

# Alcoholism

## ALCOHOLISM

### GOALS AND OBJECTIVES

#### Course Description

“Alcoholism” is a home study continuing education course for rehabilitation professionals. This course presents updated information about alcoholism including sections on causative factors, cognitive impairment, screening, comorbidity, alcoholism and aging, drug interactions, treatment and economic impact.

#### Course Rationale

The information presented in this course is applicable for rehabilitation professionals in all settings. A greater understanding of alcoholism will enable therapists and assistants to provide more effective and efficient rehabilitative care to individuals affected by this condition.

#### Course Goals and Objectives

Upon completion of this course, the therapist or assistant will be able to:

1. list the criteria defining alcoholism
2. recognize the process in which alcohol is metabolized by the body
3. identify cognitive impairments associated with alcohol use
4. recognize and apply many of the current tools utilized by health care professionals to screen for alcoholism
5. recognize how alcoholism effects nutrition
6. recognize the role alcoholism has in many comorbid conditions including cancers, hormonal dysfunction, liver disease, stress disorders.
7. list the effects alcohol has on aging and geriatric populations.
8. recognize the factors which contribute to alcohol abuse in the workplace and effective strategies to prevent them.
9. Identify risk factors associated with alcoholism in women.
10. recognize the side effects caused when alcohol is combined with other medications.
11. differentiate between all of the available alcohol treatment options.
12. recognize the economic impact of alcohol abuse.

#### Course Instructor

Michael Niss, DPT

#### Target Audience

Physical therapists, physical therapist assistants, occupational therapists, and occupational therapist assistants

#### Course Educational Level

This course is applicable for introductory learners.

#### Course Prerequisites

None

#### Criteria for issuance of Continuing Education Credits

A documented score of 70% or greater on the written post-test.

#### Continuing Education Credits

Four (4) hours of continuing education credit (4 NBCOT PDUs/4 contact hours)  
AOTA - .4 AOTA CEU, Category 1: Domain of OT – Client Factors, Context

#### Determination of Continuing Education Contact Hours

“Alcoholism” has been established to be a 4 hour continuing education program. This determination is based on an accepted standard for home-based self-study courses of 12 pages of text (12 pt font) per hour. The complete instructional text for this course is 50 pages (excluding References and Post-Test).

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### Introduction

For most people who drink, alcohol is a pleasant accompaniment to social activities. Moderate alcohol use—up to two drinks per day for men and one drink per day for women and older people—is not harmful for most adults. Nonetheless, a large number of people get into serious trouble because of their drinking. Currently, nearly 14 million Americans—1 in every 13 adults—abuse alcohol or are alcoholic. Several million more adults engage in risky drinking that could lead to alcohol problems. These patterns include binge drinking and heavy drinking on a regular basis. In addition, 53 percent of men and women in the United States report that one or more of their close relatives have a drinking problem.

The consequences of alcohol misuse are serious—in many cases, life threatening. Heavy drinking can increase the risk for certain cancers, especially those of the liver, esophagus, throat, and larynx. Heavy drinking can also cause liver cirrhosis, immune system problems, brain damage, and harm to the fetus during pregnancy. In addition, drinking increases the risk of death from automobile crashes as well as recreational and on-the-job injuries. Furthermore, both homicides and suicides are more likely to be committed by persons who have been drinking. In purely economic terms, alcohol-related problems cost society approximately \$185 billion per year. In human terms, the costs cannot be calculated.

### Defining Alcoholism

Alcoholism, also known as “alcohol dependence,” is a disease that includes four symptoms:

- **Craving:** A strong need, or compulsion, to drink.
- **Loss of control:** The inability to limit one’s drinking on any given occasion.
- **Physical dependence:** Withdrawal symptoms, such as nausea, sweating, shakiness, and anxiety, occur when alcohol use is stopped after a period of heavy drinking.
- **Tolerance:** The need to drink greater amounts of alcohol in order to “get high.”

Although some people are able to recover from alcoholism without help, the majority of alcoholics need assistance. With treatment and support, many individuals are able to stop drinking and rebuild their lives.

Alcohol abuse differs from alcoholism in that it does not include an extremely strong craving for alcohol, loss of control over drinking, or physical dependence.

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Alcohol abuse is defined as a pattern of drinking that results in one or more of the following situations within a 12-month period:

- Failure to fulfill major work, school, or home responsibilities;
- Drinking in situations that are physically dangerous, such as while driving a car or operating machinery;
- Having recurring alcohol-related legal problems, such as being arrested for driving under the influence of alcohol or for physically hurting someone while drunk; and
- Continued drinking despite having ongoing relationship problems that are caused or worsened by the drinking.

### The Metabolic Process

When alcohol is consumed, it passes from the stomach and intestines into the blood, a process referred to as absorption. Alcohol is then metabolized by enzymes, which are body chemicals that break down other chemicals. In the liver, an enzyme called alcohol dehydrogenase (ADH) mediates the conversion of alcohol to acetaldehyde. Acetaldehyde is rapidly converted to acetate by other enzymes and is eventually metabolized to carbon dioxide and water. Alcohol also is metabolized in the liver by the enzyme cytochrome P450IIE1 (CYP2E1), which may be increased after chronic drinking. Most of the alcohol consumed is metabolized in the liver, but the small quantity that remains unmetabolized permits alcohol concentration to be measured in breath and urine.

The liver can metabolize only a certain amount of alcohol per hour, regardless of the amount that has been consumed. The rate of alcohol metabolism depends, in part, on the amount of metabolizing enzymes in the liver, which varies among individuals and appears to have genetic determinants. In general, after the consumption of one standard drink (a standard drink contains about 14 grams of pure alcohol), the amount of alcohol in the drinker's blood (blood alcohol concentration, or BAC) peaks within 30 to 45 minutes. Alcohol is metabolized more slowly than it is absorbed. Since the metabolism of alcohol is slow, consumption needs to be controlled to prevent accumulation in the body and intoxication.

### Factors Influencing Alcohol Absorption and Metabolism

**Food.** A number of factors influence the absorption process, including the presence of food and the type of food in the gastrointestinal tract when alcohol is consumed. The rate at which alcohol is absorbed depends on how quickly the stomach empties its contents into the intestine. The higher the dietary fat content, the more time this emptying will require and the longer the process of absorption will take. One study found that subjects who drank alcohol after a meal that

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included fat, protein, and carbohydrates absorbed the alcohol about three times more slowly than when they consumed alcohol on an empty stomach.

**Gender.** Women absorb and metabolize alcohol differently from men. They have higher BAC's after consuming the same amount of alcohol as men and are more susceptible to alcoholic liver disease, heart muscle damage, and brain damage. The difference in BAC's between women and men has been attributed to women's smaller amount of body water, likened to dropping the same amount of alcohol into a smaller pail of water. An additional factor contributing to the difference in BAC's may be that women have lower activity of the alcohol metabolizing enzyme ADH in the stomach, causing a larger proportion of the ingested alcohol to reach the blood. The combination of these factors may render women more vulnerable than men to alcohol-induced liver and heart damage.

### Effects of Alcohol Metabolism

**Body Weight.** Although alcohol has a relatively high caloric value, 7.1 Calories per gram (as a point of reference, 1 gram of carbohydrate contains 4.5 Calories, and 1 gram of fat contains 9 Calories), alcohol consumption does not necessarily result in increased body weight. An analysis of data collected from the first National Health and Nutrition Examination Survey (NHANES I) found that although drinkers had significantly higher intakes of total calories than nondrinkers, drinkers were not more obese than nondrinkers. In fact, women drinkers had significantly lower body weight than nondrinkers. As alcohol intake among men increased, their body weight decreased. An analysis of data from the second National Health and Nutrition Examination Survey (NHANES II) and other large national studies found similar results for women although the relationship between drinking and body weight for men is inconsistent. Although moderate doses of alcohol added to the diets of lean men and women do not seem to lead to weight gain, some studies have reported weight gain when alcohol is added to the diets of overweight persons.

When chronic heavy drinkers substitute alcohol for carbohydrates in their diets, they lose weight and weigh less than their non-drinking counterparts. Furthermore, when chronic heavy drinkers add alcohol to an otherwise normal diet, they do not gain weight.

**Sex Hormones.** Alcohol metabolism alters the balance of reproductive hormones in men and women. In men, alcohol metabolism contributes to testicular injury and impairs testosterone synthesis and sperm production. In a study of normal healthy men who received 220 grams of alcohol daily for 4 weeks, testosterone levels declined after only 5 days and continued to fall throughout the study period. Prolonged testosterone deficiency may contribute to feminization in males, for example, breast enlargement. In addition, alcohol may interfere with normal sperm structure and movement by inhibiting the metabolism of vitamin A, which is essential for sperm development. In women, alcohol

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metabolism may contribute to increased production of a form of estrogen called estradiol (which contributes to increased bone density and reduced risk of coronary artery disease) and to decreased estradiol metabolism, resulting in elevated estradiol levels. One research review indicates that estradiol levels increased in premenopausal women who consumed slightly more than enough alcohol to reach the legal limit of alcohol (BAC of 0.10 percent) acutely. A study of the effect of alcohol on estradiol levels in postmenopausal women found that in women wearing estradiol skin patches, acute alcohol consumption significantly elevated estradiol levels over the short term.

**Medications.** Chronic heavy drinking appears to activate the enzyme CYP2E1, which may be responsible for transforming the over-the-counter pain reliever acetaminophen (Tylenol™) and many others, into chemicals that can cause liver damage, even when acetaminophen is taken in standard therapeutic doses. A review of studies of liver damage resulting from acetaminophen-alcohol interaction reported that in alcoholics, these effects may occur with as little as 2.6 grams of acetaminophen (four to five "extra-strength" pills) taken over the course of the day in persons consuming varying amounts of alcohol. The damage caused by alcohol-acetaminophen interaction is more likely to occur when acetaminophen is taken after, rather than before, the alcohol has been metabolized. Alcohol consumption affects the metabolism of a wide variety of other medications, increasing the activity of some and diminishing the activity, thereby decreasing the effectiveness, of others.

## Genetics and Other Causative Factors

### The Genetics of Alcoholism

Research has shown conclusively that familial transmission of alcoholism risk is at least in part genetic and not just the result of family environment. The task of current science is to identify what a person inherits that increases vulnerability to alcoholism and how inherited factors interact with the environment to cause disease. This information will provide the basis for identifying people at risk and for developing behavioral and pharmacologic approaches to prevent and treat alcohol problems.

### A Complex Genetic Disease

Studies in recent years have confirmed that identical twins, who share the same genes, are about twice as likely as fraternal twins, who share on average 50 percent of their genes, to resemble each other in terms of the presence of alcoholism. Recent research also reports that 50 to 60 percent of the risk for alcoholism is genetically determined, for both men and women. Genes alone do not preordain that someone will be alcoholic; features in the environment along with gene–environment interactions account for the remainder of the risk.

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Each gene directs the synthesis of a different protein. Abnormal gene variants may give rise to defective proteins that can contribute to disease. Genetics studies, such as the Collaborative Study on the Genetics of Alcoholism, have already identified several sites in the brain where the genes for alcoholism may be located.

In search for the genes for alcoholism, alcohol investigators are taking full advantage of the new genetic engineering techniques. For example, researchers can inactivate, or knockout, a gene, creating a line of mice that lack a particular receptor or other protein thought to influence a specific genetic trait. Conversely, scientists can insert an additional gene into an animal's genetic material. Animals with an added gene are called transgenic. The response of the genetically engineered animal to alcohol can be compared with that of a genetically unaltered animal to help determine the role of the gene in mediating a particular alcohol-induced behavior (e.g., incoordination).

Of the many different knockouts, at least three have implicated brain proteins involved in high alcohol preference in mice. Knockout and transgenic experiments also have implicated some of the same receptors and brain proteins in both sensitivity and preference to alcohol. In the animal experiments, mice that initially display high resistance to alcohol-induced sedation usually develop a high preference for alcohol consumption. These laboratory findings are consistent with Schuckit's observation that low sensitivity to alcohol may predict future development of alcoholism. If these results are confirmed in humans, they may provide a means to help identify high-risk youth for targeted intervention programs.

The use of microarrays is another powerful new technology in alcoholism research. This technique permits the simultaneous study of many genes and provides scientists with new power to understand changes in gene expression that relate to the vulnerability to developing alcoholism. The long-term adaptation of the brain's neurons to alcohol may result, in part, from changes in gene function. Genes direct the synthesis of proteins, such as receptors. A gene's level of activity, therefore, can be used to obtain indirect information about its proteins.

Because alcohol is known to affect gene-induced protein production, levels of genetic activity can be tracked to determine how genes associated with an alcohol-induced effect are expressed. Tracking the activity of a single gene takes time; given the large number of genes that may be involved in producing alcohol's effects, the task of linking specific genes to specific effects might be formidable. However, by using microarrays, alcohol scientists can track up to 10,000 selected genes simultaneously. In this approach, the genes of interest are affixed to a glass slide, silicon chip, or similar surface--often as small as a postage stamp--forming a so-called microarray. An automated operating system scans the microarray and can calculate the relative expression levels of up to 10,000 selected genes simultaneously. As more alcoholism researchers begin to

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employ this procedure, it may become possible to identify virtually every gene and its protein that play a significant role in alcohol-related behavior.

