



Conference on ‘Childhood nutrition and obesity: current status and future challenges’ Symposium 4: Strategies for reducing childhood obesity

Management of obesity in children differs from that of adults

Hilary Hoey

Department of Paediatrics, Trinity College Dublin, Ireland

Obesity in childhood is a very common disorder with an increasing prevalence. It is one of the most serious public health challenges. The objectives of the present paper are to increase the awareness of the problem of obesity in childhood, its serious complications and the need for prevention. Overweight and obese children are likely to remain obese into adulthood and more likely to develop serious complications including health problems such as diabetes and CVD, as well as psychological and social challenges. Overweight and obesity are largely preventable. In adults it is difficult to reduce excessive weight gain once it has become established, thus children should be considered the priority population for intervention strategies and prevention. Nutrition, exercise, weight gain in infancy, genetic and environmental factors, all contribute to the aetiology. Prevention and treatment of obesity in childhood requires education and empowerment of families relating to diet and exercise, along with the regulation and control of food marketing and clear nutritional labelling. The eating and physical activity behaviour of a child is strongly influenced by environmental and social factors. Therefore treatment will have only limited success in an environment where adequate physical activity is inhibited and the consumption of high-energy food is stimulated. Government investment in a health promotion programme addressing the issue of obesity in the population as a whole, with particular emphasis on the prevention and management of obesity in childhood is vital. The family doctor and multidisciplinary team play an important role. Regular visits to the family doctor, including growth assessment, will help motivate the family to restrict energy intake and to increase exercise. Therefore the prevention of childhood obesity needs high priority.

Children: Obesity: Diagnosis: Prevention: Management

Obesity is the most common chronic disorder in childhood and it is one of the most serious public health challenges. It is associated with major morbidity and mortality, including psychosocial problems, cardiovascular risk and the metabolic syndrome^(1–5). The prevalence of obesity and overweight has increased at an alarming rate in children and adults throughout the world and has been termed by the WHO as a ‘global epidemic’⁽⁶⁾. Over one quarter of children in the USA are overweight or obese⁽⁷⁾. The prevalence of obesity in Ireland has been rising and in 2009 the Growing up in Ireland Study showed that in Ireland one in four 3-year olds and 9-year olds are overweight or obese⁽⁸⁾. In Ireland, as in most other countries, while there has been a secular trend associated with a small increase in height during

the past century there has been a very substantial disproportionate increase in weight when compared with height; and in Ireland the major proportion of increment in weight has occurred during the past two decades^(9,10).

A principal predictor of adult obesity is childhood obesity⁽¹¹⁾. In addition, adults who are obese in childhood have a worse prognosis than adults who become obese later on, including health problems such as diabetes and CVD, as well as psychological and social challenges^(2,12,13).

One-third of obese pre-school-aged children become obese adults, as do 50% of obese school-aged children and 80% of obese adolescents⁽¹⁴⁾. It has been demonstrated in the Avon Longitudinal Study of Parents and Children that rapid weight gain between birth and



9 months is associated with obesity at 10 years and this study also showed that 75% of children who are obese at 7 years remain obese at 11^(15,16). Low birth weight associated with rapid weight gain in infancy is also associated with premature adrenarche, polycystic ovarian syndrome and the metabolic syndrome in later life⁽¹⁷⁾. The recent Growing up in Ireland study showed that low birth weight and rapid weight gain in infancy was associated with obesity at 3 years. In addition, lower socioeconomic status, lower breast-feeding rates and the early introduction of solids were associated with obesity at 3 years⁽¹⁸⁾.

Overweight and obesity, as well as their related diseases, are largely preventable. As overweight and obese children are likely to become obese adults with more rapid and serious complications than adults who become obese in later life, prevention and early intervention is essential. In addition, in adults it is difficult to reduce excessive weight gain once it has become established⁽²⁾.

