

# **Toxic Fungi of Western North America**

by

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## Introductory Material

### Dedication

Dedicated to my wife Ellen and my 4 children—Carolyn, Linda, Susan and John. Also in remembrance of Paul Vergeer.

### Preface

For cases in which *Amanita phalloides* or other deadly amatoxic fungi are suspected, go at once to the section titled Guide to the identification of mushroom poisoning by symptoms and, if appropriate, The amatoxic group. For a thumbnail guide to the treatment of amatoxins, go to the end of the discussion on amatoxins to Outline and summary of treatment for poisoning by amatoxins.

See after the main text for a discussion of scientific naming (nomenclature) and scientific classification (taxonomy). Species subject to taxonomic confusion with possibly unstable names are followed by the author citation when they are first mentioned. Citations are appended to all amatoxic species. Citations are customarily abbreviated for some names. One modern standard for author abbreviations is that of P.M. Kirk and A.E. Ansell in 1992.<sup>1a</sup> Two examples are “Fr.” for Elias Fries (cited for the amatoxic *Amanita phalloides*) and “Bres.” for the Abbé Giacomo Bresadola (cited for the amatoxic *Lepiota helveola*).

All descriptions of size are given in metric measurements. One inch is 2.54 centimeters or 25.4 millimeters. A centimeter (or 10 millimeters) therefore comes out to about 2/5 of an inch. A 4-inch mushroom cap is roughly 10 centimeters across. Millimeters are very handy for small measurements, replacing the awkward 1/8ths and 1/16ths inches of the English system.

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Harry D. Thiers. Special thanks go to Dr. Ammirati for the section on *Cortinarius* and to Dr. Tulloss for the sections on *Amanita* and for the examples in taxonomy and nomenclature.



# An Introduction to Mushrooms & Mushroom Poisoning

## Introduction and collection of specimens

Since the 1977 publication of “California Toxic Fungi”<sup>1</sup>, there have been numerous journal additions, more information on the fungi of western Canada and much more interest in the mushroom flora of Mexico. Toxic Fungi of Western North America covers this larger area (from Alaska through Mexico and from the Pacific Ocean through the Rocky Mountains) and includes additional information, especially on new categories of mushroom poisoning. However, except for liver transplantation and much better intensive care treatment, there have been few major breakthroughs in therapy.

## CALIFORNIA TOXIC FUNGI

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The guide below is based on clinical experience and reviews of North American and European references. The final determination of safety, however, has to be made by each collector, as does the specific treatment of individual patients by physicians. Many treatments, especially in Slavic countries (which have the most experience) are lacking in statistical significance, often routinely selecting and/or reporting on 100 or fewer samples.

Mushroom names are the scientific ones, which consist of a genus name, a noun, which is capitalized and a species epithet, an adjectival form which is not capitalized. Both are italicized. They indicate one and only one species and place it firmly in a family of names. This report avoids common names, which vary from one

area to another and often refer to more than one fungus. For a more detailed examination of scientific names see the two addenda to this paper, which review in particular names in the genus *Amanita*.

In poisoning cases, any mushroom leftovers should accompany the patient for identification. Wax paper serves well as it allows the mushroom to “breathe”. Decay in a plastic bag may render a specimen unidentifiable even for an experienced microscopist. A timely trip to the same area from which the offending mushrooms were collected may allow gathering of additional material, which should include the extreme base of mushrooms to check for universal veil tissue left in that area. That so-called “death cup” or volva may be reduced to thin membranous limbs arising from the upper part of the stalk’s bulbous base and often collapsed on it. [see glossary for “limb”] Sometimes the volva leaves only ragged rings of tissue on the lower stalk or even presents as friable flakes that may be left in the soil when specimens are excavated. The universal veil enfolds species when they are young and often leaves tissue on the cap as well.

## General overview of mushroom poisonings

The mushrooms that cause almost all deaths worldwide contain amanitins. *Amanita phalloides* (Fr.:Fr.) Link and *Amanita ocreata* Peck are the usual amatoxic species in western North America. These two Amanitas have caused all of the recorded deaths in California (which leads the nation in mushroom fatalities) and nearly all of those in the West. The majority were patients who had ingested *Amanita phalloides*. *Amanita ocreata* has caused a large number of California deaths and at least one death in Oregon.<sup>10</sup> Two serious poisonings have occurred with amatoxic lepiotas—*Lepiota helveola* Bres. and *Lepiota josserandii* Bon & Boiffard in California. There has been one fatality with *Lepiota josserandii* in Vancouver, British Columbia.<sup>9</sup> There was one death in Washington state caused by an amatoxic *Galerina* species. Other serious cases of *Galerina* poisoning have occurred in the PNW.

Unfortunately, there are no simple tests for these amatoxins or for any other toxins in mushrooms, such as silver turning black or rice turning red. Any supposed benefit is the result of pre-selection—making a distinction between “toadstools” and “mushrooms” in the home area of the collector. Since these local, non-systematic distinctions have no scientific basis, collecting in an unfamiliar area may lead to serious poisoning. For example, Americans of Asian heritage may ignore the deadly significance of a volva, since the paddy straw mushroom of east Asia is both very edible and decorated at the base of the stalk with a volva; the spores, however, are pink in contrast to those of the deadly amanitas.

Because of the delay from gastrointestinal symptoms to organ failure in some poisonings, instructions should be given to have emergency room patients return if symptoms are continued or if new symptoms occur. These instructions should

be noted on the chart. If the mushroom is known to contain amanitins, the patient should be closely monitored even if initial laboratory studies are negative. Unless it is reasonably certain that the patient can and will return in 4-8 hours, it is probably best to keep the patient in the emergency room.

The consumption of wild mushrooms in the West became increasingly popular in the 1960's because of the rush to natural foods, mushroom fairs, a return to ethnic roots and later the need of loggers to move to another forestry industry. Poisonings, however, have leveled off. Both poisonous and highly edible mushrooms are rare compared to those that are simply inedible.

## Ecology and general anatomy of fungi

A mushroom is the fruiting body of a fungus; just as a berry is the fruiting body of a green plant, bush or tree. The vegetative portion of the fungus that gives rise to mushrooms is a tangle of long cellular threads called mycelia. The substrate on which a mushroom grows may be bare soil, humus, dung, decayed wood, living trees or other organic material.

Most of our commercially sold mushrooms are decomposers, such as the white to brown varieties of *Agaricus bisporus* most commonly seen in grocery stores. Other mushrooms are parasitic, most conspicuously on trees, although often on previously damaged ones. Many fungi also combine with plant rootlets in a mutually beneficial fashion called mycorrhizal.

Since fungi do not have chlorophyll to provide energy for their growth and reproduction, mushrooms may associate with green-plant rootlets to advantageously swap the chemicals of life. The mycelia of such mushrooms closely wrap themselves around and/or penetrate the fine rootlets of trees and other green plants to form a relationship called mycorrhizal. In such an association, there is a bi-directional movement of chemical compounds in which water, mineral salts and simple nitrogenous substances flow to the green plant and simple carbon compounds along with more complicated carbohydrates flow to the mushroom. The structures themselves are called mycorrhizae.

Most of the mushrooms in our forests have winding mycorrhizal attachments to the fine rootlets of trees that are superficial in that they do not penetrate the cells of the rootlets; such mycorrhizal structures are called “ectotrophic mycorrhizae”. In contrast, those mycorrhizae that penetrate cells by one of several known structures are called “endotrophic mycorrhizae”—a type of mycorrhiza typical of non-woody green plants and some plants of the heath

family such as blueberries. The vast majority of all plant species, perhaps as high as 95%, belong to genera that characteristically form mycorrhizae with fungi.

Trees, grown with their native mycorrhizal fungi, grow much more quickly in forests or garden plots than those trees grown without their fungal partners. Part of this benefit is due to an increase in the surface area of the fungal-associated roots and their better absorption of subsurface water and minerals; part of the benefit is due to active chemical transfer by the mycorrhizae.

Some of the more ancient genera of trees, including sequoia (*Sequoiadendron*, *Sequoia* and *Metasequoia*) do not form ectomycorrhizae. All the large ectomycorrhizal mushrooms found in Redwood forests are in association with other trees or bushes, such as manzanita, oak, tanoak and madrone. *Sequoiadendron gigantea* and *Sequoia sempervirens* form endomycorrhizal structures—spores, branching hyphae and balloon-like vesicles—but all on a microscopic scale requiring special stains.

The fruiting pattern of all higher fungi is exceedingly complex and much more variable than that of green plants. Known factors include moisture, drainage, temperature, pollution, time of the last fruiting, changes in associated plants and soil disturbance. High winds rapidly dry out what would otherwise be a good crop. Fog along the coast primes many species that fruit in the fall and some fungi get enough fog drip to repeatedly fruit before the fall rains. Snow bank fungi depend on the depth of snow, rapidity of snowmelt and the amount of spring rainfall among many factors.

Mycelia can lie unproductive for many years. A retirement community (Rossmoor) near San Francisco had a hillside on which morels had not previously been found: a period close to 25 years. After disturbing the soil for new landscaping, wood chips were placed over the ensuing berm. The following spring in 1996 hundreds of morels sprang up. No morels have been seen since that time. Such an isolated fruiting may be due to pre-existing mycelia in the ground that respond to the stimulus of uncovering, followed by the placement of a moisture barrier (in the form of woodchips).

Many mushrooms do not at all look like the common store mushroom. There are club, coral, cauliflower, cup and other forms in a spectacular array of colors—white, yellow, gray, brown, orange, red, violet, but very rarely, green. However, our most familiar mushrooms have a cap, vertical plates (gills) under the cap and a "stalk"—referred to here as a *stipe*. The mushroom's spores are usually ejected from the sides of these variably spaced gills, which may be free from the stipe or

attached in several ways to it. In a variant of this tendency to form gills, there may be only “folds” or ridges as in chanterelles; occasionally even this structural feature is reduced to smooth or pimpled surfaces.

A ring or annulus may be present on the stipe as the remnant of an embryonic *partial veil* that was attached between the cap edge and the stipe. **A universal veil, when present, sheathes the entire mushroom in its embryonic stage and leaves a cup or other traces of tissue near the base of the stipe (volva) and often “warts” or “patches” on its cap. A universal veil is always present in the genus *Amanita*, the genus responsible for most deaths. Such veil tissue may be missed due to weathering of the cap or carelessness in unearthing specimens.** The color of the cap is always given as that of the tissue underlying the universal veil.

The gilled fungi like the grocery-store buttons contain the most highly toxic species of the higher fungi. It is ironic that the more bizarre or unfamiliar forms are usually less toxic. The gilled *Amanita phalloides* (mycorrhizal & non-native) and *Amanita ocreata* (mycorrhizal & native) have caused almost all of the deaths from poisonous mushrooms in Western North America. *Amanita phalloides* may have been introduced on the rootlets of imported European cork oak among others.<sup>2</sup>

Pasadena, California, was the site of the first collection of *Amanita ocreata*. This species, unlike *Amanita phalloides*, is native to California. It is now found, although less frequently, in Oregon and southern Washington.

All deaths recorded in Western North America have been from gilled fungi, *except* for a single death reported from Oregon in 1994. The cause of that death was *Boletus pulcherrimus* Thiers and Halling, a mushroom with tubes instead of gills under the cap.<sup>3</sup> *Boletus pulcherrimus* Thiers and Halling had been known for years as *Boletus eastwoodiae* (Murrill) Saccardo and Trotter. The name had to be changed as Ms. Eastwood gave a good description, but mistakenly sent *Boletus satanas* Lenz to Dr. Murrill by error for the naming as “type” collection. That name “*Boletus eastwoodiae* (Murrill) Saccardo and Trotter” was therefore dropped, since it did not apply to the specimens, which already had the name *Boletus satanas* given by Lenz. Drs. Thiers and Halling gathered new material (dried specimens) for a new type collection and named it *Boletus pulcherrimus*. The older name could not be given back to this species, lest there be confusion as to what actual herbarium material was meant.

The majority of deaths worldwide are due to species containing amatoxins, most commonly in the genus *Amanita*.

### **Description and habitat of *Amanita phalloides* and *Amanita ocreata***

These two amanitas have caused all of the recorded deaths in California and most of those in the West. The majority of these were patients who had ingested *Amanita phalloides*. In their button stages, amanitas look like puffballs due to their enclosure in a universal veil, which surrounds the embryonic cap and stipe. As the mushroom matures and the cap expands, this veil is broken and usually leaves remnants on the cap in addition to those universal veil remains at the base of the stipe. The basal remnants are referred to as a volva if  $\pm$  cup-like. The veil tissue in some species tears into voyal rings or mealy basal patches. These volval structures may be fragile and, with careless collecting, left behind in the soil. If the mushroom is pulled up, rather than dug up, even a well-formed volva may be left in the earth. A partial veil from the cap edge to the stipe often leaves a  $\pm$  persistent ring on the upper portion of the stipe. *Amanita phalloides* and *Amanita ocreata* have rather well-formed sac-like volvas and annular rings; both may collapse on the stipe.

*Amanita phalloides* usually has a large cap (6 to 15 cm across) with no or only faint striations at the edge of the cap. The cap color varies from olivaceous or greenish brown to a brassy yellow brown, at times fading to dingy yellowish or buff in the presence of sunlight. The surface is slightly to moderately tacky when moist. As the mushroom expands beyond the button stage, the cap breaks the outer or universal veil, leaving a thin white volva at the base and a few thin white patches on the cap. Sometimes there are no veil remnants on the cap. The gills, stipe and volva are also white. The gills of mature specimens are free *i.e.* they do not extend all the way to the stipe. The gills are fairly close together. The length of the stipe approximates or somewhat exceeds the cap width, giving a somewhat stately appearance to the mushroom. The thin cup or sac-like free-margined volva may not be seen, unless the whole specimen is dug up. The volva often collapses on the stipe. A white secondary veil protects the young gills on the underside of the cap. When this partial veil breaks, it leaves a thin  $\pm$  membranous ring on the stipe. This veil remnant atrophies or disappears with aging.





*Amanita phalloides* photo © Michael Wood

Specimens of *Amanita phalloides* exposed to sunlight often bleach and appear yellowish or almost white at times. Some collections in drying fade to a dingy yellow, before becoming buff or almost white (usually having a slight sheen). However, a white color variant of *Amanita phalloides* is completely white from the start. The species then looks like *Amanita ocreata*. Suggestions that a specimen is **not** *Amanita ocreata*, but rather a pallid *Amanita phalloides* are: very large specimens with caps over 12 cm., occurrence with typical specimens, lack of any warm tan or brown tints on mature caps and fruiting prior to January. Sulfuric acid turns the gills of *Amanita phalloides* vinaceous to purple, but other amatoxic amanitas show either no change or a pink coloration. Potassium hydroxide solution dropped on to the flesh of *Amanita phalloides* shows no change in color; but dropped on to the flesh of *Amanita ocreata* the solution produces a bright yellow color.

The raw flesh of the *Amanita phalloides* “death cap” is sweet or very slightly acrid and cooked it is quite tasty.<sup>15</sup> There is no odor in the very early stages, but

for a brief time, it has an odd slightly floral honeyed odor (not totally pleasant) which deteriorates rapidly to an earthy odor and then to a ghastly “rotting potato” odor.<sup>21</sup> Some coastal State Parks near San Francisco have had hundreds of *A. phalloides* in an area probably less than an acre. At such a rare mass fruiting, the whole area smells somewhat sweet yet distinctly unpleasant. Moser describes the early odor as “honey-like” in European specimens<sup>22</sup> and Oudot as a withered rose (“rose fanée”).<sup>15</sup> Bon also describes the early odor of *A. phalloides* as “smell faint, then a little unpleasant, like a rose drying”.<sup>23</sup> This feature is not specific to amatoxin-containing species, however, since other potato stinkers include the innocuous *Amanita brunnescens*, *A. citrina* and *A. porphyria*.

The fruiting bodies of *Amanita phalloides* most commonly appear from September to March. However, spring and summer fruiting occur sporadically in irrigated areas. The mycorrhizal associate for *Amanita phalloides* is principally oak, but other trees occasionally act as mycorrhizal host. Additional hosts in the Pacific Northwest (PNW) include filbert and chestnut trees. The association with old orchards suggests the mycelia were brought over from Europe on the roots of various nut trees.<sup>9</sup> One collection in Washington was made near planted juniper and dogwood.<sup>10</sup> The distribution of *Amanita phalloides* now extends into British Columbia to the north and to San Diego, California to the south. It would be expected in Tijuana, Mexico just across the U.S. border.

Over the past 40 years, this species has thrived in years with heavy fog and wet fall weather along the Pacific coast. *Amanita phalloides* has spread gradually from its first sightings in the San Francisco Bay Area. It is now common at least 100 miles north and south along the California coast. Collections in Southern California are scattered. Although uncommon in mountain areas, specimens have been found in Granite Bay (Placer County) and near Sutter Creek in Amador County, California.<sup>8</sup> *Amanita phalloides* also occurs in Oregon, Washington and British Columbia, but *Amanita phalloides* introduction there and in Southern California are presumably from separate importations.

*Amanita ocreata*, the native coastal species (California, Oregon, southern Washington and Baja California, Mexico), is just as deadly as *Amanita phalloides*. The cap is 5-12 cm broad with a stipe 6-20 cm long: slightly smaller on the average than *Amanita phalloides*. The color of the cap is white at first, but ages buff-colored and tends to discolor a light warm brown or pinkish over the apex. There are no or only faint striations at the edge of the cap. The surface is moist to somewhat tacky when young. It may or may not display thin patches or wisps of universal veil tissue. Gills, ring, stipe and volva are white. The ring collapses early and may disappear. As in *Amanita phalloides*, the volva is thin,



rather sac-like and soon collapses on the stipe. On average, the size is slightly less than that of *Amanita phalloides*.



*Amanita ocreata* photo © Michael Wood

Looking much like the white variant of *Amanita phalloides*, *Amanita ocreata* fruits most commonly in late January, February, March and early April in California. The prime fruiting time in Oregon and Washington is May. It was thought to be endemic to California, but its range probably extends from the Canadian to the Baja, California border. Although Charles Peck first described *Amanita ocreata* in 1909, it was not until 1976 that the occurrence of amanitins was documented.<sup>5,24</sup>

*Amanita ocreata* has a rare salmon-colored form.<sup>20</sup> This aberrant color may possibly be due to a parasite. This color variant of *Amanita ocreata* could be confused with the edible *Amanita velosa*, especially since both species may be fruiting at the same time in the spring.

In California, *Amanita ocreata* is usually associated with coastal live oak, but it also occurs near or under old deciduous oaks. In Oregon and Washington, it often fruits in the spring after the low-land morel season along rivers, often in silt, but not in recently flooded areas.<sup>9</sup> North of the Columbia River it is commonly associated with filbert. The Hudson's Bay Company first introduced nut trees into the Vancouver, Washington area before 1840. There has been over 150 years for these nut trees to become an established part of the woodland flora in southern Washington.

## History of *Amanita ocreata* and *Amanita phalloides* in the West

In 1909, Charles Horton Peck described *Amanita ocreata* Peck from Pasadena, a white species. Peck recognized this species as distinct from the white form of *Amanita phalloides* (Fr.:Fr.) Link. var. *alba* Britzlem. *Amanita phalloides* and *Amanita ocreata* react differently with KOH. *Amanita ocreata* turns yellow with a drop of KOH solution. *Amanita phalloides* does not.

Harkness and Moore in their "Catalogue of Pacific Coast Fungi" published by the San Francisco Academy of Sciences in 1880 reported a collection of *Amanita phalloides* from San Rafael, a city in Marin County just north of San Francisco. However, Dr. Roy Hallings searched six bins of Harkness material now located at the New York Botanical Garden and could find no *Amanita phalloides*. A March 1884 A.J. McClatchie collection from Oak Knoll in Alameda County (east of San Francisco) labeled *Amanita phalloides* was *Amanita ocreata*.<sup>2</sup> William A. Murrill reported *Amanita phalloides* from Western North America in 1912.<sup>6</sup> These collections from Santa Cruz, California, were white to pallid mushrooms and appear to have been *Amanita ocreata*. Murrill was careful to note that he did not see any fresh material. It was a Professor Campbell at Stanford that originally thought the local species was a white form of *Amanita phalloides*.

Dr. Alexander H. Smith was the first to authenticate a US identification of *Amanita phalloides* in his first edition of "The Mushroom Hunter's Field Guide" published in 1958. This 1957 collection was from San Mateo County just to the South of San Francisco, but it later turned out not to be the oldest authenticated collection. The earliest correctly identified collection of *Amanita phalloides* in the United States goes back to November 2, 1938 and is in the herbarium of the University of California, Berkeley.<sup>2</sup> Dr. Isabel Tavares authenticated this collection, which came from the garden of the Del Monte Hotel in Monterey



County and was found fruiting under an imported shrub. The oldest authenticated collection of *Amanita phalloides* under a native tree (*Quercus agrifolia*, "coast live oak") was found at Observatory Hill near the Main Library, University of California, Berkeley Campus on December 31, 1945.<sup>2</sup>



*Amanita phalloides* photo © Fred Stevens

Dr. Harry Thiers, shortly after coming to San Francisco State University in 1959, noted specimens of *Amanita phalloides* in association with coastal live oak in three "bay area" counties—San Mateo, Santa Cruz, and Alameda. San Mateo and Santa Cruz Counties are south of San Francisco; Alameda County lies across the bay east of San Francisco. The first survey of *Amanita phalloides* in California (1963–1968 by Gary Breckon) suggested that *Amanita phalloides* was relatively uncommon. By 1970 massive fruitings sporadically occurred in San Francisco Bay under live oak.

Dr. Fred Stevens, one of Dr. Thiers' students, noted repeated San Mateo fruiting with cork oak, a tree that Dr. and Mrs. William Freedman had found to have

been planted by the San Francisco Water Department in 1943 and 1944 directly from European seedlings. The hypothesis was that *Amanita phalloides* from cork and other European oaks may then have adapted and transferred to California live oak rootlets, accounting for some of the increased sightings. Greg Wright in Southern California also reported *Amanita phalloides* in association with coastal live oak in Santa Barbara and later in Los Angeles.<sup>7</sup>

Because of these two common fungi, the large population and an influx of immigrants, California currently leads other North American states or provinces in mushroom fatalities. The first recorded US fatality, however, was on a New York tombstone for William Gould, his wife and son in 1838: “Poisoned by eating Fungi (toadstools)”.<sup>11</sup> The first supposed “mushroom” death in California was reported from Chico in 1871, but the symptoms suggest a plant or mixed poisoning rather than toxic fungi.<sup>12</sup> The oldest medical journal report comes from Boston in 1890. The mushroom that was the cause of death was certainly not the postulated “*Amanita verna*”, but most likely *Amanita bisporigera* G. F. Atk. or *Amanita -magnivelaris* Peck.<sup>13,13a</sup>

## The classical history of *Amanita phalloides* and related species

The ancient Greeks and Romans were well aware of the toxicity of *Amanita phalloides*. Their favorite mushroom was *Amanita caesarea* (Fr.) Schw., but *Amanita phalloides* had enough color to pass as such when cooked. The Stoic philosopher, Seneca the Elder, who committed suicide by order of the Emperor Nero, was well aware of the long delay before symptoms appeared. He wrote a satire on this deadly confusion 9-10 years after the death of the earlier Emperor, Claudius and had it sent to his friend Lucillus. This satire appears have been a veiled reference to Claudius's death, since he was in a position to know the details.<sup>14</sup> The Roman historian, Tacitus, stated that the emperor Claudius had been killed by his wife and niece, Agrippina, one of the theories being that she gave her husband a poisonous mushroom dish. Tacitus explained that she wanted a poison with a delayed action and one causing enough mental confusion that Claudius would be unable to arrest her. The accounts of Tacitus, Suetonius and Dio Cassius differ, but all agree that Agrippina poisoned Claudius. Dio Cassius reports that a particularly large mushroom cap was specifically pointed out to Claudius.

Vaillant's term “amanite phalloïde” in 1727 was given the binomial *Amanita phalloides* almost a century later by Fries. The “phallus-like” refers to the mushroom arising from its volva or “death cup”, much as a phallus (the erect

penis) rises from the scrotal sac.<sup>15</sup> The phallus, considered a fertility symbol, was carried in the solemn Dionysiac processions of ancient Greece. The genus *Amanita* was named by Persoon in 1801 after Amanos (Amanos), a small moist mountain range in Southeastern Turkey.

## Mushroom poisoning case registry

In present day poisonings, permission of the patient to release all data to the North American Mycological Association (NAMA) is very important. Currently, patient confidentiality is a hot medical-legal topic and has led to new and often absurd rules. The chief of the emergency department refused the author's request to review the records of a hospital patient that he had seen a week earlier in its emergency room: the grounds being that this hospital's former staff president was no longer an active member of the staff and the patient had not signed a separate release of information for outside consultation. (That refusal included the author's own notes!) The Poison Control staff relayed the needed information to the author the following week and the matter was never referred to higher authorities.

NAMA mushroom poisoning report forms are available on NAMA's web site <http://www.namyco.org>.

## “Look-Alike” mushrooms

Most “look-alikes” of toxic and edible fungi only superficially resemble each other, but without close inspection in good light, terrible errors have been made. Deadly examples of mistaken identity include: picking the greenish *Amanita phalloides* instead of the yellow *Amanita (calyptroderma) lanei* (Murrill) Sacc. & Trott. with its single thick white patch of universal veil on the cap, picking a young *Amanita smithiana* Bas instead of *Tricholoma magnivelare* (Peck) Redhead (“matsutake”) with its marked heft and toughness or picking a white or pallid amatoxic *Amanita* species instead of the edible *Volvariella speciosa* with its pink spores. *Volvariella speciosa* grows in grassy areas and the stiff, thick cup of the volva flares away from the stipe and only collapses on it when old.

*Volvariella speciosa* also has a fairly distinctive earthy odor with a slippery grey cap when young. Since this species is eaten fairly often in Hawaii, confusion of it with the presumably amatoxic, white to gray-marbled, *Amanita marmorata* subsp. *myrtacearum* Miller, Hemmes and Wong<sup>19a</sup> found in the Hawaiian islands is a possible threat. However, the habitats are usually quite distinct: grass and



lawn for *Volvariella speciosa* and eucalyptus and acacia forests for *Amanita marmorata*.



*Volvariella speciosa* photo © Fred Stevens

*Chlorophyllum molybdites*, *Chlorophyllum (Macrolepiota) rachodes*, and *Chlorophyllum brunneum* are very similar in appearance, having large caps with buff “shingles” (large overlapping scales), etc. *Chlorophyllum molybdites* causes severe nausea, vomiting and diarrhea; *Chlorophyllum rachodes* and *Chlorophyllum brunneum* are good edibles. When thoroughly cooked, *Chlorophyllum molybdites* may be tolerated by some people, at least in small quantities. It is quite tasty reportedly, but hardly worth risking severe gastrointestinal poisoning. The spores are dull green in contrast to the white spores of *Chlorophyllum rachodes* and *Chlorophyllum brunneum*. The three species have scaly caps and smooth stipes, the flesh and surfaces often staining a reddish/orange when bruised or scraped. *Chlorophyllum molybdites* grows most commonly on lawns in warm areas of the U.S. and Mexico, often in large fairy rings.



*Chlorophyllum molybdites* photo © Michael Wood

Puffballs, edible when young and white through the center, have a solid undifferentiated interior, although there may be a slightly distinct sterile base. An *Amanita* button, when cut in half, will show the embryonic gills and a faint outline of the maturing mushroom, which will later rupture the universal veil that covers it. Puffballs should be cut in half, when collected or prepared for food, not only to distinguish them from *Amanita* buttons, but also because a yellow, brown, dark purplish or blackish center indicates spore maturation that will probably cause GI distress.

Pot-hunters may confuse the toxic *Amanita pantherina* sensu auct amer. with brown *Agaricus* species. *Amanita pantherina* in the United States varies markedly. What may be called the “pantherina complex” in Western North America describes a number of different species, including *Amanita pantherina* (DC.:Fr.) Krombh., *Amanita pantherina* var *pantherinoides* (Murrill) Dav. T. Jenkins and possibly *Amanita multisquamosa* Peck. *Amanita multisquamosa* is



primarily an eastern species, but it may additionally occur in the PNW. Other members of the group have not yet been sorted out.

## Identification aids

Visual findings are only part of the gross anatomical features. Texture, odor, taste and surface features such as stickiness, “hairs”, scales and veil patches are very helpful. The edible *Marasmius oreades*, for example, has a felty texture and pliant tough feel, which is very different from the fragile species that a pot-hunter is also likely to find on lawns. None of the toxic clitocybes e.g. have a velvety pliant cap or a tough stipe that bends so easily without breaking. The spicy odor and the relative toughness of *Tricholoma magnivelare* ("Matsutake") help distinguish this taxon from the kidney-toxic *Amanita smithiana*.

Common sense, however, dictates that one should best avoid tasting a mushroom suspected of containing amanitins.

If there is uncertainty about identification, a spore print may help. The stipe is cut off and the cap is placed onto paper with the gills facing down. A glass, cup or saucer is placed over the specimen so that air currents do not waft away the spores. Adding 1-3 drops of water to overly dry caps may help to get a better spore print (usually obtained in 1-4 hours, but may take overnight). Spore prints may be the same as the gill color or differently colored. The author uses white paper and a glass slide to bring out the spore print more clearly.

The use of a slide allows determination of amyloidicity (presence of starch), as determined in the iodine-rich Melzer's solution. The possibilities are amyloid (grayish blue color), dextrinoid (a reddish brown color) and inamyloid (no color except for the yellow tan of Melzer's solution). The test may be done without a microscope, but weak stains may be misleading. *Use of a magnifying glass or microscope is recommended.* The high power enlargement of a Hastings triplet is useful in the absence of a microscope.

Confusion in some genera is really due to a lack of attention to the gross anatomy of fungi. Surface features of mushrooms often change with age, usually with loss of color, viscosity and features of any veil or scale elements present. Scales are innate to a cap or stipe; patches that may be wiped away are secondary to partial or universal veils; a partial veil runs from the edge of the cap to the stipe and a universal veil covers the entire fruiting body. A viscid cap in age as it dries, for example, may display only shininess and, if the examiner is lucky, there are "pasted" leaves or debris on the cap. On moistening the cap, a tacky feeling may



be felt with the "lip test" (a slight pulling of the lip as the mushroom is removed from it). Mushrooms that are "hygrophanous" have rather thin-fleshed caps that dry a lighter color. Since details tend to be lost in aging, identification is easier if both young and older specimens are examined.

