Death caps
and other fungi responsible for amanita poisoning

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French agency for food, environmental and occupational health & safety

Investigate, evaluate, protect

Amanita phalloides, Death cap
Division of the Basidiomycota
Sub-division of the Agaricomycotina
Class of the Agaricomycetes, Sub-class of the Agaricomycetidae, Order of the Amanitales,
Family of the *Amanitaceae*.

The death cap, along with certain *Lepiota* and *Galerina*, produce toxins (amanitins or amatoxins) that are not destroyed by cooking and that cause amanita poisoning. This is manifested by late-onset (more than 6 h after ingestion of these fungi) digestive, liver and kidney disorders, and is fatal in 10 to 15% of cases.

Characteristics of the fungi responsible for amanita poisoning

Transmission/exposure routes

Amanita poisoning is caused by ingestion of the fungus.

The death cap

The death cap is responsible for a large majority of fatal poisonings from ingestion of fungi, and for 90% of cases of amanita poisoning (Enjalbert *et al.*, 2002).

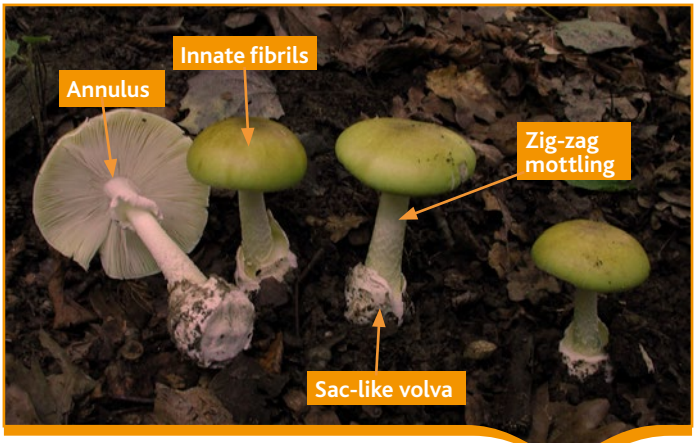
The death cap is a fungus with fibrous flesh, whose stalk and cap are easily separable. It is characterised by white gills, a white membranous annulus, and a sac-like volv(1) that is white outside but often greenish inside. The cap usually measures from 6 to 12 cm in diameter (up to 15 cm); it is hemispherical then flattened, convex, smooth, and always covered by radial grey-brown innate fibrils(2) of varying density. It is mainly greenish or yellowish in colour, but this is quite variable, ranging from a white tint for the *alba* variety to grey, copper or even brown tints. The gills are fairly dense, white and free(3). The cylindrical stalk bulges at the base and can reach up to 20 cm in height for a diameter of 1 to 2 cm (up to 3 cm). It is whitish in colour but nearly always has zig-zag mottling(4) ewith greenish yellow hints, under the annulus. The flesh is white and the adult or ageing mushrooms smell of wilted roses.

(1) Volva: membranous sac surrounding the base of the stalk of certain fungi; the volva is one of the possible signs of the universal veil (which completely surrounds the young fungus, a bit like an eggshell) after it has opened at the top to enable the entire fruiting body to emerge, and remains intact at the base.

(2) Innate fibrils: non-protruding fibrils in the cap that are enveloped in the thickness of the surface.

(3) Free gills: lamellae, generally plump in outline, which do not reach the fungus's stalk, but are embedded directly in the cap (term used to distinguish them from «adnate gills» which adhere to the stalk of the fungus).

(4) Mottling: lines or bands creating a marbling effect (like chiné fabric).



***Amanita phalloides*, typical colouring © R. Courtecuisse**

***Amanita phalloides*, atypical colouring © R. Courtecuisse**

Very young death caps are wrapped in a protective membrane called the «universal veil», which gives them the appearance of a small completely white egg. At this stage, only a longitudinal cross-section can reveal certain specific characteristics hidden by the universal veil, such as the colour of the cap surface (often faded); the future annulus is generally invisible as it adheres to the gills that are also invisible (hidden by a membrane known as the «partial veil») (Romagnesi, 1962). During its development, the identification characteristics described above appear gradually.

