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To Paul, my treasure

28
How Do We Become Addicted? From Risk Factors to a Changed Brain*

> Thou hast the keys of Paradise, oh just, subtle, and mighty opium.
>
> Thomas De Quincey,
> *Confessions of an English Opium Eater,* 1821

> What can science teach us about addictive drugs and addictive behavior? That requires a thorough analysis, drug by drug, of how each one acts and what harm each one does to users and to society.
>
> Avram Goldstein,
> *Addiction from Biology to Drug Policy,* 2001, p 13

What is addiction? It's been defined in several ways. We saw in Part IV that definitions of mental illness are strongly influenced by culture; so too are definitions of addiction. Although the World Health Organization (1992) and the American Psychiatric Association (2000) prefer the term dependence, the word addiction is still widely used today. With respect to substances of abuse, addiction is typically defined as compulsive, persistent seeking and using a substance despite major adverse consequences. Addiction to nonconsumables (such as gambling or sexual partners) is similarly understood as compulsive and

*I'm indebted to Dr Steven E Hyman, provost, Harvard University, director of the National Institute of Mental Health 1996-2001, and formerly director of Harvard's Interdisciplinary Biobehavioral Program, for permission to include material from a memorable lecture to clinicians in 1994. In an era of amazing advances in the science of addiction, the principles he enunciated a decade ago remain valid today.

persistent seeking and behavior in the face of serious harm to self or others. That is, addiction connotes not only a subjective state of urgent wanting but also behavior, for the purpose of experiencing either intense pleasure or relief from an aversive state. Substances of abuse and addictive activities act as positive reinforcers (giving a "rush" or euphoria) or as negative reinforcers (offering escape from withdrawal symptoms or painful emotional states).

What images does the word addict bring to mind? Perhaps a street person, unwashed, usually in the inner city, living outside the law, and dark-skinned? The facts belie this stereotype. The vast majority of addicted Americans are Caucasian of all socioeconomic levels who are "heavy users" of caffeine, nicotine, alcohol, or sugar—all legal and all, arguably, addictive. Obesity/overweight, now epidemic in the United States, is often a result of one kind of addiction—to the pleasures of taste.

Another way of thinking about addiction is a *vulnerable individual's response* to taking addictive substances with adequate *dose* (enough of it); *frequency* (often enough); and *chronicity* (over a long enough period of time) to enter an addicted state (Hyman, 1995).

Vulnerability is a very complicated concept. The same individual can have different levels of vulnerability in different environments or life situations at different times in his or her life. For example, you may not be a vulnerable person most of the time, but when you lose your significant other or get fired from your job, you may become vulnerable to substance abuse or addiction.

How many of us can claim honestly that we're free of addiction? For example, I know I'm addicted to at least two substances—sugar (I can't conceive of a day without several sweets) and caffeine (which has sustained me through the writing of this book). How am I different from poor homeless people addicted to crack or heroin? Answer: I've had a luckier roll of the dice in life, and my addictions are socially acceptable in the dominant culture. My addictions, however, are similar to

those of people addicted to crack or heroin with respect to the neurobiological process by which I became addicted, the eager seeking and compulsive use of the substances, and strong cravings to the point of obsession when the substance is withdrawn, not to mention headache, fatigue, and a depressed mood. Are there serious negative consequences? Though not visible to others, fairly often my body communicates unpleasant sensations related to overconsumption, and there are some ominous warnings. These substances contribute to chronic gastritis and inflammation that are known to predispose to stomach cancer.

Concepts related to addiction are tolerance, dependence, and sensitization. *Tolerance* refers to the loss of effect of a drug after repeated administration over a long time on a frequent schedule, so that more and more is needed to produce the same high.

Dependence is defined as a state in which stopping a drug suddenly ("cold turkey") causes withdrawal sickness which is dramatically relieved by another dose of the same drug (Goldstein, 2001, pp 88-90). Where does the term "cold turkey" come from? The gooseflesh symptom of withdrawal from opiates gave rise to that expression!

Sensitization is the opposite of tolerance. The drug's effect is enhanced rather than diminished with repeated use, which sometimes happens with a few particular drugs when taken rapidly and repeatedly such as cocaine (Goldstein, 2001). Sensitization refers to persistent hypersensitivity to a drug's effect in a person with a history of exposure to that drug (Cami and Farré, 2003).

From risk factors for addiction to a changed brain. In Part I, we defined risk and protective factors. Risk factors are biological or nonbiological variables in individuals and in environments, interacting through time, to cause or exacerbate problems of health, mental health, and social conditions. Protective factors are the opposite of risk factors, acting to diminish problems in these areas. With respect to addiction, the more risk factors and the fewer protective factors we have, the more likely it is that

we'll get hooked. These risk factors fall into the categories of individual vulnerability, environmental factors, and drug effects, which differ according to the drug (see Table 6).

