The International Journal of Indian Psychology
ISSN 2348-5396 (e) | ISSN: 2349-3429 (p)
Volume 6, Issue 3,DIP: 18.01.022/20180603
DOI: 10.25215/0603.022
http://www.ijip.in | July-September, 2018

**Review**

# Alcohol and Cognitive Impairment – A Review

Dr. Prosenjit Ghosh[1]*

## ABSTRACT

***Background***-Alcoholism results from an interplay between genetic and environmental factors, and is linked to brain defects and associated cognitive, emotional, and behavioral impairments. ***Objectives***-To find the impact and factors related to alcohol induced cognitive dysfunction. ***Method***- Internet search was carried out with alcohol, cognitive functions etc ***Results***-It was found that the effects of alcohol depend on various factors like – age, gender, general health and family history. Various structural, functional neuroimaging and neuropsychological tests have shown that a latge proportion of alcoholics suffer from cognitive dysfunction. ***Conclusion***-The underpinnings of alcohol-induced brain defects are multivariate; to date, the available literature does not support the assertion that any one variable can consistently and completely account for these impairments.

***Keywords:*** *Alcohol, Cognitive impairment, Executive function, Memory, Neurobehavioral deficits, Prefrontal cortex.*

Alcoholism results from interplay between genetic and environmental factors, and is linked to brain defects and associated cognitive, emotional, and behavioral impairments. A confluence of findings from neuroimaging, physiological, neuropathological, and neuropsychological studies of alcoholics indicate that the frontal lobes, limbic system, and cerebellum are particularly vulnerable to damage and dysfunction.

Alcohol abuse, as described by the American Psychiatric Association, is a psychiatric condition whereby alcoholic beverages are consumed despite negative consequences for health, well being, and interpersonal relationships. Alcohol dependence has additional physiological consequences such as increased tolerance for alcohol consumed, and withdrawal symptoms upon cessation of drinking.

Alcoholism has devastating consequences, but not all alcoholics are equally at risk for brain changes and neurobehavioral deficits. However, most problem drinkers have mild

[1]Assistant Professor, Silchar Medical College, Beside Indian Post, Ghungoor Road, Masimpur, Uttar Krishnapur Pt III, Assam, India
*Responding Author

**Received: June 14, 2018; Revision Received: July 7, 2018; Accepted: July 30, 2018**

neuropsychological difficulties, which improve within a year of abstinence. It is clear, however, that the locus and extent of brain damage, as well as the type and degree of impairment, differ across individuals. Such differences suggest that certain factors increase the likelihood of developing cognitive impairments with alcohol misuse. Among the important factors that must be considered are demographic variables (e.g., age, gender, socioeconomic background, and education), genetics and family history of alcoholism, alcohol use patterns (e.g., the age of onset of alcohol consumption, the type and amount of alcohol consumed, severity and duration of the dependency, duration of abstinence, nutritional status during periods of consumption), and the use or abuse of other psychoactive substances and nicotine.

## CONTRIBUTING FACTORS

**Age** - Normal chronological aging is associated with a number of physiological changes suggesting increased sensitivity to alcohol. Aging interferes with the body's ability to metabolize alcohol. Neuroanatomical changes seen in aging are similar to those associated with chronic alcoholism. In both, cerebral atrophy is most prominent in the frontal lobes. Other effects include greater than normal ventricular enlargement and widening of the cerebral sulci of alcoholics in relation to increasing age. Given the observed morphological similarities in the brains of alcoholic and aging nonalcoholic individuals, researchers sought to characterize parallels in functional decline associated with alcoholism and aging[9], and some investigators proposed that alcoholism is associated with premature aging. Taken together, most of the evidence from neuropathological and neuroimaging investigations supports the increased vulnerability model of premature aging. That is, certain brain structures show greater reduction in size (or blood flow) in older alcoholics than in younger alcoholics.

