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Effect of T-2 toxin and different selenium compounds on the glutathione redox and lipid peroxide status of broiler chickens

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Fusarium sporotrichoides is a widespread mould worldwide, producing 'type A' trichothecene mycotoxins, e.g. T-2 toxin, HT-2 toxin, T-2 triol, T-2 tetraol, scirpentriol and diacetoxyscirpenol. Trichothecene mycotoxins affect the antioxidant status of animals, primarily due to their pro-oxidant effect. Selenium, as active site of the Se-dependent glutathione peroxidases plays important role in the biological antioxidant system to protect the adverse effect of harmful free radicals.

The objective of this study was to evaluate the effect of T-2 toxin without and with different forms of selenium on the glutathione redox and lipid peroxide status of 21 days old broiler chickens. The birds were divided into four groups, namely control, fed with T-2 toxin contaminated feed (2.05 mg kg^{-1}) without selenium supplementation ('T-2'), fed with T-2 toxin contaminated feed and supplemented with seleno-methionine (Sel-Plex®, Alltech, 0.3 mg Se kg^{-1} feed) ('T-2+ORGSe') and fed with T-2 toxin contaminated feed supplemented with sodium selenite, Sigma, 0.3 mg Se kg^{-1} feed) ('T-2+INORGSe'). T-2 toxin was produced experimentally on maize by Fusarium sporotrichioides strain NRRL 3299. Five animals were exterminated at the start of experiment as absolute control, followed by extermination of 5 birds from each group at days 3, 7 and 14. Blood, liver, kidney and spleen samples were taken, in which reduced glutathione (GSH), malondialdehyde (MDA) concentration and glutathione-peroxidase (GSHPx) activity were measured.

In blood plasma higher ($P<0.05$) GSH concentration were measured in 'T-2' group compared to control (day 14). In red blood cell haemolysate of the 'T-2' and 'T-2+ORGSe' groups lower ($P<0.01$) MDA concentration was measured compared to control (day 3). In line with this at the same time increased GSH concentration were measured in these groups, which was higher ($P<0.01$) in 'T-2+Se' group than the control. T-2 toxin treatment resulted lower ($P<0.01$) GSHPx activity compared to control and both Se-supplemented groups (day 14). In liver homogenate of the treated groups GSH concentrations exceeded during the whole experiment the values of control one, which was significant in 'T-2' (day 7) and 'T-2 + ORGSe' (days 3 and 7) groups. In the 'T-2+INORGSe' group higher ($P<0.05$) GSHPx activity was measured compared to control (day 7). In kidney homogenate – analogously the findings in liver – elevated GSH concentrations were measured in 'T-2' and 'T2-+ORGSe' groups compared to control at each sampling, which was significant in 'T-2' (day 3) and in 'T-2+ORGSe' group (days 7 and 14). GSH concentration of spleen homogenate in 'T-2' (day 14) group and 'T-2+ORGSe' (day 7) groups were lower ($P<0.01$) compared to control.

According to the results consumption of T-2 toxin contaminated feed (2.05 mg T-2 toxin kg^{-1} feed) for two weeks affects the biological antioxidant system of broiler chickens, increasing the amount and activity of glutathione redox system during the first week of mycotoxin exposure, which efficiently protects the lipids from harmful peroxidation processes. However, in the organs which are play important role of metabolism and elimination of T-2 toxin (e.g. liver and kidney), lowered the amount/activity of glutathione redox system was found in the latter half of the T-2 toxin exposure, causing oxidative stress (as measured by the significantly higher MDA concentrations), while Se-supplementation has beneficial effect in the glutathione redox status.