Addicted to Ecstasy: A Misguided Search for Bliss

Adictos al éxtasis: una búsqueda equivocada de la felicidad

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Abstract

Addiction has been described from many perspectives, learning theory, neurobiology, and habituation among them. The eclectic model that we have advocated in this article explains why the mechanism of addiction is different for different individuals, and that each case must be considered on its own merits. As a result, it presents a challenge to social policy and to psychotherapy. Both the American Psychiatric Association's *Diagnostic and Statistical Manual for Mental Disorders* (DSM) and the World Health Organization's *International Classification of Disease* (ICD) have devoted considerable space to this topic, and the results are summarized here.

Keywords: addiction, dependency, ecstasy, DSM, ICD

Resumen

La adicción ha sido descrita desde muy diferentes perspectivas, teorías del aprendizaje, neurobiología y habituación. El modelo ecléctico que defendemos en este artículo explica por qué el mecanismo de adicción es diferente para cada individuo, y por lo tanto cada caso ha de tratarse individualmente. Como resultado, este trabajo presenta un desafío para la política social y la psicoterapia. Tanto el *Manual Diagnóstico y Estadístico de los Trastornos Mentales* (DSM) de la Asociación Americana de Psiquiatría, como la *Clasificación Internacional de las Enfermedades* (CIE) de la Organización Mundial de la Salud, han dedicado bastante atención a este tema, y aquí presentamos un resumen de los resultados.

Palabras clave: adicción, dependencia, éxtasis, DSM, CIE

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Ecstasy (from the Greek, *ekstasis*: distraction; derangement; astonishment) (Merriam-Webster, 2000) is (1) an emotional state so intense that one is carried beyond rational thought or self-control; (2) rapture associated with mystical experience.

History and Overview

Since prehistoric times, most human cultures and many animal species knew about and used psychoactive, or mind-changing, substances. Historical records indicate that many ancient societies had easy access to powerful plants and human-made concoctions, yet were able to regulate their use. Some of these substances were used for medications to alleviate distress and diseases, some were used to evoke transcendence during sacred rituals, and some were used to enhance hedonic pleasure during celebrations. In the Middle East, Eastern Asia, and North America, Cannabis compounds found a place in all three of those venues. Ancient Egyptians crushed the psychoactive blue lotus and mixed it with wine to induce euphoria. For two millennia, Grecian priests led initiates besotted with *kykeon* (a brew laced with a psychoactive ergot fungus) through the Eleusinian Mysteries. Amazonian shamans concocted *yagé* from a combination of rainforest plants. Psychoactive cacti and morning glory seeds were used by religious functionaries throughout Mexico and Latin America; in some areas, they also utilized psychoactive mushrooms, relatives of those ingested from time immemorial by Siberian shamans. Various liquors, inebriants, teas, and herbs were eaten, swallowed, and smoked to attain ecstasy, and when a participant overindulged or reacted, there were rites and antidotes to ease the hapless voyager back to consensual reality. Mircea Eliade (1964) referred to shamans as "masters of ecstasy" although he considered the use of drumming, chanting, and dancing superior to consciousness-altering substances.

Many tribes in the Americas used tobacco or coca leaves in their ceremonies, and fermented beverages were imbibed around the world to attain bliss. When Western societies learned how to distill alcohol and manufacture opium, the stage was set for epidemics of what would later be called "substance abuse." The colonists' sale of distilled spirits to Native Americans, followed by rampant alcoholism, and Britain's lucrative opium trade in China following the 18th century Opium Wars were examples of commercial greed preempting traditional patterns of controlled inebriation.

In recent decades, "drug cartels" and "drug lords" have undermined governments and corrupted legitimate businesses. In many parts of the world, thefts committed by users of illicit substances to support their "habit" are an everyday occurrence. Intravenous drug use has spread the HIV virus; entire communities are broken by drug procurement and use; impoverished young people often consider the local drug dealers their "role models" and are all too eager to serve as apprentices. Soaring health costs, broken families, and lost wages are some of the other side products of this "addiction to ecstasy." The filmmaker and actor Woody Allen once sardonically commented, "Life is full of misery, loneliness, and suffering – and it's all over much too soon."

"Addiction" as a Social Construct

Kenneth Gergen (2000) has noted that humanity delights in labeling objects and experiences, but often forgets that these social constructs might not be adequate maps of the territories they are describing. The label of "drug addiction" was socially constructed by physicians in the 19th century, who considered it a vice, a symptom of *akrasia,* a Greek term indicating the weakening of "will power." During the 20th century, the term was medicalized and "drug addiction" was reconceptualized as a "disease" (e.g., Leshner, 1997). The consequences of this "addiction" were brought to public attention, where they became the subject of negative and sensationalized media publicity and served as stimuli for restrictive legislation.

However, the resulting laws varied from region to region and from historical era to historical era (Haskell, 1993). For example, In the United States, alcoholic beverages survived the eras of prohibition to

become an economic windfall when legal distilleries reopened. For many years, opium, heroin, and cocaine were used medicinally and as ingredients in cough syrups and/or soft drinks until they were declared illegal and their manufacture and use was severely punished. In 1875, "opium dens" were outlawed in San Francisco, the first legal measure taken against illicit drugs in the United States. The Pure Food and Drug Act was passed in 1906, requiring testing and standardization of consumable items, including medicines.

Nonetheless, illegal trade has flourished and entire communities make their livelihood in the growth of these plants and the extraction of their mind-changing ingredients. Many stimulants (e.g., methamphetamine) and psychedelics (e.g., mescaline) were synthetic compounds based on ingredients found in plants that have distinct mind-changing properties. Military and medicinal uses were found for many stimulants (i.e., the amphetamines), but psychedelics were too unpredictable to find military applications and too controversial for their medical and psychotherapeutic potentials to gain widespread acceptance.

In this article, we take the position that "addiction" is a social construct, a term that reflects a societal convention as much or more so that its manifestation in the consensual world. Its definition differs from "expert" to "expert" and from culture to culture. Further, "addiction" (from the Latin *addicre*, to announce the transfer of a possession to another person) has accrued a variety of meanings (Hillman, 1991).

