



ASAM

American Society of Addiction Medicine

Public Policy Statement: Definition of Addiction (Long Version)

Addiction is a primary, chronic disease of brain reward, motivation, memory and related circuitry. Addiction affects neurotransmission and interactions within reward structures of the brain, including the nucleus accumbens, anterior cingulate cortex, basal forebrain and amygdala, such that motivational hierarchies are altered and addictive behaviors, which may or may not include alcohol and other drug use, supplant healthy, self-care related behaviors. Addiction also affects neurotransmission and interactions between cortical and hippocampal circuits and brain reward structures, such that the memory of previous exposures to rewards (such as food, sex, alcohol and other drugs) leads to a biological and behavioral response to external cues, in turn triggering craving and/or engagement in addictive behaviors.

The neurobiology of addiction encompasses more than the neurochemistry of reward.¹ The frontal cortex of the brain and underlying white matter connections between the frontal cortex and circuits of reward, motivation and memory are fundamental in the manifestations of altered impulse control, altered judgment, and the dysfunctional pursuit of rewards (which is often experienced by the affected person as a desire to “be normal”) seen in addiction--despite cumulative adverse consequences experienced from engagement in substance use and other addictive behaviors. The frontal lobes are important in inhibiting impulsivity and in assisting individuals to appropriately delay gratification. When persons with addiction manifest problems in deferring gratification, there is a neurological locus of these problems in the frontal cortex. Frontal lobe morphology, connectivity and functioning are still in the process of maturation during adolescence and young adulthood, and early exposure to substance use is another significant factor in the development of addiction. Many neuroscientists believe that developmental morphology is the basis that makes early-life exposure to substances such an important factor.

Genetic factors account for about half of the likelihood that an individual will develop addiction. Environmental factors interact with the person’s biology and affect the extent to which genetic factors exert their influence. Resiliencies the individual acquires (through parenting or later life experiences) can affect the extent to which genetic predispositions lead to the behavioral and other manifestations of addiction. Culture also plays a role in how addiction becomes actualized in persons with biological vulnerabilities to the development of addiction.

Other factors that can contribute to the appearance of addiction, leading to its characteristic bio-psycho-socio-spiritual manifestations, include:

- a. The presence of an underlying biological deficit in the function of reward circuits, such that drugs and behaviors which enhance reward function are preferred and sought as reinforcers;
- b. The repeated engagement in drug use or other addictive behaviors, causing neuroadaptation in motivational circuitry leading to impaired control over further drug use or engagement in addictive behaviors;
- c. Cognitive and affective distortions, which impair perceptions and compromise the ability to deal with feelings, resulting in significant self-deception;
- d. Disruption of healthy social supports and problems in interpersonal relationships which impact the development or impact of resiliencies;
- e. Exposure to trauma or stressors that overwhelm an individual's coping abilities;
- f. Distortion in meaning, purpose and values that guide attitudes, thinking and behavior;
- g. Distortions in a person's connection with self, with others and with the transcendent (referred to as God by many, the Higher Power by 12-steps groups, or higher consciousness by others); and
- h. The presence of co-occurring psychiatric disorders in persons who engage in substance use or other addictive behaviors.

Addiction is characterized by²:

- a. **Inability to consistently Abstain;**
- b. **Impairment in Behavioral control;**
- c. **Craving;** or increased "hunger" for drugs or rewarding experiences;
- d. **Diminished recognition of significant problems** with one's behaviors and interpersonal relationships; and
- e. **A dysfunctional Emotional response.**

The **power of external cues** to trigger craving and drug use, as well as to increase the frequency of engagement in other potentially addictive behaviors, is also a characteristic of addiction, with the hippocampus being important in memory of previous euphoric or dysphoric experiences, and with the amygdala being important in having motivation concentrate on selecting behaviors associated with these past experiences.

