

Childhood Obesity: The Health Issue

Richard J. Deckelbaum and Christine L. Williams

Abstract

DECKELBAUM, RICHARD, AND CHRISTINE L. WILLIAMS. Childhood obesity: the health issue. *Obes Res.* 2001;9:239S–243S.

Overweight and obesity in children is epidemic in North America and internationally. Approximately 22 million children under 5 years of age are overweight across the world. In the United States, the number of overweight children and adolescents has doubled in the last two to three decades, and similar doubling rates are being observed worldwide, including in developing countries and regions where an increase in Westernization of behavioral and dietary lifestyles is evident. Comorbidities associated with obesity and overweight are similar in children as in the adult population. Elevated blood pressure, dyslipidemia, and a higher prevalence of factors associated with insulin resistance and type 2 diabetes appear as frequent comorbidities in the overweight and obese pediatric population. In some populations, type 2 diabetes is now the dominant form of diabetes in children and adolescents. Disturbingly, obesity in childhood, particularly in adolescence, is a key predictor for obesity in adulthood. Moreover, morbidity and mortality in the adult population is increased in individuals who were overweight in adolescence, even if they lose the extra weight during adulthood. Although the cause of obesity in children is similar to that of adults (i.e., more energy in vs. energy utilized), emerging data suggest associations between the influence of maternal and fetal factors during intrauterine growth and growth during the first year of life, on risk of later development of adult obesity and its comorbidities. In addition, recent data suggest that varying biological responses in different racial/ethnic groups differently contribute to overweight, obesity, and their comorbidities. Although differences in gene–nutrient interactions may contribute, the role of varying cultural and socioeconomic variables still needs to be determined to understand these disparities. Novel approaches in the prevention and

treatment of childhood overweight and obesity are urgently required. With the strong evidence that a lifecycle perspective is important in obesity development and its consequences, consideration must be focused on prevention of obesity in women of child-bearing age, excessive weight gain during pregnancy, and the role of breast-feeding in reducing later obesity in children and adults. Consideration must be given to family behavior patterns, diet after weaning, and the use of new methods of information dissemination to help reduce the impact of childhood obesity worldwide.

Key words: pediatric obesity, adolescent obesity, obesity comorbidity

Introduction

Overweight and obesity are independent risk factors for increased morbidity and mortality throughout the lifecycle. For example, overweight and obesity in women are predictors of gestational diabetes during pregnancy and newborns with excessive birth weight (1). High birth weight is a predictor of overweight and obesity in adulthood and in cofactors associated with insulin resistance (2). In parallel with the worldwide increases in obesity prevalence, overweight and obesity in children are rising (3). Because obesity in childhood frequently tracks into adulthood, increases in childhood overweight and obesity clearly are major contributors to the adult obesity epidemic (4). Children express the same comorbidities that are associated with being overweight and obese as adults (4,5). Thus, being overweight during childhood brings with it comorbidities that will increase the duration of comorbidities in an individual by one to two decades, a factor that can increase the impact of a number of risk factors on adult diseases. In this article we review the increasing prevalence of childhood overweight and obesity, the impact of obesity in terms of a lifecycle perspective, comorbidities expressed in childhood obesity, and emerging evidence for different racial/ethnic responses to biological contributors to obesity. Potential approaches to fighting the obesity epidemic beginning in the childhood years are discussed.

Department of Pediatrics and the Institute of Human Nutrition, Columbia University, New York, New York.

Address correspondence to Dr. Richard Deckelbaum, Institute of Human Nutrition, PH 15-1512E, Columbia University, 630 West 168th Street, New York, NY 10032.

