1 SUBSTANCE USE DISORDER AND ADDICTION: BASIC AND BRIEF PSYCHOPHARMACOLOGICAL AND NEUROPSYCHOLOGICAL REVIEW

LEARNING OBJECTIVES	
LO 1.A	Reader can explain the components of the CNS as relevant to addiction
LO 1.B	Reader can identify the role of various neurotransmitters in the addiction condition
LO 1.C	Reader can identify various brain areas/functions relevant to the addiction condition
LO 1.D	Reader can explain the basic psychopharmacological concepts relevant to the addiction condition
LO 1.E	Reader can identify biopsychological issues associated with various substances of misuse
LO 1.F	Reader can identify biopsychological issues associated with various process addictions

Any discussion of substance use disorder and addiction must start in the brain. You are treating a psychological, neurological, and medical condition *of the brain*. Understanding *how* the substance(s) act on the brain and *how* these actions may influence thought, emotion, and behavior is critical to best understanding the substance use disorder and addiction condition. However, I want to emphasize that the information in this chapter in no way represents a complete and thorough coverage of the pertinent psychopharmacological and neuropsychological concepts. That is impossible. There are countless texts entirely devoted to the content here in Chapter 1. Consequently, my goal for this chapter is simple. I want you to learn content from

the following pages and (perhaps more importantly) learn that there are other areas you need to learn more about, and have an initial direction and leads to go address that inquiry. So, this is where we start.

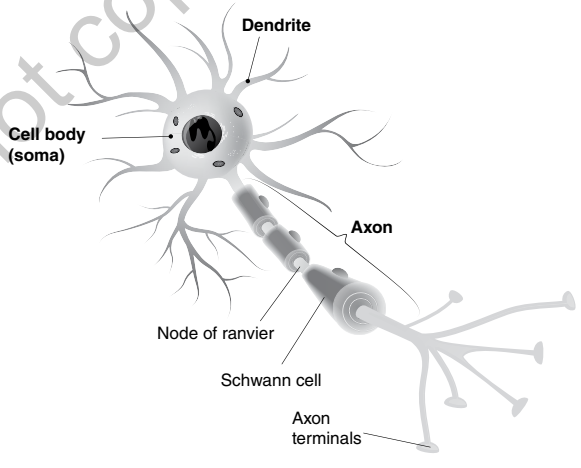
CENTRAL NERVOUS SYSTEM

The human brain is built of two cell types. Neurons (numbering in the 100 billion range) and an even larger number of glia. Each are addressed in this section.

Neurons

Neurons communicate via a series of circuits. These circuits are the foundation for all we are and experience and feel. Thus, our thoughts, emotions, and behaviors are all rooted in neurons. Figure 1.1 displays the four parts of the neuron anatomy (cell body, axon, dendrites, and synapse). The cell body consists of the nucleus and receives all the input information and is consequently the origin of all neurotransmitter and action potential activation. Action potential is when a neuron membrane is depolarized beyond its threshold. The axon is the "sending" component that transmits a signal down the neuron to the synapse. Here, in the synapse, the neurotransmitters are released. This is how neurons speak to one another as the neurotransmitter signals are received by the dendrites on nearby neurons. In brief, neurons serve three functions: inhibition, excitation, or neuromodulation. Inhibition

FIGURE 1.1 ■ Neuron Anatomy



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is the process of one neuron releasing an inhibitory neurotransmitter. Excitation is the neuron releasing an excitatory neurotransmitter. Neuromodulation involves one neuron impacting neurotransmission, typically at somewhat of a distance. Many receptors and neurotransmitter systems are involved with substance use disorder and addiction, including dopamine, serotonin, norepinephrine, glutamate, gamma- aminobutyric acid (GABA), acetylcholine, the endogenous opiate system, and the cannabinoid system (Pinel, 2013).

Glia

These are supporting cells of the central nervous system and can outnumber neurons by a margin of 10 to 1. Glia used to be considered the glue of the central nervous system that holds neurons together. However, recent work has uncovered that glia are now known to have substantial influence over various central nervous system processes. Specifically, some glia cells regulate neurotransmission and are involved in the reuptake process for various excitatory neurotransmitters.

Neurotransmitters

Numerous neurotransmitters form the language via which neurons communicate and we live and breathe. At any given moment, all you feel, think, and do can be linked all the way back to these chemicals, neurotransmitters, which are passed between neurons in the synapse. A comprehensive review of the neurotransmitters is beyond the scope of this chapter and text. However, Table 1.1 provides a brief review of the neurotransmitters involved with the substance use disorder and addiction conditions.

