

# Substance Use Disorder

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Substance use disorders include substance abuse and substance dependence. In DSM-IV, the conditions are formally diagnosed as one or the other, but it has been proposed that DSM-5 combine the two into a single condition called "Substance-use disorder".

### **Terminology and usage**

Although the term substance can refer to any physical matter, "substance abuse" has come to refer to the overindulgence in and dependence of a drug or other chemical leading to effects that are detrimental to the individual's physical and mental health, or the welfare of others.

The disorder is characterized by a pattern of continued pathological use of a medication, non-medically indicated drug or toxin, which results in repeated adverse social consequences related to drug use, such as failure to meet work, family, or school obligations, interpersonal conflicts, or legal problems. There are on-going debates as to the exact distinctions between substance abuse and substance dependence, but current practice standard distinguishes between the two by defining substance dependence in terms of physiological and behavioral symptoms of substance use, and substance abuse in terms of the social consequences of substance use.

Substance abuse may lead to addiction or substance dependence. Medically, physiologic dependence requires the development of tolerance leading to withdrawal symptoms. Both abuse and dependence are distinct from addiction which involves a compulsion to continue using the substance despite the negative consequences, and may or may not involve chemical dependency. Dependence almost always implies abuse, but abuse frequently occurs without dependence, particularly when an individual first begins to abuse a substance. Dependence involves physiological processes while substance abuse reflects a complex interaction between the individual, the abused substance and society.

Substance abuse is sometimes used as a synonym for drug abuse, drug addiction, and chemical dependency, but actually refers to the use of substances in a manner outside sociocultural conventions. All use of controlled drugs and all use of other drugs in a manner not dictated by convention (e.g. according to physician's orders or societal norms) is abuse according to this definition, however there is no universally accepted definition of substance abuse.

The physical harm for twenty drugs was compared in an article in the Lancet (see diagram, above right). Physical harm was assigned a value from 0 to 3 for acute harm, chronic harm and intravenous harm. Shown is the mean physical harm. Not shown, but also evaluated, was the social harm.

Substance use may be better understood as occurring on a spectrum from beneficial to problematic use. This conceptualization moves away from the ill-defined binary antonyms of "use" vs. "abuse" (see diagram, lower right) towards a more nuanced, public health-based understanding

of substance use.

## **Mediators & Moderators**

When a predictor variable and an outcome variable have a significant relationship, which is, in turn, influenced by a third variable, the relationship is said to be mediated by the third variable. In this relationship the predictor variable influences the mediating variable in a causal manner. This mediating variable then leads to the outcome, creating the relationship between the predictor and outcome. It is only because of this mediating variable that a relationship between the predictor and outcome exists. Also, quasi-causal inferences may be drawn from mediated relationships.

### **Mediator Model**

As demonstrated by the chart below, numerous studies have examined factors which mediate substance abuse or dependence. In these examples, the predictor variables lead to the mediator which in turn leads to the outcome, which is always substance abuse or dependence. For example, research has found that being raised in a single-parent home can lead to increased exposure to stress and that increased exposure to stress, not being raised in a single-parent home, leads to substance abuse or dependence. The following are some, but by no means all, of the possible mediators of substance abuse.

When a variable indicates the conditions under which a specific effect occurs as well as displays how the direction or strength varies within a given relationship, the variable is said to moderate the relationship. Another explanation is that a moderator variable indicates that an effect only occurs under specific conditions. Unlike a relationship containing a mediator variable, the impact of the predictor variable on the outcome is dependent on the value of the moderating variable. Also unlike a relationship involving mediation, no causal inferences can be drawn from a moderated relationship; relationships can only be described as correlated. However, moderated relationships do identify interaction effects between predictor and moderator variables. Moderation is best illustrated by the following model:

### **Moderator Model**

As demonstrated by the chart below, numerous studies have examined factors which moderate substance abuse or dependence. In these examples, the moderator variable impacts the level to which the strength of the relationship varies between a given predictor variable and the outcome of substance abuse or dependence. For example, there is a significant relationship between psychobehavioral risk factors, such as tolerance of deviance, rebelliousness, achievement, perceived drug risk, familism, family church attendance and other factors, and substance abuse

and dependence. That relationship is moderated by familism which means that the strength of the relationship is increased or decreased based on the level of familism present in a given individual.

