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Trace Metal and Organochlorine Pesticide Concentrations in Cold-Stunned Juvenile Kemp's Ridley Turtles (*Lepidochelys kempii*) from Cape Cod, Massachusetts

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ABSTRACT. – **Whole blood and keratin mercury concentrations, complete blood cell counts, and plasma biochemical health profiles were evaluated in 31 juvenile cold-stunned Kemp's ridley turtles (*Lepidochelys kempii*) from Cape Cod, Massachusetts. In addition, plasma copper, zinc, selenium, and cadmium concentrations were measured for 16 of these turtles. Liver mercury concentrations were measured for 6 turtles that were dead upon acquisition or died during rehabilitation. Concentrations of 18 organochlorine pesticides were measured in plasma of 18 live turtles, and liver, kidney, fat, and brain of 3 deceased turtles. Metal levels were generally similar to those previously published for sea turtles, with mean values (wet weight) of 24 ng/g for blood mercury, 67 ng/g for liver mercury, 389 ng/g for keratin mercury, 690 ng/g for plasma copper, 2290 ng/g for plasma zinc, and 490 ng/g for plasma selenium. Cadmium was not detected in any sample. Organochlorine levels were generally low in comparison to values published for cold-stunned juvenile Kemp's ridley turtles in the 1980s. Several significant correlations between metal levels, hematology, and plasma biochemical health parameters were detected; however, the cause of these correlations could not be determined. This study demonstrates that Kemp's ridley turtles may be exposed to contaminants at a young age. Further investigation of the sources and effects of contaminants in juvenile sea turtles is warranted.**

KEY WORDS. – **Reptilia; Testudines; Chelonidae; sea turtle; toxicology; hematology; plasma biochemistry; cold-stunning**

Heavy metal and pesticide contamination of marine animals has been widely documented in recent years (Lake et al. 1994, 1995; McKenzie et al. 1999; Becker et al. 2002; Burger et al. 2003; Gardner et al. 2003; Keller et al. 2004a, 2004b; Day et al. 2005, 2007; Mos et al. 2005). Living organisms may be exposed to such compounds via ingestion, inhalation, or dermal contact and as these compounds enter the food chain, organisms at higher trophic levels may accumulate significant concentrations within their tissues (Becker et al. 2002; Risher et al. 2002; Shaw et al. 2005). Vertebrates exposed to heavy metals and pesticides may be affected by neurologic, reproductive, gastrointestinal, respiratory, immunologic, hepatic, renal, and dermal disorders (Amin-Zaki et al. 1974; Marsh et al. 1987; Harada 1995; Castoldi et al. 2001; Risher et al. 2002; Garcia 2003; Gopal 2003; Mos et al. 2005). Two recent studies in sea turtles have demonstrated significant correlations between clinical health parameters, immune function, and exposure to organochlorine pesticides and mercury (Keller et al. 2004b; Day et al. 2007).

Kemp's ridley turtles (*Lepidochelys kempii*) are the most critically endangered of the sea turtles, having experienced a 94% reduction in the breeding population since the 1940s (Márquez-M. et al. 2005). Recently, however, a variety of successful conservation activities have resulted in a modest population increase (National Marine Fisheries Service and US Fish and Wildlife Service 2007). Although adult Kemp's ridley turtles are largely inhabitants of the Gulf of Mexico, the northwestern Atlantic is an important seasonal foraging ground for postpelagic juveniles (Lazell 1980; Morreale and Standora 2005). Late autumn stranding of "cold-stunned" juvenile Kemp's ridley turtles is relatively common in New York and New England, and coordinated efforts to rehabilitate these individuals have been underway for approximately 20 years (Burke et al. 1991; Morreale et al. 1992; Carminati et al. 1994; Matassa et al. 1994; Pisciotta et al. 1995; Gerle et al. 1998; Sadove et al. 1998; Smith et al. 2000; Still et al. 2000, 2005; Dodge et al. 2007). Between 1994 and 2007, the New England Aquarium Rescue and Rehabilitation Department (Boston, Massachusetts) admitted over 500 cold-stunned Kemp's ridley turtles (Matassa et al. 1994; Turnbull et al. 2000; Smith et al. 2000; Wyneken et al. 2005). Based on body size, it is believed that these individuals are in the 2- to 5-year-old age class, representing the transitional stage between pelagic and benthic existence (Morreale and Standora 2005).

This study was conducted to evaluate concentrations of selected metals and organochlorine pesticides in cold-stunned juvenile Kemp's ridley turtles from Cape Cod, and to determine whether these compounds were present in concentrations that were likely to impact the health of the turtles. Correlations of selected metal concentrations with clinical hematology and plasma biochemical health parameters were assessed.

