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Original Article

Correlation between cytokine profile and metabolic abnormalities in young subjects

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Abstract: Chronic systemic inflammation characterized by elevated circulating cytokines and chemokines, is an important feature of obesity. The aim of this study was to investigate the relationship between cytokines and high sensitivity C-reactive protein (hsCRP) with metabolic alterations in obese young subjects. A total of 100 subjects were recruited from the state of Guerrero, Mexico. All individuals had an age range of 18 to 30 years old and were divided into two groups: normal-weight (n = 50) and obese subjects (n = 50). The levels of circulating cytokines (IL-6, IL-1β, IFN-γ, IL-2, IL-12, IL-4, TNF-α, IL-13, IL-17 and IL-10) were measured using a bead based multiplex system. MIF levels were determined by ELISA. Serum hsCRP was analyzed by turbidimetry. We found increased serum concentrations of IL-6 and hsCRP in subjects with overall and abdominal obesity. Furthermore, subjects with hypertriglyceridemia had higher serum hsCRP levels compared to those subjects without dyslipidemia. In addition, the results showed a positive correlation between adiposity measures and circulating levels of IL-6 and hsCRP, but a negative correlation with IL-10 levels. No significant differences were found for serum levels of MIF, IL-1β, IFN-γ, IL-2, IL-12, IL-4 and IL-10 neither between both study groups nor according to metabolic abnormalities. The results show that hsCRP, IL-6 and IL-10 are the main inflammatory markers related to obesity and/or dyslipidemia in young subjects. Therefore, these markers may be useful in the early detection of cardiovascular risk in obese population.

Keywords: Obesity, inflammation, cytokines, C-reactive protein

Introduction

Obesity is associated with chronic low-grade systemic inflammation and is one of the key factors for the development of metabolic diseases such as insulin resistance (IR), type 2 diabetes mellitus (T2DM), hypertension, hyperlipidemia, atherosclerosis, metabolic syndrome and cardiovascular disease (CVD) [1, 2]. There is accumulating evidence that deregulated production of cytokines in obesity contributes to the low-grade chronic inflammation, which is recognized as an important player in the pathogenesis of obesity-associated comorbidities [3, 4]. Several studies have reported increased circulating levels of a wide range of inflammatory markers including C-reactive protein (CRP), interleukin 6 (IL-6), interleukin 8 (IL-8), monocyte chemoattractant protein 1 (MCP-1) and macrophage migration inhibitory factor (MIF) in obese and T2DM individuals, and were positively correlated to BMI (body mass index) and waist circumference [5-7].

Previous studies in our population have reported the prevalence of obesity, hypertension and other cardiovascular risk factors in children and adults [8-10]. In addition to traditional risk factors for the development of T2DM and CVD such as obesity, hypertension and dyslipidemias, chronic inflammation is now recognized as an important risk factor involved in the pathogenesis of these diseases [11, 12]. Several studies have reported the relationship of pro-inflammatory cytokines such as tumor necrosis factor alpha (TNF-α) and IL-6 with insulin resistance and atherogenesis [13, 14]. Furthermore, IL-6 and activin-A were recognized as major risk factors for cardiovascular events and mortality in T2DM subjects [15]. However,

the role of the T-helper (Th) 1 and Th2 cytokines has not been sufficiently studied in obesity, T2DM and CVD. Previously, it was reported a mixed Th1-Th2 serum cytokine profile in subjects with metabolic syndrome (MetS) as a major risk factor for T2DM (if not present already) and CAD (Coronary Artery Disease) [16]. In another study, T2DM subjects showed a mixed Th1-Th2 profile and T2DM-CAD subjects presented enhanced Th1 polarization similar to that of CAD subjects with further reduction in their Th2 cytokine levels [17]. This study assessed the relationship between a cytokine profile and high sensitivity C-reactive protein with metabolic alterations in obese young subjects.

Materials and methods

Participants

A total of 100 subjects were recruited from the state of Guerrero, Mexico. All individuals had an age range of 18 to 30 years old, and were divided into two groups: 50 with normal-weight and 50 obese subjects. Subjects were selected from the general population and exclusion criteria were acute or chronic infections, being under any medication, pregnancy and presence of autoimmune or chronic inflammatory diseases. All subjects gave written informed consent prior enrollment in the study. This protocol was approved by the Research Ethics Committee of the University of Guerrero.

