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ABSTRACT

Introduction: the Dietary Inflammatory Index (DII) provides a quantitative means for assessing the role of diet in relation to health outcomes.

Objetive: this study aimed to assess the association between the inflammatory potential of diet, as measured by the DII and IL-17A levels in young adults.

Methods: a cross-sectional study was conducted on 69 adults between 18-35 y of age in San Luis Potosi, Mexico. Fasting blood samples were collected to analyze lipid profile, glucose homeostasis, and IL-17A. Dietary intake was assessed using a 24-hour recall. DII scores were calculated from 19 available food parameters. Univariate linear regression models were estimated to evaluate the possible dependence of IL-17A levels (dependent variables) on some potential explicative variables such as anthropometric, clinical, biochemical, and dietary variables.

Results: there was a high inflammatory potential, with a mean DII score of +1.04 (range: -2.19 to +2.78). The DII was not associated with BMI, IL-17A levels or cardiometabolic risk factors.

Conclusion: the study shows that the diets of healthy college-aged Mexican adults had a high inflammatory potential.

Keywords: Dietary Inflammatory Index. Dietary intake. Serum IL-17A. Overweight/obesity. Young adults.

RESUMEN

Introducción: el Índice Inflamatorio de la Dieta (DII) proporciona un medio cuantitativo para evaluar el papel de la dieta en relación con los resultados de salud.

Objetivo: este estudio tuvo como objetivo evaluar la asociación entre el potencial inflamatorio de la dieta, medido por DII y los niveles de IL-17A en adultos jóvenes.

Métodos: se realizó un estudio transversal en 69 adultos entre 18 y 35 años de edad en San Luis Potosí, México. Se recogieron muestras de sangre en ayunas para analizar el perfil lipídico, glucosa y la IL-17A. La ingesta dietética se evaluó mediante un recordatorio de 24 horas. Las puntuaciones DII se calcularon a partir de 19 parámetros alimentarios disponibles. Se estimaron modelos de regresión lineal univariante para evaluar la posible dependencia de los niveles de IL-17A (variables dependientes) de algunas variables explicativas potenciales, como variables antropométricas, clínicas, bioquímicas y dietéticas.

Resultados: con una puntuación DII media de +1,04 (rango: -2,19 a +2,78), se determino un alto potencial inflamatorio. El DII no se asoció con el IMC, los niveles de IL-17A ni con los factores de riesgo cardiometabólico en esta población.

Conclusión: el estudio muestra que las dietas de adultos mexicanos sanos en edad universitaria muestran un alto potencial inflamatorio.

Palabras clave: Índice inflamatorio de la dieta. Ingesta dietética. IL-17A. Sobrepeso/Obesidad. Adultos jóvenes.

INTRODUCTION

Obesity is a serious medical condition associated with various non-communicable diseases (NCDs), reaching epidemic rates worldwide (1). Obesity is defined as the excessive accumulation of adipose tissue (AT) that presents a potential health risk (2). In Mexico, Obesity has become

a public health concern as its rates have been steadily increasing for the past 30 years (3). In 2021, 72.4 % percent of Mexican adults (> 20 years) were classified as having overweight (35.7) or obesity (36.7) (4). Individuals affected by obesity can present an increased risk of developing NCDs such as cardiovascular disease, type 2 diabetes mellitus, and obesity-related cancers.

Obesity is identified as a low-grade chronic inflammation state and is characterised by a raised expression of inflammatory markers into adipose tissue (AT). AT in obesity is characterized by increased lipid storage, which leads to its dysfunction, cellular lipid toxicity, inflammation, and oxidative stress, triggering the release of acute phase proteins, and pro-inflammatory adipokines including interleukin (IL)-6 (5). Furthermore, IL-6 is required for the differentiation of *naïve* CD4 T cells into the T helper 17 (Th17) subpopulation (6). The involvement of traditional inflammatory mediators, such as tumor necrosis factor α (TNF-α) and C-reactive protein (CRP), has been thoroughly examined in the context of obesity (7). Recent investigations have suggested the potential role of the Th17 T cell sub-lineage in metabolic disorders. Th17 cells participate in obesity-dependent inflammation and an increased frequency of these cells in individuals with obesity as well levels of IL-17A have been observed (8).

IL-17 family of cytokines consists of six ligands from IL-17A to IL-17F. IL-17A is the effector and the classic cytokine of Th17 cells. IL-17 plays a protective role in the host's defense against pathogens, response to injury, and physiological stress. However, excessive production of IL-17A is one of the potential mechanisms underlying chronic inflammatory conditions (9) IL-17A is involved in the induction of adipogenesis role in several inflammatory diseases (10). Obesity promotes expansion of IL-17-producing T cells in AT and periphery (11,12). Subjects with obesity had a higher level of IL-17 cytokines and a correlation was found between the level of these cytokines and the content of adipose tissue.

