A Treatment Model for Craving Identification and Management†

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Abstract—This article presents an addiction treatment model based on craving identification and management (CIM). Craving is broadly defined as the desire to use alcohol or other drugs; it increases the likelihood of use of these substances. In the CIM Model treatment interventions are referenced to craving, i.e., helping clients to identify their craving level and equipping them with strategies to avoid use. Four causes of craving are identified: (1) environmental cues (triggers): exposure to people, places, and things associated with prior drug-using experiences may cause immediate and overwhelming craving; (2) stress: addicted persons experience stress as craving; (3) mental illness; and (4) drug withdrawal: symptoms of both mental illness and withdrawal lead to craving if clients associate use with relief of these symptoms. The CIM Model incorporates four service delivery elements: Relapse Prevention Workshop, individual counseling, medical/psychiatric services, and screening for ongoing drug use. At its core, the CIM Model asks clients to be aware of craving, analyze its causes, and, based on those causes, implement specific strategies to prevent and manage craving. The CIM Model combines several treatment components, including control of exposure to environmental cues, establishment of a daily schedule, the use of behaviors that dissipate craving (tools), and treatment (with medications when appropriate) of mental health and withdrawal symptoms. The CIM Model is a client-derived approach to achieving and maintaining sobriety based on a process of analyzing craving and managing it with an individualized program of recovery activities.

Keywords—addiction, cognitive-behavioral, craving, recovery, treatment

Addiction is a disease of the pleasure-producing chemistry of the brain. Four interrelated symptoms define addiction: loss of control over pleasurable activities (such as alcohol, other drugs, gambling, sex), continued use despite adverse consequences, craving (desire to use) and denial (distortions in thinking that protect drug use). Craving is the result of neuroadaptive changes induced by overstimulation of reward chemistry. The natural history of addiction is characterized by progressive loss of control over use, so that the loss of control occurs more readily as the disease progresses. Effective treatment interrupts the pathophysiology of the disease: compulsive use in the face of adverse consequences, driven by craving and protected by denial. Though the fantasy of many addicts is to return to controlled use of substances, this appears to be unrealistic for most because addiction is not only progressive but also chronic.

The societal response to persons with addictive disease has been to decrease the availability of drugs and increase the adverse consequences of use. The 1914 Harrison Narcotic Act restricting opiate use, the anti-alcohol era of Prohibition,

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and the recent "War on Drugs" represent legislated solutions to drug abuse. This approach has had successes in reducing the availability and increasing the costs of illicit drugs, primarily by incarcerating drug sellers and drug users. However, incarceration does not treat the diseased brain chemistry, and recidivism rates for drug users are high. Early addiction treatment models were derived from the 12-Step program of Alcoholics Anonymous, a successful self-help program developed by and for addicts. For example, the Minnesota Model is an inpatient or residential treatment approach with a core curriculum derived from 12-Step principles in which spirituality anchors the addict's commitment to behavior change. The residential treatment programs added environmental isolation and rigid structure to prompt addicts to learn new skills to improve disease management. Recently, treatment models utilizing theories from several disciplines have shown potential in helping addicts manage their disease, including cognitive-behavioral (Beck et al. 1993), psycho-educational, motivational enhancement (Miller & Rollnick 1991), and hybrid models that combine aspects of several models. The Matrix Model (Rawson 1995) is a well-known model of this type. Today, the goal of most treatment programs is to provide addicts with sufficient skill to maintain control over their behavior and sustain sobriety.

This article presents a treatment model based on craving identification and management. Grounded in the medical model, it focuses on craving as the pathophysiologic consequence of drug-induced injury to reward function. The Craving Identification and Management (CIM) Model is a distillation of feedback from clients about what has worked for them in achieving and maintaining sobriety in their own prior treatment experiences. Their ideas reflect the most pragmatic and effective methods addicts use to manage craving. This interactive process has generated the hypothesis that the final common step leading to drug addiction is loss of control over craving. For addicts, use occurs when craving overwhelms their relapse prevention strategies and control over behavior is lost. In the CIM Model, treatment interventions are referenced to craving, i.e., helping clients identify how close they are to using, and equipping them with preventive strategies to avoid use. Since craving is a common denominator in addictive disorders, craving identification and management is applicable to drug, alcohol, and other behavioral addictions. The CIM and Matrix Models have been shown to have similar efficacy for methamphetamine users (Rawson et al. 2004).

