Casual Friday Series

The Real Issue with Cholesterol

A Biogenetix Clinical Presentation

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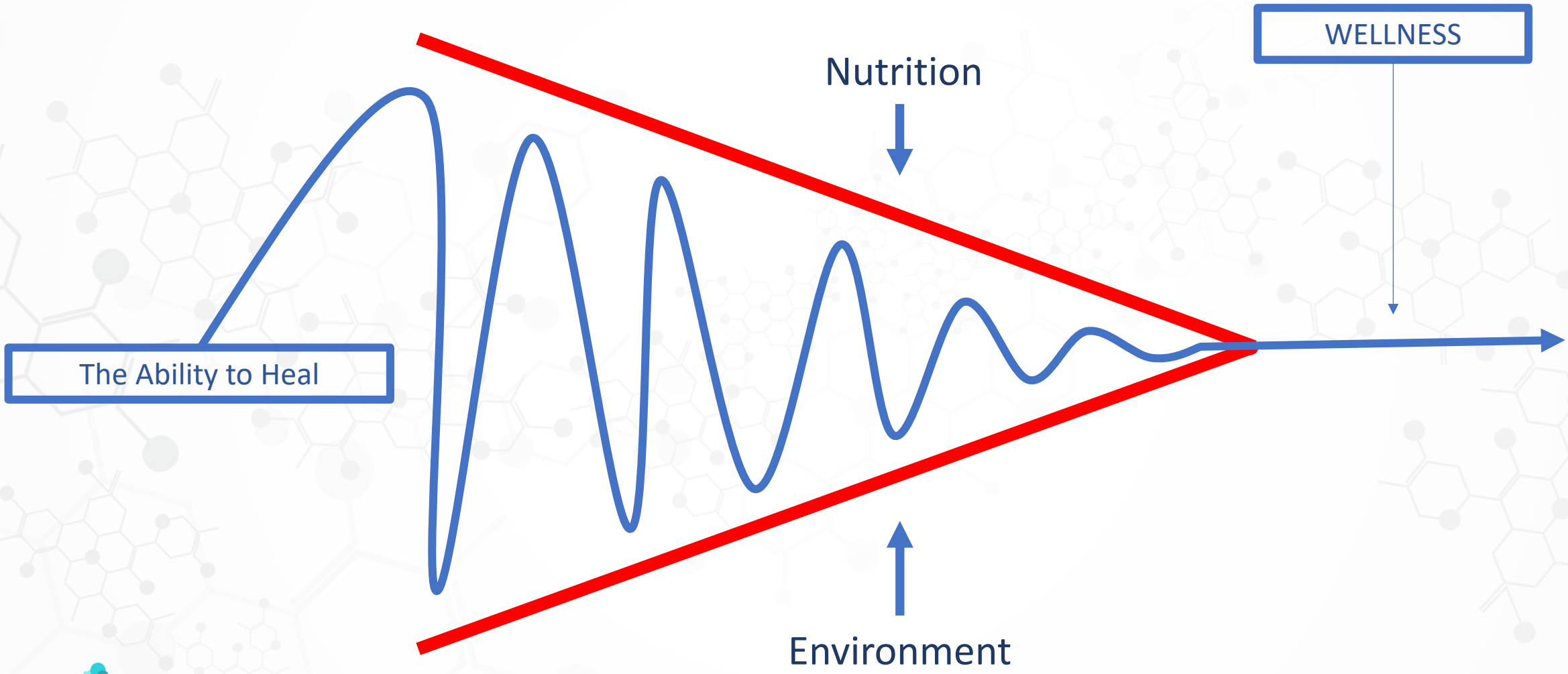
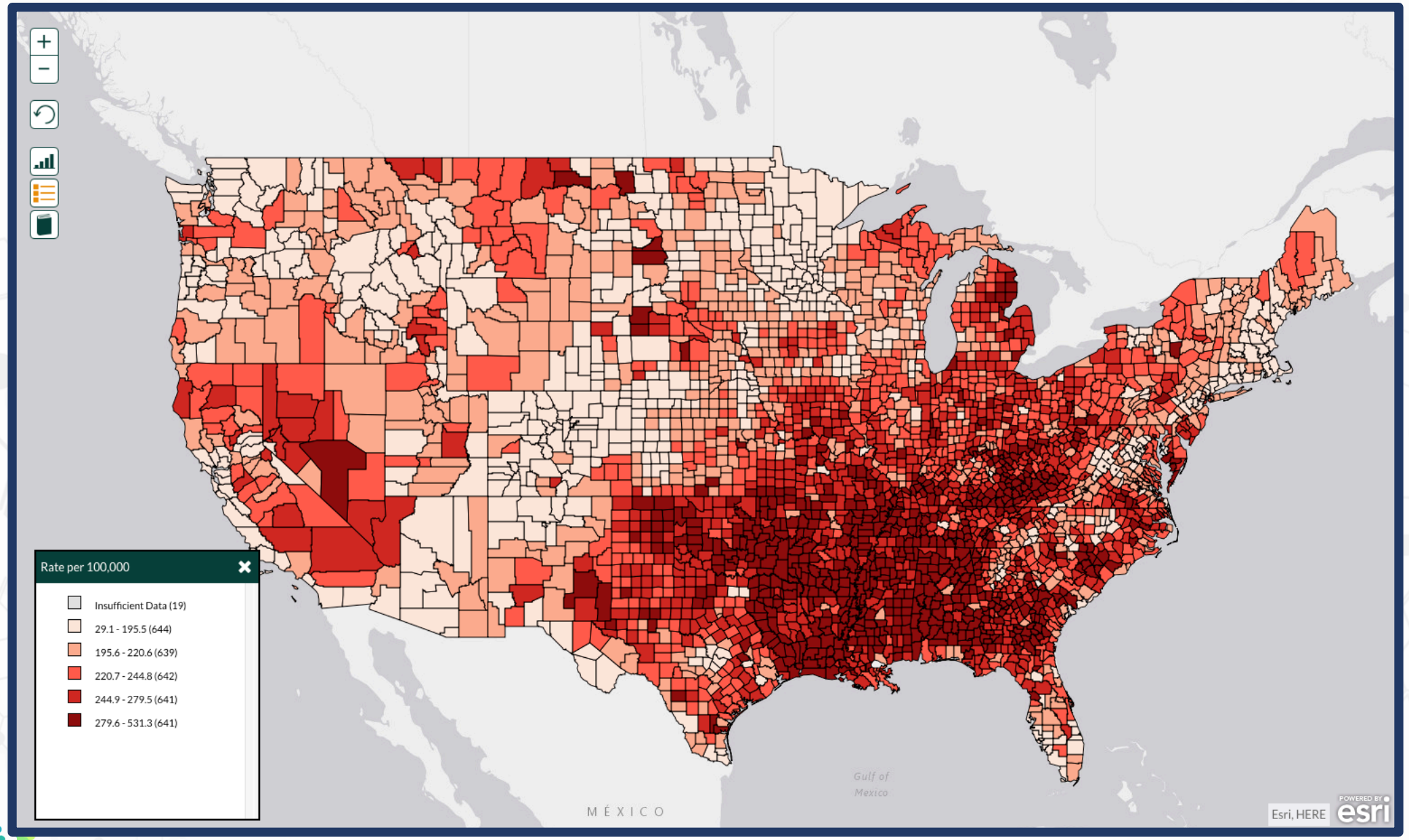
Disclaimer

