We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

6,900
Open access books available

186,000
International authors and editors

200M
Downloads

Our authors are among the

154
Countries delivered to

TOP 1%
most cited scientists

12.2%
Contributors from top 500 universities

WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.
For more information visit www.intechopen.com

Chapter 10

Prevalence of Metabolic Syndrome in Obese Pediatric Population: Relation to Serum Leptin Concentrations

Teodoro Durá-Travé, Fidel Gallinas-Victoriano,
Leyre Lloreda-Martín, Alberto Ríos-Muñoz,
Inés Niyubahwe and Ander Ernaga-Lorea

Additional information is available at the end of the chapter

http://dx.doi.org/10.5772/65098

Abstract

Childhood obesity represents the most relevant nutritional disorder in our environment. This study examines the prevalence of metabolic syndrome in an obese pediatric population and its relation to serum leptin concentrations. A cross-sectional clinical and metabolic study was accomplished in a group of 106 obese children (47 males and 59 females). Patients were classified into prepubertal group (Tanner stage I) and pubertal group (Tanner stages II–V). Prevalence of insulin resistance [homeostasis model assessment (HOMA)], hypertriglyceridemia, low high-density lipoprotein (HDL) and arterial hypertension (HTA) was 38.7, 45.3, 28.3 and 33.8%, respectively. Metabolic syndrome prevalence (30.2%) was significantly higher in the pubertal group (38%) than the prepubertal group (23.2%). There was a positive correlation between leptin and body mass index (BMI) (r = 0.529), leptin and HOMA indexes (r = 0.562) and leptin and triglycerides (r = 0.314). In addition, there was a positive correlation between HOMA indexes and triglycerides (r = 0.596). Clinical and metabolic disorders associated with obesity and related to the so-called metabolic syndrome are already present in pediatric population. Leptin could play an important role in the etiopathogenesis of the metabolic syndrome.

Keywords: childhood obesity, insulin resistance, leptin, metabolic syndrome, triglycerides, blood pressure

INTECH
open science | open minds

© 2017 The Author(s). Licensee InTech. This chapter is distributed under the terms of the Creative Commons Attribution License (http://creativecommons.org/licenses/by/3.0), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited. CC BY

1. Introduction

Childhood obesity represents the most relevant nutritional disorder in our environment [1, 2]. It usually initiates at early stages in life, when child feeding depends—almost exclusively—on feeding habits and preferences in a family setting; it is subsequently exacerbated (by the time of school attendance and/or adolescence), probably in relation to the adoption of unhealthy feeding habits and lifestyle [3, 4].

Study	Excess adiposity	Hypertension	Dyslipidemia	Abnormal glucose homeostasis
Cook et al. [12]	WC ≥ 90th percentile	SBP or DBP ≥ 90th percentile	Triglycerides ≥ 110 mg/dl or HDL-chol ≤ 40 mg/dl	Fasting glucose ≥ 110 mg/dl
De Ferranti et al. [13]	WC ≥ 75th percentile	SBP ≥ 90th percentile	Triglycerides ≥ 100 mg/dl HDL-chol ≤ 50 mg/dl	Fasting glucose ≥ 110 mg/dl
Weiss et al. [14]	BMI ≥ 97th percentile	SBP ≥ 95th percentile	Triglycerides ≥ 95th percentile or HDL-chol ≤ 5th percentile	OGTT: glucose at 120 min >140 and <200 mg/dl
Cruz et al. [15]	WC ≥ 90th percentile	SBP or DBP ≥ 90th percentile	Triglycerides ≥ 90th percentile or HDL-C ≤ 10th percentile	OGTT: glucose at 120 min >140 and <200 mg/dl
Viner et al. [16]	BMI ≥ 95th percentile	SBP ≥ 95th percentile	Triglycerides ≥ 150 mg/dl or HDL-chol ≤35 mg/dl or total-chol ≥ 95th percentile	Fasting glucose ≥ 110 mg/dl or OGTT: glucose >140 and <200 at 2 h or fasting insulin ≥15 mU/L (prepubertal) or ≥30 (pubertal)
Ford et al. [17]	WC ≥ 90th percentile	SBP or DBP ≥ 90th percentile	Triglycerides ≥ 110 mg/dl or HDL-chol ≤ 40 mg/dl	Fasting glucose ≥ 110 mg/dl
Lambert et al. [18]	BMI ≥ 85th percentile	SBP ≥ 75th percentile	Triglycerides ≥ 90th percentile or HDL-C ≤ 10th percentile	Fasting glucose ≥ 110 mg/dl or fasting insulin ≥ 75th percentile
Zimmet et al. [19]	WC ≥ 90th percentile	SBP ≥ 130 or DBP ≥ 85 mmHg	Triglycerides ≥ 75th percentile or HDL-chol ≤ 25th percentile	Fasting glucose ≥ 110 mg/dl

WC, waist circumference; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; HDL-chol, high-density lipoprotein cholesterol; OGTT, oral glucose tolerance test.

Table 1. Criteria for the diagnosis of the pediatric metabolic syndrome (all definitions considered a child as having the metabolic syndrome when three or more of the following characteristics were present).

Additional studies—except for uncommon situations such as endocrine, genetic or metabolic pathologies, which justify excess body weight—are used for the diagnosis and/or early detection of metabolic complications and, particularly, the metabolic syndrome. This syndrome is characterized by a cluster of symptoms associated with obesity, such as insulin resistance, arterial hypertension (HTA) and dyslipidemia, and its interest lies in the high

http://dx.doi.org/10.5772/65098

predictive value for cardiovascular disease and type 2 diabetes in adulthood, especially when it is already present in school children and/or adolescents [5–11].

