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# AMATOXIN MUSHROOM POISONING IN NORTH AMERICA 2015-2016

By Michael W. Beug: Chair, NAMA Toxicology Committee

Assessing the degree of amatoxin mushroom poisoning in North America is very challenging. Understanding the potential for various treatment practices is even more daunting. Although I have been studying mushroom poisoning for 45 years now, my own views on potential best treatment practices are still evolving. While my training in enzyme kinetics helps me understand the literature about amatoxin poisoning treatments, my lack of medical training limits me. Fortunately, critical comments from six different medical doctors have been incorporated in this article. All six, each concerned about different aspects in early drafts, returned me to the peer reviewed scientific literature for additional reading.

There remains no known specific antidote for amatoxin poisoning. There have not been any gold standard double-blind placebo controlled studies. There never can be. When dealing with a potentially deadly poisoning (where in many non-western countries the amatoxin fatality rate exceeds 50%) treating of half of all poisoning patients with a placebo would be unethical. Using amatoxins on large animals to test new treatments (theoretically a great alternative) has ethical constraints on the experimental design that would most likely obscure the answers researchers sought. We must thus make our best judgement based on analysis of past cases. Although that number is now large enough that we can make some good assumptions, differences of interpretation will continue. Nonetheless, we may be on the cusp of reaching some agreement. Towards that end, I have contacted several Poison Centers and NAMA will be working with the Centers for Disease Control (CDC). Dr. Denis Benjamin is taking the lead for NAMA in this endeavor.

Even though I am aware of more than 20 human amatoxin poisonings in 2016, we have not received a single complete report filed for a human poisoning from amatoxin and only a few reports for dogs. Nevertheless, we have gathered some useful information from the scientific literature, the press, NAMA toxicology identifiers, and medical professionals experienced in treatment of amatoxin poisoning.

Currently, we eagerly await the results of the open clinical trial “Intravenous Milk Thistle (Silibinin-Legalon) for Hepatic Failure Induced by Amatoxin/*Amanita* Mushroom Poisoning.” The trial, supervised by Dr. Todd Mitchell, involves use of Legalon-SIL®, an injectable form of milk thistle extract, *Silybum marianum* (L.) Gaertn. As best I can determine, Legalon-SIL® contains a complex of silibinin and the disodium salt of succinic acid. The trial was approved in June 2009, with the first recruited patient treated that October. Although initially scheduled to end by December 2016, the trial is still recruiting patients. The original estimated enrollment was for 50 patients, which number has, by my tracking, now surpassed 90. With the enrollment of 15 new participants in 2016, the trial appears to be gaining momentum. I recommend that the trial be contacted immediately about any new case in order to enroll the patient quickly by calling 1-866-520-4412 or using the websites legalonsil.com or http://www.clinicaltrials.gov/ct2/show/study/NCT00915681.

Of cause for concern, Legalon-SIL® has been purchased by Mylan, recently famous for Epipen® and rapidly raising drug prices.

*(Continued p.3)*

# UPCOMING FORAYS & OTHER EVENTS

The events page of *The Mycophile* publicizes forays and events of NAMA affiliated clubs which may be of interest to our members. If you would like to list your club's next big event, contact Dianna Smith, Editor: mycophile@namyco.org.
Include date, location, brief description, link for information, and host organization name.
To post your event on the NAMA website, contact the webmaster: webmaster@namyco.org.

**JULY 7-9: Gulf States Mycological Society 2017 Summer Foray,** Hampton Inn & Suites, 1121 East Frontage drive, Wiggins MS 39577. For information contact David and Patricia Lewis: dandplewis@gmail.com.

**JULY 21-22: West Virginia Mycological Club (WVMC) Shelly Conrad Memorial Foray,** Dry Fork, West Virginia with Gary Lincoff, Walt Sturgeon, Tom Volk and other to be announced mycologists. Registration is available at https://www.wvmushroomclub.net.

**JULY 27-30: Annual NEMF Sam Ristich Foray,** Stratton Mountain, Stratton, Vermont. Gary Lincoff is Chief Mycologist. See www.nemf.org to register.

**AUGUST 10-13: NAMA's Arizona Foray** in the White Mountain conifer forests with Dr. Scott Bates. See https://www.arizonamushroomsociety.org/event-2469863 for updated information.

**SEPTEMBER 1-4: COMA's Clark Rogerson Foray** will be returning to the completely refurbished Camp Hemlocks in Hebron CT. with Gary Lincoff, Bill Yule, Roz Lowen and Leon Shernoff. See www.comafungi.org to register**.**

**SEPTEMBER 7-10: NAMA Northwoods Foray** at Lakewoods Resort, Lake Namakagon, Wisconsin. Registration is full.

**SEPTEMBER 28- OCTOBER 1: WILDACRES 2017 Foray** with mycologist Brandon Matheny of the University of Tennessee and others. For more information about the retreat center see http://www.wildacres.org./ For questions on foray details or to register contact Glenda O'Neal, by email: glendakoneal@yahoo.com, or by phone (423) 863-2742 . The registration form can be downloaded at http://www.namyco.org/events.php. Please date checks for September 28, 2017.

**SEPTEMBER 28- OCTOBER 1**: Missouri Mycological Society (MOMS) at Lake of Ozarks State Park, Kaiser MO. Registration fee $55 members, $65 non-member, high school & college students $40, children 16 and under free. Cabin/barracks $7 per person per night. Camping or hotel on your own. Mycologists will be Denis Benjamin, Michael Kuo and Chris Crabtree. Contact Maxine Stone at VeryMaxine@aol.com.

# IN THIS ISSUE

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Historically silibinin (also called silybin) and various other treatment options have been used for amatoxin poisoning. Silibinin is both the main bioactive component of milk thistle seeds and by far the most bioactive component, thus my focus on this one compound.

Of greatest interest is death from mushrooms. Over 40 years of NAMA records indicate 1-2 deaths per year from amatoxin poisoning. In the majority of the North American cases, the cause of death has been consumption of *Amanita* species in section *Phalloideae*. Species involved have included *Amanita phalloides*, *A. bisporigera*, *A. ocreata*, and other white "Destroying Angels". The principal North American *Amanita* researcher, Dr. Rodham E. Tulloss (pers. comm.), reported four eastern North American incidents since August 2008 involving two as yet undescribed species, *Amanita sturgeonii* Tulloss et al. nom. prov. and *Amanita amerivirosa* Tulloss et al. nom. prov. Amatoxins are not confined to *Amanita* section *Phalloideae* but also occur in species of *Galerina* (most notably *G. marginata* = *G. autumnalis* = *G. venenata*), *Lepiota* (most notably *L. subincarnata* = *L. josserandii*) and possibly *Conocybe filaris*. In a disproportionate number of cases relative to the total population, the mushroom poisoning victim is an immigrant. News reports frequently attribute this to the presence of mushrooms in the United States and Canada that are not present where the immigrant is from. However, *Amanita* species in section *Phalloideae*, deadly *Galerina* species, and deadly *Lepiota* species are present worldwide and annually kill people on every inhabited continent. Indeed, elsewhere the incidence of cases and annual mortality from amatoxin is far higher than it is in North America.

The only human death in our 2015–2016 database is the 2016 autumn death of a young child on Vancouver Island, B.C. after consumption of *Amanita phalloides*. Young children (under age 10) and seniors appear to be more likely to die from amatoxin poisoning than healthy individuals 10 to 60 years of age. In the fall/winter of 2016, there was a rash of *Amanita phalloides* cases including one poisoning from Washington State and several incidents in December involving 14 people in California followed by one additional California case in January of 2017. Most were treated using what newspapers now describe as the "Santa Cruz Protocol" developed by Dr. Todd Mitchell. Because no full reports have been received by NAMA for any of these cases, we have only a partial understanding of the components of the Santa Cruz Protocol. However, NAMA toxicologists in California have been in contact with Dr. Todd Mitchell, and news articles provide additional insight. All of the California victims survived, although three (including a 19-month old child and the child's aunt) required liver transplants. There were other cases of poisoning from the consumption of species in *Amanita* sect. *Phalloideae* in the Midwest and East Coast, but we have no data on those cases. From sketchy news accounts, we understand that most or all of the U.S. patients treated were enrolled in the clinical trial and therefore received intravenous Legalon®SIL as part of the Santa Cruz Protocol.

