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*Research Article*

# Exploring The Link Between Stress and Drug Abuse: A Comprehensive Review

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## Abstract

Stress has long been considered an important factor influencing both the initiation of substance use and the risk of relapse, yet the exact pathways through which it acts remain incompletely defined. Contemporary addiction frameworks propose that stressful experiences can heighten susceptibility to drug use, particularly in individuals with pre-existing vulnerabilities. A substantial body of animal research demonstrates that exposure to stress increases drug-seeking behaviour and enhances the reinforcing effects of addictive substances. Although human studies provide indications that stress elevates craving and contributes to relapse, findings often rely on correlational designs and show considerable inconsistency.

Parallel evidence from developmental research highlights another dimension of this relationship. Prenatal drug exposure and early-life stress appear to affect overlapping neural systems involved in emotional regulation, stress responses, and reward processing. Children exposed to substances in utero often exhibit developmental and neurobehavioral deficits, but the extent to which environmental stress contributes to these outcomes is still unclear. Controlled experiments in animal models show that both perinatal substance exposure and early stress can lead to long-lasting changes in brain structure and neurotransmitter function, potentially increasing later vulnerability to addictive behaviours.

Overall, existing evidence suggests that stress plays a meaningful role in shaping both the emergence and persistence of drug use, while early developmental experiences may further modify this risk. However, key questions remain regarding how these mechanisms operate in humans. A deeper understanding of the interaction between stress, neural adaptation, and addictive behaviour is essential for improving prevention efforts and developing more targeted treatment strategies for substance use disorders.

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## 1. INTRODUCTION

The relationship between stress and drug abuse has become an important subject in today's society. Studies from the last five years have found that drug consumption causes changes in behaviour and emotions, and also affects the ability to make decisions. The possibility of consuming addictive substances also increases. At the same time, chronic drug exposure can further disturb the body's natural stress-response mechanisms, creating a cycle where stress increases drug use, and drug use amplifies stress sensitivity.

Recent research has found that stress promotes the initiation of consuming addictive substances, and in people who are already using drugs, the possibility of consuming them again also increasesAccording to research from 2021 to 2024, stress activates the hypothalamic–pituitary–adrenal (HPA) axis, which increases cortisol levels and produces changes in the brain's reward pathway, especially in the dopamine system. These alterations motivate a person to seek pleasurable experiences through drugs, alcohol, nicotine, or other addictive substances. Recent studies have also shown that childhood exposure to violence, family conflict, or stress experienced by the mother during pregnancy leaves long-term effects on brain development, which increases the likelihood of using addictive substances during adolescence or young adulthood.

In clinical studies, several limitations still exist in human research. Differences in stress-measurement methods, the lack of long-term data, and the absence of combined biological and psychological assessment remain major challenges. The purpose of this review is to understand the multidimensional relationship between stress and drug abuse, integrate recent scientific findings, and identify critical gaps for future research.

### 2. Conceptual Framework of Stress:

Understanding "stress" requires a solid conceptual framework, especially when examining its connection with the use of addictive substances. The most widely studied biological pathway of the stress response is the Hypothalamic–Pituitary–Adrenal (HPA) axis, which involves the release of CRH (Corticotropin-Releasing Hormone) from the hypothalamus, the production of ACTH (Adrenocorticotropic Hormone) from the pituitary gland, and the secretion of glucocorticoids such as cortisol from the adrenal glands.

To understand stress, we must recognise that stress is not only a biological function of the body, but also a mental and emotional process. Stress varies from person to person, and it depends on the situation. Stress occurs when a situation arises that is beyond one's control. Every individual understands and reacts to stress in a different manner.

When a person is under stress, their HPA axis becomes activated, and cortisol levels rise. This increase directly affects the brain, mainly influencing the dopamine system. Due to these changes, the individual develops a stronger attraction toward addictive substances, which ultimately increases the tendency for drug use.

Negative emotions, unstable family conditions, financial pressures, and other environmental challenges can significantly elevate a person's stress level, and this prolonged or intense stress can further increase the likelihood of developing drug-seeking behaviour or becoming attracted to addictive substances.

