

## Ecotoxicological Effects in Ecosystems: Wildlife Indicators of Environmental Contamination

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### Abstract

Conventional thinkers tend to consider the impacts of toxic substances on a species basis by measuring changes in a species' abundance following ecosystem contamination. However, when ecosystems become contaminated, effects usually ripple through the fabric of the entire community, as natural assemblages of species are altered substantially. Contaminants often interact with other factors; causality for the observed change is rarely ever simple, nor readily understood. One hypothesis often embraced in the absence of data for verification is that habitat loss is the essential driving force for ecosystem damage. New ecotoxicological data show changes to some wildlife populations after contamination that cannot be correlated to habitat changes. Often, these changes relate to the flow of contaminants through the resident species by the food web, which damages wildlife populations according to their sensitivities to contaminant exposures rather than losses of habitat or other conventionally understood stresses. Contaminant effects on the developmental processes of wildlife (and man) often project serious effects into subsequent generations from the generation which was exposed. The invasion of non-native species can complicate understandings of the often subtle role(s) of contaminants in ecosystems by changing the flows of energy and contaminants through the ecosystem's food web in unpredictable ways.

The recent development of techniques that reliably measure the biological effects of contaminant mixtures with an emphasis on the Great Lakes experience, and freshwater invertebrates is reviewed. A paradigm useful to isolate the effects of contaminants from habitat changes and exotic species invasions and a means to assess the importance of each factor in the larger ecosystem context will be suggested.

### Introduction

Over more than 40 years, wildlife has been shown to track the presence of releases of metals and synthetic chemicals with a high degree of accuracy by the responses of their exposed populations. One of the earliest sets of observations of DDT effects in wild birds was made by Charles Broley who followed the decimation of bald eagles in Florida owing to DDT-induced eggshell thinning in the late 1940s (cited in Colborn et al. 1996). Similarly, between 1957 and 1963 George Wallace at Michigan State University recorded the nearly complete extinction of robins (*Turdus migratorius*) on the campus of Michigan State University following the heavy use of DDT to control the Japanese beetle that spread the much dreaded Dutch Elm Disease. We not can state with considerable assurance that Rachael Carson's *Silent Spring* – published 33 years ago – brought the issue of chemicals “out of the closet” for our society with her remarkably accurate conjectures on the effects of chlorinated pesticides on wildlife. Because wildlife has proven to be such an effective and accurate measure of the presence of many toxins, we have chosen wild species to illustrate these effects.

After *Silent Spring*, ecotoxicology underwent a period of relative stagnation for almost two decades. The controversies created by this milestone indictment of the agricultural chemical industry created enormous controversy, generating more heat than actual understanding. The industrial and environmental communities soon came to share polar opposite view on the importance of Carson's conjectures. Regulatory groups and industry began a period of testing the toxicity of each chemical in

commerce for its carcinogenicity because a 1958 law (The Delaney clause) had focused governmental attention of the government on carcinogens in foods. Chemical detection methods were primitive, and few bioassays were developed or understood for their potent value of assessing effects in living creatures. All of these barriers to knowledge were yielding to research over the next three decades. Analysts were steadily pushing the limits of detection lower. Where chemists could only detect and discriminate chlorinated compounds reliably in the parts per million range ( $10^{-6}$ ) concentrations in the mid-1960s, by 1995 toxic chemicals were measured at environmental concentrations of  $10^{-16}$  to  $10^{-18}$  with the semi-permeable membrane sampling device (Lebo et al. 1995). By 1980, environmental scientists began close collaborations with biochemists who understood the potent discrimination and mensuration capabilities of bioassays to detect adjustments by an exposed animal to toxicants (Giesy and Grany 1989, Tillitt et al. 1990). Evidence was accumulating that many effects in wildlife chronically exposed to toxic chemicals were subtle, intergenerational, and even sometimes confounded by chemicals and their breakdown products producing agonistic or antagonistic effects when found in complex mixtures (Bannister, et al. 1987, Colborn et al. 1993).