From our perspective, the term "addiction" implies a compulsive repetition of a pleasurable behaviour to the point that other activities are neglected or overlooked, to the detriment of the "addict," his or her family, and/or society-at-large. Because many addicts realize that the compulsion is not beneficial to their superordinate goals, bizarre reactions often result. F. Scott Fitzgerald, the celebrated author, ordered his household staff to keep him away from liquor, but sneaked drinks when they were not looking. Another writer, Samuel Taylor Coleridge, moved into his physician's home under the condition that he not be allowed any opium. He immediately suffered acute withdrawal symptoms until his publisher smuggled laudanum, an opium-laced tincture, into the doctor's house.

With the development of the neurosciences, psychologists, psychiatrists, and neurologists have developed new insights into how the pleasure-seeking, hedonic use of psychoactive substances can lead to a repetition of certain actions so frequently and with such compulsivity that it qualifies as an "addiction." Some of the same mechanisms are at work in addiction to substances with few psychoactive properties and to repetitive activities that evoke ecstasy, at least at the beginning of their employment. Examples would include compulsive food consumption, compulsive gambling, compulsive sexual activity, and compulsive ritualistic behaviour (from shoplifting to religious observances), among others. Most people can enjoy the bliss provided by these experiences, finding ways to integrate them into their overall repertoire of life activities. For others, there is a disruption of responsible behaviour and social commitments to the point where one might say that these men and women have become "addicted to ecstasy". This article focuses on hedonic drug use, since that is the area that has evoked the most scientific research, especially from the perspective of neuropsychology. However, other activities will be cited to round out the spectrum.

DSM and ICD

The American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*) does not give a specific diagnostic category to "addiction," but it classifies "addictive disorders" and uses the term *dependence* synonymously with "addiction." It states that "addiction/dependence" be "considered a manifestation of a behavioural, psychological, or biological dysfunction".

The World Health Organization's *International Classification of Mental and Behavioural Disorders (ICD)* (see Appendix A) states in its clinical descriptions and diagnostic guidelines that "addiction" is considered a *dependence syndrome*, and is a cluster of physiological, behavioural, and cognitive phenomena in which the use of a substance or a class of substances takes on a much higher priority for a given individual than other behaviours that once had greater value. A central descriptive characteristic of the dependence syndrome is the desire (often strong, sometimes overpowering) to take psychoactive drugs (which may or may not have been medically prescribed), alcohol, or tobacco. From the ICD perspective here may be

evidence that return to substance use after a period of abstinence leads to a more rapid reappearance of other features of the syndrome than occurs with nondependent individuals.

We will keep these synonymous uses of "dependence" and "addiction" in mind while we examine the neuropsychological and the neurobiological substrates of addictive behaviour (Rains, 2002).

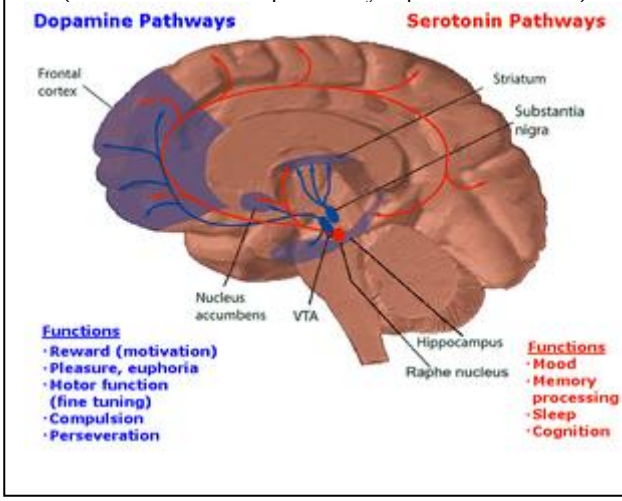
The Neuropsychology and Neurobiology of Hedonic Drug Use

Both neuropsychology and neurobiology, aim to understand how the structure and function of the brain and body relate to specific psychological and biological processes (see Figure 1). They are scientific in their approach and share an information processing view of the brain with cognitive-affective neuroscience (Kesner & Martinez, 2007; Rains, 2002). They are interdisciplinary in nature, overlapping at times with areas such as neuroscience, biology, philosophy (particularly philosophy of mind), social psychology, psychiatry, and computer science (particularly by making use of artificial neural networks). As mentioned earlier, we take the position that "addiction" is a social construct; nevertheless, it is important to determine the neuropsychological and neurobiological bases for behaviours that quality for that label (Erickson, 2007). This allows us to incorporate data into our model that indicate that there are important genetic genetic and social factors at play in hedonic drug use (Derringer, Krueger, McGue, & Iacono, 2008). We surmise that susceptibility to addiction may be epigenetic, due to an interaction between genes and their environment in the womb. This susceptibility sets the infant on a trajectory that can lead toward or away from addiction, depending on later interaction with family, peers, and the social environment. Hence, addiction can be seen as having predisposing, actuating, and maintaining factors, as is the case with many other human behaviours (Krippner & Winkler, 1996).

Figure 1

Brain Structure and Function

(Retrieved from Wikipedia Encyclopedia online 2009)



The brain's nucleus accumbens is a common neuroanatomical focus for the molecular actions of so-called "addictive" drugs in experimental animals, and probably in humans as well. Thus, addiction can be viewed as an adaptive response of the rest of the brain to the chronic effects of these agents. The nucleus accumbens is at the junction of a number of interacting memory systems in the brain that link emotions to behaviour (Kassel, 2010; Marlatt et al., 1988; Robinson & Berridge, 2003).

Hedonic drug use, when it transitions to addiction and, in some cases to relapse (or re-addiction), can be understood as forms of aberrant learning in which substances (or neurochemical changes resulting from compulsive behaviours not associated with substance ingestion) have subverted the natural "conditioning" mechanisms that organisms employ to anticipate important events and make constructive plans (Szasz, 1996). A somewhat different position has been taken by Robinson and Berridge (2003) who stressed the aberrant motivation aspect of addiction, and the triggering by cues as a learned motivation response of the brain. In either event, environmental stimuli that predict hedonic drug effects come to exert increasing influence on behaviour in the drug-dependent individual. This learning is affected by structures of the brain (e.g. the amygdala, the hippocampus, the prefrontal cortex). These structures communicate directly with themselves and the nucleus accumbens, serving to regulate its output (Robinson & Berridge, 2003). In other words, the process of addiction can be viewed from a social psychological and cognitive-affective level (involving learning, memory, emotion, and social milieu) as well as from a neuropsychological and neurobiological level (involving brain and body mechanisms).