Prevention and early detection of obesity in childhood are extremely important and this requires growth assessment and monitoring⁽¹⁹⁾. The diagnosis of overweight and obesity in childhood is more complex than in adults. Weight in childhood may fluctuate and will vary with age, gender, timing of puberty, body build and wellbeing. Accurate measurements and appropriate growth charts for the community are essential, together with clinical assessment and interpretation⁽²⁰⁾. Growth charts provide an important educational and empowerment tool for children and their family. Height and weight should be plotted on appropriate growth charts and weight should be interpreted in a clinical context including body build and wellbeing. With the recent rapid increase in weight gain in childhood growth charts should not incorporate this undesirable effect as such growth references will lead to an underestimation of overweight and obesity and an overestimation of under nutrition^(9,20). A weight two centiles (s.d.) or more above the height centile line suggests the need for careful clinical assessment for obesity⁽²¹⁾. BMI charts can be used to define overweight and obesity. BMI is determined by dividing weight (kg) by height (m²). It differs with age, gender and pubertal status; and interpretation must be made in relation to these, as well as body build and clinical wellbeing. However, BMI has its limitations as it does not allow for percentage body weight composed of lean muscle or a relationship with body build including head size and bone volume⁽²²⁾. The International Obesity Task Force in adults defines over weight as BMI >25 and obesity if BMI >30; however, in children BMI values vary with age and children have considerably lower values than adults. Thus a centile chart should be used and a BMI above the 91st centile for age indicates overweight and above the 98th centile obesity on the UK BMI charts⁽²³⁾, while on the US Centers for Disease Control and Prevention charts 85th to 95th centile are classified as 'at risk for overweight', >95th 'overweight' and exceeding the 99th is referred to as extreme paediatric obesity⁽²⁴⁾. BMI provides a common definition which allows data from different studies to be compared. Supplementary and more precise measurements of body fat can be determined by

assessing skinfold thickness, waist circumference, hydrodensitometry, bioelectric impedance assay MRI, DEXA and CT scans.

Causes of obesity in childhood

Obesity is due to energy intake being greater than energy expenditure and this is related to food intake, exercise, weight gain in infancy and childhood, metabolic, hormonal, genetic and environmental factors^(25–27). The pathogenesis is complex and is not completely clear⁽²⁸⁾. There is a central neurological aspect in the hypothalamus involving the feeding and the satiety centres which involve neurotransmitters, emotional and genetic aspects. Rarely a hypothalamic disorder such as tumour may cause hyperphagia or anorexia. At the fat cell there are various metabolic factors which influence obesity. The precise metabolic mechanism by which some people store more fat in cells or burn up more energies than others is not clear. The BMR may, in fact, be greater in obese people which is related to their greater size⁽²⁹⁾. However, there are complex factors at cell level which facilitate a greater uptake of fat into the cells or slow the breakdown of fat, including enzymes, hormonal and genetic aspects.

Clinical assessment of the overweight and obese child

Children with obesity may be divided into two groups: the very common primary obesity previously called simple exogenous which accounts for 95–99%; and the rare secondary organic causes which account for 1–5% (see Table 1).

Organic causes of obesity

The less common organic causes may be divided into three groups: endocrine, hypothalamic and genetic syndromes. All of these are generally associated with short stature, poor height velocity and abnormal clinical features.

The endocrine disorders associated with obesity are: hypothyroidism; growth hormone deficiency where the child is short with a greater weight than height centile and Cushing's syndrome where the obesity is mainly truncal, and in addition the child may have a plethoric face, hypertension and striae. The endocrine disorders are characterised by the combination of short stature, poor height velocity and obesity. The rare hypothalamic causes, such as a brain tumour, have clinical features such as recent weight gain, short stature, or may have neurological features such as raised intracranial pressure. There are also rare syndromes that cause obesity and these are generally associated with short stature, and also intellectual impairment. In the Prader–Willi syndrome, the children are hypotonic at birth and almost always require tube feeding in the newborn period; they have short stature; normal birth weight, but they develop

