Scientific Research Publishing

World Journal of Neuroscience, 2023, 13, 151-159
https://www.scirp.org/journal/wjns
ISSN Online: 2162-2019
ISSN Print: 2162-2000

A Brief Review of the Relationship between Addiction and Memory Systems

Kevin Patrick Barman

Drug Studies/Human Services, Rio Hondo College, Whittier, USA
Email: kbarman@riohondo.edu

How to cite this paper: Barman, K.P. (2023) A Brief Review of the Relationship between Addiction and Memory Systems. *World Journal of Neuroscience*, **13**, 151-159.
https://doi.org/10.4236/wjns.2023.133010

Received: May 17, 2023
Accepted: August 18, 2023
Published: August 21, 2023

Abstract

This essay will reexamine research on the relationship between human memory and addiction. This paper will review several studies that discussed how memory systems in the human brain are involved in the acquisition of behavior that is learned and is associated with the development of drug addiction and drug relapse. Additional information reveals that when individuals make the transition from recreational drug or impulsive use to compulsive drug abuse, which may result in a neuroanatomical change in areas of the brain from cognitive control guided by the hippocampus/dorsomedial striatum towards conditioned control of behavior managed by the dorsolateral striatum (DLS) [1]. This review also looked at studies that involved experiments with humans and lower animals, which suggested that the hippocampus mediates a cognitive/spatial type of memory, while the dorsal striatum manages stimulus-response (S-R) habit memory, and the amygdala governs the classical conditioning form of learning and stimulus-affective-associative relationships [1]. Overall, these studies utilize the hypothesis of the memory systems view of addiction, and the involvement of learning and memory in the context of drug addiction, which was proposed by them [2]. This theory has been proposed in response to drug addiction research and includes alcohol, amphetamine, and cocaine [1]. The research also explains how stress and anxiety can play a role in how strong emotional excitement can lead to dependent habit memory in rodents and humans [1].

Keywords

Drug Abuse, Drug Addiction, Learning and Memory, Memory Systems

1. Introduction

Throughout the history of psychological and psychiatric science, a great deal about the human brain has been discovered through the study of how addiction

affects the brain, along with disease, damage or injury (*i.e.*, dementia, epilepsy, Parkinson's disease, traumatic brain injury) to the brain as well, which includes extensive knowledge of the structure and function of human memory systems. Therefore, it is important that we continue to study the impact that the process of drug addiction has on human memory systems. Especially how it may activate or deactivate different memory systems that lead to or help support addictive behaviors.

The main theme of this research proposes that drug addiction and drug relapse are influenced by changes in the human memory during the process of addiction. It also proposes that alterations in the hippocampus/dorsomedial striatum area of the brain are made, as individuals go through the process of addiction from recreational or impulsive drug use to compulsive drug abuse. In particular, Goodman & Packard [1] reveal that alterations in the hippocampus/dorsomedial striatum are made through the development of neuroanatomical changes from cognitive control directed by that part of the brain, where it is then conditioned to control behavior by the (DLS). While White [3] also looked at studies that involved experiments with humans and lower animals, which suggested that the hippocampus mediates a cognitive/spatial type of memory, where the dorsal striatum manages stimulus-response (S-R) habit memory and the amygdala governs the classical conditioning form of learning and stimulus-affective-associative relationship. The focus of this paper is on memory and drug addiction, which primarily uses the Multiple Memory Systems View of Addiction approach [2], to explain the relationship of how drug addiction is learned and thereby makes alterations in parts of the brain that are implicated in memory.

2. Multiple Memory Systems View of Addiction

White [3] suggested that drugs can become reinforcers, just like goods or water in a learning task experiment and enhance relationships of drug-related context, stimuli behavior that encourage drug consumption, and later drug addiction.

The hypothesis of the "Multiple Memory Systems View of Addiction", included results of previous studies that the memory of both lower animals and humans is controlled through autonomous neural systems. According to White [3], the hippocampus mediates a cognitive/spatial type of memory, while the dorsal striatum manages stimulus-response (S-R) habit memory, and the amygdala governs the classical conditioning form of learning and stimulus-affective-associative relationships. Where the amygdala, dorsal striatum, and hippocampus encode special parts of memories associated with drugs. As can be seen in (**Figure 1**), the hippocampus encodes conscious information associated with cues and situations (*i.e.*, stimulus-stimulus associations) related to drug experiences [2]. Another important distinction is that even though the hippocampus does not encode behavioral responses to drug cues, it does use information that signals specific behaviors to obtain the reinforcement of a drug [2]. While the dorsal striatum encodes relationships with drug-related behaviors and stimuli associated with

