NEWSLETTER OF ISAR – DELHI CHAPTER

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Editor - Dr Parul Goyal

Co editors - Dr Rachna Agarwal

Dr S Naveen Kumar

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TABLE OF CONTENTS

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

Atherosclerosis is emerging as the major cause of morbidity and mortality in today's world especially amongst the youth. It may present as silent disease, causing no symptoms, and when it does, it can be life-threatening.

In order to update our knowledge, clinical skills and to promote research activities to enrich our understanding of the causes, prevention and treatment of atherosclerosis, cardiovascular and cerebrovascular diseases in the Indian population, Indian Society for Atherosclerosis Research (ISAR) was founded in 1987.

Delhi Chapter ISAR (ISAR-DC) was formed following a meeting of the National Executive and General body in November 2014 with the same aim to propagate knowledge on various aspects of atherosclerosis and related fields in India. Both these bodies are multi-disciplinary whose members are biochemists, molecular biologists, pathologists, cardiologists, epidemiologists, pharmacologists, physicians and vascular surgeons. Delhi Chapter of ISAR (www.delhichapterisar.co.in) is a state chapter of ISAR (www.isar.co.in) which is a member society of the International Atherosclerosis Society (www.athero.org) based in Italy. Life Membership of DC-ISAR as well as ISAR, automatically entitles one for the membership of International Atherosclerosis Society (IAS) with all its privileges

ISAR will be happy to take all clinicians and researches in its fold to work together in the quest to manage this deadly disease.

MESSAGE FROM THE DESK OF PRESIDENT (ISAR – DC)

Dr RITU SINGH
(President, DC-ISAR& Former National President ISAR)
MD, FIME, FISAR, FIMSA
WHO fellow (Lab Genetics) AIIMS, Delhi
Genomics Fellow DHR (University of Florida, USA)

Greetings from Delhi Chapter ISAR

It is a pleasure and honour to communicate with you as the President of the Delhi Chapter of this prestigious body ISAR dedicated to Atherosclerosis research with multiple specialties and disciplines brainstorming newer as well as traditional ways to tackle the Pandemic of Atherosclerosis,

DC-ISAR is a chapter of ISAR which in turn is a member society of International Atherosclerosis society based at Milan Italy.

Atherosclerosis and its myriad complications and clinical events like myocardial infarction and stroke are a dilemma to the patient and doctor alike. In an effort to understand the etiopathogenis of atherosclerosis, there is a gamut of ongoing research from dyslipidaemias, oxidative stress, inflammation, genetics and epigenetics even as the epidemic of Atherosclerosis continues to surge. Cardiac problems have also been implicated to be associated with post-covid complications.

Atherosclerosis is not a disease – it is caused by a syndrome of diseases which only effective lifestyle and holistic measures can prevent. Scientific and long-term research in these areas is lacking and needs focus.

May DC-ISAR always be a vibrant society as it contributes effectively and significantly to guide research and management of the pandemic that is Atherosclerosis.

Director Professor & HOD, Dept. of Biochemistry
& Coordinator Medical Education Unit
LHMC & Associated SSKH& KSCH hospitals
Shaheed Bhagat Singh Marg
New Delhi 110001
Ph: 9811173213
email: drritusingh19@gmail.com

MESSAGE FROM THE DESK OF SECRETARY (ISAR – DC)

Dr Harsh Vardhan Singh
Senior Biochemist, Department of Biochemistry,
North Delhi Municipal Corporation Medical College &
Hindu Rao Hospital, Delhi-110007
Mobile: +91-9810910824

Dear Esteemed Delegates, Faculty, Clinicians, Scientists and Students,

It is with great pleasure and enthusiasm that I extend a warm welcome to each one of you on behalf of the Indian Society for Atherosclerosis Research - Delhi Chapter. Our upcoming Continuing Medical Education (CME) event is set to take place on November 3, 2023, organised by Department of Biochemistry at Lady Hardinge Medical College, Delhi. As the Secretary of ISAR - Delhi Chapter, I am honoured to be a part of this esteemed organization and to have the privilege of welcoming such a distinguished gathering of the event.

The CME promises to be an enriching experience, featuring insightful sessions led by renowned experts in the field of atherosclerosis research. This event is a testament to our commitment to advancing knowledge, fostering collaboration, and promoting excellence in the study & continual research of atherosclerosis.

The venue, Lady Hardinge Medical College, is a prestigious institution known for its dedication to medical education and research. We are confident that the academic environment, coupled with the wealth of knowledge that will be shared during the CME, will contribute significantly to your professional growth. Your participation and engagement are integral to the success of this event, and we look forward to creating meaningful connections and fostering a spirit of collaboration.

Thank you for your commitment to advancing the field of atherosclerosis research. We eagerly anticipate your presence at the CME on November 3, 2023.

Best regards,

(Dr. Harsh Vardhan Singh)
Secretary, ISAR-DC

MESSAGE FROM THE DESK OF TREASURER (ISAR – DC)

Dear Delegates

The annual CME of Indian Society for Atherosclerosis Research - Delhi Chapter, is being organized by the by Department of Biochemistry at Lady Hardinge Medical College, Delhi.

As a treasurer of ISAR-DC, I feel privileged to welcome you all to this academic feast. The members of ISAR-DC are contributing immensely to the basic, translational and clinical research on various aspects of atherosclerosis. This CME will provide an excellent platform to share latest updates in this field, during interactive sessions with renowned researchers. It will also provide an opportunity to young researchers to showcase their work.

Looking forward to a fruitful interaction with all of you.

Regards

Dr Rajeev Goyal
Associate Professor
Dept. of Biochemistry
LHMC and associated hospitals

Write ups by Members

a. Association of ApoE polymorphism with Stroke in North Indian Population

Dr Rachna Agarwal[1], Ms Anuradha[2], Dr Suman Kushwaha[3], Dr C B Tripathi[4]

[1]Professor & Head, Department of Neurochemistry
[2]Department of Neurochemistry
[3]Professor & Head, Department of Neurology
[4]Associate Professor & Head, Department of Biostatistics
Institute of Human Behaviour & Allied Sciences, Delhi

Stroke is the second most frequent cause of mortality and disability worldwide with two thirds strokes occurring in individuals older than 65 years of age. It is a multifactorial disease with heterogenous etiopathogenesis. Various studies show that the human apolipoprotein E (ApoE) may have an impact on stroke occurrence which is substantiated by the fact that there is strong correlation between APOE genotyping with cholesterol metabolism, atherosclerosis, ischemic heart diseases, cerebral amyloid angiopathy and stroke. ApoE gene is located on chromosome 19 coding for apolipoprotein E. It is present in three isoforms: ApoE2, ApoE3 and ApoE4 with six genotypes E2/2, E2/3, E2/4, E3/3, E3/4 and E4/4. On this back ground the present study was taken up to find out the distribution various ApoE genotype in stroke and association of ApoE4 with stroke.

Methodology: A cross-sectional study was performed on non-diseased and diseased subjects with stroke from outpatient services of Neurology department of Institute of Human Behaviour & Allied Sciences (IHBAS), New Delhi (India). All the subjects with acute onset of persistent neurological deficit were diagnosed as stroke, confirmed by neuroimaging (CT/MRI), whereas in control group subjects included were attending the department of Neurology in same hospital for illness other than stroke and without any memory complaints. The patients of head injury presenting with stroke were excluded. APOE genotyping was done in all subjects by PCR-RFLP method.

Results: In stroke group, there were 112 subjects (Mean age 57.61 ± 15.21 years; 41 females & 71 males), and control group had 113 patients (mean age: 57.45 ± 14.25 years; 49 females & 64 males). Genetic analysis performed to identify the frequency of six possible ApoE genotypes among diseased and non-diseased subjects. The ApoE3/3 genotype was most predominant genotype in both groups, whereas ApoE3/4 had

second most frequency of occurring in stroke group (13.4%) and Control group (10.60%). No subject in stroke group had ApoE4/4. However, 01 subject in control group had ApoE4/4/ (Table 1). ApoE4 allele was present in 17 subjects in stroke as compared to 13 subjects in control group. Association study showed that ApoE4 allele as risk factor (AOR=1.36; 95% CI: 0.63 - 2.96), showing a weak association (Table 2).

Discussion: The genetic contribution in stroke is polygenic. However, very few studies have been taken up to study the role of ApoE variation in development of stroke. Luthra et al 2002 and Ganaie et al 2020 examined the association of ApoE gene polymorphism with stroke in Indian population. The present study, ApoE4 allele showed 1.32-fold odds for developing stroke. Similar findings have been reported by Ganaie et al. who also reported that ApoE4 allele had 2.74-fold odds for developing ischemic stroke in ethnic Bengali population of west Bengal. We have also found that ApoE4 allele has strong association with AD & Other dementias, whereas weak association with Parkinson's disease in our studies. However further studies need to be done to find out the association ApoE4 allele with various neurological diseases.

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Table 1: Comparison of age, gender and APOE Genotyping between Stroke & Non-diseased subjects

Variables	Diagnosis		Cramer's V	p-value
	Stroke (n=112)	Non-diseased (n =113)		
Age (mean, SD)	57.61, 15.21	57.45, 14.25	--	0.94
Gender Male Female	 63.40% 36.60%	 56.60% 43.40%	0.07	0.30
AOPE Genotyping e2e3 e2e4 e3e3 e3e4 e4e4	 15.20% 1.80% 69.60% 13.40% 0.00%	 8.80% 0.00% 79.60% 10.60% 0.90%	0.16	0.20

Table 2: Association of ApoE4 allele with stroke

Variables	AOR (95% CI)	Disease status		AOR (95% CI)
		Diseased (Stroke) (n= 112)	Non-diseased (n= 113)	
APOE4 Genotyping Present Absent	3.07 (1.39 – 6.78)	17 95	13 100	1.36 (0.63 - 2.96)
Gender Male Female	0.82 (0.45 - 1.48)	71 41	64 49	1.32 (0.77 – 2.62)
Age (Mean, SD)	1.07 (1.04 – 1.10)	57.61, 15.21	57.45, 14.25	1.00 (0.98 – 1.02)

b."Unmasking Atherosclerosis: The Silent Saboteur of Heart Health"

Dr AK Singhal

Associate Professor
Department of Biochemistry
Al Falah school of Medical Sciences & Research Centre
Faridabad

Introduction

Your arteries stiffen owing to atherosclerosis, which is caused by a slow accumulation of plaque. High blood pressure, diabetes, high cholesterol, smoking, obesity, lack of exercise, and a diet heavy in saturated fat are all risk factors. The signs of atherosclerosis may not manifest until you experience issues like a heart attack or stroke.