E-mail: rjd20@columbia.edu

Copyright © 2001 NAASO

Increasing Prevalence of Childhood Obesity

The increase in obesity prevalence has been observed in the United States and internationally from pre-schoolchildren to adolescence. These increases have been noted in all racial and ethnic groups, but some groups are affected more than others. In the United States, the National Health and Nutrition Surveys (NHANES) databases offer alarming statistics showing substantial increases in the prevalences of overweight [defined as the 85th to 95th percentiles of the weight for length growth references; (5)]. At present, nearly 8% of children 4 to 5 years of age in the United States are overweight. Whereas, in general, the increases in prevalence cross the entire pediatric population, girls have been more affected than boys. Between the NHANES I and NHANES III surveys, a period of 20 years, prevalence for overweight and obesity in young girls has increased more than 2-fold, whereas that of boys increased less, ~25%. However, in children older than 6 years of age, and particularly in adolescence, there has been an approximate doubling of obesity prevalence in boys as well as in girls in the United States in the same time (5).

Ethnicity differences are also apparent by varying rates of increase. For boys and girls, overweight is highest in Mexican American children, intermediate among non-Hispanic black children, and lowest in non-Hispanic white children. In examining what can be defined as the overweight group in childhood (i.e., above the 85th percentile cutoff), nearly 22% of pre-schoolchildren in the United States can be defined as overweight and 10% as obese. This compares with 18.6% and 8.5%, respectively, in 1983. In the Bogalusa Heart Study in Louisiana, the prevalence of overweight among 5- to 24-year-olds from the biracial community increased ~2-fold between 1973 and 1994. Of particular concern is that the yearly increases and relative weight and obesity during the latter part of the study (1983 to 1994) were ~50% greater than those between 1973 and 1982 (6). In addition, independent of racial/ethnic differences, lower socioeconomic status is another important predictor for high overweight and obesity prevalence in U.S. children (5).

Similar disturbing trends in increasing obesity prevalence are being recorded in other industrialized settings. For example, in Japan the frequency of obese schoolchildren between the ages of 6 and 14 years increased from 5% to 10% and that of extremely obese children from 1% to 2% between 1974 and 1993 (3). Childhood obesity is not limited to the industrialized countries. In a recent review, De Onis and Blössner (7) reported rapidly increasing prevalence of overweight and obesity among pre-schoolchildren in developing countries. Of interest, certain countries demonstrated high percentages of overweight at the same time as high frequencies of wasted (malnourished) children were also measured. Specific examples include Northern Africa, where the percentage of overweight children exceeded 8% and wasted children were reported at over 7%. Similarly, in

Eastern Asia, 4.3% of pre-schoolchildren were overweight and 3.4% wasted. In South America, where malnutrition and underweight were once predominant, the percentage of overweight pre-schoolchildren was close to 5%, but wasted children were now only 1.8%. In a number of countries (e.g., Egypt, Argentina, Malawi, Nigeria, Uzbekistan, Peru, Qatar, South Africa, Jamaica), the percentage of overweight children exceeds that of the United States. In 38 countries where secular data are available, 16 showed a rising trend in obesity prevalence over time, 14 were static, and only 8 showed falling rates in obesity prevalence. Rates of increase seem most marked in countries of Northern Africa, such as Morocco and Egypt, as well in some countries of the Caribbean and South America (7). Thus, obesity in children can no longer be classified as a Western problem alone; it is now shared by nearly all industrialized areas and many developing countries.

Overweight and Obesity in Children Predicts Overweight Later in Life

Data from a number of studies provide strong evidence that higher levels of body mass index (BMI) during childhood can predict overweight later in life. This was recently summarized in a review by Goran (8). Data from four longitudinal studies were reviewed and showed that the probability of overweight at 35 years of age for children with BMI in the 85th to 95th percentiles increased with increasing age. The prediction for adult weight was most accurate for BMI at 18 years of age with accuracy decreasing for BMI below 13 years of age. Goran (8) concluded that the "persistence of pediatric obesity into adulthood increases according to the age at which obesity is initially present." Similar to what has been recorded in North America, obesity during childhood in Japan is associated with increased likelihood of obesity during adulthood. In a Japanese study, approximately one-third of obese children grew into obese adults (9). Whitaker et al. (10) found that the risk of adult obesity was greater in both obese and non-obese children if at least one parent was overweight. This effect was most pronounced in children that were <10 years old; over the age of 10 years, the child's own overweight/obesity status was a better predictor than having an obese parent. These studies show the importance of the family environment in contributing to the increasing prevalence of obesity. Most likely these increases are associated with changes (increases) in food supply and caloric intake accompanied by diminishing levels of physical activity. One might consider that these family studies provide strong evidence for the genetic contribution to obesity. However, it is very unlikely that changing gene pools can explain the doubling or even tripling of obesity prevalence rates in certain groups over 20 years; too short a period to affect the genetic background in affected populations.

