LIPOPROTEIN A & APOLIPOPROTEIN B
AND CORONARY ARTERY DISEASE

How significant is Lipoprotein A as an independent risk factor for CAD? --- Addressing that question forces us to jump into the "nature versus nurture" debate. ----- Statistically speaking, elevated Lipoprotein A is indeed an independent risk factor for CAD. But, since every individual's Lipoprotein A level is almost entirely genetically determined, it gives zero information on whether a person needs therapeutic intervention to treat or prevent CAD, and, zero information on whether any instituted treatment is yielding any benefit.

Lipoprotein A comes much closer than cholesterol does to being legitimately considered an independent factor. But, as with cholesterol, there are many who experience fatal MI who have extremely low Lipoprotein A, just as there are those who have extremely high Lipoprotein A and live to old age with no coronary artery disease whatsoever.

Since it is genetically determined, there is a limit to how low it can be brought down with proper Metabolic Therapy. ----- So, monitoring a person's Lipoprotein A ends up being nothing more than an exercise in continual torment. Since the level does not consistently respond to any therapeutic intervention, once high it is going to remain high, and all the physician will do in continuously reminding the patient of elevated Lipoprotein A is create undo insecurity/fear.

Even worse, when a physician finds an elevated Lipoprotein A, that doctor is likely to institute counterproductive therapies, thinking the patient is being protected --- such as driving the patient's cholesterol down to a pathologically low level, and giving blood pressure medication in the presence of perfectly normal blood pressure.

To what extent is Lipoprotein A modifiable by therapeutic intervention? I have seen patients with no therapeutic intervention whose Lipoprotein A varied from 100 to 180. ----- So, it is very subject to influence by environmental factors. ----- Thyroid appears to be one factor controlling Lipoprotein A. Carnitine as a supplement has shown the ability to lower it. Niacin has also become a popular remedy. ----- While Lipoprotein A is very definitely an independent risk factor for heart attacks and strokes, its influence is overrated --- or perhaps I should say its "independence" is overrated.

Getting back to nature versus nurture --- think along these lines ----- there is a higher incidence of blood-borne cancers in those with Blood Type O, and a higher incidence of hard tumors in those with Blood Type A. So what? ----- There is a higher incidence of Th1-mediated autoimmune diseases in those who are left-handed, and in women, --- and particularly in left-handed women. So what? ----- Should we treat all left-handed women as if they have rheumatoid arthritis as a preventive measure?

So it is with Lipoprotein A. While there is some statistical relevance to the analyte, objective evidence indicates that it is clinically to be noted, then ignored.

Apolipoprotein B is also genetic to a large extent --- but is also subject to improvement with Metabolic Therapy. And even more than the level being subject to improvement, its degree of pathogenesis is variable.

----- Insulin resistance is a big factor. In other words, controlling sugar and carb intake is beneficial to an extreme. It is also evident from the literature that Apolipoprotein B, while an independent risk factor for heart attacks and strokes, is far less significant than the Apo B to Lipoprotein A ratio.

Apo B is inversely proportional to the HDL level. So, anything that improves HDL --- such as adequate thyroid, low sugar diet, low HOHUM-PUFA diet, high-intensity short duration exercise, and all other factors that control Insulin Resistance (--- such as the Immuno-Neuro-Endocrine Stress of Metabolic Imbalances),are important considerations. ------- In other words --- what is “good for” the Apo B to Lipoprotein A ratio is NUTRI-SPEC.

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