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Natural hallucinogens of fungal and animal origin: action and potential applications — a narrative review

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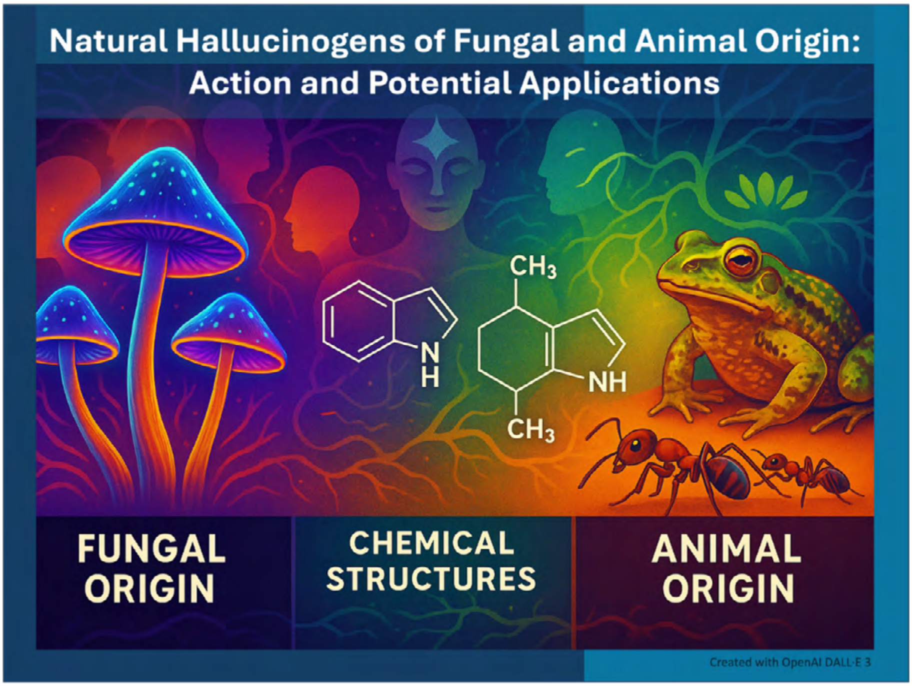
Abstract: Introduction: Natural hallucinogens derived from fungi and animals have been used for centuries in shamanic, ritualistic, and medicinal practices across diverse cultures. These compounds exhibit a wide range of structures and mechanisms of action, affecting various neurotransmitter systems pathways. Fungal hallucinogens, primarily indole alkaloids like psilocybin and ergot alkaloids, as well as animal-derived toxins, such as bufotenine, ciguatoxins, or semiochemicals from insects, can induce profound alterations in perception, cognition, and mood. Despite their traditional use and psychoactive effects, many of these substances remain underexplored in terms of pharmacology and therapeutic potential. Recent studies suggest their possible roles in treating neuropsychiatric disorders, inflammatory conditions, and chronic pain, highlighting the need for a systematic review of their biological activity and medical applications.
Aim of the study: This review aims to provide an overview of hallucinogenic compounds of fungal and animal origin, focusing on their chemical nature, pharmacodynamic properties, and current evidence for potential therapeutic use.
Methodology: The review was based on publications retrieved from databases such as PubMed, Google Scholar, and ScienceDirect, covering the period from 1983 to 2025. Search terms included: fungal hallucinogens, animal-derived psychedelics, natural psychoactive compounds, toxicity, therapeutic application of hallucinogens, and psychedelic drug research.
Results: The analyzed hallucinogens differ markedly in terms of chemical structure, receptor activity, intensity of hallucinogenic effects, and potential for clinical use. Preclinical and limited clinical data suggest beneficial effects in mood and anxiety disorders, treatment-resistant depression, pain syndromes, and potentially neurodegenerative diseases. Some compounds show promise as leads for the synthesis of novel bioactive molecules.

Conclusions: Hallucinogens of fungal and animal origin represent a biologically diverse and pharmacologically rich group of natural substances. Further interdisciplinary research is required to explore their mechanisms of action, safety profiles, and therapeutic potential. Their continued investigation may lead to the development of innovative treatments in neuropsychiatry and beyond.

