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Testing the role of contaminants in depressing avian numbers

Evaluando el rol de los contaminantes sobre la disminución del número de aves

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ABSTRACT

Environmental contaminants are ubiquitous and so are often key suspects in cases of lagging wildlife populations. How do we test hypotheses about cause-effect linkages between contaminants and wildlife health? We present three case studies in which different approaches were used to test hypotheses about effects of contaminants on wildlife. The cases involve the possible impacts of (1) polychlorinated biphenyl on Lake Superior bald eagles (*Haliaeetus leucocephalus*); (2) dioxin on osprey (*Pandion halieatus*); and (3) methyl mercury on common loons (*Gavia immer*). The different approaches were dictated by legal, logistic, and financial limitations, but the relative strengths of experimental and mechanistic approaches over correlative approaches is underscored. For all three species, the simple correlation between a single contaminant and performance was confounded by covariation with other types of contaminants and/or natural ecological factors such as food availability and predation.

Key words: Applied physiological ecology, ecotoxicology, birds, chemical contaminants.

RESUMEN

Los contaminantes ambientales son ubicuos y a menudo los principales sospechosos en los casos de disminución en las poblaciones de fauna silvestre. ¿Cómo probamos las hipótesis sobre las relaciones causa - efecto entre los contaminantes y la salud de la fauna silvestre? Presentamos tres estudios de caso en que se usaron diferentes aproximaciones para someter a prueba las hipótesis sobre los efectos de los contaminantes sobre la fauna silvestre. Los casos involucran los posibles impactos de (1) el bifenilos policlorados en el águila calva (*Haliaeetus leucocephalus*) en el Lago Superior; (2) las dioxinas sobre el águila pescadora (*Pandion halieatus*); y (3) el mercurio de metilo en colimbo grande (*Gavia immer*). Las limitaciones legales, logísticas y financieras, determinaron diferentes aproximaciones en estos estudios, pero se destaca que la fuerza relativa de las aproximaciones experimentales y mecanicistas es superior a la de un acercamiento correlacional. Se demuestra que, en todas las especies la correlación simple entre un solo contaminante y su actuación esta enmascarada por su covariación con otros tipos de contaminantes y/o los factores ecológicos naturales como disponibilidad de comida y depredación.

Palabras clave: Fisiología ecológica aplicada, ecotoxicología, aves, contaminantes químicos.

INTRODUCTION

Ecotoxicology, the study of toxicant effects on ecological processes, typically receives scant attention from animal physiological ecologists, but arguably it should occupy a more prominent position in their research portfolio. First, toxicants are ubiquitous. This point is increasingly clear based on the documented examples of contaminated biota in seemingly isolated and pristine locations (e.g., Kidd et al. 1995) and on our increasing understanding of the responsible processes that move toxicants sometimes long distances between the earth's ecosystems. We may want to study the natural ecology of our favored organism, but we must acknowledge the possibility that contaminants may be a notable factor influencing its ecology alongside more natural processes.

Second, among ecologists, it is physiological ecologists who have certain specialized background and tools to study the impact of toxicants on wild animals. Their mechanistic perspective and approach is important for thorough elucidation of cause-effect linkages and for developing predictive ability, which are two important goals of ecotoxicological study.

(Managed by F. Bozinovic)

Society is very interested in relationships between environmental contaminants and numbers or impacts on wildlife for very practical reasons. A decline in wildlife coincident with a rise in some environmental chemical can provide humans with an early warning that the chemical might pose a toxic risk to humans or their domestic animals. The classic example of this is that in the 1950's and 60's the ecological response of predatory birds to DDT (dichlorodiphenyltrichloroethane) and its metabolite DDE (dichlorodiphenylethane) stimulated the investigations that convinced most people that these chemicals were a risk to humans as well as wildlife (Peterle 1991, Walker et al. 1996). Bill malformations and apparently low egg hatchability in terns (*Hydroprogne caspia*), cormorants (*Phalacraocorax auritus*), and other communally nesting birds alerted us in the 1970's and 80's to the problems associated with polychlorinated biphenyl (PCB) contamination in the Great Lakes region of North America (Peterle 1991, Walker et al. 1996), though later research failed to find a tight cause-effect linkage (Custer et al. 1999). Some people believe that this story is being replayed again today with regard to environmental contaminants that are putative endocrine disruptors (Colborn et al. 1997).

How do we test hypotheses about cause-effect linkages between contaminants and wildlife health? In many cases what we begin with are associations between environmental chemicals and wildlife numbers. Epidemiology has established criteria to decide whether statistical association(s) might reflect a cause-effect relationship (Fox 1991). However, scientists are taught early that correlation is a relatively weak tool for assigning causation. It is common for wildlife to be exposed to multiple different contaminants at the same time and for wildlife responses to be simultaneously correlated to levels of several contaminants. In the aforementioned case of cormorants in the Great Lakes, the correlation of PCBs with increased egg mortality and bill malformations was confounded by DDE, casting doubt on exactly what factor was the causative agent (Custer et al. 1999). In the case of bald eagles in the Great Lakes, levels in eggs of PCBs, DDE, and Me-Hg were correlated with each other (Wiemeyer et al. 1993), with the paradoxical result that egg shell thinning was associated with PCBs even though these contaminants have not been shown to cause this effect. Recognizing this problem, one must also acknowledge the possibly confounding effects of other correlated factors, anthropogenic or natural, that the investigator fails even to recognize or imagine.

