Diet Content of Non-Saturated Fatty Acids is Related to Lower Plasma Adiponectin in Patients with Metabolic Syndrome

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Abstract

Introduction. The metabolic syndrome (MS) has been related to the unbalance between pro- and anti-inflammatory cytokines; where adiponectin, an anti-inflammatory adipokine, is considered to play a key metabolic role. The consumption of certain micronutrients has been claimed to modify pro- and anti-inflammatory cytokines.

Aim. To explore whether dietary micronutrients are related with plasma adiponectin in patients with MS.

Methods. Cross-sectional analysis. Quartiles of dietary bioactive compounds were compared according to values of plasma adiponectin. Interquartile variation (IQV) and correlation analyses were performed.

Results. There was a significant IQV of dietary unsaturated fatty acids (between -50% to -66% of change, p_{25} vs p_{75} , $p < 0.05$), particularly for trans-fatty acids, poly-unsaturated fatty acids, mono-unsaturated fatty acids and ω -6 fatty acids, in relation to plasma adiponectin; as well as a negative correlation ($\rho = -0.53, -0.37, -0.29$ and -0.34 , respectively; $p < 0.05$).

Conclusion. Dietary amounts of unsaturated fatty acids inversely related to plasma adiponectin in patients with MS.

Introduction

The metabolic syndrome (MS) comprises a set of widely distributed risk factors favoring the development of cardiovascular diseases and type 2 Diabetes Mellitus (DM2) [1–4].

Pathophysiological mechanisms underlying MS have been related to the unbalance between pro- and anti-inflammatory cytokines; where adiponectin, an anti-inflammatory adipokine, is considered to play a key role due to different effects like: 1) the induction of other anti-inflammatory cytokines like IL-10 and interleukin 1 receptor antagonist (IL-1RA) in human monocytes, monocyte-derived dendritic cells and macrophages, 2) the regulatory effect on insulin sensitivity, and 3) the anti-atherogenic effect [5–7].

The consumption of pro-inflammatory diets and hyperglycemia have been associated with higher prevalence of hypertension and obesity, suggesting that dietary habits affect metabolic profiles considered of higher risk [6–8].

There is an increasing interest in the study of dietary bioactive compounds with ability to modify plasma cytokines, which has been evidenced through out several longitudinal studies. For example, the consumption of food from animal source has been observed to decrease plasma adiponectin, whereas the intake of vegetable source foods, enriched with fiber and ω -3 fatty acids, may increase plasma adiponectin [5–7]. Likewise, the consumption of high fiber, complex carbohydrates-enriched diet has been reported to increase plasma adiponectin in overweight and obese subjects [9], while low fiber, fat-enriched diet reduced plasma adiponectin in patients with DM2 [10]. Nevertheless, whether other food properties modify adiponectin is less clear and sometimes controversial. Hypocaloric diets have been reported to

increase plasma adiponectin [11], while other studies have failed to achieve a significant effect of hypocaloric diets on adiponectin [12–14].

Therefore, the present study was designed to explore whether different dietary compounds modify plasma adiponectin in a higher risk population like subjects with MS.

Materials And Methods

Study Design

Observational, analytical cross-sectional study.

Setting

This study was performed at Endocrinology and Bariatric Surgery Departments, as well as Laboratory of Experimental Metabolism and Clinical Research Department, from Centro Médico Nacional “20 de Noviembre”, ISSSTE at Mexico City, between 2017 and 2019.

Study population

Patients older than 18 years old, diagnosed with MS as defined by NCEP/ATP III, meeting at least three of the following components: 1) waist circumference of more than 102 cm in men or more than 88 cm in women, 2) fasting triglyceride level of 150 mg/dL or higher, 3) HDL cholesterol $\leq$ 40 mg/dL for men or 50 mg/dL for women, 4) systolic/diastolic blood pressure $\geq$ 130/85 mmHg or receiving drug treatment, and 5) fasting plasma glucose $\geq$ 100 mg/dL [2]. Patients were excluded if presence of chronic renal failure, hepatic disease, decompensated heart failure, as well as those patients participating in other protocols involving physical rehabilitation or nutritional programs.

For sample size calculation, we estimated the effect of food content on adiponectin plasma concentration, according to previous reports [7], considering 95%CI alpha = 0.05, our sample size was n = 95, which rendered a beta power of >0.8 . The study protocol complied with the Declaration of Helsinki and was approved by the Local Ethics Committee. Informed consent was obtained from all patients for study inclusion.

