

Bufo alvarius: a potent hallucinogen of animal origin

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Abstract

Anthropologists have long speculated that ancient peoples of Mesoamerica used a toad, *Bufo marinus*, as a ritual intoxicant. This hypothesis rests on many iconographic and mythological representations of toads and on a number of speculative ethnographic reports. The authors reject *B. marinus* as a candidate for such use because of the toxicity of its venom. A more likely candidate is the Sonoran desert toad, *Bufo alvarius*, which secretes large amounts of the potent known hallucinogen, 5-methoxy-*N,N*-dimethyltryptamine (5-MeO-DMT). The authors demonstrate that the venom of *B. alvarius*, although known to be toxic when consumed orally, may be safely smoked and is powerfully psychoactive by that route of administration. These experiments are the first documentation of an hallucinogenic agent from the animal kingdom, and they provide clear evidence of a psychoactive toad that could have been employed by Precolumbian peoples of the New World.

Key words: *Bufo*; Hallucinogens; Tryptamine; Toad; Maya

1. Introduction

In the worldwide distribution of hallucinogens there is a pronounced and significant discrepancy that has attracted the attention of numerous authorities (Schultes, 1963; La Barre, 1970; Schultes and Hofmann, 1980). Of the 200 or more psychoactive plants that have been identified worldwide, close to 90% are native to the Americas; the Old World has contributed perhaps 20 (Schultes and Hofmann, 1979; Schultes and Farnsworth, 1980). How might this be explained?

In part these figures may be an artifact of the emphasis of academic research. A good many of these plants have entered the literature due to the efforts of Richard Evans Schultes and his colleagues at the Harvard Botanical Museum and elsewhere, and their interest has predominantly been in the New World. Yet, were the hallucinogenic plants a dominant feature of traditional cultures in Africa and Eurasia, surely they would have shown up in the extensive ethnographic literature and in the journals of traders and missionaries. With few notable exceptions, they do not.

Nor is this discrepancy due to floristic peculiar-

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ities. The tropical rainforests of equatorial Africa and Southeast Asia, in particular, are exceedingly rich and diverse. Moreover, the peoples of these regions have most successfully exploited them for pharmacologically active compounds for use both as medicines and poisons. In fact, as much as any other material trait, the manipulation of toxic plants remains a consistent theme throughout sub-Saharan Africa. The Amerindian, for their part, were certainly no strangers to plant toxins which they commonly exploited as fish, arrow and dart poisons. Yet it is a singular fact that while the peoples of Africa consistently used these toxic preparations on each other, the Amerindian almost never did. And while the Amerindian successfully exploited the natural world for hallucinogens, the African, with few noted exceptions, did not. This suggests the critical fact that the use of any pharmacologically active compound — remembering that the difference between hallucinogen, medicine and poison is often a matter of dosage — is firmly rooted in culture (La Barre, 1970; Weil, 1972, 1980; Davis, 1985, 1992). The ingestion of psychotropic plants represents but one means of satisfying a universal human desire to experience altered states of awareness. If the peoples of Africa did not explore their environment for psychoactive drugs, surely it is because they had discovered through trance and spirit possession an alternative vehicle for transformation (Davis, 1988a).

If culture accounts in part for the geographical range of psychotropic plants, it fails to explain yet another enigma concerning the biological distribution of hallucinogens. To date, all known and deliberate human use of natural hallucinogens has involved derivatives of higher plants and fungi. The bacteria, algae, lichens, bryophytes, ferns and gymnosperms are notably lacking in psychoactive properties. Moreover, no hallucinogenic agent has yet been found in the animal kingdom.

