

chlorocarbonate, an increase in the estimated testing costs indicates that the likelihood is now uncertain.

The likelihood of significant adverse economic impact was not addressed in the original economic analysis for 24 chemicals which were believed to be manufactured solely as pesticides or not currently manufactured or imported. For 3 of these 24 chemicals, pentabromoethane, pentabromobenzene, and maleic hydrazide, the probability of significant adverse economic impact is believed to be low. For 4-bromobenzylcyanide and endrin, there is a high likelihood of significant adverse economic impact. For 2-chloroethyl vinyl ether, the likelihood of significant adverse economic impact is uncertain.

Please refer to the revised economic analysis contained in the docket for a more detailed discussion of the economic assessment for these chemicals.

VI. Rulemaking Record

EPA has established a record for this rulemaking (docket number OPTS-42088C). This record includes all information considered in the development of the proposed rule and appropriate Federal Register notices. The Agency will continue to supplement the record with additional information as it is received.

The record includes all information referenced in support of the May 29 proposal plus the following information:

- (1) Notice of Proposed Rulemaking, Solid Waste Chemicals (52 FR 20336; May 29, 1987).
- (2) Exposure data from three sources: The Industry Studies Data Base, the Hazardous Waste Damage Incident Data Base, and the Hazardous Waste Disposal Site Data Base.
- (3) Revised economic analysis for the proposed rule.
- (4) Toxicity data on methanethiol.
- (5) Literature search information for: acetamide, 2-fluoro; 2,3-dichloropropanol; and 2,6-dinitrotoluene.

VII. Other Regulatory Requirements

The Agency discussed Executive Order 12291, The Regulatory Flexibility Act, and the Paperwork Reduction Act in detail in the May 29, 1987 proposal, and no changes are indicated for this notice.

Dated: December 30, 1987.

Susan F. Vogt,

Acting Director, Office of Toxic Substances.
[FR Doc. 88-632 Filed 1-13-88; 8:45 am]

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40 CFR Part 799

[OPTS-42008D; FRL-3215-7]

Unsubstituted Phenylenediamines; Reopening of Comment Period

AGENCY: Environmental Protection Agency (EPA).

ACTION: Proposed rule and reopening of comment period.

SUMMARY: In response to comments received by the Agency in response to the proposed rule for the unsubstituted phenylenediamines (pdas), EPA is reopening the comment period to permit public comment on modifications and additions EPA is proposing in the testing program for neurotoxic, mutagenic, oncogenic, and aquatic toxicity effects.

DATES: This document reopens the period of comment on the proposed rule, which appeared in the Federal Register of January 6, 1986 (51 FR 472), until February 29, 1988.

ADDRESS: Address written comments in triplicate identified by the document control number (OPTS-42008D) to: TSCA Public Information Office (TS-793), Office of Pesticides and Toxic Substances, Environmental Protection Agency, Room NE-G004, 401 M Street SW., Washington, DC 20460.

The public record supporting these actions is available for inspection at the above address from 8 a.m. to 4 p.m., Monday through Friday, except legal holidays.

FOR FURTHER INFORMATION CONTACT:

Edward A. Klein, Director, TSCA Assistance Office (TS-799), Office of Toxic Substances, Room E-543, 401 M Street, SW., Washington, DC 20460, (202) 554-1404.

SUPPLEMENTARY INFORMATION: In the Federal Register of January 6, 1986 (51 FR 472), EPA issued a proposed rule for unsubstituted phenylenediamines (pdas) which included testing for chemical fate and aquatic toxicity for *ortho*-phenylenediamine (*o*-pda) (CAS #94-54-5) and *para*-phenylenediamine (*p*-pda) (CAS #106-50-3) and chemical fate, aquatic toxicity, mutagenicity, and oncogenicity (if triggered by mutagenicity testing and if an oncogenicity test conducted in Japan was inadequate) for *meta*-phenylenediamine (*m*-pda) (CAS #108-45-2). EPA previously extended the comment period in a document published in the Federal Register of March 5, 1986 (51 FR 7593). In response to public comments, EPA is restructuring the proposed aquatic toxicity testing for all three isomers and the proposed mutagenicity and oncogenicity testing for *m*-pda, and is now proposing that

neurotoxicity testing be conducted on all three isomers. As regards specific modifications to proposed 40 CFR 799.3300 *Unsubstituted phenylenediamines*, the addition of which was proposed in the Federal Register of January 6, 1986 (51 FR 472), EPA is proposing to modify paragraphs (c) and (e) and add new paragraph (f) concerning the effective date, 44 days after publication of the final rule. EPA will merge the two proposals in the final rule.

I. Background

In the Federal Register of January 6, 1986 (51 FR 492), *m*-pda was proposed for testing in the *Drosophila* sex-linked recessive lethal test (SLRL), in indirect photolysis, and for acute toxicity to *Daphnia*, rainbow trout, and algae. The same environmental fate testing and aquatic toxicity testing were proposed for *o*-pda and *p*-pda as for *m*-pda. For all three isomers, it was proposed that additional aquatic toxicity testing be triggered from the results of the required acute testing. No neurotoxicity testing was proposed. The rationale for requiring testing was explained in the proposal.

Comments were received from Dow Chemical Corp. (Dow), E.I. duPont de Nemours and Company (duPont), Joseph A. Lowenstein Sons, Inc., The American Psychological Association (APA), First Chemical Company, and the Naylor Dana Institute for Disease Prevention Laboratory. The public comments presented both newly developed data and data not previously reviewed by EPA for all three isomers which, after careful review, have prompted the Agency to modify the proposed mutagenicity testing for *m*-pda and the aquatic toxicity testing for all three isomers, and to propose neurotoxicity testing for all three isomers. No modifications are being proposed in this document to the proposed environmental fate testing described in the January 1986 proposal.

II. Modifications to the Proposed Testing Program for PDAS

A. Summary of Mutagenicity and Oncogenicity Issues

1. *Mutagenicity testing.* The January 1986 proposal stated that the Agency believes that exposure to *m*-pda may present an unreasonable risk of injury to human health for mutagenic effects and that data are insufficient to assess this risk. Consequently, testing of *m*-pda in the SLRL assay was proposed; if this test was positive, the mouse specific locus assay (MSL) and oncogenicity

testing would be triggered. DuPont's comments (Ref. 2 in Unit IV, B. below) were the only ones which addressed mutagenicity testing.

DuPont supported its arguments (Ref. 2) for not testing *m*-pda with references by Lee et al. (Ref. 3), Vogel et al. (Ref. 4), studies done for the National Toxicology Program (NTP) (Ref. 2), Seiler (Ref. 6), Tanaka and Katoh (Ref. 7), Picciano, et al. (Ref. 8), and Ashby (Ref. 9) and with references in the C9 aromatic hydrocarbon fraction rule (see the 50 FR 20662; May 18, 1985). DuPont recognizes that *m*-pda causes mutations *in vitro* in the Ames test and the Chinese hamster ovary chromosomal aberration test (CHO) (Ref. 24) and that it inhibits mouse testicular cell DNA synthesis *in vitro* (Ref. 6), but DuPont argues that because *m*-pda is negative in the dominant lethal assay in male rats (Refs. 25 and 26), no significant new information would be generated by requiring additional chromosomal aberration or somatic cell gene mutation studies on this substance. DuPont also suggested alternative testing which involves dermal exposure of rat testis to labeled *m*-pda and measuring DNA binding in the testicular cells as the only justifiable testing.

