

Geachte diëtist(e),

Uw klant gebruikt mijn boek SLIM – op uw juiste gewicht om gewicht te verliezen. In mijn boek behandel ik een aantal lichamelijke stoornissen die gewichtstoename kunnen veroorzaken zoals hypothyroïdie, hyperinsulinemie, een verlaagde verbranding enz. In mijn boek ga ik er van uit dat afvallen, door op voeding en beweging te letten, meer succes heeft als er ook aandacht is voor deze onderliggende stoornissen.

Door het toepassen van mijn differentiaal diagnostische model heeft uw klant **hyperinsulinemie** bij zich zelf herkend.

Hyperinsulinemie is, zoals u weet, een vast onderdeel van het metabool syndroom. Een verhoogde taille / heup ratio (vrouwen > 0,8 mannen > 1) is één van de kenmerken. Bij hyperinsulinemie zit het gewicht bij vrouwen voornamelijk rond de taille, mannen hebben een zogenaamde “bierbuik”. Bij hyperinsulinemie is de insulinespiegel chronisch verhoogd terwijl de bloedsuiker nog een normale waarde heeft^{1,2}. Insulineresistentie is de oorzaak van hyperinsulinemie³.

Recent Nederlands onderzoek geeft aan dat bij ongeveer één miljoen Nederlanders jonger dan 60 jaar de hormonale stoornis hyperinsulinemie voor komt¹⁷. Dat is 25% van alle Nederlanders onder de 60 jaar. Door de combinatie insulineresistentie en een verhoogde insulinespiegel worden de koolhydraten (glucose) uit voeding niet verbrand maar als vet opgeslagen^{4,5}. Dit gebeurt vooral na een koolhydraatrijke maaltijd⁹.

Het is daarom belangrijk dat uw klant geen koolhydraten vlak voor of na de training gebruikt. Optimaal zou zijn als uw klant vroeg in de morgen, voor het ontbijt als de insulinespiegel op zijn laagst is, bij een sportschool traint. Hierdoor wordt insulineresistentie gedurende de verdere dag verminderd waardoor de insulinespiegel wordt verlaagd^{6,7}. Bovendien heeft sporten vroeg in de morgen het voordeel van een verhoogde verbranding gedurende de rest van de dag⁸.

Verder adviseer ik uw klant in mijn boek om laag glycemische voeding (GI < 35) te kiezen en de hoofdmaaltijden wat kleiner te maken door laag glycemische (GI < 35) tussendoortjes in te voeren. Onderzoek bevestigt dat mensen die vaker kleinere porties eten minder snel honger hebben en daardoor ook minder eten^{10,11,12}. Hierdoor worden ook pieken van de suiker- en insulinespiegel voorkomen. Zou u uw klant kunnen helpen met het invoeren van deze voedingsinterventie.

Ik zou u willen vragen om bij uw klant het gebruik van toegevoegde fructose (natuurlijke vruchtensuiker) zo veel mogelijk te verminderen. Fructose verhoogt ook het risico op insulineresistentie^{13,14}, dit veroorzaakt hyperinsulinemie en overgewicht. Bovendien wordt de aan onze voeding toegevoegde fructose in verband gebracht met een hoger risico op het krijgen van het metabool syndroom^{15,16}.

Voor eventuele vragen over dit programma ben ik via telefoon en email voor u beschikbaar.

Met vriendelijke groet,

Cora de Fluiter

Orthomoleculair gewichtsconsulent

0591 – 853377 EXLOO

<http://www.coradefluiter.nl/>

cora@coradefluiter.nl

Referenties

Belangrijk: print deze referentielijst ook uit aan uw diëtist(e)

