Alcoholism

Goals and Objectives

Course Description
"Alcoholism" is an asynchronous online continuing education course for occupational therapists and occupational therapy assistants. This course presents updated information about alcoholism including sections on causative factors, cognitive impairment, screening, comorbidity, systemic and metabolic effects, alcoholism and aging, drug interactions, and treatment.

Course Rationale
The information presented in this course is applicable for occupational therapy professionals in all settings. A greater understanding of alcoholism will enable occupational therapists and occupational therapy 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 participant 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.

Course Provider – Innovative Educational Services

Provider Contact Information – information@cheapceus.com

Course Instructor – Michael Niss, DPT

Financial/Non-financial Disclosure - Neither the Provider nor the instructor have any financial or non-financial conflict of interest related to the presentation of this CE program.

Target Audience – Occupational Therapists, Occupational Therapy Assistants

OT Scope of Practice – OT Service delivery (evaluation, intervention, outcomes); Foundational knowledge (Human body diagnoses and conditions)

Course Educational Level – This course is applicable for introductory learners.

Course Prerequisites – None

Method of Instruction – Distance Learning – Independent; Asynchronous online text-based home study

Location - Cheapceus.com

Date – Continuously available on-demand

Course Completion Requirements / Criteria for Issuance of CE Credits – Completion of instructional materials and a score of 70% or greater on the course post-test.

Continuing Education Credits – Four (4) contact hours / .4 AOTA CEUs/ NBCOT 5 PDUs

Course Fee - $39.95

Registration Information – No pre-registration required; available on-demand at Cheapceus.com

Special Needs Requests – Email: information@cheapceus.com or phone: 954-663-4101

Cancellation by the Learner – Learners may cancel their participation at any time and receive a full refund of all paid fees.

Cancellation by the Provider – Registrants for the cancelled activity will be contacted by an IES staff member via phone call or email within 12 hours of the cancellation decision. If the decision to cancel is made less than 14 hours prior to the event, each participant will be contacted no later than 2 hours prior to the scheduled start time of the activity. Registrants will receive a full refund if an activity is cancelled by the Provider. The refund will be processed back to the Registrant within 12 hours of the cancellation.

Complaint Resolution – Please call 954-663-4101 (24 hours/day, 7 days/week) to speak with a live customer service agent. Our goal is to work with our customers to resolve all issues to the customer's satisfaction with just one phone call whenever possible.

Refund Policy - Unrestricted 100% refund upon request. The request for a refund by the learner shall be honored in full without penalty or other consideration of any kind. The request for a refund may be made by the learner at any time without limitations before, during, or after course participation.

AOTA American Occupational Therapy Association Approved Provider

Innovative Educational Services is an AOTA Approved Provider of professional development. PD activity approval ID# 5471. This Distance Learning – Independent PD activity is offered at .4 CEUs; Introductory; OT Service delivery, Foundational knowledge. The assignment of AOTA CEUs does not imply endorsement of specific course content, products, or clinical procedures by AOTA.

Alcoholism

Course Outline

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.

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.

Equivalency

A standard drink is any drink that contains about 14 grams of pure alcohol (about 0.6 fluid ounces or 1.2 tablespoons). Below are standard drink equivalents as well as the number of standard drinks in different container sizes for each beverage. These are approximate, as different brands and types of beverages vary in their actual alcohol content.

A standard drink is:

- 12 oz. beer or cooler (5% alcohol)
- 8 oz. malt liquor (7% alcohol)
- 5 oz. wine (12% alcohol)
- 1.5 oz. hard liquor (40% alcohol)

Absorption

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.

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.

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. 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 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.

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. 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.

Causative Factors

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.

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.

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).

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

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.

Risk Factors

Outside influences as well as individual characteristics help determine whether a person will begin drinking and how much he or she will consume. Some of these factors increase a person's risk for problems with alcohol, whereas others serve to protect him or her from harm, as outlined below.

Many scientific studies, including research conducted among twins and children of alcoholics, have shown that genetic factors influence alcoholism. These findings show that children of alcoholics are about four times more likely than the general population to develop alcohol problems. Children of alcoholics also have a higher risk for many other behavioral and emotional problems. But alcoholism is not determined solely by the genes. In fact, more than one–half of all children of alcoholics do not become alcoholic. Many factors influence the risk of developing alcoholism. Some factors raise the risk while others lower it.
Genes are not the only things children inherit from their parents. How parents act and how they treat each other and their children has an influence on children growing up in the family. These aspects of family life also affect the risk for alcoholism. A person's risk for alcoholism increases if he or she is in a family with the following difficulties:

- an alcoholic parent is depressed or has other psychological problems;
- both parents abuse alcohol and other drugs;
- the parents' alcohol abuse is severe; and
- conflicts lead to aggression and violence in the family

Gender - Men are much more likely than women to drink in ways that are harmful.