We present three case studies that rely on correlative and other approaches to test hypotheses about effects of contaminants on wildlife. The cases involve the possible impacts of (1) PCBs on Lake Superior bald eagles (*Haliaeetus leucocephalus*); (2) 2,3,7,8-tetrachlordibenzo-*p*-dioxin (TCDD) on osprey (*Pandion halieatus*); and (3) methyl-mercury (Me-Hg) on common loons (*Gavia immer*). The lipophilic organochlorines (PCBs and TCDD), and Me-Hg which binds to sulfhydryl groups of proteins, undergo significant biomagnification as they progress through increasing trophic levels within a food chain (Paasivirta 1991). Therefore, these piscivorous species merit special attention as they provide information on the impact of maximum exposure levels in an ecosystem, and because the toxic effects might only manifest in these species. The different approaches applied in the case studies were dictated by legal, logistic, and financial limitations, and in all cases the weaknesses of correlative approaches became apparent.

TESTING THE IMPACT OF PCBS ON LAKE SUPERIOR BALD EAGLES

Between 1940 and 1970, populations of bald eagles nesting in the continental United States declined due to reproductive failure caused mainly by DDE, a metabolite of the organochlorine DDT (Wiemeyer et al. 1972, Grier 1982, Colborn 1991). After the use of DDT and other organochlorines was banned, the North American eagle population rebounded quickly throughout most of its range. However, bald eagle populations in a few regions, including the Great Lakes shorelines (Colborn 1991, Best et al. 1994), have not increased as rapidly. The mean productivity of eagles near the shores of Lake Superior from 1989-1993 was 1 young per breeding attempt, significantly ($P < 0.001$) lower than the 1.3 young produced per breeding attempt in inland Wisconsin (Dykstra et al. 1998). Thus, in the early 1990s, some researchers still considered that organochlorine contaminants were depressing productivity of eagles along the Lake Superior shoreline (Kozie 1986, Kozie & Anderson 1991). The most likely candidate contaminants seemed to be PCBs, because concentrations of DDE in addled eggs had declined significantly since 1969 to a mean level in the early 1990's (2,100 μg/kg; Dykstra et al. 1998) below the no-effect concentration associated with normal reproduction (3,000-3,600 μg/kg DDE; Wiemeyer et al. 1984, Wiemeyer et al. 1993). PCBs had also declined, but the mean level in the early 1990s (10,000 μg/kg; Dykstra et

al. 1998) still exceeded what some researchers considered to be the no-effect concentration (4,000 µg/kg; Giesy et al. 1995). A reasonable hypothesis was that PCBs were depressing eagle productivity along the Lake Superior shoreline (Colborn 1991).

An alternative hypothesis was that contaminants were not the primary cause of low productivity of bald eagles, but that the reproductive rate had stabilized at a naturally-low level, perhaps because Lake Superior is simply marginal habitat for nesting bald eagles (Dykstra et al. 1998). The lake is deep, cold, and oligotrophic with few shoals where fish can spawn and where piscivorous birds can effectively forage (Weseloh et al. 1994). Thus, natural low food availability may explain lower productivity of bald eagles on Lake Superior's shores.

Ideally, one might design an experimental study to test these hypotheses, but a manipulative field study, or a laboratory dose-response study with large numbers of captives, was not possible considering the eagle's protected status as an endangered species and as the United States' national bird. However, as discussed below, additional measures of exposure of wild eagles to PCBs cast doubt on the contaminants hypothesis, whereas physiological/ecological study of eagle foraging energetics yielded data consistent with the food availability hypothesis.

Between 1989 and 1994 blood samples were collected from 89 bald eagle nestlings at 53 nesting territories in Wisconsin (Table 1). At territories along Lake Superior nestling plasma total PCB level was higher ($P = 0.002$) and nest productivity was lower ($P < 0.001$ for 1983-1994) than at territories in inland Wisconsin, consistent with the contaminants hypothesis. The association breaks down, however, when one considers that at territories along the Wisconsin River total PCB levels were as high as at Lake Superior but nest productivity was also high. Furthermore, log-transformed concentrations of total PCBs in nestling plasma were not correlated to average 5-year productivity ($P = 0.114$) (Dykstra et al. 1998) whether the regions (Lake Superior, inland Wisconsin) were analyzed together or separately. Arguably, the absence of a correlation that is predicted from a hypothetical cause-effect linkage is cause for rejecting the hypothesis, if the test is powerful.

