

SELECTED TRACE ELEMENTS AND ORGANOCHLORINES: SOME FINDINGS IN BLOOD AND EGGS OF NESTING COMMON EIDERS (*SOMATERIA MOLLISSIMA*) FROM FINLAND

J. CHRISTIAN FRANSON,*† TUULA HOLLMÉN,‡ ROBERT H. POPPENG,§ MARTTI HARIO,|| MIKAEL KILPI,# and MILTON R. SMITH†

†U.S. Geological Survey, National Wildlife Health Center, 6006 Schroeder Road, Madison, Wisconsin 53711

‡Department of Basic Veterinary Sciences, P.O. Box 57, FIN-00014 Helsinki University, Finland

§University of Pennsylvania, School of Veterinary Medicine, Kennett Square, Pennsylvania 19348, USA

||Finnish Game and Fisheries Research Institute, P.O. Box 6, FIN-00721 Helsinki, Finland

#Department of Ecology and Systematics, P.O. Box 17, FIN-00014 Helsinki University, Finland

(Received 19 May 1999; Accepted 27 August 1999)

Abstract—In 1997 and 1998, we collected blood samples from nesting adult female common eiders (*Somateria mollissima*) at five locations in the Baltic Sea near coastal Finland and analyzed them for lead, selenium, mercury, and arsenic. Eggs were collected from three locations in 1997 for analysis of selenium, mercury, arsenic, and 17 organochlorines (OCs). Mean blood lead concentrations varied by location and year and ranged from 0.02 ppm (residues in blood on wet weight basis) to 0.12 ppm, although one bird had 14.2 ppm lead in its blood. Lead residues in the blood of eiders were positively correlated with the stage of incubation, and lead inhibited the activity of the enzyme delta-aminolevulinic acid dehydratase (ALAD) in the blood. Selenium concentrations in eider blood varied by location, with means of 1.26 to 2.86 ppm. Median residues of selenium and mercury in eider eggs were 0.55 and 0.10 ppm (residues in eggs on fresh weight basis), respectively, and concentrations of both selenium and mercury in eggs were correlated with those in blood. Median concentrations of *p,p'*-dichlorodiphenyldichloroethylene in eggs ranged from 13.1 to 29.6 ppb, but all other OCs were below detection limits. The residues of contaminants that we found in eggs were below concentrations generally considered to affect avian reproduction. The negative correlation of ALAD activity with blood lead concentrations is evidence of an adverse physiological effect of lead exposure in this population.

Keywords—Common eider Lead Mercury Organochlorines Selenium

INTRODUCTION

In some of their nesting areas in the Finnish archipelago in the Baltic Sea, the number of breeding pairs of common eiders (*Somateria mollissima*) has declined by 50% since the mid-1980s and duckling survival is as low as 1 to 5% [1]. Predation and parasitism are known to be significant causes of mortality for eider ducklings [2], but the results of several studies in the Finnish archipelago suggest that these factors alone probably do not account for the magnitude of losses there [3–5]. Epizootic mortality and impaired reproduction in wildlife can also be caused by exposure to a variety of environmental contaminants [6,7]. One of the early examples of the accumulation of persistent contaminants in wildlife was the finding of organochlorines (OCs) in eggs of the white-tailed sea eagle (*Haliaeetus albicilla*) from the Baltic Sea [8]. In addition to being very persistent in the environment, OCs continue to accumulate and a recent report estimated that the sedimentation rate of a group of these compounds in the Baltic Sea was 22 tons annually [9].

Trace elements also are of concern in the Baltic because large-scale discharges have resulted in high local and regional concentrations [10], and previous studies have reported evidence of birds being exposed to metals in the Gulf of Finland. In lesser black-backed gulls (*Larus fuscus*), for example, mercury concentrations in feathers grown on their breeding grounds in the Gulf of Finland increased through the 1980s

[11]. Common eiders found dead in the Finnish archipelago had concentrations of up to 119 and 38.5 ppm dry weight of selenium and arsenic, respectively, in their livers [12]. More than 20 ppm wet weight (about 60 ppm dry weight) of selenium in livers of birds may jeopardize their survival [13] and arsenic concentrations of 2 to 10 ppm wet weight (about 6–30 ppm dry weight) in liver are considered elevated [14]. Lead poisoning has been diagnosed in adult eiders from the Finnish archipelago and, in live birds, concentrations of up to 0.63 ppm wet weight of lead and 3.39 ppm wet weight of selenium have been found in blood [12]. As well as their potential for causing acute mortality, lead, selenium, and arsenic all have been associated with sublethal effects in birds, including immunosuppression, impaired reproduction, and growth inhibition [15–17]. Additionally, selenium may cause severe deformities and death of avian embryos [18] and lead is known to inhibit the activity of delta-aminolevulinic acid dehydratase (ALAD), an enzyme required for heme synthesis [19].