Mediation and moderation research continues to inform the field's knowledge and understanding of a pervasive and dangerous threat to public health, substance abuse and dependence. As the relationships between various predictor variables and the factors which influence them are more closely scrutinized, clinicians and researchers are provided with the necessary information to create more sophisticated and relevant methods of prevention and intervention. While these factors are important to the development of SUDs, there are plenty of other factors both known and unknown that influence the development of this disorder. As such, continued research is both necessary and invaluable.

### **Additional Mediators and Moderators of Substance Abuse**

**Mediators and Moderators Defined:** Baron and Kenny (1986) define a moderator as, "a qualitative (e.g., sex, race, class) or quantitative (e.g., level of reward) variable that affects the direction and/or strength of the relation between an independent or predictor variable and a dependent or criterion variable" (p. 1174). Moderators may operate as protective factors, decreasing the strength of the relationship between the predictor variable and the outcome. Conversely, moderators may heighten risk levels and strengthen the effects of the predictor on the outcome. In either instance, moderators do not explain why the connection exists, but rather affect the strength and direction of the relationship between the variables.

A mediator, as defined by Baron and Kenny (1986), "represents the generative mechanism through which the focal independent variable is able to influence the dependent variable of interest" (p. 1173). Unlike moderators, mediators can explain the relationship between the predictor variable and outcome. Holmbeck (1997) elaborated on Baron and Kenny's definition by adding, "the nature of the mediated relationship is such that the independent variable influences the mediator which, in turn, influences the outcome" (p. 600). Examples of mediators and moderators in empirical research:

Examples of mediators and moderators can be found in several empirical studies. For example, Pilgrim et al.'s hypothesized mediation model posited that school success and time spent with friends mediated the relationship between parental involvement and risk-taking behavior with substance use (2006). More specifically, the relationship between parental involvement and risk-taking behavior is explained via the interaction with third variables, school success and time spent with friends. In this example, increased parental involvement led to increased school success and decreased time with friends, both of which were associated with decreased drug use. Another example of mediation involved risk-taking behaviors. As risk-taking behaviors increased, school

success decreased and time with friends increased, both of which were associated with increased drug use. A second example of a mediating variable is depression. In a study by Lo and Cheng (2007), depression was found to mediate the relationship between childhood maltreatment and subsequent substance abuse in adulthood. In other words, childhood physical abuse is associated with increased depression, which in turn, is associated with increased drug and alcohol use in young adulthood. More specifically, depression helps to explain how childhood abuse is related to subsequent substance abuse in young adulthood.

A third example of a mediating variable is an increase of externalizing symptoms. King and Chassin (2008) conducted research examining the relationship between stressful life events and drug dependence in young adulthood. Their findings identified problematic externalizing behavior on subsequent substance dependency. In other words, stressful life events are associated with externalizing symptoms, such as aggression or hostility, which can lead to peer alienation or acceptance by socially deviant peers, which could lead to increased drug use. The relationship between stressful life events and subsequent drug dependence however exists via the presence of the mediation effects of externalizing behaviors.

An example of a moderating variable is level of cognitive distortion. An individual with high levels of cognitive distortion might react adversely to potentially innocuous events, and may have increased difficulty reacting to them in an adaptive manner (Shoal & Giancola, 2005). In their study, Shoal and Giancola investigated the moderating effects of cognitive distortion on adolescent substance use. Individuals with low levels of cognitive distortion may be more apt to choose more adaptive methods of coping with social problems, thereby potentially reducing the risk of drug use. Individuals with high levels of cognitive distortions, because of their increased misperceptions and misattributions, are at increased risk for social difficulties. Individuals may be more likely to react aggressively or inappropriately, potentially alienating themselves from their peers, thereby putting them at greater risk for delinquent behaviors, including substance use and abuse. In this study, social problems are a significant risk factor for drug use when moderated by high levels of cognitive distortions.