Graphical Abstract



Keywords: mushrooms-derived hallucinogens, animal-derived hallucinogens, drugs, substance abuse, toxicology, therapeutic potential.

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Introduction

Hallucinogens of natural origin have long fascinated both traditional cultures and modern science because of their profound effects on perception, cognition, and consciousness. This review examines two major groups of naturally occurring hallucinogens, fungal and animal, examining their chemical diversity, mechanisms of action, ethnomedical uses, and psychotropic profiles. Mushroom sources such as *Psilocybe*, *Amanita*, and *Claviceps* species are rich in indole alkaloids and isoxazoles, which primarily modulate serotonergic and GABA-ergic signaling. Hallucinogens of animal origin, from amphibian skin secretions and insect toxins to marine alkaloids and ichthyotoxins, exhibit a broader spectrum of molecular targets, including ion channels, cholinergic system, and serotonin receptors. In addition to their cultural and toxicological significance, these compounds represent a largely untapped reservoir of pharmacologically active molecules. Recent advances in neuropharmacology and psychedelic-assisted therapy have renewed interest in their therapeutic potential, particularly for neuropsychiatric and mood disorders. Furthermore, the structural uniqueness of many animal toxins and marine alkaloids underscores their value as lead compounds in drug discovery. This review highlights the need for further interdisciplinary research on natural hallucinogens as both biomedical tools and sources of new bioactive structures.

Hallucinogenic Fungi

A broad group of psychotropic fungi comprises approximately 200 species belonging to the phylum *Basidiomycota* (ca. 20 genera) and *Ascomycota* (genera *Claviceps* and *Cordyceps*). These fungi are found on all continents except Antarctica, growing at elevations ranging from sea level to high-altitude regions up to 4,000 meters above sea level. The genus *Psilocybe* is the most widespread among hallucinogenic fungi globally, accounting for approximately 50% of the species found in subtropical, mesophytic, and humid environments. The greatest diversity of *Psilocybe* species is observed in South America and Mexico (59 species), North America (18 species), as well as the Caribbean and Oceania [1]. According to Guzmán *et al.* [2], psychoactive fungi can be categorized into four groups based on their active compounds and psychotropic mechanisms of action:

- **Tryptamine-containing fungi**, which act as agonists at various serotonin receptors (5-HT_{2A}, 5-HT_{1A}, 5-HT_{2C}) and ion channels. This group includes *Psilocybe*, *Panaeolus*, *Gymnopilus*, *Copelandia*, *Agrocybe*, *Hypholoma*, *Galerina*, *Gerronema*, *Pluteus*, *Inocybe*, *Conocybe*, *Panaeolina*, and *Amanita* species.
- **Fungi containing isoxazole amino acids**, which are agonists of $GABA_A$ ionotropic receptors, e.g., *Amanita* species.
- **Fungi containing ergot-type indole alkaloids**, which exhibit multifaceted pharmacological activity as partial agonists at serotonin (5-$_{HT2}$), adrenergic (α_{1A}/α_2), and dopaminergic (D_2) receptors, such as *Claviceps* and *Cordyceps* species.
- **Fungi containing indole alkaloids of incompletely understood mechanisms of action**, including *Boletus*, *Heimiella*, *Russula*, and certain *Gasteromycetes* species.

Tryptamines Found in Fungi

Tryptamines isolated from fungi include psilocybin (found in *Psilocybe*, *Panaeolus*, *Gymnopilus*, *Copelandia*, *Agrocybe*, *Hypholoma*, *Galerina*, *Gerronema*, *Pluteus*, *Inocybe*, *Conocybe*, and *Panaeolina*), baeocystin and norbaeocystin (present in *Conocybe cyanopus*, *C. smithii*, *Panaeolus cyanescens*, *Inocybe* spp., and *Psilocybe* spp.), aeruginascin (*Inocybe aeruginascens*), and bufotenin (reported in *Amanita citrina*, *A. porphyria*, and *A. rubescens*) [3].

