AMINITA MUSCARIA & AMANITA PANTHERINA AND OTHERS

International Programme on Chemical Safety
Poisons Information Monograph (Group monograph) G026
Fungi

Please note that further information on Sections 1, 3.1, 3.2 and 8 is pending.

1. NAME

1.1 Scientific name

Species of the genus Amanita. The species known to cause the majority of toxic exposures are: Amanita muscaria and Amanita pantherina.

The toxins are all isoxazole derivatives.

Other Amanita mushrooms contain the same toxins and induce similar toxicity:

Amanita muscaria var. Kamtschatica Langsdorff ex. Fr.
Amanita regalis (Fr.) R. Mre. (A. muscaria var. umbrina Fr.)
Amanita muscaria var. formosa
Amanita muscaria var. alba
Amanita gemmata (Fr.) Bertillon
Amanita velatipes Atk.
Amanita cothurnata Atk.
Amanita flavovolvata Sing.
Amanita strobiliformis (Vitt.) Quel.
Amanita pantherina (DC ex Fr.) Secr.
Amanita pantherina multisquamosa
Amanita pantherina velatipes
Amanita pantherina pantherinoides
Tricholoma muscaria

1.2 Family

Agaricaceae (*Agaricales)*

The genus is Amanita *(Amanitaceae)*

1.3 Common name(s) and synonym(s)

Amanita muscaria

English	Fly Agaric
German	Fliegenpilz, Roter fliegenpilz
Spanish	Falsa oronja, Amanita matamoscas
French	Amanite tue-mouche, Agaric aux mouches, fausse oronge
Italian	Ovulo malefico, Uovolaccio
Polish	Muchomor czerwony

Amanita pantherina

English	Panther cap.
German	Pantherpilz, Braunner Knollenblätterpilz

Spanish	Amanita pantera, galipiermo falso
French	Amanite panthère, Fausse golmelle
Italian	Tignosa bigia, Tignosa regata, Agarico panterino
Polish	Muchomor plamisty

2. SUMMARY

2.1 Main risks and target organs

The most frequent cause of intoxication is the consumption of Amanita muscaria by people who mistake it and ignore its toxicity. Amanita muscaria might also be ingested in order to obtain mind-altering effects. The central nervous system is the major target organ.

2.2 Summary of clinical effects

Symptoms appear 30 to 90 minutes after ingestion and last usually for 6 hours but may persist for 12 to 24 hours. The primary effects are central nervous system depression and stimulation, which may alternate. Symptoms usually begin with drowsiness followed by a state of confusion, with ataxia, dizziness, euphoria resembling alcohol intoxication and may proceed to increased activity, illusions, or even manic excitement.
These periods of excitement may alternate with periods of somnolence, deep sleep or stupor. The illusions are primarily a misinterpretation of sensory stimuli. The prognosis is usually good with symptomatic treatment. Death from these mushrooms is extremely rare.

2.3 Diagnosis

Based upon history of ingestion and clinical features.

2.4 First aid measures and management principles

Treatment includes prevention of absorption of the toxins and treatment of the signs and symptoms of intoxication as they occur. Atropine is not recommended. Induction of emesis is NOT recommended because of the potential central nervous system depression and seizures. There is no specific antidote for Amantia muscaria and Amanita pantherina poisoning.

2.5 Poisonous parts

All parts of the fruiting body of Amanita muscaria and Amanita pantherina are toxic.

2.6 Main toxins

The main toxins are: ibotenic acid, muscimol and muscazone. These three toxins are found in certain species of mushrooms throughout the world. They are related and are all isoxazole derivatives.

3. CHARACTERISTICS

3.1 Description of the fungus

3.1.1 Special identification features

Identification: Complete and precise identification of the mushroom (if available) should be accomplished by a mycologist. If no mycologist is available, colour photographs may be helpful for a first identification. Identification is difficult when the mushrooms have been altered by cooking, eating or storage.

Description of Amanita muscaria

Cap: 8 to 12 cm diameter, occasionally over 20 cm; conical when young, flattening in age; viscid, adorned with white to pale yellow warts or small patches. This mushroom has a variety of color variants, ranging from yellow through orange, orange-red to blood-red or scarlet.

The yellow, orange or orange-red caps (A. muscaria var. formosa) occurs in eastern North America. The scarlet cap (A. muscaria var. muscaria) occurs in western North America, throughout Europe and Asia.
Flesh firm, white throughout.

