Cracking the Cardio Code pt III

A Biogenetix Clinical Presentation

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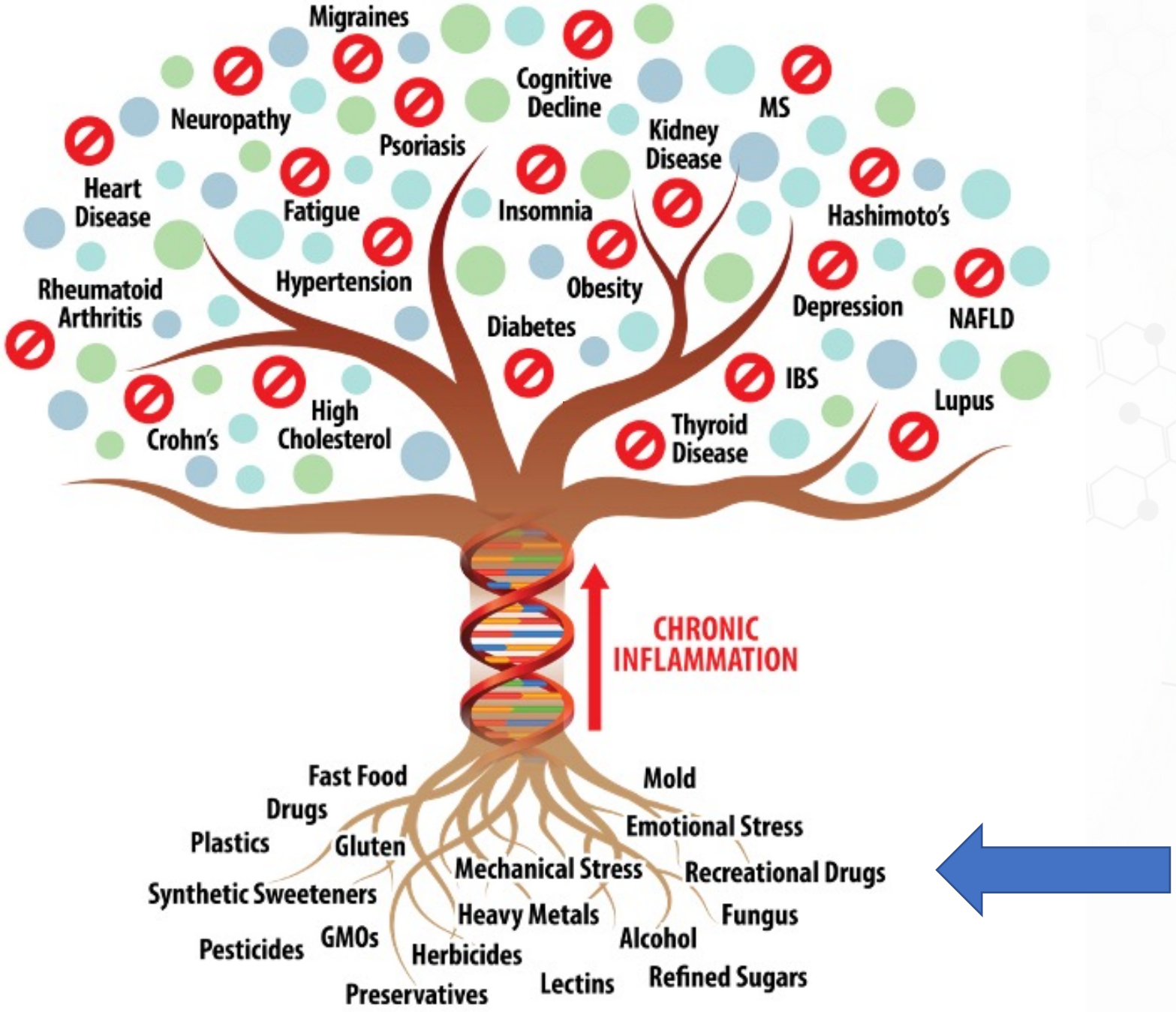
Disclaimer

- *Information in this presentation is not intended to diagnose, treat, reverse, cure, or prevent any disease. While this presentation is based on medical literature, findings, and text, The following statements have not been evaluated by the FDA.*
- *The information provided in this presentation is for your consideration only as a practicing health care provider. Ultimately you are responsible for exercising professional judgment in the care of your own patients.*