In addition, the intake of potassium, iron, vitamins B6 and C, and folic acid has been associated with decreased concentrations of IL-17 isoforms (13).

Diet is a crucial modifiable factor for reducing the risk of chronic diseases (14). Specific compounds found in nutrient-dense foods, such as omega-3 fatty acids, fiber, and polyphenols, exhibit anti-inflammatory properties. Conversely, reduced intake of fruits and vegetables and high consumption of calorie-dense ultra-processed food correlate with increased levels of inflammatory markers (14). Researchers have linked unhealthy diets, characterized by high sugar consumption, high saturated fat intake, and low fiber, vitamins, minerals, and other plant-derived molecules such as antioxidants, to an increased risk of NCDs. It is also thought that these diets cause dysbiosis, oxidative stress, the NF-B pathway, and higher levels of TNF- and IL-6, all of which lead to low-grade systemic inflammation (15,16).

The Dietary Inflammatory Index (DII) is a literature-derived population-based dietary score intended to assess the inflammatory potential of an individual's overall diet based on the balance of pro- and anti-inflammatory properties of its components, including macronutrients, vitamins, minerals, flavonoids, and specific food items (17). A positive DII score has been associated with a higher BMI (18), and several studies have confirmed that a higher DII score was associated with an increased risk of obesity (19-21). According to Sakhaei et al., participants in the top tertile of the healthy diet score had lower concentrations of serum IL-17A compared to those in the lowest tertile (22). However, the association between dietary inflammatory potential and IL-17A levels remains unclear. We aimed to investigate the association of the Dietary Inflammatory Index (DII) with L-17A levels in healthy adults and adults who are overweight or obese. We also want to find out if these cytokine levels are linked to clinical and dietary parameters in a cross-sectional sample of 62 adults in San Luis Potosí, Mexico.

MATERIALS AND METHODS

This was a cross-sectional study conducted in San Luis Potosí, Mexico. The study population included adults aged 18 years to 30 years, recruited from the surrounding community via public signage and flyers. All participants provided written informed consent, and all study procedures were approved by the University of Illinois Institutional Review Board (Protocol #15503) and conformed to standards for the use of human participants in research as outlined in the seventh revision of the Declaration of Helsinki. Subjects were compensated with US$ 15 for their participation in this study. Inclusion criteria included no previous history of physician-diagnosed gastrointestinal or metabolic disease. Adults were excluded if they had previously been diagnosed with cardiovascular, hepatic, renal, or oncological conditions, or if they were pregnant or lactating. Participants were eliminated if they did not not completed the evaluation.

Anthropometrics and biological measures

Anthropometric parameters such as weight, height, and waist circumference (WC) were measured in duplicates and the average of the two numbers were documented. Body mass index (BMI) was calculated by dividing weight (kg) by the squared value of height in meters. The reference interval of BMI was defined as 18.5-24.9 kg/m^2. Over-weight and obesity were considered with a BMI of ≥ 25 or 30 kg/m^2, respectively. Height was measured using a mobile stadiometer (Seca 213), weight was collected on a calibrated electronic weighing scale TANITA UM-081, and waist circumference was measured at the midpoint between the last floating rib and the iliac crest using a LUFKIN Executive Thin line 2 m, W606PM metal tape.

Blood pressure was measured in a seated position using an automatic blood pressure monitor (Omron Healthcare Co.). Venous blood was

collected from the antecubital vein following a 10-h overnight fast. These parameters were measured by trained research staff as previously described (23).

Blood analysis

Fasting blood samples were collected in vacutainer tubes, which were cooled to 4 °C and centrifuged 15 min to obtain serum and subsequently stored at -70 °C. Serum samples were used to measure the concentrations of total cholesterol, HDL-c, triglycerides, and fasting glucose were assessed on the automated analytical platform BS300 (Mindray®, Nanshan, Shenzhen, China) following the internal test protocols and the use of commercially available reagent kits. IL-17A was measured using ELISA kit (Bio Legend, San Diego, CA, US). The assays were carried out following the manufacturer's instructions.

