

Understanding the Disease of Addiction

Kathy Bettinardi-Angres, MS, RN, APN, CADC, and Daniel H. Angres, MD

The disease of chemical dependency can be traced to neural pathways in the brain predating a diagnosis of addiction. A genetic predisposition alone is not enough to predict addiction. Typically, psychological and social influences drive the person to use the addicting substances, and the combination of genetic predisposition and these influences triggers the disease. Chemically dependent nurses are susceptible to the scrutiny of boards of authority if their addiction affects the workplace. Therefore, those in authority should understand the disease of addiction and use an effective, compassionate approach that will benefit both the addicted nurse and nursing as a whole.

Learning Objectives

- Define addiction, substance abuse, and substance dependence.
- Identify contributing factors for addiction.
- Explain the biological neural pathways that underlie addiction.

The concept of alcoholism and other drug dependency as being a disease first surfaced early in the 19th century. In 1956, the American Medical Association (AMA) declared alcoholism an illness, and in 1987, the AMA and other medical organizations officially termed addiction a disease (Leshner, 1997). The American Nurses Association estimates that 6% to 8% of nurses have alcohol or drug abuse problems serious enough to impair their judgment, meaning that the disease of addiction profoundly affects the nursing profession.

The following description of the disease of addiction has 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 afterwards. The phenomenon of craving in some 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 with the newer imaging techniques. There are advantages for the nursing and medical community to understand these mechanisms, so the proper specialized approaches to addiction can be implemented. The status of “disease” can also assist with the necessary coverage for treatment, giving addiction its rightful parity with other diseases in psychiatry and medicine.

Not everyone accepts addiction as a disease. Some still view it as a moral failure or lack of will power. Many nurses remain silent about their addiction to mood-altering substances for a number of reasons. The most important reason is denial (Morse & Flavin, 1992). Addicted nurses also experience shame and guilt that drive the addiction underground. They do not intentionally jeopardize the safety and well-being of their patients or themselves; in fact, the workplace is often the last place the

signs and symptoms of addiction become obvious. Thus, overt signs and symptoms in the workplace usually mean the disease has already progressed (Angres, Talbott, & Bettinardi-Angres, 2001) (see Table 1).

Understanding the biological mechanisms that underlie addiction can help others recognize and treat the problem with more empathy, less stigmatization, and more effective outcomes. Alcohol and drug addiction are primary, chronic, progressive, and often fatal health problems for all of society, not just the medical and nursing community.

Defining Addiction

Addiction is defined as the ongoing use of mood-altering substances, such as alcohol and drugs, despite adverse consequences. Genetic, psychosocial, and environmental factors influence the development and manifestations of the disease (Morse & Flavin, 1992). Characteristics of alcoholism include continuous or periodic impaired control over drinking, preoccupation with alcohol, use of alcohol despite adverse consequences, and distortions in thinking—most notably denial. To the brain, alcoholism and drug addiction are the same.

The *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (DSM-IV) defines substance abuse and dependence as a maladaptive pattern of substance use, leading to clinically significant impairment or distress, although they are manifested differently.

Substance dependence is defined and manifested by three or more of the following occurring at any time in the same 12-month period:

- A need for markedly increased amounts of the substance to achieve intoxication or desired effects
- A markedly diminished effect with continued use of the same amount of the substance
- The characteristic withdrawal syndrome for the substance

TABLE 1

Recognizing Addiction in the Workplace

Typically, the workplace is the last place the signs and symptoms of addiction become obvious. Changes in mood, behavior, and appearance may be gradual or sudden. The signs and symptoms of addiction include:

- frequent tardiness and absenteeism
- poorly explained accidents and injuries
- relationship discord: marital, family, professional
- deterioration in personal appearance
- significant weight loss or gain
- long sleeves and tinted glasses inappropriate for the setting
- overuse of cologne and breath fresheners
- severe mood swings or change in personality
- withdrawal from family, friends, and coworkers—for example, refusing social invitations
- frequent disappearances during work hours
- smell of alcohol on breath during work hours
- too much time spent with opiates, or missing opiates
- dilated or pinpoint pupils
- extra work shifts to obtain substances.