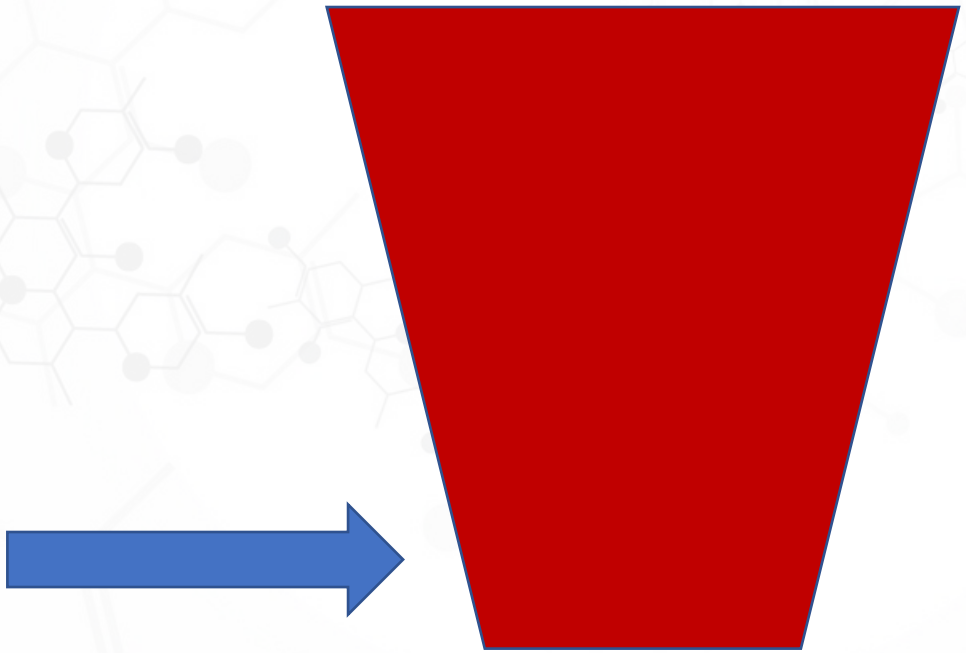

Migraines
Neuropathy
Psoriasis
Cognitive Decline
MS
Kidney Disease
Heart Disease
Fatigue
Insomnia
Hashimoto's
Rheumatoid Arthritis
Hypertension
Obesity
Depression
NAFLD
Diabetes
IBS
Crohn's
High Cholesterol
Lupus
Thyroid Disease
CHRONIC INFLAMMATION
Fast Food
Mold
Drugs
Emotional Stress
Plastics
Gluten
Mechanical Stress
Recreational Drugs
Synthetic Sweeteners
Heavy Metals
Fungus
Pesticides
GMOs
Alcohol
Herbicides
Refined Sugars
Lectins
Preservatives

My doctor says it’s genetic…nothin’ you can do.

As a clinician…

- What are we trying to accomplish?
- What variables is the patient willing to control?
- What's already been wrecked or compromised?
- What's the literature say?
    1. Ancestry
    2. Comorbidities
    3. Regional concerns
- Then put together a way forward...