In the pediatric age, there are no clearly defined parameters for its diagnosis, being several different criteria proposed [12–19] (**Table 1**) on the basis of an extrapolation from clinical guides of adult populations: WHO [20], the National Cholesterol Education Program's Adult Treatment Panel III [21], the European Group for the Study of Insulin Resistance [22] and the International Diabetes Federation (IDF) [23]; this would explain the disparity in published data with respect to the applied criteria [12–16]. Even when the International Diabetes Federation (IDF) refers to the inability for diagnosis in school age, epidemiological data allow suspecting that metabolic syndrome or its components are already present at early stages [23–29].

Leptin is an adipocytokine that, in addition to multiple neuroendocrine functions, has a role in the regulation of energy balance as well as in carbohydrate and lipid metabolism and arterial pressure regulation. In this way, many authors have suggested that leptin might be involved in the etiopathogenesis of metabolic syndrome [30–32].

The aim of this work is to determine the prevalence of metabolic syndrome and its relation to serum leptin concentrations in a group of obese pediatric population.

2. Methods

2.1. Patients

A clinical assessment and metabolic study was accomplished in all patients diagnosed with obesity who attended follow-up consultation within the year 2014. Clinical evaluation was conducted in one of the three offices of the Pediatric Endocrinology Unit of the Navarra Hospital Complex. Pubertal stage was determined in each patient according to Tanner's criteria, and patients were classified into two different groups: prepubertal group (Tanner stage I) and pubertal group (Tanner stages II–V).

All those patients with personal history of endocrine disease, malformation syndromes or iatrogenic obesity (drug treatments) were excluded.

The metabolic syndrome was defined by modified Cook's criteria [12] as the manifestation of at least three of the following features: low HDL-cholesterol (<40 mg/dl), hypertriglyceridemia (TG > 110 mg/dl), obesity, arterial hypertension and insulin resistance.

2.2. Clinical assessment

The assessment of weight and height was accomplished in underwear and barefoot. Weight was measured using an Año-Sayol scale, with a reading interval of 0–120 kg and precision 100 g, and height was measured using a Holtain wall stadiometer ranging 60–210 cm and precision 0.1 cm. Body mass index (BMI) was calculated according to the corresponding formula: weight (kg)/height^2 (m). Values of *Z* score for BMI were calculated using a nutrition application (Aplicación Nutricional) program from the Spanish Society of Pediatric Gastroenterology,

Hepatology and Nutrition (available at http://www.gastroinf.es/nutritional/). The inclusion criterion was BMI (*Z* score) values exceeding +2.0 (97th percentile) by age and sex according to the growing charts from Ferrández et al. (Centro Andrea Prader, Zaragoza 2002) [33].

Blood pressure (BP) was measured in the right arm with the patient in the supine position using Visomat comfort 20/40 (Roche Diagnostics Inc.) digital blood pressure monitor, recording the lowest of three measurements. Arterial hypertension (HTA) was considered when systolic blood pressure (SBP) and/or diastolic blood pressure (DBP) was equal to or higher than 95th percentile by age, sex and height from the American reference charts (National high blood pressure Program in Children and Adolescents) [34].

The institutionalized program for Child Care in the Community of Navarre (Comunidad Foral de Navarra, Spain) includes periodic health examinations at ages 1, 2, 3, 4, 6, 8, 10 and 12 years. The anthropometric measurements (weight and height) are recorded in the corresponding clinical history. This record has allowed the registration of the age of onset for obesity and the time of evolution at the moment of the examination.

2.3. Metabolic study

Plasma concentrations for glucose, insulin, triglycerides, total cholesterol (total-chol), high-density lipoprotein cholesterol (HDL-chol), low-density lipoprotein cholesterol (LDL-chol) and leptin were measured under basal fasting conditions using standardized methodologies.

In order to determine insulin resistance, the homeostasis model-assessment (HOMA) indexes were calculated from fasting glucose and insulin concentrations (glucose levels in mmol × insulin in μUml/L/22.5). Insulin resistance was considered when HOMA value was equal to or higher than 3.8 [35].

2.4. Statistical analysis

Results are displayed as percentages (%) and means (M) with corresponding standard deviations (SDS). Statistical analysis (descriptive statistics, Student's *T*, chi-square test and Pearson's correlation) was done using the Statistical Packages for the Social Sciences version 20.0 (Chicago, IL, USA). Statistical significance was assumed when *p* value was lower than 0.05.

Parents and/or legal guardians were informed and provided verbal consent for the participation in this study in all cases. The study was approved by the Ethics Committee for Human Investigation at our institution.

3. Results

The sample of patients consisted of 106 patients (47 males and 59 females). The prepubertal group included 56 patients (22 males and 34 females) and the pubertal group included 50 patients (25 males and 25 females).