2. Previous reports of animal hallucinogens

To be sure, there have been numerous scattered reports of psychotomimetics derived from animals. Britton (1984) cites an early 19th century travel account from eastern Brazil that suggests that the

Malalis Indians may have used *bichos de tacuara*, the larvae of a moth, tentatively identified as *Myelobia smerintha*, as an hallucinogen. La Barre (1981) refers in passing to a narcotic bamboo grub of Amazonian South America, an hallucinogenic 'dream fish,' *Kyphosus fuseus* [sic], of Melanesian Norfolk Island, and a black and red *oconenetl* bird from Tlaxcala, Mexico, whose flesh is reputedly hallucinogenic. Hoffer and Osmond (1967) also refer to an hallucinogenic fish, the silver drummer fish, found in the waters off Norfolk Island. Ichthyooallieinotoxism, or hallucinogenic fish poisoning, has been reported from the tropical Pacific and Indian oceans (Helfrich and Banner, 1960; Halstead, 1978). Several species in two families have been implicated including two species of mullet, *Mugil cephalus* and *Neomyxus chaptalli*, and two species of goatfish, *Mulloidichthys samoensis* and *Upeneus arge* (Helfrich and Banner, 1960). Finally, Carneiro (1970) noted the use of an unidentified frog in the hunting magic of Peruvian Indians in the northwest Amazon.

The Brazilian report, though provocative, is based strictly on hearsay. No voucher specimens have verified the identity of the moth, no chemical analysis has been undertaken, and, in the original report, the correspondent did not observe anyone experiencing psychoactive effects (Saint-Hilaire, 1824). Saint-Hilaire's account is noted in the widely read *Handbook of South American Indians* (Cooper, 1949) and La Barre's 'narcotic bamboo grub' may well be a reference to the same moth larvae. When recently contacted, La Barre could not recall the provenance of his references to either the grub or the purportedly hallucinogenic *oconenetl* bird (La Barre, pers. commun., 25 January 1992).

Hallucinogenic fish poisoning has been reported from South Africa, Norfolk Island and Hawaii (Jordan et al., 1927; Smith, 1953; Van Pel, 1959). In Hawaii the fish are apparently toxic in parts of Kauai, primarily in the Anini area, but spreading from Pilaa to Haena and around Molokai, in the Pilaa region. The toxin evidently is found in the heads of the fish which are considered poisonous only in the months of June, July and August. According to one early report 40 Japanese labourers became delirious and 'mentally paralyzed' after eating *weke pahala*, *Upeneus arge*, a goat-

fish also known by the vernacular name 'nightmare weke' (Jordan et al., 1927). On Molokai, local fishermen maintain that both the head and tail of these fish cause vivid dreams which are not necessarily undesirable (Lavinia Currier, pers. commun., 1992). Similar effects are apparently induced by the 'dream fish' from Norfolk Island (probably *Kyphosus vaigiensis*). Those who eat this fish before sleeping reputedly suffer terrifying nightmares (Helfrich and Banner, 1960).

Whether these fish are truly hallucinogenic and whether indigenous peoples have deliberately sought out the intoxication and interpreted it in culturally meaningful ways remains unknown. In most instances, the symptoms of the intoxication — dizziness, loss of equilibrium, partial paralysis of the legs, an itching or burning sensation in the throat, hallucinations and mental depression, delirium, a subjective perception of imminent death — appear to be highly unpleasant and difficult to distinguish from poisoning. The chemistry and pharmacology of the phenomenon remain unknown and attempts to replicate the intoxication in controlled experiments have failed (Helfrich and Banner, 1960; Halstead, 1978). From the isolated reports, it appears that the biointoxication is sporadic and unpredictable in its occurrence. Evidently, many of those who have experienced hallucinogenic fish poisoning have done so quite inadvertently, whilst seeking out fish that under most circumstances are perfectly edible.

The Peruvian frog has recently been identified as *Phyllomedusa bicolor*, and analysis of its secretions has revealed the presence of a number of vasoactive and neuroactive peptides (Daly et al., 1992). Amahuaca and Matsigenka Indians along the border of Peru and Brazil collect and dry the skin secretions of this frog, then mix them with saliva and introduce the mixture into lines of fresh burns on the arms or chest, producing a rapid, violent intoxication. Although behavioral effects are reported, it remains to be proved that this species is truly hallucinogenic.

3. *Bufo marinus*: hallucinogen or poison?

