An ILSI Center for Health Promotion Monograph

CHILDHOOD OBESITY

PARTNERSHIPS for RESEARCH and PREVENTION

Frederick L. Trowbridge

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Childhood Obesity: Partnerships for Research and Prevention

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FOREWORD

Surveys conducted in the United States and other countries over the last several decades have documented an alarming increase in the prevalence of obesity, not only in adults but also in children and adolescents. This obesity "epidemic" has significant implications for both current and future health risks, including especially increased risk for diabetes, cardiovascular disease and other chronic diseases. One might think that the causes of such a substantial increase in obesity would be easy to identify. But, in fact, the causes are complex and are linked to a variety of social, economic, and lifestyle changes that have combined to upset the balance of energy intake and expenditure.

The complexity of the causes of increasing obesity also presents a variety of challenges in addressing the problem. There is a lack of proven intervention strategies, especially considering the tendency of individuals to regain weight even after successful initial weight loss. And, although the level of awareness of child obesity as a health concern has been increasing, there is still insufficient attention and priority given to addressing this issue.

Recognizing the importance of child obesity, the International Life Sciences Institute Center for Health Promotion sponsored a conference in May, 1999 to address this issue. Co-sponsors included the Emory University School of Medicine, the Georgia Health Foundation, the Centers for Disease Control and Prevention, the National Institutes of Health and the American Cancer Society. The program included plenary and concurrent sessions addressing key issues in obesity assessment, prevention and treatment in healthcare, school and community settings. Concluding sessions considered the future directions and research needed to better address the challenges presented by the continuing increase in obesity among children and adolescents.

This monograph presents four key papers from the conference, addressing a variety of topics relating to obesity assessment, prevention, treatment and research needs. It is hoped that these papers will contribute to an increased awareness of the importance of child obesity as an international health issue and to an improved perception of the actions needed to better address this critical issue.

I. Child Obesity: Challenges and Opportunities for Prevention

James O. Hill, PhD

The papers herein by Goran,[1] Resnicow,[2] and Dietz[3] highlight critical issues relating to the growing problem of child obesity. Goran[1] reviews the linkages between childhood overweight and the risk of obesity in adulthood and provides an overview of the adverse health implications of obesity in both children and adults. He then examines the etiologic factors that appear to underlie the increasing prevalence of obesity in the United States and other countries. Given the difficulty in treating obesity once it is established, he presents a convincing rationale for a preventive approach.

The results of research relating to interventions in schools, communities, and health care settings and through environmental and policy changes have been summarized by Resnicow.[2] He notes that interventions in schools have shown inconsistent results, with only some interventions showing success in modifying diet and physical activity behaviors. Changes in physiologic endpoints such as body mass index (BMI) have proven to be even more difficult to achieve, although some promising results have been reported. Resnicow observes that the research base for community interventions for child obesity is quite thin, although initial results from studies that are currently under way suggest that programmed physical activity training may be both feasible and effective in treating obese children and adolescents. Studies in clinical settings suggest that interventions to decrease sedentary behavior may be more effective than encouraging increased physical activity. Also encouraging is the observation that children appear to maintain weight loss better than adults. Still unresolved, however, is the effectiveness of involving parents in the treatment of overweight youth. Finally, Resnicow points to a number of environ-

mental and policy changes that might be considered, such as increasing the availability of safe and accessible play areas.

Dietz[3] provides an overview of key issues relating to prevention efforts at the individual, environmental, and policy levels. He reviews key points in the development of obesity, in particular the point of "adiposity rebound" at about 6 years of age, when BMI reaches its nadir and begins to increase, and during adolescence. Dietz also assesses likely causal factors that may underlie the substantial increase in obesity prevalence observed in recent years. He then reviews potential strategies for intervention in health care, school, and community settings.

A review of these papers makes clear that efforts are under way in a variety of settings to address child obesity. But it is even clearer that the effectiveness of current intervention efforts is limited and that much more will need to be done if the trend toward increasing obesity in children is to be reversed. The question is how to proceed to identify and implement more effective efforts.

Challenges to Effective Intervention

In efforts to address the problem of child obesity effectively, three key objectives emerge:

1. to set an agenda for research to define what works and what does not,
2. to identify and demonstrate ways to improve intervention efforts within health care, school, and community settings, and
3. to raise awareness of child obesity issues in order to stimulate a higher level of support and priority at the local, state, and national levels.

In working towards these objectives, however, it becomes clear that a number of challenges must be addressed. One key challenge to effective action is the sheer complexity of the child obesity problem. In order to address the problem effectively, it is critical to understand its causes and contributing factors. At a superficial level, obesity is readily explained as an imbalance involving too much energy intake and/or too little energy expenditure. However, the causes that underlie this imbalance are complex and reflect the influence of multiple social, eco-

nomic, and lifestyle factors. This reality complicates both the setting of a focused research agenda and the implementation of specific interventions.

Another related challenge is the lack of a unified strategy involving—and, it is hoped, integrating—interventions in health care, school, and community settings as well as policy and environmental changes. Currently, efforts are often isolated and uncoordinated. Individuals concerned about child obesity in health care, school, and community settings may not even be aware of the efforts of other potential partners.

Finally, a pervasive challenge is the low level of awareness of the child obesity problem and the resulting low priority given to the problem. Because of a low level of awareness, it has proven difficult to secure the resources needed to address treatment and prevention efforts effectively. These challenges and strategies to address them are considered below.

Complexity of the Problem

The increasing prevalence of obesity may be explained, at a superficial level, as the result of some combination of increasing access to an energy-dense diet and declining energy expenditure related to an increasingly sedentary lifestyle. However, the actual extent of changes in energy intake and expenditure, and the underlying social, economic, and cultural factors responsible for such changes, are complex and difficult to quantify. This complexity presents a major challenge because it makes it difficult—in fact, inaccurate—to identify a single cause that can be remedied to fix the problem. Nevertheless, in attempts to define clear strategies for intervention, it is tempting to assign blame to some particular segment or sector of society as the cause of obesity, with the implication that changes in that sector would correct the obesity problem.

One target that has been blamed for the rise in obesity is the food industry. It is cited for marketing high-calorie, energy-dense foods in large portion sizes and for focusing advertising efforts toward children. Children's access to these food choices has increased rapidly with the proliferation of fast-food restaurants and the increased presence in schools of vending machines that stock high-fat, high-sugar snack foods. These are certainly important issues and seem likely to play a role in the

obesity problem, but food intake is only part of the equation that produces obesity.

Physical inactivity is certainly also a contributor to obesity in children. Just as the food industry and its advertising to children has been singled out as a contributor to obesity, is it not logical to single out the computer industry, the manufacturers of video games, or the explosive growth of the Internet as contributors to inactivity and sedentary lifestyles? Automobile manufacturers might also be blamed for discouraging physical activity and for inhibiting walking and cycling as forms of transportation—or is this the fault of city and community planners? The television industry might also be cited. Studies have demonstrated the contribution of excessive television viewing to inactivity and its link with obesity.

Other observers may blame parents for the rise in child obesity. Parents may be criticized for failing to teach their children healthy eating habits and for their failure to set better examples in their own diet and physical activity behaviors. Schools may also be criticized for their lack of attention to these issues. Promising strategies and programs to encourage better nutrition and physical activity in schools have been developed, but many superintendents, principals, and teachers seem uninterested in implementing them or, perhaps, are too overwhelmed with other priorities. Managed care systems and other health care providers might also be cited for giving too little attention to obesity as a health issue in their patients, whether related to a lack of time or to the lack of reimbursement for prevention or treatment services for obesity.

Although it is possible to cite many sectors of society that may be contributing to, or failing to address, the problem of child obesity, this is unlikely to be a useful process. Rather, the multiple sectors and complex factors affecting child obesity indicate that this problem is not the result of a few isolated influences, but rather is best understood as an unintentional consequence of broad social, economic, and lifestyle changes. If “blame” is to be placed for causing the increase in child obesity, it rests broadly across many segments of our society. In a real sense, we are all responsible, and therein lies the complexity of the problem. It is tempting to think that if we just eliminated fast-food restaurants or reduced television viewing, we would solve the problem. However, the solution is not that sim-

ple. Obesity is a multifactorial problem. There is no single model to explain the rise in obesity within our society, and there will be no single, isolated solutions. The two central behaviors that must be addressed—food intake and physical activity—are complex behaviors that are extremely difficult to change.

Lack of a Unified Strategy

A second challenge in addressing child obesity is the lack of a unified strategy. There is no focal point or constituency to take clear responsibility for providing direction or coordination of prevention efforts. *The health care industry, school systems, nor community organizations have stepped forward to take ownership of the problem and to provide leadership for the solution.* The shared and cross-cutting levels of responsibility for the problem, and the lack of a leadership focus, work against the development of a unified strategy. Many people and organizations in health care, school, and community settings may be concerned about the problem, but coordination and exchange of information among these individuals and organizations is often lacking.

A major factor affecting the lack of a unified strategy is our limited knowledge and lack of consensus about which strategies are effective. We need to know more about the effectiveness of strategies for changing behavior at the individual level as well as the effects of environmental strategies, such as building bike paths and improving lighting and playground equipment. We also need a better understanding and consensus on the impact of policy changes in health care, school, and community settings that can stimulate greater awareness and provide increased resources to address child obesity.

Although more knowledge and consensus about the effectiveness of intervention strategies are critical, we also need to move forward with the knowledge we have. This involves translating research results into a unified strategy of actions and programs in communities. In this translation from research into action, there is a potential disconnect between researchers and people working at the community level. This disconnect is not simply a lack of communication, but also a lack of involvement of community implementers in the design of research studies and, later, in the translation of successful programs into community settings. Researchers need to build strong, ongoing relation-

ships with health care and education professionals and with youth program leaders in the community to develop more feasible and sustainable intervention programs both within health care, school, and community settings and across multiple settings and constituencies.

Development of a unified strategy to prevent child obesity does not imply a focus on a single type of intervention. We need to explore a variety of strategies that together can have a significant impact based on the best available data. As reviewed by Resnicow,[2] a variety of strategies have been explored in school, community, and health care settings with some encouraging results. Promoting physical activity is likely to be a key component of a successful strategy. Although some might say that definitive data are lacking to show that getting children to be more active will reduce the risk of obesity, the available data are strong and suggest that this is the case. There are other potential interventions that need to be further explored, including strategies to address the availability of healthy foods in cafeterias and vending machines in schools and interventions to modify the consumption of high-fat, high-energy diets. We need to sort out what we know and what we do not know about the relationship of these factors to obesity and to understand which interventions work and which do not.

Many ongoing community programs have not been adequately evaluated. Researchers with evaluation skills need to join forces with community organizations to evaluate interventions and to implement those that work best. Also, ways must be found to ensure the sustainability of programs. Many times when initial support for a community program disappears, the program goes away. The best time to consider the sustainability of programs is in the planning phase, not after the evaluation is complete.

Low Awareness and Priority

A third challenge to effective prevention efforts is the relatively low level of awareness and priority given to obesity as a health issue. Even if effective interventions and a unified strategy could be defined, obesity prevention will not receive priority attention until awareness is increased and obesity is placed substantially higher on the list of perceived public health problems. The fact is that childhood obesity is not on the radar screen of policy makers. It does not capture attention in the same way as AIDS, teen

pregnancy, and teen violence. Childhood obesity is considered a low priority, and this translates into insufficient resources and lack of sustained support. One problem with childhood obesity has been the perception that it is not an immediate crisis and that there is plenty of time to deal with the problem. Unfortunately, children are already suffering serious social (e.g., discrimination) and health (e.g., type 2 diabetes) consequences of their obesity. How much worse do the consequences of obesity have to get before the public recognizes the seriousness of the threat it poses?

One of the issues in raising awareness is the question of how to frame the message. One option is to focus attention on the problem of obesity and its associated health problems. However, if the emphasis is on obesity and body fat, we risk stigmatizing overweight children and possibly contributing to the risk of eating disorders. However, a message that focuses more positively on the promotion of physical activity and nutrition may not instill any sense of urgency. Perhaps the message needs to be adapted to the specific setting. For example, positive messages promoting the benefits of nutrition and physical activity could be used in school and community settings. Messages citing the health impact and health care costs associated with obesity may be more effective in efforts to enlist the support of political leaders and health care managers. Many people in the obesity, nutrition, and physical activity communities want to address childhood obesity. However, we cannot form a united effort until we deal with how to frame our message and agree on the banner under which we will march together.

Future Directions

The complexity of the issue of child obesity signals the need for coordinated actions by people and organizations from many sectors of society. The problem of obesity will be addressed successfully only when partnerships are formed that link efforts among individuals and organizations working in health care, schools, and the community. The formation of partnerships involving researchers, health care professionals, educators, and community and business leaders is an obvious first step. Partners from the food industry can contribute substantially, as can partners from manufacturers of sports and exercise equipment. Some industries whose products may promote inactivity, such as producers of tel-

evision programs, computer equipment, and video games, may be unaware that they are part of the problem or that they could potentially be part of prevention efforts. Individuals and organizations working to address the problem of child obesity will benefit from considering a wide range of potential partner organizations and from involving them in developing and sustaining intervention efforts.

The recent report on obesity from a World Health Organization expert consultation[4] concluded that obesity must be dealt with as a public health problem, and challenged each country to develop a specific plan of action. However, given the complex, multifactorial causes of child obesity and the diffusion of responsibility for the problem, it is unclear who will take the leadership in formulating a comprehensive plan to address obesity in US children. The Healthy People 2010 Guidelines[5] provide appropriate goals for our battle against the epidemic of obesity. What is missing is a plan on how we are to achieve these goals. We have lost, not gained, ground in this area over the past few decades. What is clear is the need for a plan that includes prevention efforts in health care, school, and community settings and that provides for coordination to allow efforts to be synergistic.