Our objectives in the present study were to evaluate exposure of common eiders to selected trace elements by determining the concentrations of lead, mercury, selenium, and arsenic in blood samples of nesting females from five locations in the Baltic Sea. We also analyzed a sample of eggs for selenium, mercury, arsenic, and selected OCs, compared concentrations of selenium and mercury between eggs and blood, evaluated the effects of lead exposure on body weight and ALAD activity in the blood, and examined the relationship between blood lead concentration and stage of incubation.

* To whom correspondence may be addressed
(chris.franson@usgs.gov).

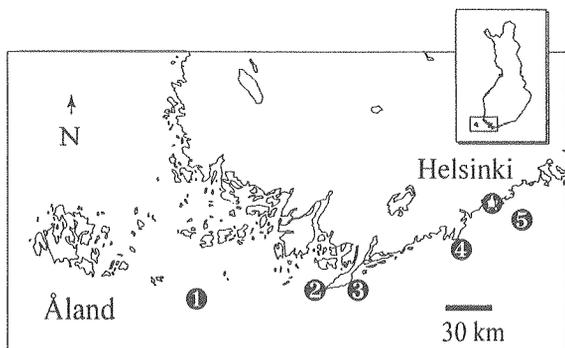


Fig. 1. Locations in the Finnish archipelago in the Baltic Sea near coastal Finland where common eider eggs and blood were collected for contaminants analysis: 1 = Utö, 2 = Hanko West, 3 = Tvärminne, 4 = Rönnskär, and 5 = Söderskär.

MATERIALS AND METHODS

Sample collection

We collected samples at five eider nesting areas in the Finnish archipelago in the Baltic Sea (Fig. 1). In April and May of 1997, we collected the first-laid egg from a total of 21 eider nests at Rönnskär (59°56'N, 24°22'E), Tvärminne (59°50'N, 23°15'E), and Söderskär (60°06'N, 25°25'E). In May and June of 1997 and 1998, we captured incubating female eiders on their nests, weighed them to the nearest 25 g with a spring scale, and collected blood by jugular or ulnar vein venipuncture. The incubation stage was determined by calculating egg density [20] or by floating eggs in water [21]. Blood samples were obtained from a total of 246 females at Hanko West (59°50'N, 22°50'E), Tvärminne, Rönnskär, and Söderskär in 1997 and 1998 and from 22 females at Utö (59°50'N, 21°25'E) in 1998. Blood was placed in plastic tubes with anticoagulant (ethylenediaminetetraacetic acid in 1997 and heparin in 1998) and stored at -20°C and -80°C for analysis of trace elements and ALAD activity, respectively. Eggs were wrapped in aluminum foil and refrigerated until being weighed to the nearest 0.1 g and measured (length and breadth) to the nearest 0.01 mm. Eggs were opened by scoring the shell around the equator with a stainless steel scalpel blade and the contents were frozen (-20°C) in glass jars (I-Chem[®], Nalge Nunc International, Rochester, NY, USA) rinsed with acid and solvent. Eggshells were air-dried for 6 months and the thickness, including the shell and shell membranes, of each was measured to the nearest 0.001 mm. A mean thickness for each eggshell was obtained from the average of five measurements taken around the equator.

Chemical analysis

Homogenized eggs were analyzed for three trace elements (selenium, mercury, and arsenic) and the following OCs: α - and β -benzene hexachloride, aldrin, dieldrin, endrin, lindane, heptachlor, heptachlor epoxide, α -chlordane, oxychlordane, methoxychlor, kelthane, endosulfan I and II, *p,p'*-dichlorodiphenyldichloroethane, *p,p'*-dichlorodiphenyldichloroethylene (DDE), and *p,p'*-dichlorodiphenyltrichloroethane. For selenium and arsenic, 1.0-g aliquots were weighed into individual 100-ml beakers and 10 ml of concentrated nitric acid containing 20% MgNO_3 was added to each beaker. The beakers were placed on a hotplate at low heat until the liquid evaporated and a yellow crust remained. The hotplate temperature was then increased until the sample residue became white. The

beakers were then placed in a muffle furnace at 500°C for 1 h, cooled, and diluted to 25 ml in volumetric flasks with 30% hydrochloric acid. The selenium and arsenic concentrations were determined by hydride generation flame atomic spectrophotometry. All trace element analyses, in both eggs and blood, were done on a GBC 906 (GBC Scientific Equipment, Arlington Heights, IL, USA) atomic absorption spectrophotometer. For mercury, 0.8 to 0.9 g of homogenized egg was weighed into 30-ml Tuftainers[®] (Savillex, Minnetonka, MN, USA) and 2 ml of concentrated sulfuric acid and 1 ml of concentrated nitric acid were added to each container. The containers were capped and placed in an oven at 90°C for 3 h, cooled, uncapped, and a saturated solution of potassium permanganate was added to each container until a purple coloration persisted. The Tuftainers were recapped and returned to the 90°C oven for 30 min, cooled, uncapped, and decolorized with a saturated solution of hydroxylamine hydrochloride, and diluted to 100 ml in volumetric flasks with 30% hydrochloric acid. Mercury concentrations were determined by cold vapor hydride generation atomic absorption spectrophotometry. A 0.200-g sample of DORM-2, certified reference material consisting of dogfish muscle and liver from the National Research Council, Canada, was prepared similarly and analyzed for selenium, mercury, and arsenic with the egg samples.