Table 1 Childhood obesity causes and major clinical features

	Major clinical features
Primary (simple/exogenous) 95–99 %	
Environmental factors	Tall stature
Food intake	Generalised obesity
Exercise	Clinical examination normal
Emotional wellbeing	Normal intellectual development
Genetic predisposition	
Organic causes 1–5 %	
Endocrine disorders	Short stature, poor height velocity
Hypothyroidism	
Growth hormone deficiency	
Cushing's syndrome	
Genetic syndromes	Short stature
Prader Willi	Developmental delay and intellectual impairment
Laurence Moon-Biedl	Abnormal clinical features
Albrights hereditary osteodystrophy	Abnormal clinical features
Single gene mutations very rare melancortin-4 receptor, leptin deficiency	Early onset obesity, positive family history
Central nervous system	Recent onset of obesity
Hypothalamic tumours/lesions	Neurological symptoms or signs

obesity in the second year secondary to hyperphagia, which if untreated, becomes progressive; they also have intellectual impairment and usually hypogonadism. Diagnosis can now be made by abnormality on chromosome 15; early dietary intervention and growth hormone treatment have greatly improved the outcome. It is usually a sporadic condition and, thus, there is no family history. In the Laurence Moon-Biedl syndrome the children are again short, obese and intellectually impaired; they may also have polydactyly, retinitis pigmentosa and hypogonadism. This is an autosomal recessive condition and, in view of the associated intellectual impairment, early diagnosis and genetic counselling is essential. Albright's hereditary osteodystrophy is associated with short stature, intellectual impairment and hypocalcaemia secondary to unresponsiveness to parathyroid hormone.

Primary or simple exogenous obesity

The most common type of obesity in childhood by far is primary or simple exogenous, which is responsible for 95–99 % of children who are obese; these children have an energy intake which exceeds their body energy expenditure and requirements. The child with this type of obesity is generally tall with a height above the 50th centile and, apart from obesity, is clinically well. They tend to have an early puberty, so that their final adult height is in fact, generally no greater than would be expected from their family height. There are many factors involved in the cause of childhood obesity including genetic, environmental, food intake, exercise and emotional wellbeing.

Genetic

Much research is being conducted in relation to genetic aspects of obesity and it is likely that excessive weight gain arises from the interactions among environmental

factors, genetic predisposition and the individual behaviour⁽³⁰⁾. Parental obesity greatly increases the risk and severity of obesity in children^(31,32). Studies have shown that if both parents are obese, 75 % of the children are obese; and if one parent is obese, 50 % of the children are obese⁽³³⁾. A genetic factor was shown in the national study, on height and weight in Sweden in which 101 pairs of twins were studied relating to whether either one or both were obese⁽³⁴⁾. In the identical twins, both twins were obese in the majority of cases whereas in non-identical twins, both twins were obese only in a minority of cases. Single gene disorders such as a melancortin-4 receptor mutation or deficient leptin production, leading to insatiable appetite and intractable obesity from early infancy do exist but are very rare. More recent genome-wide association studies have identified some genetic loci linked to childhood obesity and longitudinal analyses have revealed genetic loci linking pubertal height growth, pubertal timing and childhood obesity⁽³⁵⁾.

It is difficult however to disentangle genetic and environmental factors with common attitudes within the home towards food, eating and exercise and it seems unlikely that there has been sufficient recent change in the gene pool to account for the prevalence of obesity throughout the world today.

Environment and family lifestyle

Environmental factors play an important part as it has been shown that unrelated people living in the same household resemble each other. Garn, in Michigan, studied 429 pairs of parents who had adopted children and also had biological children of their own. He found that there was a similar degree of obesity in both the biological and the adopted children⁽³⁶⁾. In 1976, Garn in another study of 3000 pairs of spouses showed that the spouses tend to resemble each other: if a thin man marries a fat wife, there is a greater chance that he will also become fat; if a thin woman marries a fat man,

there is a greater chance she will become fat; and, conversely if a thin man marries a thin wife, he will become even thinner⁽³⁷⁾. Environmental factors, therefore, regarding the thinking of food, eating and exercise are very important in the development of obesity.

