

## Toxicidad por Micotoxinas en pequeños animales - Una revisión

### GENERAL (INCIDENCIA / METABOLISMO)

#### **Determination of mycotoxins in pet foods sold for domestic pets and wild birds using linked-column immunoassay clean-up and HPLC.**

Scudamore KA, Hetmanski MT, Nawaz S, Naylor J, Rainbird S

*Food Addit Contam* 1997 Feb-Mar 14:175-86

#### Abstract

A technique referred to as 'linked-column immunoassay clean-up', was developed for the simultaneous determination of aflatoxins and ochratoxin A in a range of dry cereal-based pet foods and wild bird food. In addition a method also based on clean-up using immunoaffinity columns was used for the determination of fumonisin mycotoxins in samples known or suspected to contain maize. One hundred samples of pet foods consisting of 35 samples of domestic bird seeds and 15 samples of wild bird food were examined for aflatoxins B1, B2, G1 and G2 and ochratoxin A. Twenty samples of these samples were also examined for fumonisins B1 and B2. Limits of detection were about 0.5 micrograms/kg for each aflatoxin and ochratoxin A and 3 and 8 micrograms/kg for fumonisins B1 and B2 respectively. Eighty-four percent of the samples contained no measurable concentrations of mycotoxins. A low level of aflatoxin B1 was found in a sample of cat food and a concentration of 370 micrograms/kg aflatoxin B1 in one sample of peanuts marketed for wild birds. Ochratoxin A was detected in 10% of samples but in low concentrations, the highest of 7 micrograms/kg occurring in a sample of bird food. **Fumonisin were found in 30% of the 20 samples tested with a maximum of 750 micrograms/kg total fumonisins being found in a sample of cat food.** Five samples each of dog and cat foods were examined for mould count and fungal species as received and after storage under controlled simulated damp conditions. Mould counts in all 10 samples examined were very low when received. The samples in which moisture content had been increased contained visible mould after storage. *Penicillium*, *Eurotium* and *Aspergillus* were the predominant species. However, no ochratoxin A or aflatoxins were detected in these spoiled samples.

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#### **Species differences in the metabolism of aflatoxin B1.**

##### **Emafo PO**

*Afr J Med Med Sci* 1976 Mar 5:55-62

#### Abstract

The metabolism of aflatoxin B1 in a number of animals was investigated. Aflatoxin B1 is metabolized relatively more slowly in liver slices of sheep than in the mouse, goat, guinea-pig, rabbit and golden hamster. The rate of metabolism of the toxin by the 10,000 g supernatant is faster than the metabolism by liver slices. This may be as a result of the substrate not penetrating the liver cells readily. Species difference exist in the in vitro metabolism of aflatoxin B1 by hydroxylation and demethylation. The sheep and White Rock cockerel demethylate aflatoxin B1 poorly but the dog and duck do not demethylate the toxin at all. Of the animals studied, the duck, mouse and White Rock cockerel do not produce aflatoxin M1 at all. The sheep and **dog produce aflatoxin M1 in comparatively large amounts**, while the rat, goat and golden hamster produce aflatoxin M1 in smaller quantities.

#### **Mycoflora in commercial pet foods.**

Bueno DJ, Silva JO, Oliver G

*J Food Prot* 2001 May 64:741-3

#### Abstract

This article reports on the identification of mycoflora of 21 dry pet foods (12 belonging to dogs and 9 to cats) that corresponded to 8 commercial brands made in Argentina and imported. The isolation frequency and relative density of the prevalent fungal genera are compared too. Ten genera and fungi classified as *Mycelia sterilia* were identified. The predominant genera were *Aspergillus* (62%), *Rhizopus* (48%), and *Mucor* (38%). The most prevalent among *Aspergillus* was *Aspergillus flavus* followed by *Aspergillus niger* and *Aspergillus terreus*. The predominant

Mucor was *Mucor racemosus* followed by *Mucor plumbeus* and *Mucor globosus*. The moisture content of these foods ranged from 5.6 to 10.0% and from 7.2 to 9.9% for dog and cat foods, respectively. A greater moisture content in food for the senior category (9.5 +/- 0.2) was observed only in comparison to adult and kitten/puppy. **If the moisture content can be maintained at these levels, mold growth would be prevented or at least it would remain at an insignificant level. Some genera and species isolated and identified from the foods analyzed are potentially producing toxins, which are known as mycotoxins. This involves a risk for animal health.**

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## **Aflatoxinas**

### **Aflatoxin in dog and horse feeds in turkey.**

Gunsen U, Yaroglu T

*Vet Hum Toxicol* 2002 Apr 44:113-4

Aflatoxin levels were determined by ELISA in 18 dog and 20 horse feed samples, collected from different firms from June 2000 to June 2001 in Turkey. The minimum and maximum levels of total aflatoxin in the dog and horse feeds were <1.75-20 microg/kg and <1.75-14 microg/kg, respectively; 3/18 dog feed samples (16.7%) and 2/20 horse feed samples (10%) exceeded the Turkish tolerance limit of 10 microg/kg in food or feed.