In over 2,000 retrospectively reviewed amatoxin cases from western Europe, the United States and Canada, the average mortality was 11.58%.[1] The mortality was 10.60% in those cases treated by some form of chemotherapy.[1] From Dr. Denis Benjamin's book, *Mushrooms: Poisons and Panaceas*, my own literature search, and discussions with Dr. Todd Mitchell, I have concluded that the most important single therapy is sustained, aggressive IV hydration sufficient to maintain strong urine output (amatoxin is primarily excreted in the urine). This fluid therapy involves careful correction of water, glucose, electrolyte imbalances, and acid-base status.[2] Correction of altered coagulation factors may also be needed (e.g. administration of vitamin K1 in patients with international normalized ratio > 2.1).[2] The retrospective multidimensional statistical analysis of 2,110 amatoxin poisoning clinical cases published in 2001 indicated that the drugs silibinin, N-acetylcysteine, and putatively ceftazidime (used with silibinin) may be associated with higher rates of patient survival.[1] The multivariate analysis revealed little or no efficacy for Penicillin G, the most frequently utilized chemotherapy, and no benefit was found for thioctic acid or steroids.[3] A retrospective study of 367 patients who were treated using silibinin alone or in combination with penicillin, found a 5.1% death rate in the 118 patients treated with silibinin monotherapy and an 8.8% death rate in 249 patients treated with both silibinin and penicillin (deemed not statistically significant due to the small sample size).[4]

Milk thistle extracts have a long history of use as liver protective agents. A (quick) literature search using the Ebscohost® academic search engine returned a list of over 5,000 peer reviewed silymarin (= silibinin) articles. Narrowing the search to "silymarin + amatoxin" still returned 64 peer reviewed articles. From these, I found that "the evidence is limited, but given the lack of alternative treatments in patients with suspected amatoxin-containing mushroom poisoning and the relatively few adverse effects, (intravenous) silibinin should be considered in some patients."[5] Silibinin is purified from an extract of milk thistle seeds. The fact that Legalon-SIL® is an injectable form of silibinin is important because oral absorption of silibinin is poor, making oral ingestion of milk thistle extract of limited utility. However, one potential route to improving oral absorption is complexing silibinin with phosphatidylcholine (lecithin).[6] Like silibinin, phosphatidylcholine is extensively researched (I found over 14,000 peer reviewed articles) and very safe. The complex, known as silipide, is well researched (I found 76 peer-reviewed articles) and commercially available under the trade name Siliphos®. This inexpensive, over-the-counter food supplement (produced by Indena® in Italy) is readily available from many different vitamin companies throughout the U.S. and Canada. Silipide is readily absorbed orally and exhibits high bioactivity (unlike oral silibinin which is absorbed but has only 1/4 to 1/10 the bioactivity of silipide).[3] Both the phosphatidylcholine and the silibinin components have liver protective effects. Indeed, the complexation with phosphatidylcholine improves the targeting of silibinin to the liver and to inflammatory cells.[6] Despite its intriguing potential, silipide has not been tested against amatoxin, and animal tests should come first.

A 2007 review summarizes a long list of findings regarding silymarin's hepatoprotective effects:[7]

- Antioxidation
- Inhibition of lipid peroxidation
- Stimulation of ribosomal RNA polymerase and subsequent protein synthesis, leading to enhanced hepatocyte regeneration
- Enhanced liver detoxification via inhibition of phase I detoxification
- Enhanced glucuronidation and protection from glutathione depletion
- Anti-inflammatory effects, including inhibition of leukotrienes and prostaglandin synthesis, Kupffer cell inhibition, mast cell stabilization, and inhibition of neutrophil migration
- Slowing or even reversing of fibrosis by reduction of the conversion of hepatic stellate cells into myofibroblasts
- Anticarcinogenesis by inhibition of cyclin-dependent kinases and arrest of cancer cell growth
- Silymarin is also found to have immunomodulatory effects on the diseased liver

Silymarin (=silibinin) exerts both important pharmacodynamic and pharmacokinetic effects in the treatment of amatoxin poisoning.[1,8] "Silymarin blocks the interaction of α-amanitin...with cellular components, including basolateral transport systems (thus preventing uptake) and its nuclear receptors (thus preventing inhibition of RNA polymerase II and the concomitant blockade of protein synthesis in the later stages of an amatoxin poisoning)."[9] Consequently, silibinin will have a role throughout the treatment of an amatoxin poisoning and not just during the first 12–24 hours. Pharmacodynamically it acts as an antioxidant preventing liver glutathione depletion. It inhibits production of advanced glycation end products. It promotes membrane stabilization and prevents cell death. It is anti inflammatory.[1] It exhibits DNA dependent RNA polymerase I stimulation promoting liver regeneration. Pharmacokinetically, silymarin not only competes with amatoxins for cell entry (thus reducing uptake), but inhibits P-glycoprotein induced cellular efflux, thus reducing amatoxin recirculation.[1]

Use of activated charcoal has only been associated with improved outcomes when used in the first hour after mushroom ingestion while mushroom material remains in the digestive tract. There is no clinical evidence for improved outcomes from using activated charcoal later in amatoxin poisoning cases.[1] Dr. Denis Benjamin (pers. comm.) notes, "the one theoretical reason to continue with activated charcoal is that it will absorb the toxin that recirculates from the biliary system back into the GI tract. On the other hand, many physicians might prefer to rest the damaged GI tract or stimulate bile flow by avoiding oral intake. There is also no evidence that cathartics provide any significant benefit and could complicate fluid management." A study of 45 French patients suffering *Amanita phalloides* poisoning, found low levels of amatoxin in the GI tract over time. This finding sharply limits any potential benefit from serial dosing with activated charcoal.[10]

A number of extracorporeal purifications have been attempted and discarded as ineffective.[3] The most recent entry into this field is the Molecular Adsorbent Recirculating System (MARS).[11] Most researchers have concluded that for these treatments to be useful, they must be started early.[12] None of these extracorporeal treatments have gained traction in North America. Consistent with pharmacokinetic data, there is broad agreement that by the time any extracorporeal treatments could be initiated, serum levels of amatoxin are already very low.[10]

In a small number of cases, attempts have been made to drain the bile directly, thus removing any retained amatoxin from the biliary tract. Dr. Kent Olson MD, Medical Director at the San Francisco Poison Control System, cautions, "We do not support the use of biliary drainage. It is invasive and the scattered reports of success are anecdotal. By the time the patient presents for treatment, it is likely that the amatoxin has already been excreted. We had a recent case where biliary drainage did not prevent the need for a liver transplant." In a study of four pigs receiving α-amanitin (two at 0.35mg/kg and two at 0.15mg/kg), researchers found that α-amanitin almost completely disappeared from systemic and enterohepatic circulation within 24 hours.[13] The authors concluded that since pig and human amatoxin responses are similar, biliary drainage would be ineffective more than 24 hours post ingestion.

A tantalizing discovery that substantial amounts of amatoxin can remain in the gall bladder more than 72 hours post ingestion comes from one 2006 Missouri case.[14] Three 18-year-old boys consumed varying amounts of *Amanita bisporigera*. One boy consumed 11 raw mushrooms between 6 PM and midnight, awaking at 5 AM with severe nausea, vomiting, and abdominal pain. Within 72 hours, he had developed severe liver dysfunction. He was listed for liver transplantation and aggressive treatment was initiated. Treatment included nasobiliary drainage. Over a 3-day period, 240 mL of bile containing 4 mg of amatoxin was collected. He survived without needing a liver transplant. To put this survival story in perspective, consider that consumption of just one *Amanita bisporigera* is widely considered lethal in an adult human.

I am only aware of one other publication describing biliary drainage. The publication is in German, but an English translation is available.[15] Three males (29, 43, and 50 years) consumed 300 g of *Amanita phalloides* they had collected. Ten to twelve hours later they began to suffer abdominal cramping and diarrhea, presenting at the hospital 26 hours post ingestion. At 48 hours post ingestion, acute liver injury was evident and all three received percutaneous drainage of the gall bladder, IV hydration and IV silibinin (for 5 days) during which no food was given by mouth. Recovery of amanitin from the gall bladder was 0.5 mg in patient one, 0.237 mg in patient number two and 0.135 mg in patient number three. All survived.

In a personal communication, Dr. Denis Benjamin noted, "The biliary drainage issue is a tricky one to deal with. The easiest is percutaneous drainage, and this would have to be maintained for a number of days with a catheter. It is not without complications." Dr. James Addison adds, "I think the question of biliary drainage is worth considering, although it is not something that is not without potential adverse consequences. Thus, I agree with Dr. Benjamin that it would perhaps best be reserved for patients who seem to have a more serious ingestion. Also, there are many fairly sophisticated centers where this may not be available in a timely way."

My own hope is that biliary drainage might be a way to treat patients who do not qualify for a transplant as well as a way to reduce the need for a liver transplant in extreme cases.

Liver transplantation will at times be required in spite of all other efforts. The challenge is to determine when a liver transplant is going to be required and to make that determination early enough that a liver can be located before the patient has died or is so far gone that they will die soon after a transplant. Several authors have proposed criteria for making this decision.[16,17,18] Factors like the amount of amatoxin in the urine and levels of liver enzymes have little correlation with outcomes. For amatoxin poisoning, an early indicator (50% probability) that a patient may require a liver transplant is diarrhea onset less than eight hours post ingestion.[14] On this matter, Dr. Denis Benjamin (pers. comm.) noted "while it is evident that the early onset of severe diarrhea (less than 8 hours) is most suggestive of severe poisoning, the 50% probability ... of needing a transplant is no better than tossing a coin. It may be prudent ... that physicians notify a transplant center in such cases, just to make them aware - a kind of "heads up". However, ... aggressive supportive care and excellent fluid management, together with intravenous silibinin (should be pursued first). The danger is transferring a patient to a liver transplant center too early or too late. This is a very fine line. It requires good clinical judgement and very good communication between the caregivers and cannot be reduced to any simple rubric."