Anthropometric measurements

Body weight was determined in subjects wearing light clothes and without shoes, using a body composition monitor (Tanita TBF-300 GS, Arlington, USA). The height was measured to the nearest 0.1 cm using a stadiometer (Seca, Hamburg, Germany). From these measurements, BMI was calculated (BMI = weight/height^2, kg/m^2). Subjects were classified by BMI: obese ≥ 30 kg/m^2 and normal-weight < 24.9 kg/m^2, based on the criteria of World Health Organization [18]. The body circumferences were measured with an anthropometric tape accurate to within ± 0.1 cm (Seca, 201, Hamburg, Germany).

Biochemical analysis

A venous blood sample of 5 mL was obtained from each subject after at least 12 hours fasting. Biochemical parameters, such as total cholesterol (TC), HDL-cholesterol (HDL-c), LDL-cholesterol (LDL-c), triglycerides (TG) and fasting glucose levels were determined in serum samples by enzymatic colorimetric methods with commercially available kits (Spinreact, Spain). Abnormal biochemical levels were identified when TC ≥ 200 mg/dL, TG ≥ 150 mg/dL, and glucose > 110 mg/dL, based on the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (NCEP-ATPIII) [19].

Determination of serum hsCRP and cytokines levels

The levels of cytokines (IL-6, IL-1β, IFN-γ, IL-2, IL-12, IL-4, TNF-α, IL-13, IL-17 and IL-10) were measured in serum samples using the Human Cytokine Magnetic 10-plex custom kit (Invitrogen Life Technologies, USA) and the MAGPIX® System (Luminex, USA). Levels of TNF-α, IL-13 and IL-17 were below the detection limit of the multiplex assay and thus were excluded from the statistical analysis. Serum samples were stored at -80°C until the day of the assay and processed according to the manufacturer's instructions. The fluorescence values of 100 events per region were considered as quantification criteria. We performed serial dilutions of the recombinant standards provided in the assay to generate standard curves of the cytokines in duplicate. Curves were adjusted to a logistic regression model (5 parameters) and showed correlation coefficients (R^2) above 0.95. Quantitative levels of cytokines in samples were interpolated from the standard curves and reported in pg/mL.

The determination of serum MIF levels was performed by enzyme-linked immunosorbent assay (LEGEND MAX™ Human Active MIF ELISA Kit, BioLegend) according to manufacturer's instructions. The MIF assay sensitivity was 17.4 ± 9.2 pg/mL. High sensitivity C-reactive protein (CRP) was measured by turbidimetry in the BS-120 chemistry analyzer (MINDRAY, China), the detection limit was less than 1 mg/L.

Statistical analysis

Data analysis was performed using STATA software (v.11.0) and GraphPad Prism (v 5.0). Differences in characteristics between groups were analyzed using the chi-square test for categorical variables (data presented as percent-

Table 1. Anthropometric, biochemical and inflammatory characteristics by group

Variable	Normal-weight N = 50	Obesity N = 50	*P* value
Age (years)*	20 (18-28)	22 (18-28)	0.15
Gender n (%)†			0.69
Male	24 (48)	22 (44)	
Female	26 (52)	28 (56)	
Weight (kg)*	59.7 (43.1-73)	89.3 (78.5-109)	< 0.001
BMI (kg/m^2)*	22.4 (18.7-24.6)	33.4 (30-38.8)	< 0.001
Waist circumference (cm)*	79.3 (70.5-89)	104 (90-120.5)	< 0.001
Hip circumference (cm)*	96 (87-104)	115.3 (106-131)	< 0.001
Waist-hip-ratio‡	0.83 ± 0.05	0.9 ± 0.07	< 0.001
Body fat mass (%)*	17.95 (9.5-32.4)	39.5 (25.7-47.4)	< 0.001
Body fat mass (kg)*	11.8 (5.2-20.9)	34.4 (23.9-47.9)	< 0.001
Metabolic profile			
Glucose (mg/dL)*	84.5 (73-104)	87.5 (76-107)	0.05
Cholesterol (mg/dL)‡	157 ± 29.6	166 ± 31	0.13
Triglycerides (mg/dL)*	84 (42-188)	119 (43-358)	0.002
LDL-c (mg/dL)*	109 (69-207)	102 (69-187)	0.42
HDL-c (mg/dL)*	40.5 (28-68)	39 (27-62)	0.68
Inflammatory markers			
MIF (ng/mL)*	3.3 (0.9-7.2)	2.5 (1.1-6.6)	0.15
IL-6 (pg/mL)*	1.3 (0.42-7.04)	2.7 (0.42-12.13)	0.004
IL-1β (pg/mL)*	4.5 (0.6-12.01)	4.8 (0.8-9.2)	0.89
IFN-γ (pg/mL)*	2.2 (0.95-5.6)	2.2 (0.95-4.2)	0.28
IL-2 (pg/mL)*	0.43 (0.05-6.6)	0.23 (0.05-5.5)	0.56
IL-12 (pg/mL)*	97 (43.2-212.7)	108 (56.5-255.6)	0.41
IL-4 (pg/mL)*	3.96 (2.9-9.7)	3.96 (2.9-16.8)	0.85
IL-10 (pg/mL)*	2.3 (2.04-4.7)	2.3 (1.82-3.9)	0.07
hsCRP (mg/L)*	0.57 (0.16-1.78)	1.28 (0.4-3.42)	< 0.001

