Chapter 2
Dioxins, Wildlife, and the Forest Industry in British Columbia, Canada

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On the Pacific coast of Canada and neighboring American states, the resident great blue heron subspecies, Ardea herodias fannini, commonly nests in colonial aggregations high in spindly alder trees. During spring, the colony is a cacophony of noise from adult birds and chicks, while the air is ripe with the smell of feces, rotting fish and carcasses of chicks displaced by siblicide. So it was unsettling in late June of 1987 to enter a once thriving colony at Crofton on Vancouver Island to find a silent place devoid of herons. The residual smells of the heronry still mingled with the equally pungent odors of a kraft pulp mill. The occasional calls of corvids and gulls broke the silence, while trucks rumbled along the gravel causeway to and from the mill. The truck noise and the smell of the cooking and bleaching of wood were a reminder that viewed from above, the heronry was on a small island of green beside a massive industrial complex. Data from previous years showed that heron eggs from this site contained some of the highest concentrations of chlorinated dioxin contaminants reported for eggs of birds. Given that the mill was discharging large amounts of chlorinated waste into the local marine environment, it did not take a lot of imagination to wonder if there was a connection with the silent colony. The failure of that heron colony provoked a series of investigations that continued for the next ten years. Working together, biologists and chemist used ecological, toxicological and chemical analytical methods to investigate the health of wildlife and to address the possible role of persistent pollutants from pulp mills and other forest industry activities. Those results were then used by engineers, regulators and, industry officials to devise solutions to the contamination problems and to monitor their effectiveness.

Abstract The exposure and effects of wildlife to persistent pollutants from forest industry sources were studied over the period 1986–2008 in British Columbia, Canada. Elevated concentrations of specific polychlorinated dibenzo-*p*-dioxin and furan (PCDD/PCDF) congeners were measured in a variety of aquatic and predatory birds and mustelid mammals, and related to sources including chlorine bleaching of wood pulp and use of chlorophenolic herbicides. Exposure was correlated to biochemical, physiological, and morphological variables in various species, and to reproductive success in great blue herons (*Ardea herodias*) and bald eagles (*Haliaeetus leucocephalus*) at specific study sites impacted by pulp mill effluents. From the late 1980s into the early 1990s, changes to the bleaching process and regulatory restrictions on chlorophenol use produced significant reductions in ambient contamination and wildlife exposure to PCDD/Fs, as well as improvements in physiological responses. My experiences with both the science and the regulatory processes are described, along with a reassessment of the wildlife effects data and some consideration of the lessons that might be learned from this work.

Introduction and Background

During the 1950s and 1960s millions of poultry in the USA died from a condition referred to as chick-edema disease (Friedman et al. 1959). Following many years of scientific investigation, ‘dioxins’, primarily the compound, 1,2,3,7,8,9-hexachlorodibenzo-*p*-dioxin (HxCDD), were identified as the cause of that disease outbreak

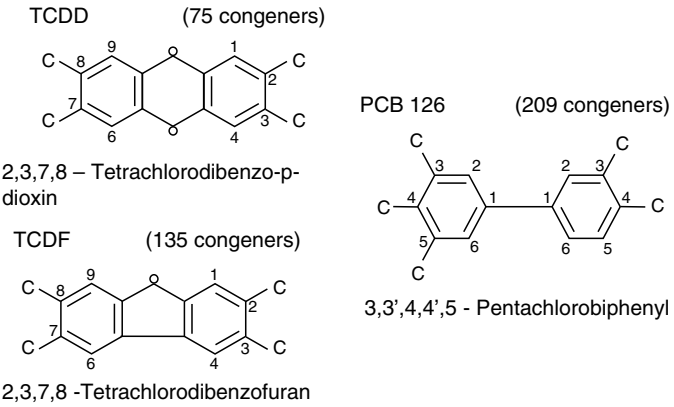

Fig. 2.1 Structure of 2,3,7,8-TCDD and related polychlorinated dibenzofurans (PCDFs) and polychlorinated biphenyls (PCBs). Nomenclature is based on presence of chlorine atoms on the molecule at sequentially numbered carbon atoms. For PCDDs and PCDFs the carbons are numbered in a clockwise order around the molecule. For PCBs, each ring is numbered separately, using a *prime* (′) designation for the second ring

(Higginbotham et al. 1968; Firestone 1973). The structure of 1,2,3,7,8,9-HxCDD can be deduced from Fig. 2.1, which shows the generic polychlorinated dibenzo-*p*-dioxin (PCDD), dibenzofuran (PCDF), and the related polychlorinated biphenyl (PCB) molecules. PCDDs and PCDFs were contaminants in the widely used chlorophenolic biocides, particularly penta- and tetrachlorophenol. Chlorophenolics along with their dioxin contaminants had entered the poultry food chain in fatty acid feed supplements obtained from waste oils and fats produced by the hide-tanning industry, where they had been used as preservatives.

By the early 1970s, the word 'dioxin' began to diffuse into the broader consciousness as something to be vaguely fearful of, something akin to a plague or other disease. *Silent Spring* (Carson 1962) and the subsequent dissemination of the book's information and had created public awareness that, like microbes, agricultural and industrial chemicals could contaminate air, water, and food. Such wider implications of technological change and the social perspectives have been referred to by some writers as the "Risks of Modernity" (Beck 1992).

Further comprehension of the potential hazards posed by dioxins began with the evidence they were formed during the manufacture of the widely used phenoxy-acid herbicides, 2,4-dichlorophenoxyacetic acid (2,4-D) and 2,4,5-trichlorophenoxyacetic acid (2,4,5-T). Agent Orange, a mixture of 2,4,-D and 2,4,5-T, became synonymous with dioxin. An estimated 400 kg of 2,3,7,8-TCDD was sprayed by the US military onto the forests of Indochina as an impurity in the 20 million kg of 2,4,5-T applied as the Agent Orange chemical warfare agent (Huff and Wassom 1974). Concerns over the impact on health of Vietnamese people, particularly children, and the American Vietnam War veterans involved in the application, as well

as the Vietnamese ecosystem continue to this day (Minh et al. 2008). During the 1970s, dioxin contamination associated with chlorophenolic manufacturing and disposal sites and with industrial accidents in Times Beach Missouri (Powell 1984), Seveso Italy (Fanelli et al. 1980a, b) and Love Canal in the USA drew increased public attention to dioxins (Pohl et al. 2002).

The Great Lakes Story

This narrative really begins during the early 1970s and with the colonial waterbirds of the North American Great Lakes. There were reports at that time of unusual reproductive problems and anomalies at colonies of cormorants (*Phalacrocorax* spp.), gulls (*Larus* spp.) and terns (*Sterna* spp.) nesting on the Great Lakes of Ontario and Michigan. At some islands on eastern Lake Ontario, nests were abandoned during mid-breeding season; eggs contained dead embryos, often with deformities. At other colonies, nests contained chicks also with various deformities, particularly of the bill, eyes, and feet (Gilbertson 1974, 1975). Failed eggs were salvaged during nest visits, and Canadian Wildlife Service biologists, such as Michael Gilbertson, aware of similar scenarios at seabird colonies in Britain, looked for a laboratory to analyze the eggs for residues for industrial and agricultural chemicals. Gilbertson worked with Lincoln Reynolds at the Ontario Research Foundation to adapt and apply methods for chlorinated hydrocarbons, such as DDE and PCBs for the analysis of wildlife tissues (Gilbertson and Reynolds 1972). Mean PCB concentrations, using methodology of the time, and expressed as a ratio of two commercial Aroclor[1] mixtures 1254:1260, were much higher than were being reported elsewhere. Eggs of herring gull (*Larus argentatus*) had 142 μg/g total PCBs at a colony in Lake Ontario and 92 μg/g, both on a wet weight basis, at a Lake Michigan colony (Gilman et al. 1977).

In 1975, the Canadian Wildlife Service opened the National Wildlife Research Centre on the grounds of a former agricultural research facility in Hull, Quebec. The problems with fish-eating birds and other wildlife on the Great Lakes had attracted wide public and political attention, which helped to shift the paradigm defining the Great Lakes Water Quality Agreement. The governments of Canada and the USA began to place priority on persistent toxic substances, rather than the original emphasis on eutrophication (IJC 1978). There was new funding to

[1] Aroclors are technical mixtures of PCBs manufactured by Monsanto Corporation from the 1930s to the 1970s. Those mixtures of PCB congeners were named according to their chlorine content, e.g. Aroclor 1254 contains 54% chlorine by weight and Aroclor 1260 contains 60%. Quantification of PCBs in environmental samples posed a challenge to analytical chemists for many years. Until analytical standards became widely available in the mid 1980s, chemists used one or two major chromatographic peaks considered representative of the major Aroclor mixtures to estimate PCBs on an Aroclor basis.

address the problem of environmental toxicants in the Great Lakes and elsewhere in Canada. New research and technical staff were hired, many of who focused on the Great Lakes. However, the establishment of cause-effect linkages between the observed health problems and specific compounds or groups of compounds proved to be challenging. During the period when signs of toxicity were overt, egg contents of fish-eating birds contained elevated concentrations of a complex mixture of halogenated aromatic contaminants in addition to PCBs, including DDTs, mirex, hexachlorobenzene and other compounds, later discussed in a paper by Peakall and Fox (1987) and examined retrospectively by Hebert et al. (1994).

Given the findings of reproductive failure among the fish-eating birds of Lake Ontario, particularly the mortality of embryos and the presence of deformed chicks, the presence of potent toxicants such as 2,3,7,8-TCDD in the Great Lakes food chains had been hypothesized by Michael Gilbertson, but could not be established with analytical methods employed at that time (Bowes et al. 1973). By the early 1980s, advances in analytical methodology and the availability of high resolution mass spectrometry combined with gas chromatography (GC/MS) enabled the quantification of PCDDs and PCDFs in tissue samples at toxicologically significant concentrations, such as the low parts per trillion range or less than 10 pg/g. A method was developed and eggs were analyzed from colonies of herring gulls in each of the Great Lakes, which demonstrated that PCDDs and PCDFs were present in food-chains from all the lakes, but with hotspots evident in Lakes Ontario, Michigan, and Huron. The temporal nature of the dioxin contamination was made possible by retrospective analysis of herring gull egg samples stored in the Canadian Wildlife Service National Specimen Bank, also one of the earliest demonstrations of the value of environmental specimen banks (Elliott 1984; Elliott et al. 1988a). The results showed that in 1971 eggs from a colony in Lake Ontario contained mean concentrations of 2,3,7,8-TCDD > 1,000 pg/g, which had decreased to about 100 pg/g by 1980 (Stalling et al. 1985). At the time of that work, the data base was very limited for comparative toxicity of dioxins in birds. Work on chickens found that LD_{50}s for 2,3,7,8-TCDD injected into chicken eggs were as low as 200 parts per trillion (pg/g, Verrett 1970).

Casting a Wider Net

Dioxins were present in wildlife from the Great Lakes, but what about other Canadian ecosystems? In 1983, the new high resolution GC/MS method was applied to a survey of wildlife samples from across Canada available from the National Specimen Bank (Norstrom and Simon 1983). Included in that survey were eggs of the Great Blue Heron collected from a colony breeding on the grounds of the University of British Columbia, and which foraged in the nearby Fraser River estuary. Along with the usual mix of halogenated contaminants, such as PCBs and organochlorine insecticides, those heron eggs contained 50 pg/g (wet weight) of

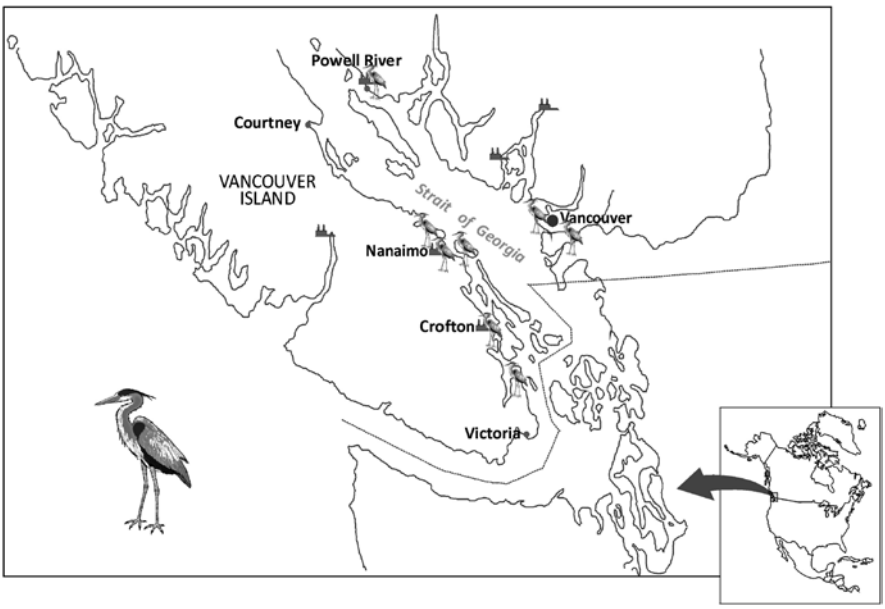

Fig. 2.2 Map of the Strait of Georgia region on the Pacific coast of Canada showing locations of pulp mills and heron colony study sites

2,3,7,8-TCDD. That amount was considered surprising, given the prevailing conception at the time that ecosystems on the west coast of Canada were relatively pristine, at least in comparison to the heavily industrialized Great Lakes. To follow up on those results, in 1983, great blue heron eggs were collected from a number of breeding colonies around the southern Strait of Georgia region on the coast of British Columbia (Fig. 2.2). The survey included two colonies on or near Vancouver Island, one on Gabriola Island located only a few kilometers across from a large bleached-kraft pulp and paper mill and other associated forest industry activities at Harmac near Nanaimo and a colony near Crofton, located on a small island immediately adjacent to a large bleached-kraft mill. The results continued to surprise and are summarized in Fig. 2.3.

