Commentary on "Understanding Addictive Vulnerability"

Brian Johnson

A NEURO-PSYCHOANALYTIC APPROACH TO ADDICTION

Psychodynamics or neuro-psychoanalysis?

Dr. Khantzian has done a masterful job of summarizing the psychodynamic literature on addiction. Khantzian states, "With some notable exception, it is surprising and unfortunate that since the 1970s there have been so few contributions from psychoanalysts addressing the dynamics of SUDs [substance use disorders]." If we add the statistics from McGinnis and Foege (1993) that drug addiction is responsible for 25% of all deaths in the United States (nicotine 19%, alcohol 5%, illegal drugs 1%) and obesity (food addiction) is responsible for another 14% of U.S. deaths, it becomes obvious that addiction is a pressing issue for psychoanalysis. Either many patients undergoing psychoanalytic therapy have active addictive issues and their therapists lack a good theoretical basis for understanding their issues and consequently ignore them, or these extremely common patients are being systematically turned away from psychoanalytic therapy because we lack a good theoretical basis for understanding their issues. I suspect both are true.

Since there is a major biological component to addiction, an important difficulty involved in using psychodynamics alone is that the biological nature of the addictive process becomes indistinct rather than standing clearly as one side of a dialectical process between psychology and biology. Neuropsychoanalysis dwells in the margin of psychoanalysis and biology from which Dr. Khantzian indicates synthetic models may be formulated.

I will use the definition of neuropsychoanalysis provided by Kaplan-Solms and Solms (2000, p. 62). Neuropsychoanalysis links mind and brain "to dissect the internal psychological structure of the various changes in personality, motivation, and complex emotion that occur following damage to different cerebral structures. Thereafter, the multiple underlying factors producing these symptoms and syndromes can be identified and each correlated with its anatomical 'scene of action.'"

There is no doubt that drug addiction causes brain sequelae, and yet these changes are not sufficient to explain the nature of addiction. In a biological review, "Molecular Basis of Long-term Plasticity Underlying Addiction," Nestler (2001) is careful to include nonbiological contributions to an addictive diathesis:

> Addiction is caused by the actions of a drug of abuse on a vulnerable brain and generally requires repeated drug exposure. This process is strongly influenced both by the genetic makeup of the person and by the psychological and social context in which the drug use occurs. Once formed, an addiction can be a life-long condition in which individuals show intense drug craving and increased risk for relapse after years and even decades of abstinence. This means that addiction involves extremely stable changes in the brain that are responsible for these long-lived behavioral abnormalities. [p. 120]

Since "repeated drug exposure" is what leads to the

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"extremely stable changes in the brain," the neuropsychoanalysis of addiction may be divided into a series of steps:

1. The premorbid states that provoke individuals to expose their brains repeatedly to potentially destructive chemicals.
2. The genetic (constitutional) and characterological bases of the transition from drug use to drug abuse or drug dependence.
3. "The internal psychological structure of the various changes in personality, motivation, and complex emotion that occur following [the stable brain changes induced by addictive drugs]." (Kaplan-Solms & Solms, 2000)
4. The relationship of drug addiction to other addictions (discussed in Johnson, 1993), such as addiction to shopping, work, gambling, sex, food (obesity, bulimia), and dieting (anorexia nervosa). This area includes the relationship of addiction to compulsion (Dodes, 1996).

On this last point, we notice that certain compulsive behaviors—shopping, work, gambling—do not have identified brain motivational systems. Appetitive systems for sex and food have delineated motivational systems that are linked to the ventral tegmental/SEEKING system of the brainstem (Panksepp, 1998, ch. 9, 12)—the same system that provokes the craving for addictive drugs (summarized in Johnson, 1999). A neuropsychological approach to linking appetitive systems, drug craving, and compulsive behaviors will be delineated later in this paper.

