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Evaluation of the Effects of Toxic Chemicals in Great Lakes Cormorants: Has Causality been Established?

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Abstract.—Toxic contaminants have influenced Double-crested Cormorant (*Phalacrocorax auritus*) reproduction on the Great Lakes for at least three decades. Four recent studies of contaminants in cormorants of the Great Lakes region are reviewed for their conformation with epidemiological criteria used to infer cause and effect linkages (CELs). Growth of the population was interrupted by DDT which led to egg-shell thinning and reduced reproduction which hid other developmental problems that were most likely caused by planar PCB, dioxin and furan contaminants. Three studies investigated embryo viability and deformities in the post-DDT era. A study of the effects of planar contaminants measured by the H4IIE bioassay technique on egg viability was judged to be strong demonstration of contaminant effects on the cormorant population. Two studies of bill defects and the co-occurrence of embryonic abnormalities with specific toxic PCB and dioxin congeners as cormorant teratogens were found to have weaknesses, but were consistent with the paradigm that planar chlorinated hydrocarbons (PHHs), especially the non-ortho-substituted PCB congeners, are now the most important toxic chemical problem for cormorants in the Great Lakes. The rapid uptake of PHHs and great interspecific variations of sensitivity to effects of these chemical between Larids and cormorants have been observed. These biochemical differences and high cormorant fecundity help account for the rapid recovery of cormorants on the Great Lakes. PHHs have relatively trivial impacts on cormorant populations, but do cause significant damage to individuals in a dose-dependant manner. Congener-specific and bioassay techniques support the development of a new toxic chemical paradigm with large implications to policy, management and water quality criteria used to make regulatory decisions. Cormorants may have actually benefited from exposure to toxic contaminants in their competition with large gulls (*Larus* spp.) on the Great Lakes in the post-DDT era.

Key words.—Chlorinated hydrocarbons, DDT, Double-crested Cormorant, embryonic abnormalities, Great Lakes, *Phalacrocorax auritus*, toxic chemicals.

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The Double-crested Cormorant (*Phalacrocorax auritus*), an enigmatic species for population ecologists, has been persecuted by humans for centuries, especially by fishermen who typically view it as a competitor. Royal edicts of Elizabeth I in England branded cormorants as pests of the crown in 1599; bounties were offered in 1603. Medieval French woodcuts show crested cormorants consorting with devils, linking the species to the underworld (Conniff 1991). Appreciation for these species came slowly even to professional resource managers, many of whom treated cormorants as nefarious pests until recently (Ludwig 1984). Altered professional and public attitudes toward cormorants, possibly due to the dramatic population crashes of related species, was coincident with widespread uses of synthetic organohalogen chemicals after 1945. The public softened its attitude toward cormorants as the concepts of biological diversity and extinction penetrated public understanding. When United States federal and state endangered species acts were passed in the 1970s, cormorant populations in North America were plummeting, possibly headed for extinction. Twenty years later, Double-crested Cormorants have staged an astounding resurgence (see Hatch 1995).

The nature of food web contamination for cormorants has changed markedly in the 20th century. The widespread use of halogenated organic chemicals in agriculture and industry had extensive impacts on many species, and pelicaniform species were identified as exceptionally sensitive (Anderson *et al.* 1969). These chemicals continue to influence the biological responses of cormorants, including biomarkers, individual responses, embryonic viability, and many subtle and poorly understood or incompletely documented phenomena (Fox 1993).In the early 1970s, wildlife biologists wondered if a breeding population of Double-crested Cormorants could persist in the Great Lakes (Weseloh *et al.* 1983). Today, the species is used as a sentinel for monitoring of certain contaminants that may have serious human health effects (Fox *et al.* 1991a,b; Gilbertson 1991). The purpose of this review is to evaluate four studies which have addressed contaminants as causes of observed population, individual and biochemical/biomarker effects in the Great Lakes Double-crested Cormorant population.

METHODS

Criteria for Assessing Contaminant Impacts

Establishing cause-effects linkages (CELs) between wildlife and contaminants beyond a reasonable doubt is a difficult and tedious process. Publication of Rachael Carson's (1962) *Silent Spring* led to two decades of study and debate. One result of her treatise and the debates it spawned was to force ecotoxicologists to formalize the criteria used to establish CELs between contaminants and the reproductive performance of populations. Some authors have been careless by reporting effects on individuals exposed to toxicants as though these were necessarily the same as effects on exposed populations (Susser 1986). Here we review four cormorant studies and assess the results of each by applying six eco-epidemiological criteria formalized by Fox (1991) and Gilbertson *et al.* (1991) for judging CELs between the effects of contaminants on individuals and populations of cormorants. These criteria comprise the following:

Consistency of the association.—Has the association been observed repeatedly by different persons, in different places, circumstances and times?