METHODS

Tissue Collection and Preparation. — This study was approved by the Institutional Animal Care and Use Committee of the New England Aquarium, the US Fish and Wildlife Service, and MassWildlife (the Massachusetts Department of Fisheries and Wildlife). Carapace keratin and blood were collected as part of the overall medical evaluation of 31 cold-stunned juvenile Kemp's ridley turtles from Cape Cod, Massachusetts, that were presented to the New England Aquarium Rescue and Rehabilitation program in 2005. In addition, tissue samples were collected from 6 Kemp's ridley turtles that were dead upon arrival or died during rehabilitation. Two to three ml of blood was collected from the dorsal cervical sinus and placed into blood collection tubes containing lithium heparin (Microtainer, Becton Dickinson and Company, Franklin Lakes, NJ). One-half of the blood sample was centrifuged at 3000 rpm for 5 minutes, and the plasma was harvested. White blood cell counts and plasma biochemistry analyses were performed at a commercial veterinary diagnostic laboratory using the Eosinophil Unopette method (Becton Dickinson and Company) and the Olympus 5400 analyzer (Olympus America, Melville, NY), respectively. Differential white blood cell counts were performed by identifying 100 white blood cells on fixed blood smears stained with modified Wright's Geimsa stain (Fisher Scientific, Middletown, VA). Hematocrit was measured after centrifugation of whole blood at 12,000 × g for 3 minutes in heparinized capillary tubes. Approximately 0.5 g of keratin was collected from 4 posterior marginal scutes (generally the left and right 12th and 13th marginals) using a plastic scraping tool and placed into plastic bags (Nasco Whirl Pak, Fort Atkinson, WI). For animals that died, approximately 5 g of liver, kidney, brain, and fat were harvested at necropsy and placed into plastic Whirl Pack bags. Samples were stored at −80°C until analysis.

Heavy Metals Analysis.. — All whole blood, liver, and keratin samples were analyzed for total mercury concentration at the Center for Environmental Sciences and Engineering at the University of Connecticut using cold vapor atomic absorption spectrophotometry (CVAAS) by US Environmental Protection Agency method 245.6. Approximately 0.5 g of sample was placed in a digestion tube with 4 ml of sulfuric acid and 1 ml nitric acid. The tube was then placed in a hot block and heated to 95°C for 30 minutes. Samples were then cooled to room temperature, and 10 ml of potassium permanganate and 5 ml of potassium sulfate were added to the digestion tube. The samples were then returned to the hot block and digested for an additional 90 minutes at 30°C. The samples were cooled and 2 ml of hydroxylmine-hydrochloride was added and finally diluted with deionized (DI) water to 50 ml. Samples were analyzed by CVAAS on a Perkin-Elmer flow injection mercury system (Perkin Elmer Life and Analytical Sciences, Inc., Waltham, MA, USA) using standard protocols. If sufficient sample volume was available, plasma was analyzed for total copper, zinc, selenium, and cadmium concentrations using Perkin Elmer/Sciex ELAN inductively coupled plasma mass spectroscopy (ICPMS). The entire sample was thawed and homogenized, and approximately 0.1 ml was placed into a hot block tube. Five milliliters of concentrated trace metal grade nitric acid was added to each tube; they were placed on the hot block and refluxed for 4 hours at 95°C. The sample was cooled, 2 ml DI water and 3 ml of trace metal grade hydrogen peroxide were added, and the sample was heated in the hot block until the effervescence subsided. The samples were once again cooled and brought up to the final volume of 50 ml with DI water. Samples were then analyzed by ICPMS using standard protocols. Interference check solutions were analyzed (ICS A and ICS A + B; High Purity Standards, Charleston, SC) with all sample runs to compensate for any baseline effects due to high concentrations of common elements, such as calcium and sodium, which can result in an overreporting of the data. Standard quality assurance procedures were employed, including analysis of duplicate samples, blanks, spikes, and standard reference material (DOLT-3; National Research Council Canada). The relative percentage of difference between the analyzed and certified values for the standard reference material was within acceptable tolerances (copper, 4.1%; selenium, 1.0%; and zinc, 2.6%).

