Pathogenesis of atherosclerosis pdf

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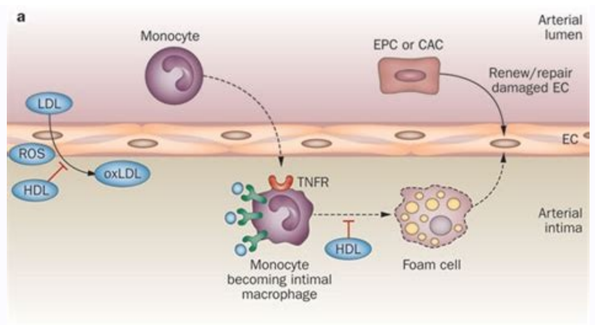
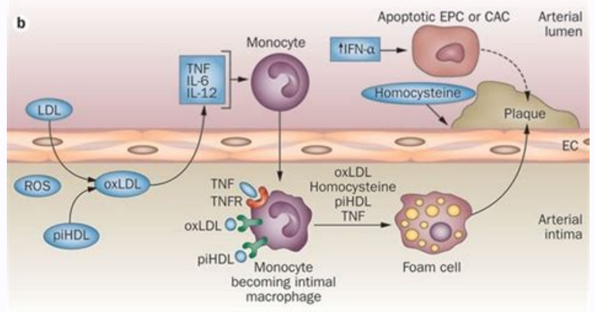
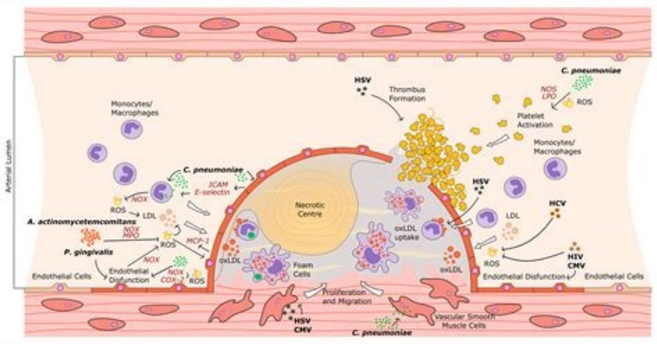
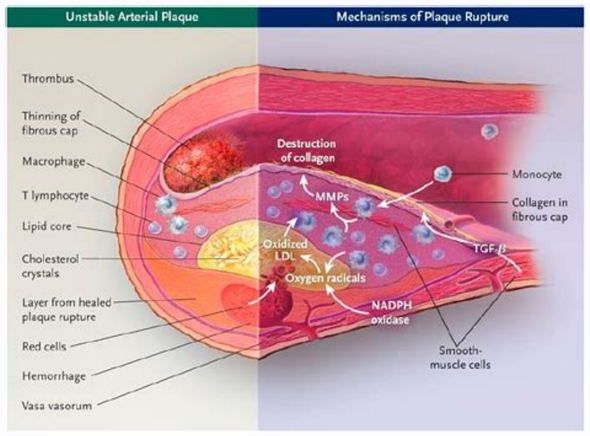
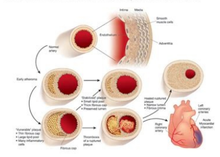




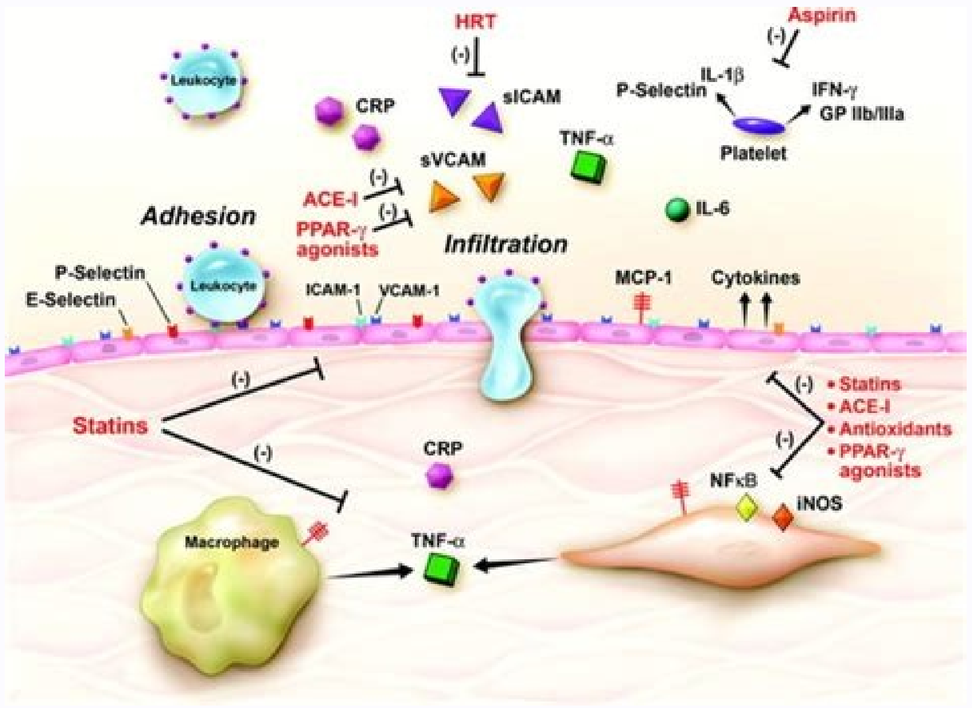




Atherosclerosis is an inflammatory disease with a critical role for monocyte recruitment



Libby, Nature, 2002;420: 868-874.



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Atherosclerosis is a multifocal, smoldering, immunoinflammatory disease of medium-sized and large arteries fuelled by lipids. Endothelial cells, leukocytes, and intimal smooth muscle cells are the major players in the development of this disease. The most devastating consequences of atherosclerosis, such as heart attack and stroke, are caused by superimposed thrombosis. Therefore, the vital question is not why atherosclerosis develops but rather why atherosclerosis, after years of indolent growth, suddenly becomes complicated with luminal thrombosis. If thrombosis-prone plaques could be detected and thrombosis averted, atherosclerosis would be a much more benign disease. Approximately 76% of all fatal coronary thrombi are precipitated by plaque rupture. Plaque rupture is a more frequent cause of coronary thrombosis in men (approximately 80%) than in women (approximately 60%). Ruptured plaques are characterized by a large lipid-rich core, a thin fibrous cap that contains few smooth muscle cells and many macrophages, angiogenesis, adventitial inflammation, and outward remodeling. Plaque rupture is the most common cause of coronary thrombosis. Ruptured plaques and, by inference, rupture-prone plaques have characteristic pathoanatomical features that might be useful for their detection in vivo by imaging. This article describes the pathogenesis of atherosclerosis, how it begets thrombosis, and the possibility to detect thrombosis-prone plaques and prevent heart attack. PDF Split View Article contents Figures & tables Video Audio Supplementary Data Hardening of the arteries (arteriosclerosis or atherosclerosis), involving principally the vessels of the brain, heart and kidneys, is a major cause of disability or death. Diseases that may follow or occur with arteriosclerosis include kidney disease, high blood pressure, uremia, apoplexy, premature senility, angina pectoris, coronary heart disease and coronary thrombosis.Hardening of the arteries may occur in the extremities, as may other diseases of the arteries and veins. Perhaps the most important form of arteriosclerosis is coronary heart disease. This leads to narrowing of the arteries that carry blood to the heart muscle, or to clots within their lumen – both of which block circulation. Knowledge of preventive measures against arteriosclerotic vascular and heart disease is in the earliest stage. Intensive research has recently produced limited advances, for example, in the use of anti-coagulant therapy in coronary thrombosis and other thrombo-embolic phenomena. Although this therapy merits widespread use, many areas lack both the facilities and personnel required for its extensive application.