- The same substance taken to relieve or avoid withdrawal symptoms
- The substance taken in larger amounts or over a longer period than was intended
- A persistent desire or unsuccessful efforts to cut down or control substance use
- A great deal of time spent in activities needed to obtain the substance, use the substance, or recover from the effects
- Reduction in or absence of important social, occupational, or recreational activities because of substance use
- Continued substance use despite knowledge of a persistent or recurrent physical or psychological problem caused or exacerbated by the substance.

Substance abuse is defined and manifested by one or more of the following in a 12-month period:

- Recurrent substance use resulting in a failure to fulfill major role obligations at work, school, or home
- Recurrent substance use in situations in which it is physically hazardous
- Recurrent substance-related legal problems
- Continued substance use despite persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of the substance.

Substance dependence and abuse are differentiated for diagnostic purposes, but often treated similarly by clinicians. Dependence is the more severe diagnosis, but substance abuse can lead to substance dependence.

Causes of Addiction

A percentage of the population has a biogenetic predisposition

to chemical or addictive behaviors; however, early-life traumatic experiences, such as isolation or abuse, can contribute to a predisposition to addiction. A predisposition alone is generally not enough to cause the disease. Often, a person is influenced by social factors, such as peers and societal and familial norms, and psychological issues, such as a history of physical or sexual abuse, other trauma, and dual diagnosis.

Dual Diagnosis

A common dual diagnosis for addicts and alcoholics is anxiety disorder. Chemical dependency is a primary disease, however, and is not caused by other diseases. Both diagnoses must be treated fully and equally. The only issue is the use of addicting substances to treat a chemically dependent person, which may fuel the addiction—for example, treating anxiety disorder with benzodiazepines. A person with a dual diagnosis needs continuity of care and caregivers who understand addiction.

Genetics

Familial transmission of alcoholism risk is in part genetically induced. Animal studies show that specific alcohol-related traits, such as sensitivity to intoxication and sedative effects, development of tolerance and withdrawal, and even susceptibility to organ damage, can have genetic origins. Studies of family illnesses, twins, and adoption support 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 that reflects an underlying genetic variation in the brain's response to alcohol (NIAAA, 2003).

The findings regarding alcoholism have generally held true for substance abuse and addicting behaviors. For example, people with an exaggerated response to alcohol (van den Wildenberg et al., 2007) and opiates may have low beta-endorphin levels. The stronger urge to drink in the alcoholic may be related to the G allele that predisposes people to drug use in general (Gianoulakis, Krishnan, & Thavundayil, 1996). In his 2003 editorial in *The American Journal of Psychiatry*, "A Predisposition to Addiction: Pharmacokinetics, Pharmacodynamics, and Brain Circuitry," Dr. Peter Kalivas (2003) 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" (p. 160). Research points to the commonality of all addictive processes, whether the addiction is to a substance or a behavior.

The Addictive Personality: Does it Exist?

Despite many discussions regarding an addictive personality, research indicates that the personalities of alcoholics are heterogeneous. Certain personality problems, such as impulsivity and poor coping skills, can result from early developmental problems,

but these personality deficits can also result from addiction. That is, addiction can interfere with the way people see themselves, cope with stress, and interact with others. Sometimes, determining whether personality problems are primary or secondary to addiction must wait until the addict is sober for an extended period.

From a psychological perspective, Khantzian and Mack have described “the heavy reliance on chemical substances to relieve pain, provide pleasure, regulate emotions, and create personality cohesion.” They have described this process as self-governance, and although no specific addictive personality may be identifiable, the maladaptive personality functioning in addiction creates a need for a cohesive sense of self and strategies to enhance self-governance capabilities. (Khantzian and Mack, 1983)

Deficits in the neurochemistry and reward circuitry in addiction, such as dopamine synthesis, likely influence personality in addicted patients. Some speculate that these circuits evolved in the brain for purposes of social attachment and are activated in addiction. It seems logical that the strong connection that can occur among sober addicts plays a pivotal role in addiction recovery. Conversely, disorders that disrupt these attachment and affiliative systems, such as borderline personality disorder, can pose significant challenges to the treatment of addiction.

In all probability, adaptive styles occur at different times in the addictive process. Before the addiction, a deficit in reward capacity could create a feeling of deprivation, leading to craving states and mood instability. During active substance use, previous temperament styles are exaggerated, and because of ongoing addiction, character development is arrested.

