

DRUG DREAMS: A NEUROPSYCHOANALYTIC HYPOTHESIS

Recent studies have shown that the ventral tegmental pathway stimulates both dreaming and drug craving. To investigate a possible clinical link between these two psychic phenomena, psychotherapy notes from the first six months of an addicted patient's treatment were reviewed, together with verbatim notes from the four years of psychoanalysis that followed. Of 240 dreams reported by the patient, 58 had manifest content involving the seeking or using of drugs. There was no particular temporal or emotional thematic pattern to these "drug dreams," which persisted through four and a half years of sobriety. Drug dreams are observable phenomena that reflect both the innate structure of the brain and neural changes produced by exposure to addictive drugs. In some addicted persons, exposure to drugs produces a fixed change in neurological functioning with which they must contend for years, possibly the rest of their lives. Drug craving meets Freud's defining characteristics for a drive: it is a constant pressure, originating from within the organism, to do work, and it constantly demands satisfaction. Because ego and libidinal drives share a common neural pathway, they should not be separated conceptually. Solms's finding (in press) that the activating systems for dreaming and for craving are identical, a finding based on observations of tumor- or stroke-provoked brain lesions, is confirmed by observation of the dreams of a patient whose brain changes were created by drug exposure. This study provides further evidence that the origin of the dream is a wish.

Drug addiction is responsible for 25 percent of all deaths in the United States; of these, nicotine accounts for 19 percent, alcohol 5 percent, and other drugs almost 1 percent (McGinnis and Feoge 1993).

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In a previous paper (Johnson 1999) I noted three forces that contribute to an addictive diathesis. Addictive illness can be a response to an inability to tolerate affect; people who can't stand their feelings may find that addictive behaviors divert them. Second, for individuals who lack object constancy, addictive behaviors can give a sense of being accompanied. For instance, adolescents who have not been able to internalize a good parent may find that they can use an addiction to separate from their family of origin, much as younger children use transitional objects to negotiate separation-individuation. The third force is hypothesized to be a change in brain functioning in addictive illness, a biologically based craving for addictive drugs. This brain change has been demonstrated in work with animals. The persistent change in neurological functioning caused by exposure to addictive drugs is demonstrated clinically in this paper by using a neuropsychanalytic method.

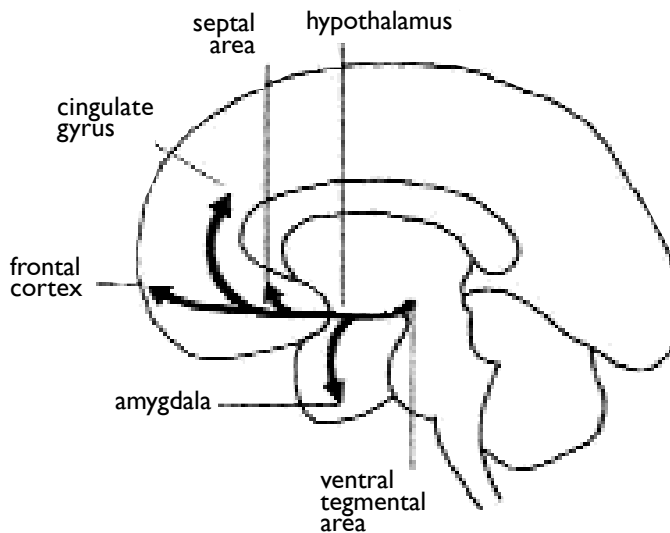
Neuropsychanalysis attempts to link mind and brain in order "to dissect the internal psychological structure of the various changes in personality, motivation, and complex emotion that occur following damage to different cerebral structures. Thereafter, the multiple underlying factors producing these symptoms and syndromes can be identified and each correlated with its anatomical 'scene of action'" (Kaplan-Solms and Solms 2000, p. 62). Recent neuropsychanalytic studies have considered alterations in psychological functioning caused by loss of brain tissue (Kaplan-Solms and Solms 2000; Solms 1997). This study examines the alteration in psychological functioning occasioned by increased neurotransmission along the ventral tegmental pathway (see figure, opposite).

The ventral tegmental pathway is a drive pathway that provokes animals to seek food, water, and sex (Miller and Gold 1993). It is hijacked by addictive drugs via irregular, episodic drug exposure producing craving for the drug and for drug-associated stimuli (Robinson and Berridge 1993, 2000; Berridge and Robinson 1998). This craving is apparently long-lasting (Robinson and Berridge 1993). Shevrin (1997) has suggested that drug craving is an instance of drive expression. The only way to help individuals who have developed this drive may be psychotherapy that helps them withstand the constant desire for repeated drug experiences despite overwhelming negative consequences (Robinson and Berridge 2000).