Apart from these factors, early-life stress—such as childhood violence, family conflicts, or stress experienced by the mother during pregnancy—can also impact the developing baby's brain. Such adverse experiences can disrupt the normal functioning of the HPA axis, and as a result, the individual may have a higher likelihood of using addictive substances during adolescence.

### 3. Biological Mechanisms Linking Stress and Drug Abuse:

To understand the relationship between stress and drug use, it is essential to examine the neurochemical and neurological reactions that occur in the body. One of the most critical systems involved is the HPA (Hypothalamic–Pituitary–Adrenal) axis.

When a person experiences stress, the hypothalamus releases CRH (Corticotropin-Releasing Hormone), which stimulates the pituitary gland to secrete ACTH (Adrenocorticotropic Hormone). ACTH then signals the adrenal glands to produce cortisol, the primary stress hormone.

Cortisol significantly affects the brain, particularly the regions involved in decision-making and the experience of reward. These changes can increase vulnerability to drug use and addiction, as individuals may seek substances to cope with or regulate stress-induced emotional states.

Apart from the HPA axis, a major biological pathway that connects stress and drug addiction is the mesolimbic dopamine system, often referred to as the brain's reward pathway. This circuit includes regions such as the ventral tegmental area (VTA), nucleus accumbens, and the prefrontal cortex, which work together to regulate pleasure, motivation, and emotional control. When a person experiences stress, the body releases cortisol, which increases dopamine activity in these regions. As a result, the pleasurable effects of drugs become stronger, motivating individuals to use substances repeatedly. With continuous exposure, the brain's natural reward response becomes weaker, and the individual begins to depend on drugs to feel pleasure, reinforcing addictive behaviour.

Stress also disrupts the balance of other important neurotransmitters, including serotonin, GABA, and glutamate, which are essential for mood stability and decision-making. Low serotonin levels may lead to depression and anxiety, encouraging people to use substances for temporary emotional relief. Likewise, disturbances in glutamate pathways impair learning and memory, causing the brain to develop strong links between stress and drug use.

In addition, chronic stress combined with long-term drug use can trigger neuroinflammation, damaging neurons and impairing communication between brain areas. This reduces emotional control and increases the likelihood of relapse. Therefore, understanding these biological processes is crucial for developing effective treatments such as pharmacotherapy, psychological counselling, and stress-management strategies.


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**3.1 Effect on Dopamine and Reward Pathways:**

Chronic stress can significantly affect the brain's dopamine pathways, which are responsible for regulating motivation and reward. When cortisol levels rise due to prolonged stress, it alters dopamine release and dopamine sensitivity in the brain.

Long-term continuing stress not only increases drug consumption but also changes dopamine receptors. Long-term stress changes the dopamine receptors, while the brain needs more stimulation to feel happy. Because of this, normal things like talking with friends, playing, and completing hobbies become difficult to find happiness in. Then the person consumes drugs for happiness. This is called reward deficiency. It forces the person to take drugs at a high level, which increases the addiction day by day. As a result, the reward experienced from drug use may feel more intense and satisfying, making the individual more vulnerable to addiction. This heightened reward response increases cravings and the likelihood of repeated drug use as a coping mechanism for stress.

**3.2 Impact on Prefrontal Cortex and Decision- Making**:

Stress weakens the efficiency of the prefrontal cortex, which is responsible for self-control and preventing risky behaviour. When stress affects this region, impulsive behaviour increases, and the likelihood of substance use also rises

If stress persists for a long time, the prefrontal cortex becomes weak, which means the brain cannot make correct decisions, the person cannot think deeply, and self-control also decreases. At the same time, the amygdala — which controls fear and anger — becomes more active, and because of this imbalance, emotions become intense. The person then uses drugs to get relief. In a stressful state, this is what happens: drug use, relapse and the possibility of making wrong decisions."

