EFFECT OF LOW DOSES OF THE MYCOTOXIN FUMONISIN B₁ ON THE BODY MASS GAIN, FEED INTAKE AND FEED CONVERSION RATE OF PIGS

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ABSTRACT

The fumonisin group of mycotoxins produced by Fusarium moniliforme is a rather newly discovered contaminant of some agricultural products, especially maize-based food and animal feed. Fumonisins were discovered in 1988 of mouldy maize inspected since 1993 has shown FB₁ contamination, the degree of this contamination increasing from year to year. Problems of mycotoxins cause significant economic losses due to the reduced feed intake, body weight gain and feed efficiency. We have very few informations about the effect of FB₁ on these in South Africa, where high evidence was found between the occurrence of human oesophageal cancer and the rate of FB₁ contamination. The discovery of this toxin lead to the explanation of equine leukoencephalomalacia (ELEM), porcine pulmonary oedema (PPC) and possibly liver cancer in rats. In Hungary almost 70 % parameters – especially in farm animals. Three experiments were carried out with weaned piglets, in order to study the dose and time-dependent effect of FB₁. Fungal culture of Fusarium moniliforme was added to the diet so that the FB₁ exposure was: 0, 10, 20 and 40 ppm for 4 weeks, 0, 1, 5 and 10 ppm for 8 weeks and 0, 1, 5 and 10 ppm for 5 months. In none of the experiments and the periods examined had FB₁ any significant effect on feed consumption, body weight gain and feed conversion of weaned pigs. In spite of these findings mild or severe pulmonary edema caused by the toxin was found in the animals by dissection carried out at the end of the experiment. Our results draw the attention to human health concerns of FB₁. The toxin consumed by the animals without any clinical signs can cumulate in the animals and then enter the human organism by means of products of animal origin (meat, milk, etc.).

Keywords: mycotoxins, Fumonisin, pig, gain of body weight

INTRODUCTION

Mycotoxins get into the human organism with contaminated plant food and consumer goods such as bread, beer or coffee. In addition to this, foods of animal origin indirectly introduce into the food chain also the mycotoxins contained in feeds consumed by the animals. Prolonged, so-called chronic exposure to low doses of mycotoxins is especially dangerous, as is the additive multitoxic effect that occurs if several toxins are present simultaneously. Fusarium moniliforme is a fungus that occurs on maize all over the world. Under certain conditions it produces mycotoxins of veterinary and medical importance. The most important representative of these mycotoxins is fumonisin B₁, which causes equine leukoencephalomalacia and porcine pulmonary oedema, and has been associated with the development of oesophageal cancer in humans. The objective of these experiments was to study the effect of fumonisin B₁ exposure of different duration and dose level (low dose) on the body mass gain, feed consumption and feed conversion rate of pigs.

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MATERIAL AND METHODS

Experimental animals
Twenty weaned barrows of identical body mass (10 kg) and of the same genotype were used in the experiments. Before the start of the experiment the animals were assigned to four groups on the basis of their body mass. The piglets were housed individually. They were fed twice a day, and the quantity of feed not consumed by the animals was weighed and recorded. Drinking water was available ad libitum. During the experimental period the animals were weighed twice a week, then in the experiments of longer term (8 weeks and 5 months) weighing was performed once a week. The clinical status of the piglets was checked twice a day. At the end of the experiment the animals were killed and examined for gross lesions by necropsy.

Design of the experiments
Experiment 1: Four random groups of weaned piglets were fed a diet containing 0, 10, 20 or 40 mg/kg fumonisin B₁ (FB₁) for a relatively short period of time, for 4 weeks. The objective was to determine whether the effect of fumonisin B₁ doses much lower than those reported in the literature could be detected already after a short period of exposure.
Experiment 2: Four random groups of weaned piglets were fed a diet containing 0, 1, 5 or 10 mg/kg FB₁ for 8 weeks. Utilising the results of the first experiment, even lower doses were applied, but for a longer time. Experiment 3: Four random groups of weaned piglets were fed a diet containing 0, 1, 5 or 10 mg/kg FB₁ for 5 months. The objective of this experiment was to study the effect of treatment of substantially longer duration, which lasted until end of fattening.

Housing of the animals
The experimental animals were housed individually in battery cages. One cage between two neighbouring groups was left empty, to prevent cross-contamination between the animals. In this way we ascertained that the animals consumed only the toxin quantity designated for them. In the 5-month experiment, the animals were kept in batteries during the first 8 weeks and subsequently they were housed in individual boxes.

Feeding
The experimental animals were fed a basal ration corresponding to their age. In the 4-week and the 8-week experiment that ration contained 187 g/kg crude protein, 12.8 MJ/kg ME, and 13.1 g/kg lysine (LYS). In the 5-month trial, after the first 8 weeks the ration contained 137 g/kg crude protein, 12.9 MJ/kg ME, and 9.4 g/kg LYS. After a 5-day period of adaptation, a Fusarium moniliforme fungal culture of known fumonisin B₁ content was mixed to the ration so as to ensure a daily FB₁ intake of 0, 10, 20 and 40 mg/kg of feed (ppm) in Experiment 1 and 0, 1, 5 and 10 mg/kg of feed (ppm) in Experiments 2 and 3. The toxin was produced at the Veterinary Institute of Debrecen according to the method of Fazekas et al (1998). The feed did not contain other mycotoxins.

