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THE EFFECT OF SELENIUM ON PHENYL MERCURY TOXICITY AND MERCURY RETENTION IN CHICKEN

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Phenylmercuric chloride in a dose 30 ppm of mercury alone and the same dose supplemented with 4 ppm of selenium were fed to chickens of both sexes for 8 weeks. Body weight, mortality rate, pathological changes and the level of mercury in the muscle, liver, and kidneys of the laying hens and cocks were analysed. After the dosage period, the mean body weight of the hens was unaffected, while in the group of cocks fed 30 ppm Hg without selenium addition, a decrease occured. In the group with Se addition no significant change in body weight of either sex was observed. Differences in the affinity of some organs to bind Hg were observed. Moreover, supplementation with 4 ppm of selenium resulted in significantly increased mercury levels in the organs analysed. The highest increase was found in the liver, but there was the same increasing tendency in the muscle and kidneys.

Key words: selenium, mercury, toxicity, retention, chicken

INTRODUCTION

It is known that selenium protects against the toxic effects of mercury (Paøizek and Oštadalova, 1967). This has been observed in a number of different organisms. The effect of selenite–mercury interaction include enhancement of the growth rate but also showed that selenite has a protective effect against renal necrosis and mortality caused by mercuric chloride (Potter and Matrone, 1974). After Parizek and Oštadalova (1967) reported the protective effect against mercury toxicity in rats, other studies about the Se-protective effectagainst Hg in organic form have been presented for various animal species (Stoewsand *et al*., 1974; Sell, 1977; Burk *et al*., 1980; Naganuma *et al*., 1981; Cuvin-Aralar and Furness, 1988, 1990; Grosicki and Kossakowski, 1993; Maretta *et al*., 1995; Pribilincova *et al*., 1996; 1997; Marettova *et al*., 2000). The aim of the present research was to determine the toxicity and distribution of mercury in three organs from chickens after long term exposure to phenylmercuric chloride in the presence of selenite.

MATERIAL AND METHODS

Animal model

Two experimental groups each of 12 laying hens and one control group were formed. The hens were all Shaver Starcross 288 hybrids, line 589, in their first year of laying. The same number of experimental and one control group of cocks (Shaver Starcross 288, line 579) was also formed. The diet for the control groups (K) was to the a complete feed mixture NVRM for high performance laying hens with added methoinine in which measurements have revealed 0.018 ppm of mercury. Mercury treatment was as follows: Group 1 = 30 ppm Hg as phenylmercury chloride, Group 2 = 30 ppm Hg + 4 ppm Se as sodium selenite added to the basal diet. The mean feed intake for hens and cocks was 120 and 140 g per bird/d, respectively. The experimental birds were placed in three floor cages.

Observations and analyses

Observations included the initial and final live weights, the mortality, and the analyses of tissue mercury level. Mercury was determined in muscle, liver and kidneys using an atomic absorption spectrometer (TM 254). Tissue samples from the selected organs were fixed in 10% neutral buffered formalin, embedded in paraffin, and sections stained with haematoxylin and eosin.

Statistical analysis

The results for Hg levels in organs were evaluated statistically by analysis of variance and by the Duncan test.

RESULTS

Body weight and mortality rate

No mortality was registered among the experimental animals during the experiment. After exposure to Hg the body weight of the hens was not affected but a decrease in body weight of 70 g was observed for the cocks in the group fed 30 ppm Hg, during the application period.

Pathological findings in chicken

Histollogically, the effect of mercury on the kidneys and liver was revealed by structural changes in bith experimental groups. The light microscopic examination of the liver showed loose and collapsed hepatic cords and dilatation of the sinusoids. Next to the portobiliary space, a noninflammatory peribiliary infiltrate was present (Fig. 1). Occasional vacuolization of hepatocyte cytoplasm was observed. The most conspicuous histological changes were observed in the kidneys, where glomerular shrinkage with cellular infiltration (Fig. 2) as well as several focal parenchymal necroses and degeneration of the tubular epithelium wewe found (Fig. 3).

Figure 1. The liver parenchyma of a bird from experimental group 2. Dilatation of the blood sinuses and portobiliary infiltration of lymphoid cells can be seen. HE. x 150

Figure 2. The kidney parenchyma of a bird from experimental group 2. Shrinkage of the renal glomeruli connected with cellular infiltration is present. HE. x 350

Residual Hg levels in the tissues

Residual Hg levels in the tissues of laying hens without and with Se supplementation are shown in Fig. 4. Mercury in different amounts was found in both experimental groups. In the first experimental group (30 ppm Hg) the lowest mean level of mercury was detected in the muscles (0.318 mg.kg $^{-1}$), whereas a considerably higher level was in the liver (2.815 mg.kg $^{-1}$) and the highest level was found in the kidneys (11.906 mg.kg $^{-1}$). In the second experimental group which

Figure 3. The kidney parenchyma of a bird from experimental group. 2. Local necrosis of the renal cortex and local dilatation and atrophy of tubules. HE. x 150

received mercury and selenium, the mercury level in all tissues analysed was higher than in the hens that received only the mercury supplement. In comparison with the 1st group (without the Se supplement) the residual mercury levels were four times higher in the muscle (1.363 mg.kg $^{-1}$), twelve times higher in the liver (33.529 mg.kg $^{-1)}$ and four times higher in the kidneys (47.095 mg.kg $^{-1)}$. The increase in residual mercury levels in all organs analysed from the experimental group supplemented with Se was highly significant ($p<0.01$). Although in our experiment a considerable increase of the Hg level in the liver was observed after Se supplementation, the affinity of the organs analyzed did not change.

