

Casual Friday Series

Functional Blood Chemistry Series

Pt. 10: Lipids (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.*



Last week...

- Lipid Panel
- NMR Lipoprofile
- APO B



Lipid Lab Ranges

	Range	
Cholesterol	145-199	Be Smart.
HDL	45-59	What happens when its too high?
Trigs	50-99	Particle size will skew.
LDL	50-99	



NMR Lipoprofile

LDL-P is the measurement of lipoprotein particles. (carriers)

LDL-C is the measurement of cholesterol mass within the LDL particles.





Moderate LDL-C and moderate LDL-P



High LDL-C and low LDL-P



Low LDL-C and high LDL-P



P1: 39 yo male

NMR LipoProfile+Lipids+Graph

Test	Current Result and Flag	Previous Result and Date	Units	Reference Interval
LDL Particle Number ⁰¹				
▲ LDL-P ^{A,01}	1296 High		nmol/L	<1000
		Low	< 1000	
		Moderate	1000 - 1299	
		Borderline-High	1300 - 1599	
		High	1600 - 2000	
		Very High	> 2000	
Lipids ⁰¹				
▲ LDL-C (NIH Calc) ⁰¹	168 High		mg/dL	0-99
		Optimal	< 100	
		Above optimal	100 - 129	
		Borderline	130 - 159	
		High	160 - 189	
		Very high	> 189	
HDL-C ^{A,01}	80		mg/dL	>39
Triglycerides ^{A,01}	58		mg/dL	0-149
▲ Cholesterol, Total ^{A,01}	257 High		mg/dL	100-199
LDL and HDL Particles ⁰¹				
HDL-P (Total) ^{A,01}	39.2		umol/L	>=30.5
Small LDL-P ^{A,01}	279		nmol/L	<=527
LDL Size ^{A,01}	21.8		nm	>20.5

**** INTERPRETATIVE INFORMATION****
PARTICLE CONCENTRATION AND SIZE
 <--Lower CVD Risk Higher CVD Risk-->
 LDL AND HDL PARTICLES Percentile in Reference Population
 HDL-P (total) High 75th 50th 25th Low
 >34.9 34.9 30.5 26.7 <26.7
 Small LDL-P Low 25th 50th 75th High
 <117 117 527 839 >839
 LDL Size <-Large (Pattern A)-> <-Small (Pattern B)->
 23.0 20.6 20.5 19.0

Comment:⁰¹
 Small LDL-P and LDL Size are associated with CVD risk, but not after LDL-P is taken into account.

Insulin Resistance Score⁰¹
 LP-IR Score^{A,01} <25 <=45
INSULIN RESISTANCE MARKER
 <--Insulin Sensitive Insulin Resistant-->
 Percentile in Reference Population



Apolipoprotein B and Cardiovascular Disease: Biomarker and Potential Therapeutic Target

[Jennifer Behbodikhah](#), [Saba Ahmed](#), [Ailin Elyasi](#), [Lora J. Kasselmann](#), [Joshua De Leon](#), [Amy D. Glass](#), and [Allison B. Reiss](#)*

M Apolipoprotein (apo) B, the critical structural protein of the atherogenic lipoproteins, has two major isoforms: apoB48 and apoB100. ApoB48 is found in chylomicrons and chylomicron remnants with one apoB48 molecule per chylomicron particle. Similarly, a single apoB100 molecule is contained per particle of very-low-density lipoprotein (VLDL), intermediate density lipoprotein, LDL and lipoprotein(a). This unique one apoB per particle ratio makes plasma apoB concentration a direct measure of the number of circulating atherogenic lipoproteins. ApoB levels indicate the atherogenic particle concentration independent of the particle cholesterol content, which is variable. While LDL, the major cholesterol-carrying serum lipoprotein, is the primary therapeutic target for management and prevention of atherosclerotic cardiovascular disease, there is strong evidence that apoB is a more accurate indicator of cardiovascular risk than either total cholesterol or LDL cholesterol. This review examines multiple aspects of apoB structure and function, with a focus on the controversy over use of apoB as a therapeutic target in clinical practice. Ongoing coronary artery disease residual risk, despite lipid-lowering treatment, has left patients and clinicians with unsatisfactory options for monitoring cardiovascular health. At the present time, the substitution of apoB for LDL-C in cardiovascular disease prevention guidelines has been deemed unjustified, but discussions continue.