Strength of the association.—The degree to which the supposed cause and outcome coincide in their distribution, and the size of the effect produced by the presumptive cause.

Specificity of the association.—Does X lead only to Y (i.e., specificity of effect), or does only X lead to Y (i.e., specificity of cause)?

Time order.—The exposure to the putative cause of the effect(s) observed must preceed the appearance of the effects.

Coherence with other information.—The effects observed should be plausible and cohere with known biochemical mechanisms and general biological knowledge. The cause-effect interpretation of the data should not conflict seriously with generally known facts of natural history of the species being studied, nor the generally known biological mechanisms of this group of animals. Is there a relationship between dose and response?

Predictive power of the cause-effect linkage.—The purported CEL should be capable of predicting similar CEL relationships and the specific effects due to the cause in many other species which have not yet been tested.

The epidemiological criteria now in use in CEL studies underway in the Great Lakes region (for details, see Fox 1991, 1993) are leading to fundamental shifts in our thinking about toxic chemical effects. This epidemiological, weight-of-evidence approach provides the means to review experimental designs before hypotheses are tested, and a set of rules to evaluate evidence for CELs. The epidemiological criteria for determining causality and how these are weighted when establishing a CEL are reviewed in Fox (1991: Table 1). Very few studies of CELs have been done with sufficient numbers of samples, sufficient quantifications of the potential causative agents, or the correct instrumental quantification methods to permit fully adequate statistical evaluations of probabilities. Wild species are never exposed to a single contaminant. Rather, they are exposed to complex mixtures of many contaminants having diverse effects, some of which may be agonistic, antagonistic, synergistic or simply unknown (McKinney *et al.* 1985, Tillitt *et al.* 1990). Consistent application of epidemiological criteria helps to determine if a particular CEL is plausible, and may eliminate isolated observations that may suggest a spurious CEL.

A causal relationship may be recognizable whenever evidence indicates that factors are a part of a complex of circumstances which increase the probability of the occurrence of the effect, and that a lessening of one or more of these factors decreases the frequency or severity of the effects observed (Lilienfeld and Lilienfeld 1980). Making CEL conclusions from effects observed in individuals are very tenuous because two of the six criteria for evaluating CELs (consistency and strength of association) cannot be satisfied with a sample size of one. When assessing individuals, only the specificity and coherence criteria can be used to infer a CEL relationship. Unless there are many individuals that demonstrate those CEL criteria consistently in a population, caution is advisable if a CEL for the individual is claimed because few contaminants produce an effect of such specificity that this criterion will over-ride the need to satisfy most or all of the other CEL criteria.

RESULTS

Cause-Effects Associations in Studies of Great Lakes Cormorants

Four studies of the relationships of persistent synthetic halogenated organic hydrocarbons and the responses of Great Lakes' cormorants are available to evaluate the strength of CELs. These include: Case I, the

Table 1. Epidemiological criteria used to establish causation.

Criterion	Importance to interpretation	Effect on hypotheses
Strength of association	Very	Helps to confirm.
Specificity of association	Moderate	Helps to confirm, but may not be critical to acceptance.
Time order	Very	The cause must precede the effect.
Consistency on replication	Very	An hypothesis gains strength if supported by studies at different times, places, populations, species and by different investigators with many designs.
Coherence	Moderate	The CEL interpretation must not conflict seriously with known facts of natural history and the biology involved.
Predictive performance	High	An hypothesis based on a CEL should predict unknown relationships correctly.

1972-73 measurement of productivity and egg-shell thinning by the Canadian Wildlife Service (Weseloh *et al.* 1983); Case II, a study of the distribution of bill deformities in North America (Fox *et al.* 1991a); Case III, a study of egg mortalities in relation to net PHH toxicity measured by the *in vitro* H4IIE-EROD bioassay (Tillitt *et al.* (1992); and Case IV, a study of PHH contaminant congeners in cormorant eggs and co-occurrence of terata and egg death at four sites in 1988 (Yamashita *et al.* 1993).

Case I: DDT-associate egg-shell thinning and population decline.

