

Understanding the Disease of Addiction*

*Adapted from “**Positive Sobriety**” Daniel H. Angres M.D. 2015

Addiction is a Brain Disease
(Alan Leshner PhD, Director, NIDA, 1996)

The concept of alcoholism and other drug dependency as being a disease first surfaced early in the nineteenth century. In 1956, the American Medical Association (AMA) declared alcoholism an illness. However, it wasn't until 1987 that the AMA and other medical organizations officially termed addiction a disease. Today, the concept of addiction as a disease has widespread acceptance. The American Society of Addiction Medicine (ASAM) created a public policy statement (2011) that captures the essence of our current understanding:

Addiction is a primary, chronic disease of brain reward, motivation, memory and related circuitry. Dysfunction in these circuits leads to characteristic biological, psychological, social and spiritual manifestations. This is reflected in an individual pathologically pursuing reward and/or relief by substance use and other behaviors.

Addiction is characterized by inability to consistently abstain, impairment in behavioral control, craving, diminished recognition of significant problems with one's behaviors and interpersonal relationships, and a dysfunctional emotional response. Like other chronic diseases, addiction often involves cycles of relapse and remission. Without treatment or engagement in recovery activities, addiction is progressive and can result in disability or premature death.

This description of the disease of addiction has definite utility when trying to understand the mechanisms responsible for the processes that occur under the direct influence of substances or addicting behaviors and for a period of time in early abstinence. The phenomenon of craving in some people can also be at least partly attributed to these neurophysiologic mechanisms. Under the direct influence of the disease, the addict is in an altered state of consciousness, one that is now measurable newer imaging techniques. When an addict is not using but close in time to use or triggered to use, there is a pull toward this altered state. This is like a gravitational pull that is particularly strong in the addict. It can help to know this, as it helps explain what can uniquely happen in the addict. This can help with denial as well as shame that often accompany use and craving states and most certainly relapse. There are also advantages for the medical community to understand these mechanisms so that the proper specialized approaches to addiction can be implemented. The status of “disease” can also assist with the necessary coverage for treatment,

giving addiction the rightful parity with other diseases in psychiatry and medicine. But a strong word of caution is necessary.

The disease model can limit the growth in character and self-awareness that need to occur in a positive sobriety. In fact, the emphasis of this book is to recognize our inherent capacity to rise above and positively influence our physiology. So, as important as the following understanding of the disease is, the limitations of the disease model that end this section need to be fully understood.

Addiction can be defined as the continued use of a mood-altering, addicting substance or behavior (e.g., gambling, compulsive sexual behaviors) despite adverse consequences. Professionals have learned that alcoholism is a primary, chronic disease with genetic, psychosocial, and environmental factors influencing its development and manifestations. It is characterized by continuous or periodic impaired control over addicting substances despite adverse consequences, and distortions in thinking, most notably denial. This is a definition forwarded in the *Journal of the American Medical Association* in 1992, and it includes the thinking of the American Society of Addiction Medicine and the National Council on Alcoholism and Drug Dependencies. Since 1992, continued exploration of the nature of addiction has included other mood-altering substances besides alcohol as well as a number of highly reinforcing behaviors. Additionally, the disease model implies that addicts are incapable of returning to controlled use; therefore, treatment should be focused on abstinence. The disease model of addiction is now well understood and accepted by both research and clinical professionals (Guze et al. 1986). To fully understand this disease allows one to fully appreciate the powerlessness and unmanageability that accompany it.

A percentage of the population (thought to be around 50 percent of all addicts) has a biogenetic predisposition to chemicals or addictive behaviors or both; however, early life traumatic experience such as isolation or abuse can also contribute to a predisposition to addiction. Furthermore, exposure to addicting substances for any reason can produce vulnerability to addiction. In fact, recent studies suggest that even a single exposure to a substance like morphine can make lasting changes in the brain, affecting memory and creating a process of pathological learning; that is, learning to crave drugs. Once there is excessive drug use, there are disturbances in stress response systems. This often leads to compulsive repetitive patterns in an effort to capture the initial reinforcement or to block withdrawal (Koob and Kreek 2007). The disease of addiction represents a spectrum of affected individuals. In any case, the end result is the same: repeated behaviors in the face of negative consequences.

