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Chapter 17

Alcohol

Inflaming wine, pernicious to mankind,
unnerves the limbs,
and dulls the noble mind.

Homer
The Iliad, VI 261 (850 B.C.)

Introduction

Alcohol (ethanol) is of importance to nutrition because it provides energy and because its ingestion can affect the requirements for and the intake, digestion, absorption, transport, storage, metabolism, and excretion of many other nutrients. Few generalizations can be made regarding the effects of alcohol on nutritional status because these depend on complex interactions between the type, quantity, and duration of alcohol consumed and the overall nutrient intake from the diet. These interactions are highly dependent upon behavioral and socioeconomic factors and predisposing genetic factors. Chronically excessive alcohol use is often associated with symptoms, signs, and biochemical evidence of nutritional deficiencies, but these are largely the secondary effects of reduced consumption of other kinds of food and drink. The effect of moderate or occasional drinking is less well documented.

Historical Perspective

Fermented beverages have been consumed in most societies since antiquity, in part because fermentation provided one of the earliest methods of food preservation (Ghalioungui 1979). In some societies, indigenous foods that contained alcohol as a result of fermentation may have offered protection against the development of deficiencies of vitamins, amino acids, and other essential nutrients (Steinkraus 1979; Darby 1979).

Socioeconomic factors have always affected the pattern of alcohol abuse and its complications in different societies. In ancient Greece, for example,

wine drinking was confined largely to the upper classes, and beer drinking to the lower classes. In industrialized England of the 18th century, gin was the drink of the poor, while port was drunk by the affluent, whose episodes of gout were attributed to the contamination of port with lead.

Distilled spirits, in contrast to fermented foods and beverages, provide concentrated sources of alcohol and calories largely without other nutrients. Although the process of distillation was known at least as early as the fourth century B.C. (Ghalioungui 1979), the consumption of distilled spirits tended for centuries to be limited to the more affluent who also had access to varied diets. In Western Europe, distilled alcoholic beverages were not widely available to the poor until the Industrial Revolution. In England, government policies that liberalized controls on production and distribution of alcoholic beverages led to the widespread “gin epidemic,” and further legislation was then required to prohibit sales and prevent adverse consequences (Roe 1979).

Out of this history grew a series of health and social reform movements, including the prohibitionist movement in the United States. In their efforts to change society’s behavior, most health reform advocates focused on the extreme consequences of alcohol abuse, using selected medical and scientific data to support their arguments (Whorton 1982). This, in turn, led to distortions in understanding the nutritional consequences of alcohol use. For example, many studies examined the habits of accessible derelict or vagrant alcoholics or provided descriptive reports that were easier to obtain than biologic determinations of specific pathophysiologic mechanisms.

The attitudes toward alcohol of the 19th and early 20th centuries remain prevalent today, and many people are uncertain and misinformed about interactions between alcohol and nutrition. Recent efforts from the U.S. Department of Health and Human Services have focused on providing the scientific community and the public with current information about this area. Thus, alcohol consumption has been identified as a dietary practice in need of change in several Federal publications. Examples include those listed below.

Healthy People: The Surgeon General’s Report on Health Promotion and Disease Prevention, 1979. This report recommends reducing misuse of alcohol. It includes information on prevention programs that stress the importance of educating the public on the effects of alcohol consumption,

altering the social climate of its acceptability, reducing individual and social stress factors that might increase consumption, and enforcing existing laws (DHEW 1979).

Nutrition and Your Health: Dietary Guidelines for Americans (second edition), 1985. One of the seven recommendations is "If you drink alcohol, do so in moderation." The discussion of this guideline emphasizes the caloric contribution of alcohol to the overall diet, especially for individuals who are overweight, but notes that one to two drinks daily appear to cause no harm in adults. It warns pregnant women that excessive alcohol consumption may cause birth defects at the same time that it acknowledges that the "level of consumption at which risks to the unborn occur has not been established" (USDA/DHHS 1985).

Promoting Health/Preventing Disease: Objectives for the Nation, 1980. These health objectives include specific recommendations for methods to reduce deaths due to alcohol-related behavior, accidents, and disease by the year 1990 (DHHS 1980).

Promoting Health/Preventing Disease: Public Health Service Implementation Plans for Obtaining Objectives for the Nation. This report identifies priority objectives for prevention of alcohol misuse and outlines Federal efforts in education, grant support, technical assistance, economic incentives, and research and surveillance measures aimed at achieving them. It also assigns to specific agencies the primary responsibility for each of the implementation steps and provides data on the anticipated date of initiation of each step (DHHS 1983).

The Sixth Special Report to the U.S. Congress on Alcohol Abuse and Health from the Secretary of Health and Human Services. This report provides the most comprehensive statement to date of current knowledge in the epidemiology of alcohol abuse and alcoholism; the genetics, psychobiologic effects, and medical consequences of alcoholism; the effects of alcohol on pregnancy outcome; the adverse social consequences of alcohol abuse; trends in treatment, research, and practice; and perspectives on prevention (DHHS 1987).

The 1990 Health Objectives for the Nation: A Midcourse Review, 1986. This report provides data on progress toward achieving the 1990 objectives for prevention of deaths due to alcohol misuse. Although it notes impressive achievements in reducing alcohol-related motor vehicle accident

fatalities and deaths due to cirrhosis, it stresses the importance of multi-disciplinary approaches to prevention of the adverse health consequences of individual behavior associated with alcohol consumption (DHHS 1986).

Significance for Public Health

Although the nutritional elements of the public health impact of alcohol abuse are still being defined, in and of itself, misuse of alcohol is one of the most preventable health problems in the United States. As mentioned above, prevention of the adverse consequences of alcohol misuse is a major health objective for the year 1990 (DHHS 1986). Excessive alcohol intake is a prominent contributor to 4 of the 10 leading causes of death in the United States—cirrhosis of the liver, motor vehicle and other accidents, suicides, and homicides (NCHS 1986). As discussed in the cancer chapter of this Report, chronic alcohol abuse also increases the risk for oral, esophageal, liver, and other types of cancer.

