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Original

Effect of a low glycemic load on body composition and Homeostasis Model Assessment (HOMA) in overweight and obese subjects

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Abstract

Objective: **The aim of this study was to compare the effects of different glycemic load diets on biochemical data and body composition, in overweight and obese subjects, during a 6-month period.**

Research design and methods: **This study was an experimental, randomized, parallel design. Anthropometric measurements and biochemical data were measured at baseline at 3 and at 6 months. All subjects completed 3-day dietary intake diaries at the baseline period and during the third and the sixth months. At the sixth month, LGL group had a mean intake of 1,360 ± 300 kcal/day and the high glycemic load group (HGL) had a mean intake of 1,544 ± 595 kcal/day.**

Results: **LGL group obtained a weight reduction of 4.5% (p = 0.006) and the HGL group of 3.0% (p = 0.18). Significant reductions in waist circumference (5%, p = 0.001) of the LGL group were observed, 10% of body fat percentage (p = 0.001), 4.3 kg (13%) of body fat (p = 0.001), 14% of total cholesterol (p=0.007), 35% of high-density lipoproteins (HDL) (p = 0.001), and 10% of HOMA (p = 0.009). In the HGL group, reductions of 4.5% of waist circumference (p = 0.02), 37% of HDL (p = 0.002), and an increase of 8 % of LDL (p = 0.04) were observed.**

Conclusions: **These results suggest that long term LGL diets are more effective for reducing body mass index, body fat, waist circumference and HOMA and, therefore, may contribute in the prevention of diabetes.**

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Key words: *Glycemic load. Obesity. Overweight. Diabetes.*

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EFECTO DE LA BAJA CARGA CLUCÉMICA EN LA COMPOSICIÓN CORPORAL Y EL HOMA EN SUJETOS CON SOBREPESO Y OBESIDAD

Resumen

Objetivo: **Comparar el efecto de dietas con diferente carga glucémica en la composición corporal y los marcadores bioquímicos, en sujetos con sobrepeso u obesidad, durante un periodo de seis meses.**

Metodología: **Estudio experimental, aleatorio, paralelo. Se realizaron mediciones antropométricas y bioquímicas al inicio, a los tres y a los seis meses. Todos los sujetos realizaron un registro de alimentos de tres días, al inicio, a los tres y a los seis meses. A los seis meses, el grupo de dieta de baja carga glucémica (DBCG) tuvo una ingesta energética promedio de 1.360 ± 300 kcal/día, y el grupo con alta carga glucémica (DACG) de 1.544 ± 595 kcal/día.**

Resultados: **El grupo con DBCG obtuvo una reducción de peso del 4,5% (p = 0,006) y el grupo con DACG del 3,0% (p = 0,18). En el grupo con DBCG se observaron reducciones significativas en la circunferencia de cintura (CC) (5%, p = 0,001), 10% en el porcentaje de grasa corporal (p = 0,001), 4,3 kg (13%) de masa grasa (p = 0,001), 14% en el colesterol total (p = 0,007), 35% en las lipoproteínas de alta densidad (HDL) (p = 0,001), y un 10% en el HOMA (p = 0,009). En el grupo con DACG se observaron reducciones del 4,5% en la CC (p = 0,02), 37% en las HDL (p = 0,002), y un incremento en las lipoproteínas de alta densidad (LDL) del 8% (p = 0,04).**

Conclusiones: **Estos resultados sugieren que a largo plazo las DBCG son más efectivas en la reducción del índice de masa corporal, la grasa corporal, la CC y la sensibilidad a la insulina (HOMA), lo que puede contribuir en la prevención de la diabetes.**

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Palabras clave: *Carga glucémica. Obesidad. Sobrepeso. Diabetes.*

Abreviations

GI: Glycemic Index.
LGI: Low glycemic index.
LGL: Low glycemic load.
HGL: High glycemic load.
GL: Glycemic load.
CHD: Coronary heart disease.
TC: Total cholesterol.
HDL: High-density lipoproteins.
TG: Triglycerides.
BMI: Body mass index.
HOMA: homeostasis model assessment.
LDL: Low-density lipoproteins.
INUBAC: Instituto de Nutrición de Baja California.
WC: Waist circumference.
IPAQ: International Physical Activity Questionnarie.
RCT: Randomized controlled trials.

