TRACE ELEMENTS STATUS OF MOOSE AND WHITE-TAILED DEER IN NOVA SCOTIA

Beth Pollock[1] and Erin Roger[2,3]

[1]P.O. Box 226, Norris Point, NL, Canada A0K 3V0; [2]Department of Natural Resources, 136 Exhibition St., Kentville, NS, Canada B4N 4E5

ABSTRACT: The province of Nova Scotia is considered to have two distinct moose populations: mainland and Cape Breton Island. In 2003, moose of the mainland area of the province were formally listed as "ENDANGERED" under the Nova Scotia Endangered Species Act. To date, the specific causes of the decline of this population have not been determined. Factors impacting health, including trace element imbalances, have been considered as potential limiting factors for the mainland population. Liver and kidney samples were collected from moose and white-tailed deer throughout Nova Scotia during the fall and winter 2000 – 2002 to compare trace element concentrations between the two species, in relation to age, gender, and geographical location, and to other areas outside the province. All samples were analysed for arsenic, cadmium, cobalt, copper, lead, manganese, nickel, selenium, and zinc. Tissue concentrations of trace elements in deer and moose in Nova Scotia are generally similar to levels reported in cervid populations elsewhere in North America and Europe with the exception of zinc and cobalt, which appear to be lower in Nova Scotia. Kidney cadmium concentrations are high in some Nova Scotia moose (geometric mean = 60.4 μg/g dry weight, 95% CI = 40.3 – 90.6, n = 21), however, similar or higher concentrations have been reported in other regions. Relative to reference values for domestic cattle, cobalt, copper, manganese, selenium, and zinc levels are deficient or marginally deficient in some animals, however, there appears to be little supporting evidence that clinical deficiencies of any of these trace elements are occurring in Nova Scotia moose or deer populations. The possibility that marginal or deficient levels of these or other trace elements and high levels of cadmium may impact the health of individual animals either directly or through interactions with other factors (e.g., infectious and non-infectious diseases, harsh environmental conditions, habitat limitations) cannot be dismissed. Some considerations for continued monitoring of trace element concentrations in these populations are discussed.

ALCES VOL. 43: 61-77 (2007)

Key words: *Alces alces*, environment, health, kidney, liver, monitoring, Nova Scotia, trace elements

The province of Nova Scotia is considered to have two moose (*Alces alces*) populations. The first population on Cape Breton Island experiences normal population growth and is harvested by recreational hunters. It was supplemented by 18 moose translocated from Alberta in 1947 and 1948 (Pulsifer and Nette 1995) and, not surprisingly, subsequent genetic analysis (Broders et al. 1999) has demonstrated the population's genetic structure to be most closely related to that of moose from Alberta. The second mainland population of moose is indigenous to the region and is representative of the eastern moose subspecies (*Alces alces americana*). It is made up of three subpopulations corresponding to the Cumberland, Tobeatic, and Guysborough regions of the province. This population has been in decline since the mid-1920s, despite being protected from legal hunting since 1981. In 2000, it was assigned a "RED" status which is defined to be a species at risk of extirpation or extinc-

[3]Present address: School of Biological Earth and Environment Sciences, University of New South Wales, Sydney, NSW 2052, Australia

tion under the General Status of Nova Scotia Wildlife Assessment Process. Following the completion of an independent commissioned status report (Parker 2003) in October of 2003, moose of the mainland area of the province were formally listed as "ENDANGERED" under the Nova Scotia Endangered Species Act. To date, the specific causes of the ongoing decline of the mainland moose population have not been determined. Factors influencing the size and distribution of moose populations in Nova Scotia may include disease (e.g., parelaphostrongylosis), habitat suitability, illegal hunting, and possibly predation of calves by black bears (*Ursus americanus*) or coyotes (*Canis latrans*) (Beazley et al. 2006). Trace element imbalances may also be a potential etiology for this population decline in part because such imbalances have been cited as possible contributing factors in population declines/mortality events of moose in other areas. Examples include possible copper (Cu) deficiency in moose in Alaska and northwestern Minnesota (Flynn et al. 1977, O'Hara et al. 2001, Custer et al. 2004) and Cu deficiency and molybdenosis in moose in Sweden (Frank et al. 1994, 2000). Results from preliminary trace element analyses of moose kidney and liver tissues have raised the possibility that high cadmium (Cd; Roger 2002) and/or low cobalt (Co; Frank et al. 2004) levels are impacting the health of moose on mainland Nova Scotia.

The white-tailed deer (*Odocoileus virginianus*) arrived in and dispersed throughout Nova Scotia in the period 1890 – 1920, and have subsequently fluctuated widely in abundance (Pulsifer and Nette 1995). In 2001, the population estimate for the province was approximately 51,500 (1.22 deer/km^2), which is below the optimal size of 80,000 animals, but still considered healthy (T. Nette, Nova Scotia Department of Natural Resources, personal communication). As with moose, white-tailed deer are present on the mainland and Cape Breton Island regions of Nova Scotia. As the only other cervid species in the province, white-tailed deer may serve as a comparison population for trace elements concentrations in moose with the caution that inter-species differences in trace element concentrations can exist.

The interpretation of trace element levels in free-ranging cervid populations can be difficult as 'normal values' have not been established and sources of variability within and between species and populations are known to exist due to differences in diet, age, gender, geographical location, and other factors (Frøslie et al. 1984, Ohlson and Staaland 2001, O'Hara et al. 2003, Custer et al. 2004, Gamberg et al. 2005). However, with this caution in mind, comparisons of trace element levels within and between populations and species can be helpful in identifying particular areas for further monitoring or study. The objectives of this study were to: (1) determine trace element concentrations in moose (liver and kidney) and deer (liver) tissue from Nova Scotia; (2) for each trace element, examine the species-specific relationship between tissue concentration and age, gender, and geographic region; (3) compare trace element concentrations found in Nova Scotia moose and deer to each other, to other free-ranging cervid populations, and to reference values for domestic cattle; and (4) discuss whether trace element imbalances may be contributing to the decline of the mainland population of moose in Nova Scotia.

METHODS

Sample Collection

During fall and winter 2000 – 2002, samples of liver (n = 48) and kidney (n = 21) from moose and liver from deer (n = 54) were collected from animals killed in vehicle accidents or by hunters, or animals killed and submitted to the Department of Natural Resources because of property intrusion or illness. All tissue samples were placed in plastic bags, labeled, and frozen at -15 to -20°C. Animal age was

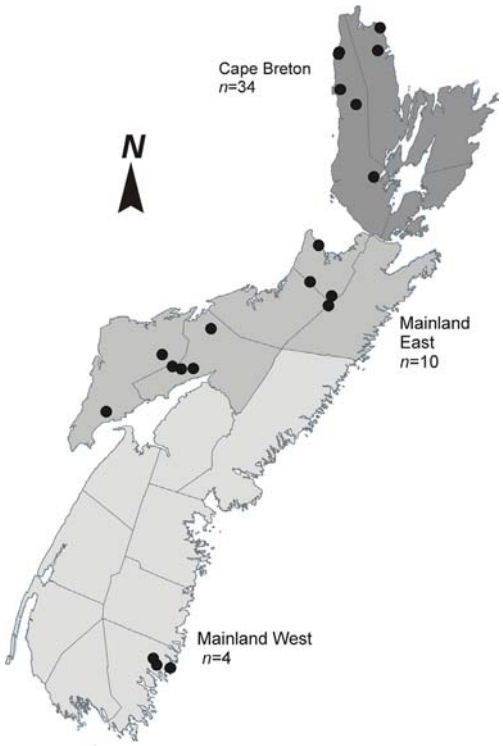

Fig. 1. Map of Nova Scotia illustrating locations of moose and number of moose sampled in each region.

determined by size (calves [moose] and fawns [deer]), and/or by analysis of cementum annuli of the lower canine and lower incisor teeth (Matson's Laboratory, Milltown, Montana, U.S.A.). Specific age data (in months) were available only for calves or fawns and those animals aged by tooth cementum analysis. Sex-specific animals were categorized by age as calves or fawns (< 12 months), yearlings (12 – 24 months), and adults (> 24 months) for descriptive and statistical analyses.

