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4th Workshop – Insulin Resistance. Early Diagnosis? Early Treatment? – Keynote Speaker

Adipokines and insulin resistance: interaction with physical activity and fitness

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Adipose tissue-derived proteins, or 'adipokines', exert a wide range of physiological actions, connecting this tissue with the rest of organs and systems in the body, and thus linking fat storage depots with central regulation of energy balance, metabolic homeostasis, immune status, reproductive capacity, angiogenesis or inflammatory response (Trayhurn and Wood 2004). Adipose tissue dysfunction in obesity takes place with an altered production of most adipokines; this outcome is also shown in related pathological conditions such as type 2 diabetes mellitus and the metabolic syndrome.

Lifestyle habits, in particular physical activity, are modifiable risk factors for obesity and obesity-related disturbances. Both physical activity and fitness have been inversely associated with all-cause mortality and chronic diseases, including CVD, in early ages as well as in adulthood (Eisenmann 2007; Kamper *et al.* 1996). Adipose tissue-derived proteins, or adipokines, have revealed themselves as key modulators of energy homeostasis and inflammatory balance, playing a determinant role in the regulation of systemic insulin sensitivity.

The mechanisms through which physical activity and fitness influence chronic disease risk have not been entirely clarified yet, although the new inflammatory markers, amongst them adipokines, are likely candidates in mediating physical activity and fitness health benefits. Physical activity and physical fitness provide many health benefits, which appear to be not only a consequence of changes in body weight and composition, but that may also be mediated by stimulation of insulin sensitivity and amelioration of the inflammatory response, through direct and indirect regulatory actions on adipokine secretion.

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What are the roles of insulin and other hormones in the development of obesity and its complications in children?

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Introduction: The epidemic of childhood obesity is associated with the occurrence of comorbidities including type 2 diabetes mellitus (T2DM), hypertension, metabolic syndrome, non-alcoholic fatty liver disease, polycystic ovary syndrome and others. Excess body fat may be stored by increasing the size (hypertrophy) and/or the number (hyperplasia) of adipocytes. White adipose tissue has been recognized as an endocrine organ that secretes a number of hormones and cytokines (adipokines: leptin, adiponectin, resistin, visfatin), which interact with brain centres and play a role in the pathogenesis of obesity-associated diseases. Obesity is the most common cause of insulin resistance. Insulin is also an important regulator of lipolysis and lipogenesis.

Examination: Oral glucose tolerance test was performed in 289 obese (mean BMI 31·1 (SD 4·6) kg/m^2) adolescents (mean age 12·9 (SD 2·7) years), among them the impaired glucose tolerance (IGT) was found in fifty (17·3 %) and T2DM in five (1·9 %) children. After a 6 months lifestyle and dietary changes the metabolic status of obese children with IGT and T2DM could improve (Table 1).

Conclusions: Childhood obesity is frequently associated with the disturbances of carbohydrate metabolism.

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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