What is Atherosclerosis?

The steady buildup of fatty deposits, cholesterol, calcium, and other chemicals within the arteries is known as atherosclerosis, a complicated medical problem. These deposits, which are collectively referred to as plaques, can impede blood flow and jeopardise the condition of the arteries that are impacted. A blood clot may form in response to a plaque rupture, which may result in potentially fatal illnesses like heart attacks and strokes.

How common is atherosclerosis?

Atherosclerosis occurs frequently. Heart attacks and strokes are just two of the problems of plaque development that are the top cause of death globally. The US National Institutes of Health estimate that half of persons between the ages of 45 and 84 have atherosclerosis but are unaware of it.

What are the complications of atherosclerosis?

Your cardiovascular system's proper operation is hampered by atherosclerosis. It may restrict or obstruct blood flow to your heart and brain, among other bodily components. The following are potential side effects of decreased blood flow:

Carotid artery disease.
Coronary artery disease.
Heart attack.
Mesenteric ischemia.
Peripheral artery disease.
Renal artery stenosis.
Transient ischemic attack (TIA).
Stroke.
Additionally, aneurysms can develop as a result of atherosclerosis weakening the walls of your arteries.

Risk Factors:

The likelihood of developing atherosclerosis is high. Risk factors that cannot be changed are those. In some circumstances, you might be able to lessen modifiable risk factors, such as some medical problems and lifestyle choices. It's crucial to remember that certain risk factors change depending on the sex you were assigned at birth. For instance, those who are born as male (AMAB) have a higher chance of developing atherosclerosis than those who are born as female (AFAB). Understanding the risk factors for atherosclerosis is crucial for prevention and early intervention. Some of the key risk factors include:

Non-modifiable risk factors

Increasing age.

People assigned male at birth face a higher risk after age 45.

People assigned female at birth face a higher risk after age 55.

Family history of premature cardiovascular disease. This means a close biological family member who's AMAB was diagnosed with cardiovascular disease before age 45. Or, one who's AFAB was diagnosed before age 55.

Medical conditions

Diabetes.

High blood pressure (hypertension).

High cholesterol (hyperlipidemia), especially high LDL cholesterol or high levels of a specific lipoprotein called lipoprotein (a).

Metabolic syndrome.

Lifestyle factors

Smoking or tobacco use.

Lack of physical activity.

A diet high in saturated fat, trans fat, sodium and sugar.

How is atherosclerosis diagnosed?

In order to identify atherosclerosis or determine your risk of acquiring it, a healthcare professional will:

- Examine your body thoroughly - You can do this by using a stethoscope to hear your heartbeat and the flow of blood through your arteries. For instance, if you hear a "bruit," your doctor will check your carotid arteries (in your neck). This noise might represent the existence of plaque.
- Inquire about your family history and medical history - These specifics can demonstrate your vulnerability to atherosclerosis and its problems.
- Make a blood test order - The traditional lipid profile, which includes the measurement of total cholesterol, triglycerides, high-density lipoprotein, and low-density lipoprotein. Several other lipid indices interplay between lipid/lipoprotein fractions, such as Lipid tetrad index, lipid pentad index, Atherogenic index, Non-HDL Cholesterol, Castelli Index I and II, Triglyceride-Rich Lipoprotein Cholesterol have emerged as potential substitute predictors of cardiometabolic risk.

“Biochemists can identify persons at risk by analysing these lipid indices and create targeted therapies to lower the risk of first-time or recurring cardiovascular events. Their work advances the creation of diagnostic instruments, therapeutic modalities, and lipid-lowering methods that are intended to slow the course of atherosclerosis.”

Tests to be done to diagnose Atherosclerotic condition:

- Angiography- In this examination, specialised X-rays are used to find and quantify bottlenecks. To make the blockages visible on the X-rays, your doctor will inject a contrast dye into your arteries.
- Ankle-brachial index -In order to evaluate blood flow in your arms and legs, this test compares the blood pressure in your ankle to the pressure in your arm.
- A chest X-ray. An X-ray of the chest produces images of your inside organs.
- MRI - This scan, which produces images of the interior of your body, can detect any hardening and narrowing of your big arteries.
- Echo (echocardiogram). An echo measures how well your heart is pumping while taking images of the chambers and valves of your heart.
- (EKG) Electrocardiogram- An EKG examines the electrical activity, rhythm, and pace of your heart.

Management for atherosclerosis:

Atherosclerosis management includes one or more of the following:

Lifestyle changes	Medications	Procedures or surgeries
• Avoid all tobacco products (including smoking and vaping). • Follow a heart-healthy eating plan like the Mediterranean Diet. • Build exercise into your daily routine.	• Lower your blood pressure. • Lower your cholesterol. • Manage your blood sugar levels. • Prevent blood clots.	• Angioplasty. • Atherectomy. • Carotid endarterectomy. • Coronary artery bypass grafting (CABG). • Peripheral artery bypass. • Stent placement. • Vascular disease bypass.

Prevention Strategies

Fortunately, atherosclerosis is preventable and manageable with the right lifestyle choices and medical interventions. Here are some strategies to help reduce your risk:

1. Eat a Heart-Healthy Diet: Consume a diet rich in fruits, vegetables, whole grains, lean proteins, and healthy fats (e.g., olive oil, nuts, and avocados).

2. Exercise Regularly: Aim for at least 150 minutes of moderate-intensity aerobic exercise or 75 minutes of vigorous-intensity exercise each week.

3. Quit Smoking: If you smoke, seek support to quit. Smoking cessation has immediate and long-term cardiovascular benefits.

4. Manage Blood Pressure: Regularly monitor your blood pressure and follow your healthcare provider's recommendations for management.

5. Control Cholesterol: Keep your cholesterol levels within a healthy range through diet, exercise, and, if necessary, medication.

6. Manage Diabetes: If you have diabetes, work closely with your healthcare team to control your blood sugar levels.

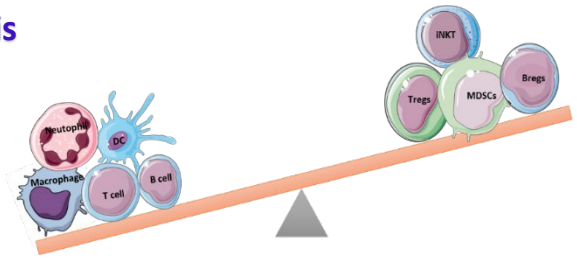
7. Reduce Stress: Find healthy ways to manage stress, such as meditation, yoga, or deep breathing exercises.

The changing views on Atherosclerosis:

Past	Present
Atherosclerosis predominantly affects developed countries	Developing countries now bear the greatest burden of atherosclerosis
Coronary thrombosis affects primarily middle-aged white men	Women, younger individuals, individuals from a range of ethnic backgrounds and the very old suffer increasingly from acute coronary syndromes
Atherosclerosis is a lipid storage disease	Inflammation links dyslipidaemia and other risk factors to atherogenesis
Oxidized LDL drives atherosclerosis	Native or aggregated LDL drives atherogenesis
HDL cholesterol protects against atherosclerosis	TGRL participate causally in atherosclerosis
Thin-capped fibroatheromata are vulnerable plaques	The 'vulnerable plaque' is a misnomer; superficial erosion is an increasing cause of arterial thrombosis
Atherosclerosis is an inevitable, steady and degenerative accompaniment to ageing	Atherosclerosis evolves episodically, can regress, and lifestyle and medical measures can modulate the process

c. Immune Regulatory Cells in Atherosclerosis

Komal Sagar, Shamima Akhtar, Alpana Sharma
Department of Biochemistry, AIIMS, New Delhi



Introduction:

Atherosclerosis is a chronic inflammatory disease of medium to large-sized arteries. Immune cells of both innate and adaptive arms have been shown to play a role in atherosclerosis. The disbalance of immune-inflammatory cells (macrophage, neutrophils, T-cells, and B-cells) and immune regulatory cells (Tregs, Bregs, MDSCs, and iNKT) is one of the important attributes of the disease. Their deregulated function in a chronic inflammatory environment aid in atherogenesis rather than checking its progression. Here we give a broad overview of different immune regulatory cells and their role identified in atherosclerosis to date**.**

Immune Regulatory Cells in Atherosclerosis:

1.) M2 Macrophages: Atherosclerotic plaques contain different macrophage subtypes, including M1 and M2 subtypes. M1 macrophages are activated by Th1 cytokines and promote inflammation, while M2 macrophages, activated by Th2 cytokines, are anti-inflammatory[1]. M2 macrophages contribute to tissue repair and efferocytosis (clearance of apoptotic cells) within the plaques. They secrete anti-inflammatory cytokines like IL-10 and play a role in plaque stabilization. The JAK-STAT pathway regulates M2 macrophage activation and proliferation[2].

2. Regulatory T Cells (Tregs) and Th2 Cells: Tregs are a T cell subset with immunosuppressive functions. They secrete transforming growth factor β (TGF-β), which reduces inflammation, promotes smooth muscle cell proliferation, and enhances collagen synthesis[3]. Tregs are categorized as naturally occurring (nTregs) and induced (iTregs) based on their origin and development. Depletion of Tregs exacerbates atherosclerosis, emphasizing their protective role. They suppress immune responses through various mechanisms, including inhibitory cytokine secretion, perforin and granzyme production, and suppression of dendritic cell function. Tregs are a T mobile subset with immunosuppressive features[4].

3. Regulatory B Cells (Bregs): Bregs are a subset of B cells with regulatory functions. They suppress inflammation and immune responses through IL-10 secretion and interactions with other immune cells. Studies have shown that deficiencies in Bregs can lead to increased atherogenesis. Modulating Bregs can impact atherosclerotic plaque size[5].

4. Myeloid Derived Suppressor Cells (MDSCs): MDSCs are immature myeloid cells that can suppress immune responses. In healthy individuals, they comprise a small fraction of immune cells but can increase during chronic inflammation and various diseases. MDSCs can inhibit T cell movement to lymph nodes and reduce atherosclerotic burden. However, the exact mechanisms of their action are not fully understood[6].

