UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
BEFORE THE ADMINISTRATOR

In the Matter of:

PROPOSED TOXIC POLLUTANT EFFLUENT STANDARDS FOR POLYCHLORINATED BIPHENYLS (PCBs)	)	FWPCA (307) Docket No. 4

REPLY BRIEF OF THE ELECTRONIC INDUSTRIES ASSOCIATION'S AD HOC PCB COMMITTEE

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The many hundreds of pages of briefs filed with the Administrator by the Agency's counsel, the Environmental Defense Fund, the Natural Resources Defense Council and others reflect the mammoth size of the record in the proceedings conducted before Law Judge Finch during September, October and November 1976. Yet 90 percent of that record concerns types of PCB which either have never been used or have not been used for many years by the capacitor industry. The only data relevant to the capacitor industry are those relating to Aroclor 1016--and the evidence presented on the alleged toxicity of Aroclor 1016 is both minimal and inconclusive.

Save for its possible rhetorical impact, there is no scientific rationale for EPA counsel's continued references to "PCBs" without any serious attempt to analyze

the differences between commercial PCB materials. Likewise, the EDF and NRDC briefs are replete with statements damning "PCBs" with only minimal attention to the central issue whether it is appropriate to regulate the discharges of capacitor manufacturers based on evidence concerning the alleged toxic effects of such highly chlorinated compounds as Aroclors 1254 and 1260. As we established in our opening brief, and as we will discuss below, there is no reputable scientific evidence demonstrating that Aroclor 1016 poses any threat to the environment if discharged at levels below 100 parts per billion.

Before reviewing the scientific issues in this proceeding, the discharge regulations proposed by the Agency must be placed in proper perspective. EPA counsel together with the EDF and NRDC contend that the Administrator should ban completely the use of polychlorinated biphenyl in capacitors and transformers under the authority granted by Section 307(a) of the Federal Water Pollution Control Act, 33 U.S.C. §1251 et seq. These parties apparently propose that such a ban take effect in January 1978.

EPA counsel have justified their decision to abandon the originally proposed 1 ppb discharge limit due in part to the fact that the capacitor industry will abandon

use of Aroclor 1016 by the second or third quarter of 1978.*/ This forced abandonment of Aroclor 1016 arises from Monsanto's decision to end production of the material, coupled with the Toxic Substances Control Act of 1976 which imposes a ban on PCB after 1978 (subject only to the grant of exemptions by the EPA Administrator). Thus, EPA counsel argue that a PCB ban under the FWPCA "will have no greater economic impact than would the standards originally proposed by the Agency on July 23, 1976." (EPA Br., pp. 20-21)**/ For that reason EPA counsel propose an outright ban on PCBs some three to six months before the capacitor industry will be obliged to cease using Aroclor 1016.

The Agency has offered no compelling reason to justify a ban on Aroclor 1016 that would contravene the carefully scheduled phase-out of PCB use mandated by TSCA. There are, however, excellent reasons why such a ban should be avoided.

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*/ The other principal reason offered for EPA counsel's new recommendation of a PCB ban is the evidence that some PCBs may be carcinogenic. See EPA Br., pp. 8-11. There is absolutely no evidence linking either Aroclor 1016 or 1242 with cancer. See page 30 infra.

**/ References to the briefs of the parties submitted on December 22, 1976 will be cited by the name of the party, e.g., EDF Br., p. ___. The Agency's Proposed Findings of Fact which were submitted as a separate document will be cited EPA PFF No. ___, p. ___.

All known substitutes for Aroclor 1016 are highly flammable. (EIA Exh. B at 5 and Exhibit B thereto) A short circuit in a capacitor case could cause a catastrophic fire in the home or any of the countless places in this country where capacitors can be found. The safety hazards posed by a ban on Aroclor 1016 cannot be dismissed as a mere "economic impact." There are very serious questions whether these safety risks outweigh any conceivable harm Aroclor 1016 might pose to the environment. It is such matters which the Administrator must evaluate under TSCA in determining whether an exemption for Aroclor 1016 (or some other similar PCB compound, such as the Prodelac dichloralbiphenyl material)*/ should be granted. Fluorescent lights, home air conditioners, high intensity discharge lighting, high voltage electrical transmission lines and many other appliances contain capacitors which will pose fire hazards once Aroclor 1016 is banned. The only known method of dealing with this fire hazard is to develop a protective device which will break the electrical circuit

*/ See EIA Exh. B at 7. The Agency has notified Prodelac that its material could not be recommended for use due to its inclusion of PCBs. The French Government, on the other hand, has exempted the material from that country's ban on PCBs. (Id.)

the instant the capacitor case ruptures, and thus prevent serious fire from developing. Unfortunately no protective device to-date has been shown to work reliably.*/ Further, all substitute materials will increase the costs of capacitors by a minimum of 20 to 30 percent.**/

In these circumstances, only a dire necessity could justify the more immediate ban on Aroclor 1016 proposed by the Agency. No emergency of this sort exists. The record provides no basis for the apparent view of Agency counsel that termination of Aroclor 1016 use in capacitors some three to six months sooner than the presently scheduled phase-out of Aroclor use by the industry will have any meaningful impact on the environment. Weighed against

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*/ The Agency misleadingly suggests that "protective devices" have been in use since 1968 and have functioned without failure. See EPA PFF XXIV.B.14, p. 173. These "protective devices" were devised solely to preclude leakage of oil from fluorescent lights and thus prevent stains (Tr. 2189-90); the record shows that on-going tests with protective devices designed to prevent fires, not just leakages, have established that they do not reliably protect against the fire hazard which will be created by the elimination of Aroclor 1016. See Tr. 2166-67; EIA Exh. B at 5-6.