Chronic stress also alters the communication between the prefrontal cortex and other brain regions involved in reward and motivation, such as the nucleus accumbens. When this connection becomes weakened, the brain finds it difficult to think about long-term outcomes and instead focuses on immediate comfort. Because of this shift, addictive substances start to appear like the quickest way to escape emotional pain, even when individuals are aware of the negative consequences.

Stress further interferes with executive functions such as attention, planning, problem-solving, memory, and judgment. As these abilities decline, a person becomes more reactive and emotionally driven instead of thinking logically. This increases their sensitivity to external triggers like peer influence, exposure to drugs, or stressful social environments, making relapse more likely—even after attempts to quit.

With time, the repeated cycle of stress and substance use forms automatic habits, where drug-seeking behaviour becomes unconscious rather than a deliberate choice. At this stage, addiction is driven by neurobiological changes rather than personal control. Therefore, strengthening the functioning of the prefrontal cortex through therapies, stress-management techniques, and behavioural interventions is essential for preventing addiction and supporting long-term recovery.

**3.3 Neuroplasticity and Long-Term Brain Changes**:

Prolonged stress can lead to structural changes in the brain, especially in areas responsible for fear response, emotional regulation, and coping ability. When stress affects these regions, a person may lose the capacity to effectively deal with emotional challenges and become more sensitive to the effects of addictive substances.

Long-term exposure to stress does not just influence brain chemicals; it also causes permanent physical and functional changes in brain structure through a process known as maladaptive neuroplasticity. Neuroplasticity refers to the brain's ability to reorganise and modify neural pathways based on experience. Under healthy conditions, neuroplasticity supports learning, memory formation, and emotional control. However, when stress becomes chronic, neuroplasticity shifts in a harmful direction, weakening connections in regions responsible for self-control and strengthening pathways related to fear and reward-seeking behaviour.

Chronic stress leads to the shrinking of neurons in the prefrontal cortex and reduces the formation of new neurons in the hippocampus, an area involved in memory and emotional balance. At the same time, it increases neural growth in the amygdala, making emotional reactions stronger and more intense. Due to these changes, individuals develop a greater sensitivity to stress and reduced ability to manage emotions, increasing the risk of using addictive substances for relief.

**4. Behavioural and Psychological Pathways**:

The relationship between stress and drug misuse is not limited to biological mechanisms alone; it also involves several behavioural and psychological pathways. When a person experiences stress, their decision-making ability becomes impaired. Negative emotional states such as anxiety, sadness, anger, hopelessness, and loneliness can drive individuals toward drug consumption as a temporary source of relief. This is why many people turn to substances as a way to cope with stress.

Prolonged stress weakens the efficiency of the prefrontal cortex, which normally helps regulate impulsive behaviour. In addition, stress activates the brain's reward pathway, making addictive substances appear more comforting and pleasurable—even though their effects are short-lived and harmful. Overall, these behavioural and psychological routes show that stress increases a person's craving for drugs, impulsivity, and tendency to make risky decisions, which ultimately deepens the risk of substance abuse and relapse.

Stress and drug use are strongly connected, and much of that link comes from the way people try to handle emotional pain. When someone lacks healthy coping tools—like problem-solving skills, emotional stability, or supportive relationships—they often turn to avoidance, trying to escape rather than face difficulties. In those moments, drugs can appear to offer fast relief because they change brain chemistry and briefly numb the pain. That short feeling of comfort convinces the brain that substances are a solution, which increases the risk of becoming dependent.


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The Self-Medication Hypothesis describes this clearly: many individuals use alcohol, opioids, or stimulants to quiet emotional suffering and reduce tension. But instead of reducing stress, drugs eventually make it worse. Over time, they damage brain circuits, increase withdrawal discomfort, and make emotions harder to control. This creates a damaging cycle—stress pushes a person toward drugs, and drug use adds even more stress.

A person's environment also shapes this cycle. Growing up surrounded by conflict, poverty, abuse, academic pressure, or negative peers can leave someone emotionally fragile. Without guidance or support, they may begin to feel hopeless and powerless—a mindset known as learned helplessness—and start believing that drugs are the only escape.

