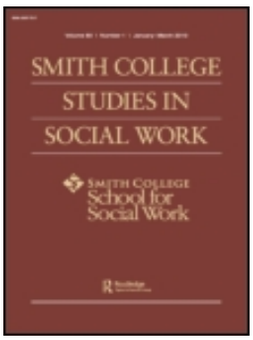

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The Neurobiology of Substance Use Disorders: Information for Assessment and Clinical Treatment

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Neuroscientific research on substance use disorders (SUD) suggests addiction is a complex, multifactorial process, resulting in changes in brain circuits and the brain reward system. This article presents definitions and stages of addiction, highlights of known research on SUD's etiology, and an overview of empirically supported integrated approaches to treatment of persons with SUD. Applying current neuroscientific and outcome research to clinical treatment of a patient with alcohol use disorder, a case discussion illustrates the etiology of addiction and the importance of matching appropriate interventions to the patient's stage of addiction and evolving recovery needs. Special emphasis is placed on attending to the physiological cognitive symptoms evident in early recovery. Five recommendations are proposed for clinical treatment of SUD.

KEYWORDS substance use disorder, neurobiology of addiction, etiology of addiction, alcohol use disorder treatment

Neurobiological research on the study of addictions suggests that substance use disorders (SUD) emerge from a complex interplay of cognitive, behavioral, environmental, genetic, and physiological factors, which lead to underlying changes in brain circuits and the brain reward system (Koob,

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2010; Pihl & Stewart, 2013). Since the founding of the National Institute on Alcohol Abuse and Alcoholism (NIAAA) more than 40 years ago, there has been increased emphasis on the effects of drugs and substances on brain functioning and on how neuroscience can inform clinical treatment (Koob, 2010). This wealth of research has led to information suggesting why some individuals develop an addiction, whereas others do not. Nevertheless, there is ongoing discussion about what neurobiology can and cannot tell us about addictions (Kalant, 2009), debate about whether addiction is a brain disease (Levy, 2013), and questions about how neurobiological research can inform best practices and explain treatment outcomes of persons with substance abuse disorders (Koob, 2010; Pihl & Stewart, 2013).

Nonmedical clinicians working with substance users often shy away from understanding the neurobiology of SUD and focus instead on treating common co-occurring behavioral disorders, such as trauma, depression, or anxiety. Some attempt to treat the psychosocial issues that accompany patients with SUD, without understanding the physiological complexities of addiction. Yet neuroscientists have conducted a massive amount of research over the last two decades on the etiology and course of SUD, particularly alcoholism, and this research is increasingly informing treatment. In this article, we present the highlights of current studies on the etiology of SUD, research on SUD treatment, and a case illustration of clinical intervention informed by this research for the treatment of alcohol use disorder (AUD). Definitions of substance use disorders and addiction begin this overview.

ADDICTION AND SUBSTANCE USE DISORDERS DEFINED

In the words of Pihl and Stewart (2013), "The definition of substance use disorders can be sweeping" (p. 242); that assessment is evident in the recent publication of *The Diagnostic and Statistical Manual of Mental Disorders* (5th ed. [*DSM-5*]; American Psychiatric Association [APA], 2013). The *DSM-5* has changed the nomenclature of SUD, dropping previous distinctions between *dependence* and *abuse* and merging these two definitions into a single disorder. Substance use disorders are based on patterns of pathological behaviors and four groupings of criteria (impaired control, social impairment, risky use, and pharmacological criteria). These criteria are related to the use of nine specific substance classes and are coded along a range of severity from mild (two to three symptoms), to moderate (four to five symptoms), to severe (six or more symptoms). In AUD, for example, anyone who meets two out of 11 criteria during a 12-month period is thought to have an AUD. Although there is considerable overlap between the *DSM-5* and earlier editions of the manual, the revised diagnostic terminology reflects recent research by adding *craving* and omitting *legal problems* as criteria. The *DSM-5* (APA,

2013) also removes the word *addiction* as part of the terminology "because of its uncertain definition and its potentially negative connotation" (p. 485).

Despite the elimination of the word addiction from the *DSM-5*, the term is common among clinicians and neuroscientists alike. Generally, *addiction* is defined as a physical dependence that develops through a repeated pattern of large doses of drugs that are self-administered, result in reinforcing psychoactive effects, and lead to difficulty in ceasing use despite the user's strong motivation (Kalant, 2009). Koob (2010), the current director of NIAAA, further defined alcoholism and addiction to other drugs as "a chronic, relapsing disorder" (p. 144) that includes three stages of an addictive process. The first stage is a binge-intoxication stage, in which increasing levels of alcohol and/or other drugs are needed for the brain to feel a positive reward. The second stage is the withdrawal-negative-affect stage, in which the user experiences negative consequences when the substance is discontinued. And, the third stage is the preoccupation-anticipated stage, when the substance user feels an exaggerated craving and motivation to continue substance use. Koob (2010) proposed that these three stages are each "associated with specific changes in the structure and function of various brain-signaling molecule (i.e., neurotransmitter) systems and in the circuits connecting various brain regions to relay information related to a specific function" (p. 144). It is because of these changes in the brain that a chronic substance user "eventually loses behavioral control over drug seeking and drug taking" (Koob, 2010, p. 144). NIAAA's emphasis on research into the underlying neuroscientific mechanisms of addictions has led to the development of new medications for the treatment of alcoholism and to the development of new models for understanding addiction as a brain disease.

