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EFFECTS OF METHYLMERCURY AND SPATIAL COMPLEXITY ON FORAGING BEHAVIOR AND FORAGING EFFICIENCY IN JUVENILE WHITE IBISES (*EUDOCIMUS ALBUS*)

EVAN M. ADAMS* and PETER C. FREDERICK
Department of Wildlife Ecology and Conservation, P.O. Box 110430, University of Florida, Gainesville, Florida 32611, USA

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Abstract—Methylmercury is a globally distributed neurotoxin, endocrine disruptor, and teratogen, the effects of which on wildlife at environmentally relevant levels are largely unknown. In birds, foraging efficiency and learning may be sensitive endpoints for sublethal methylmercury toxicity, and these endpoints also may be biologically relevant at the population level. In the present study, groups of wild-caught, prefledgling white ibises (*Eudocimus albus*) were raised in a free-flight, open-air aviary on diets that approximated the measured range of methylmercury exposure in the Everglades ecosystem (0, 0.05, 0.1, and 0.3 mg/kg/d). The effect of methylmercury exposure on group foraging efficiency was examined by allowing birds to forage on 200 fathead minnows (*Pimephales promelas*) in artificial ponds for 15 min by straining the arenas' contents through a seine net and counting all remaining prey. Additionally, we varied the difficulty of foraging by these tactile feeding birds by adding multiple levels of structural complexity (e.g., increased vegetation and prey refugia) to the pond. Structural complexity affected both foraging efficiency and the rate of increase in efficiency over time (improvement). Methylmercury exposure affected foraging efficiency ($p = 0.03$). It did not affect foraging improvement in the face of increasingly challenging environments, however, and the dose–response relationship was nonlinear (e.g., the control and high-exposure groups were the least efficient foragers). Evidence for an effect of methylmercury on foraging efficiency therefore was inconclusive because of unpredicted results and no interaction with time or habitat complexity. These data suggest a nonlinear dose–response relationship at low levels of methylmercury exposure; future research is needed to verify this hypothesis. This appears to be the first experimental demonstration of the effects of habitat complexity on foraging efficiency in long-legged wading birds.

Keywords—Methylmercury White ibis Foraging Behavior

INTRODUCTION

Methylmercury is a neurotoxin, an endocrine-disrupting contaminant, and a teratogen. Exposure has been connected to changes in behavior and health in humans and wildlife [1]. The most common acute neurological effects are a loss of motor skills, coordination, and reduction in motivation [2,3]. In captive mallards (*Anas platyrynchos*) [4], 3 ppm (wet wt in diet) of methylmercury caused changes in the duckling flight response, brain lesions, and the demyelination of neurons [5], and 5 ppm (wet wt in diet) of methylmercury caused decreases in weight and appetite of great egrets (*Ardea alba*) [6] along with changes in hematology, neurology, and histology [7]. Nocera and Taylor [8] found methylmercury exposure to be correlated with behavioral changes in free-ranging young common loons (*Gavia immer*). Bouton et al. [9] found that at a much lower dose (0.5 mg/kg wet wt in diet), juvenile great egrets showed decreased activity, altered thermoregulatory behavior, and decreased motivation to hunt. Although there appeared to be no effect of mercury dose on foraging efficacy in the egrets, any potential effects seemed to be confounded by differences in individual foraging strategies.

Evidence indicates both direct and indirect links between methylmercury exposure and learning. Methylmercury has been suggested to alter thyroid hormones in vertebrates [10] and is correlated to changes in corticosterone in chickens (*Gallus domesticus*) [11] and in testosterone, estradiol, and perhaps, progesterone in the white ibis (*Eudocimus albus*) [12]. Steroid hormones like estradiol and testosterone [13,14], thyroid hormone [15,16], and glucocorticoids [16,17] have important roles in brain development and learning. Foraging behavior also is at risk of changing in response to endocrine-disrupting contaminant exposure. Disruption of the hypothalamic–pituitary–adrenal axis [18] and steroid hormones [19] has the potential to decrease foraging effort and efficacy and to impact population demographics [20]. Therefore, it seems reasonable to hypothesize that low levels of methylmercury exposure have an impact on learning and foraging behavior in vertebrates; however, the magnitude of mercury exposure necessary to induce this kind of effect is unknown.

