. Visse R, Nagase H. Matrix metalloproteinases and tissue inhibitors of metalloproteinases: structure, function, and biochemistry. Circulation Research. 2003;92(8):827–839.

5. Newby AC. Metalloproteinases and vulnerable atherosclerotic plaques. Trends in Cardiovascular Medicine. 2007;17(8):253–258.

6. Spinale FG. Myocardial matrix remodeling and the matrix metalloproteinases: influence on cardiac form and function. Physiological Reviews. 2007;87(4):1285–1342.

7. Rosell A, Lo EH. Multiphasic roles for matrix metalloproteinases after stroke. Current Opinion in Pharmacology. 2008;8(1):82–89.

8. Newby AC. Metalloproteinase expression in monocytes and macrophages and the progression of atherosclerosis. Journal of Clinical Investigation. 2008;118(11):350–358.

R3

Novel Nanomedicine Based Investigational Approaches for the Treatment of Atherosclerosis

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Abstract

Atherosclerosis remains the principal pathological substrate of ischemic heart disease and stroke, and although lipid-lowering and antiplatelet regimens have lowered event rates considerably, a substantial residual burden of risk persists. Much of that residual risk is now attributed to shouldering intraplaque inflammation and oxidative injury, which conventional orally administered drugs address inefficiently because they distribute indiscriminately and reach the arterial wall at sub-therapeutic concentrations. Nanomedicine offers a mechanistically distinct solution: engineered carriers in the nanometer range can be steered towards diseased endothelium, triggered to release their payload locally, and used to reprogramme plaque biology rather than merely lower circulating lipids. This mini-review sets out the pharmacological rationale for nanoscale intervention in atherosclerosis, surveys the carrier platforms currently under preclinical and early clinical evaluation, and examines the molecular targets these systems exploit, including inflammatory signaling, defective efferocytosis, oxidative stress and disordered cholesterol handling. Pharmacokinetic behaviors, stimuli-responsive and biomimetic design, and the translational obstacles that still separate encouraging rodent data from clinical practice are also discussed.

Keywords: atherosclerosis; nanomedicine; targeted drug delivery; plaque inflammation; biomimetic nanoparticles; efferocytosis; nanozymes

1. Introduction

The medical application of nanotechnology has expanded rapidly over the past three decades, supported by regulatory approval of several nanoscale therapeutics and by sustained public investment in nanomedical research programmed worldwide. Although the discipline matured largely within oncology, it became apparent that the same engineering principles could be also applied in atherosclerotic disease, where nanoparticle-based targeting concepts now underpin both diagnosis and treatment. Carriers assembled from lipids, polymers, metals or biological membranes can shield labile payloads, extend circulation, and deposit cargo within the lesion at concentrations unattainable with free drug, while simultaneously accommodating imaging agents for theragnostic use [1]. Atherosclerosis itself arises from the accumulation of plaque along arterial walls, progressively narrowing the lumen until coronary occlusion produces myocardial infarction or cerebral occlusion produces stroke. Oral pharmacotherapy remains the clinical mainstay of management, yet its development has been constrained by adverse effects, low bioavailability and other unfavorable pharmaceutical properties. Recent years have seen statins and naturally derived compounds reformulated within nanoscale preparations specifically in order to raise their bioavailability. Liposomes, nanoparticles, nano emulsions and nano capsules have all been proposed as oral platforms suited to this purpose [2]. This review article consolidates progress across these strands, with emphasis on the pharmacological logic that connects carrier system to treat atherosclerotic plaque.

2. Atherosclerosis as a Therapeutic

Target Atherosclerosis is a multifactorial cardiovascular disorder defined by lipid accumulation, chronic inflammation, oxidative stress and endothelial dysfunction [3]. Its pathogenesis includes oxidative damage, lipid infiltration, inflammatory activation, and endothelial damage. Sirtuin signaling has emerged as a key regulator of plaque formation, development, and susceptibility [2]. As lesions mature, smooth muscle cells migrating from the media or proliferating locally synthesize collagen, elastin and proteoglycans, producing intimal thickening that stabilizes the plaque in its earlier phases.

That stability is subsequently eroded by matrix metalloproteinases secreted by plaque macrophages, which degrade the collagen fibers sustaining fibrous cap integrity and render the lesion vulnerable to rupture [3]. Understanding this disease cycle in full is regarded as a prerequisite for improved diagnosis, better care and more rational treatment [4].

Conventional pharmacological management has achieved considerable success but carries recognized adverse effects on the body [4]. Lipid-lowering and anti-inflammatory agents in particular deliver only limited efficacy, because they target lesions inadequately, exhibit low bioavailability and generate systemic side effects [3]. A highly effective and simultaneously safe treatment for atherosclerosis therefore remains an unresolved clinical challenge [4]. These deficiencies define the therapeutic space that nanomedicine seeks to occupy: not the replacement of lipid control, but the inclusion of an antioxidant and anti-inflammatory treatment that is specifically targeted at the plaque.

3. Why Nanomedicine in Atherosclerosis?

The central attraction of nanomedicine in this setting is that it permits therapeutics to be directed towards the diseased vessel rather than distributed uniformly through the circulation. Targeting strategies described for atherosclerosis is deliberate targeting of the vasculature, and targeting of individual plaque constituents. Nanoparticle-facilitated therapy may thus be aimed at the liver to modify systemic lipid handling, or directed locally to the lesion [1]. The pharmacological advantage this confers is illustrated by statin delivery. Statins possess potent anti-inflammatory activity that cannot be fully realized through oral dosing because systemic bioavailability is low. An injectable reconstituted high-density lipoprotein (rHDL) carrier was therefore developed to convey statin directly to atherosclerotic plaques, with the anti-inflammatory effect shown in vitro to operate through inhibition of the mevalonate pathway. In apolipoprotein E-knockout mice the particles accumulated within lesions and acted directly upon plaque macrophages. A three-month low-dose schedule of statin-rHDL arrested the progression of plaque inflammation, whereas a one-week high-dose schedule of statin-rHDL produced a marked reduction of inflammation in advanced lesions [5]. A parallel rationale applies to naturally derived agents: nanoscale delivery of nutraceuticals to atherosclerotic sites is attractive precisely because it promises high therapeutic efficiency alongside fewer side effects than conventional drugs [4].

4. Nanomedicine Platforms for Drug Delivery

Nanoscale drug delivery systems encompass a broad family of sub particulate carriers, among them nanoparticles, nanoliposomes, solid lipid nanoparticles, nano micelles, nano emulsions, nano capsules and nanogels. Collectively these systems can encapsulate insoluble, fat-soluble, protein, nucleic acid, labile or otherwise inactivated drugs. Nano formulation improves the solubility of conventional agents, prolongs systemic circulation and reduces off-target cytotoxicity, thereby lowering the dose required. It also enhances bio membrane permeability, alters biodistribution and increases bioavailability while improving the stability of the loaded drug. Nanoliposomes are nanoscale vesicles built from lipid bilayers, whereas nano emulsions disperse lipids, proteins or other constituents within water or an alternative medium at nanoscale droplet size [2].

Biologically derived carriers occupy a distinct niche. Reconstituted high-density lipoprotein exemplifies the use of an endogenous particle as an injectable vehicle for plaque-directed therapy [5]. Micro- and nanobubbles form a further category, developed as vehicles for detection, investigation and drug delivery aimed specifically at atherosclerotic sites. These constructs have already been commercialized clinically as contrast agents, since they respond to ultrasound in a manner that guides and stimulates imaging. Their assembly draws on defined substrates including proteins, lipids and polymers, chosen to tune particle characteristics and to support rational theragnostic design. Ancillary surface modification allows bubbles to be united with antibodies, inflammatory markers or genes so that cargo is delivered more selectively to the oxidized lipid-rich region and released on demand at the lesion [6]. Inorganic platforms complete the picture, with Prussian blue, cerium oxide, selenium-based and multifunctional composite nanozymes representing the principal families under investigation. These catalytic systems differ from conventional carriers in that their design principles, catalytic mechanisms and lesion-targeting strategies are interdependent, the material serving as both vehicle and active agent [3]. Platform selection is therefore governed less by any intrinsic superiority than by the intended payload, the cellular target and the administration route, and the sections that follow are organized around the biological problem each platform is recruited to solve.

5. Molecular Targets and Pharmacological Approaches

5.1 Anti-inflammatory and cytokine delivery

The interest in cytokine-directed nano therapy has remained due to the fact that approved regimens focus almost entirely on lowering the levels of lipids in the blood, with little effect on the underlying inflammation. Targeted delivery to plaque has been achieved by entrapping the multipotent anti-inflammatory cytokine interleukin-10 in cRGD-conjugated Pluronic-based nanocarriers. The carriers exhibited sustained release in mild conditions and high protein loading. The in vitro efficacy was assessed in lipopolysaccharide (LPS) stimulated macrophages and in vivo in apolipoprotein E-knockout C57BL/6 mice. Intravenous administration of the encapsulated cytokine led to a significantly improved pharmacokinetic profile and to a significantly higher accumulation in atherosclerotic lesions compared to free interleukin-10. The size of the plaque was significantly reduced, lesional production of the cytokine IL-1β was blocked and the cytokine retained bioactivity [7].

5.2 Pharmacological resolution of inflammation

Complementary approach seeks to speed up the elimination of inflammation instead of minimizing it. The peptide mimic of annexin A1, Ac2-26, has been encapsulated in sub-100 nm particles of biodegradable poly (lactic-co-glycolic acid)-b-polyethylene glycol and collagen IV-targeted analogs. These particles significantly better than the original peptide in inhibiting recruitment of neutrophils in a self-limited zymosan peritonitis model and decreased the resolution interval by up to 4 hours. Their potential for use in other chronic inflammatory diseases has been suggested given that treatment of this collagen IV-targeted formulation reduced tissue damage in hind-limb ischemia-reperfusion injury (HIRI) [8] when administered systemically. A possibility was then realized when it was shown that targeted Ac2-26 nanoparticles could prevent advanced atherosclerosis in hypercholesterolemic mice [9].

5.3 Restoring efferocytosis

Apoptotic cells are one of the criteria of the mature plaque. Although prophagocytic antibody therapies have been investigated to increase their clearance, they cause toxicities such anemia and off-target elimination of healthy tissue. In order to get around this, single-walled carbon nanotubes coated with a pharmacological inhibitor of the antiphagocytic CD47–SIRPα axis were used to create a macrophage-specific nano therapy. In apolipoprotein E-deficient animals prone to atherosclerosis, the nanotubes aggregated with the plaque, stimulated lesional phagocytosis and reduced plaque burden without sacrificing safety, thus providing a critical translational hurdle for this therapeutic class [10].

5.4 Lipid mediators and endothelial targeting

Atherosclerosis is a non-resolving inflammatory condition, and a recent clinical trial of an anti-inflammatory drug showed that it reduced cardiovascular mortality but also increased the likelihood of infection, suggesting that tissue-targeted instead of systemic immunomodulation is the most appropriate treatment for atherosclerosis. Algal oil contains docosahexaenoic acid (DHA), an omega-3 fatty acid with interesting properties as a precursor for the generation of specialized pro-resolving lipid mediators and is therefore an attractive payload. Lipid-core nano capsules were surface-functionalized with anti-PECAM-1 for targeting to inflamed endothelium and the conjugation efficiency was 94.8%. The slight increase in diameter from ca. 160–162 nm to 164 nm after functionalization was observed, and is not considered to represent aggregation. The functionalized nano capsules were found to not affect the viability of human umbilical vein endothelial cells and were determined to be appropriate for further pharmaceutical development [11].

5.5 Foam cell formation and nutraceutical payload

In vivo, sugar-based amphiphilic nanoparticles have been shown to inhibit the development of atherosclerosis by blocking lipid uptake by the lesional macrophages [12]. Similar is the use of nutraceuticals, which have become more and more popular in atherosclerosis treatment because of their low toxicity and low side effects, the data base for their clinical usefulness still needs to be reinforced [4].

6. Targeted and Stimuli Responsive Drug Delivery

The treatment of atherosclerosis can be done non-specifically via the vasculature or against individual, separate plaque constituents, either by synthetic particles or natural biological conduits [1]. The majority of publications on active targeting are based on approaches that are directed by the ligand, such as cRGD-peptides that bind to integrin receptors on activated lesional cells [7], collagen IV-targeted polymers that bind to exposed subendothelial matrix [8] and anti-PECAM-1 functionalization that targets the adhesion molecule expressed at the inflamed endothelial surface [11].

Biomimetic camouflage has become a very strong rival of synthetic ligands. By functionalizing the nanoparticles with platelet membrane, they can simultaneously target and detect atherosclerosis by mimicking several adhesive mechanisms used by platelets endogenously, in one step [13]. The membranes of platelets make an efficient targeting shell because they have intrinsic affinity for plaque, and because they are already present at atherosclerotic sites, without instructions. The same concept was used to optimize delivery of rapamycin, which is a potent anti-atherosclerotic agent with a limited clinical use due to low lesional concentration and high systemic toxicity. To demonstrate the effective homing in vivo an atherosclerotic arterial tree was used to evaluate platelet membrane-coated nanoparticles, which exhibited 4.98 times higher radiant efficiency than control particles. The rapamycin loaded particles were found to be very effective in slowing disease progression and stabilize existing plaque in apolipoprotein E-deficient mice [14].

The temporal control is added by stimuli-responsive design to this spatial precision. Cargo can be liberated on demand at the lesion site by ultrasound that both guides the imaging and triggers the release of the payloads of micro- and nanobubbles [6]. Chemical triggers can also be used, like the pH-induced dissociation of a phosphatase-inhibitor from a macrophage-specific carrier within the local acidic plaque microenvironment [10].

7. Pharmacokinetic and pharmacodynamic considerations

The pharmacokinetic case for nano formulation is straightforward: encapsulation increases solubility, prolongs systemic circulation, reduces off-target cytotoxicity and thereby lowers the dose required, while improved bio membrane permeability alters biodistribution and raises bioavailability. These carriers also stabilize the drug against premature degradation [2]. The magnitude of the effect is best appreciated by direct comparison: intravenously administered nanocarrier-encapsulated interleukin-10 achieved a vastly improved pharmacokinetic profile and profoundly greater lesional accumulation than the free cytokine. Controlled release contributes independently, since the same carriers combined high loading efficiency with sustained release characteristics [7].

It has been observed that statin-loaded reconstituted high-density lipoprotein had an anti-inflammatory action by blocking the mevalonate pathway and that this pathway is directly targeted within plaque macrophages, not within the liver [5]. An additional effect was dose sparing, where the dosages of pro-resolving nanoparticles was significantly more effective than the equivalent free peptide and remained effective at nanogram amounts per animal [8]. Length of exposure is also relevant as a three-month low dose regimen of nano therapy had a different but complementary effect compared to a one-week high dose regimen of the same nano therapy, resulting in different effects on early and advanced lesions, respectively [5]. Finally, biomimetic surfaces can also affect targeting as well as clearance since coating with an intact cell membrane preserves the inherent biological elements of the parent cell in circulation [15].

8. Recent Advances and Emerging Therapeutic Approaches

Combining intrinsic pharmacological action with targeting in biomimetic reactors is one of the most active current methods. By coating a platelet membrane around a core of selenium and ginsenoside Rb1 nanoparticles, a platelet membrane-cloaked system was developed to solve the dual problems of lesional targeting and plaque stabilization. While the membrane coating preserved the platelet's natural biological components to carry the medication into plaque, the core provided antioxidant, lipid-regulating, and anti-inflammatory activities. The construct showed significantly improved plaque-targeting ability and greater suppression of ICAM-1 compared to comparators [15].

A second horizon is shown by catalytic nanomaterials. Because they may simultaneously control oxidative stress, inflammation, and lipid metabolism, nanozymes—engineered nanomaterials with inherent enzyme-mimetic catalytic activity—have become a transformative tactic. Hydrophobic medications are solubilized using nanozyme delivery systems, which also increase drug half-life, enhance lesion-specific targeting, and reduce systemic toxicity. Hyaluronic acid-functionalized cerium oxide nanoparticles reduced aortic plaque area by about 65%, decreased intraplaque inflammatory cell infiltration by 40%, and decreased serum LDL cholesterol by 25% after eleven weeks of repeated systemic administration. These preclinical results are encouraging. Crucially, there was no apparent hepatic or renal damage based on normal histology and unchanged liver and kidney function indicators [3]. Beyond therapy, micro- and nanobubbles have been developed into multimodal theragnostic agents capable of both diagnosing and remodeling atherosclerosis [6], while the oral route continues to advance through nano preparations combining statins with natural product drugs [2].

9. Limitations and translational challenges

Clinical translation is yet hampered by a number of issues, most notably pharmacokinetic limitations, biosafety issues, and the lack of manufacturing standardization [3]. In the instance of docosahexaenoic acid nano capsules, toxicity was specifically assessed prior to considering in vivo application [11]. Toxicological characterization is not optional. Genetic hazards, low recovery rates, restricted therapeutic dosage, dubious durability, and challenges preserving cell membrane integrity are only a few of the specific drawbacks of biomimetic systems [4].

In addition, the evidence basis is not as solid as it seems. Few studies maintain treatment for twelve weeks or longer, and while they report reduced necrotic core area and improved collagen content as surrogate markers of plaque stability, they rarely look at recurrent disease risk or hard cardiovascular endpoints like myocardial infarction, stroke, or mortality [3]. Because human atherosclerosis develops over decades against a context of comorbidity and polypharmacy, efficacy shown in genetically engineered rodents cannot be considered to foretell benefit.

Two further considerations deserve emphasis. First, the safety advantage that motivates targeted design must itself be demonstrated rather than presumed, as was done when macrophage-selective inhibition of the CD47–SIRPα axis was shown to reduce plaque burden without compromising safety, in contrast to systemic antibody blockade [10]. Second, the durability of intravenous nano therapy in a chronic disease requiring lifelong management is unresolved, which explains the continued interest in oral nano formulations despite the formidable gastrointestinal barriers such routes must overcome [2].

10. Future Perspectives

New materials won't be as important to progress as thorough integration with existing care. The most promising paths for the area are seen to include polarizing pro-inflammatory M1 macrophages toward an anti-inflammatory M2 phenotype, improving nutraceutical nano delivery, and combining these strategies with proven lipid-lowering medications [4]. Platelet membrane-cloaked selenium and ginsenoside Rb1 nanoparticles have already demonstrated potential synergy with the anticoagulant warfarin [15]. Future research on manufacturing should focus on enhanced biocompatibility, optimized therapeutic specificity, and scalable, repeatable fabrication techniques [3]. As the area develops, it is anticipated that more clinical benefit will result from the ongoing development of nanoparticle targeting approaches and the theragnostic structures that enable the integration of therapy with imaging [1].

11. Conclusion

Based on the knowledge that the problem with current therapy is not the lack of active molecules but rather the inability to distribute them where they are needed, nanomedicine has evolved from a peripheral curiosity to a genuine pharmacological strategy in atherosclerosis [1]. Cytokines, pro-resolving peptides, phosphatase inhibitors, omega-3 fatty acids, immunosuppressants, and catalytic antioxidants have all been delivered into the plaque itself using carrier platforms, which at present include lipid, polymeric, inorganic, gaseous, and cell-derived architectures [3]. In comparison to free medication, the preclinical record consistently shows better pharmacokinetics, increased lesional accumulation, and decreased systemic toxicity [7]. What remains outstanding is rigorous long-term evaluation against clinically meaningful endpoints, together with the biosafety, efficacy and

manufacturing standardization required for regulatory approval [3]. Addressing these questions, rather than proliferating further carrier designs, is the work that will determine whether nanotherapeutics enter routine cardiovascular practice. The nanomedicine specially the nano capsules have shown promising results for the upcoming future in the treatment of atherosclerosis by reducing the atherosclerotic plaque [11]. In the next decade or so micro and nano bubbles have casted a reliable shadow for the treatment of atherosclerosis [6].

References

1. Lobatto ME, Fuster V, Fayad ZA, Mulder WJ. Perspectives and opportunities for nanomedicine in the management of atherosclerosis. Nature Reviews Drug Discovery. 2011 Nov;10(11):835-52.

2. Sun X, Jia X, Tan Z, Fan D, Chen M, Cui N, Liu A, Liu D. Oral nanoformulations in cardiovascular medicine: advances in atherosclerosis treatment. Pharmaceuticals. 2024 Jul 10;17(7):919.

3. Zou Z, Bi X, Ma H, Chen L, Jin H, Gong S, Li X, Liu X. Recent advances in nanozymes for the treatment of atherosclerosis. International Journal of Nanomedicine. 2025 Dec 31:9447-72.

4. Pillai SC, Borah A, Jacob EM, Kumar DS. Nanotechnological approach to delivering nutraceuticals as promising drug candidates for the treatment of atherosclerosis. Drug Delivery. 2021 Jan 1;28(1):550-68.

5. Duivenvoorden R, Tang J, Cormode DP, Mieszawska AJ, Izquierdo-Garcia D, Ozcan C, Otten MJ, Zaidi N, Lobatto ME, Van Rijs SM, Priem B. A statin-loaded reconstituted high-density lipoprotein nanoparticle inhibits atherosclerotic plaque inflammation. Nature communications. 2014 Jan 20;5(1):3065.

6. Wijaya A, Maruf A, Wu W, Wang G. Recent advances in micro-and nano-bubbles for atherosclerosis applications. Biomaterials science. 2020 Sep 15;8(18):4920-39.

7. Kim M, Sahu A, Hwang Y, Kim GB, Nam GH, Kim IS, Kwon IC, Tae G. Targeted delivery of anti-inflammatory cytokine by nanocarrier reduces atherosclerosis in Apo E−/-mice. Biomaterials. 2020 Jan 1;226:119550.

8. Kamaly N, Fredman G, Subramanian M, Gadde S, Pesic A, Cheung L, Fayad ZA, Langer R, Tabas I, Cameron Farokhzad O. Development and in vivo efficacy of targeted polymeric inflammation-resolving nanoparticles. Proceedings of the National Academy of Sciences. 2013 Apr 16;110(16):6506-11.

9. Fredman G, Kamaly N, Spolitu S, Milton J, Ghorpade D, Chiasson R, Kuriakose G, Perretti M, Farokhzad O, Tabas I. Targeted nanoparticles containing the proresolving peptide Ac2-26 protect against advanced atherosclerosis in hypercholesterolemic mice. Science translational medicine. 2015 Feb 18;7(275):275ra20-.

10. Flores AM, Hosseini-Nassab N, Jarr KU, Ye J, Zhu X, Wirka R, Koh AL, Tsantilas P, Wang Y, Nanda V, Kojima Y. Pro-efferocytic nanoparticles are specifically taken up by lesional macrophages and prevent atherosclerosis. Nature nanotechnology. 2020 Feb;15(2):154-61.

11. de Castro Leão M, Pohlmann AR, Alves AD, Farsky SH, Uchiyama MK, Araki K, Sandri S, Guterres SS, Castro IA. Docosahexaenoic acid nanoencapsulated with anti-PECAM-1 as co-therapy for atherosclerosis regression. European Journal of Pharmaceutics and Biopharmaceutics. 2021 Feb 1;159:99-107.

12. Lewis DR, Petersen LK, York AW, Zablocki KR, Joseph LB, Kholodovych V, Prud'homme RK, Uhrich KE, Moghe PV. Sugar-based amphiphilic nanoparticles arrest atherosclerosis in vivo. Proceedings of the National Academy of Sciences. 2015 Mar 3;112(9):2693-8.

13. Wei X, Ying M, Dehaini D, Su Y, Kroll AV, Zhou J, Gao W, Fang RH, Chien S, Zhang L. Nanoparticle functionalization with platelet membrane enables multifactored biological targeting and detection of atherosclerosis. ACS nano. 2018 Jan 23;12(1):109-16.

14. Song Y, Huang Z, Liu X, Pang Z, Chen J, Yang H, Zhang N, Cao Z, Liu M, Cao J, Li C. Platelet membrane-coated nanoparticle-mediated targeting delivery of Rapamycin blocks atherosclerotic plaque development and stabilizes plaque in apolipoprotein E-deficient (ApoE−/−) mice. Nanomedicine: Nanotechnology, Biology and Medicine. 2019 Jan 1;15(1):13-24.

15. Yin M, Lin J, Yang M, Li C, Wu P, Zou J, Jiang Y, Shao J. Platelet membrane-cloaked selenium/ginsenoside Rb1 nanosystem as biomimetic reactor for atherosclerosis therapy. Colloids and Surfaces B: Biointerfaces. 2022 Jun 1;214:112464.

R4

Beyond LDL-C: Can Biomarkers Identify the Vulnerable Atherosclerotic Plaque? Emerging roles of myeloperoxidase, matrix metalloproteinase-9, pentraxin-3, GDF-15 and soluble ST2

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Introduction

Atherosclerosis is now more and more seen not just as a condition involving the accumulation of lipids but as a chronic inflammatory disease. Throughout the progression of the disease, endothelial dysfunction, the retention and oxidation of lipids, activation of immune cells, oxidative stress, remodelling of the extracellular matrix and thrombosis all interact with one another. Even though traditional risk factors and lipid values such as LDL-cholesterol are still at the heart of cardiovascular risk assessment, they fail to fully account for why in some people an acute myocardial infarction or an ischemic stroke occurs even though their atherosclerotic burden appears to be only modest.

This has led to increasing interest in the concept of the vulnerable plaque—an atherosclerotic lesion with biological characteristics that increase its propensity for rupture, erosion or thrombosis. The concept has also evolved beyond the classical thin-cap fibroatheroma, with plaque erosion, fissuring and calcified nodules contributing to acute cardiovascular events. Consequently, contemporary research increasingly focuses not only on identifying the vulnerable plaque but also on identifying the "vulnerable patient." [1,2]

Circulating biomarkers may provide a relatively accessible approach to this problem. Among emerging candidates, myeloperoxidase (MPO), matrix metalloproteinase-9 (MMP-9), pentraxin-3 (PTX3), growth differentiation factor-15 (GDF-15), and soluble suppression of tumorigenicity-2 (sST2) are particularly interesting because they reflect different biological processes involved in plaque inflammation, remodelling and destabilization.

From plaque burden to plaque biology

Traditional lipid measurements provide important information about systemic atherogenic risk, but plaque complications depend on more than circulating lipid concentrations. Inflammation, oxidative stress, macrophage activation, extracellular-matrix degradation, necrotic-core formation and fibrous-cap integrity all contribute to plaque behaviour.

Thus, the degree of luminal stenosis does not necessarily correspond to the biological activity of a plaque. A lesion with relatively limited stenosis may possess a lipid-rich necrotic core and active inflammation, whereas a heavily calcified lesion may be comparatively stable. This distinction has driven interest in biomarkers that reflect plaque biology rather than plaque burden alone. [1,2]

The emerging biomarker field therefore seeks to answer a different question:

Can a blood biomarker identify active or potentially unstable atherosclerotic disease before an acute event occurs?

Myeloperoxidase: a marker of oxidative and inflammatory activity

Myeloperoxidase is a heme-containing enzyme predominantly released by activated neutrophils and monocytes. It generates reactive oxidant species and contributes to oxidative modification of lipids and proteins, endothelial dysfunction and vascular inflammation.

Within atherosclerotic lesions, MPO has been implicated in several processes associated with plaque progression and instability. Circulating MPO concentrations have also been associated with adverse cardiovascular outcomes across different clinical populations. [3]

However, biological plausibility does not automatically translate into routine clinical utility. The 2025 American College of Cardiology Scientific Statement notes that several inflammatory biomarkers, including MPO, have demonstrated predictive value but generally have not proven superior to hsCRP and are often not widely available in standardized commercial formats. [4]

Thus, MPO may ultimately have greater value as part of a multimarker assessment of residual inflammatory and oxidative risk rather than as a stand-alone diagnostic test.

Matrix metalloproteinase-9: linking inflammation to fibrous-cap disruption

Matrix metalloproteinase-9 is an extracellular-matrix-degrading enzyme produced by inflammatory cells and other vascular cells. Increased proteolytic activity within an atherosclerotic plaque can weaken the fibrous cap and contribute to plaque disruption.

This makes MMP-9 particularly interesting because it provides a mechanistic link between inflammation and structural plaque instability. Experimental and clinical studies have demonstrated increased MMP activity, including MMP-9, in vulnerable plaques, supporting a potential role for extracellular matrix degradation in fibrous-cap weakening and plaque rupture. [5]

Clinical investigations have also demonstrated associations between circulating MMP-9 concentrations and carotid atherosclerotic burden, including plaque extent, plaque instability, and intima-media thickness.[6] Nevertheless, MMP-9 is not specific to atherosclerosis and may be influenced by multiple inflammatory and tissue-remodelling conditions. Its future role may therefore lie in combination with other biomarkers or imaging rather than isolated measurement.

Pentraxin-3: a window into local vascular inflammation?

Pentraxin-3 belongs to the long pentraxin family. PTX3 can be produced locally by endothelial cells, macrophages, vascular smooth-muscle cells and other cells involved in vascular inflammation, unlike CRP, which is predominantly synthesized in the liver,

Earlier studies demonstrated increased PTX3 expression within advanced atherosclerotic plaques, with PTX3 produced principally by macrophages and endothelial cells, supporting its potential association with local plaque inflammation. [7]

More recent investigations have continued to explore associations between circulating PTX3 levels and imaging characteristics of carotid plaque vulnerability, including fibrous-cap discontinuity and focal inflammation.[8] However, PTX3 biology is complex, and an association with advanced or vulnerable plaque does not by itself establish PTX3 as a specific diagnostic marker of plaque rupture.

Further prospective validation, assay harmonization and clarification of biological variability are needed before PTX3 can be considered for routine cardiovascular risk assessment.

GDF-15: a marker of cellular stress

Growth differentiation factor-15 is a member of the transforming growth factor-β superfamily and is expressed in response to cellular stress, inflammation and tissue injury.

GDF-15 has attracted considerable attention because of its involvement in inflammatory and cellular stress responses. Experimental studies have demonstrated that GDF-15 regulates leukocyte recruitment after myocardial infarction, suggesting a role in the broader biological response to cardiovascular injury. [9]

However, its lack of vascular specificity remains an important limitation. GDF-15 is also influenced by age, renal dysfunction, malignancy and other systemic conditions. Therefore, its potential role may be more relevant to risk refinement and prognostication than to identification of atherosclerosis per se.

Soluble ST2: inflammation, remodelling and cardiovascular stress

ST2 is a member of the interleukin-1 receptor family. The soluble form, sST2, can interfere with IL-33/ST2 signalling and has been studied extensively in cardiovascular disease, particularly heart failure.

Evidence has implicated the IL-33/ST2 pathway in cardiovascular inflammation and atherosclerosis, with studies suggesting a potentially protective role for IL-33/ST2 signaling in atherogenesis. [10] The attraction of sST2 lies in its potential ability to capture inflammatory and tissue-remodelling processes that are not reflected by conventional lipid measurements.

However, its strongest established clinical application remains in cardiovascular stress and heart failure rather than direct identification of vulnerable atherosclerotic plaque. This distinction highlights an important principle: a biomarker can be biologically relevant to atherosclerosis without necessarily being sufficiently specific for routine plaque-risk assessment.

What do these biomarkers actually tell us?

Biomarker	Major biological process	Potential relevance	Major limitation
MPO	Oxidative stress and inflammation	Oxidative modification, endothelial dysfunction and inflammatory activity	Limited specificity; not routinely standardized
MMP-9	Extracellular-matrix degradation	Fibrous-cap weakening and plaque remodelling	Influenced by several inflammatory conditions
PTX3	Local vascular inflammation	Association with inflammatory/vulnerable plaque phenotype	Complex biology and limited standardization
GDF-15	Cellular stress and inflammation	Risk refinement and adverse cardiovascular outcomes	Low disease specificity; affected by age and renal function
sST2	Inflammatory signalling and tissue remodelling	Potential marker of vascular stress	Greater established clinical role in heart failure

Are these biomarkers ready for the clinical laboratory?

Despite promising biological and observational evidence, these emerging biomarkers cannot currently replace established cardiovascular risk assessment. hsCRP remains the inflammatory biomarker with the strongest clinical evidence and widespread standardized use. The 2025 ACC Scientific Statement emphasizes that alternative inflammatory biomarkers—including MPO—have predictive value but generally add limited information beyond hsCRP in routine practice. [4]

The major barriers include analytical standardization, biological variability, reference intervals, assay harmonization, cost, disease specificity and, most importantly, demonstration that biomarker-guided management improves clinical outcomes.

This distinction between association and clinical utility is crucial. A biomarker may correlate strongly with plaque vulnerability without necessarily improving patient management when added to LDL-C, ApoB, blood pressure, diabetes status, smoking history, established risk scores and imaging.

The future: multimarker panels rather than a single “magic” biomarker

Atherosclerosis is biologically heterogeneous. It is therefore unlikely that one circulating molecule will capture every process involved in plaque progression and destabilization.

A more promising approach may be a multimarker strategy, integrating complementary biological domains:

Atherogenic burden + inflammation + oxidative stress + matrix degradation + cellular stress + imaging

Such an approach could potentially combine conventional lipid markers with inflammatory and plaque-specific biomarkers and non-invasive imaging. Recent reviews of mechanism-based biomarkers similarly emphasize the potential value of integrating circulating biomarkers with imaging rather than viewing any single marker as a replacement for established assessment methods. [2]

Conclusion

The next generation of atherosclerosis biomarkers is moving beyond the traditional question of "How much cholesterol does the patient have?" towards a more biologically informative question: "How active and potentially unstable is the atherosclerotic disease?"

MPO, MMP-9, PTX3, GDF-15 and sST2 represent different components of plaque biology, including oxidative stress, inflammation, extracellular-matrix degradation and cellular stress. Their biological plausibility and growing clinical evidence make them attractive candidates for future cardiovascular risk stratification. [2–10]

However, the transition from a promising research biomarker to a routine clinical laboratory test requires rigorous analytical validation, assay standardization, prospective clinical studies and evidence that measurement changes clinical management and outcomes.

For now, these biomarkers should therefore be regarded as promising pieces of the atherosclerosis puzzle rather than replacements for established risk assessment.

The future may belong not to a single "vulnerable plaque biomarker," but to an integrated biochemical and imaging profile of the vulnerable patient.

References

1. Acoba D, Gonçalves I. Can markers predict plaque vulnerability? *Atherosclerosis.* 2026;417:120744.DOI: 10.1016/j.atherosclerosis.2026.120744

2. Choudhary D, Bali S, Bandral SS, et al. Mechanism-based biomarkers for early detection and plaque vulnerability assessment in atherosclerosis. *Biochem Biophys Res Commun.* 2026;833:154408.DOI: 10.1016/j.bbrc.2026.154408

3. Schindhelm RK, van der Zwan LP, Teerlink T, Scheffer PG. Myeloperoxidase: a useful biomarker for cardiovascular disease risk stratification? Clin Chem. 2009;55(8):1462-1470. doi:10.1373/clinchem.2009.126029.

4. **Mensah GA, Arnold N, Prabhu SD, Ridker PM, Welty FK. Inflammation and cardiovascular disease: 2025 ACC scientific statement: a report of the American College of Cardiology. J Am Coll Cardiol. 2026;87(11):1381-1404. doi:10.1016/j.jacc.2025.08.047.**

5. **Newby AC. Metalloproteinases and vulnerable atherosclerotic plaques. Trends Cardiovasc Med. 2007;17(8):253-258. doi:10.1016/j.tcm.2007.09.001.**

6. Tan C, Liu Y, Li W, Deng F, Liu X, Wang X, Gui Y, Qin L, Hu C, Chen L. Associations of matrix metalloproteinase-9 and monocyte chemoattractant protein-1 concentrations with carotid atherosclerosis, based on measurements of plaque and intima-media thickness. *Atherosclerosis.* 2014;232(1):199-203. doi:10.1016/j.atherosclerosis.2013.11.040.

7. Rolph MS, Zimmer S, Bottazzi B, Garlanda C, Mantovani A, Hansson GK. Production of the long pentraxin PTX3 in advanced atherosclerotic plaques. Arterioscler Thromb Vasc Biol. 2002;22(5):e10-e14. doi:10.1161/01.ATV.0000015595.95497.2F.

8. Li T, Li L, Wang Q, Sun Y, Lian J, Yu W. Serum PTX3 Levels Correlate with Fibrous Cap Discontinuity and Focal Inflammation in Carotid Vulnerable Plaque: A High-Resolution Magnetic Resonance Vessel Wall Imaging Study. *J Clin Med.* 2026;15(4):1452. doi:10.3390/jcm15041452.

9. **Kempf T, Zarbock A, Widera C, Butz S, Stadtmann A, Rossaint J, et al. GDF-15 is an inhibitor of leukocyte integrin activation required for survival after myocardial infarction in mice. Nat Med. 2011;17(5):581-588. doi:10.1038/nm.2354.**

10. Miller AM, Liew FY. The IL-33/ST2 pathway—a new therapeutic target in cardiovascular disease. Pharmacol Ther. 2011;131(2):179-186. doi:10.1016/j.pharmthera.2011.02.005.

R5

Extracellular Vesicles and Exosomal miRNAs: The Next-Generation Biomarkers of Atherosclerosis

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Introduction

Atherosclerosis is increasingly viewed as a dynamic and complex disease encompassing endothelial dysfunction, lipid accumulation, chronic inflammation, immune-cell activation and vascular remodeling. While conventional biomarkers such as LDL-cholesterol remain fundamental to cardiovascular risk assessment, they primarily reflect systemic lipid exposure and do not fully capture the biological activity, cellular composition or inflammatory state of an individual atherosclerotic plaque. This has prompted the search for biomarkers that can provide information closer to the underlying disease process.

Extracellular vesicles (EVs), including exosomes and other small membrane-bound vesicles, have emerged as promising candidates in this context. These particles are released by virtually all cell types and circulate in blood and other biological fluids. Importantly, EVs contain biologically active cargo, including proteins, lipids, messenger RNA and microRNAs (miRNAs), which can reflect the state of their cells of origin. As they can transport this molecular information between cells, EVs are increasingly viewed not simply as biomarkers but also as mediators of vascular biology. Their circulating presence has therefore generated interest in the concept of a molecular “liquid biopsy” for cardiovascular disease. [1–3]

Why extracellular vesicles?