Organochlorine Analysis. — Plasma and tissue samples were analyzed for 20 organochlorine pesticides at the Virginia/Maryland Regional College of Veterinary Medicine Pesticide Residue Laboratory. Plasma and tissues were extracted using matrix solid phase dispersion and analyzed for pesticide residues using gas chromatography (GC). Briefly, a 500-µl aliquot of plasma or 0.5 g of tissue were transferred to a glass mortar containing 2 g Bondesil® (C-18, 40 µm) bulk sorbent (Varian, Harbor City, CA). Using a glass pestle, the tissue and sorbent were ground until the substrate gave the appearance of fine grains. The sample–sorbent mixture was then transferred to a Bond Elut® (Florisil, 2 g, 20 ml) Solid phase extraction (SPE) cartridge (Varian) using a metal spatula. A frit (Varian) was added to the cartridge and gently pressed to compact the sample–sorbent mixture. Analytes were extracted from the column with 4 consecutive 5-ml rinses of pesticide grade dichloromethane (Fisher Scientific, Fairlawn, NJ) using a VisiPrep SPE vacuum manifold (Supelco, Bellefonte, Pennsylvania) and collected in a 25-ml glass test tube. The combined dichloromethane eluates

were concentrated under a gentle stream of nitrogen to approximately 1 ml, the solvent was exchanged to hexane, and the final volume was adjusted to 1 ml before the extract was transferred to an autosampler vial.

Tissue extracts (1 μl) were injected on an Agilent 6890 GC (Agilent Technologies, Little Falls, DE, USA) using a single injection–dual column (RTX-5, RTX-35, 30 m × 0.25 mm × 0.25 μm for both) configuration and detection using dual microelectron capture detectors (μECD). This made it possible for a single injection to yield both primary and confirmatory measured concentrations. The 6890 GC operating conditions were as follows: injector (250°C), each μECD (350°C), oven (60°C [hold 1 minute] to 100°C at 10°C/min [hold 1 minute], 100°C to 275°C at 4°C/min [hold 15 minutes]); Ultra-high purity (UHP) helium carrier gas, UHP nitrogen detector make-up gas, constant flow, initial flow 2.3 ml/min total flow, initial pressure 23.15 pounds/inch^2. Agilent Chemstation was used for instrument control and data analysis and the external standard method was used for quantitation.

Additional confirmation of all sample results was performed using a Hewlett-Packard Model 5890 Series II Plus gas chromatograph (GC) (Agilent Techologies) coupled to a 5972 mass-selective (MS) detector operated in selected ion monitoring (SIM) mode using electron impact ionization with an HP-5ms (30 m × 0.25 mm × 0.5 μm) column and the following operating conditions: injector 250°C, MS detector transfer line 280°C, oven using the same temperature program as for GC analysis, UHP helium carrier gas at 1 ml/min, injection volume 1 μl. Chemicals included in the multiresidue evaluation of tissues were as follows: aldrin, alpha-hexachlorocyclohexane (alpha-BHC), alpha-chlordane, beta-BHC, delta-BHC, dieldrin, endosulfan I and II, endosulfan sulfate, endrin, endrin aldehyde, endrin ketone, gamma-BHC (lindane), gamma-chlordane, heptachlor, heptachlor epoxide, methoxychlor, p,p′-DDE; p,p′-DDD, and p,p′-DDT. For all organochlorine chemicals evaluated, recovery was verified using standards prepared with known concentrations of each chemical. No matrix blanks were extracted. Quantitation was performed using external calibration. A reference standard containing 20 organochlorine pesticides was purchased from Restek (Mix AB No. 1 No. 32291, Bellefone, PA). A stock solution of 20 μg/ml in hexane was used to prepare calibration standards at 0.001, 0.002, 0.004, 0.008, 0.016, and 0.04 μg/ml concentrations.

Statistical Analysis. — Linear regressions of metal concentrations against each other, weight, straight carapace length (SCL), hematology, and plasma biochemistry parameters were performed (SigmaStat 3.1, Systat, Richmond, CA), and those with a significant slope were noted. To more completely test how the full suite of hematologic and plasma biochemical health parameters varied with metal concentrations, a backward, step-wise regression (SigmaStat 3.1) was conducted for 14 turtles for which all 5 metal assays were performed (keratin mercury, blood mercury, plasma copper, plasma zinc, plasma selenium). Each metal measurement was set as the dependent variable to determine which of the hematologic and plasma biochemical variables entered the model ($F = 4.00$ to remove). Comparison of liver mercury levels to hematology and plasma biochemical values was not possible because several of the turtles sampled for liver mercury were dead upon arrival, and thus were not sampled for hematology and plasma biochemistry. Statistical analysis of organochlorines was not performed because of limited sample size and marginally detectable concentrations of most compounds.

RESULTS

Mean, standard deviation, and range of SCL, weight, and metal concentrations of keratin, blood, plasma, and liver are presented in Table 1. Mean, standard deviation (SD), and range of hematology and plasma biochemistry parameters are presented in Table 2.