The Bucket Theory

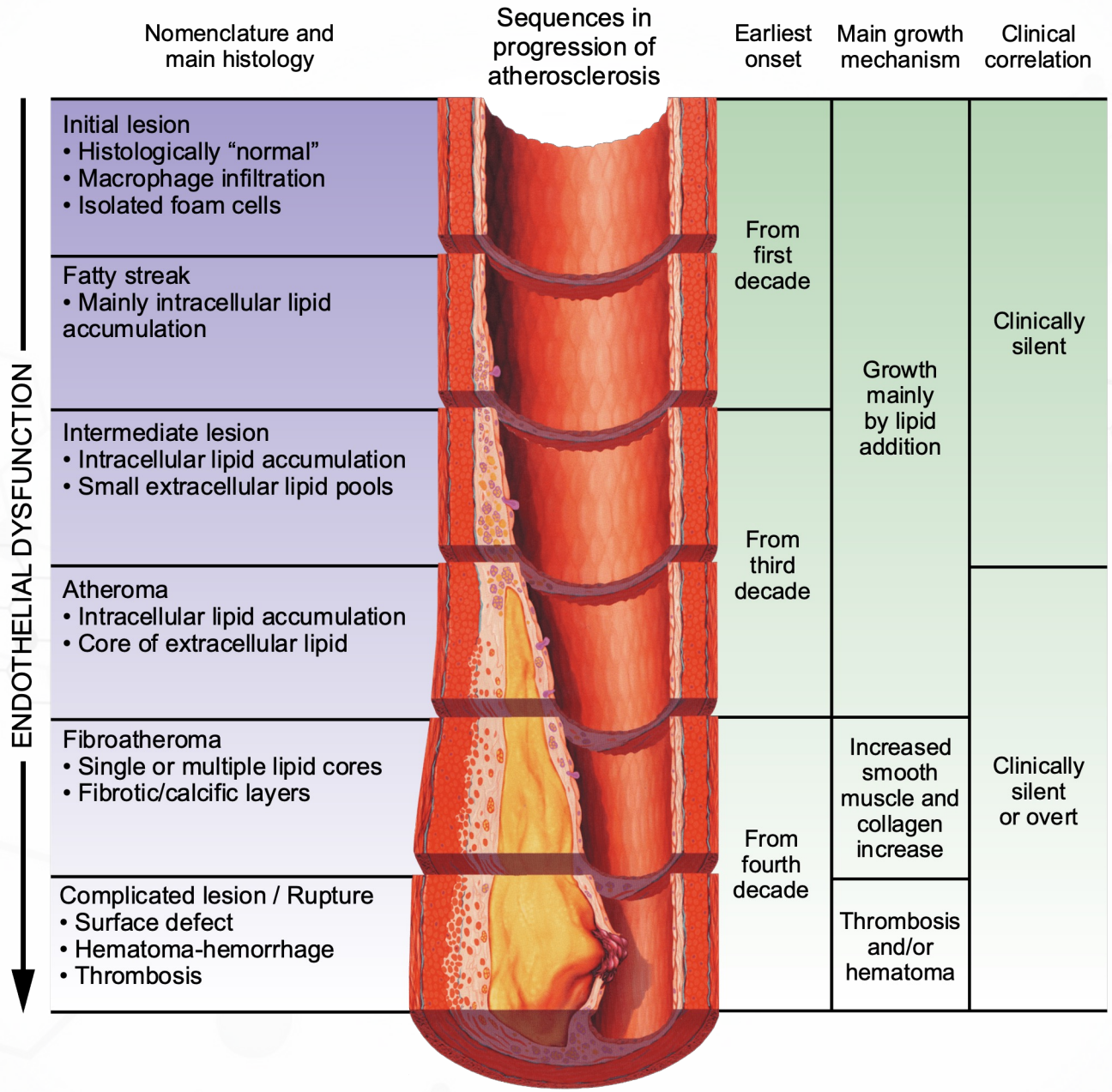


Ordered Items

NMR LipoProfile; Venipuncture

TESTS	RESULT	FLAG	UNITS	REFERENCE INTERVAL	LAB
NMR LipoProfile					
LDL Particle Number					01
LDL-P [A]	**3058**	**High**	nmol/L	<1000	01
			Low	**< 1000**	
			Moderate	**1000 - 1299**	
			Borderline-High	**1300 - 1599**	
			High	**1600 - 2000**	
			Very High	**> 2000**	
Lipids					01
LDL-C [A]	**212**	**High**	mg/dL	0 - 99	01
			Optimal	**< 100**	
			Above optimal	**100 - 129**	
			Borderline	**130 - 159**	
			High	**160 - 189**	
			Very high	**> 189**	
Comment: LDL-C is inaccurate if patient is non-fasting.					01
HDL-C [A]	52		mg/dL	>39	01
Triglycerides [A]	**200**	**High**	mg/dL	0 - 149	01
Cholesterol, Total [A]	**304**	**High**	mg/dL	100 - 199	01
LDL and HDL Particles					01
HDL-P (Total) [A]	**27.6**	**Low**	umol/L	>=30.5	01
Small LDL-P [A]	**1628**	**High**	nmol/L	<=527	01
LDL Size [A]	20.7		nm	>20.5	01

68 yo male, DM2, CVD, ED.




Omega 3
CoQ10
Nitric Oxide
Nattokinase
Cardiovascular Disease
Resveratrol
Folate
Bergamot
Hesperidin

Front Cardiovasc Med. 2022; 9: 964977.
Published online 2022 Aug 22. doi: 10.3389/fcvm.2022.964977
PMCID: PMC9441630
PMID: 36072877

Effective management of atherosclerosis progress and hyperlipidemia with nattokinase: A clinical study with 1,062 participants

Hongjie Chen,[1] Jiepeng Chen,[2] Fuping Zhang,[3] Yuanhui Li,[4] Ronghua Wang,[2] Qiang Zheng,[2] Xu Zhang,[2] Jun Zeng,[2] Feng Xu,[5,*] and Yiguang Lin[1,6,7,*]

Nattokinase (NK), known as a potent fibrinolytic and antithrombotic agent, has been shown to have antiatherosclerotic and lipid-lowering effects. However, data on human clinical studies are limited. In this clinical study involving 1,062 participants, our objective was to examine the efficacy of NK in atherosclerosis and hyperlipidemia and safety at the dose of 10,800 FU/day after 12 months of oral administration. Various factors, including lower doses that influence NK pharmacological actions, were also investigated. We found that NK at a dose of 10,800 FU/day effectively managed the progression of atherosclerosis and hyperlipidemia with a significant improvement in the lipid profile. A significant reduction in the thickness of the carotid artery intima-media and the size of the carotid plaque was observed. The improvement rates ranged from 66.5 to 95.4%. NK was found to be ineffective in lowering lipids and suppressing atherosclerosis progression at a dose of 3,600 FU/day. The lipid-lowering effect of NK was more prominent in subjects who smoked, drank alcohol, and subjects with higher BMI. Regular exercise further improved the effects of NK. Co-administration of vitamin K2 and aspirin with NK produced a synergetic effect. No noticeable adverse effects associated with the use of NK were recorded. In conclusion, our data demonstrate that atherosclerosis progression and hyperlipidemia can be effectively managed with NK at a dose of 10,800 FU/day. The lower dose of 3,600 FU per day is ineffective. The dose of 10,800 FU/day is safe and well tolerated. Some lifestyle factors and the coadministration of vitamin K2 and aspirin lead to improved outcomes in the use of NK. Our findings provide clinical evidence on the effective dose of NK in the management of cardiovascular disease and challenge the recommended dose of 2,000 FU per day.

Int J Mol Sci. 2019 Feb; 20(4): 904.
Published online 2019 Feb 19. doi: 10.3390/ijms20040904
PMCID: PMC6413130
PMID: 30791450

The Effects of Resveratrol in Patients with Cardiovascular Disease and Heart Failure: A Narrative Review

Garrison J. B. Dyck,[1] Pema Raj,[2] Shelley Zieroth,[2] Jason R. B. Dyck,[3] and Justin A. Ezekowitz[1,*]

In contrast to existing pharmacotherapies that largely focus on correcting neurohumoral factors that are altered in CVD and HF [14], alternative pharmacotherapies using nutraceuticals that directly target additional factors in CVD and HF progression are being considered for the prevention and treatment of these two conditions [19,20,21]. Indeed, certain natural compounds have been shown in preclinical studies to target the underlying causes of CVD and HF such as oxidative stress [22], inflammation [23], poor endothelial function [24], and even poor left ventricle function [25]. Therefore, these nutraceuticals may potentially target aspects of CVD and HF progression missed by, or not effectively treated with, existing pharmacotherapies. In addition, these nutraceuticals are not only being considered for independent use, but as supplements to other pre-existing HF therapies as well. While early phase trials are using these natural compounds, the goal is also to provide evidence that synthetic analogs can be made from these natural compounds in order to increase the efficacy of the compound [26]. One of these natural compounds considered to potentially help treat HF and CVD and prevent their development is resveratrol. The objective of this review is to describe the evidence of the clinical utility of resveratrol on CVD and HF treatment.

