

Clinical Cardiology 2018 :Atherosclerosis: molecular mechanisms, current/emerging therapies, and the potential of nutraceuticals as preventative/therapeutic agents for this disease -Dipak P Ramji -Cardiff University

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Atherosclerosis, an inflammatory disorder of the vasculature and the underlying cause of myocardial infarction and cerebrovascular accidents, is responsible for more global deaths than any other disease. Although some reduction in morbidity and mortality from atherosclerosis and its complications has been achieved recently by lifestyle changes and pharmaceutical intervention, this is expected to reverse in the future because of global increase in risk factors such as obesity and diabetes. Current pharmaceutical therapies against atherosclerosis are associated with considerable residual risk for cardiovascular disease together with other issues such as side effects and patient-dependent efficacy. In addition, pharmaceutical agents against many promising targets have proved disappointing at the clinical level. It is therefore essential that the molecular basis of atherosclerosis is fully understood and new therapeutic/preventative agents or targets are identified. The major focus of research in my laboratory is to understand the molecular mechanisms underlying the impact of inflammation and factors involved in orchestrating the inflammatory response, such as cytokines, on atherosclerosis with emphasis on macrophages, which are involved in all stages of the disease, together with the actions of preventative/therapeutic agents. Our research has particularly provided novel insights into the mechanisms underlying the actions of cytokines and nutraceuticals. This presentation will discuss the molecular basis of atherosclerosis and opportunities for drug discovery, current therapies against the disease and their limitations, emerging therapies targeting lipid metabolism and the inflammatory response, and the potential of nutraceuticals as preventative/therapeutic agents.

Atherosclerosis refers to the buildup of fats, cholesterol and other substances in and on your artery walls (plaque), which may restrict blood flow. The plaque can burst, triggering a grume. Although atherosclerosis is usually considered a heart problem, it can affect arteries anywhere in your body. Atherosclerosis happens when the endothelium becomes damaged, thanks to factors like smoking, high vital sign, or high levels of glucose, fat, and cholesterol within the blood. This damage allows a set of drugs, referred to as plaque, to create up within the artery wall. These substances include fat and cholesterol. Medications for treating atherosclerosis include: cholesterol-lowering medications, including statins and fibrates. Angiotensin-converting enzyme (ACE) inhibitors, which can help prevent narrowing of your arteries. Beta-blockers or calcium channel blockers to lower your vital sign. Medical treatment combined with lifestyle and dietary changes are often wont to keep atherosclerosis from getting worse, but they are not ready to reverse the disease. Some medications can also be prescribed to extend your comfort, particularly if you're having chest or leg pain as a symbol. Living healthy with atherosclerosis is feasible,

though, and it is vital. Counting on where plaques are found, the National Institutes of Health reports that atherosclerosis can lead to: Coronary heart condition, which occurs when blood flow is restricted to the guts muscle. Exercise Can Reverse Atherosclerosis. Studies combining lipid-lowering agents with aggressive lifestyle changes including exercise and a diet have had similar findings. The high-density cholesterol in your body, or good cholesterol, removes bad cholesterol from your arteries and helps fight heart attacks and strokes. Mild atherosclerosis usually doesn't have any symptoms. You always won't have atherosclerosis symptoms until an artery is so narrowed or clogged that it can't supply adequate blood to your organs and tissues. Quite sizable amount of studies on atherosclerosis are published so far. Thus, early lesions of atherosclerosis are reversible and cholesterol-lowering therapy is an efficient treatment; however, since advanced lesions seem to be irreversible, cholesterol-lowering therapy might not be effective for such lesions. You'll have an attack and not even realize it. In serious cases, medical procedures or surgery can help to get rid of blockages from within the arteries. A doctor can also prescribe medication, like aspirin, or cholesterol-reducing drugs, like statins. Eating many Cheese Won't Necessarily Clog Your Arteries. Except for years, ultra-fit types had blacklisted cheese as unnecessarily loaded with saturated fat and cholesterol, fearing that it could clog arteries. Turmeric contains powerful anti-inflammatory compounds which will help reduce damage to arterial walls. Inflammation levels are shown to possess an immediate effect on arteriosclerosis — the hardening of the arteries. Add it to your diet: the simplest thanks to add turmeric to your diet is by making turmeric tea. It can desire an uncomfortable pressure, squeezing, or pain. Discomfort in other upper-body areas, like one or both arms, the back, the neck, the jaw, or the stomach. Shortness of breath before or during chest discomfort. Breaking call at a chilly sweat, or feeling nauseated or lightheaded. Attack symptoms can last for a couple of minutes to a couple of hours. If you've got had pain continuously for several days, weeks or months, then it's unlikely to be caused by an attack. Xu says the bulk of patients experience somewhat typical symptoms, like radiating pain, heaviness or discomfort, heart palpitations, cold sweats, and shortness of breath. Both sorts of symptoms are often experienced months before an actual attack occurs.

Biography

Dipak P Ramji is Professor of Cardiovascular Science at the School of Biosciences in Cardiff University. He received his BSc (Hons) degree (Biochemistry) and his PhD (Molecular Biology) from the University of Leeds. This was followed by post-doctoral research at the European Molecular Biology Laboratory (Heidelberg) and the Istituto di Ricerche di Biologia Molecolare P. Angeletti (Rome) with fellowships from the Royal Society and the EU. He joined Cardiff University in 1992 and completed 25 years of service in August 2017. His research is focused

on understanding how the immune and inflammatory responses regulate cellular processes in heart disease with the goal of attaining deeper mechanistic insight and identifying preventative/therapeutic agents. His research has been funded by several organisations and received continuous funding from the British Heart Foundation since 1997. He has published over 150 research articles (h index 34 and i10 index 68 with over 5700 citations).

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