## Fişa suspiciunii de plagiat / Sheet of plagiarism's suspicion

Indexat la: 0140/06

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

OS	MOŞ, Liana; ZORILĂ, Corina; COTORACI, Coralia; GREC, Veronica; ALEXA,	
	Ioana Dana; WIENER, A; MARIAN, Alin. Cytokine and atherogenesis. Jurnal	
	Medical Aradean (Arad Medical Journal). 2009; 12(1). p.50-55. (număr retras	
	pe pagina web <u>www.jmedar.ro</u> dar existent în formă tipărită).	

VBWG. New Frontiers in CVD Risk Management: Optimizing Outcomes in Patients with Multiple Cardiovascular Risks - PowerPoint PPT. File VBWG06-COREMR.PPT. Vascular Biology Working Group (VBWG), Univ.of Florida. 2006. Available at: www . powershow . com / view2b / 48f6ac – MTMwZ / New \_ Frontiers \_ in \_ CVD \_ Risk \_ Management \_ Optimizing \_ Outcomes \_ in \_ Patients \_ with \_ Multiple \_ Cardiovascular \_ Risks \_ powerpoint \_ ppt \_ presentation. Accesed: 23 January 2015.

Incidența minimă a suspiciunii / Minimum incidence of suspicion	
p.50:04s-p.50:11s	slide.02:02-slide.02:04
p.50:01s-p.50:04s	slide.03:01-slide.03:02
p.50:12s-p.50:03d	slide.03:03-slide.03:05
p.51:10d-p.52:03d	slide.03:07-slide.03:17
p.51:01s-p.51:06d	slide.15:02-slide.10:10

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 <a href="https://www.plagiate.ro">www.plagiate.ro</a>

Sursă: integru.org.

Notă: p.72:00 semnifică textul de la

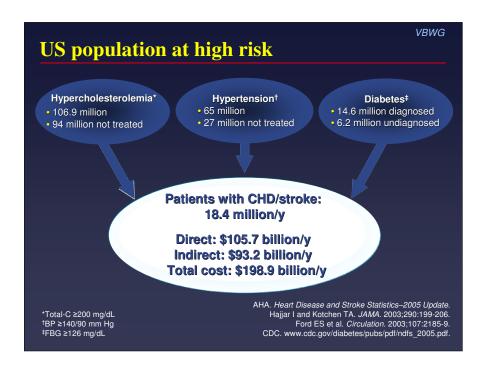
pag.72 până la finele paginii.

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

the end of the page.

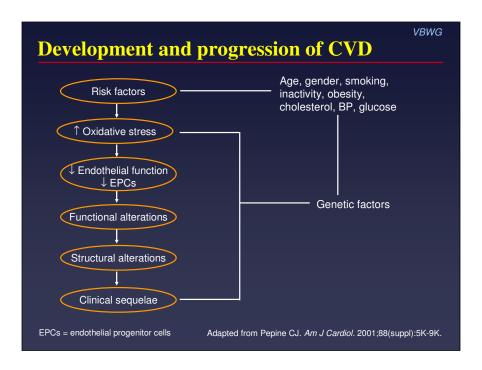
VBWG

New Frontiers in CVD Risk Management: Optimizing Outcomes in Patients with Multiple Cardiovascular Risks



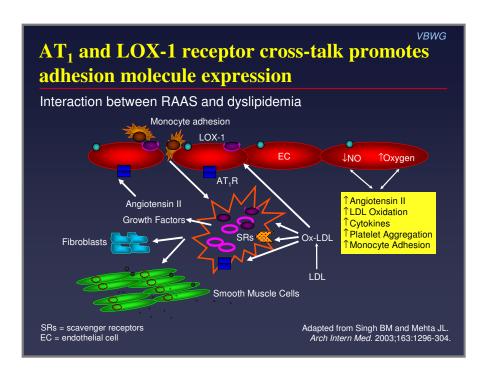
## Heart disease in the US is a national epidemic resulting in staggering costs

- According to national 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.



## Pathophysiology of vascular disease

- Atherosclerosis is a lifelong disease in which the process of development of an initial lesion to an advanced raised lesion can take decades.
- Risk factors lead to an environment in which the three principal oxidative systems in the vascular wall are activated: xanthine oxidase, NADH/NAD(P)H, and uncoupled eNOS.
- Excessive production of reactive oxygen species overwhelms endogenous antioxidant mechanisms, leading to oxidation of lipoproteins, nucleic acids, carbohydrates, and proteins. The principal target of this oxidative stress is the vascular endothelium, although there may be other targets.
- Among the functional alterations induced by reactive oxygen species are impairment of endothelium-dependent vasorelaxation (following a reduction in nitric oxide bioavailability), increase in inflammatory mediators, and development of a procoagulant vascular surface.
- Ultimately structural alterations occur, including plaque growth, vascular wall remodeling, decreased fibrinolysis, vascular smooth muscle cell proliferation and migration, and other structural alterations. They can lead to the clinical sequelae of death, MI, stroke, ischemia, and congestive heart failure (CHF).



RAAS activation has implications for ox-LDL and BP

- Formation of oxidized LDL (ox-LDL) is a key step in the pathogenesis of atherosclerosis. The ox-LDL receptor (LOX-1) is present mostly on the surface of endothelial cells, vascular smooth muscle cells, macrophages, and platelets. LOX-1-mediated ingestion of ox-LDL activates mitogen-activated protein kinases (MAPKs) in the cell, which in turn activate nuclear factor-κB (NF-κB), a transcriptional factor involved in expression of monocyte chemoattractant protein-1 (MCP-1). In turn, MCP-1 leads to adhesion molecule expression.
- Ang II, via the AT<sub>1</sub> receptor, increases LOX-1 expression. Conversely, ox-LDL, via LOX-1, upregulates the AT<sub>1</sub> receptor.