Dogs succumb to amatoxins more rapidly than humans and so treatments employed without any delay are critical. As with humans, use of IV fluids to maintain a strong urine output is of extreme importance. Drainage of the bile duct also is promising, but mainly anecdotal reports on the efficacy of this procedure exist at this point. Many vets who are experienced in treating amatoxin poisonings already use a range of liver protectants and there is great interest in IV silibinin, which is currently not available for dogs. Potential use of a complex of silibinin with phosphatidylcholine (lecithin) is intriguing, although yet untested in any animal model. The complex, known as silipide, is available under the trade name Siliphos®. It has four to ten times better oral bioavailability than pure silibinin, is inexpensive, and can be on the shelf, ready for immediate emergency use. For IV silymarin, the LD50 in beagles was 300mg/kg. The recommended dose of intravenous silibinin for amatoxin poisoning is 20 (up to 50) mg/kg/day. Since Siliphos® is untested, no recommendation can be made at this time.

An important question remains. When a patient presents at a hospital complaining of severe diarrhea, how do the physicians determine that they may be dealing with an amatoxin poisoning? If it is known or suspected that a mushroom ingestion has occurred (and even if mushrooms are not mentioned), a critical question then becomes how long has it been since a meal was consumed? If the delay between the last meal and diarrhea is 6 hours or more, it is highly likely that amatoxins are involved. In poisoning cases, NAMA stands ready to assist in mushroom identification. We have a team of over 150 identifiers in North America. They can usually (though not always) identify the species through examination of the mushrooms involved (or less ideally, identification from photos) or examination of food remains for telltale spores. We offer the same services for animal poisonings. The NAMA website also has descriptions (with photos) of the major mushroom poisoning syndromes. Additional pages are devoted to animal poisonings. Patients (or pet owners) can review the photos, looking for similarities to mushroom(s) thought to be involved. Physicians and veterinarians can review the syndromes and see how the suite of symptoms for known syndromes compare to the suite of symptoms in their case. The amatoxin poisoning syndrome is very distinctive. Most doctors dealing with an amatoxin poisoning see only one case in their life.

Physicians should not wait for confirmation that they indeed have an amatoxin case before beginning treatment. In all cases of severe diarrhea, fluid loss can damage the kidneys (and liver) and IV fluids are called for. In response to my comments in an early draft of this article, Dr. Kent Olson replied, "I do agree that some deaths result from inadequate fluid resuscitation, as most treating doctors do not appreciate how many liters

of fluid the victim has lost". Blood work to check liver enzyme levels is also important. If liver enzyme levels are elevated, OR if amatoxin poison is suspected, the IV silibinin clinical study should be contacted immediately. A repeat of the blood work the next day is an important step. If the liver enzyme levels have remained normal, it is a false positive and the patient can be considered for discharge.

In conclusion, with good hospital supportive care alone, the majority (80-90%) of human amatoxin poisonings spontaneously resolve. Use of IV-silibinin is strongly associated with improved (up to 94%) survival. Only the most severe cases currently require a liver transplant. Indeed, I believe that emphasis on IV fluid therapies, elimination of use of Penicillin and other treatments where efficacy has not been demonstrated, use of IV silibinin, and possibly in some extreme cases, use of nasobiliary drainage, hospitals will be able to reduce the need for transplantation in amatoxin poisoning cases. If for some reason IV-silibinin does not remain available, the use of oral silipide may be promising. The challenge is that in the absence of experimental data, it would be very difficult to prove whether use of silipide makes any difference in survival.

Acknowledgments

I want to thank Dr. Denis Benjamin (and many others) for their careful reading of successive drafts of this article and their insightful medical comments. I also want to thank Dr. Todd Mitchell for his advice as I have thought about amatoxin poisoning treatments. In the end, the opinions herein are my own.

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## NAMA Nominations for Treasurer and Second Vice President

**Current terms for the offices of Treasurer and Second Vice President expire at the end of 2017. The NAMA Nominating Committee is seeking nominees for these Board positions, each to serve a three-year term, 2018 – 2020. Any NAMA member – including you – may be nominated. These officers will be elected at the upcoming September meeting of the Board of Trustees.**

**Please send the nominee's contact information and a brief bio by email to Kathy Yerich: vicepresident1@namyco.org, or by mail: 932 15th Avenue SE. Forest Lake, MN 55025.**

**Submissions due on or before Aug. 25, 2017.**

# Remembering NAMA Member Marek Turowski

By Ron Spinosa

Marek Turowski, one of our most active and enthusiastic members, passed away on April 13, 2017 after over a year of treatment for lung cancer. His passing is a great loss for our club. Marek was a MMS board member, and he was honored at our last banquet by receiving MMS's highest award, The 2016 Golden Chanterelle Award, for his outstanding service to our organization.

Marek was my dear mushroom buddy. Some of my happiest times were the many hours in the woods that Marek and I shared. He was the most passionate mushroom hunter I have ever known. It all began in Poland, when he would go mushroom hunting with his father. Poles are among the most ardent mushroom lovers on the planet! Marek shared tales about his father collecting and preparing Boletes, Chanterelles, green Russulas and other edible species. It was interesting to learn that his father even consumed *Paxillus involutus* and *Gyromitra esculenta*, species that we would never use for the table. They had a special way of preparing them. In those days eastern European folk had eaten those two species for centuries with impunity. Of course we now know That *Paxillus* and *Gyromitra* contain potentially dangerous toxins and should be avoided.

Besides the thrill of the hunt, Marek enjoyed eating a huge number of mushroom species. He was an adventuresome mycophagist indeed! I remember a time when we encountered an incredible number of *Tricholoma populinum* surrounding a big old cottonwood tree. One common name for that *Tricholoma* species is "the Sandy Mushroom" It is certainly aptly named—they were thoroughly encrusted with sand. Marek wanted to try them, and we spent hours cleaning them. The verdict? Not too bad—but definitely not choice. Since we had so many, Marek thought they would be better pickled, so he took them home and put in more hours of preparation for the pickling. I still have a jar of them in my fridge. Yes, they are better pickled! We are fortunate that there is a YouTube video of Marek talking about preparing and pickling Chanterelles. It can be found here: https://www.youtube.com/watch?v=YQkUmuHPk60

Although Marek was crazy about edible mushrooms, his mycological scope went way beyond the frying pan. He was keen to learn to identify as many mushrooms as possible, even obscure LBMs. He had a knack for quickly learning to ID mushrooms, and he was quite eager to share his knowledge with new members of MMS. He served as Co-chair of the MMS ID Committee and attended most MMS forays to help identify the finds and teach others in the field.

Marek originally came to the USA to study physics at the University of Wisconsin. He completed his PhD degree in Solid State Physics and worked many years for various tech firms doing work in metallurgy. Then as he was approaching 60, he was laid off from his job. As you probably can surmise, even with a PhD, it was not easy for a man of his age to find job. But Marek was not too proud to take a job as a truck driver for a Minneapolis Polish food store. He made many round trips to Chicago to transport Polish imports. On his trips he would often stop at rest areas to check for mushrooms.

The man of science was also a man of deep faith. Marek was a devout catholic and a vital member of the Holy Cross church in North East Minneapolis. This church is home to a large Polish American Community. The catholic Masses there are delivered in the Polish language, as was his funeral service. Several MMS members attended Marek's memorial service at Holy Cross church. We did not understand what was being said, but the language of love, generated by the choir and community, was wonderfully palpable.

Marek had a deep booming bass voice, and he loved to sing. He was active in church liturgy as a lector and psalm singer. In addition he was a member of the inspirational Topola choir group, which performed songs from the Polish cultural tradition. Marek generously committed much time and energy to the Polish community, and they in turn provided loving care and support to Marek, especially during his illness.

My walks in the woods will not be the same without the company of Marek. MMS has lost one of its finest. One of his last wishes was to attend the annual NAMA foray, being held in Cable, Wisconsin this September. As a means of honoring Marek's legacy, the MMS board has created the Marek Turowski Scholarship, which will be awarded annually to an MMS club member who may not have had the opportunity or means to attend NAMA's annual foray. By paying for the foray fees, our hope is that it will broaden that person's knowledge and appreciation of mycology and introduce them to the larger mycological community. May Marek's memory live long in the Minnesota Mycological Society and NAMA.

# Mushroom Poisoning in North America

# Summary of Voluntary Reporting and News Articles for 2015 and 2016

**By Michael W. Beug**

**Chair NAMA Toxicology Committee**

A combination of patient confidentiality laws and expanded use of the internet for mushroom poisoning information has led to a decrease in the reporting of mushroom poisoning cases. As an organization, NAMA is engaged in discussions with Poison Centers across North America and with the Centers for Disease Control in an effort to improve reporting. Our hope is that all poison centers, doctors, veterinarians, and NAMA members will encourage anyone involved in a mushroom poisoning to file a report with NAMA. I believe that in the past NAMA has received reports on about 10% of all mushroom poisoning cases but feel that current reporting has fallen well below that 10% level. Never the less, we can gather some useful information from the data we have at hand. I have divided the material into three groups – poisoning by known toxic mushrooms, adverse reactions to commonly edible species and poisoning of animals (only dogs these past two years). Incidents are placed in alphabetical order by species of mushroom.