### **Detection Using Biological Markers**

Developing a reliable biological marker of recent alcohol consumption has long been on the "wish list" of alcohol researchers and clinicians alike. Such markers could enable researchers to confirm self-reported drinking behavior by study participants and could help clinicians monitor patients undergoing alcoholism treatment. Most currently available markers (e.g., gamma-glutamyl transferase) are alterations in blood chemistry that can be induced directly or indirectly by alcohol. However, many of the markers lack specificity (i.e., altered marker levels are not necessarily the result of alcohol consumption) or sensitivity (i.e., altered marker levels are difficult to detect at low levels of consumption). In addition, some marker levels are not useful until serious alcohol-related organ damage has occurred. Carbohydrate-deficient transferrin (CDT) is a blood protein that increases in concentration after alcohol consumption has exceeded approximately five standard drinks per day for 2 to 3 weeks. CDT concentrations then remain elevated for up to 2 weeks after drinking ceases, potentially enabling relatively early detection of relapse among alcoholics in treatment. Interest in using CDT as a biological marker of alcohol consumption has increased because of its relatively high sensitivity and specificity. Research is underway to improve the precision of CDT measurement. A kit designed to measure CDT levels in patients will soon be commercially available for clinical use.

### **Cognitive Impairment and Recovery**

Brain damage is a common and potentially severe consequence of long-term, heavy alcohol consumption. Even mild-to-moderate drinking can adversely affect cognitive functioning. Persistent cognitive impairment can contribute to poor job performance in adult alcoholics, and can interfere with learning and academic achievement in adolescents with an established pattern of chronic heavy drinking. A small but significant proportion of the heaviest drinkers may develop devastating, irreversible brain-damage syndromes, such as Wernicke-Korsakoff syndrome, a disorder in which the patient is incapable of remembering new information for more than a few seconds.

It stands to reason that cognitive impairment also may impede recovery from alcoholism, although evidence has not conclusively shown this to be the case. For example, Morgenstern and Bates studied whether deficits in a patient's learning and planning abilities affected recovery from alcoholism. They found that impairment was not a significant predictor of poor treatment response. On the other hand, evidence does support the possibility that brain damage, whether

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resulting from or predating alcohol use, may contribute to the development and progression of alcoholism.

Designing practical strategies to cope with the complex combination of alcoholism and cognitive impairment requires an understanding of the nature of cognitive functions and their interactions with structural and functional brain abnormalities.

### **Cognition and Alcohol**

Alcoholics may exhibit mild-to-moderate deficiencies in intellectual function, however, the most prevalent alcohol-associated brain impairments affect visuospatial abilities and higher cognitive functioning. Visuospatial abilities include perceiving and remembering the relative locations of objects in 2- and 3-dimensional space. Examples include driving a car or assembling a piece of furniture based on instructions contained in a line drawing. Higher cognitive functioning includes the abstract-thinking capabilities needed to organize a plan, set it in motion, and change it as needed.

Most alcoholics entering treatment perform as well as non-alcoholics on tests of overall intelligence. However, alcoholics perform poorly on neuropsychological tests that measure specific cognitive abilities. For example, an alcoholic who has remained abstinent after treatment may have no apparent difficulty filing office documents correctly, a task that engages multiple brain regions. However, that same person might be unable to devise a completely different filing system, a task closely associated with higher cognitive functioning.

The link between duration and lifetime quantity of drinking and the development of cognitive problems is unclear. Some investigators have proposed that cognitive performance worsens in direct proportion to the severity and duration of alcoholism. Studies suggest that social drinkers who consume more than 21 drinks per week also fit into this category. Other investigators have suggested that cognitive deficits may be detectable only in those alcoholics who have been drinking regularly for 10 years or more. Long-term, light-to-moderate social drinkers have been found to fall into this category as well, showing cognitive deficits equivalent to those found in detoxified alcoholics. Although further research is needed to determine how a person's pattern of drinking is related to cognitive impairment, some deficits are possible even in people who are not heavy drinkers.

### **Tracking Structural and Functional Brain Abnormalities**

Structural and functional brain abnormalities generally are measured by noninvasive imaging techniques that provide a picture of the living brain with minimal risk to the individual. Structural imaging techniques, such as computed tomography and magnetic resonance imaging, are used to generate computerized pictures of living tissue. Functional imaging techniques, such as

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positron emission tomography and magnetic resonance spectroscopy, permit scientists to study cell activity by tracking blood flow and energy metabolism.

Structural imaging consistently reveals that compared with non-alcoholics, most alcoholics' brains are smaller and less dense. Loss of brain volume is most noticeable in two areas: the outer layer (i.e., the cortex) of the frontal lobe, which is considered a major center of higher mental functions; and the cerebellum, which is responsible largely for gait and balance as well as certain aspects of learning. Support for these results is provided by functional imaging studies, which reveal altered brain activity throughout the cortex and cerebellum of heavy drinkers. In addition, functional imaging often is sufficiently sensitive to detect these irregularities before they can be observed by structural imaging techniques, and even before major cognitive problems themselves become manifest. This suggests that functional imaging may be particularly useful for detecting the early stages of cognitive decline.

### **Understanding the Basis of Cognitive Impairment**

Accurate measurement of cognitive abilities is challenging, and relating those abilities to a specific brain irregularity simply may not be possible with the current technology. Discrepancies among research findings have led scientists to develop improved cognitive-measuring techniques. Using a battery of tests, Beatty and colleagues have suggested that widespread cognitive impairment could arise from damage to multiple brain areas, each of which regulates distinct but related abilities. Likewise, damaging the network of brain cells that synchronizes the overall activity of those multiple areas may produce the same cognitive impairments previously attributed to localized damage.

### **Reversibility of alcohol-related cognitive function**

Certain alcohol-related cognitive impairment is reversible with abstinence. Newly detoxified adult alcoholics often exhibit mild yet significant deficits in some cognitive abilities, especially problem-solving, short-term memory, and visuospatial abilities. By remaining abstinent, however, the recovering alcoholic will continue to recover brain function over a period of several months to 1 year with improvements in working memory, visuospatial functioning, and attention—accompanied by significant increases in brain volume, compared with treated alcoholics who have subsequently relapsed to drinking.

Reversibility of alcohol-related cognitive function also may be the result of a reorganization of key brain-cell networks. Some researchers have proposed that such reorganization may contribute to the success of alcoholism treatment. Using advanced imaging techniques, Pfefferbaum and colleagues examined the brain activity of cognitively impaired alcoholic participants during a series of tests designed to assess cognitive function. They found that although the alcoholic subjects had abnormal patterns of brain activation, compared with control subjects, they were able to complete the tasks equally well, suggesting that the brain systems in alcoholics can be functionally reorganized so that tasks formerly

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performed by alcohol-damaged brain systems are shunted to alternative brain systems. This finding-that cognitively impaired alcoholic patients use different brain pathways than unimpaired patients to achieve equivalent outcome-also was suggested in a study of patients in 12-step treatment programs. Functional brain reorganization may be particularly advantageous for adolescent alcohol abusers in treatment, because their developing brains are still in the process of establishing nerve-cell networks.

### **Cognitive Function and Alcoholism Treatment**

The exact role that cognitive function has in alcoholism treatment success is unclear. Structural and functional imaging, as well as more specific cognitive tests, may provide scientists with the tools needed to reveal subtle relationships between alcohol-related cognitive impairment and recovery. Meanwhile, certain conclusions can be drawn from existing research that help to explain how cognitive function may influence alcoholism treatment:

- Cognitive deficits have been hypothesized to affect the efficacy of alcoholism treatment, although a clear association has not been established. One view finds that cognitively impaired patients may not be able to comprehend the information imparted during therapy and, thus, may not make full use of the strategies presented, thereby hampering recovery. Another view is that cognitive functioning may not directly influence treatment outcome, but may impact other factors that, in turn, contribute to treatment success. Focusing on those factors-such as improved nutrition, opportunities for exercise, careful evaluation of comorbid mental or medical disorders, and/or treatment strategies aimed at enticing the patient out of long-standing social isolation-ultimately may be more beneficial than focusing exclusively on recovery from alcoholism.
- Other types of non-alcohol-related brain damage also can produce symptoms resembling those associated with chronic alcoholism. Clinicians must be aware that no matter the cause of the impairment, it may have an impact on the patient's ability to benefit fully from alcohol-treatment strategies.
- Cognitive impairment is usually most severe during the first weeks of abstinence, perhaps making it difficult for some alcoholics to benefit from educational and skill-development sessions, which are important components of many treatment programs. For example, one study found that alcoholics tested soon after entering treatment were unable to recall treatment-related information presented in a film they had just been shown. As time goes by and cognitive function improves, however, patients may make better use of information presented to them in individual and group therapy, educational programs, and 12-step programs.

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### Screening for Alcohol Problems

The prevalence of alcohol use disorders is significantly higher among patients visiting a primary care practitioner than among the general population. For this reason, clinicians have the opportunity to play a key role in detecting alcohol problems and in initiating prevention or treatment efforts. A variety of relatively brief screening instruments are available for this purpose. These instruments do not provide a diagnosis, but help identify patients who might benefit from a more thorough assessment of their drinking behavior. Following screening, the presence of an alcohol use disorder can be confirmed using standard clinical diagnostic criteria. The success of this approach has been demonstrated. In one study, 80 percent of patients whose screening results were confirmed by a formal diagnosis of alcohol dependence accepted referrals to alcoholism treatment programs.

Patients should be screened not only for alcohol use disorders, but also for drinking patterns or behaviors that may place them at increased risk for developing adverse health effects or alcoholism. Risky drinkers who have not yet become alcohol dependent often can be treated successfully within the primary care setting.

#### Types of Instruments

Two types of alcoholism-screening instruments are available. The first type includes self-report questionnaires and structured interviews; the second type includes clinical laboratory tests that can detect biochemical changes associated with excessive alcohol consumption. The value of a screening instrument for measuring alcohol problems or other conditions is related to its sensitivity and specificity. Sensitivity refers to a test's accuracy in identifying people who have an alcohol problem (i.e., people with the condition test positive). Specificity refers to the test's effectiveness in identifying people who do not have an alcohol problem (i.e., people without the disease test negative).

No screening instrument is perfect. It is not possible to optimize both sensitivity and specificity in the same screening instrument. The likelihood of overidentifying alcohol use disorders occurs with increased sensitivity and the possibility of missing people who have an alcohol problem grows with increased specificity. Despite these limitations, research supports the use of formal screening instruments to increase the recognition of alcohol problems.

#### Questionnaires

Screening instruments vary in their ability to detect different patterns and levels of drinking and in the degree of their applicability to specific subpopulations and settings. This section compares features of some of the most widely used screening questionnaires.

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### CAGE Questionnaire

Have you ever felt that you should **Cut down** on your drinking?

Have people **Annoyed** you by criticizing your drinking?

Have you ever felt bad or **Guilty** about your drinking?

Have you ever had a drink first thing in the morning to steady your nerves or get rid of a hangover? (**Eye-opener**)

The CAGE questionnaire has been evaluated in several studies, showing sensitivities ranging from 43 to 94 percent for detecting alcohol abuse and alcoholism. CAGE is well suited to busy primary care settings because it poses four straightforward yes/no questions that the clinician can easily remember and requires less than a minute to complete. However, the test may fail to detect low but risky levels of drinking. In addition, CAGE often performs less well among women and minority populations. The performance of CAGE can be improved by incorporating questions about the quantity and frequency of drinking. A study found that this screening strategy effectively identified alcohol abuse and dependence in a general population sample. The same approach also demonstrated better sensitivity and similar specificity compared with CAGE alone among African American patients in an urban emergency room.

### TWEAK Questionnaire

Under 3 - Possible alcohol problem, but specificity too low to be helpful  
3-7 - Patient has an alcohol problem (sensitivity ~85%/specificity 86%)

Can you hold six or more drinks (**Tolerance**)

Yes (2 points)

No (0 points)

Are your friends or relatives **Worried** about your drinking?

Yes (2 points)

No (0 points)

Have you ever had an **Eye-opener** (taken a drink early in the morning to "get going")?

Yes (1 point)

No (0 points)

Have you had blackouts (**Amnesia**)?

Yes (1 point)

No (0 points)

Have you ever felt the need to "**Kut**" down on your drinking?

Yes (1 point)

No (0 points)

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### **Audit (Alcohol Use Disorders Identification Test)**

The Alcohol Use Disorders Identification Test was developed by the World Health Organization to identify persons whose alcohol consumption has become hazardous or harmful to their health. AUDIT is a 10-item screening questionnaire with 3 questions on the amount and frequency of drinking, 3 questions on alcohol dependence, and 4 on problems caused by alcohol. The AUDIT also incorporates questions about quantity and frequency of alcohol use. In contrast to CAGE, AUDIT compares favorably with other instruments in detecting risky drinking, but is less effective in identifying alcohol abuse and alcoholism. Originally developed for primary care settings, AUDIT has proven useful among medical and psychiatric inpatients, in emergency rooms, and in the workplace. AUDIT is relatively free of gender and cultural bias. In addition, it shows promise for screening adolescents and older people, populations in which standard screening instruments produce inconsistent results. The major disadvantage of AUDIT is its length and relative complexity; clinicians require training to score and interpret the test results.

### **T-ACE**

Screening pregnant women for alcohol use has become increasingly important in light of new research showing that even low levels of prenatal alcohol exposure can harm the fetus. Unfortunately, although approximately 20 percent of women consume some alcohol during pregnancy, maternal drinking can be difficult to detect. At least two questionnaires are available that are appropriate for pregnant women, both derived in part from CAGE. T-ACE takes approximately 1 minute to complete and is more accurate than AUDIT for detecting current alcohol consumption and risky drinking, as well as a history of past alcoholism; however, it is less specific.

### **Rapid Alcohol Problem Screen (RAPS4)**

Alcohol consumption plays a role in a large percentage of trauma incidents, including motor vehicle crashes. RAPS4 is a four-item questionnaire derived in part from AUDIT. In both primary care and emergency room settings, RAPS4 showed consistently high sensitivity for detecting alcoholism across gender and ethnic subgroups, although its utility for screening for risky drinking or alcohol abuse has yet to be proven.