For analysis of OCs, 1.0 g of homogenized egg was extracted with 6 ml of acetonitrile by vortexing for 15 s, followed by sonicating for 1 h. The samples were centrifuged at 1,500 rpm for 10 min, and pellets were washed with 2 ml of acetonitrile. The combined supernatants were passed through 3 g of basic alumina packed in a 10-ml syringe barrel attached to a Sep-Pac[®] (Waters, Milford, MA, USA) florisil clean-up column, which had been prewashed with 4 ml of methanol, followed by 4 ml of distilled water, and finally 4 ml of acetonitrile, at a flow rate of 2 ml/min. The columns were rinsed with 4 ml acetonitrile and the filtrates were evaporated to 0.5 ml using a nitrogen evaporator with a water bath set at 60°C . The residues were passed through a 0.45- μm filter into autosample vials. One microliter was injected into a Hewlett-Packard 5890 (Hewlett Packard, Wilmington, DE, USA) gas chromatograph, with an HP-5 60-m \times 0.25-mm column using nitrogen as the carrier gas, equipped with an electron capture detector. Standard reference materials for OCs (AccuStandard, New Haven, CT, USA), run with the samples, provided retention times and peak areas that were used to identify and quantitate concentrations in samples. For lipid determination, a 5.0-g sample of homogenized egg was ground with 75 g of Na_2SO_4 and transferred to a glass beaker in a 60°C water bath. The sample was extracted three times with 100 ml of 1:1 petroleum ether:ethyl ether (v/v) and each extract was collected in a beaker with 50 g of Na_2SO_4 . After combining the extracts, the ether solution was decanted off, and the sample was dried to constant weight.

We analyzed each of the 268 blood samples for lead and a subsample of 42 for ALAD. We determined selenium and mercury residues in 247 and 203 of the samples, respectively, and screened about half of them (129) for arsenic. Selenium and mercury levels were determined in blood samples from 15 and 14, respectively, of the same females from which eggs were collected. Concentrations of lead, selenium, and mercury in blood were determined by graphite furnace, flame, and flameless atomic absorption spectrophotometry, respectively [12]. For arsenic analysis, we used the same sample preparation and analytical procedures as previously described for selenium [12]. We analyzed DORM-2 along with the samples

Table 1. Geometric mean concentrations (range, *n*) of lead (ppm wet weight) in blood of common eider hens at five locations in the Gulf of Finland and Baltic Sea proper

Location	1997	1998	1997 + 1998
1. Utö	NS ^a	0.02 ^b (0.01–0.06, 22)	NS
2. Hanko West	0.09 (0.04–1.14, 20)	0.06 (0.03–0.18, 6)	0.08AB ^c (0.03–1.14, 26)
3. Tvärminne	0.07 (0.02–0.52, 35)	0.05 (0.02–0.25, 20)	0.06A (0.02–0.52, 55)
4. Rönnskär	0.11 (0.04–1.28, 20)	0.07 (0.02–0.18, 15)	0.09AB (0.02–1.28, 35)
5. Söderskär	0.11 ^d (0.03–1.56, 83)	0.08 (0.03–0.54, 46)	0.10B (0.03–1.56, 129)
Locations 2–5	0.10 ^e (0.02–1.56, 158)	0.06 ^f (0.02–0.06, 87)	0.09 (0.02–1.56, 245)

^a NS = no samples collected in 1997.

^b Significantly lower (one-way analysis of variance, $p < 0.0001$; Bonferroni multiple comparison test, $p < 0.05$ to $p < 0.001$) than means at each of the four other locations in 1998.

^c Means of the four locations not sharing capital letters in common are significantly different (one-way analysis of variance, $p = 0.0163$; Bonferroni multiple comparison test, $p < 0.05$).

^d One outlier (14.2 ppm lead) was not included in this mean.

^e Significantly greater (two-way analysis of variance, $p = 0.0017$) than the mean for the same locations in 1998.

^f Data from Utö are not included in this mean.

for selenium, mercury, and arsenic. For lead, we used standard reference material from the Wisconsin State Laboratory of Hygiene, Madison, Wisconsin, USA. Minimum detection limits (ppm wet weight) were 0.01 for lead, 0.025 for selenium and mercury, and 0.10 for arsenic in blood and eggs, and 0.01 for OCs (except 0.05 for methoxychlor) in eggs. Residues were not adjusted for recovery from standard reference materials, which were >90% for all analytes. We report trace elements in blood as ppm wet weight of whole blood. Residues in eggs were adjusted to account for moisture loss, using a correction factor of 0.507 [22]. Trace elements are reported in ppm, and OCs in ppb, of fresh wet egg weight. The average moisture and lipid content of the eggs were 67 and 18%, respectively.

