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Encyclopedia of Food Mycotoxins

Springer

Martin Weidenbörner

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With 96 Figures and 9 Tables

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Preface

The main emphasis of the present book is listing all foods which have been reported to be contaminated with mycotoxins (degree of contamination, concentration, country of origin/detection). To find out quickly whether a foodstuff is contaminated by a specific mycotoxin, the contaminated foods have been listed alphabetically under "Natural Occurrence" of a mycotoxin.

Products are listed by the country in which they were investigated for mycotoxin contamination. In some cases, the country of detection is **not** necessarily the country of origin, but information was lacking concerning the country of origin of such imports in the original literature. If only "imported" occurs after the country of investigation no more data were available in the original literature. Sometimes, e.g., in the case of nuts or spices, the original literature neither contained the producing country nor the addition "imported". In these cases also no indications were given in the Encyclopedia. However, in all cases where the origin of the investigated food was known, the name of the producing country was given.

The multiple listing of some countries in connection with mycotoxin contamination of food should not implicate a high rate of mycotoxin contamination of foods in these countries but primarily documents the efforts being made to detect toxic fungal metabolites in food.

The special data concerning the mycotoxin contamination of food, e.g. 6/12, means six positive (contaminated) products from a total of twelve. Means represent the mean of positively contaminated samples, except where indicated otherwise. An entry of < x generally refers to the limit of detection. Values above this level are included in calculating the mean of all positive samples.

The data concerning mycotoxin contamination of food listed in the Encyclopedia based on results predominantly published in recommended journals and scientific books in this field (mainly the literature given at the end of the book). In the case of commonly isolated mycotoxins, e.g. aflatoxins, trichothecenes, it was not possible to consider all the results published.

In the literature, sometimes contradictory information about the mycotoxin spectrum of mold species can be found. Therefore, only the "safe" and food relevant mycotoxins of a species and not all known toxic metabolites were listed. This information mainly based on Frisvad J (1988) Fungal species and their specific production of mycotoxins. In: Samson RA, Reenen-Hoekstra ES (Eds) Introduction of Food-borne Fungi, pp 239–249. Centralbureau voor Schimmelcultures, Baarn (*Aspergillus* and *Penicillium* species), Marasas WFO, Nelson PE, Tousson TA (1984) Toxigenic *Fusarium* Species, Identity and Mycotoxicology. The Pennsylvania State University Press,

University Park, PA (*Fusarium* species), Samson RA, Hoekstra ES, Frisvad JC, Filtenborg O (1998) Introduction to Food-borne Fungi. Centraalbureau voor Schimmelcultures, Baarn (*Aspergillus* species and others). According to Ainsworth & Bisby's "Dictionary of the Fungi" all mycotoxigenic fungi listed in the Encyclopedia may be grouped easily to their corresponding family, order, phylum and kingdom.

The names used for all *Penicillium* species based on Pitt JI (1979) The Genus *Penicillium* and its Teleomorphic States *Eupenicillium* and *Talaromyces*, Academic Press, London.

Although in some cases more fungal species are known to produce a mycotoxin usually only the names of food relevant molds like *Aspergillus* spp., *Penicillium* spp. and/or *Fusarium* spp. are given.

Since in some cases various toxicological data of mycotoxins do exist for better comparison only the data of the per oral application in rats/mice (as far as possible) were chosen.

Gießen, Summer 2000 Martin Weidenbörner

Abbreviations

BGY	Bright greenish yellow (fluorescence)
bm	body mass
bw	body weight
conc	concentration
d	day(s)
EC	Esophageal cancer
ELISA	Enzyme linked immunosorbent assay
EU	European Union
FAO	Food and Agricultural Organization of the United Nations World Health Organization
FDA	United States Food and Drug Administration
GC	Gas chromatography
GC-MS	Gas chromatography-mass spectrometry
h	hour(s)
HPLC	High performance liquid chromatography
HTST	High temperature short time
IARC	International Agency for Research on Cancer
ip	intraperitoneal
iv	intravenous
JECFA	Joint Expert Committee on Food Additives
kGy	kilo Gray
LD_{50}	Lethal dosis of e.g. aflatoxin that will cause acute toxicity in 50 % of the target population
mc	moisture content
min	minutes
mp	melting point
mw	molecular weight
nc	no comment (not stated, unclear)
ND	Not detected
NOAEL	No observed adverse effect level
NMR	Nuclear magnetic resonance
po	per os
PTWI	Provisional tolerable weekly intake
sa	sample(s)
sc	subcutaneous
sqd	semi-quantitative determination

