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Research Paper

Historical Aspects of Alcohol Abuse and Craving: An Overview

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ABSTRACT

Alcohol Abuse lead too many social and personal problems like; physical problem, mental health, disturbance in work life, poor family and social relationships, separation and divorce and emotional hardship in the family. Craving can also be seen as an aspect of a prolonged subclinical abstinence or withdrawal syndrome. The aim of the study has to provide information and explore towards alcohol abuse, its historical aspects and craving. Literature has been searched the both electronic databases including PubMed and manual searches for this. Neurobiological and brain-imaging studies have identified numerous brain chemicals and brain regions that may be involved in craving. A better understanding and more reliable assessment of craving may help clinicians tailor treatment to the specific needs of each patient, thereby reducing the risk of relapse.

Keywords: *Alcohol, Craving, Relapse, Neurobiology.*

Alcohol Abuse: Alcohol is a substance that has been shown acceptance for a degree so far in history similar to tobacco. It has been taken as an object of fun; consumed in social gatherings for thousands of years. Although the developed societies have started to support a negative look for both tobacco and alcohol dependence in the recent years, treatment facilities or government supported campaigns are lacking especially in terms of alcohol treatment. In Turkish culture, alcohol has its drinking customs. It has always been an object that gathers people in a place for an occasion [1]. There are rules of how to order, or how to prepare the side dishes of alcohol. For example, there are rules of how to consume an alcoholic beverage called "Rakı" in Turkey; it is consumed in group settings and rarely when the person is alone. In these groups, alcohol dependence is seen as fulfilling customs, or performing rituals that bound the group members together, therefore it is a social status symbol.

It is known that alcohol easily passes the blood-brain barrier and consequently influencing its functioning. If a person wants to slow down the effects of alcohol, there are several things to do. For example, drinking when the stomach is not empty and especially drinking it with high protein foods slows down the absorption speed. On the other hand, drinking alcohol mixed with carbonated drinks (e.g., coke, energy drinks) facilitates the absorption. Another important determinant of how alcohol affects the person is its concentration. The higher the

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concentration, the more rapidly it is absorbed from the stomach. Distilled liquors including vodka, whiskey, and gin have very highly concentrated alcohol levels (40-50 %). Body mass and gender are also effective in terms of speed of intoxication. The larger is the body, the slower it is intoxicated. Hence, women are intoxicated in a more rapid way; they usually weigh less than men, have higher percentages of body fat and lower levels of water in their body [2]. Neurobehavioral effects of alcohol can be summarized as intoxication of the metabolism, reinforcement to use again, aggression (without the significance of amount of alcohol intake), negative effects on learning and memory, brain damage and building up tolerance resulting in development of physical addiction.[3]

Alcohol abuse is defined as "A maladaptive pattern of substance use that occurs in the presence of physically hazardous situations, multiple legal problems, and recurrent social and interpersonal problems" [4]. Brachtesende states if the person who is alcohol dependent is not treated, health problems other than dependence may occur. Some of the diseases related with alcohol are diabetes, asthma, mental disorders like mood disorders, alcohol-related dementia [5]. College students have been the most extensively studied group among alcohol using populations because they were found to be inclined to become addicted. In a study, it was found that males were more likely to expect that alcohol would make dealing with stress and peer communications easier.[6]

Alcoholic beverages have been of use in human civilization. Indian people have also not been devoid of the use of alcoholic beverages since time immemorial. Scientifically, alcohol is an organic compound in which the hydroxyl functional group is bound to a carbon atom. In particular, this carbon centre is saturated, having single bonds to three other atoms. Since the primitive era, people have been using alcohol for myriad reasons, e.g. 'as a medium of enjoyment and recreation', 'a symbol associated with ethnic, racial sub-cultural practices', 'spiritual reasons', 'mode of gratification and toxic emotions (e.g. anxiety, worry and tension) buster', 'social gathering', and possible thousands of other individualized or non-individualized reasons. Historically, ardent supporters or consumers of alcoholic beverages have cited many benefits of alcohol for making it as a part and parcel of their lives. People vigorously talked about its nutritional, medicinal, antiseptic, and analgesic properties and how it can fetch boons for them. The role of such beverages as thirst quenchers is obvious and they play an important role in enhancing the enjoyment and quality of life. People reckon alcohol as a social lubricant or bonding factor, can facilitate relaxation, can provide pharmacological pleasure, and can increase the pleasure of eating. Thus, while alcohol has always been misused by a large section of people and many people as well as their kith and kin have ultimately fallen apart because of regular intake of this substance.