Gills *(lamellae):* crowded, free or just touching stalk, broad, white, minutely hairy edges.

Stalk: 8 to 15 cm long, 20 to 30 mm thick enlarging towards base and becoming bulbous; white, covered with silky hairs.

Ring: *(annulus):* large, membranous, white to yellowish, median to superior, resistant though margin usually frayed.

Cup *(volva):* the remains of the volva are often only 2 or 3 concentric rings above the bulb; white to straw.

Spores: white spore print (in mass); 8 - 11 by 6 - 8 microns, ellipsoid, thin walled, no amyloid reaction.

Description of Amanita pantherina

3.1.2 Habitat

Scattered or abundant, sometimes in fairy rings under hardwoods and conifers from spring to autumn.

3.1.3 Distribution

These species are widely distributed throughout the planet. Amanita muscaria grows in summer and autumn under coniferous and deciduous trees, from the lowland up to the subalpine zone. It occurs

practically all over the temperate and subtropical zones in Europe, North Africa, South Africa, Asia, Japan, Australia, North America (in the Western States of the USA more often than in the Eastern States) and in South America. (Seeger & Stijve, 1978).

3.2 Poisonous parts of the fungus

All parts of the fruit body of A. muscaria are toxic. The isoxazoles are NOT distributed uniformly in the mushroom. Most are detected in the cap of the fruit, then in the base, with the smallest amount in the stalk (Lampe, 1978; Tsunoda et al., 1993). Drying A. muscaria in the sun or with heater caused an increase of muscimol in the mushroom, though a lot of precursors of ibotenic acid was lost. Ibotenic acid and muscimol in the mushroom were stable on storage under dry or salt conditions (Benedict et al., 1966; Tsunoda et al., 1993).
Whilst ibotenic acid and muscimol are rapidly released from the mushrooms by cooking and boiling, these processes do not eliminate all toxic substances.

3.3 The toxin(s)

3.3.1 Name(s)

The main toxins are: ibotenic acid, muscimol and muscazone.

These three toxins are found in certain species of mushrooms throughout the world. They are related and are all isoxazole derivatives (Eugster, 1979). Ibotenic acid and muscinol are mainly responsible for the toxic effects (Takemoto et al., 1964; Bowden and Mogey, 1965; Eugster et al., 1965; Muller and Eugster, 1965).

3.3.2 Description, chemical structure, stability

Ibotenic acid and muscimol have similar structure to glutamic acid and GABA (Krogsgaard-Larsen P. et al., 2000).

Muscimol

CHEMICAL NAME: 3-Isoxazolol, 5-(aminomethyl)-
CAS REGISTRY NUMBER: 2763-96-4
SYNONYMS:
3(2H)-ISOXAZOLONE, 5-(AMINOMETHYL)-3-HYDROXY-5-AMINOMETHYLISOXAZOLE
3-Hydroxy-5-aminomethylisoxazole-agarin
3-HYDROXY-5-AMINOMETHYLISOXAZOLE-AGARIN
AGARIN
5-(Aminomethyl)-3(2H)-isoxazolone
5-(Aminomethyl)-3-isoxazolol
5-Aminomethyl-3-hydroxyisoxazole
5-Aminomethyl-3-isoxyzole

Agarin
AGARINE
Muscimol

Pantherin
PANTHERINE
RCRA waste number P007
OTHER NUMBERS: 6036
NIOSH/NY3325000
NY3325000
(Ref: IPCS INTOX CD-ROM, 2000, 1)

Ibotenic acid

CHEMICAL NAME: 5-Isoxazoleacetic acid, alpha-amino-3-hydroxy-, monohydrate
CAS REGISTRY NUMBER: 60573-88-8
SYNONYMS
alpha-Amino-2,3-dihydro-3-oxo-5-isoxazoleacetic acid
alpha-Amino-3-hydroxy-5-isoxazoleacetic acid hydrate
alpha-Amino-3-hydroxy-5-isoxazolessigsaure hydrat
Amino-(3-hydroxy-5-isoxazolyl)acetic acid
Ibotenic acid
Ibotensaeure
Isotenic acid
Pramuscimol
OTHER NUMBERS
NY2100000
(Ref: IPCS INTOX CD-ROM, 2000, 1)

The toxins are thermostable and are NOT destroyed by cooking.

