CPD article

Aflatoxicosis in dogs

Aflatoxins are toxic, naturally occurring bisfuranocoumarin compounds produced by certain strains of the moulds *Aspergillus flavus*, *A. parasiticus* and *A. nomius*. Aflatoxin metabolites cause hepatotoxicity by reacting with macromolecules (including DNA and proteins) to cause fatty liver or liver necrosis. Most cases involve dog food or, less commonly, ingestion of mouldy bread. Periodic outbreaks are reported in dogs, most recently at the end of 2020 to early 2021 in the US. Multiple dogs may be involved in incidents and the dogs usually present with gastrointestinal signs, lethargy, melaena and jaundice. Diagnosis is based on a history of possible ingestion and laboratory confirmation of aflatoxin(s) in suspect material. In the liver the typical histological changes are centrilobular necrosis of the liver and bile duct proliferation. Treatment of aflatoxicosis in dogs in supportive, with management of liver failure. Prognosis depends on the severity of liver damage, but mortality rates in dogs with aflatoxicosis are high.

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After thousands of ducklings, turkeys, chickens and swine died in England between 1960 and 1961, extensive investigation was undertaken to establish the cause. Suspicion fell on Brazilian peanut meal, which had recently been imported and used as animal feed (Austwick, 1978), resulting in the eventual discovery of compounds known as aflatoxins produced by the mould *Aspergillus*. The name aflatoxin is a contraction of '*A. flavus*' and 'toxin' (Denning, 1987) and over a dozen have been recognised.

The most common aflatoxins are B_1, B_2, G_1, G_2, M_1 and M_2. Aflatoxins fluoresce on exposure to ultraviolet light and B_1, B_2, G_1 and G_2 are distinguished on the basis of their colour (B for blue and G for green). Aflatoxins B_2 and G_2 are the dihydroxy derivatives of B_1 and G_1 respectively. Aflatoxin M_1 and aflatoxin M_2 are metabolites of aflatoxins B_1 and B_2 respectively, which were originally identified in milk (World Health Organization, 1979). Aflatoxin B_1 is the most commonly occurring aflatoxin.

Outbreaks

Following the huge outbreak in the early 1960s in England, it was suspected that deaths in large numbers of turkey poults in the late 1950s had also been a result of aflatoxicosis. Similarly, a condition known as 'hepatitis X' in dogs occurred in the early 1950s in the US (Newberne et al, 1955) and was subsequently identified as aflatoxicosis. Since then, numerous outbreaks of aflatoxicosis have been reported in dogs (*Table 1*), most recently in late 2020 to early 2021 in the US (Food and Drug Administration, 2021). Incidents may be nationwide (*Box 1*) or more localised (*Box 2*).

Sources

Aspergillus grows on plants and plant products. Most aflatoxins occur in commodities imported from developing countries and include groundnuts (peanuts), other edible nuts and their products, dried figs, spices and maize. They are also found in cotton seed, rice, barley and rye. Mould contamination of stored food is most likely during periods of high temperature and high humidity.

Animal exposure to aflatoxins usually occurs through ingestion of contaminated food. Commercial dog food or very mouldy bread has been the cause of numerous outbreaks of aflatoxicosis in dogs (*Table 1*) and aflatoxins are routinely detected in dry dog food (Martínez-Martínez et al, 2021).

Aflatoxins are not destroyed by boiling and do not confer colour, odour or flavour to affected foodstuffs. However, dogs may be reluctant to eat affected foodstuffs unless they are mixed with meat or gravy (Arnot et al, 2012).

Mechanism of action

Most of the information about aflatoxins is based on aflatoxin B_1, which is the most toxic aflatoxin. The primary toxic effect of aflatoxin exposure is liver damage. Aflatoxins are oxidised by the cytochrome P450-dependent monooxygenase system to form a highly reactive metabolite (aflatoxin B_1-8-9-epoxide), which causes hepatotoxicity by reacting with macromolecules (including DNA and proteins) to cause fatty liver or liver necrosis. Hepatocellular damage results in impaired liver function, bile duct proliferation, bile stasis and liver fibrosis. In addition, formation of DNA-adducts leads to mutation and cancer. Aflatoxins can also have immunosuppressive and nephrotoxic effects.

