



# How to Tell if Your Cat or Dog has Consumed a Psychotropic Mushroom

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**Abstract:** Accidental ingestion of psychotropic fungi by animals is a common occurrence that receives little attention due to an inability to properly diagnose and identify behavioral symptoms. The current review has organized both case studies and experimental research in an effort to define expected cat and dog symptoms caused by some common fungal toxins: isoxazoles and psilocybin-related hallucinogens. These compounds elicit diverse behaviors in dogs and cats, as reported in the reviewed material, which are compared and contrasted with human cases. In the event of onset of specific symptoms, vital procedures are suggested, and possible treatments are discussed. Information in this review is intended to inform readers/pet owners on warning signs of animal ingestion, as well as to promote better documentation of such cases.

## Introduction

Ingestion of mushrooms with psychotropic properties by animals is usually unintentional; dogs and other

pets often see a mushroom that may look and smell like food so they eat it. Fungi generally taste meaty due to their high concentration of amino acids (Hallock, 2007), and are thus attractive to dogs and other animals as potential food items. It is not until later, sometimes several hours later, that these tasty and curiously smelling mushrooms begin to create problems for the animals. They can lead to vomiting, diarrhea, disorientation and, sometimes, even coma or death. Although reports of toxic mushroom ingestion are common in humans, animal cases are not as well-documented and are severely underreported (Spoerke, 2005). The incidence of dog poisoning by mushrooms could very well be higher than the frequency of mushroom poisonings in humans. On the other hand, cats are very rarely poisoned by wild mushrooms – many reported cases are due to cats finding their owner's supply of dried mushrooms intended for recreational use (Beug and Shaw, 2009).

Animals can be exposed to psychotropic mushrooms in household yards, greenhouses, and even parks. There is a distinct lack of effective methodology for confirming whether exposure to mushrooms is actually the cause of symptoms in animals (Puschner, 2007), and a lack of standardization in

documenting these cases as well. Even if mushroom poisoning is without a doubt the cause of illness, it can be very hard (and sometimes impossible) to identify the mushrooms that animals might have consumed, even with a thorough examination of the area where the animal might have been exposed (Spoerke, 2005). For example, among mushroom poisonings in dogs between 2001 and 2004, 21 out of 70 reports (30%) involved mushrooms that could not be identified (Beug, 2006).

Treatment for cases of mushroom poisoning in animals is mainly supportive. Care focuses on the symptoms of the poisoning and does not generally change with the consumption of different species of mushrooms. That being said, knowing the identity of the ingested mushroom is important for other reasons such as deducing what behavioral effects to expect in the hours following the poisoning. Different mushroom species can contain varied concentrations of toxins throughout their life-cycles (Tuno et al., 2009). All parts of the mushroom can be critical for proper identification, including the complete cap and stipe. Additionally, any remnants of the partial or universal veil (which is often buried in the ground) can be critical for positive identification.

If possible, people with a suspected mushroom poisoning case should bring a sample of the suspected mushroom to medical personnel or to an expert on mushrooms, and take care that the specimen is not damaged in transit. In some cases, the offending mushroom can be identified from the vomitus of the afflicted animal (Yam et al., 1994). If more instances of fungal poisonings in dogs and cats were reported with correctly identified mushroom samples, it could increase the accuracy of diagnosis and lead to more effective treatments. Please see sidebar at the end of this article for instructions on how to report animal mushroom poisonings to the North American Mycological Association.

The effects of mushroom toxins on humans and animals can be vastly different; any mushrooms that are safely eaten by animals may not be safe for human consumption (Spoerke, 2005). Some mushrooms, like *Amanita muscaria*, can cause gastrointestinal discomfort in humans but can be fatal if ingested by a puppy or young cat. In this report we will focus on the mushroom toxins: isoxazoles and psilocybin-related hallucinogens, as well as the fungi genera with which they are associated (Table 1). Isoxazoles and psilocybin-related compounds are the cause of pantherine and psilocybin/serotonin syndromes, respectively, in humans (Beug et al., 2006).