Must et al. (11) presented data relating to the outcomes of overweight adolescents who were followed up to 50 years. Both men and women who were overweight at adolescence had increased age-specific morbidity and mortality relating to cardiovascular and other chronic diseases. Increased risk was also present even if adolescents who were obese had lost the excess weight during the adult period (11), suggesting that obesity during adolescence may set triggers that are associated with adverse risk in the adult.

An emerging area of research is the potential role of intrauterine growth and growth in the first year of life for predicting the emergence of increased cardiovascular risk and obesity during adulthood. These potential links were critically commented on by Dietz and Gortmaker (4). In reviewing the Dutch Famine Studies, they noted that individuals who were exposed in utero to famine in the first trimester of pregnancy were more likely to be overweight at 18 years of age compared with those exposed to famine at other periods during pregnancy. In contrast, individuals exposed to famine late in pregnancy tended to be underweight at 18 years of age. Although low birth weight and low weight gain in the first year of life may contribute to increased risk of hypertension, dyslipidemia, and cardiovascular risk in the adult population, Dietz and Gortmaker conclude that it is unlikely that low birth weight contributes significantly to obesity prevalence in the adult population. In addition, although an individual who is born overweight (≥ 4000 g) does have a higher risk of being an overweight adult, these authors suggest that $<5\%$ of adult obesity is attributable to individuals born with high birth weights.

Comorbidities of Childhood Obesity

Obesity-associated chronic disease risk factors are present in adults and also manifest in overweight and obese children. For example, data from the Bogalusa Heart Study showed that $\sim 60\%$ of overweight 5- to 10-year-old children had one cardiovascular risk factor, such as high blood pressure, hyperlipidemia, or elevated insulin levels (12). From the same cohort of 5- to 10-year-olds, $>20\%$ of overweight children had two or more cardiovascular risk factors (12)—risk factors that would increase substantially the risk of these individuals for earlier cardiovascular disease if they were tracked into adulthood.

Similar to adults, children who are moderately overweight showed that an elevation of low-density lipoprotein (LDL) cholesterol levels and hypercholesterolemia does not increase substantially with higher degrees of obesity. With more marked degrees of obesity, rises in plasma triglyceride levels and decreases of high-density lipoprotein cholesterol are more common, and blood pressure elevations are more common with significant obesity than with moderate overweight, similar to what occurs in adults.

With the rising prevalence of overweight and obesity in children, noninsulin-dependent diabetes mellitus (type 2

diabetes) is increasingly a pediatrician's problem. In one report (13), 4% of new diagnoses of diabetes before 1992 were classified as type 2 diabetes. In 1994, 16% of new diabetics were classified as type 2, a 4-fold increase. In the Cincinnati area between 1982 and 1994, there was a 10-fold increase in type 2 diabetes in children and the African American population was more severely affected than the white population (13).

Alarming findings are now emerging from Asian countries that are rapidly Westernizing their lifestyle habits. For example, in urban Japanese children, plasma total cholesterol levels and LDL cholesterol now exceed those found in U.S. children (14). A recent analysis examining differences between the Japanese and American pediatric populations suggests that dietary habits, exercise, and adiposity differences do not explain the varying lipid levels between U.S. and Japanese children. The hypothesis was raised that the populations not previously exposed to Western diets and lifestyles may have more adverse effects on expression of cardiovascular risk factors than long-exposed populations (14). In Japanese children, overweight and obesity are also associated with substantial elevations of plasma total and LDL cholesterol levels and type 2 diabetes, and in some areas of Japan, type 2 diabetes is more common in children now than type 1 diabetes (15).