Introduction

Insulin resistance is one of the main effects on human health, resulting from weight gain, and plays a principal role in the development of the metabolic syndrome.[1] It is also an important risk factor in the development of type 2 diabetes mellitus and cardiovascular diseases.[2] Additionally, in a study by Oster et al.,[3] it was estimated that 85% of all cases of type 2 diabetes were attributable to overweight and obesity. Regular consumption of high glycemic index (GI) foods can also increase the risk for obesity, type 2 diabetes, cardiovascular diseases and some types of cancer.[4] Some studies have demonstrated the beneficial effect of low glycemic index (LGI) and low glycemic load (LGL) diets in subjects with diabetes[5-10] and hyperlipidemias.[11] The glycemic load (GL) concept encompasses the idea that the overall glycemic effect of a diet may be related to disease risks.[12] Glycemic load is calculated by multiplying the GI by the amount of carbohydrates consumed.[13] Van Dam et al.[14] carried out a study in elderly healthy men and did not observe an association between high GI diets and the incidence of coronary heart disease (CHD), total cholesterol (TC), high-density lipoproteins (HDL), triglycerides (TG), insulin or glucose. On the other hand, Ebbeling et al.[15] observed a decrease in body mass index (BMI), fat mass and a lesser increase in insulin resistance with LGL diets during a 3-month interventional period and 6 months follow-up. Furthermore, LGL diets have shown a decrease in fat mass and significant improvement in insulin sensitivity (HOMA) in obese subjects during a 36-week period.[16]

It has also been observed that, in the Mexican culture, after a 3-week intervention with appropriate diets in hyperlipidemic subjects, LGI diets decrease TC and low-density lipoproteins (LDL).[11] However, we have not found long-term studies analyzing the effect of LGL hypocaloric diets on body composition and insulin sensitivity. The aim of this study was to compare the effects of diets with different GL on body composition and biochemical markers in overweight and obese subjects during a 6-month period.

Research design and methods

Study procedure

The study is a randomized, experimental, parallel design conducted in a group of Mexican subjects during a 6-month period. Anthropometric and biochemical data were determined at 0, 3 and 6 months. The Ethical Committee of the Instituto de Nutrición de Baja California (INUBAC) and the Health Science Research Evaluation Committee at UABC approved the study.

Subjects

Fifty-four adults with overweight or obesity were assigned in two groups: 27 subjects with LGL diet and 27 with high glycemic load (HGL) diet. Subjects who were pregnant or had diabetes, cancer, psychiatric disorders or physical disabilities were excluded.

Diets and dietary assessment

Two diets were designed including LGL and HGL diets. GI values of each food were estimated from the tables by Foster-Powell et al.[17] Daily GL was determined by the product of total dietary carbohydrate (grams) and GI of each food and adjusted to energetic intake using the following formulas: GI = (GI of each food X proportion of total carbohydrate contributed from each food); GL= (GI of each food × grams of total carbohydrate from each food)/1,000 kcal.

Diets were designed according to the food habits of Mexicans living in the Tijuana area. On the first day, subjects received different menus of either LGL or HGL diets, according to the randomization. A research assistant was available by mail or by phone for questions during the 6-month period. E-mail, as a reminder and reinforcement to maintain the diet, was sent every 2 weeks to all participants. Subjects who completed the 3-day dietary record (two weekdays and one weekend day) were included in the analysis.

Anthropometric measurements

Height was measured to the nearest millimeter using a portable stadiometer (model 214 Road Rod, Seca Corp., Hanover, MD). Weight was measured to the nearest 0.1 kg using an electronic scale (Bod Pod, Life Measurement Inc., Concord, CA). Subjects were dressed in light clothing and were without shoes. BMI

was calculated with the following formula: weight (kg)/height (m^2). Waist circumference (WC) was measured at the minimum circumference between the iliac crest and the rib cage. Fat mass (in kg) and total body fat percentage were measured by plethysmography with the Bod Pod. The subjects entered the Bod Pod with bodysuit and Lycra hat.