The data from 1983 were interesting, particularly the high concentrations of 1,2,3,7,8-pentachlorodibenzo-*p*-dioxin (PnCDD) and 1,2,3,6,7,8-hexachlorodibenzo-*p*-dioxin (HxCDD) in the eggs from the Crofton colony. Environment Canada biologists with support from the Department of Fisheries began to collect fish, crab, and other marine organisms around the contaminated heron colonies (Harding and Pomeroy 1990). At that time, although there had been some speculative writing and proposals, there were no published links between production of wood pulp and formation of dioxins. The putative links between the dioxin contamination of the heron eggs and the forest industry had focused on the use of chlorophenolic biocides. Chlorophenols were widely used at that time in the range of millions of kilogram per year in British Columbia as wood preservatives (PCP, pentachlorophenol) and

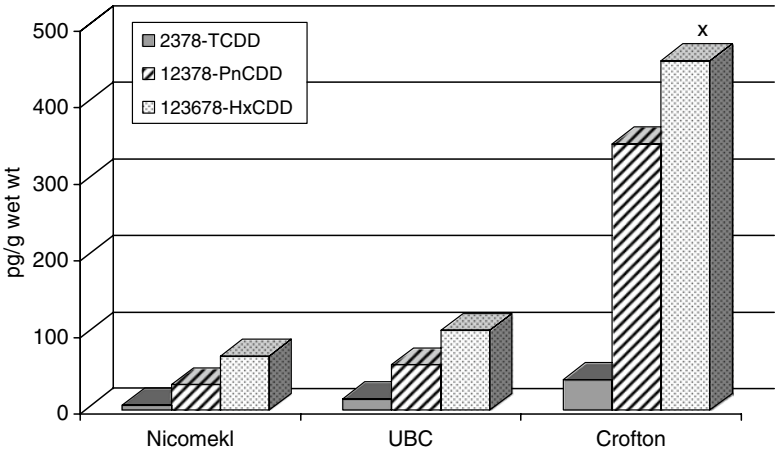

Fig. 2.3 PCDDs in eggs of great blue heron collected in 1983 from colonies around the southern Strait of Georgia, British Columbia, Canada

anti-sapstain agents (TCP, tetrachlorophenol) considered necessary to stop fungal staining of wood piled outside during the cool wet winters.[2] In 1984, the contaminants biologist, Phil Whitehead, took an acting management position and as a result there was no one in biologist position to undertake or supervise any follow-up field work that year.

In November of 1984, there was a federal election in Canada, the government of the day was defeated and a new one under Brian Mulroney came to power. Mdm Suzanne Blais-Grenier was appointed Minister of the Environment, where she made cuts to the departmental budget, particularly the Canadian Wildlife Service. The Research and Interpretation Division of the Canadian Wildlife Service was eliminated along with approximately 25% of the fulltime positions. Those budget reductions affected many projects and virtually all of the regional toxic chemical biologists across Canada, including the Pacific and Yukon Region. In Ottawa, the toxic chemicals program staff at the National Wildlife Research Centre were not made redundant, although most of their colleagues involved with research on animal health issues, including veterinarians, pathologist, and parasitologists were given 90 days notice of job termination. For months, the building was threatened with closure and transfer of the staff and facilities to the Canada Centre for Inland Waters in Burlington, Ontario. Needless to say, those changes

[2]Chlorophenolic biocides refer to the penta- and tetrachlorophenols widely used in many commercial applications to preserve wood and the U.S. Pacific Northwest and British Columbia to prevent mold and slime formation on lumber stored outside during the wet winters. Chlorophenols are unavoidably contaminated with a range of polychlorinated dioxins and furans; however, certain congeners are considered indicative of chlorophenol sources. See for example: Elliott et al. 1998b, b paper on ospreys for background references.

meant that very little new research was undertaken in 1985. However, by the following year, the budgetary and staffing situation had stabilized, and we developed a plan to study the dioxin contamination of the west coast herons.

The proposed focus of the work was the breeding biology of the herons at Crofton, and other sites in relation to the contaminant exposure. A former interpretive biologist, Rob Butler, who had been affected by the 1984 cuts to that program, was interested in undertaking a PhD as a work–study assignment. Thus, in the late winter of 1986, Butler began his study of heron biology.

At that time little was known about the ecology of the west coast subspecies of the great blue heron. There was a need for new techniques to assess breeding success and study other important topics such as diet and feeding behavior. We needed the most basic information about the local population, such as how many heron colonies were there on the south coast of B.C., where were they located in proximity to pollution sources, what was normal reproductive success, what factors could contribute to colony establishment, failure and relocation? Much of the new findings were later recounted in a book on the great blue heron (Butler 1997).

Eggs collected in 1986 confirmed the results obtained 3 years earlier, that the Crofton herons were exposed to relatively high concentrations of PCDDs and PCDFs, and in a particular pattern of the tetra:penta:hexa 2,3,7,8-subtstituted congeners Fig. 2.3. The estimated breeding success of the 64 nesting heron pairs was about 1.24 chicks per active nest, comparable to the rates measured at another dioxin contaminated colony in the Fraser River estuary (UBC) and a putative reference colony on the Nicomekl River (Harris et al. 2003a). Thus, we tentatively concluded that during the 1986 breeding season the dioxin contamination was not preventing the herons from producing what seemed to be a normal complement of fledglings.

We initially planned a 3-year study of heron reproduction and contamination at Crofton and three other colonies. The scene at the heron colony in the spring of 1987 is partially described in the introduction to the chapter. Of the estimated 200 or so eggs laid earlier by the female herons, many still lay in the nests and others were on the ground beneath the colony. Most had been pecked open and the contents eaten, probably by crows. Rob Butler was the first to enter the colony in April of 1987 and find it abandoned (Fig. 2.4). He returned immediately by car ferry across the Strait of Georgia to the Canadian Wildlife Service office in Delta. Phil Whitehead had a private pilot's license and owned a small airplane. Concerned about losing all the eggs and, therefore, evidence, he called a professional tree climber and asked him to meet them at the local Boundary Bay airport in Delta. The three of them then flew immediately to the nearest airport at Nanaimo, rented a car, and drove to the site of the Crofton heronry.

Whitehead and Butler salvaged a number of intact eggs and the contents were removed and couriered to the National Wildlife Research Centre. In the laboratory, the eggs were analyzed for chemical clues related to the colony failure. Later that summer the results came back and the concentrations of 2,3,7,8-TCDD had increased threefold from 70 to 210 parts per trillion. Interestingly, TCDD also increased about twofold over that same period in the eggs of herons at the UBC colony (Fig. 2.5).

Fig. 2.4 The bleached kraft pulp mill at Crofton, B.C., spring of 1990. The heronry was located on the island in the right foreground. Just to the right of where the causeway road bends to the left can be seen the viewing tower used to observe the herons. The new secondary treatment system was under construction at the time and is evident from the half finished tanks on the foreshore below the chip piles. The effluent pipe can also be discerned snaking under the water from near those tanks

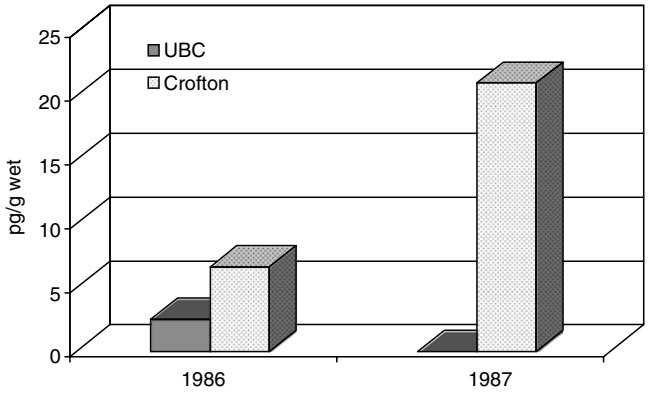

Fig. 2.5 Change in mean concentration of 2,3,7,8-TCDD in heron eggs at Crofton and UBC colonies between 1986 and 1987. Sample size was ten eggs in all cases (Elliott et al. 1989)

The Forensic Phase: I. Tracking the Sources

The steep increase in concentrations of TCDD in the eggs salvaged from Crofton forced us to confront two inter-related problems. Firstly, to reduce or ideally eliminate the contamination, we had to track and identify the source(s). Progress on

source identification required better information on the environmental chemistry of the PCDD and PCDF compounds, and on the feeding ecology of the herons. We also had to engage in further dialog with regulatory officials and engineers knowledgeable about pulp mill operations and use of chlorophenolic compounds. Secondly, we needed to investigate whether the TCDD increase was coincident or causally linked with the failure of the colony. Thus, we had to understand more about the general toxicity of the dioxin and furan contaminants in birds, particularly herons, and dose-response relations between dioxins and various aspects of reproduction, ultimately whether there was the potential to cause reproductive failure at the colony.

It now seems obvious in hindsight that cooking wood, a ready source of complex organic molecules, at high temperatures with elemental chlorine would lead to the production of complex chlorinated byproducts. However, at the time, there was no clear published data linking contamination of the ambient environment of a pulp mill with significant elevated concentrations of TCDD and TCDF. Canadian and international scientists and regulators concerned with dioxin sources were focused on waste incineration, chlorophenols, and landfills (Czuczwa and Hites 1984; Interdepartmental Committee on Dioxins 1985), and on atmospheric transport (Czuczwa et al. 1984). Whereas, we knew that the tetra- and penta-chlorophenols were heavily used as wood preservatives on the B.C. coast, they had mainly been linked to contamination with higher chlorinated PCDDs and PCDFs (Bright et al. 1999; Macdonald et al. 1992), not 2,3,7,8-TCDD or -TCDF. During the early 1980s, greater concentrations of PCDDs in human adipose tissue samples from British Columbia relative to elsewhere in Canada had been attributed to chlorophenol use (Ryan et al. 1985). Those findings and other reports of exposure and contamination of people, even industrial workers, also had been down played by some authors as of little consequence for human health (Tschirley 1986).

By March 1986, Canadian regulators were aware of substantial 2,3,7,8-TCDD residues in fish sampled in the international Rainy River drainage in Northern Ontario.[3] A sludge sample from the upstream bleached kraft pulp mill at International Falls, Minnesota contained 414 pg/g of 2,3,7,8-TCDD. By May of the same year there was high level concern at the US EPA that PCDD contamination of effluents and sludges from the pulp and paper industry could be a widespread and generic problem. Those concerns centered on the practice of applying sludges as fertilizer on forestry and agricultural land (Swanson 1988). PCDD contamination of sediments from a Wisconsin river had already been reported (Kuehl et al. 1987). In response, industry had sponsored the avian egg injection studies to assess the risk of PCCD/F contamination of forest fertilizer sludge to wild birds (Thiel et al. 1988). In February 1987, shortly before the colony failure at Crofton, a U.S. National Dioxin

[3] Memo from K. Shikaze, Regional Director, Ontario Region, Conservation and Protection Branch, Environment Canada to P. Higgins, Director General, Environmental Pollution and Protection Division, Environment Canada, Ottawa, March 12, 1986 on the subject "Dioxins – Pulp and Paper Mills". Attached is a memo from T. Tseng, Investigations of sources of dioxin in pulp and paper mills, dated March 10, 1986.

Study reported that PCDDs and PCDFs exceeded background concentrations in fish collected downstream of a number of pulp and paper mills, where no other sources of dioxins had been identified (Amendola et al. 1989). Greenpeace also produced a report in 1987 criticizing government and industry for not releasing information on dioxin contamination from the pulp and paper industry (Van Strum and Merrell 1987). Suddenly, everything was coming together fast, and it was increasingly clear that pulp mills were associated with the specific formation of 2,3,7,8,-TCDD and -TCDF.

