

## THE EFFECT OF SELENIUM ON MERCURY RETENTION IN THE OFFSPRING OF TREATED HENS

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*The levels of mercury in the chicken organs were determined after long term oral administration of mercury to laying hens. Phenylmercuric chloride was fed to laying hens and cocks for 28 and 56 days in a dose 30 ppm of mercury alone and the same dose supplemented with 4 ppm of selenium. The mortality rate of their offspring and the level of mercury in the chick muscle, liver and kidneys after seven days of rearing were analysed. The mercury accumulated in the fertilized eggs was transferred from the hens to their offspring. The mercury level in the offspring of both experimental groups was highest in the kidneys, followed by liver and muscle. The addition of selenium to the diet for laying hens resulted in a different rate of a decrease of mercury level in their offspring in all organs analysed.*

*Key words: selenium, mercury, retention, chicks offspring*

### INTRODUCTION

The developing body in its prenatal and postnatal stages may be at a higher risk from toxic metal exposure than in the adult stage (Jugo, 1979, Kostial, 1983). Mercury (Hg) distribution and its effect on rat body weight gain were found to be different in different postnatal development phases (Sakamoto *et al.*, 1993). It seems possible that the mercury distribution might reflect the toxic effects of methylmercury during a given phase of development (Sakamoto and Nakano, 1995). The protective effect of selenium (Se) against mercury toxicity has been observed in a number of different animals (Palizek *et al.*, 1969; Stiling *et al.*, 1974; Stoewsand *et al.*, 1974; Potter and Matrone, 1974; Sheline, Schmidt-Nielsen, 1977; Burk *et al.*, 1980; Cuvin-Aralar and Furness, 1990, Imura and Naganuma, 1991). No information exists on phenylmercury toxicity in the development stages of fowl nor about the protective effect of selenium against the toxic effect of phenylmercury in chicks. The aim of this study was to determine the distribution of Hg in the offspring of treated hens at the beginning of their lives.

## MATERIAL AND METHODS

Two experimental groups each consisting of 12 chicks descended from laying hens fed with phenylmercuric chloride and one control group ( $n=6$ ) were used in the experiment. The laying hens (hybrids of Shaver Starcross 288, line 589) were in their first year of laying and cocks ( $n=6$ ) (hybrids Shaver Starcross 288, line 579) of the same age were used. Except for the controls, phenylmercuric chloride and sodium selenite were added as supplements to the feed mixture (NVRM). NVRM is a complete feed mixture, with methionine added for high performance laying hens, for which measurements revealed 0.018 ppm of mercury. Mercury treatment in the laying hens and cocks was as follows: Group K = control group, Group 1 = 30 ppm Hg, Group 2 = 30 ppm Hg + 4 ppm Se. The mean feeding rations for hens and cocks were 120 and 140 g per bird/d, respectively. The experimental birds were placed in three floor cages. Insemination at 7 day intervals, at a dosage of 0.030 ml/bird of semen, was used for the fertilization of eggs. The mortality of experimental birds and viability of chicks up to 7 days of age and offspring was observed daily. Mercury was determined in the muscle tissue, liver and kidneys of 7-day-old chicks hatched from eggs collected from laying hens after 28 days (1<sup>st</sup> sampling) and 56 days (2<sup>nd</sup> sampling) of Hg application using an atomic absorption spectrometer TM 254. The results for Hg levels in organs were evaluated statistically by analysis of variance and by the Duncan test.

## RESULTS

*Chick mortality*

Chick mortality from eight hatchings, reached its minimum after seven days of rearing. It was 0.79% (3 from 378 hatched) in group 1 (hens fed 30 ppm Hg), and 0.26% (1 from 379 hatched) in group 2 (hens fed 30 ppm Hg + 4 ppm Se).

