

Is Drug Addiction a Disease?

By

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The urge to use drugs seems to go beyond the concept of social pressures into the domain of instinctual, almost primal behavior. When animals are given unlimited access to drugs, they will become addicted. This occurs, for example, even when food is readily available.

Some researchers see addicts as medicating themselves to counteract for some unspecified biochemical shortage. This conclusion has been reached because 3- to 7-percent of addicts can "just stop" without any treatment. This suggests there may be an underlying metabolic deficiency that has corrected itself. Some researchers see the "disease" as inherited because 10-percent of the population carries genes that *predispose* them to addictive behaviors.

Genetic evidence for drug addiction as a disease is slim. Studies indicate that the genetic factor to alcoholism in purebred strains of mice show varying sensitivity to opiates. The opiates are not alcohol and an increased sensitivity doesn't mean addiction. There is no direct evidence to show that opiate addiction is a disease--metabolic, genetic or otherwise. With addiction defined as being a disease this takes the responsibility for the individual's behavior *from* the addict.

The concept of drug addiction being a disease is a medical model of addiction. Prior to the medical model the "moral model" was the interpretation of addictive behavior. This earlier model said that an alcoholic or addict was somehow corrupt and depraved, essentially a sinner that needed some sort of "moral intervention."

The idea that specifically alcoholism is a disease stems back to the writings of Benjamin Rush, the Surgeon General of the American Revolutionary Army when he wrote in his paper, *An Inquiry Into the Effects of Ardent Spirits Upon the Human Body and Mind*, "This odious disease (for by that name it should be called) appears with more or less of the following symptoms, and most commonly in the order in which I shall enumerate them." He then proceeds to list 11 behaviors common to individuals *after* being inebriated. There are *no* symptoms that will indicate a person will engage in alcohol seeking behaviors. Thomas Trotter in 1804 stated, "In medical language, I consider drunkenness, strictly speaking, to be a disease ..." To him, it was a "temporary madness ... where there is a predisposition to insanity and idiotism..." In essence, he was saying that there is a mental aberration that produces alcoholism. This definition has become increasingly popular even though the evidence is quite thin. Then, in 1946 Emil Jellinek, based on a questionnaire given to individuals in Alcoholics Anonymous published the concept of alcoholism being a disease that goes through phases. (Levin, J.D., and Weiss, R.H. 1994) In the writings of Alcoholics Anonymous alcoholism was written as being "...like a disease..." and not a disease per se. Leon Wurmser in his article, *Psychodynamics in Compulsive Drug Use* admitted that the disease model has problems. But it is "humane" to treat the addict and not condemn him for condemnation leads to dehumanization. (Levin, J.D., and Weiss,

R.H. 1994)

The extensive use of the medical model came from the 1920s and 1930s when alcoholics and addicts were sent to hospitals to "dry out." The problem with addiction being considered a disease is the definition of disease. In order for a problem to be considered a disease, it must first have specific *symptoms*. That is, it must have certain indications that it is going to occur. For example, bodily aches, tiredness, nausea, and fever are the general symptoms of the flu. Second, a disease must have a specific *progression*. This means that a disease must have a particular origin, disposition and course of action. Addiction has neither of these characteristics. There are no behavioral indicators that a person will engage in alcohol seeking behaviors. Consequently, attributing addictive behavior to the disease process is incorrect. One popular definition used to claim the disease model is: "...it has an offending agent, it has specific criteria for diagnosis, it has a defined course, and it is reproducible in (laboratory, my wording) animals." (Senay, E.C. 1998) With this definition, pregnancy and emotional overreactions can be defined as a disease. As you can see, what is missing in this definition is the idea of addiction having any specific predetermining defining symptoms.

Heroin addiction alters the beta-endorphin system. Narcotics don't seem to change the number of endorphin receptors and endorphins are difficult to measure. Measuring endorphins refers to the endorphins which have jumped the blood/brain barrier from the hypothalamus into the circulatory system. The level of the endorphins doesn't reflect what's going on in the brain but actually the *stress* level of the individual.

