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MYCOTOXINS AND THEIR IMPACT ON POULTRY PRODUCTION

ABSTRACT: Only two years after the great expansion of “AGROŽIV” company, it was evident that mycotoxins have great impact on all segments of poultry production. During that year we were for the first time faced up with problems in poultry fattening. It was not possible to explain the present problems only by bacterial and viral infections, so we assumed that there is another reason for the observed clinical picture. From that time we started to pay more attention on contamination of poultry feed with mycotoxins. In the four years’ period, from 1988 to 2002, 57 samples were analyzed for the presence of mycotoxins. Mycotoxicolological investigations revealed the presence of T-2 toxin in 19 samples at concentrations less than 0.3 mg/kg, in 18 samples at concentration of 0.5 mg/kg, and in 3 samples 1.0 mg/kg. Beside this, type A trichotecene DAS was found in 6 of tested samples, and ochratoxin A and in 1 sample. Clinical picture and damages varied depending on mycotoxins’ concentrations and poultry age.

To exceed this problem in animal production we tried to use the organic and anorganic mycotoxin adsorbers as aditives of poultry feed, but the results were not satisfactory enough. So, we resumed that if we really want to resolve problem of mycotoxins we have to start from the field production of poultry feed components.

KEY WORDS: DAS, ochratoxin-A, poultry fodder, T-2 toxin

INTRODUCTION

Integral fodder mixtures used for poultry nutrition have high percentage of vegetable originated components which can be contaminated by mycotoxins, the influence of which can be more or less detrimental to animal or human organism. By this time, more than 1,100 different fungi species are known, of which more than 220 have the ability to generate over 330 different mycotoxins. Mycotoxins are respectively simple, low molecular, thermostable,
aliphatic and cyclical compounds with several carbon atoms. Depending on their chemical structure, mycotoxins taken into an organism may cause systematic effects and result in degeneration of certain organs or organic systems. Most of the mycotoxins have immunosuppressive effects due to their toxic influence over immune system, on one hand, and decreased and disordered protein synthesis on the other. Mycotoxins having been ingested enter the intestine and are discharged via faeces, often chemically modified. Mycotoxins absorption proceeds in small intestine. Certain part of absorbed mycotoxins reenters intestine along with bile, and only the remaining, usually smaller amount, enters systematic circulation, reaches predilectional organs and is consequently discharged via egg or urine after partial metabolism (G de k, Brigite,' 1980; L e i b e t s e d e r, J., 1981). Problem of mycotoxins has been actualized for the last twenty years due to factors created as a consequents of farming policy changes (H a l a m a, A. K., 1982), due to food production technology changes, intensified poultry production, high productive hybrid usage, both in agricultural and poultry production (Br a u e r, J., 1982). In further exposition factors that actualized mycotoxins’ problems, especially in poultry production, will be outlined.

Crop production. Not obeying the basic agrotechnical standards (crop rotation, inadequate mechanical land cultivation, introduction of heavy equipment in the process of land cultivation). Persisting on those cereal sorts that have high yield, and sensitive grain highly perishable while still in the ground (especially perishable are its embryonic parts-germs), leads to frequent appearance of the grains with undamaged endosperm, but with severely rotten germ. The damage of the shell and cereals’ grains by means of mechanical devices is highly present with those sorts with sensitive grain. Mechanical damage can occur mainly when harvesters or pickers are used, particularly if the rollers are not properly set. The same mechanical damage is caused by cereals’ elevation during the process of artificial drying. A grain can be damaged by insects, as well as with the work of unfavorable environmental factors. Usage of nitrogen fertilizers in large amounts, influences Fusarium to generate toxins (W o l f, Hanna, 1987).

Crops storage. Mechanical damage of grains can occur during the act of grain unloading into the elevators, particularly during the elevation into the upper strata of grain elevator. If the grain elevators are not constructed properly and if the food within is mouldy, big, heavy clots, sometimes heavier than 100kg, are created. At the same time, rise in temperature of the mixture can occur, due to the process of fermentation, pertinent to the mould growth and toxin creation. This moment is of vital importance, because when the fungi appear in normal conditions (meaning ordinary number per cm²) they use primary metabolism. It has been proved that metabolites created during the primary metabolism of some fungi species even lower the toxic rate of certain mycotoxins. The cause of such situation is still unknown. When a fungi population reaches the certain magnitude per cm², that is when it has become thick enough, secondary metabolism is activated and mycotoxins synthesis begins (K r a l j, M. et al., 1988). It appears that moulds fight for nutritive substratum
in such way; in other words, it seems as if mycotoxin production were one of the biological struggle for survival.

