Exhibit 102

STATEMENT OF THOMAS W. DUKE

(1)

Ref 5 Nimmo

p-197 — Arolor caused no deaths in shrimp

last fish or shrimp do not reproduce under test conditions

he is an Aggie

My name is Thomas W. Duke. I am the [redacted] of the [redacted] I have directed research activities at this laboratory since 1968. The mission of the Gulf Breeze Laboratory is to study the effects of toxic organics on marine organisms.

My educational background is as follows: I received my B.S. degree in Zoology from Texas A & M University in 1953, my M.S. degree in Oceanography from Texas A & M University in 1960 and my Ph.D. in Oceanography from Texas [redacted] University in 1962. A list of my scientific publications is appended to this testimony.

My testimony will be taken from completed and ongoing research, as well as pertinent results of related studies in the literature. My testimony is concerned with the routes, rates and reservoirs of Aroclor® 1254 in Escambia Bay, Florida and with the effects of various Aroclors on marine organisms as determined in the laboratory.

The following exhibits are included in my testimony:

1. Duke, T. W., J. I. Lowe, and A. J. Wilson, Jr. 1970. A Polychlorinated Biphenyl (Aroclor® 1254) in the Water, Sediment, and Biota of Escambia Bay, Florida. Bulletin of Environmental Contamination and Toxicology, Volume 5, No. 2, pages 171-180.

(X) – Move to strike because we [illegible] them –

not relevant to 1969 - to 1971 (X)

2. Nimmo, D. R., D. J. Hansen, J. A. Couch, N. R. Cooley, P. R. Parrish, and J. I. Lowe. 1974. Toxicity of Aroclor® 1254 and Its Physiological Activity in Several Estuarine Organisms. Archives Environmental Contamination and Toxicology. [redacted]

3. Nimmo, D. R., P. D. Wilson, R. R. Blackman and A. J. Wilson, Jr. 1971. Polychlorinated Biphenyl Absorbed from Sediments by Fiddler Crabs and Pink Shrimp. Nature, Volume 231, No. 5297, pages 50-52.

4. Wilson, A. J., Jr. 1972. Escambia Bay PCB Survey. [redacted]

5. Nimmo, D. R., R. R. Blackman, A. J. Wilson, Jr. and J. Forester. 1971. Toxicity and Distribution of Aroclor® 1254 in the Pink Shrimp Penaeus duorarum. Marine Biology, Volume 11, No. 3, pages 191-197.

(X) 6. Nimmo, D. R., J. Forester, P. T. Heitmuller and G. H. Cook. 1974. Accumulation of Aroclor® 1254 in Grass Shrimp (Palaemonetes pugio) in Laboratory and Field Exposures. Bulletin of Environmental Contamination and Toxicology. [redacted]

7. Staff, Gulf Breeze Environmental Research Laboratory. 1969-1974. Aroclor® 1254 in Escambia Bay System. [redacted]

8. Proceedings. 1971. Second Session of Conference in the Matter of Pollution of the Interstate Waters of Escambia River Basin (Alabama-Florida) and the Intrastate Portions of the Escambia Basin and Bay within the State of Florida. Second Session.

9. Proceedings. 1972. Third Session of Conference in the Matter of Pollution of the Interstate Waters of Escambia River Basin and the Intrastate Portions of the Escambia Basin and Bay within the State of Florida. Third Session.

10. Staff, Gulf Breeze Environmental Research Laboratory. 1969-1974. Aroclor®1254 in Selected Oysters from Escambia Bay, Florida. Unpublished data.

11. Cooley, N. R., J. M. Keltner, Jr. and J. Forester. 1973. The Polychlorinated Biphenyls, Aroclors®1248 and 1260: Effect on and Accumulation by Tetrahymena pyriformis. Journal of Protozoology, Volume 20, No. 3, pages 443-445.

12. Lowe, Jack I. 1969. Gulf Breeze Environmental Research Laboratory - Bioassay Screening Test on Aroclor® 1248 and 1260. Unpublished data.

