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IMPAIRED REPRODUCTION OF MALLARDS FED AN ORGANIC FORM OF SELENIUM

GARY H. HEINZ, U.S. Fish and Wildlife Service, Patuxent Wildlife Research Center, Laurel, MD 20708
DAVID J. HOFFMAN, U.S. Fish and Wildlife Service, Patuxent Wildlife Research Center, Laurel, MD 20708
LYN G. GOLD, U.S. Fish and Wildlife Service, Patuxent Wildlife Research Center, Laurel, MD 20708

Abstract: We fed mallards (*Anas platyrhynchos*) diets supplemented with 0-, 1-, 2-, 4-, 8-, or 16-ppm selenium in the form of selenomethionine. We fed another group of mallards a diet containing 16-ppm selenium as selenocystine. Females fed the control diet produced a mean of 8.1 ducklings that survived to 6 days of age, which was significantly greater than the 4.6 young produced by females fed 8-ppm selenium as selenomethionine and the zero surviving young of females fed 16-ppm selenium as selenomethionine. Selenocystine did not impair reproduction. Diets containing 8- and 16-ppm selenium as selenomethionine caused malformations in 6.8 and 67.9%, respectively, of unhatched eggs compared with 0.6% for controls. The most common malformations were of eyes, bill, legs, and feet. Selenium did not affect the onset or frequency of egg laying, egg size, shell thickness, fertility of eggs, or sex ratio of ducklings. Reduced survival and growth occurred in ducklings hatched from groups whose parents had received 8- or 16-ppm selenium as selenomethionine, even though all ducklings were fed a control diet. Concentrations of selenium in eggs and liver of adults could be predicted from dietary concentrations. We conclude that the dietary threshold of selenium as selenomethionine necessary to impair reproduction is between 4 and 8 ppm. It is difficult to identify 1 level of selenium in eggs that will be diagnostic of reproductive impairment in the field because different chemical forms of selenium appear to have different toxicities in eggs. However, when eggs from a wild population contain ≥1-ppm selenium on a wet-weight basis, reproductive impairment may be possible and should be evaluated in that population. At 5-ppm selenium in eggs, reproductive impairment is much more likely to occur.

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Selenium is a trace element in the earth's crust and, in small amounts, is essential to good health in animals. Too much selenium, however, is toxic to animals. Reproductive failure of chickens caused by selenium poisoning was noted in South Dakota as early as the 1930's (Poley and Moxon 1938). Grains grown on seleniferous soils proved to be the source of the toxic concentrations of selenium.

Selenium also can reach toxic levels in aquatic environments when it is leached from agricultural soils into drainage water. Selenium accumulated in evaporation ponds receiving drainage water at the Kesterson National Wildlife Refuge (NWR) in California and was believed to be responsible for severely impaired reproduction in a variety of aquatic birds (Ohlendorf et al. 1986*a*,*b*). Mortality of adult American coots (*Fulica americana*) and other birds was also linked to high dietary levels of selenium (Ohlendorf et al. 1988). Rooted aquatic plants contained a mean of 52-ppm selenium and various invertebrates contained means of 22–175 ppm on a dry-weight basis at Kesterson NWR (Ohlendorf et al. 1986*a*).

Our objectives were to estimate the dietary concentration of selenium and concentration in eggs at which reproductive problems in waterfowl begin to occur and to compare the toxicities of 2 organic forms of selenium. Knowledge of the reproductive effect threshold of selenium in diet and eggs is important because other wetlands in California and other parts of the United States that serve as breeding areas for birds have elevated levels of selenium (Cumbie and Van Horn 1978, Sorensen et al. 1982, Lowe et al. 1985). It is also important to understand the relative toxicities of different forms of selenium because many forms occur in nature.

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METHODS

Game-farm mallards that had previously gone through 1 reproductive season were randomly

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assigned to pens on 4 March 1986. One male was placed randomly in each of 115 outdoor 1-m² elevated wire breeding pens supplied with flowing water and ad libitum duck developer mash (The Beacon Milling Co., Cayuga, N.Y.). Females were randomly assigned to groups held in 7 2.5- × 6.5-m indoor pens. All females in the same pen were later placed on the same dietary treatment. To keep females from laying eggs, a cycle of 8 hours daylight : 16 hours darkness was maintained. Other males and females were kept under the same conditions to replace birds prior to placement on treated diets. Some early replacements were necessary due to cases of bumblefoot, but after selenium treatments started no further substitutions were required.

On 11 March, we switched all birds to their randomly assigned selenium diets. Thirty-five of each sex were fed a control diet and 15 of each sex were fed diets supplemented with 1-, 2-, 4-, or 8-ppm selenium in the form of seleno-DL-methionine (98% pure; Bachem, Inc., Torrance, Calif.; and Sigma Chem. Co., St. Louis, Mo.). Ten pairs were fed 16-ppm selenium as seleno-DL-methionine and 10 pairs were fed 16-ppm selenium as seleno-DL-cystine (90% pure; Sigma Chem. Co., St. Louis, Mo.).