**Fodder production.** Construction of fodder factories with large production capacities and stock space of their own, contributed to the process of poultry production intensification. However, the risk increased that, entering the process of contaminated raw material production, the problem caused by consumption of food with high mycotoxin concentration, may appear in larger amount and in greater area.

**Fodder transportation.** Constantly being on the move and being emptied daily, tank trucks used for food transportation do not represent such a frequent way of food contamination with mould, that is with mycotoxins. Nevertheless, it may happen, if a food cargo transported is mouldy and some of it remains in the tank or in discharge hose, that it will consequently contaminate healthy food. That is why it is obligatory to inspect through the cell openings, after each discharge, if there is some food leftovers stuck to the cell walls. It is understandable that hoses must explicitly be cleaned for any food remains. I, personally, find it necessary to disinfect tanks once a month by some of the chlorine preparations.

**OCHRATOXICOSES**

Ochratoxin-A is a derivate of dihydroisocumarine. Besides ochratoxin-A ochratoxines B and C are isolated. These mycotoxins were discovered in 1965 in South Africa as a result of toxicological research of a larger number of *Aspergillus ochraceus*. Later researches proved that *Penicillium verrucosum* is also a significant producer of ochratoxin and that it can be produced by some other *Aspergillus* and *Penicillum* genus. *Aspergillus* species produce ochratoxin only if humidity and temperature are high enough, while certain *Penicillium* species can produce it even at lower temperatures (even at +5°C) (Grávits, R., Salvi, G., 1988).

Consumption of ochratoxin-A rich food can cause severe dehydration with chicken, decreased growth, anorexia, deficient conversion, diarrhea, parchedness, while with laying hen further symptoms appear: lower egg production, larger number of stained eggs with thin, rubber shell and decrease in laying eggs. Higher percentage of dying embryo has been noted. The section shows hemorrhage in the glandular area of the stomach, severe renal uricosis with white urate deposits all over the body cavity and inner organs (visceral uricosis). Ochratoxin-A has cancerous, genotoxic, immunosuppressive and immunotoxic features and affects protein synthesis, and it can cause blood vessels damage along with exudative diathesis. It has been proved that it adversely affects the amount of passively acquired SN antibodies for IB virus (El-Karim, S. A. et al., 1991). Ochratoxin-A can diminish amount of gamma globulin even to 38%, as a result of immune system damages, which explicates higher frequency of aerosacculitis and lower response to vaccination (Mazija, H. et al., 1991). Ochratoxin causes decreased bone mineralization. Value of ochratoxin-A in human medicine is significantly less studied, though
there is a widely accepted hypothesis about the role of this mycotoxin for etiology of Balkan endemic nephropathy with people from Serbia, Romania and Bulgaria, as well as the chronic intestinal nephropathy with people from Tunisia (Kralj, M. et al., 1988). It is vital to mention that another mycotoxin belongs to the group of nephrotoxins and is called citrinin, which is often found in contaminated food along with ochratoxin-A.

TRICHOTECENOTOXICOSES

Trichotecenes are products of secondary metabolism of fungi from Fusarium, Trichotecium, Cephalosporium, Myrothecium and Stachybotrys genus. More than 150 mycotoxins, belonging to this group, are known today. Fusarium species belong to the group of so called 'soil moulds' the reason being that they find the arable land very propitious for their survival and growth (Gravits, R., Salyi, G., 1998).

With trichotecenic mycotoxicoses further symptoms can be observed: the loss of weight, vomiting, tachycardia, hemorrhage, edemas, skin necrosis, especially around the mouth and in it, necrosis of the tip of the tongue, hemorrhages in epithelium of the stomach lining, in small intestine, infarct in parenchymal organs, damages of hematopoietic organs, hemorrhage within meninges and, consequently, neuro disorders ( Muller, Th. et al., 1987).

