Potential Hazards of Environmental Contaminants to Avifauna Residing in the Chesapeake Bay Estuary

BARNETT A. RATTNER[1] AND PETER C. MCGOWAN[2]

[1]U.S. Geological Survey, Patuxent Wildlife Research Center, Beltsville, MD 20705
Internet: brattner@usgs.gov

[2]U.S. Fish and Wildlife Service, Chesapeake Bay Field Office, Annapolis, MD 21401

Abstract.—A search of the Contaminant Exposure and Effects-Terrestrial Vertebrates (CEE-TV) database revealed that 70% of the 839 Chesapeake Bay records deal with avian species. Studies conducted on waterbirds in the past 15 years indicate that organochlorine contaminants have declined in eggs and tissues, although *p,p'*-DDE, total polychlorinated biphenyls (PCBs) and coplanar PCB congeners may still exert sublethal and reproductive effects in some locations. There have been numerous reports of avian die-off events related to organophosphorus and carbamate pesticides. More contemporary contaminants (e.g., alkylphenols, ethoxylates, perfluorinated compounds, polybrominated diphenyl ethers) are detectable in bird eggs in the most industrialized portions of the Bay, but interpretation of these data is difficult because adverse effect levels are incompletely known for birds. Two moderate-sized oil spills resulted in the death of several hundred birds, and about 500 smaller spill events occur annually in the watershed. With the exception of lead, concentrations of cadmium, mercury, and selenium in eggs and tissues appear to be below toxic thresholds for waterbirds. Fishing tackle and discarded plastics, that can entangle and kill young and adults, are prevalent in nests in some Bay tributaries. It is apparent that exposure and potential effects of several classes of contaminants (e.g., dioxins, dibenzofurans, rodenticides, pharmaceuticals, personal care products, lead shot, and some metals) have not been systematically examined in the past 15 years, highlighting the need for toxicological evaluation of birds found dead, and perhaps an avian ecotoxicological monitoring program. Although oil spills, spent lead shot, some pesticides, and industrial pollutants occasionally harm Chesapeake avifauna, contaminants no longer evoke the population level effects that were observed in Ospreys (*Pandion haliaetus*) and Bald Eagles (*Haliaeetus leucocephalus*) through the 1970s.

Key words.—Biomarkers, contaminants, pollution, reproduction, risk assessment, stressors.

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Agricultural, industrial and urban activities are known to have had major effects on waterbird populations and habitats in the Chesapeake Bay. During the 20th century, chemical inputs into the estuary from anthropogenic activities had devastating effects on numerous species. Over 15 years have elapsed since the publication of significant reviews examining environmental contaminant exposure and effects on birds in the Chesapeake. Ohlendorf and Fleming (1988) concluded that *p,p'*-DDE in Ospreys (*Pandion haliaetus*) and Bald Eagles (*Haliaeetus leucocephalus*), cadmium and lead in seaducks, and lead in dabbling ducks were significant contaminant issues, and the hazards posed by many other chemicals (e.g., polycyclic aromatic hydrocarbons, industrial chemicals, organotin compounds, agrichemicals, and mosquito control agents) warranted investigation. Subsequently, Heinz and Wiemeyer (1991) suggested that there was little evidence indicating that organochlorine pesticides and PCBs were a significant threat to birds in the Chesapeake, but heavy metals such as lead and cadmium may be affecting Canvasbacks (*Aythya valisineria*) and other waterfowl. Both reviews suggested that research should focus on avian food chain organisms, and potential indirect effects of contaminants on food abundance.

In this review, ecotoxicological observations and research findings for avifauna residing in the Chesapeake estuary and nearby coastal barrier islands are summarized, focusing principally on data published since 1988. In addition, significant issues and potential threats warranting investigation are identified. Although environmental contamination can alter habitat quality and avian food resources, such indirect effects of pollution on Chesapeake avifauna have yet to be thoroughly investigated and thus will not be discussed here.

METHODS

The Contaminant Exposure and Effects—Terrestrial Vertebrates (CEE-TV) database (www.pwrc.usgs.gov/contaminants-online) contains over 17,000 geo-referenced data records for marine and estuarine habitats for the Atlantic, Gulf and Pacific coasts, Alaska, Hawaii and the Great Lakes (Rattner *et al.* 2005). Information in this database has been compiled using literature search tools (e.g., Wildlife Review, BIOSIS®, TOXLINE®, AGRICOLA), by examining records in various databases (e.g., U.S. Environmental Protection Agency Incident Information System, U.S. Fish and Wildlife Service Environmental Contaminant Data Management System, U.S. Geological Survey National Wildlife Health Center Mortality Database), and by contacting scientists in government agencies, conservation organizations, and academic institutions. All data are referenced with geographic collection coordinates, and if absent from the source document, coordinates were assigned based on the location description using MapExpert® version 2.0 (DeLorme Mapping, Freeport, ME) or the U.S. Geologic Survey Geographic Names Information System (http://geonames.usgs.gov/gnishome.html). If samples were collected across an entire county or state, the coordinates of the county seat or state capital were assigned to the record. Data were coded and compiled in a 118-field database in Microsoft® Access 2000 version 9.0 (Microsoft Corp., Redmond, WA) with information describing taxonomy, collection date, study location, geographic coordinates, sample matrix, contaminant concentration, biomarker or bioindicator response, and source of information. Data records for the present evaluation were identified based on the estuary or location description, and included Chesapeake Bay and nearby coastal sites.

The CEE-TV database contains 839 data records for the Chesapeake region (Fig. 1), ranging in sample size from a necropsy report of an individual bird (141 such reports) to significant research studies involving hundreds of individuals (e.g., a lead shot ingestion survey that examined 767 gizzards from Sora Rail, *Porzana carolina*). This CEE-TV subset of Chesapeake data contains information on 109 species representing an estimated 9,500 individuals. Sample collection dates ranged from 1966 to 2006, with most of the data from 1970 to 2000. Approximately 79% of these Chesapeake records contain avian ecotoxicological data, of which 292 records are for the time period from 1988 to present.

Avian data collected since 1988 were found in 78 necropsy reports, 17 peer-reviewed published studies, one dissertation, and six unpublished reports. Data are available for 89 unique contaminants (Table 1), most of which are legacy organochlorine contaminants (e.g., DDT, chlordane, dieldrin, PCBs) and heavy metals (e.g., cadmium, lead, and mercury). Very limited information is available on exposure to avicides, rodenticides, petroleum hydrocarbons, pharmaceuticals, and personal care and household cleaning products, and on biomarker or bioindicator responses.

Clearly, there are some biases in the data examined in this review. Although nearly all of the available terrestrial vertebrate ecotoxicological data have been compiled, a few investigators are reticent to share unpublished information and some data have been sequestered for legal purposes. Furthermore, the collection location of some samples was poorly described or is unknown. Finally, many studies focused on locations that are known to be highly polluted in order to document potential adverse effects. Ideally, overall condition assessments related to pollution should include randomly selected sampling locations.

ORGANOCHLORINE PESTICIDES

Initially recognized as a tremendous achievement for agricultural and human pest control, the hazardous effects of organochlorine pesticides to non-target wildlife became apparent in the 1950s and vaulted to the forefront of public concern with Rachel Carson's 1962 book *Silent Spring*. Toxicological effects of organochlorine pesticides and metabolites upon Chesapeake waterbirds occurred principally through impairment of reproduction rather than through altered survival of adults (Heinz and Wiemeyer 1991; Blus 1996). Recent findings on egg and tissue residues, and reproduction will first be presented, followed by exposure and effects data in adults.