**/ While Agency counsel devotes considerable attention to the material recently put on the market by the Dow Chemical Company (EPA PFF XXIV.B.7-9, p. 166) the proposed findings failed to note that the Dow material is 60 percent more costly than Aroclor 1016, and is flammable. While the cost of the Dow material precludes its use in most capacitors, it is worthwhile to note that the record established that more recent life-testing of the fluid showed an unacceptably short life in actual use. EIA Exh. B at 5; Tr. 2172-73.

the minimal Aroclor 1016 evidence in the record and the unavoidable hazards a PCB ban will create, as well as the conflict that would be created by a FWPCA ban in the face of the TSCA ban mandated by Congress, EPA counsel's zero discharge proposal should be rejected.

ARGUMENT

I

THE TOXIC SUBSTANCES CONTROL ACT PRECLUDES A BAN ON AROCLOR 1016 UNDER THE FEDERAL WATER POLLUTION CONTROL ACT

In our opening brief we argued that a 1 ppb discharge limitation for all PCBs was tantamount to a ban on the use of Aroclor 1016 in capacitors and that such a ban was inconsistent with the carefully designed phase-out of PCBs mandated by Congress in the recently passed Toxic Substances Control Act. TSCA provides for a 2-1/2 year schedule of termination of PCB use. It further provides that the Administrator may grant exemptions for materials such as Aroclor 1016 for which no suitable substitutes are available. The legislative history cited at length in our opening brief established that Congress considered the possibility of EPA action under the FWPCA, but determined

that the regulation of PCBs should be governed by the Toxic Substances Control Act. Indeed, the Act was signed into law while hearings in this proceeding were in progress. (See EIA Br., pp. 15-22) Surprisingly, the briefs submitted by the other parties are silent as to the relationship between TSCA and the FWPCA. Yet the Agency's new recommendation of a zero discharge level for Aroclor 1016 makes the conflict between the two statutes all the more obvious, and the Agency's failure to discuss the TSCA ban all the more striking.

In passing the PCB provisions contained in the Toxic Substances Control Act, the sponsor of the proposal in the House emphasized that such regulation "does not call . . . for immediate prohibition but, rather, a gradual phase out to assure action within a reasonable period of time." 122 Cong. Rec. H8829 (daily ed., August 23, 1976) (remarks of Rep. Dingell) In supporting the Dingell-Gude amendment, which added a PCB provision to the original House version of TSCA, Congressman Murphy explained that "the substances are necessary at the present time for use in capacitors and transformers," and accordingly the Dingell-Gude provision for a 2-1/2 phase-out of PCB use "seems reasonable. It gives at least 2 years to the in-

dustry to come up with substances other than PCB's. I think 2-1/2 years is certainly a reasonable compromise and warning to the industry." 122 Cong. Rec. H8833.

Throughout the legislative debate on the PCB proposal, members of both the House and Senate emphasized that the PCB regulation contained in TSCA provided "mechanisms for authorizing uses of PCB's beyond these time limits should that be appropriate so that we do not create worse problems than those we solve." 122 Cong. Rec. S4410 (daily ed., March 26, 1976) (remarks of Senator Tunney) Senator Nelson, the sponsor of the PCB proposal in the Senate, emphasized in debate on the floor that the Administrator was required to consider granting an exemption for certain types of PCBs, taking into account "the availability of substitutes for PCBs, presumably having less risk." 122 Cong. Rec. S4408 (daily ed., March 26, 1976)

Thus whatever the merits of the arguments of EDF and NRDC that the Administrator may not consider the availability of substitutes and the economic impact of regulation under the Federal Water Pollution Control Act, */ TSCA requires the Administrator to consider these factors in

*/ While we agree with the Agency that economic factors are relevant in considering discharge regulations under the FWPCA, we need not engage in protracted discussion of this point. First, such economic analysis has already been performed. Second, none of the arguments which we have made are based on the economic impact of the proposed regulations. Rather the issues in dispute concern the legal and scientific merits of the proposed discharge limitations.

determining whether an exemption should be granted. The ultimate inconsistency in attempts to regulate PCBs under both statutes is plain. In such a case, the more recent statute must take precedence and preempts contrary regulation under the Water Pollution Control Act.*/ Araya v. McLelland, 525 F.2d 1194, 1196 (5th Cir. 1976); Internat'l Union of Electrical, Radio and Machine Workers v. NLRB, 289 F.2d 757, 761 (D.C. Cir. 1960).

II

AN END-OF-PIPE LIMITATION OF 100 PPB FOR AROCLOR 1016 WILL PROVIDE AN AMPLE MARGIN OF SAFETY TO THE ENVIRONMENT

Both the proposed discharge regulations and the total ban now recommended by Agency counsel fail to distinguish among the various PCB mixtures despite the overwhelming evidence that PCBs differ very substantially in their toxicity and persistence. The over two hundred pages of arguments submitted by EPA counsel fail to establish any scientific basis for regulating all PCBs as if they were a single chemical, either through a single 1 ppb discharge standard, or by a total ban.

*/ In our opening brief we argued that the establishment of uniform national discharge regulations violates the FWPCA's requirement of consideration of local biological conditions. (See EIA Opening Br., pp. 22-25) No party has presented any contrary arguments in the briefs submitted to date.

With only a single exception, the Agency presented no witnesses whose experiments with Aroclor 1016 demonstrated any toxic effects at suitably low dosages paralleling the concentration which would be produced by a 100 ppb discharge limitation.*/ Rather, the "evidence" presented by the Agency on Aroclor 1016 amounts to no more than an assertion by the Agency's principal witness, Dr. Ian Nisbet, that he believed there to be no significant differences between Aroclor 1016 and the other PCB compounds tested. (See EPA Exh. M at 9-10; Tr. 1526) Dr. Nisbet was the only witness to so testify. His conclusion was squarely refuted by Dr. Gerald Lauer and Dr. Wolfgang Mueller who reviewed the evidence pertaining to Aroclor 1016 as it related to aquatic and mammalian toxicity respectively. (See EIA Exh. D at 4-5, 12-15; Tr. 2398; EIA Exh. G at 14)

The Agency's brief and proposed findings seek to discredit the testimony of these witnesses, principally on the ground neither had performed experiments with Aroclor

*/ As to that one study purporting to show toxic effects of Aroclor 1016 at low dosages, Dr. Ringer's mink experiment concededly produced few, if any, statistically significant effects. See EIA Opening Br., pp. 40-41 and discussion pages 22-23 infra.