Genetics

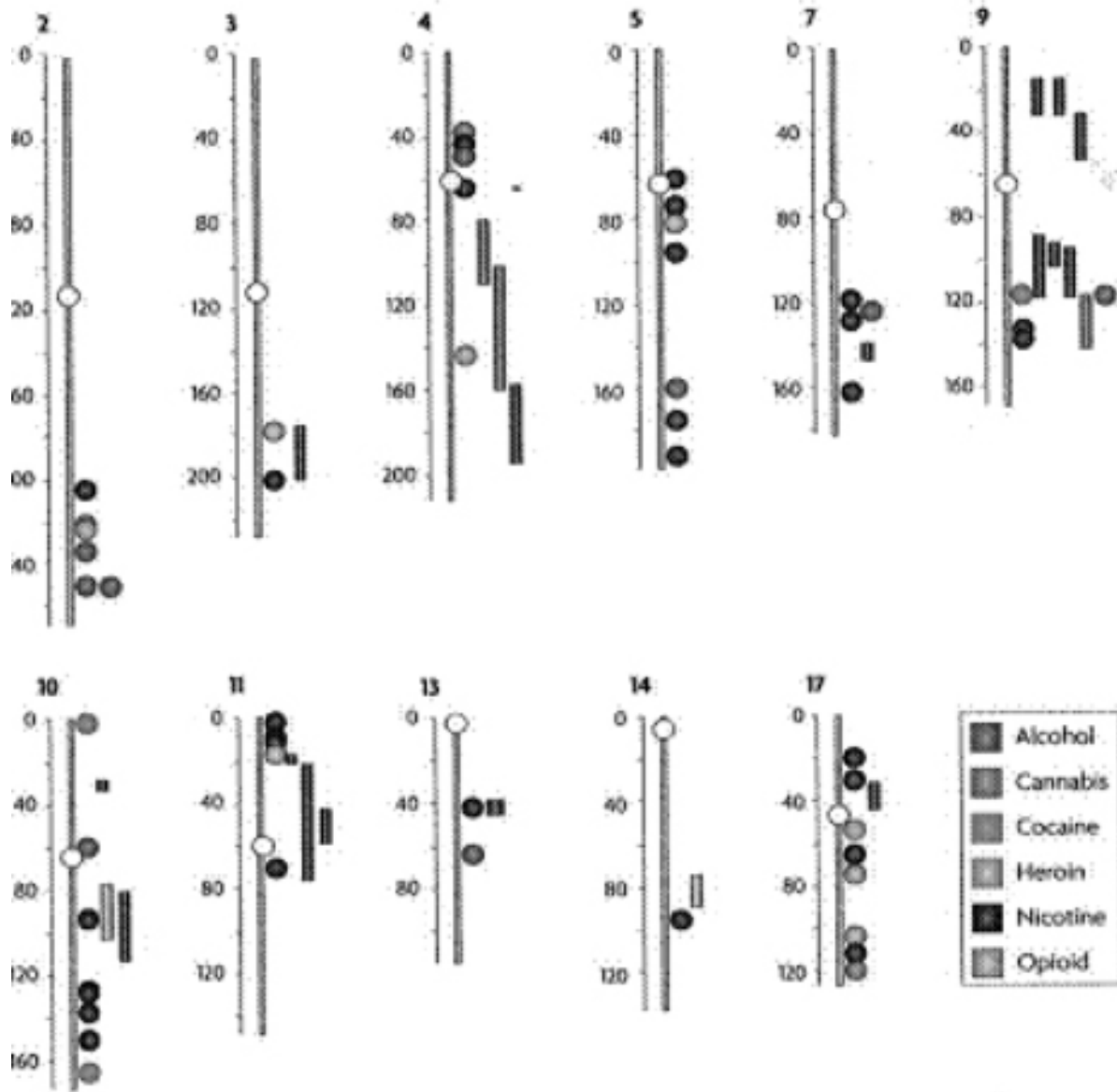
Familial transmission of alcoholism risk is, in part, genetically induced (Bohman et al. 1987; Devor and Cloninger 1989; Cloninger 1987; Edenberg 2003). Animal studies have demonstrated that specific alcoholism-related traits like sensitivity to intoxication and sedative effects, development of tolerance and withdrawal, and even susceptibility to organ damage can have genetic origins. Family illness studies, twin studies, and adoption studies have all supported a genetic contribution to alcoholism. The Human Genome Project is also contributing to our understanding of the role of genetics in alcoholism. The National Institute on Alcohol Abuse and

Alcoholism's (NIAAA) Collaborative Study on the Genetics of Alcoholism discovered reduced brain wave amplitude in subjects that reflects an underlying genetic variation in the brain's response to alcohol (National Institute on Alcohol Abuse and Alcoholism 2003). Genetic variants referred to as "polymorphisms" are being intensively studied. Some of these variants, like those that determine the metabolism of alcohol dehydrogenase, can protect some people from alcoholism by producing a flushing response (like a natural antabuse reaction). Other polymorphisms involved in this same pathway, like ADH2 and 3, seem to predispose those who possess them to alcoholism.

What has been demonstrated in alcoholism has generally held true for other abused substances and addicting behaviors. For example, beta-endorphin levels may be low in predisposed individuals with an exaggerated response to alcohol (van den Wildenberg, Wiers, and Dessers 2007) and opiates. The stronger urge to drink in the alcoholic may be related to the G allele that predisposes the individual to drug use in general (Gianoulakis, Krishnan, and Thavundayil 1996). There are certain polymorphisms or genetic variations such as with the A1 allele of the D2 receptor gene (OPRMI) that may result in reduced dopamine signaling leading to greater need for artificial means for dopamine enhancement (Parsian, Cloninger, and Zhang 2000). These genetic variations may also predict responses to certain medications. For example, naltrexone may work much better in this A1 variation group, even possibly stimulating "hidden opiate receptors," thereby producing a paradoxical sense of well-being (Kosten et al. 2002). Sometimes, for some alcoholics, a paradoxical response that occurs has been referred to as "endorphin sensitive alcoholism" and may be seen in up to one-third of alcoholics with a northern European background (Heilig et al. 2011). This group tends to have a good response to naltrexone.

