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Origins and early prevention of obesity – news?!

Friday, 09 June 2017
12:00 to 01:00 pm
The Palace of Parliament
International Conference Centre
Bucharest

Chair:
Prof. Dr. Doina Anca Plesca
Director of Pediatrics Department
University of Medicine and Pharmacy „Carol Davila“,
Bucharest, Romania
Head of Pediatric Clinic, Children‘s Hospital „Victor Gomoiu“

Prof. Dr. Berthold Koletzko
Metabolic and Nutritional Medicine
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Early prevention of obesity: the role of nutrition in the first year of life

Convincing evidence has accumulated to show that diet and lifestyle at the very beginning of life strongly impact on later health and child and adult obesity risk by modifying the slope of the lifelong risk trajectory. The World Health Organisation's 2016 report on "Ending Childhood Obesity" emphasized that particular opportunities for effective prevention of obesity exist during pregnancy, before pregnancy (including adolescence) and in infancy and early childhood. Rapid weight gain in infancy and in the second year of life has been consistently shown to markedly increase the odds of later obesity in childhood, adolescence and adulthood[1,2]. Breastfeeding is associated with less rapid weight gain in infancy than conventional bottle feeding[3]. Several meta-analyses of observational studies found breastfeeding associated with ≈10-25 % less obesity risk later[4,5]. We followed the hypothesis that the greater weight gain in formula-fed infants, relative to breastfed infants, is at least partly caused by differences in protein intakes (the "Early Protein Hypothesis")[6,7], inducing increased concentrations of insulinogenic amino acids and of insulin and IGF-1. We tested the "Early Protein Hypothesis" in a randomized double-blind clinical trial that enrolled 1678 infants born at term, the European Childhood Obesity Project Trial[8]. A non-randomized reference group was fully breastfed for at least 3 months. Formula-fed infants were randomized at an average age of 2 weeks to conventional infant formulae (followed by follow-on formulae) with high protein contents (HP), or isoenergetic intervention formulae with lowered protein (LP) contents more similar to breast milk protein levels, provided from a mean age of 2 weeks to 1 year of age. Length growth was not different between feeding groups, but weight, weight-for-length and BMI were significantly higher in HP than LP from six months of age onwards[8]. The differences in growth trajectories appear to be mediated by markedly increased plasma concentrations of indispensable amino acids, insulin and IGF-1[9,10]. Dietary effects on the IGF-1 axis outweighed by far the effects of genetics and gender[11]. There was also a significant effect on kidney growth that appears to be mediated by IGF-1[12]. Follow-up at early school age (6 yrs) showed marked persistent effects of infant feeding on median and particularly upper BMI centiles, and on obesity prevalence[13]. LP markedly reduced obesity prevalence compared to HP (adj. RR 0.35, 95%CI: 0.15, 0.82; P=0.016) (13). Feeding LP in infancy also significantly reduced body fat accumulation at 2 years and at 6 years of age, compared to HP, and led to a body composition similar to that in previously breastfed infants. Results of metabolomic and amino acid analyses suggest that not only the quantity of protein supply, but also the quality of protein provided in infancy is important for modulating infant growth[10]. In recent more recent studies we evaluated the impact of improved protein quality in infant formula and found that this significantly enhances the energetic efficacy of infant formula, reducing the gap to human milk[14,15]. With a path model analysis we determined that different amino acids have a very variable impact on the key growth factors insulin and IGF-1 in infants[16], indicating that improvements of formula quality have the potential to markedly modify growth trajectories and hence long-term health. Conclusions: Infant feeding has very powerful long-term programming effects, with very large effect sizes on obesity and adiposity at early school age. Breastfeeding appears to causally protect against later obesity, mediated at least in part by human milk protein supply. This finding further strengthens the conclusion that breastfeeding should be proactively promoted, protected and supported. Infants not fully breastfed should get infant formulae with reduced protein contents but high protein quality. The feeding of unmodified cows' and other animal milks as a drink should be avoided in the first year of life whenever feasible and affordable, because cows' milk contains three times as much protein as human milk and may induce excessive weight gain, body fat deposition and increased later obesity. Future research should lead to further optimization of protein supply in early life.

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Current insights in the genetic and endocrine origins of obesity

The rise in childhood obesity prevalence poses an enormous challenge for our health care systems because a great proportion of overweight children tend to become obese adults. The obesity associated comorbidities such as hypertension, dyslipidemia, and disturbed glucose intolerance appear already quite frequently in childhood. Both, obesity as such and also the comorbidities seem to be associated with increased morbidity and premature death. Therefore, research on the childhood obesity pandemic is of crucial importance for affected individuals, their families and also the society.

Only a very small portion of obese patients have an underlying monogenetic or endocrine disorder. However, knowledge about these disorders is important since it helps to identify affected individuals and also increases our knowledge about weight regulation which in return helps to understand and counsel patients with polygenetic obesity. This is especially true since new genes causing a monogenetic obesity keep being discovered. In addition, some of the genetic obesity diseases are treatable with recently developed drugs.

The first aim of this presentation is to review recent advances in the genetics of obesity.

One practical dilemma we face when dealing with childhood obesity is that on the one hand it is a fairly common problem and on the other hand, monogenetic forms of obesity are exceedingly rare. The second aim of this presentation is therefore to show with the help of case presentations when molecular diagnostic procedures are indicated.