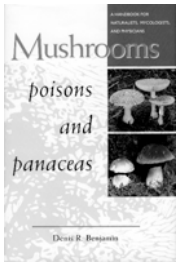
Mushroom Toxins & Poisonings (N.J. & NE U.S.A.)

POISON CONTROL: In New Jersey, "poison control" is NJPIES (New Jersey Poison Information and Education System) and can be reached by dialing **1-800-222-1222** (works in all states, but (WARNING) will be automatically connected to the home state of a cell phone) or **1-800-POISON1** (connects only to NJPIES).



These notes prepared by Rod Tulloss and Dorothy Smullen for an NJ Mycol. Assoc. workshop, 25 March 2006. When viewed with Acrobat Reader, underlined red or gray words and phrases are "hot linked." We have included a few notes on fungal poisons that are not from "mushrooms." We caution that this document has not been reviewed by a professional toxicologist or pathologist.

Main reference: Benjamin, D. R. 1995. *Mushrooms: Poisons and panaceas*. (W. H. Freeman, New York). xxvi+422 pp. Citation of this reference omits the date of publication. Other valuable references may be found in "Bibliography" on page 20. The authors have also added information from their own experiences and unpublished data and information supplied by persons experienced with identifying mushrooms in poisoning cases and tracking the course of those cases (see "Acknowledgments" on page 20).

Table 1. Benjamin's "Major Clinical Syndromes" Chart

For miscellaneous other syndromes and their causes, see the section below labeled "Miscellaneous or recently reported syndromes caused by fungi" on page 16 and "Table 2. Benjamin's list of rarely eaten toxic fungi [not otherwise covered in this summary]" on page 2.

Onset of Symptoms	Initial Signs & Symptoms	Evolution of clinical features	Syndrome
I. **Greater than 4 hours**	6-8 hrs: Vomiting, diarrhea, abdominal pain, fatigue, headache	Liver failure, hemolysis, fever, methemaglobinemia	Gyromitrin
	8-24 hrs.: Vomiting, diarrhea, abdominal pain	Liver failure (possible renal failure)	Amatoxin
	less than 48 hrs: Polyuria, polydypsia, & amatoxin like symptoms	Renal failure (possible liver failure)	Amino Acid
	greater than 48 hrs: Polyuria, polydypsia	Renal failure	Orellanine
II. **Less than 4 hours**	Nausea, ataxia, confusion, delirium [vomiting may occur with some species, but not with others]	Sleep/coma, "hallucinations," muscle fasciculations"	Pantherine
	Nausea, vomiting, diarrhea, abdominal pain		Gastrointestinal
	Perspiration, lacrimation, nausea, salivation, bradycardia		Muscarine
	Dysphora, euphoria, sense of exhilaration	Hallucinations, mydriasis (excessive dilation of the pupil)	Hallucinogens
III. Within **15 mins.** of drinking alcohol	Headaches, facial flushing, tachycardia, nausea, vomiting (Note: mushrooms may have been ingested up to 3 days previously.)		Coprine
Note: In case of ingestion of a mixture of mushrooms (e.g., *A. rubescens* var. *alba* and *A. bisporigera*, which did occur in New Jersey in RET's experience), the onset of symptoms will be the earliest onset of set of the potential syndromes. Quick occurrence of a gastrointestinal symptom can either clear other (e.g., potentially deadly) material from the stomach or mask the presence of a serious threat to health. Blood tests revealing malfunction of the liver and kidney should be utilized if there is any question of the patient having eaten more than one mushroom.			

Table 2. Benjamin's list of rarely eaten toxic fungi [not otherwise covered in this summary]

Formal and common names	Symptoms
Boletus pulcherrimus	Muscarinic effects
Clathrus cancellatus (=*C. columnatus*)	Convulsions, dysarthria, coma
Gomphus floccosus (woolly chanterelle; widely eaten in Mexico)	Nausea, vomiting, diarrhea (CNS depression, muscle weakness in rats). Onset can be delayed. May be due to norcaperatic acid.
Gomphus kauffmanii	Same as above
Gymnopus dryophilus (=*Collybia dryophila*)	Gastrointestinal
Gymnopus acervatus (=*Collybia acervata*)	Gastrointestinal
Mycena pura (lilac mycena)	Muscarinic effects
Phaeolepiota aurea	Vomiting, diarrhea, and colicky abdominal pain
Ramaria formosa (yellow-tipped coral fungus; many species of coral fungus are commonly sold in Mexican markets)	Gastrointestinal
Ramaria pallida (Bauchweh-Koralle, colic coral)	Gastrointestinal
Ramaria flavobrunnescens	Death of livestock in S. America
Scleroderma cepa and *S. aurantium* (thick-skinned pigskin, common earth-ball) Note: Bits of *Scleroderma* tissue are used to adulterate packaged truffles. Janet Lindgren reports (pers. comm.), "In [the PNW,] sclerodermas cause a lot of poisonings in dogs. Also, people try them because they think they are truffles."	Abdominal pain, nausea, generalized tingling sensation, marked spasms, rigidity (see "Scleroderma cepa "rapid rigor" syndrome" on page 18)
Stropharia coronilla	Malaise, headache, generalized aching, ataxia, dizziness, vomiting, hallucinations, confusion
Tricholomopsis platyphylla	Colicky abdominal pain, vomiting and/or diarrhea, muscle cramps and spasms
Tricholoma irinum and *Tricholoma sulphureum* (sulfurous or gas agaric)	Nausea, vomiting, neurological symptoms
Also, see "Paxillus syndrome (immune hemolytic anemia)" on page 16.	
Note: Within this group are taxa which may become more commonly eaten in N. America because of the widespread presence of Latin-American (and, especially, Mexican) immigrants. Indigenous cultures in Mexico make extensive use of a wide variety of comestible fungi, including fungi that (if, indeed, the species are correctly identified) are considered toxic in the US and Canada. Symptoms are diverse in this group.	

Acute adverse reactions not caused by a mushroom toxin Many of the reactions listed in Table 3 , below, are somewhat self-explanatory.

Benjamin repeatedly gives attention to potential confusions of mushroom poisonings with the panic reaction, which he describes as symptoms deriving from the belief that death is certain once a mushroom-eater loses faith in his/her determinations of mushroom edibility after a mushroom meal. For example, see "Muscarine (PSL or SLUDGE) syndrome" on page 14.

Poisoning due to the multitude of man-made toxins sprayed on lawns, parks, golf courses, etc. is something with which Benjamin's personal experience suggests to thim that such "treated" areas should very simply be off-limits to persons collecting fungi for the table.

The terms “idiosyncratic reaction” and “allergic reaction” probably include syndromes that may some day be separately listed in a synopsis such as this or a book such as Benjamin’s; but, at present, our ignorance is to great to say anything very useful at this time.

Table 3. Benjamin’s chart of acute adverse reactions not caused by a mushroom toxin

Panic reactions
Bacterial food poisoning (1) due to spoiled or rotten specimens (2) due to improper preparation or storage
Insecticide, herbicide, or fungicide contamination
alcohol intoxication
idiosyncratic reaction
allergic reaction (1) gastrointestinal (2) respiratory (usually due to spores)
intestinal obstruction
long term effects: (1) heavy metal poisoning (2) radioisotope contamination (3) cancer

Review of syndromes and the species/toxins that cause them The symptoms are discussed in the reverse order of the chart—with the longest onset cases first.

When a poisoning case has been caused by a mushroom, identification of the mushroom to genus, a subgeneric group, or species may be very helpful in terms of predicting the course of symptoms that may occur and their sequence in time. For example, it can be of considerable help to a victim psychologically to know that his/her experience is not life threatening or that his/her bout of gastroenteritis will end in 24 hours. In one case of a child’s poisoning by *Chlorophyllum molybdites*, vomiting was continuing beyond the normal expected time and the usually final symptom of clear diarrhea had been observed. Within 20 minutes of being told that she was “finished,” the child ceased to be symptomatic and was discharged from the emergency room.

Note: You may often hear people refer to the toxins discussed herein as “secondary metabolites.” This reflects the idea that they are not produced for a specific, known use/need of the organism that produces them. There may be no advantage to them at all for the organism. (For example, destructive slugs and burrowing insect larvae as well as some birds and mammals may do a great deal of damage to a mushroom, but they seem able to survive encounters with toxins deadly to humans.) The toxins simply have not been selected against by the process of evolution.

