PERSPECTIVE

Stress and addiction

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ABSTRACT

The great frequency of drug usage and addiction, as well as the cost to society, are undeniable. However, its classification as a brain disease is debatable, and the present treatments are insufficient. Stress and drug addiction research could help to bridge the gap and produce more effective therapies.

Drug addiction has been regarded as a chronic and relapsing brain disorder characterised by obsessive drug seeking and taking for the past two decades. Addiction, according to another viewpoint, is generated and sustained by psychosocial circumstances and learning processes that transform them into addiction, rather than being a brain illness. The question of whether the brain or the context is the most significant level of analysis for understanding and approaching addiction is still debated today. In any case, the frequency and burden of drug abuse and addiction on society are accepted, regardless of how drug addiction is defined. Only harmful alcohol usage results in more than 3 million deaths each year, or 6 deaths every minute. (According to the World Health Organization, 2018).

Key Words: *Opiod Addiction; Brain illness; Drug addiction; Psychological stress; Psychology*

INTRODUCTION

The study of drug addiction has gotten a lot of attention from neuroscientists. Animal models, which cannot fully mimic the human state, and neuroimaging research in humans have been intensively exploited to identify the neurological basis of drug addiction as a brain illness.

Surprisingly, psychological stress (hereinafter stress) has proven to be a great model for capturing how complicated social circumstances influence health, and it may help to design more effective explanations, interventions, and public policies in the area of drug addiction [1]. However, the neurobiological mechanisms underlying stress's impact on drug addiction are unknown.

Stress is triggered "when an individual feels that external demands tax or exceed his or her adaptive capability," hence the brain plays a key part in the perception of threat and the initiation of the stress response. Three main stress hormones mediate the stress response: Corticotropin-Releasing Factor (CRF) and cortisol (corticosterone in rodents) released by the Hypothalamic–Pituitary–Adrenal (HPA) axis and adrenal cortex; catecholamines, and norepinephrine or noradrenaline) released by the adrenal medulla and sympathetic nerves [2]. Stress hormones also give the brain input, controlling the HPA axis' activity. This negative feedback loop is dependent on the activation of two types of glucocorticoid receptors in the brain: high-affinity mineralocorticoid receptors, which are activated by lower doses of cortisol and prevent further release of CRF; and low-affinity glucocorticoid receptors, which are activated by higher doses of cortisol and cause an increase in CRF release.

Stress hormones have been shown to have negative effects on the limbic system's hippocampus and amygdala in previous studies: Acute stress improves memory formation in the hippocampus, whereas persistent and/or severe stress interferes with memory formation, resulting in fragmented declarative memories or missing contextual elements. Acute and even mild stresses, on the other hand, boost amygdala activity by attaching emotional importance to memories, thereby activating the locus coeruleus and triggering the traditional fear/anxiety response. Stress reduction causes anatomical alterations in the amygdala, as expected [3]. According to new research, the Bed

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Nucleus of the Stria Terminals (BNST), which is located in the central section of the amygdala, may store emotional memories that are implicated in long-term adverse stress reactions. The consequences of stress on the prefrontal cortex have gotten a lot of attention in recent years (PFC). Stress hormones impede executive functioning in the PFC, which are important for turning off the stress response once the threat has passed. Control inhibition (self-control, resisting impulsive behaviour), interference control (selective attention and cognitive inhibition), working memory, and cognitive flexibility are examples of executive functions [4].

In conclusion, the effects of stress on the limbic system strikingly reflect differences between the hippocampus and the amygdala, highlighting the amygdala's dominance over the hippocampus in enhancing implicit emotional learning and memory, particularly fear conditioning, while disrupting explicit learning and memory; the effects on the PFC aggravate the situation further, impairing the executive functions required for slow-rational decision-making based on good inhibtion.

A compulsion to seek and consume the substance, a loss of control in limiting intake despite severe effects, and the appearance of a negative emotional state when access to the drug is denied are all symptoms of drug addiction. Because it is a chronic, relapsing problem for the majority of people, similar to diabetes or hypertension, the gold standard for therapeutic success would be the management of drug use during lengthy periods of abstinence with periodic relapses [4,5]. However, it is unclear if chronicity is a hallmark of drug addiction or a result of a lack of effective treatments.

The stage of bingeing/intoxication is the dopamine reward system has long been linked to the first positive reinforcing impact of medicines. Most psychostimulant substances of abuse stimulate dopamine D1 receptors in the nucleus accumbens' mesolimbic pathway, while inhibiting D2 receptors in the striatocortical pathway in the PFC [6]. As a result, drugs and drug-related cues (wanting) or yearning are given a larger incentive value (reward) and salience, raising the risk of bingeing and intoxication. D2-agonists, like psychostimulants, consistently provide positive reinforcing effects.

First, opioids such as heroin, morphine, and endogenous endorphins (endorphin) have positive reinforcing effects that are directly mediated by their action on receptors. Opioid antagonists like naloxone and naltrexone, for example, block the rewarding effects of opioids. Second, the endocannabinoid system, which is also implicated in the reinforcing effects of natural rewards, mediates the positive reinforcement effects of cannabis.