http://dx.doi.org/10.5772/65098

Clinical data	Prepubertal group	Pubertal group	Total
Age of onset (years)			
Males	3.50 ± 1.75	5.10 ± 3.22**	4.35 ± 2.73
Females	3.38 ± 1.36	7.89 ± 3.56**	5.29 ± 3.37
Total	3.43 ± 1.51*	6.50 ± 3.65*	4.88 ± 3.12
Age at examination (years)			
Males	8.78 ± 1.01	12.42 ± 1.43**	10.72 ± 2.21
Females	8.55 ± 1.26	13.22 ± 1.15**	10.53 ± 2.62
Total	8.64 ± 1.17*	12.82 ± 1.35*	10.61 ± 2.44
Evolution (years)			
Males	5.27 ± 1.49	7.95 ± 2.86	6.64 ± 2.64
Females	5.16 ± 1.85	6.65 ± 2.71	5.71 ± 2.23
Total	5.21 ± 1.71*	7.35 ± 2.84*	6.14 ± 2.49
BMI (Z score)			
Males	4.05 ± 1.13	3.95 ± 1.31**	4.00 ± 1.21**
Females	3.55 ± 1.27	2.60 ± 0.50**	3.14 ± 1.11**
Total	3.74 ± 1.23*	3.27 ± 1.20*	3.52 ± 1.23
Systolic BP (mmHg)			
Males	113.68 ± 11.55	120.09 ± 14.74	116.88 ± 13.48
Females	108.78 ± 16.94	121.17 ± 16.10	113.96 ± 17.56
Total	110.77 ± 15.05*	120.64 ± 15.29*	115.26 ± 15.87
Diastolic BP (mmHg)			
Males	67.22 ± 11.45	70.81 ± 17.08	69.02 ± 14.49
Females	67.59 ± 11.85	70.04 ± 12.27	68.61 ± 11.98
Total	67.44 ± 11.58	70.42 ± 14.65	68.79 ± 13.08

*$p < 0.05$ among age groups.
**$p < 0.05$ between sexes.

Table 2. Average values (M ± SD) for the clinical features in both age groups according to sex.

Table 2 lists and compares the clinical features in both groups according to pubertal stage and sex. Within the pubertal group, the average values for age of onset, age at examination, years of evolution and systolic blood pressure were significantly higher ($p < 0.05$); within the prepubertal group, BMI values (Z score) at examination were higher ($p < 0.05$). There were no statistically significant differences among the average values of diastolic blood pressure between the different groups. The average BMI values (Z score) in the pubertal group were significantly higher ($p < 0.05$) in males. Finally, within the pubertal group, the average values for age of onset (obesity) were significantly lower ($p < 0.05$) in males.

Biochemical data	Prepubertal group	Pubertal group	Total
Glycemia (mg/dl)			
Males	93.40 ± 10.58	92.92 ± 10.18	93.14 ± 10.39
Females	89.12 ± 7.55	90.16 ± 8.87	89.56 ± 8.08
Total	90.83 ± 9.17	91.54 ± 9.55	91.17 ± 9.31
Insulin (uU/ml)			
Males	17.48 ± 14.58	18.22 ± 10.49	17.86 ± 12.52
Females	13.58 ± 9.70	23.82 ± 15.58	17.57 ± 13.18
Total	15.14 ± 11.93*	20.89 ± 13.31*	17.70 ± 12.82
HOMA			
Males	3.50 ± 2.09	4.01 ± 1.64**	3.77 ± 1.86**
Females	3.17 ± 1.41	3.08 ± 1.25**	3.13 ± 1.34**
Total	3.30 ± 1.70	3.54 ± 1.52	3.42 ± 1.61
Total-chol (mg/dl)			
Males	165.04 ± 27.34	152.72 ± 21.77	158.48 ± 25.05
Females	160.84 ± 27.18	157.32 ± 22.62	159.32 ± 25.17
Total	162.52 ± 27.07	155.02 ± 22.10	158.95 ± 25.00
LDL-chol (mg/dl)			
Males	93.50 ± 27.51	87.86 ± 21.26	90.62 ± 24.40
Females	94.06 ± 27.58	87.50 ± 20.64	91.25 ± 24.85
Total	93.83 ± 27.29	87.68 ± 20.72	90.97 ± 24.53
HDL-chol (mg/dl)			
Males	51.90 ± 11.31	43.30 ± 8.57**	47.51 ± 10.80
Females	49.48 ± 12.37	50.08 ± 9.93**	49.73 ± 11.32
Total	48.73 ± 9.76	47.25 ± 9.01	48.75 ± 11.09
Triglycerides (mg/dl)			
Males	115.95 ± 65.88	126.36 ± 45.59	121.48 ± 55.62
Females	98.08 ± 42.87	104.32 ± 42.08	100.72 ± 42.29
Total	105.10 ± 53.27	115.34 ± 44.83	109.93 ± 49.50
Leptin (ng/ml)			
Males	30.14 ± 11.75	34.38 ± 17.01**	32.39 ± 14.78**
Females	29.11 ± 12.43	24.66 ± 8.72**	27.22 ± 11.15**
Total	29.51 ± 12.07	29.52 ± 14.25	29.51 ± 13.08

*$p < 0.05$ among age groups.
**$p < 0.05$ between sexes.

Table 3. Average values (M ± SD) for metabolic study in both age groups according to sex.

http://dx.doi.org/10.5772/65098

Table 3 displays and compares the average values for the results of blood tests among the different groups according to pubertal stage and sex. Within the pubertal group, average values for insulin were significantly higher ($p < 0.05$) than the prepubertal group. The average values for HDL-chol within the pubertal group were significantly higher ($p < 0.05$) in females, and the average values for HOMA and leptin within the pubertal group were significantly higher ($p < 0.05$) in males.

There was a significant positive correlation ($p < 0.05$) between leptin plasma levels and BMI values ($r = 0.529$; **Figure 1**). In addition, there was a significant positive correlation between leptin plasma levels and HOMA indexes ($r = 0.562$; **Figure 2**). There was also a significant positive correlation ($p < 0.05$) between HOMA indexes and plasma concentrations of triglycerides ($r = 0.596$; **Figure 3**) as well as with age at examination ($r = 0.207$). And there was also a significant positive correlation ($p < 0.05$) between leptin plasma levels and plasma concentrations of triglycerides ($r = 0.314$; **Figure 4**).