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In France, the death cap is a fungus commonly found in woods or broadleaved forests (especially under oak trees), preferably on non-calcareous soils. It is rare under conifers. It can be seen mainly from August to October (sometimes earlier in the summer and even until winter in mild climate regions) and in general is confined to altitudes below 1,000 m (Polese and Lanceau, 2003). It does not seem to extend beyond 1,400 m.

Other species responsible for amanita poisoning

The other incriminated species are also quite common overall, especially outside forest environments (lawns, dunes, thickets, etc.). They include:

- other deadly *Amanita* such as the European destroying angel, *A. virosa* and its varieties, including the variety *levipes* (currently expanding rapidly in western France), and the fool's mushroom *A. Verna* and its varieties, *A. decipiens and A. dunensis*. All these *Amanita* are morphologically similar to the death cap but are white in colour.
- other species more recently incriminated in this syndrome: essentially small *Lepiota*[5] (white free gills, partial veil forming an annulus, often diffracted or broken into more or less labile bracelets in the small *Lepiota*) and *Galerina* such as the *Galerina marginata, G. autumnalis,* etc. (Flesch and Saviuc, 2004).

Risk of confusion

The main risks of confusion relate to:

- young *Amanita*: young specimens that are still closed (harvested before the universal veil has opened, at the «egg» stage) may be confused with many other species (young ceps, other gilled mushrooms at the *primordium* stage, puffballs, wood mushrooms, etc.);
- adult *Amanita*: once developed, deadly *Amanita* should no longer be mistaken for other species due to their combination of characteristics (presence of an annulus even though it may have been consumed by slugs, presence of a sac-like volva, free gills and a white sporeprint). However, the uninitiated could confuse them with:
  - *Tricholoma* such as the charbonnier (*Tricholoma portentosum*) or the yellow blusher (*Tricholoma sejunctum*) which is similar in colour, or the dove-coloured tricholoma (*Tricholoma columbetta*) for white forms, all without veils and with cankered adnate gills[6],
  - Some *Russula* in varying shades of green (*Russula cyanoxantha, R. virescens*),
  - Agarics, without volva and with a *brownish-black sporeprint* (the field mushroom - *Agaricus campestris* and more often *A. arvensis* or certain white forest species such as A. *silvicola*), etc.;
- *Galerina marginata* can very easily be confused with the edible sheathed woodtuft (*Kuehneromyces mutabilis*), which is recognisable mainly by its «booted» stalk (resembling a sock marked by small curly scales).

In addition, confusion is also possible with other species that are morphologically very different to the fungi responsible for amanita poisoning: for example in the cases of poisoning observed by the French poison control centres in the second half of 2013, the patients thought they had picked field mushrooms (*Agaricus campestris*) and other morphologically very different species such as *Lactarius*.

Recommendations for primary production

Ensure that no toxic or deadly fungi contaminate harvests of wild or cultivated mushrooms.

Human foodborne illness

Nature of the disease / Nature of the toxic effects

Amanita poisoning is a late-onset mycotoxic syndrome (whose latency period is more than six hours after ingestion of the fungi).

The toxins implicated in this syndrome are amanitins or amatoxins (nine distinct toxins). They are stable during cooking, sterilisation and freezing, are water-soluble and resistant to enzymatic degradation. Two toxic mechanisms have been described, the first by inhibition of protein synthesis and the second by oxidative stress (Faulstich, 1979; Leist *et al.,* 1997; Zheleva *et al.,* 2007). The liver is the organ most affected (Trueb *et al.,* 2013).

Amanita poisoning typically comprises three major steps: an asymptomatic (silent) latency period of at least six hours, a gastro-intestinal phase and a visceral phase. The severity of the symptoms depends on the quantity of fungus ingested, the species consumed, the physiological condition of the consumer, and the number of possible cumulative meals. The characteristics of the poisoning are specified in Table 2.

The early onset of symptoms must not give the clinician a false sense of security. The consumer may have ingested several poisonous species, some of which are responsible for an early mycotoxic syndrome whose first symptoms appear very quickly (in the first thirty minutes). In addition, there have been rare cases of pure acute renal failure without liver damage (in the event of exclusive ingestion of species responsible for amanita poisoning).