Int J Mol Sci. 201

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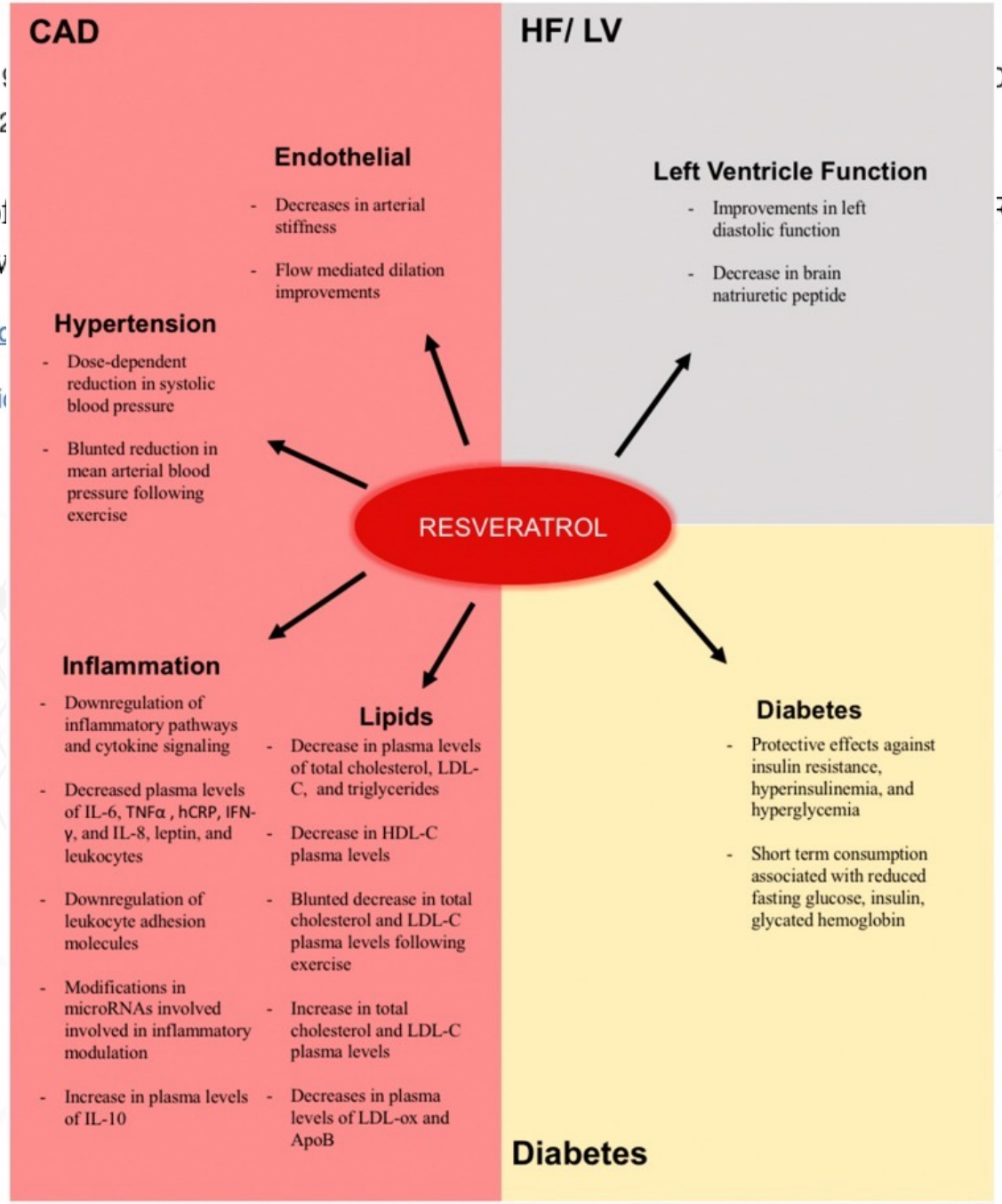

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The link between vascular inflammation and risk of CVD, most notably hypertension and atherosclerosis, is well documented [72]. In atherosclerosis, the beginning stages of the development of an atherosclerotic lesion are characterized by endothelial cells beginning to express selective adhesion molecules such as vascular cell adhesion molecule-1 (VCAM-1) that promote attachment of leukocytes to the endothelium [41]. This activity is the most pronounced in damaged areas of the endothelium with disturbed flow and a low production of nitric oxide (NO) [73]. In addition, the smooth muscle cells (SMCs) in these damaged areas may produce proteoglycans that attach to lipoproteins, promote their oxidation, and increase the adhesion of leukocytes to the lesions of the arterial walls [74]. The chemically attracted leukocytes, including lymphocytes [75] and monocytes [76], then enter the intima and stimulate a local inflammatory response [41]. Stimulation factors also causes monocytes to develop into macrophage foam cells [77] and inflammatory cytokines released by T-cells promote the smooth muscle cells of the endothelium [78] to eventually form a thick extracellular matrix of SMCs and fibrin [79]. Given this role of inflammation in atherosclerosis, pro-inflammatory cytokines are often used as biomarkers to monitor changes in atherosclerosis risk and consequently the prognosis of HF after supplementation of a potential cardio-protective drug [41]. Common biomarkers related to the inflammatory response measured in resveratrol clinical trials include interleukin (IL)-6, tumor necrosis factor (TNF)α, c-reactive protein (CRP), Intercellular Adhesion Molecule 1 (ICAM-1), P selectin, and E selectin [41]. Other inflammatory cytokines also involved with cardiac diseases used in resveratrol RCTs to assess risk of atherosclerosis and HF include IL-8; mostly as a marker of negative effects) and IL-10 (anti-inflammatory and a marker of positive effects) [80].