The coagulopathy seen in aflatoxicosis is caused by hepatic failure, but has also been suggested to occur as a result of the coumarin-like structure of aflatoxins leading to an anticoagulation effect. Disseminated intravascular coagulation occurs as a result of the depletion of clotting factors. Hyperbilirubinaemia is associated with hepatic failure and the inability to conjugate

Table 1. Summary of aflatoxin incidents in dogs

Date	Location	Source	Comment	Reference
1951–1955	USA	Commercial dog food	Reports of 31 dogs in 1951–1952, 12 dogs in 1952-1953, 18 dogs in 1954–1955	Newberne et al, 1955
1974	Rajasthan and Gujarat, India	Maize heavily contaminated with aflatoxins	A large number of dogs developed ascites and icterus and died within 2–3 weeks. The dogs shared the food of people in the home. A total of 397 people were affected and 106 died	Krishnamachari et al, 1975
1974	Alabama, USA	Cooked cornmeal, meat scraps and contaminated dry commercial dog food	Several dogs died and seven dogs presented unwell afterward. Three of these seven were dead on arrival, one died soon after, one on day 3 and another on day 8. One dog survived	Greene et al, 1977
1975	Queensland, Australia	Mixture of very mouldy bread loaves	Three dogs died after acute and subacute exposure (*Box 2*)	Ketterer et al, 1975
1985	Georgia, USA	Mixture of commercial dog food and restaurant waste	Of 48 hunting dogs in a kennel, 13 died and there were several cases with weakness, icterus, and partial anorexia	Liggett et al, 1986
1985	Georgia, USA	Ration of contaminated cornmeal, soybean meal, fat, and a mineral supplement	Nine deaths out of 20 hunting dogs in a kennel	Liggett et al, 1986
1987	Pretoria, South Africa	A brand of contaminated commercial dog food	Ten dogs died, with one acute, seven subacute and two chronic cases	Bastianello et al, 1987
1998	Texas, USA	17 different formulations of commercial dog food made with two rail cars of non-uniformly contaminated corn in a milling plant in Texas in late summer	55 confirmed deaths in dogs	Garland and Reagor, 2001
2005–2006	USA and Israel	Inadequate screening for aflatoxins in 12 corn shipments used in dog food.	46 of 72 dogs in US sample died (*Box 1*) 34 of 50 hospitalised dogs in Israel died	Stenske et al, 2006; Newman et al, 2007; Dereszynski et al, 2008; Bruchim et al, 2012
2006	Venezuela	Dog food	Several hundred dogs died over 6 months	Sogbe et al, 2006
2011	Southern Brazil	Corn meal	65 dogs on nine farms were affected; 60 dogs died	Wouters et al, 2013
2011	Gauteng Province, South Africa	Dry dog food containing 5% groundnut (peanuts)	Approximately 100 dogs presented to a veterinary hospital, but more than 220 dogs are known to have died	Arnot et al, 2012
2013	Turkey	Very mouldy bread	Ten dogs affected, three died	Ural et al, 2013
2013	Brazil	Corn and rice	17 of 18 affected dogs died	Guterres et al, 2017
2015	Turkey	Mouldy bread	Four dogs on a farm died; only one was presented and died 2 hours after admission	Eroksuz et al, 2015
2020–2021	USA	Dog food	Over 110 dogs died	Food and drug adminstration , 2021

