MERCURY CONTAMINATION IN FLORIDA PANTHERS

DRAFT

July 1991

STATUS REPORT
MERCURY CONTAMINATION IN FLORIDA PANTHERS
(JULY 1991)

EXECUTIVE SUMMARY

As a result of the death of an apparently healthy, radio-collared female Florida panther *(Felis concolor coryi)* (FP#27) in Everglades National Park in July 1989, we began to investigate contaminant impacts on this endangered species. Chemical analysis of tissue from the deceased panther revealed an elevated mercury (Hg) concentration of 110 ppm wet wt. in the liver. This was comparable with levels found to be lethal to feral cats (37-145 ppm). Analysis of hair and blood from the dead panther revealed Hg concentrations of 130 and 21 ppm, respectively.

Panthers primarily prey on white-tailed deer *(Odocoileus virginianus)* and feral hog *(Sus scrofa)*. However, in certain areas they also consume various small mammals, including raccoon *(Procyon lotor)*, armadillo *(Dasypus novemcinctus)*, and rabbit *(Sylvilagus* sp.) as a significant part of their diet. Therefore, we believed it necessary not only to study Hg contamination of panthers, but of alternate prey species as well.

Mercury concentrations were determined in tissue samples from 52 free-ranging panthers, primarily those living in the Big Cypress Swamp and Everglades ecosystems, collected opportunistically between 1978 and 1991. Whole blood and hair were routinely collected from living animals. Liver, hair and blood were collected from dead animals at necropsy. Four to seven raccoons were collected from each of nine different locations within representative panther habitat. Muscle (caudal thigh) and liver tissue from each of these animals were analyzed for Hg content.

There were significant differences in levels of mercury in panther liver when compared by geographical location or age. The mean liver Hg level for the younger group of panthers (less than 8 years old) living in southeastern Florida was significantly higher than that from the southwestern part of the State. When only the western group was considered, older animals had significantly higher liver Hg levels than did younger ones. The liver Hg burden was much higher among older animals living in the Fakahatchee Strand State Preserve than the single animal living north of Alligator Alley.

The pattern of distribution by geographical location for hair and whole blood were similar to liver. Average levels of Hg in blood and hair were greatest in panthers from the eastern portion of the range, particularly from the Shark

River Slough area and lowest values were noted in panthers from southwestern Florida north of Alligator Alley. Panthers from Pine Island, Everglades National Park, Fakahatchee Strand, and Raccoon Point in eastern Big Cypress National Preserve all had hair and blood levels significantly higher than those north of Alligator Alley.

Reproductive success in female panthers appeared to be adversely affected by elevated Hg levels. There were significantly fewer surviving kittens for females with blood Hg values >0.5 ppm compared to those females with 0.00-0.25 ppm blood Hg values.

The most probable source of Hg contamination in panthers is via the food chain. The panthers north of Alligator Alley had the lowest levels of Hg in blood, hair, and liver and fed primarily on white-tailed deer and feral hog that occurred at high densities on these largely private and protected lands. Although nothing is known about tissue Hg levels in the hog, Hg levels were less than 1.0 ppm in liver samples from approximately 100 southern Florida deer. Panthers with the highest levels of tissue Hg were those which regularly consumed non-ungulates primarily, raccoons, armadillos, rabbits, and alligators. Panther #27 fed only on small prey during the 17 months that she was monitored. Of her 12 kills recovered, all were raccoons. Mercury levels in the muscle of raccoons varied markedly between the different watersheds and habitats across the panther's range in southern Florida.

The recent drought in the Everglades may also be affecting the Hg levels in panthers. Mean hair and blood levels of Hg increased from 6.3 ppm to 33.1 ppm and from 0.18 ppm to 0.6 ppm, respectively, from pre-drought to drought years. Also, Hg levels in otter livers increased 280 percent during a similar time period.

Increased mortality and lowered reproductive success due to chronic exposure to Hg is probably responsible for lower than expected population densities of panthers in large portions of their range and is likely contributing to the extinction of this endangered mammal.

MERCURY CONTAMINATION IN

FLORIDA PANTHERS

Melody E. Roelke[1], Donald P. Schultz[2],
Charles F. Facemire[3], and Stephen F. Sundlof[4]

INTRODUCTION

Mercury contamination of freshwater fish and alligators (*Alligator mississippiensis*) in various watersheds throughout the State of Florida has recently become a human health issue (Delany et al. 1988, Hord et al. 1990). Health advisories have been issued to curtail the consumption of largemouth bass (*Micropterus salmoides*) in southern Florida and the entire Everglades watershed has been closed to the hunting of alligators due to elevated mercury residues in their flesh. Other species of fish in Florida, including the shark (a saltwater species), have also been found to be contaminated with mercury.

Mercury is accumulated and concentrated in the aquatic food chain and the highest levels occur in the longer lived species at the upper trophic levels (Clarkson and Marsh 1982, Eisler 1987). The Florida panther (*Felis concolor coryi*) is a top terrestrial mammalian carnivore in the southern Florida ecosystem. White-tailed deer (*Odocoileus virginianus*) and feral hog *(Sus scrofa)* are the preferred prey, but, in some areas, panthers also consume small mammals, e.g., raccoon *(Procyon lotor)* armadillo *(Dasypus novemcinctus)* and rabbit *(Sylvilagus sp.)*, as a significant part of their diet (Maehr et al., 1990, Roelke et al., 1986). Additionally, two adult male panthers in the Everglades National Park (ENP) have been documented to consume alligators (O. Bass, pers. comm.).

We first became aware of mercury contamination in the Florida panther when a 3- to 4-year-old adult, radio-instrumented female (FP#27) died in the ENP in the summer of 1989. Her carcass was retrieved 24-36 hours postmortem. Both gross and histopathologic examination were unremarkable, although brain tissue was too autolyzed for a definitive examination. The relatively good condition

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[1]Florida Game and Fresh Water Fish Commission, Wildlife Research Laboratory, 4005 S. Main Street, Gainesville, Florida 32601

[2]U.S. Fish and Wildlife Service, 75 Spring Street, SW., Suite 1276, Atlanta, Georgia 30303

[3]U.S. Fish and Wildlife Service, P.O. Box 2676, Vero Beach, Florida 32961

[4]Dept. of Infectious Disease, College of Veterinary Medicine, University of Florida, Gainesville, Florida 32610

of the carcass and lack of significant pathologic findings coupled with concern over the loss of a prime breeding-age female (one of only three in the ENP), prompted a more extensive examination, with selected tissues analyzed for pesticides, PCB's, and heavy metals. The only contaminant found to be present in significant levels was mercury. The liver contained 110 parts per million (ppm) of mercury. For comparison, death due to mercury toxicosis was reported in feral domestic cats with liver concentrations of 37-145 ppm (Harada and Smith, 1974).

This report presents the magnitude and distribution of mercury levels in various tissues from free-ranging Florida panthers sampled over the past 13 to 14 years. Mercury concentrations in selected tissues from white-tailed deer, raccoon, bobcat, otter, and alligator are also reported.

MERCURY IN SOUTHERN FLORIDA ECOSYSTEMS: HISTORICAL PERSPECTIVE

Mercury contamination of Florida's ecosystems has not been studied intensively until recent times. Early in the 1970's, an ENP survey measured mercury concentrations ranging from 0.05 ppm in fish to 2.62 ppm in white ibis *(Eudocimus albus)* (ref). The U.S. Fish and Wildlife Service (FWS) conducted a study in 1986 of contaminant concentrations in selected fish and bird species inhabiting the Loxahatchee National Wildlife Refuge (LNWR). Mercury concentrations in anhinga *(Anhinga anhinga)* livers ranged from 0.42 to 2.72 ppm and in largemouth bass whole body mercury concentrations ranged from 0.56 to 1.05 ppm. Largemouth bass and bullheads *(Ictalurus* spp.) were again collected from ENP and LNWR in April and May 1989, respectively. Fillets from ENP bass had levels ranging from 0.26 to 3.53 ppm, and averaged 0.96 ppm. Yellow bullhead (*I. natalis*) fillets from ENP were similar to the ENP bass with a mean concentration of 0.91 ppm (range: 0.64-1.17). At LNWR, the mean concentration in largemouth bass fillets was 0.81 ppm (range: 0.35-2.13). Concentrations in brown bullheads (*I. nebulosus*) from LNWR averaged 0.51 ppm. As a result, fishery advisories were posted at both ENP and LNWR.

During the fall of 1989, additional fish, both freshwater and marine species, were collected from ENP. Mercury concentrations ranged from a low of 0.056 ppm in a gray snapper (*Lutjanus griseus*) to a high of 1.90 ppm in an oscar *(Astronotus ocellatus)*. Mean concentrations for those species analyzed were: oscar=1.28 ppm, blue gill *(Lepomis macrochirus)*=1.01 ppm, Crevalle jack *(Caranx hippos)*=0.29 ppm, spotted sea trout *(Cynoscion nebulosus)*=0.29 ppm, and gray snapper=0.15 ppm. Ten largemouth bass were also collected from Big Cypress National Preserve (BCNP). Mercury levels in fillets from these fish ranged from 0.51 to 2.91 ppm with a mean of 1.01 ppm. The FWS sampled fish from several refuges, including LNWR and Florida Panther National Wildlife Refuge, the following year. Mercury concentrations detected in at least some fish from each of the refuges sampled exceed the Florida HRS Health Advisory for once/week consumption (0.5 to 1.5 ppm).

None of these studies, however, addressed the source of mercury contamination in Florida's aquatic ecosystems or the impact that biomagnification of methylmercury (the most toxic organic form of mercury) through the food chain may be having on top predators.

Mierle (1990) stated that atmospheric combustion pollutants are the primary source of mercury contamination in lake fish in Canada. He determined that at least 57 percent and, perhaps, as much as 70 percent, of the total mercury input into a central Ontario (Canada) lake was in the form of wet deposition (rain and snow). Atmospheric deposition was not considered a serious potential source of mercury in Florida waters in the 1989 Florida panther status report (USFWS 1989). There are, however, other sources which are evident in Florida. For example, it is estimated that approximately nine tons of mercury were emitted into the atmosphere by solid waste incinerators in Florida during 1989 (T. Rogers, Florida DER, pers. comm.). It is known that plants take up mercury from the soil (Schacklette 1980), and Simons (1991) estimated that over 10 tons of mercury is released into the atmosphere annually from burning and processing sugar cane. However, this figure was based only on mercury content of the cane (0.03 to 1.2 ppm), and did not consider the possibility of mercury on the external surface of the cane as a result of atmospheric deposition. The residence time for atmospheric mercury, of which some 25 to 30 percent is of anthropogenic origin, has been estimated to be somewhere between 6 and 90 days by Clarkson et al. (1984).

The oxidation of the natural peat soils of south Florida has been hypothesized as another source of mercury contamination (USFWS 1989). Simons (1991) noted that the South Florida Water Management District found mercury concentrations in peat ranging from 0.1 to 0.3 ppm. Assuming (1) that on the average, 1.12 inches of peat is lost to oxidation annually, (2) that the average mercury content of peat is 0.2 ppm, (3) that oxidation occurs uniformly over the approximately 430,000 acres of peat soils in south Florida, and (4) that methylation of mercury is occurring in peat as a result of natural biological activity associated with oxidation, then approximately 8.2 tons of methylmercury could be released from peat deposits annually (Simons 1991). However, mercury levels in sediment cores from lakes and estuaries have increased twofold to fivefold since precultural times (Eisler 1987), and that same phenomenon is likely true of peat sediments. Thus, it is likely that mercury concentrations in the remaining peat will increase over time due to wet deposition of atmospheric mercury.

MATERIALS AND METHODS

Tissue collections:

Tissue samples from 52 (29 male and 23 female) free-ranging Florida panthers were collected opportunistically between 1978 and 1991. Hair and whole blood from living panthers (n=43) were sampled during routine capture or when removed from the wild for rehabilitation (Roelke 1990). Liver, kidney, muscle, brain, hair, and blood were collected at necropsy from dead panthers (n=24). Seventeen of the dead panthers had also been sampled when alive. All panthers were collected in southern peninsular Florida. Forty-one individuals were collected from the Fakahatchee Strand/Big Cypress Swamp (Collier and Hendry Counties) and nine from the ENP (Dade and Monroe Counties). Two other

individuals were collected outside this range in Glades and Palm Beach counties. For discussions in this paper, panther locations within the Big Cypress Swamp and Everglades physiographic region of southern Florida, are described as follows (roughly from northwest to southeast): (1) land north of Alligator Alley/I-75 including the Florida Panther National Wildlife Refuge, Bear Island Unit of the BCNP), and adjacent private ranches to the north and east (NA); (2) the Fakahatchee Strand State Preserve (FS); (3) Raccoon Point (RP) area in eastern BCNP (now part of Corn Dance Unit); (4) Shark River Slough (SS) area of ENP (includes east Everglades (EE)); and (5) Long Pine Key/Hole-in-the-Donut (LPK) area of ENP which also encompasses Taylor Slough (Figure 1).

Non-panther carnivore location and samples:

Muscle and liver tissues were collected from four to seven raccoons captured in 1990-1991 at each of nine sites (**n=48**, total) within representative panther habitat. The capture sites included those described for the panther with the addition of the following: Loop Road (LP) Unit of BCNP; Water Conservation Area 3A (3A); Taylor Slough (TS), ENP (part of LPK); and Flamingo Visitor Center, ENP (FL) (Figure 1). The majority of the raccoons were captured in April to May 1990 with the exception of the TS, FL, and three of five LPK raccoons which were captured in the spring of 1991.

Liver tissue from 23 road- and hunter-killed bobcats were collected from areas similar to the panther collection sites, with the addition of Card Sound Road Southeast of ENP (Figure 1). Most of the bobcats were collected in 1984-85. All 20 otters were road-kills, and, therefore, the location descriptions may vary slightly from above (Figure 1); e.g., NA included animals killed along SR 29 north of I-75; a "far west" (FW) sample included animals from the Collier Seminole State Park area on the far western edge of panther range; FS includes animals killed on SR 29 south of I-75 as well as those killed on SR 41 from the western Fakahatchee Strand State Preserve boundary east to Turner River Road; the Oasis (OA) Ranger Station sample includes road-kills along SR 41 from Monroe Station east to 50-mile-Bend; and the ENP sample is from Chikika State Park on the east side of the ENP/east Everglades. The otters were collected during two time periods, 1984-1985 and 1989-1991. Muscle samples from road-killed alligators (**n=4**) were collected in the NA and OA areas in June 1990. Five additional alligators were live-captured in SS and surgically biopsied to obtain muscle tissue.

Tissue analysis:

All samples were stored at either -10^{0}C or -75^{0}C until submitted for analysis. The primary laboratory used was the Patuxent Analytical Control Facility (US Fish and Wildlife Service) in Laurel, Maryland. Tissue sample homogenates were digested under reflex in sulfuric and nitric acids (Monk 1961). Total mercury concentrations were determined by cold vapor atomic absorption spectrophotometry (Hatch and Ott, 1968) using a Spectro Products mercury analyzer equipped with a Varian VGA-76 vapor generation accessory. A limited

number of samples were similarly analyzed by Brooks Rand, Inc. in Seattle, Washington, and the Florida Game and Freshwater Fisheries Research Laboratory in Eustis, Florida. Split samples were used to validate results received from all laboratories. Muscle and liver tissue samples from other carnivores (bobcats and alligators) were analyzed at the University of Florida, Gainesville, Florida, using similar techniques.

Methylmercury analyses were performed by Brooks Rand, Inc., in Seattle, Washington. Tissues were digested in KOH/methanol to release the methylmercury. The methylmercury was then ethylated, subjected to cryogenic gas chromatography, and quantified by a cold vapor atomic fluorescence detector.

Data analysis:

All tissue results in this paper are reported in ppm of mercury on a wet weight basis. The data for mercury concentrations in tissue appeared to fit a log-normal distribution so they were log-transformed prior to data analysis. Therefore, all reported means are geometric means (GM) rather than arithmetic. When comparisons involved more than two means, one-way analysis of variance (ANOVA) and the Student-Newman-Kuels multiple range test were used to detect differences; otherwise, differences between means were detected using Student's "t" test or the Paired-sign test as appropriate. Linear regression analysis was used to develop predictive models for mercury concentrations in liver and blood. The significance of correlations in regression analyses was calculated using ANOVA.