Eagle foraging energetics at nest territories along Lake Superior and in inland Wisconsin were studied in 1992 and 1993 using behavioral observations (Dykstra et al. 1998) and the doubly labeled water method after validating it with captive (Dykstra et al. 1997) and free living bald eagles (Dykstra 1995). At inland Wisconsin nests metabolizable energy delivered per nest increased with increasing brood size, as expected (1-tailed test, $P = 0.047$, Fig. 1). At Lake Superior nests, adults with a brood of one delivered a similar amount of energy per day as parents at inland territories ($P = 0.6$), but in contrast to the inland parents they failed to increase energy delivery rate for larger broods ($P = 0.72$, Fig. 1). The ability to deliver apparently normal amounts of energy to broods of one suggests that the Lake Superior parents were not impaired by contaminants, whereas the inability to increase prey delivery to larger broods is consistent with the food limitation hypothesis. Other observations consistent with the latter hypothesis were that prey delivery rates at inland nests were highly correlated with productivity ($P = 0.001$) (Dykstra et al. 1998), observed nestling energy intake at Lake Superior nests was marginally smaller than the minimum requirement for broods of two but not one (Dykstra 1995), and the Lake Superior chicks in broods of two modified their behavior in an energy conserving fashion (Dykstra 1995).

TABLE 1

Contaminants in plasma of nestling bald eagles in Wisconsin, and nest productivity, 1989-1994

Contaminantes presentes en plasma de águila calva en Wisconsin y productividad del nido entre 1989 y 1994

Region	DDE [1] (µg/kg)	PCBs [1] (µg/kg)	Productivity[2] 5-yr avg
Superior lakeshore	19 (n = 36)	109 (n = 36)	0.99 ± 0.13 (n=15)
Inland WI	3.3 (n = 31)	28 (n = 31)	1.22 ± 0.11 (n=27)
WI River	2.6 (n = 9)	122 (n = 9)	1.76 ± 0.15 (n=7)

[1]Geometric means (n = number of samples), [2]Productivity = (number of young produced in 5 yr)/(number of times a breeding attempt occurred in 5 yr at a nest site) (n = number of nests) from Dykstra et al. (1998) Journal of Great Lakes Research 24: 32-44.

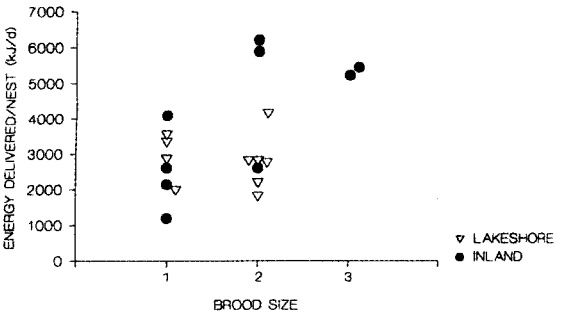

Fig. 1. Metabolizable energy delivered to nests as a function of brood size and location. Energy calculated from observed prey deliveries. Lakeshore nests are represented as open triangles (n = 10), inland nests as filled circles (n = 9). From Dykstra (1995).

Energía metabolizable aportada al nido en función del tamaño de la nidada y la ubicación. La energía fue estimada de la observación de presas aportadas. Los triángulos representan nidos de rivera (n = 10), y los círculos llenos a nidos en islas (n = 9).

In summary, the correlation of higher PCBs and lower eaglet productivity at Superior led to the erroneous assignment of causation. Along some rivers in Wisconsin similarly high levels of PCBs are associated with higher eaglet productivity. Furthermore, adult and juvenile eagles perform normally along Lake Superior, except that prey delivery is too low to larger broods. Hence, low food availability is a sufficient explanation for the low eaglet productivity along Lake Superior. This conclusion is subject to the limitations inherent in correlations. More robust conclusions might be made from a food-provisioning experiment to test the effect of food availability on productivity of Lake Superior eagles, and a dose-response study with PCBs to test whether current exposure levels of Lake Superior eagles are at a no-effects level. Such studies may be possible following the de-listing of the bald eagle as an endangered species.

Testing the effects of 2,3,7,8 TCDD on Wisconsin River osprey

Ospreys (*Pandion halieatus*) are common breeders at the Wisconsin River's Castle Rock and Petenwell system (CR/P), adjoining reservoirs contaminated with planar halogenated hydrocarbons (PHHs; polychlorinated dibenzodioxins [PCDDs], polychlorinated dibenzofurans [PCDFs], and co-planar polychlorinated biphenyls [PCBs]). Two bleached kraft pulp mills located 7 km and 12 km upstream of the CR/P, historically discharged PHHs via effluents into the Wisconsin River (Kreitlow 1996). Elevated levels of 2,3,7,8-tetra-chlorodibenzo-*p*-dioxin (TCDD) in fish sampled from the CR/P, along with warnings against human consumption of certain fish species, predicted a significant risk to piscivorous birds foraging at the CR/P. Furthermore, the mean productivity of osprey nesting at the CR/P from 1988-1993 was 0.70 young per breeding attempt, significantly ($P < 0.001$) lower than the 1.26 young produced per breeding attempt at upstream locations along the Wisconsin River (Woodford 1996). Based on these findings, a study was designed to investigate the exposure of osprey to PHHs and the impact of PHH exposure levels to osprey breeding on the CR/P.