Other comorbidities are also associated with childhood obesity. These include orthopedic problems, such as Blount's disease, skin fungal infections, and acanthosis nigricans, hepatic steatosis and steato-hepatitis; pseudotumor cerebri; and psychological and behavioral problems. Psychological problems associated with childhood obesity include negative self-esteem, withdrawal from interaction with peers, depression, anxiety, and the feeling of chronic rejection (5). Thus, a strong body of evidence suggests that BMI in childhood is associated with various adverse biochemical, physiological, and psychological effects, many of which have the possibility of tracking into chronic disease risk factors in adulthood.

There are different biological effects related to overweight and obesity in different racial/ethnic groups. Evidence is available to suggest that aerobic capacity may be lower in African American than in white children and may be more significant than energy expenditure leading to obesity (8). Goran (8) concluded that fasting insulin and acute insulin response are significantly higher, and insulin sensitivity is significantly lower in African American than in white prepubertal children; these differences are not explained by differences in body fat, body fat distribution, diet, or physical activity. These findings are important because they suggest that prevention and treatment strategies may require different approaches in different racial/ethnic populations.

Although heritability estimates of genetic population studies suggest that 40% to 70% of adult obesity is due to

genetic factors, there is little information available yet in pediatric populations. Nevertheless, statistics show that a proportionate variance within adult populations is accounted for by genetic factors, but this does not reflect the interplay between genes and environment at the individual level, and especially within the developing individual.

Prevention/Treatment of Childhood Obesity

Increase in energy intake and decrease in physical activity are the primary environmental influences on childhood obesity, similar to adult obesity. In 45 minutes of exercise, a 165-kg (75-lb) child may be expected to expend 90, 525, 135, and 180 calories during continuous bicycling, running, walking, and dancing, respectively. These expenditures can be contrasted to the difference of a regular size McDonald's meal, which provides ~600 calories vs. a super-sized McDonald's double cheeseburger meal, which provides >1800 calories. Note that the calories expended in the exercises above do not nearly cover this difference. In approaching childhood obesity, we can consider three levels of prevention: primordial prevention, which aims toward maintaining normal BMI throughout childhood and adolescence; primary prevention, directed toward preventing overweight children (BMI: 85th to 95th percentiles) from becoming obese; and secondary prevention, to treat obese children (BMI > 95%) to reduce comorbidities and reverse overweight and obesity, if possible (5). Clearly, there is a need to balance energy intake with energy output and to introduce activity in place of inactivity. In obesity prevention, an emphasis on plant-based foods and vegetable and fruit consumption would be a major step forward in avoiding energy-dense foods.

At different stages of development, we suggest the following components for obesity prevention:

- Perinatal: supply good prenatal nutrition and health care, avoid excessive maternal weight increase, control diabetes, help mothers lose weight postpartum, and offer nutrition education.
- Infancy: encourage increased breast-feeding and continuous breastfeeding to ≥ 6 months of age, delay introduction of solid foods until after 6 months of age, provide a balanced diet and avoid excess high-calorie snacks, and follow weight increase closely.
- Preschool: provide early experiences with foods and flavors, help develop healthy food preferences, encourage appropriate parental feeding practices, monitor rate of weight increases to prevent early adiposity rebound, and provide child and parent nutrition education.
- Childhood: monitor weight increase for height (slow down if excessive), avoid excessive prepubertal adiposity, supply nutrition education, and encourage daily physical activity.
- Adolescence: prevent excess weight increase after growth spurt, maintain healthy nutrition as the next generation of parents, and continue daily physical activity.

For children who are significantly overweight, the goal should be to reduce severity of obesity and to treat, reduce, and eliminate comorbidities (e.g., hypertension, dyslipidemia, insulin resistance, and type 2 diabetes). For energy balance, measures are needed for children to lose weight or to slow down the rate of gain and to grow into their expected heights. This requires some reduction in energy intake and substantial increases in energy expenditure.

