Seminar

Childhood obesity: public-health crisis, common sense cure

Cara B Ebbeling, Dorota B Pawlak, David S Ludwig

During the past two decades, the prevalence of obesity in children has risen greatly worldwide. Obesity in childhood causes a wide range of serious complications, and increases the risk of premature illness and death later in life, raising public-health concerns. Results of research have provided new insights into the physiological basis of bodyweight regulation. However, treatment for childhood obesity remains largely ineffective. In view of its rapid development in genetically stable populations, the childhood obesity epidemic can be primarily attributed to adverse environmental factors for which straightforward, if politically difficult, solutions exist.

Historically, a fat child meant a healthy child, one who was likely to survive the rigors of undernourishment and infection. In the past decade, however, excessive fatness has arguably become the primary childhood health problem in developed nations and, to some degree, in other parts of the world. Here we review the scope of the problem and discuss developments in establishment of cause, prevention, and treatment of obesity. We argue that fundamental changes in the social environment will be needed to combat this emerging public-health crisis.

International epidemic of childhood obesity

The definitions of overweight and obesity in children differ between epidemiological studies, making comparisons of cross-sectional prevalence data difficult. Nevertheless, several studies have examined change in prevalence within populations over time, and the results of these analyses are astounding. Rates have increased 2·3-fold to 3·3-fold over about 25 years in the USA, 2·0-fold to 2·8-fold over 10 years in England, and 3·9-fold over 18 years in Egypt (figure 1).[1–7] The distribution of body-mass index (BMI) has shifted in a skewed fashion, such that the heaviest children, at greatest risk of complications, have become even heavier.[8] This epidemic has affected a wide age range, most ethnic groups, and people of every socioeconomic status, though sometimes in disproportionate ways.[9,10] In the USA, prevalence rose more than twice as fast among minority groups compared with white groups, exacerbating pre-existing racial-ethnic disparities.[10] The urban poor in developed countries might be especially vulnerable because of poor diet[11] and limited opportunity for physical activity.[12] Conversely, childhood obesity is most frequent in upper socioeconomic strata of developing nations, where over-nutrition and undernutrition coexist, probably owing to adoption of an increasingly Western lifestyle.[13–15]

Lancet 2002, **360:** 473–82

Division of Endocrinology, Children's Hospital Boston, 300 Longwood Avenue, Boston, MA 02115, USA (C B Ebbeling PhD, D B Pawlak PhD, D S Ludwig MD)

Correspondence to: Dr David S Ludwig (e-mail: david.ludwig@tch.harvard.edu)

Complications of childhood obesity

Childhood obesity is a multisystem disease with potentially devastating consequences (figure 2).[16–40] Several complications warrant special attention.

As with adults, obesity in childhood causes hypertension, dyslipidaemia, chronic inflammation, increased blood clotting tendency, endothelial dysfunction, and hyperinsulinaemia.[25–29] This clustering of cardiovascular disease risk factors, known as the insulin resistance syndrome, has been identified in children as young as 5 years of age.[41] Among adolescents and young adults who died of traumatic causes, the presence of cardiovascular disease risk factors correlated with asymptomatic coronary atherosclerosis, and lesions were more advanced in obese individuals.[42,43] Furthermore, in a British cohort,[44] overweight in childhood increased the risk of death from ischaemic heart disease in adulthood two-fold over 57 years.

Type 2 diabetes, once virtually unrecognised in adolescence, now accounts for as many as half of all new diagnoses of diabetes in some populations.[45] This condition is almost entirely attributable to the paediatric obesity epidemic, though heredity and lifestyle factors affect individual risk.[32] Of particular concern, a prediabetic state, consisting of glucose intolerance and insulin resistance, seems to be highly prevalent among severely obese children irrespective of ethnic group, even before formal diagnostic criteria for diabetes have been met.[46] The emergence of type 2 diabetes in children represents an ominous development, in view of the macrovascular (heart disease, stroke, limb amputation) and microvascular (kidney failure, blindness) sequelae.

Frequent pulmonary complications include sleep-disordered breathing (sleep apnoea),[22] asthma,[23] and exercise intolerance.[24] Development of asthma or exercise intolerance in an obese child can limit physical activity

Search strategy

We identified original research, reviews, and commentaries by searching computer databases—eg, Medline, PsycINFO, Agricola, Lexis-Nexis—and by reviewing issues of journals that publish obesity research. We directed special attention towards publications since 1997. Research developments and published work were also identified by discussions with specialists in the areas of paediatric obesity, nutrition, and public health. We obtained information with respect to fast-food consumption from relevant web sites.

For personal use. Only reproduce with permission from The Lancet Publishing Group.

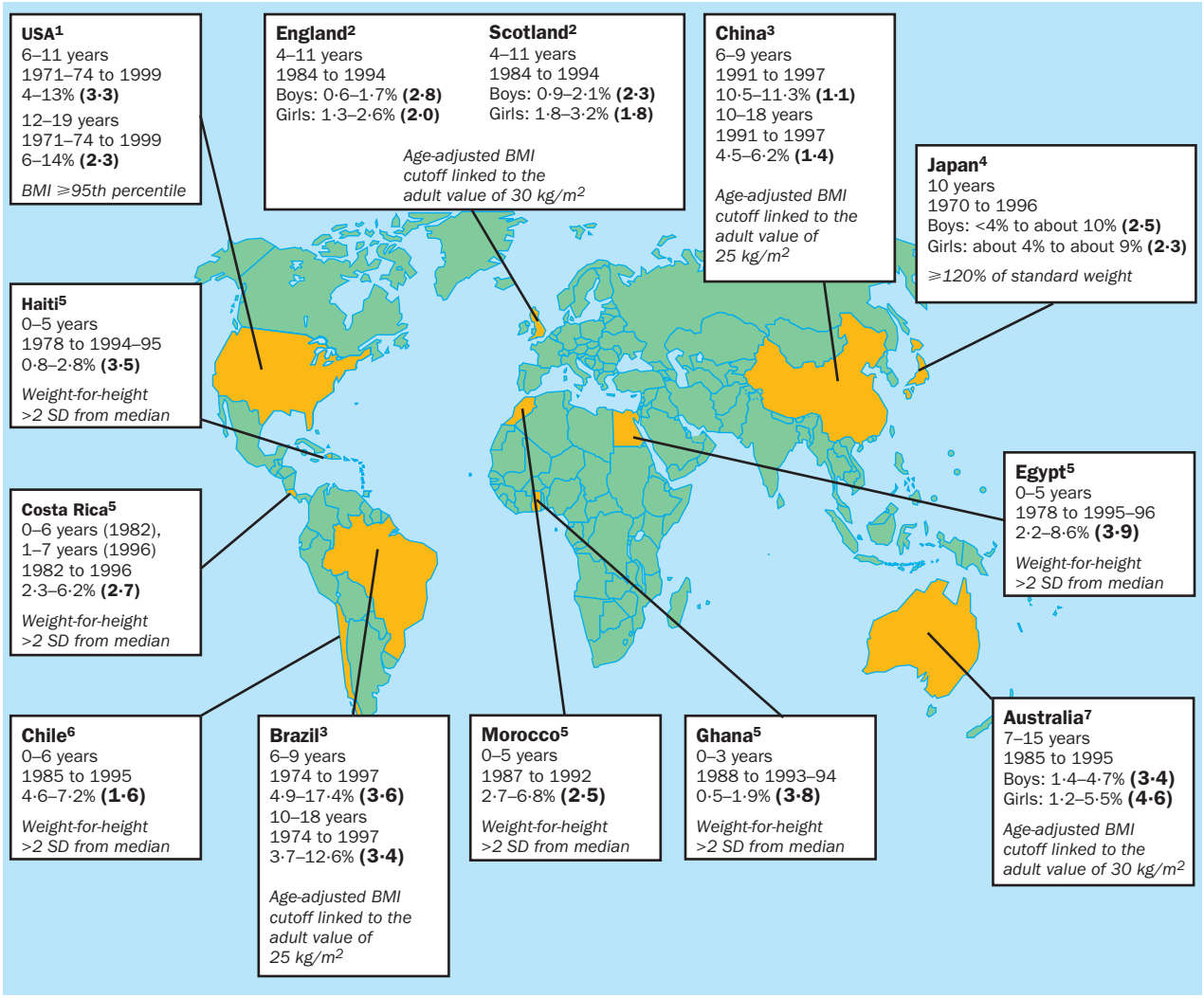

Figure 1: **Global increases in prevalence of childhood obesity**
Change factors are listed in bold for increases in prevalence over specified time intervals. Definitions of overweight and obesity are in italics.

