SYNDROME X NOTES

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THE SYNDROME

Syndrome X = Cell level insulin resistance with compensatory hyperinsulimenia.

Its clinical manifestations include:

- abdominal obesity
- elevated triglycerides
- decreased HDL cholesterol
- hypertension
- atherosclerosis and related diseases
- thrombotic diseases
- abnormal glucose tolerance (including true hypoglycemia)
- Type II diabetes

and may also include:

- osteoporosis
- clinical depression
- cognitive problems
- Alxheimer's disease
- erectile dysfunction

All the above may be related to either deficits in cell level function due to insulin resistance and the failure of insulin to facilitate the entrance of nutrients into the cell OR The pharmacological and metabolic effects of elevated insulin on the vascular system, the hormonal system, or on tissues which have not become insulin resistant. A silent condition that affects 60% or more of the U.S. population, while only about 15% have altered blood glucose tolerance or NIDDM.

INSULIN

Insulin is the body's chief anabolic hormone. It promotes the storage of nutrients in the cell, including glucose, free fatty acids, and amino acids. When dominant, it prevents the breakdown of fat and protein. The metabolism of some minerals is also affected by insulin resistance, notably chromium and magnesium, which cannot properly enter insulin resistant cells.

INSULIN RESISTANCE

The cell does not respond to normal levels of insulin. This can come from a number of factors:

CAUSES:

- Foods with high glycemic index.
- Low fat diets (fat lowers the glycemic index of a meal by delaying stomach emptying.)
- Elevated omega 6 - omega 3 ratio in the cell membranes
- Deficiencies of chromium, magnesium. These deficiencies follow a vicious cycle pattern. Deficiencies cause insulin resistance, and insulin resistance frutstrates their storage in the tissues.
- Other deficiencies: zinc, manganese, and B-vitamins
- Sedentary lifestyle. Insulin resistance in the liver increases after five days of no exercise. Trained muscle does not require insulin for the uptake of blood glucose, so lack of trained muscle mass in the body promotes systemic insulin resistance.

- Insulin. Elevated insulin promotes insulin resistance in a vicious-cycle relationship. The cell exposed to elevated insulin 'down-regulates' and reduces the number of its insulin receptors.
- Obesity. Another vicious cycle relationship. Insulin resistance promotes elevated insulin which prevents breakdown of fat and promotes obesity, while percentage of body fat increases and this in turn promotes insulin resistance.

EFFECTS

Important point: all the tissues do not become insulin resistant at once. The liver, which is exposed to insulin rich portal blood, becomes resistant first. Later, muscle and / or fat cells may become resistant, platelets, etc. Epithelial cells do not become insulin resistant. Effects depend on the tissue involved. In general the metabolism or function of the insulin resistant cells is deranged in some way. :

- - The insulin resistant liver cell, which should turn off glucose release in response to insulin does not do so. The hepatocyte also becomes resistant to the entrance of magnesium, thus creating a relative magnesium deficiency, which can alter the Phase I detoxification pathway. The insulin resistant hepatocyte also does not store blood glucose as glycogen efficiently when it is insulin resistant, and the blood glucose is shunted toward fat storage.
- - The insulin resistance muscle cell has trouble developing muscle mass because nutrients cannot enter it effectively. Insulin should be promoting the entrance of amino acids and block the breakdown of protein, but its ability to do either is hindered.
- - The insulin resistant fat cell continues to break down fats into triglycerides, which should be turned off by insulin. The triglycerides cannot be utilized, however, by other insulin resistant tissues, so blood triglycerides become elevated.
- - The insulin resistant platelet is also magnesium resistant. Deficient magnesium promotes 'stickiness" and the blood develop a tendency to clot easily.
- - The insulin resistant bone cells lose the protective effects of insulin, which normally reduce the breakdown of bone and promote collagen formation. Osteoporosis may be the long term result.

HYPERINSULINEMIA

As a mass of tissues becomes insulin resistant, uptake of blood glucose becomes more difficult, and more elevated and prolonged blood glucose curves result. The pancreas, in response, puts out progressively more insulin until the nutrients are effectively stored. Because the insulin output is not only elevated, but prolonged in time, the total hours of insulin exposure in the body may become elevated by 50-100%

Example: a normal and abnormal insulin response to a blood glucose challenge might be as follows.

Normal	**Syndrome X**
Fasting 10 microunits	40
1 hour 90	250
2 hours 80	240
3 hours 50	190
4 hours 10	90
5 hours 10	40

Both patients maintained a normal blood glucose throughout, but the patient with hyperisnulenimia is exposed to high levels of insulin around the clock, and greatly elevated levels after meals, and the post-meal curves last longer. Patient 2 is the typical curve for an obese patient.

