FACTORS IN OBESITY: CURRENT VIEWS

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This discussion of factors in obesity will address three major topics: genetic determinants of obesity, the distribution of body fat in obesity, and the influences of social factors, particularly socioeconomic status (SES).

Any consideration of the role of social factors in the determination of obesity today must consider what we know about the role of genetic determinants of obesity. This information is of recent origin and it is necessary to put it into context so that genetic influences are neither exaggerated nor minimized.

GENETIC DETERMINANTS OF OBESITY

For some time, the existence of numerous forms of genetic obesity in animals and the ease with which adiposity can be produced by the selective breeding of farm animals have suggested that genetic factors may play a similarly important role in human obesity. The stunning advances in our knowledge during the past decade have made it clear that genetic factors do indeed play an important role in human obesity.

A previous study, utilizing the classic twin method, estimated very high levels of heritability (the percentage of variance accounted for by genetic influences) for the body mass index (BMI–weight (kg)/height^2 (m)). In this study, heritability was estimated at approximately 80% (1). Even studies of identical twins separated at birth, a method that avoids some of the bias inherent in classic twin studies, estimated heritability at 66% (2). These studies are still widely cited, but there is growing consensus that they overestimate the influence of heredity.

The results of adoption studies and of complex segregation analysis agree on approximately 33% heritability of BMI (3, 4), a value now viewed as a more reasonable estimate than that of the twin studies. Genetic influences appear to play a more important role in determining the distribution of body fat, with a particularly strong influence on the critical visceral fat depot that is described in the next section.

The recent entry of molecular genetics into the study of obesity, with the identification of mutations of two genes (5–7) that cause obesity in mice, promises to greatly advance our understanding of the genetic determinants of human obesity. Nevertheless, if, as seems likely, the heritability of human obesity is no more than 33%, then 66% of the variances in BMI must be environmental. Thus, although human obesity develops within genetic constraints, environmental determinants play an enormously important role in its development.

Genetic and environmental determinants of obesity are not in conflict. It is not a question of genes or environment or of genes versus environment, but of genes and environment;

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neither acts alone to determine the clinical outcome. This outcome is determined instead by the combination of genetic vulnerability and adverse environmental events (8). This combination is diagrammed in Figure 1, in which the small inner circle represents those persons who are genetically predisposed to a disorder. The wedge represents adverse environmental conditions to which these individuals may be exposed. The model indicates that only those genetically predisposed persons who are exposed to adverse environmental conditions are clinically affected, as in the case of obesity.

**DISTRIBUTION OF BODY FAT**

In the past, various classifications of obesity have been based upon the character of the predominant adipose tissue, the severity of the obesity, and the age of onset. All of these classification methods are still used to some extent, but they have been superseded to a considerable degree by the distribution of body fat.

Interest in the distribution of body fat was aroused in the early 1980s by the finding that persons whose fat was located primarily in the upper part of the body suffered far higher mortality and morbidity from ischemic heart disease than did persons whose fat was located primarily in the lower part of the body (9). Body fat distribution is measured clinically by the waist:hip ratio, calculated from the waist circumference halfway between the lower rib margin and the iliac crest and the hip circumference at the level of the greater trochanter. Upper body obesity is defined as a waist:hip ratio of more than 1.0 for men and 0.8 for women. Risk, however, is directly proportional to the size of the waist:hip ratio, independent of gender; the greater mortality and morbidity of men is a function of their greater waist:hip ratio.

Although the waist:hip ratio is still the most widely used clinical measure of body fat distribution, a major refinement has been introduced by imaging techniques that have shown that essentially all of the risk of upper body obesity is conveyed by the visceral fat depot, within the abdominal wall (9). This finding has greatly expanded our understanding of the complications of obesity and has provided a rationale for the metabolic cascade that mediates many of the steroid hormones, which increases free fatty flux and leads to decreased hepatic insulin clearance as well as hyperinsulinemia, hyperlipidemia, hypertension, and ultimately, cardiovascular disease.