The activity of ALAD was measured colorimetrically [23] on 0.1-ml aliquots of heparinized blood with a Beckman DU-65 (Beckman Instruments, Fullerton, CA, USA) spectrophotometer. One unit of enzyme activity is defined as an increase in absorbance at 555 nm of 0.100, with a 1.0-cm light path, per milliliter of erythrocytes per hour, at 38°C.

Statistical analysis

Mercury in blood was not statistically evaluated because it was detected in less than 50% of the samples and the concentrations were very low. When residues of lead and selenium in blood were log-transformed (natural logs) and one outlier (a blood lead concentration of 14.2 ppm) was omitted, the data passed the Kolmogorov–Smirnov test for normality and Bartlett's test for homogeneity of variances [24]. Log-transformed data, excluding the outlier, were used in the statistical analysis of trace elements in blood, and results are reported as retransformed geometric means and ranges. We used two-way analysis of variance to evaluate the effects of location and year on the concentration of lead in the blood of eiders from the four sites where samples were collected in both years. A similar analysis was used for selenium. Separate one-way analyses of variance were used to compare the mean concentrations of lead and selenium in eider blood among the five locations where we sampled in 1998. The Bonferroni multiple comparison procedure [24] was used to identify differences among means. Regression analysis was used to evaluate the relationship between blood ALAD activity and blood lead concentrations. We used logistic regression to compare frequencies of blood lead concentrations of ≥ 0.20 ppm wet weight, a commonly accepted threshold suggestive of lead exposure in waterfowl [19], among locations and years. We evaluated the

effects of incubation stage, and concentrations of lead and selenium in the blood, on body weight using multiple regression with a backward elimination procedure and we calculated Pearson correlation coefficients for comparisons of lead and selenium in blood with incubation stage [24]. Nonparametric statistics were employed for comparisons of residues in eggs, because of the difficulty in evaluating normality of data with small sample sizes. The Kruskal–Wallis test was used to compare the concentrations of *p,p'*-DDE, selenium, and mercury in eggs among locations and pairs of medians were separated with Dunn's test [25]. The relationship between *p,p'*-DDE concentrations in eggs and eggshell thickness was evaluated by calculating the Spearman rank correlation coefficient. We also calculated Spearman rank correlation coefficients for comparisons of selenium and mercury concentrations in eggs versus those in blood from the same birds. Molar concentrations of selenium and mercury in eggs and blood were similarly compared. Contaminant residues in eggs are reported as medians and ranges. We used an α level of 0.05 for all statistical evaluations.

RESULTS

Trace elements and ALAD in blood

Lead was detected in each of the blood samples tested at concentrations of 0.01 to 14.2 ppm wet weight. When trapped in 1997, the bird with 14.2 ppm lead in its blood weighed 1,550 g, about 4% more than the average weight of others at the same stage of incubation. This eider completed incubation and was observed again 7 weeks later. It returned to the nesting area in 1998, but did not nest, and no blood sample was collected that year.

Significant year and location effects, but no year–location interaction, occurred for lead in blood (Table 1). In 1998, the mean lead concentration in the blood of eiders at Utö was lower than the means at each of the other four locations. In 1997, the combined mean blood lead concentration in birds at Söderskär, Tvärminne, Rönnskär, and Hanko West was significantly greater than that in 1998, but no significant year-to-year differences were found at any of the locations (Table 1). Overall results (1997 and 1998 combined) from the same four sites indicated that the mean blood lead concentration at Söderskär was significantly greater than the mean at Tvärminne (Table 1). Thirty-nine (14%) of the birds had blood lead concentrations of ≥ 0.20 ppm. Frequencies of blood lead concen-

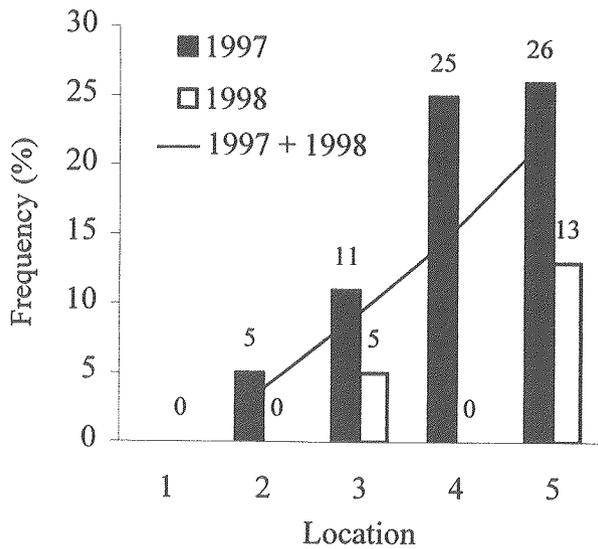


Fig. 2. Frequency (%) of lead concentrations ≥ 0.20 ppm in blood of common eiders at five locations in the Finnish archipelago in the Baltic Sea. Significant differences occurred between years ($\chi^2 = 6.74$, 1 *df*, $p = 0.01$) and among locations ($\chi^2 = 9.77$, 3 *df*, $p = 0.02$), but no year–location interaction was found. At Utö, blood samples were collected only in 1998 and none had lead concentrations of ≥ 0.20 ppm. 1 = Utö, 2 = Hanko West, 3 = Tvärminne, 4 = Rönnskär, 5 = Söderskär.