5. Tolerogenic Dendritic Cells (ToDCs): Tolerogenic DCs (ToDCs) is a subset of dendric cell with suppressive function. ToDC helps in maintaining the equilibrium between immunity and tolerance. It causes T-cell clonal deletion, T cell anergy and induces the development of Tregs. ToDCs are immature or semi-mature DCs with the low expression of co-stimulatory molecule. ToDCs have increased expression of PDL1, CTLA-4 leading to decreased T cell response. It also causes shedding of CD25 leading to decreased T-cell proliferation due to deprivation of IL-2. ToDCs are capable of producing IL-10 and TGF-β leading to enhanced Tregs proliferation [7].

6. Invariant Natural Killer T (iNKT) Cells: iNKT cells are innate-like lymphocytes that recognize lipid antigens. They express unique TCRα chains and variable TCRβ chains, allowing them to respond to lipid antigens presented by CD1d molecules. iNKT cells can modulate immune responses by producing cytokines typically associated with both Th1 and Th2 cells. In obesity, a loss of IL4-secreting resident iNKT cells has been linked to inflammation and type-2 diabetes. However, during the early stages of atherosclerosis, there is an increase in anti-inflammatory iNKT cells, which decreases as the disease progresses[8].

Therapeutic Targeting of Immune Regulatory Cells for Atherosclerosis Treatment:

Recent clinical trials explore potential therapies targeting immune regulatory cells. In the LILACS trial, low-dose interleukin-2 (IL-2) treatment with aldesleukin has shown promise in promoting the proliferation and functionality of Tregs, leading to reduced adverse effects in patients with ischemic heart disease. Additionally, IL-6 blockade with tocilizumab has demonstrated efficacy in reducing adverse effects in patients with ST-elevation myocardial infarction (STEMI) and non-ST-elevation myocardial infarction (NSTEMI). An anti-IL6 antibody, ziltivekimab, has also shown promise in reducing overall inflammation.

In conclusion, understanding the roles of immune regulatory cells in atherosclerosis provides insights into potential therapeutic strategies for mitigating this chronic inflammatory disease. Targeting these cells holds promise for improving the management and treatment of atherosclerosis and its associated cardiovascular complications.

Perspective

Atherosclerosis is a chronic inflammatory disease of arteries wherein regulatory arm of immune system becomes dysfunctional. Till date most of the studies and clinical trials have focused on the reducing the inflammatory arm of the immune system. In recent years, research have focused on normalizing the regulatory arm of the immune system which also acquires the inflammatory phenotype. A handful of clinical trials have shown promising results. Therefore, quest for new therapeutic targets still continues. Hence, further research focusing on immunophenotyping the regulatory cells in the atherosclerotic patient is warranted to develop novel targeted immunotherapy.

S.No	Name/ Trial	Target	Effect	Reference
1.	LILACS	IL-2	Tregs cells proliferation, expansion, and functionality	[9]
2.	Tocilizumab	IL-6	Reduces hsCRP in MI patients (STEMI).	[10]
3.	Ziltivekimab	IL-6	Reduced biomarkers of inflammation and thrombosis	

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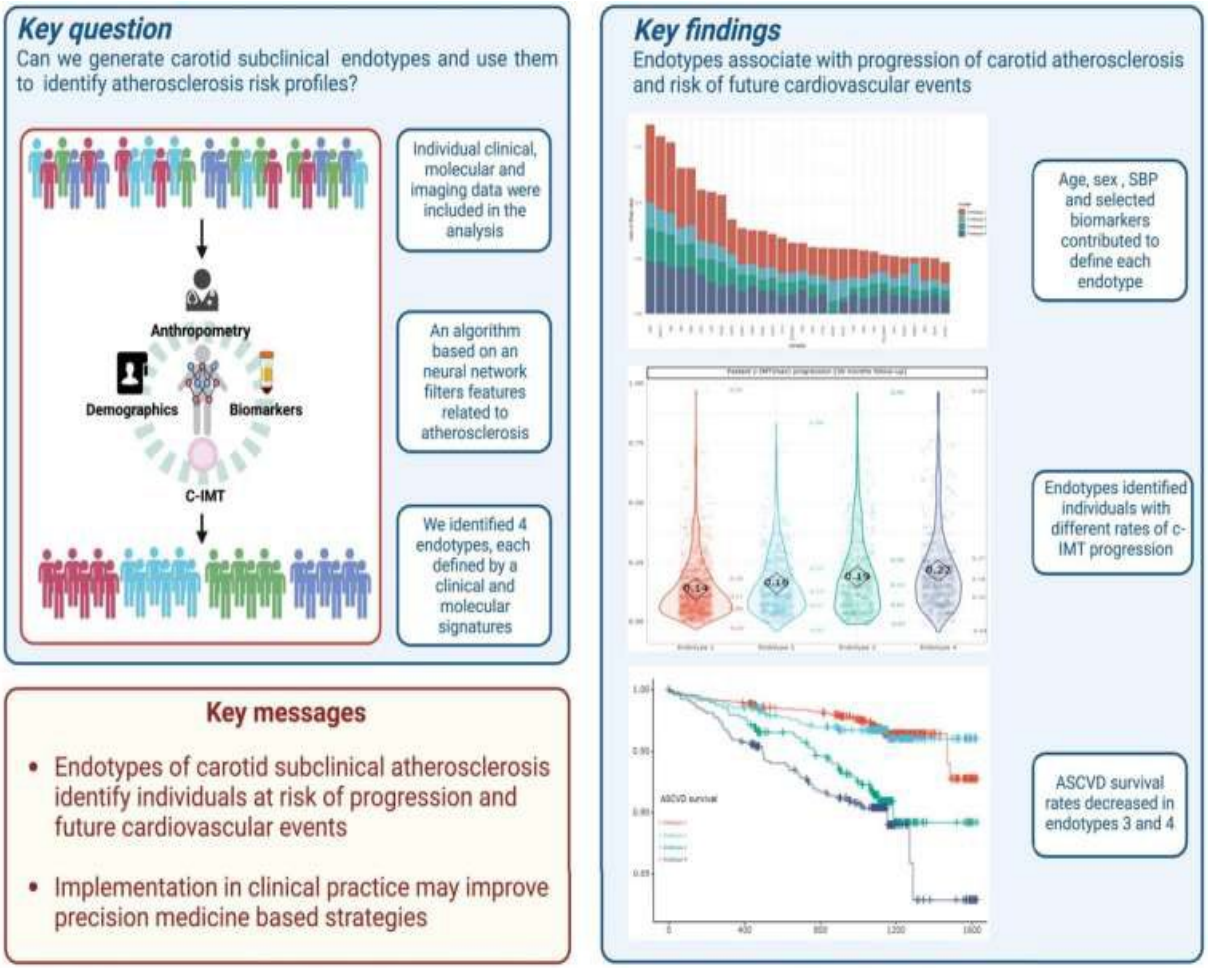
ATHEROSCLEROSIS NEWS & EVENTS

Medical Xpress

JULY 21, 2023

a. AI and precision medicine may uncover risks of cardiovascular disease

by Karolinska Institutet



Credit: *Cardiovascular Research* (2023). DOI: 10.1093/cvr/cvad106

Cardiovascular disease is one of the most common causes of death in Sweden and in the world. Researchers at Karolinska Institute, among others, have found that artificial intelligence can play a role in identifying the risk of cardiovascular disease. The study, published in Cardiovascular Research, may lead to more accurate diagnostic methods.

Cardiovascular diseases can have a variety of causes but atherosclerosis is one of the more common. Atherosclerosis is the buildup of fats, cholesterol and other substances in and on the artery walls. This plaque can cause arteries to narrow, blocking blood flow. The outcome is often acute with a heart attack or stroke.

Preventing cardiovascular disease

The recently published research study, conducted at Karolinska Institute in collaboration with Uppsala University and several European universities, has investigated how artificial intelligence (AI) can help identify the individual risk of atherosclerosis and thus provide the opportunity to intervene before cardiovascular disease develops.

"Atherosclerosis is a silent killer, and our results pave the way for precision medicine in the prevention of cardiovascular diseases related to atherosclerosis," says Bruna Gigante, last author and senior lecturer at the Department of Medicine, Karolinska Institutet, Solna.

The research team has used clinical and molecular data together with ultrasound measurements of the carotid artery from participants in a large European study.

Three identified risk groups

An unsupervised method for machine learning has integrated all the data and based on the material, the researchers have defined four endotypes to find individuals with a low, medium or high risk of heart attack and stroke. The results have been validated in a Swedish cardiovascular study, using common epidemiological and statistical methods.

"We tested whether the endotypes we had generated with machine learning could predict the risk of developing atherosclerosis in the carotid artery," says first author Qiaosen Chen, doctoral student at the Department of Medicine, Karolinska Institutet, Solna. "The results show that they can, to some extent."

The researchers now plan to investigate the genes and mechanisms behind the different endotypes of atherosclerosis and related diseases of the heart and brain. They are also interested in investigating how the results from the current study can be translated into predicting the development of atherosclerosis in other vascular beds.

b. Could plant-based diets help treat cardiovascular diseases?

By Neha Mathur Aug 22 2023 *Reviewed by Sophia Coveney*

In a recent article published in *Nutrients*, researchers identified a knowledge gap about the molecular mechanisms by which plant-based diets target cardiovascular diseases (CVDs).

Background

The American Heart Association (AHA) conducted multiple epidemiological studies to understand the influence of diet on CVDs. Based on the study results, AHA concluded that diets rich in animal-based and minimally processed plant foods were associated with poor and optimal cardiovascular health, respectively.

These observations explain why the incidence of CVDs is much higher in Western countries, including the USA, where the intake of animal-based foods is 140% more than the dietary recommendations, while the intake of raw plant foods is relatively low.

In one of their previous works, the researchers outlined how intake of an animal-based diet triggered several molecular mechanisms driving CVD pathogenesis. However, there is a scarcity of studies specifically exploring the benefits of plant-based diets and not the reduced consumption of animal products.

In this article, the researchers outlined some of the benefits of specific bioactive components of plant foods beneficial for cardiovascular health, even if one consumes animal products to some extent.

Plant food consumption and cardiovascular health

All dietary fiber comes from plant sources; thus, even fiber from unprocessed foods is a proxy for plant food consumption. A rigorous systematic analysis of ~135 million

person-years of data from a study by Reynolds et al. suggested an inverse, dose-dependent association of fiber intake with CVD incidence and subsequent mortality.