We report here on an experimental manipulation designed to test the effects of low, chronic doses of methylmercury on the ability of juvenile white ibises (referred to hereafter as ibises) to forage in differing levels of habitat complexity. Ibises are tactile-foraging aquatic birds that feed in flocks on crabs, crayfish, insects, and small fish in a variety of aquatic habitats [21]. Foraging efficiency is directly linked with conditions that produce high prey availability [22,23], which is related to reproductive success [22,24,25].

Learning novel foraging behaviors may be more difficult for birds exposed to environmentally realistic levels of methylmercury. The prediction that mercury exposure would decrease the ability of ibises to forage as conditions that influence foraging become more challenging was tested in the present study. Two major underlying assumptions were tested: Increasing structural complexity would decrease capture rates, and foraging efficiency would increase with the number of times that ibises were exposed to the challenge when controlling for all factors. We used foraging efficiency (prey de-

* To whom correspondence may be addressed (evan.adams@maine.edu).
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pleted in a given time) and motivation (numbers of birds attempting to forage over time) of groups of foraging ibises as response variables to four different exposure levels of mercury and four different levels of habitat complexity.

MATERIALS AND METHODS

We studied 168 captive ibises in a large, free-flight aviary at the U.S. Department of Agriculture Wildlife Research Center (Gainesville, FL, USA). These birds were collected as nestlings at 10 to 35 d of age from breeding colonies in the northern Everglades (Broward County, FL, USA; 26°11.179′N, 80°31.431′W) and from White Springs (Hamilton County, FL, USA; 30°19.900′N, 82°45.367′W). Young birds were randomly assigned to one of four dietary exposure groups receiving 0, 0.05, 0.1, or 0.3 mg/kg (wet wt in diet) of methylmercury beginning after approximately 90 d of age on June 17, 2005. These levels of exposure mimic the range that might be encountered by these birds in the Everglades [26,27]. Methylmercury was introduced into the diet by spraying food pellets (Mazuri Flamingo Breeder Diet; Mazuri, St. Louis, MO, USA) with a solution of methylmercury salt dissolved in corn oil while rotating the mass in a cement mixer. Sprayed food was tested routinely for methylmercury concentration throughout the study period. Mercury was measured at the Florida Department of Environmental Protection, Chemistry Section (Tallahassee, FL, USA) [26], based on U.S. Environmental Protection Agency method 245.1 (minimum detection level, 0.5 ppb; practical quantification level, 1.5 ppb) [28]. Exposure groups were housed in the same circular, open-air aviary (1,200 m^2) separated into quadrants by interior net walls; each quadrant houses one of four methylmercury exposure group, thus eliminating replication for each experimental unit—that is, the enclosed group of ibises. This arrangement was requisite because of the highly social and colonially breeding nature of the study species and our additional interest in the effects of methylmercury on their breeding biology. Despite the obvious experimental flaws in this arrangement, we believe that location effects are unlikely to affect foraging behavior.

The foraging experiment was run from October 11 to November 17, 2005. During each daily bout, all treatment groups were simultaneously presented with 200 live fathead minnow (*Pimephales promelas*) juveniles in 2.4- × 3.7-m rectangular foraging pools between 8:00 AM and 9:00 AM. All groups were given access to the fish for 15 min. The foraging arenas were all of similar proportion and arrangement with continuously varying water depths of 2 to 15 cm as a result of a sloping floor. Water depth was standardized in each pool for each experimental trial each morning because of the possibility of prey availability differing with water depth. We also placed varying levels of physical structure in the pools as needed (see below). All cages were deprived of food starting at sundown the night before each bout, and food was restored ad libitum after each trial. All trials were recorded on video and reviewed later to determine how many birds participated at standard times in each bout.

The experiment was run for six weeks, and during each week, each enclosure experienced four test bouts in each of four different levels of structural complexity. Each treatment group experienced one structural complexity trial per day and four different trials per week. Because no treatment group could receive the same structural complexity on the same day, a Latin square experimental design was employed to remove such temporal biases, and the order of given habitat complexity was pseudorandom. The four levels of structural complexity were open pools; pools with horizontal panels of rigid steel agricultural fencing (mesh size, 13–38 cm^2) supported approximately 3 cm off the bottom of the pool and occupying the entire surface of the pool; pools as just described, but with six pieces of 1- × 1-m shade cloth and approximately 30 artificial plant leaves/fronds attached to the panels; and pools as just described, but with 16 pieces of shade cloth and approximately 60 artificial plant leaves/fronds. All shade cloth and plastic plants provided a partial visual and physical barrier but were flexible (i.e., easily moved by ibises); thus, all space in the foraging arena was accessible to the ibises.

