Long-term effects of certain drugs, happen due to the addiction system in the brain. This can be known as the dopamine system. The dopamine system is the brains reward circuity, and changes within this system occur with prolonged drug usage.

The oribital cortex, is apart of the prefrtontal coretex that receives information from the medial dorsal nucles of the thalamus. This part of the brain is thought to represent emotion, taste, smell, and reward in decision making. It gets the name, as it sits abovce the eyes. This area is directly connected to brain regions involved in the reinforcing effects of drugs, creating a feedback loop that can contribute to compulsive drug use. (Norda D. Volkkow, Joanna S. Fowler 2000).

The following imaging studies done on the OFC have used positron emission tomography (PET) in conjuection with an analog of glucoge to measure brain function. It can allow researchers to map areas of the brain that change during drug administration.

* Volkow has studies 25 years of neurscinece research, addiction is a diesaede of the brain
* These studies have shown how repeated drug use can target key molecules and brain circuits, and eventually disrupt the higher order processes that underlie emotions, cognition and behavior.
* addiction is characterized by an expanding cycle of dysfunction in the brain
* impairment typically begins in the evolutionarily more primitive areas of the brain that process reward, and then moves on to other areas responsible for more complex cognitive functions. Thus, in addition to reward, addicted individuals can experience severe disruptions in learning (memory, conditioning, habituation), executive function (impulse inhibition, decision making, delayed gratification), cognitive awareness (interoception) and even emotional (mood and stress reactivity) functions.
* Drawing largely from the results of brain imaging studies that used positron emission tomography (PET),
* The disease of addiction uses the neurotransmitter dopamine as its “currency” to send information
* Interestingly, directly or indirectly, all addictive drugs work by triggering exaggerated but transient increases in extracellular DA in a key region of the reward (limbic) system [6, 7], specifically, in the nucleus accumbens (Nac) located in the ventral striatum.
* Stimulants, nicotine, and alcohol within the ventral striatum, are linked to the subjected experience (such as the high) during intoxiciation.
* PET studies can show when users experience the high, correlated with the changes in DA system. Highest displays of DA show high reported level of the “high”
* Animal and human studies have demonstrated that the speed with which a drug enters, acts upon, and leaves the brain (i.e. its pharmacokinetic profile) plays a fundamental role in determining its reinforcing effects.
* it follows that making sure that an addictive drug enters the brain as slowly as possible should be an effective way of minimizing its reinforcing potential, hence its abuse liability
* e designed an experiment to test precisely this hypothesis with the stimulant drug MPH, which, like cocaine, increases DA by slowing down its transport back into the presynaptic neuron (i.e. by blocking DA transporters), thus magnifying the DA signal. Indeed, we found that, while intravenous administration of MPH is often euphorigenic, orally administered MPH, which also increases DA in the striatum [15], but with 6- to 12-fold slower pharmacokinetics, is not typically perceived as reinforcing [16, 17]. Thus, the failure of oral MPH – or amphetamine [18] for that matter – to induce a high is likely the reflection of their slow uptake into the brain [19]. Therefore, it is reasonable to propose the existence of a close correlation between the rate at which a drug of abuse enters the brain, which determines the speed at which DA increases in the ventral striatum, and its reinforcing effects [20, 21, 22]. In other words, for a drug to exert reinforcing effects it has to raise DA abruptly. Why should this be so?
* Based on the magnitude and duration of neuronal firing, DA signaling can take one of two basic forms: phasic or tonic.
* hasic signaling is characterized by high amplitude and short burst firing, whereas tonic signaling has typically low amplitude and a more protracted or sustained time course.
* The distinction is important because it turns out that phasic DA signaling is necessary for drugs of abuse to induce “conditioned responses,” which is one of the initial neuroadaptations that follow exposure to reinforcing stimuli (including a drug).
* On the other hand, tonic DA signaling plays a role in the modulation of working memory and other executive processes. Some of the features that distinguish this mode of signaling from the phasic type are that it operates mostly through lower affinity DA receptors (DA D1 receptors).
* protracted drug exposure (and changes in tonic DA signaling through these receptors) has also been implicated through modification of NMDA, AMPA, and Glutamate receptors.
* Evidence show abrupt drug-induced increases in DA mimic phasic DA cell firing. Chronic use can cause powerful condition response to the drug itself.