Reports of toxic mushroom poisoning in both humans and domestic animals have been compiled by the North American Mycological Association (NAMA) since the 1970s (Beug, Shaw and Cochran, 2006), and those reports are used here to develop a list of symptoms for the two groups of toxins. We will use experimental studies to further detail and classify symptoms

associated with the administration of various fungal toxins to laboratory animals. The resulting behaviors were documented and are discussed here in relation to symptoms from case studies.

**I. Isoxazoles.** The isoxazole compound group includes ibotenic acid and muscimol, both of which can cross the blood-brain barrier, once ingested, and are known to cause altered visual perceptions in humans (Cope, 2007). Ibotenic acid is a derivative of  $\gamma$ -aminobutyric acid (GABA), the principal inhibitory neurotransmitter in the brain, and it is known to act as a receptor agonist and bind to GABA receptors in both humans and animals (Johnston et al., 1968). Muscimol is derived from ibotenic acid through decarboxylation, which can occur either when the mushroom becomes dehydrated, during the digestion process, or when the compound is absorbed in other body tissues (Puschner, 2007). Ibotenic acid and muscimol are the cause of pantherine syndrome, which is characterized by onset of symptoms including dizziness, disorientation, and ataxia within 30 minutes to 2 hours of ingestion. This is generally followed by periods of hyperactivity followed by deep, coma-like sleep (Gucin and Bunyard, 2006) during which the patient (human or animal) may require assistance with breathing (Puschner, 2007).

The fungal species most often associated with isoxazoles are of the *Amanita* genera. *A. muscaria* and *A. pantherina* are the most common species accidentally consumed (Hall and Hall, 1994; Spoerke, 2005). Although these two species are not lethal in humans, they can be fatal if ingested by an animal (Beug et al., 2006). Isoxazole-containing mushrooms are widespread, and grow from summer to autumn in coniferous

or deciduous forests throughout the country (Arora, 1986).

*Symptoms/Effects of ibotenic acid/muscimol poisoning:* Many of the reported poisonings in domestic animals have been associated with this type of toxin. There have even been some observations of dogs that repeatedly seek out, consume and, subsequently, get sick from *A. muscaria*, to the point their owners must muzzle them when they go outdoors (Beug, 2007). The most common symptoms reported in dogs were agitation, disorientation, salivation, ataxia, vomiting, diarrhea, panting, muscle spasms, dilated pupils, periods of hyperactivity followed by weakness and lethargy, extreme thirst and increased body temperature and heart rate (Beug, 2006; Spoerke, 2005). More severe cases can lead to respiratory failure, liver failure (Beug 2006), seizures, collapse, cyanosis (skin turning blue from lack of oxygen) and, in some cases, death (Spoerke, 2005). Of the dogs that die from these mushroom poisonings, many are puppies whose immune systems are still developing (Beug and Shaw, 2009); healthy, adult dogs most often recover fully (Beug, 2009; Beug 2010). In one case a 4-month old terrier ate half of the cap of an *A. muscaria* and in less than an hour suffered from intense salivation, diarrhea, tremors, and seizures (Beug, 2006). The animal died 4.5 hours after consuming the toxic cap. One possible cause of death in dogs after ingestion of these types of toxins is a veterinarian's decision to euthanize the animal when coma-like sleep or breathing problems occur (Beug and Shaw, 2009).

Isoxazole poisonings in cats have often been found to be the result of cats consuming dried *A. muscaria* that their owners have for recreational use (Beug and Shaw, 2009). In cats that ingested this toxin, the most common symptoms were vomiting (Spoerke, 2005) and an alternating cycle of hyperactivity accompanied by muscle spasms and deep sleep (Puschner, 2007). There have also been a few deaths associated with a cat's consumption of dried *A. muscaria* or *A. pantherina* (Beug and Shaw, 2009). Interestingly, laboratory studies involving cats and isoxazole compounds showed some similar yet different results.