The synthetic science of ecotoxicology has begun to mature with the development of these new techniques of risk assessment, especially with capabilities to measure the effects of environmental mixtures (Giesy et al. 1994a, 1994b). The basis phenomena of bioaccumulation of toxic substances followed by transfers up a food chain and bioconcentration over time in the upper levels of the food web have been understood for over three decades (Rudd 1964). Recent work and modeling have greatly refined our understandings of mechanisms of chemical movements in the environment (e.g. Clark et al. 1988). The rapid development of risk assessment has added powerful new tools to our abilities to understand the behavior and long term significance of toxic chemicals. The data that were necessary to address the endocrine-disruptor and multi-generational effects of chronic chemical exposures on populations are now appearing rapidly in the scientific literature (Mora et al. 1993; Colborn and Clement 1993; Colborn et al. 1993, 1996; Hose and Guillette 1995).

These new data have fostered new understandings of the effects of chemicals on populations, and created much controversy as old and comfortable (if ineffective) paradigms of a generations of scientists and regulators were challenged. Many scientists have been unable to assimilate these data (Cooper 1995), and even the odd career was shattered by the new information (Gilbertson and Schneider 1993). As with all emerging science, there are vigorous defenders of the old paradigms, and champions of the new, providing a rich habitat for vitriolic controversy (Cooper 1995, Ludwig 1996)! The regulatory paradigm that only human cancer should be the criterion for evaluating toxicity has suffered the most serious criticism. Multiple endpoints for quantification of damage are slowly emerging as indicators of toxicity. Cancer as the driving force in regulation is being replaced with a broader view of the effects of chemicals: development, endocrine disruption, wildlife damage, and ecological community disruptions are finally receiving attention as valid measures of effects (Ludwig et al. 1994; Giesy et al. 1994a, 1994b; Hose and Guillette 1995; Fox 1995; Ludwig 1996).

Ecology is the study of the factors and interactions among the biota that affect distribution and abundance of living species and their (communities) associations. It is very curious how many scientists and ecologists have failed to pick up on the possible influences of toxic chemicals on the ecosystems and communities they study. Many professional ecosystem and community ecologists have focused their research exclusively on the natural environment. Often, they appear to be blissfully unaware of the magnitude and distribution of contamination in the communities of organisms they study. It is a safe generalization that synthetic chlorinated organic compounds have penetrated every ecosystem and watershed in the world. The speed of dispersal of these substances from their places of manufacture and use to the most remote areas is nothing less than astonishing and emphasizes the dynamic nature of world weather processes that mix the troposphere (Iwata et al. 1995, Muir et al. 1995, Hites 1995, Jones et al. 1996). The senior author reviewed abstracts of all the papers published in the journals of the Ecological

Society of America during 1994 and found barely 4% of the published papers had even considered the possibility that synthetic toxic chemicals could have influenced their results; less than 2% addressed the possibility seriously (A recent forum in *Ecological Applications* 5: 291-310 has partly remedied this deficiency). What do these substances mean to the resident biota? Can we do anything about it? These are potent queries that few professional ecologists deign to consider. Possibly because the effects of these substances are difficult to quantify or recognize, especially when diluted in watersheds and large communities. These substances and their effects are an intractable variable that defies precise quantitation. This paper explores our views of the broad effects and principles that govern the spread into large watersheds and ecosystems and the effects of these exposures on ecological communities. Our questions are “What kinds of organisms and species will be affected predictably by individual and population level changes effected through exposures to these substances?” and “What are the patterns of damage to ecological communities that accompany the widespread release of these substances into meso-scale watersheds?”

## Methods

We review a series of peer-reviewed published papers here employing a new combination of evaluation criteria suggested by an old principle of ecosystem contamination: To wit, contaminants are generally moved from organism to organism by direct uptake as an equilibrium phenomenon from the environment (bioaccumulation) and typically increase in concentration through food chain transfers (bioconcentration). Every basic ecology text described the movement of energy through communities and the general principle that 90% of the energy in a lower trophic level is lost with each food chain transfer to the next trophic level (Odum 1971, Rudd 1964). Many toxicants, including the synthetic organochlorine contaminants, are conserved as the energy is metabolized by the consumer leaving much greater chemical concentrations in the animals in each higher trophic level. Contaminants that tend to accumulate in the lipids, or other specific tissues (e.g., cadmium in kidneys), increase with each food chain transfer. The more transfers involved, the higher the concentrations found in the animal. Species at the top of the food web, such as mink (*Mustela vison*) or bald eagles (*Haliaeetus leucocephalus*), have been reported to accumulate up to 25 million-fold higher concentrations than are found in the waters that they depend upon for food (Clark et al. 1988, Bowerman 1993, Bowerman et al. 1995). Thus, it is the movement of energy through food webs that defines the first unifying principle of wildlife toxicology:

The higher an organism is in the trophic structure of a community, the more likely it is to receive high exposures to toxicants leaked, discharged, or atmospherically moved into the system.

In recent years, a great deal of work has been directed toward understanding how sensitivity to contaminants varies among species, and how this variation translates to ecosystem-wide effects. Two laws of toxicology are:

The dose makes the poison, and its axiom that the sublethal dose determines the magnitude of the response. And, there is a threshold concentration of every toxin which begins to produce effects in organisms; concentrations below this threshold will have not effect.

Parallel to these laws of toxicology are two ecological laws that state the same basic concept in a slightly different way (Odum 1971). Leibig’s law of the minimum states: There is a minimum concentration of each substance required for life. And, Shelford’s law of tolerance states:

Every necessary condition or substance that varies in concentration in the habitat of an organism has a minimum and maximum value which, when exceeded, extinguishes the

population in that habitat. The organism tolerates and adjusts to the variation of each condition or substance so long as it remains within the range of values or concentrations it can tolerate. An axiom to this law is that adjusting to any stress requires an expense of energy and diversion of resources from other biological functions, the most critical and sensitive of which is reproduction.

To these classical laws we add one more concept from population genetics:

Each species (in a community) has a unique, genetically-determined range of tolerances for conditions and substances that defines the boundaries of its niche in the ecosystem where it lives.

Ecotoxicology is a synthetic science that harmonizes these laws of classical sciences into a single paradigm in order to explain widespread effects of chemicals in the environment; it permits one to make risk assessments. Synthetic toxic substances, and heavy metals liberated by human activities, may be correctly categorized under these concepts as substances with the power to push individuals and populations beyond their tolerances. Effects of toxicants are detected when individuals and populations adjust their responses in a characteristic manner, predetermined by their genetic capabilities to adjust to the stress at particular doses (exposures) in the environment. At chronic exposures, adjustment to the toxin is often detected by measuring an associated effect or biological marker (Fox 1993). Acutely toxic doses are rarely seen in the environment now, except when spills occur.

### **Dioxin-like Chemicals in The Great Lakes Watershed – A Detective Story**

Many years of research identified the 2, 3, 7, 8-tetrachlorodibenzo-p-dioxin congener (hereafter referred to TCDD) as a particularly toxic chemical in a family of 75 dioxin congeners. TCDD was found to have potent capabilities to induce a pleiotropic cascade of cellular and organismal toxic response (McFarland 1985). Further, TCDD was found to bind to DNA at particular locations via a specific aryl hydrocarbon hydroxylase receptor (Ah-r) that alters gene transcription to RNA and gene expression. The cellular and subcellular changes induced in the presence of TCDD occur in all vertebrate species, with varying intensities according to the genetic make-up of each species. Although all species react to TCDD, the sensitivity of the response was found to vary by about 10,000-fold for adults and 100-fold for developing embryos among exposed species (Devito and Birnbaum 1994, Theobald and Peterson 1994, Schecter, 1994). TCDD is a diaromatic dioxin congener in a planar configuration. Other groups of chlorinated compounds mimic TCDD, especially the planar congeners of PCBs, furans, and naphthalenes. All of these compounds and congeners of at least 10 other chlorinated chemical groups found in the environment also can interact with the Ah-r, and induce TCDD-like effects (Giesy et al. 1994a). All of these chemical groups have been found in the wildlife of the Great Lakes environment, as have many other pesticidal and industrial chemicals.

