Obesity in the United States and, indeed, many developed countries has reached “epidemic” proportions. Recent projections indicate that two thirds of US adults are overweight and one third are obese. Obesity is also increasing throughout the pediatric population. More than 15% of children and adolescents have a body mass index (BMI) ≥95th percentile for age and gender. There is substantial cost to these rising rates—financial, physical, psychologic, and sociologic. Thus, understanding the etiology of this epidemic has become a societal and public health priority.

A simple statement explains increasing obesity. Weight gain results when energy intake exceeds energy expenditure. Not captured in this statement, however, are the many complex and inter-related determinants of energy intake and energy expenditure and the alterations thereof that explain recent secular trends in overweight. These factors remain elusive. The rapidity with which rates have increased indicates that genetic change is not the cause. Social and ecological factors must, therefore, have shifted to create an increasingly pathologic environment.

Social factors are powerful and pervasive determinants of health. Socioeconomic status (SES) is one of the most well studied of these factors. The association between lower SES and poorer health has been well documented, as has the relationship of SES to obesity. Although this literature is growing, important conclusions can be drawn from studies to date. First, there is a clear consensus that the SES-health gradient is not artificial. Second, there is strong evidence for social causation, not social selection except in relation to certain health problems such as schizophrenia, which have early onset and severely affect functioning. Third, lifestyle and behavioral choices, such as diet and level of physical activity, do not wholly explain the influence of SES on health. In fact, SES shapes behavioral choices and also affects many other domains that influence health, such as access to medical care and environmental exposures, making it a fundamental, root cause of disease.

Current research on social inequalities in health focuses on increasing our understanding of the mechanisms that underlie the SES gradient in health. Data suggest that components of SES play distinct roles among different gender, racial, and ethnic groups. For example, income has been demonstrated to be a more powerful predictor of health than education for African Americans, whereas education is a more powerful predictor for non-Hispanic whites. Two theories dominate this field, both of which contribute to our understanding of secular trends in obesity. The materialist explanation posits that differences in income and wealth lead to differences in material conditions in life that directly influence health. These include not only differences in individual household income, but also differences in neighborhood and governmental programs and resources dependent on individual income (ie, taxes). In contrast, the psychosocial theory posits that psychosocial influences are the major social forces involved in the creation of social inequalities in health in developed countries. Proponents of this theory argue that the relative distribution of income and not absolute level is the important predictor of mortality. It is the size of the gap between the rich and the poor that influences health. A wider gap increases feelings of social anxiety and causes other psychological changes, which lead to a disinvestment in social capital, loss of social cohesion, and thus, worse health. The gap between rich and poor is referred to as income inequality, which is measured most frequently by the Gini coefficient. Mathematically, the Gini coefficient is equal to half the absolute difference in incomes, relative to the mean, between two randomly selected people in the population. However measured, greater inequality is associated with poorer health and greater mortality.

In this issue of The Journal, Hesse et al provide a compelling example of how dramatic changes in the social environment affect health, and on weight gain in particular. By using a panel design and drawing from various databases in Germany, these investigators document increases in anthropomorphic measures in all age groups since the reunification of Germany in 1990. Between 1985 and 1997, birth weight increased and childhood and young adult BMI rose. Interestingly, average BMI among mothers also increased during this same period. The findings from this study are simple associations that do not demonstrate causality. The size of the effect is modest. There are very large sample sizes for each age group, which can cause small changes to be statistically sig-

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BMI: Body mass index

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See related article, p 259.
significant but less clinically relevant. However, these findings should not be dismissed. They resonate with much of what we know about the social causation of disease.

The factors which define social status—education, income, wealth, income inequality—are directly relevant to the finding described by Hesse et al. It is likely that, in the period of social disruption after reunification, inequality increased in Germany as the citizenry of East Germany grappled with the complexities of establishing and maintaining a market economy. This increase in inequality led to more poverty and unemployment, to changes in material goods, and decreases in social investment. These dramatic social changes, in turn, are likely to have created powerful health effects, some of which are documented by Hesse et al. They suggest that changes in maternal employment and material goods explain the increases in pediatric and young adult anthropomorphic measures they document. According to Hesse et al., maternal unemployment led to less physically active mothers, and therefore, an increase in birth weight. Maternal unemployment also led to a greater maternal control of children’s diets and, thus, an “individualization and liberalization” of food intake consisting of “energy-dense snacks, chocolate biscuits, pastry, sweets, and fast food,” as opposed to the “calorie balanced,” meals provided by daycare facilities and schools. This led to increases in children’s BMIs. Car ownership led to decreased physical activity, contributing to increases in BMI among children, military recruits, and mothers.

Although maternal unemployment and lifestyle choices of postreunification families are relevant, we should not blame the mother for society’s ills. Hesse et al. ignore the fact that among children, military recruits, and mothers.

REFERENCES


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Can diagnostic evaluations to “rule out” endocarditis be improved?

Over the past several decades, the epidemiology of endocarditis in children has changed. Complex congenital heart disease (CHD) has replaced rheumatic fever as the most common risk factor for endocarditis in children. With the increased use of central venous catheters, nosocomial endocarditis is being diagnosed with greater frequency in children, including preterm infants, with structurally normal hearts. Pathogens have also changed as staphylococcal species and fungal species cause a greater proportion of cases than Viridans streptococci.

Diagnosing endocarditis is not always straightforward. Several scoring systems have been devised that are based on a compilation of signs and symptoms and rate the probability of endocarditis as definite, probable, possible, or unlikely. The definite diagnostic category used by the classic von Reyn’s criteria requires pathologic confirmation of a vegetation or intracardiac abscess or autopsy evidence of endocarditis. Such conclusive diagnostic criteria are rarely obtained today. More recently, the Duke’s criteria have been developed, which diagnose probable endocarditis by echocardiographic findings such as a vegetation or valvular dysfunction. Modifications to the Duke’s criteria have been proposed that use a complex scoring system of major and minor criteria and include newer diagnostic modalities for Q fever and Bartonella spp. All 3 scoring systems have been validated in adults with endocarditis, but there are few studies that validate their diagnostic utility in children.

In this issue of The Journal, Michelfelder et al evaluated the yield rate (YR) of echocardiography in efforts to make recommendations to improve the appropriate use of this test in children. To establish the YR, the results of echocardiography were compared with the probability of endocarditis as defined by the von Reyn’s clinical criteria (assessed by retrospective medical record review) in 101 children undergoing echocardiography to “rule out” endocarditis. Not surprisingly, the yield rate of echocardiography was highest in children (n = 10) with probable (YR = 80%) or children (n = 20) with possible (YR = 20%) endocarditis compared with an overall YR of 12%. It is reassuring that all 71 patients with a low probability of endocarditis (ie, endocarditis was rejected by the von Reyn’s criteria) had negative echocardiograms. This study suggests, therefore, that many children could have safely avoided undergoing echocardiograms.

It is common clinical practice to order transthoracic echocardiograms (TTEs) to “rule out” endocarditis in children with CHD and a positive blood culture, or in children with structurally normal hearts, a central venous catheter, and persistent positive blood cultures. Bacteremia or catheter-related bloodstream infections are the more likely diagnosis in such children than endocarditis. However, because of their low risk, TTEs are ordered without careful consideration of the likelihood of...