Blood analysis

Venous blood samples were taken at 8 a.m. from an antecubital vein after a 12-h overnight fast, again at baseline and at 3 months after beginning the study. Blood samples were centrifuged at 3,500 × g for 3 min, and plasma was removed and analyzed immediately after collection. For quantitative determination of glucose in serum, the glucose oxidase procedure based on a modified Trindler method was used (SERA-PAK Plus, Bayer, Sées, France). Serum insulin levels were determined by chemiluminescent immunoassay by the IMMULITE 2000 analyzer (Diagnostic Products Corporation, Los Angeles, CA). Homeostasis model assessment (HOMA) was used to estimate insulin resistance and was computed as follows: (fasting serum insulin [μU/ml] × fasting plasma glucose [mmol/L]/22.5).[18,19] Total cholesterol, HDL-cholesterol and triglycerides were measured by enzymatic methods (SERA-PAK Plus, Tarrytown, New York); LDL-cholesterol was calculated using the Friedewald formula: LDL (mmol/L) = total cholesterol – (TG/2.2) – HDL.

Physical activity assessment

A questionnaire containing seven questions from the International Physical Activity Questionnaire (IPAQ)[20] was used to evaluate physical activity at baseline and at 1, 3 and 6 months after study initiation.

Statistical analysis

Sample size was calculated as N = 30 per group assuming 5 cm of WC at the end of the study with 80% of power and 5% of significance level. Mean ± standard deviations were calculated using descriptive statistics in all variables. Anthropometric measurements were evaluated, and it was determined that they did not meet the normality test (Kolmogorov-Smirnov and Shapiro-Wilk). To test differences between groups on biochemical and anthropometric measurements, Mann-Whitney non-parametric test for independent samples was performed. For comparison of differences before, at 3 and 6 months after intervention in the same treatment group, we used Friedman non-parametric test for repeated measurements. To test differences between groups on diet intake at baseline and at the end of the study, Mann-Whitney non-parametric test for independent samples was performed. All analyses were performed using SPSS software (version 11.5, Chicago, Illinois)

Results

Subject characteristics

At the beginning of the study, 54 subjects were recruited (36 females and 18 males). Baseline characteristics of the subjects are shown in table I. There were no

Table I
Baseline characteristics by treatment group

	LGL group (n = 27) *Mean ± SD (range)*	*HGL group (n = 27)* *Mean ± SD (range)*
Female	66.6%	67.8%
Age (years)	36.9 ± 9.0 (22-57)	33.8 ± 8.2 (21-53)
Weight (kg)	83.1 ± 12.6 (61-107)	86.0 ± 16.1 61-120)
Height (m^2)	1-64 ± 0.1 (1.5-1.8)	1.63 ± 0.1 (1.5-1.9)
BMI (kg/m^2)	30.7 ± 4.0 (24-42)	32.5 ± 5.9 (26-46)
WC (cm)	98.9 ± 8.5 (82-116)	102 ± 13 (82-135)
FM, plethysmography (%)	39.6 ± 7.9 (23-54)	40.5 ± 9.3 (22-59)
FM, bioelectric impedance (%)	35.8 ± 7.9 (19-49)	36.9 ± 9.7 (19-53)
FFM, plethysmography (kg)	50.1 ± 10.2 (37-69)	50.6 ± 9.1 (37-67)
FFM, bioelectric impedance (kg)	53.5 ± 11 (40-74)	53.9 ± 10.9 (39-74)
Abdominal, fat, bioelectric impedance (%)	34.7 ± 6.4 (21-48)	35.4 ± 7.8 (20-52)
Glucose (mmol/L)	4.9 ± 0.8 (2.3-6.8)	4.9 ± 0.6 (3.7-6.6)
TC (mmol/L)	5.5 ± 1.2 (3.7-8.6)	6.1 ± 3.0 (3.7-20)
HDL (mmol/L)	1.6 ± 0.4 (1.0-2.3)	1.7 ± 0.4 (1.1-2.3)
LDL (mmol/L)	3.8 ± 0.8 (2-6)	4.4 ± 2.7 (3-18)
Insulin (μU/ml)	9.4 ± 3 (4-16)	10 ± 4 (4-19)
TG (mmol/L)	2.1 ± 1.4 (0.7-5.4)	1.9 ± 1.2 (0.8-6.3)
HOMA	2.0 ± 0.6 (1.2-3.5)	2.1 ± 0.7 (1.0-3.7)