1016 to support conclusions based on their experience in related experiments, their professional backgrounds, and their extensive review of the available literature. By challenging Drs. Lauer and Mueller in this way, the EPA counsel employ a curious double standard. Ian Nisbet, on whose testimony the Agency relies, performed no experiments. Indeed, he has never performed any experiments involving aquatic or mammalian toxicological responses. As a physicist, Dr. Nisbet has no expert credentials as a chemist, biologist, or a scientist in any related field. His testimony was based solely on a review of the literature--yet the Agency suggests Dr. Lauer and Dr. Mueller should not be entitled to review the literature independently and offer their expert judgments. Gerald Lauer is an aquatic biologist; he has been involved in the evaluation of the effects of pollutants on aquatic life for 17 years, and has been intimately involved in testing the toxicity of pollutants. (Tr. 2361-62; Exhibit A to EIA Exh. D) Wolfgang Mueller for the past seven years has done original research on the ability of mammals to metabolize toxicants--and for the last three years has headed a primate research center studying the effects of poly-

chlorinated biphenyl isomers on mammals. (EIA Exh. B at 1-2; Exhibit A thereto; Tr. 2672-73) */

Save for gratuitous attacks on Lauer's and Mueller's credentials, the Agency offers little response to their conclusions that

(i) Aroclor 1016 varies so substantially in its chemistry that it will not produce the toxic effects observed for other more highly chlorinated PCB compounds (EIA Exh. D at 4-5, 12, 15; EIA Exh. G at 14; Tr. 2732);

(ii) Data pertaining to Aroclor 1242, 1248, 1254 and 1260 may not be extrapolated to Aroclor 1016 (EIA Exh. D at 12; EIA Exh. G at 15); and

(iii) There is no evidence that Aroclor 1016 will produce toxic effects to either aquatic or mammalian life at low dose levels. (EIA Exh. D at 10-11; EIA Exh. G at 10-11, 15; Tr. 2764; Tr. 2798-99)

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*/ One egregious misstatement in the Agency's proposed findings concerning Dr. Mueller demands response. Despite Dr. Mueller's repeated explanations in response to questions from EPA counsel that he is not a part of the Albany Medical College, See Tr. 2793-94, EPA counsel suggest that Dr. Mueller is to be faulted for his lack of familiarity with some of the experiments which might have occurred at that facility in 1973, asserting these experiments were performed at Dr. Mueller's primate laboratory. EPA PFF IX.4, pp. 39-40. This is a most unfair and inaccurate charge, and has no record basis.

A fair reading of the record in this proceeding demonstrates that no discharge limitation lower than 100 ppb is justified for Aroclor 1016. In establishing any discharge limit, four interrelated factors must be considered. (1) What effect do ambient discharges have on PCB concentrations in the water? (2) What is a safe level for aquatic organisms in the ambient waters? (3) What effects will these concentrations have on consumers of fish and other aquatic organisms exposed to PCBs? (4) What is an ample margin of safety?

As we demonstrated in our opening brief, the record conclusively establishes that a discharge limit of 100 ppb for Aroclor 1016 provides a fully "ample" margin of safety to the environment. Such a discharge limitation will produce ambient concentrations of Aroclor 1016 no greater than 100 ppt. At this level, Aroclor 1016 poses absolutely no threat to any aquatic organism. Even assuming the maximum bioaccumulation in aquatic life found in a laboratory experiment, accumulated Aroclor 1016 in fish tissue will amount to no more than 5 ppm, which is a level wholly safe for all fish consumers. In this brief we will not undertake to repeat the extensive discussion of these points contained in our opening brief. (See EIA Br., pp. 26-50) Rather, we will review the briefs of the other parties and show that none of their arguments detract from our conclusion that a 100 ppb discharge limitation for Aroclor 1016 is fully safe.

A. A Discharge Limitation for Aroclor 1016 of 100 ppb Will Produce An Ambient Concentration Not Lower Than 100 ppt

EPA counsel agree fully that dispersion and dilution will cause the ambient concentration of Aroclor 1016 to be 1000-fold smaller than the end-of-pipe concentration.*/ Indeed, the 1 ppb discharge limit originally proposed by the Agency was based on a "ratio of 1000-fold or more" (EPA Exh. G at 3) to establish the proposed ambient standard of 1 ppt. There is no evidence to suggest that dispersion and dilution will not cause a 1000-fold decrease in the concentration of Aroclor 1016 discharged into ambient waters.

While the EDF suggests that a 1000-fold dilution and dispersion factor is invalid, its claims have no support in the record. EDF introduced no evidence to challenge the testimony of Richard Callaway that dispersion and dilution factors of 1000-fold or more "appl[ied] generally to chlorinated organic substances . . .." (EPA Exh. G at 3) Despite EDF's claims that Dr. William Brungs testified that the persistence of PCBs affected their dispersion (EDF Br., p. 48), in fact, Dr. Brungs testified that PCBs would disperse no differently from other toxicants. (Tr. 639-40)

*/ EPA PFF XXI.3, p. 137.

B. Aroclor 1016 Has No Toxic Effects On Aquatic Organisms At Levels Below 100 ppt

No party has argued that Aroclor 1016 has any toxic effects on aquatic organisms except at levels greatly in excess of the EIA's proposed ambient criterion of 100 ppt. As we showed in our opening brief, no toxic effects on any aquatic organisms for any PCB mixture were found to occur below 100 ppt. Indeed, the mildest and most equivocal effects resulting from exposure to the most highly chlorinated PCB mixtures were found to occur only at levels some two orders of magnitude above the EPA's original proposed ambient criterion of 1 ppt.

