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MERCURY AND SELENIUM INTERACTIONS DURING GROWTH AND REPRODUCTION OF CHICKENS (Progress Report)

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Introduction

Elevated levels of mercury exist in various segments of the environment largely from past industrial and agricultural activities. Bacterial conversion of inorganic mercury to the biologically more important methylmercury promotes accumulation in animal bodies. Concentrations tend to increase as methylmercury is passed up the food chain through aquatic organisms to fish and water fowl and eventually to humans. It is this form of mercury, i.e., methylmercury, that has been implicated in egg shell thinning and lowered reproduction in birds.

Past research (South Dakota A.S. Series 75-37) has demonstrated the existence of an interaction between methylmercury and selenium which reduces toxicity of methylmercury in laying hens. The study reported herein was conducted to determine the effect of age and dietary protein level on methylmercury toxicity and on the mercury-selenium interaction in hens from hatching through a complete laying cycle.

Methods

Thirty-two cages each containing eight 1-day-old pullets were allotted to eight treatments replicated four times. Dietary treatments consisted of a control, 15 ppm mercury from methylmercuric chloride, 6 ppm selenium from sodium selenite, and the combination of mercury plus selenium. Each of these was initially fed with two levels of protein, 23% (high-protein series) and 16% (low-protein series). At 12 weeks, these were changed to 16% and 12%, respectively.

At 4 weeks and again at 22 weeks of age, two birds from each cage were killed and tissues were sampled for analysis. The remaining four birds per cage were transferred to laying cages and the diets were changed to standard 16% and 12% protein layer diets for continuation of the high- and low-protein series.

This experiment is still in progress, and egg production, size, quality and shell thickness are being measured. After completion of the laying cycle, the hens will be killed and tissue samples will again be analyzed for mercury and selenium.

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Results

When fed the high-protein diet, neither mercury nor selenium alone had an effect on weight gains. However, the combination of mercury plus selenium reduced gains by 10% during the period of rapid growth to 8 weeks of age. When fed with the low-protein diet, weight gain reductions were more pronounced. Reductions in gains at 8 weeks were as follows for the various mercury and selenium treatments: mercury, 11.4%; selenium, 5.5%; and mercury plus selenium, 26.2%.

Death losses were not greatly different in the various treatments until the laying period following 22 weeks of age. In the first 19 weeks of the laying period, a protective effect of selenium was apparent as evidenced by a 37% death loss in mercury-fed birds and only 12% loss in the combination mercury plus selenium groups. Control and selenium treatment groups had 3 and 6% losses, respectively.

Mercury and selenium concentrations in selected tissues, collected at 22 weeks, are shown in table 1. No important differences in tissue mercury or selenium concentrations existed between birds fed the different protein levels. Of special interest is the extent to which dietary mercury enhanced tissue selenium accumulations, particularly in liver and kidney, at 22 weeks. In kidney, a mercury to selenium ratio averaging 7.3 for the high-protein and 6.6 for the low-protein series was observed in mercury-fed birds whether or not the 6 ppm of supplemental selenium were fed. Apparently, the 0.3 ppm selenium inherent in the basal diet were adequate to support this relationship in the early phase of the study. It remains to be determined whether this ratio is maintained in kidney or reaches this level in other tissues as the birds become older, especially in those hens afforded protection from mercury-induced deaths by use of higher levels of selenium.

Summary

Suboptimum dietary protein levels, while exerting a small influence upon the severity of mercury and/or selenium toxicity as measured by lower weight gains, did not alter the distribution or concentration of mercury or selenium in the tissues. Also, dietary selenium did not appear to alter tissue mercury levels or distribution. Conversely, dietary mercury increased tissue selenium levels, particularly in kidney and liver. Age of the birds appears to be an important factor with regard to the extent that mercury interacts with selenium and the subsequent protection that this interaction may provide against methylmercury toxicity.

Table 1. Mercury (Hg) and Selenium (Se) Accumulations in Chicken Tissues at 22 Weeks of Age (ppm)

Dietary treatment		Liver		Kidney		Brain		Breast		Feathers	
ppm Hg	ppm Se	Hg	Se	Hg	Se	Hg	Se	Hg	Se	Hg	Se
					High-Protein Series						
0	0	0.0	1.0	0.3	0.9	0.2	0.3	0.0	0.4	0.6	1.0
15	0	60.7	3.1	55.4	7.6	12.9	0.4	18.4	0.4	469.0	0.9
0	6	0.0	2.9	0.3	2.0	0.2	0.5	0.0	0.4	0.6	3.2
15	6	51.6	8.0	34.5	4.8	15.0	1.9	14.6	0.7	492.0	2.3
					Low-Protein Series						
0	0	0.0	1.0	0.2	0.9	0.2	0.3	0.0	0.3	0.6	1.0
15	0	57.9	3.3	56.8	8.3	12.4	0.4	19.7	0.4	491.0	0.9
0	6	0.0	3.3	0.3	2.8	0.2	0.5	0.0	0.5	0.8	3.4
15	6	55.6	8.9	41.2	6.5	16.6	2.7	18.2	0.6	500.0	2.4