BRAIN AREAS ASSOCIATED WITH SUBSTANCE USE AND ADDICTION

The brain is comprised of a few areas with strong connections to the addiction process. These areas handle dozens of functions, and substance use or the recovery from substance use can impact functioning within these areas as well as the many thousands of connections, or tracts, between these structures (Pinel, 2013). Some key areas are discussed in this section.

Brain Stem

The hindbrain is made of the cerebellum, pons, and medulla. Typically, the midbrain, pons, and medulla are all tied together and described as being the brain stem (Pinel, 2013). The brain stem is theorized to handle such functions as motor control, language, attention, fear and pleasure regulation, the regulation of cardiac and respiratory function, regulation of the central

TABLE 1.1 ■ Neurotransmitters Relevant to Substance Use Disorders

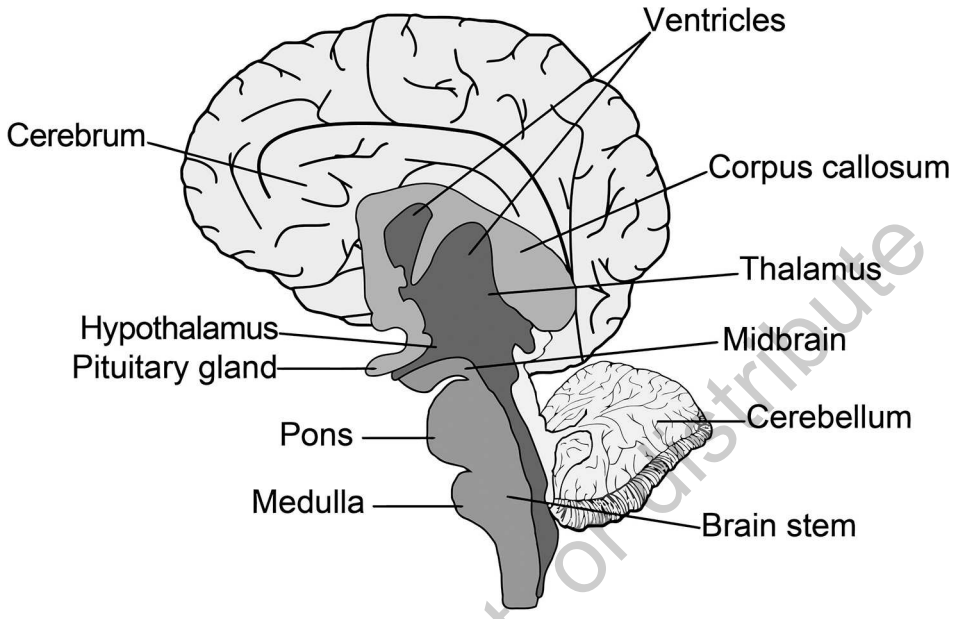
Neurotransmitter	Distribution in the Central Nervous System	Functions Affected	Drugs That Affect It
Dopamine	Midbrain, ventral tegmental area (VTA), cerebral cortex, hypothalamus	Pleasure and reward, movement, attention, memory	Cocaine, methamphetamine, amphetamine. In addition, virtually all drugs of abuse directly or indirectly augment dopamine in the reward pathway
Serotonin	Midbrain, VTA, cerebral cortex, hypothalamus	Mood, sleep, sexual desire, appetite	MDMA (ecstasy), LSD, cocaine
Norepinephrine	Midbrain, VTA, cerebral cortex, hypothalamus	Sensory processing, movement, sleep, mood, memory, anxiety	Cocaine, methamphetamine, amphetamine
Endogenous opioids (endorphin and enkephalin)	Widely distributed in brain but regions vary in type of receptors, spinal cord	Analgesia, sedation, rate of bodily functions, mood	Heroin, morphine, prescription painkillers (oxycodone)
Acetylcholine	Hippocampus, cerebral cortex, thalamus, basal ganglia, cerebellum	Memory, arousal, attention, mood	Nicotine
Endogenous cannabinoids (anandamide)	Cerebral cortex, hippocampus, thalamus, basal ganglia	Movement, cognition, and memory	Marijuana
Glutamate	Widely distributed in the brain	Neuron activity (increased rate), learning, cognition, memory	Ketamine, Phencyclidine, Alcohol
Gamma-aminobutyric acid (GABA)	Widely distributed in the brain	Neuron activity (slowed), anxiety, memory, anesthesia	Sedatives, tranquilizers, alcohol

Source: National Institute on Drug Abuse (2017).

nervous system, and the maintenance of consciousness. Figure 1.2 shows the location of the brain stem, cerebellum, pons, and medulla.