A potent confounding factor in determining causes for the damage seen in wildlife has been the complexity of environmental mixtures (Gilbertson et al. 1991; Giesy et al. 1994a, 1994b). Environmental mixtures are strongly influenced by historic and present local discharges along with a host of other ecological and biochemical factors including the availability of contaminated food (Giesy et al. 1995a., Bowerman et al. 1995), the metabolic capacities to detoxify contaminants of the exposed species population (Brunstrom and Andersson 1988, Yamashita et al. 1993), and their genetic sensitivity (Devito and Birnbaum 1994). In spite of this bewildering complexity, it was apparent by 1990 that the TCDD-like chemicals in the Great Lakes behaved in an additive manner with their direct toxic potency a function of their individual concentrations and their toxic equivalencies to TCDD. Using the new H411E cell bioassay (Tillitt et al. 1990) and toxic equivalency factor approaches (Brunstrom and Andersson 1988, Safer 1990, Ahlborg et al. 1994), several groups of scientists were able to show how the TCDD-like

toxicity was distributed across the Great Lakes by egg or blood samples that measured exposures of the colonial fish-eating waterbirds (Tillitt et al. 1991b, 1992; Mora et al. 1993; Yamashita et al. 1993; Giesy et al. 1994a, 1994b), bald eagles (Bowerman 1993; Giesy et al. 1994c, 1994d, 1995; Bowerman et al. 1995), and mink (Heaton 1995, Giesy et al. 1995, Tillitt et al. 1996). Not surprisingly, wildlife effects were strongly correlated to location and local chemical discharge history. Different species from the same locality had different responses to the same exposures owing to metabolic differences and genetic capabilities (Yamashita et al. 1993).

The available data on TCDD-like toxicity was integrated by Giesy et al. (1994a, 1994b). They found the lowest adverse effect level for the most sensitive species to be between 7 and 10 parts per trillion wet weight for actual TCDD, or for mixtures of TCDD-like chemicals with an equivalent toxicity, or TCDD-Eqs (Cheung et al. 1981, Brunstrom and Andersson 1988, Henschell 1993). The most sensitive avian species is the white leghorn chicken (*Gallus gallus*). White leghorns begin to show embryonic malformations of the heart and brain at levels in the 7-10 ppt range, have LD50s in the range of 110-150 ppt ww (Henschell 1993, Henschell et al. 1995), and an LD95 near 1,000 ppt ww (Brunstrom and Andersson 1988). The least sensitive avian species seems to be the pheasant, with an embryonic LD50 near 2,200 ppt ww. Thus, there is about a 100-fold variation in the interspecies developmental sensitivity to TCDD and TCDD-EQs.

### **An Ecological Synthesis of Great Lakes Toxic Chemical Findings**

Applying the rules for determining causality of TCDD-like toxicity in wildlife (Fox 1991, Gilbertson and Schneider 1993) has enabled many investigators to make great progress toward the goal of understanding how chemicals affect the assemblages of species in the Great Lakes that constitute the larger ecological community (Tillitt et al. 1992; Leatherland 1993; Struger et al. 1994; Ludwig et al. 1993a, 1993b, 1995; Fox 1995). Ludwig et al. (1993b) showed that a group of five fish-eating birds and mink had a 31-fold variation in their lowest observed adverse effect level from PCBs on their reproduction. Water concentrations of PCBs and TCDD-Equivalents in the Great Lakes were far above those required to provide a habitat free of TCDD-like toxicant effects in wildlife when bioconcentration and bioaccumulation through the food web were considered. The general conclusion that wildlife guilds (e.g., the secondary fish predators, the tertiary predators) were affected and damaged as a group in the same general fashion, but with different sensitivities was strongly supported by these diverse studies. Very significantly, because the studies involved species from different taxa and four vertebrate classes with very different specific metabolic capabilities but similar responses to TCDD-like chemicals, there was extremely strong support for this generalization. This satisfied one of most critical criteria for verifying cause-effect linkages: there is consistency on replication among many species from all tested taxa in the secondary and tertiary predator trophic levels in the ecosystem (Fox 1991, Leatherland 1993). This is very strong evidence of a common toxic chemical cause that is unrelated to genetics, disease, or a habitat factor that might lead to mortality of one or a few species but not many species from different taxa.