Table 6

Risk Factors for Addiction

Individual vulnerability (some aspects can change over time)
- genetic
- psychiatric condition
- chronic pain
- feeling stressed
- user goals (such as experimentation, escape).

Environmental factors
- drug availability—if you can't get it, you won't become addicted no matter how vulnerable you are.
- peer group pressure to use.
- lack of behavioral alternatives to drug use (no opportunities for fun or satisfaction).
- settings in which drugs are used such as religious ceremonies, family holidays.
- presence of conditioned cues (such as being at a place where you used to use frequently, or running into drug-using friends).

Drug effects (drugs differ)
- drug's addictiveness (some are highly addictive, such as cocaine; others are not very addictive, such as LSD).
- drug purity.
- route of administration (for example, you'll get addicted to cocaine faster if you freebase than if you snort.)
- dose.
- frequency of use.
- chronicity of use.

Chronic use of drugs causes long-lived molecular changes in the signaling properties of neurons. Depending on the drug and the circuits involved, these adaptations have different effects on behavior and different time-courses of initiation and decay.

With chronic drug use, three types of changes may take place in the brain centers that control *somatic functions* (body functions), *rewards* and *pleasures*, and *emotional memories*.

We can see physical effects of drugs that affect somatic functions when the drug is withdrawn. We can't see the physical effects of drugs on reward and pleasure pathways in the brain or on emotional memories, but these effects are physical too. They are just as real, and just as physical, as the somatic effects of withdrawal from alcohol (e.g. tremor, hypertension, grand mal seizures, tachycardia, irritability, delusions, hallucinations), caffeine (e.g. headache, fatigue), or opiates (e.g. severe muscle cramps, bone ache, diarrhea, tearing, hypothermia or hyperthermia, insomnia, restlessness, nausea, gooseflesh).

Only a few drugs involve somatic dependence, but almost all drugs of abuse are believed to induce the other two kinds of changes in brain structures and functions. With respect to brain reward and pleasure pathways, changes both in microanatomic structures and chemical processes involve motivation and volition. Motivational aspects of withdrawal are:
- dysphoria—feeling sad, blue, down in the dumps.
- anhedonia—can't experience pleasure. Things you used to enjoy aren't fun anymore.
- cravings—have to have it, fast.

The person suffering from these feelings experiences a change in behavioral priorities. Now, getting the drug of abuse often becomes the most important goal in life.

Changes in *emotional memories* are a hallmark of addiction. During a lifetime, many memories are eventually lost through decay of memory traces in the brain. However, memories of powerful experiences remain. Cues evoke these memories of either intensely pleasurable experiences, leading to cravings in addiction, or intensely painful experiences, leading to traumatic flooding, as in posttraumatic stress disorder (PTSD). These memories are referred to as "privileged" memories because they take precedence both in affecting the individual's emotional state and in motivating behavior.

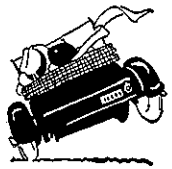
These latter two types of long-term changes—changes in structures and functions affecting motivation and volition, and changes in emotional memories—are actual physical effects in the brain that you cannot see. We used to distinguish between "physical" and "psychological" addiction by the presence or absence of somatic withdrawal, such as tremors, nausea, or muscle cramps. Now we know that *all drugs of abuse produce actual physical changes in the brain*. Even though many of the physical effects of drugs on the brain are not directly observable, they are just as real as tremors and muscle cramps.

How long do these brain changes last? Somatic withdrawal may last days, weeks, sometimes even longer. Motivational aspects of withdrawal may last from several weeks to months, even years. Emotional memories may last a lifetime—we may never shake them off. That is, once addicted, you may never "withdraw" from the memories of intense pleasure associated with drug use. That's why AA members with years of sobriety call themselves "recovering," not "recovered," alcoholics.

Each drug has its own special neurotransmitter. Drugs of abuse work in the brain through different neurotransmitter systems. Some drugs affect several transmitters through a chain of reactions. Here are some of the transmitters that are active with different drugs:

DRUG	NEUROTRANSMITTER
Opiates/Heroin	Endorphins/enkephalins
Cocaine/Amphetamine	Dopamine
Nicotine	Acetylcholine
Alcohol	GABA, opioids, and others
Marijuana (a cannabinoid)	THC receptor ligand anandamide
Hallucinogens	Serotonin
Caffeine	Adenosine

But.... most roads lead to the dopamine highway!



Hyman (1995) described the above process a decade ago, and research today supports this view (Hyman and Malenka, 2001). The complex neurobiological processes identified over the past decade that underlie these effects are reported in numerous sources (see, for example, Yang, Zheng, Wang et al, 2004; Thompson, Swant, Gosnell et al, 2004; Bolanos and Nestler, 2004; Wang, Gao, Zhang et al, 2003; NIDA, 2004). Wang and colleagues refer to the process described by Hyman as "abnormal engagement of long-term associative memory."