Stage of negative impact Drug usage causes neuroadaptations, or long-term brain alterations, which might lead to a bad emotional state or withdrawal symptoms. These neuroadaptations, according to the allostatic hypothesis of addiction, involve dynamic readjustments toward a new set point, gaining stability through change rather than just returning to homeostasis. When this is accumulated over time, it increases the likelihood of addiction and relapse. An early neuroadaptation involves the down-regulation of the dopamine reward system (also known as withinbrain reward system neuroadaptation), which reduces the availability and responsiveness of the D2 receptors in the nucleus accumbens and modifies the reward threshold, resulting in an inability to experience pleasure with natural reinforcers (anhedonia) and increasing the risk of drug intake escalation [7,8]. Changes in the cortico-striatal glutamate system also increase reactivity to drug-related stimuli and unpleasant emotional states while decreasing sensitivity to non-drug rewards.

The recruitment of brain anti-reward systems (also known as between-system neuroadaptation) is a later neuroadaptation triggered by CRF, dynorphin, and hypocretin (orexin) hormones. CRF would be responsible for an early dysregulation of the HPA axis, as well as a later dysregulation of the extra-hypothalamic system in the extended amygdala, both of which would result in an unpleasant negative emotional state. CRF antagonists, on the other hand, prevent negative emotional states brought on by medication withdrawal. On the other hand, activation of dynorphin-aversive opioid system receptors is also responsible for producing a negative emotional state in the extended amygdala by lowering reward system dopamine activity and affecting executive functioning in the PFC. Second, anti-craving medications such as naltrexone are antagonists of dynorphin-k receptors. Third, via influencing the activity of the HPA axis and the extended amygdala, hypocretin (orexin), which regulates arousal and appetite, may also be implicated in producing a negative emotional state and reward-seeking [3]. However, the action of at least four components, including agonist opioids, endocannabinoids, neuropeptide Y, and finally, oxitocin, which are implicated in reward, social attachment, and bonding, may moderate (buffer) this negative emotional state associated with withdrawal.

In summary, drugs "hijack" the brain's reward, anti-reward, and prefrontal systems, causing neuroadaptations that play a role in the ubiquitous transition from drug abuse to addiction, which gets worse with time.

Stress plays a critical role in the shift from drug usage to addiction, according to a growing body of studies. Similar to other chronic illnesses like hypertension, diabetes, or obesity, the path to drug addiction is now best understood as the outcome of an accumulation of allostatic alterations. This is significant because allostatic changes, which go beyond a simple return to the initial homeostatic state, are a major source of allostatic (over)load, resulting in brain changes that lead to a progressive imbalance between states of opposite hedonic valence (positive and negative), increasing the risk of addiction. Interestingly, chronic stress-induced brain alterations mediate and overlap drug-abuse-induced brain changes, allowing for a better understanding of the three steps involved in the shift from drug use to addiction.

For starters, persistent stress and drug misuse both cause the brain reward system to degrade or malfunction. In the case of drug misuse, the over-activation of the brain reward system, which is fueled by positive reinforcement during the binge/intoxication stage, is the direct cause. During the binge/intoxication stage, this down-regulation is engaged in the sensation of reward-craving generated by the exposure to drugs or drug-related signals. The key to understanding the stress-like state of the negative emotion/withdrawal stage, driving drug-seeking and taking through negative reinforcement, is to understand how stress exposure and drug abuse result in the progressive up-regulation or excess of the brain stress system (till now referred to as the "anti-reward" brain system), which is the key to understanding the stress-like state of the negative emotion/withdrawal stage, driving drug-seeking and taking through negative reinforcement [7,8]. The increase in responsiveness

of the HPA axis and amygdala, as well as increased hypersensitivity to stress, causes this up-regulation. As a result, it is implicated in the relief-craving process. Furthermore, recurrent drug exposure and withdrawal can be considered stresses in and of themselves, causing the same brain alterations and increasing the likelihood of relapse, which is a hallmark of addiction.

Second, both stress and drug abuse cause down-regulation of the hippocampus, disrupting learning and emotion regulation, as well as the brain's ability to inhibit the HPA axis' reactivity; on the other hand, both stress and drug abuse cause disruption of the PFC, impairing executive functions required not only for self-regulation of negative emotional states, but also for effort-related decision-making, which is required to suppress amygdala activation during the preoccupation/anticipation period [8]. Stress fills the PFC with dopamine and norepinephrine, resulting in a steady loss of functional connectivity within the PFC, undermining the ability to resist relapse in the face of need and aiding the transition to compulsive drug use, which is a defining aspect of drug addiction. In fact, stress has been identified as the single most powerful and consistent cause of seeking and relapse, with higher drug addiction severity and poor treatment outcomes.

CONCLUSION

In summary, stress promotes the motivational salience of drugs and drug-related stimuli, induces a negative emotional state, and impairs executive skills, all of which help to set up and intensify drug addiction. Drug addiction has been labelled a learning condition, a reward deficiency or anti-reward excess disorder, an executive function disorder, and, more recently, an allostatic disorder.

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