Mycotoxicosis in Swine: a Review


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Abstract

Mycotoxins are toxic substances resulting from the secondary metabolism of several strains of filamentous fungi, thereby lowering the immune system, facilitate the emergence of diseases, and reduce weight gain, leading to numerous reproductive and production losses. In tropical and subtropical climates such as Brazil, the fungal development is favored by several factors such as, excellent moisture and temperature conditions. The presumptive diagnosis of mycotoxicosis is based on observation of clinical signs of the intoxicated animals and analysis of environmental data relating to the collection and storage of cereals used in feed for pigs. The treatment presents a great challenge, and the removal of the contaminated food to the first measure to be adopted, improving the prognosis. The correct way of drying grain is the best way to avoid fungal growth and mycotoxin production. It is possible to detect fungi and aflatoxins in seeds monitored by sampling and biochemical tests, and tests used to establish acceptable limits for acceptance of products for import / export. The aim of this study was a literature review on the effects caused by mycotoxins in swine, symptoms, prevention methods and treatment. The recognition of problems caused by mycotoxins in food and feed is the first step to prevention and reduction of the problem, using methods for their removal or decontamination routine inspection, agricultural practices that prevent contamination and development of fungi to ensure raw good quality for making good food for the animals.

Key words: Mycotoxins, pigs, animal production

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Introduction

Mycotoxins are toxic substances resulting of the secondary metabolism of several strains of filamentous fungi. It is organic compound of low molecular weight and not has immunogenicity. For producers and technicians was something strange and abstract until the end of the 80's. With the intensification of research in the 90's, began to emerge the causes of numerous symptoms observed in swine, previously assigned to other events of animal health and management. (Nuvital, 2003). In tropical and subtropical climates such as Brazil, fungal development is favored by several factors such as excellent humidity and temperature conditions. Growth and mycotoxins production in cereals, especially in peanuts, corn, wheat, barley, sorghum and rice, where usually are a highly nutritious substrate for their development, can occur at various stages of development, maturation, harvest, transport, processing or storage of grain. Therefore, the reduction of grain humidity by drying is very important to reduce contamination levels.

More of four hundred mycotoxins, known at the present time, are produced by approximately one hundred of fungi, and the main mycotoxins and target organs in swine are: aflatoxins (produced by fungi of the genus Aspergillus such as A. flavus and A. parasiticus) that mainly affect the liver; zearalenone in the reproductive system; ochratoxin (produced by Aspergillus ochraceus and several species of the genus Penicillium) reaching the kidneys; fusarium toxins, which have as main representatives the trichothecenes (damaging the digestive tract) and the fumonisins (in the lung), produced by several species of the genus Fusarium (Pinto and Vaamonde, 1996) (Table 1). In Table 2, the maximum levels of mycotoxins in swine are found.

The mycotoxins decreased immunity, facilitating the emergence of diseases, reduce weight gain, cause numerous reproductive and production damages. The appearance of intoxication signs is closely related to dose and duration of consumption of each toxin. Approximately 90% of intoxications are chronic and have no specific clinical signs and can be easily confused with malnutrition, management deficiency or other chronic diseases that imply in the reduction of productivity of animals. (Dilkin, 2002). Few times mycotoxicosis manifest as acute disease, resulting in the death of animals. Therapeutic methods have low efficiency in order to reduce the impact of intoxications. Preventive measures, widely used, have good efficacy and cost benefit extremely favorable. The aim of this study was realize a review literature on the effects caused by mycotoxins in swine, symptomatology, prevention methods and treatment.

Main mycotoxins in the contamination swine

Aflatoxins

Aflatoxins B1, B2, G1 and G2, present in approximately 38% of the rations of swine, are responsible by the swine mycotoxicosis, of the point of view clinical and economic, of major important, representing a condition extremely serious for animal health. Sows that ingest aflatoxin B1 can eliminate aflatoxin M1 by milk, intoxicating piglets. The average contamination in cereals is of 18μg/kg, and can be found corn samples with up to 17 mg/kg (Dilkin, 2002), value corresponding to 850 times the limit allowed by law for this mycotoxin, in agricultural products. The LD50 of aflatoxin for swine is very low (0.6 mg/kg) and is considered the maximum safety limit of 50 μg/kg of food (Mallmann et al 1994). The aflatoxins act mainly in the liver where are biotransformed. Aflatoxin B1 can be transformed into aflatoxicol that is a reservoir metabolic of this toxin. In turn, the epoxidation of aflatoxin transforms in a radical of high covalent which determines their connection with nucleic acids. It explains the possibility of producing genetic alterations, giving to this mycotoxins carcinogenic characteristic. In turn, the hydration of aflatoxins in the liver produces aflatoxin B2-alpha, which directly interferes in the protein synthesis, leading to conditions of immunosuppression, interference in the blood coagulation and other consequences of the changes caused by these metabolic defects (Pier et al 1980).

