STATEMENT OF DOCTOR HARRY M. OHLENDORF, ASSISTANT DIRECTOR OF THE PATUXENT WILDLIFE RESEARCH CENTER, U. S. FISH AND WILDLIFE SERVICE, DEPARTMENT OF INTERIOR, BEFORE THE U.S. HOUSE OF REPRESENTATIVES MERCHANT MARINE AND FISHERIES COMMITTEE, ON THE EFFECTS OF CONTAMINANTS ON FISH AND WILDLIFE, MAY 21, 1980.

Mr. Chairman, I appreciate the opportunity to appear here today to testify on the problems related to the disposal of dredged material in ocean waters. The Fish and Wildlife Service has been concerned about the effects of contaminants on fish and wildlife resources and has contributed to the scientific data base for some time.

Fishery research in relation to environmental contaminants is conducted at the Columbia National Fishery Research Laboratory, located at Columbia, Missouri, and the Great Lakes Fishery Laboratory at Ann Arbor, Michigan. Wildlife research on the effects of contaminants is conducted primarily at the Patuxent Wildlife Research Center, Laurel, Maryland. Since being employed at the Patuxent Center in 1971, I have personally carried out several studies to determine levels of pollutants and their possible impacts on estuarine and marine birds. My testimony is based on those studies, the work of other scientists in the Fish and Wildlife Service, and published and unpublished reports on the effects of persistent environmental pollutants. One of my publications (Ohlendorf et al. 1978b, a review of literature concerning the exposure of marine birds to environmental pollutants) may be of interest to the Committee in relation to matters being considered here and is available on request.

The widespread distribution of persistent pollutants, such as organochlorine pesticides, polychlorinated biphenyls (PCBs), and heavy metals, in the environment and their effects upon populations of wild animals are matters of considerable concern. In this testimony I will focus on PCBs, but many of the observations also apply to other persistent compounds that may be present in sediments subject to dredging and ocean disposal. We know more about the direct effects of PCBs on fish, birds, and mammals than we do about their effects on other important ecosystem components. Although we do not fully understand the complex toxicological interaction of PCBs with other contaminants (such as petroleum hydrocarbons, pesticides, and heavy metals), there are indications that PCBs in sediments may pose hazards for some kinds of animals. Fish and wildlife resources in the New York Bight may be adversely affected by resuspending or recycling of PCBs that are found in sediments of the Hudson River estuary.

Our concerns focus on the effects of contaminants on waterfowl, birds that prey on marine life, marine mammals, and anadromous fish. We are specifically concerned about long-term, sublethal effects on reproduction, behavior, susceptibility to disease and predation, latent toxicity, and the synergistic and cumulative effects of ocean dumping of a wide variety of contaminated materials over a long period of time.

In a general sense, it is important to note that PCB residues in fish and birds are higher in the Northeast and Great Lakes States than in other regions of the country. Residues in fish samples collected from large river systems in the Northeast are the highest in the United States (O'Shea and Ludke 1979;

Walker 1976a, 1976b). PCB residues are higher in mallards and black ducks (White and Heath 1976; White 1979) and in wading birds (Ohlendorf et al. 1978a, 1979) from this region than elsewhere.

The New York Bight area may already be too polluted. Analyses (O'Connor et al. in ms.) of samples of dredged material from four areas of the New York Bight revealed 15 different compounds of one of the most hazardous groups of toxicants and carcinogens--the polynuclear aromatic hydrocarbons (PAH). Several of these PAH compounds occurred in the parts-per-million (ppm) range, which is high for these chemicals. PCB levels in the dredge samples ranged from 188 to 731 parts per billion (ppb). In addition, traces of organochlorine pesticides were found in three of the four areas.

PAH compounds also were found in the flesh and the eggs of edible fishes. PCBs were present in all tissues of all fish samples. The highest level was 11.9 ppm PCB in a striped bass--a favorite food and game fish. Lobsters and rock crabs also contained a broad range of chlorinated hydrocarbons, including PAH compounds and PCBs.

Clearly, the area is already contaminated. From the standpoint of man, many fish from the area would exceed the Food and Drug Administration (FDA) PCB standard of 5 ppm. FDA is presently proposing to reduce the 5 ppm standard to 2 ppm. If implemented, many organisms would exceed this new standard severalfold.