Bowerman's various long-term evaluations of bald eagles (Bowerman 1993, Bowerman et al. 1995) and the development of a mesoscale evaluation of where the contaminants located specifically within the Great Lakes watershed (Giesy et al. 1994c, 1994d) were shown to cause different effects in a dose-response manner. Tillitt et al. 1992, Ludwig et al. 1996 has added further confirmation. Toxic chemicals are the cause of the widespread decline and functional impairments to many fish-eating species in the Great Lakes. These researchers found the TCDD-like toxic contaminants in fish taken above dams on three large Michigan rivers tributary to the Great Lakes were 10- to 25-fold lower than in the same species of fish below the dams that were free to move in and out of Great Lakes waters. Lower reproductive rates measured by egg hatching and survival of chicks to fledgling and very elevated blood plasma levels of PCBs in eagles feeding on fish from Great Lakes-influenced territories were reported

(Bowerman et al. 1995, Giesy et al. 1995). For many eagle pairs that establish territories along the Great Lakes shorelines, the turnover from mortality of mates in their territories appears to be 1.5- to 2.5-fold greater than in the inland territories. The absence of mink on these rivers below the dams was also noted during field studies, and related to the presence of elevated PCBs and TCDD-Equivalents in the fish from the below-barrier dam reaches of these rivers (Giesy et al. 1994d.). Similar distributions were reported on mink and otter (*Lutra canadensis*) along Lakes Erie and Ontario tributaries for more than 20 years: only young mink were found near the Great Lakes shorelines, and little or no reproduction was documented in the townships bordering on the lakes (Wren 1991). In a recent study on mink fed diets including carp from the Saginaw River, serious reproductive damage was found with as little as 10% of carp in mink diets. The low effect level was estimated to be 2% fish from this source (Tillitt et al. 1996), and 3% to 8% of fish from the Great Lake reaches of three other Michigan rivers (Giesy et al. 1994c).

For these tertiary predator fish-eating species, contaminants appear to have a potent influence on their reproduction, adult survival, and capacity to occupy historic habitat with reproducing age individuals. In the case of the mink, all age classes were affected, with reproduction reduced to nil where the Great Lakes contaminated fish are eaten. Eagles maintain some reproduction in their Great Lakes territories but lose many more adults and raise fewer young when they eat the PCB- and TCDD-Equivalents-contaminated Great Lakes fish.

Caspian terns (*Hydroprogne caspia*) and double-crested cormorants (*Phalacrocorax auritus*) have received much attention in the past decade from ecotoxicologists owing to the appearance of many deformed chicks in their populations (Ludwig and Kurita 1988, Fox et al. 1991, Ludwig et al. 1996), and reduced reproduction in Great Lakes localities that are highly contaminated with TCDD-like chemicals, especially Green Bay and Saginaw Bay (Yamashita et al. 1993). The Saginaw Bay tern colonies were studied intensively for six years (1986-1991). Reproduction failed totally following the disturbance of contaminated Saginaw River sediments by a 100-year flood, and did not return to normal until four years later (Ludwig 1993a). Deformed tern chicks were ten-fold more frequently found in Saginaw Bay than the cleaner reference colonies of the Canadian North Channel and Georgian Bay districts. In studies of the viability of cormorant eggs and distribution of deformed chicks in the upper Great Lakes, very robust and highly significant correlations of egg death and deformities were found with embryo exposures measured as the concentrations of TCDD-Equivalents in eggs (Tillitt et al. 1992, Ludwig et al. 1996).

By the time dioxin and PCB contamination yielded to regulatory controls in the Great Lakes region during the early 1970s, large variations in contaminant burdens were established across the breeding range of Caspian terns. After 1950, the birds' U.S. Colonies, especially those in Green Bay and Saginaw Bay, were rapidly contaminated with TCDD-like chemicals. At the same time contamination was increasing rapidly, a new food source in the form of alewives (*Alosa pseudoharengus*) invaded the Great Lakes establishing an immense new food base that gulls and terns exploited (Ludwig 1968). The migratory ring-billed gull (*Larus delawarensis*) and Caspian Tern populations grew rapidly after 1960, although the year-round resident herring gull population did not (Ludwig 1974). Beginning in 1926, Caspian terns were banded in Great Lakes colonies. Banding efforts included all the upper Great Lakes after 1959 and one Lake Ontario colony that were the traditional sites of nesting. Significant numbers of tern fledglings from each geographic grouping were banded ever decade after 1960. Starting in 1966 and continuing through 1992, a program of adult trapping to recapture thousands of terns in their breeding colonies was implemented to assess population structure and movements between colonies (Ludwig 1968, 1979). By 1970, approximately 26% of nesting adults were banded in the Great Lakes population, and 21% of the birds caught in 1990 and 1992 were banded. This is likely the most thoroughly tagged population of known-age and place-of origin wild birds in North America.