The locus ceruleus functions abnormally after there has been continued

substance abuse. When an individual first begins to take opiates, this region shuts down. After continued use it returns to the normal firing rate. In detoxification (detox) a reaction occurs and the locus ceruleus becomes *hyperactive*. Since the locus ceruleus is involved in vigilance and most likely in the alarm-fear reaction, this increased sensitivity could explain the extreme anxiety and turmoil which is part of the withdrawal.

Some of the answers to addiction as a disease are found not in the brain but in the mind. The most striking observation is that, despite the apparent misery of addicts, there are no significant physical changes in the brain. Tolerance and withdrawal don't proceed according to any set formulas, as would be expected of purely physical reactions.

Though the disease concept may still hold, the individual's psychology is extremely important. An individual's predrug personality is a major indicator as to how the drug will be tolerated and the reactions occurring under the drug's influence. The conditioning the individual has undergone and the act of drug taking may be as meaningful in the formation of tolerance and withdrawal as the drug itself. (Rosenthal 1983)

The growing effects of drug addiction include: increased criminal behavior; direct effects of drugs on one's health; and the secondary costs. There are actually no effective preventive approaches at this point in time. Much of the attention and resources in the drug state is concentrated on the treatment *after* addiction. In order to produce the obsessive drug-seeking and taking behavior, an addictive substance must act on the cells and molecules of the nervous system. The sites and the mechanisms that take part in these effects have not been well determined, and

the basis for specific control in addictive accountability is unknown.

It has been theorized that drugs can invoke their peculiar patterns of behavior on a normal biological substrate and that no preexisting psychopathology or addictive sensitivity is required for induction or drug self-administration. This does not inhibit the well-recognized individual classes in addictive proneness. It does, though, raise questions of:

- 1) How are the reinforcing effects generated?
- 2) Which brain systems participate? and
- 3) What are the normal functions of these systems?

Many models of addiction are based on the intimation that physical dependence and tolerance generally develop and disintegrate along a similar time cycle. This leads to the concept that the adaptive processes are instigated to oppose the effects of the drug, and these processes persist after the drug has been cleared from the brain, leaving the opposing processes unchecked. Both behavior and cellular data indicate that tolerance and dependence are separate processes with distinct sites in the brain and with characteristic molecular mechanisms of action.

Any individual modulations in addictive sensitivity have also been ascribed theoretically to an assortment of social and biological factors. In the laboratory, animals have been bred with discriminating sensitivity to alcohol and opiates and for selectivity in drug self-distribution. Certain patterns of alcohol dependence have strong family heredity patterns which are free of social and environmental effects. The activities of specific neurons have not been delineated because the nature of the "counteradaptive" or "opposing"

mechanisms has not been described.

Dopamine-containing neurons and their terminal regions are necessary for the primary reinforcing effects of psychostimulants. With the opiates no links were found to any known transmitter or neural location before the endogenous brain opioid systems were discovered. Mapping neuronal circuits containing dopamine or the endogenous opioid peptides provided defined templates for sites at which the neuronal mechanisms of cocaine or opiate addiction could be categorized. No similar template has yet been conceived for the actions of alcohol.

Across all levels of inquiry, molecular and cellular mechanisms of the nervous system react to addictive drugs to begin and sustain patterns of drug-seeking behavior. These patterns arise primarily because the drugs are able to commandeer the decisive reinforcement systems and the small, finite number of transmitters and response sites which operate normally to shape the survival of the organism.

The same neurobiological circuits implicated in the severe hedonic or "positive reinforcing" actions of drugs may become modified through chronic use as the self-corrective homeostatic responses of the brain adapting to the drug's actions. The opposing process may neutralize the reinforcing effects and, on withdrawal, produce the antagonistic stimulus effects of the abstinence syndrome. These "negative reinforcing" effects (i.e. malaise, dysphoria, and anhedonia) are a major etiological and motivational factor in sustaining drug dependence.

