

Childhood obesity

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Obesity is the most frequent nutritional disturbance of childhood in the developed countries and can persist into adult life. This paper considers the definition, prevalence and natural history of the condition, and the effects of overnutrition on linear growth, pubertal development and the cellularity of the adipose organ.

Definition

Strictly speaking, obesity should be defined in terms of an increase in the adipose tissue mass, or of an increase in total body fat. Because, however, the techniques available for estimation of total body fat, either directly (hydrostatic weighing, absorption of gases by fat) or indirectly by determination of lean body mass (measurement of total body water or potassium) are not easily applicable to children, for technical or ethical reasons, and are certainly not useful in day-to-day clinical practice, obesity is usually defined in terms of percentage overweight. This type of definition, though practically useful, requires height and weight standards for both sexes which are up to date and preferably derived from a population of similar ethnic group. In deriving formulas to calculate the degree of overweight from such standards it must also be remembered that the standards themselves will (at least in this country) reflect the tendency of the population to be overweight. This problem applies particularly during puberty; thus, using the standards of Tanner, Whitehouse & Takaishi (1966) for a 14-year-old girl whose height of 1.72 m is on the 97th centile, the corresponding weight on the 97th centile is 72 kg. If this was the actual weight of the girl, simple inspection would identify her as being fat. However, the use of centile charts taking into account weight, height, age and sex probably still provides one of the best methods of defining 'overweight' (Newens & Goldstein, 1972), and it is generally accepted that any child whose body-weight exceeds the 'standard' by 20% is obese, provided he is not oedematous (Wolff, 1965). Inspection, however, will show that some children whose weight is only 10% above the standard are also fat.

Measurement of skinfold thickness using a suitable calliper provides a useful additional method of assessing fatness and should be used more widely in clinical practice. Tanner & Whitehouse (1962) have designed standard centile charts for subcutaneous fat-fold thickness over the triceps and subscapular regions. Durnin & Rahaman (1967) have correlated measurements made at four standard sites with body density in individuals over the age of 12 years and Brook (1971) has shown that there is a good correlation between these skinfold measurements and total

body water in younger children. Thus skinfold determinations can also be used to give an indirect estimate of total body fat.

Prevalence

To some extent the paucity of information on the prevalence of obesity is the result of problems of definition, and these are even more difficult during the 1st year of life because of technical problems in making accurate length measurements and difficulties in picking up skinfolds from underlying tissues. As a result, the 'normal' values for this age group are at present inadequate.

Despite these difficulties, however, estimates of the prevalence of obesity during the 1st year have recently been made. Hutchinson-Smith (1970) found that 35% of 200 babies exceeded the weight expected for length by more than 20%, and Shukla, Forsyth, Anderson & Marwah (1972) found that 16.7% of 300 babies weighed more than 20% above the expected value and 27.7% weighed between 10 and 20% more than the expected value.

The prevalence of obesity during the school years has been estimated at 2-3% (Howard, Dub & McMahon, 1971); this certainly increases during puberty, especially in girls, when it may become as high as 10-15% (Canning & Mayer, 1966).

Natural history

Recently more studies have been made of the problem of obesity in infancy, and one reason for this greater emphasis is based on the studies of Asher (1966) and of Eid (1970), who have shown that excessive weight gain during the first 6 months of life is associated with a greater incidence of obesity during the later years of childhood. The prevention of obesity in infancy through such measures as encouraging breast-feeding and discouraging the use of cereals before the age of 3-4 months may, in the long term, prove more effective in controlling childhood obesity than measures adopted at a later age.

Obesity in later childhood also tends to persist, and prospective studies (Haase & Hosenfeld, 1956; Lloyd, Wolff & Whelen, 1961) show that 80% of obese children may remain obese in young adult life.

The importance of childhood obesity as a precursor of adult obesity is increasingly recognized and Mullins (1958) has suggested that in approximately one-third of adults the onset of the obesity dates back to their childhood. How far this tendency for the condition to persist is the result of early feeding experience or whether intrauterine nutrition and genetic factors are also important is unknown. Intrauterine undernutrition is now known to have permanent effects on the subsequent growth of the human foetus but we are still ignorant of the effects of overnutrition at this stage of development. Studies of twins by Newman, Freeman & Holzinger (1937) and Bakwin (1973) suggest that genetic factors are of some importance and show the effect of genetic influence on the control of body-weight.

Effects on growth and pubertal development

In general terms the effect of overnutrition is the stimulation of growth, and the effect is possibly most marked when overnutrition occurs during the 1st few years of

life, at a time when the potential for accelerating the rate of cell multiplication is greatest.

Obese children as a group tend to be taller than average (Mossberg, 1948; Wolff, 1955); Wolff (1955) in a consecutive series of 100 obese children found that the height was, on average, 0.04 m above the standard. The final height reached is, however, not above average (Lloyd, Wolff & Whelen, 1961) and the reason for this is that the onset of puberty is earlier by about 1 year in obese children of both sexes, so that growth finally ceases at an earlier age.