Table 4 presents and compares the percentage values for the different clinical and metabolic parameters used as constituents of the metabolic syndrome in both groups. The percentage of patients who showed systolic blood pressure values higher than 95th percentile for the applied reference was significantly higher in the pubertal group ($p < 0.05$). There were no statistically significant differences among both groups regarding the percentage of patients who present HOMA index values higher than 3.8, plasma triglycerides higher than 110 mg/dl, HDL-chol values lower than 40 mg/dl and diastolic blood pressure values higher than 95th percentile for the applied reference charts. Within the pubertal group, the metabolic syndrome prevalence (38%) was significantly higher ($p < 0.05$) than the prepubertal group (23.2%).

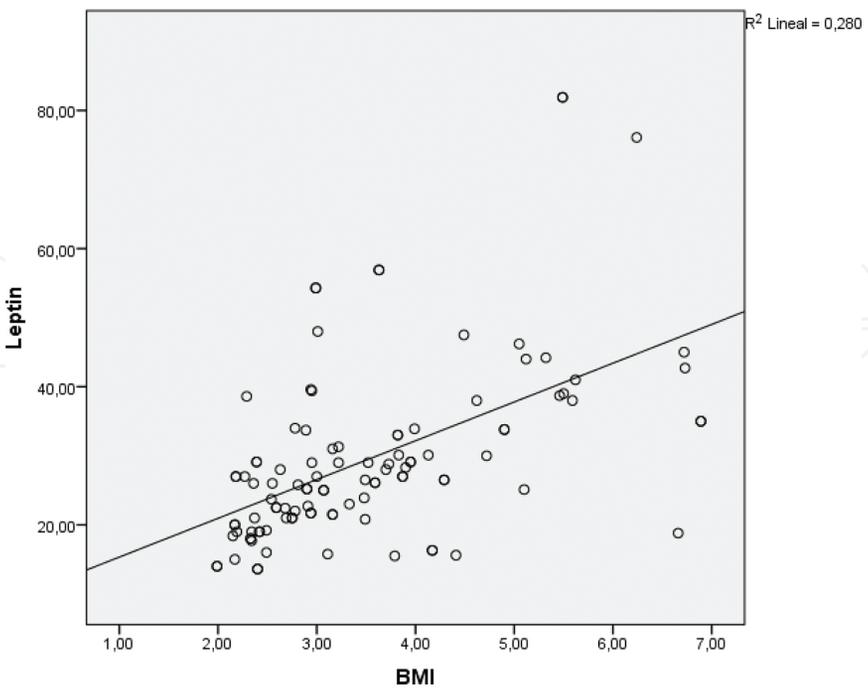

Figure 1. Leptin plasma levels (ng/ml) in relation to BMI (Z score).

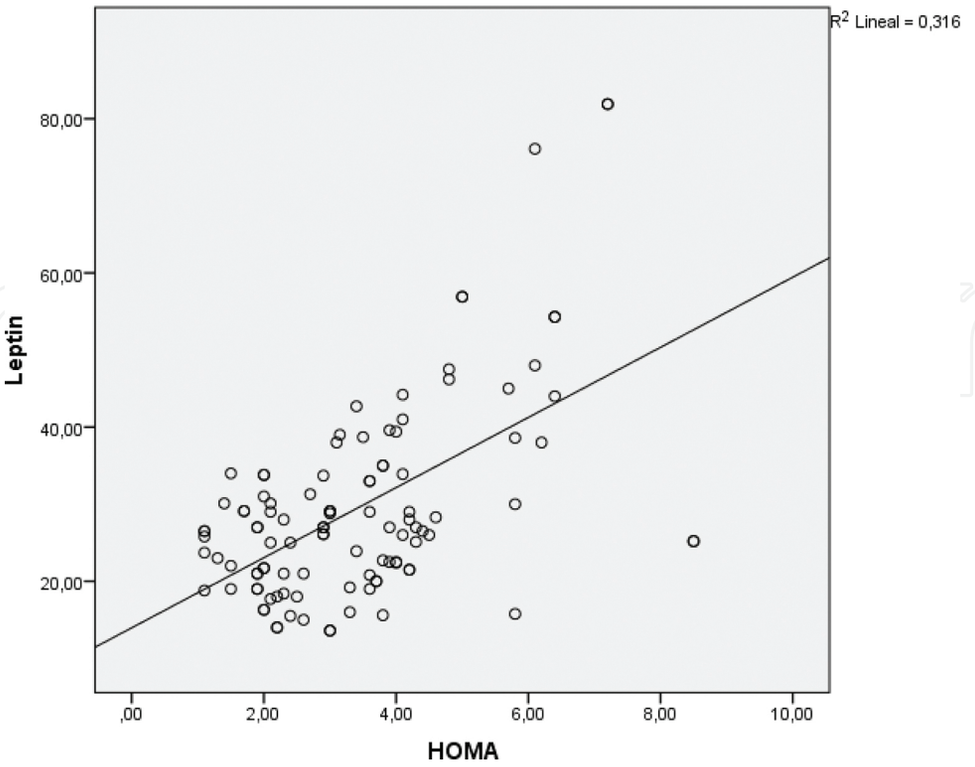

Figure 2. Leptin plasma levels (ng/ml) in relation to insulin resistance (HOMA).