Mercury levels determined in alternative blood products (saline washed red cells, whole blood clots, or hemolyzed postmortem blood) were used in a limited number of cases to calculate the blood mercury (HgWBCALC) concentration for panthers lacking archived EDTA or heparinized whole blood.[5] There was no significant difference ($p>0.5$) between the means of paired

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[5]**This was accomplished by determining the amount of mercury per unit of hemoglobin (heme) in the non-whole blood sample (97 percent of the Hg in blood is bound to heme (Albanus, et al. 1972)) followed by multiplying that value by the actual heme concentration in the original sample, if known, or by using the heme value of 10.0 g/dl (conservative mean for heme (Roelke 1990)). The results from red blood cell (RBC) and clot determinations were further multiplied by 1.03 to account for the added 3 percent Hg that would have been contained in the plasma.**

$$\text{HgWBCALC} = \frac{\text{ppm Hg in blood product}}{\text{heme gm/dl in blood product}} \times \text{Actual or estimated heme gm/dl in original whole blood sample} \times \text{RBC/Clot } 1.03$$

samples of blood and calculated blood mercury levels based on either red blood cells or clots, therefore, mercury results from blood and HgWBCALC were used interchangeably as "whole blood" in analyses in this paper. Calculated results from post-mortem serum could not be validated directly with paired blood samples and were only utilized to compare with hair and liver samples.

RESULTS AND DISCUSSION

Mercury levels in panther tissues:

Table 1 shows the concentration of mercury in various panther tissues from differing geographical locations.

Liver levels:

The total mercury values for liver samples from dead panthers are shown in Figure 2. There are striking differences when these data are examined by location and age of the panther. The mean liver mercury level for young panthers (less than eight years old) living in southeastern Florida was significantly higher than those located in southwestern Florida ($p=0.024$, geometric mean (GM) east=25.8 ppm, GM west=0.304 ppm). If only the southwestern group is considered, older animals had significantly higher liver mercury levels than the younger ones ($p=0.029$, GM old=14.6 ppm, young=0.304 ppm). The mercury burden was much higher in old animals living in the FS (19-20 ppm) than in the one older panther in NA (7.8 ppm). Old panthers have not been found in the southeastern portion of the panthers range.

Predictive models:

We utilized hair to develop a model for predicting mercury liver concentrations in living animals. There was a significant, positive correlation ($R=0.89$, $p<0.001$) between hair and liver collected from individual dead panthers (Figure 3). This also has been reported for bobcats and raccoons (Cumbie 1975). Thus, it is theoretically possible to predict liver mercury concentration from concentrations in hair collected antemortem. For example, if a liver mercury concentration of 35 ppm is considered the lower threshold for toxicity in the domestic cat, the model would predict that panthers with hair mercury levels of >57.3 ($\pm$ 95 percent C.I.) are in the toxic range. The more conservative "10 percent rule" level (10 percent of the mean toxic level) would predict that panthers with hair levels of >12.57 ppm are at risk.

Clinical toxicologic data exist for blood from the domestic cat (Charbonneau et al., 1974). From these data, one may predict mercury body burdens and potential toxicosis expected at different blood concentrations. Figure 4 demonstrates a significant, positive correlation ($R=0.75$, $p<0.001$) between mercury in blood and hair for the Florida panther. Therefore, given a hair sample, it might be possible to predict clinically relevant liver and blood

mercury concentrations. The direct relationship between blood and liver mercury concentration in the panther is strengthened by examining post-mortem blood/hair and blood/liver correlations (Figures 5 and 6) both of which are positive and significant; $R=0.98$, $p<0.001$ and $R=0.78$, $p<0.01$, respectively.

A limited number of fecal samples (n=7) were examined to determine if feces could be used as a possible means of monitoring mercury exposure and/or excretion in free-ranging panthers without the necessity of immobilization. There was a significant, positive relationship between the blood mercury level and the fecal concentration ($R=0.78$, $p<0.05$) (Figure 7). It is not known how much of the ingested mercury bound in the hair of the prey animal is absorbed by the panther and how much is passed in the feces. Likewise, nothing is known about rate of excretion of previously ingested mercury from contaminated prey. With additional analysis of fecal mercury it may be possible to obtain a rough estimate of the relative risk that an individual panther is experiencing.

Figures 8, 9, and 10 demonstrate the relationship of mercury in liver compared to three other organ systems; muscle, kidney, and brain, respectively. With these regressions, it is possible (with limited sample type) to predict potential toxic levels for selected panthers.

Distribution of mercury in whole blood and hair of panthers by location:

Mean mercury concentrations in hair and blood from living and dead panthers were utilized to assess the geographical distribution of mercury contamination (Figures 11 and 12). The pattern of distribution by location for both hair and blood is similar to that seen in the liver (Figure 2). The highest levels occurred in panthers in SS (GM: hair=55.532 ppm, GM: blood=1.986 ppm). The lowest levels were found in panthers living to the northwest in NA (GM: hair=1.77 ppm, GM: blood=0.089 ppm). Both the hair and blood mercury levels from NA were significantly lower than panthers from SS, LPK, and FS ($p<0.01$). Further, mercury levels in hair from SS panthers was significantly higher than from all other panthers ($p<0.01$) whereas blood of SS animals was not different from those in FS, but was different from LPK ($p<0.05$) panthers. RP, with only two samples, could not be included in the analysis of variance, but the mean for both hair and blood fell between FS and SS.

Southwestern Florida - Mercury concentrations north vs. south of I-75:

Panthers in the FS had significantly higher hair and blood mercury concentrations than did the panthers directly to the north across State Road 84 (now Interstate Highway I-75) (FS GM: hair=7.18 ppm, GM: blood=0.384 ppm vs. NA GM: hair=1.77 ppm, GM: blood=0.089 ppm) (Figure 1). In the fall of 1987 management actions designed to increase ungulate density and availability to the panthers were implemented in the FS. These actions included implementation of fire as a habitat management tool, creation of experimental food-plots, salt-licks, and feeders for deer, and enhancement of law enforcement efforts to curtail illegal killing of deer, and closure

of the area to public hunting. There has been a precipitous drop in mercury levels in the FS panthers coincidental with the above actions. Figure 13 displays the comparison of the mean mercury concentration in panther blood from FS and NA before and after 1987. There was a significant difference ($p<0.001$) in blood mercury values between the FS and NA prior to 1987, however, the values after 1987 were not significantly different. Further, there was no significant difference in either blood or hair values between the two time periods within the NA, while a significant difference ($p<0.005$) did exist within the FS. These data suggest that deer and hog abundance and availability was, and is, a major factor influencing diet composition and subsequent levels of mercury in panthers in the FS.

Prior to fall, 1987, panthers living in the area FS were generally underweight and anemic, had poor reproductive success, and consumed primarily raccoons and armadillos (Roelke 1986). The poor condition was considered to be primarily a nutritional problem. As mentioned above, it has now been determined that all panthers living in the FS during that period had elevated mercury levels when compared to the individuals living there since. Figure 14 displays the individual hair and blood mercury values for FS panthers and highlights the one female (FP#09) present throughout the entire period. Since 1987, FP#09 has gained 13 percent in body weight (10 lbs), has been documented killing deer (as opposed to only raccoons previously), and has experienced a 77.2 percent drop in blood mercury levels (1985=0.630 ppm, 1987=0.598 ppm, 1988=0.363 ppm, and 1990=0.140 ppm). Additionally, FP#09's two surviving 1990 offspring had considerably lower blood mercury values than did her only other surviving offspring born in 1985 (0.140 ppm and 0.170 ppm vs. 0.749 ppm, respectively).

Methylmercury composition:

A limited number of samples were submitted for methylmercury determination (Tables 1 and 2). The mercury in panther blood, hair and brain is virtually 100 percent methylmercury (range 61-119 percent), whereas liver and kidney are considerably less. Methylmercury is the neurotoxic form of mercury, therefore additional tissue types should be examined to more fully understand the partitioning and potential excretion routes of mercury within the panther.

Reproductive success:

Reproductive success in female panthers also may be adversely affected by elevated mercury levels in conjunction with sub-optimal nutritional status. Figure 15 displays the average number of surviving young (>6 mos.) per female year grouped by the mother's blood mercury level at the approximate time of pregnancy. There is a significant difference ($p<0.01$) in the number of surviving kittens for females with blood mercury values >0.5 ppm (mean=0.167 kittens/female yr) compared to those females with <0.25 ppm blood mercury values (mean 1.46 kittens/female yr). Mercury passes through the placenta and is concentrated in the fetus at levels equal to or higher than, in the mother (Khera, K.S., 1974). Further, the nervous tissue of the fetus is far more

sensitive to effects of mercury than the adult nervous tissue. Even low mercury levels in the dam have resulted in profound neuronal disarray in the offspring when the exposure occurs during critical early stages of development (T. Clarkson, pers. comm.). The disruption of normal fetal development has been documented to cause abortions, stillbirths, congenital anomalies, and behavioral changes resulting in early neonatal death (Khera, K.S., 1974). Unfortunately, no neonatal kitten carcasses have ever been retrieved nor have kittens younger than 6 months of age ever been examined. Therefore, these serious potential developmental problems cannot be ruled out in the panther, particularly for kittens born to females with elevated mercury levels.

Comparison of blood from surviving dependent kittens and their dams demonstrate that there is a significant positive correlation between the two ($R=0.59$, $p<0.05$) (Figure 16). This regression predicts that dams with low blood mercury concentrations have offspring with low levels (e.g., dam=0.015 ppm - offspring=0.06 ppm) and females with elevated levels also have offspring with increased levels (e.g., dam=1.0 ppm - offspring=0.4 ppm).

There is undoubtedly a nutritional component contributing to the documented lowered reproductive success as well. In the absence of an adequate prey base such as deer and hogs, the panther will take less desirable prey such as raccoons which may contain elevated levels of mercury and possibly a lower caloric value. Further, starvation may also mobilize mercury stored in the muscle tissue; thus the two problems may be coupled.

Potential effects of mercury on panther behavior:

A diet of contaminated prey (e.g., raccoons) may cause the panther to be unable to stalk and kill deer effectively. The panthers would then, of necessity, be forced to continue consumption of ~~raccoons~~ such prey, thus maintaining their poor nutritional status and increased reproductive failure (W. Buck, pers. com.). Tissue burdens of methylmercury also probably play a role in deaths caused by automobile accidents since methylmercury primarily effects the sensory functions of the central nervous system and motor coordination. Methylmercury may compromise night vision by constricting the visual fields and also causing loss of hearing. Damage to the cerebellum leads to ataxia manifested by a staggering type of gait and loss of movement coordination (T. Clarkson, pers. com.).

Changes in panther behavior can potentially occur with even low levels of mercury exposure. A recent behavioral study of domestic kittens fed a diet low in mercury (0.55 ppm dry wt) demonstrated a significant depression in the amount of time they spent playing, climbing, and vocalizing (Houpt et al., 1988).

Source of mercury for the panther:

Initial panther data from 1989 showed that the highest levels of mercury in panthers occurred in areas where raccoon and other small prey were consumed (USFWS, 1989) (Figure 17). The hypothesis that the source of mercury contamination in panthers was via the food chain has now been substantiated by the analysis of prey species (raccoon and alligator) and by a comparison with other carnivores in the same ecosystem with different feeding habits e.g., bobcat and otter.

Panthers in NA have the lowest levels of tissue mercury and feed primarily on white-tailed deer and feral hogs that occur at relatively high densities on these largely private, protected lands. Although little is known about tissue mercury levels in the hog, mercury concentrations in southern Florida deer liver (n=approximately 100) were all less than 1 ppm (Figure 18) (D. Forrester and S. Sundlof, unpublished data). Panthers with the highest levels of tissue mercury are those that consume non-ungulates; primarily raccoons, armadillos, rabbits, and for a couple of adult male panthers, alligators. When initially captured, FP#27 had hair and blood mercury values of 46.0 ppm and 1.7 ppm respectively. During the following 15 months of monitoring, she fed only on small prey (O. Bass, ENP, pers. comm.). Of 12 documented kills, all were raccoons. At death, her tissue mercury values were 110, 130, and 21 ppm for liver, hair, and blood, respectively.

Relationship of mercury in raccoon tissue to panther tissue:

Mercury in the muscle and liver of raccoons varies markedly between the different watersheds and habitats across the panther's range in southern Florida (Figures 19 and 20).

The overall pattern of distribution of mercury concentration in raccoon muscle is very similar to that in the blood of panthers living in the same locations (Figures 2, 11, and 12). The highest values for both species occur in SS and adjacent land whereas the lowest levels for both occur in NA. The dynamics of mercury distribution and concentration in the raccoon as it relates to its food source is unknown at this time.

At this writing only two panthers have been sampled in RP. The Hg levels of both are higher than expected if only deer were being consumed. For example, the hair and blood values of the older female (FP#38) were elevated at the time of initial capture in 1990 (47.49 ppm and 0.635 ppm respectively), yet investigation of her kills indicate that she had eaten primarily deer since capture (D. Jansen, BCNP, pers. comm.). This apparent discrepancy may be related to her periodic forays into Water Conservation Area 3A and probable consumption of raccoons whose muscle levels of mercury are twice that of those in RP (Figure 19).

Potential toxic effect of consumption of raccoon:

Considerable information exists in the domestic cat literature regarding toxicity experienced from consuming differing levels of mercury for varying lengths of time. Table 3 presents a summary of dosages and the cumulative time for clinical effects to be seen in domestic cats (Charbonneau et al, 1976, Buck et al., 1987). A tentative model for panther toxicosis was generated by extrapolating from the above literature based on the amount of mercury contained in raccoon muscle from the different sample locations (Table 4). This model assumes that an adult female panther (≈75 lb.) consumes approximately 6 lbs. of muscle from the average raccoon. The model assumes a daily consumption of one raccoon per day and the mg of mercury/kg of panther/day was then calculated. This "dosage" rate was compared to that of the domestic cat to project time to clinical effect. The model does not consider the mercury contained in the raccoon hair, hide, and organs. Several of the female panthers with the highest levels of mercury weigh less than the 75 lb. model panther, thus, if they consumed the same amount of raccoon/day they would be exposed to more Hg per kg/body weight.

The area with the shortest projected time-interval for the occurrence of clinical toxicosis includes the same area where panthers have been identified as having the highest concentrations of mercury, i.e., SS (Table 4). The model is still speculative at this point, for we know little about the dynamics of mercury toxicosis in the panther. However, the panther who died of mercury moved from the EE into the middle of SS and consumed only raccoons for approximately three months prior to her death (O. Bass, pers. comm.). This time frame compares favorably with the estimated time of 14 weeks required to show signs of toxicosis at that level of mercury consumption.

Non-raccoon sources of mercury:

Other potential food sources of mercury for the panther include alligators and otters. The two adult males that lived in, or traversed, SS regularly killed alligators estimated to be 4-5 ft. in length (O. Bass, pers. comm.). These males have the highest blood values recorded for any living panther (up to 3.4 ppm) and hair values approaching that of FP#27 (up to 100 ppm). Muscle from alligators collected in SS had higher levels of mercury than did raccoons from SS (GM 2.90 vs. 2.3 ppm) and as previously noted, animals in SS had higher levels than those in adjacent areas (Table 2, Figure 22). There appears to be a linear relationship between the length of the alligator and the amount of mercury in the muscle (Figure 23), but even small (<3 ft.) alligators had considerable levels of muscle mercury (1.66 ppm).

During the past year, FP#16 has apparently shifted his diet away from alligators and is consuming primarily deer (O. Bass, pers. comm.). His blood mercury level in February 1991 was only 0.21 ppm compared to 3.4 ppm the year before. His hair level has not dropped as markedly (down to 60 ppm from 90 ppm), but the former value may be representative of hair that was grown in the past, has not yet shed and reflects mercury being recycled within the body.

This male has apparently learned to kill deer and no longer relies on the mercury contaminated raccoon and otter of his adolescent days or the alligator of his young adult life.

Another way to validate the food source origin of mercury for panthers (raccoons) is to examine a different terrestrial carnivore, the bobcat. Published reports of bobcat food habits (Maehr and Brady, 1986), indicate that they feed on herbivorous cotton rats and marsh rabbits. It was predicted that the bobcat, like the deer, would have low mercury levels. Of the 23 bobcat livers examined, all had liver values less than 1 ppm (with the exception of one animal in 1990 from SS) (Table 2, Figure 24).

The otter, another mammalian carnivore (also a panther prey item in the ENP), is an obligate aquatic food chain forager, consuming primarily fish and crustaceans. The concentration of mercury in otter liver is 10-20 times that found in the bobcat (Table 2, Figure 25). The disparity between bobcats and otters is like that seen among panthers from different locations; the liver mercury concentrations in young panthers which consume terrestrial prey (NA) are very similar to the bobcat, whereas the mercury concentration in livers of those panthers from areas where non-ungulate prey are taken mimic the otter levels (FS<1987 and SS/EE).

The pattern of regional mercury contamination in the otter is similar to that of the raccoon and panther, supporting the hypothesis that the highest level of environmental mercury contamination is associated with the aquatic areas of the Everglades.

Absolute levels of liver mercury in the otter are distinctly lower than raccoons from the similar area (e.g., otter (OA) 5.2 ppm vs. raccoon (RP/LP) 16.0 ppm). This may indicate differential partitioning of mercury within the bodies of the respective animals and is based on published values of liver vs. muscle values for raccoon and otter (Eisler 1987). The ratio of mercury for liver:muscle is approximately 12-13:1 for raccoon and only 23:1 for otter. Therefore, if actual raccoon muscle values for RP and LR, 0.5 and 1.5 ppm, respectively, are compared to the estimated otter muscle value from OA (2.0 ppm), mercury in the otter actually exceeds that of raccoon. Muscle from Florida otters needs to be tested to validate this assumption.