PCBs are not permanently bound to sediments. They can move into the nearby water and into the animals (Young et al. 1977). Our own scientists have recently proven that in fresh water, fish can rapidly accumulate PCBs and other contaminants directly from resuspended sediments (Seelye 1980).

PCBs are freely accumulated from the water by animals. Scores of studies reveal that the amount of PCB in aquatic animals can be thousands of times that in the water. This bioconcentration may be over 100,000 times and is known to reach more than a million times in large fish that have a high fat content (International Joint Commission 1977). For this reason, mere trace amounts of PCB in water commonly create levels of PCB in animals that are dangerous to them, to their young, or to the animals that feed upon them.

What level of PCB in fish can be considered safe for the animals that eat them? Expert groups have concluded that this level is 0.1 ppm (Veith et al. 1979; International Joint Commission 1977). This level of 0.1 ppm was arrived at by considering the amount in the diet that would disrupt reproduction in mink and other animals that are equally sensitive to PCBs. However, this value is minimally protective because 0.3 ppm has been suspected of affecting reproduction in mink; 0.64 ppm had drastic effects. This safe level is far exceeded in many of the animals from the New York Bight. Many have PCB residues in the tenths of a ppm range or greater.

A short distance up the Hudson River, near Poughkeepsie, PCB residues in fish are very high. They began at 2-9 ppm in 1969, increased to 5-75 ppm in 1974, were 25-70 ppm in 1976, and reached 12-92 ppm in 1978 (Columbia National

Fishery Research Laboratory, unpublished). Much of the PCB represented by these samples is bound to have been transported downstream with water and sediments; some of it probably entered the sediments that are the subject of this hearing. We must also ask what happens to anadromous fish that work their way upstream through such contaminated reaches.

Striped bass populations have declined sharply in recent years. Ongoing studies by the Service, supplementally funded by amendments to the Anadromous Fish Conservation Act, have shown that one factor in this decline is contamination. Striped bass from the Hudson River were high in PCBs and relatively high in lead and cadmium. The Hudson River fish were exceptional in having a 42 percent reduction in the strength of their vertebrae. This reduction was related to density and chemical composition (collagen, phosphorus and calcium) of the bone. The weakness would reduce the ability of the bass to compete for food, to avoid predation, and to respond to the stresses of reproduction and migration (Mehrle 1980). The residues that correlated with this weakness were around 3 ppm of PCB in the body.

This work was supported by experiments with trout. Collagen and phosphorus declined in treated fish and the calcium concentration rose, leading to fragile vertebral columns. The level of PCB in water low enough to avoid this effect was below 0.43 ppb (Mauck et al. 1978).

Many other authors have reported ill effects with small amounts of PCB in sensitive organisms:

"Aroclor 1254 [a PCB compound] at 0.45 ug/l (parts per billion) in water produced a 50 percent reduction in midge reproduction; 1.3 ug/l caused a 50 percent reduction in Daphnia reproduction; and 1.8 ug/l produced 50 percent reduction in fathead minnows. The indirect toxicity of PCBs to predators through accumulation of PCBs in tissues of food organisms causes death." (Nebeker 1976).

"Concentrations that were lethal to selected invertebrates and fishes in chronic exposures ranged from 0.1 to 5 ug/l." (Hansen 1976). Thus, in this case as little as one-tenth part per billion was lethal.

Reproduction of the fathead minnow was reduced by as little as 0.4 ug/l in water. As often happens, the young were the most sensitive and died soon after hatching (DeFoe et al. 1978).

In a saltwater fish, the sheepshead minnow, survival of young was reduced when there was PCB in water at 0.32 ug/l (Schimmel et al. 1974).

Trout eggs that contained 2.7 ppm of PCB had 75 percent mortality and produced many abnormal young (Hogan et al. 1975).

Eggs of Atlantic salmon that contained from 0.4 to 1.9 ppm of PCB experienced mortalities of 16 to 100 percent. Mortality rose as the PCB content rose, and there was a strong coefficient of correlation (Jensen et al. 1970, reported in Walker 1976b).