*Residual Hg levels in the tissues*

Data from the examination of Hg levels in muscle, liver, and kidneys are recorded in Figs. 1 and 2. The Hg levels in the chick offspring and hens differed according to the organs analysed. The retention of mercury in the organs of chicks developed from the eggs of hens fed 30 ppm Hg in first 28 days of the experiment (Fig 1, 1<sup>st</sup> sampling) was very low, especially in the muscle, slightly higher in the liver, and highest in the kidneys. Namely, the Hg level in the muscle tissue was 0.348 mg.kg<sup>-1</sup>, in the liver 1.389 mg.kg<sup>-1</sup> and in the kidneys 2.362 mg.kg<sup>-1</sup>. In the offspring developed from hens that received 30 ppm Hg + 4 ppm Se, the Hg level was even lower being in the muscle 0.109 mg.kg<sup>-1</sup>, in the liver 1.029 mg.kg<sup>-1</sup> and in the kidneys 1.813 mg.kg<sup>-1</sup>. After 56 days of the experiment (Fig. 2, 2<sup>nd</sup> sampling) in the chicks developed from hens fed Hg without Se the Hg level, in the muscle had decreased to 0.138 mg.kg<sup>-1</sup> but in the liver and kidneys the Hg level had increased considerably to 2.228 mg.kg<sup>-1</sup> and 4.385 mg.kg<sup>-1</sup>, respectively in comparison to 1<sup>st</sup> sampling. In the chicks from hens fed Hg with the addition of Se, the Hg level, in the muscle was slightly increased to 0.142 mg.kg<sup>-1</sup> in comparison to the 1<sup>st</sup> sampling, whereas in the liver it decreased to 0.920 mg.kg<sup>-1</sup> and in the kidneys it increased to 2.372 mg.kg<sup>-1</sup>. In comparison with the former group, statistical significance ( $p < 0.05$ ) was observed only in the kidneys at the 2<sup>nd</sup> sampling. The same proportional level of Hg in the organs evaluated was

found in the hens from whom the chicks were bred, being lowest in the muscle, higher in the liver and the highest in the kidneys.

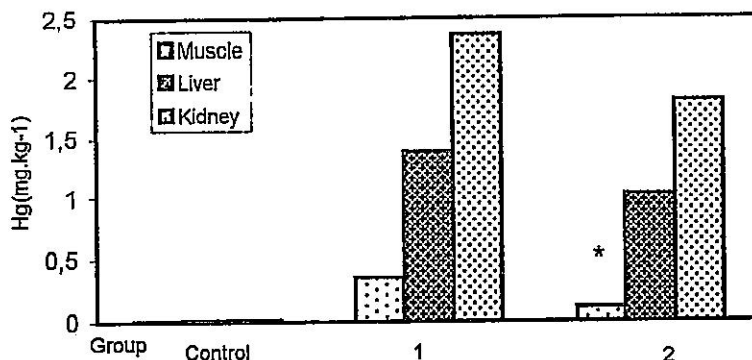


Figure 1. Levels of mercury in tissues of chicks hatched from hens after 28 days of diets without or with Hg (1) or Hg and Se (2). Values marked with an asterisk differ significantly from the 1st group (\*) ( $p < 0.05$ )

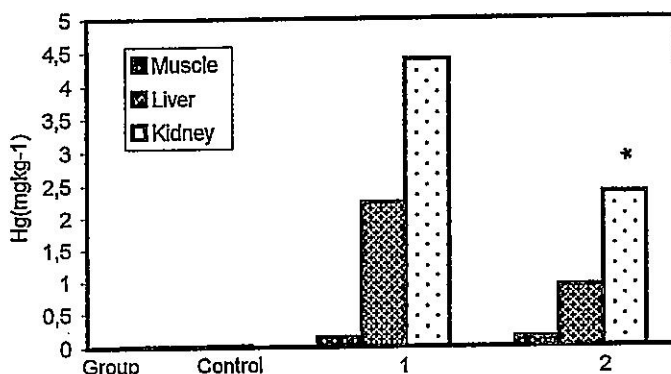


Figure 2. Levels of mercury in tissues of chicks hatched from hens after 56 days of diets without or with Hg (1) or Hg and Se (2). Values marked with an asterisk differ significantly from the 1st group (\*) ( $p < 0.01$ )

## DISCUSSION

The results we obtained in the experiment showed that Hg in the progeny of treated hens is distributed in the body in the pattern known from adult chickens (Marettova *et al.*, 2000). In adult hens it was found that the liver and kidneys accumulated more mercury on a proportional basis than did the skeletal muscles (Pribilincova *et al.*, 1996, 1997). This confirms the reports on mercury distribution in several tissues mainly parenchymatous organs – liver and kidneys. It was also found that the Hg level in the chicks during the first 56 days of treatment changed.