In 1980, it was estimated that nearly 20,000 deaths could be directly attributed to alcohol use, from alcoholic liver disease, alcoholism, alcoholic psychosis, alcoholic cardiomyopathy, alcoholic gastritis, alcoholic polyneuropathy, nondependent use of alcohol, and accidental poisoning by alcohol. Almost 78,000 fatalities could be indirectly attributed to alcohol use (Ravenholt 1984). In 1983, about 42 percent of all motor vehicle deaths were alcohol related. Problems such as homicides and automobile accidents that are indirectly related to alcohol consumption have the highest rates among young adult males ages 18 to 24. Excessive alcohol intake during pregnancy is estimated to cause birth defects in 1,400 to 2,000 babies annually. The costs of the medical and social consequences of excessive alcohol intake were estimated to be \$117 billion in 1983 (DHHS 1987). In the late 1970's, 19 percent of adolescents reported problems related to drinking alcohol. Among adults over age 18, approximately 10.6 million are alcohol dependent and another 7.3 million experience alcohol-related adverse consequences (DHHS 1986).

Recent trends suggest that both the intake of alcohol and its adverse health consequences are declining in this country. Overall per capita ethanol consumption increased annually from 1977 to 1980, reached a plateau in 1980 and 1981, and then began an annual decline until 1985, when it was slightly below the 1977 level (Laforge et al. 1987). Beer consumption in 1985 was 3 percent above the 1977 level; wine consumption per capita increased over the entire period of 1977 to 1985, and in 1985 it was 31 percent higher than in 1977. Per capita consumption of spirits, however, declined over this

entire period, and in 1985 was 15 percent below the 1977 level (Laforge et al. 1987). There has also been a slow decline in deaths attributable entirely to alcohol-related causes since 1980 (Berkelman et al. 1986).

Scientific Background

Quantitative Aspects of Alcohol Consumption Among Individuals

Different units are used to record the quantity of alcohol intake among individuals. Commonly used units are grams or ounces of pure alcohol. There are approximately 15 g of alcohol in each of the following standardized drinks: 1-½ oz of 80 proof liquor, 5 oz of table wine, and 12 oz of beer (Pennington and Church 1985).

Consumption of Alcoholic Beverages

The proportion of U.S. adults who drink alcoholic beverages depends on both the region of the country and sociocultural background. Estimates of the various categories of drinkers in the population are usually based on survey information, and the estimated quantities consumed are often based on self-reports. Such surveys indicate that about 33 percent of the general population say they do not drink alcohol at all; 34 percent are light drinkers, who say they drink from one to three drinks per week; 24 percent are moderate drinkers, who report consuming fewer than two drinks a day; and 9 percent are heavy drinkers, who average two drinks a day or more (Moore and Gerstein 1981). In studies of the heaviest drinkers (i.e., alcohol abusers and alcoholics), the quantity of alcohol consumed is very difficult to ascertain, and clinical criteria for the diagnosis of alcoholism usually must depend on factors other than alcohol intake information (Task Force on Nomenclature and Statistics 1980).

Although this classification into light, moderate, and heavy drinkers seems reasonable and is used in this chapter, it is arbitrary and not universally accepted. For example, in the Honolulu Heart Study, consumption of two to three drinks per day was considered moderate, but, according to the above classification, it would be considered heavy consumption. The use of the Michigan Alcoholism Screening Test (a short questionnaire shown to be efficient in identifying alcoholics) and other alcoholism screening tests such as the Self-Administered Alcoholism Screening Test (Swenson and Morse 1975) may prove important in separating alcoholic from non-alcoholic persons by standard criteria rather than by self-reports.

Surveys also reveal wide regional variations, especially among the percentage of individuals who say they abstain completely from alcohol (e.g., 14

percent in western New York State, 32 percent in San Francisco). The percentage of heavy drinkers, adults who report drinking two drinks or more a day, ranges from 9 percent nationwide to 23 percent in Boston and 24 percent in western New York State (Barnes and Russell 1978; Wechsler, Demone, and Gottlieb 1978).

The frequency of alcohol consumption reported by high school students differs from that reported by adults in national surveys. In a 1985 nationwide survey of about 17,000 high school seniors conducted by the National Institute on Drug Abuse, only 8 percent of seniors said they had never used alcohol. Nearly 5 percent of seniors drank every day, and 37 percent reported episodes of heavy drinking (five or more drinks per occasion) during the previous 2 weeks (DHHS 1987).

Categories of Beverages Consumed and Types of Drinkers

Since 1934, the consumption of alcohol in the United States, based on statistics from commodity sales, has increased almost continuously, and in 1984 the annual per capita intake in terms of absolute alcohol was 2.65 gal/person 14 years of age or older (DHHS 1987). As one-third of the U.S. adult population abstains from alcohol, those who drink consume an average of 1.3 oz of absolute alcohol per day. These distinctions become even more important when examined more closely: the heaviest drinking 5 percent of the population accounts for about 50 percent of total alcohol consumption, and the heaviest drinking 33 percent accounts for over 95 percent of total alcohol consumption. Given the presumed absence of impact of alcohol on the nutrition of the one-third of the population who abstain, and negligible impact on that of the one-third who are light drinkers, the group of primary interest from this perspective is this latter group.

Over time, consumption of the various types of alcoholic beverages has changed. Beer is now the most prevalent alcoholic beverage consumed, accounting for almost 50 percent of the alcohol consumption in the United States. Distilled spirits account for nearly 39 percent and wine for 12 percent. The wine industry was virtually dismantled as a result of Prohibition but has recently made significant advances in recapturing part of the market and appears to be continuing to increase its share of sales. The types of distilled spirits consumed have also changed with time; in recent decades, there has been a shift from whiskey to other forms of distilled spirits, such as gin and vodka.