Nurses at Risk

The risk to nurses is the same as it is for the general population, except for one thing: Nurses have better access to opiates. This accessibility, coupled with the culture of relieving pain with mood-altering substances, can create an ideal environment for a person who is genetically predisposed to addiction. Thus, nurses with a significant family history of addiction should either abstain from working with opiates or have an awareness of the potential for danger and incorporate stress-reducing behaviors into their lifestyle as a prevention. Currently, no one can predict if a person will become addicted in his or her lifetime.

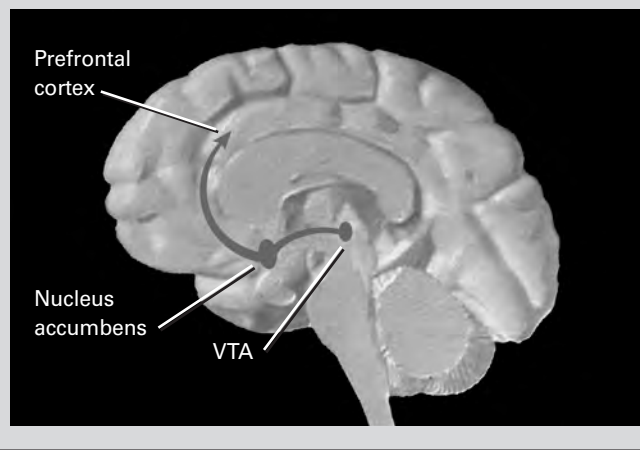
Reward Circuitry of the Brain

The mesolimbic pathways connect the brainstem and peripheral nervous system responsible for automatic body functions, and the limbic areas of the brain responsible for emotions to the prefrontal cortex, where thinking and decision making take place. Intellectually, people know that happiness does not come in a bottle, pill, or morsel. Unfortunately, the brain’s reward circuitry does not know it. In fact, what underlies addiction is reward (see Figure 1).

FIGURE 1

Reward Circuitry of the Brain

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



Reward is the term neuroscience uses to describe experiences that bear repeating, such as pleasure or relief from some discomfort. Neuroscience has come a long way in identifying the areas of the brain involved in reward and the neurochemistry of our feel-good chemicals that create reward responses. Neurotransmitters, such as dopamine and beta-endorphins, facilitate communication to the reward center. In the addict, the pathway involved in essential behaviors, such as eating, sleeping, and sex, is hijacked. The addict’s initial motivation is to feel pleasure. Eventually, the reward pathway shifts its sensitivities to the substance or behavior instead of the neurotransmitters. In other words, the brain begins to depend on outside chemicals for reward.

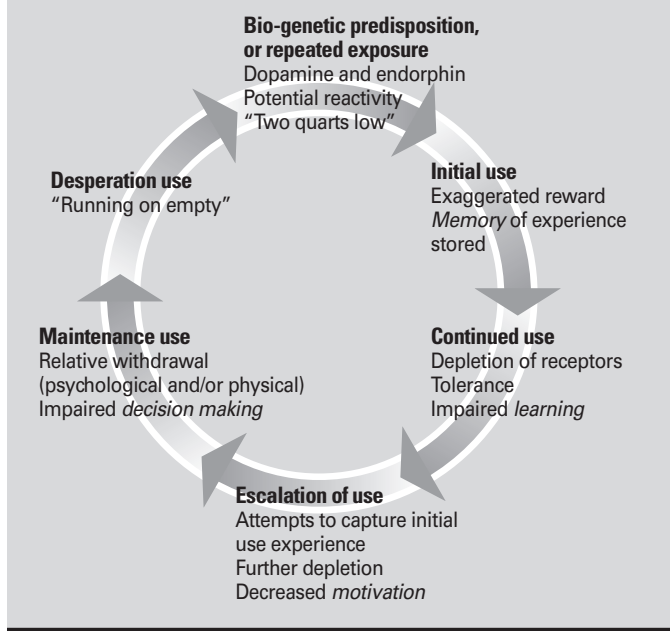
Magical Connection

The predisposed brain of the addict is like a lock, and the addicting substance or behavior is the key. When the key opens the lock the first time, the experience is extremely powerful, even magical. In the mid-1990s, positron emission tomography (PET) scans showed that the brain’s reward circuitry in people with a previous or family history of addiction lit up in a way the reward circuitry of those in control groups did not. These studies and others demonstrate that addicts experience their substances more intensely than nonaddicts. Other studies suggest that addicts run “two quarts low in feel-good chemistry” and then experience an exaggerated response when they find their drug—a phenomenon called “the magical connection.” Now, advanced techniques, such as PET scans and magnetic resonance imaging (MRI), can scientifically demonstrate this phenomenon.

