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Aflatoxins in Pet Foods: A Risk to Special Consumers

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1. Introduction

Mycotoxin contamination in pet food poses a serious health threat to pets. Cereal grains and nuts are used as ingredients in commercial pet food for companion animals such as cats, dogs, birds, fish and rodents. Cereal by-products may be diverted to animal feed even though they can contain mycotoxins at concentrations greater than raw cereals due to processing (Moss, 1996; Brera et al., 2006). Several mycotoxin outbreaks in commercial pet food have been reported in the past few years (Garland and Reagor, 2001; Stenske et al., 2006).

Most outbreaks of pet mycotoxicosis, however, remain unpublished and may involve the death of hundreds of animals (MSNBC News Services, 2006). The term "companion animal" implies the existence of a strong human–animal bond between pets and their owners (Adams et al., 2004). A pet is often regarded as a family member by its owner and a person may develop strong relationships with animals throughout his or her lifetime. Pet interactions and ownership have been associated with both emotional and physical health benefits (Milani, 1996; Adams et al., 2004). The human–animal bond has resulted in over sixty four million American households in owning one or more pets in 2006, thereby creating a huge market for the pet food industry (APPMA, 2006). Dogs and cats continue to be the most popular pet to own, found in at least one out of three US households. The breakdown of pet ownership in the USA according to the 2009-2010 National Pet Owners Survey is above of a hundred millions of dogs and cats (Table 1).

Bird	15.0
Cat	93.6
Dog	77.5
Freshwater Fish	171.7
Saltwater Fish	11.2
Reptile	13.6
Small Animal	15.9

APPA's 2009/2010

Table 1. Total Number of Pets Owned in the U.S. (millions)

The health problems of pets, are therefore more of an emotional concern as compared to a mainly financial concern in farm animals (Dunn et al., 2005; Milani, 1996). In 2009, forty five billion was spent on pets in the U.S. Seventeen billion went to Pet food (Table 2).

Food	$17.56 billion
Supplies/OTC Medicine	$10.41 billion
Vet Care	$12.04 billion
Live animal purchases	$2.16 billion
Pet Services: grooming & boarding	$3.36 billion

APPA's 2009/2010

Table 2. Expenses on Pet care in 2009 in U.S. (billion)

1.1 Aflatoxins

Mycotoxins are secondary fungal metabolites that exert toxic effects on animals and human beings. Secondary fungal metabolites are not necessary for the growth or reproduction of the fungus. Not all fungi are capable of producing mycotoxins; those that can are referred to as toxigenic. The major aflatoxins (AFs) consist of aflatoxins B_1, B_2, G_1 and G_2 produced by certain toxigenic strains of *Aspergillus flavus, Aspergillus parasiticus,* and *Aspergillus nominus* (Richard, 2007; Puschner, 2002). Aflatoxin M_1 (AFM_1), a hydroxylated metabolite, found primarily in animal tissues and fluids (milk and urine) is a metabolic product of aflatoxin B_1 (AFB_1) and this mycotoxin is not found in feed grains. Aflatoxins can be present in milk of dairy cows, meat of swine or chicken eggs if the animals consume sufficient amounts in their feed (Robens and Richard, 1992).

Many toxigenic fungi produce mycotoxins only under specific environmental conditions. Grains stored under high moisture/humidity (>14%) at warm temperatures (>20°C) and/or inadequately dried can potentially become contaminated. Warm (air temperature of 24°C–35 °C) and humid (moisture content of substrate between 25% and 35%) conditions lead to extensive mold growth and aflatoxin production (Ominski et al., 1994 Puschner, 2002). The amount of water activity (a_w) is a measure of the amount of water available for bacterial and fungal growth. The pure water value is 1.0 and the decrease of a_w confers a protective result against toxigenic molds, since the minimum a_w permitting fungal germination and growth ranged from 0.80 to 0.82, according to Pitt and Miscamble (1995). Hunter (1969) proposed the value of 0.87 as the minimum required for aflatoxin production. Grains must be kept dry, free of damage and free of insects. Initial growth of fungi in grains can form sufficient moisture from metabolism to allow for further growth and mycotoxin formation. These conditions allow mold "hot spots" to occur in the stored grain. Traditionally, mycotoxin-producing fungi have been divided into two groups: ''field'' (plant pathogenic) and ''storage'' (saprophytic) fungi. Even though production can occur after harvest under inadequate storage conditions, large-scale contamination typically occurs in the field (Puschner, 2002).

Toxic secondary fungal metabolites may pose a significant risk to human and animal health if cereal grains and animal feed become colonized by toxigenic fungi. Aflatoxins have been found in many agricultural commodities but most commonly in corn, cottonseed, ground nuts, and tree nuts. The occurrence of a toxigenic fungus on a suitable substrate does not necessarily mean that a mycotoxin is also present (CAST, 1989).

Mycotoxicoses are reported in small animals. However, it is evident that there is little in the scientific literature on mycotoxicoses in pets (Puschner, 2002). An attempt is made to compile additional information on mycotoxins that have caused disease in small animals after experimental exposure. Aflatoxins, tricothecenes, tremorgens, and other mycotoxins are discussed in view of particular hazards and concerns for small animals, but undoubtedly, aflatoxins are the most documented of all mycotoxins (CAST, 1989).

Since the 1950s, there have been many reports and studies on aflatoxin metabolism, toxicity, residues, and species susceptibilities in domestic animals (mainly swine, cattle, and poultry). Research on the toxicity of aflatoxins in dogs began in the 1960s (Armbrecht et al., 1971; Chaffee et al., 1969; Newberne et al., 1966). Compared with other species, the effects of aflatoxins in dogs are less well documented, yet there are reports of aflatoxicosis in dogs after eating moldy food (Bailly et al., 1997, Ketterer et al., 1975) or contaminated grain (Bastianello et al., 1987).

1.2 Contaminated ingredients

Aflatoxins have elicited great public health concern because of their widespread occurrence in several dietary staples such as peanuts, tree nuts, corn, dried fruits, silage, and forages, all of which are used as animal feed ingredients. Monitoring these substrates for mycotoxins, especially AFB_1, is crucial to prevent outbreaks of acute mycotoxicosis and to diminish exposure risk of animals and humans to these harmful toxins (CAST, 2003, Pereyra et al., 2008).

Sharma and Márquez reported that the samples of cat and dog foods which had high amount of AFB_1, AFM_1 and AFP_1 and the ingredients were cheese, dry milk powder, oil seed meal, soya, cereals and rice, but maize was the main ingredient in all contaminated samples. Siame et al. (1998) reported that aflatoxin was the most common toxin detected in foods and feeds samples containing sorghum and maize. Scudamore et al. (1997) has presented that aflatoxin B_1 was the mycotoxin found most frequently in rice bran, maize products, palm kernels and cottonseeds. Aflatoxins are also commonly found in peanuts, raw milk and tree nuts (Haschek et al., 2002).

A total of 35 samples of pet food of 12 different trademarks, out of which, 19 samples were of dog and 16 were of cat foods with different ingredients and flavours were analysed by Sharma and Márquez (2001) in Mexico and amounts of aflatoxins (B_1, B_2, G_1, G_2, M_1, M_2, P_1 and aflatoxicol) were determinated in these samples and the presence of aflatoxins and aflatoxicol were observed in most of the samples. Aflatoxin B_1 was the mycotoxin found with higher frequency (0.885) and its level was higher in 17% of samples (of both dog and cat foods).

The authors described also that the highest level of AFB_1 were found in cat food of three different trademarks with concentration of 46.1, 30.8 and 22.2 ng/g. In case of dog food, two samples contained 39.7 ng/g and 27.0 ng/g of AFB_1. A higher incidence of AFM_1 was observed in three samples (21.37, 19.37 and 10.8 ng/g). AP_1 was found in one sample (12.52 ng/g) of dog food and other aflatoxins were found in traces. Two samples (one each cat food and dog food) contained a high concentration of total aflatoxins (72,4 and 59.7 ng/g).

According to Joint FAO/WHO Expert Committee on Food Additives (JEFCA) the tolerance limit is 20 ng/g to AFB_1, 50 ng/g of total aflatoxins in food and 0.5 ng/g of AFM_1 in milky (Bhat, 1999).

1.3 Pet food: How is it made?

Commercially prepared pet foods are an easy and economical way to fulfill the nutrient requirements in pets. These types of foods provide more than 90% of the calories consumed by pets in North America, Japan, Northern Europe, Australia, and New Zealand. Dogs, cats, hamsters, rabbits, birds, chinchillas and fishes are the main focus to pet food industry. There are three basic forms of commercial pet foods: dry, semi-moist, and moist or canned. The main difference in this categorization scheme is based on the water content of the food with

dry foods containing usually less than 11% water, semi-moist foods containing 25 to 35% water, and moist or canned food containing 60 to 87% water (Zicker, 2008).
Most manufactured pet foods are formulated to meet specific nutrient goals to support growth, maintenance, or gestation/ lactation as recommended by the Association of American Feed Control Officials (AAFCO, 2007). The nutrients that are targeted include the calories, protein, fat, carbohydrate, vitamins, and minerals required to sustain life and, where possible, optimize performance (Zicker, 2008). Sorghum, maize, soya, rice, cereals, meal of meat and bones, by products of birds, fish, chicken, derived product of egg and milk were the main ingredients of pet food (Sharma and Márquez, 2001).