The Agency has thoroughly reviewed the comments and data submitted by DuPont. These data were insufficient to change the Agency's findings that the mutagenic potential of *m*-pda is inadequately characterized. The Agency still believes that testing of *m*-pda for gene mutation in mice (Figure 1 below) is needed to adequately characterize the mutagenic potential of this isomer. However, EPA is modifying its testing proposal for *m*-pda in response to DuPont's comment questioning whether *p*-das reach germ cell tissue and cause chromosomal aberrations by adding chromosomal aberration testing in mice to the proposed testing. The mouse is proposed as a test species for the *in vivo* tests because *m*-pda has been shown to accumulate in mouse testicular tissue (Refs. 6 and 7). These data, combined

with the negative dominant lethal data in the rat, support using the mouse as the species of choice for further chromosomal aberration testing (Refs. 25 and 26). The Agency is modifying its testing proposal for *m*-pda to include testing in the *in vivo* mammalian bone marrow cytogenetics test—chromosomal analysis (MBMC), in the mouse, according to 40 CFR 798.5385. If this test is positive, a mouse dominant lethal assay would be triggered, to be conducted according to 40 CFR 798.5450 (Figure 1 below). A positive dominant lethal assay would trigger a heritable translocation assay (subject to a public program review), to be conducted according to 40 CFR 798.5460. No further chromosomal aberration testing would be required if the MBMC is negative. As in EPA's original proposal, *m*-pda would also be tested in the SLRL assay, 40 CFR 798.5275, which if positive would, subject to a public program review, trigger MSL testing, in accordance with 40 CFR 798.5200.

The Agency continues to believe that the proposed triggers and upper-tier testing provide the most reliable description of the mutagenic potential of a chemical substance. EPA's rationale was discussed in its proposed TSCA section 4 rule for C-9's (50 FR 20662; May 17, 1985). Results from the SLRL and chromosomal testing would be included in the decision logic at the public program review stage of the weight-of-evidence determination of the need for the higher-tiered mutagenicity testing.

As to DuPont's proposed testicular binding testing in rats (Ref. 2), the Agency would consider any additional DNA binding data submitted prior to the public program review stage of the process (Figure 1 below) as part of the total evaluation of the mutagenicity potential for *m*-pda. However, since this DNA binding test does not provide evidence for the potential heritability of any effects which may be demonstrated, the Agency does not believe this test should be part of the required test

program for *m*-pda. DuPont also proposed that *m*-pda be tested in the rat, a species already shown to be negative with respect to the dominant lethal effect (Refs. 25 and 26). Therefore, the Agency is suggesting that the mouse be the species of choice for DNA binding studies, if industry elects to include results from this test for Agency consideration.

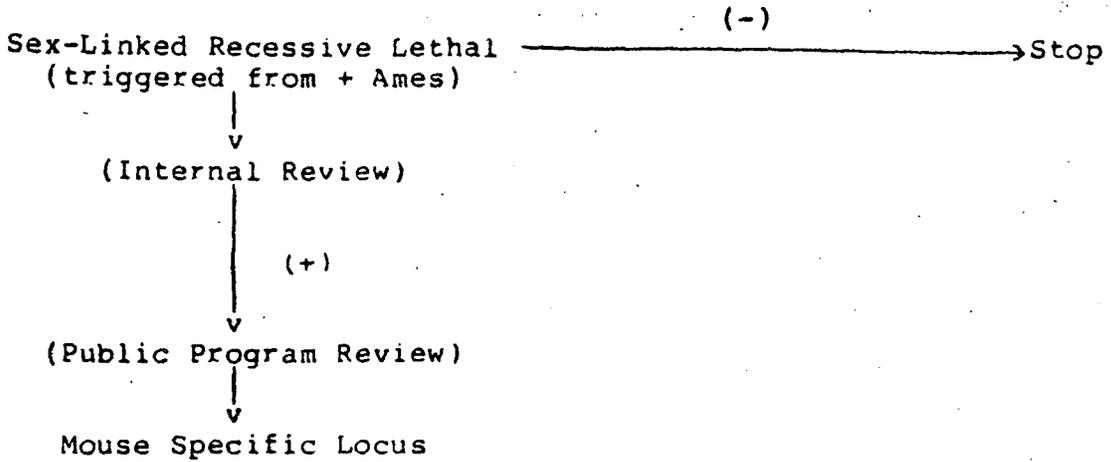
2. *Oncogenicity testing.* The proposal stated that oncogenicity testing of *m*-pda would be initiated if the SLRL were positive and the results from oncogenicity testing in progress in Japan were inadequate for Agency purposes.

In response to the public comments received, the Agency is proposing the additions to the mutagenicity testing program described above. EPA's standard chromosomal aberration testing scheme, as described in EPA's C9 rule (50 FR 20662; May 17, 1985) includes provisions for triggering oncogenicity testing from a positive *in vitro* cytogenetics test such as the Chinese hamster ovary (CHO) assay. Because a positive CHO is available (Ref. 25), EPA is proposing that the oncogenicity bioassay on *m*-pda be conducted without further triggering. However, the Agency is also proposing that review of all the available scientific evidence (including the results of the proposed mutagenicity testing program described in Figure 1 below and the oncogenicity study in progress in Japan) be concluded before the chronic assay is initiated. If, in EPA's judgment, the evidence indicates that *m*-pda oncogenicity potential is adequately characterized, the Agency proposes to solicit public comment on whether it should rescind the requirement for the oncogenicity test. If in EPA's judgment the evidence indicates a need for oncogenicity testing, the Agency will notify the test sponsors to initiate the chronic study by a certified letter or by notice in the **Federal Register**.

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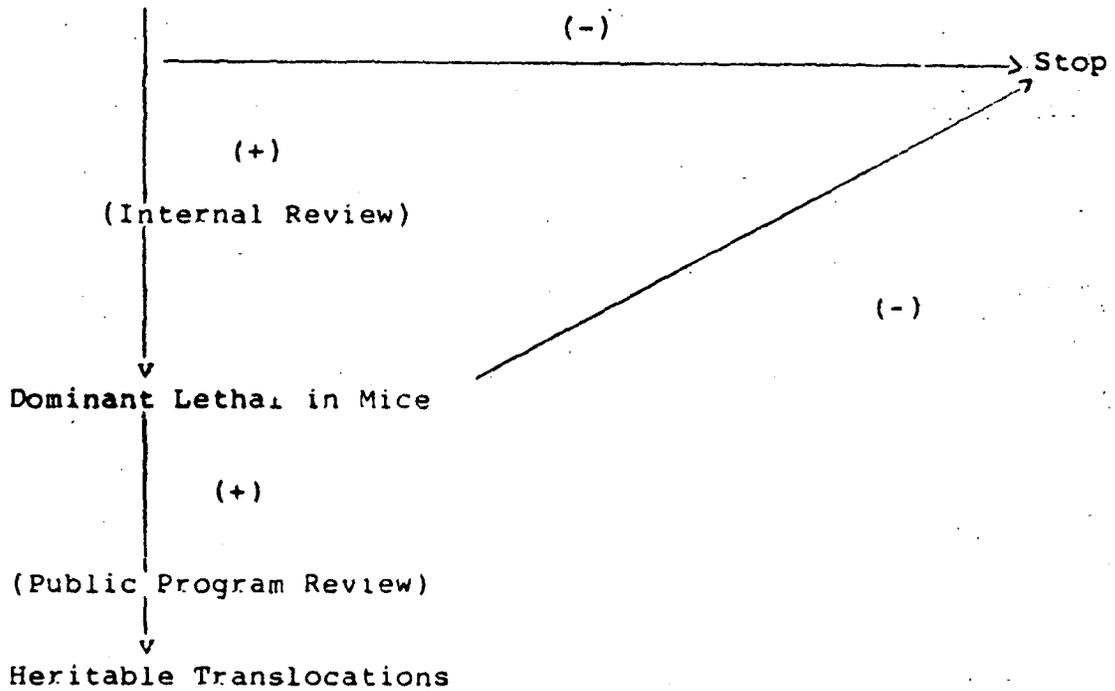
Figure 1: PROPOSED MUTAGENICITY TESTING PROGRAM

