A novel approach: deactivation of T-2 toxin by biotransformation

by Dr Arpad Bata, Dr Bata Ltd, Bajcsy-Zs ut 139, Ocsa H-2364, Hungary.

There is no farmer on earth who does not know the word mycotoxin. Mycotoxins have become an everyday risk in animal husbandry. According to the FAO (Food and Agriculture Organisation) 25% of the world's crop harvests are contaminated with mycotoxins.

Mycotoxins are highly toxic secondary metabolic products of moulds growing on the feed. They occur under natural conditions in feed as well as in food.

There are six major classes of mycotoxins that frequently occur: aflatoxins, trichothecenes, fumonisins, zearalenone and ochratoxin.

Mycotoxins differ in their chemical structure, which explains the great variation of symptoms. The main toxic effects are carcinogenicity, genotoxicity, nephrotoxicity, hepatotoxicity, oestrogenicity, reproductive disorders, or immunosuppressive effects. The main problem for farmers is the production safety that originates primarily in the lower or higher mycotoxin contamination of the feed.

Due to the immunosuppressive effects of mycotoxins efficacy of immunisation decreases or totally fails, gastrointestinal problems increase and became unmanageable in some cases.

Effects of mycotoxins

The contamination of feedstuffs with mycotoxins poses a serious threat to the health and productivity of animals. The effects of mycotoxicoses in animals are diverse; varying from immune suppression to death in severe cases, depend on the type of mycotoxin consumed, the species, sex, age, or breed of animal, on general health or the immune status and environmental factors.

T-2 toxin and trichothecenes can cause mouth and intestinal lesions as well as impair the birds' immune response, causing egg production declines, decreased feed consumption, weight loss, and altered feather patterns.

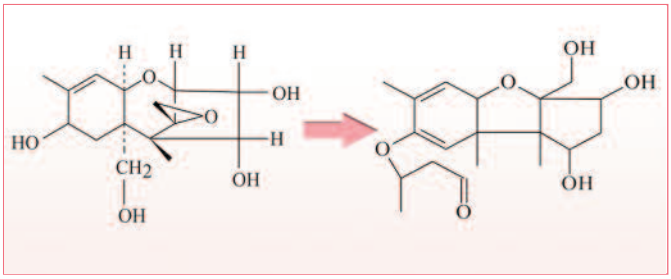
Aflatoxicosis is primarily a hepatic disease. The susceptibility of individual animals to aflatoxins varies considerably depending on species, age, sex, and nutrition. In fact, aflatoxins cause liver damage, decreased egg production, recurrent infection as a result of immunity suppression.

Grains infected with the fungus Fusarium spp. are the source of zearalenone, a mycotoxin with oestrogenic activity. Toxicity occurs chiefly in swine as reproductive failure. Chickens tolerate zearalenone better than swine but it has potential adverse effects in bird performance and egg yield.

Counteracting strategies

Detoxification procedures are divided into three categories: physical, chemical and biological methods. Use of adsorbent materials is a very common method employed to prevent mycotoxicoses. Substances scientifically investigated as potential mycotoxin-binding agents include bentonites, zeolites or activated charcoal.

Fig. 1. Detoxification of T-2 toxin.



Biological detoxification of mycotoxins by enzymes and/or micro-organisms comprises the degradation of mycotoxins within the gastrointestinal tract, before resorption into the animal occurs.

Until now, a respectable number of micro-organisms have been known to be able to counteract different mycotoxins.

Enzymatic degradation

Effectiveness of Detoxa Plus was investigated in animal trials. The trial groups were fed with T-2 toxin containing feed. The T-2 toxin is a non-polar, microcyclic type-A trichothecene. Because of its non-polar nature, the molecule binds little to any of the known mycotoxin adsorbents (for example hydrated sodium calcium aluminosilicates).

The toxicity of trichothecenes has long been attributed to the 12,13-epoxide ring, and for an almost equally long time, efforts have been made to find a method (enzymatic, microbial or both) for disintegrating this ring. Detoxa Plus is a product that is capable of disrupting the active part of the T-2 toxin (Fig. 1)

Animal trials

Day old ducks, as a rule, are extremely sensitive for the dermatotoxic effects of T-2 toxins which explains their use for bioassaying the presence of trichothecenes in contaminated feeds.

Further to this, T-2 toxin is notorious for its negative effects on a range of biological and production parameters including protein synthesis, adrenocortical activity, feed intake, growth rate, feed conversion rate, many compartments of the humoral and cellular immunity, etc.

Due to the paucity of relevant data it is customary to apply the advisory limit concentration established for domestic hens to ducks as well.