Levy (2013) proposed that scientists aim to "elucidate the neuropsychological causes and correlates of addiction" to offer a "compassionate" view of addiction as "a disease that must be treated, not something for which addicts can be blamed" (p. 4). Although addiction clearly has an effect on specific neural pathways that lead to brain adaptation, he argued that it is "misleading" to state that addiction is a brain disease, because "addiction is a disorder of a person, embedded in a social context" (Levy, 2013, p. 4). This is not to say that the sufferer of the disorder is to blame, but that the social environment mediates the development of the disorder and the outcome of effective treatment. Levy (2013) stated, "If we are to understand addiction and respond appropriately to it, we must not focus on just the addicted individual herself, much less her brain. Our focus must be on her, in her social setting" (p. 6). Levy's view is congruent with the growing body of clinical evidence that there is a complex and strong relationship between AUD, stress, and anxiety, demonstrating the important link between social context and neurobiological factors (see Silberman et al., 2009, for overview).

Although we fully accept the importance of social context and believe that psychosocial factors serve as underpinnings or triggers of addictions,

we wish to highlight the basic and applied literature on the neurobiology of SUD and the research on integrated treatment. Areas of neurobiological study have focused on the development of addictions (e.g., the role of genetics, the role of dopamine and the reward system, and the role of neurotransmitter signaling causing tolerance and cravings) and on how this body of research informs multidimensional approaches to treatment. The following findings emerge from these areas of study.

HIGHLIGHTS OF NEUROBIOLOGICAL RESEARCH

Research on Etiology of Addiction

Genetic studies

For centuries, it has been common knowledge that "alcoholism runs in families" (Kalant, 2009, p. 784), leading clinicians and persons with SUD to believe that addiction is genetically based. Indeed, family, twin, and adoption studies show that SUD has high hereditability and that genes play a role in one's risk for developing addictions (Pihl & Stewart, 2013). Studies estimate that 40% to 60% of an individual's vulnerability to addiction is due to genetic factors (National Institute of Drug Abuse [NIDA], 2010). However, Pihl and Stewart (2013) argued it is impossible to generalize these genetic findings, because biological markers vary from family to family and there have been hundreds, if not thousands, of genes identified as contributory to addictions, perhaps indirectly. Some of these genes, such as ones related to impulsivity, are evident in a number of other behavioral disorders and are not specific to addiction. Kalant (2009) also underscored that "a gene does not encode a trait," and genes "are not necessarily continuously active, i.e., they may be switched on ('expressed') or off under different circumstances" (p. 785).

Clearly, genetic factors are not the sole biological influence on substance use. Once a person begins using alcohol and/or other drugs, these substances affect nearly all brain neurotransmitters in complex ways not fully understood (G. Shean, personal communication, January 28, 2014). The following information describes some of the known neurochemical effects of substance use, yet this overview is inevitably oversimplified and subject to elaboration and revision as more knowledge becomes available.

Dopamine and its effect on rewards

Dopamine is one of the neurotransmitters present in the numerous areas of the brain that regulate feelings of pleasure and reward, as well as emotion, cognition, motivation, and movement. Scientists now recognize that people take potentially addictive drugs because alcohol and other drugs (AOD) act on cells in the brain's limbic system, an action that leads to the release

of dopamine and the subsequent experience of pleasure. This process is involved in the binge-intoxication stage of the cycle of addiction, mentioned earlier (Koob, 2010). Dopamine release motivates a first-time drug user to return to the experience, because the release results in euphoria, increased energy, and generally positive feelings (Kalant, 2009; NIDA, 2010). Although dopamine-containing cells in the brain are activated differentially based on the drug of abuse (e.g., opioid drugs, ethanol, cocaine, or amphetamines), the end result of dopamine activity in the prefrontal cortex of the brain is the same. In the brain's pleasure circuits, the natural rewards of eating and sex actually are dwarfed by the large amount of dopamine produced by drugs of abuse (NIDA, 2010). When dopamine floods the brain circuits, particularly through drugs that are ingested or smoked, the effects also last longer than natural rewards, providing further motivation for repeated use.

Cellular signaling and its effect on tolerance and cravings

Alcohol and other drugs are known to have major effects on the normal communication and functioning of the billions of neurons (e.g., individual nerve cells) in the brain (NIDA, 2010). When AODs affect neural functioning, cells attempt to restore equilibrium by adapting in an opposite manner (Kalant, 2009). For example, dopamine surges caused by AODs eventually result in abnormally lower levels of dopamine in the brain, causing the abuser to feel listless. As a result, the individual takes increasing amounts of the drug to boost the dopamine level (NIDA, 2010). These adaptive changes tend to gradually increase tolerance and physical dependence to overcome the effects of the drug's withdrawal.

During this stage of addiction, the body's hypothalamic-pituitary-adrenal (HPA) response (e.g., the stress-response system) is activated (Koob, 2010). When the normal response of the HPA is disrupted because of alcohol and other drugs, dopamine activity decreases and the hypothalamus or pituitary in the brain's limbic system is activated, releasing a molecule called corticotropin-releasing factor (CRF). Eventually, cycles of drinking and withdrawal lead to a weaker HPA response and a more sensitive CRF stress system (Koob, 2010), creating the withdrawal-negative affect stage of addiction. Acute withdrawal reactions occur when the drug is removed suddenly through a receptor blocker. In other words, the brain's adaptive changes create signaling cascades, defined as neurotransmitter molecular events. Over time, the development of tolerance can compromise the brain's long-term health and cognitive functioning, through significant changes in neurons and brain circuits (NIDA, 2010).