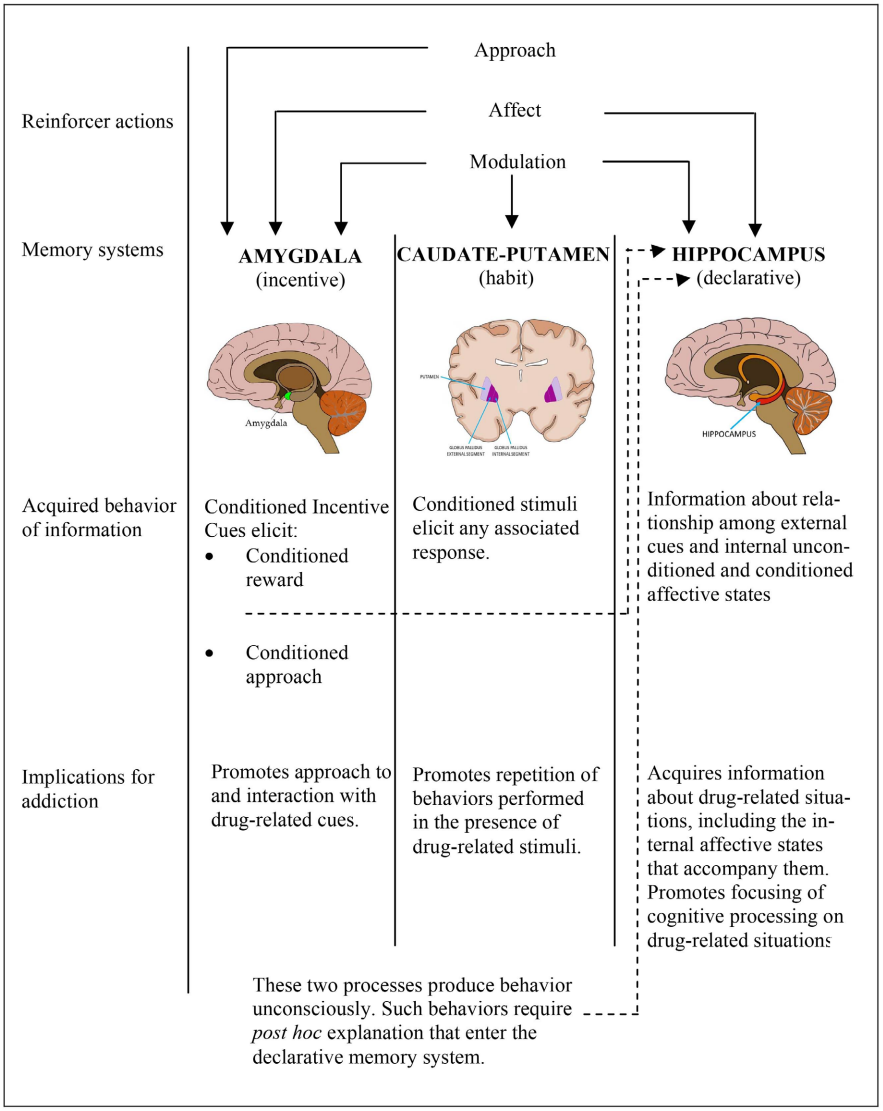

Figure 1. White's (1) multiple memory systems view of drug addiction. (Report from White with permission from John Wiley & Sons.)

drugs [2]. It can lead to the development of drug-associated cues, which may trigger automatic behavioral responses that lead to drug abuse behaviors [2]. It is in the amygdala, where classical conditioning learning takes place that provides neutral cues in the environment, which becomes related to some type of drug reward [2]. This is where the subject animals of these studies, later responded to these cues that they learned, not unlike the way they previously responded to the drug [2]. In particular, the cues that are learned trigger emotional reactions, involve internal emotional states and conditioned behaviors towards or in avoidance of a conditional cue [2].

In **Figure 1** below, White [3] explains how drugs that people become addicted to contain properties of natural reinforcement. Those drugs that may be addictive contain multiple qualities of reinforcement, which can cause positive or negative emotions, approach, and the control of memory systems [2]. Further,

White [3] discusses how the amygdala, caudate putamen (*i.e.*, dorsal striatum), and hippocampus control memory systems that are related, while every part of the system of memory more than likely encodes specific areas of memories associated with drugs. In addition, White [3] reports that due to the memory modulatory characteristics of drugs that are addictive, when individuals engage in the self-administration of drugs, they strengthen the connection of those memory systems associated with drug-related memories. Which are encoded by the amygdala, hippocampus, and dorsal striatum [2].

