A study about the comparison between the alcoholic addiction recovery and relapse.

Introduction

Alcohol addiction is a chronic relapsing disorder associated with compulsive alcohol drinking, the loss of control over intake, and the emergence of a negative emotional state when alcohol is no longer available.

Effects and Causes of alcohol addiction.

Alcohol abuse can increase your risk for some cancers as well as severe, and potentially permanent, brain damage. It can lead to Wernicke-Korsakoff syndrome (WKS), which is marked by amnesia, extreme confusion and eyesight issues. WKS is a brain disorder caused by a thiamine deficiency or lack of vitamin B-1.

Scheme of Assistance for the Prevention of Alcoholism & Substance (Drugs) Abuse and for Social Defence Services

The Ministry of Social Justice and Empowerment, Department of Social Justice and Empowerment, came into existence on 15th October 2008 through the merger of two Central Sector Schemes: the 'Scheme for the Prevention of Alcoholism & Substance (Drugs) Abuse' and 'General Grant in Aid Programme for Financial Assistance in the Field of Social Defence.' The scheme's objectives include creating awareness about the ill effects of alcoholism and substance abuse, as well as providing a comprehensive range of community-based services for indemnification, motivation, counselling, de-addiction, aftercare, and rehabilitation for whole-person recovery (WPR) of addicts.

List Of Top 10 Rehabilitation Centers in India

Sno	Top Rehabilitation Centers	Founded	Official Website

1	**Sanctum Wellness**	2019	sanctumwellness.org
2	**Rehabs India**	2021	rehabsindia.in
3	**Luxury Rehab Finder**	2021	luxuryrehabfinder.com
4	**Belgharia Well Wisher Foundation**	2014	wellwisherfoundation.in
5	**Sukoon Health**	2020	sukoonhealth.com
6	**Hope Trust**	2002	hopetrustindia.com
7	**Lotus Wellness**	2021	lotuswellness.life
8	**Blissful Mind**	2015	theblissfulmind.com
9	**Jeevan Daan Treatment**	2007	jeevandaanfoundation.org

10	**Drug Dependence Treatment Centre**	1988	aiims.edu

Review of literature

The development of alcohol dependence is a complex and dynamic process. Many neurobiological and environmental factors influence motivation to drink (Grant 1995; Samson and Hodge 1996; Vengeliene et al. 2008; Weiss 2005). At any given time, an individual's propensity to imbibe is thought to reflect a balance between alcohol's positive reinforcing (i.e., rewarding) effects, such as euphoria and reduction of anxiety (i.e., anxiolysis), and the drug's aversive effects, which typically are associated with negative consequences of alcohol consumption (e.g., hangover or withdrawal symptoms). Memories associated with these rewarding and aversive qualities of alcohol, as well as learned associations between these internal states and related environmental stimuli or contexts, influence both the initiation and regulation of intake. These experiential factors, together with biological and environmental influences and social forces, are central to the formation of expectations about the

consequences of alcohol use. These expectations, in turn, shape an individual's decision about engaging in drinking behavior.

The nature of and extent to which these factors are operable in influencing decisions about drinking not only vary from one individual to another but also depend on the stage of addiction—that is, whether the drinker is at the stage of initial experience with alcohol, early problem drinking, or later excessive consumption associated with dependence. Although many people abuse alcohol without meeting the criteria for alcohol dependence,1 continued excessive alcohol consumption can lead to the development of dependence. Neuroadaptive changes that result from continued alcohol use and abuse (which manifest as tolerance and physiological dependence) are thought to be crucial in the transition from controlled alcohol use to more frequent and excessive, uncontrollable drinking (Koob and Le Moal 2008). Indeed, for some dependent individuals, the fear that withdrawal symptoms might emerge if they attempt to stop or significantly curtail drinking may prominently contribute to the perpetuation of alcohol use and abuse.

This article will provide an overview of the basic features of alcohol dependence and the associated withdrawal syndrome, emphasizing those components of withdrawal that especially are thought to contribute to the problem of relapse. It will present evidence from both clinical and experimental studies that highlights long-lasting physiological and emotional changes which are characteristic of dependence and have been postulated to play a key role in persistent vulnerability to relapse. In particular, it will review animal models of alcohol dependence and withdrawal, as well as models of self-administration, that have helped researchers elucidate brain mechanisms underlying relapse and excessive drinking associated with dependence.