13. Cooley, N. R., Keltner, J. M., Jr. and J. Forester. 1972. Mirex and Aroclor®1254: Effect on and Accumulation by Tetrahymena pyriformis Strain W. Journal of Protozoology, Volume 19, No. 4, pages 636-638.

14. Wildish, D. J. 1972. Polychlorinated Biphenyls (PCB) in Sea Water and Their Effect on Reproduction of Gammarus oceanicus. Bulletin of Environmental Contamination and Toxicology, Volume 7, No. 2/3, pages 182-187.

15. Wildish, D. J. and V. Zitko. 1971. Uptake of Polychlorinated Biphenyls from Sea Water by Gammarus oceanicus. Marine Biology, Volume 9, No. 3, pages 213-218.

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16. Parrish, P. R. 1972. Gulf Breeze Environmental Research Laboratory - Bioassay Screening Test on Aroclors® 1242 and 1254. Unpublished data.

17. Bourquin, A. W. 1974. Growth Responses of Estuarine Bacteria to Mirex, Heptachlor and Polychlorinated Biphenyls. Unpublished manuscript.

18. Kinter, W. B., L. S. Merkens, R. H. Janicki and A. M. Guarino. 1972. Studies on the Mechanism of Toxicity of DDT and Polychlorinated Biphenyls (PCB's): Disruption of Osmoregulation in Marine Fish. Environmental Health Perspectives, Volume 1, pages 169-173.

19. Hansen, D. J., P. R. Parrish and J. Forester. 1974. Aroclor® 1016: Toxicity to and Uptake by Estuarine Animals. Environmental Research. (In press)

20. Staff, Environmental Protection Agency's Gulf Breeze Environmental Research Laboratory. 1974. Toxicity of Aroclors 1260, 1254, 1248, 1242 and 1016 to Salt Water Organisms in 48- and 96-hour Bioassays. Unpublished summary.

21. Lowe, J. I., P. R. Parrish, J. M. Patrick, Jr. and J. Forester. 1972. Effects of the Polychlorinated Biphenyl Aroclor® 1254 on the American Oyster Crassostrea virginica. Marine Biology, Volume 17, No. 3, pages 208-214.

22. Parrish, Patrick R. 1974. Aroclor® 1254, DDT and DDD, and Dieldrin: Accumulation and Loss by American Oysters (Crassostrea virginica) Exposed Continuously for 56 Weeks. Proceedings of National Shellfisheries. (In press)

23. Couch, John A. 1974. Free and Occluded Virus, Similar to Baculovirus, in Hepatopancreas of Pink Shrimp. Nature, Volume 147, No. 5438, pages 229-231.

24. Nimmo, D. R. and L. H. Bahner. 1973. Physiological Consequences of Polychlorinated Biphenyl and Salinity-stress in Penaeid Shrimp. Symposium on Pollution and the Physiological Ecology of Estuarine and Coastal Water Organisms, November 14-17, 1973, Hobcaw Barony, South Carolina.

25. Wildish, D. J. 1970. The Toxicity of Polychlorinated Biphenyls (PCB) in Sea Water to Grammarus Oceanicus. Bulletin of Environmental Contamination and Toxicology, Volume 5, No. 3, pages 202-204.

26. Hansen, D. J., P. R. Parrish, J. I. Lowe, A. J. Wilson, Jr. and P. D. Wilson. 1971. Chronic Toxicity, Uptake, and Retention of Aroclor® 1254 in Two Estuarine Fishes. Bulletin of Environmental Contamination and Toxicology, Volume 6, No. 2, pages 113-119.

27. Schimmel, Steven C., David J. Hansen, and Jerrold Forester. 1974. Effects of Aroclor® 1254 on Laboratory-reared Embryos and Fry of Sheepshead Minnows (Cyprinodon variegatus). Transactions of the American Fisheries Society. (In press)

28. Hansen, D. J., S. C. Schimmel and J. Forester. 1974. Aroclor® 1254 in Eggs of Sheepshead Minnows: Effect on Fertilization Success and Survival of Embryos and Fry. Proceedings of the Southeastern Game and Fish Commission. (In press)

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29. Johansson, N., S. Jensen and M. Olsson. 1970. PCB - Indications of Effects on Fish. In "PCB Conference, Wenner-Gren Center," September 29, 1970, pages 59-68. National Environmental Protection Board, Stockholm.