**Human poisoning from known toxic species**

Some *Agaricus* species can cause mild flu-like symptoms including vomiting and diarrhea. Many *Agaricus* species are hard to identify even for an expert. In one case a child vomited after consuming part of an unidentified *Agaricus*. What may have been *Agaricus hondensis* caused vomiting and nausea in an adult. Consumption of *Agaricus xanthoderma* (= *xanthodermus*) led to nausea, vomiting, diarrhea and elevated liver function tests (as well as a bill for a Hospital Emergency Room visit).

*Amanita phalloides*, *Amanita bisporigera* and other members of *Amanita* section Phalloideae claimed a number of victims (20 in 2016 alone, with at least one death and at least three liver transplants needed). Since we received no full reports, details are sketchy. These species are covered extensively in a separate article. The important things to know here are that these mushrooms are large, beautiful, delicious and deadly, with toxins that are not destroyed by cooking. If you wish to collect wild mushrooms for the table, it is important to learn to recognize *Amanita* species before consuming any gilled mushroom.

In 2016 we had the first ever report of the effects of consuming *Amanita magniverrucata*. A man consumed roughly one pound (cooked) and after a three hour delay suffered chills, sweating, vomiting and diarrhea. Other *Amanita* cases included one where a man suffered a rash after simply handling *Amanita phalloides*. Another man consumed an *Amanita* in the pantherina group and then felt strange, became agitated, and then vomited and had diarrhea. A couple consumed *Amanita alpinicola*. Both became dizzy and nauseated. One became confused and vomited. A child who suffered a seizure was thought to have eaten *Amanita gemmata*, but no mushroom had been eaten. *Amanita smithiana* was involved in three cases reported by the Oregon Poison Center and two incidents reported by NAMA mushroom poisoning identifiers. The poisonings were very serious, resulting in hospitalization and kidney damage that resolved after several days. In at least four of the cases, the victims reported that they thought that they were picking *Tricholoma murrillianum**, "matsutake". We have the most detail about one of the Washington cases. A midlife man collecting by Mount Rainier had picked what he thought was matsutake. About 10 hours after cooking a meal of the mushrooms, he was not

well. He reported to the hospital with gastrointestinal upset. A NAMA identifier recognized *Amanita smithiana* from the photos. Two toxins associated with *Amanita smithiana* are chlorocrotylglycine and allenic norleucine, though more research is needed to determine precisely the toxic components of this mushroom. The hospital was most appreciative to learn that they should treat for a kidney toxin. Photographs and the ways to tell *Amanita smithiana* and *Tricholoma magnivelare* apart appeared in the first 2017 issue of *The Mycophile*.

*Chlorophyllum molybdites* always claims numerous victims and the past two years were no exception. At least two individuals mistook *Chlorophyllum molybdites* for *Coprinus comatus*. If you ignore the overall shape (broad and squat versus tall and slender), ignore the gill color (white becoming greenish versus pinkish becoming black) and a few other differences (moderately spaced versus crowded gills, meaty versus soft textured, etc.), you too can enjoy a few days of vomiting and diarrhea with the added bonus of chills, headache, salivation and excessive perspiration. Of course other people who consumed this mushroom consumed it because it was growing in their yard and they assumed that anything so delicious looking in their own yard (or sometimes on their golf course) must be there for them to enjoy raw. After all, they are delicious that way. For an hour or two you can be proud of yourself for making such a meaty find. Those people learned that consumed raw, these mushrooms are even more vicious than when consumed cooked. Their vomit and diarrhea was quite bloody. Some mistook *Chlorophyllum molybdites* (gills white but eventually greenish from spores that are initially white but turn green when fully mature) for the highly similar *Chlorophyllum rachodes* (gills white and remaining white since the mature spores remain white). Unless I spore-printed every specimen, I would not eat any of the edible *Chlorophyllum* species except in climates where *C. molybdites* does not grow (e.g. the Pacific Northwest).

*Boletus huronensis* was consumed by one person who suffered vomiting and nausea. We do not know why the mistake was made in this case but do know that *Boletus huronensis*, an eastern species, is sometimes mistaken for the “king bolete”.

*Caloboletus marshii*, a bitter bolete, was consumed by one California woman who had gone to a mushroom field day and returned to her ranch to pick a wide range of mushrooms she found growing there. She had identified the “bitter bolete” as a “butter bolete” and suffered chills, flushing and nausea. I made a similar mistake myself this summer when I collected a sack of blue-staining *Caloboletus marshii* fungi from my oak grove. I sampled a tiny bit raw (and spit it out). I found them to be very sweet and tasty, not at all bitter. I concluded that I might have found a new “butter bolete” species, one that did not have a netted stipe apex. I proceeded to write up a description as well as to cook a small sample batch. I only cook and sample unknown mushrooms from groups where the known similar species are edible or thought to be relatively harmless. After all, the only way to tell whether an unnamed species is edible is to eat some. If a little bit is OK, then try more a day or two later. If still OK, serve them to a few daring individuals. While I would never do this with an unknown mushroom where similar species are toxic, I have done it a few times in the past where I had good reason to anticipate a good outcome. WARNING: DO NOT DO THIS YOURSELF, MY PRACTICE IS ONLY FOR EXPERTS! In this case, after the first extremely bitter taste, I immediately identified my find as *C. marshii*. A few months later, under those same oak trees, I found a blue staining “butter bolete”. Since I had never before eaten a “butter bolete” or any other strongly blue-staining bolete (most are extremely bitter), I cooked up my collection. I was already positive it was *Butyriboletus querciregius*. That collection was delicious!

Consumption of an unidentified *Clitocybe* species led to vomiting. Many *Clitocybe* species have considerable quantities of the toxin muscarine and many are difficult to identify.

Clearly individuals were regularly seeking to get high on hallucinogenic mushrooms, though we rarely hear about it, even when things go badly. When we do get a report, the reason why the mushroom was consumed is rarely in the report. I only uncover it when I contact the individual(s). One couple reported explosive and burning diarrhea after consuming a *Gymnopilus* species. From photos, it appeared to be in the *G. sapineus*

group. When I asked why they would even eat such a very bitter mushroom, they admitted that they had hoped to get high. Indeed, several *Gymnopilus* species are hallucinogenic, notably *Gymnopilus spectabilis*, "big laughing gym". However, one poor soul from the Pacific Northwest discovered that mistaking that for what appeared to me from pictures to be a *Pholiota*, led to days of vomiting. Had he paid more attention, he would have noted that "big laughing gym" should indeed be big and should bruise blue. His mushroom had not stained blue and was not very big. To add insult to injury, our PNW look-alike to *Gymnopilus spectabilis*, *Gymnopilus ventricosus*, is not even hallucinogenic! In another PNW case, a man suffered gastrointestinal distress after consuming a non-staining *Cortinarius* species thinking it was "big laughing gym". Consumption of what was identified as *Cortinarius vanduzerensis* by yet another individual also resulted in an inquiry, but we do not know what the effects were or why the individual consumed this particular slimy *Cortinarius*.

*Lactarius* cf. *luculentus* produced vomiting in one individual.

Consumption of a *Lepiota* similar to *Lepiota cristata* produced a case of gastrointestinal upset. I am particularly concerned about consumption of smallish *Lepiota* species because some contain amatoxins and are thus deadly poisonous.

Consumption of cooked *Leucopaxillus gentianeus* produced a case of stomach cramps and paranoia.

There were four incidents of poisoning by Jack-O-Lantern fungi (*Omphalotus* species) including one incident involving *Omphalotus olivascens* and three incidents (six individuals) involving *Omphalotus illudens*. In all cases it appears that they were mistaken for Chanterelles and the price was severe gastrointestinal distress. When your "Chanterelles" are growing in a large clump, and especially if you note that at night you can read by the light of your "Chanterelles", it is best to assume that you have made a mistake in your identification and consuming the mushrooms will be an enormous mistake, but one from which you will recover – eventually.

Consumption of *Psilocybe* mushrooms as hallucinogens is popular but sometimes can go very wrong. In one case, police were called to an apartment building to deal with a man who was destroying the apartment in his hallucinatory state. He fell to his death from the third floor window while trying to elude the police. In the past, there have been reports of individuals high on psilocybin jumping to their death from tall buildings. In a visit in October of 2016, Paul Stamets told me of reports he has received of individuals suffering temporary paralysis after consuming some of the very potent wood-chip *Psilocybe* species and the beach-grass *Psilocybe*, *P. azurescens*. The concern here is that someone might consume these mushrooms out in the field on a cold, rainy day and suffer hypothermia before they can walk again.

Consumption of a batch of red *Russula* species led to gastro-intestinal distress for two individuals in Colorado. In the South, one person suffered chills, diarrhea and sweating from consumption of a white *Russula*.