TLC	Thin-layer chromatography
tr	traces
UAE	United Arabic Emirates
WHO	World Health Organization of the United Nations

kg	kilogram
mg	milligram = 10^{-3} g; 1 mg/kg = $1:10^6$ = ppm = parts per million
µg	microgram = 10^{-6} g; 1 µg/kg = $1:10^9$ = ppb = parts per billion

l	litre
ml	millilitre = 10^{-3} l; 1 ml/l = $1:10^3$
µl	microlitre = 10^{-3} ml; 1 µl/l = $1:10^6$ = ppm = parts per million

A

AAL-toxins is the abbreviation for *Alternaria alternata* f. sp. *lycopersici* toxins which possess a "sphingosine-like" structure (see Figure AAL-toxins). AAL-toxins include the two fractions T_A and T_B. T_A ($C_{13}H_{53}NO_{15}$, MW = 679) consists of two esters (C_{13} or C_{14}) of 1,2,3-propane- tricarboxyclic acid and 1-amino-11,15-dimethylheptadeca-2,4,5,13,14-pentol. T_B ($C_{13}H_{53}NO_{13}$, MW = 647) consists of two esters (C_{13} or C_{14}) of 1,2,3-propane-tricarboxyclic acid and 1-amino-11,15-dimethylheptadeca-2,4,13,14-tetrol. These fractions contain four closely related compounds T_A-1, T_A-2, T_B-1 and T_B-2. Recently they were renamed alperisins A1, A2, B1, and B2. The alperisins are remarkably similar to the → fumonisins.

CHEMICAL DATA
Empirical formula: $C_{13}H_{53}NO_{15}$, molecular weight: 679 (T_A)
Empirical formula: $C_{13}H_{53}NO_{14}$, molecular weight: 663 (T_B)

FUNGAL SOURCES
Alternaria alternata f. sp. *lycopersici*

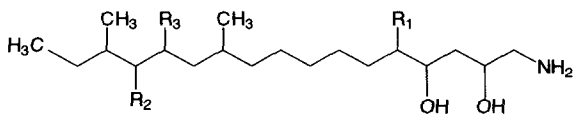
NATURAL OCCURRENCE
There are no reports on the natural occurrence of these toxins in plant products, probably because *A. alternata* f. sp. *lycopersici* is a rarely occurring pathotype of *A. alternata*. However, AAL-toxins and fumonisins (FB_1, FB_2, FB_3) occur together in spores and mycelia of *A. alternata*.

TOXICITY
Like fumonisin B_1 the AAL-toxins caused stem cancer disease in "Earlypark-7" and other susceptible tomato cultivars. In addition, AAL-toxins and the fumonisins inhibited ceramide synthase in animal cells, cell prolifeartion in rat liver and dog kidney cells.

Acacia concinna (medicinal seeds)
may contain the following → mycotoxins:
→ aflatoxin B_1
incidence: nc/nc, conc. range: 80-1130 µg / kg, country: India
→ citrinin
incidence: nc/nc, conc. range: 10-760 µg / kg, country: India

Acetoxyscirpenediol 4- or → 15-acetylscirpentriol

3-Acetyldeoxynivalenol (Syn.: deoxynivalenol monoacetate) is a 3α-acetoxy-7α,15-trihydroxy-12,13-epoxytrichothec-9-en-8-one and belongs to the → trichothecenes (→ mycotoxins) (see Figure 3-Acetyldeoxynivalenol).



AAL-TOXIN	R_1	R_2	R_3
T_A–1	OH	OH	$-O_2C-CH_2-CH(CO_2H)-CH_2-CO_2H$
T_A–2	OH	$-O_2C-CH_2-CH(CO_2H)-CH_2-CO_2H$	OH
T_B–1	H	OH	$-O_2C-CH_2-CH(CO_2H)-CH_2-CO_2H$
T_B–2	H	$-O_2C-CH_2-CH(CO_2H)-CH_2-CO_2H$	OH

AAL-toxins. *Alternaria alternata* f. sp. *lycopersici* (AAL) toxins

CHEMICAL DATA
Empirical formula: $C_{17}H_{22}O_7$, molecular weight: 338

FUNGAL SOURCES
→ Fusarium culmorum (W.G. Smith) Sacc., → Fusarium graminearum Schwabe

NATURAL OCCURRENCE
→ barley, → maize, → oats, → rye, → triticale, → wheat

TOXICITY
feed refusal (rats)
LD_{50} (ip): 49.4-49.9 mg/kg bw mice (ddS strain)

DETECTION
ELISA, TLC, GC-MS, MS

FURTHER COMMENTS
Most Japanese strains of *F. graminearum* produced 3-acetyldeoxynivalenol. The same is true for Chinese strains although the 15-acetatedeoxynivalenol could be isolated from Chinese grain.
→ deoxynivalenol

15-Acetyldeoxynivalenol belongs to the → trichothecenes (→ mycotoxins) (see Figure 15-Acetyldeoxynivalenol).