## **Terminology**

In the United States, physical dependence, abuse of, and withdrawal from drugs and other substances is outlined in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV TR). It does not use the word 'addiction' at all. It has instead a section about Substance dependence:

"Substance dependence When an individual persists in use of alcohol or other drugs despite problems related to use of the substance, substance dependence may be diagnosed. Compulsive and repetitive use may result in tolerance to the effect of the drug and withdrawal symptoms when use is reduced or stopped. This, along with Substance Abuse are considered Substance Use

Disorders..."

Terminology has become quite complicated in the field. Pharmacologists continue to speak of addiction from a physiologic standpoint (some call this a physical dependence); psychiatrists refer to the disease state as psychological dependence; most other physicians refer to the disease as addiction. The field of psychiatry is now considering, as they move from DSM-IV to DSM-V, transitioning from "substance dependence" to "addiction" as terminology for the disease state.

Addiction is now narrowly defined as "uncontrolled, compulsive use"; if there is no harm being suffered by, or damage done to, the patient or another party, then clinically it may be considered compulsive, but to the definition of some it is not categorized as 'addiction'. In practice, the two kinds of addiction are not always easy to distinguish. Addictions often have both physical and psychological components.

There is also a lesser known situation called pseudo-addiction. A patient will exhibit drug-seeking behavior reminiscent of psychological addiction, but they tend to have genuine pain or other symptoms that have been under-treated. Unlike true psychological addiction, these behaviors tend to stop when the pain is adequately treated.

Physical and psychological dependency

The medical community now makes a careful theoretical distinction between physical dependence (characterized by symptoms of withdrawal) and psychological dependence (or simply addiction).

The DSM definition of addiction can be boiled down to compulsive use of a substance (or engagement in an activity) despite ongoing negative consequences--this is also a summary of what used to be called "psychological dependency." Physical dependence, on the other hand, is simply needing a substance to function. Humans are all physically dependent on oxygen, food and water. A drug can cause physical dependence and not addiction (for example, some blood pressure medications, which can produce fatal withdrawal symptoms if not tapered) and can cause addiction without physical dependence (the withdrawal symptoms associated with cocaine are all psychological, there is no associated vomiting or diarrhea as there is with opiate withdrawal).

### **Physical dependency**

Physical dependence on a substance is defined by the appearance of characteristic withdrawal symptoms when the substance is suddenly discontinued. Opiates, benzodiazepines, barbiturates, alcohol and nicotine induce physical dependence. On the other hand, some categories of substances share this property and are still not considered addictive: cortisone, beta blockers and most antidepressants are examples. So, while physical dependency can be a major factor in the psychology of addiction and most often becomes a primary motivator in the continuation of an addiction, the initial primary attribution of an addictive substance is usually its ability to induce

pleasure, although with continued use the goal is not so much to induce pleasure as it is to relieve the anxiety caused by the absence of a given addictive substance, causing it to become used compulsively.

Some substances induce physical dependence or physiological tolerance - but not addiction -- for example many laxatives, which are not psychoactive; nasal decongestants, which can cause rebound congestion if used for more than a few days in a row; and some antidepressants, most notably venlafaxine, paroxetine and sertraline, as they have quite short half-lives, so stopping them abruptly causes a more rapid change in the neurotransmitter balance in the brain than many other antidepressants. Many non-addictive prescription drugs should not be suddenly stopped, so a doctor should be consulted before abruptly discontinuing them.

The speed with which a given individual becomes addicted to various substances varies with the substance, the frequency of use, the means of ingestion, the intensity of pleasure or euphoria, and the individual's genetic and psychological susceptibility. Some people may exhibit alcoholic tendencies from the moment of first intoxication, while most people can drink socially without ever becoming addicted. Opioid dependent individuals have different responses to even low doses of opioids than the majority of people, although this may be due to a variety of other factors, as opioid use heavily stimulates pleasure-inducing neurotransmitters in the brain. Nonetheless, because of these variations, in addition to the adoption and twin studies that have been well replicated, much of the medical community is satisfied that addiction is in part genetically moderated. That is, one's genetic makeup may regulate how susceptible one is to a substance and how easily one may become psychologically attached to a pleasurable routine.