Drought:

The drought in the Everglades ecosystem (1989-1991), may also have affected the amount of mercury accumulating in panthers. Comparison of mean hair and blood mercury values for LPK panthers prior to the drought (Dec. 1986-June 1988) vs. the two subsequent years (July 1989-Dec 1990) show a marked increase in mercury values: hair from 6.353 ppm (n=5) to 33.10 ppm (n=2) and blood from 0.184 ppm (n=10) to 0.671 ppm (n=3). The small sample sizes

do not allow statistical comparisons, however other data also support this hypothesis. Mercury levels in otter liver tissue collected from the eastern edge of the Big Cypress National Preserve in 1989-1991 have increased 280 percent over liver collected in 1984-85 ($p<0.05$) (Figure 25).

Sentinel species like the raccoon and otter should be monitored for yearly wet season/dry season changes as well as longer term drought/non-drought cycles to determine the cycling of mercury in southern Florida mammalian carnivores as models for the endangered Florida panther.

CONCLUSIONS

The significance and potential long term detrimental effects of mercury, no matter the source, in certain portions of the wild panther population should not be minimized. With so few Florida panthers, every factor that results in the depression of reproductive fitness and/or increased mortality will jeopardize the continued existence of the panther in Florida.

Mercury has been identified in this study as being particularly high in those areas associated with the historic Everglades drainage from Lake Okeechobee; 3A, the SS, and LR, with lower, but yet still significant levels, on adjacent lands. Mercury contamination may possibly be responsible for the exceedingly low density of panthers on the public land south of Alligator Alley (currently only six panthers are thought to exist there).

The apparent positive affect on panther health and reproduction afforded by the enhanced prey management in the FS since 1986 suggests that similar management actions may be applied to other selected critical areas within BCNP and EE. As prime panther habitat, largely in private ownership, continues to diminish, the ability to increase the numbers and health of panthers and to recolonize depleted areas of public land will have great bearing on the chances of recovery and maintenance of free-ranging panthers in southern Florida.

ACKNOWLEDGEMENTS

We wish to thank the veterinary assistants, field biologists, and technicians who assisted with the panther captures and/or collection and processing of samples: Steve H. Parker, Toni K. Ruth, Andrena J. Anderson, Marnie Lamm, David S. Maehr, Oron L. Bass Jr., John Roboski, Robert C. Belden, J. Walter McCown, Jayde C. Roof, E. Darrell Land, Deborah K. Jansen, Roy T. McBride and Jeff Wentworth. We wish to thank Homer E. Royals and Ted R. Lange of the FGFWFC Fisheries Laboratory in Eustis, Florida for conducting some preliminary analyses for this study. Special thanks go to William Buck, DVM, University of Illinois, Champaign, Illinois and Thomas Clarkson, Ph.D., University of Rochester, Rochester, New York, for their contributions, and to Joan Forrester Collier for her perseverance in typing the numerous drafts and final document. This project was funded in part by federal grant-in-aid funds administered through the U.S. Fish and Wildlife Service under Section 6 of the Endangered Species Act of 1973 (PL No. 93-205), the Florida Panther Research and Management Trust Fund, and the Nongame Wildlife Trust Fund.

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RECOMMENDATIONS

The Florida Panther Technical subcommittee recommends that the following actions be taken:

A. MANAGEMENT ACTIONS

1. Take action through public use regulations to increase the numbers of noncontaminated prey such as deer (and hogs where appropriate) in areas identified as mercury "hot spots". The GFC and DNR have already closed the Fakahatchee Strand State Preserve to hunting for deer and hogs. The East Everglades area has been designated for addition to the Everglades National Park. Consideration should be given to tightening public use regulations if a lengthy delay is expected until the area achieves National Park status.

2. Conduct habitat management actions that will increase the numbers of non-contaminated prey species. Specific areas that should be emphasized include:

   A. Fakahatchee Strand State Park. The DNR's existing land management program should be expanded, where appropriate.

   B. Florida Panther National Wildlife Refuge. The FWS has implemented an aggressive habitat improvement program for deer. It should continue to be given a high priority.

   C. Everglades National Park. The National Park Service plans to restore the natural ecosystem in the Hole-In-The-Donut area that is currently overrun with exotics. This restoration plan should be given a high priority.

   D. Big Cypress National Preserve. The National Park Service needs to aggressively implement a natural wildlife habitat management plan to increase the natural abundance and diversity of deer habitat. The existing fire management program should be expanded for wildlife purposes.

3. Place further emphasis on the acquisition of the Golden Gates Estates for habitat management to increase the population of non-contaminated prey in the area adjacent to the Fakahatchee Strand "hot spot". Develop a management agreement between the State and FWS for management of these lands for the Florida panther by the Panther National Wildlife Refuge upon State acquisition.

4. Implement land management actions to enhance the ungulate prey base for the panther on all land presently in public ownership or scheduled for future acquisition.

5. Work with private landowners north of I-75 to manage lands to protect and preserve panther habitat.

B. ADDITIONAL TESTING ACTIONS

1. Determine the half-life of methylmercury in the panther (using a non-Florida panther) by dosing with a single dose of methylmercury. Half-life to be determined by sampling scat on a regular basis; blood and hair-pre-treatment; within one week of treatment; and monthly thereafter for six months.

2. Determine the bioavailability, to the panther, of methylmercury from animal hair using rat and raccoon hair containing radio-labeled methlymercury. The hair from animals treated with methylmercury to be fed to a non-Florida panther. A mass balance study, using primarily scat from the treated panther to be conducted to determine the fate of the methylmercury.

3. Determine accretion and excretion rates of mercury in a non-Florida panther using archived raccoon tissue.

4. Determine the amount of total mercury and methylmercury in liver, muscle, hair, and brain from five raccoons and five otters.

5. Determine the amount of total mercury and methylmercury in panther scat.

6. Determine mercury levels in liver and muscle from six raccoons annually from Raccoon Point, Loop Road, and the Shark Slough/East Everglades area (use 3A as an alternate site for the latter). Raccoons from other sites to be monitored in even-numbered years.

7. Continue to monitor food habits of panthers. Analyze scat samples to determine potential intake and excretion of mercury. Relate mercury levels to behavioral patterns when feasible.

8. Determine levels of mercury in blood and hair of all panthers captured. When panthers are held in captivity, continue to monitor at selected intervals to determine rate of excretion of mercury.

9. Determine the presence or absence of mercury in selected tissues (liver and muscle) from feral hogs taken from the various panther habitats.

10. Monitor all panthers with levels of mercury greater than, or equal to, 0.5 ppm in their blood on at least a yearly basis.

11. Remove all panthers with mercury blood levels of 1.4 or greater from the wild and hold them in captivity until blood levels decrease below 0.5 ppm.

C. SOURCE IDENTIFICATION AND ELIMINATION

1. Strongly support the State of Florida's current efforts to identify and rectify the source of mercury contamination. Write the Federal Congressional Delegations to alert them to the problems with the panther. Attach a copy of this report to the letters. At the appropriate time, write a letter to State legislators endorsing the recommendations of the State Task Force.

CONCLUSIONS

The Technical Subcommittee makes the following conclusions based on available data.

1. Mercury contamination of the Florida panther has been documented.

2. Panther #27 contained levels of mercury in its liver consistent with mercury toxicosis reported from laboratory experiments and field observations of domestic cats.

3. It is likely that panther #27 died from mercury toxicosis.

4. Panthers with elevated mercury levels were found in the Southern Fakahatchee Strand before 1988, the eastern Big Cypress National Preserve, Shark River Slough, and the East Everglades area.

5. Raccoons, alligators, and otters contaminated with mercury are the source of contamination in the Florida panther. The former species are becoming contaminated through the aquatic food chain.

6. Those areas where panthers had the highest levels of mercury were correlated with a low availability of deer and hogs and a high consumption rate of non-ungulates, primarily raccoons.

7. Elevated mercury levels appear to be affecting reproductive success in some female panthers. In the Southern Fakahatchee Strand prior to 1988, the poor reproductive success of females may also have been due to poor nutritional status of the panther.

8. Land management practices and public use regulations that increase deer populations, or, where appropriate, hogs and other non-contaminated prey, could shift panthers away from contaminated prey species.

9. Chronic exposure to mercury, in conjunction with sub-optimum prey availability, may contribute to lower than expected population densities of panthers on all land (four panthers on 1.2 million acres) south of I-75 and could be contributing to the extinction of this endangered mammal.

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would recommend reduction of type size to allow better-looking presentation.

Table 1. Mercury concentrations (ppm, wet wt.) in tissues from free-ranging Florida panthers from various locations, 1978 - 1991.

Line up column headings

	WHOLE BLOOD		HAIR		LIVER		KIDNEY		MUSCLE		BRAIN	
LOCATION	n	Mean(S.E.)[A]	n	Mean(S.E.)	n	Mean(S.E.)	n	Mean(S.E.)	n	Mean(S.E.)	n	Mean(S.E.)
North of Alley	42	0.095(1.15)[B]	43	1.66(1.23)[B]	10	1.15(1.24)	7	0.407(1.24)	6	0.084(1.27)	3	0.052(1.43)
Fakahatchee St.	12	0.384(1.35)[CD]	7	7.18(1.62)[C]	2	20.23(1.08)	4	6.35(1.18)	4	0.974(1.26)	2	1.08(1.32)
Raccoon Point	2	0.620(1.26)	2	42.3 (1.63)	1	15.00		nd[E]		nd		nd
Shark Slough	7	0.794(1.59)[D]	8	56.4 (1.24)[D]	2	40.62(2.02)	2	2.23(2.62)	2	4.44 (1.04)	2	3.18 (1.62)
Long Pine Key	14	0.232(1.21)[C]	8	10.9 (1.35)[C]	1	12.5		nd	1	0.290	1	0.160
Corbett WMA		nd		nd	1	12.1	1	5.20	1	0.240	1	0.240

[A]The mean is the geometric mean determined from log-transformed data and the standard error (S.E.) should therefore be applied as x or ÷ of the mean.

[B,C,D] Within columns, means with different superscripts differ ($P<0.05$). Means without superscript notations were not compared statistically due to small sample size.

[E]nd - not determined.

Same general comments as Table 1.

Table 2. Mercury concentrations (ppm, wet wt.) in tissues from various southern Florida carnivores, 1984 - 1991.

LOCATION	RACCOON MUSCLE		RACCOON LIVER		BOBCAT LIVER		OTTER LIVER		ALLIGATOR MUSCLE	
Northwest to Southeast (approximate)	n	Mean(S.E.)[A]	n	Mean(S.E.)	n	Mean(S.E.)	n	Mean(S.E.)	n	Mean(S.E.)
North of Alley (Bear Island)	7	0.32(1.33)[B]	8	1.51(1.27)[B]	8[E]	0.10(1.79)	3†	0.19(1.77)	1†	0.34
Fakahatchee Strand St. Preserve	6	0.47(1.21)[BC]	6	5.46(1.35)[C]	5	0.13(1.88)		nd[F]		nd
Collier Seminole SP (far west SR 41)		nd		nd		nd	2†	1.01(1.64)		nd
Carnestown (west SR 41)		nd		nd		nd	6†	3.07(1.22)	1†	0.38
Raccoon Point	5	0.48(1.27)[BC]	5	13.4 (1.29)[CD]	7	0.16(1.95)		nd		nd
Oasis (east SR 41)		nd		nd		nd	8†	2.98(1.26)	3†	0.32(1.17)
Loop Road	6	1.43(1.30)[D]	6	15.4 (1.23)[CD]		nd		nd		nd
Water Conservation Area 3A	5	1.41(1.41)[D]	5	15.8 (1.14)[CD]		nd		nd		nd
Shark Slough	5	1.80(1.24)[D]	5	24.0 (1.15)[D]	1†	5.08	1†[G]	6.78	5	2.96(1.20)
Long Pine Key	5	1.22(1.24)[CD]	5	17.5 (1.33)[D]	1	0.08		nd		nd
Taylor Slough	4	1.01(1.68)[CD]	4	4.88(1.87)[C]		nd		nd		nd
Flamingo	6	0.22(1.24)[B]	6	1.35(1.10)[B]		nd		nd		nd
Card Sound Road		nd		nd	1†	0.22		nd		nd

[A]The mean is the geometric mean determined from log-transformed data and the standard error (S.E.) should therefore be applied as x or ÷ of the mean;
[B,C,D]Within columns, means with different superscripts differ ($P<0.05$). Means without superscript notations were not compared statistically due to small sample size;
[E]Two of these samples were collected in the Florida Panther National Wildlife Refuge (0.99 & 0.08 ppm);
[F]nd - not determined;
[G]Collected from Chikika State Park in the eastern Everglades;
†Road-killed animals.

Table 3. Toxicity of alkylmercury to cats.

Dose (mg/kg)	Duration of treatment	Cumulative dosage	Effects
2.0-3.0	2-3 wk	10-20 mg/kg	Clinical signs
0.4-1.0	10-90 d	10-20 mg/kg	Clinical signs
0.25 (fish)	55-96 d	14-25 mg/kg	Marked toxicosis
0.176	14 wk		Toxicosis
0.074	40 wk		Toxicosis
0.046	60 wk		Toxicosis
0.02	2 years		No signs

Charbonneau et al., 1976

Table 4. Potential mercury toxicosis from consumption of raccoons.

Area[1]	Mg Hg/ Meal[2]	Mg/Kg of Panther[3]	Time to Clinical Effect[4]
SS-ENP	6.3	0.185	14 wk
LR-BCNP	4.87	0.143	16 wk
3A/3B	3.63	0.11	20 wk
PI-ENP	2.13	0.063	50 wk
RP-BCNP	1.41	0.04	60 wk
FS	1.44	0.043	60 wk
BI-BCNP	0.65	0.019	> 2 yrs

[1]Area: SS-ENP = Shark River Slough, Everglades National Park; LR-BCNP = Loop Road Unit, Big Cypress National Preserve; 3A/3B = Water Conservation Area 3A & 3B; PI-ENP = Pine Island/Hole-in-the-Donut Area, ENP; RP-BCNP = Raccoon Point Area, BCNP; FS = Fakahatchee Strand State Preserve; BI-BCNP = Bear Island Unit, BCNP.

[2]6 lb. flesh from 8-12 lb raccoon;

[3]Adult female panther, est. 75 lb;

[4]Times based on domestic cat research from Charbonneau 1974

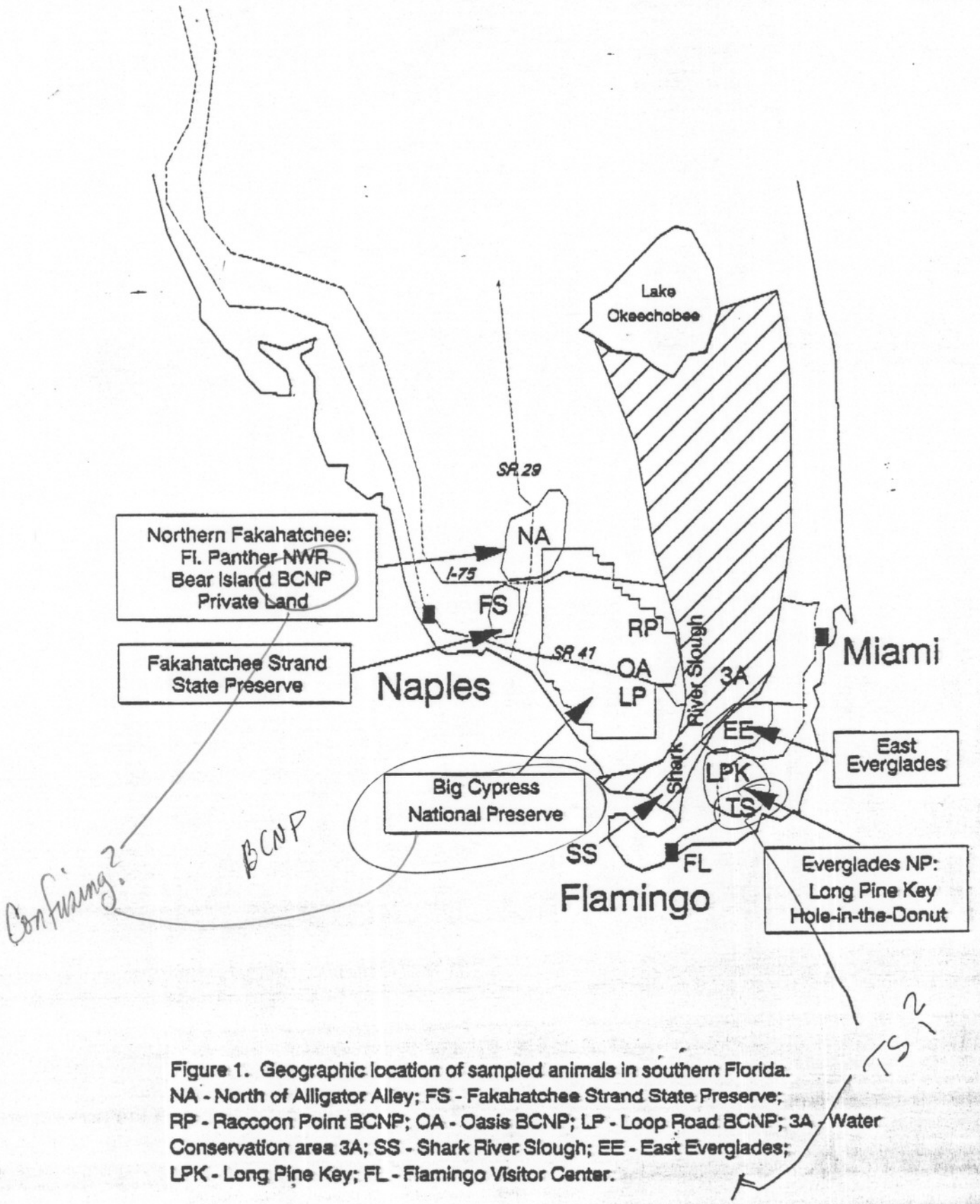

Figure 1. Geographic location of sampled animals in southern Florida. NA - North of Alligator Alley; FS - Fakahatchee Strand State Preserve; RP - Raccoon Point BCNP; OA - Oasis BCNP; LP - Loop Road BCNP; 3A - Water Conservation area 3A; SS - Shark River Slough; EE - East Everglades; LPK - Long Pine Key; FL - Flamingo Visitor Center.