Food intake

An accurate assessment of food intake is notoriously difficult to obtain as people differ greatly in their interpretation of how much they eat. The family doctor, who knows the family and is visiting the home, is more likely to get a true picture of the dietary pattern than the hospital doctor. Obesity is more common in low socio-economic groups and in large families. This is thought to be due to a higher carbohydrate diet. As obesity is due to excessive energy intake over requirements, food consumption is the cornerstone of the cause and management of this condition.

Exercise

Exercise increases the energy output and is, therefore, very important. Low levels of physical activity in modern life were demonstrated by Bradfield when he showed that children in the USA on average spend less than 1 h every day in either moderate or strenuous exercise; and 9 h of the day are spent sitting while being transported to school; sitting in school; or sitting at home watching television⁽³⁸⁾. In the UK, of children aged 2–15 years 20% of boys and 60% of girls do not attain the recommended 60 min moderate to vigorous exercise per day⁽²⁵⁾. The Growing Up in Ireland National Longitudinal Study has shown that only 25% of 9-year-olds said that they had engaged in at least 60 min physical activity for each of the last 7 d. In addition 66% typically spent between 1 and 3 h watching television on an average day and a further 9% spent between 3 and 5 h each day⁽⁸⁾. Exercise is an important mechanism for burning energy and should be encouraged while screen and sedentary time should be limited.

Emotional stress

Emotional problems may cause obesity; overeating may be a means of dealing with frustration, anxiety or depression by a child or a family. This may be associated with a negative circle where emotional disturbance causes obesity which leads to even more emotional disturbance. Obesity is more common in only children and in children of single parents; this may be due to subconscious compensation by giving excessive food and love.

Complications of childhood obesity

Obesity causes severe complications in both children and adults. It is associated with increased insulin and insulin-like growth factor 1 levels, inflammatory markers, polycystic ovarian syndrome in females and the metabolic syndrome^(28,39). In addition, social prejudice against fat children is very common; other children show a definite

prejudice against them, ranking them less favourably than children with other physical handicaps⁽⁴⁰⁾. They are teased at school, they do badly at sport and are embarrassed changing with others, and often generally underachieve academically and socially. They rarely admit that their weight causes them any problems. There is also discrimination later against fat people. Canning showed that there were fewer obese people in prestigious colleges in Boston; and more obese people in less prestigious ones. She studied the applicants to the colleges and found that they were similar in terms of weight, ability and social class. She concluded that there is an unconscious discrimination against the obese child by schools writing references, or by college interviewers, or by both. She also suggested that obesity in the lower classes may be perpetuated by preventing them from obtaining education, and hence the occupation to maintain social class⁽⁴¹⁾.

It has been clearly shown that not only do obese children become obese adults but also that adults who have been obese as children have significantly greater cardiovascular risk than those not obese in childhood and in addition children who successfully lose weight have a cardiovascular risk similar to non-obese adults. Prevention and management of obesity in children is thus extremely important⁽²⁾. The US Multicentre SEARCH Study showed that there was a tenfold increase in the prevalence of type 2 diabetes in young people in the USA between 1990 and 2000 and 90% of those who developed type 2 diabetes were overweight or obese⁽⁴²⁾. The complications of type 2 diabetes in children and adolescents progress faster than in adults including hypertension, dyslipidaemia, CVD, renal disease and psychosocial problems^(13,43).