Of all the possible animal hallucinogens, none have excited more interest than *Bufo marinus*. An-

thropologists have long speculated that ancient peoples of Mesoamerica may have used this toad as a ritual intoxicant (Coe, 1971; Furst, 1972, 1974, 1976; Dobkin de Rios, 1974; Knab, 1974; Cooke, 1979, 1981; Hamblin, 1979; Kennedy, 1982). The *Bufo marinus* hypothesis rests on many iconographic and mythological representations of toads and on a number of speculative ethnographic and ethnohistoric reports (Tozzer and Allen, 1910; Wassén, 1934a,b; Thompson, 1958, 1970; Carneiro, 1970; Furst, 1972, 1976; Dobkin de Rios, 1974; Knab, 1974; Kennedy, 1982). In addition *B. marinus* bones dominate the amphibian component of the faunal remains at a number of Classic, Late Classic and Postclassic Maya sites and have often been found in ritual contexts (Pollack and Ray, 1957; Olsen, 1972, 1978; Hamblin, 1979, 1984; Wing and Steadman, 1980; Wing and Scudder, 1991). The concentration and distribution of *B. marinus* remains at San Lorenzo led one prominent archaeologist to suggest that the Olmec civilization may have used the toad as a narcotic (Coe, 1971). Finally proponents cite a single experiment in the medical literature suggesting that one ingredient of *B. marinus* venom is psychoactive (Fabing and Hawkins, 1956).

That compound, 5-hydroxy-*N,N*-dimethyltryptamine (5-OH-DMT), also known as bufotenine, has been reported as a constituent of well-known hallucinogenic snuffs from northwest South America, derived from the leguminaceous tree, *Anadenanthera peregrina* (Altschul, 1972). However, the venom glands of the toad also produce toxic cardiac glycosides: bufogenin and bufotoxin (Daly and Witkop, 1971; Deulofeu and Rúveda, 1971; Meyer and Linde, 1971). Both are highly toxic (Abel and Macht, 1911; Chen and Jensen, 1929). Mere topical exposure to the crude venom (from handling toads, for example) may result in severe headache, nausea and violent vomiting (Allen and Neill, 1956). A recent attempt on the part of a young man to experience hallucinogenic effects from the venom resulted in seizures (Pulling, 1990). He had taken the venom orally by touching the glands and then licking his fingers. It is likely that ingesting a straight maceration of the parotoid glands would cause death by cardiac failure or respiratory arrest before the recipient would

get a chance to experience any useful states of consciousness induced by bufotenine (Alger, 1974). To date, no one has shown how the poisonous elements in the venom could be neutralized to allow human users to experience the putative hallucinogenic properties. Furthermore, many authorities doubt that bufotenine is actually psychoactive (Turner and Merlis, 1959; Holmstedt and Lindgren, 1967; Chilton et al, 1979; Schultes and Hofmann, 1980).

Virtually every report that characterizes bufotenine as a psychotomimetic dates to a single experiment completed by a medical doctor, Howard Fabing, in the 1950s. Fabing obtained permission to inject bufotenine intravenously into a number of inmates at the Ohio State Penitentiary. The recipient of the mildest dose complained of nausea, prickling sensations in the face, and slight difficulty in breathing. With higher dosage these symptoms became more pronounced and the subject's face and lips became purplish. The final dose caused mild hallucinations and delirium, and the skin turned 'the colour of an eggplant'. The hallucinations were ephemeral. Three minutes after injection, the subject vomited and 'saw red spots passing before his eyes and red-purple spots on the floor. Within 2 min, these visual phenomena were gone, but they were replaced by a yellow lens filter' (Fabing and Hawkins, 1956). That is the extent of hallucinations experienced by any of the recipients of the bufotenine injections.