Location data were obtained for 51/54 (94%) deer and 20/48 (42%) moose. Exact locations of hunted moose in Cape Breton were not recorded; however hunting is limited to two counties in Cape Breton: Inverness and Victoria. The directional location relative to Cape Breton Highlands National Park was also recorded. Moose on the mainland are found in three subpopulations corresponding to the Cumberland, Guysborough, and Tobeatic regions of the province. For comparisons among geographical locations, moose were categorized according to three regions: Mainland East (ME) which includes moose from Cumberland and Guysborough sub-populations, Mainland West (MW) which includes the Tobeatic sub-population, and Cape Breton (CB) (Fig. 1). Similar regions were used to categorize deer (Fig. 2).

Analytical Methods

All samples were analyzed for trace element concentrations (arsenic [As], cadmium, cobalt, copper, lead [Pb], manganese [Mn], nickel [Ni], selenium [Se], and zinc [Zn]) at the Environmental Quality Laboratory, Environment Canada, Moncton, New Brunswick in 2002. Subsequently, samples were placed in Freeze-Dry flasks (40 ml, 80 ml, or 120 ml), and pre-frozen for a minimum of 12 hours. After freezing, samples were placed on the

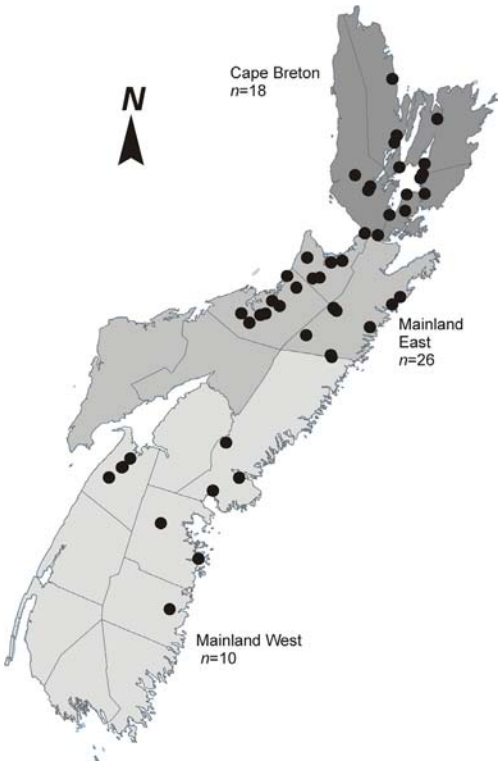

Fig. 2. Map of Nova Scotia illustrating locations of deer and number of deer sampled in each region.

Freeze-Dry System (Labconco FreeZone 4.5 Litre Freeze Dry System, Fisher Scientific Limited, Ontario, Canada) for approximately 48 hours after which they were homogenized using a mortar and pestle. Once ground, samples were weighed out (approximately 0.15 g of tissue) into the XP-1500 *Plus* vessels. Five ml of nitric acid and 2 ml of deonized water were added to each vessel. Each batch of samples contained a blank spike, duplicate, or spiked duplicate, and two quality control samples and was digested in the Microwave Accelerated Reaction System for Extraction (MARS-X, CEM Corporation, Québec, Canada). Once samples were at room temperature they were diluted to the 50 ml mark, homogenized, and transferred into 15 ml vials for ICP-MS analysis (inductively coupled mass spectrophotometer) (Perkin-Elmer Elan 6000, Perkin-Elmer Life and Analytical Sciences, Ontario, Canada). The minimum detection limit (mdl) for analysis was 0.25 µg/g for As and Se and 0.05 µg/g for Cd, Co, Cu, Mn, Ni, Pb, and Zn. Concentrations are reported in µg/g dry weight (dw).

Statistical Analysis

All transformations and analyses were carried out using statistical software (STATA v.8.0). For descriptive statistical analyses, geometric means, 95% confidence intervals (95% CI), and ranges were calculated for trace elements. Values less than the minimum detection limits (mdl) were replaced with ½ of the mdl.

Trace elements detected in at least 20% of the samples by species and tissue (all trace elements measured except for As and Pb in deer liver and As in moose liver) were included in univariate statistical analyses described below.

For each species, simple associations among trace elements and demographic variables (gender, age, geographical location) were examined. Trace element concentrations were successfully transformed (using the natural log [ln]) to render them normally distributed in order to meet the assumptions of the following parametric statistical tests: χ^2 statistics for categorical variables (gender, age group, geographical location) and pair-wise Pearson's correlation coefficients, *t*-tests and one-way ANOVA (followed by Bonferroni's adjustments for pair-wise comparisons) for normally-distributed continuous variables. For those variables for which ln transformation did not result in a normal distribution (Co and Zn in deer liver, and Cd, Mn, and Pb in moose liver), Spearman's correlation coefficients and Kruskal-Wallis tests were used. Differences between trace element concentrations in deer and moose liver samples were examined using *t*-tests. Results of $P < 0.05$ were reported as statistically significant.

For the purposes of comparison, all concentrations reported in wet weight (ww) in other studies were converted to dry weight by estimating the moisture content of liver and kidney tissues as 71.4% (Puls 1994), resulting in the conversion equation µg/g ww x 3.5 = µg/g dw. Specific reference values for tissue trace element levels in free-ranging populations of moose and white-tailed deer are unavailable for comparison. In lieu of species-specific reference values, cattle reference values were used for comparisons with the caution that species differences in trace element concentrations have been reported.

RESULTS

Demographic Data

Moose – Forty-eight liver samples and 21 kidney samples were collected from 48 moose. The geographic distribution of sampled moose is shown in Fig. 1. The majority of moose sampled were males (28 males, 8 females, 2 unknown gender). Ages ranged from 4 to 123 months. Based on age category, there were 5 calves, 11 yearlings, 25 adults, and 7 unknown age.

Males were older than females (male geometric mean age: 38 months, 95% CI =

26 – 54, n = 23; females: 18 months, 95% CI = 10 – 33, n = 12; t = 2.07, P = 0.023). No age group differences (χ^2 = 8.92, df = 4, P = 0.063) or gender differences (χ^2 = 2.47, df = 2, P = 0.29) were found among geographical locations.

Deer – Liver samples were collected from 54 white-tailed deer. The geographic distribution of all deer sampled is shown in Fig. 2. Gender and age data were collected for 51/54 animals. The majority of deer sampled were females (33 females, 21 males, 3 unknown gender). Ages ranged from 8 to 126 months (based on tooth cementum analysis). Based on age category, there were 2 fawns, 19 yearlings, 30 adults, and 3 unknown age.