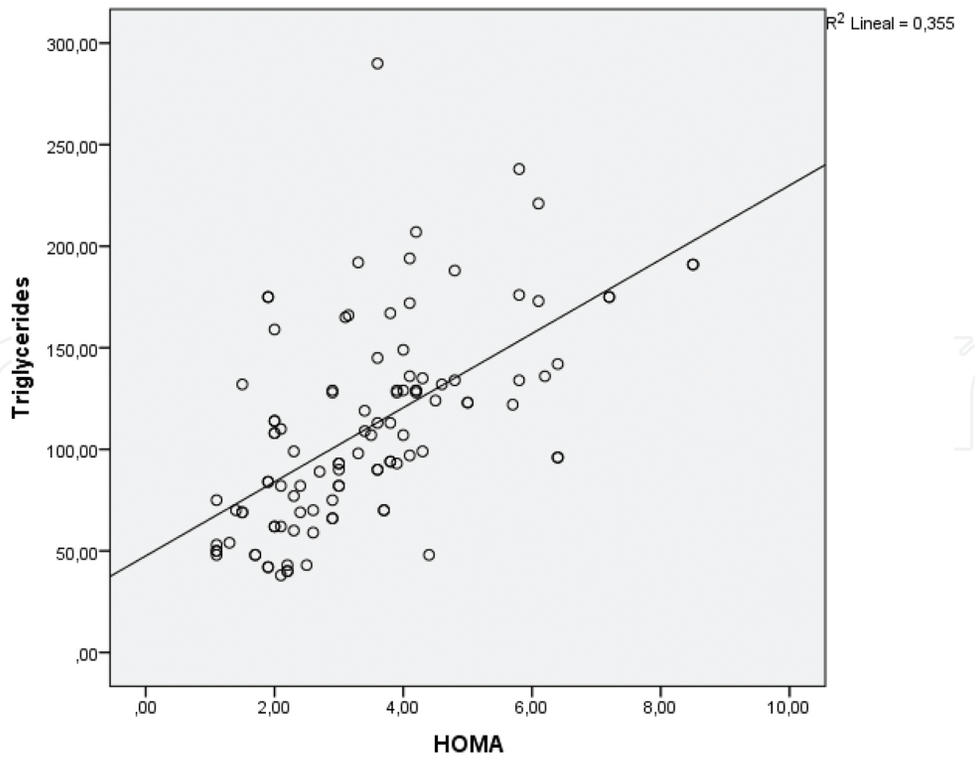

Figure 3. Triglycerides plasma concentrations (mg/dl) in relation to insulin resistance (HOMA).

http://dx.doi.org/10.5772/65098

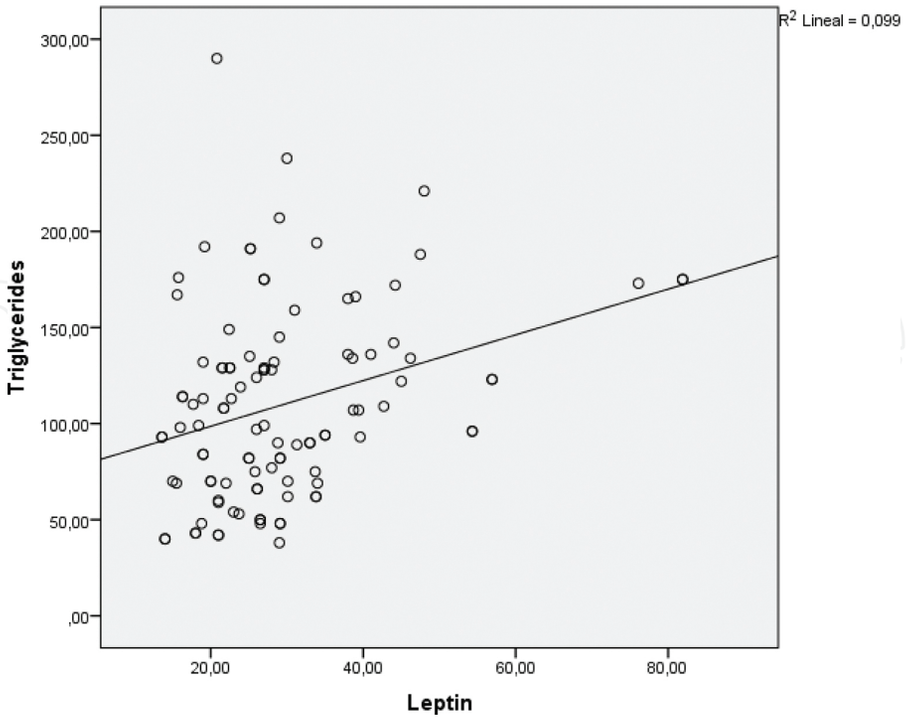

Figure 4. Triglycerides plasma concentrations (mg/dl) in relation to leptin plasma levels (ng/ml).

Diagnostic criteria	Prepubertal group *n* (%)	Pubertal group *n* (%)	Total *n* (%)
HOMA > 3.8	21 (37.5)	20 (40.0)	41 (38.7)
Triglycerides > 110 mg/dl	23 (41.1)	25 (50.0)	48 (45.3)
HDL-chol < 40 mg/dl	13 (23.6)	17 (36.2)	30 (28.3)
SBP > p95^{th*}	14 (25.9)	21 (46.7)	35 (33.8)
DBP > p95^{th*}	9 (16.7)	7 (15.6)	16 (15.1)
Metabolic syndrome*	13 (23.2)	19 (38)	32 (30.2)

$^{*}p < 0.05$ among age groups.

Table 4. Prevalence of the different diagnostic criteria of metabolic syndrome in both groups.