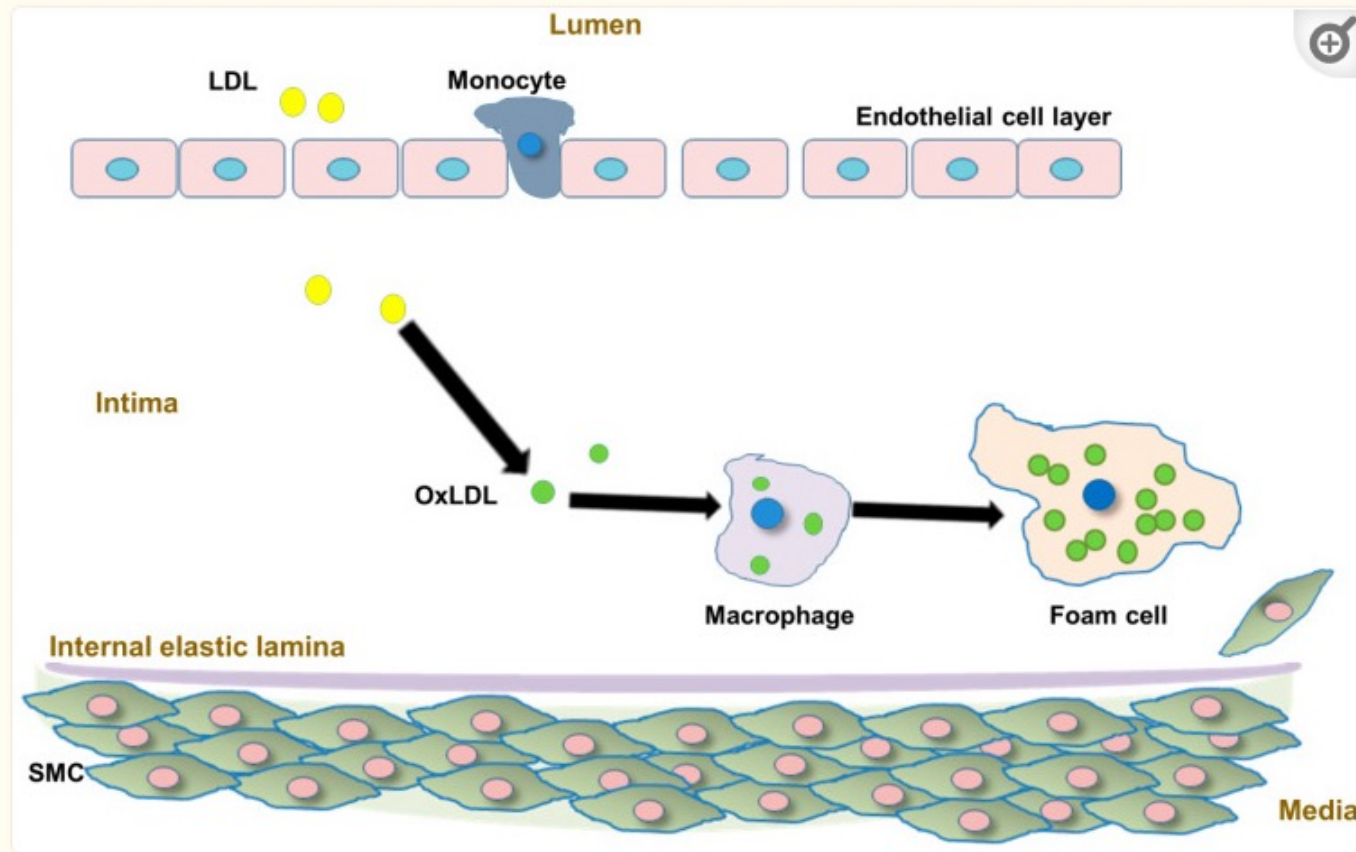


Figure 1

Atherosclerosis involves apoB-containing lipoproteins. The atherosclerotic process begins with compromise of the endothelial barrier, allowing apoB-containing LDL cholesterol to migrate into the arterial intima. Activated endothelium fosters attachment, migration and proliferation of vascular smooth muscle cells (SMC) and macrophages. Retained apoB-containing lipoproteins are oxidatively modified within the vascular intima. Oxidized (ox)LDL contains protein components, creating a net negative charge, making the particles highly attractive to macrophages. Phagocytosis allows for the accumulation of lipids within macrophages, producing foam cells. OxLDL-laden foam cells amass and form the fatty streak and eventually the lumen-narrowing atheromatous plaque that restricts blood flow. Additionally, inflammatory signaling pathways are activated, leading to increased cell migration and LDL modification.

The relation between ApoB/ApoA-1 ratio and the severity of coronary artery disease in patients with acute coronary syndrome

[Rehab Ibrahim Yaseen](#) , [Mohamed Hesham El-Leboudy](#) & [Hend Mohammed El-Deeb](#)

[The Egyptian Heart Journal](#) **73**, Article number: 24 (2021) | [Cite this article](#)

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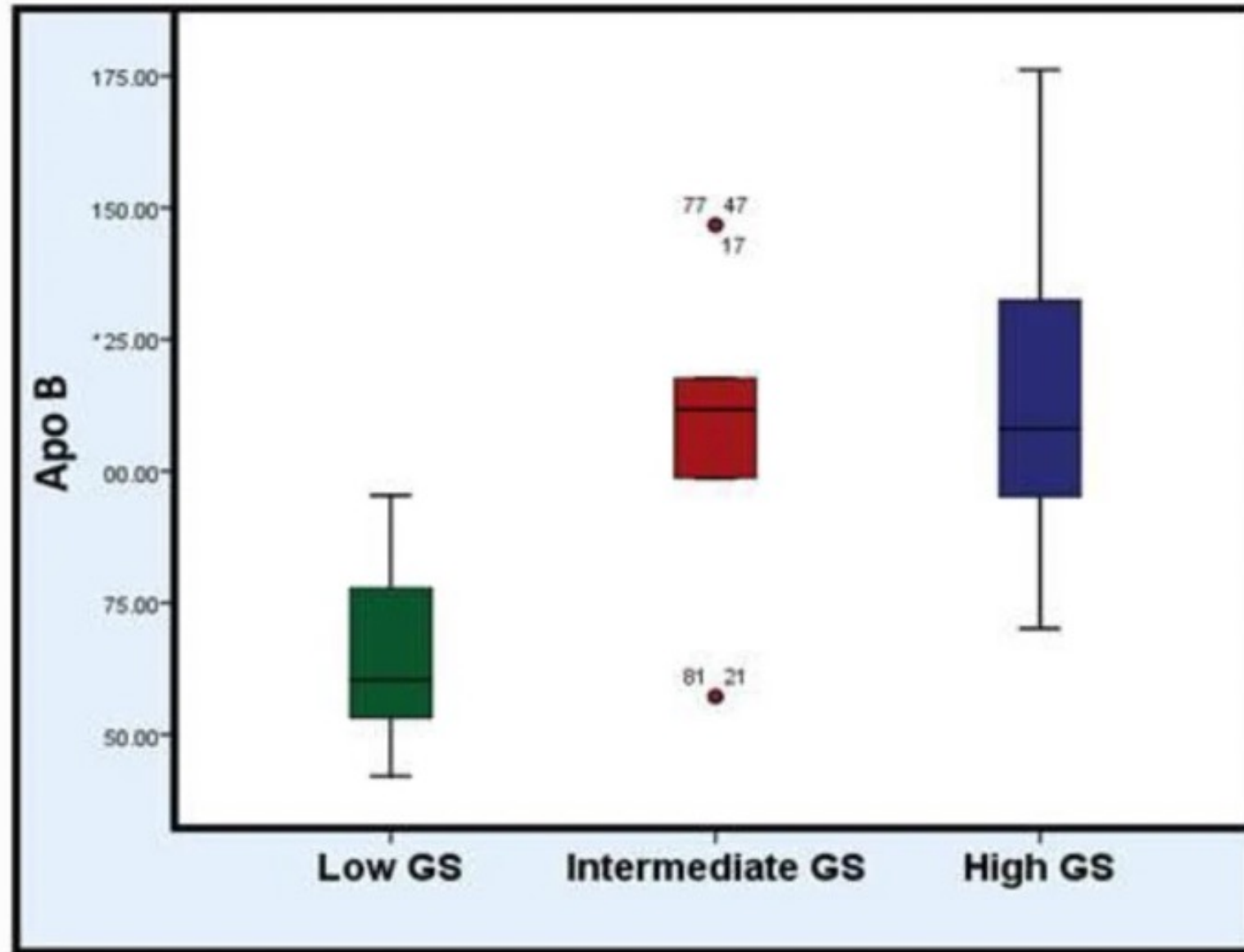
Apolipoprotein B is considered the primary protein constituent of low-density lipoprotein. LDL contains variable quantities of cholesterol, but each lipoprotein contains a single ApoB protein. Thus, ApoB is a better index for the LDL circulation if compared to LDL cholesterol. On the contrary, apolipoprotein A-1 is a main structural protein of high-density lipoprotein. It has a major role in reversing cholesterol flow and cellular cholesterol homeostasis once detected. The aim of the study is to measure apo B/apo A-1 ratio in patients with acute coronary syndrome and assess its relationship with the severity of CAD.

A total of 90 patients were enrolled in the study and subdivided into 3 groups: 30 patients of STEMI, 30 patients of NSTEMI, and 30 patients presented with unstable angina. Serum levels of apolipoprotein A-1 and apolipoprotein B were properly measured upon admission, and apo B/apo A-1 ratio was calculated.