3.3.3 Other physico-chemical characteristics

Molecular weight:

Ibotenic acid:	176.15
Muscimol	114.12

3.4 Other chemical contents of the fungus

Bufotenine (Waser, 1979)
Amavadin: a vanadium compound (Kneifel et al, 1986).
Stizolobic and Stizolobinic acid: L-DOPA oxidation products (Chilton et al. 1974; Bresinsky & Besl, 1985).
Muscaflavin, Muscaurin: colorant principles (Depovere & Moens 1984)
Muscarine (Eugster, 1979)

4. USES/CIRCUMSTANCES OF POISONING

4.1 Uses

4.1.1 Uses

4.1.2 Description

Amanita muscaria and Amanita pantherina are not and have not been used in medical practice.

In the past it has been used in different populations and cultures as a fly-killer and an inebriant

(Siberia), an ecstasy agent (India), and a hallucinogenic (Indian peoples of Meso-America). In Japan, a derivative of muscinol is presently being used as a pesticide. An extract of ibotenic acid has been found to be some 20 times more powerful than monosodium glutamate as a flavour enhancer (Wasson, 1964, 1968, 1972, 1979).

4.2 High risk circumstances

The most frequent course of intoxication is the consumption of Amanita muscaria and Amanita pantherina by people who mistake it and ignore its toxicity. Amanita muscaria and Amanita pantherina might also be ingested in order to obtain psychotic effects and especially to expand or alter spatio-temporal awareness.
Death from this kind of mushroom is rare, or rarely reported. If so, it is due to complications. However, it would be unwise to consider eating them because the toxins are complex, variable in quantity, and not completely understood.

4.3 High risk geographical areas

These species are widely distributed throughout the planet.

5. ROUTES OF EXPOSURE

5.1 Oral

Ingestion of mushrooms is the most common cause of intoxication.

5.2 Inhalation

No data available

5.3 Dermal

No data available

5.4 Eye

No data available

5.5 Parenteral

No data available

5.6 Others

No data available

6. KINETICS

6.1 Absorption by route of exposure

Based on the time of onset of clinical symptoms, the rate of absorption of the toxins of Amanita muscaria and pantherina from the gastro-intestinal tract seems to be

rapid. However, the exact rate and the proportion of absorption is still unknown.

6.2 Distribution by route of exposure

Muscimol and ibotenic acid, presumably cross the blood-brain barrier via some active transport system. Neither muscimol nor ibotenic acid is removed from the receptor by the GABA or glutamate active uptake system. Inefficient removal of these false neurotransmitters once they have passed the blood-brain barrier may be an important contributing factor to their central nervous system effect (Balcar & Johnston, 1972; Kronsgaard-Larsen & Johnston, 1975, 2000).

6.3 Biological halflife by route of exposure

Both ibotenic acid and muscimol may be detected in human urine within one hour after the ingestion of the mushrooms. The peak of excretion of ibotenic acid appears at the second hour after the ingestion.

6.4 Metabolism

Metabolites include pantherin, tricholomic acid and solitaric acid.
In humans, a substantial amount of ingested ibotenic acid is excreted in urine unmetabolized. Some is converted to muscimol which is more pharmacologically active.

6.5 Elimination and excretion

In human beings, a substantial fraction of ingested ibotenic acid is excreted in the urine unmetabolized. Virtually no muscimol is excreted when pure ibotenic acid is eaten, but muscimol is detectable in the urine after eating A. muscaria, which contains both, ibotenic acid and muscimol. The ibotenic acid that does pass through the body is excreted rapidly, between 20 and 90 minutes after ingestion (Chilton, 1975). It should be noted that major symptoms appear after the first 60 minutes to 2 hours and reach their greatest intensity after the excretion of ibotenic acid. Major signs and symptoms of major or severe intoxication lasts more than 5 hours after the peak in excretion of ibotenic acid.
According to animal experiments, most of the muscimol delivered by intra peritoneal injection in the mouse is excreted in the urine as muscimol or metabolites of muscimol within 6 hours. About 1/3 is excreted as muscimol, 1/3 as a cationic conjugate, and 1/3 as an oxidation product (Ott J. et al, 1975).

In fact, the urine retains the pharmacological activity of the Fly Agaric, and in the sacred rituals in eastern Siberia, the urine of the Shamans and their acolytes was ingested by some followers and considered a better inebriant or hallucinogen (Efron et al 1979).

