ASPECTS CONCERNING THE EFFECT OF SOME ANTIOXIDANTS ON LIVER HISTOLOGICAL STRUCTURE IN WEANED PIGS, INTOXICATED WITH DEOXYNIVALENOL (DON)

ASPECTE PRIVIND EFECTUL UNOR ANTIOXIDANȚI ASUPRA STRUCTURII HISTOLOGICE A FICATULUI LA PURCEII ÎNCĂRCAȚI ȘI INTOXICAȚI CU DEOXYNIVALENOL (DON)

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Deoxynivalenol is a mycotoxin produced by fungi of the Fusarium genera, which are abundant in various cereal crops and processed grains. In order to protect cell structure within the tissues studied, we tried various experimental variants by incorporating some selenium and boron sources into the combined fodder, and also in mixture with DON with different concentrations. To determine the morpho-physiological changes induced by the mycotoxin DON upon liver histological structure, and also the antioxidants’ effects, we took samples from 9 piglets belonging to three groups: group 1 (fed with fodder added with DON), group 2 (fed with fodder added with selenium) and group 3 (fed with fodder added with selenium and DON). In the case of the individuals in groups 2 and 3 fed with fodder added with addition of selenium, respectively of DON and selenium, the microscopic images reveal the presence of uniform hepatic lobules, delimited between them through fine conjunctive septa. The intralobular capillary network is hypertrophic. The selenium added in the fodder for the individuals from group 3 has a hepatoprotective role, aspect suggested by dystrophic processes occurred on small areas, caused by the presence of DON and by the vascular network’s hypertrophy. In the case of this group, we observed a strong stimulation on the defense system, suggested by the presence of the leukocyte infiltrates within the interlobular conjunctive and by the vascular network’s hypertrophy.

Key words: Fusarium, deoxynivalenol, liver, histology modifications, pig, antioxidant, selenium

Introduction

Deoxynivalenol is a mycotoxin produced by fungi belonging to the genus Fusarium (Fusarium culmorum and Fusarium graminearum), abundantly met in various cereal crops (wheat, maize, barley, rye) and in products made of processed
cereals (malt, beer and bread). This toxin is very thermally stable and it does not degrade at high temperatures (Rotter, B.A. et al., 1996, Ehling, G. et al., 1997, Eriksen, G.S. and Alexander, J., 1998). Pigs are exposed to DON contamination due to their diet rich in cereal grains, because they are much sensible than poultry or ruminants to the action caused by this toxin (Rotter, B.A. et al., 1996). Moreover, piglets sensitivity to DON action increases during the weaning period, a critical period that includes the adaptation to a new environment (shelter and temperature), diet changes, association with other pigs, removal from their mother, alteration of their intestinal micro-environment (pH, microflora) and loss of mother milk (immune protection). At cell level, DON inhibit AND, ANR and protein synthesis. After the acute intoxication, some necroses appear in various tissues of the digestive tract, bone marrow, lymph tissue and kidney.

Selenium is known for its antioxidant effect, contributing dramatically to body protection against the action exerted by free radicals and to their removal from cell, saving in this way, for the moment, the cell from death. This chemical element intervenes upon the immune system by stimulating the immunoglobulin in production, and also in the evolution of the leukocyte cell line, assuring vitality and maturation for leukocytes, which mean optimal functioning. So, by stimulating the immune system, selenium reduces inflammations and fights against infections (Gueguen, L., Pointillart, A., 1995).

Materials and Methods

To determine the morpho-physiological changes induced by the mycotoxin DON upon liver histological structure, and also the effects induced by the antioxidant used, we took samples from 9 piglets from three groups: group 1 (administered with NC-02 + 1.04 ppm DON), group 2 (administered with NC-02 + 0.3 ppm Selplex) and group 3 (administered with NC – 02 + 1.04 ppm DON + 0.3 ppm Selplex).

Fragments taken were fixed in solution of ethylic alcohol 80° and in neuter formalin 10%, were submitted to dehydration, clearing, then they were soaked and inclusioned in histological paraffin and sectioned at the width of 5µ with the rotating microtome Leyca and colored for an optical differentiation between the tissue and cell structures. We applied two staining methods: HE and the Mallory trichromic staining method (Mureșan E. et al., 1976). The microscopic study was performed with an optical microscope Olympus with objectives of 10x and 40x and the field glass of 10x.

Results and Discussions

Group 1 - (fodder with addition of DON)

Within the interlobular areas, some conjunctive septa fibrosis and vascular ectasies occur (fig. 1). At the level of sinusoidal capillaries located between the hepatocyte cordons Remack, ectasies sometimes associated with local hemorrhagic
processes occur on some areas. Hepatocytes, sensibly reduced in volume, have granular cytoplasm with only a few, but big granulations (fig. 3).

Group 2 – (fodder with addition of selenium)

In the case of the individual 1 from group 2 – 1(165), microscopical images reveal the presence of uniform hepatic lobules (fig. 4), delimited between them by fine conjunctive septa. The interlobular areas include the bile channel, and also the hepatic venule with a very wide lumen (fig. 5). Each lobule is centered on the centrolobular vein, to which the hepatocyte cordons Remack converge, uniform. Within the Remack cordons’ structure, hepatocytes have a polygonal aspect, with the cytoplasm finely granulated and basophile and they have 1-2 nuclei. On small areas, towards the hepatic lobules periphery, hepatocytes have a clear cytoplasm, strongly vacuolated (fig. 7). Sinusoid capillaries with a wide lumen are present between the hepatocyte cordons, and on small ectasied territories they produce reduced hemorrhagic area (fig. 6).
In the case of the individuals 2 (169) and 3 (090) from the experimental group 2, the hepatic parenchyma morphology presents similarities with the one belonging to the individual 1, a characteristic being represented by the hypertrophy of the interportal venous system (fig. 8) and of the intralobular sinusoid capillaries (fig. 9).
Interportal Kiernan areas are reduced and include vessels with a reduced lumen, compared to the control variant. Hepatocytes have an acidophil cytoplasm, loaded with reduced granulations. Within the perilobular area, hepatocytes produce dystrophic processes observed on large areas (fig. 10). Sinusoid capillaries are slightly ectasies, with hemorrhagic area observed on reduced territories.

**Conclusions**

1. The sections performed in the liver of the individuals in group 1 reveal, in the interlobular areas, conjunctive septa fibrosis and vascular ectasies. At sinusoid capillaries level located between the hepatocyte cordons Remack, ectasies sometimes associated with local hemorrhagic processes occur on some areas. Hepatocytes, sensibly reduced in volume, have granular cytoplasm with only a few, but big granulations.
2. In the case of the individuals in group 2, within the Remack cords’ structure, hepatocytes have a polygonal aspect, with the cytoplasm finely granulated and basophile and they have 1-2 nuclei. On small areas, towards the hepatic lobules periphery, hepatocytes have a clear cytoplasm, strongly vacuolated. Sinusoid capillaries with a wide lumen are present between the hepatocyte cords, and on small ectasied territories they produce reduced hemorrhagic area.

3. The microscopical images performed in the liver of the individuals in the experimental group 3 reveal the presence of hepatocytes with an acidophil cytoplasm, loaded with reduced granulations. Within the perilobular area, hepatocytes produce dystrophic processes observed on large areas. Sinusoid capillaries are slightly ectasies, with hemorrhagic area observed on reduced territories.

4. The selenium added in the fodder of the individuals in the experimental group 3 have a hepatoprotective role, an aspect suggested by the manifestation, on small territories, of dystrophic processes caused by DON presence and by the vascular network’s hypertrophy.

Bibliography