Psilocybin, the best-characterized compound among hallucinogenic mushrooms (commonly referred to as "magic mushrooms"), was discovered between 1955 and 1958 by Albert Hofmann, who studied *Psilocybe cubensis* and *P. mexicana*. However, the use of psilocybin-containing fungi predates this discovery by millennia and paleontological evidence suggests their consumption by hominins during the Pliocene epoch (approximately 5.3 million years ago) [4]. One of the oldest presumed depictions of their use is a cave painting in Algeria (circa 7000–9000 years old), featuring mushrooms preliminarily identified as *Psilocybe mairei*. *P. cubensis* and *P. mexicana* have been used ritually for thousands of years by Mesoamerican Indigenous peoples in fresh, dried, or decocted forms. Their ceremonial use is documented in sources such as the 14th-century codex *Yuta Tnoho* [3].

Psilocybin is a serotonin-like tryptamine derivative containing a phosphorus atom. After oral ingestion, it is dephosphorylated in the stomach or intestines into the active metabolite psilocin, which is responsible for its psychedelic effects [5, 6]. Due to its structural similarity to serotonin, psilocin acts primarily as an agonist at 5-HT_2 receptors, especially 5-HT_{2A} and 5-HT_{2C}, enhancing serotonergic activity in the prefrontal cortex and intensifying sensory experiences. It functions in a manner similar to LSD, although it is approximately 100 times less potent [5, 6].

Following oral administration (typically as dried or fresh mushrooms), effects begin within 15–40 minutes, peak after 60–90 minutes, and last approximately 6 hours. Psilocybin is not associated with physical dependence, although psychological dependence is possible [5, 7]. One gram of dried mushrooms contains about 12 mg of psilocybin. Typical recreational doses range from 10–50 g of fresh mushrooms (1–5 g dried), corresponding to 10–50 mg of psilocybin [5, 7]. The estimated lethal dose is approximately 6 g of pure substance (equivalent to about 0.5 kg of dried or 5 kg of fresh mushrooms) [6].

Although classified as a hallucinogen, psilocybin typically does not induce classical hallucinations but rather alters perception of reality, for instance, stationary objects may appear to move. The compound affects mood, cognition, and sensory processing [7]. Common effects include mood elevation, euphoria, introspection, mystical-type experiences, synesthesia, and distortions in time and spatial perception [5, 7]. Adverse effects may include anxiety, confusion, derealization, paranoia, and vegetative symptoms such as nausea, vomiting, and diarrhea (more frequent with whole mushrooms than with pure psilocybin) [5, 6]. Physiological symptoms such as mydriasis, tachycardia, or elevated blood pressure may result from co-occurring sympathomimetic phenylethylamines [5, 6].

Despite its low toxicity and absence of physical dependence, prolonged use of psilocybin can lead to hallucinogen persisting perception disorder (HPPD). Symptoms of HPPD may include geometric visual hallucinations, false motion perception in the peripheral visual field, color flashes, intensified colors, trailing of moving objects (palinopsia), positive afterimages, halos around objects, macropsia, and micropsia. These symptoms may persist for months or even years [6, 8]. Nonetheless, emerging research highlights the therapeutic potential of psilocybin, particularly in the treatment of depression [5].

Isoxazole Alkaloids Found in Fungi

Isoxazole alkaloids identified in fungi include muscimol, ibotenic acid, and muskazone. These compounds have been isolated from *Amanita* species, particularly *Amanita muscaria* (fly agaric) and *A. pantherina* (panther cap), but also from *A. gemmata*, *A. regalis*, *A. cothurnata*, and *A. frostiana* [9]. Muscimol is formed through the decarboxylation of ibotenic acid in the stomach, liver, and brain, whereas muskazone, a weaker compound, results from UV-induced degradation of ibotenic acid [10].

Muscimol, the principal psychoactive agent in *A. muscaria* and *A. pantherina*, is a 5-aminomethyl derivative of 3-hydroxyisoxazole and a structural analog of GABA moelcule. It acts as a potent $GABA_A$ receptor agonist, enhancing inhibitory neurotransmission in the central nervous system [11]. Its precursor, **ibotenic acid**, is a potent agonist of NMDA and metabotropic glutamate receptors (mGluR), producing excitatory effects [12]. A fresh fruiting body of *A. muscaria*

(50–70 g) typically contains approximately 70 mg of ibotenic acid and 6 mg of muscimol [13]. Ibotenic acid readily converts into muscimol during the drying process and becomes pharmacologically active after ingestion in the acidic environment of the stomach [14].