## Guidelines for pot-hunters

1. Learn the known toxic fungi first and the genera in which the major poisonings are found— *Amanita*, *Lepiota*, *Galerina*, *Conocybe*, *Inocybe*, *Clitocybe*, *Paxillus*, *Hypholoma*, *Psilocybe*, *Cortinarius* and *Gyromitra*.
2. Eat new edible fungi in small amounts at first.
3. Be cautious offering wild fungi to guests even if you have had no problems. Idiosyncratic GI reactions are common, although typical allergies such as hay fever, asthma and hives are uncommon. There are no reports to date of mushrooms causing allergic (anaphylactic) shock.
4. Remember that many mushrooms cause trouble when eaten raw. These are usually GI upsets. Gyromitrin and some other toxins are destroyed, at least in part, by cooking. The Europeans have found that, on rare occasion, a number of common genera including "edible" amanitas are able to provoke red blood cell destruction when eaten raw or partly cooked. This should be true of all western hemisphere "blushers" and of species referred to by the European name *Amanita franchetii* (Boud.) Fayod.<sup>13a</sup>
5. Don't be gluttonous. Small amounts of some toxins may be tolerated.
6. If a canned mushroom has been ingested, call attention of physicians to botulism if there are pupil dilation, muscular weakness or central nervous system symptoms. There is an anti-serum available, which may keep victims off a respirator.
7. Heed the advice of Elias Fries in his *Observationes mycologicae*— "Illustrations appeal to idlers who do not have energy enough to study descriptions".<sup>16</sup> Using a poor photograph of *Agaricus augustus*, but not reading the description below the colored plate, two California victims misidentified what was actually *Amanita phalloides*. Both required liver transplants.



*Agaricus augustus* photo © Michael Wood

## Insecticides and other toxic sprays

Collectors should watch out for insecticides and sprays used to control unwanted plants. Places, such as golf courses, walkways and railroad tracks are often heavily sprayed. Most insecticides are synaptic poisons and acute reactions are much more common in children. Mushrooms from any of the above sites should probably not be given to anyone aged 10 or under. Young children may also lack the mature enzyme systems needed to break down these chemicals for elimination from the body.

The most dangerous of the insecticides available are the organophosphates. Earlier organophosphates, such as sevin and sarin, were used by Iraq in Saddam Hussein's war against the Kurds. Organophosphates, such as chlorpyrifos, are esters of phosphorus having varying combinations of oxygen, carbon, sulfur and nitrogen attached. They inhibit cholinesterase and produce mild to severe muscarinic and nicotinic effects including headache, vertigo, nausea, blurred vision, tearing, constricted pupils, slow heart beat, muscle twitching, difficulty walking and excessive sweating and salivation. Carbamate insecticides, such as

propoxur, produce similar effects, but are much less likely to be toxic.<sup>17a</sup> Other insecticides are likely to produce symptoms (other than possible GI effects) only with exposures to amounts far higher than found in fungi. Nonetheless, bizarre or possible neurological symptoms should prompt a consideration of insecticide poisoning. *The symptoms of organophosphate and carbamate poisoning are similar to those produced by muscarine in some fungi, particularly inocybes and cltocybes.* (See the muscarin group toxins.)

## Guide to the identification of mushroom poisoning by symptoms

The latency (delay from eating the mushroom to first symptom) in the list below varies considerably. Early onsets with GI symptoms reported for toxins normally having long latency periods suggests that the mushrooms have been spoiled or eaten with other mushrooms that have early GI effects. Not all symptoms listed need occur in any one patient. Some of the mushrooms associated with a long latency period occasionally produce early gastrointestinal symptoms in some persons, but not in others. These early symptoms may be due to compounds other than the primary toxin or may represent a biphasic response to the primary toxin in susceptible patients.

### Onset of symptoms less than 4 hours after ingestion:

1. Nausea, vomiting, diarrhea, abdominal cramping with no progression beyond GI involvement—**GI toxins, irritants and idiosyncrasies.** A few GI symptoms are delayed. See #6 in the next paragraph (onset over 4 to 14 hours from ingestion).<sup>17b</sup> (many species)
2. Drowsiness, ataxia, occasional nausea, obtundation (may alternate with hyperkinetic states), muscle fasciculation, seizures. **Ibotenic acid in children.**
3. Nausea, confusion, ataxia, feelings of increased strength or euphoria, muscle fasciculation, sensations of floating or color changes, rarely progressing to coma or seizures as in children. **Ibotenic acid in adults.** (*Amanita muscaria*, *Amanita pantherina* complex)
4. Nausea, vomiting, diarrhea (usually starting in 1-4 hours); progressing to severe malaise, weakness, sweating, cold extremities, confusion, cardiovascular collapse or coma associated with autoimmune hemolysis and damage to kidney and other major organs. There is usually a history of earlier ingestions without symptoms. **Autoimmune hemolysis.** (*Paxillus involutus*).

5. GI symptoms at 4 hours rarely, usually a more delayed onset. Associated kidney failure. See *Amanita smithiana* below (#5 of “onset of symptoms after 4 hours from time of ingestion”).
6. GI symptoms, usually by 4 hours along with **hemolysis**—in association with the ingestion of **raw or poorly cooked mushrooms, especially amanitas**. Particularly suspect are “blushers” such as *Amanita novinupta* and those amanitas referred to the European name *Amanita franchetii* (Boud.) Fayod.
7. Euphoria, occasional panic reactions, hallucinations, dilated pupils and rarely nausea to the point of vomiting. *Look for seizures, fever in children.* **Psilocybin.** (*Psilocybe*, other dark spored genera)
8. Sweating, salivation, nausea, teary eyes with dilated pupils, slow heart rate and sometimes diarrhea and increased urination. Large ingestions may cause pulmonary edema and seizures. **Muscarine.** (*Clitocybe* & *Inocybe*)
9. Within 5-30 minutes of taking alcohol after ingestion of the offending mushroom, the occurrence of flushing, throbbing in the temples, headache, fast heart rate and occasionally nausea and vomiting. Symptoms may recur after alcohol consumption for up to 3 days, but are less severe over time. **Coprine.** (*Coprinus*)
10. Rare onset at 1-4 hours of malaise, vomiting, headache, dizziness usually occurring after 6 hours—see **gyromitrin** below.
11. Malaise, GI symptoms, severe headache, generalized aching, flushing, incoordination, muscular weakness, difficulty walking and confusion. One hour after ingestion (2 cases). Poisoning with *Stropharia coronilla*.

### Onset of symptoms after 4 hours from time of ingestion:

1. Sudden malaise, GI symptoms and often severe headache & dizziness at 2-25 hours after ingestion of “false morels”. The latency period, which is most commonly 6-12 hours, has been reported as late as 40 hours. Symptoms may progress to muscle cramps, shock, methemoglobinuria, liver failure, fever and CNS symptoms including convulsions. Hemolysis, hemoglobinuria and kidney failure are uncommon. **Gyromitrin.** (*Gyromitra*, *Helvella*; other ascomycetes?)
2. Diarrhea, nausea, vomiting, abdominal cramping, malaise usually at 10-16 hours (rarely 5-36 hours) after ingestion progressing to liver failure and rarely kidney failure with decreased urine, shock, acidosis etc. (*if severe renal failure, consider alternatives such as 3, 4 & 5 below or a novel toxin*) **Amanitin.** (*Amanita phalloides*, *Amanita ocreata* etc.)



3. Diarrhea, nausea, vomiting 4-10 hours after ingestion (reports range from 5-9 hours) with elevated blood liver enzymes and rare progression to liver failure associated with a description of yellow brown mushrooms clumped on living trees or dead stumps. No North American cases yet reported, although this species is common both in Europe and North America. Poisoning with *Hypholoma fasciculare*.
4. GI symptoms sometimes at 4-12 hours<sup>18</sup> with much later burning and dryness of the mouth at 2 days to 5 weeks progressing to severe thirst, frequent urination of an increased quantity of dilute urine; later having reduced quantity of urine and kidney failure. *Cortinarius rubellus* Cooke. [Poisonings have not clearly occurred in Western North America, although a few reports are suggestive of its bipyridyl toxins.<sup>19</sup>]
5. GI symptoms at 4-12 hours followed by kidney failure. (One case of liver failure.) Further characterization of the syndrome and the pathological findings are needed. Collection of ±white mushrooms including presumed matsutake. *Amanita smithiana* Bas.
6. GI symptoms only, but having latencies as long as 4-14 hours on occasion—for example, the vase-shaped *Gomphus floccosus, kauffmanii* and *bonarii*. Other species such as those in the *Armillaria mellea* complex and some boletes have also had delayed symptoms in Europe.<sup>17b</sup>

## Group I toxins: Amatoxins

### The amatoxic group

#### General description and occurrence

Fungi containing amanitin as their major toxin include species of *Amanita*, *Lepiota*, *Galerina*, *Conocybe* and possibly *Cystolepiota*. Regionally, there are 18 names or descriptions applied to fungi that are known or suspected to contain amanitin. These are *Amanita phalloides*, *Amanita ocreata*, *Amanita bisporigera* G.F. Atk., *Amanita virosa* sensu Schalkwijk-Barendsen (Sandy Lake, Alberta; possibly a 4-spored collection of *Amanita bisporigera*), *Amanita virosa* sensu Guzmán (Mexico), species close to *Amanita bisporigera* but with a negative or weak KOH reaction (Chiricahua Mountains, Arizona and neo-volcanic regions of central Mexico), *Amanita marmorata* ssp. *myrtacearum* Miller, Hemmes & Wong (Hawaii), *Amanita magnivelaris* Peck (per Guzmán, Mexico), *Amanita arocheae* Tulloss, Ovrebo & Halling (Mexico), *Galerina marginata* (Fr.) Kühner, *Galerina venenata*, *Conocybe filaris* and the six *Lepiota* names given below. Some of the species have not had amanitin assays or produced known poisonings. In addition, the taxonomic status of some of these names is unclear. Guzmán and his colleagues tend to recognize European names, which may not be appropriate for New World material.

Species identified as *Amanita virosa* may be completely or at least largely the 4-spored form of the earlier-fruiting *Amanita bisporigera*, which has only 2 spores. There is a shift from 2 to 4 spores on each basidia as the fruiting season progresses. Specimens identified as *Amanita bisporigera* tend to be smaller than specimens identified as *Amanita virosa*. These are the species most commonly identified as causing amatoxin poisoning in Mexico.

#### The Amanitas

Species of *Amanita* are medium to large, often stately in stature with rather long stipes.<sup>18a</sup> The gills are typically free (not attached to the stipe), although there is some variation. In their button stages, amanitas somewhat resemble puffballs due to their enclosure in a universal veil surrounding the embryonic cap and stipe. As the cap expands, this veil is broken and usually leaves remnants on the cap and on the lower stipe with or without a clear-cut volva (or “cup”) at the base

of the mushroom. Some amanitas have volval remnants in the form of basal rings, collar(s) or as a granular fragmentable “cup”.

Rain frequently washes off the thin veil remnants on the caps of *Amanita phalloides* and *Amanita ocreata*. Their thin collapsing volvas are readily left behind in the ground. A secondary veil from the edge of the cap to the stipe may or may not leave a ring on its upper portion. The spore print is white.

*Amanita phalloides* and *Amanita ocreata* are described in detail under the earlier heading — Description and habitat of *Amanita phalloides* and *Amanita ocreata*.



*Amanita phalloides* photo © Michael Wood

The white amanitas more or less fit the following description. The cap, veil, gills, ring, stipe and a ±sac-like volva are white. The caps are conic to convex or flat. The ring is usually skirt-like, often torn and may be persistent with age. The mushrooms are statuesque having caps (3) 5-9.5 (16) cm. broad and stipes (7) 14-

20 (24) cm tall. *Amanita bisporigera* Atkinson is generally smaller than the others with caps 5-10 cm across and rarely has a slight tan tinge to the cap.

An additional amatoxic species, which looks like a grey or grey-brown *Amanita phalloides* but without the olive tones, occurs in northern Mexico: *Amanita arocheae* Tulloss, Ovrebo and Halling.<sup>13a</sup> This greyish “hongo gris” is distributed from north central Mexico to Andean Columbia. This species, *Amanita arocheae* Tulloss, Ovrebo and Halling, is also associated with oak. It has been assayed (as presumed *Amanita phalloides*) and found to contain amanitins. Presumably this species and the next two have produced toxic events in man or animals, but the author has no data on actual poisonings. In any event, the offending mushrooms would almost certainly have been misidentified as *Amanita phalloides*.

*Amanita marmorata* ssp. *myrtacearum* Miller, Hemmes & Wong grows in Hawaii’s *Eucalyptus* and *Acacia* forests along with *Casuarina* (Iron Wood) and *Melaleuca* (Paper Bark). This presumably amatoxic species has a cap, which is pure white to greyish and marmorate (blotchy or veined like marble). The ring is membranous and may disappear at maturity. The gills are close together and remain white. The volva is white and saccate.

*Amanita longitibiale* Tulloss, Pérez-Silva & T. Herrera, another of these presumably amatoxic species, is found in the pine and fir of neo-volcanic central Mexico. This slender member of section Phalloideae (Fr.) Quélet has caps that are pale yellow, aging pale grey to umbrinous.<sup>13a,b</sup>

In California, both *Amanita phalloides* and *Amanita ocreata* cause deadly encounters not infrequently in wet years; in Europe a dry year with consequently rare amatoxin poisonings is usually a good year for local wines. No such association has been shown in California, but pinot noir would be the most likely grape to show such a correlation. There has been at least one death from *Amanita ocreata* poisoning in Oregon.<sup>19</sup>

## The Lepiotas

The genus *Lepiota* also has white spores and free gills, but with no volva. A minority have an annulus or an obvious annular zone. The genus *Lepiota* in the narrow sense contains a preponderance of small and often, fragile, fungi; typically having caps less than 6 cm wide (usually 1-4 cm across). These smaller mushrooms contain the amatoxins. Their caps are dry and often have small blackish, brown or reddish brown scales forming concentrically as the cap disc expands. The scales do not wash off, being intrinsic parts of the cap. Some species



bruise an immediate bright red; others do not bruise, or more slowly bruise some shade of brown to blackish. The white or yellow gills are close together and the stipe breaks cleanly from the cap. A ring may be present, but it often collapses to just a bare outline on the stipe and is rarely prominent. The stipe below the ring area is usually scaly. There is no volva. The spore print is white to cream. All of the species considered here have European names and many of our species are probably not the same. The toxic species are: *Lepiota helvelola*, *Lepiota josserandii*, *Lepiota castanea*, *Lepiota subincarnata* and possibly *Lepiota clypeolaria*.

The numerous *Lepiota* species are even more difficult than the *Amanita* species to sort out: whether they have produced poisoning or not, whether amanitin has been detected or not and whether U.S. counterparts are the same as the European species. Dr. Else Vellinga presented an account of the genus based on the European work of G erault, Girre and Besl at the Mycological Society of San Francisco October 2001 and noted that most lepiotas have an unpleasant rubber-like smell, with a sweet component. However, most of the toxic ones (*Lepiota josserandii*, *L. helveola*, *L. subincarnata*) often have a seductively sweetish smell.

Two small species on the west coast are highly toxic—*Lepiota josserandii* Bon & Boiffard (= *helveola* sensu Josserand) and what has been assumed to be *Lepiota helveola* Bres. (= *helveola* sensu Huijsman). Although our *Lepiota helveola* looks remarkably like the print of this species in Fries' Icones, some of the microscopic features suggest that the Icones' fungus may actually be *Lepiota subincarnata* Lange, another European taxon.<sup>25,26</sup> Both *Lepiota helveola* and *Lepiota josserandii* have caused severe, but not fatal poisonings in California. *Lepiota josserandii* has caused one death in Vancouver, British Columbia.<sup>26a</sup> *Lepiota "helveola"* also occurs in Mexico.<sup>27</sup>

The possibly toxic European *Lepiota clypeolarioides* Rea, or one very like it, is present along our west coast. This species, however, is so poorly delineated in Europe, that it is not clear as to exactly what mushroom the name refers. Dr. Joe Ammirati gives a description of U.S. material from California in *Poisonous Mushrooms of the Northern United States and Canada*.<sup>125</sup>

*Lepiota castanea* Qu el. and *Lepiota oregonensis* are probably toxic. Two other species, *Lepiota brunneoincarnata* and *Lepiota felina*, may be toxic. Other species, which are closely related to known toxic European species such as *Cystolepiota heteri*, are suspect. It is also not clear that *Cystolepiota heteri* occurs in the United States. *Lepiota elaiophylla*, a greenhouse mushroom, may also be toxic; if so, the toxin is probably not an amatoxin.

Only *Lepiota helveola* and *Lepiota josserandii* have produced definite amatoxin poisoning. Both of these species have an annulus or annular zone, although the thin tissue may weather away in age.

*Lepiota helveola* has a more or less vinaceous medium brown cap except between the scales where the underlying tissue is whitish, 1.5-4 cm wide, dry, convex becoming flatter in age, with or without an umbo (small knob) at the center, with flat concentric scales almost all towards the margins of the cap. The gills are white to a yellowish buff and free from the stipe. The cap breaks readily free from the stipe, which is 2.5-4.5 cm long and 4-9 mm wide. There is a more or less membranous ring, which may collapse on the stipe; above it, the whitish stipe is smooth and below it, the stipe is thinly covered with scales, which are concolorous with the cap.



*Lepiota helveola* © Tom Duffy

*Lepiota josserandii* has a dry cap, 2-4.5 cm wide, convex becoming flatter in age, with or without an umbo, the cinnamon brown to dull reddish cuticle broken into concentric scales over the whitish underlying tissue. These flat scales develop as the cap enlarges; they are more conspicuous than in *Lepiota helveola* and begin closer to the center of the cap. The gills are white to pale yellow, close together and free or slightly attached to the stipe. Young specimens have a cobweb-like partial veil (from the cap margin to the stipe), which quickly collapses to a ragged line on the stipe with aging. Below the ring area, the stipe is scaly and concolorous with the cap. The stipe is 2.5-5 cm long and 5-10 mm wide.

Most of the edible members of the *Lepiota* family are in the genera *Macrolepiota*, *Chlorophyllum* and *Leucoagaricus*. None of these three genera is known to contain amatoxins, although *Chlorophyllum molybdites* may cause severe vomiting and diarrhea.

### The Galerinas

*Galerina autumnalis* (Pk.) A. H. Smith & Sing. and *Galerina marginata* (Fr) Kühner are now considered conspecific species. The difference between the two species was thought to be the presence of a gelatinous cap layer in *Galerina marginata*, but this feature is minimal and inconstant. The proper species epithet is *marginata*, since that name has priority under the International Botanical Code of Nomenclature.



*Galerina autumnalis/marginata* photo © Michael Wood



*Galerina marginata* is a small fungus with convex to flat, viscid to moist caps 1.5-6.5 cm across and honey-colored to brown, the disc usually the darkest, the cap fading to dull tan or buff. The flesh is thin, white to ochre brown. The gills are broadly attached to subdecurrent on the stipe, close together and usually concolorous with the cap. The stipe is 3-9 mm across and 3-8 cm long with an evanescent thin fibrillose-membranous ring. The stipe is paler than the cap above the ring, buff to pale brown and below is  $\pm$  concolorous or more reddish brown,  $\pm$  shaggy below and with an evanescent coating of pale fibrils at the base, often displaying a white mycelial tuft. This species fruits scattered to abundantly and may be clustered on  $\pm$  decayed logs, buried wood, in deep moss or occasionally along woodland paths.

*Galerina marginata* as identified in California had a positive Meixner test for amatoxins in only 1 of 3 specimens tested at the MSSF Fungus Fairs; an additional 3 tested by the author had 2 positive tests (one weakly so). In Europe, some specimens of *Galerina marginata* have also lacked amatoxins. The Internet site [www.uio.no/conferences](http://www.uio.no/conferences) notes that European and American taxa were compatible in culture studies done at Ron Peterson's laboratory in Knoxville; additionally, nuclear rDNA studies done by Gulden, G. et al. showed that material identified earlier as *Galerina autumnalis* or *Galerina marginata* were compatible with a single species.<sup>23a</sup>

The fact that some species identified as *Galerina marginata* (or *Galerina autumnalis*) are negative for amatoxin is quite misleading as this species is often deadly in small amounts and has been postulated to contain other toxins. A small piece chewed to confirm its mealy flavor provoked a hospital stay for one experimenter. In one of the exceedingly rare cases of severe poisoning in the case of a "grazing child", a small girl was killed by what appeared to be a similarly small piece.<sup>27a</sup>

*Galerina venenata* with a lighter reddish-brown cap fades to near white and is usually grouped in grassy areas (possibly on buried wood). This species has been reported from Oregon and Washington and would be expected in British Columbia. A few have been found on lawns in Idaho. Although the species differs from *Galerina marginata* in color and habitat, molecularly it does not and Gulden et al placed all of these species along with *Galerina oregonensis* under the subgenus *Naucoriopsis*, which then forms a distinct infrageneric unit.

*Galerina venenata* Smith has the rare historical novelty that the species (or at least variety) was discovered and named only after it caused a poisoning. Two Oregon artists in 1953 thought that they had found the edible *Amanita fulva*

(Schaeff.) Fr., but they encountered the genus *Galerina* instead. The specimens could not be identified by the Oregon Mycological Society and were air mailed to Dr. A.H. Smith, who later named this new fungus *Galerina venenata*.<sup>28</sup> Galerinas are most common in the fall, but may fruit in the spring. The spores are minutely wrinkled or ornamented under the oil immersion lens of the microscope and have no germ pore; the spore print is rusty to tobacco brown.

### *Conocybe filaris*

*Conocybe filaris* (Fr.) Kühner is a small brown fungus has a smooth to finely wrinkled cap. When moist, the gills may be seen through the thin flesh at the margins as fine striations; when dry the cap becomes ± opaque and dull tan. The moist cap is typically brown to tawny brown, sometimes an attractive orangish brown. The margins may be a lighter cinnamon, medium brown or yellowish brown. The cap is ±conical when young, but expands to convex or flat. The cap is typically 1-2.5 cm across when mature; the young conic caps may measure only 3-4 mm across and up to 4-5 mm measured vertically. The pallid gills are thin and close together, soon becoming brown with an orange to yellow tinge from the cinnamon brown spores. The gill edges are finely fimbriate when young.



*Conocybe filaris* photo © Fred Stevens

The fragile stipe is hollow, usually longer than the cap (1.5-5 cm) and has a width no more than 3mm. The partial veil leaves a buff to white, skirt-like, loosely attached, fibrillous-membranous ring with its upper surface striate-grooved where it separated from the gills. It is somewhat felty and may be tinged with the color of the adjacent stipe. The ground color of the scurfy stipe above this ring is a light orange-yellow, yellow brown or light brown and below the ring the more fibrillose stipe is dark brown to dark rusty brown. The spore print is cinnamon to nutmeg brown.

This potentially deadly species is so small that it is unlikely to cause death in an adult. Dr. Harry Thiers of San Francisco State University reported a non-fatal poisoning in a child to the Mycological Society of San Francisco. The poisoning occurred not long after Dr. Thiers moved to San Francisco (probably in the early 1960's). The child was "grazing" on the lawn at San Francisco State University, but no clinical details are available.<sup>29</sup> Not all specimens of *Conocybe* (*Pholiotina*) *filaris* contained amatoxins on the basis of Meixner testing performed by the

Mycological Society of San Francisco (MSSF) and by the Los Angeles Mycological Society (LAMS).<sup>30</sup>

There appear to be no other reports of illness from this mushroom, which is found in grass, moss and on wood chips or soil near rotting wood or plant debris. There are a number of similar *Conocybe* species, which have not been studied for possible amatoxins.

## Ethnicity and amanitin poisoning

Gordon Wasson, who researched the various cultural uses of mushrooms, divided societies into “mycophobic” and “mycophilic” (fearing and loving mushrooms). He considered the mycophobic cultures to include the Scandinavian countries, Great Britain, Canada and the United States. Countries considered mycophilic included France, Austria, Italy, Switzerland and the Slavic countries. In those countries, photographs and descriptions of both toxic and edible fungi often decorate the walls of primary schoolrooms.

Americans of Italian descent have had more than their share of *Amanita phalloides* poisoning, because of their like for “coccoli” or “coccora”. Italian-American pickers may mistake *Amanita phalloides* for the edible “coccoli”—yellow or orange-yellow mushrooms described by Atkinson and Ballen as *Amanita calyptroderma*. The correct name under current ICBN rules is *Amanita lanei* (Murrill) Sacc.& Trott., but this name under Murrill’s concept refers to only the orangish fall species.<sup>25</sup> The similar pale yellow mushrooms found in the spring are probably a separate and unnamed species; these are also eaten as “coccoli”.

*Amanita phalloides* looks vaguely similar to the orange, often grayish, *Amanita lanei*, although the latter's thick membranous volva, often fish-like odor, thick single white membranous patch on the cap and striations at the cap border should suffice to distinguish them. *Amanita phalloides* has multiple thin patches of universal veil on the cap or sometimes none at all, should its fragile tissue completely weather away; the thin volva often collapses on the stipe. The thick “poison cup” is more evident in the case of the edible *Amanita lanei*. *Amanita phalloides* is not easily mistaken for the pale yellow spring form even if still fruiting, provided immature specimens are not collected. The “eggs” or young buttons are often collected but the difference from *phalloides* buttons is easily missed, even when cross-sectioned. Avoid the buttons entirely.

The greenish-yellow *Amanita phalloides*, often a dingy color, may approximate that of *Amanita lanei* quite well in poor light. At least one very experienced California pot-hunter picked one *Amanita phalloides* along with many *Amanita lanei* as darkness descended and greed gained the upper hand. A few mushrooms were eaten that night; the uneaten *Amanita phalloides* was found the next day. The rest of the collection was thrown out in horror.

The popularity of this mushroom is based on the orange *Amanita caesarea* of Mediterranean Europe, a species not found in the U.S., although a similar one may be found from Quebec to Texas (*Amanita jacksonii*) and another one in the Southern Rocky Mountains. Another species, *Amanita colchiseiana* (Tulloss nom. prov.), is found in the Chiricahua Mountains of Arizona. The Mexican species often called “caesarea” has been named *Amanita basiana* by Dr. Guzmán and Rodríguez-Guillén.

Americans of Asian extraction are poisoned more frequently than the general population because the amanitas involved resemble edible Asian species—the often off-white *Amanita princeps* and *Amanita chepangiana*. Both species tend to have a little yellow or yellowish brown, sometimes with a faint olive tinge—confined largely to the central area of the cap. Otherwise the pileus is white and it may be entirely so. Dr. Desjardin notes that both species are sold in markets, especially in Laos and Thailand, and that they tend to look somewhat like *Amanita phalloides* when the central area is colored yellowish brown.

Americans of Mexican extraction are also more likely to be poisoned than the general population by *Amanita phalloides*, since the amatoxic taxons in Mexico are typically white with the exception of the greyish *Amanita arocheae* Tulloss, Ovrebo and Halling. *Amanita phalloides* with its brassy green cap is not found or is exceedingly rare in Mexico. The problem is that amanitas are traditionally a substantial part of the mushroom diet in some areas of Mexico as well as *Hebeloma*, *Lyophyllum* and *Melanoleuca*—fungi, which are all very low on the list of US pot-hunters. Indigenous peoples in Mexico identify them by local tradition, habitat and over-all appearance. Four deaths from *Amanita phalloides* in San Diego were caused by a delay to hospital admission, because the men were fearful of their illegal immigrant status. The San Francisco Chronicle noted January 6, 2007 (page 1, section B) that six members of a family were seriously poisoned by *Amanita phalloides* because the grandmother, used to foraging in Mexico, failed to identify the *Amanita phalloides* picked.

A number of white amanitas as well as others are eaten in Mexico. The edible *Amanita tuza*, for example, has a very thick ±cottony volva similar to our

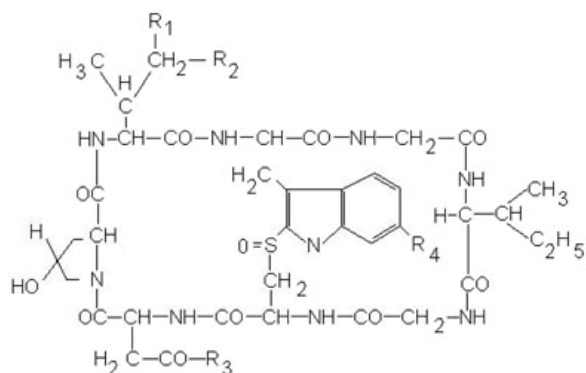


*Amanita lanei* (*calyptrata*) and is equally popular. This edible taxon would not seem likely to be confused with *Amanita magnivelaris* or the other amatoxic fungi noted above, but occasionally misidentifications occur. *Amanita tuza* not only has a very thick volva, but it has marked, slightly raised striations at the edge of the cap, which are lacking in *Amanita magnivelaris* and the other toxic taxa. *Amanita tuza* grows just below the surface of the ground initially and forms mounds resembling those of gophers (thus its species epithet). Other edible white species with a thick volva are close to the Mexican version of the edible (but orange) "Caesar's mushroom" and include *Amanita basii* Guzmán & Rodríguez-Guillén as well as *Amanita laurae* and *Amanita yema* of the same authors.<sup>13a</sup> With this abundance of edible amanitas in Mexico, latino immigrants to California continue to be in particular danger of poisoning by *Amanita phalloides* and *Amanita ocreata*.

In central Mexico, some folk beliefs are 1. Cook mushrooms with a garlic clove. If the garlic turns blue or black, the mushrooms are poisonous; 2. Do not eat mushrooms which grow on dung; 3. Do not eat mushrooms if the flesh turns a different color when cut; 4. Do not eat mushrooms with an unpleasant odor.<sup>31</sup> It is not surprising that large poisonings occasionally occur, such as the 16 or so people poisoned by white amanitas in 2005 and reported in the Spring issue of the 2007 *McIlvainea*. Eight of these Chiapas victims died.

### **Toxins of *Amanita phalloides*, other amatoxic *Amanita* and amatoxic species in the genera *Lepiota*, *Galerina* and *Conocybe***

Of the toxins in *Amanita phalloides* and *Amanita ocreata*, only the amanitins are effectively toxic in humans. The amanitins—alpha, beta, gamma and epsilon ( $\alpha$ ,  $\beta$ ,  $\gamma$ ,  $\epsilon$ )—are bicyclic octopeptides (8 amino acid chains) with an unusual sulfur bridge and are lethal in the alpha form at about 0.1 mg per kilogram of body weight.<sup>32</sup>  $\epsilon$ -amanitin is present only in minute amounts and does not contribute significantly to the toxicity. Destroying the sulfur bridge renders the compounds inactive. The amount of amatoxin in a single *Amanita phalloides* cap varies considerably, but may be enough to cause death. The amino acid group near the sulfur bridge is tryptophan and this hydroxylated amino acid is the portion of the molecule assayed in thin layer chromatography.<sup>33</sup> The phallotoxins have one less amino acid and these heptapeptides apparently play no role in human poisoning. The cause of the early GI symptoms starting between 8-14 hours is unknown.



