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STATEMENT

OF

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before
the

SENATE GOVERNMENTAL AFFAIRS COMMITTEE

Hearing on

"Tainted Water, Tainted Fish: Stewardship of the Great Lakes Basin"

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TESTIMONY of Dr. Theo Colborn, Senior Fellow with the W. Alton Jones Foundation on sabbatical from the World Wildlife Fund, to the Senate Oversight Committee on Government Affairs, April 7, 1992.

Dr. Colborn recently started a Toxics and Wildlife Project for World Wildlife Fund and in her testimony will lean on the database she developed for that program. Previous to that she provided the science for the book, Great Lakes, Great Legacy? released in 1990 by The Conservation Foundation, Washington DC and the Institute for Research and Public Policy, Ottawa, Canada. In the book, the authors suggested that contaminated fish not be fed to females until they are finished childbearing. Throughout her testimony Dr. Colborn will also refer to a Consensus Statement from 21 experts who attended a Work Session at the Wingspread Conference Center, Racine, Wisconsin, titled "Chemically Induced Alterations in Sexual Development: The Wildlife/Human Connection", a copy of which is submitted for entry into the official record.

Thank you, Mr. Chairman, for inviting me to appear before you.
In your invitation you requested that I address the following issues in my testimony:

1. **"...the adverse human and environmental health effects, especially reproductive and developmental, associated with contaminated fish consumption...";**

2. **"..the socioeconomic impacts associated with contaminated fish in the Great Lakes basin..."; and**

3. **"...whether the FDA guidelines and state fish advisories adequately consider and protect consumers against the adverse reproductive and developmental health outcomes that may be associated with contaminated fish consumption."**

Using the wildlife/human connection as the foundation for my testimony, I will focus on the toxicity of a number of chemicals that accumulate in fish tissue. These chemicals, and therefore their effects, can be passed through the women who eat contaminated fish to their babies. Many of the chemicals that accumulate in fish tissue can disrupt the development and long term function of vital organs and systems, especially if they are present during the early developmental stages of these organs and systems. The systems include the nervous, digestive, and immune systems as well as the endocrine system that controls growth, sexual development, and reproduction.

QUESTION I. DEFINING THE PROBLEM

To characterize the health problems of developmental toxicants, I shall address what is known about the health status of wildlife that eat fish, not only in the Great Lakes, but globally. Next I shall describe the parallels between the conditions reported in wildlife and what is reported from the laboratory about the effects of eating contaminated fish and the effects of chemicals that accumulate in fish tissue. In closing I will address what is known about the effects in humans of eating fish and the results of exposure to chemicals of this nature.

1. What do we know about the health of animals that eat fish?

Populations of ALL the top predator species dependent upon fish from the Great Lakes are

still suffering reproductive problems even though there was a marked decline in contamination levels in the Lakes in the late 1970s. Acute mortality and egg shell thinning have declined considerably, but other, less obvious, yet equally devastating health effects are affecting the offspring of exposed animals. Something in the fish the animals eat prevents the normal development of their offspring. The succeeding generation is deprived of physiological systems that function properly -- the endocrine, immune, nervous, and digestive systems. Ultimately this deprives offspring of their fullest potential, and in many cases reduces their fertility and shortens their lives. For example, a condition called wasting has caused high mortality among chicks for a number of years in a population of Forster's terns in Lake Michigan and appears to have been the major cause of death in the Bald Eagle chicks on the Ohio shoreline of Lake Erie last summer.

Other sublethal signs of endocrine system disruption in wildlife are widespread.

- Researchers from Guelph University have been unable to find a Great Lakes Coho Salmon, Chinook Salmon, or Lake Trout without an enlarged thyroid in the last 10 years and recently they find that the thyroids in Lake Erie fish are rupturing, growing to more than one million times their normal size.

- Similarly, the size of the thyroid in Great Lakes herring gulls correlates with the level of contamination in their territories -- the birds' thyroid hormone ratios actually provide a picture of the gradient of pollution around the Lakes.

- Additionally, the researchers from Guelph have been unable to find a Great Lakes male salmonid that has reached full sexual maturity.

- They also report high incidences of hermaphroditism in what externally appear to be normal male and female fishes.