Anatomically, the brain stem is the most interior and primitive brain area. Several components of the brain stem are theorized to be involved with substance use disorder and addiction, including the ventral tegmental area (VTA), substantia nigra (SN), and dorsal raphe nucleus

FIGURE 1.2 ■ Anatomy of the Human Brain, Showing Brain Stem and Other Areas



(DRN). The VTA is involved in the substance and natural reward circuits of the brain and is critical for cognition and emotion (Pinel, 2013). In addition, the VTA neurons project to several other key brain areas relevant to substance use disorders and addiction, such as the prefrontal cortex (PFC). The SN plays a role in reward-seeking and learning. The DRN also contributes to learning and memory functions, as well as playing a role in affect (Holtz, 2010).

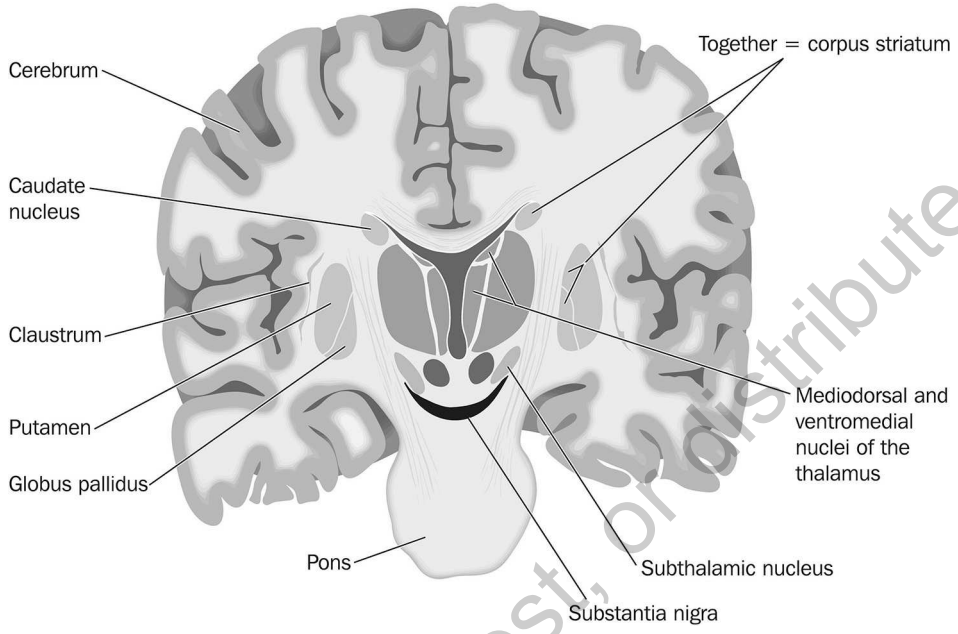
Basal Ganglia

The basal ganglia sits between the brain stem and cortex and consists of areas relevant to substance use disorder and addiction. The nucleus accumbens (NAc) contributes to the cognitive processes of motivation, pleasure, reward, and reinforcement (Pinel, 2013) as well as playing a role regarding responses to novel stimuli (Holtz, 2010). The amygdala is involved with memory, decision-making, and emotional processes, specifically the consolidation of emotional memories (Pinel, 2013). Figure 1.3 shows the location of the basal ganglia and associated areas within the brain linked with addiction, such as the subthalamic nucleus (Pellouox & Baunez, 2013) and the caudate nucleus (Bohbot et al., 2013).

Cortex

This is the outermost and most advanced brain area. Pinel (2013) and Holtz (2010) review how the cortex consists of several areas linked with substance use disorders and addiction; these areas are the anterior cingulate cortex, dorsolateral prefrontal cortex, orbitofrontal cortex, insular