30. Hansen, David J. 1974. Aroclor® 1254: Effect on Developing Estuarine Animal Communities in the Laboratory. Contributions to Marine Science. (In press)

31. Hansen, D. J., S. C. Schimmel and J. Forester. 1974. Effects of Aroclor® 1016 on Embryo, Fry, Juvenile and Adult Sheepshead Minnows (Cyprinodon variegatus). Manuscript approved by Gulf Breeze Environmental Research Laboratory.

32. Staff, Environmental Protection Agency's Gulf Breeze Environmental Research Laboratory. 1974. Toxicity of Aroclors 1254 and 1016 to Salt Water Organisms in Flow-through Bioassays Exceeding 96-hour Duration. Unpublished summary.

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ROUTES, RATES AND RESERVOIRS OF AROCLOR® 1254 IN ESCAMBIA BAY, FLORIDA

We detected the polychlorinated biphenyl, Aroclor® 1254, in the water, sediment and biota of Escambia Bay in 1969 (Exhibit 1) and continue to follow the movement and accumulation of this chemical within the bay system. Only one source of the chemical, the Monsanto chemical plant on the Escambia River, has been located at this time. The Aroclor reportedly entered the plant's effluent through accidental leakage of a heat exchange fluid. The leakage was stopped after the company was notified of the appearance of PCBs in the bay system. However, a small amount of this chemical continues to enter the system, possibly from leaching from contaminated sediments into the plant's effluent ditch. Several conclusions have been reached from this study:

(1) Much of the chemical entering the water of Escambia River appeared in particulate form in the water samples

(2) Aroclor® 1254 was distributed from the point of entry in the Escambia River to sediment and biota in the Escambia Bay system by physical or biological transport

(3) Some of the organisms containing residues of Aroclor® 1254 are used as seafood by man

(4) Although the quantity of the Aroclor in the Escambia River was reduced drastically, residues continue

to appear in oysters approximately 8 miles downstream.

Exhibit 1 summarizes the history of the discovery of the chemical in the bay. Exhibit 2, Table 1, gives Aroclor® 1254 concentrations in water, sediment and biota from September 1969 to December 1971 after the leakage was stopped. By comparison, samples of sediments had high concentrations ranging from non-detectable to 30.0 mg/kg (dry weight). In the initial survey (1970) of sediments from the river and bay, concentrations were high at the source of the pollutant (Exhibit 3, Fig. 1; see below the industry) and also at the mouth of the river (Exhibit 3, Fig. 1). Other surveys (1970-1972) show that concentrations of Aroclor® 1254 in sediments from three locations in the river and bay appear to decrease with time (Exhibit 2, Fig. 3). Also, the concentrations in sediment strata appear to decrease with depth. While the number of samples was inadequate for absolute comparison, these data indicate a trend. Data from the latest survey (1972) taken in the channel (Exhibit 4, page 4, see Transect A-middle) indicate that in November, 1972, the sediments above the trestle still had considerable amounts of Aroclor® 1254. Concentrations ranged from 0.16 to 9.0 mg/kg (dry.weight).

The sediment reservoir of Aroclor® 1254 is probably a source of this chemical to the biota of the bay because residues continued to appear after the amount in the water was reduced drastically. For example, the initial survey of biota

(Exhibit 1, Table 1) showed biota with levels as high as 12.0 mg/kg. In surveys conducted in 1969-70, shrimp collected far from the original source had whole body concentrations as high as 14 mg/kg ppm wet weight [redacted] Concentrations of Aroclor found in species collected in Escambia and East Bays are shown in [redacted] The biota in East Bay are about 35 kilometers from the original source of the chemical, and yet have detectable levels of the chemical. There is also a higher concentration of Aroclor® 1254 in higher trophic levels in both East and Escambia Bays' biota which could implicate a food chain transfer from sediment to large animals.