* * *Arteriosclerosis is a disease of the arteries affecting primarily the intimal coat and characterized in general by increasing accumulations of lipids and increasing fibrous thickening in localized areas of the intima. The frequent occurrence of coalescence of individual lesions and of further degenerative changes in them is well recognized. The further degenerative changes include necrosis of the centers of the arteriosclerotic lesions with the formation of cavities filed with lipid-rich debris (atheromata), the extension of the process to involve the media, necrosis and disintegration of the intimal lining over atheromata and calcification of the lesions. These changes progress in the absence of recognized clinical signs or symptoms, or abnormalities detectable at present by laboratory methods, until the disease has caused significant impairment of blood supply.Narrowing or occlusion of coronary, cerebral or peripheral arteries brought about by arteriosclerosis with or without thrombosis accounts in preponderant measure for the morbidity and mortality associated with the disease. The fact that there is no way of studying the lesion itself in the living patient, or of determining whether it is developing, regressing or remaining static at any particular time, makes extremely difficult the study of the factors that may be involved.Sterols and their esters make up between 85% and 90% of the lipids in arteriosclerotic lesions. About 5% consists of phospholipids and less than 4% of neutral fats. About 60% of the sterols are esterfied with fatty acids. Although most of the sterol consists of cholesterol, small amounts of dihydrocholesterol are present together with smaller quantities of oxidation products of cholesterol. These facts together with some data on changes which occur in the mineral content during calcification of the lesions and in the relative amounts of elastin, collagen, and chondroitin sulfuric acid which accompany fibrosis, constitute the bulk of the biochemical information which can with certainty be associated with arteriosclerosis.It seems probable that the lipids in the lesions come from the lipids of the blood although direct proof of this is not available. If this is true any factor that influences the chemical composition, the level, the stability or the physical state of the lipids in the blood may be of importance in the pathogenesis of the disease.The difficulties, already mentioned, of studying the development of arteriosclerosis in human subjects has forced investigators in this field to rely very largely on the study of counterparts of the human disease produced in experimental animals by rather artificial methods, most of which have in common the production of hypercholesterolemia.Arteriosclerosis in man develops earlier and more frequently in individuals with high blood cholesterol levels than in those with normal blood cholesterol, but there is no evidence that a level of blood cholesterol higher than normal is essential to the development of arteriosclerosis in man. Although experimental arteriosclerosis is commonly produced in animals with hypercholesterolemia, recent evidence shows that the absolute level of cholesterol in the blood is not the determining factor. On the contrary, the relation of cholesterol to other substances in the blood appears to be more important than its absolute quantity. A low phospholipid cholesterol ratio can be more closely correlated with the development of both experimental and human arteriosclerosis than can hypercholosterolemia itself.Preoccupation with the investigation of blood lipids in relation to the pathogenesis of arteriosclerosis has led to neglect of other factors. It is not known whether changes in endothelial permeability or in the subendothelial ground substance play a role in the deposit of