Signal transmissions that use the neurotransmitter glutamate also cause brain circuits in several parts of the brain to be affected (Koob, 2010), including the prefrontal cortex, which is the most evolved structure of the brain, affecting behaviors and regulating executive functioning; the amygdala,

which affects emotional processing; the insula, which affects disgust; and the hippocampus, which affects memory formation and recall. When AOD use decreases the amount of glutamate needed by the brain, the result is impaired cognitive functioning, problems with learning and memory, and, eventually, uncontrollable cravings (NIDA, 2010). Sound judgment and self-control are eroded, moving the user into the preoccupation-anticipation stage of addiction. At this point, users discover it is extremely challenging to stop using AODs, despite motivation or good intentions. Furthermore, long-term use of alcohol and other drugs has been found to lead to adaptations in a person's conditioned habits. For example, environmental cues that are reminders of past experiences can trigger cravings, even without the drug being present. Scientists have found that "This learned 'reflex' is extremely robust and can emerge even after many years of abstinence" (NIDA, 2010, p. 20). In other words, the changes to the brain caused by alcohol and other drugs remain, long after an individual stops using the substance.

Multifactorial interactions

Although acknowledging the importance of specific empirical findings emerging from neuroscience, researchers urge caution to avoid a reductionist viewpoint, because of the complexity of neurobiological processes and the multiplicity of factors that trigger neural mechanisms. Many researchers are currently taking a modular approach to understanding the brain, recognizing that the function of a brain region is not due to a specific brain abnormality but to interactions of various brain patterns in a complex network (Shean, 2010). For example, the neurobiological deficits scientists believe are related to AOD are probably distributed throughout many, if not all, areas of the brain, rather than in one localized region. Furthermore, understanding the mechanisms of how one neuron is linked to another and how that mechanism is altered through drug use helps to explain how changes occur in neural functioning, but not why the changes occur (Kalant, 2009). The reward system is actually "a convenient label rather than literal fact, and it provides no insight into the reasons why some drug users become addicted while the great majority of users . . . never pass from use to compulsive use" (Kalant, 2009, p. 784). Numerous other neurotransmitters beside glutamate also interact with dopamine in a complex manner, leading to different cognitive experiences and positive and negative reinforcing effects over time. Finally, it is critical to note that genetic vulnerabilities interact with environmental factors, such as stress, to lead to addiction. In other words, Kalant (2009) said, addiction is "a behavioral disorder generated within an extremely complex interactive system of drug, individual use, environment and changing circumstances. This is no longer the terrain of pharmacology or neurobiology or psychology or sociology, but an amalgam of all of them" (p. 786). Pihl and Stewart (2013) agreed that neurobiological

research confirms that SUD is a "multifactorial, multilevel, interactional process" (p. 256) and has "a potentially large number of distinct etiological pathways" (p. 251). They stated, "Complexity is the rule. Which drug, used how and how much, with whom, under what conditions, and most significantly, by whom, all impact the course of the disorder" (Pihl & Stewart, 2013, p. 244).

Clearly, this complex interaction of factors is compounded in co-occurring conditions, such as SUD and major depression (Niciu et al., 2009), SUD and anxiety disorders (Silberman et al., 2009), and SUD and trauma (Brady, Back, & Coffey, 2004). According to Brady et al. (2004), epidemiological studies reveal a particularly high prevalence of SUD among individuals with PTSD; 36% to 50% of individuals seeking treatment for SUD have a lifetime prevalence of PTSD, and 25% to 42% have post-traumatic stress disorder (PTSD) at the time of treatment. Longitudinal studies on the PTSD–SUD link suggest that people use substances to escape painful symptoms by self-medicating. With growing evidence that strong neurobiological commonalities exist between the two disorders, the question is whether chronic substance abuse causes changes in the physiological pathways that then make an individual susceptible to PTSD, or if traumatic experiences, especially in early childhood, make an individual more vulnerable to SUD (Brady et al., 2004). Understanding the interaction of these dynamic processes has implications for treatment.

Research on Treatment of Addictions

Psychopharmacology

Just as there are multiple, interactive pathways leading to addiction, there are numerous interventions that address these different pathways. Research on psychopharmacology and brain circuitry suggests a number of medications that are effective for treatment of SUD. Disulfiram, commonly known as antabuse, is a well-known medication that provides aversive treatment for persons with chronic alcoholism (Pihl & Stewart, 2013). It blocks the body's processing of alcohol by causing an unpleasant reaction when alcohol is consumed. The patient experiences nausea, elevated heart rate and respiration, and vomiting—similar to the effects of a severe hangover. A second major biological treatment is antagonist treatment, medications that cancel cravings and the pleasurable effects of the abused substance by blocking the release of dopamine. One such medication is Naltrexone, which is supported by NIAAA clinical trials (Koob, 2010) and is reportedly most effective when used in conjunction with psychotherapeutic treatment approaches. The third form of biological treatment is agonist substitution, which offers a similar drug as a substitute for the more harmful abused drug. Methodone is an example of an agonist substitution used in the treatment of opiate dependence, whereas

nicotine gum or patches are examples used in the treatment of cigarette smoking (Pihl & Stewart, 2013). Other medications may be used to manage withdrawal symptoms during detoxification. Acamprosate is an Food and Drug Administration (FDA)-approved medication for treating alcohol dependence to reduce symptoms that accompany withdrawal, such as irritability, depression, restlessness, and anxiety (NIDA, 2009). Although researchers are actively engaged in developing effective biological treatments for SUD, Koob (2010) pointed out that "none of the existing medications are effective in all patients, and additional agents need to be identified and developed that allow for effective treatment of additional patient subgroups" (p. 149).