Values are expressed as mean ± SD (range).
LGL: low glycemic load; HGL: high glycemic load; BMI: body mass index; WC: waist circumference; FM: fat mass; FFM: fat-free mass; TC: total cholesterol; HDL high-density lipoprotein; LDL: low-density lipoprotein; TG: triglyceride; HOMA: homeostasis model assessment.

Table II
Kilocalories, macronutrients (mean ± SEM), GI and GL, consumption at baseline, and after 3 and 6 months

LGL	*Baseline n = 16*	*3 m n = 16*	*6 m n = 16*	*p**
Energy (kcal)	1,860 ± 670	1,295 ± 351	1,360 ± 300	0.04
CHO (g)	222 ± 78	156 ± 38	173 ± 41	0.03
Protein (g)	90 ± 32	66 ± 19	67 ± 18	0.04
Fat (g)	68 ± 32	44 ± 21	48 ± 22	0.44
Fiber (g)	21 ± 6	20 ± 7	23 ± 12	0.76
GI	57 ± 7.6	49 ± 7	51 ± 7[2]	0.007
GL	121 ± 68	58 ± 26[1]	76 ± 22[3]	0.004
HGL	*n = 8*	*n = 8*	*n = 8*	
Energy	2,012 ± 667	1,405 ± 382	1,544 ± 595	0.02
CHO (g)	270 ± 92	194 ± 67	197 ± 82	0.09
Protein (g)	80 ± 19	66 ± 17	82 ± 31	0.07
Fat (g)	65 ± 28	43 ± 16	49 ± 39	0.10
Fiber (g)	19 ± 13	17 ± 7	14 ± 7	0.20
GI	59 ± 6	54 ± 7	59 ± 5[2]	0.15
GL	120 ± 51	97 ± 55[1]	103 ± 45[3]	0.34

*Friedman. CHO = carbohydrates; GI = glycemic index; GL = glycemic load.
Differences between diets (LGL *vs*. HGL): [1]p = 0.044; [2]p = 0.008; [3]p = 0.032.

significant differences between groups in body composition, biochemical markers and nutrient intake variables.

Participation rate

Fifty-four subjects were analyzed at the beginning of the study. At 3 months, 33 (61%) subjects returned for anthropometric and biochemical measurements (18 with LGL diet and 15 with HGL diet), and at 6 months 24 subjects (44%) completed the dietary intake diaries (16 with LGL diet and 8 with HGL diet) and returned for anthropometric and biochemical measurements.

Diet composition

No significant differences were observed at baseline between groups in the consumption of kilocalories, macronutrients, GI and GL. After 3 and 6 months of intervention, both groups had a significant decrease in caloric consumption (table II). Significant reductions of energy, carbohydrate, protein, GI and GL intake were observed in the LGL group after the intervention, and reduction of energy in the HGL group (table II).

Body composition and biochemical data

At 6 months LGL group obtained a weight reduction of 4.5% ($P = 0.008$) and the HGL group of 3.0% ($P = 0.68$). Significant reductions in waist circumference (5%, $P = 0.001$) of the LGL group were observed, 10% of body fat percentage ($P = 0.002$), 4.3 kg (13%) of body fat ($P = 0.002$), 14% of total cholesterol (P=0.007), 35% of high-density lipoproteins (HDL) ($P = 0.0001$), and 10% of HOMA ($P = 0.009$). In the HGL group, reductions of 4.5% of waist circumference ($P = 0.07$), 37% of HDL (0.002), and an increase of 8 % of LDL ($P = 0.04$) were observed (Table 3).

Physical activity

There were no significant changes in physical activity at baseline or at any time point during the intervention.