Historical aspects: The word 'alcohol' was introduced into the English language around 1543 and the origin of this word happened to be the Arabic word *"al-kuhul"*, meaning finely divided. Though, it is a difficult task to pinpoint when people started to prepare 'alcoholic beverages' and imbibed alcohol as an integral part of their lives. [] But some anecdotal information suggested that the preparation of alcoholic beverages on a routine basis was started in Ancient Egypt. Some arte facts have been found in the past which suggested that ancient Egyptians were very much proficient in the process of fermentation. Approximately 7000 years back, Chinese people also developed the skills to produce fermented liquors [7]. In India, an alcoholic beverage called *'Sura'*, distilled from rice, was in use between 3000 and 2000 B.C [8]. The Babylonians worshiped a wine goddess as early as 2700 B.C. In Greece, one of the first alcoholic beverages to gain popularity was mead, a fermented drink made from honey and water. Greek literature is full of warnings against excessive drinking.

Several Native American civilizations developed alcoholic beverages in Pre-Columbian times. A variety of fermented beverages from the Andes region of South America were created from corn, grapes or apples, called *"Chicha."* But some evidences which have relatively higher merits came from China and Transcaucasian Region of Europe. Around 8000 B.C., people took up agriculture as the regular source of livelihood and established sedentary communities in different parts of the world. The earliest concrete proof of making alcohol through crude means of fermentation was found from a place called 'Jiahu', in Northern China. From a graveyard of this place, some vessels and potteries were found and after chemical analysis, it was discovered that those vessels and potteries were used for brewing. These clay vessels, coincidentally the most ancient of their kind, contained a fermented drink made with rice, honey, grapes and hawthorn berries. Further evidence of prehistoric brewing comes from Transcaucasia, part of present day Georgia, where grape pips have been discovered around 'Neolithic Settlements' with shapes that differ slightly from those of wild grapes, suggesting that they had been cultivated.[9]

There are many forms of alcohol, in which ethanol is most frequently used. It is a colourless, volatile liquid with a mild odour. Ethanol's chemical formula was determined by Swiss chemist Nicolas-Theodore de Saussure in 1808 and was later published by Scottish chemist Archibald Scott Couper in 1858. It is obtained through the fermentation or distillation of sugars; the first examples were the use of honey fermented with water to create mead and the natural fermentation of grapes to wine. Indeed, ancient Greeks have written of alcohol in antiquity, Dionysus, the God of wine and pleasure, was first a "honey-lord", and then a God worshipped for the cultivation of the vine. Although used as a "stimulant", it is known to be the most potent and widely used central nervous system depressant in the western world; it is also the second most used drug in the world, topped only by caffeine.[10]

Craving: Craving was first elaborated by Jellinek and colleagues [11]. It can be described an irresistible need for alcohol intake that is considered to play a major role in the development and maintenance of dependent behavior.[12] Craving has been related to multiple dimensions including biological, psychological, neuro-adaptive and environmental domains. In the psychological domain, most of the literature has focused on cognitive behavioural aspects of craving acknowledging that personality traits may also play an important role in explaining individual differences in craving.Craving has been defined as a desire to experience the effects of a previously experienced psychoactive substance [13]. In short, craving is an irresistible urge to use a substance that compels drug seeking behavior[14]

Alcohol abuse and Craving

Ludwig[15] one of the pioneers in craving research, suggested that craving was a label affixed by patients to some combination of internal and external dysphoric or discontented feelings. Maisto and Schefft reviewed two theoretical constructs regarding alcoholism as a disease- one involving loss of control over alcohol, and the other involving overwhelming craving for alcohol. According to them, craving is the desire to use a drug and previous memories of pleasure superimposed upon a negative emotional state [16].