Social context interventions

Structured "12-Step" self-help groups such as Alcoholic Anonymous (AA) or Narcotics Anonymous (NA) have long been considered "the most popular model for the treatment of substance abuse" (Pihl & Stewart, 2013, p. 261). These 12-Step programs provide participants with needed structure and behavioral techniques that address re-learning and ways to avoid triggers. In addition, 12-Step self-help groups attend to social context, one of the central factors interacting with other variables in the development of addiction (Levy, 2013). The groups reinforce the importance of friends, family members, and feeling a part of a social community of people who share the struggle of recovering from SUD. Research suggests that group treatment (professionally-led and 12-Step groups) forms the most prevalent treatment approach for SUD (Weiss, Jaffee, de Menil, & Cogley, 2004). An analysis of 24 treatment outcome studies on the effectiveness of group treatment for SUD showed no differences in effectiveness between group and individual treatment or between types of group therapy. However, there was some indication from these studies that the effectiveness of "treatment as usual" was enhanced by the addition of specialized group therapy.

Individual psychotherapy

Motivational Interviewing (MI) and Cognitive-Behavioral Treatment (CBT) are two of several forms of individual psychotherapy that have been shown to be effective for particular stages of substance use or for persons with SUD and co-occurring disorders. MI is a collaborative, person-centered model of practice that is an empirically validated intervention considered effective for SUD treatment (Substance Abuse and Mental Health Services Administration [SAMHSA], 2013a). MI is especially beneficial in the early stages of treatment for persons who have co-occurring disorders and may not accept they have SUD, such as someone who is self-medicating with alcohol due to anxiety or bereavement. Nonconfrontational, MI methods enable the clinician to

build a therapeutic relationship while assessing and exploring the patient's understanding and/or acceptance of the disorder. MI may be used alone, but research suggests it is most effective for treating SUD when it is a prelude to other forms of treatment or used in conjunction with them (Pihl & Stewart, 2013).

Once a patient accepts that he or she has a SUD and is motivated to change, CBT is often used to address the distorted thinking that accompanies substance abuse and behaviors that lead to addiction. Based on learning theory, CBT is designed to enhance coping skills, increase self-efficacy, and alter thought processes about the risks and rewards of substance use. CBT is beneficial in helping patients deal with stress, a major factor that interacts with neurobiological variables and triggers cravings. According to Pihl and Stewart (2013), "CBT is among the most effective commonly employed treatments for alcohol use disorders" and "there is mounting evidence for the efficacy of CBT in the treatment of nicotine dependence" (p. 260).

Integrated treatment and treatment matching

The interaction of neurobiological factors with psychosocial factors supports interventions that combine psychopharmacology, social context interventions, and various forms of individual psychotherapy. Integrated treatment is especially critical for individuals who have co-occurring mental and substance abuse disorders. One treatment model called integrated treatment for co-occurring disorders is an empirically supported intervention supported by SAMHSA (2013b) for the treatment of co-occurring mental and substance use disorders. This model incorporates multiple approaches, including MI, CBT, and 12-Step groups, and is considered a "stage-wise treatment." Based on the belief that individuals go through different stages in the recovery process (e.g., engagement, persuasion, active treatment, and relapse prevention), the model recognizes that interventions should be matched to the recovery needs of the particular patient.

Other studies on treatment integration have examined matching to the particular co-occurring disorders, such as SUD and PTSD, in addition to the stages of recovery. For example, Brady et al. (2004) reported some evidence of the effectiveness of integrated treatment that attends to PTSD and SUD simultaneously, rather than individually, regardless of the patient's stage of recovery. This is in contrast to the traditional view that single-model therapies or sequential therapies (e.g., first for SUD, then for PTSD) work best. Although exposure-based PTSD therapy has been supported as effective in helping patients confront memories or situations that remind them of the trauma, until recently, exposure-based therapy was not considered beneficial for patients with PTSD and SUD due to concern about precipitating relapse for the patient.

The role of attachment processes in treatment

Finally, it is well known that attachment plays a major role in affect regulation, interpersonal functioning, and the structural development of the hippocampus during the first three years of life (Allen, 2001; Mikulincer & Shaver, 2007); that early attachment relationships are associated with adult attachment styles and relational patterns (Cassidy & Shaver, 2008); and that interpersonal relationships and subjective experiences affect memories, emotion, and self-regulation throughout life (Siegel, 2007, 2012). Difficulties with emotion regulation also are associated with substance abuse (Dozier, Stovall-McClough, & Albus, 2008), and Flores (2004) has called addiction an attachment disorder. Similarly, in AA, it is said that alcoholism is a disease of isolation. Although no known studies have evaluated a specific attachment-based treatment model for SUD treatment, several findings about attachment-informed treatment are relevant to this discussion (Slade, 2008).

Attachment research on adult treatment in general suggests that patients with insecure working models of attachment are more likely to form insecure specific attachments to their therapists, which can be challenging for therapists who often begin to match the style of the patient. Yet research suggests that therapists who are secure and can respond flexibly and counter to their patients' styles yield better treatment outcomes (Mikulincer & Shaver, 2007). In other words, a secure therapist ideally should use cognitive interventions when faced with an insecure anxious, preoccupied patient and use emotionally focused interventions with an insecure dismissing patient. Responding counter to the patient's insecure attachment style enables the patient to begin to establish an earned security, that is, to transition from insecure to secure attachment. Based on findings from their study on attachment and affect regulation among substance abusers, Thorberg and Lyvers (2010) suggested that a goal of SUD treatment should be to enable patients to establish earned attachment security by revising the patient's internal working model for attachment.