Although a comprehensive, national plan to deal with obesity is clearly needed, it would be a mistake to defer or lessen intervention efforts until such a plan is developed. Rather, ways must be actively sought to raise awareness about the health implications of obesity and to test and implement intervention efforts in health care, school, and community settings. Generating awareness and motivating action in the health care setting may be the most readily achievable, since health care professionals are increasingly encountering overweight children in their daily practice and there is clear evidence that obesity produces health problems and increases health care costs. Stimulating awareness and commitment in the school setting may be more difficult. Teachers, principals, and superintendents would be more likely to give priority to obesity prevention efforts if these efforts were more clearly linked to their primary concerns of increasing academic performance. It would be extremely valuable to be able to link obesity prevention efforts, such as improved nutrition education, food services, and physical activity programs, to improved academic performance, better attendance, or a reduction in disci-

pline problems. We do not at present have sufficient data to make this linkage conclusively, but we should be collecting such data and encouraging research in this area.

The incentives and motivation to address child obesity seem even less clear for the community setting. Perhaps the main deterrent for community action is the low level of awareness of obesity as a health problem. To address this issue, opportunities might be sought to link obesity prevention efforts with related social and health concerns that are a high priority in the community. For example, there is a high level of concern about child safety and the need for safe child care programs during after-school hours. These concerns may stimulate a high level of interest in well-supervised after-school programs that could emphasize physical activity as well as educate children about healthy eating practices. Such programs can be provided in partnership with local parks and recreation associations, YMCAs, and other organizations. Partnerships might also be formed with local police to improve safety around parks and recreational facilities. Safety may be a primary motivation for parents to send their children to such programs, but they may also find it appealing that their children will be learning about nutrition and participating in healthy physical activities.

If adequate resources are to be mobilized to address child obesity, a way must be found to bring this issue to the attention of policy makers at the local, state, and national levels. The most successful strategy for achieving this goal may involve both a bottom-up and a top-down approach. The bottom-up approach is developed at the community level. For example, in the school setting the key factor in success may be gaining the support of the principal or the administrator responsible for after-school programs. Getting that support may be achieved step by step by identifying the barriers, such as lack information about program options or concerns about program costs, and working to address each barrier one by one.

Development of top-down awareness is also critical. Success here will involve the development of a plan for a national campaign to increase awareness about child obesity. This campaign would provide information to policy makers and the general public about the importance of healthy diets and physical activity for children and the relationship of these factors to obesi-

ty, related health risks, and health care costs. Currently, no such plan exists. But the development of awareness at the national level is a vital long-range goal.

Conclusion

The factors leading to the increasing prevalence of child obesity are complex and will not respond to single interventions in health care, school, or community settings or to isolated policy and environmental changes. The principal determinants of future success are likely to lie in finding effective ways to raise awareness, building broad-based partnerships, and developing unified strategies. Raising awareness about the importance of obesity among parents, teachers, and community and political leaders is a particularly critical need in gaining sustained support for prevention efforts. Continued research to define successful intervention methods will be vital. Finally, the creative use of multiple communication channels, including professional publications, conferences, and the Internet, will be vital in efforts to share and update information about successful intervention efforts.

References

1. Goran MI. The rationale for a preventive approach to obesity in children. F. Trowbridge and D. Kibbe (eds.). Childhood Obesity: Partnerships for research and prevention. Atlanta, GA: ILSI Center for Health Promotion, 2002.
2. Resnicow K. Reviewing the research on child and adolescent obesity: what do we know? F. Trowbridge and D. Kibbe (eds.). Childhood Obesity: Partnerships for research and prevention. Atlanta, GA: ILSI Center for Health Promotion, 2002.
3. Dietz WH. Prevention of childhood obesity: individual, environmental, and policy issues. F. Trowbridge and D. Kibbe (eds.). Childhood Obesity: Partnerships for research and prevention. Atlanta, GA: ILSI Center for Health Promotion, 2002.
4. WHO Consultation on Obesity. Obesity: Preventing and Managing the Global Epidemic. WHO/NUT/NCD/98.1. Geneva, Switzerland: World Health Organization; 1997.
5. Department of Health and Human Services Office of Public Health and Science. Healthy People 2010 Objectives: Draft for Public Comment. Washington, DC: Department of Health and Human Services; 1999.

II. Reviewing the Research on Child and Adolescent Obesity: What Do We Know?

Ken Resnicow, PhD.

This review of research related to the prevention and treatment of obesity in children and adolescents focuses on four domains: schools, clinics, communities, and environment/policy. The quantity and quality of intervention research in these domains are highly variable, and therefore the depth and breadth of this review differ across the four settings. The review concludes with recommendations for future research.

School-based interventions

For several reasons, the school represents a potentially attractive intervention channel. Schools provide access to children. More than 95% of U.S. 5–17-year-olds are enrolled in school,[1] and no other public institution has as much continuous and intensive contact with children during their first two decades of life. Children eat one to two meals per day in school, so the school cafeteria represents a natural laboratory where students can learn and practice positive nutrition habits. Schools also contain potential positive role models in the form of teachers and peers who can serve as a rich source of behavioral reinforcement. School programs, in contrast to clinical interventions, can be delivered at little or no cost to families. Also, health and education are inextricably linked[2–4] healthy children are better prepared to learn, and conversely, educational accomplishment and aspirations are linked to better health status.[5–16]

Over the past 15 years, numerous school-based health

promotion interventions have directly or indirectly addressed obesity. Thirteen primary prevention studies[17-33] were identified that addressed obesity within the context of broad-based cardiovascular disease prevention, and four studies were identified[36,38,41,43] that targeted obesity exclusively. Excluded were studies that addressed diet or physical activity patterns but did not include assessment of adiposity.

As shown in Table 2-1, of the 13 broad-based interventions, seven were conducted in the United States (U.S.)[17-19,21,22,24,25] and six were international.[28-33] Four of the seven U.S. studies involved the Know Your Body program. Only one of the seven U.S. studies showed a positive effect on body mass index (BMI),[18] and two of the six international studies found positive effects on BMI. [28,31]For studies where skinfolds were assessed, two[18,34] U.S. studies and two international studies showed significant effects.[28,31] As reviewed elsewhere,[34–36] the methodologic rigor varied between the U.S. and international studies, with the U.S. studies more likely to employ randomization and appropriate statistical methods and to control for attrition.

In sum, multiple-risk-factor intervention studies that assessed adiposity show inconsistent effects. Whether such diffuse interventions should be expected to alter endpoints such as BMI is a matter of debate.[37] It should be noted that several of

TABLE 2-1. Results from First-Generation School-based Obesity Prevention Studies: Multiple-Risk-Factor Interventions

Obesity Prevention Studies	BMI	Skinfolds
U.S. studies		
Stanford [17,18]	Yes	Yes
KYB [119,20]	No	—
KYB 1a [21]	No	—
KYB 2 [22]	No	No
KYB 3 [23]	No	—
CHIC [34]	No	Yes
CATCH [25,26]	No	No
International studies		
Australia [27]	No	Yes
Crete [28]	Yes	No
Moscow [29]	No	Yes
Israel [30,31]	Yes	—
North Karelia [32]	No*	No
Oslo [33]	No	No

* Reverse effect.

these interventions were successful in modifying diet and physical activity behaviors. More intensive and focused interventions may be needed to alter physiologic outcomes.

Four published interventions that targeted obesity as a primary outcome were identified.[38–41] These studies include three conducted in the U.S. and one in Italy. One of the U.S. studies[39] involved 18 curriculum lessons delivered to 102 third through fifth graders over a 2-year period as well as modification of school lunch and physical activity programs. In the Italian study,[40] which lasted more than 5 years, 150 6- to 7-year-olds were provided with a curriculum by their classroom teachers and their parents received health education counseling. No control group was used. In another of the U.S. studies, the recently published Planet Health project,[38] which lasted more than 2 years, approximately 1300 sixth and seventh graders received an interdisciplinary curriculum that focused on decreasing television viewing to less than 2 hours per day, increasing physical activity, decreasing consumption of high-fat foods, and increasing consumption of fruits and vegetables. The third U.S. study[41] included a classroom curriculum to reduce television and videotape viewing and video game use. Specific intervention strategies included self-monitoring of television viewing and other sedentary behaviors; a "turnoff period" of no television or videos for 10 days followed by limiting television time to 7 hours per week thereafter and more intelligent and selective viewing; and restriction of television viewing time by an electronic timer attached to the power plug on the home television.

Two of the studies[39,40] reported no significant effects on adiposity, although the small sample sizes in both studies limited their statistical power. The results of the other two studies[38,41] were more encouraging. As shown in the Figure 2-1, the Planet Health study[38] found a significant reduction in the prevalence of obesity (defined as BMI and triceps skinfold greater than the 85th percentile) for girls in the treatment group relative to the control group. A significantly greater rate of obesity remission was also observed in girls. No effects on prevalence, incidence, or remission were observed for boys. Mediation analyses revealed that the effects of the intervention on adiposity were largely accounted for by changes in television viewing.

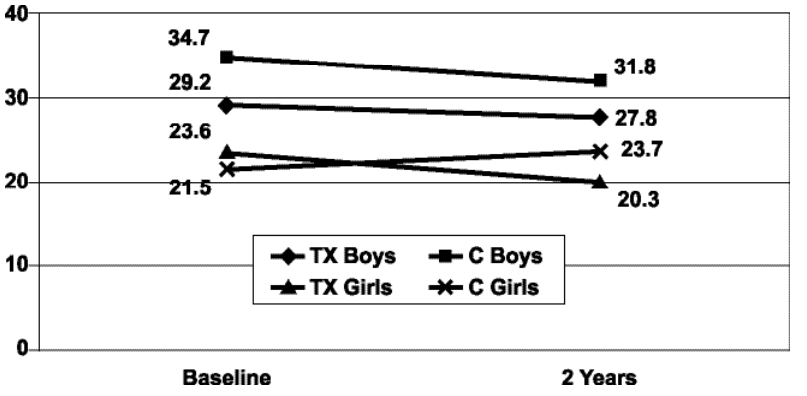

Figure 2-1. Results of Planet Health (recreated from reference 38)

Interestingly, the Planet Health program had a greater effect on African American girls than on European American girls. Earlier studies, mostly in adults, generally found African Americans to be less responsive to weight loss interventions.[42]

In one of the U.S. studies, Robinson's pilot study,[41] approximately 200 third and fourth graders and their parents from one intervention and one comparison school completed baseline and 6-month follow-up assessments. The intervention group showed a significantly lower increase in BMI, triceps skinfold, and waist circumference than the comparison group. The intervention youth also showed a significant decrease in time spent watching television based on both child and parent reports compared with the control group. There were no group differences in self-reported physical activity or cardiovascular fitness.

The Planet Health study[38] and the Robinson television reduction study[41] represent what could be considered exemplars of a second generation of school-based obesity prevention interventions. As shown in Table 2-2, other school-based obesity prevention intervention trials incorporate state-of-the-art intervention and assessment methods that may yield more promising results than earlier studies. These are randomized trials with elegant designs, appropriate statistical methods, and a strong grounding in behavioral theory. Several focus on reducing sedentary behaviors, which may be more effective than directly reinforcing diet or physical activity change.[44,45]

The school has also been used as a setting for the clinical treatment of overweight youth. Thirteen treatment studies,

TABLE 2-2. Second-Generation School-based Obesity Prevention Interventions

Study	Duration	Intervention
Reducing TV[41]	2 years	Classroom curriculum and parent newsletter to reduce TV, video tape, video game use
Planet Health[38]	2 years	Interdisciplinary curriculum to decrease sedentariness, increase physical activity, improve diet.
Pathways[43]	3 years	Physical activity, food service, classroom curriculum, family involvement
OPPrA[41]	3 years	Classroom curricula (5-A-Day, reducing TV, reducing high-fat junk food and fast food), physical education, food service, parent newsletters and video, intensive intervention for overweight children
PEACH[18]	2 years	Culturally specific classroom curricula (5-A-Day, reducing high-fat junk food and fast food), physical education, food service, parent newsletters and video

From reference 36.

most conducted before 1990, were identified in a survey of school-based obesity prevention studies.[34] Positive effects on adiposity were reported in 12 of these 13 studies. Despite the somewhat positive results of these interventions, however, the future of school-based treatment is questionable. Few if any school-based obesity treatment interventions have been published in the last 5 years. Several factors may underlie the lack of school-based treatment programs, including a decrease in school health personnel (e.g., school nurses), the low priority given to funding nutrition and chronic disease prevention activities in school health programs, fear of stigmatizing overweight youth, and difficulty obtaining third-party reimbursement for these services.

Community Interventions

Despite the intuitive appeal of engaging the broader community in the prevention and, perhaps to a lesser degree, the treatment of obesity, the research base in this area is strikingly thin. Only two intervention studies could be identified. The first, by Stolley and Fitzgibbon,[46] involved 65 low-income African American girls and their mothers. The intervention could be

classified as both primary and secondary prevention because approximately 20% of the girls and 60% of the parents were overweight. The program was conducted through the Cabrini-Green literacy program in Chicago. Over 12 weeks, youth and parents met together and received interactive lessons related to low-fat eating and increased physical activity. Many of the lessons were adapted from the Know Your Body program. The feasibility results of this pilot study were encouraging because 75% of the mother-daughter dyads attended at least 50% of the sessions. For mothers, there was a significant positive change in diet, e.g., lower saturated-fat intake and percent kilocalories from fat. There were no effects on mothers' adiposity. For daughters, there was a significant decrease in percent calories from fat. Results for girls' adiposity were not reported.