Current theories of addiction rely heavily on neurobiological evidence showing connections between addiction-related behaviors and neural structures and functions. These connections have been identified by imaging, biochemical analyses, genetic studies, and laboratory experiments.

It is widely agreed that all drugs of abuse act on dopamine systems either directly or indirectly. Determining the role of dopamine has been the predominant focus of addiction research during the past 20 years (Kalivas, 2004). Drug seeking and drug self-administering in humans and animals can be triggered either by direct exposure to drugs of abuse or by stressful events, both of which increase strength of excitatory synapses on mesolimbic dopamine neurons (Saal, Dong, Bonci et al, 2003). Moreover, it appears that other forms of addiction, such as compulsive gambling, also create feelings of pleasure, excitement, or satisfaction through dopamine pathways (Cami and Farré, 2003; Goudriaan, Oosterlaan, de Beurs et al, 2004; Ibanez, Blanco, de Castro et al, 2003).

The concept of *reward* is central to most views of addiction. Several brain circuits, structures, and neurotransmitters are involved in the reward process. Dopamine has had front-runner status in this regard for several decades, but other neurotransmitter systems (those that process serotonin, norepinephrine, opioids, GABA, and glutamate) also have important roles in the regulation of reward (see Chapters 10-12 for information about the major neurotransmitters). Among neuroscientists, it has been widely accepted for some time that dopamine mediates the

rewarding (reinforcing) properties of natural stimuli such as food and sex as well as drugs of abuse.

However, it appears that the relationship between dopamine and food or sex is more complex than was previously thought. Recent studies indicate that pleasure from food or sex ("hedonic response" in scientific terms) may continue in laboratory animals even when dopamine functions are suppressed. For some addictions, dopamine may promote effort to get and consume (food) or to perform an act (having sex) without mediating the reward process itself (another transmitter may be responsible for reward). Neuroscientist passions run high on both sides of the Great Dopamine Debate: Is dopamine the happy chemical? To find the answer, read the following authors, or if that's too much trouble, you could take my own opinion on faith. I vote for "often, yes, always, no!" (Cannon and Bseikri, 2004; Salamone, Correa, Mingote et al, 2003; Berridge and Robinson, 2003; Wise, 2004; Hajnal, Smith, and Norgren, 2004; Giuliano and Allard, 2001; Paredes and Ågmo, 2004).

Cannon and Bseikri (2004), despite their own research showing that dopamine is not *necessary* for food pleasure in laboratory animals, conclude that there's reason to think it's *important*. In a recent review article on drug addiction, Cami and Farré state unequivocally, "Both natural rewards (food, drink, and sex) and addictive drugs stimulate the release of dopamine" (2003, p 980). Studies have found the transmitter to be involved in drugs of abuse, gambling (Goudriaan et al, 2004), food (Wise, 2004; Hajnal et al, 2004), sex (Giuliano and Allard, 2001), pair-bond formation (Young, Lim, Gingrich et al, 2001), listening to music (Sutoo and Akiyama, 2004), seeing attractive faces (Kampe, Frith, Dolan et al, 2001), video games (Koepp, Gunn, Lawrence et al, 1998), positive social interactions (Vandenschuren, Niesink, Van et al, 1997; Hansen, Bergvall and Nyiredi, 1993), and best of all, humor (Mobbs, Greicius, Abdel-Azim et al, 2003).

Yes, humor is fun and feels good—is it surprising that Mobbs and colleagues, using fMRI (functional magnetic resonance imaging), found that humor activates the dopamine-pro-

cessing pleasure pathway in the brain? In answer to the question "What is the role of dopamine in reward?" Cannon and Bseikri (2004), citing Mobbs et al, quip that scientific progress would be greatly expedited if we were all simply funnier. I agree! Next assignment: Tell your boss that you were late to work because of a herniated hippocampus.

Other views of addiction expand existing theories of reward by separating its psychological components into *learning* through the experience of using, *liking* that experience (pleasure that diminishes over time), and *wanting* to repeat it (Berridge and Robinson, 2003). These researchers believe that although liking decreases over time, wanting increases, continuing to be a powerful motivator even when using is no longer enjoyable because the brain has become *hypersensitized* to effects of the drug in the context of impaired cognitions that lead to compulsive seeking and using.