For an index of exposure, eggs were collected from osprey nests and levels of PHHs were determined (Table 2). Levels of TCDD in CR/P eggs were significantly higher than eggs from either reference area ($P < 0.001$, Table 2). However, DDE, DDT, co-planar PCBs and all other PCDD/PCDF congener levels were not different. Total PCB levels from CR/P eggs were significantly greater than level in eggs collected from the Rainbow reference area ($P < 0.05$).

An egg-exchange experiment was conducted in 1995 and 1996 to measure the effect of PHH exposure on osprey embryo survival and post-embryonic growth at the CR/P, and to detect a

TABLE 2

Contaminants in eggs of osprey along the Wisconsin River, 1992-1996

Contaminantes en huevos de águila pescadora a lo largo del Río Wisconsin durante 1992 - 1996

Study Area	2,3,7,8-TCDD[1] (pg/g)	PCBs[1] (ng/g)	DDE[1] (pg/g)
Castle Rock/Petenwell	50 (n=20)	1626 (n=9)	421 (n=9)
Mead (Reference)	2 (n=3)	NM[2]	NM[2]
Rainbow (Reference)	4.5 (n=12)	480 (n=12)	248 (n=12)

[1]Geometric mean for concentration per g whole egg, wet mass (n= number of eggs) [2]NM = not measured

possible cause-effect link (Woodford et al. 1998, Table3). The study tested the hypothesis that variation in the hatching rate is caused by direct exposure of the embryo to the toxicants (an intrinsic mechanism), altered parental care (an extrinsic mechanism), or both. By following chick survival and growth through fledging age, the study also experimentally tested the hypothesis that chicks fed a PHH contaminated diet were fledging and growing at a lower rate than those fed a less contaminated diet. The egg exchange was accomplished by entering nests once the entire clutch was present (6-8 d after initiation), replacing osprey eggs temporarily with chicken eggs painted to resemble osprey eggs (to prevent nest abandonment by the adults), and transporting the osprey eggs between habitats in a heated chest. Control eggs were either unswitched or were eggs at the reference sites switched among themselves to determine if the switching procedure biased egg hatching success. After the switches were accomplished, hatching was monitored and then surviving chicks were individually marked and measured (mass, culmen length) every 7 d. Because chicks, still unable to fly, may leap from the nest when disturbed after reaching 50 d of age, nests were sampled until the oldest chick reached this age. There were not enough nests and eggs to perform the entire experiment in one year, so Groups A,B, and C were studied in 1995 and Groups C and D were studied in 1996 (Table 3).

Hatching rates were uniformly high and did not differ significantly among the experimental groups (Table 4). The fledging rate of chicks in Group B (94%) was significantly greater than that of Group C chicks (67%, P = 0.03), but the hypothesis predicted a lower, not higher fledging rate for chicks with the greatest exposure to contaminants (via ingestion of contaminated fish) when compared to those with the least exposure. In fact, the fledging rates for all experimental groups were greater than those of the Group C in either year.

TABLE 3

Overall design, group designations, and year of measurement for the egg-exchange experiment with osprey

Diseño en conjunto, identificación de grupos y año de medición para el experimento de intercambio de huevos en águila pescadora

	Reference eggs	Contaminated area eggs
Reference Adults	Control Group Group C 1995 and 1996	Intrinsic Mechanism(s) Group A 1995
Contaminated Area Adults	Extrinsic Mechanism(s) Group B 1995	Both Mechanisms Group D 1996

TABLE 4

Hatchability and survivorship of osprey eggs and chicks in the egg exchange experiment

Éxito de eclosión y sobrevivencia de huevos de Aguila pescadora en experimento de intercambio de huevos

	Reference Eggs	Contaminated Area Eggs
Hatchability of osprey eggs in egg exchange experiment		
Reference Adults	73% (1995, n=33) 86% (1996, n= 21)	74% (n = 27)
Contaminated Area Adults	82% (n = 21)	91% (n = 23)
Fledging rate of osprey in egg exchange experiment		
Reference Adults	67% (1995, n=33) 50% (1996, n= 21)	73% (n = 27)
Contaminated Area Adults	94% (n = 21)	76% (n = 23)

Though neither the hatching or fledging data indicated a significant impact of TCDD exposure on osprey chicks, there was a hint of an effect on growth. A significant group effect for mass increase occurred during the first four weeks post-hatch for chicks measured in 1995 ($P = 0.04$, Fig. 2). Multiple individual comparisons between groups (i.e., A-B, B-C, A-C) found no significant differences, but the group with the highest mean mass was the control group. When mass measurements from Group D were added to the 1995 data for the first four weeks, the group effect became non-significant ($P = 0.09$, Fig. 2), while the brood size factor became significant ($P = 0.009$). Mass increase during the entire nestling period for Groups C and D was significantly affected by brood size and gender (brood size: $P < 0.001$, gender: $P < 0.001$). No other group or interactions were statistically significant. Culmen lengths did not differ significantly among the experimental groups (Woodford et al. 1998). If TCDD exposure did slightly depress osprey growth rate, the direct