Note: Determination of a mushroom to species may often require a specialist. A non-specialist should focus first on determination to genus, then to section (or some relevant “group” within a genus). This latter may be all that is needed in the first pass at an unknown mushroom suspected of causing a poisoning.

I. Delayed onset (greater than 4 hours)

A. Amatoxin syndrome (poisoning by certain cyclopeptides)

Note: To be expected in the region of interest. Species containing the toxins are common in the region.

“Onset of the gastroenteritis is at least six hours after mushroom ingestion. The gastrointestinal symptoms are followed after a variable time (48-96 hours) with the development of evidence of acute hepatic toxicity, which may rapidly lead to liver failure.” (Benjamin, p. 179)

This toxin group is known from one section of the genus *Amanita* [sect. *Phalloideae*, which, in NE N. America includes the following: *A. bisporigera* (=*A. virosa sensu auct. amer.*), *A. magnivelaris*, *A. elliptosperma*, *A. phalloides*, and *A. tenuifolia*. Also see the key to N. American taxa of *Amanita* sect. *Phalloideae* online at

`http://pluto.njcc.com/~ret/amanita/keylist.html`

], a group of usually small taxa within the lepiotaceous fungi (*Lepiota castanea, L. helveola, L. josserandii*), species of *Galerina* (little brown mushroom with ring on stem, growing on wood—*Galerina autumnalis*—and similar taxa in lawns—*G. venenata*), and some species of *Conocybe* (e.g., *C. filaris*).

Amatoxins can be detected in a fresh mushroom sample by a simple chemical procedure called the **Weiland Test** (sometimes called the Meixner Test in the US). The test has been demonstrated to be very sensitive to the presence of amatoxins with very, very low occurrence of false negatives. However, the test's results are dependent on the composition of the newsprint that is utilized. Janet Lindgren (Vancouver, Washington, pers. comm.) reports unsatisfactory results with newspaper in the Pacific Northwest.

The test requires cheap newsprint (with significant lignin content), concentrated HCl, a hair dryer or other source of moderate heat. [Note: hydrochloric acid requires special handling and storage.] Two circles are made with a pencil in an unprinted area of a newsprint. Fresh tissue is crushed into one of the circles only; the other serves as a negative control. The newsprint is then moist from the juices of the mushroom and is allowed to dry on its own or dried with gentle heat <u>out of direct sunlight</u>. When the newsprint is dry, a drop of the HCL is placed in both circles. A <u>blue color</u> within 15 to 20 minutes in the circle containing dried juice from the tested mushroom indicates the presence of amatoxins. (Note: If both circled areas turn blue, the test has failed because of the content of the newsprint; and you must consider that you may have a false positive.). <u>Colors other than blue</u> have been reported for chemicals other than amatoxins in the Weiland Test. Non-blue response can be considered a negative response. Considering the reports of Janet Lindgren (pers. comm.), it seems wise to buy a stock of newsprint that has been proven to work well (no false positives and minimum false negatives) and to rely on this stock for the Weiland test. Note that, at least in *Amanita*, not every fruiting body contains an equal amount of toxin; and some fruiting bodies of commonly toxic species contain no measurable toxin at all.

Amatoxins are <u>cyclopeptides</u> and are **not** destroyed by cooking, freezing, or drying. (Benjamin, p. 212)

Key: If the onset timing suggests the amatoxin syndrome, in E N. America the mushroom must be a gilled-mushroom. If it is not, make another choice from the syndrome chart. In the case of a gilled-mushroom, this key may help determination of the mushroom genus. Microscopic observation is usually not possible in a hospital and relies upon fresh or well-dried material being placed in the hands of a mycologist in a timely manner. Treatment should never be delayed in order to accommodate microscopy; and, in current practice, it is not.

1. Spores are yellow-brown to brown to rust-brown; cap width about that of a US quarter or less; stem no thicker than a pencil, often thinner than an old-style, mercury thermometer, bears a small, persistent ring.....2

1. Spores are white or pale colored; stem bears a large, persistent ring.....3

2. The cap is markedly conic or rounded conic with margine flaring upward with age, fragile, white to pale tan to tan; stem very narrow, fragile; habitat in lawn ..... consider *Conocybe*.

2. The cap is not conic, often yellowish or brownish, stem not very narrow; usually found on wood, infrequently in lawn.....consider *Galerina*.

3. The mushroom is small; the cap's skin is broken up into small colored patches and distributed over the surface with much of the white flesh showing; there are no inflated cells in the flesh of the cap or stem; trees not required in habitat (e.g., could be in mulch of flower bed); the spores are <u>not</u> amyloid (not blue-black in Melzer's Reagent), but may turn reddish if treated in dilute ammonia and then in Melzer's Reagent (dextrinoid reaction).....consider *Lepiota*.

3. The mushroom may be of diverse sizes; the cap's skin does not break up into small bits and is white or shades of gray, brown, yellow, olive, etc. that may be combined in a pattern that looks somewhat like

fine, interwoven, radial hairs; habitat usually includes trees such as oaks, beeches, nut trees, conifers, etc; a drop of 5-10% KOH on the cap will produce a bright yellow or yellow-orange reaction in several white-capped species (no reaction in some); the spores are amyloid (turn blue-black in Melzer's Reagent; the flesh of the cap and stem include inflated cells[1] (elongate and vertically oriented in the stem) (for more detail, see `http://pluto.njcc.com/~ret/amanita/keylist.html`). 4

4. The cap is white. 5

4. The cap is mottled under a 10x lens, but appears radially streaked to the naked eye, with a mixture of the following colors, yellow, olive green, gray, brown, etc.; spores[2] broadly ellipsoid to ellipsoid ((7.5-) 8.0 - 10.1 (-12.5) × (5.5-) 6.1 - 8.0 (-9.0) µm, with spore length/width ratio = 1.20 - 1.33 (-1.40)) ..... *Amanita phalloides*.

5. The spores of the species are globose to broadly ellipsoid ((5.2-) 7.2 - 9.9 (-11.0) × (4.8-) 6.4 - 8.5 (-10.0) µm, with spore length/width ratio = (1.05-) 1.06 - 1.18 (-1.20)); the cap turns yellow after application of 5 - 10% KOH; the volva's free part (limb) tends to turn in toward the stipe rather than stand up straight. ..... *Amanita bisporigera*.

5. The spores of the species are broadly ellipsoid to ellipsoid, the cap may or may not turn yellow after application of 5-10% KOH; the volva's free part (limb) often stands up parallel to the stem well into maturity. 6

6. The skirt on the stem is not a thin membrane, but instead as moderately thick and clearly felted and may stand out straight from the stem after separation from the cap margin; the volva's free limb stands up parallel to the stem well into maturity; the spores are broadly ellipsoid to elongate (rarely subglobose, rarely cylindric). Note that spore data is somewhat limited at present, but probably does not make for a useful character in separating these two "taxa" 7

6. The cap either doesn't turn yellow after application of 5-10% KOH or the reaction is unknown; the skirt on the stem is a thin membrane that soon collapses against the stem; position of the volva's limb may vary; the spore may vary widely in different species from subglobose to ellipsoid in some to elongate to cylindric in others 8

7. Fruiting body surfaces yellow with exposure to air and turn bright yellow after application of 5-10% KOH (possibly parasitized specimens of *A. magnivelaris*); the spores are (8.5-) 8.8 - 12.1 (-14.4) × (5.8-) 6.5 - 8.5 (-9.0) µm, with spore length/width ratio = (1.13-) 1.25 - 1.69 (-2.01); known from SE Canada. ..... *Amanita decipiens sensu* Yves Lamoureux

7. The fruiting body remains entirely white after exposure to the air and does not react to KOH; the spores are (7.2-) 8.2 - 10.8 (-12.5) × (5.2-) 5.8 - 7.8 (-9.2) µm, with spore length/width ratio = (1.15-) 1.27 - 1.62 (-1.72); known from SE Canada and NE USA ..... *Amanita magnivelaris*

8. The cap does not turn yellow with application of KOH; the volval limb stands up parallel to the surface of the stem well into maturity; the spores are (7.0-) 8.0 - 11.2 (-12.0) × (4.8-) 5.2 - 8.2 µm, with spore length/width ratio = 1.32 - 1.66 ..... *Amanita elliptosperma "group"*

8. The position of the volva is not recorded; the color reaction to KOH is not recorded; the species are usually small; there may be an odor of garlic in one species (with relatively broad spores); spore shape ranges widely from broadly ellipsoid to cylindric in different species; current range is southeastern

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1. Note that species of *Amanita* and other agarics can be separated even when all you have is a cooked piece of stem. Just look for the longitudinally-oriented inflated cells (called acrophysalides) in the stem tissue. If they are present and plentiful (defining the character of the tissue), you have an *Amanita*.
2. Note that to accurately reproduce length/width ratios, spores for the genus *Amanita* should always be measured with the same methodology—in side view only (with apiculus and adjacent flattened surface clearly in view and with both ends of the spore in focus). Unless testing for the amyloid reaction, spores should be observed in water (not KOH solution, for example) to avoid damaging features of the spore surface or altering pigmentation of the spores.

coastal plain of N. America and some states along the Mississippi River (WARNING: could change with global warming).....Murrill's "Florida" species.