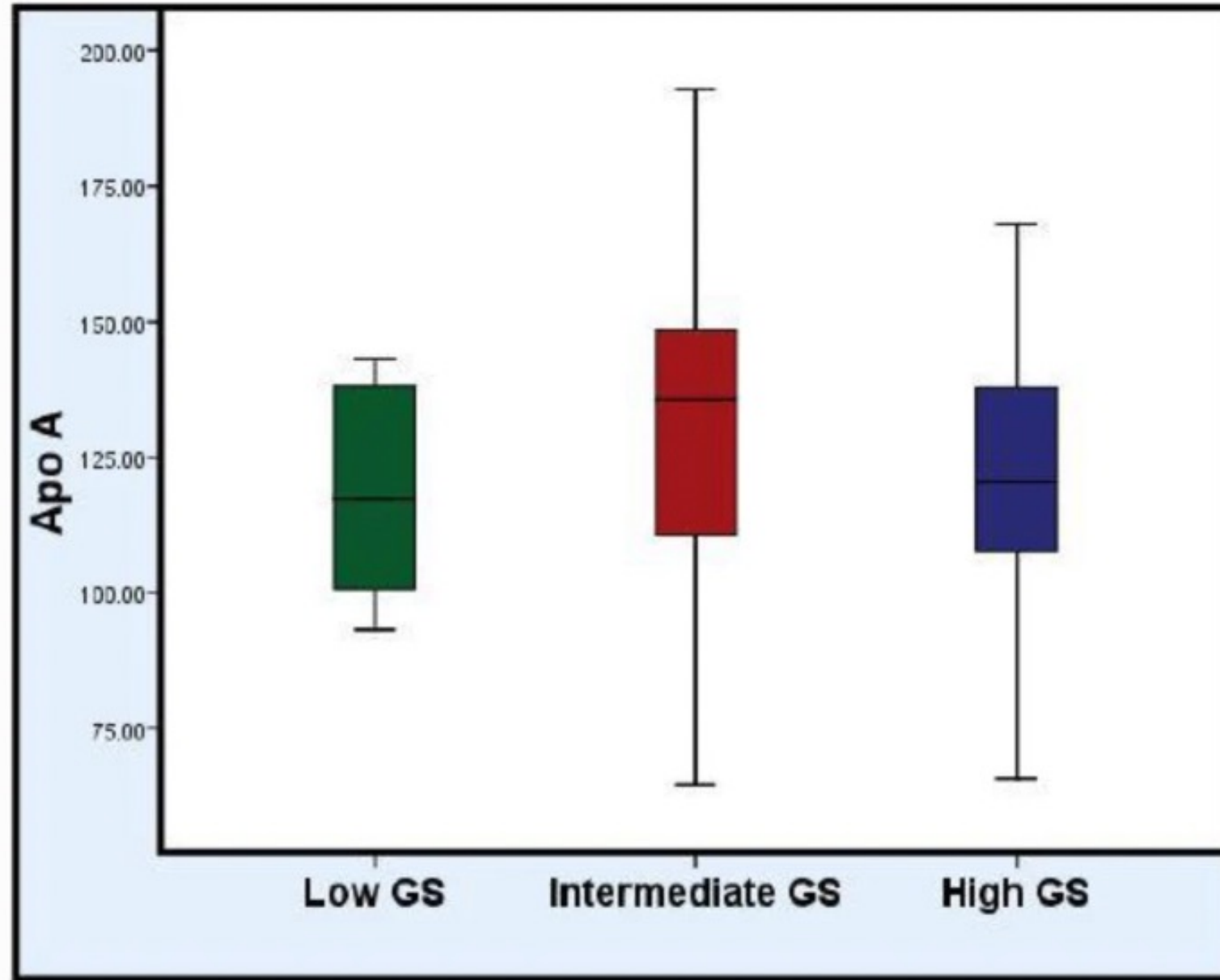
Gensini Score vs.

Fig. 1

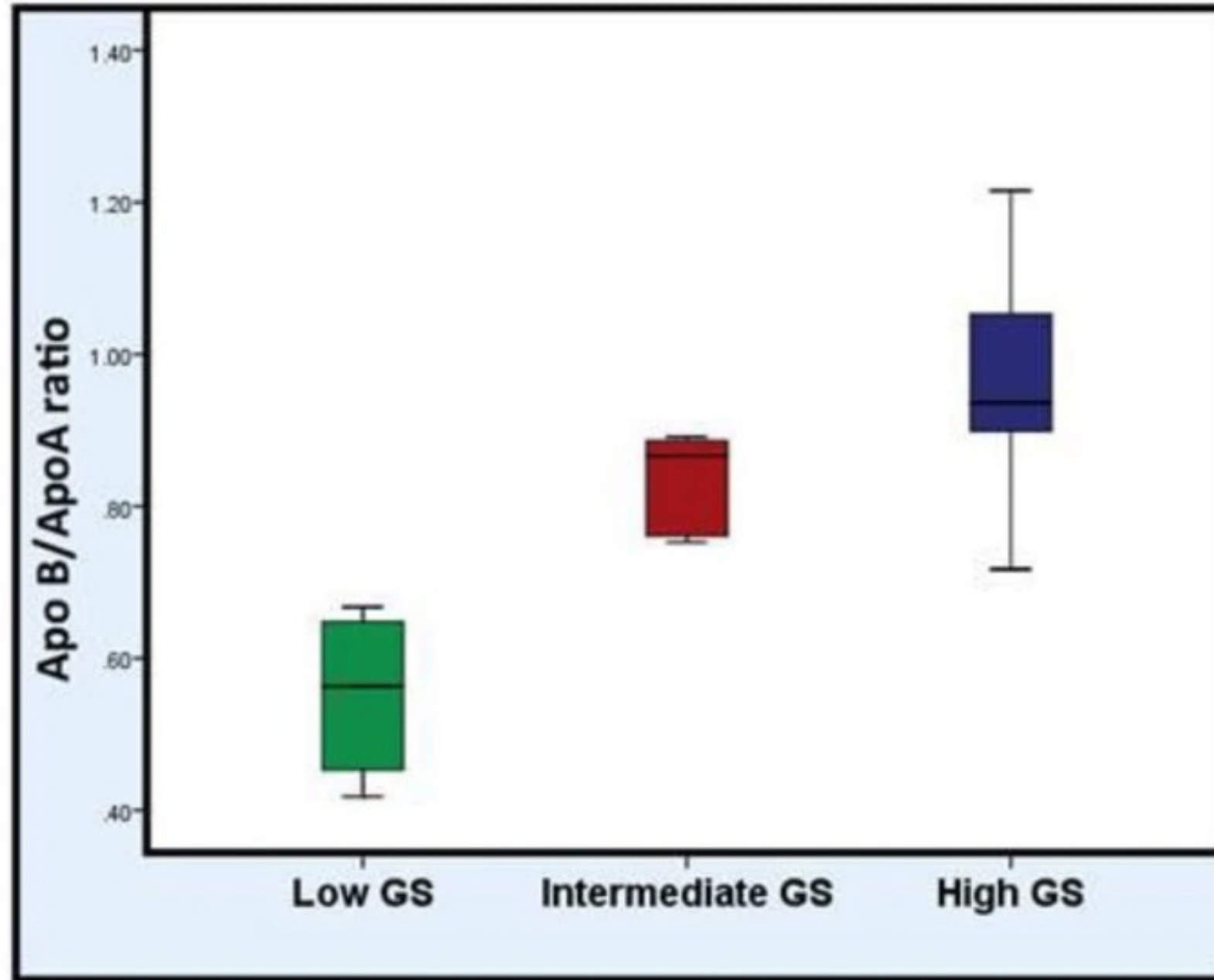


Gensini Score vs.

Fig. 2



Gensini Score vs.



