

rhages, when taken with foods or beverages high in tyramine. Long-term intake of Sinemet, used to treat parkinsonism, may cause niacin deficiency (Bender, Earl, and Lees 1979).

Caffeine is a pharmacologically active agent found in foods and drugs (Snyder et al. 1981) that can enter the brain because of its lipid solubility. Animal studies have suggested that foods can impair the efficacy of drugs by blocking their uptake into the brain. For example, a high-protein meal may diminish the efficacy of L-dopa (used to treat parkinsonism) or of Aldomet (used for high blood pressure) because these drugs are transported into the brain by the same carrier molecules that transport certain amino acids. The significance of these suggestions for either animal (Peter and Harper 1985) or human drug metabolism is uncertain at this time.

## **Implications for Public Health Policy**

### **Dietary Guidance**

#### **General Public**

Nutrients of concern in stroke are those associated with its major diet-related risk factors—hypertension, diabetes, and obesity. Evidence suggests that diets low in sodium and alcohol, as well as caloric intake and physical activity to achieve and maintain desirable body weight, should be recommended as public health measures to prevent stroke and its related conditions. Excessive drinking has been associated with stroke; hence, this practice should be avoided. Although some evidence links very large exposures of major dietary components (e.g., amino acids, choline) to nervous system disorders other than stroke, this evidence is, for the most part, preliminary and remains to be confirmed by additional clinical evidence before implications can be drawn.

Over- or underconsumption of certain vitamins and minerals can damage the nervous system as in the occurrence in alcoholics of thiamin deficiency-related Wernicke-Korsakoff's syndrome.

#### **Special Populations**

Studies in patients with major diet-related risk factors for stroke indicate that similar dietary changes can reduce the level of the risk factor and help prevent cardiovascular disease (see chapters on high blood pressure, diabetes, and obesity). Qualified health professionals should provide patients with information on the means to achieve these changes. In addition to a

focus on weight reduction and sodium restriction, this information should emphasize the importance of alcohol restriction in patients with high blood pressure and/or high glucose levels.

Suggestions that certain foods or food components might influence headache or epilepsy have yielded conflicting research results and are too preliminary to draw conclusions.

### **Nutrition Programs and Services**

#### **Food Labels**

Evidence related to the role of dietary factors in stroke and other neurologic diseases supports the need for sodium labeling of packaged food products.

#### **Food Services**

Food service programs should emphasize diets low in sodium and calories to maintain ideal body weight and to control obesity and diabetes.

#### **Special Populations**

Patients at high risk for stroke and other neurologic conditions should be provided with counseling and assistance in the development of diets appropriate to their conditions.

### **Research and Surveillance**

Research and surveillance issues of special priority related to the role of diet in neurologic disease should include investigations into:

- The role of specific dietary factors in the etiology and prevention of stroke.
- The relationship, if any, between specific dietary factors and specific brain functions such as memory, alertness, and response time.
- The mechanisms by which food components, such as dietary precursors of neurotransmitters and certain additives and toxins, might affect nervous system function.
- The role, if any, of specific dietary factors in the etiology and prevention of Alzheimer's disease.
- The nutritional needs of the brain and nervous system in health and throughout life.

- The effects of excessive intake of nutrients and supplements (vitamins A, B<sub>6</sub>, etc.) on nervous system function.
- The mechanism or mechanisms by which excessive alcohol intake increases the risk for stroke.
- The ability of diets low in calories, sodium, alcohol, and, perhaps, other dietary factors to prevent stroke.
- The most effective methods to educate the public about diet-related risk factors for stroke, and to assist the public in making recommended dietary changes.
- The comparative effects of dietary insufficiency on cognitive function and neurologic disease.
- The understanding of the blood-brain barrier nutrient transport processes and the mechanisms by which diet may influence brain function and health.
- The role of preexisting nutritional disease and nutritional status on the impact of neurotoxins.

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## Chapter 14

# Behavior

Thus it may appear, that there ought to be a great reciprocal influence between the mind and alimentary duct.

David Hartley

*Observations on Man,*

Vol. I, Chapter II, Section 2 (1749)

### Introduction

The disciplines of nutrition and behavior are not usually considered to be closely related, but there are in fact several key areas of overlap between these fields. Behavioral factors determine the choice of foods in the diet, and any attempt to change dietary patterns must necessarily involve changes in behavior. Because eating itself is a behavior controlled by the brain, all disorders of eating inherently involve the central nervous system and may be associated with mood changes. Although considerable public attention has focused on suggestions that ingestion of certain foods or nutrients might influence behavior, it has been difficult to confirm such effects. Similarly, suggestions that some nutrients can modify specific aspects of brain function and behavior also require further confirmation. This chapter reviews relationships between nutrition and behavior. For more detailed discussion of these issues, the reader is referred to recent volumes on this topic (Miller 1981; Olson 1986; Wurtman and Wurtman 1977–86) and to the chapter on neurologic disorders in this Report.