Alcohol Withdrawal

When an alcohol-dependent individual abruptly terminates or substantially reduces his or her alcohol consumption, a characteristic withdrawal syndrome ensues. In general, alcohol acts to suppress central nervous system (CNS) activity, and, as with other CNS depressants, withdrawal symptoms associated with cessation of chronic alcohol use are opposite in nature to the effects of intoxication. Typical

clinical features of alcohol withdrawal include the following (Becker 2000; Hall and Zador 1997; Saitz 1998):

Signs of heightened autonomic nervous system2 activation, such as rapid heartbeat (i.e., tachycardia), elevated blood pressure, excessive sweating (i.e., diaphoresis), and shaking (i.e., tremor);

Excessive activity of the CNS (i.e., CNS hyperexcitability) that may culminate in motor seizures; and

Hallucinations and delirium tremens in the most severe form of withdrawal.

In addition to physical signs of withdrawal, a constellation of symptoms contributing to a state of distress and psychological discomfort constitute a significant component of the withdrawal syndrome (Anton and Becker 1995; Roelofs 1985; Schuckit et al. 1998). These symptoms include emotional changes such as irritability, agitation, anxiety, and dysphoria, as well as sleep disturbances, a sense of inability to experience pleasure (i.e., anhedonia), and frequent complaints about “achiness,” which possibly

may reflect a reduced threshold for pain sensitivity. Many of these signs and symptoms, including those that reflect a negative-affect state (e.g., anxiety, distress, and anhedonia) also have been demonstrated in animal studies involving various models of dependence (Becker 2000).

Although many physical signs and symptoms of withdrawal typically abate within a few days, symptoms associated with psychological distress and dysphoria may linger for protracted periods of time (Anton and Becker 1995; De Soto et al. 1985; Martinotti et al. 2008). The persistence of these symptoms (e.g., anxiety, negative affect, altered reward set point manifesting as dysphoria and/or anhedonia) may constitute a significant motivational factor that leads to relapse to heavy drinking.

Studying Alcohol Relapse Behavior

Relapse may be defined as the resumption of alcohol drinking following a prolonged period of abstinence. Clinically, vulnerability to relapse commonly is associated with an intense craving or desire to drink. Although a precise definition for craving remains elusive (Anton 1999; Koob 2000; Littleton 2000), and there even is some debate about the role of craving in relapse (Miller and Gold 1994;

Rohsenow and Monti 1999; Tiffany and Carter 1998), there is no question that relapse represents a prevalent and significant problem in alcoholism. In fact, given the high rate of recidivism in alcoholism, relapse clearly is a major impediment to treatment efforts. Consequently, substantial research efforts have been directed at modeling relapse behavior, as well as elucidating neural substrates and environmental circumstances that are associated with or promote excessive drinking.

Events that potently trigger relapse drinking fall into three general categories: exposure to small amounts of alcohol (i.e., alcohol-induced priming), exposure to alcohol-related (i.e., conditioned) cues or environmental contexts, and stress. Clinical laboratory studies have found that compared with control subjects, alcohol-dependent people are more sensitive to the ability of these stimuli and events to elicit craving and negative affect, which in turn presumably drives an increased desire to drink (Fox et al. 2007; Sinha et al. 2008). The combination of these clinical laboratory procedures with neuroimaging techniques has proven to be a powerful tool allowing investigators to identify brain regions that are more strongly activated in alcohol-dependent subjects than in control subjects when they are

exposed to these stimuli/events (George et al. 2001; Myrick et al. 2004; Wrase et al. 2002). Similar experimental procedures have been employed to evaluate the ability of pharmacotherapeutics to quell craving and temper the brain activation provoked by alcohol-related cues in humans (Anton et al. 2004; George et al. 2008; Myrick et al. 2007, 2008; O'Malley et al. 2002).

More detailed insight regarding mechanisms underlying fundamental changes in brain function that occur as a consequence of dependence and which relate to enduring relapse vulnerability have been gained through research in animals. Several animal models have been used to study alcohol self-administration behavior and the issue of relapse (for reviews, see Le and Shaham 2002; Sanchis-Segura and Spanagel 2006; Weiss 2005). In one type of model, animals with a long history of daily access to alcohol are abruptly denied access to the drug. When alcohol is reintroduced after this period of "forced" (i.e., experimenter-induced) abstinence, the animals exhibit a transient increase in alcohol consumption. This alcohol deprivation effect has been demonstrated using both measures of voluntary alcohol consumption and operant procedures3 (Heyser et al. 1997; Sinclair 1979; Spanagel and Holter 1999). Another