Mercury was detected in the keratin, blood, and liver of every turtle tested. Mercury concentrations were consistently lowest in blood and greatest in keratin, with keratin levels 22.4- ± 20.3-fold (mean ± 1 SD, $n = 27$) greater than blood (paired t-test, $t = -7.988$, $p < 0.001$, power = 1.00). There was no association between blood and keratin mercury concentrations ($F_{1,25} = 0.32$, $p > 0.50$, $r^2 = 0.01$; Table 3). The values for liver were intermediate, being 4.0- ± 2.8-fold (mean ± 1 SD, $n = 5$) greater than blood, and 9.8- ± 8.8-fold (mean ± 1 SD, $n = 4$) less than keratin, but the concentration of mercury in the liver was not associated with the concentration in the blood or keratin ($F_{1,3} = 1.75$, $p > 0.20$, $r^2 = 0.37$, $F_{1,2} = 0.30$, $p > 0.60$, $r^2 = 0.13$, respectively; Table 3). Although there was no association between keratin or blood mercury to turtle weight or SCL (linear regression analysis, all comparisons $p > 0.12$; Table 3), liver mercury concentrations were significantly positively correlated with SCL ($r^2 = 0.81$, $p < 0.01$) and weight ($r^2 = 0.83$, $p < 0.01$; Table 3). Plasma copper and plasma zinc were positively correlated to one another ($r^2 = 0.67$).

Constructing backward step-wise regressions to determine if metal concentrations could statistically determine hematologic and plasma biochemical health parameters, significant regressions were created for 18 of the 31 (58.0%) blood parameters (Table 4). Only a single metal contaminant factored into the final equation in 11 of the 18 significant regressions; whereas, 2 metal contaminants factored into 4 of the significant regressions. All 5 metal contaminants did not factor simultaneously into any significant regression. Keratin mercury was a statistically significant factor in 9 of the 18 significant regression equations; whereas, plasma selenium was significant in 7 of the equations. Plasma copper, blood mercury, and plasma zinc added to 5, 5, and 4 regression equations, respectively (Table 4). Of the 18 significant multiple regression equations, half had a power of > 0.80 (creatine kinase, gamma-glutamyl transferase, total protein, albumin, globulin, glucose, sodium, white blood cells, and

Table 1. Mean, standard deviation (SD), and range of straight carapace length (cm SCL), weight (kg), and metal concentrations (wet weight, ng/g) of keratin, blood, liver, and plasma of cold-stunned juvenile Kemp's ridley turtles from Cape Cod, Massachusetts.

	SCL	Weight	Keratin mercury	Blood mercury	Liver mercury	Plasma copper	Plasma zinc	Plasma selenium
n	31	31	29	29	6	16	16	16
Mean	25.9	2.6	389	24	67	690	2290	490
SD	2.7	0.77	239	9	70	680	3550	60
Range	21.8–34.5	1.6–4.6	48–1058	< 5–43	21–210	330–3180	510–13,070	400–610

heterophil count). Conversely, significant multiple regressions could not be constructed for the blood parameters alkaline phosphatase, lactate dehydrogenase, bilirubin, blood urea nitrogen (BUN), cholesterol, calcium, total carbon dioxide, chloride, potassium, BUN:creatinine, hematocrit, monocyte count, and eosinophil count.

Aldrin, alpha-BHC, alpha-chlordane, beta-BHC, delta-BHC, endosulfan 1, endosulfan 2, endosulfan sulfate, endrin, endrin aldehyde, endrin ketone, heptachlor, heptachlor epoxide, and methoxychlor were not detected in any sample (plasma $n = 18$; liver, kidney, fat, and brain $n = 3$ of each tissue). Dieldrin, gamma-BHC, gamma-chlordane, p,p′-DDD, p,p′-DDE, and p,p′-DDT were detected at low concentrations in few turtles (Table 5).

Table 2. Mean, standard deviation (SD), and range of initial hematologic and plasma biochemical values of cold-stunned juvenile Kemp's ridley turtles from Cape Cod, Massachusetts ($n = 26$).