In summary, childhood obesity is increasing at epidemic rates, even among pre-schoolchildren and is accompanied by significant comorbidities and health problems. Prevention should be the primary goal and, if successful, will help reduce adult obesity. Accordingly, we will have the greatest chance to successfully reverse the obesity epidemic if we consider it a crisis, make it a funded government and public health priority, and join forces across disciplines to mount an effective public health campaign in the prevention and early treatment.

Acknowledgments

This work was supported in part by NIH grant no. HL50321.

References

1. **Institute of Medicine, National Academy of Sciences.** *Nutrition During Pregnancy.* Washington, DC: National Academy Press; 1990.
2. **Curhan GC, Willett WC, Spiegelman D, Colditz GA, et al.** Birth weight and adult hypertension and obesity in women. *Circulation.* 1996;94:1310–5.
3. **World Health Organization.** *Obesity: Preventing and Managing the Global Epidemic.* World Health Organization Technical Support Series No. 894. Geneva, Switzerland: World Health Organization; 2000.
4. **Dietz WH, Gortmaker SL.** Preventing obesity in children and adolescents. *Annu Rev Public Health.* 2001;22:337–53.
5. **Williams CL.** Can childhood obesity be prevented? In: Bendich A, Deckelbaum RJ, eds. *Primary and Secondary Preventive Nutrition.* Totowa, NJ: Humana Press; 2001, pp. 185–204.
6. **Freedman DS, Srinivasan SR, Valdez RA, Williamson DF, Berenson GS.** Secular increases in relative weight and adiposity among children over two decades: the Bogalusa Heart Study. *Pediatrics.* 1997;99:420–6.
7. **De Onis M, Blössner M.** Prevalence and trends of overweight among pre-schoolchildren in developing countries. *Am J Clin Nutr.* 2000;72:1032–9.
8. **Goran MI.** Metabolic precursors and effects of obesity in children: a decade of progress, 1990–1999. *Am J Clin Nutr.* 2001;73:158–71.
9. **Kotani K, Nishida M, Yamashita S, et al.** Two decades of annual medical examinations in Japanese obese children: do obese children grow into obese adults? *Int J Obes Relat Metab Disord.* 1997;21:912–21.

10. **Whitaker RC, Wright JA, Pepe MS, Seidel KD, Dietz WH.** Predicting obesity in young adulthood from childhood and parental obesity. *N Engl J Med.* 1997;337:869–73.
11. **Must A, Jacques PF, Dallal GE, Bajema CJ, Dietz WH.** Long term morbidity and mortality of overweight adolescents: a follow-up of the Harvard Growth Study of 1922 to 1935. *N Engl J Med.* 1992;327:1350–5.
12. **Freedman DS, Dietz WH, Srinivasan SR, Berenson GS.** The relation of overweight to cardiovascular risk factors among children and adolescents: the Bogalusa Heart Study. *Pediatrics.* 1999;103:1175–82.
13. **Pinhas-Hamiel O, Dolan LM, Daniels SR, Standiford D, Khoury PR, Zeitler P.** Increased incidence of non-insulin-dependent diabetes mellitus among adolescents. *J Pediatr.* 1996;128:608–15.
14. **Couch SC, Cross AT, Kida K, Ross E, Plaza I, Shea S, Deckelbaum R.** Rapid Westernization of children's blood cholesterol in 3 countries: evidence for nutrient-gene interactions? *Am J Clin Nutr.* 2000;72(suppl):1266S–74.
15. **Kida K, Ito T, Yang SW, Tahphaichitr V.** Effects of western diet on risk factors of chronic diseases in Asia. In: Bendich A, Deckelbaum RJ, eds. *Preventive Nutrition: The Comprehensive Guide for Health Professionals.* 2nd ed. Totowa, NJ: Humana Press Inc.; 2001, pp. 435–46.