**FIGURE 1. Combined effect of genetic predisposition to a disorder and exposure to adverse environmental conditions on clinical outcomes.**

**SOCIAL DETERMINANTS OF OBESITY**

The fact that genetic influences account for only one-third of the variance in body weight means that the environment exerts a profound influence. One measure of the extent of this influence is the dramatic increase (33%) of the prevalence of obesity in the United States during the past decade (10). Unfortunately, our understanding of these important environmental determinants is limited and systematic studies of the topic are rare. Most of these studies focus on socioeconomic status.
An influential report in 1965 delineated the influence of SES on obesity. In a stratified sample of 1660 persons in the Midtown Manhattan Study, as shown in Figure 2, obesity was six times more prevalent among women of lower SES than among those of upper SES (11). These results were important because of the strength of the relationship between current SES and obesity, but the study went further. It measured parental SES when the respondents were eight years old, the so-called "SES of origin," and assessed its relationship to the prevalence of obesity. As shown in Figure 2, SES of origin was related to the prevalence of obesity almost as strongly as was the respondents' own SES. The respondents' obesity could hardly have influenced their SES of origin, strongly suggesting that SES of origin was a determinant of obesity in this population at least. Note, however, that the prevalence of obesity in SES of origin was lower than current SES among persons of lower SES and was higher among persons of upper SES. These differences indicate that, in addition to SES influencing obesity, obesity also influences SES, in that it leads to a decrease in SES. Thus, the prevalence of obesity was nearly twice as high (22%) among women who fell in social class as it was (12%) among those who rose in social class. The relationship of social factors and obesity was far less important among men than among women. A recent prospective study had provided powerful support for the view that obesity has a deleterious influence on social functioning: women who had been obese in adolescence suffered significant social disability in adult life (12).

The Midtown Manhattan Study also revealed another social factor related to the prevalence of obesity—acculturation to the U.S. The prevalence of obesity decreased in a monotonic manner over four levels of increasing acculturation—foreign-born, second generation in the United States, one parent second generation and, finally, third and later generations.

These findings have been confirmed in no fewer than 54 studies in developed countries, which found a strong inverse relation between SES and obesity among women (13). Furthermore, two prospective longitudinal studies from England have provided strong confirmation that SES is a determinant of obesity. They have shown that both girls and boys born into a lower SES were more overweight as adults than those born into a higher SES (14, 15).

The simplest explanation for these findings is that the relation of SES and obesity is bidirectional; SES determines the prevalence of obesity and obesity leads to a decline in SES. There is, however, a complication. Another factor or factors may influence both SES and obesity (16). An example of such a common factor is heredity. As we have noted, genetic factors influence obesity. Less well-known is the fact that genetic factors may also influence SES. Thus, studies of Danish adoptees have revealed that the SES of biological parents influences the SES of their offspring, even though they have had no personal contact with them (17). A path analysis helps to explain this surprising finding. It shows that the influence of SES on obesity is mediated via the intelligence quotient (IQ). Biological parents influence the IQ of their children and IQ, in turn, influences obesity: the higher the IQ, the lower the prevalence of obesity. It is noteworthy that this influence was present even when the SES of the adoptee was controlled. It ap-

**FIGURE 2.** Prevalence of obesity among women, by own socioeconomic status and socioeconomic status of origin.

![Graph showing prevalence of obesity by socioeconomic status.](source: Goldblatt PB, Moore ME, Stunkard AJ. Social factors in obesity. JAMA 1965;192:1039–1044.)
pears that the relationship between SES and obesity is indeed complex (16).

The relationship between SES and obesity among women in developed societies is not found regularly among men or children. Among these groups, a significant percentage manifest either no relationship between SES and obesity or a direct relationship: the higher the SES the greater the prevalence of obesity. When we turn from developed to developing societies, there is a complete reversal in the relationship between SES and obesity. In every developing society that has been studied, there is a direct and often very strong relationship between SES and the prevalence of obesity in men, women, and children (13).