Figure 4. The kidney parenchyma of the 2nd experimental group. Shrinkage of the renal glomeruli connected with cellular infiltration is present. HE. x 350

DISCUSSION

Differences in the affinity to bind Hg were observed in the organs studied in the breeding hens and cocks. The affinity of Hg to be bound to muscle tissue was generally very low though an increase was found when Se was added. According Cuvin-Aralar and Furness (1990) mercury diverted away from the kidney is believed to be redistributed in the muscle.

Although an increase of Hg level in the muscle was observed after Se supplementation a much higher increase was found in the parenchymatous organs. Thus, the affinity of the liver to bind the Hg in our experiment was remarkably increased. In the broiler chicks Rubenstein and Soares (1979) observed that after methylmercury and selenium treatment the accumulation of Hg in the liver increased with the duration of exposure to Hg. According to these authors methylmercury may undergo biotransformation before it can be excreted in the bile, and the reabsorption of biliary Hg from the intestines, and its subsequent enterohepatic recirculation could be the cause of increased Hg retention. The results of our experiment confirm such a supposition. In rats a significant increase in mercury levels was observed after selenium treatment. The highest increase in the Hg level was found in the liver where, in the experimental group which received Se supplemmentation, it was twelve times higher than in the group without selenium addition (Fang, 1977).

The highest levels of mercury were found in the kidneys of the chickens in both experimental groups 1 and 2. The Hg level was about 4 times higher in the kidneys than in the liver in the group with Se addition, which confirms knowledge about the affinity of this organ for mercury. On the contrary, Potter and Matrone (1974) demonstrated a decreased percentage in the kidneys of rats fed with selenite. The authors supposed that selenium causes a reduction in the rate at which mercury is taken up by the kidney. The results we obtained with phenylmercuric chloride in the kidneys of hens may not correspond with the above mentioned observations because of differences in the form of Hg and the dosage we used in our experiment.

The results obtained confirm the existence of a biological interrelationship between mercury and selenium. The mechanisms of interaction between selenium and mercury are not well understood. However the results obtained confirm the existence of biological interrelationship between mercury and selenium. Parizek *et al.* (1969) showed that the administration of Se compounds did not increase Hg excretion, but, on the contrary, markedly increased the level of Hg in the blood, where the increase depended upon the dose of Se. *In vivo* and *in vitro* studies of mercury and selenium in rabbit blood after simultaneous administration of methyl mercuric chloride and selenite showed that the rate of mercury uptake by erythocytes was much more rapid than when methyl mercury was added alone. It has been shown that selenium treatment of methyl mercury-exposed rodents may decrease Hg accumulation in the kidneys, and enhance whole-body Hg elimination rate (Komsta-Szumska *et al.*, 1983; Wicklund Glynn *et al.*, 1993). In the female mice Wicklund Glynn and Lind (1995) found that Se supplementation did not induce alterations in Hg accumulation and distribution in the blood cells,

nor were there changes in the accumulation and distribution of Hg in the kidneys. According to Cuvin-Aralar and Furness (1990) selenite not only affects mercury uptake by the kidney but also its retention. These findings and our observations led to the conclusion that the administration of Se compounds not only increases retention of Hg but also changes its distribution within the body.

Elimination studies revealed that the presence of selenium does not improve the elimination of mercury in fish (Cuvin-Aralar and Furness, 1988) or chicken. In fact, the relese of mercury in the presence of selenium was significantly decresed compared with groups treated only with mercury. According to Cuvin-Aralar and Furness (1990) on of the effects observed in the selenium treatment of mercury intoxicated animals is an apparent modification of the distribution pattern of mercury in the different organs and tissues. However, there were no consistent effects of Se supplementation on retention of Hg in the animals. Wicklund Glynn and Lind (1995) had an indication that Se influences tissue accumulaion an the intracellular distribution of Hg through tissue-specific mechanisms rather than through a more general effect on Hg sequestration and transport in the blood. Naganuma *et al*. 1988, Imura and Naganuma, 1991), The hypothesis of these authors was that the proposed mechanisms for Se effects on Hg dynamics in blood will result in alterations of the Hg level and distribution in the blood, which subseqently will lead to alterations in te accumulation and distribution of Hg in tissues.

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UTICAJ SELENA NA TROVANJE FENOLIMA ŽIVE I NA RETENCIJU ŽIVE KOD PILIĆA

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SADRŽAJ

U toku ogleda od 8 nedelja, dve grupe pilića su hranjene smešama sa dodatkom fenilmerkuri hlorida (30 ppm žive) a jednoj grupi je dodavan i selen u koncentraciji od 4 ppm. Utvrđeno je da se tokom ogleda kod grla hranjenih smešama bez selena telesna masa kokica nije značajnije razlikovala dok je kod petlića uočen pad. U grupi koja je dobijala selen nije bilo ovih razlika. Suplementacija selenom je imala za posledicu povećanu retenciju žive, posebno u jetri.