- *Information in this presentation is not intended, in itself, 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.*

Lifestyle + Genetics = Chronic Health IMPROVEMENT

Protocols





https://nccd.cdc.gov/DHDSPAtlas/?state=State

NMR Lipoprofile

Clinical Significance: The Lipoprotein Fractionation NMR test is used to help assess the risk for cardiovascular disease (CVD) in patients with intermediate or high risk based on traditional or emerging risk factors, and to assess therapeutic response in patients undergoing lipid-lowering therapy, by quantification of the number and size of lipoprotein particles. The lipid panel is used, along with other test, during routine assessment to determine an individual's risk of cardiovascular disease. A lipid panel can also be used to monitor the efficacy of lifestyle interventions or medications.

https://www.clevelandheartlab.com/tests/lipoprotein-fractionation-nmr-with-lipids/

Diabetes Metab J. 2013 Jun; 37(3): 207–211.
Published online 2013 Jun 14. doi: 10.4093/dmj.2013.37.3.207
PMCID: PMC3689018
PMID: 23807924

Beneficial Effects of Omega-3 Fatty Acids on Low Density Lipoprotein Particle Size in Patients with Type 2 Diabetes Already under Statin Therapy

Myun[illegible]n,[4]
Youn[illegible]

Beyond statin therapy for reducing low density lipoprotein cholesterol (LDL-C), additional therapeutic strategies are required to achieve more optimal reduction in cardiovascular risk among diabetic patients with dyslipidemia. To evaluate the effects and the safety of combined treatment with omega-3 fatty acids

There were no significant difference in the initial (week 0) lipid profiles among the three groups. After 8 weeks of treatment, as shown in Table 1 and Fig. 1, mean LDL particle size increased in all groups, and the percentage change was significantly greater in patients taking 4 g of omega-3 fatty acid with statin than in patients receiving statin monotherapy (2.8%±3.1% vs. 2.3% ±3.6%, *P*=0.024). Significant reduction in TG level was shown after 8-week treatment in all groups. The percentage change from baseline TG level was significantly greater in O3FA4S group than in the control group (-41.0%±24.1% vs. -24.2%±31.9%, *P*=0.049). TC level was significantly reduced at 8 weeks from baseline only in O3FA4S group (-0.44% ±0.66 mg/dL, *P*=0.018), but the percentage change was not significantly different compared to the control group. In all groups, neither HDL-C nor LDL-C level showed any significant change during the study period. There were no significant differences between O3FA2S group and the control group after 8 weeks of respective treatment.

Omega-3

Supplement Facts

Serving Size: 1 Softgel
Servings Per Container: 60

	Amount Per Serving	%Daily Value
Calories	10	
Total Fat	1 g	1%†
MaxSimil® Fish Oil Concentrate	1.3 g	**
Total Omega-3 Fatty Acids	860 mg	**
EPA (eicosapentaenoic acid)	600 mg	**
DHA (docosahexaenoic acid)	260 mg	**

† Percent Daily Values are based on a 2,000 calorie diet.
** Daily Value not established.

Other Ingredients: Softgel (fish gelatin, vegetable glycerin, and purified water), GRAS enteric coating (ethylcellulose, sodium alginate, purified water, medium-chain triglycerides, oleic acid, vegetable stearic acid, and ammonium hydroxide), and mixed natural tocopherols.

Contains: Fish (anchovy and/or sardine [sources of fish oil], tilapia and/or pangasius [sources of fish gelatin]).

Manufactured using MaxSimil® fish oil. MaxSimil® is a registered trademark of Ingenutra Inc. Protected under US patents 8,119,690 and 8,198,324; Canadian patents 2672513 and 2677670.

Familial Hypercholesterolemia

The APOB gene provides instructions for making a protein called apolipoprotein B. This protein helps LDL cholesterol bind to LDL receptors on the surface of cells, particularly in the liver. Certain variants in this gene reduce the ability of LDL cholesterol to bind to its receptor, causing fewer LDL cholesterol particles to be removed from the blood.