Employment -Being employed full-time after high school was associated with a slight increase in current drinking and a slight decrease in heavy drinking. Unemployed men, but not women, especially tended to reduce their drinking. Homemakers reduced both their current and heavy drinking, but this may have been because of increasing responsibilities stemming

from marital and parental roles rather than the result of being a homemaker.

Military Service - Young adults in the military are more likely to drink heavily. The reasons for heavy drinking rates in the military include a workplace culture that supports alcohol use and the increased availability of alcohol both in and around military bases

Peer Influences - People entering college or the workforce may be especially vulnerable to the influence of peers because of their need to make new friendships. And they may increase their drinking in order to gain acceptance by peers. The phenomenon of perceived social norms—or the belief that "everyone" is drinking and drinking is acceptable—is one of the strongest correlates of drinking among young adults,

Marriage and Parenthood - Just as the move to adulthood leads to greater exploration of the world and experimentation with alcohol, assuming adult roles and responsibilities consistently curbs alcohol use. This reduction in drinking may be a result of limitations that adult roles place on social activities in general or may reflect a change in these young adults' attitudes toward drinking. Young married women have the greatest decreases in drinking behavior, and married men, compared with men in all other categories of living arrangements (i.e., living with parents, in a dormitory, alone, or in other arrangements) have the fewest increases.

Personality Characteristics - A number of personality traits have been associated with drinking greater amounts of alcohol and drinking more often, including impulsivity, risk-taking, and sensation-seeking—or the tendency to seek out new and exciting experiences. Sensation-seeking and impulsivity also have been linked to deviant behavior and nonconformity, both of which are predictors of heavy drinking and related problems among youth.

Then there are other personality traits, such as a feeling of invincibility, that are common among young adults and which can influence drinking. Many young people simply do not see themselves as vulnerable to any negative consequences that might occur because of drinking, such as having an accident or becoming dependent on alcohol. Negative moods, feelings of depression, and anxiety disorders also may influence alcohol use.

Alcohol Expectancies - Positive alcohol expectancies, or the belief that drinking will lead to positive, pleasurable experiences, play a key role in the drinking behavior of young adults. What a person expects from drinking not only predicts when he or she will begin drinking but also how

much he or she will drink throughout young adulthood. As people age through adolescence and into young adulthood, they increasingly expect benefits from drinking and become less convinced of the risks

Family Influences - During young adulthood parents may have less direct influence on their children's drinking behavior, but they still play a major protective role. The example set by parents with their own drinking has been shown to affect their children's drinking throughout their lifetime. Young people model their behavior after their parents' patterns of consumption (including quantity and frequency), situations and contexts of use, attitudes regarding use, and expectancies. The family's structure and aspects of the parent–child relationship (e.g., parenting style, attachment and bonding, nurturance, abuse or neglect, conflict, discipline, and monitoring) also have been linked to young people's alcohol use.

Genetics - Alcohol problems seem to "run" in some families. This family connection to alcoholism may be the result of a genetic link and/or may reflect the child's modeling of drinking behavior. Siblings also can influence drinking through modeling and by providing access to alcohol. It's unclear whether children of alcoholics have different drinking patterns and problems in young adulthood than those who do not have a family history of alcoholism. Research does show, however, that people with a family history of alcoholism are less likely than those with no family history to mature out of heavy drinking as they approach young adulthood.

Cognitive Impairment

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.

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

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.

Basis of 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. Widespread cognitive impairment arises 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

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, researchers 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 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.

Cognition and 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.

Memory

Alcohol primarily interferes with the ability to form new long–term memories, leaving intact previously established long–term memories and the ability to keep new information active in memory for brief periods. As the amount of alcohol consumed increases, so does the magnitude of the memory impairments. Large amounts of alcohol, particularly if consumed rapidly, can produce partial (i.e., fragmentary) or complete (i.e., en bloc) blackouts, which are periods of memory loss for events that transpired while a person was drinking. Blackouts are much more common among social drinkers than was previously assumed, and have been found to encompass events ranging from conversations to intercourse. Mechanisms underlying alcohol–induced memory impairments include disruption of activity in the hippocampus, a brain region that plays a central role in the formation of new auotbiographical memories.

