The Evolutionary Basis for Drug Abuse

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Abstract

Evolutionary theory, especially evolutionary psychology, should indicate that the vulnerability to drug abuse, being highly negatively adapted, would have been selected out of modern populations. Since this is observed to not be true, other mechanisms explaining the persistence of this vulnerability must be present. These are examined, and testable consequences of these explanations are given, along with suggestions for how this should influence public policy with respect to controlling drug abuse.

Introduction: Why Think About Evolution?

One of the most successful overarching frameworks for the study of biology in the last century and a half has been the theory of evolution, as originally proposed by Charles Darwin and Alfred Wallace. The central idea of this theory are the ideas that individual animals and plants are genetically diverse, and this diversity, tested against the stresses of the environment, directly contributes to their reproductive success in positive or negative ways. Those members of a species who reproduce most effectively therefore propagate their particular variations forward, and this natural selection is the source of the diversity of species found in the world today.

Charles Darwin himself, in his book *The Expression of the Emotions in Man and Animals*, hypothesized that evolution influenced the behavior and psychology of animals as well as their physical characteristics. (Darwin, 1998, p. 355-360) More recently, starting in the 1970s, evolutionary biologists, notably Edward O. Wilson, have extended the framework of natural selection and adaptation to explain aspects of the individual and social behavior of animals, including man. (Lumsden and Wilson, 1981; Wilson 1978)

But what about the abuse of psychoactive compounds? It is undeniable that these drugs exact a terrible toll, both on individuals and on society (hence, the species) as a whole. Under a naive reading of evolutionary theory, the vulnerability to drug abuse and addiction would have created such a negative adaptation that these traits would have been bred out of mankind long ago. But clearly they have not. Therefore, there must be some more subtle effect here. Either there is some positive adaptive benefit to the mechanisms underlying the cause of drug abuse, or these mechanisms are neutral in their selective value and other factors are in play.

Part I: Emotions and Psychoactive Drugs

Unlike humans, most animals (possibly with the exception of the other great apes) are not consciously self-aware. They are not reflective nor introspective. However, animals are clearly aware of their internal physiological state (hunger, thirst, fatigue, etc.). These states bear upon the animal's fitness, both in terms of individual, short-term survival, and, when integrated over the life of the animal, its reproductive success. Hence, there are neural components in the brains of higher animals which are monitors the animal's affective state, and perceptual gestalts which represent, in a visceral and pre-cognitive way, a 'short-hand' summation of that state. We can call these states 'emotions'. Emotions stimulate an animal to avoid danger or discomfort, and lead, even impel, it to survival-oriented behavior.

It is possible to ask humans to self-report emotional state, although they may be inaccurate or intentionally deceptive in reporting. But can we determine the emotional state of an animal? Pet owners may say they can tell how their dog or cat feels, but this reporting is subject to anthropomorphism, or projecting the owners' mental state upon the animal. Is it possible to objectively determine what an animal is 'feeling'?

Dr. Jaak Panksepp of Bowling Green State University, working with other experimenters, found that rats make one kind of short ultrasonic 'squeak' during or when anticipating pleasurable experiences, such as playing with other rats or familiar experimenters, being fed, anticipating sexual intercourse, and others. (Panksepp *et al*., 2002, p. 463-464) He has also observed that rats make a distinctly different (lower pitched and longer) ultrasonic 'squeak' during or anticipating unpleasant experiences, such as defeat in fighting, the presence of predators, or when anticipating an electric shock (*ibid*). Rats appear to make these noises voluntarily, without being conditioned to do so. We may therefore argue that these represent at least a crude 'self-reporting' of the emotional state of the rats.

Crucially, rats make the 'pleasure squeak' when they have received a dose of certain psychoactive drugs, notably amphetamine and morphine. This indicates that the same mechanisms which report emotions can be subverted by the administration of psychoactive compounds. They even make these noises in anticipation of receiving a drug dose. Conversely, after rats have become addicted to opiates, during withdrawal they make the 'discomfort squeak'. We can therefor conclude that the emotional component of drug addiction is not merely a construct of human experience and higher reasoning powers, but is intrinsic to the neurological functioning of these drugs.

