

Carnes, P. J., Murray, R. E., & Charpentier, L. (2005). Bargains with chaos: Sex addicts and addiction interaction disorder. *Sexual Addiction & Compulsivity: The Journal of Treatment and Prevention*, 12(2-3), 79-120. DOI: 10.1080/10720160500201371

## Bargains With Chaos: Sex Addicts and Addiction Interaction Disorder

PATRICK J. CARNES

*Pine Grove Treatment Center, Carefree, Arizona, USA*

ROBERT E. MURRAY

*Sutherland Associates, Knoxville, Tennessee, USA*

LOUIS CHARPENTIER

*The Meadows, Wickenburg, Arizona, USA*

*This paper proposes a theoretical framework by way of exploring addiction interaction in terms of addressing the following key questions: What happens when a patient population of sex addicts is assessed for other comorbid disorders? What conceptual foundations might we look to in terms of possible etiology and clinical intervention? Are there ways these addictions interact making the whole more than just the “sum” of the addictions themselves? Can we explain these interactions from what is known from neurobiology? Finally, are these interactions comprehensible to patients? In order for the paradigm to be useful to patients, they have to be able to recognize them. Clinicians who achieve a basic understanding of addiction interactions and relevant neurobiological background will be in a better position to serve the needs of their client.*

The emerging understanding of sex addiction as a mental health problem has paralleled a growing acceptance of multiple addictions, including both chemical and behavioral addictions. Some researchers have noticed how these extreme behaviors would be “balanced” by extreme deprivation or avoidance behaviors. Thus from a treatment perspective, clinicians have long noted that sex addiction was woven into an intricate web of addictions, compulsions, and avoidance strategies. Across the behaviors one is struck by how both polythetic and monothetic characteristics coexist. That is, each addiction or deprivation/avoidance has not only unique qualities but also remarkably similar characteristics. Thus the same patterns of loss of control or super efforts to control appear repeatedly driven by the same list of internal dynamics including shame, escapism, trauma, and stress. The obvious issues are: (1) Do these patterns share a common origin? (2) Are they manifestations of core dynamics? and (3) Are they interactive in fundamental ways?

Clinicians working with sex addicts see countless examples. Consider the following:

- the sex addict who buys his cocaine from his prostitutes in a ritualized predictable pattern
- the “successful” high profile sex addict whose compulsive working is relieved by compulsive prostitution use
- the alcoholic who has a sexual fantasy, but in order to carry it out has to get drunk first
- the sexual abuse victim who, when drinking, is compulsively sexual in high risk ways and when sober is sexually aversive, avoiding all possible sexual contact
- the use of ecstasy and Viagra to create “sextacy,” a compelling yet dangerous combination
- the cocaine addict who compulsively views Internet pornography while high and reports that neither activity works very well alone but does work as simultaneous activities
- the executive who, while on cocaine, masturbates compulsively for up to 12 hours in a binge, but while on alcohol hires two to three women to have sex with, during which he is very involved
- the obese sex addict whose despair about his sexual behavior is calmed by compulsive overeating and whose shame about his appearance drives him to act out sexually for relief
- the sex addict whose compulsive gambling hit the jackpot in the new Internet site where the prize was a night with a Las Vegas “escort”

Empirically we have data that have connected sex addiction with other addictions. The lead author followed a sample of 932 sex addicts (Carnes, 1991). Within that sample, 42% reported chemical dependency, 38% reported an eating disorder, 28% reported compulsive working, and 26% reported compulsive spending. As part of their recovery, they also identified multiple addictions in their mothers (22%), fathers (40%), and siblings (56%). Over time, numerous studies have documented the comorbidity of sex addiction and other addictions. These reports have documented this phenomenon in clergy (Irons & Laaser, 1994), health professionals (Irons & Schneider, 1994), chemical dependency populations (Gordon, Fargason, & Kramer, 1995), and eating disorders (Schwartz & Cohn, 1996).

Similar patterns have been noted from the perspective of other disorders. Within substance abuse, there is a wealth of documentation concerning the concurrent use of alcohol and drugs. Miller, Belkin, and Gold (1990) put it succinctly, “For the contemporary drug addict, multiple drug use and addiction that includes alcohol, is the rule. The mono-drug user and addict is a vanishing species in American culture” (p. 597). Studies of substance abuse patients also find significant comorbidity with gambling (Sweeting & Weinberg, 2000) and eating disorders (Stewart, Angelopoulos, Baker, & Boland, 2000). Conversely, studies of pathological gamblers have found symptoms of dependency on alcohol and drugs (Winters, Bengston, Dorr, & Stinchfield, 1998). Petrucelli and Stuart (2001) argue persuasively about the connections among eating disorders, sexual compulsion, and other addictions. Reflecting this emerging research have been collected efforts to show parallels across many addictions.

One of the most recent and thorough is the interdisciplinary collection by Robert Coombs (2004) titled *Handbook of Addictive Disorders: A Practical Guide to Diagnosis and Treatment*.

Clinicians who treat sex addiction must go beyond noticing the coexisting patterns. The realities of our patients are that they have made a number of “bargains with chaos.” If each addiction brings unmanageability to the patient’s life, it would be clinically negligent to think that the resulting chaos from each does not compound the problems of the others. The whole may in fact be more than the sum of its parts. Further, to borrow from Miller, Belkin, and Gold, “unless contemporary treatment methods are adapted to fit changing patient characteristics, attempts at rehabilitation may be futile” (1990, p. 596).

This opens a number of key questions. What happens when a patient population of sex addicts is assessed for other comorbid disorders? What conceptual foundations might we look to in terms of possible etiology and clinical intervention? Are there ways these addictions interact making the whole more than just the sum of the addictions themselves? Can we explain these interactions from what is known from neurobiology? Finally, are these interactions comprehensible to patients? In order for the paradigm to be useful to patients, they have to be able to recognize it. This paper is organized around these four critical questions.

### CASE STUDY: 1604 PATIENTS WITH SEXUAL DISORDERS

This case study represents a sample of 1,604 patients who participated in an inpatient residential treatment program. All of these patients presented some form of sexual disorder. They were assessed for sex addiction using conventional addiction criteria parallel to substance abuse and pathological gambling (see Carnes, 2000; Carnes, & Schneider, 2000). Also, they were assessed for sexual anorexia or sexual aversion using criteria (302.79) from the *Diagnostic and Statistical Manual, 4th edition* (American Psychiatric Association [APA], 2000). Anorexia criteria were augmented based on an earlier study of 144 patients (Carnes, 1998). Patient data were organized by dividing

**TABLE 1** Patients with Sexual Disorders: Sexual Addiction and Sexual Anorexia

<b>Patient Population</b>	<b><i>N</i></b>	<b>Addiction</b>	<b>Anorexia</b>
Heterosexual Males	893	803	325
Heterosexual Females	590	349	413
Homosexual Males	121	115	58
TOTAL	1604	1267	796

the patient population by gender and orientation. Heterosexual males, homosexual males, and females (both heterosexual and homosexual) were the subsets used.

Table 1 summarizes the assessment results of these patients in the first week of treatment. The assessments were conducted by nursing staff, the patient, and the attending psychiatrist. A cautionary note is important here. Given that these

assessments were done within the early days of treatment, it is very likely to include underreporting. As the process of treatment continues, patients and staff add additional diagnoses as the patient's situation becomes clear. For the purposes of this paper, the data are very appropriate because they represent the presenting reality of the patients at the outset of treatment.

In all three subsets, significant sexual addiction and sexual anorexia appear. This substantiates the clinical observation often made that both extremes are manifestations of the same problem. This would parallel in the eating disorders with the binge purge phenomenon of bulimia, and in many cases the long term swings of deprivation and excessive behavior. It also documents the presence of the extreme conditions in all three subsets. The occurrence of anorexia was higher for women (70%) than for either of the male populations (heterosexual, 36%; homosexual, 48%). Tables 2, 3, and 4 summarize, in descending order, the occurrence of other addictions. The reader will notice some variation in the presence of various disorders. For example, compulsive eating and bulimia were utilized far more by the women in the sample than by either male sample. An extensive analysis of this data goes beyond the scope of this paper. Yet, this summary of early assessment data clearly shows the need for differential diagnosis across the spectrum of addictions and compulsions. The bottom line is that 69% of the men, 79% of the women, and 80% of the gay men fit the diagnostic criteria for other addictions. Participants also were asked about their families. All three populations reported other family members who had addiction problems.

Another key concern was used as part of the assessment: simultaneously uses sexual behavior in concert with other addictions to the extent that desired effect is not achieved without sexual activity and other addiction present. Forty percent of the heterosexual men and 40% of the women found

**TABLE 2** Assessment of Other Addictions in Descending Order of Frequency:  
Male Population ( $N = 894$ )

<b><u>Other Addiction</u></b>	<b><u>N</u></b>	<b><u>%</u></b>
Alcoholism	411	45.8
Substance Abuse	360	40.1
Caffeine Addiction	330	36.8
Compulsive Working	330	36.8
High Risk Behavior/Danger	298	33.2
Compulsive Spending/Debting	243	27.1
Nicotine	232	25.9
Compulsive Eating	158	17.6
Compulsive Violence/Raging	126	14
Addictive Athleticism	102	11.4
Compulsive Hoarding/Saving	88	9.8
Compulsive Cleaning	80	8.9
Compulsive Gambling	74	8.2
Bulimia/Anorexia (food)	48	5.4

this to be true. It was true of 60% of the homosexual men. This is further evidence of the whole being more than the sum of the parts. That addictions actively affect each others' impact to the degree that they only achieve the de-

sired effect together, is of incredible significance to the clinician. Variations in the subset data also argue for clinical awareness, differential diagnosis, and treatment planning that involves ways to assess and understand the phenomenon of addictions that interact. Table 5 presents data about diagnostic criteria, simultaneous use, and family addictions.

**TABLE 3** Assessment of Other Addictions in Descending Order of Frequency: Female Population ( $N = 588$ )

<b>Other Addiction</b>	<b><i>N</i></b>	<b>%</b>
Alcoholism	271	45.9
Substance Abuse	241	40.8
Caffeine Addiction	217	36.7
Compulsive Working	212	35.9
Compulsive Eating	200	33.8
Nicotine	198	33.5
High Risk Behavior/Danger	176	29.8
Compulsive Working	173	29.3
Bulimia/Anorexia (food)	156	26.4
Compulsive Violence/Raging	108	18.3
Compulsive Cleaning	108	18.3
Addictive Athleticism	78	13.2
Compulsive Hoarding/Saving	77	13
Compulsive Gambling	25	4.2

**TABLE 4** Assessment of Other Addictions in Descending Order of Frequency: Gay Population ( $N = 121$ )

<b>Other Addiction</b>	<b><i>N</i></b>	<b>%</b>
Substance Abuse	65	53.7
Alcoholism	59	48.8
High Risk Behavior/Danger	55	45.5
Compulsive Spending/Debting	50	41.3
Compulsive Working	44	36.4
Caffeine Addiction	40	33.1
Nicotine	32	26.4
Compulsive Eating	24	19.8
Compulsive Cleaning	16	13.2
Addictive Athleticism	15	12.4
Compulsive Violence/Raging	14	11.6
Bulimia/Anorexia (food)	10	8.3
Compulsive Hoarding/Saving	10	8.3
Compulsive Gambling	4	3.3

## THE INTERNAL “LOGIC” OF ADDICTION INTERACTION

An internal logic exists once the premise of coexisting addictions is accepted. The logic could be expressed as follows:

- ◆ Addictions coexist and manifest common characteristics;
- ◆ They may have a common etiology, which would mean they are more intricately connected than we have assumed;
- ◆ If so, these “connections” can be analyzed into a comprehensive paradigm that would assist treatment;
- ◆ A “meta-paradigm” could, in fact, dramatically improve clinical results;
- ◆ From a biology (and disease) perspective, the meta-paradigm must fit known science;

**TABLE 5** Collateral Indicators

	<b><u>Heterosexual Males</u></b>	<b><u>Heterosexual Females</u></b>	<b><u>Homosexual Males</u></b>
Meets diagnostic criteria for other addictive disorders	69%	79%	80%
Simultaneously uses sexual behavior in concert with other addictions to the extent that desired effect is not achieved without sexual activity & other addiction present	40%	40%	60%
Reports other family members are addicts	48%	63%	55%

- ◆ From a patient perspective, the meta-paradigm must be useful and comprehensible.