B. Gyromitrin syndrome (poisoning by monomethylhydrazine and other hydrazines)

Note: Not yet experienced by RET & DS in the region of interest. Mushrooms that cause the syndrome are present in the region of interest in early to mid-spring.

"Onset of the gastroenteritis is six to eight hours after ingestion, accompanied by fever, severe headache, muscle cramps, [muscle spasms], evidence of hemolysis, and progressive liver failure." (Benjamin, p. 179)

The toxin known as gyromitrin is monomethylhydrazine (rocket fuel!). It is volatile, and people have been known to be poisoned by it because they removed the lid from a cooking pot of false morels (in the genus *Gyromitra*) and sniffed the vapors. Dogs have been killed by eating leftovers, when the humans who ate the original meal were not noticeably affected by the toxin. The human body, however, can accumulate the toxin over a period of weeks. In such a case, there may be no symptoms at first, but they may come on with a vengeance after a personal "threshold" is reached.

Ascomycetes other than species of *Gyromitra* may contain monomethylhydrazine in varying amounts. Benjamin lists these genera as suspect: *Disciotis*, *Helvella*, *Peziza*, *Sarcosphaera*, and *Verpa*. Small amounts of monomethylhydrazine have been found in some morels. Never eat morels uncooked! Modern field guides allow separation of the cited genera.

C. Orellanine syndrome (poisoning by certain cyclopeptides)

Note: Not yet observed by RET and DS in the region of interest. The only mushroom known to contain orellanine in North America does not occur in the region. However, most species of the large genus *Cortinarius* have NOT been tested for cortinarins. The genus is very well represented throughout E N. America.

The onset may take 12 hours to several weeks and so may not be connected by the patient to his/her eating mushrooms in the past. "The gastroenteritis is followed by the development of an increased frequency of urination (polyuria), intense thirst (polydypsia), and other evidence of kidney failure." (Benjamin, p. 179) It has been hypothesized that the long onset period contributed to the failure of the mushroom eating public and scientists to recognize that there were deadly problems with some species of *Cortinarius*.

The current understanding of the orellanine syndrome is that it is caused by at least one of the cyclopeptides called cortinarins.

As of 1995 (Benjamin, p. 244), no well-documented case of orellanine poisoning had been reported from N. America. BUT, why eat *Cortinarius* and related species? Since they are not often eaten in N. America, and the number of species is very large, it is not known how many American taxa of *Cortinarius* contain orellanine or similar compounds.

The most recent information on orellanine-containing species of *Cortinarius* was supplied to Marilyn H. Shaw (Denver, Colorado) by Dr. Joseph Ammirati (5 July 2005): "From my experience there is only one species that has orellanine in temperate, boreal, and montane [N. America;] the current name is *C. rubellus* (=*C. rainierensis* =*C. speciosssimus* =*C. orellanoides*). ....*C. rubellus* occurs across boreal Canada, in [British Columbia], Washington [state], [and has] not been seen...in the Rockies, but could occur there [and] eastern NA as well." The non-urban, indeed rather restricted habitat, contributes to the unlikeliness of the species being ingested.

The Basques, some of whom were professional poisoners in centuries past, had some knowledge that a few species of *Cortinarius* were deadly. The mushroom meal could be so far separated from the mysterious death that suspicion was rarely if ever directed at the provider of a forgotten meal. The knowledge is preserved at least in the Basque language. It was noted that a deadly *Cortinarius* lacking a common name in other European languages had a two word common name in Basque. One word means brown, and the other is not used on its own commonly. The clue comes from the fact that, when the second word is combined with the word for white, the resulting phrase is the Basque name for one or more white species of *Amanita* section *Phalloideae* (see "Amatoxin syndrome (poisoning by certain cyclopeptides)" on page 3).

Note: *Amanita smithiana* poisoning has been mistaken for orellanine poisoning in a few cases, prior to the appearance of *A. smithiana* in accessible toxicological literature. Onset is more rapid in the case of *A. smithiana* and other causes of the "amino-acid" syndrome and white-spored, white-capped, often rooting amanitas of *Amanita* section *Lepidella* are easily distinguishable at sight from rusty brown-spored, yellow- or brown-capped species of *Cortinarius*.

D. "Amino Acid" syndrome (poisoning by certain non-nucleotide amino acids)

Note: Nearest case to region of interest known to RET was in Baltimore, MD, and involved *A. nauseosa*. Species potentially containing such a toxin are relatively common in summer and early autumn in the region.

"...symptoms of vomiting, abdominal pain, and diarrhea developed between 4 and 11 hours after mushroom ingestion. It is entirely probable that *A. smithiana* contains a renal epithelial toxin in addition to other compounds responsible for a gastrointestinal syndrome." (Benjamin, p. 379)

When some poisonings of this type were reported in 1990, they were reported as examples of the orellanine syndrome, and it was assumed that a species of *Cortinarius* were involved. The mistake is understandable as the amino acid syndrome was not recorded in the toxicological literature. By showing colored images to one or more of the victims, it was ascertained that a large white species of *Amanita* sect. *Lepidella* was being mistaken for the desirable "matsutake" in the Pacific NW. There is obviously a cautionary tale here regarding determination of the cause of poisoning based on the symptoms alone—symptomatic treatment can resolve the immediate problem of a given patient, but a public health hazard and the mycological/biological information may not be properly identified and/or may remain hidden.

The mushroom involved was found to be *Amanita smithiana* (in *Amanita* sect. *Lepidella*) of the W and SW US states, SW Canada and (possibly) W and central Mexico. It is important to note that the same symptoms are caused by other (remotely related) taxa in sect. *Lepidella* (e.g., *A. nauseosa*, with range extending from the Gulf of Mexico to Baltimore, Maryland, and occurring commonly in lawns and parks, with OR without trees). Be wary of all taxa in sect. *Lepidella*! (Tulloss and Lindgren, 1992)

According to the few world experts in *Amanita* taxonomy, there are six or seven sections within the genus. Four of these fall in *Amanita* subgenus *Lepidella*, which is defined by having amyloid spores. Within subgenus *Lepidella*, those of section *Lepidella* are determined by having an appendiculate cap margin (powdery material usually white) and do not have a thick, membranous, saccate volva. A number of the species in the section have a strong, distinctive odor. In fact, the name of *A. nauseosa* was created to indicate that the mushroom has a rather nasty odor—like an old mouse nest or stale tiger urine(!). Another toxic *Lepidella* often found in lawns and open areas in the US is *A. thiersii*, which ranges from central Mexico at least as far north as S Illinois and S Indiana.

Odor alone is not sufficient to characterize amanitas in section *Lepidella*. *Amanita abrupta* is relatively odorless, but is suspect because a closely related species, *A. sphaerobulbosa*, is reported to have caused serious poisonings in Japan and is reported further to contain an amino acid that causes liver destruction in animals—possibly, the same toxin found in *A. smithiana*. The closest relative to *A. smithiana* in the NE USA is *A. rhopalopus*.

Determination of a specimen of *Amanita* sect. *Lepidella* to species, may require a specialist.

For a list of the taxa in sect. *Lepidella* and basic taxonomic information about many of those species, see < `http://pluto.njcc.com/~ret/amanita/sectlepi.html` >.

WARNING: The range of fruiting of southern species of section *Lepidella* is apparently expanding with global warming.

II. Onset less than 4 hours

A. Pantherine syndrome (poisoning by ibotenic acid and its derivatives such as muscimol)

Note: If "hallucinations" are reported with little other detailed information see "Hallucinogenic syndrome (poisoning by psilocybin and other tryptamine derivatives)" on page 15 and "Ergotism (poisoning by LSD precursors in Claviceps purpurea a parasite of grains)" on page 17.

Note: Poisonings of this type occur occasionally in the region of interest. The mushrooms involved are very common in the region.