Part II: Co-Evolution of Neurotransmitters and Neurotransmitter Analogues

Evolution occurs slowly; many animals which are separated by hundreds of millions of years of evolutionary divergence contain the same or very closely analogous genes. These highly conserved genes code for the production, and subsequent detection or processing, of molecules which are used in the same metabolic systems in these divergent species. By the same measure, the appearance in highly *un*related organisms of identical or analogous bio-active chemicals indicates that evolution has probably shaped *both* species together, in either a cooperative or antagonistic way. Either there is some mutual benefit to both species (as well as the benefit conferred upon each species) in sharing these chemicals, or, as happens with prey and predator species, the prey evolves to create molecules which will give it an advantage against its predator, by subverting or upsetting the predator's metabolism.

The small molecules which act as neurotransmitters (NTs) in higher animals have counterparts in the plants eaten by those animals. Convergent evolution has caused the plants to create neurotransmitter analogues (NTAs) which bind to or inhibit the same receptors in the central nervous systems of their predator animals. These NTAs disrupt the normal functioning of the animal's nervous systems in ways which cause them to, at the least, learn to refrain from eating those plants, or, *in extremis*, poison the animal. (Which, of course, keeps that particular one from eating any more of the lethal plant, and may provide a negative example to other members of the species.) (See Table 1.)

Receptor	Neurotransmitter	Neurotransmitter Analogue	Source Plant or Product
nicotinic acetylcholine	Acetylcholine	Nicotine	tobacco; pituri
muscarinic acetylcholine	Acetylcholine	Arecoline Muscarine	betel nut *Amanita* mushrooms, etc.
adrenergic	Norepinephrine / Epinephrine	Ergot Alkaloids Norpseudoephedrine Cocaine	*Claviceps* fungus *Catha edulis* (khat) coca
serotonin	Serotonin	Ergot Alkaloids Mescaline	*Claviceps* fungus *Lophophora* cactus, etc.
dopamine	Dopamine	Ergot Alkaloids Cocaine	*Claviceps* fungus coca
adenosine	Adenosine	Caffeine Theophylline Theobromine	coffee tea chocolate
opioid	Endorphins	Morphine	opium
cannabinoid	Anandamide	THC	marijuana

Table 1 – “Examples of alleochemicals that interfere with mammalian neurotransmission”

(adapted from Sullivan and Hagen, 2002, p. 393)

In fact, various mammalian NT systems were first discovered and characterized by their response to the corresponding NTAs. We talk about and study 'opioid receptors', not 'endorphin receptors'. But the exact compound to which those receptors evolved a sensitivity is an endorphin, not an opioid. Science has inadvertently put the cart before the horse; animal evolution caused the receptors to be most sensitive to the endogenous NT, and plants co-evolved to create the NTAs.

If plants synthesize NTAs to disrupt animal NT receptors, is there any evolutionary evidence that animals can consume these NTAs for any useful purpose? Evolution causes defenses to evolve in prey, but also causes counter-defenses to evolve in the predators. Early human history seems to

indicate this is exactly what happened for our species. Although we have little direct evidence for the initial dates of the consumption of plants which produce NTAs, both the archaeological record and the written records of first European contacts with native cultures provides some indications. In fact, many of these historical records must be taken as 'no later than' dates for the consumption of these plant products.

There is archaeological evidence that betel nut (*Areca catechu*) was consumed approximately 13000 years ago in Timor and 10700 years ago in Thailand. Other archaeological artifacts indicate that coca (*Erythroxylum coca*) was being used in Ecuador no less than 5000 years ago. Khat (*Catha edulis*) has been used since antiquity in Ethiopia and north-east Africa. (Sullivan and Hagen, 2002, p. 390) A survey of the plants utilized by native North Americans counts 1222 used as medicines, 745 used as foodstuffs, and 679 used for both purposes. (Moerman, 1994, p. 168) Native people did not sharply distinguish between plants used for foods and plants used for 'drugs'. There was no pharmaceutical industry producing pills or injectables; natural plant products were chewed then held in the cheek, sometimes swallowed, or less frequently smoked.

Most of these 'food drugs' were stimulant in effect. They were most commonly used in environments that were harsh and resource-poor, where nutritive plants were sparse. (See Figure 1.)