From the perspective of sex addiction, this logic would bring into focus sexual behavior as a part of a larger phenomenon, which may help in clinical understanding of the problem.

## THE CASE FOR MULTIPLE ADDICTIONS

Many professionals have approached the problem of multiple addictions. The most obvious argument is that addictions have similar structure. In the *DSM-IV* (APA, 2000) the criteria for pathological gambling and substance abuse are almost identical. They also are very similar to the criteria proposed by different authors who have written about sex addiction. Many have argued at length about the parallels and commonalities of different addictive disorders. A good example is Jim Orford's 1985 classic work *Excessive Appetites: A Psychological View of Addictions*. His book is a landmark in early contemporary addictionology because it is a methodical, systematic critique of addiction theory. Orford offers one of the best discussions of the problems of social context, normal versus excessive, and the similarities across addictions. He makes the distinction between biology and psychology in addiction, which in light of modern brain research would be considered an artificial and out-moded distinction. Yet his comparison of the dynamics of excessive appetites is one of the most thorough expositions of multiple addictions. He writes:

The way in which "alcoholism" became a specialty, divorced from the general study of behavior, was perhaps inevitable but regrettable. When it has been linked with other kinds of behaviour at all, it has usually been placed alongside "drug addiction," a marriage which has emphasized the pharmacological basis of behaviour ...Equally, if not more useful, parallels are to be drawn between drinking, gambling, and sexual behavior ...The triad of drink, sex and gambling share many features ...each has given rise to fascinating and similar logical and semantic problems (Orford, 1985, p. 6).

Part of the resolution of logical and semantic issues comes from the science of the brain. Howard Shaffer's (1997) work on the cognitive chaos in addictionology rests in his recognition of achievements in neuroscience. He points to breakthroughs in our understanding of the brain that demonstrate the artificiality of propositions that state there is a distinction between physical and psychological dependence or that addiction must involve a chemical external to the body. He points out, for example, that "pathological gamblers experience addiction, including tolerance and withdrawal, often in the absence of any drug use." A similar position was taken by Milkman and Sunderwirth in their 1987 classic book, *Craving for Ecstasy: The Chemistry and Consciousness of Escape*. They posited that all addictions accessed certain neuropathways and that those "rivers in the mind" in fact were more important to understand than the multitude of ways addicts accessed them. Perhaps Arnold Leshner (2001), the former director of the National Institute for Drug Abuse, put it best when he summarized this position of addiction as a "hijacking" of the brain. He defines addiction as a "brain disease which manifests as compulsive behavior."

An intriguing perspective on the brain and addictions is provided by another classic, H. Huebner's *Eating Disorders, Endorphins, and Other Addictive Behaviors* (1993). The writer, like the above authors, documents the similarities of the various addictions including compulsive overeating. But he extends the argument from a neuroscience point of view to include de-privation. In fact he sees the architecture of addictions as sharing common features with various forms of compulsive avoidance such as anorexia

nervosa or sexual aversion. This is an important observation echoed by others including the lead author of this article. Patients experience excessive behavior at both ends of the spectrum. This paper argues that deprivations are also part of the “dance” of addiction interaction.

One of the most powerful arguments for understanding addiction as a large cohesive whole is made by Mihaly Csikszentmihalyi. His 1990 book, *Flow: The Psychology of Optimal Experience*, is one of the classics of modern psychology. Yet its conclusions have been largely ignored by addiction professionals. *Flow* is based on over 180 studies from all over the world about the composition of optimal experience. The author describes “flow” as the state when people are at their very best. What is often overlooked is the phantom optimum, addiction, which has the same structure as optimum performance. Csikszentmihalyi argues powerfully that addiction is a problem of attention and consciousness. The perversion of flow can happen with any activity that requires focus. He writes, “Almost any activity can become addictive in the sense that instead of being a conscious choice, it becomes a necessity that interferes with other activities ...[we become] captive of a certain kind of order.” Thus, he echoes Milkman and Sunderwirth (1987), and other above mentioned authors, talking about violence, crime, and other high-risk activities becoming compulsive behaviors. Another way to describe addiction from this perspective is to see the disease as the ultimate attention deficit disorder. Addictions are an “ordering process.”

Discussions of etiology support that addictions occur in constellations. From a genetics perspective some have argued that DNA configurations are similar for those with eating disorders, alcoholism, drug addiction, and other compulsive behaviors. Trauma specialists have emphasized the role of Post-traumatic Stress Disorder in the genesis of addictive disorders. Bessel van der Kolk’s (2001) recent arguments for “complex” PTSD is virtually foundational for looking at excessive behaviors as part of traumatic reactivity and alteration in the brain. Family therapists have noted that addictions become systemic in the family. For example, mother’s obesity interacts with father’s substance abuse and compulsive infidelity. Even in examining the role of the Internet in addictive behaviors, we can note clinicians’ language about the computer as a catalyst. Examples would be Schull’s (2003) suggestion of machine video poker as the crack cocaine of gambling or Cooper’s description of Internet sex as the crack cocaine of sexual compulsivity. Even from the perspective of the genesis of addictions, clinicians note common causes and even use common language.

Nor does treatment form a barrier to the concept of multiple addictions. Every excessive behavior has 12-step fellowships that have served people who have those problems. The viability of the principles of Alcoholics Anonymous has manifested in such diverse groups as Sex Addicts Anonymous, Overeaters Anonymous, Gamblers Anonymous, and Debtors Anonymous. Perhaps one of the best examples of the evolution of understanding of multiple addictions from a patient perspective is the advent and success of Recovering Couples Anonymous. This fellowship welcomes addicts of all persuasions because similar couples dynamics are common to all forms of addictive behavior. In fact, in an earlier study, Carnes (1991) found that one of the key factors in successful outcome was attendance in a couples 12-step format group. Similarly, in every clinical genre of excessive behavior there exist numbers of articles on the importance of cognitive behavioral intervention, trauma treatment, and family system intervention.

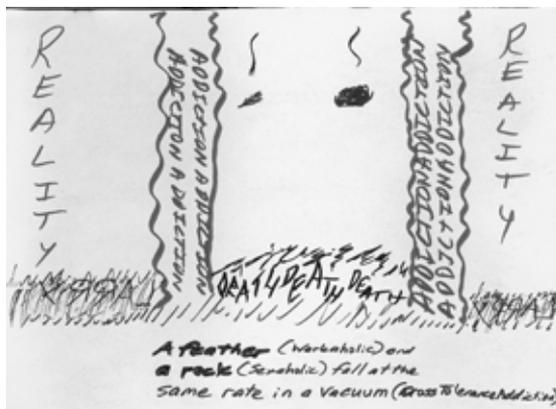
Howard Shaffer describes the addiction field as experiencing the classic stages of an emerging field of science (Shaffer, 1997). There almost always is a stage of conceptual confusion and blurring of issues. What is clearly emerging is an understanding of excessive behaviors as addictive disorders that share similar features, causes, and even cures. As part of that clarification we are proposing a model that integrates the addictions and proposes that the addictions have meta-patterns that are important and discernable clinically. We call this Addiction Interaction Disorder. What this means is that the addictions do more than coexist. They in fact interact, reinforce, and become part of one another. They, in effect, become packages. These packages can be unbundled and each addiction approached separately, which is the current level of practice. Yet equally important is that they can be approached as a whole. The following sections are criteria we propose as examples of addiction interaction.

## CROSS TOLERANCE

Cross tolerance occurs in two ways. The first is a simultaneous increase in addictive behavior in two or more addictions. This would indicate a connection between them where both addictions require increased activity. For example, consider the patient whose drinking and machine poker playing got worse at the same time. It is the parallel leap in activity that should catch the clinician's attention.

Consider also a patient who struggled with compulsive working (workaholism) and sex addiction. One hundred and ten hour work-weeks were not uncommon. Affairs with people he worked with also were not uncommon. He became sexually involved at work with employees, vendors, and a key board member who was an investor. The patient told his spouse that he had to keep up the pace because they would be ruined if he stopped, whereas if he was successful they would never have to worry about money. However, the descriptions of short-term situations stretched out to years. He clearly recognized that both the amount of sexual activity and the risks he took escalated. Similarly, the amount of his work continued to grow as did the business risks.

In treatment he made a drawing that creates a metaphor for how his addictions were escalating in similar ways. The drawing, reproduced in Figure 1, shows two pillars (representing his two addictions). They are like barriers within which reality had no sway. Reality is clearly outside of these "goal posts" to indicate how risk-taking was part of the problem. To further emphasize the point, he used the metaphor of dropping a feather (his workaholism) and a rock (his sexually compulsive behavior). He notes at



**FIGURE 1.** Cross tolerance.

the bottom that the two “fall at the same rate in vacuum,” which shows how cross tolerance affected him. To further emphasize his understanding of the concept, the rock and feather were headed for a black pile labeled “death” to underline the gravity of his addictive behaviors.

The second way to know that cross tolerance is present is when there is a sudden shift in addictive practice. A good illustration of this situation is a 38-year-old anesthesiologist who came to treatment for his cocaine addiction. He related a long history of sexual acting out in a wide variety of circumstances. He was taking risks to the point that he had breached sexual boundaries with patients. Then he discovered cocaine. He ceased all sexual behavior immediately and within two weeks was using cocaine daily. Moreover he was using cocaine at a level that usually takes years for a cocaine addict to tolerate. Later in this article we will elaborate on how neurochemically this type of transition can happen. Clearly the principle is that an addict can swap addictions including a level of tolerance for the new activity that was impacted by the previous one. To make an analogy, it would be like switching majors in college with the majority of the credits transferring.

Another clinical example involves work and sex. Our patient reported running a car wash business in which he was very successful. He worked very long hours and was hardly ever available for his family. He eventually sold his company for a great deal of money. Within days he was spending almost the same amount of time in strip shows and gentlemen’s clubs. During treatment it became very clear to him that he swapped one fairly well developed addiction for another. Usually sex addicts report that the development of their addiction has episodic use until it becomes continual and escalating. This patient reported immediate continual use that was escalating. Figure 2 is a picture he drew of working in the car wash and then a calendar to represent how brief the transition was to sex becoming a problematic part of his life.