### Historical Perspective

That diet influences behavior is an ancient human belief. Primitive people attributed friendly and unfriendly feelings to plants and animals and expected these feelings to be transferred to anyone who ate such foods. Eating the heart of an aggressive animal, for example, was thought to confer strength and courage, whereas consumption of timid or weak creatures would undermine desirable attributes (Farb and Armelagos 1980).

In religious teachings, the behavior of mankind was said to change instantly when Eve ate the apple. Solomon, suffering the pangs of love, was comforted with apples (Cussler and deGive 1952). The ancient Greeks proposed that the human body is composed of four “humors”—hot, cold, wet, and dry—that control health, feelings, and behavior, and extended these categories to foods and other factors in the environment. Imbalances in these humors were thought to cause disease; physical and mental health could be restored by consuming foods in the opposite, complementary categories. Anger, for example, was considered hot and dry, and melancholy was cold and dry. These ill humors could be corrected by consuming foods that were cold and wet, or hot and wet, respectively (Farb and Armelagos 1980). Such ideas have carried forward to the present day, when many cultural groups believe in hot/cold or yin/yang approaches to food and health (Ludman and Newman 1984).

Systematic study of cultural influences on food intake began early in this century as anthropologists examined the use of food in isolated cultures and ethnic groups. These studies recognized the importance of societal influences on food selection and use, and they indicated that food intake is not an isolated, individual response to the sensory properties of food but instead depends greatly on the symbolic meanings of food in each culture (Goode, Curtis, and Theophano 1981). Even in contemporary times, foods are endowed with magical powers (Kimmens 1975) and are believed to symbolize feelings such as those of satisfaction and security (Bruch 1973).

### **Significance for Public Health**

The reduction of behavioral risk factors for chronic disease and the development of effective means to do so are major public health goals, and improvement of the food choices and dietary practices of individuals and populations is the key to control of the conditions reviewed in this Report. Obesity, for example, is a common nutritional problem in the United States today, affecting about 34 million adults. Dietary fat and cholesterol increase the risk for a number of chronic diseases, including the Nation’s leading killer, cardiovascular disease. These conditions are discussed in the respective chapters.

Other eating-related problems such as anorexia nervosa (self-starvation) or bulimia (binge/purge syndrome) are relatively uncommon. Anorexia nervosa occurs most often in females from the upper and middle social classes and usually begins between the ages of 13 and 20. Even among persons at highest risk, the prevalence is less than 1 percent. In the general population, approximately 1 in 250 suffer from anorexia nervosa in any given year

(APA 1987). Furthermore, despite a general impression to the contrary, the incidence of anorexia nervosa does not appear to be rising significantly (Williams and King 1987). Because of difficulties in its diagnosis, estimates of the incidence of bulimia vary from 2 to 19 percent among college women, the group at highest risk (Lustic 1985; Zuckerman et al. 1986), and are about 5 percent for men (Halmi, Flak, and Schwartz 1981). A recent survey of university students, however, identified only 1.3 percent of the female and 0.1 percent of the male respondents as meeting standard diagnostic criteria for this condition (Schotte and Stunkard 1987).

Pica (consumption of nonfood substances) is another eating disorder of concern. Because definitions of pica vary and individuals who practice pica are reluctant to admit it, it has been difficult to establish the prevalence of this condition. Thus, estimates of the proportion of individuals in selected groups of children, pregnant women, or other adults who practice pica have varied from 0 to 66 percent (Danford 1982), and no conclusions can as yet be drawn about its prevalence in the general population.

Other behavioral disorders commonly associated with food choices include childhood hyperactivity, antisocial behavior, and hypoglycemia. Neither the prevalence nor the nature of these conditions is reliably established.

### **Scientific Background: Methodological Issues**

Although many research reports have concluded that food and nutrients influence behavior, most of these accounts do not meet criteria for controlled scientific research. Environmental, cultural, socioeconomic, and psychologic factors can induce symptoms such as anxiety, depression, fatigue, insomnia, or irritability, and it is difficult to demonstrate that such symptoms are caused by dietary factors unless research studies are designed to rule out other potential causes (Kanarek and Orthen-Gambill 1986).

Understanding of nutrition and behavior is also limited by unresolved questions about the applicability of much animal research to humans and by inadequacies in present knowledge of the effects of nutrients on brain function. Imprecise and inappropriate definitions and methods of measuring normal behavior also limit understanding. Such difficulties explain why research studies produce results that are often inconsistent and cause public and professional controversy.

Various authorities have emphasized the importance of methodologically rigorous research in this field and have urged investigators to design studies that include appropriate standards of measurement, adequate sample sizes, controls for confounding variables, elimination of sources of investigator and subject bias, appropriate statistical treatments, and caution in the interpretation of results (Sprague 1981; Anderson and Hrboticky 1986). These issues are especially important in establishing cause or effect as well as in establishing policies for regulation or labeling (Sobotka 1986).

