Table 1 BMI and metabolic parameters in obese children with IGT ofter a 6-month diet and lifestyle changes (*n* 32)

	1st examination		2nd examination	
	Mean	SD	Mean	SD
BMI (kg/m^2)	30·4	4·9	29·0	4·4**
Plasma glucose 120′ (mmol/l)	8·6	0·7	7·0	1·2*
Plasma insulin 0′ (ml U/ml)	29·1	9·2	18·8	8·0*
Plasma insulin 120′ (ml U/ml)	168·7	92·9	117·4	85·9*
HOMA	6·7	3·7	4·9	3·3*

IGT, impaired glucose tolerance; HOMA, homeostasis model assessment. *$P<0·001$, **$P<0·05$.

IGT or T2DM in clinically healthy obese children can – at least temporarily – be managed with dietary and lifestyle interventions, resulting in the improvement of the metabolic status of these children. It is known that many of the metabolic, cardiovascular and oncologic consequences of obesity are likely influenced through insulin resistance and production of inflammatory adipokines. Although diagnostic strategies are almost clear, and the majority of the changes of hormones and adipokines measured in obese children are reversible after weight loss, however treatment remains difficult, so prevention should be started very early in life. The current knowledge of adipokines, different hormones and the production of pro-inflammatory factors involved in the pathogenesis of insulin resistance will be also discussed.

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Vitamin D deficiency is highly prevalent in obese children and adolescents and associated with decreased insulin sensitivity

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Introduction: Low 25-hydroxyvitamin D (25(OH)D) is one of the endocrine derangements in obesity. We evaluated the prevalence of vitamin D deficiency (<15 ng/ml) in obese children and adolescents and studied the relationship with BMI, ethnicity, season and insulin sensitivity.

Method: Fasting serum 25(OH)D, glucose and insulin levels and the quantitative insulin sensitivity check index (QUICKI) were determined in ninety-one subjects aged 13·2 (SD 1·9) years (sixty-eight autochtones, twenty-three allochtones; 56% female; BMI-SDS 2·7 (SD 0·5) during fall/winter (F/W; *n* 56) and spring/summer (S/S; *n* 35).

Results: Vitamin D deficiency was present in 57% of the cohort. It was more prevalent in F/W than S/S (68% *v.* 40%, $P<0·02$). Patients with vitamin D deficiency had higher fasting insulin levels (25 (SD 14) qU/ml *v.* 19 (SD 10) qU/ml; $P<0·02$) and lower QUICKI (0·308 (SD 0·026) *v.* 0·320 (SD 0·028); $P<0·05$), but comparable BMI (2·8 (SD 0·5) SDS *v.* 2·7 (SD 0·5) SDS). Serum 25(OH)D levels were inversely related to fasting insulin levels ($r=-0·29$; $P<0·01$) and positively to QUICKI ($r=+0·31$; $P<0·005$), but not to BMI-SDS ($r=-0·16$). Multiple regression analysis revealed that serum 25(OH)D levels were related to season ($T=+3·6$; $P=0·001$), ethnicity ($T=-2·9$; $P=0·004$) and QUICKI ($T=+2·3$; $P=0·022$), but not to BMI-SDS.

Conclusions: Vitamin D deficiency is highly prevalent in obese children and adolescents; vitamin D status is influenced by season and ethnicity but not by BMI. Furthermore, serum 25(OH)D levels were positively related to insulin sensitivity suggesting that obese children and adolescents with hypovitaminosis D are at increased risk of developing impaired glucose metabolism independent of BMI.

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Correlation of ghrelin and obestatin levels with tryptophan degradation in obese children

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Introduction: Ghrelin has been shown to decrease insulin levels and increase appetite, body weight gain and fat accumulation. It is decreased in obesity and associated diseases. Obestatin has been isolated as its counterpart, with the common precursor, prepropeptid. Morbid obesity has also been associated with tryptophan metabolism, as 5-hydroxytryptophan, serotonin, is involved in regulation of satiety. We aimed to assess possible correlations between tryptophan degradation and serum ghrelin/obestatin and metabolic parameters in obese children and adolescents and non-obese controls.