Although some believe that the difference between those who have addiction, and those who do not, is the *quantity* or *frequency* of alcohol/drug use, engagement in addictive behaviors (such as gambling or spending)³, or exposure to other external rewards

(such as food or sex), a characteristic aspect of addiction is the *qualitative way* in which the individual responds to such exposures, stressors and environmental cues. A particularly pathological aspect of *the way* that persons with addiction pursue substance use or external rewards is that preoccupation with, obsession with and/or pursuit of rewards (e.g., alcohol and other drug use) persist despite the accumulation of adverse consequences. These manifestations can occur compulsively or impulsively, as a reflection of impaired control.

Persistent risk and/or recurrence of relapse, after periods of abstinence, is another fundamental feature of addiction. This can be triggered by exposure to rewarding substances and behaviors, by exposure to environmental cues to use, and by exposure to emotional stressors that trigger heightened activity in brain stress circuits.⁴

In addiction there is a significant impairment in executive functioning, which manifests in problems with perception, learning, impulse control, compulsivity, and judgment. People with addiction often manifest a lower readiness to change their dysfunctional behaviors despite mounting concerns expressed by significant others in their lives; and display an apparent lack of appreciation of the magnitude of cumulative problems and complications. The still developing frontal lobes of adolescents may both compound these deficits in executive functioning and predispose youngsters to engage in “high risk” behaviors, including engaging in alcohol or other drug use. The profound drive or craving to use substances or engage in apparently rewarding behaviors, which is seen in many patients with addiction, underscores the compulsive or avolitional aspect of this disease. This is the connection with “powerlessness” over addiction and “unmanageability” of life, as is described in Step 1 of 12 Steps programs.

Addiction is more than a behavioral disorder. Features of addiction include aspects of a person’s behaviors, cognitions, emotions, and interactions with others, including a person’s ability to relate to members of their family, to members of their community, to their own psychological state, and to things that transcend their daily experience.

Behavioral manifestations and complications of addiction, primarily due to impaired control, can include:

- a. Excessive use and/or engagement in addictive behaviors, at higher frequencies and/or quantities than the person intended, often associated with a persistent desire for and unsuccessful attempts at behavioral control;
- b. Excessive time lost in substance use or recovering from the effects of substance use and/or engagement in addictive behaviors, with significant adverse impact on social and occupational functioning (e.g. the development of interpersonal relationship problems or the neglect of responsibilities at home, school or work);
- c. Continued use and/or engagement in addictive behaviors, despite the presence of persistent or recurrent physical or psychological problems which may have been caused or exacerbated by substance use and/or related addictive behaviors;

- d. A narrowing of the behavioral repertoire focusing on rewards that are part of addiction; and
- e. An apparent lack of ability and/or readiness to take consistent, ameliorative action despite recognition of problems.

Cognitive changes in addiction can include:

- a. Preoccupation with substance use;
- b. Altered evaluations of the relative benefits and detriments associated with drugs or rewarding behaviors; and
- c. The inaccurate belief that problems experienced in one's life are attributable to other causes rather than being a predictable consequence of addiction.

Emotional changes in addiction can include:

- a. Increased anxiety, dysphoria and emotional pain;
- b. Increased sensitivity to stressors associated with the recruitment of brain stress systems, such that "things seem more stressful" as a result; and
- c. Difficulty in identifying feelings, distinguishing between feelings and the bodily sensations of emotional arousal, and describing feelings to other people (sometimes referred to as alexithymia).

The emotional aspects of addiction are quite complex. Some persons use alcohol or other drugs or pathologically pursue other rewards because they are seeking "positive reinforcement" or the creation of a positive emotional state ("euphoria"). Others pursue substance use or other rewards because they have experienced relief from negative emotional states ("dysphoria"), which constitutes "negative reinforcement." Beyond the initial experiences of reward and relief, there is a **dysfunctional emotional state** present in most cases of addiction that is associated with the persistence of engagement with addictive behaviors. The state of addiction is not the same as the state of intoxication. When anyone experiences mild intoxication through the use of alcohol or other drugs, or when one engages non-pathologically in potentially addictive behaviors such as gambling or eating, one may experience a "high", felt as a "positive" emotional state associated with increased dopamine and opioid peptide activity in reward circuits. After such an experience, there is a neurochemical rebound, in which the reward function does not simply revert to baseline, but often drops below the original levels. This is usually not consciously perceptible by the individual and is not necessarily associated with functional impairments.