### **Key Scientific Issues**

- Behavioral Determinants of Eating Habits
- Behavioral Aspects of Eating Disorders
- Effects of Foods and Nutrients on Behavior

### **Behavioral Determinants of Eating Habits**

#### **Behavioral Issues in Food Selection**

Although infants do not begin life with a choice of food, some of the most obvious reflexes at birth are those associated with eating. Infants learn to associate eating with security and relief from anxiety, tension, and distress. Later, children eat in conformance to cultural and familial standards. Throughout life, food symbolizes and mediates social relationships and is strongly linked to deep feelings of acceptance and comfort or deprivation (Bruch 1973). These ingrained meanings attached to the roles of food in society suggest reasons that food habits often can be changed only with difficulty.

Although the choice of certain foods as opposed to others may greatly affect nutritional status, surprisingly little is known about the factors that determine food selection beyond the fact that they include multiple environmental, cultural, genetic, social, and sensory variables that interact in complex ways (Rozin 1984). People must choose foods from those that are available, but the foods selected to be grown and sold vary greatly among different cultures. Within a given culture, individual food choices depend greatly on sociocultural systems that govern food production, distribution, and consumption (Goode, Curtis, and Theophano 1981; Harris 1985).

Individual preferences are the chief factors in food choices, but the elements that determine food preferences are poorly understood. Preferences for flavor and taste are learned, culturally determined, and dependent on

the degree of exposure (Story and Brown 1987). The one exception appears to be an innate preference for foods that are sweet. This preference is observed in early childhood and continues throughout life (Desor, Mallor, and Greene 1977). Aversions to foods are also learned and seem to occur most often when associated with gastrointestinal distress or the introduction of novel foods (Rozin 1984).

Selection of foods for nutritional or health reasons is also a learned behavior. Contrary to a commonly held opinion, infants have not been shown to have an inborn ability to select a balanced, nutritious diet (Story and Brown 1987). Recent studies indicate that the variety of foods available has an important effect on food consumption; the more the available foods are varied, the more of them people will eat (Rolls 1985).

#### Behavioral Aspects of Dietary Change

Behavior change—especially in dietary practices—is a key element in reducing the risk for chronic disease. Individual and public health efforts to induce beneficial changes in dietary habits are based on the assumption that people who understand the risks associated with their present practices will alter them to prevent illness. Although this assumption appears self-evident, education may not have as great an impact on food behavior as might be expected unless it can overcome counteracting barriers, including, perhaps, a general belief that some dietary practices are worth the risk (Syme 1986).

Extensive societal and behavioral forces inhibit dietary change. Eating behaviors are acquired over a lifetime; to change them requires alterations in habits that must be continued permanently—long beyond any short-term period of intervention. Dietary advice is often restrictive and viewed as depriving or unpleasant. It may also be incompatible with cultural or familial standards of appropriate food intake. Dietary changes may require increased cost, skill, time, or effort needed for food preparation (Glanz 1986).

Furthermore, environmental factors such as peer pressure, advertising of high-calorie foods and alcoholic beverages, or other cultural determinants may strongly counteract recommended changes (Syme 1986). Social and political values and economic considerations may also be obstacles (Somers and Weisfield 1986). Together, these barriers suggest that dietary advice (like much other advice) is far easier to give than to accept.

Despite these difficulties, considerable evidence supports the effectiveness of nutrition education in changing dietary intake to reduce risk factors for or symptoms of conditions such as coronary heart disease, diabetes, hypertension, and renal disease. For reasons that are poorly understood, public health efforts to reduce the incidence of obesity have been considerably less effective (Glanz 1986) and are discussed below.

Although no single method of counseling for dietary change is universally effective, the key elements of the more successful strategies have been identified repeatedly. These are (Zifferblatt and Wilbur 1977; Glanz 1986):

- To set realistic, achievable, and measurable goals.
- To tailor recommendations to individual lifestyle and dietary preferences.
- To use whatever social support systems are available to provide training in skills as well as to provide information.
- To establish good communication between educator and clients.
- To provide systematic followup, reinforcement, and monitoring.

### **Behavioral Aspects of Eating Disorders**

#### **Obesity**

Obesity is the excessive accumulation of fat in the body. As discussed in the obesity chapter, approximately one-fourth of Americans are classified as overweight (Abraham et al. 1983). The definition, prevalence, cause, and treatment of obesity are discussed in that chapter.