3. Recreational/Impulsive Drug Use and Compulsive Drug Abuse

Goodman & Packard [1] discuss how based on learning experiments, subjects (rodents) routinely engage in goal-directed behavior when they are trying to solve a task. The authors also reveal, that after complex instruction, their behavior becomes automatic and occurs involving very little attention, intention, or cognitive effort, then becomes a "habit" [1]. This revealed evidence of the shift from cognitive control behavior to habit by the subject rodents [1]. While it is the neuroanatomical shift from cognition to habit happened in the instrumental learning experiments the rodents participated in [1]. They further explain how the original cognitive control of behavior in the instrumental learning experiment is guided by the hippocampus and dorsomedial striatum (DMS), while the habitual responding is guided by the DLS [1].

Based on these experiments previously mentioned and results obtained by other researchers [1] suggest that the neuroanatomical shift to habit memory in rodent learning experiments might be able to explain the transition from recreational (impulse) drug use to compulsive drug abuse. In addition, Goodman & Packard [1] report that other researchers have found results for a variety of drugs of abuse are guided by the DMS in response to goal-directed responding to drug reinforcement and the DLS guides in habitual responding to drug reinforcement.

Therefore, since some drugs have a high risk of abuse (*i.e.*, amphetamine, cocaine…), researchers propose that drugs that are addictive may strengthen DLS-dependent memory function and hasten the shift from cognitive to habitual control of behavioral responses [1].

Another important detail to mention regarding memory is that addictive drugs strengthen habit memory directly by strengthening the function of the DLS [1]. In addition, it may be possible that abusive drugs strengthen habit memory inadvertently by other memory systems [1]. The suggestion here is that in some experiences of learning, systems of memory compete for control of learning and that if the function of one system is impaired, another memory system may be strengthened [1].

The consumption of drugs can be related to both impulsivity and compulsivity, which can lead to psychopathology along with a vast array of substance use and psychiatric disorders [4]. It seems that not every person who consumes a drug becomes addicted. What leads to us asking the obvious question of, is it the

cause for those who do become addicted? It appears that there are various drugs that are inherently more likely to lead to addiction than others. In some cases, an individual has less impulse control intrinsically or are they born with a defective mesolimbic dopamine reward system. It has been suggested that 40% - 60% of addictions are inherited, which is caused by genetic variants which can change fundamental neurobiological processes, and there is evidence of similar pathways, (e.g., mesolimbic dopamine reward system) to separate addictions [2]. In fact, the combination of a defective mesolimbic reward system and diminished impulse control could lead to susceptibility to drug abuse and misuse. Thereafter, if a person begins to consume drugs repeatedly, the impulsiveness of their drug use may lead to the inclusion of their habit system in some persons more expediently than in others ones. It can activate neuroplasticity in the circuit related to compulsive behavior that may be responsible for their drug consumption developing into a compulsive behavior [4].

How can diminished impulse control and compulsive behavior be reduced or eliminated in substance abuse and use disorders? The solution may be found in dysfunctional cortical circuits that routinely control those behaviors [4]. It has been proposed that both impulsivity and compulsivity may be neurobiological instincts that are "bottom-up", where impulsivity originates from the ventral striatum, and compulsivity emanates from the dorsal striatum, along with other parts of the prefrontal cortex working as a "top-down" to squash those actions [5]. The basic idea behind the difference between bottom-up and top-down cognitive control or processing is the hypothesis that for bottom-up processing the stimulus that our perceptions are exposed to form them [5]. While for top-down processing, the foundations of an individual's experience and probabilities are used to explain an understanding of a stimulus they are presented with, in terms of cognitive control [5].

Thereby suggesting that inhibitory voluntary control is directed by top-down cortical structures where impulsivity and compulsivity may be the result of the easing of this control. Based on this hypothesis of impulsivity and compulsivity, behavioral productivity is guided by the interaction between neurobehavioral structures [4]. While it is the outcome of the equilibrium among the "top-down" and "bottom-up", where impulsivity and compulsivity occur because of a defect in the response inhibition structures (*i.e.*, insufficient top-down cognitive regulation) or caused by too much stress emanating bottom-up from the ventral striatum for impulsivity or the dorsal striatum in the case of compulsivity [5].