A neuropsychoanalytic approach to "self-medication"

Khantzian describes the "cart-horse dilemma" as "whether there is a causal relationship between psychiatric disorders and SUDs. The distinction between what condition is primary or secondary . . . is inconclusive." The neuropsychoanalytic approach has gone a long way in elaborating Khantzian's cart-horse dilemma. Step one is that we need to know more about why individuals expose their brains repeatedly to potentially destructive chemicals. Second, we need to know the constitutional and characterological bases of the transition from exposure to brain change. Embedded within these questions is Khantzian's "self-medication hypothesis," that the goal of addictive use of drugs is to treat intolerable affects, and he discusses this in regard to the specificity of opiates: "Opiates are very effective in countering and muting intense anger and rage and appeal to individuals who endure such feelings, and/or suffer with psychiatric conditions in which such affects predominate." As he points out, physicians constantly prescribe medications with a specific goal of altering anxiety, mood, and psychosis. Kramer (1993) has described eloquently how many patients do not think specifically about symptoms but, instead, are desirous of medications from physicians which can improve character functioning. In this regard opiates are helpful in increasing tolerance to rage in general character functioning as well as possibly helping in other ways too.

There is a great deal of room for expansion of our understanding of self-medication with addictive drugs. Panksepp (1998, p. 194) notes that self-stimulation of the ventral tegmental pathway is facilitated by chlordiazepoxide, suggesting that benzodiazepines may function to increase drug hunger. Benzodiazepines may be the appetizer, and opiates the main meal, in their function as instigators of consummatory behavior.

The concept that an inability to depend on others is central to addictive psychopathology is reviewed by Khantzian. Panksepp (1998, ch. 14) describes the hard-wired need for affiliation within the brains of animals and the specific PANIC neural circuitry that results in distress vocalizations (DVs) produced by young animals separated from their mothers. Corticotropin-releasing factor (CRF) is increased during DVs, a well-known marker of stress and distress. Both opiates and nicotine reduce DVs, indicating reduced distress from maternal separation (Panksepp, 1998, p. 268). Heim, Newport, Bonsall, Miller, and Nemeroff (2001) have shown persistently elevated CRF in women who have been traumatized by abuse during childhood. These studies are consistent with earlier findings of this group using rats and nonhuman primates that maternal deprivation and adverse rearing conditions result in life-long elevation of CRF. Women with childhood abuse are five times as likely to abuse drugs and are twice as likely to abuse alcohol (McCauley et al., 1997). Thus, we can begin to construct the biological links between childhood experience and the short-term amelioration of intolerable adult affects with addictive drugs.

The neuro-psychoanalytic conceptualization of addiction leads to the recognition that opiates respond to multiple complimentary needs. The typical patient who takes benzodiazepines, uses heroin or methadone, and smokes cigarettes is intensifying her/his consummatory urges with the benzodiazepines, and then with opiates and nicotine is able to gratify her/his appetite, modulate intolerable rage generated in relationships, and at the same time decrease the distress involved in being distant from dependent relationships.

A neuro-psychoanalytic approach to the relationship between addiction and other DSM-IV disorders

Anxiety and mood disorders

While anxiety and mood disorders do not create addictive disorders, there is an obvious interrelationship that needs to be further understood. For example, anxiety disorders seem to co-evolve with alcohol-use disorders; college students with alcoholism are far more likely to develop anxiety disorders, and college students with anxiety disorders are far more likely to develop alcoholism (Kushner, Sher, & Erickson, 1999). Alcohol dependence does not seem to stay in remission, while patients who have been detoxified are depressed (Greenfield et al., 1998). Apparently, when a depressive disorder is active, it is much harder to resist the return to addictive behavior. In a large population survey, of all women with alcohol dependence, 61% had premorbid anxiety disorders and 54% had premorbid affective disorders. Of all men with alcohol dependence, 36% had premorbid anxiety disorders and 28% had premorbid affective disorders (Kessler et al., 1997). The neuro-psychoanalytic concept that addiction to drugs, food, sex, and probably gambling, shopping, and work (further discussed below), involves *appetitive* behaviors helps the connection of addictive behaviors to Axis I disorders, especially anxiety and mood disorders.

