Cracking the Cardio Code pt II

A Biogenetix Clinical Presentation

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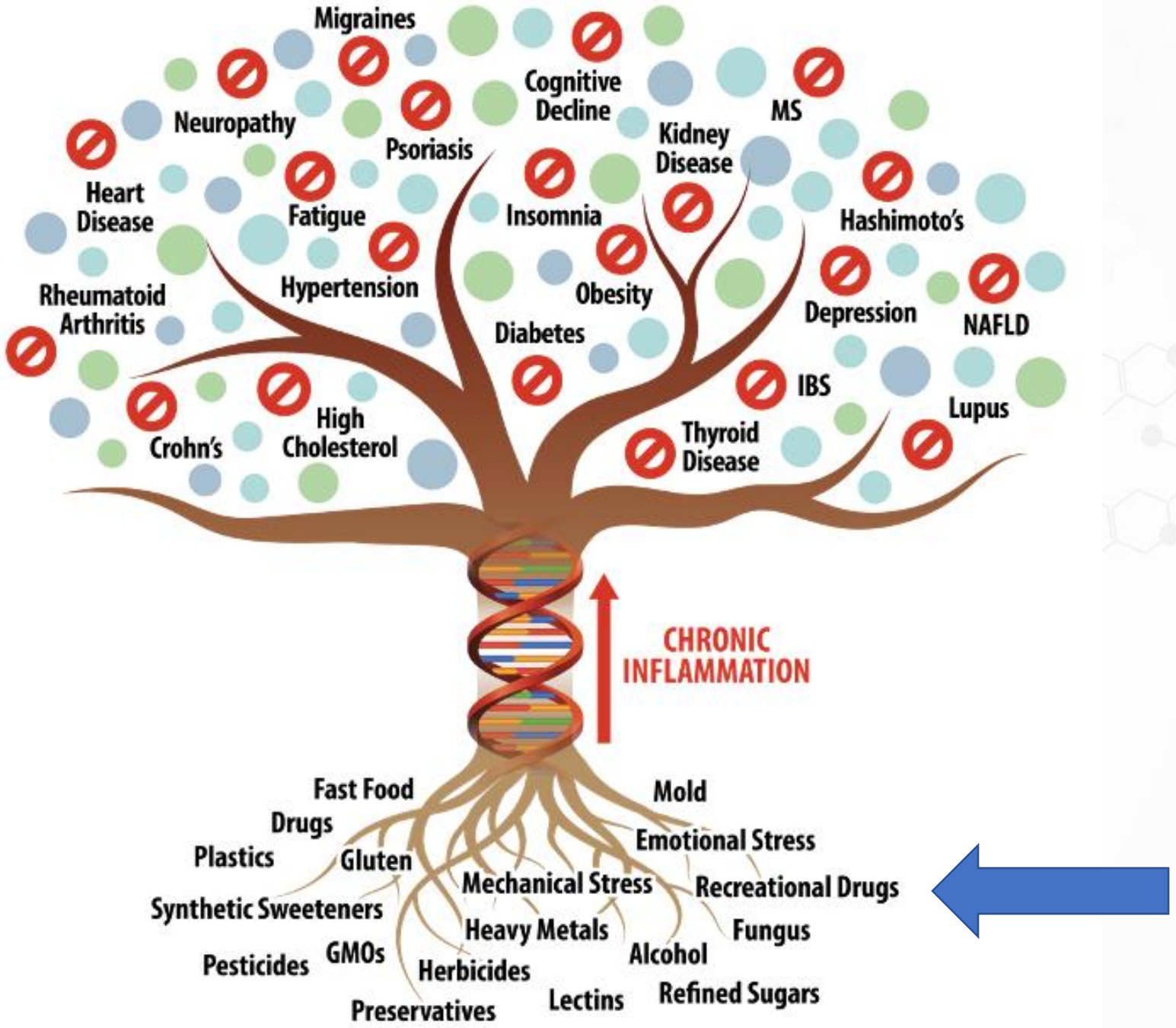
Disclaimer

