ANALYSIS OF TISSUE CADMIUM CONCENTRATIONS IN NEW ENGLAND MOOSE

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ABSTRACT: Potentially high cadmium (Cd) levels in moose have raised concern over potential impacts to moose biology and human health risks. Moose liver and/or kidney samples were collected from New Hampshire, Maine, and Vermont, and tested for Cd levels. New Hampshire samples were collected in 1988, 1989, 1993, and 1998 while those from Maine and Vermont were collected in 1993. There were significant differences in liver Cd levels between sexes and among age categories. Bulls had higher mean levels than cows and there were significant increases in Cd levels with age. Kidney Cd concentrations were approximately 5 times higher than those in liver and regression analysis indicated a significant relationship. Mean liver Cd levels in adult (age 4.5+) bulls from New Hampshire increased significantly from 1988 to 1998 (3.78 vs. 6.17 ppm respectively). No significant difference was found in mean liver Cd levels among states.

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Past research has led to concern regarding the concentrations of cadmium (Cd) in the tissues of wildlife species including moose (*Alces alces*) due to its tendency to bioaccumulate with an animal's age and its potential health effects on wildlife and human consumers of wildlife (Eisler 1985, Scanlon *et al.* 1986, Isaza and Cavalier 1988a, Kalas and Myklebust 1994). Contrary to most contaminants which bioaccumulate, herbivores tend to accumulate higher levels of Cd than carnivores (Frank 1986). Because of this, moose have been proposed as an indicator species for Cd levels (Frank 1986, Kalas and Myklebust 1994) and moose Cd levels have been suggested as bioindicators of environmental quality (Kronberg and Glooschenko 1994). Cadmium, while minimally present in muscle tissue, tends to concentrate primarily in organ tissues, particularly liver and kidney (Eisler 1985, Brazil and Ferguson 1989) which in turn are frequently consumed by humans. Specific health effects, particularly for wildlife species, are poorly documented but potentially include acute toxicity, teratogenesis, bone tissue demineralization, and renal tubular damage (Eisler 1985). Cadmium concentrations tend to increase with age, although exposure in humans seems to result in increasing kidney levels through about age 40, stabilization, and then decreasing levels following age 60 (Isaza and Cavalier 1988a). Cadmium levels in excess of 10 parts per million (ppm) wet weight in vertebrate liver or kidney should be considered evidence of "contamination" and kidney levels in excess of 200 ppm wet weight should be considered life threatening (Eisler 1985).

Environmental Cd includes natural and anthropogenic sources. Anthropogenic sources are generally reported to be related to various industrial point sources resulting in atmospheric emissions that allow long-range transport. Base soil type and acid

rain may play a role in the mobility of Cd and its uptake into the food chain (Crête *et al.* 1987, Glooschenko *et al.* 1988). Levels of Cd in moose and other wildlife high enough to cause concerns regarding environmental and human health impacts have been reported from many jurisdictions in North America and Europe (Frøslie *et al.* 1986, Scanlon *et al.* 1986, Crichton 1998).

Interest in Cd levels in New Hampshire (NH) moose arose from research (Scanlon *et al.* 1986) done in Maine (ME). Because of the geographic proximity of ME, samples of moose liver and kidney tissue were also collected in NH. Subsequent interest by the U.S. Fish and Wildlife Service led the states of NH, ME, and Vermont (VT) to collect additional samples to ascertain if geographic differences existed in northern New England, and if temporal changes in Cd levels were occurring. Assessing potential impacts of Cd on moose health or the implications of Cd in moose tissue on human health risk were beyond the scope of this study.

METHODS

New Hampshire moose tissues were first sampled for Cd in late 1986 and 1987 prior to opening the moose-hunting season in 1988. These samples were largely derived from road-killed moose. New Hampshire samples were again collected from hunter-killed moose in 1988, 1989, 1993, and 1998. The 12 samples collected in 1986-1987 were combined with the 1988 samples (56) for the purpose of these analyses. Maine and VT samples were collected in 1993 from hunter killed moose. The NH samples from 1988 and 1989 were from moose of both sexes and a variety of ages. The 1993 samples from all states focused primarily on older-aged bulls. The 1998 samples from NH were primarily from older-aged bulls but included samples from a few adult cows. Moose were aged using the *cementum annuli* technique (Sargeant and Pimlott 1959). Laboratory analyses to determine Cd concentrations for the 1988 and 1989 samples were done using flame atomic absorption spectrophotometry (the Cd detection limit for these early samples was undocumented). Samples from 1993 and 1998 were analyzed using graphite furnace atomic absorption spectrophotometry (Cd detection limit = 0.10 ppm). All Cd concentrations are reported in ppm wet weight.

