**Abstract poster Endo 2008**

**A-Reactive Hyperinsulinemia: a disease that should be treated**

Hyperinsulinemia is well known to promote conversion of energy intake into fat energy stores, as we have previously demonstarted (1).We propose the term Reactive Hyperinsulinemia (RH) be used as the name of a disease with typical features characterized by -cell hyperplasia oversecretion of insulin, increased appetite and cravings for sweets, acanthosis nigricans and skin tags, abdominal adiposity overweight/obesity, elevated triglycerides and hepatic enzymes and low HDL-C. . In a good number of patients the disease reverts when intake of Carbohydrates (CHO) is restricted to 100 130 g per day. Plasma insulin and triglyceride levels decrease and HDL-C increases. Liver enzymes normalize (Table 1). The concept implies that in a predisposed population of subjects, excessive CHO intake induces hyper secretion of insulin. Their predecessors ancestral hunters, thousands of years ago, survived on a low CHO intake (2). By having a continuous source of glucose coming from unopposed gluconeogenesis and lipolysis characterized by insulin insensitive enzymes, glucose and energy supply was thus guaranteed on a diet consistent of meat and vegetables. R H stimulates hepatic triglycerides (TG) synthesis which ends up packed as VLDL for its transport in blood and its eventual deposition as fat. The TG-rich lipoproteins lower HDL-C. The non-ketogenic Low CHO diet induces weight reduction as long as insulin levels are decreasing. When the diet alone is incapable of reducing insulin Metformin in doses of up to 3000 mg/d will ensure final control. The disease reappears when intake of CHO returns ad lib, thus indicating a causal relationship between the load of CHO intake and RH. Over the years and as -cell function diminishes, Diabetes Mellitus type 2 ensues. Hence the importance of diagnosing and treating Reactive Hyperinsulinemia.

**References**:

1- Torbay N et al. Am J Physiol 1985 Jan; 248 (1 pt 2): R 120-4
2- W. Koop: Metabolism 52, 7:840-844. 2003

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| Change in metabolic markers 6 months after a non-ketogenic low carbohydrate (NKLC) diet: |
| METABOLIC MARKERS | BASELINE | 6 MONTHS AFTER NKLC DIET |
| Total Cholesterol (mg/dl) | 190.71 http://www.marathonmultimedia.com/graphics/alphabet/plusmn.jpg8.86 | 178.79 http://www.marathonmultimedia.com/graphics/alphabet/plusmn.jpg12.42 |
| HDL-C (mg/dl) | 44.86 http://www.marathonmultimedia.com/graphics/alphabet/plusmn.jpg2.83 | 48.71 http://www.marathonmultimedia.com/graphics/alphabet/plusmn.jpg3.84 |
| LDL http://www.marathonmultimedia.com/graphics/alphabet/ndash.jpgC (mg/dl) | 123.43 http://www.marathonmultimedia.com/graphics/alphabet/plusmn.jpg7.94 | 114.57 http://www.marathonmultimedia.com/graphics/alphabet/plusmn.jpg9.01 |
| Triglycerides (mg/dl) | 177.00 http://www.marathonmultimedia.com/graphics/alphabet/plusmn.jpg24.55\* | 125.57 http://www.marathonmultimedia.com/graphics/alphabet/plusmn.jpg17.23\* |
| FBS (mg/dl) | 102.29 http://www.marathonmultimedia.com/graphics/alphabet/plusmn.jpg4.61\* | 90.59 http://www.marathonmultimedia.com/graphics/alphabet/plusmn.jpg2.78\* |
| Insulin (mIU/dl) | 31.14 http://www.marathonmultimedia.com/graphics/alphabet/plusmn.jpg7.03\* | 11.64 http://www.marathonmultimedia.com/graphics/alphabet/plusmn.jpg1.78\* |
| SGPT (UI/L) | 49.71 http://www.marathonmultimedia.com/graphics/alphabet/plusmn.jpg12.14 | 26.29 http://www.marathonmultimedia.com/graphics/alphabet/plusmn.jpg3.84 |
| SGOT (UI/L) | 30.00 http://www.marathonmultimedia.com/graphics/alphabet/plusmn.jpg8.29 | 16.60 http://www.marathonmultimedia.com/graphics/alphabet/plusmn.jpg1.29 |

n=17 Private Clinic Patients. All used same private lab \*Values in the same row are significantly different at p<0.5

**B- selecting a low fat vs. a low carbohydrates diet in obese/overweight subjects**

Variable weight reduction methods have failed to show consistent and reproducible outcomes on all obese/overweight subjects. We hypothesized that these patients could be divided into two groups for dietary prescriptions depending on their metabolic response to carbohydrates (CHO). The methodology here presented has been clinically tested in over 25.000 patients over the past 11 years, and early results have been previously published (1).It has been modified along the years, and the one here described has been tested for the past 3 years. The principle of the method is to divide the obese/overweight subjects into two groups: hyperinsulinemic (HI) and normoinsulinemic (NI). The hyperinsulinemic patient is prescribed a low carbohydrate (non-ketogenic) (LC) diet while the normoinsulinemic receives a low fat (LF) diet. To diagnose H I we use the HOMA2 (computerized version) and in addition the 1 hour post glucose insulin level. By doing so it becomes evident that we are dealing with two different types of patients. The group with insulin oversecretion has significantly higher weight, BMI, FBS, fasting insulin, HOMA, triglycerides, SGPT and uric acid and lower HDL-C