Benjamin (p. 310) describes the onset as "almost always between 30 and 120 minutes after mushroom ingestion. In very unusual circumstances, onset is delayed up to 6 hours."

Benjamin (p. 312) describes the symptoms, which peak at 2 to 5 hours and have a duration of 8 to 24 hours as the following:

- nausea (and vomiting with some species)
- confusion or delirium
- incoordination and ataxia, dizziness
- alternation between lethargy and euphoric and manic behavior
- progressive, deep, comalike sleep
- hallucinations, visual distortions
- muscle fasciculation, with cramps and spasms
- generalized seizures (rare)
- hangover headache possible.

In one infamous case of gluttony, the ingestion of more than a dozen large specimens of *Amanita muscaria* var. *guessowii* (mistaken for the European edible *Amanita caesarea*) lead to death in an adult male. The Euro-asian *A. muscaria* var. *muscaria* is used by shamans who say that it is deadly if more than 13 dried fruiting bodies are eaten. (Wasson, 1968)

Marilyn Shaw (pers. comm.) has noted that many patients experiencing the pantherine syndrome have reported to her that they were aware of their surroundings when they were "comatose," but were unable or unwilling to respond. Also, she notes that if one poisoned person is caring for another prior to hospitalization, the first can seem to be able to suppress symptoms until both are being cared for in the ER

The pantherine syndrome is caused by ibotenic acid and its derivatives including muscimol. Muscarine (see "Muscarine (PSL or SLUDGE) syndrome" on page 14) plays no significant role in intoxication by the taxa involved with the pantherine syndrome. Muscarine gets its name from the fact that it was first extracted from *Amanita muscaria*, in which it occurs in small amounts. For a period of time, the pantherine syndrome was treated with atropine (because the syndrome was thought to be caused by muscarine).

Muscimol may suppress vomiting. Marilyn Shaw (pers. comm.) recounts a veterinary case in which repeated attempts (using four different methods) to make a dog vomit were unsuccessful. She also says that her experience with *A. muscaria* subsp. *flavivolvata* is that vomiting is not common. On the other hand, in cases involving the American mushroom(s) usually misidentified as *A. pantherina* (a distinct Euro-Asian species), vomiting is common. Both *A. muscaria* and *A. pantherina* contain ibotenic acid and its derivatives.

In some parts of N. America, persons making "recreational" use of species of *Amanita* section *Amanita* (taxa related to *A. muscaria* and *A. pantherina*) are the most common patients exhibiting the pantherine syndrome. However, in New Jersey the authors have more often been confronted with this syndrome due to mistaken identification or ignorance.

RET has experienced the following: (1) ingestion of *Amanita crenulata* by a starving, homeless person, (2) ingestion of *A. muscaria* var. *guessowii* in error when the collectors thought they had *Armillaria mellea* (!), and (3) ingestion of *Amanita muscaria* var. *guessowii* by recent Mexican immigrants who mistook the mushroom for *Amanita basii*—the desirable, comestible Mexican Caesar's mushroom. ["Amarillo" (yellow), "tecomate" (squash blossom), "xical," and "yema" (yolk) are among the common names for this and related species in central Mexico.] *Amanita basii* does not occur in the US so far as we know.

Dogs (at least young ones) are also susceptible to the temptation to eat mushrooms inducing the pantherine syndrome. RET recently received a very damaged specimen of what appears to be *A. muscaria* var. *guessowii* from the case of a puppy's poisoning in Pennsylvania.

The *A. crenulata* case was one of the most dramatic that RET has ever experienced. A man was found asleep on a train track lying parallel to, and between, the rails. An emergency medical technician awoke him and was almost immediately attacked by the man who behaved in a deranged manner. His pockets were "stuffed" with *A. crenulata*. Apparently, he had attempted to assuage his hunger with a large amount of the mushroom. Symptoms lasted for 6 days, during which time the patient had to be physically restrained by being bound to his hospital bed. He repeatedly attempted to attack hospital staff.

The case involving a recent immigrant from Mexico illustrated the confusion that can occur surrounding the use of "common names." In a phone interview with the patient in the emergency room, a Spanish translator was on the line with RET and a staffer from NJPIES. The translator kept saying that the woman said the mushroom was yellow (amarillo). Finally, RET asked the translator if the woman was saying that the mushroom was yellow or that the mushroom was called "el amarillo." The latter turned out to be the case. Knowing that this term was applied to a very desirable edible mushroom in parts of Mexico, RET was able to resolve the issue of the mistaken identification. Posters warning about this error (in Spanish) were then created by RET and circulated in Spanish speaking communities by NJPIES. The poster is available from RET (ret@njcc.com).

B. Gastrointestinal syndrome

In cases of gastrointestinal poisoning, we have observed that patients can benefit from having information about the course of symptoms that they can expect. In one case, a young girl who was still repeatedly attempting to vomit was told that the expected final symptom of *Chlorophyllum molybdites* poisoning (a clear diarrhea) had occurred and that she was now "OK." The attempts at vomiting stopped almost immediately, and the child was out of the emergency room in about 20 minutes!

It should not be presumed that the biochemistry is identical in the following cases. In fact, a characteristic of treatment in literature of the mushrooms causing a gastrointestinal syndrome is that it is usually stated that while some possibly toxic compounds may have been found in some mushrooms of a given group, the knowledge is very incomplete; and the known compounds may not be the cause of rapidly appearing gastrointestinal distress. Experiments on animals often involve injecting massive doses of a purified chemical. The human stomach and the human habit of cooking food certainly produce a very different method of intake of mushroom chemicals, as Benjamin points out, and so there seems no reason to be surprised that injections that kill mice do not always correlate to poisoning of humans who eat mushrooms containing the injected chemical

In addition to lack of biological and biochemical knowledge, there are probably taxonomic errors in identification of causative fungi (at least at the species level). Many times the information about poisoning is not from N. America. The poisonous species may not be known from N. America, or they may be reported from N. America due to misidentification. Mushrooms causing poisonings in N. America have certainly been given incorrect names (based on RET's knowledge of *Amanita* alone); however, when a taxonomic error in species naming is NOT accompanied by an error in terms of the supraspecific group associated with a certain toxin, mushroom identification can still support poisoning treatment, although false information about a species goes into, or stays in, the literature.

Note: In some of the following cases, there are symptoms that are not restricted to gastrointestinal ones. For example, the nervous system may also be affected.

1. *Chlorophyllum molybdites*

Note: Poisonings commonly occurring in region of interest. In wet, warm weather, the species is common on lawns of homes and in open areas such as parks.

The single species causing the most NJPIES calls to RET relating to symptomatic patients is *Chlorophyllum molybdites*. This is a mushroom well worth knowing well. It is large and stately and appears prominently in lawns and other open spaces in rainy periods of summer and early autumn in E N. America. It is morphologically very similar to edible species of *Macrolepiota*. It is dominantly white, and the cap appears to have "flakes of oat meal" distributed over it (fragments of the cap's skin that has split during

cap expansion). The central umbo of the cap becomes somewhat brownish with age. The flesh stains yellow to yellow-orange when cut and eventually becomes brown; when the stem is cut across, the flesh shows the staining reaction but the central cylinder of the stem does not. Most distinctively, the spore print of the species is "olive green"; and, in mature specimens, the gills take on a strong "dirty green" tint.

When this mushroom is ingested for food, it has usually been mistaken for an edible species of *Macrolepiota* or *Chlorophyllum* (the term "Lepiota" or "parasol mushroom" may be used by the patient) or for an edible species of *Agaricus.* As in other case noted herein, it is valuable to know (or be able to look up) common names in language of those many countries with recent emigrants living in New Jersey. In the case of *Chlorophyllum* (we have experience the utility of having access to lists of common names for edible *Lepiota spp.* in French and Russian).

Ammirati *et al.* (1985, p. 282) provide the following account of progress of a poisoning by *C. molybdites*: "The symptoms...usually begin 1-2 hours after ingestion.... Feelings of queasiness and thirst usually develop first, followed by mental haziness, nausea, cold sweats alternating with chills, and intervals of vomiting for 4-5 hours; the victim finally has an attack of copious, watery, or sometimes bloody diarrhea, which persists from several hours to a few days. The degree of abdominal pain varies from mild to intense. Most victims recover within a day or two. ...[T]he only fatality caused by this species in North America [was] a 2-year-old girl [who] died about 17 hours after she had eaten an undetermined amount of the raw mushroom. [This death could be the result of electrolyte imbalance (Marilyn Shaw, pers. comm.).] Reports indicate that humans can be poisoned by either raw or cooked [*C.*] *molybdites*."