Dietary assessment

Before participants were interviewed, they attended a brief session where registered dietitians (RD) demonstrated how to appropriately estimate food intake using household utensils. Participants were asked to write down the type and amount of food eaten, using scales or household measures to gauge portion sizes where possible. Dietitians interviewed each participant and recorded the estimate of food and drinks consumed. The information included in the 24-hour recall was the date of record, mealtime, and amount of food consumed. Information from one 24-hours dietary recall was coded by a trained RD using the Nutrikcal computer program (Nutrikcal VO®, Mexico), which is based on the Mexican System of Food and Equivalents. Macro and micronutrient intakes were compared with sex and age-specific nutrient requirements defined by the Food and Nutrition Board of the National Academy of Science (24). VC, vitamin A (VA), vitamin E (VE), VD, thiamin (B1), riboflavin (B2), niacin (B3), pantothenic acid (B5), pyridoxine (B6), folate

(B9), cobalamin (B12), calcium (Ca), potassium (K), magnesium (Mg), sodium (Na), phosphate (P), selenium (Se), iron (Fe), and Zn.

Dietary inflammatory index (DII)

Dietary information data of participants were compared with the global standard dietary intake database, which provide a reliable estimation of the median and interquel range and the Z-score of each nutrient or food was calculated, i. e.:

Z = (actual dietary intake amount - standard global median) / standard deviation)

Subsequently, to minimize the effect of "right-skewing", the Z-value was converted into percentiles, while each percentile was multiplied by 2 and finally subtracted by 1. Then the resulting value was multiplied by the corresponding food parameter effect score to obtain the food parameter-specific DII score for an individual (18). Finally, all dietary parameter-specific DII scores were added together to calculate the overall DII score. The higher the DII score, the stronger the proinflammatory effect. In the present study, a total of 19 dietary intake parameters were obtained by the 24 h recall, and used to compute DII (namely: protein, total fat, carbohydrates, cholesterol, dietary fiber, vitamin A, thiamine, riboflavin, niacin, vitamin C, vitamin E, folic acid, vitamin B6, magnesium, iron, zinc, and selenium).

Statistical analysis

Data are shown as the mean ± the standard deviation (SD) or the median ± interquartile range. The distribution of each one of the variables was assessed by the Shapiro-Wilk normality test. Groups were compared using the Mann-Whitney U test or t-test according to the distribution of the data. Spearman correlation was employed to assess

the correlation between different parameters. Differences were considered significant at $p < 0.05$. Univariate and multivariate linear regression models were estimated to assess the possible dependence of IL-17A on some explicative variables. A multivariate model (by stepwise procedure) was estimated considering the following independent variables: overall DII score and specific DII score for the 19 dietary intake parameters. Statistical analyzes were conducted using the statistical packages SPSS (version 20.0), and InStat GraphPad software (InStat GraphPad Inc.,San Diego, CA, USA), version 5.0.

RESULTS

A total of 69 individuals (56.4 % females) were included in the cross-sectional analysis. Participant characteristics according to BMI are presented in table I. The prevalence of overweight and obesity in the sample was 48.3 %. In comparison with the healthy group, the overweight and obesity group showed increased values for body mass index, triglycerides, waist circumference, systolic and diastolic pressure, TC/HDL, triglyceride-glucose index (TyG), and visceral adiposity index (VAI) score. Adults impacted by overweight or obesity were significantly older than those with a BMI < 25 kg/m^2. The mean DII was 1.04 (SD = 1.12) and ranged from -2.18 (most anti-inflammatory) to 2.77 (highly pro-inflammatory). Finally, no differences in IL-17A levels were detected. The usual intakes of macro and micronutrients from foods according to BMI are shown in table II. Among 18-35-year-old individuals, the intakes of the following macro and micronutrients were significantly different across the two groups: Kcal/kg weight, protein (g/kg weight), carbohydrate (g/kg weight), lipids (g/kg weight), B2, and Mg. The percentage of macronutrients in relation to the total energy value (TEV) was within the values recommended by the acceptable macronutrient distribution ranges (AMDR). There was no significant difference observed

in usual dietary intakes according to sex except for Se intake which was lower in women.

Individuals with obesity had higher level of IL-17A ($p = 0.02$) than overweight groups (data not shown). When classifying the subjects according to the parameter of abdominal obesity, a tendency towards an increase in the levels of IL-17A can be observed (p =0.07). No differences on IL-17A levels between Normal weight and individuals with BMI ≥ 25 was detected (Fig. 1).

A correlation analysis to assess the possible associations between IL-17A, anthropometric parameters, traits of metabolic syndrome, macronutrient, and micronutrient intake was carried out. Glucose had a weak, but positive association when these variables were analyzed with respect to serum IL-17A in all participants ($p = 0.02$) (Fig. 2).