Alarmed by a steady 20-year decline in the nesting Double-crested Cormorant population of the Canadian waters of the Great Lakes, the Canadian Wildlife Service studied productivity and egg-shell thickness in Great Lakes populations. Ludwig (1984) and Price and Weseloh (1986) have summarized the rapid decline and almost complete disappearance of cormorants from most of the Great Lakes in the DDT era. By 1973, the breeding population had decreased to roughly 125 pairs restricted to one Lake Erie and five upper Lake Huron sites. While fecundity was normal, eggs had very thin, chalky shells in most nests examined in the Great Lakes after 1963. The mean level of DDE in the eggs was 22.4 mg/kg (ppm) wet weight in 1972, and shells were >20% thinner than in the pre-1945 museum samples (Weseloh *et al.* 1983). Few eggs remained intact long enough to hatch. Productivity had decreased to 0.1 to 0.24 fledglings per breeding pair per year.

This study is instructive because it includes data assembled two decades before the advent of formal use of the epidemiological criteria. Cormorants are one example of several species which have been shown to be damaged by a particular group of chemical compounds (the DDT-DDD-DDE family) for which there are sufficiently specific effects – egg-shell thinning, reduced hatching success, followed by population decline – to be certain that the observed effects were correctly attributed to the cause. Recent models of Great Lakes waterbird populations suggest that productivity of 0.5 to 1.0 fledglings per breeding pair per year are required to maintain a stable population (Ludwig 1981, 1984). The Great Lakes' cormorant population seemed headed for certain extirpation.

Evaluation of these observational and chemistry data confirm a strong correlation of DDT-group chemicals, egg-shell thinning and the population crash. The time-order is correct and was verified by testing the quality of pre-DDT era egg-shells. Strength and specificity of the association were great, consistency with studies of other species was robust, coherence with the known mechanism

for egg-shell thinning and resultant embryo mortality was very strong. Finally, the association was strong enough to predict accurately similar biological effects in other species of this taxon (Pelicaniiformes) at the doses of DDT group chemical residues recorded in cormorant eggs. In short, all of the epidemiological criteria for a CEL are met in the context of coherence with many similar studies of birds from other taxa.

Case II: Bill defects and teratogenic reproductive toxins.

The arguments for a causal link between synthetic planar, chlorinated, hydrocarbons (PCH) as teratogens in cormorants were formulated by Fox *et al.* (1991a,b). Available continent-wide data on the distribution of chicks with deformed bills and concentrations of planar contaminants were studied. PHHs are those dioxin-like congeners of PCBs, dioxins and furans with halogen-substitution patterns that allow the molecule to assume a planar (or flattened) configuration; this allows them to bind avidly to critical intracellular receptors which initiate numerous multisite (plieotropic) responses. The most characteristic action is Ah+ receptor-dependent P-450 enzyme induction of Aryl Hydrocarbon Hydroxylase (AHH) and Ethyoxy-Resorufin-O-Deethylase (EROD) and related enzymes in a dose dependant fashion. EROD induction by all planar contaminants in mixtures may be measured as TCDD-equivalents (TCDD-EQ) with the *in vitro* H4IIE rat hepatoma cell bioassay (Safe 1990, Tillitt *et al.* 1990). The induction of embryonic deformities is another characteristic of these PHHs (Marks *et al.* 1981, Wardell *et al.* 1982). TCDD and planar PCBs produce typical craniofacial deformities in laboratory mammals (Pratt *et al.* 1984), and bill defects in birds (Verrett 1976, Brunstrom and Andersson 1988). The hypothesis explored by Fox *et al.* (1991b) was that these PHHs were involved causally in the appearance and distribution of bill-deformities in cormorants on the Great Lakes.