Eating disorders are complicated pathological mental illnesses and thus are not the same as addictions described in this article. Eating disorders, which some argue are not addictions at all, are driven by a multitude of factors, most of which are highly different than the factors behind addictions described in this article. It has been reported, however, that patients with eating disorders can successfully be treated with the same non-pharmacological protocols used in patients with chemical addiction disorders. Gambling is another potentially addictive behavior with some biological overlap. Conversely gambling urges have emerged with the administration of Mirapex (pramipexole), a dopamine agonist.

The obsolete term physical addiction is deprecated, because of its connotations. In modern pain management with opioids physical dependence is nearly universal. While opiates are essential in the treatment of acute pain, the benefit of this class of medication in chronic pain is not well proven. Clearly, there are those who would not function well without opiate treatment; on the other hand, many states are noting significant increases in non-intentional deaths related to opiate use. High-quality, long-term studies are needed to better delineate the risks and benefits of chronic opiate use.

## **Psychological dependency**

In the now outdated conceptualization of the problem, psychological dependency leads to psychological withdrawal symptoms (such as cravings, irritability, insomnia, depression, anorexia, etc). Addiction can in theory be derived from any rewarding behaviour, and is believed to be strongly associated with the dopaminergic system of the brain's reward system (as in the case of cocaine and amphetamines). Some claim that it is a habitual means to avoid undesired activity, but typically it is only so to a clinical level in individuals who have emotional, social, or psychological dysfunctions (psychological addiction is defined as such), replacing normal positive stimuli not otherwise attained.

A person who is physically dependent, but not psychologically dependent can have their dose slowly dropped until they are no longer physically dependent. However, if that person is psychologically dependent, they are still at serious risk for relapse into abuse and subsequent physical dependence.

Psychological dependence does not have to be limited only to substances; even activities and behavioural patterns can be considered addictions, if they become uncontrollable, e.g. problem gambling, Internet addiction, computer addiction, sexual addiction / pornography addiction, overeating, self-injury, compulsive buying, or work addiction.

## **Management**

Early editions of the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders (DSM) described addiction as a physical dependency to a substance that resulted in withdrawal symptoms in its absence. Recent editions, including DSM-IV, have moved toward a diagnostic instrument that classifies such conditions as dependency, rather than addiction.

## **Addiction severity index**

Some medical systems, including those of at least 15 states of the United States, refer to an Addiction Severity Index to assess the severity of problems related to substance use. The index assesses problems in six areas: medical, employment/support, alcohol and other drug use, legal, family/social, and psychiatric.

## **Detoxification**

Early treatment of acute withdrawal often includes medical detoxification, which can include doses of anxiolytics or narcotics to reduce symptoms of withdrawal. An experimental drug, ibogaine, is

also proposed to treat withdrawal and craving.

Neurofeedback therapy has shown statistically significant improvements in numerous researches conducted on alcoholic as well as mixed substance abuse population. In chronic opiate addiction, a surrogate drug such as methadone is sometimes offered as a form of opiate replacement therapy. But treatment approaches universal focus on the individual's ultimate choice to pursue an alternate course of action.

### **Tailoring treatment**

Therapists often classify patients with chemical dependencies as either interested or not interested in changing.

Treatments usually involve planning for specific ways to avoid the addictive stimulus, and therapeutic interventions intended to help a client learn healthier ways to find satisfaction. Clinical leaders in recent years have attempted to tailor intervention approaches to specific influences that affect addictive behavior, using therapeutic interviews in an effort to discover factors that led a person to embrace unhealthy, addictive sources of pleasure or relief from pain.

### **Treatment modality matrix**

From the applied behavior analysis literature and the behavioral psychology literature, several evidenced-based intervention programs have emerged (1) behavioral marital therapy (2) community reinforcement approach (3) cue exposure therapy and (4) contingency management strategies. In addition, the same author suggests that social skills training adjunctive to inpatient treatment of alcohol dependence is probably efficacious.