The second community-based intervention, Go Girls!, was recently conducted by Resnicow et al.[47,48] Go Girls! targeted low-income overweight African American adolescent females in four Atlanta public housing developments. The intervention consisted of biweekly group sessions for 4 months and weekly sessions for 2 months. Each session comprised three elements: (a) an interactive educational/behavioral activity, (b) 30–60 minutes of physical activity, and (c) preparation and tasting of low-fat, portion-controlled recipes. The intervention used a social cognitive framework aimed at improving both diet and physical activity patterns. Pre- and post-program assessment included multiple measures of diet and adiposity, blood lipids, blood pressure, self-reported physical activity, and cardiovascular fitness, as well as knowledge and attitudes related to nutrition and physical activity.

Given the developmental nature of the program, no control group was used. Instead, results were compared for high and low attenders, with the former defined as attending at least 50% of the sessions. (Table 2-3) Fifty-four percent were classified as low attenders (attended < 50% of sessions) and 46% as high attenders (attended > 50% of sessions). Approximately 60% of the girls attended each session. At 6-month follow-up, high attenders had significantly higher nutrition knowledge scores than did low attenders. They reported significantly better low-fat practices, greater perceived positive dietary changes, and greater perceived social support from friends and family for making diet and exer-

TABLE 2-3. Preliminary 6-Month Outcomes for Go Girls!: Dietary and Physiologic Measures (1997–1998)*

	High Attenders (n=22)		Low Attenders (n=20)	
	Adjusted Mean† (SD)	Raw Change‡ (SD)	Adjusted Mean	Raw Change
24-hour recall data§				
Total kilocalories	2011 (883)	-178 (1060)	2357	+117
% kcal from fat	34.6 (4.9)	-3.2	36.5 (3.9)	+0.22
Fiber (g)	15.1 (6.2)	+1.4	14.8 (7.8)	+0.62
Cholesterol (mg)	218 (74)	-52.0	265 (102)	+60
Food frequency data				
Total kcal	2187 (1216)	-1108 (1899)	2988	-242
% kcal from fat	42.3 (4.1)	+0.84	42.7 (5.4)	+1.2
Fiber (g)	10.4 (5.3)	-8.5	13.4 (9.1)	-4.1
Cholesterol (mg)	393 (291)	-155	466 (329)	-54
Sodium (mg)	3107 (1814)	-2230	4069 (2343)	-775
Physiologic measures				
BMI	35.3 (7.9)	+0.7	35.5 (6.8)	+ 0.9
% Body fat from skinfolds	44.7 (7.9)	-0.8	46.4 (10.3)	+ 0.4
DEXA (% body fat)	44.4 (5.2)	-0.1	46.3 (4.2)	+ 1.5
Total cholesterol (mg/dL)	159 (18.9)	-12.5	162 (30.0)	-5.6
HDL cholesterol (mg/dL)	44.4 (12.6)	+0.6	44.1 (6.8)	-5.9
Systolic blood pressure (mm Hg)	108	-6.0	113	-2.8

*From reference 48.
† Means are adjusted for age and baseline values.
‡ Difference between posttest and baseline values.
§ P value based on adjusted log-transformed posttest means.

cise changes. None of the other psychosocial outcomes differed significantly between the two groups. The 24-hour recall and food frequency questionnaire data indicated that high attenders reported lower total energy and percent energy from fat as well as lower cholesterol and sodium intakes at post-test than did low attenders.

Although the findings were in the favorable direction, no statistically significant differences were observed between high

and low attenders for any of the physical measures. Nonetheless, although the magnitude of the differences was small and should not be overinterpreted, there was a slight trend favoring high attenders for most measures. Go Girls! was designed as a feasibility study rather than an efficacy trial, and the lack of statistical significance for outcomes should be tempered by the fact that the sample size was small. Several of the changes observed would have been statistically significant with an additional 20 to 30 subjects.

One set of studies that does not fit neatly into the school, clinic, or community setting is the work of Gutin and others at the Medical College of Georgia. This group has conducted several physical training interventions with obese children and adolescents,[49–54] with intervention duration ranging from 10 weeks to 8 months and frequency ranging from 3 to 5 days per week. Consistent positive changes in adiposity and cardiovascular risk factors have been observed. These studies have established both the feasibility (attendance was generally good) and the efficacy of moderate and intensive programmed physical training in the treatment of obese children and adolescents.

Clinical Interventions

The literature on clinical intervention is extensive and has been reviewed elsewhere.[55–57] Here, only some key findings and opportunities for future research are highlighted. Recent work by Epstein et al[44,45,56] has shown that efforts to decrease sedentariness may be more effective than directly encouraging increased physical activity. This work has several implications. First, these findings can be seen as contradicting the social cognitive and behavioral models on which many current research interventions are based. Rather than directly reinforcing (or coercing) behavior change, there may be a benefit to providing children with behavioral choice. Epstein et al. have inculcated choice by creating a behavioral void, ie, reducing the amount of sedentary activity, and allowing the child to fill the void with self-selected activity. This in turn may impart a greater sense of psychologic ownership over behavioral decisions, in contrast to direct reinforcement, which, as dissonance theory might predict, could result in lower personal value placed on behaviors that the individual must be coerced into changing.

Interestingly, the behavioral choice approach may not work as well for dietary behavior. In one study involving overweight youth and their parents, directly reinforcing fruit and vegetable intake led to a greater increase in fruit and vegetable intake than did an intervention that targeted reduced consumption of high-fat/high-sugar foods.[58]

Another important finding, which again derives largely from Epstein et al's studies, is that children appear to maintain weight loss better than adults.[59] Additionally, reduction in adiposity is usually accompanied by favorable changes in cardiovascular risk factors, such as blood lipids, serum insulin, leptin, and blood pressure.[49,52,60] Moreover, interventions have not been shown to increase the likelihood of undernutrition or disordered eating.[55]

One element of the clinical literature that remains unresolved is the effects of involving parents in the treatment of overweight youth. Despite an intuitive assumption that parents are essential change agents for their children's diet and physical activity behaviors, the research in this regard is mixed. In the 10-year study by Epstein et al.[61,62] the parent-child group did better than the child-only group. Others, however, found little or no added benefit of involving parents when treating overweight youth.[63–66] One moderator variable that may explain these inconsistent results is the child's age. Studies by Epstein et al., involving youth ages 6–12, found positive results whereas most of the studies that found no benefit of involving parents included somewhat older youth. As children approach adolescence, involving their parents may be counterproductive. Adolescents may reject parental efforts to shape diet and physical activity behaviors, whereas younger children may be more responsive to parental intervention.

With regard to treating children jointly with their parents, results are again equivocal. Brownell et al.[66] found that a mother-child group treated separately did better than a group treated together. However, Wadden et al.[65] found no differences between groups with children treated together with mothers and groups with children and mothers treated in separate but concurrent sessions.[65] Stolley and Fitzgibbon[46] used joint mother-daughter sessions in a low-income, African-American community and found no effects on parent or child adiposity.[46]

One final interesting aspect of the clinical literature is that most of the successful interventions were conducted by psychologists or nutritionists rather than physicians. Yet physicians remain the gatekeepers for the detection and treatment of childhood obesity. This reflects a disconnect between research and clinical practice. Whether other professions such as psychologists or dietitians should be reimbursed directly for obesity treatment should be examined. Additionally, if physicians are going to continue serving as primary interventionists, strategies to improve their behavioral skills and policies to enable them to devote time to deliver these services effectively are needed. Most successful obesity interventions were conducted in sessions lasting 30 minutes or longer, whereas the typical medical encounter is scheduled for 10–15 minutes. Additionally, many of the successful interventions included a group component, whereas most medical encounters are one on one. Clearly, changes in the health care delivery system may be needed to incorporate successful obesity treatment.

Despite the extensive literature on clinical interventions, as noted by Barlow and Dietz in their introduction to an expert committee recommendations for the prevention and treatment of obesity in children,[67] many of the standards of care have not been subjected to rigorous evaluation: "Because so few studies of this problem have been performed, the approaches to evaluation and therapy presented here rarely are evidence-based" (p. E29).

Research is needed to determine the efficacy of many practice recommendations (e.g., family involvement, use of pharmacotherapy, group versus individual intervention) and to determine how intervention strategies may function differently across various demographic populations.

Environmental/Policy Interventions

Altering the environment and creating policies and laws that facilitate healthy eating and increased physical activity can have a significant impact on the obesity epidemic. Although numerous recommendations for such macro-level interventions have been proposed, the research base in this area is virtually nonexistent. "Little or no research has been conducted to

document the effects of such changes, so the potential environmental change from these public health approaches to modify individual behaviors remains largely unknown"[68] (Page 2).

Infrastructure changes that have been proposed include increasing the availability of safe and accessible play spaces, including after-school physical education programs. This includes paths for bicycling, in-line skating, walking, and jogging as well as multipurpose play areas, tennis courts, swimming pools, and so on.

Possible policy changes include reducing taxes on sports equipment and healthy foods and increasing taxes on high-fat and unhealthful, unnutritious foods. Such tax laws, although perhaps untenable at first glance, have precedent. In Australia, a law was recently passed that eliminated the tax on sunscreens. In the United States and elsewhere, cigarettes and alcohol are taxed more heavily than other consumer products.

Legislative intervention could also be used to limit the content of advertising during children's television programming. There is considerable evidence that television advertising affects children's food preferences and consumption habits; thus, limiting the frequency to which youth are exposed to sophisticated advertisements could have a significant impact on food choices.[69,70] Approximately half of the advertisements that youth are exposed to are for food items, and in the vast majority of cases, 80%–90%, such ads are for foods with low nutritional value.[70–72]

Other legislative measures that have been discussed include warning labels on fast foods and high-fat snacks. When warning labels were initially proposed for alcohol and tobacco products in the U.S., there was considerable resistance on the part of manufacturers, and proposing food warning labels would no doubt meet with equal if not greater barriers. For each of these changes—taxation, advertising restrictions, and warning labels—determining what is considered "healthy" and "unhealthy" and which products and services would be affected would undoubtedly be an extremely complicated process, which might ultimately prove impossible. Despite the obvious classification complexities and potential conflict with a free-market system, it is recommended that expert panels comprising behavioral researchers, health economists, and representa-

tives of industry be convened to seriously examine the feasibility and potential impact of such regulations.

A related measure that has been proposed is the zoning of fast-food restaurants and vendors of other calorically dense products (e.g., donut shops). Studies are needed to determine whether a relationship exists between the concentration of fast food establishments and the eating habits and adiposity of local residents. If so, regulations regarding how many fast food establishments can exist within a geographic area might be warranted. Analogous "zoning" already exists for casinos and "adult" bookstores. Moreover, Sallis et al.,[73] in a sample of 2000 California adults, demonstrated a significant association between density of exercise facilities and frequency of exercise among local residents. Related methods to map neighborhood restaurants and food businesses have been developed.[74]

Synthesis, Summary, and Future Directions

Reconceptualization of Obesity

The effective treatment of obesity may be facilitated by its reconceptualization. First, obesity should perhaps be considered more like cancer, i.e., not as one disease but as a rubric of many diseases, each with somewhat different etiology, course, and treatment. Tailored interventions may prove more effective than standardized programs that adopt a "one size fits all" approach. As noted by Epstein et al.,[55] "Treating obesity as a homogeneous condition, with all participants receiving a common intervention, might contribute to the mixed treatment outcomes that are reported" (p. 566).

Individualizing treatment should entail not only tailoring interventions to participants' readiness to change[75–77] but also designing interventions according to individual eating and exercise patterns and preferences as well as genetic and metabolic characteristics. Dysregulation of energy balance can of course be attributed to increased caloric intake or decreased output. However, the way that increased intake or decreased output manifests across individuals can be highly variable. Nonetheless, few research interventions have attempted to tailor diet and physical activity programs according to these more

"micro-level" differences.[55] For example, excess intake could be the result of high-fat or high-sugar foods. For someone who is a "high-fat food consumer," with the use of a "tailored" study, excess caloric intake could be attributed to one or two foods, whereas for others, excess intake might result from a variety of foods. In addition to a focus on specific foods, "tailoring" could also be used to account for eating patterns such as consuming large serving sizes, finishing everything on one's plate, eating second helpings, or eating at "all you can eat" establishments. The same applies to activity patterns, because some youth may be classified as high sedentary and low activity whereas others may be classified as high activity and high sedentary. For example, children could spend many hours a week playing their favorite sport but also spend an equal number of hours watching the same sport on television. The intervention required for high-activity/high-sedentary youth may be quite different from the former case.

Genetic and metabolic opportunities can also be targeted. For example, individuals with a low resting metabolism or low thermogenic response to food may require different interventions. Similarly, individuals predisposed to storing fat abdominally may respond differently to an intervention than those who store fat in their thighs, hips, and buttocks. Interventions could also be tailored to family factors. For example, youth with two overweight parents may require different interventions than youth with two lean parents, whereas youth with psychologically high-functioning parents may require different treatment than those with parental psychopathology. Finally, as Gortmaker et al. suggest,[38] interventions may also function differently by gender and ethnicity.