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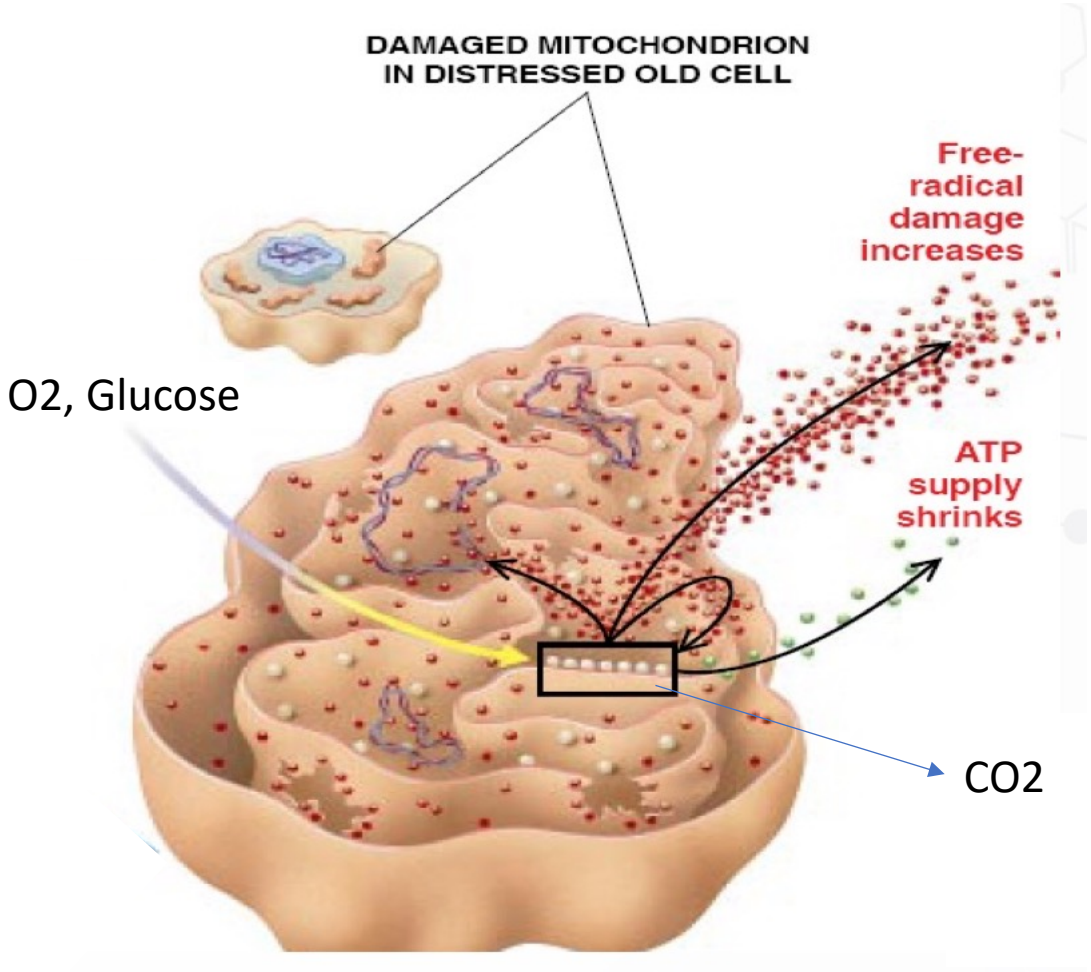
Garrison J. B. Dyck,[1] Pema Raj,[2] Shelley Zieroth,[2] Jason R. B. Dyck,[3] and Justin A. Ezekowitz[1,*]

Since it is likely that resveratrol may have beneficial effects in numerous CVDs that can contribute to HF and/or are comorbidities of HF, it stands to reason that resveratrol may hold promise for the treatment of clinical HF. Interestingly, numerous animal models of ischemic and non-ischemic HF have shown beneficial effects of resveratrol in HF that either prolongs survival [38], improves diastolic [38] or systolic function [150], reduces negative atrial and left ventricular remodeling [38,151,152], improves hemodynamics and cardiac energetics [153] and/or improves exercise capacity [154]. However, despite these preclinical studies, it is still unknown if resveratrol can improve HF in humans. That said, in a double-blind, placebo-controlled trial involving patients with stable coronary artery disease receiving 10 mg of resveratrol/day for 3 months, resveratrol improved left ventricle diastolic function [95]. Moreover, 20 mg of resveratrol/day administered for 60 days resulted in a significant decrease in b-type natriuretic peptide (BNP) in patients with angina pectoris, suggesting improved left ventricle function [64]. Although limited, these studies suggest that resveratrol may have a direct impact on myocardial function in humans. While this does not demonstrate that resveratrol will improve myocardial performance in patients with HF, it does provide interesting data that suggests that clinical trials in the area are warranted.