**Computer-Assisted Screening.** Computers have been widely and successfully used in screening and in assisting alcoholism intervention. Studies have found no significant difference in accuracy between computerized and paper-and-pencil versions of AUDIT among inpatient alcoholics. Similar results have been achieved with CAGE in the primary care setting. In addition, small laptop computers have been used in large-scale alcohol screening surveys. For example, in Audio Computer Assisted Self-Interviewing (ACASI), a recorded voice asks questions that can be answered by pressing a few keys. Advantages include ease of use for respondents with poor literacy or computer skills, as well

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as increased privacy, although the interviewer remains nearby to offer assistance if necessary.

The Internet provides an increasingly accessible, low-cost medium for screening and brief intervention. A pilot web site incorporating AUDIT and other alcohol history questions attracted more than 10,000 people during an initial 172-day trial. Of 2,253 people who took the test, 89 percent had scores suggesting harmful drinking or alcoholism, although 94 percent of participants of the total sample had never been diagnosed. The procedure is completely automated and self-administered.

### **Biological Markers**

In contrast to self-report questionnaires, clinical laboratory procedures provide objective evidence of problem drinking. They are generally less sensitive and specific than questionnaires, but are valuable for corroborating results of interviews and questionnaires. The accuracy of these markers is affected by various factors such as nonalcoholic liver damage, use of medications or drugs, and by metabolic disorders. Three widely used tests and one promising new marker are described here.

Gamma-glutamyl transferase (GGT) is the most commonly used biochemical measure of drinking. Chronic drinking of 4 or more drinks per day for 4 to 8 weeks significantly raises levels of this blood protein, at least in alcoholics. Four to five weeks of abstinence are usually required for GGT levels to return to within normal range. The ability of this test to detect long-term heavy drinking in the recent past makes GGT useful for monitoring abstinence in recovering alcoholics. However, nonalcoholic liver disease also can increase GGT levels, increasing the likelihood of false-positive results.

Carbohydrate-deficient transferrin (CDT) is another blood protein that increases in concentration with heavy alcohol consumption. CDT values become elevated substantially earlier (1 to 2 weeks) in response to prolonged excessive drinking than conventional markers such as GGT. GGT and CDT are approximately equal in their ability to identify alcoholism. However, few conditions other than heavy drinking will elevate CDT levels, decreasing the probability of false positives. Disadvantages include lower sensitivity in women and adolescents, and the high cost of the laboratory analysis.

Mean corpuscular volume (MCV), an index of red blood cell size, increases with excessive alcohol intake after 4 to 8 weeks. The sensitivity of MCV is too low to justify its use as a single indicator. However, it has higher specificity compared with other tests. MCV can detect evidence of earlier drinking after a long period of abstinence. For this reason, it is a poor indicator of recovery among alcoholics who have stopped drinking.

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Fatty acid ethyl esters (FAEEs) show promise as markers of maternal drinking. FAEEs are formed by the interaction of alcohol and natural fatty substances in the body. They have been detected in samples of meconium (the waste product of newborns). Some evidence suggests that analysis of FAEEs in meconium may indicate timing of prenatal alcohol exposure

### Alcohol and Nutrition

Nutrition is a process that serves two purposes: to provide energy and to maintain body structure and function. Food supplies energy and provides the building blocks needed to replace worn or damaged cells and the nutritional components needed for body function. Alcoholics often eat poorly, limiting their supply of essential nutrients and affecting both energy supply and structure maintenance. Furthermore, alcohol interferes with the nutritional process by affecting digestion, storage, utilization, and excretion of nutrients.

#### Impairment of Nutrient Digestion and Utilization

Alcohol inhibits the breakdown of nutrients into usable molecules by decreasing secretion of digestive enzymes from the pancreas. Alcohol impairs nutrient absorption by damaging the cells lining the stomach and intestines and disabling transport of some nutrients into the blood. In addition, nutritional deficiencies themselves may lead to further absorption problems. For example, folate deficiency alters the cells lining the small intestine, which in turn impairs absorption of water and nutrients including glucose, sodium, and additional folate.

Even if nutrients are digested and absorbed, alcohol can prevent them from being fully utilized by altering their transport, storage, and excretion. Decreased liver stores of vitamins such as vitamin A, and increased excretion of nutrients such as fat, indicate impaired utilization of nutrients by alcoholics.

#### Alcohol and Energy Supply

The three basic nutritional components found in food--carbohydrates, proteins, and fats--are used as energy after being converted to simpler products. Some alcoholics ingest as much as 50 percent of their total daily calories from alcohol, often neglecting important foods.

Even when food intake is adequate, alcohol can impair the mechanisms by which the body controls blood glucose levels, resulting in either increased or decreased blood glucose. In nondiabetic alcoholics, increased blood sugar, or hyperglycemia--caused by impaired insulin secretion--is usually temporary and without consequence. Decreased blood sugar, or hypoglycemia, can cause serious injury even if this condition is short lived. Hypoglycemia can occur when a fasting or malnourished person consumes alcohol. When there is no food to

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supply energy, stored sugar is depleted, and the products of alcohol metabolism inhibit the formation of glucose from other compounds such as amino acids. As a result, alcohol causes the brain and other body tissue to be deprived of glucose needed for energy and function.

Although alcohol is an energy source, how the body processes and uses the energy from alcohol is more complex than can be explained by a simple calorie conversion value. For example, alcohol provides an average of 20 percent of the calories in the diet of the upper third of drinking Americans, and we might expect many drinkers who consume such amounts to be obese. Instead, national data indicate that, despite higher caloric intake, drinkers are no more obese than nondrinkers. Also, when alcohol is substituted for carbohydrates, calorie for calorie, subjects tend to lose weight, indicating that they derive less energy from alcohol than from food.

The mechanisms accounting for the apparent inefficiency in converting alcohol to energy are complex and incompletely understood, but several mechanisms have been proposed. For example, chronic drinking triggers an inefficient system of alcohol metabolism, the microsomal ethanol-oxidizing system (MEOS). Much of the energy from MEOS-driven alcohol metabolism is lost as heat rather than used to supply the body with energy.

### **Alcohol and the Maintenance of Cell Structure and Function**

#### ***Structure***

Because cells are made mostly of protein, an adequate protein diet is important for maintaining cell structure, especially if cells are being damaged. Research indicates that alcohol affects protein nutrition by causing impaired digestion of proteins to amino acids, impaired processing of amino acids by the small intestine and liver, impaired synthesis of proteins from amino acids, and impaired protein secretion by the liver.

#### ***Function***

Nutrients are essential for proper body function; proteins, vitamins, and minerals provide the tools that the body needs to perform properly. Alcohol can disrupt body function by causing nutrient deficiencies and by usurping the machinery needed to metabolize nutrients.

***Vitamins.*** Vitamins are essential to maintaining growth and normal metabolism because they regulate many physiological processes. Chronic heavy drinking is associated with deficiencies in many vitamins because of decreased food ingestion and, in some cases, impaired absorption, metabolism, and utilization. For example, alcohol inhibits fat absorption and thereby impairs absorption of the vitamins A, E, and D that are normally absorbed along with dietary fats. Vitamin A deficiency can be associated with night blindness, and vitamin D deficiency is associated with softening of the bones .

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Vitamins A, C, D, E, K, and the B vitamins, also deficient in some alcoholics, are all involved in wound healing and cell maintenance. In particular, because vitamin K is necessary for blood clotting, deficiencies of that vitamin can cause delayed clotting and result in excess bleeding. Deficiencies of other vitamins involved in brain function can cause severe neurological damage.

**Minerals.** Deficiencies of minerals such as calcium, magnesium, iron, and zinc are common in alcoholics, although alcohol itself does not seem to affect the absorption of these minerals. Rather, deficiencies seem to occur secondary to other alcohol-related problems: decreased calcium absorption due to fat malabsorption; magnesium deficiency due to decreased intake, increased urinary excretion, vomiting, and diarrhea; iron deficiency related to gastrointestinal bleeding; and zinc malabsorption or losses related to other nutrient deficiencies. Mineral deficiencies can cause a variety of medical consequences from calcium-related bone disease to zinc-related night blindness and skin lesions.

### **Alcohol, Malnutrition, and Medical Complications**

#### ***Liver Disease***

Although alcoholic liver damage is caused primarily by alcohol itself, poor nutrition may increase the risk of alcohol-related liver damage. For example, nutrients normally found in the liver, such as carotenoids, which are the major sources of vitamin A, and vitamin E compounds, are known to be affected by alcohol consumption. Decreases in such nutrients may play some role in alcohol-related liver damage.

#### ***Pancreatitis***

Research suggests that malnutrition may increase the risk of developing alcoholic pancreatitis. Preliminary research suggests that alcohol's damaging effect on the pancreas may be exacerbated by a protein-deficient diet.

#### ***Brain***

Nutritional deficiencies can have severe and permanent effects on brain function. Specifically, thiamine deficiencies, often seen in alcoholics, can cause severe neurological problems such as impaired movement and memory loss seen in Wernicke/Korsakoff syndrome.

#### ***Pregnancy***

Alcohol has direct toxic effects on fetal development, causing alcohol-related birth defects, including fetal alcohol syndrome. Alcohol itself is toxic to the fetus, but accompanying nutritional deficiency can affect fetal development, perhaps compounding the risk of developmental damage.

The nutritional needs during pregnancy are 10 to 30 percent greater than normal; food intake can increase by as much as 140 percent to cover the needs of both mother and fetus. Not only can nutritional deficiencies of an alcoholic mother

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adversely affect the nutrition of the fetus, but alcohol itself can also restrict nutrition flow to the fetus.

### Alcoholism and Comorbidity

An understanding of comorbidity is essential in developing effective treatment and prevention efforts. For example, since alcoholism causes liver disease, measures to decrease alcohol consumption will help reduce the incidence of liver disease. With respect to treatment, persons exhibiting comorbid alcohol-related and medical or psychiatric disorders often fall through the cracks of the health care system because of administrative distinctions among addiction, medical, and mental health-related services. Patients are often forced to choose between clinical settings, often resulting in neglect of one condition.

Research on the nature of the relationship between comorbid disorders generally relies on surveys of either the clinical population (persons in treatment) or the general population. Most studies of comorbidity are based on clinical samples. This may result in inflated estimates of comorbidity, since persons with multiple ailments may be more likely to seek treatment (Berkson's fallacy). This trend may be countered to some extent by the reluctance of some alcoholism treatment centers to admit persons exhibiting serious psychiatric problems. Thus, the prevalence of comorbid psychiatric disorders among alcoholics in treatment does not reflect the actual prevalence of such comorbidity in the community.

Because the term comorbidity is often not applied to medical conditions, a number of medical conditions that are often comorbid with alcoholism are mentioned below.

#### Medical conditions

Alcohol has been shown to be directly toxic to the liver. Approximately 90 to 100 percent of heavy drinkers show evidence of fatty liver, an estimated 10 to 35 percent develop alcoholic hepatitis, and 10 to 20 percent develop cirrhosis. Fatty liver is reversible with abstinence, alcoholic hepatitis is usually reversible upon abstinence, and while alcoholic cirrhosis is often progressive and fatal, it can stabilize with abstinence. In addition to liver disease, heavy alcohol consumption causes chronic pancreatitis and malabsorption of nutrients.

The prevalence of alcoholic cardiomyopathy (heart muscle disease) is unknown. Alcohol-induced heart damage appears to increase with lifetime dose of alcohol.

Alcohol can damage the brain in many ways. The most serious effect is Korsakoff's syndrome, characterized in part by an inability to remember recent events or to learn new information. The incidence of alcohol-related brain

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damage is approximately 10 percent of adult dementias in the United States. Milder attention and memory deficits may improve gradually with abstinence.

Additional diseases strongly linked to alcohol consumption include failure of reproductive function and cancers of the mouth, larynx, and esophagus. Hospitalized alcoholics have also been found to have an increased prevalence of dental problems, compared with nonalcoholic psychiatric patients, including missing teeth and nonrestorable teeth.

### **Psychiatric disorders**

Based on ECA data, alcoholics are 21.0 times more likely to also have a diagnosis of antisocial personality disorder compared with non-alcoholics. Similar "odds ratios" for some other psychiatric comorbidities are as follows: drug abuse, 3.9 times; mania, 6.2 times; and schizophrenia, 4.0 times. There is only a mild increase in major depressive disorder among alcoholics (odds ratio 1.7), and essentially no increase in anxiety disorders.

***Antisocial personality disorder.*** The strongest correlate of alcoholism documented in the ECA is antisocial personality disorder (ASPD). Comorbid ASPD has prognostic and treatment implications for alcoholics. Patients with ASPD have an earlier age of onset of alcohol and other drug abuse and a more rapid and serious course.

***Bulimia.*** Bulimia is an eating disorder in which patients, usually female, binge on sugar- and fat-rich meals, and purge regularly, as by self-induced vomiting. This disorder is characterized by craving, preoccupation with binge eating, loss of control during binges, an emphasis on short-term gratification, and ambivalence about treatment--symptoms that resemble those of addictive disorders. Bulimics commonly exhibit multiple drug use disorders and have high rates of alcoholism. Between 33 and 83 percent of bulimics may have a first-degree relative suffering from alcohol abuse or alcoholism.

***Depression.*** Although it has been suggested that alcoholism and depression are manifestations of the same underlying illness, the results of family, twin, and adoption studies suggest that alcoholism and mood disorder are probably distinct illnesses with different prognoses and treatments. However, symptoms of depression are likely to develop during the course of alcoholism, and some patients with mood disorders may increase their drinking when undergoing a mood change, fulfilling criteria for secondary alcoholism. When depressive symptoms are secondary to alcoholism, they are likely to disappear within a few days or weeks of abstinence, as withdrawal symptoms subside.