### **Causes**

Several explanations (or "models") have been presented to explain addiction. These divide, more or less, into the models which stress biological or genetic causes for addiction, and those which stress social or purely psychological causes. Of course there are also many models which attempt to see addiction as both a physiological and a psycho-social phenomenon.

### **Psycho-social**

The free-will model or "life-process model" proposed by Thomas Szasz and later refined by Jeffrey Schaler questions the very concept of "addiction". Free-will model theorists argue that addiction cannot be a disease, because drug-taking is a behavior, and all behaviors are choices. Szasz views addiction as a metaphor, and that the only reason to make the distinction between habit and

addiction "is to persecute somebody." Free-will model theorists believe that individuals are capable of deliberate action in pursuit of chosen goals, and that physiology alone can never determine whether a person will take a drug, or how often they will take it. Although the free-will model of addiction has received much research support, it is opposed by groups like the American Psychiatric Association and the National Institute of Mental Health.

The pleasure model proposed by professor Nils Bejerot. Addiction "is an emotional fixation (sentiment) acquired through learning, which intermittently or continually expresses itself in purposeful, stereotyped behavior with the character and force of a natural drive, aiming at a specific pleasure or the avoidance of a specific discomfort." "The pleasure mechanism may be stimulated in a number of ways and give rise to a strong fixation on repetitive behavior. Stimulation with drugs is only one of many ways, but one of the simplest, strongest, and often also the most destructive" "If the pleasure stimulation becomes so strong that it captivates an individual with the compulsion and force characteristic of natural drives, then there exists...an addiction" The pleasure model is used as one of the reasons for zero tolerance for use of illicit drugs.

The experiential model devised by Stanton Peele argues that addictions occur with regard to experiences generated by various involvements, whether drug-induced or not. This model is in opposition to the disease, genetic, and neurobiological approaches. Among other things, it proposes that addiction is both more temporary or situational than the disease model claims, and is often outgrown through natural processes.

The opponent-process model generated by Richard Solomon states that for every psychological event A will be followed by its opposite psychological event B. For example, the pleasure one experiences from heroin is followed by an opponent process of withdrawal, or the terror of jumping out of an airplane is rewarded with intense pleasure when the parachute opens. This model is related to the opponent process color theory. If you look at the color red then quickly look at a gray area you will see green. There are many examples of opponent processes in the nervous system including taste, motor movement, touch, vision, and hearing. Opponent-processes occurring at the sensory level may translate "down-stream" into addictive or habit-forming behavior.

The allostatic (stability through change) model generated by George Koob and Michel LeMoal is a modification of the opponent process theory where continued use of a drug leads to a spiralling of uncontrolled use, negative emotional states and withdrawal and a shift into use to new allostatic set point which is lower than that maintained before use of the drug.

The cultural model recognizes that the influence of culture is a strong determinant of whether or not individuals fall prey to certain addictions. For example, alcoholism is rare among Saudi Arabians, where obtaining alcohol is difficult and using alcohol is prohibited. In North America, on the other hand, the incidence of gambling addictions soared in the last two decades of the 20th century, mirroring the growth of the gaming industry. Half of all patients diagnosed as alcoholic are

born into families where alcohol is used heavily, suggesting that familiar influence, genetic factors, or more likely both, play a role in the development of addiction. What also needs to be noted is that when people don't gain a sense of moderation through their development they can be just as likely, if not more, to abuse substances than people born into alcoholic families.

The moral model states that addictions are the result of human weakness, and are defects of character. Those who advance this model do not accept that there is any biological basis for addiction. They often have scant sympathy for people with serious addictions, believing either that a person with greater moral strength could have the force of will to break an addiction, or that the addict demonstrated a great moral failure in the first place by starting the addiction. The moral model is widely applied to dependency on illegal substances, perhaps purely for social or political reasons, but is no longer widely considered to have any therapeutic value. Elements of the moral model, especially a focus on individual choices, have found enduring roles in other approaches to the treatment of dependencies.