Exposure of Eggs and Young

As described in earlier reviews (Ohlendorf and Fleming 1988; Heinz and Wiemeyer 1991), *p,p'*-DDE concentrations in Osprey and Bald Eagle eggs in the Chesapeake were among the highest in the nation and appear to have been the principal cause of the population declines in these species from the 1950s through 1970s (Wiemeyer *et al.* 1984, 1988, 1993). Since the banning of the most toxic organochlorine pesticides in the early 1970s, Chesapeake Bay Osprey and Bald Eagle populations gradually began to rebound (Watts and Paxton 2007; Watts *et al.* 2004, 2007).

Osprey eggs collected from Glenn L. Martin National Wildlife Refuge on Smith Island, Maryland in 1986 contained *p,p'*-DDE concentrations ranging from 0.82-3.0 µg/g wet weight (Audet *et al.* 1992). The median residue concentration exceeded the threshold associated with 10% eggshell thinning (i.e., 2 µg/g wet weight), but was below the value (>2.6 µg/g) associated with population instability (production of less than one fledgling per active nest) (Wiemeyer *et al.* 1988; Audet *et al.* 1992). Notably, concentrations had not changed much compared to a

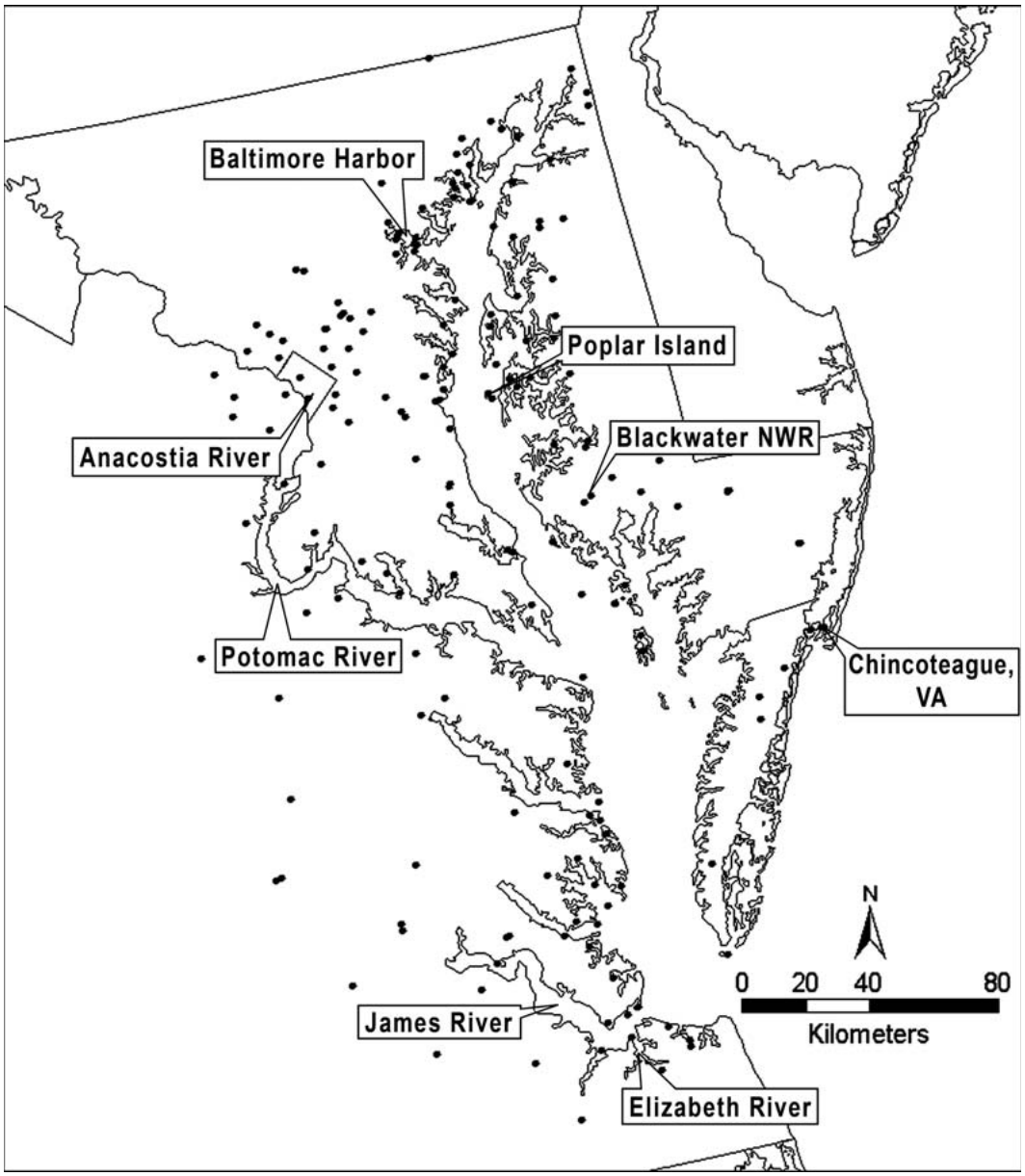

Figure 1. Geographic distribution of CEE-TV database records (n = 839) and some sample collection locations (regions of concern are bold) in the Chesapeake. Note that some records share common locations.

1973 collection from this site. Lower concentrations of *p,p'*-DDE (0.38-0.83 µg/g) were detected in Osprey eggs from the York River area, Mobjack Bay and the Rappahannock River in Virginia collected in 1987 (Audet *et al.* 1992), and were well below levels believed to cause adverse effects on eggshell thickness and productivity.

Coincident with recovery of their population in the Chesapeake (Watts *et al.* 2004; Watts and Paxton 2007), Ospreys began to nest in substantial numbers in some of the most polluted sites in the Bay (i.e., U.S. Environmental Protection Agency designated "Regions of Concern"), including Baltimore Harbor, and the Anacostia and Elizabeth

Table 1. Environmental contaminants detected in Chesapeake Bay avifauna since 1988.

Organochorine pesticides and metabolites	*p,p'*-DDD; *p,p'*-DDE; *p,p'*-DDT
	o,p'-DDD; *o,p'*-DDE; *o,p'*-DDT
	Dieldrin
	Heptachlor epoxide
	Oxychlordane
	cis-Chlordane
	trans-Chlordane
	cis-Nonachlor
	trans-Nonachlor
	Toxaphene
	Hexachlorobenzene
	Endrin
	Mirex
	alpha-hexachlorocyclohexane
	beta-hexachlorocyclohexane
	gamma-hexachlorocyclohexane
	sigma-hexachlorocyclohexane
Other organochlorine contaminants	Total PCBs (Aroclors 1254 and 1260)
	PCB congeners (66, 77, 81, 105, 114, 118/106, 123, 126, 128, 138, 156, 157, 158, 166, 167, 169, 170, 189)
	Dioxins (6 congeners)
	Dibenzofurans (2 congeners)
Brominated contaminants	Total polybrominated diphenyl ethers (PBDE)
	PBDE congeners (47, 49, 99, 100, 153, 154)
Surfactants	Nonylphenol
	Perfluorooctanesulfonate
	Perfluorodecanesulfonate
	Perfluorodecanoic acid
	Perfluoroundecanoic acid
Anticholinesterase pesticides	Organophosphorus insecticides (Fenthion, Chloropyrifos, Parathion, Phorate, Diazinon)
	Carbamate insecticides (Carbofuran, Oxamyl)
Avicides	Avitrol
Metals, metalloids and trace elements	Aluminum
	Arsenic
	Barium
	Beryllium
	Boron
	Cadmium
	Chromium
	Copper
	Iron
	Lead
	Magnesium
	Manganese
	Mercury
	Molybdenum
	Nickel
	Selenium
	Strontium
	Vanadium
	Zinc