Box 1. Case report 1: review of 72 dogs in an outbreak

In a review of 72 cases of aflatoxicosis in dogs from a contaminated commercial pet food, the severity of clinical signs varied widely and seven dogs died abruptly. In order of onset, clinical features included anorexia, lethargy, vomiting, jaundice, diarrhoea (melaena, haematochezia), abdominal effusion, peripheral oedema, terminal encephalopathy and haemorrhagic diathesis. Common clinicopathologic features included coagulopathic and electrolyte disturbances, hypoproteinaemia, increased serum liver enzyme activities, hyperbilirubinaemia, and hypocholesterolaemia. Cytologic hepatocellular lipid vacuolation was confirmed in 11 dogs examined. The best early markers of aflatoxicosis were low plasma activities of anticoagulant proteins (protein C, antithrombin) and hypocholesterolaemia. Aggressive supportive therapy (liver protectants, blood components, antioxidants and vitamin K_1) was ineffective in severely affected dogs. Overall, 46 dogs died (64%) and 26 (36%) survived (Dereszynski et al, 2008).

Box 2. Case report 2: outbreak in 3 dogs on a farm

Three dogs on a cattle farm in Australia became unwell over a period of 1 month. The dogs were fed a mixture of bread and canned food. All dogs became unwell with depression and anorexia. The most severely affected dog vomited bread and meat on the first day of illness, drank large volumes of water on the second day and died on the third. The other two dogs recovered and were eating well on the fourth day. Then two weeks later one of the dogs had severe depression, weakness and anorexia and died after 3 days. The last dog became unwell after another 2 weeks. It developed severe depression, weakness and anorexia and died after 2 days. Post-mortem findings on the last dog revealed intense jaundice, ascites, blood in the gastrointestinal tract and a yellowish-orange liver. No mould was found in the canned food but the bread the dogs had eaten was covered in a thick green growth of mould and contained 6.7 ppm of aflatoxin B_1 but was negative for G_1. A sample of vomit had a concentration of 100 parts per million (ppm) of B_1 and 40 ppm of G_1. The liver of the last dog contained 0.002 ppm B_1 (Ketterer et al, 1975).

bilirubin generated in excessive amounts in the spleen. A decrease in proteins such as albumin is a result of impaired liver function, in addition to protein loss through haemorrhage, ascites and oedema. Cholesterol concentrations fall as a result of cholestasis caused by fibrosis of bile ducts.

Susceptibility

Although all mammals, fish and birds are susceptible to aflatoxicosis, susceptibility varies with species (as there are differences in metabolism), age and nutritional status. However, dogs appear to be particularly sensitive to aflatoxins. The oral lethal dose of 50% (LD_{50}) aflatoxin B_1 in dogs is approximately 0.5–1 mg/kg (Newberne et al, 1966; Wogan, 1966; Newberne et al, 1969).

Clinical effects

Onset

The onset of clinical signs after ingestion of aflatoxins is highly variable, depending on the concentrations and the aflatoxin involved. In dogs, effects can be seen after one to several days following exposure. In more chronic, low-dose poisoning, the effects are the same but milder and are initially characterised by non-specific signs, reduced growth rate and liver damage. Clinical signs can occur over weeks or months.

Duration

In acute cases death can occur within 2–7 days of the onset of signs (usually within 3 days), but it can be several weeks (Wouters et al, 2013).

Clinical signs

Common presenting signs of aflatoxicosis in dogs are inappetence, vomiting, lethargy, melaena and jaundice (Stenske et al, 2006). Occasionally dogs die suddenly without exhibiting apparent clinical signs (Bastianello et al, 1987; Dereszynski et al, 2008) (*Box 1*). In high-dose, acute aflatoxicosis, death can occur from fulminant liver failure without marked increase in liver enzymes.

Severe gastrointestinal signs (anorexia, vomiting and diarrhoea), polydipsia and polyuria, depression, lethargy, weakness, dark yellow or orange urine and jaundice are typical of aflatoxicosis. Dehydration may occur (Chaffee et al, 1965) and evidence of bleeding with bruising, haematemesis, melaena and bloody stools are common. Epistaxis, haematuria and petechiae on mucous membranes may also be seen (Arnot et al, 2012).

Dogs with chronic low-dose exposure may present with severe ascites, pale and jaundiced mucous membranes, severe depression and weight loss (Arnot et al, 2012).

