Overweight and overwrought is a fitting epithet for modern man or woman. Up to 30 percent of the American population is obese. Stress and anxiety are harder to measure but have to be considered pervasive influences in contemporary life. There is evidence that both obesity and stress may increase the risk of serious, even life-threatening diseases, such as diabetes, heart disease, and hypertension. Medical scientists would like a better understanding of both conditions and their relation to disease, but that understanding has proved hard to come by. In fact, recent attempts to assess the causes of obesity and the connection between stress and hypertension have found the available evidence weak and often contradictory. Despite the claims to be found in popular, and sometimes even in medical, reports of these subjects, it appears that there is a good deal of uncertainty in the research. The following articles explore some of the ambiguities confronting researchers studying obesity and the connection between stress and hypertension.

Obesity

A Growing Problem

Despite the social stigma of being obese, the adverse effects of obesity on health, and the proliferation of new diets and treatments for obesity, the condition itself shows no sign of going away. In fact, many investigators believe that the prevalence of obesity is actually increasing in the United States. They note that adults examined during the years 1971 to 1974 weighed more, on the average, than those examined from 1960 to 1962. According to George Bray of Harbor General Hospital in Torrance, California, 10 to 30 percent of all Americans weigh at least 30 percent more than their "ideal" weights.

What is most disturbing is that investigators confess ignorance about what causes obesity and how best to treat it.* Discouragingly few people lose weight with any drug, diet, or other treatment, and even those who do lose weight are likely to regain it later. Moreover, many of the theories about obesity, such as the theory that fat babies become fat adults, may have little basis in fact.

Hypotheses concerning the causes of obesity abound. However, as predictors of the condition, social factors appear to be as good as or better than genetic or metabolic ones. Stanley Garn of the Center for Human Growth and Development in Ann Arbor, Michigan, points out that fat parents tend to have fat children. Lest anyone conclude that fatness is an inherited trait, he adds that this correlation holds equally well for adopted children and pets. Children of obese parents grow fatter and fatter, he says, until, by the time they reach age 17, they generally are three times as fat as those of lean parents.

Garn reports that obesity is also associated with education, sex, race, and social class. Less educated women tend to be fatter. Black women are fatter than white women but black men are leaner than white men. As they move up the socioeconomic scale, black men get fatter and black women leaner. Albert Stunkard of the University of Pennsylvania adds that ethnicity also plays a role. Americans of English, Scotch, and Irish descent tend to be thinner. "The prevalence of obesity increases as you move eastward across Europe," he says.

Researchers agree that many widely held beliefs about the causes and effects of obesity are either not established or not true. For example, Trevor Silverstone, a psychiatrist at German Hospital in London, questions the role of psychological factors in the development of obesity. Whenever investigators carefully compare obese to nonobese people, he says, they find no major psychological differences. Thus some fat people

*This is a conclusion drawn by an international group of researchers at a recent conference on Obesity and the American Public. The conference was held at the National Institutes of Health on 20 to 22 October. It was sponsored by The National Council on Obesity, The Nutrition Foundation, the National Institute of Arthritis, Metabolism, and Digestive Diseases, the National Institute of Child Health and Human Development, the National Heart, Lung, and Blood Institute, and the John E. Fogarty International Center.

Stress

Role in Hypertension Debated

The belief that stress can cause hypertension is widespread. The difficulty lies in proving that this supposition is correct. As Adrian Ostfeld of Yale University puts it: "As a physician who saw patients weekly in the clinic I was sure that stress could cause hypertension; but as an epidemiologist following patients at 6-month intervals I lost that assurance." In fact, hypertension has generally been a tough nut for medical scientists to crack. They can usually control it with drugs or diet but still do not understand what causes some 90 percent of the cases.

Recently, a multidisciplinary group of investigators, including clinicians, epidemiologists, neurobiologists, and psychologists participated in a day-long panel discussion* of the evidence regarding stress and hypertension. Although not everyone agreed, the epidemiologists on the panel concluded that the studies done thus far, many of which have produced contradictory results, have failed to establish a clear link between stress and the development of hypertension. Thus, with the evidence still unclear, they think that it is now premature to recommend that members of the public change their life-styles to avoid stress in the hope that this will prevent hypertension.