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### **Acute toxicity of aflatoxin B1 and rubratoxin B in dogs.**

Hayes AW, Williams WL

*J Environ Pathol Toxicol* 1978 Sep-Oct 1:59-70

Abstract

The effect of ip administrated aflatoxin B1 and rubratoxin B, singly and in combination, on dogs was determined by serum tests, by observations of clinical signs and survival times, and by evaluation of gross and microscopic lesions. **The dog is sensitive to the toxic effects of both mycotoxins.** Glutamic-oxaloacetic transaminase, lactic dehydrogenase and alkaline phosphatase activities and survival time varied in relation to dose and to the mycotoxin(s) administered. All three plasma enzymes were elevated regardless of dose with the combination of aflatoxin B1/rubratoxin B at 24 hr after dosing, except LDH, which was within the normal range but only at the lowest dose level. Several serum constituents including BUN, cholesterol, uric acid, and total bilirubin were elevated, whereas serum glucose was depressed in dogs treated with the multiple-toxin regimen; these changes were not seen in dogs given only aflatoxin B1 but were characteristic in rubratoxin-treated animals. In general, gross findings at necropsy were similar in all dogs regardless of the dose regimen. A striking similarity existed in the histologic changes observed between lesions experimentally induced by the mycotoxin combination and those lesions reported for dogs fed toxic feed in laboratory studies or in natural cases of hepatitis X. Of particular similarity were the severe kidney lesions observed in dogs exposed to the mycotoxin combination and kidney lesions reported in natural outbreaks of hepatitis X. There can be little doubt of an association between hepatitis X and aflatoxin B1, although it is apparent that the disease probably involves more than a single toxic factor. Our results suggest that hepatitis X in dogs includes aflatoxin B1 as a primary etiological factor but that rubratoxin B also may be involved.

### **Canine aflatoxicosis.**

Ketterer PJ, Williams ES, Blaney BJ, Connole MD

*Aust Vet J* 1975 Jul 51:355-7

Abstract

Poisoning with aflatoxin derived from mouldy bread was confirmed as the cause of death of one dog and was suspected as the cause of death of two other dogs on the same ration. A jaundiced carcass, firm bile-stained liver and haemorrhage into the gastro-intestinal tract were seen at autopsy. Swelling and foamy vacuolation of hepatocytes due to fatty infiltration, marked periportal proliferation of bile ductules and some periacinar necrosis were the microscopic

changes seen in the liver. *Aspergillus flavus* was isolated from the mouldy bread and also from a sample of vomitus. **Aflatoxin B1, 6.7 ppm, was detected in the mouldy bread and extremely high levels of 100 ppm of aflatoxin B1 and 40 ppm of aflatoxin G1 were present in a sample of vomitus.**

#### **Canine aflatoxicosis: a continuing problem.**

Liggett AD, Colvin BM, Beaver RW, Wilson DM

*Vet Hum Toxicol* 1986 Oct 28:428-30

Abstract

Aflatoxins are **hepatotoxic in many species including dogs**. In two separate outbreaks, the primary signalment was high morbidity and mortality in hunting dogs presenting with clinical signs of icterus, anorexia and listlessness. Preliminary laboratory examinations revealed toxic hepatitis, bilirubinuria and anemia. In the first case, a feed sample was not available and the diagnosis was established by confirming the presence of significant levels of aflatoxin B1 in tissues. In the second case, cornmeal utilized in formulating the ration contained 511 ng aflatoxin B1 and B2/g. **These cases illustrate that aflatoxicosis is a continuing problem despite widespread awareness and testing for aflatoxin.**

#### **DON**

#### **Overt signs of toxicity to dogs and cats of dietary deoxynivalenol.**

Hughes DM, Gahl MJ, Graham CH, Grieb SL

*J Anim Sci* 1999 Mar 77:693-700

Abstract

Studies were conducted to determine the dietary amounts of deoxynivalenol (DON; vomitoxin) in dog and cat food that are required to produce overt signs of toxicity (e.g., vomiting or reduced food intake). Wheat naturally contaminated with 37 mg of DON/kg was used to manufacture pet foods containing 0, 1, 2, 4, 6, 8, and 10 mg of DON/kg. Deoxynivalenol concentration in pet food following manufacture was unchanged, indicating that the toxin was stable during conventional extrusion processing. Dogs previously fed DON-contaminated food were able to preferentially select uncontaminated food. Dogs not previously exposed to DON-contaminated food consumed equal quantities of contaminated and uncontaminated food. There was no effect of 6 mg of DON/kg on dog food digestibility. **Food intake of dogs was significantly reduced by DON concentrations greater than 4.5 +/- 1.7 mg/kg, and DON greater than 7.7 +/- 1.1 mg/kg reduced cat food intake. Vomiting by dogs and cats was commonly observed at the 8 and 10 mg DON levels.**