*Scleroderma* species were involved in a number of cases. One case involved a child grazing in the yard. In some cases sclerodermas were consumed by people thinking that they were eating a puffball and in other cases people thought that they had found a truffle. In all cases the result was diarrhea and vomiting, often exceptionally severe. One elderly woman who I later met nearly died from the severity of the poisoning. She was one of the people who thought that she had consumed a puffball. Puffballs, when in the edible stage, are uniform pure white inside and are soft like a marshmallow. *Scleroderma* species may be whitish when very young but soon turn purple to black inside and are hard, not soft like a marshmallow. The choice edible truffles are marbled inside. Truffles are tasteless and of no culinary interest until mature. At maturity, the choice species develop an amazing aroma, often of ripe cheese and garlic. *Scleroderma* species are not marbled inside and have either no odor or a disgusting odor depending on species and maturity. In the case of the woman who nearly died from a *Scleroderma*, we collected fresh material from her site as well as a similar *Scleroderm*a from my garden and sent material off for DNA analysis. We learned that the DNA data on *Scleroderma* species is confused and that species concepts will need to be straightened out before names can accurately be

applied.

Handling of *Suillus pungens* caused a rash in one individual.

**Adverse reactions to normally edible species**

Gastrointestinal distress was experienced by one person who consumed each of the following species: *Amanita calyptroderma* (old), *Boletus edulis*, *Cantharellus formosus*, *Coprinellus micaceus* (raw), *Hydnum repandum* (left in trunk for one week before consumption, victim also passed out), *Laccaria ochropurpurea*, *Leccinum insigne*, *Leucoagaricus americanus* (also hallucinations and impaired motor function), *Pleurocybella porrigens*, *Pleurotus* c.f. *ostreatus*, *Ramaria* species (western fall, yellow), *Ramaria* species (western spring, yellow, three victims in one incident), *Sparassis crispa*, *Sparassis radicata* (old), *Sparassis spathulata*, *Suillu*s species (frozen when raw and later cooked, patient also experienced low blood pressure) .

Five cases of gastrointestinal distress resulted from consumption of various *Armillaria* species in the *A. mellea* complex ("honey mushrooms"). Two were definitely *Armillaria solidipes* (= *ostoyae*), a mostly western species that has a record of causing a significant number of adverse reactions. One was definitely *Desarmillaria tabescens* (=*Armillaria tabescens*), an eastern species. One adverse set of symptoms was the result of eating raw honey mushrooms and another was the result of eating old honey mushrooms. Looking back over years of poisoning reports, I find that it is very rare for adverse effects to occur upon consumption of honey mushrooms collected in eastern North America, where they are one of the more popular edible species. However, it is very common for adverse effects from honey mushrooms collected in western North America (where we have different species of honey mushrooms).

One case of consumption of alcohol with a meal of cooked *Coprinus comatus* led to flushing. Since the flushing reaction is normally associated with *Coprinopsis atramentaria* and alcohol rather than *Coprinus comatus*, there is a chance that there is an error either in the filing of the report or in the original identification.

"Hen of the woods", *Grifola frondosa*, was implicated in one case where the person suffered chills, dizziness, sweating, weakness, numb tongue and throat.

*Hypomyces lactifluorum* may have been involved in one hallucinatory event reported to Dr. Joe Ammirati and provides a detailed example of the kind of puzzles the NAMA toxicologists can get involved in unraveling:

> Hi Joe,
>
> Here is everything I can remember about when I ate the lobster mushrooms...
>
> My friends and I were staying at a cabin outside Mount Rainier. I had been feeling a bit sick for several days, so I had been taking a lot of Ibuprofen. At dinner, we had cooked lobster mushrooms and a bit of chanterelles mixed in with our food that we had harvested earlier that day. I had only a few sips of beer that night, and some of our food had been cooked with wine. I was also drinking the tap water.
>
> All of a sudden when I was eating, I felt a rush go through my body from my feet to my head. This rush happened a couple of times. Then, I started to have hallucinations similar to what marijuana would cause. I began to have intense anxiety and paranoia and went into a panic attack and eventual vomiting. It took a couple of hours for me to feel more or less back to normal.
>
> While everything was happening, I thought maybe the mushrooms were causing the effects, but my friends and I could not find any information about lobster mushrooms having any side effects.

I eventually wrote it off as being caused by the possibility of having too much ibuprofen. It's possible it was a mix of those different factors.

One of four people in Florida who jointly consumed a meal of *Laetiporus sulphureus*, "chicken of the woods", experienced chills, hallucinations, dizziness, spasms, vomiting and diarrhea. This suite of symptoms is often associated with the two western chicken of the woods species, *Laetiporus conifericola* and *Laetiporus gilbertsonii*. *Laetiporus gilbertsonii* caused bloody vomiting in one individual. It is worthy of note that the two eastern species, *Laetiporus sulphureus* and *Laetiporus cincinnatus* (currently considered a synonym of *L. sulphureus*), are much tastier edibles than western chicken of the woods.

*Lentinula edodes*, "shiitake", was identified in eight reports. Raw shiitake caused a severe flagellate rash in three individuals, one of whom also suffered chills and flushing. One of the reports mentioned that 10 people shared the raw mushrooms but only one developed the rash, which is consistent with Japanese research that found that fewer than 5% of people who consume raw shiitake develop a rash. The rash is caused by the lentinin in the mushrooms. Lentinin is used in cancer therapy (mainly in Asia). Lentinin is destroyed by cooking and so the rash is rarely seen in people who consumed well-cooked mushrooms. However, light cooking, a practice often seen in restaurants, does not destroy all of the lentinin. Three individuals reported a rash from cooked shiitake. In one case, the rash was accompanied by fever. In another case, the rash was accompanied by diarrhea. Rash onset is usually 2-3 days post ingestion. One restaurant poisoning involved flushing, dizziness, cramps, drowsiness and gastrointestinal distress. In a second restaurant case, the female victim suffered flushing, salivation, dizziness, sweating, disorientation, and muscle spasms. A follow-up revealed that the restaurant had frequently been cited for Listeria (a bacterial contaminant) in the previous five years.

In Hawaii there were two separate cases of purchased *Macrocybe spectabilis*, cooked, causing vomiting. This species, while generally considered edible, contains traces of cyanide that are dissipated on cooking.

As has frequently been the case in the past, *Morchella* species were cited in so many cases that were they not so delicious and so commonly eaten, I would be tempted to call morels poisonous (and have everyone send their morels to me for proper disposal). For most people, morels are definitely poisonous raw or only lightly cooked. In one case, two of five people who consumed some raw morels had chills and diarrhea. In separate incidents, two other people who consumed morels raw experienced vomiting and diarrhea. Two people shared one large cooked morel and suffered gas, stomach upset, and severe diarrhea. Another person cooked a batch of morels, had abdominal cramping, and vomited. Consumption of a meal of cooked "burn morels" produced gastrointestinal distress, hallucinations and dizziness. A Washington State woman who had eaten morels before with no problems had a stomachache three hours after her meal. She went to a hospital. Consumption of a meal reported to be of morels, helvellas and gyromitras led to the person vomiting fifteen times. Another person who consumed "morels and gyromitras" vomited and had abdominal pain.

A couple who consumed an unidentified *Suillus* species suffered gastrointestinal distress, dizziness, weakness and headache.

Five people who consumed *Verpa bohemica*, thinking they were consuming morels suffered adverse effects. I consider *Verpa bohemica* to be an edible species when thoroughly cooked, but one of the edible species where a significant number of people have adverse reactions. It should be consumed with caution and only if well cooked. Like true morels, it is definitely poisonous raw. The toxins in *Verpa bohemica* are unknown (as is also the case with true morels). In my own case, I do not find *Verpa bohemica* to be very tasty, so I stick with morels.

**Animal poisonings**

During 2015 and 2016, all actual animal poisoning reports involved dogs. One 2015 case involving a cat death had Dr. P. Brandon Matheny and me puzzled for weeks. The very unusual cat had a penchant for consuming anything and everything (something common for dogs, but rare in normally discerning cats). Brandon even did DNA work to identify the mushroom involved because neither of us was sure what species we were looking at but neither of us thought that it was likely that the mushroom was toxic either. To add to the puzzle, the reported symptoms did not match any toxic mushroom syndromes. Finally, the owner was able to attribute the poisoning to a non-mushroom toxin. A second cat was thought to have possibly consumed a *Galerina* but we have no idea what happened or whether a mushroom was even involved.

*Agaricus* cf. *meleagris* was consumed by one dog. The dog experienced vomiting and diarrhea.

*Amanita* mushrooms in section Phalloideae (including *A. phalloides*, *A. bisporigera*, and *A. ocreata*) claimed the lives of several dogs. Dogs can rarely be saved when they consume one of these species. In one case, a dog owner contacted Debbie Viess thirty minutes after the dog consumed the mushroom and she identified the mushroom (*Amanita phalloides*). The dog was rushed to a vet experienced with treating amatoxin cases and the dog was saved. One report mentions a dog saved using IV fluids and milk thistle. Another dog survived what appears to have been a possible *Amanita bisporigera* ingestion after experiencing 32 hours of chills, diarrhea, salivation, vomiting and drowsiness.

