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Abstracts

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birth (LB), Final Height (FH) relative to their Target Height (TH): (FH-TH), Body Mass Index (BMI), lipid profile, metabolism of carbohydrate, blood pressure (BP) and academic level valued as studies conducted.

Materials and Methods: Data are collected from a sample of 61 adults born PEG (treated and not treated with GH). Informed consent was obtained by phone, as well as clinical-analytical data that were contrasted with The Clinical History. Those who were premature, multiple parity and those with syndromes were excluded.

NOT CACHT-UP GROUP: 30 adults (15 female). Age (mean: 22.8 years); range: 18-30 y. At the birth: WB (sds); mean: -2 sds; range: -3.8- (-0.4); LB (SDS); mean: -2.37 (-0.55 to -4.44). Final height (cm) in female, mean: 153 (r: 148-163.5) and males: 167 (r: 160-170.5). The FH-TH (sds): -0.57 (r: -2.05-1). The actual BMI is 19.8 (r: 15.2-25). Only a patient has overweight. The 48.4% have University studies.

CACHT-UP GROUP: 31 adults (15 female). Age (mean: 27.5 years); range: 23-32 y. At the birth: WB (sds); mean: -3 sds; range: -4.5- (-1.6); LB (SDS); mean: -3.2 (r: -4.7 to -1.99). Final height (cm) in female, mean: 160.8 (r: 152-174) and males: 169.7 (r: 160-189.0). The FH-TH (sds): -0.31 (r: -2.7-1.54). The actual BMI is 23.6 (r: 17.2-31.1). Three adults have obesity and six have overweight. The 25% have abnormal lipid profile or abnormal BP. The 38% have University studies. Anthropometric data at birth are significant smaller in catch-up group ($p < 0.001$). The mean final height is significant higher in Catch-up group ($p < 0.001$). No difference in FH-TH. The BMI is significant higher in Catch-up group ($p < 0.001$).

Conclusions:

Anthropometry at birth is lower in those who recovered size, but the final size is larger.

- Both groups reach similar academic levels
- There are no differences between the two groups in FH-TH.
- BMI is higher in those who did catch-up and they presents more cardiovascular risk.

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Changes in objectively measured physical activity after 2-year lifestyle intervention in pediatric patients with abdominal obesity

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Introduction: it has been widely demonstrated that high physical activity (PA) levels prevent obesity development. The practice of moderate, regular PA reduces abdominal obesity, hypertension, insulin resistance, dyslipidemia and improves risk factors for obesity-related comorbidities (e.g. diabetes or cardiovascular diseases) independently of weight loss. However, in the childhood population, where sedentary behavior has increased in recent decades, few studies have assessed the effect of lifestyle interventions on

changes in PA, measured by accelerometry in pediatric patients with abdominal obesity.

Methods: one hundred and two children and adolescents with abdominal obesity (7-16 years old) were enrolled in a lifestyle intervention program for two years. Participants were encouraged to accumulate extra 200 min/week of PA. PA was assessed by accelerometry. Available data of objectively measured PA was obtained from 38 participants at the end of intervention.

Results: participants (mean age 11 years; 61% female) achieved a significant reduction in anthropometric (percentage of fat mass -3.99 ± 5.76 ; $p < 0.001$) and biochemical parameters (total cholesterol -15.12 ± 21.41 , $p < 0.001$; LDL-cholesterol -12.27 ± 16.18 , $p < 0.001$; triglycerides -26.81 ± 39.63 ; leptin -6.95 ± 14.03 , $p = 0.023$) after the 2-year lifestyle intervention. In addition, a significant decrease in light PA levels (-87 min/day, $p < 0.001$) was observed. In multiple regression analysis changes in light PA levels were positively associated with changes in leptin levels ($R^2 = 0.321$, $p = 0.012$).

Conclusion: participants significantly improved anthropometric and biochemical parameters. In addition, favorable changes in PA levels could affect leptin levels after 2-year lifestyle intervention in pediatric patients with abdominal obesity.

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Assessment of cardiometabolic risk factor clustering in obese children and adolescents with metabolic syndrome

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Background: Obesity in childhood and adolescence has reached epidemic proportions. Endothelial dysfunction, as a consequence of dyslipidemia, hypertension, insulin resistance and inflammation, imposes a substantial risk for the development of metabolic syndrome (MS) in childhood and compromises the health of the pediatric population by promoting premature development of atherosclerotic cardiovascular disease.

Aim: To investigate the cardiovascular risk in obese children and adolescents with MS compared with their obese counterparts without MS.