Appetitive behaviors are strongly influenced, and are dysregulated, by anxiety and mood disorders. Some people cannot eat when they are anxious, some overeat. Some people cannot eat when they are depressed, others binge on food. Some people lose their appetite for sex when they are anxious or depressed, but a subpopulation of men with these disorders are hypersexual (Kafka & Prentky, 1998) and might be said to be suffering from sexual addiction. In a psychodynamic model, individuals may be self-medicating anxiety and mood symptoms with drugs. But there are likely to be neural pathways that can eventually be described that will complement our psychodynamic model and enhance our understanding of the clinical intersection of appetitive, addictive, anxiety, and mood disorders.

Personality disorders

Khantzian expresses the need for a better delineation of the relationship between personality disorders and addictive dynamics. The character structure of the patient determines whether the analyst offers a supportive-expressive or interpretative form of psychoanalytic therapy (Kernberg, 1984; McWilliams, 1994). Some patients with addictions have a neurotic character organization, others have addictions and personality disorders. Khantzian has advocated adding "interactive and supportive elements into therapy."

I have found that the degree of optimal psychoanalytic activity is dictated by the nature of the patient's character structure, the degree of parental abuse and neglect that is reproduced in the transference, and whether the patient's problem is drug abuse or drug dependence. When dependent patients return to active use, there is a catastrophic deterioration of the function, which makes psychoanalytic therapy impossible. For example, I was forced to give direct instructions to tear up credit cards and file for bankruptcy when a patient revealed that she had switched from heroin to shopping addiction (Johnson, 1999). Conversely, a patient with substance abuse (not dependence) and relatively high functioning can tolerate a degree of inactivity and interpretation that leads to substantial character modification (Johnson, 1992).

There is a complex interaction between character and biology in addiction. Adopting an addiction may be a sign of distress in an adolescent (Wurmser, 1974), perhaps a degree of distress that corresponds to lower-level defenses that characterize a personality disorder (McWilliams, 1994). On the other hand, addictive brain changes modify character functioning because of a new set of drives to which the individual must respond (Johnson, 2001). These changes may make the addicted individual appear to have a personality disorder when they have a currently uncategorized state of biologically mediated character change.

DSM-IV categories of abuse and dependence

Another advantage of the neuro-psychoanalytic perspective regarding point 3 above—the internal psychological structure of the various changes in personality, motivation, and complex emotion that occur following a specific set of brain changes that are produced by the transition from drug abuse to drug dependence—is to use this concept for diagnosis, treatment recommendations, and prognosis. In the language of Alcoholics Anonymous (AA): "You can't turn a pickle back into a cucumber." I predict that in future editions of the *Diagnostic and Statistical Manual*, concepts of abuse and dependence will be further refined to indicate that the addictive behavior either fulfills only a psychological function (abuse) or that repeated drug exposure has caused an irreversible brain change (dependence). Certain drugs seem to create

this change rapidly (e.g., nicotine or cocaine). No one who has smoked forty cigarettes a day can return to having one occasionally. Craving and drug dreams are clinical symptoms that are likely to indicate an underlying change in ventral tegmental pathway functioning in drug dependence (Johnson, 2001).

The difference between abuse and dependence is more important in alcohol use than in the use of nicotine or cocaine because the ventral tegmental pathway seems much slower to change with exposure to alcohol than to these other drugs. I have observed craving and drug dreams for alcohol alone in the absence of any other significant exposure to addictive drugs. This was in a patient who had required detoxification from alcohol and who had significant alcoholic myopathy by age 28 (a physical finding related to extremely heavy, sustained use of alcohol). The difference between alcohol abuse and alcohol dependence has as its most important differentiating characteristic the existence of physiological withdrawal from alcohol (Schuckit et al., 1998).