Clinical signs of acute aflatoxicosis may start 6 hours after ingestion, leading to a severe depression, inappetence, blood in stools, muscle tremors, incoordination with hyperthermia (up to 41 °C), and the death may occur in 12-24 hours following. In
subacute intoxications, the clinical signs are slower evolution, observing hyporexia, lethargy and depression. In parallel, the animals may have icteric aspect, dehydrated and emaciated, with areas of red color purple in skin, and progressive weight loss. The chronic intoxication manifests by the decrease in weight gain and feed conversion, inappetence, poor general appearance and sometimes diarrhea. With progression to the final stages, there are often signs of ataxia, jaundice and sometimes convulsions (Cook and Alstine, 1989). When the toxin is ingested at higher levels, the liver presents fatty degeneration, lobular necrosis with growth of basophilic cells in the periphery of the lobe, bile duct proliferation and cirrhosis. The jaundice of the carcass, associated at the liver emaciated and yellow are very strong indications of intoxication. The gallbladder may be emaciated and friable liver and hyperemic, especially in cases of acute intoxication. It also occurs a decrease in blood coagulation time, and can observe bloody fluid collections in the cavities as well as in mucous and hemorrhage in muscle mass (Mallmann et al., 1994).

### Table 1: Major mycotoxins, species most affected clinical signs and lesions.

<table>
<thead>
<tr>
<th>Mycotoxins</th>
<th>Species most affected</th>
<th>Main clinical signs and lesions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aflatoxins</td>
<td>All</td>
<td>Decreased weight gain, digestive disorders, hepatopathy, anorexia, ataxia, tremors and death</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Vulvovaginitis</td>
</tr>
<tr>
<td>Zearalenone</td>
<td>Swine</td>
<td>Leukoencephalomalacia equine, porcine pulmonary edema and decreased performance of poultry</td>
</tr>
<tr>
<td>Fumonisins</td>
<td>Equine, Swine and Poultry</td>
<td>Reduction or refusal of feed intake, digestive disorders with ulceration and vomiting and bleeding visceral</td>
</tr>
<tr>
<td>Trichothecenes</td>
<td>Monogastric</td>
<td>Nephropathies</td>
</tr>
<tr>
<td>Ochratoxin A</td>
<td>Swine and Human</td>
<td></td>
</tr>
</tbody>
</table>

### Table 2: Maximum levels of mycotoxins in swine

<table>
<thead>
<tr>
<th></th>
<th>Deoxynivalenol (DON or Vomitoxin)</th>
<th>Zearalenone</th>
<th>Aflatoxins</th>
</tr>
</thead>
<tbody>
<tr>
<td>Swine &lt; 34 Kg</td>
<td>&lt;300</td>
<td>200</td>
<td>20</td>
</tr>
<tr>
<td>Swine 34 a 57 Kg</td>
<td>&lt;300</td>
<td>200</td>
<td>50</td>
</tr>
<tr>
<td>Swine 57 kg at slaughter</td>
<td>&lt;300</td>
<td>200</td>
<td>100</td>
</tr>
<tr>
<td>Herds of Matrices</td>
<td>&lt;300</td>
<td>100</td>
<td>50</td>
</tr>
<tr>
<td>Males of Reproduction</td>
<td>&lt;300</td>
<td>100</td>
<td>50</td>
</tr>
</tbody>
</table>

**Zearalenone (ZEA)**

Zearalenone (ZEA) occurs in practically all the cereals, especially in winter crops, contaminated by fungi of the genus *Fusarium*. The natural contamination occurs in barley, corn, sorghum, oats and rations produced based on these products, and the evaluations of contamination with ZEA showed positivity close to 5%. The average concentration of ZEA found was 18 μg/kg and the maximum level detected was 9.7 mg/kg (Dilkin, 2002). The action of this toxin is through the stimulus at the cytoplasmic estrogen receptors, increasing protein synthesis in the reproductive tract. Consequently, the secretion of the endometrial cells, uterine protein synthesis and the weight of the reproductive tract are increased. These changes can lead to pseudo-gestation by maintenance of the corpus luteum, and lead to conditions characterized by vulvovaginitis, stillborn and weak piglets, and often to splay-leg (syndrome of the members open).