## WITHDRAWAL MEDIATION

Withdrawal mediation occurs when one addiction serves to moderate, relieve, or avoid withdrawal from another addiction. A classic example would be nicotine use at an Alcoholics Anonymous meeting. Twelve-step meetings are known for strong coffee, cigarettes, and sweets. But the “smoke filled room” is the greatest irony. Cigarettes

cause more physical problems than alcohol does. Yet, people pursuing recovery use cigarettes to alter mood as they would alcohol. It has significant consequences. This brings up the issue of “giving up too many addictions at once” which is debated in professional as well as recovery circles. Yet the reality remains that nicotine use “cushions” the pain of early recovery by reducing anxiety. Many treatment facilities have gone smoke free precisely because of this issue.



**FIGURE 2.** Cross tolerance.

Oftentimes addicts will string together a number of addictive behaviors in a sequential pattern. Consider a patient who uses cigarettes and chewing tobacco to start his ritualized addictive process. He then gets on the Internet and binges on pornography. He smokes and chews while surfing sexual sites. He will go 10–12 hours. At the conclusion of the sexual binge he feels terrible about what he has been doing, the risks he took, and the time he wasted. He vows never to do it again. Then he binges on pizza and other food for days. The food becomes soothing to him after his sexual behavior. Thus the withdrawal mediation can be episodic as well as continual as in the AA example.

## REPLACEMENT

Replacement occurs when one addiction replaces another with the majority of emotional and compulsive features; one addiction can emerge after another has been addressed. Thus, when addiction professionals tell their substance abuse patients that everything gets better if people stop using drugs and drinking alcohol, they may in fact do harm. Patients need to understand that other addictions may emerge with time if they are not aware of the potential problem. Unlike cross tolerance, or withdrawal mediation, a period of time elapses (at least six months to a year) between the cessation of one addictive behavior and onset of new one.

A good example can be found in the course of recovery of a stockbroker named Lennie. He came from a wealthy family that hit hard times when Lennie was 13. He responded to the family financial crisis by getting jobs. By 15 he had three jobs in addition to school. Even more remarkable was that he had managed a small bank loan on a business he had started. After college he became a stockbroker and married into a

very wealthy family. He became the manager of his wife's trusts. He found his work on the trading floor to be extremely stressful. By his mid-twenties he was using marijuana daily as a way to cope with the intensity of the brokerage business. His wife expressed concerns that he dismissed because the business demanded that he perform well and marijuana helped him do that.

When Lennie was 31, his wife, Phyllis, went to get some money from one of her accounts without mentioning it to him. She was horrified to discover that she could access no funds. All of her funds were leveraged on margin purchases of speculative stocks. She was furious and convened an intervention on his marijuana use. In her mind it was pot that caused such a serious breach in Lennie's judgment. Lennie went to treatment for substance abuse and Phyllis attended family week. During family week they were assured that if the marijuana use stopped all the other problems would go away.

Lennie refrained from using marijuana and after a few months Phyllis returned her accounts to him for management. Lennie started to leverage the accounts again even though he was drug free. About six months after treatment he got into a significant cash flow bind. In a desperate gesture he placed some bets and won. This windfall not only saved the day financially, but encouraged him to bet further. He won again which confirmed to him that he had a "system." Now he was regularly betting using his wife's funds. In pathological gambling it is the early wins that hook the addict.

At the end of his first year of recovery, Lennie still had not used marijuana. His wife considered his treatment a success. But, Lennie was in bad shape. His betting and use of margin accounts was creating extraordinary stress. His main fear was that his wife would find out about his leveraging her funds again. A colleague suggested he might go to a massage parlor nestled in one of the office buildings in this exclusive financial district. He went and found that sex relaxed him in the way marijuana did. His visits to prostitutes became daily and sometimes twice a day. The costs of his sexual behavior added to the cash flow problems. His wife finally discovered a business card from an escort service and the house of cards came down.

In the story, notice the following: First, the treatment staff never identified compulsive debting as a problem, nor had they seen the use of the margin accounts as a form of gambling behavior. Essentially, this set of problems waited and actually blossomed into clear pathological gambling. The sex addiction problem really emerged almost a full year after treatment. When Phyllis came to treatment for family week the next time she was an angry woman. She distrusted professionals because the ones she saw the first time told her their problems were over if Lennie stopped using pot. By that standard, Lennie was in fact still sober. From our perspective, not all the problems were addressed and Lennie had found new, destructive ways to replace his old strategies to combat stress.

## ALTERNATING ADDICTION CYCLES

Addicts notice that their addictions have patterns in which the addiction-focus shifts. The addiction cycles weave back and forth in a patterned systemic way. Catherine serves as a classic example. She was physically and sexually abused as a child. Her mother was a prostitute with a compulsive gambling problem (very common in the sex

industry). In high school she was sexually promiscuous while anorexic with food. As an adult her sexual behavior was out of control and she maintained her self-starvation in order to be attractive. Her cure for this was to get married. Like many abuse victims she could be sexual even with anonymous people, but if someone really mattered she became sexually aversive or sexually anorexic. While married she was therefore compulsively non-sexual but she started to compulsively overeat and put on over 100 pounds. She admitted that the weight was, in fact, a defense against sexual overtures.

Catherine divorced, lost the weight, and went back into food starvation. Anonymous sex and sex with high-risk men became the order of the day. She got married again and repeated the pattern of sexual aversion and compulsive overeating. She married four times and each time gained 100 pounds. The sexual extremes and the food extremes became interchangeable, depending on whether she was married or not. In Figures 3 and 4 she presented the two patterns of her life: thin and sexually out of control, and obese and sexually aversive.

Catherine's pattern is quite common in our experience and raises a significant theoretical issue. If the eating extremes and the sexual extremes become interchangeable, it suggests common etiology as well as interaction. Also notice that deprivation as well as addiction is part of the exchanges. This would support Huebner's (1993) proposition that deprivation and addiction share similar psychological architecture to be able to serve similar functions. Clearly the two illnesses are interactive.

A different example is provided by Betty, who also noticed a set of rhythms. She would have a period of serious sexual bingeing with many partners. Then she would have a very intense, dramatic relationship that would break up. This would be followed by serious alcohol and drug use that would last quite some time. Then there would be another period of sexual bingeing followed by another all consuming relationship followed by an extended time of drug and alcohol abuse characterized by its isolation and



**FIGURE 3.** Alternating addictions cycles (Catherine).

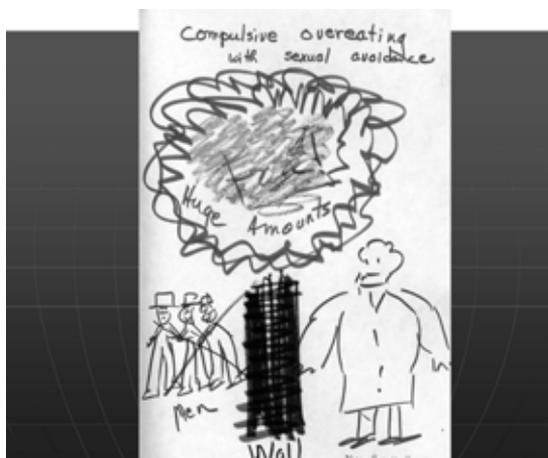
despair. In the timeline she presented in treatment she realized that she had repeated the same cycle 11 times. Further, she recognized that each phase of the cycle was getting longer. The numbers of partners and the risks she took in the sexual phase was growing. The "love" relationship was progressively more violent. The drugs became

more severe including heroin in the later cycles. There emerged a predictable pattern and a progressively deteriorating situation.

Obviously these different periods of compulsive behavior have some relationship with one another. Predictable sequencing implies an order to the behaviors. The fact that the progressive nature of each becomes longer and more risky suggests that each phase is worsening in some collective meta-pattern of progression. Both Catherine and Betty had prior treatment experience. Both made significant progress when they could see the larger patterns of the weave of their sexual behaviors.

## MASKING

Often in treatment we hear the phrase, “I did it because I was drinking.” What are clearly problematic behavior patterns are dismissed by the patient as the result of being under the influence. For example, it is less shameful to be an alcoholic than a sex addict. Yet under closer scrutiny it becomes clear



**FIGURE 4.** Alternating addictions cycles (Catherine).

that while the patient is under the influence, that patient’s sexual behavior becomes compulsive. The patient saw the problem resulting entirely from the alcohol use. Yet this type of patient will admit with time that always when they drink they are sexual in unwanted ways. In these cases, often times we find that when sober they would be sexually aversive.

These situations also fit other addiction interaction criteria, including the alternating cycles patterns as well as what we call “fusion.” Fusion occurs when the addictions are always present together. Masking occurs in these situations because the patient perceives the problem as the less shameful addiction. In fact using one addiction to cover up for another, perhaps more destructive addiction is a clinical sign that they are tied together. When most patients recognize that masking is an essential part of their denial system, they then discover the many ways the behaviors related.

## RITUALIZING

Rituals have long been recognized as part of addictive behavior. Rituals are used to induce trance and to prepare for special experiences. They are usually part of a sequence leading to significantly altered mood. Thus the drug addict has very specific preparations as part of getting high. The gambler has formulaic rituals as part of ensuring the windfall. The sex addict uses extensive rituals to enhance sexual pleasure. Food preparation rituals are frequently part of compulsive overeating. Interaction among addictions occurs when the rituals for one addiction are the same or significantly overlap the rituals for another. Many counselors miss significant data when they fail to ask how their patients acquired cocaine, which is often sexually. They may buy it from their prostitutes or use cocaine as part of highly ritualized behavior such as extended periods of masturbation.

One of the classic examples of the merging of rituals is the movie "Looking for Mr. Goodbar (1977)." It is the story of a young woman who is a sex addict, drug addict, and alcoholic. In the movie she dies ultimately from her drug use. Yet the story line is about her unremitting search for sexual conquest. Elaborate dressing and cruising rituals precede her picking up men in bars. In a famous scene she asks the bartender to join her in a drink. He refuses, telling her that he never takes the first drink because he cannot say no after having the drink. As he walks away she mutters to herself, "I know, I have the same problem with men."

The movie illustrates how her rituals of preparation to find men involve going to bars, drinking, and using drugs. They are in fact the same behaviors. The rituals then link the various compulsive behaviors before they start or one behavior enhances or is ritualistic to the other behavior. We had a patient who was compulsive sexually on the Internet and gambled pathologically on the Internet as well. The rituals of starting his computer and accessing his modem would initiate both sexual fantasies and windfall fantasies. Part of recovery for him was to recognize that he could not access a computer without getting into trouble.

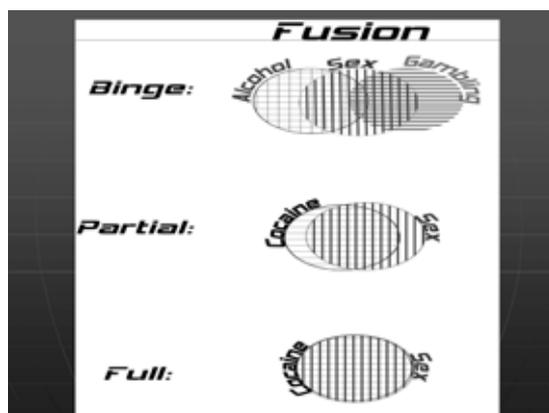
## INTENSIFICATION

Addictions intensify each other. In its most complete form, addiction fully potentiate so their addictive behavior. We call this Fusion Dependence. Neither addiction separately is sufficient: only simultaneous use suffices. To make concrete, think of the cocaine addict who has certain compulsive sexual behaviors and only does them on cocaine. He does not do the sex separate from the cocaine, nor will he do the cocaine without the sexual behavior. The addictions have become fused or inseparable. Another example is the patient who hyperventilates tobacco smoke and compulsively masturbates. These patients find confined spaces to concentrate the smoke such as a closet or bathroom and masturbate while hyperventilating the smoke. They describe a unique high which results ultimately in never smoking without the masturbation nor is there masturbation without smoking in the confined area. The neurochemical effects of the two addictions are only reproducible in this fashion. In a later section we describe how this likely occurs within the brain.