Geographic variation in deformity rates was significant ($P<0.05$) with the highest rates co-occurring with the highest reported concentrations of TCDD-EQ in cormorant eggs (Tillitt *et al.* 1991). However, there were too few bioassay results available to test the statistical significance and variance in those data. What data were available was based on pooled samples rather than individual eggs so that toxic doses to individuals and the variance of individual responses was unknown. Time order was indeterminant since no population-wide data on the natural occurrence of bill deformities prior to the widespread use of planar substances was presented. Consistency with other field studies (e.g., Hoffman *et al.* 1987, Kubiak *et al.* 1989) and numerous laboratory studies of contaminant mixtures (Tumasonis *et al.* 1973, Rifkind *et al.* 1984) and individual planar substances (Brunstrom 1988, Brunstrom and Andersson 1988) was great. Specificity of the observed effects matched the known effects of planar compounds very well (Safe 1990). Although there are other substances and exposures that could produce *some* of the effects observed, only the PHHs are known to produce the entire suite of effects observed. Recently, this consistent grouping of effects has been termed the GLEMEDs syndrome (Great Lakes Embryo Mortality, Edema and Deformities syndrome) by Gilbertson *et al.* (1991). Consistency upon replication was great since different investigators in different years reported the same high rates of bill defects from more contaminated areas (e.g., Green Bay) and the same lesser rates from areas of lesser contamination in cormorants and other species (Hoffman *et al.* 1987; Kubiak *et al.* 1989; Yamashita *et al.* 1993; J. P. Ludwig unpubl. data). Coherence with known biological mechanisms and theory was excellent. Overall, a convincing CEL is presented which is only slightly less robust than that for DDT and egg-shell thinning. The principal limitations to acceptance of the CEL hypothesis were the paucity of measurements of the concentrations of TCDD-EQs and rates of bill deformities for other similarly exposed species, and the indeterminant time-order criterion.

Case III: TCDD-EQ in cormorant eggs correlated with embryo mortality.

Much speculation has resulted from the widespread appearance of deformities and reduced waterbird embryo survival in the last 20 years (e.g., Ludwig and Kurita 1988), following the first systematic observations reported for Herring Gulls (*Larus argentatus*) and other Lake Ontario waterbirds in the late 1960s and early 1970s (Gilbertson and Hale 1974, Gilbertson *et al.* 1976, Gilbertson and Fox 1977). Waterbird colonies persist in areas with greater residual concentrations of halogenated contaminants, but these colonies usually have depressed hatching and elevated deformity rates (Kubiak *et al.* 1989; Ludwig *et al.* 1993a,b; Yamashita *et al.* 1993). These relationships were explored by relating the dose-response of H4IIE EROD bioassay-measured TCDD-EQ in cormorant eggs to hatchability (Tillett *et al.* 1991, 1992). Toxic chemical exposures are but one of many factors known to influence egg hatchability. Even so, embryonic death and deformities are reported to occur frequently in the presence of toxic chemicals. TCDD-EQs were measured by the H4IIE *in vitro* bioassay in 41 egg sample pools from 22 waterbird colonies and five species in the upper Great Lakes (Tillitt *et al.* 1991). Concentrations of extraction-corrected TCDD-EQ ranged from 100.4 to 839.7 with a mean of 409 pg/g. The lethal dose for 50 percent of white leghorn chicken eggs (LD50) for TCDD was established by egg injections to be 147 pg/g (Verrett 1976) and the LD95 value is 1,000 pg/g (Higgenbotham *et al.* 1968). Brunstrom and Danerud (1983) and Brunstrom and Andersson (1988) found LD 95s of the much more abundant PCB congeners 126 and 77, of 8,000 and 22,000 pg/g, respectively. Since these much more environmentally-abundant planar PCB compounds act through the same toxic mechanism as TCDD, albeit with lesser potencies, there was ample reason to suspect that planar compounds were a major factor in reduced egg hatchability at most, if not all, affected Great Lakes waterbird colonies.

A cooperative study by three independent research groups which used the same field and egg collection protocols for measuring egg hatchability was conducted between 1987 and 1989. They found a very strong correlation and high statistical significance (r^2 = 0.703, P=0.0003) of TCDD-EQ with egg hatchability, but a much poorer correlation (r^2 = 0.319, P=0.045) with concentrations of total PCBs. They also found that the PCB mixture extracted from cormorant eggs had a much greater toxic potency than PCB in parent Aroclor™ mixtures indicating selective enrichment of the most toxic congeners, probably owing to environmental weathering, biomagnification and metabolism. The study included one less contaminated colony outside of the Great Lakes basin as an external control and to expand the spectrum of dose-response. This design was a clever means to measure egg hatchability at very low levels of planar contaminants which partially addressed the time-order criterion for a Great Lakes CEL since the vast majority of the TCDD-EQs in waterbird eggs resulted from PCBs (Tillitt *et al.* 1991). PCBs entered commerce in quantity only after 1940. The range of observed hatchabilities was from 92% to 58% of eggs laid across a TCDD-EQ range of 35 to 344 pg/g. The dose-response curve was linear above 150 TCDD-EQs, which meets the strength of association and coherence criteria.