Trapping and census work in 1966-67 revealed the nesting population was evenly divided between the Canadian and U.S. colonies as contamination was increasing; 54% of breeding age terns

originated in cleaner Canadian and U.S. colonies as contamination was increasing, 54% of breeding age terns originated in cleaner Canadian source areas (Ludwig 1968). In the next 11 years, the population grew from 2,800 nesting pairs to 3,597, but the proportion of breeding age terns in the whole population that had been raised in the more contaminated U.S. colonies eroded rapidly. By 1978, 73% of the banded terns nesting in the U.S. colonies had been raised in the cleaner Canadian sites (Ludwig 1979), but 97% of the terns nesting in Canada were raised in Canada. After 1986, the nesting numbers in Canadian colonies stabilized or slowly declined as local populations of alewives became scarce, but the U.S. colonies continued to grow, probably because terns were attracted to eat abundant alewives. Adult trapping in 1986, 1990 and 1992 continued to show heavy recruitment of birds raised in the Canadian colonies; less than 2% of birds nesting in the Canadian sites were raised at U.S. colonies in this six-year period, but close to half of the U.S. nesting birds were raised in Canada. L'Arrivee and Blokpoel (1988) analyzed all banding data available on Caspian terns from 1926 to 1986. Because few birds are found dead in their colonies, more than 98% of the in-colony recovery records had come from the adult recapture programs. Of 22,745 terns banded as chicks, 621 had been recaptured as known age and place of origin breeding birds in this population between 1966 and 1986. If the probability of survival to breeding age was identical from all colonies, then the birds should have returned at the same rate to all sites, and recruitment should have been at the same rate. However, clearly the Canadian colonies provided proportionally much greater numbers of reproductively competent adults than did the U.S. colonies ( $P < 0.001$ ), and the most contaminated U.S. area of Green Bay contributed breeding birds to the Great Lakes population at less than half the rate of the Canadian colonies.

Paradoxically, a review of the fledging rates for colonies shows the rates of chick fledging were significantly higher ( $P < 0.004$ ) in the U.S. colonies between 1962 and 1982 (U.S. colonies' mean of 21 colony years = 1.24 chicks/pair, range 0.46 – 1.93; Canadian colonies mean of 15 colony years 0.98 chicks per pair, range 0.59 – 1.66). On the surface, these data sets seem incompatible: a lower fledging rate from Canadian colonies, but higher adult recruitment three to five years later at maturity. However, to conclude that this was true, survival from the time of fledging to the time of first nesting would have to be equal for the U.S. – and Canadian-raised birds. Recently, a possible explanation for this situation emerged from work on immunocompetence and contaminants. The immune responses of terns raised in the highly contaminated Green Bay and Saginaw Bay colonies of the upper Great Lakes to challenges by antigens and T-cell stimulators that mimic disease agents were 30 to 50% lower than the immune responses of Canadian source chicks in 1992 – 94 year tests. This occurred in spite of the fact that the Canadian colony chicks grew more slowly, had higher helminth parasite loads, and appeared to be fed less often (Grasman 1995, Grasman et al. 1996) than those from the U.S. sites. TCDD-like contaminant burdens were 2- to 3-fold greater in the eggs and chicks from the more contaminated U.S. sites.