Selenomethionine is believed to be the predominant form of selenium in commercial grains (Olson et al. 1970), although little is known about the chemical forms found in aquatic vegetation. Selenocystine may also occur in nature (Butler and Peterson 1967, Girling 1984), although its existence in plant and animal material has been questioned (Olson et al. 1970, Osman and Latshaw 1976). Selenocystine is, however, similar in chemical makeup to selenocysteine, which has been identified in animal tissue (Beilstein et al. 1981), but is not stable and therefore not commercially available.

We prepared the diets by dissolving the selenium compounds in distilled, deionized water and then mixing these solutions into the duck mash so that 1% water had been added to the diet. To dissolve selenocystine, we used warm water whose pH had been reduced to 2 by the addition of hydrochloric acid. Controls also received 1% water added to their diet. Three samples of each dietary concentration of selenium were saved for selenium analysis. Selenium recoveries in these samples averaged 95%. We mixed diets every week. Samples of feed set aside for 4 weeks showed no decline in selenium content.

We switched all birds after 3 weeks on treated diets to a duck breeder mash diet with the same selenium levels as in the developer mash, and the females were randomly paired with the males in the outdoor 1-m² breeding pens. Developer mash contained about 14.5% protein, 2.75% crude fat, and 7% crude fiber. Breeder mash contained about 17, 3, and 6% of these constituents. Developer and breeder mashes were supplemented by the supplier with 0.1- and 0.2-ppm selenium as sodium selenite.

We fed 3 additional pairs of mallards 4-ppm selenium as selenomethionine. From each female, we saved the first, fourth, seventh, tenth, thirteenth, and sixteenth eggs for selenium analysis to determine if there were any changes in selenium levels over the course of the laying period. All adults were weighed on the day selenium treatments started, when the females were paired with the males, and when each pair was sacrificed.

We collected a minimum of 16 eggs from each female. The eighth egg was saved for eggshell thickness measurements, egg weight, and selenium analysis, and the other 15 were placed in an incubator. Additional eggs were collected to replace any that were cracked. Eggs were labeled according to pen, stored at 10 C, and set each week in an incubator maintained at 37.6 C and at a relative humidity of 60–68%. Eggs were candled each week to check for fertility and embryo mortality. All nonviable eggs were opened to confirm that they were infertile or to examine dead embryos for malformations. We saved the seventeenth egg from 4 females fed 16-ppm selenium as selenomethionine and 4 fed 16-ppm selenium as selenocystine. The yolk was separated from the white and each portion was analyzed separately for selenium. In chickens, selenomethionine causes more selenium to be deposited in white than yolk (Latshaw and Osman 1975); we wanted to compare this deposition pattern with that of selenocystine.

We sacrificed each female and her mate after all the required eggs were collected. We saved a sample of liver for selenium analysis from 5 randomly chosen females and their mates in each treatment.

Hatchlings were weighed, banded, and transferred to 2.5- × 6.5-m pens where they were reared for 6 days on a diet of duck starter mash containing no added selenium. We monitored the survival of ducklings and determined the

sex of all ducklings after they were sacrificed on day 6.

Analysis of variance was used to test for differences among treatments for adult weight, number of days on treatment until the first egg was laid, mean number of days between eggs, egg weight, eggshell thickness, percent fertility of eggs, percent hatch of fertile eggs, percent survival of hatchlings to 6 days of age, number of 6-day-old ducklings produced, duckling weights at hatching and 6 days of age, and percent ducklings that were males. When an analysis of variance was significant at $P < 0.05$, Dunnett's 1-tailed procedure (Dunnett 1955) was used to separate the control mean from other group means, also at $P < 0.05$. Percentage data were normalized by an angular transformation before statistical analysis.

For eggs that failed to hatch, the proportion of malformed embryos for controls was compared to the proportions in each selenium treatment by Chi-square tests. We defined malformed embryos as those with ≥ 1 external, skeletal, or soft tissue structural defect. Embryotoxic effects such as edema and stunted growth were not classified as malformations.

The concentration of selenium in the liver of females was compared to the concentration in males fed the same diet by 1-tailed t-tests ($P < 0.05$). We used 1-tailed paired t-tests ($P < 0.05$) to compare the concentrations of selenium in yolk and white of the seventeenth eggs from females fed 16-ppm selenium as selenomethionine or selenocystine. Based on the separate concentrations of selenium in the yolk and white of these seventeenth eggs and the ratios of yolk to white, we calculated whole egg ppm of selenium. The whole egg concentration of selenium in seventeenth eggs was compared to the concentration in the eighth eggs from the same females using a 2-tailed paired t-test.

We used correlation analyses to look for relationships between selenium levels in the eighth eggs and days until egg laying began or number of 6-day-old ducklings produced. Regression equations were derived to explain relationships between dietary concentrations of selenium and concentrations of selenium in tissues, and selenium in the liver of adult females and their eighth eggs.

Selenium was measured by the method of Krynitsky (1987). A 0.5-g (wet wt) subsample of tissue was digested for 2 hours in a nitric acid/hydrogen peroxide solution in a polyethylene tube, partially immersed in a hot water bath at 95 C. The digested samples were then quantified directly, using peak area integration, by graphite furnace atomic absorption spectrometry in combination with Zeeman background correction (Slavin et al. 1983, Welz et al. 1983). The lower limit of detection on a wet-weight basis was 0.05-ppm selenium. The mean percent recovery on spiked tissue and National Bureau of Standards standard reference materials was 97%.