Biochemical component	Mean	SD	Range
Alkaline phosphatase (IU/L)	285	417	40–1646
Aspartate aminotransferase (IU/L)	610	50	184–2531
Creatine kinase (IU/L)	21,979	24,298	3497–100,000
Alanine aminotransferase (IU/L)	26	50	0–257
Lactate dehydrogenase (IU/L)	5694	10,674	292–55,180
Gamma glutamyltransferase (IU/L)	3	2	0–7
Total protein (g/dL)	2.6	0.4	2–3.9
Albumin (g/dL)	1.0	0.2	0.6–1.5
Globulin (g/dL)	1.7	0.3	1.1–2.4
Albumin:globulin	0.6	0.1	0.4–0.8
Blood urea nitrogen (mg/dL)	33	22	12–94
Uric acid (mg/dL)	3.7	2.3	0.1–8.7
Creatinine (mg/dL)	0.25	0.11	0.1–0.5
Blood urea nitrogen:creatinine	137	57	47–246
Glucose (mg/dL)	141	50	19–253
Calcium (mg/dL)	6.6	1.1	4.7–8.7
Phosphorus (mg/dL)	7.4	1.2	5.4–9.9
Chloride (mEq/L)	117	8	100–136
Potassium (mEq/L)	3.2	0.8	2.3–6.3
Sodium (mEq/L)	155	6.0	144–171
Anion gap (mEq/L)	17	11	1–43
Cholesterol (mg/dL)	334	141	25–665
Bilirubin (mg/dL)	0.03	0.04	0–0.1
Total carbon dioxide (bicarbonate, mEq/L)	25	6	12–40
Hematocrit (%)	33	6.3	23–47
White blood cells (10^3 cells/μL)	11.2	4.9	4.5–23.3
Heterophils (10^3 cells/μL)	7.6	3.8	2.2–17.0
Lymphocytes (10^3 cells/μL)	3.1	2.0	0.8–7.4
Basophils (10^3 cells/μL)	0.03	0.05	0–0.2
Monocytes (10^3 cells/μL)	0.5	0.5	0–1.5
Eosinophils (10^3 cells/μL)	0.03	0.06	0–0.16

DISCUSSION

Mean keratin mercury concentrations in this study were approximately half that of Kemp's ridley, green (*Chelonia mydas*), and hawksbill turtles (*Eretmochelys imbricata*) in the Gulf of Mexico, but 64% greater than loggerhead turtles (*Caretta caretta*) in the Gulf of Mexico, and over 7 times greater than black turtles (*Chelonia mydas agassizii*) in the Gulf of California (Presti 1999; Presti et al. 1999, 2000). Mean keratin mercury concentration in this study was similar to that reported for live loggerhead turtles off the southeastern coast of the United States (Day et al. 2005).

Blood mercury concentrations in this study were nearly identical to values reported for larger Kemp's ridley turtles from the Gulf of Mexico (Presti 1999; Presti et al. 1999, 2000; Kenyon et al. 2001). In comparison to other species, mean blood mercury concentration of the Cape Cod turtles was higher than loggerhead, hawksbill, and 1 green turtle from the Gulf of Mexico, and very similar to live loggerhead turtles off the southeastern coast of the United States but approximately 10 times lower than blood mercury concentrations recently reported for leatherback turtles (*Dermochelys coriacea*) in Gabon (Presti 1999; Day et al. 2005, 2007; Deem et al. 2006).

The lack of correlation of blood and keratin mercury concentration to each other, to liver mercury concentration, to carapace length, and to weight in Kemp's ridley turtles from Cape Cod is consistent with several previous reports, and may be because of the relatively small range of turtle sizes in this study. The lack of correlation of mercury concentrations among tissues of the same animal has previously been reported in Kemp's ridley turtles, where there was no correlation between blood mercury and keratin mercury concentrations (Presti 1999). No correlation was found between mercury concentration in blood and keratin, and SCL for Kemp's ridley turtles from the Gulf of Mexico < 42 cm SCL; and only weak correlation was seen in turtles > 42 cm SCL, and black turtles from the Gulf of California (Presti 1999; Presti et al. 1999, 2000). Gordon et al. (1998) found no correlation of liver or kidney mercury concentration with curved carapace length in green turtles from Australia. In contrast, Kenyon et al. (2001) found that blood mercury concentration correlated to SCL of Gulf of Mexico Kemp's ridley turtles, when evaluating a wide range of sizes (21.6–65.8 cm SCL); and Day et al. (2005) found that blood and keratin mercury concentrations correlated to each other

Table 3. Paired simple correlations (*r*, above the diagonal) and *p* values from a linear regression analysis (below diagonal) for metal concentrations of cold-stunned juvenile Kemp's ridley turtles from Cape Cod, Massachusetts.