Later investigators attempted but failed to replicate these results. Harris Isbell, a researcher at the Public Health Service Hospital in Lexington, Kentucky, experimented with bufotenine as a snuff. Neither inhalation of pure bufotenine in aerosol suspension, or oral ingestion of bufotenine in doses as high as 100 mg elicited any psychoactive effect (Holmstedt and Lindgren, 1967). Turner and Merlis (1959) tried injecting bufotenine intramuscularly. They noted that with a dose of 40 mg, the recipient 'suddenly developed an extremely rapid heart rate; no pulse could be obtained; no blood pressure measured . . . onset of auricular fibrillation . . . extreme cyanosis developed'. Resuscitative procedures were immediately implemented, and, fortunately, the pulse returned to normal (Chilton et al., 1979). After the failure of

this and other experiments the investigators concluded that 'we must reject bufotenine as capable of producing the acute phase of cohoba (*Anadenanthera peregrina*) intoxication' (Chilton et al., 1979, p. 64).

This conclusion is supported by other experimental evidence. One measure of the ability of compounds to penetrate the nervous system is lipid solubility. Gessner and Page (1962) showed that bufotenine has a very low lipid solubility and is relatively incapable of crossing the blood–brain barrier, making it unlikely that the drug would have any effect on the central nervous system. Therefore, even assuming that a folk preparation could eliminate the toxic constituents in *Bufo marinus* venom, it is very doubtful that bufotenine itself is hallucinogenic.

4. *Bufo alvarius*: a potent and proven hallucinogen from the Sonoran Desert

We think it highly unlikely that *B. marinus* could, under any circumstances now or in the past, be employed as a psychoactive drug (Davis, 1988b; Davis and Weil, 1992). If the ancient civilizations of Mesoamerica did have a toad-based hallucinogen it may well have come from another species, whose psychoactivity has, with one exception (Furst, 1972, 1976), been overlooked by anthropologists. We report here the definite psychoactivity of *Bufo alvarius*. It is, as far as we can ascertain from the literature, the first proven instance of the use of an hallucinogenic agent obtained from an animal source.

Bufo alvarius, the Sonoran Desert toad, is a semi-aquatic amphibian found only in the Sonoran Desert, an area of approximately 120 000 square miles that reaches from southeastern California across the southern half of Arizona and south approximately 400 miles into Mexico. For most of the year, from September to April, the toads remain underground in a dormant state. During the breeding season, which coincides with summer rains, they are highly active at night, and the desert comes alive with thousands of the animals (Wright and Wright, 1949; Stebbins, 1985).

One of more than 200 species of *Bufo*, the Sonoran toad is a large amphibian, and like *B.*

marinus it has prominent parotoid glands that secrete a viscous milky-white venom. The two species are morphologically similar and in iconographic representations would be impossible to distinguish. Their secretions, however, are very different. Toad venom is biochemically complex, with particular combinations of constituents peculiar to each species. *Bufo alvarius* is unique within the genus in its possession of an unusual enzyme, *O*-methyl transferase, which, among other reactions, converts bufotenine (5-OH-DMT) to the potent hallucinogen 5-methoxy-*N,N*-dimethyltryptamine (5-MeO-DMT). In fact, the activity of this enzyme leads to the production and accumulation of enormous amounts of 5-MeO-DMT, up to as much as 15% of the dry weight of the parotoid and tibial glands (Erspamer et al., 1965, 1967; Cei et al., 1972).

One of the most powerful hallucinogens known from nature, 5-MeO-DMT accounts for much of the psychoactivity of South American snuffs derived from *Anadenanthera peregrina* as well as those derived from various species of *Virola*, a genus of trees in the nutmeg family (Holmstedt and Lindgren, 1967; Schultes and Hofmann, 1980). In the plant kingdom it usually occurs together with *N,N*-dimethyltryptamine (DMT). Orally inactive due to the activity of an enzyme in the human gut (monoamine oxidase), these compounds are usually smoked and rarely injected. They may be ingested orally if taken in combination with monoamine oxidase inhibitors, as in the case of certain sophisticated indigenous preparations reported from the northwest Amazon (McKenna et al., 1984a,b). Both DMT and 5-MeO-DMT are easily synthesized compounds that appeared as recreational psychedelics in the American drug subculture during the 1960s. DMT is a controlled substance under Federal law, but its 5-methoxy derivative is not. Some chemical supply houses sell 5-MeO-DMT, and supplies are occasionally diverted to human users.