Hypertension is a very serious complication in children. Rames, in Iowa, showed that 43% of adolescents with hypertension were obese. This may be associated with a high morbidity or with sudden death⁽⁴⁴⁾. A recent Irish study showed that approximately one-third of children referred to a children's hospital with obesity had features of the metabolic syndrome such as hypertension, hyperlipidaemia and hyperinsulinism⁽⁴⁵⁾. The 'Pickwickian syndrome' is a serious complication in children with obesity; this is a combination of severe obesity associated with somnolence, sleep apnoea and hypoventilation. The hypoventilation results from increased oxygen requirements due to their large size, upper airways obstruction due to fatty tissue in the upper airways, and inefficient respiratory movement and collapsed areas of the lungs. There is also a central decrease in the ventilator drive; this progresses to pulmonary hypertension, cardiac failure and possibly sudden death. Orthopaedic side-effects may occur with slipped femoral epiphyses, knock knees and flat feet.

Prevention and treatment of obesity in children

Obese children have a high risk of becoming obese adults with all the associated complications which include CVD, hypertension, gallstones, arthritis, skeletal



deformities, hernia and cancer. The complications of obesity are, therefore, very serious and thus the prevention and treatment of obesity is very important. This is a global problem and involves governments, health professionals, communities, parents and families^(46,47). Education, empowerment and positive support of families relating to diet and exercise are fundamental and education should be provided in the antenatal period⁽⁴⁸⁾. Regulation and control on food marketing and clear nutritional labelling are also extremely important^(49,50).

Family diet and lifestyle is fundamental and the whole family must be involved in order to establish healthy eating at home and parents must take responsibility for supervising food intake in a positive manner^(51,52). In many cases, there is conflict at home between parents concerning the child's weight and diet and often there is ineffectual control within the family.

Lack of parent awareness of overweight and obesity in their children has shown, even when their children are obese; overweight and obese parents express less concern regarding their overweight and obese children than parents with a normal BMI⁽⁵³⁾. Health professionals may also appear unconcerned about childhood obesity⁽⁵⁴⁾.

Interviews by health professionals must involve both parents and where appropriate the extended family and child minder. Breast feeding should be encouraged. The diet should be healthy, palatable, high in fibre and protein, low in refined carbohydrate, fat and salt, relatively inexpensive and eaten by the whole family. Whole fruit and vegetable intake should be eaten on a daily basis. Drinking of water should be encouraged and easy access to water should be available in schools. Sweets, crisps and soft drinks should be avoided. In the obese child, energy intake should be reduced by food restrictions, and exercise must be encouraged. The energy intake for a school child should be appropriate and should be less if the child is obese⁽⁵⁵⁾. This should result in some weight loss and no weight gain, while height gain continues. The presence of ketones in the urine will indicate whether the child is adhering to a diet. The dietary restrictions should not be severe especially during puberty as this may diminish the adolescent growth spurt and reduce the final adult height.

Exercise

The whole family should be encouraged to undertake regular exercise with at least 1 h relatively vigorous exercise per day. Sedentary behaviour should be limited with no more than 1 h screen time per day. The placement of televisions in children's rooms/bedrooms should be discouraged. Physical activity should be provided as a vital part of the school curriculum with a wide range of enjoyable options. Access to exercise is very important and government investment is essential in road safety schemes to promote walking and cycling and free or low-cost sports facilities such as swimming pools, football pitches and skating rinks.

Motivation

Family education and support relating to healthy lifestyle, including diet and exercise by all members of the family is fundamental in order to motivate the child to lose weight. Measures to increase self-esteem and social skills are likely to enhance children's enthusiasm for activities other than television and computer games. Government and community support is very important, including education, healthy diet and exercise promotion by schools and health professionals. School environment is very important and influences nutrition knowledge and eating patterns and physical activity behaviour as well as sedentary behaviour. School-based programmes for weight control can be effective⁽⁵⁶⁾. Vending machines and tuck shops with high-energy fast foods should not be available and lunch time should be on the school premises. Medication and bariatric surgery have little to no role to play in children. A fortnightly visit to the General Practitioner, where the child is weighed, including a 15 min counselling session with the whole family has been shown to be successful⁽⁵⁷⁾.