Not in question, however, is the unfavorable effect stress may have on the course of the disease once it begins. Stress, which can cause transient increases in blood pressure even in normal individuals, may drive elevated pressures even higher, thus accelerating blood vessel damage or even precipitating a stroke in someone with uncontrolled high blood pressure. The uncertainty revolves around the theory that prolonged or repeated exposures to stress may produce permanent physiological changes in some persons who consequently develop chronic hypertension.

One panelist, S. Leonard Syme of the University of California at Berkeley, says that in the past he has concentrated on the more positive findings that seemed to indicate that stress is a cause of hypertension. But he has been increasingly troubled by his observation that "every one of the hypotheses presented by psychosocial epidemiologists regarding the etiology of essential hypertension is contradicted by as much evidence as exists in its support." Syme now says that he thinks the epidemiological research has reached a dead end.

As examples of the problems that concern him, he cites

*The discussion was held during a conference on The Crisis in Stress Research: A Critical Reappraisal at Boston University Hospital on 20 to 21 October. It was sponsored by the Department of Psychosomatic Medicine of the Boston University School of Medicine and supported by a grant from the Roche Psychiatric Service Institute. The panelists were: Alvin P. Shapiro (chairman), University of Pittsburgh School of Medicine; Herbert Benson, Harvard Medical School; Aram Chobanian, Boston University School of Medicine; J. Alan Herd, Harvard Medical School; Stevo Julius, University of Michigan Medical School; Norman Kaplan, University of Texas Health Science Center; Richard S. Lazarus, University of California at Berkeley; Adrian Ostfeld, Yale University; S. Leonard Syme, University of California at Berkeley.

Obesity

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as well as some lean people eat under stress. But stress-related eating is actually less prevalent among obese than nonobese people. Similarly, fat people are no more likely than lean people to be stimulated to eat by the mere sight or smell of food. There is a spectrum of eating behavior for both obese and nonobese people. Some eat quickly, some slowly, for example. The obese, however, cannot be distinguished by their eating behavior.

Another widely held belief is that fat babies are doomed to be fat adults. Lester Salans of the National Institute of Arthritis, Metabolism, and Digestive Diseases in Bethesda, Maryland, reports that the evidence for this hypothesis is conflicting. Although severely obese children are likely to remain obese, it is not clear that this relation holds for mildly obese children. In fact, studies done in England indicate that most fat babies become lean during childhood and that obese people who became obese in infancy are only a small portion of the total number of obese adults.

Investigators do have some evidence that people who become fat as infants and who remain fat may have more fat cells than people who become fat as adults. In lean children, fat cells proliferate during the first 2 years of life, remain constant in number from age 2 to 10, then proliferate again from age 10 to 16. Jerome Knittle of Mt. Sinai School of Medicine and others found that the number of fat cells in obese children increases almost continuously from birth to age 14 to 16.

A number of investigators have postulated that there may be critical periods in early life when fat cells are particularly sensitive to nutritional, hormonal, metabolic, and other factors that influence cell division and development. According to Salans, however, no one has yet been able to determine whether there are any critical periods for fat cell development in humans. In fact, he says, fat cells can increase in number at any time in life. Furthermore, no one has been able to show that obesity that is manifest early in life is any more resistant to treatment than adult-onset obesity.

It is generally believed that obesity is not one but a multitude of disorders, and that treatment should be tailored to the patient's particular type of obesity. For example, some people have very large fat cells, some have very many fat cells, and some have both large and numerous fat cells. But researchers are a long way from knowing which people will do best with which treatment and whether or how prognoses are related to these cellular characteristics.

In general, only 5 to 20 percent of obese people can lose weight and keep it off after dieting, and no one diet has been found to be any better, in terms of long-range results, than any other. Even the currently highly promoted "liquid protein" diets yield discouraging results. Saul Genuth, of Mt. Sinai Hospital in Cleveland, reports that "Cure of obesity is virtually unheard of and even control is only rarely achieved."

Diets have been supplemented with drugs, but many investigators are wary of drug treatments. The long-term effects of the drugs used to treat obesity have not been demonstrated, they say, and many of the drugs are dangerous or addictive.

