Editorial

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Childhood obesity at the crossroads

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Introduction and scope of the problem

Overweight and obesity is frequently seen in children and adolescents across most developed, and developing, countries with a 20–35% prevalence. Although the prevalence of obesity at a young age seems to stabilise or even slightly decline in some countries, the number of adolescents with obesity is still increasing (1). This is a major concern, considering the well-described association of overweight and obesity with long-term health problems, such as cardiovascular disease, type 2 diabetes and cancer. In the majority of individuals, weight gain is the result of exposure to an ‘obesogenic’ environment, superimposed on a background of genetic susceptibility brought about through evolutionary adaptation (2).

Approximately 40–70% of inter-individual differences in body weight and fat content is thought to be due to genetic variation (3). A large number of genes have been identified by genome wide association studies (GWAS) and candidate gene approaches that are associated with the regulation of body weight. According to the thrifty gene hypothesis, evolutionary selection pressure has selected genes which allow individuals to survive periods of food deprivation. Within our modern obesogenic environment, however, these same genetic susceptibility traits now appear to be detrimental by promoting obesity and its associated metabolic diseases (2).

In this issue of our journal, three review papers, one on behavioural aspects of obesity (4), one on hypothalamic forms of obesity (5) and lastly and importantly one on the role of obesigenic environments address the important public health issue of childhood obesity. These reviews are followed by a series of papers that have been submitted recently to the journal and which cover a wide range of childhood obesity related topics mainly dealing with cardiovascular and metabolic sequelae of obesity.

Scientific dilemma

Before addressing obesity research and the material discussed in this issue of the Journal of Paediatric Endocrinology and Metabolism the scientific and clinical dilemmas related to obesity have to be mentioned: Translating science into clinical practice is not easy. Research in biology, biomedicine, psychology, and the social sciences and humanities do follow their own rules: career plans of scientists, their sometimes narcistic nature and personal fights between them often impair the advancement of knowledge. The requirement to apply for financial funds and resources both within an institution and from outside greatly and substantially influences the choice of topics, area of interest and even the questions that are being asked and the answers that one will arrive at. Once financial funds have been secured researchers very often focus on easy to reach goals and follow mainstream research: working in an area where everyone else works, researching topics with tools that everyone else is using will enable scientists too (6).

Hence, the questions that are asked are not necessarily the questions that need to be answered in order to improve quality of life of patients, reduce suffering from a disease, or increase the survival of patients let alone increase our chances to successfully prevent disease. Failure to concentrate on the real causes of the obesity epidemic worldwide has actually led to an inability to treat or prevent the disease. Importantly, if one asks questions without asking why one should answer the question in the long run, will not benefit the patient (6).

Clinical dilemma

Any meta-analysis on the efficacy of treatment of obesity and even more so on the prevention of overweight and obesity at a young age has failed to show large effects of treatment and/or prevention strategies. Barriers to participate and obstacles to access treatment and prevention programs are but one reason for the failure of therapeutic and
preventive strategies in the long term (7). Very often families with an obese child will have low educational and socioeconomic resources and even will sometimes completely fail to understand the importance and or relevance of treating childhood obesity. In addition, very often the affected child will have obese family members whose obesigenic environment and lifestyle will ultimately influence the child’s environment and lifestyle. Since in many cases the comorbidities of obesity are not felt at a young age, there is little individual and personal incentive to change habits and or unhealthy lifestyles. Lastly, if one is to live with obese peers, the motivation to lose weight or change one’s unhealthy behaviour will be very limited indeed (1, 6).

**Behaviour, hypothalamus and obesigenic environment**

In our review on environmental approaches to obesity prevention, we stress the point that ultimately only multidimensional interventions will be effective and no single behavioural or environmental intervention can possibly be effective on its own. Access to playgrounds, restrictions on food marketing for children, prizing of healthy food stuffs and changes in food supplies in cafeterias, walkability of living quarters and the planning of cities all have to be considered if one is to seriously prevent childhood obesity on a large scale (2). Haliloglu and Bereket outline, in their outstanding review article, why hypothalamic obesity has to be seen as a special and unique entity: it is a complex neuroendocrine disorder indeed usually caused by direct damage to the hypothalamus. Hypothalamic obesity is frequently a feature of serious diseases and disorders and in itself is very difficult to treat (5). Behavioural diversity of children includes a wider variety of responses to the obesogenic environment. In fact, eating behaviour and in particular, responses to satiety, energy compensation, eating rate and meal frequency, responsiveness to food and food advertisement, food reward and importantly taste and dietary preferences vary greatly among individuals. Hence, addressing behavioural aspects of childhood obesity should be considered an area of research and a target for intervention strategies (4).