FIGURE 1.3 ■ Drawing of the Brain Showing the Basal Ganglia and Thalamic Nuclei

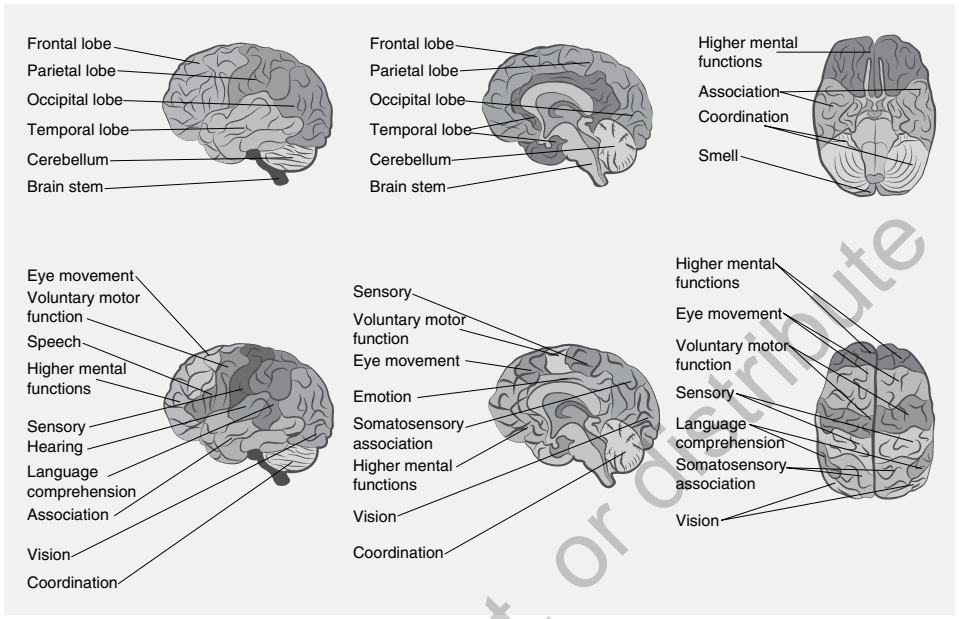


cortex, and the hippocampus. The anterior cingulate cortex is responsible for such functions as reward anticipation, empathy, emotion, impulse control, and emotion. It helps to modulate emotional responses. The dorsolateral prefrontal cortex is involved in executive functions, which include working memory as well as cognitive flexibility and planning. This area is frequently discussed as relevant to problems with attention and motivation. In addition, this area is activated in risky or moral decision-making processes involving a cost–benefit analysis of several potential decisions. The orbitofrontal cortex may be involved with linking affect to reinforcement as well as the decision-making processes. The insular cortex is associated with exposure to substance-related triggers; this brain area is involved with a host of functions, including the processing of negative emotional experience (Critchley et al., 2004) as well as the integration of sensory input from multiple sources (K. S. Taylor et al., 2009). Finally, the hippocampus plays a critical role in the integration of emotion and memory as well as playing an influence on long-term memory (Pinel, 2013). Figure 1.4 displays the general human brain anatomy and the functions associated with these areas. Just about all of these areas are in some manner influenced by the addiction process, whether during the active use and/or recovery periods.

Dopamine Pathways

As discussed in Table 1.1, dopamine plays a pivotal role across most substances discussed in this text. There are four dopamine pathways in the brain, with the first three being involved in

FIGURE 1.4 ■ Human Brain Anatomy, Function Area, and Mind System



the substance use disorder and addiction process: mesolimbic, mesocortical, nigrostriatal, and tuberoinfundibular. Each will be discussed briefly here.

The mesolimbic pathway runs between the ventral tegmental area to the nucleus accumbens. However, the dopamine cells of the mesolimbic pathway also project to other areas relevant to substance use disorders and addictions, including the amygdala, bed nucleus of the stria terminalis (BNST), and lateral hypothalamus. The mesocortical pathway extends to the frontal lobes and includes several structures believed to have an important role in the addictive process, such as the dorsolateral prefrontal cortex (Dagher et al., 1999), the orbitofrontal cortex (Elliott, Dolan, & Frith, 2000; Kringelbach, 2005), and the anterior cingulate (Bush et al., 2000). For example, the prefrontal cortex facilitates the control of impulsive behavior. Any alteration in this area, such as via substance use, could lead to increased impulsivity and possible substance use. The third dopamine pathway in the brain is the nigrostriatal pathway, which primarily controls movement and may explain some motor deficits in substance using individuals (Gardner & Ashby, 2000). The fourth pathway, the tuberoinfundibular pathway does not play a role in substance use and addiction.

PSYCHOPHARMACOLOGY: BASICS

Route of Administration

The specific format in which a substance is administered will have a major impact on four key concepts: (1) the speed in which the substance begins influencing the body, (2) how this substance is distributed throughout the body, (3) the intensity of the effect, and (4) the speed

in which any side effects will be experienced. There are a few ways that substances are administered, with most being divided among the enteral or parenteral routes.