The gender distribution differed among geographical locations (males: CB = 10/17, ME = 8/24, MW = 0/10; χ^2 = 9.6, df = 2, P = 0.008). No age group differences were found among geographical locations (χ^2 = 3.09, df = 4, P = 0.54) or between genders (χ^2 = 4.48, df = 2, P = 0.11).

Tissue Concentrations of Trace Elements and Determination of Deficiency/Toxicity

Table 1 provides a summary of the occurrence and concentrations of trace elements for deer liver, moose liver, and moose kidney. For calculations involving Ni concentrations, one sample was dropped because of an erroneously high value (44.5 μg/g dw). Domestic cattle reference values for marginal and toxic levels of each trace element are also listed where applicable. In general, based on reference values for domestic cattle, concentrations of As, Cu, Pb, and Ni in deer and moose tissues were within normal ranges (Puls 1994). Results for other trace elements are listed below.

Analytical Results

Moose liver samples had higher Cd and Cu and lower Co, Mn, and Se levels than deer liver samples (Table 1). Some regional differences were seen and are listed below along with detailed age and gender results for each trace element.

Cadmium – Relative to reference values for domestic cattle, Cd concentrations in moose and deer were below the chronic toxicity level (liver: 175 μg/g dw; kidney: 350 μg/g dw; converted from ww) (Puls 1994).

Regional differences were seen with Cd in moose kidney (F = 4.29, df = 20, P = 0.030). Bonferroni comparison indicated that MW concentrations (geometric mean = 166.6 μg/g dw, 95% CI = 48.5 – 572.1, n = 4) were greater than ME (45.0 μg/g dw, 95% CI = 17.0 – 119.2, n = 7) (P = 0.043) or CB (49.5 μg/g dw, 95% CI = 34.6 – 70.9, n = 10) (P = 0.048). A similar relationship was seen with liver concentrations (MW: 27.5 μg/g dw, 95% CI = 12.4 – 60.7, n = 4; ME: 4.6 μg/g dw, 95% CI = 1.1 – 19.1, n = 10; CB: 5.2 μg/g dw, 95% CI = 3.8 – 7.0, n = 34; P = 0.005). Female moose had higher kidney Cd concentrations than males (female geometric mean = 92.4 μg/g dw, 95% CI = 43.5 – 196.0, n = 10; male geometric mean = 41.1 μg/g dw, 95% CI = 29.7 – 57.3, n = 11; t = -2.30, P = 0.017).

No significant differences were found between age (in months) or age groups with respect to Cd concentrations in deer or moose.

Cobalt – In relation to reference values for domestic cattle, 29.2% of moose liver samples and 7.4% of deer liver samples were marginally deficient (0.06 μg/g dw) (Fig. 3a) (Puls 1994).

Regional differences were found in moose liver Co concentrations (F = 3.90, df = 47, P = 0.028). Bonferroni comparison indicated that ME moose had higher liver Co concentrations (geometric mean = 0.14 μg/g dw, 95% CI = 0.08 – 0.26, n = 10) than CB moose (0.07 μg/g dw, 95% CI = 0.06 – 0.09, n = 34) (P = 0.042). There was a significant negative association between age (in months) and Co concentrations in kidney (Pearson's r = -0.53, P <0.05): as age increased, kidney Co concentrations decreased.

Copper – In relation to reference values

Table 1. Mean trace element concentrations (μg/g dry weight) in white-tailed deer liver and moose liver and kidney in Nova Scotia in 2000–2001.

Trace Element	Concentration[1]			Reference Values[2]			
	Deer Liver	Moose Liver	Moose Kidney	Liver		Kidney	
n	54	48	21	Marginal	Toxic	Marginal	Toxic
Arsenic	na	na	0.19 (0.15 – 0.24)	na	> 24.5	na	> 17.5
	[< 0.25 – 0.60]	[< 0.25 – 1.42]	[< 0.25 – 0.59]				
	13.00%	6.30%	33.30%				
Cadmium	1.1 (0.8 – 1.5)[3]	5.8 (4.0 – 8.3)[4]	60.4 (40.3 – 90.6)	na	> 175	na	> 350
	[0.05 – 28.1]	[< 0.05 – 51.9]	[14.3 – 346.1]				
	100%	97.90%	100%				
Cobalt	0.15 (0.13 – 0.17)[3]	0.08 (0.07 – 0.10)[4]	0.10 (0.08 – 0.13)	0.06	> 17.5	< 0.05	> 105
	[< 0.05 – 0.41]	[< 0.05 – 0.39]	[< 0.05 – 0.21]				
	96.30%	85.40%	95.20%				
Copper	122.0 (88.9 – 167.5)[3]	251.6 (188.2 – 336.5)[4]	16.1 (14.8 – 17.5)	< 35 def	> 875		
	[2.0 – 506.3]	[2.9 – 625.5]	[12.5 – 23.6]	35 – 87.5			
	100%	100%	100%				
Manganese	11.6 (10.1 – 13.4)[3]	9.2 (7.2 – 11.7)[4]	13.0 (11.3 – 15.1)	< 3.5 def		3.3 – 4.2	
	[1.8 – 27.9]	[0.3 – 57.8]	[8.5 – 29.7]	3.5 – 10.5			
	100%	100%	100%				
Nickel	0.57 (0.41 – 0.80)[3]	0.37 (0.26 – 0.52)[3]	0.56 (0.33 – 0.95)	< 0.035		< 0.035	
	[< 0.05 – 4.46]	[< 0.05 – 5.24]	[0.13 – 5.76]				
	94.40%	93.80%	100%				
Lead	na	0.07 (0.05 – 0.09)	0.05 (0.04 – 0.08)	na	> 17.5	na	> 17.5
	[< 0.05 – 0.51]	[< 0.05 – 2.69]	[< 0.05 – 0.19]				
	14.80%	56.30%	47.60%				

[1] Geometric mean μg/g dry weight (95% confidence interval)[range] % samples above minimum detection limit.

[2] Marginal and toxic reference values for cattle in μg/g dry weight (Puls, 1994). Concentrations converted from wet weight based on estimate of 71.4% moisture (conversion factor of 3.5); def = deficient, marg = marginal.

[3,4] For each trace element in deer and moose liver, means having different superscripts were significantly different, $P < 0.05$.

na = not applicable.

Table 1 (continued). Mean trace element concentrations (μg/g dry weight) in white-tailed deer liver and moose liver and kidney in Nova Scotia in 2000–2001.

Trace Element	Concentration[1]			Reference Values[2]			
	Deer Liver	Moose Liver	Moose Kidney	Liver		Kidney	
n	54	48	21	Marginal	Toxic	Marginal	Toxic
Selenium	1.4 (1.2 – 1.6)[3]	0.7 (0.6 – 0.8)[4]	2.9 (2.5 – 3.3)	< 0.6 def	> 4.4	< 3.5	> 8.8
	[0.6 – 4.0]	[0.3 – 3.6]	[1.8 – 5.3]	0.6 – 0.88			
	100%	100%	100%				
Zinc	79.7 (70.9 – 89.6)[3]	75.0 (60.7 – 92.6)[3]	99.7 (87.4 – 113.7)	< 70 def		56 – 70	> 455
	[33.3 – 461.9]	[9.5 – 476.9]	[68.6 – 159.5]	70 – 87.5		def/marg	
	100%	100%	100%				

[1] Geometric mean μg/g dry weight (95% confidence interval)[range] % samples above minimum detection limit.