The relation between ApoB/ApoA-1 ratio and the severity of coronary artery disease in patients with acute coronary syndrome

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We found a strong positive correlation of both Apo B and ApoB/ApoA1 in predicting high Gensini scores (scores ≥ 47) in patients who suffered from acute coronary syndromes (P value < 0.001). Patients with NSTEMI and STEMI had significantly higher Apo B/Apo A 1 ratio and Apo B levels than those with UA (P value < 0.05). On the contrary, no significant correlation between Apo A and Gensini scores was found (P value > 0.05). It is known that each particle of the atherogenic lipoproteins like low-density lipoprotein, very-low-density lipoprotein, intermediate-density lipoprotein, and lipoprotein (a) carries a single ApoB100 molecule. Hence, the plasma ApoB100 concentrations notably reflect proatherogenic potentials. On the other hand, ApoA is the key apolipoprotein component of high-density lipoprotein which represents the ApoA serum content whereby antiatherogenic potentials are witnessed. Thus, the ApoB100/ApoA1 ratios represent the balance between the harmful and beneficial potentials. The higher the ApoB100/ApoA1 ratio, the more developed atherogenic potentials we get and/or the less antiatherogenic potentials we attain [12].



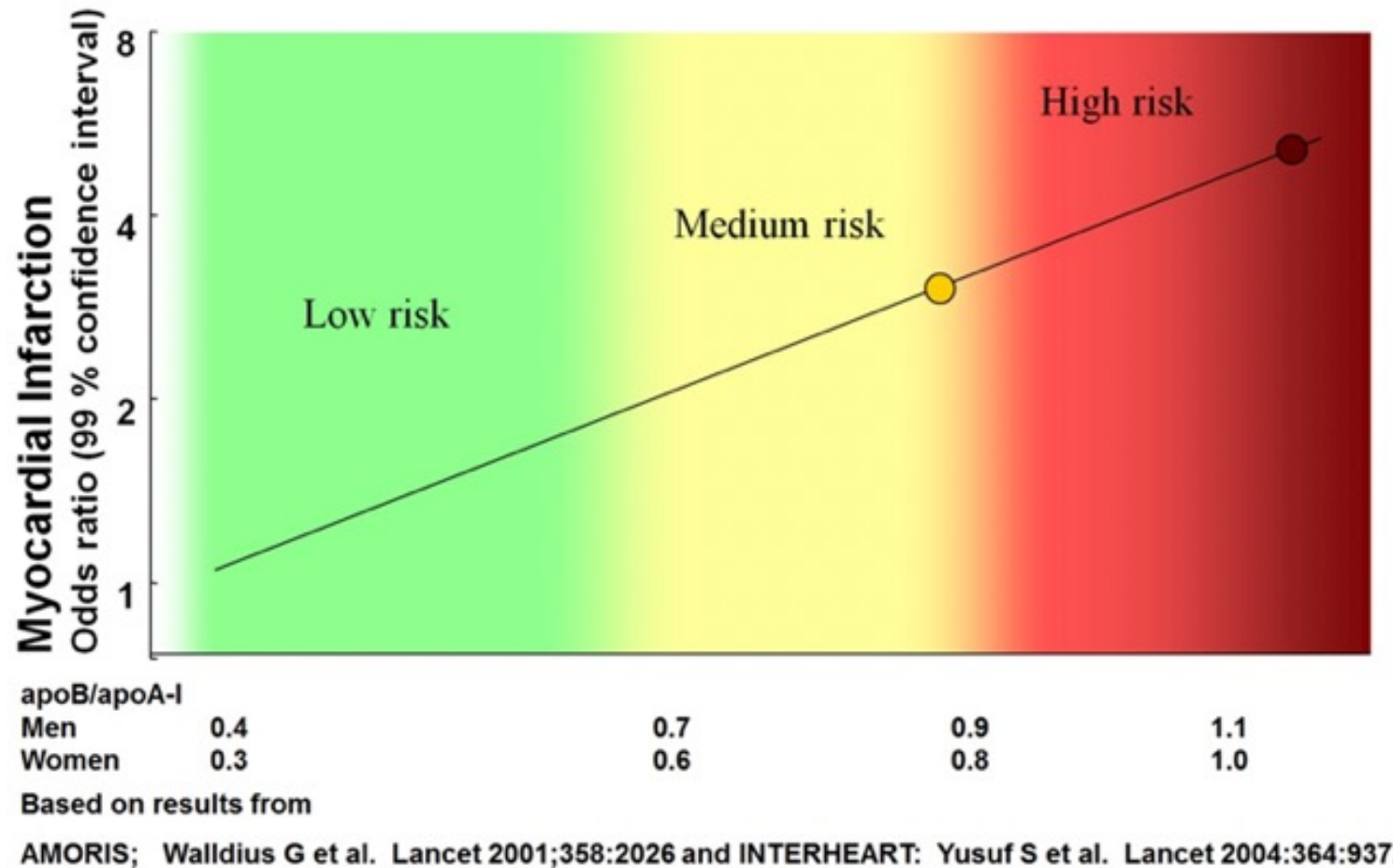
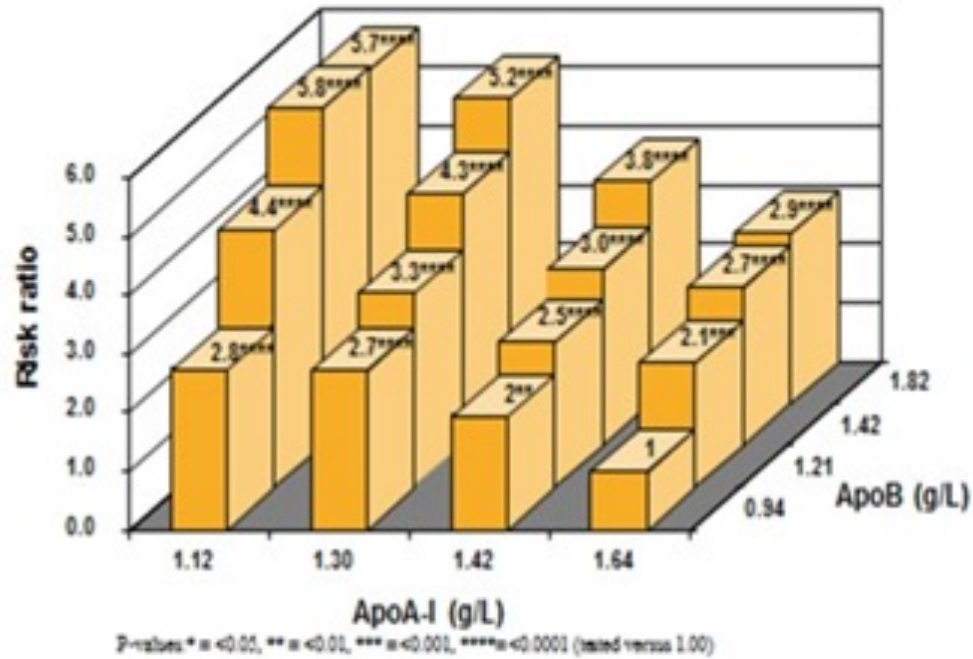


Figure 10.