In severe cases there is liver failure, ascites, peripheral oedema, hypotension and encephalopathy. Hepatic encephalopathy may result in neurological effects with behavioural change, vocalisation, lethargy, stupor, tremor, weakness, seizures and coma having been documented (Bruchim et al, 2012). Secondary renal impairment can occur as a result of excretion of bile pigment, hypotension or haemorrhage. Immunosuppression and secondary infection may also occur. Death is usually caused by haemorrhage and sudden death may occur.

Laboratory analysis in dogs with aflatoxicosis will show liver damage with raised liver enzymes (aspartate transaminase and alanine transaminase) and bilirubin, coagulopathy (increased prothrombin time and activated partial thromboplastin time), thrombocytopenia, mild-to-moderate anaemia, hypoalbuminaemia and hypocholesteraemia (Greene et al, 1977; Stenske et al, 2006). Disseminated intravascular coagulation (Greene et al, 1977; Hagiwara et al, 1990; Bruchim et al, 2012), increased fibrinolysis (measured by thromboelastography) (Connor and Goddard, 2012) and reduced fibrinogen (Greene

Table 2. Antioxidant drug dosages

Drug	Dosage	Mechanism
Acetylcysteine	A loading dose of 140 mg/kg intravenously, followed by 70 mg/kg every 4–6 hours for 1–3 days. Given as a 5% solution diluted in sterile water or saline over 20 minutes.	Liver protectant, glutathione precursor
S-adenosylmethionine	20 mg/kg orally per day	Generates sulphur-containing compounds important for conjugation reactions and as a precursor to glutathione. Also, a methyl donor for protein synthesis
Vitamin E	10 IU/kg orally per day	Antioxidant, major terminator of lipid peroxyl reaction
Vitamin K_1	2–5 mg/kg orally or subcutaneously per day	Will not resolve coagulopathy but can cause improvement
L-carnitine	25–100 mg/kg orally per day	Essential nutrient that can enhance fatty acid mobilisation from the liver

et al, 1977; Hagiwara et al, 1990) have been reported and hyperammonaemia may occur (Bruchim et al, 2012). Leucocytosis (caused by neutrophilia) may also occur.

Liver biopsy will typically reveal hepatocellular fatty vacuolation, hepatic necrosis, periportal necrosis, portal fibrosis, perivenular necrosis and inflammation and bile duct proliferation. Bile canaliculi may be plugged with casts (Furrow, 2007).

In post-mortem examination, dogs with acute aflatoxin poisoning have massive fatty degeneration, centrilobular necrosis of the liver and widespread haemorrhage. Typically, severe icterus of mucous membranes and sclera, submucosal oedema, severe haemorrhage of the intestines, spleen and kidneys with centrilobular fatty liver changes and necrosis, and hyperplasia of the bile duct are also seen. The liver and kidneys are usually swollen and discoloured. The gut may be filled with haemorrhagic fluid (Chaffee et al, 1965; Newberne et al, 1966; Greene et al, 1977; Liggett et al, 1986; Bastianello et al, 1987; Newman et al, 2007; Ural et al, 2013). Pulmonary oedema may also be seen (Wouters et al, 2013).

Histopathological changes include cytoplasmic vacuolar degeneration brought on by the accumulation of hepatocellular lipids, portal fibrosis and hepatocellular cholestasis (Stenske et al, 2006). There may be multifocal haemorrhages of the kidneys with degeneration and necrosis of the proximal and distal renal tubular epithelium (Ural et al, 2013). Alveolar haemorrhage may also be seen (Ural et al, 2013).

Prognosis

Although prognosis depends on the extent and severity of the liver damage, mortality rates in dogs with aflatoxicosis are high (*Box 1*). Animals that present as clinically well but with laboratory evidence of hepatic and haematological abnormalities appear to have a more favourable outcome with aggressive supportive care, than those presenting with clinical signs (Bruchim et al, 2012). Prognosis is poor in animals with severe liver damage, even with aggressive treatment (Dereszynski et al, 2008), severe enteric haemorrhage (Dereszynski et al, 2008) or neurological abnormalities (Bruchim et al, 2012).