lipids. Virtually nothing is known of the possible importance of phagocytosis of lipids by lining endothelial cells or by other cells in the subendothelial layer. Neither is anything known of the metabolic activities of these cells in relation to the local deposition of lipids.The localization of arteriosclerotic lesions may be determined by variations in the structure and composition of the vessel wall itself or by mechanical or hydrodynamic forces acting at particular points in the arterial walls. However, with regard to the relative importance of such localizing factors practically no dependable evidence exists. Diseases of the coronary arteries Definition For its ability to function effectively as a pump the myocardium is dependent upon an adequate supply of blood, brought to it almost entirely by the coronary arteries. The role of other accessory channels is minimal. Disease may interfere with coronary flow by (1) diminishing the elasticity of the vascular walls; (2) narrowing the lumina; (3) occlusion. Ischemia and hypoxia induce changes in the heart muscle which impair its functional capacity and eventually may lead to disturbances in the general circulation. To this syndrome is applied the designation “coronary heart disease.” Incidence The number of deaths from diseases of the coronary arteries, as well as the number of individuals disabled by them, is increasing. The growing incidence can be ascribed in part to the aging of the population and in part to better diagnosis. Those in the older age groups are more likely to suffer from arteriosclerosis; greater familiarity with the symptoms and signs, as well as refinements in diagnostic techniques, has placed the proper label on mild or atypical cases. Whether other factors are operative is unknown. Among these have been mentioned the control of many infectious diseases occurring particularly in early life, excessive smoking, the accelerated pace of modern existence and a dietary regimen rich in fats in countries in which the scale of living is relatively high.Coronary disease occurs 3 or 4 times more often in males than in females. Its importance is greatest in middle life, although it may occur in youth and is common in persons of advanced years.There is no sound evidence that occupation, social status or residence in urban or rural surroundings affects the incidence. Etiology In over 97% of cases, coronary disease is due to arteriosclerosis and its sequelae. Other less common causes are syphilis of the aorta, in which the coronary ostia are involved and narrowed or occluded; periarteritis nodosa; rheumatic fever; and pressure on a coronary vessel from without, as for example by intrapericardial aneurysm, a tumor or amyloid deposits in the heart muscle. Embolism of a coronary artery is rare; it is seen occasionally when fragments from vegetations break off from the mitral or aortic valve in bacterial endocarditis or when a bit of calcium is dislodged from an atheromatous ulcer in a sclerotic aorta. Fat embolism from bone has been described.