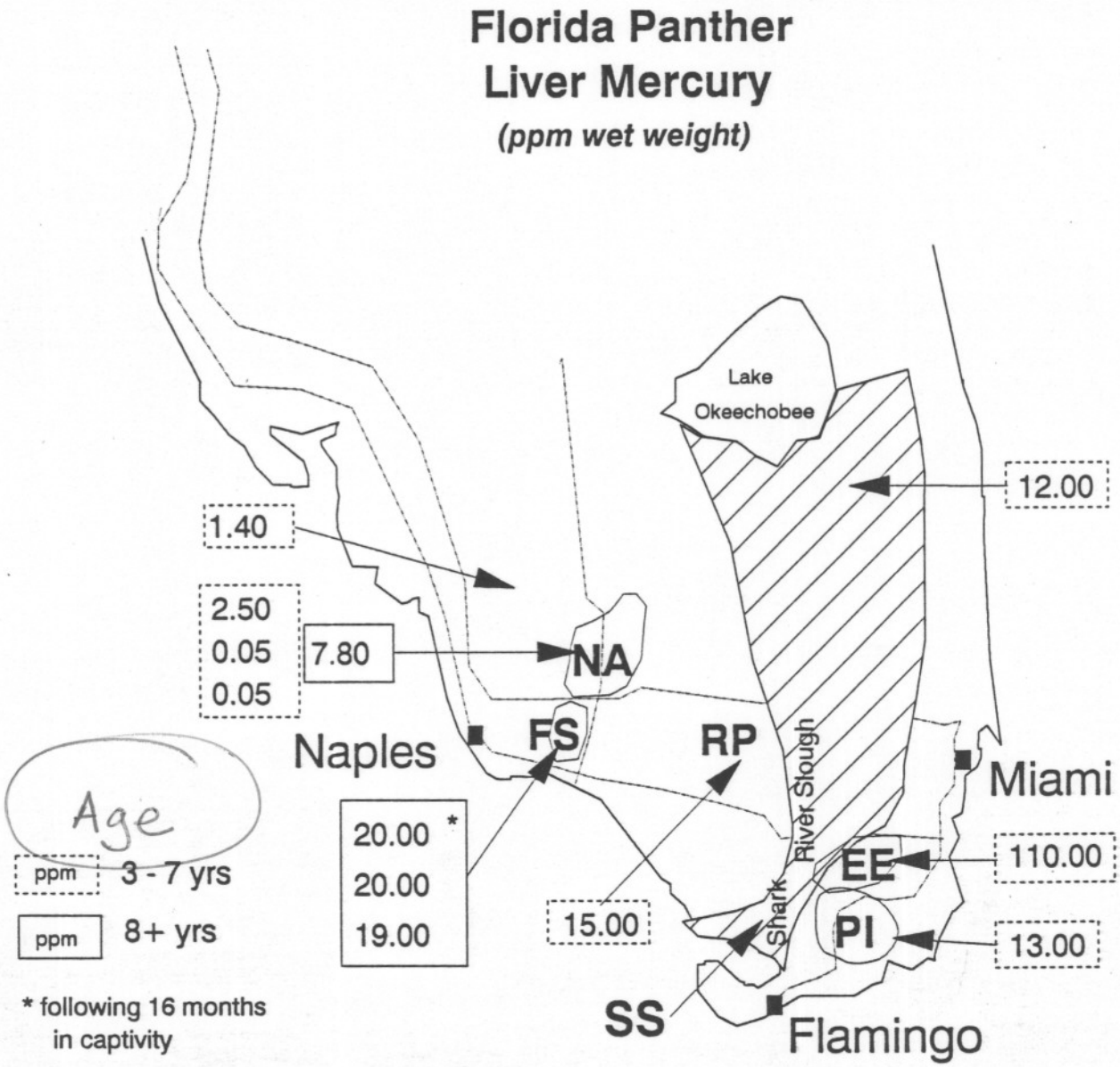

Figure 2. Total mercury values in liver samples from dead panthers showing approximate geographic distribution of contaminants.

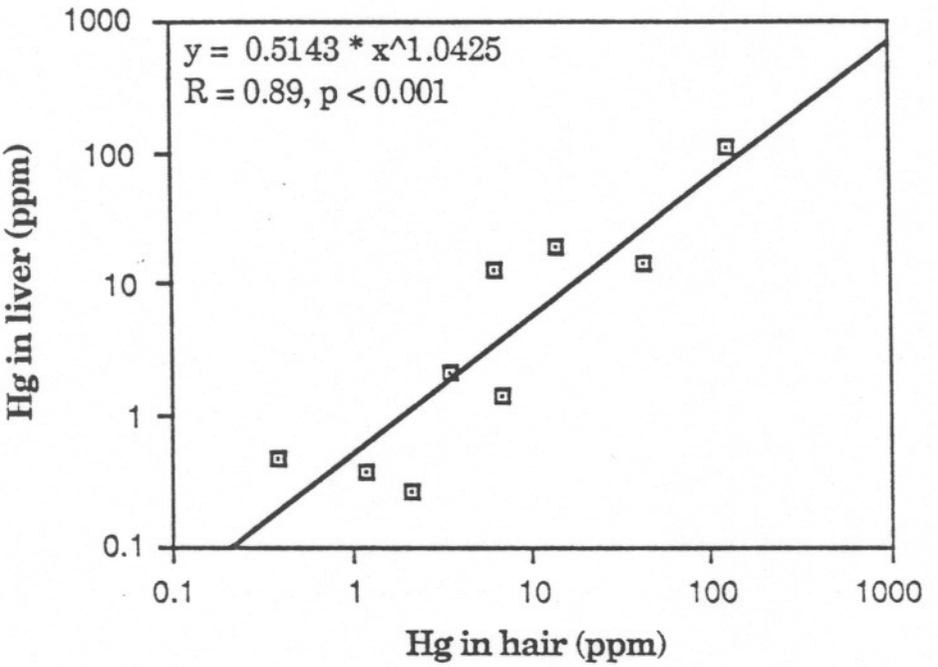

Figure 3. Relationship between mercury (Hg) concentrations in liver and hair of Florida panthers.

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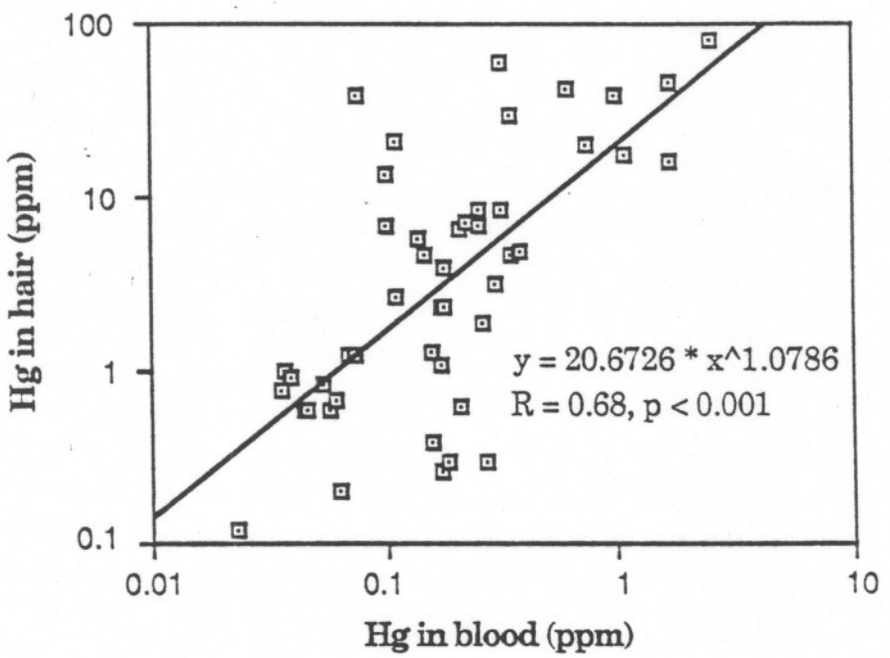


Figure 4. Relationship between mercury (Hg) concentrations in blood and hair from Florida panthers.

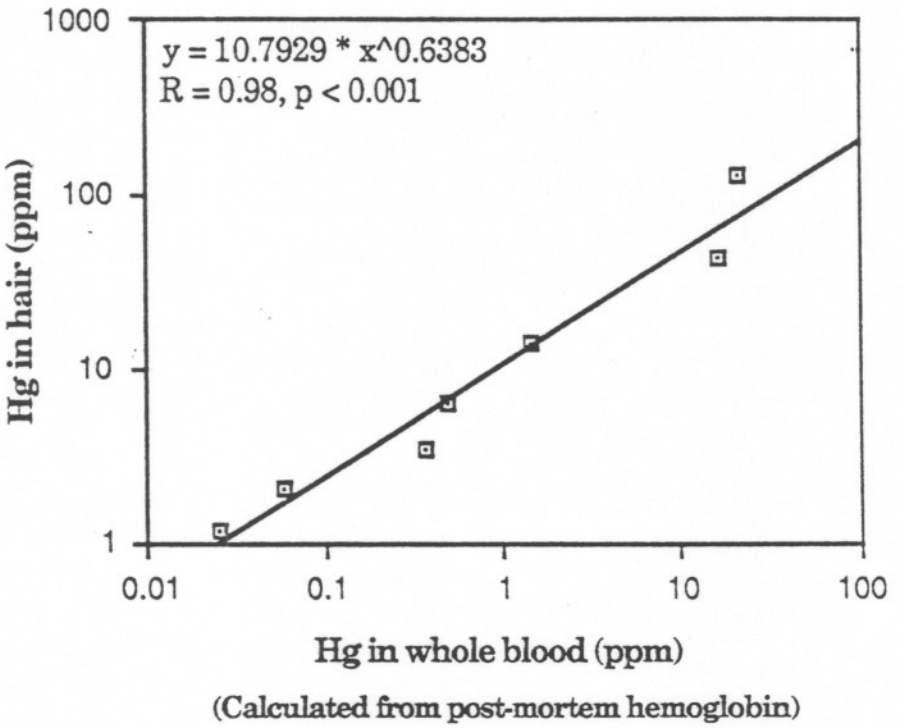


Figure 5. Relationship between mercury (Hg) concentrations in blood (as calculated from post-mortem hemoglobin) and hair from Florida panthers.

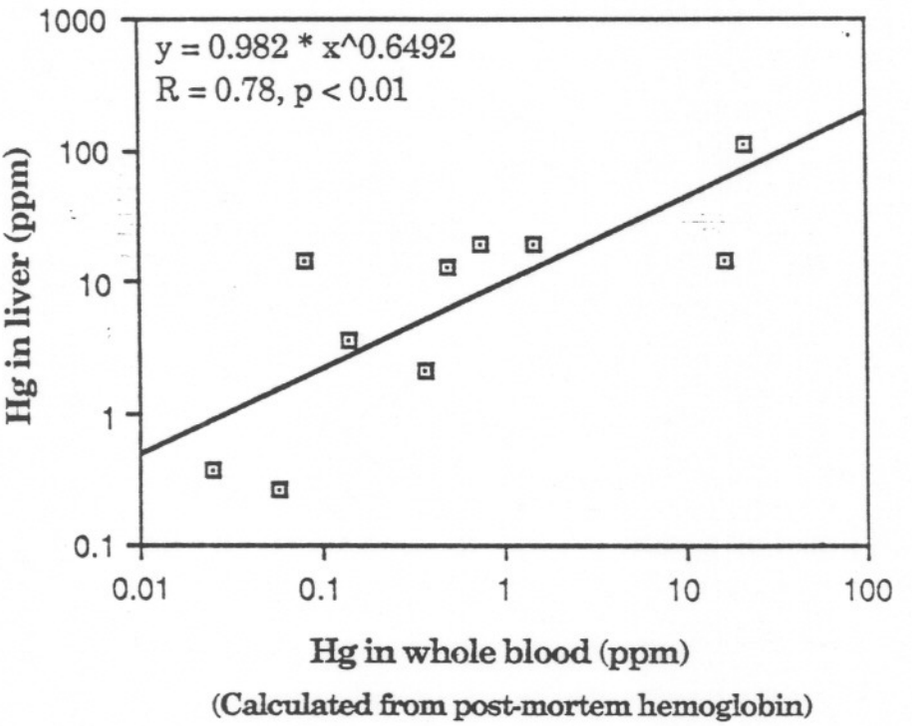


Figure 6. Regression of mercury (Hg) concentration in panther liver on concentration in blood (calculated from post-mortem hemoglobin).

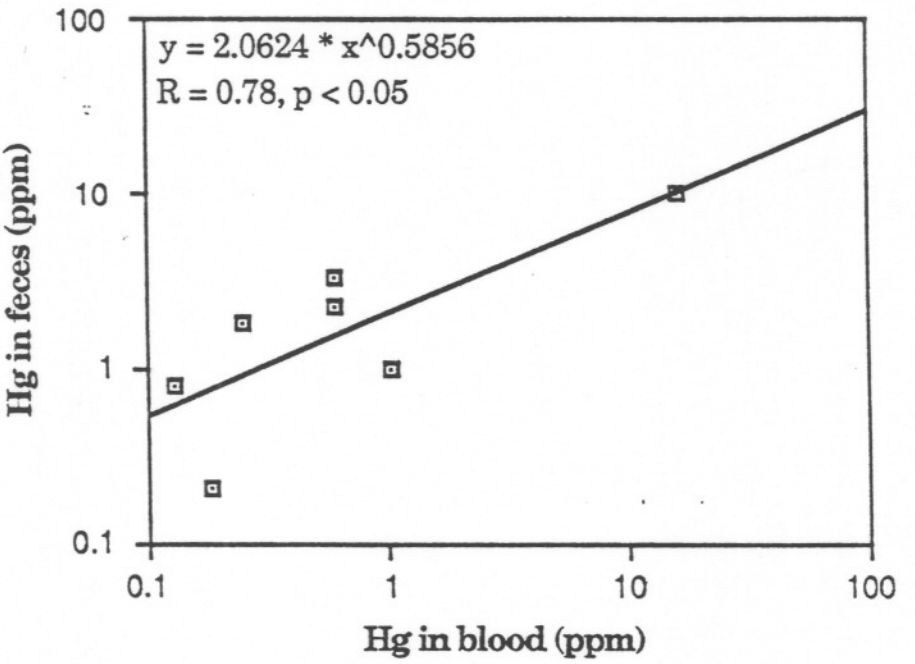


Figure 7. Relationship between mercury (Hg) concentrations in blood and feces of Florida panthers.