and thus cause further weight gain. Furthermore, serious hepatic, renal, musculoskeletal, and neurological complications have been increasingly recognised.[21,30,36,37]

Findings of many studies indicate substantial psychosocial consequences of childhood obesity. Obese children are stereotyped as unhealthy, academically unsuccessful, socially inept, unhygienic, and lazy.[47] Health-care providers with expertise in obesity treatment share these negative stereotypes to some degree.[48] Overweight children as young as age 5 years can develop a negative self-image,[17] and obese adolescents show declining degrees of selfesteem associated with sadness, loneliness, nervousness, and high-risk behaviours.[18]

Risk of obesity-related complications can differ by ethnic origin and as a result of cultural factors. Black and Hispanic youths in the USA, for example, are at greater risk for type 2 diabetes and cardiovascular disease than their white counterparts.[45,49] Obesity only partly explains this raised disease risk, since fasting serum insulin concentration and prevalence of the insulin resistance syndrome remain much higher in minority youths after statistical adjustment for BMI or adiposity.[50,51] By contrast, adverse psychosocial effects are often more severe in white children, particularly girls, than in other ethnic groups.[52]

Causes of childhood obesity

Bodyweight is regulated by numerous physiological mechanisms that maintain balance between energy intake and energy expenditure.[53] These regulatory systems are extraordinarily precise under normal conditions—eg, a positive energy balance of only 500 kJ (120 kcal) per day (about one serving of sugar-sweetened soft drink) would produce a 50-kg increase in body mass over 10 years. Thus, any factor that raises energy intake or decreases energy expenditure by even a small amount will cause obesity in the long-term. Genetic factors can have a great effect on individual predisposition; however, rising prevalence rates among genetically stable populations indicate that environmental and, perhaps, perinatal factors must underlie the childhood obesity epidemic.

Genetic, perinatal, and early-life factors

In 1997, two massively obese Pakistani children of consanguineous parents were found to have a mutation in the gene encoding leptin,[54] a hormone normally produced by adipocytes and secreted in proportion to body-fat mass.[55] Since then, five genetic mutations that cause human obesity have been identified,[56] all presenting in childhood. Additionally, many candidate alleles, such as those in the variable nucleotide tandem repeat region of

For personal use. Only reproduce with permission from The Lancet Publishing Group.

the insulin gene, have been discovered that seem to affect risk of early-onset obesity.[57] Progress has been made in mapping the genetic loci of the Prader-Willi, Bardet-Biedl, Cohen, and Alstrom syndromes, though the molecular causes of these obesity syndromes have not yet been identified.[56] Single gene defects, however, account for a small fraction of human obesity.[56] Instead, predisposition to obesity seems to be caused by a complex interaction between at least 250 obesity-associated genes[58] and, perhaps, perinatal factors.

Whitaker and Dietz[59] advanced the intriguing hypothesis that prenatal overnutrition might affect lifelong risk of obesity. According to this hypothesis, maternal obesity increases transfer of nutrients across the placenta, inducing permanent changes in appetite, neuroendocrine functioning, or energy metabolism. Results of observational studies[59] show a direct relation between maternal obesity, birthweight, and obesity later in life; however, the relative contributions of shared maternal genes versus intrauterine factors are difficult to differentiate.[59] Findings of studies in animals indicate the potential long-term consequences of maternal obesity per se—the offspring of female rats with diet-induced obesity were heavier than the offspring of rats with the same genotype but without obesity.[60] The implications of these findings are formidable: the obesity epidemic could accelerate through successive generations independent of further genetic or environmental factors. However, undernutrition at important stages of fetal development can also induce permanent physiological changes that result in obesity, as indicated by an analysis of the Dutch famine cohort.[61] For this reason, the nutrition transition, as described by Popkin,[62] could place children in developing nations at particularly high risk of obesity. In view of these possibilities, an opportune time to initiate obesity prevention might be before conception.

Children who were bottle fed seem to be more at risk of obesity later in childhood than those who were breast fed.[63,64] The explanation for this finding could relate to permanent physiological changes caused by some intrinsic factor unique to human milk or to psychological factors, such as locus of control over feeding rate (baby versus parent) or taste preference.

During early childhood, BMI normally decreases until age 5–6 years, then increases through adolescence. The age at which this BMI nadir occurs has been termed the adiposity rebound.[65] Several observational studies[66,67] have described an increased risk for obesity later in life in individuals who have an early adiposity rebound. However, the biological importance and predictive value of this association remains a matter of debate.[68,69]

Physical activity

A lifestyle characterised by lack of physical activity and excessive inactivity (particularly television viewing) might cause obesity in children. Findings of a cross-sectional study[70] suggest that obese children in South Carolina spent less time in moderate and vigorous physical activity than their non-obese counterparts, and in a nationally representative cross-sectional study in the USA,[71] children who engaged in the least vigorous physical activity or the most television viewing tended to be the most overweight. Among children from Mexico City, obesity risk decreased by 10% for each hour per day of moderate-to-vigorous physical activity, and increased by 12% for each hour per day of television viewing.[72] Prospectively, physical activity was inversely associated with BMI change in girls, and media time (watching television or videos, playing video or computer games) was directly associated with BMI change in both sexes.[73] Moreover, low aerobic fitness predicts increased adiposity in black and white children.[74] However, as summarised by Goran and colleagues,[75] there are few data with respect to how qualitative aspects of physical activity, such as frequency and intensity, affect body composition and health risk.

The effect of television viewing on obesity risk is of particular interest. Television viewing is thought to promote weight gain not only by displacing physical activity, but also by increasing energy intake.[76,77] Children seem to passively consume excessive amounts of energy-dense foods while watching television. Furthermore, television advertising could adversely affect dietary patterns at other times throughout the day. US and British children are exposed to about ten food commercials per hour of television time (amounting to thousands per year), most for fast food, soft drinks, sweets, and sugar-sweetened breakfast cereal.[78–80] Exposure to 30-second commercials increases the likelihood that 3–5-year-olds would later select an advertised food when presented with options.[81] Moreover, television viewing during mealtime is inversely associated with consumption of products not typically advertised, such as fruits and vegetables.[82] In an experimental study by Robinson,[83] measures of adiposity increased significantly over an academic year in children in a control school who continued to watch television at their usual rates, compared with children in an intervention school who decreased television viewing by about 40%.

Diet

Fat—Opinions vary with respect to optimum macronutrient composition of paediatric diets, and dietary fat is central to deliberations.[84] Because fat is the most energy dense macronutrient, excessive

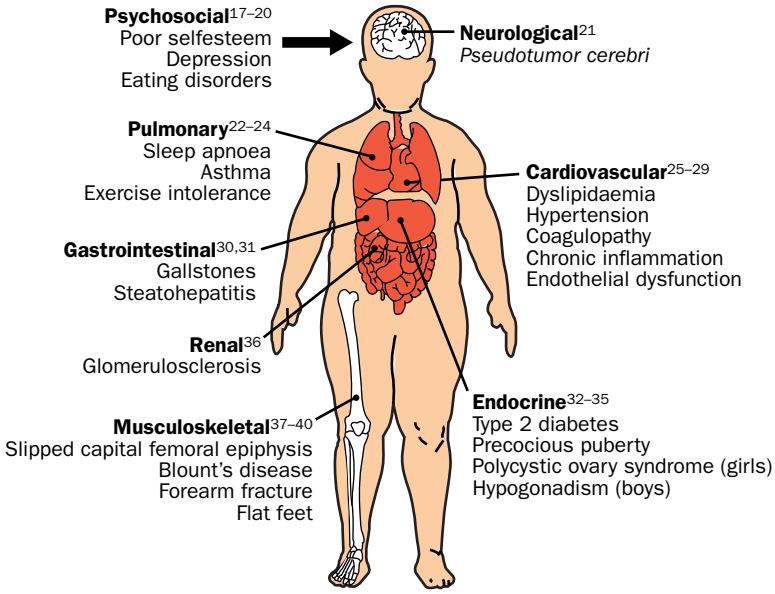

Figure 2: **Complications of childhood obesity**

For personal use. Only reproduce with permission from The Lancet Publishing Group.

consumption is often believed to cause weight gain.[84] However, the relation between dietary fat and adiposity has been questioned. Findings of epidemiological studies do not consistently show an association between dietary fat and adiposity in children and young adults.[85,86] Moreover, the prevalence of obesity has greatly increased, despite an apparent decrease in proportion of total calories consumed as fat in the diet of US children.[87,88] Thus, the potential effect of other dietary factors on bodyweight warrants careful consideration.