The same ventral tegmental pathway activates dreaming (Solms in press). The activation-synthesis hypothesis states that a pontine

pathway stimulates dreaming during REM sleep (Hobson 1999). Solms's assertion (1999, in press) is that REM sleep now seems merely to reflect general activation of the brain. He explains that lesions of the ventral tegmental pathway completely obliterate dreaming in a way seen with only one other type of lesion, a lesion in the parieto-temporo-occipital area, which integrates dream images (Solms in press).

Drug dreams are seen ubiquitously in the treatment of various addictions: cocaine (Jerry 1997), alcohol (Denzin 1988), opiate (Looney 1972) and nicotine (Persico 1992). Flowers and Zweben (1998) reported a cocaine craving dream in a client completely sober for ten years. A Medline search for "drug dreams" and "using dreams" found no papers suggesting a mechanism by which these dreams might be produced.



MARK SOLMS'S ELEGANTLY SIMPLE DIAGRAM OF THE DREAMING/
DRUG-CRAVING PATHWAY (VENTRAL TEGMENTAL PATHWAY).

Reprinted with permission from Solms (in press).

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CLINICAL CASE REPORT

Jane was a fifty-year-old divorced woman who asked for psychotherapy following heroin detoxification.

History

Jane's father was a bullying workaholic. He made a lot of money as a plumbing contractor, but everyone hated working for him. He constantly insulted the people around him.

Jane's mother was addicted to alcohol until she switched over to prescription opiate addiction in middle age. As Jane was growing up, she would often find her mother passed out in the house. The mother also hurt herself in various ways. Jane remembered hearing that one time, after her parents made love, her mother asked her father to go to the garage to get her cigarets. Upon returning, he found her in a pool of blood, having cut her arms.

Jane's brother King was two years older. As far back as she could remember, he had bullied her. She remembered him throwing darts into her back and asking her mother to pick them out. King used to smother her until she felt she might die. She would go limp, and see if he stopped. Or she would fight him and escape. At one point he threw her bike off a bridge into the river. At no point did her parents intervene to protect her. When King was eighteen he wrecked his new car while drunk. Two girls in the back seat died in the accident. All her father did was buy King a new car. Jane stayed away from King, who was drinking himself to death during her analysis.

Conflicts in the parents' marriage seem initially to have been addressed by the father's repeatedly buying a better house for the mother. Consequently, Jane attended one school through the third grade, another in the fourth, and two in the fifth, when the parents separated. Of her sixth-grade year, when she was 11, Jane said, "I was very alone. In school I could not divide or multiply, and probably not much of anything else either."

Six months after the divorce, the father married Marni, a classic wicked stepmother. Marni seemed to hate Jane, constantly denigrating her with comments, for example, about her developing breasts and how they would never be as large and attractive as her own. Jane lived with her mother and visited her father and Marni on weekends. The mother lived next door to a kind neighbor who helped, and who

knew what to do when Jane would find her mother passed out on the floor.

When Jane was thirteen, her mother started working from three in the afternoon to eleven at night. As a result, Jane was almost never around an adult. Her mother would be asleep when Jane went to school, and Jane would be asleep by the time her mother came home. King had been sent to live with the father.

Drug Exposure

Jane's exposure to addictive drugs is summarized in Table 1. Addiction is a complex process, and its neurobiological description does not explain why drug use begins. The need for an addiction as an object to ward off annihilation anxiety during adolescent separation from abusive and abandoning parents (Johnson 1993, 1999) is illustrated by the onset of Jane's use of addictive drugs. Jane's terrifying loneliness ended when she began drinking, and began idealizing her drinking, as seen below. In addition, it formed a symbolic link to her mother; they were both drinking.

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Table 1. Exposure to Addictive Drugs

Age	Addictive Drugs
15–18	alcoholic drinking only (alcohol continues with other drugs until sober)
18–44	nicotine
20	LSD, marijuana, oral amphetamines taken with oral opiate
21–23	intravenous amphetamine and barbiturates
23	intravenous opiates, including heroin, and benzodiazepines
24–28	methadone, barbiturates, benzodiazepines
28–31	only nicotine and alcoholic drinking
31–42	cocaine (nasal, smoked, intravenous), amphetamines, opiates
42–43	only nicotine and alcoholic drinking for a year, then heroin
43–44	only nicotine
44–47	sober
47	heroin
48	sober
49–50	heroin
50–55	no relapse to use of any addictive drug

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At fifteen Jane met her first boyfriend, and her first time drinking “got disgustingly drunk while with him.” She joined the local Y. “The next two times I drank was in this club. I got very physically ill from getting so drunk. . . . Its members consisted of female ‘friends.’ Finally, I belonged.” She and her two female friends got drunk together for the next three years. “I also started to take my mother’s wine each time she got drunk, which was every night.”