7. TOXINOLOGY

7.1 Mode of action

Ibotenic acid is structurally similar to glutaminic acid and mimics its effects in animals. Ibotenic acid is rapidly converted to muscimol, which structurally resembles GABA. Muscimol has a high affinity for GABA receptor sites and imitates the action of $GABA_B$ in animals and humans, inhibiting and controlling the recruitment and multiplication of nerve impulses mediated by many positive neurotrasmitters (Page, 1984).

7.2 Toxicity

7.2.1 Human data

7.2.1.1 Adults

The threshold for observation of central nervous system disturbances in human is about 6 mg of muscimol or 30 to 600 mg of ibotenic acid (Waser, 1979). This dose is potentially available in a single A. muscaria or A. pantherina mushroom.

In human volunteers, effects were measurable about 1 hour after ingestion of 7.5 to 10 mg of muscimol, or 50 to 90 mg of ibotenic acid. These effects continued for 3 to 4 hours, with some residual effects lasting as much as 10 to 24 hours in some subjects. Hangover was noted the next day (Chilton, 1975; Eugster, 1979).

Purified ibotenic acid and muscimol produced hallucinations, delirium, muscular spasm, and sleep in volunteers (Theobald et al., 1968, Waser, 1979).

7.2.1.2 Children

No data available.

7.2.2 Relevant Animal data

Muscimol lacks cholinergic effect at the neuromuscular junction. It inhibits tremor induced by tremorin but does not stop the associated salivation and lacrimation. A low dose of muscimol affects the EEG of cats and rabbits (Scotti et al., 1969; Theobald et al., 1968). These observations further support a localization of action of muscimol in the brain rather than in the peripheral nervous system.

Muscimol and ibotenic acid administered to rats and mice intraperitoneally affects brain in the levels of serotonin (5-hydroxytryptamine), noradrenaline and dopamine as do LSD, psilocybin and mescaline (Koenig-Bersin et al., 1970; Waser, 1979).

Ibotenic acid and glutamic acid produce convulsions in

immature rats, in which the blood-brain barrier is not completely developed (Johnston, 1973). Muscinol has been shown to produce electroencephalographic alterations distinct from hallucinogens such as LSD or mescaline (which is in accord with the clinical observations). Neither ibotenic acid nor muscimol appears to act on receptors acetylcholine, dopamine, or 5-hydroxytryptamine receptors in the central nervous system. It has been suggested that both muscimol and ibotenic acid act similarly by activation of the gamma-aminobutyric acid (GABA) receptor (Brehm et al., 1972; Walker et al., 1971).

The acute LD_{50} of muscimol in rats ranges from 4.5 mg/kg intravenously to 45 mg/kg, p.o. Experiments in dogs suggest that the effects of 20 mg/kg/day, p.o. are not cumulative (Waser, 1979).

7.2.3 Relevant in vitro data

No data available.

7.3 Carcinogenicity

No data available.

7.4 Teratogenicity

No data available.

7.5 Mutagenicity

No data available.

7.6 Interactions

Muscimol-treated animals, administered small doses of diazepam or phenobarbital, displayed flaccid paralysis and an electroencephalographic pattern similar to deep anesthesia Theobald et al. (1968) and Scotti de Carolis et al. (1969). These data cannot be extrapolated to humans.