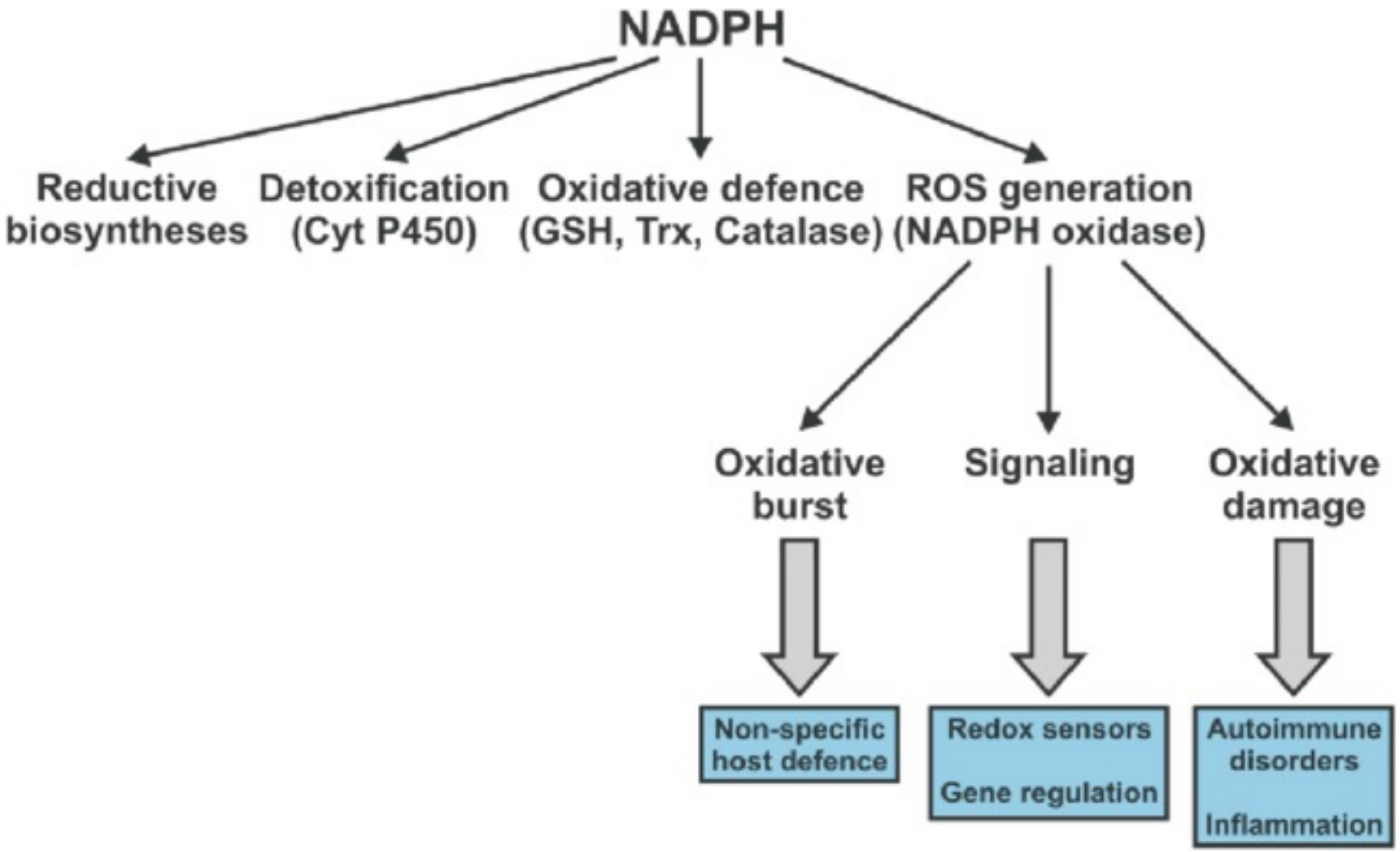

DAMAGED MITOCHONDRION
IN DISTRESSED OLD CELL
Free-
radical
damage
increases
ATP
supply
shrinks
O2, Glucose
CO2