From these points of view, addictive behaviour can be seen as a repetitive, compulsive self-defeating pattern of activities accompanied by intense emotion and that is impervious to rational thought and self-regulation. We would not describe frequent lovemaking or skydiving, extended periods of crocheting or meditation, or the enjoyment of dark chocolate or kumquats as "addictive" unless they became self-defeating rather than directed toward goals that the individual considers to be conducive to life mastery.

Both of these perspectives are necessary to resolve some of the contradictory data that appear in the literature about addictions. On the one hand, it is claimed that a person who uses a drug frequently becomes "tolerant" and "dependent," undergoing withdrawl symptoms when the drug is no longer available. However, this does not apply to gambling and compulsive shopping, which trigger endorphin-like opioids that produce a "high." It does not apply to patients who receive large doses of morphine-like opioids with little or no subsequent addiction. It does not apply to people (musicians, writers, and psychiatrists themselves) who are casual users of substances that the media considers so addictive that one injection will "hook" a person for life. It does not apply to the U.S. combat veterans who became "addicted" to high grade heroin in Vietnam, most of whom quit "cold turkey" on their return to the United States. It does not explain the mounting evidence that there are strong genetic, social, and environmental determinents of addiction that can mitigate drug use and other behaviours (Cheung & Erickson, 1997; Cohen, 1989; Robins et al., 1975). It is significant that the American Psychiatric Association shifted pathological gambling from being an impulse-control disorder to the addiction and related disorders category, reflecting these complexities and the severity of the condition (Gold & Werner, 2011).

The Development of an Addiction

There is controversy over whether activities such as compulsive gambling, compulsive sex, or compulsive computer use should be placed in the same category as shooting, snorting, or sniffing heroin (e.g., Robinson & Berridge, 2003, p. 46). Although we understand the logic of this argument, for the purposes of this article, we will tilt toward those writers who consider them "addictions," since all lead to the experience of ecstasy, bliss, and euphoria, at least in their initial stages. Therefore, we conceptualize addiction as a compulsive life-denying craving for the effects of a substance or behaviour that takes precedence over life-affirming activities. These individuals are not addicted to gambling per se, but to the "rush" that the experience provides. Nor are they addicted to methamphetamine per se, but to the "high" it yields. Someone might joke that they are "addicted" to chanting mantras, to working out in a gym, or to playing chess. From our perspective, these pastimes all enhance a person's quality, enjoyment, and affirmation of life. Only if they are done to the exclusion of social engagements, work commitments, and healthy living habits could they be considered life-denying. Until recently, this description would have been considered unduly "subjective," but the advent of "positive psychology" has brought with it a number of operational definitions of "strengths" and "virtues," as well as ways in which they can be measured (e.g., Snyder & Lopez, 2007).

In an attempt to construct an integrative and eclectic biopsychosocial model that could help resolve these contradictions, we propose that the development of an "addiction" involves a simultaneous process of 1) increased focus on and engagement in a particular behaviour and 2) the attenuation or "shutting down" of other behaviours, especially those that could service as life-enhancing alternatives. 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 for that activity that they will forgo food, sleep, and sex for continued access (Goeders & Smith, 1983). The neuroanatomical correlate of this is that the brain regions involved in driving goal-directed behaviour grow increasingly selective for particular motivating stimuli and rewards, to the point that the brain regions involved in the inhibition of behaviour can no longer effectively send "stop" signals. A useful 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 orbito-frontal cortex is the substrate of the top-down inhibition. However, consistent with our

model, the animals in the cited study did not compulsively choose to ingest drugs if other enjoyable alternatives were available.

A specific portion of the limbic circuit known as the mesolimbic dopaminergic system has been hypothesized to play an important role in translation of motivation to motor behaviour- 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, it's important that addiction research goes beyond its role in what is considered to be "natural" motivation. While the specific site or mechanism of action may differ, all known drugs with the propensity for addiction elevate the level of dopamine in the nucleus accumbens. This may happen directly, such as through blockage of the dopamine re-uptake mechanism (as with cocaine and tobacco). It may also happen indirectly, such as through stimulation of the dopamine-containing neurons of the VTA that synapse onto neurons in the accumbens (as with the morphine-like opiates). The euphoric effects of these drugs of abuse are considered to be a direct result of the acute increase in accumbal dopamine (Pich et al., 1997).

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, in other words 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 many longtime 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 an activity 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 behaviour. We suspect 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, one in which the conditioned cues are no longer operating.

In cases of physical dependency on depressants of the central nervous system such as morphine-type 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 (such as the valium family) can result in seizures and even death. By contrast, withdrawal from morphine-type opioids (such as opium and heroin), which can be extremely uncomfortable, is rarely if ever life-threatening. In cases of physical 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 (the 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 (such as alcohol, barbiturates, or the 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*.

This discussion has described how a person's brain and body become physically dependent on a substance (Jacobs-Stewart, 2010). However, we have emphasized that the addict typically becomes psychologically dependent upon the experiences evoked by that substance (or activity). Hence, our eclectic model proposes that a neuropsychological and neurobiological understanding of addiction needs to be understood in tandem with associated social and cognitive-affective data. For the former disciplines, the term "dependence" would seem to be a more appropriate descriptor than the term "addiction".

In either event, we would consider many people not to be "addicts" but "users", drinking regularly without becoming alcoholics or experimenting with illicit drugs; 90% of Americans who have tried illegal stimulants, depressants, psychedelics, or marijuana do not become addicted (Heyman, 1996, p. 563). We

would identify another group as "abusers," for whom drug use leads to serious problems but not to withdrawal symptoms or compulsive use. Users and abusers might end up as addicts, but we conjecture that most of them do not cross that line.