The Agency's proposed findings on the adverse effects of PCBs on aquatic microorganisms, invertebrates and fish do not contradict the conclusion that Aroclor 1016 is entirely safe to aquatic organisms at levels of 100 ppt.*/ Thus, the only basis for the Agency's claims that Aroclor

*/ All studies referred to in the EPA's proposed findings indicated that Aroclor 1016 posed no risk whatever to aquatic life at levels of 100 ppt. The Agency's counsel misleadingly state that "PCBs inhibited the growth of a number of estuarine bacteria in laboratory cultures." EPA PFF VI.1, p. 27. In fact, in the EPA-referenced study, only 15 of the 85 culture studies showed any effects on growth from Aroclor 1016 at 100 ppb, and only 26 of the cultures reflected any sensitivity to Aroclor 1016 even at levels of 500 ppb. This study in no way suggested Aroclor 1016 would produce any harmful effects at levels of 100 ppt.

1016 should be banned is the alleged toxic effects of the material on fish consumers.*/

C. Aroclor 1016 Will Have No Toxic Effects on Mammals at Levels Below 5 ppm

In evaluating the possible adverse effects of Aroclor 1016 on mammals, it is necessary first to determine what concentrations of the material will accumulate in aquatic tissue consumed by animals and humans. The maximum bioaccumulation factor found for Aroclor 1016 is 50,000. Based on this factor, a 100 ppb discharge limit will produce concentrations of Aroclor 1016 in fish tissue no greater than 5 ppm, a level entirely safe for fish consumers.

1. Aroclor 1016 Will Bioaccumulate In Aquatic Organisms By A Factor No Greater Than 50,000

The EPA's proposed findings on bioaccumulation factors for Aroclor 1016 neglected to include all of the laboratory bioassays in which the bioaccumulation of Aroclor

*/ Neither the EDF nor NRDC contend that the possible effects of Aroclor 1016 on aquatic organism justify the proposed ban. They too rely on the alleged toxicity of Aroclor 1016 to fish consumers as a basis for regulation.

1016 has been measured.*/ The proposed findings of EPA did include the two long-term studies (one of approximately 32 days and the other of 42 days) that produced the highest reported values for Aroclor 1016 of approximately 50,000.**/

The Agency attempts to justify its claim that a single bioaccumulation factor should be used for all PCBs because Dr. Nisbet testified "the differences in retention between tetra- and higher CBs are small, no more than a factor of 2-3 in the circumstances of these experiments." (EPA PFF XV.12, p. 85)***/ But this is no

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*/ In particular, no mention is made of studies performed upon aquatic invertebrates which demonstrated bioaccumulation factors for Aroclor 1016 substantially below the 50,000 maximum figure noted in the two studies referred to above. (See, e.g., Exhibit E to EIA Exh. D)

**/ The Agency's proposed findings imply that the 50,000 figure produced by these two studies is not quite as high a figure as would have been produced had the studies been of longer duration. There is no record basis for the apparent claim that the 50,000 bioaccumulation value for Aroclor 1016 was faulty. Dr. Veith (the author of the experiment) expressly testified that "steady state" was reached by the end of his experiment and that at that time his data indicated a bioaccumulation factor for Aroclor 1016 of 50,000. (EPA Exh. B at 22; see also Tr. 2450-51)

***/ The Agency also weakly defends its use of the 274,000 bioaccumulation factor, which was based on a single experiment producing plainly anamolous results (see EIA Exh. D at 10), on the dubious assertion that "this figure . . . is as reliably based as any other in the record, and is derived from the only experiment involving long-term exposure of any fish to Aroclor 1242." EPA PFF XV.8, pp. 82-83. The histogram prepared by Dr. Lauer (Exhibit D to EIA Exh. D)vividly demonstrates that the 477,000 value on which this 274,000 figure was based bore no

[footnote continued]

explanation for the failure of the Agency to recognize that each PCB mixture has different bioaccumulation tendencies. Variations by factors of 2 and 3 are undisputably significant. Further, the claim that tetra-CBs are retained to a substantially lesser extent than higher CBs does not begin to account for the differences between Aroclor 1016 (78 percent of which is composed of mono-, di- and tri-CBs) and the other Aroclors. (See EIA Br., p. 9) The Agency introduced no evidence that these important differences in bioaccumulation may be disregarded in calculating the appropriate bioaccumulation factor for Aroclor 1016.

The EDF is similarly content to rely on isolated field data showing that the PCB level in some fish may exceed the PCB concentrations of the water from which they were taken by factors of one million or more (EDF Br., p. 53) But these data were rejected by both the Agency's Chief of its Criteria and Standards Division, Dr. Leonard Guarraia, and by Dr. Lauer. (See Tr. 1885; 2487)

[footnote continued]

relationship to any other study conducted with Aroclor 1242. The Agency introduced no evidence to rebut Dr. Lauer's expert judgment that the 477,000 value should be disregarded because it was not reliably based in good scientific practice.

The unconfirmed field studies relied on by EDF cannot substitute for controlled laboratory experiments. The fish taken in these field samples may have spent their lifetimes in waters containing much higher PCB concentrations than the specific waters from which they were taken. (Tr. 2488-89; EPA Exh. 2 at 355) Moreover, these isolated field studies reported only generalized "PCB" concentrations, with no identification as to specific PCB isomers. Thus, there was no showing that these studies had any relation to Aroclor 1016.

Both the Agency counsel and EDF misleadingly suggest without evidentiary basis that data on wild fish are relevant because wild fish may bioaccumulate differently and in higher concentrations. But, as Dr. Lauer explained, high PCB concentrations in fish tissue is likely the result of high PCB concentrations in the water body in which the fish spent its lifetime. In such a case, the PCB level in the tissue compared to the level of PCB actually in the water would produce a ratio no higher than the ratios produced in the laboratory. Of course, the ratio in nature cannot be calculated because it is impossible to determine where each fish has spent its lifetime. (Tr. 2489)

In the last analysis, counsel for both the EPA and EDF have failed to appreciate that this proceeding is designed to regulate future discharges of Aroclor 1016 and to determine a safe level for those discharges. The existence of a sizeable quantity of PCB already in the environment may not be used as a basis for rejecting conclusive evidence demonstrating that the discharges actually subject to regulation will add little to current PCB levels. The field studies suggesting that high levels of "PCBs" may occur on occasion in some fish is irrelevant to the task of regulating future discharges of Aroclor 1016.