About this time, as Jane started to feel that she had somewhere she belonged, her mother warned her not to go out with her friends, “Or you’ll be sorry.” Jane remembers rebelliously sticking to her plans. When she returned, she found a trail of blood through the living room and found her mother lying passed out with her arms cut. Jane coolly called the next-door neighbor and then watched as the doctor stitched up her mother’s slashes.

At sixteen Jane met Bennie, a boy from an ethnic group of which her father disapproved. At seventeen Bennie raped her at his house while his mother was home. Jane was afraid to say anything, as if it was her fault, and decided that the proper thing to do under the circumstances was to marry him. Her father’s response was to disown her.

Her drinking intensified over the next two years. The only way Jane has ever been able to have any sexual contact is while intoxicated. She felt conflicted about whether to leave her mother to be with Bennie, and tended to oscillate back and forth between them. Her mother threatened suicide were Jane to leave her and dived head-first out a third-floor window. Her injuries left her partially crippled, and with chronic pain that occasioned her switching from alcohol to prescription opiate dependence.

Jane left Bennie when she was twenty (later to divorce him) and went to California with her next boyfriend, Joey. “The first drug I did in California was grass, which I hated from the get-go, but I continued to smoke it when I came back to Boston that July. I was escalating in my drinking while hanging around Beacon Hill. I was getting occasional blackouts along with horrible hangovers. Then quickly came LSD. I started ingesting p.o. [oral] speed, mostly ‘Black Beauties,’ and loved mixing them with p.o. Demerol. I did lots of acid with Joey and for some reason was now living with my father in Brookline.”

There were many violent stories from this time. Once she was drunk in an apartment where one man almost beat another to death. She sat there helpless and terrified. On another occasion, Joey and another

boyfriend, Jackie, got into a fight, and Jackie stabbed Joey through the heart. At first they thought Joey was dead, but then they decided it was worth trying to save him. Jackie dragged him down the stairs by his feet, threw him into the trunk of his car, dumped his body on the pavement in front of the Massachusetts General Hospital Emergency Room, and drove off. Joey lived.

“At twenty-one I met Buster, who was very willing to get me off with a minor request. He got me off with thirty Desoxyns [amphetamine pills that are ‘cold shaken’ with water to dissolve the drug and then injected] and I thought I was going to die. But of course, this did not deter me in any way. I continued shooting Desoxyns and crystal meth on a daily basis. I always shot barbs to come down at night. One time I shot at least twelve to sixteen Nembutals at once and found myself lying face down on someone’s bathroom floor thirty to forty hours later. . . .

“After a severe kicking from Buster I decided it was time to end that sick relationship so I went to Mass General because my arms were all infected and I couldn’t move my wrists. They applied casts to both arms and called my father. He came and got me and took me first to Bournewood [a private psychiatric hospital], whereupon they told him I was hopeless. Then he took me to Westboro [a state hospital] and had me committed. I ran away, never to return.

“A month after leaving the hospital I got septic from shooting Desoxyns and was pronounced clinically dead at Mass General. . . .

“At twenty-three I met boyfriend Gary and started injecting Numorphans [a cold-shaken opiate], “blues” [10 mg diazepam], and heroin. I absolutely loved it. This was the perfect drug I was looking for all along and in fact I felt great when I knew I had my first habit. I felt I really belonged.

“At twenty-four I got into Khantzian’s new methadone clinic with Gary in Cambridge. I continued to mix the methadone with barbiturates and benzos of course, in keeping with the standards and guidelines of any good methadone addict at a clinic.

“At twenty-eight I left the clinic while still on 35 mgs, after meeting Ralph. I got my GED and only drank wine minimally on Friday nights. No further drug use.

“At twenty-nine I started LPN [Licensed Practical Nurse] school, drinking on Friday nights only. No drugs.

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“At thirty I graduated from LPN school and left Ralph. I was class secretary and top of my class. I met Rob in Boston and traveled through Europe. I was drinking heavily now with many hangovers. No drugs.

“At thirty-one I met Steve in Europe and stayed with him for four years. . . . A member of the ski team offered me p.o. speed, which started the ball rolling again. I really wasn’t interested in doing it, I just did it to belong. I also did a lot of THC. Steve was a cocaine dealer, unbeknownst to me initially. I started snorting cocaine at his insistence, which I didn’t even like. Eventually I realized what I really wanted was Dilaudids, which I really liked. I mixed the cocaine with Dilaudids for the four years, mostly just on weekends for the first two years.