CHEMICAL DATA
Empirical formula: $C_{17}H_{22}O_7$, molecular weight: 338

FUNGAL SOURCES
→ Fusarium graminearum Schwabe

NATURAL OCCURRENCE
→ maize, → wheat

TOXICITY
In combination with → deoxynivalenol and → zearalenone the aforementioned contaminated samples caused feed refusal in swine.

DETECTION
GC-MS

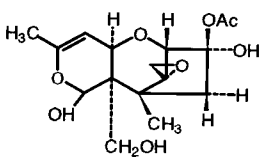
FURTHER COMMENTS
Nearly all strains of *F. graminearum* isolated in North America are able to produce this mycotoxin. This trichothecene mycotoxin occurred in naturally infected field maize samples (ca. 16,300 and 1510 µg/kg) used for feed. 15-acetyldeoxinvalenol co-occurs with → deoxynivalenol and → zearalenone.

4-Acetylnivalenol → fusarenon X

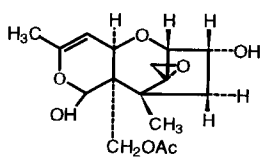
4-Acetylscirpentriol (Syn.: 15-acetylscirpentriol)

Acute aflatoxicosis → aflatoxicosis

Acute cardiac beriberi (Syn.: Shoshin-kakke) A probable → mycotoxicosis which belongs to the complex of "yellow rice diseases" (→ yellow rice disease). It was first described in Japan at the end of the last century. The disease has mainly been reported from Asian countries where → rice is a staple food and has been recognized for the past three centuries. The mold damaged rice is mainly contaminated with → Penicillium citreonigrum Dierckx (synonyms *P. citreoviride*, *P. toxicarium*). → Citreoviridin the most important mycotoxin (neurotoxin) of this mold which causes a very rapid → paralysis of the respiratory muscles. In combination with → convulsion, vomition, ascending → paralysis, and lowering



3-Acetyldeoxynivalenol



15-Acetyldeoxynivalenol

of the body temperature, the patient usually dies within a short period of 1-3 days, once the disease started. There is no method available of saving the patient from acute cardiac beriberi.
Because moldy "yellow rice" was thought to be responsible for this disease the sale of this rice was prohibited in Japan in 1910. Subsequently no more cases of acute cardiac beriberi have been reported. The disease is now of only historical interest in Japan. However, in other parts of Asia *P. citreonigrum* and its mycotoxin citreoviridin which is also produced by *P. ochrosalmoneum* may still contribute acute cardiac beriberi.
It is under discussion whether there are several types of beriberi (e.g. atropic and wet beriberi) having the same etiological origin. The difference in symptoms compared to acute cardiac beriberi may be due to dose and duration of intake of the mycotoxin. In these cases severe → paretic signs were not observed.
In contrast to acute cardiac beriberi, the cause of beriberi is a nutritional disease, an avitaminosis (vitamin B). This is proved by the following facts: the slower course of the disease, no dilation of the right ventricle, and no hypertrophy of adrenal medulla. In addition, administration of liver removed from typical shoshin-kakke patients led to the recovery of vitamin B_1-deficient animals. This indicates that adequate amounts of vitamin B_1 were present in the liver of these patients at the time of death.
However, to prove beyond doubt that citreoviridin is the cause of acute cardiac beriberi, the etiology of the chemical pathway of this neurotoxin has to be clarified.

Aflatoxicol (Abbr.: AFL, AFR_0) AFL was first reported in microorganisms (→ mycotoxins) and is the cyclopentanol derivative (2,3,6a,9a-tetrahydro-1-hydroxy-4-methoxy-cyclopenta[c]furo[3′,2′:4,5]furo[2,3-h][1]-benzopyran-11(1H)-one) of → aflatoxin B_1 (see Figure Aflatoxicol).