After birds had foraged for 15 min, two researchers entered the cages and placed large pieces of shade cloth over the arena to halt foraging. Once foraging was stopped in all cages, we drained each foraging arena through a seine net and counted the remaining fish. This technique removed error associated with the detectability of fish swimming in the arena.

Video recordings were used to obtain an accurate measure of the number of birds in each treatment group that were foraging at standardized times. Each video was analyzed at 30, 60, 240, 420, 600, and 780 s by counting the number of ibises in (not around or on the edges of) the wading pool. We estimated the total number of bird-minutes foraged during the entire bout by summing rectangles bounded by the total time foraged and the number of birds to roughly estimate the area under the curve. Different levels of habitat complexity yielded different amounts of time spent by birds in the foraging arena; this made simple means impractical for describing differences in foraging motivation between groups. This approximation was a consistent underestimate of the true area under the curve, but it gave us a consistent measure that could describe and compare a wide variety of foraging effort curves.

We used a repeated-measures general linear model for statistical analysis. The response was the proportion of fish remaining, and the dependent variables were day, week, methylmercury exposure group, and structural complexity. To normalize our proportional response, we added one to the number of fish remaining and to the number of fish presented and then arcsine square-root transformed the data to yield the resulting modified proportion. This transformation was found to produce a set of residuals with an approximately normal distribution. We also modeled group motivation with the same factors to test our assumption of equal motivation among groups and across time and structural complexity levels. We used analysis of variance to compare numbers of birds foraging across exposure groups while controlling for the effects of structural complexity. We selected models from an a priori set of models that included all three base factors (methylmercury exposure, habitat complexity, and time), and all higher-order interactions with time included a time × time interaction. The linear models were analyzed using SAS® Version 7.2 (PROC MIXED; SAS Institute, Cary, NC, USA), and all other statistics were analyzed using JMP IN Version 5.1 (SAS Institute). Alpha was equal to 0.05.

RESULTS

Time, structural complexity, and exposure group were all included as main effects in our best model (Table 1) of foraging efficiency. The major differences between factors was in the interactions between them: The interaction between structural complexity and time was significant (and quadratic), whereas the interaction between methylmercury exposure and time was

Table 1. Effect of time, structural complexity, and methylmercury exposure group on white ibis (*Eudocimus albus*) foraging efficiency in the highest-ranked model[a]

Effect	p
Time	<0.0001
Exposure group	0.0038
Time × time	<0.0001
Structural complexity	<0.0001
Structural complexity × time	<0.0001
Structural complexity × time × time	0.0061

[a] Significance was determined using an F test based on the Kenward-Rogers estimation for degrees of freedom.

not. The resulting model fit well, and all these terms were considered to be biologically important and plausible. The repeated-measures aspect of our analysis (date) explained a small amount of the variance; this implies that our Latin square experimental design was effective in removing the potential bias of day effect.

The hypothesis that habitat complexity would decrease foraging efficiency was supported by our data (Fig. 1). Foraging efficiency decreased with increasing vegetation/structure (Table 2), and this effect varied with time. All methylmercury exposure groups showed both linear and exponential increases in foraging efficiency over time that depended on habitat complexity. The highest and second-highest habitat complexity exhibited significantly greater positive changes in foraging efficiency (the time × complexity term) and different curve shapes (the time × time × complexity effect) when compared to the two lower levels of complexity (Table 2). Thus, the assumption that feeding efficiency would increase over time was supported, and the degree of improvement was related to the degree of structural complexity.

Although we predicted that methylmercury exposure would decrease foraging efficiency, result in a change in foraging efficiency over time, or both, we did not find any interaction of methylmercury exposure group with time. Foraging efficiency differed between methylmercury exposure groups (Fig. 2). The medium- and low-treatment groups were statistically more efficient foragers than the control, and the high-dose group was not significantly different from the control (Table 2). Contrary to our predictions, this relationship was nonlinear. These results imply that methylmercury did not alter the improvement in foraging by these birds over time and, thus, does not support our initial hypothesis of learning impairment.

One assumption of the experiment was that foraging efficiency during a foraging bout would not decline as a result of decreased foraging effort. The number of birds foraging during the course of the bouts was first analyzed as a response variable using the same model, factors, and interactions as used with foraging efficiency. This model showed no significant effects. When the model was stripped down to the main effects, structural complexity was significant ($p < 0.0001$), whereas time and exposure group were not (Table 3). In summary, our attempt to control motivational differences between exposure groups via temporary food restriction appeared to be successful.