Some patients exhibit what we call Partial Fusion. Addicts combine addictions in

such a fashion to be more potent than each addiction separately. Some of the behavior is independent part of the time. A well-known example is compulsive prostitution. Over two thirds of men who use prostitutes compulsively also have significant financial disorders such as compulsive debting or spending. The eroticization of money becomes so complete that they can tell you the dollar amount of credit or cash that becomes sexualized. Diligent therapists are very thorough about following the money trail to disrupt the compulsive behavior. Most often we find that spending/debting problems extend to other parts of the addict's life as well. In other words the addictions are not exact overlays of one another.

Many patients describe Multiple Binge Behavior. The alcoholic, sexually addicted, compulsive gambler goes to the topless casino for one-stop shopping. Often addicts will binge in additive fashion to enhance mood alteration. Yet, they will pursue the behaviors independently. What intrigues us is that many patients who fit the criteria of fusion dependence report that they started with multiple binges. They progressed then to partial fusion, and then full fusion. Figure 5 creates a graphic representation of the phases of addictive fusion.



**FIGURE 5.** Fusion.

Another way of understanding intensification from a fusion perspective is what we call “inclusive.” In this situation one addiction subsumes all the rest. Thus, a sex addict also may be addicted to marijuana, cocaine, alcohol, gambling, and caffeine. Sex, if not totally fused, is partially fused with the others. Figure 5 is a graphic representation of how such a constellation of mutual intensification might look in a patient.

## NUMBING

In the trauma literature, van der Kolk (1998) and others talk about how trauma survivors assemble addictive behaviors that are highly stimulating, followed by a

collection of behaviors that are calming or soothing. High arousal is followed by numbing. One of the trademarks of Posttraumatic Stress Disorder is seeking high-risk repetitions of earlier victimization. Then, numbing behavior is designed to calm the system as a way to help regulate affect. An example of that is spending the evening in the streets doing risky, dangerous sexual behavior, then coming home and using alcohol, compulsive masturbation, and compulsive eating to soothe. Some patients include in the soothing such mundane things as television and such self-destructive behaviors as cutting (self-harm). Clinically what stands out most is how compulsive behavior is used to soothe or numb out. These behaviors are then inextricably tied to the original arousal behaviors.

## DISINHIBITING

Another way that addictions interact is for one to lower inhibitions for another addiction. One patient said, "I would think of what I wanted to do sexually, but I would have to get drunk to do it." Historically, we may have diagnosed such a patient as an alcoholic. Yet clearly his starting point and end goal are sexual. His drinking then enables the other compulsive behavior. The two addictions are tied together in a fundamental way. Neither behavior is necessary without the other.

## COMBINING

All professionals are familiar with the practice of combining uppers and downers. Yet many addicts take the practice of mixing addictive behaviors far beyond tailoring drug interactions. Usually the goal is to preserve a specific high by prolonging the feeling in whatever way possible. An analogy would be like surfing an ocean wave. The idea is to get on the edge of the wave and then make adjustments to stay there until it crashes. The addict will create a wave and try to stay on it for weeks until the body gives out. Then the addict will rest, recover, and gather strength for another run.

This phenomenon is common enough to appear in popular literature. Novelist John Sanford writes of the adventures of Lucas Davenport, detective and police chief. Consistently in his stories there is an antagonist who is pharmacologically savvy. He is a physician, a pharmacist, or a scientist who understands drugs and their impact on the brain. Usually this character is up to no good with high-risk, illegal, often sexual activities. Sanford manages to capture in his characters this "riding the wave" phenomenon where sex, high-risk activities, and various drugs are used to stay on the edge of the wave. Patients who combine to prolong or adjust highs are metaphorically surfing on the interactions of their addictions.

Many clinicians resist understanding brain functions because they are not trained in biology. The vocabulary of the brain's anatomy and circuitry seems incomprehensible and complex to the point of overwhelming. Yet in another ten years we all will become familiar with how the brain affects addiction. So we must become good consumers of medical literature to understand the potential of addictions to interact. This next section in that sense contains technical writing that may be unfamiliar to some of our readers. We ask that you make the best sense of it as you can

to understand this issue.

## THE NEUROCHEMISTRY OF ADDICTION INTERACTION

Addiction has been frequently described as related to basic biological drives. Hijacking is a term used to describe the maladaptive re-direction of basic biological drives to serve the initiation and maintenance of an addiction (Leshner, 2001). Addictive processes “tap into” and subvert existing neural circuits that have evolved to process information about natural drives and their reinforcers. With prescient insight James Johnston described the high-jacking process well in 1855:

Every feeling of joy and gladness even when the cause of it is elusively moral—that those enjoyments which are least connected with material objects, the most spiritual, the most ideal—may be nothing else but sensations purely physical developed in the interior of the system, in the same way as those which are produced by the means of haschisch ...that there is no distinction to be made between these two orders of sensations, in spite of the diversity of causes to which they are due (Johnston, 1855, p. 150).

Traditional drives focus on food, water, and reproduction. The reproductive drive is manifested both via sexual and attachment behavior. All of these drives can be observed in lower vertebrates manifested by repetitive stereotypical behavior. This reflects a brain organization that stresses speed and invariability of response over flexibility and adaptation. The evolution of higher vertebrates has produced a brain organization that is capable of responding to a variety of stimuli with flexibility. In addition, the development of explicit memory allows the creation of as-if scenarios and conscious planned modification of behavior based on those explicit memories. These developments place a premium on the desirability of seeking novel stimuli that may provide survival advantage. Each such novel stimulus must be processed through a learning module to determine if it is indeed advantageous. MacLean’s model of the triune brain is very helpful in understanding how all of this has happened (MacLean, 1990). Described simply, the triune brain is a sequentially developed organ that has evolved over eons and can be divided into three components. At its most primitive level, the brain stem contains the mechanism of reflexive, stereotypical action. Further evolution produced a second “brain” loosely patched into the brain stem. This adds primitive values, drives, and affect to the emotionless reflexive behavior of the lizard. Third, the cortex evolved in mammals and reached a pinnacle in the human cortex. The cortex contributes the ability to formulate as-if scenarios that makes judgment or revision of the affective states and emotional values possible. To summarize: A lizard reacts without thought or emotion; a primitive mammal reacts with limited thought and great emotion expressed through facial and verbal means. Lastly the cortex allows finely tuned, thoughtful control of affect (and hence lying). Controlling the balance between emotion and logical evaluation is the essence of the interactions between the cortex and primitive brain regions.

## THE TRIUNE BRAIN COMMITTEE

In order to make all of this work each part of the brain must teach the other parts what it knows. It is easy to think of the brain as a committee, each of the three parts represented. Like any committee each member defends the interests of its constituents. The committee meeting is the site of the high-jacking. The highjacking agent is recognized as a novel stimulus (chemical or behavioral) followed by a learning sequence involving both implicit and explicit memory processes that embrace the brain's own reward system in the support of addictive behavior. When the system is working well an item that instinctively is recognized as having survival value is assigned an affective value. For example, if a brown waxy material has survival value the taste of chocolate may evolve as a "reward" and thus reinforce its repetitive use.

## TRANSMISSION OF NEURAL REWARD INFORMATION

Transmission of neural reward experiences begins in neurons in the pre-mammalian midbrain ventral tegmental area (VTA), which projects to multi-polar "spiny neurons" in the nucleus accumbens (NAc). These spiny neurons communicate chemically with neurons from the cortex, the amygdala, and the hippocampus. The chemical communication occurs at a physical junction between cells called a synapse; one cell is pre-synaptic, the other post. The result is a neural hub of interaction among the pre-mammalian VTA, mammalian cortex, and structures dedicated to the assimilation of memory and to processing traumatic, endangering experiences. This hub is the committee meeting site that directs assimilation and transfer of information from primitive to more recently evolved structures (Wolf, 2003). Activation of this sequence of neurochemical events leads to production of motivating factors such as thirst, hunger, and sexual desire to satisfy the organism's basic and reproductive needs. In its more highly evolved form, exploratory drives such as "curiosity" emerge. In the parlance of the drug culture this curiosity produces drug-seeking behavior. The ability to create as-if scenarios facilitates problem solving as well as making possible self-reflective fantasies.

## PHARMACOLOGICAL AGENTS

Exogenous agents that have pharmacological effects on any part of this sequence may play a role in producing addictive interactions. Any agent producing a brisk increase in dopamine release in the NAc is likely to produce an addictive level response. Agents acting in combination may produce additive or synergistic effects if they act on different steps in the sequence. The agents that produce the most direct effect on this process are psychostimulants, which have been shown to facilitate bringing dopamine to sites where it may be released with appropriate stimulation. Cocaine increases the available dopamine by blocking the re-uptake of dopamine into the pre-synaptic cell after it has been released following stimulation. The cocaine step clearly depends on a pre-existing pool of releasable dopamine. Since these two classes of agents act on sequential steps, it seems likely that interactions take place. This indeed has been shown at the neurochemical level in rats. Other agents produce effects that are additive or synergistic to those produced by psychostimulants. A clearly described clinical example is found for opiates. Opiates increase dopamine in the NAc, but do so at a different point than cocaine in the dopamine cascade (Figure 6, steps 2 & 3). The synergism resulting from combining the actions of opiates and cocaine is the neurochemical basis

for the “speedball” effect. This satisfies the description of fusion presented earlier.

It is common to clinically observe addiction interactions that involve agents acting at complementing steps in the dopamine cascade. The image of a glass of wine followed by a hint of cocaine and a dose of sexual orgasm followed by the traditional cigarette comes to mind. Switching from one

Brain reward system (keyed to numbered steps)	Addictive agents involved and comments	Anatomic site of action
1. Dopamine brought to releasable site	Amphetamines have been shown to act on this step as well as promoting the release of dopamine into the synaptic cleft between the pre and post synaptic neurons.	Presynaptic DA neuron in NAc
2. Reuptake of dopamine into presynaptic cell is blocked	Cocaine acts on this step. Several antidepressants share this pharmacological feature without liability for addiction. The difference lies in the rate of dopamine increase.	Synaptic cleft, NAc
3. Decreasing GABAergic activity	This step is enhanced by opiates (direct action on GABA neurons) and alcohol (decreasing synthesis of GABA receptors). Cannabis may have a similar effect.	Presynaptic GABAergic neuron
4. Stimulation of cholinergic neurons	Acetyl choline binds to a variety of receptors with widespread action on neurotransmitters including increasing release of dopamine in the NAc. Nicotine also binds to these receptors, stimulating dopamine release.	Multiple synapses on DA cell.
5. Mono-amine oxidase inhibition	Tobacco smoke inhibits monoamine oxidase, an enzyme that facilitates the breakdown of dopamine and other mono-amines	Synaptic cleft
6. Inhibition of presynaptic dopamine receptors	This is a little like the float in the toilet tank. As dopamine builds up in excess, it binds to presynaptic receptors stopping it's own outflow allowing the unrestrained rapid release of dopamine	Presynaptic receptors: NAc or VTA
7. Increased cAMP in post synaptic spiny neurons	Caffeine shares its ability to produce this increase in cAMP with a number of pharmacological agents; many are used in the treatment of asthma, generally having mild stimulant properties.	Within post synaptic spiny cell
8. Direct stimulation of corticostriatal glutamatergic neurons	Opiates and alcohol are both thought to have this effect. This action makes the level of dopamine in the presynaptic cleft appear to be irrelevant by bypassing the involvement of the spiny neuron in stimulation of the cortex. This effect is not sufficiently robust to replace the previous 7 steps but serves to enhance.	Post synaptic spiny cell.