2. Aroclor 1016 Has No Toxic Effects On Consumers of Aquatic Organisms At A Dietary Level Below 15 ppm

No party denies that with the one arguable exception of the Ringer study on mink, there is no evidence that Aroclor 1016 has toxic effects on consumers of aquatic organisms at low dietary levels.*/ Although EPA and EDF

*/ EPA counsel claim that the lowest dose level producing adverse effects with Aroclor 1016 is 8 ppm, citing the Iverson rat study (Reference 243 to the Criteria Document). EPA PFF XVIII.11(b)(2) page 113 [footnote] In fact, both the Criteria Document and its author, Dr. Nisbet, reviewed this study and stated that it involved a 15 ppm dosage. EPA Exh. M at 9; Criteria Document at 194. EPA counsel, in a footnote without transcript citation, have undertaken to rewrite Dr. Nisbet's testimony to change all references to 8 ppm. This extra-record testimony is grossly improper. We are unable to determine on what basis counsel now assert that 8 ppm is a proper estimate of the feeding levels involved in the study, other than the fact that Dr. Nisbet twice appeared to refer to 8 ppm rather than 15 ppm in the course of his testimony. (See

[footnote continued]

both assert that the data produced by other more highly chlorinated mixtures should be used to determine the toxicological effects of Aroclor 1016, neither party introduced any evidence to rebut the conclusions of Dr. Wolfgang F. Mueller that the chemical difference between Aroclor 1016 and the other PCB mixtures make such toxicological comparisons invalid.

In the absence of any direct evidence establishing the toxicity of Aroclor 1016, EPA and EDF counsel have been forced to rely exclusively upon the assertions of Dr. Ian Nisbet that all PCB mixtures should be regulated together. Because Dr. Nisbet believed that Aroclor 1242 is "generally similar" in molecular constitutution to Aroclor 1016, he asserted that generally their toxicities should be similar. This largely unsupported opinion is decisively refuted by the extensive and detailed testimony of Dr. Wolfgang Mueller.

[footnote continued]

Tr. 1640, 1744-45) At no time did Dr. Nisbet explain the reason why these references varied from the figures used in his prepared testimony, or from the figures contained in the Criteria Document. In the absence of evidence relating to the claim that 8 ppm is a proper figure, this effort to alter the record must be rejected.

Dr. Mueller concluded, based upon his extensive review of all of the data contained in the record on this particular issue:

> "Particularly in mammals, continued intake of small amounts of Aroclor 1016 will not result in elevated total body burdens or organ concentrations of PCB. Results from studies which have been carried out with Aroclors 1260, 1254, 1248 and 1242 cannot be directly applied for the toxicological evaluation of 1016, since all these products contain higher chlorinated isomers which are accumulated and stored in mammalian tissues." EIA Exh. G at 15. (emphasis supplied)

To buttress Dr. Nisbet's hypothesis that the toxicities of Aroclor 1016 and 1242 are comparable, the Agency's proposed findings of fact refer to three studies in which toxic effects arguably comparable to those found for Aroclor 1242 are asserted to have been produced in mammals by Aroclor 1016. (See EPA PFF XVIII.11, pp. 112-116) In fact, these studies provide no support whatever for Nisbet's theory that the two Aroclors are so comparable that they should be regulated together.

1. The first study discussed in the proposed findings is the Ringer study on reproduction in mink. We discussed the fact that this study produced no statistically significant adverse effects for Aroclor 1016 at low dose levels in our opening brief. (See EIA Br., pp. 45-47) The only results which were shown to have any statistical significance were at 25 ppm dosages. Even these results indicated no adverse effects from exposure to Aroclor 1016.

The Ringer study offers no support for claims that Aroclor 1016 is toxic at levels below 15 ppm, much less at 5 ppm.

2. The Iverson rat study is also cited by the Agency in the proposed findings. (Reference 243 to the Criteria Document) As we discussed in our opening brief, however, this study showed only the most minimal effects on rats when exposed to Aroclor 1016 at 15 ppm, which is the lowest dose level which produced any adverse effects in any animal tested with Aroclor 1016. Further, the Iverson study reported very substantial differences between the effects of Aroclor 1016 and 1242 on female rats (with somewhat less significant differences between the effects of the two mixtures on male rats). Thus the study also contradicts the claim that the toxicities of the two compounds are generally similar.

3. Finally, the Agency refers to an Industrial Bio-Test Laboratory study (Reference 209 to the Criteria Document) which purportedly showed that Aroclor 1016 has adverse effects because it produced differences in gonad weights in rats. The Agency's proposed findings seize upon this report as establishing that Aroclor 1016 is harmful, but the findings fail to note (i) that the authors of the report drew no conclusions whatever from the differences in

gonad weights reported, (ii) there was no evidence that the weight differences had any effect on reproduction, and (iii) the results were obtained only after feeding the rats high dosages of Aroclor 1016 (100 ppm). Assuming this study offers any evidence that Aroclor 1016 may be harmful, at most it suggests that possible adverse effects might occur at levels far in excess of the concentrations which would be produced by the 100 ppb discharge limitation proposed by the EIA.