In females, lL-17A levels were negatively associated with triglycerides ($p = 0.02$, r -0.34), and TyG index ($p = 0.02$, r -0.33). A positive correlation with cholesterol intake ($p = 0.03$, r 0.30) was found. In males, a significant positive correlation with glucose was observed ($p = 0.03$, r 0.357, and carbohydrates percentage from diet ($p = 0.04$, r 0.338).

We analyzed the levels of IL-17A according to the adequacy in the intake and found that individuals with an adequate intake of sodium, and those with inadequate intake of B3 present higher levels of IL-17A. The categorization of the individuals according to the adequacy of the intake of the other micronutrients evaluated, did not show differences in the levels of IL-17A (Fig. 3).

Neither components of metabolic syndrome nor adiposity index predict IL17-A levels. Furthermore, we investigated the independent effect of several macro and micronutrients dietetic intake. B1 was the strongest predictor of IL-17A levels in both males and females, following by Niacin (Table III). Each unit increase in B1 was associated with 1.08-fold

increased odd of IL-17A levels, suggesting that B1 intake is useful in predicting IL-17A levels in young adults.

The mean DII was 1.04 (SD = 1.12), ranging from -2.18 (most anti-inflammatory) to 2.77 (highly pro-inflammatory). No association between overall or specific DII and IL-17A levels was found. Also, no statistical association was observed between DII and other biochemical indices (data do not show). We further investigated the independent effect of specific DII. The β coefficients of correlation between specific DII and IL-17A are shown in table IV. Moreover, the specific DII score for niacin was positively associated with the IL-17 in the unadjusted model 1 ($\beta = 0.275$, $p < 0.001$). In addition, there was a negative relationship between IL-17 and specific DII score for Vit B1 in model 2 ($\beta = -0.376$, $p = 0.02$).

DISCUSSION

Adipose tissue (AT) in obesity presents a progressive infiltration of pro-inflammatory immune cells, which together with increased inflammatory adipokine secretion including IL-17 and other cytokines, to promote chronic inflammation-associated metabolic syndrome and insulin resistance (25). The current study was designed to examine the DII and dietary intake in association with IL-17A (a systemic proinflammatory marker) and cardiometabolic risk factors among young adults. We hypothesized that DII might be positively associated with IL-17A levels. However, in this cross-sectional study of Mexican adults, we found no association with DII, neither with macro nor micronutrient-specific intakes.

Obesity and obesity-related diseases are closely connected to the serum levels of IL-17A (26). We did not find differences on IL-17A levels according to BMI status. Polak-Szczybył et al., reported that IL-17F, IL-17E but not IL-17A levels were positively correlated BMI (27). Nevertheless, the participant's BMI ranged from 30.0 to 58.1 kg/m^2, with a mean of 36.65 ± 5.27 kg/m^2, and in our study the mean BMI was

25.1 ± 3.8 kg/m^2. The above, may explain why in the overweight/ obese group IL-17A levels did not differed from healthy individuals. However, we initially included 85 participants in our study, 13 of whom had IL-17A levels below the detection threshold, leading to their exclusion from the overall analysis. Additionally, we employed the graphpad outlier calculator, and the Grubbs' test successfully identified three significant outliers.

Healthy dietary pattern is inversely related with inflammatory markers (28). Thus, diet is a key modifiable factor since DII score for a more pro-inflammatory diet, were associated with a higher risk of obesity (29). Our study, using the DII score calculation, indicated that participants' diets were more pro-inflammatory. Nevertheless, DII did not show a correlation with IL-17A levels or any cardiometabolic risk factor. The analysis, stratified by BMI and sex, produced similar findings. The above suggests that our population does not show significant systemic markers of inflammation levels, despite a pro-inflammatory diet.

We calculated the DII score computed from 19 food parameters, ranged from -2.19 to + 2.78 with a mean of 1.04, which indicates a proinflammatory diet. These results contrast with a prior study conducted in Mexico (30), where an anti-inflammatory diet had a mean score of −0.68 in adults. These conflicting results may be partly due to the differences in sample size and region representation. On the other hand, it is important to note that our study population is younger and might be less interested in a healthy diet, but this is just a speculation. However, one study reports that the diet of young adults showed a high inflammatory potential, with a mean DII score of +1.10 (range: -4.69 to +5.28). Also, DII was not associated with Metabolic syndrome components in the study population (31).