Enteral Substance Administration

Substances administered via the enteral route enter into the bloodstream through the gastrointestinal system (Stahl, 2013). This route is most typically administered orally via a liquid or tablet. Another enteral route is sublingual via the blood-rich tissues found under the tongue.

Parenteral Substance Administration

This route entails the injection of the substance directly into the body (Stahl, 2013) and is typically the preferred route if a rapid onset of substance effects is wanted. There are a few types of parenteral administration. Subcutaneous administration is the injection of a substance right below the skin surface, with the advantage being slow absorption into the body. This process (called "skin popping" by illegal substance users) thus provides the substance available for absorption over a period of time. Intramuscular administration injects the substance directly into the muscle. Since muscles are blood-rich, the intramuscular process administers the substance into the body quicker than the subcutaneous method. The intravenous method injects the substance direct into the vein, thus providing a direct and immediate access to the bloodstream (Stahl, 2013).

Other Routes of Administration

Transdermal administration involves the absorption of a substance through the skin surface. Intranasal administration involves "snorting" the substance through the nose, allowing the substance to enter the system through blood-rich sinus tissues. Inhaling administers the substance into the system via passing through the microscopically thin (1/100,000th of an inch) layer between the air and the circulatory system via the blood-rich tissues in the lungs (Stahl, 2013).

Distribution and Transport

DeVane (2004) underscores the case-by-case nature of substance distribution, with the process influenced by countless variables such as gender, age, muscle tissue ratio, or degree of hydration at time of substance administration. Substances also need to move to a site of action in the body. However, some types of chemicals move more freely than others. For example, water-soluble compounds can mix easily with blood plasma and can thus be easily moved throughout the body. Alcohol is an example of a water-soluble compound. Other compounds must bind with fat molecules in order to move throughout the body. These compounds are called lipid-soluble. While a lipid-soluble compound is bound to the fat molecules the compound cannot be eliminated from the body, but it also cannot produce the intended effect. Thus, the compound must detach from the lipid molecules and enter the bloodstream to reach the site of action desired.

Biotransformation

Biotransformation is usually focused in the liver, although other organ(s), such as the kidneys, might also be involved. There are essentially two forms of biotransformation: zero-order

biotransformation and first-order biotransformation process. In the zero-order biotransformation process the biotransformation mechanism(s) quickly become saturated if a large amount of the substance is taken. Despite the potentially heavy concentration of that substance in the blood, only a set amount can be biotransformed each hour. For example, alcohol works from a zero-order biotransformation process in that if an individual drinks alcohol more rapidly than the body can metabolize it, intoxication occurs. First-order biotransformation process entails a set percentage of the substance biotransformed each hour independent of the substance concentration in the blood. Many substances are biotransformed through a first-order biotransformation process (Doweiko, 2015; Pinel, 2013).

The First-Pass Metabolism Effect

The human digestive tract is designed to filter all content first through the liver. This is called the first-pass metabolism effect (DeVane, 2004). Thus, any toxin ingested can be isolated and the biotransformation process started before any damage occurs. However, one consequence of the first-pass metabolism is that the effectiveness of oral medication is diminished. For instance, much of an orally administered dose of morphine is biotransformed by the first-pass metabolism before it reaches the site of action in the brain, thus greatly limiting the analgesic effectiveness; this is why morphine is administered via IV (Doweiko, 2015).

Elimination

Biotransformation changes the chemical structure of a substance so that it becomes more water soluble and then is removed from the circulation by the organs filtering the blood. Kidneys are the most common blood filtering organ, but the lungs, sweat glands, and biliary tract are sometimes also involved in the process of drug elimination (Doweiko, 2015; Pinel. 2013). Depending on the substance, this process might take hours or even days. Again, the ultimate goal of the biotransformation process is to allow the enzymes to transform the substance molecule(s) into a water-soluble metabolite that can be easily eliminated from the body.

Half-Life

The concept of a half-life provides an estimate of substance effectiveness, duration of the effect, and the length of time that the substance will remain in the body. There are several different types of half-life, with each discussed briefly here.

The distribution half-life is the length of time a substance takes to move into the general circulation post administration. For example, this is critical in pain management as the physician would obviously want a medication to quickly get into the circulation to reach the site of action to control pain. Another example is Narcan (naloxone), which is used to treat opiate overdoses. Clearly, the emergency condition of an overdose mandates a substance having rapid action once administered. Cloos (2010a, 2010b) reviews the two subtypes of distribution half-life. The alpha half-life is the period after the peak blood concentration for the substance. The beta half-life is the amount of time for the concentration to decline as the substance biotransforms

and evacuates the body. Beta half-life is the criteria used to classify benzodiazepines as short, intermediate, or long-acting.