In 1969, researchers conducted a series of experiments in which muscimol was administered to cats and rabbits

**Table 1**

List of mushroom toxins and the mushroom genera they are associated with\*

Toxin	Associated Mushroom Genera
Isoxazoles	<i>Amanita</i> sp.
Psilocybin-related	<i>Conocybe</i> sp., <i>Gymnopilus</i> sp., <i>Inocybe</i> sp., <i>Mycena</i> sp., <i>Panaeolus</i> sp., <i>Pluteus</i> sp., and <i>Psilocybe</i> sp.

\*Adapted from Spoerke (2005)

(Scotti de Carolis et al., 1969). Cats were trained to press a lever in response to a specific sound in order to receive a pellet of food. This task was performed before and after administration of muscimol, and performance on the task and recordings of brain waves using an electroencephalogram (EEG) was also recorded. Cats exhibited noticeable signs of intoxication at 1 mg/kg (Scotti de Carolis et al., 1969): one cat waved its paw in the air, and made unusual head movements and ear twitching. The authors speculated that waving the paw in the air was an effort to complete the conditioned task and obtain the food reward. The cat exhibited attenuated locomotor activity, and EEG recordings revealed continuous and synchronous activation throughout the brains of the cats, similar to what would be expected during a seizure. This activity presumably precluded coordinated activation necessary for meaningful thoughts and movements. Rabbits underwent a similar conditioning paradigm, and doses as small as 0.5 mg/kg resulted in lethargy during trials.

Schmied et al. (1990) showed that cats' reaction times became slower with the administration of muscimol. The brain region researchers focused on was the red nucleus, which plays an important role in coordination. GABA is prominent in the red nucleus and functions to halt the movement of limbs (Schmied et al., 1990). Since muscimol can bind to GABA receptors in the brain, Schmied et al. (1990) injected it directly into multiple regions of the red nucleus to see if it had an effect on reaction time in response to a conditioned stimulus. In larger doses, severe motor impairments were observed. The common symptoms of dystonia (repetitive twisting and rolling over and abnormal sustained muscle contractions) caused some cats to stop pressing the lever in response to the conditioned stimulus. The cats actively consumed freely available food pellets, so motivation was not the issue. Lower doses led to significant increases in reaction times, due to the agonistic effects of muscimol on GABA receptors. In sum, these animal experiments describe the neural mechanism responsible for lethargy in the case studies discussed above, and there is evidence the effects of GABA may also underlie the respiratory

failure in animals. Human cases of overdosing on GABA-like drugs leading to respiratory failure are common and well-documented (Jones and Holmgren, 2009). One case study involving intentional overdoses of diazepam (a drug that binds to GABA receptors) in two humans resulted in spontaneous recovery within 48 hours after a deep coma (Greenblatt et al., 1978). It has long been argued that the same would be true in most animal cases of ibotenic acid/muscimol poisoning, though many end in euthanasia as decided by the pet owner (Beug and Shaw, 2009). With these considerations we urge pet owners to be patient when labored breathing/coma-like sleep due to mushroom ingestion is exhibited in their pets.

**Possible treatments:** Treatments are mainly supportive. Detoxification measures, such as activated charcoal, can be effective if administered soon after exposure. Dogs suffering from pantherine syndrome often recover within 12-24 hours. Aggressive supportive care (such as mechanical ventilation during respiratory distress) may be required. Cats usually fully recovered within 24 hours, especially if early detoxification measures were taken (Puschner, 2007). Administering IV fluids for rehydration is common (Beug, 2006). Keeping the animal confined in a quiet, dark space is often recommended (Cope, 2007). In cases of poisoning by *A. muscaria* or *A. pantherina*, vets may administer atropine. This drug, however, is only effective at treating muscarine poisoning (a compound associated with *Amanita* mushrooms but only present in trace amounts in these two species) and can exacerbate the symptoms caused by ibotenic acid and muscimol (Beug and Shaw, 2009).

