           When evaluating the long-term effects of drugs on consciousness and cognitive function, it is crucial to explore the neurological mechanisms through which drugs can alter consciousness and cognitive function. This can also include changes in neurotransmitter activity and brain structure. The last two statements can be best understood as what changes in the brain occur that cause drug use to happen, allow it to become long-term, and what changes occur as a result of the long-term use.

           Before exploring the research done on this topic, it is important to gain an understanding of the regions of the brain and the relationship between those regions and the addiction mechanisms. A model proposed four circuits within the brain: reward, motivation/drive, memory/learning, and planning/control (Volkow N.D. 2010). The reward circuitry in the brain can be found within several nuclei in the basal ganglia, specifically the ventral striatum. Motivation and drive are located in the orbitofrontal cortex, dorsal striatum, and motor cortex. Memory and learning are located in the amygdala and hippocampus. Planning and control are found in the prefrontal cortex. These four regions of the brain all receive dopamine and are connected through either indirect or direct means (Volkow, N.D., 2010). The model suggests that long-term drug use can cause dysfunction in the addiction circuitry of the brain, such as learning, executive function, cognitive awareness, and emotional regulation.

           Studies done by Volkow were meant to show how long-term drug use can affect the regions of the brain associated with dopamine. Dopamine is a neurotransmitter that relays information about methods described in the model. The studies done draw largely on the use of positron emission tomography (PET). PET allows users to induce and introduce positron-emitting isotopes into the body to allow the precise location of physiological processes through the detection of the gamma rays produced by the isotopes. Using PET, Volkow was able to monitor which regions of the brain produced dopamine in subjects.

           One study done by Volkow used PET to monitor dopamine levels in the reward system. The study used Craclopride, a dopamine receptor that competes with endogenous dopamine for occupancy of the receptors, to measure changes in dopamine levels within the reward system following different doses of I.V. methylphenidate in 14 healthy controls. What was found in this study was that the level of “high” found in the individuals was correlated with higher levels of dopamine. Subjects who did not experience any kind of “high” showed low levels of dopamine release. The results of this study suggest that “highs” experienced from stimulants are followed by a direct correlation in dopamine release. Volkow goes on to state that the speed at which a drug enters the brain correlates with the high levels of dopamine found in the brain.

           The research supports the hypothesis that long-term drug use is based on repeated arousal of the reward system. The stressors in the reward system can cause neuroadaptations in the circuits outlined in the four-circuit model. Another study conducted by Volkow suggested that prolonged drug use would cause an individual to enter a hypodopaminergic state, meaning that the individual had low dopamine receptor availability and produced low levels of dopamine from natural rewards, like eating food. The study used PET technology on cocaine, meth, alcohol, and heroin users. The imaging found that the control group all had high amounts of specifically type 2 dopamine receptors, whereas the test group showed reduced levels of available receptors. Further research showed that individuals who were specifically long-term cocaine users showed reduced metabolic activity in the orbitofrontal cortex following a 3- to 4-month detox, whereas the control group showed an increase in OFC activity (Volkow et al., 1993). Long-term users of drugs with dopaminergic dysfunction in the striatum do not account for the other traits of addiction like impulsivity, cravings, and relapse triggered by drug cues (Volkow N.D., 2010).

           Exploring impulsivity, it is hypothesized that dysfunction in impulse control is due to dysfunctions in the frontal regions of the brain. Previous studies showed the connection between lower type 2 dopamine receptor availability and lower brain activities in the PFC, such as the OFC and cingulate gyrus (Volkow N.D., 2010). Another study was done investigating individuals with alcoholic fathers and at least two other relatives who were alcoholics, along with a control group. The study found that those individuals who were not alcoholics but had a family history of alcoholism had an increased amount of type 2 dopamine receptor availability. The conclusion from that study was that higher amounts of D(2) receptor availability protect against addiction. This is due to regulating the circuits involved in inhibiting behavioral responses and controlling emotions (COPED WORD, PLEASE FIX).

Finally, one more important study done by Volkow to understand the long-term effects on the neurological mechanisms of the brain from drug use is one theorizing how stimuli can release dopamine, causing the brain to start needing the reward. Meaning that the ritual someone might do before consuming their drug of choice has a strong impact on the release of dopamine. The final study discussed 18 active cocaine users for at least 6 months. Individuals watched two videos: one showing someone smoking cocaine, the other showing nature stories. Using the PET, upon viewing the cocaine video, individuals showed less available DA receptors, meaning there was a release of dopamine. The subjects also reported a strong urge to smoke after viewing the video.

Once again exploring the model produced by Volkow (2003), the neurological effects of long-term use can be discussed. Prolonged exposure to drug use can cause changes in the limbic (reward) system of the brain, changing an individual’s motivation, memory, regulation, and emotions. This is due to the unnatural amount of dopamine released from long-term drug use. When a drug is consumed, large amounts of dopamine will be released, which stresses the PFC (this is the part of your brain that you have control over, "No, I don’t want to eat cookies; I am dieting”), causing a loss of inhibitory control and creating a positive feedback loop (Volkow, N. D., 2010).

Furthermore, long-term exposure causes the brain to become “desensitized” to tasks that would produce dopamine in sober individuals. Meaning that, when the drug is consumed, the large release of dopamine teaches the limbic system to only seek whatever creates that reward system. Naturally, the point of the limbic system is to keep us alive. For example, if an individual finds food while hungry, dopamine is released, and the memory circuits remember that finding food while hungry increases dopamine. Whereas, when individuals create powerful dopamine connections to drugs, that connection is so powerful that normal tasks no longer produce the dopamine they once did. This is due to a lack of available type 2 dopamine receptors over time and reduced activity in the PFC.

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 suggests 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., 1999). 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, 2006 Sep.).

One significant study conducted by Volkow et al. (2006, Jun.) 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 (2003) 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.

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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 Sycle 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 stubbily. “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.