Diagnosis

Diagnosis of aflatoxicosis is based on history of possible exposure and laboratory confirmation of aflatoxin in suspect material or, less commonly, in tissue samples, particularly the liver. Laboratory changes include increased alanine transaminase, hyperbilirubinaemia, hypocholesteraemia and prolonged clotting times. Note that the magnitude of liver enzyme activity may not reflect the severity of hepatocellular injury seen at post-mortem examination (Dereszynski et al, 2008). Typical liver histological changes in aflatoxicosis are centrilobular necrosis of the liver and bile duct proliferation.

Treatment

There is no specific treatment for aflatoxicosis and treatment is supportive, focusing on management of liver failure. Recovery in survivors may be prolonged.

Gut decontamination is generally not appropriate in aflatoxin poisoning since exposure is usually chronic. The liver enzymes and bilirubin, clotting parameters, electrolytes, cholesterol, blood glucose and a full blood count should be monitored. All animals in the household that have eaten the same food, even if asymptomatic, should be evaluated for liver dysfunction.

Fluid support is essential in aflatoxicosis to correct hypovolaemia and hypoperfusion and facilitate renal elimination of aflatoxin metabolites. Antiemetics are recommended to control vomiting, such as a constant rate infusion of metoclopramide or, if refractory, ondansetron. Nutrition is also important in dogs with liver damage and the diet should be balanced. Protein-restricted diets should be avoided unless the animal has hepatic encephalopathy (Furrow, 2007). Water-soluble vitamins should be provided to prevent deficiency (Cornell University, 2006).

Bleeding is a serious complication in aflatoxicosis. Fresh frozen plasma or a blood transfusion (if there is moderate to severe anaemia) should be used where necessary. High doses may be

needed to replace coagulation factors and albumin (Bruchim et al, 2012), but may be ineffective in dogs with severe, irreversible liver failure (Bruchim et al, 2012). Vitamin K_1 should also be given in animals with clotting abnormalities. This will not reverse coagulopathy but may result in some improvement (Cornell University, 2006).

Liver protectants and antioxidants are recommended, including acetylcysteine, vitamin E, S-adenosylmethionine, milk thistle (silymarin) and L-carnitine (Cornell University, 2006; Furrow, 2007), although the efficacy of these drugs in canine aflatoxicosis has not been evaluated (*Table 2)*. Antibiotic cover is suggested because animals are at risk of sepsis from the compromised gastrointestinal tract. Antibiotics against Gram-negative bacteria are suggested, alone or in combination, such as metronidazole, ampicillin, amoxicillin (Furrow, 2007). A reduced dose (50%) of metronidazole should be used because it is metabolised in the liver. Gastric protectants may also be given, such as omeprazole, and sucralfate is recommended in dogs with haematemesis (Cornell University, 2006).

Dogs that survive aflatoxicosis may develop chronic liver disease or possibly neoplastic hepatic disorders. Hepatotocarcinogenicity is well recognised in humans surviving aflatoxicosis, but not well studied in dogs (Cornell University, 2006). Consequently, periodic health assessment is recommended in survivors (monitoring liver enzymes, bilirubin and biochemistry) (Cornell University, 2006).

In a suspected incident

If there is a suspicion of aflatoxicosis, the suspected product should not be fed. Owners should be advised to consult a vet if their pet develops inappetence, yellow discolouration of eyes, gums or skin, persistent vomiting, bloody diarrhoea, discoloured urine or fever. It is important to document the medical history of suspected cases and where possible, histological examination of biopsy or post-mortem liver samples should be undertaken. Samples of whole blood (in ethylenediaminetetraacetic acid tubes), serum, urine, fresh or frozen tissues, gastrointestinal contents and food should be collected. Samples of suspected food, including original packaging that has batch numbers and use by dates, should be obtained as this can help manufacturers determine the source and affected batches. Opened food should be stored frozen before testing and unopened containers can be stored at room temperature (Stenske et al, 2006). The relevant organisation for investigating these incidents should be contacted for further advice and investigation of possible outbreaks. In the UK this is the Food Safety Agency or trading standards and in the US it is the Food and Drug Administration.