Kozlowski and Wilkinson (1987) argued that while craving existed, the problems related to defining it came from mixing the traditional layman's definition of craving with technical or scientific definitions predicted on specific hypotheses [17]. Scientists have taken craving variously to be representative of physical dependence, or to be the result of physical

symptoms, or to be one of several withdrawal symptoms, or to indicate a subset of cognitive processes about drug use, but have always connected the observed phenomenon of craving to an interpreted explanation. Instead, Kozlowski and Wilkinson suggested that "craving" should be used only in a descriptive way, indicating a strong desire or urge to use the substance. Childress et al.(1987) documented the association between negative mood states and craving and withdrawal symptoms and recommended that treatment should address both internal and external conditioned stimuli. They suggested that extinction procedures would be useful as a treatment technique [18]. O'Brien and Childress (1990) demonstrated that there were strong signs of physiological arousal associated with cue-exposure in addition to the subjective feelings of craving [19]. Russian scientists like Nemtsov demonstrated changes in skin conductance response with utterance of words like 'Vodka' or 'Beer'. [20]. Marlatt (1990) suggested that craving was a component of expectation and anticipation, a psychological rather than a physiological phenomenon. He also addressed the importance of cue-exposure and stressed on extinction of the pleasurable expectations associated with alcohol. Several investigators in 80's and 90's talked about 'Priming'- a feed-forward effect which causes a substantial increase in the drive to use the substance [21]. Ludwig et al.(1986) suggested that at low doses, alcohol has a priming effect, known as "First Drink" phenomenon [22] and priming proved to be instrumental in increasing the subjective phenomenon of craving [23]. An association between "kindling" and craving has also been postulated based on neuronal super sensitivity on animal models. Craving can also be seen as an aspect of a prolonged subclinical abstinence or withdrawal syndrome [24]. According to Modell et al (1992) craving shares specific features in common with the obsessions of obsessive-compulsive disorder and that the existence of craving is dependent on the presence of obsessive thoughts about drinking. Positive correlations between craving and measures of compulsive drinking behavior also exists; compulsive drinking behavior, however, may reflect the consequences of craving rather than a fundamental characteristic of craving itself and despite difficulties in defining the term craving, it is clearly a phenomenon that is experienced or endorsed by most alcoholic subjects and is not experienced by most persons who do not abuse alcohol.[25]

MODELS OF ALCOHOL CRAVING

(a) Conditioning models

Conditioning models are based on principles of classical conditioning. Alcohol related cues (like sight of a bar or beer bottle etc.) after repeatedly being paired with alcohol consumption, become conditioned stimuli and they evoke the same physiological and psychological response as alcohol consumption itself and if not followed by alcohol consumption, results in craving, either to have the rewarding pleasant effect or to get rid of the aversive withdrawal. It was conceptualized that craving is entirely a conditioned response among alcohol dependent associated with environmental and intra-psychic cues relevant to drinking in the same way that fear is a conditioned response in phobias.[26]

(i) Conditioned incentive and appetitive model

Craving results from the desire to experience the positive or reinforcing effects associated with alcohol consumption. Alcohol associated cues further strengthen this reinforcement-seeking behavior. It is long known that craving for alcohol can be elicited by exposure to alcohol and alcohol-associated stimuli in abstinent alcoholics [27] and alcohol craving is induced by the mood-enhancing, positive-reinforcing effects of alcohol. [28]

(b) Conditioned tolerance model

Craving results from the desire to avoid the negative (i.e. aversive) experience resulting from physiological changes involved in conditioned tolerance (i.e. responses to counteract the effects of alcohol in the brain, which also occurs in the absence of alcohol consumption in situations that were previously associated with drinking).

(c) Conditioned withdrawal model

Craving results from the desire to avoid the aversive experience of conditioned withdrawal (i.e. withdrawal that is induced by stimuli that have been associated with previous withdrawal experiences). In the initial phases of drug and alcohol use, self-administration is maintained primarily by the positive reinforcing effects of the alcohol. As dependence develops, the negative aspects of withdrawal could play an increasingly important role in increasing the motivation for substance use [29].It was observed that alcohol-associated stimuli evoke conditioned physiological reactions. Such conditioned compensatory responses help to maintain a homeostatic balance during alcohol intake and may manifest as conditioned withdrawal if alcohol-associated cues are presented but not followed by actual intake. [30]

(d) Auto shaping model

Craving reflects the drinker's monitoring of internal and external stimuli that in the past have been reliably associated with alcohol consumption.