***Anxiety.*** Studies indicate that approximately 10 to 30 percent of alcoholics have panic disorder, and about 20 percent of persons with anxiety disorders abuse alcohol. Among alcoholics entering treatment, about two-thirds have symptoms that resemble anxiety disorders. The relation between major anxiety disorders

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and alcoholism is unclear. Several studies indicate that anxious patients may use alcohol or other drugs to self-medicate, despite the fact that such use may ultimately exacerbate their clinical condition.

The strongest correlation between alcoholism and severe anxiety symptoms occurs in the context of alcohol withdrawal. The severe tremors, feelings of tension, restlessness, and insomnia associated with withdrawal begin to subside after 4 or 5 days, although a vulnerability to panic attacks and to generalized anxiety may continue for months. Because these symptoms decrease with abstinence, they are unlikely to represent an independent anxiety disorder. Interestingly, subjects suffering from both alcoholism and panic disorder are unable to distinguish between a number of symptoms common to both disorders.

**Other drug abuse.** Based on ECA data, alcoholics are 35 times more likely than non-alcoholics to also use cocaine. Similar odds ratios for other types of drugs are: sedatives, 17.0 times; opioids, 13.0 times; hallucinogens, 12.0; stimulants, 11.0; and marijuana and related drugs, 6.0. Surveys of both clinical and non-clinical populations indicate that at least 90 percent of alcoholics are nicotine dependent .

## Alcohol and Hormones

Alcohol can impair the functions of the hormone-releasing glands and of the target tissues, thereby causing serious medical consequences.

Hormones control four major areas of body function: production, utilization, and storage of energy; reproduction; maintenance of the internal environment (e.g., blood pressure and bone mass); and growth and development.

### Regulation of Blood Sugar Levels

Glucose is derived from three sources: from food; from synthesis (manufacture) in the body; and from the breakdown of glycogen, a form of glucose that the body stores in the liver. Hormones help to maintain a constant concentration of glucose in the blood. Two hormones that are secreted by the pancreas and that regulate blood glucose levels are insulin and glucagon. Insulin lowers the glucose concentration in the blood; glucagon raises it.

Alcohol consumption interferes with all three glucose sources and with the actions of the regulatory hormones. Chronic heavy drinkers often have insufficient dietary intake of glucose. Without eating, glycogen stores are exhausted in a few hours. In addition, the body's glucose production is inhibited while alcohol is being metabolized. The combination of these effects can cause severe hypoglycemia 6 to 36 hours after a binge- drinking episode.

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Even in well-nourished people, alcohol can disturb blood sugar levels. Acute alcohol consumption, especially in combination with sugar, augments insulin secretion and causes temporary hypoglycemia. In addition, studies in healthy subjects and insulin-dependent diabetics have shown that acute alcohol consumption can impair the hormonal response to hypoglycemia.

Chronic heavy drinking, in contrast, has been associated with excessive blood glucose levels (hyperglycemia). Chronic alcohol abuse can reduce the body's responsiveness to insulin and cause glucose intolerance in both healthy individuals and alcoholics with liver cirrhosis. In fact, 45 to 70 percent of patients with alcoholic liver disease are glucose intolerant or are diabetic. In animals, chronic alcohol administration also increases secretion of glucagon and other hormones that raise blood glucose levels.

Alcohol consumption can be especially harmful in people with a predisposition to hypoglycemia, such as patients who are being treated for diabetes. Alcohol can interfere with the management of diabetes in different ways. Acute as well as chronic alcohol consumption can alter the effectiveness of hypoglycemic medications. Treatment of diabetes by tight control of blood glucose levels is difficult in alcoholics, and both hypoglycemic and hyperglycemic episodes are common. In a Japanese study, alcoholics with diabetes had a significantly lower survival rate than other alcoholics.

### **Reproductive Functions**

The human reproductive system is regulated by many hormones. The most important are androgens (e.g., testosterone) and estrogens (e.g., estradiol). They are synthesized mainly by the testes and the ovaries and affect reproductive functions in various target tissues. Other reproductive hormones are synthesized in the hypothalamus and pituitary. Although men and women produce many of the same hormones, their relative concentrations and their functions vary.

In men, reproductive hormones are responsible for sexual maturation, sperm development and thus fertility, and various aspects of male sexual behavior. In women, hormones promote the development of secondary sexual characteristics, such as breast development and distribution of body hair; regulate the menstrual cycle; and are necessary to maintain pregnancy. Chronic heavy drinking can interfere with all these functions. Its most severe consequences in both men and women include inadequate functioning of the testes and ovaries, resulting in hormonal deficiencies, sexual dysfunction, and infertility.

Alcohol is directly toxic to the testes, causing reduced testosterone levels in men. In a study of normal healthy men who received alcohol for 4 weeks, testosterone levels declined after only 5 days and continued to fall throughout the study period. Prolonged testosterone deficiency may contribute to a "feminization" of male sexual characteristics, for example breast enlargement.

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In addition, animal studies have shown that acute alcohol administration affects the release of hormones from the hypothalamus and pituitary. Even without a detectable reduction of testosterone levels, changes in these hormones can contribute to the impairment of male sexual and reproductive functions. Alcohol also may interfere with normal sperm structure and movement by inhibiting the metabolism of vitamin A, which is essential for sperm development.

In premenopausal women, chronic heavy drinking can contribute to a multitude of reproductive disorders. These include cessation of menstruation, irregular menstrual cycles, menstrual cycles without ovulation, early menopause, and increased risk of spontaneous abortions. These dysfunctions can be caused by alcohol's interfering directly with the hormonal regulation of the reproductive system or indirectly through other disorders associated with alcohol abuse, such as liver disease, pancreatic disease, malnutrition, or fetal abnormalities.

Although most of these reproductive problems were found in alcoholic women, some also were observed in women classified as social drinkers, who drank about three drinks per day during a 3-week study. A significant number of these women had abnormal menstrual cycles and a delay or lack of ovulation.

Alcohol also affects reproductive hormones in postmenopausal women. After menopause, estradiol levels decline drastically because the hormone is no longer synthesized in the ovaries, and only small amounts are derived from the conversion of testosterone in other tissues. This estradiol deficiency has been associated with an increased risk for cardiovascular disease and osteoporosis in postmenopausal women. Alcohol can increase the conversion of testosterone into estradiol. Accordingly, postmenopausal women who drank were found to have higher estradiol levels than abstaining women.

### **Calcium Metabolism and Bone Structure**

Calcium absorption, excretion, and distribution between bones and body fluids are regulated by several hormones, namely parathyroid hormone (PTH); vitamin D-derived hormones; and calcitonin, which is made by specific cells in the thyroid.

Alcohol can interfere with calcium and bone metabolism in several ways. Acute alcohol consumption can lead to a transient PTH deficiency and increased urinary calcium excretion, resulting in loss of calcium from the body. Chronic heavy drinking can disturb vitamin D metabolism, resulting in inadequate absorption of dietary calcium.

Studies in alcoholics also have shown that alcohol is directly toxic to bone-forming cells and inhibits their activity. In addition, chronic heavy drinking can adversely affect bone metabolism indirectly, for example by contributing to nutritional deficiencies of calcium or vitamin D. Liver disease and altered levels of

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reproductive hormones, both of which can be caused by alcohol, also affect bone metabolism.

Calcium deficiency can lead to bone diseases, such as osteoporosis. In alcoholics, the risk of osteoporosis is increased. Because many falls are related to alcohol use, adverse alcohol effects on bone metabolism pose a serious health problem.

Studies with abstinent alcoholics have found that alcohol-induced changes in bone metabolism, including toxic effects on bone-forming cells, are at least partially reversible after cessation of drinking.

### **Hormones and Alcohol-Seeking Behavior**

The effects of alcohol on different hormonal pathways may in turn influence alcohol-seeking behavior. For example, in animals, alcohol-seeking behavior appears to be regulated in part through a system called the renin-angiotensin system, which controls blood pressure and salt concentrations in the blood. In rats, activation of this system through alcohol consumption caused the animals to reduce their alcohol intake. The mechanism and relevance of this effect are currently under investigation.

## Alcohol and Cancer

Cancer kills an estimated 526,000 Americans yearly, second only to heart disease. Cancers of the lung, large bowel, and breast are the most common in the United States. Considerable evidence suggests a connection between heavy alcohol consumption and increased risk for cancer, with an estimated 2 to 4 percent of all cancer cases thought to be caused either directly or indirectly by alcohol.

A strong association exists between alcohol use and cancers of the esophagus, pharynx, and mouth, whereas a more controversial association links alcohol with liver, breast, and colorectal cancers. Together, these cancers kill more than 125,000 people annually in the United States.

### **Alcohol's Link to Cancer**

Two types of research link alcohol and cancer. Epidemiologic research has shown a dose-dependent association between alcohol consumption and certain types of cancer; as alcohol consumption increases, so does the risk of developing certain cancers. More tenuous results have come from research into the mechanism by which alcohol could contribute to cancer development.

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### **Epidemiologic Research**

The strongest link between alcohol and cancer involves cancers of the upper digestive tract, including the esophagus, the mouth, the pharynx, and the larynx. Less consistent data link alcohol consumption and cancers of the liver, breast, and colon.

**Upper digestive tract.** Chronic heavy drinkers have a higher incidence of esophageal cancer than does the general population. The risk appears to increase as alcohol consumption increases. An estimated 75 percent of esophageal cancers in the United States are attributable to chronic, excessive alcohol consumption.

Nearly 50 percent of cancers of the mouth, pharynx, and larynx are associated with heavy drinking. People who drink large quantities of alcohol over time have an increased risk of these cancers as compared with abstainers. If they drink and smoke, the increase in risk is even more dramatic.

**Liver.** Prolonged, heavy drinking has been associated in many cases with primary liver cancer. However, it is liver cirrhosis, whether caused by alcohol or another factor, which is thought to induce the cancer. In areas of Africa and Asia, liver cancer afflicts 50 or more people per 100,000 per year, usually associated with cirrhosis caused by hepatitis viruses. In the United States, liver cancer is relatively uncommon, afflicting approximately 2 people per 100,000, but excessive alcohol consumption is linked to as many as 36 percent of these cases by some investigators.

**Breast.** Chronic alcohol consumption has been associated with a small (averaging 10 percent) increase in a woman's risk of breast cancer. Recent studies suggest that alcohol may play an indirect role in the development of breast cancer. These studies indicate that alcohol increases estrogen levels in premenopausal women, which, in turn, may promote breast cancer.

**Colon.** Epidemiologic studies have found a small but consistent dose-dependent association between alcohol consumption and colorectal cancer, even when controlling for fiber and other dietary factors. Despite the large number of studies, however, causality cannot be determined from the available data.

### **Mechanisms of Alcohol-Related Cancers**

The epidemiologic data provide little insight into whether or how alcohol increases the risk for various cancers. For some cancers, such as mouth and esophageal, alcohol is thought to play a direct causal role. For others, such as liver and breast cancers, alcohol is thought to play an indirect role by enhancing mechanisms that may cause cancer.

**Oncogenes.** Preliminary studies show that alcohol may affect cancer development at the genetic level by affecting oncogenes at the initiation and

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promotion stages of cancer. It has been suggested that acetaldehyde, a product of alcohol metabolism, impairs a cell's natural ability to repair its DNA, resulting in a greater likelihood that mutations causing cancer initiation will occur. It has recently been suggested that alcohol exposure may result in overexpression of certain oncogenes in human cells and, thereby, trigger cancer promotion.

**Alcohol as a cocarcinogen.** Although there is no evidence that alcohol itself is a carcinogen, alcohol may act as a cocarcinogen by enhancing the carcinogenic effects of other chemicals. For example, studies indicate that alcohol enhances tobacco's ability to stimulate tumor formation in rats. In humans, the risk for mouth, tracheal, and esophageal cancer is 35 times greater for people who both smoke and drink than for people who neither smoke nor drink, implying a cocarcinogenic interaction between alcohol and tobacco-related carcinogens.

Alcohol's cocarcinogenic effect may be explained by its interaction with certain enzymes. Some enzymes that normally help to detoxify substances that enter the body can also increase the toxicity of some carcinogens. One of these enzymes is called cytochrome P-450. Dietary alcohol is able to induce cytochrome P-450 in the liver, lungs, esophagus, and intestines, where alcohol-associated cancers occur. Subsequently, carcinogens such as those from tobacco and diet can become more potent as they, too, pass through the esophagus, lungs, intestines, and liver and encounter the activated enzyme.

**Nutrition.** Chronic alcohol abuse may result in abnormalities in the way the body processes nutrients and may subsequently promote certain types of cancer. Reduced levels of iron, zinc, vitamin E, and some of the B vitamins, common in heavy drinkers, have been experimentally associated with some cancers. Also, levels of vitamin A, hypothesized to have anticancer properties, are severely depressed in the liver and esophagus of rats during chronic alcohol consumption.

A recent study indicates that as few as two drinks per day negate any beneficial effects of a "correct" diet on decreasing risk of colon cancer. Although the study suggests that a diet high in folic acid, a B vitamin found in fresh fruits and vegetables, decreases the risk for colon cancer, it also warns that alcohol consumption may counter this protective action and increase the risk for colon cancer by reducing folic acid levels.

**Suppression of immune response.** Alcoholism has been associated with suppression of the human immune system. Immune suppression makes chronic alcohol abusers more susceptible to various infectious diseases and, theoretically, to cancer.

### Alcohol and Liver Disease

Alcohol-induced liver disease (ALD) is a major cause of illness and death in the United States. Fatty liver, the most common form of ALD, is reversible with abstinence. More serious ALD includes alcoholic hepatitis, characterized by persistent inflammation of the liver, and cirrhosis, characterized by progressive scarring of liver tissue. Either condition can be fatal, and treatment options are limited. During the past 5 years, research has significantly increased our understanding of the mechanisms by which alcohol consumption damages the liver.