The therapeutic half-life is the time for the body to inactivate 50% of a substance. It is usually linked to a single dose of a substance, but regular dosing can alter the therapeutic half-life of the substance. The elimination half-life is the time required to eliminate 50% of a single dose of the substance from the body (Doweiko, 2015).

Tolerance/Neuroadaptation

Tolerance is a shortened duration and decreased intensity of substance effects after repeated and chronic administration of the substance (i.e., the neurons have adapted to the presence of the substance in the system). Doweiko (2015) notes that if the substance is prescribed by a physician, this process is called neuroadaptation though it is called tolerance in the language of illegal substance use. There are a few types of tolerance, which will be addressed here.

Metabolic tolerance involves the body becoming more efficient at biotransforming the substance. For instance, many individuals with use disorders (e.g., alcohol, heroin) report the need to administer more of the substance in order to reach the same degree of intoxication. Behavioral tolerance involves the brain and body appearing normal despite the heightened amount of substance in the system. Cross tolerance occurs when two or more substances focus on a similar receptor site. For example, alcohol and benzodiazepines share a receptor site for the inhibitory neurotransmitter GABA. Thus, as the drinker grows tolerant of alcohol, that tolerance will cross over to benzodiazepines, thus limiting their effectiveness even if there is no history of prior benzodiazepine use (Pinel, 2013; Stahl, 2013).

The Blood–Brain Barrier

Twenty percent of the blood pumped through the circulatory system makes it to the brain. Consequently, the brain is vulnerable to toxins delivered via the bloodstream. As a protection, the brain has a circulatory system of various cells (endothelial, pericytes) that operate a selective screening process. Of note, lipid-soluble molecules can pass through the endothelial cells. This blood–brain barrier weakness plays an important role in addiction as numerous substances are lipid-soluble (Holtz, 2010).

INTERFACE BETWEEN SUBSTANCE USE, NEUROPSYCHOLOGY, AND PSYCHOPHARMACOLOGY: BASIC CONSIDERATIONS

Substance use disorder and addiction can be classified as a chronic disorder, primarily located in the brain, that impacts cognition, emotion, and behavior (Goldstein & Volkow, 2002). However, this text is a practical and applied exercise. No counselor needs to know everything (as that is impossible). However, a counselor must know what is needed to be known, including where to seek additional information. As I am not a neuropsychologist or psychopharmacologist, my knowledge and experience with the issues reviewed in Chapter 1 focuses on the application of these concepts in the counseling process. If seeking more detailed reviews of these

issues, please consult the numerous quality sources that provide a detailed neuropsychological and psychopharmacological overview on substance use disorder and addiction (e.g., J. S. Meyer et al., 2022; Volkow et al., 2018).

In the following sections, neuropsychological issues relevant to specific substance use (and some process addictions) are reviewed from a counseling perspective of influences on client thought, emotion, and/or behavior.

Alcohol

Numerous cognitive deficits are documented in clients with alcohol use disorder (e.g., Fama & Sullivan, 2014). Two of the more severe examples of adverse influences of alcohol on cognition are Wernicke-Korsakoff syndrome (WKS) and alcohol-induced dementia. WKS is a common alcohol-induced cognitive impairment and caused by a lack of vitamin B1 (thiamine) in the brain as a result of malnutrition. WKS primarily presents as a dramatic decline in memory capability while other cognitive properties remain unchanged (Kopelman et al., 2009). Alcohol-induced dementia, caused by overconsumption of alcohol, produces severe cognitive impairments in such areas as executive functioning and emotion regulation (Asada et al., 2010).