Effects on the adipose organ

Recently techniques have been developed for estimating the size and number of adipose cells in samples of tissues obtained by aspiration of subcutaneous tissue. If at the same time an estimate of total body fat is made, the total number of adipose cells in the body can be calculated. Using the method of Hirsch & Gallian (1968) to analyze samples of tissue, and skinfold thickness measurements to estimate total body fat content (Brook, 1971), Brook, Lloyd & Wolff (1972) have studied adipose tissue cellularity in a group of obese children. The total number of adipose cells was found to be increased (compared with the values obtained for non-obese children undergoing elective surgery) in about half the children, and a retrospective enquiry suggested that those obese children with increased numbers of adipose cells had already become obese during the 1st year of life. By contrast, those obese children with a normal number of adipose cells had only become obese later in childhood. Studies of adipose tissue cellularity in adults have shown that obese adults with increased numbers of cells are likely to have been obese during childhood (Hirsch & Knittle, 1970; Brook, Lloyd & Wolff, 1972). Therefore the capacity for adipose cells to increase their rate of multiplication in response to overnutrition apparently decreases after childhood and may be greatest in early infancy.

Studies of the size of adipose cells have shown that on average the mean cell size in obese children is increased compared with that found in non-obese children (Bonnet, Gosselin, Chautrain & Seutterre, 1971; Brook *et al.* 1972). During a period of weight loss, cell size has been shown to decrease, but cell number remains unchanged, at least in the short term (Brook *et al.* 1972).

Although it is tempting to speculate about the consequences of increased adipose tissue cellularity, there is at present insufficient information to justify this. Many technical and methodological problems in the estimation of adipose cells, especially in young babies, are still unsolved and this aspect has recently been reviewed by Widdowson (1973). Our knowledge of the normal rate of cell multiplication during infancy and childhood is still incomplete. Prospective studies are required before we can be certain whether increased cellularity is usually the result of overnutrition during the 1st year of life, or indeed that overnutrition at this time always results in an increased rate of cell multiplication. Long-term follow-up studies are needed to show whether increased cellularity will persist indefinitely if weight is lost, and whether the tendency for obesity to recur even after successful weight loss is the result of increased cellularity.

REFERENCES

- Asher, P. (1966). *Archs Dis. Childh.* **41**, 672.
- Bakwin, H. (1973). *Devl Med. Child Neurol.* **15**, 178.
- Bonnet, F., Gosselin, L., Chautraîne, J. & Scuterre, J. (1971). *Proc. XIII int. Congr. Pediat.* Vol. 7, p. 231.
- Brook, C. G. D. (1971). *Archs Dis. Childh.* **46**, 182.
- Brook, C. G. D., Lloyd, J. K. & Wolff, O. H. (1972). *Br. med. J.* **2**, 25.
- Canning, H. & Mayer, J. (1966). *New Engl. J. Med.* **275**, 1172.
- Durnin, J. G. V. A. & Rahaman, M. M. (1967). *Br. J. Nutr.* **21**, 681.
- Eid, E. E. (1970). *Br. med. J.* **2**, 74.
- Haase, K-E & Hosenfeld, H. (1956). *Z. Kinderheilk.* **78**, 1.
- Hirsch, J. & Gallian, E. (1968). *J. Lipid Res.* **9**, 110.
- Hirsch, J. & Knittle, J. L. (1970). *Fedn Proc. Fedn Am. Socs exp. Biol.* **29**, 1516.
- Howard, A. N., Dub, I. & McMahan, M. (1971). *Practitioner* **207**, 662.
- Hutchinson-Smith, B. (1970). *Med. offr* **123**, 257.
- Lloyd, J. K., Wolff, O. H. & Whelen, W. S. (1961). *Br. med. J.* **2**, 145.
- Mossberg, H. O. (1948). *Acta Paediat., Stockh.* **35**, Suppl. 2.
- Mullins, A. G. (1958). *Archs Dis. Childh.* **33**, 307.
- Newens, M. E. & Goldstein, H. (1972). *Br. J. prev. soc. Med.* **26**, 33.
- Newman, H. H., Freeman, F. N. & Holzinger, K. J. (1937). *A Study of Heredity and Environment*. Chicago: University of Chicago Press.
- Shukla, A., Forsyth, H. A., Anderson, C. M. & Marwah, S. M. (1972). *Br. med. J.* **4**, 507.
- Tanner, J. M. & Whitehouse, R. H. (1962). *Br. med. J.* **1**, 446.
- Tanner, J. M., Whitehouse, R. H. & Takaishi, M. (1966). *Archs Dis. Childh.* **41**, 454.
- Widdowson, E. M. (1973). *Archs Dis. Childh.* (In the Press.)
- Wolff, O. H. (1955). *Q. Jl Med.* **24**, 109.
- Wolff, O. H. (1965). In *Recent Advances in Paediatrics* 3rd ed., p. 216 [D. Gairdner, editor]. London: Churchill.