As government scientists located in a small regional wildlife centre, we were in no isolated ivory tower. As awareness of the issue widened, political and economic factors came increasingly into play. We tried to focus on the intricacies of dioxin chemistry and toxicology interfaced with heron population and foraging ecology, while trying to absorb explanations on how chlorophenols were used and pulp mills operated. Meanwhile, we went to numerous meeting with managers and officials from our own and other departments, prepared summary and briefing notes for parliamentary question period, and answered questions and requests for interviews and presentations from the media and the public. As with most similar problems, the questions distilled down to the main issue of the significance of the problem in relation to costs of reducing the release of pollutants to the environment. That quickly became a public and political debate waged in many forums and with many competing voices.

Throughout that period there was considerable public pressure on the government and industry, evidenced by the widespread media coverage. Much of the public had grasped the idea that dioxins were not something that you wanted in your bacon or sushi, or even washing around in the water at your local beach. In British Columbia the publicity escalated rapidly after January 5, 1988 when Greenpeace released data on PCDD and PCDF contamination of sediments collected near the Harmac bleached kraft pulp mill at Nanaimo. Consistent with the importance of the forest industry to the provincial economy, the Crofton herons had already received full color press on the front page of the Vancouver Sun. In 1989, data on the increasing trend of 2,3,7,8-TCDD in herons at the UBC colony was featured in national radio and television coverage. Lobbyists for industry and their political allies countered in the media, hiring respected scientists such as Bruce Ames to speak at industry sponsored forums. In media reports, Ames contended there was no elevated threat of cancer from the dioxins in pulp effluent and products, but missed the point that wildlife concerns were not driven by cancer, but by affecting reproductive and basic physiological processes.

The Canadian pulp and paper industry did respond quickly to evidence that their operations were producing and releasing dioxin contamination into local marine and aquatic environments. PAPRICAN (Pulp and Paper Research Institute of Canada) researchers identified the role of elemental chlorine bleaching in TCDD and TCDF formation; subsequently, chemical defoaming agents were also found to be a significant source of TCDF in particular (Berry et al. 1989). The specific pattern in wildlife from the Strait of Georgia, where 1,2,3,7,8-PnCDD and 1,2,3,6,7,8-HxCDD were so prevalent (Elliott et al. 2001a), was traced to the pulping of

chlorophenol contaminated wood chips. Luthe et al. (1993) later reported how HxCDDs in particular were produced from a condensation reaction when polychlorinated phenoxyphenol impurities in anti-sapstain treated wood chips were cooked in a pulp mill digester.

It was clear that the Crofton mill in particular had a dioxin problem. A bleached kraft and chemi–thermo–mechanical pulp mill had operated since 1935 at Crofton, processing a variety of local wood species, and discharging liquid effluent into adjacent Stuart Channel. Effluents were discharged under 1971 federal regulations, which exempted mills, such as Crofton, built before the implementation of those regulations. In the spring of 1988, the mill began to test their feedstock chips for chlorophenols, and to divert non-compliant materials. Later that year, they converted first their 'A' bleach plant, and then their 'B' plant to 50% chlorine dioxide, which reduced production of the most toxic, 2,3,7,8-substituted dioxins and furans (Berry et al. 1989). In October 1989, the mill began using defoamers reformulated with purified oils free of precursor dioxins and furans, as had been recommended by researchers at Paprican (Berry et al. 1989). By early 1990, the bleaching sequence was modified by introducing molecular chlorine first and chlorine dioxide last, also in response to the Paprican research.

Those process and product changes had rapid effects on contamination levels in the great blue herons, which are detailed in articles by Elliott et al. (2001a) and Harris et al. (2003a). Within a year of the changes at the mill and over the period 1989–1991, concentrations of 2,3,7,8-substituted TCDD, PnCDD, and HxCDD all dropped significantly. Concentrations of 2,3,7,8-TCDF in heron eggs also declined, although more slowly. We turn from chemistry to ecology to understand why the contaminants dropped so fast in the heron eggs. Diet studies had shown that herons ate mainly small fish between 1 and 2 years of age (Harfenist et al. 1993). Once the tap was essentially turned off, the mill effluents were cleaner, the active tidal and current movements flushed the marine system and the newly hatched fish were growing in a much cleaner environment than had the parent fish. Thus, it was not surprising that a reduced uptake of contaminants in herons quickly followed reductions in contaminant discharges.

Different dioxin congeners did show different patterns of decline, likely due to varying principal sources within and around the pulp mill at Crofton. Throughout the 1980s, the gradual decline of 1,2,3,6,7,8-HxCDD was consistent with the earlier theory that the pervasive contamination of the Georgia Basin with that compound originated from chlorophenol use, which was being regulated and declining by the mid-1980s. At Crofton, the major source of HxCDD in heron eggs was probably contaminated wood chips in the pulp-mill, but local sawmills may have also contributed to contamination. The increased contamination of eggs with TCDD and, to a lesser extent TCDF, in the early 1980s was consistent with increased production and sales of bleached pulp over the decade at that mill. The dramatic declines after 1990 were clearly linked to process changes, as pulp production continued to increase during that time. Because several changes to bleaching technologies and defoamer use were implemented within a short time span, it is virtually impossible to sort out which of those effected the most change in TCDD and TCDF production.

The Forensic Phase: II. Toxicology of Dioxins and Wildlife

By the time that chlorine bleaching of wood pulp was shown to cause formation of 2,3,7,8-substituted dioxins and furans, there had been some progress in understanding the basic toxicology of dioxins and related chemicals. The role of the cellular Ah or arylhydrocarbon receptor had been identified and widely investigated (Poland et al. 1976; Poland and Knutson 1982). The dioxin-like toxicity of some polychlorinated biphenyl (PCB) molecules had also been examined (Safe 1984). A scheme had been developed to rank the toxicity of the 75 PCDD, 135 PCDF, and 209 PCB congeners and produce a single number, the dioxin equivalent or TEQ, to estimate the toxicity of the complex mixtures commonly found in the environment (Bradlaw and Casterline 1979). The finding of a receptor led to a general theory of how dioxin-like chemicals caused toxicity, depicted in Fig. 2.6.

Following the 1987 failure of the Crofton heron colony and the increase in 2378-TCDD burden of the eggs, we initiated a toxicological study for the 1988 breeding season. The Crofton colony eventually failed again that year; however, partially incubated eggs were collected prior to the abandonment and at two other colonies, and transported to the Animal Science laboratories at the University of British Columbia. Using information on incubation conditions obtained from the San Diego zoo, the eggs were placed into incubators designed for chickens, and were tended carefully until they began to hatch. Under those controlled conditions in the laboratory, and

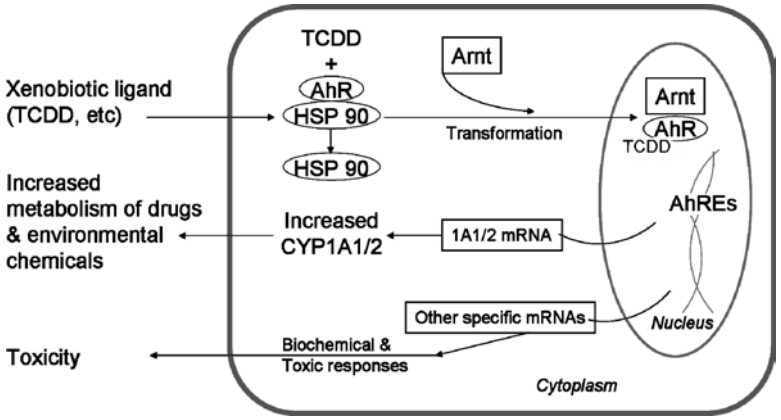

Fig. 2.6 Mode of action for dioxin-like chemicals at the cellular and molecular level. The xenobiotic ligand such as TCDD is able to move passively across the lipid rich cell membrane due to its fat solubility. Once inside the cell, it binds with the Ah-receptor in the process displacing a heat shock protein (HSP 90); it then further binds with the Arnt, a nuclear translocation protein, which facilitates transport into the nucleus where it interacts with responsive elements on the genome. A variety of messenger RNAs (mRNAs) are produced, which leave the nucleus and proceed to code for the production of various proteins, including cytochrome P450s (CYPs) such as CYP1A, involved in metabolism of xenobiotics, including contaminants and drugs. Similarly, other proteins are produced leading to various biochemical and toxic responses

Table 2.1 Result of great blue heron eggs incubated in the laboratory, 1988

Variable	Nicomekl	UBC	Crofton
Mean 2378-TCDD	8 pg/g	84 pg/g	170 pg/g
Fertility	100%	100%	100%
Hatchability	100%	92%	92%
Growth	Normal	Normal	Chicks significantly smaller than Nicomekl
Incidence of edema	0	15%	30%
EROD induction	Normal	Elevated	Significantly elevated over Nicomekl

thus without the influence of other factors that can affect the outcome of the eggs in the wild, it was clear that despite the dioxin burden, there were no significant differences in hatching success or fertility of the eggs among the different colonies (Table 2.1). Thus, a first and important element was determined; there was no overt toxicity to developing embryos. The colony was not failing because eggs were not hatching. There were, however, other physiological and biochemical effects in the heron embryos, which correlated significantly with TCDD exposure. In that study and in further assessments during 1990–1992, there were statistically significant correlations between concentrations of 2,3,7,8-TCDD and of dioxin toxic equivalents (TEQs) in eggs with activity of a liver enzyme referred to as ethoxyresorufin-0-deethylase or EROD, a known marker of exposure to dioxin-like chemicals (Bellward et al. 1990; Sanderson et al. 1994a, b; Harris and Elliott 2011). Exposure to 2,3,7,8-TCDD also correlated with several measures of chick growth, such as body, organ, and bone weights (Hart et al. 1991). Some chicks at Crofton and UBC had jelly-like deposits of fluid or edema under the skin of the neck, breast and leg regions, and one chick from UBC had a crossed bill. No gross abnormalities were observed in chicks from the reference colony at Nicomekl River.

The findings from those studies, including similarities and differences compared to what was reported in the Great Lakes water birds, often referred to as GLEMEDS (Great Lakes Embryo Mortality Edema and Deformities Syndrome, Gilbertson et al. 1991) were examined in more detail in the publications cited above and later in Elliott et al. (2001a, b), and Harris et al. (2003a, b). Among other items of importance, we learned that great blue herons, along with the other species studied, cormorants, bald eagles and ospreys, are not particularly sensitive to dioxin-like toxicity. In 1988, the dioxin-TEQs in heron eggs chicks averaged 370 pg/g at Crofton and 360 pg/g at UBC. Those TEQ tissue loads were exceeded in eggs collected from Crofton in all of the years up to 1991. At UBC, they were exceeded in 1985 and 1989. Comparable toxic burdens were also measured in 1989 at a second colony near near Crofton. We suspect, given the statistical relationships between TEQs and developmental effects in chicks, that there was at least EROD induction and possibly growth effects in chicks at those other sites. With LD_{50}s in the range of 200–250 pg/g, TEQs (Harris and Elliott 2011), chicken embryos would have died at the exposures commonly found in avian predators near pulp mills during the 1980s and to the mid-1990s.

In 1988 there was also an attempt to do an egg swap study. That approach had been applied successfully in contaminant studies of osprey (*Pandion haliaetus*), and involved switching eggs between locations (Wiemeyer et al. 1975; Spitzer et al. 1978; Woodford et al. 1998). The objective is to separate effects 'intrinsic to the egg' such as infertility or direct embryo toxicity from those caused by parental behaviors such as failing to adequately incubate eggs. Egg swap studies are logistically complex at the best of times; however, the approach did not work for great blue herons. There were two main problems; the first was the tendency of herons at some sites to abandon nests if disturbed early in incubation. The second problem was the lack of synchrony in breeding at various colonies, likely due to variation in local availability of food resources (Butler 1997), which made it very difficult to successfully switch eggs among colonies.

Overall, the productivity of great blue herons in the Strait of Georgia, although variable, was similar to that recorded for colonies in Washington and Oregon (Thomas and Anthony 1999). Poor reproductive success, where less than one chick was produced per active nest, occurred at Crofton (5 years), Holden Lake (1 year), Powell River (3 years), UBC (1 year), and Victoria (2 years). The first three colonies (comprising 75% of the cases) were located within 10 km of pulp mills, while the latter two colonies were located within urban centers. Breeding at Crofton and Powell River failed completely in several years, and none of the colonies surveyed around those mills were in apparent use from 1996 to 1999 (Moul et al. 2001). The poorest effort recorded for a more remote colony in our subset of data was one young per active nest (2.11 young per successful nest); however, colony failures have been recorded in rural sites, including Sidney Island in 1989 and 1990 (Butler 1997; Moul et al. 2001). Reproductive success is affected by a variety of ecological as well as anthropogenic factors; nonetheless, there is an apparent trend of lower nest success of great blue herons near areas of significant industrial and urban pollutant sources.