I. GENE MUTATION



II. CHROMOSOMAL ABERRATION TESTING

Mouse Bone Marrow



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B. Summary of Neurotoxicity Issues

The January 1986 proposal stated that neurotoxicity testing was not being proposed for the pdas. The neurotoxicity concerns identified by the Interagency Testing Committee (ITC) were based upon the potential formation of methemoglobin in people exposed to pdas. Secondary effects from the methemoglobin development could include neurotoxic effects. Pda manufacturers supplied information showing that methemoglobinemia could not be documented in people working in the manufacture of these substances. Consequently the Agency did not propose neurotoxicity testing for pdas. Comments were received from the Neurobehavioral Toxicity Test Standard Committee (NTTSC), Psychopharmacology Division of the American Psychology Association (Ref. 14). The NTTSC argued that the pdas produce adverse effects on the central nervous system and that additional testing is necessary to characterize this damage.

NTTSC argued that the pdas could metabolize into chemicals which are highly reactive with tissue nucleophiles. This reactivity could lead to biological effects on the central nervous system. These potential effects could result in behavioral modifications at levels substantially below those that may cause methemoglobinemia. According to NTTSC, convulsive activity in humans resulting from chemical toxicity is already known to occur with some non-pdas at levels substantially below those which may cause methemoglobin formation. NTTSC states that 3 to 6 people per 1,000 are epileptic and that these people would be especially susceptible to potential convulsive agents. Both *m*-pda and *p*-pda have been reported to induce seizures in experimental animals.

NTTSC (Ref. 14) argued that there are enough people exposed and enough unsubstituted phenylenediamines production to warrant testing. NTTSC further argues that sufficient information exists to suggest the pdas may pose a risk of neurotoxic effects and therefore these tests should be included in the required testing program. NTTSC also suggested a strategy to evaluate neurotoxic potential which was discussed in detail in their comments. To support their arguments, NTTSC provided the following documentation.

Effects in humans caused by the pdas according to NTTSC include: sleepiness, headache, paresthesia, gastrointestinal irritation, changes in reflex excitability, increased respiration, body temperature, and pulse rate (Close, Ref. 18 and

Berger, Ref. 19), and visual disturbances (Berger, Ref. 19).

Toxicity of *p*-pda in rabbits was studied by Erdmann and Vahlen (Ref. 15), Pollak (Ref. 16), and Puppe (Ref. 17). Oral administration, subcutaneous injection, or topical application resulted in both clonic and tonic spasms just prior to death. In dogs, both oral administration and subcutaneous injections resulted in lethargy followed by clonic and tonic spasms prior to death (Refs. 15 and 17). The dogs also demonstrated edema of the eyes, reddening of the conjunctiva (Ref. 15), exophthalmus and increased intraocular pressure (Ref. 17).

A major review article in the Journal of the American College of Toxicology provided additional information which indicates that *p*-pda interferes with the normal metabolism of isolated guinea pig brain tissue (Ref. 19). In mice, *p*-pda accumulated in the brain within 24 hours after application to shaved areas of the animals. The *p*-pda was not detected in brain tissue 48 hours after the treatment. In both humans and monkeys, *p*-pda in hair dyes accumulated in the hair shaft and was excreted in the urine of both species up to seven days after the application of the dye.

EPA has evaluated the NTTSC arguments and reviewed their submissions. Even though some of these data are over 50 years old, the Agency finds that (1) the manufacturing, processing, and use of the pdas may present an unreasonable risk of neurotoxic effects because as many as 1,000 workers involved in the manufacture, processing, and use of the three pda isomers are potentially exposed to all three isomers, and the data presented by NTTSC suggest that the pdas may cause acute and subchronic neurotoxic effects; (2) the data are insufficient to characterize the neurotoxic potential of the pdas; and (3) testing for neurotoxic effects is necessary to answer these questions.

Therefore, EPA is now proposing that all three pda isomers be tested for neurotoxic effects in rats in the neurotoxicity functional observational battery (FOB), according to 40 CFR 798.6050 and the motor activity test (MAT), according to 40 CFR 798.6200. These tests are designed to be conducted either independently or as an additional parameter of another acute or subchronic health effects test. Because no subchronic testing is being proposed, EPA is combining the FOB and the MAT to provide the neurotoxicity testing program specifically for the pdas. EPA is proposing that the pdas be initially

tested for acute neurotoxic effects and that they be administered by oral exposure. Clinical observations would be made, at a minimum, before dosing and at 1, 4, 24, and 48 hours and at 7 days. Both positive and negative controls would be used and the dose range would be as required by the FOB, according to 40 CFR 798.6050. Motor activity would be measured at time of peak effects as determined using FOB. The two acute studies would be structured as described in 40 CFR 798.6200 and conducted so that the requirements of the two tests are not violated.

If positive neurotoxic effects are observed at 24 hours or later, a 90-day subchronic FOB, MAT, and neuropathology test would be conducted according to 40 CFR 798.6050, 798.6200, and 798.6400, respectively. At the end of the subchronic testing, animals would be sacrificed and the nervous tissue preserved for histopathological examination as described in 40 CFR 798.6400.

At the completion of the histopathological examination, data would be submitted to the Agency. The final report for the acute toxicity shall be received by EPA within 6 months, and those for subchronic neurotoxicity testing and neuropathological examination shall be received by EPA within 15 months of the effective date of the final test rule.

C. Summary of Chemical Fate Issues

The Agency states its findings in the January 1986 proposal that pdas may enter the aquatic environment in sufficient quantities and persist long enough that exposure to the pdas may present an unreasonable risk of injury to aquatic organisms. However, persistence data are lacking, and testing is therefore necessary to estimate pda persistence in ambient waters. The proposal presented a new test guideline, the indirect photolysis test, to predict removal of chemicals in ambient waters and proposed it as a test standard for pdas. The indirect photolysis test requires that humic acids be added to the test waters because humic acids may play a key role in indirect photolysis of pdas in the environment. Comments on the chemical fate testing were received from DuPont (Ref. 2).

DuPont argued (Ref. 2) that the aquatic oxidation rate study which it submitted on the three pda isomers closely parallels the conditions under which the aquatic toxicity studies (see Unit II.D below) were conducted and adequately simulate the removal of pdas from ambient waters by oxidation.