		R <sub>1</sub>	R <sub>2</sub>	R <sub>3</sub>	R <sub>4</sub>
α	Amanitin	-OH	-OH	-NH <sub>2</sub>	-OH
β	Amanitin	-OH	-OH	-OH	-OH
γ	Amanitin	-OH	-H	-NH <sub>2</sub>	-OH
δ	Amanitin	-OH	-H	-OH	-OH

**Figure 1. The Amanitins**

At low concentrations, amanitins bind specifically to the enzyme RNA polymerase II (or B) in cell nuclei. With reduced amounts of polymerase II to catalyze the transcription (“reading”) of messenger RNA from DNA, there is less protein synthesis in the body.<sup>34</sup> At very high doses RNA I (or A) and RNA III (or C) are also affected.<sup>35</sup> Phallotoxins & virotoxins are present in *Amanita phalloides*, but do not produce symptoms in humans.<sup>36</sup>

In 1966, Tyler in Washington found that the amount of beta-amanitin was generally higher in our specimens than in those from central Europe where the amounts of α- and β-amanitins are about equal.<sup>37</sup> This geographical difference was confirmed in 1974 by Dr. Udo Boerner studying over 100 specimens of *Amanita phalloides* collected by the MSSF.<sup>38</sup> Similar chemical differences have been noted for many geographical “strains” of fungi and it is not clear how much is directly environmental and how much is a genetic adaptation to different conditions including available mycorrhizal hosts. Some fruiting bodies, even from what is presumably the same mycelial mass, contain no or little toxin.

The amount of amanitins in western species averaged 2.3 mg/gram dry weight for *Amanita phalloides*. *Amanita ocreata* is thought to contain about the same

amount of amatoxins as eastern amatoxic species, such as *Amanita verna* containing 1.5 mg/gram or less.<sup>32,37,39</sup> Amatoxin amounts for *Galerina autumnalis* averaged 1.5 mg/gram and *Galerina marginata* 0.45 mg/gram.<sup>32,40</sup> These two species are now considered conspecific and the correct name is *Galerina marginata*. Since the toxin was given per dry weight, it is surprising that there was a difference in amatoxin amount for the two presumed species. Possibilities include chemical races and age differences between the specimens.

## The Wieland-Meixner qualitative test for amatoxins

This is a simple and fairly accurate screening test for the presence of amatoxins *if done correctly*. Wieland-Meixner testing should never outweigh the clinical findings or another identification of an amatoxic species. The Wieland-Meixner test is often done incorrectly. ***Hospital laboratories notoriously obtain false negatives by using dilute hydrochloric acid or by performing the test on low lignin paper such as filter paper.*** The reaction is an acid-catalyzed one (using concentrated HCl acid) with the lignin present in low-grade paper, such as newspaper. A grayed greenish blue color appears with dried amatoxin. Dr. Theodor Wieland first studied the test on high-lignin paper, but Dr. Axel Meixner's article of 1979<sup>41</sup> specifically noted that it would work on ordinary newspaper and the test is usually referred to simply as the "Meixner test". The color change was actually noted first on wood chips by W. Dilger, while working on a thesis in Mainz, Germany.<sup>42</sup>

The test should be done on a surface **away from high heat or sunlight**. A control spot of concentrated HCl turns bluish when exposed to direct sunlight or to a temperature range of 85 to 95°F over a 20-30 minute period. Left in the sun longer, these bluish control spots slowly turn light pink. The Meixner test can be done with a hair drier held at a distance, but overheating is difficult to avoid.

Avoiding sunlight or high heat, a small circle is made on newspaper (or other high-lignin paper) with a pencil. A small amount of mushroom juice is then squeezed on to this and left to dry at room temperature. When dry, a single tiny drop of 8-16 concentrated hydrochloric acid is placed in the center of the circle. The grayed bluish-green that is typical of amatoxins slowly appears over 1-3 minutes, but the test sheet should be observed up to 20 minutes. The color on high lignin paper is usually 25B3-5 in the *Methuen Handbook of Colour*,<sup>43</sup> but may vary from 25B3-5 to 25B5-26B4. There has been considerable concern about false positives.<sup>44</sup> However, if done with reasonable knowledge of colors seen with various genera, falsely positive tests are not of great concern.

The test results from California were combined with taxa presumably identical to those in North America, but collected in Germany and reported by Ruth Seeger.<sup>45,46</sup> The group total was 526 tests, but there were 56 species in common so that the total number of different species tested was 470. Many mushrooms gave an array of colors from yellow, orange and red to green, blue and even violet. Nine species of *Agaricus* gave a color change from yellowish to reddish, but only twenty nine species of this common genus were tested. There was a 15% false positive result for the total species tested, but this included some easily distinguished genera such as *Russula*, *Lactarius* and *Coprinus*. Excluding these and very bright blue or green colors, the number of false positives drops to 6.8%.

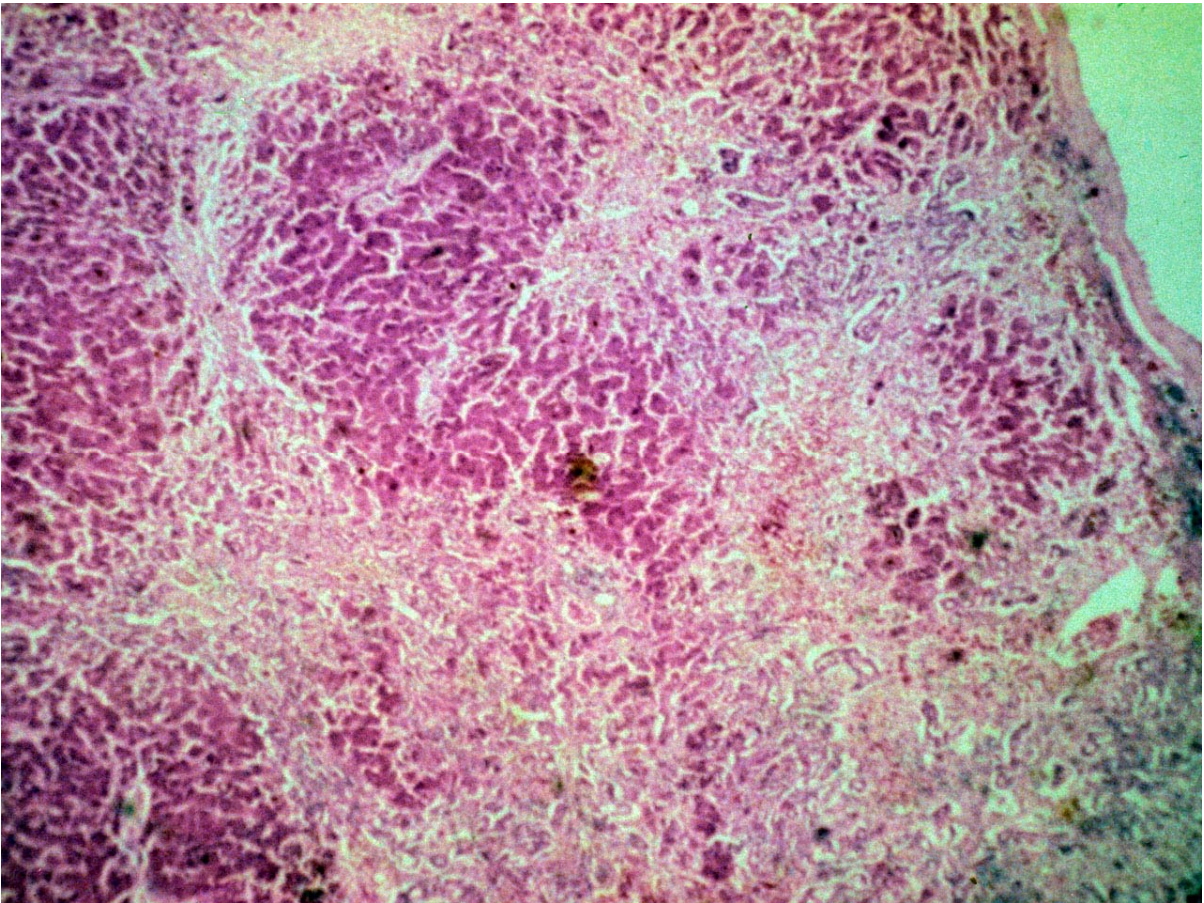
There were a few anomalous single Meixner tests. *Galerina marginata* was positive for *amatoxins* in two of the three specimens tested. One specimen of presumed *Conocybe filaris* was negative, although two other specimens gave a positive test. *Lepiota helveola* Bres. and *Lepiota josserandii* Bon & Boiffard were positive as expected.

## The pathologic picture of amanitins

The pathologic picture reflects the attack of *amatoxins* on numerous tissues, principally by inhibiting RNA polymerase II in cell nuclei. In addition to the liver, pathologic findings may also include small hemorrhages in the tissues and cloudy swelling in the cells of the pancreas, adrenal glands, kidneys, heart, lungs, muscles, intestines and brain. Cardiac muscle occasionally shows fatty degeneration in addition to cloudy swelling. Swollen lymph nodes may occur. The central cells of the liver lobules regularly show fatty degeneration and destruction. Microscopy of a 1970 California fatality is included in the photo section at the end of this paper.

Changes in the kidney glomeruli (the primary blood filter) may show mild enlargement. The kidneys often display cloudy swelling and destruction in the convoluted tubules near each glomerulus.<sup>57</sup>





**Figure 2. Liver cell necrosis in an amatoxin death**

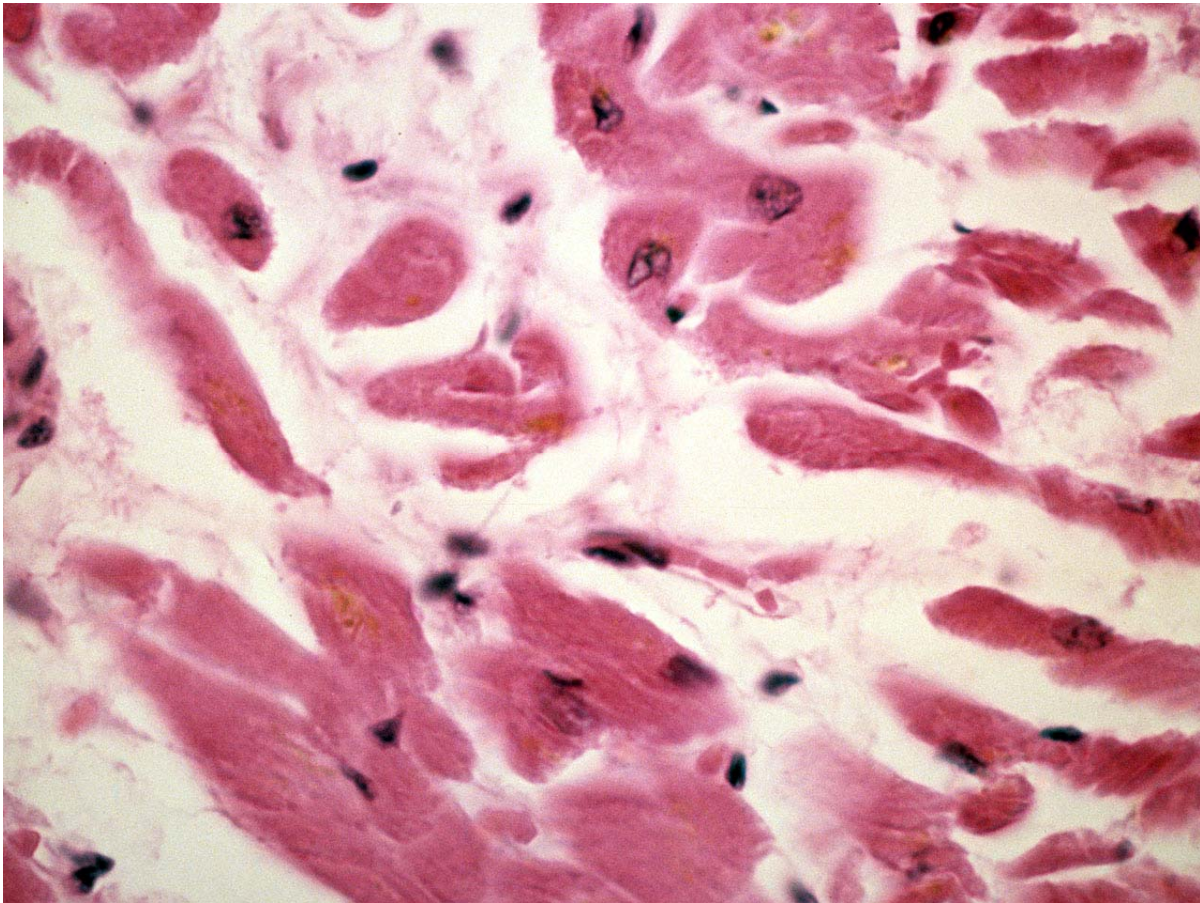
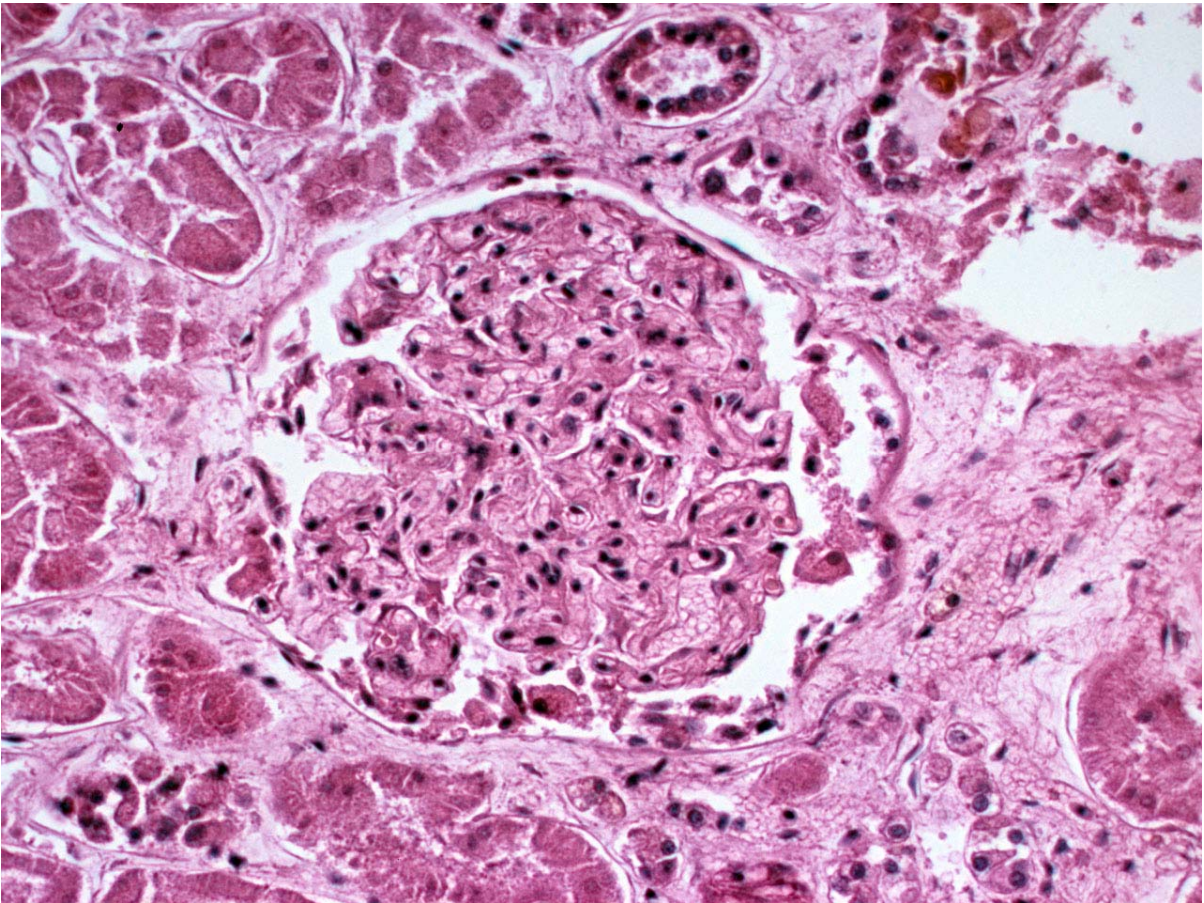


Figure 3. Cardiac muscle with cloudy swelling in same victim





**Figure 4. Kidney tubules & glomeruli in same victim as Figures 2,3**

## Pregnancy and amanitin poisoning

There is a large report on twenty two surviving women from Budapest, Hungary, who had been poisoned by *Amanita phalloides* and related species. The only abnormality noted was a slightly low birth weight. Five of these women were poisoned in the first trimester of pregnancy, which is the most likely to produce fetal abnormalities.<sup>64</sup> There are also smaller reports of one or two survivals in the second trimester, where toxins generally have less effect on the fetus.<sup>65</sup> There has been one US abortion performed in the first 3 months because of amatoxin poisoning.<sup>66</sup> There is one case of mild toxicity in the nursing child of a mother with amanitin poisoning.<sup>67</sup>

## Fatality rates from selected studies published since 1970

Dr. George Floersheim and his associates at Basel, Switzerland in 1982 studied 205 European patients poisoned by *Amanita phalloides*<sup>68</sup> using the statistical method of multiple regression. In this mathematical model, a selected individual treatment and its patient outcome is compared to the average outcome for all the other treatments given. His group also made adjustments for age and for severity (the latter as judged by delay of symptom onset). The death rate was 16.5% for adults and 51.3% for children. Other than charcoal, standard ER and intensive care unit (ICU) management, the only treatment that reduced mortality was massive doses of penicillin, although silibinin compounds and hyperbaric oxygen showed borderline effectiveness.<sup>68</sup> Floersheim tried Penicillin-G, because it reduced the entry of amanitins into the cells of experimental animals. The study protocol used Penicillin-G sodium rather than the potassium salt, to avoid kidney toxicity. No subsequent human study has had the numbers of subjects needed to confirm or refute the findings.

Barrter *et al.* reported on severe US poisoning cases from 1974-1978. Sixty seven of the seventy five patients were thought to be amatoxin cases. All had good criteria for amanitin poisoning, but not all specimens were available or identified by mycologists. The mortality rate was 10.4%.<sup>48d</sup> With education and migrant assimilation, the north American rate should approximate the 5.5-9% mortality found in the developed nations of Europe.

The current mortality rate in California appears to be about 8%, an estimate based on the reported rates over the past 25 years and the gradual improvement in these rates. Reports from the San Francisco and Los Angeles Poison Control Centers, San Francisco General Hospital, Los Angeles County Hospital, UCSF Pharmacology Department and consultations with local mycological societies



provide the core of this estimate.<sup>47,60,61</sup> Some of the California amanitin poisonings were not reported to the North America Mycological Association, although they were reported to the appropriate Poison Control Centers.

There are inadequate data on children under the age of 10. Their death rates are probably between 15 and 22% extrapolating from Floersheim's European study.<sup>68</sup>

Fatality rates from Mexico show that untreated poisonings have roughly 50% mortality rates. Those patients that are treated in hospital have a prognosis close to that in the United States.

## Evaluation of amanitin treatment

Except for Florsheim's study, current therapy is largely based on animal experiments and the current treatments for liver and kidney malfunction. Most studies of survival are simply too small to be of statistical value. The problem of gathering enough patients to study the efficacy of new therapies occurs because of the few deaths that now occur. Researchers need to study additional end points, such as days of hospitalization, return to normal activity or avoidance of dialysis. As the death rate becomes smaller and smaller, it becomes more and more difficult to obtain the required numbers of poisoning cases to show likely efficacy. With a current mortality about 8%, even a 50% reduction to 4% is hard to demonstrate statistically.

High dose intravenous Penicillin G was efficacious in European studies, but this treatment has safety problems. It has to be given as the sodium salt. At 40 million units/day (or for children, 1 million units per kilogram of body weight), the dosage is close to the seizure threshold in children and makes sodium restriction difficult to achieve in adults. In the United States, this treatment has fallen out of favor and there is little likelihood of reaching the patient numbers needed for a North American study.

In addition to activated charcoal, European treatment also includes silibinin intravenously in a dose of 20-30 mg per Kg of body weight per day given in 4 equal doses over a period of 2 hours. Vogel did the original work showing that extracts of the European milk thistle antagonized phalloiden. The extracts also seemed to protect experimental animals from amanitin. A later study of his showed purified silibinin (the most potent compound from the milk thistle) to protect poisoned beagles.<sup>69</sup> The German drug company A.G. Mahaus was unable to get even an "orphan" drug approval from the FDA in this country.<sup>70</sup> The

mechanism of silibinin protection appears to be interference with uptake by liver cells, thus interrupting the enterohepatic recirculation.

Glowing reports with silibinin claiming near-perfect survival rates use small numbers of patients. There are statistical methods (such as chi square) designed for small research numbers, but these methods are easily abused. The patients in question must be very homogenous with significant characteristics similar to that of a control or historical group of cases. Such characteristics include the variables of age and severity of poisoning (judged by the latency period before symptom onset and liver/kidney/coagulation tests). However, reports usually show marked improvement in liver function following the intravenous administration of silibinin.<sup>87b</sup>

A large co-operative study using multiple European, Asian and North American medical centers with a good clear protocol should be done on silibinin. Its use in Europe is now standard, since the medication is readily available there and has no apparent side-effects. It is not generally available here, although it has been flown in for the treatment of at least one patient.

Hyperbaric oxygen in Floersheim's large study (3 atmospheres of pressure in the oxygen chambers) seemed possibly effective. Its use, however, was restricted to high-tech centers such as the one in Nancy. Few such centers exist and the care there would be presumed to be of the best available.

## **Prognostic indicators for likely fatality**

A simple classification for cases of amatoxin poisoning can be formed from the latency period and the prothrombin time. Most commonly that measurement of clotting time becomes prolonged in amanitin poisoning when the damaged liver produces less prothrombin. One can readily classify mild poisonings as those that have a latency period over 18 hours and a normal prothrombin time; moderately severe cases, as those with a latency period from 13-18 hours and an abnormally long prothrombin time (but less than 55 seconds) and severe cases as those having a latency of 12 hours or less and a prothrombin time of 55 seconds or more. As expected with biologic phenomena, the three criteria do not always correlate. The prothrombin time probably is the most closely correlated with death.

A 1991 review of 140 cases where some of a severely poisoned group died without recourse to transplantation showed that the fatality cases in these patients were best separated out on the basis of thromboplastin time (liver) and serum

creatinine (kidney). In this study, the two best indicators for likely fatality were a thromboplastin time (35% of normal or less) *despite moderate replacement of these factors* and a creatinine of 1.2 or more for at least 2 days. The authors concluded that patients whose thromboplastin time and creatinine were in the possibly fatal range should be transferred to liver transplant centers for further evaluation and possible surgery.<sup>87a</sup>

A few patients with these criteria have been shown in other reviews to go on to semi-coma and even to coma and still survive. Bartels and Seeger, as early as 1977, presented 13 cases treated with charcoal hemoperfusion in the days prior to liver transplantation, who had encephalopathy grades II-IV.<sup>56a</sup> Ten of the 13 survived, but more importantly 3 of 5 patients with grade IV encephalopathy lived. Similar cases have been sporadically reported.<sup>88,89</sup>

A German group found that a quick thromboplastin time and a coagulation factor V under 10% of normal on admission were the best prognostic tests. Encephalopathy and renal failure were additional indicators, but not as good as the coagulation factors. The presence of either one was a very bad omen.

## The clinical course of amanitin poisoning

The clinical picture of amanitin poisoning is characterized by delayed onset of gastrointestinal symptoms, often a transient remission of these symptoms, during which time the liver and kidney damage increases, and finally either spontaneous recovery or death. The course is best described by the latency period: the number of hours or days since ingestion, rather than the onset of symptoms. Thus day 2 would start 24 hours from ingestion. Between days 2 and 4, there are frequently some hours during which most or all symptoms subside. This “honeymoon” period (rarely more than 24 hours) may not occur at all.<sup>47,48e,49a,50,51,52,53</sup> Experience with numerous cases in the San Francisco area from 1970 on would not support statements in the literature that the remission period frequently lasts longer than 36-48 hours. Statements to the contrary are baffling; some reports may include the latency period.

One report notes occasional absence of abdominal pain.<sup>54</sup> Diarrhea is often continuous in severe poisonings for 3-6 days, after which jaundice and progressive liver failure usually dominate the clinical picture. Blood liver enzymes such as ALT and AST (alanine transaminase and aspartate aminotransferase) are with rare exception elevated by the time of admission to the emergency room. Levels may go up into thousands of units. Prothrombin, a protein manufactured by the liver and essential to blood clotting, may be low.

Vomiting and diarrhea usually begin between 10 and 16 hours, but may be present as early as 6 hours in severe poisoning and as late as 36 hours. The diarrhea is usually profuse and may be bloody and painful. Occasionally diarrhea is mild or the clinical picture may present as severe ileus, a condition in which normal propulsive bowel waves are markedly reduced. This unusual complication is accompanied by bloating, reduced bowel sounds and decreased or absent fecal output. Mild ileus is fairly common and presents only with slightly decreased, but high-pitched, bowel sounds and minor bloating. Earlier speculations that phalloidin produced the GI symptoms have been proven incorrect.<sup>55</sup>

The liver is often tender on admission or becomes so in the first 24-48 hours. The liver may enlarge in the first few days to as much as 5-15 cm below the right rib margin.<sup>56c</sup> The spleen also sometimes enlarges during the course of acute poisoning.<sup>47</sup> Such enlargement presumably occurs only during the acute phase of the illness.

Kidney failure is almost never an early finding and most often occurs, if at all, along with severe acidosis and liver failure as a late event. When renal failure does occur early, a study of 106 patients has shown oliguria (decreased urine output).<sup>57</sup> Renal failure most commonly occurs between the fifth and tenth days, preceded by increasing protein, protein casts and blood cells in the urine. Such kidney failure may occur in mild to moderate poisoning, most commonly when dehydration goes untreated 36 hours or more.

Liver failure manifests itself by jaundice, fluctuations of blood sugar, sometimes bleeding and changes in mentation (encephalopathy). The stages of encephalopathy are grade I (confusion), II (somnolence), III (coma, but responses to painful stimuli present) and IV (coma with no response to painful stimuli). Abnormal cardiac rhythms, shock and lactic acidosis may appear. Systemic acidosis is a regular feature of severe poisonings. However, lactic acidosis caused by poorly oxygenated cells is usually a fatal event.

Both high and low blood sugar levels have been reported in the course of poisoning. The liver stores glucose as a more complex carbohydrate called glycogen and a sudden toxic insult may release sugar causing a high blood sugar. As it turns out, either hyperglycemia (high glucose) or hypoglycemia (low glucose) may be present early. When hypoglycemia is present, insulin levels have been elevated, providing at least one mechanism for an early low blood glucose.<sup>58</sup> Late hypoglycemia also occurs and presumably reflects depletion of glycogen stores in a badly damaged liver.



Both low calcium and low phosphate levels have been noted along with varying levels of the two hormones—parathyroid hormone and calcitonin—that control calcium and phosphate metabolism. A Polish study of 21 poisoned children found low phosphate levels predominant in cases of moderate severity. Low calcium levels were more commonly seen in severe poisonings.<sup>59</sup>

Intestinal perforation, mesenteric venous thrombosis (clotting in the bowel veins), rhabdomyolysis (death of muscle tissue), septicemia (toxic blood infection), seizures due to cerebral edema and respiratory distress syndrome are rare complications.<sup>60,61</sup> Occasional patients require a transvenous pacemaker for temporary heart block presenting as marked slowing of the heart rate. A number of fatal cases have had septicemia, an event due to the patient's poor resistance and/or the venous/arterial lines inserted.

Disseminated intravascular coagulopathy (DIC) is a rare complication of amatoxin poisoning. This is a dreaded catastrophe in which systemic clotting is inappropriately set off with small clots forming in tiny vessels, followed by depletion of clotting agents with often fatal bleeding. Although reported in amanitin poisoning, the incidence is probably very small.<sup>62</sup> Most of the severe cases of coagulation factor depletion can be explained not by DIC, but by liver failure with decreased liver production of these coagulating proteins.<sup>48c,63</sup>

Other than death, outcomes in severe poisonings include possible brain damage and chronic active hepatitis, the latter up to 20% of survivors in one series.<sup>56a</sup> Most reports show a lower incidence. Persistent poor kidney function is extremely rare.<sup>57</sup>

## Detailed treatment of amanitin poisoning

Currently penicillin G sodium treatment has fallen out of favor in the United States. No further studies with adequate statistical power have been performed and with the dramatically fallen death rates, sufficiently large studies now seem unlikely. The Penicillin G sodium doses used have to be given intravenously as the sodium salt rather than the potassium preparation or potassium toxicity occurs. Furthermore, the amounts of sodium salt required for 40 million units per day (or 1 million units per kg of body weight in children) are almost equal to the daily need for sodium and are sufficiently high that seizure precautions should be considered in children. Cephalosporins (another antibiotic class) will also bind amatoxins and should be much safer, but their efficacy remains unproven with only a few patients so treated in Europe.<sup>71</sup>

American conservatism leads to marked differences between most European and American treatment programs. A typical European protocol is that of Centre-Anti-Poisons CHU of Grenoble.<sup>72</sup> This group advises that at least one tube be advanced just within the duodenum by visual control. Their next step is chemotherapy with IV penicillin G and silibinin.

Some European treatment sheets show as many as 20 chemical interventions in the first 36 hours. European treatment also often includes hemoperfusion. In this procedure, arterial blood is sent through an activated charcoal or resin cartridge to bind amanitin, although viable pig and human liver have been used on occasion. Pig liver was used in two San Francisco cases as a last resort, but both patients died after severe acidosis could not be reversed.<sup>60</sup>

American treatment varies somewhat. Double lumen tubes with gastric and duodenal openings have been used. The gastric lumen is used to cleanse the stomach and to suck out gastric fluid. The duodenal lumen is used to give activated charcoal to absorb the amatoxins. Presently the use of wide bore gastric tubes is the most common. The first step is washing out the stomach with saline to remove all food and then pushing in a slurry of activated charcoal in 6-8 ounces of fluid. Another option is the use of the more highly absorbing resins such as Amberlite XAD-2, but this remains experimental. The initial dose of activated charcoal is usually 50-100 grams or 1 mg per kg of body weight.