At 28 days in the offspring of hens receiving Hg only, the Hg level in the liver was four times and in the kidneys almost seven times higher than in the muscle. At 56 days the Hg level in the liver and in the kidneys increased near doubled. This coincides with the observation of Sakamoto and Nakano (1995) for Hg concentration and the % of total Hg administered to rats found in the kidney, which increased markedly with the postnatal phase. The high Hg level in the kidneys in the postnatal period explained to be a result of the functional maturation of the kidney as the glomerular filtration rate is known to be very low in immature kidneys (Spitzer, 1985). Drasch *et al.* (1993) observed an increase of Hg in the kidneys of human infants after birth. These authors suppose this was caused by a redistribution of Hg in the body from the other organs to the kidneys or by a new intake of Hg. We can confirm this findings also in the kidneys of chicks during the second period of the experiment (28 - 56 days), when the Hg level in the kidneys increased considerably in comparison with the Hg level in the liver and muscle, and from the first period it doubled. According to Sakamoto and Nakano (1995), who used methylmercury, the Hg in the body may undergo biotransformation before it can be excreted in the bile, and the reabsorption of biliary excreted Hg from the intestines, and its subsequent recirculation via enterohepatic circulation could be the cause of the increased Hg retention. The reason for this phenomenon can be found in the slow blood flow into the renal tissue which may lead to less accumulation of methylmercury in the kidney of neonatal rats.

As for the liver, our results for chicks coincide with the observations of Sakamoto and Nakano (1995), who showed that Hg concentration and the % of total Hg administered were highest in the rat liver in the first 14 postnatal days. The explanation for this phenomenon can be based on the level of metallothioneine in chicks after birth or their greater ability to eliminate Hg. In the foetal liver, an extremely high level of metallothionein-like protein is present, which prevents further distribution of Hg from the foetal liver (Drasch *et al.*, 1993). We suppose that the rather high level of Hg in the chick liver can be related to the amounts of Hg in the yolk and white (Pribilincova *et al.*, 1996) of the egg during embryonal development before hatching and with the higher ability of the liver to bind mercury.

Mercury level at 28 days in the chick offspring descended from hens exposed to mercury with Se supplementation, displayed a lower Hg level in all organs analysed in comparison to the group without Se supplementation. A proportionally higher Hg level was found in the muscle and liver. In hens, proportionally more Hg was present in the kidneys in comparison to the liver and muscle (Pribilincova *et al.*, 1997). The same pattern of Hg distribution was found in the group with Se, though in lower amounts than in the group without Se. At 56 days, the same Hg level was found in the muscle, as in group 1, but there was a greater decrease in the liver and kidneys. In any case, the results we obtained with the chicks confirm the Se-protective effect against Hg for the organic and inorganic forms as has been found in various animals (Stoewsand *et al.*, 1974; Potter and Matrone, 1974; Burk *et al.*, 1980; Cuvín-Aralar and Furness, 1990). This effect was not observed in hens in which the Hg level in the liver and kidneys was enormously high (Pribilincova *et al.*, 1997).

Studies on the effect of different mercury compounds (mercuric chloride, methylmercuric chloride, and phenylmercuric acetate) on the tissue distributions of selenium gave no definite trend in terms of one compound being more effective than the others in altering tissue. Wicklund-Glynn and Lind (1995) indicated that

Se influences tissue accumulation and the intracellular distribution of Hg through tissue-specific mechanisms. Elimination studies revealed that the presence of selenium did not improve the rate of elimination of mercury in fish (Cuvin and Furness, 1988) and neither for chickens, but improved in chick offspring at hatching. In fact, the release of mercury in the presence of selenium was significantly decreased compared with groups treated only with mercury. According to Cuvin-Aralar (1990) one 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 which, according to this study, is different in adult chickens and very young offspring.

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#### UTICAJ SELENA NA ZADRŽAVANJE ŽIVE KOD PODMLATKA TRETIRANIH KOKOŠAKA

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#### SADRŽAJ

Ova studija je imala za cilj da utvrdi uticaj selena dodatog u hrani na nivo žive u organima izleženih pilića posle dugotrajnog peroralnog unošenja ovog teškog metala od strane njihovih roditelja. Kao izvor žive je korišćen fenil merkuri hlorid koji je dodavan sam u dozi od 30 ppm ili ova ista doza + Se (4 ppm). Živa koja se akumulirala u oplodjenim jajima preneti je na potomstvo i njena koncentracija je bila najveća u bubrezima a zatim u jetri i miškulaturi. Dodavanje selena u hranu za koke imalo je za posledicu smanjenje nivoa žive u svim ispitivanim organima pilića.