Extracellular vesicles (EVs), including exosomes and other vesicle subtypes, have emerged as promising candidates in this context. These particles are released by virtually all cell types and circulate in blood and other biological fluids. Importantly, EVs contain biologically active cargo, including proteins, lipids, messenger RNA and microRNAs (miRNAs), which can reflect the state of their cells of origin. As they can transport this molecular information between cells, EVs are increasingly viewed not simply as biomarkers but also as mediators of vascular biology. Their circulating presence has therefore generated interest in the concept of a molecular “liquid biopsy” for cardiovascular disease. [1–3]

The biological importance of EVs lies in their molecular cargo. Their lipid bilayer provides protection to nucleic acids from degradation, allowing molecules such as miRNAs to remain relatively stable in the circulation. As EV cargo is influenced by the physiological state of the cell from which the vesicle originates, circulating EVs may provide information about endothelial injury, platelet activation, macrophage activity, vascular smooth-muscle-cell responses and other processes occurring during atherosclerosis. [1–3]

This cellular origin is particularly relevant to atherosclerosis. Endothelial cells, platelets, vascular smooth-muscle cells, monocytes and macrophages can all release EVs. These vesicles can participate in intercellular communication and influence endothelial function, inflammatory signaling, platelet activation, vascular remodeling and plaque biology. Thus, the quantity of circulating EVs, their cellular origin and their molecular cargo may each provide complementary information about vascular disease. [1–3]

An important conceptual shift is that EVs should not be considered merely passive “markers” released from damaged cells. Experimental evidence suggests that EVs themselves can influence disease mechanisms. For example, EV-mediated transfer of proteins, lipids and regulatory RNAs can modify recipient-cell behaviour, potentially affecting inflammatory responses, endothelial dysfunction,

vascular smooth-muscle-cell phenotype and macrophage function. In advanced atherosclerotic lesions, EV-associated molecular signals may therefore participate in processes that influence plaque progression and stability. [1,2]

Exosomal miRNAs: the molecular cargo

Among the different components carried by EVs, miRNAs are particularly attractive as potential biomarkers. miRNAs are short, non-coding RNA molecules that regulate gene expression predominantly at the post-transcriptional level. Individual miRNAs can regulate multiple target genes and therefore influence interconnected pathways such as inflammation, endothelial function, lipid metabolism, proliferation, apoptosis and vascular remodeling. [4]

The incorporation of miRNAs into EVs provides an additional level of biological stability. Rather than remaining freely exposed to circulating RNases, EV-associated miRNAs are enclosed within a membrane structure, potentially allowing them to persist in the circulation and reach distant cells. Consequently, EV-associated miRNA profiles may contain information about both the cellular source and the biological state of the vascular system. [1,3,4]

Several miRNAs have attracted attention in atherosclerosis. These include **miR-126, miR-146a, miR-223, miR-21 and miR-155,** among others. miR-126 is closely associated with endothelial biology and vascular integrity, whereas miR-146a and miR-155 are involved in inflammatory signaling. miR-21 has been linked to vascular remodeling and cellular responses, while miR-223 has been studied in relation to inflammatory and lipid-related pathways. The biological effects of individual miRNAs can, however, vary according to their cellular source, target cells and disease context. [4]

This complexity may actually be advantageous from a biomarker perspective. Rather than relying on a single molecule, a panel of EV-associated miRNAs could potentially provide a broader molecular representation of disease activity. Such a panel might capture several pathological processes simultaneously—for example, endothelial injury, macrophage activation, inflammatory signaling and vascular remodeling. This approach could potentially be more informative than measuring an isolated circulating protein or a single miRNA. [1,2,4]

EVs and plaque biology

The relationship between EVs and atherosclerotic plaque biology is particularly interesting as different vascular and inflammatory cell populations produce distinct EV populations. Endothelial-derived EVs may reflect endothelial activation or injury, while platelet-derived EVs can be associated with platelet activation and thrombo-inflammatory processes. EVs released by macrophages and vascular smooth-muscle cells may provide information about inflammatory activity, extracellular-matrix remodeling and changes in plaque architecture. [1,2]

EVs may also participate directly in plaque progression. Their uptake by recipient cells can modify cellular signaling, potentially influencing inflammation, apoptosis, lipid handling and vascular-cell phenotype. In this way, EVs occupy an interesting position at the interface between biomarker biology and disease mechanism: the same particles that may provide information about the state of atherosclerosis can also contribute to communication between cells within the vascular environment. [1,2]

This raises an important possibility for clinical laboratory medicine. Instead of simply asking whether a patient has atherosclerosis, EV profiling could eventually help answer a more biologically relevant question: *what is happening within the atherosclerotic vascular compartment?* Such information could complement traditional risk factors and imaging rather than replace them.

Emerging clinical evidence

The transition from experimental observations to clinical biomarker studies is now becoming increasingly visible. A particularly interesting 2025 investigation evaluated plasma EV-derived miRNAs in individuals with subclinical coronary atherosclerosis. The study employed a discovery and validation approach and identified six differentially expressed miRNAs. A three-miRNA combination consisting of **miR-146b-5p, miR-4701-3p and miR-1180-3p** demonstrated promising discrimination of

subclinical coronary atherosclerosis, with an area under the receiver operating characteristic curve (AUC) of **0.8281**, along with 93.75% sensitivity and 93.75% specificity in the reported analysis. [5]

The study is particularly interesting as it addresses an important clinical objective: identifying vascular disease before the occurrence of overt cardiovascular events. Functional enrichment analysis suggested that the candidate miRNAs were related to pathways involving inflammation, lipid metabolism and vascular remodeling. However, the findings should be interpreted as an early biomarker-development signal rather than as evidence of an immediately deployable clinical test. The investigators themselves noted important limitations, including the relatively small cohorts, restriction to male participants, cross-sectional design and absence of longitudinal outcome assessment. [5]

Nevertheless, this type of study illustrates the potential of EV-miRNA signatures to detect subtle biological changes. A multimarker molecular signature could theoretically detect subtle biological changes before they become apparent through conventional clinical manifestations. Such an approach may ultimately complement imaging modalities such as coronary CT or carotid ultrasound by providing molecular information about disease activity.

From research biomarker to clinical laboratory test

Despite considerable enthusiasm, several barriers must be addressed before EV-based biomarkers can become routine clinical laboratory tests. One major challenge is pre-analytical variability. Factors such as the type of blood collection tube, processing time, centrifugation conditions, storage temperature and number of freeze–thaw cycles can influence EV measurements and miRNA profiles. [1,2]

A second challenge is the absence of complete standardization in EV isolation and characterization. Different approaches—including ultracentrifugation, size-exclusion chromatography, precipitation-based methods and immunoaffinity techniques—can recover EV populations with different characteristics. Consequently, results obtained using one platform may not be directly comparable with those obtained using another. [1–3]

Normalization of EV-associated miRNA measurements is another important analytical issue. Unlike conventional biochemical analytes for which established reference materials and standardized calibrators may be available, EV-miRNA assays currently lack universally accepted approaches for normalization and quality control. Selection of endogenous controls, spike-in controls and normalization strategies can substantially influence reported miRNA expression levels. [1,2,4]

For clinical laboratory implementation, therefore, analytical validity will be just as important as biological discovery. Future studies will need to establish reproducible workflows covering blood collection, EV isolation, characterization, RNA extraction, miRNA quantification and data normalization. Large prospective studies will then be required to determine whether these biomarkers provide incremental value beyond established cardiovascular risk factors and imaging.

Where could EV-miRNA biomarkers fit clinically?

The most realistic future role for EV-miRNA profiling may not be to replace established cardiovascular biomarkers but to complement them. LDL-cholesterol provides information about lipid exposure; hsCRP and related inflammatory markers provide information about systemic inflammation; and imaging provides structural information about atherosclerotic burden. EV-associated miRNAs could potentially add a molecular layer describing cellular activation and intercellular signaling.

This integrated approach could eventually combine conventional risk factors, biochemical biomarkers, imaging findings and molecular signatures into a more comprehensive assessment of cardiovascular risk. In particular, EV-miRNA panels may be valuable in research settings focused on early or subclinical atherosclerosis, treatment response and residual cardiovascular risk. [1,2,5]

Another attractive possibility is longitudinal monitoring. If EV cargo changes in response to disease progression or therapeutic intervention, serial measurements could potentially provide dynamic information rather than a single snapshot. However, this application remains investigational and requires prospective studies demonstrating that changes in EV-miRNA profiles correspond reliably to clinically meaningful changes in vascular disease. [1,2]

Future Prospects

The next phase of EV research is likely to move beyond the discovery of individual biomarkers toward multimarker signatures. Integration of EV-derived miRNAs with EV proteins, lipids and other nucleic acids may provide a more complete molecular picture of vascular disease. Multi-omics approaches could also help identify signatures associated with specific stages or phenotypes of atherosclerosis. [1,2]

Artificial intelligence and machine-learning approaches may further facilitate the interpretation of complex EV datasets. Rather than relying on a single threshold for one biomarker, computational models could potentially integrate multiple EV features with clinical variables and imaging data. However, such approaches will require large, well-characterized and externally validated cohorts before they can be considered clinically reliable.

Equally important will be the development of standardized laboratory protocols. Reproducibility across laboratories is essential if EV-miRNA testing is ultimately to become part of cardiovascular laboratory medicine. International consensus regarding EV nomenclature, isolation, characterization, quality control and reporting will therefore be critical.

Conclusion

Extracellular vesicles and their miRNA cargo represent an emerging interface between molecular biology, vascular pathology and clinical laboratory medicine. Their ability to carry relatively stable molecular information from specific cell populations makes them attractive candidates for atherosclerosis biomarker development. Unlike conventional circulating biomarkers that primarily reflect systemic processes, EVs may provide information about cellular activation and intercellular communication occurring within the vascular environment. [1–4]

Recent clinical work, including the identification of plasma EV-miRNA signatures associated with subclinical coronary atherosclerosis, demonstrates the potential of this approach while also highlighting the need for larger and more diverse validation studies. [5] The major challenge is now to translate promising molecular signatures into standardized, reproducible and clinically validated laboratory assays.

Rather than simply asking whether atherosclerosis is present, future biomarker strategies may increasingly aim to determine which biological processes are active within the vascular system. EV-miRNA profiling could become an important component of this next generation of cardiovascular risk assessment—but its clinical utility will depend on rigorous analytical standardization and prospective validation.

The next generation of cardiovascular biomarkers may come not simply from measuring more proteins, but from decoding the molecular messages carried by extracellular vesicles.

References

1. Rizzuto AS, Fichtner I, Carugo S, Radeghieri A, Macchi C, Ruscica M. The role of extracellular vesicle-derived miRNA in the atherosclerotic burden. Am J Pathol. 2025;195(12):2274-2287. doi:10.1016/j.ajpath.2025.08.006.

2. Deng H, Qiu W, Zhang Y, Hua J. Extracellular vesicles in atherosclerosis cardiovascular disease: emerging roles and mechanisms. Front Cardiovasc Med. 2025;12:1611557. doi:10.3389/fcvm.2025.1611557.

3. Boulanger CM, Loyer X, Rautou PE, Amabile N. Extracellular vesicles in coronary artery disease. Nat Rev Cardiol. 2017;14(5):259-272. doi:10.1038/nrcardio.2017.7

4. Letonja J, Petrovič D. A review of microRNAs and lncRNAs in atherosclerosis as well as some major inflammatory conditions affecting atherosclerosis. Biomedicines. 2024;12(6):1322. doi:10.3390/biomedicines12061322.

5. Peña-Peña M, Zepeda-García Ó, Posadas-Sánchez R, Sánchez-Muñoz F, Domínguez-Pérez M, Martínez-Greene JA, et al. A plasma extracellular vesicle-derived microRNA signature as a potential biomarker for subclinical coronary atherosclerosis. Int J Mol Sci. 2025;26(17):8727. doi:10.3390/ijms26178727.

R6

Measure Cholesterol: BUT Understand the LDL Particle and Recognise the Cardiovascular Risk

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For many years, doctors have used LDL Cholesterol (LDL-C) levels to assess the risk of cardiovascular disease. LDL-C tells us how much cholesterol is carried by LDL in the blood. However, a low LDL-C does not always mean that the risk of heart disease or stroke is completely absent. Some patients may develop cardiovascular disease even when their LDL-C is below 70mg/dL. This is because the arteries doesn't experience cholesterol as a dissolved fluid but as a preassembled particle.

Diagnostic Illusion of LDL-C:

Cardiovascular risk depends on LDL-C mass to particle ratio. Some lesions underestimate the cardiovascular risk. The diagnostic blind spot is especially significant in patients with metabolic syndrome, insulin resistance, visceral obesity and type 2 diabetes. Some patients carry cholesterol mass with small number of large buoyant LDL particles but holds large burden of cholesterol.

While some people have exact same quantity of LDL-cholestrol, with higher number of small dense LDL particles. Hence these are smaller particles holding less cholesterol per particle. This is called LDL mass to particle discordance.

Why LDL particle number matters:

The risk of atherosclerosis is related not only to the amount of cholesterol but also to the number of LDL particles circulating in the blood. Therefore, Small dense LDL particles are considered more likely to contribute to atherosclerosis in contrast to large, buoyant LDL particles.

The rate of particle entry in the arterial wall is directly proportional to total concentration of circulating LDL-particle and does not depend on total cholesterol mass. Increased particle count will collide more and even cross the endothelial barrier more.

Large LDL particle are buoyant and less prone to retention and can deposit out the intimal space back to systemic circulation via lymphatic drainage. In contrast Small dense LDL have more affinity for proteoglycans within extracellular matrix of arterial wall and gets physically trapped. Small dense LDL particle also undergoes surface conformation affecting Apo B100 molecule. It reduces binding affinity for hepatic LDL receptor. Thus sdLDL particle remain in systemic circulation for a longer time in comparison to large LDL particle. This longer circulation time increases exposure to oxidative stress and further boosts chances of entering the vascular wall. Oxidised LDL acts as a major pro inflammatory trigger. Endothelial cell reacts with oxLDL and adhesion molecule like ICAM-1 and VCAM-1which releases chemoattractants that draw monocytes into the intima. These monocytes mature into macrophages and engulfs the modified molecule. Macrophage engorged with lipid changes to foam cell. Foam cell acts as the fundamental structural building block of Atherosclerotic Plaque.

ApoB -A way to count harmful lipoprotein particles:

Apoprotein is one molecule present on the outer shell of every atherogenic lipoprotein particle like VLDL, IDL, Large LDL and Lipoprotein(a). There is approximately one Apo-B molecule per atherogenic particle. Thus Apo B estimation is a better predictor of cardiovascular event especially significant for patients who have low LDL-C levels but still have residual high Triglyceride rich small dense particle.

Lipoprotein (a)- an additional risk factor:

Lipoprotein (a) consist of LDL like particle containing Apo B 100 linked with Disulphide bridge to glycoprotein Apo (a). It acts as genetic accelerator of vascular risk. Lp (a) doesn't alter with diet, exercise and lipid lowering drugs unlike LDL-C. Lp(a) crosses endothelium, tightly binds to intimal proteoglycans and deliver cholesterol to macrophages and form foam cell. It also acts as carrier of phospholipid and activate endothelial cells and trigger proinflammatory cytokines and drive calcification. Thirdly, it impairs normal fibringenolysis by inhibiting Plasminogen activation. Moreover, rupture of unstable plaque make increased Lp (a) and cause occlusive thrombus, Triglyceride rich lipoprotein and remnant cholesterol. Therefore, increased Lp(a) can contribute to atherosclerotic cardiovascular disease.

Remnant Cholesterol and Remnant Particles:

Endothelial lipoprotein lipase breaks down VLDL and chylomicron to form remnant lipoprotein. These are much more enriched with cholesterol and can penetrate intima like LDL particle and can accelerate foam cell formation and intensify the inflammatory microenvironment of plaque.

Thus, to translate particle biology into advanced lipid testing following tests should be integrated for patient care:

1. Universal Lp(a) measurement-every patient must get their Lp (a) levels checked once in a lifetime.
2. Apo B measurement is suggested routinely for patient with insulin resistance, visceral obesity, metabolic syndrome and hypertriglyceridemia.
3. Remnant cholesterol assessment can be considered while evaluating total burden of atherogenic particle.

Conclusion:

Measuring and lowering LDL-C provides an essential base for preventive cardiology from very beginning. However residual cardiovascular risk demonstrate that only. cholesterol mass management cannot halt the progression of vascular disease.

Hence, by evaluating Apo B, Lp (a), small dense LDL remnant particles we may get a clear picture of plaque development. Measuring particle count remains an important baseline, while understanding the type and number of particles helps us identify the hidden risks and may guide in appropriate management.

References

1. Mach F. 2025 Focussed update of 2019 ESC/EAS Guidelines for the management of Dyslipidemia. European Heart Journal.2025;46(42):4359-4378.
2. Blumanthal RS, Morris PB. Guidelines on management of Dyslipidemia.Circulation.2026;153(17):e1154-e1276.

R7

Could ApoB Predict COVID-19 Risk? Exploring a New Biomarker

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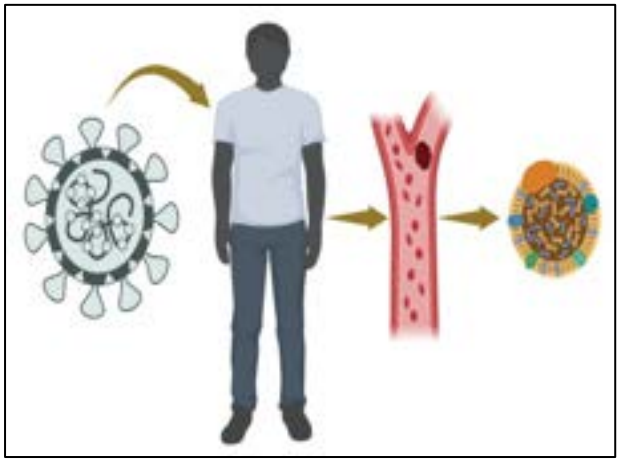
The COVID-19 pandemic has presented unprecedented challenges to global health, particularly concerning cardiovascular complications. Understanding and mitigating these risks has become paramount as we navigate the ongoing crisis. Scientists are still looking for biomarkers that can assist anticipate the severity of diseases and direct early therapies, even though the introduction of vaccinations and antivirals has saved countless lives. ApoB protein that has historically been linked to cardiovascular health, can be one such biomarker.

Could the severity of COVID-19 also be predicted by this lipid transport protein? In the battle against pandemic viral infections, this newsletter examines the most recent research findings, therapeutic applications, and potential future paths for ApoB as a novel biomarker.

Lipid Metabolism and COVID-19: An Odd Association



Cholesterol regulates the entry of the virus causing COVID-19, SARS-CoV-2, into host cells as cholesterol-enriched rafts are specific platforms that enable viral endocytosis through the Angiotensin converting enzyme (ACE2) receptor. This causes immunological dysregulation and an inflammatory cascade and the resulting inflammation and cytokine storms inturn negatively impacts natural lipid biosynthesis pathways. As SARS-CoV-2 comprises a bilayer lipid envelope, the viral replication inside the host cell also disrupts lipid metabolism and lipid resources. This has been observed that patients with COVID-19 infection, who are hospitalised frequently have dyslipidaemia with increased triglycerides and decreased HDL-C, and that is quite common in critical cases. These changes imply that the pathogenesis of COVID-19 may include lipid-related mechanisms, such as lipoproteins containing ApoB.

Apolipoprotein B (APO B): The Key Player

APO B is a primary protein component of atherogenic lipoproteins, such as low-density lipoprotein (LDL), very-low-density lipoprotein (VLDL), and intermediate-density lipoprotein (IDL) essential for cholesterol transport in the bloodstream. ApoB comes in two main forms: ApoB-100 produced by Liver, a structural protein for LDL and VLDL particles and ApoB-48 produced in intestine aids Chylomicron production. Unlike LDL cholesterol (LDL-C), which measures the cholesterol within these particles, APO B provides a more accurate assessment of cardiovascular risk by quantifying the actual number of lipoprotein particles since there is just one ApoB molecule per atherogenic particle. Elevated ApoB levels are thus linked to a higher risk of cardiovascular disease due to the increased number of atherogenic particles capable of penetrating arterial walls and triggering inflammation.

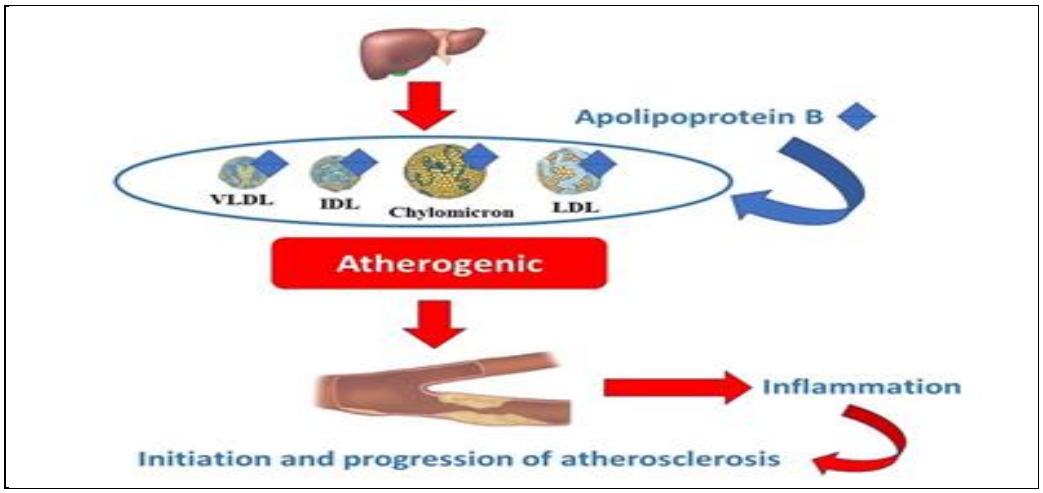
Mechanistic Perspectives: The Potential Impact of ApoB on COVID-19 Results

How ApoB may contribute to COVID-19 risk is explained by a number of tenable mechanisms:

1. Endothelial dysfunctioning and ApoB are involved in endothelial inflammation and atherosclerosis. These are the two conditions that are related to the vascular damage brought on by COVID-19. Endothelial damage caused by the virus results in: Coagulopathies, thrombosis of the microvascular system and an embolism in the lungs.

2. Elevated ApoB levels thus may intensify these consequences, perhaps making infected patients' prognosis worse.

3. The virus's inflammatory response can destabilize atherosclerotic plaques, further leading to acute cardiovascular events.



The link between Apo B and atherosclerosis.

Immune modulation and ApoB

ApoB-rich lipoproteins have the ability to interact with immune cells, including dendritic cells and macrophages. According to certain research, ApoB-lipoproteins may change viral entrance or clearance by binding to viral particles. The cytokine storm observed in severe COVID-19 may be exacerbated by elevated ApoB, which may trigger the production of pro-inflammatory cytokines.

Shared Risk Factors: Obesity and Metabolic Syndrome

Both elevated ApoB and severe COVID-19 are associated with obesity, diabetes, and metabolic syndrome. ApoB may act as a proxy marker reflecting underlying metabolic derangements that predispose individuals to worse COVID-19 outcomes.

ApoB as a Hospitalisation Risk Predictor

The lipid profiles in COVID-19 patients were examined in many cohort studies. The findings demonstrated that, regardless of LDL-C or triglyceride levels, increased ApoB levels were associated with a greater probability of hospitalisation and intensive care unit admission. Researchers assessed the extended lipid profiles of hospitalised COVID-19 patients and found that ApoB-to-ApoA1 ratios were much greater in patients with increased levels of inflammatory markers- C-reactive protein (CRP), Interleukin-6 (IL-6- a cytokine that promotes inflammation) and D-dimer, a coagulopathy marker. One reliable indicator of respiratory failure and the requirement for mechanical ventilation can be ApoB/ApoA1 ratio.

ApoB and Long COVID: A Path Forward

Long COVID is one of the most confusing problems of the pandemic, since patients continue to suffer symptoms like as exhaustion, fogginess, and dyspnoea months after they have recovered. These symptoms might be caused by chronic metabolic and endothelial dysfunction, according to some studies. ApoB may be an indicator of persistent inflammation linked to lipids, offering information on the long-term pathophysiology of COVID-19. Long-term COVID therapy regimens may include an investigation of ApoB-lowering techniques.

A Hidden Biomarker in the Public Eye

Integrative biomarkers spanning immunological, cardiometabolic health, and infectious illness have become increasingly important as a result of the COVID-19 pandemic. As a long-standing indicator of

cardiovascular risk, ApoB may provide important information about: Immune and endothelial dysfunction, post-acute sequelae of viral infections, and susceptibility to severe COVID-19.

Although clinical guidelines has not yet included ApoB-based COVID-19 screening, the mounting evidence is however strong. Future studies may confirm ApoB's status as a vital pandemic preparation tool, with potential uses in other inflammatory and viral illnesses.

Call to Action: What Are the Next Steps for Researchers and Clinicians?

1. Risk Stratification: Measuring APO B levels and ApoB/ApoA1 ratio in addition to inflammatory markers in COVID-19 patients could become a standard practice to identify those at higher risk for severe disease, enabling more tailored and proactive treatment approaches.

2. Preventive Measures: For individuals with known high APO B levels, stringent preventive measures against COVID-19, such as vaccination and booster shots and lifestyle modifications, are highly recommended.

3. Therapeutic Interventions: Incorporating lipid-lowering treatments in the management plan for COVID-19 patients with elevated APO B levels might be beneficial.

4. Researchers may Conduct prospective research correlating COVID-19 results to pre-infection ApoB levels. Investigate the mechanisms that link ApoB to viral clearance and immunological respons alonside examining the possibility of ApoB-targeting therapies for both acute and chronic COVID or viral infections

References

1. Myrou A, Barmpagiannos K, Papathanasiou E, Kachtsidis V, Kiouli C, Tziomalos K. Association Between Lipid Profile and COVID-19 Severity: Insights from a Single-Center Cross-Sectional Study in Northern Greece. J Clin Med. 2025 Jun 9;14(12):4082.

2. Kowalska K, Sabatowska Z, Forycka J, Młynarska E, Franczyk B, Rysz J. The Influence of SARS-CoV-2 Infection on Lipid Metabolism-The Potential Use of Lipid-Lowering Agents in COVID-19 Management. Biomedicines. 2022 Sep 18;10(9):2320.

3. Abu-Farha M, Thanaraj TA, Qaddoumi MG, Hashem A, Abubaker J, Al-Mulla F. The Role of Lipid Metabolism in COVID-19 Virus Infection and as a Drug Target. Int J Mol Sci. 2020 May 17;21(10):3544.

4. Garcia-Gil M, Barrios V, Benito B. Dyslipidemia and COVID-19: Possible Role in Severe Disease. *Curr Cardiol Rep.* 2021;23(10):140.

5. Dorobanțu M, Halațiu VB, Gheorghe-Fronea O, Bala CG, Moldovan H, Irinel-Parepa R, Rodean IP, Benedek I, Benedek T. The Association between Apolipoprotein B, Cardiovascular Risk Factors and Subclinical Atherosclerosis-Findings from the SEPHAR National Registry on Hypertension in Romania. Int J Mol Sci. 2023 Feb 1;24(3):2813.

R8

Dyslipidemia: Current Practices and Guidelines

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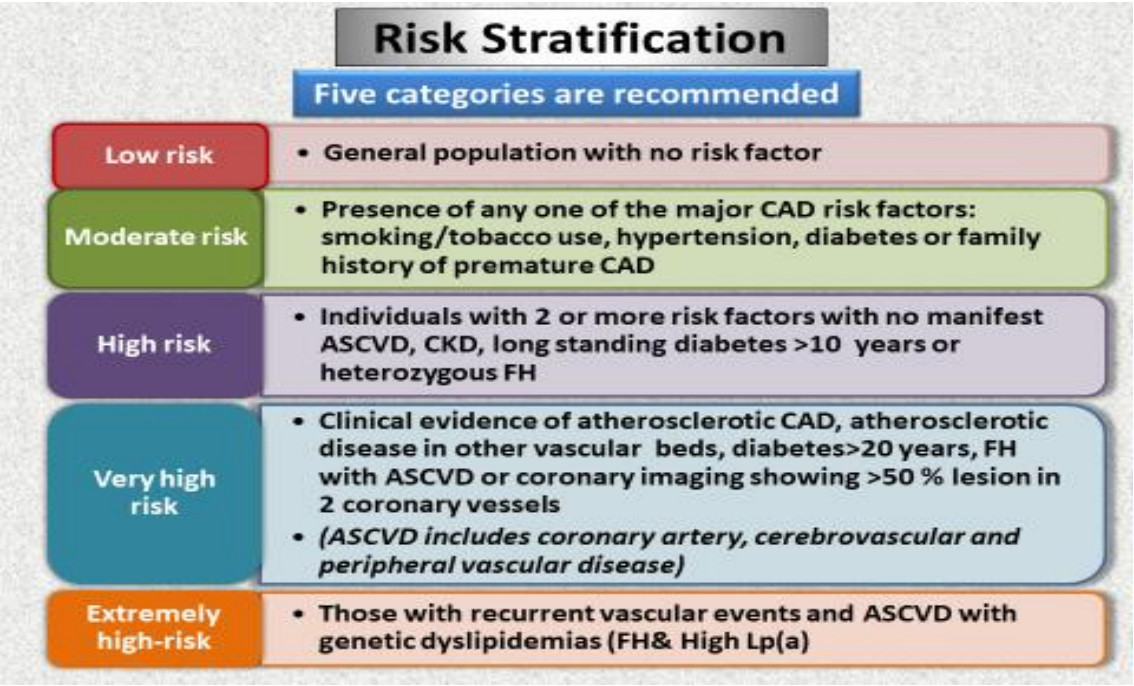
Dyslipidemia, one of the most commonly detected & treated chronic conditions, is a vital risk factor due to its significant association with cardiovascular disease (CVD). Its importance lies in the fact that it is a modifiable risk factor as about 80% of lipid disorders are related to diet & lifestyle, with the rest being familial. There are no single comprehensive dyslipidemia guidelines, but what is common among them all is that they all identify the routine lipid panel as a practical starting point. For CVD, elevated LDL cholesterol is essentially causal and guidelines world wise recommend treatment targets for this lipid fraction. Currently, dyslipidemia is an expanding area of research due to recent insight into their role in the molecular basis and genetic origin of the condition. Hence, it ispertinent to discuss, very briefly, the salient recent guidelines for the management of dyslipidemia so as to consider an updated clinical approach for its management.

In the recent Clinical Practice Guidelines for Dyslipidemia Management given by Sawhneyet al in 2024 (1), the focus was on the management of dyslipidemia, specifically in the Indian population. Since India has the distinction of the highest number of dyslipidemia patients globally, the study highlighted specific points as follows:

- A significant epidemiological, genetic and causal association exists between total cholesterol, non-HDL-c, LDL-c &triglycerides with ASCVD events and morality.
- Increasing population level total cholesterol, LDL-c& triglycerides trends have been observed in emigrant South Asians vis-à-vis the developed countries where a declining trend has been observed.
- Raised triglycerides are a unique feature of dyslipidemia in India.
- Hypercholesterolemia prevalence in southern states is greater than the north Indian states with urban more than rural, while triglycerides& HDL-care similar throughout the country.
- Importantly, the level of awareness in the population and individual levels remains low.

Recommended Levels of Various Lipid Parameters					
Lipid Parameter		**Low Risk**	**Moderate Risk**	**High Risk**	**Very High Risk**
Total Cholesterol		**<200**	**<200**	**200-239**	**≥240**
L.D.L-C		**<100**	**<100**	**<70**	**<55**
H.D.L-C	**M**	**>40**	**>40**	**>40**	**>40**
	F	**>50**	**>50**	**>50**	**>50**
Non H.D.L-C		**<130**	**<130**	**<100**	**<85**
Triglycerides		**<150**	**<150**	**<150**	**<150**



Sawhney JPS, Ramakrishnan S, Madan K et al. CSI clinical practice guidelines for dyslipidemia management: Executive Summary. Indian Heart Journal 2024: 76: S6-S19

Sawhney et al recommended accurate measurement of the different lipid parameters and measurement of non-fasting levels of total cholesterol, non-HDL-c, LDL-c& triglycerides for risk estimation and guiding treatment. They also advised the importance of risk stratification in guiding lipid lowering therapy and to identify goals. They suggested that Lp(a) should be measured at least once in a person's lifetime. Significantly, non-HDL-c has been included as a co-primary goal in their guidelines and measurement of non-HDL-c has been recommended as it is a simple calculation and highly predictive of ASCVD events.

The 2025 Focussed Update of the 2019 ESC/EAS Guidelines for the management of dyslipidemia (2) has also stated that epidemiologic and genetic studies strongly support a causal and direct continuous association between high Lp(a) levels and higher risk of ASCVD and aortic valve stenosis. Also, Lp(a) levels above 50 mg/dL (105 nmol/L) should be considered in all adults as a risk enhancing factor. They stated that triglyceride levels are associated with CVD risk, independent of LDL-c levels and recommended statins as the first drug of choice to reduce CVD risk in high-risk patients.

Hence in conclusion, atherosclerosis is a chronic progressive disease that begins early, progresses slowly and along with exposure to high levels of lipids, is associated with a higher risk of CVD. Statins are the drugs of choice for the management of dyslipidemia. A non-fasting lipid profile is acceptable and in acute coronary syndrome, the goal is to achieve LDL-c levels below 55 mg/dL as early as possible. Lifestyle interventions like increased physical activity and dietary modifications are important. Better awareness of dyslipidemia management can go a long way in reducing the morbidity and mortality associated with CVD in our country.

References

1. Sawhney JPS, Ramakrishnan S, Madan K et al. CSI clinical practice guidelines for dyslipidemia management: Executive Summary. Indian Heart Journal 2024: 76: S6-S19

2. 2025 Focused Update of the 2019 ESC/EAS Guidelines for the management of Dyslipidemias. Developed by the Task Force for the management of Dyslipidemias of the European Society of Cardiology (ESC) and the European Atherosclerosis Society (EAS). European Heart Journal 2025; 00;1-20.

R9

Stroke: A Overview

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Stroke in India: where we stand and what to do next

Stroke is now one of India's most urgent cardiovascular problems. Globally, there are almost 12 million new strokes each year and one in four adults over 25 will have a stroke in their lifetime. (1,2) In India, stroke caused an estimated 699,000 deaths in 2019 (7.4% of all deaths) with wide state-to-state variation, reflecting uneven prevention and care. (3) Recent analyses of Indian data from 1990-2021 confirm a heavy and shifting burden driven by modifiable risks: hypertension, diabetes, tobacco, dyslipidaemia, atrial fibrillation, air pollution, and sedentary lifestyles. (4)

Why atherosclerosis matters in stroke

Most strokes are ischaemic, caused by a blocked brain artery. Atherosclerosis is a key mechanism either large-artery disease (carotid or intracranial plaques) or artery-to-artery embolism from unstable plaque. Atherosclerosis also amplifies risk in cardioembolic stroke via shared factors like age, hypertension, and diabetes. Controlling BP and low-density lipoprotein cholesterol (LDL-C), eliminating tobacco, and treating diabetes and atrial fibrillation are the levers that bend the curve.

Prevention: the highest-yield actions

Blood pressure. After a stroke or transient ischaemic attack (TIA), most patients with hypertension should target <130/80 mm Hg with lifestyle and medication. Tight BP control also prevents first strokes. (5)

Lipids. LDL-C lowering is central. For secondary prevention and very-high-risk patients, guidelines recommend ≥50% LDL-C reduction and a target <55 mg/dL (<1.4 mmol/L). Add ezetimibe and then a PCSK9 inhibitor if goals are not met on maximal statin. (6) The 2025 focused update reinforces early, aggressive LDL-C lowering across risk tiers to reduce cardiovascular events, including stroke. (7)

Atrial fibrillation (AF). Screen systematically after ischaemic stroke; extended rhythm monitoring detects more AF. Most patients with AF and prior stroke should receive oral anticoagulation (preferably a direct oral anticoagulant unless contraindicated). (8)

Lifestyle and cardiometabolic risks. Smoking cessation, salt reduction, weight control, regular physical activity, Mediterranean-style eating patterns, glycaemic control in diabetes, and adherence to antihypertensive and lipid-lowering therapy together deliver the largest absolute risk reduction.

Acute care: time windows are real and expanding

For acute ischaemic stroke, rapid recognition and hospital transfer remain decisive. Intravenous thrombolysis is time-sensitive, and mechanical thrombectomy (MT) is now standard of care for large-vessel occlusions when eligibility criteria are met. Landmark trials show MT benefit up to 6–24 hours from last-known-well in selected patients using perfusion or clinical-core mismatch criteria (DAWN/DEFUSE - 3). Major guidelines reflect this extended window. (9,10) Emerging data explore MT beyond 24 hours in carefully selected cases, but this remains investigational and centre-specific. (12)

For India, the practical message is simple: build prehospital stroke pathways (recognition, dispatch, prenotification), expand 24 by 7 thrombolysis capability, and develop regional MT hubs with tele-stroke support for spoke hospitals.

Secondary prevention package after ischaemic stroke/TIA

1. **High-intensity statin** (e.g., atorvastatin 80 mg) with LDL-C target <55 mg/dL; add ezetimibe/PCSK9 inhibitor as needed. (6)

2. **BP control** to <130/80 mm Hg with ACEi/ARB-based regimens plus thiazide-like diuretic or calcium-channel blocker as required. (5)

3. **Antithrombotic therapy** tailored to mechanism:

    - Non-cardioembolic stroke: single antiplatelet (aspirin or clopidogrel) long-term; consider short dual antiplatelet therapy (DAPT) for minor stroke/TIA per local protocol.

    - AF-related stroke: oral anticoagulation unless contraindicated. (5)

4. **Lifestyle and diabetes management** as above; treat triglycerides and consider icosapent ethyl if triglycerides 135-499 mg/dL on statin and LDL-C 41-100 mg/dL. (10,11)

5. **Carotid disease**: evaluate for revascularization when symptomatic stenosis is present; ensure best medical therapy for all.