MEASUREMENTS

Anthropometry

Clinical-demographic data were obtained through individual interviews performed by experienced researchers. Anthropometric variables (weight, height, BMI, arm and waist circumferences) were obtained by clinical nutrition specialists, according to standard methods [15].

Dietetic habits

A 24-hour recalls were used to register habitual type of diet, with emphasis in their food schedules and size portions that patients consumed a day before the interview. In case that food consumption on the day before did not reflect the usual diet, patients were asked to describe a habitual day. NASCO® food replicas kit was used to help patients in order to describe the size portions more accurately. All 24-hour recalls were analyzed with the Food Processor® program v.7, allowing estimating the amount of energy (kilocalories), macro- and micro-nutrients. All measurements were performed by 2 previously experienced nutritionists.

Adiponectin determination

All blood samples were obtained after 8 hours fasting, by antecubital venopuncture. Then, blood was immediately centrifuged 5000rpm for 5 min, at room temperature and plasma was recovered. Plasma adiponectin was determined by immunomagnetic multiplexing assay (Milliplex Map Human Adiponectin Magnetic Bead Panel Thermo Fischer Scientific, USA), and readings were performed in a MAGPIX System 40–072–EM (Millipore, Austin, Texas, USA). The measurements were processed according to provider specifications.

Statistical analysis

Normality data analyses were performed with Shapiro-Wilks test. All continuous variables were expressed as mean $\pm$ SD or median and InterQuartile Range (IQR, P_{25} , P_{75}), as appropriate; qualitative data were shown as n (%). For inferential analyses, one-tailed, non-paired U-Mann Whitney or T-test were used as appropriate; likewise, Spearman test was used to analyze relation between quantitative data. All statistics tests were performed using software PRISM (v. 6.0) and SPSS (v. 20). Statistical significance was considered if $p \leq 0.05$.

Results

The study population was constituted by 95 patients with metabolic syndrome, aged 58 years-old, 78.7% female, with higher prevalence of insulin resistance, dyslipidemia and obesity. Likewise, anthropometric and biochemical parameters indicate cardiometabolic risk (Tables 1 and 2), despite general intake of macronutriments was normal (Table 3).

Table 1
Clinical-Demographic Characteristics (n = 95)

Age (years-old)	58.5 ± 9.9
Male (n [%])	20.0 (21.3)
Co-morbidities	
Type 2 Diabetes Mellitus (n [%])	76.0 (80.9)
Dyslipidemia (n [%])	58.0 (61.7)
Obesity (n [%])	47.0 (50)
HAS (n [%])	59 (62)
Biochemical Data	
Plasma Glucose (mg/dL)	106.0 (93.5 to 126.0)
Total Cholesterol total (mg/dL)	142.9 (100.0 to 176.0)
c-HDL (mg/dL)	45.9 ± 14.7
c-LDL (mg/dL)	92.4 (62.7 to 120.3)
Triglycerides (mg/dL)	134.3 ± 47.1
Adiponectin (pg/dL)	5.44 (2.28 to 12.68)
Quantitative data are resumed as mean ± SD or p50 (p25 to p75) as appropriate; while qualitative data are described as n (%). Abbreviations: c-HDL: high-density cholesterol; c-LDL, low-density cholesterol.	

Table 2
Anthropometric Characteristics

Weight (Kg)	78.3 ± 17.1
BMI (Kg/m ²)	28.9 (26.3 to 32.6)
Brachial Circumference (cm)	32.5 (29.5 to 35.4)
Wrist Circumference (cm)	99.5 (91.2 to 106.0)
Hip Circumference (cm)	102.0 (96.3 to 111.8)
Quantitative data are resumed as mean ± SD or p50 (p25 to p75) as appropriate; while qualitative data are described as n (%). Abbreviations: BMI, Body Mass Index.	

Table 3
Macronutriments Consumption

Energy (Kcal)	1430.5 (1083.7 to 1874.1)
Proteins (g)	71.4 ± 27.5
Carbohydrates (g)	185.0 (116.5 to 252.8)
Lipids (g)	51.2 ± 30.5
Quantitative data are resumed as mean ± SD or p50 (p25 to p75) as appropriate; while qualitative data are described as n (%). Abbreviations: BMI, Body Mass Index.	

The study population was divided by quartiles according to the amount of nutriment composition in the diet. Then, value of plasma adiponectin was distributed between the quertiles of nutriments. A significant progressive reduction of adiponectin accross the increasing quartiles of nutriments was observed for trans- fatty acids, PUFAs, mono-unsaturated fatty acids and ω-6 fatty acids (Table 4).