	Keratin mercury	Blood mercury	Liver mercury	Plasma copper	Plasma zinc	Plasma selenium	Weight	Length
Keratin mercury	—	−0.06	0.36	0.08	0.00	−0.12	0.05	−0.51
Blood mercury	0.57	—	0.61	−0.33	−0.29	0.26	0.54	0.33
Liver mercury	0.64	0.27	—	NA[a]	NA	NA	0.91	0.90
Plasma copper	0.74	0.22	NA	—	0.87	−0.36	−0.15	−0.09
Plasma zinc	0.91	0.29	NA	< 0.001	—	−0.44	−0.24	−0.22
Plasma selenium	0.72	0.34	NA	0.19	0.10	—	0.31	0.18
Weight	0.44	0.14	0.01	0.59	0.04	0.25	—	0.69
Length (SCL)	0.12	0.44	0.01	0.75	0.45	0.49	< 0.001	—

[a] NA = not applicable.

and to other tissue concentrations and body weight in loggerhead turtles in the southeastern United States (weight range 20–140 kg). However, in some studies showing a positive correlation among tissue metals and body size (e.g. Day et al. 2005), much of the positive relationship was driven by the largest animals. In the present study, all animals were of relatively small body size, thus correlations dependent on sampling of large animals may not be apparent.

This is the first report of liver mercury concentrations for Kemp's ridley turtles. In comparison to other species, liver mercury concentrations in the present study were similar to those reported for leatherback turtles, and green turtles from the Torres Strait and Mediterranean Sea but

Table 4. Results of statistically significant backward-stepwise regressions of selected metals (columns 2 through 6) on hematologic and plasma biochemical health parameters (column 1) of cold-stunned juvenile Kemp's Ridley sea turtles from Cape Cod, Masachusetts ($n = 14$). Number in cells indicates *p*-value of metal and (+,–) below the *p*-value indicates the sign of the coefficient (i.e., positive or negative correlation); r^2 and power refer to the overall fit of the resultant regression equation.

	Tissue and metal						
Health parameter	Keratin mercury	Blood mercury	Plasma copper	Plasma zinc	Plasma selenium	r^2	Power
Alanine aminotransferase	0.01 +					0.41	0.75
Aspartate aminotranferase	0.05 +					0.26	0.50
Creatine kinase	0.03 +	0.05 +				0.47	0.83
Gamma glutamyltransferase	0.02 +				0.03 +	0.46	0.82
Albumin	< 0.001 +		0.03 −	0.06 +		0.76	1.00
Total protein	< 0.001 +		0.005 −	0.01 +	0.005 +	0.82	1.00
Globulin	0.01 +		0.01 −	0.02 +	< 0.001 +	0.81	1.00
Creatinine				0.05 −		0.28	0.53
Glucose	0.02 −	0.03 +				0.54	0.90
Phosphorus		0.04 −				0.30	0.56
Sodium		0.05 +			0.03 −	0.54	0.90
Albumin:globulin					0.01 −	0.43	0.78
Uric acid		0.01 +				0.39	0.72
Anion gap					0.03 +	0.30	0.57
White blood cells			< 0.001 +			0.59	0.94
Heterophils			0.005 +			0.46	0.82
Lymphocytes					0.04 −	0.27	0.52
Basophils	0.03 −					0.31	0.59

Table 5. Organochlorine pesticide concentrations of plasma, liver, kidney, and fat of cold-stunned juvenile Kemp's ridley turtles from Cape Cod, Massachusetts. Limit of quantitation = 10 ng/g. Limit of detection not determined.

	Tissue Type				
Organochlorine	Plasma	Liver	Kidney	Fat	Brain
Dieldrin	ND[a] ($n = 18$)	ND ($n = 3$)	ND ($n = 2$) < 10 ng/g ($n = 1$)	< 10 ng/g ($n = 2$) 10 ng/g ($n = 1$)	ND ($n = 3$)
Gamma-BHC	ND ($n = 14$) < 10 ng/g ($n = 4$)	ND ($n = 2$) < 10 ng/g ($n = 1$)	ND(n= 3)	ND ($n = 3$)	ND (n=3)
Gamma-chlordane	ND ($n = 18$)	ND ($n = 2$) < 10 ng/g ($n = 1$)	ND ($n = 3$)	ND ($n = 3$)	ND ($n = 3$)
p,p′-DDD	ND ($n = 15$) < 10 ng/g ($n = 3$)	ND ($n = 2$) < 10 ng/g ($n = 1$)	ND ($n = 3$)	ND ($n = 3$)	ND ($n = 3$)
p,p′-DDE	ND ($n = 13$) < 10 ng/g ($n = 5$)	< 10 ng/g ($n = 1$) 14 ng/g ($n = 1$) 15 ng/g ($n = 1$)	< 10 ng/g ($n = 3$)	51 ng/g ($n = 1$) 54 ng/g ($n = 1$) 209 ng/g ($n = 1$)	ND ($n = 1$) < 10 ng/g ($n = 2$)
p,p′-DDT	ND ($n = 14$) < 10ng/g ($n = 4$)	ND ($n = 3$)	ND ($n = 3$)	ND ($n = 3$)	ND ($n = 3$)