Impact on Memory

The impact of alcohol on the formation of new long–term “explicit” memories (e.g., names and phone numbers) and events is far greater than the drug’s impact on the ability to recall previously established memories or to hold new information in short–term memory.

Intoxicated individuals are typically able to repeat new information immediately after its presentation and often can keep it active in short–term storage for up to a few minutes if they are not distracted. Similarly, they are also capable of retrieving information placed in long–term storage prior to acute intoxication. In contrast, alcohol impairs the ability to store information across delays longer than

a few seconds if the person is distracted between the time they are given the new information and the time they are tested. As the amount of alcohol increases, the resulting memory impairments can become much more profound, sometimes culminating in blackouts—periods for which a person is unable to remember critical elements of events, or even entire events, that occurred while he or she was intoxicated.

Blackouts

Blackouts represent episodes of amnesia, during which individuals are capable of participating in salient, emotionally charged events—as well as more mundane events—that they later cannot remember. Like milder alcohol–induced memory impairments, these periods of amnesia are primarily anterograde, meaning that alcohol impairs the ability to form new memories while the person is intoxicated, but does not typically erase memories formed before intoxication.

People experiencing blackouts are unable to recall any details whatsoever from events that occurred while they were intoxicated, despite all efforts by the drinkers or others to cue recall. it is as if the process of transferring information from short–term to long–term storage has been completely blocked. These memory impairments tend to have a distinct onset. It is usually less clear when these blackouts end because people typically fall asleep before they are over. Interestingly, people appear able to keep information active in short–term memory for at least a few seconds. As a result, they can often carry on conversations, drive automobiles, and engage in other complicated behaviors. Information pertaining to these events is simply not transferred into long–term storage.

The Hippocampus

The hippocampus is critically involved in the formation of memories for events. Hippocampal CA1 pyramidal cells assist the hippocampus in communicating with other areas of the brain. The hippocampus receives information from a wide variety of brain regions, many of them located in the tissue, called the neocortex, that blankets the brain and surrounds other brain structures. The hippocampus somehow ties information from other brain regions together to form new autobiographical memories, and CA1 pyramidal cells send the results of this processing back out to the neocortex.

Alcohol disrupts activity in the hippocampus via several routes—directly, through effects on hippocampal circuitry, and indirectly, by interfering with interactions between the hippocampus and other brain regions. The impact of alcohol on the frontal lobes remains poorly understood, but probably plays an important role in alcohol–induced memory impairments.

Diagnostic Imaging

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 positron emission tomography and magnetic resonance spectroscopy, permit clinicians and researchers 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 frontal lobes (of the cortex), which are 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.

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.

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.

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.

Cage Questionnaire - The CAGE questionnaire has sensitivities ranging from 43 to 94 percent for detecting alcohol abuse and alcoholism. CAGE 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.

- *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 Alcohol Use Disorders Identification Test (AUDIT) - The Alcohol Use Disorders Identification Test (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.

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.

TWEAK - The five-item TWEAK performs similarly to T–ACE and can be used to detect a range of drinking levels from moderate to high-risk consumption.

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 (2 points) __NO (0 points)

Have you had blackouts ***(Amnesia)?***
__Yes (2 points) __NO (0 points)

Have you ever felt the need to ***"Kut"*** down on your drinking?
__Yes (2 points) __NO (0 points)

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

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

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) - 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) - 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) - 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.

Fatty acid ethyl esters (FAEEs) - 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 (i.e., the waste product of newborns). Some evidence suggests that analysis of FAEEs in meconium may indicate timing of prenatal alcohol exposure.

Comorbidities

An understanding of alcohol-related 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 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

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 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

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 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

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-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.

Epidemiology

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

Interaction Mechanisms

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.

Specific Drug-Alcohol 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 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.

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 light-headedness 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
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.

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.

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.

Nutrient Digestion

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.

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 non-diabetic 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 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.

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. 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
>
> 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.

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

Hormonal Effects

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.

Blood Sugar Regulation

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.

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 "femininization" of male sexual characteristics, for example breast enlargement.

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 pre-menopausal 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.

Bone Metabolism

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.

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 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.

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-Related Cancers

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.

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.

Epidemiology

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. Research suggests that alcohol may play an indirect role in the development of breast cancer. The data indicates 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

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
Alcohol may affect cancer development at the genetic level by affecting oncogenes at the initiation and 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.