“At thirty-two I started UMass for the nursing associate degree program and was using cocaine, Dilaudids, and occasional heroin. I left a year later, three weeks short of being capped and pinned. I had a large habit on Dilaudids, which I was mixing occasionally with coke. I was dope sick all the time. I was working three jobs at the time; one was director of a nursing home in Somerville.

“At thirty-four I met Irv, another cocaine dealer. I was really going nuts with cocaine, shooting it.”

She remembered running out of veins. After Irv had shot his coke, Jane would lie on her bed with her head hanging off the end so that a vein in her forehead would pop out. Irv injected her there.

“At thirty-five I met Jim. I was doing mucho drugs. At thirty-six I was pregnant with my son, so I got married. I shot two bags of heroin before the church wedding. Jim knew all about my addiction beforehand. I told him, and he didn’t seem concerned.

“At thirty-seven I had my son, Ethan. I was on 5 mg methadone. I was refused a detox while pregnant, and he was born addicted. I didn’t do drugs for a while, but I don’t know how long. Eventually I started shooting Dilaudids and cocaine. I loved speedballs. I used to feel so awful when I wasn’t using.”

When Ethan was two, Jane got divorced. She continued to use drugs as above.

“When I was forty-one I was using the same drugs and I learned to ‘base’ cocaine by cooking it. Crack was not available at the time. I started smoking it and I really got whacked. I hated cocaine, but I was unable to stop. It was the easiest drug around. Bags of heroin were up to fifty dollars.

“When I was forty-two I went to detox. They tried me on Naltrexone (an opiate blocker). I was hoping if I tried to override it I would die. I thought that would be an appropriate, well-deserved ending. I stopped taking the Naltrexone and just drank booze. I was introduced to Narcotics Anonymous (NA). I thought I could drink, that I was different. I did only alcohol for approximately a year. Of course, I used heroin again and went to detox.

“I was sober for almost five years. I graduated with an RN, highest honors, and was class president. My father had a stroke which left him terribly impaired. Coincidentally I picked up booze again. That September I started my BSN. I was deathly ill from drinking too much. I was on the dean’s list the entire time I went for my BSN.

“At forty-seven I picked up Darvocets, which progressed to Percocets, then heroin. I ended up in detox again.

“At forty-eight I graduated summa cum laude. I was clean.

“At forty-nine I relapsed on my mom’s Percocet. I progressed to heroin as usual.

“At fifty I went to detox again, and started treatment with you.”

Jane was also addicted to nicotine, from the age of eighteen until she was forty-four.

All the way through her drug using, when Jane wanted to be held, she would go home and sleep with her mother. Her mother never offered to help her get treatment. Her mother did ask to watch her inject heroin.

Neurocognitive Considerations

Cocaine causes a storm of neurotransmitter release, including the acetylcholine that innervates the smooth muscle around arteries. The resulting vasospasm is most commonly experienced as chest pain (angina pectoris). Just at the moment when the heart is racing, and pumping harder against increased blood pressure, circulation through the coronary arteries is being choked off by smooth muscle spasm. Characteristically, users will put down the pipe for a few minutes until the blood cocaine level diminishes, and then go back to smoking.

However, there are no pain receptors in the brain. Decreased brain perfusion, with concomitant cognitive deficits, is common in cocaine use. It is especially common in “speedballing,” the combination of cocaine and opiates (Tumeh et al. 1990). In addition, Jane presented as an inattentive, foot-wagging, trunk-turning, head-bobbing tornado of constant motion.

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An initial cognitive screen came up normal. My diagnosis of attention deficit hyperactivity disorder (ADHD), however, was confirmed by an independent ADHD specialist. Stimulants were found to be effective (see Table 2). After two years of analysis, because Jane still seemed to be having so much cognitive trouble, a SPECT scan was done that showed "slight asymmetry of perfusion to the frontal lobes, with decreased perfusion most prominent in the right prefrontal cortex." This is congruent with ADHD, and not with cocaine-induced damage (Lou et al. 1990).

During the first three and a half years of psychoanalysis, every time Jane stopped her medication she would grow confused and angry. She would gyrate on the couch. After a week off medication she would threaten to quit psychoanalysis. I would explain that she couldn't use psychoanalysis without medication; she couldn't take in what was being said to her. She would then resume her medication.

Each medication tried for ADHD seemed to work better than the last. Eventually we decided that Jane's ADHD was improving as a result of her psychoanalysis, in terms of both attention and motor hyperactivity. We agreed to a trial off medication. At the time of this report, six months after her stimulant was discontinued, she has been able to do the work of analysis and function well in her life.