These cognitive effects have been connected to events in the frontal cortex. Goldstein and Volkow (2002) have expanded the focus on limbic subcortical structures to include structures in the frontal cortex using findings from neuroimaging studies. They found that the orbitofrontal cortex and the anterior cingulate, regions neuroanatomically connected with limbic structures, are the frontal cortical areas most frequently implicated in drug addiction. These structures are activated in addicted subjects during intoxication, craving, and bingeing; deactivated during withdrawal; and also involved in higher-order cognitive and motivational functions. That is, addiction connotes cognitive and emotional processes, regulated by the frontal cortex, which result in overvaluing drug reinforcers, decreasing sensitivity to alternative reinforcers (that is, perceiving them as less desirable), and deficient inhibitory control for drug responses (Volkow, Fowler, and Wang, 2002). These changes in addiction, called *salience attribution* and *impaired response inhibition*, expand traditional concepts of drug dependence that emphasize limbic system responses to pleasure and reward.

A related view cites evidence that compulsive drug use and its persistence arise from pathological usurpation of molecular

mechanisms involved in memory (Hyman and Malenka, 2001), a process we spelled out earlier. This process characterizes the takeover of the addicted person's life so familiar to users, their families, and those of us who try to help them grapple with addiction.

Another theory proposes that specific brain reward and stress circuits become dysregulated during the development of alcohol dependence (Koob, 2003). Parts of the amygdala and the nucleus accumbens (a grouping called the "extended amygdala") mediate multiple neurotransmitter systems that process GABA, opioid peptides, glutamate, serotonin, and dopamine. Withdrawal from drugs of abuse is associated with subjective negative affect, accompanied by action of stress hormones (see Chapter 19). This "toxic" effect of chronic drug use creates an ongoing state of vulnerability for relapse.

There is increasing evidence that the excitatory transmitter glutamate also plays a central role in processes underlying the development and maintenance of addiction (Kalivas, 2004; Tzschentke and Schmidt, 2003). Glutamate dispatched from the prefrontal cortex to the nucleus accumbens appears to promote reinstatement of drug-seeking behavior. That is, glutamate may be a major contributor to relapse. In particular, context-specific aspects of behavior (control over behavior by conditioned stimuli) seem to depend heavily on transmission of glutamate.

The influence of genetics has emerged in conjunction with the publication of the Human Genome study in 2003. Roles of genes in addiction are elusive because substance abuse is a product of polygenetic action and is strongly influenced by environments through epigenetic systems in the brain (see Chapter 7). Data have been available for many years showing much higher risks for alcohol addiction in biological children of substance abusing parents adopted at birth into non-substance abusing homes, than for children without a family history of addiction (Schuckit, 1985). Recently, specific genes and variants of genes (alleles) have been identified that either protect individuals from addiction by, for example, causing aversive effects from ingesting certain substances, or put individuals at

risk for various types of substance abuse (for a review of this literature, see Cami and Farré, 2003; Comings and Blum, 2000).

As Cami and Farré (2003) have pointed out, these various theories overlap in some ways and are not mutually exclusive. None alone can explain all facets of addiction. In Parts I-IV we've considered the mind-boggling intricacy of the brain, the most complex entity in the known universe. Substance abuse and addiction happen through interaction between that entity and the world around it, so it's not surprising that these phenomena are extremely complex as well. Little by little, we're piecing together various aspects that give us guidance to work with people.

Popular psychology has expanded the usage of the word "addiction" to include "workaholics" (who work too long and too hard), women who "love too much" (have a pattern of attachments to hurtful partners), and "codependents" (significant others of addicted persons who supposedly have a need to have an addicted partner) (Whitfield, 1991). These groups are thought to have addictive characteristics because they have seemingly overpowering cravings that they compulsively and repetitively seek to satisfy, despite very adverse consequences.

If the underlying neurobiology of attachment to compulsive work or hurtful partners is found to resemble the process that has been identified for addictive drugs and gambling, that interpretation might be justified. However, to the best of my knowledge no such evidence has been uncovered. There are possible alternative explanations for these behavior patterns, for example, anxiety about not making enough money to meet expenses (leading to compulsive working), or an attachment to an addicted partner because of that person's other qualities *despite* the addiction, not *because of* it. In this case, the therapeutic issue is not to determine the partner's "codependency"—connoting that the partner of the addicted person has a sickness equivalent to that of the addicted person—but rather to weigh costs and benefits of the relationship as it is, and to consider strategies for bringing about change.

A body of published critiques of the codependency concept has noted its pejorative connotations, characterizing interpersonal behaviors as addictions or diseases, pathologizing women, promulgating a value-laden Anglo cultural narrative, and requiring partners of addicted persons to assume responsibility for their partner's addiction (Montgomery, 2001; Anderson, 1994; Collins, 1993; Inclan and Hernandez, 1992). Although recent validation studies of instruments measuring problems and issues of family members have shown reliability and validity with respect to characteristics on the inventories (Dear, 2004), the designation "codependent" remains offensive. Studies that simply gather data about the kinds of challenges experienced by families with a member experiencing addiction, in the context of a stress and coping conceptual framework, can supply information that may help practitioners support families (Hurcom, Copelle, and Orford, 2000). Unlike the codependency frame, a stress and coping model normalizes individuals and their behaviors.