Other genetic variations that involve, for example, the GABA, neuropeptide Y, and glutamate systems are implicated in multiple addiction scenarios. Some of these are linked with personality variable, age of likely onset of addiction, and vulnerability to stress and depression. The future of addiction medicine will be closely linked to these specific variants, providing an even greater ability for individualized treatments. It also appears that drug of choice is at least in part determined in many by genetic variants (see the chart on the following page).

Genetic Markers for Addiction



Sweeney, M. S. 2009. *Brain: The complete mind*. Washington, D.C.: National Geographic.

In a 2003 editorial in *The American Journal of Psychiatry* titled “Predisposition to Addiction: Pharmacokinetics, Pharmacodynamics and Brain Circuitry,” Dr. Peter Kalivas states, “There is little doubt that the development of addiction to drugs of abuse is in part a function of predisposing factors in an individual’s genome as well as factors associated with childhood and adolescent development.” Furthermore, more research is pointing to the commonality of all addictive processes, whether substance or behavioral in origin. This concept will be discussed in

more detail later on. It is important to note that genetic vulnerability doesn't ensure addiction. These vulnerabilities still need to be expressed (gene expression), and that depends on many factors, including environment, personality, and comorbid illnesses like depression.

In the article, "Personality Traits and Vulnerability or Resilience to Substance Use Disorders" (Belcher and Volkow et.al 2014) the authors describe that genes that moderate personality traits and how they interact with the environment and drugs may give us the best clues as to susceptibility in becoming an addict.

It is the disease model that informs treatment programs that address the psychosocial and environmental factors that contributed to the addictive behaviour in an attempt to combat the genetic influences. In fact, current evidence has suggested that 50 to 60 percent of addiction is genetically informed, leaving 40 to 50 percent to environmental and psychosocial factors (Dick and Bierut 2006).

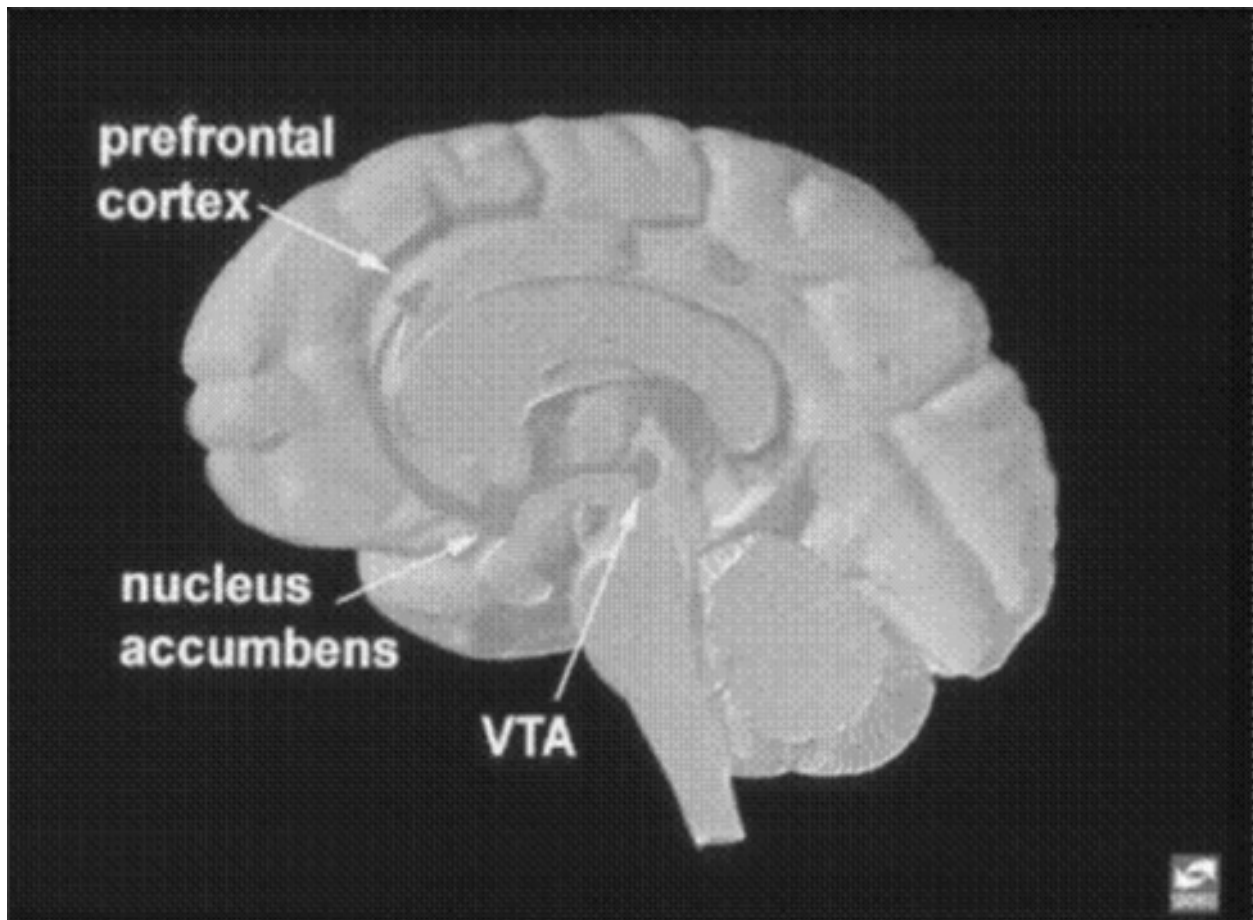
The combination of the treatment interventions described throughout this manual addresses that remaining 40 to 50 percent. The goal of treatment is to replace the addiction with personal growth and satisfaction with lifestyle, or a *positive sobriety*.