**II. Psilocybin/Psilocin and other recreational hallucinogens.** Ingestion of psilocybin/psilocin causes symptoms such as scenery hallucinations, changed meanings of percepts, facilitated imagination, positive mood, and a changed sense of time (Studerus et al., 2011). These compounds produce the effects of the hallucinogenic mushrooms, and are usually associated with the intentional ingestion of humans looking to get high (Gucin and Bunyard, 2006). Many species of *Gymnopilus*, *Panaeolus*, and *Psilocybe* mushrooms contain both psilocybin and psilocin. There are also

a few species in the genera *Conocybe*, *Inocybe*, *Mycena* and *Pluteus* that contain both of these compounds. Some of these mushrooms are generally coprophilic and can be found growing in pasteurized fields, some are grassland species closely associated with specific species of grasses, and many are lignicolous, either growing on lignin-rich soil or directly on wood (Stamets, 1996). Because psilocybin cannot cross the blood-brain barrier until it is dephosphorylated into psilocin, the latter compound is most likely the cause of all clinical symptoms (Puschner, 2007). Psilocin is believed to act as a serotonergic agonist.

**Symptoms/Effects of psilocybin/psilocin poisoning:** Among the reported cases of psilocybin poisoning were several reports of ingestion by dogs, and one case of cat consumption. As a caveat, many cases of mushroom ingestion, especially those that contain psilocybin and psilocin, by animals do not get reported. For example, some recreational mushroom hunters who bring along their dogs have found that the dogs will get conditioned to liking *Psilocybe* mushroom hunting once they have tried such psychotropic mushrooms, often seeking them out again and again (M.W. Beug, Personal communication, March 16, 2011). These cases are not found in the NAMA database because they are not considered toxic incidents. Of the reported cases, the symptoms most often observed in the dogs were vomiting, disorientation and ataxia, increased body temperature, aggression and barking, and possible hallucinations (Beug, 2006; Puschner, 2007; Spoerke, 2005).

The cat suspected of eating psilocybin-containing fungi suffered from vomiting, excessive salivation, diarrhea, erratic breathing and constricted pupils. These symptoms are in contrast to human cases where vomiting very rarely occurs, and if it does, it may be the result of other compounds present in some varieties of psilocybin mushrooms. Humans also respond with pupillary dilation, not constriction.

Kirsten and Bernardi (2010) observed mice behavior after psilocybin injections. Behaviorally, treated mice showed increased gnawing, decreased movement, and increased "wet-dog" shaking. Davis and Walters (1977) found a biphasic effect of psilocybin on startle responses in rats. The rats would receive



## How to Report Cases to NAMA

Accurately reporting cases of animal poisoning to the North American Mycological Association (NAMA) will increase our knowledge of toxicology cases in pets. If you have a suspected mushroom poisoning in a pet, you should contact your veterinarian or a pet emergency hospital. Another option is to contact the animal poison control center (1-888-426-4425), although this service currently requires a \$65 consultation fee.

You should also try to get the consumed mushroom identified. In addition to resources in your local mushroom clubs (see <http://www.namyco.org/clubs/index.html>) or colleges/universities, NAMA has a list of volunteer identification consultants, organized by state, who can assist with the identification of mushrooms (see <http://www.namyco.org/toxicology/identifiers.html>). If possible, you should save any remnants of the ingested mushroom and other samples of the same species if they are growing nearby. Specimens should be placed in a brown paper bag (not plastic) until an expert can examine and identify them.

Afterwards, please submit an online report of the incident (it is the same form whether a person or a pet is the victim) to the NAMA Poison Case Registry at [http://www.namyco.org/toxicology/email\\_report\\_form.html](http://www.namyco.org/toxicology/email_report_form.html). Every reader should view this form to become better familiar with it: it is easy to follow and should take about 10 minutes to fill out. The first questions have to do with general demographics about the victim (species, age, sex) and how the mushroom was eaten (cooked or raw?). It also asks details about where the incident occurred. The form has a checklist of symptoms (including chills, fever, sweating, intestinal cramps, vomiting, and muscle spasms) that the reporter simply checks off if present. The form concludes with questions about treatment and outcome, and then asks about the genus and species of the culprit, and specifically how the identification was determined.