Three separate studies have been conducted to study the capacity of benthic animals to accumulate Aroclor® 1254 from contaminated sediments. The first of these [redacted] showed that shrimp placed on various naturally-occurring substrates containing this PCB accumulated the chemical in proportion to the concentration of the chemical in the sediment. The second study showed that penaeid shrimp (Penaeus duorarum), placed on Aroclor® 1254-contaminated sediments, in upper Escambia Bay, accumulated the material [redacted] Grass shrimp, Palaemonetes pugio, exposed for 3 months to sediments in the bay accumulated Aroclor® 1254 to about 0.4 mg/kg whole body [redacted] In the third study.

Additional data on the levels of Aroclor® 1254 in the water of the Escambia Bay system are shown in Exhibit 7. We analyzed both filtered and unfiltered samples of surface and bottom water and monitored water from the new outfall that the industry constructed in 1971. Duplicate samples were also analyzed periodically, and in general, there was good agreement. The water column was fairly homogeneous judging from the consistency of surface to bottom samples. But in filtered samples of water, little Aroclor® 1254 was recoverable. This observation probably means that most of the chemical was attached to suspended particulates in the river water. Other evidence of this is shown in Exhibit 8, Fig. 1; Exhibit 9, Fig. 1, where waters in Escambia Bay show only trace levels (0.05 to 0.05 µg/l), whereas the levels in the sediments are relatively high (Exhibit 8, Fig. 2; Exhibit 9, Fig. 2).

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The Aroclor® 1254 concentrations from a new outfall constructed by the industrial plant in 1971 are listed in Exhibit 7. Levels of this PCB continue to be above 0.03 µg/l but well below those found in the initial surveys (1969). Some Aroclor® 1254 is now found in the river above the industry and we assume this is due to the effects of high tides and winds moving surface waters slightly up the river.

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We originally discovered Aroclor® 1254 in the bay in oysters from Devil's Point Light and we have continued to

monitor these mollusks (Exhibit 10). The increases in Aroclor® 1254 levels in May to July each year are probably due to the increase in egg mass which could have a high affinity to these chemicals because of a high lipid content. The levels drop shortly after the oysters spawn. This phenomenon also is observed in the laboratory. If the data from Devil's Point Light are analyzed by linear regression analysis it is projected that these mollusks would reach the <0.03 µg/g level by 1985. This prediction assumes that no more chemical is put into the system and that climate, hydrologic or other factors remain the same.

EFFECTS OF AROCLORS ON MARINE ORGANISMS

At the time Aroclor® 1254 was discovered in Escambia Bay, essentially no data existed on the effects of this chemical or other PCBs on marine organisms. Since it is difficult and sometimes impossible to determine toxic effects of specific chemicals in the natural environment because of the presence of other chemicals and fluctuating environmental conditions, we conducted specific toxicity test with Aroclor® 1254 and other Aroclors under controlled conditions in the laboratory. In order to make our bioassay information as meaningful as possible, we conducted most of our tests with indigenous organisms from Escambia Bay or similar areas, and utilized flowing seawater when possible. The toxicity of the test chemicals was determined for specific marine organisms for a given period of time. Detailed test conditions are given in the exhibits.

Residue analyses were conducted where possible to determine if the animals concentrated the chemical from the water in which they were tested. This is an important aspect of these bioassays because Aroclor® 1254 occurred in animals from Escambia Bay that are used by man for food. Obviously, these animals could serve as a vector for the movement of Aroclor® 1254 from site of application to humans. If residues in marine organisms were to exceed levels deemed harmful for human consumption, the fishery resource could not be harvested and would be adversely affected. Consequently, the well-being of commercial fishermen and those with related occupations would be adversely affected.

The following conclusions were reached from laboratory bioassay experiments:

(1) All of the Aroclors tested are acutely toxic for certain estuarine organisms.

(2) Bioassays lasting longer than 96 hours demonstrate that short-term or acute tests underestimate toxicities of some of the Aroclors.

(3) Bioassays lasting over 96 hours indicate that Aroclor® 1254 is toxic to commercially valuable shrimps at concentrations less than 1 µg/l and its toxicity is greatest to young shrimp and to shrimp that are subjected to stress.