(e) Incentive sensitization model

Craving is defined as a conscious experience that occurs when the drinker pays excessive attention to alcohol related stimuli or considers those stimuli to be excessively attractive; the attention to or perceived attractiveness of alcohol-related stimuli increases with repeated alcohol exposure (i.e. sensitization occurs). Associative learning may transform positive mood states and previously neutral environmental stimuli into alcohol associated cues that acquire positive motivational salience and induce reward craving.[31]

(b) Cognitive models

The assumption behind cognitive models is that responses to alcohol and alcohol-related cues involve various cognitive processes such as expectations regarding the pleasant effects of alcohol and a person's belief in his or her own ability to cope with the desire to drink.[32]

(i) Cognitive-behavioral model

Craving is considered as a subjective state mediated by the expectation that drinking will improve an existing negative mood state i.e. have a positive effect and craving occurs when drinker's confidence regarding the ability to resist alcohol is lacking. It was suggested that craving for alcohol really represented a cognitive correlate for an alcohol withdrawal syndrome that was subclinical in nature and it summated the internal and external cues and presented the addict with a potential source of relief, i.e., alcohol. Craving and urges are considered non-automatic cognitive processes that result when plans for drinking are blocked or impeded; craving is not required for either drinking or relapse to occur[33]

(ii) Neurocognitive model

Craving influences and is influenced by other cognitive processes and during craving, the brain regions associated with affect and memory are activated.

(c) Neuro-adaptive model of Craving

A gradual and perhaps permanent adaptation of brain function or neuro-adaptation to the presence of alcohol is a central feature in the development of alcohol dependence. Chronic alcohol use produces numerous neurobiological changes in several brain regions, including brain regions involved in regulating addictive behavior. These changes occur at virtually every level of information processing, ranging from neurotransmitters and receptors to intracellular signaling pathways and regulation of gene expression to long-term structural changes that alter synaptic plasticity. This neuro-adaptation, or sensitization, leads to certain characteristics of alcohol dependence, such as tolerance and withdrawal as well as to a condition that might be called reward memory, a memory that has evolved from amygdala. Neuro-adaptations in the mesolimbic dopamine system, especially the nucleusaccumbens (NAc), are commonly found with chronic exposure to alcohol and this reflects the ability to activate a common reinforcement substrate and to produce similar symptoms characteristic of dependence. Neuro-adaptationoccurs to a greater extent and more permanently in people who are at increased risk for developing alcoholism, either because of inherited genetic predisposition or being exposed to severe stress. Neuro-adaptation in alcohol craving involves following aspects:

(i) Adaptations in nucleus accumbens neurotransmitter and receptor systems.

Repeated exposure to alcohol produces progressively larger psychomotor responses to a fixed challenge dose. Sensitization involves the development of hypersensitivity in the mesolimbic dopamine system to drugs, stress, and drug associated stimuli. Hypersensitivity in the dopamine system is related to greater dopamine release from dopamine nerve terminals in the nucleus accumbens. Chronic alcohol use also produces alterations in glutamatergic input to the mesolimbic dopamine system.

(ii) Adaptations in dopamine receptor signaling.

The significant role of dopamine in substance dependence involves receptor regulation of intracellular second messenger responses, such as cAMP formation. D1 and D2 receptors are coupled to the cAMP second messenger system but in opposite ways. D1 receptors are coupled to stimulatory Gproteins (Gs and Golf) that stimulate cAMP formation by activating adenylate cyclase. In contrast, D2 receptors are coupled to inhibitory G proteins (Gi and Go) that inhibit adenylate cyclase activity and cAMP formation. Opposite neuroadaptations in D1 and D2 responsiveness could produce tolerance to the rewarding effects of drugs and alcohol while simultaneously enhancing the incentive to seek them.

