TRIGLYCERIDES AS A RISK FACTOR FOR CARDIOVASCULAR DISEASE

A. Independent Risk Factors for Cardiovascular Disease include:

1. [Elevated total cholesterol is not an independent risk factor for heart attacks, unless the total cholesterol is close to 300 (>280)]

2. Elevated triglycerides are the Number 3 independent risk factor for cardiovascular disease.

3. Low HDL cholesterol is the Number 2 independent risk factor for heart attacks and strokes.

4. An elevated triglyceride to HDL ratio is the Number 1 independent risk factor for heart attacks and strokes.

5. Elevated C-reactive protein

6. Elevated homocysteine

7. Elevated fibrinogen

8. Hypothyroid or Thyroid Insufficiency

9. Low testosterone

10. Electrolyte Stress Imbalance with Hypertension associated with the kidney hormone renin

11. All the manifestations of Metabolic Syndrome === [elevated triglycerides + abdominal obesity + elevated Blood Sugar + Hypertension]

12. There are other blood analytes that are independent risk factors for heart attacks and strokes --- but they are expensive and beyond the scope of routine clinical testing. They rarely yield more/better information than the risk factors listed above.

B. Causes of high triglycerides:

1. Sugar Intake

2. Alcohol Intake

3. High Carb + Low Protein Diet

4. Insulin Excess caused by 1-3 above === Insulin Resistance === Metabolic Syndrome

5. Insulin Excess = Insulin Resistance from frequent eating (snacking) === Maximum = 3 feedings daily

6. Hypothyroid or Thyroid Insufficiency

7. Birth Control Pills; Estrogen Replacement Therapy; Excess Endogenous Estrogen

8. Thiazide Diuretics

9. Some Beta Blockers

10. Familial Hyperlipidemias

C. Drug Treatment for Elevated Triglycerides: There are three drugs generally recognized as effective in lowering triglycerides ----- Clofibrate, Gemfibrozil, and Nicotinic Acid.

1. Clofibrate is associated with few side effects --- but gall stones, myalgias and (rarely) ventricular arrhythmias have been reported. --- Strangely (alarmingly!), in large clinical trials to assess the efficacy of Clofibrate in patients who have high cholesterol in addition to high triglycerides, there is an increase in drug-associated mortality that offsets the beneficial response from lowering the triglycerides and total cholesterol. Also, the lowering of triglycerides by Clofibrate is often associated with an actual increase in LDL cholesterol --- which is causing many in the medical/pharmaceutical establishment to question whether a net gain is achieved in overall lipoprotein metabolism with Clofibrate.

2. Gemfibrozil has many of the actions as Clofibrate. Again, triglyceride reduction often results in an actual rise of LDL cholesterol. The side effects of Gemfibrozil resemble those of Clofibrate, but are probably less severe since an overall increase of mortality has not been demonstrated.

3. Nicotinic Acid results in flushing and itching as early side effects, but most people develop tolerance. But more severe side effects of Nicotinic Acid include hyperuricemia, increased blood sugar, and liver damage --- with rising liver enzymes. Gastrointestinal complaints are also common. Heart arrhythmias have also been reported at high doses of niacin. There is also increased prothrombine time and decreased platelet count --- which is significant in patients who are also taking anticoagulants. The time-release niacin products are designed to decrease the flushing reaction --- but can cause acute toxic reactions. These time-release

products increase the side effects listed above --- insulin resistance leading to diabetes, liver dysfunction/damage, along with gastrointestinal symptoms, fatigue, and low blood pressure.

[The flushing is mediated by Prostaglandin D2 (PGD2) as the primary cause, with serotonin playing a secondary role. The PGD2, along with Prostaglandin E2 (PGE2) activates Langerhans cells (a type of macrophage) in the skin. The Langerhans cells use Cyclooxygenase Type 1 (COX-1) for PGE2 production and are more responsible for acute flushing, while keratinocytes of the skin use COX-2 and are active in continued vasodilation. (Flushing was originally thought to involve histamine, but it appears now that histamine is not involved in the niacin reaction.)]