THEORETICAL FRAMEWORK

An addictive drug is a pleasure-producing chemical, whether legal or illicit, which affects brain pleasure and reward chemistry. These drugs activate pathways in the brain associated with feelings of well being, pleasure, and euphoria. Pleasure serves to reward completion of instinctive drives, adds interest to learning, and motivates behavior toward rewarding activities. In order for a drug to have abuse potential, it must interact with specific brain chemistry in the pleasure and reward centers of the brain.

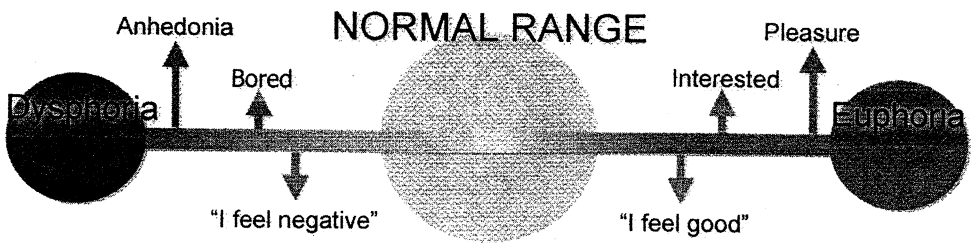
Generally, the intensity of the high is related to the degree to which the pleasure chemistry is activated. In addition to receptor-binding properties of the drugs, their pharmacokinetic properties have a major role in producing euphoria. Drugs that are delivered to the brain in high concentrations over a short time concentrate the high; the dopamine and endorphin generated by the drug are abruptly dumped in the pleasure centers proportional to the sudden appearance of high concentrations of the drug. The resultant rush of euphoria is intensely pleasurable, leading to loss of control over use of the drug more quickly than occurs with lower-intensity drugs such as marijuana or alcohol.

Drugs of abuse produce their euphoric "high" by augmenting dopamine and endorphin functions in the pleasure and reward centers of the brain. By overstimulating reward circuits, drugs force adaptive changes that blunt the drug's effect. Neuroadaptation is the process by which receptors in the reward and pleasure centers adjust to the high concentrations of dopamine and endorphin produced by the drugs. The principal mechanism of addictive disease is the relentless neuroadaptation that drugs induce through overstimulation of pleasure chemistry.

Cessation of drug use is characterized by a rebound towards dysphoria. Sobriety results in dramatic changes: inability to mobilize energy (anergia), the loss of ability to experience pleasure (anhedonia), and appearance of drug hunger or craving. Essentially, drugs injure sobriety by rendering it pleasureless. Drugs damage the ability of the user to experience normal pleasure, to obtain enjoyment from the activities of daily life (Figure 1). Daily life becomes unrewarding, and the user withdraws from activities that were formerly enjoyable. The user's normal mental state changes from feeling well to feeling empty, bored or flat. For many users, sobriety itself becomes so unpleasant and dysfunctional that the risk of relapse to substance use is heightened.

Craving is broadly defined as the desire to use. Underlying the awareness of craving are changes that occur in the brain's reward circuitry. These changes manifest as boredom, restlessness, irritability, and distractibility and may occur without conscious awareness of the desire to use. Higher levels of craving produce feelings of anger, anxiety, frustration, a feeling of entitlement, depression and mood swings. Occasionally there is a feeling of breezy and superficial elation consistent with manic swings of mood. Though not all negative affective states represent craving, "feeling negative" is a common antecedent to conscious awareness of a desire to use. Neuroimaging studies of the brain in the craving state document a characteristic pattern of physiologic changes. As the physiological/psychological discomforts increase, desire to use increases. Intense

FIGURE 1
Pleasure Scale



levels of craving produce elevations in blood pressure, pulse, sweating, and dysphoria. Ultimately, craving induces an intense psychological preoccupation with getting and using the drug. Craving that is too intense, too severe, or too uncomfortable results in the loss of control over behavior.