Method: Serum ghrelin, obestatin (RIA, Phoenix, Germany), fasting and postprandial (oral glucose tolerance test) insulin, neopterin (ELISA, Brahms Hennigsdorf), tryptophan, kynurenine (HPLC) in 356 overweight and obese (mean age 11·3 (SD 2·97) years; f = m), otherwise healthy children and thirty-two non-obese controls.

Results: No difference was found in ghrelin serum levels between obese and non-obese controls. With the exception of ghrelin all investigated parameters differ significantly as compared with the obese group. Furthermore we found a significant association of ghrelin with weight and BMI, but not with obestatin. Tryptophan degradation significantly correlated with ghrelin, but not with obestatin. In addition, tryptophan correlated significantly with parameters of the metabolic syndrome.

Conclusions: Our results suggest a strong negative correlation of ghrelin and weight/BMI in obese children and adolescents. Furthermore, we found a significant correlation between tryptophan degradation rate and ghrelin suggesting central biochemical collaterals. Thus, ghrelin may play a role in mood disturbances, which in turn ultimately lead to increased energy intake.

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Obese children with normal insulin sensitivity respond differently from those with insulin resistance to lifestyle modifications

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Background: Insulin resistance is considered the unifying mechanism for the derangements associated with metabolic syndrome and its related diseases and is a frequent feature of childhood obesity. Lifestyle intervention is recommended to treat insulin resistance. Recently it has been reported that weight loss induced by diet and exercise, paradoxically increases health risk in metabolically healthy individuals. This finding raised the doubt that uncomplicated obesity would not benefit from lifestyle-induced weight loss.

The aim of our study was to assess the effects of a 3-month lifestyle intervention on insulin sensitivity and its related variables (blood pressure, HDL-cholesterol, triglycerides, fasting and 2 h post-OGTT glucose, ALT, CRP, white blood cells) in 202 obese children (36·4 % prepubertal, BMI *Z*-score 3·2 (SD 0·7)). In particular the study analysed if a short-term lifestyle intervention may have detrimental effects on metabolic variables in young people with normal insulin sensitivity. Lifestyle intervention consisted in (i) mild caloric restriction with an appropriate protein intake according to the Italian recommendations for age, and 0·5 g/kg of ideal body weight of soluble and insoluble fibres, 25 % of fat (10 % saturated fat) and 55–65 % of carbohydrates (45–55 % of complex sugars and 10 % of simple sugars, (ii) one-hour weekly session at the gym dedicated to the education to the physical activity under the guidance of trainer + 240 min of physical activity at home during the week, (iii) family psychological counselling and psycho-educational groups. HOMA-IR was used as a surrogate marker of liver insulin resistance and the OGTT-derived Matzuda index as a marker of muscular insulin sensitivity. Metabolically healthy obese (MHO) was defined by normal values of blood pressure, lipids and glucose.

Results: In the whole cohort, the 3-month lifestyle intervention induced a significant decrease in weight, waist circumference, fat mass and ALT levels with an increase in fat-free mass. However, it was possible to distinguish two groups of subjects, one in which insulin sensitivity improved (Group 1) at the end of the intervention and one in which it worsened (Group 2 represented by 45 % of subjects). Before the intervention, Group 1 and Group 2 subjects were comparable in BMI, waist circumference, body composition, % of pre-pubertal children and males, family history of diabetes, dietary intake and heart rate and % MHO subjects. Group 1 children were more insulin resistant (insulin 16·2 (SD 7·1) mU/ml *v.* 10·4 (SD 5·3) mU/ml, $P < 0·0001$; HOMA-IR 3·4 (SD 1·6) *v.*