At one level of understanding, the cause of obesity is quite simple: fat accumulates when more calories are consumed than are expended. It is difficult to demonstrate a positive correlation between total food consumption and the extent of obesity in specific individuals, although such relationships can be observed in population studies (Rolland-Cachera and Bellisle 1986). Human obesity is often a familial disorder; obese parents tend to produce obese offspring. While this observation does not distinguish the influences of heredity and environment, studies of twins suggest that there is a substantial genetic component to human obesity (Stunkard et al. 1986). It is also possible that some people become obese and sustain their obesity because mechanisms that normally regulate food intake are impaired (Wurtman et al. 1981; Rosenthal, Sack, James, et al. 1984). Obesity is more common among women than among men at all ages, and its prevalence increases with age but decreases among individuals of higher socioeconomic status and greater levels of physical activity.

*Behavioral Determinants.* Behavioral and psychiatric contributions to obesity—those factors that affect eating behavior—have been the focus of extensive investigation, based largely on the assumption that overeating is the primary cause of obesity. The evidence for this assumption is, at best, only partially correct (see chapter on obesity). Once obesity is established, food choices and caloric intake are no longer “normal,” and personality differences between obese and normal-weight individuals may be due more to the results of physiologic changes, social discrimination, or dieting than to behavioral factors.

Because of these complexities, which impede scientific understanding at basic levels, numerous behavioral hypotheses have been proposed to account for the differences in weight between obese and normal-weight individuals. These hypotheses have been reviewed recently (Striegel-Moore and Rodin 1986) and are summarized in Table 14-1.

Considerable research supports many of these theories, but none has yet been demonstrated to account consistently for group differences between obese and normal-weight individuals. For most variables measured, no group differences have been found. Instead, the many tests of these hypotheses suggest that variation between individuals is greater than that between the groups; that behavioral correlates might be the result of obesity rather than its cause; and that even when a behavioral effect is identified, its relation to the etiology of obesity is uncertain (Striegel-Moore and Rodin 1986). The precise causal role of behavioral factors in obesity is still to be determined.

**Table 14-1**  
**Behavioral and Psychologic Hypotheses to Explain Obesity**

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Unconscious personality conflicts
Response to anxiety or depression
Neurotic personality traits
External locus of control
Preference of immediate gratification
Abnormal eating styles
Excessive food intake
External eating cues
Response to food palatability
Taste perception
Response to food variety
Restrained eating (response to dieting)
Arousal misperception

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Source: Striegel-Moore and Rodin 1986.

*Behavioral Associations.* Of the various emotional disturbances to which obese persons are subject, three are specifically related to their obesity: disparagement of body image, psychologic complications of dieting, and binge eating (Wadden and Stunkard 1985). A fourth complication—fear of obesity—has also been reported. Binge overeating occurs in only a small percentage of obese persons and is discussed below with regard to bulimia.

Disparagement of body image affects a larger percentage of obese persons than nonobese persons who characteristically dislike their own bodies. Such feelings are closely associated with self-consciousness and impaired social functioning, and these feelings are not unexpected in view of the pervasive social biases and discrimination against the obese in this society (Brownell 1984). They are usually observed in persons who have been obese since childhood. Even among this group, however, fewer than half suffer from impaired body image.

Psychologic complications of dieting include anxiety, depression, irritability, and preoccupation with food. These disturbances may arise from the same mechanisms that occur in normal-weight persons who restrict their food intake (Stunkard and Rush 1974) and are discussed further in the chapter on obesity. Fear of obesity has been reported as a cause of self-induced dieting and subsequent weight loss, growth retardation, and delayed puberty in a small group of boys and girls ages 9 to 17 (Pugliese et al. 1983).

*Behavioral Treatment.* Although weight reduction ought to confer great benefits upon obese persons and be simple to accomplish, clinical experience has shown obesity to be remarkably resistant to treatment. The basis of weight reduction is deceptively simple: Establish an energy deficit by consuming fewer calories than are expended or by expending more calories than are consumed. As discussed in the obesity chapter, the most effective treatment methods involve combinations of diet, exercise, and behavior modification. Most such treatment in the United States is carried out under the direction of nonmedical groups and counselors in programs that pose minimal hazard, although they are of uncertain long-term effectiveness.

The role of behavioral strategies to enhance the effectiveness of weight loss programs has been the subject of active investigation. These strategies derive from the key assumptions and characteristics of behavior therapy: Behavior is acquired; treatment measures should be specified and outcomes evaluated; treatment should be individualized; and treatment effectiveness should be assessed (Wilson and O'Leary 1980). Behavioral weight control programs usually include group participation at weekly meetings

for periods of 2 months or longer and involve techniques of stimulus control (e.g., shop from a list, do not save leftovers), eating behavior (do nothing else while eating, chew thoroughly), reward, self-monitoring, nutrition education, physical activity, and cognitive restructuring in which patients are encouraged, for example, to set reasonable weight goals and to think about progress rather than failure (Stunkard and Berthold 1985).