Table 2. Medications Prescribed for ADHD

Months Sober	Medication	Response
1	imipramine	intolerant of side effects
2	desipramine	intolerant of side effects
4	methylphenidate	markedly helpful at a dose of 60 mg/day, eventually refused because it caused insomnia
12	pemoline	ineffective at dose to 90 mg
15	bupropion	helped, but patient intolerant of headache, insomnia
24	dexedrine	definitely helpful, but patient refused to take more than 15 mg/day because of insomnia, muscle pain. (Given with trazodone 50 mg for insomnia)
42	Adderall	more helpful than dexedrine at 30 mg/day
48	medication stopped	

Psychoanalysis

After six months of once-a-week and then twice-a-week psychotherapy, we began psychoanalysis with the understanding that Jane had a life-threatening condition that had not responded to Narcotics Anonymous or Alcoholics Anonymous. She tended to denigrate the people she met in these programs, seeing herself as intellectually superior.

Jane began a special study of psychoanalysis as she began her own analysis. She started with Greenson's *Technique and Practice of Psychoanalysis* and became a sophisticated student of psychoanalytic literature. While this might not be right for every psychoanalysis, it had the effect of filling in an impoverished background. It helped anchor her alliance with me to know that there was a group of people who thought this way, in contrast to the people she had grown up with, who were all, in their own ways, unsocialized and unconnected.

Given her history of caregivers, establishing an initial working alliance was difficult. Of course, the four-days-a-week frequency and the analyst's relative silence are calculated to allow the patient to play out problems experienced in previous relationships. Jane's transference was constantly that I was trying to trick her into relying on me, and saying something open, only so I could then zap her with a humiliating comment, as her mother, father, brother, and Marni used to do. Jane consciously withheld associations for years, fearing the repetition of these attacks. Her mantra was that information was on a "need-to-know" basis, and there was a lot I did not need to know. The transference was conveyed with this military phrase, which reflects an environment in which enemies are everywhere and may kill at any time, making it essential, for one's safety, to compartmentalize information.

Jane has been a prolific dreamer. Within the hostile transference, dreams seemed a relatively safe method of association. On many occasions she would remind me, "It's only a dream," as she associated to difficult material.

Another central aspect of our relationship was the way that urges to use opiates were played out directly between us. Jane's last relapse had been initiated by her mother's having urged her to take one of her Percocets. Three days after her mother's death, Jane began talking about not letting her mother's fentanyl (a powerful synthetic opiate) transdermal patches go to waste. As we talked about this, it turned out that she had the patches in her purse, which lay next to the

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analytic couch (somehow she telegraphed this information, if I cared to notice). When I demanded the patches, she got up and handed them to me, and then resumed talking. Over the next several years, this enactment between us recurred several times. Jane was enacting the same pattern she had enacted with her mother, who wanted to watch her shoot up, or gave her a Percocet when she knew Jane would end up once again addicted. We repeated her feelings of closeness with her mother, but in this new relationship, she was kept safe by my maintaining a level of assertiveness that matched the danger of her intended action.

At this point, four years into analysis, Jane's struggle seems to be whether she can begin depending on people with loving dispositions. Jane is consistently doing well, both in her work and in her mothering of Ethan. However, she has not established any stable relationships in which she can be open with her feelings and receive something in return for the immense amount she has to give.

Informed Consent

"Jane" has read this paper, and freely agrees to having it published. All identifying data have been altered to preserve her anonymity. Consultation regarding the transference and countertransference impact of this intrusion into the analysis has been obtained.

METHOD

Drug Dreams

I reviewed the first forty-eight months of verbatim notes of Jane's analysis, along with notes taken after her hours during psychotherapy, a period in all of four and a half years without a relapse. This meets the DSM-IV definition of sustained remission: "none of the criteria for dependence or abuse have been met at any time during a period of 12 months or longer."

Dreams were rated "drug dreams" if, within the manifest content, at least one person was getting high, or there was drug seeking or buying. Multiple dreams presented in an hour were rated as a single dream.

Examples of Drug Dreams

Cocaine/Percocet (dream 13, hour 30). "I was doing drugs with Joey, shooting coke. I was having a hard time getting high. I couldn't

get a vein, or it would blow. Then he was my cousin Larry. I said, 'You got Percs right?' I'm thinking, 'If I use Percs, is it a relapse? While I'm shooting coke. I took the Percs.'

Alcohol (dream 15, hour 37). "My mother and I were in our summer house. There were three of us celebrating. My mother says, 'We'll have to drink to that.' She poured expensive champagne. I thought, 'I already did RediWhip. That will *really* be relapsing.' Maybe I could hide it. I didn't think I could say no to her. I said fuck it and drank the champagne. I thought, 'What does this mean? Should I take Percs?' I was thinking, 'Why is she giving me booze?' The third person is a he, balancing my mother's morals, caring." (Later associations made it clear that I was this person.)