DII consistently reflects the levels of six inflammatory markers: interleukin (IL)-1β, IL-4, IL-6, IL-10, tumoral necrosis factor-α and C-reactive protein (17). However, we could not find any correlation

between IL-17A and overall DII. We analyzed the specific DII for dietary macro and micronutrients and found that niacin and B1 specific DII can predict IL-17A levels. Nevertheless, it is important to mention that a specific micro or macronutrient DII has not been reported or discussed in any previous publication. Regarding nutrient intake, we observed that subjects with the most pro-inflammatory diet (overweight and obesity) had lower intakes of riboflavin and magnesium. In addition, dietary intake of riboflavin and niacin are predictors of IL-17A levels in Model 4.

The strength of our study is that we use a validated DII score specially constructed to assess the inflammatory potential of any diet. Then, DII provides results that can be compared to those from studies based on diverse populations. Some potential weaknesses of our study need to be highlighted. Because of its cross-sectional design, this study cannot infer causality. The sample size in this study was relatively small, which may have contributed to less precise estimates. Finally, we recognize the limitation of using a single measurement of 24-hour recall to evaluate the dietary factors to calculate the DII, as it is subject to random error that would tend to underestimate the true association between DII and the tested variables.

Our data suggest that Mexican population presents a pro-inflammatory diet with a mean DII of 1.10. However, this study did not contribute to the hypothesis that a higher BMI, higher DII, we did not find an association of overall DII with IL-17A levels. However, specific DII for niacin and B1 are predictors of IL-17A levels which are in accordance with the dietary intake of the same nutrients. In a population of young adults with a proinflammatory diet, IL-17A levels, and overall DII do not show changes between study groups. Nevertheless, it is important to monitor populations that follow a proinflammatory diet that, if perpetuated over time without intervention, could be related to the increase in inflammatory markers, as demonstrated by scientific evidence. Finally, the DII may be a good tool to characterize the diet of

the Mexican population and further explore associations with non-communicable diseases. Our results may contribute to improved dietary recommendations. They could increase public awareness of adhering to a healthy and anti-inflammatory diet.

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Table I. Sample characterization in all subjects and according to BMI status

Variables	**All** ***n* = 69**	**Healthy** ***n* = 34**	**Overweight and obesity** ***n* = 35**	***p*[1]**
Age, years	21 (18-30)	20 (18-28)	23 (19-30)	*0.03*
Sex	M 43 % F 57 %	M 44 % F 56 %	M 42 % F 58 %	
BMI (kg/m^2)	25.1 ± 3.8	22.06 ± 1.9	28.28 ± 2.3	*< 0.0001*
IL-17A (pg/mL)	2.4 (0.04-6.6)	2.5 (0.12-6.6)	2.6 (0.04-4.8)	0.99
TC (mg/dL)	124 (100-215)	123 (100-203)	124 (100-215)	0.26
LDL-C (mg/dL)	70.2 ± 21.3	64.8 ± 20.4	75.4 ± 21.2	0.07
Glucose (mg/dL)	84.6 ± 6.7	84.6 ± 5.5	84.6 ± 7.9	0.49
HDL-C (mg/dL)	45.5 ± 11.7	46.7 ± 10.6	44.2 ± 12.7	0.10
TG (mg/dL)	110 (46.9-316.7)	102.5 (46.9-250.8)	123.1 (60.7-316.7)	*0.01*
WC (cm)	86 ± 11	77.7 ± 6.5	94.3 ± 8.1	*< 0.001*
DBP (mmHg)	117 (96-147)	112.8 (96-142)	119 (99-147)	*0.02*
SBP (mmHg)	69 (51-132)	66 (51-80)	73 (57-132)	*< 0.01*
TC/HDL	2.9 ± 0.6	2.7 ± 0.6	3.1 ± 0.6	*0.02*
TyG index	4.5 ± 0.2	4.5 ± 0.2	4.6 ± 0.2	*0.01*
VAI score	1.7 (0.5-5)	1.6 (0.5-3.6)	1.9 (0.8-5)	*< 0.01*
Overall DII score	1.04 ± 1.12	0.88 ± 1.08	1.2 ± 1.1	*0.09*

BMI: body mass index; WC: waist circumference; HDL-C: high-density lipoprotein cholesterol; TC: total cholesterol; TG: triglycerides; SBP: systolic blood pressure; DBP: diastolic blood pressure; TC/HDL: total cholesterol/high-density lipoprotein cholesterol ratio; TyG index:

triglyceride-glucose index; VAI: visceral adiposity index; DII: Dietary Inflammatory Index. p^1 Healthy vs overweight and obesity.