[2] Marginal and toxic reference values for cattle in μg/g dry weight (Puls, 1994). Concentrations converted from wet weight based on estimate of 71.4% moisture (conversion factor of 3.5); def = deficient, marg = marginal.

[3,4] For each trace element in deer and moose liver, means having different superscripts were significantly different, $P < 0.05$.

na = not applicable.

for domestic cattle, 4.2% and 2.1% of moose liver samples were deficient (< 35 μg/g dw) and marginally deficient (35 – 87.5 μg/dw), respectively, and 7.4% and 3.7% of deer liver samples were deficient and marginally deficient, respectively (Fig. 3b) (Puls 1994).

Regional differences were found in moose kidney Cu concentrations ($F = 5.07$, df = 20, $P = 0.018$). Bonferroni comparison indicated that MW moose had higher Cu (geometric mean = 19.0 μg/g dw, 95% CI =15.3 – 23.6, $n = 4$) compared to CB moose (14.5 μg/g dw, 95% CI =13.4 – 15.7, $n = 10$) ($P = 0.025$). Significant differences in liver Cu were found between age categories in moose ($F = 7.33$, df = 40, $P = 0.002$). Bonferroni comparison indicated that yearling moose had lower Cu in liver (geometric mean = 95.2 μg/g, 95% CI = 31.4 – 288.5, $n = 11$) compared to adult moose (geometric mean = 338.4 μg/g dw, 95% CI = 280.6 – 408.0, $n = 25$) ($P = 0.002$).

Manganese – In relation to reference values for domestic cattle, 4.2% and 41.7% of moose liver samples were deficient (< 3.5 μg/g dw) and marginally deficient (3.5 – 10.5 μg/g dw) , respectively, and 3.7% and 27.8% of deer liver samples were deficient and marginally deficient, respectively (Fig. 3c) (Puls 1994).

In deer, females had higher liver Mn concentrations (geometric mean = 12.9 μg/g dw, 95% CI = 11.1 – 14.9, $n = 33$) compared to males (geometric mean = 9.1 μg/g dw, 95% CI = 6.7 – 12.5, $n = 18$; $t = -2.36$, $P = 0.011$).

Nickel – The marginal reference value for liver for domestic cattle (< 0.035 μg/g dw) (Puls, 1994) is lower than the mdl for moose and deer liver samples (0.05 μg/g dw), thus the number of samples below the marginal reference value could not be calculated.

In moose, regional differences were found in liver Ni concentrations ($F = 29.24$, df = 46, $P < 0.0001$). Bonferroni comparison indicated that Ni in CB was higher (geometric mean = 0.65 μg/g dw, 95% CI = 0.52 – 0.80, $n = 33$) than ME (0.08 μg/g dw, 95% CI = 0.04 – 0.13, $n = 10$) ($P < 0.001$) and MW

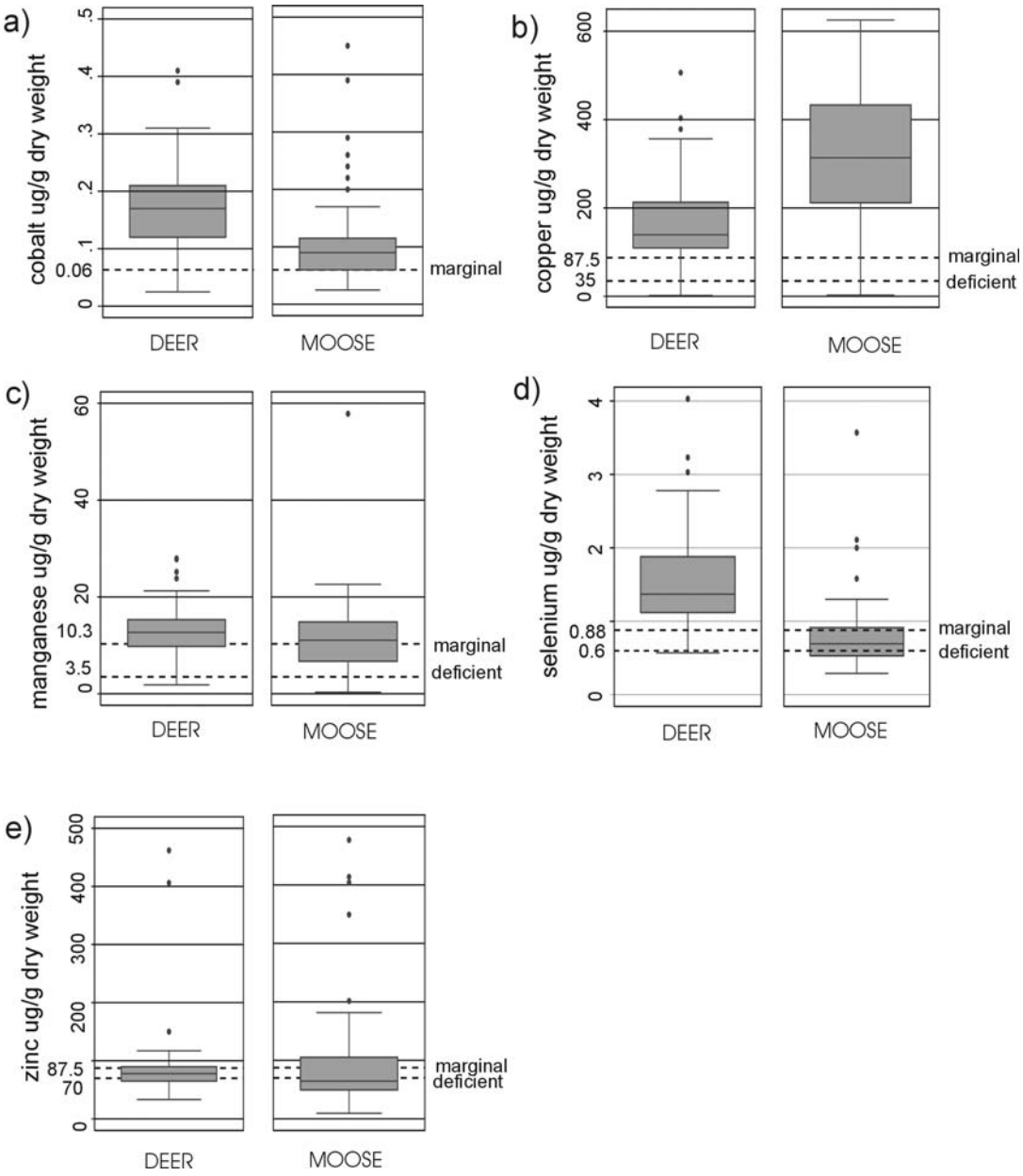

Fig. 3. Box plots representing trace element concentrations of cobalt (a), copper (b), manganese (c), selenium (d), and zinc (e) in white-tailed deer and moose liver from Nova Scotia. Shaded areas represent the distribution of the middle 50% of the data, short horizontal lines show the median and the 95% confidence limits. Broken lines indicate levels considered marginal and deficient for domestic cattle.

(0.18 µg/g dw, 95% CI = 0.01 – 3.60, $n = 4$) ($P = 0.012$). Similar differences were seen with kidney samples ($F = 12.54$, df = 20, $P = 0.0004$): CB: 1.37 µg/g dw, 95% CI = 0.72 – 2.60, $n = 10$; ME: 0.21 µg/g dw, 95% CI = 0.16 – 0.28, $n = 7$; $P < 0.001$; and MW: 0.33 µg/g dw, 95% CI = 0.06 – 1.88, $n = 4$; $P = 0.023$).

