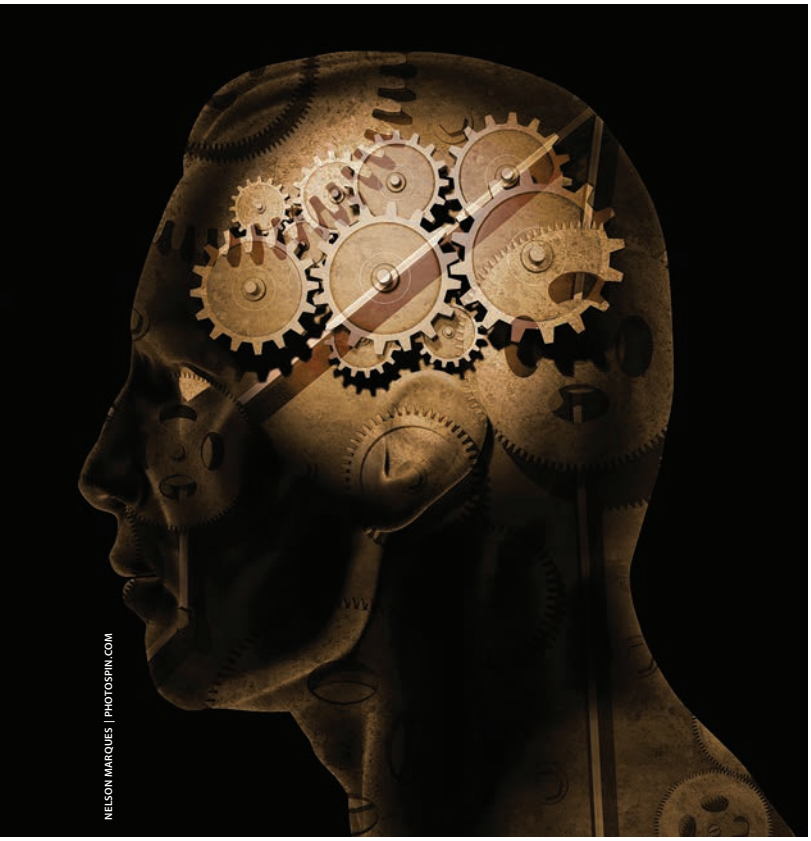
Neurobiology-Informed Responsive Addiction Treatment: One Therapist's Experience

By Joe Terhaar, PhD



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Since the inception of the first systematic, successful efforts to survive alcohol addiction through 12-step fellowship, there have been adaptations for other addictions (e.g., Narcotics Anonymous, Cocaine Anonymous, Overeaters Anonymous). From the application of 12-step principles in the Minnesota Model of treatment, to a variety of subsequent philosophical approaches (e.g., Cognitive Behavioral Therapy, Dialectic Behavioral Therapy, Choice Theory), our profession has diversified. We have a more holistic view of the person with a substance use disorder within the family (codependency, family behavioral therapy, etc.) and the community (sober housing, drug court, recovery-oriented systems of care, etc.) and appreciate the complexity of addressing co-occurring disorders and the complicated influence of past trauma. The profession continues to improve efforts to support life-preserving recovery.

Increasingly, treatment professionals help those with substance use disorders and families recognize that all drugs of abuse rewire the brain reward system away from survival and comfort to a drug-induced compulsion for exaggerated euphoric pleasure. These distortions of basic neurobiological mechanisms create the generic trap and craving cycle for reuse of drugs. Many chemical dependency professionals are familiar with the cycle: environmental cues and internal sensations are recognized by the amygdala brain processing center and trigger a small tempting dose of dopamine in the nucleus accumbens; then, with ingestion of the drug, there is a dopamine spike that temporarily quenches the ever-increasing appetite with ever-diminishing pleasure. With every use, the desire grows progressively and the euphoric effect lessens. All chemical dependency counselors should be well versed in conveying to clients and families this basic craving mechanism for drugs of abuse.