DuPont also believes humic acid is present in its well water. However, no documentation quantifying the humic acid was included in the oxidation rate studies. DuPont further argues that the oxidation rate studies in its submission included rate-constant determinations under both light and dark laboratory conditions and that the rate constants for each isomer were similar under both light and dark testing conditions. Also, duPont argued EPA did not provide reference to situations where oxidation occurs in the absence of light, to justify testing these chemicals in the dark. DuPont argues that under the conditions of its study, *p*-pda is so reactive in aqueous solutions that no additional significant information would be gained from the required testing.

EPA analyzed the oxidation rate studies and disagrees with duPont that the oxidation rate studies submitted in the public comments (Ref. 2) and EPA's proposed indirect photolysis study are comparable and that additional testing is not necessary.

The proposed indirect photolysis test measures oxidative rates: (1) In sunlight; (2) under controlled conditions to minimize, or eliminate, biodegradation, volatilization, sorption, etc.; and (3) in the presence of dissolved humic acids, a critical ingredient in indirect photolysis (oxidation). DuPont measured loss of pdas under conditions with fluorescent light, no dark controls, and no dissolved humic acids. Fluorescent light does not resemble sunlight in wavelength distribution and light intensity. Dissolved humic acids play a key role in indirect, oxidative photolysis of the pdas in the environment and were not included in duPont's studies in measured quantities.

The duPont data from the experiments on *o*-, *m*-, and *p*-pda in Haskell well water were fitted to a first-order loss of diamine at the initial diamine concentration of 2.5 and 25 mg/L. For all three isomers, the half-life increased at the higher concentration. In a first-order rate process, the half-life is independent of the concentration of the

substrate. Biodegradation probably did not play a role in the results for *p*-pda, since microbial counts were relatively low and the duration of the experiment was 8 hours. However, for *m*- and *o*-pda biodegradation may have had significant influence on the results since the microbial counts were relatively high and the experimental duration of 21 days was quite long. In all experiments, the loss of diamine was all that was demonstrated. In no case was it shown that decomposition products were formed. Consequently, EPA is unsure that oxidation of the diamine was being measured in these experiments.

In the experiments measuring loss of *p*-pda in Delaware River water, diamine loss was measured with and without aeration and the half-lives were very similar (aerated half-life was approximately 4.0 hours and the non-aerated half-life was approximately 4.7 hours). If oxidation had occurred, the aerated sample of *p*-pda should have decomposed considerably faster than the non-aerated sample.

Consequently, the Agency has not received any information in the public comments which causes it to modify the indirect photolysis testing proposed and thus continues to propose that it be conducted.

D. Summary of Aquatic Toxicity Issues

EPA proposed aquatic toxicity testing for all three isomers, according to a specific environmental effects testing scheme. Positive results would trigger additional acute or chronic testing. Comments were received from duPont (Ref. 2) and Dow (Ref. 21) addressing the aquatic toxicity program for pdas.

DuPont argued that the aquatic toxicity data submitted in October 1985 (Ref. 22) are sufficient to characterize the aquatic toxicity of all three isomers. Moreover, duPont argued the use of fathead minnow and static test systems provided useful data, as indicated by the broad range of sensitivity exhibited by the fathead minnow to these three isomers and the extreme sensitivity of the minnow to *p*-pda. In claiming that testing in more than one fish species

was unnecessary, duPont calculated predicted environmental concentrations (PEC) of 3.5 ppd and 6.5 ppb for *o*- and *m*-pda from the data submitted in October 1985 (Ref. 23). Application of 100X and 1,000X PEC as prescribed in the proposed rule led DuPont to conclude that no additional fish toxicity testing would be triggered. DuPont also argued in response to issues raised in the proposal, that if fish testing was necessary, precedent for using freshwater fish toxicity studies to predict chemical toxicity to brackish and saltwater fish has been adequately established in the open literature. Therefore, duPont contended no testing in saltwater fish could be justified.

EPA agrees that for pdas, testing of brackish or saltwater organisms is not necessary since pdas are not expected to enter saltwater and is therefore now proposing that any testing of pdas in fish be conducted only in freshwater species. However, EPA disagrees that testing only in fathead minnows adequately characterizes the toxicity of pdas in aquatic vertebrates.

The Agency has evaluated the acute aquatic toxicity data submitted by duPont for *o*-, *m*-, and *p*-pda in fish, invertebrates, and algae in the following Table 1, and believes that additional aquatic toxicity testing is necessary for all three isomers.

TABLE 1.—Acute Toxicity of PDAS to Aquatic Organisms (ppm)

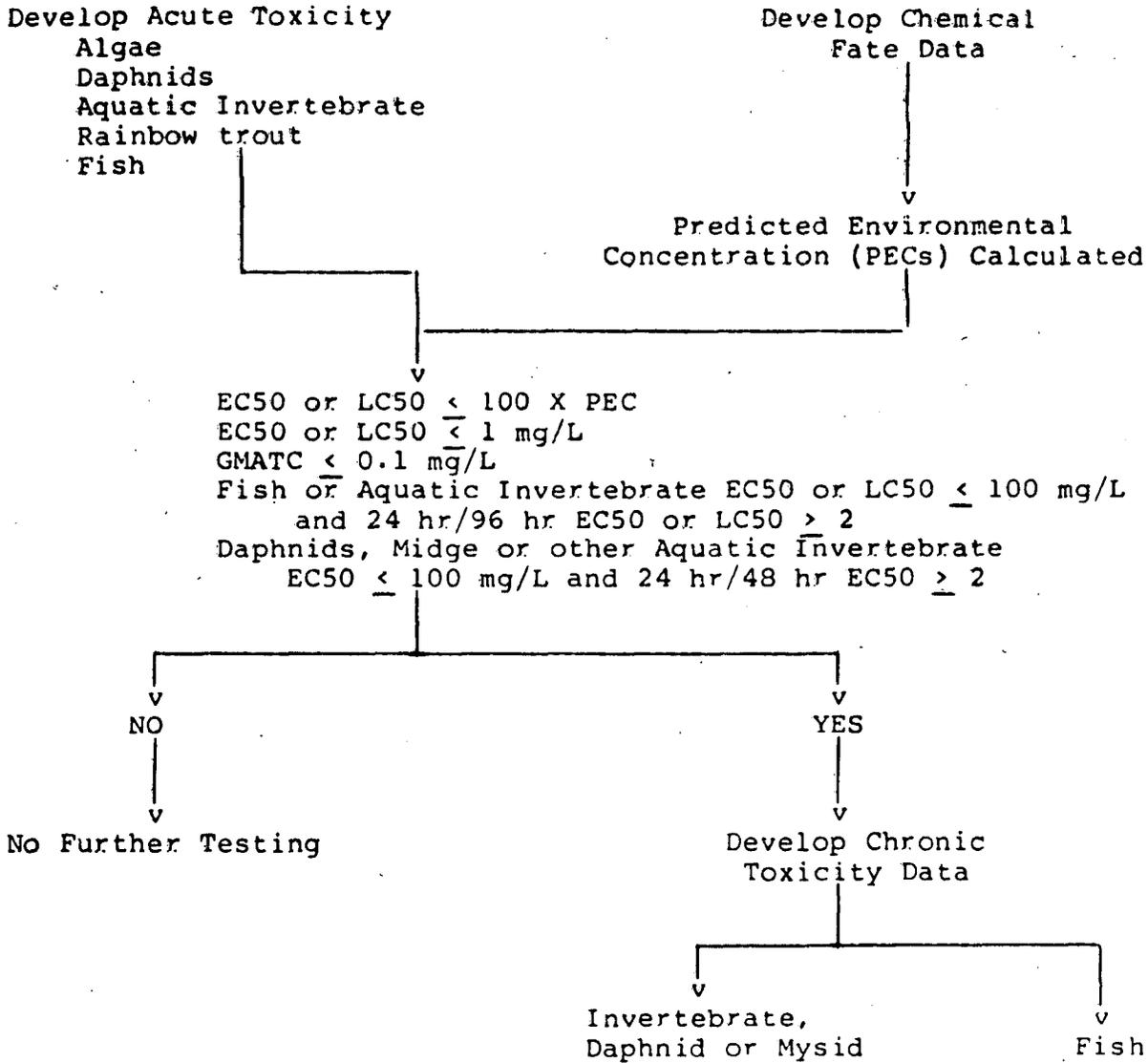
Substance	Aquatic organisms		
	Fathead minnow (FM)	Daphnia magna (DM)	Selenastrum capricornutum
<i>o</i> -pda	44	0.88	0.16
<i>m</i> -pda	1614	5.9	2.4
<i>p</i> -pda	0.057	0.28	0.28