Many addicts describe this initial experience as finally feeling normal. Sometimes, a paradoxical response occurs. For example, an opiate that typically causes sedation instead produces

FIGURE 2

Vicious Circle of Addiction



stimulation and increased energy. This response helps explain why many health-care professionals addicted to oral analgesics describe a feeling of being more alert. Consequently, they feel they can work more hours and even be more effective at what they do, thus feeding their denial. This initial connection is relatively short-lived. Invariably, a vicious circle takes over. In the pursuit of reward, the receptors that naturally mediate reward become desensitized, which creates the need for more substances, contributing to tolerance and withdrawal. The more addicts use, the more they need, creating the progressive, vicious circle that is the hallmark of all addictions (see Figure 2).

Learning and Memory

Hyman (2005) discusses the role of addictive behaviors in usurping the neural mechanisms of learning and memory that normally shape survival behaviors related to rewards and predictive cues. If survival is too intimately associated in the addict's mind with securing the addictive substance, rewards and predictive cues develop around the substance. Chronic substance use results in impaired reward-related learning (Koob & Kreek, 2007). Addicts may believe that the hedonic properties of the substance far exceed other goals and 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 an environment where he or she previously used a substance may be vulnerable to an emerging pattern of brain stimuli and connec-

tions that can motivate the patient to use again.

This research suggests a circular pattern of reinforcement with diminished capacity for the addict to incorporate new learning strategies. Addicts are trapped in a system of drug acquisition and the consistent reward pattern of ingestion. Awareness of other rewarding stimuli or the need to invest energy in other rewarding activities decreases. More often, addicts maintain a limited consciousness of the destructive and alienating cycles of addiction and only enter treatment because of a consequence of their use, such as a spouse's threat to leave, a job intervention, licensing problems, or legal difficulties. Addicts rarely seek treatment because of insights into their behavior and addiction.

A person with an addictive disease who has engaged in chronic substance use maintains a series of intact or collaboratively fragmented memories of the addictive behaviors and likely recalls these memories with ease during periods of craving. In early recovery, memories of successful sobriety and newly learned behaviors do not have the same level of intensity; thus, they are vulnerable to being overridden. Also, addicts experience a period called post-acute withdrawal in early sobriety. The most common symptoms are lack of concentration, irritability, and insomnia.

Motivation

Motivation is another factor with biological components, and 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. MRI studies on cue-induced craving demonstrate increased reaction between the amygdala, the fear-based part of the brain, and the prefrontal cortex when people are actively reminded of their addicting agent. The prefrontal cortex, responsible for decision making, gets activated with the amygdala, 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 to reduce this discomfort through drug use.

Besides the legal, financial, and psychosocial consequences of addictive behavior, the addict also risks neuronal recircuiting that results in physiologic cycles of addictive behaviors. These circuits are increasingly difficult to break.

Decision Making

Addictive behaviors negatively affect decision making, as well. Noel, van der Linden, and Bechara (2006) suggest 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 nonaddicts, they

make detrimental decisions and fail to learn from their mistakes, contrary to their pre-injury personality. The authors make 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 people to reflect and choose wisely appear to be weakened, and addicts move from self-directed behavior to automatic sensory-driven behavior. The study's authors hypothesize that some people have a weak decision-making mechanism in the brain and that the weakness makes them vulnerable to addiction. The source of the weakness can be genetic or environmentally induced.

Recent MRI 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 memories of when the addiction worked as well as negating options other than engaging in the addiction. Not only are some people predisposed to a sluggish reward circuitry before ever using a substance, they also appear to have some degree of difficulty in decision making. Deficits in these areas constitute the vicious circle of addiction.

Denial, the close companion of addiction, feeds off the progressive deterioration of the ability to freely choose. Denial is reinforced by the powerful reward of the addiction and the deficits in learning, motivation, memory, and decision making.

Conclusion

Addiction is a biopsychosocial disease process, not a choice. Martha Morrison, MD, in her book *White Rabbit*, stated that she grew up wanting to be a physician, not a drug addict. Alcoholic and addicted nurses unanimously report the same sentiments.