Rivers. In 2000 and 2001, a large-scale study was undertaken in which Osprey eggs were collected from nests in these regions of concern and nearby tributaries (e.g., Patapsco River and middle Potomac River), and from a reference area (South, West and Rhode Rivers near Annapolis). Sample eggs were analyzed for various contaminants, and the fate of the eggs remaining in each nest was monitored through fledging of young to examine potential adverse effects of pollutants on reproduction (Rattner *et al.* 2004). Of 27

organochlorine pesticides and metabolites quantified, 17 or more were detected in half of the sample eggs from each region of concern, and concentrations of *p,p'*-DDE, *p,p'*-DDD, heptachlor epoxide, α-chlordane, *cis*-nonachlor, *trans*-nonachlor and oxychlordane were statistically greater in one or more of the regions of concern compared to the reference site. Concentrations of *p,p'*-DDE ranged from 0.26-1.92 µg/g wet weight in the regions of concern compared to 0.24-0.98 µg/g in the reference area. On a temporal basis, *p,p'*-DDE, dieldrin and *cis*-chlordane levels in Osprey eggs collected from the middle Potomac in 2000 (Rattner *et al.* 2004) were less than half of that observed in Potomac Osprey eggs collected in the 1970s. In 2000 and 2001, there were no significant differences in eggshell thickness, egg hatchability, reproductive success, or productivity of successful nests among study sites. However, shell thickness of eggs collected from the Anacostia and middle Potomac River study site averaged 8.7% less than in the pre-DDT era, and fell in the range of mean values (0.402-0.468 mm) for Osprey eggs from the lower Potomac River between 1968 and 1977 (Wiemeyer *et al.* 1975, 1988). Furthermore, over half of the sampled eggs from the Anacostia and middle Potomac Rivers had *p,p'*-DDE concentrations within the 95% confidence interval (1.2 to 3.0 µg/g) associated with 10% percent eggshell thinning in Osprey eggs (Wiemeyer *et al.* 1988). Eggshell thinning of greater than 18% is believed to be related to population declines in raptorial birds (Blus 1996). Concentrations of dieldrin, heptachlor epoxide, chlordane and other organochlorine pesticides or metabolites in this study were well below known reproductive effect levels for species that have been studied in detail. Kepone, a pesticide manufactured in Hopewell, Virginia that polluted the James River, had been found at concentrations of up to 5.0 µg/g wet weight in Osprey eggs collected from Chesapeake tributaries in Virginia in 1977 (Wiemeyer *et al.* 1988), but was not detected in a subset of Osprey eggs from the Elizabeth River in 2001.

Three Peregrine Falcon (*Falco peregrinus*) eggs that failed to hatch during the 1986 and 1987 breeding season were analyzed for a number of organochlorine contaminants (Jarman *et al.* 1993). An egg collected from Chincoteague, Virginia had the greatest concentrations of DDT and metabolites (25.2 µg/g wet weight), approaching the threshold associated with 20% eggshell thinning and reduced productivity for this species (i.e., 15 to 30 µg/g wet weight; Blus 1996). Concentrations of DDT and its metabolites were much lower in the eggs from Smith Island, Maryland and the Key Bridge in Baltimore Harbor (9.5 and 3.4 µg/g, respectively). The sum of chlordane metabolites in these eggs ranged from 0.92-2.4 µg/g. Avian reproductive effects thresholds are not known for chlordane. Up to 15 parts per million in the diet of Northern Bobwhite (*Colinus virginianus)* apparently has no effect on reproduction; unfortunately, egg residue values are not available from this study (J. W. Spann, unpubl. data cited in Wiemeyer 1996). In 1992, nonviable Peregrine Falcon eggs were collected from seven nests in the Chesapeake (Morse 1994), and were analyzed for chlorinated hydrocarbon contaminants and metals. Values of *p,p'*-DDE ranged from 1.0-11.7 µg/g wet weight. Dieldrin ranged from 0.05-0.90 µg/g, and was interpreted to be below the adverse effect threshold for reproduction in raptors (Wiemeyer *et al.* 1986). Other organochlorine pesticides and metabolites were lower, and suggested to be below reproductive effect levels (Morse 1994). Between 1993 and 1999, post-term and addled Peregrine Falcon eggs were collected from the Chesapeake region (e.g., Chincoteague and Martin National Wildlife Refuges, Clay, Cobb, Fisherman, Metompkin, South Marsh, and Wallops Islands, Baltimore, Wachapreague, and Fort Eustis) as part of a mid-Atlantic states monitoring effort (U.S. Fish and Wildlife Service *et al.* 2004). Concentrations of *p,p'*-DDE ranged from 1.14 to 23.9 µg/g wet weight, with the highest value being from Chincoteague National Wildlife Refuge. Some of these values might be artificially high as they were not adjusted for moisture loss. Notably, over a quarter of the Peregrine Falcon eggs collected from the Chesapeake region exhibited >15% shell thinning compared to pre-1947 shell

thickness values. Concentrations of chlordane metabolites and other organochlorine pesticides were low, rarely exceeding one µg/g wet weight. Unfortunately, productivity and nest success data for this study are incomplete, and thus it is not possible to fully evaluate the effects of contaminants on reproduction of this species in the Chesapeake.

In the past 15 years, numerous studies have reported organochlorine pesticide concentrations in Black-crowned Night-Herons (*Nycticorax nycticorax*), but these compounds have not been monitored in other wading bird species in the Chesapeake. Of the major Black-crowned Night-Heron colony sites in Maryland, most are on Bay islands or along the Atlantic shoreline, where human access and disturbance are minimal (Brinker *et al.* 1996). As part of a biomarker development study (Rattner *et al.* 1993, 1996), pipping Black-crowned Night-Heron embryos and eleven-day-old nestlings were collected in 1989 from Chincoteague Bay, Virginia. In pipping embryos, *p,p'*-DDE concentrations ranged from 0.06-5.2 µg/g wet weight, which was below the eight µg/g threshold associated with impaired reproduction in this species (Henny *et al.* 1984). Concentrations of *p,p'*-DDE in carcasses of young were considerably less than observed in embryos, ranging from non-detectable to 1.3 µg/g. The sum of other compounds (e.g., *p,p'*-DDD, dieldrin, heptachlor epoxide, α-chlordane, *cis*-nonachlor, *trans*-nonachlor, and oxychlordane) in pipping embryos averaged 0.74 µg/g, whereas residues of these pesticides were rarely detected in nestlings. These findings and others (Ohlendorf *et al.* 1978) verified the use of this colony as a reference site for subsequent studies.

In 1979, a small colony (25 pairs) of Black-crowned Night-Herons was found in a wooded area on the Baltimore Gas and Electric Plant fronting the Patapsco River in the highly industrialized Baltimore Harbor (Erwin 1990). This colony flourished to 325 pairs by 1988, making it the largest in Maryland's portion of Chesapeake Bay (Erwin 1990, 1991). This single species heronry, and two others (Rock Creek Park in Washington, D.C. and Chincoteague Bay, Virginia, a reference area), were studied in 1991 in order to examine contaminant residues and biomarkers of exposure and potential harm (Rattner *et al.* 1997). Organochlorine pesticide and metabolite residues were consistently greater in pipping embryos from Baltimore Harbor and Rock Creek Park compared to the reference area. Concentrations of *p,p'*-DDE in pipping embryos averaged between 0.23-1.59 µg/g, with only three of 45 embryos containing more than eight µg/g, the threshold associated with impaired reproduction in this species (Henny *et al.* 1984). Dieldrin, heptachlor epoxide, chlordane and other organochlorine pesticides rarely exceeded one µg/g. Concentrations of organochlorine pesticides on a µg/g wet weight basis were lower in ten-day-old nestlings than pipping embryos. However, on a body burden basis (µg/carcass), several pesticides, including heptachlor epoxide and several components of chlordane, were actually two to ten times greater in nestlings than in pipping embryos, indicating direct accumulation from food items of local origin.