CHEMICAL DATA
Empirical formula: $C_{17}H_{13}O_6$, molecular weight: 313

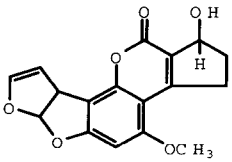
NATURAL OCCURRENCE
→ human breast milk, → pistachio nuts

TOXICITY
AFL resulted from the *in vitro* and *in vivo* metabolism of AFB_1 by soluble NADPH-dependent reductases of submitochondrial liver fractions from humans and several animal species (e.g. poultry, rabbits, trouts). A microsomal AFL-dehydrogenase catalyzes the enzymatically reversible reaction. AFL therefore may represent a storage reservoir of AFB_1 that enhances the toxicity of AFB_1. Mice or rats which are relatively resistant to AFB_1 produce only very little AFL. Therefore, the minor rate of transformation might be a determinant in the susceptibility of animals to the acute toxic action of AFB_1. AFL is reported to be 18 times less toxic than AFB_1 in the duckling biliary → hyperplasia assay. In Fischer rats AFL shows nearly one half the hepatocarcinogenic potency of AFB_1. Carcinogenicity and mutagenicity (→ mutagenic) were almost the same as for AFB_1 in rainbow trout and in *Salmonella typhimurium*, respectively. Biological activity of aflatoxicol B is unknown.

DETECTION
see → aflatoxins

FURTHER COMMENTS
Two stereoisomers of AFL are known, the "A" isomer, also referred to as aflatoxin R_0, and the "B" isomer. The latter is only formed by microorganisms whereas AFR_0 also resulted from animal metabolism.

Aflatoxicol H_1 (Abbr.: $AFLH_1$) is the hydroxylated oxidative metabolite of



Aflatoxicol

→ aflatoxicol. It resulted from the metabolism of → aflatoxin B_1 by microsomal and soluble enzymes of primate and human liver and from → aflatoxin Q_1 incubated with cytosol enzymes.

TOXICITY

No toxicity has been reported in chick embryos and bacteria but it was → mutagenic (2% that of AFB_1) in the case of *Salmonella typhimurium.*

Aflatoxicosis is caused by → Aspergillus flavus Link and → Aspergillus parasiticus Speare due to the formation of → aflatoxins. Although these molds are of ubiquitous distribution, *A. parasiticus* predominates in tropical and subtropical countries. These → storage fungi invade seeds and → grains, particularly → peanuts, → maize (before harvest), and edible → nuts. Saprophytic growth on a wide range of foodstuffs is possible. Certain climatic conditions favour preharvest invasion and aflatoxin contamination of maize and peanuts. Countries with colder climates do not support aflatoxin production. Here, aflatoxicosis may be imported by contaminated feeds and foods. Species which are mainly affected by aflatoxins are humans, → cattle, dogs, → poultry, pigs, and trout.

The aflatoxicosis can be divided into two forms: primary aflatoxicosis with the acute and chronic forms, and secondary aflatoxicosis.

Acute aflatoxicosis results from high and moderate aflatoxin concentrations which cause the death of the animal. The main symptoms are: fatty, pale, and decolorized livers; interference of normal blood clotting mechanisms with subsequent hemorrhages (→ haemorrhage); decrease in total serum proteins and increase in certain serum enzymes of the liver; accumulation of blood in the gastrointestinal canal. In addition, lesions of the kidney (glomerular → nephritis) and congestions (→ congestion) in the lungs are possible.

The most severe case of acute aflatoxicosis has been observed in north-west India (1974). Ca. 25% of the exposed population (397 affected, 106 died) died after eating molded → maize with aflatoxin levels ranging from 6250 to 15,600 µg / kg. In contrast to females males were affected twice as often. Patients suffered from → icterus, in general vomiting and → anorexia preceded. → Ascites and → edema of the lower extremities subsequently occurred. In another case of acute aflatoxicosis (Kenya) patients showed similar clinical signs. Pathological changes in the liver were characteristic of toxic → hepatitis. In addition, three children in the Province of Taiwan, China and one child in Uganda died from acute liver necrosis. Their death was associated with the ingestion of → rice (200 µg aflatoxins / kg) and → cassava (1700 µg aflatoxins / kg), respectively, which most probably caused the disease. The reported outbreaks are only seen as the tip of the iceberg of worldwide occurring aflatoxicosis.