Conclusions

Obesity is a common disorder in childhood with very serious complications and is generally a preventable condition. It occurs when energy intake exceeds body requirements. Diet, exercise, genetic and environmental and socioeconomic factors contribute to its aetiology. Primary or simple exogenous obesity is by far the most common type; these children are tall, usually with a height above the 50th centile. They are intellectually and clinically normal and generally do not require investigations. Only 1–5% of children with obesity have an organic cause; they usually have short stature with a height below the 50th centile, poor height velocity and may have intellectual or clinical abnormalities. These children require investigation. Health care costs of patients with a BMI >35 are approximately 44% more than those of non-obese⁽¹⁵⁾ and at present in Ireland approximately 10% of the healthcare budget is spent on treating obesity related conditions, and this figure is likely to increase. The prevention and treatment of obesity requires education and empowerment of families relating to diet and exercise, along with regulation and control on food marketing and clear nutritional labelling. Health education should commence in the antenatal period and continue while the children are growing up. Eating and physical activity behaviour of an individual child is strongly influenced by environmental and social factors. Therefore, treatment will have only limited success in an environment where adequate physical activity is inhibited and the consumption of high-energy food is stimulated. Government investment with a health promotion programme addressing the issue of obesity in the population as a whole with particular emphasis on the prevention and management of obesity in childhood is vital. The family doctor and multi-disciplinary team play a very important role. It has

been shown that regular visits to the family doctor including growth assessment will help motivate the family to restrict energy intake and to increase exercise. Prevention of childhood obesity needs high priority. There is a need for ongoing research relating to health promotion, intervention strategies and the management of the obese child.

Acknowledgements

I am grateful to Professor Edna Roche and the staff in the Paediatric Endocrine Clinic in the National Children's Hospital, Tallaght for their help and to the children and their families attending the clinic.

Financial support

None.

Conflicts of interest

None.

Authorship

The author was solely responsible for all aspects of preparation of this paper.