https://you.23andme.com/reports/ghr.fh/details/

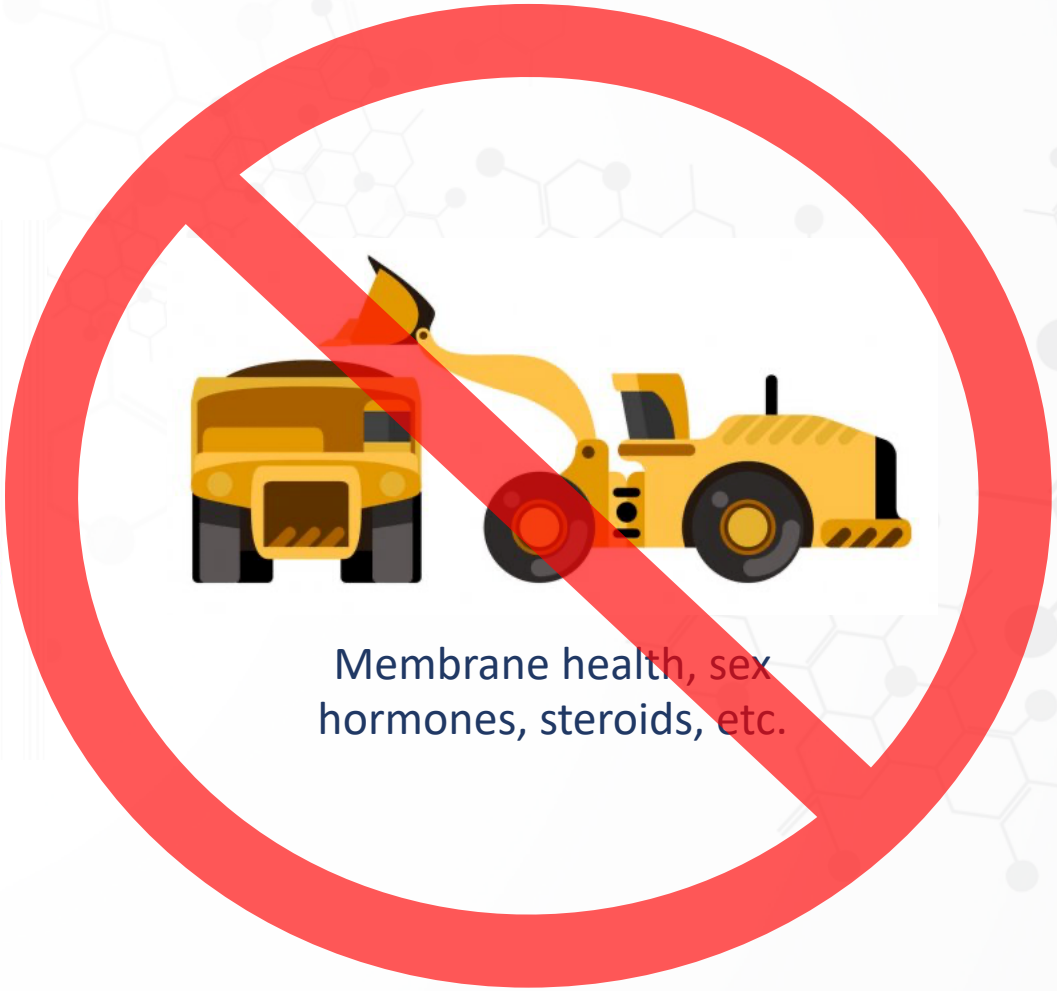
Familial Hypercholesterolemia

B-48

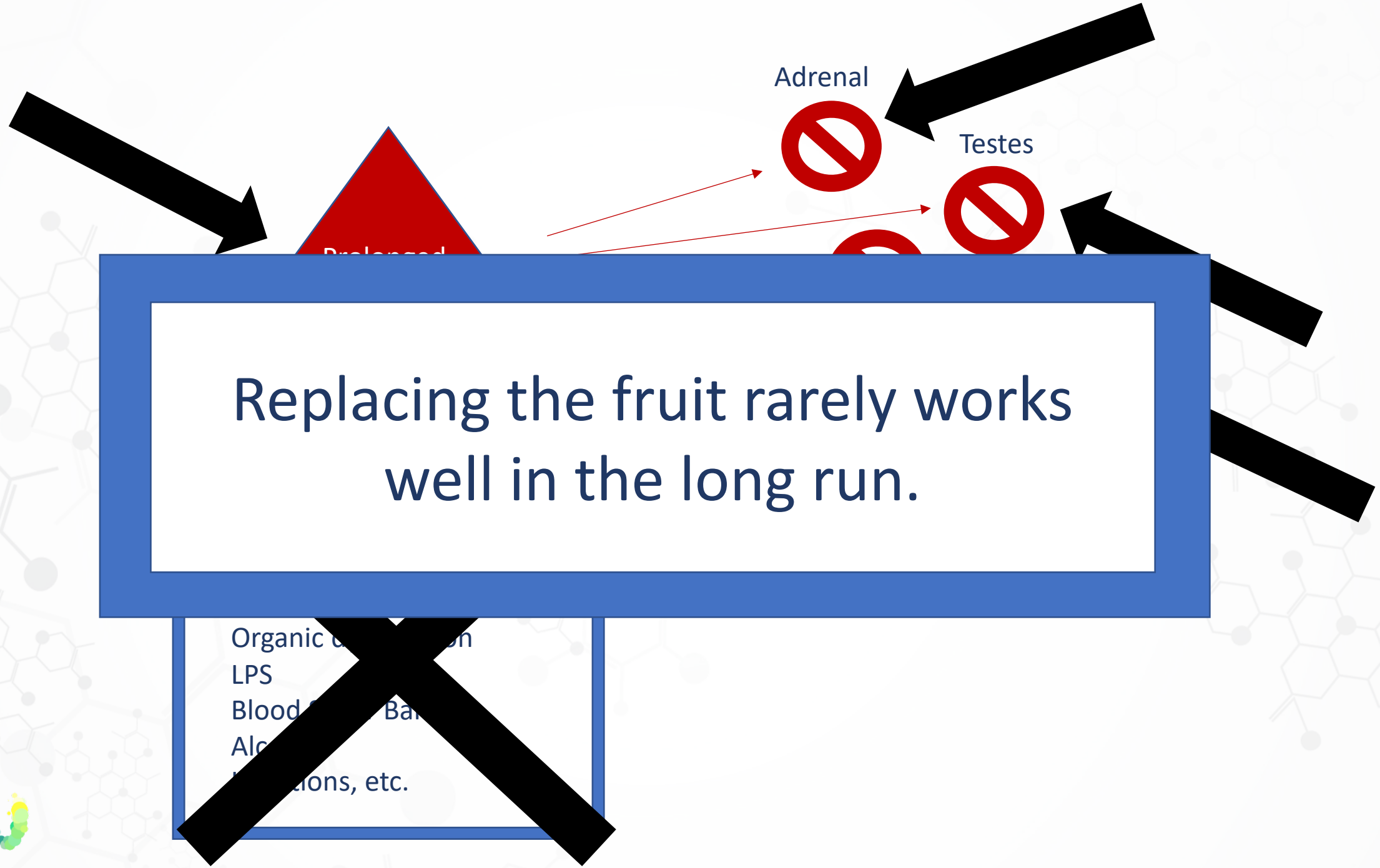
Chylomicrons, gut level.

B-100

Repackaged in the liver with B-100 to form trig rich VLDL, and chol rich LDL.



Membrane health, sex hormones, steroids, etc.


Adrenal
Testes
Replacing the fruit rarely works well in the long run.
LPS
ions, etc.

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Nat Rev Immunol. 2015 Feb; 15(2): 104–116.

doi: 10.1038/nri3793

PMCID: PMC4669071

NIHMSID: NIHMS740909

PMID: 25614320

Cholesterol, inflammation and innate immunity

Alan R. Tall[1] and Laurent Yvan-Charvet[2]

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Hypercholesterolaemia leads to cholesterol accumulation in macrophages and other immune cells, which promotes inflammatory responses, including augmentation of Toll-like receptor (TLR) signalling, inflammasome activation, and the production of monocytes and neutrophils in the bone marrow and spleen. On a cellular level, activation of TLR signalling leads to decreased cholesterol efflux, which results in further cholesterol accumulation and the amplification of inflammatory responses. Although cholesterol accumulation through the promotion of inflammatory responses probably has beneficial effects in the response to infections, it worsens diseases that are associated with chronic metabolic inflammation, including atherosclerosis and obesity. Therapeutic interventions such as increased production or infusion of high-density lipoproteins may sever the links between cholesterol accumulation and inflammation, and have beneficial effects in patients with metabolic diseases.

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doi: 10.36660/abc.20200038
PMCID: PMC7077584
PMID: 32215497

Atherosclerosis, Inflammation, and Genetics - And you Thought it Was Just LDL-cholesterol

Luis Henrique Wolff Gowdak

Arq Bras Cardiol

As the twentieth century unfolded, based on numerous epidemiological observations and intervention trials, cardiovascular risk factors were identified and targeted with the aim of decreasing cardiovascular disease burden worldwide. Along the centuries, changes in human eating patterns, a progressive decrease in physical activity, and a higher prevalence of obesity, all of which are contributing factors to the alarming rates of diabetes, hypertension, and hypercholesterolemia we see in our daily practice, have led us to assume that atherosclerosis-related disorders (myocardial infarction, ischemic cardiomyopathy, stroke, and peripheral artery disease) are an inevitable consequence of the evolutionary process we have to face in present times. However, due to advances in noninvasive imaging of the vascular system, atherosclerotic lesions in the aorta and coronary and carotid arteries happen to be found in mummies from ancient Egypt,[1] whose estimated mean age at the time of death was only 45 years.