On the other hand, I have reported a complete psychoanalysis (Johnson, 1992) and vignettes of another psychoanalysis and a three-times-per-week psychotherapy (Johnson, 1993) of patients with alcohol abuse where addressing the psychopathology in analytic therapy restored the patients' ability to drink alcohol in an asymptomatic manner. In the psychotherapy case, I was able to publish the patient's liver enzymes at the beginning and end of treatment and to show that they had returned to normal with moderation of the patient's intake (Johnson, 1993). These three patients did not show any physical dependence on alcohol, despite a plethora of symptoms of alcohol abuse. They had not sustained permanent brain changes despite substantial symptoms of alcohol abuse, i.e. had not "turned into pickles." This differentiation could be used to answer the universal question of patients with alcoholism: "Can I ever drink again?" The answer is: "Only if you have alcohol abuse rather than alcohol dependence, and only if you undergo comprehensive treatment for the psychological conditions underlying your alcohol abuse."

Complementarity of psychodynamic and neuroscience explanations

I have suggested that the phenomenon of "substitution" is explained by the symbolization of an early object by any addictive behavior, and that when one addictive behavior becomes impossible to continue, the addicted person frequently shifts to a second compulsive and self-abusive behavior (Johnson, 1993). In a case presentation, I showed that a patient who had entered psychoanalysis after detoxification from heroin switched to shopping addiction in order to avoid depending on me, in the context of a transference reactivating early relationships with a terrifying set of caregivers (Johnson, 1999).

Panksepp (1998, p. 153) covers the same territory with rat studies! Stimulation of the ventral tegmental area triggers a diversity of "stimulus-bound" behaviors such as feeding, drinking, wood gnawing, sexual behavior, pup carrying, and tail preening. When the animal's preferred goal objects were taken away, and electrical brain stimulation of the ventral tegmental area was continued, the animal shifted to another behavior: "When the originally preferred goal object was returned, the newly acquired consummatory behaviors competed effectively with the original behaviors" (Panksepp, 1998, p. 153). Robinson and Berridge (1993) discuss an analogous set of animal-study findings that suggest that once the ventral tegmental area has been upregulated by exposure to one class of addictive drug, compulsive acquisition of a second addictive drug is induced more rapidly, suggesting that there is a long-lasting or permanent upregulation in functioning in this pathway. Feeding, drinking, and sexual behavior have known neural pathways. Could it be that wood gnawing and tail preening are rat equivalents of shopping, working, and gambling?

The addition of neuroscience findings to psychoanalytic findings does not invalidate concepts of self-medication or symbolization. It adds information. As Khantzian indicates, we are in a process of integrating different sources of information in order to construct, reconstruct, and expand the models that guide our interventions.

Neuro-psychoanalytic contributions to psychoanalytic theory

Drives

A final advantage of a neuropsychoanalytic approach to addiction is that the discoveries of this approach can be returned to psychoanalytic theory. Freud, of course, began as a neurologist. Frustrated by the underdevelopment of nineteenth-century neurobiology, he expanded his thinking with psychological constructs. As psychoanalysis has grown over the hundred years since Freud, it has lacked the corrective of the dialectical materialism between psychology and biology. For example, Mitchell (2000) summarizes,

> Some continue to use the terminology of drive theory, but a closer look reveals that the meanings

> of the classical terms have changed. Freud's concept of "drive," perfectly consistent with the intellectual currents of his own time, was of an endogenous force of nature, pushing from within, that only secondarily comes into interaction with the external world. The most common concept of "drive" in the recent psychoanalytic literature is something quite different. Shaped by the highly influential metapsychological revisions of Loewald and Kernberg, "drive" now generally indicates a body-based sexual or aggressive motive that has derived from, and has been shaped by, dense affective interactions with early caregivers. [p. xiii]

Immersed once again in neurobiological material, this "relational" concept of drives seems completely incorrect. Addictive chemicals produce new drives by keying into the ventral tegmental pathway which already motivates animals to seek food, water, and sex. Drives *are* an endogenous force of nature pushing from within that only secondarily come into interaction with the external world.