Particularly relevant to counseling, chronic alcohol drinkers (especially those who would quality for a *DSM-5-TR* alcohol use disorder diagnosis) typically demonstrate significant neuron loss in the frontal cortex area. This area governs working memory, attention, mood regulation, and various skills involved in judgment and risk-taking (Sullivan & Pfefferbaum, 2005). Pitel et al. (2010; as reviewed in Fama & Sullivan, 2014) note a pattern of cognitive deficits as heterogeneous and dependent on factors such as (but not limited to) age, gender, drinking history, and concurrent psychiatric or medical diagnoses. Executive functioning deficits are also linked with relapse, and these executive functioning deficits remain well into extended-term alcohol recovery (Fama & Sullivan, 2014). Other neurological deficits are also relevant. For instance, Fein and colleagues (2006) note the lasting visuospatial deficits, which impact the ability to focus on relevant stimuli for a task. In addition, critical deficits exist in the social-cognition arena where Townshend and Duka (2003) underscore the difficulty in emotional processing for those with an alcohol use disorder history, where individuals responded intensely to facial expressions of fear but showed deficits to anger and disgust expressions. Similarly, Monnot et al. (2002) found deficits in individuals with alcohol use disorder in recognizing the emotional tone in conversation. Memory, however, seems to produce mixed results. For instance, Nöel et al. (2012) noted how recovering individuals struggle with memory coding and retrieval issues, whereas Fama and Sullivan (2014) found that both remote (information from months ago) and implicit memory (learning from prior exposure to the content) for this population remains intact.

Cocaine

Literature suggests cocaine use leads to changes in several brain areas, including the prefrontal cortex (Franklin et al., 2002) and anterior cingulate cortex (Kaufman et al., 2003; C. R. Li et al., 2008). Sudai et al. (2011) discusses how cocaine use may inhibit new cell development in the

hippocampus, thus impairing memory. In addition, cocaine use may cause new extensions on dendrites (T. E. Robinson & Berridge, 2001). More dendrite branches in the NAc may collect a greater volume of nerve signals coming from areas such as the hippocampus and amygdala. The additional linkages to the hippocampus and amygdala may explain the intense craving paired with cocaine-associated memories (Nestler, 2005).

R. Hester and colleagues (2006) found a strong attentional bias for cocaine-related words and visual stimuli, which seems to highlight the role of attention as involved in the neuropsychological pathology linked with cocaine use and how this diminished attention may be a variable to predict cocaine relapse (Verdejo-Garcia, 2014). Other issues include prefrontal and temporal brain region changes (e.g., reduced gray matter) potentially contributing to memory deficits (Hulka et al., 2013), poor facial emotion expression (Fernandez-Serrano et al., 2010), and executive functioning deficits predicting poor treatment engagement and poor treatment outcomes (Verdejo-Garcia, 2014).

Opioids

Soyka et al. (2011) highlights that opiates (such as heroin) are known to impair cognitive functioning with specific impairments to the frontal cortex and hippocampus. Specific to heroin, Pau and colleagues (2002) found that heroin addiction has a negative effect on impulse control but not on attention and mental flexibility/abstract reasoning. Other studies document the deterioration of the brain's white matter, which may impact decision-making abilities, behavior regulatory ability, and responses to stress (Q. Li et al., 2012). Overall, as the abstinence period lengthens, some see a return to better functioning for verbal memory but not inhibitory control or psychomotor function. However, the research on neurological recovery following opiate use disorder is mixed (Rass et al., 2014).

Cannabis

There still exists a debate regarding specific neuropsychological impairment due to cannabis use disorder, though some evidence indicates possible problems with daily life tasks (Cattie & Grant, 2014). Two meta-analyses that examined lasting effects of cannabis found deficits in learning, memory, verbal language, and various executive functions (I. Grant et al., 2003; Schreiner and Dunn, 2012).

Methamphetamine

Numerous neuropsychological deficits in long-term users of methamphetamine are noted in the literature, including abstract reasoning, cognitive flexibility (Scott et al., 2007), and behavioral regulation (Kim et al., 2005). Most individuals with methamphetamine use disorder show behaviors common for those with frontal systems dysfunction, such as impulsivity, apathy, and sensation-seeking (Iudicello et al., 2014). Furthermore, others have discussed how the risky decision making and poor judgment demonstrated may be linked to the toxic effects of methamphetamine on the dopaminergic system (Paulus et al., 2005).

Rippeth et al. (2004) found between 33% and 50% of all individuals with methamphetamine use disorder experience learning and memory issues. Iudicello et al. (2011) found prospective memory (remembering to perform an action at some point in the future based on a time period or in response to a specific environmental cue relevant to daily life tasks, such as adhering to a medication schedule or attending scheduled counseling sessions) failures in those with a chronic methamphetamine use history. In addition, failure to sustain long-term attention may be linked to damaged areas such as the anterior cingulate cortex (London et al., 2004; McKetin & Mattick, 1998). Other deficient brain areas (e.g., prefronto-limbic) may be responsible for the difficulties with facial affect recognition (Iudicello et al., 2014).