Additionally, the brain learns to associate certain people, places, or feelings with drug use. Because of this conditioning, emotions like sadness, loneliness, or anxiety—or even familiar environments—can instantly trigger cravings. That's why therapies such as Cognitive Behavioural Therapy (CBT), mindfulness practices, and stress-management training are essential. They help people break harmful patterns, build healthy coping skills, and move toward long-term recovery.

**5. Evidence from Animal Studies**:

Over the past several decades, research has revealed that the relationship between stress and drug abuse is not limited to humans—it is a deep biological process observed in animals as well. Studies conducted on rats show that chronic stress significantly increases the self-administration of addictive substances such as cocaine, nicotine, alcohol, and opioids. To induce stress in animal models, researchers use various methods, including social defeat stress and foot shock stress. Across these models, it has been consistently observed that stress activates the mesolimbic dopamine pathway, which enhances the tendency for reward-based drug intake.

Further research has shown that stress strongly activates the hypothalamic-pituitary-adrenal (HPA) axis, leading to elevated cortisol levels. These hormonal changes affect the brain's reward and motivation systems, causing animals to repeatedly press drug-lever mechanisms and consume higher doses of addictive substances.

In some experiments, when animals are deprived of drugs for a few days and then exposed to stress, they exhibit drug-seeking behaviour again—closely resembling relapse patterns seen in humans.

These animal studies suggest that stress not only increases drug use but also rewires the brain in a way that heightens the risk of addiction and relapse. This highlights the profound impact of stress on neurobiological pathways associated with substance abuse.

**6. Evidence from Human Studies:**

Numerous clinical studies on humans have found that individuals who experience persistent psychological, social, or financial stress are significantly more likely to use substances such as alcohol, nicotine, and opioids.

Several longitudinal studies have revealed that people exposed to childhood trauma—including domestic violence, neglect, parental conflict, or emotional insecurity—are at a much higher risk of developing substance use disorders later in life. This is primarily because early-life stress has a lasting impact on the HPA axis and neural circuits involved in emotional regulation. These changes increase stress reactivity and alter reward sensitivity, making individuals more vulnerable to addiction.

Clinical research has also shown that stress directly increases craving. When individuals are exposed to stress, their cortisol levels rise sharply, and brain imaging reveals heightened activity in the reward pathways. This is why stress is considered one of the strongest predictors of relapse in addiction recovery.

Additionally, research conducted during the COVID-19 pandemic highlighted that social isolation, economic insecurity, and uncertainty led to a global rise in substance use. Among adolescents and young adults, factors such as academic pressure, family stress, the burden of online education, and unemployment contributed to increased risk-taking behaviour and drug experimentation.

However, human research still faces limitations. These include: Variability in stress measurement standards, Difficulty in controlling confounding variables, Lack of integrated evaluation of biological and psychological factors.

**7. Interaction Between Chronic Drug Use & Stress Response**:

Chronic drug use rewires the brain's reward circuits—such as the amygdala, prefrontal cortex, and nucleus accumbens—in a way that the brain begins to adopt drug use as a coping mechanism instead of normal stress regulation. This condition is known as *stress-induced drug seeking*, where an individual, upon experiencing stress, spontaneously feels drawn toward substance use.

Research has clearly shown that chronic substance use blunts the response of the HPA axis—meaning that during stress, cortisol levels fail to rise adequately. As a result, the ability to manage stress declines, and the individual becomes emotionally unstable, which further drives repeated drug use.

Long-term consumption of addictive substances deeply affects the body's natural stress-response system. Continuous drug intake alters the normal functioning of the HPA axis, leading to either excessive cortisol production or its gradual suppression. Overall, chronic drug use and the stress response form a vicious cycle—where stress increases drug use, and drug use progressively weakens the stress-response system.