Selenium – In relation to reference values for domestic cattle, 39.6% and 33.3% of

moose liver samples were deficient (< 0.6 μg/g dw) and marginally deficient (0.6 – 0.88 μg/g dw), respectively, 3.7% and 11.1% of deer liver samples were deficient and marginally deficient, respectively (Fig. 3d), and 61.9% of moose kidney samples were deficient or marginally deficient (< 3.5 μg/g dw) (Puls 1994).

In deer, there was a significant positive association between age (in months) and Se concentrations in liver (r = 0.49, P<0.05). Male deer had higher concentrations of Se in liver (geometric mean = 1.70 μg/g dw, 95% CI = 1.33 – 2.17, n = 18) compared to females (1.28 μg/g dw, 95% CI = 1.13 – 1.45, n = 33; t = 2.36, P = 0.011).

Zinc – In relation to reference values for domestic cattle, 58.3% and 12.5% of moose liver samples were deficient (< 70 μg/g dw) and marginally deficient (70 – 87.5 μg/g dw), respectively, and 37.0% and 35.2% of deer liver samples were deficient and marginally deficient, respectively (Fig. 3 e), and 9.5% of moose kidney samples were deficient/marginal (56 – 70 μg/g dw) (Puls 1994).

DISCUSSION

Based on our analyses of tissue concentrations, evidence for widespread deficiencies or toxicities of trace elements in Nova Scotia moose or deer populations is lacking. Moose had higher liver concentrations of Cd and Cu and lower Co, Mn, and Se levels than deer. In general, trace element concentrations among mainland and Cape Breton populations were similar; however, some regional differences were seen in moose for Cd, Co, Cu, and Ni. Caution must be used when making these comparisons as many trace elements have been shown to vary with age, gender, season, locality, and health status.

Compared with cattle, it was found that Co, Cu, Mn, Se, and Zn levels in some Nova Scotia moose and deer are deficient or marginally deficient. However, to date, supporting evidence for clinical deficiencies or toxicities (Cd) of these trace elements in the form of clinical signs or pathological changes in individual animals has not been consistently found (Beazley et al. 2006).

The following discussions for each trace element of concern include tissue concentrations found in relation to levels in other studies on Nova Scotia moose and cervid populations elsewhere. However, caution must again be used when making these comparisons as many trace elements have been shown to vary with age, gender, season, locality, and health status. Some considerations for continued monitoring of trace element concentrations in these populations are also discussed.

Cobalt

Overall, liver Co concentrations in the present study were higher in deer compared to moose. Between regions, it was found that ME moose had higher liver Co levels than CB moose. Few other studies were found for comparison of tissue Co concentrations. One recent study (Frank et al. 2004), cites Co/vitamin B12 deficiency as a contributing factor in observed "moose sickness" in Nova Scotia. However, recent investigations into causes of morbidity and mortality in moose from the mainland population have not substantiated these conclusions as gross or microscopic lesions compatible with Co deficiency were not identified in any of 22 moose necropsied (Beazley et al. 2006).

Median concentrations of Co reported by Frank et al. (2004) were 0.09 μg/g dw (range = 0.01 – 0.29) in liver and 0.06 μg/g dw (range = 0.01 – 0.19) in kidneys (converted from ww). Similar concentrations were found in both moose and deer in the present study in mainland and Cape Breton populations (Table 1). Based on criteria for domestic cattle (Puls 1994, Radostits et al. 2000), of the moose livers sampled by Frank et al. (2004), 7/17 (41%) had marginal or deficient levels of Co (< 0.02 μg/g wet weight) compared to 29% of moose livers and 7% of deer livers in the

present study.

Liver cobalt levels in both moose and deer in Nova Scotia appear to be low compared to moose in the Yukon (mean = 0.46 μg/g dw, SD = 0.28; converted from ww) (Gamberg et al. 2005), and Sweden (median = 0.42 μg/g dw, range = 0.26 – 0.60; converted from ww) (Frank et al. 2000).

The diagnosis of Co/vitamin B12 deficiency in Nova Scotia moose made by Frank et al. (2004) appears to have been based on historical reports of diseased animals with signs of weakness, emaciation, and neurological lesions, and they cite McBurney et al. (2001) to support the diagnosis. However, the moose McBurney et al. (2001) described with neurological disease of uncertain etiology were in excellent body condition in contrast to the emaciation described by Frank et al. (2004). Also, any emaciated moose diagnosed with neurological disease in Nova Scotia have had parelaphostrongylosis and meningoencephalitis to account for their debilitated physical condition (Beazley et al. 2006). Therefore, the case for Co/vitamin B12 deficiency in Nova Scotia moose as presented by Frank et al. (2004) appears to be unsubstantiated.

As the levels in both moose and deer in Nova Scotia appear to be lower than those in other areas, continued monitoring of Co levels in these species is warranted. This could be accomplished through analysis of serum and liver samples from both healthy and diseased animals throughout the province. As supporting evidence for clinical Co deficiency is lacking at this point in time, it is strongly recommended that data also be collected on the health status, particularly body condition, of sampled animals to help determine if an association is present between low liver Co levels and poor health in moose populations in Nova Scotia.

Copper

Apparent or possible Cu deficiency has been reported in free-ranging moose from Alaska (Flynn et al. 1977, O'Hara et al. 2001), northwest Minnesota (Custer et al. 2004), and Sweden (Frank et al. 1994, Frank 1998).

In general, liver Cu concentrations in the present study appeared to be adequate to high, although a small number of moose and deer had deficient or marginally deficient levels relative to reference values for domestic cattle. Cape Breton moose had lower liver Cu levels than MW moose and among age groups, yearlings had lower liver Cu concentrations than adults.

Comparable levels of Cu in liver were found in other studies on apparently healthy moose from Norway (means = 80.5 - 354 μg/g dw; converted from ww) (Frøslie et al. 1984) and the Yukon (mean = 141.1 μg/g dw, SD = 167.0; converted from ww) (Gamberg et al. 2005). Moose with apparent Cu deficiency (Frank et al. 1994, O'Hara et al. 2001, Custer et al. 2004) had lower liver Cu levels than moose and deer in the present study.

Clinical signs of Cu deficiency reported in free-ranging cervids include antler deformities (Gogan et al. 1988), faulty hoof keratinization and reduced reproductive rates (Flynn et al. 1977), and diarrhea, anorexia, emaciation, osteoporosis, and loss of hair colour (Frank et al. 1994, Frank 1998). At the population level, Custer et al. (2004) found an association between lower liver Cu concentrations in moose and reduced calf-to-cow ratios in Minnesota. In general, young animals and fetuses are more susceptible to Cu deficiency than adults (Smith 1990, Radostits et al. 2000)

Notably, yearling moose in the present study had lower liver Cu concentrations than adults, however levels considered deficient were found in only a small number of animals. Since antler and hoof deformities have been reported in Nova Scotian moose (Beazley et al. 2006), continued monitoring of the Cu status of healthy and diseased moose, particularly yearlings, may be of value. Where Cu deficiency is suspected, analysis of liver samples for sulfur and molybdenum may aid

in diagnosis and interpretation of Cu results.