LITERATURE REVIEW

The CIM Model is the result of an ongoing collaboration between treatment staff and the hundreds of clients who have attended the Relapse Prevention Workshops (RPW) conducted at the New Leaf Treatment Center. Over the 10 years of model development these clients have identified four common causes of craving as noted below. The literature for each cause of craving is reviewed briefly.

1. Environmental cues (triggers): cues cause immediate, catastrophic, overwhelming craving stimulated by the people, places, and things associated with prior drug-using experiences (O'Brien et al. 1990).
2. Stress: addicted persons experience stress as craving (Wallace 1989).
3. Mental illness: inadequately treated or untreated symptoms may lead to self-medication with alcohol or other drugs (Rohsenow, Corbett & Devine 1988; Khantzian 1985).
4. Drug withdrawal: inadequately treated or untreated symptoms may be experienced as intolerable and hence lead to use (Kosten 1990).

Environmental Cues

The literature supports a clear relationship between environmental cues and drug dependence both in clinical research and in animal studies. The conditioned place-preference test, widely used to assess the reinforcing properties of drugs, relies on this relationship. In conditioned place preference testing, animals are repeatedly administered a drug, and then permitted to choose between being in that environment and a dissimilar one. When drugs of abuse are tested in this paradigm, a preference usually develops for the environment paired with administration of the drug. In addition to this conditioned response, presumably based on the reinforcing effects of drugs, the pairing of drug-withdrawal effects with environmental cues has long been hypothesized to form a classical conditioning basis for craving (Ludwig & Wikler 1974; Wikler 1948).

An abundance of clinical data also supports the role of the environment in drug craving and use. In the laboratory setting, the task of imagining being in a cocaine-use environment was associated with a higher level of craving for both alcohol and cocaine than was imagining being in a neutral setting (Sinha et al. 2000). Presentation of opiate- (Grüsser, Heinz & Flor 2000; Kasvikis et al. 1991; Sideroff & Jarvik 1980), marijuana- (Grüsser, Heinz & Flor 2000), cocaine- (Grüsser, Heinz & Flor 2000; Kranzler & Bauer 1992; Childress et al. 1988), and alcohol-related (Schneider et al. 2001; Grüsser, Heinz & Flor 2000) cues increases craving for those substances. In a sample of 35 crack cocaine addicts, 12 (34%) identified environmental cues as relapse triggers (Wallace 1989). Environmental stimuli have been reported to lead to alcohol craving (Westerberg 2000; Ludwig 1988) and relapse (Ludwig 1988). With respect to cigarettes, environmental stimuli are associated with low abstinence self-efficacy (Gwaltney et al. 2001) and craving (Steuer & Wewers 1989). A variety of environmental factors have been identified as precipitants of relapse to opiates (Bradley et al. 1989).

Stress

Several lines of evidence support the hypothesis that stress plays a role in acquisition and continuation of drug use. Behavioral stress in animals has been shown to facilitate the acquisition of self-administered morphine (Shaham & Stewart 1994; Alexander, Coambs & Hadaway 1978) and cocaine (Miczek & Mutschler 1996; Goeders & Guerin 1994; Ramsey & van Ree 1993). Stress in animals has also

been shown to reinstate drug-seeking behavior in alcohol- (Lê et al. 1998), nicotine- (Buczek et al. 1999), heroin- (Shaham & Stewart 1995), and cocaine-experienced animals (Erb, Shaham & Stewart 1996).

Human laboratory work has been conducted with cocaine users in order to define more precisely the role of stress in drug craving (Sinha et al. 2000; Sinha, Catapano & O'Malley 1999). Subjects prepared a script describing a situation they had found very stressful, but which was not associated with drug use; then, they were read these scripts and asked to imagine themselves in the same situation. Compared to a neutral imagery task, this situation led to higher levels of cocaine (Sinha et al. 2000; Sinha, Catapano & O'Malley 1999) and alcohol (Sinha et al. 2000) craving. Data from clinical settings has been inconsistent with respect to the role of stress in relapse to cocaine use (Hall, Havassy & Wasserman 1991; Wallace 1989), perhaps in part due to methodologic inconsistencies. In opiate addicts, stress is associated with greater drug use (Brewer et al. 1998; Kosten, Rounsaville & Kleber 1986).