Type of dietary fat could be of greater importance than total fat consumption in the cause of obesity-related morbidities. The adverse effects of saturated fat on risk of cardiovascular disease are well documented.[89] Intake of partially hydrogenated (trans) fat, commonly found in commercial bakery products and fast foods,[90] increases risk for both cardiovascular disease[91] and type 2 diabetes[92] in adults. By contrast, unsaturated fats from vegetable and marine sources decrease risk of these diseases.[91,92]

Carbohydrate—The decrease in dietary fat observed at a population level has been accompanied by a compensatory increase in carbohydrate consumption, especially in the form of refined foods—eg, breads, ready-to-eat cereals, potatoes, soft drinks, cakes, and biscuits.[88,93] High glycaemic index foods like these produce fairly large increases in postprandial blood glucose concentrations[94] and could play a part in appetite regulation.[95,96] Consumption of meals composed predominately of high glycaemic index foods induces a sequence of hormonal events that stimulate hunger and cause overeating in adolescents.[97] A high glycaemic index diet has been linked with risk for central adiposity,[98] cardiovascular disease,[99] and type 2 diabetes[100] in adults. Nevertheless, the importance of glycaemic index in the cause of obesity and related morbidities has not been substantiated in long-term clinical trials.

Sugar-sweetened soft drinks have been the subject of several studies,[88,101] in part because of the rapid increase in their rate of consumption by children. Results of a cross-sectional study[102] showed that total energy intake was about 10% greater among school-age children who consumed soft drinks than in those who did not. Additionally, findings of a prospective observational study[103] indicated a 60% increased risk of development of obesity in middle-school children for every additional daily serving, after controlling for the effects of potentially confounding factors. Sugar-sweetened soft drinks might promote energy intake and excessive weight gain because of their high glycaemic index[104] or because compensation for calories consumed in liquid form is less complete than for calories consumed in solid form.[103] By contrast, milk, a low glycaemic index beverage, seems to protect overweight young adults from becoming obese.[105]

Energy density—As reviewed by Rolls,[106] energy density seems to affect satiety and food consumption, at least in the short term. Adults consumed substantially less energy when served test meals with a low, rather than a high, energy density, irrespective of fat content (25%, 35%, or 45% of total energy).[107] The energy density of children's diets is directly associated with not only fat but also a range of starchy foods, including breakfast cereal, bread, and potatoes.[108]

Portion size—Despite pervasive commercial trends toward large portions, there is little research into the effects of portion size on food intake. In a study[109] in which preschool children were given lunches that contained small, medium, or large amounts of macaroni and cheese, and in whom voluntary energy consumption was measured, younger children (mean age 3·6 years) ate the same amount irrespective of portion size, whereas older children (5·0 years) consumed more energy when given a large versus a small portion. These findings suggest that, as children grow older, they become less responsive to internal hunger and satiety cues and more reactive to environmental stimuli.

Fast food—The rise in consumption of fast food, in developed and developing nations, might have particular relevance to the childhood obesity epidemic. Fast food typically incorporates all of the potentially adverse dietary factors described above, including saturated and trans fat, high glycaemic index, high energy density, and, increasingly, large portion size. Additionally, these foods tend to be low in fibre, micronutrients, and antioxidants; dietary components that affect risk of cardiovascular disease and diabetes.[110,111] Results of several studies[112–114] suggest an association between fast-food consumption and total energy intake or bodyweight in adolescents and adults. Although there are no data on fast food and obesity in children, adolescent girls who ate fast food four times a week or more consumed about 770–1095 kJ (185–260 kcal) per day more than those who did not.[115] A large fast food meal (double cheeseburger, french fries, soft drink, dessert) could contain 9200 kJ (2200 kcal), which, at 350 kJ (85 kcal) per mile, would require a full marathon to burn off.

Family factors

Parent-child interactions and the home environment can affect behaviours related to risk of obesity. Family life has changed a lot over the past two decades, with trends towards eating out and greater access to television than previously. Children consume more energy when meals are eaten in restaurants than at home,[116] possibly because restaurants tend to serve larger portions of energy dense foods. A bedroom television increases viewing by 38 min per day.[117] By contrast, eating family dinner seems to decrease television viewing[117] and improve diet quality (less saturated and trans fat, less fried food, lower glycaemic load, more fibre, fewer soft drinks, and more fruits and vegetables).[118] Moreover, social support from parents and others correlates strongly with participation in physical activity.[119] In view of these results—relating psychosocial factors to dietary and physical activity behaviours that affect energy balance—it is not surprising that children who suffer from neglect, depression, or other related problems are at substantially increased risk for obesity during childhood and later in life.[120–123]

Prevention and treatment

Prevention and treatment of obesity ultimately involves eating less and being more physically active. Though this action sounds simple, long-term weight loss has proven exceedingly difficult to achieve. A US National Institutes of Health consensus statement indicated that adults who remain in conventional weight loss programmes can realistically expect a maximum weight loss of only 10% (a small fraction of excess adiposity). About half of this modest weight loss is regained within a year, and virtually all is regained within 5 years.[124] The relative intellectual and psychological immaturity of children compared with adults, and their susceptibility to peer pressure present additional practical obstacles to the successful treatment of childhood obesity. For this reason, most efforts to reduce obesity in children have used either family-based or school-based approaches, though pharmacological and surgical treatments are also available.[125]

For personal use. Only reproduce with permission from The Lancet Publishing Group.

Family-based intervention

Family intervention is implemented on the premise that parental support, family functioning, and home environment are important determinants of treatment outcomes. Although this premise cannot be refuted, a review of randomised controlled studies led Epstein and co-workers[126] to conclude that "most pediatric obesity interventions are marked by small changes in relative weight or adiposity and substantial relapse". For instance, one study[127] reported that children who received dietary counselling, encouragement to exercise, and family therapy for 14–18 months had a smaller increase in BMI than controls, who received no treatment (1·1 *vs* 2·8 kg/m^2, 1 year after therapy stopped), though drop-out rate was substantial. In another study,[128] 34 children showed moderate decreases in proportion overweight after 6-months' family interventions that used cue control and a reward system to foster behaviour change. However, at 3-years' follow-up, the proportion overweight approached or exceeded baseline values. In another study,[129] cognitive behavioural treatment was used to promote dietary change and aerobic exercise. Decrease in proportion overweight at 4·6 years among 109 of 136 children available for follow-up was greater in response to treatment (15%) compared with only advice (7%), but most children remained substantially obese. In possibly the only successful long-term intervention, Epstein and colleagues[130] used behavioural strategies (contracting, self-monitoring, social reinforcement, modelling) with obese children and their parents to limit consumption of high-calorie foods and increase aerobic exercise. A decrease in proportion overweight (7·5%) was noted at 10-years' follow-up in the experimental group compared with an increase in untreated controls (14·3%). However, individuals were selectively recruited based on motivation to change and likelihood of success, and less than half of the children in the experimental group maintained a 20% decrease in proportion overweight.[131,132]

School-based intervention

School-based efforts have been oriented towards prevention, targeting all students in selected classes to avoid stigmatisation of obese children. Planet Health is an interdisciplinary curriculum that aims to decrease dietary fat consumption, increase consumption of fruits and vegetables, promote physical activity, and limit television time. Over 2 academic years, prevalence of obesity significantly decreased among girls, but not boys, in intervention versus control schools.[133] The intervention effect was attributed to reduced television viewing. The APPLES (Active Programme Promoting Lifestyle Education in Schools) intervention involved nutrition education, provision of healthy cafeteria lunches, a fitness programme, improved playground facilities, and extracurricular activities. Implementation during an academic year elicited an increase in vegetable consumption, but did not favourably alter other targeted behaviours or BMI.[134,135] The Pathways programme for American-Indian children, who are at high risk for cardiovascular disease and type 2 diabetes,[136] was a large-scale cooperative effort among food-service personnel, classroom and physical education teachers, and families.[137] The aim of the programme was to reduce dietary fat consumption and augment physical activity. Preliminary reports state that the 3-year programme produced a significant decrease in fat consumption and a trend toward increased physical activity, but BMI did not differ between children in intervention and control schools at the end of the programme.[138–140] The CATCH (Child and Adolescent Trial for Cardiovascular Health) intervention, using an approach similar to Pathways although not specifically designed for obesity prevention, also aimed to reduce dietary fat consumption and increase physical activity. Once again, the intervention caused changes in the targeted behaviours, but BMI did not differ between intervention and control schools after 2 academic years.[141] Thus, with the exception of Planet Health, school-based interventions, involving multiple sites, have not reduced obesity prevalence, despite their intensive and, in some instances, multi-year designs.