1.3.1 Dry food

Dry food is by far the major segment of the pet food industry attributable to its convenience to store and feed. Dry food particles are usually formed through a process called extrusion, which utilizes the same technology as that to produce breakfast cereals for people. Other methods include baking, flaking, pelleting, and crumbling of foods to achieve a dry form. Dry foods are protected against spoilage due to their low water content. To produce extruded foods, ingredients determined by the formulation are compounded and mixed homogeneously and then passed through an extruder. The extruder uses a combination of steam, pressure, and temperature to rapidly cook foods, then pushes the mixture through a faceplate where a revolving knife slices the extruded mix into the final kibble product. The extrusion process puts the ingredients through a temperature between 100 to 200°C and 34 to 37 atm pressure, which is high enough to effectively achieve a food sterilization process that meets industry standards The resultant extruded material has a moisture of approximately 25% before drying, where the final moisture content of 8 to 10% is attained. At this level of moisture mold formation is inhibited (Zicker, 2008; Crane et al., 2000; Miller and Cullor, 2000).

1.3.2 Canned food

Moist or canned foods historically comprised a much greater segment of the manufactured pet foods market but they have decreased in use. Moist foods are high in water content, usually 60 to 87%, and require the presence of gelling agents such as starch or gums to achieve their final consistency. Moist foods go through a process that results in a well sterilized final product similar to canned products for human consumption. Ingredients are mixed, ground together, and then cooked into a hot mixture for transfer to the can. The slurry is allotted into the cans and the top is sealed under steam, which displaces any air, resulting in an anaerobic environment. Finally the cans are sterilized in a machine called a retort where temperatures of 121°C are maintained for a minimum of 3 minutes (Zicker, 2008).

1.3.3 Semi-moist food

Semi-moist foods are a smaller but significant portion of the manufactured pet food market. Semi-moist foods require the use of humectants and acidification to control water content and inhibit mold growth. Semi-moist foods also have a low fiber content and relatively high sugar content, which make them highly palatable but also not an ideal choice to deliver weight control applications based on fiber. Semi-moist foods are manufactured in a way similar to extruded food but the water content is maintained at a higher level because of the

added humectants. The final moisture content of 25 to 35% is more prone to mold and spoilage, which is mitigated by mold or bacterial inhibitors as well as managing the a_w component of the food. The addition of humectants helps to keep this at a low level of a_w, which effectively inhibits their growth despite higher total water content. It is apparent that much effort is put toward producing products that not only meet nutrient targets but that are also safe for their intended purposes. In addition to the care paid to details during the formulation and manufacturing process, companies maintain a quality control programs that further ensure safety and adequacy or products (Zicker, 2008).

Thermal inactivation is a good alternative for products that are usually heat processed. The processes described use high temperature (100 to 200 ^oC) that is an important physical factor to fungal control, because when heated to high temperatures, bacteria and fungi can be killed. However, the temperatures applied in the pet food processes are not enough to control the pre-formed aflatoxins in the ingredients. Mycotoxins are, in general, chemically and thermally stable, rendering them unsusceptible to commonly used feed manufacturing techniques (Kabak et al., 2006; Leung et al., 2006).

By-products commonly used in animal feeds (e.g. dried distillers grains and solubles) may also contain concentrated (i.e. higher) levels of mycotoxins relative to the grains (corn) they are derived from (Schaafsma et al., 2009). Aflatoxins are stable to moderately stable in most food processes. Aflatoxins are stable up to their melting point of around 250 ^oC and are not destroyed completely by boiling water, autoclaving, or a variety of food and feed processing procedures (Feuell 1966; Van Der Zijden et al., 1962).

1.4 Outbreaks of mycotoxicoses in pets

The effects of mycotoxins on companion animals are severe and can lead to death. As early as 1952, a case of hepatitis in dogs was directly linked to consumption of moldy food (Devegowda and Castaldo, 2000). A careful survey of the early outbreaks showed that they were associated with Brazilian peanut meal and the mycotoxin contaminated feed was groundnut cake. This outbreak occurred in the 1960 when more than 100,000 young turkeys, ducklings and young pheasants on poultry farms in England died in the course of a few months from an apparently new disease that was termed "Turkey X disease", because the cause of the disease was unknown. An intensive investigation of the suspect peanut meal was undertaken and it was quickly found that this peanut meal was highly toxic to poultry and ducklings with symptoms typical of Turkey X disease. Speculations made during 1960 regarding the nature of the toxin suggested that it might be of fungal origin. In fact, the toxin-producing fungus was identified as *Aspergillus flavus* and the toxin was given the name Aflatoxin. This discovery has led to a growing awareness of the potential hazards of these substances as contaminants of food and feed causing illness and even death in humans and other mammals (Bradburn and Blunden, 1994; Asao et al., 1963). Following the discovery of AF the agent responsible for the 1952 case was identified as AFB_1 (Newberne et al., 1966) and the symptoms of aflatoxicoses in dogs were also elucidated (Newberne et al., 1966; Ketterer et al., 1975).

Mycotoxins were detected in food for dogs, cats, birds, rodents and fishes with different prevalences across regions. Wild bird seed, for instance, has been found to be most contaminated among different pet food products (Henke et al., 2001). In the case study realized by Ketterer et al. (1975), three dogs on a farm in Queensland became ill (severe depression, anorexia, and weakness) and died at different times within a month following consumption of a commercial dog food mixed with AF-contaminated bread.

In 1998, 55 dogs died in Texas after eating dog food containing levels of aflatoxin that varied between 150 and 300 ppb (parts per billion). The corn in the diets was contaminated with aflatoxin (Bingham et al., 2004). Aflatoxins have been the most common cause of acute mycotoxin outbreaks in commercial dog food because corn is the usual source of aflatoxins in these cases. A commercial dog food with a high aflatoxin level was responsible for the acute deaths of 23 dogs in the United States in 2005 (Lightfoot and Yeager, 2008).

Pereyra et al. (2008) described an acute aflatoxicosis case on a chinchilla farm in Argentina. Chinchillas (*Chinchilla lanigera*) are rabbit-sized crepuscular rodents native to the Andes Mountains in South America. Chinchillas are farm raised and are currently used by the fur industry and as pets. Chinchillas are known to be very sensitive to mycotoxins, and a large number of animals often die if acute aflatoxicosis occurs. Clinical signs that may indicate mycotoxicosis on a farm include low feed intake, diarrhea, weight loss, poor condition of the skin, fur discoloration, sudden death, and a predisposition to secondary infections (Pereyra et al., 2008). In this case of chinchilla's farm the feed samples had undergone a pelleting process by an expander at 90°C for 60 min. This oat-based commercial feed was suspected to have caused the death of 200 animals.

The available reports of acute mycotoxicosis, however, cannot provide the whole picture of the mycotoxin problem associated with pet foods since only a small number of food poisoning cases are published. Veterinarians, furthermore, often overlooked mycotoxins as the cause of chronic diseases such as liver and kidney fibrosis, infections resulting from immunosuppression and cancer. These findings suggest that mycotoxin contamination in pet food poses a serious health threat to pet species. The public has recently begun a shift to organic pet foods. The public perception is that organic foods are safer due to the lack of pesticide residues. In the case of mycotoxins, however, the avoidance of insecticides and fungicides may result in increased crop pest damage, fungal growth and mycotoxin production (Boermans and Leung, 2007).

2. Mycotoxin risks assessment

"Risk assessment" is the systematic scientific characterization of potential adverse effects resulting from exposure to hazardous agents (NRC, 1993; Faustman and Omenn, 2001). Risk is the probability that a substance will produce a toxic effect. Risk involves two components: toxicity and exposure. Thus mycotoxins of relatively low toxicity may pose significant risks if exposure is great, frequent, and long. Conversely, mycotoxins of high toxicity, such as aflatoxins, may pose virtually no risk if exposure can be substantially reduced. "Exposure assessment" determines what type, levels, and duration of exposures are expected. Although the exposure of pet animals to mycotoxins in grain-based pet food is generally low, it is unavoidable and occurs throughout the entire life of the animal. The toxicity of a substance is dependent on its chemical, physical and biological properties. Often referred to as "hazard", toxicity is an inherent property of the compound and the animal being exposed (Faustman and Omenn, 2001).

The objectives of classical mammalian toxicity studies developed for risk assessment are as follows:

1. Hazard identification — determine the kinds of adverse effects
2. Dose–response assessment — determine the potency or sensitivity of effects
3. No observable adverse effect level (NOAEL) and lowest observed adverse effect level (LOAEL).

Toxicological information on mycotoxins is important because it allows us to judge the relative risk that may result from exposure to these toxic substances. Today's short-term repeat dose toxicity testing is used to derive symptoms as the main objective with mortality as a secondary objective (Paine and Marrs, 2000).

From this data, LD50 can be calculated as an indicator of acute response but LD50 alone gives little information on chronic response. Subchronic toxicity studies of 90 day exposures are used to determine the chemical dose an animal can consume daily without any demonstrable effect (NOAEL), and to characterize the effects of the chemical when administered at doses above the NOAEL. Chronic toxicity studies measure the effect of doses below the NOAEL on the normal life span of the animal. Chronic studies are often used to determine if the substance causes "delayed" effects on reproduction, development or cancer. One dose in chronic studies should cause subtle signs of toxicity such as reduced weight gain or a minor physiological response (LOAEL) (Boermans and Leung, 2007).

These classical toxicity studies using dosages in both effects and no effect range are designed to derive data to be applied to risk determination. Toxicity testing has made great strides, ever increasing our ability to detect sensitive toxic endpoints. Routine haematology, blood biochemistry, histology, and cytology are being supplemented by sophisticated diagnostic equipment including ultrasound imaging, magnetic resonance imaging (MRI), and electron microscopy. New technologies are extremely sensitive at detecting effects at sub-clinical dosages. Molecular techniques (e.g. DNA Microarray) detect alterations at the molecular level and help elucidate modes of action. Toxic endpoints should, however, have a level of clinical significance. What should be the most sensitive toxicological parameter may soon have to be answered (Boermans and Leung, 2007).

