

Cognitive Impairment and Recovery From Alcoholism

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 (i.e., mental activities that involve acquiring, storing, retrieving, and using information) (1). 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 (2). 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 (3).

It stands to reason that cognitive impairment also may impede recovery from alcoholism, although evidence has not conclusively shown this to be the case. For example, Morgenstern and Bates (4) studied whether deficits in a patient's learning and planning abilities-core aspects of many treatment strategies-affected recovery from alcoholism. They found that impairment was not a significant predictor of poor treatment response. On the other hand, evidence does support the possibility that brain damage, whether resulting from or predating alcohol use, may contribute to the development and progression of alcoholism (5).

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. This issue of *Alcohol Alert* describes the nature and consequences of common alcohol-associated cognitive defects, explores the extent to which some cognitive abilities recover with abstinence, and summarizes recent research on the effects of cognitive deficits on alcoholism treatment outcome.

Cognition and Alcohol

Most alcoholics exhibit mild-to-moderate deficiencies in intellectual functioning (6), along with diminished brain size and regional changes in brain-cell activity. The most prevalent alcohol-associated brain impairments affect visuospatial abilities and higher cognitive functioning (7). 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 (2).

Most alcoholics entering treatment perform as well as nonalcoholics on tests of overall intelligence. However, alcoholics perform poorly on neuropsychological tests that measure *specific* cognitive abilities (8). 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.

How Much Is Too Much?

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 (6,9). Studies suggest that social drinkers who consume more than 21 drinks per week also fit into this category (6). Other investigators have suggested that cognitive deficits may be detectable only in those alcoholics who have been drinking regularly for 10 years or more (8,10). 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 (8). Although further research is needed to determine how a person's pattern of drinking is related to cognitive impairment, some deficits are possible even in people who are not heavy drinkers.

Tracking Structural and Functional Brain Abnormalities

Structural and functional brain abnormalities generally are measured by noninvasive imaging techniques that provide a picture of the living brain with minimal risk to the individual. Structural imaging techniques, such as computed tomography and magnetic resonance imaging, are used to generate computerized pictures of living tissue. Functional imaging techniques, such as positron emission tomography and magnetic resonance spectroscopy, permit scientists to study cell activity by tracking blood flow and energy metabolism. For more information about imaging, see *Alcohol Alert* No. 47, "Imaging and Alcoholism: A Window on the Brain."

Structural imaging consistently reveals that compared with nonalcoholics, most alcoholics' brains are smaller and less dense (11,12). Loss of brain volume is most noticeable in two areas: the outer layer (i.e., the cortex) of the frontal lobe, which is considered a major center of higher mental functions (7,12,13); and the cerebellum, which is responsible largely for gait and balance as well as certain aspects of learning (14). Support for these results is provided by functional imaging studies, which reveal altered brain activity throughout the cortex and cerebellum of heavy drinkers (15). 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 (15).

Understanding the Basis of Cognitive Impairment

Accurate measurement of cognitive abilities is challenging, and relating those abilities to a specific brain irregularity simply may not be possible with the current technology (16). Discrepancies among research findings have led scientists to develop improved cognitive-measuring techniques. Using a battery of tests, Beatty and colleagues (9) have suggested that widespread (i.e., diffuse) cognitive impairment could arise from damage to multiple brain areas, each of which regulates distinct but related abilities. Likewise, damaging the network of brain cells that synchronizes the overall activity of those multiple areas may produce the same cognitive impairments previously attributed to localized damage (9).

Is Impairment Reversible?

Certain alcohol-related cognitive impairment is reversible with abstinence (17). Newly detoxified adult alcoholics often exhibit mild yet significant deficits in some cognitive abilities, especially problem-solving, short-term memory, and visuospatial abilities (18). By remaining abstinent, however, the recovering alcoholic will continue to recover brain function over a period of several months to 1 year (19)-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 (18).