Side effects

No side effects were observed with the diets.

Discussion

This study, conducted in overweight and obese men and women, showed higher beneficial effects of LGL hypocaloric diet controlled by macronutrient consumption and fiber on most body composition and biomarkers data when compared to the HGL diet. HDL was significantly reduced in both groups. A few randomized controlled trials (RCT), parallel or crossover, had been reported on the effect of GI on overweight or obese adolescents,[15] young adults,[21,22] and adults,[23] which assessed the diet on a duration ranging from 9

Table III
Body composition and biochemical data (mean ± SEM) before and after intervention

	Low glycemic load diet n = 16				*High glycemic load diet n = 8*			
	0	*3 m*	*6 m*	*p**	*0*	*3 m*	*6 m*	*p**
Weight (kg)	80.5 ± 12	78.4 ± 13	76.9 ± 13	0.008	89.2 ± 15	85.8 ± 14	86.8 ± 14	0.68
WC (cm)	97.4 ± 6.7	93.4 ± 8.6	92.2 ± 9.7	0.001	101.3 ± 8	98.4 ± 6.9	97.8 ± 7.5	0.07
BMI (kg/m^2)	30.1 ± 4	29.3 ± 4.4	28.8 ± 4.6	0.008	32.9 ± 5.2	31.8 ± 5.2	31.9 ± 4.9	0.68
FM (%)	39.3 ± 7.3	37.1 ± 8.2	35.3 ± 8.2	0.002	38.5 ± 7.9	36.7 ± 11	36.6 ± 8.8	0.41
FM (kg)	31.6 ± 8.1	29.2 ± 9.1	27.3 ± 9.2	0.002	34.9 ± 12	32.1 ± 14	32.1 ± 12	0.41
FFM (kg)	48.9 ± 10	49.1 ± 9.9	49.6 ± 10.3	0.21	54.3 ± 8.6	53.4 ± 9.3	54.4 ± 9.4	0.19
Glucose (mmol/L)	5.0 ± 0.9	4.9 ± 0.8	5.1 ± 0.6	0.79	5.2 ± 0.7	5.3 ± 0.5	5.3 ± 0.5	0.78
TC (mmol/L)	5.7 ± 1.3	5.2 ± 0.8	4.9 ± 0.7	0.007	5.7 ± 1.0	5.6 ± 1.1	5.3 ± 0.6	0.38
HDL (mmol/L)	1.7 ± 0.4	1.0 ± 0.1	1.1 ± 0.1	0.0001	1.9 ± 0.4	1.0 ± 0.1	1.2 ± 0.1	0.002
LDL (mmol/L)	3.8 ± 0.9	4.1 ± 0.9	3.8 ± 0.6	0.01	3.6 ± 0.9	4.5 ± 1.2	3.9 ± 0.6	0.04
TG (mmol/L)	2.1 ± 1.6	1.6 ± 1.0	1.6 ± 0.9	0.15	2.7 ± 1.8	2.4 ± 1.2	2.2 ± 1.1	0.19
Insulin (μU/mL)	9.6 ± 3.3	9.5 ± 3.2	09.0 ± 2.8	0.13	8.4 ± 4.6	8.4 ± 4.6	8.9 ± 3.7	0.75
HOMA	2.0 ± 0.5	1.81 ± 0.4	1.81 ± 0.5	0.009	1.9 ± 1.0	1.8 ± 0.6	1.8 ± 0.8	0.88

*Friedman. BMI = body mass index; WC = waist circumference; FM = fat mass; FFM = fat free mass; HOMA = homeostasis model assessment; FBG = fasting blood glucose; TC = total cholesterol; LDL = low density lipoprotein cholesterol; HDL = high density lipoprotein cholesterol; TG = triglycerides.

months[23] to 29 months.[21] The four studies compare LGI or LGL meals with low-fat meals or conventional diet. Pereira et al.,[21] Ebbeling et al.[15] included a restricted conventional diet vs. ad libitum LGL, and Maki et al.[23] included 15 intensive clinic visits and a low-fat portion-controlled diet.