Similarly, the rational addiction model hypothesizes that addictions (to heroin, tobacco, television, etc.) can be usefully modeled as specific kinds of rational, forward-looking, optimal consumption plans. In other words, addiction is perceived as a rational response to individual and/or environmental factors.

The chemical model : Nearly all drugs, directly or indirectly, target the brain's reward system by flooding the circuit with dopamine. As a person continues to overstimulating the "reward circuit", the brain adapts to the overwhelming surges in dopamine by producing less of the hormones or by reducing the number of receptors in the reward circuit. As a result, the chemical's impact on the reward circuit is lessened, reducing the abuser's ability to enjoy the things that previously brought pleasure. This decrease compels those addicted to dopamine to increase the drug consumption in order to attempt to bring their "feel-good" hormone level back to normal --an effect known as tolerance. Development of dopamine tolerance can eventually lead to profound changes in neurons and brain circuits, with the potential to severely compromise the long-term health of the brain. Modern antipsychotics are designed to block dopamine function. Unfortunately, this blocking can also cause relapses in depression, and can increase addictive behaviors.

Finally, the blended model attempts to consider elements of all other models in developing a therapeutic approach to dependency. It holds that the mechanism of dependency is different for different individuals, and that each case must be considered on its own merits.

## **Physiological**

The disease model of addiction holds that addiction is a disease, coming about as a result of either the impairment of neurochemical or behavioral processes, or of some combination of the two.

Within this model, addictive disease is treated by specialists in Addiction Medicine. Within the clinical field, the American Medical Association, National Association of Social Workers, and American Psychological Association all have policies which are predicated on the theory that addictive processes represent a disease state. Most treatment approaches, as well, are based on the idea that dependencies are behavioral dysfunctions, and, therefore, contain, at least to some extent, elements of physical or mental disease. Organizations such as the American Society of Addiction Medicine believe the research-based evidence for addiction's status as a disease is overwhelming.

The genetic model posits a genetic predisposition to certain behaviors. It is frequently noted that certain addictions "run in the family," and while researchers continue to explore the extent of genetic influence, many researchers argue that there is strong evidence that genetic predisposition is often a factor in dependency.

The development of addiction is thought to involve a simultaneous process of 1) increased focus on and engagement in a particular behavior and 2) the attenuation or "shutting down" of other behaviors. For example, under certain experimental circumstances such as social deprivation and boredom, animals allowed the unlimited ability to self-administer certain psychoactive drugs will show such a strong preference that they will forgo food, sleep, and sex for continued access. The neuro-anatomical correlate of this is that the brain regions involved in driving goal-directed behavior grow increasingly selective for particular motivating stimuli and rewards, to the point that the brain regions involved in the inhibition of behavior can no longer effectively send "stop" signals. A good analogy is to imagine flooring the gas pedal in a car with very bad brakes. In this case, the limbic system is thought to be the major "driving force" and the orbitofrontal cortex is the substrate of the top-down inhibition.

A specific portion of the limbic circuit known as the mesolimbic dopaminergic system is hypothesized to play an important role in translation of motivation to motor behavior- and reward-related learning in particular. It is typically defined as the ventral tegmental area (VTA), the nucleus accumbens, and the bundle of dopamine-containing fibers that are connecting them. This system is commonly implicated in the seeking out and consumption of rewarding stimuli or events, such as sweet-tasting foods or sexual interaction. However, its importance to addiction research goes beyond its role in "natural" motivation: while the specific site or mechanism of action may differ, all known drugs of abuse have the common effect in that they elevate the level of dopamine in the nucleus accumbens. This may happen directly, such as through blockade of the dopamine re-uptake mechanism (see cocaine). It may also happen indirectly, such as through stimulation of the dopamine-containing neurons of the VTA that synapse onto neurons in the accumbens (see opiates). The euphoric effects of drugs of abuse are thought to be a direct result of the acute increase in accumbal dopamine.