**Gender** - In the past decade, there has been an increasing interest in alcoholism-related gender differences with respect to possible changes in brain and behavior. However, the degree to which men and women differ with respect to these changes remains controversial. For example, in a recent cross-sectional, population-based study in which gender differences in cognitive performance were explored in relation to alcohol consumption, drinking data were collected from men and women between 35 and 85 years of age, and the participants were classified into non, light, moderate, and heavy drinking subgroups. There were clear gender differences in episodic memory (favoring women) and visuospatial tasks (favoring men). When these gender differences were examined by drinking group, visuospatial performance favoring men disappeared for the moderate to heavy drinking groups, but higher performance by women on episodic memory tasks was consistent across all levels of alcohol consumption. The results suggested that moderate alcohol intake may be less detrimental to women than to men.

Neuroimaging studies that measured gender differences in alcoholics' brain functioning have yielded contradictory evidence, with some studies showing women to be more susceptible than men to brain impairments and other studies showing no such distinction. Using

functional magnetic resonance imaging (fMRI), found decreased activity in parietal and frontal cortex, particularly in the right hemisphere, in alcohol-dependent women during performance of a spatial working memory task. Other studies, however, did not find functional differences based on gender, or even found that alcohol intoxication decreased brain metabolism in men more than in women as measured with positron emission tomography. Using structural MRI, Kroft et al found that the average ventricular volume in alcoholic women was within the typical range found in MRI studies of nonalcoholic women of similar ages. Hommer et al. reported clear gender differences in the brain structure of alcoholics. In that study, alcoholic men and women had smaller volumes of gray and white matter, as well as greater volumes of sulcal and ventricular CSF, than nonalcoholic men and women, but these differences were largest for the women. Clearly, many questions remain concerning the nature and extent of gender differences in the effects of alcoholism on brain and behavior. These potential differences deserve close scrutiny in the context of other variables such as age, drinking history, perceived social sanctions for drinking, impulsivity, genetic risk factors, etc.

**Health** - Medical conditions concomitant with alcoholism that are most likely to influence neurobiological and neurobehavioral functioning include liver disease, cardiovascular disease, and malnutrition. Thus, poor liver function and hypertension have been associated with drinking outside of meals. Thiamine deficiency, a consequence of poor diet, can contribute to Alcohol-Induced Persisting Amnestic Disorder (Korsakoff's syndrome), a severe disorder characterized by permanent cognitive and emotional deficits . Other common neurological conditions in alcoholics are head injury, encephalopathy, and fetal alcohol syndrome (or fetal alcohol effects), all of which can have an impact on neurobehavioral outcome. Various findings emphasize that consideration must be given to the potential influence of a host of psychiatric and medical conditions on neurocognitive functioning in studies of alcoholism as the primary condition. However, it is difficult to quantify the significance of comorbid conditions, whether they are secondary or tertiary illnesses, into a single, predictive variable that measures neurobehavioral or neurobiological outcomes in alcoholism. Instead, comorbid conditions deserve independent consideration, in addition to examining multivariate effects and interactions.

**Family history** - Results of twin, family, and adoption studies have shown that hereditary factors influence vulnerability to alcoholism. Additionally, the pharmacogenomics of alcohol response is well established, and genetic variants for the principal enzymes of alcohol metabolism are thought to influence drinking behavior and protect against alcoholism. Convergent evidence supports the view that vulnerability to alcoholism is likely to be due to multiple interacting genetic loci of small to modest effects.

Collaborative Studies on Genetics of Alcoholism (COGA) investigators have successfully recruited thousands of individuals from hundreds of extended families densely affected by alcoholism. The investigators have collected detailed and extensive clinical, neuropsychological, electrophysiological, biochemical, and genetic data. Evidence from these

studies has led to the identification of chromosomal regions containing genes that influence alcoholism risk and related phenotypes. Subsequently, single nucleotide polymorphisms (SNPs) have been genotyped in positional candidate genes located within the linked chromosomal regions, and analyzed for association with these phenotypes. Using this sequential approach, COGA investigators have reliably detected and identified associations with specific genes contributing to the risk for alcoholism.

**Acute Effects of Ethanol Ingestion -** Alcohol's effects on the brain and behavior depend upon an individual's blood alcohol concentration (BAC). Low doses can have a stimulating effect, and higher levels can have depressant effects. Impairments in mental functions such as attention and vigilance can be detected at BAC levels much lower than the legal intoxication levels, such as 0.02–0.03% .Alcohol intoxication disrupts neurophysiological indices of stimulus processing in attentional, semantic, and psychomotor domains. Furthermore, consistent with the evidence obtained from chronic alcoholics, acute intoxication results in a disproportionate impairment of executive functions such as planning, working memory, or complex behavioral control.