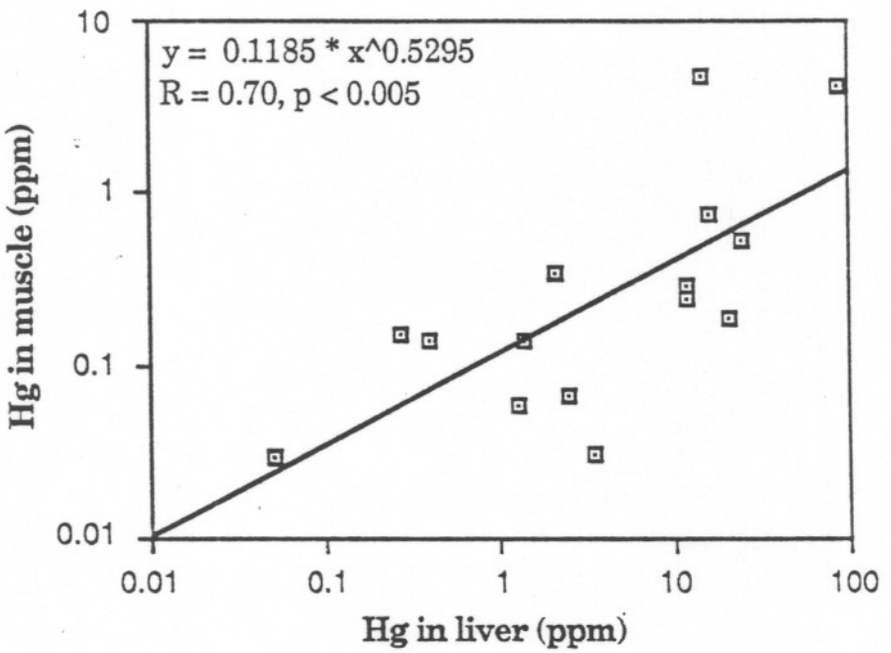


Figure 8. Relationship between mercury (Hg) concentrations in muscle and liver from Florida panthers.

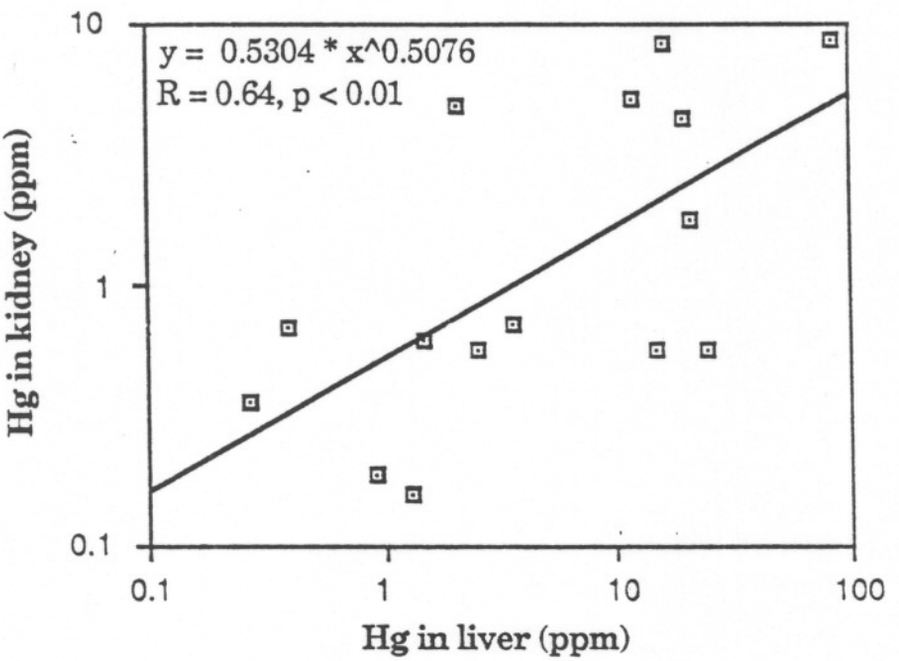


Figure 9. Relationship between mercury (Hg) concentrations in the kidney and liver of Florida panthers.

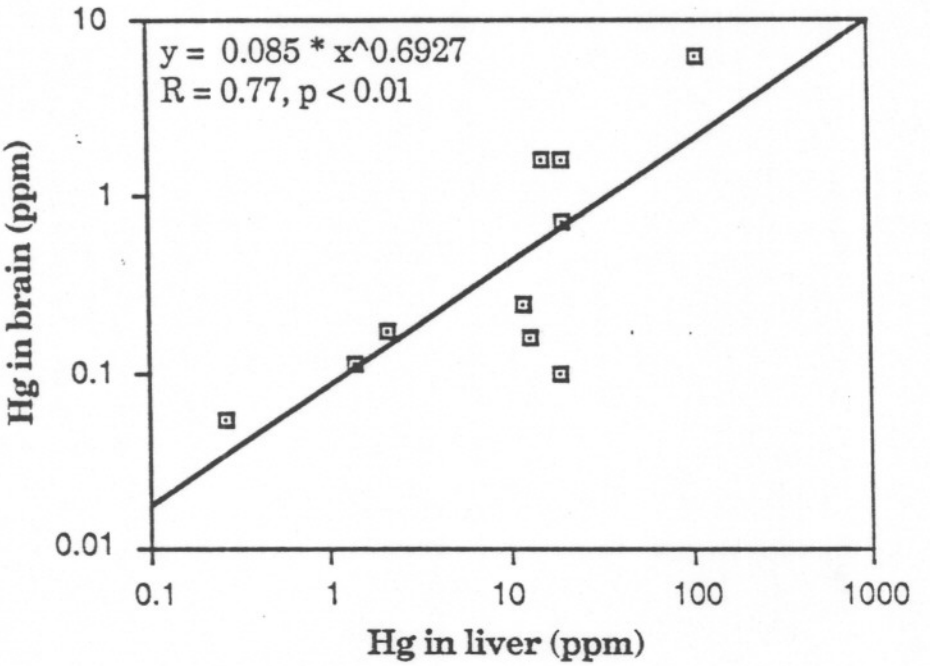


Figure 10. Relationship between mercury (Hg) concentrations in the brain and liver of Florida panthers.

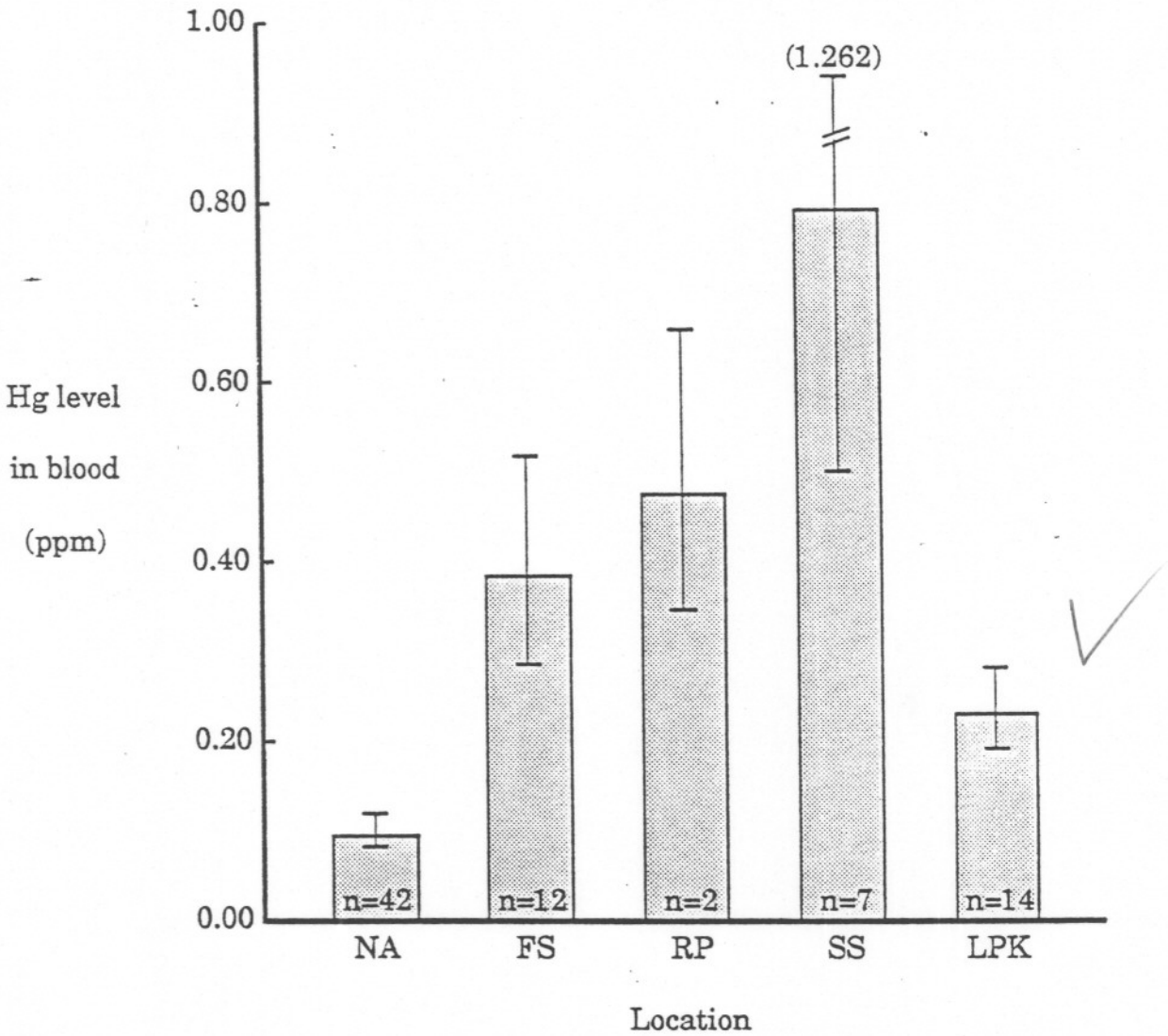


Figure 11. Geometric mean mercury concentrations (± S.E.) in whole blood from panthers from north of Alligator Alley (NA), Fakahatchee Strand State Preserve (FS), Raccoon Point (RP), Shark Slough (SS) and Long Pine Key (LPK). n = number of samples.

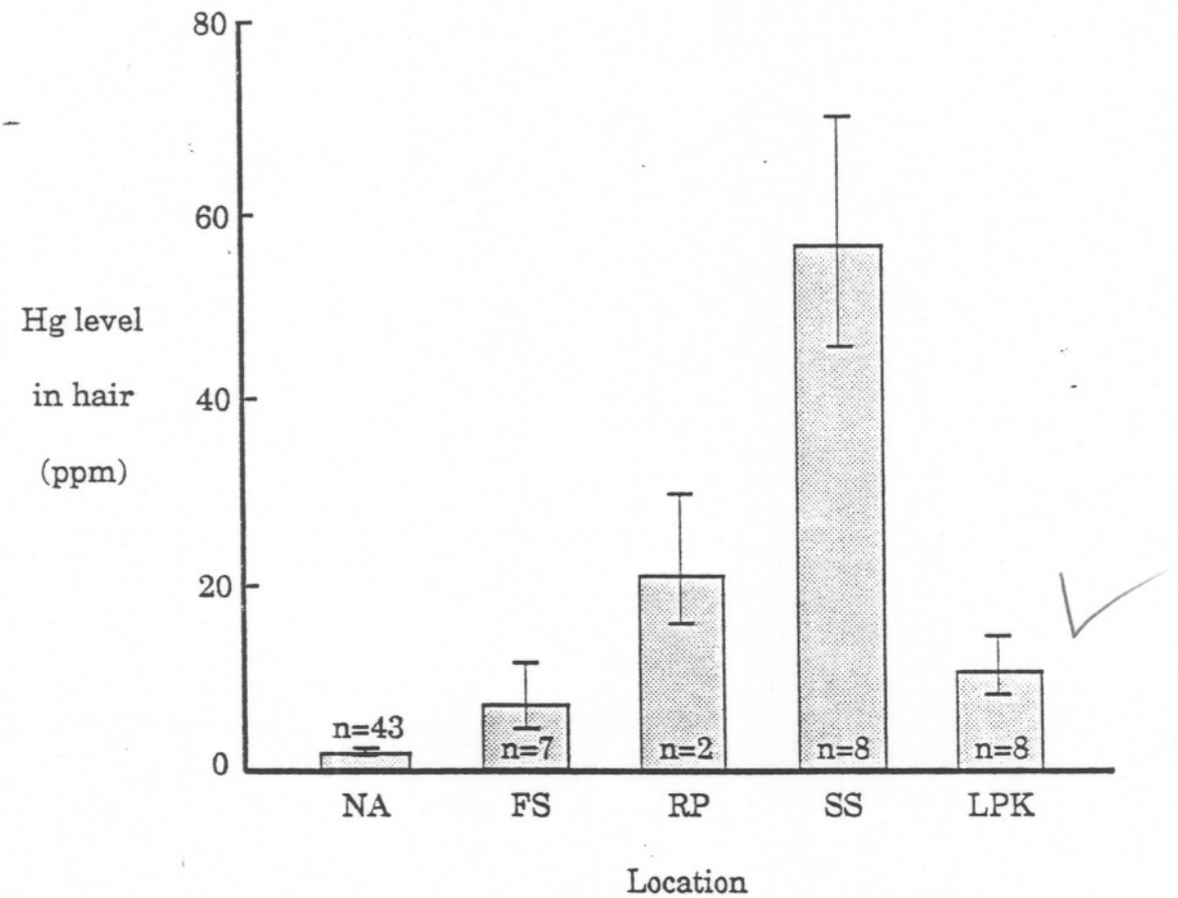


Figure 12. Geometric mean mercury concentrations (± S.E.) in hair from Florida panthers from north of Alligator Alley (NA), Fakahatchee Strand State Preserve (FS), Raccoon Point (RP), Shark Slough (SS) and Long Pine Key (LPK). n = number of samples.

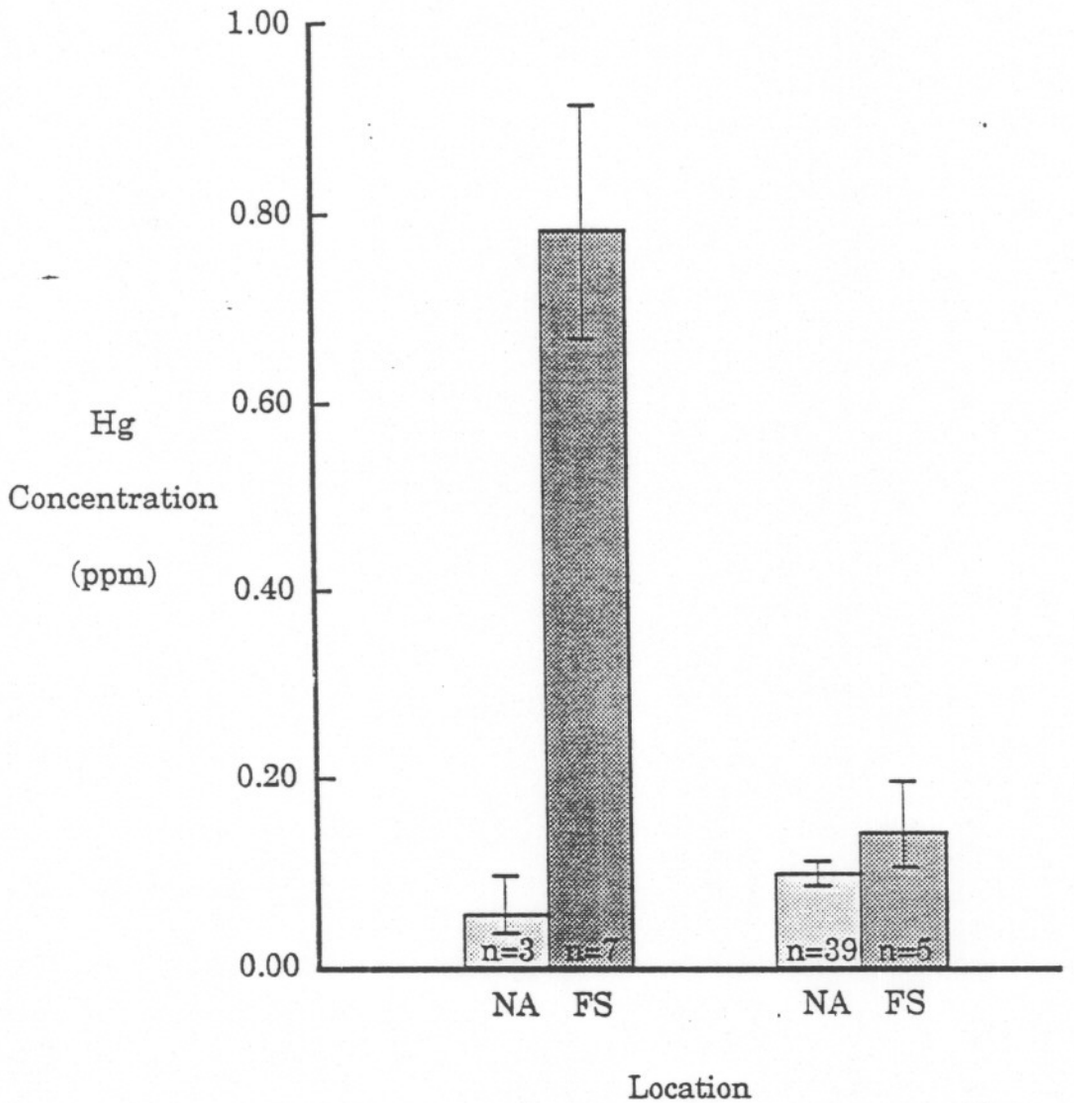


Figure 13. Comparison of geometric mean mercury concentrations (± S.E.) in blood from panthers north of Alligator Alley (NA) and Fakahatchee Strand State Preserve (FS) prior to (left) and after (right) cessation of hunting and changing land management practices in Fakahatchee Strand. n = number of blood samples.