Trichotecenes inhibit DNA and protein synthesis and because of that have a strong immunosuppressive effect. Besides, they can cause cell damages particularly of the actively dividing ones (thymus, lymph glands, testis, intestine, milk). Trichotecenes cause acute hepatic dystrophy with hemorrhages. T-2 toxin is strong irritant and causes necroses of proventriculus’ mucosa, of the muscular part of the stomach, and of epithelium feather follicle. Smaller amounts of trichotecene, result, along with the loss of weight, in feathering disorder and flock unhomogeneousness. DON taken clean and in large amounts affects neither food consumption nor broiler growth, that is it affects neither egg production nor their quality when laying hen are concerned. On the other hand, according to some authors, DON produces the process of yolk retraction to run late, which precipitates decreased vitality of chickens. Some authors have published that DON concentration of 0.50 mg/kg effected wet extremities (legs) with laying hens of heavy line (Gravits, R., Salyi, G., 1998).

Humans and animals can get poisoned via ingestion of contaminated food, via skin or inhalation. When a chicken takes a larger amount of T-2 toxin, it reaches the highest concentration in liver in 2.5—3 hours, but already in 4—5 hours it is out of the organ.

In 2000, broiler poisoning was registered in Hungary. A small flock of broilers was in question where 78 out of 300 specimen died, i.e. 26%. Clinical picture said the following: bristle feather, apathy, mild diarrhea, subcutaneous edemas and heavy breathing. The section outlined muscular degeneration, necroses in oral cavity and esophagus, lymph tissue (Bursae fabricii and Thymus) atrophy, as well as heavy kidneys nephrosis. At the first stages of the disease, the cause was unfamiliar. The food having been changed, clinical picture beca-
me stable respectively and later fodder analyses demonstrated that the food contained T-2 toxin with 2.5 mg/kg concentration (Biataj, Z. et al., 1981).

During the same year broilers in Scotland had the same clinical picture of T-2 toxicosis characterized by loud piping, strong vexation, running to and from, pecking the walls. Further on, certain number of chickens started to form groups, while the rest of them stood individually being pecked by other chickens, especially being pecked over their feet that had dry necroses and relinquished a finger, respectively. Their liver was enlarged with augmented gallbladder. Decreased feathering was noted, as well as thickening of cartilaginous discs in limbs. With those chickens that grouped themselves quaver, ataxia, uncoordinated movements, loss of getting up reflex and, consequently, exitus were observed (Robb, J. et al., 1982). Stachybotryotoxin is a product of the secondary metabolism of Stachybotrys alternans mould. This mould lives as saprophyte on necrotic parts of plants, which are rich in cellulose. The most important of these toxins is satratoxin-N which is five times more toxic than T-2. Strachybotryotoxicosis is noted in almost all animals, as well as in people (in 1944 described as alimentary aleuky) (Kralj, M. et al., 1988). Acute course is followed by depression, ailment, loss of weight, ataxia, and abrupt death. Chronic course is characterized by necrosis and appearance of pseudomembranes all over oral lining and tongue, and over digestive tract, respectively. A case of strachybotryotoxicosis has been described in Hungary, within broilers aged 3 weeks when in a flock counting 15,000 pieces 0.6% of them died (Grávits, R., Salyi, G., 1998). The skin of the scalp, crest and neck, as well as of the legs was erythematic, infiltrated with serum, while surface necroses with necrotic areas of variable areas respectively appeared. Hemorrhages and diphtheric layers with necroses occurred even on the lining of the beak and muscular part of the stomach.

MATHERIALS AND WORKING METHODS

Only two years after the great expansion of “AGROŽIV” company, it was evident that mycotoxins have the great impact on all segments of poultry production. During that year, we were for the first time faced up with problems in poultry fattening. It was not possible to explain the present problems only by bacterial and viral infections, so we assumed that there is another reason for the observed clinical picture. From that time we started to pay more attention on contamination of poultry feed with mycotoxins. In the four years’ period, from 1988 to 2002, 57 samples were analyzed for the presence of mycotoxins. Mycotoxicological investigations revealed the presence of T-2 toxin in 19 samples in concentrations less than 0.3 mg/kg, in 18 samples in concentration of 0.5 mg/kg, and in 3 samples 1.0 mg/kg. Beside this, type A trichothecene DAS was found in 6 of tested samples, and ochratoxin A and in 1 sample. Clinical picture and damages varied in dependence on mycotoxins’ concentrations and poultry age.
RESULTS AND DISCUSSION

*T-2* toxicosis

For broiler production mycotoxins from the group of trichotecenes were the most interesting, first of all *T-2* toxin and DAS. Clinical picture and damages varied depending on mycotoxins’ concentrations and poultry age. Since the most of the specimen sent to be analyzed referred to starter, it was evident that one-day old chickens and starter chickens were the most sensitive.

