Healthy Outcomes
MEDICAL WEIGHT LOSS

OBESITY TREATMENT: TESTS USED TO ASSESS METABOLISM, APPETITE AND ENERGY CONTROL

FAT METABOLISM

Tests	Significance	Treatment
LDL (calculated by Friedwall formula) as in standard lipid panel	"Bad cholesterol". Independent risk factor for CVD. It can be elevated by genetic factors, high carb/high fat diet, high transfat diet, certain medications (e.g., beta-blockers, thiazides, estrogen, androgen), certain diseases (e.g., hypothyroidism, kidney disease, cystic fibrosis).	Carbohydrate restricted, low saturated fat/low transfat diet, healthy weight loss, exercise, target insulin resistance, smoking cessation, and medications when appropriate.
LDL Direct (directly measured, not calculated)	Calculated LDL that is often inaccurate, especially in situations when patient is not fasting, triglycerides are over 200 mg/dl, or LDL is less than 100 mg/dl. LDL value in standard lipid panel is calculated by a formula, but the error rate ranges from 7-41% based on published studies.	Carbohydrate restricted, low saturated fat/low transfat diet, healthy weight loss, exercise, target insulin resistance, smoking cessation, and medications when appropriate.
HDL/HDL Subclasses: **HDL-2:** most protective **HDL-3:** less protective	"Good cholesterol". Low HDL can be caused by genetics, ageing, high fat/refined carbohydrate diets, insulin resistance, over-weight/obesity, smoking, inactive lifestyle, certain illnesses (e.g., liver and thyroid disease, renal failure, diabetes), certain medications (e.g., beta-blockers, anabolic steroids).	Carbohydrate restricted diets, low saturated fat/ transfat diets, healthy weight loss, exercise, smoking cessation, target insulin resistance, medications when indicated: Niacin, Fibrates, Statins, Omega-3 supplements.
Triglycerides (TG)	Produced in the liver, mostly from excess carbohydrate and/or alcohol consumption. Main storage form of fat in the fat tissue. Independent predictor of CVD. High TG levels can interfere with leptin action in the brain (what can lead to increased food intake). High TG production can cause fat deposits in the liver (fatty liver) and pancreatitis. High TG can be caused by states of insulin resistance, diabetes, certain illnesses (e.g., hypothyroidism, renal failure, alcoholism, pancreatitis, and certain medications (e.g., beta-blockers, thiazides, glucocorticosteroids, oral contraceptives, anabolic steroids, HIV meds)	Carbohydrate restricted diet, Omega-3 supplements, medication changes when appropriate.
VLDL Calculated and **VLDL Direct**	Lipoprotein that mostly transports TG. Also considered atherogenic. VLDL Direct (not calculated, but measured) helps determine main source of high TG (chilomicrons vs VLDL) which helps guide treatment.	Carbohydrate restricted diet, Omega-3 supplements, medications when appropriate.
TG/HDL Ratio	Elevated TG/HDL ratio correlates with insulin resistance (IR): Normal: <3 IR: >5	Target insulin resistance. Carbohydrate restricted diet, exercise, healthy weight loss.