Most mycotoxin research has been designed to investigate toxic effects and therefore dosages used are in the toxic range. In such experiments the lowest experimental dose causing a toxic effect may be far greater than the threshold of the adverse effect and therefore overestimates the true LOAEL. Furthermore, if an experimental dose falls into the no-effect range, it may be far below the threshold dose and therefore greatly underestimates the true NOAEL. These factors introduce variability and uncertainty in the estimation of NOAEL and LOAEL (USEPA, 1995).

As for all risk assessments, pet health risk assessment requires data on toxicity and exposure. Pet species are seldom used for toxicity studies and therefore data obtained from other species are used in the risk assessment for pets. This results in a level of uncertainty when extrapolating toxicity data from experimental animals to pet species (Faustman and Omenn, 2001). The process of human health risk assessment (Covello and Merkhofer, 1993) can be applied to the risk determination in animals. In order to estimate the risk associated with mycotoxin exposure, we need to determine the dose a pet can consume in the food on a daily basis for their entire life with no adverse effect (i.e. NOAEL). This level can then be divided by an appropriate safety factor. Safety Factors (SF) (i.e. uncertainty factors) are numerical values applied to the NOAEL or other effect levels to account for any uncertainty in the data (Boermans and Leung, 2007).

Uncertainty includes species extrapolation, the nature and severity of effect, differences in pet breeds, and variability in LOAEL estimation. SF's could be adjusted according to epidemiological data on pets. Human SF numbers are often selected as a factor of 10, 100, or 1000 and set by the World Health Organization/Food and Agriculture Organization (WHO/FAO). Pet food SF's could be set by the pet food industry to apply standard safety guidelines. This process of combining the qualitative and quantitative aspects of toxicity and

exposure to derive a quantitative level of risk is called "risk characterization" (Faustman and Omenn, 2001).
To complete the process a safe pet dietary level (SPDL) can be determined using a pet specific food factor (FF). The food factor is the amount of food consumed daily and accounts for the differences in the quantity of food consumed by different animal species. A safe pet dietary level (SPDL) would be equivalent to the human Maximal Permissible (tolerance) Level in foods (MPL). Risk management refers to the process by which policy actions are chosen to control hazards identified in the risk assessment/risk characterization processes (Faustman and Omenn, 2001; Covello and Merkhofer, 1993).
Calculation of SPDL would provide producers with a pet specific maximal permissible (tolerance) level of mycotoxin in food. Managers would consider scientific evidence and risk estimates along with processing, engineering, economic, social and political factors in evaluating alternative options (Boermans and Leung, 2007).

3. Mechanisms of toxicity in pets

The aflatoxins are primarily hepatotoxic or cause liver damage in animals; aflatoxin B_1 is the most toxic, followed by aflatoxins G_1, B_2, and G_2. Susceptibility varies with breed, species, age, dose, length of exposure and nutritional status. Aflatoxins may cause decreased production (milk, eggs, weight gains, etc.), are immunosuppressive, carcinogenic, teratogenic and mutagenic (Miller and Wilson, 1994). Aflatoxins are acutely toxic, carcinogenic, teratogenic, mutagenic, and immunosuppressive to most mammalian species. Animal species display differing degrees of susceptibility to aflatoxins, however, and it is now recognized that young animals are more susceptible. The primary clinical effects in aflatoxicosis are related to hepatic damage in all species studied (Boermans and Leung, 2007; Puschner, 2002; Plumlee, 2004).
After ingestion, aflatoxins are absorbed into the circulatory system, from which they are largely sequestered into the liver. Aflatoxins are then metabolized in the liver by microsomal mixed-function oxidases and cytosolic enzymes (Eaton et al., 1994a). The toxicity of aflatoxins is a result of the formation of the reactive aflatoxin B_1 8,9-epoxide, which binds covalently to cellular macromolecules such as DNA, RNA, and protein enzymes resulting in damage to liver cells (Cullen and Newberne, 1994).
Binding to these macromolecules results in adduct formation and is thought to ultimately result in damage to and necrosis of hepatocytes and other metabolically active cells. Typically, hepatocellular damage leads to impaired liver function, bile duct proliferation, bile stasis, and liver fibrosis. Epoxide formation may also occur in other tissues such as renal proximal tubular epithelium. Primary metabolites are further detoxified by conjugation with glutathione, glucuronic acid, amino acids, sulfate, or bile salts, and they are eliminated via feces and urine (Cullen and Newberne, 1994; Eaton et al., 1994b).
In addition to their hepatotoxic properties, aflatoxins are also carcinogenic. The binding of DNA causes genotoxicity and mutation in cells. Aflatoxin B_1 (AFB_1) has become an important model agent in the fields of experimental mutagenesis, carcinogenesis and biochemical and molecular epidemiology (Groopman et al., 1988). AFB_1 is metabolized by the microsomal mixed function oxygenase enzyme system (localized mainly on the endoplasmic reticulum of liver cells, but also present in kidney, lungs, skin, and other organs) to a variety of reduced and oxidized derivatives including an unstable reactive epoxide (Busby and Wogan, 1984). Epoxidation of the double bond of the terminal furan

ring of AFB_1 results in AFBi-8,9-epoxide, which can form adducts with nucleophilic sites in DNA, primarily at the N 7 atom guanine, as well as reacting with RNA and protein (Croy et al., 1978; Neal et al., 1986).

In dogs, the most prominent clinical signs of aflatoxicosis are related to the impairment of liver function. In most reported cases, dogs either died suddenly or after a short clinical course. In addition to hepatitis and sudden death in dogs, symptoms of acute aflatoxicoses in both dogs and cats include vomiting, depression, polydipsea, and polyuria, weakness, anorexia, diarrhea, icterus, epistaxis, and petechiae on mucous membranes. It is thought that hemorrhagic diathesis secondary to protein synthesis inhibition and clotting factor deficiency is the cause of death in affected dogs (Hussein and Brasel, 2001; Puschner, 2002).

Necropsy observations revealed enlarged livers, disseminated intravascular coagulation, and internal hemorrhaging. In subacute aflatoxicosis (at 0.5–1 mg/kg of pet food over 2–3 week), dogs and cats become lethargic, anorexic, and jaundiced (Newberne et al., 1966). This can be followed by disseminated intravascular coagulation and death. The vomitus specimens from one dog contained high levels of AF (100 μg/g of AFB_1 and 40 μg/g of AFG_1). Death usually occurs in 3 days with LD50 levels ranging from 0.5 to 1.0 mg/kg in dogs and 0.3 to 0.6 mg/kg in cats depending on the age of the animal (Newberne et al., 1966).

The oral median lethal dose (LD50) of purified aflatoxin found by Cullen and Newberne (1994) in dogs was 0.80 mg/kg of body weight (BW). The authors reported the LD50 for cats as 0.55 mg/kg of BW. Dogs have developed acute, subacute, and chronic aflatoxicosis from batches of commercial dog food containing 0.1 to 0.3 mg/kg (ppm) of aflatoxin and after eating moldy bread with aflatoxin concentrations of 6.7 and 15 mg/kg (ppm), respectively (Bailly et al., 1997; Bastianello et al., 1987; Ketterer et al., 1975).

In acute aflatoxicosis, dogs exposed to > 0.5–1 mg aflatoxin/kg body weight (BW) typically die within days, showing enlarged livers, disseminated intravascular coagulation and internal hemorrhaging (Bohn and Razzai–Fazeli, 2005). Sub-acute aflatoxicosis (0.5–1 mg aflatoxin/kg pet food) is characterized by anorexia, lethargy, jaundice, intravascular coagulation and death in 2–3 weeks. Similar hepatotoxic effects can also be produced by chronic aflatoxin exposure with 0.05– 0.3 mg aflatoxin/kg pet food over 6–8 weeks. The chronic carcinogenic dose of aflatoxins is much lower than the acute dose. Newberne and Wogan (1968) have experimentally induced malignant tumors in rats with a continual exposure of <1 mg aflatoxin B_1/kg feed. Since aflatoxins are both acute and chronic hepatotoxins and carcinogens, the actual number of dogs affected by aflatoxins would be far more than the total number reported in acute poisoning cases (Boermans and Leung, 2007).

A 2-kg feed sample taken from a chinchilla farm located in the province of Córdoba, in the central region of Argentina, was analyzed. This study evaluated macroscopic and histologic changes in the livers of dead chinchillas. The authors reported that all chinchillas were kept under the same husbandry conditions on the farm. The hatchery had 200 animals that all received the same feed. All of these animals died naturally after the consumption of feed by an acute aflatoxicosis. Analyses of the pelletized feed for AFs by TLC revealed that the feed sample was contaminated at a mean level of 212 ppb ± 8.48 ppb of AFB_1 (Pereyra et al., 2008). Macroscopic inspection of the livers revealed general enlargement, pale-yellowish coloration, hypertrophy, rounded hepatic borders, and increased friability. Livers from chinchillas with aflatoxicosis were 38–71% larger than those from control animals. The color of the livers from chinchillas with acute aflatoxicosis was yellowish gray to pale yellow with gray spots (8 of 9 affected livers). Histopathology revealed severe, diffuse cytoplasmic

vacuolation, with the appearance of many large and fewer small cytoplasmic vacuoles in hepatocytes in HE-stained tissue sections. Frozen sections of liver stained confirmed the presence of lipid within the cytoplasmic vacuoles (Pereyra et al., 2008).