Further, the National Health and Nutrition Examination Survey (NHANES) data revealed that ~6% of people in the USA barely meet the minimum fiber intake recommendations.

Researchers also gathered stirring *in vivo* data on how dietary fiber positively alters gut microbiota to prevent metabolic disorders. Specifically, they showed how maternal low-fiber intake adversely affected the gut microbial health of neonates, increasing the likelihood of obesity due to obesogenic diet intake.

Given the role of gut microbiota in CVD pathogenesis, it is highly clinically relevant to increase plant food consumption for improved gut health. Apart from fiber, plants are a great source of several polyphenols and secondary metabolites. Each plant has a distinctive polyphenol profile; thus, a heterogeneous plant-based diet facilitates potential synergistic effects. In mice, the consumption of blackberries and raspberries improved cardiac inflammatory signaling more than the consumption of one of the berries alone.

Researchers have outlined numerous molecular pathways through which polyphenols mediate cardiovascular function. Examples include apoptotic pathways, redox, and inflammatory pathways, and the renin-angiotensin system. However, these studies assessed single polyphenols using *in vitro* and *in vivo* animal models, while each plant food has a multitude of polyphenols.

A previous study demonstrated that higher polyphenol consumption reduced cardiovascular risk by 47% in middle-aged individuals. Likewise, the PREDIMED trial showed that the highest polyphenol intake reduced all-cause mortality risk by 37%.

Together, these observations suggested that of all plant products, polyphenols were most protective against CVDs; however, there is a need for more work in this research area.

Furthermore, the authors outlined that in the literature, only a strict plant-based diet has shown promise to clinically reverse atherosclerosis, improve myocardial perfusion, and even treat heart failure. They also identified studies showing that a diet rich in fruits and vegetables (raw) reduced low-density lipoproteins (bad cholesterol) to an extent comparable to statin treatment.

Journal reference:

Najjar RS, Gewirtz AT (2023). Plant-Based Diets: A Path to Ending CVD as We Know it? *Nutrients*, *15*, 3608.
doi: 10.3390/nu15163608.
https://www.mdpi.com/2072-6643/15/16/3608

MEDICAL NEWS TODAY

c. Heart disease: Experimental cancer drug may slow inflammation from atherosclerosis

Experts note that atherosclerosis is linked to heart disease risk.
The medication saracatinib has been researched in the past as a potential cancer treatment.

- Researchers say the drug is now showing promise as a treatment for atherosclerosis, an inflammation linked to heart disease.
- Experts say the findings are intriguing, but more research needs to be done.

An experimental drug, saracatinib , might slow the progression of atherosclerosis, according to a study published today in the journal Nature Cardiovascular Research.

The medication was initially tested for cancer treatment. However, researchers in a 2015 study determined the drug's efficacy was not high enough. Since then, saracatinib has been or is being tested for Alzheimer's disease Trusted Source and pulmonary fibrosis. The current study at NYU Grossman School of Medicine in New York shows that the drug might slow the progression of atherosclerosis, an inflammation linked to heart disease.

Why saracatinib was tested

The researchers analyzed blood samples from 34 men and women with atherosclerotic cardiovascular disease. All participants were taking statins at the time. There were 24 other participants without atherosclerotic cardiovascular disease in the study for comparison.

The scientists determined that the plasma – the liquid part of the blood – from the people with atherosclerosis triggers an unusually high inflammatory signal in blood immune cells.

Rather than try to create a new drug, the researchers looked at a series of datasets with hundreds of thousands of test results and decided to work with saracatinib because of its anti-inflammatory properties.

"While many people are aware of the risks of high cholesterol and heart disease, the role of inflammation in plaque progression and heart attacks and strokes is being increasingly appreciated," Dr. Jeffrey Tyler, an interventional cardiologist with Providence St. Joseph Hospital in California who was not involved in the study, told Medical News Today.

One expert noted that based on how saracatinib works, there is potential for studying its use against leukaemia and atherosclerotic disease.

"It's important to understand that these studies are in very early stages," said Dr. Sameer Amin, the chief medical officer and a cardiologist for LA Care Health Plan who was not involved in the study.

"Though a drug may theoretically work to treat a condition, treatments often affect the body in unexpected ways," Amin told Medical News Today. "This leads to the possibility of drug repurposing but also may mean that medication in development may never work. We need to be rigorous in differentiating theoretical potential from proven clinical benefit."

What researchers uncovered in atherosclerosis drug study

The researchers reported that saracatinib reduced inflammatory signalling by about 90% in diseased human cells.

When tested in a rabbit model, plaque-based inflammation was reduced by 97%.

In a mouse model, an 80% decrease in cells associated with inflammation in atherosclerosis was seen.

Additionally, plaques shrunk by between 48% and 70% in the mouse model, depending on the dose of saracatinib used.

"Our study employed an unbiased human systems immunology approach to investigate potential drugs that target the concerted action of multiple molecules involved in inflammation," said Dr. Chiara Giannarelli, an associate professor of cardiology and pathology at NYU Cardiovascular Research Centre at the NYU Grossman School of Medicine who has a patent pending for this therapeutic approach she developed for treating atherosclerosis cardiovascular disease.

"By directly examining inflammation responses in human samples, we discovered that saracatinib can diminish these inflammatory responses," Giannarelli told Medical News Today.

The researchers noted that the drug positively mitigated inflammation within atherosclerotic lesions.

What's next for potential treatments

Experts say it's too soon to tell if saracatinib can effectively treat atherosclerosis.

"Before a medication establishes itself as a viable form of treatment, it needs to go through multiple levels of testing in large populations to show that it is improving patient outcomes," Amin said. "There are currently many cardiovascular medications that are in early stages that may never pan out."

The next step is to test the drug on larger animals, according to Dr. Sanjiv Patel, an interventional cardiologist at Memorial Care Heart & Vascular Institute at Orange Coast Medical Centre in California who was not involved in the study.

"In addition, testing needs to be done for the safety of humans. Although other studies tested safety, the researchers need to look at safety based on the dosage they are testing for reducing human inflammation," Patel told Medical News Today.

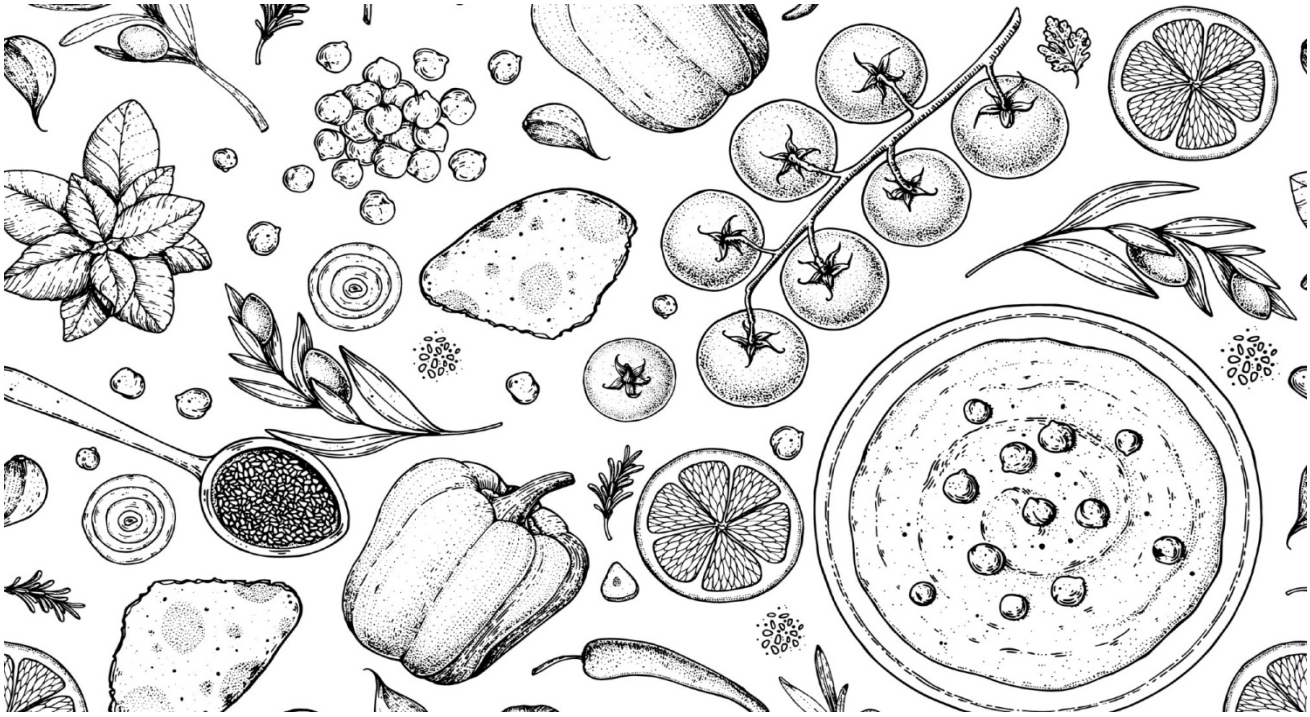
The approach used by the researchers at NYU Grossman School of Medicine might work to introduce new treatments for atherosclerosis.

"By combining cutting-edge technologies and computational tools, we can identify promising drug candidates more efficiently," Giannarelli said. "This streamlined process holds promise for expediting the development of effective treatments and bringing them to market sooner, benefiting individuals with atherosclerosis.

d. Mediterranean diet with a dairy twist shows promise in lowering heart disease risk

By Dr. Priyom Bose, Ph.D. Aug 31 2023

A recent Nutrients journal study evaluated how the combination of Mediterranean diet (MedDiet) supplement and dairy foods affected the gut microbiome in Australians at a high risk of cardiovascular disease (CVD).



Study: Interactions between Mediterranean Diet Supplemented with Dairy Foods and the Gut Microbiota Influence Cardiovascular Health in an Australian Population.

Background

Diet plays an essential role in regulating immunity and maintaining metabolic health. Improper diet is often linked to the development of obesity, cardiovascular diseases, and type 2 diabetes. Long-term adherence to a specific dietary pattern aids in shaping the intestinal commensal microbiota. The gut microbes produce several bioactive compounds by metabolizing dietary components, influencing host metabolic and immune homeostasis.