Benzodiazepines (dream 93, hour 226). "A woman doctor gave me Valiums and thought it was okay, even though I was an addict. I was smiling. I *really* got over."

Heroin (dream 184, hour 480). "I did some dope. I got high and I was going to buy more. The dealer said, 'This is a package deal for five hundred dollars.' Effie and Sammy [two female cousins] were there. I had two hundred. I said, 'Give me three hundred and I'll pay you back with a credit card. I can go right to the cash machine.' They said no. There was a little man under my window spying on me. I was in Madeleine's house doing something that was not right." (Madeleine was her mother's alcoholic sister.)

Stimulation of multiple drives (dream 184, hour 494). "I was looking to buy dope. I couldn't find the number or something, to make contact with the person who had it. I was at Grant Street [location of a former apartment owned by her father]. They were tearing it down or fixing it. Some guy came up. He was part of fixing the building. He said, 'You want to have something to eat?' This was an invitation on a date. I said, 'Yeah, it seems like a good thing to do.' We were walking to a black car. At first it looked new, but it was a junk box when I got closer. There were people in it. It made me leery. The frustration was that I couldn't get what I wanted. I wanted to cop, and I couldn't get to the person."

I interpreted this dream as reflecting drives (for food, sex, heroin) active while Jane was asleep. Note her repeated use of the word *fixing* and the mention of *junk*. She responded that she had wanted to lose weight and had skipped dinner the evening before the dream. Dream 237 (hour 669) conveys the experience of intense thirst, buying a friend

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a shot and a beer and looking for Coca-Cola (associations, respectively, to shooting up and to coke, suggestive again of the common ventral tegmental pathway's activation).

With each drug dream, I looked at the hour in which it was reported to see if I could find a particular cause. Sometimes there were intense feelings toward me, or toward Jane's mother or other family members. But this was true of many hours in which drug dreams were not reported. After careful review of verbatim notes, I can offer no explanation for the dreams other than a wish to use drugs.

Finally, it is of interest that in the fifty-first month the dreams change, with the emergence of the new theme of using me to inhibit her drug use. This theme occurs in four of twenty dreams within a three-month period.

One of them is dream 232 (hour 658): "My mother looked sad. I walked away from her. I felt so sad. I said, 'Dope is the only thing that will take away the feeling. I know who has dope. I'll call.' There was a guy standing there. I had to call without him knowing. Someone interrupted the call. I heard his voice, 'I know what you are doing. You are *not* buying dope.' I thought, 'Shit! How did he hook into my phone line?' I figured I'd try again, find a way around him. I had to have it! But I didn't get the dope." These dreams are a further development of dream 184 above, where I am a witness but she is still able to use.

Table 3. Drug Dreams

#	Dream#	Hour#	# Months Sober	Drug
1	1	2x/wk tx	4	speedball
2	13	30	9	IV cocaine, Percocet
3	15	37	9	alcohol
4	16	41	9	alcohol, heroin
5	18	48	10	IV Demerol
6	23	58	11	alcohol, speed, heroin
7	26	64	11	alcohol
8	28	72	12	alcohol
9	30	75	12	heroin, amphetamine
10	31	78	12	speed, heroin
11	32	79	12	heroin
12	33	81	13	cocaine, heroin
13	34	82	13	cocaine
	(Mother dies)			
14	44	125	16	alcohol
15	49	140	17	Desoxyn (amphetamine)
16	55	156	18	heroin

Table 3 (Continued)

#	Dream#	Hour#	# Months Sober	Drug
17	59	169	19	snowballs (a drug in dream), Demerol
18	62	174	19	cocaine
19	65	177	19	heroin
20	71	186	20	heroin
21	82	209	21	alcohol
22	83	210	22	cocaine
23	85	215	22	heroin
24	88	218	22	heroin
25	91	223	22	heroin
26	93	226	23	diazepam
27	94	227	23	diazepam
28	104	246	24	speed, Numorphan
29	109	261	25	heroin
30	111	269	25	heroin
31	113	278	26	alcohol
32	121	293	28	cocaine
33	124	297	28	heroin
34	126	306	28	heroin
35	127	309	29	heroin
36	129	319	29	cocaine
37	132	327	30	Desoxyn
38	137	343	31	alcohol, heroin
39	138	344	32	"drugs"
40	146	365	32	IV drugs
41	147	369	32	speed, heroin
42	151	375	33	crack
43	152	376	33	methadone
44	161	406	35	beer
45	162	408	35	speed, heroin
46	166	415	36	Dilaudid, alprazolam, amphetamine
47	176	443	37	nicotine
48	178	463	40	heroin
49	184	480	41	heroin
50	188	494	41	heroin
51	192	509	42	heroin
52	195	523	43	heroin
53	206	576	47	heroin
54	217	608	49	cocaine
55	221	634	51	heroin, Dilaudid
56	232	658	52	heroin
57	237	669	54	alcohol
58	239	675	54	methadone