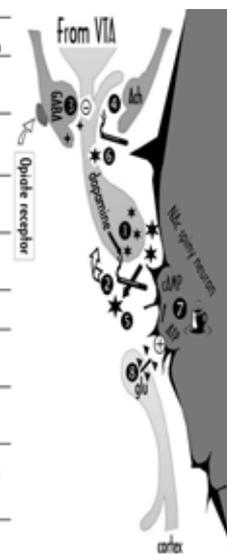


Table Six illustrates the NAc spiny neuron as a focal point of interaction. As shown, this neuron receives projections from the midbrain using dopamine as a neurotransmitter and from the cortex using glutamate as a neurotransmitter. While there are relatively few dopamine cells, they are positioned to influence activity in important functional areas. Each mammalian spiny cell has at least 5000 synapses with glutamate-producing cells from cortex (executive function), hippocampus (memory) and amygdala (trauma processing). The mammalian NAc contains at least several million spiny neurons. That works out to less than 250,000 dopamine cells with the potential to influence over one billion cortical neurons. Other neurotransmitters reach this area from other areas of the brain bringing the neurotransmitter actions of serotonin, norepinephrine, GABA, and acetyl choline. The NAc projects back to the cortex via the thalamus, thus completing a NAc-cortex continuous loop. The entire chemical scenario involves all of the above neurotransmitters in orchestrating the interaction among the triune committee governing the brain.

**FIGURE 6.** NAc spiny neuron: The reward system cascade.

addiction to another simply changes the point on the cascade where the neurochemical effect takes place.

Dopamine however is only a stepping stone in the transmission of information to the pre-frontal cortex. In fact, the role of dopamine fades with the arrival of another small molecule. Dopamine bound to external receptors on the spiny neurons facilitates synthesis of cyclic AMP (cAMP) within post-synaptic NAc spiny neurons. Cyclic AMP is a small molecule that is widely involved in many systems regulating which genes are “read” in the cell. It is a non-specific “second” messenger that is activated by a variety of neurohormonal agents that are specific to a given cell function. It would follow from this that agents known to increase cAMP may have a potentiating effect on most substances of abuse. A commonly used agent that stimulates cAMP formation is caffeine. Caffeine is largely regarded as a benign stimulant with little reinforcing potential. Cocaine and amphetamine activities are potentiated by pre-treatment with

caffeine (Derlet, Tseng, & Albertson, 1992). In the abstinent state, reduction of cAMP may lead to craving. Thus, there may be a state-dependent effect of alterations in cAMP that may enhance the action of addicting agents. Many different pharmacological agents modulate the cAMP system in addition to caffeine, including medications commonly used for asthma (Albuterol) and inflammation and pain (Ibuprofen). In general, these agents all have the properties of a mild stimulant. In my practice it is not uncommon to find patients overusing these medications.

Alcohol also has been shown to have an effect on the dopaminergic brain reward system through the dual mechanisms of diminishing GABAergic effects and altering glutamatergic effects. Chronic alcohol use has been shown to suppress the synthesis of certain components of the GABA receptor, resulting in increased dopamine availability. For those interested in detail, Figure 6 summarizes an eight-step sequence involved in the dopamine cascade of neurochemical events.

## BEHAVIORAL ADDICTIONS

Behavioral addictions are certainly the most controversial of the potential interactions among addictions. However, the evidence is clear that behavioral disorders such as gambling, compulsive shopping, sexual addictive behavior, and eating disorders substantially co-occur with substance abuse disorders. There is a collection of data that attributes behavioral compulsions to the same brain reward system as chemical addictions. Prior use of amphetamines increases frequency and intensity of sexual behavior (Fiorino & Phillips, 1999). This sensitization is both appetitive (prior to sexual behavior) and consummatory (during copulation). Neurobiological study of the sensitization process demonstrates augmentation of dopamine efflux in the NAc. This effect persisted for at least three weeks after administration of amphetamine was discontinued. Pathological gambling has been shown to be more prevalent in populations that were users of alcohol, nicotine, and cocaine. Intermittent, excessive sugar intake results in dependence on endogenous opioids. Even the anticipation of money selectively activates the NAc. It is likely that all similar compulsions are reinforced through increased activity in the brain reward system and are therefore targets of interaction with chemicals known to affect that system. These neurochemical interactions are the basis of alternating addictive behaviors.

A great deal of attention has been focused on subcortical structures, which, in some ways, is where the action is. The putative direction of information transmission is from the VTA through the NAc to the pre-frontal cortex. The overall function of the prefrontal cortex has been the subject of much study going back to 1848 with the case of Phineas Gage. Mr. Gage was a hard working railroad man from New England who was injured by a steel rod penetrating his skull from the cheek bone through the cranium.

This injury destroyed the pre-frontal area of his brain. He exhibited profound character change, with loss of moral behavior and motivation and a strange compulsive attraction to certain objects. He was not able to maintain his previous employment even though he was physically capable. Mr. Gage became incapable of exercising the good judgment that made him a successful and respected railroad foreman. He died leaving a reputation as unreliable, disruptive and lazy. If the pre-frontal cortex in

mammals is destroyed, they will exhibit compulsive reflexive actions and behavior reflecting uncontrolled drives that can be compared to Mr. Gage's behavior. This is apparent in grooming, drinking, eating, and uninhibited sexual behavior. Studies have shown pre-frontal cortical function is altered by psychostimulants and alcohol (Goldstein & Volkow, 2002). Specifically, the process of assigning value to reward-based decisions seems to be distorted in such a way that short-term gains (such as the brief pleasures of cocaine, orgasm, or methamphetamine) are overvalued, culminating in long-term net loss of function.

The ritualistic alternation between tobacco and sex is evident in the drawing made by a 45-year-old Caucasian male (Figure 7). His frustration concerns compulsive use of cigarettes followed by further compulsion to act out sexually. The intensity of his failing efforts to control his behavior is evident in the intensity of repetitious drawing. As will be discussed below, this



**FIGURE 7.** Ritualistic alternation.

may reflect a loss of control by the neocortex over more primitive ritualistic modes of behavior ingrained in subcortical brain pathways.

Drive states that seek novelty have their origins in cortical abilities to evaluate the “meaning” of stimuli. However, the quality of novelty itself may become an overriding goal. An illustration of this is provided by hallucinogens. MDMA (or Ecstasy) is an amphetamine derivative known to act as a stimulant. The dominant action of MDMA, however, is to increase extracellular serotonin. In sufficient doses, Ecstasy will result in hallucinatory activity and death of serotonergic neurons. Ecstasy is frequently used socially in high-intensity raves along with other drugs. The fusion of drugs and high intensity activates the brain reward system in conjunction with the serotonergic, hallucinatory (novel) actions of Ecstasy. A clear connection of this novelty-seeking state to drive theory is through the recognition of exploratory activity as a basic “drive” guiding mammalian behavior. A hallucination serves to satisfy the drive for novelty. Other hallucinogens, such as LSD, act in a similar manner.

From a sex addiction perspective, there are two concrete ways to illustrate novelty seeking. One is to note that part of the compelling nature of cybersex is stimulation

through bombardment of images. An unending stream of images can float across the screen. These images can become progressively more refined through the “marketing loops” of porn websites to closely fit an “ideal” fantasy. While the search is for the ideal, the stimulation is the diversity within that ideal.

Another example is romantic fantasy. Addicts report attaching incredible significance even to the most casual of social contacts. The extremes the “fantasy addict” or “romance junkie” goes to are sometimes hard for a clinician to accept. For example, consider a physician in her late thirties whose various romantic involvements were constantly turning her life upside down—including changing hospitals and practices. It became clear only after treatment was well underway that her affairs were all in her mind. None of these men were aware of all that she was bringing to the relationship with them. This type of behavior has very clear antecedents (e.g., emotional incest with opposite sex parent), collateral issues including sexual aversion with partner, and compulsive masturbation. From a courtship disorder perspective (Carnes, 2001) both patients are stuck in early phases of noticing and attraction. Here is where novelty seeking fits from a biology point of view. Illustration Six represents a Caucasian female patient’s representation of the constant pursuit of the unavailable. The flowers represent the escapism of yet another discovery, whereas the truth, represented by the desert images, reflects reality. In effect, she is receiving an “error message” from her brain and she now knows that.

Understanding this pre-frontal cortex-subcortical relationship is key to full acceptance of the most controversial area of addictions—the behavioral addictions. In a normal individual, pre-frontal cortical feedback to the NAc



**FIGURE 8** Fantasy.

regulates drive behavior. In the addicted state this regulation is distorted. The association between chemical dependence and a variety of behavioral addictions is well known. The ultimate common pathway of behavioral and drug reinforcement is likely to involve altered pre-frontal activation and ultimately impaired cortical regulation during addictive behaviors. Such effects have been demonstrated in animal models for psychostimulants, alcohol, and sucrose. Early efforts to study this in humans involve evaluation of complex interactions between prefrontal cortex, NAc, amygdala,

hippocampus, and other brain structures thought to be relevant to motivation, salience, and reward. As outlined in Table 6 the NAc serves as a hub receiving input into spiny neurons from each of these components. A process involving simultaneous activation of both dopamine receptors (from the lower brain) and glutamate receptors (from the cortex) in the NAc spiny neurons reflects this “hub” status. This simultaneous activation of neurons from primitive and cortical levels of the brain is the sine qua non of appetitive learning (Smith-Roe & Kelley, 2001) and is likely experienced as the “lightbulb” effect we all experience after gaining a hard-fought insight. In this manner, primitive and cortical functions are tightly linked, leading to long-term changes in the pattern of neural networking. At the point of simultaneous stimulation of the Nucleus Accumbens (reward center) by both primitive and cortical inputs in response to addictive behavior, the highjacking of a functional exploratory drive is complete.

**TABLE 6** Patient Recognition of Addiction Interactions (AIs) per Person in Ascending Order of Frequency

<b><u>No. of AIs/person</u></b>	<b><u>Percent</u></b>	<b><u>Number</u></b>
0	6.5	42
1	5.8	38
2	7.1	46
3	12.0	78
4	12.2	79
5	11.4	74
6	12.5	81
7	11.2	73
8	9.7	63
9	7.2	47
10	4.4	29
<b>TOTAL</b>	100.0	650

Further evidence of completion of the highjacking sequence is provided by the phenomena of craving/cueing. The “lightbulb” effect may occur with the slightest cue to the addictive process and indeed may be transformed from simple lightbulb to screaming ecstasy. Non-specific stimuli become conditioned to stimulate the reward center even in the absence of the chemical agent. An example is the well-known cueing of white powder for the cocaine addict. Craving may lead to use of the craved agent or substitution of another more readily available agent. The role of dopamine in this circuit seems to be to signal the cortex reward circuit that a reward may be available.