Evaluations of the effectiveness of such programs have indicated that when spouses of obese patients were included during treatment, followup weight loss is greater (Brownell et al. 1978). Obese patients who participated in regular peer group meetings following the completion of formal treatment have been observed to maintain their weight loss better than those who do not participate in such meetings (Perri et al. 1984). Other behavioral studies have described factors associated with prevention of relapse following weight loss programs, such as strategies to cope with the presence of favorite foods (Marlatt and Gordon 1985).

*Effects of Mood Disorders.* It has been known for many years that disturbances of appetite and weight regulation are important symptoms of certain mood disorders. In pioneering work on manic-depressive disorders, for example, body weight was observed to fall early in a depressed phase, while a rise in body weight was often one of the earliest signs of recovery (Kraepelin 1921). Other patients, however, gained weight during protracted depression. In later large-scale studies of depression, eating and weight disturbances were found in 70 percent of depressed subjects (Leckman et al. 1984), and weight gain was observed in 70 percent of 263 depressed subjects during their recovery from depression (Kraepelin 1921). Depression can be accompanied by either gain or loss of weight, and in general, disturbances in appetite and weight regulation are accepted as central features of mood disorders (Herzog and Copeland 1985; Rosenthal, Sack, James, et al. 1984b; Slochower 1983), although it is important to distinguish such effects from those of antidepressant medications. Nevertheless, because disturbances in eating behavior may be characteristic of specific subcategories of depression, the study of eating behavior may provide an excellent source of objective information regarding the physiologic mechanisms that underlie particular psychiatric disorders.

Different patterns of appetite, eating behavior, and weight gain may reflect fundamental differences in brain biochemistry. Although there is considerable information on central neurotransmitters that may be involved in the regulation of appetite, much less is known about the neurophysiology of human mood states. Certain hypothalamic nuclei implicated in the control of feeding appear to be associated with reward or pleasure centers in the

brain (Morley and Levine 1983), and abnormal neurotransmission in these areas might produce concurrent disorders of eating habits and mood. Disturbed hypothalamic function has for some time been postulated as a fundamental defect in depression (Sachar 1976). Unfortunately, interpretation of data concerning such centers is complicated by the fact that the destructive lesions used to study them also damage other areas of the brain.

Several types of neurotransmitters are thought to play a role in appetite regulation, including the monoamines, opioid peptides, other neuropeptides, and gamma-aminobutyric acid. Disturbed regulation of these neurotransmitters has been postulated to be associated with depression, because abnormalities of both serotonin and norepinephrine metabolism, for example, seem to be present in depression (Murphy, Campbell, and Costa 1978; Jimerson 1984). Serotonin may also play a role in regulating carbohydrate intake, because ingestion of carbohydrate foods increases brain serotonin synthesis and release (Fernstrom and Wurtman 1972). Its agonist, fenfluramine, has been reported to decrease the proportion of dietary carbohydrates chosen by a group of so-called "carbohydrate cravers" (Wurtman and Wurtman 1983).

While decreased appetite and weight loss have long been recognized as prominent symptoms in endogenous depression (also known as endomorphogenic depression or melancholia), this weight loss is not usually observed in patients with nonendogenous, or neurotic, depression (Hopkinson 1981), whose symptoms instead are overeating, weight gain, and oversleeping (Davidson et al. 1982). In one study, patients observed to have an increased appetite during depression were predominantly female, neurotically rather than endogenously depressed, less severely depressed, and possibly more obese prior to the onset of depression (Paykel 1977). Although it might be beneficial for clinical and research purposes to categorize depressed patients into those who do and those who do not have increased appetite and weight gain, few investigators have evaluated these matters in depressed populations. In one attempt, decreased appetite was the symptom with the greatest value for discriminating between endogenous and nonendogenous types of depression (Feinberg and Carroll 1982), but in another, it was the severity of depression that determined whether weight was gained or lost (Weissenburger et al. 1986).

In a study of seasonal depression (with episodes during the winter but not in spring or summer), 71 percent of 125 patients experienced an appetite increase during their depressions, 17 percent a decrease, 9 percent a mixed reaction, and only 2 percent reported no appetite change (Rosenthal, Sack, Gillin, et al. 1984; Rosenthal, Sack, James, et al. 1984). Changes in weight,

as expected, paralleled appetite changes, with 72 percent reporting increased weight, 13 percent decreased weight, 3 percent a mixed picture, and 12 percent no change. These patients were predominantly women with a less severe, nonendogenous type of depression (Paykel 1977).

An association between weight change and sleeping patterns in depression has been noted (Crisp and Stonehill 1973). Depressives who lost weight differed from those who gained. The sleep duration for those losing weight was shorter, they awakened earlier, and their sleep was more disrupted. In a recent study, sleep-disrupted depression was associated with increased appetite and weight gain (Garvey, Mungas, and Tollefson 1984). In similar studies, overeating and weight gain were also associated with sleep disorders in patients with seasonal mood problems (Rosenthal, Sack, Gillin, et al. 1984).