Plainly Stated

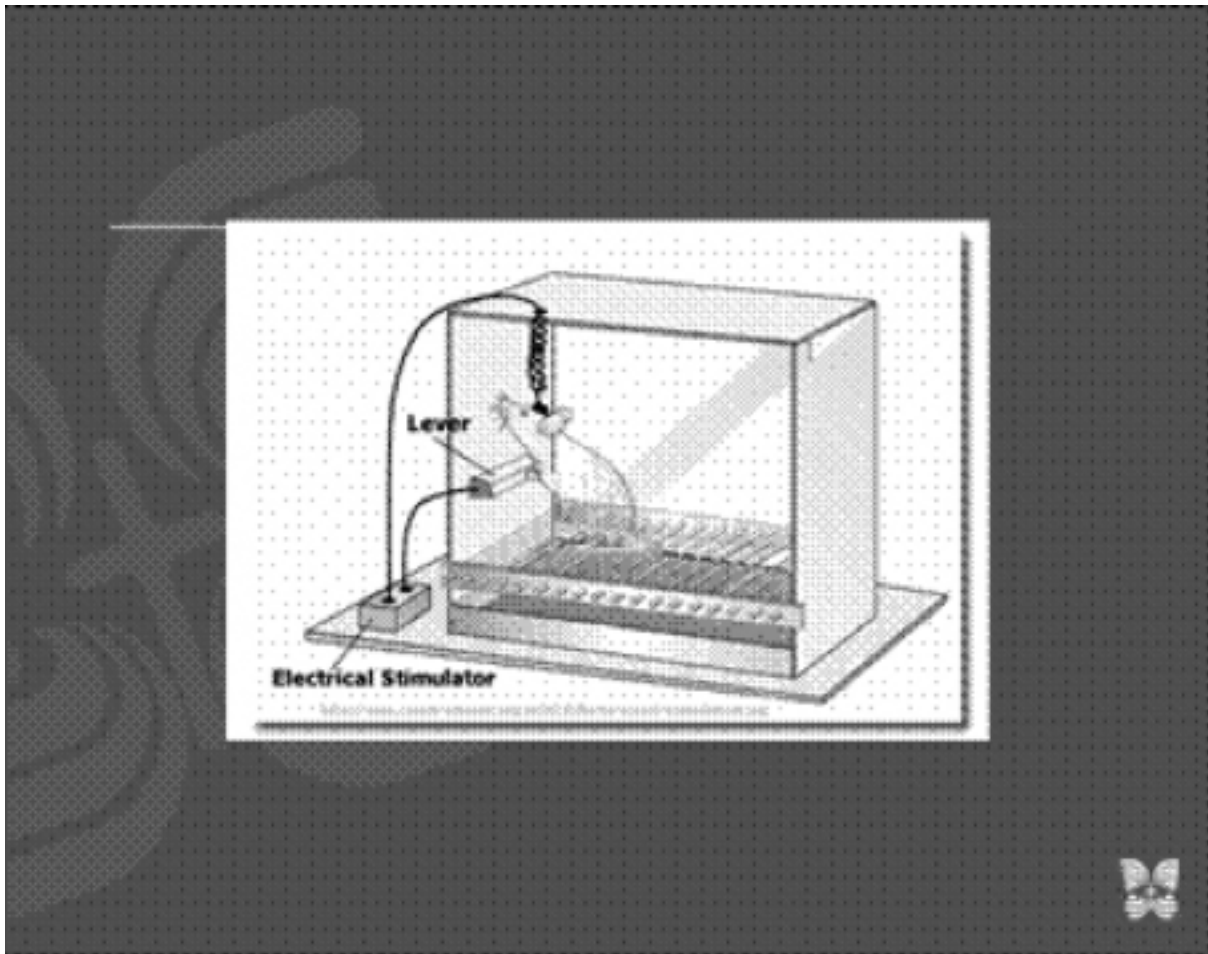
Those studying addiction have taken great strides in understanding addiction as a biogenetic disease. The genetics of this disease is highly complex, and there will be continued study for some time to come. One finding does stand out: genetics plays a role in most that are addicted. It appears to explain the unique reaction—that "magical connection"—most addicts feel when they use initially. It may explain drug (and behavior) of choice and even associated psychological issues like depression, anxiety, or even some personality dysfunctions as genetic subtypes. This is giving us an important way of understanding the unique presentations of this disease and allowing for more targeted treatments. Environment and experience (like prolonged stress) also shape the brain along with the use of drugs themselves. We always need to remember that genes are important but are not the only reason addiction occurs. When involved, genes are expressed in an environment that promotes that expression.