(iii) Adaptations in Gene Expression

Sustained or repeated activation of cyclic adenosine monophosphate (cAMP) response element binding protein (CREB) gene with chronic alcohol use also could contribute to the development of tolerance and craving

(iv) Neurobiological model of craving

It correlates the neuroanatomical and neurochemical substrates of craving. Reward system consists of dopaminergic neurons in the A9 and A10regions of the ventral tegmental area (VTA) and their axon projections to forebrain regions, such as the nucleus accumbens (NAc) and the prefrontalcortex (PfC). Alcohol-associated cues can trigger appetitive or approach behavior and can activate dopamine neurons in the VTA, thus the mesolimbic dopamine system plays a major role in cue-induced craving ·Nucleus accumbens has connection with amygdala and frontal cortex. Amygdala, an integral part of limbic system, plays a role in modulation of stress and mood. The amygdala is involved in the conditioning processes for appetitive and aversive stimuli and also is necessary for recalling memories evoked by cues related to substance use. Descending neuronal projections from the amygdale activate VTA dopamine neurons

through monosynaptic and polysynaptic pathways, leading to increased dopamine levels in the NAc. Frontal cortex areas integrate incoming sensory information, such as sights, mells and sounds. Dorsolateral prefrontal cortex (DLPC) is the area where the memories for the positive reinforcing aspects of alcohol use and their salience is located and it maintains intimate connections with those parts of the brain which control emotion and reward, i.e. amygdala and nucleus accumbens[34].Studies on neurochemical basis of craving helps to explain the conditioning models. In alcoholism, conditioned withdrawal may be caused by an imbalance between central glutamatergic and aminobutyric acid (GABA)-ergic neurotransmission. Acute alcohol intake potentiates GABAergic sedation and inhibits glutamatergic excitation. [35,36] Chronic alcohol intake down-regulates GABAA receptors and thus ensures homeostatic regulation [37] .When the sedative effects of alcohol are suddenly withdrawn during detoxification, reduced GABAergic inhibition and increased glutamatergic excitatory neurotransmission may manifest as anxiety, seizures and autonomic dysregulation. Cues associated with prior alcohol intake that are not followed by actual alcohol consumption, stress exposure, or negative mood states may induce conditioned withdrawal and withdrawal relief craving. Alcohol consumption may then be reinstated to reduce withdrawal stress, thus acting as a negative reinforce [38]. Currently the role of serotonin is under research. Several studies report reduced serotonin (5HT) in alcohol-dependent subjects. Furthermore, alcohol increases 5HT in animals. Thus, craving may be an attempt to self-medicate reduced 5HT.[39, 40].In recent years, group therapy has been the primary form of treatment in structured in-patient and out-patient's addiction programs. Group provides opportunities to the individual with alcohol dependence for mutually identification and reduced feelings of isolation and shame; peer acceptance, support and role modelling for positive changes; acquisition of new coping skills; exchange of factual information; and instillation of optimism and hope. The gathering together of people who share a common problem often creates a common bond between them, stemming from a sense of belonging and an expectation of being intuitively understood.

Many researchers and clinicians consider craving an important contributor to the development and maintenance of alcoholism. Craving has been described as a powerful urge to drink or as intense thoughts about alcohol. The *International Classification of Diseases (ICD–10)* includes craving as an optional diagnostic criterion for addiction to alcohol or other drugs, defining the term as a strong desire or sense of compulsion to take the drug Understanding the exact nature of craving has been difficult. Nevertheless, scientists have accumulated a large amount of data on its mechanisms and manifestations [41]

CONCLUSION

It can be said that numerous models of the mechanisms underlying craving have been suggested, however. One of those model "the neuroadaptive model "suggests that the prolonged presence of alcohol induces changes in brain-cell function. In the absence of alcohol, those changes cause an imbalance in brain activity that results in craving. Furthermore, the adaptive changes generate memories of alcohol's pleasant effects that can be activated when alcohol-related environmental stimuli are encountered, even after prolonged abstinence, thereby leading to relapse. Similarly, stressful situations may trigger memories of the relief afforded by alcohol, which could also lead to relapse. Neurobiological and brain-imaging studies have identified numerous brain chemicals and brain regions that may be involved in craving. Psychiatric conditions that affect some of these brain regions, such as depression or anxiety, also may influence craving. A better

understanding and more reliable assessment of craving may help clinicians tailor treatment to the specific needs of each patient, thereby reducing the risk of relapse.

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