Such patients also appear to crave carbohydrates (Rosenthal, Sack, James, et al. 1984), a symptom that has been associated with premenstrual tension, depression, and fluid retention in a survey of 300 nurses (Smith and Sauder 1969) and that has also been reported as a side effect of treatment with the antidepressant drug amitriptyline (Paykel, Mueller, and de la Vergne 1973). Studies of these and other relationships between mood and weight require further explanation and confirmation before their significance for obesity can be determined.

### Anorexia Nervosa

Anorexia nervosa is a condition characterized by extreme weight loss, amenorrhea, and a constellation of psychologic problems that have been described as "the relentless pursuit of thinness" (Bruch 1979; Garfinkel and Garner 1982). The term anorexia is technically incorrect because there is no loss of appetite in this condition until advanced stages of cachexia have been reached. The American Psychiatric Association's diagnostic criteria for anorexia nervosa are listed in Table 14-2.

The most common course of the disease is a single episode with full recovery, but anorexia nervosa can also be episodic or unremitting until it causes death by starvation (APA 1987). Thus, it may become necessary to hospitalize anorexics to prevent death. Followup studies indicate that rates of overall mortality are between 15 and 21 percent, among the highest levels recorded for psychiatric disorders (Herzog and Copeland 1985).

*Etiology.* Most teenage girls who develop anorexia nervosa are described as having been model children (Bruch 1973). Unlike many other psychiatric disorders, anorexia nervosa tends to occur in intact families and is

**Table 14-2**  
**Diagnostic Criteria for Anorexia Nervosa and Bulimia**

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*Anorexia Nervosa*

- A. Refusal to maintain body weight over a minimal normal weight for age and height, e.g., weight loss leading to maintenance of body weight 15 percent below that expected; or failure to make expected weight gain during period of growth, leading to body weight 15 percent below that expected.
- B. Intense fear of gaining weight or becoming fat, even though underweight.
- C. Disturbance in the way in which one's body weight, size, or shape is experienced, e.g., the person claims to "feel fat" even when emaciated, believes that one area of the body is "too fat" even when obviously underweight.
- D. In females, absence of at least three consecutive menstrual cycles when otherwise expected to occur (primary or secondary amenorrhea). (A woman is considered to have amenorrhea if her periods occur only following hormone, e.g., estrogen, administration.)

*Bulimia*

- A. Recurrent episodes of binge eating (rapid consumption of a large amount of food in a discrete period of time).
- B. A feeling of lack of control over eating behavior during the eating binges.
- C. The person regularly engages in either self-induced vomiting, use of laxatives or diuretics, strict dieting or fasting, or vigorous exercise to prevent weight gain.
- D. A minimum average of two binge eating episodes a week for at least 3 months.
- E. Persistent overconcern with body shape and weight.

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Source: American Psychiatric Association. 1987. *Diagnostic and statistical manual of mental disorders, third edition, revised*, pp. 67-69. Washington, DC: American Psychiatric Association. Copyright 1987, reprinted with permission.

often precipitated by seemingly minor events during adolescence. Various psychologic and physiologic hypotheses have been advanced to explain anorexia nervosa, but none is generally accepted. Most theories fall into one of four categories: (1) organic, (2) psychodynamic, (3) familial, and (4) sociocultural (Johnson, Thompson, and Schwartz 1984). The organic theories of anorexia nervosa are predominantly genetic and endocrinologic and have focused on the hypothalamic-pituitary axis (Gold et al. 1986). Support for a genetic basis of the disease is weak, but the disorder does occur with greater frequency among sisters, with a high incidence of various psychopathologies seen in relatives of such patients. Most theories of anorexia nervosa focus on psychologic trauma or unempathetic and overly domineering mothering as possible underlying causes of the disease (Johnson, Thompson, and Schwartz 1984), but these too are unproved. Sociocultural

theories suggest that the disease represents an exaggeration of the current inordinate weight consciousness of adolescent girls at a time when high-calorie foods are readily available and fewer calories are expended through exercise (Strober 1986).

*Clinical Features.* Typical symptoms of the disease include depression and obsessive-compulsive behaviors, although it is not clear whether these psychiatric problems precede weight loss or occur as a later result of semistarvation. Depression is often the first visible sign of anorexia nervosa, but sometimes it develops only after recovery. Abnormal hormonal patterns characteristic of starvation also occur (Mitchell 1986).

The clinical features of anorexia nervosa are personality characteristics such as rigidity or perfectionism, fear of obesity preceding the onset of the disorder, and the symptoms of starvation accompanying it. Serious body image disturbance is common, manifested by a lack of recognition of the severe emaciation and a belief that one is too fat. Individuals are usually preoccupied with food, thinking about it much of the time, preparing meals for others, and often engaging in bizarre eating rituals. Many anorexics engage in very extensive physical exercise. The disorder is also associated with a pervasive sense of personal ineffectiveness.