Reward

The reward circuitry of the brain involves the mesolimbic dopamine system including the prefrontal cortex, the nucleus accumbens, and the ventral tegmental (VTA) areas of the brain.



The mesolimbic pathways connect the more automatic bodily functions of the brain stem and peripheral nervous system and the emotional, or limbic, areas of the brain to the prefrontal cortex, which is the thinking or reflective and decision-making part of the central nervous system. Happiness doesn't come in a bottle, pill, powder, or morsel. Intellectually, everyone knows that. The brain reward circuitry people possess does not catch on to this fact as quickly. In fact, what underlies addiction is this reward problem. "Reward" is the term neuroscience uses to describe experiences that bear repeating, like pleasure or relief from some discomfort. Neuroscience has come a long way in specifically identifying the areas of the brain involved in reward (the brain reward circuitry) and the neurochemistry (our "feel-good chemicals") that create these reward responses (e.g., dopamine and beta-endorphins). Neurotransmitters (including dopamine and beta-endorphins) facilitate the communication of these systems in the reward center. This pathway is involved in essential behaviors such as eating, sleeping, and sex and is essentially hijacked in the addict. This pleasure pathway in the brain, which we share with other animals, was discovered by Dr. James Olds who, through electrical stimulation of this pleasure center (specifically the hypothalamus), demonstrated that lab animals will self-stimulate this area and completely ignore food and water in the process (1956).



The addict's initial motivation is to feel pleasure. Eventually the reward pathway shifts its sensitivities to the substance or behavior instead of the internal neurotransmitters.

The initial experience of drug use for some people can be described as a “magical connection.” The predisposed brain of the addict can be like a lock with the addicting substances (or behaviors) of choice representing the key. This vulnerability, as previously discussed, may have a genetic component or be shaped by environment or both. In any case, the drug does something to the person that “hooks” them. In some cases where this initial connection may not occur but where repeated use happens anyway (for example, through peer pressure), the brain responds to the repeated use by shifts in the reward system that over time create this unique connection. In either case, the experience that opens the door is extremely and abnormally powerful, even “magical,” in its effects.

Many addicts describe this initial experience as finally feeling “normal.” Sometimes a paradoxical (opposite) response occurs, such as an opiate like hydrocodone or alcohol (both sedating drugs) producing stimulation and increased energy. This is one reason so many health care professionals addicted to oral painkillers describe initially feeling these drugs help them be more alert. Consequently, they feel they can work more hours and even be more effective at what they do, which in turn feeds their denial that their use is a problem. This initial connection is relatively short-lived. Invariably, a vicious cycle is produced. In the pursuit of reward, the

receptors that naturally mediate reward become desensitized or diminished, creating the need for more substances, contributing to tolerance and withdrawal. The more the addict uses, the more they need, creating the progressive, vicious cycle that is the hallmark of all addictions (Berridge and Kringelbach 2008).

Plainly Stated

The brain reward center is now well understood. It plays a critical role in the relationship between addict and substance (or behavior). On one hand this reward center is essential for the experience of pleasure and our survival as a species. Unfortunately, it is hijacked in addiction, which explains why the addict continues in the addiction despite adverse consequences.

Learning and Memory

Learning and memory faculties are negatively impacted in addictive behaviors. Hyman (2005) defines addiction in terms of learning and memory and discusses the impact of addictive behaviors in usurping the neural mechanisms of learning and memory that under normal circumstances shape survival behaviors related to pursuit of rewards and predictive cues. If survival is too intimately associated in the addict's mind with securing the substance of use, then rewards and predictive cues are developed around the substance. Chronic substance use results in impaired reward-related learning, to the extent that addicts may believe that the hedonic properties of the substance far exceed any other goals and thereby devote their lives to attaining the substance.

Dopamine, a powerful neurotransmitter, can shape stimulus-reward learning, or the behavioral response to reward-related stimuli. Cueing involves significant associational memories, and connectionist brain theory suggests that these associations are wired into the brain. For example, a patient placed in the environment in which they previously used a substance may be vulnerable to an emerging pattern of brain stimuli and connections that can motivate the patient to use.