Table 4
Dietary Micronutrients and Plasma Adiponectin

	Plasma Adiponectin (pg/dL) according to Micronutrients					% Change <i>P25 vs P75</i>	
	Q1	Q2	Q3	Q4	p-value		p-value
Trans- FA (g)	7.2 (4.4 to 10.5)	4.5 (2.9 to 7.4)	4.5 (2.3 to 7.8)	2.4 (1.9 to 4.8)	0.01	-66.62	0.01
PUFA (g)	6.2 (2.2 to 10.9)	7.1 (4.0 to 11.9)	3.0 (2.3 to 5.8)	2.6 (2.1 to 4.5)	0.004	-57.95	0.01
Mono-unsaturated FA (g)	5.6 (2.2 to 9.9)	5.6 (3.0 to 9.8)	3.6 (2.3 to 6.1)	2.6 (1.6 to 4.9)	0.06	-53.75	0.03
ω-6 FA (g)	6.7 (2.3 to 9.9)	6.2 (3.0 to 10.6)	4.0 (2.2 to 6.2)	3.3 (2.3 to 4.5)	0.05	-50.97	0.04
Potassium (mg)	3.0 (2.1 to 11.9)	5.4 (3.0 to 9.5)	4.5 (2.6 to 7.2)	5.4 (2.2 to 6.7)	0.49	77.74	0.88
Calcium (mg)	6.5 (3.8 to 12.8)	4.5 (2.4–6.9)	3.8 (2.2 to 5.7)	3.3 (2.3 to 7.7)	0.14	-49.15	0.05
Vitamin B6 (mg)	5.8 (3.9 to 6.9)	4.5 (1.9 to 9.0)	4.4 (2.2 to 8.8)	3.0 (2.4 to 4.9)	0.23	-47.77	0.04
Vitamin B2 (mg)	6.3 (3.9 to 9.4)	4.5 (2.3 to 8.3)	4.5 (1.9 to 7.7)	3.3 (2.4 to 7.0)	0.37	-47.61	0.07
Cholesterol (mg)	5.2 (4.0 to 9.9)	5.4 (2.2 to 7.2)	4.5 (2.3 to 8.3)	2.9 (2.3 to 6.5)	0.45	-42.33	0.08
Vitamin E (UI)	7.0 (2.5 to 12.0)	5.4 (2.4 to 10.6)	4.1 (2.3 to 7.0)	5 (2.3 to 5.4)	0.25	-41.46	0.06
Vitamin C (mg)	4.0 (1.5 to 7.1)	3.8 (2.6 to 5.4)	3.8 (2.6 to 5.4)	5.8 (2.6 to 9.1)	0.82	40.59	0.88
Vitamin B1 (mg)	5.6 (3.7 to 9.9)	3.8 (2.0 to 6.5)	4.5 (2.0 to 8.9)	3.5 (2.2 to 6.5)	0.37	-36.96	0.04
Sodium (mg)	6.2 (2.7 to 9.9)	3.1 (2.1 to 5.4)	4.3 (2.4 to 8.1)	4.1 (2.8 to 8.0)	0.1	-33.6	0.41
Carbohydrates (g)	5.2 (2.4 to 9.9)	4.5 (2.3 to 6.6)	4.1 (2.1 to 7.1)	3.7 (2.3 to 8.5)	0.9	-28.6	0.58
Vitamin B3 (mg)	5.4 (2.3 to 6.8)	5.0 (3.1 to 1.7)	2.5 (2.1 to 8.9)	4 (2.5 to 5.8)	0.49	-26.65	0.4
ω-3 FA (g)	4.5 (1.9 to 6.8)	5.4 (2.3 to 9.4)	5.4 (2.9 to 7.5)	3.9 (2.5 to 6.1)	0.57	-14.61	0.84

Plasma Adiponectin (pg/dL) according to Micronutrients						% Change <i>P25 vs P75</i>	
Selenium (mcg)	5.0 (2.2 to 10.2)	5.4 (3.4 to 8.3)	3.8 (2.2 to 8.9)	4.5 (2.4 to 5.6)	0.55	-10.1	0.34
Folate (mcg)	4.3 (2.3 to 9.1)	3.9 (2.9 to 6.4)	5.8 (1.7 to 9.6)	3.8 (2.3 to 6.2)	0.57	-6.86	0.57
Saturated FA (g)	4.7 (2.1 to 9.4)	4.7 (2.1 to 9.4)	3.1 (2.3 to 6.1)	4.9 (2.1 to 12.0)	0.45	3.16	0.99
Vitamin D (UI)	3.8 (2.2 to 11.5)	6.3 (3.9 to 7.5)	2.3 (1.7 to 4.6)	5.4 (2.8 to 6.0)	0.16	0	0.81
Adiponectin was distributed according to quartiles of dietary micronutrients (Q1 refers to < p25; Q2 refers to p25-50; Q3 refers to p50-p75 and Q4 refers to > p75). Interquartile variance was compared by Kruskal-Wallis test. Then, the magnitude of change was estimated by U-Mann-Whithney test. Abbreviations: FA, Fatty Acids; PUFA, Poly-unsaturated fatty acids.							