What the Indian Society for Atherosclerosis Research can drive

1) **Bench-to-community lipid control.** Promote routine LDL-C goal-oriented care in cardiology, neurology, and primary care clinics; publish simple treatment algorithms and dose-titration schedules aligned to the <55 mg/dL target in very-high-risk patients. (6)

2) **Hypertension action.** Support practical home BP monitoring programs with monthly adherence nudges, focusing on states with the highest stroke DALYs. (4)

3) **Tobacco and air quality.** Partner with state programs on cessation services and clean-air advocacy; both yield stroke gains. (4)

4) **Stroke-ready districts.** Advocate for district-level stroke pathways: EMS training, hospital prenotification, standard thrombolysis kits, and defined referral routes to thrombectomy hubs using tele-stroke. Align messaging with the extended MT window to reduce nihilism about late presenters. (13)

5) **Data and equity.** Support audits of door-to-needle and door-to-groin times, LDL-C attainment rates, and medication adherence, with a focus on rural and underserved regions where burden is high and services are thin. (3)

India can cut stroke deaths and disability by doing the basics well - lower BP, lower LDL-C, treat AF, stop tobacco, and organize acute stroke systems. Science is clear; the opportunity is large; the tools are in hand.

References

1. Feigin VL, Brainin M, Norrving B, Martins SCO, Lindsay P, Roth GA, et al. World Stroke Organization (WSO) Global Stroke Fact Sheet 2025. Int J Stroke. 2025;20(2):[epub ahead of print]. doi:10.1177/17474930241308142.

2. GBD 2021 Stroke Risk Factor Collaborators. Global, regional, and national burden of stroke and its risk factors, 1990–2021: a systematic analysis for the Global Burden of Disease Study 2021. Lancet Neurol. 2024;23(10):973–1003. doi:10.1016/S1474-4422(24)00369-7.

3. India State-Level Disease Burden Initiative Neurological Disorders Collaborators. The burden of neurological disorders across the states of India: the Global Burden of Disease Study 1990–2019. Lancet Glob Health. 2021;9(8):e1129–e1144.

4. Behera DK, Rahut DB, Mishra S. Analyzing stroke burden and risk factors in India using data from the Global Burden of Disease Study. Sci Rep. 2024 Sep 30;14(1):22640. doi: 10.1038/s41598-024-72551-4. PMID: 39349946; PMCID: PMC11443044.

5. Kleindorfer DO, Towfighi A, Chaturvedi S, Cockroft KM, Gutierrez J, Lombardi-Hill D, et al. 2021 guideline for the prevention of stroke in patients with stroke and transient ischemic attack. Stroke. 2021;52(7):e364–e467. doi:10.1161/STR.0000000000000375.

6. European Society of Cardiology; European Atherosclerosis Society. 2025 Focused Update of the 2019 ESC/EAS Guidelines for the management of dyslipidaemias. 2025. Available from: https://www.escardio.org (accessed 24 Sep 2025).

7. Koskinas KC, Mach F, Catapano AL, et al. 2025 ESC/EAS Focused Update on dyslipidaemias. Atherosclerosis. 2025;[epub ahead of print]. doi:10.1016/j.atherosclerosis.2025.08.XXX.

8. Nogueira RG, Jadhav AP, Haussen DC, Bonafe A, Budzik RF, Bhuva P, et al. Thrombectomy 6 to 24 hours after stroke with a mismatch between deficit and infarct (DAWN). N Engl J Med. 2018;378(1):11–21. doi:10.1056/NEJMoa1706442.

9. Albers GW, Marks MP, Kemp S, Christensen S, Tsai JP, Ortega-Gutierrez S, et al. Thrombectomy for stroke at 6 to 16 hours with selection by perfusion imaging (DEFUSE-3). N Engl J Med. 2018;378(8):708–718. doi:10.1056/NEJMoa1713973.

10. Bhatt DL, Steg PG, Miller M, Brinton EA, Jacobson TA, Ketchum SB, et al. Cardiovascular risk reduction with icosapent ethyl for hypertriglyceridemia (REDUCE-IT). N Engl J Med. 2019;380(1):11–22. doi:10.1056/NEJMoa1812792.

11. Bhatt DL, Steg PG, Miller M, Brinton EA, Jacobson TA, Ketchum SB, et al. Effects of icosapent ethyl on total ischemic events: the REDUCE-IT trial. J Am Coll Cardiol. 2019;73(22):2791–2802. doi:10.1016/j.jacc.2019.02.032.

12. Endovascular Treatment Beyond the 24-Hour Mark. https://eso-stroke.org/endovascular-treatment-beyond-the-24-hour-mark/

13. Turc, Guillaume. Mechanical Thrombectomy in Late-Time Windows: Time to Treat More Patients. Stroke 2023; 54: 3: pp 731-732. doi:10.1161/STROKEAHA.122.041420

14. India State-Level Disease Burden Initiative Neurological Disorders Collaborators. The burden of neurological disorders across the states of India: the Global Burden of Disease Study 1990–2019. Lancet Glob Health. 2021;9(8):e1129–e1144.

15. GBD 2021 Stroke Risk Factor Collaborators. Global, regional, and national burden of stroke and its risk factors, 1990–2021: a systematic analysis for the Global Burden of Disease Study 2021. Lancet Neurol. 2024;23(10):973–1003. doi:10.1016/S1474-4422(24)00369-7.

16. Powers WJ, Rabinstein AA, Ackerson T, Adeoye OM, Bambakidis NC, Becker K, et al. 2019 update to the 2018 guidelines for the early management of acute ischemic stroke. Stroke. 2019;50(12):e344–e418. doi:10.1161/STR.0000000000000211.

R10

Inflammatory Mechanisms in Coronary Artery Disease: The Complement and Membrane Complement Regulatory Proteins

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Abstract

Complement system, the first line of defense against invading microbes contributes to the pathophysiology of many chronic inflammatory disorders. Several lines of evidence suggest that the complement system is involved in the pathogenesis of atherosclerosis. Beside its traditionally well-known proinflammatory effects, complement activation is also necessary for removal of immune complexes, debris and apoptotic cells [5C] and may therefore exert proatherogenic or atheroprotective effects within the vessel wall. Recent studies suggest leucocyte membrane complement regulatory proteins as modulators of inflammatory processes in health and disease. This mini review brings forth a brief account of the complement system, its physiological functions, mechanisms of its protective role against invading pathogens and bystander injurious effects to the self along with importance of membrane complement rgulatory proteins in modulating disease activity in atherosclerosis.

Key words: Atherosclerosis, Inflammation, complement system, membrane complement regulatory proteins, pathogenesis.

1.Methodology: Literature search for last 10 years using the standard search methodology.

2.Introduction

Coronary artery disease is a major killer worldwide. South Asians (including Asian Indians) make up roughly 25% of the global population but account for nearly 60% of the world's cardiovascular disease burden. [1] and the disease strikes about 5 to 10 years earlier in life. [1]. Despite increasing awareness to life style management, the menace continues. The disease once considered a metabolic and life style related disorder, in recent years is being identified as an immune-inflammatory disorder. In this context, the levels of elevated CRP had been the first observation followed by further studies on inflammatory markers like IL6, TNFα and others. Although, studies are mounting in current years, understanding of the immune-inflammatory mechanisms in relation to human CAD is inadequate especially because information came mainly from the studies on animal models and mechanistic insights into the components of the immune-inflammatory mechanisms are inadequate.

Complement and inflamosomes are two key components of inflammatory mechanisms in health and disease. Their importance in the pathophysiology of coronary artery disease is increasingly being envisaged.

Complement system on activation generates inflammatory peptides promoting inflammatory reactions and cell signaling. The bystander effects can cause severe self-tissue injury if complement activation goes unabated. A large number of complement regulatory proteins (Cregs)protects the normal health by controlling aggravated complement activation. Thus the complement mediated inflammatory tissue injury is largely modulated by Cregs.

This brief article attempts at buiding up some baseline understanding

3.The current knowledge

3.1. The complement system: Complement is a proinflammatory system consisting of about 50 soluble and cell surface proteins enfolding plasma zymogens, receptors and regulators. Central to the system are the zymogens which are organized in a sequencial cascade, the components are named as C1, C2, C3, C4, C5.C6, C7, C8 and C9, C6,7,8 and 9are non -enzyme components of the complement cascade. on activation by molecular patterns, biologically active peptides are generated

Additional components are factor D, B, P (Properdein) and multisubunit multimeric MBL (Mannose binding lectin) consisting of MASP1, MASP2 and Masp3, the mannose binding serine proteases.

3.2 Complement activation occurs through three different pathways, the classical, lectin, or alternative pathways. The activation of C2, C4, C3 and C5 takes place by the proteolytic cleavage of the parent molecule to a small (a) and a bigger fragment (b).

The classical pathway is initiated with the activation of C1 by antigen antibody complexes formed during the humoral response, by natural antibodies, and by other molecules that are generated as a result of an inflammatory reaction such as CRP (C-reactive protein) or serum amyloid protein. C1 consists of three subunits namely, C1q, C1r and C1s. C1q binds with any of the molecular triggers following the sequential activation of C1r, then, C1s, the esterases.

The lectin pathway begins with binding of the complex MBL and mannose-binding lectin-associated proteases 1 and 2 (MASP1 and MASP2, respectively) to a bacterial cell wall. MASP2, similarly to C1s, leads to the formation of the C3 convertase C4b2a. The alternative pathway is initiated by the spontaneous hydrolysis of C3, leading to the formation of C3(H2O). C3(H2O) forms a complex with factor B, followed by the cleavage of factor B within this complex by factor D. The final product of these enzymatic reactions is the C3 (H2O) Bb complex. The C3bBb complex has the capacity to cleave C3 to C3b and C3a. Once deposited on the surface of cells or pathogens, C3b binds more factor B, and this binding gradually amplifies the activation cascade. The binding of properdin stabilizes the C3bBb complex, the C3 convertase of the alternative pathway C3 convertases generated through various pathways cleave C3 to C3a and C3b. C3b contributes to the formation of the C5 convertase which cleaves C5 to C5a and C5b. Complement activation can take place at the cell surface when the antigen is a present on the cell surface and immune complexes are form there C1q gets attached and all the active cascade components assemble there.The activation takes place at the fluid phase by the circulating immune complexes and triggers are present in the fluids.

Whatever be the pathway of activation, the component (b) assembles sequentially as the cascade enzymes where as the component (a) are released out. Further, C3 is the central complement component where all the pathways merge leading to the formation of the Cb5b, C6, C7, C8andC9(n), the membrane attack complex (MAC) or Terminal complement complex.

and the C5b-9 complex are complement effectors that contribute to inflammation [3-5]

3.3 Physiological functions and inflammatory mechanisms of complement peptides.

It had long been known that C3b, C4b are opsonins that adhere to the phagocyte surface facilitating the phagocytosis. C3a, C5a are chemotactic and anaphylactic peptides. The membrane attack complex makes a hole on the cell membrane leading to cell lysis. Currently, a close interaction of complement componentC1q, C3a and C5a with inflamosomes and downstream signaling of inflammatory pathways along with activation of NF kappa had been documented.

During an inflammatory flare (e.g., in sepsis or arthritis), IL-1β and other cytokines can increase complement synthesis, ensuring abundant substrate for ongoing complement activation. This creates an inflammatory feed-forward loop in which complement activation products promote inflammasome-driven cytokine release, and those cytokines, in turn, increase local complement synthesis, fueling further inflammation and tissue damage [6–8].

A detailed account or analysis of these pathways and resultant release of inflammatory molecules like generation of ROS and inflammatory cytokines have limited scope here in this review. Summarily all the active complement fragments especially C5a, C3a and MAC make the complement system as the key mediators of inflammation and can be trememdously injurious to the self-tissues by their bystanders effects if activation goes unabated as is the3 case of many inflammatory disorders.

Whatever the mechanism of activation, the components of complement are often deposited on the surface of endothelial cells. Although, inflammation is now identified as the key mechanism involved in endothelial dysfunctions and atherosclerosis, the complex interplay of complement and other inflammatory molecules is better understood in ischaemia reperfusion injury. The endothelium, together with the underlying basement membrane, builds a vascular barrier, which has particular

significance for the pathogenesis of I/R injury [7]. It has been postulated that natural antibodies reacting with antigen epitopes that are exposed after damage to endothelial cells play an important role in the activation of complement during I/R injury [9] iC3b, formed by the proteolytic cleavage of C3b deposited on the endothelial surface, acts as a ligand for ß2 integrin CD11b/CD18 (CR3). This adhesion molecule is up-regulated in activated leukocytes and contributes to the accumulation of these cells in the extravascular compartment. The deposition of several complement proteins on the surface of endothelium ultimately leads to the formation of terminal complement complex, which, when incorporated into the cellular membrane, activates endothelial nuclear factor B. Thereby, it increases the transcription and expression of several adhesion molecules, including vascular cell adhesion molecule-1, intracellular adhesion molecule-1, and E- and P-selectins[10]Terminal complement complex also stimulates the endothelium to produce IL-8 and monocyte chemoattractant protein-1 and directly affects vascular tone by inhibiting endothelium-dependent relaxation and decreasing endothelial cyclic guanosine monophosphate(10). Terminal complement complex also induces the activation of platelets, contributing to platelet-leukocyte aggregation [12] Anaphylatoxins C3a and C5a activate endothelial cells and leukocytes. C5a, more potent as an inflammatory mediator than C3a, up-regulates the expression of adhesion molecules on endothelium and induces the release of various cytokines, including IL-1, IL-6, monocyte chemoattractant protein-1, and TNF-alpha[11]C5a is also a strong chemoattractant, contributing to the accumulation of inflammatory cells at the site of injury[12-18].Thus, several mechanisms used by complement to orchestrate the events of acute inflammation are also important for the pathogenesis of I/R injury.

Endothelial cells also represent one of the extrahepatic sources of complement components and regulators, and, because of the strategic position along the surface of the vessel wall, they may supply both the circulating blood and the extravascular fluids with these proteins. Most of the information on the production of complement components by endothelial cells has been obtained using human umbilical vein endothelial cells (HUVECs)[19]

Therapeutic blocking of anaphylactic peptides, though were successful in animal models of CAD and 1/R injuries, in humans results were of mixed nature.) It might partially be due to lack of insight into the overall dynamics and interlinks of complement proteins in CHD in human subjects.

Complement regulatory proteins protect the host against complement mediated tissue damage. Membrane-bound complement regulatory proteins are ubiquitously expressed throughout the cells of the hematopoietic system and protect host cells from complement-mediated lysis. The balance between acceleration and inhibition of complement activation is critical to whether complement activation leads to host defense or tissue injury of host organs. [20]

A large number of complement regulatory proteins are known of which CR1, DAF, MCP, MIRL and CD59 are membrane bound. Soluble form of these membranes bound complement regulatory proteins are also known.

Recent investigations on complement regulatory proteins have unveiled their role in the pathophysiology of chronic inflammatory diseases. Simultaneously, their significance as disease activity markers and therapeutic targets is being realized. Studies however, remained more focussed to autoimmune disorders and cancers. [3]

Mishra etal. Demonstrated very significant association of Leukocyte CD59 with severity of CAD along with modulation of Leukocyte surface C-Regs.[21]

Discussion

An increasing body of evidence supports a functional role for complement activation in the pathogenesis of CVD not only by generation of inflammatory complement activation product rendering their classically known effects, namely immune adhesion, opsonisation, anaphylaxis, chemotaxis along with direct cellular damage by the membrane attack complex but, predominantly through pleiotropic effects on endothelial and haematopoietic cell function and haemostasis. The complement anaphylatoxins C5a and C3a bind to their G-protein–coupled receptors (GPCRs), C5aR1/C5aR2 and C3aR, on immune cells. They are extremely potent inflammatory mediators, regulating a wide range of immune and non-immune functions such as proinflammatory cytokine production, vasodilation, histamine release, chemoattraction, and tissue regeneration (3). By binding to GPCRs, they trigger

signaling cascades such as PI3K–Akt, p38 MAPK, and calcium mobilization, ultimately leading to the activation of NF-κB and other pathways that promote inflammation and immune responses (83, 84). This can serve as a priming signal for inflammasome activation and initiates a cascade that increases the production of key components such as NLRP3 protein and pro-IL-1β, thereby priming the inflammasome. It can also directly promote a second activation signal through mechanisms such as ROS production and ionic fluxes [20]). Complement component C1q, the initiator of the classical pathway, acts as a dual regulator of inflammasomes, either promoting or suppressing their activation depending on context and molecular interactions. Beyond its canonical role in recognizing immune complexes, microbes, and apoptotic cells to trigger complement, C1q directly modulates immune cell signaling and inflammasome activity.

During an inflammatory flare (e.g., in sepsis or arthritis), IL-1β and other cytokines can increase complement synthesis, ensuring abundant substrate for ongoing complement activation. This creates an inflammatory feed-forward loop in which complement activation products promote inflammasome-driven cytokine release, and those cytokines, in turn, increase local complement synthesis, fueling further inflammation and tissue damage.

In addition, C3a, C5a plays a central role in inflammasome regulation in response to sterile danger signals, particularly crystalline materials. Cholesterol crystals (CC), MSU, and calcium phosphate crystals activate complement pathways, leading to C5a generation [22-23]. These crystals, which are classical NLRP3 activators through lysosomal damage and ROS production, show enhanced inflammasome activation in the presence of C5a. Samstad et al. demonstrated that CC activate both the classical and alternative complement pathways, with C5a enhancing IL-1β release and ROS generation in a complement-dependent manner, and identified CR3 as the receptor mediating CC uptake [24]It has been shown that MSU crystals induce C5a–C5aR1–dependent IL-1β production in whole blood via caspase-1 activation, K+ efflux, intracellular Ca2+ flux, and cathepsin B activity (86). Moreover, mitochondrial C5aR1 signaling has been linked to increased ROS generation and IL-1β gene expression, reinforcing a feed-forward loop of inflammasome activation. Recently, a study brought new evidence that complement activation and NLRP3 inflammasome signaling work together to cause cytokine release syndrome after CAR-T cell therapy [25].] Significance of complement proteins as disease activity marker and exploration of complement inhibition either by monoclonal antibodies or peptide inhibitors is going on along with some formulations in market, their success in CAD is yet to be realized.

Conclusion

Complement and complement regulatory proteins play a crucial role in the immunoinflammatory mechanisms of CAD. New information is mounting progressively that needs to be explored into depth and detail. Translations implications of the knowledge body presented is very promising in searching out new complement-based biomarkers and therapeutics.

Conflict of Interest: None

Ethical clearance. This paper work did not require an ethical clearance.

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References

1. Sattar, N. "Atherosclerotic Cardiovascular Risk Before and After Type 2 Diabetes Onset and the Roles of Ectopic Fat and Ethnic Variation: The 2025 Edwin Bierman Award Lecture." Diabetes (American Diabetes Association, published online/print 2025–2026).

2. Singh A, Bind MK, Sinha N etal. (2026) The Role of Inflammatory Markers in Coronary Artery Disease Severity: Insights from a High vs. Low Inflammation, Group EurJ Cardiovas. med.; Volume 14: 1368 – 1373

3. Nibhriti Das, (2015) Complement and membrane-bound complement regulatory proteins as biomarkers and therapeutic targets for autoimmune -inflammatory disorders, RA and SLE. Ind.J. Exp. Biol. Vol 53 :701-713

4. Das N, Srivastava N, Panchamurthy G. and Srivastava, LM (1988) Circulating immune complexes in myocardial infaraction. Ind. J. Med. Res. Vol 36: 74-75.

5. Janžič L, Kouter K (2026) Complement and inflammasome crosstalk in chronic inflammation. Frontiers in Immunology Volume 17 - article citation number: 1778759 2026 |

6. Rallidis LS, Zolindaki MG, Manioudaki HS, Laoutaris NP,etal. (2002) Prognostic value of C-reactive protein, fibrinogen, interleukin-6, and macrophage colony stimulating factor in severe unstable angina. Clin Cardiol. Vol 25: 505-510.

7. Ronald P. T, Margaret A. Lindorfer (2018) Mechanisms of Complement-Mediated Damage in Hematological Disorders. Science Direct Seminars in Hematology:118-123

8. Cortes C, Desler C, Mazzoli A, Chen JY, Ferreira VP (2022). The role of properdin and Factor H in disease. Adv Immunol. Vol 153:1-90.

9. Nesargikar P N, Spiller B, Chavez R (2012). The complement system: history, pathways, cascade and inhibitors Eur J Microbiol Immunol (Bp) Vol 13: 103–111.

10. YaoJ, Sterling K, Wang ZZ, hang Y, Song W (2024) The role of inflammasomes in human diseases and their potential as therapeutic targets. Signal Transduct Target Ther. 9:10. doi: 10.1038/s41392-023-01687-y

11. BultéD, Rigamonti C, Romano A, Mortellaro A. (2023) Inflammasomes: mechanisms of action and involvement in human diseases. Cells. Vol 12:1766 doi: 10.3390/cells12131766

12. Dubey SR, Turnbull C, Pandey A, Zhao A, Kurera MAl, Zidan R et al. (2025) Molecular mechanisms and regulation of inflammasome activation and signaling: sensing of pathogens and damage molecular patterns. Cell Mol Immunol. Vol 22:1313–44.

13. Vella R Panci D Carini F Malta G Vieni S David S et al. (2025) Cytokines in sepsis: a critical review of the literature on systemic inflammation and multiple organ dysfunction. Front Immunol. Vol 16:1682306.

14. Moon MR, Parikh AA, Pritts TA., Fischer JE, Cottongim, S et al. (1999) Complement component C3 production in IL-1beta-stimulated human intestinal epithelial cells is blocked by NF-kappaB inhibitors and by transfection with ser 32/36 mutant Ikappa Balpha. J Surg Res. Vol 82:48–55.

15. West EE, Kemper C. (2023) Complosome — the intracellular complement system. Nat Rev Nephrol. Vol 19, 426–39.

16. Dobó J, Kocsis A, Farkas B, Demeter F, Cervenak L, Gál P. (2024) The lectin pathway of the complement system-activation, regulation, disease connections and interplay with other (Proteolytic) systems. Int J Mol Sci Vol 25 :1566. doi: 10.3390/ijms25031566

17. Markiewski M M, Lambris JD (2007) The Role of Complement in Inflammatory Diseases From Behind the Scenes into the Spotlight Biological Perspectives. Am J Pathol. Vol 171, 715–727.

18. Kim, D D, Song WC (2006) Membrane complement regulatory proteins. Clin. Immunol. Vol 118: 127-136

19. Laufer J, Katz y, Passwell JH (2001) Extrahepatic synthesis of complement proteins in inflammation. Molecular Immunology Vol 38: 221-222.

20. Mastellos DC, Hajishengallis G, Lambris JD. (2024) A guide to complement biology, pathology and therapeutic opportunity. Nat Rev Immunol Vol 24:118–141.

21. Mishra N, Mohata M, Narang R, Lakshmy R, Hazarika A, Pandey RM, Das N and Luthra K (2019) Altered Expression of Complement Regulatory Proteins CD35, CD46, CD55, and

CD59 on Leukocyte Subsets in Individuals Suffering From Coronary Artery Disease. Front. Immunol, Vol 10:2072. doi: 10.3389/fimmu.2019.02072

22. Samstad E O Niyonzima N Nymo S A une MHRyan LBakkeSSet al. (2014) Cholesterol crystals induce complement-dependent inflammasome activation and cytokine release. J Immunol. Vol 192 :2837–45.

23. Niyonzima N BakkeSS Gregersen I etal. 2020 Cholesterol crystals use complement to increase NLRP3 signaling pathways in coronary and carotid atherosclerosis. EBioMedicine. Vol 60:102985. doi: 10.1016/j.ebiom.2020.102985

24. Wierzbicka D, Thet Chinamoorthy K, Jaromir T.Ratajczak M. (2025) New evidence that complement activation and NLRP3 inflammasome signaling work together to cause cytokine release syndrome after CAR-T cell therapy. Blood. Vol 146:2328. doi: 10.1182/blood-2025-2328

25. Speidl WS· (2011) Complement in atherosclerosis: friend or foe? J Thromb Haemost. (2011) Vol 9:428–40.

26. Kiss MG and Binder CJ. (2022) The multifaceted impact of complement on atherosclerosis. Atherosclerosis Vol 351:29–40.

R11

CARDIOMETABOLIC DISEASE – WHAT'S NEW

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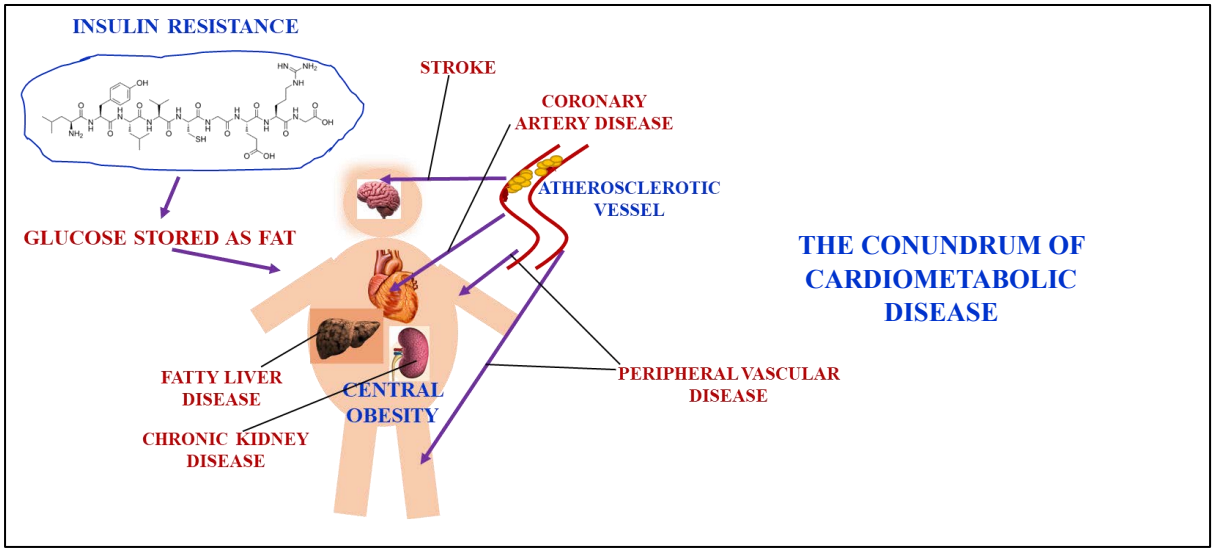
When the human body was envisaged by nature, each organ system was made in such a way as to not only co-exist with the other systems, but also they were made interdependent. Thus, every deviations to overcome a defect in another system and continue a mutually symbiotic existence with each other. Then how can we separate cardiac disease from liver disease or kidney disease or brain disease? Thus far, each organ system has been addressed in isolation, but today we are realising what we considered as cardiovascular disease (CVD), or diabetes, or stroke or fatty liver disease or even chronic kidney disease are all so interconnected in their occurrence that we have been forced to give it all a new name - cardiometabolic disease. Insulin resistance to hyperglycaemia to insulin resistance to altered handling of glycogen and its excessive deposition in various organs to plaque formation to CVD and stroke; also from hyperglycaemia to renal damage and ultimately chronic kidney disease. This is the conundrum of cardio metabolic syndrome.

And atherosclerosis is a process that sits right in the middle of many crossing pathways!

Being the pathologic driver of CVD which is responsible for the maximum deaths worldwide, atherosclerosis is the prime villain amongst all pathological processes. More than a century of research has led to current knowledge which has enabled the institution of many corrective therapies and lifestyle changes, but we are far from the ultimate solution.

The best solutions always start with prevention, then go on to early detection and finally to treatment or correction. So we need to catch it before it insidiously rears its head and becomes so difficult to eradicate. We must continue the search for a marker, or a combination of markers, that will enable very early detection or one that will predict atherosclerosis.

Now, we are faced with the conglomerate of cardiometabolic disease (CMD) and need a composite solution – one that will predict insulin resistance as well as atherosclerosis. Maybe, now that we have recognized the problem better, we are better enabled to find a solution.



ISAR DCCON 2025 CONFERENCE: HIGHLIGHTS

The Annual Conference of the Indian Society for Atherosclerosis Research, Delhi Chapter (ISAR-DCCON 2025) was organized by the Department of Biochemistry, ABVIMS & Dr RML Hospital, on Saturday, 1st November 2025 at Amaltas Hall, Civil Services Officers Institute (CSOI), KG Marg, New Delhi. The Organizing Secretary was Dr Parul Goyal, Department of Biochemistry, ABVIMS & Dr RML Hospital, and Vice President, DC-ISAR. Dr Smita Roy and Dr Neera Sharma were the Organizing Chairpersons. It was a full day conference that was well attended by more than 200 delegates, which included students, scientists and faculty from various fields of medicine from Delhi & NCR.

The inauguration ceremony was graced by the Chief Guest, Dr (Prof.) Ashok Kumar, Director, Atal Bihari Vajpayee Institute of Medical Sciences & Dr Ram Manohar Lohia Hospital, New Delhi, and the Guest of Honor, Dr S.C. Manchanda, former Professor and Head, Cardiology, AIIMS, New Delhi, and retired Consultant, Cardiology, Sir Ganga Ram Hospital. The conference had a very relevant theme, “Changing Face of Coronary Artery Disease – The Young at Risk”, and eminent speakers from Delhi and abroad were invited to deliver lectures on pertinent topics.

The scientific programme of the conference included the following lectures:

1. **Unravelling age-diet interactions regulating atherosclerosis susceptibility: novel precision nutrition strategies for cardiovascular disease prevention** by Prof. Rajendra K. Tangirala, Division of Clinical Chemistry, Department of Laboratory Medicine, Karolinska Institute, Stockholm, Sweden.

2. **CAD in the young – How early, how often and why?** By Dr Safal, Professor, Department of Cardiology, G.B. Pant Hospital, New Delhi.

3. **Lipoprotein (a) – Neglected risk marker for ASCVD** by Dr J.P.S. Sawhney, Chairperson, Department of Cardiology, Sir Ganga Ram Hospital, New Delhi.

4. **Panel discussion: Stress, Speed and Sudden Death – The modern epidemic of early CAD.** The panel was moderated by Dr Piyush Jain, Professor, Department of Medicine, ABVIMS & Dr RML Hospital. The panelists were Dr Ritu Singh (Head, Department of Biochemistry, LHMC & SSK Hospital), Dr Vijay Grover (Head, Department of CTVS, ABVIMS & Dr RML Hospital), Dr R.K. Nath (Head, Department of Cardiology, ABVIMS & Dr RML Hospital), Dr Jagriti Bhatia (Professor, Department of Pharmacology, AIIMS), Dr Ajay Chauhan (Director & Professor, Department of General Medicine, ABVIMS & Dr RML Hospital) and Dr Anshita Aggarwal (Professor, Department of Endocrinology, ABVIMS & Dr RML Hospital).

The day began with oral presentations by young researchers, followed by poster display and poster evaluation. There were 26 poster presentations and 16 oral presentations in the conference. The results were declared after the judges submitted declarations of no impartiality and no conflict of interest. The best presentations were awarded first, second and third prizes. The award recipients in both the categories were as under:

ORAL PRESENTATIONS

Name and affiliation of the presenter	Award	Title of the paper
Komal Sagar Department of Biochemistry, AIIMS, New Delhi	First	Targeting CCR5 to modulate MDSCs and Tregs mediated immunosuppression in atherosclerotic disease
Pratip Jana Department of Biochemistry, LHMC & Associated Hospitals, New Delhi	Second	Comparative analysis of an insulin sensitivity surrogate marker versus HOMA indices for detecting abnormal

		cardiometabolic risk
Pratibha Sharma Institute of Human Behaviour and Allied Sciences, Delhi	Third	Comparative quantitative proteomic profiling from exosomes of non-invasive saliva, urine and blood identifying and validating early diagnostic biomarkers in patients with coronary artery disease
Divya Parashar Department of Pharmacology, Delhi Institute of Pharmaceutical Sciences and Research University, New Delhi	Third	Prospective study determining the role of angiotensin receptor neprilysin inhibitor: ARNI versus telmisartan in resistant hypertension

POSTER PRESENTATIONS

Name and affiliation of the presenter	**Award**	**Title of the paper**
Pulkit Soni Dr B.R. Ambedkar Centre for Biomedical Research (ACBR), University of Delhi, New Delhi	First	Harnessing TGF-β and PDGF signaling to drive MSC differentiation into vascular smooth muscle cells: a novel approach for cardiovascular regeneration
Mayank Deep Singh Dr B.R. Ambedkar Centre for Biomedical Research (ACBR), University of Delhi, New Delhi	Second	Identification of novel PTX3 inhibitors as therapeutic candidates for cardiovascular diseases
Lamthrila Mongzar Department of Biochemistry, ABVIMS & Dr RML Hospital, New Delhi	Second	Association of serum adiponectin and IGF-1 with atherosclerosis in polycystic ovarian syndrome
Anshul Jangra Department of Laboratory Medicine, AIIMS, New Delhi	Third	Evaluating the impact of NABL accreditation preparedness, with a focus on training, on the attitudes and performance of lab technicians
Parul Bansal Department of Biochemistry, Lady Hardinge Medical College, New Delhi	Third	Comparison of inflammatory markers (MCP-1, IFN-γ) and PON-1 enzyme levels in patients of myocardial infarction with and without family history of MI

The executive committee meeting and GBM was conducted by Dr. Harsh Vardhan Singh, Secretary ISAR Delhi Chapter. The prize distribution and valedictory function were followed by a vote of thanks and high tea, after which all the delegates dispersed.