In this experiment day-old white Pekin ducks were randomly assorted into nine groups of 10 ducks.

One of the groups served as control and no mycotoxin was added to their feeds. The feeds of the experimental groups were completed with 0.2, 0.4, 0.6, 0.8, 1.0, 2.0, 3.0 and 4.0mg/kg (ppm) purified T-2 toxin, respectively.

Dermatotoxic oral lesions caused by T-2 toxin.

Dermatotoxic oral lesions developed in most experimental ducks within two days after start of feeding T-2 toxin contaminated feeds. The gradual disappearance of macroscopic signs indicated the development of some sort of tolerance in groups treated with lower T-2 toxin content. No heeling was found in the 3.0 and 4.0ppm groups.

Dietary concentrations of T-2 ≤0.4ppm had no effect on the average weekly weight gain in the first six weeks, but severe decrease was found in the last week of the experiment. The 0.6ppm dietary T-2 toxin had no effect on the weight gain in the first three weeks.

On week four and later the weekly weight gain was significantly reduced and the final live weight of this group was also significantly lower than that of the control. Dietary T-2 concentrations of 1.0ppm and above unanimously depressed the growth rate from week one to week seven.

Only the 3.0 and 4.0ppm groups showed feed refusal in the first week. From week three the feed intake of the 0.6-4.0ppm groups was most of the time lower than that of the controls indicating feed refusal.

Feed conversion rate of the

Continued on page 9

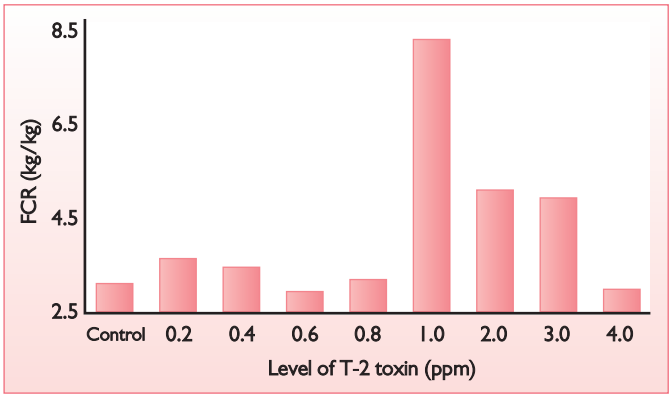


Fig. 2. Feed conversion rate of the ducks after 49 days of treatment.

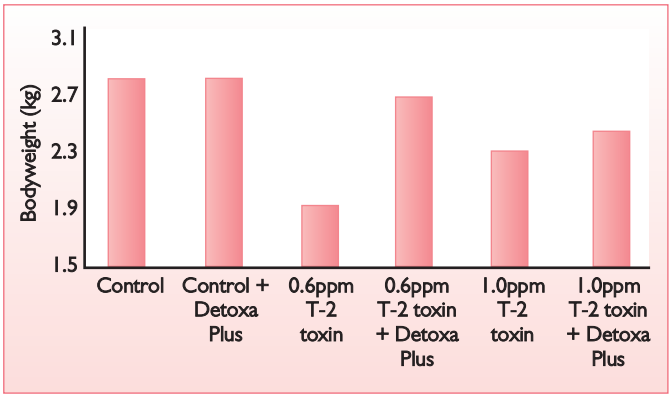


Fig. 3. Body weight of the ducks at slaughter (kg).

Continued from page 7

groups showed no dose dependent consistent changes with the weeks of the treatment. In the average of the 49 days of treatment the control ducks used 3.31kg feed for production of 1kg live weight. Average FCR of the 0.2-0.8ppm groups was similar to the control (Fig. 2).

Ducks that consumed feeds contaminated with 1.0 and 2.0ppm T-2 toxin had 8.31 and 5.06kg/kg FCR, respectively, indicating impaired growth rate.

The 4.98 and 3.26kg/kg average FCR in the 3.0 and 4.0ppm groups refers both to depressed feed intake and weight gain (Fig. 2).

Antigen or non-specific mitogen induced proliferation of peripheral blood lymphocytes is a good indicator of the initiation of immune response, which ultimately determines host resistance to disease.

The blastogenic response of lymphocytes to non-specific and specific mitogens was distinctly impaired by the T-2 toxin at all levels in the feed.

In the 3.0 and 4.0ppm groups the histological examination revealed lymphocyte depletion in the spleen and Bursa of Fabricius.

Data of the above investigation show that dietary T-2 toxin can affect the health, performance and an important element of cellular immune response of growing White Pekin ducks. Therefore implementation of counteracting measures is important.