[Editorial note: Sub-sections on Pathology, Abnormal Physiology, Clinical Manifestations, Evolution of lesions, and Diagnosis have been omitted in the interest of brevity] Treatment The therapy in cases of coronary heart disease resolves itself into two parts: (1) management of the patient subject to paroxysms of anginal pain; (2) treatment of the patient who has suffered an attack of coronary occlusion with infarction of the myocardium. These therapeutic regimens are well known, so that for this discussion they may be presented in outline form. Measures which are of accepted value will be given first, those of doubtful or as yet unproven benefit will be indicated in a second list. Others generally regarded as without value will be mentioned last. The Anginal Syndrome Current Practice Nitrites, especially nitroglycerine.Reduction of weight by diet, in the obese.Low sodium diet, in congestive failure.Digitalis or quinidine when indicated.Regulation of activity and rest.Modification in occupational habits.Curtailment or elimination of smoking.Removal of gallbladder containing stones, if attacks of colic are recurrent.Surgical operation for intractable pain. (Sympathectomy, paravertebral alcohol block). Of Doubtful Value Diet low in fats, especially cholesterol.Use of drugs to lower cholesterol levels in the blood and lessen likelihood of deposition in the arteries (choline, inositol).Use of drugs to lower blood pressure when elevated.Vasodilators, given by mouth (aminophyllin, thesodate, khellin).Alcoholic beverages.Spa therapy.Psychotherapy alone.Present surgical operations designed to promote the development of a collateral circulation in the heart. Of No Value Organ and tissue extracts, given by mouth or parenterally.Vitamin E.Testosterone.Papaverine.Sympatholytic drugs, such as tetraethylammonium chloride.Irradiation of the adrenal glands.Total thyroidectomy.Propylthiouracil, radioactive iodine and other measures employed to reduce thyroid activity when this is not increased. Cardiac Infarction Current Practice Rest in bed.Opiates and sedatives, as needed.Low caloric diet.Low sodium diet digitalis and mercurial diuretics, for congestive failure.Oxygen, when indicated.Anticoagulants (heparin and Dicumarol).Regulation of activity, rest and occupational habits after healing has taken place. Of Doubtful Value Atropine and aminophyllin, in the acute phases.Intravenous injections of blood or plasma for the type of shock seen in this condition. Of No Value Conclusion It is clear that the core of the problem of coronary heart disease lies in learning the causes and mechanisms responsible for the lesions of arteriosclerosis. It is only by such understanding that an approach to preventive measures can be attempted. Thrombosis and embolism are directly concerned, both in initiating the chain of events and in causing grave, and sometimes fatal complications. Hypertension may likewise play a contributing role in aggravating the clinical course. Inasmuch as all of these conditions are being discussed by other members of this panel, they will not be considered further here.It is possible that drugs capable of exerting a prolonged dilator effect on the coronary arteries may be found. Effective surgical procedure to produce a collateral circulation in the heart conceivably may be developed. Diet may prove to be a basic factor in prevention. Syphilis can be reduced to insignificant proportions in its role as a cause of aortitis and ostial stenosis by materially lowering its incidence and treating it thoroughly in its early stages.Current practice, by means of better diagnosis and improved therapeutic procedure, has lessened disability and lowered mortality. Yet coronary heart disease, striking predominantly during the years of greatest productivity, remains one of the chief scourges of our day. The search for its fundamental causes must be pursued diligently so that prevention may become a reality.

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