10th ANNUAL CON RENCE

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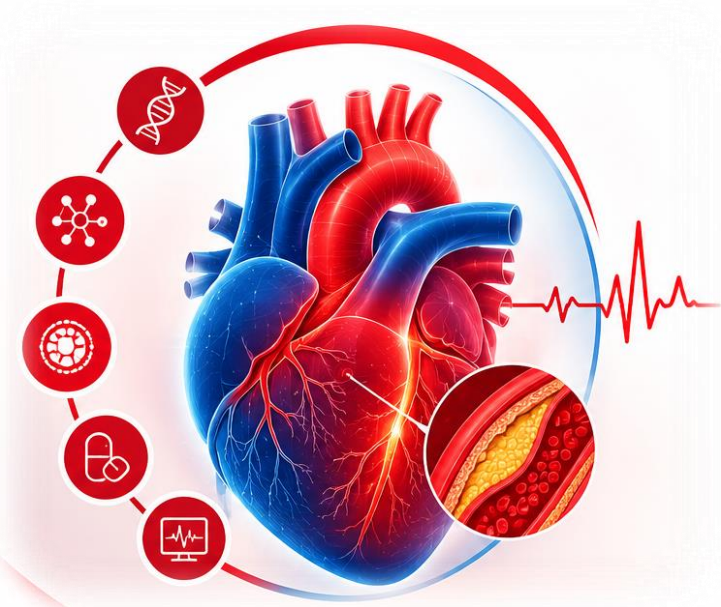
11th Annual Conference of the Indian Society for Atherosclerosis Research – Delhi Chapter

ISAR–DCCON 2026

ABSTRACT BOOK

Scientific Programme · Invited Speakers · 16 Oral · 52 Poster Abstracts



Bridging the Gap: *From Molecular Mechanisms to Precision Management of Atherosclerosis*

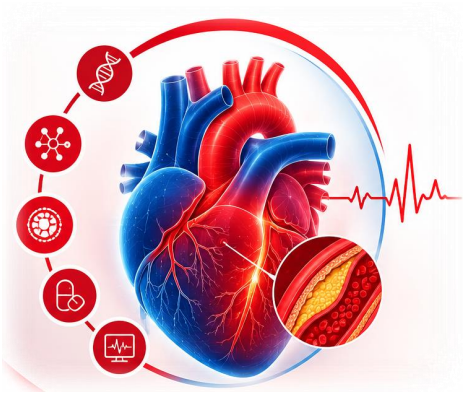
SATURDAY, 10 OCTOBER 2026 | FET AUDITORIUM, JAMIA MILLIA ISLAMIA, NEW DELHI

Organised by DDU Kaushal Kendra, Faculty of Life Sciences, Jamia Millia Islamia, with the Indian Society for Atherosclerosis Research – Delhi Chapter

SECTION I

SCIENTIFIC PROGRAMME

10 October 2026 · FET Auditorium, Jamia Millia Islamia



11th Annual Conference

ISAR–DCCON 2026

INDIAN SOCIETY FOR ATHEROSCLEROSIS RESEARCH

DELHI CHAPTER

Bridging the Gap: *From Molecular Mechanisms to* ***Precision Management*** *of Atherosclerosis*

10 OCTOBER 2026 | AUDITORIUM, FACULTY OF ENGINEERING & TECHNOLOGY, JMI, NEW DELHI

Organised by DDU Kaushal Kendra, Faculty of Life Sciences, Jamia Millia Islamia, New Delhi, with the Indian Society for Atherosclerosis Research – Delhi Chapter

SCIENTIFIC PROGRAMME AT A GLANCE

OC	10:00–10:40 AM	Opening Ceremony
PL	10:45–11:15 AM	Plenary Lecture
S1	11:30 AM–12:30 PM	Session 1 · Molecular Mechanisms
S2	12:30–1:30 PM	Session 2 · Risk to Precision Care
PS	1:30–3:00 PM	Lunch & Poster Session
S3	3:00–4:00 PM	Session 3 · Translational Frontiers
YI	4:00–5:00 PM	Young Investigators' Forum · Oral Abstracts (Two parallel sessions)
VL	5:00–5:30 PM	Vision & Guidelines Lecture Series
CL	5:30–6:00 PM	Valedictory, Awards & Induction

11:15–11:30 AM High Tea ·6:00–6:30 PM High Tea 6:30 PM ISAR–DC Executive Committee & General Body Meeting

OC — OPENING CEREMONY

10:00 – 10:40 AM | FET Auditorium, JMI

TIME	SEGMENT	PROGRAMME
10:00–10:05	INVOCATION & FELICITATION	**Recitation of the Holy Quran · Felicitation of the Hon'ble Guests**
10:05–10:10	WELCOME	**Prof. Md. Imtaiyaz Hassan** Convener, ISAR–DCCON 2026 · Director, DDU Kaushal Kendra, Jamia Millia Islamia
10:10–10:15	OVERVIEW	**Prof. Luqman A. Khan** Co-Patron, ISAR–DCCON 2026 · Dean, Faculty of Life Sciences, Jamia Millia Islamia
10:15–10:20	PRESIDENTIAL ADDRESS	**Prof. Jagriti Bhatia** President, ISAR–DC · Professor, Department of Pharmacology, AIIMS, New Delhi
10:20–10:25	INTRODUCTORY ADDRESS	**Dr. Harsh Vardhan Singh** Secretary, ISAR–DC · Senior Biochemist, North DMC Medical College & Hindu Rao Hospital, Delhi
10:25–10:30	SPECIAL ADDRESS	**Prof. Md. Mahtab Alam Rizvi** Registrar, Jamia Millia Islamia
10:30–10:35	INAUGURAL ADDRESS	**Prof. Mazhar Asif** Vice-Chancellor, Jamia Millia Islamia
10:35–10:40	VOTE OF THANKS	**Prof. Mohammad Zahid Ashraf** Coordinator, ISAR–DCCON 2026 · Department of Biotechnology, Jamia Millia Islamia

PL — PLENARY LECTURE

10:45 – 11:15 AM | FET Auditorium, JMI

CHAIRS: **Prof. Jayashree Bhattacharjee**, Former Director, VMMC, New Delhi
Prof. Ejaz Hussain, Founder Director, DDU Kaushal Kendra, Jamia Millia Islamia

TIME	FORMAT	SPEAKER & PRESENTATION
10:45–11:15	PLENARY	**Contemporary Interventional Cardiology and Precision Management of Coronary Artery Disease** **Prof. Rajiv Narang** Professor & Head, Department of Cardiology, AIIMS, New Delhi

HIGH TEA · 11:15 – 11:30 AM

S1 SESSION 1 · MOLECULAR MECHANISMS

11:30 AM – 12:30 PM | FET Auditorium, JMI

Molecular mechanisms and emerging insights in atherosclerosis

CHAIRS: **Prof. Ritu Singh**, Director Professor, Department of Biochemistry, Lady Hardinge Medical College, New Delhi

Prof. Shyamal K. Goswami, School of Life Sciences, Jawaharlal Nehru University, New Delhi

TIME	FORMAT	SPEAKER & PRESENTATION
11:30–11:50	KEYNOTE	**Phenome-India: From Population Signals to Sex-Divergent Biology** **Dr. Shantanu Sengupta** Chief Scientist, CSIR–Institute of Genomics and Integrative Biology (IGIB), New Delhi
11:50–12:10	INVITED LECTURE	**Coronary Atherosclerosis, Inflammation and Lipid Disorders: Emerging Biomarkers and Therapeutic Frontiers** **Dr. Preeti Gahlan** Department of Cardiology, Fortis Hospital, Sukhdev Vihar, New Delhi
12:10–12:30	INVITED LECTURE	**Targeting the oxLDL–LOX-1 Axis: Structure-Guided Discovery and Preclinical Evaluation of Small-Molecule LOX-1 Inhibitors** **Dr. Arockiasamy Arulandu** Group Leader, Structural Biology, International Centre for Genetic Engineering and Biotechnology (ICGEB), New Delhi

S2 SESSION 2 · RISK TO PRECISION CARE

12:30 – 1:30 PM | FET Auditorium, JMI

From cardiovascular risk to precision clinical management

CHAIRS: **Prof. S. B. Sharma**, Former Director-Professor, UCMS, Delhi

Prof. Meryam Sardar, Head, Department of Biosciences, Jamia Millia Islamia

TIME	FORMAT	SPEAKER & PRESENTATION
12:30–12:50	KEYNOTE	**From Vulnerable Plaque to Precision PCI** **Dr. Amitabh Yaduvanshi** Chief of Services & Senior Consultant, Cardiology, Holy Family Hospital, New Delhi
12:50–1:10	INVITED LECTURE	**The Genetic Landscape of Hypertriglyceridemia-Associated Genetic Variants in Patients from a Cardiology Setting** **Dr. Archna Singh** Professor, Department of Biochemistry, AIIMS, New Delhi

TIME	FORMAT	SPEAKER & PRESENTATION
1:10–1:30	SPECIAL INVITED LECTURE	**Empowering the Scientific Ecosystem: Navigating National Funding and Project Formulation at ANRF** **Dr. Balachandar Venkatesan** Senior Scientist & Member Secretary – Health Science & Ayurvedic Biology, Anusandhan National Research Foundation (ANRF)

PS

LUNCH, GBM & POSTER SESSION

1:30 – 3:00 PM | FET Auditorium, JMI

Poster viewing and evaluation

Clinical Research / Studies

CHAIRS: **Prof. S. P. Singh**, Professor & Head, Department of Physiology, Amrita School of Medicine, Faridabad

Basic Sciences Research / Studies

CHAIRS: **Prof. Mohammad Mahfuzul Haque**, Department of Biotechnology, Jamia Millia Islamia

Poster judges: confidential — not printed

S3

SESSION 3 · TRANSLATIONAL FRONTIERS

3:00 – 4:00 PM | FET Auditorium, JMI

Translational research and new frontiers in atherosclerosis

CHAIRS: **Prof. Seema Bhargava**, Chairperson, Department of Biochemistry, Sir Ganga Ram Hospital; President-Elect, ISAR–DC

Prof. Ehteshamul Haque, Head, Department of Biochemistry, School of Pharmacy, Jamia Hamdard

TIME	FORMAT	SPEAKER & PRESENTATION
3:00–3:20	KEYNOTE	**Perspectives on Molecular and Precision Therapeutic Targets of Atherosclerosis** **Dr. Rajendra K. Tangirala** Division of Clinical Chemistry, Department of Laboratory Medicine, Karolinska Institutet, Stockholm, Sweden · Gut Leben Inc., San Diego, USA
3:20–3:40	INVITED LECTURE	**When Vessels Fail: From Vulnerable Plaques to Hibernating Hearts and Ageing Tissues** **Dr. Saran Kumar** Assistant Professor, Kusuma School of Biological Sciences, IIT Delhi
3:40–4:00	INVITED LECTURE	**Modeling Pathological Ocular Neovascularization in Zebrafish for Mechanistic Studies and Therapeutic Screening** **Dr. Rudra Nayan Das** Assistant Professor, Department of Life Sciences, Shiv Nadar University, Delhi-NCR

YI

YOUNG INVESTIGATORS' FORUM

4:00 – 5:00 PM | FET Auditorium & Lecture Hall, DDU Kaushal Kendra

Oral abstract presentations · competing for the Best Oral Presentation Award · two parallel sessions

Clinical Research / Studies · Lecture Hall, DDU Kaushal Kendra

CHAIRS: **Prof. Parul Goyal**, Director Professor, Department of Biochemistry, ABVIMS & Dr. RML Hospital; Vice President, ISAR–DC

Prof. Rachna Agarwal, Professor & Head, Department of Biochemistry, IHBAS, Delhi; Executive Committee Member, ISAR–DC

Basic Sciences Research / Studies · FET Auditorium (Main Hall)

CHAIRS: **Prof. Kapil Dev**, Head, Department of Biotechnology, Jamia Millia Islamia

Dr. Aastha Mishra, Principal Scientist, CSIR–Institute of Genomics and Integrative Biology, New Delhi

Oral judges: confidential — not printed

TIME	FORMAT	TRACK & VENUE
4:00–5:00	ORAL PRESENTATIONS	**Clinical Research / Studies** Lecture Hall, DDU Kaushal Kendra
4:00–5:00	ORAL PRESENTATIONS	**Basic Sciences Research / Studies** FET Auditorium (Main Hall)

VL

VISION & GUIDELINES LECTURE SERIES

5:00 – 5:30 PM | FET Auditorium, JMI

Perspectives from senior members of the Society

CHAIRS: **Prof. D. K. Srivastava**, Patron, ISAR–DC · Professor of Biochemistry, HIMSR, Jamia Hamdard, New Delhi

Prof. Nibhriti Das, Former Professor, Department of Biochemistry, AIIMS, New Delhi

Prof. Anita Bhatia, Director Professor & Head, Department of Biochemistry, Lady Hardinge Medical College, New Delhi

TIME	FORMAT	SPEAKER & PRESENTATION
5:00–5:10	VISION LECTURE	**Lipoprotein(a) as a Causal Risk Factor for CAD: From Molecular Mechanisms to Targeted Treatment Modalities — 60 Years: Still an Unresolved Enigma** **Prof. Jasvinder K. Gambhir** Former Senior Professor & Head, Department of Biochemistry, UCMS, Delhi
5:10–5:20	VISION LECTURE	**Why Dyslipidemia among Indians Must Be Treated Differently than the Western Population?** **Prof. Harmeet Singh Rehan** Director Professor & Head, Department of Pharmacology, Lady Hardinge Medical College, New Delhi
5:20–5:30	VISION LECTURE	**Beyond Barker's Hypothesis to Precision Prevention** **Prof. Shridhar Dwivedi** Patron, ISAR–DC · Senior Consultant Cardiologist, National Heart Institute, New Delhi

CL — VALEDICTORY, AWARDS & INDUCTION

5:30 – 6:00 PM | FET Auditorium, JMI

CHAIRS: **Dr. Harlokesh Narayan Yadav**, Professor, AIIMS, New Delhi; Joint Secretary, ISAR–DC

Dr. Rajeev Goyal, Professor, Department of Biochemistry, LHMC, New Delhi; Treasurer, ISAR–DC

Prof. Majumi M. Noohu, Director, Centre for Physiotherapy and Rehabilitation Sciences, Jamia Millia Islamia

Prof. Rajan Patel, Director, Centre for Interdisciplinary Research in Basic Sciences, Jamia Millia Islamia

TIME	SEGMENT	PROGRAMME
5:30–5:40	VALEDICTORY REMARKS	**Prof. Bhuvnesh Kumar** Dean Research, Sharda University; Former Director, DIPAS–DRDO
5:40–5:50	AWARDS	**Awards & Prize Distribution** Best oral and poster presentation awards · certificates · acknowledgements
5:50–5:55	INDUCTION CEREMONY	**Induction of New Office Bearers of ISAR–DC**
5:55–6:00	CONCLUDING REMARKS	**Prof. Tanuja** Dean, Academic Affairs, Jamia Millia Islamia

HIGH TEA · 6:00 – 6:30 PM

ISAR–DC EXECUTIVE COMMITTEE & GENERAL BODY MEETING · 6:30 PM

LOCAL ORGANISING COMMITTEE

Chief Patron	Prof. Mazhar Asif, Vice-Chancellor, Jamia Millia Islamia
Patron	Prof. Md. Mahtab Alam Rizvi, Registrar, Jamia Millia Islamia
Co-Patron	Prof. Luqman A. Khan, Dean, Faculty of Life Sciences, Jamia Millia Islamia
Convener	Prof. Md. Imtaiyaz Hassan, Director, DDU Kaushal Kendra, Jamia Millia Islamia
Coordinator	Prof. Mohammad Zahid Ashraf, Department of Biotechnology, Jamia Millia Islamia
Organising Team	Faculty members, DDU Kaushal Kendra: Dr. Amena Mahmood · Dr. Sheikh Tousif Reza · Dr. Anam Aseem · Dr. Aaliya Taiyab · Dr. Sobia Zaidi · Dr. Moquitol Haque · Dr. Mohammad Misbah

Advisory Committee	Prof. Luqman A. Khan — Dean, Faculty of Life Sciences Prof. Meryam Sardar — Head, Department of Biosciences Prof. Kapil Dev — Head, Department of Biotechnology Prof. Rajan Patel — Director, Centre for Interdisciplinary Research in Basic Sciences (CIRBSc) Prof. Majumi M. Noohu — Director, Centre for Physiotherapy and Rehabilitation Sciences (CPRS) Prof. Mohammad Husain — Director, Multidisciplinary Centre for Advanced Research and Studies Prof. Syed Naqui Kazim — Hony. Director, Unani Medicine & Allied Sciences
Scientific Committee	Prof. Mohammad Mahfuzul Haque · Prof. Aman Jairajpuri · Prof. Mushahid Rizvi · Prof. Kazi Rizwanul Haque · Prof. Nikhat Manzoor · Prof. Mohammad Abid · Prof. Nida Jamal · Dr. Mohammad Mohsin · · Prof. Asimul Islam · Prof. Shama Parveen · Prof. Raven · Prof. Zubaida Ansari · Prof. Fareeda · Prof. Meetu Gupta · Prof. Abdur Rab · Dr. Syed Mansoor Ali · Dr. Sadaf Fatima

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SECTION II

INVITED SPEAKERS

Plenary · Keynote · Invited · Vision Lectures

PLENARY LECTURE **Plenary Lecture · 10:45–1:15 AM**

Contemporary Interventional Cardiology and Precision Management of Coronary Artery Disease

Prof. Rajiv Narang

Professor & Head, Department of Cardiology, All India Institute of Medical Sciences (AIIMS), New Delhi

ABSTRACT

Summary prepared by the organising committee.

Coronary artery disease (CAD) remains the leading cause of death in India, where it presents at a younger age and often follows a more aggressive course than in Western populations. Over the past two decades, interventional cardiology has moved from angiography-guided treatment of stenoses towards a physiology- and imaging-informed approach to the whole patient. Invasive physiology helps decide which lesions merit revascularisation, while intravascular ultrasound and optical coherence tomography optimise stent sizing, expansion and apposition and reduce stent failure in complex lesions. Contemporary drug-eluting stents, drug-coated balloons and calcium-modification tools such as intravascular lithotripsy have widened the range of patients who can be treated safely by percutaneous coronary intervention (PCI), including those with left main, bifurcation and heavily calcified disease.

Equally important is what follows the procedure: antiplatelet therapy tailored to ischaemic and bleeding risk, intensive lipid lowering to guideline LDL-cholesterol targets, and attention to lipoprotein(a), diabetes and lifestyle. This plenary will review how these advances come together in the precision management of CAD — from risk stratification and the choice between medical therapy, PCI and surgery, to individualised secondary prevention — and will consider the challenges of applying them in India's high-volume, resource-diverse settings.

Keywords: *Coronary artery disease; Percutaneous coronary intervention; Intravascular imaging; Coronary physiology; Secondary prevention*

BIOSKETCH

Prof. Rajiv Narang is Professor and Head of the Department of Cardiology at the All India Institute of Medical Sciences (AIIMS), New Delhi. An interventional cardiologist, his clinical and academic interests include coronary artery disease, coronary intervention and the delivery of cardiovascular care in resource-diverse, developing-world settings. The department he leads offers coronary angioplasty, balloon valvuloplasty, device closure of congenital heart defects and electrophysiology services, and trains DM cardiology residents from across the country.

SESSION 1 · MOLECULAR MECHANISMS **Keynote · 11:30–11:50 AM**

Phenome-India: From Population Signals to Sex-Divergent Biology

Dr. Shantanu Sengupta

Chief Scientist, CSIR–Institute of Genomics and Integrative Biology (IGIB), New Delhi

Email: shantanus.igib@csir.res.in

ABSTRACT

The Phenome-India (PI-CHeCK) cohort is a deeply phenotyped cohort of more than 10,000 CSIR employees, pensioners, and their spouses recruited across 37 laboratories in 24 Indian cities. Integrating dietary and multi-omic data, Phenome-India provides a unique platform to investigate how nutritional status shapes metabolic health at scale. Its balanced male and female representation (~57:43) enables investigation of sex-specific associations often missed in male-skewed cohorts. We use vitamin B12 status as a case study of how a population-level signal can be traced to underlying biology. Among 4,326 Phenome-India participants, circulating active vitamin B12 (holotranscobalamin) was inversely associated with an atherogenic lipid profile, including ApoB, LDL, VLDL, and total cholesterol, in males, whereas in females it was positively associated with ApoA1 and HDL cholesterol. This pattern extended across dozens of metabolites and biochemical markers and remained robust across sensitivity analyses. To test whether this dimorphism reflected reproducible B12-responsive biology, we developed a paternal vitamin B12-restricted Wistar rat model spanning two generations. Hepatic multi-omics in F1 offspring, never directly B12-restricted, revealed a similarly sex-divergent phenotype. Males showed hepatic fatty acid accumulation, impaired β-oxidation, PPAR-pathway rewiring, and compensatory VLDL secretion, recapitulating key features of the human male signature. Females instead showed glycolytic-to-TCA disruption and one-carbon metabolic insufficiency, with relative protection from the atherogenic lipid phenotype. Phosphatidylcholine remodeling tracked with impaired PEMT-mediated phospholipid synthesis, linking one-carbon and lipid metabolism. Together, these findings demonstrate how Phenome-India can connect population-level nutritional associations to sex-specific metabolic mechanisms and reveal how micronutrient status may shape metabolic health across generations.

Keywords: *Phenome India, Vitamin B12, Cholesterol, Rat model*

BIOSKETCH

Shantanu Sengupta is Chief Scientist at the CSIR-Institute of Genomics and Integrative Biology (CSIR-IGIB), New Delhi, where he leads research in clinical proteomics, metabolomics, and cardiovascular disease. He holds a Master's degree in Organic Chemistry from Jadavpur University and a PhD from CSIR-Indian Institute of Chemical Biology, followed by postdoctoral training at the National Institute of Immunology, New Delhi, and the Cleveland Clinic Foundation, USA.

His research focuses on integrating genetic, proteomic, and metabolomic approaches to understand cardiovascular disease, with particular emphasis on the role of vitamin B12 deficiency in coronary artery disease. He leads the Phenome-India longitudinal cohort, a flagship CSIR initiative aimed at understanding determinants of health and disease in the Indian population. He has also played a key role in establishing proteomics and metabolomics capabilities at CSIR-IGIB.

Dr. Sengupta is President of the Proteomics Society, India, and a Fellow of NASI. He is a recipient of the DBT Biosciences Award and serves on the editorial board of the Journal of Proteins and Proteomics.

SESSION 1 · MOLECULAR MECHANISMS **Invited Lecture · 11:50 AM–12:10 PM**

Coronary Atherosclerosis, Inflammation and Lipid Disorders: Emerging Biomarkers and Therapeutic Frontiers

Dr. Preeti Gahlan

Department of Cardiology, Fortis Hospital, Sukhdev Vihar, New Delhi

ABSTRACT

Coronary artery disease (CAD) remains the leading cause of cardiovascular morbidity and mortality worldwide. Despite major advances in lipid-lowering therapy, antithrombotic treatment and coronary revascularisation, recurrent adverse cardiovascular events remain common, underscoring the need for novel therapeutic strategies. Persistent inflammatory activity is now recognised as an important contributor to residual cardiovascular risk, even in patients receiving guideline-directed preventive therapy.

This talk will review biomarkers that capture the inflammatory and lipid dimensions of coronary atherosclerosis. Inflammatory biomarkers include C-reactive protein (CRP), a member of the pentraxin family; interleukin-6 (IL-6) and other cytokines; lipoprotein-associated phospholipase A2 (Lp-PLA2); fibrinogen; and D-dimer. Among lipid markers, apolipoprotein B (apoB) reflects the number of atherogenic particles rather than simply their cholesterol content, while lipoprotein(a) [Lp(a)] carries an inherited atherogenic and pro-inflammatory signal; both complement conventional measures of LDL cholesterol, total cholesterol and triglycerides. Imaging biomarkers, including computed tomography coronary angiography, assessment of plaque composition and the perivascular fat attenuation index, allow direct visualisation of plaque burden and vascular inflammation.

Therapeutic strategies span lifestyle and risk-factor control; standard guideline-directed medical therapy with statins, antiplatelet agents, angiotensin-converting enzyme (ACE) inhibitors and beta-blockers; and anti-inflammatory therapy targeting residual inflammatory risk. Emerging approaches, including epigenetic therapies and microbiome-targeted interventions, may further extend treatment options. Integrating inflammatory, lipid and imaging biomarkers offers a pathway towards more precise risk stratification and individualised management of patients with coronary atherosclerosis.

Keywords: *Coronary artery disease; Residual inflammatory risk; Biomarkers; Lipoprotein(a); Anti-inflammatory therapy*

BIOSKETCH

Dr. Preeti Gahlan is a cardiologist with interests in structural heart disease interventions, coronary artery disease and cardiac imaging. She has undertaken fellowship training in structural heart disease interventions. Her publications include a study of the clinical predictors of coronary artery ectasia (Angiology, 2023), reports on echocardiographic diagnosis in valvular and aortic-root disease (Journal of Invasive Cardiology, 2020; Journal of Cardiovascular Imaging, 2020), and transcatheter endovascular management of complex coarctation of the aorta in adults (JACC: Case Reports, 2025).

SESSION 1 · MOLECULAR MECHANISMS **Invited Lecture · 12:10–12:30 PM**

Targeting the oxLDL–LOX-1 Axis: Structure-Guided Discovery and Preclinical Evaluation of Small-Molecule LOX-1 Inhibitors

Dr. Arockiasamy Arulandu

Group Leader, Structural Biology Group, International Centre for Genetic Engineering and Biotechnology (ICGEB), New Delhi

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ABSTRACT

Despite advances in lipid-lowering therapy, substantial residual cardiovascular risk persists in patients with atherosclerotic cardiovascular disease (ASCVD). Oxidized low-density lipoprotein (oxLDL) and its vascular receptor, lectin-like oxidized LDL receptor-1 (LOX-1), contribute to endothelial dysfunction, oxidative stress, inflammation, and atherosclerosis progression. Targeting this axis may therefore offer a complementary approach to addressing disease mechanisms that persist despite conventional lipid lowering.

We undertook a structure-guided drug-discovery programme to identify and optimize small-molecule inhibitors of LOX-1. Our integrated approach combined structural biology, computational design, biophysical characterization, cellular assays, and an experimental animal model of atherosclerosis to evaluate target engagement, bioavailability, toxicity, and pharmacological activity. Structural and biophysical studies demonstrated direct binding of selected compounds to LOX-1 and provided insights into their mechanism of inhibition. Lead compounds inhibited LOX-1-mediated oxLDL uptake in vitro. Evaluation in an experimental model of atherosclerosis demonstrated a reduction in atherosclerotic burden, providing in vivo proof of concept for this therapeutic approach.

These findings support the therapeutic potential of small-molecule LOX-1 inhibition and warrant further preclinical development. LOX-1-directed therapeutics could complement established lipid-lowering treatments, with the longer-term aim of reducing residual cardiovascular risk.

Keywords: *LOX-1; Oxidized LDL; Structure-guided drug discovery; Small-molecule inhibitors; Residual cardiovascular risk*

BIOSKETCH

Dr. Arockiasamy Arulandu is Group Leader of Structural Biology at ICGEB, New Delhi. His group focuses on structure-guided preclinical drug discovery for cardiovascular disease and cancer, integrating structural biology, computational design, and experimental validation in vitro and in vivo. A major focus of his current research is the development of small-molecule inhibitors targeting the oxLDL–LOX-1 axis for the treatment of atherosclerotic cardiovascular disease.

SESSION 2 · RISK TO PRECISION CARE **Keynote · 12:30–12:50 PM**

From Vulnerable Plaque to Precision PCI

Dr. Amitabh Yaduvanshi

Chief of Services & Senior Consultant, Cardiology, Holy Family Hospital, New Delhi

ABSTRACT

Coronary atherosclerosis becomes clinically dangerous not when it narrows the lumen, but when a plaque acquires the biology of rupture. The vulnerable plaque — archetypally the thin-cap fibroatheroma, with its lipid-rich core, macrophage-infiltrated cap beneath sixty-five micrometres, and spotty microcalcification — is now understood less as a fixed morphology than as a dynamic state: an equilibrium of collagen turnover, a focus of mechanical stress where clustered microcalcification amplifies local force, and a failure of clearance in which macrophage apoptosis outpaces efferocytosis. Low endothelial shear stress completes the picture, upregulating matrix metalloproteinases and cathepsins that thin the cap from within. Crucially, this biology has become measurable in the living coronary tree. Intravascular ultrasound, optical coherence tomography, and hybrid near-infrared spectroscopy resolve burden, cap, and lipid composition; ^{18}F-sodium fluoride positron emission tomography exposes active mineralisation; and landmark studies — Lipid-Rich Plaque, PROSPECT II, and COMBINE OCT-FFR — now link these high-risk phenotypes to events with quantified, site-specific weight, even where physiology is negative. That prognostic yield converts diagnosis into decision. Imaging-guided PCI improves outcomes in complex lesions, intravascular lithotripsy and drug-coated balloons extend the interventional repertoire, and the PREVENT trial offers the first randomised evidence that a vulnerable plaque can be preempted before it ruptures. Molecular insight and clinical intervention are thus not separate disciplines but a single continuous chain — from mechanism, through detection and risk adjudication, to tailored intervention — reframing atherosclerosis as a disease to be measured, stratified, and, increasingly, preempted rather than simply stented.

Keywords: *Vulnerable plaque; Thin-cap fibroatheroma; Intravascular imaging; Imaging-guided PCI; Plaque preemption*

BIOSKETCH

Dr. Amitabh Yaduvanshi is Chief of Services and Senior Consultant in Cardiology at Holy Family Hospital, New Delhi, and an Honorary Professor. He obtained his MBBS and MD (Medicine) from Maulana Azad Medical College, New Delhi, and his DM in Cardiology from G.B. Pant Hospital, New Delhi. In more than two decades of practice he has performed over 7,500 coronary angioplasties, 5,000 electrophysiology procedures and 1,000 pacemaker and AICD/CRT-D implants. His interests include complex coronary intervention, electrophysiology and device therapy, structural heart interventions, mechanical circulatory support and renal denervation for resistant hypertension. He is a Fellow of the American College of Cardiology (FACC), the Society for Cardiovascular Angiography and Interventions (FSCAI), FICC and FICN, a member of the Asia Pacific Heart Rhythm Society, and has 30 publications in national and international journals.

SESSION 2 · RISK TO PRECISION CARE **Invited Lecture · 12:50–1:10 PM**

The Genetic Landscape of Hypertriglyceridemia-Associated Genetic Variants in Patients from a Cardiology Setting

Dr. Archna Singh

Professor, Department of Biochemistry, AIIMS, New Delhi

Authors: Archna Singh, Aanchal Singh, Ashikh Seethy, Satyavir Yadav, Ethayathulla AS, Ambuj Roy

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ABSTRACT

Triglycerides (TGs) are the primary energy-storage molecules in mammals. Impaired TG metabolism causes hypertriglyceridemia (HTG), defined as fasting serum TG >150 mg/dL. HTG is highly prevalent in India (~30% of adults). Moderate HTG increases Atherosclerotic Cardiovascular Disease (ASCVD) risk. Besides classical TG genes (GPIHBP1, LPL, APOC2), non-canonical genes like SMPD1, PLIN1, and CREB3L3 may affect TG metabolism via indirect pathways. Limited data on the genetic background of HTG is available from the Indian population.

The study aims to evaluate variant presence and frequency in male HTG patients using Whole Exome Sequencing and predict structural and functional consequences using in-silico approaches. WES was performed on an Illumina platform on samples from 61 males with primary HTG. Analysis was done using a standardized bioinformatics pipeline and ACMG classification to classify them as Pathogenic/Likely Pathogenic (P/LP), Variants of Uncertain Significance (VUS) and Benign/Likely Benign (B/LB). In-silico protein structure prediction was performed using ColabFold and ChimeraX.

The WES analysis identified 128 variants across 84 canonical and non-canonical lipid-metabolism genes including 11 P/LP (e.g., GPIHBP1, CREB3L3), 49 VUS, and 68 LB. Among these, 17 were novel (5 LP, 6 VUS, 6 LB). In-silico modeling of a pathogenic GPIHBP1 frameshift variant revealed complete loss of the C-terminal GPI-anchoring site, predicting a loss of LPL-anchoring function as a possible loss-of-function mechanism. Thermodynamic analysis of missense variants in SMPD1 (sphingomyelin phosphodiesterase 1), a non-canonical sphingolipid metabolism gene, predicted destabilization. We observed multiple HTG-associated variants. These may contribute to HTG pathogenesis through mechanisms beyond classical LPL-pathway dysfunction.

Keywords: *Hypertriglyceridemia, whole exome sequencing, Coronary artery disease*

BIOSKETCH

Dr. Archna Singh is Professor of Biochemistry at AIIMS, New Delhi. She holds an MBBS from Nalanda Medical College, an MD and PhD in Biochemistry from AIIMS, and an MPH from the University of Michigan. She has worked at AIIMS since 2001, rising through successive academic ranks. Her research links lipid metabolism, nutrition and public health. As PI and Co-PI she has drawn more than ₹5 crore in funding over the past decade, including a SERB project on fatty acid metabolism in paediatric B-cell leukemias, DBT support on insulin sensitivity in obesity, and ICMR and India Alliance grants on nutrition in tribal communities. She has published over 75 papers in journals including Mol Cell Biochem, Journal of Clinical Lipidology and Frontiers in Oncology. Her work covers lipid metabolism and mitochondrial changes in pediatric leukemia, anti-inflammatory diets in coronary artery disease, and indigenous foods in Jharkhand, Madhya Pradesh and Meghalaya.

SESSION 2 · RISK TO PRECISION CARE **Special Invited Lecture · 1:10–1:30 PM**

Empowering the Scientific Ecosystem: Navigating National Funding and Project Formulation at ANRF

Dr. Balachandar Venkatesan

Senior Scientist & Member Secretary – Health Science & Ayurvedic Biology, Anusandhan National Research Foundation (ANRF)

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ABSTRACT

Summary prepared by the organising committee.

The Anusandhan National Research Foundation (ANRF), established under the ANRF Act, 2023, has subsumed the Science and Engineering Research Board (SERB) and is mandated to seed, grow and promote research and innovation across India's universities, colleges, research institutions and R&D laboratories. Alongside continuing support for individual investigators, ANRF places new emphasis on mission-mode research in areas of national priority and on building research capacity in institutions that have so far had limited access to competitive funding.

This address will introduce ANRF's principal funding instruments relevant to biomedical and health scientists — including the Advanced Research Grant (which replaces the SERB Core Research Grant and supports projects of up to five years), the Prime Minister Early Career Research Grant for researchers early in their independent careers, the Mission for Advancement in High-Impact Areas (MAHA), and Partnerships for Accelerated Innovation and Research (PAIR), together with fellowship, travel and capacity-building schemes. It will offer practical guidance on project formulation: identifying the right scheme, framing a focused and fundable research question, building realistic objectives, budgets and timelines, addressing ethics and data requirements, and avoiding common reasons for rejection — with particular reference to opportunities in health sciences and Ayurvedic biology.

Keywords: *ANRF; Research funding; Grant writing; Project formulation; Research ecosystem*

BIOSKETCH

Dr. A. V. Balachandar (Balachandar Venkatesan) is a Scientist at the Anusandhan National Research Foundation (ANRF), New Delhi, where he is associated with programmes in health sciences and Ayurvedic biology, including the PM Early Career Research Grant and MAHA schemes. He obtained his PhD in Biochemistry from the University of Madras and carried out postdoctoral research on vascular remodelling and metabolic conditions at the University of Texas Health Science Center and Tulane University, USA. His research has examined IL-18/EMMPRIN signalling in cardiomyocytes and smooth muscle cells, high-glucose-induced endothelial dysfunction, and Sestrin2-mediated monocyte activation in diabetes with dyslipidaemia. He is a recipient of the Lady Tata Memorial Trust award and the DBT Rapid Grant for Young Investigators.

SESSION 3 · TRANSLATIONAL FRONTIERS **Keynote · 3:00–3:20 PM**

Perspectives on Molecular and Precision Therapeutic Targets of Atherosclerosis

Dr. Rajendra K. Tangirala

Division of Clinical Chemistry, Department of Laboratory Medicine, Karolinska Institutet, Stockholm, Sweden · Gut Leben Inc., San Diego, USA

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ABSTRACT

Atherosclerotic cardiovascular disease (ASCVD) remains the leading cause of global morbidity and mortality, driven by an intricate interplay between lipid accumulation and chronic arterial inflammation. While standard lipid-lowering therapies lower clinical event rates, substantial cardiovascular disease risks persist. Drawing from 30 years of translational molecular target and therapeutic discovery, this presentation will dissect the core molecular mechanisms driving atherosclerosis progression and critically evaluate emerging therapeutic strategies designed to actively induce atherosclerosis regression. The interconnected pathophysiological pillars of ASCVD include the beginning of how oxidative stress accelerates low-density lipoprotein (LDL) modification, intracellular cholesterol accumulation and metabolic dysregulation which trigger a pro-inflammatory cascade that destabilizes advanced plaques. Furthermore, several next-generation therapeutic avenues include novel inflammatory and metabolic gene targets, and metabolic reprogramming strategies. There needs to be a shift in the clinical and drug-development paradigm from merely stalling lesion progression to actively stimulating plaque regression. However, a key caveat is that despite rapid technological advancements, true precision therapy for atherosclerosis is not yet clinical-ready. Ultimately, integrated multi-omics based molecular target identification and next generation precision model development will provide a comprehensive roadmap for cardiologists, scientists, and industry innovators a definitive frontier for eradicating ASCVD.

Keywords: *Atherosclerosis regression; Vascular inflammation; Oxidative stress; Multi-omics; Precision therapeutics*

BIOSKETCH

Dr. Rajendra K Tangirala received his PhD from Medical College of Pennsylvania, Philadelphia PA and completed his postdoctoral research at UC San Diego School of Medicine. Later, he joined Research Faculty at University of Pennsylvania Medical Center, Philadelphia. After several years as Head of Pharmacology at X-Ceptor Therapeutics Inc in San Diego, he returned to academia. In 2004, he joined the Faculty of Division of Endocrinology, Diabetes & Hypertension at UCLA David Geffen School of Medicine, Los Angeles. Until 2015 at UCLA, Dr. Tangirala served as Director of Phenotyping and Co- Director of Inflammation Cores of the UCLA/UCSD Diabetes Endocrinology Research Center of NIH/NIDDK. Currently, he is the President and Chief Executive Officer of GUT LEBEN, INC. in San Diego with focus on innovative therapeutic approaches to target cardiometabolic diseases. He is Professor associated with Division of Clinical Chemistry at Karolinska Medical Institute, Stockholm, Sweden. Dr. Tangirala received many National/International awards including International Atherosclerosis Society Award for Scientific Excellence (Stockholm, 2000). Dr. Tangirala is a fellow of American Heart Association (AHA) and has served as Chair and Co-chair on National Research Committees of the AHA for two decades. He was a Principal Investigator of AHA and NIH- funded projects in metabolic and cardiovascular diseases. He has published many leading scientific research articles in top peer-reviewed journals and serves as editorial reviewer for many leading scientific journals. He delivered many invited scientific presentations at national and international conference/meetings.

SESSION 3 · TRANSLATIONAL FRONTIERS **Invited Lecture · 3:20–3:40 PM**

When Vessels Fail: From Vulnerable Plaques to Hibernating Hearts and Ageing Tissues

Dr. Saran Kumar

Assistant Professor, Vascular Biology and Cancer Laboratory, Kusuma School of Biological Sciences, Indian Institute of Technology Delhi

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ABSTRACT

Atherosclerosis compromises tissue oxygen supply at three scales: the vulnerable plaque that ruptures, the chronically under-perfused myocardium downstream of a stenosis, and the progressive capillary loss that accompanies ageing. I will present three mouse studies that address each scale. First, ADAMTS4, a versican- and aggrecan-cleaving metalloproteinase elevated in human plaques and in the plasma of patients with coronary artery disease, increases in plaques and plasma of ApoE−/− mice as disease progresses. Genetic ablation of Adamts4 in ApoE−/− mice reduced high-fat diet-induced plaque burden without altering plasma lipids and produced a stable plaque phenotype: less lipid and fewer macrophages, more smooth muscle cells and collagen, a thicker fibrous cap, a smaller necrotic core and a two-thirds reduction in plaque vulnerability index, together with a less inflammatory plasma cytokine profile. Second, using a heart-specific, tetracycline-regulated VEGF decoy receptor, we generated a tunable and reversible microvascular deficit that drives cardiomyocytes into hibernation without infarction. Loss of contractility tracked microvascular density and was fully restored upon revascularisation, accompanied by reversible isoform switching, alternative splicing of sarcomeric genes and recruitment of new transcriptional enhancers; prolonged hypoperfusion, however, culminated in a point of no return. Third, we found that VEGF signalling declines during ageing owing to increased production of decoy receptors. A modest, sustained rise in circulating VEGF prevented age-related capillary rarefaction, improved perfusion and extended both lifespan and healthspan, alleviating hepatic steatosis, sarcopenia, osteoporosis and inflammaging. Together, these studies identify matrix proteolysis and microvascular maintenance as targetable determinants of cardiovascular resilience and healthy ageing.

Keywords: *ADAMTS4; plaque stability; myocardial hibernation; VEGF; vascular ageing*

BIOSKETCH

Dr. Saran Kumar is an Assistant Professor at the Kusuma School of Biological Sciences, Indian Institute of Technology Delhi, where he has led the Vascular Biology and Cancer Laboratory since 2021. He trained in vascular biology at the National University of Singapore, where he studied the role of ADAMTS proteases in atherosclerosis, and at the Hebrew University–Hadassah Medical School, Jerusalem, in the laboratory of Prof. Eli Keshet, where he worked on VEGF-regulated microvascular homeostasis, myocardial hibernation and vascular ageing. His work has been published in Science and Scientific Reports, among other journals. His laboratory studies how blood vessels and their microenvironment shape tissue fate in disease, with interests spanning tumour spatial heterogeneity, vascular biology, cancer metabolism, patient-derived organoids and explants, spatial transcriptomics, CAR T-cell therapy and DNA secondary structures (i-motifs and G-quadruplexes). He collaborates closely with clinicians at AIIMS New Delhi on translational research.

SESSION 3 · TRANSLATIONAL FRONTIERS **Invited Lecture · 3:40–4:00 PM**

Modeling Pathological Ocular Neovascularization in Zebrafish for Mechanistic Studies and Therapeutic Screening

Dr. Rudra Nayan Das

Assistant Professor, Department of Life Sciences, Shiv Nadar University, Delhi-NCR

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ABSTRACT

The health of our eyes depends critically on their vascular system. Neovascularization is the pathological growth of new blood vessels into tissues where vascularization is normally tightly restricted. In the anterior segment of the eye, such aberrant vascular growth is a major feature of several blinding disorders, including neovascular glaucoma. These vascular responses can arise from genetic predisposition, metabolic disease, inflammation, or tissue damage. However, the mechanisms governing their initiation, progression, and resolution remain incompletely understood and require new experimental models. Zebrafish provide an optically accessible and genetically tractable vertebrate system compatible with preclinical drug testing. Using high-resolution imaging, CRISPR-based genome editing, and single-cell transcriptomics, we are developing complementary models of anterior-segment vasculopathy.