Mental Illness/Psychiatric Comorbidity

A primary relapse factor cited in the literature (Marlatt & Gordon 1985) is the presence of negative emotional states originating from a variety of sources, including acute or long-term withdrawal, dysphoria related to depression or emptiness related to a borderline or narcissistic personality disorder. It is clear from the literature that a high percentage of persons with addictive disease also have other Axis I or Axis II diagnoses. Making another diagnosis has always been a tricky endeavor given that both drug intoxication and drug withdrawal mimic various Axis I and II diagnoses.

Regier and colleagues (1990) found that of the people with a cocaine use disorder, 76.1% had a comorbid disorder. The primary diagnoses often cited in relation to addiction are mood disorders and anxiety disorders. The percentage of people using cocaine who also have a mood disorder ranges from 33% to 54% (Galanter 1993; Rounsaville et al. 1991; Regier et al. 1990; Crowley et al. 1987; Gawin & Kleber 1986; Weiss et al. 1986). Anxiety disorders are also reported frequently. Ross (Ross, Glaser & Germanson 1988) reported that 60% of patients being treated for addictive disease also had a lifetime diagnosis of anxiety disorder. Forty-five percent reported symptoms of an anxiety disorder in the past month, including generalized anxiety disorder, panic disorder, and post-traumatic stress disorder. Other studies show a high correlation between substance use and a history of sexual abuse (PTSD). Grice and colleagues (1995) reported that 80% of women entering treatment for addiction reported a history of sexual abuse.

Khantzian (1985) posited that addiction was underpinned by psychological dysfunction, including problems with affect regulation, self-esteem, interpersonal relations, and self-care. Foote and colleagues (1994) modified the neurobehavioral treatment program to address these underlying issues and thereby improve treatment for more dysfunctional clients. They defined four areas of difficulty: regulation of affect, interpersonal conflicts, "self" deficits, and externalizing defenses. These Axis 1 issues as well as the underlying characterological issues can increase turmoil and subsequent craving for many addicts.

Withdrawal/Abstinence Syndrome

One of the primary causes of craving is withdrawal from the drug itself. Physical dependence means the individual experiences physical symptoms when use of the substance is discontinued. The symptoms that appear, constituting an abstinence syndrome, include physical dysfunction (pain, stomach cramps, diarrhea) as well as disturbances of sleep, disorders of mood, anhedonia and anergia, drug craving, and occasionally, psychotic symptoms. Similar to adaptive changes in the brain, the overstimulating effects of the drug on other bodily processes induce counterbalancing changes. Hence, withdrawal symptoms are the mirror image of the drug effects: if a drug increases blood pressure, in withdrawal blood pressure is decreased. The process is one of tolerance to the drug and the physiological need to use more, both to achieve the same effect and to avoid symptoms that develop if use is below the tolerance point.

Many individuals have both the disease of addiction and physical dependence. If the addict is physically dependent, the appearance of withdrawal symptoms activates the drive to use, drug craving becomes extremely intense, and often the individual loses control. Withdrawal symptoms require immediate intervention when there is interference with daily function and/or when the physiological symptoms may have adverse medical consequences. If the client reports symptoms that interfere with daily function for more than three consecutive days or the client's daily function is deteriorating or the client is unable to sustain sobriety, the use of medications to treat the withdrawal symptoms is indicated. Detoxification is defined as the use of medications to treat withdrawal symptoms. "Symptom-driven" detoxification provides medications proportional to the severity of the symptoms and for the length of time needed to ameliorate the withdrawal symptoms. Good symptom control increases the client's participation in treatment and reduces a major source of craving.

Craving is the final common mediator in drug use. The utility of craving in predicting drug and alcohol use lies at the heart of the theoretical basis of the model. Although there has been debate about the definition of craving and whether it is a useful construct, various investigators have addressed the issue and have typically defined craving as a desire for a drug or alcohol. Retrospective assessment of reasons for relapse has shown a significant but not predominant role for craving (Marlatt 1996; Miller