Previous studies have reported that data of this type may exhibit heteroscedasticity (Crête *et al.* 1987, Glooschenko *et al.* 1988) and the homogeneity of variances was assessed using the method of Glejser (1969) as outlined in Zar (1984). The SAS General Linear Models procedure (GLM) was used for analysis of variance (ANOVA) and regression analyses, while multiple comparison tests were done using Tukey's Studentized Range (HSD) or least squares means (SAS Institute Inc. 1990) in cases where class means may have been affected by unbalanced sampling and/or covariates. For some analyses, moose ages were combined into age categories to minimize the effects of aging techniques (Glooschenko *et al.* 1988). The age categories were calf (0.5 years), yearling (> 0.5 and 1.5 years), young adult (> 1.5 and < 4.5 years), and adult (4.5+ years).

RESULTS

Assessment of homogeneity of variances of liver Cd samples showed significant (F = 8.55; 1, 250 df; P = 0.0038) heteroscedasticity. Kidney Cd samples gave similar results (F = 53.44; 1, 100 df; P = 0.0001). All subsequent statistical analyses were based on natural log (ln) transformed data (Sokal and Rohlf 1981, Zar 1984) which stabilized the variances. All means and measures of dispersion are reported in the original units (ppm) by back transformation resulting in geometric means. Liver and kidney Cd samples summarized by year,

state, and sex are given in Table 1.

ANOVA of liver Cd levels by sex and age categories for all years and states combined showed significant differences between sexes (F = 9.79; 1, 245 df; P = 0.0020) and among age categories (F = 60.63; 3, 245 df; P = 0.0001), with no significant interaction. Analysis of least squares means indicates that bulls exhibited higher Cd levels than cows. Multiple comparison tests based on least squares means also indicate that liver Cd levels for all age categories differed significantly from each other ($P \leq 0.0101$). Calves had the lowest mean Cd levels followed by yearlings, young adults, and adults (Table 2). No significant differences were found between sexes within age categories. Regression analysis of liver Cd as a function of age confirmed a significant relationship (Fig. 1) with marked increase in liver Cd in younger animals followed by an apparent slowing of the bioaccumulation rate in older animals.

ANOVA of Cd levels for adult (age 4.5+) bulls for samples collected in 1993 from NH, ME, and VT showed no significant differences (F = 0.33; 2, 43 df; P = 0.7175) among the states. These data are summarized in Table 3. New Hampshire had the highest mean level followed by VT and ME.

ANOVA of Cd levels for adult (age 4.5+) bulls from NH collected in 1988, 1989, 1993, and 1998 indicated a significant difference among years (F = 3.14; 3, 69 df; P = 0.0309). Multiple comparison tests using Tukey's HSD indicate that this difference is due to 1998 levels being significantly higher than levels in 1988. Mean liver Cd levels sampled in the intervening years (1989 and 1993) show an increasing trend but were not significantly different from the earliest (1988) or most recent (1998) samples. No significant difference among years was found for adult (age 4.5+) cows from New Hampshire although sample sizes were

Table 1. Summary of moose liver and kidney cadmium levels in ppm wet weight by year, state, and sex.

Year	State	Sex	Sample Type	*n*	Minimum	Maximum	Geometric Mean	Std. Error
1988	NH	Bull	Liver	45	0.20	9.10	2.27	1.11
			Kidney	33	0.55	38.00	10.49	1.16
		Cow	Liver	23	0.39	14.00	1.93	1.19
			Kidney	18	0.79	41.00	8.94	1.26
1989	NH	Bull	Liver	32	0.86	7.90	2.80	1.11
			Kidney	32	1.50	61.00	11.94	1.14
		Cow	Liver	23	0.43	7.10	1.90	1.16
			Kidney	20	1.20	51.00	11.25	1.22
1993	ME	Bull	Liver	21	2.41	7.33	4.14	1.07
	NH	Bull	Liver	21	0.81	12.27	4.81	1.15
	VT	Bull	Liver	6	2.54	8.14	4.53	1.21
		Cow	Liver	4	1.57	4.72	2.94	1.26
1998	NH	Bull	Liver	69	1.12	16.18	5.00	1.05
		Cow	Liver	9	1.42	11.28	3.82	1.23

Note: NH = New Hampshire, ME = Maine, VT = Vermont

Table 2. Summary of moose liver cadmium levels in ppm wet weight by sex and age category for all years and states combined.

Sex	Age Category	*n*	Minimum	Maximum	Geometric Mean	Std. Error
Bull	Calf	6	0.20	1.70	0.79	1.36
	Yearling	26	0.56	4.70	1.75	1.09
	Young Adult	65	1.12	7.30	3.78	1.05
	Adult (4.5+)	97	0.81	16.18	4.86	1.05
Cow	Calf	7	1.30	7.10	0.66	1.17
	Yearling	10	0.81	12.27	1.40	1.17
	Young Adult	19	2.21	16.18	2.66	1.18
	Adult (4.5+)	23	2.44	5.43	3.25	1.11

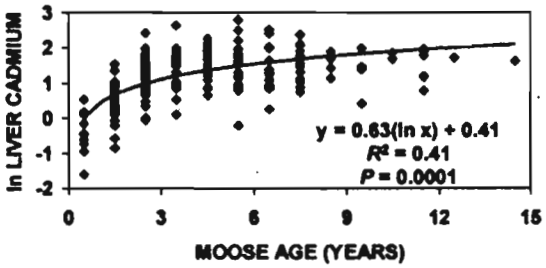

Fig. 1. Relationship between natural log (ln) of liver cadmium level (ppm wet weight) and age of New England moose.