Pharmacological and surgical treatments

Elucidation of the physiological basis of bodyweight regulation, and sequencing of the human genome provide the opportunity to develop new antiobesity agents. Each of a growing number of endogenous molecules known to affect bodyweight—eg, leptin, hypothalamic melanocortin 4 receptor, and mitochondrial uncoupling proteins—are potential targets for pharmacological manipulation. Enthusiasm for a pharmacological cure of obesity must be tempered, however, by three observations. First, most drugs used for treatment of obesity over the past century, from thyroid extract in the 1890s to Phen/fen in the 1990s,[142] have had potentially life-threatening complications. Second, because drugs do not produce permanent changes in physiology or behaviour they are effective only so long as they are taken, raising the spectre of life-long treatment. Third, the two agents most often used in the treatment of adult obesity, sibutramine and orlistat, produce modest weight loss, ranging from about 3–8% compared with placebo.[143] Four experimental drugs have produced weight loss in small-scale studies that involved children with special conditions—namely, metformin in obese adolescents with insulin resistance and hyperinsulinaemia,[144] octreotide for hypothalamic obesity,[145] growth hormone in children with Prader-Willi syndrome,[146] and leptin for congenital leptin deficiency.[147] When there is no inherent biological cause of obesity, pharmacological treatment should be prescribed only for children who have complications, and even then only after careful consideration of immediate and long-term risks and benefits and in the context of a comprehensive weight-management programme, as advocated by Yanovski.[148]

Scattered case reports, dating back several decades, have examined the use of bariatric surgery for the treatment of severe obesity in childhood.[148] With current techniques—generally, the roux-en-y gastric bypass—dramatic weight loss has been reported. However, serious complications of this procedure can result, including perioperative mortality, wound dehiscence, bowel obstruction, gastrointestinal bleeding, cholelithiasis, infection, and chronic nutritional deficiencies. As emphasised by Strauss and co-workers,[149] this approach constitutes, at best, a last resort for severely obese adolescents.

Limitations of current approaches

Although a few family-based studies produced significant long-term weight loss in motivated individuals, the overall success of non-surgical approaches has been disappointing, leading some specialists to conclude that treatment of obese children, which aims to establish a normal bodyweight, is unrealistically optimistic.[128] Why is substantial long-term weight loss so difficult to obtain? One explanation is that the dietary and physical activity prescriptions used in family-based and school-based programmes might not be particularly efficacious. Indeed,

For personal use. Only reproduce with permission from The Lancet Publishing Group.

most dietary interventions focus on reduction of fat intake, even though dietary fat might not be an important cause of obesity. Remarkably few paediatric obesity studies have sought to ascertain the effect of dietary composition on bodyweight, controlling for treatment intensity, physical activity, and behavioural modification techniques. With respect to physical activity, many studies have used conventional programmed exercise prescriptions, although increasing lifestyle activity or reducing sedentary behaviours might be better for long-term weight control.[83,131] A second explanation for the difficulty in obtaining long-term weight loss is that adverse environmental factors overwhelm behavioural and educational techniques designed to reduce energy intake and augment physical activity.

The toxic environment

6 years ago, Battle and Brownell wrote, "it is hard to envision an environment more effective than ours [in the USA] for producing . . . obesity".[150] This statement probably applies to much of the developed world and, increasingly, to some developing countries. Several pervasive environmental factors promote energy intake and limit energy expenditure in children, undermining individual efforts to maintain a healthy bodyweight.

Food quality, policy, and advertising

In the late 1970s, children in the USA ate 17% of their meals away from home, and fast foods accounted for 2% of total energy intake. By the mid-1990s to late-1990s, the proportion of meals eaten away from home nearly doubled to 30%, and fast food consumption increased five-fold, to 10% of total energy intake.[151] From 1965 to 1996, per capita daily soft drink consumption among 11–18-year old children rose from 179 g to 520 g for boys and from 148 g to 337 g for girls.[88] There are 170 000 fast food restaurants in the USA alone. These trends have been driven, in part, by enormous advertising and marketing expenditures by the food industry, including an estimated US$12·7 billion directed at children and their parents.[152] Marketing campaigns specifically target children, linking brand names with toys, games, movies, clothing, collectibles, educational tools, and even baby bottles.[152] By contrast, the advertising budget for the US National Cancer Institute's "5-A-Day" programme to promote consumption of fruits and vegetables was $1·1 million in 1999.[153] Large meals, often containing a child's total daily energy requirements, can be purchased for little additional cost over smaller portions, whereas fresh fruits and vegetables tend to be less readily available and comparatively more expensive.[154,155] Furthermore, fast-food and soft-drink vending machines pervade schools.[152] That US children overconsume added sugar and saturated fat, and underconsume fruits, non-starchy vegetables, fibre, and some micronutrients, is therefore not surprising.[88]

Sedentary lifestyle

Availability of sedentary pursuits, including television, video games, computers, and the internet, has risen greatly. Children in the USA spend 75% of their waking hours being inactive, compared with remarkably little time in vigorous physical activity; estimated at only 12 min per day.[156] Opportunities for physical activity have decreased for various reasons. Physical education, typically considered less important than academic disciplines, has been eliminated in some school districts. In schools that do offer physical education, large class size and lack of equipment present barriers to successful programme implementation.[157] After-school participation in unstructured activities can be limited, because of absence of pavements (sidewalks), bike paths, safe playgrounds, and parks in many neighbourhoods. Moreover, our culture places a premium on convenience: the car is preferred to walking, the lift to stairs, and the remote control to manual adjustment. These cultural forces arguably culminate in the drive-through window of fast-food restaurants, where a maximum of energy can be obtained with a minimum of exertion.

Barriers to change

Many special interests contribute to this problem of obesity, actively or passively, for financial reasons. As detailed by Nestle,[152] the food industry, which generated almost $1 trillion in sales in 2000, spends enormous amounts of money to promote consumption of high calorie processed foods of poor nutritional quality. Underfunded school districts make money by establishing pouring rights contracts with soft drink companies, allowing them to place vending machines on school property and to sell beverages at school events.[158] To save money, schools have subcontracted lunch programmes to corporate food services, encouraging the sale of high profit, low quality foods, including fast food.[152] At the same time, budgetary pressures have led to reduction or elimination of physical education classes. Many communities do not adequately invest in urban environments that encourage physical activity, and instead pursue policies that favour real estate development to open space. Parents, for various socioeconomic reasons, work excessively long hours, leaving little time to prepare home-cooked meals and supervise non-sedentary activities. Professional nutritional societies maintain lucrative relations through sponsorships and endorsement with the food industry, creating a potential conflict of interest.[159] According to the Center for Responsive Politics, candidates for the US congress and presidency received more than $12 million between 1989 and 2000 from the sugar industry (http://www.opensecrets.org/industries, accessed Jan 30, 2002). Might these political contributions have a corrosive effect on regulatory efforts to revise national nutritional policy?[160] Finally, the US health insurance industry reimburses poorly, if at all, for medical treatment of childhood obesity.[161] However, all these short-term financial incentives are trivial when compared with the long-term costs to individuals and society. Annual hospital costs alone related to paediatric obesity in the USA approximate $127 million,[162] and the effect of obesity on individuals is incalculable. Sadly, 10% of children with type 2 diabetes develop renal failure, requiring dialysis or resulting in death by young adulthood, according to a preliminary report.[163]

Conclusion

Almost three decades ago, an editorial in *The Lancet* called for efforts to prevent obesity in childhood.[164] Since then, the worldwide prevalence of childhood obesity has risen several-fold. Obese children develop serious medical and psychosocial complications, and are at greatly increased risk of adult morbidity and mortality. The increasing prevalence and severity of obesity in children, together with its most serious complication, type 2 diabetes, raise the spectre of myocardial infarction becoming a paediatric disease. This public-health crisis demands increased funding for research into new dietary, physical activity, behavioural, environmental, and pharmacological approaches for prevention and treatment of obesity, and improved

For personal use. Only reproduce with permission from The Lancet Publishing Group.