8. TOXICOLOGICAL ANALYSES AND BIOMEDICAL INVESTIGATIONS

8.1 Material sampling plan

8.1.1 Sampling and specimen collection

8.1.1.1 Toxicological analyses

8.1.1.2 Biomedical analyses

8.1.1.3 Arterial blood gas analysis

8.1.1.4 Haematological analyses

8.1.1.5 Other (unspecified) analyses

8.1.2 Storage of laboratory samples and specimens

8.1.2.1 Toxicological analyses

8.1.2.2 Biomedical analyses

8.1.2.3 Arterial blood gas analysis

8.1.2.4 Haematological analyses

8.1.2.5 Other (unspecified) analyses

8.1.3 Transport of laboratory samples and specimens

8.1.3.1 Toxicological analyses

8.1.3.2 Biomedical analyses

8.1.3.3 Arterial blood gas analysis

8.1.3.4 Haematological analyses

8.1.3.5 Other (unspecified) analyses

8.2 Toxicological Analyses and Their Interpretation

8.2.1 Tests on toxic ingredient(s) of material

8.2.1.1 Simple Qualitative Test(s)

8.2.1.2 Advanced Qualitative Confirmation Test(s)

8.2.1.3 Simple Quantitative Method(s)

8.2.1.4 Advanced Quantitative Method(s)

A convenient analytical method for ibotenic acid (IBO) and muscimol (MUS) in a toxic mushroom, Amanita muscaria (A. muscaria), was developed. IBO and MUS in the mushroom were extracted with 70% methanol. After filtration, IBO and MUS in the extract were determined by high performance liquid chromatography (HPLC) with a UV detector set at 210 nm. The HPLC system adopted was ion-pair chromatography in the reverse-phase mode on an IRICA RP-18 (C18) column (4.0 mm with sodium dodecyl sulfate as a counter ion. Recoveries of IBO and MUS added to the sample were more than 98% and the minimum detectable concentration of IBO or MUS was about 1 ppm. The concentrations of IBO and MUS in A. muscaria ranged from 258 and 471 ppm and from 18 to 27 ppm, respectively. Neither of the compounds was detected in commercial edible mushrooms (Abstract: Tsunoda K, Inoue N, Aoyagi Y, Sugahara T, J Food Hyg Soc Jpn; 34(1). 1993. 12-17).

8.2.2 Tests for biological specimens

8.2.2.1 Simple Qualitative Test(s)

8.2.2.2 Advanced Qualitative Confirmation Test(s)

8.2.2.3 Simple Quantitative Method(s)

8.2.2.4 Advanced Quantitative Method(s)

8.2.2.5 Other Dedicated Method(s)

8.2.3 Interpretation of toxicological analyses

8.3 Biomedical investigations and their interpretation

8.3.1 Biochemical analysis

8.3.1.1 Blood, plasma or serum

8.3.1.2 Urine

8.3.1.3 Other fluids

8.3.2 Arterial blood gas analyses

8.3.3 Haematological analyses

8.3.4 Interpretation of biomedical investigations

8.4 Other biomedical (diagnostic) investigations and their interpretation

8.5 Overall interpretation of all toxicological analyses and toxicological investigations

8.6 References

9. CLINICAL EFFECTS

9.1 Acute poisoning

9.1.1 Ingestion

Symptoms appear within 30 to 90 minutes and are most marked at 2 or 3 hours. They may include: drowsiness, confusion, dizziness, ataxia, euphoria, delirium, visual and auditory disturbances with hallucinations, muscle cramps and spasms. Gastrointestinal disturbances and convulsions may also be seen.

9.1.2 Inhalation

No data available.

9.1.3 Skin exposure

No data available.

9.1.4 Eye contact

No data available.

9.1.5 Parenteral exposure

No data available.

9.1.6 Other

No data available.

9.2 Chronic poisoning

9.2.1 Ingestion

No data about chronic toxicity available.

9.2.2 Inhalation

No data available.

9.2.3 Skin exposure

No data available.

9.2.4 Eye contact

No data available.

9.2.5 Parenteral exposure

No data available.

9.2.6 Other

No data available.

9.3 Course, prognosis, cause of death

Toxic effects appear 30 to 90 minutes after ingestion and last usually for 6 hours but may persist for 12 to 24 hours. Hangover is often observed the following day. The prognosis is usually good with symptomatic treatment. Death from these mushrooms is extremely rare (Chilton, 1978). Although they sometimes produce dramatic intoxications with extensive psychological and neurological effects these mushrooms have a totally unwarranted reputation for being "deadly poisonous".

9.4 Systematic description of clinical effects

9.4.1 Cardiovascular

Pulse and blood pressure are usually normal. In one case, the patient developed cardiac fibrillation (Lincoff & Mitchel, 1977), also bradycardia was observed (Benjamin, 1992).

9.4.2 Respiratory

Respiration is not usually affected (Bosman et al., 1965); but respiratory depression is possible as a result of over treatment (Benjamin, 1992).