The clinical presentation of isoxazole intoxication is highly variable due to the opposing actions of ibotenic acid (stimulant) and muscimol (depressant) and depends on the species consumed. *A. muscaria* generally contains more ibotenic acid than *A. pantherina*. In patients poisoned with *A. muscaria*, disorientation (26/32 cases) and agitation (20/32) were more frequently observed, while *A. pantherina* was more often associated with coma (5/17 vs. 2/32; $p = 0.03$) [15]. The typical intoxication syndrome includes alternating phases of sedation and excitation with hallucinations. Excitatory symptoms begin 1–4 hours post-ingestion and include paresthesias, dizziness, ataxia, floating sensations, followed by perceptual distortions (e.g., altered color perception, size distortion), pressured speech, hallucinations, and a subsequent sleep-like phase with vivid dreams or depersonalization. Coma may also occur, accompanied by hypotension and neuromuscular hyperexcitability. Symptoms usually persist for 8–24 hours, but coma may last up to 72 hours. After intoxication, patients may experience headaches, fatigue, depression, and occasionally psychotic symptoms [16]. In children, seizures and myoclonic jerks may occur [17]. In severe cases, *A. muscaria* intoxication may result in death due to cardiorespiratory failure [18] and the estimated mortality rate is 2–5%, with a lethal dose approximated at 15 mushroom caps. Toxin concentrations vary seasonally, so spring and summer specimens can contain up to ten times more active compounds than those collected in the fall [19].

Recent studies have shown that muscimol inhibits cell lysis by blocking the oligomerization of ninjurin-1, a process critical in pyroptosis. This finding suggests potential therapeutic applications of muscimol in treating diseases associated with excessive inflammation and programmed cell death, such as septic shock and other inflammatory conditions [20]. Additionally, muscimol has been shown to upregulate TREK-2 potassium channels on GABAergic neurons in the CNS, indicating a possible mechanism for centrally acting anesthetic agents. Activation of TREK-2 channels is believed to reduce the excitability of GABAergic neurons. This mechanism underlies current investigations into muscimol's analgesic properties, with studies demonstrating its efficacy in alleviating allodynia and hyperalgesia [21].

Ergot Alkaloids

Ergot alkaloids are a complex group of over 80 mycotoxins, typically categorized into **clavines** (e.g., chanoclavine, agroclavine), **ergopeptines** (e.g., ergovaline, ergotamine), and **lysergamides** (e.g., LSA, ergonovine) [22]. These compounds are primarily found in the sclerotia of fungi from the genus *Claviceps*, which parasitize cereal crops. In Europe, the most significant species is *Claviceps purpurea*, which infects rye, wheat, and barley. Other species, such as *C. africana* and *C. fusiformis*, occur in Africa and Asia, and ergot alkaloids have also been isolated from endophytic fungi like *Epichloë* and *Aspergillus fumigatus* [23]. Ergot alkaloids (e.g., LSA, ergonovine, LAH) are also present in the seeds of *Argyreia nervosa* and *Ipomoea corymbosa*, although they are not produced by the plants themselves but rather by symbiotic fungi of the genus *Periglandula*, which transport the alkaloids to the host's glandular trichomes [24].

The history of ergot dates back to antiquity, and it is speculated that it may have been an ingredient in the hallucinogenic beverages used during the Eleusinian Mysteries. Ergot was known in Egypt (ca. 1550 BCE), China (ca. 1100 BCE), and was described by Hippocrates (ca. 370 BCE) as a “corn disease” later named *melanthion*, which was effective in halting postpartum hemorrhage [25]. From the 9th century onwards, Europe experienced outbreaks of ergotism caused by consumption of bread and other baked goods made from flour contaminated with ergot-infected rye, such as in Aquitaine (944–945 CE), where approximately 20,000 deaths were reported [26]. Poisonings were also reported in modern times, such as in Pont-Saint-Esprit in 1951 [27].