The frequency of chromosomal aberrations in bone marrow cells, after a single i.p. aflatoxin B_1 (AFB_1) dose, was examined in male Chinese hamsters *(Cricetulus griseus)*. There was a significant increase in aberrant cells within 5 days of administration of a dose of 0.1 µg-5 mg AFB_1/kg, and on the 36th day. After a single dose of 5 mg AFB_1/kg the enhanced frequency of aberrant cells was monitored up to day 104 with no sign of a decrease to control level. The results indicate that the minimum mutagenic effect of an AFB_1 dose in this system is 0.1 µg/kg. Attention is drawn to the long-term presence of chromosomal aberrations even after a single i.p. exposure to AFB_1 (Bárta et al., 1990). According to Schmidt and Panciera (1980) aflatoxin caused primarily foetal growth retardation in hamsters and hepatic and renal necrosis occurred in the pregnant females.

Rabbit is considered as one of the most suitable and sensitive animal model for studying the teratogenic potential of a chemical (World Health Organization, 1993). Wangikar et al. (2005) showed that AFB_1 was found to be teratogenic in rabbits when given by oral route during gestation days 6–18 and the dose of 0.1 mg/kg could be considered as the minimum oral teratogenic dose. In this study the mean fetal weights were significantly reduced and the gross anomalies observed included wrist drop and enlarged eye socket whereas, skeletal anomalies were agenesis of caudal vertebrae, incomplete ossification of skull bones and bent metacarpals. The visceral anomalies of microphthalmia and cardiac defects were observed. The characteristic histological findings of fetal tissues were distortion of normal hepatic cord pattern and reduced megakaryocytes in liver, fusion of auriculo-ventricular valves, mild degenerative changes in myocardial fibers, microphthalmic eyes and lenticular degeneration. There was no dead fetus in any group.

Avian species are more susceptible than other affected species, such as dogs, cattle, swine, and humans, to aflatoxicosis (Robens and Richard, 1992). Aflatoxin and fusariotoxin are often responsible for avian mycotoxicosis. Clinical signs of chronic aflalotoxicosis often include lethargy, weight loss, anorexia, regurgitation, and polydipsia (Degernes, 1995; Rauber, 2007). Mycotoxins are hepatotoxic and histologic changes include increased content of hepatic glycogen, portal infiltrate of monocytes, increased lipid droplet accumulation, hepatic necrosis and bile duct hyperplasia (Degernes, 1995; Ergün et al., 2006).

Changes in levels of specific neurotransmitters in the pons and brain stem have also been noted in some species (Yegani et al., 2006). The commercial product to birds are presented as a mixed of grains, that are more susceptible to fungal attack. Hepatic changes have been shown to occur in turkeys at levels as low as 100 to 400 ppb (Schweitzer et al. 2001). In the United States, the acceptable level of total aflatoxins in food for human consumption is less than 20 mg/kg, except for Aflatoxin M_1 in milk, which should be less than 0.5 mg/kg (Lightfoot and Yeager, 2008).

There are no reports about aflatoxicosis in aquarium small fishes. Aflatoxin contamination has been generally detected in fish farmed widely in the tropical and subtropical regions. Shi et al. (2010) studied tilapias that were fed six diets containing different levels of AFB_1 (19, 85, 245, 638, 793 and 1641 µg/kg), which were prepared with AFB_1-contaminated peanut meal. The results indicated that dietary AFB_1 led to aflatoxicosis effects in tilapia in a dose- and duration-dependent manner. No toxic effects of AFB_1 were found during the first 10 weeks, but by 20 weeks, the diet with 245 µg AFB_1/kg or higher doses reduced the growth and induced hepatic disorder, resulting in decreased lipid content, hepatosomatic

index, cytochrome P450 A1 activity, elevated plasma alanine aminotransferase activity and abnormal hepatic morphology in these fishes.
The aflatoxin-treated Indian major carp (1.25 mg/kg body weight) revealed a reduction of total protein, globulin levels, bacterial agglutination titre, NBT and serum bactericidal activities, as well as an enhanced A:G ratio without change in albumin concentration. Thus, AFB_1 proved to be immunosuppressive to fishes even at the lowest dose of toxin treatment (Sahoo and Mukherjee, 2001).

4. Pet food regulation and recall

To ensure safety, pet foods and individual pet food ingredients are regulated by several governmental agencies in addition to meeting manufacturer's quality control and storage standards (Miller et al., 2000). Considering that the intrinsic toxicological properties of a chemical cannot be altered, regulatory agencies consider exposure mitigation the only meaningful opportunity for risk reduction (NRC, 1993). Government regulations of mycotoxin contamination, however, are often compromised by the analytical detection limits, regional prevalence, as well as trade relationships amongst different countries instead of fulfilling the scientific approach of risk assessment and safety determination (Leung et al., 2006).
Scientifically based regulations for the acceptable limit of mycotoxins in pet food would be beneficial. Strict regulations, however, would create greater competition with the human food chain resulting in increased pet food costs and decreased industry profits. It is also possible that the avoidance of severe regulations will promote mycotoxin outbreaks (Boermans and Leung, 2007). Safety and efficacy of foods intended for cats and dogs are of prime interest to manufacturers. Long-lived, healthy consumers (pets) contribute to greater sales, so breakdowns in product quality can have catastrophic effect on profits or even company viability. Recent problems with contamination, while affecting only a small percentage of commercial pet foods, impacted the entire pet food industry (Williard et al., 1994; Anonymous: FDA, 2005; Anonymous: FDA, 2007).
Such experiences have reaffirmed the need for manufacturers to devote extensive resources to documenting product quality. In many cases the processes already in place exceed the recognized standards within the industry. Nonetheless, most companies have increased the screening and sourcing control on ingredients used in pet foods. Regulatory standards are provided at several levels to ensure safety and adequacy of commercial products. In addition, the manufacture and regulation of pet foods is continually progressing forward, which should result in even more veterinary and consumer confidence in commercially manufactured foods (Zicker, 2008).
The FDA has action levels for aflatoxins regulating the levels and species to which contaminated feeds may be fed (CAST, 2003). The European Community levels are more restrictive (FAO Food and Nutrition Paper No. 81, 2004). In the United States, the FDA regulates foods and ingredients that are shipped across state or international boundaries under the authority of the Federal Food, Drug and Cosmetic Act (FFDCA) (Price et al. 1993; Van Houweling et al., 1977).
The FDA regulates enclose cat food, bag of dog food, box of dog treats or snacks. The FDA's regulation of pet food is similar to that for other animal feeds (Table 3). The FFDCA requires that pet foods, like human foods, be safe to eat, produced under sanitary conditions, contain no harmful substances, and be truthfully labeled. AFB_1 is the most toxic

type and is regarded as the "sentinel" substance for all other aflatoxins. Aflatoxin control limit adopted in the US is 20 parts per billion for aflatoxin B_1 (Phillips, 2007). Harmonized regulations for aflatoxins exist in MERCOSUL, a trading block consisting of Argentina, Brazil, Paraguay and Uruguay. Worldwide limits for total aflatoxins in feed may vary (from 0.01 to 50 µg/kg), depending on the destination of the feedstuff as for dairy cattle, for example, that is 50 µg/kg. A relatively flat distribution is apparent with the most occurring limits set at 20 mg/kg (FAO, 2011).

Mycotoxin	Grain for human food		Grain for animal feed	
	USA [a]	EU [b]	USA [a]	EU [b]
Aflatoxins	20 ppb	2-4 ppb [c]	20-300 ppb [d]	10-50 ppb [d]

[a] Munkvold, 2003a

[b] Commission Regulation (EC) Nº 1126/2007

[c] Varies among specific food items

[d] Varies among livestock species

Adapted from Schmale and Munkvold, 2011

Table 3. Recommendations and regulations for safe limits on mycotoxin concentrations in grain in the United States and European Union, as of 2008.

FDA ensures that the ingredients used in pet food are safe and have an appropriate function in the pet food (FDA, 2011). There is no requirement that pet food products have pre-market approval by the FDA. However, depending on the ingredient, the quality control steps may include testing for nutrient content, aflatoxins, or other contaminants that may pose safety risks. Careful testing of susceptible commodities for aflatoxins is necessary and the contaminated lots are eliminated. These standards must meet regulatory requirements for the particular industry standard. However, in many cases company quality control standards exceed the minimal regulatory requirement to insure safety and efficacy of product for dogs and cats. Specifically, in the United States, the FDA monitors pet food and individual pet food ingredients for pesticides, mycotoxins, and heavy metals as part of its Feed Contaminants Program (Van Houweling et al., 1977). For contaminants not covered by a tolerance, an action level, or advisory level, the limit remains unknown, although it is assumed to be theoretically at zero. In the present day analytical methods have become so sensitive that minuscule amounts of contaminants can be detected (Zicker, 2008).

Recalls are actions taken by a firm to remove a product from the market. Recalls may be conducted on a firm's own initiative, by FDA request, or by FDA order under statutory authority (FDA, 2011).

Class I recall: A situation in which there is a reasonable probability that the use of or exposure to a violative product will cause serious adverse health consequences or death.

Class II recall: A situation in which use of or exposure to a violative product may cause temporary or medically reversible adverse health consequences or where the probability of serious adverse health consequences is remote.

Class III recall: A situation in which use of or exposure to a violative product is not likely to cause adverse health consequences.