trations of ≥ 0.20 ppm differed among years and locations, but no year–location interaction was found. The proportion of birds with ≥ 0.20 ppm lead in their blood was greater in 1997 than in 1998 and greater at Söderskär and Rönnskär than at Hanko West, but none of the eiders sampled at Utö had blood lead concentrations of ≥ 0.20 ppm (Fig. 2). Combining results for 1997 and 1998, the frequency of birds with ≥ 0.20 ppm lead in their blood was greater at study sites in the east than those in the western part of the study area (Fig. 2). A negative relationship was found between ALAD activity and lead concentration in eider blood (Fig. 3). The model that best fit the data was $\ln \text{ALAD} = 8.4 - 0.92 \ln \text{blood lead (ppb)}$. The median ALAD activity for birds having blood lead concentrations of ≥ 0.20 ppm was 16 units, whereas the median for birds with < 0.20 ppm blood lead was 113 units. Concentrations of lead and selenium in the blood were removed in the backward elimination multiple regression model, leaving incubation stage as the only variable of the three to significantly ($p < 0.0001$) affect body weight. The concentration of lead in the blood was positively correlated with incubation stage (Pearson $r = 0.53$, $p < 0.0001$).

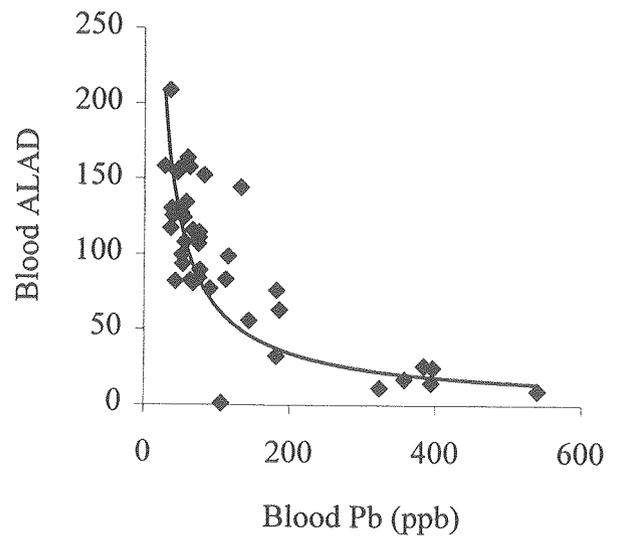


Fig. 3. Relationship between delta-aminolevulinic acid dehydratase (ALAD) activity and lead concentrations (ppb wet weight) in the blood of common eiders. The regression model that best fit the data was $\ln \text{ALAD} = 8.4 - 0.92 \ln \text{blood Pb}$ ($r = -0.69$, $p < 0.0001$, $n = 42$). One unit of ALAD activity is defined as an increase in absorbance at 555 nm of 0.100, with a 1.0-cm light path, per milliliter of erythrocytes per hour, at 38°C.

Selenium also was detected in each blood sample tested, at concentrations from 0.30 to 9.25 ppm wet weight, but selenium was not correlated with incubation stage. Selenium concentrations in the blood of eiders differed by year and location, but no year–location interaction occurred (Table 2). At the four study sites where blood was collected in both years, the combined mean blood selenium concentration was significantly greater in 1997 than in 1998. In combined results from 1997 and 1998, birds from Hanko West and Rönnskär had greater selenium residues in their blood than those sampled at Tvärminne and Söderskär (Table 2). Mercury was detected in 95 of 203 (47%) samples tested and the maximum concentration was 0.31 ppm. Arsenic was not detected in blood from any of the eiders sampled.

OCs and trace elements in eggs

Residues of *p,p'*-DDE were present in each of the 21 eggs and the median concentration of DDE in eggs from eiders at Söderskär was greater than the median in eggs from Rönnskär (Table 3). Eggshell thickness ($\bar{x} = 0.433$ mm, SEM = 0.005, range = 0.375–0.481 mm, $n = 21$) was not correlated with DDE residues. Selenium and mercury also were detected in

Table 2. Geometric mean concentrations (range, *n*) of selenium (ppm wet weight) in blood of common eider hens at five locations in the Gulf of Finland and Baltic Sea proper

Location	1997	1998	1997 + 1998
1. Utö	NS ^a	1.57 (0.67–3.85, 22)	NS
2. Hanko West	2.86 (1.36–9.25, 20)	1.88 (1.15–3.95, 6)	2.56A ^b (1.15–9.25, 26)
3. Tvärminne	1.41 (0.30–5.93, 34)	1.34 (0.57–3.70, 20)	1.34B (0.3–5.93, 54)
4. Rönnskär	2.45 (0.85–4.36, 20)	1.55 (1.02–2.44, 15)	1.99A (0.85–4.36, 35)
5. Söderskär	1.64 (0.30–3.37, 84)	1.26 (0.65–3.03, 26)	1.52B (0.30–3.37, 110)
Locations 2–5	1.75 ^c (0.30–9.25, 158)	1.37 ^d (0.65–3.95, 67)	1.63 (0.30–9.25, 225)

^a NS = no samples collected in 1997.