Two ground censuses of the Baltimore Harbor Black-crowned Night-Heron colony in 1996 revealed a dramatic reduction to only 111-113 nests. A study assessing reproduction and contaminant exposure of this colony was undertaken in 1998 (Rattner *et al.* 2001), focusing on potential reproductive effects of PCBs, as this industrial contaminant had been linked to cytochrome P450 induction in earlier work at this site (Rattner *et al.* 1997). However, it became apparent that the Night-Heron colony had relocated to a mixed wading bird colony about two kilometers south at Fort Carroll, a 19th century installation in the middle of the Patapsco River. Based on analyses of a large number of sample eggs, concentrations of *p,p'*-DDE (0.023-1.29 µg/g wet weight) and dieldrin (not detected to 0.26 µg/g) were clearly lower than observed in 1991, and levels of other organochlorine pesticides were also quite modest. Pesticides in Night-Heron eggs concurrently collected from Holland Island, a remote reference colony in the southern Chesapeake, were but a fraction of those observed in Baltimore Harbor. There was nei-

ther eggshell thinning nor impaired reproduction in Night–Herons that could be associated with DDT exposure in this 1998 study.

Two studies examined organochlorine pesticides in Common Tern (*Sterna hirundo*) eggs. In 1994, ten eggs were collected from South Sand Point off of Barren Island, and were found to contain very modest levels of *p,p'*-DDE (0.04-0.21 µg/g wet weight) and *trans*-chlordane (not detected to 0.06 µg/g) (J. B. French, USGS Patuxent Wildlife Research Center, unpubl. data). In 1997, ten three-egg clutches were collected from Bodkin Island, and organochlorine pesticide concentrations (e.g., DDT and metabolites, chlordane and metabolites, dieldrin, endrin, heptachlor epoxide and mirex) were below the detection limit of 0.061 µg/g dry weight (French *et al.* 2001).

Exposure and Effects in Adults

In the final phase of the National Contaminant Biomonitoring Program of the U.S. Fish and Wildlife Service, wings of adult Mallards (*Anas platyrhynchos*) and American Black Ducks (*Anas rubripes*) collected from the Chesapeake region (Maryland and Virginia) in 1981-1982 were found to contain less *p,p'*-DDE than the overall average for the Atlantic Flyway (Prouty and Bunck 1986; Schmitt and Bunck 1995). With the exception of DDT and *trans*-nonachlor, other compounds (DDD, DDMU, dieldrin, heptachlor epoxide, *cis*-nonachlor and mirex) were not detected in the 1981-82 collection (Prouty and Bunck 1986). Starling (*Sturnus vulgaris*) carcasses collected from Maryland and Virginia in 1982 and 1985 also indicated that *p,p'*-DDE concentrations were moderate to low compared to much of the United States (Bunck *et al.* 1987; Schmitt and Bunck 1995). These monitoring studies demonstrated the overall decline in the concentration of persistent organochlorine pesticides in the continental United States (Schmitt and Bunck 1995), and the subset of these CEE-TV data for the Chesapeake supports this conclusion.

Eight Osprey carcasses were collected in 1993 from Tabbs Creek, a small tributary of the northwest branch of Back River in York County, Virginia (Hale *et al.* 1996). DDT metabolites were quantified in mesenteric tissue, and *p,p'*-DDE concentrations ranged from 5.3 to 12.9 µg/g dry weight. These values are lower than most observed in adult Ospreys collected between 1964 and 1982 (Wiemeyer *et al.* 1980, 1987) when rough adjustments are made for moisture and lipid content to normalize reporting units.

The New York State Department of Environmental Conservation performed necropsies on numerous adult birds that were collected from the Chesapeake Bay region, and at least six appear to have succumbed to organochlorine pesticide intoxication. For example, in 1988 and 1989, brain tissue of a number of dead individuals (American Kestrel, *Falco sparverius*; Red-shouldered Hawk, *Buteo lineatus*; Barred Owl, *Strix varia*; American Robin, *Turdus migratorius*, Blue Jay, *Cyanocitta cristata*, all from Prince Georges and Montgomery Counties in Maryland) contained substantial quantities of oxychlordane (2.41-4.10 µg/g wet weight) and heptachlor epoxide (2.63-6.35 µg/g wet weight), and the cause of death was attributed to chlordane intoxication (NYDEC Cases 88-30-20, 88-39-23, 88-39-25, 89-07-34, and 89-62-29). In addition, the death of a Cattle Egret (*Bubulcus ibis*) that succumbed during rehabilitation efforts in 1990 was attributed to dieldrin intoxication following detection of 3.28 µg/g wet weight in brain tissue (NYDEC 90-22-26).

ORGANOPHOSPHORUS AND CARBAMATE PESTICIDES

Use of organophosphorus and carbamate pesticide in the United States increased greatly following the ban of many organochlorine pesticides in the 1970s. These compounds or their activated metabolites inhibit the enzyme acetylcholinesterase, thereby disrupting neurotransmitter processes in the central nervous system and normal neural functioning of the sensory, integrative, and neuromuscular systems (Ballantyne and Marrs 1992). Generally, these pesticides have a short environmental half-life, but some remain quite toxic for days or weeks after application. Exposure of non-target wildlife to

these compounds has been demonstrated to cause alterations in behavior, endocrine function, thermoregulation, and reproduction (Grue *et al.* 1997). Death can result from respiratory failure due to constricted airways, decreased ventilation associated with paralysis of intercostal muscles and diaphragm, and the direct depression of respiratory centers in the brain. Definitive evidence of poisoning in wildlife usually entails detection of the parent compound in the digestive tract and substantial inhibition of brain acetylcholinesterase activity (Hill 2003).

For the period of 1988 to 2006, the CEE-TV database contains 133 records documenting at least 50 incidents of unintentional bird poisonings in the Chesapeake Bay region that involved anticholinesterase pesticides, and most of these incidents occurred in agricultural settings. These die-offs involved six orders of birds, most frequently passerines, birds of prey, and waterfowl. These incidents were principally caused by carbofuran exposure of prey (including one Bald Eagle) (Stinson *et al.* 1994). Use of granular formulations of this carbamate was restricted by the U.S. Environmental Protection Agency in 1994 because of bird die-offs. The remaining incidents involved chlorpyrifos, diazinon, fenthion, phorate, parathion and the carbamate oxamyl.

Other Economic Poisons

Two avian die-offs involving the avidicide Avitrol® (4-aminopyridine) occurred in Virginia in 1991 and 1993. One case was permitted use near a granary, and resulted in the death of Starlings (Virginia Department of Game and Inland Fisheries Case Number 3-91). The other instance entailed unapproved use of Avitrol® at a country club, and resulted in the death or intoxication of at least 39 Canada Geese (*Branta canadensis*) (Virginia Department of Game and Inland Fisheries Case Number 32-93). There were no reports found that documented instances of bird die-offs related to rodenticide poisoning, although this is certainly a problem in Atlantic coast states (e.g., New York; Stone *et al.* 2003).

Polychlorinated Biphenyls, Dioxins and Dibenzofurans

Use of PCBs dates back to the 1930s, and commercial applications of these compounds include heat transfer agents, flame retardants and waterproofing agents. The manufacture and distribution of these compounds in the United States was prohibited in 1979, but their massive production (1.3 billion pounds over a 50-year period) and their environmental stability has resulted in the distribution of the 209 congeners plus metabolites in abiotic and biotic matrices on a global scale (Rice *et al.* 2003). Polychlorinated dibenzo-*p*-dioxins and polychlorinated dibenzofurans are highly toxic contaminants with no commercial use that are released by chemical and combustion processes (Rice *et al.* 2003). In general, birds are more tolerant of acute exposure to these compounds than mammals, but a range of effects (e.g., enzyme induction, altered growth and reproduction, chick edema disease, immune dysfunction and endocrine disruption) have been linked to exposure (Rice *et al.* 2003).