In our study the energy intake was lower for both the LGI and HGI diets than the previously reported RCTs. There were no differences in the consumption of fiber and fat content of the diet as in the study conducted by Ebbeling et al.[15] in obese adolescents. However, in our study, the LGL had more fiber than the adolescents in the study by Ebbeling et al.[15]. The groups in the study conducted by Pereira et al.[21] had different carbohydrate, fiber, fat and protein consumption. In the study by Ebbeling et al. conducted in young adults,[22] there were differences in carbohydrate and fat consumption, and in the study conducted by Maki et al.[23] there were differences in the carbohydrate and fat content of the diets.

In our study, significant differences were observed in both groups in most anthropometric measurements. The LGL group obtained a weight reduction of 4.5 % ($p = 0.008$) and the HGL group 3.0% ($p = 0.68$).

These results are consistent with those observed by Raatz et al.[16] in overweight and obese subjects. In this study, after six months with LGL and LGI diets, a greater reduction in fat mass was observed with maintenance of fat-free mass and a significant improvement in HOMA, compared with HGI diets.[16] The reduced effects observed on body weight, fat mass and HOMA in the LGL group are consistent with those reported by others.[22] In addition, Pereira et al.[21] and Ebbeling et al.[15] also reported between-group differences on those parameters. However, the study by Pereira et al.[21] excluded those with weight loss < 10%. In the study by Ebbeling et al.,[15] a lower consumption of carbohydrate and fiber and higher consumption of fat showed a reduction in triglycerides between groups. The study by Pereira et al.[21] also showed a reduction between groups on triglycerides, and the study by Maki et al.[23] showed a beneficial effect on HDL cholesterol. Similarly, in our study a reduction of triglycerides (18%) was shown but did not reach statistical significance (table III); however, a decrease in HDL cholesterol was also shown in both groups without changes in physical activity. In this study, in the LGL diets showed a significant reduction in dietary GI and GL, calories, carbohydrate and protein intake, while in the HGL diets showed a significant reduction in calories. Dietary fiber consumption in both diets was maintained constant. This could imply that the changes obtained by the LGL might be attributed to the GL of the diet. These results also suggest that hypocaloric diets with LGL may be more efficient measures for the reduction of BMI, body fat, and HOMA and may contribute to the prevention of diabetes in a highly susceptible population. The limitations of this study were the low participation at the beginning of the study, low participation rate at three (61%) and six months (40%) of the intervention.

These results suggest that long term LGL diets are more effective for reducing body mass index, total body fat, waist circumference, HOMA, total cholesterol, and LDL-cholesterol; therefore, may contribute in the prevention of diabetes among subjects with Mexican dietary habits.