Zinc

Zinc concentrations in Nova Scotia moose and deer appear on the lower end of, or lower than, the range of values reported in free-ranging cervids elsewhere. In relation to reference values for domestic cattle, approximately 70% of moose and deer livers in the present study had deficient or marginal concentrations of Zn.

Liver Zn concentrations from moose and deer in the present study were lower than concentrations in moose from a previous study on moose in Nova Scotia (median = 161 μg/g dw; converted from ww) (Frank et al. 2004) from the Yukon (mean = 122.0 μg/g dw; converted from ww) (Gamberg et al. 2005), northern Alaska (mean = 232.8 μg/g dw) (O'Hara et al. 2001), northwestern Minnesota (geometric means = 167 μg/g dw and 219 μg/g dw) (Custer et al. 2004) and were similar to the lower range of concentrations found in moose from Norway (means = 73.5 - 112.0 μg/g dw; converted from ww) (Frøslie et al. 1984) and caribou (*Rangifer tarandus*) from northern Alaska (geometric means = 77.0 - 246.4 μg/g dw; converted from ww]) (O'Hara et al. 2003) and the Northwest Territories (means = 75.8 - 114.1 μg/g dw) (Elkin and Bethke 1995).

It is unclear whether the lower levels seen in Nova Scotia moose and deer are sufficiently low to result in clinical signs of Zn deficiency in individual animals, which generally manifest as hair and skin lesions (Radostits et al. 2000), or at the population level. However, signs of Zn deficiency have not been reported in moose or deer in Nova Scotia to date. Zinc may also interact with other trace elements including Cd and Cu (Puls 1994). Continued monitoring of Zn levels in moose may be of value, particularly in debilitated or diseased animals.

Manganese

Based on reference values for domestic cattle, approximately 30% of deer liver samples and 45% of moose liver samples had deficient or marginally deficient Mn concentrations. However, it appears unlikely that Mn concentrations are truly deficient in deer and moose in Nova Scotia as Mn levels in the present study were similar to those of apparently healthy caribou from the Northwest Territories (liver means = 8.6 - 15.9 μg/g dw) (Elkin and Bethke 1995) and moose from northwestern Minnesota (liver geometric means = 7.9 and 8.0 μg/g dw) (Custer et al. 2004).

Selenium

In the present study, moose had lower liver Se concentrations than deer. Based on reference values commonly reported for cattle, 15% of deer liver samples and 73% of moose liver samples were deficient or marginal (Puls 1994, Radostits et al. 2000).

Overall, tissue Se concentrations in moose and deer in the present study were similar to those reported in cervid populations elsewhere. Liver Se concentrations in deer and moose were similar to those of caribou and reindeer from Greenland (geometric means = 0.30 - 3.4 μg/g dw; converted from ww) (Aastrup et al. 2000), and moose from Norway (means = 0.28 - 3.2 μg/g dw; converted from ww) (Frøslie et al. 1984), northwestern Minnesota (geometric means = 1.2 and 2.7 μg/g dw) (Custer et al. 2004), and Sweden (medians = 0.33 - 1.02 μg/g dw; converted from ww) (Galgan and Frank 1995).

Soils throughout eastern North America, including Nova Scotia, are known to be generally deficient in Se. The role of Se in animal health is complex. In domestic animals, clinical signs of Se deficiency are commonly seen in juveniles in the form of weakness, muscle stiffness, inability to stand, and possible sudden death (nutritional muscular dystrophy) (Radostits et al. 2000). In a study of free-ranging black-tailed deer (*Odocoileus hemionus columbianus*) in northern California, Flueck (1994) found that pre-weaning fawn

survival increased with Se supplementation of adult females compared to unsupplemented controls.

It is unclear what effect subclinical deficiencies of Se may have on the health of Nova Scotia moose and/or deer either directly or through interaction with other trace elements, however continued monitoring of Se levels in these species appears warranted. Where Se deficiency is suspected (clinical signs observed) concentration in liver is a better indicator of Se status than kidney.

Cadmium

Cadmium concentrations in moose kidneys analyzed in the present study are among the highest reported for the species. Although the highest concentrations found were in adults from the MW region, calves and yearlings from all regions sampled had high kidney concentrations when compared to those found in young animals in other studies.

As discussed in other studies, age (Glooschenko et al. 1988, Paré et al. 1999, O'Hara et al. 2001), gender (Scanlon et al. 1986, Gustafson et al. 2000), geographical location (Paré et al.1999), species (Gamberg and Scheuhammer 1994), and season (Crête et al. 1989) have all been found to influence Cd concentrations. Thus, direct comparison of tissue concentrations among studies can be difficult and should be interpreted with caution.

Relative to other regions, overall geometric mean kidney concentrations in moose from the present study are higher than those reported in Newfoundland (mean = 19.3 μg/g dw; converted from ww) (Brazil and Ferguson 1989), New England (geometric means by age and gender = 31.3 - 41.8 μg/g dw; converted from ww) (Gustafson et al. 2000), Maine (mean = 23.8 μg/g dw), and Norway (means by geographical location = 8.4 - 20.5 μg/g dw) (Scanlon et al. 1986), and similar to or lower than mean kidney concentrations reported in Québec (means by gender and geographical location = 31.8 - 100.0 μg/g dw, Crête et al. 1987; mean = 72.4 μg/g dw, Paré et al. 1999), New Brunswick (means by county = 31.8 - 225.8 μg/g dw; converted from ww) (Ecobichon et al. 1988), Ontario (means by age group = 2.1 - 179.9 μg/g dw; converted from ww) (Glooschenko et al. 1988), Alaska (geometric means by geographical location = 4.9 - 70.7 μg/g dw; converted from ww) (Arnold et al. 2006), and the Yukon (mean = 98.4 μg/g dw; converted from ww) (Gamberg et al. 2005).

In the present study, kidney Cd concentrations in moose calves (16.6 and 83.3 μg/g dw, n = 2) and yearlings (geometric mean 54.4 μg/g dw, 95% CI = 25.0 – 83.7, n = 6) are among the highest reported for these age groups (Gamberg et al. 2005), however, the number of young moose sampled was low. Comparable levels of kidney Cd were reported in moose calves and yearlings sampled from the area surrounding the community of Rouyn-Noranda, Québec (Paré et al. 1999), and in the Yukon (Gamberg et al. 2005).

Paré et al. (1999) attributed the high level of Cd contamination found in moose of all ages surrounding Rouyn-Noranda to natural mineralization of the soil and historical and recent human contributions of Cd to the environment (including a Cu smelter). Gamberg et al (2005) concluded that high levels of Cd in Yukon moose are a result of naturally occurring geological sources, likely via the ingestion of Cd-accumulating plants such as willow, a preferred browse species for moose (Renecker and Schwartz 1998). Other explanations put forth for regional differences seen in Cd accumulation in free-ranging cervids also include: differences in buffering capacity of the soil, the degree of environmental acidification (Frøslie et al. 1986, Crête et al. 1987, Glooschenko et al. 1988), the composition and diversity of forage species in an area (Ohlson and Staaland 2001), and other habitat differences (Custer et al. 2004). In relation to the present study, a discussion of bedrock and soil composition

in relation to Cd levels in moose, porcupines, and willow spp. in Nova Scotia can be found in Roger (2002).

Liver Cd concentrations in deer in the present study were similar to those found in white-tailed deer in other regions of North America (Ecobichon et al. 1988, Glooschenko et al. 1988, Crichton and Paquet 2000).