Alcohol Abuse and Alcoholism

The essential feature of alcohol abuse is a pattern of pathologic moderate to heavy alcohol use for at least a month that causes impairment in social or

occupational functioning. The accepted diagnostic criteria, as defined in the *Diagnostic and Statistical Manual of Mental Disorders (DSM-III)* of the American Psychiatric Association, are useful for such patterns of chronic, pathologic alcohol consumption (Task Force on Nomenclature and Statistics 1980). DSM-III criteria describe three main patterns of alcohol abuse: (1) regular drinking of large amounts, (2) regular heavy drinking but limited to weekends, and (3) episodic binges of heavy daily drinking lasting weeks or months, interrupted by long periods of sobriety. These criteria for the diagnosis of alcoholism also include either a pattern of pathologic alcohol use or impairment in social or occupational functioning and either central nervous system tolerance or withdrawal symptoms.

The number of Americans with alcohol problems is estimated to be as high as 18 million (DHHS 1987). In an outpatient medical setting using the Self-Administered Alcoholism Screening Test, the prevalence of abstainers was 13 percent and the prevalence of possible or probable alcoholism was 5.4 percent (Hurt, Morse, and Swenson 1980). The prevalence of alcoholism in general hospitals, estimated by using the Michigan Alcoholism Screening Test, was 18 percent for men and 5.5 percent for women (Moore 1971). In emergency room patients, the prevalence of alcoholism may be as high as 30 percent of patients during the evening hours (Rund, Summers, and Levin 1981). However, precise statistics for the prevalence of alcoholism are not available now, nor are they likely to become so in the near future, because alcoholism is diagnosed infrequently, especially in its earlier stages.

Physiology of Alcohol Use

Absorption. Alcohol is readily absorbed from all levels of the gastrointestinal tract, and blood concentrations rise rapidly after ingestion. Overall absorption of alcohol depends partly on the rate of gastric emptying. Food, particularly fat, delays gastric emptying and stimulates gastric secretion, thereby diluting the concentration of alcohol and blunting its rapid absorption into blood.

Absorption across the gut mucosa appears to occur as a result of passive diffusion. The rate of absorption varies as a function of the concentration gradient, the area and permeability of the absorbing surface, and the volume of regional blood flow (Kalant 1971). During the first 15 minutes after oral administration, maximum alcohol levels are rapidly found in the stomach, duodenum, and proximal jejunum; at 90 minutes, peak levels are found in the mid-jejunum. Alcohol levels in the ileum parallel those in the blood, suggesting that the ileal concentration results from passage of alcohol from the blood back into the lumen rather than from transit along the

small bowel (Halsted, Robles, and Mezey 1973). Infusion of alcohol directly into the duodenum, the first segment of the small intestine where most nutrients are absorbed, causes blood levels of alcohol to rise as high as those following direct intravenous administration, and these levels rise much more rapidly than those that occur when alcohol is infused into the stomach.

Distribution. Once absorbed, alcohol diffuses rapidly across capillary and other cell membranes and is distributed uniformly throughout all extracellular and intracellular body water. At equilibrium, the concentration of alcohol in all tissues is proportional to the tissue water content, so that differences in the volume of body water among people can help to explain why some individuals are apparently more susceptible than others to the pharmacologic and toxic effects of alcohol. Smaller, lighter people are more susceptible to the effects of alcohol than larger, heavier people simply because their fluid volume is less. For a given body weight, the volume of body water is less in females than in males, and less in older people than in younger people; therefore, a given dosage of alcohol can be expected to produce higher blood concentrations in women and older persons and to affect them more (Vestal et al. 1977).

Metabolism. Alcohol is metabolized by the liver first to acetaldehyde, then to acetate, and, finally, to carbon dioxide and water. Three separate enzyme systems can account for the initial oxidation of alcohol to acetaldehyde (Badawy 1978; Lieber 1984; Pirola 1978). Quantitatively, the most important system is that involving alcohol dehydrogenase, an enzyme found in the cytoplasm of liver cells. The acetaldehyde produced is rapidly oxidized to acetate by the enzyme acetaldehyde dehydrogenase, resulting in little accumulation of acetaldehyde in either the liver or blood.

Alcohol and acetaldehyde dehydrogenases exist in several forms, which oxidize alcohol and acetaldehyde at different rates. These enzyme variations may account in part for observed differences in rates of alcohol metabolism among individuals of different genetic backgrounds and may help explain, for example, why some people of Oriental or American Indian heritage have lower tolerances for alcohol consumption (DHHS 1985).

A second enzyme system located in the liver, called the microsomal ethanol-oxidizing system (MEOS), probably plays a relatively minor role in alcohol metabolism, at least at low concentrations, but it has significant activity at higher concentrations of alcohol. This system can be stimulated by chronic exposure to high levels of alcohol. This is associated with cross induction of the metabolism of other drugs, such as the anticonvulsants

hydantoin and phenytoin, the barbiturates, the sedative meprobamate, the tricyclic antidepressants, the phenothiazine tranquilizers, and oral anticoagulants (Pirola 1978). The inducibility of this system results in interactions between alcohol and other drugs that may alter the rate of disappearance of alcohol and other drugs from the body (see chapter on drug-nutrient interactions) and may activate a variety of hepatotoxic agents and carcinogens.

A third enzyme system, the catalase system, appears to be of minor importance in the metabolism of alcohol and is not stimulated by chronic consumption of alcohol. Several other minor metabolic pathways, such as glucuronide and sulfate conjugation and fatty acid esterification, have been identified, but their importance remains unclear.

Some of the metabolic consequences of alcohol consumption can be explained by shifts in oxidation-reduction balance that occur as a consequence of ethanol oxidation. Reduced nicotinamide-adenine dinucleotide (NADH) is produced as a result of the oxidation of ethanol by the alcohol dehydrogenase enzyme system and by the further oxidation of acetaldehyde to acetate. An increased supply of NADH relative to NAD may affect carbohydrate, lipid, and protein metabolism and account for alterations in the hepatic metabolism of steroids, biogenic amines, some drugs, and, perhaps, even some toxins. Other deleterious effects of alcohol ingestion have been attributed to aldehyde or its cytotoxic interactions with body proteins (Wickramasinghe, Gardner, and Barden 1987).

Excretion. More than 95 percent of the alcohol ingested is oxidized in the liver to carbon dioxide and water, and the remainder is excreted in the urine, feces, perspiration, and expired air.