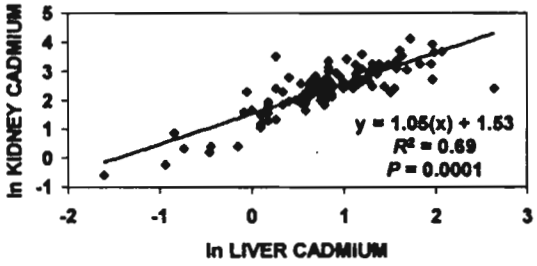

Fig. 2. Relationship between natural log (ln) of liver and kidney cadmium levels (ppm wet weight) for New Hampshire moose.

much smaller. These data are summarized in Table 4.

Past research has indicated a significant relationship between liver and kidney Cd levels (Frøslie *et al.* 1986). This was also the case for samples from NH collected in 1988 and 1989. Results of regression analysis describing this relationship are presented in Fig. 2. Kidney Cd levels tend to run on average about 5 times higher than that in liver.

DISCUSSION

Based on the earliest NH samples collected in 1986-1987, it was recommended that liver and kidney organ tissues not be consumed due to the high levels of Cd (Isaza and Cavalier 1988a). A similar recommendation was made in ME in 1985 following early Cd sampling. Results of samples collected over the 11 year period from 1988 to 1998 in NH suggest that mean liver Cd levels in adult (age 4.5+) bull moose

Table 3. Summary of moose liver cadmium levels in ppm wet weight for adult (age 4.5+) bulls from New Hampshire (NH), Maine (ME), and Vermont (VT) in 1993.

State	*n*	Minimum	Maximum	Geometric Mean	Std. Error
ME	20	2.57	7.33	4.22	1.07
NH	21	0.81	12.27	4.81	1.15
VT	4	2.54	8.14	4.53	1.31

Table 4. Summary of moose liver cadmium levels in ppm wet weight for adult (age 4.5+) bulls and cows from New Hampshire in 1988, 1989, 1993, and 1998.

Year	Sex	*n*	Minimum	Maximum	Geometric Mean	Std. Error
1988	Bull	13	2.30	9.10	3.78	1.14
	Cow	8	1.50	5.90	3.06	1.19
1989	Bull	9	2.20	7.90	4.62	1.16
	Cow	11	1.30	7.10	3.10	1.16
1993	Bull	21	0.81	12.27	4.81	1.15
1998	Bull	30	2.21	16.18	6.17	1.08
	Cow	4	2.44	5.43	4.26	1.21

are significantly higher in 1998 than they were in 1988 while samples from intervening years show a steady (but statistically insignificant) increase. The current mean liver Cd level in adult (age 4.5+) bulls from NH (6.17 ppm) and the highest level found (16.18 ppm) are still below the 50-160 ppm level which may adversely affect the health of cattle (Isaza and Cavalier 1988b). Current kidney Cd levels, while not measured in recent years, were predicted to average about 31 ppm and at the highest level could reach about 85 ppm (based on the regression in Fig. 2). This is less than, but is approaching, the 100-250 ppm kidney Cd levels which may adversely affect the health of cattle (Isaza and Cavalier 1988b). It is unknown what concentration levels will adversely affect individual moose.

Considerable evidence supports the tendency for Cd to bioaccumulate with age. Moose tend to have higher Cd levels than white-tailed deer (*Odocoileus virginianus*), perhaps related to differences in diet or the relatively greater longevity of moose (Crête *et al.* 1987, Isaza and Cavalier 1988a). It is common to see older moose harvested in NH, ME, and VT where the lack of predators and reduced harvest rates likely result in lower overall mortality rates and an "older population" compared to jurisdictions with more predation. The detected difference in mean Cd levels between bulls and cows and differences in the pattern of temporal change may arise from physiological differences such as lactation or antler growth (Crête *et al.* 1987).

In summary, it appears that Cd levels in moose organ tissues have risen over the past 11 years and this potentially has environmental and moose management implications. Both liver and kidney Cd levels in NH moose are creeping upwards toward the lower limits of Cd levels which may adversely affect the health of cattle. The Cd levels which adversely affect moose, and how these manifest themselves in relation to moose biology and management, requires additional research. From a human health risk perspective, the current health advisories by the Department of Health and Human Services against the consumption of moose liver and kidney in NH appear more warranted than in the past.

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