Chronic aflatoxicosis is caused by long term consumption of moderate to low aflatoxin concentrations. Much more serious veterinary problem may arise compared to acute aflatoxicosis. Symptoms are: liver congestions with hemorrhagic and necrotic regions; proliferation of the hepatic parenchyma and epithelial cells of the → bile duct; kidney congestion accompanied by occasional hemorrhagic → enteritis. Reduced feed efficiency and retarded growth rate are common, the

reproducive efficiency is decreased. Development of liver cancer (e.g. hatchery-reared trout) may result from long-term consumption of low levels of → aflatoxins as extremely potent hepatocarcinogenes.

Secondary aflatoxicosis (low aflatoxin concentrations) impairs the native resistance by reduction of phagocytic effectiveness of macrophages and nonspecific humoral substances (complements). The immunosuppressive effects of aflatoxins predispose animals to secondary infections by bacteria, fungi and viruses. Epidemiological studies in different parts of Africa and Asia show that aflatoxins may cause liver cancer in humans, albeit in combination with the hepatitis B virus. People e.g. living in Kenya, Mozambique, Swaziland and Thailand showed a high incidence of hepatic carcinomas. In these countries → foods and feeds are often contaminated with aflatoxins. In the Philippines AFM_1 has been detected in the 24 h urine samples of people who ingested → peanut butter containing aflatoxin. A level as high as 10-15 µg → aflatoxin B_1 in the diet seems to be sufficient for detection of → aflatoxin M_1 in urine.

Aflatoxin B_1 (Abbr.: AFB_1) is a 2,3,6a,9a-tetrahydro-4-methoxy-cyclopenta[c]furo[3´,2´:4,5]furo[2,3-h][1]-benzopyran-1,11-dione (→ mycotoxins) generally produced in the largest amount both in nature and in culture (see Figure Aflatoxin B_1).

CHEMICAL DATA

Empirical formula: $C_{17}H_{12}O_6$, molecular weight: 312

FUNGAL SOURCES

→ Aspergillus flavus Link, → Aspergillus nomius Kurtzman et al. → Aspergillus parasiticus Speare.

NATURAL OCCURRENCE

→ Acacia concinna, almonds, → ammi, → apples, → baby food, → bacon, → barley, → bean jam, → beans, → beefburger, → beer, burukutu, → beer, pito, → beer, sorghum, → Blepharis edulis, → bondakaledkai, → Brazil nuts, → bread, → buckwheat, → buckwheat flour, → cabbage, → Caesalpinea digyna, → Cassia fistula, → cardamom, → cardamom, greater, → cashew nuts, → cayenne pepper, → cereals, → cheese, → cheese, blue, → cheese, pepper, → cheese, Tilsit, → cheese rind, → cheese trimmings, → cherries, → chicken liver, → cocoa beans, → congressbele, → copra, → coriander, → corn flakes, → cumin, → curcuma, → dairy products, → duck, → emu aran, → equsi meal, → fennel, → fenugreek, figs, → galgant, → garlic, → garlic / onions, → ginger, → groundnut toffee, → ham, → hare, → hazelnuts, → hot dog, → human breast milk, → Hydnocarpus laurifolia, → Indian cassia, → ingwer, → job's-tears, → kubeba, → lemmons, → lentils, → libritos, → lineseed oil, → lineseeds, → mackarel, → maize flour, → maize grits, → mango, → meat, luncheon, → milk, → milk powder, → miso, → muesli, → nutmeg, → nuts (mixed), → oats, → oat flakes, → ogbono, → ogiliugba, → ogoro, → oil seeds, → oil seed rape, → olive oil, → olives, → oranges, → pastries, → peaches, → peanut brittle, → peanut butter, → peanut oil, → peanut products, → peas, → pecans, → persipan, → pheasants, → pig liver, → pine nuts, → Piper betle, → pipian paste, → pop corn, → rice, → rice cake, → roe deer, → rye, → sago, → salami, → sausages, → shrimp, → sorghum, → soybean, → spices, → sunflower seeds, → sunflower seed oil, → taro, → tomatoes, → tomato ketchup, → tumeric, → vegetables, walnuts, → wheat

For further information see → aflatoxins and → aflatoxin G_2.

Plant commodities which may be highly contaminated with → aflatoxins are → nuts such as → peanuts, Brazil nuts, → pistachio nuts as well as copra,

→ maize, and cottonseeds. Agricultural products with a slightly lower potential of aflatoxin contamination are → almonds, → figs, pecans, spices, and → walnuts. Animal products are less likely substrates, e.g. → milk, animal tissue.