DISCUSSION

One of our objectives was to establish a foraging environment that would challenge birds regardless of methylmercury

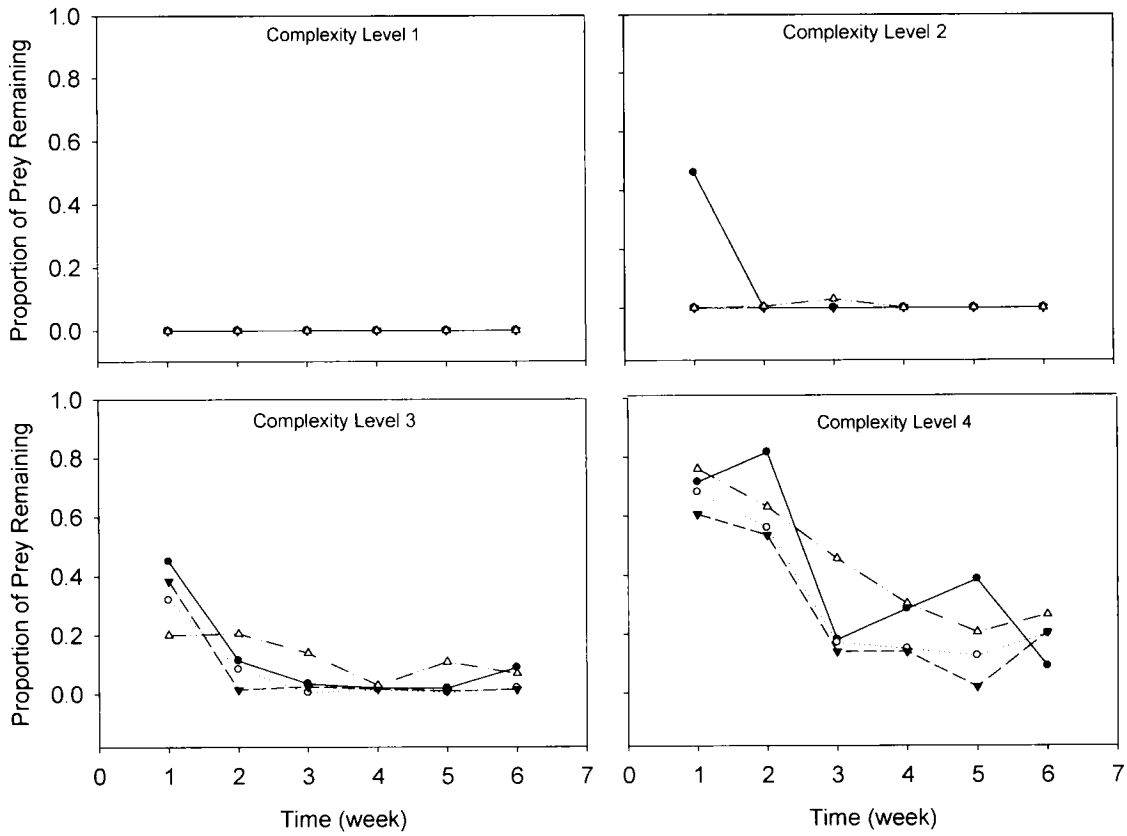

Fig. 1. Mean proportion of fish remaining after each 15-min foraging trial, with each line representing a methylmercury exposure group and each graph a level of foraging habitat difficulty. Thus, we see the pattern of foraging efficiency improvement for each treatment group controlling for habitat complexity. —●— = control; - -○- - = low treatment; —▼— = medium treatment; - -△- - = high treatment.