Rewiring Brain Networks

Reversibility of alcohol-related cognitive function also may be the result of a reorganization of key brain-cell networks. Some researchers have proposed that such reorganization may contribute to the success of alcoholism treatment. Using advanced imaging techniques, Pfefferbaum and colleagues (20) 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 (4). 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 (21).

Cognitive Function and Alcoholism Treatment

The exact role that cognitive function has in alcoholism treatment success is unclear. Structural and functional imaging,

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

- Cognitive deficits have been hypothesized to affect the efficacy of alcoholism treatment, although a clear association
 has not been established. One view finds that cognitively impaired patients may not be able to comprehend the
 information imparted during therapy and, thus, may not make full use of the strategies presented, thereby hampering
 recovery. Another view is that cognitive functioning may not directly influence treatment outcome, but may impact other
 factors that, in turn, contribute to treatment success (22). 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 (22,23). 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 (4). 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.

Cognitive Impairment and Recovery From Alcoholism-A Commentary by NIAAA Director Enoch Gordis, M.D.

The new noninvasive imaging techniques that allow us to "see" how the brain operates have been a boon to the study of cognition. Through this medium, we now know that the brain is capable of "rewiring" itself. In doing so, the brain can regain some of the cognitive abilities previously diminished as a result of damage from alcohol or other diseases. The brain's remarkable ability to recover is important for at least two reasons. First, alcohol use over a period of time, even at low levels of drinking, can produce varying degrees of cognitive damage, a problem that is of particular concern because alcohol use is so widespread. Thus, the brain's self-repairing ability may help defer or reduce alcohol-induced cognitive problems among a large portion of the population. Second, the brain's ability to rewire itself may have implications in terms of adolescent drinking. Recent evidence suggests that the adolescent brain, which is still forming important cellular connections, is more vulnerable than the adult brain to alcohol-induced damage. This is particularly troubling, given the problems associated with chronic binge drinking, which is all too common among young people. The brain's ability to rewire important neurological systems might help mitigate a lifetime of cognitive difficulties resulting from chronic drinking during adolescence, but we do not yet know if this is true. Future research will help clarify this and other important questions about alcohol's effect on cognition.

References

(1) Evert, D.L., and Oscar-Berman, M. Alcohol-related cognitive impairments: An overview of how alcoholism may affect the workings of the brain. Alcohol Health Res World 19(2):89-96, 1995. (2) Giancola, P.R., and Moss, H.B. Executive cognitive functioning in alcohol use disorders. In: Galanter, M., ed. Recent Developments in Alcoholism: Volume 14. The Consequences of Alcoholism.New York: Plenum Press, 1998. pp. 227-251. (3) Oscar-Berman, M. Severe brain dysfunction: Alcoholic Korsakoff's syndrome. Alcohol Health Res World 14(2):120-129, 1990. (4) Morgenstern, J., and Bates, M.E. Effects of executive function impairment on change processes and substance use outcomes in 12-step treatment. J Stud Alcohol 60(6)846-855, 1999. (5) Bowden, S.C.; Crews, F.T.; Bates, M.E.; et al. Neurotoxicity and neurocognitive impairments with alcohol and drug-use disorders: Potential roles in addiction and recovery. Alcohol Clin Exp Res 25(2):317-321, 2001. (6) Parsons, O.A. Neurocognitive deficits in alcoholics and social drinkers: A continuum? Alcohol Clin Exp Res 22(4):954-961, 1998. (7) Oscar-Berman, M.; Shagrin, B.; Evert, D.L.; and Epstein, C. Impairments of brain and behavior: The neurological effects of alcohol. Alcohol Health Res World 21(1):65-75, 1997. (8) Parsons, O.A., and Nixon, S.J. Cognitive functioning in sober social drinkers: A review of the research since 1986. J Stud Alcohol 59(2):180-190, 1998. (9) Beatty, W.W.; Tivis, R.; Stott, H.D; Nixon, S.J.; and Parsons, O.A. Neuropsychological deficits in sober alcoholics: Influences of chronicity and recent alcohol consumption.