Opioids and psychostimulants have specific endogenous ligands on which they act at specific places to produce discrete patterns of each behavior apropos to dependence. Alcohol appears to act at many sites in the brain, each with its own dose

threshold, to produce anxiety reduction, euphoria, motor incoordination, and cognitive depression. (Koob and Bloom 1988)

The statistics for drug abuse seem to be plausible inferences of survey results, but they may be invalid in some cases and in others show the proper use of raw data. The statistics, it appears, are usually "soft." The problem related to drug abuse is complex and difficult to quantify. In some instances the data are not statistically sound nor can they be inferred beyond the limits of the survey. We can also have a problem with the survey questions. They may not include questions concerning addiction per se, but focus on the problems which are related to ingestion. (Barnes 1988a)

Researchers have described two types of alcohol dependency. *Type I* alcohol dependency is characterized by a passive-dependent or anxious personality. In this person there is a high reward dependence, high harm avoidance, and low novelty seeking behavior. High reward dependence means that this person is eager to help, emotionally dependent, warmly sympathetic, sentimental, sensitive to social cues, and persistent. High harm avoidance means that this person is cautious, apprehensive, pessimistic, inhibited, shy, and susceptible to fatigue. The low novelty seeking trait means that this individual tends to be rigid, reflective, loyal, orderly, and attentive to details. The abstaining Type I is over-alert and anxious with a lot of expectational worrying. Women tend to develop into Type I dependency with a later onset and more rapid development of complications which are associated with guilt, depression, and medical difficulties from sustained high blood-alcohol levels.

Type II alcohol dependence is characterized by antisocial personality traits. They are high novelty seeking, low harm

avoidance, and low reward dependence. High novelty seeking means this person is impulsive, exploratory, excitable, disorderly, and distractable. Low harm avoidance means a tendency to be confident, relaxed, optimistic, uninhibited, carefree, and energetic. Low reward dependence makes this person is tough-minded, socially detached, emotionally aloof, practical, and independently self-willed. The abstaining Type II has lowered alertness, is distractible, impulsive, and easily bored.

Alcohol dependency has variable predispositional patterns to seek out alcohol and become tolerant and dependent upon it. There are various combinations of personality traits which reflect the differences in the brain systems which determine one's abilities to seek behavioral reinforcement from alcohol and become tolerant and dependent. (Cloninger 1987)

The determination gauge for psychoactive substance dependence contains at least three of the following:

1. The substance is often taken in larger amounts or over a longer period of time than has been expected by the person.
2. There is a recurring craving or one or more unsuccessful attempts to cut down or control the substance being used.
3. There is a great deal of time spent in activities necessary to get the substance (e.g. theft), taking the substance (e.g. chain smoking), or recovering from the effects.
4. There is recurrent intoxication or withdrawal symptoms when one is expected to fulfill one's major role responsibilities at work, school, or at home (e.g. doesn't go to work due to hangover, or goes to work "high," or is intoxicated while taking care of

- the children), or when substance use is physically dangerous (e.g. DUI).
5. Important social, occupational, or recreational activities have been given up or reduced due to substance use.
 6. There is persistent substance use despite the knowledge of having frequent or recurrent social, psychological, or physical problems that are caused or heightened by the use of the substance (e.g. keeps on using the drug despite family arguments about it, cocaine-induced depression, or having an ulcer made worse by drinking).
 7. There is marked tolerance. This means that there is a need for distinctly larger amounts of the substance to reach the same level of intoxication (e.g. at least a 50-percent increase) or the desired effect, or noticeably diminished effect with continued use of the same amount.
 8. The substance is often taken to ease or evade withdrawal symptoms.

Some of the features of the distress must have continued for at least one month, or have occurred repeatedly over a longer period of time in order to demonstrate dependence. (Barnes 1988b)

If drug or alcohol addiction isn't a disease what could be the reason for considering it a disease?

The main reason for the constant reference to and labeling drug and alcohol addiction as a disease is funding. The federal government funds many drug addiction centers both public and faith-based.* By classifying drug addiction as a "disease" and in the same category as any other disease funding is freed up. If addiction was

classified as a "choice" funding would be impossible.

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* Note: In recent research it has been found that faith-based programs do not work.