Selenium concentrations in feed, eggs, and liver samples are reported on a wet-weight basis. Feed contained about 10% water; eggs and liver contained approximately 70% moisture. Selenium concentrations cited from the literature are also on a wet-weight basis unless specified as dry weight.

RESULTS

No mortality or signs of selenium intoxication occurred among adults during the study. Adults fed the 2 forms of selenium at 16 ppm did not appear to accept these diets as well as other dietary treatments; they moistened their food more which, under our pen conditions, seems to be related to food aversion. When the pairs were formed (3 weeks after treatments started), females fed either of the 16-ppm selenium diets weighed significantly less than did controls (Table 1).

After 100 days on treatment, 1 control female had laid no eggs and 1 female fed 16-ppm selenium as selenomethionine had laid only 13 eggs. Both females and their mates were killed and necropsied. No obvious lesions were found in the control, but the female fed 16-ppm selenium had yolk peritonitis. Two other controls and 1 female fed 16-ppm selenium as selenocystine produced eggs, but none were fertile. In the statistical analysis of fertility, hatchability, survival of young, and number of young produced, these 5 females were eliminated from the study.

No statistically significant differences were noted between controls and selenium-treated females in the number of days on treatment until the first egg was laid or in the average interval of days between successive eggs (Table 2). Fertility of eggs was unaffected by selenium treatment, but 16-ppm selenium as selenomethionine caused a severe reduction in hatching success of

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Table 1. Mean weights of adult mallards fed a control diet or diets containing 1-, 2-, 4-, 8-, or 16-ppm selenium as selenomethionine, or 16-ppm selenium as selenocystine.

		Wt (g)											
		M						F					
		Onset of treatment		When pairs were formed		At sacrifice		Onset of treatment		When pairs were formed		At sacrifice	
Selenium added to diet (ppm)	n	$\bar{x}$	SE	$\bar{x}$	SE	$\bar{x}$	SE	$\bar{x}$	SE	$\bar{x}$	SE	$\bar{x}$	SE
0	35	1,181	22.4	1,219	19.9	1,155	16.0	1,046	37.9	1,053	17.1	1,224	22.5
1	15	1,124	36.5	1,188	34.1	1,085	24.4	1,019	30.4	1,021	24.9	1,225	32.8
2	15	1,183	25.7	1,235	30.4	1,193	35.4	1,001	22.3	1,035	27.6	1,183	28.3
4	15	1,195	27.2	1,227	28.7	1,200	34.5	1,036	22.1	1,077	24.4	1,212	32.1
8	15	1,218	36.7	1,199	33.8	1,191	41.0	1,020	27.8	1,012	26.6	1,225	46.0
16	10	1,196	30.6	1,184	30.6	1,082	26.4	958	27.1	918[a]	33.3	1,100	49.4
16 (selenocystine)	10	1,212	49.1	1,202	45.3	1,129	54.2	985	33.3	938[a]	22.4	1,172	55.1

[a] Significantly lower than controls at $P < 0.01$ by Dunnett's procedure (Dunnett 1955).

fertile eggs. Fewer eggs hatched from pairs fed 8-ppm selenium; however, this decrease was not statistically significant.

In eggs that did not hatch, the percentages of malformed embryos for controls, mallards fed 1-, 2-, 4-, 8-, or 16-ppm selenium as selenomethionine, and 16-ppm selenium as selenocystine were 0.6, 0.9 0.5, 1.4, 6.8, 67.9, and 2.3%, respectively. The diets containing 8- and 16-ppm selenium as selenomethionine produced a significantly greater percentage of malformed embryos than did the control diet.

All 10 females fed 16-ppm selenium as selenomethionine produced ≥1 unhatched egg containing a malformed embryo. Individual embryos often had multiple malformations. The most common malformations were microphthalmia (abnormally small eyes); hydrocephaly; bill defects such as a reduced or absent mandible or spoon-shaped upper bill; twisted legs and feet, often with ectrodactyly (≥1 missing toe); and frequently micromelia (small legs). Wing malformations were less common.

In the group fed 8-ppm selenium as selenomethionine, 7 of 15 females produced ≥1 unhatched egg containing a malformed embryo. Malformations in embryos of females fed 8-ppm selenium were similar to those seen in the 16-ppm selenium group, but were generally less severe and less often multiple. The malformations included microphthalmia, shortened mandible, and absence or severe reduction in the size of the hind toe. Hydrocephaly and twisted feet occurred occasionally. Three of 32 control females produced ≥1 malformed embryo.

Females fed the diet containing 16-ppm selenium in the form of selenocystine did not produce a significantly greater percentage of deformed embryos than did controls, and the small number of malformations were generally unlike those caused by selenomethionine.

All deformed embryos failed to hatch; no hatchlings in any treatment had any obvious external deformities. None of the ducklings hatched from females fed 16-ppm selenium as selenomethionine survived to 6 days. Among ducklings from parents fed 8-ppm selenium, 95% of all mortality occurred within 3 days of hatching. All mortality of ducklings from parents fed 16-ppm selenium occurred within 3 days of hatching.