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| Table 1- Blood Variables in HI vs. NI Obese Subjects |
| Diet Assigned | Low CHO (n=123) | Low Fat (n=68) |
| BMI | 37.81 http://mail.google.com/mail/?ui=2&ik=5425238ede&attid=0.1&disp=emb&view=att&th=118b12a9fd0308190.25 | 38.58 http://mail.google.com/mail/?ui=2&ik=5425238ede&attid=0.1&disp=emb&view=att&th=118b12a9fd0308190.49 |
| FBS | 95.80 http://mail.google.com/mail/?ui=2&ik=5425238ede&attid=0.1&disp=emb&view=att&th=118b12a9fd0308191.19\* | 85.85 http://mail.google.com/mail/?ui=2&ik=5425238ede&attid=0.1&disp=emb&view=att&th=118b12a9fd0308191.19\* |
| Insulin | 22.88 http://mail.google.com/mail/?ui=2&ik=5425238ede&attid=0.1&disp=emb&view=att&th=118b12a9fd0308191.19\* | 9.30 http://mail.google.com/mail/?ui=2&ik=5425238ede&attid=0.1&disp=emb&view=att&th=118b12a9fd0308190.26\* |
| HDL-C | 49.41 http://mail.google.com/mail/?ui=2&ik=5425238ede&attid=0.1&disp=emb&view=att&th=118b12a9fd0308190.98\* | 53.53 http://mail.google.com/mail/?ui=2&ik=5425238ede&attid=0.1&disp=emb&view=att&th=118b12a9fd0308191.55\* |
| Triglycerides | 184.87 http://mail.google.com/mail/?ui=2&ik=5425238ede&attid=0.1&disp=emb&view=att&th=118b12a9fd03081911.16\* | 145.47 http://mail.google.com/mail/?ui=2&ik=5425238ede&attid=0.1&disp=emb&view=att&th=118b12a9fd03081911.25\* |
| SGPT | 45.44 http://mail.google.com/mail/?ui=2&ik=5425238ede&attid=0.1&disp=emb&view=att&th=118b12a9fd0308194.59\* | 29.15 http://mail.google.com/mail/?ui=2&ik=5425238ede&attid=0.1&disp=emb&view=att&th=118b12a9fd0308192.52\* |
| Uric Acid | 6.18 http://mail.google.com/mail/?ui=2&ik=5425238ede&attid=0.1&disp=emb&view=att&th=118b12a9fd0308190.14\* | 5.46 http://mail.google.com/mail/?ui=2&ik=5425238ede&attid=0.1&disp=emb&view=att&th=118b12a9fd0308190.29\* |

\*Values in the same row with superscript are significantly different at p< 0.5

. This difference is independent of weight, because if the two groups are made to equal BMI the differences remain significantly higher in the HI group.

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| FBS | 95.80 http://mail.google.com/mail/?ui=2&ik=5425238ede&attid=0.1&disp=emb&view=att&th=118b12a9fd0308191.19\* | 85.85 http://mail.google.com/mail/?ui=2&ik=5425238ede&attid=0.1&disp=emb&view=att&th=118b12a9fd0308191.19\* |
| Insulin | 22.88 http://mail.google.com/mail/?ui=2&ik=5425238ede&attid=0.1&disp=emb&view=att&th=118b12a9fd0308191.19\* | 9.30 http://mail.google.com/mail/?ui=2&ik=5425238ede&attid=0.1&disp=emb&view=att&th=118b12a9fd0308190.26\* |
| HDL-C | 49.41 http://mail.google.com/mail/?ui=2&ik=5425238ede&attid=0.1&disp=emb&view=att&th=118b12a9fd0308190.98\* | 53.53 http://mail.google.com/mail/?ui=2&ik=5425238ede&attid=0.1&disp=emb&view=att&th=118b12a9fd0308191.55\* |
| Triglycerides | 184.87 http://mail.google.com/mail/?ui=2&ik=5425238ede&attid=0.1&disp=emb&view=att&th=118b12a9fd03081911.16\* | 145.47 http://mail.google.com/mail/?ui=2&ik=5425238ede&attid=0.1&disp=emb&view=att&th=118b12a9fd03081911.25\* |
| SGPT | 45.44 http://mail.google.com/mail/?ui=2&ik=5425238ede&attid=0.1&disp=emb&view=att&th=118b12a9fd0308194.59\* | 29.15 http://mail.google.com/mail/?ui=2&ik=5425238ede&attid=0.1&disp=emb&view=att&th=118b12a9fd0308192.52\* |
| Uric Acid | 6.18 http://mail.google.com/mail/?ui=2&ik=5425238ede&attid=0.1&disp=emb&view=att&th=118b12a9fd0308190.14\* | 5.46 http://mail.google.com/mail/?ui=2&ik=5425238ede&attid=0.1&disp=emb&view=att&th=118b12a9fd0308190.29\* |

\*Values in the same row with superscript are significantly different at p< 0.5

. Average weight loss in the H I group is -10.67 0.39 % of initial at 4 months (n=109). For the NI group the weight loss is -9.79 O.57 % of initial (n= 35) with no significant difference among the two groups. At the end of the weight loss period patients learn what macronutrient specifically makes them gain weight and maintenance of the lost weight becomes much easier.

**References:**

1- Baba NH, Sawaya S, Torbay N et al. Int J Obes Relat Metab Disord 1999 Nov, 23 (11):1202-6