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Table 4. Summary of Drug Types in Manifest Content

opiates only	24
cocaine only	7
alcohol only	7
amphetamine only	2
benzodiazepine only	2
nicotine only	1
mixed drugs	13
unspecified drugs	2
Total Drug Dreams	58/240 (24%)

RESULTS

The drug dreams are listed in Table 3 and summarized in Table 4. Whether or not drugs were involved, one-third of all the patient's dreams had sexual themes in the manifest content, such as being asked on a date, having an affair with Tom Cruise, having sex with me, or my getting on the couch next to her while she is asleep.

DISCUSSION

A Neuropsychanalytic Hypothesis about Drug Dreams

The hypothesis of Berridge and Robinson (1993) is that the dopaminergic ventral tegmental pathway is irreversibly upregulated by drug exposure. This is congruent with the position of Alcoholics Anonymous that every sober alcoholic is but one day away from a drink or a drug, and that the disease continues to progress despite sobriety, ever ready to reemerge with its former strength. In my experience, only addicted individuals have drug dreams. This paper gives circumstantial evidence that suggests that their brains are changed by drug exposure. The persistence of these drug dreams shows that the urge to use drugs goes on for years, maybe forever.

Why would drug craving be expressed in dreams? This question has been answered recently by Solms (in press), whose work shows that the ventral tegmental area of the brain is the "dream-on" initiator of dreaming. Solms's evidence is that "dreaming and REM are dissociable states. . . . dream onset and offset can be manipulated by dopamine agonists and antagonists without any concomitant change in REM

frequency, duration and density. Dreaming can also be induced by focal forebrain stimulation and by complex partial (forebrain) seizures during non-REM sleep, when the involvement of brainstem REM mechanisms is precluded. Likewise, dreaming is obliterated by focal lesions along a specific (probably dopaminergic) forebrain pathway, and these lesions do not have any appreciable effects on REM frequency, duration and density. These findings suggest that the forebrain mechanism in question [the ventral tegmental pathway] is the final common path to dreaming.”

The mechanism of drug dreams would be as follows. Initially the ventral tegmental pathway provokes dreams that may show a wish for food, water, or sex. Dreaming then takes place in many other regions of the brain, which encompass other neuronal systems and other concerns. Exposure to addictive drugs upregulates dopaminergic neurotransmission in the ventral tegmentum, a process termed “reverse tolerance” by Berridge and Robinson (1993). This phenomenon includes increased drug effect on this pathway with further drug exposure. This may be why patients in detoxification programs constantly describe drug dreams; they are craving drugs because of upregulation of their ventral tegmental pathway, and they are dreaming intensely and frequently for the very same reason. That is, upregulation both stimulates increased dreaming and introduces a new drive, whose object is addictive drugs. Since the pathway also expresses the drive for food, water, and sex, these drives might be overrepresented relative to dreams of non-addicted patients. There is no way to compare the one-third incidence of sexually themed dreams in this study with what that incidence might have been without Jane’s drug exposure. But Berridge and Robinson (2000) use independent evidence to suggest that the sex drive in addicted patients is upregulated: increased mounting behavior in drug-addicted male rats, and the fact that 70 percent of patients admitted to cocaine addiction treatment programs suffer from compulsive sexuality.

Consequences for Psychoanalytic Theory

This dream study is relevant to the ongoing neuropsychanalytic discussion of exactly what constitutes a “drive” (Solms and Nesserian 1999; Panksepp 1999; Shevrin 1999; Yorke 1999). The study supports Shevrin’s assertion (1997) that drug addiction is a drive expression.

Freud’s view in “Instincts and Their Vicissitudes” (1915) was that an instinct constantly “impinges not from without but from within the

organism, no flight can avail against it. A better term for an instinctual stimulus is a 'need'. What does away with it is 'satisfaction'" (pp. 118–119). Further, "By the pressure of an instinct we understand its motor factor, the amount of force or the measure of the demand for work which it represents. The characteristic of exercising pressure is common to all instincts; it is in fact their very essence" (p. 122). Freud then differentiated ego or self-preservative instincts and sexual instincts, and explained, that "this supposition has not the status of a necessary postulate. . . . it is merely a working hypothesis, to be retained only so long as it proves useful, and it will make little difference to the results of our work of description and classification if it is replaced by another" (p. 124). He explained too that analysis of transference neuroses makes it possible to investigate the vicissitudes of the sexual drive, but not those of the ego. Any distinguishing of food, water, or drugs from sex, as representing a different category of drive, is undercut by the demonstration from neurobiology, and in this dream study, that all these intensely felt needs are subserved by the same neurological pathway.