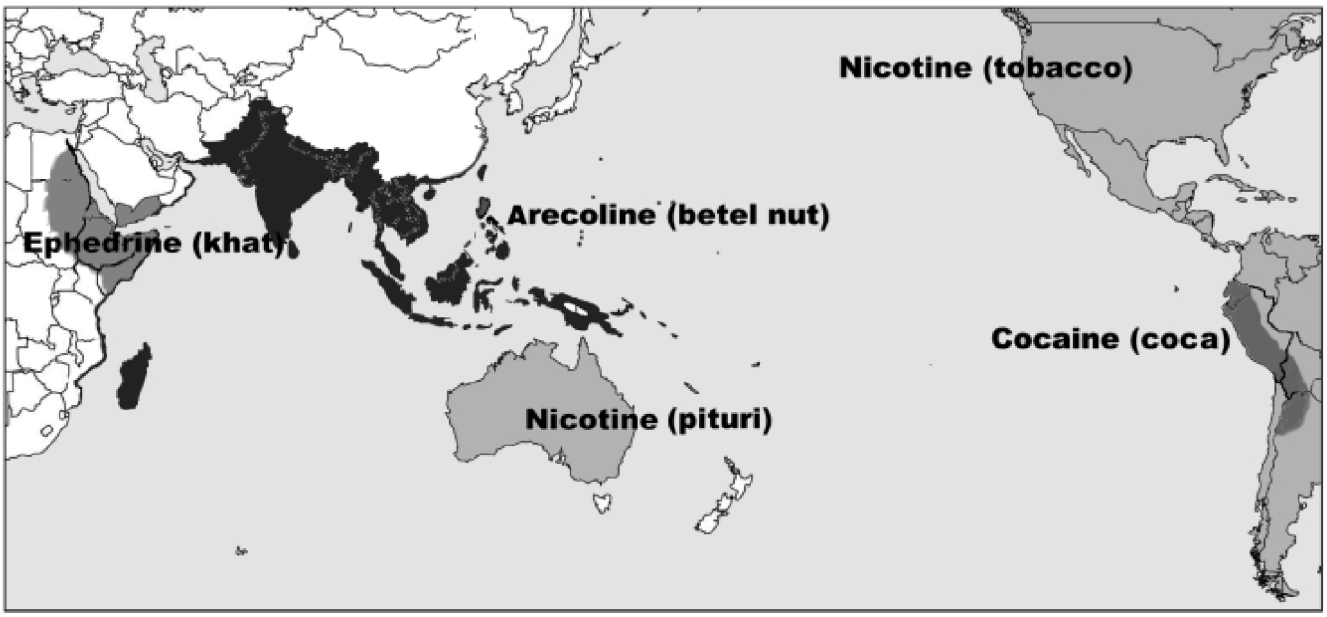

Figure 1 – "Examples of ubiquitously exploited alleochemicals in the precolonial world"

(from Sullivan and Hagen, 2002, p. 391)

Their perceived effects were interpreted the same as foods – they provided energy or relieved fatigue. (Sullivan and Hagen, 2002, p. 392) However, unlike modern refined, high-potency drugs, the NTAs available from unprocessed plants were limited in both dose per use and in the frequency of dosing. Thus, there was no evolutionary pressure to moderate their intake. Any evolved 'desirability' of these plants, or of the NTAs they provide, was not offset by any evolved 'undesirability' due to overdosing or the difficulties associated with physical addiction to them.

Part III: Non-Obvious Drivers of Addictive Behavior

John Falk, in his address upon receiving the 1997 Solvay Award, described a series of experiments performed in his lab at Rutgers. By cleverly adjusting the feeding schedule and amounts given to hungry experimental rats, they could be induced to consume almost ten times the normally expected amount of water in a three hour period. (Falk, 1998, p. 91) This 'schedule-induced excess behavior' is not limited to rats and water drinking; many species (including human beings) can be induced to polydipsia, and other behaviors, including aggression and hyperactivity, can be induced as well. These 'adjunct' behaviors are “excessive in amount and persistent over weeks and months, as long as the generator-schedule conditions remain in force.” (*ibid*, p. 92)

Now these are strange results indeed. At first glance, excessive and persistent behaviors which are not linked to useful functions strongly resemble the behavior of drug addicts. But similarity does not mean equivalence, although it may give some insight into further experiments to perform. Dr. Falk goes on to use a modification of the original setup, giving the rats a choice of water or a bitter solution of lidocaine. As expected, the rats preferred the water. But if the lidocaine solution was made more attractive by adding glucose and sodium saccharin, the rats would preferentially drink that, in the 'expected' excess. Further, if the concentration of sweeteners was slowly faded out over several weeks, the rats continued to prefer the lidocaine over the water, and continued to drink in excess of their needs. Even more bizarrely, a third experiment gave the rats a choice between cocaine and lidocaine solutions. When the rats had been 'trained' (via the decreasing sweetener method described above) to prefer lidocaine, they stuck with that preference. (See Figure 2.)