Exposure and Effects in Eggs and Young

In studies of Chesapeake Bay raptors from the late 1960s to the mid-1980s, total PCB concentrations in Bald Eagle eggs averaged about 25 µg/g wet weight (ranging from 8.9-218 µg/g) (Wiemeyer *et al.* 1984, 1993), whereas concentrations in Osprey eggs were somewhat lower, ranging up to 18 µg/g (Wiemeyer *et al.* 1988). Although a topic of some controversy, PCBs were not associated with adverse reproductive effects in these species in the Chesapeake. To the best of our knowledge, there are no published data on PCB concentrations in Chesapeake Bay Bald Eagle eggs since 1984.

Total PCB concentrations in Osprey eggs collected from Glenn L. Martin National Wildlife Refuge in 1986 appeared to be lower than found at this site in 1973 (1.0 µg/g wet weight versus 2.8 µg/g), although values from the York River area, Mobjack Bay and the Rappahannock River in Virginia aver-

aged 3.7 µg/g, and ranged up to 5.7 µg/g (Audet *et al.* 1992). However, total PCB concentrations (principally Aroclors 1254 and 1260) in Osprey eggs collected from Baltimore Harbor and the Patapsco River, and the Anacostia and middle Potomac Rivers in 2000 were greater, averaging 7.25 µg/g and 9.28 µg/g, respectively. The upper extreme value was 19.3 µg/g from an egg collected near the Naval Research Laboratory on the middle Potomac (Rattner *et al.* 2004), and was actually similar in concentrations to the greatest historical values reported in Osprey eggs from the Chesapeake (Wiemeyer *et al.* 1988). Osprey eggs from the Elizabeth River, the location of the largest military naval port in the world, contained the lowest total PCB values in this study. Surprisingly, total PCB concentrations in eggs from the South, West and Rhode rivers reference areas averaged 4.60 µg/g, and ranged up to 12.4 µg/g. Compared to total PCB values reported in collections in the 1970s and 1980s by Wiemeyer and coworkers (1988), it would appear that concentrations in Osprey eggs have not declined; this trend has also been noted for Osprey eggs for much of the Atlantic coast (Rattner *et al.* 2005). Levels of 15 arylhydrocarbon (*Ah*) receptor-active PCB congeners were quantified in these Osprey eggs. Concentrations of the toxicologically most potent coplanar and semi-coplanar congeners (i.e., numbers 77, 81, 105, 126 and 169) did not differ much between study sites, although values were consistently higher in 2000 compared to 2001 samples. Toxic equivalents of 15 *Ah* receptor-active congeners did not differ among sites in this Chesapeake Bay study, and on a national scale, values were greater than those observed in the Pacific Northwest (Elliott *et al.* 2000; Henny *et al.* 2003), but not unlike toxic equivalents observed in Delaware Bay (Toschik *et al.* 2005) and the Great Lakes (Martin *et al.* 2003).

Cytochrome P4501A, one of the most sensitive biomarkers of exposure to polyhalogenated compounds, was quantified in liver and other tissues of pipping Osprey embryos collected from Back River, York County Virginia, a site in close proximity to Tabbs Creek that is known to be polluted with polychlorinated terphenyls (PCT) and PCBs (Stegeman *et al.* 1995). Pipping embryos were also collected from Mobjack Bay, in Gloucester and Matthews Counties, Virginia, which served as a presumed reference area. Catalytic activity of ethoxyresorufin dealkylase in hepatic microsomes and immunohistochemical measurements of cytochrome P4501A content in hepatic parenchymal cells did not differ among sites. Aroclor 1260 was detected in eggs from both sites, but PCT was not. The investigators concluded that Ospreys may not have been feeding on fish from the PCT contaminated outfall.

Peregrine Falcon eggs that failed to hatch in 1986 and 1987 contained total PCBs in concentrations ranging from 5.8-25.0 µg/g wet weight; an egg from Chincoteague, Virginia had the largest PCB levels and also had remarkably high concentrations of DDT metabolites (Jarman *et al.* 1993). In this study, east coast Peregrine Falcon eggs were noted to have much greater PCB concentrations than falcon eggs from the western United States. Nonviable Peregrine Falcon eggs collected from nests in the Chesapeake in 1992 contained Aroclor 1254 values ranging from 2.0-5.7 µg/g wet weight, and Aroclor 1260 values ranging from 4.1-10.9 µg/g (Morse 1994). Peregrine Falcon eggs collected between 1993 and 1999 exhibited total PCB concentrations ranging from 3.62-44.3 µg/g wet weight (U.S. Fish and Wildlife Service *et al.* 2004). Apparently, these addled and post-term eggs are the only avian samples in the Chesapeake region that have been analyzed for dioxins (not detected to 97 pg/g wet weight, not adjusted for moisture loss) and dibenzofurans (not detected to 128 pg/g). Mathematically predicted toxic equivalent of dioxins, dibenzofurans and *Ah* receptor-active PCB congeners were presented for the entire mid-Atlantic study area, and appear to be slightly above estimates of the no observed adverse effect level for developmental impairment in raptors, but well below the lowest observable adverse effect level for embryo mortality (U.S. Fish and Wildlife Service *et al.* 2004).

Total PCB and *Ah* receptor-active congener exposure was determined in Black-crowned Night-Herons from Chincoteague

Bay, Virginia. In pipping embryos and eleven-day-old nestlings, total PCB concentrations averaged 1.13 µg/g wet weight and 0.36 µg/g, respectively; these values were considerably lower than observed in concurrently collected samples from the Great Lakes and San Francisco Bay (Rattner *et al.* 1993, 1996). Likewise, concentrations of twelve *Ah* receptor-active congeners, mathematically predicted toxic equivalents, and H4IIE bioassay-derived dioxin equivalents in pipping embryos from the Chincoteague Bay reference site were but a small fraction of those observed in samples from the Great Lakes and San Francisco Bay (Rattner *et al.* 1994). Mean activity and variability of several cytochrome P450-associated monooxygenases (arylhydrocarbon hydroxylase, benzyloxyrsorufin-O-dealkylase, ethoxyresorufin-O-dealkylases, and ethoxycoumarin O-dealkylase) in hepatic microsomes of pipping embryos from Chincoteague were significantly lower than those observed in the Great Lakes, in part validating the use of these biomarkers as indicators of exposure to some PCB mixtures in Night-Herons (Rattner *et al.* 1993). Activities of arylhydrocarbon hydroxylase and benzyloxyrsorufin-O-dealkylase tended to be lowest in eleven-day-old nestlings from Chincoteague Bay compared to other more polluted sites in this study, although cytochrome P450-associated monooxygenase activities seem to be a less robust biomarker during this rapid-growth life stage (Rattner *et al.* 1996).

Studies conducted with Black-crowned Night-Heron pipping embryos in 1991 documented total PCB, *Ah* receptor-active PCB congeners, and toxic equivalents to be up to 37-fold greater in Baltimore Harbor and Rock Creek Park compared to levels in embryos from Chincoteague Bay, Virginia (Rattner *et al.* 1997). Hepatic microsomal activities of benzyloxyrsorufin-O-dealkylase and ethoxyresorufin-O-dealkylase of embryos from the two most PCB polluted sites were six- to nine-fold greater than those found in embryos from Chincoteague Bay. Accumulation of total PCBs in ten-day-old nestlings from Baltimore Harbor was much greater than that observed at Chincoteague Bay (252 µg/carcass versus 33 µg/carcass). Findings of high concentrations of PCB congeners and toxic equivalents, as well as cytochrome P450 induction in Baltimore Harbor herons, was the impetus for testing the hypothesis that PCBs might be leading to the declining size of the Baltimore Harbor heron colony (Rattner *et al.* 2001). Although twelve *Ah* receptor-active PCB congeners and toxic equivalents were up to 37 times larger in sample eggs from Baltimore Harbor compared to those from the reference area in the southern Chesapeake, overall nest success (0.74) and productivity (2.05 young/nest) were adequate to maintain a stable population. Furthermore, no significant relation was found between hatching, fledging and overall reproductive success and concentrations of PCBs and toxic equivalents. It was concluded that contaminants were not having a dramatic effect on reproduction in the Baltimore Harbor heronry.