Strikingly, EPA counsel are obliged to concede that two other experiments demonstrated substantial differences between Aroclor 1016 and 1242. These two experiments showed that Aroclor 1016 had none of the effects found for Aroclor 1242 at similar dose levels. (See EPA PFF XVIII.11(c)(1)-(2), p. 116)

In sum, the only three studies which are cited to support Dr. Nisbet's assertion that Aroclor 1016 will produce toxic effects analogous to those of the more highly chlorinated Aroclor mixtures are of at most dubious significance--and are contradicted by two studies which showed

important differences between Aroclor 1016 and other PCB compounds.*/

Agency counsel in their proposed findings also refer to increased levels of enzyme activity as a possible harmful effect of Aroclor 1016. (EPA PFF XVIII.11(a)(5), p. 114) In fact, all scientific witnesses who testified expressly declined to draw similar conclusions.**/ As

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*/ EPA counsel also rely on a statement by Dr. Nisbet that he believed Aroclor 1016 would cause hepatic effects at 0.5 ppm in the diet. EPA PFF XVIII.11(b)(7), p. 114-15. There is absolutely no basis for Dr. Nisbet's assertion in this regard. In fact, EPA's primary support for low level hepatic effects, the Iverson Study, specifically concluded that no hepatic effects occurred even at significantly higher levels. (EPA Exh. 2, Ref. 243, at p. 457)

**/ Even Dr. Harry Gelboin, the Agency's principal witness on the subject of MFO reactions, while expressing the view that any degree of enzyme induction represents "a violation of the integrity of the human organism," refused to claim concrete ill-effects. See EPA Exh. O at pp. 5-6. As Dr. Mueller pointed out, Gelboin deliberately chose only to describe the possible ill-effects of enzyme reduction. At no time did Gelboin assert that any persuasive evidence existed of harm resulting from such activity. See EIA Exh. G at pp. 8-9.

Dr. Mueller explained, the mild enzyme activity attributed to Aroclor 1016 is just as likely to be beneficial to mammalian organisms, as it is to be harmful. (EIA Exh. G at 8-9; Tr. 2740-41)

> "Although the enzyme inducing effects of low chlorinated biphenyls are far less marked than those demonstrated for higher chlorinated isomers, a certain (although small) effect of Aroclor 1016 on mixed function oxidases does exist. It cannot be said with assurance, however, that some small degree of induction of mixed function oxidases should be regarded as undesirable or injurious." EIA Exh. G at 16.

EPA also has argued that Aroclor 1016 might be toxic because it can be readily metabolized. Based on fragmentary and inclusive test results EPA counsel hypothesize that PCBs might be metabolized via an intermediate toxic substance known as arene oxides into a non-toxic metabolite known as dihydrodiol. (EPA PFF XVIII.15-17, pp. 117-120)

EPA bases its hypothesis on two studies, one by Allen with nursing infant rhesus monkeys and the other by Gardner with rabbits. Both of these studies dealt with exposures to a single tetrachlorobiphenyl isomer (2, 5, 2', 5') at levels much higher (100 times in the case of the monkeys and 2000 times in the case of the rabbits) than the 5 ppm maximum exposure that would result from the standards proposed by the EIA. Thus, the two studies provide no support for claims that a 100 ppb discharge limit

is unsafe. Further, the test results appear questionable in light of Dr. Mueller's attempt to repeat Allen's experiment on infant rhesus monkeys (five to seven months old) using 5 ppm of a trichlorobiphenyl isomer (2, 5, 4'). Dr. Mueller produced no evidence of the formation of dihydrodiol and thus no evidence of an arene oxide metabolic pathway. Dr. Mueller concluded that there was no evidence of an arene oxide pathway "under the more relevant conditions of daily intake of small amounts of PCB with the mother's milk." (EIA Exh. G at 12) Dr. Mueller also concluded that studies on the metabolism of infant rhesus monkeys "should not be generalized for the non-human primates." (Ibid.)*/

D. A Discharge Limitation of 100 ppb for Aroclor 1016 Would Provide An Ample Margin of Safety to the Environment

The suggested ban on Aroclor 1016 is grounded on the possible adverse effects of the material on mammalian

*/ There is clear evidence, for example, that rats do not use an arene oxide metabolic pathway. (EPA Exh. Q at 6 and Exhibit 8 thereto)

consumers of fish.*/ All parties have acknowledged that there is no evidence in the record that Aroclor 1016 poses any threat to aquatic life at levels of 100 ppt. While both EPA and EDF counsel have argued that evidence exists that Aroclor 1016 is toxic to fish consumers if discharged at levels of 100 ppb or below, this "evidence" is inconclusive. The assertion made by Dr. Nisbet concerning a possible analogy between Aroclor 1016 and other PCB compounds was properly rejected by Drs. Lauer and Mueller as unfounded; the three studies purporting to show toxic effects for Aroclor 1016 are either inconclusive, or fail to support any findings of toxicity below 5 ppm; and the hypothesis put forward by EPA counsel that Aroclor 1016 may have toxic effects as it is metabolized has no solid foundation.

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*/ There is no evidence as to non-mammalian fish consumers at levels below 5 ppm for Aroclor 1016. No party has argued that the material should be banned because of its possible toxic effects on non-mammalian organisms.

In apparent recognition that no evidence demonstrates that Aroclor 1016 is toxic, EPA and EDF counsel have engaged in rhetorical assertions that Aroclor 1016 may be harmful to humans. These claims concern the issue of PCB found to be contained in mother's milk and the possibility that some PCB compounds might be carcinogenic. Both are obviously matters of legitimate concern. But the fact that past levels of PCB in the environment may have contaminated mother's milk, as is seemingly suggested in EDF Exh. 3,*/ provides no basis for regulation of Aroclor 1016. There is no evidence Aroclor 1016 was contained in the milk.**/ There is no evidence that Aroclor 1016 at 5 ppm levels in fish would pose the slightest threat to any mammalian consumer--animal or human. All the evidence demonstrates that the metabolism and excretion of Aroclor 1016 by humans renders it entirely safe.

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*/ It is difficult to draw conclusions from this fragmentary document. It was introduced without any explanation of its background or methodology, and its author was not presented for cross-examination. At most it purports to show levels of PCB found in mother's milk, with no data as to the type of PCB isomer involved, or the concentrations of PCB to which the mothers were exposed. Tr. 1152-56.

**/ Given the fact Aroclor 1016 is rapidly metabolized by humans (EIA Exh. G at 3-4), there is no reason to expect that it would be passed from mothers to nursing infants as would the higher chlorinated mixtures which would concentrate in lipid tissue.