4. Discussion

The prevalence of metabolic syndrome within the whole sample was 30.2%, being significantly higher in the pubertal group (38%) than the prepubertal group (23.2%). The comparison of these results with those described by the other authors reveals that the prevalence found is similar to references from Cook (28.7%) [12], De Ferranti (31.2%) [13], Weiss (38.7%) [14], Cruz (30%) [15] and Viner (30%) [16] in obese United Kingdom or American children and adoles-

cents, although slightly higher than references from Lopez-Capapé (18%) [25], Tapia (18.6%) [27] and Olza (16.8%) [28] in Spanish obese pediatric population. Nevertheless, the contrast of the rate of prevalence from different studies has a relative value, since the criteria applied are different, and even different cut points for each component of metabolic syndrome are used [26, 28].

This study follows Cook modified criteria (abdominal perimeter has been replaced by BMI in the assessment of obesity, and fasting plasma glucose higher than 110 mg/dl has been replaced by HOMA index higher than 3.8). These criteria have gradually acquired clinical relevance in the assessment of metabolic syndrome in pediatric age and support from the scientific community [12, 25, 27, 28, 31]; this allows, on one side, the achievement of comparisons among the results of the different national and international studies and, on the other side, justifies its use as reference diagnosis criteria in this work.

The IDF considers fat distribution and, concretely, central or visceral obesity—which is defined by abdominal perimeter—as a "sine qua non" criterion for the diagnosis of metabolic syndrome due to its high predictive value for cardiovascular disease in adult life [36, 37]. However, there is some controversy regarding the adequacy of its use as a main and/or necessary diagnostic criterion [38]; in fact, recent studies conducted in pediatric population have used both abdominal perimeter [12, 13, 15, 39] and BMI [14, 16, 18, 25] interchangeably. In this case, the inclusion criterion was BMI value (*Z*-score) higher than +2.0 (97th percentile) by age and sex according to the growing charts from Ferrández et al. (Centro Andrea Prader, Zaragoza 2002) [33].

Insulin resistance, as several authors have highlighted [16, 25], has been a very frequently noted metabolic disorder in the population studied. It is worth indicating that, when Reaven [40] described the syndrome X, he considered insulin resistance as the determining pathophysiological factor and, in fact, the WHO included it as main and necessary criterion in order to diagnose the metabolic syndrome [20]. However, the diagnosed criteria subsequently proposed by the National Cholesterol Education Programs Adult Treatment Program III [21] and the IDF [23] opted for a "lipid centric" theory, with special focus on dyslipidemia and/or fat distribution.

Even though several criteria have been used to evaluate peripheral insulin sensitivity and/or alterations in glucose metabolism (fasting glucose, glycemia after an oral glucose tolerance test (OGTT), fasting insulin levels, etc.), the use of a mathematical model called *homeostasis model assessment* (HOMA) as a criterion for insulin resistance has been widely contrasted as an early disorder in glucose homeostasis (hyperinsulinemia with euglycemia). In this case, despite the application of a quite restrictive cut point [21], insulin resistance was already detected in 39% of the patients included in the study. In addition, the existing correlation between the HOMA indexes and the age of the patients at the moment of examination suggests that the onset of this metabolic comorbidity associated with obesity is related to hormonal changes concomitant with puberty rather than to the evolution time of obesity.

The situation of insulin resistance usually involves a disturbance in lipid profile by stimulating lipolysis and, therefore, an increase in plasma exchange of fatty acids that, at the same time,

http://dx.doi.org/10.5772/65098

stimulate the hepatic triglyceride synthesis. This explains its correlation with the HOMA index of the patients. In addition, the concentration of low-density lipoproteins is usually in normal range while the concentration of high-density lipoproteins is usually low, being this considered as a side effect of hypertriglyceridemia [41–43]. Dyslipidemia observed in patients with insulin resistance corresponded with the situation expected in this metabolic condition. Furthermore, hyperinsulinemia causes water and sodium retention and activates the sympathetic nervous system, contributing to the development of hypertension.

Leptin participates directly in the regulation of energy homeostasis through an anorectic effect and an increase in thermogenesis. Plasma concentrations reflect the reserve of organic fat and are considered as a predictive factor for insulin resistance [44]. This would explain, on one hand, the existing correlation between plasma concentrations and BMI, and, on the other hand, the correlation with the HOMA index in the patients included in this work. In addition, leptin stimulates lipolysis in adipocytes and, consequently, contributes to dyslipidemia (there was a correlation with leptin plasma levels and triglycerides); it also stimulates angiogenesis and/or endothelial dysfunction and would explain, to a great extent, the development of hypertension, which is frequently associated with obesity [30]. Instead, it has been reported that adiponectin has a paradoxical effect. Adiponectin levels correlate negatively with insulin and triglyceride concentrations and BMI and positively with insulin sensitivity and high-density lipoprotein levels. Adiponectin increases fatty acid combustion and decreases triglyceride content in the liver and skeletal muscle and thus increases insulin sensitivity [45, 46]. Recently, several authors had suggested that leptin/adiponectin ratio could be a better biomark for metabolic syndrome [47–49].

As a conclusion, we remark the finding of clinical and metabolic disorders associated with obesity and related to the so-called metabolic syndrome, which, to a great extent, are already present in pediatric population. In the same way, the positive correlation between leptin plasma levels and BMI values, HOMA indexes and plasma concentrations of triglycerides suggests that leptin could play an important role in the etiopathogenesis of metabolic syndrome and/or comorbidities that are associated with obesity.