Egg weight and eggshell thickness were not affected by selenium treatments. Hatchling sex ratio (51.8% M:48.2% F across all treatments) was unaffected by selenium treatment. Although 8- and 16-ppm selenium as selenomethionine produced the lowest duckling weights at hatching, these weights were not significantly lower than that of controls (Table 3). At 6 days of age, ducklings from parents fed 8-ppm selenium weighed significantly less than did controls (Table 3). All ducklings in the 16-ppm treatment died before 6 days of age.

Concentrations of selenium in eggs and liver of adults increased with increases in dietary concentrations of selenomethionine (Table 4). Regressions of dietary concentrations of selenium versus levels of selenium in eggs, liver of adult females, and liver of adult males were all statistically significant. The regression equations and r^2 values, where y is the wet-weight concentration of selenium in egg, liver of females,

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Table 2. Mean reproductive success of mallards fed a control diet or diets containing 1-, 2-, 4-, 8-, or 16-ppm selenium as selenomethionine, or 16-ppm selenium as selenocystine.

Measurement	ppm selenium added to diet											
	0		1		2		4		8		16		16 (selenocystine)	
	$\bar{x}$ (95% CL)	SE	$\bar{x}$ (95% CL)	SE	$\bar{x}$ (95% CL)	SE	$\bar{x}$ (95% CL)	SE	$\bar{x}$ (95% CL)	SE	$\bar{x}$ (95% CL)	SE	$\bar{x}$ (95% CL)	SE
Days on treatment until first egg	46.5	2.38	39.8	1.78	45.7	3.89	46.1	3.17	45.6	3.02	52.3	5.41	48.7	4.20
Days between eggs	1.2	0.04	1.1	0.05	1.3	0.12	1.1	0.04	1.1	0.03	1.2	0.05	1.3	0.14
% fertility of eggs[a]	98.6 (96.5–99.6)		96.3 (89.1–99.8)		97.0 (90.9–99.8)		97.9 (93.7–99.9)		96.6 (89.3–99.9)		99.6 (99.1–100)		98.7 (95.1–100)	
% hatch of fertile eggs[a]	59.6 (48.7–70.1)		70.7 (44.8–91.0)		60.0 (41.6–77.1)		53.4 (35.8–70.7)		36.9 (13.8–63.8)		2.2[b] (0–10.7)		50.3 (13.1–87.3)	
% survival of ducklings to 6 days of age[c]	99.4 (98.3–100)		97.7 (90.8–100)		98.9 (94.9–100)		99.7 (98.3–100)		80.6[b] (60.1–94.9)		0[b] (0–0)		98.0 (90.9–100)	
N 6-day-old ducklings produced/hen[a]	8.1	0.67	8.5	1.32	8.2	0.97	7.5	0.91	4.6[d]	1.04	0.0[b]	0.00	6.6	1.70
Pairs of breeders	34		15		15		15		15		10		10	

[a] *n* was reduced to 32 for controls, 9 for mallards fed 16-ppm selenium as selenomethionine, and 9 for mallards fed 16-ppm selenium as selenocystine. These reductions reflect the fact that 1 F did not lay eggs, 1 F had yolk peritonitis, and 3 F produced no fertile eggs and were not included in the analysis.

[b] Significantly lower than controls at $P < 0.01$ by Dunnett's procedure (Dunnett 1955).

[c] *n* was reduced to 32 for controls, 14 for mallards fed 1-, 2-, or 4-ppm selenium; 11 for mallards fed 8-ppm selenium, 3 for mallards fed 16-ppm selenium as selenomethionine, and 8 for mallards fed 16-ppm selenium as selenocystine. These reductions reflect the no. mallards that hatched 0 young and for whom no percentage survival could be calculated.

[d] Significantly lower than controls at $P < 0.05$ by Dunnett's procedure (Dunnett 1955).

Table 3. Mean weights of mallard ducklings from parents fed a control diet or diets containing 1-, 2-, 4-, 8-, or 16-ppm selenium as selenomethionine, or 16-ppm selenium as selenocystine.

Selenium added to diet (ppm)	n[a]	Wt (g) Hatching $\bar{x}$	Hatching SE	6 days old $\bar{x}$	6 days old SE
0	32	33	0.6	72	2.0
1	14	32	1.0	69	3.8
2	14	33	0.8	71	3.3
4	14	31	0.7	69	2.5
8	11	30	0.6	58[b]	4.0
16	3	29	3.0		
16 (selenocystine)	8	32	1.6	67	5.4

[a] The no. F with ducklings represented. A $\bar{x}$ wt for all ducklings from the same F was calculated; these means were then averaged to produce the $\bar{x}$'s presented in this table. No ducklings from parents fed 16-ppm selenium as selenomethionine survived to 6 days of age.

[b] Significantly lower than controls at $P < 0.01$ by Dunnett's procedure (Dunnett 1955).

or liver of males, and x is the dietary concentation of selenium, where $y = -0.32 + 1.18\ x$, $r^2 = 0.98$; $y = 0.35 + 0.43\ x$, $r^2 = 0.99$; and $y = -1.20 + 1.70\ x$, $r^2 = 0.96$, respectively.