It also can observe a marked reduction in conception rates, accompanied of repetition of estrus. The intoxication mimics the estrus and the newborns piglets may show clinical signs, characterized as vulvovaginitis infant (Edwards et al., 1987a). In young males, the toxin causes feminization, including edema of prepuce, testicular atrophy and increased mammary gland, but these changes apparently do not lead to effects on reproductive capacity when adult. In boars, the
reduction of the libido, as well as a slight reduction on the sperm quality can be observed (Edwards et al., 1987b).

**Ochratoxins (OTA)**

The ochratoxins (OTA) are produced by *Penicillium* and *Aspergillus* fungi presenting an optimized development at temperatures between 5 and 24 °C. The incidence of OTA in the south hemisphere is low, less than 5%, being practically restricted to the north hemisphere with contamination levels 10 times higher. The change in glomerular filtration and losses function of the proximal convoluted tubule is the major damage of the intoxication by OTA, leading to loss of capacity of urinary concentration. The ochratoxicosis in swine cause reduction of weight gain, clinical signs characterized by polydipsia and polyuria, and renal lesions. Doses of 200 μg/kg of OTA in the ration were sufficient for cause nephropathy in the animals, leading to a negative effect on feed conversion and weight gain. The mortality can reach 90% in the affected lots (Krogh et al., 1979).

**Fumonisins**

The fumonisins belong to a large group of mycotoxins produced by fungi of the genus *Fusarium*, natural contaminants of cereals, mainly corn and byproducts. The occurrence of fumonisin B1 in food produced in Brazil has been described by several researchers (Rodriguezamaya, 2000), reaching a positivity of 90% with levels up to 300 mg/kg food. This is the main mycotoxin of this group primarily affecting swine and poultry. Fumonisin B1 is the most abundant metabolite of this group of mycotoxins, representing about 70% in naturally contaminated foods. Fumonisins B2 and B3 occur in lower concentrations (Shephard et al., 1996).

The swine have a high sensitivity to fumonisins, supporting only concentrations below 10 mg/kg food. This verification has been observed in several natural outbreaks and experimental (Haschek et al., 1992). In swine, the main target organs are the lungs, liver and heart, and the specific syndrome in this species is the porcine pulmonary edema, usually with hydrothorax (Osweiler et al., 1992; Smith et al., 2000). This change is caused by ingestion of high doses of the mycotoxin for short periods. The larger damages are due to the ingestion of low doses of the toxin, which induce hepatic and hyperplastic lesions in the esophageal mucosa in weaned pigs (Casteel et al., 1993). In these cases, it can observe mainly the decrease of weight gain of swine.

**Trichothecenes (TCT)**

The trichothecenes are a chemical group of fungal metabolites with the same basic structure, produced mainly by fungi of the genus *Fusarium* such as *F. graminearum* and *F. tricinctum*. More than a hundred of TCT are known. According to the molecular structure are divided into two major groups: single-chain and macrocyclic. Only some have economic importance in Brazil, and deoxynivalenol (DON or vomitoxin) and the T-2 toxin the main representatives. The occurrence of TCT is significant in winter crops such as wheat, barley, oats, rice and rye, cultivated at low temperatures, varying between 6 and 24 °C. The concentrations of DON often limited between 0.1 to 41.6 mg/kg with an average of 2.4 to 4 mg/kg. Natural contamination levels of DON, DAS, T-2 and NIV often reach up to 10 mg/kg, with few exceptions showing levels of 15-40 mg/kg (Dilkin 2002). Globally, DON is the contaminant of cereals more common, accompanied in some areas by nivalenol (NIV). The presence concomitant of others TCT and others *Fusarium* toxins in the same lot of cereals may occur (OMS, 1983). Swine and other monogastric animals have increased sensitivity to TCT, followed by poultry. The NIV and DON induce food refusal and weight loss, have similar toxicities and a combined level less than 0.4 mg/kg is described as acceptable (for swine, which are relatively susceptible), while more than 2.0 mg/kg is always unacceptable (Dilkin and Mallmann, 2004).