Despite the diarrhea, there is often a mild ileus with slight abdominal bloating and reduction of normal bowel sounds. Moderate ileus is an indication for low dose charcoal. Very severe ileus is extremely rare, but other methods of amatoxin removal such as biliary cannulation, extremely vigorous diuresis (preferably with right atrial intracardiac pressure measurements) and hemoperfusion should be considered within the first 36 to 48 hours of ingestion.

Amanitins are absorbed from the duodenum, carried to the liver by portal veins and a portion is then re-secreted into the systemic circulation by the hepatic veins. However, a goodly amount is excreted in the bile and goes back by the common bile duct to the duodenum to be absorbed again.<sup>73</sup> This vicious cycle of re-absorption and excretion leads to repeated bombardment of the liver by amatoxins.

After the initial dose of charcoal, 10-30 grams of activated charcoal is given every 2-4 hours into the stomach or duodenum. If the double lumen tube is used or a duodenal tube only, then 2-4 hours of charcoal placement into the duodenum is followed by at least 1 hour of duodenal suction. A possible hazard, if a long

duodenal tube with a weight is placed into the duodenum using only x-ray confirmed placement, is the possibility of passing the tube into an area, which is not near or even past the actual entry of the bile duct. There is considerable variation of bile duct anatomy. A long duodenal tube that is not anchored firmly may also drift after the abdominal x-ray is taken.

Another option is passing a thin plastic catheter directly into the bile duct and removing amatoxin by very low pressure suction. After discussion with the author in 1982, a Santa Cruz (California) gastroenterologist, Dr. Larrimore Cummins, performed the first (and only) biliary cannulation on an *Amanita phalloides* victim. The patient did extremely well, but the biliary drainage and serum samples for amatoxin analysis were lost.<sup>74</sup> Since that time, the concerns noted above (and the obscure journal in which it was published) have been followed by no further trials. A simpler treatment has been used in Europe: aspiration of bile from the duodenum without using any charcoal.<sup>75</sup>

Various methods of toxin removal directly from the systemic blood circulation have been explored over the past 25 years and the best of these appears to be hemoperfusion, if done within the initial 36 hours.<sup>48f</sup> This procedure in which the patient's blood is directly passed through a resin cylinder has been uncommonly done. Most of these procedures have been done on very ill patients. These fatality rates, however, are about the same as those of patients treated in the standard way. A paper from the Slovak Republic found that the resin, Amberlite XAD-2, was experimentally more effective in removing amatoxins than Amberlite XAD-4 and that activated charcoal hemoperfusion was less effective than either resin.<sup>76</sup>

With just adequate fluids alone, about 85% of amatoxins are excreted by the kidneys within six hours of poisoning. It is not known how low the levels of amanitins in blood have to be in order to cease damaging organs. Sensitive assays show low blood toxin levels after 36 hours. However, amatoxins have been found in urine as late as 96 hours. The authors of that study suggest forcing urine output to 100-200 ml each hour with isotonic fluid during the initial 48 hours after amanitin exposure.<sup>77</sup> The amount of amatoxin present in the urine, however, correlates poorly with the severity of poisoning.<sup>49b</sup> Amatoxins in blood and bile are usually assayed by a very sensitive radioimmunoassay or with high-pressure liquid chromatography.

Piqueras and his group in Barcelona have shown that forced diuresis (a drug and water induced increase in urine flow) is effective. A urine output of 150-200 mL/h is able to remove 20,000-350,000 ng of amatoxin.<sup>78</sup> A blood purification method called plasmaphoresis removed no more than 10,000 ng.<sup>78,79</sup> The Barcelona group

using forced diuresis and plasmaphoresis in select patients has had the best mortality rate of any good sized study to date: a 5.5% death rate of their 91 patients reported in 1989.<sup>51</sup> Another group showed that with rapid diuresis there was no tubular re-absorption of toxin and little, if any, damage to the kidney tubules (although this has been a major concern of another expert, Dr. Faulstich in Germany).<sup>80,81</sup>

ICU orders for severe poisonings should include hematology, electrolytes, calcium and phosphate levels, liver and kidney function panels, amylase, random blood glucose levels and acid/base studies. Standard cardiac and pulmonary monitoring are usually adequate. Patients in shock require more extensive evaluation including right atrial monitoring. Any changes in blood clotting or prothrombin time indicate a need for more extensive work-up including thromboplastin time, fibrinogen levels and measurements for split fibrin products. What tests are necessary rests on clinical judgment.

Renal function requires more than one gauge. Blood urea levels may be up because of dehydration, but present no problem if vascular status and fluid input/output are carefully followed. Creatinine levels will be elevated if there are severe muscle cramps, but then the muscle enzyme CPK would also be elevated. Sophisticated renal failure indices, such as the fractional excretion of filtered sodium, are not needed. The best test may be the ratio of urine osmolality to plasma osmolality. This ratio is  $\leq 1.3$  if the plasma volume is low (causing too little blood flow to the kidneys) and  $\geq 1.2$  in acute renal failure where the kidney itself is damaged.

Should muscle cramps occur, magnesium as well as calcium and phosphate levels should be checked. For grade IV and probably also for rapidly advancing grade III encephalopathy, intracranial pressure monitoring is now a standard procedure in most liver transplant centers.

There should be a near-by dialysis unit in case of kidney failure. Peritoneal dialysis is an alternative in patients with unstable blood pressures, but this procedure is contraindicated by previous abdominal surgery or infection. Liver transplantation is available only at select medical centers.

There may be a need for fresh frozen plasma or specific coagulation factor replacement. Vitamin K has little effect on the prothrombin time.<sup>82</sup> Since disseminated intravascular clotting (DIC) or liver depletion of clotting factors may occur, a hematology consultation is advisable if the prothrombin time is 20% less than normal, blood platelet count is diminished or unusual bleeding occurs.



Fibrin split products from dissolving blood clots are found in DIC. Depletion of coagulation factors by a damaged liver is the usual cause of unexpected bleeding. Although DIC appears to be uncommon, the early stage of this disorder involves clotting and there is one report of mesenteric thrombosis.<sup>83</sup>

Most cardiac arrhythmias are supraventricular and not serious. There have been cases requiring a temporary transvenous pacemaker for low-grade heart block with a very slow cardiac rate. The treatment of shock is no different than in other disorders. Chest x-rays may reveal pneumonia or pulmonary bleeding.

The standard treatment of liver failure includes maintenance of normal or high normal glucose levels and avoidance of protein loads, which are poorly handled by a failing liver. Toxic nitrogenous substances are formed from protein fragments, then released into the blood and may cause brain damage. Broad-spectrum antibiotics have been used to reduce nitrogenous toxins from intestinal bacterial activity and to reduce the incidence of septicemia. The latter is a frequent and very dangerous occurrence in patients with multiple catheters and intravenous lines. When antibiotics are used to reduce nitrogenous products from intestinal bacterial activity, monitoring of antibiotic blood levels is usually necessary. Abnormally high antibiotic levels may damage organs and, in particular, kidney function and their ability to clear not only the antibiotics, but also the toxins.

Aminoglycoside antibiotics as well as atropine, barbiturates, phenothiazines, contrast media used in some x-rays and other potential liver toxins should be avoided if possible. Pain may require cautious doses of meperidine HCl (Demerol®), morphine or hydromorphone HCl (Dilaudid®).

In acute liver failure, cerebral edema (brain swelling) may occur. Cerebral edema causes a quick rise in intracranial pressure, since the skull's rigidity allows the brain little room for expansion. Using a burr-hole, intracranial monitoring is safest when the monitor is placed over the dura (outer membrane around the brain).<sup>85</sup> Treatments for increased intracranial pressure include intravenous mannitol to move fluid from the brain to the blood stream.

A bioartificial liver may be helpful at tiding the patient over, but the technology is still evolving.<sup>86</sup> At present liver transplantation is the only solution for severely deteriorating liver function. Criteria for transplantation have usually been the same as those used for acute hepatitis with hepatic failure.<sup>87</sup> The best option is usually partial liver transplantation leaving the patient's left liver lobe. This procedure allows that portion of the patients' own liver a chance to regenerate.

Liver regeneration therapies, which may become important in the near future, include insulin, growth hormone and liver growth factor. If blood glucose levels are normal, intravenous administration of 1 unit of insulin per hour has been suggested in severe poisoning.<sup>75</sup> Pure growth hormone has now been synthesized using recombinant DNA grown in cultures of the bacterium *Escherichia coli*. Growth hormone's primary use is in short stature due to growth hormone deficiency, but it has been used in some cases of non-specific short stature and in some burn patients as an anabolic agent. Since growth hormone, insulin and liver growth factor reduce liver necrosis in experimental animals, cautious trials would seem reasonable in amatoxin poisoning. The recommended dose of growth hormone is 40 units per hour or the equivalent of the active portion of growth hormone. Antibodies active against amanitins have been produced in the laboratory,<sup>90</sup> but there has been little clinical progress in the past 10 years.

## Outline and summary of treatment for poisoning by amatoxins

1. Identify the mushroom. Do a Meixner test if possible, but proceed with treatment on the basis of the patient's history. **Do not delay for examination of the mushroom or a Meixner test. More than one mushroom species may have been ingested.**
2. Intravenous line with a goal of 150-200 ml/hr output of urine.
3. Wash out the stomach if within 6 hours of food and proceed with one of the methods to capture the re-circulation of amatoxin in the bile (gastric tube, gastroduodenal tube or duodenal tube for delivery of activated charcoal 50-100 grams (1 mg/kg body weight approximately) at once and then 10-30 grams every 2-4 hours—see above for details if there is evidence of ileus or it is elected to place a tube within the duodenum).
4. Alternate considerations are early hemoperfusion with resin cartridges in severe ileus, direct cannulation of the common bile duct under endoscopic control or simple suction of fluid from the duodenum near the opening of the common bile duct (usually the second portion of the duodenum).
5. ICU monitoring of blood volume, input and output as well as cardiac, renal and hepatic function, blood cultures, electrolytes, blood glucose, coagulation studies (including thromboplastin time and fibrin split products) and other lab studies as noted above.
6. Hematology consultation in the event of coagulation problems.
7. Availability of renal dialysis and transfer to a liver transplant center if indicated.
8. Standard treatment of hepatic failure, awareness of cerebral edema and seizures.

9. Consideration of massive Penicillin G early for heavily poisoned patients, if the cardiovascular status is stable and the potential problems with this treatment are recognized.
10. Trial of intravenous silibinin (Legalon®) while monitoring changes in liver function tests.
11. Consideration of treatment with liver growth factors for severely ill patients. Use of these growth factors is experimental, but probably safer than Penicillin G, which has been widely used in Europe.

## Group II toxins: Ibotenic Acid/Muscimol

### ***Isoxazole poisoning from *Amanita muscaria* & *Amanita pantherina* (pantherine syndrome)***

Two *Amanita* species (*Amanita muscaria* (L.:Fr.) Pers. and *Amanita pantherina* (DC.:Fr.) Krombh.) are responsible for a quite different type of poisoning caused by the toxins ibotenic acid and muscimol. Other toxins appear to be present as well. Such additional toxins may explain, for example, why vomiting is more likely with *Amanita muscaria* as compared to *Amanita pantherina* (the most toxic over-all). Both of these amanitas have several variants.<sup>91</sup>

*Amanita muscaria* is present in large quantities along the coasts of Northern California through British Columbia, most commonly in the variety *muscaria* and in the subspecies *flavivolvata*. The former is not uncommon in California, but gives way to the latter in the PNW and in Colorado.





*Amanita muscaria* photo © Michael Wood

*Amanita muscaria* var. *muscaria* Pers. has a red cap whose surface has white warts (easily removed), white gills, a white  $\pm$  persistent ring and a white stipe. The volva consists of 2-3 (4)  $\pm$  complete rings just above the swollen base of the stipe. This mushroom is widely distributed in Western North America, including Mexico. The red cap fades to reddish orange in sunlight. The more detailed description below for subspecies *flavivolvata* holds in general for this and the other varieties of *Amanita muscaria*, except for the coloration and some variation in size.

*Amanita muscaria* subsp. *flavivolvata* Singer has removable creamy to yellow warts on a deep golden tan, reddish orange to red cap,  $\pm$  viscid, smooth, 5-17 (39) cm across. Bright red caps in full sun quickly fade to a more orange color. The cap begins as a convex to almost hemispheric structure and becomes plane to

depressed in age, sometimes humping slightly in the center. The margin has short striations in maturity. The flesh is white with yellow to reddish tissue just below the cuticle. The white to pale cream gills are attached then free, broad, crowded with numerous shorter gills. The edges of the gills are smooth to rough. The stipe is white to cream, tan when handled or bruised, 6-16 (20) cm long and 1-3 (4) cm wide at the top, enlarging to a ± bulbous base. The surface is smooth above the ring and fibrillose to scaly below. The ample ring is persistent, membranous, white with a ± yellow edge and often a yellowish tint to the undersurface of the ring. The volva is poorly formed and presents more as a series of fragmented concentric rings. Found from California originally; this variety is uncommon there, but common in the PNW and in the Rocky Mountains. Its range also extends from New Mexico and Arizona down into Mexico's Sierra Madre Orientale.

*Amanita muscaria* var. *guessowii* Veselý [= *Amanita muscaria* var. *formosa* sensu. auct. amer.] has a yellow-orange, yellow or yellow-tan cap when young. It is found in the PNW, California and Mexico, but is uncommon. In California, what is probably this subspecies, is found in the mountains from 3500 feet up to the subalpine zone in the Sierra Nevada and Cascade mountains. There is one low-land collection from Napa county.<sup>93</sup> In the PNW, it is found occasionally in both montane and sea-level areas. In Mexico, the range of this species is restricted to the higher elevations.

Presumably, other varieties of *Amanita muscaria* are toxic and, possibly, also the closely related *Amanita breckonii* Ammirati & Thiers.

*Amanita muscaria* var. *alba* Peck is a white to yellowish-buff form in the PNW. There are 2 California collections of this form from the northern coastal forests of Humboldt and Del Norte counties.<sup>93</sup>

Jenkins noted the presence in Alaska of what he thought was identical to a Scandinavian taxon—*Amanita muscaria* var. *regalis*. However, this mushroom is now considered to be a separate, but closely related species—*Amanita regalis* (Fr.) Michael.<sup>94</sup> Dr. Rodham Tulloss and Dr. Joe Ammirati have also compared this taxon with the Scandinavian one and agree they are not the same.<sup>95</sup> The cap is yellow to golden brown with yellowish or light tan floccules.

*Amanita pantherina* (DC.:Fr.) Krombh. resembles *Amanita muscaria* with its warty cap and ± persistent ring; but the cap color is different, usually a yellow brown to dark brown. The bulbous stipe base also differs in that the volva adheres to it at the base with an abrupt inrolled collar at its apex; sometimes



there are 2-3 volval rings from the universal veil just above the major portion of the volva. *Amanita pantherina* is quite common in California and Oregon, but is found less frequently in southern California and Baja California.



*Amanita pantherina* photo © Michael Wood

There is enough variation in *Amanita pantherina* that it is best to give a standard description. The description chosen is that of the San Francisco Bay area mushroom.

The cap is 4-15 cm broad with ground color dark brown or tan, sometimes buff yellow, although often with a darker apical area. The shape of the cap is convex to subglobose, becoming plano-convex to plane, depressed or umbonate in age. The margin of young caps is incurved to decurved becoming uplifted in senescence and is striate to tuberculate-striate. The striations are 6-10 mm long and less evident in age. The surface is viscid to subviscid, usually with flat to pointed, floccose, loosely attached universal veil remnants, which are whitish to buff, sometimes arranged in concentric rings. The cap is often glabrous in age.

The flesh is white except for a pale yellow next to the cuticle, firm, 3-14 mm thick; odor and taste mild. The gills are bluntly attached, usually adnexed but becoming deeply notched to free. The gills are close to subdistant, white becoming buff; margin fimbriate then smooth. The stipe is 5-15 cm long, 8-20 mm broad at the apex, equal to tapering or clavate, bulbous at base. The stipe surface is moist to subviscid, pruinose to appressed fibrillose and longitudinally striate above the superior skirt-like ring, which collapses in age. The color of the stipe is white to buff, staining buff or brown when bruised. The volva is white, consisting of one to several rows of irregular tissue, which is inrolled at the bulb apex forming a ± distinct collar, occasionally disappearing with age. The identification depends primarily on the distinctive arrangement of the universal veil remnants on the cap along with the inrolled volva. The mushroom is commonly found under Monterey pine in the San Francisco Bay area.

*Amanita pantherina* var. *pantherinoides* (Murrill) Dav.T.Jenkins, having either a free-limbed short portion of the volva or an abrupt collar, occurs at least in Oregon and Washington. This variety is usually brown at the apex, but the rest of the cap is a warm pale tan or honey yellow.

A form of *Amanita pantherina* in which the young buttons are white is found in Colorado, but apparently has not been studied extensively.<sup>91</sup>

Both *Amanita muscaria* and *Amanita pantherina* occasionally have specimens low in ibotenic acid/muscimol.<sup>96</sup> Dr. Joe Ammirati notes that one or another variety of *Amanita pantherina* may be found in every month of the year.

Isoxazole poisonings have been occasionally reported with what were thought to be *Amanitas* intermediate in character between *Amanita gemmata* and *Amanita pantherina*. However, *Amanita gemmata* may be a complex of closely related species, ±yellow or yellow tan, whose volvas are not always the typical membranous sheath. The intermediate forms have also been thought to be “hybrids” in Northern California and in the PNW.<sup>97</sup>

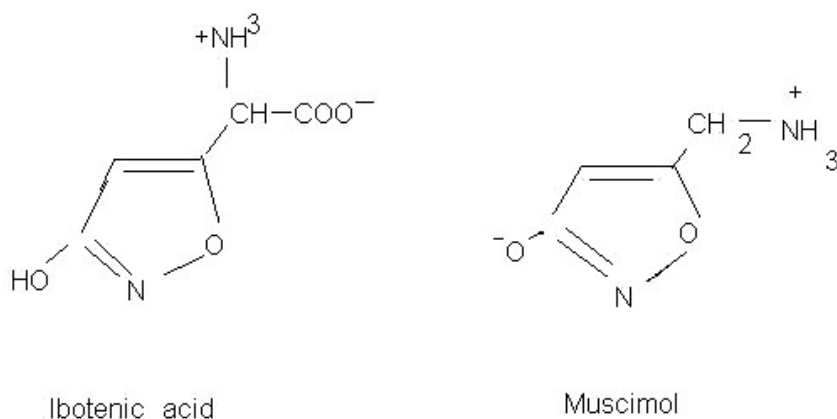
## Toxins of the ibotenic acid/muscimol group (pantherine poisoning)

*Amanita muscaria* derives its name from the ability of its juice to stun and sometimes kill house flies (*Musca domestica*). The fly attractant is 1-3 diolein.<sup>98</sup> and the “flycidal” agents are ibotenic acid and erythro-DL-tricholomic acid.<sup>99,100</sup>



The major toxins present in the mushrooms are ibotenic acid (an acidic amino acid in the form of an isoxazole ring) and muscimol, its decarboxylation product. Unfortunately muscarine was the first toxin to be identified in *Amanita muscaria*, albeit in minute, non-physiologic amounts: about 0.00025% was extracted from this mushroom by Holmstedt and Liljestrand in 1863.<sup>106</sup> It took years to rid American textbooks of the idea that muscarine was the major toxin in *Amanita muscaria*, rather than ibotenic acid and muscimol. The result was treatment with useless and mildly toxic atropine, generally increasing the patients' misery if not worsening the toxic effects. Even if muscarine were the effective toxin in *Amanita muscaria*, it would take over 100 pounds of that amanita to cause major muscarinic symptoms and roughly 250 pounds to cause death. [calculation in Lincoff/Mitchell]

In 1964, Takemoto and colleagues isolated the true toxins. An international agreement in 1967 named these toxins ibotenic acid and muscimol. Muscimol, supposedly the more important toxin, structurally resembles GABA (g-aminobutyric acid), the brain's most important inhibitory neurotransmitter.<sup>49a</sup> Competing with GABA, the toxin attaches itself to GABA receptor sites. There, muscimol may in part stimulate GABA receptors, at least initially, thus first producing an inhibitory effect similar to that of GABA itself with transient drowsiness and sleep. The major effects, however, are a loss of GABA's suppressive effect on neuronal activity and a marked increase in the synthesis of glutamate and other excitatory amino acids. Thus the illusions and other fragmentations of thought suggest an over-all inhibition of GABA by muscimol's competition for its receptor site.



**Figures 5, 6. Ibotenic acid and muscimol**

It has been shown in monkeys that loss of GABA inhibition allows an increase of the constant stream of stray neuronal signals (“cerebral static”). The result is interference with the main path of thought. The suppression of “garbage” signals is a paramount necessity where concentration holds sway: memory, decision-making, calculation and inference. By 2005 GABA treatment was found to markedly help these activities in Alzheimer’s and similar disorders.

Both muscimol and ibotenic acid pass the blood-brain barrier better than GABA or glutamic acid. Within rat brain slices, isoxazole compounds do inhibit GABA uptake.<sup>102</sup> Benjamin notes, however, that ibotenic acid appears to have an excitatory effect closer to glutamic acid.<sup>101</sup> Presumably ibotenic acid binds and also stimulates glutamate receptors. The further clinical course suggests activation of a series of glutamate excitatory effects, which would be severe when accompanied with partial loss of GABA inhibition.

Scott Chilton in 1978 was among the first to propose a possible role for glutamic acid and glutamate in isoxazole poisoning.<sup>50a</sup> Although glutamic acid and glutamate do not readily cross the blood-brain barrier and are quickly destroyed at that interface, ibotenic acid easily crosses and once entering brain cells, is able to stimulate glutamate receptors.

A number of intermediate compounds have been found to be part of an excitatory aspartate/glutamate “hierarchical system”. This system transports unintegrated “information” from areas of the brain stem to the higher centers of consciousness

in the cerebrum. The "glutamate cascade" is now thought so important that it may be a common pathway for many CNS disease states.<sup>103</sup> Presumably, it is muscimol stimulation of the glutamate cascade in these amanitas that produces the illusion of time change, the increased perception of strength, the changes in vision and even seizures.

Muscazone, an ultraviolet radiation product, is found in minute and variable quantities in most European specimens, but is not present in North American specimens.<sup>104</sup> Stizolobic acid, stizolobinic acid and tricholomic acid are also present as derivatives of ibotenic acid.<sup>105</sup> These three amino acids can activate excitatory amino acid receptors, but there is probably not enough of these compounds to have an effect, at least in most *Amanita muscaria* poisonings. The first two—stizolobic acid and stizolobinic acid—are present in *Amanita pantherina* in amounts that might be clinically significant. These three compounds are related to L-DOPA oxidation products, which are known to cause anticholinergic activity.

## Symptoms of ibotenic/muscimol poisoning (isoxazol poisoning)

The initial symptoms of ibotenic acid poisoning are drowsiness, nausea and occasionally vomiting, usually followed an hour later with a group of central nervous system symptoms: confusion, mild euphoria, visual distortions, sensations of floating, subjective changes in bodily strength, distractibility and retrograde amnesia. True visual hallucinations such as brightly lit tunnels and swirling colors occasionally occur.

Waser tested pure muscimol on himself and noted confusion, difficulty finding the proper words, colored visual distortions and finally sleep.<sup>106</sup> Ibotenic acid caused only lassitude, sleep and migraine headache with the classical one-sided visual disturbance. The headache continued in milder form for two weeks. There was no nausea or vomiting, which are not constant features of this poisoning and may be due to individual sensitivity or to an undescribed toxin.

Symptoms in children do not follow the adult course, but are more grimly excitatory in form. In a review of 1 case of *Amanita muscaria* poisoning and 8 cases of *Amanita pantherina* poisoning, all in children from age 11 months up, symptoms began 30-180 minutes after ingestion.<sup>106a</sup> The dominant presenting features were ataxia, obtundation and lethargic to euphoric states. Physicians and nurses commonly observed myoclonic jerking and muscle fasciculation. Typical generalized tonic/clonic seizures were seen in two children and minor

seizure-like activity was noted in two others. Diazepam readily controlled seizures.

Although seizures may occur with either *Amanita pantherina* or *Amanita muscaria* early in the course with adults, these neurologic symptoms are rare after puberty. In three journal reports, normal neurologic reflexes (corneal, abdominal wall and knee) were lost in poisoned adults and abnormal ones appeared (Babinski's, Chvostek's, Trousseau's). Neither ibotenic acid nor muscimol cause GI irritation, the cause of which remains unknown.<sup>107,108,109</sup>

Although children are more susceptible than adults to the more severe neurological effects, severe poisoning in adults may cause muscle fasciculations, brief muscular jerks or generalized seizures. In a report of ten heavily poisoned cases, three patients had seizures witnessed by physicians.<sup>106a</sup> At the second Aspen Mushroom Conference, Dr. Barry Rumack presented one adult case of massive ingestion of *Amanita muscaria* in which a psychotic patient developed status epilepticus (where one seizure blends into another).

Drying and cooking convert most of the ibotenic acid in these amanitas to muscimol. Only blanching and throwing out the cooking water significantly lower the toxicity.

Japanese found that muscimol in very small amounts was twenty times more effective in enhancing food flavors than sodium glutamate, the commonly used salt of glutamic acid.<sup>54,104</sup>

*Amanita muscaria* has had a higher fatality rate than *Amanita pantherina* in Germany.<sup>110</sup> In the United States, more deaths have been reported from *Amanita pantherina* than from *Amanita muscaria*, which almost never has a fatal outcome here.<sup>111</sup> One particularly notorious death from *Amanita muscaria*, that of the diplomat, Italian Count Achilles de Vecchj in Washington, DC. at the turn of the 19th century, proved an early stimulus to American amateur mycology.<sup>111a</sup> The Pacific Northwest has had 2-3 deaths over many years from *Amanita pantherina*. There was also one fatality from *Amanita muscaria* and one from what appeared to be *Amanita gemmata*.<sup>16</sup> A pot-hunter mistook *Amanita muscaria* for the European *Amanita caesaria* and ate roughly two dozen caps! The unique *Amanita gemmata* death occurred in a two-year-old child, who had shared a meal with her parents. She became listless, had a convulsive seizure and died.

*Amanita muscaria* has been eaten without symptoms in small quantities after boiling and throwing out the water. Eaten raw or nearly so, *Amanita muscaria* acts as a “poor man’s hallucinogen” and was very fashionable in the 60’s. However, it is far less potent in this respect than psilocybin mushrooms and much more likely to have unpleasant results. Recreational use of *Amanita pantherina* in California has been less frequent than in the PNW.

"Mushroom the Journal" had an educational story in its summer 1991 issue.<sup>112</sup> A busload of high-school students, coming back from a jazz competition mushroomed in woods near their dinner stop. Thinking to get high from *Amanita muscaria* they picked *Amanita pantherina*. They shortly began to vomit and hallucinate and even a few became semi-comatose. Three boys ended up in an intensive care unit, two others were kept overnight in hospital and six more were treated and released after ipecac and activated charcoal treatment. Oregon State police rushed the samples to Oregon Health & Science University in Portland. The university in turn got Janet Lindgren, chairman of the Toxicology Committee of the Oregon Mycological Society, out of bed at 2 a.m. The symptoms, the time of year and the fragments clearly identified the offending mushrooms as *Amanita pantherina*. The school temporarily suspended those, who poisoned themselves; the others hopefully learned by example.

The 18th century Siberians seem to have used *Amanita muscaria* to increase their feeling of strength before battle. Dr. Angus McDonald did a 1978 study in California with human volunteers using placebo capsules of whole wheat toast and capsules with dried powdered *Amanita muscaria* using specimens provided by the MSSF.<sup>113</sup> Among the symptoms was nausea, increased salivation, time changes and improved color vision. There were also auditory, visual and bodily distortions including a sensation of increased strength. Motor coordination (standing on one foot, putting nuts on bolts) was decreased, but dart throwing was not. If any conclusion can be drawn about Siberian berserkers, they probably slept before battle, wobbled around, but threw lances just as well.

The six human volunteers and McDonald himself had enough in the way of unpleasant symptoms to outweigh any pleasant feelings. Only two wanted to repeat the experiment at 1/3 of the dose used in the study.

## **Treatment of ibotenic acid/muscimole poisoning**

Treatment is symptomatic and requires close monitoring of symptoms, fluid and electrolyte balance and, in severe cases, seizure precautions. Extremely rare reports of kidney or liver changes suggest poisoning from mixed collections.



Thorazine-like compounds should be avoided, since they may decrease the seizure threshold. Medications for seizures include diphenylhydantoin, gabapentin and sodium valproate. Diazepam and barbiturates are faster acting anticonvulsants intravenously, but have caused paralysis and abnormal EEG changes in laboratory animals previously given isoxazoles.<sup>114</sup> Metoclopramide is a safe anti-emetic. It is probably best to manage any "highs" without any medication if possible. Many of these medications are GABA enhancers and might conceivably worsen any "lows" occurring after the excitatory effects.<sup>115</sup>

## Group III toxins: *Amanita smithiana*

### ***Amanita smithiana* poisoning & unclassified renal/hepatic toxicity**

*Amanita smithiana* Bas, which is found frequently in coastal forests of Northern California and the PNW contains toxins quite different from those present in other *Amanita* species. The appearance of *Amanita smithiana* is somewhat variable and is similar to some other white species of *Amanita*. *Amanita smithiana* is usually seen in the fall, but it also occurs in the spring. The base of the stipe, even without much of a “root” or “bulb” is unevenly swollen much like the root of a horse-radish.<sup>116</sup>



*Amanita smithiana* photo © Darwin DeShazer

*Amanita smithiana* is white with a convex/expanded or centrally depressed, subviscid when moist, 6-16 cm wide cap. There may be no special odor, but in age there is often an unpleasant meaty odor. Janet Lindgren notes that the taste is mild to somewhat sweet. The cap has no striations, but has white floccose to flattened bits of universal veil tissue on it or hanging from the edge. In age, these floccules tend to disappear and the cap becomes smooth like *Amanita ocreata*. The white to pinkish-buff gills typically reach the stipe and so the specimen may be not recognized immediately as an *Amanita* since *Amanita* gills are typically free. The edges of the gills are finely irregular or minutely floccose, a feature best appreciated with a hand lens. The ring is poorly formed and may collapse on the stipe. The white stipe may bruise pinkish buff or brownish and is thicker at the base, which tends to split and may have a root-like portion. The volva is poorly formed and usually consists of one or two white rings at the base of the stipe, often evanescent.