Continued Behavioral Research

Additional basic research regarding the development and modification of eating and exercise habits is needed. One avenue for exploration is satiety training, or what others have called "conditioned satiation."[55] Satiety training entails increasing individuals' sensitivity to cues that ingestion should cease and helping them redefine hunger impulses that may arise from dysphoric affective states such as anxiety and depression rather

than from pure physiologic hunger. There is evidence that overweight children are less sensitive to satiety cues than normal-weight youth and that they eat faster and decelerate their eating more slowly toward the end of a meal.[78] There has been surprisingly little research into strategies to help individuals bridge the 10–20-minute gap between consumption and physiologic satiety. The concept of reconditioning overweight individuals to become more sensitive to internal cues dates back to the 1970s, when it was proposed that overweight individuals are more "externally bound" to hunger and satiety cues than are nonoverweight people; that is, they are more likely to stop eating when all the food is gone from their plate rather than in response to internal sensations of satiety. Initial interventions to address this problem largely involved the micromanagement of eating, eg, cutting food into smaller pieces, chewing longer, and so on. These strategies did not prove effective. However, interventions that help individuals redefine and tolerate hunger and satiety cues based on the analogous approaches that have been used successfully for the management of chronic pain[79] may be applicable to obesity control.

Other avenues for future research include exploring the role of sugar and fat replacements. Although there is often reluctance to incorporate into the diet foods with artificial sweeteners and products that contain substances such as olestra, evaluation of the impact of these technologic foods on weight management in youth may be warranted. Similarly, the role of pharmacologic therapy in youth, e.g., Meridia (sibutramine) or Xenical (orlistat),[80] should be considered.[81] Candidates for drug therapy might include youth with an immediate medical need to lose weight, such as those with severe weight-related sleep apnea, with morbid obesity, or for whom surgery needed for other problems is precluded because of their weight.[82] The use of technology to limit television viewing also holds considerable promise. Devices that limit the amount of time youth can watch television as well as devices rigged to a television so that it operates only when a bicycle or related apparatus is being pedaled have been tested in small studies. Additional research is needed to test the feasibility and effectiveness of incorporating these devices into commercially viable products. Finally, research is needed to test interventions

using the Internet or multimedia technology such as interactive CD-ROM and virtual reality devices.

In summary, the amount, quality, and success of obesity interventions vary across the four domains reviewed here. For schools, despite the limited impact of the first generation of preventive interventions, initial results from the second generation of interventions are more encouraging.

For clinical interventions, perhaps the greatest challenge lies in technology transfer: how do we enable practicing physicians to incorporate into their practices successful research-based interventions? Structural changes in how obesity treatment is delivered and reimbursed are needed both to improve physicians' ability and willingness to provide these services and to better engage psychologists, dietitians, and other behavioral specialists who may be better prepared to treat this disorder. Third-party reimbursement for the evaluation and treatment of pediatric obesity is limited. In one pediatric hospital, only 11% of obesity-related charges were reimbursed.[83] Without financial incentives, it is unlikely that practitioners will invest the time and effort required to effectively treat the condition. Community-based intervention research is in its infancy. Basic studies demonstrating the efficacy of these interventions are needed, including studies examining their potential institutionalization.

There is reason for optimism. Over the past few years, there has been a marked increase in federally funded behavioral research related to obesity prevention in youth. Two notable National Institutes of Health initiatives include four prevention studies targeting African American youth funded by the Heart, Lung, and Blood Institute (RFA HL 98-010) and a trans-NIH initiative to develop innovative approaches to obesity prevention and treatment (RFA DK 99-010). Although the recent increase in pediatric obesity is sobering, the silver lining may be the increased research it has spawned.

References

1. US Department of Education. National Center for Educational Statistics Digest of Educational Statistics Washington DC NCES; 1990: 41-159.
2. Novello A. Healthy children ready to learn: the Surgeon General's initiative for children. J School Health. 1991;61:359–360.

3. Kolbe L. Why school health education? an empirical point of view. Health Educ. 1985;16:116–120.
4. Kolbe L, Green L, Forety J. Appropriate functions of health education in schools: improving health and cognitive performance. In: Kraisnger N, Arasteli J, Cataldo M, eds. Child Health Behavior: A Behavioral Pediatrics Perspective. New York: John Wiley; 1986.
5. James WP, Nelson M, Ralph A, Leather S. Socioeconomic determinants of health: the contribution of nutrition to inequalities in health. Br Med J. 1997;314:1545–1549.
6. Novotny TE, Warner KE, Kendrick JS, Remington PL. Smoking by blacks and whites: socioeconomic and demographic differences. Am J Public Health. 1988;78:1187–1189.
7. Winkleby M, Fortmann S, Barrett D. Social class disparities in risk factors for disease: eight-year prevalence patterns by level of education. Prev Med. 1990;19:1–12.
8. Flay B, Phil D, Koepke D, Thomson SJ, Santi S, Best A, et al. Six-year follow-up of the first Waterloo school smoking prevention trial. Am J Public Health. 1989;79:1371–1376.
9. Pirie P, Murray D, Luepker R. Smoking prevalence in a cohort of adolescents, including absentees, dropouts, and transfers. Am J Public Health. 1988;78:176–178.
10. Hawkins JD, Catalano RF, Miller JY. Risk and protective factors for alcohol and other drug problems in adolescence and early adulthood: implications for substance abuse prevention. Psychol Bull. 1992;112:64–105.
11. Johnston L, O'Malley P, Bachman J. National Survey Results on Drug Use from the Monitoring the Future Study, 1975–1994. Rockville, MD: U.S. Department of Health and Human Services; 1995.
12. Bachman JG, Johnston LD, O'Malley PM. Monitoring the Future: A Continuing Study of the Lifestyles and Values of Youth, 1994. Ann Arbor, MI: University of Michigan, Survey Research Center; 1996.
13. Tershakovec AM, Weller SC, Gallagher PR. Obesity, school performance and behaviour of black, urban elementary school children. Int J Obes Relat Metab Disord. 1994;18:323–327.
14. Pollitt E, Gersovitz M, Gargiulo M. Educational benefits of the United States school feeding program: a critical review of the literature. Am J Public Health. 1978;68:477–481.
15. Meyers A, Sampson A, Weitzman M, Rogers B, Kayne H. School breakfast program and school performance. AJDC. 1989;143:1234–1239.
16. Lindeman A, Clancy K. Assessment of breakfast habits and social/emotional behavior of elementary schoolchildren. J Nutr Educ. 1990;22:226–231.
17. Killen J, Robinson T, Telch M. The Stanford Adolescent Heart Health Program. Health Educ Q. 1989;16:263-283.
18. Killen JD, Telch MJ, Robinson TN, Maccoby N, Taylor CB, Farquhar JW. Cardiovascular disease risk reduction for tenth graders: a multiple-factor school-based approach. JAMA. 1988;260:1728–1733.
19. Walter HJ. Primary prevention of chronic disease among children: the school-based "Know Your Body" intervention trials. Health Educ Q. 1989;16:201–214.
20. Resnicow K, Cross D, Wynder E. The Know Your Body program: a review of evaluation studies. Bull N Y Acad Med. 1993;70:188–207.
21. Walter HJ, Hofman A, Vaughan R, Wynder EL. Modification of risk factors for coronary heart disease. N Engl J Med. 1988;318:1093–1100.
22. Bush PJ, Zuckerman AE, Taggart VS, Theiss PK, Peleg EO, Smith SA. Cardiovascular risk factor prevention in black school children: the "Know Your Body" evaluation project. Health Educ Q. 1989;16:215–227.
23. Resnicow K, Cohn L, Reinhardt J, Cross D, Futterman R, Kirschner E, et al.

A three-year evaluation of the Know Your Body program in minority schoolchildren. Health Educ Q. 1992;19:463–480.
24. Harrell JS, McMurray RG, Bangdiwala SI, Frauman AC, Gansky SA, Bradley CB. Effects of a school-based intervention to reduce cardiovascular disease risk factors in elementary-school children: the Cardiovascular Health in Children (CHIC) study. J Pediatr. 1996;128:797–805.
25. Luepker RV, Perry CL, McKinlay SM, Nader P.R., Parcel G.S., Stone, E.J., et al. Outcomes of a field trial to improve children's dietary patterns and physical activity. JAMA. 1996;275:768–776.
26. Nader P, Stone E, Lytle L, Perry CL, Osganian SK, Kelder S, et al. Three-year maintenance of improved diet and physical activity. Arch Pediatr Adolesc Med. 1999;153:695–704.
27. Vandongen R, Jenner D, Thompson C, Taggart AC, Spickett EE, Burke V, et al. A controlled evaluation of a fitness and nutrition intervention program on cardiovascular health in 10- to 12-year-old children. Prev Med. 1995;24:9–22.
28. Lionis C, Kafatos A, Vlachonikolis J. The effects of a health education intervention program among Cretan adolescents. Prev Med. 1991;20:685–699.
29. Alexandrov A, Maslennikova G, Kulikov S, Propirnij G, Perova N. Primary prevention of cardiovascular disease: 3-year intervention results in boys of 12 years of age. Prev Med. 1992; 21:53–62.
30. Tamir D, Brunner S, Edelstein P, Feuerstein A, Palti H, Halfon ST. Segev health promotion project in Jerusalem elementary schools. Harefuah. 1990;118:527–531.
31. Tamir D, Feurstein A, Brunner S. Primary prevention of cardiovascular diseases in childhood: changes in serum total cholesterol, high density lipoprotein, and body mass index after 2 years of intervention in Jerusalem school children age 7–9 years. Prev Med. 1990;19:22–30.
32. Puska P, Vartiainen E, Pallonen U, Salonen JT, Poyhia P, Koskela K, et al. The North Karelia youth project: evaluation of two years of intervention on health behavior and CVD risk factors among 13- to 15-year old children. Prev Med. 1982;11:550–570.
33. Tell GS, Vellar OD. Noncommunicable disease risk factor intervention in Norwegian adolescents: the Oslo youth study. In: Hetzel B, Berenson G, eds. Cardiovascular Risk Factors in Childhood: Epidemiology and Prevention. Amsterdam: Elsevier Science Publishers; 1987.
34. Resnicow K. School-based obesity prevention: population vs high-risk interventions. Ann N Y Acad Sci. 1993;699:154–166.
35. Resnicow K, Robinson TN. School-based cardiovascular disease prevention studies: review and synthesis. Ann Epidemiol. 1997;7:14–31.
36. Stone EJ, McKenzie TL, Welk GJ, Booth ML. Effects of physical activity interventions in youth: review and synthesis. Am J Prev Med. 1998;15:298–315.
37. Resnicow K, Robinson T, Frank E. Advances and future directions for school-based health promotion: commentary on the CATCH trial. Prev Med. 1996;25:373–383.
38. Gortmaker S, Peterson K, Wiecha J, Sobol AM, Dixit S, Fox MK, et al. Reducing obesity via a school-based interdisciplinary intervention among youth. Arch Pediatr Adolesc Med. 1999;153:409–418.
39. Donnelly JE, Jacobsen DJ, Whately JE, Hill JO, Swift LL, Cherrington A, et al. Nutrition and physical activity program to attentuate obesity and promote physical and metabolic fitness in elementary school children. Obes Res. 1996;4:229–243.

40. Angelico F, Ben M, Fabiani L, Lentini P, Pannozzo F, Urbinati GC, et al. Management of childhood obesity through a school-based programme of general health and nutrition education. Public Health. 1991;105:393–398.
41. Robinson T. Reducing children's television viewing to prevent obesity: a randomized controlled trial. JAMA. 1999;282:1561–1567.
42. Kumanyika SK, Obarzanek E, Stevens VJ, Hebert PR, Whelton PK. Weight-loss experience of black and white participants in NHLBI-sponsored clinical trials. Am J Clin Nutr. 1991;53:1631S–1638S.
43. Davis SM, Going SB, Helitzer DL, Teufel NI, Snyder P, Gittelsohn J, et al. Pathways: a culturally appropriate obesity-prevention program for American Indian schoolchildren. Am J Clin Nutr. 1999;69:(4 Suppl), 796S-802S.
44. Epstein LH, Saelens BE, Myers MD, Vito D. Effects of decreasing sedentary behaviors on activity choice in obese children. Health Psychol. 1997;16:107–113.
45. Epstein LH, Valoski AM, Vara LS, McCurley J, Wisniewski L, Kalarchain MA, et al. Effects of decreasing sedentary behavior and increasing activity on weight change in obese children. Health Psychol. 1995;14:109–115.
46. Stolley MR, Fitzgibbon ML. Effects of an obesity prevention program on the eating behavior of African American mothers and daughters. Health Educ Behav. 1997;24:152–164.
47. Resnicow K, Yaroch A, Davis A, Wang DT, Lyn R, London J, et al. GO GIRLS!: Development of community-based nutrition and physical activity program for African American adolescent females. J Nutr Educ. 1999;31:283-185.
48. Resnicow K, Yaroch AL, Petty A, Wang TD, Carter S, Slaughter L, et al. Go Girls!: results from a pilot nutrition and physical activity program for low-income overweight African American adolescent females. Health Educ Behav. 2000;27:616-631.
49. Gutin B, Ramsey L, Barbeau P, Cannady W, Ferguson M, Litaker M, et al. Plasma leptin concentrations in obese children: changes during 4-mo periods with and without physical training. Am J Clin Nutr. 1999;69:388–394.
50. Owens S, Gutin B, Allison J, Riggs S, Ferguson M, Litaker M, et al. Effect of physical training on total and visceral fat in obese children. Med Sci Sports Exerc. 1999;31:143–148.
51. Gutin B, Owens S, Okuyama T, Riggs S, Ferguson M, Litaker M. Effect of physica training and its cessation on percent fat and bone density of children with obesity. Obes Res. 1999;7:208–214.
52. Gutin B, Cucuzzo N, Islam S, Smith C, Stachura ME. Physical training, lifestyle education, and coronary risk factors in obese girls. Med Sci Sports Exerc. 1996;28:19–23.
53. Gutin B, Cucuzzo N, Islam S, Smith C, Moffatt R, Pargman D. Physical training improves body composition of black obese 7- to 11-year-old girls. Obes Res. 1995;3:305–312.
54. Barbeau P, Gutin B, Owens S, Okuyama T, Bauman M, Vemulapalli S, et al. Effects of moderate and high intensity physical training on cardiovascular fitness, adiposity, and the insulin resistance syndrome in obese teenagers. In: Childhood Obesity: Partnerships for Research and Prevention. Atlanta, GA: International Life Sciences Institute; 1999.
55. Epstein L, Myers M, Raynor H, Saelens BE. Treatment of pediatric obesity. Pediatrics. 1998;101:554–570.
56. Epstein LH, Coleman KJ, Myers MD. Exercise in treating obesity in children and adolescents. Med Sci Sports Exerc. 1996;28:428–435.
57. Epstein LH. Family-based behavioural intervention for obese children. Int J Obes Relat Metab Disord. 1996;20:S14–S21.
58. Raynor H, Epstein L, Gordy C. Effects of increasing fruits and vegetables and