Chronic drug use doesn't just affect the body—it deeply changes the way the brain handles stress. Normally, after a stressful situation, the brain is able to calm down and return to balance. But long-term stress and substance use interfere with this ability. This condition is called allostatic load, where the body's stress system stays constantly switched on or becomes completely worn out. When this happens, natural sources of happiness—like spending time with friends or doing hobbies—stop feeling rewarding, and the brain begins to rely on drugs just to feel normal. Even small stressful moments can trigger strong cravings, making it very hard for someone to stay in


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recovery. Chronic drug use also weakens communication between the prefrontal cortex, which helps with self-control and decision-making, and the amygdala, which controls emotional responses like fear and anger. As a result, emotions become harder to manage, impulsive actions increase, and resisting triggers becomes more difficult. Over time, the brain learns to connect stress relief only with drug use, creating a powerful cycle where stress leads to drug use, and drug use leads to more stress.

Withdrawal makes the situation even worse. It brings anxiety, irritability, and emotional discomfort, which activate the stress system again and push the person back toward relapse. Because of this strong cycle, breaking addiction requires support, including stress-management techniques, Cognitive Behavioural Therapy (CBT), mindfulness, and medical treatment to help the brain recover and reduce dependence on substances.

In this way, chronic stress and drug addiction become tightly linked, each making the other stronger, increasing both vulnerability to addiction and the risk of relapse.

## 8. Research Gaps & Future Directions:

Despite extensive research on stress and drug abuse, several important gaps remain. One major challenge is that many human studies are correlational in nature, making it difficult to clearly establish cause-and-effect relationships. Moreover, the tools, scales, and standards used to measure stress vary significantly across studies, leading to inconsistencies in conclusions.

Additionally, very few studies are available that demonstrate how prolonged stress during childhood or adolescence gradually leads an individual toward addiction. Specifically, the neurodevelopmental changes linking early life stress—such as prenatal stress, domestic violence, neglect, and parental conflict—with later substance misuse are still not adequately understood.

Another critical gap is that many studies focus only on psychological or biological aspects, whereas addiction is actually a biopsychosocial phenomenon. Factors like social stress, insecurity, family environment, financial pressure, and coping mechanisms need to be studied together in an integrated framework, which is still lacking.

Multidimensional research that simultaneously uses neuroimaging, hormonal analysis, and behavioural assessment is currently limited. This makes it difficult to understand how, during stress, the brain's reward pathways, HPA axis, emotional regulation circuits, and decision-making systems interact with one another.

### 8.1 Future direction

To advance research in this field, it is essential that:

- Large-scale longitudinal, multi-site studies are conducted to track patterns over time and across populations.
- A standardised international protocol is developed for measuring stress, ensuring consistency and comparability across studies.
- Research integrates stress, genetics, social environment, and coping strategies to provide a holistic understanding of addiction vulnerability.
- Neuroimaging techniques (such as fMRI and PET scans) and hormonal profiling are combined with behavioural data to map the complex interactions between brain, body, and behaviour.
- Intervention models are designed that simultaneously target stress regulation and addiction treatment, offering more comprehensive and effective solutions.

## 9. CONCLUSION

Available research data clearly demonstrate that stress is a central determinant in the misuse of addictive substances and relapse. Through biological mechanisms such as heightened activity of the hypothalamic-pituitary-adrenal (HPA) axis, alterations in the brain's reward pathways, and impaired decision-making, stress significantly influences the propensity for substance use. Both human and animal studies consistently show that stress increases craving and strongly triggers relapse.

Notably, stress experienced during early life stages—such as childhood abuse, neglect, or family conflict—can have lasting effects on brain development and stress regulation. Such adverse experiences make individuals more vulnerable to addiction at a young age. Therefore, early-life interventions and supportive environments play a critical role in preventing substance abuse.

From a scientific perspective, future research should adopt multidimensional models that integrate biological, psychological, and social factors. In terms of policy-making, priority should be given to programs that reduce socioeconomic stress, expand access to mental health services, and strengthen substance-abuse prevention efforts. Clinically, it is essential to incorporate stress-management therapies, early screening, and relapse-prevention strategies as integral components of treatment.

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