Table II. Description of the energy values, macronutrients, micronutrients, fiber and references in all subjects and according to BMI status

Nutrient intake	**All** ***n* = 69**	**Healthy** ***n* = 34**	**Overweight and obesity** ***n* = 35**	***p*[1]**
Energy (kcal) Kcal/kg weight	1948 ± 636.4 29 ± 10.5	2043 ± 710 34.2 ± 11.2	1847 ± 540.6 23.54 ± 6.3	0.07 *< 0.0001*
Protein g/kg weight %TEV	1.1 ± 0.4 17.15 ± 6.6	1.4 ± 0.5 16.8 ± 4.6	0.95 ± 0.3 17.50 ± 8.3	*< 0.0001* 0.39
Protein g per 1000 kcal	42.7 ± 16.4	41.85 ± 11.6	43.74 ± 20.7	0.87
Carbohydrate g/kg weight %TEV	3.7 ± 1.7 52.1 ± 12.5	4.4 ± 1.8 53.1	3.0 ± 1.1 50.97 ± 11.4	*0.0003* *0.75*
CHO g per 1000 kcal	130.4 ± 31.2	133.2 ± 33.6	127.4 ± 28.7	*0.23*
Fiber g/kg weight g/day	0.25 ± 0.1 17.3 ± 10.5	0.25 ± 0.1 17.1 ± 9.6	0.22 ± 0.1 17.6 ± 11.4	0.40 0.47
Fiber g per 1000 kcal	9.6 ± 6.9	8.8 ± 5.6	10.5 ± 8.1	0.33
Lipids g/kg weight %TEV	1.0 ± 0.5 33.0 ± 11.8	1.2 ± 0.6 31.7 ± 12.0	0.9 ± 0.4 34.5 ± 11.6	*0.01* 0.19
Lipids g per 1000 kcal	36.67 ± 13.1	35.1 ± 13.4	38.2 ± 12.8	0.18
% saturated fat	8 (2-21)	9 (4-20)	8 (2-21)	0.40

% monounsaturated	9 (2-29)	9.5 (2-28)	9 (3-29)	0.49
% polyunsaturated	4 (1-17)	3.5 (1-15)	5 (1-17)	0.10
Saturated fat (g)	17.4 (3.2-60)	19.1 (3.9-57)	17 (3.2-60.9)	0.20
Monounsaturated (g)	17.9 (2-69)	19.3 (2.1-69)	17.8 /3.5-66)	0.20
Polyunsaturated (g)	9.1 (1.5-35)	9.3 (1.5-35)	8.9 (2.9-34.3)	0.35
Cholesterol (mg)	340 ± 262.8	303.5 ± 267	379.8 ± 256.9	0.06
Sugar (g)	32 (0-140)	43 (5.4-140)	22.9 (0-119.2)	0.09
Liquids (ml)	1752 ± 1002	1718 ± 975	1788 ± 1044	0.43
Vitamin A retinol (mcg)	999.1 ± 1407	852.3 ± 1200	1156 ± 1604	0.20
Vitamin B1 thiamin (mg)	1.4 ± 0.9	1.6 ± 1.1	1.22 ± 0.5	0.05
Vitamin B2 riboflavin (mg)	1.7 ± 1.1	2.0 ± 1.4	1.4 ± 24.4	*0.01*
Vitamin B6 piridoxin (mg)	3.5 ± 17.0	1.6 ± 1.6	5.6 ± 1.9	0.17
Vitamin B12 cobolamin (mg)	3.1 ± 2.1	3.21 ± 2.2	2.9 ± 119.2	0.27
Vitamin C ascorbic acid (mg)	118.4 ± 123.6	128.4 ± 128.7	107.7 ± 382.7	0.25
Folic acid (mg)	290 ± 352.1	308.2 ± 325.9	270.6 ± 1.8	0.33
Pantothenic acid (mg)	2.9 ± 1.7	2.9 ± 1.605	2.8 ± 1.8	0.33
Niacin (mg)	18.0 ± 13.7	20.4 ± 16.2	15.4 ± 10.2	0.07
Vitamin E (mg)	4.4 ± 4.5	4.1 ± 4.9	4.7 ± 4.6	0.30
Calcium (mg)	1083 ± 623.1	1175 ± 672.5	984.7 ± 560.2	0.11
Iron (mg)	16.2 ± 18.1	19.0 ± 24.8	13.3 ± 4.4	0.10
Potassium (mg)	2353 ± 1130	2417 ± 903.7	2284 ± 1343	0.32
Magnesium (mg)	319.8 ± 266	388.6 ± 32	246.4 ± 156.	*0.01*

	.2	6.4	1	
Sodium (mg)	2200 ± 149 3	2471 ± 157 5	1911 ± 1366	0.07
Phosphorus (mg)	907.2 ± 474 .2	981.8 ± 48 3.9	827.6 ± 458. 2	0.10
Selenium	62.45 ± 32. 9	68.59 ± 36. 2	55.9 ± 28.08	0.06
Zinc (mg)	7.0 ± 3.7	7.6 ± 3.9	6.4 ± 3.4	0.08

Total energy value percent (%TEV). p^1 Healthy vs overweight and obesity.