Key Scientific Issues^a

- Effect of Alcohol on Nutritional Status and Energy Balance
- Role of Alcohol in Diseases of the Liver
- Role of Alcohol in Diseases of the Nervous System
- Role of Alcohol in Cardiovascular Diseases
- Role of Alcohol in Reproductive Disorders

^aThe role of alcohol in cancer, diabetes, and osteoporosis is not covered here but is reviewed in the chapters devoted to those conditions.

Effect of Alcohol on Nutritional Status and Energy Balance

Whether alcohol injures organs directly because of its toxic effects on tissues or indirectly because of the nutritional deficiencies it causes has been a subject of debate for decades. In the mid-18th and 19th centuries, alcohol was considered a toxin and was classified with other poisons, such as arsenic, mercury, and ergot. The discovery of vitamins in the 20th century gave momentum to studies of the association between alcohol consumption and nutritional status, especially between 1920 and 1940. Similarities between complications of alcoholism such as those affecting the nervous system and various vitamin deficiency states led to the belief that most physical manifestations of alcoholism had a nutritional basis.

The malnutrition observed in alcoholics was proposed as the primary reason for the organ and tissue damage found so frequently in this population. Patients with severe alcoholic liver disease and ascites who were given a diet high in protein and B-complex vitamins had a better prognosis than those provided with a regular diet, and there was a significant association between the occurrence of nutritional deficiency and the occurrence of alcoholic cirrhosis (Patek and Post 1941). This finding suggested that eating a diet high in protein and B-complex vitamins would protect an individual from the effects of alcohol. Because so many nutritional deficiencies were observed in derelict alcoholics, the assumption naturally was made that similar but milder nutritional deficiencies were present in all other alcoholics.

By the early 1960's, however, many of the clinical consequences of alcoholism were shown to be the direct toxic effects of alcohol itself (Lieber 1966). Most recent evidence points to the toxic effect of alcohol or its metabolic byproducts as the primary mode of liver injury. In addition, possible genetic differences in predisposition to the direct toxic effects of alcohol have been recognized. The effects of alcohol on the nutritional status of nonalcoholic social drinkers remains to be defined.

Excessive alcohol intake increases the risk for malnutrition through a variety of pathophysiologic mechanisms, including direct and indirect alterations in both nutrient intake and requirements, digestion, absorption, transport, storage, metabolism, and excretion. For any nutrient, alcohol may affect one or more of these processes. The following sections summarize what is known about the most important interactions between alcohol and each major nutrient. The reported effects of alcohol vary greatly, depending on how much and how long it is consumed. In some cases,

marked differences among animal species have led to apparently contradictory and confusing experimental results.

Energy

At the turn of this century, Atwater determined that ethanol provides 7 kcal/g, and ever since then this amount has been used as the basis for calculating the contribution of alcohol to total energy intake (Passmore 1979). The use of this conversion factor assumes that the energy in alcohol is fully available to the body. Based on this conversion factor, only 3 to 7 percent of the calories consumed by all Americans over the age of 14 are derived from alcohol, but if nondrinkers are excluded, alcohol provides over 10 percent of the calories consumed by adult drinkers in the United States (Williamson et al. 1987).

Many observations suggest that the caloric contribution of alcohol is more complex than can be explained by the Atwater conversion values, and therefore, the concept of alcohol and energy balance has recently been revised. If, for example, alcohol provides an average of 20 percent of the calories in the diet of the average drinking American adult, then many alcoholics consuming much larger amounts ought to be obese. Instead, national data indicate that despite higher energy intakes, drinkers are no more obese than nondrinkers (Gruchow et al. 1985). Alcoholics tend to lose weight over time, and abstinence is commonly associated with a gain in weight (Morgan 1982). In one study, eight malnourished alcoholic patients lost an average of 9 lb while they were consuming at least 200 g (about 1,400 kcal) of ethanol daily for a minimum of 3 weeks. They then gained an average of 7 lb after abstaining from alcohol for 2 weeks and eating an adequate diet (Halsted, Robles, and Mezey 1971). In another study, alcohol was associated with a substantial reduction in weight among women (Williamson et al. 1987). The conclusion usually drawn from such studies is that alcoholics do not consume an adequate diet, which is often true.

Increasing alcohol intake while maintaining an adequate diet, however, does not necessarily lead to a weight gain. For example, when 56 alcoholic patients were admitted to a hospital and fed a diet of 2,600 kcal, those who received an additional 256 g, or 1,800 kcal, of alcohol each day experienced no greater weight gain than those who received the diet of 2,600 kcal alone (Mezey and Faillace 1971). Similarly, when alcoholic patients were fed diets supplemented with 2,000 kcal of alcohol, no consistent change in body weight occurred, but when the 2,000 extra kcal were provided as chocolate, there was a consistent weight gain (Pirola and Lieber 1972). This study showed that substitution of an equal number of kcal of alcohol for carbohy-

drate at a level of 50 percent of total kcal was followed by weight loss, and the same result was obtained at 25 percent of total kcal (McDonald and Margen 1976).

The mechanisms accounting for the apparent inefficiency in conversion of potential energy from alcohol are complex and incompletely understood (World et al. 1984). Metabolic rate, as measured by oxygen consumption, is higher in rats fed alcohol than in those fed the same number of calories as carbohydrate (Pirola and Lieber 1972). Alcohol increases oxygen consumption in normal human subjects and is reported to increase it even more in alcoholic persons (Lieber 1984).

Several mechanisms have been proposed to explain how the oxidation of alcohol could result in "inefficient" energy transfer. The MEOS pathway of alcohol oxidation requires a reducing equivalent, from nicotinic adenine dinucleotide phosphate (NADPH), and results in the production of heat rather than high-energy phosphate bonds. Subsequent reduction of NAD to NADH during the oxidation of acetaldehyde to acetate would result in no net change in total reducing equivalents. Furthermore, the alteration in the ratio of NADH to NAD in the cell cytoplasm might temporarily shift various substrates to the more reduced state and thereby impair the flux of reducing equivalents into the mitochondria for subsequent oxidative phosphorylation. A third mechanism could result from abnormalities in mitochondrial membranes, but this would be more likely to occur in alcoholic individuals and therefore would not explain the acute increase in metabolic rate following alcohol ingestion in normal drinkers (Lieber 1984).