Table 2. Parameter estimates for effects of experimental variables on foraging efficiency of white ibis (*Eudocimus albus*) using the highest-rank model[a]

Effect	Parameter estimate (β)	SE	*p*
Time	−0.354	0.06221	<0.0001
Time × time	0.03337	0.008699	0.0002
Methylmercury exposure			
Control			
Low	−0.07665	0.03069	0.0145
Medium	−0.09188	0.03069	0.0036
High	−0.00482	0.03069	0.8756
Habitat complexity			
High			
Medium	−0.4599	0.1345	0.001
Low	−1.0468	0.1345	<0.0001
Control	−1.2913	0.1345	<0.0001
Habitat complexity × time			
High			
Medium	0.007242	0.08797	0.9346
Low	0.2388	0.08797	0.0081
Control	0.3547	0.08797	0.0001
Habitat complexity × time × time			
High			
Medium	0.006126	0.0123	0.008
Low	−0.02057	0.0123	0.0983
Control	−0.03347	0.0123	0.6199

[a] The parameter estimate is the relative magnitude of difference between the reference group (the control for methylmercury exposure and the high-complexity group for habitat) and the group of interest. SE = standard error of the test.

exposure. This was achieved, as evidenced by the inhibitory effect of increasing structural complexity. Another goal was to establish an environment in which the birds might learn how to improve foraging efficiency over time and, thus, test whether learning might be affected by treatment. This goal was achieved, as evidenced by the significantly negative effect of time on foraging efficiency. We also were able to demonstrate that motivation was similar across treatment groups, suggesting that it was not a confounding factor.

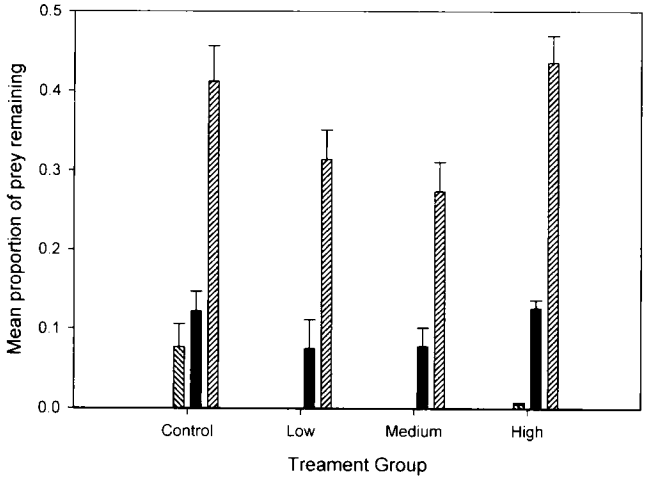

Fig. 2. Mean proportion of prey remaining for all weeks for each methylmercury exposure group and each structural complexity group. Error bars represent the standard error of the mean for each group. Different patterns represent different levels of habitat complexity. □ = control; ▨ = low treatment; ■ = medium treatment; ▨ = high treatment.

Table 3. Mean foraging motivation score and standard errors by exposure group, habitat complexity, and week of experiment[a]

Factor	Group	Mean score (bird-seconds)	SE
Exposure group	Control	13,209	845
	Low	13,605	827
	Medium	12,918	827
	High	13,361	827
Habitat complexity	Control	8,413	488
	Low	12,490	488
	Medium	15,743	499
	High	16,554	488
Period	1	13,896	1,013
	2	12,438	1,046
	3	13,133	1,013
	4	14,285	1,013
	5	13,283	1,013
	6	12,559	1,013

[a] Mean score is the average cumulative bird-time for each group within a factor, an estimate of area under the number of birds versus time curve. SE = standard error of each group mean.

Although we found a significant effect of methylmercury exposure in this experimental context, the effect of methylmercury did not increase with exposure. The 0.05 and 0.1 ppm Hg/d groups were more efficient foragers than the control, and the control and high-dose groups were similar in efficiency. Our strongest prediction was that the high-dose and control groups should have provided the greatest contrast in mercury effects, yet they were not significantly different. Additionally, the effect of methylmercury—even though statistically significant—was quite weak when compared to habitat complexity or time.

Over time, all treatment groups improved in their foraging efficiency. The results indicate that all groups learned at an exponential rate (the time × time term in the model) when challenged by higher habitat complexity and that the degree of improvement in foraging efficiency depended on the difficulty of the task (the time × complexity and time × time × complexity terms). Therefore, the prediction that methylmercury exposure (at the dose rates given) would inhibit learning seemed to have little supporting evidence. This finding does not rule out the possibility that learning may be affected at higher dose rates, in different behavioral endpoints, or in the offspring of methylmercury-exposed birds, but our current study finds no evidence to support this hypothesis.