Statistical analysis
Comparison of the treated and control groups in terms of feed consumption, body mass gain and feed conversion rate (FCR) was done by one-factor analysis of variance using the ANOVA programme of the SPSS 7.5 programme package.

RESULTS AND DISCUSSION

Clinical signs
The animals did not show clinical signs attributable to toxin effect in any of the experiments. No difference was found between the treated and the control groups in the behaviour of the animals either.

Body mass gain, feed consumption and feed conversion rate
Comparing the three experiments, there was no significant difference in the starting average body mass values. During the study, no significant differences were found between the treated and the control groups in daily average body mass gain and feed consumption in any case. There was no difference in the feed conversion rate either (at least within the sensitivity limits of the method, which in the case of the feed conversion rate means approximately 6–8% body mass/feed consumption difference, assuming 5% error of first kind and 20% error of second kind). Comparison of the groups was always done by single-factor analysis of variance. The level of significance markedly exceeded 0.05 in all cases.

Gross pathological findings
In all three experiments, necropsy revealed gross lesions in the lungs which were indicative of the toxic effect of fumonisin B₁. Table 1 show the incidence of these lesions and the average mass of the lungs in the treated groups.

**Table 1.** The relative frequency indicate that the incidence of lung lesions depends not only on the amount of toxin ingested but also on the duration of toxin intake

<table>
<thead>
<tr>
<th>Experiments</th>
<th>Doses</th>
<th>Macrossopic alterations (number of lungs with pathological alterations / number of lungs in the groups)</th>
<th>Weight of the lungs (mean) (a,b,c: P&lt;0.05)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>10 ppm</td>
<td>3 / 4 (75 %)</td>
<td>168&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>1.</td>
<td>20 ppm</td>
<td>4 / 5 (80 %)</td>
<td>210&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>40 ppm</td>
<td>5 / 5 (100 %)</td>
<td>266&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>1 ppm</td>
<td></td>
<td>1 / 4 (25 %)</td>
<td>276&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>2.</td>
<td>5 ppm</td>
<td>2 / 5 (40 %)</td>
<td>294&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>10 ppm</td>
<td>3 / 4 (75 %)</td>
<td>367&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>1 ppm</td>
<td>2 / 5 (40%)</td>
<td>588&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>3.</td>
<td>5 ppm</td>
<td>3 / 4 (75 %)</td>
<td>660&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>10 ppm</td>
<td>6 / 6 (100 %)</td>
<td>769&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

**CONCLUSION**

Very few data are available in the literature regarding the effect of fumonisin B₁ on growth and body mass gain. Especially little is known about the effects exerted by this toxin in farm animals (including the pig) and in the case of long-term toxin exposure.

In an 8-week experiment on weaned piglets (6 weeks old, weighing 6–12 kg), Rotter et al. (1996) studied the effect of feeding a diet supplemented with purified FB₁. While in male animals the body mass gain achieved in weeks 4–6 decreased in a linear ratio to increasing concentrations of the toxin (1 and 10 ppm), in the gilts no difference could be detected. Body mass gain showed marked fluctuation in the first four weeks, then became more balanced in the second 4-week period. The feed conversion rate was impaired already by toxin levels as low as 0.1 ppm, though the decrease was slight and non-significant. No significant difference was detectable in the feed consumption either; still, it was found that at an FB₁ dose of 10 ppm the toxin-treated male piglets ate approximately 10% less than the control males.

The toxin doses fed in these experiments did not exert a detectable effect on the body mass gain, feed consumption and feed conversion rate of the animals. This finding somewhat differs from the results reported by Rotter et al. (1996). In their experiments, Rotter et al. fed a diet containing purified FB₁ mycotoxin to male and female piglets, while in these studies we added an FB₁-containing mycotoxin mixture to the diet of barrows. Rotter et al. could demonstrate a negative effect of FB₁ on the performance data only in boar piglets.

According to data of the literature, other fusariotoxins affect feed intake and body mass gain already in very low amounts. For example, T-2 toxin was found to cause substantial feed rejection already at a level as low as 3 mg/kg of feed (Rafai et al., 1995).

In the experimental animals used in this study, the toxin exerted its harmful effects; however, this was found out only at necropsy, as the animals did not show abnormal clinical signs during the experiment and their performance parameters did not change as an indication of toxicosis. This calls the attention to human health implications, as the mycotoxin may accumulate in animals unnoticed, and humans may ingest it with foods of animal origin. This poses an particularly high risk, as FB₁ belongs to the group of potentially carcinogenic substances.

The absence of clinical signs in the experimental animals of this study suggests that it may well happen also in humans that this toxicosis is diagnosed only at an already irreversible stage.

It is known that certain mycotoxins may interact with each other. Thus, when several mycotoxins are simultaneously present in the feed, they may mutually amplify the toxic effects of each other. In experiments conducted on piglets, Friend et al. (1992) proved that the negative effect exerted by T-2 toxin on body mass gain and feed intake was substantially enhanced by the presence of deoxynivalenol (DON). In the present experiment the piglets’ feed contained only the studied Fusarium moniliforme mycotoxin. Thus, these doses probably exert a different effect under natural conditions when the contaminated feed often contains several different mycotoxins.

The presence of mycotoxins is a constant problem. It seems that mycotoxins cannot be eliminated from the environment, and thus we must live together with the hazard they pose. Mycotoxicoses represent an important human
health problem in Hungary because the population consumes large quantities of cereals, which are the foods most likely to become contaminated with different mould species.

REFERENCES