It is known from many studies that PCBs and other pollutants can reduce the resistance of animals to disease and that unrelated pollutants can be additive, or at times even synergistic, in their effects. Unfortunately, most of this work

has been done with relatively high dosages and therefore provides little proof of what will happen in the field with low residue levels. However, the work of Bills et al. (1977) is noteworthy. They exposed one group of trout to 0.01 ug/l of Aroclor 1254 in water for 30 days and another group to 0.1 ug/l. The fish then contained averages of 0.46 ppm and 3.40 ppm of PCB. Both groups were then tested against cyanide and chromium. Both groups were more sensitive to cyanide than were the controls. The 0.1 ug/l group was also more sensitive to chromium. Such results were not obtained with other pollutants. The study shows that low exposures to PCBs can alter the susceptibility of fish to certain other contaminants.

When administered as an acute or sub-acute dosage, PCBs are not as toxic to birds as are DDT, dieldrin, and most other organochlorine insecticides (Heath et al. 1972; see also Stickel 1975 and Stendell 1976 for review). Aroclor 1254 was was about as toxic as DDE (the persistent breakdown product of DDT) to four species of blackbirds. With coturnix quail the toxic effects of Aroclor 1254 and DDE were additive but not synergistic. It is unlikely that birds would acquire lethal levels of PCBs through acute exposure via their food webs as a result of ocean dumping of dredge material from New York Harbor. When various small birds were killed with heavy dietary dosages of Aroclor 1254, they displayed signs of neurotoxicity - tremors and traces of convulsive behavior. Residues of PCBs in brains were highly diagnostic: from 349 to 763 ppm in the dead and not above 301 ppm in survivors (W. H. Stickel, Patuxent Wildlife Research Center, unpublished report). Other workers have reported high PCB residues in brains of dead birds (see Stickel 1975). In the field, long-term, low-level intake would be usual and may kill by causing edema

and related phenomena; signs vary between individuals and between species. Under these circumstances, mortality resulting from PCBs would not necessarily be accompanied by high residues of PCBs in brains, and for these birds no good diagnostic technique is available, for the signs are nonspecific and the residue levels vary greatly.

Effects of PCBs on reproductive and survival ability are difficult to evaluate but are apt to have the most serious impact on populations (Stendell 1976). Few data are available for wild species, but some pertinent studies have been published.

Reproductive success of mallards was not affected when they received 25 ppm Aroclor 1254 in their diet. In one study (Heath et al. 1972) the eggs were artificially incubated and young were reared apart from the female. More recently another study (Custer and Heinz 1980) was conducted to test the effects of 25 ppm Aroclor 1254 on the reproductive success and nest attentiveness of mallards allowed to incubate their own eggs. The latter study was conducted because other investigators (Peakall and Peakall 1973) had reported reduced reproductive success in ring doves fed 10 ppm Aroclor 1254 and allowed to incubate their own eggs. They demonstrated that incubation temperature was more variable in the nests of treated doves and suggested that lower success was due to abnormal incubation behavior. These effects, however, were not exhibited by the mallards, as noted above.

In these studies, as well as some others discussed later, the dietary concentrations of PCBs were mixed into a commercially available mash diet that contained less than 10 percent water. Had the mash contained as much water

as do natural diets of wild mallard ducks (75-90%), the concentrations of PCBs in the diet would be about 3.8 ppm (see Heniz 1975 for further discussion).

Residue concentrations of PCBs in the mallards averaged about twice those in their food (55 ppm in hens, 64 ppm in drakes); those in eggs (23 ppm) were similar to levels in the food (Custer and Heinz 1980). When residues in the feed are expressed on the basis of the normal wet diet (3.8 ppm) the concentrations found in the duck carcasses were about 15 times those in the diet, those in the eggs about 6 times. However, these findings suggested no reproductive impairment in wild mallards due to PCB since mean PCB levels for carcasses were considerably higher than those found in field-collected samples (White and Heath 1976; White 1979).

Field levels of PCBs in other species can be very high and may be adversely affecting their reproduction (Stendell 1976). For example, the median level of PCBs in eggs of herring gulls from Lake Ontario was 142 ppm and the hatching success of artificially incubated eggs from this area was lower than in other areas with lower egg residues (Gilman et al. 1977).

Lake Ontario herring gulls did not defend their nests in the normal manner and were less attentive than those from less polluted colonies (Fox et al. 1978). Incubating Lake Ontario gulls appeared to apply less heat to their eggs. Within Lake Ontario, a comparison of nest attentiveness and air temperatures in the gull nests between successful and unsuccessful nests revealed similar differences. The decreased nest defense of Lake Ontario gulls was sufficient to account for the high incidence of egg loss observed. The variability in nest air temperature was sufficient to increase embryonic mortality

based on studies in other species. The authors suspected that these abnormalities resulted from pollutant-induced endocrine dysfunction in the colonies that were more heavily contaminated with PCBs and other organochlorine pollutants.