Long-term potentiation, or LTP, is an important concept in learning. Like long-term memory, LTP involves the strengthening of the connections between neurons secondary to repeated exposures. Researchers are studying LTP and recognizing that addiction represents a powerful form of learning and memory. As previously stated, addiction is a complex neurobehavioral disease involving various parts of the brain reward circuitry, such as the ventral tegmental area (VTA) and nucleus accumbens (NAc). Studies have demonstrated that VTA and NAc synapses are capable of undergoing LTP (Kauer and Malenka 2007) and that this LTP may be responsible for the behaviors that characterize addiction (Wolf 2003).

Because of the power of addiction in this reward center, this research suggests a circular pattern of reinforcement with diminished capacity for the addict to incorporate new learning strategies. Addicts are encased in a system of acquisition of a drug and the consistent reward pattern of ingestion, with decreasing awareness of other rewarding stimuli or the need to invest energy in other rewarding activities. More often, the addict maintains a limited consciousness of the destructive and alienating cycles of addiction and only comes into treatment as a result of some

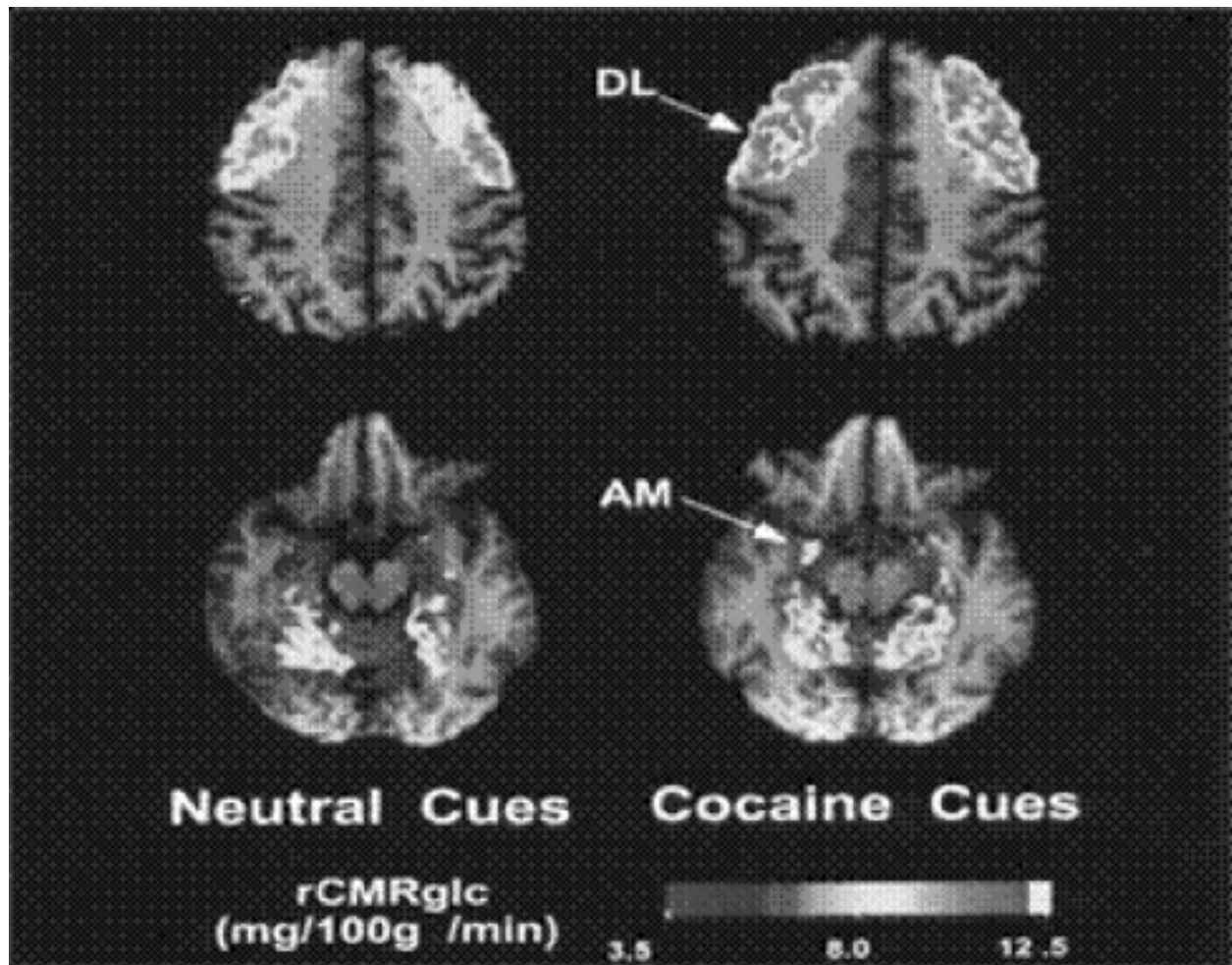
consequence of use (e.g., spouse's threat to leave, job intervention, licensing problems, legal difficulties, and so on) and rarely as a result of insight into their behavior and addiction.