Gut microbes produce short-chain fatty acids (SCFAs), such as acetate, butyrate, and propionate, through the fermentation of non-digestible fiber. These SCFAs are the primary sources of energy for colonic tissues to modulate inflammatory pathways, maintain gastrointestinal tissue integrity, and inhibit the proliferation of pathogenic bacteria. Alteration of specific gut bacteria lowers triglyceride levels, reduces systemic inflammatory markers (e.g., C-reactive protein), and improves liver function.

All gut microbial products are not beneficial to humans. For instance, microbial metabolism of L-carnitine and choline results in the production of trimethylamine N-oxide (TMAO), which is a metabolite associated with the manifestation of atherosclerosis and inflammation. Both L-carnitine and choline are commonly found in nuts, dairy, meat, fish, and eggs.

MedDiet contains fruits, vegetables, nuts, extra virgin olive oil (EVOO), legumes, and cereals. This diet type recommends the consumption of moderate amounts of fish, dairy foods, eggs, poultry, and red wine. Furthermore, MedDiet is associated with low consumption of red meat, processed food, and discretionary foods (e.g., cakes and sweets).

An abundance of bioactive nutrients, such as fiber, polyphenols, vitamins and minerals, antioxidants, and monounsaturated fats, has been associated with MedDiet, which promotes beneficial effects through the gut microbiota. Long-term adherence to the MedDiet significantly improves obesity, metabolic syndrome, diabetes, and dyslipidemia.

It must be noted that a typical MedDiet provides calcium much below the Australian recommended daily intake (RDI). Calcium plays an important role in the formation and maintenance of bone, vascular dilation and contraction, cell differentiation, neuronal activity, and cell signaling for muscle function. Insufficient calcium intake leads to reduced bone strength and enhances pregnancy complications. It also enhances the risk of CVDs. The MedDiet supplemented with dairy products, such as milk, yogurt, and cheese, would fulfill Australian calcium intake recommendations. It is essential that a MedDiet fulfills all nutritional requirements before recommendation.

About the Study

The current randomized controlled trial (RCT) followed a 2 × 2 cross-over design to compare the benefits of MedDiet supplemented with dairy food (MedDairy) and low-fat (LFD) diet (control) in Australians at high risk of CVD.

This study recruited adults between the ages of 45 and 75 years. All participants had high systolic blood pressure (SBP) but were not under any medication. Individuals who consumed medicinal levels of calcium or omega-3 supplements daily were excluded.

Participants were randomly assigned to any one of the groups, i.e., MedDairy (Group 1) or LFD (Group 2), and dietary interventions continued for 8 weeks, separated by an 8-week washout phase where participants followed their habitual diet. Complete fecal and clinical samples were collected at baseline and at 8 weeks to assess both groups.

Study Findings

At baseline, there were no significant differences between the study groups. Group 1 contained 18 participants, and group 2 contained 16 participants. All participants who were not following MedDiet at baseline exhibited increased MedDiet adherence through the MedDairy intervention. Along with the MedDiet, participants received 3 to 4 servings of any one of the dairy products, such as low-fat Greek yogurt, low-fat milk, cheese (hard, soft, semi-soft), and tzatziki dip.

Fecal microbiota analysis indicated no significant difference in the overall structure and composition of the fecal microbiota between the two study groups. However, a modest decrease in microbial diversity was observed in the LFD group. It must be noted that the MedDairy diet did not result in a significant change in the gut microbiota but considerably altered the abundance of selected bacterial taxa, such as *Butyricicoccus, Lachnospiraceae*, and Streptococcus, and a reduction in *Colinsella* and *Veillonella.*

Conclusions

The findings of the current study highlighted that 8 weeks of a Mediterranean diet supplemented with dairy foods resulted in changes in the relative abundance of certain bacterial taxa. MedDairy diet enhanced *Butyricicoccus ,* which has a positive effect on systolic blood pressure. Therefore, adherence to the MedDairy diet could reduce CVD risks.

THE HealthSite.com

e. Half Of World's Adults Over 40 Might Have Hidden Heart Disease: Study

As per experts, the surprising element was that 10 per cent had obstructive illness without any symptoms.

Written by Kashish Sharma | Published: March 29, 2023 7:10 PM IST

A new study has found people without known cardiovascular disease that have obstructive coronary atherosclerosis can increase their risk of heart attack by eight folds. The study suggested that half of the world's adults over 40 years of age might have hidden or undetected conditions. Heart disease is one of the major causes of death worldwide.

Coronary artery disease is a common heart disease in which the coronary artery that supplies blood to the heart gets blocked with plaque. This causes it to become narrower and inflexible. This can further result in reduced blood supply to the heart and can give rise to conditions like a heart attack. This narrowing down of the artery is termed atherosclerosis. Sometimes the bursting of this plaque can give rise to blood clots that can travel to various parts of the body such as the brain and can cause a stroke.

Why it goes undetected?

The study published in the Annals of Internal Medicine suggests that nearly half of the world's adults over the age of 40 might have undetected heart conditions. As per the study researchers, atherosclerosis is a leading reason for heart attack and cardiac death. The condition can develop many years before it gets detected. The study involved over 9,500 subjects over 40 years of age. They had no previous diagnosis of cardiovascular disease. Computed tomography angiography (CTA) was used to identify any undetected obstructive coronary illness. The results showed that 46 per cent of the participants had previously undetected heart disease. Among them, 36 per cent had a non-obstructive illness and 10 per cent had an obstructive disease. As per experts, the surprising element was that 10 per cent had obstructive illness without any symptoms.

ATHEROSCLEROSIS - RECENT ADVANCES & UPDATES

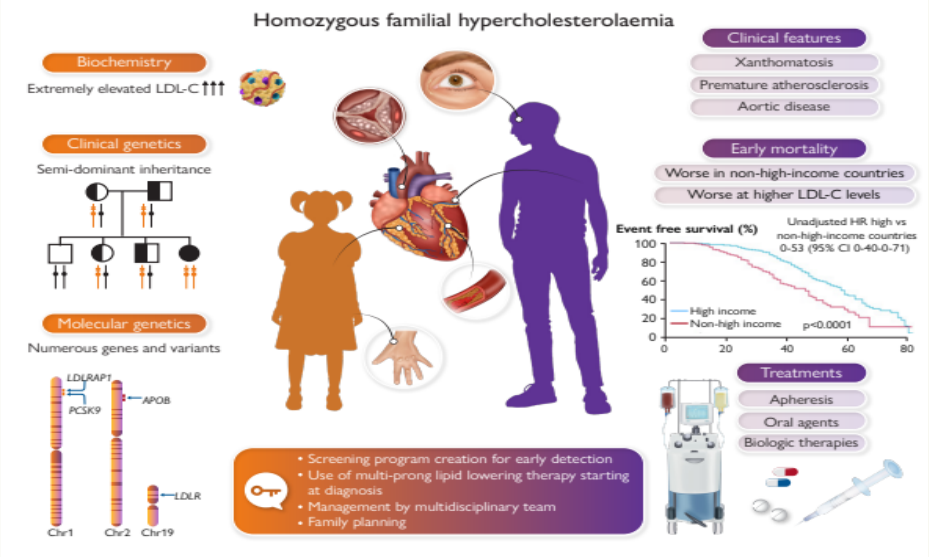
ESC European Society of Cardiology
European Heart Journal (2023) **44**, 2277–2291
https://doi.org/10.1093/eurheartj/ehad197

SPECIAL ARTICLE
Dyslipidaemias

2023 Update on European Atherosclerosis Society Consensus Statement on Homozygous Familial Hypercholesterolaemia: new treatments and clinical guidance

Marina Cuchel [1†], Frederick J. Raal [2†], Robert A. Hegele [3†], Khalid Al-Rasadi [4], Marcello Arca [5], Maurizio Averna [6,7], Eric Bruckert[8],

Graphical Abstract



Homozygous familial hypercholesterolaemia is a rare autosomal semi-dominant disease affecting males and females equally, characterized by markedly elevated levels of low-density lipoprotein cholesterol (LDL-C) from conception and accelerated atherosclerotic cardiovascular disease, often resulting in early death. Homozygous familial hypercholesterolaemia is diagnosed late and undertreated; both established and novel therapies offer hope to patients, but treatment inequity exacerbates poorer outcomes in less affluent countries.[12] *APOB*, apolipoprotein B gene; CI, confidence interval; HR, hazard ratio; *LDLR*, low-density lipoprotein receptor gene; *LDLRAP1*, low-density lipoprotein adaptor protein 1 gene; *PCSK9*, proprotein convertase subtilisin/kexin type 9 gene.

Abstract

This 2023 statement updates clinical guidance for homozygous familial hypercholesterolaemia (HoFH), explains the genetic complexity, and provides pragmatic recommendations to address inequities in HoFH care worldwide. Key strengths include updated criteria for the clinical diagnosis of HoFH and the recommendation to prioritize phenotypic features over genotype. Thus, a low-density lipoprotein cholesterol (LDL-C) >10 mmol/L (>400 mg/dL) is suggestive of HoFH and warrants further evaluation. The statement also provides state-of-the art discussion and guidance to clinicians for interpreting the results of genetic testing and for family planning and pregnancy. Therapeutic decisions are based on the LDL-C level. Combination LDL-C-lowering therapy—both pharmacologic intervention and lipoprotein apheresis (LA)—is foundational. Addition of novel, efficacious therapies (i.e. inhibitors of proprotein convertase subtilisin/kexin type 9, followed by evinacumab and/or lomitapide) offers potential to attain LDL-C goal or reduce the need for LA. To improve HoFH care around the world, the statement recommends the creation of national screening programmes, education to improve awareness, and management guidelines that account for the local realities of care, including access to specialist centres, treatments, and cost. This updated statement provides guidance that is crucial to early diagnosis, better care, and improved cardiovascular health for patients with HoFH worldwide.