The TCT act by inhibiting the enzyme peptidyl transferase, thereby decreasing protein synthesis, which mainly affects cells in active division, such as the gastrointestinal tract, skin and lymphoid cells, erythroid and vital organs. (Dilkin, 2002). The trichotheccenes are immunosuppressive and are also associated with hemorrhages, and prothrombin time is increased significantly, but the primary factor of the hemorrhage is the diminution in blood coagulation factor VII. The intoxications by TCT
lead refusal feeds, vomit, reduction in the feed conversion and diarrhea. The bloody syndrome produced by T2 toxin is characterized by the occurrence of dermatitis, abortions, and nervous disorders, gastric and visceral hemorrhages. All TCT can be acutely lethal. But the biggest problems tend to be subacute toxicosis reaching chronicity, which lead to nonspecific effects associated with poor performance. Macroscopic lesions after necropsy are not always obvious, but an increase in the volume of the liver, lymph node hemorrhage and erosions in the stomach and intestines can be observed (Ueno, 1983).

The aim of this study was performed a literature review through data collection of the various effects caused by mycotoxins in swine showing the importance that these effects have on animal production, in relation to damage, symptoms, prevention methods and treatment.

**Material and methods**

Several articles were consulted to carry out the development of this work, using as basis, experiments performed with the most important mycotoxins in swine.

In an experiment conducted by Hauschild 2006 to evaluate the digestibility of diets and metabolic balance of swine fed diets containing 800ug kg⁻¹ of aflatoxins, eight swine were used, half-brothers, with initial average weight of 13 kg, housed in metabolic cages, in environment semi-acclimatized. The experimental design was completely randomized with two treatments (control diet and control + 800ug kg⁻¹ of aflatoxins) and four replications, being the animal the experimental unit. The digestibility coefficients of dry matter, crude protein and gross energy were not affected (P>0.05) by addition of 800ug kg⁻¹ of aflatoxins in the diet. The metabolism of gross energy was 6% higher (P<0.05) compared to the control diet. Urinary excretion of N increased (P<0.05) in 52% and the relative retention to the absorption decreased (P<0.05) in 31% in animals fed diets containing aflatoxins. In the energy balance, the gross energy intake was not affected (P>0.05) by the addition of aflatoxins. Urinary excretion of energy increased (P<0.05) 52% in animals fed diets containing aflatoxins. The presence of 800ug kg⁻¹ of aflatoxins in the diet did not affect the digestibility, but altered protein and energy metabolism of piglets.

In the face of an outbreak of aflatoxicosis occurred in fall of 2004 in a pig farm in the town of Sentinela do Sul, RS, Zlotowski 2004, conducted an experiment to evaluate the infection that caused this outbreak. The corn used in feeding of the animals and that caused the intoxication, was produced and processed on the farm. 7 sows and 8 piglets died, and were reported two cases of abortion. The clinical signs were apathy, anorexia, jaundice, yellow urine with blood and photosensitization. The gamma-glutamyl transferase (GGT) and total bilirubin were elevated in necropsy of the animals. The main necropsy findings included generalized jaundice, orange yellow liver, edema of the gallbladder wall and the presence of yellow fluid in the abdominal cavity and pericardial. The most important microscopic lesions were found in the liver and included tumefaction, degeneration and necrosis individual of hepatocytes, bile duct proliferation and cholestasis. The diagnosis was based on clinical signs, the findings of necropsy and histopathology and high levels of aflatoxin B1 (3140ppb in corn, 4670ppb in ration) found.