*Amanita smithiana* is usually seen in the fall, but it also occurs in the spring. The species occurs in pine and mixed conifer forests from the northern Mexican highlands to coastal British Columbia. *Amanita smithiana* has also been reported from Idaho, Arizona and New Mexico and presumably may be found in the adjacent states. *Amanita smithiana* may be confused at times with the so-called “matsutake” or *Tricholoma magnivelare* (*Armillaria ponderosa*). *Tricholoma magnivelare* has a delicious spicy odor; a more persisting (although also collapsing) cottony ring on the stipe, no volva and the flesh is unusually firm. *Amanita smithiana*, in contrast, is fairly fragile and may have no odor or a slightly pungent to “meaty” odor, becoming unpleasant in age.

## Clinical picture, presumed toxin and treatment

The clinical presentation begins with gastrointestinal symptoms (especially nausea and vomiting) usually beginning 2-12 hours after ingestion, although in one atypical case GI symptoms began very early at 20-30 minutes.<sup>116,117</sup> These symptoms usually go on to include kidney failure (or rarely liver failure) within a few days (usually 2-6 days). The atypical findings may represent mixed collections. The initial urine output is dilute and the flow normal or even increased despite abnormal kidney tests. Patients later display the usual oliguria (reduced flow) of acute kidney failure. Almost all of these patients had been searching for autumn specimens of “matsutake”.

The first two reported cases (1976,1982) had only nausea, abdominal discomfort, anxiety and malaise. Neither one had renal failure. The Toxicology Committee of the Oregon Mycological Society reviewed a more severe case near The Dalles.

After a delay of 8 hours, the man had nausea and repeated vomiting before both renal and liver failure ensued. The liver failure was reversible, but he required intermittent kidney dialysis. In 1988, another poisoning occurred, but this time near Vancouver, British Columbia. After a local physician examined him, and found nothing the patient went back to Vancouver after having vomited for three days. He had renal failure, but did not require dialysis.<sup>116</sup>

Another patient, a Laotian immigrant, began vomiting after a 10½ hour delay.<sup>116</sup> On hospital admission, he had a slightly elevated temperature and a very alkaline urine (pH 9). Starting the next day, he became oliguric with the urine output dropping increasingly. He then went into overt renal failure without any evidence of liver toxicity. He was intermittently dialyzed and finally discharged. The patient's relatives gathered a bag of mushrooms from the original collection site, which included *Amanita smithiana*. The very alkaline urine reported is very unusual unless excessive amounts of bicarbonate were given intravenously to combat acidosis. The pH of the urine in other possible cases of *Amanita smithiana* poisoning should be reported.

In 1998, four more patients were reported from the Northwest with *Amanita smithiana* poisoning. All of these recovered after hemodialysis with return to normal kidney function. One of these cases is notable because the patient was seen in the ER and diagnosed as irritant GI poisoning; the patient later returned to the same facility and was admitted with kidney failure (this time with the proper diagnosis).<sup>117</sup>

The exact toxins have not yet been fully identified, but are not similar to the renal toxins in some species of *Cortinarius*. Including Europe, a total of nine cases is known. One compound isolated from *Amanita smithiana*, allenic norleucine (an amino sugar), was toxic in animal cells.<sup>17e,118</sup> The amino acid, pentynoic acid, may also play a role. These compounds were first found in Europe's *Amanita proxima* poisonings. *Amanita smithiana*'s potential toxins require much more study.

One California case in the early 1960's was puzzling at the time and may have been a case of *Amanita smithiana* poisoning.<sup>119</sup> The University of California ascribed the case to *Amanita phalloides* without mushroom identification. It may have been *Amanita smithiana* toxicity, although the symptoms were more severe than in any case noted by Tulloss and Lindgren.<sup>116</sup> Two hours after a meal, this lady had three episodes of urinary urgency and slightly watery eyes followed by vomiting. She was seen at a local hospital for "muscarine poisoning" and treated with atropine, but put out only 3 1/3 ounces of urine over the next three days. She

was then transferred to the University of California. There was no evidence of any liver injury and she survived after intermittent peritoneal dialysis (cleansing solution flushed in and out of the abdomen using the gut wall membranes instead of the artificial membranes of a kidney dialysis machine). Just before discharge, a kidney biopsy was done which showed marked diffuse interstitial fibrosis (microscopic scarring between cells) and regenerating kidney tubules without inflammation.



## Group IV toxins: Orellanine

### Delayed renal failure with *Cortinarius* species

The genus *Cortinarius*, although having no choice edibles, had no significant poisonings attributed to it until a mass poisoning of 102 people occurred in Poznan, Poland in 1952. Stanislaw Grzymala, an epidemiologist, identified the offender as *Cortinarius orellanus* Fr.<sup>120</sup> An unusual cluster of symptoms was usually present in addition to very mild GI symptoms: decreased kidney function with increased urination, intense thirst, marked dryness and burning of the mouth. Some cases progressed to terminal renal failure. Eleven deaths ensued despite renal dialysis. This startling epidemic had a very marked delay in onset of symptoms—from 2 days to 3 weeks. As with amanitin poisoning, early appearance of symptoms usually indicated more severe toxicity. In 1959, Dr. Grzymala reviewed signs and symptoms in 132 *Cortinarius orellanus* poisonings in detail. All but 2 patients had thirst. Some symptoms were rare, but usually not found in other mushroom poisonings—ringing in the ears, pain in the arms and legs, visual defects, difficulty breathing and swelling of the feet. Neurological symptoms and slight jaundice with abnormal liver tests were rare.<sup>121</sup>



*Cortinarius orellanus* photo © Flora Photographica Cortinarius

*Cortinarius orellanus* Fr. has a reddish orange brown cap and gills, a color similar to that of annatto powder; this mushroom fluoresces blue in ultraviolet light. The cap is 3-8 cm across, fibrillose-tomentose to slightly tomentose-scaly, almost smooth in age, dry to moist, convex to expanded, sometimes broadly bossed. The gills are fairly thick, wide and distant from each other. The fibrillose stipe is gold to orange or tawny yellow, darkening towards the base, usually without yellowish remains of the partial veil; it is  $\pm$  equal and measures 3-9 cm long, 4-12mm thick. The spores in mass are rusty yellow brown.

Subsequently, a second species, rather lighter in color, *Cortinarius speciosissimus* Kühner & Romagnesi, produced identical poisonings in several European countries.<sup>122</sup> *Cortinarius speciosissimus* and *Cortinarius orellanoides* Hry appeared to be the same species.<sup>123</sup> Brandrud et al in 1990, however, noted that the oldest name for this taxon (and therefore the correct one) was *Cortinarius rubellus* Cooke.<sup>125b</sup> [Brandrud, T.E., H. Lindström et al: 1990 *Cortinarius*. Flora Photographica, *Cortinarius* HB, Klövervägen, Matfors, Sweden.]



*Cortinarius rubellus* photo © Flora Photographica Cortinarius

Smith and Stunz in the United States had described *Cortinarius rainirensis* from the Cascade Mountains in 1950. The type locations were Mt. Rainier National Park and Barlow Pass.<sup>124,125</sup> These mushrooms had a tawny to orange brown cap with small scales and pale flesh. Attempts to find it again in the same area failed. Ammirati et al in 1985 noted the likelihood of finding *Cortinarius orellanoides* or or closely related taxa in the PNW.<sup>125,125a</sup> Dr. Ammirati suspected

that *Cortinarius rainerensis* and *Cortinarius orellanoides* were in fact the same mushrooms. Without fresh material, however, it was not possible to be sure.

A few years ago, however, Sharmin Gamiet of Abbotsford, British Columbia, found a collection of *Cortinarius rainirensis* A.H.Smith and Stunz in that province.<sup>125a</sup> Review of the Canadian and Washington material showed no difference. As noted, however, the correct name appears to be *Cortinarius rubellus* Cooke.<sup>125b</sup>

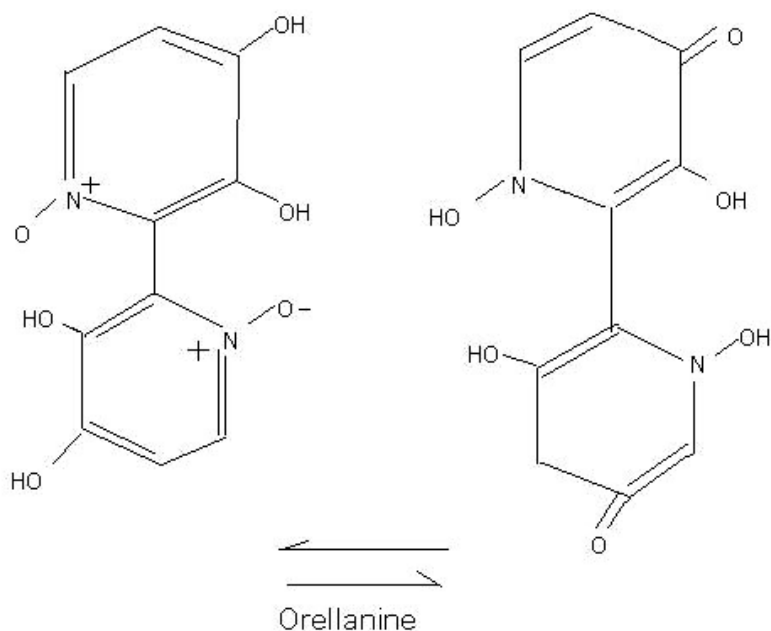
## The genus *Cortinarius* in general

The genus *Cortinarius* has a variety of shapes, but the common feature is a spider-web like veil, the "cortina", in the early stages. The cortina later collapses on the stipe, usually leaving a band colored by rust-brown roughened spores. The genus is subdivided into a number of subgenera using cap and stipe features of dry *or* viscid and base of the stipe bulbous *or* non-bulbous. Added to these are pigmentary differences, microscopic cap features and differences in ultraviolet fluorescence. This genus, fortunately, has never been high on the list of edibles, except for *Cortinarius (Rozites) caperatus*, which has an atypical membranous ring.

Dr. Rolf Singer in his *Agaricales* 4th edition gave over the difficult task of *Cortinarius* classification to Dr. Meinhard Moser of the Institute for Microbiology (University of Innsbruck, Austria).<sup>126</sup> Dr. Moser placed the two toxic species considered *Cortinarius orellanoides* in the sub-genus *Leprocybe*, section *Orellani*. These species are dry-capped, usually 3-8.5 cm in cap width and fluoresce blue or blue-green under ultraviolet light. *Cortinarius gentilis* Fr., a species described from Europe by Fries, also occurs commonly in Western North America. This member of sub-genus *Leprocybe* may also contain orellanine or a similar toxin.

## Toxins of *Cortinarius orellanus*

An extract of *Cortinarius orellanus* has yielded a crystalline substance—orellanine. Orellanine now appears to be a group of bipyridyl compounds.<sup>127,128</sup> Dried specimens of *Cortinarius orellanus* have retained toxicity up to 60 years.<sup>129</sup> A major effect of orellanine is on alkaline phosphatase, an enzyme system required for the energy derived from transport of phosphate into cells. The energy-producing organelles in cells known as mitochondria are the most damaged.<sup>22</sup>



**Figure 7. Orellanine**

Another group has described the presence of compounds similar to the amatoxins.<sup>130</sup> Orellanine was not always present and a group of cyclopeptides, named cortinarins, was proposed. The presence of cortinarin A and cortinarin B, however, was not confirmed by later studies.<sup>131</sup>

Other than chromatography, two qualitative tests have been described. In the first, the fresh or dried mushroom is crushed in water and after 10 minutes the mixture is filtered. The filtered solution is mixed in equal portions with 3% ferric chloride hexahydrate dissolved in 0.5 N hydrochloric acid. A dark gray blue color suggests orellanine.<sup>132</sup> Pöder and Moser have proposed a more rapid test on fresh or dried specimens.<sup>133</sup> The fresh, dried or even cooked specimen is added to a small amount of water, just enough to pound or grind to a pulp. The mixture is then centrifuged to obtain the liquid test material. A drop of this liquid is then placed onto previously prepared white blotting paper or laboratory "bibulous paper" (paper used to mop up excess fluid from microscope slides). The bibulous paper is pretreated by soaking with a 2 percent solution of ferric chloride in 0.5 N HCl acid and allowed to dry. A control spot is done on a second piece of non-treated bibulous paper. A positive test for orellanine is one showing a central reddish color with an encircling lilac halo.

## Clinical course and treatment

Following a delay of 3-16 days intense thirst, often with burning in the mouth, occur. The victims usually urinate large amounts despite increasing kidney failure. Later urination decreases as kidney tubule cells completely fail and die. Gastrointestinal symptoms may also be present. In severe cases renal tubular necrosis and fatty degeneration of the liver may result in death, usually several weeks later. At least 85%-90% of patients recover over a period of some months. Current intensive therapy should greatly reduce the 10-15% death rates reported from Eastern Europe.

Treatment is largely symptomatic: intravenous fluids and electrolytes, cardiovascular monitoring and early kidney dialysis before irreversible damage occurs. Furosemide, a “loop” diuretic (one which works principally on the thin loops of the kidney tubules), appears to increase toxicity; other diuretics may also be dangerous. However, during renal dialysis one can adjust fluid and electrolyte levels at the same time that one is removing the body’s waste products. Renal damage is primarily confined to the proximal tubules with fibrosis around them. This scarring, when severe, seals off these tubules.<sup>135</sup> Kidney tubule scarring, once done, is not reversible and such patients may require intermittent life-long dialysis. Some improvement occurs in occasional patients, presumably because some tubular cells were swollen with inflammation and not blocked by fibrosis of the lumen.



## Group V toxins: Gyromitrin

### Gyromitrin group

The toxic species of *Gyromitra* are brown, yellow brown or reddish brown. Like other species of *Gyromitra* they do not have gills under the cap, but bear their spores on the wrinkled saddle-shaped cap or on one with a ±brain-like convoluted surface. Grey or black species of *Helvella*, which are similar, but simpler in configuration, contain little or none of the pretoxin, gyromitrin (the protein framework plus the active hydrazine toxin it holds).

Western *Gyromitra* species that contain gyromitrin are *Gyromitra esculenta* (Fr.) Fr. and *Gyromitra infula* (Schaeff. Ex Fr.) Qué. *Gyromitra gigas* and *fastigiata* contain *Gyromitrin* in Europe, but probably not here.<sup>135</sup> There is no evidence that *Gyromitra gigas* (Krombh.) Qué. sensu McKnight and *Gyromitra korfii* (= *Gyromitra fastigiata* sensu McKnight) have caused any poisonings in North America.<sup>125</sup>

The taxonomy of *Gyromitra gigas* and closely allied species is somewhat unsettled because of European confusion about the type collections.<sup>136</sup> Although *Gyromitra esculenta*, *fastigiata* and the *gigas/montana* group are eaten regularly, poisonings are quite rare in Western North America. There are traces of gyromitrin in *Helvella crispa*, *lacunosa* and *elastica* and possibly in *Gyromitra sphaerospora*. The distribution of *Gyromitra sphaerospora* appears to be restricted to Montana and adjacent areas of the Northern Rocky Mountains.



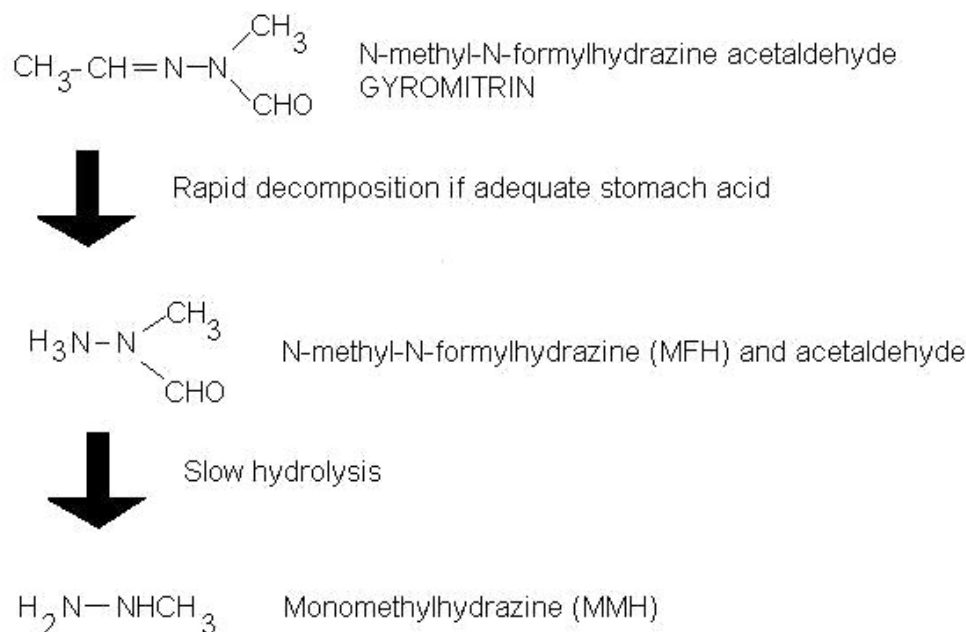
*Gyromitra esculenta* photo © Fred Stevens

*Gyromitra esculenta* has the usual dry to moist, hollow, brain-shaped convolutions on a 3-10 cm irregular subglobose head on a hollow stipe. The color of the convolutions externally varies from clay brown to a darker bay brown; internally the convolutions and the stipe are a pallid off-white. The stipe is 2-5 cm long and 1-2.5 cm thick, white to pale brown, but often tinted with a dark pinkish to reddish brown color reminiscent of the cap. The stipe is fairly smooth, round to somewhat compressed or grooved, hollow, although the remaining central canal may be small.

*Helvella* and the other non-gilled genera containing gyromitrin (usually in lesser amounts) have other non-standard mushroom shapes—saddle, disk, spatula etc. and are not described here, except to note that *Helvella* is similar, but with a more simple shape, commonly resembling a saddle. These other non-gilled genera include such species as *Otidea onotica*, *Cyathipodia macropus*, *Spathularia flavida*, *Leotia lubrica* and *Cudonia circinans*.<sup>137</sup>

## Gyromitrin toxins

Gyromitrin is one of a group of protein-bound hydrazines, whose pre-toxins are found mostly in the genus *Gyromitra*. The principal pre-toxin is gyromitrin (N-methyl-N-formylhydrazine) that breaks down quickly in the stomach and duodenum to acetaldehyde and N-methyl-N-formylhydrazine. Hydroxylation of this compound forms monomethylhydrazine (MMH) and other hydrazines. The methylformyl-hydrazones of pentanal, hexanal and also 3-methyl-butanol are also present in *Gyromitra esculenta* and other toxic *Gyromitra* species.



**Fig.8. Gyromitrin hydrolysis to monomethylhydrazine (MMH)**

MMH has a very narrow range of toxicity, which may account for some of the marked variation in actual symptoms. In addition, there may be less toxicity in western forms, although this hypothesis is unproven. The French have reported decreased MMH in the Spring and also at higher elevations, the latter possibly due to enhanced ultraviolet light degradation.<sup>137</sup> Western *Gyromitra* species commonly occur in the Spring and usually at relatively high altitudes where ultraviolet light is increased. These conditions help explain the benign course of poisoning in the Western areas of the US as compared to the Mid-West, where poisonings have often been severe, even causing death. Comparison is difficult, however, since *Gyromitras* are infrequently collected in the West, appearing only slightly ahead of the highly edible morel in mountain areas. The US military first

studied MMH at Wright-Patterson Air Force Base in Ohio, because this volatile compound had been used as rocket propellant.<sup>138</sup>

MMH interferes with the action of pyridoxal phosphate and others of the vitamin B6 group by inhibition of the enzyme pyridoxine kinase. Vitamin B6 is needed as a co-factor in many enzymatic processes and a lack of pyridoxine kinase results in a decrease of GABA (g-aminobutyric acid), an inhibitory neurotransmitter in the brain. GABA reduces electrical overload on cerebration by decreasing stray signals—what one might call “static”—which tend to overwhelm our thinking. To do without GABA in our brain cells has been likened to attempts to hear a whisper at a rock concert.

MMH also attacks the enzyme GAD (glutamic acid decarboxylase), which reduces the conversion of glutamic acid to glutamate. Glutamate is needed for the synthesis of GABA and, along with glutamic acid, acts as an excitatory neurotransmitter. The decrease of both cerebral GABA and of cerebral glutamate probably account for most of the CNS symptomatology. The gastrointestinal symptoms, methemoglobinemia, liver and kidney failure require other mechanisms and remain unexplained. Monomethylhydrazine induces fetal abnormalities in experimental animals. *Gyromitra* species and other ascomycetes should not be eaten during pregnancy, especially in the first 3 months.

## The clinical picture of gyromitrin poisoning

Since MMH is unstable with heating: boiling, drying and canning usually remove all or most of this toxin. The cooking fumes have caused dizziness, nausea and malaise.

Recovery from gyromitrin poisoning ensues in one to three days, unless severe methemoglobinemia, hemolysis, central nervous system symptoms, liver or kidney failure supervene. Liver failure occurs more often than renal failure. The fatality rate in the 1960's was as high as 2-4% in the Mid-West, but deaths are rare now. No deaths have occurred in the Western states. Other than lightheadedness from inhaling cooking fumes and GI symptoms from eating them, gyromitras are singularly symptom-free in the West. Five poisoning cases presenting themselves to Idaho hospitals survived.<sup>91</sup> Wells and Kempton reported a case of *Gyromitra infula* poisoning in Alaska with symptoms compatible with MMH toxicity. However, the species was later determined to be *Gyromitra ambigua*, a species in which the presence of gyromitrin had not previously been documented.<sup>125,139</sup>

The usual presence of headache, often fever and some of the CNS symptoms (vertigo, loss of coordination, seizures) and particularly a history of false morel ingestion distinguish this syndrome from amatoxin poisoning. Without a history of *Gyromitra* ingestion, the clinical picture is to some extent a peculiar combination of the symptoms found in amanitin and ibotenic acid poisonings.

However, there also may be hemolysis (destruction of circulating red blood cells) with free hemoglobin in the urine. Methemoglobinemia may also occur in which the hemoglobin (a ferrous heme) is oxidized to methemoglobin (a ferric heme). The ferric iron form picks up oxygen very poorly and cyanosis occurs along with shortness of breath. An initially rapid pulse due to dehydration and stress may give way to a slow one and more serious cardiac arrhythmias may occur. Neither hemolysis nor methemoglobinemia occur in amanita poisoning.

### **Treatment of gyromitrin toxicity**

The initial treatment is charcoal (50-100 grams or 1 mg/kg of body weight), antiemetics, fluid and electrolyte replacement. Sorbitol may be used to provoke diarrhea, but this treatment is hazardous unless central venous pressure or right atrial pressure is measured. The one-time dose of sorbitol is 1-2g/kg of body weight for adults and 1-1.5 g/kg for children under 12 (roughly 150 grams for adults and 50 grams for children as the total amount). If methemoglobinemia is high (over 20-30% or with the patient showing signs or symptoms), the use of 1% methylene blue (0.1-0.2 ml/Kg body weight of 1% solution) intravenously is indicated. This dose may be repeated every four hours if methemoglobinemia is refractory.

Seizure precautions should be in effect. If convulsions do occur, they may be controlled by the standard anticonvulsants given intravenously. If an IV line has not been established and a sedative medication is desired, a minor tranquilizer with fairly rapid onset of action such as oxazepam is best. If convulsions occur in a child, the pediatric dose of diazepam is 0.1 mg per kilogram of body weight. Newer anticonvulsants are likely to replace diazepam and neurological consultation should be obtained if time and circumstances allow. Moderately large doses of pyridoxine such as 1 mg per kg IV will combat to some degree CNS toxicity due to vitamin B6 inhibition by the toxin. High doses of B6 (such as the 25 mg/Kg once advised) should be avoided, since vitamin B6 in large dosage may cause neuritis.

In addition to routine chemistry tests and complete blood cell count, the blood should be monitored for free hemoglobin, methemoglobin and haptoglobin.



Severe hemolysis may require infusion of packed red cells or exchange transfusions. Kidney and liver failure are treated as usual. Some of these drugs may be unsafe in pregnancy as little data is available. However, the hydralazines of MMH poisoning are probably much more toxic to a fetus.

## Group VI toxins: *Paxillus involutus*

### **Autoimmune hemolysis with *Paxillus involutus***

Raw or poorly cooked *Paxillus involutus* may cause severe gastrointestinal symptoms, but the red cell destruction and hemoglobin changes of severe poisoning are due to sensitization to the mushroom. Although rare exceptions have been noted, this mushroom poisoning usually reflects repeated ingestion of the mushroom over a period of years, gradually producing antibodies directed against red blood cells. Then the acute syndrome intervenes following a new ingestion of the mushroom. Most patients are elderly, reflecting the years it may take to build up sensitivity and to allow a decrease in the body's auto-antibody defenses.



*Paxillus involutus* photo © Michael Wood

There have been only two cases in the West. Janet Lindgren, chairing the Toxicology Committee of the Oregon Mycological Society, noted two cases just across the Oregon border from Portland in Vancouver, Washington. Of these two cases, one victim had some loss of vision due to central retinal necrosis. These appear to be the only North American cases. Seattle collections over a number of years—“cooked, pickled and raw”—have led to no toxicity.<sup>142</sup>

This medium-sized, squat, brown, rather ugly mushroom with olivaceous tints does not seem unusual at first glance except for its depressed cap and inrolled cap edges. However, on picking, it readily bruises a dark brown, which may be reddish at first, but almost immediately becomes a dingy or slightly olivaceous dark brown. The gills are striking in that they are close and run down the stipe. Like the pores in boletes, one's thumbnail readily scoops out the gills in mass from the cap.

The cap of *Paxillus involutus* is dry to subviscid, dull, matted with fibrils and has a hairy margin that becomes very strongly inrolled early in its development. The color varies from a slightly yellowish to slightly rusty brown, usually with olive tones. It bruises as noted above and the crowded, deeply decurrent gills tend to fork, again suggesting the fruiting body of a bolete: one can scoop out the gills from under the cap, much as one can scoop out the tubes from boletes with their sponge-like, but more regular tube mouths. The cap is 4-12 cm across and perched on a 2-10 cm long stipe, equal or enlarged downwards. The spore print is yellow-brown. These mushrooms spring up solitary or scattered in small groups, most commonly on decaying wood near conifers in the fall. They also occur on garden paths and in mixed woods.

Eaten primarily in Europe, this mushroom usually produces only gastrointestinal symptoms, which begin in 1-4 hours. Malaise, weakness and sweating may be present. However, if autoimmune hemolysis has taken place, the clinical course may progress to changes in consciousness, cardiovascular collapse, kidney failure and other organ problems associated with autoimmune hemolysis.<sup>143,144</sup> A hemoagglutination test for this poisoning using cooked, freeze-dried powder from *Paxillus involutus* is available in Europe.<sup>18</sup>

A variant of the hemoagglutination test noted above can be carried out with fresh *Paxillus involutus* gently heated in a small amount of physiologic saline for 30 minutes. 0.2 ml of this extract is added to 0.1 ml of the patient's serum and incubated at 37° C (98.7° F) for 30 minutes. In the next step, 0.2 ml of a 10% suspension of normal donor red blood cells is added and incubated at 37° C for 60 minutes. Then the resultant mixture is briefly centrifuged to allow examination

of a more concentrated sample of red blood cells. A positive test is agglutination (clumping of red cells) under the microscope.<sup>145</sup>

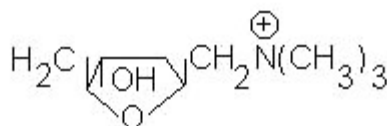
Usually a description of eating a squat brownish mushroom (cap wider than height of stalk), a history of repeated ingestion and the occurrence of hemolysis make diagnosis a rather easy task. Laboratory abnormalities include a decreased red cell count, elevated white cell count, free hemoglobin in the serum with a corresponding fall in haptoglobin (to which it binds) and abnormal kidney function tests. Tests for standard autoantibodies are negative.

Treatment is largely directed at stabilizing the cardiovascular system, managing fluid and electrolyte requirements and reducing symptoms. Intravenous high-dose dexamethasone (an anti-inflammatory steroid) has been used to reduce the immune response, but its efficacy is not proven. Renal dialysis should be available. The best treatment for severely ill patients seems to be plasma exchange transfusion with 5% albumen.<sup>145</sup>

## Group VII toxins: Muscarine

### The muscarine group toxins

Muscarine, although found in 1869, did not have its simple amine structure identified until 1954 by Eugster and Waser—a span of 85 years.<sup>146</sup> Three years later the compound was synthesized and it was determined to be an oxoheterocyclic quaternary salt, 2-methy-3-hydroxy-5-trimethyl-ammoniummethyltetrahydrofuran chloride.<sup>147</sup> Fortunately, the ring structure is simpler to inspect than it is to visualize from its long name.



L-(+)-Muscarine

**Fig. 9. Muscarine**

This toxin produces muscarine poisoning by strongly attaching to muscarinic acetylcholine receptors in the parasympathetic nerves and initiating an acetylcholine effect on organs supplied by these nerves (part of the non-voluntary nervous system). The enzyme acetylcholinesterase, which breaks down acetylcholine, is not effective against muscarine.

Muscarine toxicity produces SLUDGE, the acronym for Salivation, Lacrimation (formation of tears), Urination, Defecation, Gastrointestinal upset and pulmonary Edema. Pulmonary edema (fluid in the lung spaces) occurs only with very severe poisonings.



## General description and occurrence

Muscarine poisoning occurs primarily in the genera *Clitocybe* and *Inocybe*. The genus *Clitocybe* has white spores and white or pale-colored gills that are bluntly attached or run down the stipe. The caps are convex, plane or centrally depressed. The species of *Clitocybe* considered here are  $\pm$  white.<sup>148</sup>

Widely occurring *Clitocybe* species in Western North America reported to contain muscarine are *Clitocybe dealbata* (Fries) Kummer subspecies *sudorifica* (Peck) Bigelow, *Clitocybe cerussata* (Fr.), possibly *Clitocybe dilatata* Pers. ex Karsten and species apparently close to the European *Clitocybe gibba* and *Clitocybe rivulosa*<sup>125</sup> Dr. Bigelow in his *North American Species of Clitocybe Part I* considers *Clitocybe gibba* (Fr.) Kummer and *Clitocybe rivulosa* (Fr.) Kummer to be species close to or identical with *Clitocybe catina* (Fr.) Quélet and *Clitocybe splendoides* Bigelow respectively. In addition, many *Clitocybes* contain smaller or variable amounts of muscarine and the various assays do not always agree.