The dietary treatment of 16-ppm selenium in the form of selenocystine resulted in lower concentrations of selenium in eggs and liver of adults than did 16 ppm as selenomethionine. At a dietary concentration of 16-ppm selenium as selenocystine, less selenium accumulated in eggs than when 1-ppm selenium as selenomethionine was fed.

The selenium concentration was greater ($P < 0.05$) in egg white than in egg yolk when selenomethionine was fed at 16-ppm selenium (8.7 ± 0.92 [SE] vs. 6.4 + 0.46 ppm) but, with selenocystine, there was no difference between the mean selenium concentration in white and yolk (1.1 ± 0.26 vs. 1.2 ± 0.15 ppm).

The correlation between selenium concentration in eighth eggs and number of 6-day-old ducklings produced was not statistically significant for females fed 8-ppm selenium ($r = -0.36$, $P > 0.05$). At the dietary level of 16-ppm selenium as selenomethionine, ducklings did not survive to 6 days of age, and a correlation coefficient could not be calculated.

Selenium concentrations changed little from the first to the sixteenth eggs of the extra 3 females fed 4-ppm selenium (Fig. 1). A regression of selenium in egg versus egg number was not significant ($P > 0.05$). However, it appeared that selenium in eggs of females fed 16-ppm selenium in either form changed over time. For selenomethionine, the mean selenium concentration was 17 ± 4.0 ppm for 4 eighth eggs, which was almost significantly greater ($P = 0.08$, 2-tailed paired t-test) than 7.8 ± 0.60 ppm in the seventeenth eggs of the same 4 females. For selenocystine, however, eighth eggs (0.59 ± 0.095 ppm) contained significantly less than seventeenth eggs (1.2 ± 0.21 ppm).

No statistically significant correlations were noted in any dietary treatment between selenium concentration in the eighth egg and days on treatment until the first egg was laid. A significant relationship existed between the concentration of selenium in the liver of adult females and the concentration in their eighth eggs (Fig. 2).

Table 4. Mean selenium concentrations in eggs and liver of mallards fed a control diet or diets containing 1-, 2-, 4-, 8-, or 16-ppm selenium as selenomethionine, or 16-ppm selenium as selenocystine.

Selenium added to diet (ppm)	Selenium (ppm, wet wt) Egg n[a]	Egg $\bar{x}$	Egg SE	Liver n[b]	Liver F $\bar{x}$	Liver F SE	Liver M $\bar{x}$	Liver M SE
0	5	0.18	0.021	5	0.57	0.039	1.2[c]	0.08
1	15	0.83	0.062	5	0.64	0.078	1.4[c]	0.17
2	15	1.6	0.09	5	1.1	0.15	2.2[c]	0.21
4	15	3.4	0.18	5	2.4	0.39	4.0	0.93
8	15	11	0.7	5	3.5	0.25	9.1[c]	2.57
16	10	18	1.7	5	7.3	0.37	28[c]	2.3
16 (selenocystine)	10	0.57	0.049	5	1.6	0.24	6.9[c]	2.49

[a] The eighth egg from each F was analyzed.

[b] Selenium levels in the livers of 5 randomly selected F were compared to levels in 5 M from the same pens. At a dietary concentration of 1-ppm selenium, $n = 4$ for M.

[c] Significantly higher than F at $P < 0.05$ by a 1-tailed t-test.

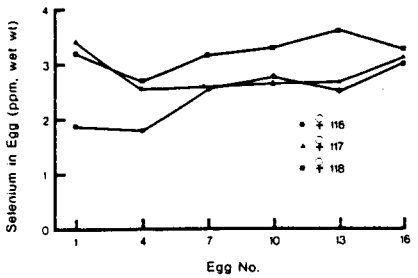


Fig. 1. Selenium concentrations in the first, fourth, seventh, tenth, thirteenth, and sixteenth eggs laid by 3 mallards fed a continuous diet of 4-ppm selenium in the form of selenomethionine.

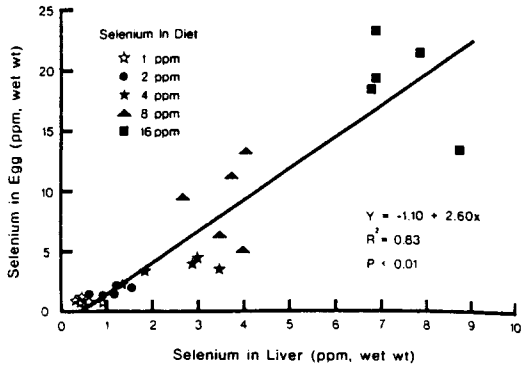


Fig. 2. Relationship between the concentration of selenium in the liver of female mallards and the concentration in their eighth eggs for females fed diets containing 1-, 2-, 4-, 8-, or 16-ppm selenium as selenomethionine.