Behavioral aberrations have been suspected as a cause of lowered reproduction in heavily contaminated birds in other areas (Milstein et al. 1970; Ratcliffe 1970; Cooke et al. 1976). Because it is difficult to determine whether environmental contaminants have caused abnormal behavior in wild birds, contaminant-induced changes in behavior have been studied primarily in the laboratory. Aroclor 1254 at 10 and 100 ppm in the diet of ring doves caused severe depletions in two neurotransmitters (dopamine and norepinephrine) in the brains of treated birds (Heinz et al. 1980). Neurotransmitters play a role in many basic functions, such as eating, drinking, locomotion, sleep, coordination, grooming, and aggression, in a wide variety of species (see Heinz et al. 1980 for further discussion). Depletions of these neurotransmitters could result in abnormal behavior of contaminated birds in the wild, and the detection of such depletions could become an important tool in assessing contaminant-induced behavioral aberrations in birds.

When common murres were fed high doses of Aroclor 1254 they showed dose-related effects on the thyroid (Jefferies and Parslow 1976). These changes suggest a direct effect of the PCB on the avian pituitary, causing a dose-related decrease in thyroid function. This action on the pituitary, producing secondary hypothyroidism, could be an important sublethal lesion caused by these materials.

Because so few experimental studies of PCB effects have been conducted with wild species, work done with chickens by U.S. Department of Agriculture, and others, should be considered. The chief reproductive effects of PCBs in chickens are lowered egg production, reduced hatchability, deformities in chicks, and reduced survival and growth of young (see Stickel 1975 and Stendell 1976).

Reproduction of chickens was not adversely affected when they were fed 2 ppm of PCBs in their diet (Platonow and Reinert 1973; Lillie et al. 1974). Five ppm of Aroclor 1254 reduced egg production erratically and with 50 ppm, mortality occurred. Residues over 10 to 15 ppm in eggs were accompanied by heavy embryotoxicity, but those below 5 ppm showed no effect.

PCB residues in eggs of wild birds have often equalled or exceeded the levels known to have caused reproductive problems in chickens. For example, 11 eggs of bald eagles had from 2.2 to 28 ppm of PCBs with a median of 9.7 ppm (Wiemeyer et al. 1972). The PCB concentrations in three bald eagle eggs from a declining population in northwestern Ontario were 25, 30, and 166 ppm (Grier 1976). PCB residues in osprey eggs collected in Connecticut in 1968 and 1969 varied from 3.6 to 51 ppm (Wiemeyer et al. 1975). When the average residue content for eggs from a nest equalled or exceeded 12 ppm DDE, 1 ppm dieldrin, or 15 ppm PCB, no eggs from that nest were known to hatch. Brown pelican eggs from the Eastern United States contained 1.9 to 36.5 ppm PCBs (Blus et al. 1974).

In 1972 and 1973, we collected eggs of wading birds (herons, ibises, and related species) throughout the Eastern United States, and especially along the Atlantic Coast from Florida to Massachusetts. For great egrets, snowy egrets, and glossy

ibises average PCB residue levels in samples from Long Island were higher (8.6, 7.9, and 0.59 ppm) than those from any other nesting colonies we sampled (Ohlendorf et al. 1979). In black-crowned night herons the average PCB residues in eggs from Long Island also were high (8.4 ppm), but average residues in eggs from Massachusetts (22 and 12 ppm), Rhode Island (10 ppm), and Michigan (9.9 ppm) were still higher. PCB residues reached 35 ppm in one egg from the House Island, Massachusetts colony and exceeded 50 ppm in some eggs from Boston Harbor, Rhode Island, and Long Island colonies. The black-crowned night heron experienced population declines in New England and the Great Lakes States during the 1950s and 1960s. Although we could not positively relate the organochlorine or heavy metal residues we found in eggs to the declines of night heron populations, evidence suggests that environmental pollutants may contribute to impaired reproduction in the more contaminated areas (Ohlendorf et al. 1978a).