*Amanita pantherina* and *Amanita muscaria* are frequently eaten by dogs. In one reported case, a dog consumed *Amanita pantherina* and then vomited, became ataxic and could not stand. At least one other dog could not stand after consuming *Amanita pantherina* and another dog vomited. Several other dogs were sickened by *Amanita pantherina*, but we have no details. There was also a case of poisoning from *Amanita gemmata* and one from *Amanita aprica*, but again no details. Of two dogs (two incidents) that consumed *Amanita muscaria*, one died and the other experienced salivation and disorientation. Another dog consumed a mixture of a *Russula* species and *Amanita* species and experienced classical ibotenic acid poisoning symptoms of vomiting, diarrhea, ataxia and disorientation.

A *Calvatia* species was for the first time implicated in a dog poisoning. The dog vomited. In a first report for a *Conocybe albipes* ingestion, a dog experienced vomiting and diarrhea.

A bite or two of *Chlorophyllum molybdites* produced seizure and a drooping head in a dog. In another case, the dog vomited, had diarrhea and appeared weak, disoriented and drowsy.

*Clitocybe* species, many of which contain significant levels of muscarine, caused significant problems for at least three dogs. One suffered violent vomiting, diarrhea and was unable to stand. Another had diarrhea and excessive drooling. A third had what were described as classical muscarinic (PSL) poisoning symptoms. PSL standing for perspiration (though dogs pant and do not perspire), salivation and lachrymation.

Some *Entoloma* mushrooms are notorious for their toxicity, though I have never before had a specific report of a dog poisoning from them. This first case involved a dog with muscarinic syndrome poisoning (PSL) that appeared disoriented and seemed to be hallucinating.

*Inocybe* species, a large number of which have significant levels of muscarine, were involved in several dog poisonings. NAMA is often contacted to identify the mushroom, but we are rarely informed of the symptoms or the outcome. That was certainly the case for some of the *Inocybe* poisonings. One dog had classic muscarine PSL symptoms – panting, salivation and lachrymation accompanied by vomiting and diarrhea. Another simply vomited. However, one dog consumed *Inocybe* cf. *fibrosa*, showed classic PSL symptoms, vomited,

developed a bloody stool, and liver failure and died.

We had a first ever report of a dog consuming *Lysurus cruciatus*. The dog was found vomiting afterwards. Consumption of a *Leucoagaricus* caused another dog to vomit. *Phyllotopsis nidulans*, another mushroom never before implicated in a dog poisoning, caused neurologic problems and liver damage in one dog. Another first ever report involved *Polyporus squamosus*. The dog experienced vomiting and profuse diarrhea, exhibited small pupils, elevated liver enzymes and was unresponsive.

Consumption of *Panaeolina foenisecii* produced nausea and fatigue in one dog.

*Scleroderma* species were involved in several dog-poisoning reports. One dog consumed 1/5 of one mushroom, vomited and was weak. Another dog vomited. A third dog was found drooling, vomited, experienced diarrhea and had an elevated heart rate. We have no details on a fourth and fifth dog (two different cases) at a vet after consuming a *Scleroderma*. *Scleroderma citrinum* produced vomiting and diarrhea in another dog. *Scleroderma* species in the past have led to deaths for both dogs and pigs.

A dog found to have consumed *Stropharia coronaria* vomited and was ataxic.

A dog took bites from a *Suillus* and was found with PSL symptoms and unable to stand. Another dog consumed a *Suillus* and vomited.

Consumption of a *Xerocomellus* species (identified as *Boletus chrysenteron*) caused vomiting in a dog. The vet treated the animal with activated charcoal.

**Summary**: While we have fewer cases and fewer details than ever, we have learned about some new toxic mushrooms. I hope that we can find ways to encourage more reporting so that we can both minimize ingestion of toxic species and improve treatments.

Of interest to some, the species of *Armillaria* that lack an annulus are now in a new genus, *Desarmillaria*. Koch, R.A., A.W. Wilson, O. Séné, T.W. Henkel, and M.C. Aime resolved phylogeny and biogeography of the root pathogen *Armillaria* and its gasteroid relative, *Guyanagaster*. *BMC Evolutionary Biology*, 17:33 (16 pages). Other new genera appear in this report because, for example, the genus *Boletus* has proven to contain diverse lineages of mushrooms. Furthermore, we are learning both about more cryptic species and about more misapplied names. *Xerocomellus chrysenteron* (*Boletus chrysenteron*) is a misapplied name for a group of at least three different species.

* The western matsutake is genetically distinct from the eastern matsutake (*T. magnivelare*) and an old name (*T. murrilianum*) for our western species has been resurrected, however this change is not yet recognized by Index Fungforum which gives *T. murrillianum* as a synonym for *T. magnivelare* while Mycobank gives *T. murrillianum* as a synonym for *T. ponderosum* . (Thanks to Steve Trudell for for coauthoring the recent publication recognizing *T. murrillianum*).

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## RECOMMENDED LINKS TO FUNGI ARTICLES

Rich Malhotra, "Fungus creates zombie beetles that crave flowers before death", *New Scientist* Zoologger, June 9, 2017 www.newscientist.org.

Tom Fisher, "The Ballerina with Fangs: A Cau onary Tale for Foragers" from *Whidbey Life Magazine*, May 31, 2017. https://www.whidbeylifemagazine.org/the-ballerina-with-fangs-a-cautionary-tale-for-foragers/

# President's Message

The NAMA Board of Trustees held a meeting via conference call on Sunday, June 11. Our main topic was a series of Foray Guide proposals brought by Sam Landes, Chair of the Foray Committee.

The most important change is how forays are organized. In the past we've sought out locations where affiliated clubs do most of the site selection, registration, contacting speakers, putting together a program, and organizing forays, while NAMA runs the voucher program and provides guidance and assistance. The benefit to clubs is where the bottom line is profitable, they get 80% of the profit, while NAMA gets 20%. Historically, 9 out of 10 annual forays have used the club hosted model.

With electronic registration forms and online payment – and a Foray Committee chair and a Registrar willing to do the work – NAMA feels we should take more responsibility for running our own event. We felt that this change will encourage affiliated clubs to partner with us in this joint event process.

The first proposal from the Foray Committee was to offer a stipend to defray travel expenses for some presenters; the committee feels this action will allow us to continue to bring in top-notch professionals to do presentations and assist in the identification process. This change could increase registration fees by about $8. However, by limiting the number of presenters and changing the basis for the foray budget, this cost will probably be lower.

Next, we considered a proposal to treat "mandatory" presentations on photography, toxicology, education, cultivation and mycophagy as part of the foray budget, rather than NAMA paying directly. This will add about $5 to registration.

The final item was standardizing foray cancellation fees. In the past, we've had a range of cancellation policies from non-refundable registration, to partial refund up to a certain date, to registrar discretion. The proposal which passed, reads as follows. "NAMA will offer a refund minus a $10 handling fee for cancellations up to 30 days prior to the beginning of the Foray and a full refund minus a $50 handling fee for cancellations 30 days or less prior to the beginning of the Foray. Exceptions (medical, family, work issues) are left up to the registrar who may consult with other Foray Committee members."

Some of these changes affect only the Foray Guide and some will have to be incorporated into the Policy Manual. Thanks again to Sam and the Foray Committee for putting this major policy change together.

## NAMA Needs You!

Several key positions are still open. If you'd like to help NAMA by participating with a committee or leadership role, please contact me at president@namyco.org or call me at 510.468.5014.

## Update on California Death Cap Poisonings

You may have seen news in the past few weeks about a report by the Centers for Disease Control and Prevention (CDC) about *Amanita phalloides* poisonings in Northern California in December 2016. You may recall my message in the January/February 2017 issue of *The Mycophile* on the same topic. I urge you to read the highly detailed report.

A total of 14 people were poisoned in December 2016 by *Amanita phalloides*, which is the highest number that we know of since this aggressively invasive mushroom was introduced to California back in the 1930s. Last fall, with record rains, Death Caps were prolific. While the total poisonings is alarming, two of the incidents involved multiple people. Of the total poisonings, all survived, although three individuals, including an 18 month old toddler, required a liver transplant. One contributing factor for two liver transplantations may have been the time between ingestion and admission to the hospital. One liver recipient waited 64 hours before seeking treatment (at the local Alameda county hospital and trauma center).

The family cohort consisted of the mother, father, daughter, sister, and a family friend; all ate the same meal. According to the CDC report, "The mushrooms had been given to her by a person she did not know, who reportedly picked them earlier in the day in the mountains."

Another cohort, "four men aged 19–22 years who developed hepatotoxicity after ingesting what they thought were psychedelic mushrooms picked from the wild." Yikes! Two cases occurred with women aged 86 and 93 years old who "received wild-picked mushrooms from a friend".

The good news is that the report includes details about the poisonings which we rarely receive (except through the NAMA Poison Case Registry, thanks to Michael Beug, Chair of the Toxicology Committee). We've asked the California Poison Control System (CPCS), source of the CDC report, to provide this level of poisoning information in the future. My local club, BAMS, was credited with making initial alerts to CPCS. We hope to continue the dialogue to work more closely with Poison Control in the future.