*Data are presented as median and 5th to 95th percentile. Mann-Whitney test. †Data are presented as n and percentage. Chi-square test. ‡Data are presented as mean ± SD. Student *t*-test. Abbreviations: BMI, Body Mass Index; LDL-c, Low Density Lipoprotein-Cholesterol; HDL-c, High Density Lipoprotein-Cholesterol; Macrophage migration inhibitory factor, MIF; Interferon-γ, IFN-γ; Interleukin, IL; hsCRP, High sensitivity C-reactive protein.

ages), Student's *t*-test for parametric variables (data presented as mean ± SD) and Mann-Whitney *U*-test for nonparametric variables (data presented as median and 5th to 95th percentiles). Correlations between variables were expressed as Spearman's correlation coefficients. $P < 0.05$ was considered statistically significant.

Results

Anthropometric, biochemical and inflammatory characteristics by group are summarized in **Table 1**. As expected, obese subjects had higher body weight, BMI, waist and hip circumferences, waist-hip-ratio and body fat mass ($P < 0.001$) as well as triglycerides concentrations ($P = 0.002$) but no total cholesterol, HDL-c and LDL-c, in comparison to normal-weight subjects. In the comparative analysis of inflammatory markers levels by group we only found a significant increase in both IL-6 ($P = 0.004$) and hsCRP concentrations ($P < 0.001$) in obese subjects in comparison to normal-weight subjects. There were no significant differences for MIF, IL-1β, IFN-γ, IL-2, IL-12, IL-4 and IL-10 serum levels between groups.

In **Table 2**, are shown the concentrations of serum inflammatory markers that were analyzed according to metabolic abnormalities in all subjects. We found increased IL-6 ($P = 0.0007$) and hsCRP serum levels ($P < 0.001$), but significantly decreased IL-10 levels ($P = 0.013$) in subjects with abdominal obesity when compared to those without abdominal obesity. Besides, subjects with hypertriglyceridemia had higher serum hsCRP levels ($P = 0.0034$) than those subjects without dyslipidemia.

The correlation between inflammatory markers and anthropometric measures are shown in **Table 3**. Levels of hsCRP were significantly correlated with all body measures and adiposity ($P < 0.001$). Similarly, IL-6 concentrations were correlated with most measures but not with waist-hip-ratio. IL-10 levels were negatively correlated with all measures, but only significantly with hip circumference ($P = 0.023$) and body mass ($P = 0.03$).

Table 4 shows the correlation between serum cytokine concentrations that was performed in the total sample. We observed a positive cor-