The ultimate evaluation and experience of reward is managed in the cortex. If the cue is followed by an expected dose of rewarding agent, the dopaminergic response to the cue will be enhanced; if not it will diminish. The net effect is to produce a conditioned response to a previously unconditioned stimulus. Thus, Pavlov described the basis of addictive reinforcement in 1903.

## HORMONAL MODULATION OF ADDICTIVE BEHAVIOR

Many other neurohormonal systems may be important in modulating addictive behavior, including sex steroids, stress steroids, and peptide hormones. An interesting example is found to underlie the separation of sexual compulsion and compulsive attachment. Interactions appear to be abundant among the spheres of sexual function, attachment, and opioid activity. Oxytocin is a peptide hormone known to modulate alcohol, opiate, and cocaine tolerance and dependence. Oxytocin is a likely important mediator of sexual-attachment interactions, including well known roles in regulation of orgasm, birthing, and lactation. Oxytocin release with ejaculation in males is known to be dependent on available opioid receptors. In turn, oxytocin has been the subject of comparative studies among various species of monogamous and non-monogamous voles (Fisher, 2004). Many patients have close attachment but little sexual activity with a spouse, and simultaneously have multiple sexual partners with no attachment. This supports the concept of sexual activity and attachment as separable, interacting phenomena. The peptide hormones, oxytocin, and a related peptide, vasopressin, appear to play a key role in this separation. The action of oxytocin appears closely linked to modulation of the dopamine reward cascade via opiate receptors (Fisher, 2004; Money, 1986).

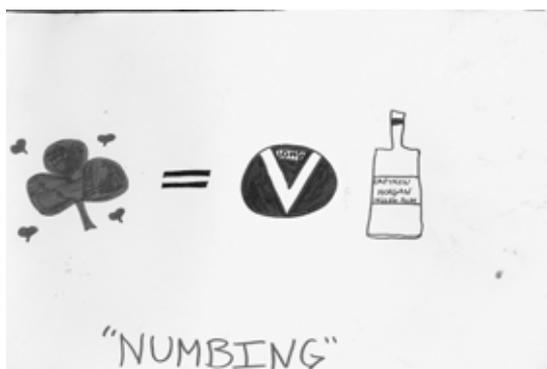
Not all addictive agents activate the brain reward system. One that seems to diminish activity of the brain reward system is any benzodiazepine. All benzodiazepines enhance the action of GABA. They do so by binding to the GABA-A receptor, altering that receptor so that it binds GABA more tightly (Nutt & Malizia, 2001). The result is a reduction in exploratory drive motivation and a decrease in dopamine activity in the NAc. The exact mechanism involves changing the properties of the GABA-A receptor so that GABA itself may be more potent. Other agents producing similar results (barbiturates, alcohol) are known to act on the GABA-A receptor. The result is a potential for synergism based on a summation of these actions.

All drugs of this class are frequently sought by abusers of a variety of agents to ease anxiety resulting from hyperarousal of the brain reward system. Of the drugs in this group, certainly the most ubiquitous is alcohol. As discussed previously, alcohol does have significant brain reward system effects. When used chronically, it impairs the synthesis of one of the components of the GABA-A receptor, increasing NAc dopamine efflux by diminishing GABA activity. More acutely however, it potentiates GABA by acting to open the chloride channel of the receptor. Thus, alcohol is involved over the entire spectrum of exploratory drive, both activating and diminishing it in a phasic manner. We believe this fits well with commonly observed clinical effects of alcohol. Cross tolerance between alcohol and benzodiazepines has been heavily documented. No endogenous benzodiazepine-like compounds have yet been identified despite extensive searching. Until these are identified, it is difficult to describe the

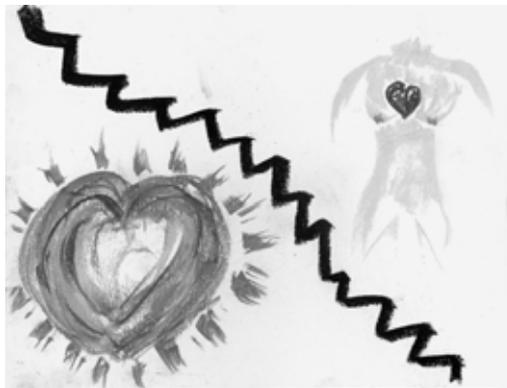
natural role of GABA and its receptor in neural regulation. We believe that its function lies in protection from the disruptive anxiety associated with high levels of exploratory drive activity. Conceptually it would be anchoring the low end of a continuum of exploratory drive intensity. All of the GABAergic addicting drugs are known to have powerful interactions with other drugs that stimulate the brain reward system in the NAc by facilitating withdrawal mediation, cross tolerance, disinhibition, combining, numbing, masking, and replacement.

In our experience, sexual addiction presents with at least two principal patterns of response to sex. First, the majority of behavioral events involve seeking ever more stimulating and arousing experiences. This pattern of behavior reflects activity in the brain reward system and the association with novelty that has been emphasized above. Second, a significant number of patients frequently exhibit a more passive sexual response that reflects a feeling of satiety. This pattern may reflect the opposite pole of the exploratory drive represented by the action of benzodiazepines. It is easy to speculate that endogenous benzodiazepines may play an important role. Sexual satiety has been associated with a decrease in NAc dopamine and an increase in hypothalamic serotonin (Lorrain, Riolo, Matuszewich, & Hull, 1999). In essence, a continuum of responses exists along an axis that is correlated with exploratory drive. GABA activity is stimulated at one end and inhibited at the other. The role of GABA seems to define a “state” that serves to modulate the primary dopamine-driven reward system. Serotonin is known to have a reciprocal relationship with dopamine and probably serves as a marker of satiety in general. The clinical model of arousal, satiation, and fantasy developed by Milkman and Sunderwirth (1987) correlates well with the neurochemical model presented here. This clinical model will be discussed further in a later section.

Figure 9 was made by a 26-year-old Caucasian female college student. Her diagnoses were sedative/hypnotic, alcohol dependence, and cannabis abuse. She also identified herself as a “love addict.” The patient argues that she is not a true alcoholic or drug addict. Rather she is “codependent” to the point that she uses pills and alcohol to medicate herself against the realities



**FIGURE 9.** Numbing.



**FIGURE 10.**State dependence.

of a hopeless love affair. The majority of her energy, behaviorally and chemically, is dedicated to a common goal of maintaining a state of tranquil satiety through as much of her life as possible. Exploratory activity is minimal. Her life is minimal. In the second author's experience, this patient may best respond to medication with dual action serotonin/norepinephrine inhibitors (Effexor or Cymbalta) in conjunction with psychotherapy.

The vulnerability of the brain reward system to "hijacking" may be modified by mood states. Depressed patients exhibit a hypersensitive response of the brain reward system. This hypersensitivity seems to reflect a baseline hypofunctional state present in severely depressed patients. Figure 10 was drawn by a 41-year-old Caucasian female, married with three children. She suffers from Major Depressive Disorder, Dysthymic Disorder, and Sexual Disorder NOS. The lower left portion of the page is filled with a brightly colored heart design with a range of bright colors: greens, reds, purples, yellows, and pinks. It is large, congested to the point of bursting with the threat of released pent up emotion. In sharp contrast is the image presented on the right side of a prominent schism. The patient is depicted as anonymous, helpless, and virtually transparent. In further contrast is a highly visible heart that is both small and dark. This heart appears open at the top, which may represent a point of access to fill the empty void within.

The patient does not feel either image is healthy. She uses the excitement of illicit love affairs to ameliorate her emptiness. In the long run, she simply becomes more estranged from her authentic self and her husband.

Social defeat has a complex effect on the self-administration of cocaine in an experimental setting. The importance of "state dependence" is illustrated by the fact that patients treated for cancer pain with addictive opiates rarely become psychologically dependent. In these circumstances, intact processing in the pre-frontal cortex identifies a specific need for the opiates and limits runaway reinforcement by upstream regulatory feedback. Similarly "state dependence" invoked by a committed relationship may serve to regulate sexual behavior.

## NEUROBIOLOGY SUMMARY

In summary, brain neurochemical interactions are understood at a level that allows the development of a broad model involving neurochemical events associated with a “highjacking” of exploratory drive in mammals. This exploratory drive may be considered as a continuum with extensive modulation by benzodiazepine receptors at one extreme and psychostimulant receptors at the other. The agents activating these receptors may either lie within or be encountered in the environment. This model is predictive of a substantial number of neurobiological interactions between addicting agents that are fully consistent with the full range of behavioral interactions discussed previously. Many of these interactions have been clinically and biochemically demonstrated. More detailed review of these interactions is certainly available. The process of expanding this model to include all relevant applications is ongoing. Biological aspects involving structural modification of the central nervous system are beyond the scope of this paper, but represent another important layer in the neurobiology of addictive interactions (Kelly, 2004). These structural changes produce an ever-decreasing correlation between psychosocial stressors and addictive acting out, making the highjacking a durable change. The ensuing behavior reflects a distorted control over cortical feedback by primitive reward systems emanating from the midbrain. The subcortical part of this learning process reflects the automatic stereotypical responses of lizards leading to ritualization and repetitive compulsions refractory to rational redirection.

Successful treatment mandates the restoration of cortical control over such pre-conscious primitive motivation. These pre-conscious functions emerge on a daily basis in psychotherapy under the rubric of “feelings.” These feelings become the center of a cognitive reframing process that leads to behavioral change reflecting extensive cortical action. Ultimately, the pathological reinforcement of addictive agents will be replaced by a restored cortical regulation reflecting additional structural changes in the brain superimposed on the pattern of addiction. The underlying addictive pattern remains susceptible to re-activation. Twelve-step programs have provided an ideal venue to achieve this reframing through affirmation and consistent self-evaluation and recognition that addictive disease reflects a lack of conscious control (powerlessness). Clinicians who achieve a basic understanding of addiction interactions and relevant neurobiological background will be in a better position to serve the needs of their client. This background will allow inclusion of the predictive value of a theoretical framework as well as empirical technique in treatment protocol.

## ADDICTION INTERACTION IN CLINICAL PRACTICE

The obvious implications of addiction interaction as a metamodel concept start with screening and assessment. The model provides a framework for not only accessing all the addictions involved, but also determining how they affect one another and the patient’s functioning. More importantly, it provides a more useful tool to understand addictions as a “package” and not as coexisting pathologies to be approached individually and perhaps sequentially. One of the initial challenges is deciding which addictions to approach first or if they can be approached simultaneously. Our position

is that they can be addressed simultaneously and sequentially.

A hierarchy exists in approaching multiple addictions. Violence is the first clinical issue. Violence towards others and violence to self must be contained. Then an assessment of all the issues involved must take place. Often the outlines of all the addictions do not appear at once. The clinician starts with the ones that are apparent with full confidence that more will be revealed. Yet the clinician's best efforts should be to initiate a withdrawal process. Clearly if substance abuse is present, ceasing chemical use is mandatory; otherwise therapy is fruitless.