Keywords Homozygous familial hypercholesterolaemia • Clinical guidance • Diagnosis • Genetics • Treatment • Women

AHA STATISTICAL UPDATE

Heart Disease and Stroke Statistics—2023 Update: A Report From the American Heart Association

Connie W. Tsao, MD, MPH, FAHA, Chair, Aaron W. Aday, MD, MSc, FAHA, Zaid I. Almarzooq, MBBCh, MPH, Cheryl A.M. Anderson, PhD, MPH, FAHA, Pankaj Arora, MD, FAHA, Christy L. Avery, PhD, MPH, FAHA, Carissa M. Baker-Smith, MD, MPH, FAHA, Andrea Z. Beaton, MD, MS, FAHA,

Background: The American Heart Association, in conjunction with the National Institutes of Health, annually reports the most up-to-date statistics related to heart disease, stroke, and cardiovascular risk factors, including core health behaviors (smoking, physical activity, diet, and weight) and health factors (cholesterol, blood pressure, and glucose control) that contribute to cardiovascular health. The Statistical Update presents the latest data on a range of major clinical heart and circulatory disease conditions (including stroke, congenital heart disease, rhythm disorders, subclinical atherosclerosis, coronary heart disease, heart failure, valvular disease, venous disease, and peripheral artery disease) and the associated outcomes (including quality of care, procedures, and economic costs).

Methods: The American Heart Association, through its Epidemiology and Prevention Statistics Committee, continuously monitors and evaluates sources of data on heart disease and stroke in the United States to provide the most current information available in the annual Statistical Update with review of published literature through the year before writing. The 2023 Statistical Update is the product of a full year's worth of effort in 2022 by dedicated volunteer clinicians and scientists, committed government professionals, and American Heart Association staff members. The American Heart Association strives to further understand and help heal health problems inflicted by structural racism, a public health crisis that can significantly damage physical and mental health and perpetuate disparities in access to health care, education, income, housing, and several other factors vital to healthy lives. This year's edition includes additional COVID-19 (coronavirus disease 2019) publications, as well as data on the monitoring and benefits of cardiovascular health in the population, with an enhanced focus on health equity across several key domains.

Results: Each of the chapters in the Statistical Update focuses on a different topic related to heart disease and stroke statistics.

Conclusions: The Statistical Update represents a critical resource for the lay public, policymakers, media professionals, clinicians, health care administrators, researchers, health advocates, and others seeking the best available data on these factors and conditions.

Key Words: AHA Scientific Statements ■ cardiovascular diseases ■ epidemiology ■ risk factors ■ statistics ■ stroke

The official journal of the Japan Atherosclerosis Society and the Asian Pacific Society of Atherosclerosis and Vascular Diseases

Original Article

J Atheroscler Thromb, 2023; 30: 000-000. http://doi.org/10.5551/jat.64304

Association between Non-Lipid Residual Risk Factors and Cardiovascular Events in Patients with Stable Coronary Artery Disease Treated with Pitavastatin: An Observation from the REAL-CAD Study

Kiwamu Kamiya[1], Makoto Takei[2], Toshiyuki Nagai[1], Toru Miyoshi[3], Hiroshi Ito[4], Yoshihiro Fukumoto[5], Hitoshi Obara[6], Tatsuyuki Kakuma[6], Ichiro Sakuma[7], Hiroyuki Daida[8], Satoshi Iimuro[9], Hiroaki Shimokawa[10, 11], Takeshi Kimura[12], Ryozo Nagai[13] and Toshihisa Anzai[1] for the REAL-CAD study investigators

Kiwamu Kamiya and Makoto Takei contributed equally to this work.

[1]Department of Cardiovascular Medicine, Faculty of Medicine and Graduate School of Medicine, Hokkaido University, Sapporo, Japan
[2]Department of Cardiology, Saiseikai Central Hospital, Tokyo, Japan
[3]Department of Cardiovascular Medicine, Okayama University Graduate School of Medicine, Dentistry and Pharmaceutical Sciences, Okayama, Japan
[4]Department of General Internal Medicine 3, Kawasaki Medical School, Okayama, Japan
[5]Division of Cardiovascular Medicine, Department of Internal Medicine, Kurume University School of Medicine, Fukuoka, Japan
[6]Biostatistics Center, Kurume University, Fukuoka, Japan
[7]Caress Sapporo Hokko Memorial Clinic, Sapporo, Japan
[8]Department of Cardiovascular Medicine, Juntendo University Graduate School of Medicine, Tokyo, Japan
[9]Innovation and Research Support Center, International University of Health and Welfare, Tokyo, Japan
[10]Graduate School, International University of Health and Welfare, Chiba, Japan.
[11]Tohoku University Graduate School of Medicine, Sendai, Japan.
[12]Hirakata Kohsai Hospital, Department of Cardiology, Osaka, Japan
[13]Jichi Medical University, Tochigi, Japan

Aims: We aimed to investigate the association between non-lipid residual risk factors and cardiovascular events in patients with stable coronary artery disease (CAD) who achieved low-density lipoprotein cholesterol (LDL-C) <100 mg/dL from the Randomized Evaluation of Aggressive or Moderate Lipid Lowering Therapy with Pitavastatin in Coronary Artery Disease (REAL-CAD) study.

Methods: The REAL-CAD study was a prospective, multicenter, open-label trial. As a sub-study, we examined the prognostic impact of non-lipid residual risk factors, including blood pressure, glucose level, and renal function, in patients who achieved LDL-C <100 mg/dL at 6 months after pitavastatin therapy. Each risk factor was classified according to severity. The primary outcome was a composite of cardiovascular death, nonfatal myocardial infarction, nonfatal ischemic stroke, and unstable angina requiring emergency hospitalization.

Results: Among 8,743 patients, the mean age was 68±8.2 years, and the mean LDL-C level was 84.4±18 mg/dL. After adjusting for the effects of confounders, an estimated glomerular filtration rate (eGFR) ≤ 60 mL/min/1.73 m^2 showed the highest risk of the primary outcome (hazard ratio [HR] 1.92; 95% confidence interval [CI] 1.45-2.53). The combination of eGFR ≤ 60 and hemoglobin A1c (HbA1c) ≥ 6.0% also showed the highest risk of all-cause death (HR, 2.42; 95% CI, 1.72-3.41).

Conclusions: In patients with stable CAD treated with pitavastatin and who achieved guidelines-directed levels of LDL-C, eGFR and HbA1c were independently associated with adverse events, suggesting that renal function and glycemic control could be residual non-lipid therapeutic targets after statin therapy.

International Journal of *Molecular Sciences*

MDPI

Review

Novel Biomarkers of Atherosclerotic Vascular Disease—Latest Insights in the Research Field

Cristina Andreea Adam [1], Delia Lidia Șalaru [1,2,*], Cristina Prisacariu [1,2], Dragoș Traian Marius Marcu [2], Radu Andy Sascău [1,2] and Cristian Stătescu [1,2]

[1] Institute of Cardiovascular Diseases "Prof. Dr. George I.M. Georgescu", 700503 Iași, Romania; adam.cristina93@gmail.com (C.A.A.); prisacariu88@yahoo.com (C.P.); radu.sascau@gmail.com (R.A.S.); cstatescu@gmail.com (C.S.)
[2] Department of Internal Medicine, University of Medicine and Pharmacy "Grigore T. Popa", 700115 Iași, Romania; dragos.marcu11@yahoo.com
* Correspondence: deliasalaru@gmail.com

Abstract: The atherosclerotic vascular disease is a cardiovascular continuum in which the main role is attributed to atherosclerosis, from its appearance to its associated complications. The increasing prevalence of cardiovascular risk factors, population ageing, and burden on both the economy and the healthcare system have led to the development of new diagnostic and therapeutic strategies in the field. The better understanding or discovery of new pathophysiological mechanisms and molecules modulating various signaling pathways involved in atherosclerosis have led to the development of potential new biomarkers, with key role in early, subclinical diagnosis. The evolution of technological processes in medicine has shifted the attention of researchers from the profiling of classical risk factors to the identification of new biomarkers such as midregional pro-adrenomedullin, midkine, stromelysin-2, pentraxin 3, inflammasomes, or endothelial cell-derived extracellular vesicles. These molecules are seen as future therapeutic targets associated with decreased morbidity and mortality through early diagnosis of atherosclerotic lesions and future research directions.

Keywords: biomarkers; atherosclerosis; prognosis; development; research; midkine; pentraxins; inflammasomes

check for updates

Citation: Adam, C.A.; Șalaru, D.L.; Prisacariu, C.; Marcu, D.T.M.; Sascău, R.A.; Stătescu, C. Novel Biomarkers of Atherosclerotic Vascular Disease—Latest Insights in the

Current Atherosclerosis Reports
https://doi.org/10.1007/s11883-023-01141-y

Check for updates

Treatment of Lp(a): Is It the Future or Are We Ready Today?

Alexandros D. Tselepis[1]

Accepted: 15 August 2023

Abstract

Purpose of Review The goal of this review is to present the pharmacodynamic effectiveness as well as the clinical efficacy and safety of investigational antisense oligonucleotides (ASOs) and small interference RNAs (siRNAs) drugs that specifically target lipoprotein(a) (Lp(a)). The review will discuss whether the existing lipid-lowering therapies are adequate to treat high Lp(a) levels or whether it is necessary to use the emerging new therapeutic approaches which are based on the current RNA technologies.

Recent Findings Lipoprotein(a) (Lp(a)) is a causal risk factor for atherosclerotic cardiovascular disease (ASCVD), independent of other conventional risk factors. High Lp(a) levels are also independently associated with an increased risk of aortic stenosis progression rate. Plasma Lp(a) levels are primarily genetically determined by variation in the LPA gene coding for apo(a). All secondary prevention trials have demonstrated that the existing hypolipidemic therapies are not adequate to reduce Lp(a) levels to such an extent that could lead to a substantial reduction of ASCVD risk. This has led to the development of new drugs that target the mRNA transcript of LPA and efficiently inhibit Lp(a) synthesis leading to potent Lp(a) reduction. These new drugs are the ASO pelacarsen and the siRNAs olpasiran and SLN360. Recent pharmacodynamic studies showed that all these drugs potently reduce Lp(a) up to 98%, in a dose-dependent manner. Ongoing clinical trials will determine the Lp(a)-lowering efficacy, tolerability, and safety of these drugs as well as their potential effectiveness in reducing the ASCVD risk attributed to high plasma Lp(a) levels.

Summary We are not ready today to significantly reduce plasma Lp(a). Emerging therapies potently decrease Lp(a) and ongoing clinical trials will determine their effectiveness in reducing ASCVD risk in subjects with high Lp(a) levels.