References

- Pratt CA, Stevens J & Daniels S (2008) Childhood obesity prevention and treatment recommendations for future research. *Am J Prev Med* **35**, 249–252.
- Juonala M, Magnussen CG, Berenson GS *et al.* (2011) Childhood adiposity, adult adiposity, and cardiovascular risk factors. *N Engl J Med* **365**, 1876–1885.
- Daniels SR, Arnett DK, Eckel RH *et al.* (2005) Overweight in children and adolescents: pathophysiology, consequences, prevention, and treatment. *Circulation* **111**, 1999–2012.
- Roche E (2003) Childhood obesity. *Irish Med J* **96**, 100–102.
- Berenson GS, Srinivasan SR, Bao W *et al.* (1998) Association between multiple cardiovascular risk factors and atherosclerosis in children and young adults. The Bogalusa heart study. *New Engl J Med* **338**, 1650–1656.
- World Health Organisation. Obesity: preventing and managing the global epidemic – report of a WHO Consultation on Obesity, 3–5 June 1997, Geneva: WHO/NUT/NCD/98.1.
- Dehghan M, Akhtar-Danesh N & Merchant AT (2005) Childhood obesity, prevalence and prevention. *Nutr J* **4**, 24.
- Growing up in Ireland. National Longitudinal Study. <http://www.growingup.ie>
- Perry IJ, Whelton H, Harrington J *et al.* (2009) The heights and weights of Irish children from the post-war era to the Celtic Tiger. *J Epidemiol Commun Health* **63**, 262–264.
- Hoey H, Tanner JM & Cox L (1987) Clinical growth standards for Irish children. *Acta Paediatr Scand Suppl* **338**, 1–31.
- Sinha A & Kling S (2009) A review of adolescent obesity: prevalence, etiology, and treatment. *Obes Surg* **19**, 113–120.
- Pietrobelli A, Malavolti M, Battistini NC *et al.* (2008) Metabolic syndrome: a child is not a small adult. *Int J Pediatr Obes* **8**, 3 Suppl. 1, 67–71.
- Dart AB, Martens PJ, Rigatto C *et al.* (2014) Earlier onset of complications in youth with type 2 diabetes. *Diab Care* **37**, 436–443.
- Diamond FB Jr (1998) Newer aspects of the pathophysiology, evaluation, and management of obesity in childhood. *Curr Opin Pediatr* **10**, 422–442.
- Ong KK, Emmett P, Northstone K *et al.* (2009) Infancy weight gain predicts childhood body fat and age at menarche in girls. *J Clin Endocrinol Metab* **94**, 1527–1532.
- Wright CM, Emmett PM, Ness AR *et al.* (2010) Tracking of obesity and body fatness through mid-childhood. *Arch Dis Child* **95**, 612–617.
- Idkowiak J, Lavery GC, Vivek D *et al.* (2011) Premature adrenarche: novel lessons from early onset androgen excess. *Eur J Endocrinol* **165**, 189–207.
- Madden D (2013) The relationship between low birth weight and socioeconomic status in Ireland. *J Biosoc Sci* **30**, 1–18.
- Hoey H, Roche EF & Meehan J (2005) Auxology and growth assessment. *Irish Med J* **98**, 187–188.
- Hoey H (2009) The use of growth references for school-aged children and adolescents. *Int J Pediatr Obes Suppl.* **1**, 23.
- Hulse JA & Shilg S (1996) Relation between height and weight centiles may be more useful. *BMJ* **312**, 122.
- Speiser PW, Rudolf MC, Anhalt H *et al.* (2003) Consensus statement. Childhood obesity. *J Clin Endocrinol Metab* **90**, 1871–1887.
- Cole TJ, Bellizzi MC, Flegal KM *et al.* (2000) Establishing a standard definition for child overweight and obesity worldwide: international survey. *BMJ* **6**, 1240–1243.
- Dietz WH & Robinson TN (2005) Overweight children and adolescents. *N Engl J Med* **352**, 2100–2009.
- Poskitt E. (2008) *Management of Childhood Obesity*. pp. 132–152. Cambridge: Cambridge University Press.
- Dina C, Meyre D, Gallina S *et al.* (2007) Variation in FTO contributes to childhood obesity and severe adult obesity. *Nat Genet* **39**, 724–726.
- Farooqi IS (2005) Genetic and hereditary aspects of childhood obesity. *Best Pract Res Clin Endocrinol Metab* **19**, 359–374.
- Kiess W, Blüher S, Kapellen T *et al.* (2006) Physiology of obesity in childhood and adolescence. *Curr Paediatr* **16**, 123–131.
- James WPT, Davies HL, Balies S *et al.* (1978) Elevated metabolic rates in obesity. *Lancet* **1**, 1122.
- Svensson V, Jacobsson JA, Fredriksson R *et al.* (2011) Associations between severity of obesity in childhood and adolescence, obesity onset and parental BMI: a longitudinal cohort study. *Int J Obes* **35**, 46–52.
- Keane E, Layte R, Harrington J *et al.* (2012) Measured parental weight status and familial socio-economic status correlates with childhood overweight and obesity at age 9. *PLoS ONE* **7**, e43503.
- Campión J, Milagro FI & Martínez J (2009) Individuality and epigenetics in obesity etiology and pathophysiology. *Obes Rev* **10**, 383–392.
- Gurney R (1936) Hereditary factor in obesity. *Arch Int Med* **261**, 557.