Anorexics have been divided into two general types, each comprising about half of the afflicted population. "Restricters," who confine their eating disorder to restriction of food intake, differ in a number of psychological dimensions from "bulimics," who engage in eating binges and subsequent vomiting and purging (see below). Bulimic anorexics tend to be older, manifest other impulsive behaviors such as kleptomania, alcohol and drug abuse, and sexual promiscuity, and have a poorer prognosis (Garfinkel and Kaplan 1986).

Anorexics typically deny their weight-losing behaviors and the existence of any illness and avoid treatment even when they have become severely emaciated. Family members often must insist upon medical treatment. Some anorectic persons effectively hide their weight-losing behaviors even after they are forced to seek medical assistance, which makes establishment of an accurate diagnosis difficult.

*Treatment.* Most persons with anorexia nervosa are resistant to entering treatment because of their fear of weight gain and so are usually brought by their families under protest. No treatment method has proved unusually effective. Because starvation plays such an important role in the clinical picture of anorexia nervosa, some clinicians begin treatment with a period

in the hospital designed to restore body weight. In patients 35 to 40 percent below normal weight, the provision of enteral nutritional support may be necessary (Winston 1987). Such treatment, which removes the patient from the environment supporting the illness, may permit the use of behavioral rewards for weight gain and provides the opportunity to work on issues of control. Some medications have proved helpful in the treatment of anorexia nervosa. These include cyproheptadine (an appetite stimulant), antidepressant medication, and chlorpromazine. Family therapy is increasingly used in treatment, a course that seems reasonable in view of the disordered functioning of many of these families. In addition, cognitive-behavioral therapy is useful for inpatient and outpatient therapy (Garner 1986).

### **Bulimia**

Bulimia is a disorder characterized by recurrent episodes of binge eating in which large amounts of food are consumed over a short period of time (Hawkins, Fremouw, and Clement 1984). These episodes are usually terminated by abdominal pain, self-induced vomiting, sleep, or the appearance of another person on the scene. Standard diagnostic criteria for this condition are given in Table 14-2.

As noted above, bulimia is present in half the patients with anorexia nervosa but in a much smaller percentage of obese patients. Recent interest in bulimia has focused on the disorder as it occurs among persons of normal weight. The severity of binge eating ranges from occasional episodes of morbid overeating at a party to the severe form of the disorder, bulimia nervosa, in which vomiting or purging follows frequent episodes of binge eating.

There are many similarities between patients with anorexia nervosa and those with bulimia. Both occur primarily in young women, although the onset of bulimia occurs in slightly older individuals; both may relate to the current preoccupation with thinness and dieting; and both usually begin with a period of dieting (Boskind-White 1986.)

First-degree relatives of bulimic patients have an increased prevalence of affective disorders, and probably of obesity as well. Thus, the symptoms of depression in the majority of bulimic patients and the presence of biologic markers of depression suggest that bulimia may represent a variant of a mood disorder (Agras and Kirkley 1986). Treatment for bulimia, still in the early stages of development, is cognitive-behavioral and pharmacologic. Behavioral treatments include modification of the behavioral program designed for obese patients and a combination of cognitive-behavioral and

insight-oriented approaches. Controlled clinical trials of these methods have produced encouraging results, although the size and number of studies are too small to permit firm conclusions about efficacy. The results of studies using antidepressant medications have similar difficulties, and conclusions are also uncertain (Wilson 1986).

### Pica

Pica is the intentional and compulsive consumption of substances not commonly regarded as food. It occurs worldwide and is common among people of either sex and of all ages and races. Because pica is often associated with nutritional deficiencies or toxicities, it is of special concern among young children and pregnant women (Danford 1982).

Pica is usually classified into four categories distinguished by the substance consumed. Geophagia is the consumption of earth and clay; amylophagia is the consumption of starch and paste; and pagophagia is the eating of ice. The fourth (miscellaneous) category includes consumption of ash, chalk, antacids, paint chips, plaster, wax, and other substances (Crosby 1976).

The origin of this practice is poorly understood. One common hypothesis is that people are driven to consume these substances by nutritional deficiencies, but pica is not necessarily correlated with poor nutritional status (Grivetti 1978). Theories that it represents a method for reducing stress, preventing nausea during pregnancy, or obtaining calcium, iron, or other nutrients are equally unproven. Despite recent reports of similarities between clay and standard antidiarrheal medications (Vermeer and Ferrell 1985), geophagia has not been shown to prevent diarrheal diseases.

The nutritional hazards most frequently associated with pica are lead poisoning and iron deficiency anemia, but whether pica is a cause of these problems, their result, or both is as yet uncertain (Danford 1982).