However, beyond the generic craving cycle and despite some shared neurotransmitters and pathways, each category of drug (e.g. cannabis, alcohol, opioids, methamphetamine, Spice, benzodiazepines) has neuromechanics that leave the drug user with characteristically different patterns of devastation and recovery needs. Most treatment centers have compelling films or presentations of the generic craving cycle, though almost none address drug-specific impacts on the brain. Tailoring the recovery plan and raising motivation with guidance that reflects the drug-specific impact on the brain is almost entirely overlooked. As a treatment community, we have not yet fully adapted and articulated our psycho-education and therapeutic responses to the actual drug-specific signature on the brain as reflected in an individualized service/treatment plan.

The Matrix Model for stimulant dependence (Rawson et al., 1995; Rawson, Obert, McCann, & Ling 1991) was a hallmark in recognizing the benefits of tailoring care to the primary drug of choice. It provided addiction professionals with an understanding of the need for and support to structure the recovering person's routine to reduced exposure to triggers. Structuring activities incompatible with drug use counteracts the frequent discharge of compulsive dopamine urges triggered a dozen times a day by environmental cues and internal sensations. However, looking at each drug's signature on the brain's wiring offers more specific guidance beyond introducing structure into the drug-user's hourly life. With treatment, we often facilitate clients and patients proceeding through generic, primarily time-driven denominations of care (by days for inpatient and hours of contact over days or months for outpatient). As professionals, we can be more responsive to the neurobiological needs of the individual suffering the drug-specific brain trap and impairments.

This article offers a sample of guiding points for providing care responsive to drug-specific brain traps for the individual, consistent with the Finlay's (1966) research on what motivated individuals to accept treatment. The crystalline element of motivation for treatment and change is in facilitating for the drug user a raised level of "concern about [him]self" (p. 76). Showing the drug user and family the mechanics of the drug-specific trap—with visual and kinesthetic examples relatable to their immediate lives—deepens comprehension of the debilitating downward spiral and the imperative for change and abstinence. This motivates far beyond the impact of the generic 'reward system being hijacked or rewired.' Presentation of the drug-specific impact unites the recovering person and family in their experiences. They can mutually understand the mechanical trap of addiction and collaborate to develop a recovery

plan that responds first to the recent adverse impact of the drug.

The most easily understood trap of drug impact is from the opioids. Often with the family and drug user together, I illustrate opioid action with the following story:

> An in-law of mine was distracted from his farm equipment, his right hand got caught in the machinery, and he lost three fingers and part of a fourth. Two days after the accident, I spoke to Phil in the hospital after his first of many surgeries. He spoke of the incredible pain, but I found it notable that despite such intense discomfort he was able to hold a conversation. Phil was being given powerful opioid 'painkillers.'

I then go on to explain that only about 20% of opioid action blocks the transmission of pain signals from the injury to the conscious receptors in the brain. With only 20% of the pain blocked, how could Phil have a cogent conversation? The other 80% of the opioid drug action is on the pain centers of the brain. The opioids were primarily reducing Phil's experience of the discomfort of the pain by triggering massive dopamine release in the nucleus accumbens pleasure center (the same area that produces the euphoria of sexual orgasm). Thus, the pain has not been killed, but overshadowed with drug-induced contentment and euphoria. Drawing the neuron-to-neuron transmission pattern and the mu receptor site on the nucleus accumbens facilitates comprehension and acceptance for the family and drug user of the physical nature of drug behavior. For the opioid user with no physical injury, there is a beyond-normal experience of euphoria and contentment.

I further explain that after weeks, months, or years of drug-induced addictive euphoria, if the appetite for the opioid drug is not satisfied, the pain receptors in the body and pain perception center in the brain scream pain. The pain is in the absence of an injury, but the pain sensing system reacts the same as if to an injury. This is happening in a person who has not developed skills and emotional capacities to cope with the physical and emotional pains of life.

When the impact of opioid drug use is explained in this way to opioid users, and in my practice family members (especially parents), motivation and relational participation palpably coalesce to provide healthy recovery supports. The necessity for abstinence is easy to accept. For the family, compassion and resolve of healthier non-enabling relational boundaries are strengthened. In family intervention preparation, the resolve to intervene is solidified and the process is tailored to target the perspective of the drug user. The family speaks with compassion to the pain, increasing the opioid user's acceptance of treatment. In treatment planning, neuro-informed compassion facilitates shifting from personalities and unresolved issues to collaboration on a plan that supports abstinence and recovery. It neither eliminates nor reduces the anger and pain of the harm from the addiction, but it motivates change to escape the neuro-trap for the individual and unites the family in meaningful relational change toward sobriety. This process nullifies denial.