**Obesity indices**

Body mass index has been used as a marker of obesity and is the most frequently used tool to diagnose overweight and obesity also in childhood. However, BMI has surprisingly low sensitivity and specificity when compared to other body mass indices such as skinfold measurements, waist circumference and such. In the study by Marković-Jovanović et al. the unreliability of BMI for diagnosing childhood obesity is revisited (8). The clinical importance of determining abdominal obesity is highlighted in the paper by Ozturk et al. It is visceral and organ adipose tissue rather than subcutaneous fat that is thought to contribute most to the development of co-morbidities in obese subjects. In their study 5358 elementary and secondary school students from Turkey were recruited and the mid-upper arm circumference was found to be a strong predictor of abdominal obesity (9).

**Obesity and co-morbidities**

Insulin resistance that eventually may lead to type 2 diabetes and therefore represents one of the most clinically relevant co-morbidities of obesity has been in the focus of obesity research for many years. In this issue of JPEM, Maggio et al. report on the relation between cardiorespiratory fitness and markers of insulin resistance. It is interesting to note that maintenance and/or improvement of cardiorespiratory fitness prevents the development of insulin resistance. Hence, the importance of physical activity on metabolic health is again documented (10). Insulin resistance and cardiometabolic risk are also studied in the work by Sakou et al. (11). A putative link between thyroid function, obesity and insulin resistance is the topic of the study by Santos et al. It looks as if children with higher TSH levels had higher degrees of insulin resistance as, for example, expressed by HOMA-IR (12). A common feature of all three of these papers is that they report on rather small numbers of subjects and follow-up if required is very limited. These limitations are typical of many studies in the field of childhood obesity research and indeed prevent generalisable conclusions to some extent.

**Obesity and endocrine surrogate markers**

In order to better understand the pathogenesis of co-morbidities and also in order to find out what surrogate markers could be useful to differentiate between metabolically and cardiovascularly healthy from unhealthy...
obesity many a surrogate marker be it adipokytines, lipoproteins or lipids or growth factors have been studied. In this issue of our journal Gultom et al. investigate the putative influence of apolipoprotein E polymorphisms on obesity morbidity and try to develop a personalised approach to treat dyslipidemia in obese adolescents (13). A study from Estonia reports that osteocalcin serum levels are inversely related to markers of body adiposity and leptin levels. A relation between obesity and bone health has been suggested in the past and this paper adds to our understanding of putative pathophysiological relations (14). Bone health and obesity is also being discussed in the following paper: in particular, putative associations between insulin-like growth factor-I and IGF-binding proteins, bone turnover and obesity have been studied by Gajewska et al. (15). New markers of obesity, namely endogenous antioxidant levels, have been studied by Cayr et al. Since oxidative stress is linked to morbidity the measurement of markers of oxydative stress might be of clinical relevance (16).

Cardiovascular risk

It comes to no surprise that obesity in youth is linked to higher blood pressure, and in fact ethnicity, gender and markers of adrenal function have been studied before. In this issue of JPEM a relationship between dehydroepiandrosterone is proven (17), the variability of blood pressure and in fact ethnicity, gender and leptin levels. A relation between obesity and bone health has been suggested in the past and this paper adds to our understanding of putative pathophysiological relations (14). Bone health and obesity is also being discussed in the following paper: in particular, putative associations between insulin-like growth factor-I and IGF-binding proteins, bone turnover and obesity have been studied by Gajewska et al. (15). New markers of obesity, namely endogenous antioxidant levels, have been studied by Cayr et al. Since oxidative stress is linked to morbidity the measurement of markers of oxydative stress might be of clinical relevance (16).

References


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