Table 2. Inflammation markers levels according to metabolic abnormalities

Variable	MIF	IL-6	IL-1β	IFN-γ	IL-2	IL-12	IL-4	IL-10	CRP
Abdominal obesity									
No	3.3 (0.9-7.2)	1.25 (0.4-7.04)	4.4 (0.8-10.1)	2.2 (0.95-4.2)	0.43 (0.1-4.7)	95.4 (46.2-194.4)	3.96 (2.9-9.7)	2.3 (2-3.9)	0.62 (0.16-1.8)
Yes	2.5 (1.1-6.4)	2.7 (0.42-9.12)	4.9 (0.9-8.4)	2.2 (0.95-4.2)	0.14 (0.1-5.5)	109 (56.5-255.6)	3.96 (2.9-14.4)	2.3 (1.8-3.4)	1.4 (0.4-3.42)
	$P = 0.11$	$P = 0.0007$	$P = 0.60$	$P = 0.86$	$P = 0.60$	$P = 0.31$	$P = 0.76$	$P = 0.013$	$P < 0.001$
Glucose (> 110 mg/dL)									
No	2.7 (1-7.2)	1.8 (0.42-7.04)	4.6 (0.8-9.9)	2.2 (0.95-4.2)	0.23 (0.1-5.5)	107 (52.1-194.4)	3.96 (2.9-14.4)	2.3 (1.8-3.7)	0.9 (0.2-2.42)
Yes	4.4 (1.5-6.4)	2.7 (0.7-9.12)	4.4 (0.9-8.6)	2.9 (0.95-3.5)	0.1 (0.1-0.43)	255.6 (62.3-329.2)	3.96 (3.96-3.96)	2.3 (1.8-4.9)	1.8 (0.28-4.64)
	$P = 0.50$	$P = 0.62$	$P = 0.88$	$P = 0.69$	$P = 0.29$	$P = 0.23$	$P = 0.55$	$P = 0.91$	$P = 0.36$
Total cholesterol (≥ 200 mg/dL)									
No	2.9 (1.1-7.2)	2.1 (0.42-7.9)	4.5 (0.8-9.9)	2.2 (0.95-4.2)	0.23 (0.1-5.5)	107 (56.5-195)	3.96 (2.9-14.4)	2.3 (1.8-3.9)	0.9 (0.22-2.41)
Yes	2.0 (0.9-5.2)	1.3 (0.42-4.1)	4.8 (4-8.4)	2.2 (1.6-4.2)	0.14 (0.1-2.0)	80.3 (46.2-212.7)	3.96 (2.9-6.2)	2.3 (2-2.73)	1.04 (0.15-2.5)
	$P = 0.088$	$P = 0.22$	$P = 0.46$	$P = 0.86$	$P = 0.52$	$P = 0.12$	$P = 0.69$	$P = 0.17$	$P = 0.81$
Triglycerides (≥ 150 mg/dL)									
No	2.8 (1.1-7.2)	2.1 (0.42-7.9)	4.4 (0.8-9.9)	2.2 (0.95-4.2)	0.43 (0.1-5.5)	107 (53.6-212.7)	3.96 (2.9-9.7)	2.3 (2-3.4)	0.72 (0.18-3.2)
Yes	2.7 (0.9-6.4)	1.8 (0.42-4.1)	4.9 (0.8-9.3)	1.9 (0.95-5.6)	0.23 (0.1-4.2)	107 (52.1-195.1)	3.96 (2.9-22.9)	2.3 (1.8-3.9)	1.13 (0.51-2.27)
	$P = 0.81$	$P = 0.67$	$P = 0.81$	$P = 0.41$	$P = 0.91$	$P = 0.99$	$P = 0.95$	$P = 0.34$	$P = 0.0034$

Data are presented as median (5^{th}-95^{th} percentile). Mann-Whitney test.

Table 3. Correlation between inflammatory markers and anthropometric measures

Variables	CRP		IL-6		IL-10	
	r	*P*	*r*	*P*	*r*	*P*
Weight	0.49	< 0.001	0.26	0.02	-0.16	0.16
BMI	0.55	< 0.001	0.38	0.0008	-0.22	0.05
Waist circ.	0.56	< 0.001	0.34	0.003	-0.19	0.09
Hip circ.	0.51	< 0.001	0.37	0.001	-0.26	0.023
Waist-hip-ratio	0.39	0.0001	0.19	0.10	-0.04	0.74
Body mass (%)	0.52	< 0.001	0.38	0.0008	-0.31	0.005
Body mass (kg)	0.55	< 0.001	0.36	0.002	-0.25	0.03

r = Spearman correlation coefficient; *P* = *P* value. Abbreviations: BMI, body mass index; Waist circ., waist circumference; Hip circ., hip circumference.

relation between IL-6 with IFN-γ (r = 0.24, *P* = 0.04) and IL-4 (r = 0.25, *P* = 0.03); IFN-γ with IL-2 (r = 0.37, *P* = 0.001), IL-12 (r = 0.23, *P* = 0.04), IL-4 (r = 0.30, *P* = 0.007) and IL-10 (r = 0.26, *P* = 0.023); IL-2 with IL-4 (r = 0.53, *P* < 0.001) and IL-10 (r = 0.31, *P* = 0.007); IL-12 with IL-4 (r = 0.24, *P* = 0.04) and IL-10 (r = 0.29, *P* = 0.01); and IL-4 with IL-10 (r = 0.39, *P* = 0.0005).