Concerns over PCBs and other pollutants near Mason Neck National Wildlife Refuge, prompted an investigation of its Great Blue Heron (*Ardea herodias*) colony, the largest in Virginia. In 1997, eggs were collected from Mason Neck and from Coaches Island (near Tilghman Island on Maryland's Eastern Shore, the reference site in the Chesapeake), and were artificially incubated until they hatched (Johnson *et al.* 2001). There was no evidence of cytochrome P450 induction or oxidative stress that might be linked to polyhalogenated pollutant exposure.

In a preliminary study examining potential endocrine disruptive effects of PCBs, Common Tern eggs collected in 1994 from South Sand Point, off of Barren Island, contained relatively low concentrations of Aroclor 1260 ranging from 0.44-1.50 µg/g wet weight (J. B. French, unpubl. data). In testing this hypothesis, eggs were collected from Bodkin Island, which served as a comparative reference site for the more contaminated samples from Ram Island in Buzzards Bay, Massachusetts. Total PCBs concentrations (<ten µg/g lipid) were much lower at Bodkin Island compared to Ram Island, and no evidence was obtained to suggest that PCBs were evoking toxic effects in embryos (French *et al.* 2001).

Tree Swallow (*Tachycineta bicolor*) eggs and nestlings from the USGS Patuxent Wildlife Research Center on the Patuxent River, a tributary of the middle Chesapeake Bay, generally contained considerably lower concentrations of total PCBs than samples collected from PCB-polluted sites in Indiana, New York and Pennsylvania (eggs: 0.69 µg/g versus 0.94 to 4.6 µg/g; nestling body burdens: 0.294 µg/g versus 0.169 to 18.46) (Yorks 1999).

Exposure and Effects in Adults

Wings of adult Mallards and American Black Ducks collected from the Chesapeake region (Maryland and Virginia) in 1981-1982 as part of the National Contaminant Biomonitoring Program contained moderate amounts of total PCBs that were less than the overall average for the Atlantic Flyway (Prouty and Bunck 1986). Starling carcasses collected from Maryland and Virginia in 1982 contained total PCB concentrations that were slightly higher than the national average (Bunck *et al.* 1987). These monitoring studies indicated that total PCB concentrations did not decline as dramatically as levels of organochlorine pesticides and metabolites.

Concentration of total PCBs in mesentery of dead adult Ospreys from Tabbs Creek in York County, Virginia, ranged from 11.3-45.3 µg/g dry weight (Hale *et al.* 1996), which appears to be slightly lower than the range of normalized values reported for adult osprey carcasses and brain tissue collected from 1964-1982 (Wiemeyer *et al.* 1980, 1987). In necropsy reports of the New York State Department of Environmental Conservation describing adult birds that apparently succumbed to organochlorine pesticide intoxication from the Chesapeake Bay region, Aroclors 1254 and 1260 were below the limit of detection (NYDEC Cases 89-07-34, 89-62-29, and 90-22-26).

NEWER ORGANIC CONTAMINANTS

Recent studies have detected with increasing frequency the presence of chemicals and pharmaceuticals in the environment that have not been historically detected or examined, and thus never considered environmental contaminants (Kolpin *et al.* 2002). These "emerging contaminants" include compounds such as steroids, caffeine, antimicrobials, fire retardants and nonionic detergent metabolites that may be released by municipal, agricultural and industrial wastewater sources. Often serving as focal points of commerce and human populations, estuaries such as the Chesapeake Bay may receive and actually trap large quantities of such chemicals (Hale and La Guardia 2002). Although widely recognized as a potential concern to biota, exposure to and the effects of many of these compounds are only just now being investigated from an ecotoxicological perspective. Remarkably, exposure data in Chesapeake Bay avifauna is limited to one study in Osprey eggs (Rattner *et al.* 2004).

Perhaps the most interesting group detected is the polybrominated diphenyl ether flame retardants. Using archived Herring Gull (*Larus argentatus*) eggs collected over the past 25 years from the Great Lakes, these flame retardants appear to be doubling in concentration every three to five years over the past 25 years (Norstrom *et al.* 2000). Concentrations in Chesapeake Osprey eggs of total polybrominated diphenyl ethers (PBDEs) approach one µg/g on a wet weight basis, and on a lipid weight basis, concentrations in Chesapeake Bay Osprey eggs are some of the highest values reported in bird eggs to date (Hale *et al.* 2004). The toxicity of these compounds is currently under investigation in avian egg injection studies (McKernan *et al.* 2006).

Perfluorinated compounds are surface protectors and surfactants that are distributed worldwide. Perfluorooctanoic acid, an active ingredient of Scotch Guard®, was voluntarily removed form the market in 2000 because it was increasingly being detected in the environment, wildlife and people. Several perfluorinated compounds have been detected in Chesapeake Osprey eggs, with concentrations of perfluorooctanesulfonate ranging up to 428 ng/g wet weight (Rattner *et al.* 2004). Recent controlled exposure studies and risk assessments in Northern Bobwhite and Mallards concluded that current environmental concentrations do not

pose a significant risk to avian populations (Newsted *et al.* 2005).

Alkylphenol and ethoxylate surfactant and cleaning agents are produced in multi-million ton quantities, and were occasionally detected in low nanogram quantities in Chesapeake Bay Osprey eggs (Rattner *et al.* 2004). Some members of this group of surfactants have been suggested to be endocrine disruptors, although such effects have yet to be clearly documented in birds.

PETROLEUM SPILLS

Since 1988, two moderate-sized oil spills have occurred in the Chesapeake Bay region. On 28 March 1993, a 90-cm (36 inch) high-pressure pipeline, running from the Gulf of Mexico to Maine, ruptured and released over 400,000 gallons of No. 2 heating oil into Sugarland Run Creek and the Potomac, affecting a 15-km stretch. Twenty-three oiled birds were observed, including Wood Ducks (*Aix sponsa*), Canada Geese, Mallards, and a Kingfisher (*Megaceryle alcyon*), and 18 of these succumbed despite rehabilitation efforts (Research Planning, Inc. 1993). Undoubtedly, more birds were affected by this spill. Another oil spill occurred on 7 April 2000 involving a pipeline rupture releasing 126,000 gallons of No. 2 and No. 6 fuel oil at the Potomac Electric Power Company Chalk Point Facility near Aquasco, Maryland. The spill spread to Swanson Creek, a tributary of the Patuxent; 55 dead birds (principally waterfowl, but also Ospreys, herons, gulls and terns) were found, and 109 birds were collected for rehabilitation (twelve of these died) (Cardano *et al.* 2001). This spill occurred near Osprey nests, and although ten adult Ospreys were observed to be oiled, there was no evidence of effects on reproductive success (Michel *et al.* 2001). Records of the U.S. Fish and Wildlife Service and U.S. Coast Guard indicate about 500 smaller spill events occur annually in the Chesapeake.

Although chronic low level oil pollution has been a long-term concern, effects in birds have been difficult to detect. One study examined cellular DNA content of Black-crowned Night-Heron embryos and nestlings, a sensitive genetic damage biomarker of exposure to petroleum and some metals (Custer *et al.* 1994). Although eggs and nestlings from petroleum polluted regions in Texas and Louisiana exhibited effects, there was no evidence of genetic damage in samples from Baltimore Harbor when compared to the Chincoteague Bay reference site.

METALS, METALLOIDS AND OTHER ELEMENTS

Considerable historic data exist for the concentrations of metals in tissue and eggs of wild birds in the Chesapeake Bay region. Heinz and Wiemeyer (1991) pointed out that metals did not seem to be involved in the decline of Bald Eagle and Osprey populations in the Chesapeake, and with the exception of lead exposure from spent shot, concentrations of other metals were below adverse effect thresholds for several species of waterfowl. Mercury and selenium have been measured in eggs, liver, and kidney, whereas other metals (e.g., lead, cadmium, nickel) have generally been quantified in liver and kidney because they are poorly transferred into eggs.