DISCUSSION

Condition of Adults

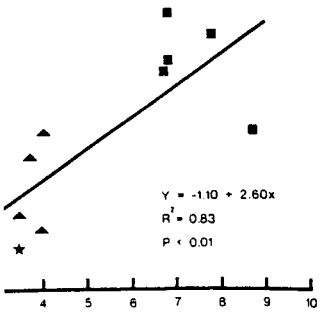
Neither form of selenium appeared to adversely affect adult mallards, apart from some food avoidance of diets containing 16-ppm selenium as selenomethionine or selenocystine and a temporary weight loss in females fed these diets. In another study (Heinz et al. 1988), a dietary concentration of 20-ppm selenium as selenomethionine reduced mallard duckling growth, but mortality did not occur until the diet contained 40-ppm selenium. When adult mallards were fed sodium selenite, mortality began at a dietary concentration of 25-ppm selenium and reached almost 100% at 100-ppm selenium (Heinz et al. 1987). Jensen (1975) reported 40% mortality from hatching to 2 weeks of age in chickens fed 40-ppm selenium as sodium selenite. Recently collected data show that mortality of adult mallards can occur at a dietary concentration of selenomethionine as low as 15-ppm selenium, but exposure was for a prolonged period over winter (G. H. Heinz, unpubl. data).

In 1984 at the Kesterson NWR in California, adult American coots and other aquatic birds died from selenium poisoning (Ohlendorf et al. 1988). Although the chemical forms of selenium and dietary concentrations ingested by these coots and other birds at Kesterson NWR were unknown, mean selenium concentrations in 1983 on a dry-weight basis were 22–175 ppm in various insects, 170 ppm in mosquitofish (*Gambusia affinis*), 35 ppm for algae, 52 ppm for rooted aquatic plants, and 85 ppm for net plankton (Ohlendorf et al. 1986*a*).

Reproduction

Selenomethionine at dietary concentrations of 8- and 16-ppm selenium impaired mallard reproduction, but 16-ppm selenium as selenocystine had no harmful effects. Selenomethionine, thus far, has proven to be the most harmful chemical form of selenium when mallard reproduction is measured. In an earlier study, selenomethionine at a dietary concentration of 10-ppm selenium reduced the number of mallard ducklings produced by about 80% compared to controls, whereas duckling production was not significantly reduced by 10-ppm selenium as sodium selenite (Heinz et al. 1987). When injected into the air cells of embryonated chicken eggs, the lethal dose for 50% of the population (LD_{50}'s) of selenomethionine, sodium selenite, and selenocystine were 0.13-, 0.30-, and 0.64-ppm selenium in the egg (Palmer et al. 1973).

Selenocystine and selenomethionine are both organic forms of selenium in which selenium is substituting for sulfur in the amino acids cystine and methionine; however, selenocystine was not nearly as toxic as selenomethionine. Latshaw and Osman (1975) suggested that selenocystine is metabolized to an inorganic selenium compound, whereas selenomethionine is incorporated into proteins in place of methionine. Ochoa-Solano and Gitler (1968) found that selenomethionine injected into the wing vein of chickens later showed up as selenomethionine in their egg white proteins. This metabolic difference between the 2 selenium-containing ami-



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no acids may be a reason for much of their differences in toxicity.

An increase in teratogenic effects above controls was seen only in embryos produced by females fed 8- and 16-ppm selenium as selenomethionine. The defects we observed were similar to those described in aquatic birds nesting at the Kesterson NWR in California (Ohlendorf et al. 1986*a*). In an earlier study (Heinz et al. 1987), a diet containing 10-ppm selenium as selenomethionine also caused deformities of eyes, beak, head, legs, and toes. When seleniferous grain was fed to chickens at a dietary concentration of 15-ppm selenium, embryos exhibited hydrocephaly, twisted necks and legs, missing toes, missing eyes, deformed beaks, and poorly developed wings (Poley et al. 1937). Approximately 50% of the selenium in wheat seeds was selenomethionine (Olson et al. 1970), which suggests that the teratogenic effects reported by others for chickens fed seleniferous grain were due, at least in part, to selenomethionine.

Teratogenic effects and embryo mortality are most pronounced when selenomethionine is the form of selenium fed to birds. Sodium selenite has also been reported to be teratogenic to chickens (Franke et al. 1936). At dietary concentrations of 10- and 25-ppm selenium, sodium selenite produced a few teratogenic effects in mallards, but not as many as were caused by selenomethionine (Heinz et al. 1987). Palmer et al. (1973) injected 8 selenium compounds including selenomethionine, selenocystine, and sodium selenite into chicken eggs and reported that selenomethionine was especially capable of producing abnormal embryos.

Embryo development and survival appear to be the most sensitive measures of selenomethionine poisoning in mallards. We found no effects, even at 16-ppm selenium in the diet, of selenomethionine on the date of initiation or frequency of egg laying, egg weight or eggshell thickness, fertility of eggs, or sex ratio of hatchlings. Survival and growth of hatchlings were reduced by 8- and 16-ppm selenium but, because the young were fed a control diet, even these effects must have been ones lingering from embryonic exposure to selenomethionine. Studies with chickens also suggest that effects on embryos are the most sensitive measures of selenomethionine poisoning in birds (Poley et al. 1937, Poley and Moxon 1938).