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▸ Aut

If the so-called "classical risk factors" were less prevalent in ancient times, different, non-traditional factors must have played a significant role in the development and progression of atherosclerosis.[2] Microbial and parasitic inflammatory burdens that were likely present in ancient cultures inherently lacking modern hygiene and antimicrobials could have evoked a chronic inflammatory status. Given that patients with today's chronic systemic inflammatory diseases, including human immunodeficiency virus infection, systemic lupus erythematosus, and rheumatoid arthritis experience early-onset atherosclerosis and coronary events, is it possible that the chronic inflammatory load secondary to infection resulted in atherosclerosis in ancient times? Moreover, atherosclerosis is a complex, multifactorial biological process, and, as such, it is also subject to gene-environment interplay; therefore, although the contribution of today's classical risk factors to the development of atherosclerosis is inquestionable, their role in the appearance of atherosclerotic lesions in the vascular tree involves not only inflammation and activation of the immune system but also genetic factors that facilitate or oppose the formation of lipid accumulation in the arterial wall.

Ferritin, Serum	**175**	**High**	ng/mL	15-150	01
Cholesterol, Total	**317**	**High**	mg/dL	100-199	01
Triglycerides	**157**	**High**	mg/dL	0-149	01
HDL Cholesterol	66		mg/dL	>39	01
VLDL Cholesterol Cal	29		mg/dL	5-40	
LDL Chol Calc (NIH)	**222**	**High**	mg/dL	0-99	

Comment:

Possible Familial Hypercholesterolemia. FH should be suspected when fasting LDL cholesterol is above 189 mg/dL or non-HDL cholesterol is above 219 mg/dL. A family history of high cholesterol and heart disease in 1st degree relatives should be collected. J Clin Lipidol 2011;5:133-140

T. Chol/HDL Ratio	**4.8**	**High**	ratio	0.0-4.4	

CBC, Platelet Ct, and Diff					01
WBC	8.0		x10E3/uL	3.4-10.8	01
RBC	5.23		x10E6/uL	3.77-5.28	01
Hemoglobin	14.8		g/dL	11.1-15.9	01
Hematocrit	45.8		%	34.0-46.6	01
MCV	88		fL	79-97	01
MCH	28.3		pg	26.6-33.0	01
MCHC	32.3		g/dL	31.5-35.7	01
RDW	13.1		%	11.7-15.4	01
Platelets	300		x10E3/uL	150-450	01
Neutrophils	48		%	Not Estab.	01
Lymphs	39		%	Not Estab.	01
Monocytes	8		%	Not Estab.	01
Eos	4		%	Not Estab.	01
Basos	1		%	Not Estab.	01

63 yo female diabetic

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Investigators from different research facilities have produced a complex, most likely still incomplete picture of the intricate relationship between inflammation-infection, genetics, and atherosclerotic diseases like CAD. In this issue of the *Brazilian Archives of Cardiology*, Rocha et al.,[14] sought to investigate the link between periodontal disease (as a model of chronic inflammatory condition) and two specific polymorphisms in genes knowingly related to inflammation (C-reactive protein and interleukin-6), with the presence of CAD in 80 patients from the South Region of Brazil referred for invasive coronary angiography. They found in the multivariate model that male gender and the CRP gene +1444C > T variant were significantly associated with the presence of CAD.

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Atherosclerosis, Inflammation, and Genetics - And you Thought it Was Just LDL-cholesterol

Luis Henrique Wolff Gowdak

Progress in cell and molecular biology has allowed us to refine our understanding of the mechanisms involved in the onset of atherosclerosis. LDL-cholesterol particles play a significant role in the genesis of atherosclerotic plaque in the presence of endothelial dysfunction, an omnipresent feature in individuals with cardiovascular risk factors.[3] Proliferation and migration of smooth muscle cells in response to the release of growth factors and the accumulation of mononuclear phagocytes rich in plasma-derived lipids (foam cells) contribute to the development of atheroma.[4] Further studies revealed that the immune system played a role in atherosclerosis through not only innate (macrophages) but also adaptive (T cell and other lymphocytes) pathways.[5] Cells directly involved in atherosclerosis establish a complex network of cross-talking by the release of cytokines, notably interleukin-1.[6]