The family and drug user comprehend that the imperative for abstinence goes beyond supporting engagement in activities incompatible with drug use to avoid re-experiencing the euphoria. The drug user, family, and recovery team are motivated to address the anxiety and fear of horrible pain while receptors gradually rebalance in a person ill-equipped to intrapersonally and interpersonally deal with the day-to-day discomforts of physical and emotional pain. Relapse is reduced by informing of the necessity for the drug user to engage in activities, marked by the half-hour, that are incompatible with access to any drugs of abuse in the balance with compassion for the challenges of abstinence. The family and drug user can now benefit from counseling support together, increasing their capacity to mutually support when dealing with pain. The family and the drug user are now better disposed to consider partial agonist (buprenorphine) and full antagonist (naltrexone) medication and many supports of addiction recovery.

For families with a loved-one suffering established chronic pain conditions, the mechanism for neuropathic pain can be sketched in 15 minutes. It is simple and accurate to encircle the neuron-to-neuron graphic with a red marker showing inflammation of the surrounding and supportive glia (Bland, Hutchinson, Maier, Watkins, & Johnson, 2009) that is, at least for many, caused by the short-acting opioid medications like morphine. When withdrawn from the short-acting pain medications (e.g. oxycontin, hydrocodone) and especially with the support of non-opioid pain management strategies, the pain either subsides or is manageable.

Visualizing the drug mechanism on the brain can powerfully motivate the drug user and family into action for each category of drug,

even cannabis (often viewed as only a recreational drug). When I draw the brain with arrows locating the pons and amygdala, it is easy to show the reduction of inhibitory action of GABA and the associated CB-1of cannabis use. The resulting drug-induced heightened experience of 'interest and vividness' can be illustrated in 15 minutes. Life feels more vibrant, even though one is less engaged. There is also a drug-induced sense of camaraderie and fellowship. To the observer, this is more noticeable when there is no one present or the cannabis user is not meaningfully engaged in relationship-building conversation. Families and drug users can now see the mechanism and understand why he or she has been experiencing a drug-induced contentment in the mundane, a lack of initiative or interest in novelty (with little motivation to accomplish), and deteriorating memory. This information engages the drug user and family to work together to increase structure for abstinence and informs them of the need to break down tasks into manageable and systematically rewarded steps. This is critical for the drug user with reduced neuro-biological capacity for intrinsic motivation. It also guides the team to responsively offer meaningful supports for problem-solving the alienation, lack of sober social connections, and feelings of abandonment experienced by early recovering cannabis users.

For alcohol, many counselors highlight its impact on GABA as it lowers inhibitions and impairs judgment, with the eventual impairments to gross intoxication. I find it far more motivating and transformational, in 15 to 20 minutes of an individual or family session, to highlight the impact on serotonin. Serotonin provides the sense of contentment after a good meal, a restful night's sleep, or the incredible sense of satisfaction after the euphoria of sexual orgasm. However, for the alcoholic, especially in those genetically vulnerable, as with the SLC 6A4 pattern (Seneviratne, Huang, Ait-Daoud, Li, & Johnson, 2009), serotonin levels are exaggerated with alcohol consumption and then drop well-below normal. This pattern manifests in an initial enhanced sense of contentment, as many an alcoholic has reported, 'finally feeling normal.' I draw a blue line showing the slight rise from and return to baseline of the serotonin level for the social drinker and a red line showing the alcoholic cycle that spikes up with contentment and euphoria and then drops far below normal with malaise and irritability manifesting themselves. The family and the drinker see the rollercoaster of behavior patterns they have long experienced reflected in the red line. The compulsion to feel contentment from the artificial serotonin rise and the compensatory drop afterwards into dissatisfaction and hangover are explained. The family and drinker can see the necessity for complete abstinence and transform it into resolve not to tolerate the rollercoaster ride of desperation, bargaining, and promises that have already failed. The visual, kinesthetic, and emotional interactions motivate recovery.