Based on clinical descriptions from ergot poisoning outbreaks, two forms of ergotism have been distinguished: gangrenous and convulsive. Gangrenous ergotism resulted from acute ischemia of distal extremities due to the vasoconstrictive properties of ergot alkaloids. The hands, feet, nose, ears, and occasionally entire limbs became swollen and inflamed, accompanied by intense burning pain; eventually, the ischemic tissue became pale and insensate, progressed to dry gangrene (black discoloration), and underwent spontaneous autoamputation. This type of ergotism was referred to in the Middle Ages as “holy fire” (*ignis sacer*) or “St. Anthony’s fire,” with the former name alluding to the burning pain and the latter to the Order of St. Anthony, whose monks cared for ergotism victims [26].

Convulsive ergotism was initially characterized by a sensation of heaviness in the head, mild diarrhea, paresthesia of the hands and feet, and fasciculations (myokymia) of the periorbital and perioral muscles. This progressed to painful tonic-clonic contractions of limb muscles, and sometimes spasm of facial muscles, vocal cords, and the diaphragm. Severe cases included opisthotonus, marked hyperextension of the head and trunk due to neck and back muscle spasms, as well as status epilepticus, hallucinations with delirium, cognitive impairment, and mania [28]. The mortality rate for convulsive ergotism ranged from 10–20%. Historically, outbreaks of convulsive ergotism were more common east of the Rhine River, while gangrenous ergotism prevailed to the west. These differences are attributed to variations in the alkaloid composition of ergot produced by distinct *Claviceps purpurea* strains growing in different soil types. Alkaloids in eastern ergot specimens may have been present at levels sufficient to induce convulsive symptoms but insufficient to cause peripheral ischemia [29]. Ergot alkaloids have also been shown to significantly affect embryonic development during pregnancy. In utero exposure to methylergonovine has been implicated in cases of sirenomelia (fused lower limbs, caudal spinal defects, and malformations of the genitourinary and lower gastrointestinal tracts) [30]. Use of ergotamine for migraine treatment during the second month of pregnancy has been associated with increased risk of neural tube defects, posterior cleft palate, congenital cataracts, and clubfoot [31].

The mechanisms of action of ergot alkaloids include interactions with serotonergic, adrenergic, and dopaminergic receptors, leading to vasoconstriction (via D_1 and α_2 receptors), inhibition of prolactin secretion (via D_2 receptors), uterine contractions (ergometrine), and anorectic effects (via 5-HT receptors). Hallucinogenic effects are attributed to 5-HT_{2A} receptor agonism and increased glutamate release [32]. Chronic exposure to ergot derivatives (e.g., ergotamine) is believed to stimulate 5-HT_{2B} receptors, inducing myofibroblast proliferation, followed by thickening of heart valve leaflets and vocal cords. In the heart, this may lead to acquired valvular disease [33].

In modern medicine, ergometrine and ergotamine are infrequently used to manage postpartum hemorrhage. Ergometrine, due to its strong smooth muscle contractile properties, was formerly used as an oxytocin substitute [34]. Derivatives of ergot alkaloids, such as bromocriptine and cabergoline, are used in the treatment of Parkinson’s disease and hyperprolactinemia [35]. Ergotamine (rarely) and dihydroergotamine (more commonly) are still occasionally employed to treat vascular headaches, including migraines and cluster headaches [36].

Lysergic acid is the precursor to the hallucinogenic compound LSD, which was once used in the treatment of anxiety disorders, depression, psychosomatic illnesses, and addictions, and is now primarily a recreational psychedelic substance [37]. According to the legislation in Poland, the use of LSD is illegal [38] and currently research is underway on non-hallucinogenic LSD derivatives that could be used in the treatment of affective disorders [39].

Hallucinogenic Boletaceae Mushrooms

Certain mushrooms from the Boletaceae family found in parts of Asia, such as Papua New Guinea, China, and the Philippines, may exert hallucinogenic effects. Missionary and ethnographic accounts report that in western Papua New Guinea, species like *Boletus manicus* and *Tylopilus nigerrimus* have long been consumed during shamanic rituals by the Kuma people, inducing a state of "madness." These experiences often involved microptic hallucinations, a visual phenomenon where objects or people appear smaller than in reality [40]. Chemical analysis of *Boletus manicus* revealed three unidentified indole compounds, but in concentrations too low for precise identification [1].