Market withdrawal: Occurs when a product has a minor violation that would not be subject to FDA legal action. The firm removes the product from the market or corrects the violation. For example, a product removed from the market due to tampering, without evidence of manufacturing or distribution problem, would be a market withdrawal.
Medical device safety alert: Issued in situations where a medical device may present an unreasonable risk of substantial harm. In some case, these situations also are considered recalls.
Miller and Cullor (2000) compared commercial pet foods with other sources of poisoning in dogs and cats. Food ranked well below drugs, insecticides, plants, rodenticides and cleaning products, in terms of frequency of occurrence. Only 1.7% of reported poisonings of dogs and cats could be attributed to food of any type. Despite these statistics, adverse signs in pets are very frequently blamed on the pet's food. Incidents of pet food contamination and illness still occur. In 2005, more than 75 dogs died in the United States after consuming pet food contaminated with aflatoxins, and hundreds more experienced severe liver problems associated with the intoxication (FDA, 2005).
The contaminated pet food was shipped to 22 different states and at least 29 different countries. Diamond Pet Food has discovered aflatoxins in many products manufactured in South Carolina and the problem was associated with the growth of the fungus *Aspergillus flavus*, on corn and other crops. The U.S Food and Drug Administration posted a recall on December 20, 2005, and nineteen different types of pet food (cats and dogs) produced at a single facility in Gaston, South Carolina were removed from sale. Sixteen batches of pet food were found to be contaminated with aflatoxins at levels greater than or equal to 20 ppb. The veterinarians were alarmed because this outbreak caused 100 dog deaths in weeks. The widespread panic that followed this tragic event motivated many pet food companies to set-up routine testing services for aflatoxins (Schmale III, D.G. and Munkvold, 2011, FDA, 2005).
In the end of 2010, the Kroger Company recalled pet food packages that could be contaminated with aflatoxins distributed in stores around many states in USA (Alabama, Arkansas, Georgia, Indiana, Illinois, Kentucky, Texas, Louisiana, Mississippi, Michigan, Missouri, North Carolina, Ohio, South Carolina, Tennessee, Virginia and West Virginia). The recall involved five different kinds of cat and dog foods. The Kroger Company advised the customers to consult veterinarians if their animals showed any signs of sluggishness or lethargy combined with reluctance to eat. Yellowish tints to the eyes or gums, severe blood or diarrhea were also included in the alert of warning signs divulged by industry.
A product recall may be the most effective means of containing the risk in a swift manner. Most commonly, pet food recalls are limited in scope (eg, a single manufacturer) and involve a quickly identified and understood contaminant (eg, *Salmonella* spp., mycotoxins). While recalls may be expensive to conduct, the potential repercussions of failure to honor the request in terms of legal liability and company/brand reputation may be much more costly in the long term. Veterinarians who suspect a case of pet food-borne illness should collect as much information on the food in question as feasible. In fact, a record of the dietary history of a sick animal is always prudent and may become important later if a pattern emerges or a notice of a recall is announced at a later date. Pertinent information may include the manufacturer's or distributor's name and address, the product and variety names, a description of the type of product, and any lot or date codes on the packaging. Effort should be made to determine the place and date where the food was obtained (Dzanis, 2008).

Pet food companies report that even minor changes to color, odor or texture of a pet food that have no bearing on safety are frequently reported to increase complaints to the companies' consumer relations department. Except for overt moldiness, obvious rancidity, or visible inclusion of foreign materials, most incidents of pet food contamination are unlikely to be apparent on gross inspection. Thus, collection of samples for laboratory analysis may be indicated when the food is suspect. Proper handling of the sample as legal evidence may be critical if there is a possibility of a lawsuit at a later date (Miller and Cullor, 2000).

In submission of pet food samples suspected of contamination, effort should be made to improve the chances of detecting the possible contaminant. Vague references to "look for poison" on a sample submission form does not give much assistance. A tentative diagnosis, or at least a thorough description of clinical signs and laboratory findings, may give clues to the facility running the analysis on the suspected food as to which contaminants are likely and hence which analyses to conduct (Dzanis, 2008).

5. Diagnostic and treatment

The diagnosis of mycotoxicosis is a common challenge for veterinarians, because the mycotoxin-induced disease syndromes can easily be confused with other diseases caused by pathogenic microorganisms. The liver is the primary target organ of acute injury from AF ingestion in all species. Although it is difficult to prove that a particular disease outbreak was caused by a mycotoxin (CAST, 2003).

A diagnosis of mycotoxicosis is usually made by feed analysis and histopathology because clinical signs of aflatoxicosis can be nonspecific and confusing. Histologic evaluation of the livers of affected animals and analysis of the feed for mycotoxin content are crucial to confirm the clinical diagnoses. Histopathology signs as bile-duct hyperplasia, hepatocellular degeneration, fatty change of hepatocytes, and mononuclear-cell infiltration of the hepatic parenchyma were observed in broiler chickens fed 1 ppm AFs (Eraslan et al., 2006; Ortatali and Oguz, 2001).

In a 2005 research study, broilers were fed a combination of AFs and fumonisins. The livers of affected birds were enlarged, yellowish, friable, and had rounded borders (Miazzo et al., 2005).

The HE-stained tissue sections were characterized by multifocal cytoplasmatic vacuolation, with a variable location within hepatic lobes. Hepatocellular damage manifested by marked cytoplasmic vacuolation and pyknotic nuclei was reported in a 2006 study of rats administered 2 mg/kg body weight of AFB_1 (Sakr et al., 2006).

Testing for mycotoxins in food and in the patient can be difficult because of variation in toxic concentration and the inconsistent production of toxins (LaBonde, 1995). A complete blood cell count, serum chemistry panel, and analysis of bile acids, ammonia, and urine help to rule out other causes of acute or chronic liver disease (e.g., infectious, neoplastic, chemical, drug-induced, congenital). Serum activity of hepatic enzymes (alanine aminotransferase, aspartate aminotransferase, and alkaline phosphatase) is usually elevated. Serum ammonia and bilirubin concentrations are often increased. If bleeding disorders are found on clinical examination, determination of coagulation times may be helpful. If an animal has died, macroscopic findings may include generalized icterus, liver damage, ascites, widespread hemorrhage, and edema of the gallbladder (Bastianello et al., 1987).

Histologically, varying degrees of liver damage are observed depending on the length of exposure to aflatoxins and their concentrations in the diet. Typical lesions in chronic and subacute cases are bile duct proliferation, varying degrees of fibrosis, hepatocellular fatty degeneration, and megalocytosis. Acutely poisoned dogs show massive fatty degeneration and centrilobular necrosis of the liver as well as widespread hemorrhage. In addition to liver lesions, renal proximal tubular necrosis is often present in dogs poisoned by aflatoxins. Confirmation of aflatoxicosis should include testing of the suspect feed source for aflatoxins (Trucksees and Wood, 1994).

Even if the feed is not visibly moldy, mycotoxins may be present. It is recommended to contact a veterinary diagnostic laboratory for sampling and shipping instructions. Some laboratories also offer testing of fresh liver for aflatoxin B_1. Additionally, a liver biopsy may be useful in ruling out other etiologies of liver disease (Puschner, 2002).

Treatment for hepatic dysfunction is symptomatic and supportive (e.g., fluids, B-complex vitamins, glucose). In many cases, lactated Ringer's solution supplemented with potassium (20 mEq/L) is administered as a maintenance solution. In cases with hypoalbuminemia, administration of dextrose is recommended. Aflatoxicosis resulting in severe hepatic failure may lead to a hypocoagulable status, requiring correction with frozen plasma or whole blood. No antidote is available. The prognosis depends on the extent and severity of liver dysfunction. Monitoring serum biochemical parameters may help to evaluate the extent of liver damage. If liver damage is extensive, the prognosis is guarded to poor. Ammoniation and certain adsorbents are effective in reducing or eliminating the effects of aflatoxins in animals (Park et al., 1988; Puschner, 2002).

While there is no specific treatment for mycotoxicosis, birds that are at high risk of exposure may benefit from supplementation with glucomannans and organic selenium, which appear to decrease the hepatotoxic and CNS changes associated with exposure (Ergün et al., 2006; Dvorska et al., 2007). The best way to protect pet birds from exposure to mycotoxins is to feed only human-grade grain, corn, and peanut products; avoid spoiled foods; and store grain products in cool, dry places (Lightfoot and Yeager, 2008).

6. Preventative strategies

Such experiences have reaffirmed the need for manufacturers to devote extensive resources to documenting product quality. In many cases the processes already in place exceed the recognized standards within the industry. Nonetheless, most companies have increased the screening and sourcing control on ingredients used in pet foods. Regulatory standards are provided at several levels to ensure safety and adequacy of commercial products. In addition, the manufacture and regulation of pet foods is continually progressing forward, which should result in even more veterinary and consumer confidence in commercially manufactured foods (Anonymous: FDA, 2005; 2008, 2011).

A control program for mycotoxins from field to table should involve the criteria of an Hazard Analysis Critical Control Point (HACCP) approach which will require an understanding of the important aspects of the interactions of the toxigenic fungi with crop plants, the on-farm production and harvest methods for crops, the production of livestock using grains and processed feeds, including diagnostic capabilities for mycotoxicoses, and to the development of processed foods for consumption as well as understanding the marketing and trade channels including storage and delivery of foods to the consumer. A good testing protocol for mycotoxins is necessary to manage all of the control points for finally being able to ensure a

food supply free of toxic levels of mycotoxins (Richard, 2007). This system could be applied to prevent the risks of mycotoxins in animals by the pet food industry.