^b Means of the four locations not sharing capital letters in common are significantly different (one-way analysis of variance, $p < 0.0001$; Bonferroni multiple comparison test, $p < 0.05$ to $p < 0.001$).

^c Significantly greater (two-way analysis of variance, $p = 0.0006$) than the combined mean for the same locations in 1998.

^d Data from Utö are not included in this mean.

Table 3. Median concentrations and ranges of p,p'-dichlorodiphenyl-dichloroethylene (ppb fresh wet weight) in first-laid eggs of common eider hens at three locations in the Gulf of Finland

	Söderskär (n = 11)	Tvärminne (n = 7)	Rönnskär (n = 3)
Median	29.6A ^a	16.3AB	13.1B
Range	15.6–60.5	8.9–29.6	8.9–16.9

^a Medians not sharing capital letters in common are significantly different (Kruskal–Wallis test, $p = 0.0122$; Dunn's multiple comparison test, $p < 0.05$).

each of the 21 eggs, but residues did not differ among sampling sites. The median (range) concentrations (ppm fresh wet weight) of selenium and mercury in eggs were 0.55 (0.42–0.94) and 0.10 (0.04–0.46), respectively. The other OCs and arsenic were below detection limits in eggs.

Selenium–mercury relationships in eggs and blood

Concentrations of selenium and mercury in eggs were significantly correlated with corresponding concentrations of selenium (Spearman $r = 0.61$, $p = 0.0164$) and mercury (Spearman $r = 0.89$, $p < 0.0001$) in the blood of females from which the eggs were collected (Fig. 4). The median concentration of selenium in blood was 3.8 times higher than the median concentration in eggs. For mercury, the blood:egg ratio was 0.3:1. Both selenium and mercury were detected in 95 blood samples. The molar ratios of selenium to mercury in blood and eggs were 55:1 and 14:1, respectively. Molar concentrations of selenium and mercury were not correlated in blood or in eggs.

DISCUSSION

ALAD and lead in blood

We found that the proportion of eiders with blood lead concentrations of ≥ 0.20 ppm increased from west to east, suggesting greater exposure to lead shot or other sources of lead contamination in the eastern part of the study area. Although inhibition of ALAD activity may occur at blood lead concentrations lower than 0.20 ppm wet weight [26], this level is frequently considered to be the lower threshold of lead exposure in waterfowl that results in subclinical poisoning [19]. Our findings in eiders are consistent with the assumption of adverse effects at a blood lead concentration of 0.20 ppm, because birds with ≥ 0.20 ppm of lead in their blood had a median ALAD activity of 16 units, compared with the median of 113 units for birds that had blood lead concentrations of < 0.20 ppm. The negative relationship that we found between ALAD activity and lead concentrations in the blood is further evidence of a physiologically significant effect of lead exposure in eiders. The relationship between ALAD activity and lead in the blood of eiders in our study was similar to that reported in a study of canvasbacks (*Aythya valisineria*) [27]. The model described in the canvasback study was: $\ln \text{ALAD activity} = 7.7 - 0.82 \ln \text{lead concentration}$ [27], whereas the model that best fit our eider data was $\ln \text{ALAD activity} = 8.4 - 0.92 \ln \text{lead concentration}$.

Lead poisoning has been previously reported in common eiders found dead in the Gulf of Finland, and lead concentrations of 0.11 to 0.63 ppm wet weight were found in five blood samples from incubating females [12]. However, other reports of blood lead concentrations in eiders from the Gulf of Finland