Table III. Multivariate linear regression analysis that evaluates the association between IL-17A

Model 1

		β	*p*	r^2
IL-17A	TC/HDL	-0.009	0.949	0.031
	TyG	-0.277	0.200	
	VAI	0.152	0.557	
	BMI	-0.009	0.949	

Model 2

		β	*p*	r^2
IL-17A	TG	-0.140	0..329	0.068
	HDL	0.108	0.442	
	Glucose	0.174	0.193	
	SBP	0.015	0.935	
	DBP	0.098	0.593	
	WC	-0.077	0.614	

Model 3

		β	*p*	r^2
IL-17A	% fat	-1.006	0.205	0.083
	% sat fat	.902	0.223	
	%MUFA	0.396	0.467	
	%PUFA	0.323	0.653	
	g sat fat	0.003	0.275	
	cholesterol	-.224	0.226	

Model 4

		β	*p*	r^2
IL-17A	Zinc	0.065	0.776	0.275
	Selenium	-0.071	0.690	
	Fosfote	0.100	0.680	
	Sodioum	-0.119	0.594	
	Magnesium	-0.193	0.329	
	Potasium	0.286	0.349	
	Iron	0.175	0.755	
	Calcium	-0.127	0.528	

Vit E	-0.044	0.768
Niacin	-0.793	*0.021*
Pantoteic acid	0.120	0.530
Folic acid	-0.566	0.051
C	-0.200	0.403
B12	-0.140	0.541
B6	0.013	0.928
B2	-0.196	0.658
B1	1.087	*0.043*
A	0.138	0.403

Table IV. Multivariate linear regression analysis that evaluates the association between IL-17A with specific DII

Model 1			
	β	***p***	***r²***

IL-17A	DII niacin	0.275	0.031	0.075

Model 2

		β	*p*	r^2
IL-17A	DII niacin	0.537	0.002	0.148
	DII B1	-0.376	0.029	

Model 3

		β	*p*	r^2
IL-17A	DII niacin	0.415	0.021	0.204
	DII B1	-0.617	0.004	
	DII iron	-0.413	0.048	

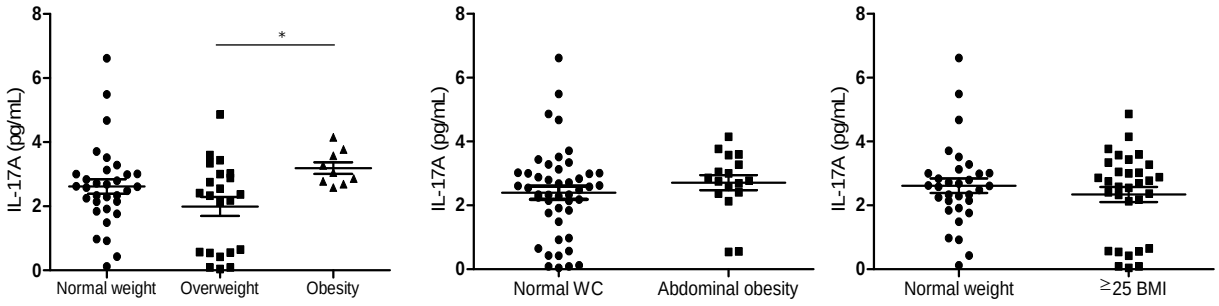

Figure 1. IL-17A levels according to body mass index.