For analysis of infection rates affected by zearalenone, Hauschild et al 2007, conducted an experiment to evaluate the digestibility of diets and metabolic balance of swine fed diets containing zearalenone (ZEA) with and without addition of organoalumino-silicate (OA). 12 sows were used with an initial weight of 12 kg, housed in metabolic cages. The design was completely randomized with three treatments (control, control + 2 ppm of ZEA and control + 2 ppm of ZEA with addition of 0.3% of OA in the diet) and four replications, with the animal as experimental unit. The ZEA and the OA did not influence (p>0.05) the dry matter intake, digestibility of dry matter and gross energy, energy metabolism, digestible protein and digestible and metabolizable energy of the diets. The ZEA and OA did not alter the balance of N (p>0.05), but modified (p<0.05) the fecal excretion of P. In diets containing ZEA and ZEA + OA, the fecal excretion of P was 15 and 10% lower than in the control group. The ZEA and the OA did not change (p>0.05) absorption of P in function of the ingestion. The consumption of 2 ppm of ZEA with or without the addition of 0.3% of OA does not
affect the digestibility of diets and metabolism of swine.

Another experiment consulted, where zearalenone was added in the diet of swine, it was carried out by Andretta et al., 2008, in order to evaluate the performance, the weight of some organs and vulvar morphology of prepubertal gilts, fed for 28 days with diets containing zearalenone. The experimental design was completely randomized with two treatments, control diet (CD) and control diet + 2 mg kg\(^{-1}\) of zearalenone (DZ), and six replicates each. There was no difference (P>0.05) among treatments for average daily feed intake (1.24 x 1.19 kg), average daily weight gain (0.68 x 0.71 kg), feed conversion (1.86 x 1.71) and body weight (BW) (30.9 x 30.4 kg). Zearalenone did not change (P>0.05) the absolute and relative weights of the heart (137 x 141g and 0.45 x 0.45% BW), liver (699 and 699g x 2.31 % x 2.26% BW), kidneys (47 x 49g and 0.15 x 0.16% BW) and spleen (166 x 171g and 0.55 x 0.55% BW). There was an increase (P<0.05) in length (17 x 27cm) and weight (23 x 157g and 0.07 x 0.51% BW) of the reproductive tract of gilts in the group DZ. The volume vulvar at the end of the period was 820% higher (P<0.05) in animals fed with zearalenone (941 x 8658mm/kg BW). The results indicate that in swine, the zearalenone and its metabolites have estrogenic activity, but not interfere in the performance of animals.

In order to evaluate the effect of fumonisins in swine, an experiment carried out by Dilkin et al., 2004 aimed to study the effects of prolonged oral exposure (28 days) in young swine to fumonisin. The animals were divided into three experimental groups and fed with rations ad libitum containing: A) 0mg of FB1.kg\(^{-1}\) (control); B) 10mg of FB1.kg\(^{-1}\); C) 30mg of FB1.kg\(^{-1}\). The swine were observed twice per day, their weights and feed intake were recorded weekly. Between the 20\(^{th}\) and 24\(^{th}\) days of intoxication, swine of the C treatment had decreased feed intake and weight gain, characteristic signs of pulmonary edema, and death of an animal at 23 days of intoxication. After 28 days, the animals were necropsied and the organs subjected to macroscopic analysis, histopathological and at the study of intestinal morphology and histology. In swine that slowed clinical signs of intoxication, it was observed pulmonary lesions, hepatics and decreased villus height and crypt depth and glandular hyperplasia in intestinal segments. It is concluded that substantial productivity losses can be induced in swine fed with ration contaminated with low levels of fumonisins.

Lovatto et al., 2007, performed an experiment to evaluate the effect of suction vice in piglets fed diet with or without fumonisins on the performance and characteristics of some organs. 32 piglets were used, paternal half-brothers, distributed in a factorial 2 x 2 (animals with vice and without suction vice, with or without addition of fumonisins in the diet), with four replications and two animals per experimental unit. There was no interaction (P>0.05) of the suction vice with the addition of dietary fumonisins on the variables studied. The final weight of piglets with suction vice was 8% lower (P <0.05) compared to the control group (25.2 x 27.5 kg). The addition of fumonisins in the diet reduced (P<0.05) in 9% the final weight of animals compared to the control group (25.8 x 28.3 kg). The suction vice not affected (P>0.05) the feed intake. The addition of fumonisins in the diet reduced (P<0.05) feed intake at 20% of the 22 to 28 days of experiment. The total weight gain was 14% lower (P<0.05) in piglets with suction vice (0.51 x 0.59 kg). The weight gain of animals fed diet with fumonisins was 15% lower (P<0.05) at the gain of the control animals. The suction vice decreased (P<0.05) the feed conversion (1.52 x 1.68) in 11%. The addition of fumonisins in the diet decreased (P<0.05) the feed conversion in 13% for piglets of the 15 to 21 days of experiment. The suction vice did not change (P>0.05) the weight of the organs of piglets. Fumonisins increased (P<0.05) liver weights (820 x 693g) and reduced heart weight (126 x 148), stomach (291 x 384g), intestine (2,015 x 2.577g), pancreas (55 x 74g) and lung (291 x 350 g). The suction vice and fumonisins adversely affect the animal performance, but the vice did not change the mass of organs.