Yet, if they are chemically dependent and sexually addicted at the same time, focusing only on the chemicals also becomes self-defeating. Clear boundaries around sexual behavior would be the minimum. Hence most treatment facilities ask for an abstinence agreement for a minimum amount of time. This contract is an opportunity to facilitate the withdrawal process from the sexual acting out. Not to do this will create untenable situations within the treatment milieu. For example, seducing other patients may become the focus of the sex addict who had no guidelines or awareness of multiple issues. It amazes us how "cosmic relationships" can blossom in a withdrawal context. Clearly, multiply addicted patients will require a residential stay or intensive outpatient supervision to accomplish a comprehensive withdrawal process. To focus on one addiction at a time allowing other addictive behaviors to persist is self-defeating. Once engaged in the treatment process, patients must explore each addiction in depth. If chemically dependent, there is much to learn about pharmacology, the culture of drugs, and the impact of drug use on behavior. If sexually addicted, there is much to learn about sexual development, the arousal template, and issues specific to relapse prevention. If problem gambling is the issue, the patient needs to learn about risk and the brain, the role of the windfall fantasy, and money obsession. Each area presents the challenge of information, specific strategies critical to recovery, and personal understanding of the etiology of the illness.

There also must be a way for patients to see how their compulsive behaviors fit together. Combining depth of experience with the breadth of interaction is the chief challenge of addiction today. We must develop curriculum and treatment designs that allow patients to get what they need to address each addiction and yet integrate them in a substantive way. We believe this to be the central factor in reducing recidivism rates in addiction treatment. What follows are three examples of clinical strategies that could be used in a more comprehensive approach to addictive illness. They are the multiple addiction timeline, the Neural pathway interview, and the addiction interaction self-assessment.

### Multiple Addiction Timeline

We ask patients to construct a timeline of the major events of their life. They then stratify the timeline by making a line for each addiction in which they determine the onset, worst moments, examples of powerlessness and unmanageability, and key events (e.g., DUIs or arrests). This allows the patient to notice how the addictions relate to one another. For example, phases become quite clear in which one addiction becomes prominent and then switches with another in some patterned way. Or the patient will notice periods in which all addictions were escalating simultaneously.

The timelines also allow the patient and therapist to notice what events triggered

addictive behavior. Specific family issues might appear for example. The therapist has the opportunity to notice how losses, grieving, and trauma are woven into addictive bingeing. The more comprehensive picture sets the stage for exploring the underlying issues driving the addictive process. Even at this point the issues of long-term treatment start to emerge. Patients report that this process is relieving because they understand the mechanisms of their use. They begin to see they are not hopeless degenerates but responding to wounds that if addressed will allow them to live a very different life. The multiple addiction timeline also facilitates acceptance of the “package” their combined illness presents. While it is important to see each one, taken together there is new appreciation of the pressures of their lives.

### The Neural Pathway Interview

Another strategy is the Neural pathway interview. Patients receive a brief lecture on the addiction neuropathways of the brain. We have found the model of arousal, satiation, and fantasy developed by Milkman and Sunderwirth (1987) a useful way to talk to patients about how their brain uses different behavior to achieve different ends. We also include deprivation as another strategy the brain uses to cope with stress. It is important that the patient accepts that a full understanding of addiction requires knowledge of how an addict uses compulsive behavior to achieve various ends. Knowing the end results opens up a discussion on how to prevent relapse.

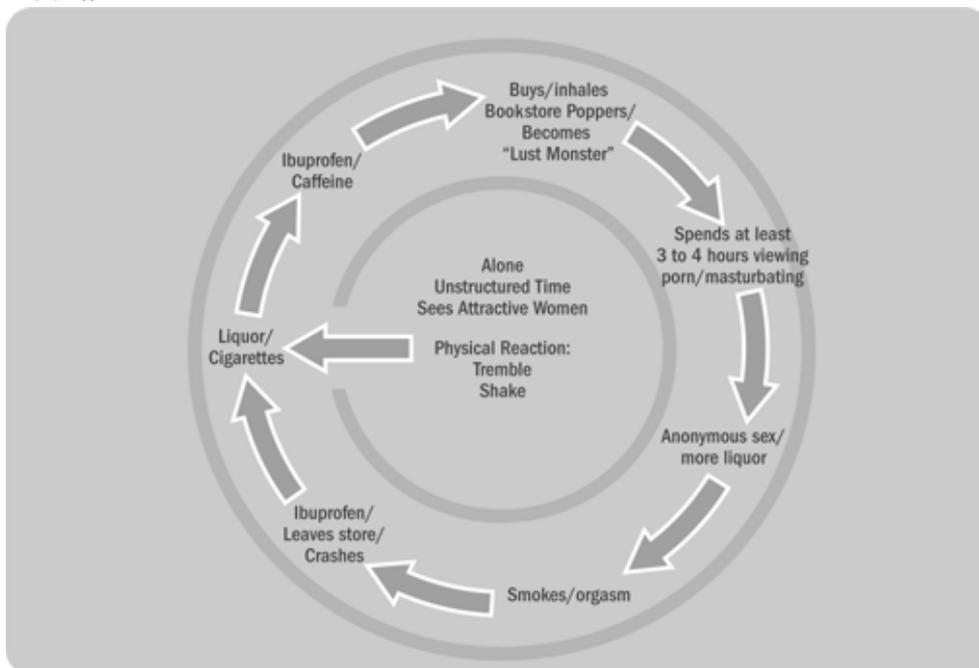
Then the counselor uses a large piece of newsprint to “interview” patients on the typical scenarios that emerge in their lives as addicts. Usually this is done in a group context so that all can benefit from one another in understanding how the complex weave of compulsive behaviors work. Each patient tells their typical patterns and the counselor then draws the sequence of events on the newsprint. As the counselor interviews the patient, care is taken to tease out if the behavior was about arousal, efforts to relax, or to escape. The counselor also guides the patient in exploring the role of deprivation. As each patient presents, there is often excitement as the patients start to get it. Literally there is a bandwagon effect in seeing how much they have in common.

Important realizations emerge out of the process. Patients start to discriminate that their addiction is not the same all the time. Consider the male patient who described acting out in a men’s room. Often it was exhilarating and he feared no consequences. At other times he felt very fearful and just wanted to find someone he could have sex with in order to sleep. Two distinct patterns emerge with very different affective sets to them. Yet the same compulsive behavior was part of each pattern.

The other discovery has to do with how ritualized and woven together the compulsive behaviors are. Consider the following examples. In Figure 10, a woman patient described a very predictable cycle in her life. She used LSD, Ecstasy, and cocaine prior to sex. This cycle began with guilt about her sexual behavior followed by the use of marijuana to calm down, which led to binge eating. After sleeping she would use Ecstasy to get up in the morning so she could send her kids to school. Once patients experience the Neural pathway interview, they are able to make the patterns explicit, label the affect and purpose of each compulsive phase, and name the triggers for the behavior.

Figure 11 was developed from a Neural pathway interview with a 37-year-old

professional man. It begins in the middle with an awareness that being alone with unstructured time would start a cycle in him in the form of noticing an attractive woman. He was aware that as it started his body would tremble and shake—the result of a combination of fear, arousal, and anticipation. He then would find hard liquor, drink and smoke. Next he would get in the car and head for the adult bookstore. On the way he would take caffeine tablets and ibuprofen, knowing from experience that loading up on these would help with the headache that would ensue. At the adult bookstore he would buy inhalants (poppers) which heighten viewing pornography. The inhalants



**FIGURE 11.** Neural pathway interview

made him into what he termed a “lust monster.” He would spend the next three to four hours masturbating and watching pornography.

The next stage was to initiate sex with others in the bookstore. In order to do that, he would drink more liquor. Before an orgasm he would smoke; after orgasm he would leave the store taking additional doses of ibuprofen. After getting home he would “crash” and sleep it off, days passing before recovering. This pattern was highly ritualized, with each step needing to be in place to work. He was combining compulsive behaviors, creating an architecture embedded in his brain’s cravings. To label him an alcoholic would be beyond simplification. To call him a sex addict trivialized the stress that he put his body through. By assisting the delineation of the pattern, therapy can trace back to how it emerged and the functions it performed for the brain.

Our patients attend an addiction interaction workshop designed to understand the patterns in the interaction of their compulsive behaviors. We present a standardized list of criteria of all addictions and talk about how various addictions might fit the criteria. Using a standard Likert scale, patients then rate each addiction they have. Then they total up their ratings for each addiction. Figure 12 is a sample of the worksheet they use in the workshop. The goal of this phase of the experience is to teach them the common characteristics of addictions. The net impact is that the patients' own self-assessment puts a valence on how powerful their addictions are in their lives. It is routine to hear patients make comments like, "I used to think my problems were about chemicals. What I realized is that sex addiction was my most serious problem."

Criteria	Example: Drug Addiction	—	—	—	—	—	Row Totals
1. Loss of control	4						
2. Compulsive...behavior	4						
3. Efforts to stop	2						
4. Loss of time	3						
5. Preoccupation	5						
6. Inability to fulfill obligations	2						
7. Continuation despite consequences	3						
8. Escalation	3						
9. Losses	5						
10. Withdrawal	1						
Totals							

**FIGURE 12.** Criteria. Rate each criterion in each addiction separately. Use the following scale to rate each addiction: 1-never a problem, 2-occasionally a problem, 3-sometimes a problem, 4-often a problem, 5-consistently a problem.

The second phase of the workshop continues the process with a lecture on addiction interaction disorder. The ten criteria described earlier in this paper are presented, followed by a discussion of each characteristic. Patients are then given worksheets in which they record various ways they fit the ten patterns. They are then asked to think through the implications of what they have recognized for relapse prevention. Next they discuss the key question of how addictions interact to make them more vulnerable to relapse. The patients then bring this information back to their groups and treatment process.

The original patterns of addiction interaction evolved out of discussions with patients and clinical observation. While clinically useful, we were concerned whether patients themselves would be able to identify and use the concepts. We believe that for a concept to be useful it needs to be understood by patients as relevant to their experience. Between June of 1999 and January of 2003, 650 patients participated in the addiction interaction workshop. Through content analysis of patient worksheets we were able to pinpoint when patients thought particular dimensions fit their addiction

pat-terns. Table 6 summarizes the patients' ability to recognize the patterns in their own lives.

Only 6.5% (42 out of 650) of the participants found none of the concepts relevant. On the other extreme, 4.4% felt they could identify all ten criteria.

**TABLE 7** Patient Recognition of Specific Addiction Interaction Dynamics

<b><u>Addiction interactions</u></b>	<b><u>Percent</u></b>	<b><u># Patients per addiction interaction</u></b>
1. Cross tolerance	60.8	395
2. Withdrawal mediation	55.8	362
3. Replacement	42.8	278
4. Alternating cycles	41.5	269
5. Masking	45.3	294
6. Ritualizing	41.5	267
7. Fusion	61.4	399
8. Numbing	53.8	350
9. Disinhibiting	41.7	271
10. Combining	46.1	299
11. Inhibiting (As a mostly spontaneous write-in by patients)	6.8	44
<b>TOTAL</b>		650

Table 7 summarizes the patient recognition by each criterion. The very least recognized criterion was alternating addiction cycles (41.5%) and the most identified was fusion (61.5%). One of the most interesting outcomes was a write-in category. A number of patients individually added another criterion: inhibiting. They explain that they used one addiction to inhibit the use of another. This "lesser of two evils" option emerged spontaneously in 6.8% of the sample.

This research was conducted to see if the concepts were recognizable to patients as a viable way to validate the concepts. The data reflect the teaching of the workshop as well as the influence of the groups. Further content analysis is subject to error and rater evaluation. Yet the strong results are suggestive of the viability of addiction interaction disorder as a viable concept that is useful to patients. Further validation comes from this "hypothesis seeking" approach since a new category also emerged out of patient experience.