Recent studies on nonshivering and postprandial thermogenesis (Rothwell and Stock 1986) may cast some light on another possible mechanism to explain the observed rise in oxygen consumption following alcohol ingestion. Landsberg and Young (1984) have shown that sympathetic nervous activity plays a central role in the increased heat production following feeding and the reduced heat production associated with starvation. Alcohol ingestion, especially in large amounts, has been associated with increases in catecholamine levels, specifically norepinephrine (Beilin and Puddey 1984).

The caloric value of alcohol (at least when ingested in large amounts) cannot be regarded as equivalent to the caloric value of other dietary sources of energy, as measured by the synthesis of high-energy phosphate bonds. Loss of weight when alcohol is substituted isocalorically for carbohydrate and the failure to gain weight when an otherwise adequate diet is supplemented with alcohol have led to the suggestion that alcohol-derived

calories should be disregarded completely as an energy source in predicting dietary-induced changes in weight (World et al. 1984), but the factors may work differently by pattern of alcohol use.

For moderate drinkers, another factor that must be considered is the effect of alcohol on caloric intake from other foods, especially in individuals who are chronically dieting. Recently, the concept of the "restrained eater" has been developed (Herman and Polivy 1980). Briefly stated, the restrained eater attempts to maintain body weight at a lower-than-set-point level by chronically undereating. When obligated to eat, or when the self-restraint is temporarily suspended, rebound overfeeding occurs. This hypothesis is supported by studies in which volunteers were asked to taste an appealing food after some of them had been provided a preliminary dose of alcohol. Among restrained eaters, alcohol increased the amount of food consumed, but alcohol did not affect the subsequent intake of unrestrained eaters (Polivy and Herman 1976a, 1976b).

These findings are consistent with the frequent clinical observation that obese people have greater difficulty dieting and maintaining lower body weights if they continue to drink alcohol. Whether the signals involved are psychologic or biologic remains to be determined. Animal studies suggest that biologic signals may be responsible because similar overeating can be demonstrated in animals who have been underfed before testing (Herman and Polivy 1980).

Lipids

Abnormalities in lipid metabolism are common in both alcoholics and mild to moderate drinkers (Janus and Lewis 1978). Body fat stores usually are depleted when weight loss results from poor diets. Alcoholics with chronic pancreatitis and pancreatic insufficiency may not digest fat efficiently because of a reduced output of pancreatic lipases, the primary enzymes for digesting lipids. Hepatic insufficiency can also cause fat malabsorption due to impaired secretion of the bile salts that emulsify fats and make them more digestible and absorbable. In addition, abnormalities in intestinal mucosal cells have been found to occur as a direct result of alcohol toxicity and, more commonly, as an indirect result of malnutrition (Green 1983).

Carbohydrate

Differences among animal species and differences in experimental conditions have led to apparently conflicting results regarding the effects of alcohol on carbohydrate metabolism (Marks 1978). Two major syndromes warrant discussion: (1) impaired glucose tolerance and (2) alcohol-induced

hypoglycemia. These effects are of special concern in patients with diabetes mellitus.

Impaired Glucose Tolerance. Impaired glucose tolerance is most easily demonstrated if the dosages of alcohol are sufficient to increase sympathetic nervous system activity and, perhaps, to decrease peripheral uptake of glucose. Doses of alcohol sufficient to produce blood alcohol levels of approximately 80 mg of alcohol per 100 ml of blood and mild euphoria do not activate the sympathetic nervous system and do not greatly affect blood glucose concentrations (Marks 1978). Indeed, some studies have shown an improvement in glucose tolerance following alcohol ingestion. Mild fasting hyperglycemia has been observed in more than 20 percent of middle-class alcoholics at the time of admission for alcoholism treatment (Hurt et al. 1986). It appears that the conditions under which alcohol is consumed, the associated dietary intake, the level of alcohol consumption, the history of alcohol consumption, and the presence of organ pathology such as hepatic insufficiency are responsible for the wide variation in the effects of alcohol on glucose tolerance.

Hypoglycemia. A rare but often fatal complication of alcohol abuse is profoundly low blood sugar (Williams 1984). This condition usually occurs in the context of acute alcohol ingestion after minimal food intake for several days or more. Victims are found stuporous or deeply comatose, with blood glucose levels below 20 mg/100 ml of blood. It has been proposed that alcohol blocks gluconeogenesis by metabolic shifts that result in reduced conversion of gluconeogenic precursors into glucose and glycogen. In the absence of glycogen stores, profound hypoglycemia occurs. Alcohol-induced hypoglycemia can be produced experimentally in normal healthy volunteers by inducing them to fast for 36 to 72 hours before administering alcohol (Freinkel et al. 1965). Obesity, or pretreatment with corticosteroids, tends to blunt the hypoglycemic effect of alcohol. In contrast, malnutrition, adrenocortical insufficiency, thyrotoxicosis, and consumption of diets high in protein and low in carbohydrate increase sensitivity to the hypoglycemic effects of alcohol ingestion.

Diabetes Mellitus. The effects of alcohol on the management of diabetes have been reviewed (McDonald 1980). Of particular importance are alterations in the metabolism of oral hypoglycemic drugs. Sulfonylurea compounds stimulate insulin release from the pancreatic beta cells and, in turn, inhibit hepatic gluconeogenesis, thus augmenting the hypoglycemic effects of alcohol. Prolonged alcohol ingestion can stimulate hepatic metabolic pathways responsible for drug metabolism and thereby shorten the half-life

of certain drugs (see chapter on drug-nutrient interactions). In such cases, the effectiveness of these drugs diminishes with prolonged alcohol ingestion.