(Ref. 22)

The inconsistencies in the toxicity data between chemicals and among organisms also leads EPA to propose refined decision criteria for aquatic testing in the following (Figure 2).

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Figure 2 -- PROPOSED PDA DECISION LOGIC FOR DEVELOPING DATA FOR AQUATIC ORGANISMS



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These data demonstrate that for unsubstituted pdas there are large differences in LC50 values in the different species tested. Because of these large differences, the decision criteria in Figure 2, and the requirements outlined in the NPRM, the Agency believes that additional data should be developed for pdas.

Both DuPont (Ref. 2) and Dow (Ref. 21) argued that *Gammarus* is an inappropriate test species for two reasons: *Daphnia* tend to be a more sensitive species than *Gammarus*; and the gammarid test has not been subjected to the same intense peer review as the daphnid test. They therefore contend that any data generated by the required testing would provide little useful information for hazard assessment at this time.

EPA has used toxicity data developed for *Gammarus* as part of its evaluation of chemical impact on aquatic systems; examples are included as references 31, 32, and 33, and more recent examples of *Gammarus* being tested for toxic effects are included as references 27, 28, 29, and 30. Industry did not provide data which showed *Gammarus* to be an inadequate test organism. EPA finds no evidence to cause it to modify its proposed use of *Gammarus* as a test species.

DuPont further argued that the chronic *Daphnia* test submitted by duPont (Ref. 22) is adequate to judge the chronic toxicity of *m*-pda to the animals, and therefore no additional testing is needed.

EPA believes that the toxicity of *m*-pda to aquatic invertebrates is still inadequately characterized and that the proposed testing (below) in both *Daphnia* and *Gammarus* is necessary to adequately characterize differences in species sensitivity to *m*-pda.

DuPont (Ref. 2) also argued that the flow-through system required by EPA's proposed test standards would not provide different information from the static data which they submitted since these chemicals, especially *m*-pda, are very volatile and under flow-through systems these substances would rapidly assume the same levels as those found in the static test systems. DuPont further argues that the flow-through system would create logistical problems with *Daphnia* testing, namely, loss of animals resulting from flushing the test chambers with fresh test water.

EPA disagrees that static systems would be better for testing the pdas in aquatic systems. Flow-through systems are designed to maintain constant exposure levels to unstable chemicals and are therefore being proposed for the testing described below. However, the

Agency recognizes that several of the static acute toxicity tests indicate that *o*- and *p*-pdas are highly toxic to selected organisms, and to repeat the acute toxicity testing of *o*- and *p*-pdas using the same organisms and flow-through conditions would not be a cost-effective use of resources. Therefore, EPA is proposing certain additional tests as described below.

In response to EPA's request for comment on the appropriateness of using one isomer as a surrogate for testing toxicity of the pdas, duPont contends that sufficient acute toxicity data are available for all three isomers and that the chronic data for *m*-pda are adequate for making a prediction of chronic toxicity for this category of chemicals. The data submitted by duPont indicate that the toxicity of the three isomers may vary widely among the species tested and that *o*- and *p*-pda may be more toxic than *m*-pda. Consequently, the Agency disagrees that the chronic toxicity of *m*-pda to either fish or invertebrates would provide an adequate prediction of the chronic toxicity of the other isomers to these organisms, and is therefore proposing chronic toxicity testing for any of the isomers which meet the decision criteria for triggering chronic testing.

The Agency is proposing that *p*-pda be tested for acute toxicity with rainbow trout and *Gammarus* in accordance with 40 CFR 979.1400 and 795.120, respectively. On the basis of LC50 values, early lifestage testing would be conducted with the more sensitive fish (fathead minnow or rainbow trout) in accordance with § 797.1600. The concentration of pda would be measured before, during, and at the end of testing. The results from the acute studies on *p*-pda would be incorporated into the pda test scheme to determine whether all chronic toxicity testing is triggered and the appropriate organism(s) in which to conduct the chronic testing.

The Agency is proposing that *o*-pda be tested for acute toxicity with the rainbow trout and *Gammarus*, in accordance with 40 CFR 797.1400 and 795.120, respectively. Using the fathead minnow LC50 value, and the 24/96 hr LC50 ratio to be calculated from the testing data, early life stage testing would be conducted with the more sensitive fish (fathead minnow or rainbow trout) in accordance with 40 CFR 797.1600. The concentration of *o*-pda would be measured during and at the beginning and end of the study. The results from the acute studies on *o*-pda would be incorporated into the pda test scheme to determine whether chronic toxicity testing is triggered and the

appropriate organism(s) in which to conduct the chronic testing.

The Agency is proposing that *m*-pda be tested for acute toxicity with the rainbow trout and the *Gammarus* in accordance with 40 CFR 797.1400 and § 797.120 respectively. *m*-Pda is moderately toxic to *Daphnia*. During the acute toxicity study, the concentration of *m*-pda was 63 percent of the nominal concentration at 48 hours. The Agency requires these tests to be repeated when the measured concentration is substantially less than the nominal concentration, unless a decision criterion is satisfied that requires a subsequent test to be conducted. Since the *Daphnia* EC50 < 100 × PEC, the Agency is proposing to require a *Daphnia* chronic test to be conducted in accordance with 40 CFR 797.1330. Although duPont submitted chronic data for *Daphnia*, they are inadequate for regulatory purposes because an acceptable MATC was not determined.

Testing of all three isomers would be conducted in flow-through systems. Reporting requirements would remain as in the January 6 proposal.

E. Issues for Comment

The Agency solicits comments on issues related to the proposed environmental effects testing scheme and for the proposed neurotoxicity and mutagenicity testing.

1. Dow (Ref. 21) argued that more scientific rationale is needed for justification of the decision criteria (i.e., 100 X) proposed for triggering additional testing in Figure 2 above.

The Agency recognizes that decision criteria have certain weaknesses and that toxic effects for different classes of chemicals in different species of organisms may vary widely. Toxic effects may also vary widely within specific categories of chemicals, as is the case for pdas. However, the Agency believes that the decision criteria in the testing scheme presented in Figure 2 are adequate for purposes of this rule. If data exist which support use of different decision criteria for more efficient assessment of chemical toxicity to the environment, the Agency encourages submission of these data during this extension of comment for the pdas proposed test rule.