Over time, repeated experiences with substance use or addictive behaviors are not associated with ever increasing reward circuit activity and are not as subjectively rewarding. Once a person experiences withdrawal from drug use or comparable behaviors, there is an anxious, agitated, dysphoric and labile emotional experience,

related to suboptimal reward and the recruitment of brain and hormonal stress systems, which is associated with withdrawal from virtually all pharmacological classes of addictive drugs. While tolerance develops to the “high,” tolerance does not develop to the emotional “low” associated with the cycle of intoxication and withdrawal. Thus, in addiction, persons repeatedly attempt to create a “high”--but what they mostly experience is a deeper and deeper “low.” While anyone may “want” to get “high”, those with addiction feel a “need” to use the addictive substance or engage in the addictive behavior in order to try to resolve their dysphoric emotional state or their physiological symptoms of withdrawal. Persons with addiction compulsively use even though it may not make them feel good, in some cases long after the pursuit of “rewards” is not actually pleasurable.⁵ Although people from any culture may choose to “get high” from one or another activity, it is important to appreciate that addiction is not solely a function of choice. Simply put, addiction is not a desired condition.

As addiction is a chronic disease, periods of relapse, which may interrupt spans of remission, are a common feature of addiction. It is also important to recognize that return to drug use or pathological pursuit of rewards is not inevitable.

Clinical interventions can be quite effective in altering the course of addiction. Close monitoring of the behaviors of the individual and contingency management, sometimes including behavioral consequences for relapse behaviors, can contribute to positive clinical outcomes. Engagement in health promotion activities which promote personal responsibility and accountability, connection with others, and personal growth also contribute to recovery. It is important to recognize that **addiction can cause disability or premature death, especially when left untreated or treated inadequately.**

The qualitative ways in which the brain and behavior respond to drug exposure and engagement in addictive behaviors are different at later stages of addiction than in earlier stages, indicating progression, which may not be overtly apparent. As is the case with other chronic diseases, the condition must be monitored and managed over time to:

- a. Decrease the frequency and intensity of relapses;
- b. Sustain periods of remission; and
- c. Optimize the person’s level of functioning during periods of remission.

In some cases of addiction, medication management can improve treatment outcomes. In most cases of addiction, the integration of psychosocial rehabilitation and ongoing care with evidence-based pharmacological therapy provides the best results. Chronic disease management is important for minimization of episodes of relapse and their impact. Treatment of addiction saves lives †

Addiction professionals and persons in recovery know the hope that is found in recovery. Recovery is available even to persons who may not at first be able to perceive this hope, especially when the focus is on linking the health consequences to the disease of addiction. **As in other health conditions, self-management, with mutual support, is very important in recovery from addiction.** Peer support such as

that found in various “self-help” activities is beneficial in optimizing health status and functional outcomes in recovery. ‡

Recovery from addiction is best achieved through a combination of self-management, mutual support, and professional care provided by trained and certified professionals.

† See ASAM Public Policy Statement on **Treatment for Alcohol and Other Drug Addiction**, Adopted: May 01, 1980, Revised: January 01, 2010

(<http://www.asam.org/1TREATMENT%20AND%20AOD%201-10.pdf>)

‡ see ASAM Public Policy Statement on **The Relationship between Treatment and Self Help: A Joint Statement of the American Society of Addiction Medicine, the American Academy of Addiction Psychiatry, and the American Psychiatric Association**, Adopted: December 01, 1997

(<http://www.asam.org/1TREATMENT%20AND%20SELF-HELP%20-%20JOINT%2012-972.pdf>)

Explanatory footnotes:

1. The neurobiology of reward has been well understood for decades, whereas the neurobiology of addiction is still being explored. Most clinicians have learned of reward pathways including projections from the ventral tegmental area (VTA) of the brain, through the median forebrain bundle (MFB), and terminating in the nucleus accumbens (Nuc Acc), in which dopamine neurons are prominent. Current neuroscience recognizes that the neurocircuitry of reward also involves a rich bi-directional circuitry connecting the nucleus accumbens and the basal forebrain. It is the reward circuitry where reward is registered, and where the most fundamental rewards such as food, hydration, sex, and nurturing exert a strong and life-sustaining influence. Alcohol, nicotine, other drugs and pathological gambling behaviors exert their initial effects by acting on the same reward circuitry that appears in the brain to make food and sex, for example, profoundly reinforcing. Other effects, such as intoxication and emotional euphoria from rewards, derive from activation of the reward circuitry. While intoxication and withdrawal are well understood through the study of reward circuitry, understanding of addiction requires understanding of a broader network of neural connections involving forebrain as well as midbrain structures. Selection of certain rewards, preoccupation with certain rewards, response to triggers to pursue certain rewards, and motivational drives to use alcohol and other drugs and/or pathologically seek other rewards, involve multiple brain regions outside of reward neurocircuitry itself.

2. These five features are not intended to be used as “diagnostic criteria” for determining if addiction is present or not. Although these characteristic features are widely present in most cases of addiction, regardless of the pharmacology of the

substance use seen in addiction or the reward that is pathologically pursued, each feature may not be equally prominent in every case. The diagnosis of addiction requires a comprehensive biological, psychological, social and spiritual assessment by a trained and certified professional.

3. In this document, the term "addictive behaviors" refers to behaviors that are commonly rewarding and are a feature in many cases of addiction. Exposure to these behaviors, just as occurs with exposure to rewarding drugs, is facilitative of the addiction process rather than causative of addiction. The state of brain anatomy and physiology is the underlying variable that is more directly causative of addiction. Thus, in this document, the term "addictive behaviors" does not refer to dysfunctional or socially disapproved behaviors, which can appear in many cases of addiction. Behaviors, such as dishonesty, violation of one's values or the values of others, criminal acts etc., can be a component of addiction; these are best viewed as complications that result from rather than contribute to addiction.

4. The anatomy (the brain circuitry involved) and the physiology (the neuro-transmitters involved) in these three modes of relapse (drug- or reward-triggered relapse vs. cue-triggered relapse vs. stress-triggered relapse) have been delineated through neuroscience research.

Relapse triggered by exposure to addictive/rewarding drugs, including alcohol, involves the nucleus accumbens and the VTA-MFB-Nuc Acc neural axis (the brain's mesolimbic dopaminergic "incentive salience circuitry"--see footnote 2 above). Reward-triggered relapse also is mediated by glutamatergic circuits projecting to the nucleus accumbens from the frontal cortex.

Relapse triggered by exposure to conditioned cues from the environment involves glutamate circuits, originating in frontal cortex, insula, hippocampus and amygdala projecting to mesolimbic incentive salience circuitry.

Relapse triggered by exposure to stressful experiences involves brain stress circuits beyond the hypothalamic-pituitary-adrenal axis that is well known as the core of the endocrine stress system. There are two of these relapse-triggering brain stress circuits – one originates in noradrenergic nucleus A2 in the lateral tegmental area of the brain stem and projects to the hypothalamus, nucleus accumbens, frontal cortex, and bed nucleus of the stria terminalis, and uses norepinephrine as its neurotransmitter; the other originates in the central nucleus of the amygdala, projects to the bed nucleus of the stria terminalis and uses corticotrophin-releasing factor (CRF) as its neurotransmitter.

5. Pathologically pursuing reward (mentioned in the Short Version of this definition) thus has multiple components. It is not necessarily the amount of exposure to the reward (e.g., the dosage of a drug) or the frequency or duration of the exposure that is pathological. In addiction, pursuit of rewards persists, despite life problems that accumulate due to addictive behaviors, even when engagement in the behaviors ceases to be pleasurable. Similarly, in earlier stages of addiction, or even before the outward manifestations of addiction have become apparent, substance use or

engagement in addictive behaviors can be an attempt to pursue relief from dysphoria; while in later stages of the disease, engagement in addictive behaviors can persist even though the behavior no longer provides relief.

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