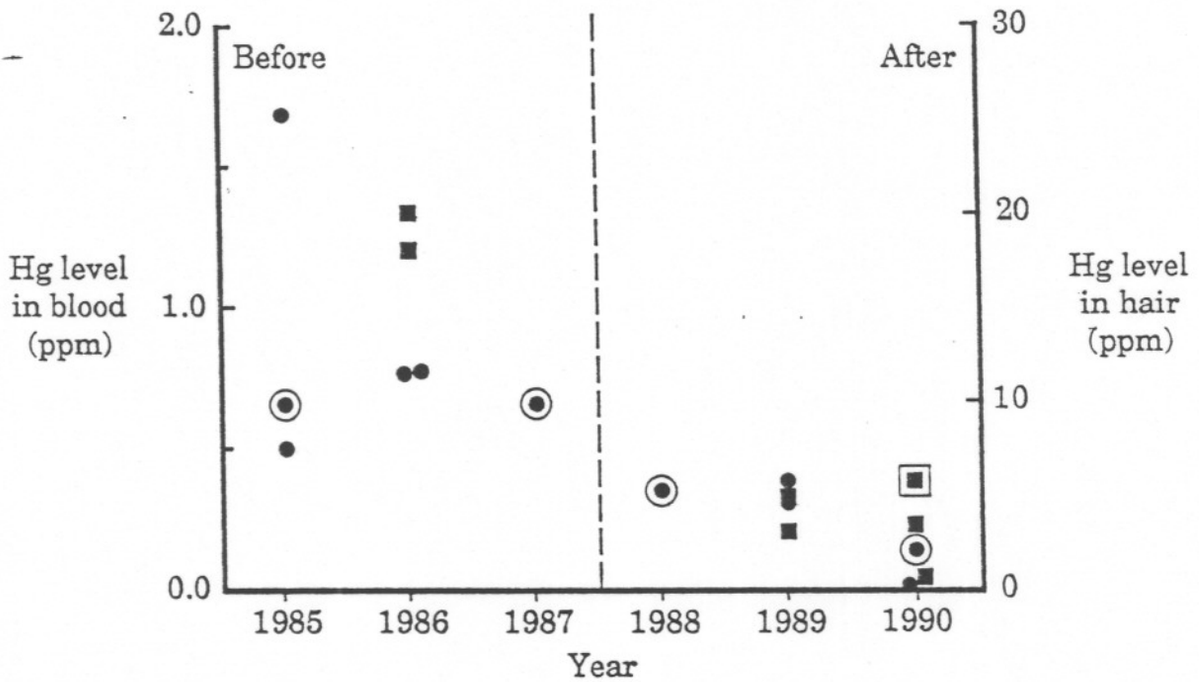


Figure 14. Mercury (Hg) concentrations in blood (●) and hair (■) of panthers living in Fakahatchee Strand State Preserve (FS) before (left) and after (right) commencement of game and land management practices (see text). ◉ and ▣ are samples from panther FP09, the only animal that has lived in the FS throughout the entire period.

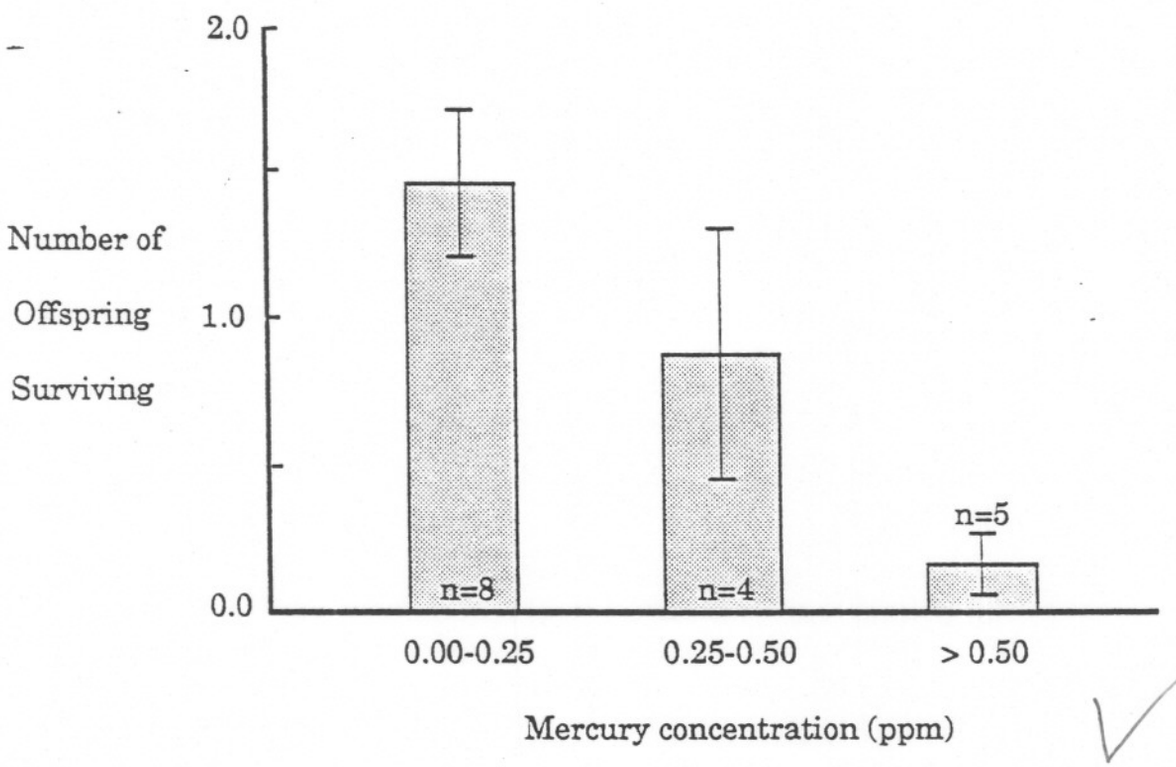


Figure 15. Average number of offspring (± S.E.) surviving to age 6 months per female year $^{-1}$ in relation to mercury concentration in the whole blood of the female parent. n = number of females.

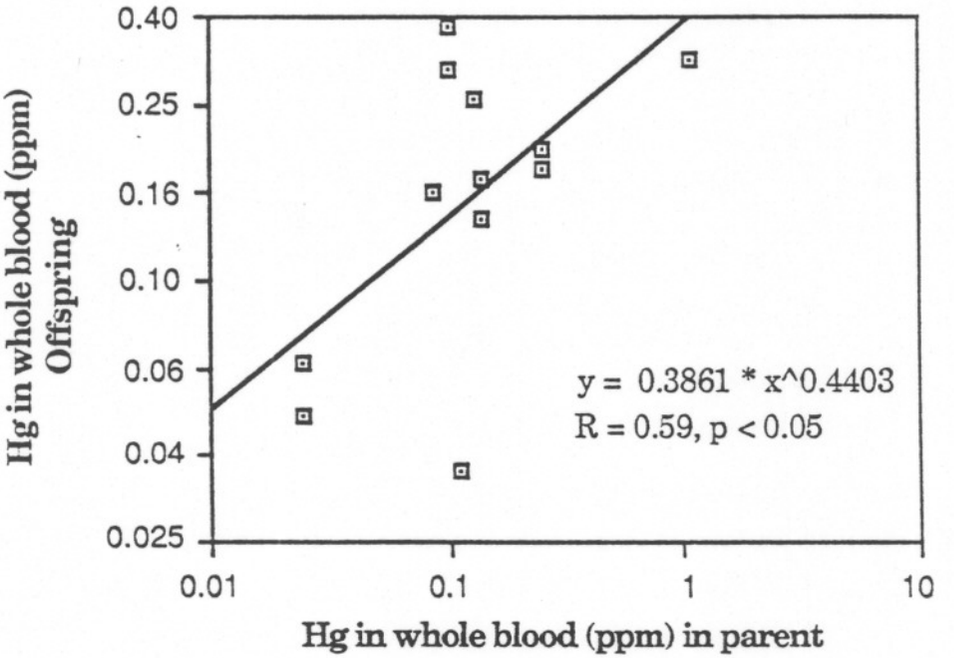


Figure 16. Relationship between mercury concentration in whole blood of the female parent and that of dependent offspring.

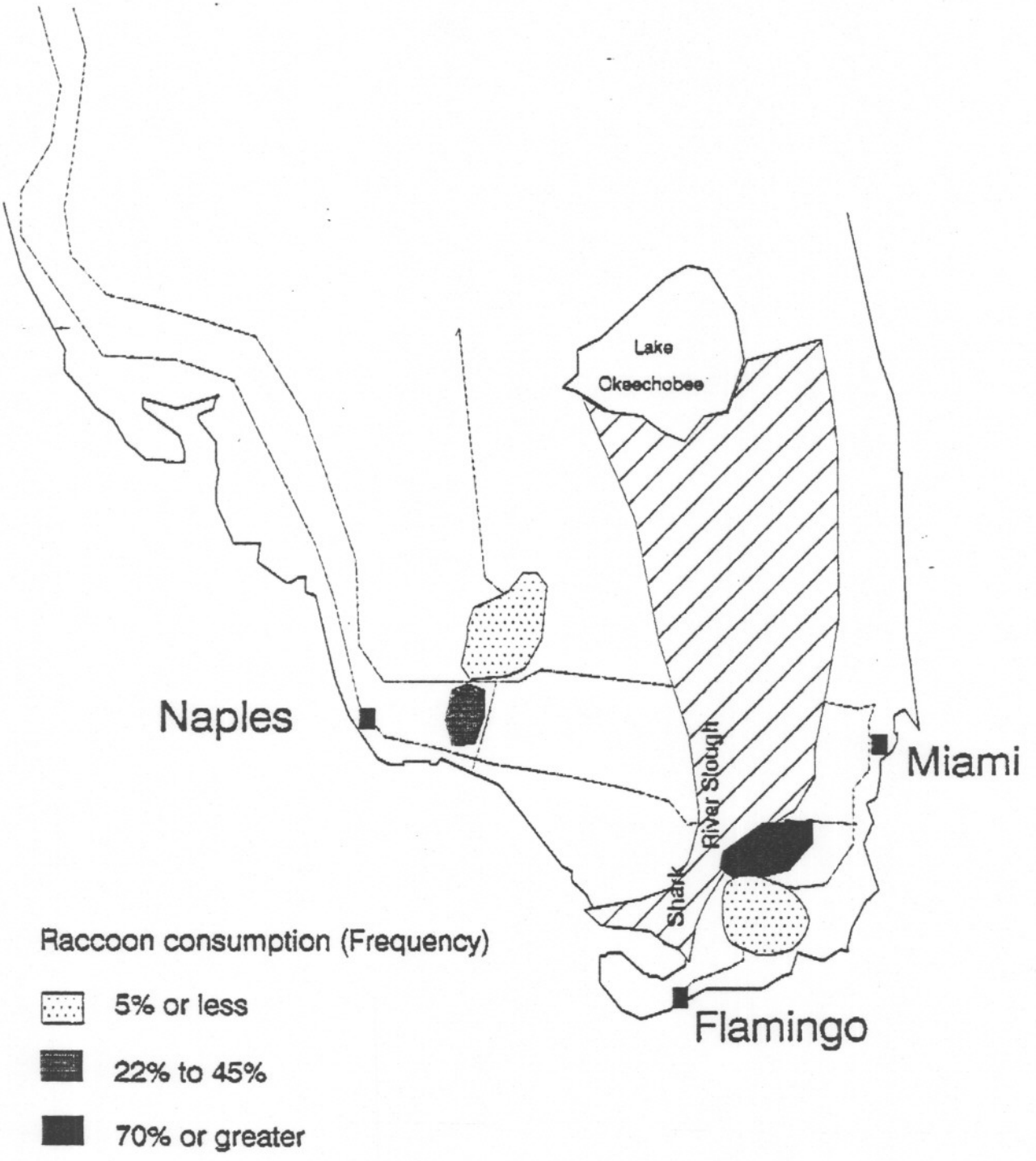


Figure 17. Panther food habits: raccoon consumption by location (Roelke et al., 1986, Maehr et al., 1990, and USFWS, 1989).

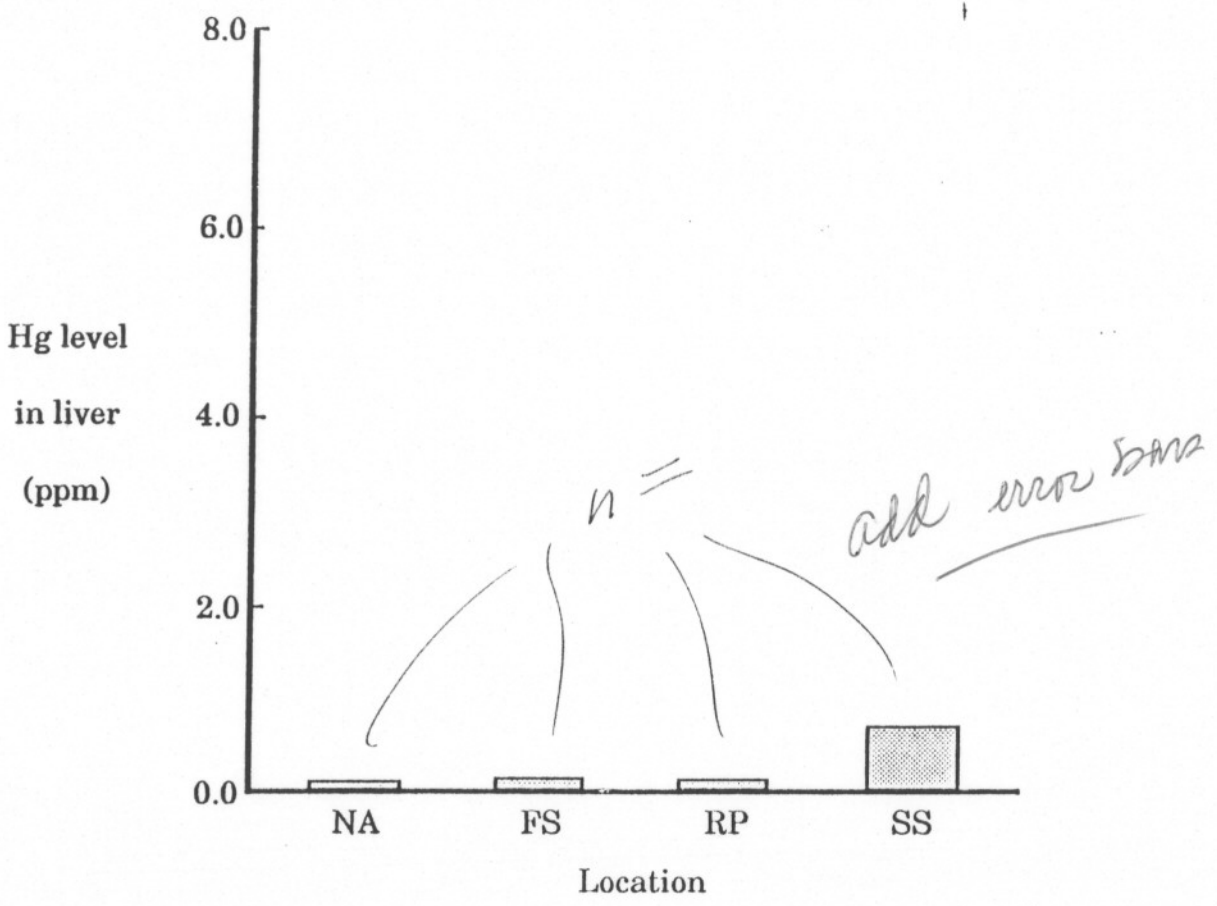


Figure 18. Arithmetic mean mercury concentrations (± S.E.) in livers of deer collected from north of Alligator Alley (NA), Fakahatchee Strand State Preserve (FS), Raccoon Point (RP) andShark Slough (SS). n = number of animals. The scale is the same as for river otters and bobcats for ease of comparison (after Sundloff and Forrester, unpubl. ms.).