* The evidence showing that fast dopamine increases are likely for addiction, they are not sufficient.
* Repeated drug exposure causes changes in dopamine brain functions that develop over time, due to secondary neurodapdatrin in neurotransmitter systems (glutamate and maytbe GAGA).
* While dopamine is the first affected circuit, other circuits of the brain will be affected. This will be shown, at the second half
* The other circuits affected, will be motivation/drive/ inhibotry control/ executive function/ and memory/conditioning, those are modulated by dopamine.
* Addicted individuals have the neuro adaption of lower dopamine type 2/3 receptors and the amount of dopamine released naturally.
* Associated with lower activity in areas of the prefrontal cortex, that are necessary for executive performance.
* Reduced dopamine would explain addicted subjects reduced dopamine in natural rewards (such as food, sex, work etc), and the means of drug use to compensate for this deficit.
* Addicted individuals have less D2R and lower activity in orbito-frontal cortex
* Imaging studies show a decrease in dopamine in the ventral striatum and other regions of striatum. This was unexpected since hypothesis suggested that addiction reflectred ehanced sensitivity to the rewading (hence dopaminergic). This could be explained due the neurological changes, making it so that drug use replaces the dopamine feeling associated with normal activities.
* Dysfunctional regions of the frontal brain, might explain impaired control when confronted with drugs.
* Study done by author looked at a family with a history or alcoholism, but the family were not alcholics. They showed an increase in type 2 and 3 receptors. Mean of 24 years old and a SD of 3. The family showed higher than control levels of type 2 and 3, lead author to speculate that people with genetic history of addiction, might have increased type 2 and 3 receptors, to protect against that addiction. The data suggested that higher levels of type and 3 receptors can protect agains impulsivity by regulating the circuits involved with behavioral response and in controlling emotions.
* Author did another study testing the hypothesis that prefrontal regions are involved with reduced release of DA in the stratium. Tested the relationship between baseline PFC activity and increases in DA by administration of MPH (drug). In acholhics, failed to detect the expected correlation between PFC activity and DA release in the straium.
* Between two studies, a link was found between reduced baseline activity in the PFC and reduced straitial D2R in drug-addicted subjects, and between baseline PFC activity and DA release in controls that is not present in addictes subjects.
* These studies indicate that there are strong connections between neuroadaption in the PFC pathways and downstream dsyfunctions in the DA reward and motivation system. Likely due to the influence of PFC impulsivity and compulsivity.
* The studies failed to account for additional behavioral phenomena such as the effects odff drug associated cues in trigeering craving, which would likely implicate memory and learning circuits.
* The over-stimulation of dopamine in the ventral stiratum, establishes powerful connections in the brain to satisfy the urge, and the events around that urge (ie, where are you, what do you do, your ritual for the drug). Suggesting that there are many cues that can begin the firing of dopamine, or rather stimuli that affect the release of DA, and tell the brain it is time to use the drug. Can suggest why someone who detoxes for awhile, can relapse, they experience the stimuli once again.
* In studies done using PET scans, two independent sutides were down showing different stimuli to cocaine abusers. One showed a video of someone smoking cocaine, and the other showed nature footage. The people who viewed the video of smoking cocaine, had an increase in dopamine, whereass the other group had no increase. This further strengthens the theory that when exposed to addiction related stimuli, the brain will release to dopamine to essentially beging the “quest” to receive the reward (drugs).
* Lingering puzzles in drug addiction is the overwhelming ucraving to take drugs that can reemerge after years of abstinence, comprosied ability of addicited individuals to iinhibit drug seeking once the craving erupts, even though they are aware of the consequences.
* The author proposes a multidimensional model to explain this disease. A network of four interreletaed circuits, when combined, can show the dysfunctional behaviors of the regular features of addiction. Reward,in the ventral striatum / motivation and drive located in the OFC, subcallosal cortex, dorsal striatum and motor cortex. Memory and learning, in the amygdala and the hippocampus. Plaaning and control, in the dorsolateral prefrontal cortex and infrerior frontal corext. The following circuits receive dopamine but are also connected through direct or indirect means

Introduction to Drug Use and its Impact (Grace Conley) :