**Diagnosis**

A presumptive diagnosis of mycotoxicosis is based on observation of clinical signs of intoxicated animals and through analysis of environmental data relating to the collection and storage of cereals used in swine feed. Usually, the story of the introduction of a new batch of food, sometimes with
macroscopic characteristics altered, is associated with the condition of intoxication. However, the definitive diagnosis is performed through the analysis of the presence of mycotoxins in food of the animals intoxicated. The techniques most used are analyzed by ELISA kits, Thin Layer Chromatography (TLC) and High Performance Liquid Chromatography (HPLC) (Dilkin et al., 2001).

**Treatment**

The treatment of mycotoxicosis presents a major challenge in the veterinary clinic. Removal of contaminated food is the first measure to be adopted. Support treatment appears to improve slightly the prognosis. The addition of higher levels of sulfur amino acids in foods is adopted by clinicians, but the efficacy needs further scientific studies.

Prophylactic measures consist in adopting cultivation and management techniques that prevent fungal growth, as the grain harvest immediately after physiological maturity, leaving the grain less exposed to the weather, drying and storage in appropriate storage for each type of cereal or a byproduct. The monitoring of cereals and by-products through appropriate sampling techniques and analysis mycotoxicological before of the utilization are also good practise, especially when the cereals were exposed to environmental conditions favorable to the development of fungi. The use of organic acid can help in preservation of food when in risky situations. The use of adsorbents natural or modified by the addition of enzyme or biological compounds in food need further researches scientifics, but in field situations, some have shown promising results (OMS 1983).

**Prevention**

Fungi cannot grow, or mycotoxins to be produced, in properly dried foods. For this reason efficient drying of the product and without moisture conservation is an effective measure against fungal growth and mycotoxins production. To reduce or prevent the production of most of the mycotoxins, the drying process should be done soon after harvest and as soon as possible. The critical quantity of water for safe storage corresponds to the water activity (Aw) of about 0.7. The maintenance of food below 0.7 Aw is an effective technique used worldwide to control damage caused by fungi and mycotoxins production in foods.

Problems in maintaining of an adequately low Aw often occur in the tropics, where high ambient humidity difficult to control of the humidity of the product. Where the grain is stored in bags, methods that use careful system of drying and subsequent storage can resolve these problems.

The correct way of drying is the best manner to avoid fungal growth and mycotoxins production in grain after harvest. Sometimes, when the drying at sun is not possible, it is necessary use some form of mechanical drying, and in the market, there are dryers with accessible price. It is possible the detection of fungi and aflatoxins in seeds, monitored by sampling and biochemical tests. These tests are used to establish the acceptance limits of acceptability of products for importation/exportation.

For aflatoxin, for example, the international limit is 4 to 50 micrograms/kg (or parts per billion - ppb). For the reliability of results, it is important to collect representative samples of the seed lot analyzed by techniques predetermined, which offers some difficulties.

**Conclusions**

The contamination of feeds and rations by mycotoxins is a serious health problem, causing in the animals, significant losses in production beyond suffering caused by injuries.

In Brazil, the recommendation for levels of aflatoxins in foods used for animal consumption is of 50 mg/kg, according to the Ordinance MA/SNAD/SFA nº 183, Ministry of Agriculture (Federal Official Gazette of 09/11/1988). But Brazilian law contemplate only the aflatoxins, further research should be conducted with other important mycotoxins such as citrinin, fumonisins, ochratoxin A, patulin, trichothecenes and other less frequent.

The recognition of the problems caused by mycotoxins in foods and rations require a first step towards the implementation of programs for the prevention and reduction of the problem; should be adopted the use of methods for removal or decontamination; a routine inspection, carry out
agricultural practices that prevent contamination and development of fungi to ensure good quality of the food for the animals.

References