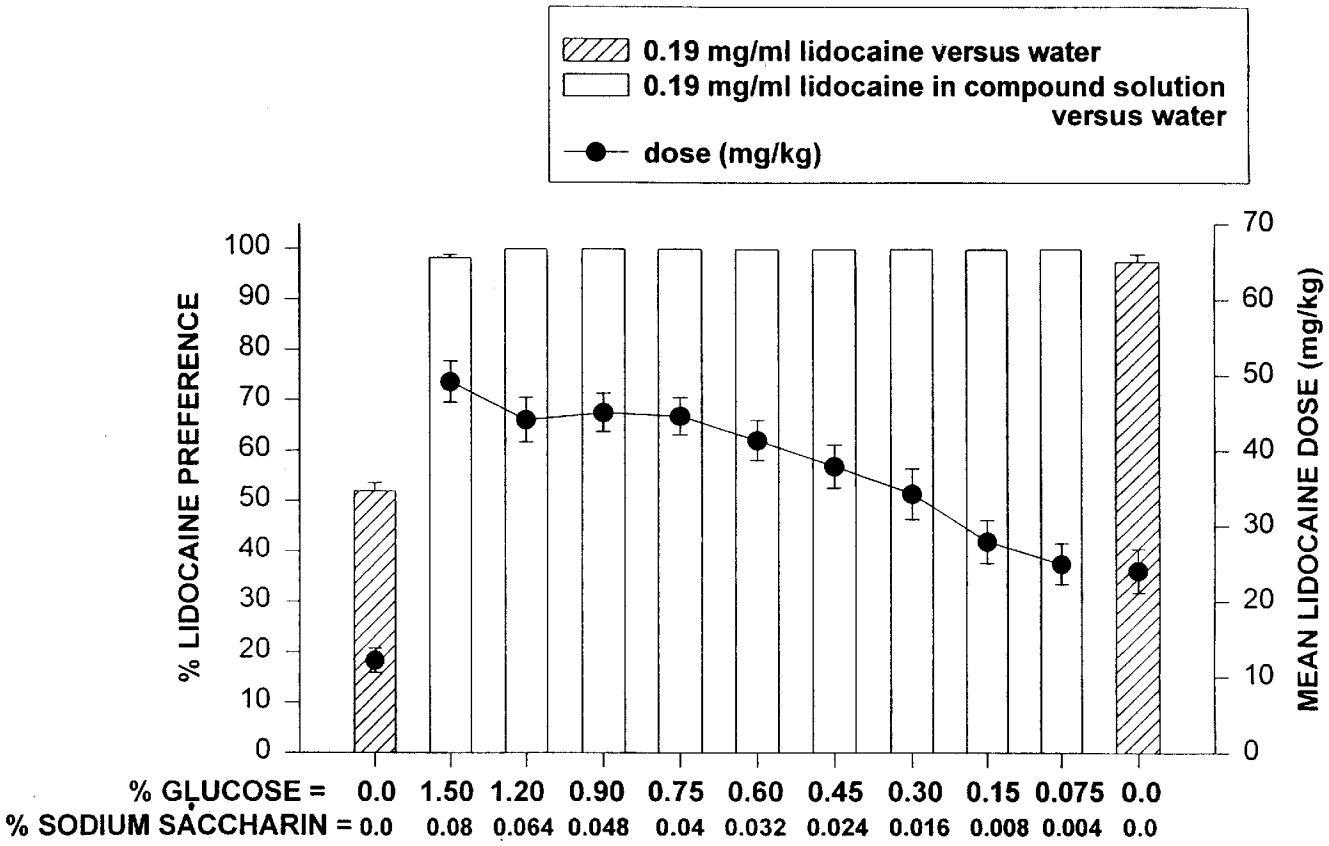

Figure 2 – Initial preference for lidocaine solution, and time change of preference for lidocaine solutions when delivered with decreasing amounts of sweeteners.