During the 1990 adult trapping program, a set of blood samples was taken from Caspian terns of known age and tested for total PCB burdens (Mora et al. 1993). Eggs from these same colonies also were tested for TCDD-Equivalents in other research projects between 1986 and 1990 (Tillitt et al. 1991b., Ludwig et al. 1996). The TCDD-equivalents data provided the same contaminants ranking as did the total PCBs blood plasma data. Results were combined for plasma PCB analyses by region, and the rate of return of breeding birds to their natal region was determined from the banking data. The three Canadian (cleanest) colonies averaged a return rate of 73%, the moderately contaminated colonies in northern Lake Michigan returned 43% of their fledglings to nest as adults, but the most highly contaminated birds from Green Bay and Saginaw Bay returned just 21% of their chicks to their natal area (chi square  $P < 0.002$ ). Over all colonies, return to natal colonies correlated to total PCBs in plasma ( $r^2 = 0.83$ ). Furthermore, the average age of the breeding adults in the three Canadian colonies ( $N=16$ ) was 12.3 years, 8.9 years in the northern Lake Michigan colonies ( $N=21$ ) and only 7.3 years in the three most contaminated colonies ( $N = 29$ ;  $P < 0.003$ ). In the period of 1981-1995, 1,604 tern chicks were banded in Saginaw Bay. Only two (0.12%) returned to nest in any colony in the Great Lakes, and only three were found as recoveries more than 20 miles from their banding site. No immatures were recovered on the wintering grounds. By

comparison, L'Arrivee and Blokpoel (1988) recorded 505 recoveries of birds >20 miles from the banding site (2.14%) and 621 recaptures in the trapping studies (2.73%) from the banding of 23,643 chicks in the whole population 1959-1986. Clearly, while the food is abundant and the habitat appears to be very favorable in these contaminated areas, nesting in them is a very dangerous business for a tern! Yamashita et al. (1993) reported on eggs collected 15 and 49 days after terns arrived back in Saginaw Bay from winter quarters. They found the second clutch eggs to be more than twice as contaminated as first clutch eggs with total PCBs and TCDD-EQs, and that the eggs built-up residues at the rate of 0.26 ppm/day ww for each day in residence (Ludwig et al. 1993).

The conclusions of all of these field banding studies correlated with actual contaminants data may be summarized by the following statements. Terns raised in contaminated areas appear to be healthy when fledged. However very few survive to breeding age; perhaps as few as 3% of those that fledge. Immune system damage from TCDD-like contaminants may be the primary mechanism that lowers tern survival in spite of better food and a superficially more hospitable habitat in the more productive, though highly contaminated areas. The growth and maintenance of this population is dependent on the terns nesting in the cleanest Canadian colonies. Colonies in highly contaminated area function as population sinks where the turnover of birds is greatest and adult survival is lowest. These are identical findings to the bald eagle studies discussed above (Bowerman et al. 1995, Giesy et al 1995), and are fully consistent with the observations made on mink and otter (Wren 1991, Giesy et al. 1994c). It also reminiscent of the work done on the St. Lawrence Beluga whales which are highly contaminated with organochlorine chemicals (especially PCBs), suffer debilitating diseases, and give strong evidence of suppressed immune function (Beland et al. 1993).

Critics of the hypothesis that it is the contaminants controlling the trajectory of Great Lakes communities often point to the population growth of ring-billed gulls and double-crested cormorants as proof that the case against contaminants is overstated and could not possibly be true (Cooper 1995). Both of these species are migratory, and less exposed than the resident species. Samples of ring-billed gull eggs suggest that they are generally about half as contaminated with the TCDD-like chemicals as their nearest competitor, herring gulls (Tillitt et al. 1991). Further, they eat fewer Great Lakes fish, more insects and earthworms than herring gulls and probably are much less exposed to synthetic chemicals (Ludwig 1974). Cormorants have been found to have a much more robust detoxification metabolism for these chemicals than Caspian terns feeding on the same fish species at the same sites (Yamashita et al. 1993), a situation that probably pertains to other larids such as the Forster's tern (Kubiak et al. 1989).