A person who has developed some type of drug addiction is very unlikely to be able to maintain cognitive and conscious control of their decision-making [4]. Due to the fact that they have learned to make choices by default to the habit of consuming drugs, rather than making a cognitive choice or decision to use or not use a drug [4], in response to an already learned and practiced behavioral to an environmental stressor [4]. That has already been encoded in their memory system [4]. Therefore, it is necessary to differentiate between bottom-up processing and top-down processing to determine the potential nature of cognitive control and

the maintenance of an addiction [4].

The neuroanatomical areas for impulsivity and compulsivity appear to interact in separate neuronal circles [4]. Where impulsivity is an action-response dependent on the learning system and compulsivity located dorsally is a habit system [4]. It seems that many behaviors begin as impulses located in the ventral loop of the motivation and reward system [4]. Later, some of those behaviors move in a dorsal direction because of a flow of neuroadaptations and neuroplasticity which connect with the habit system where an impulsive action develops into a compulsive act [4]. The coils of data are transferred from one neuronal loop to the next and seem to include regulatory direction from the amygdala, hippocampus and other parts of the prefrontal cortex [4].

Ventral-to-dorsal migration is very common during the process of an individual becoming addicted to a drug [4]. Even though it is believed that the onset of drug use is voluntary and connected to some type of impulsive trait, inevitably they lose voluntary control over their pursuit of drugs and their impulsive drug-taking behavior progresses into a compulsion [4]. The impulse to consume drugs or engage in behaviors that primarily cause "euphoria" leads to a sense of pleasure and enjoyment [4]. If this does not occur on a frequent basis, where it does not produce neuroplasticity surges from the ventral to the dorsal area, it will remain under the control of that individual [4]. It is also true that engaging in impulsive drug consumption or impulsive behaviors too often can develop into compulsive behaviors caused by the need to avoid the painful symptoms of withdrawal that happen over the course of their drug consumption and behavior that occurs over time [4]. It is also true that people who have a history of behavioral or drug addictions may experience arousal and tension as they anticipate engaging in the behavior [4]. It will lead to a dysphoric disposition, if they are not able to consume the drug or perform the addictive behavior [4]. Later, the pleasure and reward that the drug/behavior originally initiated, fades with time, possibly leading to the necessity of increased amounts or frequency of the drug use to reach the same effect [4].

It is often believed that the first use of a drug will always be the most reinforcing and rewarding experience and sensation [4]. Unfortunately, people do not pursue pleasurable and rewarding behaviors only one time. Thereafter excessive impulsivity can trigger the acquisition of compulsions, which can lead to over-dependence on habit learning [4]. This enhanced habit creation might cause the development of habits and compulsions for individuals who express excessive impulsivity [4]. Whereby compulsions are expressed by a recurrence of maladaptive addictive behaviors [4]. This results in a person's conditioned compulsion developing into a compulsive habit that is beyond the reach of their cognitive control over the maladaptive drug-seeking behavior [4]. This phenomenon of differentiating between impulsive drug use and compulsive drug use helps draw a connection between the selection of habit-learned behaviors as opposed to cognitive selection, which have altered the brain's memory systems caused by drug abuse and drug addiction [4]. Additional information reveals

that when individuals make the transition from recreational/impulsive drug use to compulsive drug abuse, may result in a neuroanatomical change in areas of the brain from cognitive control guided by the hippocampus/dorsomedial striatum towards conditioned control of behavior managed by the dorsolateral striatum (DLS) [4].

4. Influence of Stress and Anxiety

Psychosocial stressors like stress or anxiety can strengthen habit memory and become a cue or trigger for drug abuse [1]. It has also been suggested that daily life situations that lead to chronic prolonged periods of stressful or anxious life experiences are related to increased vulnerability to addiction to drugs and drug relapse in human beings [1]. According to Goodman & Packard [1], additional researchers have discussed how emotional arousal influences multiple memory systems and continual exposure to stress may strengthen drug addiction and relapse in human beings by participating in the DLS-dependent habit memory process [1]. A good example of this is when they studied individuals who had a cocaine dependency and were exposed to stress, later they discovered that stress in cocaine-dependent individuals was related to a reduction in blood-oxygen- level-dependent activity (BOLD) in the hippocampus and increased activity in the dorsal striatum [1]. And these BOLD activity changes were related to stress- induced cocaine cravings [1].