NADPH and NOX

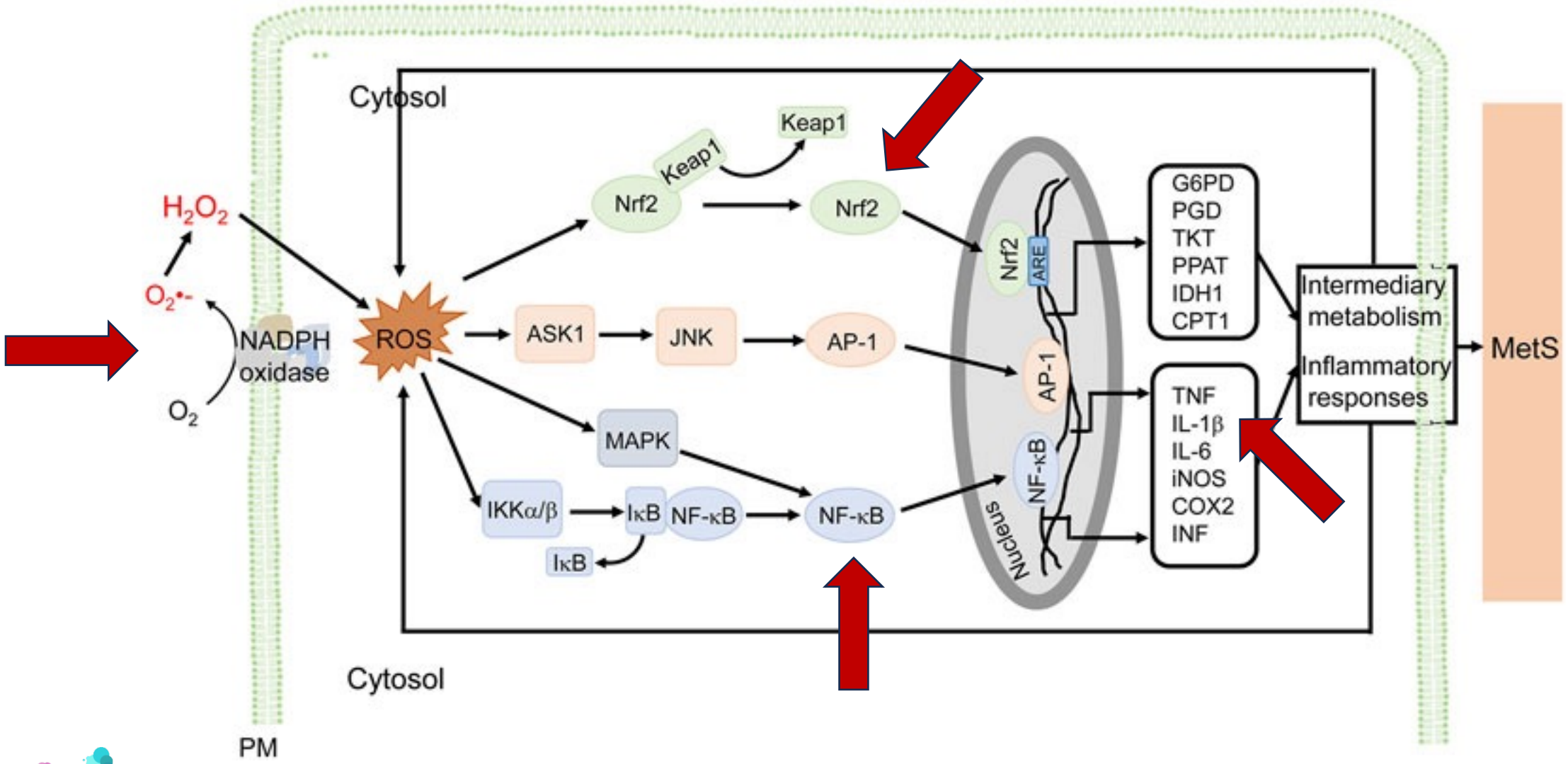
NADPH: Nicotinamide adenine dinucleotide phosphate is a vital electron donor found in all organisms that plays a role in many biological processes. Low levels expose a person to metabolic syndrome and further metabolic disease windup.

NADPH oxidase: (NOX) is a family of membrane-bound enzymes that generate reactive oxygen species (ROS). NOX is located in intracellular organelles and the plasma membrane, and it's also found in the membranes of phagosomes used by white blood cells to engulf microorganisms (including biotoxins). The enzyme complex is made up of membrane and cytosolic components that communicate during a host's response to stimuli like bacterial and viral infections.

OR?



https://www.researchgate.net/publication/41668746/figure/fig2/AS:667871755640848@1536244443982/Functions-of-NADPH-The-major-function-of-NADPH-is-to-donate-electrons-to-reduce-oxidized.png


Cytosol
Keap1
Keap1
Nrf2
Nrf2
H_2O_2
$O_2^{\bullet-}$
NADPH oxidase
O_2
ROS
ASK1
JNK
AP-1
MAPK
IKKα/β
IκB
NF-κB
NF-κB
IκB
Nrf2
ARE
AP-1
NF-κB
Nucleus
G6PD
PGD
TKT
PPAT
IDH1
CPT1
TNF
IL-1β
IL-6
iNOS
COX2
INF
Intermediary metabolism
Inflammatory responses
MetS
Cytosol
PM

Br J Pharmacol. 2017 Jun; 174(12): 1633–1646.
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PMCID: PMC5446570
PMID: 27058985

Antioxidant effects of resveratrol in the cardiovascular system

Ning Xia, [1] Andreas Daiber, [2] Ulrich Förstermann, [1] and Huige Li [1]

The antioxidant effects of resveratrol (3,5,4′-trihydroxy-*trans*-stilbene) contribute substantially to the health benefits of this compound. Resveratrol has been shown to be a scavenger of a number of free radicals. However, the direct scavenging activities of resveratrol are relatively poor. The antioxidant properties of resveratrol *in vivo* are more likely to be attributable to its effect as a gene regulator. Resveratrol inhibits NADPH oxidase-mediated production of ROS by down-regulating the expression and activity of the oxidase. This polyphenolic compound reduces mitochondrial superoxide generation by stimulating mitochondria biogenesis. Resveratrol prevents superoxide production from uncoupled endothelial nitric oxide synthase by up-regulating the tetrahydrobiopterin-synthesizing enzyme GTP cyclohydrolase I. In addition, resveratrol increases the expression of various antioxidant enzymes. Some of the gene-regulating effects of resveratrol are mediated by the histone/protein deacetylase sirtuin 1 or by the nuclear factor-E2-related factor-2. In this review article, we have also summarized the cardiovascular effects of resveratrol observed in clinical trials.