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Atherosclerosis, Inflammation, and Genetics - And you Thought it Was Just LDL-cholesterol

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Once recognized as an inflammatory disease, a highly sensitive assay for the measurement of C-reactive protein (hsCRP) proved to be a marker for patients at high risk for cardiovascular events due to atherosclerosis and a useful tool in selecting patients for aggressive lipid control for risk reduction. In the JUPITER trial, statin therapy in patients with hsCRP values above the median for the population (> 2 mg/L) but with LDL-cholesterol level < 130 mg/dL had a 44% reduction in first-ever cardiovascular events.[7] More recently, the CANTOS trial allocated the anti-interleukin-1 antibody (canakinumab) to patients with stable post-acute coronary syndromes who had hsCRP values > 2 mg/L on statin therapy.[8] Individuals who achieved a reduction of hsCRP to < 2 mg/L in response to anti-inflammatory therapy had a > 30% reduction in cardiovascular and all-cause mortality.[9]

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T. Chol/HDL Ratio	**4.8**	**High**	ratio	0.0-4.4	

Please Note: 01

T. Chol/HDL Ratio

	Men	Women
1/2 Avg.Risk	3.4	3.3
Avg.Risk	5.0	4.4
2X Avg.Risk	9.6	7.1
3X Avg.Risk	23.4	11.0

C-Reactive Protein, Cardiac	2.13		mg/L	0.00-3.00	01

Relative Risk for Future Cardiovascular Event

Low	<1.00
Average	1.00 - 3.00
High	>3.00

The most important link of your
FM career:

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5986484/

The hidden Link: PAF (Platelet Activating Factor)

Nutrients. 2018 May; 10(5): 604.
Published online 2018 May 12. doi: 10.3390/nu10050604
PMCID: PMC5986484
PMID: 29757226

Inflammation, not Cholesterol, Is a Cause of Chronic Disease

Alexandros Tsoupras, Ronan Lordan, and Ioannis Zabetakis*

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This arti

For example, in cases of dyslipidaemia, increased cholesterol levels are not the causative agent or the underlying biochemical mechanism responsible for endothelial dysfunction and atherosclerosis development. The accumulation of excess plasma LDL cholesterol is addressed by the innate immune system as an undesired event. Therefore, an inflammatory response at the endothelial wall is promoted to reduce the threat by the removal of excess LDL and oxidised-LDL (Ox-LDL) cholesterol from the blood stream to the subendothelium, where they are engulfed by comigrated monocytes for final removal [63,64]. During chronic inflammatory diseases, inflammation and infections can also induce a variety of alterations in lipid metabolism, including decreases in serum HDL cholesterol, increases in triglycerides, lipoprotein(a), and LDL levels. These changes of the lipid levels may initially dampen inflammation or fight infection; however, the sustained inflammation can contribute to the increased risk of atherosclerosis [65]. In addition to affecting serum lipid levels, inflammation also adversely effects lipoprotein function; LDL is more easily oxidised, as the ability of HDL to prevent the oxidation of LDL is diminished, while several steps in the reverse cholesterol transport pathway are also adversely affected during inflammation. The greater the severity of the underlying inflammatory disease, the more consistently these abnormalities in lipids and lipoproteins are observed [65]. Thus, it is not serum cholesterol and lipoproteins that influence the endothelium but the inflammatory response that affects the well integrity and functionality of the endothelium.

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ISSN 2470-4563

The Expression of Platelet-Activating Factor is Induced by Low Extracellular Mg^{2+} in Aortic, Cerebral and Neonatal Coronary Vascular Smooth Muscle; Cross Talk with Ceramide Production, NF–kB and Proto-Oncogenes: Possible Links to Atherogenesis and Sudden Cardiac Death in Children and Infants, and Aging; Hypothesis, Review and Viewpoint

PAF ---- NF-KB ---- PAF ---- NF-KB ---- PAF

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B-48

Chylomicrons, gut level.