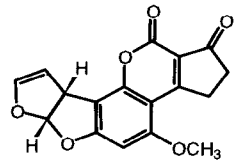
TOXICITY

It is the strongest natural carcinogen and the main hepatocarcinogen in animals, although effects vary with species, age, sex, and general nutrition. For example trout, duckling, and pig, are highly susceptible, whereas e.g. sheep and → cattle, are more resisant. The liver is the primary organ affected (induction of liver lesions, liver carcinoma, bile duct proliferation). In Fischer rats and rainbow trout AFB_1 is the most potent hepatocarcinogen. Changes in other organs (e.g. kidneys, lung) have been observed. From primate data the doses of AFB_1 required to cause acute → aflatoxicosis in humans were extrapolated. It was estimated that the intake of → food contaminated with 1700 µg / kg bw for a short time could be sufficient for severe liver damage while a single dose of 75,000 µg / kg bw could result in death. Apparent acute aflatoxicosis would not occur if 340 µg AFB_1 / kg bw is consumed per day. In the USA the ingestion of AFB_1 with maize and peanut products contributes to a greater risk of hepatic cancer in adults than AFM_1 in milk and → dairy products. In comparison to these agricultural products the human intake of aflatoxins by meat and meat products is negligible. The IARC (1993) evaluated AFB_1 as a Class 1 human carcinogen.

LD_{50} (po): 5.5-7.2 mg / kg bw male rats (weight: 100 g), 17.9 mg / kg bw female rats (weight: 150 g)

DETECTION

see → aflatoxins



Aflatoxin B_1

FURTHER COMMENTS

Spiking commercially manufactured cigarettes with AFB_1 (100-300 µg / kg) did not result in any contamination of the gas phase or the ashes.

Aflatoxin B_2 (Abbr.: AFB_2) is the dihydro derivative of → aflatoxin B_1 (2,3,6a,8,9a-hexahydro-4-methoxy-cyclopenta[c]furo[3′,2′:4,5]furo[2,3-h][1]-benzopyran-1,11-dione) and synthesized by the reduction of the single double bond in the terminal dihydrofuran ring (→ mycotoxins) (see Figure Aflatoxin B_2).

CHEMICAL DATA

Empirical formula: $C_{17}H_{14}O_6$, molecular weight: 314

FUNGAL SOURCES

→ Aspergillus flavus Link, → Aspergillus nomius Kurtzman et al., → Aspergillus parasiticus Speare

NATURAL OCCURRENCE

AFB_2 occurs in the same commodities as AFB_1 but AFB_2 is found in smaller amounts. Via milk it is secreted as → aflatoxin M_2.

TOXICITY

This carcinogenic (?) and → genotoxic substance shows toxic properties similar to AFB_1 but has markedly reduced toxic potency in comparison to AFB_1. Instead of 3.9 µg AFB_1 50 µg AFB_2 are necessary to produce similar bile duct proliferation in ducklings. Estimated lethal dose for human beings 1-10 mg / kg.

LD_{50} (po): 84.8 µg / 50 g bw one-day old ducklings

DETECTION
see → aflatoxins

Aflatoxin B_{2a} (Abbr.: AFB_{2a}) (Syn.: AFB_1 hemiacetyl, aflatoxin W, hydroxydihydroaflatoxin B_1) represents the corresponding "water adduct" (2-hydroxy derivative) of → aflatoxin B_1 (→ mycotoxins) which resulted from the hydration of the 2,3-vinyl ether bond of this aflatoxin (2,3,6a,8,9,9a-hexahydro-8-hydroxy-4-methoxy-cyclopenta[c]furo[3´,2´:4,5]-furo[2,3-h][1]-benzopyran-1,11-dione). Conversion occurs rapidly under mildly acidic conditions. Although this blue fluorescing compound is 60-100 (200) times less toxic to ducklings it may be dehydrated to the highly toxic AFB_1. Furthermore, AFB_{2a} is a biotransformation / detoxification product of AFB_1 produced by hepatic microsomes *in vitro* of some animals (e.g. mouse, guinea-pig, avian). It is under discussion whether AFB_{2a} reacts readily with free amino groups of functional proteins (see Figure Aflatoxin B_{2a}).

CHEMICAL DATA
Empirical formula: $C_{17}H_{14}O_7$, molecular weight: 330

FUNGAL SOURCES
→ *Aspergillus flavus* Link, → *Aspergillus parasiticus* Speare

TOXICITY
In the standard duckling assay (initiation of → bile duct proliferation) both AFB_{2a} and AFG_{2a} are very much less toxic than AFB_1 (60-100 times) after oral application. In Khaki Campbell ducklings (day-old) no acute toxicity was noted at levels up to 1200 µg / duckling.