Educational Models: Learning, Memory, and Choice in Addiction

Some psychologists and psychiatrists have suggested that the terms "addict" and "addiction" have become unduly reified and that this has led to punitive legislation and counterproductive social practices. For example, Thomas Szasz (1996) has denied that "addiction" should be considered a psychiatric problem. Rather, he has considered it a metaphor for a ritualized *social habit* that reflects a choice, and that a "drug addict" is a person who simply prefers to ingest a socially taboo substance rather than, say, to engage in team sports. In his 1996 book, *Our Right to Drugs*, Szasz cited a statement of the social activist Malcolm X to corroborate his views towards the way that the term "addiction" is applied to users of some substances and not to others. Malcolm X claimed that quitting cigarettes was more difficult than shaking his heroin "addiction" but society did not label him a nicotine "addict". Szasz postulated that humans inevitably have a choice, and it is simplistic to call someone an "addict" just because he or she prefers a drug-induced euphoria to a more conventional pastime.

Szasz is not alone in questioning the disease model of addiction. John Booth Davies (1998) in *The Myth of Addiction* has proposed 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 position that "they are compelled to by the pharmacology of the drugs they take" (p. 18) Davies used an adaptation of attribution theory (one he called the *theory of functional attributions*) to argue that the statement "I am addicted to drugs" is functional, rather than veridical; it refers to behaviour rather than to the person engaging in that behaviour. Stanton Peele (1989) put forward similar views, arguing that addiction occurs with regard to educational experiences generated by various involvements, whether drug-induced or not. He proposed that addiction is both more temporary and situational than the medical model claims, and (in accord with our eclectic model) is often outgrown through natural processes. To Peele, the fallacy of the disease model of any kind of addictive behaviour is that it does not treat the activity in question as something for which the individual assumes some degree of responsibility. Peele insisted that the aim of treatment should be to help people replace the concept of themselves as "recovering victims" with a self-concept of self-efficacy. The disease model promulgates a dependence on others and an excuse to avoid taking responsibility for one's own behaviour.

Heyman (1996) agreed that drug consumption is a goal-oriented act, but asked how such an act, one that often requires considerable planning and preparation can be "out of control"? In their definitions of "addiction", both the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders* (4th edition) and the World Health Organization's *International Classification of Diseases* (10th edition) use the term "out of control." This dilemma is at the heart of the disagreement between the medical model of drug addiction, one that sees it as "involuntary" and the educational models of Szasz, Davies, and Peele who see it as a voluntary preference.

The author Williams Burroughs (1977) wrote, "You become a narcotics addict because you do not have strong motivations in any other direction. Junk wins by default" (p. xv). In examining this and other autobiographical accounts, Heyman (1996) found several common themes: recreational drug use slides into addiction, sometimes imperceptibly; addiction wreaks havoc on friendships, occupations, family life, and health; attempts to stop the addiction have mixed results; drug use becomes the focal point of everyday life. The street addict's daily routines become organized around the ability to "score" drugs; the professional worker's social life become organized in terms of the amount of liquor the host is likely to provide. If the addict ever gains control over the drug use, it frequently follows "hitting bottom" or some other dramatic event (p. 562). For example, the "bottom of the barrel" is a common theme in the stories told by members of Alcoholics Anonymous (AA), a group that never quite accepted the medical model, preferring to replace the

"spiritual rapture" reported by many alcoholics with "surrender" to a "higher power." For AA, there is nothing amiss about searching for bliss as long as one finds it in all the right places.

An Attempt to Resolve the Contradictions in Addiction Research

As we noted early in this chapter, there are many ways of ingesting a natural substance or a chemically constituted drug just as there are many different cultural ways of eating or drinking. Many ancient cultures knew how to regulate taboo substances and high risk social behaviour. (Before we romanticize indigenous societies, we need to recall that several of them provided leadership opportunities to women and to sexual minorities, and nurtured their children well; but other societies were sexist, homophobic, and abused children both emotionally and physically.) Nonetheless, in our opinion, too many contemporary cultures are maintaining taboos on ecstatic experience that have outlived what usefulness they ever had, a restriction that is especially punitive to those seekers of pleasure and euphoria in ways that if properly regulated would harm nobody but themselves, and may even be of social value.

The disease model holds that addicion comes about as a result of either the impairment of neurobiological or neuropsychological processes, or some combination of the two. In the United States, the American Medical Association, the American Psychiatric Association, the National Association of Social Workers, and the American Psychological Association all have policies which are predicated on the theory that addictive processes represent a diseased state. Most treatment approaches, as well, are based, at least to some extent, on elements of physical or mental disease (Prescott & Kendler, 1999).

An example of the educational model has been proposed by Nils Bejerot (1980) for whom addiction is an emotional fixation (or sentiment) acquired through learning, either intermittently or through the 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 behaviour. 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 an "addiction" exists. The pleasure model is used as one of the reasons for advocates of "zero tolerance" programs for illicit drug use.

Numerous models of addiction compete for attention, for example, those that highlight genetic predispositions (Iacono, Malone, & McGue, 2008), "opponent processing" (in which an addict's pleasure turns to pain, leading to more drugs, more food, or more alcohol; Solomon, 1980), and the influence of an addiction-prone culture, ethnicity, or family (Bennett & Holloway, 2005).

Heyman (1996) has read numerous biographies of addicts, both unknown and celebrated, finding exceptions to each of these explanations. For example he has cited evidence that cost, probability of arrest, and embarasssment are potent factors in curbing addictive behaviour but did not easily fit into existing explanations, especially the disease model. Elicitng stimuli and reinforcing consequences are diffficult to separate becaue they act in concert. Withdrawal symptoms indicate that drug consumption has brought about biological changes, but not all addictions produce withdrawal when terminated, hence it is neither a necessary nor sufficient condition for addiction (p. 566). Most educational models postulate that behaviour can be controlled by its consequences. However, in operant conditioning, aversive consequences are held to decrease behaviour, a phenomenon not found in addiction (p. 567). Learning theory also holds that pleasure is reinforcing, but the addict has many other reinforcements in his or her life, many of them at odds with the addictive behaviour (p, 567).

Instead, Heyman proposed a "matching law" theory of choice for addicts. The elementary process governing choice is not maximization of costs and benefits (which hardly explains addictive behaviour) for "ameliorization" where existing choices compete against each other and the choice that seems best at a given moment is the one that prevails. Immediate pleasure may win out over long-term gains. A quick "fix" and the euphoria it brings is chosen over future adverse effects. Many drug effects are marked by *tolerance;* the addict must take more and more of a drug to obtain a similar effect. In hospital settings, a patient with access

to his own dispenser might increase his daily dosage tenfold. Tolerance also occurs with alcohol, stimulants, and nicotine. The increased demand for the addictive substance of activity exacts steep costs on the addict's everyday responsibilities. The process of ameliorization causes these activities to retain a diminishing value as the addiction continues. As what seems to be the "best" local value goes up, the value of the competing local and global rewards goes down. It is as if there is a hidden bookkeeper, one whose function needs to be swtiched from focus on immediate local values to longterm global values. When drugs are inacessible and choices are under scrutinty (such as by fellow members of an AA or other 12-step program), the addict is less likely to use them. But if the drugs become available at a time when the support group is not present, relapse may easily occur.