Two potential explanations exist for the nonlinear effect of mercury seen in the present experiment: Hormesis, and/or confounding effects. Hormesis suggests that certain toxicants stimulate an animal in apparently positive ways at low doses, yet cause negative effects at higher doses [29,30]. Nonlinear patterns have been found increasingly in low-exposure studies using endocrine disruptors, especially with behavioral endpoints [16]. The amount of methylmercury to which these birds were exposed could be considered quite low by comparison with the existing literature measuring effects [2], and it is possible that we approached a threshold for hormesis for the foraging efficiency endpoint. Verification of this hypothesis would require repeating the experiment and expanding the dose range. Two potential variables could confound the effects of methylmercury exposure: Individual composition of groups, and cage location. Because of unknown differences in the social composition of treatment groups and asymmetrical location effects (e.g., the differential effect of light or shade on different cages), some cages might have been predisposed to

forage more or less efficiently than others. We do not know of any ready mechanisms that suggest such a location effect. We also cannot rule out such an effect, however, because each group contained only a single replicate.

Increasing structural complexity made it more difficult for ibises to forage and encouraged the birds to forage for longer periods within bouts. Whereas availability of prey to visual and tactile aquatic birds is known to be affected by prey density, hydroperiod, temperature, dissolved oxygen, and water depth [23,25,31–33], the effect of vegetation and obstructions has received relatively little attention [33,34]. The ibises showed improvement in the more challenging habitat structure, but they never achieved the levels of efficiency seen in the low-complexity environment. Such an effect has been suggested by habitat-selection studies of wading birds [34–36]; however, we believe this is the first experimental evidence for an effect of structural complexity on foraging by long-legged wading birds. It therefore seems likely that vegetation density and type is an important determinant of foraging success and habitat selection for tactile-foraging waders.