The association of TCDD-EQs with egg hatchability must be judged to be exceptionally strong. Statistical significance was robust, time order was partially addressed, the measured dose:response supporting strength of association was strong. Specificity of the cause was high, consistency among three independent field research teams was good, as was the consistency with the literature (Gilbertson *et al.* 1991). Controlled laboratory studies by egg injections of TCDD (Verrett 1976), the planar PCBs (Brunstrom 1988, Brunstrom and Andersson 1988) and even PCB feeding experiments using parent Aroclor mixtures (Tumasonis *et al.* 1973) all had produced similar results. This uniform coherence strongly supports causality.

Case IV: Investigations of relative contributions of the planar PCB, dioxin and furan congeners on egg viability and deformity rates.

This study by Yamashita *et al.* (1993) measured the presence of six dioxin, six furan and seven planar PCB congeners in the eggs of cormorants from four of the same colonies studied by Tillitt *et al.* (1991, 1992); samples of Caspian Tern (*Sterna caspia*) eggs were also examined from colonies adjacent to two of the cormorant colonies chosen for study. Duplicate collections were made in colonies known to exhibit a three-fold range in TCDD-EQ concentrations in their eggs from 1986 and 1987 year samples (Tillett *et al.* 1991). Egg hatchability and embryonic deformity/abnormality rates were measured in the field by marking over 100 nests in each test colony and opening 100 eggs after 12 and 23 days of incubation to identify viable, dead, and abnormal embryos.

Large differences in concentrations of the coplanar PCB congeners 77, 126 and 169 between the Lake Superior and Green Bay eggs, and intermediate concentrations in the eggs from elsewhere in Lake Michigan and Lake Huron were reported (Yamashita *et al.* 1993). These concentrations were calculated as TCDD-EQs using the published relative potencies to TCDD (i.e., toxic equivalency factors) for each congener measured. Further, they found a large difference in the concentrations of coplanar PCB 77 in the eggs of terns and cormorants from colonies on the same islands, which indicates that cormorants have a more effective metabolism or capacity to excrete this very toxic congener than do Caspian Terns. Increased rates of abnormal embryos were found in eggs that carried higher calculated TCDD-EQ in a linear dose-dependant fashion. The same types of craniofacial abnormalities reported by Fox *et al.* (1991b) and Pratt *et al.* (1984), and the edema conditions cited by Gilbertson *et al.* (1991) as evidence of the GLEMED syndrome, were found in both species studied.

Although this study was consistent with the other studies linking specific PHH compounds to the GLEMED syndrome and particular deformities, the essential weakness was the small sample size, only four colonies studied for one year. The study did not address time-order or include an off-Great Lakes colony to provide a greater dose-response range. Statistical significance was weak. However, specificity was great, the strength of the putative association was high, and the results agree very strongly with statistically more robust studies. Further, this study confirmed the relatively small role of TCDD, other dioxins, and the dibenzofurans relative to the planar PCBs in the embryonic toxicology and deformity problems that many authors have reported or speculated about in other freshwater and marine ecosystems (e.g., Tanabe *et al.* 1987, Ludwig and Kurita 1988, Kubiak *et al.* 1989, Smith *et al.* 1990).

The Caspian Tern chemical residue data reported for the Saginaw Bay colony in Yamashita *et al.* (1993) was investigated further by Ludwig *et al.* (1993a). Ludwig *et al.* (op. cit) made a strong case for the planar PCBs contributing most of the toxic load in the eggs of that species from Saginaw Bay. Their conclusion, based on a six-year study of productivity of Saginaw Bay terns, adds weight to the coherence criterion and validity of the CEL of planar contaminants to egg viability and deformity problems in Great Lakes cormorants. This interpretation gives validation through coherence with responses of another species with a different metabolic capacity within the same Great Lakes basin ecosystem.

DISCUSSION

The strengths and weaknesses of the four case studies are summarized in Table 2. By themselves, none of these studies can stand alone as an unequivocal demonstration of a CEL with the exception of the Tillitt *et al.* (1992) study of egg mortality, which fulfills all of the CEL criteria adequately. But even this study fails to fulfill Koch's fourth postu-

Table 2. Evaluation of the strength of four potential Cause-Effects Linkages of Great Lakes cormorant responses to toxic chemical contaminants.