The first published analysis of the venom of *B. alvarius* appeared in 1965 and a more comprehensive study came out in the *Journal of Pharmacology* in 1967 (Erspamer et al., 1965, 1967). The research was later reported in a book on the evolution of the genus *Bufo* (Blair, 1972). These publications

probably inspired experimentation with the venom of *B. alvarius* that led to the appearance in 1984 of an underground pamphlet titled '*Bufo Alvarius*, the Psychedelic Toad of the Sonoran Desert' (Most, 1984). This pamphlet gave detailed instructions for collecting and drying the venom:

Fresh venom can easily be collected without harm to the toad. Use a flat glass plate or any other smooth, non-porous surface at least 12-inches square. Hold the toad in front of the plate, which is fixed in a vertical position. In this manner, the venom can be collected on the glass plate, free of dirt and liquid released when the toad is handled.

When you are ready to begin, hold the toad firmly with one hand and, with the thumb and forefinger of your other hand, squeeze near the base of the gland until the venom squirts out of the pores and onto the glass plate. Use this method to systematically collect the venom from each of the toad's granular glands: those on the forearm, those on the tibia and femur of the hind leg, and, of course, the parotoids on the neck. Each gland can be squeezed a second time for an additional yield of venom if you allow the toad a one-hour rest period. After this the glands are empty and require four to six weeks for regeneration.

The venom is viscous and milky-white in color when first squeezed from the glands. It begins to dry within minutes and acquires the color and texture of rubber cement. Scrape the venom from the glass plate, dry it thoroughly, and store it in an airtight container until you are ready to smoke it. (Most, 1984: 10–12)

These instructions are remarkable in view of the known toxicity of the Sonoran Desert toad (Allen and Neill, 1956). There are many instances, for example, of dogs being poisoned after mouthing the animal. In one case an owner reported that he was able to remove the toad from his dog's mouth within 10 s. Nevertheless, after 30 min, the dog began to salivate profusely, quickly went into convulsions, and died, apparently in respiratory arrest. Human morbidity has also been reported. In 1986 a 5-year-old boy with profuse salivation and continuous seizures was admitted to the University of Arizona Medical Center; seizure activity had begun within 15 min of his licking a toad, later identified as *Bufo alvarius*. The child survived, but it took a full week for him to return to normal (Hitt and Ettinger, 1986).

Since 1987 one of us (Andrew T. Weil) has interviewed a number of informants in southern Arizona who claim to have safely smoked toad venom and experienced positive psychoactive ef-

fects. No one reported toxicity. Based on these interviews, we hypothesized that smoking selectively denatures the toxic constituents. Therefore, we felt confident in initiating a series of self experiments with venom obtained from the parotoid glands of Sonoran Desert toads collected in Pima County, Arizona. We later repeated the experiments with venom that had been collected 2 years previously in Gila County, Arizona and stored in a closed vial at room temperature. The results of these experiments are noteworthy.

Single deep inhalations of vaporized venom proved powerfully psychoactive within 15 s. Consistent with the known effects of 5-MeO-DMT, the intoxication was intense and short-lived, marked by auditory and visual hallucinations. The strongest effects dissipated after 5 min, but residual changes in perception persisted for 1 h. No toxic effects were experienced during or after the experiments. The 2-year-old venom was equally active.

One *Bufo alvarius* toad yields 0.25–0.5 g of dried venom. Since concentrations of 5-MeO-DMT may be as high as 15%, one toad may yield 75 mg of an hallucinogenic drug that, when smoked, is effective in humans at doses of 3–5 mg. In other words, a single toad produces 15 or more doses of one of the most potent psychoactive drugs found in nature. A matchbox-sized container would represent thousands of effective doses.

These experiments provide clear evidence of the existence of a psychoactive toad that could have been employed by pre-Columbian peoples of the New World. The implications for anthropology and Mesoamerican archaeology in particular are significant and are the subject of a longer report (Davis and Weil, 1992). In the meantime we offer this review as the first documentation of the use of an hallucinogenic agent from the animal kingdom.

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