(5) *Lepiota helveola, L. josserandii, L. brunneoincarnata, L. brunneolilacina, L. kuehneri, L. subincarnata, L. helveoloides, L. pseudohelveola, etc.*

(6) Adnate gills: gills adhering to the stalk of the fungus.

Table 2. Characteristics of amanita poisoning

Phase	Onset time	Duration of phase	Clinical picture
I - Silent, so-called «incubation»	From ingestion	Between 6 and 48 h after ingestion;10 to 12 h on average	Totally asymptomatic
II - Gastro-intestinal	Between 6 and 48 h after ingestion 10 to 12 h on average	Between 24 and 48 h on average	**Symptoms appearing simultaneously:** Vomiting Very abundant watery diarrhoea Abdominal cramps **Possible complications:** Severe dehydration Intense thirst Tachycardia and hypotension
III - Visceral	Between 36 and 48 h after ingestion	Highly variable (from almost non-existent to several days)	**Symptoms appearing successively:** Hepatic cytolysis Hepatocellular insufficiency (+/- kidney failure with oliguria or anuria) Haemorrhage Hepatic encephalopathy Coma, and possibly death

Susceptible population groups[7]: Any individual likely to develop intoxication due to ingestion of the toxin in the fungus. Serious cases primarily concern children, pregnant women and the elderly.

Dose-effect relationship[8]

The lethal dose of amanitins is 0.1 mg/kg, or the ingestion of 7 mg for a person weighing 70 kg, contained in a single death cap or 50 g of this fresh fungus (Faulstich, 1980; Wieland, 1986).

Epidemiology

To this day, in France, there is no surveillance of cases of amanita poisoning. A retrospective study identified 1,165 cases of accidental poisoning linked to the consumption of fungi reported to Poison Control and Monitoring Centres (CAPTV) from July to December 2013 in France, with the second half-year concentrating almost 90% of cases of fungi poisoning recorded during a calendar year. All the French regions were affected by these poisoning cases, although their incidence was higher south of the Loire, and in western (Midi-Pyrénées) and eastern (Rhône-Alpes) regions. The age of the poisoned subjects varied from 16 months to 90 years old, an average of 44.5 years (median 46 years). The sex ratio was equal to 1.0.

The aim of this retrospective study was not to list all of the cases of amanita poisoning (benign, serious[9] or deadly disorders). However, for the second half of 2013, 80% of the serious cases of poisoning by ingestion of fungi were amanita poisoning (16 cases out of 20). Among these 16 serious cases of amanita poisoning, the ingestion of death caps was confirmed for six cases and consumption of *Lepiota* for two cases. Three cases were fatal, three others led to sequelae (liver transplant or kidney failure) and in the other ten cases the patients recovered.

Surveillance and prevention

Surveillance in food

Because these mushrooms are toxic, their sale is prohibited. There is no specific monitoring of this hazard in food.

Inactivation treatments

There is no conventional physical or thermal treatment (cooking, sterilisation or freezing) likely to destroy the toxins in these fungi.

Recommendations

The information presented in this sheet indicates firstly that amanita poisoning is often caused by the ingestion of fungi that have been poorly identified during gathering, and secondly that the toxins are not destroyed during preservation or preparation of the fungi. Prevention therefore relies solely on the general recommendations below.

Recommendations for operators

Ensure that any wild or cultivated mushrooms intended for canning or drying are edible.