decreasing high-fat and/or high-sugar foods during obesity treatment [abstract]. Society of Behavioral Medicine 20th Annual Meeting,. San Diego,1999.
59. Epstein LH, Valoski AM, Kalarchian MA, McCurley J. Do children lose and maintain weight easier than adults? a comparison of child and parent weight changes from six months to ten years. Obes Res. 1995;3:411–417.
60. Epstein LH, Kuller LH, Wing RR, Valoski A, McCurley J. The effect of weight control on lipid changes in obese children. AJDC. 1989;143:454–457.
61. Epstein LH, Wing RR, Koeske R, Andrasik F, Ossip DJ. Child and parent weight loss in family-based behavior modification programs. J Consult Clin Psychol. 1981;49:674–685.
62. Epstein L, Valoski A, Wing R, McCurley J. Ten-year follow-up of behavioral, family-based treatment for obese children. JAMA. 1990;264:2519-2523.
63. Kirschenbaum DS, Harris ES, Tomarken AJ. Effects of parental involvement in behavioral weight loss therapy for preadolescents. Behav Therapy. 1984;15:485–500.
64. Coates TJ, Killen JD, Slinkard LA. Parent participation in a treatment program for overweight adolescents. Int J Eat Disord. 1982;1:37–48.
65. Wadden TA, Stunkard AJ, Rich L, Rubin CJ, Sweidel G, McKinney S, et al. Obesity in black adolescent girls: a controlled clinical trial of treatment by diet, behavior modification, and parental support. Pediatrics. 1990;85:345–352.
66. Brownell KD, Kelman JH, Stunkard AJ. Treatment of obese children with and without their mothers: changes in weight and blood pressure. Pediatrics. 1983;71:515–523.
67. Barlow SE, Dietz WH. Obesity evaluation and treatment: expert committee recommendations: the Maternal and Child Health Bureau, Health Resources and Services Administration and the Department of Health and Human Services. Pediatrics. 1998;102:E29.
68. Story M, Fitzgibbon M, Donato K. Community strategies for addressing the prevention and treatment of child and adolescent obesity. Background paper for the conference Childhood Obesity: Partnerships for Research and Prevention, ILSI, May, 1999.
69. Taras HL, Sallis JF, Patterson TL, Nader PR, Nelson JA. Television's influence on children's diet and physical activity. J Dev Behav Pediatr. 1989;10:176–180.
70. Birch L, Fisher J. Development of eating behaviors among children and adolescents. Pediatrics. 1998;101:539–459.
71. Cotugna N. TV ads on Saturday morning children's programming: what's new? J Nutr Educ. 1988;20:125–127.
72. Taras HL, Gage M. Advertised foods on children's television. Arch Pediatr Adolesc Med. 1995;149:649–652.
73. Sallis JF, Hovell MF, Hofstetter CR, Elder J. P., Hackley M, Caspersen CJ, et al. Distance between homes and exercise facilities related to frequency of exercise among San Diego residents. Public Health Rep. 1990;105:179–185.
74. Dixit S, Wang X. Using neighborhood mapping in the design and implementation of nutrition education programs. In: Childhood Obesity: Partnerships for Research and Prevention. Atlanta, GA: International Life Sciences Institute; 1999, p. 94.
75. Campbell MK, Symons M, Demark-Wahnefried W, Polhamus B, Bernhardt, J.M., McClelland, J.W. et al. Stage of change and psychosocial correlates of fruit and vegetable consumption among rural African-American church members. Am J Health Promotion. 1998;12:185–191.
76. Booth ML, Macaskill P, Owen N, Oldenburg B, Marcus BH, Bauman A. Population prevalence and correlates of stages of change in physical activity.

Health Educ Q. 1993;20:431–440.
77. Calfas KJ, Long BJ, Sallis JF, Wooten WJ, Pratt M, Patrick K. A controlled trial of physician counseling to promote the adoption of physical activity. Prev Med. 1996;25:225–233.
78. Barkeling B, Ekman S, Rossner S. Eating behaviour in obese and normal weight 11-year-old children. Int J Obes Relat Metab Disord. 1992;16:355–360.
79. Sarafino E. Managing and controlling clinical pain. In: Health Psychology: Biopsychosocial Interactions. 2nd ed. New York: John Wiley & Sons; 1994:371–398.
80. Davidson MH, Hauptman J, DiGirolamo M, Foreyt JP, Halsted, CH, Heber D, et al. Weight control and risk factor reduction in obese subjects treated for 2 years with orlistat: a randomized controlled trial. JAMA. 1999;281:235–242.
81. Aronne LJ. Modern medical management of obesity: the role of pharmaceutical intervention. J Am Diet Assoc. 1998;98:S23–S26.
82. Dietz W. Health consequences of obesity in youth: childhood predictors of adult disease. Pediatrics. 1998;101:518–525.
83. Tershakovec AM. Insurance reimbursement for the treatment of obese children. In: Childhood Obesity: Partnerships for Research and Prevention. Atlanta, GA: International Life Sciences Institute; 1999:40.

III. The Rationale for a Preventive Approach to Obesity in Children

Michael I. Goran, PhD

Specific prevalence estimates for obesity in children vary depending on the population studied and the methods and definitions used. Current estimates suggest that overall, approximately 25% of children in the U.S. are obese, but significant differences have been observed in regard to ethnic group. For example, data from schoolchildren in Birmingham, AL,[1] suggest that when obesity is defined as an ideal body weight above 120% of the reference median, the prevalence in African-American girls at age 10 years is 38% compared with 21% in white girls. Obesity prevalence also varies in relation to cultural setting, gender, and geographic location. Despite these variations in prevalence, obesity is increasingly viewed as a global epidemic, as outlined by the World Health Organization.[2]

In this chapter, the rationale for a preventive approach for the management of the obesity epidemic in children is presented, with attention to the following five factors:

- epidemiologic factors and future projections,
- health risk factors,
- etiologic factors,
- economic factors, and
- preventive versus treatment strategies.

Epidemiologic Factors

The secular increase in body weight in children has been well documented over the last few decades. Freedman et al.[3] exam-

ined 26,000 measurements of body weight in 11,000 children and showed that the secular increase in body weight was 0.2 kg per year. Obesity is also a persistent problem,[4,5] and the degree of persistence into adulthood changes with the age at which obesity originally develops. For example, if obesity is apparent at 7 years of age, the likelihood of persistence into adulthood is about 41%. If obesity is apparent at adolescence, the persistence into adulthood is almost a certainty, at 80%. Also, as shown by Whitaker et al.,[6] the odds ratio for obesity in adulthood (adjusted for parental obesity) increases with age. Finally, obesity is familial: the likelihood of obesity in children is 9% if both parents are lean, but rises to 60%–80% when both parents are obese.[7] The projected outcome of these epidemiologic observations is a tremendous increase in the prevalence of obesity in future generations. Thus, obesity is a substantial current problem that is projected to worsen in future generations.

Health Risk Factors

Child obesity is not an issue solely related to body weight and physical appearance. Even in childhood, obesity has serious health implications. From a health perspective, there are numerous implications for our nation and for global health in general. The relationship between obesity and increased risk for cardiovascular disease is apparent early in life.[8] In addition, obesity during childhood significantly increases the risk for non-insulin-dependent diabetes mellitus (Type 2 Diabetes) later in life.[8] Recent epidemiologic data show that even though NIDDM is typically thought of as an adult disease, its incidence is increasing dramatically in children and is related to increased obesity. The major risk factors for Type 2 Diabetes in children are obesity and family history of Type 2 Diabetes, with the highest prevalence observed in African American females,[9] a subgroup of the population with a higher prevalence of obesity.[1] Other health risks of obesity in children include psychosocial maladjustment, early maturation, orthopedic problems, and sleep apnea.[10]

Increased obesity is hypothesized to increase the risk of NIDDM and cardiovascular disease through the effects of visceral fat;[8] the exact mechanism(s) of this effect, however, is not clearly known. Visceral fat, or fat surrounding the organs, is associated with the negative health effects of obesity in adults.[11] Visceral fat begins to build up early in life and is present in chil-

dren,[12] and it is significantly related to negative health risk factors for Type 2 Diabetes and cardiovascular disease.[8] These observations are apparent across the spectrum of adiposity in children.[12] In addition, African American children display an increased risk for Type 2 Diabetes that is not explained by differences in adiposity.[13,14] Growing evidence also supports the relationship between obesity and cancer risk.[15,16] Moreover, obesity-related behaviors, including physical inactivity, increase risk for breast cancer[17] and colon cancer.[18,19] In total, dietary factors and physical inactivity associated with obesity result in approximately 300,000 deaths per year, second only to smoking.[20] Thus, regardless of the mechanism, obesity in children has serious and numerous negative health implications.

Etiologic Factors

The possible etiologic factors that cause obesity include genetic abnormalities, dysregulation of energy balance, excessive caloric intakes, and inadequate physical activity, or more likely, a multifactorial, complex interaction of all of these factors. Obesity is frequently defined as a simple problem of energy balance. The principle of energy balance—in which the energy input into the body (food intake) equals the energy output from the body (energy expenditure)—is based on the First Law of Thermodynamics. When energy input equals energy output, there is a maintenance of energy stores and no change in body weight. An average 5-year-old consumes and expends approximately half a million calories per year. This consumption and expenditure of energy is controlled such that under usual circumstances, the only energy gain in the body is that needed for growth (about 2% of ingested calories). Disturbances in this control result in a failure to match energy intake with energy expenditure, which may result in excess or insufficient energy storage.

It is not yet known whether obesity is explained by excess intake relative to expenditure or by defective expenditure relative to intake, or a combination. Both are likely such that obesity is best understood as a failure of the control mechanisms to match intake with expenditure. In fact, only a very small error in this matching over time can lead to obesity. Studies have shown that in children, an excess of intake over expen-

diture of less than 50 kcal (209 kJ) per day can result in obesity over time.[21]

Abnormalities in energy expenditure have been widely examined as a possible contributing factor to the increased prevalence of obesity. The simple hypothesis is that an abnormally low energy expenditure and/or increased energy intake increases the risk of obesity because of a greater likelihood of positive energy balance. From an evolutionary perspective, it is implausible that basal metabolic rates in humans have plummeted in the last 20 years. It is far more likely that energy expenditure through voluntary physical activity has declined. Dietary factors such as increased food availability are obviously important from the standpoint of environmental influences. However, recent decreasing trends in fat and calorie intake have not been linked to a reduction in obesity; in fact, obesity has continued to increase despite increased consumption and availability of reduced-fat foods.[22]

For these reasons, physical activity, or more precisely physical inactivity, is an important etiologic factor in understanding obesity trends. Modernization and technology have led us to become a more sedentary society because of the decreased need for physical activity. In fact, small reductions in physical activity can contribute to obesity. Between 1976–1980 and 1988–1991, the average weight gain in adults was 3.6 kg, which could be explained by a calorie surfeit of 50 kcal (209 kJ) per day.[23] This calorie surfeit can occur, for example, by sitting rather than walking for just 15 minutes per day.[24] Epidemiologic data suggest that 20% of U.S. children do not get more than 2 hours of vigorous physical activity per week and that 67% of U.S. children watch more than 2 hours of television per day.[25] Fewer than 36% of U.S. children get physical education on a daily basis, and often most of the class time is spent being physically inactive.[26] Recess has practically disappeared from U.S. schools.

Examination of the role of physical activity in the etiology of obesity is complicated by the multidimensionality of physical activity. Depending on how the data are expressed, some cross-sectional studies show the expected inverse correlation between adiposity and physical activity in children, whereas others do not.[27] We previously showed that hours per

week of reported activity may be more significantly related to adiposity in children than the combined energy cost.[27] There is a paucity of longitudinal and cause-and-effect data to provide compelling evidence that reduced physical activity in children leads to obesity and disease outcome later in life. Several studies in adults, however, have clearly linked physical activity to disease outcome. In a Finnish twin study of 16,000 men and women followed for 20 years,[28] those who exercised moderately had a significantly reduced odds ratio for death. The effect was more pronounced in women.

There is clearly a genetic and familial aspect to obesity, which is likely to be complex and involve multiple genes.[29] Genetic factors alone, however, cannot explain the sudden increased prevalence of obesity in populations around the globe, because changes in the gene pool cannot occur that rapidly. It therefore seems likely that the involvement of genetic factors will prove to be important in terms of gene-environment interactions.[30] Specific changes in the environment may trigger expression of certain genes that occur only in genetically susceptible individuals. The increased obesity and diabetes in Pima Indians living in Arizona may be an example of this phenomenon.[31]

Thus, from an etiologic perspective, the mechanism of obesity remains unknown, but it may be the result of the interaction of a normal physiologic system with an environment that promotes obesity because of an increasing sedentary lifestyle coupled with increased food availability.