Discussion

In this study, circulating levels of hsCRP and a panel of ten cytokines and their relationship with obesity were studied in Mexican young subjects; we found increased serum levels of IL-6 and hsCRP in subjects with overall and abdominal obesity. Individuals with hypertriglyceridemia had higher serum hsCRP levels compared to those without this abnormality. Moreover, we detected a positive correlation between adiposity measures and circulating levels of IL-6 and hsCRP, but a negative correlation of these parameters with IL-10 levels.

The chronic low-grade systemic inflammation, characterized by elevated circulating cytokines and chemokines, is a prominent feature of obesity. In both children and adults, several studies have shown high circulating levels of IL-6, IL-18, MCP-1 and CRP in obese individuals [5, 6, 20, 21]. Similarly, we found increased IL-6 and hsCRP serum concentrations in abdominal and overall obese subjects compared with normal-weight subjects. It is known that during obesity, IL-6 is released by the visceral adipose tissue into the portal circulation and that CRP is mainly synthesized in the liver in response to IL-6 stimulation, which would explain their proportional increase [22]. CRP has an important effect on amplifying the inflammatory response and is used as a marker of obesity-related inflammation and as a predictor of cardiovascular events and diabetes [23, 24].

In our study, we did not find significant differences between MIF, IL-1β, IFN-γ, IL-2, IL-12, IL-4 or IL-10 serum levels when comparing normal-weight versus obese, nor according to metabolic abnormalities. However, previous reports on serum levels of these cytokines were inconsistentin subjects with obesity, T2DM and MetS. IL-12 levels were elevated in obesity [25], MIF levels were increased in obese adolescents [26], and high circulating levels of IL-12, IFN-γ, IL-4, IL-5 and IL-13 were reported in subjects with MetS [16]. In obese adolescent girls, IL-1β, IL-4 and IL-5 levels were higher in those with central obesity than in controls [27]. Also, serum levels of IL-5, IL-10, IL-12, IL-13, IFN-γ and TNF-α were found elevated in obese subjects [28]. In other studies, inconsistent results have been reported regarding IL-10 concentrations; increased levels of this cytokine were found in obese women [29], whereas a reduction on IL-10 was reported in other study evaluating obese women, additionally, no changes were detected for this cytokine after body weight reduction in response to diet [30]. Another report detected increased IL-10 levels associated with visceral fat loss [31]. Furthermore, one of the most studied comorbidities associated with obesity is T2DM. Previously, it was reported that the presence of T2DM favors a Th1 cytokine profile in subjects with T2DM and CAD, with suppression of the Th2 cytokine profile [17]. However, it is important to mention that in our study, obese subjects do not have T2DM, only 3 obese patients had impaired fasting glucose.

In addition, the distribution of the number of metabolic abnormalities in obese subjects was as follows: 22% displayed at least one alteration, 48% exhibited two abnormalities and 30% presented three or more metabolic alterations. Thus, it is possible that obese young subjects may have an early inflammatory process where circulating levels of IL-6 and hsCRP are increased but the levels of other cytokines are difficult to be detected in periph-

Table 4. Correlation between serum cytokine levels

Cytokines	MIF		IL-6		IL-1β		IFN-γ		IL-2		IL-12		IL-4	
	r	*P*	*r*	*P*	*r*	*P*	*r*	*P*	*r*	*P*	*r*	*P*	*r*	*P*
MIF	-	-	-	-	-	-	-	-	-	-	-	-	-	-
IL-6	0.18	0.11	-	-	-	-	-	-	-	-	-	-	-	-
IL-1β	-0.13	0.27	0.12	0.30	-	-	-	-	-	-	-	-	-	-
IFN-γ	-0.05	0.68	0.24	0.04	0.19	0.09	-	-	-	-	-	-	-	-
IL-2	-0.05	0.69	0.18	0.13	0.15	0.18	0.37	0.001	-	-	-	-	-	-
IL-12	0.025	0.83	0.20	0.08	0.0001	0.99	0.23	0.046	0.16	0.16	-	-	-	-
IL-4	0.03	0.81	0.25	0.03	0.035	0.76	0.30	0.007	0.53	< 0.001	0.24	0.04	-	-
IL-10	-0.03	0.78	0.14	0.23	-0.013	0.91	0.26	0.023	0.31	0.007	0.29	0.01	0.39	< 0.001