(4) Fishes, particularly sheepshead minnows, are extremely sensitive to Aroclor® 1254. Concentrations

of about 0.1 µg/l in water are lethal to fry and affect reproduction of this fish. About 7 µg/g in eggs of the fish is lethal to fry hatching in water free of this chemical.

(5) The acute or short-term toxicity of Aroclor® 1016 to estuarine organisms is similar to other Aroclors but it appears less toxic to fishes in long exposures than does Aroclor® 1254.

BIOASSAYS OF 96 HOURS OR LESS DURATION

Aroclor 1260: Acute bioassays lasting as long as 96 hours show that Aroclor® 1260 is toxic to a wide variety of animals. Growth of the ciliate, Tetrahymena pyriformis, was reduced after 96 hours in growth media containing 1,000 µg/l of Aroclor® 1260 (Exhibit 11). Pinfish were apparently unaffected and 10% of the pink shrimp died in 100 µg/l of the PCB (Exhibit 12) during a 48-hour test. Oysters, however, were sensitive to Aroclor® 1260 with growth diminished by 44% in 10 µg/l and 52% in 100 µg/l in a 96-hour test.

Aroclor 1254: Acute bioassays indicate that Aroclor® 1254 may be more toxic to some estuarine organisms than Aroclor® 1260. Growth rates of the ciliate protozoan, T. pyriformis, were significantly reduced by 96 hours exposure to 1 µg/l (part per billion) of Aroclor® 1254 in 10 ml of growth medium (Exhibit 13). A concentration of 100 µg/l of Aroclor® 1254 in 48-hour flowing water bioassays appeared to have no effect on

juvenile pinfish, Lagodon rhomboides, but killed 100% of the juvenile pink shrimp, Penaeus duorarum. Shell growth of oysters, Crassostrea virginica, exposed for 96 hours was completely inhibited by 100 µg/ℓ and decreased 41% over that of unexposed controls by 10 µg/ℓ of Aroclor® 1254 (Exhibit 1[illegible]. All four species accumulated Aroclor® 1254 with quantities in oysters, pinfish, and pink shrimp exposed to 10 µg/ℓ in excess of 1 µg/g (part per million). The Aroclor®1254 is toxic to the amphipod, Gammarus oceanicus, (Exhibit 14). The amphipod also accumulates Aroclor®1254 from water (Exhibit 15).

Aroclor 1248: The toxicity of Aroclor®1248 to T. pyriformis (Exhibit[illegible]) is about 1,000 times less than that of Aroclor®1254. Its toxicity to shrimp, oysters, and pinfish was similar to that of Aroclor®1254 (Exhibit 12).

Aroclor 1242: Bioassays lasting 96 hours demonstrate that this PCB is toxic to oysters and shrimps at concentrations of 100 µg/ℓ or less (Exhibit 16). This PCB, like the others tested, had no apparent effect on pinfish. Aroclor®1242 inhibits the growth of certain strains of estuarine bacteria (Exhibit 17).

Aroclor 1221: In static bioassays lasting 96 hours, 80% of the killifish, Fundulus heteroclitus, exposed to 25,000 µg/ℓ of Aroclor®1221 died (Exhibit 18). All control and 7,500 µg/ℓ exposed fish survived the 96-hour experiment. Lethal concentrations of Aroclor®1221 did decrease the ability of this killifish to osmoregulate. The osmolarity and Na concentration of the serum was consistently increased in fish exposed

to lethal levels of Aroclor®1221.

Aroclor 1016: Bioassays lasting 96 hours show that Aroclor®1016, like other PCBs, is acutely toxic to many estuarine organisms at concentrations of 100 µg/l or less (Exhibit 19). About 10 µg/l was estimated to be lethal to 50% of the oysters, brown shrimp (Penaeus aztecus) and grass shrimp (Palaemonetes pugio) in flowing water bioassays. Only 18% of the pinfish exposed to 100 µg/l died in the 96-hour exposure. All four of these species accumulated the PCB with the quantities accumulated being related to concentrations in the test water and apparently not to species of animal. Aroclor®1016 inhibits the growth of certain strains of estuarine bacteria (Exhibit 17).

Data on the acute toxicity PCBs tested is summarized in Exhibit 20.