By David Rust, NAMA President

# NAMA PHOTO CONTEST

The photo contest is open to all mushroomers. For Pictorial and Documentary divisions, organisms from the myxomycetes (slime molds, which are not true fungi) and the classes Basidiomycetes and Ascomycetes of the Fungi Kngdom are eligible. For Judge's Option, nearly anything goes, so long as the theme relates to fungi, and fungi are a key element of the photograph. Up to 15 images may be entered per person, with a maximum of 6 in the Pictorial, 6 in the Documentary and 3 in the Judges Option to make a total of up to 15 images. **Closing date**: All entries must be received by the Contest Director on or before **August 4, 2017**. Allow at least one week for mailing.

**Entry Divisions:** Limited or Advanced. If you won 1st, 2nd, or 3rd place two or more times previously, then you must enter in the Advanced division. If you won less than twice before you can enter the Limited Division.

**Marking, Listing and Submitting Digitals**: Let us know if you are entering the limited or advanced division. The digital photos file name should include 3 things, D (for Documentary) JO (for Judges Option) or P (for Pictorial), and the photographer's initials, followed by the Genus and species of the fungi or the title for the Judges Option photo. Please also include a photo of yourself and the location the mushroom photo was taken. Digital images may be emailed or mailed on a CD or DVD and will not be returned. If emailing in images please include your name, address and phone number. Images can also be submitted using free file mailing programs such as http://www.mailbigfile.com/ or Dropbox etc.

**Photography Contest Entry**: To enter the NAMA photography contest, mail or email your entries to:
John Plischke III, 411 Center Avenue, Greensburg PA 15601, (724) 832-0271, Email: fungi01@aol.com.

# Knocking Off the Dust – Exposing the Value of Past NAMA Collections

By Stephen Russell, Mycoflora Committee Chair

For 20 years, starting in 1997, the NAMA Voucher Collection Program has documented, photographed, and saved collections of all species from the annual national forays. This program has preserved thousands of collections during this time, covering different regions of the continent. The bulk of these specimens currently reside at the Field Museum in Chicago, Illinois. The general protocol for vouchering NAMA collections has not changed much over the years, until a new component was added in 2015: saving fresh tissue for DNA analysis. As the specimens are being photographed and dried, a small piece of mushroom flesh – about the size of a grain of rice – is being retained. A tiny portion of material will often allow us to accurately confirm the identity of the species by examining its genetic code.

The ongoing NAMA initiatives in this realm are encouraging. More than 100 DNA sequences of specimens were generated from the North Carolina foray at Black Mountain in 2015. These DNA sequences will be added to the public DNA and protein sequence repository (GenBank), aiding researchers across the world who are working to sort out which species occur in North America. This process of tissue collection and genetic sequencing continued at the 2016 Shenandoah Foray in Virginia and is planned for the 2017 North Woods Foray in Wisconsin later this year.

Continuing the current sequencing program at the annual foray will create a valuable database of fungal DNA from across the continent and aid our efforts with the Mycoflora 2.0 Project. A key benefit of these sequences is providing additional reference data for North American specimens - without reference sequences to make comparisons to, a DNA sequence has little value. The more high-quality reference sequence data that exists, the higher the likelihood club-based DNA projects (and professional-led research) have of producing successful outcomes. While current NAMA efforts in DNA focus on new collections at the annual forays, the organization possesses thousands of historical collections at the Field Museum that could also serve an important purpose. Making the DNA data from these specimens available to the public will help to advance the knowledge of fungi worldwide, and is within the scope of what NAMA can achieve.

As a start, the NAMA board recently approved a plan to start sequencing past NAMA vouchers in the family *Amanitaceae*. The *Amanitaceae* contains some of the most charismatic, enigmatic, and culturally significant macrofungi known to exist. The broad interest in this family makes it a natural group to start with for sequencing historical collections. Two more technical reasons for starting with the *Amanitaceae* are the amount of high-quality reference sequences available for this group and existing taxonomic experience within NAMA. Amanitologist and NAMA member Rod Tulloss currently has more than 2000 sequences available in his personal registry, covering 290 different species[1]. Public repositories of genetic data, such as GenBank, offer thousands more reference sequences. Moreover, I will also be examining the *Amanitaceae* from Indiana through 2017, providing more than 200 additional sequenced voucher collections that can be used as reference data for the current project.

The remainder of this article describes the protocols that will be employed for the project, as well as the intended scope of what we hope to achieve. A portion of the project description delves into highly technical jargon, so just a word of caution before you proceed. If after reading this you find yourself wanting to learn more about these topics, wanting to participate in the NAMA Mycoflora Committee or the Mycoflora 2.0 project, or if you would like to chat about implementing fungal sequencing projects at the club level, I encourage you to contact me at steve@hoosiermushrooms.org.

## Research Objectives

The current project will perform a genetic survey of NAMA voucher collections of the *Amanitaceae* from 1997 to the present. There are 241 vouchers from the genus *Amanita* and three from *Limacella* collected by the NAMA Voucher Project over this time period. The collections are currently under the care of Patrick Leacock, who had graciously allocated time in November 2016 and May of 2017 for my visits to collect tissue from these specimens.

### Number of NAMA *Amanitaceae* vouchers by year

| Year | # of Vouchers | Year | # of Vouchers | Year | # of Vouchers |
|---|---|---|---|---|---|
| 2015 | 26 | 2008 | 10 | 2001 | 21 |
| 2014 | 14 | 2007 | 19 | 2000 | 40 |
| 2013 | 6 | 2006 | 0 | 1999 | 2 |
| 2012 | 18 | 2005 | 4 | 1998 | 15 |
| 2011 | 18 | 2004 | 27 | 1997 | 4 |
| 2010 | 12 | 2003 | ? | | |
| 2009 | 8 | 2002 | ? | | |

**Molecular Methods** - Standard molecular methods are being employed for the voucher specimens involved in this study. (Prepare for the technical jargon.) DNA will be extracted from specimens utilizing the Promega Wizard protocol.[2] PCR amplification will utilize ITS1F and ITS4 primer combinations for all specimens. Additional regions including LSU, TEF1, and RPB2 may be examined for specimens without a corresponding ITS reference sequence. It is estimated that 25% of the 244 samples (~60 vouchers) will need to have additional loci sequenced. For each locus to be sequenced, two reads will be attained, one from the forward primer and one from the reverse primer. Extractions and amplifications will be done at the Aime Lab at Purdue University. Sequencing will be done by a commercial service used by the Aime Lab.

**Bioinformatics** - (Prepare for a bit more technical jargon.) Forward and reverse reads will be combined and edited using Sequencher (or equivalent) software. These curated sequences will be compared against the public databases, GenBank and UNITE. All sequences will also be compared against the in-house databases of Rod Tulloss and Stephen Russell. High-quality reference sequences will be aligned with target sequences using the MUSCLE algorithm. Phylogenetic trees will be constructed using Maximum-likelihood.

**Taxonomic Work** - Voucher collections will be analyzed using established morphological and molecular methods. A summary of the outcomes of this project will be submitted to *The Mycophile* within a year after the conclusion of the study. If species new to science are discovered, they will be published in a mycological journal (e.g. *Mycologia*, *Mycotaxon*, *Fungal Diversity*) in collaboration with mycoflora committee members and outside experts. Rod Tulloss has agreed to help with the analysis for this study. Mycoflora Committee members, as well as other NAMA members, are invited to help with the analysis of the acquired data. Seventy-five percent of the vouchers should require little analysis in order to gain a solid species hypothesis. Most of the analytical time for this study will be spent on the remaining 25% of the species that are expected to lack informative reference sequences.

**Project Cost** – The estimated cost for sequencing the ITS region is $10/sample, for a total of $2440 for all 244 vouchers. Each additional region sequenced (LSU, TEF-1, and RPB2) will cost an additional $5 for forward and reverse reads. For 60 select vouchers to receive three additional sequenced genes, the total cost will be $900. Thus, the total budget request for this project in 2017 was $3340.

## Dissemination of results

All of the metadata produced by this study will be publicly accessible via the MycoMap framework (www.mycomap.com), an online mycology data integration portal. This will include the raw forward and reverse sequence files, edited sequences, alignments, and phylogenetic trees. All final sequences will be included with the original record reported on Mushroom Observer and deposited into GenBank. Any alignments used in published works will be made available on TreeBASE. Initial results from this study will be available by September 2017, with a final report to be published in *The Mycophile* in 2018.

## Conclusion

The interest in obtaining genetic information from mushroom specimens has been significantly increasing among the citizen science community in recent years. The process to obtain a DNA sequence is now simple/economical enough that a growing number of mycological societies have already launched sequencing initiatives at the local level. The NAMA Mycoflora Committee is playing an active role in organizing these various local level initiatives into a coordinated national effort with the Mycoflora 2.0 Project, as described in recent *Mycophile* and *FUNGI Magazine* articles by Bill Sheehan. As a general interest in molecular mycology continues to expand at the amateur level, so will opportunities for NAMA to advance its educational and scientific missions, and to support the efforts that are currently being pursued by member organizations.