The influence of emotional arousal on behavior has been studied extensively, with a specific emphasis on the effects of anxiety and/or stress on learning and memory processes [6]. It is well known that an individual's experience in their environment and their exposure to stress can shape their memory for the rest of their lives, whether it was traumatic or triumphant. It appears that the memory systems of the brain are designed to support human behavior [7]. For instance, a negative childhood experience like being bitten by a small snake can lead to a long-standing declarative memory for the episode as well as a long-term no declarative fear of snakes (phobia) that is associated with a personality feature rather than a memory [4]. It is part of a clinician's training to identify psychosocial stressors (*i.e.*, physical illness, poverty, trauma, unemployment...) that may cause stress and/or anxiety to interfere with an individual's ability to function in a healthy way. It has been demonstrated that exposure to stressful life circumstances diminished the volume of grey matter located in the right hippocampus of human beings [2]. While exposure to chronic stress may effect change in several memory systems, it can change the comparative volume of the dorsal striatum and hippocampus [1].

It has been found in some case studies of human beings that the experience of acute or chronic anxiety, may be the basis for various anxiety-related psychiatric disorders and symptoms (*i.e.*, substance use disorder (SUD), obsessive-compulsive disorder (OCD), and post-traumatic stress disorder (PTSD) with habitual traits expressing behavioral qualities [1]. For example, someone who is suffering from OCD may reduce their germ fears by habitually washing their hands. While

people who suffer from symptoms of PTSD may acquire a non-context particular cued recollection of a traumatic memory. For instance, if they experienced a traumatic car accident driving on a freeway, the signal cue for driving on a freeway may trigger anxiety, which leads to the habit of avoiding the freeway. In regard to addiction, anxiety may lead to continued use of the drug or relapsing back into addiction. Due to the fact that they rely on memory and memory cues based on their habit, which guides their maladaptive drug using and seeking behavior [1], which could result in their experiencing anxious symptoms or the expression of an anxiety disorder.

5. Conclusion

Overall, this research argued how the transition from recreational or impulsive drug use to compulsive drug abuse can be understood through the study of how human and lower animal memory systems of the brain are altered throughout the process [3]. It can be explained by White's multiple memory systems approach to drug addiction [4], where it indicates that the hippocampus facilitates contextual control of the self-administration of a drug, while the DLS facilitates the S-R habitual responding for drug reinforcement, and the amygdala facilitates drug seeking that is learned [4]. This phenomenon of discerning between impulsive drug use and compulsive drug use helps draw a link between the selection of habit-learned behaviors as opposed to cognitive selection, which have altered the brain's memory systems caused by drug abuse and drug addiction [5]. It is equally important to study the impact that stress and anxiety have on the memory systems that are involved in triggering drug use and relapse as well. In the future, it would be very helpful to continue with this research. In order to find more evidence of how drugs hijack human neural networks and alter human memory systems.

Conflicts of Interest

The author declares no conflicts of interest regarding the publication of this paper.

References

[1] Goodman, J. and Packard, M.G. (2016) Memory Systems and the Addicted Brain. *Front Psychiatry*, **7**, 24. https://doi.org/10.3389/fpsyt.2016.00024

[2] Papagni, S.A., Benetti, S., Arulanantham, S., McCrory, E., McGuire, P. and Mechelli, A. (2011) Effects of Stressful Life Events on Human Brain Structure: A Longitudinal Voxel-Based Morphometry Study. *The International Journal on the Biology of Stress*, **14**, 227-232. https://doi.org/10.3109/10253890.2010.522279

[3] White, N.M. (1996) Addictive Drugs as Reinforcers: Multiple Partial Actions on Memory Systems. *Addiction*, **91**, 921-949, Discussion 951-965. https://doi.org/10.1046/j.1360-0443.1996.9179212.x

[4] Robbins, T.W., Ersche, K.D. and Everitt, B.J. (2008) Drug Addiction and the Memory Systems of the Brain. *Annals of the New York Academy of Sciences*, **1141**, 1-21. https://doi.org/10.1196/annals.1441.020

[5] Stahl, S.M. (2013) Stahl's Essential Psychopharmacology: Neuroscientific Basis and Practical Applications. 4th Edition, Cambridge University Press, Cambridge.

[6] Koob, G.F. and Volkow, N.D. (2016) Neurobiology of Addiction: A Neurocircuitry Analysis. *Lancet Psychiatry*, **3**, 760-773. https://doi.org/10.1016/S2215-0366(16)00104-8

[7] Squire, L.R. (2004) Memory Systems of the Brain: A Brief History and Current Perspective. *Neurobiology of Learning and Memory*, **82**, 171-177. https://doi.org/10.1016/j.nlm.2004.06.005