- *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.*

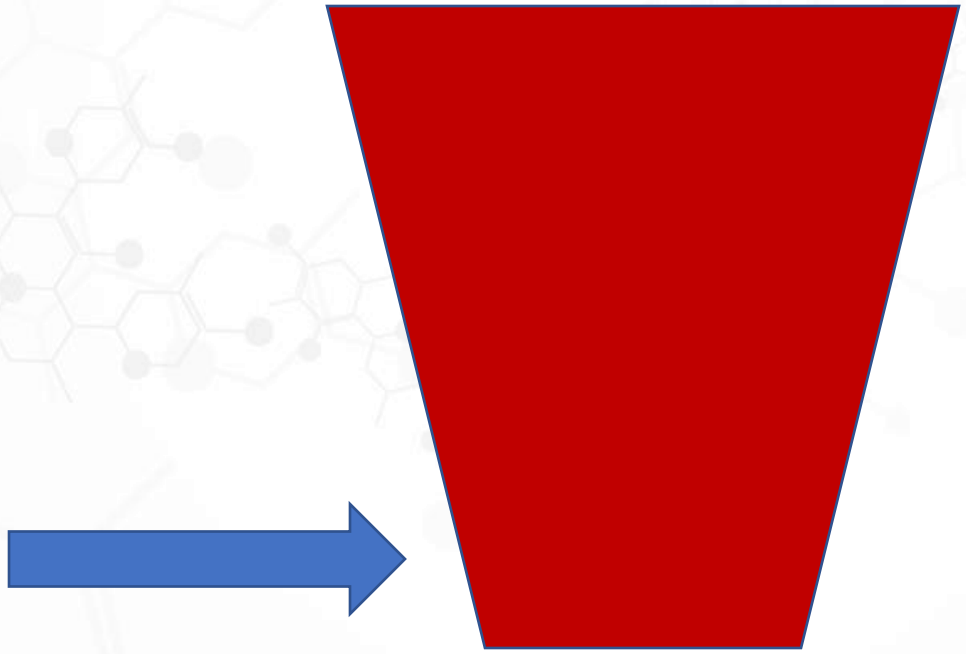

Migraines
Neuropathy
Psoriasis
Cognitive Decline
MS
Kidney Disease
Heart Disease
Fatigue
Insomnia
Hashimoto's
Hypertension
Obesity
Depression
NAFLD
Rheumatoid Arthritis
Diabetes
IBS
Lupus
Crohn's
High Cholesterol
Thyroid Disease
CHRONIC INFLAMMATION
Fast Food
Mold
Drugs
Emotional Stress
Plastics
Gluten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



LipoFraction NMR
(and LipoFraction NMR with Lipids)

CPT Code **83704, 80061 (with Lipids)***
Order Code **37847 – LipoFraction NMR**
37849 – LipoFraction NMR with Lipids†
Specimen Type **Serum**
Tube Type **Red-Top (without Gel Barrier)**

Evaluation of lipoprotein particles has been used to support management of cardiovascular disease (CVD) risk for over 15 years, and lipoprotein subclass analysis has become a valuable tool to help clinicians better stratify patients at risk.

In situations where LDL-C or HDL-C levels determined as part of a conventional lipid panel are optimal, additional LDL-C and HDL-C subclass analysis may identify patients with increased CVD risk.1,2

Enhanced identification of these previously unidentified at-risk patients can help physicians incorporate treatment that can help reduce atherosclerotic CVD and significantly reduce cardiovascular events.3,4

The LipoFraction NMR test utilizes the most up-to-date nuclear magnetic resonance technology to measure lipoprotein particles.

https://www.clevelandheartlab.com/wp-content/uploads/2020/04/CHL-D040-SEPT2019-NMR-Practitioner-One-Pager.pdf

The NMR Lipoprofile includes measurements for:

- LDL Particle Number.
- LDL Cholesterol. (volume)
- HDL Cholesterol. (volume)
- Triglycerides.
- Total Cholesterol.
- HDL Particle Number.
- Small LDL Particle Number.
- LDL Size.

*ApoA, ApoB, Lipoprotein (a)

Apolipoprotein A

- ApoA-I is the major protein component of HDL and plays essential roles in the biogenesis and functions of HDL.
- Approximately 70% of the HDL protein mass is comprised of ApoA-I, with another 15–20% comprised of ApoA-II.

https://www.sciencedirect.com/topics/medicine-and-dentistry/apolipoprotein-a#:~:text=ApoA%2DI%20is%20the%20major,been%20found%20in%20heterozygous%20subjects.