Conclusions

Aflatoxicosis in dogs is not common but can be devastating, with large numbers of animals often being involved and high rates of mortality. Aflatoxins are produced by some strains of the mould *Aspergillus* and dog food is a common source. Aflatoxicosis causes liver failure and typical liver histological changes are centrilobular necrosis of the liver and bile duct proliferation. There is no specific treatment for aflatoxicosis and management is supportive. The prognosis is poor in dogs with severe liver damage.

KEY POINTS

- Aflatoxins are toxic compounds produced by some strains of the mould *Aspergillus.*
- Dogs are particularly susceptible to aflatoxins and the target organ is the liver.
- Dog food is a common source of aflatoxin exposure in outbreaks of aflatoxicosis in dogs.
- Management of aflatoxicosis is supportive focused on treatment of liver failure.
- Mortality in dogs with aflatoxicosis is high.

References

Arnot LF, Duncan NM, Coetzer H, Botha CJ. An outbreak of canine aflatoxicosis in Gauteng Province. J S Afr Vet Assoc. 2012;83(1):2. https://doi.org/10.4102/jsava.v83i1.2

Austwick PKC. Aflatoxicosis in poultry. In: Willey TD, Morehouse LG (eds). Mycotoxic fungi, mycotoxins and mycotoxicosis. an encyclopedic handbook. vol. 2. New York: Marcel Dekker; 1978:279–301

Bastianello SS, Nesbit JW, Williams MC, Lange AL. Pathological findings in a natural outbreak of aflatoxicosis in dogs. Onderstepoort J Vet Res. 1987;54(4):635–640

Bruchim Y, Segev G, Sela U et al. Accidental fatal aflatoxicosis due to contaminated commercial diet in 50 dogs. Res Vet Sci. 2012;93(1):279–287. https://doi.org/10.1016/j.rvsc.2011.07.024

Chaffee VW, Edds GT, Himes JA, Neal FC. Aflatoxicosis in dogs. Am J Vet Res. 1969;30(10):1737–1749

Connor B, Goddard A. Aflatoxicosis causing a hyperfibrinolytic syndrome in 12 dogs [abstract]. Vet Emerg Crit Care. 2012;22(S2):54

Cornell University. Canine foodborne aflatoxicosis synopsis. 2006. http://www.allnaturalpetcare.com/Holistic_Pet_Remedies/Canine_Foodborne_Aflatoxicosis.pdf (Accessed 2 September 2021)

Denning DW. Aflatoxin and human disease. Adverse Drug React Acute Poisoning Rev. 1987;6(4):175–209

Dereszynski DM, Center SA, Randolph JF et al. Clinical and clinicopathologic features of dogs that consumed foodborne hepatotoxic aflatoxins: 72 cases (2005-2006). J Am Vet Med Assoc. 2008;232(9):1329–1337. https://doi.org/10.2460/javma.232.9.1329

Eroksuz Y, Kaya E, Baydar E et al. Subacute aflatoxicosis due to moldy bread consumption in a dog. Revue Med Vet. 2015;166(9–10):259–265

Food and Drug Administration. Certain lots of Sportmix Pet Food Recalled for potentially fatal levels of aflatoxin. 2021. https://www.fda.gov/animal-veterinary/outbreaks-and-advisories/fda-alert-certain-lots-sportmix-pet-food-recalled-potentially-fatal-levels-aflatoxin (accessed 4 August 2021)

Furrow E. The critical care of aflatoxin-induced liver failure in dogs. Vet Med. 2007;102(10):644–651