Assessments of Other Wildlife

Herons were not the only species on the BC coast using habitat affected by pollution from forest industry activities. Beginning in the early 1970s, eggs of two species of *cormorant*, the widely distributed, double-crested cormorant, *Phalacrocorax auritus*, and the smaller Pacific coast species, *P. pelagicus* or the pelagic cormorant, were monitored for the exposure to persistent contaminants. Double-crested cormorant eggs contained on average 50% greater concentrations of 2,3,7,8-TCDD than pelagics, while being in turn about 30% less on average than heron eggs from the same locations (Harris et al. 2003a, b). Trends in cormorant eggs followed the same temporal patterns as were measured in herons, falling dramatically in the early 1990s at the forest industry dominated sites. Although cormorants and herons share many ecological niche characteristics, there are important differences, which contributed to overall lower dioxin burdens in cormorants. Most importantly, there are marked differences

in fine scale habitat use. Herons tend to feed in slow moving or even stagnant waters where particulate matter tends to settle out, often carrying adsorbed contaminants as compared to the open, faster moving waters where cormorants prefer to forage. Larger scale seasonal differences in feeding area are also likely important; a radio-telemetry study showed that double crested cormorants can move away from breeding areas, traveling in winter down into areas of Puget Sound (unpublished data).

We also conducted an artificial egg incubation experiment with double-crested cormorants. Hepatic EROD activity in hatchlings from three British Columbia colonies, including Crofton and a second pulp mill influenced colony, was significantly elevated as compared to hatchlings from a reference colony in Saskatchewan (Sanderson et al. 1994b). There were no gross abnormalities, edema, or changes in morphological measurements in hatchlings from the pulp mill colonies. Henshel (1998) did report that some hatchlings exhibited at least one type of brain asymmetry, possibly indicating neurotoxic effects of contaminants.

Waterfowl and waterbird populations from breeding sites across western North America overwinter in the Strait of Georgia and many other protected bays and estuaries of the west coast (Campbell et al. 1990). Because waterfowls are hunted for human consumption, their contamination by industrial and pulp mill effluents was a human health issue (Braune et al. 1999). There have also been recent conservation concerns for some North Pacific populations of eider and scoter species (Henny et al. 1995). A study of wood ducks (*Aix sponsa*) suggested that species, and possibly other waterfowl, were very sensitive to dioxin-like effects on deformities and reproduction (White and Segnak 1994), although that was not confirmed by later egg injection studies (Augspurger et al. 2008).

To address both conservation concerns and risk to human consumers, samples of seaducks and grebe species were collected in the late 1980s and early 1990s near pulp mills, and from reference sites, including the Yukon Territory. Sampling occurred in late winter or early spring and assumed exposure would be maximized after overwintering in the collection area (Elliott and Martin 1998). Generally, of all samples collected in 1989, those from near the bleached kraft pulp mill site at Port Alberni were the most contaminated with PCDDs and PCDFs (Vermeer et al. 1993). Piscivorous or fish-eating species, including western grebes (*Aechmophorus occidentalis*) and common mergansers (*Mergus merganser*), contained the greatest concentrations of all contaminants (Elliott and Martin 1998). Those samples also contained other chlorinated hydrocarbons such as DDE and PCBs. The only compound detected in all samples was 2,3,7,8-TCDF, although 2,3,7,8-TCDD was regularly present. International TCDD toxic equivalents (I-TEQs) in some western grebe samples were comparable to herons and cormorants (200–400 pg/g). Top predators, particularly bald eagles, resident near pulp mill sites on the British Columbia coast probably accumulated high levels of chlorinated hydrocarbons in part from feeding on fish-eating birds, including grebes and seaducks (Elliott et al. 1996b, c).

The greater liver PCDD/F concentrations in 1989 at Port Alberni prompted the Canadian federal health agency (Health Canada) to issue an advisory limiting the human consumption of common merganser liver to <50 g/week and of surf scoters liver to <75 g/week (Whitehead et al. 1990). Waterfowl samples were collected at

the end of winter, potentially following maximum exposure to pulp mill effluents; therefore, it was supposed that by the time of the important fall hunting season, and a summer on less contaminated breeding grounds, tissue contaminant levels would be much lower, and less of a concern for human consumers.

Among the resident birds on the Pacific coast, the *bald eagle* is the largest predatory species. Eagles are generally considered to be top predators; however, we found that based on their stable isotope or $\delta^{15}N$ signature (Elliott et al. 2009), an independent measure of trophic level, that at least during the breeding season, eagles were similar to other piscivorous birds on the British Columbia coast. They appear to have been eating a variety of medium-sized forage fishes such as herring (*Clupea*), mackerel (*Scomber*), and midshipman (*Porichthys*) species (Gill and Elliott 2003; Elliott et al. 2005).

In 1990, I began a study of eagle biology in the context of contaminant exposure and effects on the British Columbia coast. In 1990 and 1991, eagle eggs from around Crofton and other pulp mill and reference sites had elevated PCDDs and PCDFs with patterns similar to those in other fish-eating birds (Elliott et al. 1996a). In 1992, we collected eggs for an artificial incubation study of the bald eagle. We measured significant hepatic EROD induction, and using molecular techniques found that specific toxicant metabolizing enzymes, including a cytochrome P450 or CYP1A-like enzyme was expressed (Elliott et al. 1996b). That year we also began to monitor productivity of eagles across a broad area of the British Columbia coast, and to use non-destructive blood sampling of nestling eagles, rather than egg collection. The results of the 4-year study revealed that nest success was significantly lower in the bald eagle nests within the dioxin fishery closure zone around the Crofton mill compared to nests outside that zone (Elliott and Norstrom 1998). However, productivity of bald eagles was also low at other areas of the coast that had low exposure to PCDDs and PCDFs. At those sites, such as areas of the west coast and north-eastern coasts of Vancouver Island, and the Queen Charlotte Islands, low production of young eagles was linked to low food availability during the breeding season (Elliott et al. 1998a).

Later we studied both contaminants and food supply more intensively in eagles from the Crofton area. At territories south of the mill, average productivity was 50% lower than territories north of the mill. At individual nests south of the mill, reproductive success was correlated to lower rates of prey delivery in comparison to nests north of the mill. That in turn was linked to topographic differences, which limited feeding areas south of the mill, compared to the habitat further north where there were greater shallows and mudflats. However, contaminants, particularly PCDDs and PCDFs also were higher at the territories south of the mill, and we recognized the potential for those contaminants to have acted in concert with food stress to affect eagle breeding success (Gill and Elliott 2003; Elliott et al. 2005). Most recently, we have shown that more than 30 years after regulatory bans on PCB use, that nestling bald eagles, similar to birds from many other locations, exhibit apparent thyroid hormone disruption caused by PCB exposure (Cesh et al. 2010).

Forest sector industrialization of the British Columbia landscape was not confined to the coast. Well before 1990, the major waterways of the interior of the province were dotted with numerous log sorts and lumber mills. At strategic

locations, integrated pulp and paper installations were constructed. Moving east in British Columbia, the moderating influence of the Pacific wanes while successive mountain ranges create a diversity of climate and habitats and with it changes in the wildlife community, and some key life history strategies. Marine species such as pelagic cormorants disappear, and species common to the coast, such as the great blue heron or the bald eagle, may now be forced to migrate to avoid subzero winters with ice cover of rivers and lakes. To expand our study of forest industry pollution to the interior, we needed a different approach and sometimes different species.

The osprey (*Pandion haliaetus*) is an obligate fish-eating bird of prey with a nearly worldwide breeding distribution. Like the bald eagle, the osprey was particularly sensitive to DDE-induced effects on eggshell quality, and populations in many parts of the world declined during the organochlorine era (Poole 1989). Exposure and effects of chlorinated hydrocarbon and mercury pollutants has been widely studied in this species (Ames 1966; Spitzer et al. 1978; Wiemeyer et al. 1988; Steidl et al. 1991). Many features of osprey life history make it a useful species for monitoring and research of contaminants (Grove et al. 2009).

Over a number of years, we studied ospreys breeding up and downstream of bleached-kraft pulp mills on the Fraser and Columbia River drainage systems of British Columbia, Washington, and Oregon. We learned that mean concentrations of 2,3,7,8-TCDD were significantly higher in eggs collected at downstream compared to upstream nests near pulp mills at Kamloops and Castlegar, British Columbia (Elliott et al. 1998b). We monitored a number of sites over time, and found that, like the coastal birds by the late 1990s, concentrations of 2,3,7,8-TCDD and -TCDF were significantly lower than in the early 1990s, consistent with changes in bleaching technology and evidence from sediments (Elliott et al. 1998b; Macdonald et al. 1998).

We found an unusual pattern of higher chlorinated PCDDs and PCDFs in many of the osprey eggs collected in that study, with highest concentrations from nests on the Thompson River, a tributary of the Fraser River, and contrary to expectations, concentrations were generally higher at nests upstream of pulp mills. That contamination was related to use of chlorophenolic wood preservatives by lumber processors based on patterns of trace PCDFs in eggs, essentially a fingerprint particular to that source, and significant positive correlations between egg concentrations of pentachlorophenol. It had been widely thought that octachlorodioxin (OCDD) had a very low capacity to bioaccumulate. Bioavailability of chemicals in food chains is largely determined by their tendency to move or partition between water (aqueous) and octanol (lipid) phases, called the octanol:water partition coefficient, and expressed as a logarithm, log K_{OW}. It was thought that OCDD and other higher chlorinated compounds had such a high log K_{OW} that it inhibited their capacity efficiently bioaccumulate (Segstro et al. 1995). However, the theory does not apply to some benthic or bottom feeding food chains where, for example, fresh water clams or aquatic insects appear to efficiently bioaccumulate higher chlorinated PCDDs and PCDFs (Fig. 2.7). A connection with the ospreys and such food chains was made from the work of fishery biologist, Don McPhail, from the University of British Columbia who showed that some subspecies of mountain whitefish, that he referred to as “Pinocchios”, because of their longer snouts, had adapted to specialize on benthic invertebrates (McPhail and Troffe 1998). Thus it appeared that those

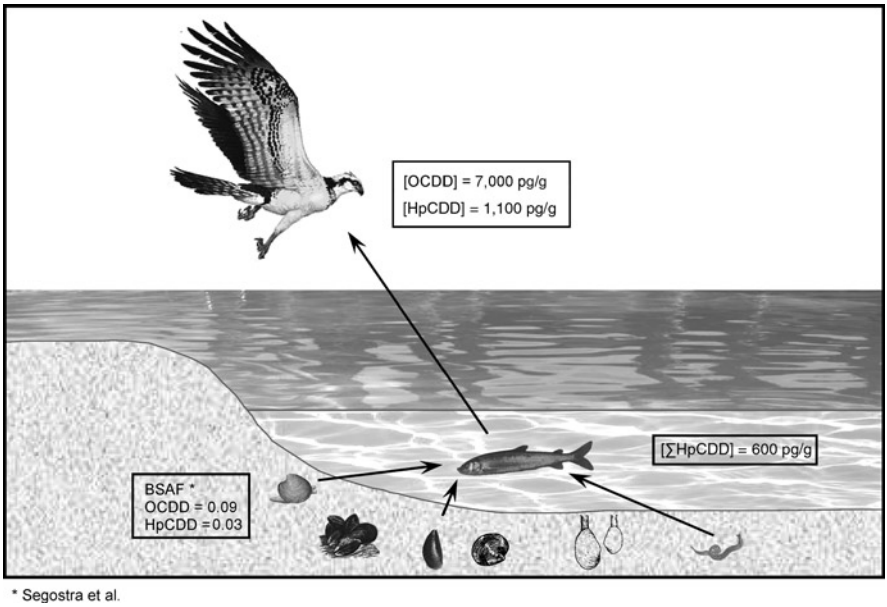

Fig. 2.7 Ospreys accumulated unusually high concentrations of supposedly non-bioaccumulative compounds, such as OCDD. Some ospreys probably fed on 'pinnochio' forms of the mountain whitefish, adapted to feed on the benthic filter-feeding invertebrates that are able to accumulate such chemicals

bottom feeding whitefish were accumulating OCDD and other higher chlorinated compounds (Nener et al. 1995) and transferring them to ospreys.