Fish-eating birds in the vicinity of the lower Great Lakes are heavily contaminated by PCBs and other pollutants, and severe reproductive failure has been related to these contaminants (Gilbertson 1974; Gilbertson and Hale 1974; Gilbertson and Fox 1977). Whereas eggshell thinning has been correlated with DDE content of eggs, there is a positive correlation between early embryonic mortality and PCB contamination. Chicks also exhibit symptoms of chick edema disease; symptoms are subcutaneous and abdominal edema, porphyria, liver necrosis, and mortality.

Justifiable concern about the effects of PCBs on man himself was expressed by Allen et al. (1976): "Female rhesus monkeys exposed to dietary levels as low as 2.5 and 5.0 ppm of PCB (Aroclor 1248) developed facial acne, erythema, subcutaneous edema, conjunctivitis, and loss of eyelashes. Reproductive

dysfunctions were manifested by irregular menstrual cycles, early abortions, and stillbirths. As a result of transplacental migration of the compounds, all infants born of PCB-exposed animals contained PCBs in their tissues at birth. The infants, which continued to be exposed to PCBs by ingestion of milk from their lactating mothers, developed skin lesions and 50 percent expired within 4 months." It would not have been expected from work with the usual laboratory mammals, but reproductive effects did appear at the surprisingly low level of 2.5 ppm in the diet.

Even more surprising and unusual were the results with mink. Growers found that they could not rear mink if they fed them fish from the Great Lakes. This led to a series of studies (Aulerich et al. 1971, 1977; Ringer et al. 1972; Platonow et al. 1973). The latter dosed cows with Aroclor 1254 and used the meat as part of the mink ration. The concentration of PCB in the two diets was 3.57 ppm and 0.64 ppm. When the 3.57 ppm diet was fed to mink, there was no reproduction and all breeders (12 females and 4 males) died within 105 days. With the 0.64 ppm diet, 2 of the 12 females died. Of the rest, only one produced a litter, and those kits died at once. Among the many untreated mink, reproduction fell from the expected 4 per litter to 1.8. Analysis of the untreated diet revealed 0.3 ppm PCB, and the authors considered this responsible for the effects they observed. No sign of disease was found in the mink.

In both sets of mink studies, tissue from fish or cows was more toxic than expected from the amount of PCB it contained. Various authors have concluded that the increased toxicity of PCB that has gone through an animal is caused by the formation of a toxic metabolite in animals. This means that the genuine danger in the field is considerably greater than experimenters would judge from

relatively short-term tests in which straight PCBs were used. The mink work has received much attention from those concerned with setting water quality standards and determining safe levels in animal tissues. It has also been of interest to researchers studying the impacts of contaminants on marine mammals.

Sea lions of Southern California produced a small percentage of premature young, most of which died (DeLong et al. 1973). Affected females proved to contain several times as much PCB and DDE as did normal females. PCBs in livers ranged from 3.4 to 9.7 ppm. This does not seem high for a mammal, but some scientists believe that PCBs explain the problem.

Koeman suggested in 1973 that deaths of seals in the Netherlands might be explained by PCBs. The idea is difficult to prove or disprove, for neither residue levels nor pathology are highly diagnostic of death from PCBs.

The occurrence of aborted seal pups in the Baltic was tentatively attributed to large residues of PCBs and DDE by Olsson et al. (1975). Similarly, Helle et al. (1976a) pointed out that in the Bothnian Bay, where residues were high, only 27 percent of ringed seals of reproductive age were pregnant. Helle suggested that PCB was the probable cause, and therefore the probable cause of the rapid decrease of seals in the Baltic Sea. Later (1976b) Helle et al. pointed out that some 40 percent of Baltic ringed seal females of reproductive age had their uterine horns closed by stenosis and occlusions, which blocked passage of the ova. He felt that PCB was strongly indicated as the cause of the difficulties.

PCB, as well as several metals, was shown to alter the production of hormones by testes of gray seals with *in vitro* studies (Freeman et al. 1977).

The case for PCBs reducing the reproductive success of seals and sea lions is not wholly proven, but there is strong indicative evidence, and when compared to the mink studies the probability is increased. The levels of PCB that may affect reproduction of seal populations are not well known, but reports of trouble come from areas of relatively high PCB pollution.

Mr. Chairman, this concludes my formal statement. I will be happy to answer any questions you may have.

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