In summary, Heyman's model is not compatible with the idea that addiction is an involuntary state or that the addict is "out of control". The addict is very much in control, but is making choices, even if the reward is small in comparison with a "larger but later" reward (p. 572). Since addiction depends on elementary choice mechanisms, "everyone is a potential addict" (p. 573). Addictive behaviour is voluntary even though its effects are aversive. "The matching law" emphasizes local and immediate rather than global, overall values. Indeed, Heyman's (2009) "matching law" and "meliorization" can be applied to gambling, credit card abuse, sexual exhibitionism, and other behaviours often considered "addictive." He concluded; "Meliorization is the product of an experimental procedure that unfortunately confounds two processes, over evaluation of immediate consequences and mis-estimation of delayed consequences" (p. 574).

The eclectic model that we have advocated in this article finds eloquent expression in Heyman's resolution of the contradictions in addiction research. It explains why the mechanism of dependency is different for different individuals, and that each case must be considered on its own merits. As a result, it presents a challenge to social policy and to psychological therapy. However, a collection of essays published by the American Psychological Association (Marlatt & Witkiewitz, 2009) is an example of an attempt to present what is currently known about the effects of drug action, the epidemiology and etiology of drug and alcohol abuse, the design of prevention programs, understanding the trajectory of substance abuse and family risk factors, screening and assessment, identifying the most suitable models of treatment, and comprehending the needs of specific populations.

Coda

Some readers of this article may be puzzled why we have not mentioned the popular drug "Ecstasy" in our discussion. We have saved it to make a final provocative remark. In 2009, the Chair of the British Advisory Council on the Misuse of Drugs called for Ecstasy (methylenedioxymethaphetamine or MDMA) to be downgraded to a Class B (rather than a Class A) substance. He observed that an "addiction to horseback riding" caused some 100 road accidents and 10 deaths in 2008 compared with about 30 deaths attributed to organ failure due to ingestion of contaminents found in street MDMA or Ecstasy (Bates, 2009). Although heavily criticized for this remark, it serves as a reminder that social policy often uses drugs as scapegoats for political reasons, when there are other social ills, and even socially approved paths to ecstacy (e.g., horseback riding, deep-sea diving, installing home electrical appliances, and mountain climbing) that put more people at risk for health, for safety, and for life itself. Eckhart Tolle (1999) has reminded us that:

For most humans, the only respite they find from their own minds is to occasionally revert to a level of consciousness below thought. Everyone does that every night during sleep. But this also happens to some extent through sex, alcohol, and other drugs that suppress excessive mind activity. If it weren't for alcohol, tranquilizers, antidepressants, as well as the illegal drugs, which are all consumed in vast quantities, the insanity of the human mind would become even more glaringly obvious than it is already. The drugs, of course simply keep you stuck in dysfunction. The widespread use only delays the breakdown of the old mind structures and the emergence of higher consciousness. While individual users may get some relief

from the daily torture inflicted on them by their minds, they are prevented from generating enough conscious presence to rise above thought and so find true liberation (p. 102).

In other words, ecstasy, euphoria, and bliss are great – but let not your search be misguided.

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Appendix A

The World Health Organization's categories of addictive behaviours due to psychoactive substances.

<u>The ICD-10 Classification of Mental and Behavioural Disorders: Clinical descriptions and diagnostic guidelines</u>

F10 - F19
Mental and behavioural disorders due to psychoactive substance use.
Overview of this block

F10. – Mental and behavioural disorders due to use of alcohol
F11. – Mental and behavioural disorders due to use of opioids
F12. – Mental and behavioural disorders due to use of cannabinoids
F13. – Mental and behavioural disorders due to use of sedative hypnotics
F14. – Mental and behavioural disorders due to use of cocaine
F15. – Mental and behavioural disorders due to use of other stimulants, including caffeine
F16. – Mental and behavioural disorders due to use of hallucinogens
F17. – Mental and behavioural disorders due to use of tobacco
F18. – Mental and behavioural disorders due to use of volatile solvents
F19. – Mental and behavioural disorders due to multiple drug use and use of other psychoactive substances

Four- and five-character codes may be used to specify the clinical conditions, as follows:

F1*x*.0 Acute intoxication
.00 Uncomplicated
.01 With trauma or other bodily injury
.02 With other medical complications
.03 With delirium
.04 With perceptual distortions
.05 With coma
.06 With convulsions
.07 Pathological intoxication

F1*x*.1 Harmful use

F1*x*.2 Dependence syndrome
.20 Currently abstinent
.21 Currently abstinent, but in a protected environment
.22 Currently on a clinically supervised maintenance or replacement regime [controlled dependence]
.23 Currently abstinent, but receiving treatment with aversive or blocking drugs
.24 Currently using the substance [active dependence]
.25 Continuous use
.26 Episodic use [dipsomania]

F1*x*.3 Withdrawal state
.30 Uncomplicated
.31 With convulsions

F1*x*.4 Withdrawal state with delirium
.40 Without convulsions
.41 With convulsions

F1*x*.5 Psychotic disorder
.50 Schizophrenia-like
.51 Predominantly delusional
.52 Predominantly hallucinatory
.53 Predominantly polymorphic
.54 Predominantly depressive symptoms
.55 Predominantly manic symptoms
.56 Mixed

F1*x*.6 Amnesic syndrome

F1*x*.7 Residual and late-onset psychotic disorder
.70 Flashbacks
.71 Personality or behaviour disorder
.72 Residual affective behaviour
.73 Dementia
.74 Other persisting cognitive behaviour
.75 Late-onset psychotic disorder

F1x.8 Other mental and behavioural disorders

F1x.9 Unspecified mental and behavioural disorder.