In China's Yunnan Province, the local population frequently consumes Boletaceae species such as *Butyriboletus roseoflavus*, *Lanmaoa asiatica*, *Sutorius magnificus, Rubroboletus sinicus*, and *Boletus speciosus*. These mushrooms are not hallucinogenic if properly cooked for 15–25 minutes, which likely destroys heat-sensitive toxins. However, ingestion of raw, undercooked, or excessive amounts may lead to microptic hallucinations known locally as *xiao ren ren* ("many little people") [41]. One report described 300 poisoning cases involving *Boletus speciosus*, in which lilliputian hallucinations emerged within 6–24 hours post-ingestion and persisted for 10–30 days. More severe intoxications presented with schizophrenia-like symptoms, auditory hallucinations, pareidolia, thought disorders, aberrant behavior, personality disintegration, and stupor. EEG recordings in stuporous patients revealed LSD-like patterns [42].

Another study detailed 81 cases of poisoning from *Lanmaoa asiatica*, with neuropsychiatric symptoms, including visual hallucinations of "little people," dizziness, weakness, auditory hallucinations, ataxia, and headache, alongside gastrointestinal symptoms such as nausea, vomiting, diarrhea, and abdominal pain. Gastrointestinal symptoms appeared within 1-6 hours, while neuropsychiatric effects manifested after 12–24 hours [43]. To date, the specific toxins responsible for these effects remain unidentified.

Hallucinogens Derived from Other Fungi

Dictyonema huaorani is a lichenized fungus that was only recently described by science. Although samples were first collected from the Amazonian rainforest in Ecuador in 1981, species-level identification was not completed until 2014 [44]. This basidiolichen consists of a fungal mycobiont in symbiotic association with green algae, forming a unique composite organism. The hallucinogenic properties of *D. huaorani* were known to the Waorani (Huaorani) indigenous people (Amazonia), who referred to it as *nęnęndapę*. It was used sporadically by shamans during ritual practices, primarily for casting spells and invoking magical effects [45].

Chemical analysis of the lichen revealed the presence of tryptamine, 5-methoxy-N,N-dimethyltryptamine (5-MeO-DMT), and psilocybin. Further LC/MS studies indicated the likely presence of 5-methoxy-N-methyltryptamine (5-MeO-NMT), 5-MeO-DMT, and 5-methoxytryptamine [44].

Hallucinogens of animal origin

Amphibian-Derived Hallucinogens

The psychoactive properties of amphibian skin and parotid gland secretions have long been documented. Ancient Mesoamericans, Olmecs and Mayans, used *Incilius alvarius* (formerly *Bufo alvarius*) and *Rhinella marina* (formerly *Bufo marinus*) in ritual intoxications, and some *R. marina* toxins served as pre-Columbian "zombie powder" in North America [46]. Dried secretions from Chinese toad (*Bufo gargarizans*), known as Chansu, have been used in traditional medicine for infections and inflammation for centuries [47]. These secretions are defensive compounds released only when the amphibian is damaged.

Parotid and skin secretions contain two main compound groups: bufadienolides and indole alkaloids. The former (ca. 96 compounds such as bufalin, cinobufagin) exhibit cardiotoxicity in acute poisoning, while the latter (ca. 23 compounds like bufotenine, bufowiridine) are responsible for hallucinogenic effects [48]. **Bufotenine** (5OHDMT) acts as an agonist at serotonin receptors $5HT_{1A}$, $5HT_{2A/C}$, $5HT_{3/4}$ and stimulates serotonin release, but has limited blood-brain barrier penetration, leading to peripheral symptoms, e.g. tachypnea, chest tightness, flushing, nausea, vomiting [49].

In animal studies, bufotenine has shown anti-inflammatory and analgesic effects, likely via downregulation of COX, LOX, CYP_{450}, and lipid metabolism in pathways involving the σ_1 and $5HT_{3A}$ receptors [50]. In vitro, dehydrobufotenine exhibited cytotoxicity in human tumor cells via inhibition of topoisomerase II, while bufotionine inhibited proliferation in hepatocarcinoma and leukemic cells [51].