It is a common belief that alcohol ingestion leads to aggression and reduced impulse control, and there is high association of alcohol intoxication with violent crimes. Results of laboratory research have shown that alcohol intoxication increases the likelihood of aggressive behaviors. Some evidence suggests that alcohol may have disinhibitory effects on behavior. Alcohol induced disinhibition is also reflected in premature motor preparation based on incomplete stimulus evaluation. The disinhibitory effects may reflect a disruption in the inhibitory control of behavior subserved by prefrontal regions. A cluster of traits termed "antisocial personality disorder" inclusive of impulsivity, hyperactivity, and sensation/novelty seeking correlates with the early-onset alcoholism, increased drinking, and chronic alcohol use and dependence and may reflect a disruption in the inhibitory control of behavior subserved by prefrontal regions. Recent models of vulnerability to alcoholism emphasize the importance of executive functions in mediating, as well as moderating the effects of alcohol.

## NEURAL SYSTEMS AFFECTED AND CONCOMITANT

### Neurobehavioral Deficits

Results of research employing a variety of different techniques have determined that the brain structures most vulnerable to the effects of alcoholism are the neocortex (especially the frontal lobes), the limbic system, and the cerebellum.

**Frontal lobe structure and function -** Although alcoholism related cortical changes have been documented throughout the brain, many studies consistently have found the frontal lobes to be more vulnerable to alcohol-related brain damage than other cerebral regions. Harper and his collaborators established that 15–23% of cortical neurons are selectively lost from the frontal association cortex following chronic alcohol consumption. Structural MRI studies have shown frontal lobe volume losses in alcoholic subjects, reduced regional blood

flow measurements, and with measurements of lower glucose metabolism throughout the brain (including prefrontal cortex) during alcohol intoxication. Frontal lobe blood flow and metabolism may decrease in alcoholics before significant shrinkage or major cognitive problems become detectable. The anterior region of the frontal lobes (prefrontal cortex) plays a kind of executive regulatory role within the brain. Executive functions (which depend upon many of our cognitive abilities, such as attention, perception, memory, and language) are defined differently by different theorists and researchers. Most agree, however, that executive functions are human qualities, including self-awareness, that allow us to be independent individuals with purpose and foresight about what we will do and how we behave.

Prefrontal neurobehavioral dysfunction has been frequently observed in alcoholics with and without the dense amnesia of Korsakoff's syndrome. In addition to causing changes in cognitive functions, damage to frontal brain systems often leads to aberrations of emotion and personality. Frontal personality traits have been described in terms of "disinhibition" and impulsivity, including aggression and a lack of concern for the consequences of untoward behaviors. Investigators have found that shared neurochemical markers may underlie the commonalities between alcohol abuse and traits associated with antisocial personality disorder. Thus, impulsive behavior may be a premorbid trait predisposing individuals to a spectrum of disorders including alcohol dependence.

**Limbic system** - The limbic system monitors internal homeostasis, mediates memory and learning, and contributes to emotional feelings and behaviors. The limbic system also drives important aspects of sexual behavior, motivation, and feeding behaviors.

**Amygdala** - The amygdala is a small almond-shaped structure, deep inside the anteroinferior region of the temporal lobe. A number of studies have linked the amygdala to the processing of motivational significance of stimuli and to the mediation and control of major emotions such as love, fear, rage, anxiety, and general negative affective states. The amygdala is partially controlled by the brain's dopamine system, as an essential part of the brain-reward circuitry—the same system that responds to alcohol and produces feelings of pleasure when good things happen. In a recent study using fMRI, observed clear evidence of differences between abstinent long-term alcoholics and nonalcoholic controls in amygdala activation to emotional materials. The subjects were scanned while viewing emotional words and emotional facial expressions. Faces with negative and positive emotional expressions evoked significantly stronger bilateral amygdala activity in the controls than in the alcoholics, whose activations were blunted. The observation that alcoholics respond to emotionally-valenced stimuli in an undifferentiated manner is consistent with clinical evidence of their interpersonal difficulties.