A common sense approach to prevention and treatment of childhood obesity

Home	Set aside time for Healthy meals Physical activity Limit television viewing
School	Fund mandatory physical education Establish stricter standards for school lunch programmmes Eliminate unhealthy foods—eg, soft drinks and candy from vending machines Provide healthy snacks through concession stands and vending machines
Urban design	Protect open spaces Build pavements (sidewalks), bike paths, parks, playgrounds, and pedestrian zones
Health care	Improve insurance coverage for effective obesity treatment
Marketing and media	Consider a tax on fast food and soft drinks Subsidise nutritious foods—eg, fruits and vegetables Require nutrition labels on fast-food packaging Prohibit food advertisement and marketing directed at children Increase funding for public-health campaigns for obesity prevention
Politics	Regulate political contributions from the food industry

reimbursement for effective family-based and school-based programmes. However, because this epidemic was not caused by inherent biological defects, increased funding for research and health care, focusing on new treatments, will probably not solve the problem of paediatric obesity without fundamental measures to effectively detoxify the environment (panel). Although these measures require substantial political will and financial investment, they should yield a rich dividend to society in the long term.

Contributors
All authors conceived and wrote the seminar.

Conflict of interest statement
None declared.

Acknowledgments
We thank Benjamin Brown, Steven Gortmaker, Gerald Hass, Robert Lustig, Joseph Majzoub, Marion Nestle, Norman Spack, and Joseph Wolfsdorf for their critical review of our Seminar. The authors were supported by grants from the National Institute of Diabetes and Digestive and Kidney Diseases (1R01DK059240, 5T32DK07699-18) and the Charles H Hood Foundation. The funding sources had no direct role in writing this seminar.

References

1 National Center for Health Statistics. Prevalence of overweight among children and adolescents: United States, 1999. http://www.cdc.gov/nchs/products/pubs/pubd/hestats/overwght99.htm (accessed Jan 29, 2002).
2 Chinn S, Rona RJ. Prevalence and trends in overweight and obesity in three cross-sectional studies of British children, 1974–94. *BMJ* 2001; **322:** 24–26.
3 Wang Y, Monteiro C, Popkin BM. Trends of obesity and underweight in older children and adolescents in the United States, Brazil, China, and Russia. *Am J Clin Nutr* 2002; **75:** 971–77.
4 Murata M. Secular trends in growth and changes in eating patterns of Japanese children. *Am J Clin Nutr* 2000; **72** (suppl)**:** 1379S–83S.
5 deOnis M, Blossner M. Prevalence and trends of overweight among preschool children in developing countries. *Am J Clin Nutr* 2000; **72:** 1032–39.
6 Filozof C, Gonzalez C, Sereday M, Mazza C, Braguinsky J. Obesity prevalence and trends in Latin-American countries. *Obes Rev* 2001; **2:** 99–106.
7 Magarey AM, Daniels LA, Boulton TJC. Prevalence of overweight and obesity in Australian children and adolescents: reassessment of 1985 and 1995 data against new standard international definitions. *Med J Aust* 2001; **174:** 561–64.
8 Flegal KM, Troiano RP. Changes in the distribution of body mass index of adults and children in the US population. *Int J Obesity* 2000; **24:** 807–18.
9 Bundred P, Kitchiner D, Buchan I. Prevalence of overweight and obese children between 1989 and 1998: population based series of cross-sectional studies. *BMJ* 2001; **322:** 1–4.
10 Strauss RS, Pollack HA. Epidemic increase in childhood overweight, 1986–1998. *JAMA* 2001; **286:** 2845–48.
11 James WPT, Nelson M, Ralph A, Leather S. Socioeconomic determinants of health: the contribution of nutrition to inequalities in health. *BMJ* 1997; **314:** 1545–49.
12 Gordon-Larsen P, McMurray RG, Popkin BM. Determinants of adolescent physical activity and inactivity patterns. *Pediatrics* 2000; **105:** e83.
13 Martorell R, Khan LK, Hughes ML, Grummer-Strawn LM. Obesity in Latin American women and children. *J Nutr* 1998; **128:** 1464–73.
14 Doak C, Adair L, Bentley M, Fengying Z, Popkin B. The underweight/overweight household: an exploration of household sociodemographic and dietary factors in China. *Public Health Nutr* 2002; **5:** 215–21.
15 Popkin BM. An overview on the nutrition transition and its health implications: the Bellagio meeting. *Public Health Nutr* 2002; **5** (suppl)**:** 93–103.
16 Must A, Strauss RS. Risks and consequences of childhood and adolescent obesity. *Int J Obesity* 1999; **23** (suppl)**:** S2–11.
17 Davison KK, Birch LL. Weight status, parent reaction, and self-concept in five-year-old girls. *Pediatrics* 2001; **107:** 46–53.
18 Strauss RS. Childhood obesity and self-esteem. *Pediatrics* 2000; **105:** e15.
19 Erickson SJ, Robinson TN, Haydel KF, Killen JD. Are overweight children unhappy? Body mass index, depressive symptoms, and overweight concerns in elementary school children. *Arch Pediatr Adolesc Med* 2000; **154:** 931–35.
20 Field AE, Camargo CA, Taylor CB, et al. Overweight, weight concerns, and bulimic behaviors among girls and boys. *J Am Acad Child Adolesc Psychiatry* 1999; **38:** 754–60.
21 Balcer LJ, Liu GT, Forman S, et al. Idiopathic intracranial hypertension: relation of age and obesity in children. *Neurology* 1999; **52:** 870–72.
22 Redline S, Tishler PV, Schluchter M, Aylor J, Clark K, Graham G. Risk factors for sleep-disordered breathing in children: associations with obesity, race, and respiratory problems. *Am J Respir Crit Care Med* 1999; **159:** 1527–32.
23 Figueroa-Munoz JI, Chinn S, Rona RJ. Association between obesity and asthma in 4–11 year old children in the UK. *Thorax* 2001; **56:** 133–37.
24 Reybrouck T, Mertens L, Schepers D, Vinckx J, Gewilling M. Assessment of cardiovascular exercise function in obese children and adolescents by body mass-independent parameters. *Eur J Appl Physiol* 1997; **75:** 478–83.
25 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.
26 Ford ES, Galuska DA, Gillespie C, Will JC, Giles WH, Dietz WH. C-reactive protein and body mass index in children: findings from the Third National Health and Nutrition Examination Survey, 1988–1994. *J Pediatr* 2001; **138:** 486–92.
27 Ferguson MA, Gutin B, Owens S, Litaker M, Tracy RP, Allison J. Fat distribution and hemostatic measures in obese children. *Am J Clin Nutr* 1998; **67:** 1136–40.
28 Tounian P, Aggoun Y, Dubern B, et al. Presence of increased stiffness of the common carotid artery and endothelial dysfunction in severely obese children: a prospective study. *Lancet* 2001; **358:** 1400–04.
29 Srinivasan SR, Myers L, Berenson GS. Predictability of childhood adiposity and insulin for developing insulin resistance syndrome (syndrome X) in young adulthood: the Bogalusa Heart Study. *Diabetes* 2002; **51:** 204–09.
30 Strauss RS, Barlow SE, Dietz WH. Prevalence of abnormal serum aminotransferase values in overweight and obese adolescents. *J Pediatr* 2000; **136:** 727–33.

For personal use. Only reproduce with permission from The Lancet Publishing Group.

31 Friesen CA, Roberts CC. Cholelithiasis: clinical characteristics in children—case analysis and literature review. *Clin Pediatr* 1989; **28:** 294–98.

32 Ludwig DS, Ebbeling CB. Type 2 diabetes mellitus in children: primary care and public health considerations. *JAMA* 2001; **286:** 1427–30.

33 Kaplowitz PB, Slora EJ, Wasserman RC, Pedlow SE, Herman-Giddens ME. Earlier onset of puberty in girls: relation to increased body mass index and race. *Pediatrics* 2001; **108:** 347–53.