Incilius alvarius secretes an Omethyltransferase that converts bufotenine to 5MeODMT, which is a potent, fast-acting hallucinogen. Toad venom is toxic not only if ingested orally, but produces also strong psychoactive effects when smoked [52]. Oral consumption (licking or ingestion) can cause digoxin-like toxicity with excessive salivation, perioral numbness, gastrointestinal distress, arrhythmias, blood pressure changes, and seizures [53]. Smoked 5MeODMT induces immediate, intense visions lasting 10–20 minutes, including perceptual distortions, emotional intensification, ego dissolution, and occasionally anxiety, disorientation, or panic; it acts as an agonist at $5HT_{1A}$ (highest affinity) and $5HT_{2A}$ [54]. It is currently under investigation for treatment of depression, addiction, and neurological disorders, with reports suggesting rapid and sustained reduction of depressive, anxiety, and stress symptoms, along with neuroendocrine and anti-inflammatory effects [54, 55].

Kambo (or *sapo*) is the skin secretion of Phyllomedusa tree frogs, used traditionally in Amazonian cleansing rituals. It causes vomiting, diarrhea, tachycardia, and blood pressure fluctuations, and side effects may include psychosis, seizures, syndrome of inappropriate antidiuretic hormone secretion (SIADH), kidney and liver damage, pancreatitis, dermatomyositis, and esophageal rupture [56]. Its bioactive peptides, phyllomedusin, phyllokinin, cerulein and sauvagine, act on smooth muscle and vasculature, causing nausea, bile secretion, hypotension, tachycardia, flushing, and angioedema. Reported well-being and motor clarity may derive from dermorphin or deltorphin (opioid peptides). Cerulein increases digestive secretions, while sauvagin stimulates corticotropin release and mimics stress responses [57]. In one retrospective study, the acute effects of Kambo were found to be dominated by intense physical responses followed by increased mental clarity and concentration, rather than psychedelic ego-dissolution. In addition, benefits such as "glow" (relaxation, elevated mood, openness, empathy) were reported without a prior psychedelic state [58].

Although scientific evidence is insufficient to support therapeutic use of Kambo, peptides such as dermorphin, cerulein, and deltorphin show promise. Dermorphin and cerulein, with high-affinity μ-opioid receptor agonists, have demonstrated efficacy in treating postoperative and cancer pain in humans [59]. Deltorphin, a selective δ-opioid agonist, is under investigation for cardioprotective effects in reperfusion injury and as a potential antidepressant [60, 61].

Fish-Derived Hallucinogens

Hallucinogenic intoxication from fish toxins has been reported across a wide range of species. Coral reef fish from the Mediterranean Sea and the Indian and Pacific Oceans, particularly those from families such as Sparidae, Mullidae, Kyphosidae, Acanthuridae, and Mugilidae, contain bioactive compounds capable of inducing psychoactive effects. These toxins appear to be thermostable, as symptoms have been reported after ingestion of cooked, fried, or baked fish [62].

The clinical picture, known as **ichthyoallyeinotoxism**, typically emerges within minutes to two hours after fish consumption and may persist for up to 24 hours. Symptoms include vivid and often terrifying auditory and visual hallucinations, dizziness, balance disturbances, motor discoordination, psychological depression, intense fear, nightmares, pruritus, throat burning, muscle weakness, and occasionally gastrointestinal upset [46].

In ancient Rome, *Sarpa salpa* (salema porgy) was reportedly consumed for its hallucinogenic effects [46]. Contemporary case reports describe mild gastrointestinal symptoms along with frightening hallucinations, nightmares, altered behavior, and in one instance, anterograde amnesia during the hallucinatory episode [63]. The precise toxins responsible remain unidentified, though experimental studies suggest that the liver and viscera are particularly toxic [64]. Some hypotheses point to toxic dinoflagellates (*Dinoflagellata*) consumed by the fish, or to heavy metals bioaccumulated in their tissues, as potential sources [65].