The individual with an addictive disease who has engaged in chronic substance use will maintain a series of intact or collaboratively fragmented memories of the addictive behaviors and will likely recall these memories with ease during periods of craving. Memories of successful sobriety and newly learned behaviors have not likely been practiced with the same level of intensity in early recovery and are therefore vulnerable to being overridden. Also, addicts will experience a period of time referred to as "post-acute withdrawal" early in sobriety. The most common symptoms are lack of concentration, irritability, and insomnia. (See "Core Treatment Lectures and Workshops" for more information about post-acute withdrawal).

The addict has adopted a reactive response to feeling uncomfortable, and that reaction is to use a substance. Addicts will need physiological, psychological, and social support to counteract their impulsive need to medicate these uncomfortable states. Educational support is also critical, but education alone will not deter an addict from relapse. Physiological changes that impact behavior do not respond to the intellect, and the very engagement in extended addictive behaviors minimize the power of the individual's will, which results in cyclical and self-rewarding patterns of addiction.

Motivation

Motivation is another factor with biological components, and the pursuit of goals that produce desired outcomes is an integral aspect of addiction and recovery. Kalivas and Volkow (2005) support the theory that addiction involves a dysregulation in the motive circuitry, and the repetitive use of addictive drugs reorganizes brain circuitry to establish behaviors characteristic of addiction. PET studies on cue-induced craving clearly demonstrate increased reactions between the amygdala and the prefrontal cortex when people are actively reminded of their addicting agent. The next graphic shows how cues such as pictures of drug paraphernalia activate the prefrontal cortex's dorsolateral (DL) area and the amygdala (AM). The prefrontal cortex, responsible for decision making, gets activated with the amygdala, the fear-based part of the brain, creating a connection for craving. This activates a neurotransmitter called glutamate, which creates an unpleasant feeling associated with craving that can cause the addict to try and reduce this discomfort through drug use.



From Kaufman 2001

In addition to the obvious consequences of engaging in addictive behavior (i.e., legal, financial, psychosocial), there is a risk of neuronal recircuiting that results in physiological cycles of addictive behaviors, and these circuits are increasingly difficult to break. Kalivas and Volkow (2005) propose three temporally distinct phases of addiction:

Stage 1: Acute Drug Effects

Stage 2: Transition to Addiction

Stage 3: End-Stage Addiction

In stage 1, acute drug administration results in molecular consequences that are widely distributed in the brain circuitry that impact motivation. Stage 2 reflects neuronal changes, such as D1-receptor-mediated stimulation of proteins. Stage 3 introduces the possibility that changes in protein content, function, or both move from temporary to permanent features. The researchers concluded that cellular adaptations in the prefrontal glutamatergic innervation of the accumbens are responsible for the promotion of compulsive-character drug seeking in addicts through decreasing the value of natural rewards, reducing cognitive control (choice), and increasing glutamatergic drive in response to drugs and association with drugs. In other words,

the addict's brain progresses to a point where it is driven to use, sensing that only use will bring pleasure, while having diminished ability to find healthy ways to receive reward (e.g., connection with people or spiritual pursuits).

Decision Making

Decision making is another area of cognitive function negatively impacted by addictive behaviors. A publication in *Psychiatry* (Noel, van der Linden, and Bechara 2006) suggested that addiction is an imbalance between the neural system that is reactive for signaling pain or pleasure and another neural system that is reflective and controls the reactive system. When the ventromedial prefrontal cortex (VMPC) is injured in patients who are not addicts, they make disadvantageous decisions and fail to learn from their mistakes, contrary to their preinjury personalities. The authors made striking comparisons between patients with VMPC injuries and addicts: both deny they have a problem and appear to ignore the consequences of their actions. In addiction, the neural mechanisms that enable an individual to reflect and choose wisely appear to be weakened, and they move from self-directed behavior to automatic sensory-driven behavior. The authors hypothesized that for certain people, the decision-making mechanism—the process by which one reflects and considers consequences prior to an action—in the brain is weak, and this weakness makes them vulnerable to addiction. The source of the weakness can be genetic or environmentally induced, but it is always a consequence of addiction. *Two decision-making areas of the brain are particularly affected in addiction: the orbitofrontal cortex, involved in deciding what is really best for us, and the anterior cingulate cortex, which helps us understand if the decision we made was the right one. Both these pathways are disrupted in addiction, which invariably leads to making bad decisions and, to some degree, not even realizing the decision was bad.*

Recent fMRI and PET studies demonstrate a split between the ability to make appropriate decisions as the compulsive drive for the chemical or addiction progresses. Goldstein and Volkow (2002) demonstrated that as addiction progresses, one's ability to make appropriate choices diminishes. Increased impulsivity is accompanied by old memories of times when the addiction “worked” as well as by negation of options other than engaging in the addiction. Not only are some addicts predisposed to a sluggish reward circuitry before ever using a substance or engaging in addiction, but they also now appear to have some degree of difficulty in decision making. Deficits in the aforementioned areas constitute the vicious cycle of addiction