BIOASSAYS EXCEEDING 96 HOURS DURATION

Aroclor 1254:

Mollusca - Young oysters were continuously exposed to Aroclor®1254 for 24 or 30 weeks in flowing, unfiltered sea water (Exhibit 21). Growth rates (height and in water weights) were significantly reduced in oysters exposed for 24 weeks to 5 µg/l (3.9 µg/l measured), but apparently were not affected by 30 weeks exposure to 1 µg/l (0.64 µg/l measured). Survival of oysters was not affected by these concentrations of the chemical. Oysters accumulated as much as 101,000 X the concentration in the test water. Concentration was <0.3 µg/g in 5 µg/l exposed oysters after 28 weeks of depuration in PCB-free

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water and <0.2 µg/g in 1 µg/l exposed oysters after 12 weeks of depuration. Oysters exposed to 5 µg/l for 24 weeks displayed general tissue alterations in the vesicular connective tissue around the digestive diverticula of the hepatopancreas.

In another experiment, oysters were exposed continuously, with no significant mortality, for 56 weeks to 0.01 µg/l of Aroclor® 1254 and showed a concentration factor of 165,000 [redacted]. Maximum concentration of 1.65 µg/g occurred in oysters exposed for eight weeks. Patterns of accumulation and loss were seasonal. Residues decreased 45% to 81% in early July and late October, apparently as a result of spawning, and increased following these periods. This data indicates that life-history of oysters must be known when evaluating residue data from monitoring programs.

We have not received Exhibit 22 Strike it

Crustacea - blue crabs. Juvenile blue crabs, Callinectes sapidus, exposed for 20 days to [redacted] (3.5 to 4.2 µg/l measured) of Aroclor® 1254 were apparently not affected (Exhibit 1). Average concentration in five crabs was 23 µg/g. The Aroclor® 1254 was slow to flush from crabs. After one week in PCB-free water concentrations in a second group of five crabs averaged 22 µg/g and after 4 weeks six crabs contained 11 µg/g.

Fiddler crabs. Fiddler crabs, Uca pugilator, were exposed to Aroclor® 1254 contaminated sediment from Escambia Bay, Florida to determine if the crabs would accumulate the Aroclor®

1254 from the sediment (Exhibit 3). Fiddler crabs accumulated Aroclor in quantities directly related to the amounts in sediments. The chemical was accumulated by ingestion or by its leaching into the water.

Pink shrimp. Pink shrimp were exposed for 20 days to 5µg/l of Aroclor®1254 (3.5 to 4.2µg/l measured) in flowing unfiltered water (Exhibit 1). Seventy-two percent (18 of 25) died during the exposure, no controls died. The first shrimp died on the tenth day of exposure, and a few died each day during the next 10 days. Several died during molting. Symptoms of insecticide poisoning were not exhibited by dying shrimp.

A series of additional bioassays using pink shrimp showed that Aroclor®1254 is differentially toxic to juvenile and adult shrimp (Exhibit 5). In flowing water bioassays, a measured concentration of Aroclor of 0.94µg/l killed 51% of the juvenile shrimp (2.5 to 3.8 cm) within 15 days. Exposure to 3.5µg/l (measured concentration) for 35 days resulted in a mortality of 50% of a group of (9.5 to 12.5 cm) adult shrimp. Dying shrimp exhibited no apparent outward symptoms of poisoning. Mortality was delayed, they died at the rate of one or two per day and seemed most susceptible during molting. Shrimp obtained the PCB from water and food and concentrated it in the greatest amounts in the hepatopancreas. Shrimp placed in PCB-free water lost about 40% of their total body burden of PCB within 5 weeks. If Aroclor®1254 caused shrimp mortality in the environment, it would be unlikely that it could be observed because:

(1) shrimp would die over an extended period of time, (2) dying shrimp would decompose rapidly or be eaten by predators, and (3) dead shrimp would not be visible, because unlike fish, they do not float on the surface of the water.