The taxonomy of the genus *Chlorophyllum* is discussed in detail in a recent paper by Dr. Else Vellinga ("Bibliography" on page 20).

The species *C. rachodes* is widely eaten although some individuals report gastroenteritis. Some Asian species of *Chlorophyllum* are reported as edible.

Cause of poisoning in humans unknown as of 1995.

2. The *Russulaceae*

Note: Poisoning frequency data for the area of interest is unknown. The family is extremely common in the region of interest.

The *Russulaceae* include fungi with a variety of forms of fruiting bodies. Here we consider the taxa with the gilled-mushroom form. Dr. Bart Buyck, a contemporary expert in the *Russulaceae* believes that there is little reason to maintain a separate genus for *Russulaceae* that exude a "latex"; for the moment, all the available literature makes this separation. Therefore, the two generic names are used here.

Members of the *Russulaceae* are determinable as such even when cooked because of the unusual structure of the tissues of the fruiting bodies—distributed throughout the tissues (including cap, stem, and gills) are spherical clusters of roughly spherical cells. Under a microscope, these clusters appear as "rosettes." Spores are also very distinctive, having an inamyloid surface decorated by raised, amyloid projections, lines, and networks. The variety of forms of decoration of spores is very large and is employed in specialist determination of taxa. Amyloidity of the spores can be seen on a spore print (on aluminum foil, glass, etc., not on paper) without a microscope.

Within the family, the caps range from white to brightly colored (all colors of the spectrum can be present). Sometimes the colors of the cap can be mixed or very variable. In mixed color caps, the colors may blur into one another or be arranged in concentric zones. In many cases, the caps have a matte appearance. Across the family, cap size can range from smaller than a quarter to larger than a dinner plate. The stem is often suggestive of a piece of chalk, with or without stains or dots of color. Sometimes the stem is completely colored. The fruiting body is exceptionally fragile because of the microscopic structure of the flesh. It is generally believed that the bitter, acidic, or hot tasting species include the species that cause gastrointestinal problems.

Determination to species can be very difficult without extensive, up-to-date literature; a microscope; and a selection of reagents that are specific to the family; however, determinations to species are usually not necessary in poisoning cases.

Russula species. The gills and flesh do not exude latex when cut.

Lactarius species. The gills and flesh exude at least some latex when cut. The latex can range from colorless to brightly colored. Some times the gills or flesh will change color in an area from which latex has been exuded.

Benjamin (pp. 364, 369) reports that at least one group of causes for poisoning by the *Russulaceae* comprises sesquiterpenes. Mutagenic compounds are reported in some species of *Lactarius*. (Benjamin, p. 365)

3. *Agaricus* species

Note: Poisoning frequency data for the area of interest is unknown. The genus is common in the region.

The species of *Agaricus* that are of concern are those with an odor of ink or of phenol and those which stain yellow when the flesh is bruised, scraped, or cut. In addition to containing one or more toxins, yellow-staining species of *Agaricus* tend to concentrate heavy metals—another reason to avoid them. Odors such as "anise" are not thought to be indicators of a toxic *Agaricus. Agaricus arvensis*, which is widely considered edible, stains yellow.

Cause of poisoning in humans unknown as of 1995.

4. *Armillaria mellea* "complex"

Note: Poisoning frequency data for the area of interest is unknown. The genus is common in the region.

This complex has been demonstrated to include a number of distinct species in N. America. The confusing picture of edibility in the group seems to be related to a mixture of edible and inedible species. Some of the species cannot be easily identified even with molecular methods.

Species growing on pine and eucalyptus are said to be toxic.

Some species are liable to being parasitized by *Entoloma abortivum*, in which cases, the fruiting body is reduced to an irregular lump, more or less (sometimes the suggestion of a stem or cap can be seen). See *"Entoloma species" on page 11*.

Cause of poisoning in humans unknown as of 1995.

5. Red-pored species of *Boletus*

Note: Poisoning frequency data for the area of interest is unknown. Taxa of the *Boletaceae* occur very commonly in the region.

Once again, the group is poorly known; the mixture of toxins is not known; the literature is scanty; and at least one species is noted as an exception to the general rule that red-pored species are toxic. At least one death attributable to *Boletus pulcherrimus* (=*B. eastwoodiae*) has been reported; an autopsy showed "mid-gut ischemia (reduction of blood supply) and infarction (death of tissue from lack of blood supply)." (Benjamin, p. 360)

In the New Jersey Mycological Association, members are taught to avoid blue-staining species of *Boletus* and closely related taxa (in the *Boletaceae*)—in addition to red-pored boletes. Bitter-tasting boletes are also avoided—such as several *Tylopilus spp.*

Cause of poisoning in humans unknown as of 1995. A toxic substance from boletes named bolesatine is understood biologically and biochemically to some degree; however, based on current knowledge it is unlikely to be the cause of rapid onset gastrointestinal distress. (Benjamin, p. 360)

6. *Entoloma* species

Note: Poisoning frequency data for the area of interest is unknown. The genus is common in the region.

"Generally, symptoms develop in one-half to two hours after the meal and are characterized by vomiting, diarrhea, and headache...[that] may persist for up to two days." (Benjamin, p. 362)

See "Armillaria mellea "complex"" on page 11.

A number of taxa in this pink-spored genus are reputed to be toxic. The fact that *E. sinuatum* is the species most commonly reported as causing poisonings may be because it is one of the few larger entolomas present in field guides.

Benjamin (p. 362) notes that (1) choline, muscarine, and muscaridine are some of the toxins responsible for poisonings by a Japanese species and (2) a hemolysin has been reported for some species, but it would be destroyed by cooking.

Complete list of causes of poisoning in humans unknown as of 1995.

7. *Hebeloma* species

Note: Frequency of poisoning in the region is unknown. The genus is rather common in the region.

Many of the taxa in this brown-spored genus are reputed to be toxic; however, in Mexico, at least one taxon is regularly collected for personal use and for the market by some indigenous peoples. RET has eaten the Mexican mushroom with no ill effect whatever. This may be an indicator of a potential public health hazard if immigrants to the U.S. and Canada attempt to collect and eat a species of *Hebeloma*. A poisoning would be most likely to happen if a person who was not fully endowed with the traditional knowledge of his/her region attempted to reproduce a "Mexican" meal in N. America. The elderly women who collect the edible *Hebeloma* make minute distinctions NOT including cap color, which is identical in a species occurring in the same (Mexican) habitat and believed to be poisonous.

Cause of poisoning in humans unknown as of 1995.

8. *Laetiporus* species

Note: Frequency of poisoning in the region is unknown. Mushrooms of the genus occur commonly in the region.

There are five species of *Laetiporus* in the U.S., some possibly unnamed as yet (Banik et al., 1998). The one that has been eaten the most frequently is the brilliant orange and yellow shelf, *L. sulfureus*. RET has eaten this and enjoyed it. It seems limited to hardwoods, particularly to oak. It does not grow on the ground.

Laetiporus sulfureus is said to become poisonous to humans when growing on *Eucalyptus*, and other persons have reported being poisoned by it. However, this may be a correct observation, but not about *L. sulfureus*. The yellow-pored or white-pored material largely from *Eucalyptus* was proposed to be a separate taxon by Banik et al. (1998) on molecular and habitat grounds. Therefore, the contribution of the host to the poisonous nature of the putative taxon (called LRG II) is open to question.

Reports that *L. sulfureus* growing on conifers has different toxicity may be incorrect because a probably distinct taxon (called LRG III) apparently occurrs only conifers (Banik et al., 1998) and was the only entity doing so in the study.

LRG IV of Banik et al. was identified by them as *L. cincinnatus* (white pore layer, growing only on the ground with hardwoods, synonymous with *L. sulfureus* var. *semialbinus*). Tom Volk (2001) and Michael Kuo (2005) report this species as edible. This entity occurs in our region of interest.

LRG V has a white pore layer, does not grown on the ground, and has a midwestern distribution according to the limited data of Banik et al.

Banik et al. argue that their LRG I, LRG VI, and LRG VII are not reproductively isolated, always have a yellow pore surface, and are probably representative of the true *L. sulfureus*. This entity occurs in our region of interest.