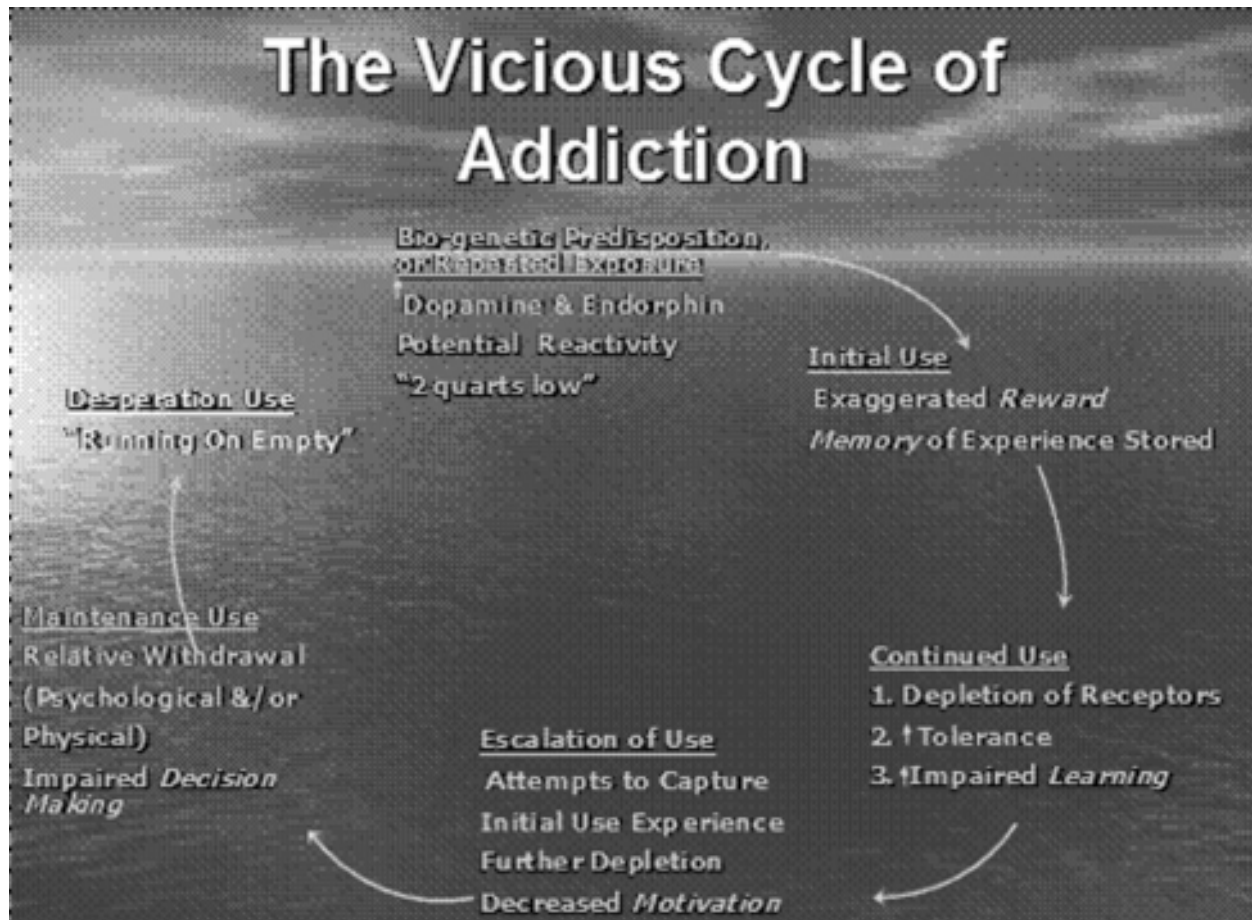


Figure 1: The above figure outlines how a predisposition (being “two quarts low”) creates a vicious cycle through initial intensified reward and continued and escalating use. The more one uses, the more they need, and the more they need, the more they use and the more they shut down their own endogenous feel-good chemistry, leading to a downward spiral. Increasing deficits in learning, memory, motivation, and decision making accompany this process, accelerating the downward spiral.

Plainly Stated

Addiction may start with that magical connection, often referred to as “Reward Sensitivity” but other factors quickly take over. These include issues with memory, learning, motivation, and decision making. Over time, repeated use stores the memory deep in various parts of the brain, especially the ones associated with strong emotion. It is as if we learn to be addicted and learn it too well! The motivation for use becomes overwhelming, and eventually, as the disease progresses, we lose our ability to make decisions—in a sense, our disease makes them for us. This is particularly related to triggers or ‘cues’ and referred to as “Cue Sensitivity” The disease and its progression can be summarized as “Reward Sensitivity and Cue Reactivity”. Recovery involves a whole new learning process: one that needs to be strong enough to overcome the power of addiction.

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