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Obesity in childhood and adolescence: epidemiology, management and mechanisms

AJ Drake
Lecturer in Child Life and Health, University of Edinburgh, Edinburgh, Scotland

ABSTRACT There is increasing public awareness of the rising incidence of obesity in childhood and adolescence, and its consequences for long-term health – type 2 diabetes, cardiovascular disease and cancer. This year's symposium meeting focused on epidemiology, management and mechanisms of obesity. The trends in obesity in children and adolescence, at both a global and national level were reviewed. The UK House of Commons Select Committee's report on obesity suggests that obesity 'will soon surpass smoking as the greatest cause of premature loss of life'. If we can understand the cause we can potentially make an impact on future healthcare. The review covered primary prevention, early intervention and management of the significantly obese. People want to understand why they are overweight and what impact obesity will have on their health. Rare monogenic causes of severe obesity give insight into central mechanisms underlying weight control. Hormones are intrinsically linked with obesity, but do not provide the easy solution hoped for by many. A discussion around three case studies concluded the symposium.

KEYWORDS Childhood obesity, epidemiology, management

LIST OF ABBREVIATIONS 11β-hydroxysteroid dehydrogenase type 1 (11β-HSD1), α- and β-melanocyte stimulating hormone (α- and β-MSH), adrenocorticotrophic hormone (ACTH), body mass index (BMI), Child and Adolescent Mental Health Services (CAMHS), free fatty acids (FFA), Genetics Of Obesity Study (GOOS), impaired glucose tolerance (IGT), International Obesity Task Force (IOTF), melanocortin 4 receptor (MC4R), pro-opiomelanocortin (POMC), randomised controlled trial (RCT), standard deviation score (SDS), World Health Organization (WHO).

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Correspondence to **Dr A Drake, Dept. of child life and health, University of Edinburgh, 20 Sylvan Place, Edinburgh, EH9 1UW**

tel. **+44 (0)131 536 0825**

fax. **+44 (0)131 536 0821**

e-mail **mandy.drake@ed.ac.uk**

CME

UPDATE ON CURRENT EPIDEMIOLOGY OF OBESITY IN CHILDREN

Dr R Viner (London) discussed global trends. Obesity is common in richer 'developed' nations, particularly the USA. However, in some developing countries, the number of overweight now exceeds the number who are underweight, particularly in urban areas, creating a 'dual burden' of overweight and underweight.[1] The International Obesity Task Force estimates that the world prevalence of childhood obesity is around 2%, with up to 10% being overweight. The most common cause of childhood obesity is 'primary obesity' influenced by environmental factors, but with some contribution from, and probably interaction with, genetic factors. Parental obesity is the strongest predictor of childhood and later obesity. Childhood obesity is associated with insulin resistance, type 2 diabetes, polycystic ovarian syndrome, obstructive sleep apnoea, fatty liver disease and psychosocial disorders. He discussed problems with the use of BMI to monitor weight change in obese individuals and populations (for example, exercise may increase BMI despite a reduction in fat mass). Additionally, there are various definitions of obesity in childhood.

Dr J Reilly (Glasgow) gave the national perspective for the UK. The prevalence of childhood obesity in the UK is increasing;[2] in Scotland, 10% of 4–5 year olds and 20% of 11–12 year olds are obese. He argued that there is good evidence for the use of BMI to identify the fattest children and those at high risk of co-morbidities. Using current BMI reference ranges (e.g. IOTF), the scale of the obesity problem has been underestimated and there is a trend to increased central fatness which may be missed using BMI.[3] The International Obesity Task Force BMI reference ranges have high specificity, but low sensitivity in contrast to National (UK) definitions. Correlates with paediatric obesity in the UK include age and lower socioeconomic status and there is emerging evidence for an increased prevalence in ethnic minorities. He discussed obesity prevention studies, including the SPARKLE study of Glasgow pre-school children, showing as little as 3–5% of their time awake is spent in moderate to vigorous intensity activity. Most RCTs are

methodologically flawed and it is unclear how applicable the results are to other populations. In terms of routine management, an Edinburgh audit of attendance at a dietetic clinic showed that more than 50% of obese children did not attend for review. There are few ongoing intervention studies, mostly US-based and in high-risk groups. He briefly discussed two Scottish studies, the MAGIC study (in pre-school children) and the Scottish Childhood Overweight Treatment Trial, a dietetic programme, results from which are awaited.

CLINICAL MANAGEMENT

In session two Dr M Rudolf (Leeds) began by discussing what we can learn from studies so far. A multifaceted approach is needed, parents must be involved, communication is important and frequent appointments are needed over a long period of time. She discussed who should be delivering care and the competencies needed. There are many initiatives and opportunities in existence in the community, but fewer services available for younger children and there is little ongoing evaluation. Offering help within schools may increase stigmatisation. In Leeds, the WATCH IT project, for 8–16 year olds, provides individual and group sessions, activity sessions and parenting sessions within Sports Centres, run by 'trainers', non-health professionals recruited for their personal skills and supervised by health professionals. The group had excellent attendance, most individuals showed decreased BMI at six months and reported improved nutrition, decreased self-harm and misery and increased self-confidence. She discussed her concerns that we should focus on obesity in young children. She highlighted the need to focus on those families requesting help, services should be community-based and funding is required for multidisciplinary tertiary services for those with morbid obesity and special needs. There is a high level of misery associated with obesity; therefore CAMHS must be involved.