9.4.3 Neurological

9.4.3.1 Central nervous system (CNS)

The primary effects are CNS depression and stimulation, which may alternate. Symptoms usually begin with drowsiness followed by a state of confusion, with ataxia, dizziness, euphoria resembling alcohol intoxication and may proceed to increase activity, illusions, or even manic excitement.
These periods of excitement may alternate with periods of somnolence, deep sleep or stupor (Ammirati et al., 1985; Benjamin, 1992).
The illusions are primarily a misinterpretation of sensory stimuli such as changes in color vision, echo images (seeing through walls), identification of hospital personnel as divine figures, and the like, rather than true hallucinations caused by Psilocybe, or Paneolus.
However, vivid hallucinations associated with accidental poisoning by this Amanitas have been reported occasionally (McDonald, 1980; Carter et al., 1983).
Seizures are observed primarily in children (Benjamin, 1992).

9.4.3.2 Peripheral nervous system

Muscimol lacks cholinergic effects at the neuromuscular junction

9.4.3.3 Autonomic nervous system

Neither muscarinic nor atropinic effects have been observed in poisoning due to A. muscaria or A. pantherina.
Occasionally, sweating and salivation have been reported (Lampe, 1978; Waser, 1979; Benjamin, 1992).

9.4.3.4 Skeletal and smooth muscle

Muscle jerks, fasciculation and spasms in the extremities are observed (Chilton, 1978; Benjamin, 1992).

9.4.4 Gastrointestinal

Dyspepsia and vomiting may occur (Chilton, 1978, own data).

9.4.5 Hepatic

Amanita muscaria has no hepatotoxic effects.

9.4.6 Urinary

9.4.6.1 Renal

No data available.

9.4.6.2 Other

No data available.

9.4.7 Endocrine and reproductive systems

No data available.

9.4.8 Dermatological

Skin may be warm and flushed (Benjamin, 1992).

9.4.9 Eye, ear, nose, throat: local effects

Miosis as well as mydriasis or intermittent mydriasis were observed in children (Benjamin, 1992)

9.4.10 Haematological

No data available.

9.4.11 Immunological

No data available.

9.4.12 Metabolic

9.4.12.1 Acid-base disturbances

No data available.

9.4.12.2 Fluid and electrolyte disturbances

Light or mild dehydration may be observed, as a consequence of vomiting.

9.4.12.3 Others

No data available.

9.4.13 Allergic reactions

No data available.

9.4.14 Other clinical effects

No data available.

9.4.15 Special risks

No data available.

9.5 Other

No data available.

9.6 Summary

Symptoms onset is usually within 30 to 90 minutes, peaking at 2 to 3 hours. Initial drowsiness is followed by ataxia, confusion, agitation, illusions or even manic excitement. These periods of excitement may alternate with periods of somnolence, deep sleep or stupor. Muscle jerks, fasiculations and spasms in the extremities were observed.

10. MANAGEMENT

10.1 General principles

Treatment includes prevention of absorption of the toxins and treatment of the signs and symptoms of intoxication as they occur, especially sedation. Induction of emesis is NOT recommended because of the potential CNS depression and seizures.

10.2 Life supportive procedures and symptomatic/specific treatment

Make a proper assessment of airway, breathing, circulation and neurological status of the patient. Control convulsions with appropriate drug regimen, sedation with benzodiazepines is required (see IPCS Treatment Guidelines).

10.3 Decontamination

Emesis is not recommended.
Administer activated charcoal, most effective when administered within one hour of ingestion.
Gastrointestinal emptying and/or charcoal is however, rarely indicated and only in very recent ingestion, while the patient is asymptomatic.

10.4 Enhanced elimination

Forced diuresis
Not necessary, although possibly effective on theoretical grounds.

Hemodialysis
Although the toxins may be removed by hemodialysis (Mitchell and Lumack, 1978) this procedure is considered unnecessary in view of the good prognosis of clinical cases.

10.5 Antidote/antitoxin treatment

10.5.1 Adults

There is no specific antidote for Amanita muscaria and Amanita pantherina poisoning.

10.5.2 Children

There is no specific antidote for Amanita muscaria and Amanita pantherina

poisoning.

10.6 Management discussion

Treatment includes prevention of absorption, with activated charcoal, of the toxins and treatment of the signs and symptoms of intoxication as they occur.
Induction of emesis is not recommended because of the potential central nervous system depression and seizures.
There is no specific antidote for Amantia muscaria and pantherina poisoning.

11. ILLUSTRATIVE CASES

11.1 Case reports from literature

Accidental poisoning

There are numerous reports in the medical literature of this type of intoxication. Intoxication with A. muscaria and pantherina have more recently become more common as the result of deliberate attempts by individuals to induce hallucinations. Special recipes are even now appearing for ways to prepare a broth from these mushrooms so that one can retain their psychoactive effects without the gastrointestinal irritating effects.