Conventional detection methods for AFB_1 require trained personnel, a laboratory environment, expensive equipment and often several hours or days in analytical time. Several commercial rapid test kits for use in determining the aflatoxin concentration are present in market. These test kits are self contained and thus no additional equipment is required. The kit system provide all the necessary instructions to complete an analysis and it also enables visual evaluation of the results of grains samples on farm or at buying point. It is possible to detect AFB_1 in cereals, nuts, spices and their derived products. Food samples are prepared for analysis by simply shaking the sample by hand in the presence of an extraction solution. However, the biggest challenge is the detection of minimum level of aflatoxin on feed or ingredients. But, a representative sample is essential, because aflatoxins can be concentrated in a few kernels that contaminate an entire load. A multi-level probe sampling at several sites and depths will give the best results. AOAC approved methods generally agree that an initial sample weight of 10 pounds (5 kilograms) is desirable (Byrne, 2008; Phillips, 2007).

Pet food amelioration is often considered a practical solution for mycotoxin contamination. Food processing techniques such as sieving, washing, pearling, ozonation, and acid-based mold inhibition can reduce the mycotoxin content of cereal grains. Dietary supplementation with large neutral amino acids, antioxidants, and omega-3 polyunsaturated fatty acids as well as inclusion of mycotoxin-sequestering agents and detoxifying microbes may ameliorate the harmful effects of mycotoxins in contaminated pet food. Amelioration of pet food, however, should be used as an additional safety factor but not to replace the sound application of risk and safety determination (Leung et al., 2006).

Sorption methods for the detoxification of aflatoxins are being studied and applied for the enterosorption and inactivation of aflatoxins in the gastrointestinal tract. Hydrated sodium calcium aluminosilicate (HSCAS) is a phyllosilicate clay commonly used as an anticaking agent in animal feeds. HSCAS tightly and selectively adsorbs aflatoxin and it has been shown to prevent the adverse effects of aflatoxins in various animals when included in the diet. Studies have also confirmed that HSCAS can alter the bioavailability of aflatoxin in dogs. HSCAS does not interfere with the utilization of vitamins and micronutrients in the diet and protects dogs fed diets with even minimal aflatoxin contamination. However, it does not protect animals against other mycotoxins. Despite regular and careful ingredient screening for aflatoxin, low concentrations may reach the final product undetected. Therefore, HSCAS may provide the petfood industry further assurance of canine diet safety (Bingham et al., 2004).

Bingham et al. (2004) realized a crossover study, using six dogs randomly fed a commercial dog food (no-clay control) or coated with HSCAS (0.5% by weight) were subsequently administered a sub-clinical dose of aflatoxin B_1. Diets were switched and the process repeated. The HSCAS-coated diet significantly reduced urinary aflatoxin M_1 by 48.4% (+/-16.6 SD) versus the control diet. It was demonstrated that HSCAS protected dogs fed diets with even minimal aflatoxin contamination. Despite regular and careful ingredient screening for aflatoxin, low concentrations may reach the final product undetected. Therefore, HSCAS may provide the pet food industry further assurance of canine diet safety.

7. Conclusion

It is known that mycotoxin contamination in pet food poses a serious health threat to pets and recent problems with contamination, while affecting only a small percentage of

commercial pet foods, impacted the entire pet food industry, affecting the confidence of veterinarians and owners. Long-lived, healthy consumers (pets) contribute to greater sales, so breakdowns in product quality can have catastrophic effect on profits or even company viability. More research is needed to better address the pet mycotoxin problem. Safety and efficacy of foods intended for animals are of prime interest to manufacturers because the health problems of pets are of a highly emotional concern, besides the pet food safety is the responsibility of the pet food industry. In the other hand, pet owners must care to store the animal's food at home with regard to avoid fungal contamination, putting the open bags in a clean and dry place, with aeration and protected against humidity from environment. The shelf-life of commercial products must be observed, even at home.

8. References

Association of American Feed Control Officials –AAFCO (2007). Industry Statistics & Trends. In: *American Pet Products Association, Inc.,* Accessed january 09, 2011, Available from: <http://www.americanpetproducts.org/press_industrytrends.asp>.

Adams, C.L., Conlon, P.D. & Long, K.C. (2004). Professional and veterinary competencies: addressing human relations and the human–animal bond in veterinary medicine. *Journal of Veterinary Medical Education,* Vol. 31, pp. 66–71.

Food and Drug Administration- FDA (2005). Diamond Pet Food Recalled Due to Aflatoxin. In: *Recall – Firm Press Release*. Accessed december 19, 2010, Available from: <www.fda.gov/Safety/Recalls/ArchiveRecalls/2005>.

Food and Drug Administration- FDA (2007). Pet Food Recall (Melamine)/Tainted Animal Feed Updated. In: *Recalls & Withdrawals,* Accessed december 19, 2010, Available from: <http://www.fda.gov/oc/opacom/hottopics/petfood.html>.

American Pet Products Association Inc. – APPA (2011). Industry Statistics & Trends. In: 2011 Pet Products Trend Report. Accessed january 16, 2011, Available from: <http://www.americanpetproducts.org/press_industrytrends.asp 2009/2010>.

American Pet Products Manufacturers Association – APPMA (2006). National Pet Owners Survey 2005/2006. In: *APPMA News*, Accessed january 21, 2010, Available from: < http://www.americanpetproducts.org/newsletter/october2006/index.html>.

Armbrecht B.H., Geleta J.N., Shalkop W.T. & Durbin C.G. (1971). A subacute exposure of beagle dogs to aflatoxin. *Toxicology and Applied Pharmacology*, Vol. 18, pp. 579–85.

Asao, T., Buchi, G., Abdel-Kader, M.M., Chang, S.B., Wick, E.L. & Wogan, G.N. (1963). Aflatoxins B and G. *Journal of the American Chemical Society*, Vol. 85, pp. 1706–1707.

Bailly J.D., Raymond I., Le Bars P., Leclerc J.L., Le Bars J. & Guerre P. (1997). Aflatoxine canine: cas clinique et revue bibliographique. *Revue de Médecine Vétérinaire.*Vol. 148, pp. 907–14.

Bárta, M. Adámková, T. Petr & J. Bártová. (1990). Dose and time dependence of chromosomal aberration yields of bone marrow cells in male Chinese hamsters after a single i.p. injection of aflatoxin B_1. *Mutation Research*, Vol. 244, pp. 189-195.

Bastianello, S.S., Nesbit, J.W., Williams, M.C. & Lange, A.L. (1987). Pathological findings in a natural outbreak of aflatoxicosis in dogs. *Onderstepoort Journal of Veterinary Research*, Vol. 54, pp. 635–40.

Bhat, R. (1999). Mycotoxin contamination of food and feeds. Mycotoxin contamination of foods and feeds. *Proceedings of Working document of the Third Joint FAO/WHO/UNEP*.Tunis, Tunisia, March 1999.

Bingham A.K., Huebner H.J., Phillips T.D. & Bauer J.E (2004). Identification and reduction of urinary aflatoxin metabolites in dogs. *Food and Chemical Toxicology*, Vol. 42, 11, pp. 1851-1858.

Boermans, H. J. & Leung, M. C.K. (2007). Mycotoxins and the pet food industry: Toxicological evidence and risk assessment. *International Journal of Food Microbiology, Vol.* 19, pp. 95-102.

Bradburn, R.D.C. & Blunden, G. (1994). The aetiology of turkey "x" disease. *Phytochemistry*, 35, 3, pp. 817.

Brera, C., Catano, C., de Santis, B., Debegnach, F., de Giacomo, M., Pannunzi, E. & Miraglia, M. (2006). Effect of industrial processing on the distribution of aflatoxins and zearalenone in corn-milling fractions. *Journal of Agricultural and Food Chemistry*, Vol. 54, pp. 5014–5019.

Busby, W.F., & G.N. Wogan (1984). Aflatoxins, In: *Chemical Carcinogens*, C.D. Searle, pp. 945-1136, ACS Monograph 182, Washington, DC.

Byrne, J. Aflatoxin detection kit validated by USDA (2008). In: *Quality and Safety*, Accessed december 23, 2010, Available from: <http://www.foodproductiondaily.com/Quality-Safety/Aflatoxin-detection-kit-validated-by-USDA>.

CAST - Council for Agricultural Science and Technology (2003). Mycotoxins: risks in plants animal and human systems. *Task Force Report 139*. Ames.

CAST - Council for Agricultural Science and Technology (1989). Mycotoxins: economic and health risks. *Task Force Report 116*. Ames, pp. 1–9.

Chaffee V.W., Edds G.T., Himes J.A. & Neal F.C. (1969). Aflatoxicosis in dogs. *American Journal of Veterinary Research,* Vol. 30, pp. 1737–49.

Covello, V.T. & Merkhofer, M.W. (1993). *Risk Assessment Methods Approaches for Assessing Health and Environmental Risks*. pp. 318, Plenum, New York.

Cowell, C. S., Stout, N. P., Brinkmann, M. F., Moser, E. A. & Crane, S. W. (2000). Making of commercial pet foods, In: *Small Animal Clinical Nutrition*, Hand, M. S., Thatcher, C. D., Remillard & R. L., Roudebush, pp. 127–146, Walsworth Publishing Company, Marceline, USA.

Cable News Network- CNN (2010). Kroger announces select pet food recall. In: *Pet Food*. Accessed january 14, 2011, Available from: <http://articles.cnn.com/2010-12-19/us/pet.food.recall_1_pet-food-aflatoxin-toxic-chemical-byproduct?_s=PM:US>.

Crane, S.W., Griffin, R.W. & Messent, P.R. (2000). Introduction to commercial pet foods, In: *Small Animal Clinical Nutrition*, Hand, M.S., Thatcher, C.D., Remillard, R.L. & Roudebush, P., pp. 111-126, Walsworth Publishing Company, Marceline, USA.