Similar to the studies with other birds, in 1995 and 1996, we collected 54 osprey eggs at seven sites along the Fraser and Columbia Rivers in BC, Washington and Oregon, USA and brought them to the laboratory for incubation (Elliott et al. 2001b). That work was done five years after the pulp mills had substantially reduced dioxin and furan contamination of effluent, and may have been in factor in our finding no differences in contaminant concentrations between eggs which hatched and those that did not. As with the other fish-eating birds, EROD and related proteins were induced with exposure to toxic equivalents and PCBs. Tissue concentrations of retinols or vitamin A compounds also varied among sites and correlated positively with concentrations of TEQs and PCBs, indicating there were subtle effects of those chemicals on the physiology of ospreys.

Although fish-eating birds are among the most visible top predators in temperate aquatic food webs, they may not be indicative of contaminant exposure and effects on other wildlife, such as mammalian predators. Pacific killer whales in the Strait of Georgia (*Orcinus orca*) have substantial body burdens of persistent contaminants, particularly PCBs (Ross et al. 2000). Harbor seals (*Phoca vitulina*) collected in the mid 1990s near the Strait of Georgia pulp mill sites showed the same distinctive patterns of PCDDs and PCDFs, but body burdens were lower than exhibited by their avian counterparts (Ross et al. 2004; Addison et al. 2005). *Mink* (*Mustela vison*) and

river otter (*Lontra canadensis*) have a similar trophic status, but their home ranges are relatively small and seasonally more constant compared to their larger marine counterparts. In the 1960s, minks were identified as particularly sensitive to PCBs, when a connection was made between mink reproductive failure and diets of contaminated Great Lakes fish (Aulerich and Ringer 1977; Smit et al. 1994). Declines of European otter (*Lutra lutra*) populations (Macdonald and Mason 1988; Kruuk and Conroy 1966) and North American mink populations (Henny et al. 1981) have been linked tentatively to elevated environmental PCB concentrations. In a later study, mink exposed via the diet to bleached kraft mill effluent had sublethal effects on immune function (Smit et al. 1994).

During the early 1990s, I worked with Chuck Henny of the US Geological Survey in Corvallis, Oregon to study semiaquatic mammals on the Fraser and Columbia Rivers. (Elliott et al. 1999) Using carcasses obtained from commercial trappers, we measured chlorinated hydrocarbon residue concentrations in livers of American mink and river otter collected from the Fraser River and Columbia River watersheds. For the most part, significant amounts of the two congeners 2,3,7,8-TCDD and TCDF were not found in mink and otter. International TCDD toxic equivalent levels in mink (31 pg/g) and otter (93 pg/g) from the lower Columbia River approached toxicity thresholds for effects on reproduction in ranch mink.

Elevated concentrations of higher chlorinated PCDDs and PCDFs, probably resulting from use of chlorophenolic wood preservatives, were found in both species. Most samples also contained other contaminants including DDE and PCBs, although there was substantial variability in patterns and trends among neighboring samples.

More recently we have used a nonintrusive technique to study river otter populations, by collecting their feces at communal latrine sites (Elliott et al. 2008). We found that otter feces collected in 1998 from pulp mill sites at Crofton, Nanaimo, and Powell River did not contain elevated TCDD, although one latrine near the pulp mills at Powell River still had higher TCDF present. Elevated concentrations of higher chlorinated PCDDs and PCDFs were found at some sites, particularly in Victoria Harbour and Nanaimo. Based on a fingerprint pattern of specific PCDF compounds, the source was attributed to use of chlorophenolic biocides, as we had found elsewhere and in other species, most likely by previous forest industry operations. Recently, we have combined molecular DNA genotyping techniques with contaminant analyses in feces to show movement and population structuring of river otters in the Victoria area (Guertin et al. 2009).

Forensic Phase: III. Reappraising the Toxicological Data

With the benefit of hindsight and new information what can we now conclude about causal relationships between the forest industry contaminants and health effects in wildlife? Significant exposure was clearly evident; in habitats impacted by forest industry effluents, predatory and fish-eating wildlife accumulated much greater

PCDD and PCDF concentrations than at reference sites. The only exceptions were the mink and otter, which did not appear to forage extensively on the more contaminated main stems of those rivers (Elliott et al. 1999).

Local populations of three coastal bird species (herons, cormorants, and eagles) exhibited measurable and significant biochemical responses, including increased activity of the enzyme(s) measured by the EROD assay and in some cases specific induction of CYP1A-like enzymes. Interpreting the ecological or even basic toxicological significance of induction of CYP1 enzymes remains complex and somewhat unresolved (Nebert et al. 2004). In avian species, we now know that there are at least two forms of the enzyme associated with the measured EROD response, as well as more than one form of the Ah-receptor (reviewed by Harris and Elliott 2011). It may be that the CYP1 induction measured in those various birds was simply an adaptive physiological response to xenobiotic exposure – with no toxicologically significant consequences. However, if viewed as a biomarker of an Ah-receptor mediated response, CYP1A induction also indicates that other physiological processes were affected. Effects on immune function, for example, appear to be Ah-receptor mediated, but are more difficult to measure reliably in the field, but have potentially greater significance to the overall fitness of the bird. In the ospreys, we initiated the laboratory incubation study because breeding success downstream of some mills, such as Kamloops, was lower than upstream and not explained by food availability or other discernible factors (Elliott et al. 2001a). However, by the time of that study, PCDDs and PCDFs had declined significantly in those systems. Nevertheless, we detected a CYP1A type response which correlated with exposure to PCBs.

The population level parameters, such as the failure of the Crofton colony during the years of highest PCDD and PCDF contamination, and the lower nest success of bald eagles within the fishery closure zone are confounded by other ecological factors, including disturbance, weather and food supply. Thus it is always difficult to relate cause and effect, which is no different, however, from the complexity facing human epidemiological work, and is not a reason for avoiding attempts to understand the system. In 1988 and 1989 we used a basic ecological technique of intensive observation of individual birds. Observation of the Crofton heron colony during two breeding seasons revealed periodic disturbance and subsequent predation and scavenging by crows (*Corvus caurinus*) or ravens (*Corvus corax*) of the vacated nests. The proximal cause of the abandonment appeared to have been that disturbance, either from humans or bald eagles (Norman et al. 1989; Moul 1990). There is then the question of relative sensitivity of herons to disturbance, also not a simple topic. At some sites, herons are more easily provoked to abandon a breeding attempt, particularly early in the incubation cycle and before a lot of investment of time and energy. However, elsewhere, such as a colony located for years above the old Stanley Park zoo in Vancouver, herons bred successfully in trees overhanging the howler monkey cages (Butler 1997). The role of disturbance by bald eagles has also proven more complex than originally thought. Some authors considered the recovering bald eagle populations in the Pacific Northwest to pose a threat to long-term viability of heron populations by disturbance and predation at breeding

colonies (Vennesland and Butler 2004). In my experience, however, during the breeding season most eagle pairs consume and feed their young on fish (Elliott et al. 1998a, 2005). Recent studies also showed the eagle–heron relationship to be more complicated. Herons which nested in close proximity to eagle nests appear to obtain protection from other foraging eagles in return for some loss of chicks (Jones 2009). That finding is consistent with a so-called predator protection or 'sleeping with the enemy' hypothesis (Quinn and Ueta 2008).

There remains the potential for a negative relationship between PCDD and PCDF contamination and the sensitivity of the herons' to disturbance by humans or predators. Although the physiological mechanisms are not understood, some chlorinated hydrocarbon compounds have been associated with aberrant parenting behavior in avian species in the laboratory (Peakall and Peakall 1973; McArthur et al. 1983) and in some field situations (reviewed in: Harris and Elliott 2011). Thus, contaminants in incubating adult herons may have affected their nest attentiveness and subsequent susceptibility to disturbances.

In addition to the 'realtime' exposure and effects of dioxins on adult herons, another mechanism may have been in play. In the early 1990s, research with laboratory rodents showed that exposure to very low concentrations of 2,3,7,8-TCDD during early development, in the womb or from maternal milk, could have significant consequences for later sexual development particularly of male animals (Mably et al. 1992; Peterson et al. 1993). Subsequent studies with a variety of PCBs and polybrominated diphenyl–ether flame retardant compounds have corroborated those findings (e.g. Dickerson and Gore 2007). Overall there is a lack of such research on birds. Avian work has focused primarily on the possible estrogenic or anti-androgenic effects of early exposure to DDE (Fry and Toone 1981; Helander et al. 2002; Holm et al. 2006; Iwaniuk et al. 2006) rather than delayed responses to PCBs or PCDDs. However, some more recent laboratory work with avian models has shown later effects on reproductive and related endpoints from in ovo exposure to PCBs (Fernie et al. 2001; Hoogesteijn et al. 2008).

The embryos of herons, cormorants, eagles, and ospreys collected during the 1980s and 1990s near any of the bleached kraft pulp mills had PDDD and PCDF TEQ concentrations in the range of hundreds of pg/g. Although speculative, it possible that male birds exposed during early development to pulp mill derived dioxins, later suffered such neuroendocrine impairments with subtle effects on reproductive behaviors during mating or later during incubation There are repeated hints from the data, such as the consistent poor reproductive success of herons near Crofton and other mill sites, and of the more highly exposed bald eagle south of the Crofton mill.

The Regulatory Process

I have often wondered if the evidence for wildlife alone would have been sufficient cause for the pulp and paper industry to have spent any money, never mind hundreds of millions of dollars, to reduce output of chlorinated waste, especially

dioxins?[4] Perhaps it is a moot point, as the concerns for wildlife were only a part of the issue. Large areas of the coast were closed to commercial harvest of crab, shellfish, and fishes. The industry also faced consumer boycotts and 'Reach for Unbleached' campaigns. Regulators in many major markets in the USA and Europe were defining low limits on the amount of dioxins permitted in food contact products, such a coffee filters, diapers, and paper towels.

Involvement with the regulatory process meant many meetings with government and industry representatives to present and discuss the implications of our findings. Phil Whitehead and I made numerous presentations to technical and public forums. At the request of management, we published the first account of the heron contamination in a Canadian government technical forum (Elliott et al. 1988a, b). The contamination of herons at Crofton and by implication their food chain as evidenced by the fishery closures, and the intense publicity played some role in the pre-emptive changes made at that mill site, and soon after at all Canadian mills (see for example: Servos et al. 1996).

In 1992 the Pulp and Paper Regulatory Framework was made law, consisting of the *Pulp and Paper Effluent Regulations* (PPER) under the *Fisheries Act* and two regulations under the *Canadian Environmental Protection Act* (Government of Canada 1992). The PPER revoked and replaced an earlier set of regulations passed in 1971. The 1971 regulations were considered deficient in that they were legally binding only on new mills built after the regulation's November 2, 1971 promulgation date. That exempted many of the mills in British Columbia, such as Crofton, which were built before that date. The new regulations were designed to ensure that all mills were subject to regulatory requirements. The PPER set discharge limits for Biological Oxygen Demand (BOD) and Total Suspended Solids (TSS), and prohibited the discharge of effluent that was acutely lethal to rainbow trout, based on a standard assay.[5] The new regulations developed under CEPA specifically targeted the formation of chlorinated dioxins and furans during pulp bleaching. The *Pulp and Paper Mill Effluent Chlorinated Dioxins and Furans Regulations* prohibited the release of measurable concentrations of the 2,3,7,8-chlorinated dioxin and furan in effluent from mills that used chlorine or chlorine dioxide to bleach pulp. The *Pulp*

[4]Cost estimates for 96 Canadian pulp and paper mills to become compliant with new effluent regulations were 2.2 billion dollars at an average cost of $ 23.8 million per mill. Costs of meeting PCDD and PCDF regulations were estimated at $500 million for 47 affected mills (Environment Canada, 1990, Cleaning up pollution in the pulp and paper industry: an overview of the federal regulatory strategy. Environmental Protection Branch, Environment Canada, Ottawa, ON).

[5]For more details on treatment of pulp mill effluent, consult Biermann (1996). Processes are labeled as primary, secondary, and sometimes, tertiary treatment. Primary treatment, removes solids by allowing them to settle out was widely implemented in the 1950s. Currently, most mills also employee at least a secondary treatment process involving use of oxygen and microorganisms to remove oxygen-consuming materials, which significantly decreases toxicity of the effluent. In Canada the most common secondary steps are aerated stabilization basins and activated sludge. Some mills also have a tertiary process following the secondary treatment normally to reduce odor and color.

and Paper Mill Defoamer and Wood Chip Regulations, targeted the defoamers used in chlorine bleaching processes and prohibited the manufacture of pulp from wood chips treated with polychlorinated phenols.