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However, Freud's more fundamental concept of drive is strongly supported by the findings of this study, and the neurobiological events to be inferred contribute to the clinical expression of drug craving in dreams. The evidence of sexual interest expressed in the manifest content of one-third of these dreams, the 24 percent incidence of drug dreams, and direct expression of the wish for food and water within such drug dreams suggest that craving food, water, sex, and addictive drugs ought all to be regarded as drives.

Finally, Freud (1900) insisted that every dream expresses a wish, and that the dream borrows from the unconscious "the instinctual force which is at the disposal of the repressed wish" (p. 564). This view has been criticized by dream researchers, for example by Hartmann (1998), according to whom Freud's "mistakes were in fact related to his powerful drive to find a simple, satisfying answer to complex problems. He obviously desired, and felt he had achieved, a simple solution when he came up with 'every dream, when the work of analysis is complete, turns out to be the fulfillment of a wish'" (p.171; see also Fisher and Greenberg 1985).

Although this study cannot give evidence regarding *every* dream, it provides substantial evidence that the neurological origin of the mechanism of dreams is the ventral tegmental pathway, and that this

pathway is also the origin of “instinctual force” within the brain. In light of this understanding, it may be said that Freud in some sense anticipated the discovery of this pathway. He discusses this concept in “An Outline of Psycho-Analysis” (Freud 1940): “a dream is invariably an attempt to get rid of a disturbance of sleep by means of a wish-fulfilment, so that the dream is a guardian of sleep” (p. 171). He gives examples of a hunger dream, and of a dream prompted by sexual desire: “He has a dream of sexual intercourse” (p. 170); “A need for food makes itself felt in a dreamer during his sleep: he has a dream of a delicious meal and sleeps on” (p. 170).

As dreams are elaborated through other sections of the brain during dreaming, they will begin to contain other concerns, motivations, and feelings. Nonetheless, the origin of the dream would be exactly the ventral tegmental area of the brain expressing a “wish.”

In concluding the Outline, Freud says, “Experience has shown that the unconscious mechanisms which we have come to know from our study of the dream-work and which gave us the explanation of the formation of dreams also help us to understand the puzzling symptoms which attract our interest to neuroses and psychoses. A conformity of such a kind cannot fail to excite high hopes in us” (p. 171). The dreamwork discussed in this paper similarly helps us to understand the puzzling symptoms of addiction.

CONCLUSION

Drug addiction provides an important “experiment of nature” regarding drives and dreaming. Drug addiction presents observable phenomena that can reflect both the innate structure of the brain and the changes produced by drug exposure. Freud’s defining requirements for a drive—that it be a constant pressure, originating from within the organism, to do work, and that it constantly demand satisfaction—are completely satisfied by drug craving. Solms’s finding (in press) that the activating systems for dreaming and for craving are identical, a finding based on observations of tumor- or stroke-provoked brain lesions, is substantiated by the observation of dreams of a patient whose brain changes were created by a different type of neurological insult. Persico’s study (1992), as well as everyday clinical experience, indicates that drug dreams are common in cigaret addiction. Persico’s conclusion is that drugs dreams might have a protective effect against relapse to smoking.

Experiencing drug dreams and being able to discuss the unconscious pressure to relapse has helped keep the patient described in this report sober. Once the addictive neural mechanism is established, the only choices are to be conscious of craving, and to resist it, or to relapse to drug use.

The awful news for some individuals suffering from drug addiction is that drug exposure, often initially experienced during childhood with “gateway” drugs such as nicotine, in all likelihood causes an irreversible neurological change that sends out constant drive signals demanding satisfaction. Addiction to other drugs is more easily induced once the first addictive drug is craved, as the ventral tegmental pathway has been upregulated. Exposure to other addictive drugs then increases the drive for whichever drugs have already been experienced. This would be at least a partial explanation for the findings of Lai et al. (2000). In an epidemiological study with data from 17,809 respondents, those who had smoked cigarettes were 7.3 times more likely to use marijuana, 13.9 times more likely to smoke crack, and 16 times more likely to use heroin. Those who had used cigarettes before the age of fifteen were 80 times more likely to use illegal drugs.

This new evidence regarding the nature of addiction may have some impact on governmental regulation of addictive drugs, especially nicotine. We must now be aware that the cigarettes available to children at any corner store can produce an irreversible neurological change. Ventral tegmental upregulation through tobacco use might then fuel the huge drug hunger in our population, a hunger catered to by criminal cartels against which the government currently fights a losing war.

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