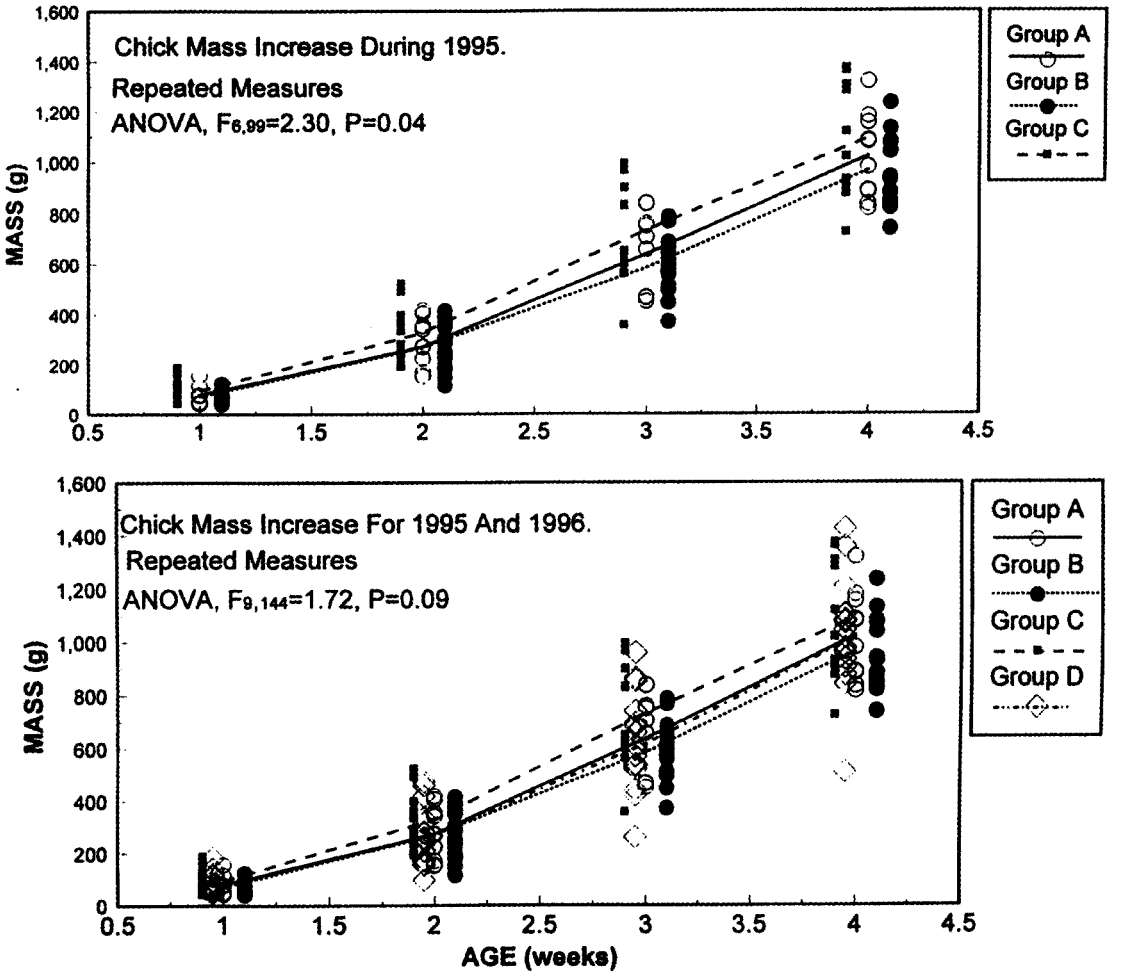

Fig. 2. Mass increases through the first 4 weeks of age for osprey chicks measured in 1995 and with the addition of group D chicks (measured in 1996). Symbols indicate masses of individual chicks with group means represented by lines. Group A chicks were from the contaminated area and raised at the reference areas. Group B chicks were from the reference areas and raised at the contaminated area. Group C chicks were eggs from the reference areas and raised at the reference areas. Group D chicks were eggs from the contaminated area and raised at the contaminated area. (From Woodford et al. 1998).

Incremento de masa durante las primeras cuatro semanas de edad en polluelos de águila medidos en 1995 y con la adición del grupo de pollos D (medidos en 1996). Los símbolos indican el peso corporal individual y las líneas representan el peso promedio grupal. Los polluelos del grupo A provenían del área contaminada y crecieron en las áreas de referencia. Los polluelos del grupo B provenían de las áreas de referencia y crecieron en el área contaminada. Los polluelos del grupo C corresponden a huevos provenientes y que crecieron en el del área de referencia. Los polluelos del grupo D corresponden a huevos provenientes y que crecieron en el área contaminada (en Woodford et al. 1998).

cause was most likely an intrinsic mechanism such as an endocrine or biochemical disruption in the chicks, because there was no difference among the experimental groups in parental food delivery rate or attentiveness (Woodford et al. 1998).