34 Lewy VD, Danadian K, Witchel SF, Arslanian S. Early metabolic abnormalities in adolescent girls with polycystic ovary syndrome. *J Pediatr* 2001; **138:** 38–44.

35 Castro-Magana M. Hypogonadism and obesity. *Pediatr Ann* 1984; **13:** 494–97.

36 Adelman RD, Restaino IR, Alon US, Blowey DL. Proteinuria and focal segmental glomerulosclerosis in severely obese adolescents. *J Pediatr* 2001; **138:** 481–85.

37 Goulding A, Jones IE, Taylor RW, Williams SM, Manning PJ. Bone mineral density and body composition in boys with distal forearm fractures: a dual-energy X-ray absorptiometry study. *J Pediatr* 2001; **139:** 509–15.

38 Dowling AM, Steele JR, Baur LA. Does obesity influence foot structure and plantar pressure patterns in prepubescent children? *Int J Obesity* 2001; **25:** 845–52.

39 Loder RT, Aronson DD, Greenfield ML. The epidemiology of bilateral slipped capital femoral epiphysis. *J Bone Joint Surg* 1993; **75:** 1141–47.

40 Dietz WH, Gross WL, Kirkpatrick JA. Blount disease (tibia vara): another skeletal disorder associated with childhood obesity. *J Pediatr* 1982; **101:** 735–37.

41 Young-Hyman D, Schlundt DG, Herman L, DeLuca F, Counts D. Evaluation of the insulin resistance syndrome in 5- to 10-year-old overweight/obese African-American children. *Diabetes Care* 2001; **24:** 1359–64.

42 Strong JP, Malcom GT, McMahan CA, et al. Prevalence and extent of atherosclerosis in adolescents and young adults: implications for prevention from the Pathobiological Determinants of Atherosclerosis in Youth Study. *JAMA* 1999; **281:** 727–35.

43 McGill HC, McMahan A, Zieske AW, et al. Association of coronary heart disease risk factors with microscopic qualities of coronary atherosclerosis in youth. *Circulation* 2000; **102:** 374–79.

44 Gunnell DJ, Frankel SJ, Nanchahal K, Peters TJ, Smith GD. Childhood obesity and adult cardiovascular mortality: a 57-y follow-up study based on the Boyd Orr cohort. *Am J Clin Nutr* 1998; **67:** 1111–18.

45 Fagot-Campagna A, Pettitt DJ, Engelgau MM, et al. Type 2 diabetes among North American children and adolescents: an epidemiologic review and a public health perspective. *J Pediatr* 2000; **136:** 664–72.

46 Sinha R, Fisch G, Teague B, et al. Prevalence of impaired glucose tolerance among children and adolescents with marked obesity. *N Engl J Med* 2002; **346:** 802–10.

47 Hill AJ, Silver EK. Fat, friendless and unhealthy: 9-year old children's perception of body shape stereotypes. *Int J Obesity* 1995; **19:** 423–30.

48 Teachman BA, Brownell KD. Implicit anti-fat bias among health professionals: is anyone immune? *Int J Obesity* 2001; **25:** 1525–31.

49 Winkleby MA, Robinson TN, Sundquist J, Kraemer H. Ethnic variation in cardiovascular disease risk factors among children and young adults: findings from the Third National Health and Nutrition Examination Survey, 1988–1994. *JAMA* 1999; **281:** 1006–13.

50 Gower BA, Nagy TR, Trowbridge CA, Dezenberg C, Goran MI. Fat distribution and insulin response in prepubertal African American and white children. *Am J Clin Nutr* 1998; **67:** 821–27.

51 Reaven P, Nader PR, Berry C, Hoy T. Cardiovascular disease insulin risk in Mexican-American and Anglo-American children and mothers. *Pediatrics* 1998; **101:** e12.

52 Kimm SY, Barton BA, Berhane K, Ross JW, Payne GH, Schreiber GB. Self-esteem and adiposity in black and white girls: the NHLBI Growth and Health Study. *Ann Epidemiol* 1997; **7:** 550–60.

53 Lustig RH. The neuroendocrinology of childhood obesity. *Pediatr Clin North Am* 2001; **48:** 909–30.

54 Montague CT, Farooqi IS, Whitehead JP, et al. Congenital leptin deficiency is associated with severe early-onset obesity in humans. *Nature* 1997; **387:** 903–08.

55 Zhang Y, Proenca R, Maffei M, Barone M, Leopold L, Friedman JM. Positional cloning of the mouse *obese* gene and its human homologue. *Nature* 1994; **372:** 425–32.

56 Farooqi IS, O'Rahilly S. Recent advances in the genetics of severe obesity. *Arch Dis Child* 2000; **83:** 31–34.

57 LeStunff C, Fallin D, Bougneres P. Paternal transmission of the very common class I *INS* VNTR alleles predisposes to childhood obesity. *Nat Genet* 2001; **29:** 96–99.

58 Rankinen T, Perusse L, Weisnagel SJ, Snyder EE, Chagnon YC, Bouchard C. The human obesity gene map: the 2001 update. *Obes Res* 2002; **10:** 196–243.

59 Whitaker RC, Dietz WH. Role of the prenatal environment in the development of obesity. *J Pediatr* 1998; **132:** 768–76.

60 Levin BE, Govek E. Gestational obesity accentuates obesity in obesity-prone progeny. *Am J Physiol* 1998; **275:** R1374–79.

61 Ravelli GP, Stein ZA, Susser MW. Obesity in young men after famine exposure in utero and early infancy. *N Engl J Med* 1976; **295:** 349–53.

62 Popkin BM. The nutrition transition and its health implications in lower income countries. *Public Health Nutr* 1998; **1:** 5–21.

63 Gillman MW, Rifas-Shiman SL, Camargo CA, et al. Risk of overweight among adolescents who were breastfed as infants. *JAMA* 2001; **285:** 2461–67.

64 vonKries R, Koletzko B, Sauerwald T, et al. Breast feeding and obesity: cross sectional study. *BMJ* 1999; **319:** 147–50.

65 Rolland-Cachera M-F, Deheeger M, Bellisle F, Sempe M, Guillound-Bataille M, Patois E. Adiposity rebound in children: a simple indicator for predicting obesity. *Am J Clin Nutr* 1984; **39:** 129–35.

66 Whitaker RC, Pepe MS, Wright JA, Seidel KD, Dietz WH. Early adiposity rebound and the risk of adult obesity. *Pediatrics* 1998; **101:** e5.

67 Wisemandle W, Maynard M, Guo SS, Siervogel RM. Childhood weight, stature, and body mass index among never overweight, early-onset overweight, and late-onset overweight groups. *Pediatrics* 2000; **106:** e14.

68 Freedman DS, Kettel Khan L, Serdula MK, Srinivasan SR, Berenson GS. BMI rebound, childhood height and obesity among adults: the Bogalusa Heart Study. *Int J Obes* 2001; **25:** 543–49.

69 Dietz WH. "Adiposity rebound": reality or epiphenomenon? *Lancet* 2000; **356:** 2027–28.

70 Trost SG, Kerr LM, Ward DS, Pate RR. Physical activity and determinants of physical activity in obese and non-obese children. *Int J Obes Relat Metab Disord* 2001; **25:** 822–29.

71 Andersen RE, Crespo CJ, Bartlett SJ, Cheskin LJ, Pratt M. Relationship of physical activity and television watching with body weight and level of fatness among children: results from the Third National Health and Nutrition Examination Survey. *JAMA* 1998; **279:** 938–42.

72 Hernandez B, Gortmaker SL, Colditz GA, Peterson KE, Laird NM, Para-Cabrera S. Association of obesity with physical activity, television programs and other forms of video viewing among children in Mexico City. *Int J Obesity* 1999; **23:** 845–54.

73 Berkey CS, Rockett HRH, Field AE, et al. Activity, dietary intake, and weight changes in a longitudinal study of preadolescent and adolescent boys and girls. *Pediatrics* 2000; **105:** e56.

74 Johnson MS, Figueroa-Colon R, Herd SL, et al. Aerobic fitness, not energy expenditure, influences subsequent increase in adiposity in black and white children. *Pediatrics* 2000; **106:** e50.

75 Goran MI, Reynolds KD, Lindquist CH. Role of physical activity in the prevention of obesity in children. *Int J Obesity* 1999; **23** (suppl)**:** S18–33.

