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The Herbalist's Corner

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TREATING METABOLIC SYNDROME (TYPE II DIABETES)

Metabolic Syndrome describes a cascade of symptoms and physiological presentations due to elevated insulin and glucose in the blood. It is synonymous, or at least directly leads to, Diabetes Type II.¹ It's important to know the difference between Type I and Type II diabetes. Both show increased blood glucose, but for different reasons. Type I diabetes is due to a genetic or autoimmune process that destroys the pancreatic beta cells that produce insulin, and therefore the elevated blood glucose levels are due to an absence of insulin, the chemical that transports glucose into cells. In diabetes Type II, blood sugar is elevated because of the inability of cells to recognize insulin, with consequential accumulations of sugar and insulin in the blood. It corresponds directly with weight gain and obesity.

Metabolic Syndrome is a direct consequence of diet, specifically intake of large amounts of refined carbohydrates and sugars. The American diet has been rich in sugar and breads since 1945, but the blame of the increase in obesity and Metabolic Syndrome lies our heavy consumption of sugars and corn syrup. In 1999, per capita intake of sugars was 158 pounds per person, 30 percent higher than in 1983.² Since introducing high fructose corn syrup in 1980, obesity rates have doubled.³ Corn syrup sugars more readily turns to fats in the liver, raising triglyceride and LDL levels, and it can affect the hormone leptin, which regulates appetite and body weight.⁴ It is now estimated that 24% of America's population is at risk for Metabolic Syndrome and Type II diabetes.⁵

Physiologically, Metabolic Syndrome occurs because of excessive amounts of carbohydrates (including sugar) in the diet. The pancreas secretes insulin when glucose is present in the blood, in order to transport it to the cells of the body. (Glucose is the main source of cellular energy, being used to make ATP.) In our evolutionary development as hunter-gatherers over the last million years or so, humans ate a diet of animal protein, nuts, seeds, fruits and berries, roots, and digested contents within animal guts. This is how our bodies developed, and actually is the diet best suited to our evolutionary origins. It is only the last 12,000 years or so that humans expanded their

diets to include, first, dairy from domesticated herds, and then to agricultural cultivation of grains, specifically rice or wheat. Even so, we did not see the advent of Type II diabetes until this past century when two events occurred. First was the creation of labor saving devices involving electricity and gasoline that allowed Western societies to lead a more sedentary lifestyle. The second was the increased consumption of animal fat, breads and sugars which increased dramatically after World War II. The average weight in 1950 in men was around 150 lbs, moving up to 200 lbs fifty years later.

The increased dietary load placed more glucose into the blood, and more demand to create insulin to manage the glucose load. Levels of insulin in someone fasting should actually be 2 uU/ml. although no one seems to have this low level. Medical doctors consider fasting insulin to indicate Type II diabetes if it is greater than 15 units, but those in Functional Medicine define Metabolic Syndrome at 6 uU/ml.

Elevated insulin is not good for the body. My view, based on scientific evidence, is that insulin itself is harmful to the interior epithelial lining of blood vessel walls.⁶ Insulin acts like a solvent: it tells cell walls to open up, that glucose is coming in. Left in the blood stream, however, it can damage and inflame the interior walls of blood vessel. When the body goes to heal and repair this inflammation, LDL (low density lipid) cholesterol is trapped in the repair. This is the main reason arteriosclerosis happens with LDL cholesterol. Without elevated insulin, however, I do not believe that LDL cholesterol can cause arterial plaquing. While doctors are busy chasing LDL cholesterol with statin drugs, it would be more important to evaluate and lower blood insulin levels. (Glucophage, which lowers blood sugar, also lowers blood insulin). Why does the body allow elevated insulin to stay in the blood? Because if the cells have glucose, they signal the cell walls to ignore insulin. This is called insulin resistance. The origin of this problem, of course, is over-abundance of glucose in the diet. (All refined carbohydrates break down into glucose).

The damaging effect of blood insulin – ignored in conventional medicine - would explain why Type I diabetics have so many problems as their disease progresses. The consequences in Diabetes Type I – heart and kidney disease, gangrene in the limbs, diabetic retinopathy - may not be due to diabetes per se, but to excessive amounts of insulin in their systems⁷. Type I diabetics typically take 60-80 combined units of insulin a day. With diet control, they could reduce that to 20 units a day.

The second consequence of Metabolic Syndrome, following elevated blood insulin, is elevated blood glucose. This causes sticky blood, much the same as adding sugar to water. The stickiness correlates to many disease processes, including heart disease, atherosclerosis and senility. In Chinese medicine, sticky blood implies blood stagnation, which inhibits organ function to the heart, kidneys and brain. It is involved in many of the health consequences we associate with aging.

Type II Diabetes, and Metabolic Syndrome, can be controlled by diet. Excess weight is clearly correlated to Type II Diabetes. (A typical sign would be to have a belt size less than half of one's height.) The best approach is the Paleolithic diet, one absent of all refined carbohydrates, dairy and sugars.⁸ This diet - animal protein, vegetables, fruits and nuts - allows fairly quick weight loss, perhaps 13 lbs. per month. Once the diet has reduced weight, one can add in rice, and then one essentially has a typical Chinese diet

In China, control of diet has become an important directive in the management of patients. From a modern TCM text describing Type II Diabetes, the following advice appears: "The (obese) patient represents the most frequent and important challenge of the physician. Treatment requires an energetic, vigorous program directed by persons who are aware of the mechanisms by which weight reduction is known to effectively lower hyperglycemia, and who are convinced of the profoundly beneficial effects of weight control on blood lipid levels and hyperglycemia in obese patients. Weight reduction is an elusive goal that can only be achieved by close interaction and communication with the obese patient."⁹

Treatment with Chinese Herbal Medicine.