The threshold concentration of selenomethionine that causes reproductive impairment in mallards appears to lie somewhere between 4- and 8-ppm selenium on what is close to a dry-weight basis in the diet. The commercial mashes we fed our mallards contained about 10% water. In determining whether the natural foods of ducks from a particular area contain potentially harmful concentrations of selenium, one would have to take into account the water content of the food items.

There were no apparent adverse effects at a dietary concentration of 4-ppm selenium, but the diet containing 8-ppm selenium resulted in a mean decrease of 43% in the number of 6-day-old ducklings compared to controls. In an earlier study with mallards, a diet containing 10-ppm selenium as selenomethionine caused nearly an 80% reduction in production of ducklings (Heinz et al. 1987). Poley and Moxon (1938) fed chickens a diet containing 10-ppm selenium from seleniferous grains and reported that hatching success of fertile eggs decreased to zero after 4 weeks; hatching "appeared to be slightly reduced" when the diet contained 5-ppm selenium. Ort and Latshaw (1978) reported 5-ppm selenium as sodium selenite to be "borderline toxic" when hatching success of fertile chicken eggs was measured.

Selenium in Eggs and Adults

Our results indicate that it is possible, at least in a laboratory study, to predict the levels of selenium in mallard eggs and liver of adults if the dietary concentration of selenium as selenomethionine is known. Moksnes (1983) fed chickens diets supplemented with selenomethionine at levels from zero to 6.0-ppm selenium and reported highly significant correlations between dietary concentration of selenium on a dry-weight basis and selenium concentrations in eggs and female liver on a wet-weight basis. For the chicken eggs and liver, the regression equations were $y = 0.27 + 0.81\ x$ and $y = 0.50 + 0.99\ x$, respectively. For eggs, the slope is similar to that for our study of mallards (1.18), but for liver of females the slope for chickens is greater than for mallards (0.43). The range of dietary concentrations for the chickens did not go as high as for our mallards; this may be 1 reason for the difference, but there may also be species differences. We do not know how applicable these predictions would be in the field where dietary intake of selenium would not be constant and

where other factors could affect accumulation of selenium.

With mallards on constant selenium treatment in our laboratory study, we were able to predict selenium concentration in egg if the concentration in the laying female was known. How well the predictive equation (Fig. 2) would apply to wild mallards is unknown.

In the field it is often easier to collect breeding adults, and especially eggs, than representative food items for contaminant analyses. For this reason, it is helpful to know how much of a chemical in adults or eggs is associated with reproductive impairment. In our study, a dietary concentration of 4-ppm selenium as selenomethionine caused no detectable decrease in mallard reproduction; the mean concentration of selenium in eggs and in liver of adult females was 3.4 and 2.4 ppm, respectively. At a dietary concentration of 8-ppm selenium, mallard reproduction was impaired and eggs and female liver contained 11- and 3.5-ppm selenium, respectively. Based on these results, we can infer that the threshold level of selenium, from selenomethionine, associated with reproductive impairment in mallards fell somewhere between 3.4 and 11 ppm in eggs and 2.4 and 3.5 ppm in the liver of females. The values for eggs are probably more useful because our work and that of others provides strong evidence that it is the selenium in the egg which harms the embryo and consequently impacts reproduction.

In an earlier study (Heinz et al. 1987), reproductive impairment in mallards fed 10-ppm selenium as selenomethionine was associated with a mean of 4.6-ppm selenium in eggs and 4.7 ppm in liver of females. When chickens were fed 10-ppm selenium from seleniferous grains, reduction of reproductive success was associated with a mean of 6.40-ppm selenium in egg white and 3.92 ppm in egg yolk (Moxon and Poley 1938, Poley and Moxon 1938).

In a field study, Ohlendorf et al. (1986*b*) discovered severe reproductive impairment in American coots, black-necked stilts (*Himantopus mexicanus*), and eared grebes (*Podiceps nigricollis*) nesting at Kesterson NWR in California. In 1983, eggs from coots, stilts, and grebes contained means of about 8.3-, 8.5-, and 16.7-ppm selenium, respectively, on a wet-weight basis. A 1984 sample of stilt eggs contained 7.2-ppm selenium. There was an estimated 50% chance of embryo death or deformity in coots when selenium reached about 4.9 ppm (wet wt) in eggs. For stilt eggs, the 50% level was about 7.1-ppm selenium (wet wt). A 50% level could not be calculated for grebes because even the lowest selenium concentration in eggs (about 10.6 ppm, wet wt) was embryotoxic.

Ort and Latshaw (1978) reported 7-ppm selenium as sodium selenite to impair hatching success of chicken eggs. This diet resulted in a mean of 0.87- and 2.02-ppm selenium in egg white and yolk, respectively, after 16 weeks on treatment.

In another study with chickens, 8-ppm dietary selenium as sodium selenite impaired hatching success and resulted in from 1.46- to 1.86-ppm selenium on a wet-weight basis in whole eggs (Arnold et al. 1973). These studies with chickens fed sodium selenite suggest that reproductive impairment may occur at lower levels of selenium in the egg than when selenomethionine is fed. Mallards fed 25-ppm selenium as sodium selenite produced eggs containing only 1.3-ppm selenium, yet reproduction was significantly impaired (Heinz et al. 1987). This suggests that when selenium in the egg is the result of feeding sodium selenite the concentration in eggs may not have to reach as high a level to harm reproduction as when selenomethionine is fed. The other important point, however, is that high dietary levels of sodium selenite are required to produce these effect levels in eggs. These results are at odds with those from the egg injection study of Palmer et al. (1973), in which less injected selenomethionine was needed to kill embryos than injected sodium selenite. One explanation may be that biologically incorporated selenium compounds have different toxicities than the same compounds injected into eggs. In addition, there could be changes in a selenium compound by the female before she incorporates it into her eggs.