Benjamin reports a 6-year-old girl in British Columbia exhibiting what sounds like the pantherine syndrome after eating what was claimed to be *L. sulfureus* raw. She recovered completely in 20 hours after her stomach was emptied. Other cases have indicated that toxic properties are not destroyed by cooking. Symptoms include "nausea, vomiting, dizziness, and disorientation." (Benjamin, p. 366)

Phenolethylamine tyramine and its derivatives known as hordenine have been reported in *L. sulfureus* and "could be responsible for the central nervous system manifestations." (Benjamin, p. 366)

Otherwise, cause of poisoning in humans unknown as of 1995.

9. *Omphalotus* species

Note: Poisonings encountered occasionally in region of interest. *Omphalotus illudens* occurs occasionally in the region.

"The onset of symptoms is generally between one and three hours. Nausea and vomiting are the most striking features and are associated with abdominal pain, headache, and a sense of exhaustion, weakness, and dizziness. Some patients have increased sweating and salivation and others complain of a bitter taste in the mouth." (Benjamin, p. 366) Diarrhea may sometimes occur as may a "marginal increase in hepatic enzymes."

Some cases resolve in as little as 18 hours, other patients may have protracted symptoms that almost always are gone in a week. "Rare patients have complained of excessive tiredness for up to a month." (Benjamin, p. 367) More rapid resolution is typical of *O. illudens.* Poisonings seem to be more severe and the symptoms more prolonged with the European *O. olearius*, which also seems to produce muscarinic symptoms not reported for *O. illudens.* The two species apparently contain toxins that are different or are in different concentrations. (Benjamin, p. 367)

The one case of poisoning by *Omphalotus* that was encountered by RET involved a European immigrant or tourist that mistook *Omphalotus illudens* for a chanterelle. This is reported as a common occurrence in *Omphalotus illudens* poisonings.

The genus *Omphalotus* is very interesting taxonomically and biologically (Hughes and Petersen, 1998; Kirchmair et al., 1994; Petersen, undated). The upshot of these studies is that *O. olearius*, and *O. subilludens* (whether or not they are synonymous) do not occur in our region.

The clusters of fruiting bodies are on old stumps. The gills are always reported to be luminescent. RET has seen this only once after sitting with the mushroom in a closed closet in a dark room with a heavy blanket over the mushroom and the observer for 10 or 15 minutes.

The toxin involved is reportedly one of the bioluminescent compounds, illudin S. Benjamin speculates that it may have a primary effect on central nervous system. It is a powerful cytotoxin that has been explored as a cancer treatment. "Other terpenoids may be involved." (Benjamin, p. 367)

10. *Pholiota squarrosa*

Note: Frequency of poisoning unknown in region of interest. The species occurs in the region.

Benjamin (pp. 367-369) notes that a small number of people experience gastrointestinal problems after consuming this fungus.

Cause of poisoning in humans unknown as of 1995.

11. *Scleroderma* species

Note: Frequency of poisoning unknown in region of interest. Several species of the genus occur commonly in the region.

Note: WARNING. Sometimes, the dark centers of *Scleroderma* fruiting bodies are used to adulterate European-originated packages of black truffle pieces. Disreputable suppliers will also dye imperfectly black filler material with black ink.

A veterinary case (pot-bellied pig) of poisoning by *Scleroderma* (?*citrinum*) resulted in death in less than 12 hours (Half Moon Bay, Calif.). Unfortunately, the literature is no longer available online. (Marilyn Shaw, pers. comm.)

See "Scleroderma cepa "rapid rigor" syndrome" on page 18 and "Table 2. Benjamin's list of rarely eaten toxic fungi [not otherwise covered in this summary]" on page 2.

12. *Suillus* species

Note: Frequency of poisoning unknown in region of interest. The genus occurs commonly in the region.

Poisoning by species in this genus appears to be related to failure to peel the slimy skin off the cap before cooking. The onset of watery diarrhea is quite rapid (about 15 minutes after ingestion in a case cited by Benjamin, p. 370). The toxin "whatever it is" is apparently "nonvolatile, heat stable, not readily extracted...by boiling, and can withstand drying."

Cause of poisoning in humans unknown as of 1995.

13. *Tricholoma pardinum* and related species

Note: Frequency of poisoning unknown in region of interest.

Tricholoma pardinum cases are reported from Europe. Benjamin suggests that some species of *Tricholoma* found in the U.S. may produce a similar set of symptoms.

Symptoms develop in 15 mins. to 2 hrs. The mushroom "produces both vomiting and diarrhea, which can be severe." (Benjamin, p. 370)

Rapid recovery is usual, although some patients may complain of lingering symptoms for several days.

Cause of poisoning in humans unknown as of 1995.

C. Muscarine (PSL or SLUDGE) syndrome

Note: Frequency in region of interest is unknown to RET and DS. Species of the two genera most frequently considered to be causes of the muscarine syndrome are extremely common in the region.

"...effects of muscarine develop rapidly, often within 15 to 30 minutes of eating the mushrooms. Certainly by one hour, almost all muscarine-poisoned victims will manifest symptoms." (Benjamin, p. 346)

Muscarine stimulates the parasympathetic nervous system, and this is the cause of the clinical features of the syndrome.

Clinical features of muscarine poisoning include the following (Benjamin, p. 347):

- Onset is 5-30 mins. post ingestion
- perspiration
- salivation
- lacrimation (production of tears)
- nausea, vomiting, and diarrhea
- colicky abdominal pain
- bradycardia
- miosis, blurred vision
- urge to urinate

Benjamin (pp. 347-348) goes to some length to distinguish the muscarine syndrome from the panic reaction. In the latter situation the skin is cold and clammy rather than flushed as in the latter. And, although tears and nausea may be present in the panic reaction, it is further differentiated from muscarine poisoning through the following: "...pupils are dilated as a result of fright and anxiety, and the pulse rate is always elevated, unlike the slow pulse produced by muscarine."

Muscarine was discovered first in *Amanita muscaria* and is present in pharmacologically insignificant amounts in that species and many other mushrooms. Unfortunately, the false belief that muscarine was the active ingredient responsible for what is now called the pantherine syndrome persists. Indeed, there are still those who believe that atropine (a very appropriate treatment for the muscarine syndrome) is appropriate for all mushroom poisons—another unfortunate element of the "muscarine myth." (Benjamin, pp. 305-306)

Muscarine reaches toxic levels in quite a few species of *Clitocybe* and *Inocybe* and a handful of *Omphalotus* taxa (see "Omphalotus species" on page 13). It is suspected, in other mushrooms. The acronymic names for

the syndrome stand for "perspiration, salivation, and lacrimation" (PSL) and "salivation, lacrimation, urination, defecation, gastrointestinal distress, and emesis" (SLUDGE)—which are sets of common symptoms of muscarine poisoning.

Muscarine does not cross the blood-brain barrier; and, consequently, central nervous system manifestations are lacking.

D. Hallucinogenic syndrome (poisoning by psilocybin and other tryptamine derivatives)

Note: Poisonings occasionally encountered in area of interest. Taxa to which the present syndrome are attributed occur naturally in the region and are illegally imported for so-called "recreational" use.

Note: If "hallucinations" are reported with little other detailed information, see also "Pantherine syndrome (poisoning by ibotenic acid and its derivatives such as muscimol)" on page 7 and "Ergotism (poisoning by LSD precursors in Claviceps purpurea a parasite of grains)" on page 17.

"From the clinical standpoint, few specific signs are present other than the unusual behavior of the patient." (Benjamin, p. 329)

The reaction of an individual to the hallucinogen syndrome is variable not only based on dosage, but also on (1) social circumstances and surroundings, (2) psychological make-up and ethnic heritage, (3) previous experience, and (4) method of preparation of the mushrooms. Benjamin provides this list of key clinical features:

- onset rapid, 10 - 30 minutes
- sense of exhilaration, uncontrollable laughter
- hallucinations, mostly visual, involving colors and shapes
- distortion of time sense
- euphoria, introspective and meditative state
- dilated pupils
- confusion, vertigo
- muscular weakness
- increased deep tendon reflexes

Tachycardia is usually only noted in teenagers expressing anxiety and having unpleasant experiences. In general, about half of persons under 25 report frightening and unpleasant experiences. Such experiences are considerably less frequent in reports of older individuals. (Benjamin, p. 329)

"The duration of the effects with the average dose of mushrooms used for 'recreational' purposes is generally from four to five hours. Neither headache nor hangover is common, in contrast to the effects caused by *A. muscaria*.... Moreover, a sense of peace and serenity that lasts for a number of days is not uncommon. This mood may border on euphoria. [Rarely,] flashback phenomena have been described. [But this may be based on a single case.]" (Benjamin p. 332)

Benjamin (p. 326) provides a summary of taxa that are known to cause the hallucinogenic syndrome. The genera involved or suspected are *Psilocybe* and *Panaeolus* (with many taxa in these two genera containing hallucinogens) as well as *Conocybe, Inocybe*, *Gymnopilus* (a bitter or metallic taste is often mentioned for this genus), *Lycoperdon* (reported from Mexico), and *Pluteus* (reported from Germany). See "Table 2. Benjamin's list of rarely eaten toxic fungi [not otherwise covered in this summary]" on page 2.