For example, the drive to obtain cigarettes does not derive from an interaction with an early caregiver. It would be produced in 100% of individuals (human or rat) exposed to nicotine in a certain manner. As described by Khantzian and others, a lack of self-care somehow allows some individuals to expose themselves to an addicting drug. In a dialectical manner, the drug exposure causes neural changes. The neural changes include a new drive—nicotine craving—which further influences thinking, such as producing a denial system. The need to acquire the drug, and protect its use, further changes the nature of relationships.

Panksepp (1998) uses neurobiology to innovate a completely unexpected solution to the problem of how to explain why humans are object-driven. He proposes a series of motivational systems within the brainstem: SEEKING (drives), RAGE (anger and rage), FEAR (amygdala-based learned anticipation of harmful events), LUST (modulating sexual desire in complex ways), CARE (nurturance), PANIC (fear of separation), PLAY (drive for social interaction), SELF (essential for generating subjective emotional feelings in all mammalian brains). We do not have to throw away drives in order to accept that humans are internally motivated to be affiliated with others in a variety of ways. The midbrain nature of the SEEKING/drive system helps to understand why Freud suggested that there is no direct expression of drive, but that drives manifest through "drive derivatives." Midbrain endogenous forces must stimulate motor and sensory cortical areas in order to be represented in secondary process, and acted on in the world. But accepting that the drives of the twenty-first century are the same as Freud's of the nineteenth and twentieth centuries is in no conflict with separate PANIC, CARE, or PLAY neural subsystems that drive relatedness.

Ego defenses

In addictive disorders, a permanent brain change in the ventral tegmental/SEEKING system is manifest in wordless craving and drug dreams. Freud's concept of the ego—the interface between internal and external through which we constantly strive to cope with wordless cravings within our social context—makes perfect sense. Khantzian's focus on shame in addiction and alliance building can be further articulated by suggesting that any therapist needs to imagine what it is like to cope with intensified drives produced by exposure to addictive drugs. Imagine all the times that you meant to stick to your diet, yet found yourself eating your favorite fattening food. Think about how carefully we all contain our sexual feelings for each other, and the catastrophic results of unconsciously allowing these feelings to be expressed in inappropriate contexts. What would it be like for *us* to have to appear before a therapist or other authority and admit that we had been unable to contain our drive expressions, and that there had been embarrassing consequences?

Ego defenses are employed to try to cope with these intensified drives. We can observe them. In the case of a "denial system," the individual seeks to gratify her/his wish to use addictive drugs and yet to disguise to her/himself this expression of the drive. Whether a psychoanalyst or AA sponsor, the outside observer can name and interpret the defenses of the denial system. Thus, a return to the original Freudian concept of drive prompts, as a corollary, a return to the original Freudian concept of ego defense.

Repetition compulsion

Robinson and Berridge (1993), writing with rat and other animal studies as the basis for their discussion, ask the question: "Why do addictive behaviors persist when the pleasure of drug use ebbs?" Their formulation may be taken as a contribution to the problem of repetition-compulsion. On a neurobiological basis, drug seeking and drug-seeking-related behaviors become fixed by the drive (ventral tegmental) pathway. Under its influence, pleasure becomes irrelevant.

They explain that there are separate neural systems for wanting and liking: wanting is a drive, liking is a hedonic experience. Thus, the compulsion to repeat runs on a neural system separate from neural systems that underlie the experience of pleasure. This may not completely explain repetition-

compulsion, but it is essential information for theory building.

The brain changes involved in drug addiction allow us to observe the mind functioning under altered conditions. From this new vantage point, certain properties of the mind stand out more clearly.

Conclusion

Psychoanalysts need neurobiology, and neurobiologists need psychoanalysis (Kandel, 1998, 1999). This loose set of responses to Khantzian's paper is not meant to be a complete work, but a scaffolding, with expansion slots available for future developments of all kinds.

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