Apo-B/Apo-A1	ApoB is present in all the atherogenic lipoproteins (chilomicrons, LDL, IDL, Lp(a), VLDL). The Apo B: Apo A-I ratio appears to be superior to cholesterol measures and cholesterol ratios for predicting risk for myocardial infarction based on recent large case control studies. Even in the setting of normal cholesterol values, people can have high number of atherogenic lipoproteins. ApoB traverses in the blood in a 1:1 ratio with all the atherogenic lipoproteins (chilomicrons, LDL, IDL, lp(a), VLDL), and therefore is an indicator of number of atherogenic lipoproteins in the blood, even when individual lipoprotein values are normal.	Decreasing Apo B:Apo A-I ratio can be achieved by lowering Apo B and/or increasing Apo A-I. Statins, fibrates and niacin can lower Apo B. Exercise, fish oil, niacin and alcohol consumption in moderation can increase Apo A-I.
LDL-P/HDL-P	This test determines the particle numbers of LDL and HDL. Abnormal particle numbers/concentrations are associated with increased CV risk, even in the setting of normal cholesterol and normal LDL values. Cholesterol travels in the blood in the form of lipoproteins. LDL is a type of lipoprotein that carries mainly cholesterol. It is well known that LDL particles vary in size. The smaller the LDL particles, the more atherogenic they are. For any given LDL value, the smaller the LDL particles are, the higher the LDL-P (LDL particle number) there is. Therefore, a high LDL-P reflects higher number of smaller LDL particles.	Statins, fibrates and niacin can lower LDL-P. Exercise, fish oil and alcohol consumption in moderation can increase HDL-P. Some studies have shown that low carbohydrate diets, and targeting insulin resistance, as well as the use omega-3 supplements can favorably affect the size of LDL and HDL particles.
Free Fatty Acids (FFA)	FFA can be released from the adipose tissue (to be used as a source of energy) or produced and released by the liver in response to high carbohydrate and/or alcohol intake. FFA levels can increase in setting of starvation, prolonged fasting, ketogenic diets, adipose tissue insulin resistance, obesity, and high fat or high carbohydrate/alcohol intake. Lipoactive hormones such as epinephrine, norepinephrine, glucagon, thyroid hormone, adrenocorticotropin can induce release of FFA. Elevated FFA can lead to metabolic syndrome, development of diabetes, insulin resistance (in muscle and liver), stimulate triglyceride/VLDL production, decrease HDL, stimulate vasoconstriction, increase sodium reabsorption, promote inflammation, decrease nitrous oxide (leading to endothelial dysfunction), and increase free radical formation.	Carbohydrate restricted diet. Avoid high-fructose foods and beverages. Limit alcohol. Of note, ketogenic diets can cause a transient increase in FFA.

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Fatty Acid Profile	Blood levels of specific fatty acids provide important information on fat, cholesterol, and carbohydrate metabolism, inflammation risk, oxidative stress risk, and overall cardiometabolic risk. High levels of blood saturated fat leads to elevated cholesterol production. In the setting of a low carbohydrate diet, high intake of saturated fat will not lead to high cholesterol production. High carbohydrate and alcohol intake can lead to overproduction of saturated fat. AA/EPA (arachidonic acid/ eicosapentanoic acid) ratio provides information on pro- or anti-inflammatory status in the body. Ideal AA/EPA ratio is 1.5. Average American AA/EPA ratio is about 20, consistent with an active pro-inflammatory state.	Carbohydrate restriction will prevent elevation of blood saturated fat levels. Mediterranean style diet and promotion of higher intake of polyphenols and natural antioxidants will help quell inflammatory state. Increase consumption of omega-3 (food and/or supplements). Decrease consumption of omega-6 (chicken, grain-based desserts, salad dressings, chips, pizza, yeast breads, pasta, fried potatoes, mayonnaise, eggs, popcorn, sausage, franks, bacon, and ribs). Use oils with higher concentration of monoun-saturated fats: olive, canola, macadamia nut, and pecan oil.
Omega-3 Index	Helps assess possible benefits of Omega-3s such as: modulation of inflammation, improved insulin resistance, improved CNS response to leptin, improved lipids (lower triglycerides, raise HDL, lower Lp(a), increase LDL particle size), reduced risk of CAD, arrhythmias and risk of sudden cardiac death, reduced hepatic steatosis, and potentially help with weight loss efforts and appetite control. Low omega-3 index has been linked to higher risk of CV events, mainly sudden cardiac death, as reported in the GISSI-P study, JELIS trial, and the US Physician's Health Study.	Omega-3 supplements with proper concentration of DHA and EPA. Increase consumption of Omega-3-rich seafood: Atlantic salmon, Atlantic herring, pickled herring, bluefin tuna, chum salmon, coho salmon, mackerel (canned), oysters (steamed), sardines (canned in oil).