Economic Factors

Providing treatment for health problems associated with obesity has been projected to incur health care costs of about $100 billion per year in adults.[32] Because obesity can be a lifelong disease beginning in childhood, it clearly may be more cost effective to prevent rather than treat obesity. The cost of obesity prevention is unknown, but is likely to be far less than $100 billion per year. Furthermore, changing behavior in children may have long-lasting effects. Also, prevention has been very successful for a number of other health outcomes requiring health behavior change, including such major public health challenges as smoking and Human Immuno-Deficiency Virus.

Prevention Versus Treatment

With regard to the management of obesity in children, there are many advantages of prevention over treatment. From a metabolic perspective, prevention may be more effective because it is likely that the obese state may be associated with potentially metabolic and physiologic alterations that may be difficult to reverse. Thus, even if weight loss is achieved, these alterations may make it more difficult to lose and/or maintain weight. Prevention of obesity in the first place may promote a more optimal metabolic physiology in terms of body weight regulation. Prevention may be more cost effective because strategies can be applied at the population level, whereas treatment is usually targeted at the level of the individual and therefore is generally more labor intensive and costly.

Treatment may be useful for obese, higher-risk children, and it can be effective.[33] However, some treatment options, especially drug therapy, may not always be applicable for children or appropriate for targeting at the population level. Treatment options also do not usually address the root cause of the problem, whereas preventive approaches can. Moreover, preventive strategies are unlikely to be harmful and may have secondary beneficial effects such as general disease risk reduction associated with improved diet and physical activity–related behaviors. Thus, a preventive strategy would provide a more favorable risk-benefit ratio for larger numbers of individuals.

Conclusion

Obesity in children is a serious and worsening problem that will have dramatic health effects on future generations. There is no evidence for a rapid shift in genotype to explain the rise in obesity, and there is very little evidence of a major metabolic defect. Potential defects in energy balance resulting in obesity are more likely explained by changes in environmental factors caused by changes in physical activity and diet. Thus, it is more likely that obesity is the end result of an interaction of a normal physiologic system with an obesity-promoting environment. This obesity-promoting environment is an obvious target for preventive interventions. The environmental modifications

that promote obesity in our society include an abundance of food coupled with a decreased requirement or stimulus for physical activity such that energy accumulates in the body. This type of adaptation to the environment is not an unusual phenomenon. These are normal physiologic adaptations that developed evolutionarily for survival but that are dysfunctional and counterproductive in the context of our highly mechanized and generally affluent life-style.

Obesity is a complex disease, and additional insight is required to design effective preventive programs. However, because of the seriousness of the problem, preventive efforts must begin immediately and continue to be modified as we learn more about the etiology of the obesity epidemic. This approach mirrors the strategy currently being used for other complex diseases associated with behavior, such as HIV, heart disease, and some cancers, especially those related to smoking behavior. For example, smoking prevention efforts have had major effects in reducing lung cancer, even though the mechanism for lung cancer is not fully elucidated.

Conclusion

The increased obesity in our society is likely the result of the modernization of our environment, in which there is an increased availability of food and a decreased requirement and stimulus for physical activity. Because obesity in children has long-lasting and serious negative health consequences that may affect the health of future generations, efforts must begin immediately to mount a unified and cooperative strategy to prevent obesity in children.

References

1. Figueroa-Colon R, Franklin FA, Lee JY, Aldridge R, Alexander L. Prevalence of obesity with increased blood pressure in elementary school-aged children. J South Med Assoc. 1997;90:806–813.
2. WHO Consultation on Obesity. Obesity: Preventing and Managing the Global Epidemic. WHO/NUT/NCD/98.1. Geneva, Switzerland: World Health Organization; 1997.
3. 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;99:420–426.
4. Charney E, Goodman HC, MacBride M, Lyon B, Pratt B. Childhood antecedent of adult obesity: do chubby infants become obese adults? N Engl J Med. 1976;295:6–9.

5. Stark D, Atkins E, Wolff DH, Douglas JWB. Longitudinal study of obesity in the National Survey of Health and Development. Br Med J. 1981;283:12–17.
6. Whitaker RC, Wright JA, Pepe MS, Seidel KD, Dietz W. Predicting obesity in young adulthood from childhood and parental obesity. N Engl J Med.1997;337:869–873.
7. Garn SM, Clark DC. Trends in fatness and the origins of obesity. Pediatrics. 1976;57:443–456.
8 Goran MI, Gower BA. Relation between visceral fat and disease risk in children. Am J Clin Nutr. 1999;70:149S–156S.
9. 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;128:608–615.
10. Dietz WH. Obesity in infants, children, and adolescents in the United States: identification, natural history, and aftereffects. Nutr Res. 1981;1:117–137.
11. Despres JP, Allard C, Tremblay A, Talbot J, Bouchard C. Evidence for a regional component of body fatness in the association with serum lipids in men and women. Metabolism. 1985;34:967–973.
12. Goran MI, Nagy TR, Treuth MT, Trowbridge C, Dezenberg C, McGloin A, et al. Visceral fat in Caucasian and African-American prepubertal children. Am J Clin Nutr. 1997;65:1703–1709.
13. Gower BA, Nagy TR, Trowbridge CA, Dezenberg C, Goran MI. Fat distribution and insulin response in pre-pubertal African-American and Caucasian children. Am J Clin Nutr. 1997;67:821–827.
14. Gower BA, Nagy TR, Goran MI. Visceral fat, insulin sensitivity, and lipids in prepubertal children. Diabetes. 1999;48:1515–1521.
15. Huang Z, Hankinson SE, Colditz GA, Stampfer MJ, Hunter DJ, Manson JE, et al. Dual effects of weight and weight gain on breast cancer risk. JAMA. 1997;278:1407–1411.
16. Moller H, Mellemgaard A, Lindvig K, Olsen JH. Obesity and cancer risk: a Danish record-linkage study. Eur J Cancer [A]. 1994;30A:344–350.
17. Thune I, Brenn T, Lund E, Gaard M. Physical activity and risk of breast cancer. N Engl J Med. 1997;336:1269–1275.
18. Slattery ML, Schumacher MC, Smith KR, West DW, Abd-Eighany N. Physical activity, diet and risk of colon cancer in Utah. Am J Epidemiol. 1998; 128: 989-999.
19. McTiernan A, Ulrich C, Slate S, Potter J. Physical activity and cancer etiology: associations and mechanisms. Cancer Causes Control. 1998;9:487-509.
20. McGinnis JM, Foege WH. Actual causes of death in the United States. JAMA. 1993;270:2207–2212.
21. Goran MI, Shewchuk R, Gower BA, Nagy TR, Carpenter WH, Johnson R. Longitudinal changes in fatness in white children: no effect of childhood energy expenditure. Am J Clin Nutr. 1998;67:309–316.
22. Heini AF, Switzerland B, Weinsier RL. Divergent trends in obesity and fat intake patterns: the American paradox. Am J Med. 1997;102:259–264.
23. Weinsier RL, Bracco D, Schutz Y. Predicted effects of small decreases in energy expenditure on weight gain in adult women. Int J Obes. 1993;17:693–700.
24. Weinsier RL, Hunter GR, Heini AF, Goran MI, Sell SM. The etiology of obesity: relative contribution of metabolic factors, diet and physical activity. Am J Med. 1999;105:145–150.
25. 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. JAMA. 1998;279:938–942.
26. Ross JG, Pate RR. The National Children and Youth Fitness Study. J Phys Educ Recr Dance. 1987;58:51–56.
27. Goran MI, Hunter G, Nagy TR, Johnson R. Physical activity related energy

expenditure and fat mass in young children. Int J Obes. 1997;21:171–178.
28. Kujala UM, Kaprio J, Sarna S, Koskenvou M. Relationship of leisure-time physical activity and mortality: the Finish twin cohort. JAMA.1998;279:440–444.
29. Bouchard C, Perusse L. Genetic aspects of obesity. Ann N Y Acad Sci.1993;699:26–35.
30. Hill JO, Peters JC. Environmental contributions to the obesity epidemic. Science. 1998;280:1371–1374.
31. Bogardus C, Lillioja S. Pima Indians as a model to study the genetics of NIDDM. J Cell Biochem. 1992;48:337–343.
32. Wolf AA, Colditz GA. Current estimates of the economic cost of obesity in the United States. Obes Res. 1998;6:97–106.
33. Epstein LH, Valoski A, Wing RR, McCurley MA. Ten-year follow-up of behavioral, family-based treatment for obese children. JAMA. 1990;264:2519–2523.

IV. Prevention of Childhood Obesity: Individual, Environmental, and Policy Issues

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Obesity has become the most prevalent nutritional disease in the U.S. Estimates from the Third National Health and Nutrition Examination Survey (NHANES III) suggest that 10%–15% of children and adolescents in the United States now have a body mass index (BMI) ≥ 95th percentile.[1] Furthermore, the prevalence of obesity in children has increased by more than 100% between 1980 and 1991.[1] Although the rate of the recent increase in prevalence appears comparable in black and white children and adolescents, the prevalence of obesity is higher in African American children. Obesity in children is not simply a cosmetic problem. For example, more than 60% of obese children and adolescents have at least one additional cardiovascular risk factor, and 20% have two or more.[2] Despite the prevalence of obesity and its associated morbidities, few specialized referral centers are available to treat the morbidly obese. Likewise, almost no research exists to inform the treatment of obesity in primary care settings.

The prevalence of obesity and the lack of adequate treatment protocols or clinics to provide care suggest that efforts should focus on both primary and secondary prevention. The most vital elements to successful prevention are the development of a better understanding of the critical periods in childhood for the development of obesity that persists into adulthood and of additional factors related to the onset of obesity that promote its persistence; identification of causal factors that represent reasonable targets for intervention; and the development of successful interventions. Each of these areas will be considered in turn.

Critical Periods in Childhood for the Development of Adult Obesity

Identification of periods of risk in childhood for the development of adult obesity and the morbidity that accompanies it will help identify the appropriate target of intervention efforts. Although childhood-onset obesity appears to account for less than one-third of adult obesity,[3] adult obesity that begins in childhood may be disproportionately severe.[4] There appear to be several critical periods in childhood for the development of adult obesity. These include prenatal experience, the period of "adiposity rebound," and adolescence.[5]

Experience from the Dutch famine of World War II suggests that children whose mothers were exposed to famine early in their pregnancies had an increased likelihood of obesity at the age of 18 years.[6] Although it seems increasingly clear that low-birth-weight infants have an increased risk of later diabetes, hypertension, and cardiovascular disease, it appears unlikely that low birth weight is accompanied by an increased risk of adult obesity. For example, infants who are small for gestational age tend to remain small.[7] The infants who appear at greatest risk for adult obesity appear to be those with increased birth weights.[8–10] Therefore, low birth weight infants who develop the adult symptoms usually associated with obesity may represent the "metabolically obese."[11] Even if low-birth-weight infants go on to become overweight adults, their contribution to the prevalence of adult syndrome X characterized by hypertension, hyperlipidemia and glucose intolerance is likely small. Our experience suggests that low-birth-weight infants (<2500 g) account for less than 10% of all births (Zuguo Mei, personal communication). Therefore, the maximal contribution to adult disease, or the population-attributable risk, will be less than 10%.

In contrast to low birth weight, the period of "adiposity rebound" may make a greater contribution to the prevalence of adult obesity. The period of "adiposity rebound" describes the period of development where the BMI reaches a nadir at approximately 6 years of age and subsequently begins to increase again.[12] Several studies have demonstrated that children with an early "adiposity rebound" have increased BMIs as adults.[13–15] Our calculations suggest that

the onset of obesity for approximately 30% of obese adults may occur at the time of "adiposity rebound". However, several caveats still apply. No clear evidence exists that children with early "adiposity rebound" are fatter than children whose BMI begins to accelerate later. Also, no studies have yet demonstrated that the increased BMI observed in adults who had early "adiposity rebound" is associated with increased fatness. Finally, the BMI at rebound may be a more powerful predictor of future BMI then the time of rebound. Therefore, the term "adiposity rebound" must be used tentatively until increased adiposity is demonstrated at the time of rebound and in adults with increased BMI who had early rebound. Furthermore, even if this period proves a period of true adiposity rebound, the biologic mechanisms that predispose to increased fatness remain unspecified.

Adolescence appears to be the final critical period in childhood, and may account for the remainder of adult obesity that begins in childhood. Data from the third Harvard Growth Study[16] showed that obesity present in adolescence was associated with increased early mortality in men and with increased morbidity in both men and women that appeared to be independent of the effect of adolescent weight on adult weight. Approximately 70% of adult obesity that begins before adulthood appears to begin in adolescence.

Few studies have examined the effects of childhood obesity on the likelihood of persistence into adulthood, after control for parental obesity. The one exception is a recent study by Whitaker et al.[17] This study demonstrated that the risk of obesity persisting into adulthood was not significantly increased among children 1-3 years of age. However, the risk was elevated in children older than 3 years. The greatest risk for adult obesity occurred in obese adolescents. Young children with two obese parents incurred a risk of adult obesity that approximated the risk associated with adolescent obesity.

These observations suggest that adolescents are at the highest risk of adult obesity, perhaps followed by children with "adiposity rebound". Thus, demonstration of increased fatness in children with early "adiposity rebound" and in adults whose increased BMI began in childhood must be a high research priority. Studies to identify the behaviors that promote either increased

food intake or reduced energy expenditure that lead to obesity in children and adolescents are essential to establish the behavioral focus of interventions designed to prevent adult obesity.

