In recent months Per Björntorp and his colleagues in Gothenburg announced the surprising results of a 13-year prospective study of 975 randomly selected male patients with obesity: they found that the rate of new events of coronary heart disease (CHD) was directly and importantly related to the regional distribution of adiposity — that is, the more male the type of obesity, the higher the CHD rate. And what is male obesity? As first noted by Vague in 1953, males tend to grow fat in the abdominal region, females in the hips and buttocks. Analysis of the Swedish test population (all males) showed that the larger the ratio of waist/hip circumference, the higher the CHD rate (the rate in the highest quintile being more than twice that in the lowest). A full report of the basic methodology and characteristics of the test population can be found in the October 1983 issue of the Journal of Clinical Investigation; the CHD data were presented at the October 1983 meeting of the 4th International Congress on Obesity in New York.

The observations of Kissebah and his colleagues are relevant to these new findings. In their recently published metabolic studies of obesity in 80 premenopausal women, they showed that there is an inverse relationship between the plasma sex hormone-binding globulin capacity and the waist/hip girth ratio (WHG), but a direct relationship of WHG with the percentage of free plasma testosterone. In other words, women who show male-type obesity and the associated metabolic abnormalities of glucose intolerance and insulin resistance have high levels of androgens in the blood, relative to estrogens.

Thus, it now becomes reasonable to speak of "androgenic obesity" as a CHD risk factor. We must wonder why males gather their excess adiposity so characteristically in the belly: "executive spread" and "beer-belly" are typical white collar and blue collar descriptions of paunchy men. What is it about the male hormonal balance that leads to the hypertrophy of adipose cells within the abdomen, and of femaleness that leads to fat cell hyperphasia in the periphery? Lipolysis of omental fat is more inducible by epinephrine than peripheral fat; does an increased mobilization of free fatty acids to the liver via the portal vein play some role in androgenic obesity in VLDL production and hyperglyceridemia? Although these questions are of central interest to endocrinologists and students of obesity, they apparently will prove to be equally interesting to investigators in the field of arteriosclerosis.

Accordingly, it is timely to readdress the public health hazards of obesity and to ask ourselves the price of doing little or nothing to acquaint the public with the costs of obesity. Obesity is more common today in the industrialized Western world than ever before in recorded history. However obesity is defined (and there are no clear-cut boundaries between obesity, overweight, and healthful weight), it affects more men, women, and children than ever before. Nevertheless, because it is so common and so difficult to reverse, the public seems to accept it as one of those inevitable consequences of the higher standards of living enjoyed by populations with increasing life expectancies.

If obesity could be reversed, that is, if overweight people could be thinned and kept at lower total body weights, what benefits can be promised? Diabetes (Type II) and hypertension, two of the major risk factors for new events of CHD and stroke, would be reduced in incidence and severity. Gallstone disease, the most common reason for major surgery, is closely associated with overweight; in turn, obesity is
associated with increased rates of cholesterol synthesis in the body and with increased flux of cholesterol through the biliary tree. These metabolic changes (as well as glucose intolerance and insulin resistance) are reversed when body weight is reduced. The orthopedic problems of the obese could be avoided, and the increased risks of surgery in the obese could be reduced.

There is sound scientific evidence to support all these claims for benefits to obese persons who reduce their body weight and keep it reduced. This statement of fact raises an important question, however. Is there evidence that prevention of obesity can produce the same health benefits? That is to say: if the general population never becomes obese, will there be less diabetes, hypertension, and gallstone disease? However strong the logic supporting such a conclusion, I cannot cite evidence to support or refute this hypothesis; I know of no population study in which, prospectively, the incidence of these diseases has been tabulated in groups held at physiologically desirable weights for comparison to the incidence in other groups not so managed. Thus, any claim that prevention of obesity confers definite health benefits rests on logic, on retrospective epidemiologic evidence, and on the clinical observation that the reduction of overweight is beneficial for diabetics and hypertensives.

This important distinction between the benefits we know will accrue if obesity is successfully managed, and the benefits we believe will follow if obesity is prevented, cannot be passed over lightly. If the public is faced with a national health program to control body weight, people must be indoctrinated in the implications of this distinction. Although the public must not be given false promises, the success of such a program will depend on persuading the public of the strength of the logic. These caveats aside, I suggest that one of the strongest reasons for urging the population to remain lean in adult life is the difficulty that every overweight person experiences in losing weight and then maintaining a lean weight indefinitely thereafter. Success in the prevention of obesity seems more likely than the hope that the obese, rid of their excess weight, will long succeed in remaining lean. The moral must be: convince the public that it must not become obese.

Can obesity be prevented? I truly believe it can be, despite the fact that our track record on this public health issue has been extremely disappointing. The place to begin, I believe, is in the maternity clinic where expectant mothers must be persuaded that fatness in infancy is probably not desirable. The choice of food offered to infants and toddlers will, I believe, affect their long-term attitudes toward food. Since heavily sweetened foods are of no special benefit to a child, parents should be encouraged to avoid them in their child's diet. The result might be that the child accumulates less body fat during the early years and is less inclined to choose heavily sweetened foods as an adult.

With lean children entering kindergarten, the educational process must begin and continue through the school years; new ways to teach physiology and hygiene must be devised to assure optimal eating habits during all the years of growth. Equal attention must be paid to the health benefits of aerobic exercise and to an enjoyment of sports that can be continued throughout adult life. Since obesity represents a metabolic imbalance that is as much a matter of sloth as of gluttony, the population must learn how to balance energy input with an equal output of energy; that calories are calories, no matter what the source; that liquor, beer, and wine are rich in calories; that eating specific foods does not lead to overweight, only the total daily intake of energy; that most occupations pursued by adults are increasingly sedentary with age and that caloric intakes must, therefore, decrease with age. Running to catch a bus is not an adequate amount of daily exercise; a minimal exercise program must include at least two 20- to 30-minute sessions each week in which the heart rate is sustained at the high level set by exercise physiologists for each individual. The public can learn that a skinfold thickness of an inch or more in the flank area is palpable evidence of obesity.

A concerted attack on obesity has never been adequately mounted by any nation, to my knowledge. While it is essential to have and hold the explicit support of the medical profession in designing this attack, it would be unwise today to depend on physicians for its execution. Until the day when physicians are trained to preserve health and not simply to attend the sick, we must rely on the professionals in this field, the nutritionists. Increasingly in the U.S., doctors are referring patients for
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nutritional guidance to nutritionists, both in private practice and in institutional settings. This professional group can supervise the education of school teachers in those matters that relate to energy balance as well as to nutritional deficiencies; this group also constitutes the strongest counterbalance to the advocacy of the fad diets that seem so appealing to dieters. The medical profession must assure that adequate numbers of nutritionists and exercise physiologists are brought into these essential service roles. It is equally important that the hazards of bulimia and anorexia nervosa be recognized and addressed through education of the young, especially teen-aged girls.4

In conclusion, I submit that an effective attack on the public health hazards of obesity requires the joint efforts of physicians, nutritionists, sociologists, physiologists, and teachers. It must be supported not only by the government but also by the food industry. Indeed, it is my sincere belief that no nutritional program can offer such enormous benefits today as the proper control of body weight.

References

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Obesity and coronary heart disease. New dimensions.
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