*Clitocybe dealbata* photo © Fred Stevens

The genus *Inocybe* tends to have small conic to bell-shaped caps that are often brown or display rather pallid colors. The caps are also typically radially

fibrillose, splitting or scaly and less commonly smooth. The margin may be lacerated in some of the older specimens. The gill edges are finely fringed in some species (most easily seen under a strong hand lens such as the Hastings Triplet). There is a fine web-like partial veil seen in young specimens, which disappears at maturity, but often leaves a roughened area on the stipe. If a ring is present it is fibrillose and often incomplete. Often the upper portion of the stipe is white and finely stippled. Odors of *Inocybe* species are often peculiar—earthy, spermatic, fruity or like corn silk. The spore print is a dull brown to dull yellow brown, often “rusty brown”.

Because of the studious work of Malone and others on specimens identified by Dr. Daniel Stuntz (Washington), the list of western muscarinic *Inocybes* is quite long.<sup>149,150</sup> Toxic species that occur in California include *Inocybe albodisca*, *I. decipientoides*, *I. fastigiata* (= *I. rimosa*), *I. geophylla*, *I. kauffmanii*, *I. lacera*, *I. lilacina* (= *I. geophylla* var. *lilacina*), *I. mixtilis*, *I. olympiana*, *I. pudica* (= *I. whitei*), *I. pyriodora* (= *I. fraudans*), *I. sororia* and *I. subdestricta*. At least four toxic *Inocybes* species are found in Mexico: *Inocybes fastigiata*, *I. geophylla*, *I. lacera* and *I. lilacina*.<sup>26</sup> Pacific Northwest species include the additional species of *Inocybe cinnamomea*, *I. gausapata*, *I. griseolilacina*, *I. napipes*, *I. nigrescens*, *I. oblectabilis*, *I. obscuroides*, *I. picrosma*, *I. praetervisita*, *I. terrifera* and *I. xanthomelas*. Almost all of these species extend eastwards to Colorado and the southern Rocky Mountains.





*Inocybe geophylla* var. *lilacina* photo © Fred Stevens

The most common muscarine poisoning in California, however, appears to be that caused by *Boletus satanas*. Possible muscarinic symptoms with this bolete have been reported most years in California's Grass Valley and in other Sierra Nevada foothill areas. However, no members of the MSSF toxicology committee have confirmed the presence of SLUDGE. Lillian Mott, a long-time member of the MSSF, described possible muscarinic symptoms in one patient.<sup>151</sup> This large bolete has a massive bulbous stipe and red pores on the undersurface that blue with injury. Some people eat *Boletus satanas* with impunity. It appears that at least the GI symptoms are avoided by careful cooking.





*Boletus satanas* photo © Michael Wood

Stadelmann and his co-workers noted that muscarine is present in *Boletus calopus* and *Boletus luridus*, but only in small amounts.<sup>152</sup> Muscarine presumably is present in *Boletus pulcherrimus*.<sup>24</sup> *Boletus pulcherrimus* produced what appeared to be muscarine poisoning in at least one report.<sup>153</sup> *Boletus haematinus* has not been studied and is readily confused with either *Boletus pulcherrimus* or *Boletus satanas*. These large red-pored boletes and similar species should be avoided. Dr. Thier's *California Mushrooms—A Field Guide to the Boletes* (California Boletes on this web site) gives good descriptions of *Boletus calopus* var. *frustosis* (our Western species) and *Boletus erythropus*.<sup>154</sup> *Boletus calopus* var. *frustosis* usually has red on the cap or stipe and has ± yellow tubes bruising blue; it may be a different species from the European *Boletus calopus* and may or may not contain muscarine.

*Mycena pura*, a delicate gilled mushroom which is widespread in North America and can cause gastrointestinal upsets, may also contain muscarine. *Omphalotus olivascens*, a large dingy yellow-green fungus, which grows in shelves on

senescent western trees and stumps, apparently contains muscarine, although it usually causes only gastrointestinal symptoms. *Panellus serotina*, a common small greenish-yellow to somewhat purplish shelving species also clusters on dead wood. European authors report that their specimens contain muscarine.<sup>56d</sup>

## The clinical picture of muscarine poisoning

Muscarine activates the parasympathetic nervous system. Symptoms of sweating, salivation, nausea, teary eyes, small pupils and/or pupils sluggishly reacting to light, slow heart rate, increased urination and sometimes diarrhea occur within 20-30 minutes. Severe poisonings may produce more serious symptoms, including bronchial constriction, slow heart rate with faster “escape rhythms” and pulmonary edema. Tremors and dizziness apparently reflect diminished blood flow to the brain or mild activation of the glutamate hierarchical system. Muscarine itself passes only with difficulty into the brain. One Colorado case of *Amanita muscaria* poisoning appeared to cause muscarinic effects and excessive lactation in a nursing woman.<sup>155,40b</sup> There are no reports of muscarine damage to the fetus. There is one case of a mild asthma attack.<sup>91</sup>

## Treatment of muscarine poisoning

The treatment for this symptom complex is atropine titrated to reduce symptoms, induce slight dryness of mouth and restore normal or nearly normal pupil size. The initial adult dose of atropine is 0.5 to 1.0 mg given very slowly intravenously. Repeat doses of 0.5-1.0 mg can be given at 10-20 minute intervals to either of two endpoints: A. 1 mg doses until bronchial hypersecretion, pulmonary edema and significant cardiac arrhythmias have cleared or B. 0.5 mg of atropine until the pupils are normal or nearly so. The second end-point may take longer to achieve; its use requires increased diligence and slower administration of atropine to avoid overdosage. Usually no more than 2.5 mg of atropine is required.

For severe poisoning with marked bradycardia (slowing of heart rate), AV or ventricular block (slowing of heart rate by blocking of cardiac electrical conduction), escape rhythms (rapid rhythms to escape cardiac conduction block) or pulmonary edema, give 1.5 mg IV at once and titrate from there. The pediatric dose is not strictly according to weight; the range is 0.25-1.0 mg total for children under 12. Dr. Denis Benjamin, a US physician, gives the pediatric dose as 0.01-0.3 mg/kg body weight with a repeat dose at 10 minute intervals up to a total of 1 mg in children.<sup>82,101</sup>



Always consider the possibility of insecticide poisoning. The organophosphates, some carbamates and at least one organochloride (Malathion) also produce muscarinic symptoms, but do so by interfering with acetylcholinesterase. This enzyme inactivates acetylcholine. The toxicity is usually severe and may require very high doses of atropine. These insecticides are also able to enter the central nervous system. In skeletal muscle, in addition to acetylcholine receptors, there are also receptors responsive to nicotine. Some of these insecticides bind to these nicotinic receptors and cause such skeletal muscle symptoms as weakness, fasciculations and paralysis.<sup>156,157</sup> Since atropine does not bind to nicotinic receptors, treatment of skeletal muscle symptoms requires the use of pralidoxime hydrochloride, a cholinesterase reactivator.

## Group VIII toxins: Hallucinogenic indoles (psilocybin)

### The "New World" history of psilocybin containing mushrooms

This group of fungi is interesting as they played an important role in some Mexican and South American religions. These rites were suppressed so vigorously by the Spanish Christians that a prominent botanist, William Safford, proclaimed in 1915 that only peyote cactus buttons were used.<sup>158</sup> R.J. Weitlaner rediscovered them in the state of Oaxaca in 1936.<sup>159</sup> Richard Schultes followed up on this finding and placed some species in the Harvard herbarium.<sup>159</sup> Gordon Wasson, a former New York banker and self-trained ethnomycologist, studied them in 1953 with the aid of the French mycologist, Roger Heim, and publicized their use in shamanic ceremonies.<sup>160</sup> Wasson not only reported these ceremonies and took part in them, but also recorded four cassette tapes of the religious rite as performed by the shaman, Maria Sabina.<sup>161</sup>

Dr. Rolf Singer, a world expert on tropical and subtropical fungi, also headed for the Mexican state of Oaxaca where Gordon Wasson and Roger Heim had found most of the hallucinogenic species. Gordon Wasson, just prior to the second Aspen Conference on mushrooms, recounted for the participants a rather colorful story of Dr. Singer "stalking" Gordon and Valentina Wasson's party in Mexico and finally the Singer/Wasson meeting in a hut where both parties had sought protection from heavy rains.<sup>162</sup> Given Dr. Singer's extensive travels and expertise (and rather less flamboyance), one must discount some of this story. However, R. Gordon Wasson's account provides some of the background for further events and there may have been other observers in the rain forest.

A few years later Roger Heim published in French a full description of what he intended to name *Psilocybe wassonii* (Heim). Gordon and Valentina Wasson first collected this species in 1955. Their colleague, the mycologist Roger Heim, had first described it provisionally as *Psilocybe mexicana* var. *brevispora* in 1956. The following year he recognized this as a new species and he re-described the species (without the Latin diagnosis) for a report to the French Academy of Sciences.<sup>163</sup> Dr. Heims' formal description and Latin diagnosis was not published until the Mycologia issue of 4/29/58, 25 days after Rolf Singer and Alexander Smith named that *Psilocybe* species *Psilocybe muliercula* in the same journal's preceding issue. The correct name, therefore, is *Psilocybe muliercula* Singer and Smith. Roger Heim and others threw a few uncomplimentary darts at the editorial board of

Mycologia for what they saw as favoritism, but then the rivalries seemed to settle down.

Twenty years later, distressed by a lot of non-scientific babble about “magic mushrooms”, Dr. Smith wrote for Mycologia an article entitled *Comments on hallucinogenic agarics and the hallucinations of those who study them*.<sup>164</sup> This brief article faulted, for example, a footnote in Jonathon Ott’s *Hallucinogenic Plants of North America* where the latter states that Gordon Wasson discovered the Mexican mushroom cults for the English-speaking world, although at the same time citing Schultes’ prior work. Dr. Smith also noted that, in another article, Jonathan Ott considered *Psilocybe subaeruginascens* and *Psilocybe aeruginascens* to be the same species. Dr. Smith retorted that the surface of the cap and other features of these two taxa were not the same. In the 4th edition of his “Agaricales”, Dr. Singer, like Dr. Smith, considers these two species to be different.<sup>126</sup>

Jonathon Ott then demanded a nine-page response in Mycologia. The editorial board of Mycologia accepted the first 2½ pages, but not the full text. The complete text was later published as “Mr. Jonathan Ott’s Rejoinder to Dr. Alexander H. Smith” as part of the Botanical Museum of Harvard University’s series #6 in *Ethnomycological studies*. In this response to Dr. Smith, the mycological competition of the various workers on the genus *Psilocybe* was re-hashed all the way back to 1952, when the Wassons first went to Mexico.

The whole episode has an air of black humor about it that is worthy of a movie script. In studying what actually happened, Jonathon Ott wondered if the CIA (who had studied LSD) had also looked at hallucinogenic psilocybin mushrooms. Using the new Freedom of Information Act, he discovered that the CIA had wasted some of the taxpayer’s money by putting the Wassons, Roger Heim, Rolf Singer and Alexander Smith under surveillance for a period of time. Were some of the “stalkers” CIA personnel burning our hard earned dollars?

## Taxonomic and legal problems

Western gilled mushrooms containing hallucinogenic indoles are primarily found in the dark spored genera of *Psilocybe*, *Stropharia* and *Panaeolus*. The last genus has black spores that do not fade in concentrated sulfuric acid. Mycelia at the base of the stipe in *Panaeolus* may bruise blue, but not the flesh. Dr. Guzman, the definitive authority on these fungi, places all of these segregates from *Panaeolus* as subgenera under that genus.<sup>165</sup>

The federal drug enforcement act lists mushrooms containing psilocybin and psilocin as controlled substances under Schedule I (no medical use). The original listing of psilocin in the 1970 “Comprehensive Drug Abuse and Control Act” was misspelled as “psylocin”. Hallucinogenic indole-containing fungi are on Schedule I, primarily because such mushrooms are often used for “recreation”. Schedule I lists substances that cannot be prescribed by a physician even on a “triplicate” (legal narcotic form). Mere possession of psilocin in any amount is technically illegal. However, collectors of LBM’s (little brown mushrooms) without these indoles have been cited occasionally. That section of the 1970 law is further flawed in that most encounters between man and possibly hallucinogenic fungi are quite innocent. Most mycologists collect for identification, photography, mushroom fairs and university herbaria. *Psilocybes* and related genera (if non-bluing) are difficult to identify and may or may not contain hallucinogens. Some species are only “latently” indole-hallucinogenic.

"Latently indole-hallucinogenic" is a vague term. Use of the word “latent” or its forms covers a good number of interpretations: 1. The mushroom in question may have these alkaloids, but only at a given phase of its growth. 2. The compounds are present, but only in minute amounts and may be toxic only to susceptible persons or children. 3. The compounds are present only in specimens from one site or habitat, but not from another. 4. Alkaloids are present, but require specific growth requirements or handling to be biologically available. 5. The “latently indole-hallucinogenic” compounds are only detected after certain laboratory manipulations.

Collecting these “magic mushrooms” for recreational use, however, is quite hazardous unless careful rules are used to gather only the bluing species. Additionally, dried “street” specimens have been laced with LSD or worse. See below for what is almost certainly a death from *Psilocybe cyanescens*, a bluing species, in a six-year-old child.

## General description and occurrence

The genus *Psilocybe* has a variety of cap configurations, sometimes with an obtuse or acute umbo, but rarely with much of a central depression. The caps are usually ± brown; typically viscid, smooth and fading to ochre or yellow brown with drying. Caps and stipe are not particularly fragile and some viscid caps have the upper gelatinous layer separable. The gills are attached to the stipe. The spore print is typically dark purple brown. Species with a high amount of active indoles usually bruise blue or blue-green. The genus *Psilocybe* occurs in a variety of habitats including wood chips, but never on living or dead trees.

(In the following lists of species for Western North America, those *Psilocybe* species also found in Mexico are underlined.)

Active species of *Psilocybe* known to occur in California include *Psilocybe aeruginosa* (Curtis:Fr.) Noordeloos [= *Stropharia aeruginosa* (Curtis: Fr.) Quélet], *Psilocybe azurescens*, *Psilocybe baeocystis*, *Psilocybe caeruleascens* {but almost certainly imported from central Mexico}, *Psilocybe caerulipes* (?), *Psilocybe cyanescens* [San Francisco to Alaska], *Psilocybe cyanofibrillosa*, *Psilocybe inquilina* (Fr. ex Fr.) Bresadola, *Psilocybe mairei*, *Psilocybe merdaria*, *Psilocybe pelliculosa*, *Psilocybe physaloides* (Bull. ex. Merat) Quélet [Santa Barbara to Alaska], *Psilocybe semilanceata*, *Psilocybe squamosa* and *Psilocybe subaeruginascens*.<sup>166,167,168,169</sup> The citations are from Paul Stamet's *Psilocybin Mushrooms of the World*.<sup>167</sup> The material labeled *Psilocybe caerulipes* (Peck) Saccardo in San Francisco State's Harry D. Thiers Herbarium from Golden State Park may be an undescribed species or variety as the spores are longer (up to 11µm) than those described by Singer and Smith in 1958.<sup>170</sup> Additionally, *Psilocybe caerulipes* has an Eastern U.S. distribution. Dr. Guzman made two collections of what he called *Psilocybe caerulipes* in Mexico<sup>167</sup> and those collections should be compared with the California material and with the Eastern *Psilocybe caerulipes*.





*Psilocybe cyanescens* photo © Fred Stevens

The PNW has additional *Psilocybe* species: *Psilocybe caerulea* (Kriesel) Noordeloos [= *Stropharia caerulea* Kriesel], *Psilocybe fimetaria* (Orton) Watling [= *Stropharia fimetaria* Orton], *Psilocybe liniformans* var. *americana*, *Psilocybe luteonitens* (Peck) Saccardo [= *Stropharia umbonatenscens* (Peck) Saccardo], *Psilocybe moellerii*, *Psilocybe montana*, *Psilocybe pseudocyanea* (Desmazieres: Fr.) Noordeloos [= *Stropharia pseudocyanea* (Desmazieres) Morgan], *Psilocybe silvatica*, *Psilocybe strictipes* Singer and Smith, *Psilocybe stunzii* Guzmán and Ott and *Psilocybe subfimetaria*.<sup>171</sup> Most of these taxa occur in British Columbia in addition to the reported *Psilocybe baeocystis*, *cyanescens*, *semilanceata*, *strictipes* and *stunzii*.<sup>167</sup> *Psilocybe physaloides* (Bull. ex. Merat) Quélet) has a stupendous range from Santa Barbara to Alaska.

Many more *psilocybe* species probably occur only in Mexico, the gulf states and Middle America. These include two collections of *Psilocybe caerulipes* (a primarily Eastern US species), *Psilocybe herrerae*, *Psilocybe hoogshagenii* Heim sensu lato, *Psilocybe jacobsonii*, *Psilocybe mammillata*, *Psilocybe muliericula*, *Psilocybe*

pelliculosa (Smith) Singer and Smith. Those species favored by the shamans of Oaxaca are *Psilocybe aztecorum*, *Psilocybe caerulescens*, *Psilocybe mexicana* and *Psilocybe zapotecorum*. The shamans also use raw *Lycoperdon mixtecorum* and *Lycoperdon marginatum* in some areas of Mexico. Their use in these rites apparently is symbolic as these puffballs have no hallucinogenic effects.<sup>178a</sup>

The genus *Panaeolus* typically has a ± white, yellow-brown or brown cap, often ± conic or bell-shaped, viscid or dry, occasionally with remnants of a partial veil at the edges. A separable surface layer is not present. The pallid gills are attached to the stipe and typically are mottled black with the dark spores, because the spores do not all mature at the same time and the immature spores contain little pigment. A ring may be present.

Western North America has 1-2 active species of *Panaeolus*—*Panaeolus subbalteatus* and one possibly active species—*Panaeolus semiovatus*.<sup>167,166</sup> Other species such as *Panaeolus castaneifolius*, *Panaeolus fimicola* and possibly *Panaeolina foenisecii* (= *Panaeolus foenisecii*) are latently hallucinogenic. Stamets notes that a form of the latter, which occasionally bruises blue, has been found in the grassy coastal areas of California and may be a new species.<sup>166</sup>

*Inocybe aeruginascens*, which bruises blue at the base, is widely distributed in western America and is weakly active. Two possibly hallucinogenic, but quite small, *Conocybes* have been reported in the West: *Conocybe cyanopus* (Washington, Colorado, British Columbia) and *Conocybe smithii* (Oregon and Washington). The base, or at least the mycelia just below the stipe, blue in these species. These mushrooms are similar to other *Conocybe* species that contain amanitins. *Hypholoma (Naemataloma) popperianum* is the only indole-containing *Hypholoma* and there is only one voucher specimen from Western North America (Golden Gate Park in San Francisco).<sup>172</sup>

The genus *Gymnopilus* has a rusty brown spore print and is usually found on wood. The caps are usually dry and are often brightly colored. A partial veil is often present and varies from web-like to fibrillose. Psilocybin was found initially in five species: *Gymnopilus luteus*, *G. spectabilis*, *G. validipes*, *G. aeruginosus* and *G. viridans*.<sup>173</sup> *Gymnopilus spectabilis* is a common large bright orange mushroom found clumped on logs or stumps in the west, but it does not appear to be toxic as the eastern mushrooms are. Paul Stamets and others have noted that this species with a stipe usually 1-6 cm wide is easily confused with *Gymnopilis ventricosus*, which is known to be hallucinogenic. This species has a wider swollen stalk and very shallow gills.<sup>166</sup> Active species in California and in the PNW are *Gymnopilus aeruginosus* with its dull blue-green or mottled blue-green



cap when young and *Gymnopilus luteofolius* with its somewhat purplish scales and its thick reddish to purplish flesh when young. **Warning:** at least one species of *Gymnopilus* in Austria may contain amatoxins, *Gymnopilus spadiceus* (Singer, 4th edition Agaricales). No amatoxin-containing species of *Gymnopilus* have been found on this continent.



*Gymnopilus spectabilis* photo © Michael Wood



*Gymnopilus luteofolius* photo © Michael Wood

*Cortinarius infractus*, a common species found in temperate zones, contains indole alkaloids.<sup>174</sup> *Conocybe cyanopus* and *Conocybe smithii* are found in the PNW and may occur in Northern California. The basal mycelia stain blue when handled.

*Stropharia coronilla*, an edible mushroom that occurs in the West, presumably caused 2 bizarre Arkansas poisonings in 1976. Two healthy young visitors from Iowa made a soup from fresh *Stropharia coronilla*. About an hour later malaise, severe headache, generalized aching, flushing, incoordination, muscular weakness, difficulty walking and confusion occurred in addition to GI symptoms. Both victims had hallucinations and delirium. One had fever and one lost consciousness. Even the following day they had headache and bone pain.<sup>175</sup>

The University of Arkansas identified specimens from the collecting areas as *Stropharia coronilla*. Photos were sent to Dr. Barry Rumack at the Rocky Mountain Poison Control Center in Denver where both he and Dr. A.H. Mitchell agreed on the identification as best as that could be done without actual material. Although Arkansas locals did look for blue staining *Psilocybes* under cow pies,



the two visitors insisted they had not done so. Some of their symptoms, such as severe headache, flushing and bone pain were not psilocybin symptoms. Another toxin or possibly a chemical contaminant appears to have been present.

*Pluteus salacinus* occurs along the California coast and in other areas of the West. Like the genus *Psilocybe*, it contains psilocybin, but recreational use is probably rare.<sup>8</sup> Its smooth spores are pink in mass.

A few US polypores are stimulants or mild hallucinogens. *Phaeolus schweinitzii* is the most common species. Since it is woody, about the only way it can be used is as a bitter herbal tea. The psychotropic agents may be hispidin and bis-noryangonin. These compounds are also found in kava-kava, a pleasant tasting tea greatly appreciated in Indonesia. Hordenine and probably tyramine & N-methyltyramine are found in two polypores: *Bondarzewia berkeleyi* and *Bondarzewia montanus*. Possession of kava-kava is legal in the United States and is only mildly stimulating.

*Laetiporus "sulphureus"* produced hallucinations in a six year old child, after she had nibbled away at a sulfur shelf.<sup>176</sup> The MSSF Toxicology Committee Survey in 1975 reported one ingestion in which an adult had a tingling sensation in the fingers, a floating sensation, some dizziness and a feeling of disorientation.<sup>177</sup> Western collections of *Laetiporus sulphureus* contain hordenine, tyramine, N-methyltyramine and two unidentified alkaloids.<sup>178</sup> We now know that Western North American *Laetiporus "sulphureus"* is actually 2 or more different species. The *Laetiporus* that grows on Eucalyptus and probably on oaks is *Laetiporus gilbertsonii*.<sup>188a</sup> The *Laetiporus* growing on conifers is *Laetiporus conifericola*.

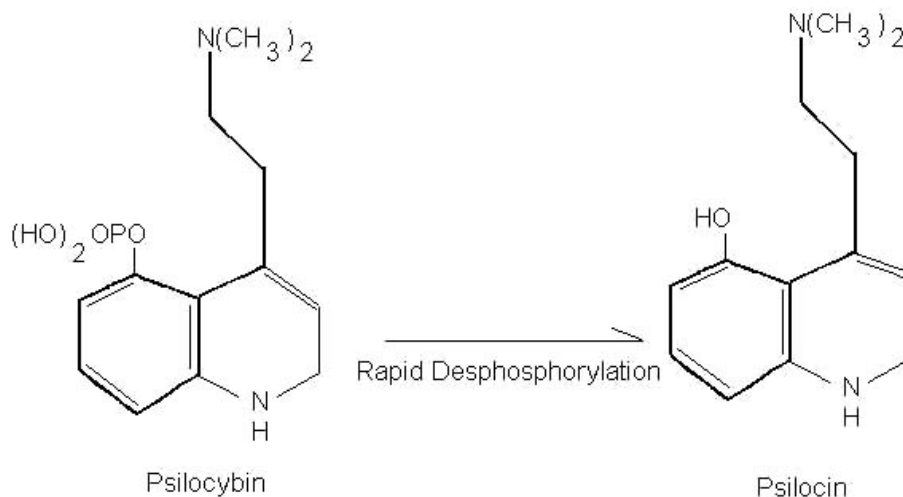
In the Yukon Territory, the native residents chew a polypore found on birch trees—*Phellinus igniarius*. The tough mushroom is burned to an ash and mixed with chewing tobacco and tea leaves. There is only a sedative effect, but first-time users may experience a mild headache.<sup>179</sup>

Occasionally, other fungi produce unexpected hallucinogenic effects. For example, *Hygrocybe (Hygrophorus) punicea* was reported in one California case to have caused GI symptoms with hallucinations and disorientation. Other people, who have eaten this species, have not noted any CNS toxicity. Such isolated reports of toxicity require confirmation of the species involved. They do serve to remind us that little is known of the taxonomy or possible toxicity of many fungi or of the idiosyncratic ways in which particular victims may react.<sup>177</sup>



## Psilocybin and psilocin toxins

Dr. Albert Hoffman at Sandoz isolated the active compounds: first psilocybin and then its dephosphorylated form, psilocin. Psilocybin was the first phosphorylated indole found in nature. Because of the rapid enzymatic conversion of psilocybin to psilocin, psilocin is the toxin actually producing effects in humans. Psilocin mimics the effect of the neurotransmitter, serotonin, by strongly attaching to its receptors and provoking an excessive serotonin-like effect. Normal serotonin effects include regulation of circadian rhythms and of vasoconstriction. Excessive stimulation of serotonin receptors, however, can produce high blood pressure, rapid pulse, sensory distortions and hallucinations.



**Figure 10. Psilocybin**

Many psilocybes blue with bruising or cutting, apparently a reaction catalyzed by cytochrome oxidase. This reaction in hallucinogenic genera corresponds roughly with the amount of psilocin. The exact compound producing this bluing reaction remains to be isolated, although it appears to be a quinone derivative.

## The clinical picture of psilocybin toxicity

Psilocybin-containing fungi vary greatly in the amount actually present or available. Initial symptoms, starting around 20 minutes after ingestion, present with a variety of very individual responses—relaxation, anxiety, light-headedness, nausea and vague abdominal discomfort usually followed by a

sharpness of outline or heightened color for objects and visual imagery with closed eyes. Usually 20 grams of fresh mushroom or 2 grams of dried mushroom is enough to produce true hallucinations. Sometimes panic reactions occur in association with a fear of death or of insanity and an inability to distinguish reality from fantasy.

During the second hour, the closed-eye visual effects increase and are marked by the perception of intense, brilliant colors, often geometric and undulating or bursting forth like fireworks. Sometimes these intense visual effects are superimposed on dream-like shapes, which rapidly appear and disappear. Often there is a perception that time has slowed. The effects continue through a third hour and gradually wind down. An alcoholic hang-over does not usually occur, although mild headache and fatigue have been reported. Subjects in laboratory situations usually found the experience less thrilling, suggesting that the context of ingestion is important.

Systemic effects are due mostly to stimulation of the brain-stem and the sympathetic nervous system. Persons taking insulin or having cardiac problems or high blood pressure are at some hazard, since the systemic effects include lowered blood sugar, increased heart rate and blood pressure. The elderly in general probably have an increased risk.

Children are more susceptible to the toxins than adults; fever, seizures and one death have been reported from the PNW.<sup>180</sup> Paul Stamets reviewed that 1962 case: a 6 year old child who died on the third hospital day. The mushroom photograph was that of *Psilocybe cyanescens*.<sup>166</sup> The child developed fever from 102-106° F and had seizures. Continuous convulsions then occurred despite intravenous therapy with anticonvulsants. She became unable to breathe, had an artificial airway inserted, but eventually died despite good treatment. Autopsy showed brain swelling due to excess fluid and mild pulmonary edema.<sup>180</sup> A San Francisco physician informed the MSSF toxicology committee that he had seen two similar cases in children with seizures. This death almost certainly was *not* the result of an amanitin poisoning. It was due to a bluing *Psilocybe*.

## Treatment of psilocybin toxicity

A panic reaction or accidental ingestion is best treated by supportive therapy; what is usually called “talk-down” treatment. Results of pharmacologic intervention have not been satisfactory. Chlorpromazine and thiorazine-like compounds do not terminate psilocybin effects and may be contraindicated because of their slight atropine-like effects. Psilocybin mushrooms on occasion

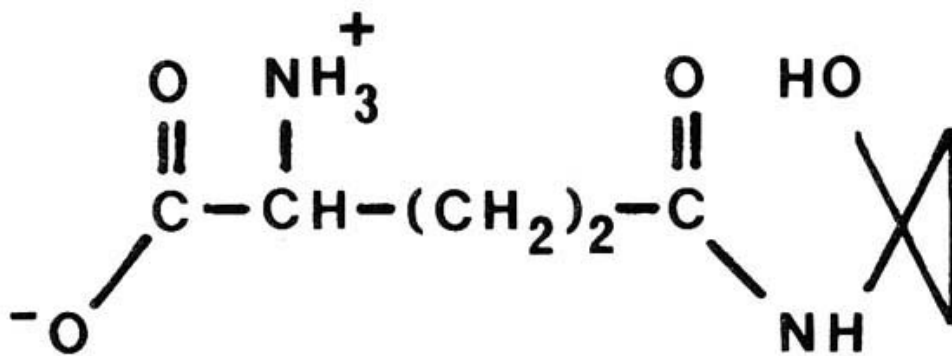
cause mild atropine-like symptoms.<sup>181</sup> If a sedative is desired, a minor tranquilizer with fairly rapid onset of action such as oxazepam is best. If convulsions occur in a child, the pediatric dose of diazepam is 0.1 mg per kilogram of body weight.

Not all reactions are unpleasant. Although 30% of patients in a 2006 John Hopkins study reported fear and paranoia, some 60% found peace and harmony after taking the drug, frequently lasting beyond the immediate experiment. 38% regarded this experience as one of the 5 most meaningful episodes of their lives, up with birth of a first child or death of a parent.<sup>181a</sup>

## Group IX toxins: Coprine

### Disulfiram-like reactions to alcohol

*Coprinopsis (Coprinus) atramentaria*, whose gills deliquesce to a black ink to spread its spores, are the common cause of this syndrome. *Coprinopsis insignis* is an Eastern species with the same toxins, but a few sightings have been reported from Western North America. These inky caps in the former *Coprinus* genus contain a protoxin “coprine” which is converted to 1-aminocyclopropanol— a compound with an antabuse-like reaction with alcohol.



**C o p r i n e**

Fig. 11. Coprine

Antabuse® is used as an adjunct in the treatment of alcoholics. This compound prevents the normal breakdown of alcohol to carbon dioxide and water. It inhibits the enzyme aldehyde dehydrogenase and the excess acetaldehyde causes flushing, throbbing in the temples and usually headache starting 5-30 minutes after ingestion of alcohol.