[a] ND = not detected.

generally lower than reported for loggerhead turtles (Davenport and Wrench 1990; Sis and Landry 1992; Sakai et al. 1995; Gladstone 1996; Godley et al. 1998, 1999; Storelli 1998; Anan et al. 2001; Day et al. 2005). The lowest liver mercury concentrations in the present study are similar to mean concentrations reported for green, loggerhead, and hawksbill turtles in Australia (Gordon et al. 1998).

The mean plasma zinc concentration in this study was approximately one-third of the mean whole blood zinc concentration reported for Kemp's ridley turtles in the Gulf of Mexico (Kenyon et al. 2001). Plasma zinc concentrations in this study were, in general, an order of magnitude lower than those reported for liver, kidney, and fat of several sea turtle species (Sis and Landry 1992; Aguirre et al. 1994; Sakai et al. 1995; Gordon et al. 1998; Caurant et al. 1999; Anan et al. 2001; Gardiner et al. 2003). Mean plasma zinc concentration in this study is similar to whole body zinc concentrations of olive ridley turtle (*Lepidochelys olivacea*) hatchlings, and similar to liver and muscle zinc concentrations reported in a leatherback turtle (Davenport and Wrench 1990; Sahoo and Sahoo 1996).

Plasma selenium concentrations have not previously been reported for sea turtles, but concentrations in this study (400–610 ppb) were similar to those reported for whole blood of several species of reptiles (Elsey and Lance 1983; Hopkins et al. 2001; Campbell et al. 2005; Burger et al. 2006, 2007). Although selenium is believed to play a protective role against mercury toxicity in some species, this study did not find a correlation between tissue mercury concentrations and plasma selenium concentrations (Chen et al. 2006).

Plasma copper concentrations in this study were very similar to those reported for whole blood of Kemp's ridley turtles in the Gulf of Mexico (Kenyon et al. 2001) and for fat of several sea turtle species in the Gulf of California (Gardner et al. 2003); they were within the low end of the ranges reported for liver, muscle, and kidney of several sea turtle species (Sis and Landry 1992; Aguirre et al. 1994; Sahoo and Sahoo 1996; Gordon et al. 1998; Caurant et al. 1999; Anan et al. 2001; Gardner et al. 2003).

Organochlorine concentrations were generally not detectable in this study. In rare cases, low concentrations of dieldrin, gamma-BHC, p,p′-DDD, p,p′-DDE, and p,p′-DDT were detected (Table 3). Concentrations were much lower than those reported for juvenile cold-stunned Kemp's ridleys from Long Island, New York, in the 1980s (Lake et al. 1994) and were similar to those found in juvenile cold-stunned Kemp's ridleys from Cape Cod in the 1990s (Keller et al. 2004a). This discrepancy between the Long Island turtles and the Cape Cod turtles could be due to a number of factors, including geographic and temporal differences in environmental organochlorine exposure, and differences in laboratory methods. Over the past 2 to 3 decades, reductions in polychlorinated biphenyls and chlorinated pesticides have been reported for seals in the northwestern Atlantic (Lake et al. 1995; Shaw et al. 2005), and a similar trend may be occurring for Kemp's ridley turtles. The relatively low concentrations of organochlorines found in this study are consistent with concentrations reported for olive ridleys, green turtles, leatherbacks, and some loggerheads, but lower than reported for *Caretta* in other studies (Sis and Landry 1992; Aguirre et al. 1994; Godley et al. 1998; McKenzie et al. 1999; Gardner et al. 2003; Keller et al. 2004b). It should be noted, however, that even very low concentrations of organochlorine contaminants have been associated with changes in health parameters in sea turtles. For example, mean blood total organochlorine levels of only 6.5 ppb for loggerheads have been correlated with changes in hematologic, plasma biochemical, and immune function assays (Keller et al. 2004b, 2006). Several turtles in the present study had organochlorine concentrations within this range. Future researchers studying organochlorine concentrations in free-ranging Kemp's ridley turtles should consider modifying the methodologies used in this study in

order to detect lower concentrations. Specifically, assays should allow detection of concentrations < 100 pg/g, utilize an internal standard rather than an external standard, use negative chemical ionization rather than electron impact ionization, extract and inject larger plasma volumes for analysis, and specifically determine limits of detection. In this study, relatively small volumes of plasma were collected because of concern over extracting excessive blood volume from these debilitated turtles. Because of limited sample size and inability to quantify very low organochlorine concentrations in this study, we were unable to correlate organochlorine concentrations with hematologic and plasma biochemical values.