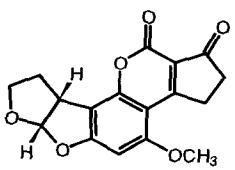
Aflatoxin B_3 (Abbr.: AFB_3) (Syn.: parasiticol) Older cultures of → *Aspergillus flavus* Link and → *Aspergillus parasiticus* Speare may contain high amounts of this 6-methoxy-7-(2´hydroxyethyl) difurocoumarin (7a,10a-dihydro-4-(2-hydroxyethyl)-5-methoxy-2H-furo[3´,2´:4,5]-furo[2,3-h]-1-benzopyran-2-one) as a possible precursor of → aflatoxins. On the other hand it seems to be the first step in the biological degradation of → aflatoxin G_1 by e.g. *Rhizopus* spp. (see Figure Aflatoxin B_3).

CHEMICAL DATA
Empirical formula: $C_{16}H_{14}O_6$, molecular weight: 302

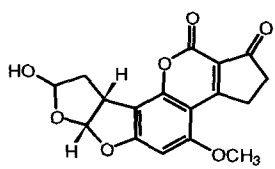
TOXICITY
Parasiticol has the same acute toxicity to ducklings as → aflatoxin B_1. However, the tendency to cause biliary → hyperplasia is low. In chick embryo studies toxicity was only 1/100 than that of AFB_1.

Aflatoxin D_1 is a major product (10-30%) - besides the 206-molecular weight compound (3-10%) - from the reaction of aflatoxin B_1 with heated ammonium hydroxide. aflatoxins

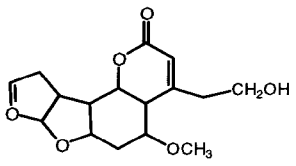
Aflatoxin G_1 is a mycotoxin (→ mycotoxins) that has a structure very similar to that of → aflatoxin B_1 (3,4,7a,10a-tetrahydro-5-methoxy-1H,12H-furo[3´,2´:4,5]-furo[2,3-h]pyrano[3,4-c][1]-benzopyran-1,12-dione) but there are two lactone functions rather than one and the two



Aflatoxin B_2



Aflatoxin B_{2a}



Aflatoxin B_3

dihydrofuran rings are fused in a *cis* configuration (see Figure Aflatoxin G_1).

CHEMICAL DATA
Empirical formula: $C_{17}H_{12}O_7$, molecular weight: 328

FUNGAL SOURCES
→ Aspergillus flavus Link, → Aspergillus nomius Kurtzman et al., → Aspergillus parasiticus Speare

NATURAL OCCURRENCE
Same commodities as AFB_1, in addition, → celery seeds.

TOXICITY
This carcinogenic (liver- and kidney carcinoma) and → genotoxic mycotoxin possesses a similar toxicity to that of AFB_1, although acute toxicity was less than AFB_1 but greater than AFB_2. It is a slightly less potent liver carcinogen but a slightly more potent kidney carcinogen, with a comparable carcinogenic potency to aflatoxin B_1 i.e. within a factor of 10. Ducklings treated with AFG_1 showed the same lesions as AFB_1-treated animals. The zone in affected rat liver lobule was the same as in B_1. However, a consistent pattern as seen with AFB_1 was absent. The LD_{50} in the rat was twice that of AFB_1.

DETECTION
see → aflatoxins

FURTHER COMMENTS
Optimum temperature for AFG_1 production is 30 °C.

Aflatoxin G_2 is the dihydro derivative of → aflatoxin G_1 (3,4,7a,9,10,10a-hexahydro-5-methoxy-1H,12H-furo[3′,2′:4,5]-furo[2,3-h]pyrano[3,4-c][1]-benzopyran-1,12-dione) and synthesized by the reduction of the single double bond in the terminal dihydrofuran ring (see Figure Aflatoxin G_2).

CHEMICAL DATA
Empirical formula: $C_{17}H_{14}O_7$, molecular weight: 330

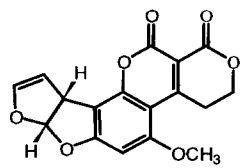
FUNGAL SOURCES
→ Aspergillus flavus Link, → Aspergillus nomius Kurtzman et al., → Aspergillus parasiticus Speare

NATURAL OCCURRENCE
→ beans, → brazil nuts, → cocoa beans, → cumin, → ginger, → Indian cassia, → lemons, → maize, → mango, → olive oil, → oranges, → peanuts, → peanut brittle, → pepper, → pop corn, → rice, → sausages, → sesame seeds, → shoyu, → sunflower seeds, → tumeric, → walnuts
For further information see → aflatoxins and → aflatoxin B_1.