(from Falk, 1998, p. 94)

Lidocaine is not psychoactive, nor is it palatable unless highly sweetened. The only conclusion that can be drawn is that the rats' behavior is being mostly determined as an 'adjunct' to the feeding schedule.

We can expect that this has implications for human behavior surrounding drug abuse also. Psychoactive drug taking in humans is nearly always a social activity performed with others. Not only is there the psychological effect of the drug to consider, but the social context, including any stereotypical behaviors or 'rituals' associated with the acquiring, preparing, or consuming the drug. As

Falk succinctly puts it, “[T]he world of drug abuse contains a host of reinforcers associated with drugs that are **independent** of the pharmacological effects of the abused drug.” (emphasis added. Falk, 1998, p. 95)

Part IV: A Comprehensive Explanation of Drug Abuse

Tying together these several observations, we can now propose a comprehensive theory of human psychoactive drug abuse that is consistent with evolutionary theory and the evidence at hand. This theory can be summarized as follows:

I. Animals and plants have co-evolved related compounds (neurotransmitters and neurotransmitter analogues (NTAs)) which function to mediate or disrupt the activity of the central nervous system. Humans in particular, especially in environments where food is scarce, have evolved a desire to search for and consume plants containing certain NTAs as an alternative or supplement to food plants, because the perceived effects of the NTAs are energizing or fatigue-reducing. Additionally, some plant NTAs have pain-reducing or sedative effects, which are desirable to relieve environmental stress and discomfort. Therefore, these desires confer a positive evolutionary advantage to the humans possessing them. In this early environment, the doses of NTAs available from plants were limited in both quantity and frequency, so there was no counter-balancing evolutionary pressure to create any 'limits' to the desirability of these plants.

II. Humans, like other social animals, communicate their emotional (affective) state to each other. Both positive and negative affects, caused by the consumption of NTA-containing plants, provide important clues to other members of the immediate social group to influence their desire to also consume these plants. We reinforce each others' behavior by communicating the emotional result of our own behavioral choices.

III. Humans, like other animals, can be trained in so-called 'adjunctive' behaviors, that is, one can be trained to perform a series of behaviors, which by themselves might be avoided, by merely adjusting the correct intermittent schedule of another, time-related

behavior. In simple terms, humans can be trained (or train themselves) to do an undesirable X when that happens in conjunction with a highly desirable Y at the right periodic schedule.

Therefore, the worst effects of modern psychoactive drug abuse occur when large quantities of high-potency NTAs are readily available in a environment which simultaneously provides both social promotion and 'schedule-induction' to reinforce their use.

Conclusion: Evolutionary Evidence Is Both Dismaying and Hopeful

Correcting 'the drug abuse problem' will involve modification of both personal (individual) and social (collective) behaviors. Because there has been evolutionary adaptation towards (limited) drug taking, "just say no" cannot be very effective. Instead, treatments should focus (after any necessary 'de-tox') on individuals being 'retrained' to avoid the adjunctively-reinforcing behavioral contexts they find themselves in. Former drug abusers will have to be made aware of, or sensitized to, alternative methods of altering their emotional state in the desired direction which are socially acceptable and psycho-physiologically safer. As a society, we will need to be aware of how we may have (inadvertently or unconsciously) built up psycho-social environments that reinforce adjunctive behavior, including illicit (financial and social hierarchical) rewards for producing and supplying drugs. We will want to build environments that reinforce (adjunctively or not) the desired positive behaviors.

This will not be easy, and will probably not be cheap. Intervention for individuals will take concentrated, long-term assistance from highly trained (and hopefully well-compensated) practitioners. Intervention for the entire society will take mass education, mobilization of the money and political will necessary to effect the required social changes, and again, long-term follow-through to measure the impact of these efforts, and to improve them where necessary. But if the alternative is either to let individuals suffer from their drug abuse, and inflict the indirect cost (in crime and social disorder) on all of us, or to flail around with ineffective attempts at solutions that do not respect the evolutionary basis of drug abuse as outlined here, we will be simply wasting our time, money, and priceless human capital.

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