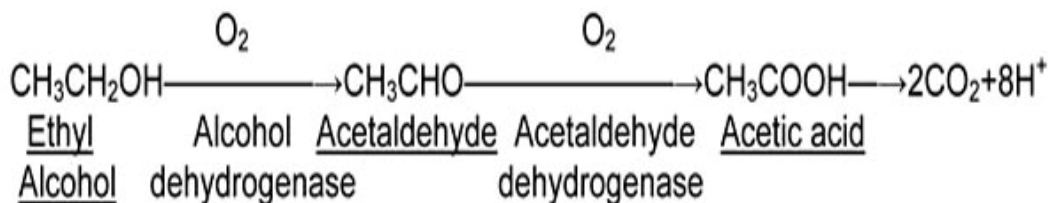


Fig. 12. Acetaldehyde pathway from alcohol to acetic acid

Other symptoms may include nausea, sweating and occasional diarrhea. The normal pathway is shown below, but disulfiram type reactions are blocked to a major extent at acetaldehyde and do not go on to acetic acid and CO<sub>2</sub>.

Because of a very thin, tightly adhering veil, *Coprinopsis atramentaria* has a white cap when very young. The caps rapidly become gray or gray brown, marshmallow-shaped, conical to ellipsoid, then finally campanulate. The young still closed ovoid-elliptical mushrooms have caps 3-7 cm X 2.5-5 cm; the campanulate caps may measure up to 9.5 cm across. The moist caps are finely grooved and decorated with flat grayish scales mostly at the center; the caps may be almost smooth in contrast to many species of the *Coprinus* group. The crowded gills are first white, then grayish brown; finally becoming black as they begin to deliquesce. The stipe is 6-14 cm long and 7-14 mm thick, white with a slightly clubbed base, sometimes slightly rimmed.



*Coprinopsis atramentaria* photo © Tom Duffy



A *Coprinopsis atramentaria* meal ingested with alcohol (or minutes to 8 hours after the alcohol) most commonly sets off the syndrome 3-15 minutes later. Much longer periods for symptoms to appear with *Coprinopsis* meals following alcohol have been reported. The usual recurrence period is given as up to 48 hours later. The author and his wife had a lunch-time reaction with beer after an earlier *Coprinopsis atramentaria* breakfast. We tried the same amount of beer with lunch again at 24, 48 and 72 hours after the initial symptoms and both of us had recurrent, but progressively milder symptoms each time. Both of us had freely ingested *Coprinopsis atramentaria* in the past with alcohol and had had no symptoms previously, a not uncommon report.

*Clitocybe clavipes* (= *Ampulloclitocybe clavipes*), with a wide distribution from Mexico to Canada, produces a similar syndrome.<sup>183,184</sup> Similar symptoms, but usually with more of a GI component, have also been reported with *Pholiota squarrosa*, *Boletus luridus* and *Boletus pulcherrimus*. These mushrooms have little in common and other toxins are likely.



*Ampulloclitocybe clavipes* photo © Michael Wood

It is best to avoid therapeutic interventions. The symptoms are usually transient. Vital signs should be monitored until symptoms have subsided and the blood pressure is stable. Some of the symptoms are caused or worsened by panic and it is very important that the patient understand the expected benign course. By 45 minutes, the episode is usually over. Occasionally, symptoms persist for 2-3 hours.

However, there have been a few severe poisonings requiring monitoring for cardiac arrhythmias. For an extremely rapid heart rate, beta blockers such as propranolol have been suggested.<sup>185</sup> A 53 year old man with unusually severe vomiting had a fatal rupture of the esophagus—a dreadful emergency which requires immediate surgery.<sup>186</sup> Experimental animals have shown testicular abnormalities, but even if that damage could be translated to humans, over a pound a day of *Coprinopsis atramentaria* for a week would be needed to replicate loss of sperm production!<sup>187</sup>

## **Group X toxins: Gastrointestinal irritants, toxins and idiosyncrasies**

### **GI reactions due to fungi usually considered edible**

Most mushroom “poisonings” cause no more than GI distress: abdominal cramping, diarrhea, nausea and vomiting. Although a number of toxins are known to cause only GI symptoms, most of these are poorly understood. There are too many GI toxic fungi to describe here and other sources should be consulted. There is a description of additional GI toxic species appended to this text.

The great variation in the presence of any symptoms between people eating the same collection for a meal suggests that individual susceptibility is important. The leading American mycologist of the 20th century, Alexander H. Smith, picked specimens of *Lepista irina*, an edible mushroom, from a 'fairy ring' of this species in 1958 and checked several specimens under the microscope. 'Fairy rings' of mushrooms, often with 15-40 individuals, grow outward as nutrients in the central area are used up by the fungi. All such mushrooms are believed to be the fruiting bodies of the same mass of mycelia. Therefore, the mushrooms from such a collection should be nearly identical chemically. Although students at the University of Michigan had been picking them for food and Dr. Smith had done so for several years, this time everyone partaking of his collection enjoyed them, except for a husband and wife who woke up in the middle of the night with

gastrointestinal symptoms. The most likely assumption was individual idiosyncrasy.<sup>188</sup>

The true *Laetiporus sulphureus* does not occur along the Pacific Coast. The *Laetiporus* that grows on *Eucalyptus* and probably on oaks is *Laetiporus gilbertsonii*.<sup>188a</sup> The *Laetiporus* growing on conifers is *Laetiporus conifericola*. Both species are edible and highly sought after. Each can cause occasional idiosyncrasies. The San Francisco Toxicology Committee in 1975 surveyed its members along with some amateur mycologists in the PNW.<sup>177</sup> Examination of the 158 reports of fungal poisoning surprisingly revealed that seven persons had had almost identical reactions to *Laetiporus*—nausea and often vomiting within 5-45 minutes of ingestion of the sulfur shelf. Symptoms lasted a few minutes to 2 ½ hours. Numerous other cases surfaced later, especially with raw or nearly raw specimens. There also was initially thought to be a difference in symptoms dependant on the various substrates. It seemed that *Laetiporus “sulphureus”* growing on eucalyptus more frequently caused symptoms, but that thesis was later in question as more cases produced by *Laetiporus “sulphureus”* growing on oak and conifers were reported. It remains possible that *Laetiporus gilbertsonii* more often causes symptoms than *Laetiporus conifericola*.



*Laetiporus gilbertsonii* photo © Fred Stevens



About the same time, members of MSSF also recorded two rare, but dramatic and presumably idiosyncratic reactions to chanterelles. These two initial cases were similar and unusual in that nausea and short-lived vomiting began almost immediately after ingestion. The first victim vomited after a small plate of European chanterelles (*Cantharellus cibarius*). This unpleasant experience was repeated when he ate a supposedly mushroom-free soup in Switzerland. The cook admitted that a small amount of chanterelles had been added to the soup for extra flavor. The second poisoning was similar, but occurred in Western North America.

Most of the chanterelles along the northern coast are *Cantharellus formosus* (not the European *Cantharellus cibarius*) and *Cantharellus cibarius* var. *roseocanus*.<sup>189</sup> There are a number of unnamed species in Western North America, including the *Cantharellus* most common in the San Francisco Bay area. There appear to be at least five chanterelle species in California and more in Oregon, Washington and Idaho.<sup>189a</sup> These chanterelles usually fruit in moist areas, but may be found in dry areas as well occasionally. When this occurs, they are usually found later in the year. In the dry San Joaquin Valley east of the coastal mountains in California, they sometimes fruit in 3-4 year cycles.<sup>190</sup>

*Armillariella mellea*, the honey mushroom, which produces occasional gastrointestinal symptoms both in North America and in Europe, is now known to be several species—of which the most common in California is *Armillariella ostoyae* with a light orangish tan to medium brown cap. *Armillariella gallica*, which has a smooth cap and more bulbous base is more common from the PNW to Colorado, than it is in California. There is some uncertainty as to whether occasional GI toxicity is more related to cooking or individual sensitivity to one or both of these *Armillariella* species. Many Europeans blanch *Armillariella mellea* in boiling water before eating or pickling.



*Armillariella mellea* photo © Michael Wood

Another genus that may cause GI upsets is *Leccinum*, a member of the family *Boletaceae* (boletes) that has scaly stipes and is most commonly found under birch. Although most cases of reported *Leccinum* toxicity come from Colorado, this fact is likely an artifact of active reporting and the fact that Colorado has so much *Leccinum* in association with birch. Most problems appear to be with poorly cooked or raw *Leccinum*. Dr. Dennis Desjardin and Marilyn Shaw believe that, as in the blue-staining *Boletus*, there may be a GI irritant present at very low levels.<sup>91</sup>

Morels should not be eaten raw, but they are occasionally served that way in salads. The first report came from Canada and the morels were eaten with alcohol.<sup>191</sup> Gastrointestinal symptoms occurred in 77 out of 483 persons at a Vancouver Banquet.<sup>82</sup>

*Leucoagaricus leucothites* (*Lepiota naucina*), a white-capped mushroom with white to faintly pinkish gills is a good edible. Some field guides refer to a grey



“variant” that may cause gastrointestinal upset. This mushroom is actually a separate species, *Leucoagaricus barsii* (= *L. pinguipes*), and it causes occasional GI distress.

## Mushrooms known for their GI toxins or irritants

It is often difficult to distinguish GI toxins and irritants from various intolerances. An example of intolerance is *Agaricus bisporus* and trehalose malabsorption. Young buttons of *Agaricus bisporus* have more of the complex carbohydrate trehalose. Some people lack the enzyme trehalase to break down this carbohydrate to sugar.<sup>192</sup> The result is diarrhea without vomiting. The lack of trehalase sometimes runs in families.<sup>193</sup>

Other authors have distinguished GI irritants that work locally in the gut (eg. the phenolic compounds in some yellowing species of *Agaricus*) from GI toxins that work deeper on peripheral nerve receptors or activate central nervous system centers. The CNS vomiting center, for example, may be activated by the terpenes in the genus *Omphalotus*. The toxins of *Chlorophyllum molybdites* are unknown, but may produce its symptoms by both GI irritation and CNS toxicity.

It has already been noted that raw mushrooms are generally more toxic and some genera should never be eaten raw. Even with "safe" raw edibles, it is best to eat only small amounts uncooked. Relatively safe raw mushrooms include *Agaricus bisporus* and *Agaricus campestris* along with *Boletus edulis*, *Boletus aereus* and *Boletus barrowsii*. As noted previously, boletes have tubes instead of gills on the undersurface of the caps. The highly sought-after edulis group (*Boletus edulis*, *Boletus aereus*, *Boletus barrowsii* etc.) is particularly safe. When these boletes are immature, the pore-like mouths of their tubes are closed by a whitish flap.

Ingestion of *Agaricus californicus* instead of *Agaricus campestris* produces GI symptoms in many, but not all persons, suggesting personal idiosyncrasy. Back in the 1960's, the author collected a few “odd *Agaricus campestris*” on a nearby golf course and had some nausea. They were less felty and more reflective of light, so only a few were eaten. We now recognize these specimens as the often GI symptomatic: *Agaricus californicus*. This noxious domestic weed is now more common than *Agaricus campestris* in many California areas. It may also invade groups or fairy rings of *Agaricus campestris* on lawns. Peck described *Agaricus californicus* in 1895 from a Pasadena, California collection, but it was not a wide spread nuisance until the last half of the 20th century.<sup>194</sup>



*Agaricus californicus* photo © Fred Stevens

The anatomical differences between *Agaricus campestris* and *Agaricus californicus* are relatively subtle. The *Agaricus campestris* cap feels and looks more felty to the touch, the ring is exceedingly thin and without definite patches of a secondary veil on the undersurface of the ring. The partial veil tears in a stellate fashion like thin tissue paper and the cap does not turn yellow with 2.5% KOH. *Agaricus campestris* has a pleasant mushroomy odor and the young gills are pink from the start.

*Agaricus californicus* frequently has a silky sheen, a more fibrillose cap and the partial veil and ring are much more substantial, the ring having small patches of a double veil on the underside. There usually is a faint phenolic odor to *Agaricus californicus*, but it may be slight and some people seem unable to smell it. Current disinfectants no longer smell of phenol and the exact odor is best taught by smelling certain other *Agaricus* species. Crushing and smelling the base of *Agaricus* (“meleagris”) *praeclaresquamosus* provides a good example. Sometimes there is very slight yellowing of the carpophore with bruising, but this is not dependable. The gills are whitish in small buttons.



Other gilled fungi commonly producing GI symptoms include other members of the *Agaricus* group, *Xanthodermati*: *Agaricus hondensis*, *Agaricus placomyces* var. *microsporus*, *Agaricus praeclaresquamosis* and *Agaricus xanthodermus*. These latter *Agaricus* species are easily distinguished by their yellow staining and unpleasant phenolic odors. Sometimes members of another *Agaricus* group cause trouble—primarily *Agaricus albolutescens* and *Agaricus silvicola*.



*Agaricus xanthodermus* photo © Michael Wood

Although *Clitocybe (Lepista) inversa* is listed in most texts as edible, what appears to be the same mushroom has caused poisonings in France.<sup>195</sup> One member of the MSSF has had mild nausea and moderately severe diarrhea from its ingestion on two occasions.

Other offenders are species of *Gymnopilus*, *Hypholoma* and *Hebeloma* (especially *Hebeloma crustiliniforme*, *H. fastibile*, *H. mesophaeum* and *H. sinapizans*); very acrid species of *Lactarius* and some species of *Lactarius* that stain lilac (especially *Lactarius glaucescens*).<sup>196</sup> The first *Hebeloma crustiliniforme* poisoning reported from North America was in 1927.<sup>197</sup> This mushroom is known as "poison pie" in Europe, because of its intense diarrhea. Many *Hebeloma*

species have a radish-like odor; that and brown gills on maturity should cause any such to be bypassed for culinary use.



*Hebeloma crustuliniforme* photo © Michael Wood

Occasionally, the *Russula laurocerasi* group, *Pholiota* species and *Phaeolepiota aurea* produce GI symptoms. *Megacollybia (Tricholomopsis) platyphylla* and a number of tricholomas: *Tricholoma pardinum*, *T. pessundatum*, *T. saponaceum*, *T. sejunctum*, *T. sulphureum*, *T. vaccinum*, *T. zelleri* and possibly *T. virgatum* are others that often cause GI upsets. Of these, the most vicious offender is *Tricholoma pardinum*, which may cause prolonged vomiting and diarrhea.

Collectors of coccoli (*Amanita lanei*) may mistakenly gather a mushroom in the spring that has even brighter yellow to orange caps—the toxic *Amanita aprica* J. Lindgr. & Tulloss. A frost-like layer of universal tissue tends to persist on the caps. This amanita usually occurs on open banks or along road cuts or paths in Douglas Fir and pine forests at elevations from 600-1850 meters. It is a typical species of the Cascade and Sierra Nevada mountains. This beautiful *Amanita* produces nausea, vomiting, diarrhea, intestinal cramps and muscle spasms.



*Cortinarius* species are rarely collected for the table in Western North America. There have been no definite *Cortinarius orrelanoides* poisonings at the time of this publication. Raw *Cortinarius* species (*C. camphoratus* & *C. purpurascens*) have been reported in the West to cause gastrointestinal problems.<sup>122</sup> There seem to be no *Dermocybe* poisonings reported from our area of concern, although some species of this genus (which Moser segregates from *Cortinarius*) apparently contain anthroquinones which act as GI irritants.<sup>126</sup>

The acrid species of *Russula* and *Lactarius* have GI irritants, whereas the non-acrid species are often edible. *Russula emetica* (an acrid species native to European sphagnum bogs) is replaced in North America by *Russula silvicola*. *Russula sanguinea*, equally acrid, has both a red cap and a stipe flushed rosy to reddish. *Russula nigricans* in a 1985 Oregon poisoning produced not only GI symptoms, but also dizziness and disorientation.<sup>198</sup>

*Entoloma* species often produce severe GI toxicity. All of them should be avoided; data on American species is very limited. The spores are pink in mass. Under the microscope, these spores are angular (frequently 5-6 sided); rarely long and longitudinally ridged. The PNW has the toxic *Entoloma grande*.<sup>199</sup> Several forms of the toxic *Entoloma rhodopolium* are present in California. Other forms are found in the Pacific Northwest. *Entoloma rhodopolium* has occasionally also produced muscarinic symptoms. *Entoloma lividoalbum* is considered toxic in Colorado.

*Tricholoma pardinum* poisoning often causes such severe vomiting and diarrhea that hospitalization is needed. Sometimes other *Tricholoma* species with gray, black or reddish-brown caps may cause lesser gastrointestinal distress. *Tricholomas* with orange or reddish caps found grouped under various trees need to be carefully examined. Some of these, such as *Tricholoma populinum*, usually found in sandy soil under cottonwood trees are good edibles. However, they may be confused with members of the poisonous *Tricholoma pessundatum* group: *Tricholoma pessundatum*, *T. manzanitae*, *T. albobrunneum*, *T. ustaloides*, *T. flavobrunneum*, *T. acerbum* and *T. ustale*. Any of these mushrooms may cause quite severe gastrointestinal symptoms.

*Chlorophyllum molybdites* is usually found on lawns or in grassy areas (often in huge fairy rings). This large and inviting mushroom has green spores, although buff spores have been rarely seen. Although *Chlorophyllum molybdites* has been reported safe to eat after boiling, apparently well-cooked meals occasionally cause nausea and vomiting. More than one toxin may be present. A 2-year-old child died after eating *Chlorophyllum molybdites* in 1900, apparently becoming

dehydrated.<sup>202</sup> As noted in XII Miscellaneous Toxins, there have been a few reports of hemorrhage, when this mushroom had been eaten raw. One possible toxin has been reported to be a polymeric protein.<sup>203</sup>

*Sceleroderma* species, unlike puffballs, usually have shades of black, brown or dark purplish centers even in the early stages of development and, additionally, they have a thick tough outer layer. *Scleroderma citrinum* and *Scleroderma cepa* may cause very violent vomiting, diarrhea and prostration. *Scleroderma cepa* has a nearly white interior. A Tacoma, Washington, woman in 1978 collected what she thought were puffballs, since two of the smaller ones were pure white inside.<sup>204</sup> Sauteéd in butter, they were delicious, but 1½ hours later she suffered vomiting, profuse sweating, prostration and two brief episodes of loss of consciousness. A half hour later, she was nearly back to normal. Dr. Beug at the Evergreen State College noted a small amount of purple in one specimen. Dr. David Hosford at Central Washington State University identified the specimen as *Scleroderma areolatum*. Dr. Beug then advised that, in addition to a white center for a field description of a puffball, one should add soft and with a texture resembling a marshmallow. Young *Scleroderma* specimens may also have white centers, but the tissue is then tough or hard.



*Scleroderma cepa* photo © Fred Stevens

*Morchella* species, especially the black specimens, occasionally cause nausea, vomiting and diarrhea. Additional symptoms of fever, headache, sweating and weakness are sometimes reported.<sup>204a</sup>

Although *Gomphus floccosus*, *G. bonari* and *G. kauffmanii* are occasionally eaten without problem, they may cause GI symptoms. Symptoms may occur as late as 8-14 hours after the meal, a latency period which is startlingly different from other GI toxins. At least one toxin, norcaperatic acid, has been detected. Fortunately, these ice-cream cone shaped fungi are easy to identify. Dr. Alexander Smith (who got hay fever-like symptoms from many fungi) reported for *Gomphus floccosus* that “I have tried it on three different occasions and always experienced considerable (GI) discomfort within eight to fourteen hours”.<sup>205</sup>

*Sarcosphaera coronaria* is a common montane species that occurs in the spring as an inhabitant of forest duff. Starting out below ground as a hollow sphere with a beautiful lilac tone to its interior, it soon penetrates the duff splitting from the top down as a crown-like cup. Eaten raw, it produces severe vomiting and

diarrhea. Although said to be a good edible when thoroughly cooked, this mushroom is best avoided.

Orange or red-pored boletes often cause GI symptoms. Muscarine may also be present in a few. Collectors should generally avoid these boletes in Section *Boletus* and *Subsection Luridi*, although *Boletus erythropus* is edible and very tasty. All of the following have produced GI symptoms: *Boletus amygdalinus*, *B. haematinus*, *B. luridus*, *B. satanas* and *B. pulcherrimus*. *Boletus luridus* also interacts with alcohol.<sup>206</sup> The general advice to avoid all boletes that stain blue regardless of pore mouth coloring proscribes the ingestion of some boletes that are quite tasty in the West such as *Boletus appendiculatus*. It remains best to avoid those that have orange or red tube mouths that stain blue or are bitter when tasted raw.



## Group XI toxins: Miscellaena

### Miscellaneous toxins

*Auricularia polytricha*, a flattened ear-shaped mushroom used in Chinese cooking, produces an anticoagulant effect. In 1980, a U.S. case of “Szechwan Purpura” was reported in the New England Journal of Medicine. A 32-year-old U.S. researcher (during an experiment using his own blood platelets for clotting) found after some 50 trials one morning that his platelets would not agglutinate or release serotonin on exposure to epinephrine.<sup>211</sup> On physical examination, he had a few small bruises on the legs. Tests, not requiring platelets, were normal. He and his co-workers were able to trace the aspirin-like effect back to Mo-er (the Mandarin name for *Auricularia polytricha*). This mushroom inhibited platelet response in the researcher and other volunteers for 3-24 days. Like aspirin (another inhibitor of platelets), this mushroom should not be ingested prior to surgery or by patients with poor blood clotting.



*Auricularia polytricha* photo © Tom Duffy

Western physicians thus became aware that *Auricularia polytricha* inhibits the tiny blood platelets that initiate blood clotting in the small vessels and capillaries of the body. This mushroom is a dark rubbery, irregular ear-like fungus known in Asian markets as “wood ears”. This mushroom is especially common on trees along canals and waterways in the damp, fertile province of Szechwan. Like the discovery of penicillin, it occurred in the course of a different investigation.

Cookbooks commonly confuse the fleshier *Auricularia polytricha* with the smaller and thinner *Auricularia auricularia*, which has no effect on platelets and which Cantonese cooking uses in its contrast of textures. The more spicily sauced cuisines of Szechwan and Hunan use the thicker *Auricularia polytricha* more for soaking up their richer sauces than for texture.

The red-pored *Boletus pulcherrimus* Thiers and Halling [formerly *Boletus eastwoodiae* (Murril) Saccardo and Trotter] ordinarily produces only gastrointestinal symptoms, but may have additional toxicity. An Oregon husband and wife began with typical gastrointestinal symptoms in a 1974 poisoning. The husband, however, died suddenly and his autopsy showed small bowel death from lack of blood supply.<sup>3</sup> There was either clotting of vessels leading to the mid-gut or a lack of blood supply due to a drop of blood pressure. Causes could have been dehydration, a narrow artery to the small bowel or a direct toxic effect on blood pressure or on blood clotting.



*Boletus pulcherrimus* photo © Fred Stevens

*Suillus* species have generally not caused any symptoms, other than occasional gastrointestinal ones, in North America. However, there have been a few reports of other toxicity, including one from Poland, of hemolysis.<sup>207</sup>

*Boletus amygdalinus*: The San Francisco Poison Control Center reported in 1998 that at least two persons of Russian extraction, seen in a San Francisco Bay Area emergency room for mushroom poisoning, were subsequently hospitalized with markedly abnormal liver tests. The Poison Control Center asked two of the MSSF member to examine the boletes they had collected. One of the sons brought the remaining boletes to the Webmaster of MykoWeb.com, Michael Wood, and to a former pupil of Dr. Harry Thiers, Fred Stevens. The specimens were readily identified as *Boletus amygdalinus* (Thiers) Thiers a species indigenous to central California. The MSSF's Toxicology Committee did not examine the actual hospital records. The identification, however, was solid and the specimen showed the pip-shaped spores as well as other characteristic features.<sup>208a</sup> **Any bolete causing liver or kidney damage should be reported in as much detail as**



possible; boletes have always been considered relatively safe for amateur collecting, causing at worst only gastrointestinal misery.



*Boletus amygdalinus* photo © Michael Wood

Two *Clitocybe* species with high acromelic acid levels are known to produce a bizarre acromelalgia. A *Clitocybe* in France (*Clitocybe amoenolens*) has caused swelling, reddening and severe pain in the limbs. *Clitocybe acromelalga* in Japan produces the same syndrome. The toxins (acromelic acids) were the same. Such a poisoning may eventually be found here, possibly in one of the brown clitocybes.<sup>214</sup> The pain produced by these fungi responded poorly to opiates. However, neuralgic pain often responds to higher doses of narcotics, to antidepressant drugs (such as nortryptine) and to some of the anticonvulsant medications (neurontin, lamictal, mexiletine and tegretol).

*Morchella* and *Gyromitra/Helvella*. See rhabdomyolysis in *Psilocybe cubensis* below.



*Phallus impudicus* poisoning was reported to produce severe allergic symptoms in two patients. One lady had dramatic reddening of her hands followed by hives over her entire body with this "stink horn".<sup>91</sup> The common name refers to the unpleasant odor which is so distinctive and strong in the mushroom that one is thereafter able to smell the fungal mycelia in the ground, even in the absence of the fruiting body.

*Psilocybe cubensis*. This psychoactive (and often cultivated) mushroom surprisingly caused rhabdomyolysis in one poisoning. The NAMA national data base maintained by Dr. Kenneth Cochran recorded that case and additionally two more cases of muscle break-down following mixed meals of morels and false morels (*Morchella* and *Gyromitra/Helvella*).<sup>212</sup>

*Ptychoverpa bohemica* (Kromb.) Boud. was previously placed in the genus *Verpa*. This mushroom has produced GI symptoms at times, but the most remarkable poisonings have produced decreased muscular coordination, even to the point of being unable to write.<sup>209,210</sup> One theory is that small amounts of Gyromitrin are present. However, this conception is inconsistent with Gyromitrin's threshold effect. What occurs with remarkable consistency, however, is a predominant CNS symptom of incoordination suggesting a novel toxin. The Thiers Herbarium in San Francisco contains only two collections of *Ptychoverpa bohemica*. This species is more common in the PNW and in the East.

*Tricholoma flavovirens*, a choice edible in the U.S., has caused rhabdomyolysis in twelve pot-hunters in France who had eaten large amounts at three or more meals. The victims developed some muscle symptoms at 24-72 hours with pain and weakness. The group of 12 pot-hunters delayed coming in for medical treatment and also had dark urine and severe sweating.<sup>213,214</sup>

## Addendums, Glossary, & References

### Addendum 1: Scientific names and the conventions used in the text

[After this section, there is an addendum by Dr. Rodham Tulloss, it being felt that my original material on this complicated issue needed further clarification and expansion. This section by Dr. Tulloss is labeled “Addendum 2”. The tenor of that section is clarification of the relationship between fungal taxonomy and fungal names, using specific examples in the genus, *Amanita*.] Errors, if any, are those of TJ Duffy since that material has been modified to follow this addendum.

Genus names and species names (epithets) are italicized, the genus name being capitalized with the following species name being in lower case.

Terms, which follow the italicized species epithet, identify the person or persons responsible for that name according to the specifications of *The International Congresses of Botanical Nomenclature*. The study of these specifications for a particular species epithet constitutes taxonomy in the strict sense. The word taxonomy is also employed in the less strict sense of correctly determining what names should be applied to particular fungi. This is the sense in which Dr. Rolf Singer uses the term in his *The Agaricales in Modern Taxonomy*.

The “type” collection is the defining one for that species or any subspecies, variety or form. Without a note to the contrary this is the holotype or original collection on which the author based the name. If the original material has been lost, heavily depleted or damaged, another type may be used or designated e.g. an isotype (a duplicate or part of the type collection other than the holotype) or e.g. a neotype (a new collection carefully made to duplicate the original material and newly designated as that on which the name is to be based).

Few fungi have common names. The same common name may be used for *different* species and more than one common name may apply to the *same* species. Thus there was a need for a more rigid designation following the concepts of Linnaeus, the father of systematic botany. In his Latin binomial or two-name system, each species is designated by both a genus name and a species name, the latter referred to as a species “epithet”. The genus name is Greek or Latin in derivation or form; the species epithet is usually Latin (or a latinized name, e.g. species named after various botanists).

*The International Congresses of Botanical Nomenclature* regulate scientific naming for living, but non-animal species. The capitalized genus name comes first followed by the uncapitalized species epithet. The citation *Chroogomphus vinicolor* (Peck.) O.K.Mill., for example, recognizes the fact that Charles Peck, the father of American mycology, named the species "*vinicolor*" in the genus *Gomphus* and that Orson K. Miller later transferred it to his new genus *Chroogomphus*. Mushroom names have been much more unstable than green plant names, primarily because fungi have many characteristics that are not macroscopic in nature, but still need to be considered in defining a species.

Two names within a parenthesis denote earlier authors of name and validation of that name for species later accepted by the definitive author of that genus. For most of the higher fungi, the validating author is Elias Fries—the founder of modern mycology. Names assigned by authors prior to official validation in the botanical code are followed by “ex” or “per”, which expressions are used in the sense of “was validated by”. Thus *Amanita muscaria* (L. ex Fr.) Pers. is an example of a species named by Linnaeus, that name validated by Fries and the species then moved from the genus *Agaricus* to the genus *Amanita* by Persoon. Names for species that have been validated by publication in Elias Fries’ “Systema Mycologicum” may use a colon instead of ex or per. This more concise notation for Fries’ validations will be used in the text below e.g.—*Amanita muscaria* (L.:Fr.) Pers. or *Gyromitra infula* Schaeff.:Fr.) Quélet.

Names frequently used, especially earlier authors or those with longer names are often abbreviated, such as Fr. for Elias Fries and Quélet. for Lucien Quélet. A standard list is that of Kirk and Ansell.<sup>215</sup>

Citations are left out of the text, except where there are new names or possible ambiguity. In such a case (and for all amanitas), at least the first mention of the species will include the citation. "Sensu" means in the sense of a particular mycologist and "sensu auct. amer." in the sense of American authors. The term “comb. nov.” refers to a new combination made by joining the names of two species in a different way. The example in this text is *Clitocybe dealbata* (Fries) Kummer ssp. *sudorifica* (Peck) Bigelow, comb. nov. Howard Bigelow in his North American Species of *Clitocybe* notes that the North American species studied by Peck and Murrill is so closely related to the European interpretations of *Clitocybe dealbata*, that it is best described at the subspecies level, hence the new combination. "Sp. affin." refers to a closely related species and "nom. prov." refers to a proposed species name not yet validly published. In addition to “sp.” for species, the abbreviation “ssp” is used to represent a subspecies. A species’ name

in parentheses denotes, in this text, a former (and often, a more familiar) name that may be found in some texts.