Finally, many scholars and clinicians (Doctors, 2008; Fonagy, 2003; Schore, 2002; Slade, 2008) have integrated attachment theory with self-psychology to inform clinical practice, and several have written about the treatment of substance use disorders through self-psychological methods of intervention. Attuned to the substance abuser's stage of recovery, self-psychologists Ulman and Paul (1989) proposed that clinicians should "make abstinence and sobriety basic treatment issues in the initial phase of therapy" (p. 131), because this emphasis is ultimately empathic to the patient's needs. In the following case discussion of a patient with AUD, we illustrate attunement to the patient's initial phase of therapy, her need for affect regulation, and the attachment processes that unfold within the therapeutic relationship. The clinical approach presented is based on a model of practice informed by attachment theory and self-psychology, as well as attunement to the patient's evolving recovery needs and the neurobiology of addictions.

APPLICATION TO CLINICAL PRACTICE

The Case of Cheryl

A 30-year-old professional, Cheryl was referred to me (P. Petrash) for psychotherapy by a counselor from her professional organization following a "Driving Under the Influence" (DUI). The counselor initially referred her to AA, but Cheryl was finding it impossible to connect with the program. Uncomfortable in AA meetings, she was unable to hear or benefit from what was being said, could not connect with people in the meetings, and would flee meetings as soon as possible. The counselor recognized that Cheryl needed additional psychotherapy to optimize her treatment of chronic AUD.

At the time of our first interview, Cheryl had been sober several months. What immediately impressed me was her overall presentation. She wore a beautiful designer suit with carefully coordinated stockings that were marred by a large hole in them. I thought to myself that it was too bad she had a "run" in her stockings, but in future meetings, she continued to wear the same stockings with an increasingly larger hole. I wondered how someone could wear such elegant, expensive suits and choose to put on torn stockings. Did she not notice? The second striking feature in her presentation was her voice. Although she spoke about her family as "okay" and described family life as "uneventful," her voice sounded as if someone was trying to strangle her. What was the source of her anxiety? Did she know she was anxious? Finally, Cheryl spoke of her graduate school accomplishments and subsequent career successes, yet she seemed unable to figure out the most basic problems of daily living. I noted the discrepancy between her considerable intelligence and intellectual accomplishments, in contrast to her lack of problem solving ability and, quite frankly, daily living skills.

Cheryl was the oldest of four children born to her mother over the course of 6 years. She described her father as "bright," mentioning his having earned a PhD. The example she gave of her mother as a "good mother" was that "she cooked us a good dinner every night." Reportedly, neither parent was alcoholic, but her mother's sister and several other relatives on her mother's side of the family were alcoholic. Her childhood was "uneventful" until she was in junior high school and the family moved from the Midwest to the West Coast. Afterwards, she felt she never fit in and became isolated. She turned to food for comfort and proceeded to gain 60 pounds, though her mother failed to notice her weight. I noted she did not mention people as a resource.

Cheryl's parents severed ties with her upon graduation from college, expecting her to be independent. Subsequently, she had her first drink following her first panic attack during the process of leaving home. Alcohol proved to be the perfect antidote to her anxiety. For the first time in her life, Cheryl felt "okay" and like "my shoulders dropped." Not surprisingly, her drinking quickly escalated. She found she had a high tolerance and was

capable of out-drinking many of her peers. Using alcohol to medicate her anxiety, her functioning improved for a period of time. She was able to begin dating, complete graduate school, and start a career. However, in fewer than 10 years, her increasing tolerance led to dependence and craving. At the time of her DUI, she was drinking up to a fifth a day, in a struggle to avoid symptoms of withdrawal.

Our initial work together focused on the concrete issues of self-care and on how to relate in an AA meeting. "Do I talk or not talk?" "What do I do if someone invites me for coffee?" "Do I go to a meeting if I am going to be late?" "Have I eaten?" "Am I tired?" As a self-psychological and relationally oriented therapist, I felt drawn to an empathically oriented approach, identifying and responding to her selfobject needs. Clearly, she needed me to be an active participant who would provide her with concrete information, answers to her questions, and directions about issues of daily living—a process that has been described as a "selfobject in action, rather than one merely in thought or fantasy" (Ulman & Paul, 1992, p. 123). Eventually, someone in an AA meeting chose Cheryl as her sponsee, although a new member typically chooses her own sponsor. For Cheryl, the experience of being claimed by her sponsor was initially helpful as it bypassed her inability to ask someone for help. Her sponsor embraced the role of providing information, direction, and problem solving—also functioning as a selfobject in action. She told Cheryl which meetings to attend and then would meet her there and sit with her. Her sponsor was repeatedly reminding her of the AA slogans like "keep coming back," "one day at a time," or "go to a meeting."

With that provision from her sponsor, I broadened my responses to include understanding and mirroring her internal experiences. I was struck by the discrepancy between her facility with language in her professional capacity and her inability to put her emotional life into words. I had to take an active role, asking questions, offering my description of her experience, and clarifying if my mirroring fit for her. Cheryl gradually began to internalize these reflections with me, resulting in her increased self-regulation and self-care. She also began to feel connected to the program and several people in it. Yet eventually, as she remained sober and her sense of self began to consolidate, she came to resent the total accommodation required by her sponsor and wished to begin making some of her own decisions. Her desire for autonomy precipitated an irreparable rupture with her sponsor. At that point, her connection to me, including my mirroring of her wish for autonomy and her frustration with her sponsor, enabled her to remain in the program and to find a more appropriate, collaborative sponsor.