Inhalants

Tagaki and colleagues (2014) note the limited scope of inhalant research, thus impeding a fuller knowledge of the neurological deficits lasting from inhalant use. However, some work has found that chronic inhalant use has serious neurological and neuropsychological effects, likely due to damage of neuronal membranes (Meadows & Verghese, 1996). Inhalants cause brain stem dysfunction and a variety of motor, cognitive and sensory deficits (Rosenberg et al., 2002). Symptoms could include irritability, tremor, ataxia, slurred speech, or decreased visual acuity, and areas of attention and executive function difficulties (Tagaki et al., 2014).

Sample Process Addictions: Gambling and Sex

Process addictions and substance-related addictions share similar neurological traits. One key difference is that the substance-related disorders typically cause some degree of damage and/or change to the brain as a result of repeated exposure to the substance. Though dissimilar in that manner, the process addictions do have a neurological foundation of relevance for counselors who encounter these individuals. Two common process addictions, gambling and sexual behavior, will each be briefly addressed.

Pathological gambling is associated with frontotemporal dysfunction (Brand et al., 2005) and shows impaired attention and concentration abilities (Forbush et al., 2008) as well as elevated levels of impulsive behavior and novelty seeking coupled with diminished levels of self-directedness and cooperation (Forbush et al., 2008). However, despite pathological gambling consistently associated with a blunted mesolimbic-prefrontal cortex activity to nonspecific rewards but increased activity when exposed to gambling-related stimuli, little is actually known about the neuropsychological components of impulsivity and decision making in people with pathological gambling (van Holst et al., 2010).

Drug cue reactivity and craving studies of nicotine, cocaine, and alcohol highlight the role of some key brain areas, including the ventral striatum, dorsal anterior cingulate and amygdala (Kuhn & Gallinat, 2011). In regard to compulsive sexual behavior, Voon et al. (2014) found that these regions were activated during viewing of sexually explicit materials across the groups with and without compulsive sexual behavior, but the activation was stronger for the compulsive sexual behavior group. This finding suggests neurological similarities across the substance use and compulsive sexual disorders.

CLOSING THOUGHTS

Addiction is a brain disease (Hellig et al., 2021; Volkow et al., 2018). I am not a neurologist, neuropsychologist, or psychopharmacologist, but I embrace this paradigm. You do not have to be deeply trained in these areas to appreciate the neurological and psychopharmacological aspects of substance use disorders and addictions. When reading this book, think of how different substances (and process addictions) may impact client behavior in the early, mid, or long-term recovery period when you see them in counseling. The next few chapters will focus on interviewing, assessment, and later counseling practices. Consider the deficits noted earlier regarding executive functioning, attention, memory, emotion regulation and recognition, and impulse control and ask yourself the following questions. (1) Are treatment programs and counselors asking clients who are new to recovery more than they are cognitively capable of providing? For example, a neuropsychological argument could be made that many of the standard practices, such as a multihour intensive outpatient group (attention deficits) or a biopsychosocial assessment requesting self-report for past mental health or addiction behaviors (memory deficits), fall outside of what the client is able to perform (Bates et al., 2002) and thus contribute to client frustration and negative early treatment experiences. (2) Treatment for substance use disorder (especially in early recovery) is focused on new coping skill development. This requires a lot of cognitive work both in session and out (e.g., homework). Is there a mismatch between the client neurocognitive capability and the higher order cognitive processing required for effective early counseling progress (Bates et al., 2013)? (3) Does the profession need a new definition of the construct we currently call *denial*? Neurocognitive deficits resulting in poor planning, rational thought, and impulse control may better explain how and why clients seem to downplay or deny the severity of their problem(s). As Goldman (1995) argued nearly 30 years ago, neuropsychology may better conceptualize what the treatment profession considers denial.

CONCLUSION AND QUESTIONS TO CONSIDER

This is more of an introduction to this section of each chapter. Here, I will offer some concluding thoughts and I will also pose some questions to consider that build exponentially upon one another across the chapters. For example, in all of the remaining chapters, I will pose at least one question to consider here related to that chapter content as seen through the perspective of the neurocognitive and psychopharmacology content reviewed in Chapter 1.

Questions to Consider as You Move On to Chapter 2

Question 1. How much of this content in Chapter 1 did you NOT know (or know as well) prior to reading this chapter?

Question 2. Does this content alter your perceptions of addiction in any manner? If so, how? And, how would that changed perspective influence your treatment philosophy?