The human body has a natural tendency to maintain homeostasis, and the central nervous system is no exception. Chronic elevation of dopamine will result in a decrease in the number of dopamine receptors available in a process known as downregulation. The decreased number of receptors changes the permeability of the cell membrane located post-synaptically, such that the post-synaptic neuron is less excitable- i.e.: less able to respond to chemical signaling with an electrical impulse, or action potential. It is hypothesized that this dulling of the responsiveness of the brain's reward pathways contributes to the inability to feel pleasure, known as anhedonia, often observed in addicts. The increased requirement for dopamine to maintain the same electrical activity is the basis of both physiological tolerance and withdrawal associated with addiction.

Downregulation can be classically conditioned. If a behavior consistently occurs in the same environment or contingently with a particular cue, the brain will adjust to the presence of the conditioned cues by decreasing the number of available receptors in the absence of the behavior. It is thought that many drug overdoses are not the result of a user taking a higher dose than is typical, but rather that the user is administering the same dose in a new environment.

In cases of physical dependency on depressants of the central nervous system such as opioids, barbiturates, or alcohol, the absence of the substance can lead to symptoms of severe physical discomfort. Withdrawal from alcohol or sedatives such as barbiturates or benzodiazepines (valium-family) can result in seizures and even death. By contrast, withdrawal from opioids, which can be extremely uncomfortable, is rarely if ever life-threatening. In cases of dependence and withdrawal, the body has become so dependent on high concentrations of the particular chemical that it has stopped producing its own natural versions (endogenous ligands) and instead produces opposing chemicals. When the addictive substance is withdrawn, the effects of the opposing chemicals can become overwhelming. For example, chronic use of sedatives (alcohol, barbiturates, or benzodiazepines) results in higher chronic levels of stimulating neurotransmitters such as glutamate. Very high levels of glutamate kill nerve cells, a phenomenon called excitatory neurotoxicity.

## **Epidemiology**

### **Addiction and drug control legislation**

Most countries have legislation which brings various drugs and drug-like substances under the control of licensing systems. Typically this legislation covers any or all of the opiates, amphetamines, cannabinoids, cocaine, barbiturates, hallucinogens (tryptamines, LSD, phencyclidine, and psilocybin) and a variety of more modern synthetic drugs, and unlicensed production, supply or possession may be a criminal offense.

Usually, however, drug classification under such legislation is not related simply to addictiveness.

The substances covered often have very different addictive properties. Some are highly prone to cause physical dependency, whilst others rarely cause any form of compulsive need whatsoever.

Also, although the legislation may be justifiable on moral grounds to some, it can make addiction or dependency a much more serious issue for the individual. Reliable supplies of a drug become difficult to secure as illegally produced substances may have contaminants. Withdrawal from the substances or associated contaminants can cause additional health issues and the individual becomes vulnerable to both criminal abuse and legal punishment. Criminal elements that can be involved in the profitable trade of such substances can also cause physical harm to users.

### **Opposition to common views**

Thomas Szasz denies that addiction is a psychiatric problem. In many of his works, he argues that addiction is a choice, and that a drug addict is one who simply prefers a socially taboo substance rather than, say, a low risk lifestyle. In *Our Right to Drugs*, Szasz cites the biography of Malcolm X to corroborate his economic views towards addiction: Malcolm claimed that quitting cigarettes was harder than shaking his heroin addiction. Szasz postulates that humans always have a choice, and it is foolish to call someone an 'addict' just because they prefer a drug induced euphoria to a more popular and socially welcome lifestyle.

Professor John Booth Davies at the University of Strathclyde has argued in his book *The Myth of Addiction* that 'people take drugs because they want to and because it makes sense for them to do so given the choices available' as opposed to the view that 'they are compelled to by the pharmacology of the drugs they take'. He uses an adaptation of attribution theory (what he calls the theory of functional attributions) to argue that the statement 'I am addicted to drugs' is functional, rather than veridical. Stanton Peele has put forward similar views.

Experimentally, Bruce K. Alexander used the classic experiment of Rat Park to show that 'addicted' behaviour in rats only occurred when the rats had no other options. When other options and behavioural opportunities were put in place, the rats soon showed far more complex behaviours.