After her support system was solidly in place to maintain a more advanced recovery, we began to focus on her attachment issues. Several major themes emerged from our discussions, which are especially relevant to understanding her functioning and treatment needs. Minimizing her need and affect, she had presented with an avoidant attachment strategy. But,

what became clear was that her experience of early developmental trauma was continuous and resulted in a pattern of disorganized attachment. She expanded her description of her mother with a casual and telling remark: "My mother never made eye contact." Although her mother performed household functions adequately, her limited caregiving responses were traumatizing, leaving Cheryl without the needed vitalizing and soothing responses necessary for the development of a coherent sense of self. As an illustration, Cheryl recalled being hospitalized at age 6 with mononucleosis, reflecting her massive depletion and exhaustion from her efforts to deny her feelings and needs. She remembered her mother visiting her in the hospital, but after seeing the terrified look in her mother's eyes, she told her mother not to visit again. Her mother complied.

As therapy continued to unfold, Cheryl explored her past development and the stressors that triggered her anxiety and alcohol abuse. After many years of treatment, she appropriately terminated her psychotherapy while remaining actively involved in AA. Eventually she also began attending Alanon, a 12-Step program that provided a forum for her to continue to learn how to function in relationships. Cheryl was able to stay sober without relapsing and eventually married someone who provided the interpersonal relationship and support she had not experienced as a child.

Discussion of Cheryl's Treatment

Although Cheryl's experiences are unique to her life course, the development of her addiction and her early recovery are common processes. We present her story to illustrate two main points about the neurobiology of addictions. First, we wish to emphasize that the etiology of SUD is complicated and multifactorial and, second, that therapeutic techniques must match the patient's stage of recovery. With that in mind, we stress that the physiological symptoms of addiction must be the primary focus of early treatment, as Cheryl's recovery demonstrates.

Etiology of addiction

Similar to many individuals with SUD, Cheryl was born into a family with a history of alcoholism, perhaps making her genetically vulnerable to developing the disorder when she discovered alcohol's capacity to soothe her anxiety. She may have been genetically predisposed to anxiety as well, and her early attachment traumas compromised the development of her self-regulatory and self-care capacities, creating significant vulnerabilities to anxiety and dysregulation. Her unfolding family narrative was replete with stories of separation and loss, loneliness and fear, and maternal indifference and abandonment. Without doubt, her mother's lack of attuned responsiveness contributed to Cheryl's difficulties, leaving her struggling to manage

her internal states through dissociation. When we think about the strangled voice she had when she began therapy, we are reminded of Tronick's "Still Face" experiments (Zero to Three, 2007) of the baby's frustrated struggle and ultimate collapse in the face of a nonexpressive mother. This picture is reminiscent of Cheryl's depleted, empty self-experience and the fragmentation of being caught between protest and despair.

We also were struck by the story of the young Cheryl in the hospital. It is understandable that a mother would be frightened visiting her small sick child in the hospital. But, to not return in response to the child's reassurance that she was "okay" reflected a level of fearful anxiety associated with a caregiver who had a disorganized attachment. Cheryl's sparse, concrete language about her mother (e.g., good, because she cooked good meals) revealed interpersonal avoidance and a lack of awareness about her childhood neglect, cues of dismissing attachment. Her language was intermittently incoherent, suggesting signs of disorganized attachment as well, paralleling her mother's attachment. Indeed, the theme of a mother and child relationship marked by incoherence and disorganization was rampant in Cheryl's stories, culminating with the cut-off that a frightened Cheryl felt when she left home, triggering panic and her first use of alcohol to medicate her anxiety. She learned to cope with her anxious fear throughout life by cutting off her affect and expression of need.

In other words, the etiology of Cheryl's addiction seemed to emerge from the interaction of genetic factors that became activated by her early attachment disorganization, making her more vulnerable to stress and loss as she moved into adulthood. Although insecure attachment does not exist for all persons with SUD, the link between attachment disorganization and substance abuse appeared prevalent in Cheryl's case. Alcohol use helped her block the dysregulated affect associated with her disorganization, and she withdrew into alcohol as a means of survival. Although the neurobiological changes caused by alcohol intake are too complex to fully understand, we can assume that the pleasure from the drug-induced dopamine surges became addictive for Cheryl because the pleasure blunted her pain. It is thought that her alcohol use eventually led to decreased dopamine levels and a weakened, more sensitive stress–response system. This phenomenon in turn created increased tolerance for alcohol, uncontrollable cravings, and significant deficits in her cognitive functioning.

Therapeutic matching

Cheryl's story further demonstrates the importance of understanding a patient's level or stage of addiction and matching that knowledge with appropriate treatment. When she was first referred to therapy, the prefrontal cortex of her brain was severely impaired by acute alcoholism, causing damage to her executive functioning. The drug that served initially to regulate anxiety

left her in a constant dysregulated state due to physiological adaptation. As a result, she had abnormally low levels of dopamine—a reaction to alcohol-induced dopamine surges—leaving her listless and tired. The effects of the early developmental trauma further compromised her executive functioning. Cheryl really did not know what to do. The hole in her stockings (matched with designer suits) was just one small example of her impaired judgment, her lack of body awareness, and even her inability to think flexibly to address a problem. She literally needed someone to tell her what to do to function on a daily level.

It was important to match Cheryl's cognitive deficits with a form of treatment that would rebuild her executive functioning and increase her cognitive skills. AA provided the necessary structure and behavioral approach that her cognitive impairments required at that time. For the first two years of recovery, she needed simple, consistent, and repetitive directions to rebuild cognitive skills she lost due to the damage to her brain. Over time, the structure and skill building that AA provided served as a secure base-safe haven for Cheryl. In terms of psychotherapy, Cheryl's cognitive impairments and limited functioning required a supportive, nonconfrontational form of treatment that complemented the messages she was receiving in AA. As Cheryl's experiences were reflected and her affect and words were mirrored, she eventually began to use the therapist as a secure base as well.