Based on studies that relate to the metabolism of sodium selenite and selenomethionine in chickens, the form of selenium in the egg may not be the same when sodium selenite is fed as when selenomethionine is fed (Latshaw 1975, Latshaw and Osman 1975). Until more is known about the various forms of selenium that may be present in waterfowl foods, the significance of the differences between effect levels for sodium selenite versus selenomethionine will remain unclear and the interpretation of selenium levels in the eggs of wild waterfowl should be made with caution.

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Our results with mallards illustrate the difficulty in setting a single threshold of selenium in eggs that is associated with the start of reproductive impairment. For mallards fed 8-ppm selenium, we were unable to show a statistically significant correlation between selenium concentration in the eighth eggs and reproductive success. Although there appeared to be a difference between level of selenium in a small sample of eighth eggs and seventeenth eggs of females fed 16-ppm selenium, a larger sample of eggs from females fed 4-ppm selenium suggested little change in selenium concentrations within eggs of the same clutch. One might expect, therefore, that a female with a higher level of selenium in her eighth egg would have poorer hatching success of her remaining eggs than one fed the same diet but who deposited less selenium in her eggs. The fact that this was not the case shows that, although a range of selenium concentrations in eggs was associated with reproductive problems, there must be considerable variability in the sensitivity of embryos to selenium.

When we put together our results for mallards with the results of others for chickens we cannot set a simple, single threshold of selenium in eggs above which reproductive impairment will definitely occur. However, we can say that when eggs of wild birds contain wet-weight concentrations of selenium >1 ppm, one should consider the possibility of reproductive impairment and look closely at the reproductive success of birds in that area. The 1-ppm value in eggs is based on the possibility that the 1-ppm selenium could have resulted from a bird eating high levels of sodium selenite in nature; this is a possibility that we cannot rule out. As selenium in eggs approaches 5 ppm, even if derived solely from foods containing selenomethionine, it is much more likely that reproductive problems are occurring. Before we can refine these rough estimates, more needs to be learned about the chemical forms of selenium in foods and eggs of birds and species differences in sensitivity to selenium.

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MALLARD SURVIVAL FROM LOCAL TO IMMATURE STAGE IN SOUTHWESTERN SASKATCHEWAN

JAY B. HESTBECK, U.S. Fish and Wildlife Service, Patuxent Wildlife Research Center, Laurel, MD 20708
ALEXANDER DZUBIN, Canadian Wildlife Service, Prairie Migratory Bird Research Center, Saskatoon, SK S7N 0X4, Canada
J. BERNARD GOLLOP, Canadian Wildlife Service, Prairie Migratory Bird Research Center, Saskatoon, SK S7N 0X4, Canada
JAMES D. NICHOLS, U.S. Fish and Wildlife Service, Patuxent Wildlife Research Center, Laurel, MD 20708

Abstract: We used 3,670 recoveries from 32,647 bandings of mallards (*Anas platyrhynchos*) in southwestern Saskatchewan during 1956–59 to estimate the probability of surviving from the local, flightless (classes II and III) stage to the flighted, immature stage. The probability of surviving from the local to the immature stage was 0.84 ± 0.05 ($\widehat{SE}$) for males and females. The geographic distribution of direct recoveries was similar for the birds banded as local and immature. Probabilities of survival for banded mallards can only be estimated from late summer to late summer. The estimate of survival from local to immature stage fills a gap in our knowledge of mallard mortality from female–brood breakup to the time of banding in late summer.

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Very little is known about survival of young mallards prior to the initiation of banding in late summer (Anderson 1975:25). Young mallards may experience substantial mortality, most of which occurs before 3 weeks of age (Dzubin and Gollop 1972, Ball et al. 1975, Talent et al. 1983). For example, the probability of mallard ducklings surviving from hatch to female–brood breakup was 44% in north-central Minnesota (Ball et al. 1975) and 35% in south-central North Dakota (Talent et al. 1983). The female–brood breakup generally occurs at about 7 weeks of age in early to midsummer (Ball et al. 1975, Talent et al. 1983).

Probabilities of survival for banded birds can only be estimated from banding date to banding date (Brownie et al. 1985). Because most mallard bandings occur in late summer, survival estimates apply from late summer to late summer. Estimates of the probability of surviving between the later stages of brood-rearing and the time of banding flighted young do not exist. We present estimates of this probability for 1 population.

We acknowledge the efforts of all individuals who banded mallards from 1956–59 in southwestern Saskatchewan and acknowledge the assistance of the U.S. Fish and Wildlife Service (USFWS) Bird Banding Laboratory in maintaining the banding and recovery records used in the analysis. Programming assistance was provided by J. E. Hines. Comments on earlier drafts