This line of risk of myocardial infarction is based on the findings in the AMORIS (reference 3) and the INTERHEART (reference 58) studies. Tentative cut-values are indicated in green (low risk), yellow (medium risk), and red (high risk). Values for a particular patient can be indicated by the dots on the line. During lipid-lowering treatment it is easy to monitor how a patient moves upwards or downwards in the risk line for the apo-ratio. <https://www.intechopen.com/chapters/39545>

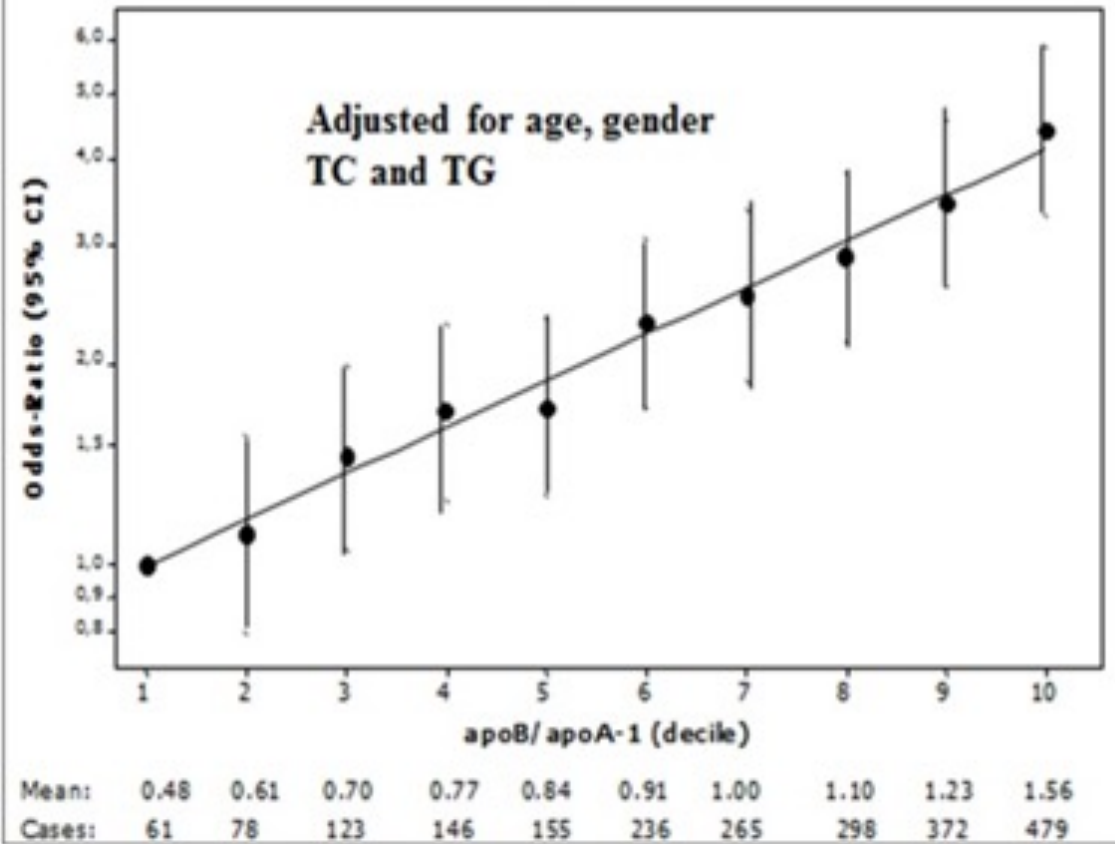


AMORIS: Fatal MI Men, n = 1,267: adjusted for age, TC, and TG

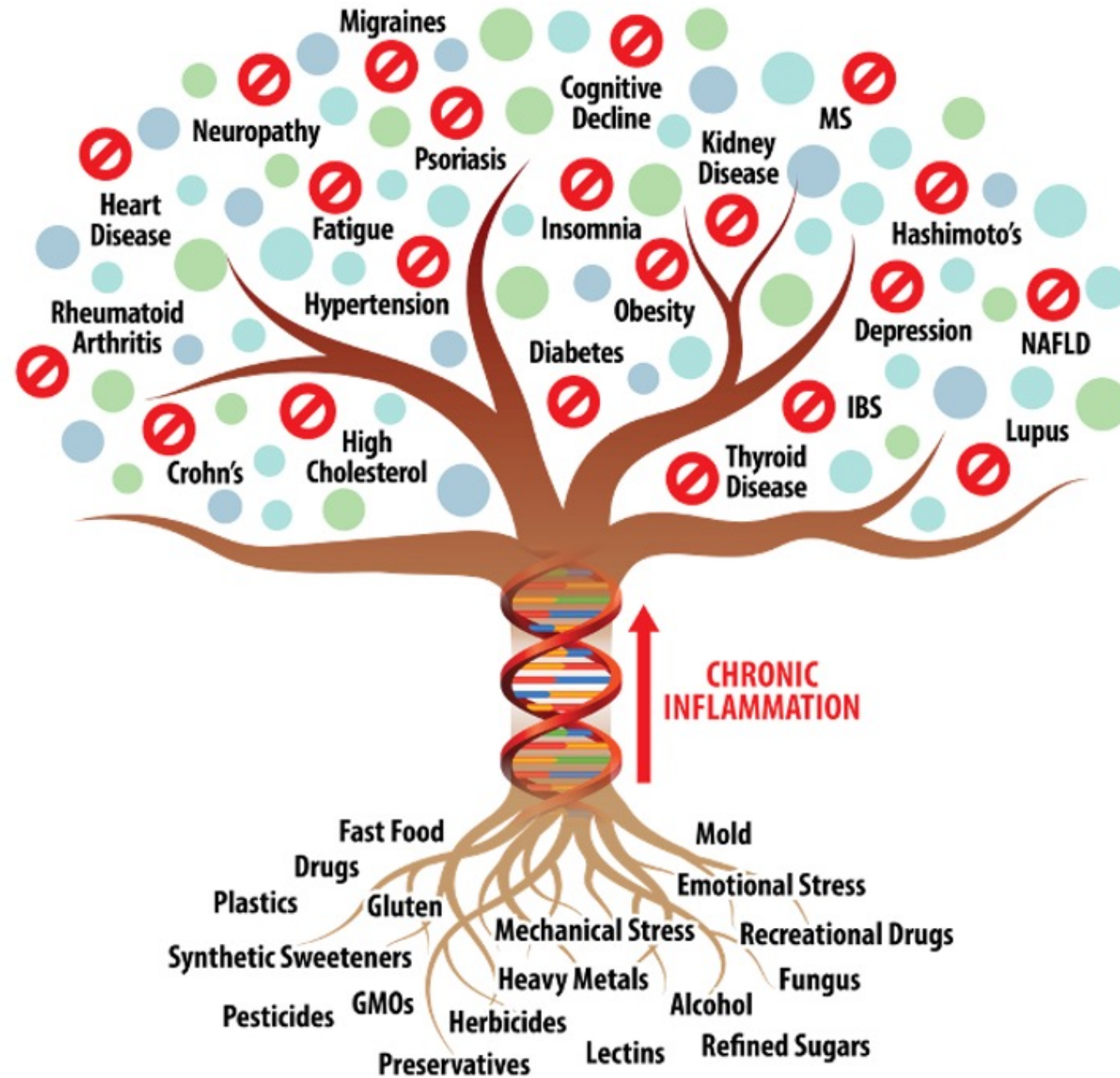


Walldius G, Jungner I J Intern Med 2004;255:947-56.

AMORIS: fatal myocardial infarction



Pause.