Ciguatoxins, produced by dinoflagellates of the genera *Gambierdiscus* and *Fukuyoa*, are another group of marine neurotoxins that may induce hallucinogenic symptoms. These toxins biomagnify through the food chain and are found in fish such as *Epinephelus spp.* (groupers), *Lutjanus spp.* (snappers), *Sphyraena spp.* (barracudas), *Acanthurus spp.* (surgeonfish), and members of the *Muraenidae* (moray eels) family [62]. Ciguatoxins are polycyclic polyether compounds that cause prolonged activation of voltage-gated sodium channels and inhibition of potassium channels, leading to sustained membrane depolarization and repetitive action potential firing in excitable tissues, including neurons, skeletal muscle, smooth muscle, and cardiac tissue. This condition, known as ciguatera poisoning, may be accompanied by hallucinations, mood disturbances, impaired concentration, confusion, and sensorimotor dysfunction [66].

Invertebrate-Derived Hallucinogens

Certain insects and other invertebrates have been used for their hallucinogenic properties in traditional medicine and rituals. In southern and south-central California, Native American groups such as the Chumash, Kitanemuk, and Tübatulabal consumed large quantities of live red harvester ants (*Pogonomyrmex spp.*) to induce prolonged catatonic and hallucinatory states, often during initiation rites for young boys [67]. These ants were also employed in traditional medicine for conditions such as diarrhea, arthritis, and paralysis. Their venom contains formic acid and peptide kinins, which cause pain, inflammation, and hypotension. Some kinins exhibit nicotinic cholinergic activity, possibly underlying the hallucinogenic effects, typically requiring ingestion of 250 or more ants [68].

In southeastern Brazil, the Malali people used dried and powdered caterpillars of *Myelobia smerintha* ("bicho de taquara") to promote wound healing. When the caterpillar's head was left intact during drying, users reported entering a full-day ecstatic sleep with vivid visions of lush forests and successful hunts, suggesting a hallucinogenic compound concentrated in the salivary glands of the head [69].

In rural northeastern India, bugs of the genus *Coridius* (e.g., *C. nepalensis*, *C. singhalanus*, *C. chinensis*), known locally as *tari*, are consumed as food or spice. About 6% of users report adverse effects, including hallucinations and agitation following excessive intake [69, 70]. Affected individuals exhibited unusual behaviors such as attempting to fly, crawling into small spaces, or mistaking strings for snakes. Additional symptoms included paralysis, motor incoordination, postural instability, dizziness, vomiting, and general weakness. Symptoms could persist untreated for six months or longer. The effects may result from semiochemicals released by scent glands, microbial toxins, or pesticide residues [71].

Marine sponges such as *Smenospongia aurea*, *S. echina*, and *Verongula rigida* produce brominated tryptamine derivatives including 5-bromo-DMT and 5,6-dibromo-DMT, which act on 5-HT_{2A} receptors and exhibit psychedelic effects [46]. Marine indole alkaloids also show promise for pharmaceutical development targeting infectious diseases, cancers, and psychiatric disorders [72].

Summary

This review provides a comprehensive overview of naturally occurring hallucinogens from mushrooms, emphasizing their ethnopharmacological significance, chemical structures, mechanisms of action, and related psychotropic effects. Mushroom hallucinogens, primarily from *Psilocybe spp.* (psilocybin, psilocin), *Amanita muscaria* (ibotenic acid, muscimol), and *Claviceps purpurea* (ergot alkaloids), have been used for centuries in ritual and therapeutic contexts. These compounds act primarily through serotonergic pathways, especially the 5-HT_{2A} receptor, producing altered perceptions, mystical experiences, and in some cases toxic effects.

In contrast, hallucinogens of animal origin encompass a variety of chemical classes and mechanisms. These include tryptamines such as 5-MeO-DMT from toads (*Incilius alvarius*), neurotoxins such as ciguatoxins from reef fish, formic acid-derived kinins from *Pogonomyrmex* ants, and halogenated indole alkaloids (e.g., 5-bromo-DMT) from marine sponges. These substances can induce vivid auditory and visual hallucinations, dissociative states, or prolonged neurotoxic symptoms, depending on the compound and route of exposure.

Despite their distinct biological origins, mushroom and animal hallucinogens have shaped cultural practices and inspired scientific research into consciousness, neuropharmacology, and potential therapeutic applications. Ongoing research into their pharmacodynamics and biosynthetic origins continues to expand our understanding of naturally occurring psychoactive agents in the biosphere.

Founding

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