If appropriately treated, addiction can remain in remission, and nurses who have peer support and monitoring have a greater chance of long-term sobriety than the general population. The recovering addict must begin an exploration into self, cultivate a program of well-being, and maintain a long-term goal of sobriety.

The bodies of authority in the nursing profession must understand the disease of addiction and its treatment. This understanding may lead to more options for addicted nurses, greater opportunities for them to heal and return to the profession, and a compassionate approach to peers that is congruent with the values of the nursing profession.

References

Angres, D., Talbott, D., & Bettinardi-Angres, K. (2001). *Healing The Healer: The Addicted Physician*. Madison, CT: The Psychosocial Press.

- Gianoulakis, C., Krishnan, B., & Thavundayil, J. (1996). Enhanced sensitivity of pituitary beta-endorphin to ethanol in subjects at high risk of alcoholism. *Archives of General Psychiatry*, 53, 250–257.
- Goldstein, X. R., & Volkow, N. D. (2002). Drug addiction and its underlying neurobiological basis: Neuroimaging evidence for involvement of the frontal cortex. *American Journal of Psychiatry*, 159(10), 1642–1652.
- Hyman, S.E. (2005). Addiction: A disease of learning and memory. *American Journal of Psychiatry*, 162, 1414–1422.
- Kalivas, P. (2003). A predisposition to addiction: Pharmacokinetics, pharmacodynamics, and brain circuitry. *American Journal of Psychiatry*, 160(1), 1–2.
- Kalivas, P. W., & Volkow, N. D. (2005). The neural basis of addiction: A pathology of motivation and choice. *American Journal of Psychiatry*, 162(8), 1403–1413.
- Khantzian, E. J., & Mack, J. E. (1983). Self-preservation and the care of the self—ego instincts reconsidered. *Psychoanalytic Study of the Child*, 38, 209–232. New Haven, CT: Yale University Press.
- Koob, G., & Kreek, J. (2007). Stress, dysregulation of drug reward pathways, and the transition to drug dependence. *American Journal of Psychiatry*, 164(8), 1149–1159.
- Leshner, A. (1997). Addiction is a brain disease, and it matters. *Science*, 278(5335), 807–808.
- Morse, R., & Flavin, D. (1992). The definition of alcoholism. *JAMA*, 268(8), 1035–1039.
- NIAAA. (2003). The genetics of alcoholism. *Alcohol Alert*, No. 60. Retrieved from <http://pubs.niaaa.nih.gov/publications/aa60.htm>.
- Noel, X., van der Linden, M., & Bechara, A. (2006). The neurocognitive mechanisms of decision making, impulse control, and loss of will power to resist drugs. *Psychiatry*, 3(5), 30–41.
- van den Wildenberg, E., Wiers, R. W., Deters, J., Janssen, R. G. J. H., Lambrichs, E., H., Smeets, H. J. M., & van Breukelen, G. J. P. (2007). A functional polymorphism of the u-opioid receptor gene (ORPMI) influences cue-induced craving for alcohol in male heavy drinkers. *Alcoholism, Clinical and Experimental Research*, 31(1), 1–10. doi: 10.1111/j.1530-0277.2006.00258.x

Kathy Bettinardi-Angres, MS, RN, APN, CADC, and Daniel H. Angres, MD, are clinicians in the field of addiction with a focus on health-care professionals.

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Learning Objectives

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- Identify contributing factors for addiction.
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CE Posttest

Understanding the Disease of Addiction

If you reside in the United States and wish to obtain 1.6 contact hours of continuing education (CE) credit, please review these instructions.

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Contact hours: 1.6

Posttest passing score is 75%.

Expiration: July 2013

Posttest

Please circle the correct answer.