Croy, R.G., Essigman, J.M., Reinhold, V.N. & Wogan, G.N. (1978). Identification of the principal aflatoxin BrDNA adduct formed in vivo in rat liver, *Proceedings of the National Academy of Sciences*, Vol. 75.

Cullen J.M. & Newberne, P.M. (1994). Acute hepatotoxicity of aflatoxins, In: *The toxicology of aflatoxins: human health, veterinary and agricultural significance*. Eaton, D.L. & Groopman, J.D., pp. 3–26, Academic Press, San Diego, USA.

Degernes, L.A. (1995).Toxicities in waterfowl. *Seminars in Avian and Exotic Pet Medicine*, Vol. 4, (1), pp. 15–22.

Devegowda, G. & Castaldo, D. (2000). Mycotoxins: hidden killers in pet foods. Is there a solution? In: *Technical Symposium on Mycotoxins*. Alltech Inc, Nicholasville, Kentucky, USA.

Dog Food Advisor. In: Kroger Dog Food Recalled for Aflatoxin Accessed january 25, 2011, Available from: <http://www.dogfoodadvisor.com/dog-food-recall/kroger-dog-food-recall/>.

Dunn, K.L., Mehler, S.J. & Greenberg, H.S. (2005). Social work with a pet loss support group in a university veterinary hospital. *Social Work in Health Care*, Vol. 41, pp. 159–170.

Dvorska, J.E., Pappas, A.C., Karadas, F., Speake, B.K. & Surai, P.F. (2007). Protective effect of modified glucomannans and organic selenium against antioxidant depletion in the chicken liver due to T-2 toxincontaminated feed consumption. *Comparative Biochemistry and Physiology. Part C, Pharmacology, Toxicology and Endocrinology*, Vol. 145, (4), pp. 582–7.

Dzanis, D.A. (2008). Anatomy of a Recall. *Topical Review*, 23, 3, pp. 133 – 136.

Eaton DL, Ramsdell HS, Neal GE. (1994a). Biotransformation of aflatoxins. In: *The toxicology of aflatoxins: human health, veterinary and agricultural significance*, Eaton, D.L., Groopman, J.D., pp. 45–47, Academic Press, San Diego, USA.

Eaton, D.L. & Gallagher, E.P. (1994b). Mechanisms of aflatoxin carcinogenesis. *Annual Review of Pharmacology and Toxicology*, Vol. 34, pp. 135–72.

Eraslan, G., Essiz, D., Akdogan, M., E. Karaoz, M. Oncu & Z. Ozyildiz (2006). Efficacy of dietary sodium bentonite against subchronic exposure to dietary aflatoxin in broilers. *Bulletin of the Veterinary Institute in Pulawy*, Vol. 50, pp. 107-112.

Ergün, E., Ergün, L. & Esxsiz, D. (2006). Light and electron microscopic studies on liver histology in chicks fed aflatoxin. Dtsch Tierarztl Wochenschr, 113, 10, pp. 363–368.

FAO Food and Nutrition. (2004). Worldwide Regulations for Mycotoxins in Food and Feed in 2003, Paper. nº 81, pp. 180.

Faustman, E.M. & Omenn, G.S. (2001). Risk assessment. In: *Casarett and Doull's Toxicology: The Basic Science of Poisons*, Klaassen, C.D., pp. 83–104, McGraw-Hill, USA.

Food and Drug Administration – FDA (2005). Center for Food Safety and Applied Nutrition. In: *Diamond Pet Food Recalled Due to Aflatoxin.* Accessed january 14, 2011, Available from: <http://www.fda.gov/Safety/Recalls/ArchiveRecalls/2005/ucm111929.htm>.

Food and Drug Administration – FDA (2009). Center for Food Safety and Applied Nutrition. In: *Interim melamine and analogues safety/risk assessment.* Accessed january 14, 2011, Available from: <http://www.cfsan.fda.gov/_dms/melamra. html.>.

Federal Food, Drug and Cosmetic Act, as Amended. (2004). Superintendent of Documents. Washington, DC: U.S. Government Printing Office.

Feuell, A.J. (1966). Aflatoxin In groundnuts. Problems of detoxification. *Tropical Science*, Vol. 8, pp. 61-70.

Garland, T. & Reagor, J. (2001). Chronic canine aflatoxicosis and management of an epidemic, In: *Mycotoxins and Phycotoxins in Perspective at the Turn of the Millennium*, deKoe,W., Samson, R., van Egmond, H., Gilbert, J., Sabino, M., pp. 231–236, Ponsen & Looven, Wageningen, Netherlands.

Groopman, J.D., Cain, L.G. & Krusler, C.C. (1988). Aflatoxin exposure in human populations: measurements and relationship to cancer, *CRC Critical Reviews in Toxicology,* Vol. 19, pp. 113-145.

Haschek, W.M., Voss, K.A. & Beasley, V.R. (2002). In: *Selected Mycotoxins Affecting Animal and Human Health, Handbook of Toxicologic Pathology* (Second Edition), pp. 645-699, Academic Press, London.

Henke, S.E., Gallardo, V.C., Martinez, B. & Balley, R. (2001). Survey of aflatoxin concentrations in wild bird seed purchased in Texas. *J Wildl Dis*, 37, pp. 831–835.

Hooser, S.B. & Talcott, P.A. (2006). In: *Mycotoxins* in *Small Animal Toxicology* (Second Edition), Peterson, M.E. & Talcott, P.A., pp. 888-897, Elsevier Inc, USA.

Hunter, J.H. (1969). Growth and aflatoxin production in shelled corn by the *Aspergillus flavus* group as related to relative humidity and temperature. Ph.D. thesis. Purdue University, West Lafayette, IN.

Hussein, S.H. & Brasel, J.M. (2001). Review: Toxicity, metabolism, and impact of mycotoxins on humans and animals. *Toxicology,* Vol. 167, pp. 101–134.

Kabak, B., Dobson, A.D., Var, I. (2006). Strategies to prevent mycotoxin contamination of food and animal feed: a review. *Food Science and Nutrition*, Vol. 46, pp. 593–619.

Ketterer, P.J., Williams, E.S, Blaney, B.J. & Connolle, M.D. (1975). Canine aflatoxicosis. *Australian Veterinary Journal,* Vol. 51, pp. 355–357.

LaBonde, J. (1995). Toxicity in pet avian patients. *Seminars in Avian and Exotic Pet Medicine*, Vol. 4 (1), pp. 23–31.

Leung, M.C.K., Díaz-Llano, G. & Smith, T.K. (2006). Mycotoxins in Pet Food: A Review on Worldwide Prevalence and Preventative Strategies. *Journal of Agricultural and Food Chemistry,* Vol. 54, 26, pp. 9623–9635.

Lightfoot, T.L. & Yeager, J.M. (2008). Pet Bird Toxicity and Related Environmental Concerns., Vol. *Veterinary Clinics of North America: Exotic Animal Practice*, Vol. 11, pp. 245–246.

Miazzo, R., Peralta, M.F., Magnoli, C., Salvano, M., Ferrero, S. & Chiacchiera, S.M. (2005). Efficacy of sodium bentonite as a detoxifier of broiler feed contaminated with aflatoxin and fumonisin. *Poultry Science*, Vol. 84, pp. 1-8.

Milani, M.M. (1996). The importance of the human–animal bond. *Journal of the American Veterinary Medical Association,* Vol. 209, pp. 1064–1065.

Miller, D.M. & Wilson, D.M. (1994). Veterinary diseases related to aflatoxin. In: *The Toxicology of Aflatoxins: Human Health, Veterinary, and Agricultural Significance*. Eaton, D.L. & Groopman, J.D., pp. 347–364, Academic Press, San Diego, USA.

Miller, E.P. & Cullor, J.S. (2000). Food safety. In: *Small Animal Clinical Nutrition*, Hand, M.S., Thatcher, C.D., Remillard, R.L., pp. 183-198, Topeka, KS, Mark Morris Institute.

Moss, M.O. (1996). Mycotoxic fungi, In: *Microbial Food Poisoning*, Eley, A.R., pp. 75-93, Chapman & Hall, New York, USA.

MSNBS News Services (2006). Toxic pet food may have killed dozens of dogs. In: *Pet Health*. Accessed january 23, 2011, Available from: <http:// www.msnbc.msn.com/id/10771943/>.

Munkvold, G.P. (2003). Cultural and genetic approaches to managing mycotoxins in maize. *Annual Review of Phytopathology,*Vol. 41, pp. 99–11.

Neal, G.E., Judah, D.J. & Green, J.A. (1986). The in vitro metabolism of aflatoxin B_1 catalysed by hepatic microsomes isolated from control or 3-methylcholanthrene stimulated rats and quail, *Toxicology and Applied Pharmacology,* Vol. 82, pp. 454-460.

Newberne, P.M., Wogan, G.N. (1968). Sequential morphologic changes in aflatoxin B carcinogenesis in the rat. *Cancer Research*, Vol. 28, pp. 700–781.

Newberne, P.M., Russo, R. & Wogan, G.N. (1966). Acute toxicity of aflatoxin B_1 in the dog. *Veterinary Pathology,* Vol. 3, pp. 331–340.

National Research Council (NRC), 1993. Issues in Risk Assessment. National Academy Press, Washington, DC, USA.

Ominski, K.H., Marquardt, R.R., Sinah, R.N & Abramson, D. (1994). Ecological aspects of growth and mycotoxin production by storage fungi. In: *Mycotoxins in Grain-compounds other than Aflatoxin*, Miller, J.D. & Trenholm, H.L., pp. 287–312, Eagan Press, St. Paul.