Potential Causes of Childhood Obesity

Causality encompasses causes of origin and causes of course. Although few studies of the causes of obesity have been performed, most have focused on causes of origin. For the purposes of this discussion, we assume that obesity includes a genetic component. It is thus appropriate to consider the genes associated with obesity as factors that influence susceptibility to obesity. For example, the genetic makeup of the US population did not change during the period 1980–1994, which was when the obesity epidemic was documented. Therefore, the rapid increase in the prevalence of obesity must reflect environmental changes during the same time period. The finding that most of the upward changes in the BMI of the population occurred in the 50% of the population with BMIs above the 50th percentile suggests either that the prevalence of the genes that influence susceptibility occurs in 50% of the population or that the environmental factors that produced the epidemic affected only half of the population.

Causes of Origin

Because food intake and the energy spent on activity represent the only discretionary elements of energy balance, obesity can result only from increased food intake or reduced activity, or both. Neither careful dietary assessments nor measures of energy expenditure are likely to detect the modest caloric imbalance necessary to initiate obesity. For example, comparisons of self-reported caloric intake with energy expenditure measured by doubly labeled water demonstrated that both obese and nonobese adolescents significantly underestimated energy intake.[19] Whether certain foods were less likely to be reported than others cannot be established. Similarly, the energy spent on activity measured by doubly labeled water may under- or overestimate nonbasal energy expenditure by as much as 30% (Michael Goran, personal communication). Because these measures lack the capacity to identify the small daily imbalances probably associated with the development of obesity,

techniques to measure patterns of diet or activity may be more appropriate strategies for identifying the behavioral targets of interventions.

Examination of the changes in diet and physical activity that coincided with the epidemic of obesity suggests a variety of potential candidate behaviors. Potential dietary factors are shown in the Table 4-1. In the mid-1970s, the advent of the microwave oven made it possible for children to select and prepare their own meals with ease. The erosion of parental control of food intake has also been promoted by the increase in food consumed outside the home. In the United States between 1977–1978 and 1987–1988, the percentage of food expenditures on food consumed outside the home increased from 25% to more than 35%.[20] More recent data suggest that the upward trend in food consumed outside the home is continuing and that food consumed outside the home contributes disproportionally to calorie and fat intake.[21] Per capita consumption of soft drinks increased by 63% between 1972 and 1992, from approximately 27 to 44 gallons per year.[20] Furthermore, soft drink consumption by 13–18-year-olds has more than doubled since 1977.[22]

The relationship of other eating patterns to obesity has not been examined. For example, 12% of middle school and high school students and 7% of elementary school students consume no food before school in the morning.[23] Anecdotal experience suggests that the hunger induced by meal skipping may impair the regulation of energy intake at the next meal, but careful studies of this problem have not been done. Sixteen

Table 4-1. Changes in Food Consumption that Coincided with the Obesity Epidemic in the United States

Use of the microwave oven
Changes in food consumed outside the home
7%–12% of older children and of 6–18-year-olds skip breakfast
Per capita consumption of soft drinks increased from 27 to 44 gallons per year from 1972 to 1992
12,000 new food products introduced annually (doubled since 1986)
16% of 2–19-year-olds meet none of the Food Guide Pyramid recommendations
1% of 2–19-year-olds meet all of the Food Guide Pyramid recommendations

percent of 2–19-year-olds met none of the Food Guide Pyramid recommendations, and only 1% met all of the recommendations. In addition, only 1% met the grain, fruits, and vegetables recommendation.[24] Almost 15% of food energy is derived from snacks for 6–19-year-olds.[20] Whether these food patterns contribute to obesity has not been thoroughly studied, either cross-sectionally or prospectively.

In 1997, more than 12,000 new food products were introduced. The number of new products introduced has doubled since 1986. The greatest number of new products were in the categories of candy/gum/snacks (2500), condiments (2600), bakery foods (1200) and beverages (1600).[25] A recent cross-sectional study[26] found a direct relationship between BMI and consumption of an increased variety of foods in the sweet, snack, condiment, entree, and carbohydrate categories, but an inverse relationship with BMI between the number of foods consumed in fruit and vegetable categories. These observations are consistent with the increased obesity observed in rats fed a varied and palatable diet[27] and the relationship between variety and meal intake in humans.[28] However, the cross-sectional nature of this study[28] does not allow causal inferences. Because overweight people have higher energy requirements, they require more food, and the variety of foods they consume may simply reflect their increased energy requirement.

One of the potentially most productive approaches to the causality of obesity is the examination of the factors that may operate to produce obesity at the time of "adiposity rebound". Assuming that this period represents a period of risk for the development of adiposity, it appears reasonable to investigate the factors responsible for the impairment of energy balance that may occur at this time. This period may represent the time when interactions regarding eating between parents and children become internalized. Several important studies emphasize that parental efforts to control the quantity of food that a child eats[29] and a parent's inability to control his or her own food intake[30] may be associated with impairment of the child's capacity to self-regulate food intake.

Several crucial concerns require clarification. First, these studies have not yet been replicated. Second, studies of the relationship of parent:child interactions and obesity to date have been cross-sectional, so it is not clear whether parental efforts

to control a child's food intake occurred because the child lacked the capacity to self-regulate or because parental control altered the capacity of the child to self-regulate. Third, because longitudinal studies have not been performed, it is not clear that the lack of self-regulation of food intake in experimental settings increases the likelihood of later obesity. Finally, we lack any information about how parental behaviors affect a child's activity level.

Extensive trend data are not available for physical activity or inactivity. Between 1991 and 1999, daily physical education classes for adolescents in ninth through 12th grades declined from 42% to 29% of schools.[31]

Television viewing has been associated with obesity in several studies,[32,33] and we have argued that this association may be causal.[32] Furthermore, reductions in television time appear to represent an effective strategy to induce weight loss in both clinic-based studies[34] and school-based studies.[35] However, data regarding the amount of time spent watching television by children and adolescents are incomplete. For example, the mean time spent watching television between the 1960s and 1990 increased from 2–3 hours per day[32] to 4.8 hours per day.[33] However, by 1990, 33% of 10–15-year-olds were watching more than 5 hours of television daily. Between 1990 and 1997, Neilsen data, which may not be comparable, suggest that the amount of time spent watching television by 12–17-year-olds declined from approximately 22 hours a week to 20 hours a week.[36,37] An important problem with these observations is that the estimates for television viewing by children and adolescents may not be valid (T. Robinson, unpublished observations).

No sound data regarding daily activity levels or patterns, or changes in daily activity levels or patterns, have been published. The increased frequency of working parents and the increased reliance on caretakers for after-school child care suggest that opportunities for free play have decreased during the last 15 years, but no objective data are available. Because the accuracy of self-reported activity levels is questionable, population-based studies of directly measured physical activity are essential.

The broad changes in diet and physical activity that have occurred coincident with the obesity epidemic offer a variety of testable hypotheses. The challenge is to examine the effect of

these patterns on incident obesity and to identify the barriers, opportunities, and mediating behaviors that promote both activity and inactivity.

Causes of Course

Most studies of childhood obesity have examined incident obesity. Very few have examined the factors that influence the remission of obesity or that maintain obesity after it has been established. At least one study has suggested that television viewing affects both the onset and the persistence of obesity in children.[33] Nonetheless, efforts to distinguish the causes that affect the onset of obesity from those that determine its course may suggest alternative approaches to treatment. For example, the observation that no amount of difficulty associated with the choice of sedentary behavior could induce very obese children to choose vigorous physical activity[38] suggests that efforts to induce weight loss in this subset should focus on dietary rather than physical activity interventions.

Prevention

The high incidence and prevalence of obesity indicate that effective strategies for both primary and secondary prevention must be developed. Primary prevention will target children and adolescents before obesity develops. Because most of the morbidity associated with obesity occurs in adulthood, secondary prevention should be directed at children and adolescents who are already overweight either to prevent further weight gain or, more importantly, to reduce weight to the normal range. Preventive efforts can occur in clinical, school, and community settings. Because so little is known about the dietary behaviors that induce or maintain obesity, and because physical activity reduces the comorbidities associated with obesity in adults, such as elevated blood pressure, glucose intolerance, and hyperlipidemia,[39] the promotion of increased physical activity represents a logical strategy across the lifecycle. Therefore, school and community efforts should probably focus on physical activity. Before school-based or community-based dietary interventions directed at obesity can be recommended for general implementation, a stronger scientific basis must be developed.

Environmental modification represents the most effective intervention for other prevalent nutritional diseases. For example, the addition of iodine to salt eliminated goiter in the United States; fluoridated water has substantially reduced dental caries; and enrichment of flour with folic acid may prove effective in reducing neural tube defects (levels of plasma homocysteine levels have apparently already decreased in response to this intervention).[40] Therefore, modifications of the school and community environments to promote physical activity to prevent obesity must be a high priority.

Clinical Settings

Neither primary nor secondary prevention in clinical settings has been carefully explored. The most appropriate food-related goal for parents would be to share responsibility for food intake with their children. Parents should be in control of what their children are offered and when, and children should be in charge of whether they choose to eat what is offered and how much they eat.[41,42] However, the manner in which appropriate food interactions are best promoted through anticipatory guidance has not yet been established, nor are there any data to indicate that the diets of children are improved in families where responsibilities for food intake are partitioned appropriately between parents and children. In addition, although the American Academy of Pediatrics has recommended counseling parents to limit television time to 1 to 2 hours per day,[43] no studies have yet explored the most appropriate approach to such counseling or the impact of such counseling on the activity level, food preferences, or incident obesity of children.

Secondary prevention in primary care settings has also received limited attention. Although an expert committee recently released guidelines for the treatment of obesity in primary care settings,[44] no studies have been done to demonstrate that the implementation of these guidelines produces weight loss. A study currently in progress to assess the knowledge and attitudes of pediatricians, nurse practitioners, and pediatric nutritionists should help identify the gaps that should become the focus of education and training.

A network of tertiary care referral centers also represents a high priority for the most severely obese children and adolescents, and efforts to establish such a network have been initiat-

ed by the Centers for Disease Control and Prevention. The function of such a network will be to provide aggressive care for children and adolescents with morbid complications of obesity, to share solutions to common problems, and to educate medical students, house staff, and primary care providers.

Schools

Schools represent a logical focus of efforts to prevent obesity. Most children attend school, many receive one or two meals at school daily, and schools have the resources to support physical activity. It is unclear at present whether schools are part of the problem, part of the solution, or both. For example, although schools have recently instituted closer oversight of the caloric and fat content of school meals, items purchased a la carte fall outside the nutrient standards applied to the school lunch menu. Furthermore, vending machines exist in many schools, and "pouring contracts" with major beverage-producing corporations offer an additional incentive for schools to make vending machines available to their students.[45] Items sold a la carte in the cafeteria and in vending machines tend to be high-caloric-density foods, such as ice cream and cookies. Furthermore, many schools depend on the revenue generated by a la carte foods in the cafeteria and vending machines to support their lunch programs.

The contribution of foods consumed at school to the usual dietary intake has been studied, but the role that such foods may play in the onset of obesity is unknown. However, the observation that competitive pricing of low-fat snacks through subsidization promoted their increased consumption[46] offers a reasonable strategy for altering the consumption of high-caloric-density foods. After the subsidy of the low-fat alternatives was discontinued, consumption returned to former levels. These data suggest that a more permanent alternative to price subsidies might be to charge more for foods of increased caloric density so that lower-calorie foods cost less.

Schools also offer an important and protected site for physical activity. The decline in daily physical education classes represents an important opportunity for interventions. Data that demonstrate that participation in physical activity improves learning would provide important support for the

argument to restore physical education programs in schools, but we are not aware of any studies that have rigorously examined this association. In schools with physical education programs, techniques to increase the amount of active time spent in physical education classes will enhance the effectiveness of physical activity.

Schools also provide the opportunity for school-based interventions. Although the highly popular Child and Adolescent Trial for Cardiovascular Health (CATCH) produced significant institutional and individual behavioral changes, such changes were not accompanied by changes in fatness, blood pressure, or blood lipids.[47] However, a more recent intervention demonstrated that a middle-school curriculum integrated within existing classes that focused on reduced fat and increased fruit and vegetable intake, increased moderate and vigorous physical activity, and reduced inactivity produced weight loss in girls.[35] African-American girls appeared particularly responsive to this approach. This intervention is now being expanded to a larger school system.

Community

Dietary data that link obesity to specific food practices will be necessary for the development of effective community-based approaches to change food consumption. If the data are credible, one could argue that the recent increases in obesity despite the widespread reduction in dietary fat consumption suggest that the reductions in dietary fat represent a community-level intervention that has failed. Nonetheless, the reductions in dietary fat that have been achieved indicate that major cultural shifts in food consumption are possible.

Modification of community environments to promote physical activity offers a major challenge for the new century. The term “connectedness” is used to define the number of alternatives available for travel from one location to another. In older communities, a high degree of connectedness exists; such communities already have the infrastructure to support walking, such as sidewalks, traffic lights, and pedestrian crossing signals. In many of these communities, perceived neighborhood safety constitutes a major barrier. In contrast, many newer communities lack sidewalks and community shopping

areas accessible to pedestrians. Although studies suggest that increased pedestrian traffic occurs in communities with existing infrastructure,[48–50] no studies have compared physical activity in highly connected communities with physical activity in communities affected by urban sprawl.