76 Robinson TN. Does television cause childhood obesity? *JAMA* 1998; **279:** 959–60.

77 Epstein LH, Paluch RA, Consalvi A, Riordan K, Scholl T. Effects of manipulating sedentary behavior on physical activity and food intake. *J Pediatr* 2002; **140:** 334–39.

78 Kotz K, Story M. Food advertisements during children's Saturday morning television programming: are they consistent with dietary recommendations? *J Am Diet Assoc* 1994; **94:** 1296–300.

79 Lewis MK, Hill AJ. Food advertising on British children's television: a content analysis and experimental study with nine-year olds. *Int J Obesity* 1998; **22:** 206–14.

80 Taras HL, Gage M. Advertised foods on children's television. *Arch Pediatr Adolesc Med* 1995; **149:** 649–52.

81 Borzekowski DLG, Robinson TN. The 30-second effect: an experiment revealing the impact of television commercials on food preferences of preschoolers. *J Am Diet Assoc* 2001; **101:** 42–46.

82 Coon KA, Goldberg J, Rogers BL, Tucker KL. Relationships between use of television during meals and children's food consumption patterns. *Pediatrics* 2001; **107:** e7.

83 Robinson TN. Reducing children's television viewing to prevent obesity: a randomized controlled trial. *JAMA* 1999; **282:** 1561–67.

84 Jequier E. Is fat intake a risk factor for fat gain in children? *J Clin Endocrinol Metabol* 2001; **86:** 980–83.

For personal use. Only reproduce with permission from The Lancet Publishing Group.

85 Atkin L-M, Davies PSW. Diet composition and body composition in preschool children. *Am J Clin Nutr* 2000; **72:** 15–21.
86 Ludwig DS, Pereira MA, Kroenke CH, et al. Dietary fiber, weight gain and cardiovascular disease risk factors in young adults: the CARDIA Study. *JAMA* 1999; **282:** 1539–46.
87 Troiano RP, Briefel RR, Carroll MD, Bialostosky K. Energy and fat intakes of children and adolescents in the United States: data from the National Health and Nutrition Examination Surveys. *Am J Clin Nutr* 2000; **72** (suppl)**:** 1343S–53S.
88 Cavadini C, Siega-Riz AM, Popkin BM. US adolescent food intake trends from 1965 to 1996. *Arch Dis Child* 2000; **83:** 18–24.
89 Kris-Etherton P, Daniels SR, Eckel RH, et al. AHA scientific statement: summary of the Scientific Conference on Dietary Fatty Acids and Cardiovascular Health. *J Nutr* 2001; **131:** 1322–26.
90 Litin L, Sacks F. Trans-fatty-acid content of common foods. *N Engl J Med* 1993; **329:** 1969–70.
91 Hu FB, Stampfer MJ, Manson JE, et al. Dietary fat intake and the risk of coronary heart disease in women. *N Engl J Med* 1997; **337:** 1491–99.
92 Salmeron J, Hu FB, Manson JE, et al. Dietary fat intake and risk of type 2 diabetes in women. *Am J Clin Nutr* 2001; **73:** 1019–26.
93 Subar AF, Krebs-Smith SM, Cook A, Kahle LL. Dietary sources of nutrients among US children, 1989–1991. *Pediatrics* 1998; **102:** 913–23.
94 Foster-Powell K, Brand Miller J. International tables of glycemic index. *Am J Clin Nutr* 1995; **62** (suppl)**:** 871S–90S.
95 Ebbeling CB, Ludwig LS. Treating obesity in youth: should dietary glycemic load be a consideration? *Adv Pediatr* 2001; **48:** 179–212.
96 Ludwig DS. The glycemic index: physiological mechanisms relating to obesity, diabetes, and cardiovascular disease. *JAMA* 2002; **287:** 2414–23.
97 Ludwig DS, Majzoub JA, Al-Zahrani A, Dallal GE, Blanco I, Roberts SB. High glycemic index foods, overeating, and obesity. *Pediatrics* 1999; **103:** e26.
98 Toeller M, Buyken AE, Heitkamp G, Cathelineau G, Ferriss B, Michel G. Nutrient intakes as predictors of body weight in European people with type 1 diabetes. *Int J Obes* 2001; **25:** 1815–22.
99 Liu S, Willett WC, Stampfer MJ, et al. A prospective study of dietary glycemic load, carbohydrate intake, and risk of coronary heart disease in women. *Am J Clin Nutr* 2000; **71:** 1455–61.
100 Salmeron J, Manson JE, Stampfer MJ, Colditz GA, Wing AL, Willett WC. Dietary fiber, glycemic load, and risk of non-insulin-dependent diabetes mellitus in women. *JAMA* 1997; **277:** 472–77.
101 Morton JF, Guthrie JF. Changes in children's total fat intakes and their food group sources of fat, 1989–91 versus 1994–95: implications for diet quality. *Fam Econ Nutr Rev* 1998; **11:** 44–57.
102 Harnack L, Stang J, Story M. Soft drink consumption among US children and adolescents: nutritional consequences. *J Am Diet Assoc* 1999; **99:** 436–41.
103 Ludwig DS, Peterson KE, Gortmaker SL. Relation between consumption of sugar-sweetened drinks and childhood obesity: a prospective, observational analysis. *Lancet* 2001; **357:** 505–08.
104 Ludwig DS, Peterson KE, Gortmaker SL. Causes of obesity. *Lancet* 2001; **357:** 1978–79.
105 Pereira MA, Jacobs DR, VanHorn L, Slattery ML, Kartashov AI, Ludwig DS. Dairy consumption, obesity, and the insulin resistance syndrome in young adults: the CARDIA study. *JAMA* 2002; **287:** 2081–89.
106 Rolls BJ. The role of energy density in the overconsumption of fat. *J Nutr* 2000; **130** (suppl)**:** 268S–71S.
107 Bell EA, Rolls BJ. Energy density of foods affects energy intake across multiple levels of fat content in lean and obese women. *Am J Clin Nutr* 2001; **73:** 1010–18.
108 Gibson SA. Associations between energy density and macronutrient composition in the diets of pre-school children: sugars *vs* starch. *Int J Obesity* 2000; **24:** 633–38.
109 Rolls BJ, Engell D, Birch LL. Serving portion size influences 5-year-old but not 3-year-old children's food intake. *J Am Diet Assoc* 2000; **100:** 232–34.
110 Hu FB, vanDam RM, Liu S. Diet and risk of type II diabetes: the role of types of fat and carbohydrate. *Diabetologia* 2001; **44:** 805–17.
111 Slavin JL, Martini MC, Jacobs DR, Marquart L. Plausible mechanisms for the protectiveness of whole grains. *Am J Clin Nutr* 1999; **70** (suppl)**:** 459S–63S.
112 French SA, Story M, Neumark-Sztainer D, Fulkerson JA, Hannan P. Fast food restaurant use among adolescents: associations with nutrient intake, food choices and behavioral and psychosocial variables. *Int J Obesity* 2001; **25:** 1823–33.
113 French SA, Harnack L, Jeffery RW. Fast food restaurant use among women in the Pound of Prevention study: dietary, behavioral and demographic correlates. *Int J Obesity* 2000; **24:** 1353–59.
114 Binkley JK, Eales J, Jekanowski M. The relation between dietary change and rising US obesity. *Int J Obesity* 2000; **24:** 1032–39.
115 McNutt SW, Hu Y, Schreiber GB, Crawford PB, Obarzanek E, Mellin L. A longitudinal study of the dietary practices of black and white girls 9 and 10 years old at enrollment: the NHLBI Growth and Health Study. *J Adolesc Health* 1997; **20:** 27–37.
116 Zoumas-Morse C, Rock CL, Sobo EJ, Neuhouser ML. Children's patterns of macronutrient intake and associations with restaurant and home eating. *J Am Diet Assoc* 2001; **101:** 923–25.
117 Wiecha JL, Sobol AM, Peterson KE, Gortmaker SL. Household television access: associations with screen time, reading, and homework among youth. *Ambulatory Pediatr* 2001; **1:** 244–51.
118 Gillman MW, Rifas-Shiman SL, Frazier AL, et al. Family dinner and diet quality among older children and adolescents. *Arch Fam Med* 2000; **9:** 235–40.
119 Sallis JF, Prochaska JJ, Taylor WC. A review of correlates of physical activity of children and adolescents. *Med Sci Sports Exerc* 2000; **32:** 963–75.
120 Lissau I, Sorensen TIA. Parental neglect during childhood and increased risk of obesity in young adulthood. *Lancet* 1994; **343:** 324–27.
121 Pine DS, Goldstein RB, Wolk S, Weissman MM. The association between childhood depression and adult body mass index. *Pediatrics* 2001; **107:** 1049–56.
122 Mellbin T, Vuille J-C. Further evidence of an association between psychosocial problems and increase in relative weight between 7 and 10 years of age. *Acta Paediatr Scand* 1989; **78:** 576–80.
123 Johnson JG, Cohen P, Kasen S, Brook JS. Childhood adversities associated with risk for eating disorders or weight problems during adolescence or early adulthood. *Am J Psychiatry* 2002; **159:** 394–400.
124 Anon. Methods for voluntary weight loss and control: NIH technology assessment conference panel. *Ann Intern Med* 1993; **119:** 764–70.
125 Barlow SE, Dietz WH. Obesity evaluation and treatment: expert committee recommendations. *Pediatrics* 1998; **102:** e29.
126 Epstein LH, Myers MD, Raynor HA, Saelens BE. Treatment of pediatric obesity. *Pediatrics* 1998; **101:** 554–70.
127 Flodmark C-E, Ohlsson T, Ryden O, Sveger T. Prevention and progression to severe obesity in a group of obese schoolchildren treated with family therapy. *Pediatrics* 1993; **91:** 880–84.
128 Israel AC, Guile CA, Baker JE, Silverman WK. An evaluation of enhanced self-regulation training in the treatment of childhood obesity. *J Pediatr Psychol* 1994; **19:** 737–49.
129 Braet C, VanWinckel M. Long-term follow-up of a cognitive behavioral treatment program for obese children. *Behav Ther* 2000; **31:** 55–74.
130 Epstein LH, Valoski A, Wing RR, McCurley J. Ten-year follow-up of behavioral, family-based treatment for obese children. *JAMA* 1990; **264:** 2519–23.
131 Epstein LH, Valoski AM, Wing RR, McCurley J. Ten year outcomes of behavioral family-based treatment for childhood obesity. *Health Psychol* 1994; **13:** 373–83.
132 Epstein LH, Roemmich JN, Raynor HA. Behavioral therapy in the treatment of pediatric obesity. *Pediatr Clin North Am* 2001; **48:** 981–93.
133 Gortmaker SL, Peterson K, Wiecha J, et al. Reducing obesity via a school-based interdisciplinary intervention among youth. *Arch Pediatr Adolesc Med* 1999; **153:** 409–18.
134 Sahota P, Rudolf MCJ, Dixey R, Hill AJ, Barth JH, Cade J. Evaluation of implementation and effect of primary school based intervention to reduce risk factors for obesity. *BMJ* 2001; **323:** 1027–29.
135 Sahota P, Rudolf MCJ, Dixey R, Hill AJ, Barth JH, Cade J. Randomised controlled trial of primary school based intervention to reduce risk factors for obesity. *BMJ* 2001; **323:** 1029–32.
136 Story M, Evans M, Fabsitz RR, Clay TE, Rock BH, Broussard B. The epidemic of obesity in American Indian communities and the need for childhood obesity-prevention programs. *Am J Clin Nutr* 1999; **69** (suppl)**:** 747S–4S.
137 Gittelsohn J, Evans M, Story M, et al. Multisite formative assessment for the Pathways study to prevent obesity in American Indian schoolchildren. *Am J Clin Nutr* 1999; **69** (suppl)**:** 767S–72S.
138 Lohman TG, Going S, Stewart D, et al. The effect of Pathways obesity prevention study on body composition in American children. *FASEB J* 2001; **15:** A1093 (abstr).