Criterion	Potential Cause-Effects Linkage			
	DDT compounds and shell thinning	Bill deformities and H4IIE bioassay	Egg hatching and H4IIE bioassay	Planar compounds and abnormal embryos
Strength of association	++	++	+++	-
Specificity of association	+++	+	++	-
Time order	+++	0	-	0
Consistency	++	++	+++	--
Coherence	+++	+++	+++	--
Predictive power	+++	++	+++	--

+++ = very strong support.
++ = strong support.
+ = weak support.
0 = not addressed in study design.
- = negative support, invalidates hypothesis.

late since the putative cause (PHHs) were not introduced into clean cormorant eggs to elicit the effect (such a study is now in the planning stage). However, taken together with other reports reviewed in Gilbertson *et al.* (1991), these four studies of cormorants allow researchers to make several significant advances. First, after nearly two decades of field monitoring studies it was clear that DDT had impacted many avian populations through egg-shell thinning. However, very little more could be stated as a CEL for the effects of toxicants on populations of waterbirds with any degree of confidence. The impacts of other synthetic organohalogens on wildlife populations were confounded by the presence of DDT, the complexity of contaminant mixtures, and a lack of analytical means to separate these factors from each other. The great advance of these combined field and laboratory studies, made possible by the biochemical work of McKinney *et al.* (1985), Rifkind *et al.* (1984), and summarized by Safe (1990), has been the development of tools that measure the effects of complex mixtures which result in a plausible and understandable toxicity paradigm (Tillitt *et al.* 1990).

Second, two innovative techniques, the *in vitro* H4IIE bioassay for measuring TCDD-EQ of mixtures directly in a living system (Tillitt *et al.* 1990), and the additive model of planar halogenated congeners calculated as the sum of congener equivalents to actual TCDD (Kannan *et al.* 1988, Safe 1990), are useful new tools which allow advances in our understanding of PHH toxicity to wildlife. By considering the three more recent studies which used these techniques under the six rules for judging the validity of CELs, such studies combine with older literatures to support the new paradigm that planar halogenated hydrocarbon (PHHs) contaminants are now the most important toxic chemicals faced by cormorants and probably other Great Lakes species. This is a paradigm shift of huge significance. The new paradigm is especially important to other details, such as the relatively small importance of actual TCDD, the phenomenon of selective bioaccumulation of the most toxic of the planar PCB contaminants in waterbirds, and the general disappearance of DDT as a problem.

There are regulatory large implications of this new information. For example, knowing the actual concentrations of individual congeners and how these interact with other compounds in mixtures will support water quality criteria developed from real-world field data for wildlife. Instead of using criteria modeled from imprecise total contaminants toxicity data that was produced in laboratories with domestic test species and unweathered parent mixtures which do not

account for selective bioaccumulation or metabolic differences, more realistic criteria are now possible (Ludwig *et al.* 1993b).

Another implication is that influences of PHHs will be present in the waterbirds for many years. Avian field biologists should understand that toxic PHH burdens will continue to be a confounding factor for almost all reproductive studies of colonial waterbirds for the forseeable future, especially on the Great Lakes. Tanabe *et al.* (1987) showed that the planar halogenated contaminants, especially the non-ortho-substituted PCBs, are a worldwide problem of aquatic ecosystems and a potential threat to fish-eating species of all taxa. Contamination and the physiological sensitivity of each exposed species and population are large confounding factors to population and ecosystem studies that seek to understand the interactions between populations by studies of classic habitat parameters only. The value of employing the new bioassay approaches and CEL criteria lies in their rigor and capacity to discriminate toxic chemicals in complex mixtures in a defensible and uniform manner. When used rigorously, those researchers who incorporate the CEL criteria into their studies will reassure peers of the degree of contamination problems affecting the populations they study, thereby eliminating a major confounding factor to classic habitat-based studies.

Finally, it is clear from this special publication (Nettleship and Duffy 1995) that Great Lakes Double-crested Cormorant populations are only slightly influenced by contaminants (see Weseloh *et al.* 1995). The exceptional fecundity and reproductive capacity of undisturbed cormorants has overridden the damage from toxic chemicals to individuals in this population. Colonies in some more contaminated areas are likely less productive than those from cleaner areas, but all this has served to do is lessen the magnitude of the population explosion in this species. The Double-crested Cormorant is uniquely positioned to exploit food reserves unavailable to its competitors in the Great Lakes (Ludwig *et al.* 1989). In fact, because cormorants have been shown to have both greater access to available forage fish (op. cit.) and a more effective metabolic detoxification capability than smaller larids for certain highly toxic planar contaminants, especially the planar PCBs with four or fewer chlorinations (Yamashita *et al.* 1993), cormorants may have significant competitive advantages over the other species with which they compete for nest sites in these contaminated Great Lakes ecosystems.

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