These interactions are also especially critical for benzodiazepine addiction recovery. Benzodiazepines very specifically enhance the inhibitory influence of GABA, particularly in the locus coeruleus, part of the brain's alarm system. Drawing this takes only about 10 minutes and makes it apparent how benzodiazepine action dampens the brain's ability to respond with alarm. Showing benzodiazepine action in the nucleus accumbens explains the creation of the highly rewarding fake euphoria. Presenting these basic mechanisms empowers and guides the drug user when responding to the chemically-induced anxiety and depression of benzodiazepine withdrawal. Recovery is motivated toward building coping capacity for stress, and in increasing relational capacity in family relationships.

With these few examples, it is clear how the neuro-biological impact of each drug category on the brain can be presented efficiently in a visually and kinesthetically representative manner to support comprehension of the addiction trap, guiding informed behavior change for the drug user, family, and interdisciplinary team. Presenting and collaboratively responding to the individual signature imprint of each drug category on the brain is the next iteration in the long evolution of saving lives.

REFERENCES

Bland S. T., Hutchinson M. R., Maier S. F., Watkins L. R., Johnson K. W. (2009). The glial activation inhibitor AV411 reduces morphine-induced nucleus accumbens dopamine release. *Brain, Behavior, and Immunity,* 23, 492–97.

Finlay D. (1966). Effect of role network pressure on an alcoholic's approach to treatment. *Social Work,* 11, 71–77.

Rawson, R. A., Obert, J. L., McCann, M. J., & Ling, W. (1991). Psychological approaches to the treatment of cocaine dependence: A neurobehavioral approach. *Journal of Addictive Diseases,* 11, 97–119.

Rawson, R. A., Shoptaw, S. J., Obert, J. L., McCann, M. J., Hasson, A. L., Marinelli-Casey, P. J., Ling, W. (1995). An intensive outpatient approach for cocaine abuse treatment: The Matrix model. *Journal of Substance Abuse Treatment,* 12, 117–127.

Senevirante C., Huang W., Ait-Daoud N., Li M. D., Johnson B. A. (2009). Characteristics of a functional polymorphism in the 3'UTR of SLC 6A4 and its association with drinking intensity. *Alcoholism: Clinical and Experimental Research,* 33, 332–339.

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Fortier, J. P. & Shaw-Taylor, Y. (1999). *Cultural and Linguistic Competence Standards and Research Agenda Project: Part One: Recommendations for National Standards.* Resources for Cross-Cultural Health Care. Silver Springs, MD.: Office of Minority Health.

Hanson, K. L. (2001). Patterns of insurance coverage within families with children. *Health Affairs, 20,* 240–246.

Macias, E. P., and Morales, L. S. (2000). Utilization of health care services among adults attending a health fair in south Los Angeles County. *Journal of Community Health, 25,* 35–46.

Morales, L. S. et al. (1999). Are Latinos Less Satisfied with Communication by Health Care Providers? *Journal of General Internal Medicine, 14.*

Ortiz, A. A. (1997). Learning Disabilities Occurring Concomitantly with Linguistic Differences. *Journal of Learning Disabilities.* May/June 1997.

Substance Abuse and Mental Health Services Administration. (2013). *Results from the 2012 National Survey on Drug Use and Health: Mental Health Findings,* NSDUH Series H-47, HHS Publication No. (SMA) 13-4805. Rockville, MD.

Table 1. Georgia Department of Behavioral Health and Developmental Disabilities (2014). FY12 *Utilization of Community Services per 1000 population By Disability Program & Ethnicity,* Table 1.

United States Census Bureau. (2002). Census: Hispanic dropout numbers soar. Retrieved from http://www.usatoday.com/news/nation/census/2002-10-10-censushispanic-dropouts_x.htm.

U.S. Department of Health and Human Services. (2001). *Mental Health: Culture, Race, and Ethnicity—A Supplement to Mental Health: A Report of the Surgeon General.* Rockville, MD: U.S. Department of Health and Human Services, Substance Abuse and Mental Health Services Administration, Center for Mental Health Services.

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