It is instructive to look at other species Great Lakes' populations for which there are fewer contaminants data. Assuming that all terns share a similar metabolic capability to detoxify contaminants, one may ask which species and populations would one expect to find effects in, and which should be affected first. It is well known that homeothermic species' standard metabolic rates are inversely related to body mass: smaller species have higher standard metabolic rates just to stay warm owing to their greater surface to volume ratios. This requires energy, and translates directly into a higher rate of food consumption per unit of body mass in the small species. All three of the smaller terns [black (*Chilodias niger*), common (*Sterna hirundo*), and Forster's (*Sterna forsteri*)] on the Great Lakes have suffered drastic population declines since 1960. Black and Forster's terns are substantially extinguished from Saginaw Bay and barely holding on in Green Bay (Kubiak et al. 1989). Common tern populations in Michigan 1995 were about 12% of the size of the population in the 1960-62 period (Ludwig, 1962, J.P. Ludwig unpublished data). Black terns, once common in Saginaw, Green and Thunder Bay areas are virtually extinguished there, while barely surviving in the Les Cheneaux district of Lake Huron, and St. Mary's River connecting Lake Superior to Lake Huron (J.P. Ludwig, unpublished observations). Among the terns, one would predict the largest member of this guild, the Caspian, would be the slowest to be affected by contaminants because of a lower metabolic rate. Among all guilds of species in a fish-consuming food web, one would predict that the secondary predators (e.g., Caspian terns, cormorants, and

right-billed gulls) would be less exposed and affected than the secondary-tertiary predators (eagles, mink, snapping turtles, salmonids) which in turn should be less affected than the true tertiary predators (peregrines). In fact, this is an abundance pattern that has developed for these predators throughout the Great Lakes watershed since 1960.

### **Summary and Conclusions.**

Just as many others have discussed the effects of point source discharges and sedimentation on watershed resilience and values, the same is true for contaminants. Where there were large point source discharges to the Great Lakes, contaminant problems have appeared in virtually all of the wildlife taxa from every vertebrate class. Which species have been damaged seriously has been controlled by a complex set of intrinsic and extrinsic factors, but chiefly these:

- Species feeding at the secondary-tertiary and tertiary predator trophic levels were universally affected, regardless of taxon. The higher in the food web, the more damaged the species.
- Smaller homeothermic species were damaged more seriously than larger species in the same taxa and guilds.
- Within taxa and guilds, some species were genetically very sensitive and seriously damaged (e.g., mink) while others (e.g., cormorants and Caspian terns) were more resistant or less exposed, and consequently less affected.

These observations support the speculations that contaminants, as major agents of mortality in the Great Lakes, are important determinants of the trajectories of evolution (Ludwig 1974, Fox 1995). Evolution's two handmaidens are reproduction and death. Reproduction generates variation in populations that make some individuals more fit to withstand a stress than others. Death selects the best fit survivors, thereby altering gene frequencies to make a more fit population. The TCDD-like contaminants both limit the numbers of animals reproduced, and alter the capability of those that do survive their early life states to resist disease (Grasman et al. 1996). In some ways, these selective pressures from contaminants are similar to epizootic diseases. However, unlike a parasitic or bacterial infestation, the mechanisms available to adjust to the selection may push exposed populations in directions that are ultimately harmful, and evolutionary "dead ends." For example, it has been noted that eggshell thinning caused by DDT selected against those females that laid eggs with average or below average shell thickness in the pelicaniforms. In places where this selection was severe, such as the Southern California Bight that was severely polluted by the world's largest DDT manufacturing facility for nearly three decades, the local pelicaniforms were drastically affected by thinned eggshells. Only those with a genetic predisposition to lay the thickest eggs had their eggs survive. In 1993, 21 years after the DDT-manufacturing plant closed and 16 years after reproduction of these species resumed, the average cormorant nesting in this area laid eggs with shells 5% to 8% thicker than eggshells were in the pre-DDT exposure period (Fry, unpublished data). Thicker shells take more energy and material to assemble. Unless the delicate pore spaces remain as functional as those in unthickened eggshells, embryos in the thick-shelled eggs may not lose enough water during development to be normal, nor have the extra energy to break out and hatch from these thickened eggs. The long-term effect of DDE-mediated eggshell thinning may be to make an exposed population less fit to survive other "normal" stresses that will not be detectable with traditional ecotoxicological research and monitoring studies. Indeed, it may be dangerous to tinker with wildlife gene frequencies (Fox 1995).

### **References**

Research for this paper is based on more than 40 references of technical materials. Contact AES for the complete bibliography.