Lifestyle and Dietary Determinants of Serum Apolipoprotein A1 and Apolipoprotein B Concentrations: Cross-Sectional Analyses within a Swedish Cohort of 24,984 Individuals

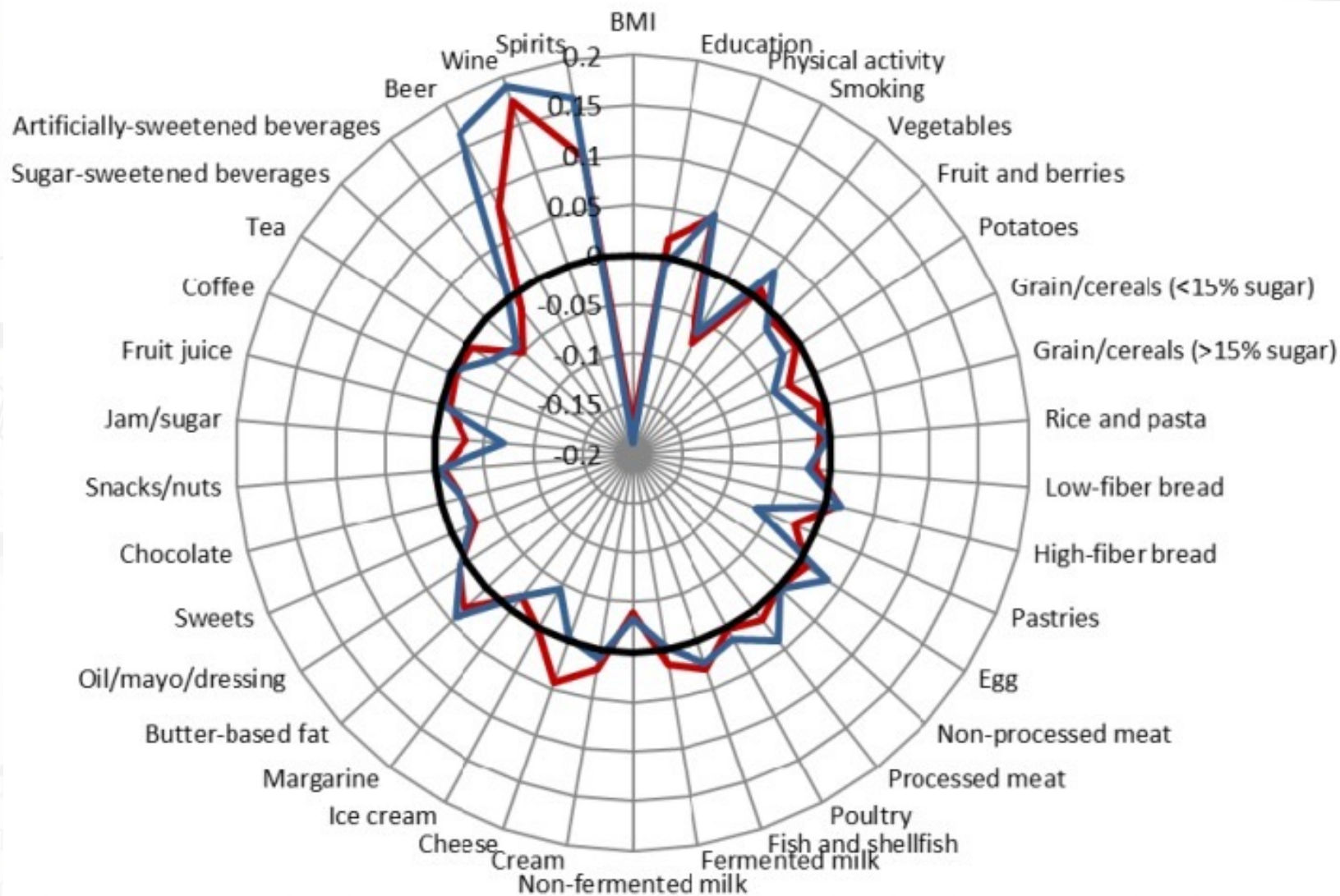
[Kasper Frondelius](#),¹ [Madelene Borg](#),¹ [Ulrika Ericson](#),¹ [Yan Borné](#),² [Olle Melander](#),³ and [Emily Sonestedt](#)^{1,4,*}

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Low serum apolipoprotein (Apo) A1 concentrations and high serum ApoB concentrations may be better markers of the risk of cardiovascular disease than high-density lipoprotein (HDL) and low-density lipoprotein (LDL). However, the associations between modifiable lifestyle factors and Apo concentrations have not been investigated in detail. Therefore, this study investigated the associations between Apo concentrations and education, lifestyle factors and dietary intake (macronutrients and 34 food groups). These cross-sectional associations were examined among 24,984 individuals in a Swedish population-based cohort. Baseline examinations of the cohort were conducted between 1991 and 1996. Dietary intake was assessed using a modified diet history method. The main determinants of high ApoA1 concentrations (r between 0.05 and 0.25) were high alcohol consumption, high physical activity, non-smoking, and a low body mass index (BMI), and the main determinants of high ApoB concentrations were smoking and a high BMI. The intake of sucrose and food products containing added sugar (such as pastries, sweets, chocolate, jam/sugar and sugar-sweetened beverages) was negatively correlated with ApoA1 concentrations and positively correlated with ApoB concentrations and the ApoB/ApoA1 ratio, whereas the intake of fermented dairy products, such as fermented milk and cheese, was positively correlated with ApoA1 concentrations and negatively correlated with the ApoB/ApoA1 ratio. These results indicate that smoking, obesity, low physical activity, low alcohol consumption and a diet high in sugar and low in fermented dairy products are correlated with an unfavorable Apo profile.

