Fişa suspiciunii de plagiat / Sheet of plagiarism's suspicion	Indexat la: 0139/06

Opera suspicionată (OS) Suspicious work	Opera autentică (OA) Authentic work

OS	MOŞ, Liana; ZORILĂ, Corina; COTORACI, Coralia; GREC, Veronica; ALEXA, Ioana; WIENER, A; and MARIAN, Alin. Cytokine and atherogenesis. Jurnal Medical Aradean (Arad Medical Journal). 2009; 12(1). p.50-55.
OA	DAUGHERTY, A.; WEBB, N.R.; RATERI, D.L.; and KING, V.L. Cytokine regulation of macrophage functions in atherogenesis. *Journal of Lipid Research.* Vol. 46, 2005. p.1812 – 1822.

Incidența minimă a suspiciunii / Minimum incidence of suspicion	
Abstract.p.50:01 – Abstract:p.50:05	Abstract.p.1812:07 - Abstract.p.1812:11
Abstract.p.50:05 – Abstract:p.50:10	p.1816:06s-p.1816:10s
p.53:01s-p.53:05s	p.1814:14s-p.1814:p.18
p.53:05-p.53:02d	p.1815:10d-p.1815:25d
Fişa întocmită pentru includerea suspiciunii în Indexul Operelor Plagiate în România de la Sheet drawn up for including the suspicion in the Index of Plagiarized Works in Romania at www.plagiate.ro	

Notă: p.72:00 semnifică textul de la pag.72 până la finele paginii.

Notes: p.72:00 means the text of page 72 till the end of the page.

Anul XII
Nr. 1
Martie 2009

Jurnal medical arădean

JURNAL MEDICAL ARĂDEAN

CUPRINS

Cytokine and atherogenesis

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Rezumat

Numarul mare de citokine care au fost identificate in procesul de ateroscleroza, impreuna cu numarul mare de receptori de la nivelul macrofagelor, constituie importanti participanti in modificarile lezionale din cadrul aterosclerozei. Combinatia citokinelor prezente in leziuniel aterosclerotice cu receptorii de la nivelul macrofagelor determina interactiunea citokine-macrofage care are rol important in dezvoltarea lezionala aterosclerotica.

Abstract

The numerous cytokines that have been detected in atherosclerosis, combined with the expression of large numbers of cytokine receptors on macrophages, are consistent with this axis being an important contributor to lesion development. The combination of the many cytokines present in atherosclerotic lesions and the abundant cytokine receptors on macrophages is consistent with an important role of cytokine-macrophage interactions in lesion development.

Atherosclerosis is a lifelong disease in which the process of development of an initial lesion to an advanced raised lesion can take decades. According to international statistics, heart disease is the primary cause of morbidity and mortality across all ethnicities and genders. Hypertension, hypercholesterolemia, and diabetes are increasing at alarming rates and many individuals remain undiagnosed and untreated.

Risk factors lead to an environment in which the three principal oxidative systems in the vascular wall are activated: xanthine oxidases, NADH/NAD(P)H, and uncoupled e-NOS.

Inflammatory response is generalized and can be triggered by microbial invaders, mechanical stress, chemical stress, oxidative stress, other.

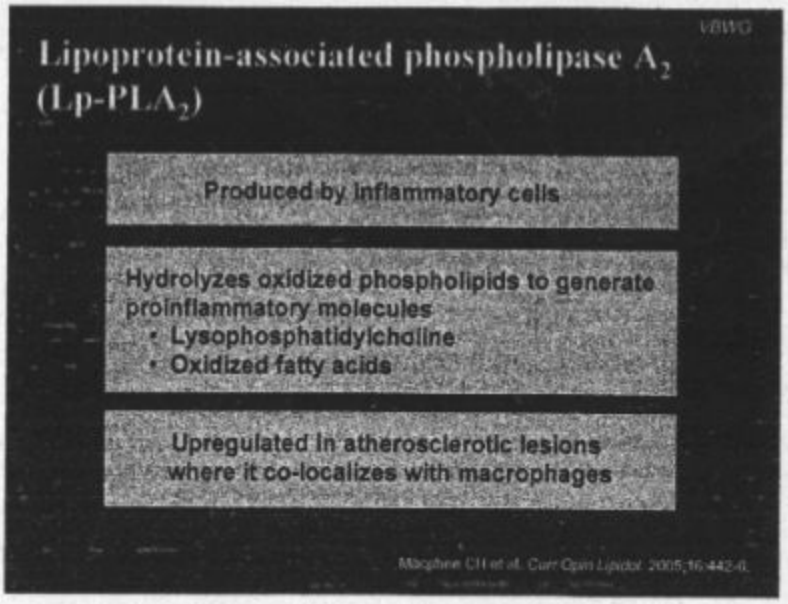
Inflammatory response includes four basic phenomena: changes in vascular tone of blood vessels, increased oxygen utilization by cells facilitating the response, changes in blood vessel walls (short term: inc. capillary permeability; long term: smooth muscle proliferation), changes in coagulation.

Origination of free radicals/ ROS is absorption of extreme energy sources, ultraviolet light, x-rays, endogenous (oxidative) reactions, enzymatic metabolism of exogenous chemical or drugs.

Atherogenesis can be related to an inflammatory response to endothelial damage:

- Inflammatory/Immune response
- Endothelium
- Cytokines
- Functions of "Good" Cholesterol
- Renin Angiotensin Aldosterone System (RAAS)

Tabel 3.



One of the most prominent changes in macrophages after entry into the sub endothelial space of developing atherosclerotic lesions is the engorgement of these cells with lipid. There have been numerous studies to determine the role of specific cytokines in the development of atherosclerosis.

As described above, one cytokine that has been studied extensively in cell culture studies is IFN-alfa, which is also one of the more extensively investigated cytokines in in vivo studies of atherogenesis.

Studies with cultured cells have demonstrated many effects of IFN-alfa on the intracellular accumulation of lipids in macrophages. These findings lead to the notion that IFN-alfa would retard atherosclerosis, especially by minimizing intracellular lipid accumulation in macrophages. In contrast, the effects of IFN- alfa on the development of atherosclerosis in mouse models of the disease have been quite consistent, but they have contradicted the original concept of IFN-alfa being anti-atherogenic.

HDL has anti-inflammatory, a oxidative, anti-aggregatory, anti-coagul and pro-fibrinolytic role.

HDL Inhibits chemotaxis monocytes, adhesion of leukocyt endothelial dysfunction, apoptosis, L Oxidation, complement activation, plate activation and Factor X activation.

HDL promotes endothelial repair/regeneration, smooth mus proliferation, synthesis of prostacyc synthesis of naturietic peptide, activat of Protein C and Protein S.

Insults to endothelium increa production of AGEs - advanc glycosylation endproducts, react oxygen species, hyperinsulinem hypertension, activated the rtesp[onses T-Cells/Lymphocytes, small dense LDL

Smoking causes intimal inju promotes oxidation, promo inflammatory response in respiratory tra enhances platelet aggregation, promo vasoconstriction

Diabetes mellitus increa production of AGEs. hyperglycem induces inflammatory response, frequen

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