What is the reason for this striking difference in the relationship between SES and obesity in developed and developing societies? Four factors may mediate the inverse relationship between SES and obesity among women in developed societies, particularly factors that control obesity among women of upper SES.

One influence, and probably the most important one, constraining the prevalence of obesity among women of upper SES in developed societies is dieting and dietary restraint. Women of higher SES diet more often than do women of lower SES, have greater access to resources that facilitate dieting, have greater knowledge of nutrition, and are more committed to the view that slimness is desirable and, therefore, are more motivated to achieve it.

A second direct influence on the control of obesity among upper SES women is their greater physical activity, derived from their greater leisure time and greater opportunity for recreational exercise. Interestingly, pathologically increased physical activity characterizes the young women of upper SES whose dieting leads to anorexia nervosa.

A third factor mediating the inverse relationship between SES and obesity among women is social mobility. As noted in the Midtown Manhattan Study, the prevalence of obesity is nearly twice as high among women who fall in social class than it is among women who rise in it (11). One of the longitudinal studies in Britain confirmed and extended this finding. It showed that obesity was significantly less prevalent (5%) among women who rose in social status than among those (11%) who remained in their social class of origin (14). As is the case with SES itself, social mobility plays a far less important part in determining the prevalence in obesity among men.

A fourth factor influencing the relationship between SES and obesity is heredity. As we have seen, studies of Danish adoptees have revealed a significant influence of the SES of biological parents upon the prevalence of obesity in their children, with whom they have had no personal contact. This influence appears to be genetically transmitted via IQ.

The strong direct relationship between SES and obesity in developing societies has a more straightforward rationale than does the inverse relationship in developed societies. The low prevalence of obesity in developing societies appears to be due to a lack of food, coupled with cultural values favoring fat bodies. Obesity may be a sign of health and wealth in these societies, the opposite of its significance in developed societies. In such societies, the biological propensity to store fat is associated with, and may even influence cultural evolution that selects “fatness” as a valued trait. Among most of the 58 traditional cultures for which information is available, “plumpness” is viewed as an ideal of feminine beauty and a symbol of prestige (18). In circumstances of relative deprivation, members of the upper SES may have access to sufficient food to become fat; members of lower SES do not.

Because of the enormous importance of environmental factors in determining obesity, it is unfortunate that our information about factors influencing the prevalence of obesity is largely limited to SES. The effort to control obesity will require a far better understanding of the social factors that promote it than is now available. There is, therefore, an urgent need to explore other social factors in addition to SES. A few recent examples illustrate the direction in which such study may profitably be conducted.
Among adolescent girls in the United States, the inverse relationship between SES and obesity, which had been so firmly established among women, is absent among African-American adolescent girls. Evidently these girls were not affected by the messages on body shape that prevail in U.S. culture. Analysis of the recent Hispanic Health and Nutrition Examination Survey (HANES) has revealed a similar lack of relationship between SES and obesity among certain groups: Puerto Rican women and Mexican-Americans and Cuban-Americans of both genders (19). This study also suggests social determinants different from those that have been the subject of most of the research to date (Kahn LK, personal communication).

Along with these essentially negative findings, an interesting positive finding has emerged from the Hispanic HANES analysis. Acculturation to the American lifestyle by Mexican-American men is associated with an increased prevalence of obesity.

**FUTURE DIRECTION**

In the future, epidemiologic studies of obesity will benefit from measurement and control of genetic influences. A measure of genetic influence may be obtained through assessment of parental obesity, but a more precise measure awaits the detection of genetic markers.

There is an urgent need to move beyond the traditional environmental measure of socioeconomic status as a determinant of the prevalence of obesity. One useful measure has been the disaggregation of the three traditional components of SES—income, occupation, and education—relating each of them individually to the prevalence of obesity. Among the three components, years of education is the easiest to measure and has the greatest cross-cultural relevance. It may also be the most powerful.

New measures of social influences should be sought and Latin America provides an excellent opportunity for such a search. Acculturation is a promising candidate. Other measures undoubtedly will present themselves to the inquisitive investigator; they should be explored with vigor and dispatch.

**REFERENCES**