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REFERENCES

1. Scheuhammer AM, Meyer MW, Sandheinrich MB, Murray MW. 2007. Effects of environmental methylmercury on the health of wild birds, mammals, and fish. *Ambio* 36:12–19.
2. Wolfe MF, Scharzbach S, Sulamen RA. 1998. Effects of mercury on wildlife: A comprehensive review. *Environ Toxicol Chem* 17: 146–160.
3. Scheuhammer AM. 1987. The chronic toxicity of aluminum, cadmium, mercury, and lead in birds: A review. *Environ Pollut* 46: 263–295.
4. Heinz GH. 1979. Methylmercury: Reproductive and behavioral effects on three generations of mallard ducks. *J Wildl Manag* 43: 394–401.
5. Heinz GH, Locke LN. 1976. Brain lesions in mallard ducklings from parents fed methylmercury. *Avian Dis* 20:9–17.
6. Spalding MG, Frederick PC, McGill HC, Bouton SN, McDowell LR. 2000. Methylmercury accumulation in tissues and its effects on growth and appetite in captive great egrets (*Ardea alba*). *J Wildl Dis* 36:411–422.
7. Spalding MG, Frederick PC, McGill HC, Bouton SN, Richey LJ, Schumacher IM, Blackmore CGM, Harrison J. 2000. Histologic, neurologic, and immunologic effects of methylmercury in captive great egrets. *J Wildl Dis* 36:423–435.
8. Nocera JJ, Taylor PD. 1998. In situ behavioral response of common loons associated with elevated mercury (Hg) exposure. *Conserv Ecol* 2:10.
9. Bouton SN, Frederick PC, Spalding MG, McGill H. 1999. Effects of chronic, low concentrations of dietary methylmercury on the behavior of juvenile great egrets. *Environ Toxicol Chem* 18: 1934–1939.
10. Facemire CF, Gross TS, Guillette LJ Jr. 1995. Reproductive impairment in the Florida panther: Nature or nurture? *Environ Health Perspect* 103(Suppl 4):79–86.
11. Thaxton JP, Gilbert J, Hester PY, Brake J. 1981. Mercury toxicity as compared to adrenocorticotropin-induced physiological stress in the chicken. *Arch Environ Contam Toxicol* 11:509–514.
12. Heath JA, Frederick PC. 2005. Relationships among mercury concentrations, hormones, and nesting effort of white ibises (*Eudocimus albus*) in the Florida Everglades. *Auk* 122:255–267.
13. Schlinger BA. 1997. Sex steroids and their actions on the birdsong system. *J Neurobiol* 33:619–631.
14. Gahr M. 2004. Hormone-dependent neural plasticity in the juvenile and adult song system: What makes a successful male? *Ann NY Acad Sci* 1016:684–703.
15. Schantz SL, Widholm JJ. 2001. Cognitive effects of endocrine-disrupting chemicals in animals. *Environ Health Perspect* 109: 1197–1206.
16. Zala SM, Penn DJ. 2004. Abnormal behaviors induced by chemical pollution: A review of the evidence and new challenges. *Anim Behav* 68:649–664.
17. Kitayski AS, Kitaiskania EV, Piatt JF, Wingfield JC. 2003. Benefits and costs of increased levels of corticosterone in seabird chicks. *Horm Behav* 43:140–149.
18. Wingfield JC, Maney DL, Breuner CW, Jacobs JD, Lynn S, Ramenofsky M, Richardson RD. 1998. Ecological bases of hormone-behavior interactions: The "emergency" life-history stage. *Integrative and Comparative Biology* 38:191–206.
19. Ketterson ED, Nolan V Jr. 1992. Hormones and life histories: An integrative approach. *Am Nat* 140:S33–S62.
20. Vos JG, Dybing E, Greim HA, Ladetoged O, Lambre C, Tarazona JV, Brandt I, Vethaak AD. 2000. Health effects of endocrine-disrupting chemicals on wildlife, with special reference to the European situation. *Crit Rev Toxicol* 30:71–133.
21. Kushlan JA. 1974. The ecology of the white ibis in southern Florida, a regional study. PhD thesis. University of Miami, Miami, FL, USA.
22. Frederick PC, Collopy MW. 1989. Nesting success of five ciconiiform species in relation to water conditions in the Florida Everglades. *Auk* 106:625–634.
23. Gawlik DE. 2002. The effects of prey availability on the numerical response of wading birds. *Ecol Monogr* 72:329–346.
24. Frederick PC, Spalding MS. 1994. Factors affecting reproductive success in wading birds (Ciconiiformes) in the Everglades ecosystem. In Dabis SM, Ogden JC, eds, *Everglades: The Ecosystem and Its Restoration*. St. Lucie, Del Ray Beach, FL, USA, pp 659–691.
25. Frederick PC. 2001. Wading birds in the marine environment. In Schreiber BA, Burger J, eds, *The Biology of Marine Birds*. CRC, Boca Raton, FL, USA, pp 617–655.
26. Frederick PC, Spalding MG, Dusek R. 2002. Wading birds as indicators of mercury contamination in Florida, USA: Annual and geographic variation. *Environ Toxicol Chem* 21:213–255.
27. Loftus WF. 2000. Accumulation and fate of mercury in an Everglades aquatic food web. PhD thesis. Florida International University, Miami, FL, USA.
28. Florida Department of Environmental Protection. 2007. Trace-level total mercury analysis in tissue by cold-vapor atomic fluorescence (CVAF). Method HG-007, Revision 1.9. HG-007-1.9. Bureau of Laboratories, Tallahassee, FL, USA.
29. Noel HL, Hopkin SP, Hutchison TH, Williams TD, Sibly RM. 2006. Towards a population ecology of stressed environments: The effects of zinc on the springtail *Folsomia candida*. *J Appl Ecol* 43:325–332.
30. Calabrese EJ. 2005. Historical blunders: How toxicology got the dose–response relationship half-right. *Cell Mol Biol* 51:643–654.
31. Maccarone AD, Brzorad JN. 2005. Foraging microhabitat selection by wading birds in a tidal estuary, with implications for conservation. *Waterbirds* 28:383–391.
32. Smith SM, Gawlik DE, Rutchey K, Crozier GE, Gray S. 2003. Assessing drought-related ecological risk in the Florida Everglades. *J Environ Manag* 68:355–366.
33. Bancroft GT, Gawlik DE, Rutchey K. 2002. Distribution of wading birds relative to vegetation and water depths in the northern Everglades of Florida, USA. *Waterbirds* 25:265–277.
34. Surdick JA Jr. 1998. Biotic and a biotic indicators of foraging site selection and foraging success of four ciconiiform species in the freshwater Everglades of Florida. PhD thesis. University of Florida, Gainesville, FL, USA.
35. Stolen ED. 2006. Habitat selection and foraging success in wading birds in impounded wetlands in Florida. PhD thesis. University of Florida, Gainesville, FL, USA.
36. Bancroft GT, Strong AM, Sawicki RJ, Hoffman W, Jewell SD. 1994. Relationship among wading bird foraging patterns, colony locations, and hydrology in the Everglades. In Dabis SM, Ogden JC, eds, *Everglades: The Ecosystem and its Restoration*. St. Lucie, Del Ray Beach, FL, USA, pp 615–657.