Garland T, Reagor J. Chronic canine aflatoxicosis and management of an epidemic. In: de Koe W, Samson R, van Egmond H (eds). Mycotoxins and phycotoxins in perspective at the turn of the millennium. Wageningne, The Netherlands: Ponsen and Looven; 2001:231–236

Greene CE, Barsanti JA, Jones BD. Disseminated intravascular coagulation complicating aflatoxicosis in dogs. Cornell Vet. 1977;67(1):29–49

Guterres K, Silva C, Giordani C et al. [Outbreak of acute aflatoxicosis in dogs in the municipality of Pelotas/RS, Brazil.] Surto de aflatoxicose aguda em cães no município de Pelotas/RS. Pesq Vet Bras. 2017;37(11):1281–1286. https://doi.org/10.1590/s0100-736x2017001100014

Hagiwara M, Kogika H, Malucelli B. Disseminated intravascular coagulation in dogs with aflatoxicosis. J Small Anim Pract. 1990;31(5):239–243. https://doi.org/10.1111/j.1748-5827.1990.tb00793.x

Ketterer PJ, Williams ES, Blaney BJ, Connole MD. Canine aflatoxicosis. Australian Vet J. 1975;51(7):355–357. https://doi.org/10.1111/j.1751-0813.1975.tb15946.x

Krishnamachari KA, Bhat RV, Nagarajan V, Tilak TB. Hepatitis due to aflatoxicosis. An outbreak in Western India. Lancet. 1975;305 (7915):1061–1063. https://doi.org/10.1016/S0140-6736(75)91829-2

Liggett AD, Colvin BM, Beaver RW, Wilson DM. Canine aflatoxicosis: a continuing problem. Vet Hum Toxicol. 1986;28(5):428–430

Martínez-Martínez L, Valdivia-Flores AG, Guerrero-Barrera AL et al. Toxic effect of aflatoxins in dogs fed contaminated commercial dry feed: a review. Toxins (Basel). 2021;13(1):65. https://doi.org/10.3390/toxins13010065

Newberne JW, Bailey WS, Seibold HR. Notes on a recent outbreak and experimental reproduction of hepatitis x in dogs. J Am Vet Med Assoc. 1955;127(940):59–62

Newberne PM, Russo R, Wogan GN. Acute toxicity of aflatoxin B1 in the dog. Pathol Vet. 1966;3(4):331–340. https://doi.org/10.1177/030098586600300403

Newberne PM, Butler WH. Acute and chronic effects of aflatoxin on the liver of domestic and laboratory animals: a review. Cancer Res. 1969;29(1):236–250
Newman SJ, Smith JR, Stenske KA et al. Aflatoxicosis in nine dogs after exposure to contaminated commercial dog food. J Vet Diagn Invest. 2007;19(2):168–175. https://doi.org/10.1177/104063870701900205
Sogbe E, Ascanio E, Zerpa H et al. An outbreak of mycotoxins poisoning in dogs in Venezuela. J Vet Pharmacol Therap. 2006;29(Suppl 1):334Stenske KA, Smith JR, Newman SJ, Newman LB, Kirk CA. Aflatoxicosis in dogs and dealing with suspected contaminated commercial foods. J Am Vet Med Assoc. 2006;228(11):1686–1691. https://doi.org/10.2460/javma.228.11.1686
World Health Organization. Mycotoxins. Environmental health criteria 11. Geneva: World Health Organization; 1979
Ural K, Ulutas B, Tunca R et al. Aflatoxicosis in rottweilers after eating moldy bread: clinicopathologial features and effective tetrasulphate therapy. Vet Archiv. 2013;83(4):403–412
Wogan GN. Chemical nature and biological effects of aflatoxins. Bacteriol Rev. 1966;30(2):460–470. https://doi.org/10.1128/br.30.2.460-470.1966
Wouters AT, Casagrande RA, Wouters F et al. An outbreak of aflatoxin poisoning in dogs associated with aflatoxin B1-contaminated maize products. J Vet Diagn Invest. 2013;25(2):282–287. https://doi.org/10.1177/1040638713477409