model frequently used to study alcohol (and other drug) relapse behavior involves operant reinstatement procedures (Shaham et al. 2003). In this model, animals first are trained to respond for access to alcohol (i.e., to receive the reinforcement provided by alcohol). Then, the response-contingent reinforcement is interrupted with extinction training—that is, even if the animals perform the required response, they do not receive alcohol; as a result, the animals eventually reduce or even completely stop responding. When the animals then are exposed again to small alcohol doses, environmental stressors, or stimuli previously associated with delivery of alcohol (i.e., conditioned cues), they resume responding (to varying degrees)—as if "seeking" alcohol reinforcement (Le et al. 1998, 2000; Weiss et al. 2001). This renewed alcohol-seeking behavior becomes even more robust when several of these relevant stimuli are presented in combination (Backstrom and Hyytia 2004; Liu and Weiss 2002b). Interestingly, this reinstatement of alcohol responding occurs even though the animals still do not receive alcohol reinforcement.

This experimental design can be further modified by the use of discriminative contextual cues. This means that certain

contextual cues (e.g., a unique odor or testing environment) will indicate to the animal that responding will pay off with delivery of alcohol reinforcement, whereas a different contextual cue is used to signal that responding will not result in access to alcohol. If the responding is extinguished in these animals (i.e., they cease to respond because they receive neither the alcohol-related cues nor alcohol), presentation of a discriminative cue that previously signaled alcohol availability will reinstate alcohol-seeking behavior. This renewed alcohol-seeking behavior can be observed even after a long period of time has elapsed since the animals last were given an opportunity to self-administer alcohol, suggesting that these contextual cues can serve as powerful triggers for relapse-like behavior (Ciccocioppo et al. 2001; Katner and Weiss 1999; Katner et al. 1999). Additional studies (Chaudhri et al. 2008; Zironi et al. 2006) found that reexposure of the animals to the general environmental context in which they could self-administer alcohol not only enhanced subsequent alcohol responding but also modulated the ability of alcohol-conditioned cues to reinstate alcohol-seeking behavior.

Finally, and perhaps most importantly, animals used in all of these models generally have demonstrated sensitivity to

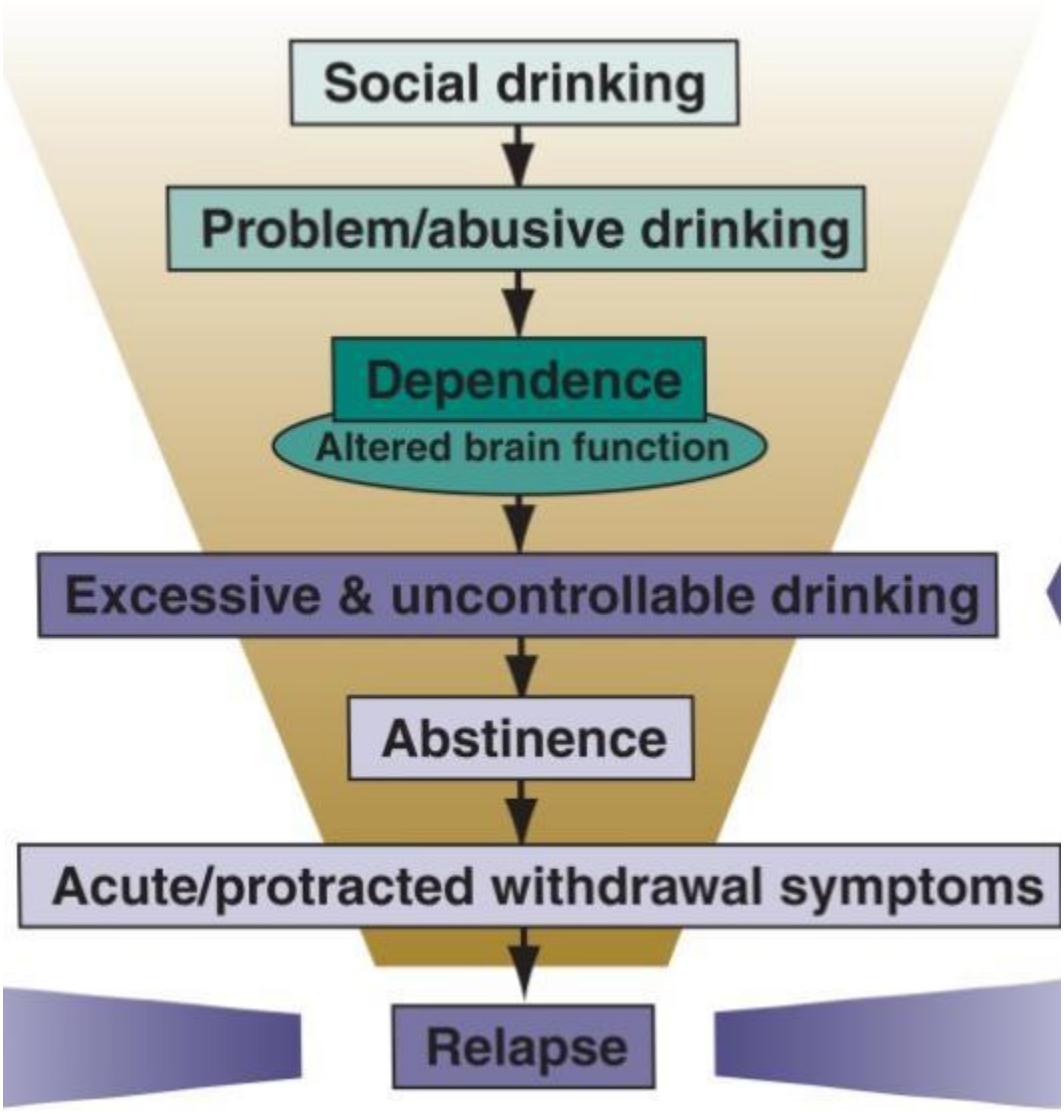
treatment with various medications that have been shown to be clinically effective in preventing and/or retarding alcohol relapse (Burattini et al. 2006; Heilig and Egli 2006; Le and Shaham 2002; Marinelli et al. 2007b; Spanagel and Kiefer 2008). From a clinical standpoint, this is important because it underscores the value of these models in identifying and evaluating new treatment strategies that may be more effective in battling the problem of relapse.