Dr J Shield (Bristol) highlighted the appearance of type 2 diabetes in childhood. Studies in UK children report that 10–11% of obese children have IGT and 30% have features of the metabolic syndrome,[4] there is also evidence for endothelial dysfunction. Weight loss can improve a number of the metabolic sequelae of obesity. He then discussed the multidisciplinary obesity clinic in Bristol involving an exercise specialist, dietician, psychologist and medical staff. Of 140 significantly obese children, 83% showed reduced BMI SDS after one year. 'Achievers' (losing >0·5 BMI SDS) tended to be younger, male and had less parental obesity but there was no association with socioeconomic status or the presence of metabolic abnormalities at baseline. However there is a 26% drop-out rate, free group exercise sessions were poorly attended and funding is difficult to obtain. He discussed potential medical strategies including Orlistat, particularly as an adjunct in individuals with learning difficulties, Sibutramine, Remonibant and bariatric surgery. The 'Mandometer' trial aimed to retrain children to reduce their speed of eating. Pilot data suggests the technique can be effective and a RCT is in progress.

Dr L Edmunds from Bristol outlined a number of psychological issues. Children measure 'self-worth' by assessing their own 'competencies' against a number of domains, attaching importance to things they excel at and discounting things they are not good at (for overweight children this is typically physical activity). We are judged by physical appearance, which cannot be discounted; children are conscious of this from a young age. Being overweight is less important for children if they have friends. Bullying can begin at a mild degree of overweight, becoming worse as children get older and more overweight. She discussed some of her interviews with parents. Many overweight children lacked confidence; some were withdrawing from school and other social contact. Overweight children may be more immature than their chronological age, but look older, causing problems due to unrealistic expectations of their behaviour. Parents want to be listened to, to be taken seriously, help with portion sizes, motivational and problem solving tools. Helpful approaches may include parenting classes, counselling, cognitive behavioural therapy, support from other agencies (CAMHS, charities, social services). There is little evidence that healthy eating and exercise strategies are associated with the development of eating disorders.

A short discussion followed. Paediatricians appear hesitant to use medication in obese children. The panel agreed that these therapies are likely to become increasingly used in paediatric practice, in addition to surgery, although lifestyle modification must remain the first line of treatment. Initial weight loss following bariatric surgery may be regained later and surgery may not be a long-term solution. The panel agreed that there should be public health strategies to manage the problem of obesity, although they disagreed somewhat on what such strategies might achieve. They also agreed that an increase in psychology input would be useful.

MECHANISMS IN OBESITY

Dr S Farooqi (Cambridge) suggested that some people are genetically predisposed to gain weight in the current environment. In the GOOS cohort of 1,700 severely obese children, genes associated with altered appetite were studied. She described children with leptin deficiency resulting in severe obesity in whom leptin treatment has lead to fat loss, reduced hyperphagia, spontaneous puberty onset and reduced infections. Obese individuals have been identified with an abnormality in the leptin receptor in the absence of markedly increased circulating leptin levels. She then discussed the leptin–melanocortin pathway. Leptin

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activates POMC which is cleaved to ACTH in the pituitary and α- and β-MSH in the hypothalamus (which then act at MC4R to control food intake). Individuals homozygous for mutations in POMC have red hair, pale skin, adrenal insufficiency and obesity. Melanocortin 4 receptor mutations are the most common genetic form of obesity (5–6% of patients in the GOOS cohort), are dominantly inherited and may be amenable to treatment in the near future. Children with these mutations have increased fat and lean body mass and are tall, possibly secondary to marked hyperinsulinaemia. She showed new data on neurotrophins (molecules involved in neurone growth and connections); a mutation in the neurotrophin TrkB receptor has been identified in one patient with obesity, impaired memory and hyperactivity.

Professor B Walker (Edinburgh) discussed mechanisms linking obesity with its adverse metabolic consequences. Central (visceral) obesity is particularly associated with increased cardiovascular risk and insulin resistance. Adipose tissue has a role in storage of FFA as triglyceride. A finite adipose storage capacity results in extra-adipose fat storage, e.g. in liver. In addition, excess FFA can induce insulin resistance. The use of stable isotope tracers has shown increased FFA flux particularly from visceral adipose in obesity,[5] although the importance of this is unclear. Studies in mice suggest that adipokines released from adipose tissue influence insulin sensitivity in peripheral tissues, e.g. liver and muscle. He then discussed the potential role of steroids. Individuals with Cushing's syndrome have elevated circulating cortisol levels causing obesity and features of the metabolic syndrome. However, idiopathic obesity is not associated with increased circulating cortisol levels. 11β-HSD1 converts inactive cortisone into active cortisol. Mice overexpressing 11β-HSD1 specifically in fat, are obese and show features of the metabolic syndrome;[6] conversely, loss of 11β–HSD1 in fat is protective.[7] Obese adults have increased 11β-HSD1 activity in subcutaneous fat. Drugs inhibiting 11β-HSD1, lower blood glucose in mice and prevent obesity. New tracer studies can be used to quantify cortisol regeneration. Future studies envisage the development of agents to specifically inhibit this enzyme in fat.

INTERACTIVE CASE DISCUSSIONS

In session four, three case studies from Edinburgh were put to the expert panel of Dr S Greene (Dundee), Dr P Padfield (Edinburgh) and Ms L Stewart (Edinburgh). Dr M Strachan presented an 18-year-old man with severe obesity, type 2 diabetes, hypertension and hyperlipidaemia. Dr Louise Bath presented an 11-year-old girl with insulin resistance and IGT who was commenced on metformin and showed weight loss, normalisation of glucose tolerance and improved insulin resistance. Dr C Oxley presented a girl with diabetes who is heterozygous for a mutation in the PPARγ gene.

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