We encourage you to be familiar with NAMA's form, as the increased knowledge gained by more submissions and a greater accuracy in the reports helps us to better understand the risks that mushroom poisonings pose to pets. Access to a better record of pet mushroom poisonings will increase the effectiveness of treatment research and implementation.

an injection of either saline solution or psilocybin and were subjected to a loud tone that would normally result in the rats becoming startled. Lower doses of psilocybin increased the startle response, whereas doses larger than 2.5 mg/kg nearly eliminated the startle response.

Another interesting study was conducted on the inhibiting effects of

psilocybin on the obsessive compulsive disorder (OCD)-like behavior of marble-burying mice (Matsushima et al., 2009). Marble-burying has been used as an animal model of OCD. In response to unwanted materials on their bedding, mice will bury them in a repetitive manner similar to OCD sufferers. Matsushima et al. (2009) compared the effects of

pure psilocybin with those of whole *Psilocybe argentipes* on the OCD-like behavior of the mice. They injected low doses of pure psilocybin or powdered *P. argentipes* into different groups of mice. Results showed that larger doses of pure psilocybin were required to significantly inhibit the OCD-like behavior than an equivalent dosage of powdered *P. argentipes*. The authors suggested that *P. argentipes* contains other tryptamine derivatives, in addition to the psilocybin, that resulted in the behavioral differences between the two groups of mice. Changes in marble-burying were observed absent of locomotor inhibition, which is good for possible human usage as treatment for OCD. Indeed, preliminary research on the use of psilocybin to treat OCD in humans shows promising results (Moreno et al., 1997).

*Possible treatments:* Treatment for psilocybin or psilocin poisoning is mainly supportive including rehydration with IV fluids, if necessary. In many cases, treatment is not necessary at all (Puschner, 2007); the biggest threat to the health of these animals is their altered perceptions and behaviors leading to some physical trauma, which can be treated by confining the animal to a dark, padded cage (Cope, 2007). Because some animal poisonings involving these compounds occur during their owner's attempts to get high, veterinarians might want to investigate the possibility of co-ingestion with other hallucinogenic drugs such as LSD, PCP and marijuana (Cope, 2007).

**Conclusion.** The main psychotropic agents involved in mushroom poisonings are isoxazoles and psilocybin, and as shown above, each causes different sets of symptoms. Although fungal poisoning is common in both humans and animals, relatively fewer animal poisonings are reported, so less is known about the effects of mushroom toxins on animals. Mushroom poisonings in humans and domestic animals are different. The fact that a pet or wild animal can eat a mushroom without experiencing any adverse effects does not mean that they are safe for humans. Many species have caused no reported deaths in humans but have proven to be lethal in domestic animals (Beug et al., 2006). When an animal

becomes sick and mushroom ingestion is suspected, the identification of the mushroom, though not essential to determining proper treatment, is of great importance in helping veterinarians and pet owners anticipate what may lie ahead in the progression of symptoms. Classification of the mushroom culprit can also assist other animal owners in recognizing the types of mushrooms that may pose a threat to their animals. The best way to prevent mushroom poisonings in pets is to limit their exposure to potentially dangerous mushrooms (Cope, 2007).

Experimental research on the isolation of the behavioral effects of psychotropic fungi will undoubtedly prepare veterinarians and responsible pet owners for the occasional accidental consumption. Taken collectively, the controlled experiments reviewed here elucidate the underlying neural mechanisms of individual symptoms described from the animal case studies. This information increases our understanding and aids in the best treatment of animals to ensure the best outcome in cases of accidental cases of toxic mushrooms.

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