EFFECTS:

- Hyperinsulinemia disrupts sodium metabolism and promotes water retention.
- May directly promote hypertension.
- Promotes growth of non-insulin resistant epithelial tissues. May promote growth on any epithelial-derived cancers. Promotes growth of atherosclerotic plaque
- May increase oxidative load in some tissues, promoting oxidative damage and initiation of atherosclerosis and neoplasia.
- Hyperinsulinemia decreases the total daily secretion of growth hormone, with ill effects throughout the body. One notable effect is depressing the conversion of T-4 to T-3 creating 'functional hypothyroidism" with normal circulating blood levels of the hormones.
- Hyperinsulinemia promotes rapid and dramatic lowering of blood sugar, with increased secretion of the counter hormones of cortisol, glucagon, growth hormone, and insulin-like growth factor. Chronic hyperinsulinemia may be accompanied by chronic compensatory hypercortisolemia as well, with poor tolerance of stress, depressed immunity, and, eventually, adrenal exhaustion.

DIAGNOSIS OF SYNDROME X

- Definitively diagnosed by measures of fasting insulin, or insulin changes to glucose challenge. Fasting levels should be below 10 units of insulin (though labs lists below 20 as "normal" Most labs and physicians will not run these tests. Most labs cannot run an insulin test. The blood sample must be shipped to a lab on dry ice.
- May be diagnosed clinical by abdominal obesity. For a male, waist circumference at the navel is greater than the widest part of the hips. For a female the circumference at the navel is more than 80% of the widest part of the hips.
- Another good indicator is the triglyceride level on routine blood tests, which tends to closely follow the insulin level.

TREATMENT

The treatment has three legs, all of which must be done simultaneously:

- Supplement the nutritional factors whose deficiencies lead to insulin resistance.
- Enagage in a minimum of 15-20 minutes of resistance-type exercise most days.
- Eat a low carbohydrate diet.

SUPPLEMENTS

The "oil change" to restore 6:3 ratios to the area of 2:1 or 3:1 in tissues. Accomplishing this require great restriction of 6 and high intake of 3, in the form of regular fish consumption, and supplementation with fish oils and flax oil Supplements must be given in greater than the RDA range in order to replenish tissue reservoirs.

- 60 mg Zinc
- 800 mg magnesium
- 600-800 mcg chromium
- B-100 complex, complete
- Lots of antioxidants: Vitamin C, E, etc
- L-carnitine, from 500-4000 mg

EXERCISE

Should be muscle-building exercise that mimics the effects of regular manual labor, such as humanity has always engaged in. Military type calisthenics, weight programs at the gym, etc. Running, cycling, etc is OK provided it gets into the anaerobic phase, such as running or cycling uphill, or doing training intervals at a race pace.

LOW CARBO DIET.

These are now made famous through various fad diets, proving that this portion alone can have some effect on insulin levels and resulting weight loss. This alone does not correct underlying nutrient deficiencies or the ill effects of sedentary lifestyle.
See the diet handout from Dr Rosedale, which includes both the low carbohydrate portion and the "oil change." The diet should also be -very- high in vegetables, fiber, and dietary antioxidants.

RESULTS

Results of the above treatments are usually evident within one week.

- - rapid loss of water weight
- - rapid reduction of hypertension
- - sustained weight loss of * - 1 lb per week
- - reduced food cravings and appetite
- - improved mental clarity
- - improved energy level, steadier throughout the day.
- - better tolerance of stress.
- - radically improved glycemic control in diabetics. (One patient brought 2-hour post-meal blood glucose levels from 225+ down to the 90s within 8 days. A type I diabetic brought insulin requirements from 25+ to below 5 in 3 weeks.)
- - No long term clinical trials have been done, but, presumably, reduced risk for the chronic diseases of civilization.

Note: The transition to the low carbohydrate diet is difficult, both because of dietary habits and also because the body is shifting its metabolism toward burning fat. This can be depressing for 2-3 weeks

MM's daily personal protocol

High protein, low carbo diet.
45 minutes swimming

Multi
Vitamin C - 1.5 grams
Vitamin E - 800 i.u.
Magnesium - 800 mg
Selenium - 50 mcg
Chromium picolinate - 200 mcgs
Fish oil - 3 gms
Innositol hexaniacinimide - 2 gm
Co-Q10 - 200 mg.
Garlic extract - 400 mgs
Commiphora mulmul standardized - 2 gm.
Coleus forskohlii standardized - 2 gm
Red Yeast Rice - 400 mg
Gingko standardized - 200 mg
Serenoa standardized - 1 gm