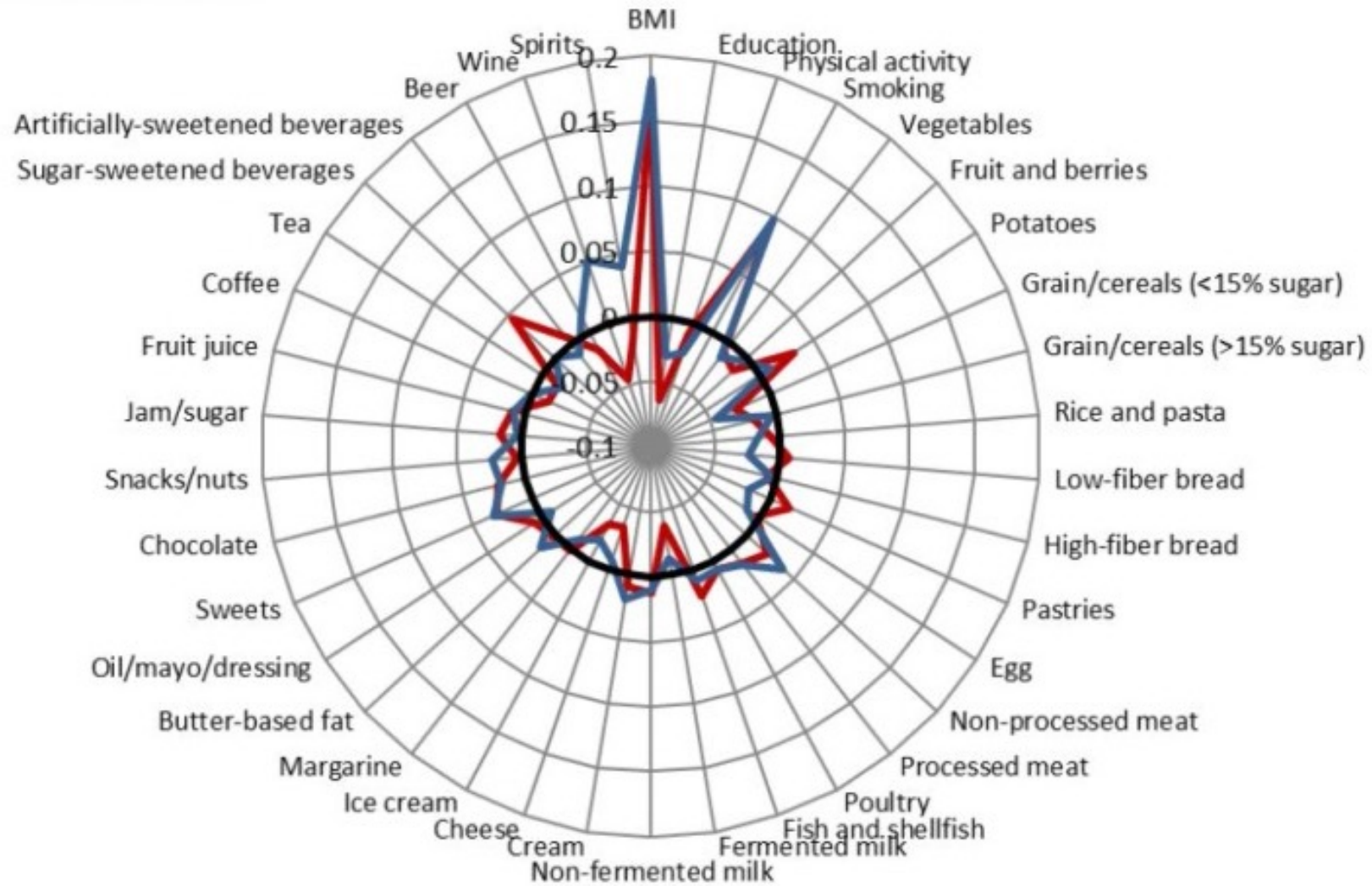


ApoA1

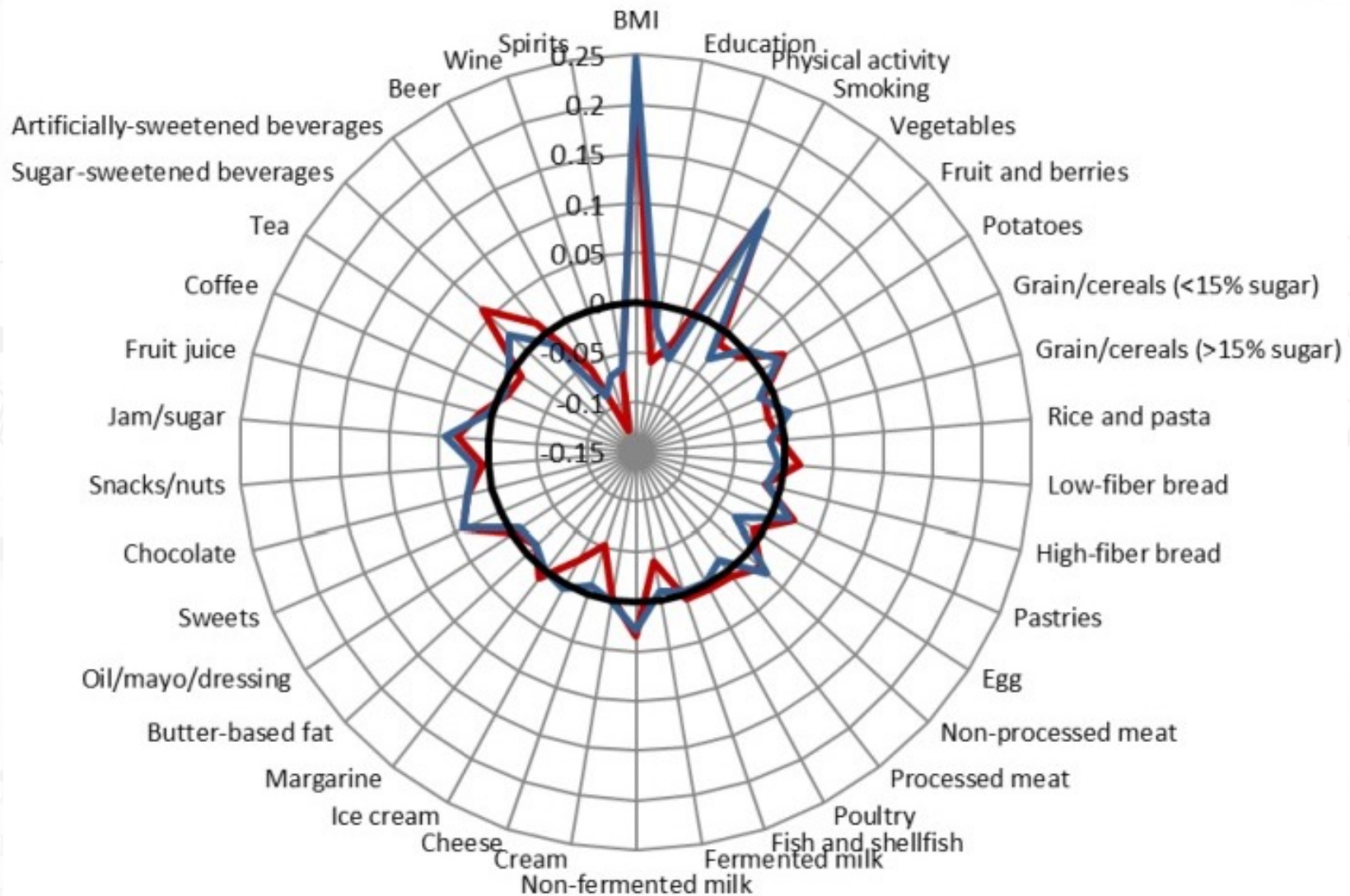


Click on image to zoom

ApoB



ApoB/A1



Adipokines and Obesity. Potential Link to Metabolic Disorders and Chronic Complications

[Katarzyna Zorena](#),^{1,*} [Olga Jachimowicz-Duda](#),² [Daniel Ślęzak](#),³ [Marlena Robakowska](#),⁴ and [Małgorzata Mrugacz](#)⁵