Alcohol Clin Exp Res 24(2):149-154, 2000. (10) Eckardt, M.J.; File, S.E.; Gessa, G.L.; et al. Effects of moderate alcohol consumption on the central nervous system. Alcohol Clin Exp Res 22(5):998-1040, 1998. (11) Pfefferbaum, A.; Rosenbloom, M.; Crusan, K.; and Jernigan, T.L. Brain CT changes in alcoholics: Effects of age and alcohol consumption. Alcohol Clin Exp Res 12(1):81-87, 1988. (12) Pfefferbaum, A.; Lim, K.O.; Zipursky, R.B.; et al. Brain gray and white matter volume loss accelerates with aging in chronic alcoholics: A quantitative MRI study. Alcohol Clin Exp Res 16(6):1078-1089, 1992. (13) Lyvers, M. "Loss of control" in alcoholism and drug addiction: A neuroscientific interpretation. Exp Clin Psychopharmacol8(2):225-249, 2000. (14) Sullivan, E.V.; Rosenbloom, M.J.; Deshmukh, A.; et al. Alcohol and the cerebellum: Effects on balance, motor coordination, and cognition. Alcohol Health Res World 19(2):138-141, 1995. (15) Eberling, J.L., and Jagust, W.J. Imaging studies of aging, neurodegenerative disease, and alcoholism. Alcohol Health Res World 19(4):279-286, 1995. (16) Parsons, O.A. Determinants of cognitive deficits in alcoholics: The search continues. Clin Neuropsychologist 8(1):39-58, 1994. (17) Volkow, N.; Wang, G.J.; and Doria, J.J. Monitoring the brain's response to alcohol with positron emission tomography. Alcohol Health Res World 19(4):296-299, 1995. (18) Sullivan, E.V.; Rosenbloom, M.J.; Lim, K.O.; and Pfefferbaum, A. Longitudinal changes in cognition, gait, and balance in abstinent and relapsed alcoholic men: Relationships to changes in brain structure. Neuropsychology 14(2):178-188, 2000a. (19) Sullivan, E.V.; Rosenbloom, M.J.; and Pfefferbaum, A. Pattern of motor and cognitive deficits in detoxified alcoholic men. Alcohol Clin Exp Res 24(5):611-621, 2000 b. (20) Pfefferbaum, A.; Desmond, J.E.; Galloway, C.; et al. Reorganization of frontal systems used by alcoholics for spatial working memory: An fMRI study. NeuroImage 13:1-14, 2001. (21) Spear, L. Modeling adolescent development and alcohol use in animals. Alcohol Res Health 24(2):115-123, 2000. (22) Allen, D.N.; Goldstein, G.; and Seaton, B.E. Cognitive rehabilitation of chronic alcohol abusers. Neuropsych Review 7(1):21-39, 1997. (23) McCrady, B.S., and Smith, D.E. Implications of cognitive impairment for the treatment of alcoholism. Alcohol Clin Exp Res 10(2):145-149, 1986.

All material contained in the *Alcohol Alert* is in the public domain and may be used or reproduced without permission from NIAAA. Citation of the source is appreciated.

Copies of the Alcohol Alert are available free of charge from the National Institute on Alcohol Abuse and Alcoholism Publications Distribution Center P.O. Box 10686, Rockville, MD 20849-0686.

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES

Public Health Service * National Institutes of Health

September 2001

[NIAAA Home Page | Site Map | Accessibility | Privacy statement | Disclaimer | Contact Us]

National Institute on Alcohol Abuse and Alcoholism (NIAAA) 5635 Fishers Lane, MSC 9304 Bethesda, MD 20892-9304 email: Web Sponsor (<u>niaaaweb-r@exchange.nih.gov</u>)



National Institutes of Health

Department of Health and Human Services