Lead

The CEE-TV database contains 48 records for Chesapeake waterbirds from 1988 to the present that describe lead concentration in liver. For 26 of these records (representing 84 individuals including waterfowl, geese, and Bald Eagles), lead concentration in liver exceeded two μg/g wet weight, a value associated with sub-clinical poisoning in waterfowl (Pain 1996), and ranged up to 183 μg/g dry weight in one of the Bald Eagles (>five μg/g wet weight is compatible with death; Franson 1996). One striking feature of these observations is their variability relative to the sub-clinical toxicity threshold. For example, in an unpublished study of dabbling and diving ducks collected in Baltimore Harbor between 1987 and 1989 (M. W. Tome, USGS Patuxent Wildlife Research Center, unpubl. data), liver lead concentrations in Canvasbacks and Lesser Scaup (*Aythya affinis*) averaged <0.5 μg/g dry weight (extreme value 1.2 μg/g), whereas for

Black Ducks, Mallards and Scaup liver lead averaged >two µg/g dry weight (extreme value for each species >15 µg/g). In a study of Long-tailed Ducks (*Clangula hyemalis*) collected throughout the Chesapeake during an outbreak of avian cholera, only one of forty individuals had detectable quantities of lead (>0.5 µg/g dry weight) in liver (Mashima *et al.* 1998). Concentrations of lead in livers of Mute Swans (*Cygnus olor*) collected in 1995 from several presumably unpolluted locations (Bloodsworth Island, Horseheads Wetland Center and Eastern Neck and Blackwater National Wildlife Refuges) averaged <1.5 µg/g dry weight (within the range of background; Pain 1996), yet one individual had 7.6 µg/g dry weight (Beyer *et al.* 1998). These concentration differences among species of waterfowl and swans are due to a number of factors, the foremost being feeding habits. Although lead may be incorporated in food items, ingestion of contaminated sediment or a single spent shot or fragment can greatly elevate liver lead concentration. Remarkably, exposure of waterfowl to spent lead shot (e.g., examination of gizzards for shot fragments, radiography or blood lead and protophorphyrin determinations) has not been systematically investigated in Chesapeake waterbirds since the ban of lead shot for hunting waterfowl in 1991.

Lead can be incorporated into feathers of growing birds (Golden *et al.* 2003a), and has been proposed as a sensitive minimally invasive indicator of exposure. Feathers of Black-crowned Night-Heron nestlings from Chincoteague Bay and Holland Island were lower (≤0.13 µg/g dry weight) than feathers of 14- to 16-day-old nestlings from Baltimore Harbor (0.32 µg/g) (Golden *et al.* 2003b). A similar tendency was noted in feathers of 40-45-day-old Osprey nestlings collected from Baltimore Harbor in 2000 (1.25 µg/g dry weight) compared to nestlings from the South River reference area near Annapolis (0.66 µg/g) (Rattner *et al.* 2008).

Mercury

Rising mercury concentrations in the environment and widespread fish consumption advisories have been a cause for national concern. However, in Chesapeake Bay and its tributaries, PCBs and pesticides, but not methylmercury, are the risk drivers for fish consumption advisories. The only current methylmercury fish consumption advisory is in Spring Gap in the Upper Potomac River near Hagerstown, Maryland, and is well above the fall line. Historically, mercury has not posed a threat to waterbirds in the Chesapeake Bay and its tributaries (Ohlendorf and Fleming 1988; Heinz and Wiemeyer 1991). Since 1988, five studies involving 119 eggs from Ospreys (Rattner *et al.* 2004), Peregrine Falcons (Morse 1994; U.S. Fish and Wildlife Service *et al.* 2004) and Common Terns (French *et al.* 2001) indicated that mercury concentrations averaged well below 0.5-1.5 µg/g wet weight, the currently accepted reproductive effect toxicity threshold in bird eggs (Wiener *et al.* 2003).

In an unpublished study of dabbling and diving ducks collected in Baltimore Harbor and the Rhode River between 1987 and 1989 (M. W. Tome, USGS, unpubl. observations), mercury concentration in 174 liver samples from Mallards, Black Ducks, Canvasbacks, Ruddy Ducks (*Oxyura jamaicensis*) and scaup ranged from 0.03-1.6 µg/g dry weight. In Long-tailed Ducks collected in 1994, mercury concentrations in liver ranged from 0.11-1.2 µg/g dry weight (Mashima *et al.* 1998). Mercury concentration in the kidney of a Bald Eagle found dead along the James River in 2001 was determined to be 8.80 µg/g wet weight (Southeastern Cooperative Wildlife Disease Study Case Number CC63-02). These values in adult birds are well below the 20 µg/g level in liver or kidney associated with toxicity (Wiener *et al.* 2003).

Mercury is readily incorporated into feathers of growing birds and adults following molt. Mercury levels in feathers of 14- to 16-day-old nestling Black-crowned Night-Herons from Baltimore Harbor and Holland Island did not differ, and ranged only from 0.04-0.23 µg/g dry weight (Golden *et al.* 2003b). Mercury concentrations in feathers of 40- to 45-day-old Osprey nestlings were greater in the Elizabeth River (0.26-2.40 µg/g dry weight) compared to Baltimore Har-

bor and the Patapsco River, Anacostia and the middle Potomac, and the South, West and Rhode Rivers (values ranged from 0.01-1.53 µg/g) (Rattner *et al.* 2008). However, these values were quite low compared to concentrations found in feather at mercury polluted sites. Based on the aforementioned mercury data, this metal does not appear to be a significant threat to birds in Chesapeake Bay proper.

Cadmium

Cadmium concentrations in livers of dabbling and diving ducks from Baltimore Harbor and the Rhode River averaged <two µg/g dry weight (M. W. Tome, USGS, unpubl. data). The mean concentration in livers of Long-tailed Ducks collected during the Chesapeake avian cholera outbreak in 1994 was 2.65 µg/g dry weight, but was much greater in kidney, averaging 22.7 µg/g and ranging up to 81 µg/g (Mashima *et al.* 1998). The mean cadmium concentration in livers of Mute Swans collected at Blackwater and Eastern Neck National Wildlife Refuges was 16 µg/g dry weight, and ranged up to 94 µg/g (Beyer *et al.* 1998). Cadmium is rarely detected in eggs, and poorly transferred into feathers of nestlings. The toxicity threshold for cadmium has not been adequately established for birds (perhaps >100 µg/g wet weight; Beyer 2000), and seemingly elevated concentrations in individuals may merely reflect increasing cadmium values with age.

Selenium

Selenium concentrations in Common Tern and Peregrine Falcon eggs averaged <2.6 µg/g dry weight (Morse 1994; French *et al.* 2001; U.S. Fish and Wildlife Service *et al.* 2004; J. B. French, USGS Patuxent Wildlife Research Center, unpubl. data), and were clearly below levels causing embryotoxicity, although this threshold is somewhat contentious (Ohlendorf 2003). Concentrations of selenium in the liver of waterfowl (M. W. Tome, USGS, unpubl. observations) and Mute Swans (Beyer *et al.* 1998) averaged <20 µg/g dry weight, and when converted to wet weight was below the ten µg/g wet weight concentration associated with sublethal adverse effects in young and adult Mallards (Heinz 1996). Notably, livers of male Ruddy Ducks collected on the Rhode River in 1987 to 1989 appeared to be higher, averaging 29.8 µg/g dry weight with one value ranging to 140 µg/g (M. W. Tome, USGS, unpubl. data).