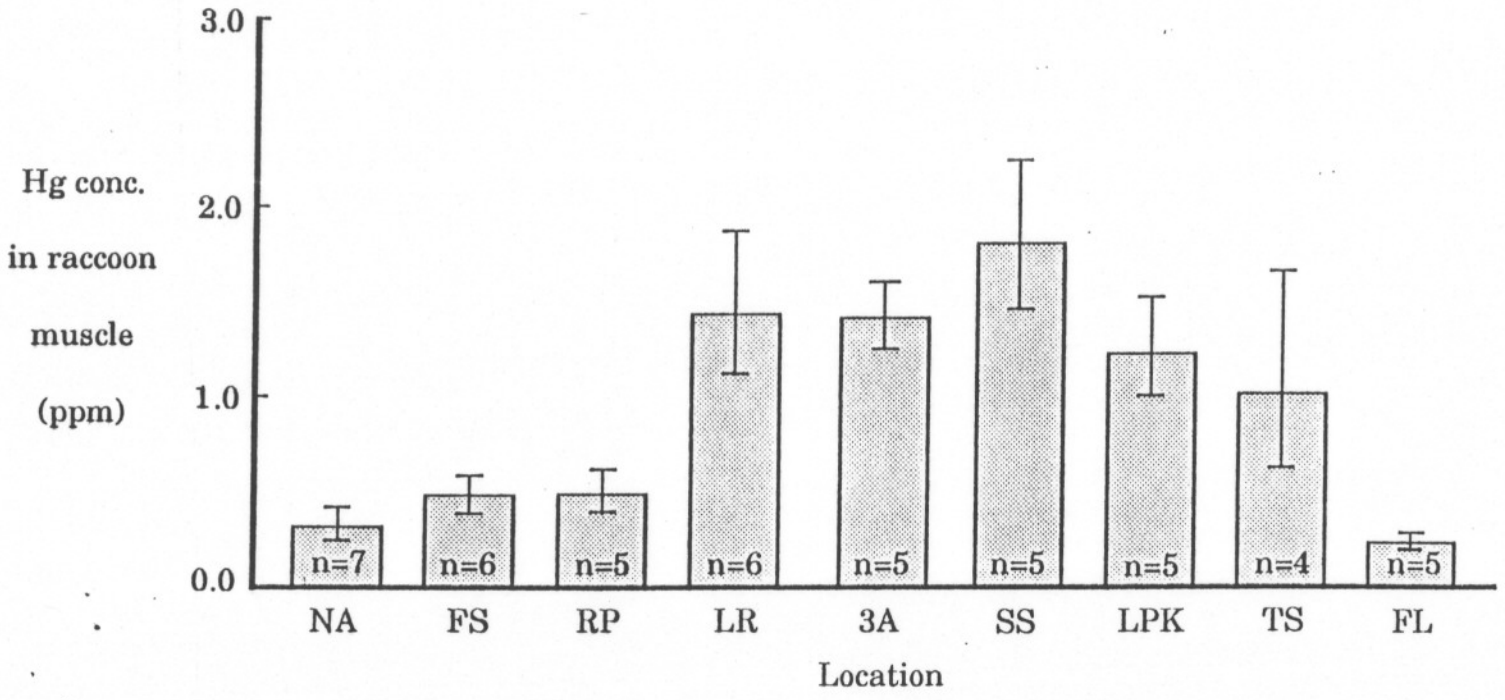


Figure 19. Geometric mean mercury concentrations (± S.E.) in muscle from raccoons collected from north of Alligator Alley (NA), Fakahatchee Strand State Preserve (FS), Raccoon Point (RP), Loop Road (LR), Water Conservation Area 3A (3A), Shark Slough (SS), Long Pine Key (LPK), Taylor Slough (TS) and Flamingo Key (FL). n = number of raccoons.

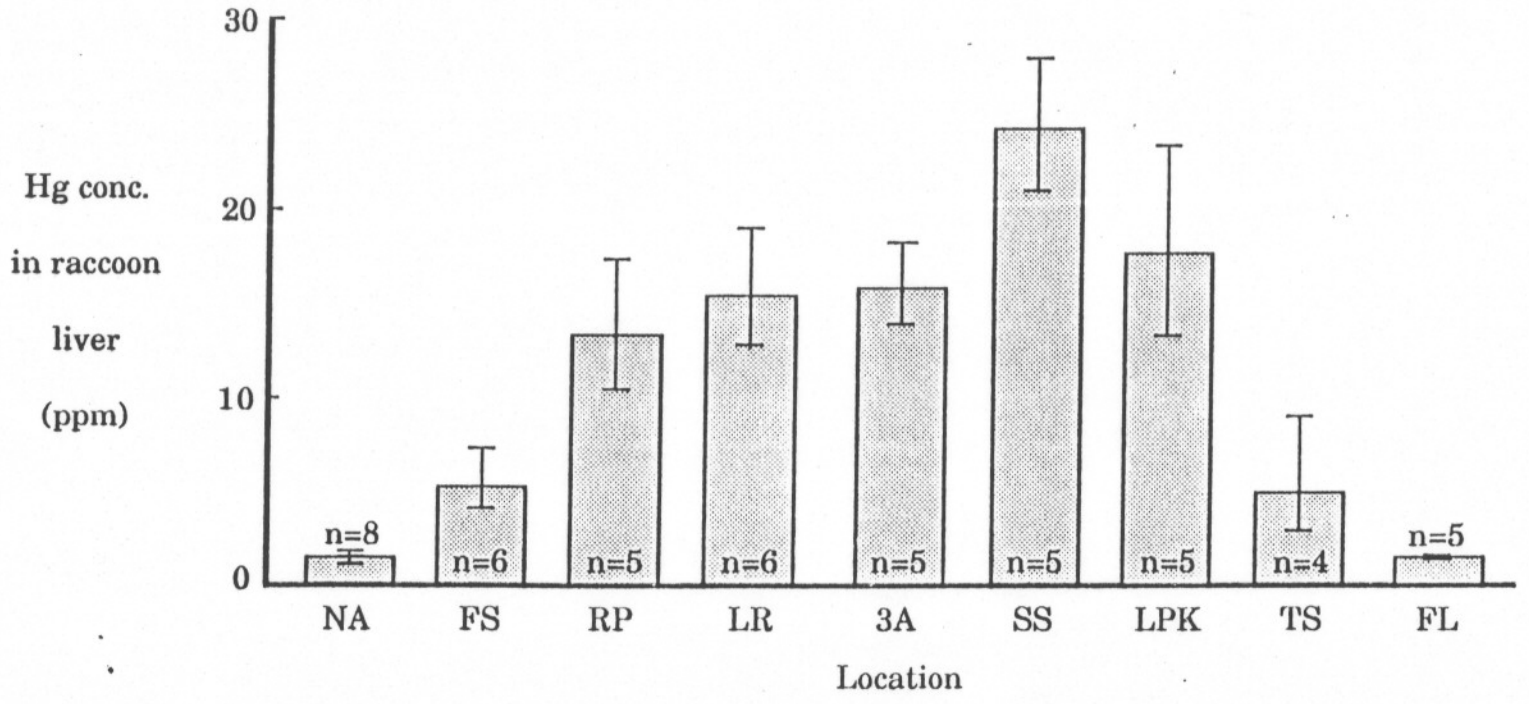


Figure 20. Geometric mean mercury concentrations (± S.E.) in liver from raccoons collected from north of Alligator Alley (NA), Fakahatchee Strand State Preserve (FS), Raccoon Point (RP), Loop Road (LR), Water Conservation Area 3A (3A), Shark Slough (SS), Long Pine Key (LPK), Taylor Slough (TS) and Flamingo Key (FL). n = number of raccoons.

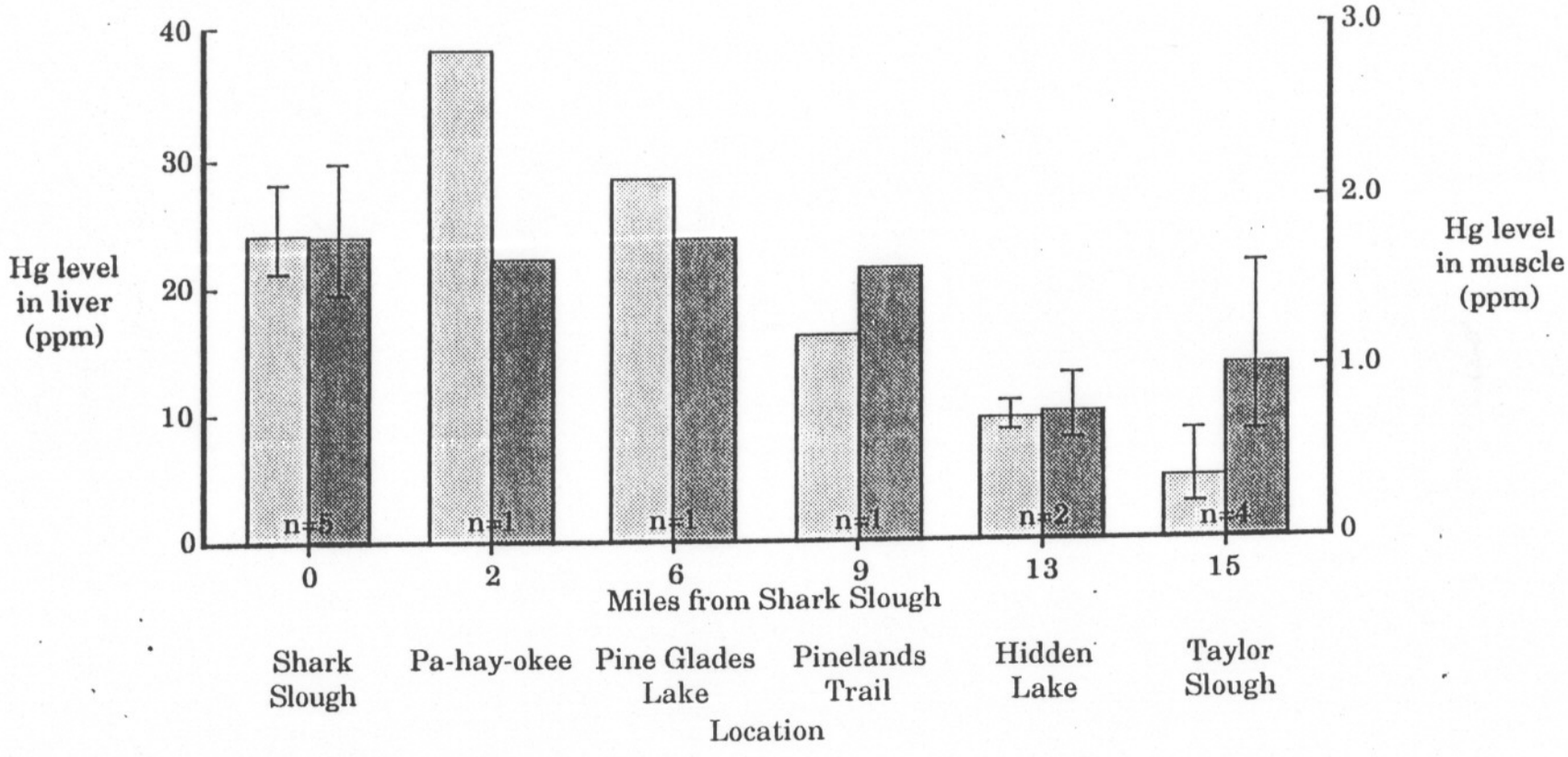


Figure 21. Levels of mercury (Hg) in raccoon muscle and liver with distance from Shark Slough. n = number of animals.

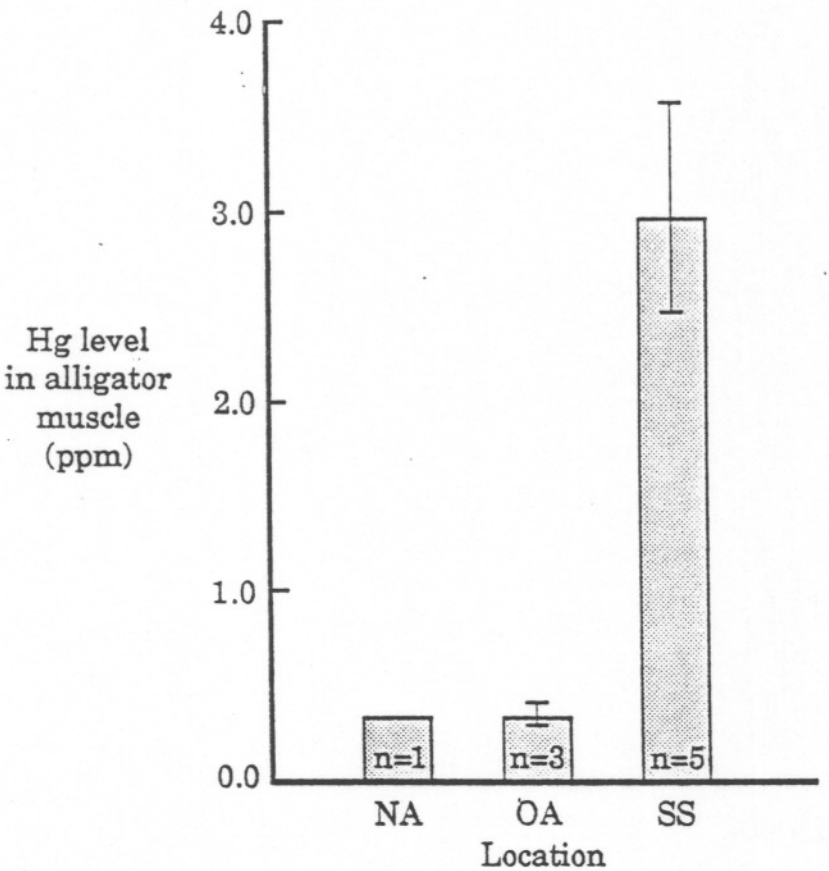


Figure 22. Geometric mean mercury (Hg) concentrations (± S.E.) in alligator muscle tissue from animals collected from north of Alligator Alley (NA) the Oasis (OA) and Shark Slough (SS). n = number of animals.

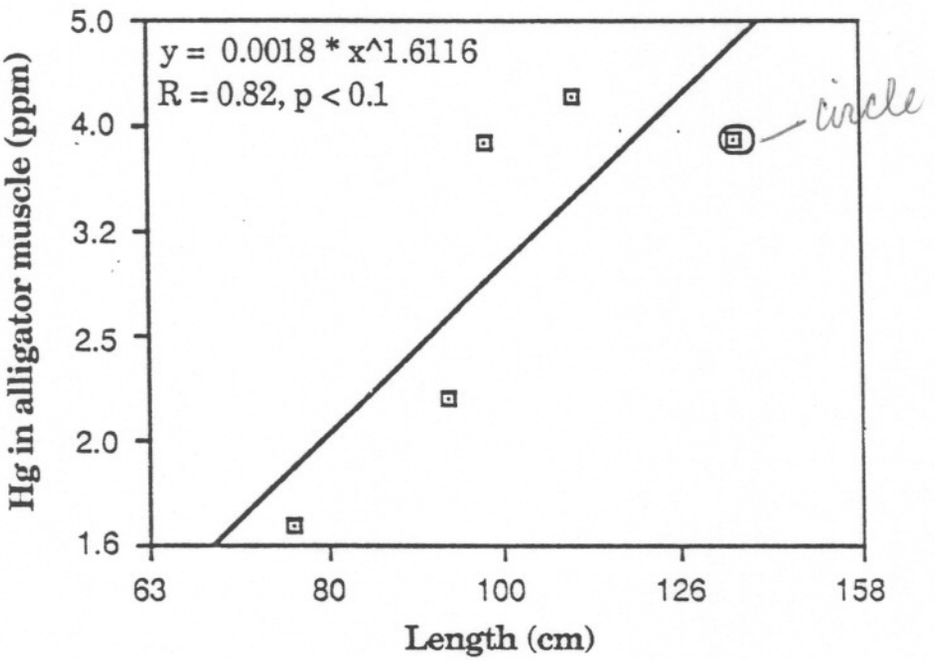


Figure 23. Relationship between snout-vent length and mercury (Hg) concentration in alligator muscle. All animals except the circled one were females.

credit ?