The second issue of frightening dimension is that PCBs might be carcinogenic.*/ We cannot fault EPA counsel for the concern they have expressed that a PCB compound such as Aroclor 1260 might be carcinogenic. But it is absolutely essential to distinguish between those Aroclor mixtures for which there is any evidence of carcinogenicity,**/ and those for which no carcinogenicity has been reported. There is absolutely no evidence whatever linking Aroclors 1016 or 1242 with cancer--either in humans or in test animals. No study introduced into

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*/ The only evidence that a PCB caused hepatic carcinomas in test animals was an Aroclor 1260 study conducted by Dr. Renate Kimbrough. She expressly refused to generalize from this experiment and made no claim that her work had any bearing on the other PCB mixtures. (Tr. 1309) In studies conducted by Dr. Kimbrough involving Aroclors 1016 and 1242 at 100 ppm dosages, no carcinogenic or pre-carcinogenic effects of any sort were found. See Reference 208 to the Criteria Document at pages 306-307. See also Tr. 1280-94.

**/ EDF also asserts that there is evidence that Aroclor 1254 may produce cancer in humans. But EDF was obliged to concede that the Mobil Oil Paulsboro study purportedly establishing this possibility offered no basis for any conclusions. It was a partial study conducted with fewer than 100 sets of medical records and with no data on the degree of exposure to other chemicals. Mobil refused to permit the epidemiologist conducting the study to have access to other medical records, or to continue her work. See Tr. 2814-15, 2819, 2823-24, 2838. See also Exhibit 3 to EDF Exh. A at 13. There were many other defects in the study which were brought out under cross-examination, and which are outlined by the EDF itself. See EDF Br., p. 37. It is enough that the witness EDF called to sponsor the study stated, without qualification, that in his opinion the Mobil Oil Study offered no basis for drawing any conclusion. Tr. 2822.

the record reflects any carcinogenic, co-carcinogenic, or pre-carcinogenic effects as a result of exposure to either Aroclor 1016 or Aroclor 1242. (See EIA Exh. G at 4-6) (Mueller]) This fact was implicitly conceded by both the EPA and the EDF. (EPA PFF XI.8, p. 64; EDF Br., p. 33)*/ The possibility that Aroclor 1260 might have links to cancer has no relevance whatever to regulation of Aroclor 1016.

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*/ In its proposed findings, EPA counsel state that the Industrial Bio-Test Laboratories experiment on Charles River strain rats "reported results [that] constitute prima facie evidence of the carcinogenic effects of Aroclors 1242, 1254 and 1260 . . . .." (PFF XI.15, p. 66) This statement is entirely unfounded. To check its original conclusion that the study produced no evidence of carcinogenicity, Industrial Bio-Test had its slides reviewed by three independent pathologists. All concluded that there was "no evidence of hepatocellular carcinogenicity" at any dose level (1 ppm, 10 ppm, 100 ppm) for any of the three Aroclors tested (Aroclor 1242, 1254, 1260). (EPA Exh. 9 at 35) One of these three pathologists, who EPA claims produced evidence of carcinogenicity, specifically stated that the tumors he identified as 'hepatomas' and 'chlangiohepatomas' were "benign tumors rather than malignant tumors (carcinomas)." (Reference 416 to the Criteria Document, p. 3; EPA Exh. 2 at D-12)

III

NO DISCHARGE LIMITATION FOR AROCLOR 1016 BELOW 20 PPB CAN BE MEASURED ACCURATELY AND RELIABLY

If the Administrator declines to set a 100 ppb discharge limitation for Aroclor 1016, the question remains what discharge limitation should be established from a purely administrative perspective. The answer to this question is that no standard lower than 20 ppb can be administered fairly. At lower levels it is impossible to produce accurate and reproducible test results.

There is no dispute that a discharge limitation set below the level at which accurate measurements can be made exposes all companies subject to the regulation to arbitrary and erratic enforcement. Both EPA counsel and EDF have attempted to establish that reliable measurements are possible at levels of 1 ppb and below, yet the only basis for this assertion is the testimony of witnesses who have had no experience testing industrial effluents for PCB levels, and whose test results (for the most part using distilled water samples) were never subject to comparison testing to determine their relative accuracy. See EPA PFF XX.7-9, pp. 128-130; EDF Br., pp. 13-17.

The witnesses on whom EPA counsel and EDF rely asserted that reputable testing laboratories should

be able to measure for PCB levels in industrial effluent accurately to 1 ppb. But no witness attempted to rebut the evidence that each time reputable testing laboratories have attempted to measure PCB levels to 1 ppb, using identical samples of effluent, they have been unable to agree as to their results.

The results of three such comparison testing programs were introduced into evidence. All demonstrate the unreliability of test reports below 20 ppb. The first was a round-robin conducted by the American Society for Testing and Materials with the cooperation of Monsanto Chemical. The ASTM/Monsanto round-robin was conducted to determine the ability of commercial testing laboratories to establish PCB levels in distilled water and river water (not industrial effluent itself). The results demonstrated variations between 1 and 23 ppb. (EIA Exh. F at 5 and Exh. B thereto) These results did not include the anamolous test results of two laboratories which had erred in their test procedures.*/

*/ James Laubscher, who heads the Woodson-Tenent laboratory, testified that his laboratory had reported PCB test results to 1 ppb and below, but that he had no confidence in the accuracy of these test results, given the substantial disparities between results reported by other laboratories testing identical samples. EIA Exh. F. at 5; Tr. 2632. Both EDF and EPA counsel argue that Laubscher's testimony should be limited only to the ability of his laboratory to test accurately for PCB levels in effluent water. Yet counsel overlook the fact that all of the laboratories participating in the ASTM round-robin reported great variations in the level of PCB inserted into the water sample. Neither the EPA nor EDF called any representative from any other commercial laboratory to rebut Mr. Laubscher's expert opinion.