Patients and Methods: Eighty eight ($n = 88$) obese children and adolescents [mean age \pm SD: 13.1 ± 1.9 years; 53 males (60.2%) and 35 females (39.8%); 14 prepubertal (15.9%) and 74 pubertal (84.1%)] with MS and sixty ($n = 60$) obese children and adolescents without MS [12.2 ± 2.1 years; 37 males (61.7%) and 23 females (38.3%); 18 prepubertal (30%) and 42 pubertal (70%)] were studied prospectively for one year. All participants received personalized

advice on diet and exercise, while 26 (29.5%) participants in the MS group and 13 (21.7%) participants in the control group also received metformin. Biochemical and endocrinologic investigations, oral glucose tolerance test, echocardiography, ultrasonography of the carotid arteries and liver were performed at the beginning and at the end of study.

Results: Systolic and diastolic blood pressure was significantly higher in children with MS (127.9±13.1 and 76.7±11.3mmHg, respectively) than those without MS (116.6±10.5 and 68.2±9.2mmHg, P<0.001). At the beginning of the study (t₀), subjects with MS had significantly lower HDL [39.1±1.2 vs. 45.2±1.2 mg/dL, p<0.001], and higher triglycerides (125.1±1.6 vs. 86.5±1.5 mg/dL, p<0.001) and insulin (30.4±1.6 vs. 20.7±1.4 µIU/mL, p<0.001) concentrations compared with the control group. The carotid intima-media thickness (cIMT) at t₀ was 0.8±1.1mm in the MS group and 0.7±1.1mm in the non MS group, respectively (normal range: 0.49±0.03mm), whereas after 1 year of intervention cIMT_{t₁₂} improved significantly only in children with MS (0.5±0.2mm, p<0.05). Furthermore, patients with MS and ≥ 3 risk factors had significantly higher cIMT compared to those with 2 or less risk factors. Interventricular septal end diastole and systole were significantly higher in patients with MS (8.4±1.7 mm and 9.0±1.3 mm, respectively) than the control group (7.8±1.2 mm and 8.2±1.3 mm, p<0.05). Hepatic steatosis was identified in 69 (78%) of the participants with MS and in 34 (56.7%) obese without MS.

Conclusions: Our findings demonstrate increased cardiovascular risk in children and adolescents with MS, as well as an improvement in certain cardiovascular parameters following intervention.

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HOMA-IR in obese children with BMI ≥2.5SDS, but not <2.5SDS, differ significantly from normal weight children

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Objective: Compare HOMA-IR between obese and normal weight children.

Methods: 292 children (156 females), BMI (≥0SD), Tanner stage 1/>2: 163/129, divided in four groups (Group 1: 0SD<BMI<2SD, Group 2: 2SD≤BMI<2.25SD, Group 3: 2.25SD≤BMI<2.5SD, Group 4: BMI≥2.5SD) were analyzed retrospectively from the medical records. Age, sex, fasting blood glucose and insulin as well as the homeostasis model assessment of insulin resistance (HOMA-IR) [(fasting glucose-mmol/l×fasting insulin mIU/l)/22.5] were recorded and insulin resistance was defined as HOMA-IR≥ 3.4. One-Way and Two-Way Analysis of variance (ANOVA), p<0.05, were calculated among groups using the SPSS statistics program.

Results: Mean age was 9.5 yrs (SD, 3.07), BMI: 2.28 (SD, 0.59), HOMA-IR: 3.88 (SD, 3.6), fasting glucose: 87.3 (SD, 8.3) mg/dl, fasting insulin: 17.9 (SD, 16) mIU/l. Clinical and laboratory characteristics are summarized in table 1. HOMA-IR was significantly higher in group 4 only as opposed to group 1 (p:0.02) but not group 2 or 3. Prepubertal boys in groups 2, 3, 4 and overall had higher HOMA-IR as opposed to girls (4.1/3.5, boys/girls), but in puberty this finding was reversed (3.6/4.4 boys/girls). However, there was no statistically significant difference among groups in relation to sex or puberty.

Conclusion: Obese children with BMI ≥2.5SDS, but not BMI<2.5SDS, present significantly higher HOMA-IR as opposed to normal weight children, irrespectively of sex or Tanner stage.

Table 1. Clinical and laboratory characteristics, Mean (SD).

	Group 1 (n=55)	Group 2 (n=78)	Group 3 (n=70)	Group 4 (n=89)	p
Age (yrs)	10.9 (2.4)	9.7 (2.2)	9.5 (2.8)	7.6 (3.6)	*
Sex (boys/girls)	20/35	37/41	32/38	47/42	
Tanner stage I/II-IV	20/35	47/31	36/34	60/29	*
BMI z-score	1.7 (0.27)	2.1 (0.06)	2.4 (0.08)	2.9 (0.65)	*
HOMA IR	3.1 (1.0)	3.6 (3.0)	3.9 (2.7)	4.6 (5.1)	
< 3.4	69%	62%	52%	52%	*
≥ 3.4	31%	38%	48%	48%	
Fasting Glucose (mg/dl)	89(8.6)	87 (8.3)	87 (7.8)	86 (8.2)	*
Fasting Insulin (mU/l)	14 (8)	17 (14)	18 (11)	21 (23)	*