Voluntary ingestion

Agnus McDonald experimented the self-administration of whole A. muscaria (1978). He prepared capsules with dried and powdered mushrooms, collected in Northern California. With 12 g of dried A. muscaria a noticeable effect was felt.

"In summary, 12 g of dry red A. muscaria produced mainly these following symptoms: a marked nausea that tapered off over the first three hours; 2) a conspicuous absence of reflective thought combined with a sense of tiredness, and 3) a slight transient euphoria around the fourth hour that alternate with and finally was overwhelmed by a general sense of fatigue. It was not an inspiring experience, and the initial nausea was so great that I had no desire to repeat it. I decided on a compromise. I made an infusion by soaking 30 g of dried mushrooms in a cup of water. Within one hour I was obviously feeling a greater effect than I had from eating 12 g. I felt again as if I were in a state of suspended animation, this time with a much stronger desire to sleep. Although my environment seemed somehow "bright", there were no hallucinations of obvious visual distortions. My stream of consciousness seemed notably empty, and when I contemplated writing down something about how I felt, I could think of nothing to say. I noticed a marked increase in my

usual level of saliva production. By 5 1/2 hours after ingestion, the effects were waning. By 7 hours later, they were nearly gone, and I succumbed to my desire to sleep. There were no sequelae the following morning".

McDonald conducted also an assay with six human volunteers who ate the 12 g does of dried-powdered Fly Agaric. All experimented nausea, although only two of them vomited. All

six experienced tiredness, and three of the six reported increased salivation. Only two of the subjects related visual distortions that might pass for low-grade hallucinations.

The experiences of Waser (1979), who experimented himself the effects of pure substances (ibotenic acid and muscimol), merit to be mentioned here:

"A 20 mg ibotenic acid dose ingested in water tastes like mushrooms, but produces little immediate action. Within half an hour a warm and slightly flushed face was noticed, without changes in blood pressure or heart rate, with no physic stimulation, but lassitude followed by sleep. A day later a migraine with classical one-sided visual disturbance developed for the first time in my life. The occipitally localized headache continued in a milder form for two weeks.

Next I turned to muscimol. A dose of 5 mg in water orally ingested had little effect except a feeling of laziness. Ten mg produced a slight intoxication after 90 minutes with dizziness, ataxia and elevated mood, psychic stimulation (in psychological tests), no hallucinations but slight changes in taste and color vision. Some myoclonic muscle twitching followed, then sleep with dreams. After two to three hours I felt normal, rested and able to undertake anything, even work. During the next night I slept well, deep and long. No other signs followed.

With 15 mg of muscimol administered orally the intoxication started after 40 minutes and was more pronounced. Dizziness made walking with closed eyes impossible, but reflexes were not changed. Speech was sometimes inarticulate and dysarthric. Appetite and taste were diminished. After a phase of stimulation, concentration became more difficult. Vision was altered by endlessly repetitioned echopictures of situations a few minutes before. Hearing became noisy and sometimes was followed by echo. Most disturbing were repeated myoclonic cramps of different muscle groups. I felt sometimes as if I had lost my legs, but never had hallucinations as vivid and colorful as with LSD. The pupils remained always the same size. After 2 hours I fell asleep,

but I cannot remember any dreams. Two hours later I awoke again and was glad that the muscle twitching was less frequent. I did not feel relaxed and fresh as after 10 mg muscimol but rather dull and uncertain. Blood pressure was only a little elevated during the psychoactive phase".

Muscimol induces a state of psychosis with confusions, dysarthria, disturbance of visual perception, illusions of colour vision, myoclonia, disorientation in place and time, weariness, fatigue and sleep. Concentration tests showed improved performance with small doses (5 mg) but diminished performance and learning with an increased number of errors with higher doses (10 to 15 mg).

12. Additional information

12.1 Specific preventive measures

12.2 Other

13. REFERENCES

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14. AUTHOR(S), REVIEWER(S) DATA (INCLUDING EACH UPDATING), COMPLETE ADDRESSES

Author: J. Piqueras
Department of Haematology & Haemotherapy
General Hospital Vall d'Hebron
Autonomous University of Barcelona
08035 Barcelona
Spain

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