In 1992, the British Columbia government moved to further regulate the industry by enacting the Pulp and Paper Mill Liquid Effluent Control Regulations, which provided a 10-year staged process to eliminate chlorinated organic compounds from effluent by requiring increasingly strict controls on AOX. Alberta had already set a high standard in its 1988 legislation, which required all mills to implement best available technologies to minimize the impact of effluent on receiving waters, a decision at that time based largely on application of the precautionary principle.

Implementation of those new regulations led to industry-wide improvements in the treatment processes. Significant changes in the chemical composition and toxicity of the final effluent and significant reductions in hazard to aquatic organisms were demonstrated (e.g. Bothwell 1992; Servos et al. 1996; Dubé and McLatchy 2000). Previous sections have discussed how PCDD and PCDF contamination of wildlife decreased both at coastal and interior aquatic sites. Those declines were also documented in sediments and other biota (Hagen et al. 1997; Macdonald et al. 1998; Bright et al. 1999; Yunker et al. 2002). In the late 1990s, studies were beginning to document early signs of recovery in fish reproductive parameters at a number of mills that had modernized their processes (Munkittrick et al. 1997). Those improvements to pulp mill effluent treatment have reportedly also reduced toxicity events and the effects of eutrophication on benthic invertebrate communities (Felder et al. 1998; Chambers et al. 2000; Culp et al. 2000a, b; Lowell and Culp 2002). In addition, the PPER required implementation of an Environmental Effects Monitoring (EEM) program at each mill in order to monitor the quality of mill effluent and to provide long-term data on the impacts on Canadian aquatic ecosystems. As a result of those changes, all marine and freshwater systems in Canada receiving pulp and paper effluents or runoff from sawmills and log sorts are now much cleaner than they were prior to the early 1990s.

Conservation Gains

Presently, on both the south coast and the interior rivers of British Columbia, the eggs and offspring of wildlife species, including herons, cormorants, grebes, seaducks, eagles, ospreys, mink, and otter are exposed to concentrations of PCDDs and PCDFs, which are in many cases several orders of magnitude lower than pre 1990s. In the case of the herons, we documented the improvement of the physiological and morphological responses in concert with the reduced exposure in the Crofton area (Sanderson et al. 1994a, b). It seems reasonable to suppose that other species would have exhibited similar improvements in those health parameters. Given variation in species sensitivity and lack of firm information about developmental and behavioral effects of those chemicals, it is difficult to confidently assess the degree of later injury caused by exposure during early development. Some impact at the population level is possibly evident from the consistent poor reproductive success of both herons

and eagles in the number of the zones most heavily impacted by pulp mill pollution. Unfortunately, there is limited subsequent information other than reports that herons have returned to breed successfully in the Crofton area.

Overall, removal of that contamination reduced a significant anthropogenic stress on the populations. In the process, we learned a great deal about the basic ecology and reproductive biology, of some key species, which has aided in assessment of overall habitat loss and degradation.

Summary and Lessons Learned

These are complex societal issues eventually fought out, at least in democratic societies, in courts of public opinion and, therefore, at political levels (e.g. Harrison 2002; Lougheed 2009, this volume). Truth and facts may often be obscured in the arguments made by the various opponents and proponents of the given commercial activity causing the pollution (Elliott et al. Introduction to this volume). So, it is incumbent on the scientists working for both parties to make truthful and factual arguments, and to resist tendencies to over extend interpretation, whether in defense of the wildlife resource or the commercial client. Involvement in this and related issues has personally emphasized the need to focus on producing the best scientific data with the available resources. It may become difficult if the issue expands along with related and extraneous demands on one's time. It is also important, not to ignore, but to remain skeptical about threats such as job losses and damage to commerce. That is for the regulators and ultimately politicians to work out using cost-benefits models. In this case, most of the mills continue to operate, and profitably it seems. Some mills have closed for wider reasons, such as depleted timber supplies and market factors including competition from lower cost producers elsewhere, related to labor costs and environmental and other regulations. That is a complex topic in environmental economics and beyond the scope of this article (see, for example: Norberg-Bohm and Rossi 1998; Clarkson et al. 2004).

This work provides another lesson in how wildlife and particularly fish-eating and predatory birds make elegant and efficient sentinels of ecosystem contamination by persistent bioaccumulative contaminants. In the examples discussed here from British Columbia, but also from the Great Lakes, there was evidence of a contamination problem in wildlife many years before there was corroborating evidence from other levels of the foodchain, or from sampling of nonbiota, such as sediment. The work also further underscores the power of charismatic wildlife to attract public attention and galvanize people to respond to such evidence.

Looking ahead, there are a number of observations if we wish to maintain the capability for this type of investigation, and ways to improve. These include:

- The need for in-house expertise in government agencies, both in the laboratory to adapt and develop methods for new chemicals and other threats to wildlife health, and in the field to identify and address problems before they reach a critical stage.

- The need to apply that expertise to assess new and emerging chemical threats as was done here in the case of dioxins, and more recently in the case of the brominated flame retardant chemicals (e.g. Elliott et al. 2005; McKinney et al. 2006; Cesh et al. 2010).
- The fostering of close cooperation between researchers, risk assessors, and regulators in the evaluation and monitoring of new chemicals.
- The maintenance of tissue archives such as the National Specimen Bank.
- The continuous development and refinement of wildlife monitoring techniques, particularly the use of nondestructive and ideally non-intrusive techniques such as fecal sampling.
- The development and application of appropriate study designs, many of which, such as the sample egg technique, have been around for some time but are not always applied (e.g. Blus 1984; Custer et al. 1999; Henny et al. 2009), including use of blood, feather, feces, etc.
- Continuous awareness and adaptation of improvement in data analysis and statistical methods such as information theoretic, and probabilistic approaches (Burnham and Anderson 2001; and see examples: Elliott et al. 2009; Custer et al. 2010; Cesh et al. 2010; Best et al. 2010).
- The continued development and application of a landscape approach using spatial analysis methods.
- The development and refinement of biomarkers, particularly using new genomics technologies.
- The application of other molecular technologies, such a DNA genotyping, into population level assessments (e.g., Guertin et al. 2010).

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References

Addison RF, Ikonomou MG, Smith TG (2005) PCDD/F and PCB in harbour seals (*Phoca vitulina*) from British Columbia: response to exposure from pulp mill effluents. Mar Environ Res 59:165–176

Amendola G, Barna D, Blosser R, LaFleur L, McBride A, Thomas F, Tiernan T, Whittemore R (1989) The occurrence and fate of PCDDs and PCDFs in five bleached kraft pulp and paper mills. Chemosphere 18:1181–1188

Ames PL (1966) DDT residues in the eggs of the osprey in the northeastern USA and their relation to nest success. J Appl Ecol 3:87–97

Augspurger TP, Tillitt DE, Bursian SJ, Fitzgerald SD, Hinton DE, Di Giulio RT (2008) Embryo toxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin to the wood duck (Aix sponsa). Arch Environ Contam Toxicol 55:659–669

Aulerich RJ, Ringer RK (1977) Current status of PCB toxicity to mink, and effect on their reproduction. Arch Environ Contam Toxicol 6:279–292

Beck U (1992) Risk society: towards a new modernity. Sage, London

Bellward GD, Norstrom RJ, Whitehead PE, Elliott JE, Bandiera SM, Dworschak C, Chang T, Forbes S, Cadario B, Hart LE, Cheng KM (1990) Comparison of polychlorinated dibenzodioxin levels with hepatic mixed-function oxidase induction in great blue herons. J Toxicol Environ Health 30:33–52

Berry RM, Fleming BI, Voss RH, Luthe CE, Wrist PE (1989) Towards preventing the formation of dioxins during chemical pulp bleaching. Pulp Paper Can 90:48–58

Best DA, Elliott KH, Bowerman WW, Shieldcastle M, Postupalsky S, Nye PE, Kubiak TJ, Tillitt DE, Elliott JE (2010) Productivity, embryo and eggshell characteristics and contaminants in bald eagles from the Great Lakes, U.S.A. Environ Toxicol Chem 29:1581–1592

Biermann CJ (1996) Pulping and paper making. Academic, San Diego, California

Blus LJ (1984) DDE in birds' eggs: comparison of two methods for estimating critical levels. Wilson Bull 96:268–276

Bothwell ML (1992) Eutrophication of rivers by nutrients in treated kraft pulp mill effluent. Water Pollut Res J Can 27:447–472

Bowes GW, Simoneit BR, Burlingame AL, de Lappe BW, Risebrough RW (1973) The search for chlorinated dibenzofurans and chlorinated dioxins in wildlife populations showing elevated levels of embryonic death. Environ Health Perspect 5:191–198

Bradlaw JA, Casterline JL (1979) Induction of enzyme activity in cell culture: a rapid screen for detection of planar polychlorinated organic compounds. J Assoc Off Anal Chem 62:904–916

Braune BM, Malone BJ, Burgess NM, Elliott JE, Garrity N, Hawkings J, Hines J, Marshall H, Marshall WK, Rodrigue J, Wakeford B, Wayland M, Weseloh DV, Whitehead PE (1999) Chemical residues in waterfowl and gamebirds harvested in Canada, 1987–1995. Canadian Wildlife Service, Technical Report Series, No 326, Ottawa ON, Canada

Bright DA, Cretney WJ, Macdonald RW, Ikonomou MG, Grundy SL (1999) Differentiation of polychlorinated dibenzo-p-dioxin and dibenzofuran sources in coastal British Columbia, Canada. Environ Toxicol Chem 18:1097–1108

Burnham KP, Anderson DR (2001) Kullback-Leibler information as a basis for strong inference in ecological studies. Wildl Res 28:111–119

Butler RW (1997) The great blue heron. UBC, Vancouver BC, Canada, p 167

Campbell RW, Dawe NK, McTaggert-Cowan I, Cooper JM, Kaiser GW, McNall MCE (1990) The birds of British Columbia, vol 1. Victoria, BC, Canada, 514 pp

Carson R (1962) Silent spring. Houghton Mifflin, New York

Cesh L, Elliott KH, McKinney M, Quade S, Maisonneuve F, Garcelon DK, Sandau CD, Letcher RJ, Williams TD, Elliott JE (2010) Polyhalogenated aromatic hydrocarbons and metabolites: relation to circulating thyroid hormone and retinol in nestling bald eagles (*Haliaeetus leucocephalus*). Environ Toxicol Chem 29:1301–1310

Chambers PA, Dale AR, Scrimgeour GC, Bothwell ML (2000) Nutrient enrichment of northern rivers in response to pulp mill and municipal discharges. J Aquat Ecosyst Stress Recov 8:53–66

Clarkson PM, Li Y, Richardson GD (2004) The market valuation of environmental capital expenditures by pulp and paper companies. Accounting Rev 79:329–353

Culp JM, Lowell RB, Cash KJ (2000a) Integrating mesocosm experiments with field and laboratory studies to generate weight-of-evidence risk assessments for large rivers. Environ Toxicol Chem 19:1167–1173

Culp JM, Podemski CL, Cash KJ (2000b) Interactive effects of nutrients and contaminants from pulp mill effluents on riverine benthos. J Aquat Ecosyst Stress Recov 8:67–75

Custer TW, Custer CM, Hines RK, Gutreuter S, Stromborg KL, Allen PD, Melancon MJ (1999) Organochlorine contaminants and reproductive success of double-crested cormorants from Green Bay, Wisconsin, USA. Environ Toxicol Chem 18:1209–1217

Custer TW, Custer CM, Gray BR (2010) Polychlorinated biphenyls, dioxins, furans, and organochlorine pesticides in spotted sandpiper eggs from the upper Hudson River basin, New York. Ecotoxicology 19:391–404

Czuczwa JM, Hites RM (1984) Environmental fate of combustion-generated polychlorinated dioxins and furans. Environ Sci Technol 18:444–450

Czuczwa JM, McVeety BD, Hites RA (1984) Polychlorinated dibenzo-p-dioxins and dibenzofurans in sediments from Siskiwit Lake, Isle Royale. Science 226:568–569

Dickerson SM, Gore AC (2007) Estrogenic environmental endocrine-disrupting chemical effects on reproductive neuroendocrine function and dysfunction across the life cycle. Rev Endocr Metab Disorders 8:143–159

Dubé MG, McLatchy DL (2000) Endocrine responses of *Fundulus heteroclitus* to effluent from a bleached-kraft mill before and after installation of reverse osmosis treatment of a waste stream. Environ Toxicol Chem 19:2788–2796

Elliott JE (1984) Collecting and archiving wildlife specimens in Canada. In: Lewis RA, Stein N, Lewis CW (eds) Environmental specimen banking and monitoring as related to banking. Martinus Nijhoff, The Hague, pp 45–64

Elliott JE, Martin PA (1998) Chlorinated hydrocarbons in grebes and seaducks wintering on the coast of British Columbia, Canada, 1988–93. Environ Monitor Assess 53:337–362