Overall, there was no evidence of a notable effect of TCDD exposure on osprey hatching success, survival, or growth rate. These results were not consistent with the hypothesis that TCDD or PHH exposure depressed osprey productivity at the CR/P. Concurrent with these studies, however, the Wisconsin Department of Natural Resources (WDNR) was engaged in other habitat manipulations whose results suggested a different explanation for depressed productivity at CR/P. After installing special physical guards at the base of osprey nest trees and poles to limit access by nest predators (e.g., racoons), the nest productivity of ospreys at the CR/P more than doubled from 0.70 young/occupied territory (1988-1993) to 1.73 (1994-1996) ($P < 0.001$), whereas a similar increase was not observed at the reference sites where a similar manipulation had been undertaken (Woodford 1996). Though these results are not from a study properly designed to test a hypothetical link between predation and osprey nest productivity, they are suggestive and serve as a cautionary note that correlations between contaminant exposure and lower nest productivity can be confounded by other correlated factors, anthropogenic or natural, that the investigator fails even to recognize or imagine at the outset.

Testing the effects of methyl-mercury on the common loon in Wisconsin

The ecological risk posed by atmospheric Hg deposition is a priority resource management concern for regulatory agencies and industries in the United States and elsewhere (EPA 1996; EPRI 1994). Hg deposition into aquatic environments with low pH is associated with relatively high risk because at low pH there is apparently increased generation of methyl mercury (Xun et al. 1987) which biomagnifies in aquatic food chains (Wiener 1987, Scheuhammer 1991) and is neurotoxic to vertebrates (Thomspon 1996, Goyer 1986) and causes reproductive impairment in birds at low exposure levels (Heinz 1979, Fimreite 1971).

Common loons are piscivorous aquatic birds that nest on numerous lakes in northern Wisconsin. Loons nesting on low-pH lakes receive greater Hg exposure than loons nesting on neutral-pH lakes (Meyer et al. 1995), and production of loon chicks was significantly lower at lakes where chicks had elevated blood Hg levels (Meyer et al. 1998). Establishing a cause-effect linkage between chick Hg exposure and loon productivity is confounded by the covariance between chick blood Hg and lake pH. An alternative hypothesis is that loon productivity is reduced by lesser prey abundance at low pH lakes (Meyer et al. 1998).

A dosing experiment is one experimental approach that can be used to test directly the effect of Hg exposure on loons. Furthermore, certain designs can provide information that permits a quantitative prediction of level of exposure, knowledge necessary to develop management scenarios that will protect loons and other avian piscivores. For example, applying pharmacokinetic methodology, one can predict the body Hg concentration (amount normalized to body mass) at time t (C_t) based on the fractional absorption of Hg in the food (a), the feeding rate (R, normalized to body mass), the Hg concentration in food (C_f), and the elimination rate constant for Hg (k_e) (Newman 1995, pg. 93): $C_t = a RC_f(1-e^{-ke\,t})/k_e$. Parameters R and C_f can come from approaches in physiological ecology (Nagy 1987) and measures on field-collected prey, respectively, and the other parameters can come from a dosing study. In our preliminary experiments with captives, for example, we dosed 85-day-old loon chicks with Me-Hg both orally (500 µg/kg body mass in a gelatin capsule inside a fish) and by intravascular injection (200 µg/kg body mass). Following a rapid decline probably due to initial distribution, blood Hg level of orally dosed birds declined in a log-linear fashion (Fig. 3). The slope, -0.006 d^{-1}, pro-

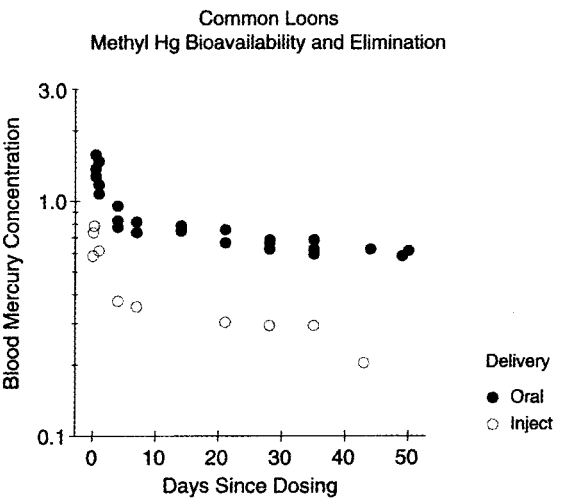

Fig. 3. Blood mercury concentration in common loons following oral (filled circles, n = 3) or intravascular dosing (unfilled circles, n = 1) with methyl mercury.