A “-----” around a species epithet in the text indicates that the species may not be identical to the species generally assumed to be correctly named (often an eastern or European name) or that the “species” may really be a complex of closely related ones. When a word in parenthesis begins with a capital letter and occurs in the middle of a scientific name, such as *Hygrophoropsis (Clitocybe) aurantiaca*, the **genus** in parentheses is not the name considered correct in the taxonomic sources used by the author. The “(*calyptroderma*)” in “*Amanita (calyptroderma) lanei*” begins with a lower case letter and refers to a former **species** epithet not considered correct in the author’s current taxonomic sources.

Citations for *Amanita* species are those used by Dr. Rodham Tulloss. Other citations are those used in standard reference works on toxic fungi. A number of these citations are incorrect (especially among the older taxa), partly because of persistent tinkering by the ICBN in recent decades and partly because many mycologists were more interested in taxonomy specifically as the naming of described species rather than untangling the needed nomenclature as dictated by the ICBN.

Historical reasons have given rise to peculiar citation problems. The citation for the highly toxic *Amanita phalloides* is a good example. Dr. Singer gives this species the scientific name of *Amanita phalloides* (Vaill. ex Fr.) Secretan. However, Vaillant himself did not use a Latin name, but instead used the French descriptive phrase “Amanite phalloïde”. The result was that when Fries re-described the species as a binomial on the basis of Vaillant’s work, the re-described species name became the earliest legitimate one and also, under the code, its formal validation. Link was the first author to transfer this species to the genus *Amanita* (from *Venenarius*), so that the correct latinized name is *Amanita phalloides* (Fr.:Fr.) Link.

The most difficult problems are with the oldest names. In the case of *Amanita verna* (Bull.:Fr.) Lam. (the European springtime “Destroying Angel”), the holotype no longer exists. Dr. Tulloss notes that in the recent informal study by Francis Massart, his colleagues and correspondents, all specimens collected in Europe as *Amanita verna* gave a yellow-orange stain with KOH despite that taxon’s original designation as a species negative to KOH. The suggestion has been made that the original holotype may have contained at least some *Amanita phalloides* var. *alba* Britzlem. Sidestepping the issue of whether there exists a correctly named *Amanita verna* in Europe, the species with a yellow-orange stain



on KOH testing has been designated *Amanita decipiens* (Trimbach) Andary & Bon.

## **Addendum 2: Fungal nomenclature, fungal taxonomy, and effect on fungal names**

**by Rodham Tulloss, PhD.**

(This section has been modified by the author of Toxic Fungi of Western North America, Tom Duffy, to follow and flesh out the prior discussion of taxonomy. Any errors should be attributed to him.)

There are two complementary forces that end up producing a “currently correct name” in the life sciences in general. They play this role in mycology. One is nomenclature, which includes a set of rules governing the formation and publication of names in a way that is deemed valid by common agreement (the International Code of Botanical Nomenclature). The other is taxonomy as the study of the fungi in nature that bear (or eventually will bear) a valid name.

Nomenclature governs issues such as the set of rules for publishing a new name, the conditions under which a name may or may not be changed, the conditions under which a name can be changed in rank (a variety made into a species, for example), etc.

Taxonomy reveals the meaning behind a name and, consequently, causes names to disappear when two different names are found to belong to a single taxon.

Few fungi have common names and these names vary disconcertingly from area to area. The same common name may be used for different species (for example, in Nepal, the words meaning “devil’s apple” are applied both to a plant and a fungus) and more than one common name can be applied to a single species (for example, Jack-in-the-Pulpit and Lords-and-Ladies). Thus, for scientific communication purposes, there is a need for a more rigid designation. In the binomial system by the father of systematic botany, Linnaeus, each species is designated by two names. The first name is the name of the genus in the form of a noun and is always capitalized. The second name is called the species epithet and is in the form of an uncapitalized adjective or an uncapitalized noun serving as an adjective. Originally the names were largely limited to Latin and to Greek; however, currently pronounceable acronyms and even nonsense terms are permitted for epithets. There are a number of examples of epithets taken from Maori or aboriginal Australia.

Names get their meaning (even if it isn't detailed at the time of naming) by having a type designated. For a species or a rank below species (such as variety or form), the type collection must be a preserved specimen. Methods are provided for replacing types if lost or destroyed or if they never existed (a problem with some old names). For a genus, the type is a species in that genus.

Some of the history of a name is preserved in the authorial citation that follows the name (usually the first time the name appears in a work).

When a newly described mushroom is published, the author(s) publishing the name get his/her/their names appended to the name of the newly recognized entity. An example that we can track through various times in its history is *Amanita calyptрата* Peck. Peck deposited a specimen from Oregon in the herbarium of what is now the New York State Museum. Let's just call that "Peck's type."

Nomenclature leads to name changes if library research leads to an old, forgotten publication in which a name was used earlier than previously thought. For example, *Agaricus citrinus* Schaeff. was made invalid when an old Norwegian journal was found in which the combination *Agaricus citrinus* had been made by another author several years earlier than Schaeffer's publication. This made "*citrinus*" unavailable or occupied at species rank. This led to the present day question of whether we should follow the core rules and stop using "*Amanita citrina* (Schaeff.) Pers." or use the "rule for breaking the rule" and have a vote to conserve Schaeffer's "*Agaricus citrinus* (and its subsequent "*citrinus*" transfer to the genus, *Amanita*).

To continue the *calyptрата* story...Several decades after Peck named *Amanita calyptрата*, W.A. Murrill, who followed the then existing American Code of Nomenclature, found that Lamarck had already used the combination *Amanita calyptрата* late in the eighteenth century. Following the rules of the time in America, Murrill changed Peck's name. He recognized *Venenarius* as the oldest equivalent of *Amanita* and he picked *lanei* as his new species epithet. Peck's type was still the type. So the equivalence of Murrill's name to Peck's name is guaranteed by definition. This kind of synonymy is called "nomenclatural synonymy". Some authors indicate this by the 3 barred equivalence symbol ( $\equiv$ ). Soon thereafter, Saccardo and Trotter, who did not recognize the American Code, changed the genus name of *Venenarius* to *Amanita*. This change gets recorded in the authorial citation as follows: *Amanita lanei* (Murrill) Sacc. & Trott.

Changes in the accepted code and within that code also lead to name changes that were not planned in advance, but are required by a newly approved version of the code. When a group of works of E.M. Fries were collectively assigned as the starting point of international mycological nomenclature, Lamarck's "*Amanita calyptrata*" was made irrelevant because he published decades before the Friesian "starting point." Back came "*Amanita calyptrata*" Peck as the correct name defined by Peck's type.

When the starting point for fungi was moved back again to the canonical publication of Linnaeus, Lamarck became relevant again! Now we are caught up to the present day and the correct name of Peck's type is, once again, *Amanita lanei* (Murrill) Sacc & Trott. Notice that all the changes in the correct name to be applied to Peck's type had little to do with what the type was, what it looked like, what size or shape the spores had, etc. The changes all had to do with rules, changing between sets of rules, and changes to the rules themselves. All the names generated so far in our story are nomenclatural synonyms.

Now, let's see how taxonomy plays its role. About the time that Murrill was following his American rules and creating *Venenarius lanei*, Ballen collected some large edible amanitas in California and posted them off with notes and photos to G.F. Atkinson at Cornell. Atkinson could not recognize the characteristics listed by Ballen as being those of anything previously named. In the fullness of time, there appeared a description of a proposed new species with a new name—*Amanita calyptroderma* G.F. Atkinson & Ballen. Decades went by before D.T. Jenkins looked at the Atkinson and Ballen type and Peck's type, examined them, and proposed they were the same species. Those who have followed Jenkins, treat the name *Amanita calyptroderma* as an additional synonym to those we've met so far in this story. This time, however, the synonymy has everything to do with the study of the two types in detail. This time it is taxonomic synonymy. When there is a case of taxonomic synonymy, the older of the two names is the correct name. So here is a case of a name (*Amanita calyptroderma*) that is perfectly valid by nomenclatural rules alone, but succumbs to a physical review of the specimens that define two names.

Taxonomy can also lead to new names in other ways. The recognition of a taxonomic need for a new genus (say, *Chroogomphus* O.K. Mill.) will require the making of new combinations. Since Miller split *Chroogomphus* from *Gomphus*, species such as *Gomphus vinicolor* Peck became *Chroogomphus vinicolor* (Peck) O.K. Mill. This procedure is not automatic. Each name requires separate recombination. In our previous tale, *Venenarius* and *Amanita* were exchanged back and forth for purely nomenclatural reasons. In the present case, the



originating motivation is taxonomic. However, since the type of *Gomphus vinicolor* Peck is also the type of *Chroogomphus vinicolor* (Peck) O.K. Mill., the two are nomenclatural synonyms.

Special abbreviations and other notations used in names and author citations are also worth understanding. Most readers of field guides are familiar with some abbreviations—those for species (sp. for the singular and spp. for the plural), the ranks below species (the infraspecific ranks): subspecies (subsp. or ssp.), variety (var.), and form (f). When someone proposes to change the rank of a species to a variety or a variety to a form, then this is another example of recombination. The author of the latest combination is placed to the right of parentheses as in the change of names of species in our earlier examples: *Amanita pantherina* var. *pantherinoides* (Murrill) Dav. T. Jenkins is a taxonomically motivated nomenclatural synonym of *Amanita pantherinoides* (Murrill) Murrill. [Murrill didn't like having Saccardo and Trotter move all his species in *Venenarius* to *Amanita*, so he formed the habit of giving both names when he named a new species. *Venenarius* was the genus of choice for Murrill, but (for example, on the last page of an article) Murrill would say “for those who follow Saccardo” and list all his *Venenarius* species recombined as *Amanita*. [Thus Murrill achieved instantaneous name change, the fastest nomenclatural species sixgun in the West.]

When a name is being applied without certainty (a choice of the author), certain abbreviations may be inserted between the genus name and the species name. These follow idiosyncratic conventions (if any). These abbreviations can all be read as “close to (in my opinion, at least for the moment)”: ca., cf., affin.

There are also abbreviations that are sometimes seen embedded in authorial citations. Some have very specific uses associated with one or a very few authors, such as the colon preceding “Fr.” (meaning E.M. Fries). The name *Amanita muscaria* (L.:Fr.) Lam. is an example of a sanctioned name. Names sanctioned by Fries are accepted no matter whether or not there is an older one for the same mushroom. This is a very special kind of nomenclatural provision. In the example, Linnaeus created the name *Agaricus muscarius* in his canonical, starting point work. Fries used the name (accepted it) in his set of books that are no longer the starting point for fungal names, but are the sanctioning works for agarics. Lamarck was the first known person to make the combination *Amanita muscaria*. This sanctioning applies to the epithet and not to the genus.

## Glossary

**Acetaldehyde**—step in the metabolism of alcohol.

**Acetylcholine**—quaternary amine and neurotransmitter. See amine below.

**Adnate**—referring to a very blunt attachment of the gills to the stipe or to an attachment that is about an average gill width.

**Adnexed**—referring to a thin attachment of a gill where the gill edge at that point is narrowed but the attachment is still at right angles to the stipe, not sinuate.

**Agonist**—a chemical substance capable of combining with a receptor site on the surface of a cell and initiating a reaction within the cell. See receptor site for lock and key concept.

**Alkaloid**—a broad class of amines found mainly in plants, including fungi.

**Amberlite XAD**—an ion exchange resin that removes amines from solution and permits their recovery.

**Amino acid**—building blocks of peptides. See peptides, which includes proteins.

**Amide**—carbon to nitrogen bond in peptide.

**Amine**—an organic nitrogen compound capable of forming salts with acids. (see salt).

**Amyloid**—blue, gray or black in iodine solutions such as Melzer's solution.

**Anaphylactic shock**—an extreme allergic reaction using a different pathway than minor allergies such as "hay fever". The immediate shock (loss of life-sustainable blood pressure) occurs within seconds or minutes. The most common deaths from anaphylactic shock are from bee stings and food such as eggs (but not or exceeding rare with fungi).

**Angular**—4-7 sided cells, usually referring to spores of the Entolomaceae.

**Annulus**—ring on the stipe; from the partial veil originally from the cap edge to the stipe and covering the gills.

**Annular zone**—remains of a ring; usually a roughened area which is noted by catching colored, often brownish or blackish spores, drifting onto the stipe.

**Antagonist**—see inhibitor.

**Antibody**—shape sensitive protein that recognizes and destroys specific foreign cellular components.

**Antigen**—substance that triggers an immune system cell to produce antibodies.

**Arcuate**—forming an arch of gills, lower edge of the middle of the gills are higher than the lower edge of the ends of the gills.

**Ataxia**—wobbling or unsteady gait due to nervous system dysfunction.

**Autoantibodies**—see below.

**Autoimmune**—a state in which higher organisms produce antibodies that are directed against their own tissue antigens.

**Bicyclopeptide**—See peptide.

**Bicyclic**—an organic chemical containing two rings.

**Broadly attached**—said of gill attachment at the stipe where the attachment is slightly decurrent, adnate or adnexed with a wide attachment at the stipe. See adnate, adnexed, decurrent.

**Catalyst**—substance that can increase the rate of a chemical reaction without being consumed itself.

**Chromatography**—separation of a mixture by adsorption on insoluble particles by a moving solvent.

**Central nervous system ("CNS")**—that portion of the brain and spinal cord within the upper skull (cranium).

**Confidence level**—statistical estimation of population means from random sample means. The 95th confidence level (roughly 2 standard deviations from the population mean) is most commonly employed; such a statistical finding is likely to be correct 5% of the time at that confidence level, all other considerations being equal.

**Coprine**—derivative of aminocyclopropanol. An acetaldehyde dehydrogenase inhibitor.

**Creatinine**—a final product primarily from the break-down of skeletal muscle creatine. Its measurement changes little in human dehydration and is an excellent measure of kidney function.

**Cyclopeptides**—closed loop derivatives of a peptide (protein fragment).

**Decarboxylation**—loss of carbon dioxide (CO<sub>2</sub>) by the decomposition of an organic acid.

**Decurrent**—gills that run down the stipe or where the attachment at the stipe is wider than the average height of the gill. Short decurrent refers to attachments that run down the stipe, but less than the average height of the gills.

**Deliquescent**—melting to a liquid state; usually referring to the gills and caps of *Coprinus* and some species of the *Bolbitiaceae*.

**Dextrinoid**—tissue or spores staining golden brown to reddish brown in Melzer's or other iodine solution.

**Diolein**—breakdown product of a fatty compound.

**Dopamine**—an amine (3,4-dihydroxy-phenylethylamine), a neurotransmitter found in the brain and also in peripheral nerves. In addition, it is a precursor of noradrenaline and adrenaline.

**DNA**—Desoxyribonucleic acid; the building blocks in the nucleus of the cell that form the double helical structure transmitting the genetic code. The double strands are made of nucleotides (a backbone of alternating sugar and phosphate molecules with a base attached to each sugar molecule), residing in the nucleus of the cell. The bases for DNA, abbreviated A, C, G and T (adenine, cytosine, guanine & thymine), encode proteins. A sequence of bases on the DNA—called a promoter—signals the enzyme RNA polymerase to begin the RNA sequence called “messenger RNA”, which transfers the information of DNA to the protein-building machinery of the cell. See RNA.

**Ectomycorrhizal**—referring to a mutually beneficial relationship between mushrooms and the rootlets of green plants, bushes and trees in which the nutrients of life are exchanged through mycelia branching freely around and



between the outer rootlet cells thus forming a connecting mesh fine rootlets. The mushroom provides phosphorus and other simple chemicals to the plant, which provides the mushroom with the more complicated carbon compounds produced by photosynthesis.

**Endomycorrhizal**—referring to a mutually beneficial relationship between mushrooms and the rootlets of green plants in which the nutrients of life are exchanged through mycelia branching freely around and into rootlet cells. Some species of *Armillaria* associate in this way with orchids.

**Edema**—swelling of the body, usually of its dependent parts such as the ankles; a manifestation of over-retention of water in the space between the cells.

**Elliptic**—elongated, circular structure. By one criteria with a ratio of length to width of 1.15-1.60. **Elliptoid**—more correctly used for 3-dimensional objects such as spores.

**Enzymes**—protein catalysts that speed up chemical reactions.

**Euphoria**—a rosy feeling of happiness and energy.

**Fasciculations**—small irregular muscle contractions due to the spontaneous contraction of small groups of muscle; usually noted as twitching of bundles of muscle fibers.

**Ferrous/Ferric**—ferrous iron has an oxidation state of two (charge of 2); ferric refers to iron with an oxidation state of three. Ferrous salts such as ferrous chloride are useful in identification of mushrooms, especially in the genus *Russula*.

**Fibrillose**—composed of fine thread-like fibers, longitudinally or radially arranged.

**Fibrous**—composed of tough, string-like tissue.

**Fleshy**—soft and relatively bulky; not tough or cartilaginous.

**Floccose**—with soft, fluffy, cotton-like tufts that readily wipe off a cap.

**GABA**—gamma-aminobutyric acid, a central nervous system inhibitory transmitter. Increases memory in primates by suppressing “noise” or “static” in information processing.

**GI distress or toxicity**—nausea, vomiting, diarrhea or epigastric pain.

**Glutamic acid and glutamate**—an amino acid/salt of that amino acid. (See salt.) Excitatory neurotransmitters in the brain.

**Gyromitrin**—the precursor of hydrazines in the genus *Gyromitra*, in particular monomethylhydrazine (MMH) which is a vitamin B6 compound inhibitor. However, not all of MMH toxicity can be explained by this mechanism.

**Habit**—the characteristic appearance of a mushroom, including its general shape and manner of growth.

**Habitat**—the natural place of growth.

**Haptoglobin**—a globulin protein in the plasma that binds free hemoglobin in plasma; should red blood cell destruction take place, then the lab test values of haptoglobin go down since the haptoglobin is bound; bound haptoglobin is immeasurable with standard tests.

**Heme**—an iron-containing break-down product of hemoglobin.

**Hemoglobinuria**—free hemoglobin in the urine due to the destruction of red blood cells.

**Hemolysis**—destruction of red blood cells with spillage of hemoglobin into the plasma.

**Heterocyclic**—referring to a ring of carbon atoms with a least one other element inserted.

**High pressure chromatography**—rapid chromatography employing an adsorption column under pressure. See chromatography.

**Holotype**—the collection on which the describing author named the given mushroom species.

**Humus**—decaying organic material, often the leaves of deciduous trees.

**Hydrazine**—nitrogen compound with an N-N bond. See gyromitrin and monomethylhydrazine (MMH).

**Hydroxylated**—Adding a water molecule as an –OH building block to a chemical compound; benzene ring compounds oxidized to phenols during detoxification.

**Hygrophanous**—changing color on drying, usually referring to a cap which changes from a water-soaked appearance to a ±opaque appearance.

**Ibotenic acid**—an isoxazole alkaloid that inhibits GABA receptors and activates a glutamate cascade. The name derives from the Japanese "Ibo-Tengu" or long nosed goblin. However, Ibo tengu apparently was applied originally to *Amanita "solitaria"*, not to *Amanita muscaria* or *Amanita pantherina*. The chemical formula is  $\alpha$ -amino-2,3-dihydro-3-oxo-5-isoxazoleacetic acid. Other oxazoles are muscimol and tricholomic acid.

**Immunoassay**—Analysis using a reaction of antigen with an antibody and tracer.

**Immune system**—cells that recognize and destroy specific foreign proteins (or those mistaken for such antigens). See antigen, antibody, autoimmune.

**Inamyloid**—same as non-amyloid (not changing color in iodine solutions such as Melzer's agent. See amyloid, dextrinoid for blue-black or brown colors.)

**Indole**—heterocyclic skeleton of the amino acid tryptophan.

**Inhibitor (antagonist)**—a molecule that blocks the reaction of a neurotransmitter or enzyme.

**Interaction of toxins and drugs**—more than a simple additive effect, one toxin or drug *increases* the effect of the other. A drug or toxin, for example, may increase the effect of a mushroom toxin above that expected by simply adding the two effects together.

**Isomer**—a compound having the same molecular weight and the same chemical structure except for 3-dimensional differences. When shown on ordinary 2-dimensional paper, a backwards branch (from the main portion of the molecule) is represented by dotting or similar device rather than a continuous line.

**Isoxazole ring**—an isomer of oxazole.

**Lactic acidosis**—a difficultly treatable acidosis usually due to an inadequate supply of oxygen to bodily tissues. Often fatal.

**Lignin**—a complex organic material (a phenolic polymer) that binds cellulose fibers together to form stiff cell walls in trees and woody plants.

**Limb**—in botany and mycology, a colored zone at the edge of a leaf or mushroom cap or a membranous extension from a plant or mushroom part are examples of what may be called a “limb”.

**Malaise**—general but non-specific feeling of being sick.

**Methemoglobinemia**—a form of hemoglobin which carries oxygen poorly. See methylene blue below.

**Methylene Blue**—a dye which, though a staining agent in microscopy, is also used medically to reduce ferric heme (methemoglobin) to ferrous heme (normal hemoglobin).

**Mitochondria**—organelle within cells where energy from respiration is stored and released by oxidative phosphorylation reactions.

**Mycorrhizal**—referring to a mutually beneficial relationship between mushrooms and the rootlets of green plants (including bushes and trees) in which the nutrients of life are exchanged.

**Muscarinic**—a compound that stimulates the sympathetic beta receptors directly or indirectly. See discussion under Parasympathetic nervous system.

**Muscimol**—An isoxazole alkaloid (acts as a GABA receptor inhibitor).

**Neurotransmitter**—a compound that carries information through a small space of extracellular fluid from one neuron to an adjoining neuron and activates the latter.

**Nicotine**—an alkaloid that acts as an acetylcholine receptor agonist, but primarily in specific tissues, such as skeletal muscle. (other types of muscle are smooth muscle lining arteries and cardiac muscle)

**N [normal]**—1 mole per liter. A mole is an international unit that expresses the number of molecules that take place in a chemical reaction.

**Octopeptides**—An eight unit peptide. (see peptide)

**Oliguria**—decreased urine output.



**Oxoheterocyclic quarternary salt**—Oxo (designating the presence of a carbonyl group) heterocyclic (a ring of carbon compounds with at least one other element inserted) quarternary (a set of four ammonium compounds) salt.

**Organelle**—specialized structure in the gel-like fluid of a cell. Less commonly used to refer to specialized portions of organs.

**Parasympathetic nerves and parasympathetic nervous system**—One of the two major autonomic peripheral nervous system divisions. This division uses acetylcholine as its neurotransmitter and over-stimulation produces atropine-like effects with dry mouth, slowing of heart rate etc; also see nicotine. Parasympathetic fibers go to the heart, lungs, ordinary sweat glands etc. Brain cells also trigger using acetylcholine as the neurotransmitter. The other autonomic nervous system division is the sympathetic nervous system.

**“Patches”**—used to describe fragments of universal veil on the cap (as opposed to “scales” which are innate to the substance of the mushroom cap.

**Peptides**—chains of amino acids linked by phosphate bonds. Proteins are peptides (commonly referring to the larger chains). Peptides linked head to tail are cyclic peptides.

**pH**—the common form of reporting acidity. It is the reciprocal of the log to the base 10 of the H<sup>+</sup> ion concentration in millimoles per liter. A pH below 7 is acidic; a pH of 7 or higher is alkaline.

**Phosphorylation**—storage and release of energy in phosphate ester bonds.

**Plasma**—blood with the red cells removed. Serum is plasma allowed to clot; coagulating factors are removed.

**Plasmaphoresis**—the selective removal of plasma from whole blood by membrane filtration or other methods. Plasma or a substitute then needs to be replaced.

**Plasma exchange**—removal of plasma after plasmaphoresis and substitution by donated plasma or albumen (the major protein in plasma).

**Platelets**—small ±flat organelles in the blood that usually initiate clotting.

**Phosphorylated and Dephosphorylated**—adding or subtracting phosphate— (PO<sub>4</sub><sup>-</sup>)—having to do with the storage of energy.

**Prothrombin time**—The protein prothrombin is part of the clotting cascade. It is produced by the liver. Liver damage, however, is only one way that clotting occurs in association with a long prothrombin time.

**Proximal tubules and distal tubules**—in the kidney, the proximal tubules are the ones nearest the round glomerulus or filtering apparatus; the proximal tubules are followed by the thin loop of Henle and then by the distal tubules (which are microscopically near the glomeruli and thus indistinguishable (in the cortical or outer area of the kidney); these distal tubules are followed by the collecting tubules which run down into the hollow portion of the kidney. These are easily seen as they are microscopically close to that inner portion of the kidney (the medullary area). Loop diuretics used to treat amatoxin poisoning by diuresis (increased urine flow). The loop diuretics work on the loop of Henle and on the distal tubules.

**Pulmonary edema**—water around and in the air sacs of the lung; an emergency.

**Pyridoxal**—an oxidized derivative of vitamin B12 which is a co-enzyme for many biochemical reactions.

**Quinone**—an oxidation product of a dihydroxybenzene compound.

**Radioimmunoassay**—radioimmunoassay systems use the specificity of antibody reactions and the sensitivity of radioactive tracers to quantitatively measure components in very dilute solution (such as most compounds in serum).

**Receptors**—portions of cell membranes that are so configured that they accept only certain oppositely configured compounds that fit into these areas like a lock and key. When these receptors accept such compounds, a chemical cascade occurs in which a final reaction occurs within the cell.

**RNA**—Ribonucleic acid; a chain of nucleotides with a backbone of alternating sugar and phosphate molecules with a nitrogen base attached to each ribose sugar molecule. The strands are made of nucleotides (a backbone of alternating sugar and phosphate molecules with a base attached to each sugar). The bases of RNA are abbreviated A, C, G and U (adenine, cytosine, guanine & uracil) and encode proteins in the ribosomes—tiny structures found in all cells. Free ribosomes scattered in the cytoplasm produce proteins used mainly within the cells; those attached to a membrane called the endoplasmic reticulum produce proteins that are secreted by the cells.

**Salts**—A chemical compound in which the first portion has a positive ion charge and the second a negative ion charge. Sodium chloride (table salt) is an example, but the salts noted in the text are biochemical ones such as the various amine salts.

**Scale**—an intrinsic, but differentiated, often slightly elevated flap of cap tissue; note the surface of common *Agaricus*, *Lepiota* and *Macrolepiota* species.

**Seceding**—refers to ±blunt gill attachments where the gills have now have pulled away from the stipe, lending the appearance of their being free; longitudinal lines are left on the stipe.

**Sensu**—in the sense of (from the Latin).

**Sensu lato**—Latin for “in a wide sense”.

**Sensu stricto**—Latin for “in a narrow sense”.

**Serotonin**—central nervous system neurotransmitter controlling sleep/waking centers, anaphylactic reactions, some sympathetic nervous system and brain stem reactions. Frequently it acts as a modulator of other neurotransmitter for the autonomic nervous system.

**Serum**—see plasma.

**Slides of fatal *Amanita phalloides* poisoning**—(EL, 1970 California poisoning):

1. **Liver**—Centrilobular necrosis (pale areas, mostly in the center of each liver lobule shows death of cells, although the severity is such that often the areas somewhat interconnect). Some fatty replacement is present. The area between the liver lobules show that the lining of tiny bile ducts (ductal epithelium) is increased and some of the cells are swollen.
2. **Kidney**—The round structures are glomeruli (filtering apparatus) and are minimally swollen. The surrounding tubules are swollen and fuzzy with loss of portions of the lining.

**Sorbitol**—a complex sugar which in concentrated oral solution acts to pull water out of the space between the body cells (and eventually from the cells) producing a "hyperosmotic" diarrhea, since the solution is far more concentrated than bodily fluids.

**Stipe**—stalk part of a mushroom.

**Stizolobic and stizolobinic acids**—these isomers of ibotenic acid probably have little effect in pantherine poisoning, but they are present and activate excitatory amino acid receptors. Tricholomic acid is another isomer.

**Sympathetic nervous system**—that portion of the autonomic nervous system, which peripherally has alpha and beta receptors using noradrenaline and adrenaline respectively. Alpha receptor effects include constriction of blood vessels and increased force of the heart beats; beta receptor effects peripherally produce muscarine effects such as salivation and rapid heart rate. Dopamine is a precursor of noradrenaline and adrenaline (as well as a CNS neurotransmitter for nervous tissue in the brain). See parasympathetic nervous system.

**Taxon**—a taxonomic entity; usually refers to a fungus or fungi whose taxonomic status is unclear (especially as to its status as species, subspecies or form).

**Taxonomy**—the classification of fungi by genetic, structural and chemical changes; an effort made to find their place in the order of plants according to an evolutionary tree or diagram of such findings.

**Thin-layer chromatography**—an assay performed in an electromagnetic field on thin silica gel plates. Known and unknown compounds are applied in a solvent and allowed to migrate along the surface. The colors and distances are matched for known and unknown compounds. The colors are brought out by sprays of one or more colorizing compounds (usually cinnamaldehyde spray and then exposure to hydrogen chloride gas for amanitins)

**Transvenous**—through or into a vein.

**Tricholomic acid**—an oxazole similar to ibotenic acid and muscimol, but its toxicity is unproven. It is dihydro-ibotenic acid (L-erythro- $\alpha$ -amino-3-oxo-5-isoxazolidine acetic acid). See Ibotenic acid.

**Trimester**—the 3 divisions of pregnancy, each consisting of 3 months. The first trimester is very important as medications and poisonings occurring here are most likely to damage the fetus.

**Tryptophan**—an indole-containing aminoacid and the precursor to serotonin. (See serotonin).



**Tyrosine**—an amino acid and the precursor to the neurotransmitter dopamine and noradrenaline.

**Type (collection)**—There is or was always an original type collection, whose material may be sent to various institutions for taxonomic study. Unfortunately, the original specimens may be lost from the original herbarium as well as from those donated to other herbariums. In such a situation, a new collection from that area or a previously documented area is matched with descriptions and any fragments in place of the original. Errors also occur.

**Urea**—an end product of protein breakdown.

**Umbo**—a central knob left on the cap of a mushroom from an earlier ±conical shaped cap.

**Vitamin B6**—Pyridoxine: one of 3 forms of B6. Vitamin B6 will combat to some degree CNS toxicity due to vitamin B6 inhibition by gyromitrin.

**Viscid, subviscid and glutinous caps** vary in the amount of surface “slime”. A viscid cap is slippery to the touch; a subviscid cap is only “tacky” (pulls at the lip when touched to the lip, but needs moistening first); a glutinous cap has an obvious veil of slimy material.

**Volva**—remnants of a universal veil around the early embryo; it arises from the basal portion of the veil and appears at the base of the stipe as a sac, collar, rings and sometimes, looser, (more amorphous) material.

**Warts**—here used to indicate agglutinated patches of universal veil tissue on mushroom caps (as opposed to “scales” which are innate to the cap).

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