The second comparison testing program was one conducted by all nine capacitor manufacturers, eight of whom used samples collected by the EPA and distributed for the purpose of conducting parallel comparison testing. All nine capacitor manufacturers reported great variations in the results received on these samples. The variations existed both between the results of EPA regional laboratories and private testing and between the results of different private laboratories. (EIA Exh. E at 2-3 and Exhibit B thereto)

Finally, Ray Clark of Universal Manufacturing Company testified that he had engaged in a series of comparison testing projects in an attempt to determine what level of PCB actually existed in his company's effluent water. He testified that after a number of tests he was unable to obtain any unanimity in test results involving identical samples of effluent. (EIA Exh. C at 7-8 and Exhibit B thereto; EIA Exhs. 9-12)

These three testing programs establish that, in practice, commercial laboratories have been unable to measure PCB levels in industrial effluent with any degree of accuracy at levels below 20 ppb. The over twelve different laboratories involved in the various studies all have had similar difficulties. There is no evidence that these laboratories are incompetent or employed faulty test procedures. Indeed, these are the same laboratories to which

the capacitor industry must turn for tests to determine whether plant effluent complies with the discharge limitations ultimately set by the Administrator.

IV

THE FEDERAL WATER POLLUTION CONTROL ACT REQUIRES THAT DISCHARGE LIMITATIONS EXCLUDE PREEXISTING PCB CONCENTRATIONS IN INFLUENT WATER

The proposed discharge regulations made no allowance for the existence of PCB in plant influent water, save for those cases where the influent water comes from the same source as that into which effluent is discharged. No capacitor manufacturer (with one possible exception) falls into this category. (EIA Exh. C at 9-10) Thus the regulations fail to take preexisting PCB levels into account in regulating the discharges of the capacitor industry. As we argued in our opening brief, there is no legal justification for the imposition of a penalty upon the industry requiring capacitor manufacturers to treat their effluent water for PCB over which they had no control. In its response to comments by General Motors (EPA PFF at A-8), the Agency stated that it was aware of the possibly substantial levels of PCB existing in influent water. As yet, however, the Agency has offered no rationale for its failure to take this into account in formulating discharge regulations for the capacitor industry.

CONCLUSION

The widespread use for some forty years by many different industries of highly chlorinated PCB compounds has produced a contamination of the environment by these PCB mixtures which do not degrade but persist, both in sediment, water, fish and aquatic tissue, and other organisms. Through their persistence, these PCBs pose toxic dangers to many different organisms as the PCB compounds move up the food chain. Unhappily, the nature and scope of this problem became apparent only years after the bulk of the damage had been done. The regulations involved in this proceeding cannot cure the problem. Indeed, in its opening brief the Agency acknowledged that the two industries subject to the proposed regulations "represent a relatively small part of the total environmental PCBs problem . . .." EPA Br., p. 21.

One of the two industries subject to the proposed regulations, the capacitor industry, abandoned all use of highly chlorinated Aroclor mixtures years ago in an attempt to respond to legitimate ecological concerns. Those who seek to regulate the discharges of capacitor manufacturers lightly disregard this highly significant fact. EPA counsel rely principally on loose analogies with other

PCB compounds to support their case against Aroclor 1016--resorting ultimately to the novel theory that Aroclor 1016 is "presumptively"*/ toxic. Counsel for the EDF shrilly accuse the capacitor industry of "molecule manipulation for the purpose of staying 'one step ahead of the sheriff'. . ."**/ Such inflammatory rhetoric hardly excuses their failure to introduce any evidence that Aroclor 1016 is in fact toxic.

Aroclor 1016 was in use two years before the EPA's first attempt to regulate it. That earlier proceeding of the EPA was dismissed due to the lack of any persuasive evidence that Aroclor 1016 was, in fact, toxic. In the two-year interim prior to commencement of this proceeding, the Agency did virtually nothing to improve the data base supporting regulation of Aroclor 1016. As before, the Agency relies in this proceeding on the mounting evidence that persistent and nondegradable PCB mixtures such as Aroclors 1248, 1254 or 1260 are toxic to justify its effort to ban Aroclor 1016. Yet strikingly, because Aroclor 1016 does not persist in the environment due to the substantial differences in its chemical composition, the Agency speculates that Aroclor 1016's non-persistence and its ability to be metabolized might, in some manner,

*/ EPA PFF XVI.9, p. 100.

**/ EDF Br., p. 29.

evidence its toxicity. This theory turns the alleged analogy between Aroclor 1016 and the other PCB mixtures on its head. The more highly chlorinated Aroclors should be regulated because they persist in the environment. Aroclor 1016 should be banned because it does not. Accordingly, the non-sequitur -- all "PCBs" should be banned.

The capacitor industry manufactures a product used in so many different applications that is is unthinkable that capacitors could be abandoned. Without AC capacitors, it would be impossible to have the type of lighting, home appliances, air conditioners and hundreds of other appliances on which we have come to rely. The capacitor industry uses Aroclor 1016 because it is safe for use in capacitors, economical, gives long and stable life, and creates no risk of fire. No non-PCB capacitor dielectric fluid is equally safe for use. All substitutes for Aroclor 1016 are flammable; all are dramatically more costly; no device has been shown to protect reliably against the risk of fire posed by use of the presently available substitutes. A ban on Aroclor 1016 thus presents serious safety implications, wholly removed from the fact such a ban will increase the costs of AC capacitors by 20 or 30 percent. Plainly such a ban should be adopted only on the basis of convincing evidence that Aroclor 1016 poses a greater threat than its elimination would create.

No such evidence has been produced in this proceeding. Taking the evidence presented by the Agency in its best light, the only regulation of Aroclor 1016 which could be supported is a discharge limit of 100 ppb. Certainly a total ban on use of the material in capacitors has not been shown to be necessary. Indeed, given the new program of PCB regulation created in the Toxic Substances Control Act, no regulation of Aroclor 1016 is justified under the Federal Water Pollution Control Act. In a few months the Administrator will be required to evaluate whether an exemption should be granted for PCB compounds necessary for use, and for which no suitable substitute exists. That will be the appropriate time for a determination whether Aroclor 1016 should be banned. Any premature action under the FWPCA is unjustified and improper.

Respectfully submitted,

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CERTIFICATE OF SERVICE

I, Steven S. Rosenthal, hereby certify that a copy of the foregoing Reply Brief of the Electronic Industries Association's Ad Hoc PCB Committee was hand-delivered this 5th day of January, 1977, to:

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