Pathological examinations of exposed pink shrimp demonstrated that, even if there were no outward symptoms of poisoning, there were alterations at the cellular and sub-cellular level. The hepatopancreas of shrimp exposed to Aroclor® 1254 did exhibit dramatic tissue changes which includes the presence of pyramidal crystalloids in the nuclei of epithelial cells (Exhibit 24). Along with the presence of this crystalloid were rod-shaped, free (non-occluded) and occluded virons which appear similar to the Baculovirus group of invertebrate viruses (Exhibit 24). Aroclor® 1254 may facilitate the transmission or enhance the expression of latent viral infections.

Brown shrimp - adult brown shrimp (Penaeus aztecus) were as sensitive to Aroclor® 1254 as adult pink shrimp (Exhibit 24). Three µg/l was lethal to both shrimps within 30 days. Brown shrimp exposed to a sublethal quantity of the Aroclor® 1254 for seven days died if the salinity of the test water decreased gradually in 8 hours from 30‰ to 10‰ and 7‰. Salinity changes such as this occur during tidal cycles. Osmoregulation seemed impaired because concentrations of most major ions in sera of PCB-exposed shrimp became significantly less as the

ambient salinity decreased. This was reflected as a decrease in the sum of the major ions (e.g. Na, Ca, Mg, K, Cu, and Cl) and particularly in the sodium, chloride, and calcium ions.

Grass shrimp - grass shrimp, Palaemonetes pugio, were exposed to measured concentrations of Aroclor® 1254 ranging from 0.17 to 12.5 µg/l for 7 or 16 days (Exhibit 6). A concentration of 4.0 µg/l produced significant mortality but mortality of 1.3 µg/l exposed shrimp was not significantly different from that of control shrimp. Shrimp exposed for seven days to from 0.17 to 9.1 µg/l contained Aroclor in amounts ranging from 3,200 to 11,000 X the measured concentration in the test water. Grass shrimp accumulated the Aroclor® 1254 in ever increasing amounts in exposures lasting as long as 35 days.

Amphipod - the amphipod, Gammarus oceanicus, was exposed to nominal concentrations of Aroclor® 1254 of from 1 to 10,000 µg/l for up to 30 days (Exhibit 25). Aroclor® 1254 was lethal at concentrations as low as 10 µg/l. Some Gammarus had severely necrosed branchiae. Branchial necrosis was found in some animals exposed to 1 µg/l of the Aroclor® 1254.

Fishes - Spot. In three separate flow-through bioassays, 5 µg/l of Aroclor® 1254 (measured concentration with ± 20% of nominal) was lethal to juvenile spot (Leiostomus xanthurus) exposed in flowing water for 20, 26 or 45 days (Exhibit 26). Spot exposed to 1 µg/l showed no apparent effects. Affected

spot usually ceased feeding, became emaciated, and developed ragged fins and lesions on the body. In some instances, fish continued to die even though they were place in PCB-free water. Aroclor® 1254 was accumulated by exposed spot with amounts related to duration and extent of exposure and not apparently to mortality. Spot exposed to 1 µg/ℓ of the Aroclor® 1254 for 56 days rapidly stored this chemical, maximum levels were attained in 14 to 28 days. Maximum amount stored in whole fish was 37,000 times that in the test water. After 84 days in PCB-free water, 39% of the Aroclor® 1254 remained in the fish.

Pinfish - in two separate bioassays lasting 14 or 35 days, 5 µg/ℓ of Aroclor® 1254 was lethal to juvenile pinfish (Exhibit 26). Affected fish usually developed fungus-like lesions on the body, especially around the mouth. Concentration of the Aroclor® 1254 in whole pinfish exposed for 35 days to 5 µg/ℓ was 109 µg/g (21,800 X the amount in the test water).

Sheepshead minnows. Sheepshead minnows are the most sensitive estuarine organism to Aroclor® 1254 that we have tested (Exhibit 27). Intermittent flow bioassays lasting three weeks were conducted with embryo, fry, juvenile and adult fish. Newly hatched fry were the most sensitive life-stage tested. Significant fry mortality occurred in 0.3 µg/ℓ (0.16 µg/ℓ measured) and 0.1 µg/ℓ (0.06 µg/ℓ measured) had no apparent effect. Fry did not die immediately upon hatching but were killed over most of the two weeks following hatching. Embryos

and juveniles of this fish were affected by 10 µg/l (3.5 µg/l measured) but this concentration was not lethal to adults. Many fry, juvenile and adult fish developed fin-rot.

