Dietary Selenium Protection of Methylmercury Intoxication of Japanese Quail[1]

by

G. S. STOEWSAND

Department of Food Science and Technology
New York State Agricultural Experiment Station
Cornell University, Geneva, N. Y. 14456

and

C. A. BACHE and D. J. LISK

Pesticide Residue Laboratory
Cornell University, Ithaca, N. Y. 14850

Selenium is both an essential nutritional element required for growth (THOMPSON and SCOTT 1969) and a toxicant when present in the diet in excess of 5 ppm (CAMERON 1947). A series of studies on the interaction of inorganic mercury and selenium (PARIZEK and OSTADALOVA 1967; PARIZEK et al. 1968; PARIZEK et al. 1969) indicated that selenium decreases the toxicity of injected inorganic mercury in rats. This "protective effect" apparently is caused by a decrease in vivo distribution of mercury. Decreased mercury intoxication of rats fed diets containing both mercury and selenium has been observed previously (GANTHER et al. 1972), yet there appears to be an enhanced organ accumulation of both mercury and selenium when diets contain both elements (JOHNSON 1972).

Foods, such as tuna and swordfish, containing relatively high levels of methylmercury together with selenium, tend to increase the retention of each toxicant with a concomitant depressed syndrome of mercury intoxication when fed to Japanese quail (GANTHER et al. 1972). The purpose of this study was to determine when methylmercury intoxication occurs in Japanese quail fed diets containing selenium. In addition, we sought to determine if high methylmercury residues in liver, kidney, brains or produced eggs is correlated to the development of methylmercury toxicosis.

EXPERIMENTAL

Day-old Japanese quail (Coturnix coturnix japonica), housed in a commercial type chick brood unit, were fed purified diets (STOEWSAND and ROBINSON 1970) with added methylmercury or selenium alone or in combination. Specifically the treatments included:

---

[1] Approved by the Director of the New York State Agricultural Experiment Station for publication as Journal Paper No. 2012.

Bulletin of Environmental Contamination & Toxicology,
Vol. 11, No. 2 © 1974 by Springer-Verlag New York Inc.

A. diet with added methylmercury for four weeks
B. diet alone for four weeks followed by diet with added methylmercury for five additional weeks
C. diet with added selenium for four weeks followed by diet with added methylmercury for five additional weeks
D. diet with added methylmercury and selenium for four weeks followed by diet with added methylmercury for five additional weeks
E. diet with added methylmercury and selenium for nine weeks.

Methylmercury was added to the diets as methyl mercuric chloride, in corn oil, equivalent to 20 ppm of mercury, and selenium was added as sodium selenite, dissolved in water, equivalent to 5 ppm of selenium. Methylmercury was isolated (WESTÖÖ 1967) from composite samples of liver, kidney, brain and eggs and analyzed by gas chromatography with a microwave-powered emission detector (BACHE and LISK 1970). The recoveries of methylmercury listed as a footnote in Table 2 were in the range of 70% since this is the level of recovery which Westöö obtained owing to unfavorable partition coefficients which result in about a 30% loss of methylmercury.

RESULTS AND DISCUSSION

Adding methylmercury to the diets of quail produced over 90% mortality within 2 weeks (Table 1). The addition of selenium to methylmercury-containing diets produced 21% mortality, close to that of the controls, or that of selenium fed alone. No additional quail died in these latter three treatments after the first two weeks of the experiment. Very young Japanese quail have a poor homeothermic mechanism and are susceptible to slight drafts (NATIONAL ACADEMY OF SCIENCES 1969).

TABLE 1

Mortality of Young Japanese Quail Fed Methylmercury and Selenium

Treatment[1]	Initial No. of Quail	Mortality at 2 weeks %
0	78	21.8
Se	39	20.5
CH_3Hg + Se	38	21.0
CH_3Hg	77	92.2

[1] CH_3HgCl equiv. to 20 ppm. Hg; $Na_2SeO_3.5H_2O$ equiv. to 5 ppm. Se

Pretreatment for the first four weeks with selenium, delayed the onset of death caused by the methylmercury intake during the last five weeks of the feeding experiment.(Figure 1). The delay of death was even more pronounced when the pretreatment diet included both selenium and methylmercury. Feeding the combined selenium and methylmercury for the entire nine weeks produced the most dramatic reduction in mortality.

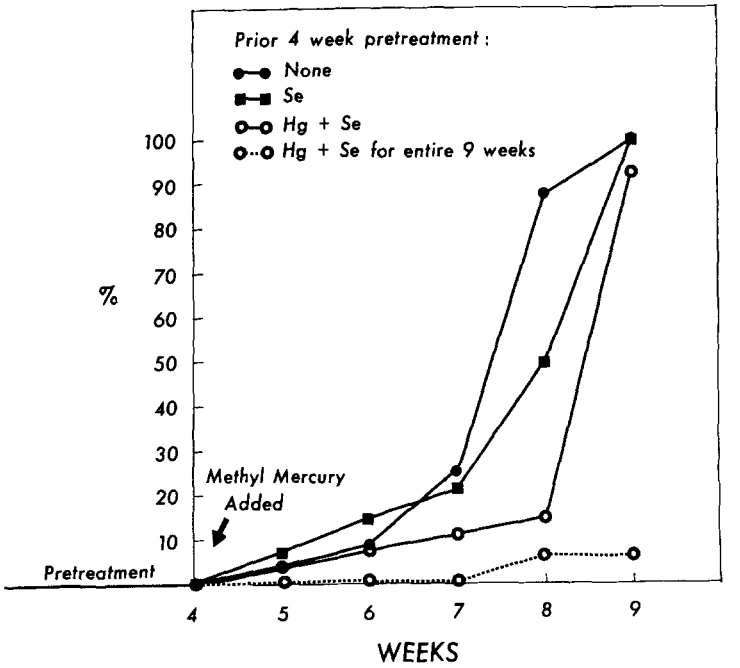

Fig. 1. Cumulative % mortality of Japanese quail fed methylmercury or methylmercury + selenium after a 4 week dietary pretreatment of selenium or methylmercury + selenium. Control quail received neither element during the pretreatment period.