These observations suggest a rich source of potential studies. For example, ensuring neighborhood safety may prove a less challenging, less costly, and more effective approach to increasing physical activity than the development of a connected infrastructure in newer communities. However, strategies to promote common recreation areas or facilities to support physical activity, and a promotional campaign designed to foster their use, may be less challenging, less costly, and more effective in newer communities affected by urban or suburban sprawl. They may also foster the development of new alliances. For example, community coalitions may more readily emerge from connected communities, or connected communities may foster the development of community coalitions that enhance livability. Transportation agencies and the Environmental Protection Agency are promoting reduced reliance on the automobile and the development of alternative forms of transportation. These developments might promote alliances around the development of pedestrian-friendly communities or the use of bicycles as transportation. Such developments will also foster increased levels of physical activity.

Conclusion

Effective prevention will depend on a sound knowledge base, development of an effective social strategy, and the political will to change policy.[51] Although the specific behaviors that lead to obesity have not been well defined, the environmental changes that have accompanied the epidemic of obesity provide an initial focus for primary and secondary preventive efforts. Perhaps the first barrier to effective prevention is the lack of recognition that obesity represents more than a cosmetic problem and that resources must be mobilized to explore the physical and dietary behaviors that cause this nutritional disease. Increased attention to the medical, emotional, and economic costs of obesity may increase social pressure to develop effective primary and secondary prevention programs. The research outlined above

will be required to develop an effective scientific base for interventions. Because obesity has not yet become a public health priority, however, the development of the political will to direct the necessary resources to the prevention of obesity represents a significant challenge.

References

1. Troiano RP, Flegal KM. Overweight children and adolescents: description, epidemiology, and demographics. Pediatrics. 1998;101:497–504.
2. 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: 1175-1182.
3. Braddon FEM, Rodgers B, Wadsworth MEJ, Davies JMC. Onset of obesity in a 36-year birth cohort study. Br Med J. 1986;293:299–303.
4. Rimm IJ, Rimm AA. Association between juvenile onset obesity and severe adult obesity in 73,532 women. Am J Public Health. 1976;66: 479–481.
5. Dietz WH. Critical periods in childhood for the development of obesity. Am J Clin Nutr. 1994;59:955–959.
6. Ravelli G-P, Stein AZ, Susser MW. Obesity in young men after famine exposure in utero and early infancy. New Engl J Med. 1976;295:349–353.
7. Strauss RS, Dietz WH. Effects of intrauterine growth retardation in premature infants on early childhood growth. J Pediatr. 1997;130:95–102.
8. Curhan GC, Willett WC, Rimm EB, Spiegelman D, Ascherio AL, Stampfer MJ. Birth weight and adult hypertension, diabetes mellitus, and obesity in US men. Circulation. 1996;94:3246–3250.
9. Seidman DS, Laor A, Gale R, Stevenson DK, Danon YL. A longitudinal study of birth weight and being overweight in late adolescence. AJDC. 1991;145:782–785.
10. Whitaker RC, Dietz WH. The role of the prenatal environment in the development of obesity. J Pediatr. 1998;132:768–776.
11. Ruderman NB, Schneider SH, Berchtold P. The "metabolically obese," normal weight individual. Am J Clin Nutr. 1981;34:1617–1621.
12. Rolland-Cachera M-F, Dheeger M, Guilloud-Bataille M. Tracking the development of adiposity from one month of age to adulthood. Ann Hum Biol. 1987;4:219–229.
13. Siervogel RM, Roche AF, Guo S, Mukherjee D, Chumlea WC. Patterns of change in weight/stature2 from 2 to18 years: findings from long-term serial data for children in the Fels Longitudinal Growth Study. Int J Obes. 1991;15:479–485.
14. Prokopec M, Bellisle F. Adiposity in Czech children followed from 1 month of age to adulthood: analysis of individual BMI patterns. Ann Hum Biol. 1993;20:517–525.
15. Whitaker RC, Pepe MS, Wright JA, Seidell KD, Dietz WH. Adiposity rebound and the risk of adult obesity. Pediatrics [electronic]. 1998;101:1–6.
16. 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. New Engl J Med. 1992;327:1350–1355.
17. Whitaker RC, Wright JA, Pepe MS, Seidel KD, Dietz WH. Predicting obesity in young adulthood from childhood and parental obesity. New Engl J Med. 1997;337:869–873.
18. Troiano RP, Flegal KM. Overweight children and adolescents: description, epidemiology, and demographics. Pediatrics. 1998;101 Suppl:497–504.

19. Bandini LG, Schoeller DA, Cyr HN, Dietz WH. Validity of reported energy intake in obese and non-obese adolescents. Am J Clin Nutr. 1990;52:421–425.
20. Third Report on Nutrition Monitoring in the United States: Executive Summary. Washington, DC: US Government Printing Office; 1995.
21. Lin B-H, Frazao E, Guthrie J. Away-from-Home Foods Increasingly Important to Quality of American Diet. Agriculture Information Bulletin 749. Washington, DC: US Department of Agriculture, 1999.
22. Center for Science in the Public Interest. Liquid Candy [corrected version]. Washington, DC: Center for Science in the Public Interest; 1998.
23. Devaney B, Stuart E. Eating Breakfast: Effects of the School Breakfast Program. Alexandria, VA: USDA Food and Nutrition Service; 1998.
24. Munoz KA, Krebs-Smith SM, Ballard-Barbash R, Cleveland LE. Food intakes of US children and adolescents compared with recommendations. Pediatrics. 1997;100:323–329.
25. New Products Slip a Trifle in '97. Food Institute Report. Fair Lawn NJ, Am Inst Food Distrib Inc. 1998.
26. McCrory MA, Fuss PJ, McCallum JE, Yao M, Vinken AG, Hays NP, et al. Dietary variety within food groups: association with energy intake and body fatness in men and women. Am J Clin Nutr. 1999;69:440–447.
27. Rolls BJ, van Duijvenvoorde PM, Rowe EA. Variety in the diet enhances intake in a meal and contributes to obesity in the rat. Physiol Behav. 1983;31:21–27.
28. Rolls BJ, Rowe EA, Rolls KT, Kingston B, Megson A, Gunary R. Variety in a meal enhances food intake in man. Physiol Behav. 1981;26:215–221.
29. Johnson SL, Birch LL. Parents' and children's adiposity and eating style. Pediatrics. 1994;94:653–661.
30. Cutting TM, Fisher JO, Grimm-Thomas K, Birch LL. Like mother, like daughter: familial patterns of overweight are mediated by mothers' dietary disinhibition. Am J Clin Nutr. 1999;69:608–613.
31. Department of Health and Human Services Office of Public Health and Science. Healthy People 2010 Objectives: Draft for Public Comment. Washington, DC: Department of Health and Human Services; 1999.
32. Dietz WH, Gortmaker SL. Do we fatten our children at the TV set? obesity and television viewing in children and adolescents. Pediatrics. 1985;75:807–812.
33. Gortmaker SL, Must A, Sobol AM, Peterson K, Colditz GA, Dietz WH. Television viewing as a cause of increasing obesity among children in the United States, 1986–1990. Arch Pediatr Adolesc Med. 1996;150:356–362.
34. Epstein LH, Valoski AM, Vara LS, McCurley J, Wisniewski L, Kalarchain MA, et al. Effects of decreasing sedentary behavior and increasing activity on weight change in obese children. Health Psychol. 1995;14:109–115.
35. Gortmaker SL, Peterson K, Wiecha J, Sobol AM, Dixit S, Fox MK, et al. Reducing obesity via a school-based interdisciplinary intervention among youth: Planet Health. Arch Pediatr Adolesc Med. 1999;153:409–418.
36. AC Nielsen. 1990 Report on Television. Northbrook, IL: Neilsen Media Research; 1990.
37. AC Nielsen. 1997 Report on Television. Northbrook, IL: Neilsen Media Research; 1997.
38. Epstein LH, Smith JA, Vara LS, Rodefer JS. Behavioral economic analysis of activity choice in obese children. Health Psychol. 1991;10:311–316.
39. National Heart, Lung, and Blood Institute. Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults. Bethesda, MD: National Heart, Lung, and Blood Institute; 1998.
40. Jacques PF, Selhub J, Bostom AG, Wilson PWF, Rosenberg IH. The effect of folic acid fortification on plasma folate and total homocysteine concentrations.

New Engl J Med. 1999;340:1449–1454.
41. Satter E. How to Get Your Kid to Eat but Not Too Much. Palo Alto, CA : Bull Publishing Co; 1987.
42. Dietz WH, Stern L, editors. A Guide to Your Child's Nutrition. New York, NY: Villard Books; 1999.
43. Committee on Communication. Children, adolescents, and television. Pediatrics. 1995;96:786–787.
44. Barlow SE, Dietz WH. Assessment and treatment of obesity in children and adolescents: recommendations of an expert committee. Pediatrics [electronic]. 1998;102:e29.
45. Kaufman M. Health advocates sound alarm as schools strike deals with Coke and Pepsi. Washington Post. 1999 March 23; p Z12.
46. French SA, Jeffery RW, Story M, Hannan P, Snyder MP. A pricing strategy to promote low-fat snack choices through vending machines. Am J Public Health. 1997;87:849–851.
47. Webber LS, Osganian SK, Feldman HA, Wu M, McKenzie TL, Nichaman M, et al. Cardiovascular risk factors among children after a 2 1/2-year intervention: the CATCH study. Prev Med. 1996;25:432–441.
48. Moudon AV, Hess PM, Snyder MC, Stanilov K. Effects of site design on pedestrian travel in mixed-use, medium-density environments. Transport Res Record. 1997:no. 1578;48–55.
49. Sallis JF, Bauman A, Pratt M. Environmental and policy interventions to promote physical activity. Am J Prev Med. 1998;15:379–397.
50. Brownson RC, Housemann RA, Brown DR, Jackson-Thompson J, King AC, Malone BR, et al. Promoting physical activity in rural communities: walking trail access, use, and effects. Am J Prev Med. 2000;18:235-241.
51. Atwood K, Colditz GA, Kawachi I. From public health science to prevention policy: placing science in its social and political context. Am J Public Health. 1997;87:1603–1606.

V. Summary

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The Obesity Epidemic

From 10% to 15% of U.S. children and adolescents now have a body mass index (BMI) > 95th percentile. Furthermore, the prevalence of obesity in children increased by more than 100% between 1980 and 1991. In reality, there is no single cause that can be remedied to fix this complex problem.

Another challenge to effective intervention is the lack of a unified strategy that incorporates intervention methods proven to be effective. Research has demonstrated some promising models for intervention in schools, in communities, and in the context of health care, but much remains to be done to demonstrate the feasibility and effectiveness of these models when applied beyond the research setting. Interventions are also hampered by a low level of awareness of the child obesity problem and by the low priority it is given in communities and at the state and national levels. Because of these challenges, the future determinants of success are likely to lie in finding effective ways to raise awareness, build broad-based partnerships, and develop a unified strategy for treatment and prevention.

Intervention Strategies

Interventions for the prevention and treatment of obesity in childhood and adolescence have largely been conducted in four broad domains: schools, clinics, communities, and environment/policy. The school represents the most frequent intervention setting because school-based studies can address obesity as

part of a broad-based cardiovascular disease prevention program. Although such studies show inconsistent outcomes related to adiposity changes, encouraging findings have been noted for changes in the behavioral intermediary factors of physical activity and nutrition. School-based studies that target obesity as the primary outcome have been more successful than multiple-risk-factor studies, owing to improvements in intervention techniques and assessment methods. Community-based interventions hold promise for obesity prevention and treatment, although little is known about their effectiveness. Clinical interventions also show promise for successful interventions, with current work suggesting that efforts to reduce sedentary activity (rather than focus solely on promoting increased activity) may be the most effective treatment. Environment and policy interventions represent a vast potential for a positive effect on obesity in youth, but their effects are unknown. Public infrastructure changes, such as the construction of facilities that promote physical activity and legislative strategies to promote healthful choices, have been discussed recently as having a potential effect. The treatment and prevention of obesity in youth must be conceptualized as a heterogeneous condition, and interventions must be tailored to provide the greatest likelihood of success, whatever the intervention domain.

Obesity Prevention

Data suggest that prevention is the key to controlling the current epidemic of in children. Current evidence suggests that obesity is the end result of the interaction of a normal physiologic system with an obesity-promoting environment that includes an abundance of food coupled with a decreased requirement or stimulus for physical activity. Because obesity is a complex disease, additional insight related to specific etiology is required to design the most effective preventive programs. However, because of the seriousness of the problem, preventive efforts must begin immediately and continue to be modified as we learn more about the specific causality of the alarming increase in obesity prevalence. This approach mirrors the strategy currently being used for other complex diseases

associated with health behavior, such as HIV, heart disease, and some cancers, especially those related to smoking behavior.

Challenges and Opportunities

Several key elements will be necessary to address the prevention and management of childhood obesity in the future. One of these key elements is gaining a better understanding of the critical periods in childhood during which obesity may develop. Current research indicates that the most critical periods for the development of obesity are the period of "adiposity rebound" and during adolescence. These periods appear to account for approximately 30% and 70%, respectively, of childhood obesity that persists into adulthood. More research is needed to better define these critical periods for obesity development. Research is also needed to identify the factors that promote the onset and persistence of obesity during these vulnerable periods. Once better defined, these causal factors could serve as potential targets for the development and testing of intervention strategies.

Because genetic factors influencing obesity cannot have changed appreciably in a short time span, the recent marked increase in obesity can only have resulted from increased food intake or reduced activity, or both. The challenges now are to: (a) Establish the effect of these behavioral patterns on incident and persistent obesity; (b) Identify the underlying factors and mediating behaviors that lead to patterns of food intake, activity, and inactivity; and (c) Develop the clinical, environmental, and policy approaches necessary to prevent the continuation or worsening of the obesity epidemic.

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