The issue of *enabling* (significant others doing things that inadvertently support rather than discourage the addicted person's habit) is separate from the concept of codependency. Enabling does not assume pathology in significant others and is an important target for educational and therapeutic efforts (Rotunda, West, and O'Farrell, 2004).

Brain structures and systems involved in substance abuse and addiction. Brain structures referred to here are described in Chapters 10 and 11. Two systems, the *mesolimbic dopamine pathway*, also called the pleasure pathway, and the *mesocortical dopamine circuit*, originate in neurons in the *ventral tegmental area* (VTA), where neurons manufacture dopamine (see figures 16 and 17 pp 268-269). All drugs of abuse act on these systems at different levels. The two systems act in parallel and also interact with each other to mediate the addiction process.

The mesolimbic dopamine pathway is probably the most important circuit involved in reward. In this system, a bundle of

nerve fibers (axons) project from the VTA to the *nucleus accumbens* (NAc) in the limbic system bordering on the basal ganglia. Messages are relayed through the *amygdala* as part of this system. Another limbic structure involved in addiction is the *hippocampus*, which is involved in short-term memory formation. Opioid pathways also participate in reward.

The mesolimbic dopamine pathway has a role in creating privileged memories of highly rewarding novel stimuli. These memories cause addicts to have cravings and risk relapse even after years of abstinence. The limbic system plays a key role in determining what is salient enough to be remembered.

The mesocortical dopamine system projects from the VTA to the prefrontal cortex, the anterior cingulate (mediates response inhibition and initiation), and the orbitofrontal cortex (mediates ability to evaluate future consequences and balance immediate rewards against long-term negative consequences). These structures are involved in the conscious experience of drug-taking, cravings, and compulsions (Cavedini, Riboldi, Keller et al, 2002).

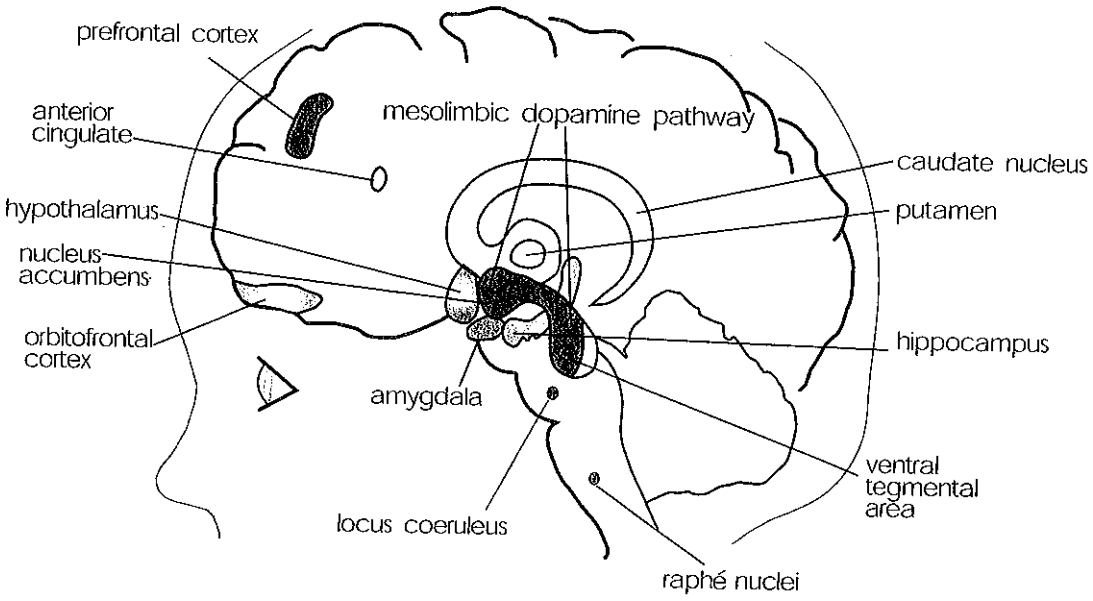
Basal ganglia structures, the *caudate nucleus* and the *putamen* (together called the *striatum*) are also involved. The *hypothalamus,* located near the limbic region and the basal ganglia, is activated when stress is contributing to addictive responses. The hypothalamus controls many hormones, including those that help the individual cope with stress, such as cortisol (see Chapter 19).

The *prefrontal cortex*, critical for higher cognitive functions such as executive planning, working memory, hypothesis generation, response inhibition, action initiation, and problem solving, is also importantly involved in substance abuse and addiction. Some cognitive dysfunctions seen fairly often in heavy users such as poor appraisal of likely consequences of behavior, overvaluation of drug effects, difficulty making decisions, and poor response inhibition, suggest interactions between mesolimbic structures and prefrontal and other cortical structures that mediate cognitive functions.