Specifically, we have developed zebrafish models of neovascular glaucoma (NVG) and corneal neovascularization. In our NVG model, mutation-driven alteration of the anterior-segment microenvironment induces pathological angiogenesis, with dense vessels growing over the iris. This is accompanied by inflammatory-cell infiltration and visual impairment. Single-cell RNA sequencing reveals extracellular-matrix remodeling associated with disease progression. In parallel, we established a robust corneal neovascularization model using alkali-induced injury. Neovascularization peaks during the second week after injury, followed by spontaneous vascular regression and restoration of corneal transparency within one month. Together, these models enable mechanistic analysis of pathological angiogenesis, endothelial plasticity, and tissue repair, while providing experimentally tractable platforms for therapeutic and drug-screening studies.

Keywords: *Neovascularization, Vascular Remodeling, Ocular Vasculopathy, Zebrafish, CRISPR*

BIOSKETCH

Dr. Rudra Nayan Das is an Assistant Professor in the Department of Life Sciences at Shiv Nadar Institution of Eminence, India, where he leads a research program in vascular biology. His work focuses on endothelial cell identity and plasticity, organ-specific vasculature, lymphatic biology, and mechanisms underlying vasculopathies. He uses zebrafish as a primary vertebrate model, integrating live imaging, genetic manipulation, lineage tracing, and single-cell transcriptomics. During his postdoctoral research, he studied lymphatic endothelial plasticity and demonstrated lymphatic-to-blood vessel transdifferentiation in vivo, work published in Nature. Subsequently, he also discovered aspects of vena cava development, work published in Science Advances. His current research investigates vascular development and pathology in the eye and intestine, and regulators of lymphatic plasticity. His laboratory aims to combine mechanistic vascular biology with disease modeling and therapeutic discovery.

VISION & GUIDELINES LECTURE SERIES **Vision Lecture · 5:00–5:10 PM**

Lipoprotein(a) as a Causal Risk Factor for CAD: From Molecular Mechanisms to Targeted Treatment Modalities — 60 Years: Still an Unresolved Enigma

Prof. Jasvinder K. Gambhir

Former Senior Professor & Head, Department of Biochemistry, University College of Medical Sciences & GTB Hospital, Delhi

ABSTRACT

Lipoprotein(a) [Lp(a)] was identified by Berg in 1963 as an LDL-variant. Later work showed Lp(a) to be a structurally unique modified LDL having apo-B100 covalently linked to apo(a) molecule, enriched with oxidized phospholipids, thereby possessing atherogenic, thrombogenic as well as pro-inflammatory properties. Over the last 60 years several genetic, epidemiologic and pathological studies have suggested Lp(a) to be a causal risk factor for CAD, myocardial infarction and stroke. Thus, as per NCEP-ATP III guidelines (2001) Lp(a) was classified as an emerging, genetically determined risk factor for CVD. Plasma Lp(a) levels are genetically determined by LPA gene, show remarkable polymorphism having up to 40 isoforms, adult Lp(a) levels are attained by age five, remain relatively stable during an individual's lifetime and are unresponsive to lifestyle changes and typical lipid lowering drugs such as statins and PCSK9 inhibitors. Despite its well established role as a CVD risk factor, the integration of Lp(a) testing in routine clinical practice and risk calculation remains limited due to: lack of awareness among clinicians, non-availability of standard estimation methodology, inconsistent guidelines and limited treatment modalities.

However, recently updated 2026 ACC/AHA guidelines now recommend universal Lp(a) testing, at least once in a lifetime for all individuals, thereby recognizing growing evidence of its significance in CVD risk assessment. Recent development of RNA-based therapies, which are in the Phase 1-3 trials (results are expected later this year), offer a new chance to specifically target and significantly lower Lp(a) up to eighty percent. Additionally, CRISPR/Cas9 based editing of LPA gene in liver offer new hope and reignite interest in Lp(a) as a recognized and treatable risk factor of ASCVD. These approaches will allow for the first time to answer the question, whether or not lowering of Lp(a) translates into clinical benefit to patients. Therefore, the Lp(a) landscape has now moved from basic science phase to active trial and treatment phase.

Keywords: *Lipoprotein(a); Apolipoprotein(a); LPA gene; Coronary artery disease; RNA-based therapeutics*

BIOSKETCH

Prof. Jasvinder K. Gambhir is a clinical biochemist who served as Senior Professor and Head of the Department of Biochemistry at the University College of Medical Sciences (UCMS) and GTB Hospital, Delhi. She obtained her MSc in Biochemistry from Panjab University and her PhD from the Postgraduate Institute of Medical Education and Research (PGIMER), Chandigarh. Over more than four decades her research has focused on lipoprotein(a) and apolipoprotein(a) polymorphism in coronary artery disease, homocysteine metabolism, oxidised LDL and antioxidants in atherosclerosis, and inflammation in diabetic nephropathy. Her early work established lipoprotein(a) as an independent risk factor for coronary artery disease in young Indians. She has around 50 publications and is a member of the American Association for Clinical Chemistry and the Association of Clinical Biochemists of India.

VISION & GUIDELINES LECTURE SERIES **Vision Lecture · 5:10–5:20 PM**

Why Dyslipidemia among Indians Must Be Treated Differently than the Western Population?

Prof. Harmeet Singh Rehan

Director Professor & Head, Department of Pharmacology, Lady Hardinge Medical College, New Delhi

ABSTRACT

Summary prepared by the organising committee.

Indians develop coronary artery disease nearly a decade earlier than Western populations, and dyslipidaemia is a major contributor. The Indian lipid phenotype differs in important ways: atherogenic dyslipidaemia with high triglycerides and low, often dysfunctional, HDL cholesterol; a greater number of small dense LDL particles and higher apolipoprotein B at any given LDL-cholesterol level; frequent coexistence of insulin resistance and metabolic syndrome; and elevated lipoprotein(a) in a substantial proportion of individuals. Risk calculators derived from Western cohorts tend to underestimate risk in Indians, so treatment decisions based on them may come too late.

The Lipid Association of India therefore recommends assessing lifetime rather than 10-year risk, starting statin therapy early, pursuing lower LDL-cholesterol goals than most Western guidelines, measuring lipoprotein(a) at least once, and using non-HDL cholesterol and apolipoprotein B as additional targets. This lecture will examine the pharmacological implications of these differences — the choice and intensity of statins, combination therapy with ezetimibe and PCSK9-directed agents, the place of triglyceride-lowering drugs, adherence and persistence, drug interactions, and safety monitoring through pharmacovigilance — and will argue for an India-specific approach to lipid management.

Keywords: *Dyslipidaemia; South Asians; Lipoprotein(a); LDL-cholesterol targets; Statins*

BIOSKETCH

Prof. Harmeet Singh Rehan is Director-Professor and Head of the Department of Pharmacology at Lady Hardinge Medical College, New Delhi. He obtained his MBBS from UCMS and his MD in Pharmacology from Maulana Azad Medical College, University of Delhi, and has over 27 years of experience in medical education and research. A Commonwealth Fellow (Durham University, UK) and Member of the National Academy of Medical Sciences, he coordinates the Adverse Drug Reaction Monitoring Centre at LHMC under the Pharmacovigilance Programme of India and the Materiovigilance Programme of India, chairs the Quality Control Panel of PvPI (IPC) and an ICMR Data Safety Monitoring Board, and serves on several national programmes and ethics committees. His research covers pharmacovigilance, rational use of medicines, and dyslipidaemia and statin therapy, for which he received Lipid Association of India best-paper awards. He has published 128 original research papers.

VISION & GUIDELINES LECTURE SERIES **Vision Lecture · 5:20–5:30 PM**

Beyond Barker's Hypothesis to Precision Prevention

Prof. Shridhar Dwivedi

Patron, ISAR–DC · Senior Consultant Cardiologist, National Heart Institute, New Delhi

Authors: S. Dwivedi, Harsh Arora

ABSTRACT

Atherosclerosis is traditionally regarded as a disease resulting from cumulative exposure to conventional cardiovascular risk factors such as dyslipidaemia, hypertension, diabetes, obesity and tobacco use during adult life. Increasing evidence, however, suggests that susceptibility to cardiovascular disease may be influenced much earlier, during fetal development and childhood. The Developmental Origins of Health and Disease (DOHaD) concept, originating from the observations of David Barker and colleagues, proposed that adverse intrauterine conditions and altered fetal growth may have long-term consequences for cardiovascular and metabolic health. Maternal undernutrition, obesity, diabetes, hypertension, smoking, alcohol exposure and environmental pollutants may influence placental function, fetal growth, vascular development and metabolic programming. These exposures may interact with genetic susceptibility and epigenetic mechanisms, potentially contributing to endothelial dysfunction, insulin resistance, altered lipid metabolism, hypertension and increased lifetime cardiovascular risk. Maternal obesity and diabetes have been associated with adverse cardiometabolic outcomes in offspring, although the strength and causality of individual associations vary across studies.

It is important to trace the evolution from the original Barker hypothesis to the contemporary DOHaD framework, highlighting the interaction between genes, placenta, nutrition, inflammation, oxidative stress, endocrine signalling and epigenetic regulation. It is also important to examine the relationship between fetal growth patterns, childhood adiposity and subsequent cardiovascular risk.

Importantly, the developmental origins concept should not be interpreted as deterministic. Recent systematic evaluation has highlighted methodological limitations and potential confounding in some of the classical Barker studies, emphasizing the need for cautious interpretation of causal relationships. We have been deliberating this concept very stoutly for last twenty years that intrauterine factors be it maternal or paternal, albeit passive, play significant role in early development of vascular disease in offspring. Situations like fetus papyraceus, familial hypercholesterolemia, familial hypertriglyceridemia, hyperlipoproteinemia(a) , obesity running in generations are a few examples. Not to forget is the ancient textbook 'Garbh Upanishad' mentioning obliquely few of above points.

The emerging concept of precision prevention therefore shifts the focus from treating established atherosclerosis to identifying modifiable risk across the life course—from preconception and pregnancy through childhood and adulthood. We shall be discussing some interesting clinical examples illustrating above concept.

Keywords: *Atherosclerosis; Barker hypothesis; DOHaD; fetal programming; maternal health; epigenetics; premature cardiovascular disease; precision prevention.*

BIOSKETCH

Prof. Shridhar Dwivedi is Senior Consultant Cardiologist at the National Heart Institute, New Delhi, and a Patron of the Indian Society for Atherosclerosis Research – Delhi Chapter. He holds MBBS and MD (Internal Medicine) degrees and a PhD in Cardiology from Banaras Hindu University, and is a Fellow of the National Academy of Medical Sciences (FAMS), the Royal College of Physicians, London (FRCP) and the International Academy of Cardiovascular Sciences (FIACS). A long-standing advocate of preventive cardiology, he lectures widely on the prevention of non-communicable diseases through lifestyle change and has written on the history of Indian cardiology.

SECTION III

ORAL PRESENTATIONS

Young Investigators' Forum · Clinical and Basic Sciences

ORAL PRESENTATIONS

Young Investigators' Forum · 4:00 – 5:00 PM · two parallel sessions competing for the Best Oral Presentation Award. The abstracts below were selected for oral presentation from the submitted abstracts.

A · Clinical Research / Studies — Lecture Hall, DDU Kaushal Kendra

CODE	TITLE · PRESENTING AUTHOR
C1	**Triglyceride–HDL Axis as a Marker of Dyslipidemia in Advanced Chronic Kidney Disease** *Anisha Sharma*
C2	**Validation of Signature Proteins as Diabetic Biomarkers** *Gunjan Sharma*
C3	**Beyond the Conventional Lipid Profile: Elevated Lipoprotein(a) as a Residual Cardiovascular Risk Marker in Coronary Artery Disease** *Choudhary Kuldeep Singh*
C4	**India's Atherosclerotic Cardiovascular Prevention Priorities: A Comparative Analysis of NFHS-5 and NFHS-6** *Kalika Singh*
C5	**Association of Cardiac Troponin I with Glycemic and Lipid Parameters in Patients with Acute Coronary Syndrome** *Krishna Parmar*
C6	**Integrated Quantitative Proteomics and Advanced Structural Characterization of Biofluid Exosomes for Early CAD Diagnostic Signature** *Pratibha Sharma*
C7	**Association of Alpha-1 Antitrypsin with Blood Pressure, Fasting Blood Sugar and Lipid Profile in COPD Patients** *Sharad Singh*
C8	**Sudden Cardiac Death beyond Traditional Risk: Age-Stratified Insights in Coronary Artery Disease Associated Mortality** *Sunil Kumar Mallick*

B · Basic Sciences Research / Studies — FET Auditorium (Main Hall)

CODE	TITLE · PRESENTING AUTHOR
B1	**Erdosteine Attenuates Experimental Autoimmune Myocarditis by Modulating Inflammation, Oxidative Stress, and MAPK Signaling** *Arti Srivastava*
B2	**Mechanistic Evaluation of Platelet Hyperactivity and Arterial Thrombosis in Insulin Resistance** *Ilma Ansari#[a]*
B3	**Terpinolene as a Cardioprotective Candidate Against Cyclophosphamide-Induced Cardiotoxicity: Evidence from Biochemical, Immunohistochemical, and Histopathological Studies** *Adeeba Laeeq*
B4	**Investigating the role of Heat Shock Protein 10 (HSP10) as a novel Clinical Biomarker in Coronary Artery Disease (CAD)** *Annu Kumari*
B5	**Atorvastatin Attenuates Methotrexate-Induced Renal Injury Through Modulation of Oxidative and Inflammatory Pathways: Implications for Cardiovascular-Renal Risk** *Jayadevan K*
B6	**Small Molecule Targeting Sialin Increases NO Bioavailability via Phosphatidylinositol Pathway in Dysfunctional Endothelium** *Shamima Akhtar*
B7	**Mechanistic Insights into the Role of Circulating cfDNA in Vascular Inflammation and Atherosclerotic Plaque Progression** *Manzoor Ali*
B8	**Beyond the Usual Suspects: Whole Exome Sequencing Reveals the Genetic Complexity of Inherited Cardiac Disease** *Shraddha Handa*

C1 ORAL · CLINICAL RESEARCH / STUDIES

Triglyceride–HDL Axis as a Marker of Dyslipidemia in Advanced Chronic Kidney Disease

Dr Anisha Sharma[1], Dr Seema Bhargava[1], Dr Anjali Manocha[1], Dr Mamta Kankra[1], Dr Reetika Saini, Dr Krishna Parmar[1]

[1] Department of Biochemistry, Sir Ganga Ram Hospital, New Delhi, India

Background: Cardiovascular diseases are the leading cause of mortality in Chronic kidney disease (CKD), especially in end stage renal disease patients. Thus, therapies to reduce cardiovascular risk are urgently needed in CKD. The triglyceride-to-high-density lipoprotein cholesterol (TG/HDL-C) ratio may serve as a biochemical marker in CKD.

Aim: To compare triglycerides, HDL-C and the TG/HDL-C ratio between patients with CKD (KDIGO G4–G5) and controls, and to examine their relationship with renal dysfunction.

Methods: This retrospective case-control study included 80 participants: 40 patients with CKD G4–G5 and 40 controls with G1 renal function. Serum triglycerides and HDL-C were measured using routine laboratory methods. The TG/HDL-C ratio was calculated. Continuous variables were compared using t-test or Mann–Whitney U test, as appropriate. Correlations with serum creatinine were assessed using Spearman correlation. Significance was set at $p<0.05$.

Results: Among the 40 CKD patients, 21 (52.5%) were G4 and 19 (47.5%) were G5. Mean age was 57.45±17.15 years in cases and 46.73±13.24 years in controls. HDL-C was significantly lower in CKD patients than controls (32.98±15.28 vs 41.98±13.11 mg/dL; $p=0.006$). Mean triglyceride concentrations were 185.35±119.33 and 210.18±154.13 mg/dL, respectively, with no significant difference ($p=0.423$). The mean TG/HDL-C ratio was higher in cases (8.85±10.39) than controls (5.94±6.47), but the difference was not statistically significant ($p=0.138$). The TG/HDL-C ratio in this cohort showed no significant correlation with serum creatinine ($\rho=0.016$, $p=0.922$).

Conclusion: Advanced CKD was characterized by significantly reduced HDL-C, whereas triglycerides and TG/HDL-C ratio did not differ significantly from controls. Larger studies with adjustment for age, sex, diabetes, medications and dietary factors are needed to establish the TG/HDL-C ratio as a useful marker of dyslipidemia in advanced CKD patients.

Keywords: *Chronic kidney disease; Dyslipidemia; TG/HDL-C ratio; HDL cholesterol; Cardiovascular risk*

C2 ORAL · CLINICAL RESEARCH / STUDIES

Validation of Signature Proteins as Diabetic Biomarkers

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Introduction: Diabetes, earlier considered primarily as a high-risk factor for cardiovascular disease, now has become a far above the ground public health concern, due to the increasing epidemic of diabetes in younger population. Type 2 Diabetes Mellitus (T2DM) is referred to as a progressive disorder characterized by hyperglycemia, peripheral insulin resistance and a decrease in insulin secretion. A bio-marker is defined as a characteristic that is objectively measured and evaluated as an indicator of normal biologic processes, pathogenic processes or pharmacologic responses to a

therapeutic intervention. The present study reports differential gene expression in serum proteins with disease progression in animal model of T2DM.

Aim & Objectives: To identify protein based pharmacodynamic biomarkers associated with diabetes progression.

Methods: These signature proteins were identified using, High Fat Diet (HFD) and Multi Low Dose Streptozotocin (MLD – STZ), a novel animal model for Type 2 Diabetes Mellitus in Albino Wistar rats. Biochemical Studies and Histopathological analysis were conducted to confirm the pathology establishment. Identification of differentially expressed proteins was done through 1D & 2D Electrophoresis, MALDI-TOF-MS/LC-MS with disease progression.

Confirmation of identified proteins through Immunohistochemistry (IHC) as diabetic biomarkers.

Results: It was observed that certain proteins were upregulated & some are downregulated in different stages of the model. Identified 5 biomarkers (Sodium Glucose Cotransporter – 2, Alpha actinin - 1, A Kinase anchor protein – 4, Calcium transporting ATPase, Extracellular Sulphatase) as diabetic signatures for using them as Pharmacodynamic biomarkers.

Summary: A novel set of signature proteins were identified which can assess Diabetes prognosis before the metabolic changes are detectable in blood.

Conclusion: This study concludes with the identification of novel proteins which can be therapeutically explored to reverse the pathological changes of Diabetes Mellitus at molecular level.

Keywords: *Type 2 Diabetes Mellitus, Pharmacodynamic Biomarkers, Signature Proteins*

C3 ORAL · CLINICAL RESEARCH / STUDIES

Beyond the Conventional Lipid Profile: Elevated Lipoprotein(a) as a Residual Cardiovascular Risk Marker in Coronary Artery Disease

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Introduction: Conventional lipid parameters, particularly LDL cholesterol (LDL-C), are routinely used for cardiovascular risk assessment. However, apparently well-controlled lipid levels may not completely reflect residual atherosclerotic cardiovascular risk. Lipoprotein(a) [Lp(a)] is an additional risk marker that may help identify individuals with persistent cardiovascular risk.

Materials and Methods: An 80-year-old male with established coronary artery disease and a history of percutaneous coronary intervention with LAD stenting was evaluated. His conventional lipid profile and serum Lp(a) levels were assessed. Lp(a) was measured by immunoturbidimetry using the Beckman Coulter AU5800.

Results: The conventional lipid profile was largely well controlled, with total cholesterol 103 mg/dL, triglycerides 111 mg/dL, LDL-C 48.5 mg/dL, and non-HDL-C 70.7 mg/dL; HDL-C was low at 32.3 mg/dL. In contrast, Lp(a) was markedly elevated at 95.1 mg/dL (reference: <30 mg/dL) in a patient with established coronary artery disease.

Conclusion: This case demonstrates that well-controlled conventional lipid parameters do not exclude significant cardiovascular disease. Markedly elevated Lp(a) may represent an important component of

residual cardiovascular risk. Incorporating Lp(a) measurement alongside the conventional lipid profile may improve cardiovascular risk assessment.

Keywords: *Lipoprotein(a); Coronary artery disease; LDL cholesterol; cardiovascular risk; Atherosclerosis*

C4 **ORAL · CLINICAL RESEARCH / STUDIES**

India's Atherosclerotic Cardiovascular Prevention Priorities: A Comparative Analysis of NFHS-5 and NFHS-6

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Introduction: Atherosclerotic cardiovascular disease (ASCVD) prevention requires surveillance of multiple modifiable risk factors. We examined changes in India's cardiometabolic indicators to identify priorities for integrated prevention.

Materials and Methods: We conducted a descriptive secondary analysis of official NFHS-6 (2023–2024) national fact-sheet estimates and their NFHS-5 (2019–2021) comparators. Sex-specific absolute changes, relative changes and urban–rural differences were calculated. Blood-sugar and blood-pressure indicators covered adults aged ≥15 years; overweight/obesity covered ages 15–49 years. Survey definitions and denominators were preserved; estimates were not pooled.

Results: The random blood-sugar indicator (>140 mg/dL or medication) increased from 13.5% to 17.8% in women (+4.3 percentage points; 31.9% relative increase) and 15.6% to 20.9% in men (+5.3 points; 34.0%). Overweight/obesity (BMI ≥25 kg/m²) rose from 24.0% to 30.7% in women and 22.9% to 27.3% in men (+6.7 and +4.4 points). The elevated-blood-pressure/medication indicator decreased from 21.3% to 19.4% in women and 24.0% to 22.1% in men (−1.9 points each). NFHS-6 urban–rural overweight/obesity gaps were 17.3 points in women and 13.3 in men.

Conclusions: These divergent patterns support integrated prevention addressing adiposity, glycaemic risk and blood pressure. Ayushman Arogya Mandirs should link risk assessment with lifestyle counselling, diagnostic confirmation, appropriate treatment and longitudinal follow-up. Aggregate comparisons cannot establish changes in ASCVD incidence or programme effectiveness; the random blood-sugar indicator is not equivalent to diagnosed diabetes.

Keywords: *Atherosclerosis; Cardiovascular Risk; Population Surveillance; Primary Health Care; India.*

C5 **ORAL · CLINICAL RESEARCH / STUDIES**

Association of Cardiac Troponin I with Glycemic and Lipid Parameters in Patients with Acute Coronary Syndrome

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Background: Acute coronary syndrome (ACS) is a major cardiovascular condition associated with metabolic abnormalities and dyslipidemia. Cardiac troponin I (cTnI) is an established biomarker of myocardial injury, while glycated hemoglobin (HbA1c), lipoprotein(a) [Lp(a)], triglycerides (TG), and high-density lipoprotein cholesterol (HDL-C) may provide additional information regarding cardiometabolic risk. The TG/HDL-C ratio has also been proposed as a useful marker of an adverse lipid and metabolic profile.

Objectives: To compare cTnI, HbA1c, Lp(a), TG, HDL-C, and the TG/HDL-C ratio between patients with ACS and apparently healthy controls, and to assess the correlations between cTnI and selected metabolic and lipid parameters.

Methods: This comparative observational study included 60 subjects, comprising 30 patients diagnosed with ACS and 30 apparently healthy age- and sex-matched controls. Blood samples were collected for estimation of cTnI, HbA1c, Lp(a), TG, and HDL-C using standard laboratory methods. The TG/HDL-C ratio was calculated from the measured TG and HDL-C concentrations. Biochemical parameters were compared between the ACS and control groups using appropriate statistical tests. Correlations between cTnI and the metabolic and lipid parameters were assessed using suitable correlation analysis.

Results: Patients with ACS demonstrated differences in cardiac and metabolic biochemical parameters compared with healthy controls. The study further evaluated the relationship of cTnI with HbA1c, Lp(a), TG, HDL-C, and the TG/HDL-C ratio to determine their association with myocardial injury and cardiometabolic abnormalities. Statistically significant differences and correlations were interpreted based on the observed data and appropriate significance levels.

Conclusion: Assessment of cTnI alongside HbA1c, Lp(a), lipid parameters, and the TG/HDL-C ratio may provide a broader biochemical profile of patients with ACS. These parameters may contribute to understanding the association between myocardial injury and metabolic or lipid abnormalities.

Keywords: *Acute coronary syndrome; Cardiac troponin I; HbA1c; Lipoprotein(a); TG/HDL-C ratio*

C6 ORAL · CLINICAL RESEARCH / STUDIES

Integrated Quantitative Proteomics and Advanced Structural Characterization of Biofluid Exosomes for Early CAD Diagnostic Signature

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Background: Coronary artery disease (CAD) is a major global cause of mortality, characterized by a long asymptomatic phase involving fatty-fibrous deposit development in the arteries. Early identification of the disease is essential for improved patient outcomes. Small extracellular vesicles (sEVs) or exosomes from human biofluids- such as blood plasma, saliva, and urine are emerging as valuable non-invasive "liquid biopsies" because they carry stable, cell-specific protein signatures protected from degradation by their lipid bilayer membranes. Our study aimed to isolate and characterize sEVs from these biofluids to identify unique protein signatures for early CAD diagnosis.

Methods: sEVs were isolated from clinically relevant volumes of blood plasma, saliva, and urine of CAD (n=60) and age-gender matched healthy controls (n=60). Advanced characterization was performed using immuno-Transmission Electron Microscopy and Scanning Electron Microscopy for morphology, Nanoparticle Tracking Analysis for size distribution and concentration, and Western Blotting for exosome-specific markers including CD63, TSG101, and Flotillin-1. Quantitative protein profiling was achieved through high-throughput label-free LC–MS/MS Orbitrap and iTRAQ-based mass spectrometry, followed by bioinformatics analysis using DAVID, KEGG, and STRING databases.

Results: Saliva exosome proteomic analysis identified 506 proteins, with 18 significant differentially expressed proteins (DEPs). Cystatin-S (CST1/CST2/CST4), Protein S100, alpha-amylase, and Gelsolin were notably upregulated, while Serum albumin and Apolipoprotein A1 (APOA1) were downregulated. In Urinary exosome a total of 508 proteins were identified, revealing significant downregulation of Uromodulin (UMOD), KNG1, and AMBP, alongside upregulation of ORM1, CD59, and Gelsolin. Validation showed that UMOD levels were lowest in recent myocardial infarction patients

compared to stable CAD and healthy controls. For Plasma exosomes, 545 proteins were identified qualitatively, with sEVs exhibiting a more uniform round shape and less morphological heterogeneity compared to saliva and urine in CAD patients. sEVs typically ranged from 30–150 nm in diameter. CAD-derived sEVs in saliva and urine exhibited distinct structural variations, including shape distortions, blebbing, and mixed elongated morphologies, which were absent in healthy controls.

Conclusions: The integration of sEV protein signatures from multiple biofluids provides a robust framework for the non-invasive early detection of CAD. Specifically, Cystatin-S in saliva and Uromodulin in urine represent high-potential biomarkers for identifying early-stage disease and monitoring progression. These findings on validating further in larger cohort, can potentially support the transition toward early diagnosis and risk prediction for preventive cardiovascular medicine.

References: Sharma P, Sancheti M, Inampudi KK, Roy A, Dhamija RK. Salivary small extracellular vesicles reveal protein signatures in young patients with coronary artery disease. BMC Clinical Proteomics. 2025. DOI: 10.1186/s12014-025-09541-9.

Sharma P, Dhamija RK. A streamlined method for isolating and characterizing small extracellular vesicles from various human biofluids for high-throughput downstream applications. BMC Methods 2025. Sep 1;2(1):18. DOI: 10.1186/s44330-025-00040-9.

Sharma P, Roy A, Dhamija RK, Bhushan S, Baswal K, Kulandaisamy R, Yadav S, Kumar S, Inampudi KK. A comprehensive proteomic profiling of urinary exosomes and the identification of early non-invasive biomarker in patients with coronary artery disease. J Proteomics. 2024 Feb 20;293:105059. doi: 10.1016/j.jprot.2023.105059. Epub 2023 Dec 25.

Sharma P, Dhamija RK, Nag TC, Roy A, Inampudi KK. Different Biofluids, Small Extracellular Vesicles or Exosomes: Structural Analysis in Atherosclerotic Cardiovascular Disease Using Electron Microscopy Techniques. Microsc Microanal. 2023 Jun 9;29(3):1168-1177. doi: 10.1093/micmic/ozad025.

Keywords: *Coronary artery disease; Exosomes; Small extracellular vesicles; Proteomics; Biomarkers*

C7 ORAL · CLINICAL RESEARCH / STUDIES

Association of Alpha-1 Antitrypsin with Blood Pressure, Fasting Blood Sugar and Lipid Profile in COPD Patients

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Introduction: Chronic obstructive pulmonary disease (COPD) frequently coexists with hypertension, glycemic disturbance and dyslipidemia. Alpha-1 antitrypsin (AAT), a major circulating antiprotease, may link systemic inflammation with these abnormalities. We compared AAT, fasting blood sugar (FBS), lipid profile and blood pressure in COPD patients with and without hypertension.

Materials and Methods: This cross-sectional study enrolled 212 COPD patients (aged 40–88 years): 106 with hypertension (Group 1) and 106 without (Group 2). Systolic (SBP) and diastolic blood pressure (DBP) were recorded; serum AAT, FBS, total cholesterol, triglycerides, HDL and LDL were estimated. Groups were compared by one-way ANOVA and chi-square test, and associations by Pearson correlation ($p<0.05$ significant).

Results: Mean SBP and DBP of the cohort were 143.03±25.16 and 86.18±11.36 mmHg. Group 1 had significantly lower AAT (87.46±12.13 vs 129.00±19.69 mg/dL) and HDL (44.92±5.18 vs 51.42±4.76 mg/dL), and higher FBS (119.49±9.03 vs 102.85±10.58 mg/dL), total cholesterol (215.32±16.94 vs 182.85±18.58 mg/dL), triglycerides (178.30±22.10 vs 137.05±24.23 mg/dL) and LDL (133.81±17.73 vs 104.68±19.65 mg/dL) (all p<0.001). AAT correlated negatively with SBP (r=−0.750) and DBP (r=−0.738). FBS, total cholesterol, triglycerides and LDL correlated positively with both SBP and DBP (r=0.584–0.678), while HDL correlated negatively (r=−0.530 and −0.539) (all p<0.001).

Conclusions: Hypertensive COPD patients show lower AAT, higher FBS and an atherogenic lipid profile. Reduced AAT together with metabolic dysregulation is strongly associated with elevated blood pressure in COPD, supporting routine AAT, FBS and lipid assessment in these patients.

Keywords: *Alpha-1 antitrypsin; COPD; Hypertension; Fasting blood sugar; Dyslipidemia*

C8 ORAL · CLINICAL RESEARCH / STUDIES

Sudden Cardiac Death beyond Traditional Risk: Age-Stratified Insights in Coronary Artery Disease Associated Mortality

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Introduction: Sudden cardiac death (SCD) is a critical and often unpredictable manifestation of coronary artery disease (CAD), affecting individuals across a broad age spectrum.

Methods: This study uses verbal, conventional and molecular autopsy and investigates the genetic landscape of SCD in two distinct age-groups—young adults (19-45-years) and older adults (46-65-years) - within the South Asian population, where the prevalence of CAD is steadily increasing.

Results: In the younger cohort (n=32; mean age: 36.3-years; male-to-female ratio: 5.4:1), postmortem examination confirmed severe CAD and myocardial infarction (MI). Molecular autopsy revealed genetic variants in 56.25% of cases, involving KCNE1, MYH7, RYR2, ANK2, SYNE2, and single instances in 16 additional genes. According to ACMG guidelines, FBN2 was classified as likely pathogenic and ACTA1 as pathogenic, while the remaining variants were considered of uncertain significance (VUS). Lipid metabolism genes were notably absent; most variants were associated with arrhythmias, long QT syndrome, or cardiomyopathies, with 92.3% exhibiting autosomal dominant inheritance. In the older cohort (n=46; mean age: 53.4-years; male-to-female ratio: 6.7:1), 78.3% of individuals harbored variants in genes linked to structural cardiomyopathies (TTN, FLNC, FHOD3), ion channelopathies (SCN5A, SCN10A, RYR2), and lipid metabolism (PCSK9, APOB, LDLR, ABCA1, LPA).

Conclusion: Several individuals carried multiple variants, suggesting a cumulative genetic burden that may elevate SCD risk. Despite differences in variant distribution between age groups, both cohorts emphasize the value of molecular autopsy in uncovering hidden genetic susceptibilities. These findings support the integration of postmortem genetic screening into routine practice to inform family risk assessment and guide preventive strategies in the era of precision medicine.

Keywords: *Sudden cardiac death, autopsy, coronary artery disease, molecular autopsy, young death*

B1 ORAL · BASIC SCIENCES RESEARCH / STUDIES

Erdosteine Attenuates Experimental Autoimmune Myocarditis by Modulating Inflammation, Oxidative Stress, and MAPK Signaling

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Background: Autoimmune myocarditis is an inflammatory cardiac disorder that may progress to ventricular dysfunction, dilated cardiomyopathy, and heart failure. Erdosteine, a mucolytic agent with antioxidant and anti-inflammatory properties, has potential to modulate inflammatory and oxidative stress pathways; however, its role in experimental autoimmune myocarditis (EAM) remains unexplored.

Objective: The present study aimed to standardize a BALB/c mouse model of EAM and evaluate the cardioprotective effects of erdosteine on cardiac function, myocardial injury, inflammation, oxidative stress, histopathological alterations, and MAPK signaling.

Methods: EAM was induced in BALB/c mice by subcutaneous administration of porcine cardiac myosin emulsified with Freund's Complete Adjuvant on Days 1 and 8. The standardized model was evaluated over 28- and 56-day periods using echocardiography, histopathology, cardiac injury markers, and biochemical parameters. In the therapeutic study, EAM mice received erdosteine at 20, 40, or 80 mg/kg. Cardiac function was assessed by echocardiography, while CK-MB and LDH were evaluated as markers of myocardial injury. Inflammatory responses were assessed by flow cytometric analysis of $CD4^+$/$CD8^+$ cytokine-producing T cells and serum IFN-γ, TNF-α, and IL-6. Cardiac MAPK signaling was evaluated by measuring phosphorylated ERK1/2, JNK, and p38.

Results: EAM induction produced marked cardiac dysfunction, characterized by reduced ejection fraction and fractional shortening, ventricular remodeling, and increased cardiac injury markers. Histopathological examination demonstrated inflammatory cell infiltration, cytoplasmic vacuolization, early fibrosis, and myocyte loss. Erdosteine treatment improved cardiac function and attenuated ventricular dilation, with the 80 mg/kg dose showing marked improvement. Erdosteine also reduced CK-MB and LDH levels and ameliorated myocardial histopathological changes. Furthermore, treatment reduced pro-inflammatory $CD4^+$ and $CD8^+$ T-cell responses and decreased circulating IFN-γ, TNF-α, and IL-6 levels. Importantly, erdosteine attenuated the increased phosphorylation of ERK1/2, JNK, and p38 MAPK observed in EAM.

Conclusion: Erdosteine demonstrated cardioprotective effects in experimental autoimmune myocarditis by improving cardiac function, reducing myocardial injury and histopathological damage, suppressing pro-inflammatory immune responses, and attenuating MAPK pathway activation. The effects were particularly evident in the advanced 56-day EAM model, supporting further investigation of erdosteine as a potential therapeutic strategy for autoimmune myocarditis.

Keywords: *Experimental autoimmune myocarditis; Erdosteine; Oxidative stress; Inflammation; MAPK signaling*

B2 ORAL · BASIC SCIENCES RESEARCH / STUDIES

Mechanistic Evaluation of Platelet Hyperactivity and Arterial Thrombosis in Insulin Resistance

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Introduction: Cardiovascular diseases (CVDs) remain the leading cause of mortality globally. The prevalence of CVDs is compounded in patients with insulin resistance (IR), the hallmark of Type 2 diabetes mellitus (T2D). However, how IR is linked with CVDs and the mechanism through which it promotes thrombotic risk remains poorly understood.

Methodology: We retrospectively studied a clinical cohort of patients who underwent Coronary artery bypass grafting (CABG), different valve surgeries, and pericardiectomy. We then characterized an insulin-resistant (IR) model using intraperitoneal glucose & insulin tolerance tests; we assessed arterial thrombosis in real time using intravital microscopy and platelet function by aggregometry.

Results: We retrospectively analyzed five-year hospital data for patients who underwent different cardiac surgeries and identified metabolically dysfunctional patients using HbA1c. We found that almost 77% of patients recommended for CABG surgery were diabetic/prediabetic (n=425/553 patients) compared to aortic valve replacement surgery (28%, n=43/151), Double valve replacement (26%, n=40/153), mitral valve replacement (31%, n=113/362), and Pericardiectomy surgery (50%, n=17/34). Metabolic dysfunction was therefore markedly more prevalent in CABG patients than in any valve surgery group. Further, IR rats showed impaired glucose and insulin tolerance, confirming the model. Arterial thrombosis revealed a significant decrease in the mean time required to form the first thrombus, as well as the time to complete occlusion, and an accelerated rate of thrombus growth in the animals subjected to IR compared to control. Similarly, we found a significant increase in platelet aggregation in IR animals in response to both ADP and thrombin compared to control. However, TLR4 inhibitor TAK-242 reversed this, with a prolonged time to form the first thrombus and complete occlusion, a decelerated thrombus growth rate, and a reduction in platelet aggregation compared to IR. These results suggest that TLR4 signalling contributes directly to platelet dysfunction in insulin resistance.

Conclusion: Insulin resistance significantly increases cardiovascular risk by enhancing platelet activity and arterial thrombosis. TLR4, an innate immunity receptor, plays a key regulatory role in this process, making it a promising therapeutic target.

Keywords: *Insulin resistance; Platelet hyperactivity; Arterial thrombosis; TLR4; Type 2 diabetes mellitus*

B3 ORAL · BASIC SCIENCES RESEARCH / STUDIES

Terpinolene as a Cardioprotective Candidate Against Cyclophosphamide-Induced Cardiotoxicity: Evidence from Biochemical, Immunohistochemical, and Histopathological Studies

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Cyclophosphamide (CP), a widely used chemotherapeutic agent, is associated with cardiotoxicity mediated by oxidative stress, inflammation, and apoptosis. This study investigated the protective effect of Terpinolene, a naturally occurring monoterpene possessing antioxidant, anti-inflammatory, and anti-apoptotic properties, against CP-induced cardiac damage in Wistar albino rats. Forty-eight Wistar rats (200–250 g) were divided into seven groups; CP (200 mg/kg, single i.p. dose on 7th day) induced cardiotoxicity, followed by oral Terpinolene treatment (12.5, 25, and 50 mg/kg) for 14 days. Resveratrol was used as a standard comparator. Biochemical evaluation of oxidative stress markers, including SOD, TBARS, CAT, and GSH, along with assessment of cardiac injury markers including LDH, BNP, CK-MB and cTnT, and inflammatory mediators like IL-1B, IL-6 and TNF-Alpha were also assessed. Immunohistochemical evaluation of caspase-3 and histopathological examination using H&E and MT staining were also carried out. CP administration significantly increased oxidative stress, inflammatory responses, apoptotic activity, and cardiac tissue injury. Treatment with Terpinolene, particularly at 50 mg/kg, markedly attenuated these alterations and restored the evaluated parameters toward normal levels. Histopathological findings further supported the cardioprotective effect of Terpinolene, with improvements in CP-induced structural alterations comparable to those observed with resveratrol.

