Results: Waist/height ratio (W/h) is greater in M general sample ($P = 0{\cdot}01$) but also in POST-F:PRE-F ($P = 0{\cdot}05$) and in PRE-M:PRE-F ($P = 0{\cdot}0001$). At large, blood glucose was higher in M, independently from puberty ($P = 0{\cdot}01$), while insulin was similar in F/M. After sub-grouping, insulin was higher in post-F/M (both $P = 0{\cdot}0001$) *v.* PRE, while glucose was higher in POST-F:POST-M ($P = 0{\cdot}01$).

Similar behaviour for insulin resistance-homeostasis model assessment (IR-HOMA): higher in POST-F/M *v.* PRE (both $P = 0{\cdot}0001$). Besides, IR-HOMA >2·5 risk is higher in POST (whole sample, F, M), but POST-M have a greater risk (OR = 2·11 POST:PRE, $P = 0{\cdot}0001$; OR = 2·45 POST-M:PRE-M, $P = 0{\cdot}02$; OR = 1·94 POST-F:PRE-F, $P = 0{\cdot}01$).

Conclusions: M attending our outpatients service seems in poorer nutritional (higher W/h) and metabolic conditions (higher pathologic IR-HOMA risk) than F, with a slight indication that abdominal fat distribution might not be the only explanation for IR appearance: other factors should be considered and studied.

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29 – Insulin resistance risk among ex-preterm overweight/obese patients

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Introduction: According to the 'thrifty phenotype' hypothesis, ex-preterm (ExP) children, if overfed in infancy, show a greater insulin resistance (IR) risk than AGA (appropriate gestational age) children. ExP also shows a larger waist circumference (W), due to greater extent of abdominal fat: this might trigger off IR.

Method: 655 valid overweight/obese patients (F 356, M 309; average age = 10·43 (SD 2·84) years) were considered: ExP 118 (F 62, M 56), AGA 547 (F 294, M 253). Anthropometric indexes studied were W and waist/height ratio (W/h). Insulin resistance-homeostasis model assessment (IR-HOMA), studied in 569 patients (ExP 102: F 54, M 48; AGA 467: F 254, M 213), led to sub-grouping them in: IR-HOMA >2·5 (ExP 53: F 26, M 27; AGA 226: F 129, M 97) and IR-HOMA <2·5 (ExP 49: F 28, M 21; AGA 241: F 125, M 116). Statistical analysis used Student's t and χ^2 tests.

Results: W was >95thC in 97·4% of ExP *v.* 91·4% of AGA; W/h was pathologic (>0·5) in 92·4% of ExP *v.* 89·0% of AGA. ExP have a higher risk of W > 95thC and W/h > 0·5 than AGA (W OR: F = 5·33, M = 2·23; W/h OR = 1·5 in both F/M, respectively). ExP also have IR-HOMA > 2·5 more frequently, with higher risk for M (OR: F = 0·9; M = 1·5).

Conclusions: In our experience, ExP of both genders show a greater extent of W > 95°C and W/h > 0·5 than AGA, but an IR risk just slightly higher (OR = 1·25). M ExP seems to be at higher risk than F: literature lacks of data about this point.

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30 – Relationship among insulin resistance, blood lipids and blood pressure in a population of paediatric overweight/obese patients

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Introduction: Within the ample debate about Metabolic Syndrome, its *primum movens* and its pathophysiology, a relationship among high insulin resistance-homeostasis model assessment (IR-HOMA), blood lipids (BL: triglycerides

TG, total cholesterol CHOL, LDL-CHOL) and blood pressure (BP) in both genders is well known. Some authors (see Sinalko 2004) even documented a constant, progressive increase of BL and BP, proportional to IR-HOMA values.

Method: 288 patients with IR-HOMA >2·5 (F 160, M 128), out of 683 overweight/obese ones, aged 9–14 years (average age = 11·09 (SD 2·62) years) were divided into seven IR-HOMA and one unit stepped groups. Every group's BL and BP were confronted with the groups around and with the opposite gender's corresponding group. Statistical analysis: Student's *t* test and parametric/non-parametric correlation tests.

Results: In both genders TG decrease ($P < 0·05$) in IR-HOMA groups 1–3, then increase ($P < 0·05$); F/M CHOL increases only in groups 6 and 7 ($P < 0·05$). Among F, LDL-CHOL ($P < 0·05$) and BP ($P < 0·01$) also increase in groups 6 and 7, while among M, LDL-CHOL is increased only in some IR-HOMA groups (1, 3, 6) and BP increases in group 5 (all $P < 0·05$). Pairing opposite gender groups, BL and BP differences are significant in single groups, not at large. Parametric/non-parametric correlations were non-significant.

Conclusions: Notwithstanding our large data allow a quite accurate comparison of metabolic parameters, a progressive, much less constant, increase of any BL or BP, proportional to IR-HOMA increase, could not be demonstrated. Our contribution outlines once more that BL, BP and IR-HOMA are undoubtedly bound, but more factors that IR alone (like genetics, overweight degree and hyperhomocysteinemia) presumably influence this link.

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31 – C-reactive protein: a marker of adiposity or cardiometabolic comorbidities of paediatric obesity?

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Background/aims: Childhood obesity is a public health problem. The association between obesity and low-grade inflammation is well established. Our aim is to evaluate the association between C-reactive protein (CRP) and cardiometabolic comorbidities in paediatric obesity.

Material and method: Obese children/adolescents with nutritional obesity followed in our outpatient clinic (*n* 354) were included. Duration of disease (years), BMI *Z*-score (Center for Disease Control), percentage of fat mass (dual energy X-ray absorptiometry) and waist circumference were evaluated. Blood pressure, lipid profile and CRP were measured and homeostasis model assessment-insulin resistance (HOMA-IR) was calculated.

Results: The mean chronological age was 10·1 years (SD 3·2; min = 1·7; max = 16·9) with no differences between gender. Same data related to descriptive analyses can be observed in Table 1. CRP was positive and significantly correlated with BMI *Z*-score ($r = 0·271$; $P < 0·001$), %fat mass ($r = 0·366$; $P < 0·001$) and waist circumference ($r = 0·198$; $P < 0·001$). A strong positive correlation was observed between CRP and fat mass, even for short duration of disease (<2 years: $r = 0·731$; $P < 0·001$). No correlations were observed between CRP and lipid profile variables (total, HDL- and LDL-cholesterol, Apo lipoproteins A1 and B and triglycerides), systolic and diastolic blood pressure and HOMA-IR, independently of duration of disease.

Conclusions: Magnitude of obesity and adiposity as also intraabdominal fat deposition are predictors of early expression of low-grade inflammation. CRP seems not to be a sensitive/early marker of cardiometabolic comorbidity of paediatric obesity.

	Total (*n* 354)		Females (*n* 182)		Males (*n* 172)		
	Mean	SD	Mean	SD	Mean	SD	*P*
BMI *Z*-score	4·1	1·7	4·0	1·7	4·2	1·8	0·465
Waist (%90th Pc)	117·7	12·4	118·2	15·9	116·4	11·4	0·076
%Fat mass – (DXA)	45·8	6·1	47·2	5·7	44·3	6·2	0·002
CRP	0·31	0·4	0·32	0·4	0·31	0·4	0·581

CRP, C-reactive protein.