For example, alcohol can impair the action of oral hypoglycemic agents. Both phenformin—a drug no longer available in the United States, but used elsewhere—and alcohol inhibit the decarboxylation of pyruvate to acetyl-CoA and favor the reduction of pyruvate to lactic acid, which would tend to increase the risk for lactic acidosis. Patients who take chlorpropamide experience flushing and other symptoms similar to those induced by disulfiram (Antabuse) when alcohol is consumed. Evidence indicates that there are genetic predispositions to this drug-alcohol interaction. Finally, alcohol-related deteriorations in judgment may be especially serious for persons with insulin-requiring diabetes if inebriation leads to errors in insulin dosage, administration, and diagnosis. In rare cases, profound hypoglycemia with permanent neurologic deficit has occurred.

Protein

Data from animal studies suggest that low-protein diets, sufficient to produce protein malnutrition, affect the biosynthesis of the alcohol dehydrogenase enzyme system and the availability of other cofactors required for alcohol oxidation (Orten and Sardesai 1971). Furthermore, the ability of rats, and possibly humans, to oxidize alcohol varies with both the amount of dietary protein and the types of protein ingested. For example, egg and milk proteins have been found to be more beneficial in maintaining normal rates of alcohol oxidation than proteins from cereals or other plant sources. When measured by indicators such as degree of inebriation and incidence of death due to alcohol intoxication, however, a mixture of proteins from vegetable sources has been shown to provide protection comparable to that from animal protein alone (Lucas, Ridout, and Lumchick 1968).

Water-Soluble Vitamins

Thiamin. Poor dietary intake and poor selection of foods, frequently seen in alcoholics, reduce thiamin intake. Alcohol may interfere with absorption of thiamin, with its activation to thiamin pyrophosphate, or with the ability of thiamin pyrophosphate to combine with the enzymes for which it is a cofactor (Hoyumpa 1983). In addition, hepatic storage of thiamin may be reduced due to fatty infiltration of the liver, hepatocellular damage, or cirrhosis. Increased losses of thiamin from the body, as well as increased requirements due to increased metabolic demands, especially following

refeeding with carbohydrates, have been reported. Wernicke-Korsakoff's syndrome caused by thiamin deficiency in the alcoholic population is discussed later in this chapter.

Riboflavin and Niacin. Riboflavin deficiency has been documented in alcoholics, but the mechanism for this deficiency, other than a presumed inadequate intake, has yet to be delineated.

In the 1940's, reductions in mortality rates from 90 to 14 percent following therapy with niacin (nicotinic acid) were reported in a large series of alcoholic patients who presented with impaired consciousness, delirium, a particular type of muscular rigidity called cogwheel rigidity, and uncontrollable grasping and sucking reflexes (Jolliffe 1940, 1941). In some patients, paralysis of the eye muscles and memory defects developed during therapy. Although these symptoms were attributed to niacin deficiency, deficiencies of other nutrients, especially thiamin, were probably present as well.

Vitamin B₆. Evidence suggests that alcohol inhibits the absorption of vitamin B₆ (Baker et al. 1975) and its release from the liver (Sorrell et al. 1974). Alcohol also increases the rate of degradation of pyridoxal-5-phosphate, one of the active forms of vitamin B₆. However, the magnitude of vitamin B₆ deficiency, its contribution to morbidity, and the precise mechanisms involved in its effects on alcoholic persons are unknown.

Folate and Vitamin B₁₂. Folate deficiency is probably the most common vitamin deficiency observed in alcoholics. Conversely, alcoholism is probably the most common cause of folate deficiency in the U.S. adult population. As with other nutrients, many factors contribute to folate deficiency in alcoholics, although poor dietary intake is undoubtedly the major cause. Alcoholics with good diets are less likely to have a folate deficiency than alcoholics with poor diets. The type of alcoholic beverage typically consumed may be significant because beer contains considerably more folate than wine or distilled spirits.

Folate malabsorption has been reported to occur among alcoholics, but this condition may be secondary to the toxic effects of alcohol or to malnutrition, both of which can damage the intestinal mucosa and impair nutrient absorption. Accordingly, correction of folate deficiency has been followed by improved absorption both of folate and other vitamins and minerals.

Urinary excretion of folate is increased by alcohol intake, and its tissue utilization is decreased (Russell et al. 1983). Alcohol may directly inhibit

enzymes involved in folate metabolism. Several investigators (Sullivan and Herbert 1964; Lindenbaum 1977) have shown that alcohol antagonizes the ability of folate to reverse the megaloblastic bone marrow changes seen in deficiency states, but larger doses of folate can overcome this antagonism. The suppressive effects of alcohol have been shown to be present whether folate is given intravenously or orally, suggesting that a metabolic function subsequent to absorption is involved.

Others have observed that macrocytosis (large red blood cells) occurs frequently in alcoholics and that this symptom persists despite folate supplementation unless alcohol intake is curtailed. Because of the high prevalence of folate deficiency and related anemia in derelict alcoholics, some investigators have proposed that low concentrations of folate be added to inexpensive domestic wine and spirits as a way of preventing this deficiency (Kaunitz and Lindenbaum 1977). However, it should be noted that even though folate deficiency can cause macrocytosis, the macrocytosis often seen in heavy drinkers is usually not related to folate deficiency and is not corrected by administration of the vitamin. Instead, this macrocytosis appears to be due to the direct effect of alcohol on the architecture of the red cell and is correctable only with abstinence. Because this kind of macrocytosis is usually unaccompanied by anemia, the presence of macrocytosis without anemia is a useful clue to alcohol abuse.

Symptoms of vitamin B₁₂ deficiency are much less common than those of folate deficiency in alcoholics. Among malnourished alcoholics with liver disease, the prevalence of vitamin B₁₂ deficiency is similar to that seen among randomly selected municipal hospital patients. Indeed, in some studies, the circulating levels of vitamin B₁₂ have been found to be higher in alcoholics than in normal controls (Bonjour 1980).

The major manifestation of either folate or vitamin B₁₂ deficiency is macrocytic megaloblastic anemia. Macrocytic anemia, without megaloblastosis, is also seen in chronic liver disease. Megaloblastic changes in the intestinal mucosal cells of persons with folate or vitamin B₁₂ deficiency may further impair nutrient absorption and bioavailability.