Concentración de mercurio en la sangre del colimbo grande luego de ingestión oral (circulos llenos, n = 3) o dosis intravascular (circulos vacios, n = 1) de mercurio de metilo.

vides an estimate of k_e. The fractional absorption of the orally administered Hg can be estimated by comparing the areas under the curves (AUC) of the oral and injected doses, normalized to differences in the dose levels (D): $a = [AUC^{0\rightarrow\infty}]_{oral} D_{injected} / [AUC^{0\rightarrow\infty}]_{injected} D_{oral}$ (pg. 103, Newman, 1995);). Values for AUCs are determined from the blood Hg plots following typical procedures in pharmacokinetics: the area from $t = 0$ to $t = x$ days (when the final blood sample was taken) is calculated using the trapezoidal method (Newman, 1995; pg. 101), the area from $t = x$ days to $t = \infty$ is calculated as $AUC^{x\rightarrow\infty} = (C \text{ at } t=x)/k_e$, and the total $AUC^{0\rightarrow\infty}$ is obtained by summing the two areas. The preliminary results to date (Fig. 3) indicate a fractional absorption of MeHg in fish of 90%, similar to values reported in laboratory animals (Clarkson 1972, Nielsen & Andersen 1991).

Using these data, along with estimates of feeding rates of loons, one can predict body Hg concentration of loon chicks eating fish with differing Hg levels (Fig. 4). In this example we used our data on age-specific feeding rates of captive loon chicks, but ultimately we will use feeding rates determined from our concurrent studies of field energetics using the doubly labeled water method. The model can be further refined by including a term for dilution of body Hg concentration during growth (Thomann 1981), and can be validated by measuring body Hg concentration in captives dosed at different levels and in free-living birds that are exposed to different dose levels because they feed on lakes with differing pH and hence fish Hg levels. Concurrent behavioral and physiological measures on captive birds with different experimentally determined body Hg concentrations are underway and will permit an assessment of the ecological consequences of exposure at different levels.

DISCUSSION AND NEW DIRECTIONS

These case studies on piscivorous birds in Wisconsin, U.S.A. highlight some of the common problems in ecotoxicological studies but also their overall success. Certainly among the successes