The highest correlations between nutriment amounts and plasma adiponectin are shown in Fig. 1, highlighting the moderate negative correlation between trans- fatty acids (-0.53, CI95% -0.70 to -0.31, $p = 0.0001$); PUFA (-0.37, CI95% -0.57 to -0.12, $p = 0.0019$); ω -6 fatty acids (-0.33, CI95% -0.54 to 0.08, $p = 0.0043$) and mono-unsaturated fatty acids (-0.29, CI95% -0.51 to 0.03, $p = 0.012$) with plasma adiponectin.

Discussion

Our main finding was the inverse relation adiponectin with diet amount of unsaturated fatty acids, particularly trans-fatty acids, poly-unsaturated fatty acids, mono-unsaturated fatty acids and ω -6 fatty acids.

Our results are similar to observations reported by Esposito et al, where consumption of a high fat, low fiber diet for 1 week induced a reduction of plasma adiponectin, either in healthy individuals and patients with DM2 [10].

In the other hand, Peake et al. showed that consumption of a saturated fat-rich breakfast for approximately 1 week did not significantly modified plasma adiponectin in healthy individuals or patients with DM2; while Paniagua et al. did not observed difference in mRNA adiponectin between consumption of saturated- vs unsaturated fatty acid-rich breakfast for 3 days in healthy individuals with metabolic risk factors. Such controversies with our findings may be explained by variations in the study design, methods to measure adiponectin, the type of fatty acids and time of consumption [16, 17], suggesting that the effect of unsaturated fatty acids on adiponectin is specific and may depend on the time of consumption, for at least 1 week.

Our findings are in line with some mechanisms proposed to explain why specific unsaturated fatty acids, calcium and pyridoxin may decrease adiponectin. PUFA may modulate enzymes responsible for

adiponectin expression through AMPK pathway in the liver and adipocytes; or peroxidation particularly of linoleic acid produces hydroxyalkenal 4-hydroxynonenal derivate, which may reduce adiponectin gene [18, 19]. Other responsible mechanisms may be related with calcium and piridoxin. There are reports showing that calcium-content reduces adiponectin, which may be explained that calcium reduces production of fatty acid and promotes lipolysis; whereas piridoxin may be modulating intracellular calcium signaling and lipid metabolism regulation [20].

Our findings contribute to the understanding of how diet composition and the time of specific diet consumption are able to induce higher adiponectin and the associated cardiometabolic benefits. However, some limitations of the study should be considered. First, the heterogenicity of the population regarding life styles, co-morbidities and drug therapies; while a healthy control group was not included. Second, the low sample size, which may be a source for potential bias. Third, the nutriments content in the diet was estimated through a 24-hour recall approach, whereas a direct measure of nutriments would be more accurate.

Conclusion

Results obtained from the present study suggest that diet content of non-saturated fatty acids decreases plasma adiponectin in patients with metabolic syndrome.

Declarations

Ethical approval and consent to participate

The study was designed and performed according to ethical guidelines of the 1975 Declaration of Helsinki, and it was approved by the Local Committees of Research, Ethics in Research and Biosafety of the Centro Médico Nacional '20 de Noviembre' ISSSTE, Mexico City. All participants provided written informed consent.

Consent for publication

The authors state that they have read and approve the present manuscript and give their consent for publication.

Availability of data and materials

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests

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Authors' contributions

JAS-C, DED-J, JAP-J, were responsible for the conception, design and revision of the study and drafting the manuscript. AGM-M, ODV-A, SN-A, EV-G, AH-P, ME-T, PL-M, JAL-V, RP-CdeV, JRA-M, AAG-P, BB-F, JM-R, MAD-A, RCC-H, AM-L, SG, JG-S and PM-T were responsible for the generation, collection, analysis and interpretation of data.

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Figures

Figure 1

Dietary components most related with plasma adiponectin. The figure shows the correlations between micronutrients most significantly related to plasma adiponectin. Spearman correlation.