Keywords Antisense oligonucleotides · Apolipoprotein (a) · Coronary artery disease · CRISPR/Cas9 · Small interference RNAs · Atherosclerosis · Calcific aortic valve stenosis · Lipoprotein (a)

Volume 50, June 2023, 101822

Review

Two decades of vaccine development against atherosclerosis

Author links open overlay panelMiguel A. Moreno-Gonzalez [a b 1], Oscar A. Ortega-Rivera [a b 1], Nicole F. Steinmetz [a b c d e f]
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Abstract

Atherosclerosis is an immune-mediated chronic inflammatory disease that leads to the development of fatty plaques in the arterial walls, ultimately increasing the risk of thrombosis, stroke, and myocardial infarction. The immune response in this complex disease is both atheroprotective and pro-atherogenic, involving both innate and adaptive immunity. Current treatments include the adjustment of lifestyle factors, cholesterol-lowering drugs such as statins, and immunotherapy, whereas vaccine development has received comparatively little attention. In this review, we discuss the potential of antigen-specific vaccination as a preventative approach based on more than 20 years of research and innovation. Vaccination targets include proteins that are more abundant in atherosclerotic patients, such as oxidized low-density lipoprotein (LDL), apolipoprotein B-100, proprotein convertase subtilisin/kexin type-9 serine protease (PCSK9), cholesteryl ester transfer protein (CETP), and heat shock proteins HSP60 and HSP65. Immunization with such proteins or their peptide epitopes has been shown to induce T-cell activation, produce antigen-specific antibodies, reduce the size of atherosclerotic lesions, and/or reduce serum cholesterol levels. Vaccination against atherosclerosis therefore offers a new strategy to address the burden on healthcare systems caused by cardiovascular disease, the leading cause of death worldwide.

Current Atherosclerosis Reports
https://doi.org/10.1007/s11883-023-01140-z

Check for updates

Updates in Drug Treatment of Severe Hypertriglyceridemia

Ioanna Gouni-Berthold[1] · Jonas Schwarz[1] · Heiner K. Berthold[2]

Accepted: 15 August 2023

Abstract

Purpose of Review To provide an insight into the new pharmacological options for the treatment of severe hypertriglyceridemia (sHTG).

Recent Findings sHTG is difficult to treat. The majority of the traditional pharmacological agents available have limited success in both robustly decreasing triglyceride levels and/or in reducing the incidence of acute pancreatitis (AP), the most severe complication of sHTG. Therapeutic options with novel mechanisms of action have been developed, such as antisense oligonucleotides (ASO) and small interfering RNA (siRNA) targeting *APOC3* and *ANGPTL3*. The review discusses also 2 abandoned drugs for sHTG treatment, evinacumab and vupanorsen.

Summary The ASO targeting *APOC3*, volanesorsen, is approved for use in patients with familial chylomicronemia syndrome (FCS) in Europe. Olezarsen, an N-acetylgalactosamine (GalNAc)-conjugated ASO with the same target, seems to have a better safety and efficacy profile. siRNA targeting *APOC3* and *ANGPTL3*, namely ARO-APOC3 and ARO-ANG3, are also promising for the treatment of sHTG. However, the ultimate clinical goal of any sHTG treatment, the decrease in the risk of AP, has not been definitively achieved till now by any pharmacotherapy, either approved or in development.

Keywords Triglycerides · Hypertriglyceridemia-induced acute pancreatitis · Volanesorsen · Olezarsen · ARO-APOC3 · ARO-ANG3

Eur J Clin Invest. 2023 Aug 12;e14083. doi: 10.1111/eci.14083. Online ahead of print.

Evaluation of high-density lipoprotein-bound long non-coding RNAs in subjects with familial hypercholesterolaemia

Roberto Scicali [1], Giosiana Bosco [1], Alessandra Scamporrino [1], Stefania Di Mauro [1], Agnese Filippello [1], Francesco Di Giacomo Barbagallo [1], Salvatore Spampinato [1], Chiara Pavanello [2], Alice Ossoli [2], Antonino Di Pino [1], Laura Calabresi [2], Francesco Purrello [1], Salvatore Piro [1]

Affiliations + expand

PMID: 37571980 DOI: 10.1111/eci.14083

Abstract

Background: Long non-coding RNAs (lncRNAs) could be attractive circulating biomarkers for cardiovascular risk stratification in subjects at high atherosclerotic cardiovascular disease risk such as familial hypercholesterolaemia (FH). Our aim was to investigate the presence of lncRNAs carried by high-density lipoprotein (HDL) in FH subjects and to evaluate the associations of HDL-lncRNAs with lipoproteins and mechanical vascular impairment assessed by pulse wave velocity (PWV).

Methods: This was a retrospective observational study involving 94 FH subjects on statin treatment. Biochemical assays, HDL purification, lncRNA and PWV analyses were performed in all subjects.

Results: LncRNA HIF1A-AS2, LASER and LEXIS were transported by HDL; moreover, HDL-lncRNA LEXIS was associated with Lp(a) plasma levels ($p < .01$). In a secondary analysis, the study population was stratified into two groups based on the Lp(a) median value. The high-Lp(a) group exhibited a significant increase of PWV compared to the low-Lp(a) group (9.23 ± .61 vs. 7.67 ± .56, $p < .01$). While HDL-lncRNA HIF1A-AS2 and LASER were similar in the two groups, the high-Lp(a) group exhibited a significant downregulation of HDL-lncRNA LEXIS compared to the low-Lp(a) group (fold change -4.4, $p < .0001$). Finally, Lp(a) and HDL-lncRNA LEXIS were associated with PWV (for Lp(a) $p < .01$; for HDL-lncRNA LEXIS $p < .05$).

Conclusions: LncRNA HIF1A-AS2, LASER and LEXIS were transported by HDL; moreover, significant relationships of HDL-lncRNA LEXIS with Lp(a) levels and PWV were found. Our study suggests that HDL-lncRNA LEXIS may be useful to better identify FH subjects with more pronounced vascular damage.

RECENT EVENTS

ISAR-DCCON 2022 CONFERENCE: HIGHLIGHTS

The 7th Annual Conference of Indian Society for Atherosclerosis Research, Delhi Chapter (ISAR-DCCON 2022) was organized by Department of Biochemistry, ESIC Medical College & Hospital Faridabad on 10th September, 2022 at Dhanwanti Hall, ESIC Medical College & Hospital. The Organizing Secretary was Prof SB Sharma, Professor, Department of Biochemistry, ESIC Medical College & Hospital, Faridabad and President, ISAR – Delhi Chapter. It was a full day conference that was well attended by more than 150 delegates which included students, scientists and faculty from various fields in medicine from Delhi & NCR. This conference was also accredited by Delhi Medical Council for 5.5 Credit hours vide their letter no 3458/DMC/CME/16C/2/2022 dated 7.7.2022. The news media was also present to cover the proceedings of the conference.

The inauguration ceremony was presided by eminent personalities which included the glittering presence of Chief Guest, Prof Randeep Guleria, Director, AIIMS New Delhi, and Guest of Honor, Prof O P Kalra, Vice-Chancellor, SGT University, Gurgaon, Ex-Vice Chancellor, Pandit Bhagwat Dayal Sharma University of Health Sciences, Rohtak along with Prof Asim Das, Dean, and Prof A K Pandey, Dean (Academics) and Registrar, from ESIC Medical College & Hospital. The conference had a very relevant theme, 'New Horizons in the Diagnosis & Treatment of Atherosclerosis' and a galaxy of eminent speakers from Delhi and also abroad (Prof Hari Singh, The Netherlands) were invited to deliver lectures on pertinent topics.

The scientific programme of the conference included Key note address on 'Statin therapy for atherosclerotic plaque: regression or healing' by Prof Sanjay Tyagi, Professor of Excellence, Department of Cardiology, GB Pant Institute of Post Graduate Medical Education, Delhi and Ex-DG Health Services, GOI and several Invited lectures on some very interesting and pertinent topics as given below:

1. COVID and cardiovascular disease by Prof Vinod Sharma, Interventional Cardiologist, National Heart Institute, New Delhi
2. Updates in the Management of CAD: Focus on Acute MI by Prof Mohan Nair, Consultant cardiologist ESIC Medical College & Hospital, Faridabad
3. Stunted angiogenesis and vascular remodeling in the failing heart by Prof Hari S Sharma, Department of Pathology & Clinical Bioinformatics, Erasmus MC, University Medical Center, Rotterdam, The Netherlands
4. Atherosclerosis since antiquity by Prof S Dwivedi, Consultant Cardiologist, National Heart Institute, New Delhi

5. Cardiovascular complications of long COVID-19 by Prof Ambuj Roy, Department of Cardiology, AIIMS, New Delhi
6. Primordial prevention of adult disease in the first 1000 days of life by Dr Harsh Pal Singh Sachdev, Consultant Pediatrician, Sita Ram Bhartia Institute of Science Research, New Delhi
7. Sub-clinical atherosclerosis and arterial stiffness by Prof A K Pandey, Head, Department of Physiology, ESIC Medical College & Hospital, Faridabad
8. Integrated multidisciplinary approach to atherosclerosis by Prof Praveen Malik, Prof Mukta Pujani, Prof Monica Aggarwal, Prof Gini Garima from Departments of Medicine, Pathology, Pharmacology and Biochemistry respectively, ESIC Medical College & Hospital, Faridabad.

The various sessions were chaired by eminent personalities like Prof Asim Das, Prof S Dwivedi, Prof D K Srivastava, Prof Jayashree Bhattacharya, Prof Ritu Singh, Prof S B Sharma, Prof Nibhriti Das, Prof Praveen Malik, Prof Gajendra Singh Ranga, Prof Amitesh Agarwal, Prof Zahid Ashraf, Prof Anita Khalil, Prof Kalpana Luthra and Prof Jagriti Bhatia.