**Fourteen-day old chickens and 0.5 mg/kg *T-2* toxin concentrations.** Five-day old chickens, that is six-day old chickens clinically seem normal. The only fact that can be observed is that till the third day of their life chickens lose interest in food, start to rummage through the food and pick by beak those components of food that suit them (note: the size of food particles is appropriate to technology). After this unapparent period, the first clinical signs appear—chickens form groups around hand feeders and under the heaters. Thinking that they were cold, we tried to raise the temperature in the room for 1—2°C, but the results were not satisfying enough. Soon after that mild diarrhea appears. About 5% of chickens suffers from neuro disorders, such as the loss of rising reflex. Morbidity is present in 20% and mortality in 1—3%. Average body weight of the chickens during the test measurement is by, approximately, 40% lower than the technological one (90 g). With the chickens that died during the peracute period pathologic-anatomic test is very often negative. Catarrh enteritis can be observed sometimes. During the subacute and chronic period, with those chickens that have lost their rising reflex and the ability to walk, section displays kidney changes shaped as one strong nephrosis, which can be manifested via renal or visceral uricosis. Hemorrhages in glandular area of stomach along with necrosis of cuticula of the muscular part of stomach can occur as a consequence of thirst.

**Fourteen-day old chicken and 1 mg/kg *T-2* toxin concentrations.** Two-day or three-day old chickens appear normal. However, already after 24 hours lower food consumption is observed. After this unapparent period very intensive chicken grouping occurs, which cannot be explained by the low temperatures in the room. Risen temperature had no effect. Consequently, profound diarrhea is noted. During 24 h the spreading becomes so wet on those places where the grouped chickens were located that damp reaches the conceit. The chickens seem as if they were scald. Down covering neck, scalp and orbital arches thins. Around 30% of the chickens gain neuro disorders such as loss of the rising effect. Certain specimens even lose pecking reflex. Other neuro disorders like one-sided, seldom both-sided, paresis and paresis of wings and legs appear. Morbidity is 60%, approximately, and mortality 3—6%. Average body weight of the chickens, during the test measurement that was undertaken for seven days, was by approximately 60% lower than the technological one (around 70 g).

The pathologic-anatomic results are similar to those when the concentration of *T-2* toxin was lower than 0.5 mg/kg; only the changes were more obvious. Subcapsular hematomas in liver, fatty liver dystrophy, dilatation of gal-
bladder and hemorrhage on kidneys are evident (peracute period). During the subacute and chronic period, kidneys demonstrate mild changes indicating fatty dystrophy, kidneys are hypertrophic, filled with urate, and if the process lasts longer severe renal or visceral uricosis occurs. Hemorrhages on Burse Fabricii plica and milt atrophy are noted.

**Fourteen till fifty-six-day old chicken and 0.25—1 mg/kg T-2 toxin concentrations.** Clinically, with chickens seven to fourteen days old weaker food consumption can be observed, weaker feathering, along with chicken disintegration according to average egg weight, which is some 10—20% below the technological one. The section outlines catarrh enteritis, hemorrhage at the juncture of esophagus and glandular stomach area. Sporadically, erosions are traced in muscular stomach area. Liver is still fatty dystrophic and hypertrophic with pale stripes originated as a consequence of ischemia and rib pressure over the area. Subcapsular hematomas are observable. Gallbladder is dilated and enlarged 2—3 times. Necroses of the tip of the tongue are present with older poultry that consummated T-2 toxin rich food for a longer period of time. This necroses is sometimes so strong that the tip of the tongue becomes cleft (snake’s tongue). Atrophy of thymus, milt, and B. Fabricii is noted. Those chickens that had consummated contaminated food were more prone to get fibrous aerosacculitis compared to those that consummated trichotecene free food. It is noted that T-2 toxin and DAS have cumulative impact. After serological blood research on the virus of New Castle disease, via method of hemagglutination inhibition, bad immune response to vaccination is recorded. However, we have observed that the immunity of those chickens from the rooms where the food was previously healthy and their immune system good, did not deteriorate even when the chickens consumed trichotecene rich food. This leads us to conclude that trichotecenes do not affect already created immunity.