National Library of Medicine
National Center for Biotechnology Information

Utility of Advanced Lipoprotein Testing in Clinical Practice

Kenneth R. Feingold, MD.

Author Information and Affiliations

Last Update: January 3, 2023.

APOLIPOPROTEIN B

Go to:

All of the pro-atherogenic lipoproteins (chylomicron remnants, VLDL remnants, IDL, LDL, and Lp(a)) carry one apolipoprotein B on their surface such that apolipoprotein B levels reflect the total number of atherogenic particles (76). Most of the circulating apolipoprotein B is associated with LDL particles (76). However, the contribution of very high Lp(a) levels to total Apo B levels can be substantial (Estimated Apo B in LDL/VLDL = Apo B mg/dl – (Lp(a) mg/dl x 0.16) (77). Apo B levels measured in the non-fasting state are similar to fasting values.

The levels of apolipoprotein B, LDL-C, and non-HDL-C are strongly correlated. Almost all studies have shown that apolipoprotein B levels are more closely associated with ASCVD than LDL-C levels and the general consensus is that apolipoprotein B levels are a more accurate predictor of ASCVD events than LDL-C (41,42,65,78-85). Apolipoprotein B levels are equivalent to non-HDL-C levels in predicting ASCVD but when these measurements are discordant apolipoprotein B levels are a more accurate predictor of ASCVD.

Lipoprotein (a) (Lp(a))

Lp(a) is an LDL-like lipoprotein with an apoB bound to an apo(a) protein. 20-25% of the global population has an elevated Lp(a), which increases the risk of atherosclerotic cardiovascular disease, independent of other traditional risk factors. Elevated Lp(a) precipitates vascular inflammation, atherosclerosis, calcification, and blood clotting.

https://www.rupahealth.com/post/a-functional-medicine-protocol-for-coronary-artery-disease