Other Elements

Concentrations of aluminum, boron, chromium, copper, iron, manganese, strontium and zinc were often detected in tissues and eggs of Chesapeake waterbirds collected since 1988, and were generally in the range of values for healthy captive birds. Arsenic, barium, beryllium, molybdenum, nickel and vanadium were rarely detected in tissues and eggs.

Plastics and Fishing Tackle

Plastic pollution in aquatic environments is recognized as a worldwide problem (Derraik 2002), and ingestion of plastic, and entanglement in fishing line, nets and other polymer debris affects untold numbers of birds annually. Birds may be coincidentally exposed to lead and even PCBs during such encounters. While undertaking Chesapeake Bay Osprey studies in 2000 and 2001, the presence of plastic material (e.g., bags, tapes, fishing tackle) in nests was commonplace (Rattner *et al.* 2004 and unpubl. data). Subsequently, surveys conducted along several Maryland western shore tributaries of the Chesapeake Bay documented that plastic materials were present in more than 60% of the 139 nests surveyed, and many of these nests contained fishing line (P.C.M., unpubl. data). Discussions with Maryland Department of Natural Resources personnel suggest that >50% of the Osprey nests on the Patuxent River visited by biologists during banding of young have been noted to contain fishing line. Such debris in nests can pose a reproductive hazard by concealment of eggs (Ellis and Lish 1999), and entanglement of nestlings (Watson 1989). It is also well recognized

that fishing tackle and nets can be lethal to foraging adult waterbirds (e.g., Schrey and Vauk 1987; Kelly and Kelly 2004).

CONCLUSIONS AND RECOMMENDATIONS

Based on earlier reviews (Ohlendorf and Fleming 1988; Heinz and Wiemeyer 1991) and the present analysis, the widespread reproductive effects of organochlorine pesticides on waterbirds in the Chesapeake have subsided, although eggshell thinning in a few locations and even occasional lethality have been observed in the past 15 years. There have been numerous instances in which organophosphorus and carbamate pesticides have been linked to avian die-off events in agricultural regions of the Chesapeake, although use of the most hazardous anticholinesterases (e.g., parathion and granular formulations of carbofuran and diazinon) has been curtailed. Unlike organochlorine pesticides, concentrations of PCBs in bird eggs do not seem to have declined, and despite substantial exposure and biochemical biomarker responses, it is not clear as to their overall effects on waterbird populations. Despite nationwide interest and concern about potential effects of organic chemicals released from wastewater treatment plants and landfills, only limited information exists on exposure and effects of these chemicals on birds in the Chesapeake and elsewhere. At least two newsworthy petroleum spills occurred in the Chesapeake since 1988, and it is indeed fortunate that their effects on bird populations were apparently minimal. Although use of lead shot for hunting waterfowl was phased out from 1986 to 1991, it would seem that lead exposure and effects are still commonplace in the Chesapeake, particularly in industrialized regions. The threat of other metals, including cadmium and mercury, do not seem to be a significant stressor on waterbirds in most of Chesapeake Bay and its tributaries. With expanding growth, development and recreational use of the region, it is not surprising to find plastic pollution throughout the Bay. In comparison to contemporary avian data in other estuaries in the United States (Rattner *et al.* 2005), it appears that environmental contaminant exposure of Chesapeake avifauna for the past 15 years has been moderate, occasionally harming individuals, but not evoking widespread adverse effects at the population level.

Some research and monitoring recommendations of previous reviews have been addressed (Ohlendorf and Fleming 1988; Heinz and Wiemeyer 1991), while others have not. Data collected over the past 15 years has documented contaminant exposure, sublethal effects, and reproduction in some Chesapeake waterbird species, most notably Black-crowned Night-Herons, Ospreys and waterfowl. However, many wading bird species have largely been overlooked. Some of the controlled exposure and field investigations recommended 15 years ago have been pursued (e.g., heron productivity in industrialized Baltimore Harbor, lead exposure from sources other than shot), whereas others have been addressed to a limited degree (e.g., concentrations of selenium in waterbird tissues and eggs, toxicity of pesticides used in mosquito abatement programs, contaminant levels in avian food chain items, and indirect effects of toxicants that limit food availability), or not at all and still deserve some attention (e.g., potential effects of cadmium on health and reproduction of birds).

Several new ecotoxicological priorities for Chesapeake waterbirds have arisen, and will be briefly mentioned. Information in the CEE-TV database for the Chesapeake Bay region indicates that we have exposure data on a very small subset (N = 81 for Chesapeake Bay avifauna since 1988) of the >75,000 discrete chemicals that are in commerce in the United States. Many new groups of pesticides that have entered the market are not bioaccumulative (e.g., chlorfenapyr, imidacloprid, ivermectins). However, because of current registration processes, their effects on birds and other wildlife are incompletely known. Although some exposure monitoring has been undertaken for a few emerging contaminants (alkylphenols and ethoxylates, perfluorinated compounds, and polybrominated diphenyl ether flame retardants), it has become apparent that a wide array of

antibiotics, drugs, steroids, cleaning agents and fragrances are making their way through many wastewater treatment plants into tributaries (Kolpin *et al.* 2002), albeit at low concentrations. Although the toxicities of many of these compounds are understood from a human health perspective, neither their potential toxicity to birds nor the ecological consequences of chronic low level exposure are adequately known. In addition to potential concerns about new pesticides and emerging contaminant issues, some relatively well studied compounds deserve special attention in the Chesapeake. Remarkably, dioxin and dibenzofuran exposure in Chesapeake Bay wildlife has not been thoroughly examined, yet fish consumption advisories are in effect in northern portions of the Potomac and in the Chesapeake and Delaware Canal.

There has been increased interest on biotoxin effects on wildlife, particularly those associated with harmful algal blooms. In the fall of 2001, harmful algal blooms coincident with reports of dead and dying waterbirds at the Poplar Island Complex, Kent Island, and Grasonville in the Chesapeake Bay were received by federal, state and private conservation agencies. Most prominent was the mortality event at the Poplar Island Complex, involving about 100 individuals including Great Blue Herons. Necropsies were consistent with steatitis (inflammation of the adipose tissue). Microcystins, a cyanobacterial (*Anabaena* spp.) product that is a potent hepatotoxin, were detected in both water samples from constructed wetland cells at Poplar Island and in tissue samples of dead herons (P.C.M., unpubl. data). These algal blooms and bird die-offs recurred in 2004 and 2005. Whether microcystin exposure, steatitis and mortality are independent findings or associated in some way is currently under investigation. The expanding use of created wetland and storm water impoundments in urbanized portions of the Chesapeake could possibility foster algal blooms, and may be a looming issue for waterbirds.

From an ecotoxicological perspective, some parts of the Chesapeake have been intensively studied in the past 15 years, most notably Baltimore Harbor, and the Anacostia and Elizabeth Rivers, whereas other regions have not. An examination of the geographic density and collection dates of information in the CEE-TV database suggests at least two tributaries that may warrant special research and monitoring attention in the future: the Susquehanna River, the largest freshwater inflow to the Chesapeake, that provides a very significant component of the total nutrient pollution entering the Bay, and the Rappahannock River, a region for which there are limited waterbird contaminants data. Aside from these two locations other sites probably deserve study, particularly in view of the downward trend in new CEE-TV records since 2001 (only 45 records from 2001 to present) compared to previous decades (343 records from 1991-2000 and 160 records from 1981-1990).

In the past there have been serious consequences of environmental contamination on waterbird populations in the Chesapeake. In our opinion there is a need for an ongoing monitoring program that examines contaminant exposure and potential effects in birds and other wildlife. Such a program should include thorough necropsies and toxicological analyses of individuals found dead, and a rigorously designed collection and analysis scheme of waterfowl tissue (perhaps collected from recreational hunters) and Osprey eggs to document spatial and temporal trends. Ultimately, such information will enhance the prediction, monitoring and understanding of contaminants in the Chesapeake Bay with the ultimate goal of a Bay "free of toxic effects".

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