Effects of two dietary levels (0.6 and 1.0ppm) of T-2 toxin, and the possible protective capacity of Detoxa Plus were investigated in growing White Pekin ducklings in a 49-day trial comprising six treatment groups of 10 ducks/group.

Experimental design

The experimental design consisted of a negative and a positive control and four test groups, as follows:

- Negative control group: no T-2 toxin and no feed additive added to the feed.
- Positive control group: no T-2 toxin added but the feed was supplemented with Detoxa Plus at 2kg/t level.
- Trial group 1: the feed was complemented with 0.6ppm of purified T-2 toxin.
- Trial group 2: the feed was complemented with 0.6ppm of purified T-2 toxin and 2kg/t of Detoxa Plus.
- Trial group 3: the feed was complemented with 1ppm of purified T-2 toxin.
- Trial group 4: the feed was complemented with 1ppm of purified T-2 toxin and 2kg/t Detoxa Plus.

The main production parameters were investigated during the trial. The daily growth rate of the control groups followed the pattern characteristic of the breed. The negative and positive control groups produced almost identical body weight at slaughter.

There was a significant difference in the body weight in the groups where the feed was complemented with 0.6 or 1.0ppm T-2 toxin. The final body weight was significantly lower in these groups compared to the control group.

In the trial group two, where the feed was complemented with 0.6ppm T-2 toxin and Detoxa Plus, the final body weight was as high as the control group.

The adverse effect of 0.6ppm T-2 toxin was fully counteracted by Detoxa Plus (Fig. 3). No such effect was observed with the higher dietary concentration of T-2 toxin. Although the treatment with the feed additive failed to close the gap between the control group and the trial group three.

Feed conversion ratio was calculated at the end of the trial. As the results show in Fig. 4, FCR was similar in the negative and the positive control group.

Mycotoxin addition resulted in the impairment of the feed conversion in the trial group 1 (0.6ppm T-2 toxin supplementation) and group three (1.0ppm T-2 toxin supplementation).

This value is impaired almost 40% in the case of 0.6ppm T-2 toxin addition that suggests an impaired metabolic process of the animals. Detoxa Plus supplementation resulted in an improvement in the feed conversion in the trial group 2 (0.6ppm T-2 toxin and Detoxa Plus complementation) and this ratio was close to the value measured in the control group (Fig. 4).

The adverse effects of T-2 toxin on the immunocompetence of birds have been known for many years. In our trial, the blastogenic response of lymphocytes to non-specific mitogens was distinctly impaired by T-2 toxin treatment. Both levels of dietary T-2 toxin caused depression in this important element of cellular immunity (Fig. 5).

As our results show, treatment with Detoxa Plus significantly ($P \leq 0.05$) alleviated the depressive effect of T-2 toxin on the blastogenic response at a dietary level 0.6ppm.

The higher T-2 concentration depressed this parameter of the cellular immunity, but Detoxa Plus failed to yield significant improvement.

Conclusion

In the present experiment, no significant differences were observed between the negative control and the positive control group (Detoxa Plus supplementation). Therefore it can be concluded that Detoxa Plus does not impair the health and production of broiler ducks.

Based on the results Detoxa Plus is able to counteract the adverse effect of a dietary concentration of T-2 toxin that might be encountered under feed conditions.

Based on the literature simultaneous occurrence of several mycotoxins should be expected in the feed mixtures. Therefore, Detoxa Plus is effective against the contamination of other trichothecene compounds like deoxynivalenol, ochratoxin A, fumonisin and zearalenone or co-occurrences of them. ■

Fig. 4. The feed conversion ratio of the animals during the trial (kg/kg).

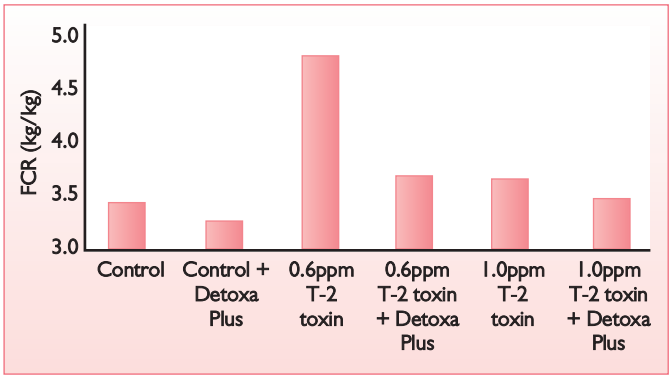


Fig. 5. Blastogenic response of lymphocytes to a specific mitogen concanvalin-A.