Elliott JE, Norstrom RJ (1998) Chlorinated hydrocarbon contaminants and productivity of bald eagle populations on the Pacific coast of Canada. Environ Toxicol Chem 17:1142–1153

Elliott JE, Butler RW, Norstrom RJ, Whitehead PE (1988b) Levels of polychlorinated dibenzo-dioxins and polychlorinated dibenzofurans in eggs in Great Blue Herons (*Ardea herodias*) in British Columbia, 1983–1987: possible effects on reproductive success. Canadian Wildlife Service Progress Notes, Ottawa, ON, No. 176, p 7

Elliott JE, Norstrom RJ, Kennedy SK, Fox GA (1988a) Trends and effects of environmental contaminants determined from analysis of archived wildlife samples. In: Wise SA, Zeisler GM (eds) Progress in environmental specimen banking. National Bureau of Standards (U.S.) Spec. Publ. No. 740:131–142

Elliott JE, Butler RW, Norstrom RJ, Whitehead PE (1989) Environmental contaminants and reproductive success of Great Blue Herons *Ardea herodias* in British Columbia, 1986–87. Environ Pollut 59:91–114

Elliott JE, Norstrom RJ, Lorenzen A, Hart LE, Philibert H, Kennedy SW, Stegeman JJ, Bellward GD, Cheng KM (1996a) Biological effects of polychlorinated dibenzo-*p*-dioxins, dibenzofurans, and biphenyls in bald eagle (*Haliaeetus leucocephalus*) chicks. Environ Toxicol Chem 15:782–793

Elliott JE, Norstrom RJ, Smith GEJ (1996b) Patterns, trends and toxicological significance of chlorinated hydrocarbons and mercury in bald eagle eggs. Arch Environ Contam Toxicol 31:354–367

Elliott JE, Wilson LK, Langelier KE, Norstrom RJ (1996c) Bald eagle mortality and chlorinated hydrocarbons in liver samples from the Pacific coast of Canada, 1988–1995. Environ Pollut 94:9–18

Elliott JE, Machmer MM, Henny CJ, Whitehead PE, Norstrom RJ (1998a) Contaminants in ospreys from the Pacific Northwest: I. Trends and patterns in polychlorinated dibenzo-p-dioxins and dibenzofurans in eggs and plasma. Arch Environ Contam Toxicol 35:620–631

Elliott JE, Moul IE, Cheng KM (1998b) Variable reproductive success of bald eagles on the British Columbia coast. J Wildl Manage 62:518–529

Elliott JE, Henny CJ, Harris ML, Wilson LK, Norstrom RJ (1999) Chlorinated hydrocarbons in livers of American mink (*Mustela vison*) and river otter (*Lutra canadensis*) from the Columbia and Fraser River basins, 1990–1992. Environ Monit Assess 57:229–252

Elliott JE, Harris ML, Wilson LK, Whitehead PE, Norstrom RJ (2001a) Monitoring temporal and spatial trends in polychlorinated dibenzo-*p*-dioxins (PCDDs) and dibenzofurans (PCDFs) in eggs of great blue heron (*Ardea herodias*) on the coast of British Columbia, Canada, 1983–19. Ambio 30:416–428

Elliott JE, Wilson LK, Henny CJ, Trudeau SF, Leighton FA, Kennedy SW, Cheng KM (2001b) Assessment of biological effects of chlorinated hydrocarbons in osprey chicks. Environ Toxicol Chem 20:866–879

Elliott KH, Gill CE, Elliott JE (2005) Influence of tides and weather on bald eagle provisioning rates. J Raptor Res 39:99–108

Elliott JE, Guertin DA, Balke JME (2008) Chlorinated hydrocarbon contaminants in faeces of coastal marine river otters. Sci Total Environ 397:58–71

Elliott KH, Cesh LS, Dooley JA, Letcher RJ, Elliott JE (2009) PCBs and DDE, but not PBDEs, increase with trophic level and marine input in nestling bald eagles. Sci Total Environ 407:3867–3875

Fanelli R, Bertoni MP, Castelli MG, Chiabrando C, Martelli GP, Noseda A, Garattini S, Binaghi C, Marazza V, Pezza F (1980a) 2,3,7,8-tetrachlorodibenzo-p-dioxin toxic effects and tissue levels in animals from the contaminated area of Seveso, Italy. Arch Environ Contam Toxicol 9:569–577

Fanelli R, Castelli MG, Martelli GP, Noseda A, Garattini S (1980b) Presence of 2,3,7,8-tetrachlorodibenzo-p-dioxin in wildlife living near Seveso, Italy: a preliminary study. Bull Environ Contam Toxicol 24:460–462

Felder DP, D'Surney SJ, Rodgers JH Jr, Deardorff TL (1998) A comprehensive environmental assessment of a receiving aquatic system near an unbleached kraft mill. Ecotoxicology 7:313–324

Fernie KJ, Smits JE, Bortolotti GR, Bird DM (2001) In ovo exposure to polychlorinated biphenyls: reproductive effects on second-generation American kestrels. Arch Environ Contam Toxicol 40:544–550

Firestone D (1973) Etiology of chick-edema disease. Environ Health Perspect 5:59–66

Friedman L, Firestone D, Horwitz W, Banes D, Anstead M, Shue G (1959) Studies of the chick edema disease factor. J Assoc Offic Agr Chem 42:129–140

Fry DM, Toone CK (1981) DDT-induced feminization of gull embryos. Science 213:922–924

Gilbertson M (1974) Pollutants in breeding herring gulls from the lower Great Lakes. Can Field Nat 88:273–280

Gilbertson M (1975) A Great Lakes tragedy. Nat Can 4:22–25

Gilbertson M, Reynolds L (1972) DDE and PCB in Canadian birds 1969 to 1972. Canadian Wildlife Service. Occasional Paper No. 19, p 17

Gilbertson M, Kubiak T, Ludwig J, Fox G (1991) Great Lakes embryo mortality, edema, and deformities syndrome (GLEMEDS) in colonial fish-eating birds: similarity to chick-edema disease. J Toxicol Environ Heath 33:455–520

Gill CE, Elliott JE (2003) Influence of food supply and chlorinated hydrocarbon contaminants on breeding success of bald eagles. Ecotoxicology 12:95–112

Gilman AP, Fox GA, Peakall DB, Teeple SM, Carroll TR, Haymes GT (1977) Reproductive parameters and egg contaminant levels of Great Lakes herring gulls. J Wildl Manage 41:458–468

Government of Canada (1992) Pulp and paper effluent regulations. Canada Gazette Part II, Registration SOR/92-269 126, 1967–2006

Grove RA, Henny CJ, Kaiser JL (2009) Osprey: worldwide sentinel species for assessing and monitoring environmental contamination in rivers, lakes, reservoirs, and estuaries. J Toxicol Environ Health B Crit Rev 12:25–44

Guertin DA, Ben-David M, Harestad A, Drouillard KG, Elliott JE (2009) Non-invasive fecal sampling links individual river otters to chlorinated hydrocarbon contaminant exposure. Environ Toxicol Chem 29:275–284

Guertin DA, Ben-David M, Harestad A, Drouillard KG, Elliott JE (2010) Non-invasive fecal sampling links individual river otters to chlorinated hydrocarbon contaminant exposure. Environ Toxicol Chem 29:275–284

Hagen ME, Colodey AG, Knapp WD, Samis SC (1997) Environmental response to decreased dioxin and furan loadings from British Columbia coastal pulp mills. Chemosphere 34:1221–1229

Harding LE, Pomeroy WM (1990) Dioxin and furan levels in sediments, fish and invertebrates from fishery closure areas of Coastal British Columbia. Environment Canada Regional Data Report 90-09. Environment Canada, Pacific and Yukon Region, North Vancouver, BC, Canada, p 77

Harfenist A, Whitehead PE, Cretney WJ, Elliott JE (1993) Food chain sources of polychlorinated dioxins and furans to great blue herons (*Ardea herodias*) foraging in the Fraser River Estuary, British Columbia. Canadian Wildlife Service (CWS) Technical Report Series No. 169. CWS, Pacific and Yukon Region, Environment Canada, Delta, BC, Canada

Harris ML, Elliott JE (2011) Polychlorinated biphenyls, dibenzo-*p*-dioxins and dibenzofurans in birds. In: Beyer WN, Meador J (eds) Environmental contaminants in wildlife – interpreting tissue concentrations. CRC, New York, NY, pp 471–522

Harris ML, Elliott JE, Wilson LK, Butler RW (2003a) Reproductive success and chlorinated hydrocarbon contamination of resident great blue herons (*Ardea herodias*) from coastal British Columbia, Canada, 1977 to 1998. Environ Pollut 121:207–227

Harris ML, Wilson LK, Norstrom RJ, Elliott JE (2003b) Egg concentrations of polychlorinated dibenzo-p-dioxins and dibenzofurans in double-crested (*Phalacrocorax auritus*) and pelagic (*P. pelagicus*) cormorants from the Strait of Georgia, Canada, 1973–1998. Environ Sci Technol 37:822–831

Harris ML, Wilson LK, Elliott JE (2005) An assessment of chlorinated hydrocarbon contaminants in eggs of double-crested (*Phalacrocorax auritus*) and pelagic (*P. pelagicus*) cormorants from the west coast of Canada, 1970 to 2002. Ecotoxicology 14:607–625

Harrison K (2002) Ideas and environmental standard-setting: a comparative study of regulation of the pulp and paper industry. Governance 15:65–96

Hart LE, Cheng KM, Whitehead PE, Shah RM, Lewis RJ, Ruschkowski SR, Blair RW, Bennett DC, Bandiera SM, Norstrom RJ, Bellward GD (1991) Dioxin contamination and growth and development in great blue heron embryos. J Toxicol Environ Health 32:331–344

Hebert CE, Norstrom RJ, Simon M, Braune BM, Weseloh DV, Macdonald CR (1994) Temporal trends and sources of PCDDs and PCDFs in the Great Lakes: herring gull egg monitoring, 1981–1991. Environ Sci Technol 28:1268–1277

Helander B, Olsson A, Bignert A, Asplund L, Litzén K (2002) The role of DDE, PCB, coplanar PCB and eggshell parameters for reproduction in the white-tailed sea eagle (*Haliaeetus albicilla*) in Sweden. Ambio 31:386–403

Henny CJ, Blus LJ, Gregory SV, Stafford CJ (1981) PCBs and organochlorine pesticides in wild mink and river otters from Oregon. In: Chapman JA, Pursley D (eds) Worldwide furbearer conference proceedings, August 1980, vol 3. Frostburg, MD, USA, pp 1763–1780

Henny CJ, Rudis DD, Roffe TJ, Robinson-Wilson E (1995) Contaminants and sea ducks in Alaska and the circumpolar region. Environ Health Perspect 103:41–49

Henny CJ, Kaiser JL, Grove RA, Johnson BL, Letcher RJ (2009) Polybrominated diphenyl ether flame retardants in eggs may reduce reproductive success of ospreys in Oregon and Washington, USA. Ecotoxicology 18:802–813

Henshel DS (1998) Developmental neurotoxic effects of dioxin and dioxin-like compounds on domestic and wild avian species. Environ Toxicol Chem 17:88–98

Higginbotham GR, Huang A, Firestone D, Verrett J, Ress J, Campbell AD (1968) Chemical and toxicological evaluations of isolated and synthetic chloro derivatives of dibenzo-p-dioxin. Nature 220:702–703

Holm L, Blomqvist A, Brandt I, Brunstrom B, Ridderstale Y, Berg C (2006) Embryonic exposure to o, p'-DDT causes eggshell thinning and altered shell gland carbonic anhydrase expression in the domestic hen. Environ Toxicol Chem 25:2787–2793

Hoogesteijn AL, Kollias GV, Quimby FW, De Caprio AP, Winkler DW, DeVoogd TJ (2008) Development of a brain nucleus involved in song production in zebra finches (*Taeniopygia guttata*) is disrupted by Aroclor 1248. Environ Toxicol Chem 27:2071–2075

Huff JE, Wassom JS (1974) Health hazards from chemical impurities: chlorinated dibenzodioxins and chlorinated dibenzofurans. Int J Environ Stud 6:1–17

Interdepartmental Committee on Dioxins (1985) Dioxins in Canada: the federal approach. Environmental Protection Branch, Ottawa ON, Canada

International Joint Commission (IJC) (1978) Great Lakes Water Quality Agreement of 1978. Agreement with annexes and terms of reference, between the United States of America and Canada. 22 November 1978, Ottawa, ON, Canada, p 52

Iwaniuk AN, Koperski DT, Cheng KM, Elliott JE, Smith LK, Wilson LK, Wylie DRW (2006) The effects of environmental exposure to DDT on the brain of a songbird: changes in structures associated with mating and song. Behav Brain Res 173:1–10