Aroclor® 1254 in eggs from adult sheepshead minnows exposed in bioassays to as little as 0.3 µg/l (0.14 µg/l measured) of the Aroclor® 1254 can decrease survival of embryos and fry that are maintained in water free of this chemical (Exhibit 28). Fertilization success was unimpaired by concentrations in eggs as high as 201 µg/g but survival of embryos and fry was significantly reduced. Usually, fry from eggs containing 7.0 µg/g or more began dying 24-48 hours after hatching. Some preliminary research indicates that PCBs in eggs may decrease fertility and survival in early stages of embryonic development in Atlantic salmon, Salmo salar (Exhibit 29).

Communities of Estuarine Organisms:

Aroclor® 1254 affected the composition of communities of estuarine animals that developed from planktonic larvae in salt water that flowed through 10 control aquaria and 10 aquaria contaminated with 0.1, 1, or 10 µg/l of the Aroclor® 1254 (Exhibit 30). Communities that developed in control aquaria and aquaria that received 0.1 µg/l of PCB in water for four months were dominated (>75%) by arthropods, primarily the amphipod Corophium volutator. In aquaria receiving 1 and 10 µg/l, the number of arthropods decreased and the number of chordates, primarily the tunicate Molgula manhattensis, increased; over 75% of the animals in 10 µg/l aquaria were tunicates. Numbers of phyla, species, and individuals (particularly

amphipods, bryozoans, crabs, and mollusks) were decreased, but there was no apparent effect on the abundance of annelids, brachipods, coelenterates, echinoderms or nemerteans. The Shannon-Weaver index of species diversity was not altered by Aroclor®1254.

Aroclor 1016 - Pinfish. The chronic toxicity and uptake of Aroclor®1016 by pinfish was tested in three separate flow-through bioassays lasting 42 days (Exhibit 19). Mortality of pinfish exposed to 32 µg/l (13 µg/l measured in one experiment and 21 µg/l in the second) or 100 µg/l (59 µg/l measured) was significantly greater than mortalities of unexposed pinfish. Mortality of 10 µg/l (about 7 µg/l measured) exposed fish was negligible. Color of most dying fish darkened, they stopped feeding and they swam erratically with their head inclined downward. Difficulty in swimming progressed until the fish swam upside down just prior to death. Pathological examination of 32 µg/l exposed fish revealed several liver and pancreatic alterations that distinguished them from control fish. Concentrations of Aroclor®1016 stored by the pinfish exposed for 42 days ranged from 11,000 to 24,000 times the nominal concentration in the test water. Concentrations in pinfish exposed to 1 µg/l for 56 days increased for three or four weeks and then stablized. When placed in PCB-free water for 56 days, concentrations (µg/g) in these fish decreased by 51%.

Sheepshead minnows: The chronic toxicity of Aroclor® 1016 to fry, juvenile and adult sheepshead minnows was investigated in intermittent flow bioassays lasting 28 days [redacted] Fry, juvenile, and adult fish were apparently not affected by nominal concentrations of 0.1, 0.32, 1.0, 3.2 or 10 µg/l but died in 32 and 100 µg/l of this chemical. Chemical analyses of the test water showed about 4 µg/l in the 10 µg/l aquaria, 15 µg/l in 32 µg/l aquaria and 42 µg/l in the aquaria contaminated with 100 µg/l of Aroclor® 1016. Responses to poisoning included darkened body coloration, uncoordinated swimming, cessation of feeding, lesions on the body of many juveniles and adults, and death. Sheepshead minnows accumulated the PCB in proportion to its concentration in the test water. Fry contained [redacted] the nominal concentration in the test water, adult[redacted] and juveniles[redacted] Eggs from exposed adults were fertilized and placed in PCB-free water. As much as [redacted] of the PCB in these eggs apparently did not affect survival of embryos and fry for two weeks.

Information on flow-through bioassays lasting longer than 96 hours is summarized in Exhibit 32.