Drug use can lead to fundamental changes in the brain that affect how a person thinks and acts. All you can think about is when is the next time I am going to get/use them. It makes it extremely hard to enjoy the things that used to give you joy. In 2017, 8.5 million American adults suffered from both a mental health disorder and a substance use disorder (AAC,2024). Drug use also has a significant impact on society, people's families are getting torn apart from excessive amounts of drug use. Childrens whose parents abuse drug use normally are physically and emotionally abused. Shortly after use, effects can include altered consciousness, impaired memory, disinhibition, euphoria, inattention, altered judgment, and more. Meanwhile, long-term use can lead to impairments across multiple cognitive domains, including memory, attention, and executive function (EMC 2015).

cognitive impairments resulting from traumatic brain injury (TBI) and explores various rehabilitation strategies. TBI, a major public health concern, leads to a wide range of cognitive deficits, necessitating comprehensive neuropsychiatric evaluation and rehabilitation. Common impairments include attention deficits, memory issues, language and communication disorders, visuospatial perception changes, and executive function impairments. Rehabilitation involves a multidisciplinary approach with tailored interventions such as attention process training, errorless learning for memory deficits, pragmatic language skills training, metacognitive strategy training, and problem-solving training. Both restorative and compensatory approaches are utilized, often in combination with pharmacotherapy. Additionally, family therapy, cognitive behavior therapy, noninvasive brain stimulation techniques, and comprehensive holistic rehabilitation programs play crucial roles in addressing the multifaceted challenges posed by TBI-related cognitive impairments. Despite advancements, the heterogeneous nature of brain injuries necessitates further research to establish evidence-based treatment protocols, particularly in developing countries like India, where effective and low-cost interventions are essential.

Neurological Mechanisms (Kyler Suess):

When assessing the long-term effects of drugs on consciousness and cognitive function, it is imperative to explore the neurological mechanisms through which drugs can alter consciousness and cognitive function, including changes in neurotransmitter activity and brain structure. These changes elucidate the brain alterations that precipitate drug use, facilitate its long-term effects, and manifest consequent changes.

Before delving into the research on this topic, it is essential to grasp an understanding of the brain regions and their interplay with addiction mechanisms. Volkow et al. (2010) proposed a model delineating four circuits within the brain: reward, motivation/drive, memory/learning, and planning/control. The reward circuitry resides in several nuclei in the basal ganglia, particularly the ventral striatum, while motivation and drive are centered in the orbitofrontal cortex, dorsal striatum, and motor cortex. Memory and learning are associated with the amygdala and hippocampus, while planning and control are governed by the prefrontal cortex. These regions receive dopamine and are interconnected either directly or indirectly (Volkow et al., 2010). The model posits that long-term drug use can lead to dysfunction in the brain's addiction circuitry, affecting learning, executive function, cognitive awareness, and emotional regulation.

Studies conducted by Volkow aimed to demonstrate how long-term drug use can impact the brain regions associated with dopamine. Dopamine, a neurotransmitter, plays a pivotal role in conveying information within the model. These studies predominantly utilized positron emission tomography (PET), enabling the precise localization of physiological processes through the detection of gamma rays emitted by positron-emitting isotopes introduced into the body.

In one such study, PET was employed to monitor dopamine levels in the reward system. Craclopride, a dopamine receptor antagonist, was utilized to measure dopamine level changes within the reward system following different doses of I.V. methylphenidate in 14 healthy controls. The study revealed a correlation between the intensity of the "high" experienced by individuals and the levels of dopamine release. Subjects who did not report experiencing any euphoria exhibited low dopamine release levels. These findings suggest a direct correlation between stimulant-induced "highs" and dopamine release (Volkow et al., 1999). The researchers further suggest that the rate at which a drug enters the brain correlates with the observed high dopamine levels.

The research corroborates the hypothesis that long-term drug use entails repeated stimulation of the reward system. Stressors on the reward system can induce neuroadaptations in the circuits outlined in the four-circuit model. Another study by Volkow et al. (1993) suggested that prolonged drug use could lead individuals into a hypodopaminergic state, characterized by low dopamine receptor availability and diminished dopamine production in response to natural rewards, such as food consumption. PET imaging conducted on cocaine, methamphetamine, alcohol, and heroin users revealed reduced levels of available dopamine receptors in the test group compared to the control group. Long-term cocaine users, specifically, exhibited decreased metabolic activity in the orbitofrontal cortex following a 3- to 4-month detox, contrasting with the increased activity observed in the control group (Volkow et al., 2002). However, dopaminergic dysfunction in the striatum among long-term drug users does not fully account for other addiction traits like impulsivity, cravings, and relapse triggered by drug cues (Volkow et al., 2010).