Author details

Teodoro Durá-Travé[1,2,3*], Fidel Gallinas-Victoriano[2], Leyre Lloreda-Martín[1], Alberto Ríos-Muñoz[2], Inés Niyubahwe[2] and Ander Ernaga-Lorea[2]

*Address all correspondence to: tduratra@cfnavarra.es

1 Department of Pediatrics, School of Medicine, University of Navarra, Pamplona, Spain

2 Department of Pediatrics, Navarra Hospital Complex, Pamplona, Spain

3 Navarra Institute for Health Research (IdisNa), Pamplona, Spain

References

[1] Odgen CL, Flegal KM, Carroll MD, Johnson CL. Prevalence and trends in overweight among US children and adolescents, 1999–2000. JAMA. 2002; 88:1728–32.

[2] Tzotzas T, Krassas GE. Prevalence and trends of obesity in children and adults of South Europe. Pediatr Endocrinol Rev. 2004; 1 Suppl 3:448–454.

[3] Durá-Travé T, Hualde-Olascoaga J, Garralda-Torres I, Grupo Colaborador de Navarra. Overweight among children in Navarra (Spain) and its impact on adolescence. Med Clin (Barc). 2012; 138:52–56.

[4] Durá-Travé T, Gallinas-Victoriano F, Grupo Colaborador de Navarra. Natural evolution of excess body weight (overweight and obesity) in children. An Pediatr (Barc). 2013; 79:300–6.

[5] Srinivasan SR, Myers L, Berenson GS. Predictability of childhood adiposity and insulin for developing insulin resistance syndrome (syndrome X) in young adulthood: the Bogalusa Heart Study. Diabetes. 2002; 51:204–9.

[6] Bao W, Srinivasan SR, Wattigney WA, Berenson GS. Persistence of multiple cardiovascular risk clustering related to syndrome X from childhood to young adulthood. The Bogalusa Heart Study. Arch Intern Med. 1994; 154:1842–7.

[7] Freedman DS, Khan LK, Dietz WH, Srinivasan SR, Berenson GS. Relationship of childhood obesity to coronary heart disease risk factors in adulthood: the Bogalusa Heart Study. Pediatrics. 2001; 108:712–8.

[8] Brambilla P, Lissau I, Flodmark CE, Moreno LA, Widhalm K, Wabitsch M, et al. Metabolic risk-factor clustering estimation in children: to draw a line across pediatric metabolic syndrome. Int J Obes (Lond). 2007; 31:591–600.

[9] Yusuf S, Hawkwn S, Ounpuu S, Dans T, Avezum A, Lanas F, et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTER-HEART study): case-control study. Lancet. 2004; 364:937–52.

[10] Goodman E, Dolan LM, Morrison JA, Daniels SR: Factor analysis of clustered cardiovascular risks in adolescence: obesity is the predominant correlate of risk among youth. Circulation. 2005; 111:1970–77.

[11] Freedman DS, Dietz WH, Srinivasan SR, Berenson GS. The relation of overweight to cardiovascular risk factors among children and adolescents: the Bogalusa Heart Study. Pediatrics. 1999; 103:1175–82.

[12] Cook S, Weitzman M, Auinger P, et al. Prevalence of a metabolic syndrome phenotype in adolescents. Findings from the third National Health and Nutrition Examination Survey, 1988–1994. Arch Pediatr Adolesc Med. 2003;157:821–7.

[13] De Ferranti SD, Gauvreau K, Ludwig DS, et al. Prevalence of the metabolic syndrome in American adolescents: findings from the third National Health and Nutrition Examination Survey. Circulation. 2004;110:2494–7.

[14] Weiss R, Dziura J, Burgert TS, et al. Obesity and the metabolic syndrome in children and adolescents. N Engl J Med. 2004;350:2362–74.

[15] Cruz ML, Weigensberg MJ, Huang TT, Ball G, Shaibi GQ, Goran MI. The metabolic syndrome in overweight Hispanic youth and the role of insulin sensitivity. J Clin Endocrinol Metab. 2004; 89:108–113.

[16] Viner RM, Segal TY, Lichtarowicz-Krynska E, et al. Prevalence of the insulin resistance syndrome in obesity. Arch Dis Child. 2005; 90:10–4.

[17] Ford ES, Ajani UA, Morkdad AH. The metabolic syndrome and concentration of C-reactive protein among U.S. youth. Diabetes Care. 2005; 28: 878–81.

[18] Lambert M, Paradis G, O´Loughlin J, Delvin EE, Hanley JA, Levy E. Insulin resistance syndrome in a representative sample of children and adolescents from Quebec, Canada. Int J Obes Relat Metab Disord. 2004; 28:833–41.

[19] Zimmet P, Alberti KG, Kaufman F, Tajima N, Silink M, Arslanian S, et al. IDF Consensus Group: The metabolic syndrome in children and adolescents – an IDF consensus report. Pediatr Diabetes. 2007; 8:299–306.

[20] Alberti KG, Zimmet PZ. Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: Diagnosis and classification of diabetes mellitus. Provisional report of a WHO consultation. Diabet Med. 1998;15:539–93.

[21] Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. Executive summary of the Third Report of The National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, And High Blood Cholesterol in Adults (Adult Treatment Panel III). JAMA. 2001;285:2486–97.

[22] Balkau B, Charles MA. Comment on the provisional report from the WHO consultation. European Group for the Study of Insulin Resistance (EGIR). Diabet Med. 1999;16:442–3.

[23] Alberti KG, Zimmet P, Shaw J, IDF Epidemiology Task Force Consensus Group. The metabolic syndrome—a new worldwide definition. Lancet. 2005;366:1059–62.

[24] Goodman E, Daniels S, Morrison J, Huang B, Dolan LM. Contrasting prevalence of and demographic disparities in the World Health Organization and National Cholesterol Education Program Adult Treatment Panel III definitions of metabolic syndrome among adolescents. J Pediatr. 2004;145:445–51.