Conclusion: Terpinolene demonstrated significant cardioprotective potential against CP-induced cardiotoxicity by attenuating oxidative stress, inflammation, apoptosis, and cardiac tissue damage, suggesting its potential as a therapeutic candidate for mitigating chemotherapy-associated cardiac toxicity.

Keywords: *Terpinolene, Cyclophosphamide, Cardiotoxicity, Oxidative stress, Apoptosis, Inflammation*

B4 ORAL · BASIC SCIENCES RESEARCH / STUDIES

Investigating the role of Heat Shock Protein 10 (HSP10) as a novel Clinical Biomarker in Coronary Artery Disease (CAD)

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Introduction: Coronary artery disease (CAD) remains a leading cause of global mortality, underscoring the critical need for reliable, non-invasive diagnostic and prognostic biomarkers. Heat shock protein 10 (HSP10), an essential mitochondrial co-chaperone, plays a critical regulatory role in cellular stress responses, protein folding, endothelial dysfunction and anti-apoptotic pathways within the cardiovascular system. This study aims to investigate the expression profile of HSP10 in clinical CAD samples to evaluate its potential as a novel diagnostic biomarker.

Materials and Methods: A case-control study was conducted using a clinical cohort (n = 200), comprising angiographically proven CAD patients with ≥70% stenosis [30-75 years age] (n = 100) having ≥70% stenosis and healthy controls [30-75 years age] (n = 100). Total RNA and protein were extracted and the transcript expression levels of target HSP10 were quantified using real-time reverse transcription PCR (RT-qPCR). Corresponding protein expression and accumulation were subsequently evaluated using Western blotting.

Results: RT-qPCR analysis demonstrated a statistically significant upregulation of HSP10 mRNA transcripts in the CAD cohort compared to the control group ($p < 0.05$). These transcriptional findings were corroborated by Western blot analysis, which showed corresponding alterations in HSP10 protein levels among CAD patients.

Conclusions: Our preliminary findings demonstrate that HSP10 is differently expressed in patients with coronary artery disease, highlighting its viability as a promising non-invasive biomarker for CAD. Further validation through larger cohorts is essential to establish the robustness of these findings and support translation role into clinically applicable risk stratification tools.

Keywords: *Coronary artery Disease, Heat shock protein 10, oxidative stress, molecular biomarker, clinical study*

B5 ORAL · BASIC SCIENCES RESEARCH / STUDIES

Atorvastatin Attenuates Methotrexate-Induced Renal Injury Through Modulation of Oxidative and Inflammatory Pathways: Implications for Cardiovascular-Renal Risk

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Background: Methotrexate (MTX) is widely used for the treatment of inflammatory and immune-mediated disorders, conditions that may themselves be associated with increased cardiovascular risk. Inflammation, oxidative stress, and renal dysfunction represent important interconnected components of cardiovascular–renal pathophysiology. MTX-associated renal injury may therefore have implications beyond the kidney, particularly in individuals with concomitant cardiovascular risk. Atorvastatin, an

established cardiovascular therapeutic agent, exerts effects beyond lipid lowering, including modulation of oxidative stress and inflammatory signaling. However, its potential to attenuate MTX-induced renal injury and the associated molecular pathways remains insufficiently explored. This study evaluated the renal protective effects of atorvastatin against MTX-induced injury and examined its potential relevance to cardiovascular–renal risk modulation.

Methods: Wistar albino rats were used to establish MTX-induced renal injury. This study compared normal control rats, MTX-treated rats, and rats receiving MTX with atorvastatin 3 mg/kg orally. Atorvastatin was administered daily for 7 days before and 21 days after a single intraperitoneal dose of MTX. Renal function was assessed using serum urea, blood urea nitrogen, and creatinine. Renal oxidative stress was evaluated using thiobarbituric acid reactive substances (TBARS), reduced glutathione (GSH), and superoxide dismutase (SOD). Renal inflammatory responses were assessed using nitrite, myeloperoxidase (MPO), tumor necrosis factor-α (TNF-α), and prostaglandin E_2 (PGE_2). Renal cyclooxygenase-2 (COX-2) and inducible nitric oxide synthase (iNOS) expression were evaluated by immunohistochemistry.

Results: MTX administration produced significant renal dysfunction, with marked elevations in serum urea, blood urea nitrogen, and creatinine compared with normal controls. Atorvastatin significantly attenuated these alterations, demonstrating improvement in MTX-induced renal functional impairment. MTX exposure was accompanied by pronounced renal oxidative stress, characterized by increased lipid peroxidation and depletion of endogenous antioxidant defenses. Atorvastatin significantly reduced renal TBARS and restored GSH and SOD activity compared with MTX-treated rats. MTX also produced significant increases in renal nitrite, MPO, TNF-α, and PGE_2, indicating enhanced and inflammatory responses. Atorvastatin significantly reduced each of these abnormalities. Immunohistochemical assessment demonstrated increased renal COX-2 expression following MTX administration, which was significantly attenuated by atorvastatin. Although atorvastatin reduced renal iNOS immunoreactivity, this change did not reach statistical significance at the studied dose.

Conclusion: Atorvastatin attenuated MTX-induced renal dysfunction and was associated with restoration of antioxidant defenses and suppression of renal inflammatory and COX-2-associated responses. These findings suggest that the pleiotropic actions of an established cardiovascular therapeutic agent may extend to modulation of drug-induced renal injury. From a translational perspective, the study identifies renal oxidative and inflammatory injury as a potentially relevant component of the cardiovascular–renal interface during MTX therapy. Although direct cardiovascular and atherosclerotic endpoints were not evaluated, these findings provide a preclinical rationale for investigating whether preservation of renal function through established cardiovascular therapies could contribute to integrated cardiovascular–renal risk management. Further studies incorporating vascular and cardiac endpoints are warranted to establish the cardiovascular implications of this approach.

Keywords: *Methotrexate; Atorvastatin; Renal injury; Oxidative stress; Inflammation; Cardiorenal risk; Atherosclerosis*

B6 ORAL · BASIC SCIENCES RESEARCH / STUDIES

Small Molecule Targeting Sialin Increases NO Bioavailability via Phosphatidylinositol Pathway in Dysfunctional Endothelium

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Background: Mechanism underlying reduced NO bioavailability in dysfunctional endothelium remain incompletely understood, particularly since exogenous nitrate supplementation has shown limited therapeutic benefit. Recently, sialin, a nitrate transporter, has emerged as a potential regulator of intracellular nitrate/nitrite homeostasis.

Aim: We screened novel small-molecule inhibitor of sialin using in silico approach and evaluated its therapeutic potential in vitro.

Methods: Small molecule inhibitors of Sialin were identified through in silico screening and molecular docking. MTT assay was performed to measure cytotoxicity. Intracellular and extracellular total NO, nitrite, and nitrate levels were quantified. Whole transcriptome profiling of sialin inhibitor treated ECs were performed. Quantitative real-time PCR, western blotting and in vitro assays were performed to assess EC functionality.

Results: In silico screening identified 34 high-affinity sialin-binding candidates. Top 4 compounds were selected for functional validation. Sialin inhibition restored intracellular NO, and nitrate levels while reducing extracellular nitrate efflux in stimulated ECs. Whole transcriptomics data analysis revealed that sialin regulates pathways related to eNOS signalling via phosphatidylinositol pathway. Inhibitor treatment enhanced eNOS expression, and restored autophagy-associated genes.

Conclusion: Targeting sialin with novel small-molecule inhibitor restored endothelial NO bioavailability, highlight sialin as a promising novel therapeutic target for endothelial dysfunction.

Keywords: *Sialin; Nitric oxide bioavailability; Endothelial dysfunction; Small-molecule inhibitor; Phosphatidylinositol pathway*

B7 **ORAL · BASIC SCIENCES RESEARCH / STUDIES**

Mechanistic Insights into the Role of Circulating cfDNA in Vascular Inflammation and Atherosclerotic Plaque Progression

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Vascular inflammation, endothelial injury, oxidative stress, and cell death are central mechanisms underlying vascular dysfunction and atherosclerotic plaque progression. However, the contribution of circulating cell-free DNA (cfDNA) to these processes and its potential as a marker of vascular tissue injury remain incompletely understood. Using high-altitude pulmonary edema (HAPE) as a model of acute hypoxia-induced vascular and endothelial injury, we investigated the structural characteristics, biological origin, and inflammatory signatures of circulating cfDNA using molecular, biochemical and proteomic approaches. A case-control study included 145 HAPE patients and 139 healthy high-altitude sojourners. HAPE patients were further classified as mild (n = 45), moderate (n = 50), and severe (n = 50). HAPE patients exhibited significantly higher cfDNA concentrations and a lower DNA Integrity Index (DII), reflecting an increased proportion of shorter cfDNA fragments in circulation. cfDNA levels increased with disease severity and correlated inversely with SpO_2. Further analyses revealed significantly elevated 8-hydroxy-2'-deoxyguanosine (8-OxodG), Annexin A5, High Mobility Group Box 1 (HMGB1), Cytokeratin 18 (CK18), and caspase-cleaved Cytokeratin 18 (ccK18), indicating oxidative stress, apoptosis, and secondary necrosis as major contributors to cfDNA release. Targeted proteomics identified significant alterations in OSM, IL-1β, IL-6, OLR1, VEGFA, and HGF, highlighting inflammatory activation, endothelial dysfunction, and vascular remodeling. Collectively, these findings demonstrate that hypoxia-associated vascular injury is accompanied by characteristic cfDNA release and fragmentation signatures, providing mechanistic insights into how cfDNA may reflect inflammatory and cellular injury pathways relevant to vascular disease and atherosclerotic plaque progression. These findings support cfDNA fragmentomics as a promising non-invasive approach for investigating vascular injury and disease progression.

Keywords: *Cell-free DNA; Vascular inflammation; High-altitude pulmonary edema; Endothelial injury; Atherosclerosis*

B8 ORAL · BASIC SCIENCES RESEARCH / STUDIES

Beyond the Usual Suspects: Whole Exome Sequencing Reveals the Genetic Complexity of Inherited Cardiac Disease

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Cardiovascular diseases (CVDs) are the leading cause of mortality in India, with sudden cardiac death (SCD) representing a major public health challenge. While structural heart diseases such as coronary heart disease predominate among individuals above 40 years of age, inherited arrhythmia syndromes contribute substantially to SCD in younger individuals. Long QT syndrome (LQTS), one of the most common, life threatening inherited cardiac channelopathies, is characterized by delayed cardiac repolarization, prolongation of corrected QT interval (QTc), and increased susceptibility to syncope, seizures, ventricular arrhythmias and SCD. In this study, we investigated the genetic architecture of suspected LQTS probands and phenotypically positive first-degree relatives (PPFDRs) in a multicentric Indian cohort using whole-exome sequencing (WES), with subsequent clinical, familial and computational evaluation of identified variants. Among 167 clinically suspected individuals, pathogenic/likely pathogenic variants were identified in 49.7%, while 28.75% individuals harboured VUS. Overall, 128 unique variants across 27 genes were identified, demonstrating genetic variation extending beyond the canonical LQTS-associated genes, *KCNQ1, KCNH2* and *SCN5A*. Familial screening further identified 28.2% asymptomatic first-degree relatives (AFDRs) to be silent carriers. These findings demonstrate substantial genetic complexity underlying LQTS in the Indian population and emphasize the importance of integrating genomic, facilitate familial risk identification and provide a foundation for precision approaches to cardiovascular risk assessment and management.

Keywords: *Long QT syndrome; Whole exome sequencing; Sudden cardiac death; Inherited arrhythmia; Indian cohort*

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SECTION IV

POSTER PRESENTATIONS

Lunch & Poster Session · 1:30 – 3:00 PM

P01 POSTER

Multimorbidity and Preventable Cardiovascular Risk Factors in Septuagenarians and Above

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Introduction: Ageing is associated with an increasing burden of chronic diseases and multimorbidity. Elderly patients with coronary artery disease (CAD) may have multiple coexisting conditions and modifiable cardiovascular risk factors. This study assessed multimorbidity and selected cardiovascular risk factors among elderly patients with documented CAD.

Materials and Methods: A retrospective observational study was conducted using cardiology outpatient records from January 2016 to December 2025. Patients aged ≥70 years with documented CAD and complete clinical records were included. Demographic characteristics, hypertension, type 2 diabetes mellitus, obesity, tobacco exposure, and other chronic conditions were recorded. Multimorbidity was defined as two or more chronic conditions. Data were analysed descriptively using frequencies, percentages, and mean ± standard deviation.

Results: During the study period, 608 patients aged ≥70 years were screened, of whom 166 (27.3%) had documented CAD and were included. The mean age was 77.3 ± 5.8 years; 112 (67.5%) were males and 54 (32.5%) females. Hypertension was present in 113 (68.1%), type 2 diabetes mellitus in 89 (53.6%), and obesity in 67 (40.4%). Tobacco exposure was documented in 40 (24.1%) patients. Multimorbidity was present in 153 (92.2%). Hypertension with type 2 diabetes mellitus was the most frequent combination, occurring in 40 (24.1%) patients.

Conclusions: A high burden of multimorbidity was observed among elderly patients with CAD. Hypertension and type 2 diabetes mellitus were the most frequent conditions, while obesity and tobacco exposure remained prevalent. These findings highlight that important cardiovascular risk factors remain preventable even in advanced age through blood pressure and diabetes control, tobacco cessation, and weight management.

Keywords: *Coronary artery disease; Multimorbidity; Elderly; Hypertension; Obesity*

P02 POSTER

Protein-Templated DNA Synthesis by an Antiphage Reverse Transcriptase

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Introduction: Nucleic acid polymerases generally require a nucleic acid template to direct sequence-specific synthesis. Defense-associated reverse transcriptases (DRTs) are bacterial antiphage systems that exhibit unconventional modes of nucleic acid synthesis. The DRT3 system consists of two distinct reverse transcriptases, Drt3a and Drt3b, together with a non-coding RNA (ncRNA). This study investigated the mechanism by which DRT3 synthesizes DNA and contributes to antiphage defense.

Materials and Methods: The study employed bacterial expression and phage-defense assays, biochemical DNA-synthesis experiments, DNA sequencing, protein purification, mutational analysis, and cryo-electron microscopy. The individual activities of Drt3a and Drt3b were examined using active-

site mutants and different dNTP combinations. Cryo-EM structures were determined to characterize the molecular organization and DNA-synthesis mechanism of the DRT3 complex.

Results: DRT3 synthesized alternating poly(GT/AC) double-stranded DNA. Drt3a generated poly(GT) DNA using a conserved ACACAC sequence within the ncRNA as a template. In contrast, Drt3b synthesized the complementary poly(AC) strand in the absence of a nucleic acid template. Cryo-EM structures at 2.6 Å resolution showed a D3-symmetric complex containing six copies each of Drt3a, Drt3b, and ncRNA. Conserved Drt3b residues, including Glu26 and Arg253, interacted with the nascent DNA to enforce nucleotide alternation, while Tyr650 mediated protein-primed DNA synthesis. Mutations disrupting these residues impaired DNA synthesis and antiphage defense.

Conclusions: The DRT3 system demonstrates an unusual mechanism of sequence-specific DNA synthesis in which protein residues can substitute for a nucleic acid template. The coordinated activities of Drt3a and Drt3b generate complementary repetitive DNA and contribute to bacterial antiphage defense. This finding expands the known functional diversity of reverse transcriptases and reveals an unconventional mechanism of biological information transfer.

Keywords: *DRT3; Reverse transcriptase; Protein-templated DNA synthesis; Antiphage defense; Dinucleotide repeats*

P03 **POSTER**

Artificial Intelligence-Enabled ECG for Early ASCVD Risk Stratification

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Introduction: Atherosclerotic cardiovascular disease (ASCVD) remains a major cause of cardiovascular morbidity and mortality. Conventional risk assessment relies on demographic, clinical and laboratory variables, but may not capture all cardiovascular risk-related information. Electrocardiography (ECG) is widely available, inexpensive and routinely performed. Recent advances in artificial intelligence (AI) have enabled analysis of subtle ECG features that may not be apparent through conventional interpretation. This review explores the potential of AI-enabled ECG as an adjunct for early ASCVD risk stratification.

Materials and Methods: A focused literature review was conducted on recent studies investigating AI-enabled ECG for coronary disease detection and prediction of future ASCVD and cardiovascular outcomes. Studies were examined for population characteristics, model performance, prognostic value and potential clinical applications.

Results: Large-scale studies demonstrate promising findings. A study involving more than 7.1 million patients showed that AI-enabled ECG models could identify coronary disease-related features and provide risk information additional to conventional assessment. The AIRE platform, developed using 1,163,401 ECGs from 189,539 patients and externally validated across international cohorts, demonstrated prediction of future ASCVD, mortality and other cardiovascular outcomes. Emerging studies also suggest that ECG-derived age may provide additional prognostic information.

Conclusions: AI-enabled ECG has potential as a scalable adjunct to conventional ASCVD risk assessment. However, prospective validation, diverse population studies and evaluation of clinical utility are required before routine clinical implementation.

Keywords: *Artificial intelligence; Electrocardiography; ASCVD; Risk stratification; Cardiovascular prevention*

P04 POSTER

Genetic Testing for Familial Hypercholesterolemia: Role of Molecular Diagnostics in Early Detection and Cascade Screening

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Introduction: Familial hypercholesterolemia (FH) is an inherited condition in which the level of low-density lipoprotein cholesterol (LDL-C) remains high throughout life. This increases the risk of developing atherosclerosis and coronary artery disease at a younger age. Heterozygous FH affects approximately 1 in 250–311 people, while the rarer homozygous form affects around 1 in 300,000 people. Despite this, many individuals with FH are never diagnosed.

Traditional diagnostic methods, such as the Dutch Lipid Clinic Network and Simon Broome criteria, mainly rely on cholesterol levels and family history. Although these methods are useful, they may fail to identify some affected individuals. Genetic testing can provide additional information by identifying the specific genetic changes responsible for FH. This review focuses on how molecular genetic testing can improve early diagnosis and screening of family members.

Materials and Methods: A narrative review of published research was carried out using PubMed, Scopus, and ScienceDirect. The review included studies covering the genetic causes of FH, molecular testing methods, the relationship between genetic findings and clinical features, and cascade screening of family members. Meta-analyses, large cohort studies, and recent consensus guidelines were given greater priority.

Results: Most cases of FH are linked to pathogenic variants in the LDLR gene. Other important genes include APOB and PCSK9, while LDLRAP1 is associated with a rare recessive form of the condition.

Several molecular testing methods are available, including Sanger sequencing, next-generation sequencing (NGS) gene panels, and multiplex ligation-dependent probe amplification (MLPA), which can identify large rearrangements in the LDLR gene. NGS panels are increasingly preferred because they can examine several genes at the same time, while reducing cost and testing time.

A pathogenic variant can be identified in approximately 60–80% of people with definite FH, compared with around 20–30% of those with possible FH. When no pathogenic variant is found, the high LDL-C level may have a polygenic cause. Importantly, people carrying a pathogenic FH variant have a substantially higher risk of premature coronary artery disease. In one large study, this risk was approximately 22 times higher in variant carriers compared with about 6 times higher in individuals without an identified variant but with similarly elevated LDL-C levels.

Genetic confirmation can also make it easier to screen close relatives through cascade screening. This approach can help identify affected family members before symptoms or complications develop. Early diagnosis allows treatment with medicines such as statins, ezetimibe, and PCSK9 inhibitors, ideally beginning during childhood when appropriate.

Challenges: Although genetic testing has clear benefits, several challenges remain. Correctly interpreting genetic variants, particularly variants of uncertain significance (VUS), can be difficult. Limited access to genetic testing and affordability are also important barriers.

Conclusion: Genetic testing can complement traditional clinical criteria by helping confirm the diagnosis of FH and providing a clearer picture of an individual's cardiovascular risk. It also makes cascade screening of family members more effective, allowing affected individuals to be identified and treated earlier.

Greater use of NGS-based testing could help improve early detection and reduce the risk of premature atherosclerotic disease. To make genetic testing more widely available, there is also a need for more population-specific genetic data, particularly from India, along with affordable testing options.

Keywords: *Familial hypercholesterolemia; Genetic testing; Next-generation sequencing; Cascade screening; Atherosclerosis*

P05 **POSTER**

Site-specific acetylation and deacetylation at conserved Lys839 and Lys842 in the AI loop modulate functional dynamics, conformational equilibrium and coupling efficiency of Neuronal Nitric Oxide Synthase

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Catalytic activity of neuronal nitric oxide synthase (nNOS) is governed by the autoregulatory insert (AI) of the flavoprotein reductase domain, although the contribution of post-translational modifications within this element remains undefined. Here, two AI lysine residues, Lys839 and Lys842, were investigated using acetylmimetic (K→Q) and deacetylmimetic (K→R) substitutions. Substitution at either position produced charge- and chemistry-dependent effects on reductase function. K842Q supported a 1.6-fold increase in NO synthesis relative to wild-type (WT) and enhanced coupling, reducing NADPH consumption from 2.89 to 2.17 molecules per NO formed, whereas K842R was less active than WT. K839Q was comparable to WT, while K839R was substantially compromised: cytochrome c reduction decreased to approximately one-third of WT, coupled heme flux declined to ~27 min^{-1}, corresponding to ~28% coupling efficiency, approximately half the WT value. Ferricyanide reduction was unaltered in all mutants, indicating that these residues act downstream of the initial FAD reduction step. Flavin fluorescence amplitudes and autooxidation rates indicate that acetylmimetic substitution favours the open, FMN-deshielded conformation, whereas deacetylmimetic substitution stabilises the closed state. Collectively, these findings identify Lys842 acetylation as a regulatory mechanism that optimizes electron transfer through the reductase domain, improves catalytic coupling, and enhances NO production without altering the intrinsic requirement for calmodulin activation. This study provides new mechanistic insight into AI-mediated regulation of nNOS and identifies Lys839/Lys842 acetylation and deacetylation as a potential target for modulating NO signaling under physiological and pathological conditions.

Keywords: *Neuronal Nitric Oxide Synthase, Autoinhibitory Insert, Post-Translational Modifications, Acetylation, Oxidative Stress*

P06 **POSTER**

Genomic Signatures of High-Altitude Adaptation in Ladakhi Highlanders: An Evolutionary Perspective on Cardiovascular Homeostasis

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High-altitude environments are characterized by reduced barometric pressure and oxygen availability, leading to chronic hypobaric hypoxia and substantial physiological stress on human populations. Long-term residence under these conditions requires coordinated adaptations involving oxygen sensing, erythropoiesis, angiogenesis, endothelial function, vascular regulation, and cellular stress responses. These processes are also central to cardiovascular physiology, as dysregulated hypoxia signalling, endothelial dysfunction, altered nitric oxide bioavailability, oxidative stress, and inflammation contribute to vascular pathology and atherosclerosis. Ladakh, a Trans-Himalayan region of northern India extending from approximately 2,700 to 5,500 m above sea level, provides a natural model for investigating the genetic basis of high-altitude adaptation. This study aimed to identify genomic signatures of natural selection in Ladakhi highlanders and explore their potential relevance to cardiovascular biology. Genome-wide genotyping was performed using the Illumina Infinium Global

Screening Array, followed by standard quality-control procedures. Population structure was evaluated using ancestry-inference approaches. Complementary selection scans, including population branch statistic (PBS), integrated haplotype score (iHS), cross-population extended haplotype homozygosity (XP-EHH), were employed to identify candidate regions under selection. Regions supported by multiple independent approaches were prioritized for functional annotation and pathway analysis. Multiple genomic regions showed convergent evidence of selection in highlanders relative to lowland populations, including overlapping signals across PBS, iHS, and XP-EHH. Candidate regions included genes and pathways involved in hypoxia response, oxygen homeostasis, vascular regulation, cellular stress, and metabolism. Characterization of these adaptive mechanisms may provide candidate pathways for subsequent investigation of broader cardiovascular disease susceptibility, thereby linking evolutionary adaptation to cardiovascular health and disease.

Keywords: *High-altitude adaptation; Hypobaric hypoxia; Natural selection; Ladakhi highlanders; Cardiovascular homeostasis*

P07 **POSTER**

Efficacy and Safety of Sarpagandha Mishran Versus Amlodipine in Stage-I Primary Hypertension: A Prospective Double-Blind Randomised Controlled Trial

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Background: Sarpagandha Mishran is an Ayurvedic formulation used for hypertension, but comparative evidence against established antihypertensive therapy is limited. This study evaluated its efficacy and safety compared with amlodipine in Stage I primary hypertension.

Materials and Methods: This prospective, double-blind, randomised, double-dummy, controlled Phase III trial was conducted at AIIMS, New Delhi. Participants aged 30–65 years with Stage I primary hypertension were randomised 1:1 to Sarpagandha Mishran 500 mg twice daily plus placebo amlodipine 5 mg once daily, or amlodipine 5 mg once daily plus placebo Sarpagandha Mishran. Both groups received hydrochlorothiazide 12.5 mg once daily for 12 weeks. The primary outcomes were changes in systolic and diastolic blood pressure. Secondary outcomes included inflammatory and cardiovascular biomarkers, quality of life, and safety parameters.

Results: Of 120 participants enrolled, 112 were analysed. Both groups showed significant reductions in systolic and diastolic blood pressure (within-group $p<0.001$). In the Sarpagandha Mishran group, systolic blood pressure decreased from 148.27±7.21 to 134.48±11.16 mmHg and diastolic blood pressure from 93.80±3.65 to 89.13±7.56 mmHg. In the amlodipine group, corresponding reductions were from 146.16±7.20 to 131.98±16.85 mmHg and from 94.05±3.29 to 85.86±6.09 mmHg. The between-group difference in final systolic blood pressure was not significant ($p=0.357$). IL-6, pro-BNP, and oxidative stress markers improved in both groups. Safety laboratory parameters were generally stable.

Conclusion: Sarpagandha Mishran demonstrated significant blood-pressure reduction over 12 weeks, with no significant between-group difference in final systolic blood pressure compared with amlodipine. Further studies may clarify its role in hypertension management.

Keywords: *Sarpagandha Mishran; Primary hypertension; Amlodipine; Blood pressure; Randomized controlled trial*

P08 **POSTER**

Experimental Evaluation of the Effect of 4-Octyl Itaconate in DOCA-Salt-Induced Hypertension in Rats

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Introduction: Hypertension is a major cardiovascular disorder affecting people worldwide. It is associated with oxidative stress, inflammation and progressive myocardial damage. 4-Octyl itaconate (4-OI), a cell-permeable derivative of itaconate, exhibits antioxidant and anti-inflammatory properties. The present study investigated the cardioprotective effects of 4-OI in an experimental model of deoxycorticosterone acetate (DOCA) salt-induced hypertension in rats.

Materials and Methods: Adult male Wistar albino rats were used in the study. Rats received DOCA (20 mg/kg, s.c., twice weekly) along with 1% NaCl drinking water for five weeks to induce hypertension. 4-OI (10 mg/kg, ip) dissolved in olive oil was administered as treatment. At the end of 5 weeks, hemodynamic parameters were determined. Further, myocardial oxidative stress, antioxidant level, and inflammation were assessed by measuring malondialdehyde (MDA), reduced glutathione (GSH), superoxide dismutase (SOD), tumor necrosis factor-alpha (TNF-α), and interleukin-6 (IL-6). Serum creatine kinase-MB (CK-MB) and lactate dehydrogenase (LDH) were measured as markers of myocardial injury. Histopathological examination was performed to assess structural changes in cardiac tissue.

Results: Administration of DOCA-salt led to marked increase in blood pressure and myocardial lipid peroxidation. Levels of inflammatory markers exhibited elevation whereas antioxidant levels decreased. Treatment with 4-OI attenuated these alterations, improving hemodynamic parameters, restoring antioxidant status, and reducing oxidative and inflammatory responses. These findings support the cardioprotective effects of 4-OI.

Conclusion: 4-Octyl itaconate exhibited cardioprotective effects in DOCA-salt-induced hypertension in rats. Its effects were associated with attenuation of oxidative stress and inflammation, preservation of antioxidant defenses, improvement in cardiovascular parameters, and reduction in damage to cardiac tissues. These findings suggest that 4-OI ameliorates high blood pressure through modulation of antioxidant and anti-inflammatory pathways.

Keywords: *4-Octyl itaconate; DOCA-salt; Hypertension; Oxidative stress; Inflammation.*

P09 **POSTER**

Intravascular Ultrasound: A Closer Look at Coronary Artery Disease

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Introduction: Heart arteries are usually examined with angiography, which outlines only the open channel of the vessel. Plaque growing within the wall can therefore go unnoticed in early disease. Intravascular ultrasound (IVUS) places a very small ultrasound tip inside the artery to produce cross-sectional pictures. This review discusses how IVUS supports diagnosis and treatment planning.

Materials and Methods: Published articles on coronary IVUS were reviewed and summarised in the author's own words. They covered comparisons with angiography, studies following plaque changes over time, and reports on its role in stenting.

Results: IVUS lets clinicians see how much plaque is present, where it lies, and roughly what it is made of. Its advantage over angiography is greatest when the artery is irregular in shape. Patients carrying more plaque, or showing faster plaque growth, were more likely to later need repeat procedures or suffer cardiac events. During stenting, IVUS helps choose the correct stent size and position, notably in the left main artery and at branching points. It also helps identify moderate narrowings that can safely be left alone. Its limits include difficulty in very small arteries and extremely tight narrowings, and it cannot reliably show whether a narrowing restricts blood flow.

Conclusions: IVUS is best used together with angiography rather than instead of it. By revealing the true extent of plaque, it supports risk assessment and more accurate stent placement, and so helps clinicians make better treatment decisions.

Keywords: *intravascular ultrasound; coronary artery disease; atheromatous plaque; stenting; angiography*

P10 **POSTER**

Evaluation of antioxidant activity of Gallic acid loaded phytosome

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Free radicals are produced during biochemical reactions occurred in the body. They are responsible for developing various types of disease such as heart disease, cancer, diabetes and atherosclerosis. Gallic acid has free radical scavenging activity and reduce risk of cancer, heart disease and diabetes etc. Gallic acid loaded phytosomes were prepared by standard methods and evaluated for antioxidant activity. It was observed that the developed phytosomes were found to be within the range of nano-size and were of spherical shape. Antioxidant activity was evaluated by DPPH method. The antioxidant activity of the developed phytosomes was found to be comparable with reference standard ascorbic acid. It can be concluded that the developed phytosomes have potent antioxidant/free radical scavenging potential. Further studies on this formulation, including in vivo studies and clinical trials, are necessary to further evaluate its efficacy and safety.

Keywords: *Phytosomes, Gallic acid, DPPH, antioxidant*

P11 **POSTER**

Pharmacological studies to delineate the effect of Hesperidin in an experimental model of Cardiac Hypertrophy

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Objectives: Cardiac hypertrophy is an adaptive process of the heart. It is a leading cause for heart failure, which in turn has a high mortality rate. Hesperidin (HES) is a bioactive flavanone glycoside with agonistic activity on Peroxisome Proliferator-Activated Receptor gamma (PPAR-γ), reported to be an inhibitor of cardiac hypertrophy signalling pathways. Therefore, we investigated the cardio-protective effect of this bioactive flavonoid in isoproterenol induced cardiac hypertrophy.

Methods: The work was started after getting approval from the institutional animal ethics committee (IAEC). Male albino Wistar rats were divided into 6 groups (n=6) and administered isoproterenol (3 mg/kg/day s.c.) or hesperidin (200 mg/kg/day, p.o.) and isoproterenol or enalapril (30 mg/kg/day p.o.) and isoproterenol or a combination of hesperidin and enalapril for 28 days. On the 29th day, rats were anaesthetized followed by cannulation of coronary artery to record hemodynamic parameters. Blood sample was collected and heart was excised to evaluate for parameters like biochemical, histopathological, ultrastructural and immunohistochemical studies.

Results: Hesperidin significantly normalized heart weight/body weight ratio and cardiomyocyte area in the treated rats. Moreover, hesperidin improved cardiac function and attenuated pathological changes as seen in the heart tissue sections. A decline in the levels of cardiac injury markers (CK-MB, LDH) and inflammatory markers (TNF-α, IL-6) was observed. Additionally, hesperidin suppressed oxidative stress and apoptosis along with an increased PPAR-γ expression.

Conclusion: Hesperidin has a good potential for attenuation of cardiac hypertrophy and acts by virtue of its PPAR-γ agonistic, anti-inflammatory, anti-apoptotic and anti-oxidant properties.

Keywords: *Flavonoids, Hesperidin, Cardiac hypertrophy, TNF, IL-6*

P12 **POSTER**

Evaluation of Natural and Synthetic Iron Chelators Against Ferroptosis-Associated Bone Loss in Iron Overload-Induced C57BL/6 Mice

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Background: Iron overload is increasingly recognized as a contributor to skeletal deterioration; however, the involvement of ferroptosis in iron overload-associated bone loss remains insufficiently elucidated.

Objective: To investigate ferroptosis-associated alterations in iron overload-induced bone loss and comparatively evaluate the osteoprotective effects of natural and synthetic iron chelators administered individually and in combination.

Methods: Iron overload-induced bone loss was established in C57BL/6 mice using iron dextran. Animals were treated with a natural iron chelator, a synthetic iron chelator, or their combination. Serum receptor activator of nuclear factor-κB ligand (RANKL), glutathione peroxidase 4 (GPX4), and iron levels were quantified. Femoral osteoprotegerin (OPG) expression was assessed by immunohistochemistry, while histopathological alterations were evaluated using hematoxylin and eosin staining. Micro-computed tomography (micro-CT) was performed to assess femoral bone mineral density and microarchitectural parameters.

Results: Iron overload resulted in marked deterioration of femoral bone microarchitecture, accompanied by increased serum RANKL and iron levels and decreased GPX4 levels. The natural iron chelator provided comparatively greater protection against iron overload-associated bone deterioration than the synthetic chelator, as evidenced by improved micro-CT parameters and favorable modulation of RANKL, GPX4, and serum iron levels. Combination treatment demonstrated protective effects but did not exhibit superior efficacy compared with the natural chelator alone.

Conclusion: Natural iron chelation demonstrated promising osteoprotective potential against iron overload-induced bone loss and may provide a therapeutic approach for modulating iron dysregulation and ferroptosis-associated skeletal damage.

Keywords: *Iron overload; Ferroptosis; Bone loss; Iron chelation; RANKL; GPX4; OPG; Micro-CT.*

P13 **POSTER**

Biomechanical Stress in Coronary Atherosclerosis: Insights from Computational Modelling

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Introduction: Coronary atherosclerosis is a major contributor to coronary artery disease and acute coronary syndrome. Plaque rupture followed by thrombus formation is an important mechanism responsible for acute coronary events. Although conventional imaging can identify morphological features associated with plaque vulnerability, accurately predicting plaques at risk of rupture remains

challenging. Computational modelling has emerged as a promising approach for investigating the relationship between plaque morphology, coronary blood flow, and biomechanical forces.

Materials and Methods: A literature-based review was performed focusing on coronary atherosclerosis, plaque vulnerability, endothelial shear stress, biomechanical stress, and image-based computational modelling. Published evidence describing the application of computational fluid dynamics and biomechanical analysis to coronary imaging was examined to understand their potential role in evaluating plaque progression and rupture.

Results: Reviewed evidence indicates that local haemodynamic and biomechanical factors may influence atherosclerotic plaque progression and vulnerability. Endothelial shear stress appears to contribute to changes in plaque characteristics, while computational modelling can provide detailed information regarding blood-flow patterns and mechanical stresses. Integration of imaging data with computational analysis may therefore help identify high-risk plaque features that are difficult to characterize using conventional imaging alone.

Conclusions: Image-based computational modelling provides valuable insights into the mechanisms underlying coronary plaque progression and rupture. Assessment of biomechanical parameters, particularly endothelial shear stress, may improve future identification of vulnerable plaques. Further clinical validation and standardization are required before these techniques can be routinely applied in clinical practice.

Keywords: *Atherosclerosis, Coronary artery disease, Plaque rupture, Shear stress, Computational modelling*

P14 **POSTER**

Targeting NLRP3 Inflammasome Activation: Identification of Small-Molecule Inhibitors with Therapeutic Potential in Atherosclerosis

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Inflammasomes have emerged as important mediators of inflammation in atherosclerotic cardiovascular disease, with clinical evidences implicating the role of inflammasome-associated IL-1β signaling in cardiovascular risk. Recent studies suggest that the contribution of NLRP3 inflammasome may be particularly pronounced under conditions of heightened metabolic or inflammatory stress. In this study, we performed a cell-based screening approach to identify small-molecule inhibitors of NLRP3 inflammasome activation. Candidate compounds were evaluated in an LPS primed, Nigericin/ATP-activated macrophage model, with IL-1β release as the primary functional readout, followed by assessment of cellular viability and mechanistic characterization of selected candidates. Screening identified compounds that markedly suppressed inflammasome-associated IL-1β release, with selected candidates additionally reducing IL-18 production without significant cytotoxicity. The inhibitory activity of selected candidates was further validated using the established NLRP3 inhibitor MCC950, while preliminary mechanistic studies supported modulation of the NLRP3 inflammasome pathway. These findings identify pharmacological candidates capable of modulating NLRP3-associated inflammatory responses and provide a basis for further therapeutic development. Future studies will investigate the lead NLRP3 inhibitor in preclinical models of atherosclerosis, focusing on plaque burden, vascular inflammation, macrophage accumulation, plaque stability, and atherothrombotic complications. This study provides a foundation for developing targeted NLRP3-directed anti-inflammatory strategies to address inflammation-associated residual cardiovascular risk.