Regarding impulsivity, it is hypothesized that impaired impulse control stems from dysfunctions in the frontal brain regions. Previous studies have demonstrated a correlation between reduced type 2 dopamine receptor availability and diminished brain activity in the prefrontal cortex (PFC), including the orbitofrontal cortex and cingulate gyrus (Volkow et al., 2010). Another study investigated individuals with a familial history of alcoholism alongside a control group. Individuals with a family history of alcoholism, despite not being alcoholics themselves, exhibited increased type 2 dopamine receptor availability. The study concluded that higher levels of D(2) receptor availability confer protection against addiction by regulating circuits involved in inhibiting behavioral responses and emotional control (Volkow, et. al, 2006a).

One significant study conducted by Volkow et al. (2006b) aimed to elucidate the long-term neurological effects of drug use by theorizing how stimuli can trigger dopamine release, fostering a dependency on the reward. This study involved 18 active cocaine users who had used the drug for at least 6 months. Participants viewed two videos: one depicting someone smoking cocaine and the other showcasing nature scenes. PET imaging revealed a decrease in available dopamine receptors upon viewing the cocaine video, indicating dopamine release. Participants also reported a strong urge to use cocaine after viewing the video.

Revisiting Volkow's et. al, (2010) model, the neurological effects of prolonged drug use can be expounded upon. Sustained exposure to drug use can induce alterations in the limbic (reward) system of the brain, thereby influencing an individual's motivation, memory, regulation, and emotions. This results from the excessive dopamine release elicited by long-term drug use. Upon drug consumption, a surge of dopamine overwhelms the PFC, impeding inhibitory control and perpetuating a positive feedback loop (Volkow et al, 2010).

Furthermore, prolonged exposure desensitizes the brain to tasks that typically induce dopamine release in sober individuals. Consequently, the limbic system learns to prioritize activities associated with the drug-induced reward system. While the limbic system's primary function is to ensure survival, powerful dopamine connections to drugs diminish the rewarding effects of normal activities over time. This phenomenon is attributed to the dwindling availability of type 2 dopamine receptors and decreased PFC activity.

(Chadia Salhi: Impact on Consciousness, and Recovery and Rehabilitation)

Drug use can have a significant impact on cognition, which refers to mental processes like thinking, memory, and decision-making. Certain drugs, such stimulants like cocaine or amphetamines, can increase alertness and concentration in short term but may lead to impaired cognition over time. Other drugs, like cannabis, can affect memory and decision-making abilities. Chronic drug can also alter brain structure and function, leading to long-lasting cognitive deficits. Overall, drug use can disrupt normal cognitive processes and impair overall mental functioning.

Drug use and mental illness are two significant public health issues that often intersect, creating complex challenges for individuals and society as whole. “Reports published in the Journal of the American Medical Association indicate that roughly 50% of individuals with severe mental disorders are affected by substance abuse, 37% of alcohol abusers, and 53% of drug abusers who also have at least one serious mental illness, and of all people diagnosed as mentally ill, 29% abuse either alcohol or drugs [9]”.( Assessment of anxiety and depression among substance use disorder patients: a case-control study. Spring Open) with substance use abuse often leading to various mental health conditions, including changes in mood and emotions, depression, suicidal thoughts, schizophrenia, and anxiety. Let’s explore how drug use can contribute to the development or exacerbation of various mental health conditions, shedding light on the detrimental effects of substance abuse on individual’s psychological well-being. Changes in mood and emotions are common symptoms experienced by individuals who engage in drug use. Substance abuse can alter brain chemistry and neural pathway, leading to fluctuations in mood ranging from euphoria and increased energy to irritability and aggression. The use of drug like stimulant and depressants can disrupt the brain’s natural balance of neurotransmitters, causing individuals to experience heightened emotions and erratic mood swings. Over time, chronic drug use can impair the brain’s stability to regulate emotions effectively, resulting in long-term changes in mood and emotional stability.