Ascorbic Acid (Vitamin C). Serum ascorbic acid levels have been found to be lower in alcoholics than in nonalcoholics, probably because of inadequate diets. However, some data suggest that even when diets are adequate, increasing levels of alcohol consumption are associated with lower serum levels of ascorbic acid. Whether ascorbic acid affects alcohol metabolism is uncertain, as is knowledge of the extent to which this interaction may have clinical significance (Bonjour 1979).

Fat-Soluble Vitamins

Vitamin A. Vitamin A, as a fat-soluble vitamin, requires for its absorption a certain level of fat in the diet and adequate quantities of pancreatic lipid-digesting enzymes (lipases) and bile salts in the small intestine. Some complications of alcoholism, such as pancreatic insufficiency and biliary insufficiency, can therefore lead to malabsorption of vitamin A. In addition, alcoholics with liver disease may have impaired storage or transport of vitamin A because of an inadequate synthesis of retinol-binding protein, the protein formed in the liver that transports vitamin A in the blood. Even moderate alcoholic liver disease is associated with severely decreased vitamin A concentrations, and these levels are reduced in the liver even when blood levels of vitamin A, retinol-binding protein, and prealbumin are normal (Leo and Lieber 1982).

The storage form of vitamin A, retinol, is oxidized to its active form, retinal, by retinol dehydrogenase, an enzyme similar to alcohol dehydrogenase. Impairments in the metabolism of vitamin A have been reported in alcoholics, and some evidence suggests that retinol and ethanol compete for the alcohol dehydrogenase enzyme in the liver, testes, and retina (Shaw and Lieber 1983). Experimental deficiencies of several nutrients, including vitamin A, have been shown to enhance carcinogen-induced tumors in laboratory animals. Whether vitamin A deficiency plays a role in the reported association between alcohol intake and certain types of tumors remains to be determined (Committee on Diet, Nutrition, and Cancer 1982).

Vitamin D. Vitamin D and its metabolites are involved in regulating calcium and phosphorus metabolism, bone formation and resorption, and various other physiologic functions, including some aspects of immune function. Alcoholics have been reported to have low circulating levels of vitamin D, especially 25-hydroxyvitamin D, the metabolite formed in the liver. Not surprisingly, alcoholic persons with liver disease have lower levels of this metabolite than persons without liver disease. Higher rates of osteomalacia and osteoporosis have been reported in alcoholics (see chapter on skeletal diseases). The specific mechanisms to account for these findings remain unclear. Certainly, conditions associated with fat malabsorption may lead to malabsorption of vitamin D. In addition, some evidence suggests that alcohol induces enzymatic changes in the liver that favor the production of inactive metabolites of vitamin D. As discussed below in the section on minerals, the actions of vitamin D are closely linked with the metabolism of calcium and phosphorus, the levels of which may be altered in response to heavy alcohol ingestion.

Vitamin E. Alcoholic persons may develop severe malabsorption of vitamin E due to pancreatic or biliary insufficiency, but actual reports of vitamin E deficiency in alcoholics have been uncommon (Losowsky and Leonard 1967). Available data suggest that vitamin E malabsorption occurs most commonly in the presence of cirrhosis and steatorrhea, or fat malabsorption (Leevy, Tanribilir, and Smith 1971). Whether antioxidants such as vitamin E protect against alcohol-induced fatty changes in the liver remains uncertain (French 1971).

Vitamin K. Evidence suggests that at least half of the vitamin K required by humans is normally synthesized by bacteria in the intestine. In rare cases, vitamin K deficiency may occur with fat malabsorption, which is common in alcoholics. In addition, persons with alcoholic liver disease demonstrate a variety of impairments in the synthesis of vitamin K-dependent and other blood clotting factors. Bleeding disorders are, therefore, common in alcoholics. Alcohol also has been shown to affect blood platelets, coagulation inhibitors, and fibrinolysis (Larkin and Watson-Williams 1984).

Minerals

Iron. Iron deficiency in alcoholics may occur as a result of repeated gastrointestinal bleeding or clotting disorders. Iron overload, with hemosiderosis, may occur because of increased iron intake, either from alcoholic beverages (especially certain wines) or from iron-containing vitamin-mineral supplements that are often taken because of otherwise unexplained anemia. Iron absorption may be increased due to the increased solubility of ferric iron in the small intestine caused by stimulation of gastric acid secretion by alcohol. Increased iron absorption has also been reported in persons with pancreatic insufficiency, folate deficiency, and cirrhosis. In addition, disorders in heme synthesis can result in iron deposition in bone marrow and other organs. Alcoholics may develop sideroblastic anemias, characterized by the presence of ringed sideroblasts, in which iron is deposited between the membrane folds of the mitochondria (Eichner and Hillman 1971). Such anemias may result from vitamin B₆ deficiency, usually in association with folate deficiency or other impairments in activity of enzymes involved in heme synthesis (Larkin and Watson-Williams 1984). Hepatic hemosiderosis also appears to occur more frequently in alcoholics and may be exacerbated by coexisting hemolysis, blood transfusion, or the prolonged administration of therapeutic iron. In most such cases, it is not possible to separate the causes from the consequences of iron deposition in the development of hepatic failure (Larkin and Watson-Williams 1984).

Magnesium. Low magnesium levels have been demonstrated in the blood, skeletal muscle, and heart muscle of alcoholics (Wheeler et al. 1977). Chronic alcohol use can result in both reduced intake and malabsorption of magnesium. Alcohol ingestion acutely increases the urinary excretion of magnesium. Because magnesium is necessary for certain thiamin-dependent enzymes, there has been speculation that magnesium plays a role in the development of some of the neurologic complications of alcohol abuse, including Wernicke-Korsakoff's syndrome (discussed later in this chapter). However, the data are inconsistent, and the precise role, if any, of magnesium remains to be determined (Thomson 1978). Finally, magnesium insufficiency can be associated with hypocalcemia, perhaps because hypomagnesemia causes resistance to parathyroid hormone and impairment of its secretion.