Keywords: *NLRP3 inflammasome; Atherosclerosis; IL-1β; Small-molecule inhibitors; Macrophages*

P15 POSTER

Pregnancy-Specific Biological Reference Intervals for Metabolic Biomarkers and TSH: A Step Towards Precision Cardiometabolic Assessment

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Background and Aim: Pregnancy causes physiological and metabolic alterations affecting thyroid function and glycemic status. Fructosamine, albumin-corrected fructosamine (ACF), and fructosamine–albumin ratio (FAR) may improve glycemic assessment, but trimester-specific reference intervals are limited in the Indian population. To establish biological reference intervals for TSH, fructosamine, albumin, ACF, and FAR in normal third-trimester pregnancy.

Methods: A cross-sectional study was conducted among 120 healthy pregnant women without thyroid disorders, diabetes, GDM, or other comorbidities. Serum TSH, fructosamine, and albumin were measured using Alinity i and Beckman Coulter AU480 analyzers. ACF and FAR were calculated using published formulas. Reference intervals were established according to CLSI EP28-A3c guidelines using the non-parametric method (2.5th–97.5th percentile).

Results: TSH ranged from 1.2–3.8 µIU/mL (2.51 ± 0.67) and was within the standard range (0.35–4.94 µIU/mL). Fructosamine ranged from 181–280 µmol/L (215.3 ± 21.56) versus 205–280 µmol/L. Albumin ranged from 3.0–5.0 g/dL (3.72 ± 0.38) versus 3.5–5.2 g/dL. ACF ranged from 180–275 µmol/g (228.46 ± 24.13) versus 205–280 µmol/g. FAR ranged from 48–72 µmol/g (59.84 ± 5.92) versus 54–86 µmol/g. Fructosamine, albumin, ACF, and FAR showed relatively lower values than standard reference ranges.

Conclusion: This study establishes population-specific biological reference intervals for TSH, fructosamine, albumin, ACF, and FAR in third-trimester pregnancy. These differences may reflect physiological metabolic adaptations during pregnancy and support pregnancy-specific laboratory interpretation.

Keywords: *Pregnancy; Biological reference intervals; Metabolic biomarkers; TSH; Fructosamine; ACF; FAR.*

P16 POSTER

Deciphering the Cardiovascular Relevance of OR51X1: Insights from Disease-Associated Expression

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G-protein-coupled receptors (GPCRs) constitute a major class of membrane signaling proteins involved in cardiovascular homeostasis and disease. Although considerable attention has been directed toward classical cardiovascular GPCRs, the contribution of non-canonical receptors, including olfactory receptors expressed in extra-olfactory tissues, remains largely unexplored. OR51X1 is an orphan olfactory receptor whose potential involvement in cardiovascular signaling has not yet been defined. Cellular and animal approaches were combined to investigate the association of OR51X1 with cardiovascular disease-related conditions.

OR51X1 expression was examined in H9c2 cardiomyoblasts and vascular smooth muscle cells (VSMCs) under normoxic and hypoxic conditions to assess its response to cellular stress. Hypoxia-associated changes in OR51X1 expression were observed in these cellular models. In parallel, analysis of aortic tissues from normotensive, L-NAME-induced hypertensive, and enalapril-treated hypertensive Wistar rats revealed reduced OR51X1 expression in the hypertensive condition, with treatment-associated changes following antihypertensive intervention. These observations suggest that OR51X1 expression may be responsive to cardiovascular stress and altered during hypertension.

By integrating expression-based functional investigations, this work seeks to advance understanding of OR51X1 as a non-canonical GPCR and to evaluate its potential significance in cardiovascular pathophysiology.

Keywords: *Olfactory receptor, Non-canonical GPCR, Cardiovascular disease, Hypertension, Hypoxia*

P17 **POSTER**

The Rho/ROCK Signaling Pathway: ROCK Inhibitors as Potential Therapeutic Targets in Atherosclerosis

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Introduction: Atherosclerosis is a chronic, progressive vascular disorder contributing to cardiovascular disease. Management targets modifiable risk factors, including dyslipidemia, hypertension, obesity, and inflammatory and pro-thrombotic states. Although these conventional pharmacotherapies substantially reduce morbidity and mortality, residual cardiovascular risk remains considerable.

Rho activation and its downstream kinases (ROCK1 and ROCK2) contribute to endothelial dysfunction, inflammatory signaling, oxidative stress, and plaque remodeling in atherosclerosis. Thus, inhibition of the ROCK signaling pathway may attenuate multiple stages of atherosclerotic disease.

Objectives: To elucidate the role of the Rho/ROCK signaling pathway in atherosclerosis pathogenesis.

To critically review the potential of Rho-kinase inhibition as a newer therapeutic target in atherosclerosis.

To explore the emerging roles of ROCK isoforms in the molecular mechanisms involved in ROCK-targeted therapy.

Materials and Methods: In this review, the signaling pathway of Rho-kinase and the therapeutic potential of ROCK inhibition have been explored through an extensive and systematic search across several electronic databases, including PubMed, Scopus, and ClinicalTrials.gov.

Results: The RhoA/ROCK signaling pathway plays a critical role in the pathogenesis of atherosclerosis. Preclinical studies show that ROCK inhibition can attenuate these processes and promote endothelial function, reduce vascular inflammation and oxidative stress, and limit atherosclerosis plaque progression and remodeling. Early clinical studies support the therapeutic potential of ROCK inhibitors, particularly Fasudil. Identifying ROCK isoforms further facilitates selection of targeted therapy.

Conclusion: ROCK-directed therapy may emerge as a new target in atherosclerosis management, bridging molecular vascular mechanisms with precision medicine.

Keywords: *Rho/ROCK, ROCK Inhibitors, Atherosclerosis, Rho-signaling pathway, Fasudil*

P18 POSTER

An Integrated Experimental and Structure-Guided Investigation of Pentraxin 3 in Cardiovascular Inflammation

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Introduction: Pentraxin 3 (PTX3) is an inflammation-associated long pentraxin implicated in vascular and cardiovascular disease. Although PTX3 has emerged as a potential biomarker and molecular mediator of cardiovascular inflammation, its molecular interactions and potential as a therapeutic target remain incompletely understood. This study aims to investigate the molecular relevance of PTX3 in cardiovascular inflammation and explore its potential as a molecular target through an integrated experimental and computational approach.

Materials and Methods: PTX3 expression is being investigated at the transcript and protein levels in the context of coronary artery disease, supported by analysis of publicly available transcriptomic datasets. A PTX3 expression construct was generated and validated to establish an experimental platform for subsequent functional studies. The interaction of PTX3 with C1q was investigated using molecular docking, followed by structure-based virtual screening to identify potential PTX3-binding compounds. Selected leads were further evaluated using pharmacological profiling and molecular dynamics simulations.

Results: Transcriptomic analysis demonstrated differential PTX3 expression in cardiovascular disease-associated datasets, supporting its relevance to cardiovascular inflammatory processes. The PTX3 expression construct was successfully generated and validated. Structural analysis predicted a favourable PTX3-C1q interaction, while virtual screening identified candidate compounds with favourable predicted binding characteristics. Molecular dynamics simulations indicated overall stability of the ligand-associated systems and identified key PTX3 residues contributing to ligand interactions.

Conclusion: The integrated approach connects the cardiovascular relevance of PTX3 with its molecular interactions and structure-guided ligand discovery. The study provides a framework for further experimental validation of PTX3-associated mechanisms and candidate compounds, supporting continued investigation of PTX3 as a potential molecular target in cardiovascular inflammation and atherosclerotic disease.

Keywords: *PTX3; coronary artery disease; atherosclerosis; molecular dynamics; drug discovery.*

P19 POSTER

Rotundic acid protects against cyclophosphamide-induced hepatic damage in Swiss albino mice: Integrated biochemical, immunohistochemical, and histopathological evidences

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Background: Cyclophosphamide (CP) is a nitrogen mustard alkylating agent extensively used in cancer therapy. However, its clinical application is associated with severe hepatotoxicity. CP undergoes rapid hepatic metabolism to form phosphoramide mustard (PM), which mediates its anticancer activity, and acrolein, a toxic metabolite that contributes considerably to CP-induced hepatic injury.

Objective: To assess the protective effect of rotundic acid (RA) against cyclophosphamide (CP)-induced liver injury in Swiss albino mice.

Materials and Methods: Animals were separated into five groups; Normal control, CP 200, RA 40 *per-se*, RA10 + CP 200, and RA 40 + CP 200. RA (10, 40 mg/kg, p.o.) were given daily for 14 days and CP 200 mg/kg/i.p, once on the 7th day. On the 15th day animals were weighed, sacrificed, blood was withdrawn, and livers were collected to assess the biochemical and histopathological parameters.

Results: CP 200 mg/kg/i.p. enhanced the level of ALT, AST, GGT, TNF-α, interleukin-6, and reduced the level of CAT, and SOD, resulting in hepatic toxicity due to liver injury, oxidative stress and inflammation. Beside this CP also increased the level of cleaved caspase-3 which further substantiated hepatic-toxicity. The concurrent administration of rotundic acid substantially reversed these pathological modifications towards normal.

Conclusion: The findings suggest that RA has potential to be used as an adjuvant to mitigate cyclophosphamide-induced toxicity, however, further clinical investigation is needed to substantiate this fact.

Keywords: *Cyclophosphamide, Rotundic acid, oxidative stress, inflammation, hepatic toxicity*

P20 **POSTER**

Impact of Faecal Microbiota Transplantation on Transcriptomic Signatures of Gut Dysbiosis in Diabetes Mellitus

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Type 2 diabetes mellitus (T2DM) is a complex metabolic disorder characterized by hyperglycaemia, insulin resistance, β-cell dysfunction, and chronic low-grade inflammation, with increasing evidence implicating gut microbiota dysbiosis in its pathogenesis. The present study investigated the therapeutic potential of faecal microbiota transplantation (FMT) in ameliorating diabetes-associated dysfunctions using a high-fat diet (HFD) and streptozotocin (STZ)-induced rat model of T2DM. Male Wistar rats were allocated into three groups: normal control (NC), diabetic control (DC), and diabetic + FMT treatment (DC+FMT). Diabetes was induced by HFD feeding for four weeks followed by a single STZ injection (45 mg/kg), and FMT was administered daily via oral gavage using faecal suspensions from healthy donor rats. Physiological endpoints (fasting blood glucose, body weight) were monitored longitudinally, and targeted transcriptomic profiling of pancreatic tissue was performed using quantitative real-time PCR to evaluate seven candidate genes implicated in inflammation, barrier integrity, lipid metabolism, and detoxification (ADAMDEC1, AGR2, APOC3, AQP10, CYP3A4, MSMB, MUC2). Diabetic control rats exhibited persistent hyperglycaemia, impaired weight gain, and marked transcriptional dysregulation, including upregulation of inflammatory and metabolic stress markers (ADAMDEC1, APOC3, CYP3A4) and suppression of immune-regulatory and barrier-associated genes (MSMB, MUC2, AGR2, AQP10). In contrast, FMT-treated rats showed a progressive decline in fasting blood glucose, partial restoration of body weight, and transcriptional reprogramming towards normal levels. Specifically, FMT downregulated ADAMDEC1 and APOC3, restored MSMB, MUC2, AGR2, and AQP10 expression, and normalized CYP3A4, indicating reduced inflammation, improved barrier integrity, and balanced lipid and xenobiotic metabolism. These findings provide strong preclinical evidence that FMT ameliorates metabolic dysfunction in T2DM by restoring microbial–host interactions, reducing systemic inflammation, and reprogramming host gene expression. The study underscores the therapeutic potential of microbiota-targeted interventions as adjuncts to conventional diabetes management and establishes a mechanistic framework for future translational and clinical investigations.

Keywords: *Type 2 diabetes mellitus; Faecal microbiota transplantation; Gut dysbiosis; Transcriptomics; Inflammation*

P21 POSTER

Molecular Mechanisms Underlying miR-145 Downregulation in Thrombosis

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Background: Thrombosis is a multifactorial vascular disorder involving dysregulation of endothelial and immune cell responses and ultimately activation of coagulation pathways. MicroRNAs are important post-transcriptional regulators of vascular and inflammatory processes, such as, miR-145 which is well known for regulation of coagulation-associated genes, including tissue factor (F3). Downregulation of miR-145 is well established with thrombosis and increased thrombotic burden, supporting its role as an important endogenous antithrombotic regulator. However, the regulatory mechanisms controlling miR-145 expression and processing during thrombotic conditions remain poorly defined.

Objectives: The objective of this study was to investigate the regulatory mechanisms governing altered miR-145 expression and maturation in the context of thrombosis, with particular emphasis on the transcriptional and post-transcriptional regulation of the pri-miR-145/pre-miR-145/mature miR-145 pathway and its potential relationship with tissue factor-mediated thrombotic signaling.

Methods: In silico analyses were performed to characterize DNA methylation patterns at the MIR-145 promoter and to predict RBPs interacting with the full-length pri-miR-145 transcript using structure-aware binding algorithms. Candidate RBPs were prioritized based on functional relevance, m6A-reader activity, and reported dysregulation under thrombosis-associated stress conditions. Integration of epigenetic and RBP-interaction datasets was used to construct a unified regulatory model. Experimental validation of key interactions is currently in progress.

Results: Computational epigenetic analysis revealed altered DNA methylation patterns at the MIR-145 promoter, consistent with potential transcriptional regulation. RBP interactome profiling identified a highly competitive binding landscape enriched for RNA-binding proteins, with several candidates showing potential interactions with the pri-miR-145 transcript. Candidate RBPs were prioritized based on their predicted interaction with regulatory regions of pri-miR-145, functional relevance, and potential involvement in miRNA processing and stress-responsive regulatory pathways. These findings support a model in which epigenetic regulation and RBP-mediated post-transcriptional control converge to modulate miR-145 expression and processing, providing a potential molecular mechanism underlying miR-145 dysregulation in thrombosis.

Conclusion: This study proposes a regulatory framework linking MIR-145 transcription, pri-miR-145 processing and mature miR-145 abundance to thrombotic signaling. Identification of RBP-mediated regulatory checkpoints may provide new insight into the molecular control of miR-145 during thrombosis and establish a basis for investigating the miR-145 and F3 axis as a potential regulatory pathway in thrombotic disease.

Keywords: *miR-145, MIR-145, Tissue Factor, F3, RNA-binding proteins, miRNA processing, vascular homeostasis, coagulation.*

P22 POSTER

Fisetin Protects the Heart Against Isoproterenol-Induced Injury by Targeting RAGE/NF-κB–driven Oxidative Stress, Inflammation, and Apoptosis

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Background: Fisetin is a naturally occurring flavonoid found in fruits such as strawberries and apples. We investigated whether fisetin could protect the heart against isoproterenol (ISO)-induced cardiac injury in rats and explored the molecular mechanisms involved.

Study Design: The study included 48 male Wistar rats. Fisetin was given orally at doses of 10, 20, or 40 mg/kg/day for 28 days. To induce cardiac injury, isoproterenol (85 mg/kg, subcutaneously) was administered twice at a 24-hour interval on days 27 and 28. On day 29, blood pressure and left ventricular function were measured, followed by biochemical, histological, and molecular analyses.

Fisetin reduced oxidative stress by restoring the balance between oxidants and antioxidants in myocardial tissue. It also reduced apoptosis, or programmed cell death. ISO increased the pro-apoptotic proteins Bax, caspase-3, and cytochrome-c and decreased the anti-apoptotic protein Bcl-2. Fisetin reversed these changes.

At the molecular level, the cardioprotective effects were associated with suppression of the RAGE/NF-κB signaling pathway, which is involved in oxidative stress and inflammatory responses. Interestingly, the highest fisetin dose, 40 mg/kg, did not provide similar protection and produced detrimental changes resembling those observed in the ISO-treated group.

Results: The study suggests that fisetin at appropriate doses can reduce ISO-induced cardiac injury by suppressing RAGE/NF-κB-mediated oxidative stress, inflammation, and apoptosis.

Conclusion: Hence, through our study, the role of fisetin in cardioprotection has been uncovered via a molecular pathway.

Keywords: *Apoptosis; Fisetin; Inflammation; Isoproterenol; Oxidative stress; RAGE.*

P23 **POSTER**

Effect of Cardiac Rehabilitation on Functional Capacity and Pulmonary Function in Patients after Coronary Artery Bypass Graft: A Systematic Review

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Purpose: This review aims to systematically evaluate the effectiveness of cardiac rehabilitation (CR) in improving functional capacity and pulmonary function in patients after coronary artery bypass grafting (CABG).

Methods: A systematic search was performed at PubMed, Google Scholar, and ScienceDirect databases, from March 2014 to December 2024, to retrieve relevant literature. Studies were eligible if they included adults (≥18 years) diagnosed with coronary artery disease and treated with CABG, and employed a randomized controlled or quasi-experimental design.

Results: A total of 39,164 studies were retrieved, of which 57 full-text articles were assessed for eligibility according to the specified criteria. After filtering, eight articles were included in the present review. The quality assessment revealed fair-quality evidence. CR was associated with a significant improvement in functional capacity, while pulmonary function outcomes showed mixed results, with only one RCT demonstrating significant improvements in FVC (Forced Vital Capacity) and FEV_1 (Forced Expiratory Volume in the 1st second) at hospital discharge.

Conclusion: CR appears to be effective in improving functional capacity in patients following CABG, as evidenced by consistent improvements in 6-minute walk distance across most studies. However, its effect on pulmonary function remains inconclusive; further high-quality RCTs with standardized CR protocols, adequate sample sizes, and longer follow-up are needed to support the use of a superior protocol.

Keywords: *Cardiac Rehabilitation; Exercise Therapy; Coronary Artery Bypass; Functional Capacity; Quality of Life*

P24 POSTER

Phenotypic and Metabolic Characterization of Isoproterenol-Induced Myocardial Injury Across Experimental Exposure Paradigm

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Introduction: Isoproterenol (ISO)-induced myocardial injury is widely used as a preclinical model of myocardial necrosis however, metabolic alterations associated with different ISO exposures remain incompletely characterized. This study investigated the effects of varying ISO doses and exposure frequency on cardiac injury, hemodynamic alterations, myocardial pathology, and metabolic perturbations in adult male Wistar rats.

Methods: Adult male Wistar rats (n=32) were allocated into four groups (n=8/group): Sham, ISO 85 mg/kg single dose (ISO 85-1D), ISO 85 mg/kg administered as two doses 24 h apart (ISO 85-2D), and ISO 150 mg/kg single dose (ISO 150-1D), administered subcutaneously. Animals were evaluated 24 h after the last ISO or saline injection for hemodynamic parameters, cardiac injury markers, biochemical alterations, and histopathological and ultrastructural changes. Untargeted LC-MS/MS-based metabolomic profiling was performed, followed by normalization, multivariate analysis, and pathway enrichment using MetaboAnalyst 6.0.

Results: ISO administration produced myocardial injury, with alterations in hemodynamic parameters and increased CK-MB and LDH levels. Histopathological and ultrastructural analyses demonstrated myocardial structural alterations across ISO-treated groups, varying according to dose and exposure pattern. Metabolomic profiling revealed distinct metabolic signatures. Altered pathways included arginine biosynthesis/arginine and proline metabolism, alanine, aspartate and glutamate metabolism, taurine and hypotaurine metabolism, glutathione metabolism, and the citrate cycle, indicating perturbations in amino-acid metabolism, antioxidant defense, and cellular energy metabolism.

Conclusion: Varying ISO dose and exposure frequency produced distinct phenotypic and metabolic alterations associated with myocardial necrosis. Integrated cardiac injury and metabolomic assessment provides comprehensive characterization of ISO-induced myocardial injury and identifies metabolic pathways associated with disease development.

Keywords: *Isoproterenol; Myocardial necrosis; Myocardial injury; Metabolomics; Metabolic pathways*

P25 POSTER

Effect of Iron Deficiency Anaemia on HbA1C and Serum Insulin Levels Among Non-Diabetic Individuals

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Background: Anaemia is a significant public health challenge worldwide, affecting more than 30% of the global population. Iron deficiency anaemia (IDA) develops when iron availability is insufficient due to inadequate dietary intake, blood loss, or impaired intestinal absorption. Glycated haemoglobin (HbA1c), which constitutes approximately 5% of total haemoglobin in non-diabetic individuals, is widely

used as an indicator of long-term glycaemic status. However, iron deficiency may influence HbA1c levels independently of blood glucose concentrations, potentially affecting its clinical interpretation.

Methods: This hospital-based prospective analytical study included 200 non-diabetic participants, comprising 100 individuals with iron deficiency anaemia and 100 age- and sex-matched healthy controls. Haematological parameters, iron profile, HbA1c, and fasting serum insulin levels were measured using standard automated analytical methods. Statistical analysis was performed using SPSS version 24. A p-value < 0.05 was considered statistically significant.

Results: The two groups were comparable in terms of age; however, females were significantly more predominant in the IDA group. Significant differences were observed between the two groups in haemoglobin, red blood cell indices (MCV, MCH, and MCHC), serum iron, ferritin, transferrin saturation, TIBC, HbA1c, and fasting serum insulin levels (all $p < 0.001$). In contrast, fasting and postprandial blood glucose levels did not differ significantly between the groups. Serum haemoglobin showed a strong positive correlation with HbA1c ($R = 0.827$, $p < 0.001$) and a weak but statistically significant positive correlation with fasting serum insulin ($R = 0.161$, $p = 0.022$).

Conclusion: Iron deficiency anaemia significantly affects HbA1c and fasting serum insulin levels in non-diabetic individuals despite the absence of significant differences in blood glucose levels. The strong association between haemoglobin and HbA1c, along with the weak association between haemoglobin and fasting serum insulin, highlights the importance of considering iron status when interpreting HbA1c values in non-diabetic individuals.

Keywords: *Iron Deficiency Anaemia; HbA1c; Serum Insulin; Non-Diabetic Individuals; Iron Profile; Red Blood Cell Indices.*

P26 **POSTER**

Atherosclerosis: Mechanisms of LDL-Driven Inflammation and Therapeutic Targets

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Atherosclerosis which we all know about which results in plaque formation and the key role for the formation of that plaque is Low-Density Lipoprotein (LDL) which is the central driver of atherosclerosis, a complex, active inflammatory disease rather than a passive accumulation of arterial plaque. The biological pathogenesis initiates with endothelial dysfunction, allowing excess circulating LDL to penetrate the intima. Here, it becomes chemically trapped via ionic bonds between ApoB-100 and proteoglycans. Once trapped, LDL undergoes oxidation, triggering an immune cascade where macrophages indefinitely consume oxidized LDL via unregulated scavenger receptors. This creates lipid-engorged foam cells. Due to defective efferocytosis, these cells undergo secondary necrosis, spilling toxic contents to form a highly inflammatory necrotic core covered by a fibrous cap. If this cap ruptures, it exposes thrombogenic material to the bloodstream, precipitating critical clinical events like myocardial infarction or stroke.

To cure this lipid toxicity and cardiovascular risk, PCSK9 inhibitors have emerged as highly effective targeted therapies. By inhibiting the PCSK9 protein, these agents prevent the degradation of hepatic LDL receptors, dramatically increasing the clearance of circulating LDL. Clinical studies indicate that PCSK9 inhibitors reduce LDL-C concentrations by 60% to 70% in vivo. This significant reduction translates to improved clinical outcomes, effectively lowering cardiovascular morbidity and all-cause mortality. Furthermore, PCSK9 inhibitors provide a safe and vital alternative for patients unable to reach target cholesterol levels due to statin intolerance.

Keywords: *LDL, Low-Density Lipoprotein, Atherosclerosis, Foam Cells, PCSK9 Inhibitors, Cardiovascular Risk.*

P27 POSTER

Global Longitudinal Strain as an Early Marker of Myocardial Dysfunction in Coronary Artery Disease

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Introduction: Coronary artery disease (CAD) is a major cause of myocardial dysfunction and heart failure. Left ventricular ejection fraction (LVEF) is commonly used to assess cardiac systolic function; however, LVEF may remain preserved despite early myocardial abnormalities. Global Longitudinal Strain (GLS), assessed using two-dimensional speckle-tracking echocardiography (2D-STE), provides a sensitive measure of myocardial deformation and may detect subtle myocardial dysfunction before a significant reduction in LVEF occurs.

Materials and Methods: A prospective case series was conducted in 8 patients with coronary artery disease (CAD). Patients underwent routine clinical evaluation and transthoracic echocardiography. Left ventricular ejection fraction (LVEF) was assessed using conventional echocardiographic methods, while Global Longitudinal Strain (GLS) was measured using two-dimensional speckle-tracking echocardiography from standard apical views. GLS values were analyzed in relation to LVEF to assess early left ventricular myocardial dysfunction.

Results: A total of 8 patients with CAD underwent clinical assessment and transthoracic echocardiography. LVEF was assessed using conventional echocardiography, while GLS was measured using 2D speckle-tracking echocardiography. Reduced GLS was observed in patients with preserved or near-normal LVEF, indicating subtle myocardial dysfunction. GLS provided additional information on left ventricular systolic function beyond conventional LVEF assessment. These findings support the potential role of GLS as an early marker of myocardial dysfunction in CAD.

Conclusion: GLS detected early myocardial dysfunction in CAD patients despite preserved or near-normal LVEF. It may provide additional information beyond conventional LVEF and serve as a useful early marker of left ventricular dysfunction.

Keywords: *Global longitudinal strain; Coronary artery disease; Speckle-tracking echocardiography; Left ventricular function; Myocardial dysfunction.*

P28 POSTER

Nanobots: The Future of Reversing Atherosclerosis Without Surgery

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Atherosclerosis is a chronic inflammatory disease of the arteries, the underlying cause of about 50% of all deaths in westernized society. It is principally a lipid-driven process initiated by the accumulation of low-density lipoprotein and remnant lipoparticles, and an active inflammatory process in focal areas of arteries, particularly at regions of disturbed non-laminar flow at branch points in the arteries, and is considered a primary cause of atherosclerotic cardiovascular disease (ASCVD), resulting in heart attacks, stroke and peripheral arterial disease. In this review, we outline the vascular biology, sequelae, and potential management. It is caused by diabetes, family history, high blood pressure, poor lifestyle, older age etc. Treatments include traditional pharmacological treatment — statins, fibrates, ezetimibe, PCSK9 inhibitors, antiplatelet therapies, hypertensive management therapies and diabetes mellitus therapy. Revascularization interventions: PCI, CABG, endarterectomy. Emerging anti-atherosclerosis therapies: anti-inflammation therapy, nucleic acid-based therapy, immunomodulating therapy, nanotechnology-based therapy. As all these treatments include some of these side effects — poor

tolerance, gastrointestinal side effects, myopathy and rhabdomyolysis, neurocognitive effects, invasive surgery, long trauma, strict surgical indication — adverse effects indicate a need for personalised approaches and many more. Nanobots, which are biologically compatible robots called nanobio-robots, are machines between 1 and 100 nm in dimensions and engineered to function at a scale that facilitates direct interaction within cells. The concept of "Fantastic Voyage" was conceived by Feynman, 1959, Fleischer, 1966. Its applications include biosensors, targeted drug delivery, and diagnosis. It is still facing many challenges in proper implementation, including size, biocompatibility, transportation, and control power supply, etc.

Keywords: *Atherosclerosis, Nanobots, Targeted Drug Delivery, Biosensors, Biocompatibility, Nanotechnology based therapy.*

P29 **POSTER**

Isoproterenol-Induced Alterations in Myocardial Antioxidant Defense, Hemodynamics, and Left Ventricular Contractile Function in Rats

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Introduction: Oxidative stress contributes to myocardial injury and impaired cardiac function. Isoproterenol (ISO), a synthetic β-adrenergic agonist, produces oxidative stress and myocardial damage at high doses. This study evaluated ISO-induced changes in myocardial antioxidant defense, systemic hemodynamics, and left ventricular function in rats.

Materials and Methods: Rats received subcutaneous ISO (85 mg/kg) on two consecutive days at a 24-h interval. Myocardial oxidative stress was assessed by superoxide dismutase (SOD), catalase, glutathione peroxidase (GPx), reduced glutathione (GSH), and thiobarbituric acid-reactive substances (TBARS). Cardiac injury was assessed using CK-MB and LDH. Hemodynamic and left ventricular parameters included arterial pressure, heart rate, rate-pressure product, LVEDP, +LVdP/dt, and −LVdP/dt.

Results: ISO significantly reduced myocardial SOD, catalase, GPx, and GSH, with increased TBARS, indicating enhanced oxidative stress and lipid peroxidation. CK-MB and LDH activities were depleted, consistent with cardiomyocyte injury. ISO also decreased arterial pressure, heart rate, rate-pressure product, +LVdP/dt, and −LVdP/dt, while increasing LVEDP, demonstrating impaired ventricular contractile and relaxation function.

Conclusions: High-dose ISO induces myocardial oxidative stress, weakens endogenous antioxidant defenses, and produces marked hemodynamic and left ventricular dysfunction. These findings support the utility of this model for evaluating cardioprotective interventions.

Keywords: *Isoproterenol; Oxidative stress; Left ventricular dysfunction; Cardiomyocyte injury; Cardioprotection*

P30 **POSTER**

Relationship of kisspeptin levels with insulin resistance and glycemic control in adults with Type 2 Diabetes Mellitus

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Introduction: Kisspeptin, encoded by the KISS1 gene, is well established as a regulator of GnRH release and pubertal onset; however, its role in glucose homeostasis remains unclear, with conflicting evidence regarding insulin secretion. Given the rising burden of Type 2 Diabetes Mellitus (T2DM) in India and limited data on kisspeptin in Indian populations, this study investigated its relationship with insulin resistance and glycemic control. Serum kisspeptin levels were compared between adults with T2DM and healthy controls and assess their correlation with insulin resistance (HOMA-IR), fasting blood glucose (FBG), and HbA1c.

Methods: This cross-sectional study included 80 adults with T2DM and 80 age-, sex-, and socioeconomically matched healthy controls. Serum kisspeptin-1 was measured by ELISA, plasma glucose by the hexokinase method, HbA1c by HPLC, and insulin resistance by HOMA-IR. Group comparisons were performed using Student's t-test, and correlations were assessed using Pearson's correlation analysis.

Results: Serum kisspeptin levels were significantly higher in T2DM patients than in healthy controls (1427.5 ± 327.5 vs. 752.7 ± 191.8 pg/mL; $p < 0.05$). Kisspeptin showed a significant positive correlation with insulin resistance ($r = 0.629$, $p < 0.05$). Correlations with FBG ($r = 0.117$, $p = 0.299$) and HbA1c ($r = 0.119$, $p = 0.293$) were weak and non-significant.

Conclusion: Serum kisspeptin levels were elevated in adults with T2DM and significantly associated with insulin resistance, but not with FBG or HbA1c. These findings indicate a potential association between kisspeptin and insulin resistance in T2DM and warrant further mechanistic and longitudinal investigation.

Keywords: *Kisspeptin; KISS1; Type 2 Diabetes Mellitus; Insulin Resistance; HOMA-IR; Glycemic Control; HbA1c; Fasting Blood Glucose.*

P31 **POSTER**

Statin Intolerance in Subclinical Atherosclerosis: A Case-Based Pharmacotherapeutic Analysis

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Introduction: Statins are central to the pharmacological management of atherosclerotic cardiovascular disease because of their low-density lipoprotein cholesterol (LDL-C)-lowering effects. However, statin-associated muscle symptoms may complicate treatment and influence long-term adherence. This case-based analysis describes the therapeutic challenge of statin intolerance in a patient with hyperlipidemia and subclinical coronary atherosclerosis and considers its implications for cardiovascular risk management.

Materials and Methods: A published clinical case describing a middle-aged woman with hyperlipidemia, subclinical coronary atherosclerosis and intolerance to multiple statin regimens was examined. Clinical history, lipid parameters, coronary calcium findings, treatment attempts and adverse-effect profile were reviewed to identify the principal pharmacotherapeutic considerations.

Results: The patient had total cholesterol of 253 mg/dL and LDL-C of 172 mg/dL, with a coronary artery calcium score of 49 localized to the left anterior descending artery. Rosuvastatin 20 mg daily produced thigh aching and was discontinued, followed by similar symptoms with atorvastatin 40 mg and intermittent simvastatin. Creatine kinase remained within the normal range during the reported episodes. The case illustrates the difficulty of balancing LDL-C reduction, tolerability and adherence when multiple statin regimens produce muscle symptoms.

Conclusions: Statin intolerance can complicate preventive management even when objective evidence of subclinical atherosclerosis is present. Individualized assessment of symptoms, treatment history and cardiovascular risk is important when selecting an ongoing lipid-lowering strategy.

Case basis: American College of Cardiology, "A Middle-Aged Woman With Hyperlipidemia, Subclinical Atherosclerosis, and Statin Intolerance."

Keywords: *Atherosclerosis; Statins; Statin intolerance; LDL-C; Cardiovascular risk*

P32 **POSTER**

Association of Soluble fms-like Tyrosine Kinase-1 (sFlt-1), Placental Growth Factor (PlGF), 8-Isoprostane and Endothelin-1 Markers with Gestational Obstructive Sleep Apnea and Maternal-Fetal Outcomes

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Background: Gestational obstructive sleep apnea (OSA) is increasingly recognized as an important pregnancy-related sleep disorder associated with adverse maternal and fetal outcomes. Intermittent hypoxia associated with OSA may contribute to oxidative stress, endothelial dysfunction, and placental angiogenic imbalance. However, the combined association of these pathophysiological pathways with gestational OSA and pregnancy outcomes remains inadequately studied, particularly in the Indian population.

Objective: To evaluate the association of soluble fms-like tyrosine kinase-1 (sFlt-1), placental growth factor (PlGF), 8-isoprostane, and endothelin-1 (ET-1) with gestational OSA and maternal-fetal outcomes.

Methods: A prospective case-control study will be conducted among pregnant women aged 19–40 years with singleton pregnancies at 24–32 weeks of gestation. Participants will be screened for OSA using the STOP-BANG questionnaire, Berlin Questionnaire, Epworth Sleepiness Scale, and Mallampati score. Women identified as having gestational OSA based on the screening criteria will be compared with individually matched non-OSA controls. Approximately 5–8 mL of maternal venous blood will be collected, and serum concentrations of sFlt-1, PlGF, 8-isoprostane, and ET-1 will be estimated using ELISA. Maternal outcomes, including gestational hypertension, preeclampsia, and gestational diabetes mellitus, and fetal/neonatal outcomes, including preterm birth, fetal growth restriction, low birth weight, Apgar score, and NICU admission, will be assessed. A total of 140 participants (70 cases and 70 controls) is planned.

Expected Results: It is anticipated that women with gestational OSA will demonstrate altered biomarker levels and that these alterations will be associated with adverse maternal and fetal outcomes.

Conclusion: Integrated assessment of angiogenic, oxidative stress, and endothelial dysfunction biomarkers may improve understanding of the biological mechanisms linking gestational OSA with adverse pregnancy outcomes and may support early risk stratification.

Keywords: *Gestational obstructive sleep apnea; sFlt-1; PlGF; Endothelin-1; 8-Isoprostane*

P33 POSTER

Effects of Exercise-Based Rehabilitation on Spirometric Outcome in Adults with Bronchiectasis

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Introduction: Evidence on the effects of exercise-based rehabilitation on spirometric outcomes, particularly FEV_1 and FVC in bronchiectasis remains limited. This systematic review and meta-analysis therefore evaluated its impact on these pulmonary outcomes.

Materials and Methods: Following PRISMA 2020, PubMed, Web of Science, Scopus, and PEDro were searched from inception to 16 September 2026. Two reviewers (SB, AS) independently screened, extracted, and assessed bias; a third author (AM) resolved disagreements. RoB 2 was applied to randomised controlled trials (RCTs), and ROBINS-I and the NIH Quality Assessment Tool to non-randomised studies. Pooled mean differences (MDs) with 95% confidence intervals (CIs) were estimated using random-effects models in RevMan Web; heterogeneity was assessed by I^2 (significance: $p<0.05$).

Results: Of 9,947 records, 4 studies (2 RCTs, 2 non-RCTs) were included. Qualitative analysis depicted high risk of bias in one RCT and some concerns for the other; among non-randomised studies, one had serious risk (ROBINS-I), the other poor quality (NIH). Short-term effects (MD [95% CI]) were non-significant: FEV_1 (-0.00 L [-0.17, 0.17]; p=0.97; I^2=0%) and FVC (0.08 L [-0.03, 0.19]; p=0.97; I^2=0%), whereas long-term improvements were significant: FEV_1 (0.22 L [0.11, 0.33]; p<0.0001; I^2=61%) and FVC (0.27 L [0.17, 0.37]; p<0.0001; I^2=96%). Overall effects favoured exercise: FEV_1 (0.15 L [0.06, 0.24]; p=0.0008; I^2=59%) and FVC (0.19 L [0.12, 0.26]; p<0.00001; I^2=90%).

Conclusions: Exercise-based rehabilitation improves long-term pulmonary function; short-term effects are favourable but non-significant, warranting cautious interpretation.

Keywords: *Bronchiectasis; Exercise-based rehabilitation; Spirometry; Pulmonary function*

P34 POSTER

Adrenergic Regulation of Hypoxia-Induced Metabolic and Inflammatory Dysfunction in Neuronal and Microglial Cells

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Introduction: Hypoxia is a feature of several cardiovascular and critical-illness conditions and can promote oxidative stress, metabolic dysfunction, and inflammation, contributing to tissue injury. Noradrenergic signaling is an important regulator of cellular stress responses; however, its contribution to hypoxia-induced metabolic and inflammatory alterations in neuronal and microglial cells remains incompletely understood. We investigated the cellular responses to prolonged hypoxia and examined the involvement of adrenergic signaling.

Methods: SH-SY5Y neuronal and N9 microglial cells were exposed to hypoxic conditions (1% O_2) for defined durations. Reactive oxygen species (ROS), mitochondrial membrane potential (JC-1), cellular ATP, and nitrite release were assessed. Expression of mitochondrial stress- and inflammatory-associated genes was evaluated by RT-qPCR. Pharmacological modulation of adrenergic signaling was performed using adrenergic receptor agonists/antagonists. Publicly available transcriptomic data were additionally analyzed to characterize hypoxia-responsive pathways.