- 1. Which of the following is defined as the ongoing use of mood-altering substances or behaviors despite adverse consequences?**
 - a. Dual diagnosis
 - b. Substance dependence
 - c. Substance abuse
 - d. Addiction
- 2. Which of the following is a criterion of substance abuse according to the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV)*?**
 - a. Characteristic withdrawal syndrome for the substance
 - b. Markedly diminished effect with continued use of the same amount of the substance
 - c. Recurrent substance use in situations in which it is physically hazardous
 - d. The same substance is taken to relieve or avoid withdrawal symptoms
- 3. Which statement about predisposition to addiction is correct?**
 - a. A percentage of the population has a biogenetic predisposition to chemical or addictive behaviors.
 - b. A predisposition is usually enough to cause the disease.
 - c. Early childhood experiences have little influence on the manifestation of addiction.
 - d. Studies of family illnesses, twins, and adoption do not support a genetic contribution to opioid abuse.
- 4. The stronger urge to drink in the alcoholic may be related to:**
 - a. the C allele.
 - b. the G allele.
 - c. high beta-endorphin levels.
 - d. high alpha-endorphin levels.
- 5. The risk for addiction in nurses:**
 - a. is lower than that for the general population.
 - b. is higher than that for the general population.
 - c. is the same as the general population.
 - d. depends on the nurse's specialty.
- 6. Which statement about the reward circuitry of the brain is correct?**
 - a. Neurotransmitters facilitate communication to the reward center.
 - b. Neurotransmitters inhibit communication to the reward center.
 - c. The reward circuitry of the brain includes the prone tegmental areas.
 - d. The reward circuitry of the brain involves the limbic epinephrine system.
- 7. Which statement about the mesolimbic pathway is correct?**
 - a. It connects the brainstem and peripheral nervous system responsible for automatic functions, and the limbic areas of the brain responsible for emotions to the frontal cortex.
 - b. It connects the brainstem and peripheral nervous system responsible for automatic functions, and the limbic areas of the brain responsible for emotions to the prefrontal cortex.
 - c. It connects the peripheral nervous system responsible for automatic functions and the limbic areas of the brain responsible for emotions to the prefrontal cortex.
 - d. It connects the brainstem responsible for automatic functions, and the limbic areas of the brain responsible for emotions to the frontal cortex.
- 8. Which neurotransmitter can shape stimulus-reward learning?**
 - a. Alpha-endorphin
 - b. Norepinephrine
 - c. Epinephrine
 - d. Dopamine
- 9. Which statement about addicts and treatment is correct?**
 - a. Addicts usually do not enter treatment because of a consequence of the substance use.
 - b. Addicts rarely seek treatment because of insights into their behavior and addiction.
 - c. Abstinence from the substance is usually sufficient treatment for an addict.
 - d. Isolation from other recovering addicts is a helpful treatment strategy.
- 10. Which neurotransmitter causes the unpleasant feeling associated with craving?**
 - a. Norepinephrine
 - b. Epinephrine
 - c. Glutamate
 - d. Dopamine

11. Which of the following is NOT a common symptom of post-acute withdrawal in early sobriety?

- a. Sleepiness
- b. Lack of concentration
- c. Irritability
- d. Insomnia

12. Magnetic resonance imaging studies show that cue-induced craving in addicts is associated with increased reaction between the:

- a. posterior cortex and ventral tegmental area.
- b. prefrontal cortex and nucleus accumbens.
- c. amygdala and posterior cortex.
- d. amygdala and prefrontal cortex.

13. Injury to which area in a nonaddicted person results in decision making similar to that of the addicted person?

- a. Ventromedial prefrontal cortex
- b. Ventromedial posterior cortex
- c. Lateral prefrontal cortex
- d. Lateral posterior cortex

14. Continued use of an addicting substance causes:

- a. depletion of receptors.
- b. improved learning.
- c. decreased tolerance.
- d. enhanced decision making.

15. Escalation of use occurs in an effort to:

- a. increase motivation.
- b. reduce rewards.
- c. capture the first-use experience.
- d. enhance neurotransmitters.

Evaluation Form (required)

1. Rate your achievement of each objective from 5 (high/excellent) to 1 (low/poor).

- Define addiction, substance abuse, and substance dependence.

1 2 3 4 5

- Identify contributing factors for addiction.

1 2 3 4 5

- Explain the biological neural pathways that underlie addiction.

1 2 3 4 5

Rate each of the following items from 5 (very effective) to 1 (ineffective):

2. Was the author knowledgeable about the subject?

1 2 3 4 5

3. Were the methods of presentation (text, tables, figures, etc.) effective?

1 2 3 4 5

4. Was the content relevant to the objectives?

1 2 3 4 5

5. Was the article useful to you in your work?

1 2 3 4 5

6. Was there enough time allotted for this activity?

1 2 3 4 5

Comments: _____

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