Figure 17 (p 269) shows some of the routes for neural mes-

Hot Spots For Highs
Structures

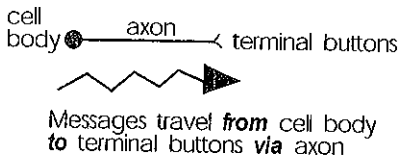
figure 16



Hot Spots For Highs
Pathways

figure 17

= mesocortical dopamine circuit
GLU = glutamate
DA = dopamine
5-HT = serotonin
NE = norepinephrine
GABA = gamma-aminobutyric acid
OP = opioid



Messages travel ***from*** cell body ***to*** terminal buttons ***via*** axon

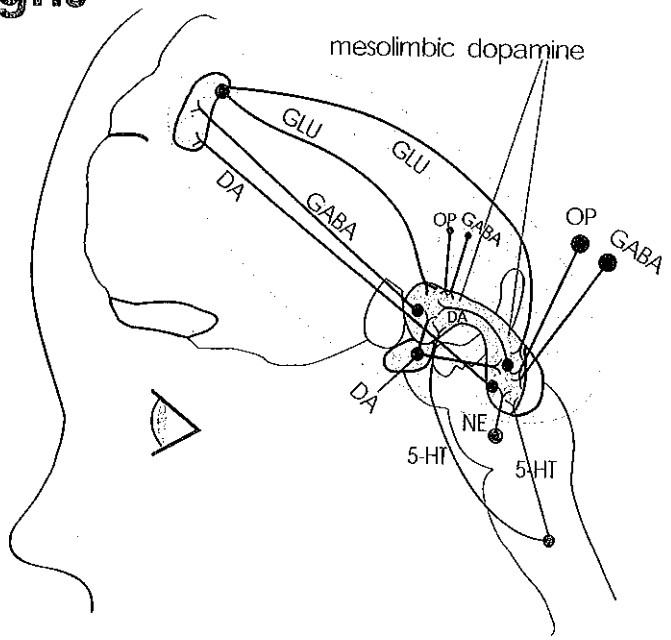


figure 18

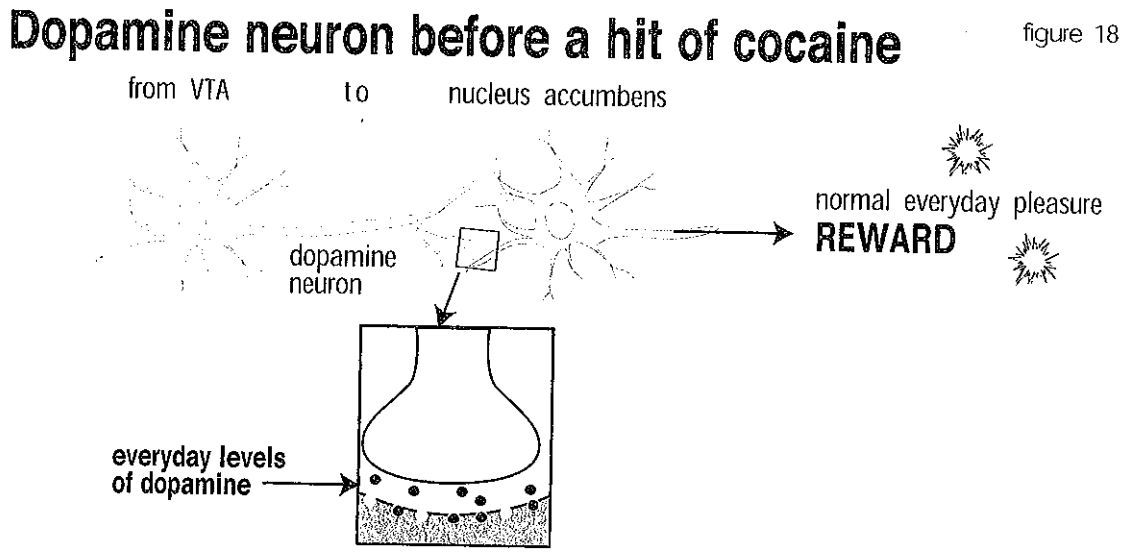


figure 19

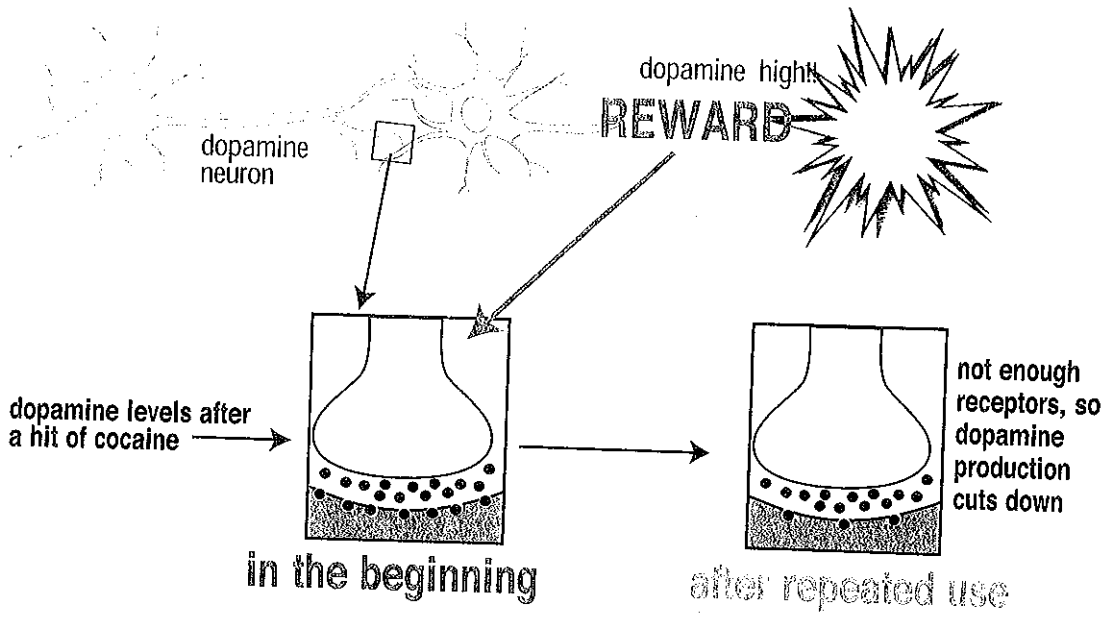


figure 20

Inhibitory interneuron (GABA) keeps the dopamine neuron in check

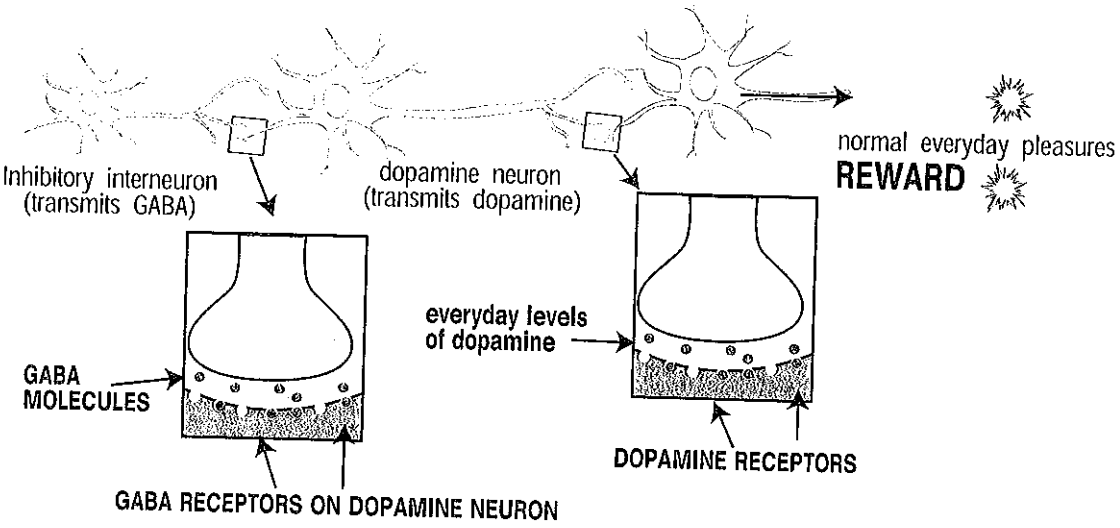
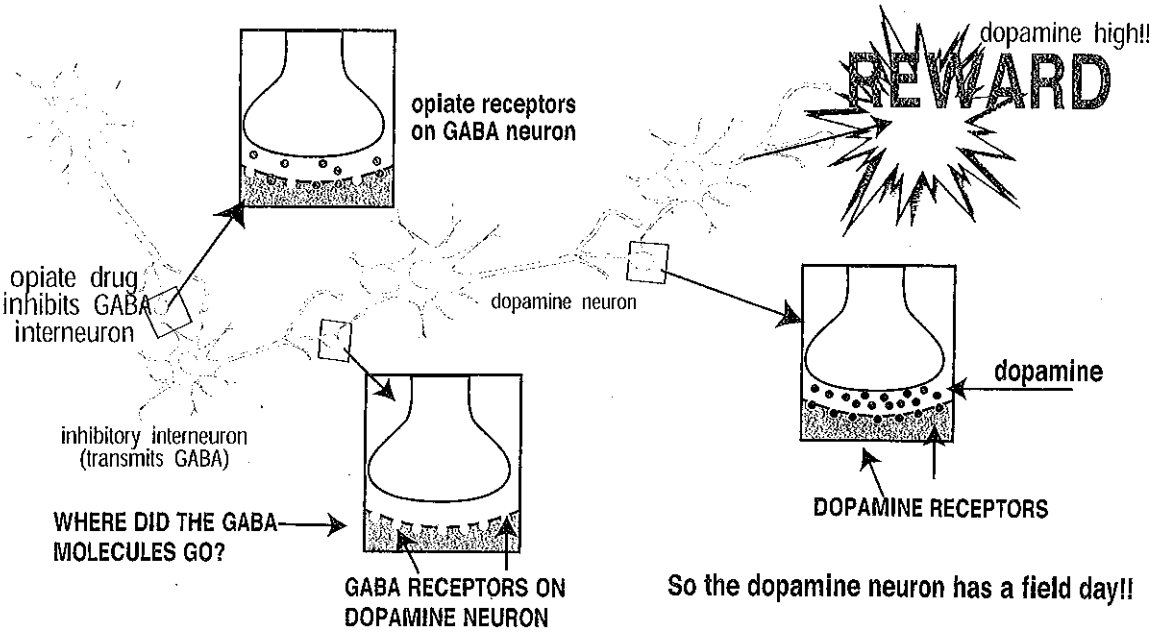