Another trend is to combine behavior modification programs with diets. Henry Jordan of the Institute for Behavioral Education at King of Prussia, Pennsylvania, says that this approach has been the focus of a tremendous amount of research, but the research has progressed very slowly over the past decade. The whole idea of behavioral modification, he explains, is based on the false assumption that there is a characteristic obese eating style and that this is learned behavior that can be changed.

With the failure of conservative treatments, some investigators turn to radical treatments for obesity. Some of these treatments, such as intestinal bypass operations, have higher success rates than the more conservative methods, but they also are very expensive and more dangerous to the health of patients. Other treatments, such as jaw-wiring, offer no better long-term effects than the conservative treatments. All of the radical treatments raise the question of whether the treatment is worse than the disease.

Obesity is associated with a number of adverse effects on health. For example, obese people are more prone to develop gallbladder disease, strokes, diabetes, heart disease, and hypertension. They are also likely to suffer damage to the weight-bearing bones and joints, and this damage may be aggravated by gout or arthritis. Obese women are prone to menstrual disorders and endometrial cancer. But there is such a thing as a healthy fat person. In fact, according to Stunkard, people who become mildly obese in childhood and remain so are likely to be fairly healthy—at least compared to those with adult-onset obesity.

Researchers are still hopeful that promising results on how to treat the obese will be forthcoming. One major advance would be the ability to distinguish between different types of obesity and to establish which treatment is best for each type of the disorder. But it is still maintained that prevention, not treatment, is the ultimate solution to the problem of obesity. Prevention could be aided by improving education in such subjects as health and nutrition, and by providing information on the sizes of "normal" portions of food. Increased exercise, especially for children, could also help. Some evidence indicates that people are changing their diets, stopping smoking, and controlling the amount of cholesterol and saturated fats in their diets in response to a public education campaign for the prevention of heart disease. It is possible, then, that they may change their life-styles, or at least their children's life-styles, to prevent obesity as well.—GINA BARI KOLATA

Stress

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some studies that have reported higher blood pressures in urban populations than in rural ones, whereas other studies have found the opposite. (Urban areas are supposed to be more stressful places to live than rural environments.) Even relationships generally thought to be well established may not be as clear-cut as they once appeared. The incidence of hypertension is supposed to be higher in women than in men and in blacks than in whites. But most investigations have actually shown that younger women's blood pressures are usually lower than those of younger men; sometime around the age of 45 the situation reverses.

Moreover, Syme has evidence that socioeconomic class may account for much of the apparent higher incidence of hypertension among blacks than whites. Analysis of data from some 22,000 persons, most of whom are members of the Kaiser Foundation Health Plan in the San Francisco area, indicates that the blood pressures of members of both races increase as their socioeconomic class decreases. Thus, whatever the cause—stress, obesity, and less adequate medical care have all been suggested—members of the lower socioeconomic classes appear to be at increased risk of developing chronic high blood pressure. Since a higher proportion of

blacks belongs to the lower socioeconomic classes, the incidence of the condition appears to be higher among them than among whites. However, there are studies in which a greater incidence of high blood pressure in the lower socioeconomic classes has not been found, and these serve as a further indication of the confusion surrounding the etiology of hypertension.

Ostfeld and Richard S. Lazarus of the University of California at Berkeley are also dismayed by the failure to establish that stress causes at least some cases of hypertension. The investigators point to a number of possible reasons for the failure. One of the most important, according to Lazarus, is the lack of recognition that stress is not just an environmental condition; rather it is an interaction between a particular external environment and a particular kind of person. In other words, not every individual will appraise and react to a situation in the same way. If the psychological responses differ, then the physiological reactions might also differ. Lazarus says that few studies have included consideration of these individual differences.

In addition, Lazarus maintains that an individual's responses vary from time to time, even from moment to moment, and that this variability is not studied in most epidemiological research. Large numbers of people are included in the studies and their physical and psychological states are assessed only at long intervals, and sometimes only once. In a moment of frustration, Norman Kaplan of the University of Texas Health Science Center pointed out that "you cannot put these people in a cage and watch them all day." But Lazarus thinks that it is possible to gain significant information by studying fewer people more intensively than is usually done, and is taking that tack in his own research. By repeatedly measuring the psychological and physiological responses of individuals in varying life situations, he hopes to spot the characteristics of those at greater risk and clarify the role of stress—if there is one—in producing chronically elevated blood pressures.