Co-carcinogen
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, 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.

As few as two drinks per day negate any beneficial effects of a "correct" diet on decreasing risk of colon cancer. A diet high in folic acid, a B vitamin found in fresh fruits and vegetables, decreases the risk for colon cancer, however, 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-Induced 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.

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.

Damage Mechanisms

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 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 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.

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 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

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.

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.

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 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.

Influence 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. 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. High levels of stress may influence drinking when alternative resources are lacking, when alcohol is accessible, and when the individual believes that alcohol will help to reduce the stress.

Individuals may differ in the amount of alcohol they consume in response to stress. Such differences may be related in part to 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.

A positive correlation between stress and alcohol consumption suggest that drinking may take place in response to chronic stress that is perceived as unavoidable.

Whether humans drink in response to uncontrollable stress is less clear. In both humans and animals, drinking appears to follow stress. Some research, however, shows that drinking may take place in anticipation of or during times of stress.

Women and Alcohol

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.

Epidemiology

Household surveys indicate that alcohol use is more prevalent among men than women in the United States. 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 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

Data 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.
- ***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 amounts of alcohol the women reported drinking each 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.

Drinking and College Students

People typically drink the heaviest in their late teens and early to mid-twenties. This high level of alcohol use comes at an age when people are moving away from parental restrictions but before they take on the full responsibilities of adult life. As young people begin to assume more adult roles—full-time employment, marriage, and parenthood—they often reduce their drinking. This reduction in alcohol use may be a result of the limitations that adult roles place on social activities or may reflect a change in young people's attitudes toward drinking. Young adults who drink in ways that are especially harmful—those who fit the diagnostic criteria for alcohol dependence—may have predisposing personality characteristics and other factors that place them at greater risk for problems with alcohol.

Prevention strategies that may be especially useful in curbing young adult alcohol use are those that focus on restricting the availability of alcohol. Such measures include raising the cost of alcohol through taxes, limiting when and where alcohol can be consumed, and enforcing policies that help to reduce problems such as drinking and driving.

High-risk alcohol use among college students is a major problem. There are many adverse effects with high levels of mortality and serious morbidity.

A number of factors contribute to high-risk alcohol use and alcohol-related adverse events. Some of these factors can be modified or changed. Others are more resistant to prevention efforts.

Individual factors that may play an important role in alcohol use and risk-taking behavior include a number of pre-college variables. These include

- Family history of alcoholism;
- Parental alcohol use;
- Age at first drink;
- Use of tobacco and marijuana in high school or middle school;
- Regular church attendance prior to college;
- Personality factors

- Untreated depression, anxiety, bipolar disorders and Post Traumatic Stress Disorder;
- Alcohol and drug use among peers and the student's home community.

Alcohol and the Elderly

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.

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 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.

Alcohol Sensitivity

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.

Alcohol and the Aging 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.

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.

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 are estimated at more than $119 billion.

Contributing Factors

Drinking rates vary among occupations, but alcohol-related problems are not characteristic of any specific 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.
- **Alcohol Availability -** The availability and accessibility of alcohol influences employee drinking.
- **Supervision -** Limited work supervision, often a problem on evening shifts, has been associated with employee alcohol problems.
- **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.

Managing Workplace Alcohol Problems

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 follow up after treatment.

Treatment

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.

Self-Help Programs

Self-help groups are the most commonly used treatment intervention 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.

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. 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 Intervention
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. 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.

Brief intervention for alcohol problems is more effective than no intervention and often as effective as more extensive intervention. The six key elements of brief intervention are summarized by the acronym FRAMES: feedback, responsibility, advice, menu of strategies, empathy, and self-efficacy. 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 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.

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

Effectiveness of Brief Intervention
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. 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.

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.

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

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.

Naltrexone

> **Mechanism:** Naltrexone blocks opioid receptors that are involved in the rewarding effects of drinking alcohol and the craving for alcohol. It's available in two forms: oral (Depade®, ReVia®), with once daily dosing, and extended-release injectable (Vivitrol®), given as once monthly injections.

Efficacy: Oral naltrexone reduces relapse to heavy drinking, defined as 4 or more drinks per day for women and 5 or more for men. It cuts the relapse risk during the first 3 months by about 36 percent (about 28 percent of patients taking naltrexone relapse versus about 43 percent of those taking a placebo). Thus, it is especially helpful for curbing consumption in patients who have drinking "slips." It is less effective in maintenance of abstinence.