Results: Hypoxia produced time-dependent oxidative and metabolic alterations in SH-SY5Y cells, with increased ROS during early exposure and mitochondrial membrane depolarization following prolonged hypoxia. Twenty-four-hour hypoxia was associated with reduced cellular ATP levels, indicating impaired energy homeostasis. In N9 microglial cells, prolonged hypoxia increased nitrite release and was accompanied by increased expression of NOS2, suggesting activation of the inducible nitric oxide pathway. Pharmacological modulation of adrenergic signaling altered hypoxia-induced oxidative and inflammatory responses. Transcriptomic analyses further indicated hypoxia-associated metabolic remodeling and HIF-1-related pathways.

Conclusion: These findings indicate that hypoxia induces coordinated metabolic dysfunction in neuronal cells and inflammatory activation in microglia, while adrenergic signaling modulates these responses. The findings provide mechanistic insight into hypoxia-associated cellular dysfunction at the intersection of cardiovascular stress and neuroinflammation.

Keywords: *Hypoxia; Adrenergic signaling; Neuroinflammation; Mitochondrial dysfunction; Nitric oxide*

P35 **POSTER**

Endothelin-1 Gene Polymorphism, Serum Endothelin-1, Blood Pressure and FEV1 in COPD with and without Hypertension

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Introduction: Hypertension is among the commonest extrapulmonary comorbidities of chronic obstructive pulmonary disease (COPD). Endothelin-1 (ET-1), a potent vasoconstrictor encoded by EDN1, may link vascular disease in COPD with hypertension, and the EDN1 rs5370 polymorphism has been variably associated with ET-1 and hypertension. We compared ET-1 gene polymorphism and serum ET-1, and their relation to blood pressure and FEV1, in COPD with and without hypertension.

Materials and Methods: This cross-sectional study included 212 COPD patients (40–88 years): 106 with hypertension (Group 1) and 106 without (Group 2). Systolic (SBP) and diastolic (DBP) blood pressure and FEV1 (%) were recorded, serum ET-1 was measured by ELISA, and EDN1 rs5370 was genotyped by PCR-RFLP in 60 subjects (30 per group). Data were analysed using chi-square/Fisher's exact test, one-way ANOVA and Pearson correlation ($p<0.05$).

Results: The rs5370 variant was present in 90.0% of Group 1 versus 60.0% of Group 2 ($p=0.007$; Fisher's exact $p=0.015$). Serum ET-1 was higher in Group 1 (8.54±0.76 vs 5.08±1.17 pg/mL, $p<0.001$). Overall mean SBP, DBP and FEV1 were 143.03±25.16 mmHg, 86.18±11.36 mmHg and 46.98±8.44%. ET-1 correlated positively with SBP ($r=0.796$) and DBP ($r=0.806$) and negatively with FEV1 ($r=-0.612$) (all $p<0.001$).

Conclusions: Carriage of the EDN1 rs5370 variant and raised serum ET-1 are associated with hypertension in COPD, and ET-1 is closely linked to blood pressure and airflow limitation, supporting ET-1 as a candidate marker of cardiovascular risk in COPD.

Keywords: *Endothelin-1; EDN1 rs5370; FEV1; COPD; Hypertension*

P36 POSTER

Comparative Evaluation of LDL-C, Non-HDL-C and Atherogenic Index of Plasma in Fasting and Postprandial States: Implications for Cardiovascular Risk Assessment

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Background: Low-density lipoprotein cholesterol (LDL-C) remains a central marker and treatment target for atherosclerotic cardiovascular disease (ASCVD). However, LDL-C represents cholesterol within LDL particles and does not encompass cholesterol carried by other atherogenic lipoproteins. Non-high-density lipoprotein cholesterol (non-HDL-C), calculated as total cholesterol minus HDL-C, captures cholesterol contained in all major atherogenic lipoproteins and is particularly relevant in hypertriglyceridemia and non-fasting samples. The Atherogenic Index of Plasma (AIP), calculated as log10[triglyceride/HDL-C] using molar concentrations, provides an integrated measure of the triglyceride–HDL phenotype.

Objective: To compare LDL-C, non-HDL-C and AIP between fasting and postprandial states and to evaluate their relative utility as complementary markers of lipid-related cardiovascular risk.

Methods: A cross-sectional paired analysis was performed in 200 individuals who underwent fasting and postprandial lipid testing between April to September 2026. Total cholesterol, triglycerides, HDL-C and LDL-C were measured in paired samples. Non-HDL-C was calculated as total cholesterol minus HDL-C. AIP was calculated as log10(TG/HDL-C), after conversion of triglycerides and HDL-C from mg/dL to mmol/L. Paired comparisons were performed using the paired t-test, and Pearson correlation was used to assess relationships between lipid-derived markers. A two-sided p value <0.05 was considered statistically significant.

Results: The study included healthy females and males, with an age range of 18–81 years. Mean fasting versus postprandial concentrations were: total cholesterol, 190.35 ± 53.36 versus 181.82 ± 51.15 mg/dL; triglycerides, 180.31 ± 112.19 versus 208.14 ± 125.97 mg/dL; HDL-C, 43.92 ± 12.59 versus 42.00 ± 12.91 mg/dL; LDL-C, 107.36 ± 38.36 versus 100.96 ± 35.80 mg/dL; and non-HDL-C, 146.43 ± 50.15 versus 139.82 ± 47.83 mg/dL. The mean change was −6.40 mg/dL for LDL-C ($p<0.001$), −6.62 mg/dL for non-HDL-C ($p<0.001$), and +27.83 mg/dL for triglycerides ($p<0.001$). AIP increased significantly from 0.208 ± 0.247 to 0.290 ± 0.254 ($p<0.001$). LDL-C and non-HDL-C demonstrated a strong positive correlation in both fasting ($r=0.922$) and postprandial ($r=0.921$) states. In contrast, AIP showed only a weak correlation with LDL-C, indicating that AIP provides information complementary to LDL-C and non-HDL-C.

Conclusion: In this paired dataset, non-HDL-C demonstrated a fasting–postprandial behavior broadly similar to LDL-C while retaining the advantage of incorporating cholesterol carried by triglyceride-rich atherogenic lipoproteins. AIP demonstrated the largest relative postprandial change, reflecting the influence of triglyceride elevation and HDL-C on this index. These findings support the use of non-HDL-C as a practical complementary marker to LDL-C, particularly in individuals with elevated triglycerides or when non-fasting samples are used, while AIP may provide additional information regarding the triglyceride–HDL-associated atherogenic phenotype. Non-HDL-C should therefore be viewed as complementary to, rather than a replacement for, LDL-C.

Keywords: *LDL-C; non-HDL-C; Atherogenic Index of Plasma; AIP; triglycerides; HDL-C; non-fasting lipid profile; cardiovascular risk; ASCVD.*

P37 POSTER

Beyond Glycaemic Control: Do Oral Hypoglycemic Agents Suppress CD36-Dependent Foam Cell Formation? A Review in Diabetic with Atherosclerosis Risk

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Background: Cardiovascular disease (CVD) is a major complication in diabetes, significantly contributing to patient morbidity and mortality. Type 2 diabetes mellitus (T2DM) is linked to dyslipidaemia, hypertension, insulin resistance, and chronic inflammation, which accelerate atherosclerosis. Reports suggest that dipeptidyl peptidase-4 (DPP-4) inhibitors, primarily used for their antihyperglycemic effects in T2DM may also have anti-atherosclerotic properties. Foam cell formation, facilitated by CD36-mediated uptake of oxidized low-density lipoprotein (oxLDL), is a pivotal process in atherogenesis. CD36 is a significant regulator of lipid homeostasis. CD36 is considered the most important scavenger receptor for taking up ox-LDL in atherosclerotic plaque formation.

Objective: This study aims to analyse the impact of DPP-4 inhibitors on foam cell formation and CD36 expression in CVD and associated morbidities. Methods: Studies were identified and selected through comprehensive searches on PubMed, Google Scholar, and Scopus using the keywords "DPP-4 inhibitors, CD36, T2DM and CVD".

Result: This study seeks to explore whether DPP-4 inhibitors can suppress foam cell formation and CD36 expression in diabetes associated cardiovascular risk. However, more research is needed to conclusively determine if DPP-4 inhibitors have direct anti-atherosclerotic effects independent of their glucose-lowering properties. These studies indicated that this potential benefit may be linked to their impact on macrophage foam cells, which play a crucial role in the development of atherosclerosis.

Summary and Conclusion: This review findings underscore the potential of DPP-4 inhibitors as a therapeutic agent for lowering cardiovascular risk in diabetic patients.

Keywords: *Atherosclerosis, Type 2 diabetes, CD36 expression, Dipeptidyl peptidase-4 (DPP-4) inhibitors.*

P38 POSTER

Investigation of cardio-protective effect of *Juniperus virginiana L.* derived sesquiterpene in cyclophosphamide induced Wistar albino rats

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Background: Worldwide, the occurrence of cancer is alarming. Numerous anticancer medications have been developed to treat cancer and cyclophosphamide (CP) is one of them. Besides anticancer effect, CP also shows cardiotoxicity which is a major concern regarding the usage of this medication. Thus, to lessen toxicity an adjuvant therapy for the cancer patients has to be developed. With this intention we tried to examine *Juniperus virginiana L.* derived sesquiterpene (JVDS) for its cardioprotective potential against the cyclophosphamide induced toxicity in rats.

Objective: To assess the protective effect of sesquiterpene JVDS on cyclophosphamide induced cardio-toxicity in Wistar albino rats.

Methodology: The animals were treated for 14 days after getting divided into four groups (n=6). Control, Cyclophosphamide (CP200); JVDS 40 + CP200; and JVDS 40 *per se*. JVDS was administered as a daily dose for 14 days, with a single dose of CP 200 on the seventh day. The animals were sacrificed on 15th day and blood and heart tissues were collected for biochemical and histopathological examinations.

Result: Administration of cyclophosphamide significantly reduced the activity of CAT and SOD whereas elevated the level of TBARS, pro-inflammatory cytokines (TNF-α and IL-6), cardiac injury markers (LDH, CK-MB and cTnT), and apoptotic marker (caspase-3). Histopathological analysis showed severely damaged cardiac tissue marked by cellular disintegration, pyknosis, vacuolation, and fibrotic changes in CP-treated group which were significantly reversed to normal by our test drug.

Conclusions: JVDS has shown significant cardio protection and attenuated cyclophosphamide-induced cardiotoxicity, effectively.

Keywords: *Cyclophosphamide, Sesquiterpene, Cardio-protection, Oxidative stress, Inflammation.*

P39 **POSTER**

Study of IDRS as a Screening Tool for Identifying the High-Risk Adults for Development of Diabetes: An Interim Analysis

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Introduction: India is among the top 3 countries in the world where adults (20–79 yrs) are suffering from diabetes. More than 77 million people (adults 20–79 yrs) with diabetes are there in India. However, more than 50% of people are undiagnosed for prediabetes or T2DM. These undiagnosed cases may land up into the complications of T2DM if not managed appropriately with an early diagnosis & management (including lifestyle management).

Methods: We conducted an online survey using IDRS (Indian Diabetes Risk Score) as a screening tool for adults who are undiagnosed with either prediabetes or T2DM. We shared an online questionnaire (based on key parameters: age, waist circumference, physical activity & family history of diabetes) with people who provided their consent to participate & shared their responses to our survey questionnaire. This is an interim analysis with survey responses of two hundred one responders. People with IDRS >60 is considered as high risk for development of diabetes.

Result: 36% of people were found to have IDRS >60 & are at high risk of development of diabetes. 18% of people were found to have waist circumference ≥90 cm (female) / ≥100 cm (male) in our survey responders. 77% of people had one or both parent with diabetes. Sedentary lifestyle with no exercise was found in 15% of people.

Conclusion: IDRS can be a cost-effective screening tool for identifying undiagnosed adult people with high risk of development of diabetes. People with high risk (found in screening) should undergo diagnostic testing (HbA1C, FBS, PPBS) to diagnose prediabetes or diabetes.

Keywords: *Indian Diabetes Risk Score (IDRS); Type 2 Diabetes Mellitus; Prediabetes; Diabetes Screening; Risk Assessment*

P40 **POSTER**

Nerolidol as an adjuvant to cyclophosphamide in diethylnitrosamine-induced hepatocellular carcinoma in Wistar rats

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Hepatocellular carcinoma (HCC) is a major cause of cancer-related mortality, often developing in patients with cirrhotic liver disease. Cyclophosphamide (CP), a widely used anticancer drug, is associated with hepatic and systemic toxicities, highlighting the need for adjunct therapies to improve its therapeutic profile. Therefore, this study evaluated nerolidol (NER), a natural sesquiterpene alcohol, alone and in combination with CP in a diethylnitrosamine-induced HCC model in Wistar rats. Forty-two albino rats were divided into seven groups (n=6). Except for the Control, NER, and CP per se groups, all animals received DEN (50 mg/kg, i.p.) once weekly for eight weeks to induce HCC, and followed up to 16th week. Treatment groups were administered NER (400 mg/kg), CP (50 mg/kg), and their combination (400 + 50 mg/kg, p.o.) daily for four weeks. Body, liver weights, number of hepatic nodules, haematological, biochemical, histopathological and immunohistochemistry parameters were assessed. DEN-treated rats showed significant increase in incidence and number of hepatic nodules, along with elevation in serum tumour markers, alpha-fetoprotein (AFP), carcinoembryonic antigen (CEA), liver enzymes (AST & ALP), and reduction in red blood cells and haemoglobin, indicating anaemia and hepatic damage. Decrease in superoxide dismutase confirmed oxidative stress. Treatment with NER, CP, and particularly their combination significantly attenuated DEN-induced alterations, as evidenced by reduced hepatic nodule incidence and number, along with improved haematological, biochemical, histopathological, and immunohistochemical parameters. These findings suggest that NER, CP, and their combination exert chemo-preventive effects against DEN-induced hepatocellular carcinoma in Wistar albino rats, with the combination showing the greatest protective effect.

Keywords: *Hepatocellular carcinoma, Diethyl nitrosamine, Nerolidol, Cyclophosphamide, Wistar rats.*

P41 **POSTER**

Investigating the Role of KCNX in Atherosclerosis and Hypertension Using In Vivo Rat Models

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Cardiovascular diseases (CVDs) involve complex alterations in vascular and cardiac signaling, while the contribution of non-canonical ion channels remains poorly understood. KCNX, a sodium-activated potassium channel, has emerged as a potential regulator of cardiovascular homeostasis; however, its functional significance and structural determinants remain underexplored. This study integrates functional and structural approaches to investigate the role of KCNX in atherosclerosis and hypertension to explore its therapeutic potential. Wistar rat models of high-fat-diet-induced atherosclerosis and L-NAME-induced hypertension were utilized to investigate the expression of KCNX and disease-specific markers. Changes in cardiovascular phenotype, tissue pathology, oxidative stress, and disease-relevant signaling were analyzed in conjunction with KCNX expression to define KCNX-associated molecular responses. KCNX was upregulated under cardiovascular stress conditions, indicating its potential involvement in disease-associated remodeling. Notably, pharmacological activation of KCNX reduced blood pressure in L-NAME-induced hypertensive rats, suggesting a potential protective role for KCNX in regulating cardiovascular function. Collectively, this integrated approach aims to establish KCNX as a potential druggable target in atherosclerosis and hypertension.

Keywords: *KCNX, potassium channel, hypertension, atherosclerosis, cardioprotection*

P42 **POSTER**

Targeting CD80/86–CD28 Signaling in Cardiovascular Remodeling: Molecular Modulation of MAPK, Nrf2/HO-1, and TGF-β/Smad Pathways in Experimental Cardiac Hypertrophy

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Background: Abatacept, a synthetic CTLA4 analogue, inhibits T-cell co-stimulation, reducing inflammation by limiting immune cell infiltration at injury sites. Primarily used for psoriatic diseases and rheumatoid arthritis, its role in isoproterenol (ISO)-induced cardiac hypertrophy in male albino Wistar rats was investigated. This study hypothesized that Abatacept could reduce cardiac T-cells and macrophage infiltration, preventing cardiac hypertrophy. Hemodynamic, structural, biochemical, and molecular analyses were conducted to assess Abatacept's effects, showing its potential in mitigating ISO-induced cardiac hypertrophy.

Methods: In this study, male Wistar albino rats were divided into six groups (n=6): Group 1 (control, PBS), Group 2 (ISO-induced hypertrophy, 3 mg/kg s.c.), and Groups 3-5 (ISO + Abatacept at 2.5, 5, and 10 mg/kg, respectively). Group 6 received only Abatacept (10 mg/kg s.c.). The treatment lasted 40 days. On day 41, post-hemodynamic assessments, blood and hearts were collected to investigate physiological parameters, cardiac injury markers, oxidative stress, inflammation, T-cell infiltration, fibrosis, and apoptosis, evaluating Abatacept's effects on pathological cardiac hypertrophy.

Results: Abatacept reduced cardiac hypertrophy, stabilized heart function, and minimized cardiac injury markers (CK-MB, LDH). Histopathological evidence and decreased inflammatory cytokines supported its benefits. Immunofluorescence and pathway analyses (MAPK/Nrf-2-HO1/Smad-Tgf-beta) confirmed Abatacept's cardioprotective action through anti-apoptotic, anti-necrotic, and anti-inflammatory effects.

Conclusion: Abatacept enhances hemodynamics and mitigates cardiac hypertrophy in ISO-induced dysfunction, offering robust protection against oxidative stress via Nrf-2/HO-1 and combating inflammation through MAPK/NF-κB modulation. It prevents cell damage by inhibiting apoptosis and balancing Bax/Bcl-2/caspase-3 ratios, making it a promising candidate for managing cardiac diseases.

Keywords: *Cardiac Hypertrophy, Abatacept, Fibrosis, Signalling pathways*

P43 **POSTER**

Whole Exome Sequencing Guided Cascade Screening in Long QT Syndrome: Pedigree-Based Case Studies from Indian Familial Cohort

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Long QT Syndrome (LQTS) is a life-threatening, genetically heterogeneous cardiac disorder associated with ventricular arrhythmias, Torsades de Pointes (TdP), and sudden cardiac death (SCD), particularly among young individuals with structurally normal hearts. Early diagnosis remains challenging because of variable penetrance and phenotypic expression, with affected individuals often remaining asymptomatic until the occurrence of a trigger-induced cardiac event. This study aimed to characterise the genetic architecture of LQTS in the Indian population using next-generation sequencing, complemented by Sanger sequencing to investigate variant co-segregation among asymptomatic first-degree relatives (AFDRs). The Indian multicentre familial cohort included clinically affected probands and phenotypically positive first-degree relatives (PPFDRs) who underwent whole-exome sequencing (WES), while familial co-segregation analysis was extended to AFDRs to delineate patterns of inheritance and identify clinically silent carriers. Across the cohort, 204 variant calls were identified involving canonical LQTS genes (*KCNQ1, KCNH2,* and *SCN5A*) as well as non-canonical genes, including *RYR2, SCN10A, and ANK2*. Of these, 104 were classified as pathogenic/likely pathogenic (P/LP) calls, whereas 100 were variants of uncertain significance (VUS) calls, highlighting a substantial diagnostic and interpretative gap. Familial segregation identified 28.2% to be silent carriers, revealed diverse patterns of genetic transmission and phenotypic expression, including a possible additive

contribution of two co-occurring variants associated with a severe phenotype, biparental transmission of a pathogenic variant with incomplete penetrance resulting in identification of asymptomatic carriers, and reclassification of a VUS following incorporation of additional ACMG evidence. These findings underscore the value of integrating genomic analysis with familial screening to enhance diagnosis, variant interpretation, risk stratification, and management of at-risk individuals and silent carriers.

Keywords: *Long QT syndrome; Whole exome sequencing; Cascade screening; Silent carriers; Variant interpretation*

P44 **POSTER**

Association between the blood DNA methylation derived pace of aging and clinical characteristics of HAPE patients

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The epigenome acts like a layer over the genome to control the regulatory programs at the cellular level. Particularly DNA methylation in terms of epigenome has been attributed to the high altitude maladies like High-altitude pulmonary edema (HAPE). DNA methylation-based epigenetic clocks have been widely utilized to estimate biological age and assess the pace of aging in an individual. The pace of aging represents the rate of decline across multiple organ systems and has been associated with several age-related conditions, including cardiovascular diseases. Based on this, we hypothesized that individuals who develop HAPE may have an underlying accelerated biological aging process. To investigate this hypothesis, we designed a case-control study comprising individuals who successfully acclimatized as controls and HAPE patients who experienced acclimatization failure. We generated DNA methylation profiles covering ~930,075 CpG sites using the Illumina Infinium MethylationEPIC v2.0 BeadChip. Biological aging was assessed using multiple epigenetic measures, including the DunedinPoAm and DunedinPACE measures to estimate the pace of aging. We further estimated age acceleration using the residuals obtained by regressing DNAmPhenoAge and DNAmGrimAge2 on chronological age. The epigenetic clocks, including DNAmAge, DNAmAgeHannum, and Zhang's clock, showed good concordance with the reported chronological age of the study participants. Our analysis revealed a significantly increased pace of aging in HAPE patients compared with controls ($p = 0.00036$). Overall, these findings suggest that HAPE is associated with an increased pace of aging, along with a trend towards accelerated epigenetic age.

Keywords: *High-altitude pulmonary edema; DNA methylation; Epigenetic clock; Pace of aging; DunedinPACE*

P45 **POSTER**

Remote ischemic conditioning confers protection against diesel exhaust-induced pulmonary fibrosis in an experimental mouse model

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Remote ischemic conditioning (RIC) is a promising non-pharmacological intervention involving short, non-lethal, and repeated episodes of alternating ischemia and reperfusion applied to a remote organ,

which mitigates ischemic injury in cardiovascular complications and stroke. However, the effects of RIC in pulmonary fibrosis (PF), a progressive and degenerative lung condition with poor survival rates and limited pharmacological treatments, remain poorly understood. Thus, the present study aimed to explore the safety and efficacy of RIC in a diesel exhaust (DE)-induced PF mouse model. The study comprised age-matched mice divided into nine groups, including six experimental groups exposed to DE fumes 5 days/week for 8 weeks to recapitulate PF pathological features. Interventions consisted of optimized RIC given as three cycles of 5 min ischemia followed by 5 min reperfusion, 3 days/week, for four weeks, and a standard pharmacotherapy, pirfenidone. The effect of RIC in the PF model was determined by assessing physiological parameters, circulating fibrotic markers, lung inflammatory profile, mitochondrial dysfunction in lung cells, and lung transcriptome profiling. RIC-receiving groups showed significant improvements in physiological and molecular parameters, as indicated by increased weight and restoration of autonomic balance, and a reduction in fibrotic and inflammatory markers and mitochondrial dysfunction. Lung transcriptome profiling revealed downregulation of PF-progressive genes in RIC-receiving groups and highlighted six potential PF-relevant genes that may be involved in RIC-mediated beneficial effects. Thus, the findings of the present study demonstrated the therapeutic relevance of RIC in the PF model; however, further investigation and validation are warranted.

Keywords: *Remote ischemic conditioning; Pulmonary fibrosis; Diesel exhaust; Pirfenidone; Lung transcriptome*

P46 **POSTER**

Endothelial-to-mesenchymal transition (EndMT) in atherosclerosis: An emerging molecular mechanism

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Atherosclerosis is a chronic inflammatory disease of the arterial wall characterized by endothelial dysfunction, lipid accumulation, inflammation, and plaque formation. Recent research has identified Endothelial-to-Mesenchymal Transition (EndMT) as an important mechanism contributing to vascular remodeling and atherosclerotic progression. During EndMT, endothelial cells gradually lose their normal endothelial identity and acquire mesenchymal-like characteristics. This transition is associated with reduced expression of endothelial markers such as VE-cadherin and CD31 and increased expression of mesenchymal markers including α-SMA, vimentin, and fibronectin.

EndMT is influenced by disturbed blood flow, oxidized LDL, oxidative stress, inflammatory signals, and metabolic changes. Major regulatory pathways include TGF-β/SMAD, Wnt/β-catenin, and Notch signalling. Recent studies indicate that EndMT is not always a simple irreversible switch. Instead, endothelial cells may exist in partial or intermediate transition states, depending on the disease stage and vascular environment.

Emerging research has also linked EndMT with vascular calcification and plaque instability. EndMT-derived cells may acquire osteogenic and chondrogenic characteristics, providing a possible connection between endothelial dysfunction and vascular calcification. Recent studies have further identified SMS2 and SRXN1 as potential molecular targets involved in regulating EndMT and atherosclerotic progression.

Conclusion: EndMT represents an important link between endothelial dysfunction, inflammation, cellular plasticity, metabolism, and plaque remodeling. Understanding this process may help identify new biomarkers and therapeutic targets for limiting atherosclerotic progression and improving plaque stability.

Selected References: Ilyas I, Evans PC, Berk BC, et al. Endothelial-to-mesenchymal transition (EndMT) in atherosclerosis: mechanisms, models and therapies. Cardiovascular Research, 2026.

You N, Liu G, Yu M, et al. Reconceptualizing Endothelial-to-mesenchymal transition in atherosclerosis: Signaling pathways and prospective targeting strategies. Journal of Advanced Research, 2025;77:419–441.

Zheng X, Ye H, Zhang T, et al. Inhibiting Endothelial SMS2 Alleviates Atherosclerosis by Blocking Endothelial-Mesenchymal Transition Through Boosted Fatty Acid Oxidation. Arteriosclerosis, Thrombosis, and Vascular Biology, 2026.

Laminar shear stress inhibits endothelial-mesenchymal transition and mitigates atherosclerosis through SRXN1. Atherosclerosis, 2026.

Keywords: *EndMT; Atherosclerosis; Endothelial dysfunction; Vascular remodeling; Plaque stability; Cellular plasticity*

P47 **POSTER**

Imeglimin Attenuates Established Isoproterenol-Induced Cardiac Hypertrophy in Rats: A Comparison with Ramipril

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Introduction: Pathological cardiac hypertrophy, driven by oxidative stress and inflammation, is a key precursor of heart failure. Imeglimin, a novel oral antidiabetic that improves mitochondrial function, may offer cardioprotection beyond glycaemic control. This is a post-treatment study evaluating treatment of imeglimin against isoproterenol (ISO)-induced cardiac hypertrophy in rats and comparing it with ramipril.

Materials and Methods: Male Wistar rats (n = 6/group) received ISO (3 mg/kg/day, s.c.) for 35 days, followed by oral imeglimin (50, 100 or 200 mg/kg/day) or ramipril (3 mg/kg/day) for 28 days. Normal control and imeglimin per se (200 mg/kg) groups were included. Heart weight/body weight (HW/BW) ratio, serum CK-MB and LDH, myocardial MDA, GSH and SOD, serum TNF-α and IL-6 (ELISA), histopathology and cardiomyocyte cross-sectional area were assessed. Data were analysed by one-way ANOVA with Bonferroni post-hoc test.

Results: ISO significantly increased HW/BW ratio, CK-MB, LDH, MDA, TNF-α, IL-6 and CSA, and depleted GSH and SOD versus control ($p < 0.05–0.001$). Imeglimin attenuated these changes dose-dependently; 200 mg/kg significantly reduced HW/BW ratio, MDA, CK-MB, LDH, TNF-α, IL-6 and CSA and restored GSH and SOD versus ISO ($p < 0.05–0.001$), with near-normal myocardial architecture. Effects of high-dose imeglimin were comparable to ramipril. Imeglimin per se produced no adverse changes.

Conclusions: Imeglimin attenuates established ISO-induced cardiac hypertrophy through antioxidant and anti-inflammatory actions, with efficacy comparable to ramipril at 200 mg/kg, supporting its potential repurposing for pathological cardiac remodelling.

Keywords: *Imeglimin; Cardiac hypertrophy; Isoproterenol; Oxidative stress; Inflammation*

P48 **POSTER**

Myocardial Ischemia in Atherosclerosis: Electrophysiological Mechanisms, ECG Changes and Arrhythmic Risk

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Introduction: Atherosclerosis is a chronic inflammatory disease characterized by plaque formation within the arterial wall. When coronary arteries are affected, narrowing or blockage can reduce blood flow to the myocardium, causing myocardial ischemia. Reduced oxygen supply produces metabolic and ionic disturbances in cardiac cells, altering electrical activity and increasing the risk of arrhythmias.

Materials and Methods: This proposed observational study aims to assess electrophysiological changes associated with myocardial ischemia in patients with atherosclerotic coronary artery disease. Clinical information including age, sex and cardiovascular risk factors will be recorded. ECG findings including ST-segment changes, T-wave abnormalities, QT interval and rhythm disturbances will be assessed. Findings will be compared according to the presence and severity of ischemic changes using descriptive analysis.

Results: It is anticipated that patients with myocardial ischemia will show a higher frequency of ST-segment and T-wave abnormalities. Some patients may also demonstrate QT interval changes, conduction abnormalities or ventricular ectopic beats. Hypertension, diabetes and dyslipidemia are expected to be more common among patients with ischemic ECG changes.

Conclusions: Myocardial ischemia associated with atherosclerosis can produce significant electrophysiological changes and increase arrhythmic risk. ECG may serve as a useful, non-invasive tool for identifying electrical abnormalities associated with ischemia. Understanding these changes may support early risk assessment and cardiovascular management.

Keywords: *Atherosclerosis; Myocardial Ischemia; Electrophysiology; ECG; Arrhythmia*

P49 **POSTER**

Relationship of pregnancy to coronary atherosclerosis

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Pregnancy induces substantial hormonal, metabolic, and cardiovascular changes that may influence long-term cardiovascular health. Earlier necropsy-based studies found no significant association between reproductive rate and coronary atherosclerotic blockage. However, contemporary evidence suggests that adverse pregnancy outcomes, particularly pre-eclampsia, gestational hypertension, gestational diabetes, preterm birth, and pregnancy loss, are associated with increased future coronary atherosclerosis. These associations may be mediated through endothelial dysfunction, chronic inflammation, hypertension, insulin resistance, and metabolic abnormalities.

Objectives: To investigate the association between pregnancy-related factors and coronary atherosclerosis, identify major pregnancy-related risk factors, and evaluate potential mechanisms contributing to later cardiovascular disease.

Study Design: An observational, analytical approach can be used to compare women with and without adverse pregnancy outcomes and assess the presence and severity of coronary atherosclerosis while adjusting for potential confounders such as age, smoking, hypertension, diabetes, obesity, lipid profile, and family history.

Causes & Risk Factors: Important factors include pre-eclampsia, hypertension, gestational diabetes, obesity, dyslipidaemia, insulin resistance, smoking, chronic inflammation, advanced maternal age, and family history of cardiovascular disease.

Conclusion & Importance: Pregnancy itself is not considered a direct cause of coronary atherosclerosis; however, adverse pregnancy outcomes may serve as early markers of increased cardiovascular risk. Identifying these women can support earlier cardiovascular screening, prevention, and long-term risk management.

Keywords: *Pregnancy, coronary atherosclerosis, pre-eclampsia, gestational hypertension, gestational diabetes, endothelial dysfunction, cardiovascular disease.*

P50 POSTER

Cigarette Smoking and New-Onset Chronic Kidney Disease: Evidence from the ISSA-CKD Study

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Chronic kidney disease (CKD) is an important public-health problem, and cigarette smoking is a potentially preventable renal risk factor. This study evaluated whether smoking contributes to the development of CKD among adults without pre-existing CKD. The retrospective ISSA-CKD cohort included 4,540 Japanese adults followed through annual health examinations from 2008–2016. New-onset CKD was defined by eGFR <60 mL/min/1.73 m² and/or newly developed proteinuria. During a mean follow-up of 4.6 years, CKD developed in 759 participants. Current smokers had a higher incidence of proteinuria than non-smokers and demonstrated an adjusted hazard ratio of 1.39. The association was particularly marked in participants younger than 50 years, with an HR of 2.55. A meta-analysis of prospective studies similarly reported increased incident CKD risk among current smokers (RR 1.34).

Objective: To determine whether cigarette smoking increases the risk of new-onset CKD.

Key Lines: Smoking → endothelial injury → renal vascular damage → glomerular injury → proteinuria → reduced eGFR → CKD.

Importance: Smoking is a modifiable risk factor; early smoking cessation may help prevent renal dysfunction.

Pathology/Clinical Correlation: Nicotine and tobacco toxins promote vasoconstriction, oxidative stress, inflammation, endothelial dysfunction and atherosclerosis, reducing renal microvascular health and promoting glomerular injury.

Conclusion: Cigarette smoking is associated with increased risk of new-onset CKD, particularly proteinuria, with a stronger association observed in younger smokers. Smoking cessation should be encouraged for kidney-disease prevention.

Keywords: *Smoking, CKD, Proteinuria, eGFR, Renal function, Epidemiology.*

P51 POSTER

Atherosclerosis in Young Adults: Emerging Cardiovascular Risk Factors and Prevention Strategies.

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Introduction: Atherosclerosis is increasingly recognized as a lifelong process that may begin early in adulthood. In young Indians, obesity, sedentary behaviour, tobacco use, hypertension, dysglycaemia and dyslipidaemia are important modifiable cardiovascular risk factors. Early identification and lifestyle modification may therefore help reduce future atherosclerotic cardiovascular disease.

Materials and Methods: Published Indian studies involving young adults were reviewed to identify major cardiovascular risk factors and prevention strategies. Evidence relating to tobacco use, obesity, blood pressure, glucose abnormalities, lipid profile, physical activity and dietary behaviour was considered, with emphasis on findings relevant to early cardiovascular prevention.

Results: Indian evidence demonstrates a substantial burden of cardiovascular risk factors among younger populations. A national analysis of Indians aged 15–49 years reported an increase in overweight/obesity from 11.4% to 23.6% and raised blood sugar from 6.2% to 8.5% between survey

periods. The real young study of 8,135 Indian adults aged 18–45 years found sedentary lifestyle in more than half of participants, while atherogenic dyslipidaemia was particularly common in younger age groups. These findings indicate the presence of modifiable risk factors early in life.

Conclusions: Atherosclerosis prevention should begin before the development of overt cardiovascular disease. Early screening, healthier dietary practices, regular physical activity, weight management, tobacco avoidance and appropriate control of blood pressure, glucose and lipids may reduce long-term cardiovascular risk among young Indian adults. Community-based awareness, routine risk assessment and targeted preventive interventions in colleges and workplaces may provide opportunities to identify high-risk individuals before clinically evident atherosclerotic disease develops.

Keywords: *Atherosclerosis; Young Adults; Cardiovascular Risk; Dyslipidaemia; Prevention*

P52 **POSTER**

Physiotherapy and Exercise Rehabilitation in Diabetes-Associated Peripheral Artery Disease.

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Introduction: Peripheral artery disease (PAD) is a manifestation of systemic atherosclerosis and an important vascular complication of type 2 diabetes mellitus. Diabetes-related endothelial dysfunction, chronic inflammation, and metabolic abnormalities may accelerate atherosclerotic changes, contributing to lower-limb ischemia, intermittent claudication, and reduced walking capacity. This is particularly relevant in India, where the growing burden of diabetes is associated with an increasing risk of peripheral vascular complications. Physiotherapy-based exercise provides a practical non-pharmacological approach to improving functional mobility, walking capacity, and exercise tolerance in individuals with PAD.

Materials and Methods: Published clinical evidence on physiotherapy-based exercise interventions for adults with type 2 diabetes and peripheral artery disease was evaluated. Relevant interventions included supervised progressive walking, lower-limb strengthening, mobility exercises, and aerobic conditioning. Outcome measures considered included ankle-brachial index (ABI), six-minute walk distance, resting heart rate, blood pressure, oxygen saturation, and perceived exertion assessed using the Borg scale. Evidence regarding changes in walking capacity, exercise tolerance, and functional performance following exercise-based physiotherapy was examined.

Results: Published evidence indicates improved functional performance following exercise-based physiotherapy. An individual-participant meta-analysis involving 719 participants reported improvements of 32.9 m in six-minute walk distance after supervised treadmill exercise and 50.7 m after structured home-based walking, compared with non-exercise controls. Supervised resistance training also improved walking distance by approximately 49.4 m.

Conclusions: Physiotherapy-based exercise may improve walking capacity and exercise tolerance in diabetes-associated PAD. Structured walking, aerobic conditioning, and strengthening may reduce functional limitations and support cardiovascular rehabilitation. Integrating physiotherapy into multidisciplinary care may be particularly relevant for addressing atherosclerotic disease and cardiovascular risk in India overall.

Keywords: *Atherosclerosis; Peripheral Arterial Disease; Type 2 Diabetes Mellitus; Exercise Therapy; Borg Scale*

P53 POSTER

Association of Soluble fms-like Tyrosine Kinase-1 (sFlt-1), Placental Growth Factor (PlGF), 8-Isoprostane and Endothelin-1 Markers with Gestational Obstructive Sleep Apnea and Maternal-Fetal Outcomes

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Background: Gestational obstructive sleep apnea (OSA) is increasingly recognized as an important pregnancy-related sleep disorder associated with adverse maternal and fetal outcomes. Intermittent hypoxia associated with OSA may contribute to oxidative stress, endothelial dysfunction, and placental angiogenic imbalance. However, the combined association of these pathophysiological pathways with gestational OSA and pregnancy outcomes remains inadequately studied, particularly in the Indian population.

Objective: To evaluate the association of soluble fms-like tyrosine kinase-1 (sFlt-1), placental growth factor (PlGF), 8-isoprostane, and endothelin-1 (ET-1) with gestational OSA and maternal-fetal outcomes.

Methods: A prospective case-control study will be conducted among pregnant women aged 19–40 years with singleton pregnancies at 24–32 weeks of gestation. Participants will be screened for OSA using the STOP-BANG questionnaire, Berlin Questionnaire, Epworth Sleepiness Scale, and Mallampati score. Women identified as having gestational OSA based on the screening criteria will be compared with individually matched non-OSA controls. Approximately 5–8 mL of maternal venous blood will be collected, and serum concentrations of sFlt-1, PlGF, 8-isoprostane, and ET-1 will be estimated using ELISA. Maternal outcomes, including gestational hypertension, preeclampsia, and gestational diabetes mellitus, and fetal/neonatal outcomes, including preterm birth, fetal growth restriction, low birth weight, Apgar score, and NICU admission, will be assessed. A total of 140 participants (70 cases and 70 controls) is planned.

Expected Results: It is anticipated that women with gestational OSA will demonstrate altered biomarker levels and that these alterations will be associated with adverse maternal and fetal outcomes.

Conclusion: Integrated assessment of angiogenic, oxidative stress, and endothelial dysfunction biomarkers may improve understanding of the biological mechanisms linking gestational OSA with adverse pregnancy outcomes and may support early risk stratification.

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