Organ methylmercury residues are presented in Table 2. Although methylmercury alone added to the diet caused early symptoms and mortality (Figure 1), the organ residues of methylmercury in general were lower than in those (especially in the males) pretreated with selenium and methylmercury. The brains of male quail fed methylmercury and selenium throughout the pretreatment and treatment (9-week feeding period) had extremely high methylmercury residues, yet no symptoms or mortality occurred. In general, female quail had lower levels of methylmercury in their organs, compared

with males, as previously observed with inorganic mercury (STOEWSAND et al. 1971). It is apparent that the level of methylmercury in the organs, or in produced eggs is not correlated with mercury toxicosis.

TABLE 2

Methylmercury Residues in Liver, Kidney, Brain, and Eggs of Japanese Quail Fed Selenium and Methylmercury

Pretreatment (4 weeks)	Treatment (5 weeks)	Sex	Methylmercury Residues[1] Liver	Kidneys	Brain	Eggs
None	None	M	0.05	0.04	0.23	
		F	0.08	0.03	0.25	0.04
Se	Se	M	0.02	0.02	0.04	
		F	0.02	0.01	0.03	0.01
None	CH_3Hg	M	53.7	50.5	20.7	no
		F	42.4	24.4	18.7	eggs
Se	CH_3Hg	M	63.4	58.9	27.7	
		F	52.0	50.5	15.7	48.1
CH_3Hg + Se	CH_3Hg	M	74.2	88.6	33.9	
		F	76.4	69.1	31.6	74.5
CH_3Hg + Se	CH_3Hg + Se	M	60.0	38.5	40.4	
		F	37.9	25.0	22.7	34.6

[1] Recoveries of methylmercury added to tissues and eggs at .043 ppm were 74.0% and 71.0% respectively. Duplicate determinations.

In vivo retention of inorganic mercury or cadmium had been reported when mice are injected with selenite (EYBL et al. 1969). This mechanism of increased retention of various mercury compounds with selenium suggests that there is a marked depressed availability of methylmercury for toxic reactivity at the receptor sites within the organism. Apparently selenium must constantly be present with methylmercury, perhaps forming intermediate complexes, since in our studies the birds pretreated with methylmercury and selenium, and then fed diets containing only methylmercury, showed a delayed but eventual mercury-toxic response. Continued feeding of selenium prevented this reaction.

SUMMARY

Selenium, as sodium selenite, added at 5 ppm to purified diets of Japanese quail protected against methylmercury intoxication. Selenium fed simultaneously with methylmercury to quail for 9 weeks gave complete protection. However, feeding selenium with methylmercury for 4 weeks, followed by a diet containing only methylmercury, delayed the onset of methylmercury intoxication for 1-2 weeks as compared to quail not pretreated with selenium. On diets which contained 20 ppm of methylmercury but no selenium, over 90% mortality was observed for young quail within 2 weeks, and mature quail within 4 weeks. Methylmercury residues in liver, kidney, and brain are higher in male than female quail. High methylmercury content of these organs, or in produced eggs, does not indicate that birds will show evidence of methylmercury toxicosis.

REFERENCES

BACHE, C. A. and D. J. LISK: Anal. Chem. 43, 950 (1971).
CAMERON, G. R.: J. Path. Bact. 59, 539 (1947).
EYBL, V., SYKORA, J. and F. MERTL: Experientia 25, 504 (1969).
GANTHER, H. E., GOUDIE, C., SUNDE, M. L., KOPECKY, M. J., WAGNER, P., OH, S. W., and W. G. HOEKSTRA: Science 175, 1122 (1972).
JOHNSON, S. L.: M.N.S. Thesis, Cornell University, Ithaca, N.Y. (1972).
NATIONAL ACADEMY OF SCIENCES: Publ. 1073, Printing Office, Washington, D. C. (1969).
PARIZEK, J. and I. OSTADALOVA: Experientia 23, 1 (1967).
PARIZEK, J., BENES, I., OSTADALOVA, I., BABICKY, A., BENES, J. and J. PITHA: In Mineral Metabolism in Paediatrics, Symposium at Glaxo Laboratories, Greenford, Middlesex, England (D. Baltrop and W. L. Burland, eds.) 117-134 (1968).
PARIZEK, J., BENES, I., OSTADALOVA, I., BABICKY, I., BENES, J. and J. LENER: Physiol. Bohemoslov. 18, 95 (1969).
STOEWSAND, G. S., ANDERSON, J. L., GUTENMANN, W. H., BACHE, C. A. and D. J. LISK: Science 173, 1030 (1971).
STOEWSAND, G. S. and W. B. ROBINSON: Amer. J. Enol. Viticul. 21, 174 (1970).
THOMPSON, J. N. and M. L. SCOTT: J. Nutrition 97, 335 (1969).
WESTÖÖ, G.: Acta Chem. Scand. 21, 1790 (1967).