Alcohol Dependence, Withdrawal, and Relapse

As mentioned earlier, alcohol addiction is a complex and dynamic process (see figure 1). Prolonged excessive alcohol consumption sets in motion a host of neuroadaptive changes in the brain's reward and stress systems (for reviews, see Hansson et al. 2008; Heilig and Koob 2007; Koob and Le Moal 2008; Vengeliene et al. 2008). The development of alcohol dependence is thought to reflect an allostatic state—that is, a state in which the chronic presence of alcohol produces a constant challenge to regulatory systems that attempt (but ultimately fail) to defend the normal equilibrium of various internal processes (i.e., homeostatic set points). In the dependent individual, this allostatic state is fueled by progressive dysregulation of the brain's reward and stress systems beyond their normal

homeostatic limits (Koob 2003; Koob and Le Moal 2001). These neuroadaptive changes associated with dependence and withdrawal are postulated to impact the rewarding effects of alcohol and, consequently, contribute to the transition from controlled alcohol use to more excessive, uncontrollable drinking. Manifestations of these perturbations in brain reward and stress systems also appear to mediate the myriad symptoms of alcohol withdrawal, as well as underlie persistent vulnerability to relapse.

Schematic illustration of how problem drinking can lead to the development of dependence, repeated withdrawal experiences, and enhanced vulnerability to relapse. Alcohol dependence is characterized by fundamental changes in the brain's reward and stress systems that manifest as withdrawal symptoms when alcohol consumption is stopped or substantially reduced. These changes also are purported to fuel motivation to reengage in excessive drinking behavior. Repeated bouts of heavy drinking interspersed with attempts at abstinence (i.e., withdrawal) may result in sensitization of withdrawal symptoms, especially symptoms that contribute to a negative emotional state. This, in turn, can lead to enhanced vulnerability to relapse as well as favor perpetuation of excessive drinkin



Methodology:

This research uses a cross-sectional comparative study design with a sample size of 60 drawn from a population of clinically diagnosed patients of alcohol dependence (n = 30) or opioid dependence (n = 30) and seeking treatment for relapse. In addition to collecting sociodemographic data, other factors such as craving, affect, self-efficacy, and

expressed emotions were measured using standardized instruments including brief substance craving scale, Bradburn affect balance scale, drug avoidance and self-efficacy scale and family emotional involvement, and conflict scale. The data were statistically analyzed.

Results:

Disparity in sociodemographic factors was seen in both the groups with opioid group being more likely to be single, unemployed, belonging to lower socioeconomic status, and having a criminal record (P = 0.025). Among factors associated with relapse, the opioid group scored significantly higher on craving, perceived criticism (P = 0.0001), and lower on self-efficacy (P = 0.016). Most common reason cited for relapse in both the groups was desire for positive mood.

Conclusion:

This study highlights the role of social determinants in drug dependence and relapse. Relapse was found to be a complex multifactorial phenomenon. Despite differences in presentation, somewhat similar relapse mechanisms were seen in both groups.