#### **The Prevalence of ALD**

Approximately 10 to 35 percent of heavy drinkers develop alcoholic hepatitis, and 10 to 20 percent develop cirrhosis. In the United States, cirrhosis is the seventh leading cause of death among young and middle-age adults. Approximately 10,000 to 24,000 deaths from cirrhosis may be attributable to alcohol consumption each year.

#### **How Alcohol Damages the Liver**

Normal liver function is essential to life. Alcohol-induced liver damage disrupts the body's metabolism, eventually impairing the function of other organs. Multiple physiological mechanisms, discussed in the following sections, interact to influence the progression of ALD. Medications that affect these mechanisms may help prevent some of the medical complications of ALD or reduce the severity of the illness.

***Alcohol Metabolism.*** Most of the alcohol a person drinks is eventually broken down by the liver. However, some products generated during alcohol metabolism (e.g., acetaldehyde) are more toxic than alcohol itself. In addition, a group of metabolic products called free radicals can damage liver cells and promote inflammation, impairing vital functions such as energy production. The body's natural defenses against free radicals (e.g., antioxidants) can be inhibited by alcohol consumption, leading to increased liver damage.

***The Inflammatory Response.*** Inflammation is the body's response to local tissue damage or infection. Inflammation prevents the spread of injury and mobilizes the defense mechanisms of the immune system. One such defense mechanism is the generation of free radicals that can destroy disease-causing microorganisms. Long-term alcohol consumption prolongs the inflammatory process, leading to excessive production of free radicals, which can destroy healthy liver tissue.

Bacteria that live in the human intestine play a key role in the initiation of ALD. Alcohol consumption increases the passage of a noxious bacterial product called endotoxin through the intestinal wall into the bloodstream. Upon reaching the liver, endotoxin activates specialized cells (i.e., Kupffer cells) that monitor the

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blood for signs of infection. These cells respond to the presence of endotoxin by releasing substances called cytokines that regulate the inflammatory process.

**Cytokines.** Cytokines are produced by cells of the liver and immune system in response to infection or cell damage. Alcohol consumption increases cytokine levels, and cytokines in humans produce symptoms similar to those of alcoholic hepatitis. Recent studies implicate cytokines in scar formation and in the depletion of oxygen within liver cells, processes that are associated with cirrhosis. Each of the disease mechanisms described above contributes to the death of liver cells. The presence of damaged cells triggers the body's defensive responses, including the release of additional cytokines, resulting in a vicious cycle of inflammation, cell death, and scarring.

**Scar Formation.** Normal scar formation is part of the wound-healing process. Alcohol-induced cell death and inflammation can result in scarring that distorts the liver's internal structure and impairs its function. This scarring is the hallmark of cirrhosis. The process by which cirrhosis develops involves the interaction of certain cytokines and specialized liver cells (i.e., stellate cells). In the normal liver, stellate cells function as storage depots for vitamin A. Upon activation by cytokines, stellate cells proliferate, lose their vitamin A stores, and begin to produce scar tissue. In addition, activated stellate cells constrict blood vessels, impeding the delivery of oxygen to liver cells.

Acetaldehyde activates stellate cells directly, promoting liver scarring in the absence of inflammation. This finding is consistent with the observation that heavy drinkers can develop cirrhosis insidiously, without preexisting hepatitis.

### **Factors That Influence Vulnerability to ALD**

Susceptibility to ALD differs considerably among individuals, so that even among people drinking similar amounts of alcohol, only some develop cirrhosis. Understanding the mechanisms of these differences may help clinicians identify and treat patients at increased risk for advanced liver damage.

**Genetic Factors.** Structural or functional variability in any of the cell types and biochemical substances discussed above could influence a person's susceptibility to ALD. Researchers are seeking genetic factors that may underlie this variability. Results of this research may provide the basis for future gene-based therapies.

**Dietary Factors.** Nutritional factors influence the progression of ALD. For example, a high-fat, low-carbohydrate diet promotes liver damage in alcohol-fed rats, and high amounts of polyunsaturated fats may promote the development of cirrhosis in animals.

**Gender.** Women develop ALD after consuming lower levels of alcohol over a shorter period of time compared with men. In addition, women have a higher

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incidence of alcoholic hepatitis and a higher mortality rate from cirrhosis than men. The mechanisms that underlie gender-related differences are unknown.

**Hepatitis C.** Many patients with ALD are infected with hepatitis C virus (HCV), which causes a chronic, potentially fatal liver disease. The presence of HCV may increase a person's susceptibility to ALD and influence the severity of alcoholic cirrhosis. For example, alcohol-dependent patients infected with HCV develop liver injury at a younger age and after consuming a lower cumulative dose of alcohol than do those without HCV (20). Patients with HCV are often treated with an antiviral substance called interferon. However, interferon is less effective in patients with chronic HCV who are heavy drinkers, compared with those who are not.

### Treatment Effectiveness

Abstinence is the cornerstone of ALD therapy. With abstinence, fatty liver and alcoholic hepatitis are frequently reversible, and survival is improved among patients with ALD, including those with cirrhosis. For terminally ill patients, liver transplantation remains the only effective treatment. Research has established the effectiveness of liver transplantation in patients with alcoholic cirrhosis. More recently, Belle and colleagues summarized followup medical data on all persons who received liver transplants in the United States between 1988 and 1995. Deaths among these subjects were not alcohol related. That is, alcohol-dependent patients died from the same conditions that caused deaths among patients without alcoholism (e.g., infection, cancer, or heart disease). Recurrences of liver disease among alcohol-dependent patients are rare.

Hepatitis C infection in patients with ALD does not appear to affect survival after liver transplantation, despite the continued presence of the virus in the bloodstream.

### Prospects for Future Treatment

The multiple mechanisms of ALD development provide several potential targets for medical intervention. Some promising lines of inquiry are summarized below.

The role of endotoxin in the inflammatory response suggests the possibility of inhibiting ALD development at its earliest stages. For example, suppression of endotoxin-producing intestinal bacteria reduced signs of liver damage in alcohol-fed rats.

An adequate daily supply of total carbohydrates is important in treating ALD. In addition, researchers are investigating certain nutritional supplements for patients with ALD. One such supplement is polyunsaturated lecithin (PUL), a mixture of fatty substances extracted from soybeans. PUL protected against liver scarring in alcohol-fed baboons. Another dietary factor, S-adenosyl-L-methionine (SAM), can reduce liver cell damage in animals that is induced by alcohol or other toxic

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substances. The safety and effectiveness of these supplements for treating human ALD are under investigation.

Finally, an important goal of ALD research is to develop medications that can moderate the toxic effects of inflammatory cytokines while sparing their essential defensive functions. In one study, administration of antibodies designed to recognize and inactivate key inflammatory cytokines markedly decreased liver injury in rats.

## Alcohol and Stress

The stress response is a complex process; the association between drinking and stress is more complicated still. Because both drinking behavior and an individual's response to stress are determined by multiple genetic and environmental factors, studying the link between alcohol consumption and stress may further our understanding of drinking behavior.

### The Stress Response

The stress response is a highly complex, integrated network involving the central nervous system, the adrenal system, and the cardiovascular system. When homeostasis is threatened, the hypothalamus gland, at the base of the brain, initiates the stress response by secreting corticotropin releasing factor (CRF). CRF coordinates the stress response by triggering an integrated series of physiological and behavioral reactions. CRF is transported in blood within the brain and in seconds triggers the pituitary gland to release adrenocorticotropin hormone (ACTH), also referred to as corticotropin. ACTH then triggers secretion of glucocorticoid hormones by the adrenal glands. Glucocorticoid hormones play a key role in the stress response and its termination.

### Stress Influences on Drinking

Human research to clarify the connection between alcohol and stress usually has been conducted using either population surveys based on subject self-reports or experimental studies. In many but not all of these studies, individuals report that they drink in response to stress and do so for a variety of reasons. Studies indicate that people drink as a means of coping with economic stress, job stress, and marital problems, often in the absence of social support, and that the more severe and chronic the stressor, the greater the alcohol consumption. However, whether an individual will drink in response to stress appears to depend on many factors, including possible genetic determinants of drinking in response to stress, an individual's usual drinking behavior, one's expectations regarding the effect of alcohol on stress, the intensity and type of stressor, the individual's sense of control over the stressor, the range of one's responses to cope with the perceived stress, and the availability of social support to buffer the effects of stress. Some researchers have found that high levels of stress may influence

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drinking when alternative resources are lacking, when alcohol is accessible, and when the individual believes that alcohol will help to reduce the stress.

Numerous studies have found that stress increases alcohol consumption in animals, and that individual animals may differ in the amount of alcohol they consume in response to stress. Such differences may be related in part to an animal's experiencing chronic stress early in life: Prolonged stress in infancy may permanently alter the hormonal stress response and subsequent reactions to new stressors, including alcohol consumption. For example, monkeys who were reared by peers, a circumstance regarded as a stressor compared to mother-rearing, consumed twice as much alcohol as monkeys who were mother-reared. According to Viau and colleagues, adult rats handled for the first 3 weeks of life demonstrate markedly reduced hormonal responses to a variety of stressors compared with rats not handled during this time. In humans, Cloninger reported an association between certain types of alcoholism and adverse early childhood experiences.

Animal studies reporting a positive correlation between stress and alcohol consumption suggest that drinking may take place in response to chronic stress perceived as unavoidable. For instance, rats chronically exposed to unavoidable shock learn to be helpless or passive when faced with any new stressor--including shock that is avoidable--and to demonstrate increased alcohol preference compared with rats that received only avoidable shock. The rats exposed to unavoidable shock exhibit the hormonal changes indicative of the stress response, including increased levels of corticosteroid hormones.

Whether humans drink in response to uncontrollable stress is less clear, according to Pohorecky. In a review investigating the connection between alcohol consumption and stress, Pohorecky notes several studies in which researchers sampled individuals from areas affected by natural disaster. One study found that alcohol consumption increased by 30 percent in the 2 years following a flood at Buffalo Creek, West Virginia. Similarly, there was evidence of increased drinking in the towns surrounding Mount St. Helens following eruption of the volcano.

In both humans and animals, drinking appears to follow stress. Some human research, however, shows that drinking may take place in anticipation of or during times of stress.

### **Alcohol's Effects on Stress**

Some studies have reported that acute exposure to low doses of alcohol may reduce the response to a stressor in animals and humans. For example, low doses of alcohol reduced the stress response in rats subjected to strenuous activity in a running wheel. In humans, a low dose of alcohol improved performance of a complex mental problem-solving task under stressful conditions. However, in some individuals, at certain doses, alcohol may induce rather than reduce the body's stress response.

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Much research demonstrates that alcohol actually induces the stress response by stimulating hormone release by the hypothalamus, pituitary, and adrenal glands. This finding has been demonstrated in animal studies. In one study with rats, the administration of alcohol initiated the physiological stress response, measured by increased levels of corticosterone. In addition to stimulating the hormonal stress response, chronic exposure to alcohol also results in an increase in adrenaline.

### **Stress, Alcoholism, and Relapse**

Stress may be linked to social drinking, and the physiological response to stress is different in actively drinking alcoholics compared with non-alcoholics. Researchers have found that animals preferring alcohol over water have a different physiological response to stress than animals that do not prefer alcohol.

There is a clear connection in the alcoholic between stress and relapse: Among abstinent alcoholics, personally threatening, severe, and chronic life stressors may lead to alcohol relapse. Brown and colleagues studied a group of men who completed inpatient alcoholism treatment and later experienced severe and prolonged psychosocial stress prior to and independent of any alcohol use. The researchers found that subjects who relapsed experienced twice as much severe and prolonged stress before their return to drinking as those who remained abstinent. In this study, severe psychosocial stress was related to relapse in alcoholic males who expected alcohol to reduce their stress. Those most vulnerable to stress-related relapse scored low on measures of coping skills, self-efficacy, and social support. Stress-related relapse was greatest among those who had less confidence in their ability to resist drinking and among those who relied on drinkers for social support. Although many factors can influence a return to drinking, Brown and colleagues note that stress may exert its greatest influence on the initial consumption of alcohol after a period of abstinence.

## Alcohol and Aging

Persons age 65 and older constitute the fastest growing segment of the American population. Although the extent of alcoholism among the elderly is debated, the diagnosis and treatment of alcohol problems are likely to become increasingly important as the elderly population grows.

### **Drinking Prevalence and Patterns Among the Elderly**

Surveys of different age groups in the community suggest that the elderly, generally defined as persons older than 65, consume less alcohol and have fewer alcohol-related problems than younger persons. However, some surveys that track individuals over time suggest that a person's drinking pattern remains relatively stable with age, perhaps reflecting societal norms that prevailed when the person began drinking.

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In contrast to most studies of the general population, surveys conducted in health care settings have found increasing prevalence of alcoholism among the older population. Surveys indicate that 6 to 11 percent of elderly patients admitted to hospitals exhibit symptoms of alcoholism, as do 20 percent of elderly patients in psychiatric wards and 14 percent of elderly patients in emergency rooms. In acute-care hospitals, rates of alcohol-related admissions for the elderly are similar to those for heart attacks. Yet hospital staff are significantly less likely to recognize alcoholism in an older patient than in a younger patient.