- In the late 1970s, male herring gull chicks and embryos were discovered with ovarian tissue and the females with a second oviduct.

- A fully hermaphroditic Beluga whale was found at the mouth of the St. Lawrence River: 1 of the 60 beached male whales that have been examined.

- White suckers below paper and pulp mills in northern Lake Superior are being neutered -- males becoming feminized and females masculinized.

- Fish that are normally hermaphroditic are being masculinized in streams in Florida below a paper mill.

- Forster's terns from a contaminated colony in Green Bay exhibited less parental attention to their eggs and chicks when compared with a "clean" colony.

- Herring gull females abnormally share nests in which they both lay a clutch of eggs. The hatching success of these nests is poor.

- Turning to the literature on marine mammals, we find that female seals along the shores of the Wadden Sea have suffered severe reproductive problems because of an abnormal condition discovered in the female reproductive organs.

- And since 1987 a series of major die-offs have decimated populations of seals, porpoises, and toothed whales, all dependent upon fish. In each instance, high concentrations of organochlorine chemicals were reported in the dying animals. And the immune systems of the animals appeared to be compromised, although the immediate cause of death was a new virus, similar to canine-distemper virus, but specific to seals or dolphins.

2. Is there laboratory evidence to support concerns about the problems reported in wildlife?

Yes, there is evidence. For example, from Texas A & M there is a large body of information that dioxin and dioxin-like PCB compounds cause wasting, immune suppression, liver enlargement, birth defects in mice, brain damage, and edema to mention a few. From the University of Wisconsin, there is evidence that one meal contaminated with very low levels of dioxin during pregnancy produces demasculinization and feminization in male rat pups, the extent of which depends upon the dose. The male rats differed in body measurements and in a number of biochemical parameters. But, it was not until adulthood that loss of fertility and behavioral changes were fully manifested in the males. The results of this study read like a science fiction best-seller when one considers the small doses of dioxin administered to the pregnant females and the delay between fetal exposure and the full-blown manifestation of the effects a generation time later.

I should like to point out here that because of our fixation on the obvious endpoints such as cancer, gross birth defects, and acute toxicity, it is easy to disregard the results of a study like the dioxin/rat study from Wisconsin. For example, it was reported as early as 1950 that DDT causes demasculinization in young roosters, very obviously reducing the size of their combs and changing their behavior. The report has been buried in the literature for over 40 years.

Recently, the female hormone-like effects of DDT were brought to light again. Researchers at the University of California, Davis, found that like the Great Lakes herring gulls, when researchers exposed western gulls and sparrow hawks in the laboratory to DDT, its metabolites, and the new substitute products for DDT -- kelthane, dicofol and methoxychlor at concentrations found in environmental samples -- the male birds grew oviductal tissues and behaved aberrantly, while the females grew an extra oviduct.

It is also important to mention a number of ongoing studies looking at the effects of feeding Great Lakes fish to animals in the laboratory. At SUNY, Oswego, rats fed Lake Ontario salmon could not cope with stress. They became hyperreactive to slight changes in scenery, food deprivation, and slight electric shock. The time required before the aversive behavior became evident was dependent upon the concentration of fish in the diet. Unlike the low odds when testing for cancer, every rat was affected in these studies. The offspring of these rats also displayed the same behavior, although they had never been fed Lake Ontario fish.

At Michigan State University, white leghorn chickens fed Saginaw Bay carp developed all the common birth defects reported in wild bird colonies around the Great Lakes in a dose response manner. The effects included edema of the abdomen, back, head, and neck; hemorrhaging in the neck and muscles; beak deformities such as missing upper mandibles;

asymmetrical eyes or no eyes; small brains; club feet; and other miscellaneous deformities. Hatchlings died with "unabsorbed and fluid-filled yolk sacs" still attached, suggesting a metabolic disturbance where the chicks were unable to convert the energy in the yolk into body mass. Similar results were reported when mink were fed the same fish. These results confirm earlier studies concerning the exquisite sensitivity of mink to PCBs and explains why mink and otter do not fare well on the Great Lakes shorelines.