The primary target of chronic Cd toxicity in mammals and birds is the kidney (Scheuhammer 1987, Alden and Frith 1991) and the earliest light microscopic change in this organ is proximal tubular necrosis (Alden and Frith 1991). In addition to the renal toxicity, there is also evidence that exposure to Cd can result in disturbances in calcium balance and decreases in bone density (Taylor et al. 1999, Larison et al. 2000). The threshold for significant renal tubular damage in mammals and birds from Cd accumulation is generally reported as 100 – 200 μg/g ww (approximately 350 – 700 μg/g dw) (Cooke and Johnson 1996), although a renal threshold of 30 μg/g ww (approximately 105 μg/g dw) for mammals has also been published (Outridge et al. 1994).

Among published studies on Cd in ungulates, evidence for biological effects associated with high concentrations is lacking. Paré et al. (1999) found no lesions characteristic of renal disease in 33 kidney samples with a mean Cd concentration of 123.1 (± 17.98) μg/g dw submitted for histopathological examination. O'Hara et al. (2003) found no histopathologic evidence of renal lesions in caribou kidneys with Cd concentrations of 1.9 – 115.5 μg/g dw (converted from wet weight) from northern Alaska. Kidneys from two moose sampled in the present study (Cd concentrations 96.1 and 346.2 μg/g dw) were submitted to the Atlantic Veterinary College Diagnostic Services for histopathological examination and no evidence of renal proximal tubular lesions was found, although autolysis and freezing artifacts may have masked subtle changes in the tissues (S. McBurney, Canadian Cooperative Wildlife Health Centre, personal communication). It may be that large ungulates such as moose that tend to accumulate high levels of Cd are less susceptible to the toxic effects of Cd than experimental animals; however, the possibility that elevated Cd concentrations in individual animals may lead to subclinical or clinical disease cannot be dismissed.

In the future, monitoring of moose tissue Cd concentrations in Nova Scotia should be carried out in conjunction with detailed health assessment of individuals including histopathological examination of kidneys.

CONCLUSIONS AND RECOMMENDATIONS

A good foundation of trace element data has been collected for moose and deer in Nova Scotia, however, some age groups and regions are under-represented. Some differences were found in trace element status between the mainland and Cape Breton moose populations which warrant continued monitoring. Tissue concentrations of trace elements in moose and deer in Nova Scotia appear to be generally similar to levels reported elsewhere in North America and Europe with the exception of Zn and Co.

Although kidney Cd concentrations in some Nova Scotia moose are high, particularly in the MW region, similar or higher concentrations have also been reported elsewhere. To date, individual or population-level health effects in relation to elevated tissue Cd levels have not been reported in free-ranging ungulates in North America, including Nova Scotia.

In relation to reference values for domestic cattle, marginal or deficient levels of Se, Cu, Co, Mn, and Zn were found in some moose and deer in Nova Scotia. At the present time, there appears to be little supporting evidence that clinical deficiencies of any of these trace elements are occurring in Nova Scotia moose or deer populations. However, the possibility that marginal or deficient levels of these or other trace elements and high levels of Cd

may impact the health of individual animals either directly or through interactions with other factors that may be contributing to the decline of the mainland moose population (e.g., infectious and non-infectious diseases, harsh environmental conditions, habitat limitations) cannot be dismissed.

Continued monitoring of trace element concentrations in both populations of Nova Scotia moose is of value because of the endangered status of the mainland population. Future monitoring efforts should include relevant demographic data and health assessment for all sampled animals in order to help establish "normal" values for these populations and to identify possible health effects in relation to possible trace element deficiencies (Co, Cu, Se, and Zn in particular) or toxicities (Cd). For each animal sampled, demographic data including geographical location, age, gender, and species are essential. As well, body condition should be assessed as many disease conditions related to trace element deficiencies can lead to loss of body condition or emaciation. The timely necropsy of debilitated or diseased animals is also strongly recommended. Collection of this supporting data is highly recommended and is essential for meaningful interpretation of trace element data collected in the future.

ACKNOWLEDGEMENTS

We gratefully acknowledge the cooperation of hunters and employees of the Nova Scotia Department of Natural Resources (NSDNR) from throughout the province for providing the samples. Ken Eagle (New Brunswick Department of Natural Resources) helped with age estimates for the sampled animals in addition to the tooth cementum analysis. The trace element analyses of tissue samples were completed at and funded by the Environmental Quality Laboratory (EQL), Environment Canada, Moncton, New Brunswick. The technical assistance provided by Marc Bernier and Kim Davis during the sample analyses was very much appreciated as was the help with follow up questions and clarifications provided by Martin Leger at the EQL. Tony Nette from the NSDNR and Pierre-Yves Daoust and Scott McBurney from the Canadian Cooperative Wildlife Health Centre (CCWHC), Atlantic Region, were instrumental in realizing the completion of the paper and provided helpful input and editorial comments throughout. Thanks also to S. Taylor for his assistance with the maps. Funding for the data analysis and completion of the manuscript was generously provided by the CCWHC and NSDNR.

REFERENCES

Aastrup, P., F. Riget, R. Dietz, and G. Asmund. 2000. Lead, zinc, cadmium, mercury, selenium and copper in Greenland caribou and reindeer (*Rangifer tarandus*). Science of the Total Environment 245:149-159.

Alden, C. A., and C. H. Frith. 1991. Urinary System. Pages 333-361 *in* W. M. Haschek and C. G. Rousseaux, editors. Handbook of Toxicologic Pathology. Harcourt Brace Jovanovich, San Diego, California, USA.

Arnold, S., R. Zarnke, T. Lynn, M.-A. Chimonas, and A. Frank. 2006. Public health evaluation of cadmium concentrations in liver and kidney of moose (*Alces alces*) from four areas of Alaska. Science of the Total Environment 357:103-111.

Beazley, K., M. Ball, L. Isaacman, S. McBurney, P. Wilson, and T. Nette. 2006. Complexity and information gaps in recovery planning for moose (*Alces alces americana*) in Nova Scotia, Canada. Alces 42:89-109.

Brazil, J., and S. Ferguson. 1989. Cadmium concentrations in Newfoundland moose. Alces 25:52-57.

Broders, H. G., S. P. Mahoney, W. A. Montevecchi, and W. S. Davidson. 1999. Population genetic structure and the effect of founder events on the genetic vari-

ability of moose, *Alces alces*, in Canada. Molecular Ecology 8:1309-1315.

COOKE, J. A., and M. S. JOHNSON. 1996. Cadmium in small mammals. Pages 389-404 *in* W. N. Beyer, G. H. Heinz, and A. W. Redmon-Norwood, editors. Environmental Contaminants in Wildlife: Interpreting Tissue Concentrations. Lewis Publishers, CRC Press, Incorporated, Boca Raton, Florida, USA.

CRÊTE, M., R. NAULT, P. WALSH, J.-L. BENEDETTI, M. LEFEBVRE, J.-P. WEBER, and J. GAGNON. 1989. Variation in cadmium content of caribou tissues from Northern Québec. Science of the Total Environment 80:103-112.

_____, P. POTVIN, P. WALSH, J.-L. BENEDETTI, M. LEFEBVRE, J.-P. WEBER, G. PAILLARD, and J. GAGNON. 1987. Pattern of cadmium concentration in the liver and kidneys of moose and white-tailed deer in Québec. Science of the Total Environment 66:45-53.