Footnotes

1 - http://www.amanitaceae.org/?Amanitaceae+genetic+sequence+collection

2 – https://www.promega.com/resources/protocols/technical-manuals/0/wizard-genomic-dna-purification-kit-protocol/

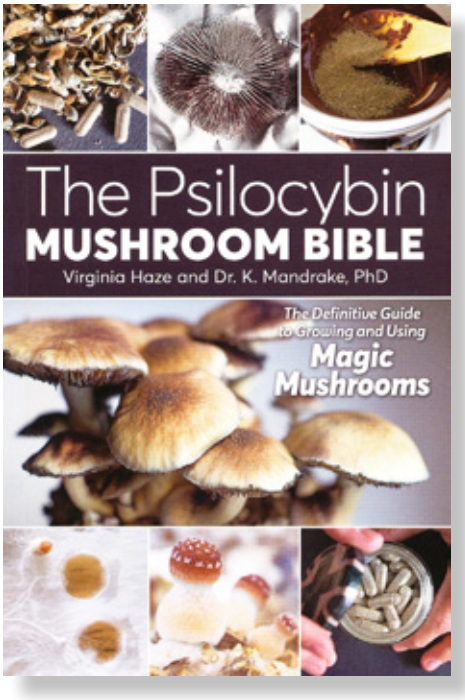
## WINNING JUDGES' OPTION PHOTOS from 20016 NAMA Photo Contest!

**1st Place:** Frosty the Fungus
by Mark Bower

**2nd Place**: Bunny shaped *Fuligo septica*
by Autumn Mallett

**3rd Place**: Mushroom Rainbow
by Alissa Allen

# *The Psilocybin Mushroom Bible: The Definitive Guide to Growing and Using Magic Mushrooms*




Virginia Haze and Dr. K. Mandrake, PhD

2016, Green Candy Press (www.greencandypress.com)

978-1-937866-28-0 (Paper, xxv+358 pages)

$30.00

And yet another guide for growing mushrooms at home — this time focused on a single species, Psilocybe cubensis. So, does anyone really need another book about growing magic mushrooms? Certainly there are many out there, plus a lot of information on the internet. Let's see what this one provides.

The contents: Dedication, Disclaimer, Introduction, Chpt 1—Mushroom Basics, 2—Sourcing Your Spores, 3—Good Practice and Sterile Technique, 4—Equipment and Supplies, 5—Making Your Own Hardware, 6—Basic PF Tek, 7—Liquid Culture, 8—Grain, 9—Bulk Substrate Method and Pasteurization, 10—Agar, 11—Troubleshooting and Contaminants, 12—Drying Mushrooms and Storing Spores, 13—Consuming Your Shrooms, 14—Microdosing and the Future of Psilocybin as Medicine, Glossary (including definitions of such esoteric terms as "candy thermometer" and "contamination," and where we learn that "strain isolation" is "the process of isolating a strain"), Resources, Index, and, finally, About the Authors.

Starting with the last item … Given the emphasis the UK-based authors place on security and anonymity, including wearing a Mexican wrestler mask when their faces might otherwise appear in a photograph, I'm guessing their names are made up. Although the claim is made that one of the authors (Mandrake) is a scientist (with both "Dr." and "PhD" attached to his name on cover and title page), the author information gives no indication of what institution awarded the degree and in what field. The presentation of the "science," especially the mycological/biological material, is flawed (more on this below) and leads me to wonder about the advertised credential.

On to the main purpose of the book — to teach one how to grow magic mushrooms. It is abundantly clear that the authors know how to grow *Psilocybe cubensis* and their advice for doing that is what most people who buy this book will be after. The advice and procedures seem sound and the many images of dense fruitings of *P. cubensis* provide ample proof that their version of the PF Tek method works. However, occasional inconsistencies or mistakes occur, such as listing recipe ingredients in one place as volumes and elsewhere as "parts," and the relative volumes not agreeing with the "parts." In discussing lemon tek as a means of consuming the mushrooms, they first state that there's little evidence that soaking the mushrooms in the acidic lemon juice promotes the conversion of psilocybin to psilocin, leading to a much quicker onset of the "therapeutic effects." But later, in the recipe for ginger and lime chocolates, they state that "The lime juice works in the same way as in the lemon tek method above, meaning that there is a fast come-up and the trip seems incredibly intense." These sorts of details aren't critical but they will cause some head-scratching and necessitate trial-and-error on the part of a potential grower/user.

The many step-by-step photos for the different parts of the grow process are effective. However, the "decorative" and other supporting photos are mostly disappointing and largely unnecessary. All of the more or less close-ups suffer from being out of focus over much of the image — in a few cases, nearly the whole picture is fuzzy. Of the decorative photos, about 175 are redundant or contribute nothing to illustrating the techniques. Instead they show, for instance, a photo of four buttons captioned "They're not pretty, but they're still potent!" and an out-of-focus singleton "This mushroom has made it! Good work, mushroom." The book

could have been shortened by probably 40 pages or more by leaving them out, with no downside, and it might even have resulted in a lower price.

I'm not much of a mushroom eater, but I do know that munching on dried mushrooms is not at the top of most mycophiles' favorite recipes list (although I have seen a recipe for bolete chips). So, for those wishing to indulge and not looking forward to spending "10 minutes choking them down while trying not to retch everything up like a cat with a magical hairball," the chapter "Consuming Your Shrooms" should be quite welcome. Several of the recipes include ginger to help reduce the nausea that many users experience and I was drawn particularly to the one for ginger and lime chocolates. When it comes to consumption (especially if using capsules), one should note that Haze and Mandrake apparently grow only, or at least primarily, *P. cubensis* and likely have developed strains that are relatively consistent in potency. Thus they feel confident in specifying amounts of mushroom that will provide a specific dose ("One of the benefits of making capsules is that you can be sure of getting an exact dose every time"). However, different species vary quite a bit in their psilocybin content and it is likely that different strains of a given species also do. So stay aware of this and err on the side of caution. As the authors say, "always dose low and take more if necessary."

Most folks know that possessing psilocybin-containing materials is illegal in North America. So it is probably no surprise that, like most similar books, this one offers a silly disclaimer, "The material offered in this book is presented as information that should be available to the public. The aim of the Publisher is to educate and entertain. Whatever the Publisher's view on the validity of current legislation, we do not in any way condone the use of prohibited substances or breaking the law." Even though the publisher produced a book that tells one exactly how to break the law and offers advice for not getting caught. At least the authors' version is funnier, "we recommend that all cultivation be done in Spain, where it's legal and where they serve a damn good local beer on a hot afternoon."

Although I'm not sure another book on how to grow mushrooms at home with the PF Tek method is needed, this one will serve its purpose of showing you how to do that, albeit for only one species. A little trial and error will be required, and North American products comparable to the UK ones mentioned in the book will have to be identified, but these shouldn't present major challenges.

However, if you do opt for this book, be aware that there are many errors in the presentation of the science. For instance, mushrooms are sexual reproduction structures produced by fungus mycelia and can be considered as analogous to the fruits of plants. The mycelium is not the mushroom, and referring to "fruits of the mushroom" and "The mycelium is really the mushroom itself, while the top part is simply the expression of the fruit" are just plain wrong. Molds are filamentous fungi and yeasts are unicellular fungi. Haze and Mandrake often refer to them as though they are something separate from fungi with statements such as "it's hard to tell mold from mycelium." Of course it is, because the "mold" is the mycelium of a fungus. Spores do not mate. Most molds do no harm to human health yet Haze and Mandrake seem paranoid about any exposure to them. They seem to forget bleu and camembert cheeses, and Quorn mycoprotein, for instance. Why do they wear gloves when picking the mushrooms and when making spore prints? "Rhizomorphic" and "tomentose" mycelia are different hyphal morphologies produced by a single fungus. They aren't different "strains" and, throughout, it appears the authors don't understand what strains actually are. And they get the physics backwards in saying that things move from areas of low pressure to areas of high pressure.

Obviously there are aspects of this book that could stand improvement. However, if growing magic mushrooms is your goal then, with a modicum of trial and error, it should provide most of the guidance you need, including how to avoid having the local sheriff catch wind of your endeavor. Just look elsewhere if you want to understand fungal biology and the other science that underlie successful cultivation.

Steve Trudell

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Newsletter of the North American Mycological Association
THE MYCOPHILE

## Mushroom of the Issue

***Leucopholiota decorosa*** **Photo and Description by Walt Sturgeon**

In Eastern North America and the upper Midwest there is a species that beginning collectors often mistake for a *Pholiota* and it does resemble the scaly *Pholiota* species. The misidentification is corrected easily by simply making a spore print. Unlike the brown spored *Pholiota* species this mushroom has white spores. *Leucopholiota decorosa* was originally described by Charles Horton Peck in 1873 and over the years has been placed in several different genera. Its current name was made by a collaboration of Orson Miller, Thomas Volk and Alan Bessette. It is the only North American species in this genus. It is most common in Pennsylvania, Ohio and New York but can be found from Ontario and Maine south to North Carolina and west to Wisconsin. It is a saprobe and occurs singly or in small groups on the dead wood of broadleaf trees. Sugar maple is a common host. It typically fruits from August to October.This is a very attractive medium sized mushroom. Its scaly tan cap and tan scaly lower stem contrast with its white gills and silky white upper stem. The scales are actually tufts of fibers. It has a mild odor and taste. Some find it slightly bitter. Its edibility is unknown although there are reports of it being edible.