Ordered Items

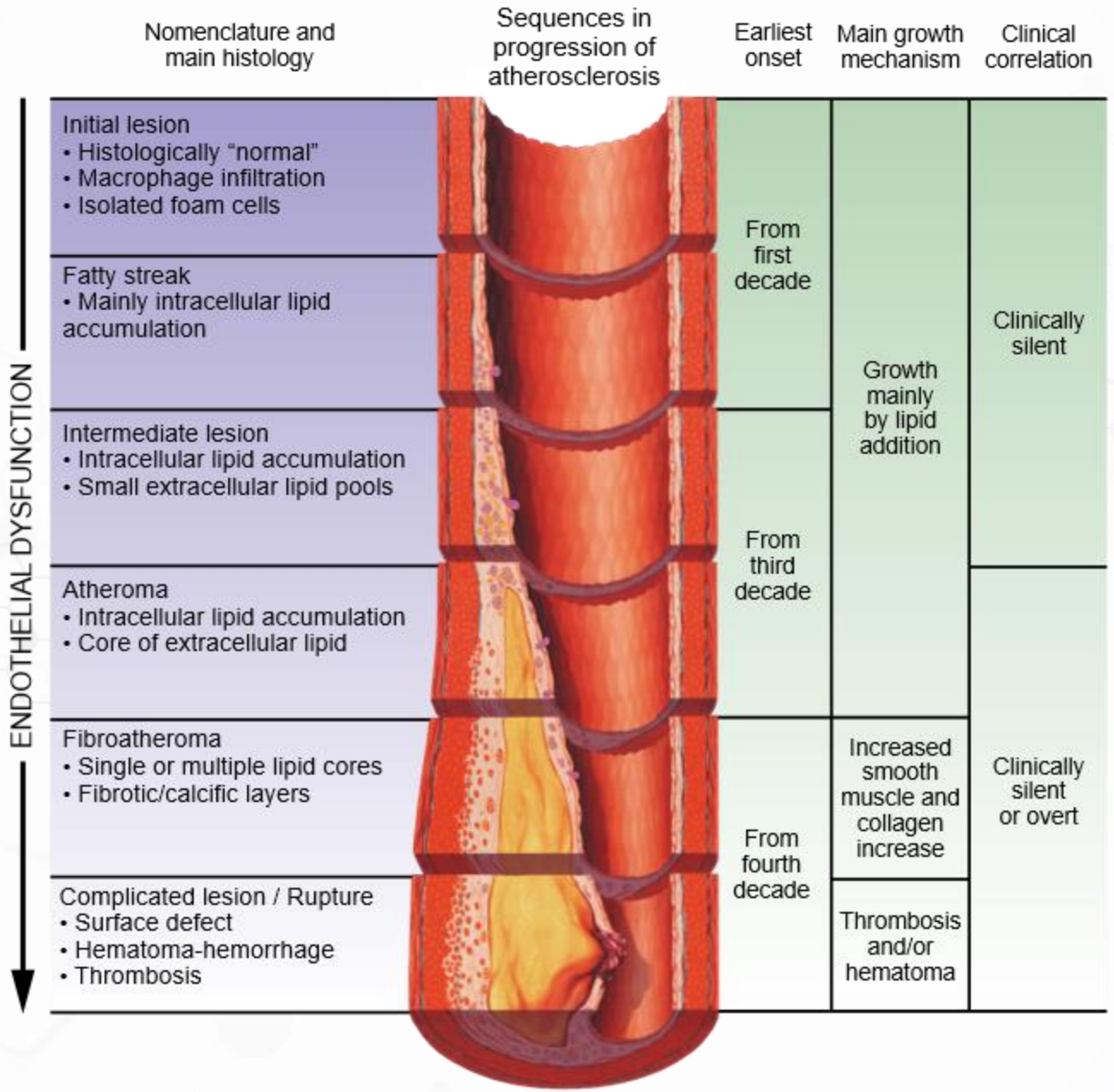
NMR LipoProfile; Venipuncture

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.

LIPIDS							
Cholesterol, Total	110		<200	N/A	≥200	mg/dL	
Direct LDL Cholesterol	39		<100	100-129	≥130	mg/dL	
HDL Cholesterol	55		≥40	N/A	<40	mg/dL	
Triglycerides	131		<150	150-199	≥200	mg/dL	
Lipoprotein Fractionation, NMR							
LDL-P[15]	596		<935	935-1816	>1816	nmol/L	817
Small LDL-P	265		<467	467-820	>820	nmol/L	<154
LDL Size	20.8		>20.5	N/A	≤20.5	nm	21.1
HDL-P	38.5		>32.8	29.2-32.8	<29.2	umol/L	35.4
Large HDL-P		5.9	>7.2	5.3-7.2	<5.3	umol/L	10.9
HDL Size		8.9	>9.0	8.7-9.0	<8.7	nm	9.5
Large VLDL-P	<1.5		<3.7	3.7-6.1	>6.1	nmol/L	<1.5
VLDL Size	46.8		<47.1	47.1-49.0	>49.0	nm	39.7
Apolipoproteins							
Apolipoprotein A1	154		≥115	N/A	<115	mg/dL	168
Apolipoprotein B	46		<90	90-119	≥120	mg/dL	
ApoB/ApoA1 Ratio	0.30		<0.77	0.77-0.95	>0.95		
Lipoprotein (a)		82	<75	75-125	>125	nmol/L	64

62 yo male, DM2.




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

Sci Rep. 2023; 13: 22469.
Published online 2023 Dec 18. doi: 10.1038/s41598-023-48562-y
PMCID: PMC10728071
PMID: 38110459

Natto consumption suppresses atherosclerotic plaque progression in LDL receptor-deficient mice transplanted with iRFP-expressing hematopoietic cells