We wish to emphasize that the initial approach to treatment integrated cognitive and behavioral techniques to help Cheryl learn new coping skills for dealing with anxiety and maintaining her sobriety. This approach tended to the physiological symptoms of early sobriety and focused on increasing her executive functioning skills, such as judgment, thought processes, and impulse control. It took several years of sobriety and ongoing AA involvement for Cheryl to develop enough cognitive improvement and regulatory capacity to tolerate a more in depth exploration of her life history. The therapist could not address the underlying attachment issues that influenced Cheryl's alcohol use until she learned new ways of managing her stress and the physiological symptoms of her addiction were no longer overwhelming her daily functioning. By the time Cheryl terminated psychotherapy, she had developed a state of earned attachment security, because she had resolved the loss and trauma of her childhood and no longer needed alcohol to mask the painful memories of her past. Despite her significant therapeutic gains, however, she understood that the permanent physiological changes caused by her addiction prevented her from being able to safely drink alcohol again.

SUGGESTIONS AND CONCLUSION

This case discussion and the neurobiological research presented earlier support the value of using a multidimensional framework for understanding the

etiology and treatment of substance use disorders. In addition, the context of psychotherapy—the social context and reason for referral, the patient's interpersonal capacities, the patient's acceptance of the disorder, and the quality of the therapeutic relationship—determines how best to intervene. Keeping this context in mind, we summarize our approach by suggesting the following guidelines we find beneficial for treating patients with alcohol use disorder.

1. Keeping in mind the progression of the addiction, evaluate the patient's stage of substance use and encourage a thorough medical exam. This intervention helps the nonmedical clinician discern the necessity of medication and the appropriate intensity of treatment. Initially, patients in advanced stages of addiction often require ongoing medical supervision to undergo a medically safe detoxification. This can occur in the form of hospitalization, a residential 30-day program, or a structured outpatient day treatment program.
2. Provide structured, active, and direct supportive interventions, especially in the initial stages of recovery; encourage patients to participate in a 12-Step self-help group or a formal treatment program in addition to individual psychotherapy. Based on the level of impairment, the clinician should provide the level of support required to facilitate someone attending a group meeting. If necessary, go with patients to open 12-Step meetings to help them begin the process; use multiple providers, collaborating with all who provide care; and employ multiple treatment modalities, such as group and individual treatment. Such direction and structure provides compensation for the patient's inadequate and damaged cognitive abilities, whereas group and family modalities address the importance of social context. Patients in early recovery do not have the neurocognitive capacities to engage in unstructured, interpretive dynamic treatment.
3. Assess the patient's history of substance use within the context of the patient's family development and the family's substance use, as well as the patient's historical patterns of dealing with stress. This intervention incorporates knowledge that patients often have hereditary vulnerabilities and that stress activates genetic traits. History taking helps to sensitize patients to the connection between their minds and their bodies and to become aware of triggers for relapse. For example, many patients relapse following loss, the death of a loved one, or a particularly stressful work situation, and they must learn new methods of coping with such stress.
4. Use multiple models of individual intervention (e.g., cognitive, behavioral, relational, motivational interviewing) that are interactive, focused on the present, and match what works best with the patient's particular stage of recovery and co-occurring issues. Many patients in early recovery need

clinicians to employ more structured CBT interventions to enhance their cognitive skills and to learn behavioral techniques for managing anxiety and stress. Still other patients may be sufficiently stable physiologically that they can tolerate relational methods of intervention to treat the psychosocial underpinnings of their substance use. Note, however, that no one method is appropriate for all SUD patients at all stages of recovery. When addressing co-occurring issues (e.g., trauma, depression, anxiety), maintain focus on physiological symptoms and triggers for substance use or relapse. For example, most patients are prone to distancing or dissociation when dealing with their complex trauma history, and it becomes essential to keep them focused on the present to avoid retraumatization and relapse.

5. Attend to the patient's extended social context through family or couple's interventions; group interventions; and AA, NA, and Alanon. Although difficulty in spousal or family relationships is a certainty, it is often beneficial for the family members of the patient in early recovery to engage in treatment separate from the identified patient due to the patient's cognitive impairment and reactivity. Groups for family members or Alanon can be particularly beneficial. Such interventions provide support and education for the patient and the patient's significant others and may increase the partner or family's awareness of how they sometimes trigger the patient's repeated use. It is especially important to educate patients and their families regarding the chronic nature of substance use disorders and how relapse may be prevented through awareness of personal triggers and specific methods to address them. Finally, 12-Step groups are vital because they help patients develop friendships and a sense of belonging to a social community, which lessens shame about addiction and reinforces hope that recovery is feasible. Twelve-Step groups also provide the skill building and structure that is essential for early recovery.

We believe these multidimensional guidelines attend to the complex, yet dynamic needs of patients who have SUD. Clinicians should provide treatment that is attuned to the relational needs of these patients, because they often have histories of significant loss, trauma, and disorganized attachments. Further, clinicians who are sensitively attuned to the particular environmental, behavioral, and psychosocial factors shaping patient capacities will be more likely to establish a secure base for treatment and maintain a therapeutic working alliance. However, it is essential to take note of the neurobiological factors influencing the patient's stage of addiction as well, because these factors shape the patient's cognitive abilities and capacities for treatment. With these multiple factors in mind, the unfolding therapeutic process should match the patient's functioning and level of substance use disorder—be it early recovery or long-term abstinence.

REFERENCES

Allen, J. (2001). *Traumatic relationships and serious mental disorders*. New York, NY: John Wiley & Sons.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington, VA: American Psychiatric Publishing.

Brady, K., Back, S., & Coffey, S. (2004). Substance abuse and post-traumatic stress disorder. *American Psychological Association*, *13*, 206–209.