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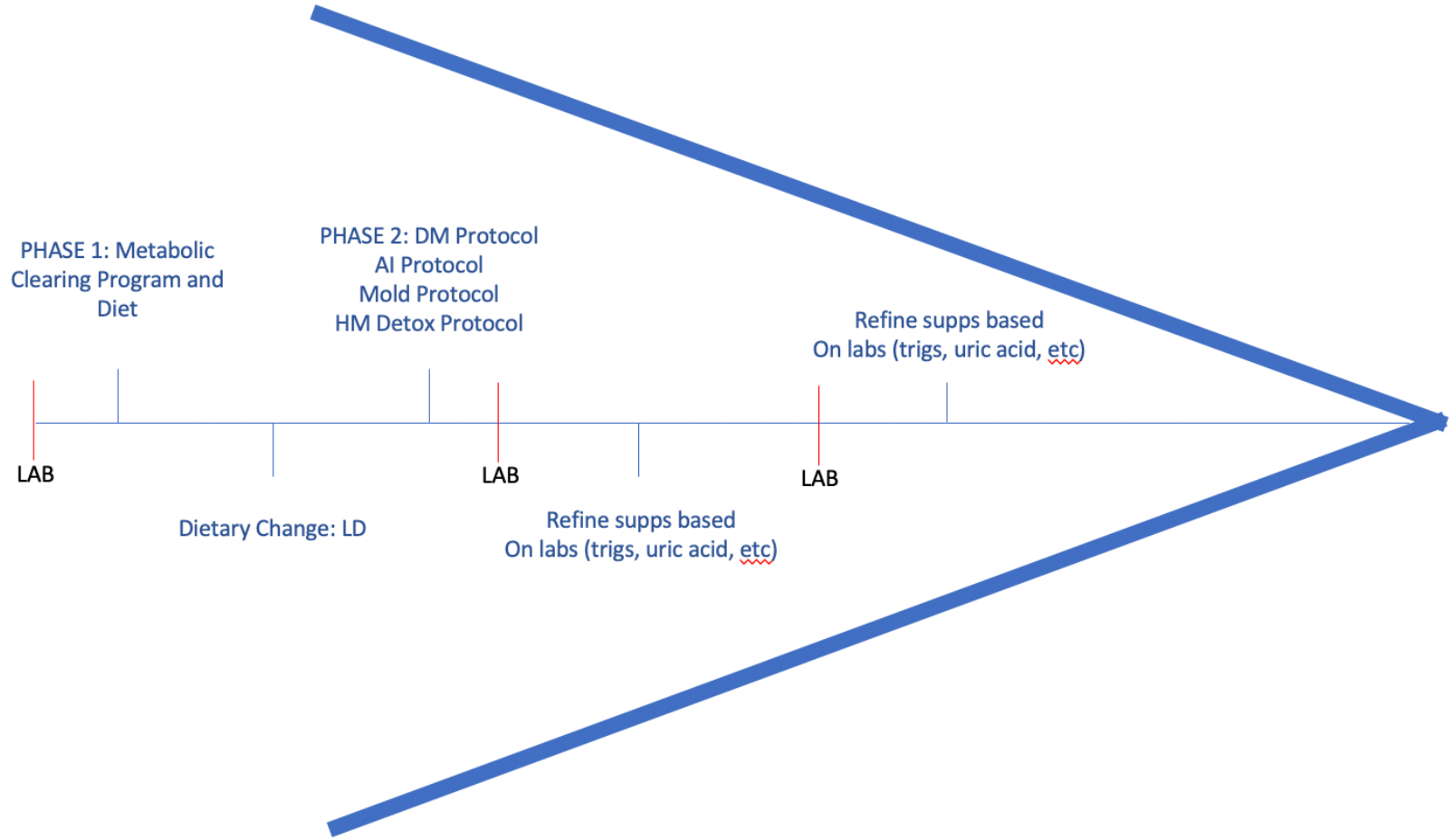
Repackaged in the liver with B-100 to form trig rich VLDL, and chol rich LDL.

Membrane health, sex hormones, steroids, etc.

Biogenetix Cardio Support Bundle

Supplement and Diet Protocols



Retest a lab at least every 60 days.

85% of patients will improve with basic structures and healthy eating.

% of problem analysis: this is what the cleanse is for.

General → Fine Tune

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