Acamprosate

Mechanism: Acamprosate (Campral®) acts on the GABA and glutamate neurotransmitter systems and is thought to reduce symptoms of protracted abstinence such as insomnia, anxiety, restlessness, and dysphoria. It's available in oral form (three times daily dosing).

Efficacy: Acamprosate increases the proportion of dependent drinkers who maintain abstinence for several weeks to months.

Disulfiram

Mechanism: Disulfiram (Antabuse®) interferes with degradation of alcohol, resulting in accumulation of acetaldehyde which, in turn, produces a very unpleasant reaction including flushing, nausea, and heart palpitations if the patient drinks alcohol. It's available in oral form (once daily dosing).

Efficacy: The utility and effectiveness of Disulfiram are considered limited because compliance is generally poor when patients are given it to take at their own discretion. It is most effective when given in a monitored fashion, such as in a clinic or by a spouse. Some patients will respond to self-administered disulfiram, however, especially if they're highly motivated to abstain. Others may use it episodically for high-risk situations, such as social occasions where alcohol is present.

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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
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
Internet address: http://www.niaaa.nih.gov

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Alcoholism

Post-Test

1. A standard drink contains about _____ of pure alcohol. (p.4)
   A. 14 grams
   B. 1.2 fluid ounces
   C. 0.6 tablespoons
   D. 7.8 milliliters

2. Chronic heavy drinking may transform acetaminophen (Tylenol™) into chemicals that can cause liver damage. (p. 6) A. True B. False

3. Young married women have the greatest decreases in drinking behavior. (p. 9) A. True B. False

4. The most prevalent alcohol-associated brain impairments affect _____. (p. 11)
   A. fine motor coordination
   B. visuospatial abilities and higher cognitive functioning
   C. long-term memory
   D. intelligence and creativity

5. An individual experiencing an alcohol induced blackout typically is unable to _____. (p 13-14)
   A. drive a car
   B. recall events that occurred while they were intoxicated
   C. transfer information from their long-term memory to their short-term memory
   D. respond to noxious stimuli

6. Structural imagery reveals that alcoholics experience loss of volume and density in which two regions of the brain? (p. 15)
   A. Temporal lobes and neocortex
   B. Occipital lobes and substantia nigra
   C. Frontal lobes and cerebellum
   D. Parietal lobes and corpus collosum

7. Which alcoholism screening instrument consists of 5 questions and is used to detect a range of drinking levels from moderate to high risk? (p. 16-18)
   A. CAGE Questionnaire
   B. The Alcohol Use Disorders Identification Test (AUDIT)
   C. TWEAK Questionnaire
   D. Rapid Alcohol Problem Screen (RAPS4)

8. Approximately 10 to 20 percent of heavy drinkers develop cirrhosis. (p. 19) A. True B. False

9. Alcohol increases the sedative effect of tricyclic antidepressants (p. 23) A. True B. False

10. A patient who combines alcohol with nitroglycerin may be affected in which of the following ways while performing therapy related activities? (p. 24)
   A. Hyperactivity
   B. Sedation / sluggishness
   C. Hot flashes
   D. Dizziness or light-headedness

11. Alcohol inhibits fat absorption and impairs absorption of vitamins A, E, and D. (p. 27)
A. True B. False

12. Chronic alcohol abuse can reduce the body's responsiveness to insulin and cause glucose intolerance. (p. 29) A. True B. False

13. A strong association exists between alcohol use and cancers of the esophagus, pharynx, and mouth. (p. 32) A. True B. False

14. Alcohol consumption decreases liver cytokine levels. (p. 35) A. True B. False

15. Women achieve lower concentrations of alcohol in the blood after drinking equivalent amounts of alcohol. (p. 39) A. True B. False

16. People typically drink the heaviest in their late teens and early to mid-twenties. (p. 40) A. True B. False

17. Geriatric patients who abuse alcohol have an increased incidence of _____, and should therefore be monitored carefully by the clinician. (p. 41-42)

A. cardiac arrest
B. dementia
C. falls and fractured hips
D. vision problems

18. Employee drinking has been associated with low job autonomy, lack of job complexity, and lack of control over work conditions. (p. 43) A. True B. False

19. The most commonly used treatment intervention for alcohol-related problems is _____. (p. 44-47)

A. self-help groups
B. motivational enhancement therapy
C. behavioral marital therapy
D. brief intervention

20. Which of the following is NOT a medication commonly used to treat alcoholism? (p. 47-48)

A. Depade
B. ReVitrol
C. Campral
D. Antabuse

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