Jones I (2009) Bald eagle and great blue heron. Thesis, Masters of Science, Simon Fraser University, Department of Biological Sciences

Kruuk H, Conroy JWH (1966) Concentrations of some organochlorines in otters (*Lutra lutra* L.) in Scotland: implications for populations. Environ Pollut 92:165–171

Kuehl DW, Cook PM, Batterman AR, Lothenbach D, Butterworth BC (1987) Bioavailability of polychlorinated dibenzo-p-dioxins and dibenzofurans from contaminated Wisconsin River sediment to carp. Chemosphere 16:667–679

Lougheed T (2009) Outside looking in: understanding the role of science in regulation. Environ Health Perspect 117:105–110

Lowell RB, Culp JM (2002) Implications of sampling frequency for detecting temporal patterns during environmental effects monitoring. Water Qual Res J Can 37:119–132

Luthe CE, Berry RM, Voss RH (1993) Formation of chlorinated dioxins during production of bleached kraft pulp from sawmill chips contaminated with polychlorinated phenols. Tappi J 76:63–69

Mably TA, Bjerke DL, Moore RW, Gendron-Fitzpatrick A, Peterson RE (1992) In utero and lactational exposure of male rats to 2,3,7,8-tetrachlorodibenzo-p-dioxin. III. Effects on spermatogenesis and reproductive capability. Toxicol Appl Pharmacol 114:118–126

Macdonald SM, Mason CF (1988) Observations on an otter population in decline. Acta Theriol 33:415–434

Macdonald RW, Cretney WJ, Crewe N, Paton D (1992) A history of octachlorodibenzo-p-dioxin, 2,3,7,8-tetrachlorodibenzofuran, and 3,3′,4,4′-tetrachlorobiphenyl contamination in Howe Sound, British Columbia. Environ Sci Technol 26:1544–1550

Macdonald RW, Ikonomou MG, Paton DW (1998) Historical inputs of PCDDs, PCDFs, and PCBs to a British Columbia interior lake: the effect of environmental controls on pulp mill emissions. Environ Sci Technol 33:331–337

McArthur MLB, Fox GA, Peakall DB, Philogene BJR (1983) Ecological significance of behavioural and hormonal abnormalities in breeding ring doves fed an organochlorine chemical mixture. Arch Environ Contam Toxicol 12:343–353

McKinney MA, Cesh LS, Elliott JE, Williams TD, Garcelon DK, Letcher RJ (2006) Novel brominated and chlorinated contaminants and hydroxylated analogues among North American west coast populations of bald eagles (Haliaeetus leucocephalus). Environ Sci Tech 40: 6275–6281

McPhail JD, Troffe PM (1998) The mountain whitefish (Prosopium williamsoni): a potential indicator species for the Fraser System. DOE FRAP 1998-16 Report, Environment Canada, Vancouver, BC, Canada

Minh TB, Iwata H, Takahashi S, Viet PH, Tuyen BC, Tanabe S (2008) Persistent organic pollutants in Vietnam: environmental contamination and human exposure. Rev Environ Contam Toxicol 193:213–290

Moul IE (1990) Environmental contaminants, disturbance and breeding failure at a great blue heron colony on Vancouver Island. Unpubl. MSc. Thesis, University of British Columbia, Vancouver, BC, Canada

Moul IE, Vennesland RG, Harris ML, Butler RW (2001) Standardizing and interpreting nesting records for Great Blue Herons in British Columbia. Canadian Wildlife Service (CWS) Progress Note No. 217. CWS, Pacific and Yukon Region, Vancouver, BC, Canada. Available from http://www.cws-cf.ec.gc.ca/pub/pnotes/index_e.html

Munkittrick KR, Servos MR, Carey JH, Van Der Kraak GJ (1997) Environmental impacts of pulp and paper wastewater: evidence for a reduction in environmental effects at North American pulp mills since 1992. Water Sci Technol 35:329–338

Nebert DW, Dalton TP, Okey AB, Gonzalez FJ (2004) Role of aryl hydrocarbon receptor-mediated induction of the CYP1 enzymes in environmental toxicity and cancer. J Biol Chem 279:23847–23850

Nener J, Kieser D, Thompson JA, Lockhart WL, Metner DA, Roome R (1995) Monitoring of mountain whitefish (*Prosopium williamsoni*), from the Columbia River system near Castlegar, British Columbia: health parameters and contaminants in 1992. Can Tech Rep Fish Aquat Sci, No. 2036, p 89

Norberg-Bohm V, Rossi M (1998) The power of incrementalism: environmental regulation and technological change in pulp and paper bleaching in the US. Technol Anal Stateg Manage 10:225–245

Norman D, Breault AM, Moul IE (1989) Bald eagle incursions and predation at great blue heron colonies. Colonial Waterbirds 12:215–217

Norstrom RJ, Simon M (1983) Preliminary appraisal of tetra- to octachlorodibenzodioxin contamination in eggs of various species of wildlife in Canada. In: Miyamoto J (ed) IUPAC pesticide chemistry: human welfare and the environment. Pergamon, Oxford, UK, pp 165–170

Peakall DB, Fox GA (1987) Toxicological investigations on pollutant-related effects in Great Lakes gulls. Environ Health Perspect 71:187–193

Peakall DB, Peakall ML (1973) Effects of a polychlorinated biphenyl on the reproduction of artificially and naturally incubated dove eggs. J Appl Ecol 10:863–868

Peterson RE, Theobald HM, Kimmel GL (1993) Developmental and reproductive toxicity of dioxins and related compounds: cross-species comparisons. CRC Rev 23:283–335

Pohl HR, Hicks HE, Jones DE, Hansen H, De Rosa CT (2002) Public health perspectives on dioxin risks: two decades of evaluations. Hum Ecol Risk Assess 8:233–250

Poland A, Knutson JC (1982) 2,3,7,8-tetrachlorodibenzo-*p*-dioxin and related halogenated aromatic hydrocarbons: examination of the mechanism of action. Ann Rev Pharmacol Toxicol 22:517–554

Poland A, Glover E, Kende AS (1976) Stereospecific, high affinity binding of 2,3,7,8-tetrachlorodibenzo-p-dioxin by hepatic cytosol. J Biol Chem 251:4936–4946

Poole AF (1989) Ospreys: a natural and unnatural history. Cambridge University Press, New York

Powell RL (1984) Dioxin in Missouri: 1971–1983. Bull Environ Contam Toxicol 33:648–654

Quinn JL, Ueta M (2008) Protective nesting associations in birds. Ibis 150:146–167

Ross PS, Ellis GM, Ikonomou MG, Barrett-Lennard LG, Addison RF (2000) High PCB concentrations in free-ranging Pacific killer whales, *Orcinus orca*: effects of age, sex and dietary preference. Mar Pollut Bull 40:504–515

Ross PS, Jeffries SJ, Yunker MB, Addison RF, Ikonomou MG, Calambokidis JC (2004) Harbor seals (*Phoca vitulina*) in British Columbia, Canada, and Washington State, USA, reveal a combination of local and global polychlorinated biphenyl, dioxin, and furan signals. Environ Toxicol Chem 23:157–165

Ryan JJ, Lizotte R, Lau BPY (1985) Chlorinated dibenzo-p-dioxins and chlorinated dibenzofurans in Canadian human adipose tissue. Chemosphere 14:697–706

Safe S (1984) Polychlorinated biphenyls (PCBs) and polybrominated biphenyls (PBBs): biochemistry, toxicology, and mechanism of action. Crit Rev Toxicol 13:319–393

Sanderson JT, Elliott JE, Norstrom RJ, Whitehead PE, Hart LE, Cheng KM, Bellward GD (1994a) Monitoring biological effects of polychlorinated dibenzo-p-dioxins, dibenzofurans and biphenyls in great blue heron chicks. J Toxicol Environ Health 41:435–450

Sanderson JT, Norstrom RJ, Elliott JE, Hart LE, Cheng KM, Bellward GD (1994b) Biological effects of polychlorinated dibenzo-*p*-dioxins, dibenzofurans, and biphenyls in double-crested cormorant chicks (*Phalacrocorax auritus*). J Toxicol Environ Health 41:247–265

Segstro MD, Muir DCG, Servos MR, Webster GRB (1995) Long-term fate and bioavailability of sediment-associated polychlorinated dibenzo-*p*-dioxins in aquatic mesocosms. Environ Toxicol Chem 14:1799–1807

Servos ME, Munkittrick KR, Carey JH, van der Kraak GJ (1996) Environmental fate and effects of pulp and paper mill effluents. St. Lucie, Delray Beach, FL

Smit MD, Leonards PEG, van Hattum AGM, de Jongh AWJJ (1994) PCBs in European otter (*Lutra lutra*) populations, report nr 4-97/7. Institute for Environmental Studies, Vrije Universiteit, Amsterdam, p 45

Smit MD, Leonards PEG, van Hattum AGM, de Jongh AWJJ (1994) PCBs in European otter (Lutra lutra) populations. Report R-94/7. Institute for Environmental Studies, Vrije Universiteit, Amsterdam, The Netherlands

Spitzer PR, Risebrough RW, Walker W, Hernandez R, Poole A, Puleston D, Nisbet ICT (1978) Productivity of ospreys in Connecticut–Long Island increases as DDE residues decline. Science 202:333–335

Stalling DL, Norstrom RJ, Smith LM, Simon M (1985) Patterns of PCDD, PCDF and PCB contamination in Great Lakes fish and birds and their characterization by principal components analysis. Chemosphere 14:627–643

Steidl RJ, Griffin CR, Niles LJ (1991) Contaminant levels of osprey eggs and prey reflect regional differences in reproductive success. J Wildl Manage 55:601–608

Swanson SE (1988) Dioxins in the Bleach Plant. Ph.D. Thesis, Institute of Environmental Chemistry, Umea, Sweden

Thiel DA, Martin SG, Duncan JW, Lemke MJ, Lance WR, Peterson RE (1988) Evaluation of the effects of dioxin-contaminated sludges on wild birds. In: Proceedings of the 1988 Technical Association of Pulp and Paper Environmental Conference. Charleston, SC, p 145–148

Thomas CM, Anthony RG (1999) Environmental contaminants in great blue herons (*Ardea herodias*) from the lower Columbia and Willamette Rivers, Oregon and Washington, USA. Environ Toxicol Chem 18:2804–2816

Tschirley FH (1986) Dioxin. Sci Am 254:19–22

Van Strum C, Merrell PE (1987) No margin of safety: a preliminary report on dioxin pollution and the need for emergency action in the pulp and paper industry. Greenpeace, Toronto, ON

Vennesland RG, Butler RA (2004) Factors influencing Great Blue Heron nesting productivity on the Pacific coast of Canada from 1998 to 1999. Waterbirds 27:289–296

Vermeer K, Cretney WJ, Elliott JE, Norstrom RJ, Whitehead PE (1993) Elevated polychlorinated dibenzodioxin and dibenzofuran concentrations in grebes, ducks and their prey near Port Alberni, British Columbia, Canada. Mar Pollut Bull 26:431–435

Verrett MJ (1970) Effects of 2,4,-T on man and the environment. In: Hearings before the Subcommittee on Energy, Natural Resources and the Environment of the Committee on Commerce, US Senate, Serial 91-60. US Government Printing Office, Washington, DC

White DH, Seginak JT (1994) Dioxins and furans linked to reproductive impairment in wood ducks. J Wildl Manage 58:100–106

Whitehead PE, Elliott JE, Norstrom RJ, Vermeer K (1990) PCDD and PCDF contamination of waterfowl in the Strait of Georgia, British Columbia, Canada, 1989-90. Organohalogen Compounds 1:459–462

Wiemeyer SN, Spitzer PR, Krantz WC, Lamont TG, Cromartie E (1975) Effects of environmental pollutants on Connecticut and Maryland ospreys. J Wildl Manag 39:124–139

Wiemeyer SN, Bunck CM, Krynitsky AJ (1988) Organochlorine pesticides, polychlorinated biphenyls and mercury in osprey eggs – 1970–79 – and their relationships to shell thinning and productivity. Arch Environ Contam Toxicol 17:767–787

Woodford JE, Krasov WH, Meyer ME, Chambers L (1998) Impact of 2,3,7,8-TCDD exposure on survival, growth, and behaviour of ospreys breeding in Wisconsin, USA. Environ Toxicol Chem 17:1323–1331

Yunker MB, Cretney WJ, Ikonomou MG (2002) Assessment of chlorinated dibenzo-*p*-dioxin and dibenzofuran trends in sediment and crab hepatopancreas from pulp mill and harbour sites using multivariate- and index-based approaches. Environ Sci Technol 36:1869–1878

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