TOXICITY
This carcinogenic (?) and → genotoxic mycotoxins possesses the least acute toxicity of the 4 major naturally occurring → aflatoxins.
LD_{50} (po): 172.5 µg / 50 g bw one day old ducklings.

DETECTION
see → aflatoxins

Aflatoxin G_{2a} (Abbr.: AFG_{2a}) Aflatoxin G_1 is converted by strong acids to the corresponding "water adduct" (2-hydroxy derivative = AFG_{2a}) which retains its



Aflatoxin G_1

toxicity (3,4,7a,9,10,10a-hexahydro-9-hydroxy-5-methoxy-1H,12H-furo[3′,2′:4,5]furo[2,3-h]pyrano[3,4-c][1]-benzopyran-1,12-dione). Livers of certain animals ingesting → aflatoxin G_1 produce AFG_{2a} which might be a detoxification mechanism (see Figure Aflatoxin G_{2a}).

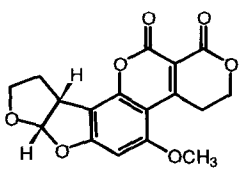
CHEMICAL DATA
Empirical formula: $C_{17}H_{14}O_7$, molecular weight: 330

FUNGAL SOURCES
→ Aspergillus flavus Link, → Aspergillus parasiticus Speare

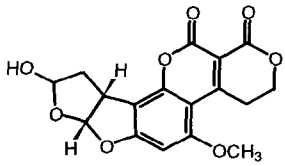
TOXICITY
No significant differences in growth and characteristic liver lesions occurred in day-old Khaki Cambell ducklings (1600 µg / duckling). LD_{50} of AFB_1 in the same assay was 18.2 µg / duckling.

Aflatoxin GM_1 is a 4-hydroxylated derivative of → aflatoxin G_1 but only minor quantities have been detected in → Aspergillus flavus Link cultures.

Aflatoxin M_1 (Abbr.: AFM_1) is the 4-hydroxylated derivative of → aflatoxin B_1 (2,3,6a,9a-tetrahydro-1,9a-dihydroxy-4-methoxy-cyclopenta[c]furo[3′,2′:4,5]-furo[2,3-h][1]-benzopyran-11(1H)-one). It is found in liver, kidneys, blood, bile, feces, urine, and → milk of mammals (→ mycotoxins). Hydroxylation mainly occurs in the liver in the benzylic position at the junction of the two furan rings. It was the first → aflatoxin B_1 metabolite identified which was originally (early 1960s) found in cow′s milk. Structural elucidation was first achieved in 1966. Subsequently isolation of AFM_1 has also been reported from other kinds of milk as well as → dairy products (see Figure Aflatoxin M_1).



Aflatoxin G_2



Aflatoxin G_{2a}

CHEMICAL DATA
Empirical formula: $C_{17}H_{12}O_7$, molecular weight: 328

FUNGAL SOURCES
→ Aspergillus flavus Link, → Aspergillus parasiticus Speare

NATURAL OCCURRENCE
→ cheese, → cheese, blue, → cheese, Blue Haverti, → cheese, Brie, → cheese, butter, → cheese, Camembert, → cheese, Camembert & Brie, → cheese, Cheddar, → cheese, Cheshire, → cheese, Chester, → cheese, Cottage, → cheese, Comte, → cheese, Cream, → cheese, Double Gloucester, → cheese, Edam, → cheese, Emmental, → cheese, Fresh, → cheese, Gouda, → cheese, Grana Padano, → cheese, Lancashire, → cheese, Leicester, → cheese, Maribo, → cheese, Mozarella, → cheese, Parmesan, → cheese, Romadur, → cheese, Samsoe, → cheese, Stilton, → cheese, Wensleydale, → cheese, Wine, → cream, full, → human breast milk, → milk, → milk powder, → milk, pasteurized, → milk, sterilized, → milk, UHT, → milk, camel, → pistachio nuts, → soybean milk powder, → whey powder, → yogurt
Besides milk and dairy products this mycotoxin (→ mycotoxins) is also a contaminant of stored white and yellow → maize, freshly harvested yellow maize, and acid treated stored yellow maize (1-