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#### **Stability of deoxynivalenol in heat-treated foods.**

Wolf-Hall CE, Hanna MA, Bullerman LB

*J Food Prot* 1999 Aug 62:962-4

Abstract

The effects of high-temperature and -pressure processing of foods spiked with deoxynivalenol (DON) were examined. In extruded corn grits, extruded dry dog food, and autoclaved moist dog food, there were no significant reductions ( $P < 0.05$ ) in DON after processing. Autoclaved cream-style corn showed a reduction in DON of only 12%. Overall, DON was stable to the high temperature and pressure processes tested. The use of an alpha-amylase in the extraction method for analysis by an enzyme-linked immunosorbent assay (ELISA) improved the recovery of DON from the spiked extruded and autoclaved products by as much as 26% over the standard ELISA method.

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#### **Ochratoxinas**

#### **Ochratoxin A and citrinin induced nephrosis in Beagle dogs. I. Clinical and clinicopathological features.**

Kitchen DN, Carlton WW, Tuite J  
*Vet Pathol* 1977 Mar 14:154-72

Abstract

Ochratoxin A and citrinin, both mycotoxins, were given separately and combined to young Beagle dogs for 14 days. Ochratoxin A, 0.1 and 0.2 mg/kg, was given by capsule, and citrinin, 5 and 10 mg/kg, was dissolved in ethanol and given by intraperitoneal injection. **Clinical signs of toxicosis in dogs given 10 mg/kg citrinin and the higher combined doses included anorexia, retching, tenesmus, weight loss, prostration and death. Severity of the clinical disease and mortality were increased when the mycotoxins were combined, which indicated synergism.** The clinicopathological abnormalities reflected renal damage, in that glutamic oxaloacetic transaminase and lactic dehydrogenase increased in the urine of the dogs with clinical signs of poisoning. Serum lactic dehydrogenase was increased in dogs given 10 mg/kg citrinin. Cellular and granular casts, ketones, protein and glucose were in the urine of dogs given large doses of citrinin alone or combined with ochratoxin A. Serum concentrations of sodium, potassium and chloride in the dogs given high doses of each group.

**Ochratoxin A and citrinin induced nephrosis in Beagle dogs. II. Pathology.**

Kitchen DN, Carlton WW, Tuite J  
*Vet Pathol* 1977 May 14:261-72

Abstract

Beagle dogs were given ochratoxin A (0.1 and 0.2 mg/kg) and citrinin (5 and 10 mg/kg) alone and in two dose combinations for 14 days. The gross lesions included focal peritonitis and intestinal intussusceptions in dogs given citrinin. **Changes in the kidneys of dogs given ochratoxin A were degeneration and necrosis with desquamation of tubular epithelial cells, primarily in the straight segment of the proximal tubules.** Dogs given 10 mg/kg citrinin had similar changes in the distal tubules and collecting ducts. Dogs given combined doses of citrinin and ochratoxin A had degeneration and necrosis in proximal and distal tubules, and in thin segments and the collecting ducts; there were desquamated cells and granular casts in the lumina. Dogs given ochratoxin A had necrosis of lymphoid tissues in the spleen, tonsil, thymus, peripheral lymph nodes and lymph nodules of the ileum, colon and rectum. There was ulceration of the mucosa of the intestine in dogs given large combined doses of ochratoxin A and citrinin.

**Ochratoxin A and citrinin induced nephrosis in Beagle dogs. III. Terminal renal ultrastructural alterations.**

Kitchen DN, Carlton WW, Hinsman EJ  
*Vet Pathol* 1977 Jul 14:392-406

Abstract

The extent and type of renal ultrastructural changes in Beagle dogs varied with the administration of ochratoxin A and citrinin alone and in the two dosage combinations. The three predominant changes were cytoplasmic vacuolation, myelin figure formation and lesions designated as cytoplasmic disarray. These changes were mainly of the endomembrane system of the tubular epithelial cells. Cytoplasmic vacuoles were within proximal and distal tubules and collecting ducts and were most numerous in dogs given 10 mg/kg citrinin. Vacuolation of similar distribution, but less severe, was seen in renal tubular cells of dogs given the higher dose of the combined mycotoxins (0.2 mg/kg ochratoxin A + 10 mg/kg citrinin). This damage was limited to the proximal tubular cells in dogs given only ochratoxin A (0.1 or 0.2 mg/kg). Myelin figures were in proximal epithelial cells of dogs given ochratoxin A alone or combined with citrinin. There was cytoplasmic disarray in dogs of all groups except for dogs given 5 mg/kg citrinin. This lesions was usually limited to the proximal tubules. The lesions, however, was found in cells of the distal tubules of dogs given 10 mg/kg citrinin alone.