Variability of individual responses was also stressed by Syme who thinks that it is necessary to distinguish between a generalized susceptibility to becoming ill and the development of a specific illness. Investigators have reported that increased susceptibility to illness in general—everything from influenza to cancer and heart disease—is associated with stress. Thus, Syme thinks that attempts to relate stress to one or another specific disease will continue to be unsatisfactory; a more productive approach would be to look for situations that increase general disease susceptibility.

A related question to be explored is what determines which particular disease an individual may develop under conditions favoring increased susceptibility. Even if they cannot agree on the cause of hypertension, investigators do agree that there are large individual differences in susceptibilities to the condition. A great deal of current effort is aimed at identifying those persons who are at higher risk.

However, personality tests are not much help in this regard, according to the panelists. There have been suggestions that there is a "hypertensive personality" characterized by a high degree of suppressed anger or hostility and "tender-mindedness" (meaning an increased tendency to develop hurt feelings). Efforts to confirm these suggestions have, if anything, been even less productive than those directed at pinning down the relationship between external stresses and hypertension.

If stress does cause hypertension then a mechanism to explain how psychological responses are converted to permanent physiological changes is required. Several investigators are examining the function of the sympathetic nervous system in this regard because it is known to mediate normal responses to stress and may also be involved in the pathological ones. But Ostfeld thinks that a nervous mechanism may not be needed to explain how stress might cause chronic high blood pressure, and suggests that obesity and excess salt intake could be the true culprits.

He points out that the relationship between excess weight and elevated blood pressure is one of the most consistently demonstrated linkages of all. Hypertensive patients tend to be heavier than persons with normal blood pressures. Conversely, persons with normal pressures are less likely to develop hypertension than obese individuals. And when hypertensives lose weight, their blood pressures drop. Excessive intake of dietary salt is another factor that is widely, if not universally, accepted as predisposing some individuals to the development of hypertension.

According to Ostfeld, stress may lead indirectly to hypertension by increasing appetite and thus the intake of food, including salt. He says that the relationships between sociocultural variables and obesity parallel those between the variables and the incidence of hypertension. For example, moving from a traditional agricultural or hunting society to an urban environment has been associated with an increased incidence of hypertension in many studies (although not in all of them). This change may be stressful but it is also usually accompanied by decreased physical activity and increased food consumption, both of which contribute to a gain in weight. Moreover, some studies indicate that the incidence of both obesity and hypertension increase as socioeconomic class decreases in highly developed countries. Thus, Ostfeld says obesity and high salt intake may be more plausible explanations for the development of hypertension than stress-induced neurological changes.

Although concerned by some of the inconsistencies in the data, several panelists, including Alvin P. Shapiro of the University of Pittsburgh School of Medicine and Herbert Benson of Harvard Medical School, held out for the view that the preponderance of the evidence favors a connection between stress and chronic high blood pressure. Investigators, including Benson, have shown, for example, that "relaxation techniques" that produce physiological changes opposite those evoked by stress, do produce modest decreases in blood pressures of hypertensive patients. The decreases are prolonged and not just limited to the times that the patients are practicing the techniques. Benson says that the methods should be useful as adjuncts to, but not as replacements for, drug therapy.

Shapiro emphasizes that investigators generally agree that disturbances in any of several interacting systems or organs, such as the endocrine system, brain, and kidneys, could result in chronically elevated blood pressures. The problem is that the more factors that are involved the harder it is to sort out the role played by each. Considering how difficult it is to pinpoint the roles of the physiological factors, which are easy to quantitate compared to stress, it is not surprising that the results of some studies designed to test the stress hypothesis have been unsatisfactory.—JEAN L. MARX

Science Writing Award

Jean L. Marx and Gina Bari Kolata have won an American Heart Association Blakeslee Award for their series of articles on heart research that appeared in Research News last fall and winter.