R = Spearman correlation coefficient; *P* = *P* value.

eral blood. Also, our population of obese subjects had fewer metabolic abnormalities in comparison to former studies assessing other comorbidities. Therefore obesity alone seems to be insufficient to induce pro-inflammatory cytokine profile at an early age and the presence of other abnormalities is probably required for an increase on Th1 cytokine profile to occur. Besides, other factors that may contribute to the differences between studies are the sample size and their inclusion criteria, as well as the racial influence among populations with different ethnic origin. Despite differences in some studies, it appears to be an unregulated production of pro-inflammatory and anti-inflammatory cytokines in obesity, which probably play an important role in the pathophysiology of the disease and the development of metabolic comorbidities.

Furthermore, we detected that IL-6 and hsCRP levels were correlated with body adiposity, whereas IL-10 levels were negatively correlated with body fat mass. Similarly, other studies have reported positive correlations between IL-6 and CRP with BMI, waist and hip circumferences, and body fat percentage [5, 32, 33]. IL-6 and CRP are strongly associated because one of the main effects of IL-6 is the induction of hepatic CRP production. Therefore, both markers appear to simultaneously increase as a consequence of the inflammatory condition in obese subjects. However, the fact that hsCRP was significantly elevated in abdominal obesity and hypertriglyceridemia, and that cytokines may drastically vary due to external influences, hsCRP may be considereda better marker of cardiometabolic risk in comparison to cytokines.

Obesity is associated with a chronic inflammatory response, abnormal adipokines production, and the activation of some pro-inflammatory signaling pathways, resulting in the induction of several biological markers of inflammation. However, the exact mechanisms have not yet been clearly elucidated. Recently, several mechanisms have been proposed as contributors to obesity-related inflammation: 1) hyperplasic and hypertrophic adipocytes synthesize pro-inflammatory adipokines such as TNF-α and IL-6; 2) macrophages migrate into the adipose tissue, where polarization from M2 to M1 macrophages is enhanced, but this polarization state depends on environmental stimuli [34]; 3) the Th2/Th1 ratio and Treg cell activity is reduced [35]. These processes are suggested to lead to a shift in cytokine levels in obesity.

In this study, we have observed a correlation between pro- and anti-inflammatory cytokines. IFN-γ was correlated with IL-6, IL-12, IL-4 and IL-10; IL-4 was correlated with IL-6, IL-2, IL-12 and IL-10, and IL-10 was correlated with IL-2 and IL-12. Some previous studies on correlations between cytokines and associated comorbidities were reported in obesity. In 2006, Ranjbaran and colleagues found a correlation between pro- and anti-inflammatory cytokines in patients with coronary atherosclerosis; they demonstrated a relationship between IFN-γ with IL-12 and IL-10 levels [36]. IL-12 is a pro-inflammatory cytokine that induces the production of IFN-γ in T cells and natural killer cells, and promotes the differentiation of Th1 cells [37]. IFN-γ is a key mediator for IL-12 and IL-6 release by classically activated macrophages [38]. An inverse relationship was found between circulating levels of IL-10 and adiposity mea-

sures. IL-10 is a potent anti-inflammatory cytokine produced mainly by monocytes and macrophages in response to inflammatory stimulus such as IL-6 and also by regulatory T cells (T_{reg}) [39]. One may speculate that IL-10 produced by obese subjects is insufficient to decrease their inflammatory state. In fact, a recent study by Wagner et al. reported decreased circulating T_{reg} in obese individuals compared with non-obese. Moreover, the proportion of circulating T_{reg} cells was inversely correlated with indices of adiposity such as body weight and BMI, particularly in obese subjects, supporting the idea of defective anti-inflammatory pathways in obese subjects [40].

The main limitation of the present study is the small sample size. Additionally, due to the cross-sectional nature of our study we cannot determine the causal relationship between inflammatory markers and cardiometabolic abnormalities.

In conclusion, our study show that hsCRP, IL-6 and IL-10 are the main inflammatory markers related to obesity and/or hypertriglyceridemia. Therefore, these biomarkers may be a link between obesity and cardiometabolic abnormalities in young subjects.

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Disclosure of conflict of interest

None.

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