CRICHTON, V., and P. PAQUET. 2000. Cadmium in Manitoba's wildlife. Alces 36:205-216.

CUSTER, T. W., E. COX, and B. GRAY. 2004. Trace elements in moose (*Alces alces*) found dead in Northwestern Minnesota, USA. Science of the Total Environment 330:81-87.

ECOBICHON, D. J., R. HICKS, and G. REDMOND. 1988. A survey of cadmium concentrations in liver and kidney of deer and moose in New Brunswick. Department of Natural Resources, Fish and Wildlife Branch, Fredericton, New Brunswick, Canada.

ELKIN, B., and R. W. BETHKE. 1995. Environmental contaminants in caribou in the Northwest Territories, Canada. Science of the Total Environment 160-1:307-321.

FLUECK, W. T. 1994. Selenium deficiency in free-ranging black -tailed deer. Ecology 75:807-812.

FLYNN, A., A. W. FRANZMANN, P. D. ARNESON, and J. L. OLDEMEYER. 1977. Indications of copper deficiency in a subpopulation of Alaskan moose. Journal of Nutrition 107:1182-1189.

FRANK, A. 1998. 'Mysterious' moose disease in Sweden; similarities to copper deficiency and/or molybdenosis in cattle and sheep; biochemical background of clinical signs and organ lesions. Science of the Total Environment 209:17-26.

_____, R. DANIELSSON, and B. JONES. 2000. The 'mysterious' disease in Swedish moose; concentrations of trace elements in liver and kidneys and clinical chemistry; comparison with experimental molybdenosis and copper deficiency in the goat. Science of the Total Environment 249:107-122.

_____, V. GALGAN, and L. R. PETERSSON. 1994. Secondary copper deficiency, chromium deficiency and trace element imbalance in the moose (*Alces alces* L.): Effect of anthropogenic activity. Ambio 23:315-317.

_____, J. MCPARTLIN, and R. DANIELSSON. 2004. Nova Scotia moose mystery--a moose sickness related to cobalt and vitamin B12 deficiency. Science of the Total Environment 318:89-100.

FRØSLIE, A., A. HAUGEN, G. HOLT, and G. NORHEIM. 1986. Levels of cadmium in liver and kidneys from Norwegian cervides. Bulletin of Environmental Contamination and Toxicology 37:453-460.

_____, G. NORHEIM, J. P. RAMBAEK, and E. STEINNES. 1984. Levels of trace elements in liver from Norwegian moose, reindeer and red deer in relation to atmospheric deposition. Acta Veterinaria Scandinavia 25:333-345.

GALGAN, V., and A. FRANK. 1995. Survey of bioavailable selenium in Sweden with the moose (*Alces alces* L.) as monitoring animal. Science of the Total Environment 172:37-45.

GAMBERG, M., M. PALMER, and P. ROACH. 2005. Temporal and geographic trends

in trace element concentrations in moose from Yukon, Canada. Science of the Total Environment 351-352:530-538.

_____, and A. M. Scheuhammer. 1994. Cadmium in caribou and muskoxen from the Canadian Yukon and Northwest Territories. Science of the Total Environment 143:221-234.

Glooschenko, V., R. Downes, R. Frank, H. E. Braun, E. M. Addison, and J. Hickie. 1988. Cadmium levels in Ontario moose and deer in relation to soil sensitivity to acid precipitation. Science of the Total Environment 71:173-186.

Gogan, P. J., D. A. Jessup, and R. H. Barrett. 1988. Antler anomalies in Tule elk. Journal of Wildlife Diseases 24:656-662.

Gustafson, K., K. M. Bontaites, and A. Major. 2000. Analysis of tissue cadmium concentrations in New England moose. Alces 36:35-40.

Larison, J. R., G. E. Likens, J. W. Fitzpatrick, and J. G. Crock. 2000. Cadmium toxicity among wildlife in the Colorado Rocky Mountains. Nature 406:181-183.

McBurney, S., D. Brannen, and T. Nette. 2001. Neurological disease in moose in Nova Scotia. Canadian Cooperative Wildlife Health Centre Newsletter 8(1). Saskatoon, Saskatchewan, Canada.

O'hara, T. M., G. Carroll, P. Barboza, K. Mueller, J. Blake, V. Woshner, and C. Willetto. 2001. Mineral and heavy metal status as related to a mortality event and poor recruitment in a moose population in Alaska. Journal of Wildlife Diseases 37:509-522.

_____, J. C. George, J. Blake, K. Burek, G. Carroll, J. Dau, L. Bennett, C. P. Mccoy, P. Gerard, and V. Woshner. 2003. Investigation of heavy metals in a large mortality event in caribou of northern Alaska. Arctic 56:125-135.

Ohlson, M., and H. Staaland. 2001. Mineral diversity in wild plants: benefits and bane for moose. Oikos 94:442-454.

Outridge, P. M., D. D. Macdonald, E. Porter, and I. D. Cuthbert. 1994. An evaluation of the ecological hazards associated with cadmium in the Canadian environment. Environmental Review 2:91-107.

Paré, M., R. Prairie, and M. Speyer. 1999. Variations of cadmium levels in moose tissues from the Abitibi-Temiscamingue region. Alces 35:177-190.

Parker, G. 2003. Status Report on The Eastern Moose *(Alces alces americana* Clinton) in Mainland Nova Scotia. An independent commissioned report for Nova Scotia Department of Natural Resources, Kentville, Nova Scotia, Canada.

Puls, R. 1994. Mineral Levels in Animal Health: Diagnostic Data. Second edition. Sherpa International, Clearbrook, British Columbia, Canada.

Pulsifer, M. D., and T. L. Nette. 1995. History, status and present distribution of moose in Nova Scotia. Alces 31:209-219.

Radostits, O. M., C. C. Gay, D. C. Blood, and K. W. Hinchcliff. 2000. Veterinary Medicine: A Textbook of the Diseases of Cattle, Sheep, Pigs, Goats and Horses. Ninth edition. Harcourt Publishers Limited, London, U.K.

Renecker, L. A., and C. C. Schwartz. 1998. Food habits and feeding behaviour. Pages 403-440 *in* A. W. Franzmann and C. C. Schwartz, editors. Ecology and Management of the North American Moose. Smithsonian Institution Press, Washington, D.C., USA.

Roger, K. E. 2002. Plant and mammal tissue cadmium concentrations in Nova Scotia, and possible effects on moose (*Alces alces*) populations. B.Sc. Honors Thesis. Acadia University, Wolfville, Nova Scotia, Canada.

Scanlon, P. F., K. I. Morris, A. G. Clark, N. Fimreite, and S. Lierhagen. 1986. Cadmium in moose tissues: comparison of data from Maine, USA and from Telemark,

Norway. Alces 22:303-312.

SCHEUHAMMER, A. M. 1987. The chronic toxicity of aluminum, cadmium, mercury and lead in birds: a review. Environmental Pollution 46:263-295.

SMITH, B. P. 1990. Large Animal Internal Medicine: Diseases of Horses, Cattle, Sheep, and Goats. The C.V. Mosby Company, St. Louis, Missouri, USA.

TAYLOR, J., R. DEWOSKIN, and F. K. ENNEVER. 1999. Toxicological profile for cadmium (update). Agency for Toxic Substances and Disease Registry, U.S. Department of Health and Human Services, Atlanta, Georgia, USA.