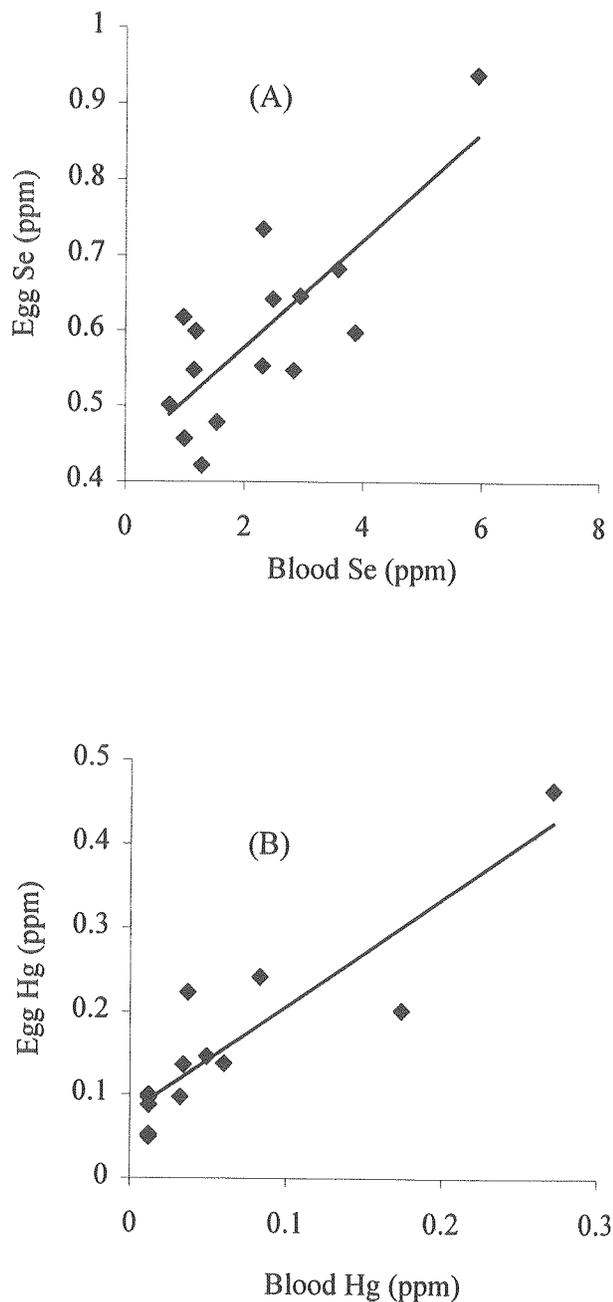


Fig. 4. Correlations of selenium (A) and mercury (B) in first-laid eggs and blood of common eider females. Selenium: Spearman $r = 0.61$, $p = 0.0164$, $n = 15$; egg Se = $0.44 + 0.07$ (blood Se). Mercury: Spearman $r = 0.89$, $p < 0.0001$, $n = 14$; egg Hg = $0.08 + 1.28$ (blood Hg).

are lacking. On eider breeding grounds in Alaska, USA, few incubating common eiders exhibited evidence of lead exposure, but about 25% of spectacled eiders (*Somateria fischeri*) and 20% of oldsquaw ducks (*Clangula hyemalis*) captured during incubation had ≥ 0.20 ppm lead in their blood [28]. Much of the lead exposure in birds sampled in Alaska was apparently the result of ingestion of lead shot, because shot was visible on radiographs of more than 11% of the spectacled eiders that were examined [28]. The ingestion of shot can result in extremely high blood lead concentrations in wild waterfowl. A spectacled eider caught in Alaska, and that had an ingested shot visible on X-ray, had a blood lead concentration of 14.37 ppm wet weight [29]. We found a similarly high concentration

(14.2 ppm) of lead in a blood sample from an eider that completed incubation and was observed on the nesting area a year later. Ingested lead shot may have contributed to the high lead value in this bird, and to the lead exposure in birds with blood lead concentrations of ≥ 0.20 ppm, because at least one eider was found dead with an ingested lead shot in the Finnish archipelago in 1998 (T. Hollmén, unpublished data). Lead shot was banned for waterfowl hunting in Finland in 1996. However, studies in the United States have shown that waterfowl continued to be exposed to lead shot in some areas even after the use of lead for waterfowl hunting was prohibited [30]. The higher prevalences of elevated lead concentrations that we found in birds from the two easternmost sampling sites could be the result of greater exposure to lead shot, or could reflect a west to east gradient of increasing lead exposure from other anthropogenic sources. The fact that we found lower mean blood lead concentrations in 1998 versus 1997 suggests an overall reduction in lead exposure between those two years.

The positive correlation of blood lead concentrations with incubation stage of eiders may be related to reproductive physiology. The medullary bone of birds is a reserve of mineral that undergoes destruction during the laying cycle, providing calcium for inclusion in developing eggshells [31]. Accumulated lead, also stored in medullary bone of ducks [32], may be simultaneously mobilized during the remodeling process. In a study with pigeons (*Columba livia*), the authors reported that some resorption of medullary bone continued after eggs were laid [33]. In another study, bony spicules were absent in marrow cavities of female house sparrows (*Passer domesticus*) trapped when their young were fledging [34]. If, as these studies suggest, mobilization of bone continues postovulation, one might expect lead to be released from medullary bone during incubation, with a resultant increase in blood lead concentrations.

Selenium and mercury in blood

Previous studies suggest that birds from marine environments tend to have higher selenium levels in their blood than nonmarine birds that are not exposed to high dietary selenium. For example, marine-feeding Eurasian oystercatchers (*Haematopus ostralegus*) had selenium concentrations in their blood that were about 3.5 times higher than birds of the same species that fed inland [35]. Sooty terns (*Sterna fuscata*), which inhabit marine areas where high levels of metals may occur naturally, had a mean selenium concentration of 8.76 ppm wet weight in their blood [36], and emperor geese (*Chen canagica*) in Alaska had mean blood selenium residues of up to 5.6 ppm wet weight [37]. The concentrations of selenium that we found in the blood of eiders were lower than those that were found in sooty terns and emperor geese, and may reflect normal marine exposure in the Baltic Sea. However, the maximum blood selenium concentrations at most of our study sites were higher than the maximum concentration found in blood samples collected from several eiders in the Gulf of Finland in 1994 [12].