2. EPA is considering expanding the analytical portion of the *p*-pda aquatic toxicity testing by requiring a quantitative analysis of the breakdown products present in the test solution at the onset and termination of the acute test. The acute toxicity data submitted by DuPont for *p*-pda indicated continued toxic effects throughout the 96-hour test

period at levels below the detection limit for this substance. The Agency is concerned that either *p*-pda is extremely toxic to aquatic organisms of that it is oxidized very rapidly into toxic compounds which are causing the observed toxicity. The Agency believes that the mode of toxicity for *p*-pda should be identified. Consequently, the Agency is soliciting comment on the degree of sensitivity of current analytical techniques and whether they could be modified to provide more sensitive detection of *p*-pda. The Agency is also requesting comments on the analytical methodology for the *p*-pda oxidation products, their level of sensitivity, and the materials balance necessary to account adequately for the *p*-pda added to the test chamber.

3. Neurotoxicity testing has been proposed for *o*-, *p*- and *m*-pda. The Agency solicits comments on the testing program presented above.

4. The mutagenicity testing program for *m*-pda has been modified to include chromosomal aberration testing in the mouse. Comments are sought on the addition of the chromosomal aberration testing and the selection of the mouse as the test species.

F. Reporting Requirement

The January 6, proposed rule for pdas contains language about the submission of study plans, for some tests, that applies only to two-phase rules. In accordance with 40 CFR Part 790 under single-phase rulemaking procedures, test sponsors for pdas would be required to submit individual study plans at least 45 days before the initiation of each study.

III. Economic Analysis of Proposed Rule

EPA has analyzed the potential economic impact of the total testing program proposed for all three isomers. The estimated costs for testing *p*-, *m*-, and *o*-pda, assuming maximum testing, are \$182,000, \$1,330,000, and \$182,000, respectively, or an estimated total cost for all three isomers of \$1.69 million. The total estimated annualized cost (7 percent interest for 15 years) is \$186,000. Based upon 1984 production figures of 35 million pounds, the total unit cost of testing is estimated to be 0.0053 \$/lb. The worst-case estimated costs of testing as percentages of current market price for *p*-, *m*-, and *o*-pda are 0.13, 0.26, and 0.16 percent, respectively. This is not considered to be a significant economic impact.

IV. Rulemaking Record

EPA has established a record for this rulemaking (docket number OPTS-42008D). This record includes all information considered in the

development of the proposed rules and appropriate Federal Register notices. The Agency will continue to supplement the record with additional information as it is received.

The record includes all information referenced in support of the January 6 proposal plus the following information:

(1) Notice of Proposed Rulemaking, unsubstituted phenylenediamines (51 FR 472).

(2) DuPont. "Comments of E.I. DuPont de Nemours & Co., Inc., Wilmington, Delaware 19898. 40 CFR Parts 796, 797, & 799, Unsubstituted Phenylenediamines—Proposed Test Rule. Document Control No. OPTS-42008B." Washington, DC: Office of Toxic Substances, U.S. Environmental Protection Agency, 1986.

(3) Lee, W.R., S. Abrahamson, R. Valencia, E.S. von Halle, F.E. Wurgler, and S. Zimmering. "The sex-linked recessive lethal test for mutagenesis in *Drosophila melanogaster*. A report of the U.S. Environmental Protection Agency Gene-Tox Program." *Mutation Research*. 123:183-279.

(4) Vogel, E.W., H. Frei, M.K. Fujikawa, et al. "Summary report on the performance of *Drosophila* assays." In *Progress in Mutation Research*. Vol. 5. Elsevier, Amsterdam. J. Ashby and F.J. deSerres, pp. 47-57. 1985.

(5) Vogel, E.W., J.A. Zijlstra and W.G.H. Blijleven. "Mutagenic activity of selected aromatic amines and polycyclic hydrocarbons in *Drosophila melanogaster*." *Mutation Research*. 107:53-77. 1983.

(6) Seiler, J.P. "Inhibition of testicular DNA synthesis by chemical mutagens and carcinogens. Preliminary results in the validation of a novel short term test." *Mutation Research*. 46: 305-310. 1977.

(7) Tanaka N. and M. Katoh. "Unscheduled DNA synthesis in the germ cells of male mice *in vivo*." *Japanese Journal of Genetics*. 54(6): 405-414. 1979.

(8) Picciano, J.C., W.E. Morris, S. Kwan, and B.A. Wolf. "Evaluation of teratogenic and mutagenic potential of the oxidative dyes, 4-chlororesorcinol, *m*-phenylenediamine, and pyrogallol." *Journal of the American College of Toxicology*. 2(4): 325-333. 1983.

(9) Ashby, J. "Gonadal genotoxicity assays as practical surrogates for germ-cell mutagenicity assays." *Environmental Mutagenesis*. 7: 263-266. 1985.

(10) First Chemical Corporation. "Response to proposed test rule on unsubstituted phenylenediamines by Sorell L. Schwartz, Ph.D., Georgetown University." Washington, DC: Office of Toxic Substances, U.S. Environmental Protection Agency, 1986.

(11) Holland, J.M., D.G. Gosslee and N.J. Williams. "Epidermal carcinogenicity of bis(2,3-epoxycyclopentyl) ether, 2,2-bis(*p*-glycidylloxyphenyl)propane, and *m*-phenylenediamine in male and female C3H and C57BL/6 mice." *Cancer Research*. 39: 1718-1725. 1979.

(12) Burnette, C., B. Lanman, R. Giovacchini, et al. "Long-term toxicity studies on oxidation hair dyes." *Food and Cosmetic Toxicology* 13:353-357. 1975.

(13) Weisburger, E.K., A.B. Russfield, F. Hamburger, et al. "Testing of twenty-one environmental aromatic amines and

derivatives for long-term toxicity or carcinogenicity." *Journal of Environmental Pathology and Toxicity*. 2: 325-356. 1978.

(14) Comment on the proposed test rule for the unsubstituted phenylenediamines (51 FR 472; January 6, 1986). Cover Memo: Psychopharmacology Division of the American Psychological Association. Ronald W. Wood, Chairman. Washington, DC: Office of Toxic Substances, U.S. Environmental Protection Agency, 1985.

(15) Erdmann, E. and E. Vahlen. "On the effects of *p*-phenylenediamine and quinonediaimine." *NS Archiv für Experimentelle Pathologie und Pharmakologie* 401-418. 1905. (German with English translation)

(16) Pollak, E. "A Case of Paraphenylene Diamine Poisoning." *Wiener Klinische Wochenschrift*, 31: 712-715. 1900. (German with English Translation)

(17) Puppe, E. "On Paraphenylene Intoxication." *New York Academy of Medicine*. 116-127. 1896. (German with English translation)

(18) Close, W.J. "A case of poisoning from hair dye (paraphenylenediamine)." *Medical Journal of Australia*. January 9: 53-54. 1932.

(19) Berger, E. "Visual disturbance due to the use of hair dye containing anilin." *Archives of Ophthalmology*. 38: 397-400. 1909 (July).

(20) Final Report on the Safety Assessment of *p*-Phenylenediamine. *Journal of the American College Toxicology*. 4(3): 203-266. 1985.

(21) Dow Chemical Co. Letter from Carlos M. Bowman, Ph.D. "Response to Notice of Proposed Rule Making." Washington, DC: U.S. Environmental Protection Agency, Office of Toxic Substances. March 12, 1986.