34. Bojeson M (1976) The aetiology of obesity in children: a study of 101 twin pairs. *Acta Paediatr Scand* **65**, 279–287.
35. Cousminer DL, Berry DJ, Timpson NJ *et al.* (2013) Genome-wide association and longitudinal analyses reveal genetic loci linking pubertal height growth, pubertal timing and childhood adiposity. *Hum Mol Genet* **22**, 2735–2747.
36. Garn SM, Bailey SM & Higgins ITT (1976) Fatness: similarities in adopted pairs. *Am J Clin Nutr* **29**, 1067–1068.
37. Garn SM, Clark DC (1976) Trends in fatness and the origins of obesity. *Paediatrics* **57**, 443–456.
38. Bradfield RB, Paulos J & Grossman L (1971) Energy expenditure and heart rate of obese high school girls. *AMJ Clin Nutr* **24**, 1482–1488.
39. Vilmann LS, Thisted E, Baker JL *et al.* (2012) Development of obesity and polycystic ovary syndrome in adolescents. *Horm Res Paediatr* **78**, 269–278.
40. Staffieri JR (1967) A study of social stereotype of body image in children. *J Personal Soc Psychol* **7**, 101–104.
41. Canning H & Mayer J (1966) Obesity – its possible effect on college acceptance. *N Engl J Med* **275**, 1172–1174.
42. Liu LL, Lawrence JM, Davis C *et al.* (2009) Prevalence of overweight and obesity in youth with diabetes in USA: the SEARCH for diabetes in youth study. *Pediatr Diab* **11**, 4–11.
43. Kiess W, Böttner A, Blüher S *et al.* (2004) Type 2 diabetes mellitus in children and adolescents – the beginning of a renal catastrophe? *Nephrol Dial Transplant* **19**, 2693–2696.
44. Rames LK, Clarke WR, Connor WE *et al.* (1978) Normal blood pressures and the evaluation of sustained blood pressure elevation in childhood: the Muscatine study. *Paediatrics* **61**, 245–251.
45. Hussey J, Gormley J, Bell C *et al.* (2006) Exercise tolerance and physical activity Levels in children referred to a weight reduction clinic. *Irish Med J* **99**, 49–47.
46. American Academy of Pediatrics (2003) Policy statement. Prevention of pediatric overweight and obesity. *Pediatrics* **112**, 424–430.
47. National Task Force on Obesity (2005) Obesity – the policy challenges. Department of Health, Ireland. Available at www.dohc.ie.
48. Oude Luttikhuis H, Baur L, Jansen H *et al.* (2009) Interventions for treating obesity in children. *Cochrane Database Syst REV.* (1) CD001872.
49. McMaster C (2006) *National guidelines for prevention and management of childhood overweight and obesity: evidence review and recommendations for good practice*. Sligo, Ireland: Health Service Executive of Ireland.
50. O'Malley G, Hussey J, Hannon-Fletcher M, *et al.* (2009) Moving towards health. *Int J Pediatr Obes Suppl.* 1, 4.
51. Epstein LH, Raja S, Daniel TO *et al.* (2012) The built environment moderates effects of family-based childhood obesity treatment over 2 years. *Ann Behav Med* **44**, 248–258.
52. Xanthopoulos MS, Moore RH, Wadden TA *et al.* (2013) The association between weight loss in caregivers and adolescents in a treatment trial of adolescents with obesity. *J Pediatr Psychol* **38**, 766–774.
53. Jeffery AN, Voss LD, Metcalf BS *et al.* (2005) Parents' awareness of overweight in themselves and their children: cross sectional study within a cohort (EarlyBird 21). *BMJ* **330**, 23–24.
54. White A, O'Brien B, Houlihan T *et al.* (2012) Childhood obesity: parents fail to recognise, general practitioners fail to act. *Irish Med J* **105**, 10–13.
55. Butte N (2008) Energy requirements of infants, children and adolescents. In *Pediatric Nutrition in Practice*, pp. 31–36 [Koletzko Beditor]. Basel: Karger.
56. Sharma M (2006) School based interventions for childhood and adolescent obesity. *Obes Rev* **7**, 261–269.
57. Truswell AS (1985) Obesity: diagnosis and risks. *BMJ* **291**, 655–657.