3. What do we know about fish consumption and human health?

We know that the same hormonally active chemicals found in fish tissue and wildlife are found in human tissue as well. A number of surveys also show an association between the concentration of certain chemicals in humans and the amount of fish they eat. Since we cannot force people to eat fish in large quantities and then monitor their health and the health of their children, researchers have chosen two routes to reach some conclusions concerning the health effects of eating contaminated fish:

> (1) they have followed the outcome of a group of children whose mothers consumed contaminated fish, and
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> (2) they have turned to a human model that depicts the results of exposure to chemicals that behave like those that accumulate in fish.

(1) In the first case, infants born to women who ate two to three Lake Michigan fish meals for at least six years preceding their pregnancies experienced low birth weight, shorter gestation period, and smaller skull circumference; and cognitive, motor, and behavioral deficits. At age four the results of postnatal testing prevailed. It was determined that there was a "...reasonably consistent pattern of physical growth and short-term memory deficits that appear to be related specifically to prenatal exposure..."[1]. During administration of the psychological tests at age four, 17 youngsters became intractable and refused to take the tests. This behavior is analagous to the aversive behavior of the rats fed Lake Ontario fish mentioned earlier. Later, it was determined that these were the 17 children of seventeen women with the highest PCBs breast milk concentrations in the study. The average PCBs breast milk concentrations of these women fall within the range of normal background. It must also be pointed out here that these children are not mentally retarded -- they are not obviously affected. However, their future potential has been compromised in ways that may never be fully understood or quantified.

(2) In the second approach, scientists from a number of disciplines have turned to the literature concerning the health effects of individuals who were exposed in the womb to an estrogen-like chemical, diethylstilbestrol or DES. "Daughters born to mothers who took DES now suffer increased rates of vaginal [cancer] clear cell adenocarcinomas, various genital tract abnormalities, abnormal pregnancies, and some changes in immune response. Both sons and daughters exposed in utero [in the womb] experience congenital anomalies of their reproductive system and reduced fertility. The effects seen in in utero [in the womb]

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[1] Jacobson JL, Jacobson SW. 1991. Follow-up on children from the Michigan fish-eaters cohort study: performance at age 4. In Cause-Effect Linkages II Symposium Abstracts, Eds. Schneider S, Campbell R. Michigan Audubon Society, p34.

DES exposed humans parallel those found in contaminated wildlife and laboratory animals, suggesting that humans may be at risk to the same environmental hazards as wildlife" (Wingspread Statement of Consensus, 1991).

It was pointed out at the Wingspread meeting that, "The bird model is the best described model to date." since fish are a major pathway of exposure for birds, a shared protein food source for humans. The bird model "...provides support for the wildlife/human connection because of similarities in the development of the avian and mammalian endocrine systems." "Impacts on wildlife and laboratory animals as a result of exposure to these contaminants [found in fish tissue] are of such a profound and insidious nature that a major research initiative on humans must be undertaken."

It is imperative that more groups of fish eating individuals are studied, subjects selected from populations that depend daily on food resources from aquatic systems. For example, it would be expected that Native American infants whose mothers are dependent upon high fish and marine mammal diets are among the most vulnerable. I estimate that PCBs could reach 14 ppm in infant body fat at 6 to 9 months as the result of consuming the highly contaminated breast milk of the mothers who have some of the highest recorded breast milk PCBs concentrations in the world -- this is twice the concentration of PCBs found in the troubled population of Forster's terns that I mentioned earlier.

What I have presented to you is a comprehensive review of the literature on toxics in wildlife and humans in the Great Lakes Basin. It is a collection of independent studies done in isolation: study designs were different; the questions addressed in each study were different; the animal species were different; the human subjects were from different populations; and each study had flaws, limitations, and different analytical chemical and statistical approaches for arriving at its conclusion. However, the results, when reviewed in relation to each other, are consistent and coherent, and call for a concerted effort on the part of the government to seek new approaches to address this 40 year-old problem. The $2 million dollar appropriation to the Agency for Toxics Substances and Disease Registry (ATSDR) for fy 1992 under "The Great Lakes Critical Program Act of 1990" is a step in the right direction. It is imperative that the government continue this support by appropriating the full $3 million for fy 1993 and again, for fy 1994, support that has been authorized under the Act. With this money, the ATSDR can coordinate the assorted wildlife/human health studies in progress around the Great Lakes region in order to fill information gaps and to assure using established cohorts (study groups) to their fullest potential. Many studies in the Great Lakes region are only partially funded and therefore address only part of the questions their studies could address. With adequate resources the ATSDR could respond to situations such as this. By facilitating multi-disciplinary activities, ATSDR can synergize research that has practical application for addressing wildlife and human health, nationwide and globally.