For personal use. Only reproduce with permission from The Lancet Publishing Group.

139 Himes JH, Cunningham-Sabo L, Gittelsohn J, et al. Impact of the Pathways intervention on dietary intake of American Indian school children. *FASEB J* 2001; **15:** A1092 (abstr).
140 Going SB, Stone E, Harnack L, et al. The effects of the Pathways obesity prevention program on physical activity in American Indian children. *FASEB J* 2001; **15:** A1092 (abstr).
141 Luepker RV, Perry CL, McKinlay SM, et al. Outcomes of a field trial to improve children's dietary patterns and physical activity: the Child and Adolescent Trial for Cardiovascular Health (CATCH). *JAMA* 1996; **275:** 768–76.
142 Connolly HM, Crary JL, McGoon MD, et al. Valvular heart disease associated with fenfluramine-phentermine. *N Engl J Med* 1997; **337:** 581–88.
143 Glazer G. Long-term pharmacotherapy of obesity 2000: a review of the efficacy and safety. *Arch Intern Med* 2001; **161:** 1814–24.
144 Freemark M, Bursey D. The effects of Metformin on body mass index and glucose tolerance in obese adolescents with fasting hyperinsulinemia and a family history of type 2 diabetes. *Pediatrics* 2001; **107:** e55.
145 Lustig RH, Rose SR, Burghen GA, et al. Hypothalamic obesity caused by cranial insult in children: altered glucose and insulin dynamics and reversal by somatostatin agonist. *J Pediatr* 1999; **135:** 162–68.
146 Myers SE, Carrel AL, Whitman BY, Allen DB. Sustained benefit after 2 years of growth hormone on body composition, fat utilization, physical strength and agility, and growth in Prader-Willi syndrome. *J Pediatr* 2000; **137:** 42–49.
147 Farooqi IS, Jebb SA, Langmack G, et al. Effects of recombinant leptin therapy in a child with congenital leptin deficiency. *N Engl J Med* 1999; **341:** 879–84.
148 Yanovski JA. Intensive therapies for pediatric obesity. *Pediatr Clin North Am* 2001; **48:** 1041–53.
149 Strauss RS, Bradley LJ, Brolin RE. Gastric bypass surgery in adolescents with morbid obesity. *J Pediatr* 2001; **138:** 499–504.
150 Battle EK, Brownell KD. Confronting a rising tide of eating disorders and obesity: treatment vs prevention and policy. *Addict Behav* 1996; **21:** 755–65.
151 Lin B-H, Guthrie J, Frazao E. American children's diets not making the grade. *Food Rev* 2001; **24:** 8–17.
152 Nestle M. Food politics: how the food industry influences nutrition and health. Berkeley: University of California Press, 2002.
153 National Institutes of Health. National Cancer Institute. 5 A Day for Better Health (monograph), 2001. http://www.5aday.gov/pdf/masimaxmonograph.pdf (accessed July 12, 2002).
154 French SA, Story M, Jeffery RW. Environmental influences on eating and physical activity. *Ann Rev Public Health* 2001; **22:** 309–35.
155 Hill JO, Peters JC. Environmental contributors to the obesity epidemic. *Science* 1998; **280:** 1371–74.
156 Strauss RS, Rodzilsky D, Burack G, Colin M. Psychosocial correlates of physical activity in healthy children. *Arch Pediatr Adolesc Med* 2001; **155:** 897–902.
157 Anon. A report to the president from the secretary of health and human services and the secretary of education: promoting better health for young people through physical activity and sports. Silver Spring: CDC at Healthy Youth, 2000. http://www.cdc.gov/nccdphp/dash/presphysactrpt (accessed July 12, 2002)
158 Nestle MS. Soft drink "pouring rights": marketing empty calories to children. *Public Health Rep* 2000; **115:** 308–19.
159 Nestle M. Food company sponsorship of nutrition research and professional activities: a conflict of interest? *Public Health Nutr* 2001; **4:** 1015–22.
160 Loewenberg S. Big sugar is bitter about diet advice. *Legal Times*, April 3, 2000.
161 Tershakovec AM, Watson MH, Wenner WJ, Marx AL. Insurance reimbursement for the treatment of obesity in children. *J Pediatr* 1999; **134:** 573–78.
162 Wang G, Dietz WH. Economic burden of obesity in youths aged 6 to 17 years: 1979–1999. *Pediatrics* 2002; **109:** e81.
163 Dean H, Flett B. Natural history of type 2 diabetes diagnosed in childhood: long term follow-up in young adult years. *Diabetes* 2002; **51:** A24 (abstr).
164 Anon. Infant and adult obesity. *Lancet* 1974; **1:** 17–18.

For personal use. Only reproduce with permission from The Lancet Publishing Group.