(22) DuPont. Voluntary submission by E.I. DuPont de Nemours & Co., Inc., of aquatic toxicity testing. Washington, DC: U.S. Environmental Protection Agency, Office of Toxic Substances. 1985.

(23) Naylor Dana Institute for Disease Prevention, American Health Foundation, comments from John H. Weisburger. March 20, 1986.

(24) Ishidate, Jr. M. and K. Yoshikawa "Chromosomal aberration tests with Chinese hamster cells *in vitro* with and without metabolic activation. A comparison study on mutagens and carcinogens" *Archives of Toxicology* (Suppl. 4): 41-44. 1980.

(25) Burnette, C., R. Loehr, and J. Corbett. "Dominant lethal mutagenicity study on hair dyes." *Journal of Toxicology and Environmental Health*. 1: 325-328. 1977.

(26) Sheu, C.W. and S. Green. "Dominant lethal assay of some hair-dye components in random-bred male rats." *Mutation Research*. 68: 85-98. 1979.

(27) Spehar, R.L., H.P. Nelson, M.J. Swanson, and J.W. Renous. "Pentachlorophenol toxicity to amphipods and fathead minnows at different test pH values." *Environmental Toxicology and Chemistry*. 4(3):389-397. 1985.

(28) Sanders, H.O., J.B. Hunn, E. Robinson-Wilson, and F.L. Mayer. "Toxicity of seven potential polychlorinated biphenol substitutes to algae and aquatic

invertebrates." *Environmental Toxicology and Chemistry*. 4(2):149-154. 1985.

(29) Nebeker, A.V., M.A. Cairns, J.H. Gaskstatter, et al. "Biological methods for determining toxicity of contaminated freshwater and sediments to invertebrates." *Environmental Toxicology and Chemistry*. 3(4):617-630. 1984.

(30) Ewell, W.S., J.W. Gorsuch, R.D. Kringle, et al. "Simultaneous evaluation of the acute effects of chemicals on seven aquatic species." *Environmental Toxicology and Chemistry*. 5(9):831-840. 1986.

(31) U.S.E.P.A. "Ambient water quality criteria for aldrin/dieldrin." U.S. Environmental Protection Agency. EPA 440/5-80-019. 1980.

(32) U.S.E.P.A. "Ambient water quality criteria for toxaphene." U.S. Environmental Protection Agency. EPA 440/5-80-076. 1980.

(33) U.S.E.P.A. "Ambient water quality criteria for polychlorinated biphenyls." U.S. Environmental Protection Agency. EPA440/5-80-068. 1980.

(34) Brooke, L.T., D.J. Call, D.L. Geiger, and C.E. Northcott (eds). "Acute toxicities of organic chemicals to fathead minnows. Volume I." Center for Lake Superior Environmental Studies, University of Wisconsin-Superior, Superior, Wisconsin. 54880 (Available for purchase from Center for Lake Superior Environmental Studies 715/394-8426). 1984.

(35) Geiger, D.L., C.E. Northcott, D.J. Call, and L.T. Brooke (eds). "Acute toxicities of organic chemicals to fathead minnows. Volume II." Center for Lake Superior Environmental Studies, University of Wisconsin-Superior, Superior, Wisconsin. 54880 (Available for purchase from Center for Lake Superior Environmental Studies 715/394-8426). 1985.

(36) Geiger, D.L., S.H. Poirier, and D.J. Call (eds). "Acute toxicities of organic chemicals to fathead minnows. Volume III." Center for Lake Superior Environmental Studies, University of Wisconsin-Superior, Superior, Wisconsin. 54880 (Available for purchase from Center for Lake Superior Environmental Studies 715/394-8426). 1986.

(37) Significant new uses of chemical substances; General Provisions for New Chemical follow-up (52 FR 15594; April 29, 1987).

(38) U.S.E.P.A. "Testing costs for unsubstituted Pdas." Computer printout, U.S. Environmental Protection Agency. November 13, 1986.

V. Other Regulatory Requirements

The Agency discussed Executive Order 12291, The Regulatory Flexibility Act, and the Paperwork Reduction Act in detail in the January 1986 proposal, and no changes are indicated for this notice.

List of Subjects in 40 CFR Part 799

Chemicals, environmental protection, hazardous substances, reporting and recordkeeping requirements.

Dated: December 30, 1987.

Susan F. Vogt,

Acting Director, Office of Toxic Substances.

Therefore, it is proposed that 40 CFR Part 799 be amended as follows:

PART 799—[AMENDED]

1. The authority citation for Part 799 would continue to read as follows:

Authority: 15 U.S.C. 2603, 2611, 2625.

2. In proposed § 799.3300 by adding new paragraphs (c)(1)(i) (C), (D), and (E) and (c)(3) and (f) and by revising the following paragraphs: (c)(1)(ii) and (2); (e)(1)(i) (A), (B), and (C); (e)(1)(ii)(C); (e)(2)(i) (A) and (B); and (e)(2)(ii)(C), to read as follows:

§ 799.3300 Unsubstituted phenylenediamines.

* * * * *

(c) * * *

(1) * * *

(i) * * *

(C) The *in vivo* mammalian bone marrow cytogenetics test: chromosomal analysis (MBMC) shall be conducted in the mouse on *m*-pda in accordance with § 798.5395 of this chapter.

(D) If the MBMC conducted pursuant to paragraph (c)(1)(i)(C) of this section is positive, the dominant lethal assay (DL) in mice shall be conducted on *m*-pda in accordance with § 798.5450 of this chapter.

(E) If the DL conducted pursuant to paragraph (c)(1)(i)(D) of this section is positive, heritable translocation (HT) testing in the mouse on *m*-pda shall be conducted in accordance with § 798.5460 of this chapter, if after a public program review, EPA issues a **Federal Register** notice or sends a certified letter to the test sponsor specifying that testing shall be initiated.

(ii) *Reporting requirements.* (A) The final results and final report for the SLRL assay and the MBMC assays shall be submitted to EPA no later than 12 months after the effective date of this section.

(B) The final results and final report of the DL and the mouse specific-locus tests shall be received by EPA no later than 48 months after the effective date of this section.

(C) The final results and the final report of the HT shall be received by EPA no later than 36 months after the effective date on which EPA notifies the test sponsor under paragraph (c)(1)(i)(E) of this section to begin testing.

(D) Interim reports for the SLRL assay, MBMC, DL, HT, and mouse specific-locus studies are required at 6-month intervals beginning 6 months after the effective date of this section or the date

of notification by EPA that testing shall be initiated and ending when the final report is submitted.

(2) *Oncogenicity*—(i) *Required testing.* A 2-year dermal oncogenicity bioassay shall be conducted with *m*-pda in both rats and mice in accordance with § 798.3320 of this chapter if *m*-pda yields positive test results in: the SLRL test conducted pursuant to paragraph (c)(1)(i)(A) of this section, or the MBMC and DL tests conducted pursuant to paragraphs (c)(1)(i)(C) and (c)(1)(i)(D) of this section if, after a public program review, EPA issues a **Federal Register** notice or sends a certified letter to the test sponsor specifying that the testing shall be initiated.

(ii) *Reporting requirements.* (A) The final results and final report for the oncogenicity bioassay shall be submitted to EPA no later than 53 months after the date of EPA's notification of the test sponsor by certified letter or **Federal Register** notice under paragraph (e)(2)(i) of this section that testing shall be initiated.