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A high plasma insulin concentration in the presence of a normal or high plasma glucose level appears to be a common feature of glucose intolerance, obesity, and hypertension.
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Resistance to insulin consists in a decrease in insulin's biologic action and is manifested mainly by hyperinsulinism.
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Lipogenesis is the process by which simple sugars such as glucose are converted to fatty acids. Insulin stimulates lipogenesis in three main ways.
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Our study suggests that acute exercise decreases insulin resistance at the first exercise session with no effect on leptin levels. Significant leptin decrement was evident at the first week and lasted during the entire four weeks exercise session.
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Favorable changes in glucose tolerance and insulin sensitivity usually deteriorate within 72 h of the last exercise session: consequently, regular physical activity is imperative to sustain glucose-lowering effects and improved insulin sensitivity.
<http://www.ncbi.nlm.nih.gov/pubmed/10912903>
8. [Speakman JR, Selman C. Proc Nutr Soc.](#) 2003 Aug;62(3):621-34 **Physical activity and resting metabolic rate**
Resting metabolic rate (RMR) is the largest component of the daily energy budget in most human societies and, therefore, any increases in RMR in response to exercise interventions are potentially of great importance. Long-term effects of training include increases in RMR due to increases in lean muscle mass. Many studies of human subjects indicate a short-term elevation in RMR in response to single exercise events (generally termed the excess post-exercise O₂ consumption; EPOC). This EPOC appears to have two phases, one lasting < 2 h and a smaller much more prolonged effect lasting up to 48 h.
<http://www.ncbi.nlm.nih.gov/pubmed/14692598>
9. [Brand-Miller JC, Holt SH, Pawlak DB, McMillan J. Am J Clin Nutr.](#) 2002 Jul;76(1):281S-5S **Glycemic index and obesity**

Many high-carbohydrate, low-fat diets may be counterproductive to weight control because they markedly increase postprandial hyperglycemia and hyperinsulinemia. Many high-carbohydrate foods common to Western diets produce a high glycemic response [high-glycemic-index (GI) foods], promoting postprandial carbohydrate oxidation at the expense of fat oxidation, thus altering fuel partitioning in a way that may be conducive to body fat gain.

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Obese males fed an isoenergetic pre-load sub-divided into a multi-meal plan consumed 27% less at a subsequent ad libitum test meal than did the same men when given the pre-load as a single meal. Prolonged but attenuated increases in serum insulin concentration on the multi-meal programme may facilitate this acute reduction in appetite.
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These data suggest that when the nutrient load was spread into equal amounts and consumed evenly through the day in lean healthy males, there was an enhanced control of appetite. This greater control of satiety when consuming smaller multiple meals may possibly be linked to an attenuation in insulin response although clearly both other physical (gastric stretch) and physiological (release of gastric hormones) factors may also be affected by the periodicity of eating.
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A protective effect of an increased daily meal frequency on obesity in children was observed and appeared to be independent of other risk factors for childhood obesity. A modulation of the response of hormones such as insulin might be instrumental.
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we have reported that consumption of a high-fructose diet, but not a high-glucose diet, promotes the development of three of the pathological characteristics associated with metabolic syndrome: visceral adiposity, dyslipidemia, and insulin resistance.
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Because leptin production is regulated by insulin responses to meals, fructose consumption also reduces circulating leptin concentrations. The combined effects of lowered circulating leptin and insulin in individuals who consume diets that are high in dietary fructose could therefore increase the likelihood of weight gain and its associated metabolic sequelae.
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There is much evidence from both animal models and human studies supporting the notion that fructose is a highly lipogenic nutrient that, when consumed in high quantities, contributes to tissue insulin insensitivity, metabolic defects, and the development of a prediabetic state. Recently evidence has helped to decipher the mechanisms involved in these metabolic changes.
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Emerging evidence suggests that increased dietary consumption of fructose in Western society may be a potentially important factor in the growing rates of obesity and the

metabolic syndrome.

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17. [Bos MB](#), [de Vries JH](#), [Wolffenbuttel BH](#), [Verhagen H](#), [Hillege JL](#), [Feskens EJ](#). [Ned Tijdschr Geneeskd](#). 2007 Oct 27;151(43):2382-8 **The prevalence of the metabolic syndrome in the Netherlands: increased risk of cardiovascular diseases and diabetes mellitus type 2 in one quarter of persons under 60**

Approximately 1 million Dutch adults below 60 years of age had the metabolic syndrome in the 1990's. Based on the total prevalence of the metabolic syndrome and hypercholesterolaemia, one quarter of the Dutch population younger than 60 runs an increased risk of cardiovascular disease and type 2 diabetes mellitus.

<http://www.ncbi.nlm.nih.gov/pubmed/18019216>