RET reports a case in which an Italian-American male of about 80 years and living in a retirement community saw a film on television of the collection of wild mushrooms in Italy. He got up from his chair and went onto the communal lawn where he found a very large cluster of mushrooms at the base of a dead tree stump. He brought the mushroom cluster into his kitchen expecting a delicious spaghetti sauce that was soon prepared and poured over a mound of pasta. To his dismay, the taste of the mushrooms was metallic and bitter. Not wanting to waste his meal, he carefully removed as many pieces of mushroom as possible, throwing them out the kitchen window. He then ate the remainder. In a few minutes, he felt very unusual and called a

relative who was related to a doctor. By the time help arrived, 30 minutes after ingestion, he was "stone blind." He reported he could not see and exhibited signs of the hallucinogen syndrome. The doctor rushed to the backyard to gather the mushrooms that had been thrown out the window and brought what he found with a flashlight to the ER. These were duly passed forward to RET. The elderly gentleman was much improved by the time that RET reported that the doctor (via a police relay) had sent fresh orange peels for identification! During the next week, the doctor went back and found a few groups of branching mushroom stems. The few spores still on the stems made it possible to diagnose the poisoning's cause as a cluster of *Gymnopilus*. Identification to species was not possible given the amount of material and the available literature.

III. Onset within 15 minutes of ingesting alcohol

A. Coprine syndrome

Note: Frequency in region of interest unknown. Mushrooms causing the coprine syndrome occur commonly in the region.

The affect is very similar to that of the drug Antabuse (disulfiram), which is used to discourage alcohol consumption. The chemical causing the coprine syndrome is not disulfiram, but "an unusual cyclopropylglutamine" that has a "mechanism of action identical to that of disulfiram." (Benjamin, p. 284)

Benjamin (p. 289) lists the following clinical features of coprine intoxication:

- onset occurring 5-10 mins. after alcohol ingestion by a person who has eaten *Coprinus atramentarius* (common inky cap) or a related species 30 mins. to 3 (7?) days previously
- sensation of warmth, flushing, and possible swelling of the face
- sensation of tingling in arms and legs
- nausea and vomiting
- metallic taste in mouth
- tachycardia and palpitations
- severe headache
- sweating, anxiety, vertigo
- confusion
- hypotension and collapse
- (rarely) cardiac arrhythmia lasting up to three [seven?] days.

The taxa known to produce the coprine syndrome in humans include several in *Coprinus* in the broad sense of the name (a black-spored genus including several taxa commonly eaten in N. America) and *Clitocybe* (a white-spored genus with cap often having a funnel-like shape). Reportedly (Benjamin, p. 284), tested specimens of *Clitocybe* did not contain coprine; hence, the "coprine syndrome" in *Clitocybe* may be caused by another chemical.

IV. Miscellaneous or recently reported syndromes caused by fungi

A. *Paxillus* syndrome (immune hemolytic anemia)

Note: Poisoning frequency unknown in area of interest. Species of *Paxillus* are rather common in the region.

"Hemolytic anemia generally develops in individuals who have eaten *Paxillus involutus* for many years with no ill effect. For reasons presently unclear, a few people produce IgG antibodies to an unidentified antigen in the mushroom. During the course of a subsequent meal, antigen-antibody complexes form, agglutination occurs, complement is fixed, and the red blood cells undergo intravascular hemolysis. The onset of the symptoms is rapid, developing within two hours of the mushroom meal. The initial symptoms include vomiting and diarrhea, abdominal pain, and collapse with hypotension. A rapidly developing anemia, with a rise in indirect bilirubin and free hemoglobin (if the hemolysis is massive), a fall in the level of haptoglobin, and

hemoglobinuria are all part of the syndrome. The usual renal complications may follow, with kidney failure and renal pain." (Benjamin, p. 384)

The immune response described above can be fatal in as little as 3.5 days. While a number of hemolytic compounds in fungi are destroyed by cooking (e.g., in *Amanita rubescens* and its relations), such is not the case with *Paxillus involutus*. Despite its long culinary history in some cultures, the latter should never be eaten.

B. Ergotism (poisoning by LSD precursors in *Claviceps purpurea* a parasite of grains)

Note: If "hallucinations" are reported with little other detailed information, see also "Pantherine syndrome (poisoning by ibotenic acid and its derivatives such as muscimol)" on page 7 and "Hallucinogenic syndrome (poisoning by psilocybin and other tryptamine derivatives)" on page 15.

Note: Ergotism, is not caused by a mushroom as the word is commonly understood in North America. The occurrence of ergotism is extremely rare world wide.

"*Claviceps purpurea* [is] a fungus that infects grains of rye and related grasses. One of the psychoactive components of [the] ergot fungus is the alkaloid ergine (d-lysergic acid amide)....

"...[*Claviceps purpurea* (an Ascomycete),] forms a dark, compact, fungal mass called a sclerotium where [a]...grain would normally develop. One or several of these pelletlike sclerotia can be seen in an infected grain spike, typically extending outward...[at an angle]. When separated from the grain spike, the sclerotia superficially resemble rat droppings (rat pellets). The sclerotia are the source of the potent alkaloids in *Claviceps purpurea*. In late spring, when rye plants are in bloom, the overwintering sclerotia from the previous year's grain crop produce stalked[,]...microscopic,... fungal fruiting bodies." (Armstrong, 1998)

It is the sclerotia getting mixed with grain that is ingested by animals, including humans, and leads to cases of ergotism.

"During the Middle Ages, tens of thousands of people in Europe were afflicted with ergotism, a malady characterized by gangrenous extremities, convulsions, [hallucinations,] ["]madness,["] and death. They ate rye bread [unintentionally adulterated] with ergot fungus containing several peptide alkaloids of the ergotamine group (including ergotamine, ergosine and ergocristine) that affect blood vessels. Since they are potent vasoconstrictors, these alkaloids can cause gangrene if ingested in sufficient dosages. Between 990 and 1129, more than 50,000 people died of this disease in France. The disease became so devastating that in 1093 in southern France the people formed a [religious] order to take care of the afflicted, and they chose St. Anthony as their patron saint. One of the symptoms of the disease was an intense burning sensation; hence the name St. Anthony's Fire. It wasn't until 1597 (500 years after the first epidemic of ergotism) that physicians finally associated this horrendous disease with the ergot on rye." (Armstrong, 1998)

A World Health Organization web site (Peraica et al. 1999) provides the following:

"Ergot alkaloids are also secondary metabolites of some strains of *Penicillium*, *Aspergillus* and *Rhizopus spp*.

"The ca. 40 ergot alkaloids isolated from *Claviceps* sclerotia can be divided into three groups:

- derivatives of lysergic acid (e.g. ergotamine and ergocristine);
- derivatives of isolysergic acid (e.g. ergotaminine);
- derivatives of dimethylergoline (clavines, e.g. agroclavine).

"The source of the ergot strongly influences the type of alkaloids present, as well as the clinical picture of ergotism. *Claviceps purpurea* produces ergotamine-ergocristine alkaloids, which cause the gangrenous form of ergotism because of their vasoconstrictive activity. The initial symptoms are oedema of the legs, with severe pains. Paraesthesias are followed by gangrene at the tendons, with painless demarcation. The last-recorded outbreak of gangrenous ergotism occurred in Ethiopia in 1977-78; 140 persons were affected and the mortality was high (34%).

"The other type of ergotism, a convulsive form related to intoxication with clavine alkaloids from *Claviceps fusiformis* was last seen during 1975 in India when 78 persons were affected. It was characterized by gastrointestinal symptoms (nausea, vomiting and giddiness) followed by effects on the central nervous system (drowsiness, prolonged sleepiness, twitching, convulsions, blindness and paralysis). The onset of symptoms occurred 1-48 hours following exposure; there were no fatalities.

