supplementation of the experimentally T-2 contaminated diet resulted improvement of the antioxidant and moderately in lipid peroxide status.

The possible causes of the lack of dose-relation would be the environmental factors, e.g. temperature and light regimen, also partially different genetic background of the experimental animals, even all of them was the same hybrid. The other possible cause would be the presence or lack of natural metabolites of T-2 toxin or some other not-identified trichothecene mycotoxins, because the experimental contamination was carried out using crude extract from the mainly T-2 toxin producing moulds, *Fusarium sporotrichioides* or *Fusarium tricintum.*

In conclusion it can be stated that T-2 toxin exposure has some pro-oxidant effect also activated or impaired the amount/activity of the glutathione redox system but its effect depends on the duration of the study also some other factors. Additionally, the question, that T-2 toxin has direct or indirect pro-oxidant effect remains open.

Effects of postconditioning on kidney ischemia/reperfusion injury in hypercholesterolemic rats

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Ischemia/reperfusion injury frequently threats the integrity of the organs during surgery. The protective effect of postconditioning (PK), the short repetitive ischemia/reperfusion cycles, applied in the beginning of reperfusion, has been improved the outcome in vital organs. Signaling cascades are induced by PK interfere in several points to preconditioning, which is blocked by metabolic diseases, such as insulin resistance and type 2 diabetes.

The aim of our study was to compare the efficacy of PK after reperfusion injury of both kidneys in metabolically healthy and hypercholesterolemic rats.

Male Wistar rats (N=30) were divided into two groups. Control group of the animals were fed by normal rat chow, the treated group (n=18) was fed with 1.5% cholesterol containing diet for 8 weeks. Both groups of rats were divided to further two subgroups, and were anaesthetized by ketamin: diazepam. One subgroup of rats was subjected to 45 min ischemia and 2 hours reperfusion, in the other subgroups 4x5 min ischemia/reperfusion cycles were applied in the early phase of reperfusion. After 2 hours of reperfusion blood and tissue (kidney, heart, liver, lung) samples were taken. Serum cholesterol, glucose and triglyceride levels were determined by photometric methods. Kidney function was characterized by serum urea, and creatinin levels. Inflammation and oxidative stress were characterized by the measurement of TNF-α and oxLDL concentrations (ELISA) and PMA induced free radical production capacity of whole blood by chemiluminometric method. Tissue injury in kidney was determined by formaline-fixed, paraffin embedded tissue sections (5 μm), stained with PAS and HE. TNF-α levels were also determined by immunohystochemistry.

Serum cholesterol and triglyceride levels were significantly higher in cholesterol fed rats than in control ones. Serum urea and creatinine levels were same in control and hypercholesterolemic groups. A significant elevation was observed in TNF-α level ($p<0.01$), PMA-induced free radical production ($p<0.05$), and in lipid peroxydation (oxLDL; $p<0.05$) after I/R injury in healthy rats, which reduced almost to the normal levels in PK ones. In hypercholesterolemic rats neither the elevation, nor the postconditioning induced reduction were not as significant as in the healthy rats. Surgical intervention caused a great elevation in serum glucose and insulin levels ($p<0.01$). PK caused a further elevation in insulin levels, while the TNF-α concentration and free radical levels were reduced. Tissue TNF-α level, measured in hypoxia sensitive papilla, was significantly higher in cholesterol fed animals, than in control rats, and this high level was not able to change in response to PK. In healthy animals PK caused a significant reduction in tissue TNF-α level, as well.

PK proved to be a very effective defense against I/R in healthy animals, but it was ineffective in hypercholesterolemic ones.