### **Combined Effects of Alcohol and Aging**

Although many medical and other problems are associated with both aging and alcohol misuse, the extent to which these two factors may interact to contribute to disease is unclear. Some examples of potential alcohol-aging interactions include the following:

- The incidence of hip fractures in the elderly increases with alcohol consumption. This increase can be explained by falls while intoxicated combined with a more pronounced decrease in bone density in elderly persons with alcoholism compared with elderly nonalcoholics.
- Studies of the general population suggest that *moderate* alcohol consumption (up to two drinks per day for men and one drink per day for women) may confer some protection from heart disease. Although research on this issue is limited, evidence shows that moderate drinking also has a protective effect among those older than 65. Because of age-related body changes in both men and women.
- Alcohol-involved traffic crashes are an important cause of trauma and death in all age groups. The elderly are the fastest growing segment of the driving population. A person's crash risk per mile increases starting at age 55, exceeding that of a young, beginning driver by age 80. In addition, older drivers tend to be more seriously injured than younger drivers in crashes of equivalent magnitude. Age may interact with alcoholism to increase driving risk. For example, an elderly driver with alcoholism is more impaired than an elderly driver without alcoholism after consuming an equivalent dose of alcohol, and has a greater risk of a crash.
- Long-term alcohol consumption activates enzymes that break down toxic substances, including alcohol. Upon activation, these enzymes may also break down some common prescription medications. The average person older than 65 takes two to seven prescription medications daily. Alcohol-medication interactions are especially common among the elderly, increasing the risk of negative health effects and potentially influencing the effectiveness of the medications.
- Depressive disorders are more common among the elderly than among younger people and tend to co-occur with alcohol misuse. Data from the National Longitudinal Alcohol Epidemiologic Survey demonstrate that, among persons older than 65, those with alcoholism are three times more likely to have a major depressive disorder than are those without

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alcoholism. Thirty percent of alcoholics over the age of 65 were found to have concurrent psychiatric disorders. Among persons older than 65, moderate and heavy drinkers are 16 times more likely than nondrinkers to die of suicide, which is commonly associated with depressive disorders.

### **Aging and Increased Alcohol Sensitivity**

Limited research suggests that sensitivity to alcohol's health effects may increase with age. One reason is that the elderly achieve a higher blood alcohol concentration (BAC) than younger people after consuming an equal amount of alcohol. The higher BAC results from an age-related decrease in the amount of body water in which to dilute the alcohol. Therefore, although they can metabolize and eliminate alcohol as efficiently as younger persons, the elderly are at increased risk for intoxication and adverse effects.

Aging also interferes with the body's ability to adapt to the presence of alcohol. Through a decreased ability to develop tolerance, elderly subjects persist in exhibiting certain effects of alcohol (e.g., incoordination) at lower doses than younger subjects whose tolerance increases with increased consumption. Thus, an elderly person can experience the onset of alcohol problems even though his or her drinking pattern remains unchanged.

### **Aging, Alcohol, and the Brain**

Aging and alcoholism produce similar deficits in intellectual (i.e., cognitive) and behavioral functioning. Alcoholism may accelerate normal aging or cause premature aging of the brain. Using magnetic resonance imaging techniques, Pfefferbaum and colleagues found more brain tissue loss in subjects with alcoholism than in those without alcoholism, even after their ages had been taken into account. In addition, older subjects with alcoholism exhibited more brain tissue loss than younger subjects with alcoholism, often despite similar total lifetime alcohol consumption. These results suggest that aging may render a person more susceptible to alcohol's effects.

The frontal lobes of the brain are especially vulnerable to long-term heavy drinking. Research shows that shrinkage of the frontal lobes increases with alcohol consumption and is associated with intellectual impairment in both older and younger subjects with alcoholism. In addition, older persons with alcoholism are less likely to recover from cognitive deficits during abstinence than are younger persons with alcoholism.

Age-related changes in volume also occur in the cerebellum, a part of the brain involved in regulating posture and balance. Thus, long-term alcohol misuse could accelerate the development of age-related postural instability, increasing the likelihood of falls.

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### **Treatment of Alcoholism in the Elderly**

Studies indicate that elderly persons with alcohol problems are at least as likely as younger persons to benefit from alcoholism treatment. The outcomes are more favorable among persons with shorter histories of problem drinking (i.e., late onset). Additionally, although evidence is not entirely consistent, some studies suggest that treatment outcomes may be improved by treating older patients in age-segregated settings.

The use of medications to promote abstinence has not been studied extensively in elderly subjects. However, one study has suggested that naltrexone (ReVia®) may help prevent relapse to alcoholism in subjects ages 50 to 70. Results of research in animals suggest that age-related alterations in specific chemical messenger systems in the brain may alter the effectiveness of medications used to treat alcoholism and mental disorders.

### **Alcohol in the Workplace**

Drinking among U.S. workers can threaten public safety, impair job performance, and result in costly medical, social, and other problems affecting employees and employers alike. Productivity losses attributed to alcohol were estimated at \$119 billion for 1995.

#### **Factors Contributing to Employee Drinking**

Drinking rates vary among occupations, but alcohol-related problems are not characteristic of any social segment, industry, or occupation. Drinking is associated with the workplace culture and acceptance of drinking, workplace alienation, the availability of alcohol, and the existence and enforcement of workplace alcohol policies.

**Workplace Culture.** The culture of the workplace may either accept and encourage drinking or discourage and inhibit drinking. A workplace's tolerance of drinking is partly influenced by the gender mix of its workers. Studies of male-dominated occupations have described heavy drinking cultures in which workers use drinking to build solidarity and show conformity to the group. Some male-dominated occupations therefore tend to have high rates of heavy drinking and alcohol-related problems. In predominantly female occupations both male and female employees are less likely to drink and to have alcohol-related problems than employees of both sexes in male-dominated occupations.

**Workplace Alienation.** Work that is boring, stressful, or isolating can contribute to employees' drinking. Employee drinking has been associated with low job autonomy, lack of job complexity, lack of control over work conditions and products, boredom, sexual harassment, verbal and physical aggression, and disrespectful behavior.

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**Alcohol Availability.** The availability and accessibility of alcohol may influence employee drinking. More than two-thirds of the 984 workers surveyed at a large manufacturing plant said it was "easy" or "very easy" to bring alcohol into the workplace, to drink at work stations, and to drink during breaks. Twenty-four percent reported drinking alcohol at work at least once during the year before the survey. In a survey of 6,540 employees at 16 worksites representing a range of industries, 23 percent of upper-level managers reported drinking alcohol during working hours in the previous month.

**Supervision.** Limited work supervision, often a problem on evening shifts, has been associated with employee alcohol problems. In one study of 832 workers at a large manufacturing plant, workers on evening shifts, during which supervision was reduced, were more likely than those on other shifts to report drinking at work.

**Alcohol Policies.** There is wide variation in the existence of alcohol policies, in employees' awareness of them, and in their enforcement in workplaces across the country. Researchers found that most managers and supervisors in one large manufacturing plant had little knowledge of the company's alcohol policy. In addition, supervisors were under constant pressure to keep production moving and were motivated to discipline employees for drinking only if the drinking was compromising production or jeopardizing safety. Workers' knowledge that policies were rarely enforced seemed to encourage drinking.

### Effects of Employee Drinking

Alcohol-related job performance problems are caused not only by on-the-job drinking but also by heavy drinking outside of work. Ames and colleagues found a positive relationship between the frequency of being "hungover" at work and the frequency of feeling sick at work, sleeping on the job, and having problems with job tasks or co-workers. The hangover effect was demonstrated among pilots whose performance was tested in flight simulators. Yesavage and Leirer found evidence of impairment 14 hours after pilots reached blood alcohol concentrations (BACs) of between 0.10 percent and 0.12 percent. Morrow and colleagues found that pilots were still significantly impaired 8 hours after reaching a BAC of 0.10 percent. Drinking at work, problem drinking, and frequency of getting drunk in the past 30 days were positively associated with frequency of absenteeism, arriving late to work or leaving early, doing poor work, doing less work, and arguing with co-workers.

### Preventing Alcohol Problems in the Workplace

Health promotion programs offered in the workplace may reduce employees' alcohol-related problems. An employee health promotion program delivered in three 2-hour sessions at one manufacturing plant was designed to increase participants' awareness of the health risks related to stress and drinking. More than one-half of the 294 workers attended the sessions. Researchers based their results on data from 120 employees who completed prestudy and poststudy

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evaluations. After 6 months, 76 percent of the heaviest drinkers reportedly reduced their alcohol consumption. Moderate drinkers also reduced their consumption, and participants reported changes in their attitudes toward drinking and drinking and driving, knowledge about problem drinking, and recognition of signs of a drinking problem.

### **Managing Alcohol Problems in the Workplace**

One function of employee assistance programs (EAPs) is to identify and intervene in employees' alcohol problems. EAPs may be provided by labor unions, management (as part of the employee benefit package), or through a union-management collaboration.

Although the services offered vary, EAPs usually train supervisors to recognize problems and refer workers to the EAP; provide confidential and timely assessment; refer employees for diagnosis, treatment, and other assistance; work with community resources to provide needed services; and conduct followup after treatment.

From 1992 to 1993, a national survey estimated that 33 percent of U.S. worksites with 50 or more full-time employees had an EAP. A 1992 survey of the alcohol programs offered through EAPs at 1,507 worksites with 50 or more employees found that 16 percent offered individual counseling, 22 percent had group sessions, and 41 percent provided employees with written materials. Unionized and larger worksites were more likely to offer alcohol programs than were nonunionized, smaller worksites.

## Alcohol and Women

Women appear to be more vulnerable than men to many adverse consequences of alcohol use. Women achieve higher concentrations of alcohol in the blood and become more impaired than men after drinking equivalent amounts of alcohol. Research also suggests that women are more susceptible than men to alcohol-related organ damage and to trauma resulting from traffic crashes and interpersonal violence.

### **Prevalence of Women's Drinking**

Household surveys indicate that alcohol use is more prevalent among men than women in the United States. In one survey, 34 percent of women reported consuming at least 12 standard drinks during the previous year compared with 56 percent of men. Among drinkers surveyed, 10 percent of women and 22 percent of men consumed two or more drinks per day on average. Men are also more likely than women to become alcohol dependent.

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Women's drinking is most common between ages 26 and 34 and among women who are divorced or separated. Binge drinking (i.e., consumption of five or more drinks per occasion on 5 or more days in the past month) is most common among women ages 18 to 25. Among racial groups, women's drinking is more prevalent among whites, although black women are more likely to drink heavily.

### **Metabolism**

Women absorb and metabolize alcohol differently than men. In general, women have less body water than men of similar body weight, so that women achieve higher concentrations of alcohol in the blood after drinking equivalent amounts of alcohol. In addition, women appear to eliminate alcohol from the blood faster than men. This finding may be explained by women's higher liver volume per unit lean body mass, because alcohol is metabolized almost entirely in the liver.

### **Consequences of Alcohol Use**

Research suggests that women are more vulnerable than men to alcohol-related organ damage, trauma, and legal and interpersonal difficulties.

**Liver Damage.** Compared with men, women develop alcohol-induced liver disease over a shorter period of time and after consuming less alcohol. In addition, women are more likely than men to develop alcoholic hepatitis and to die from cirrhosis. Animal research suggests that women's increased risk for liver damage may be linked to physiological effects of the female reproductive hormone estrogen.

**Brain Damage.** Views of the brain obtained by magnetic resonance imaging (MRI) suggest that women may be more vulnerable than men to alcohol-induced brain damage. Using MRI, researchers found that a brain region involved in coordinating multiple brain functions was significantly smaller among alcoholic women compared with both nonalcoholic women and alcoholic men. These differences remained significant after measurements were adjusted for head size. Conversely, a study measuring metabolic energy utilization in selected brain regions found a significant difference between alcoholic and nonalcoholic men but no significant difference between alcoholic and nonalcoholic women. These results are not consistent with a greater vulnerability to alcoholic brain damage in women. However, the female alcoholics reported less severe alcohol use compared with the male alcoholics studied.

**Breast Cancer.** Many studies report that moderate to heavy alcohol consumption increases the risk for breast cancer, although one recent study found no increased breast cancer risk associated with consumption of up to one drink per day, the maximum drinking level reported by most women.

**Violent Victimization.** A survey of female college students found a significant relationship between the amount of alcohol the women reported drinking each

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week and their experiences of sexual victimization. Another study found that female high school students who used alcohol in the past year were more likely than non-drinking students to be the victims of dating violence.

**Traffic Crashes.** Although women are less likely than men to drive after drinking and to be involved in fatal alcohol-related crashes, women have a higher relative risk of driver fatality than men at similar blood alcohol concentrations. Laboratory studies of the effects of alcohol on responding to visual cues and other tasks suggest that there may be gender differences in how alcohol affects the performance of driving tasks.

Women's lower rates of drinking and driving may be attributed to their lower tendency toward risk taking compared with men. Women are also less likely to view drinking and driving as acceptable behavior. In a 1990 national household survey, 17 percent of women, compared with 27 percent of men, agreed that it was acceptable for a person to drink one or two drinks before driving. Nevertheless, the proportion of female drivers involved in fatal crashes is increasing. In 1996, 16 percent of all drivers involved in alcohol-related fatal crashes were women, compared with 13 percent in 1986 and 12 percent in 1980.

### **Risk Factors for Women's Alcohol Use**

Factors that may increase women's risk for alcohol abuse or dependence include genetic influences, early initiation of drinking, and victimization.

**Genetic Factors.** The relative contribution of genetic factors to women's risk for alcoholism has been debated. A survey of 2,163 female twins revealed greater similarity between identical twins compared with fraternal twins on measures of alcohol consumption. Similar studies including more than 12,000 twin pairs from the general population have confirmed that among both male and female twin pairs, identical twins are more likely than fraternal twins to have similar rates of alcohol dependence, alcohol abuse, and heavy alcohol consumption.