"Ergotism is extremely rare today, primarily because the normal grain cleaning and milling processes remove most of the ergot so that only very low levels of alkaloids remain in the resultant flours. In addition, the alkaloids that are the causative agents of ergotism are relatively labile and are usually destroyed during baking and cooking." (Peraica et al., 1999)

C. *Scleroderma cepa* "rapid rigor" syndrome

Note: Not known from region of interest and, apparently, rare. The genus *Scleroderma* and the species *S. cepa* are both common in the region.

This remarkable reaction to a thick-skinned puffball of the genus *Scleroderma* has rarely been reported. The entire body is reported to become so stiff that the victim cannot be "folded" to be put into an automobile.

In the reported case known to the compilers of this document, a teenager, who was mowing a lawn, picked a fruiting body of *Scleroderma cepa* and ate it raw while working. The onset was very rapid. A complete recovery was reported.

Scleroderma cepa has a white surface and is smaller than or about the size of the last joint of an adult man's thumb. It has no decoration or powder on the surface, the bottom of the fungus becomes red-wine-colored when (in its fresh state) it is rubbed rigorously. Like other species of *Scleroderma*, there is a noticeable skin several mm thick; and the spore mass in the puffball's center becomes dark purple or black as the mushroom matures.

The chemical causes of the present syndrome are unknown. It is possible that the reported case of "rapid rigor" syndrome records an individual's dramatic immune response.

Note: Information on similar cases is solicited.

D. *Auricularia* syndrome = Szechuan restaurant syndrome (easy bruising and excessive bleeding)

Note: Frequency of poisoning in area of interest is unknown, despite a large number of Chinese restaurants that commonly serve one or more species of *Auricularia*. Possibly, excessive bleeding is not associated with a mushroom by persons involved in any cases that occur.

After eating at restaurants serving Wood Ears (*Auricularia sp.*) in their dishes, "a few patients sought treatment because of small, blotchy hemorrhages in the skin. This condition has subsequently been dubbed the Szechwan restaurant syndrome or Szechwan purpura." (Benjamin, p. 380)

Another symptom after the ingestion of Wood Ears is difficulty in stopping a nose bleed or a nick from shaving—excessive bleeding.

E. *Hypholoma fasciculare* syndrome ("fasciculic" acid)

Note: Poisoning frequency in area of interest is unknown. A species often identified as *H. fasciculare* is present in the region.

"A characteristic feature of this poisoning is the long latent period—from 5 to 10 hours—prior to the onset of nausea, vomiting, diarrhea, proteinuria, and possible collapse. Impaired vision and paralysis also have been described in connection with this poisoning. The symptoms gradually improve over the ensuing days. One recorded fatality, caused by a fulminant hepatitis-like disorder, resembled amatoxin poisoning.... However, this patient had eaten a mixture of mushrooms..." thus making placement of blame for the symptoms difficult. (Benjamin, p. 382)

This mushroom is not normally sought on purpose, but might be mistaken for edible species of *Armillaria*.

F. *Tricholoma equestre* syndrome

Note: Not reported from N. America, questioned here and elsewhere. The species in question does not occur in N. America; related taxa do occur in our region.

"*Tricholoma equestre*, as it is known in Europe, has caused poisonings in southwestern France where it is found in sandy soil under pine trees. The kind of poisoning it causes is called rhabdomyolysis (where the iron containing red pigment myoglobin leaks out of muscle cells and into the blood. As myoglobin degrades it produces kidney toxins that, untreated, can lead to kidney failure.) Symptoms in one case in which the mushrooms were eaten at several consecutive meals caused fatigue, muscle weakness (muscles stiffened), myalgia, loss of appetite, mild nausea, and profuse sweating. In most cases there is also a red-brown coloration of the urine." (Lincoff and Tschekunow, 2005)

It is possible that eating the same mushroom repeatedly at consecutive meals is necessary for the syndrome to be generated. This syndrome has not been reported from N. America.

The toxicity of the European species has rapidly and widely been included (whether accepted or questioned) in new European field guides and many publications of European amateur societies. The original study is now questioned in terms of its basic science, including experimental design, by Benjamin (2002).

G. *Pleurocybella porrigens* syndrome

Note: Poisonings not reported in North America. The species is locally common to the N of our region.

"... 'In September and October, 2004, an outbreak of encephalopathy of unknown etiology occurred in certain areas of Japan…These patients had a history of chronic renal failure, most of them had undergone hemodialysis, and also had a history of eating Sugihiratake (*Pleurocybella porrigens*), an autumn [mushroom] without known toxicity. Each patient had a history of eating the mushroom within 2-3 weeks of the onset of neurological symptoms…The onset was subacute; the initial symptoms were tremor…weakness of the extremities, consciousness disturbance and intractable seizures…Three to eight days after onset, however, conspicuous lesions appeared in (the cerebral cortex area of the brain)…Of ten cases studied, three patients died at 13, 14 and 29 days after onset.' The other 7 recovered, but only 3 recovered completely, the others showing different symptoms lingering for different periods of time.

"In Japan, in 2004, it was hotter than usual, it rained a lot in August, the Pleurocybella porrigens came up in early September, earlier than usual, and were both very abundant and twice their usual size. Individual caps had become the size of an adult human palm. The mushrooms grow in stumps in pine and cedar trees. By early November, scattered over 8 prefectures in Japan, there were 46 cases of brain illnesses from these mushrooms, and 14 deaths.

"No poisonings from this mushroom have been reported in the U.S. or anywhere else, to date. A pre-existing kidney condition appears to be required. Brain lesions appear to be the proximate cause of death." (Lincoff and Tschekunow, 2005)

This syndrome has only been reported from Japan.

H. *Clitocybe amoenolens* & *Clitocybe acromelalga* syndromes

Note: Poisoning frequency unknown in the area of interest. See below for potentially relevant local species.

"The poisoning report "said, in part: 'Seven cases (of *Clitocybe amoenolens* poisoning) observed and followed over 4 years are reported. All ill patients had eaten the same mushroom species, gathered in the same French alpine valley. Clinical features of erythromelalgia were observed. This syndrome was first described in Japan after *Clitocybe acromelalga* ingestion. It had never been observed in Europe before.' Erythromelalgia is a maldistribution of blood flow with some areas not getting enough blood and calling for more. Extra blood gets through other open vessels… This continues until the appearance of the skin shows too much blood flow…The skin, especially of the hands and feet, appears and remains bright red and feels warm to hot to the touch, and these symptoms are painful. Patients avoid warm weather, some need to have their legs elevated for extended periods of time, and some are confined to bed. Symptoms can last for months. All digits

can be affected. Even the tip of the nose can be affected. No specific treatment is known to be effective. Pain relievers, such as aspirin, or aspirin-free analgesics, are taken as needed.

"This mushroom is not known to exist in the U.S., but it and closely related species that can cause this kind of poisoning might very well occur here.... For our area, besides looking like the false chanterelle, *Hygrophoropsis aurantiaca*, [*C. amoenolens*] also looks somewhat like *Clitocybe gibba*, except that *Clitocybe amoenolens* is much more colorful (with orange or rusty pigments) and distinctly fragrant (perfume like)." (Lincoff and Tschekunow, 2005)

I. Molds

"Of course, when fungi elaborate mycotoxins that are useful, we call them antibiotics." (Benjamin, p. 59.)

Note: The taxa in this group are not usually classed as "mushrooms" in North America.

The following summarizes Turner and Szczawinski (1991, pp. 65-66):

Microscopic fungi commonly known as molds can sometimes contaminate food and cause poisoning. Contaminated forage and grain have caused illness and death of thousands of cattle, horses, swine and poultry. *Penicillium*, *Aspergillus* and *Monascus* have been incriminated. The mycotoxins are varied in structure and belong to several chemical groups.

A common mold, *Aspergillus flavus*, on peanut meal was responsible in 1961 for the death of thousands of turkeys in Britain. The mycotoxin is known as aflatoxin which causes tumors of the liver. Human health problems around the world from aflatoxin have not been directly proven, but improved harvest and storage procedures have reduced the risks of mycotoxicosis in humans.

Food may contain mycotoxins even if the food is not visibly moldy. Milk and meat may contain mycotoxins if the animals have eaten contaminated feed.

The spores of many molds can produce an allergic respiratory condition in humans and other animals. Some *Fusarium spp*. are known to cause this problem.

Note: Also see Peraica et al. (1999). The latter work is available on a World Health Organization website and goes into much more detail.

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