Ortatali M. & Oguz H. (2001). Ameliorative effects of dietary clinoptilolite on pathological changes in broiler chickens during aflatoxicosis. *Research in Veterinary Science*, Vol. 71, pp. 59-66.

Paine, A.J., Marrs, T.C. (2000). The design of toxicological studies, In: *General and Applied Toxicology*, Ballantyne, B., Marrs, T., Syversen, T., pp. 321–334, MacMillan References Texts Ltd, London, United Kingdom.

Park, D.L., Lee, L.S. & Pohland, A.E. (1988). Review of the decontamination of afltoxins by ammoniation: current status and regulation. *Journal Association of Official Analytical Chemists*, Vol. 71, pp. 685–703.

Pereyra, M.L.G., Carvalho, E.C.Q., Tissera, J.L., Keller, K.M., Magnoli, C.E., Rosa, C.A.R., Dalcero, A.M. & Cavaglieri, L.R. (2008). An outbreak of acute aflatoxicosis on a chinchilla (*Chinchilla lanigera*) farm in Argentina. Case Reports. *Journal of Veterinary Diagnostic Investigation,* Vol. 20, 6, pp. 853-856.

Phillips, T. (2010). Aflatoxin insurance, In: *A clay called HSCAS can help petfood manufacturers avoid a crisis*. Accessed january 11, 2011, Available from: < http://www.petfoodindustry.com/4019.html>.

Pitt, J.I. & Miscamble, B.F. (1995). Water relations of *Aspergillus flavus* and closely related species. *Journal of Food Protection*, Vol. 58, pp. 86–90.

Plumlee, K.H. (2004). Chapter 23 – Mycotoxins. *Clinical Veterinary Toxicology*, pp. 231-281. Mosby Inc., St. Louis.

Price, W.D., Lovell, R.A. & McChesney, D.G. (1993). Naturally occurring toxins in feedstuffs: Center for Veterinary Medicine Perspective. *Journal of Animal Science*, Vol. 71, pp. 2556-2562.

Puschner, B. (2002). Mycotoxins. *Veterinary Clinics of North America: Small Animal Practice*, Vol. 32, pp. 409–419.

Rauber, R.H., Dilkin, P., Giacomini, L. Z., Araújo de Almeida, C. A. & Mallmann, C. A. (2007). Performance of turkey poults fed different doses of aflatoxins in the diet. *Poult Science*, Vol. 86, 8, pp. 1620–1624.

Razzai–Fazeli, E., 2005. Effects of mycotoxins on domestic pet species. In: *The Mycotoxin Blue Book*. Diaz, D., pp. , 77–92, Nottingham University Press, Nottingham, UK.

Richard, J.L. (2007). Some major mycotoxins and their mycotoxicoses—An overview. *International Journal of Food Microbiology*, Vol. 119, pp. 3–10.

Robens, J.F. & Richard, J.L. (1992). Aflatoxins in animal and human health. *Reviews of Environmental Contamination & Toxicology*, pp. 127.

Sahoo, P.K. & Mukherjee, S.C. (2001). Immunosuppressive effects of aflatoxin B_1 in Indian major carp (*Labeo rohita*). *Comparative Immunology, Microbiology & Infectious Diseases*, Vol. 24, (3), pp. 143-149.

Sakr, S.A., Lamfon, H.A. & El-Abd, S.F. (2006). Ameliorative effects of Lupinus seeds on histopathological and biochemical changes induced by aflatoxin-B_1 in rat liver. *Journal of Applied Science Research*, Vol. 2, pp. 290-295.

Schaafsma, A.W., Limay-Rios, V., Paul, D.E. & Miller, J.D. (2009). Mycotoxins in fuel ethanol co-products derived from maize: a mass balance for deoxynivalenol. *Journal of the Science of Food and Agriculture,* Vol. 89, pp. 1574–1580.

Schmale III, D.G. & Munkvold, G.P., (2009). Mycotoxins in Crops: A Threat to Human and Domestic Animal Health. In: *The Plant Health Instructor*, Accessed january 14, 2011, Available from: <http://www.apsnet.org/edcenter/intropp/topics/Mycotoxins/Pages/References.aspx, The American Phytopathological Society>.

Schmidt, R.E. & Panciera, J.R. (1980). Effects of aflatoxin on pregnant hamsters and hamsters foetuses. *Journal of Comparative Pathology*, Vol. 90, pp. 339-346.

Schweitzer, S.H., Ouist, C.F., Grimes, G.L & Forest, D.L. (2001). Aflatoxin levels in corn available as wild turkey feed in Georgia. *Journal of Wildlife Diseases*, Vol. 37, 3, pp. 657–659.

Scudamore, K.A., Hetmanski, M.T., Chan, H.K. & Collins, S. (1997). Occurrence of mycotoxins in raw ingredientsused for animal feeding stuffs in the United Kingdom in 1992. *Food Additives & Contaminants*, Vol. 14, pp. 157-173.

Sharma, R. & Márquez, C. (2001). Determination of aflatoxins in domestic pet foods (dog and cas) using immunoaffinity column and HPLC. *Animal Feed Science and Technology,* Vol. 93, pp. 109-114.

Shi, X.D., Li, X.T., Fu, J.L., Sheng, J.J., Gui, Y.L., Hui-J.Y., Zhen, Y.D. & Yong, J.L. (2010). Toxic effects and residue of aflatoxin B_1 in tilapia (*Oreochromis niloticus* × *O. aureus*) during long-term dietary exposure. *Aquaculture*, Vol. 307, pp. 233-240.

Siame, B.A., Mpuchane, S.F., Gashe, B.A., Allotey, J. & Teffera, G. (1998). Occurrence of aflatoxins fumonisin B_1 and zearalenone in foods and feeds in Botswana. *Journal of Food Protection*, Vol. 61, pp. 1670-1673.

Stenske, K.A., Smith, J.R., Newman, S.J., Newman, L.B. & Kirk, C.A. (2006).Aflatoxicosis in dogs and dealing with suspected contaminated commercial foods. *Journal of the American Veterinary Medical Association*, Vol. 228, pp. 1686–1691.

Trucksees, M.W. & Wood, G.E. Recent method of analysis for aflatoxins in food and feeds. In: *The toxicology of aflatoxins: human health, veterinary and agricultural significance*. Eaton, D.L. & Groopman, J.D., pp. 73-88, Academic Press, San Diego, USA.

United States Environmental Protection Agency –USEPA (1995). The use of the benchmark dose approach. Health Risk Assessment. USEPA, Washington, DC, USA.

Van Der Zijden, A.S.M., Blanch Kolensmid, W.A.A. & Boldingh J. (1962). Isolation in crystalline form of a toxin responsible for turkey X disease. *Nature*, London, 195, pp. 1062.

Van Houweling, C.D., Bixler, W.B. & McDowell, J.R. (1977). Role of the Food and Drug Administration concerning chemical contaminants in animal feeds. *Journal of the American Veterinary Medical Association*, Vol. 171, pp. 1153-1156.

Wangikar, P.B., Dwivedi, P., Sinha, N., Sharma, A.K. & Telang, A.G. (2005). Effects of aflatoxin B_1 on embryo fetal development in rabbits, *Food and Chemical Toxicology*, Vol. 43, pp. 607–615.

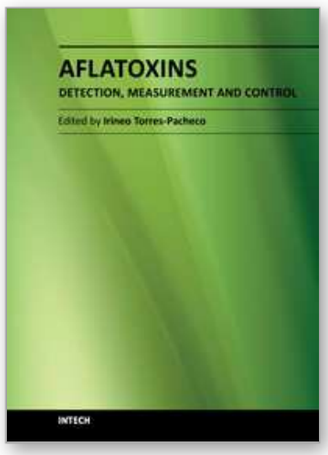
Willard, M. D., Simpson, R. B. Delles, K. , Cohen, N. D., Fossum, T. W. Kolp, E. D. L. & Reinhart G. A. (1994). Preparation with Food Safety. Retrieved from < www.dogcathomeprepareddiet.com/food_safety_and_preparation.html>.

World Health Organization – WHO (1993). International Agency for Research on Cancer (IARC), Monographs on evaluation of carcinogenic risks to humans.

Yegani, M., Chowdhury, S.R., Oinas, N., MacDonald E. & Smith, T. (2006). Effects of feeding grains naturally contaminated with Fusarium mycotoxins on brain regional neurochemistry of laying hens, turkey poults, and broiler breeder hens. *Poultry Science*, Vol. 85 (12), pp. 2117–23.

Zain, M.E. Impact of mycotoxins on humans and animals. J. Saudi Chem. Soc. Article in Press, doi:10.1016/j.jscs.2010.06.006.

Zicker, S.C. (2008). Evaluating Pet Foods: How Confident Are You When You Recommend a Commercial Pet Food? *Topics in Companion Animal Medicine,* Vol. 23 (3), pp. 121-126.



Aflatoxins - Detection, Measurement and Control

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This book is divided into three sections. The section called Aflatoxin Contamination discusses the importance that this subject has for a country like the case of China and mentions examples that illustrate the ubiquity of aflatoxins in various commodities The section Measurement and Analysis, describes the concept of measurement and analysis of aflatoxins from a historical prespective, the legal, and the state of the art in methodologies and techniques. Finally the section entitled Approaches for Prevention and Control of Aflatoxins on Crops and on Different Foods, describes actions to prevent and mitigate the genotoxic effect of one of the most conspicuous aflatoxins, AFB1. In turn, it points out interventions to reduce identified aflatoxin-induced illness at agricultural, dietary and strategies that can control aflatoxin. Besides the preventive management, several approaches have been employed, including physical, chemical biological treatments and solvent extraction to detoxify AF in contaminated feeds and feedstuffs.

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