[25] López-Capapé M, Alonso M, Colino E, Mustieles C, Corbatón J, Barrio R. Frequency of the metabolic syndrome in obese Spanish pediatric Population. Eur J Endocrinol. 2006; 155:313–19.

[26] Reinehr T, de Sousa G, Toschke AM, Andler W. Comparison of metabolic syndrome prevalence using eight different definitions: a critical approach. Arch Dis Child. 2007; 92: 1067–72.

[27] Tapia-Ceballos L, López-Siguero JP, Jurado-ortiz A. Prevalence of metabolic syndrome and its componentes in obese children and adolescents. An Pediatr (Barc). 2007 ; 67 : 352–61.

[28] Olza J, Gil-Campos M, Leis R, Bueno G, Aguilera CM, Valle M, et al. Presence of the metabolic syndrome in obese children at prepubertal age. Ann Nutr Metab. 2011; 58:343–50.

[29] Martos-Moreno GA, Gil-Campos M, Bueno G, Bahillo P, Bernal S, Feliu A, et al. Obesity associated metabolic impairment is evident at early ages: Spanish collaborative study. Nutr Hosp. 2014; 30:787–93.

[30] Marti A, Martinez JA. Leptin and body weight regulation. ANALES Sis San Navarra. 1999; 22:353–63.

[31] Moreno LA, Pineda I, Rodríguez G, Fleta J, Giner A, Juste MG, et al. Leptin and metabolic syndrome in obese and non-obese children. Horm Metab Res. 2002; 34:394–9.

[32] Zuo H, Chu NF, Rimm EB, Wang D-J, Liou H-S, Shie S-M. Clustering of cardiovascular disease risk factors among obese schoolchildren: the Taipei Children Heart Study. Am J Clin Nutr. 1998; 67:1141–6

[33] Ferrández A, Baguer L, Labarta JL. Longitudinal study of normal Spanish children from birth to adulthood (anthoprometric, pubertal, radiological and intellectual data. Pediatr Endocr Rev. 2005; 2:423–559.

[34] Task Force on Blood Pressure Control in Children: Report of the Second Task Force on Blood Pressure Control in children – 1987. Pediatrics. 1987; 79: 1–25.

[35] Tresaco B, Bueno G, Pinerda I, Moreno LA, Garagorri JM, Bueno M. Homeostatic model assessment (HOMA) index cut-off values to identify the metabolic syndrome in children. J Physiol Biochem. 2005; 61:381–8.

[36] Pouliot MC, Després JP, Lemieux S, Moorjani S, Bouchard C, Tremblay A, et al. Waist circumference and abdominal sagittal diameter: best simple anthropometric indexes of abdominal visceral adipose tissue accumulation and related cardiovascular risk in men and women. Am J Cardiol. 1994; 73:460–8.

[37] Savva SC, Tornaritis M, Savva ME, Kourides Y, Panagi A, Silikiotou N, et al. Waist circumference and waist-to-height ratio are better predictors of cardiovascular disease risk factors in children than body mass index. Int J Obes. 2000; 24:1453–8.

[38] Cook S, Auinger P, Daniels S. What best predicts medical complications of obesity? BMI, waist circumference pr both. Obes Res. 2003; 11 Suppl:A27–28.

[39] Velásquez-Rodríguez CM, Velásquez-Villa M, Gómez-Ocampo L, Bermúdez-Cardona J. Abdominal obesity and low physical activity are associated with insulin resistance in overweight adolescents: a cross-sectional study. BMC Pediatr. 2014; 14:258.

[40] Reaven G. Role of insulin resistance in human disease. Diabetes. 1988; 37:1595–607.

[41] Arner P. Human fat cell lipolysis: biochemistry, regulation and clinical role. Best Pract Res Clin Endocrinol Metab. 2005; 1:471–82.

[42] Cardona F, Gónzalo-Marín M, Tinahones FJ. Association between postprandial hypertriglyceridemia and insulin resistance in patients with the metabolic syndrome. Endocrinol Nutr. 2006; 53:237–41.

[43] Klop B, Elte JW, Castro-Cabezas M. Dislipidemia in obesity: mechanisms and potential targets. Nutrients. 2013; 5:1218–40.

[44] Zuo H, Shi Z, Yuan B, Dai Y, Wu G, Hussain A. Association between serum leptin concentrations and insulin resistance: a population-based study from China. PLoS ONE. 2013; 8(1): e54615.

[45] Yadav A, Kataria MA, Saini V, Yadav A. Role of leptin and adiponectin in insulin resistance. Clin Chim Acta. 2013; 417:80–84

[46] Falahi E, Rad AHK, Sajjad Roosta S. What is the best biomarker for metabolic syndrome diagnosis?. Diab Met Syndr: Clin Res Rev (2013), http://dx.doi.org/10.1016/j.dsx.2013.06.014 (article in press)

[47] Labruna G, Pasanisi F, Nardelli C, Caso R, Vitale DF, Contaldo F, Sacchetti L. High leptin/adiponectin ratio and serum triglycerides are associated with an "at-risk" phenotype in young severely obese patients. Obesity. 2011; 19: 1492–96.

[48] Donoso MA, Muñoz-Calvo MT, Barrios V, Martínez G, Hawkins F, Argente J. Increased leptin/adiponectin ratio and free leptin index are markers of insulin resistance in obese girls during pubertal development. Horm Res Paediatr. 2013; 80:363–70

[49] Angın Y, Arslan N, Kuralay F. Leptin-to-adiponectin ratio in obese adolescents with 16 nonalcoholic fatty liver disease. Turk J Pediatr. 2014; 56: 259–66

IntechOpen

IntechOpen