## Effects of Foods and Nutrients on Behavior

### Normal Behavior

At least three types of food constituents have been evaluated in controlled studies for their possible behavioral effects on healthy people: amino acid precursors of brain neurotransmitters, other protein and carbohydrate foods, and caffeine.

*Amino Acid Neurotransmitter Precursors.* Certain large neutral amino acids found in substantial amounts in protein-containing foods are the

precursors of important brain neurotransmitters. These amino acids are transported across the blood-brain barrier into the brain where they could affect behavior. One of these is tryptophan, the precursor of the neurotransmitter serotonin. When pure tryptophan is administered to experimental animals, it increases brain tryptophan and serotonin levels. Tryptophan supplements have been reported to affect normal human mood, sleep, and pain sensitivity, functions believed to be regulated in part by serotonin neurons (Hartmann, Spinweber, and Ware 1976; Hartmann 1983; Lieberman et al. 1983; Seltzer et al. 1982; Young, 1986). They have also been reported to modify the appetites of certain patients (Wurtman, Hefti, and Melamed 1981). Tyrosine, the precursor of the neurotransmitters dopamine and norepinephrine, has also been reported to affect the neurotransmitter biosynthesis.

There is some evidence that certain doses of tryptophan are sedative and/or hypnotic. Investigators have demonstrated the effects of gram quantities of tryptophan on human alertness, as measured by self-report mood questionnaires, and on the period of time needed to fall asleep (Hartmann 1983). These effects are consistent with reports implicating brain serotonin neurons in the regulation of sleep. Although tryptophan does not appear to be as potent as prescription hypnotic drugs, it may have some clinical utility as a treatment for mild insomnia.

*Protein and Carbohydrate Foods.* In experimental animals, high-protein and high-carbohydrate meals have opposite effects on brain levels of tryptophan and its neurotransmitter product, serotonin. Meals of relatively high carbohydrate proportions tend to increase brain levels of tryptophan and serotonin, even though protein contains tryptophan but carbohydrate does not (Wurtman, Hefti, and Melamed 1981). Because serotonin-responsive neurons participate in the onset and maintenance of sleep, meals with a high ratio of carbohydrate to protein would be expected to increase sleepiness. The results of the few behavioral tests of this hypothesis have not been definitive (Lieberman, Spring, and Garfield 1986).

*Caffeine.* About three-fourths of the caffeine consumed in the United States is in coffee. Of the types of coffee regularly consumed in the United States, drip-method coffee usually contains the highest amounts of caffeine, about 112 mg per cup, and instant coffee the least, about 60 mg per cup. A cup of tea made with a tea bag contains about 42 mg of caffeine, and a typical serving (12 oz) of a cola beverage contains about 38 mg of caffeine (Roberts and Barone 1983).

Caffeine classically has been considered to have stimulant-like effects on behavior, and it has clear behavioral and pharmacologic effects at high doses. However, considerable controversy exists concerning its effects at the lower doses present in ordinary food and drink. The scientific literature on the behavioral effects of caffeine is quite extensive and quite contradictory. Many studies have failed to detect any effects on mood, such as increased alertness and improvements in vigilance, with even high doses of caffeine, while others have shown rather large effects as measured by tests of performance (Curatelo and Robertson 1983; Raebel and Black 1984). The most likely effects of moderate doses of caffeine are an improvement in aspects of performance (such as vigilance) and an increase in feelings of alertness, but these effects almost certainly depend on the individual's usual level of caffeine intake and inherent sensitivity to this substance.

#### Childhood Hyperactive Behavior

Numerous unconfirmed reports have found effects of various food and food constituents on children. Some of the more common ones are discussed below. Behavioral responses to food allergies are reviewed in the chapter on infections and immunity.

*Food Colors.* The childhood behavior problem most commonly discussed in relation to nutrition is termed Attention Deficit Hyperactivity Disorder, or, more popularly, hyperactivity. This condition is characterized by problems of inattention, excessive motion, impulsivity, learning disabilities, and related problems of conduct.

The contention that food additives, especially artificial dyes and colors, are causes of childhood hyperactivity (Feingold 1975) has stimulated many studies of the behavioral toxicity of food additives in children, notwithstanding the implausibility that such a wide variety of chemical structures would lead to a common result. A statistical analysis of the results of 23 of these studies concluded that artificial colors have, at most, a negligible effect on the behavior of children (Kavale and Forness 1983). This conclusion is consistent with that of other reviewers (Mattes 1983; Rumsey and Rapoport 1983; NIH 1982; Lipton and Golden 1984). At most, a few predisposed preschool and school-aged children may be adversely affected by artificial colors (Connors 1984b; Rimland 1983; Weiss et al. 1980). Because it remains possible that a few children may respond to diets that eliminate food colors, and such diets are not harmful, there is no reason to advise against therapeutic trials of food additive avoidance in individual cases.