There were 28 Poster presentations in the conference; 10 in basic research category and 18 in clinical research category. The posters were chaired and judged by Dr Pradeep Dabla, Dr Archna Singh, Dr Rajeev Goyal, Dr Anita Bhatia and Dr Rachna Agarwal. In both basic and clinical categories, the best three presentations were awarded 'Young Investigator Award' first, second and third prizes. The award recipients in both the categories were as under:

Name and affiliation of the Presenter	Award (Category)	Title of the paper
	BASIC	
Dr. Shamima Akhtar Department of Biochemistry, AIIMS, New Delhi E-mail: ashamima05@gmail.com	First (Basic)	Autophagy-regulated Sialin (SLC17A5) protein controls endothelial dysfunction by affecting nitrate flux
Ms Sadia Shah Department of Pharmacology, AIIMS, New Delhi E-mail: shah.syed1990@gmail.com	Second (Basic)	SMA signaling pathway in vitamin D-induced amelioration of pulmonary artery hypertension in ratsα/βInvolvement of eNOS/TGF

Raishal Safdar Department of Biotechnology, Jamia Millia Islamia E-mail: raishalsafdar@gmail.com	Third (Basic)	Antagonistic effects of 6-shogaol, an active constituent of Zingiber officinale on inflammasome mediated thrombotic complications
	CLINICAL	
Ms Komal Sagar Department of Biochemistry, AIIMS, New Delhi E-mail: komalsagar28@gmail.com	First (Clinical)	Defective homing of MDSCs and Tregs contributes to atherosclerosis progression
Mr Dharmsheel Shrivastava Amity Institute of Biotechnology, Amity University, Jaipur E-mail: dharmsheel.shrivastav1@gmail.com	Second (Clinical)	The severity of coronary artery stenosis with type II diabetes mellitus
Dr. Pankaj Kumar Gupta Department of Pharmacology, Al Falah School of Medical Science & Research Centre, Dhouj, Faridabad (Haryana) Email: drpankajgupta2000@yahoo.com	Third (Clinical)	Atherosclerosis: A Review & Analysis of Drug Development Pipeline

The Executive Meeting and General Body Meeting of ISAR Delhi Chapter were held followed by presentation of cultural programme by the students of ESIC Medical College & Hospital. The Prize distribution and Valedictory function was organized after the end of the cultural programme. The valedictory session ended with a vote of thanks from Dr Gini Garima and Prof S B Sharma and this was followed by tea and dispersal of all the delegates.


ESIC Medical College & Hospital, Faridabad
7th ANNUAL CONFERENCE OF
INDIAN SOCIETY FOR ATHEROSCLEROSIS RESEARCH
DELHI CHAPTER
EXIT


DELHI CHAPTER


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ESIC Medical College & Hospital
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ORGANIZER

ANNUAL PICNIC EXTRAVAGANZA OF ISAR DELHI CHAPTER

All members of ISAR-Delhi Chapter were invited along with family and friends for a gala picnic at Madhavgarh Farms Aravali, Village Tikli, Gurugram on 18th December, 2022 (Sunday) for a full day of fun and frolic from 9 am to 5 pm. Approximately 90 people attended the gala picnic including ISAR-DC members and their family and friends. The picnic started with a welcome drink for everyone, followed by breakfast, lunch and evening tea and snacks. There were plenty of games, zip line, commando net, rock climbing, rappelling wall and rides (horse carriage, bullock cart, camel cart, tractor ride), pottery making and painting, mehandi, cultural performance and live DJ and much more. The tickets were nominally charged at Rs 700/- per person (children up to 3 ft height were complimentary), for children between 3 ft to 4 ft height, the ticket price was Rs 400/- and for children above 4 ft height, the ticket price was Rs 700/-.

UPCOMING EVENTS

1. DC-ISAR 2023 on 3rd November at LHMC

DEPARTMENT OF BIOCHEMISTRY
LADY HARDINGE MEDICAL COLLEGE, NEW DELHI

UNDER THE AEGIS OF

THE DELHI CHAPTER OF
INDIAN SOCIETY FOR ATHEROSCLEROSIS RESEARCH

ANNUAL C.M.E DC-ISAR 2023

"Holistic Approach to the Management of Coronary Artery Disease"

3RD NOVEMBER 2023
11.00 A.M. - 05.00 P.M.

Venue:
Mini-Auditorium, 5th Floor, New Academic Block

Se. No.	Time	Programme Schedule	
		Speaker	Topic
1	11.00 A.M. – 01.00 P.M.	Convenor: **Dr. Parijat Gogoi & Dr. Preeti Chauhan** Professors, Biochemistry, LHMC	Parallel Sessions: Oral/Poster Presentation
2	01.00 P.M. – 02.00 P.M.	**Lunch**	
3	02.00 P.M. – 02.20 P.M.	**Dr. Zahid Ashraf** Prof. & HOD Dept. of Biotechnology Dean, Jamia Milia Islamia University	"Genetic Predisposition to Cardiovascular Diseases: An Indian Perspective"
4	02.20 P.M. – 02.50 P.M.	**Dr. Sanjay Tyagi** Clinical Director & Head, Apollo Hospitals Group, Northern Region. President-Elect: Cardiological Society of India *Former:* Director General of Health Services Government of India HOD Cardiology, MAMC	"Atherosclerotic Vascular Disease: Non-Surgical Management"
5	02.50 P.M. – 03.10 P.M.	**Inauguration, Felicitation & Release of Abstract Booklet**	
6	03.10 P.M. – 03.20 P.M.	**Dr. Pratip Jana & Dr. Roshnara PP** Senior Residents, Biochemistry, LHMC	**Real-time Audience Digital Interaction for Self-Analysis of Current Lifestyle.**
7	03.20 P.M. – 03.50 P.M.	**Dr. Shridhar Dwivedi** Senior Consultant Cardiologist & Academic Head National Heart Institute *Former:* Dean, Hamdard Institute of Medical Sciences & Research HOD Medicine, UCMS & GTB hospital	"Half a Century Journey with Atherosclerosis."
8	03.50 P.M. – 04.00 P.M.	**DC-ISAR Patrons & Dr. S. K. Rasania** Vice Principal, LHMC & Associated Hospitals	**Prize Distribution Ceremony for winners of oral and poster presentations.**
9	04.00 P.M. – 04.05 P.M.	**Dr. Smita Tripathi** Professor, Biochemistry, LHMC	**Vote of Thanks**
10	04.05 P.M. – 04.30 P.M.	**High Tea**	
11	04.30 P.M. – 05.00 P.M.	**DC – ISAR General Body Meeting**	

Organizing Secretary & President DC-ISAR
Dr. Ritu SIngh

Joint Organizing Secretaries:
Dr. Mukesh Kumar Meena **Dr. Parul Goyal** **Dr. Rajeev Goyal**

REGISTRATION
& ABSTRACT SUBMISSION

Scientific Chairpersons:
Dr. Seema Bhargava **Dr. Harshvardhan Singh** **Dr. Jagriti Bhatia**

For Registration either
Scan the QR Code or press button below

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Registration Fee:
For ISAR members: Rs 250/-
Non-Members: Rs 500/-
Transaction ID has to be submitted in the Google form during registration

Account Details:

- Account Name: **Delhi Chapter ISAR**
- Bank Name: **Central Bank of India, Lady Hardinge Medical College New Delhi - 110001**
- Account Number: **3432232021**
- IFSC: **CBIN0283462**

Last Date of Abstract Submission: 28th October 2023

Registration entitles one to:

1. **Conference Material & Bag**
2. **Lunch**
3. **Participation in Oral/Poster Presentation**

Cash Prizes!

FOR WINNERS!!!

For Any Queries, Please mail to:
isar.dc.lhmc2023@gmail.com

2. 35th Annual Cardiologists Conference on November 13-14, 2023 Barcelona, Spain

3. 39th World Cardiology Conference on January 25-26, 2024 London, UK

4. 4th Immuno-Metabolic Mechanisms of Atherosclerosis Conference

   04 May 2024 - 07 May 2024, Cancun, Mexico

5. Vascular Discovery 2024 is sponsored and organized by the AHA Councils on Arteriosclerosis, Thrombosis and Vascular Biology and Peripheral Vascular Disease, and in collaboration with the AHA Council on Genomic and Precision Medicine.
   May 15–18, 2024
   Chicago Hilton | Chicago, Illinois

MEMBERSHIP FORM

LIFE MEMBERSHIP OF INDIAN SOCIETY FOR ATHEROSCLEROSIS RESEARCH – NATIONAL BODY & DELHI CHAPTER

Life Membership of Delhi Chapter and National body of Indian Society of Atherosclerosis Research – Rs 5000/-. Membership of the ISAR National body automatically gives membership of the International Atherosclerosis Society.

Please paste your recent colour passport size photograph

First Name .....

Last Name .....

Academic Qualification .....

Designation .....

Office Address .....

.....

Office Tel No .....

Email.....

Mobile Number .....

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Residence Address

.....

.....

Residence Tel No .....

Area of Research

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Details of Publication/Presentation/Poster/Thesis in Atherosclerosis:

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Nominated by (Name of Life Member of ISAR National Body and their Life Membership Number)

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Life Membership fee: Rs 5,000 (ISAR + IAS + Delhi Chapter)

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PRESIDENT

Dr Ritu Singh
Director Professor & HOD,
Dept. of Biochemistry
& Coordinator Medical Education Unit
LHMC & Associated SSKH& KSCH hospitals
Shaheed Bhagat Singh Marg
New Delhi 110001
Mobile : +91 - 9811173213
Email : drritusingh19@gmail.com

SECRETARY

Dr Harsh Vardhan Singh
Senior Biochemist,
Department of Biochemistry,
North Delhi Municipal Corporation Medical College & Hindu Rao Hospital, Delhi-110007
Mobile: +91-9810910824
Email :
Secretary.delhi.chapter.isar@gmail.com

TREASURER

Dr Rajeev Goyal
Associate Professor
Dept. of Biochemistry
LHMC and associated hospitals
Shaheed Bhagat Singh Marg
New Delhi 110001
Mobile: +91 – 8191861234
Email : docrajeev@gmail.com

OFFICE BEARERS

S. No.	Name	Designation
1	Dr Ritu Singh	President
2	Dr Jagriti Bhatia	President Elect
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4	Dr Harsh Vardhan Singh	Secretary
5	Dr Rajeev Goyal	Treasurer
6	Dr Anita Bhatia	Joint Secretary
7	Dr Parul Goyal	Executive members
8	Dr Abhishek Jaiswal	Executive members
9	Dr Kamna Srivastava	Executive members
10	Dr Anjali Manocha	Executive members
11	Dr Harlokesh Narayan Yadav	Executive members
12	Dr Pradeep Dabla	Executive members
13	Dr Sudheer Arava	Executive members
14	Dr Rachna Agarwal	Executive members
15	Dr Archna Singh	Executive members
16	Dr Bhumika Upadhyay	Executive members