**Hippocampus** - As part of the limbic system, the hippocampus is intimately involved in motivation and emotion, and it also plays a central role in the formation of memories. Results of a nonhuman animal study have suggested that the deleterious effect of ethanol on the survival of newlyformed neurons in the adult rat hippocampus could result in impairment of

hippocampal-dependent cognitive functions. Neurogenesis declines until it ceases in the young adult mammalian brain, with two exceptions: The olfactory bulb and the hippocampus produce new neurons throughout adult life. The ethanol induced reductions in hippocampal neurogenesis can be attributed to two general mechanisms: an effect on cell proliferation or on cell survival. These changes in hippocampal structure could be part of the anatomical basis for cognitive deficits observed in alcoholism. Structural neuroimaging studies have demonstrated a reduction of hippocampal volume in alcoholics. The loss of hippocampal volume has been attributed to changes in white matter.

**Hypothalamus** - The hypothalamus literally means "under the thalamus." The hypothalamus is involved in learning and memory, as well as in regulatory functions such as eating and drinking, temperature control, hormone regulation, and emotion. Long-term alcoholism and concomitant dietary insufficiencies have been associated with damage to the mammillary bodies of the hypothalamus, and memory deficits (amnesia) often follow. When amnesia occurs as a consequence of chronic alcoholism, it is referred to as alcohol-induced persisting amnestic disorder or alcoholic Korsakoff's syndrome[21]. The specific memory impairments include severe anterograde amnesia for newly learned information, and some retrograde amnesia, i.e., loss of memory for events that happened long ago (prior to the appearance of obvious symptomatology). Because new events are forgotten a few seconds after they occur, virtually nothing new is learned, and patients with Korsakoff's syndrome live perpetually in the past. However, in contrast to patients with alcoholic dementia, who have generalized cognitive decline (including widespread memory loss), patients with Korsakoff's syndrome retain old memories formed prior to the onset of alcohol-related brain damage.

***Implications for Treatment and Recovery***

Clinicians must consider a variety of treatment methods to promote cessation of drinking, maintenance of sobriety, and recovery of impaired functioning. In a comprehensive review of the pharmacogenomics of alcohol response and addiction, Enoch noted that treatment is complicated by the comorbidity of alcoholism with other disorders (e.g., anxiety, depression, antisocial personality disorder, smoking, and other addictions).

First-line therapeutic targets for alcoholism are neurotransmitter pathway genes implicated in alcohol use. Of particular interest are the reward pathways (serotonin, dopamine, GABA, glutamate, and beta endorphin) and the behavioral stress response system (corticotrophin-releasing factor and neuropeptide Y). The current pharmacological therapies are only modestly effective in preventing relapse and dependence in alcoholics, prompting more research. Additionally, treating co-occurring disorders remains a challenge, and the use of creative approaches that would encompass individualized psychosocial support, as well as a combination of treatments, might be the most effective way to address this problem.

Neuroimaging research already has shown that abstinence of less than a month can result in an increase in cerebral metabolism, particularly in the frontal lobes, and that continued abstinence can lead to at least partial reversal in loss of brain structure and function.

## CONCLUSION

Alcoholics are a diverse group. They experience different subsets of symptoms, and the disease has different origins and modulating influences for different people. Therefore to understand the effects of alcoholism, it is important to consider the influence of a wide range of variables on a particular behavior or set of behaviors. The underpinnings of alcohol-induced brain defects are multivariate; to date, the available literature does not support the assertion that any one variable can consistently and completely account for these impairments. An integrative approach that recognizes the interconnectivity of the different functional systems to account for the heterogeneity of outcome variables associated with alcoholism-related impairments and recovery of functions will be ideal.

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***Acknowledgements***

The authors profoundly appreciate all the people who have successfully contributed in ensuring this paper is in place. Their contributions are acknowledged however their names cannot be able to be mentioned.

***Conflict of Interest***

The authors colorfully declare this paper to bear not conflict of interests

**How to cite this article:** Ghosh, P (2018). Alcohol and Cognitive Impairment – A Review. *International Journal of Indian Psychology*, *6*(3), 15-25. DIP:18.01.022/20180603, DOI:10.25215/0603.022