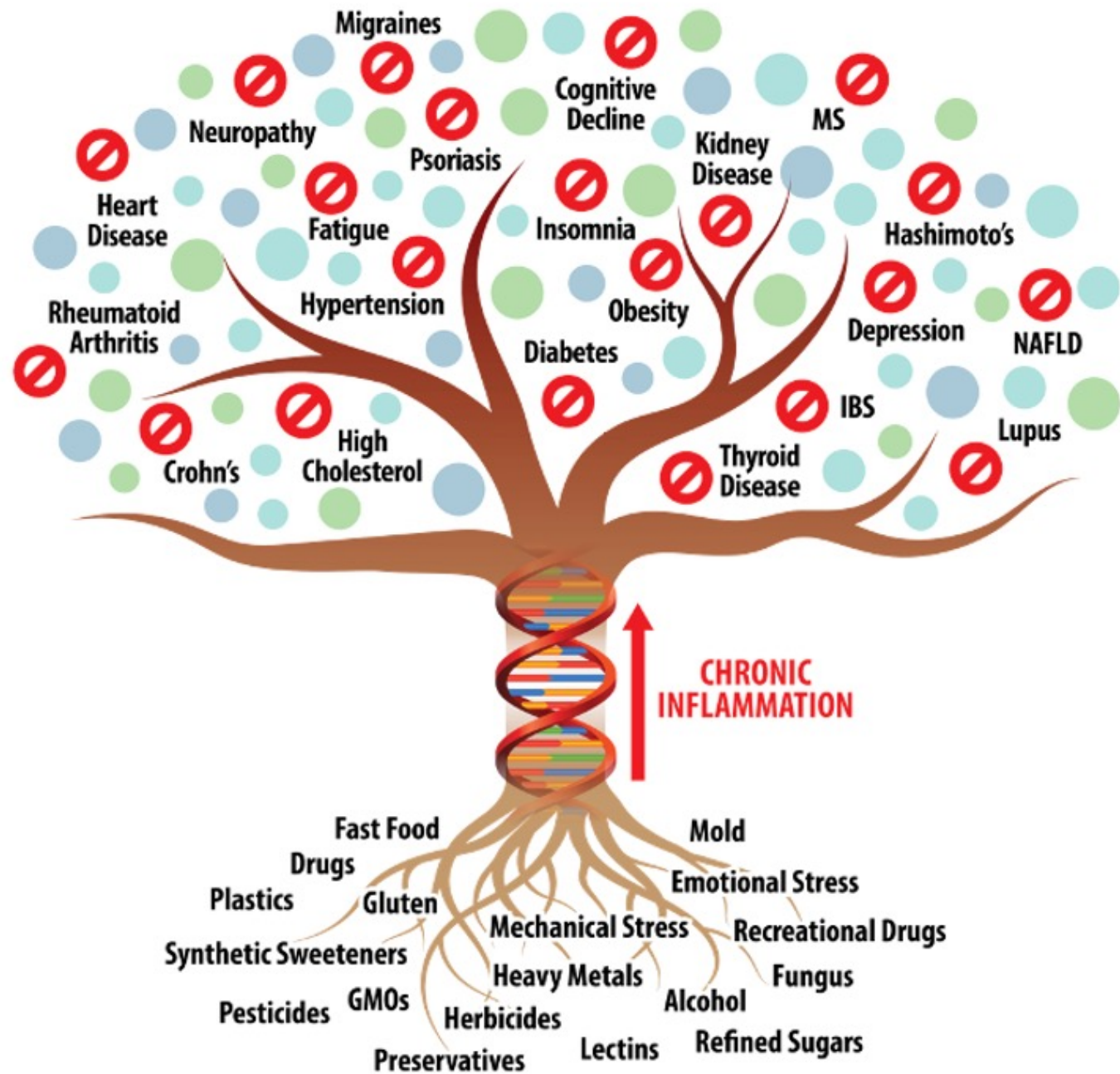
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Abstract

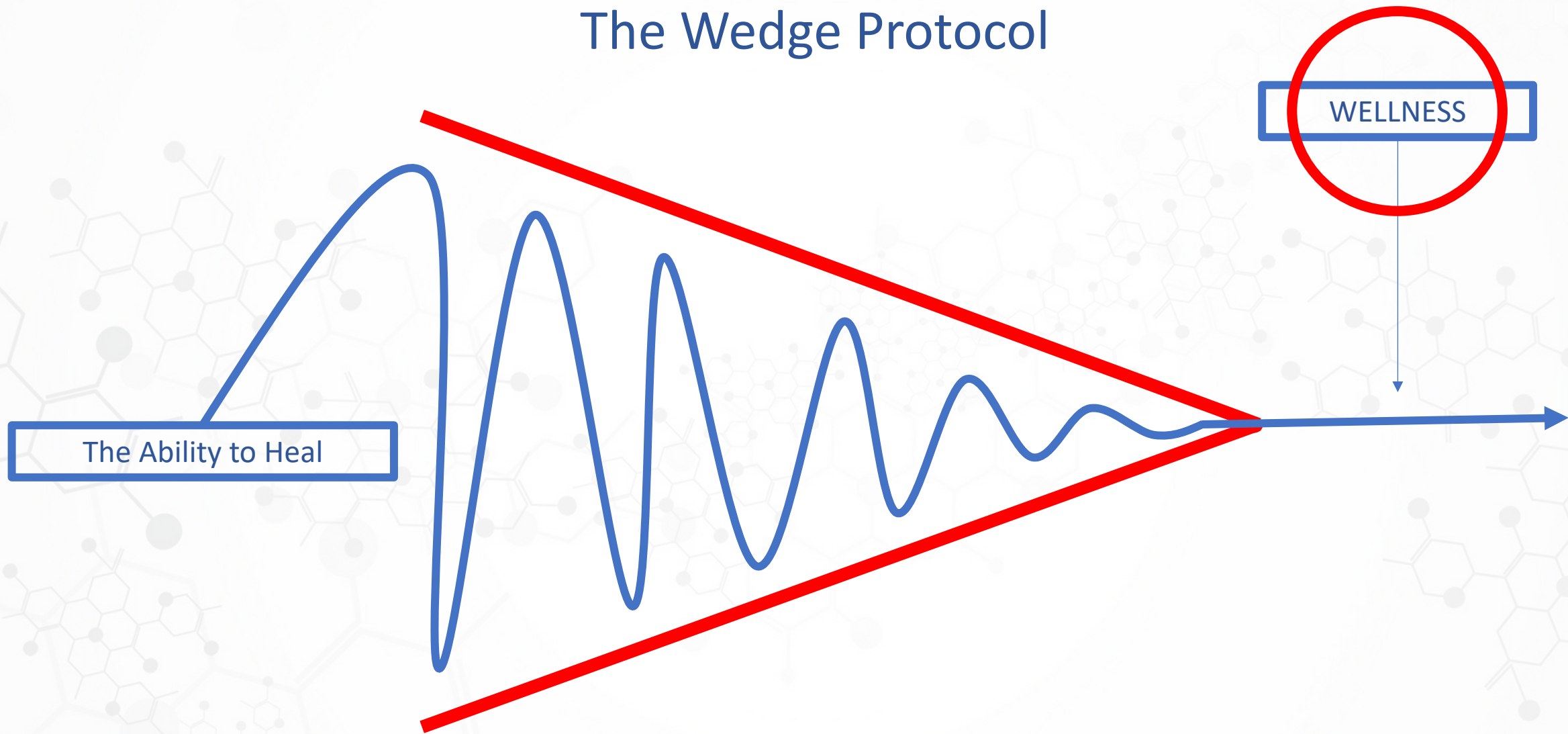
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The World Health Organization (WHO) has recognized obesity as one of the top ten threats to human health. It is estimated that the number of obese and overweight people worldwide exceeds the number of those who are undernourished. Obesity is not only a state of abnormally increased adipose tissue in the body, but also of increased release of biologically active adipokines. Adipokines released into the circulating blood, due to their specific receptors on the surface of target cells, act as classic hormones affecting the metabolism of tissues and organs. What is more, adipokines and cytokines may decrease the insulin sensitivity of tissues and induce inflammation and development of chronic complications. Certainly, it can be stated that in an era of a global obesity pandemic, adipokines may gain more and more importance as regards their use in the diagnostic evaluation and treatment of diseases. An extensive search for materials on the role of white, brown and perivascular fatty tissue and obesity-related metabolic and chronic complications was conducted online using PubMed, the Cochrane database and Embase.





The Wedge Protocol



The Ability to Heal

WELLNESS