General Recommendations for gatherers and consumers of wild mushrooms

- **Only pick mushrooms that you know perfectly well, in surroundings with which you are familiar. Take the time to check methodically all the fungus's characteristics (by systematically examining the colour of the gills, whether or not there are signs of a universal veil, such as a volva, whether there is an annulus, etc.); if in doubt, discard any suspect fungus.**
- Pick only specimens in good condition and take the entire mushroom (stalk and cap; under no circumstances cut it with a knife), to facilitate its identification (some key characteristics may be found at the very base of the stalk!).
- Avoid picking after heavy rains or frost, because such weather conditions can alter the characteristics of fungi, especially their colour.
- Do not pick young mushrooms because very often their distinctive characteristics can only be observed in adult fungi in a perfect state of development.
- Never go picking near polluted sites (by the side of a road, industrial zones, landfill sites) because even when perfectly edible, mushrooms concentrate pollutants that can cause poisoning.
- Place the mushrooms separately in a box, crate or basket (ideally in different compartments) but never in a plastic bag which accelerates their decomposition. After gathering, the mushrooms should be sorted and checked.
- **Take photos of your mushrooms to keep along with anything left from what was gathered (whole specimens as examples or, failing this, peelings) for subsequent identification, in order to adapt treatment in the event of poisoning.**
- **If there is the slightest doubt about the identification of any of the mushrooms harvested, seek advice from a specialist (pharmacists, local mycology associations and societies, or even other gatherers may be consulted).**
- Keep the mushrooms separate, in the refrigerator, and consume them within two days of picking.
- Consume edible wild mushrooms in reasonable quantities and cooked thoroughly for at least fifteen minutes; never eat them raw. Bear in mind that cooking does not destroy certain toxins.
- **If symptoms should appear, especially digestive ones such as diarrhoea, vomiting, nausea, or any other signs (sweating, discomfort, neurological signs with agitation, confusion, vision disorders) following one or more meals with the mushrooms, note the time of the meals and of the first symptoms, and call the emergency services (15) or the poison control centre.**

(7) Susceptible population groups: people with a higher than average probability of developing symptoms of the disease, or severe forms of the disease, after exposure to a foodborne hazard.

(8) Relationship between the dose (the quantity of toxins ingested during a meal) and the effect on an individual.

(9) Clinical and/or paraclinical impairment (biological examinations, etc.) that is potentially fatal, and/or with sequelae responsible for a significant disability.

References and links

General references

- Courtecuisse, R., Duhem, B., 2011. Guide des champignons de France et d'Europe. 3rd edition. Delachaux and Niestlé, 544p.
- Enjalbert, F., Rapior, S., Nouguier-Soule, J., Guillon, S., Amouroux, N., Cabot, C., 2002. Treatment of amatoxin poisoning: 20-year retrospective analysis. Journal of Toxicology - Clinical Toxicology, 40, 715-757.
- Faulstich, H., 1979. New aspects of amanita poisoning. Klinische Wochenschrift, 57, 1143-1152.
- Faulstich, H., 1980. Mushroom poisoning. Lancet 2, 794-795.

- Flesch, F., Saviuc, P., 2004. Intoxications par les champignons: principaux syndromes et traitement. EMC - Médecine, 1, 70–79.
- Graeme, K.A., 2014. Mycetism: a review of the recent literature. Journal of Medical Toxicology: Official Journal of the American College of Medical Toxicology, 10, 173-189.
- Leist, M., Gantner, F., Naumann, H., Bluethmann, H., Vogt, K., Brigelius-Flohe, R., *et al*. 1997. Tumor necrosis factor-induced apoptosis during the poisoning of mice with hepatotoxins. Gastroenterology, 112, 923-934.
- Polese, J.-M., Lanceau, Y., 2003. Le guide des champignons des Alpes. Tétras éditions, 179p.
- Romagnesi, H., 1962. Petit atlas des champignons. Bordas, Paris, France.
- Trueb, L., Carron, P.N., Saviuc, P., 2013. Intoxication par les champignons. Revue Médicale Suisse, 394, 1465-1472.
- Wieland, T., 1986. Peptides of poisonous Amanita mushrooms. Springer, New-York, 181-206.
- Zheleva, A., Tolekova, A., Zhelev, M., Uzunova, V., Platikanova, M., Gadzheva, V., 2007. Free radical reactions might contribute to severe alpha amanitin hepatotoxicity: a hypothesis. Medecine Hypotheses, 69, 361-367.

Useful links

- Poison Control and Monitoring Centres (CAPTV): **www.centres-antipoison.net**
- French Mycological Society: **www.mycofrance.fr**
- List of French mycological societies: **www.mycodb.fr**