Studies of women who had been adopted at birth have shown a significant association between alcoholism in adoptees and their biological parents. In addition, antisocial personality (e.g., aggressiveness) in biological parents may predict alcoholism in both male and female adoptees.

**Age of Initiating Drinking.** Results of a large nationwide survey show that more than 40 percent of persons who initiated drinking before age 15 were diagnosed as alcohol dependent at some point in their lives. Rates of lifetime dependence declined to approximately 10 percent among those who began drinking at age 20 or older. The annual rate of this decline was similar for both genders. Although in the past women generally started drinking at later ages than men, more recent survey data show that this difference has nearly disappeared.

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**Victimization.** Using data collected in a large general population survey, Wilsnack and colleagues found that women who reported being sexually abused in childhood were more likely than other women to have experienced alcohol-related problems (e.g., family discord or household accidents) and to have one or more symptoms of alcohol dependence. Another study found that women in alcoholism treatment were significantly more likely to report childhood sexual abuse and father-to-daughter verbal aggression or physical violence compared with women in the general population.

Physical abuse during adulthood has also been associated with women's alcohol use and related problems. One study found that significantly more women undergoing alcoholism treatment experienced severe partner violence (e.g., kicking, punching, or threatening with a weapon) compared with other women in the community. In addition, among women in the community group, those with AOD-related problems reported significantly higher rates of severe partner violence than women without such problems. Although the findings indicate that partner violence and AOD problems co-occur among women, the data do not indicate whether the association is causal.

### Alcohol-Medication Interactions

Many medications can interact with alcohol, leading to increased risk of illness, injury, or death. For example, it is estimated that alcohol-medication interactions may be a factor in at least 25 percent of all emergency room admissions. An unknown number of less serious interactions may go unrecognized or unrecorded.

### Alcohol-Drug Interactions

More than 2,800 prescription drugs are available in the United States, and physicians write 14 billion prescriptions annually; in addition, approximately 2,000 medications are available without prescription.

About 60 percent of men and 30 percent of women have had one or more adverse alcohol-related life events. Together with the data on medication use, these statistics suggest that some concurrent use of alcohol and medications is inevitable.

The elderly may be especially likely to mix drugs and alcohol and are at particular risk for the adverse consequences of such combinations. Although persons age 65 and older constitute only 12 percent of the population, they consume 25 to 30 percent of all prescription medications. The elderly are more likely to suffer medication side effects compared with younger persons, and these effects tend to be more severe with advancing age. Among persons age 60 or older, 10

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percent of those in the community--and 40 percent of those in nursing homes--fulfill criteria for alcohol abuse.

### How Alcohol and Drugs Interact

To exert its desired effect, a drug generally must travel through the bloodstream to its site of action, where it produces some change in an organ or tissue. The drug's effects then diminish as it is processed (metabolized) by enzymes and eliminated from the body. Alcohol behaves similarly, traveling through the bloodstream, acting upon the brain to cause intoxication, and finally being metabolized and eliminated, principally by the liver. The extent to which an administered dose of a drug reaches its site of action may be termed its *availability*. Alcohol can influence the effectiveness of a drug by altering its availability. Typical alcohol-drug interactions include the following:

First, an acute dose of alcohol (a single drink or several drinks over several hours) may inhibit a drug's metabolism by competing with the drug for the same set of metabolizing enzymes. This interaction prolongs and enhances the drug's availability, potentially increasing the patient's risk of experiencing harmful side effects from the drug.

Second, in contrast, chronic (long-term) alcohol ingestion may activate drug-metabolizing enzymes, thus decreasing the drug's availability and diminishing its effects. After these enzymes have been activated, they remain so even in the absence of alcohol, affecting the metabolism of certain drugs for several weeks after cessation of drinking. Thus, a recently abstinent chronic drinker may need higher doses of medications than those required by nondrinkers to achieve therapeutic levels of certain drugs.

Third, enzymes activated by chronic alcohol consumption transform some drugs into toxic chemicals that can damage the liver or other organs. Fourth, alcohol can magnify the inhibitory effects of sedative and narcotic drugs at their sites of action in the brain. To add to the complexity of these interactions, some drugs affect the metabolism of alcohol, thus altering its potential for intoxication and the adverse effects associated with alcohol consumption.

### Some Specific Interactions

**Anesthetics.** Chronic alcohol consumption increases the dose of propofol (Diprivan) required to induce loss of consciousness. Chronic alcohol consumption increases the risk of liver damage that may be caused by the anesthetic gases enflurane (Ethrane) and halothane (Fluothane).

**Antibiotics.** In combination with acute alcohol consumption, some antibiotics may cause nausea, vomiting, headache, and possibly convulsions; among these antibiotics are furazolidone (Furoxone), griseofulvin (Grisactin and others), metronidazole (Flagyl), and the antimalarial quinacrine (Atabrine). Isoniazid and

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rifampin are used together to treat tuberculosis, a disease especially problematic among the elderly and among homeless alcoholics. Acute alcohol consumption decreases the availability of isoniazid in the bloodstream, whereas chronic alcohol use decreases the availability of rifampin. In each case, the effectiveness of the medication may be reduced.

**Anticoagulants.** Warfarin (Coumadin) is prescribed to retard the blood's ability to clot. Acute alcohol consumption enhances warfarin's availability, increasing the patient's risk for life-threatening hemorrhages. Chronic alcohol consumption reduces warfarin's availability, lessening the patient's protection from the consequences of blood-clotting disorders.

**Antidepressants.** Alcoholism and depression are frequently associated, leading to a high potential for alcohol-antidepressant interactions. Alcohol increases the sedative effect of tricyclic antidepressants such as amitriptyline (Elavil and others), impairing mental skills required for driving. Acute alcohol consumption increases the availability of some tricyclics, potentially increasing their sedative effects; chronic alcohol consumption appears to increase the availability of some tricyclics and to decrease the availability of others. The significance of these interactions is unclear. These chronic effects persist in recovering alcoholics.

A chemical called tyramine, found in some beers and wine, interacts with some anti-depressants, such as monoamine oxidase inhibitors, to produce a dangerous rise in blood pressure. As little as one standard drink may create a risk that this interaction will occur.

**Antidiabetic medications.** Oral hypoglycemic drugs are prescribed to help lower blood sugar levels in some patients with diabetes. Acute alcohol consumption prolongs, and chronic alcohol consumption decreases, the availability of tolbutamide (Orinase). Alcohol also interacts with some drugs of this class to produce symptoms of nausea and headache such as those described for metronidazole.

**Antihistamines.** Drugs such as diphenhydramine (Benadryl and others) are available without prescription to treat allergic symptoms and insomnia. Alcohol may intensify the sedation caused by some antihistamines. These drugs may cause excessive dizziness and sedation in older persons; the effects of combining alcohol and antihistamines may therefore be especially significant in this population.

**Antipsychotic medications.** Drugs such as chlorpromazine (Thorazine) are used to diminish psychotic symptoms such as delusions and hallucinations. Acute alcohol consumption increases the sedative effect of these drugs, resulting in impaired coordination and potentially fatal breathing difficulties. The combination of chronic alcohol ingestion and antipsychotic drugs may result in liver damage.

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**Antiseizure medications.** These drugs are prescribed mainly to treat epilepsy. Acute alcohol consumption increases the availability of phenytoin (Dilantin) and the risk of drug-related side effects. Chronic drinking may decrease phenytoin availability, significantly reducing the patient's protection against epileptic seizures, even during a period of abstinence.

**Antiulcer medications.** The commonly prescribed antiulcer medications cimetidine (Tagamet) and ranitidine (Zantac) increase the availability of a low dose of alcohol under some circumstances. The clinical significance of this finding is uncertain, since other studies have questioned such interaction at higher doses of alcohol.

**Cardiovascular medications.** This class of drugs includes a wide variety of medications prescribed to treat ailments of the heart and circulatory system. Acute alcohol consumption interacts with some of these drugs to cause dizziness or fainting upon standing up. These drugs include nitroglycerin, used to treat angina, and reserpine, methyldopa (Aldomet), hydralazine (Apresoline and others), and guanethidine (Ismelin and others), used to treat high blood pressure. Chronic alcohol consumption decreases the availability of propranolol (Inderal), used to treat high blood pressure, potentially reducing its therapeutic effect.

**Narcotic pain relievers.** These drugs are prescribed for moderate to severe pain. They include the opiates morphine, codeine, propoxyphene (Darvon), and meperidine (Demerol). The combination of opiates and alcohol enhances the sedative effect of both substances, increasing the risk of death from overdose. A single dose of alcohol can increase the availability of propoxyphene, potentially increasing its sedative side effects.

**Nonnarcotic pain relievers.** Aspirin and similar nonprescription pain relievers are most commonly used by the elderly. Some of these drugs cause stomach bleeding and inhibit blood from clotting; alcohol can exacerbate these effects. Older persons who mix alcoholic beverages with large doses of aspirin to self-medicate for pain are therefore at particularly high risk for episodes of gastric bleeding. In addition, aspirin may increase the availability of alcohol, heightening the effects of a given dose of alcohol.

Chronic alcohol ingestion activates enzymes that transform acetaminophen (Tylenol and others) into chemicals that can cause liver damage, even when acetaminophen is used in standard therapeutic amounts. These effects may occur with as little as 2.6 grams of acetaminophen in persons consuming widely varying amounts of alcohol.

**Sedatives and hypnotics ("sleeping pills").** Benzodiazepines such as diazepam (Valium) are generally prescribed to treat anxiety and insomnia. Because of their greater safety margin, they have largely replaced the barbiturates, now used mostly in the emergency treatment of convulsions.

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Doses of benzodiazepines that are excessively sedating may cause severe drowsiness in the presence of alcohol, increasing the risk of household and automotive accidents. This may be especially true in older people, who demonstrate an increased response to these drugs. Low doses of flurazepam (Dalmane) interact with low doses of alcohol to impair driving ability, even when alcohol is ingested the morning after taking Dalmane. Since alcoholics often suffer from anxiety and insomnia, and since many of them take morning drinks, this interaction may be dangerous.

The benzodiazepine lorazepam (Ativan) is being increasingly used for its antianxiety and sedative effects. The combination of alcohol and lorazepam may result in depressed heart and breathing functions; therefore, lorazepam should not be administered to intoxicated patients.

Acute alcohol consumption increases the availability of barbiturates, prolonging their sedative effect. Chronic alcohol consumption decreases barbiturate availability through enzyme activation. In addition, acute or chronic alcohol consumption enhances the sedative effect of barbiturates at their site of action in the brain, sometimes leading to coma or fatal respiratory depression.

### Treatment for Alcoholism

More than 700,000 Americans receive alcoholism treatment on any given day. However, the techniques of alcoholism therapy have traditionally been based on clinical experience and intuition, with little rigorous validation of their effectiveness. Over the past 20 years, modern methods of evaluating medical therapies have been increasingly applied to alcoholism treatment. These methods include the use of control groups for comparison purposes, random assignment of study participants to different treatment groups and, to the greatest extent possible, follow-up of all patients who entered the study.

#### Twelve-Step Self-Help Programs

Self-help groups are the most commonly sought source of help for alcohol-related problems. Alcoholics Anonymous (AA), one of the most commonly known self-help groups, outlines 12 consecutive activities, or steps, that alcoholics should achieve during the recovery process. Alcoholics can become involved with AA before entering professional treatment, as a part of it, or as aftercare following professional treatment. Although AA appears to produce positive outcomes in many of its members, its efficacy has rarely been assessed in randomized clinical trials.

One randomized study of patients entering employee assistance programs compared inpatient treatment combined with AA with referral to AA alone. This

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study found that inpatient treatment, a combination of professional treatment and AA, will achieve better results for more people than AA alone.

The beneficial effects of AA may be attributable in part to the replacement of the participant's social network of drinking friends with a fellowship of AA members who can provide motivation and support for maintaining abstinence. In addition, AA's approach often results in the development of coping skills, many of which are similar to those taught in more structured psychosocial treatment settings, thereby leading to reductions in alcohol consumption.

### **Psychosocial Therapy**

The following sections deal with selected recent approaches or considerations relevant to the psychosocial treatment of alcohol-related problems.

### **Motivational Enhancement Therapy**

Motivational enhancement therapy (MET) begins with the assumption that the responsibility and capacity for change lie within the client. The therapist begins by providing individualized feedback about the effects of the patient's drinking. Working closely together, therapist and patient explore the benefits of abstinence, review treatment options, and design a plan to implement treatment goals. Analysis suggests that MET may be one of the most cost-effective of available treatment methods. In one study, the motivational interviewing technique—a key component of MET—was shown to overcome patients' reluctance to enter treatment more effectively than did conventional techniques.

### **Couples Therapy**

Evidence indicates that involvement of a nonalcoholic spouse in a treatment program can improve patient participation rates and increase the likelihood that the patient will alter drinking behavior after treatment ends.

There are various approaches to marital family therapy. Behavioral-marital therapy (BMT) combines a focus on drinking with efforts to strengthen the marital relationship through shared activities and the teaching of communication and conflict evaluation skills. O'Farrell and colleagues combined couples therapy with the learning and rehearsal of a relapse prevention plan. Among alcoholics with severe marital and drinking problems, the combination approach produced improved marital relations and higher abstinence rates through 30 months of follow-up compared with patients undergoing only BMT.

### **Brief Interventions**

Many persons with alcohol-related problems receive counseling from primary care physicians or nursing staff in the context of five or fewer standard office visits. Such treatment, known as brief intervention, generally consists of straightforward information on the negative consequences of alcohol consumption along with practical advice on strategies and community resources to achieve moderation or abstinence. Two controlled trials in the United States

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and Canada demonstrated that this approach reduced drinking, alcohol-related problems, and patients' use of health care services. Most brief interventions are designed to help those at risk for developing alcohol-related problems to reduce their alcohol consumption. Alcohol-dependent patients are encouraged to enter specialized treatment with the goal of complete abstinence.