Depression is another mental health condition that is closely linked to drug use. “Two core features of depression are a markedly reduced interest or pleasure in activities and low mood (feelings of sadness), thus depressive personality traits are also linked to dysfunction of brain reward and motivational systems and may specifically relate to hypofunctioning of the mesolimbic dopaminergic system (Pizzagalli et al., 2009).” (The Detrimental effects of emotional process dysregulation on decision-making in substance dependence. National Library of Medicine). Many individuals turn to drugs as means of self-medicate and cope with feelings of sadness and hopelessness. However, substance abuse can worsen symptoms of depression over time by altering brain chemistry and affecting serotonin levels, a neurotransmitter associated with mood regulation. Prolonged drug use can lead to a vicious cycle of dependence and deteriorating mental health, making it challenging for individuals to break free from the grip of addiction and overcome depressive symptoms.

Suicidal thoughts are a serious concern for individuals struggling with drug addiction and mental illness. The risk of suicide is significantly higher among individuals with co-occurring disorders, as the combination of substance abuse and mental health symptoms can create overwhelming feelings of this pair and hopelessness, increasing the risk of suicidal ideation and self-harm. Drug use can impair judgment, lower inhibition, and exacerbate suicidal thoughts and behaviors. It is essential for health care providers to address both the substances use and mental health aspects of an individual’s condition to prevent tragic outcomes and provide comprehensive care.

Schizophrenia is a severe mental illness characterized by hallucinations, delusions, and disorganized thinking. Drug use, particularly the abuse of substances like methamphetamine or cannabis, can trigger psychotic episodes in individual predisposed to schizophrenia, leading to heightened paranoia and cognitive disturbances. Moreover, the use of drugs can also interfere with the effectiveness of antipsychotic medications making it challenging to manage symptoms and maintain stability. “Ferguson et al (2013) reported that individuals with cannabis use disorder at the ages of 18 and 21 had significantly higher rates of psychosis when compared to non-cannabis using participants (Fergusson et al., 2003), and Arseneault et al., (2002) found that adolescents using cannabis at the age of 15 were more likely to develop a schizophreniform disorder by the age of 26 when compared to non-using adolescents, even when controlling for prior psychotic symptoms Lastly, Schubar and colleagues demonstrated that cannabis use at the age of 12 was associated with a nearly 5-fold increase in odds of being hospitalized for psychosis later in life (2011).” (The Link Between Schizophrenia and Substance Use Disorder: A Unifying Hypothesis. National Library of Medicine).

Anxiety is a common mental health condition characterized by excessive worry and nervousness, can be exacerbated by drug use. While some individuals may use drugs to alleviate symptoms of anxiety temporarily, long-term substance abuse can acutely increase feelings of agitation and panic. “Anxiety can be caused by drug addiction. Anxiety commonly occurs during the acute withdrawal phase of alcohol and can persist for up to 2 years as part of a post-acute withdrawal syndrome, in about a quarter of people recovering from alcoholism [11]”(.Assessment of anxiety and depression among substance use disorder patients: a case-control study. Spring Open) Additionally, withdrawal from certain substances can cause intense anxiety symptoms, making it difficult for individuals to quit using drugs without professional support. Integrated treatment approaches that address both anxiety disorder and substance use are essential for long-term recovery.

Recovery and rehabilitation from drug addiction is a challenging journey that requires a combination of support, treatment, and dedication. There are various treatment options and strategies available to help individuals overcome their addiction and achieve long-term sobriety. Let’s explore the different approaches rehabilitation from drugs, including their effectiveness and potential challenges. There is study research done by Paul Duffy and Helen Baldwin, using a strategy that aims to offer support for individuals to choose recovery as a way out of dependency, moving beyond harm reduction. This study used A purposive sample of 45 participants was recruited from 11 drug treatment services in northern England. Semi-structured qualitative interviews lasting between 30 and 90 minutes were conducted one to three months after participants completed treatment. Interviews examined key themes identified through previous literature but focused on allowing participants to explore their unique recovery journey.