QUESTION II. SOCIO-ECONOMIC FACTORS

You asked if I would address the socio-economic impacts associated with contaminated fish in the Great Lakes. I am not an economist nor a sociologist, however, it is apparent that the presence of thousands of chemicals in the Great Lakes ecosystem cannot be counted as a benefit. The fact that there are 43 areas of concern around the Great Lakes where the harbors are so contaminated that the fish are not edible and the waters are not swimmable -- and that 16 top predator fish-eating species, including the endangered bald eagle, cannot

sustain stable populations in the region -- must have a negative impact on tourism and recreation. Certainly, recreation dollars will be much greater when fish consumption warnings are no longer issued with fishing licenses.

As a matter of fact, the most difficult task we undertook for our book, Great Lakes Great Legacy?, was to produce a cost/benefit analysis chapter. The chore was so difficult that we finally opted for a box in the book instead. I refer you to page 205 for details in the box "What Will it Cost to Clean up Areas of Concern?" We arrived at a figure of between $100 to $500 billion dollars to clean up the Lakes, and went on to say

> Thus, the damage already done to the health of the Great Lakes ecosystem is not likely to be repairable in total, or perhaps even in large part.

The most important socio-economic impacts of contaminants in fish are expressed in our children. To try to address this, one child at time, is impossible. However, from a population perspective we might be able to derive a figure based on loss of functionality, or loss of potential. First, it is important to understand that in their current risk model for arriving at a tolerance for a contaminant in fish, the FDA includes economic factors. These are factored as losses in commercial fish-harvest income. In order to balance the equation, howver, the FDA should also calculate in lost human potential as the result of second generation maternal transfer of the chemical to an offspring. A basis for calculating the cost of this maternal transfer of contaminants found in fish lies in the work of Drs. Sandra and Joe Jacobson, Wayne State University[2]. The Jacobsons administered tests to determine the performance of children exposed in the womb to the chemicals their mothers accumulated over a lifetime of eating Lake Michigan fish. The tests were administered to infants at birth, seven months, and at four years. Although the tests were not designed to predict IQ, if the children's short term memory problem persists as it did for the first four years of their lives, it could lead to lowered IQ scores. To place a price on this lost human potential, I refer you to Weiss (1990)[3] in which he determined that in a population of 100 million, a 5% shift in IQ to the left, representing 1/3 standard deviation, can lead to a loss of 1,310,000 individuals scoring above an IQ of 130. Costing the loss of productivity and talent of these individuals at the top of the curve is probably impossible, but at the other end of the curve, costing the special education required for those individuals dropping below an IQ of 80 is possible.

QUESTION III. THE ADEQUACY OF FDA GUIDELINES AND STATE FISH ADVISORIES

The answer to question three is no. In most instances there is little or outdated scientific data to support the present fish advisories. In many instances, economic considerations

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[2] Jacobson JL, Jacobson SW, Humphrey HEB. 1990. Effects of in utero exposure to polychlorinated biphenyls and related contaminants on cognitive functioning in young children. The Journal of Pediatrics 116(1):38-45.

[3] Weiss, B. 1990. The scope and promise of behavioral toxicology. In Behavioral Measures of Neurotoxicity. Russell RW, Flattau PE, Pope AM Eds. National Academy Press, Washingtno, DC 395-413.

outweighed scientific evidence when setting the levels of concern. In addition, current fish advisories are not adequate to protect consumers against the adverse reproductive and developmental health outcomes that may be associated with contaminated fish consumption.

Current advisories for PCBs and dioxins are based on the the risk of contracting cancer, DNA damage, and in the case of mercury, neurological damage by the individual who consumes the fish. The individuals who may be most at risk -- the children of people who eat contaminated fish -- have not been considered.

Until transgenerational effects are included in the FDA's evaluation of the hazards of eating fish, public health problems will not be adequately addressed. These effects should include the difficult-to-recognize, insidious loss of function of the major organ systems -- the digestive, endocrine, immune, and nervous systems -- in the children of those who eat fish.