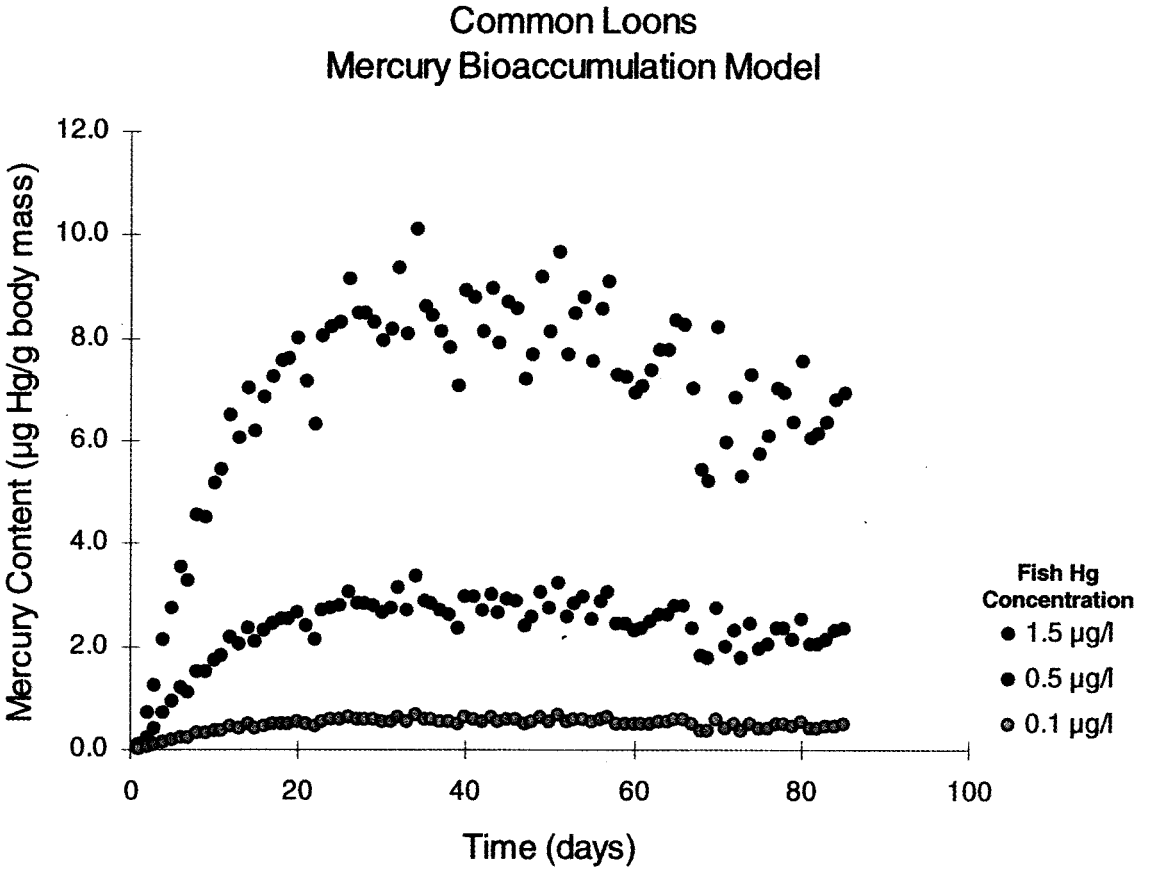

Fig. 4. A simulation for common loon chicks of whole-body mercury content over time following chronic ingestion of methyl mercury at three dietary concentrations (0.1, 0.5, 1.5 µg/g fish) (see text).

Una simulación del contenido corporal total de mercurio presente en polluelos del colimbo grande luego de la ingestión crónica de mercurio del metilo en tres concentraciones dietarias (0,1, 0,5, 1,5 µg/g pez) (ver texto).

one must count the identification of the ecological and human health problems associated with the presence of persistent, bioaccumulative organochlorines such as DDE and PCBs in the environment. Following legal restrictions on industrial production and/or use of these compounds in many countries, environmental levels have declined. In the Great Lakes region the levels of many toxicants are generally falling or have reached stable concentrations (Stow et al. 1995). The studies with eagles and osprey illustrate that even though present in some ecosystems, contaminant levels may be low enough not to be causing substantial effects on wildlife, even those at high tropic levels in a food chain.

Of course, there are important caveats to the general conclusion that the eagles and osprey we studied do not seem to be suffering any major effects from the levels of toxicants to which they are exposed. Important technical considerations particular to each study include questions about the relative potencies of the specific PCB congeners to which eagles were exposed at Lake Superior vs. inland Wisconsin sites (Dykstra et al. 1998), and questions about statistical independence of osprey eggs collected from the same nests (Woodford et al. 1998). Other important considerations apply to both studies. Possibly, the toxicants have their major impact on points in the life cycle that we did not study. Without further study one cannot easily discount hypotheses that eagles and osprey exposed early in life may have survived to fledging normally but then suffered lower long term survivorship or lower reproductive output. Also, tolerance of PHHs by birds and vertebrates generally is species specific (Hoffman et al. 1996; Peterson et al. 1993) and so we must be careful not to generalize the conclusions to all animals in these ecosystems. Comparisons indicate that the levels of PCBs that appeared to have little effect on bald eagles do negatively affect mink, at least in captivity (Eisler 1986, Peterson et al. 1993). As another example, the level of TCDD in osprey eggs that appeared to have no effect on hatching success is an order of magnitude higher than the level that reduces hatching success of chickens, the most sensitive avian species studied to date.

Considering these and other theoretical and practical limits to scientific knowledge, it is important not to discount the possible impact of toxicants on wildlife, even when not apparent in a study. However, these case studies provide ample warning of the pitfalls in attributing wildlife declines to contaminants. Both eagles and osprey were exposed to multiple different contaminants at the same time, a phenomenon common in wildlife that confounds the attempt to link exposure to one chemical to lower wildlife performance. Establishing these cause-effect linkages are also confounded by covariance with natural ecological factors. The correlation of PCBs and Me-Hg with eagle and loon productivity, respectively are arguably confounded by a correlation with food availability, and the correlation of TCDD with osprey productivity is arguably confounded by a correlation with predation.

It is important to disentangle these factors and solve these problems. For a compound like Hg, important regulatory changes are still on the horizon and the quantitative determination of exposure and toxicity to wildlife can contribute towards risk analysis. It is conceivable to develop models of environmental transport and fate of Hg linked to models of exposure and toxicity in fish and then piscivores, all leading to scientifically defensible regulatory goals and a detailed understanding of how, and at what cost, various regulatory scenarios will achieve the goals (Meyer, 1998). Increased understanding of cause-effect links between wildlife and contaminants are also important for toxicants already regulated. First, regulations are periodically modified based on new information. Second, even if production and release of a toxicant is banned, increased knowledge is necessary for planning ecosystem remediation and possibly for litigation considerations. For the Fox River-Green Bay ecosystem in Wisconsin, for example, decisions on whether to physically remove PCH-contaminated sediments (WIDNR, 1999), and to seek compensatory damages for the past industrial pollution, will hinge partly on the quantitative assessment of the effect of PCHs on wildlife (Exponent 1998).

Wildlife ecotoxicology presents important problems whose solutions can be usefully advanced by physiological ecologists. Tools and approaches associated with animal energetics, nutritional ecology, and pharmacokinetics were incorporated into the ecotoxicological studies of eagles, osprey, and common loons. If you are a researcher whose passion is to study how animals work, and how their functions influence their ecology, then you should consider doing research in ecotoxicology. Otherwise, you are missing an important opportunity to contribute something to society that it considers very important, and on a practical note, you are missing an important opportunity to secure funding from society for your scientific program.

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