This study says the individual’s ability to recover from substance misuse can be understood in terms of their ‘recovery capital’, the initiation and maintenance of recovery. Recourses may stem from their social networks, education, employment, financial assets, health, beliefs, and values etc. recovery capital is believed to accumulate over time as a person stays abstinent from drugs and alcohol. Recovery capital and the usefulness of interventions aiming to boost aspects of recovery capital for longer term outcomes such us abstinence and preventing re-admission to treatment. Supportive relationships with peers, families and communities are suggested to be critical for ongoing recovery from substance misuse. Employment increases legitimate income and can improve living standards, both of which are important for recovery. Studies highlight a need for joint working between drug treatment commissioners, drug treatment services, employment services and employers to help substance users find work. Without forgetting to mention the importance of providing housing. Treating the co-occurring mental health is very important in the recovery process. The study result was, whilst most interviewees had remained completely abstinent (from all substances) since completing treatment, lapses had occurred for some clients and a small number saw no problem with continuing some substance use (generally not of the substances they had viewed as problematic) because they felt they had established a better level of control. By considering the various treatment options and strategies available, individuals can find a personalized approach that works best for their needs. It is important to remember that recovery is a lifelong process, and ongoing support and resources are utilizing a combination of treatment options and strategies, individuals can increase their chances of successful recovery and lead fulfilling, drug-free lives.

Social and Ethical Confederations: (Grace Conley)

Social and Ethical confederations with people facing cognitive impairment from drug abuse are intertwined with stigma, access to treatment, and legal considerations. The stigma is that mark of disgrace that people will see you as. Depending on where you live, and your financial stability it can be difficult to get access to treatment. There could be legal consequences if you choose to get help.

First, a stigma is a mark of a disgrace associated with a particular circumstance, quality, or person. Substance use disorders are chronic and treatable medical conditions studies show that people with these disorders still face discrimination and a stigma that can impact their mental health in many ways (U.S. DOHHS, 2023). This stigma makes people feel shame and not seek help when they need it. Some individuals experience cognitive impairment from drugs, and might not seek help, because of the stigma that goes around. It is that shameful embarrassing feeling that they get when they just want to better themselves. There are life changing tools out there that can help people recover from drug use, but the stigma connected with it is a key factor in why people do not seek help when needed.

Furthermore, for people with cognitive impairment of drug abuse and wanting to seek help, getting access to treatment is extremely limited. Some factors that may get in the way of seeking help could be geographic location, financial cost, and sigma. In some areas, depending on where you live, there is no treatment available, especially in marginalized communities. Changes in insurance may open the door for more people to be able to seek help. In 2016, 27.6 million people ages 0-64 did not have health insurance (ADC, 2024). States with high populations like California, New York, and Florida have many treatment centers. States with extremely low populations like Vermont and Wyoming have very few treatment centers.

Lastly, substance abuse has been historically seen as a crime then as a disorder. When a person who has a cognitive impairment of drug abuse is normally overlooked with that stigma. A major reason people do not seek help is because they do not want to face the legal consequences. In some states, the legal consequence requires you to go to mandatory rehabilitation programs. The federal law does protect information about individuals' substance misuse. The Confidentiality of Alcohol and Drug Abuse Patients Records regulation provides statutory authority for the confidentiality of patient records (RHI, 2024). This makes people want to seek help, by preventing any discrimination.

This review was designed to explore the long-term effects of drug use on cognitive function and consciousness. By exploring the neurological mechanisms underlying addiction, elucidating how long-term drug use can lead to dysfunctions in the brain’s reward circuity, impairing cognition, emotional regulation, and decision-making processes. The insights into the brain structure highlighted the complex nature of long-term drug usage and its effects on the brain. Moving beyond the neurological mechanisms, the social and ethical confederations were explored. The long-term usage of drugs on mental health can precipitate various mental illnesses such as depression, anxiety, schizophrenia, and suicidal thoughts. By examining the correlation between substance abuse and mental health disorders, an urgent need for treatment that addresses an individual’s condition, providing comprehensive care and support.

Furthermore, this review explored the social and ethical confederations associated with drug addiction, recognizing the societal stigma that prevents individuals suffering from long-term addiction from seeking treatment. These societal barriers included location, financial constraints, and logical considerations, which created concerns for the destigmatizing of addiction and improving access to evidence-based intervention.

In essence, this literature review serves as an explanation for long-term drug usage from a neurological perspective and provides an explanation of the difficulties an individual might experience dealing with this addiction. Based on the evidence, there is a lack of understanding of addiction in society. In order to combat this issue, requires a sustained effort from policymakers, healthcare professionals, and community stakeholders to prioritize the development and implementation of comprehensive strategies that address the root causes of addiction, reduce barriers to treatment, and promote holistic approaches to recovery.

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