**OCHRATOXICOSES**

So far we had the opportunity to detect ochratoxin at the lower boarder of allowed concentration in one food sample. The chickens were eleven days old and the first clinical sign that something was arising was larger water consumption and appearance of whitish diarrhea, that is the presence of watery substance, admixed with urates over the spreading. Thinking that the reason for that was increased food salinity, we sent the sample urgently to be tested. The amount of traced NaCl was even lower than the recommended one for that chicken category. It was only then, when we demanded mycotoxic analyses to be done. The food was removed from the premises for 24 h, and was mixed with other food stocks until laboratory results appeared. In the food sample ochratoxin was traced. Its concentration was 0.25 mg/kg. Direct damages from consumption of such food were not enormous. During that year epizooologic situation was fairly unfavorable since we had the IB virus present. The chickens were vaccinated against IB on the first and seventeenth day of their life. On the premises where ochratoxin rich food was consumed, clinical manifestation of the illness appeared.
CONCLUSIONS

Based on previously stated facts, further conclusions can be drawn concerning mycotoxicoses:

1. Mycotoxicoses have been known as causes of certain illnesses, or, perhaps it would be better to say, symptoms of certain illnesses, for more than a few hundred years.

2. Diagnosis of mycotoxicoses is very complex and includes thorough anamnesis and unspecific clinical picture that characterizes all mycotoxicoses. Evident in development disorders, lower production capacities with animals, and only in those cases of timely long consumption of food with extremely high mycotoxin concentration, exitus can occur. Uncontagiousness of mycotoxicoses, abrupt betterment in health of the ill animals and return of production to technological norms based solely on food change, without treatment, are safe data for final diagnosis establishment.

3. The most frequent mycotoxins carriers are crops, especially harvest remnants, oil industry waste and warehouse food remains.

4. Mycotoxicoses, when occurring in large systems such as 'Agroživ' company, can cause major problems both by means of increased mortality and by decreased production results. Qualified personnel that lead this production sector had realized that beforehand, and there have been several years since we have started fighting this battle. We opted for the situation where mycotoxins are fodder. This is achieved via appropriate agro technical measurements applied in the fields, and certain precautions are undertaken even in the fodder factories and farms. A thorough analysis of the row materials of foreign origin is done. Quality of our raw materials is more or less known to us, so that we can decide by ourselves what to give to which animal species and category. This way of work resulted in betterment of production results that can be equally measured with the world.

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МИКОТОКСИНИ И ЊИХОВ УТИЦАЈ НА ЖИВИНАРСКУ ПРОИЗВОДЊУ

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Резиме

У компанији „Агрожив” Панчево смо две године након експанзије компаније — 1998. год. схватили значај који микотоксини могу имати у свим сегментима наше производње: почев од одграја родаитеља тешке линије, преко њихове експлоатације па до финалне бројдерске производње. Те године смо се први пут сусрели са проблемима пилића у току чију етиологију нисмо могли да објаснимо само бактеријским и вирусним инфекцијама, него смо претпостављали да постоји још неки чинилац који је искомплексовао клиничку слику. Тада је послат и први узорак хране ради испитивања на садржај микотоксинов. У периоду 1998—2002. год. послато је 57 узорака хране ради испитивања на садржај микотоксинова. У 19 узорака је утврђен Т-2 токсин у концентрацији < 0,3 mg/kg. У 18 узорака је нађена концентрација Т-2 токсина од 0,5 mg/kg, а у 3 узорака је нађена концентрација од 1 mg/kg. У 6 узорака хране је осим Т-2 токсина био присутан и ДАС. У једном узорку хране био је присутан и охратоксин. Клиничка слика и штете су варирали у зависности од концентрације микотоксинова и старости пилића. У борбу против микотоксинов смо кренули са додавањем различитих органских и неорганских адсорбентата, али смо тиме успели само да делимично решимо проблем. Схватили смо да борбу против микотоксинова морамо да поведемо на широком фронту, у свим сегментима производње, а да је морамо почети у ратарској производњи — на самој њиви.