This block contains a wide variety of disorders that differ in severity (from uncomplicated intoxication and harmful use to obvious psychotic disorders and dementia), but that are all attributable to the use of one or more psychoactive substances (which may or may not have been medically prescribed). The substance involved is indicated by means of the second and third characters (i.e. the first two digits after the letter F), and the fourth and fifth characters specify the clinical states. To save space, all the psychoactive substances are listed first, followed by the four-character codes; these should be used, as required, for each substance specified, but it should be noted that not all four-character codes are applicable to all substances.

Diagnostic guidelines

Identification of the psychoactive substance used may be made on the basis of self-report data, objective analysis of specimens of urine, blood, etc, or other evidence (presence of drug samples in the patient's possession, clinical signs and symptoms, or reports from informed third parties). It is always advisable to seek corroboration from more than one source of evidence relating to substance use. Objective analyses provide the most compelling evidence of present or recent use, though these data have limitations with regard to past use and current levels of use. Many drug users take more than one type of drug, but the diagnosis of the disorder should be classified, whenever possible, according to the most important single substance (or class of substances) used. This may usually be done with regard to the particular drug, or type of drug, causing the presenting disorder. When in doubt, code the drug or type of drug most frequently misused, particularly in those cases involving continuous or daily use.

Only in cases in which patterns of psychoactive substance taking are chaotic and indiscriminate, or in which the contributions of different drugs are inextricably mixed, should code F19. – be used (disorders resulting from multiple drug use).

Misuse of other than psychoactive substances, such as laxatives or aspirin, should be coded by means of F55. – (abuse of non-dependence-producing substances), with a fourth character to specify the type of substance involved. Cases in which mental disorders (particularly delirium in the elderly) are due to psychoactive substances, but without the presence of one of the disorders in this block (e.g. harmful use or dependence syndrome), should be coded in F00 – F09. Where a state of delirium is superimposed upon such a disorder in this block, it should be coded by means of F1*x*.3 or F1*X*.4.

The level of alcohol involvement can be indicated by means of a supplementary code from Chapter XX of ICD-10: Y90. – (evidence of alcohol involvement determined by blood alcohol content) or Y91. – (evidence of alcohol involvement determined by level of intoxication).

F1*x*.0 Acute intoxication

A transient condition following the administration of alcohol or other psychoactive substance, resulting in disturbances in level of consciousness, cognition, perception, affect or behaviour, or other psychophysiological functions and responses. This should be a main diagnosis only in cases where intoxication occurs without more persistent alcohol- or drug-related problems being concomitantly present. Where there are such problems, precedence should be given to diagnoses of harmful use (F1*x*.1), dependence syndrome (F1*x*.2), or psychotic disorder (F1*x*.5).

Diagnostic guidelines

Acute intoxication is usually closely related to dose levels (see ICD-10, Chapter XX).

Exceptions to this may occur in individuals with certain underlying organic conditions (e.g. renal or hepatic insufficiency) in whom small doses of a substance may produce a disproportionately severe intoxicating effect. Disinhibition due to social context should also be taken into account (e.g. behavioural disinhibition at parties or carnivals). Acute intoxication is a transient phenomenon. Intensity of intoxication lessens with time, and effects eventually disappear in the absence of further use of the substance. Recovery is therefore complete except where tissue damage or another complication has arisen.

Symptoms of intoxication need not always reflect primary actions of the substance: for instance, depressant drugs may lead to symptoms of agitation or hyperactivity, and stimulant drugs may lead to socially withdrawn and introverted behaviour. Effects of substances such as cannabis and hallucinogens may be particularly unpredictable. Moreover, many psychoactive substances are capable of producing different types of effect at different levels. For example, alcohol may have apparently stimulant effects on behaviour at lower dose levels, lead to agitation and aggression with increasing dose levels, and produce clear sedation at very high levels.

Includes:
Acute drunkenness in alcoholism "bad trips" (due to hallucinogenic drugs) drunkenness NOS

Differential diagnosis.
Consider acute head injury and hypoglycemia. Consider also the possibilities of intoxication as the result of mixed substance use.

The following five-character codes may be used to indicate whether the acute intoxication was associated with any complications:

F1*x*.00 Uncomplicated Symptoms of varying severity, usually dose-dependent, particularly at high dose levels.
F1*x*.01 With trauma or other bodily injury
F1*x*.02 With other medical complications. Complications such as haematemesis, inhalation of vomitus.
F1*x*.03 With delirium
F1*x*.04 With perceptual distortions
F1*x*.05 With coma
F1*x*.06 With convulsions
F1*x*.07 Pathological intoxication. Applies only to alcohol. Sudden onset of aggression and often violent behaviour that is not typical of the individual when sober, very soon after drinking amounts of alcohol that would not produce intoxication in most people.

F1*x*.1 Harmful use
A pattern of psychoactive substance use that is causing damage to health. The damage may be physical (as in cases of hepatitis from the self-administration of injected drugs) or mental (e.g. episodes of depressive disorder secondary to heavy consumption of alcohol).

Diagnostic guidelines
The diagnosis requires that actual damage should have been caused to the mental or physical health of the user. Harmful patterns of use are often criticized by others and frequently associated with adverse social consequences of various kinds. The fact that a pattern of use or a particular substance is disapproved of by another person or by the culture, or may have led to socially negative consequences such as arrest or marital arguments is not in itself evidence of harmful use.

Acute intoxication (see F1*x*.0), or "hangover" is not itself sufficient evidence of the damage to health required for coding harmful use. Harmful use should not be diagnosed if dependence syndrome (F1*x*.2), a psychotic disorder (F1*x*.5), or another specific form of drug- or alcohol-related disorder is present.

F1*x*.2 Dependence syndrome

A cluster of physiological, behavioural, and cognitive phenomena in which the use of a substance or a class of substances takes on a much higher priority for a given individual than other behaviours that once had greater value. A central descriptive characteristic of the dependence syndrome is the desire (often strong, sometimes overpowering) to take psychoactive drugs (which may or may not have been medically prescribed), alcohol, or tobacco. There may be evidence that return to substance use after a period of abstinence leads to a more rapid reappearance of other features of the syndrome than occurs with nondependent individuals.