Research indicates that brief intervention for alcohol problems is more effective than no intervention and often as effective as more extensive intervention. To identify the key ingredients of brief intervention, Miller and Sanchez proposed six elements summarized by the acronym FRAMES: feedback, responsibility, advice, menu of strategies, empathy, and self-efficacy. The importance of these elements in enhancing effectiveness has been supported by further review. Goal setting, followup, and timing also have been identified as important to the effectiveness of brief intervention.

***Feedback of Personal Risk.*** Most health professionals delivering brief intervention provide patients with feedback on their risks for alcohol problems based on such factors as their current drinking patterns; problem indicators, such as laboratory test results; and any medical consequences of their drinking. For example, a physician may tell a patient that his or her drinking may be contributing to a current medical problem, such as hypertension, or may increase the risk for certain health problems.

***Responsibility of the Patient.*** Perceived personal control has been recognized to motivate behavior change. Therefore, brief intervention commonly emphasizes the patient's responsibility and choice for reducing drinking. For example, a doctor or nurse may tell patients that "No one can make you change or make you decide to change. What you do about your drinking is up to you."

***Advice To Change.*** In some types of brief intervention, professionals give patients explicit advice to reduce or stop drinking. While expressing concern about the patient's current drinking and the related health risks, the physician may discuss guidelines for "low-risk" drinking.

***Menu of Ways To Reduce Drinking.*** Health professionals providing brief intervention may offer patients a variety of strategies from which to choose. These may include setting a specific limit on alcohol consumption; learning to recognize the antecedents of drinking and developing skills to avoid drinking in high-risk situations; planning ahead to limit drinking; pacing one's drinking (e.g., sipping, measuring, diluting, and spacing drinks); and learning to cope with the everyday problems that may lead to drinking. Health care professionals often give their patients self-help materials to present such strategies and to help them carry these strategies out. Self-help materials often include drinking diaries to help patients monitor their abstinent days and the number of drinks consumed on drinking days, record instances when they are tempted to drink or experience social pressure to drink, and note the alternatives to drinking that they use. When

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working with alcohol-dependent patients, abstinence, rather than reduced drinking, is the goal of brief intervention.

***Empathetic Counseling Style.*** A warm, reflective, and understanding style of delivering brief intervention is more effective than an aggressive, confrontational, or coercive style. Miller and Rollnick found that when they used an empathetic counseling style, patients' drinking was reduced by 77 percent, as opposed to 55 percent when a confrontational approach was used.

***Self-Efficacy or Optimism of the Patient.*** Health professionals delivering brief intervention commonly encourage patients to rely on their own resources to bring about change and to be optimistic about their ability to change their drinking behavior. Brief intervention often includes motivation-enhancing techniques (e.g., eliciting and reinforcing self-motivating statements, such as "I am worried about my drinking and want to cut back," and emphasizing the patient's strengths) to encourage patients to develop, implement, and commit to plans to stop drinking.

***Establishing a Drinking Goal.*** Patients are more likely to change their drinking behavior when they are involved in goal setting. The drinking goal usually is negotiated between the patient and physician and may be presented in writing as a prescription from the doctor or as a contract signed by the patient.

***Followup.*** The health care professional continues to follow up on the patient's progress and provide ongoing support. Followup may take the form of telephone calls from office staff, repeat office visits, or repeat physical examinations or laboratory tests.

***Timing.*** Much of the research investigating the relationship between an individual's readiness to change and actual behavior change is based on studies of smoking cessation. Research findings have been applied to reducing drinking. Individuals are most likely to make behavior changes when they perceive that they have a problem and when they feel they can change. Some patients may not be ready to change when brief intervention begins, but may be ready when they experience an alcohol-related illness or injury.

### **Effectiveness of Brief Intervention**

Studies have examined the effectiveness of brief intervention for motivating alcohol-dependent patients to enter long-term alcohol treatment. Among alcoholics identified in an emergency care setting, 65 percent of those receiving brief counseling kept a subsequent appointment for specialized treatment, compared with 5 percent of those who did not receive counseling.

Some studies conducted among alcohol-dependent patients have found that brief intervention is as effective as more extensive treatment approaches used in specialized alcohol treatment settings. Edwards and colleagues compared the effectiveness of one session giving brief advice to stop drinking with standard

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alcohol treatment among 100 alcohol-dependent men. The brief advice emphasized personal responsibility to stop drinking and encouraged group members to return to work and improve their marriages. Group members also received a monthly followup telephone call. The group receiving standard alcohol treatment was admitted for an average of 3 weeks' inpatient alcoholism treatment, attended an average of ten 30-minute psychiatric outpatient counseling sessions, and received monthly followup visits. One year later, both groups reported a 40-percent decrease in alcohol-related problems. After 2 years, patients with less severe problems were more likely to report improvement if they received brief intervention than if they received intensive treatment. However, patients with more severe problems were more likely to report improvement if they received intensive treatment.

In summary, variations of brief intervention have been found effective for helping non-alcohol-dependent patients reduce or stop drinking, for motivating alcohol-dependent patients to enter long-term alcohol treatment, and for treating some alcohol-dependent patients.

### **Pharmacotherapy**

More recently, research has focused on the development of medications for blocking alcohol-brain interactions that might promote alcoholism. In 1995 the U.S. Food and Drug Administration approved the use of the medication naltrexone (ReVia™) as an aid in preventing relapse among recovering alcoholics who are simultaneously undergoing psychosocial therapy. This approval was based largely on two randomized controlled studies that showed decreased alcohol consumption for longer periods in naltrexone-treated patients compared with those who received a placebo.

As is the case with all diseases, however, naltrexone is only effective if taken on a regular basis. Like all medications, naltrexone has side effects. One recent study reported a high rate of side effects, which probably explains why this study, in contrast with most other studies, failed to find naltrexone effective.

Acamprosate showed promise in treating alcoholism in several randomized controlled European trials involving more than 3,000 alcoholic subjects who were also undergoing psychosocial treatment. Analysis of combined results showed that more than twice as many alcoholics receiving acamprosate remained abstinent up to 1 year compared with subjects receiving psychosocial treatment alone.

Research suggests that some medications may be more effective for certain types of alcoholics. For example, when ondansetron (Zofran®) was combined with psychotherapy, alcoholics who had begun drinking heavily before age 25 (i.e., early-onset alcoholics) decreased their alcohol consumption and increased their number of abstinent days, but later onset alcoholics did not. Sertraline (Zoloft®), in contrast, appears to reduce drinking in late-onset, but not early-

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onset, alcoholics. However, fluoxetine (Prozac<sup>®</sup>), a medication related to sertraline, has not been found to be effective in late-onset alcoholism.

In conclusion, research supports the concept of using medications as an adjunct to the psychosocial therapy of alcohol abuse and alcoholism. However, additional clinical trials are required to identify those patients most likely to benefit from such an approach, to determine the most appropriate medications for different patient types, to establish optimal dosages, and to develop strategies for enhancing patient compliance with medication regimens.

### **Economic Impact of Alcoholism**

Economic analysis is used to estimate the costs of alcohol-related problems and to assist in the study of alcoholism prevention and treatment methods. The costs of alcohol abuse and alcoholism are of continuing concern to policymakers and the general public. Changes in alcoholic beverage prices and taxes are of interest, because they can affect alcohol consumption and related problems. Finally, understanding the costs and cost-effectiveness of alcoholism treatment can lead to more efficient use of health care resources.

#### **The Economic Costs of Alcohol Abuse**

During the past two decades, five major studies have estimated the economic costs of alcohol abuse in the United States using the "cost of illness" approach, which expresses the multidimensional impact of a health problem in dollars. The most recent estimate of the overall economic cost of alcohol abuse was \$185 billion for 1998, which is a projection based on the comprehensive cost estimate of \$148 billion for 1992.

More than 70 percent of the estimated costs of alcohol abuse for 1998 were attributed to lost productivity (\$134.2 billion), including losses from alcohol-related illness (\$87.6 billion), premature death (\$36.5 billion), and crime (\$10.1 billion). The remaining estimated costs included health care expenditures (\$26.3 billion, or 14.3 percent of the total), such as the costs of treating alcohol abuse and dependence (\$7.5 billion) and the costs of treating the adverse medical consequences of alcohol consumption (\$18.9 billion); as well as property and administrative costs of alcohol-related motor vehicle crashes (\$15.7 billion, or 8.5 percent); and criminal justice system costs of alcohol-related crime (\$6.3 billion, or 3.4 percent).

#### **Alcoholism Treatment Cost Analysis**

A better understanding of treatment costs and cost-effectiveness can support informed decision making about the allocation of health care resources. In a review of 141 previously published studies comparing a combined total of 36 treatment types, Finney and Monahan found no significant relationship between

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cost and treatment effectiveness. Results of a randomized, nationwide clinical trial of three treatment types found that each produced comparable treatment outcomes, but at different costs. The lowest costs were associated with motivational enhancement therapy (MET), a strategy designed to mobilize the patient's own motivation and resources for change. Cost data were obtained for three of the nine treatment sites in the study. Estimated average costs for patient care were \$359 for MET; \$407 for 12-step facilitation (TSF), an approach designed to facilitate participation in 12-step programs, such as Alcoholics Anonymous; and \$433 for cognitive-behavioral therapy (CBT). However, MET was a less intensive therapy, requiring 4 treatment sessions compared with 12 sessions for either TSF or CBT. Therefore, the least expensive therapy to deliver per patient was not the least expensive per session.

Health benefits associated with alcoholism treatment programs have also been measured in terms of cost offsets, which are net reductions in health care costs among persons who have been treated for alcoholism. A recent survey of 15,000 alcoholics found modest decreases in health care costs among patients receiving treatment, mostly within the first 6 months of initiating therapy. Cost offsets were greater for persons who initially received residential (i.e., inpatient) rather than outpatient treatment.

## Resources

For more information on alcohol abuse and alcoholism, contact the following organizations:

**Al-Anon Family Group Headquarters, Inc.**

1600 Corporate Landing Parkway  
Virginia Beach, VA 23454-5617  
Phone: (757) 563-1600; Fax: (757) 563-1655  
Email: WSO@al-anon.org  
Internet address: <http://www.al-anon.alateen.org>

**Alcoholics Anonymous (AA) World Services, Inc.**

475 Riverside Drive, 11th Floor  
New York, NY 10115  
Phone: (212) 870-3400; Fax: (212) 870-3003  
Email: via AA's Web site  
Internet address: <http://www.aa.org>

**National Council on Alcoholism and Drug Dependence, Inc. (NCADD)**

20 Exchange Place, Suite 2902  
New York, NY 10005  
Phone: (212) 269-7797; Fax: (212) 269-7510  
Email: [national@ncadd.org](mailto:national@ncadd.org)  
HOPE LINE: (800) NCA-CALL (24-hour Affiliate referral)  
Internet address: <http://www.ncadd.org>

**National Institute on Alcohol Abuse and Alcoholism (NIAAA)**

Scientific Communications Branch  
6000 Executive Boulevard, Willco Building, Suite 409  
Bethesda, MD 20892-7003  
Phone: (301) 443-3860; Fax: (301) 480-1726  
Email: [niaaaweb-r@exchange.nih.gov](mailto:niaaaweb-r@exchange.nih.gov)  
Internet address: <http://www.niaaa.nih.gov>

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**ALCOHOLISM**

**POST-TEST**

1. Alcohol Dehydrogenase functions to convert alcohol into:
  - A. Acetaldehyde
  - B. Acetate
  - C. Carbon Dioxide and Water
  - D. Cytochrome P450IIE1
  
2. Which therapy activity would most likely present the greatest challenge to a patient affected by alcoholism:
  - A. Fine motor tasks such as writing
  - B. Cognitive tasks such as following and adapting a written home exercise program containing multiple sequential activities.
  - C. Balance and coordination activities such as ascending and descending stairs
  - D. Alternating gross motor activities such as such as rolling side to side
  
3. Which of the following screening mechanisms has ten questions and was developed by the World Health Organization?
  - A. CAGE Questionnaire
  - B. TWEAK Questionnaire
  - C. Alcohol Use Disorders Identification Test
  - D. Rapid Alcohol Problem Screen
  
4. Approximately 75% of all \_\_\_\_\_ cancers in the U.S. are caused by alcoholism.
  - A. Breast
  - B. Colon
  - C. Liver
  - D. Esophageal
  
5. Stellate cells in the liver do which of the following when activated by cytokines?
  - A. Diminish in number
  - B. Increase vitamin A storage capacity
  - C. Produce scar tissue
  - D. Dilate blood vessels

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6. Geriatric patients who abuse alcohol have an increased incidence of \_\_\_\_\_, and should therefore be monitored carefully by the therapist or assistant.
  - A. Cardiac arrest
  - B. Dementia
  - C. Falls and fractured hips
  - D. Vision problems
  
7. Which of the following is NOT a risk factor associated with alcohol abuse in the workplace?
  - A. Culture
  - B. Alienation
  - C. Alcohol availability
  - D. Worker socioeconomic status
  
8. A patient who combines alcohol with antihistamines will most likely be affected in which of the following ways while performing therapy related activities?
  - A. Hyperactivity
  - B. Sedation / sluggishness
  - C. Hot flashes
  - D. Dizziness
  
9. Which of the following is NOT one of the six elements of Brief Intervention?
  - A. Feedback
  - B. Advice
  - C. Consistency
  - D. Self-efficacy
  
10. The greatest percentage of costs associated with alcohol abuse are attributed to:
  - A. Lost productivity
  - B. Health care expenditures
  - C. Property and administrative costs
  - D. Criminal justice administrative costs