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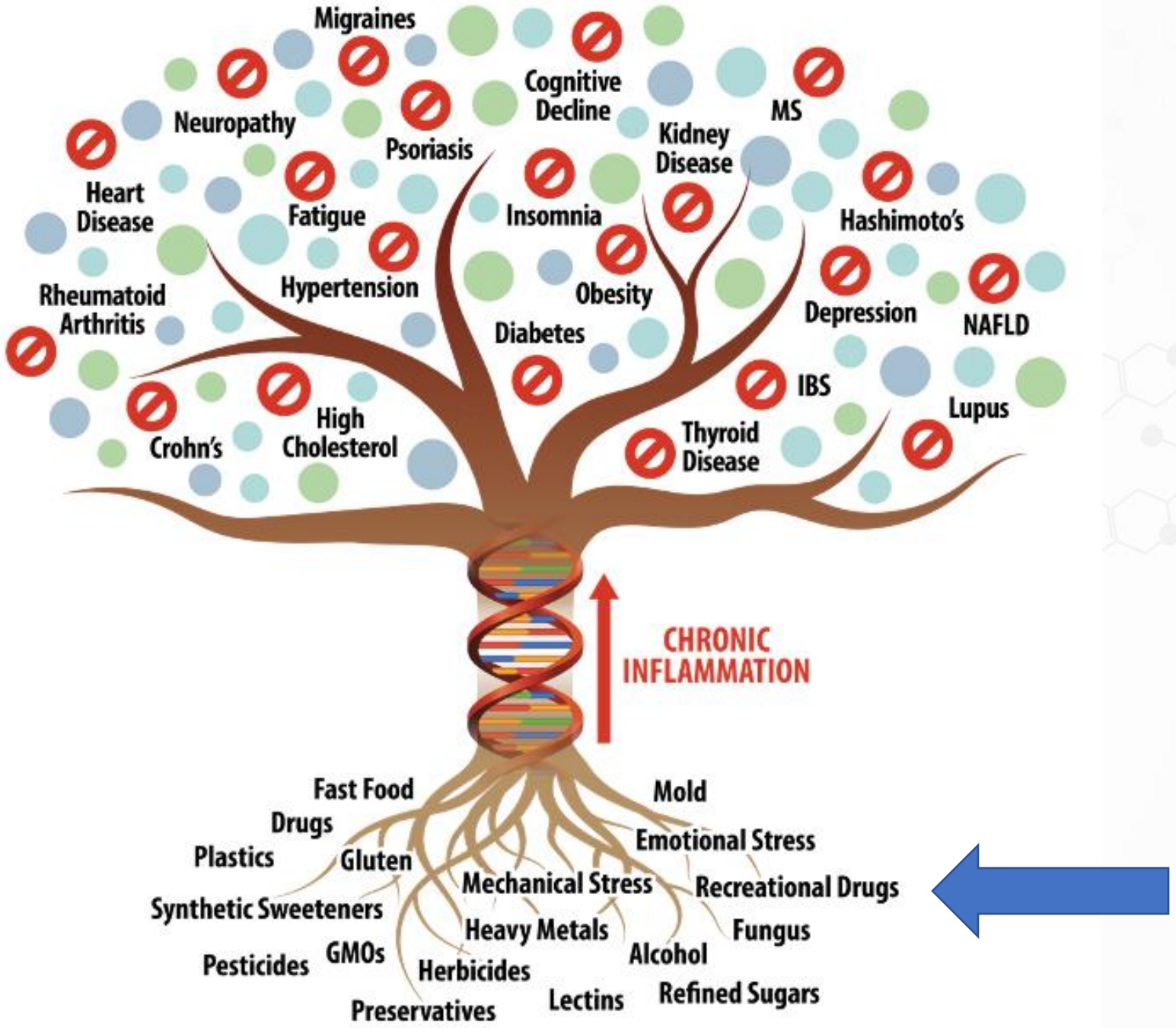
Natto, known for its high vitamin K content, has been demonstrated to suppress atherosclerosis in large-scale clinical trials through a yet-unknown mechanism. In this study, we used a previously reported mouse model, transplanting the bone marrow of mice expressing infra-red fluorescent protein (iRFP) into LDLR-deficient mice, allowing unique and non-invasive observation of foam cells expressing iRFP in atherosclerotic lesions. Using 3 natto strains, we meticulously examined the effects of varying vitamin K levels on atherosclerosis in these mice. Notably, high vitamin K natto significantly reduced aortic staining and iRFP fluorescence, indicative of decreased atherosclerosis. Furthermore, mice administered natto showed changes in gut microbiota, including an increase in natto bacteria within the cecum, and a significant reduction in serum CCL2 expression. In experiments with LPS-stimulated macrophages, adding natto decreased CCL2 expression and increased anti-inflammatory cytokine IL-10 expression. This suggests that natto inhibits atherosclerosis through suppression of intestinal inflammation and reduced CCL2 expression in macrophages.

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.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