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Published online 2021 Sep 21. doi: 10.3390/ijms221810152
PMCID: PMC8466271
PMID: 34576315

The Effect of Resveratrol on the Cardiovascular System from Molecular Mechanisms to Clinical Results

Roland

Mathias

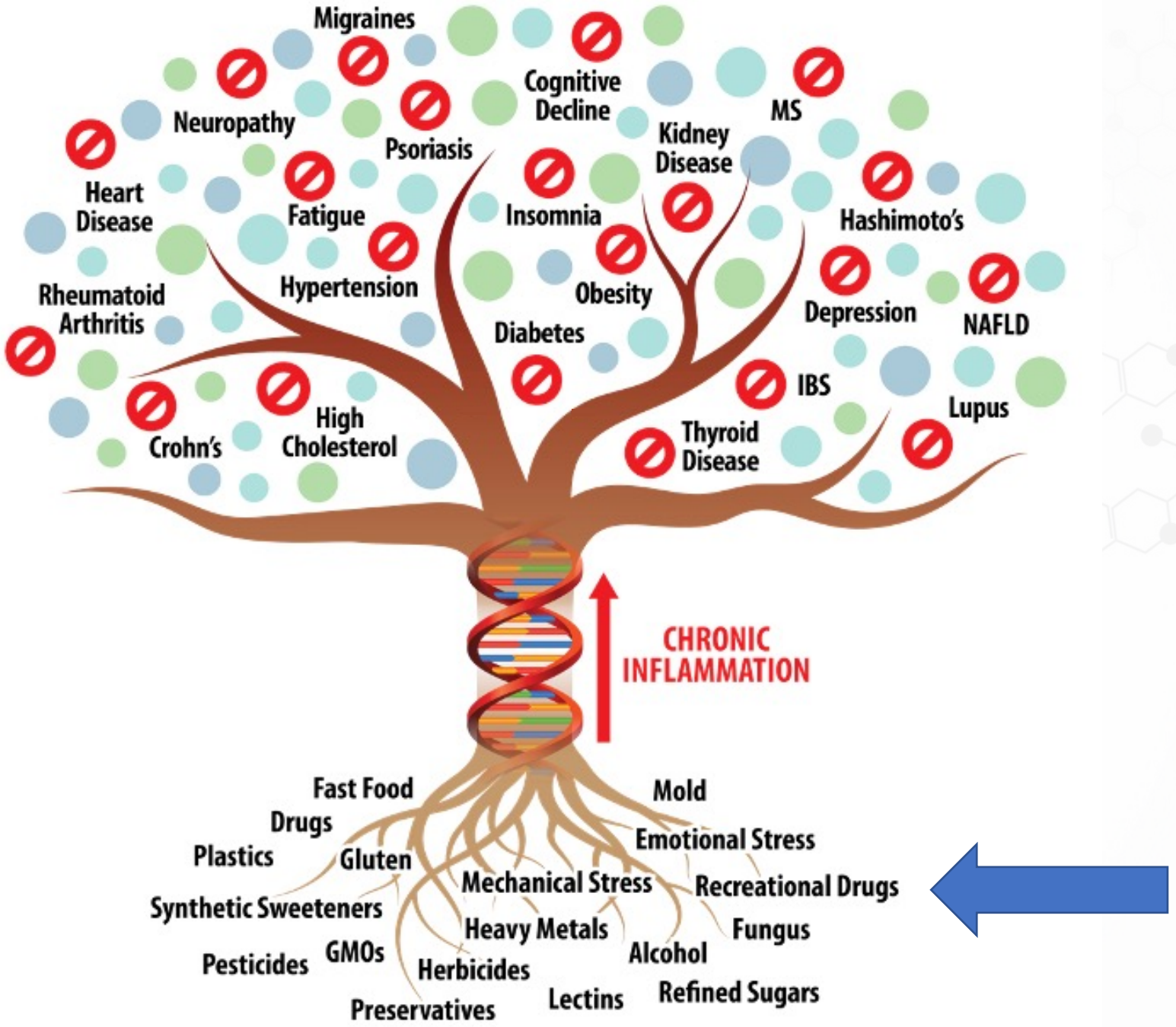
9. Future Perspectives on Treatment with Resveratrol in COVID-19 Associated with CVDs

Go to:

Based on the results of recent years, CVDs seem to be associated with higher morbidity and mortality in patients with COVID-19 infection. Myocardial and vascular injury in chronic cardiovascular diseases are strongly associated with SARS-CoV-2 infection, possibly due to the overactivation of inflammatory processes, namely due to hypercytokinemia ("cytokine storm") triggered by viral infection [181]. Systemic inflammation and hypercoagulability results in myocardial damage, with consequent CV complications including myocarditis, myocardial infarction, heart failure, arrythmias, or venous thromboembolism (deep vein thrombosis or pulmonary embolism) [182,183,184,185]. RES has shown a high antiviral potential that can be explored in both human and animal viral infections. The antiviral mechanisms and effects of RES (inhibition of viral protein synthesis and some gene expressions) have been widely studied in various viruses, including coronavirus [186,187]. Moreover, RES can influence several mechanisms associated with CVD related to COVID-19, including upregulation of the expression of angiotensin-converting enzyme 2 (ACE2) [188]. The downregulation of ACE2 protein by SARS-CoV-2 is leading to dysfunction of the renin-angiotensin-aldosterone-system (RAAS), with overproduction of pro-inflammatory and pro-oxidant agents [189]. Furthermore, as a potent anti-inflammatory and antiviral agent, RES can inhibit the overactivation of pro-inflammatory responses as well as enhance the antiviral immune system (macrophages, cytotoxic T lymphocytes, and natural killer immune cells) [187].

In addition, based on its anti-thrombotic effects, RES can reduce the risk of vascular thrombotic events in the course of COVID-19 infection [190].

Although no preclinical or human clinical trials with RES have been published to date in COVID-19, the aforementioned mechanisms of action of RES suggest that it may contribute to the prevention of cardiovascular toxicity in COVID-19. Thus, in the future, RES may be an adjunctive agent in the treatment of SARS-CoV-2 infection.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