The presence of mercury in juvenile Kemp's ridley turtles indicates exposure during the early life stages. Although mercury concentrations of Kemp's ridley eggs or hatchlings have not been reported, mercury has been detected in eggs and hatchlings of several sea turtles species (Hillestad et al. 1974; Stoneburner et al. 1980; Sakai et al. 1995; Sahoo and Sahoo 1996). It is, therefore, possible that Kemp's ridley turtles are exposed to mercury during embryonic development, with further mercury accumulation via dietary sources after hatching. Although the diet of pelagic, posthatchling Kemp's ridley turtles is not known, elevated mercury levels have been documented in a number of pelagic cetacean species, indicating that mercury exposure may occur in pelagic environments (Booth and Zeller 2005). Upon entering the juvenile benthic life stage, Kemp's ridley sea turtles are known to feed primarily upon crustaceans (Hardy 1962; Lutcavage and Musick 1985; Bellmund et al. 1987; Morreale and Standora 1991; Burke et al. 1993, 1994). Tissue mercury concentrations of some benthic crustaceans are within the range that may pose a developmental risk to juvenile vertebrates, and elevated mercury concentrations have been found in avian and chelonian species that feed on benthic prey within the coastal habitat and geographic range of Kemp's ridley turtles (Eisler 1987; Brooks et al. 1992; Burger and Gochfeld 1997, 2004; Atlantic States Marine Fisheries Commission 1998; Burger 2002; Burger et al. 2003). Blood mercury concentrations in this study were slightly lower than those shown to affect certain aspects of the immune response of loggerhead sea turtles (Day et al. 2007). However, mean blood mercury concentrations were approximately 5 times higher than levels that may be associated with increased risk for abnormal human fetal neurodevelopment (Mahaffey et al. 2004).

This study is the third to demonstrate significant correlations between contaminant levels and clinical health parameters in sea turtles; however, the pathophysiologic mechanisms that may explain these correlations cannot be determined from this study (Keller et al. 2004b; Day et al. 2007). Day et al. (2007) provided several hypotheses for correlations of blood mercury and health parameters, including cellular damage (e.g., positive correlation between blood mercury and creatine kinase) and reduced immune cell proliferation (e.g., negative correlation between blood mercury and lymphocyte counts). Some correlations found by Day et al. (2007) are supported by the present study (e.g., mercury vs. creatine kinase); whereas, others are not (e.g., positive correlation of mercury and glucose in the present study). In light of the large number of correlations found in this study, it is impractical and perhaps overly speculative to try to explain the nature of each correlation. Future studies should investigate the mechanisms by which exposure to specific contaminants may lead to changes in individual hematologic and plasma biochemical analytes.

Although the present study demonstrates the presence of contaminants in juvenile cold-stunned Kemp's ridley turtles in the northwestern Atlantic, it is not intended to evaluate contaminant exposure as a causal factor for cold-stunning. Although contaminant accumulation could affect the health and behavior of these turtles, further studies are required to critically evaluate this possibility. The hematologic and plasma biochemical values in this study are consistent with those previously reported for cold-stunned Kemp's ridley turtles (Carminati et al. 1994; Smith et al. 2000; Turnbull et al. 2000; Innis et al. 2007). Values for several parameters (e.g. aspartate aminotransferase, creatine kinase, uric acid, BUN) are abnormal in comparison to healthy Kemp's ridley turtles (Carminati et al. 1994; Turnbull et al. 2000; Innis et al. 2007). Upon initial hospitalization, cold-stunned Kemp's ridley turtles are often affected by metabolic and respiratory acidosis; elevated levels of electrolytes, uric acid, and tissue enzymes; and reduced BUN levels (Carminati et al. 1994; Smith et al. 2000; Turnbull et al. 2000; Innis et al. 2007). These changes likely reflect the combined effects of hypothermia, poor perfusion, reduced ventilation, exertion, dehydration, and impaired renal function. In light of this, caution must be exercised in comparing the health parameter correlations found in this study to those correlations found in studies of healthy sea turtles.

In light of the very long potential lifespan of sea turtles, it is possible that accumulation of contaminants over time may become problematic, particularly if high concentrations accumulate in reproductive females and their eggs. Further investigation of the temporal accumulation of contaminants in sea turtles, and their effects on embryonic and juvenile development and behavior are indicated. In addition, studies to identify specific sources of contaminant exposure of sea turtles are warranted.

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