While Diabetes Type I has been reported in China as far back as Zhong Zhongjing's time (3rd century C.E.), Type II diabetes is a modern phenomenon. Current teaching materials from China, however, have recognized it as a growing health problem in China, and are seeking to address it with Traditional Chinese Medicine.

A recent TCM text distinguishes seven differentiations for Type II diabetes: (1) deficiency of *yin* with heat; (2) deficiency of *qi* and *yin*; (3) deficiency of spleen *qi* with accumulation of heat; (4) deficiency of spleen *qi* with accumulation of damp; (5) deficiency of *qi* and blood; (6) deficiency of kidney *jing*; and (7) stagnation and accumulation of phlegm.¹⁰ These differentiations all have their own individualized

formulas, and the best approach with a Type II diabetic is individualized and customized treatment. For the general public, however, the authors recommend to popular patent medicine for the general public, *Yu Quan Wan*.

This prescription was originally formulated for *xiao ke*, “emaciation-thirst disease”, which corresponds to our understanding of Type I diabetes. There is a history to this formula. Andrew Ellis sites the origin in *Zhu Danxi’s Essential Teachings (dan xi xin fa)*, compiled in 1481, with later modifications by Ye Tianshi in 1746 (*wen re lun*, Epidemic Fevers Treatise).¹¹ A third variation appears with Shen Jin-ao in 1773.¹² In any event, the formula addresses the presentation of extreme thirst due to deficiency of *yin* with preponderance of heat. The formula is composed of the following: *sheng di huang* (Radix Rehmanniae Glutinosae), *mai men dong* (Tuber Ophiopogon), *ren shen* (Radix Panax Ginseng), *gua lou ren* (Semen Trichosanthis), *ge gen* (Radix Puerariae), *huang qi* (Radix Astragalus), *wu wei zi* (Fructus Schisandrae chinensis), *fu ling* (Sclerotium Poria Cocos), *wu mei* (Fructus Pruni mume), and *zhi gan cao* (Radix Glycyrrhizae uralensis prep). This prescription is available from the Taiwan extract manufacturers, and the original formula or variations of it are available as patent medicines from at least eight different companies.¹³

Extreme thirst and emaciation are not characteristic of Type II diabetes, yet the formula is being recommend for this type. Numerous rat and mice studies show that it effectively reduces blood glucose levels whether thirst is a symptom or not. ¹⁴ As an herbalist, two aspects about this formula are intriguing. First of all, the formula uses moistening herbs to thin the blood to reduce blood stickiness, rather than using blood moving herbs. These are *ge gen*, *mai men dong* and *sheng di huang*. As a treatment strategy, this may be useful for other “sticky blood” presentations. Thick blood viscosity can damage blood vessel walls and cause heart attacks. Secondly, I also wonder if the sour astringent herbs *wu wei zi* and *wu mei* have a benefit because, in the classical sense, they counter the sweet nature of sugar when addressing elevated blood glucose.

In any event, modern studies support that fact that this classical prescription, formulated to address TCM signs and symptoms, is effective within the paradigm of Western scientific analysis. Is there a place for *Yu Quan Wan* in a combined approach for Metabolic Syndrome patients in the West? This remains to be seen. I would like to see studies that demonstrate, beyond lowering blood glucose levels, whether the formula is

also effective in reducing insulin resistance at the cellular level, or that it reduces blood insulin levels. Clinically, I think that diet should be the first and foremost therapy, but using the formula *Yu Quan Wan* might prove a valuable adjunct.

¹ For a medical overview of Metabolic Syndrome, see See:
http://www.medscape.com/viewarticle/567635_print

² Center for Science in the Public Interest. http://www.cspinet.org/new/sugar_limit.html

³ San Francisco Chronicle, February 18, 2004. <http://www.sfgate.com/cgi-bin/article.cgi?f=/chronicle/archive/2004/02/18/FDGS24VKMH1.DTL>

⁴ Ibid. Also, an interesting discussion of fructose versus other sugars can be found at:
<http://www.westonaprice.org/modernfood/highfructose.html>

⁵ “The age-adjusted prevalence of the metabolic syndrome for adults is 23.7 percent.”
 American Heart Association. See:
<http://www.americanheart.org/downloadable/heart/1081492779297FS15META4.pdf>

⁶ Erzin and Kowalski, *The Type 2 Diabetes Diet Book*, McGraw-Hill, 1999.

⁷ Before the arrival of insulin after 1928, the real consequence of Diabetes Type I was an early death from cellular deprivation of glucose. No one survived long enough to demonstrate diabetic blindness, heart disease, etc.

⁸ See Loren Cordain, *The Paleo Diet*, Wiley Press, 2002.

⁹ From: *Traditional Chinese Treatment for Senile Diseases*, Academy Press [Xue Yuan] Beijing, 1997; Chapter 3, p.

¹⁰ Wang Xu and Yang Yaping, *Typical TCM Therapy for Diabetes Mellitus*, Shanghai University Of Traditional Chinese Medicine Press, 2004, p. 56.

¹¹ Andrew Ellis, *Notes from South Mountain*, Thin Moon Publishing, Berkeley, 2003, p. 393.

¹² *Yu Quan Wan*, from *Wondrous Lantern for Peering into the Origin and Development of Miscellaneous Diseases, za bing yuan liu xi zhu*, 1773. Sited in *Commonly Used Chinese Herb Formulas, Volume 1*. Hong-Yen Hsu, Chau-Shin Hsu. Revised edition by Qing-Fu Hu. Oriental Healing Arts Institute, 2006, p. 437.

¹³ See Jake Fratkin, *Chinese Herbal Patent Medicines, The Clinical Desk Reference*, Shya Publications, 2001, Chapter 11B.

¹⁴ Hsu and Hu, p. 438.