Calcium and Phosphorus. Alcohol ingestion increases urinary calcium excretion, but the mechanisms for this action have yet to be determined. The possible roles of magnesium, parathyroid hormone, and vitamin D in this effect are unclear. Chronic alcoholics may have low serum calcium levels, usually secondary to reduced serum albumin levels from malnutrition, liver disease, or acute pancreatitis. As noted above, vitamin D intake, absorption, and metabolism may also be impaired in alcoholics, resulting in abnormalities in phosphorus, calcium, and magnesium metabolism. For example, low levels of vitamin D impair calcium absorption. Low levels of dietary calcium result in increased urinary retention of calcium and increased urinary excretion of phosphate. By whatever combination of mechanisms, hypophosphatemia occurs in some alcoholics and is easily detectable by routine laboratory testing at the time of admission to the hospital, although its symptoms are similar to those seen in other syndromes associated with alcohol abuse. In some groups of alcoholics, mild hyperphosphatemia has been observed more frequently than hypophosphatemia (Hurt et al. 1986).

Zinc. Reduced concentrations of zinc have been found in the plasma, red blood cells, and liver of humans and rats following chronic alcohol ingestion. Alcohol appears to increase the urinary excretion of zinc, perhaps because of increased release of zinc from hepatic stores. Protein catabolism is also associated with increased urinary zinc losses. The effect of zinc deficiency on alcohol metabolism has not been established, but preliminary evidence suggests that zinc-dependent enzymes, such as those involved in vitamin A metabolism, may be inhibited by alcohol (Thomson 1978). Some alcoholic men with night blindness have required both zinc and vitamin A treatment to correct visual dysfunction (McClain et al. 1979), but

whether the origin of this problem is liver dysfunction or nutrient malabsorption is as yet uncertain.

Nutritional Status of Alcoholics

Three types of malnutrition may be observed in alcoholics (Lieber 1983): (1) primary malnutrition due to a decreased intake of nutrients, (2) secondary malnutrition caused by an impairment in the digestion and absorption of nutrients due to the effects of alcohol, and (3) tertiary malnutrition due to an alteration in the ability to convert nutrients to their active coenzyme forms, resulting in nutritional complications that potentiate the direct toxic effects of alcohol.

The nutritional status of alcoholics remains ill defined because of variations in the types of studies performed and the groups of alcoholics that have been studied. The derelict alcoholic has a different baseline nutritional status than the middle-class alcoholic, and findings derived from studying one group cannot necessarily be extrapolated to the other.

The preponderance of studies have examined indigent alcoholics who are readily available and willing to cooperate at least temporarily with researchers. These individuals are probably as different in the type and amount of alcohol and food consumed as they are in every other aspect of day-to-day living when compared with nonindigent and middle-class alcoholics. Although indigent alcoholics make up less than 5 percent of the total alcoholic population, there is a persistent misconception that this group is representative of the alcoholic population at large. The almost total exclusion of women in this population is an indication that indigent alcoholics are not representative of all alcoholics.

Because of the association of alcoholism with serious medical illnesses such as liver cirrhosis or pancreatic insufficiency, another tendency is to study the nutritional status of persons with these conditions rather than the much larger—and healthier—portion of the alcoholic population. A clear distinction needs to be made between each of these groups. For example, the dietary intake of a large group of hospitalized alcoholic patients has been reported to be poor when judged by “traditional standards” (Patek et al. 1975). The mean daily energy intake in this group was over 3,000 kcal, but more than 50 percent was derived from alcohol. All of these patients were hospitalized for the treatment of medical complications of alcoholism, and two-thirds had cirrhosis, a much higher prevalence than expected for the general alcoholic population (Lelbach 1975).

Individuals hospitalized in alcoholism treatment centers may also have severe alcohol-associated medical problems, but the primary reason for their hospitalization is alcoholism. Therefore, these patients are probably more representative of the overall alcoholic population. Despite the high proportion of caloric intake from alcohol in this group, only a few studies have reported evidence of malnutrition. Among a small group of male alcoholics in an inpatient alcoholism treatment program, total caloric intake was estimated to be 2,600 to 2,700 kcal, with alcohol providing 22 to 36 percent of calories (Neville et al. 1968). In a similar group of middle-class alcoholics, the mean intake measured by diet history at admission to an alcoholism treatment unit was 3,100 kcal, with alcohol accounting for 35 percent of the daily energy intake (Hurt et al. 1981). No severe nutritional deficiencies were found using anthropometric measurements and blood studies of protein status in a small group of alcoholic patients hospitalized in a veterans hospital alcoholism treatment program (Dickson et al. 1983). Despite adequate protein intake, alcoholic patients, with and without liver disease, had significantly lower body weights, triceps skin folds, and arm muscle circumferences than abstainers (Simko, Connell, and Banks 1982), but no assessment of the socioeconomic status of this group was included. Thirteen percent of nonindigent alcoholics in another study were malnourished, but because of the method used to analyze the anthropometric data, the percent was probably higher (Tomaiolo and Kraus 1980). In yet another study, 13 percent of low-income alcoholics and 10 percent of middle-income alcoholics were judged to be malnourished, but obesity occurred in 2 percent and 4 percent, respectively. This latter research suffered from the vagueness of the nutritional data, which was based on the overall medical status of the subjects. In a group of 179 middle-class males, 47 of whom were alcoholic inpatients, the consumption of alcohol was associated with a decrease in energy intake from other sources and a reduction in the nutrient density of the diet at both moderate and high levels of alcohol consumption (Hillers and Massey 1985).

A comparison of alcoholics of lower socioeconomic status with those of higher socioeconomic status showed small but clear differences in the ratio of height-to-weight, triceps skin fold, mid-arm muscle circumference, and hematocrit (Goldsmith, Iber, and Miller 1983). Only 8 percent of this middle-income group were moderately malnourished, compared with 24 percent of the low-income group. In the low-income group, 8 percent were severely malnourished. Thus, socioeconomic class is an important factor that often is not taken into full account in nutritional studies of alcoholics. Furthermore, very few studies have been conducted on middle-income alcoholics, although these indicate that the nutritional status of this group is