Marine birds may also have higher residues of mercury in their tissues, without apparent toxic effects, than terrestrial birds [38]. However, we detected mercury in less than one half of the eider blood samples and the maximum concentration of 0.31 ppm was much lower than the concentration of 4.47 ppm wet weight reported from blood of sooty terns [36]. Unless eiders were feeding in areas of heavy mercury contamination, we would expect them to have lower tissue residues

than fish-eating birds because eiders are lower on the food chain. The relationship between selenium and mercury in bird tissues is variable but, in some studies, marine birds have had relatively higher levels of selenium than mercury in their tissues [39]. Our results agree with this because, in eider blood samples with detectable levels of both selenium and mercury, relatively more selenium was present on a molar basis. The molar ratio of selenium to mercury that we found in eider blood (55:1) was greater than the 36:1 ratio reported in red blood cells of Eurasian oystercatchers [40].

Contaminants in eggs and relationships with blood

The concentrations of selenium and mercury in eggs that may indicate reproductive problems are reported to be about 3 ppm wet weight and 0.5 to 2 ppm wet weight, respectively [13,38] and marine birds may tolerate even higher residues. Therefore, the median concentrations of selenium (0.55 ppm wet weight) and mercury (0.10 ppm wet weight) that we found in eider eggs probably do not pose a threat to reproduction. Few comparative data exist for selenium in eggs of birds from the Baltic, but the residues of mercury that we found in eider eggs were nearly twice as great as the mean (0.06 ppm wet weight) found in eider eggs collected in the early 1990s in northern Norway [41]. Mercury residues reported in eggs of fish-eating birds from the Baltic tend to be greater than those we found in eiders. For example, guillemot (*Uria aalge*) eggs collected in the early 1990s in the Baltic proper contained about 250 to 300 ng/g (0.25–0.30 ppm) of mercury, fresh weight [42].

We have found little previously published information on the concentrations of selenium or mercury in whole blood versus concentrations in eggs from the same birds. However, our finding of greater concentrations of selenium in blood than in eggs of eiders agrees with a study of Eurasian oystercatchers in which concentrations of selenium in red blood cells were greater than the levels found in egg yolk and egg white [35]. Although mercury in the blood and in the eggs of eiders were significantly correlated, the concentrations were low and we found lower levels in blood than in eggs. Based on our sample of blood and eggs, we found no evidence of arsenic exposure at our sampling sites in 1997 and 1998, although an earlier study reported up to 38.5 ppm arsenic (dry weight) in livers of several adult female eiders found dead at Söderskär [12].

A high degree of interspecific variability exists regarding sensitivity to the adverse reproductive effects of DDE, but the concentrations that we found in eider eggs are much lower than those that affect the most sensitive avian species [43]. The levels of *p,p'*-DDE that we found are also lower than those reported in common eider eggs collected in Scandinavia, Holland, and Maine, USA, in the 1970s [44,45]. The lack of correlation between DDE residues and eggshell thickness is not surprising because of the low DDE concentrations that we found. The mean eggshell thickness of 0.433 mm for the Finnish eiders was the same as that reported for eggshells from common eiders in Maine, although the contents of the Maine eider eggs had DDE residues that were more than 10 times greater [45] than those in our study. Greater DDE residues in eggs from Söderskär than in those from Rönnskär suggest the possibility of an increasing exposure gradient from west to east.

CONCLUSIONS

We conclude that the concentrations of lead that we found in the blood of common eiders captured in the Finnish archi-

pelago caused adverse physiological effects, as evidenced by the inhibition of ALAD activity. Although the mean concentrations of selenium in the blood of eiders ranged up to about 3 ppm, the fact that we found low residues in eggs suggests that selenium may not be a reproductive threat to the eiders. Relatively high residues of selenium in tissues of marine birds may be, at least in some instances, a natural phenomenon. The concentrations of mercury and *p,p'*-DDE in our sample of eider eggs were lower than the concentrations associated with reproductive problems in other species. Monitoring of contaminants, particularly selenium and lead, should be continued in this population of eiders. Sampling sites should include the easternmost nesting areas, because our results for lead and *p,p'*-DDE suggest an increase in exposure to those contaminants from west to east.

Acknowledgement—Funding support was provided by a research travel grant from The American-Scandinavian Foundation. P. Byholm, Y. Deligiannis, J. Högmänder, P. Ikonen, M. Nordström, T. Pankasalo, V. Ranki, H. Selin, K. Selin, W. Velmala, and M. Öst assisted with sample collection. We consulted with M. Samuel regarding statistical analysis and L. Locke provided helpful comments on the manuscript.

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