References

1. Brennan CS. Dietary fiber, glycemic response and diabetes. *Mol Nutr Food Res* 2005; 49: 560-570.
2. Kemper HCG, Stasse-Wolthuis M, Bosman W. The prevention and treatment of overweight and obesity Summary of the advisory report by the Health Council of the Netherlands. *J Med* 2004; 62 (1): 10-17.
3. Oster G, Edelsberg J, O'Sullivan A, Thompson D. The clinical and economic burden of obesity in a managed care setting. *Am J Managed Care* 2000; 6 (6): 681-689.
4. Ludwig D. The glycemic index, physiological mechanisms relating obesity, diabetes and cardiovascular disease. *JAMA* 2002; 287 (18): 2414-2423.
5. Willett W, Manson J, Liu S. Glycemic index, glycemic load and risk of type 2 diabetes. *Am J Clin Nutr* 2002; 76 (Suppl.): 274S-280S.
6. Brand-Miller JC, Hayne S, Petocz P, Colagiuri S. Low-glycemic index diets in the management of diabetes. A meta-analysis of randomized controlled trials. *Diabetes Care* 2003; 26 (8): 2261-2267.
7. Jiménez-Cruz A, Bacardí-Gascón M, Turnbull WH, Rosales-Garay P, Severino-Lugo I. A flexible, low glycemic index Mexican style diet in overweight and obese subjects with type 2 diabetes improves metabolic parameters during a 6-week treatment period. *Diabetes Care* 2003; 26: 1967-1970.
8. Jiménez-Cruz A, Turnbull WH, Bacardí-Gascón M, Rosales-Garay P. A high-fiber, moderate-glycemic-index, Mexican style diet improves dyslipidemia in individuals with type 2 diabetes. *Nutr Res* 2004; 24: 19-27.
9. Rizkalla SW, Taghrid L, Laromiguiere M, Huet D, Boillot J, Rigoir A, Elgably F, Slama G. Improved plasma glucose control, whole-body glucose utilization, and lipid profile on a low-glycemic index diet in type 2 diabetic men. *Diabetes Care* 2004; 27: 1866-1872.
10. Bacardí-Gascón M, Dueñas-Mena D, Jiménez-Cruz A. Lowering effect on postprandial glycemic response of nopales added to Mexican breakfasts. *Diabetes Care* 2007; 30 (5): 1264-1265.
11. Jiménez-Cruz A, Seimandi-Mora H, Bacardí-Gascón M. Efecto de dietas con bajo Indice glucémico en hiperlipidémicos. *Nutr Hosp* 2003; 18: 331-335.
12. Salmeron J, Ascherio A, Rimm E, Colditz G, Spiegelman D, Jenkins D, Sampfer M, Wing A, Willet W. Dietary fiber, glycemic load and risk or NIDDM in men. *Diabetes Care* 1997; 20: 545-550.
13. Brand-Miller JC, Thomas M, Swan V, Ahmad ZI, Petocz P, Colagiuri S. Physiological validation of the concept of glycemic load in lean young adults. *J Nutr* 2003; 133: 2728-2732.
14. Van Dam RM, Visscher AWJ, Feskens EJM, Verhoef P, Kromhout D. Dietary glycemic index in relation to metabolic risk factors and incidence of coronary heart disease. The Zutphen Elderly Study. *Eur J Clin Nutr* 2000; 54: 726-731.
15. Ebbeling CB, Leiding MM, Sinclair KB, Hangen JP, Ludwing DS. A reduced-glycemic load diet in the treatment of adolescent obesity. *Arch Pediatr Adolesc Med* 2003; 157: 773-779.
16. Raatz SK, Torkelson CJ, Redmon B, Reck KP, Kwong CA, Swanson JE, Liu C, Thomas W, Bantle J. Reduced glycemic index and glycemic load diets do not increase the effects of energy restriction on weight loss and insulin sensitivity in obese men and women. *J Nutr* 2005; 135: 2387-2391.
17. Foster-Powell K, Holt SH, Brand-Miller C. International table of glycemic index and glycemic load values: 2002. *Am J Clin Nutr* 2002; 76: 5-56.
18. Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: insulin resistance and beta cell function from fasting plasma glucose and insulin concentrations in man. *Diabetologia* 1985; 28: 412-419.
19. Bonora E, Targher G, Alberiche M, Bonadonna RC, Saggiani F, Zenere MB, Monauni T, Muggeo M. Homeostasis model assessment closely mirrors the glucose clamp technique in the assessment of insulin sensitivity. *Diabetes Care* 2000; 23 (1): 57-63.
20. International Physical Activity Questionnaire. (Available at http:/www.ipaq.ki.se) (accessed January 2006).
21. Pereira MA, Swain J, Goldfine AB, Rifai N, Ludwig DS. Effects of a low-glycemic load diet on resting energy expenditure and heart disease risk factors during weight loss. *JAMA* 2004; 292 (20): 2482-2490.
22. Ebbeling CB, Leiding MM, Sinclair KB, Seger-Shipppe LG, Feldman HA, Ludwig DS. Effects of an ad libitum low-glycemic load diet on cardiovascular disease risk factors in obese young adults. *Am J Clin Nutr* 2005; 81: 976-982.
23. Maki KC, Rains TM, Kaden VN, Raneri KR, Davidson MH. Effects of a reduced glycemic load diet on body weight, body composition, and cardiovascular disease risk markers in overweight and obese adults. *Am J Clin Nutr* 2007; 85: 724-734.