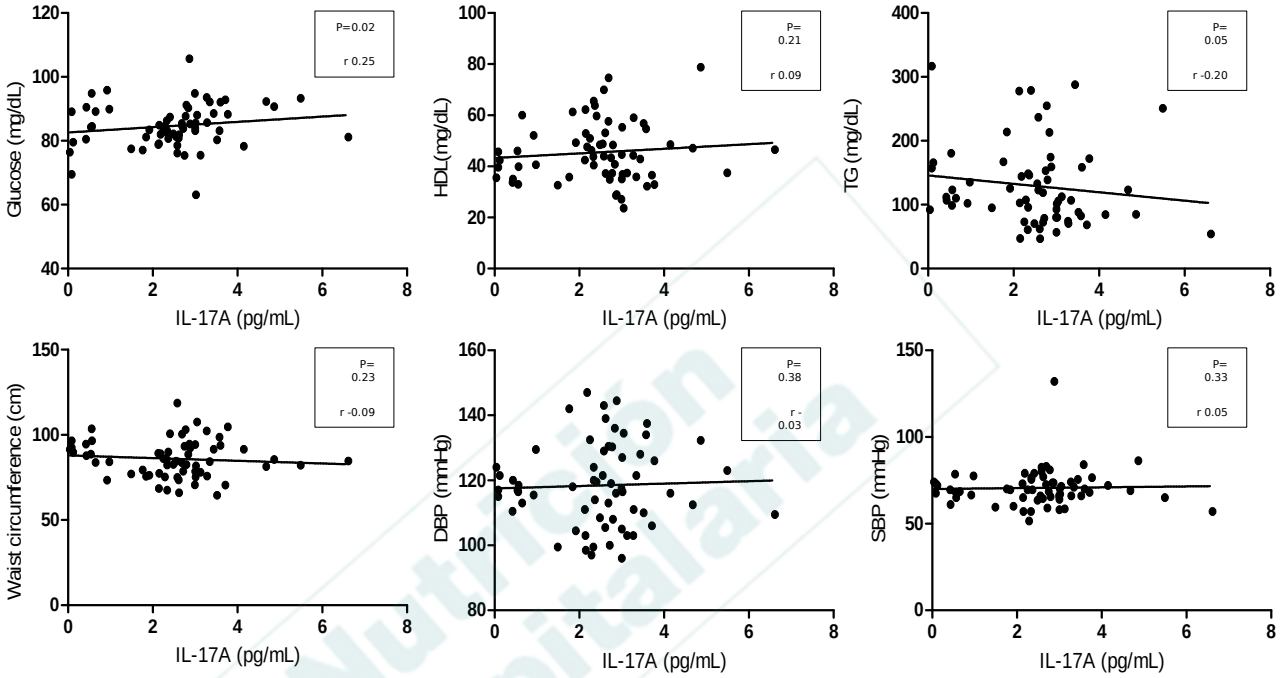

Figure 2. Associations between IL-17A levels and anthropometric parameters, traits of metabolic syndrome, and dietary intake.

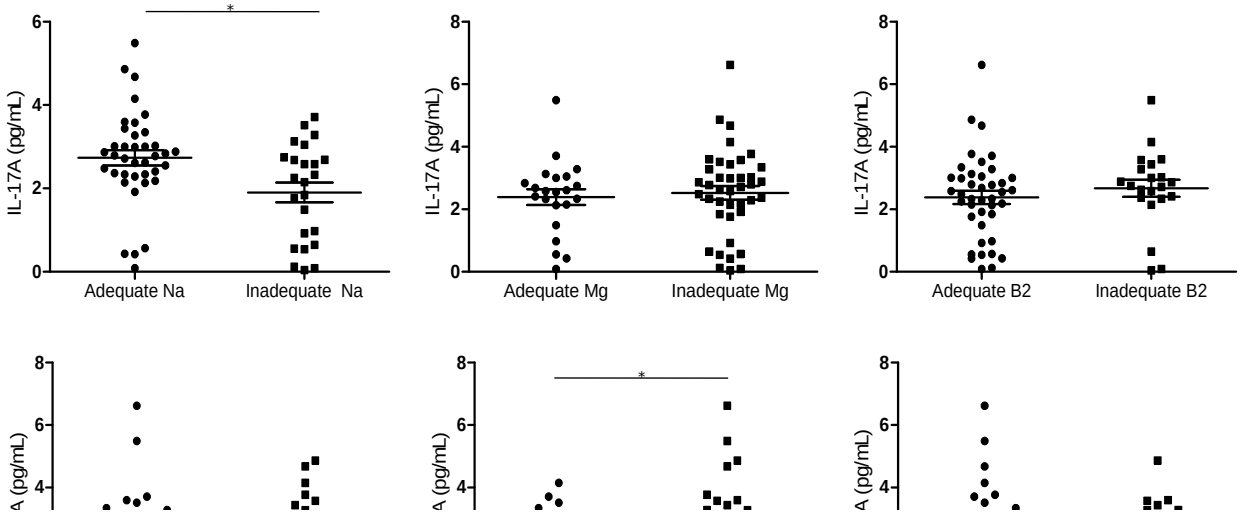

Figure 3. IL-17A levels according to the adequacy in the intake of micronutrients.