Diagnostic guidelines

A definite diagnosis of dependence should usually be made only if three or more of the following have been present together at some time during the previous year:

(a) a strong desire or sense of compulsion to take the substance;
(b) difficulties in controlling substance-taking behaviour in terms of its onset, termination, or levels of use;
(c) a physiological withdrawal state (see F1*x*.3 and F1*x*.4) when substance use has ceased or been reduced, as evidenced by: the characteristic withdrawal syndrome for the substance; or use of the same (or a closely related) substance with the intention of relieving or avoiding withdrawal symptoms;
(d) evidence of tolerance, such that increased doses of the psychoactive substances are required in order to achieve effects originally produced by lower doses (clear examples of this are found in alcohol- and opiate-dependent individuals who may take daily doses sufficient to incapacitate or kill nontolerant users);
(e) progressive neglect of alternative pleasures or interests because of psychoactive substance use, increased amount of time necessary to obtain or take the substance or to recover from its effects;
(f) persisting with substance use despite clear evidence of overtly harmful consequences, such as harm to the liver through excessive drinking, depressive mood states consequent to periods of heavy substance use, or drug-related impairment of cognitive functioning; efforts should be made to determine that the user was actually, or could be expected to be, aware of the nature and extent of the harm.

Narrowing of the personal repertoire of patterns of psychoactive substance use has also been described as a characteristic feature (e.g. a tendency to drink alcoholic drinks in the same way on weekdays and weekends, regardless of social constraints that determine appropriate drinking behaviour). It is an essential characteristic of the dependence syndrome that either psychoactive substance taking or a desire to take a particular substance should be present; the subjective awareness of compulsion to use drugs is most commonly seen during attempts to stop or control substance use. This diagnostic requirement would exclude, for instance, surgical patients given opioid drugs for the relief of pain, who may show signs of an opioid withdrawal state when drugs are not given but who have no desire to continue taking drugs.

The dependence syndrome may be present for a specific substance (e.g. tobacco or diazepam), for a class of substances (e.g. opioid drugs), or for a wider range of different substances (as for those individuals who feel a sense of compulsion regularly to use whatever drugs are available and who show distress, agitation, and/or physical signs of a withdrawal state upon abstinence).

Includes:
Chronic alcoholism
Dipsomania

Drug "addiction"
The diagnosis of the dependence syndrome may be further specified by the following five-character codes:

F1*x*.20 Currently abstinent
F1*x*.21 Currently abstinent, but in a protected environment (e.g. in hospital, in a therapeutic community, in prison, etc.)
F1*x*.22 Currently on a clinically supervised maintenance or replacement regime [controlled dependence] (e.g. with methadone; nicotine gum or nicotine patch)
F1*x*.23 Currently abstinent, but receiving treatment with aversive or blocking drugs (e.g. naltrexone or disulfiram)
F1*x*.24 Currently using the substance [active dependence]
F1*x*.25 Continuous use
F1*x*.26 Episodic use [dipsomani]

F1*x*.3 Withdrawal state
A group of symptoms of variable clustering and severity occurring on absolute or relative withdrawal of a substance after repeated, and usually prolonged and/or high dose, use of that substance. Onset and course of the withdrawal state are time-limited and are related to the type of substance and the dose being used immediately before abstinence. The withdrawal state may be complicated by convulsions.

Diagnostic guidelines
Withdrawal state is one of the indicators of dependence syndrome (see F1*x*.2) and this latter diagnosis should also be considered. Withdrawal state should be coded as the main diagnosis if it is the reason for referral and sufficiently severe to require medical attention in its own right. Physical symptoms vary according to the substance being used. Psychological disturbances (e.g. anxiety, depression, and sleep disorders) are also common features of withdrawal. Typically, the patient is likely to report that withdrawal symptoms are relieved by further substance use.

It should be remembered that withdrawal symptoms can be induced by conditioned/learned stimuli in the absence of immediately preceding substance use. In such cases a diagnosis of withdrawal state should be made only if it is warranted in terms of severity.

Differential diagnosis.
Many symptoms present in drug withdrawal state may also be caused by other psychiatric conditions, e.g. anxiety states, and depressive disorders. Simple "hangover" or tremor due to other conditions should not be confused with the symptoms of a withdrawal state.
The diagnosis of withdrawal state may be further specified by using the following five-character codes:

F1*x*.30 Uncomplicated
F1*x*.31 With convulsions

F1*x*.4 Withdrawal state with delirium
A condition in which the withdrawal state (see F1*x*.3) is complicated by delirium (see criteria for F05. -).
Alcohol-induced *delirium tremens* should be coded here. *Delirium tremens* is a short-lived, but occasionally life-threatening, toxic-confusional state with accompanying somatic disturbances. It is usually a consequence of absolute or relative withdrawal of alcohol in severely dependent users with a long history of use. Onset usually occurs after withdrawal of alcohol. In some cases the disorder appears during an episode of heavy drinking, in which case it should be coded here. Prodromal symptoms typically include insomnia, tremulousness, and fear. Onset may also be preceded by withdrawal convulsions. The classical triad of symptoms includes clouding of consciousness and confusion, vivid hallucinations and illusions affecting any

sensory modality, and marked tremor. Delusions, agitation, insomnia or sleep-cycle reversal, and autonomic overactivity are usually also present.

Excludes:
*D*elirium, not induced by drugs and alcohol (F05. -)
The diagnosis of withdrawal state with delirium may be further specified by using the following five-character codes:

F1*x*.40 Without convulsions
F1*x*.41 With convulsions

F1*x*.5 Psychotic disorder
A cluster of psychotic phenomena that occur during or immediately after psychoactive substance use and are characterized by vivid hallucinations (typically auditory, but often in more than one sensory modality), misidentifications, delusions and/or ideas of reference (often of a paranoid or persecutory nature), psychomotor disturbances (excitement of stupor), and an abnormal affect, which may range from intense fear to ecstasy. The sensorium is usually clear but some degree of clouding of consciousness, though not severe confusion, may be present. The disorder typically resolves at least partially within 1 month and fully within 6 months.

Diagnostic guidelines
A psychotic disorder occurring during or immediately after drug use (usually within 48 hours) should be recorded here provided that it is not a manifestation of drug withdrawal state with delirium (see F1*x*.4) or of late onset. Late-onset psychotic disorders (with onset more than 2 weeks after substance use) may occur, but should be coded as F1*x*.75.
Psychoactive substance-induced psychotic disorders may present with varying patterns of symptoms. These variations will be influenced by the type of substance involved and the personality of the user. For stimulant drugs such as cocaine and amphetamines, drug-induced psychotic disorders are generally closely related to high dose levels and/or prolonged use of the substance.