The next step would be to construct a dichotomous questionnaire and interview protocol that could be tested for reliability and validity. Once developed, these assessments could be used with large samples to research prevalence in a more

systematic, empirical fashion. Once established, the most important question could be asked and answered—Do programs that use multiple addiction approaches and use addiction interaction disorder as a concept have better outcomes? We believe that would be the case.

## IMPLICATIONS AND DISCUSSION

Addiction interaction disorder as a concept cuts to the core of much of the controversy about addictive disorders. If addictions interact, then at some primary levels they share etiology and structure. In the past, each issue was often treated separately, with specialists in each area largely disconnected from specialists in other areas. The result of such an approach is a piecemeal approach with often scattered results. We vastly underestimate what we are up against and keep searching for the silver bullet that will increase our recovery rates. Further, addiction professionals must develop the political strength not to be subsumed by a pharmacologically driven mental health community which often has an anti-addiction bias.

It is bad enough that some mental health professionals still do not accept alcoholism. Even worse is to see addiction stigmatized as a willpower problem that is really about character. The most unacceptable is that addiction professionals have not banded together across various specialties to martial political support, funding, and research so that success for our patients is more achievable. A more thorough understanding of the neurobiological realities that are common to each of the disciplines involved could evolve into a unified concept and a common language accepted by all.

If addiction interaction were to prove a viable, validated concept, it would clear up much of the conceptual chaos that Howard Shaffer (1997) and others have so patiently documented exists among addiction researchers. Further, the diagnostic frameworks of the *DSM* could more systematically be shaped to fit our patient realities rather than schools of thought and professional training. The biggest challenge would come to us as addiction professionals. If each patient was to receive the depth of treatment in each addiction and the breadth of treatment necessary across issues, the 28 day program loses its legitimacy. Recovery will be a three-to five-year process involving many specialties and formats. What we do would change. Consider how understanding addiction interaction would change the breadth of a “first step.” Or, think about the person who has a number of addictions and how they handle going to 12-step meetings. If they went to every meeting they qualified for they would not have a life. This is not to negate the value of 12-step programs, but rather to point out the level at which things would have to change.

The field of sex addiction is in many ways subversive to the larger field of addiction. Understanding gambling or compulsive overeating from an addiction framework does not offer the same challenges that sexual compulsivity does. Part of the struggle has been resistance to seeing sex as yet another manifestation of the eternal struggle of too much and too little. Acceptance of this concept has been clouded to some degree by larger issues of cultural and even scientific resistance to talking about sexual issues in general. Factor in sexual politics, divergences, and even competition among professional allegiances, and it becomes rapidly much more convoluted than

some of the other addictions. Yet for sex addiction to take its place at the table of clinical deliberations, we must not only show how the addiction works, but also how it interacts with other addictions. Addiction interaction as a paradigm may help with that transition.

## REFERENCES

- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC.
- Amico, J. (1997). Assessing sexual compulsivity/addiction in chemically dependent gay men. *Sexual Addiction & Compulsivity: Journal of Treatment and Prevention*, 4, 291–297.
- Bateson, G. (1978). Breaking out of the double bind. *Psychology Today*, 12(3), 43–51.
- Campbell, R. J. (1996). *Psychiatric Dictionary* (7th ed.). New York: Oxford University Press.
- Carnes, P. (1998). The case for sexual anorexia: An interim report on 144 patients with sexual disorders. *Sexual Addiction Compulsivity: Journal of Treatment and Prevention*, 5, 293–309.
- Carnes, P. (1991). *Don't call it love: Recovery from sexual addiction*. New York: Bantan.
- Carnes, P. (2000). Sexual addiction and compulsion: Recognition, treatment, and recovery. *CNS Spectrums*, 5(10), 63–72.
- Carnes, P. (September 2001). *Addiction Interaction Disorder Core Lab*. Information presented at The September Institute, Scottsdale, AZ.
- Carnes, P., & Schneider, J. (2000). Recognition and management of addictive sexual disorders: Guide for the primary care clinician. *Primary Care Practice*, 4(3), 302–318.
- Combs, R. (Ed.). (2004). *Handbook of addictive disorders: A practical guide to diagnosis and treatment*. New Jersey: John Wiley & Sons.
- Cooper, A. (2000). Cybersex: The dark side of the force: A special issue of the journal of sexual addiction and compulsivity. New York: Brunner-Rutledge.
- Cooper, A. C., Putnam, D. A., Planchon, L. A., & Boies, S. C. (1999). Online sexual compulsivity: Getting tangled in the net. *Sexual Addiction & Compulsivity: Journal of Treatment and Prevention*, 6(2), 79–104.
- Csikszentmihalyi, M. (1990). *Flow: The psychology of optimal experience*. New York: Harper & Row.
- Derlet, R. W., Tseng, J. C., & Albertson, T. E. (1992). Potentiation of cocaine and d-amphetamine toxicity with caffeine. *American Journal of Emergency Medicine*, 10(3), 211–216.
- Fiorino, D. F., & Phillips, A. G. (1999). Facilitation of sexual behavior and enhanced dopamine efflux in the nucleus accumbens of male rats after d-amphetamine induced behavioral sensitization. *Journal of Neuroscience* 19(1), 456–463.
- Fisher, H. (2004). *Why we love: The nature and chemistry of romantic love*. New York: Henry Holt & Co.
- Goldstein, R. Z., & Volkow, N. D. (2002). Drug addiction and its underlying neurobiological basis: Neuroimaging evidence for the involvement of the frontal cortex. *American Journal of Psychiatry*, 159, 1642–1652.
- Gordon, L. III, Fargason P., & Kramer J. (1995). Sexual behaviors of patients in a residential chemical dependency program: Comparison of sexually compulsive physicians and nonphysicians with non-sexually compulsive physicians and nonphysicians. *Sexual Addiction & Compulsivity: Journal of Treatment and Prevention*, 2(4), 233–255.
- Griffin-Shelley, E., Sandler, K. R., & Lees, C. (1992). Multiple addictions among dually diagnosed adolescents. *Journal of Adolescent Chemical Dependency*, 2, 35–44.
- Huebner, H. (1993). *Eating disorders, endorphins, and other addictive behaviors*. New York: Norton.
- Irons, R., & Laaser, M. (1994). The abduction of fidelity: Sexual exploitation by clergy—Experience with inpatient assessment. *Sexual Addiction & Compulsivity: Journal of Treatment and Prevention*, 2(2), 119–129.
- Irons, R., & Schneider, J. (1994). Sexual addiction: Significant factor in sexual exploitation by health care professionals. *Sexual Addiction & Compulsivity: Journal of Treatment and Prevention*, 1, 198–214.
- Johnston, J. F. (1855). *The chemistry of common life (vol. II)* New York: D. Appleton & Co.
- Johnson, M. (1999). *Cross-addiction: The hidden risk of multiple addictions*. New York: Rosen.

- Kelly, A. E. (2004). Memory and addiction: Shared neural circuitry and molecular mechanisms. *Neuron*, 44(1), 161–179.
- Leshner, A. I. (2001). *Addiction is a brain disease*. The Addiction Recovery Guide Website. Available [www.addictionrecoveryguide.org/articles/article151.html](http://www.addictionrecoveryguide.org/articles/article151.html)
- Lorrain, D. S., Riolo, J. V., Matuszewich, L., & Hull, E. M. (1999). Lateral hypothalamic serotonin inhibits nucleus accumbens dopamine: Implications for sexual satiety. *Journal of Neuroscience* 19(17), 7643–7652.
- MacLean, P. (1990). *The triune brain in evolution: Role in paleocerebral functions*. New York: Plenum.
- Milkman, H., & Sunderwirth, S. (1987). *Craving for ecstasy: How our passions become addictions and what we can do about them*. San Francisco: Josey-Bass.
- Miller, N. S., Belkin, B. M., & Gold, M. S. (1990). Multiple addictions: Co-synchronous use of alcohol and drugs. *New York State Journal of Medicine*, 90, 596–600.
- Miller, N. S., & Gold, M. S. (1995). The role of the psychiatrist in treatment or relapse in addictive disorders. *Psychiatric Annals*, 25, 673–678.
- Minuchin, S., Rosman, B., & Baker, L. (1978). *Psychomatic families*. Boston: Harvard University Press.
- Money, J. (1986). *Lovemaps: Clinical concepts of sexual/erotic health and pathology, parahilia, and gender transposition in childhood, adolescence, and maturity*. New York: Irvington.
- Nutt, D. J., & Malizia, A. L. (2001). New insights into the role of GABA a benzodiazepine receptor in psychiatric disorder. *British Journal of Psychiatry*, 179, 390–396.
- Orford, J. (1985). *Excessive appetites: A psychological view of addictions*. New York: John Wiley.
- Pavlov, Ivan (1903). The experimental psychology and psychopathology of animals. Presented at the 14th international medical congress. Madrid, Spain.
- Petrucci J., & Stuart, C. (Eds.). (2001). *Hungers and compulsions: The psychodynamic treatment of eating disorders & addictions*. Northvale: Jason Aronson.
- Robertson, N. (1988). *Getting better*. New York: Morrow.
- Schull, N. (2003). *Living with the machine: An ethnography of gambling addiction in Las Vegas*. Doctoral dissertation, University of California, Berkeley.
- Schwartz, M. F., & Cohn, L. (Eds.). (1996). *Sexual abuse and eating disorders: A clinical overview*. New York: Brunner/Mazel.
- Shaffer, H. J. (1997). The most important unresolved issue in the addictions: Conceptual chaos. *Substance Use and Misuse*, 32(11), 1573–1580.
- Smith-Roe, S. L., & Kelley, A. (2001). Coincident activation of NMDA and dopamine D1 receptors within the nucleus accumbens core is required for appetitive instrumental learning. *Journal of Neuroscience*, 20(2), 7737–7742.
- Stewart, S. H., Angelopoulos, M., Baker, J. M., & Boland, F. J. (2000). Relations between dietary restraint and patterns of alcohol use in young adult women. *Psychology of Addictive Behaviors*, 14, 77–82.
- Sweeting, P. D., & Weinberg, J. L. (2000). Gambling: The secret invisible addiction. *Counselor: The Magazine for Addiction Professionals*, 1, 46–50.
- van der Kolk, B. (1988). The trauma spectrum: The interaction of biological and social events in the genesis of the trauma response. *Journal of Traumatic Stress*, 1(3), 273–290.
- van der Kolk, B. (2001). The assessment and treatment of complex PTSD. In R. Yehuda (Ed.) *Traumatic stress*. American Psychiatric Press.
- van der Kolk, B., McFarlane, A., McFarlane, C., Weisqeth, L. (1996). Assessment of post traumatic stress disorder in clinical and research settings, 11, 242–271. New York: Guilford Press.
- Washton, A. M. (1989). Cocaine may trigger sexual compulsivity. *U.S. Journal of Drug and Alcohol Dependency*, 13(6), 8.
- Wines, D. (1997). Exploring the applicability of criteria for substance dependence to sexual addiction. *Sexual Addiction & Compulsivity: Journal of Treatment and Prevention*, 4, 195–215.

- Winters, K. C., Bengston, P., Dorr, D., & Stinchfield, R. (1998). Prevalence and risk factors of problem gambling among college students. *Psychology of Addictive Behaviors, 12*, 127–135.
- Wolf, M. E. (2003). LTP may trigger addiction. *Molecular Interventions, 3*(5), 248–252.