Cassidy, J., & Shaver, P. (Eds.). (2008). *Handbook of attachment: Theory, research, and clinical applications* (2nd ed.). New York, NY: Guilford Press.

Doctors, S. (2008). Notes on incorporating attachment theory and research into self psychological and intersubjective clinical work. *International Journal of Psychoanalytic Self Psychology*, *3*, 34–49. doi:10.1080/15551020701743451

Dozier, M., Stovall-McClough, C., & Albus, K. (2008). Attachment and psychopathology in adulthood. In J. Cassidy & P. Shaver (Eds.), *Handbook of attachment: Theory, research, and clinical applications* (2nd ed., pp. 718–744). New York, NY: Guilford Press.

Flores, P. J. (2004). *Addiction as an attachment disorder*. New York, NY: Jason Aronson.

Fonagy, P. (2003). The development of psychopathology from infancy to adulthood: The mysterious unfolding of disturbance in time. *Infant Mental Health Journal*, *24*, 212–239. doi:10.1002/imhj.10053

Kalant, H. (2009). What neurobiology cannot tell us about addiction. *Addiction*, *105*, 780–789. doi:10.1111/j.1360-0443.2009.02739.x

Koob, G. (2010). The potential of neuroscience to inform treatment. *Alcohol Research & Health*, *33*(1/2), 144–151.

Levy, N. (2013). Addiction is not a brain disease (and it matters). *Frontiers in Psychiatry*, *4*(24), 1–7. doi:10.3389/fpsyt.2013.00024

Mikulincer, M., & Shaver, P. (2007). *Attachment in adulthood: Structure, dynamics, and change*. New York, NY: Guilford Press.

National Institute on Drug Abuse. (2009). *Drug facts: Treatment approaches for drug addiction*. Retrieved from http://www.drugabuse.gov/publications/drugfacts/treatment-approaches-drug-addiction

National Institute on Drug Abuse. (2010). *Drugs, brains, and behavior: The science of addiction* (NIH Pub No. 10-5605). Washington, DC: National Institutes of Health, U.S. Department of Health and Human Services. Retrieved from http://www.drugabuse.gov/publications/science-addiction

Niciu, M., Chan, G., Gelernter, J., Arias, A., Douglas, K., Weiss, R., . . . Kranzler, R. (2009). Subtypes of major depression in substance dependence. *Addiction*, *104*, 1700–1709. doi:10.1111/j.1360-0443.2009.02672.x

Pihl, R., & Stewart, S. (2013). Substance use disorders. In L. Castonguay & T. Oltmanns (Eds.), *Psychopathology: From science to clinical practice* (pp. 241–274). New York, NY: Guilford Press.

Schore, A. (2002). Advances in neuropsychoanalysis, attachment theory, and trauma research: Implications for self psychology. *Psychoanalytic Inquiry*, *22*, 433–485. doi:10.1080/07351692209348996

Shean, G. D. (2010). *Recovery from schizophrenia: Etiological and evidence-based treatments*. New York, NY: Hindawi Publishing.

Siegel, D. (2007). *The mindful brain: Reflection and attunement in the cultivation of well-being*. New York, NY: W. W. Norton.

Siegel, D. (2012). *The developing mind: How relationships and the brain interact to shape who we are* (2nd ed.). New York, NY: Guilford Press.

Silberman, Y., Bajo, M., Chappell, A., Christian, D., Cruz, M., Diaz, M., . . . Weiner, J. (2009). Neurobiological mechanisms contributing to alcohol-stress-anxiety interactions. *Alcohol*, *43*, 509–519. doi:10.1016/j.alcohol.2009.01.002

Slade, A. (2008). The implications of attachment theory and research for adult psychotherapy: Research and clinical perspectives. In J. Cassidy & P. Shaver (Eds.), *Handbook of attachment: Theory, research, and clinical applications* (2nd ed., pp. 762–782). New York, NY: Guilford Press.

Substance Abuse and Mental Health Services Administration. (2013a). *About co-occurring disorders training. Motivational interviewing*. Retrieved from http://www.samhsa.gov/co-occurring/topics/training/motivational.aspx

Substance Abuse and Mental Health Services Administration. (2013b). *About co-occurring disorders training. Stage-wise treatment: A key component in an evidence-based approach to treating co-occurring disorders*. Retrieved from http://www.samhsa.gov/co-occurring/topics/training/stage-wise-treatment.aspx

Thorberg, F., & Lyvers, M. (2010). Attachment in relation to affect regulation and interpersonal functioning among substance use disorder in patients. *Addiction Research and Theory*, *18*, 464–478. doi:10.3109/16066350903254783

Ulman, R., & Paul, H. (1989). The self-psychological theory and approach to treating substance abuse disorders: The "intersubjective absorption" hypothesis. In A. Goldberg (Ed.), *Dimensions of self experience: Progress in self psychology* (Vol. 5, pp. 121–141). Hillsdale, NJ: Analytic Press.

Ulman, R., & Paul, H. (1992). Dissociative anesthesia and the transitional selfobject transference in the intersubjective treatment of the addictive personality. In A. Goldberg (Ed.), *Dimensions of self experience: Progress in self psychology* (Vol. 8, pp. 109–139). Hillsdale, NJ: Analytic Press.

Weiss, R., Jaffee, W., de Menil, V., & Cogley, C. (2004). Group therapy for substance use disorders: What do we know? *Harvard Review of Psychiatry*, *12*(6), 339–350. doi:10.1080/10673220490905723

Zero to Three. (2007). *Still face experiment: Dr. Edward Tronick*. Retrieved from http://www.youtube.com/watch?v=apzXGEbZht0

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