A diagnosis of psychotic disorder should not be made merely on the basis of perceptual distortions or hallucinatory experiences when substances having primary hallucinogenic effects (e.g. lysergic (LSD), mescaline, cannabis at high doses) have been taken. In such cases, and also for confusional states, a possible diagnoses of acute intoxication (F1*x*.0) should be considered. Particular care should also be taken to avoid mistakenly diagnosing a more serious condition (e.g. schizophrenia) when a diagnosis of psychoactive substance-induced psychosis is appropriate. Many psychoactive substance-induced psychotic states are of short duration provided that no further amounts of the drug are taken (as in the case of amphetamine and cocaine psychoses). False diagnosis in such cases may have distressing and costly implications for the patient and for the health services.

Includes:
alcoholic hallucinosis
alcoholic jealousy
alcoholic paranoia
alcoholic psychosis NOS

Differential diagnosis.
Consider the possibility of another mental disorder being aggravated or precipitated by psychoactive substance use (e.g. schizophrenia (F20. -); mood [affective] disorder (F30-F39); paranoid or schizoid

personality disorder (F60.0, F60.1). In such cases, a diagnosis of psychoactive substance-induced psychotic state may be inappropriate. The diagnosis of psychotic state may be further specified by the following five character codes:

F1*x*.50 Schizophrenia-like
F1*x*.51 Predominantly delusional
F1*x*.52 Predominantly hallucinatory (includes alcoholic hallucinosis)
F1*x*.53 Predominantly polymorphic
F1*x*.54 Predominantly depressive symptoms
F1*x*.55 Predominantly manic symptoms
F1*x*.56 Mixed

F1*x*.6 Amnesic syndrome
A syndrome associated with chronic prominent impairment of recent memory; remote memory is sometimes impaired, while immediate recall is preserved. Disturbances of time sense and ordering of events are usually evident, as are difficulties in learning new material. Confabulation may be marked but is not invariably present. Other cognitive functions are usually relatively well preserved and amnesic defects are out of proportion to other disturbances.

Diagnostic guidelines
Amnesic syndrome induced by alcohol or other psychoactive substances coded here should meet the general criteria for organic amnesic syndrome (see F04). The primary requirements for this diagnosis are:

(a) memory impairment as shown in impairment of recent memory (learning of new material); disturbances of time sense (rearrangements of chronological sequence, telescoping of repeated events into one, etc.);
(b) absence of defect in immediate recall, impairment of consciousness, and of generalized cognitive impairment;
(c) history or objective evidence of chronic (and particularly high-dose) use of alcohol or drugs.
Personality changes, often with apparent apathy and loss of initiative, and a tendency towards self-neglect may also be present, but should not be regarded as necessary conditions for diagnosis. Although confabulation may be marked it should not be regarded as a necessary prerequisite for diagnosis.

Includes:
Korsakov's psychosis or syndrome, alcohol- or other psychoactive substance-induced.

Differential diagnosis.
Consider: organic amnesic syndrome (nonalcoholic) (see F04); other organic syndromes involving marked impairment of memory (e.g. dementia or delirium) (F00-F03; F05. -); a depressive disorder (F31 – F33).

F1*x*.7 Residual and late-onset psychotic disorder
A disorder in which alcohol- or psychoactive substance-induced changes of cognition, affect, personality, or behaviour persist beyond the period during which a direct psychoactive substance-related effect might reasonably be assumed to be operating.

Diagnostic guidelines
Onset of the disorder should be directly related to the use of alcohol or a psychoactive substance. Cases in which initial onset occurs later than episode(s) of substance use should be coded here only where clear and strong evidence is available to attribute the state to the residual effect of the substance. The disorder should represent a change from or marked exaggeration of prior and normal state of functioning. The disorder

should persist beyond any period of time during which direct effects of the psychoactive substance might be assumed to be operative (see F1*x*.0, acute intoxication). Alcohol- or psychoactive substance-induced dementia is not always irreversible; after an extended period of total abstinence, intellectual functions and memory may improve. The disorder should be carefully distinguished from withdrawal-related conditions (see F1*x*.3 and F1*x*.4). It should be remembered that, under certain conditions and for certain substances, withdrawal state phenomena may be present for a period of many days or weeks after discontinuation of the substance. Conditions induced by a psychoactive substance, persisting after its use, and meeting the criteria for diagnosis of psychotic disorder should not be diagnosed here (use F1*x*.5, psychotic disorder). Patients who show the chronic end-state of Korsakov's syndrome should be coded under F1*x*.6.

Differential diagnosis.
Consider: pre-existing mental disorder masked by substance use and re-emerging as psychoactive substance-related effects fade (for example, phobic anxiety, depressive disorders, schizophrenia, or schizotypal disorder). In the case of flashbacks, consider acute and transient psychotic disorders (F23. -). Consider also organic injury and mild or moderate mental retardation (F70 – F71), which may coexist with psychoactive substance misuse. This diagnostic rubric may be further subdivided by using the following five-character codes:

F1*x*.70 Flashbacks. May be distinguished from psychotic disorders partly by their episodic nature, frequently of very short duration (seconds or minutes) and by their duplication (sometimes exact) of previous drug-related experiences.
F1*x*.71 Personality or behaviour disorder. Meeting the criteria for organic personality disorder (F07.0).
F1*x*.72 Residual affective disorder. Meeting the criteria for organic mood [affective] disorders (F06.3).
F1*x*.73 Dementia. Meeting the general criteria for dementia as outlined in the introduction to F00-F09.
F1*x*.74 Other persisting cognitive impairment. A residual category for disorders with persisting cognitive impairment, which do not meet the criteria for psychoactive substance-induced amnesic syndrome (F1*x*.6) or dementia (F1*x*.73).
F1*x*.75 Late-onset psychotic disorder

F1*x*.8 Other mental and behavioural disorders
Code here any other disorder in which the use of a substance can be identified as contributing directly to the condition, but which does not meet the criteria for inclusion in any of the above disorders.

F1*x*.9 Unspecified mental and behavioural disorder

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