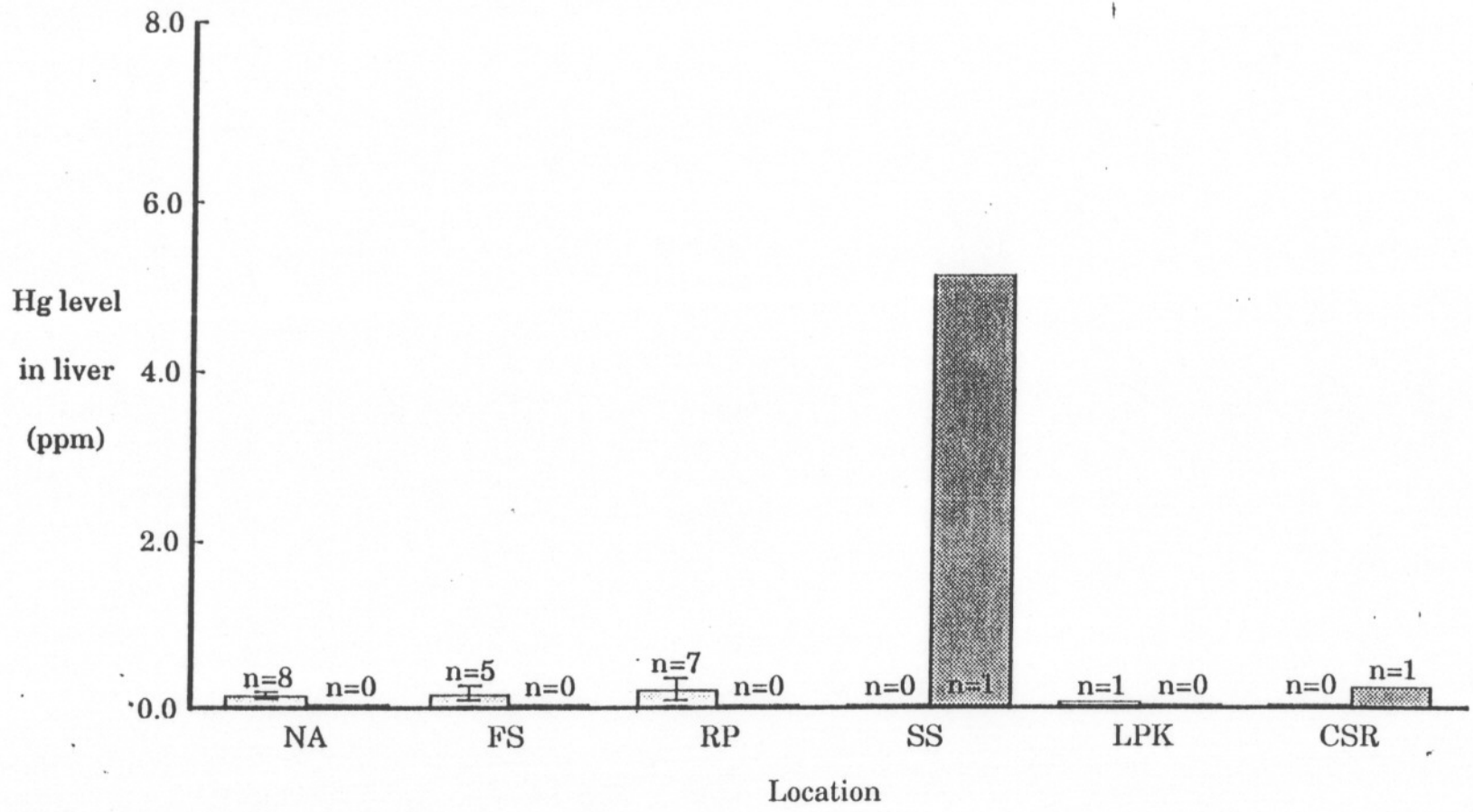


Figure 24. Geometric mean mercury concentrations (± S.E.) in livers of bobcats before (□) and after (▩) the drought of 1988-1990. Animals were collected from north of Alligator Alley (NA), Fakahatchee Strand State Preserve (FS), Raccoon Point (RP), Shark Slough (SS), Long Pine Key (LPK) and Card Sound Road (CSR). n = number of animals. The scale is the same as that for river otters for comparison.

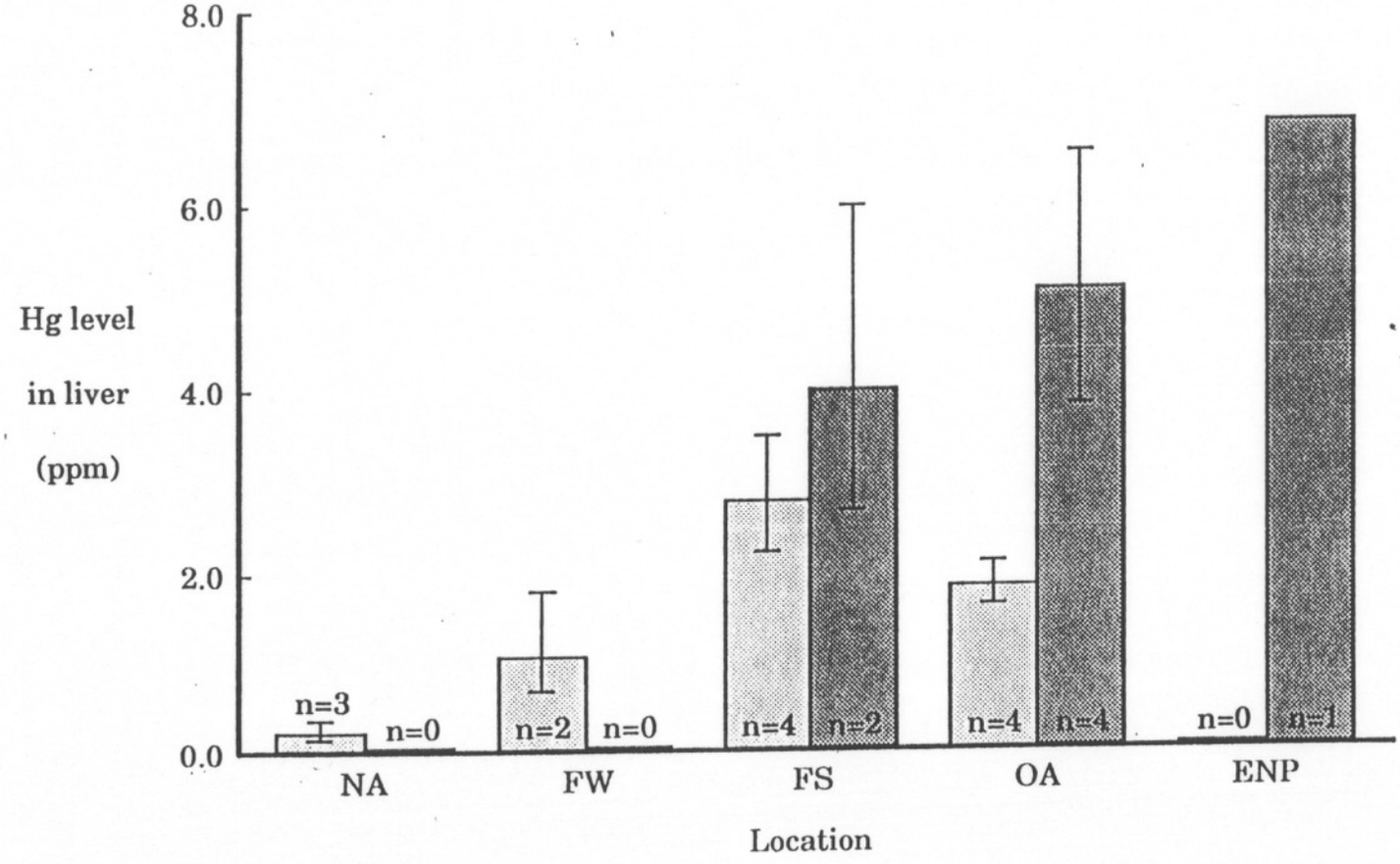


Figure 25. Geometric mean mercury concentrations (± S.E.) in livers of river otters before (□) and after (▩) the drought of 1988-1990. Animals were collected from north of Alligator Alley (NA), the far western edge of known panther range (FW), Fakahatchee Strand State Preserve (FS), the Oasis (OA) and Everglades National Park (ENP). n = number of animals.

1988-91 ?
or change text

APPENDIX

RAW DATA FOR ~~PANTHERS~~, RACCOONS, OTTERS, ALLIGATORS, AND BOBCATS

Mercury Contamination of Raccoons (in connection with the panther study)

ID No.	Location	Tissue	Hg (ppm wwt)
R11M2	NA	Muscle	0.240
R12M2	NA	Muscle	0.325
R13M2	NA	Muscle	0.115
R14M2	NA	Muscle	0.153
R15M2	NA	Muscle	0.374
N91-01P	NA	Muscle	0.620
N91-02P	NA	Muscle	1.020
R21M2	RP	Muscle	1.199
R22M2	RP	Muscle	0.315
R24M2	RP	Muscle	0.420
R26M2	RP	Muscle	0.355
R27M2	RP	Muscle	0.454
R31M2	FS	Muscle	0.352
R32M2	FS	Muscle	0.459
R33M2	FS	Muscle	0.904
R34M2	FS	Muscle	0.778
R35M2	FS	Muscle	0.352
R37M2	FS	Muscle	0.279
R41M2	LR	Muscle	1.035
R42M2	LR	Muscle	0.930
R43M2	LR	Muscle	1.909
R44M2	LR	Muscle	4.647
R46M2	LR	Muscle	1.070
R47M2	LR	Muscle	0.954
R52M2	3A	Muscle	1.516
R53M2	3A	Muscle	1.516
R54M2	3A	Muscle	1.230
R55M2	3A	Muscle	0.959
R56M2	3A	Muscle	2.028
R61M2	SS	Muscle	1.433
R62M2	SS	Muscle	3.341
R64M2	SS	Muscle	2.495
R65M2	SS	Muscle	1.630
R66M2	SS	Muscle	0.960

Raccoon Muscle (cont.)

R72M2	FL	Muscle	0.200
R73M2	FL	Muscle	0.320
R75M2	FL	Muscle	0.250
R76M2	FL	Muscle	0.330
R77M2	FL	Muscle	0.100
R81M2	PI	Muscle	0.970
R82M2	PI	Muscle	0.585
R83M	PI	Muscle	1.620
R84M	PI	Muscle	1.790
R86M	PI	Muscle	1.660
R91M2	TS	Muscle	2.140
R93M2	TS	Muscle	0.240
R94M2	TS	Muscle	1.550
R95M2	TS	Muscle	1.290

Mercury Contamination of Raccoons (in connection with the panther study)

ID No.	Location	Tissue	Hg (ppm ww)
R11L2	NA	Liver	1.074
R12L2	NA	Liver	1.092
R13L2	NA	Liver	0.753
R14L2	NA	Liver	0.678
R15L2	NA	Liver	2.284
R16L2	NA	Liver	5.185
N91-01P	NA	Liver	2.130
N91-02P	NA	Liver	1.800
R21L2	RP	Liver	14.306
R22L2	RP	Liver	7.839
R24L2	RP	Liver	7.736
R26L2	RP	Liver	29.619
R27L2	RP	Liver	16.917
R31L2	FS	Liver	6.525
R32L2	FS	Liver	13.066
R33L2	FS	Liver	4.564
R34L2	FS	Liver	10.610
R35L2	FS	Liver	1.788
R37L2	FS	Liver	3.590
R41L2	LR	Liver	33.919
R42L2	LR	Liver	11.467
R43L2	LR	Liver	14.679
R44L2	LR	Liver	18.657
R46L2	LR	Liver	7.494
R47L2	LR	Liver	16.677
R52L2	3A	Liver	14.862
R53L2	3A	Liver	20.977
R54L2	3A	Liver	13.130
R55L2	3A	Liver	21.914
R56L2	3A	Liver	10.913
R61L2	SS	Liver	17.519
R62L2	SS	Liver	24.555
R64L2	SS	Liver	17.385
R65L2	SS	Liver	36.065
R66L2	SS	Liver	29.658

R72L2	FL	Liver	1.110
R73L2	FL	Liver	1.780
R75L2	FL	Liver	1.290
R76L2	FL	Liver	1.120
R77L2	FL	Liver	1.560
R81L2	PI	Liver	10.869
R82L2	PI	Liver	8.628
R83L	PI	Liver	16.070
R84L	PI	Liver	28.520
R86L	PI	Liver	38.420
R91L2	TS	Liver	14.474
R93L3	TS	Liver	0.900
R94L2	TS	Liver	7.740
R95L2	TS	Liver	5.630

Mercury Contamination of Otters (in connection with the panther study)

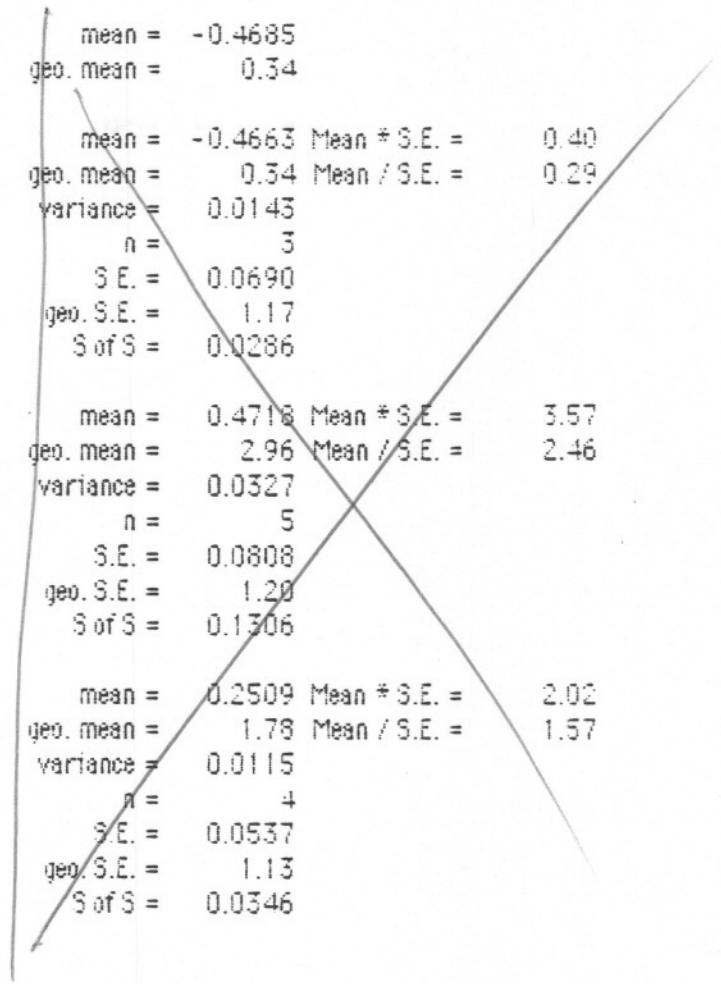
ID No.	Location	Tissue	Hg (ppm wwt)
Pre-drought			
5	NA	Liver	0.060
6	NA	Liver	0.340
7	NA	Liver	0.320
8	FW	Liver	0.620
13	FW	Liver	1.660
9	FS	Liver	4.390
10	FS	Liver	1.510
11	FS	Liver	2.380
12	FS	Liver	3.440
1	OA	Liver	2.060
2	OA	Liver	2.300
3	OA	Liver	1.600
4	OA	Liver	1.330

Mercury Contamination of Otters (in connection with the panther study)

ID No.	Location	Tissue	Hg (ppm wwt)
Post-drought			
N91-03L	FS	Liver	2.600
N91-04L	FS	Liver	5.900
N91-01L	OA	Liver	8.440
N91-02L	OA	Liver	5.480
N91-09L	OA	Liver	2.470
N91-08L	OA	Liver	5.390
N90-07L	SS	Liver	6.780

Mercury Contamination of alligators (in connection with the panther study)

ID No.	Location	Tissue	Hg (ppm wwt)				
1	NA	Muscle	0.340	mean =	-0.4685		
				geo. mean =	0.34		
2	OA	Muscle	0.250	mean =	-0.4663	Mean * S.E. =	0.40
3	OA	Muscle	0.380	geo. mean =	0.34	Mean / S.E. =	0.29
4	OA	Muscle	0.420	variance =	0.0143		
				n =	3		
				S.E. =	0.0690		
				geo. S.E. =	1.17		
				S of S =	0.0286		
5	SS	Muscle	1.660	mean =	0.4718	Mean * S.E. =	3.57
6	SS	Muscle	2.190	geo. mean =	2.96	Mean / S.E. =	2.46
7	SS	Muscle	3.830	variance =	0.0327		
8	SS	Muscle	3.870	n =	5		
9	SS	Muscle	4.240	S.E. =	0.0808		
				geo. S.E. =	1.20		
				S of S =	0.1306		
1	OA	Liver	2.060	mean =	0.2509	Mean * S.E. =	2.02
2	OA	Liver	2.300	geo. mean =	1.78	Mean / S.E. =	1.57
3	OA	Liver	1.600	variance =	0.0115		
4	OA	Liver	1.330	n =	4		
				S.E. =	0.0537		
				geo. S.E. =	1.13		
				S of S =	0.0346		



Bobcat Data

Pre-drought (1985-87)

ID No.	Location	Tissue	Hg (ppm)
B01	RP	Liver	0.49
B02	RP	Liver	0.62
B03	RP	Liver	0.18
B04	RP	Liver	0.27
B05	RP	Liver	0.01
B06	RP	Liver	0.02
B07	RP	Liver	1.03
B11	NA	Liver	0.02
B12	NA	Liver	0.73
B13	NA	Liver	0.01
B14	NA	Liver	0.03
B15	NA	Liver	0.14
B16	NA	Liver	0.16
B26	NA	Liver	0.99
B27	NA	Liver	0.08
B21	FS	Liver	0.36
B22	FS	Liver	0.87
B23	FS	Liver	0.03
B24	FS	Liver	0.08
B25	FS	Liver	0.05
B26			
B27			
B31	ENP	Liver	0.04

Bobcat Data
Post-drought (1988-91)

ID No.	Location	Tissue	Hg (ppm)
SVBCL	SS	Liver	5.08
N91-01B	CS	Liver	0.22