(B) Interim progress reports for the oncogenicity bioassay shall be submitted every 6 months after notification of the test sponsor by certified letter or **Federal Register** notice that testing shall be initiated and ending when the final report is submitted.

(3) *Neurotoxicity*—(i) *Required testing.* (A) Acute neurotoxicity testing in the neurotoxicity functional observational battery (FOB) in accordance with § 798.6050 of this chapter, and the motor activity test (MAT) in accordance with § 798.6200 of this chapter, shall be conducted simultaneously in the same animals. Each isomer, *o*-, *m*-, and *p*-pda, shall be tested in the FOB and MAT. The test substances shall be administered as a single oral dose in mice. Clinical observations shall be made at a minimum of 1, 4, 24, and 48 hours and at 7 days after dosing.

(B) If neurotoxic effects are observed at 24 hours, or longer, during the testing conducted pursuant to paragraph (c)(3)(i)(A) of this section, then 90-day subchronic neurotoxic FOB, MAT, and neuropathology shall be conducted in accordance with §§ 798.6050, 798.6200, and 798.6400 of this chapter, respectively, for each pda isomer showing such effects. At the end of the subchronic tests, the animals shall be sacrificed and the nervous tissue preserved and examined as described in § 798.6400 of this chapter.

(ii) *Reporting requirements.* (A) The final data and final report for the acute neurotoxicity testing shall be submitted to the EPA no later than 6 months after

the effective date of this section. If triggered, the final report for the subchronic neurotoxicity testing and neuropathological examination shall be submitted to the EPA no later than 15 months after the effective date of this section

(B) [Reserved]

- (e) * * *
- (1) * * *
- (i) * * *

(A) Flowthrough fish acute toxicity tests (LC50) in the rainbow trout (*Salmo gairdneri*) shall be conducted with *o*-, *m*-, and *p*-pda in accordance with § 797.1400 of this chapter.

(B) Acute flow-through studies on the fresh water invertebrate *Gammarus* shall be conducted with *o*-, *m*-, and *p*-pda in accordance with § 795.120 of this chapter.

(C) If the concentration affecting 50 percent of the population (EC50) for any study conducted pursuant to paragraphs (e)(1)(i)(A) and (B) of this section is less than or equal to 100 X Predicted Environmental Concentration (PEC), less than or equal to 1 milligram/liter (mg/L), or less than or equal to 100 mg/L and shows indications of chronicity, chronic toxicity testing shall be conducted pursuant to paragraph (e)(2) of this section. Indications of chronicity shall be the following: for fish or aquatic invertebrates, the ratio of 24 hr/96 hr LC50 is greater than or equal to 2; for daphnids or gammarids, the ratio of 24 hr/48 hr LC50 is greater than or equal to 2.

- (ii) * * *
- (A) * * *
- (B) * * *

(C) An interim report for each acute toxicity test is required 6 months after the effective date of this section.

- (2) * * *
- (i) * * *

(A) A fish early life cycle flow-through test shall be conducted in the most sensitive fish species, either *Pimephales promelas* or *Salmo gairdneri*, with each isomer, *o*-, *m*-, and *p*-pda, demonstrating an LC50, determined by testing of fish pursuant to paragraph (e)(1)(i)(A) of this section, equal to or less than 100 X PEC: less than 1 mg/L; or less than 100 mg/L with indications of chronicity.

Chronicity indicators are defined in paragraph (e)(1)(i)(C) of this section. Testing shall be conducted in accordance with § 797.1600 of this chapter.

(B) An invertebrate life cycle flow-through toxicity test shall be conducted in *Daphnia magna* for each of the *o*-, *m*-, or *p*-pda isomers demonstrating an

EC50, determined by testing of invertebrates pursuant to paragraph (e)(1)(i)(B) of this section, equal to or less than 100 X PEC, or less than 1 mg/L, or less than 100 mg/L with indications of chronicity. Chronicity indicators are defined in paragraph (e)(1)(i)(D) of this section. Testing shall be conducted in accordance with § 797.1330 of this chapter.

- (ii) * * *

(C) Progress reports shall be submitted at 6-month intervals beginning 6 months after the submission of acute toxicity testing which triggers the chronic toxicity test requirement and ending when the final report is submitted.

(f) *Effective date.* The effective date of this section shall be [44 days after the date of publication of the final rule in the Federal Register].

(Information collection requirements have been approved by the Office of Management and Budget under Control Number 2070-0033)

[FR Doc. 88-633 Filed 1-13-88; 8:45 am]

BILLING CODE 6560-50-M

FEDERAL EMERGENCY MANAGEMENT AGENCY

44 CFR Part 7

Nondiscrimination on the Basis of Age in Program or Activities on the Basis of Age in Financial Assistance from FEMA

AGENCY: Federal Emergency Management Agency.

ACTION: Proposed rule.

SUMMARY: These proposed regulations implement provisions of the Age Discrimination Act of 1975, and the general government wide regulation, codified at 45 CFR Part 90.

The Age Discrimination Act of 1975 (hereinafter "the Act") prohibits discrimination on the basis of age in programs or activities receiving federal financial assistance. The Act also contains certain exceptions that permit, under limited circumstances, use of age distinctions or factors other than age that may have a disproportionate effect on the basis of age. The Act applies to persons of all ages.

These regulations are designed to guide the actions of recipients of financial assistance from FEMA. The regulations incorporate the basic standards for determining age discrimination, which are set forth in the general regulations, 45 CFR Part 90. They discuss the responsibilities of FEMA recipients and the investigations,

conciliation and enforcement procedures FEMA will use to ensure compliance with the Act.

DATE: Comments are due March 14, 1988.

FOR FURTHER INFORMATION CONTACT: John R. Curran, Director of Personnel and Equal Opportunity, Room 8110, Federal Emergency Management Agency, 500 C Street SW., Washington, DC 20472, (202) 646-3962 (Voice), (202) 646-4117 (TTD).

ADDRESS: Send comments to: Rules Docket Clerk, Office of General Counsel, Federal Emergency Management Agency, 500 C Street SW., Washington, DC 20472.

SUPPLEMENTARY INFORMATION: The preamble containing supplementary information is divided into the following four sections:

I. Background—provides a brief history of the development of the Act and these regulations.

II. Regulatory procedures—explains compliance with various regulatory requirements.

III. Overview of the Regulations—summarizes the contents of the regulations.

IV. Important questions about the Regulations—answers various questions raised during the development of these regulations.

I. Background

In November 1975, Congress enacted the Age Discrimination Act (42 U.S.C. 6101, *et seq.*) as part of the amendments to the Older Americans Act (Pub. L. 94-135). The Act prohibits discriminating on the basis of age in all programs and activities receiving federal financial assistance.

The Act prohibits recipients of federal financial assistance from taking actions that result in denying or limiting services or otherwise discrimination on basis of age. The Act also contains certain exceptions that permit, under limited circumstances, use of age distinctions or factors other than age that may have a disproportionate effect on the basis of age.

Like other civil rights statutes, the Act applies only to programs or activities in which there is an intermediary (recipient) standing between the federal financial assistance and the ultimate beneficiary of that assistance. The Act does not apply to programs of direct assistance (such as the Social Security program in which federal funds flow directly and unconditionally from the federal government to the individual beneficiary of those funds.)