figure 21

Opiate inhibits the inhibitory interneuron



sages related to reward and cognition..

Let's try to follow these arrows to help us visualize these routes. Dopamine (DA) neurons originate in the VTA and project their axons to the nucleus accumbens, the amygdala, and the prefrontal cortex. Neurons processing glutamate (GLU), the major excitatory transmitter in the brain, send messages from the prefrontal cortex to the VTA and the nucleus accumbens. Neurons processing GABA, the major inhibitory transmitter in the brain, send messages from the nucleus accumbens to the prefrontal cortex and also act on the VTA and the nucleus accumbens. Opioid-processing neurons (OP on the diagram) modulate GABA's inhibitory influence on the release of dopamine in the VTA and also affect the release of norepinephrine (NE) from the locus coeruleus. Serotonin neurons (5-HT) originate in the raphé nuclei and project to the VTA, the nucleus accumbens, and the striatum, where they modulate release of dopamine. These processes are described by Cami and Farré (2003).

These networks are very complicated, and it's not necessary to "master" this information in order to use it to help your clients. You do need a basic understanding of the ways some rewarding and cognitive events take place in relation to specific classes of drugs such as cocaine, opiates, and nicotine, and the ways using drugs or engaging in addictive behaviors leads us to become addicted. We'll explain that later in this chapter.

The prominent role of neurobiology in serious medical, psychological, and social problems related to addiction has led to efforts to identify and use pharmacological agents (i.e. more drugs) to treat drug addiction. Later in this chapter we'll consider the pros and cons of this direction in addiction treatment. First, we'll briefly review the kinds of medications in use.

There are at least three types of drugs for treating addictions: agonists, antagonists, and aversive agents. *Agonists* bind with receptor molecules in a similar fashion to the drug of abuse, prevent withdrawal symptoms, but do not give the high of the drug of abuse. For example, methadone binds to opiate receptors in place of heroin, prevents heroin withdrawal, and

takes away the craving for heroin. Nevertheless, the street marketability of some treatment drugs such as methadone suggests that they do give some kind of a high. Another problem with agonists is that they are also likely to be addictive, so the user substitutes one addiction for another. The advantages are that the person may be able to function (e.g., hold a job) better than when using the original drug, and that the prevention of withdrawal symptoms and cravings diminishes the need for criminal behavior to get money for the drug of abuse.

Antagonists bind with receptors in a different way from the drug of abuse (competitive binding at the receptor site), so taking the drug of abuse gives no high because the antagonist drug now occupies the receptors. The craving, however, is not satisfied. For example, naltrexone binds to opiate receptors, blocks the effects of heroin, but does not remove the craving for heroin and other opiates. Since the antagonist therapeutic drug now blocks the effect of the original drug, the user may just substitute a different drug of abuse for the original drug. Researchers are developing and testing new drugs that combine agonist and antagonist actions in the hope of remedying the limitations of each type of medication.

Aversive agents, such as disulfiram (Antabuse) for alcohol addiction, deter drug use by making you very sick if you use the drug while taking the medication.

29
Some Street Drugs--How They Can Get You Hooked

We'll look briefly at the actions of some classes of drugs. Let's start with *stimulants.*

Cocaine, amphetamine, and other stimulants produce euphoria and increase arousal, alertness, concentration, and