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40 CFR Part 372

Addition of Certain Chemicals; Toxic
Chemical Release Reporting; Community
Right-to-Know; Final Rule

ENVIRONMENTAL PROTECTION AGENCY**40 CFR Part 372**

[OPPTS-400082B; FRL-4922-2]

RIN 2070-AC47

Addition of Certain Chemicals; Toxic Chemical Release Reporting; Community Right-to-Know

AGENCY: Environmental Protection Agency (EPA).

ACTION: Final rule.

SUMMARY: EPA is adding 286 chemicals and chemical categories, which include 39 chemicals as part of two delineated categories, to the list of toxic chemicals subject to reporting under section 313 of the Emergency Planning and Community Right-to-Know Act of 1986 (EPCRA) and section 6607 of the Pollution Prevention Act of 1990 (PPA). The additions of these chemicals and chemical categories are based on their acute human health effects, carcinogenicity or other chronic human health effects, and/or their adverse effects on the environment. EPA is taking this action pursuant to its authority to add to the list those chemicals and chemical categories that meet the EPCRA section 313(d)(2) criteria for addition to the list of toxic chemicals. EPCRA section 313 reporting for the newly listed chemicals and chemical categories will be required beginning with the 1995 calendar year. As such, the first reports for the added chemicals and chemical categories must be submitted to EPA and States by July 1, 1996.

EFFECTIVE DATE: This rule is effective November 22, 1994.

FOR FURTHER INFORMATION CONTACT: Maria J. Doa, Project Manager, 202-260-9592, for specific information regarding this final rule. For further information on EPCRA section 313, contact the Emergency Planning and Community Right-to-Know Information Hotline, Environmental Protection Agency, Mail Stop 5101, 401 M St., SW., Washington, DC 20460, Toll free: 800-535-0202, TDD: 800-553-7672.

SUPPLEMENTARY INFORMATION:**I. Introduction***A. Statutory Authority*

This rule is issued under section 313(d) of the Emergency Planning and Community Right-to-Know Act of 1986 (EPCRA), 42 U.S.C. 11001 *et seq.* EPCRA is also referred to as Title III of the Superfund Amendments and Reauthorization Act of 1986.

B. Background

Section 313 of EPCRA requires certain facilities manufacturing, processing, or otherwise using listed toxic chemicals to report their environmental releases of such chemicals annually. Beginning with the 1991 reporting year, such facilities also must report pollution prevention and recycling data for such chemicals, pursuant to section 6607 of the Pollution Prevention Act, 42 U.S.C. 13106. Section 313 established an initial list of toxic chemicals that was composed of more than 300 chemicals and 20 chemical categories. Section 313(d) authorizes EPA to add or delete chemicals from the list, and sets forth criteria for these actions. Under section 313(e), any person may petition EPA to add chemicals to or delete chemicals from the list. EPA issued a statement of petition policy and guidance in the **Federal Register** of February 4, 1987 (52 FR 3479), to provide guidance regarding the recommended content and format for petitions. On May 23, 1991 (56 FR 23703), EPA issued guidance regarding the recommended content of petitions to delete individual members of the section 313 metal compound categories.

II. Background

On January 12, 1994 (59 FR 1788), EPA issued a proposal in the **Federal Register** to add 313 chemicals and chemical categories to the list of toxic chemicals under EPCRA section 313 based on their acute human health effects, carcinogenicity or other chronic human health effects, and/or their environmental effects. EPA's decision to add the chemicals and chemical categories in today's rule to the section 313 list is based on a further assessment, in light of public comments of both the relative toxicity of the chemicals--the potency of the chemical's inherent toxicity--and a careful consideration of the type of adverse effect the chemical causes or can reasonably be anticipated to cause. Under section 313(d)(2)(A) (acute human toxicity), the effect must be "significant." Under section 313(d)(2)(B) the effect must either be cancer or teratogenicity, or some other "serious or irreversible" chronic health effect. Under section 313(d)(2)(C) (environmental toxicity) the effect must be "significant" and "of sufficient seriousness in the judgment of the Administrator" to warrant reporting.

The statute does not specify how serious or significant an effect must be in order for a chemical to be listed under any of the criteria. This determination is left to the EPA's discretion and scientific judgment. The Agency recognizes that not every

adverse effect is sufficiently significant or serious to satisfy the criteria. For chemicals with effects that satisfy the criteria, Congress made it clear in section 313 that communities have a right to know about releases of such chemicals. The Agency's goal in implementing section 313 is to ensure that the communities are provided with that release information to allow them to further educate themselves and, if appropriate, take or recommend action.

A brief description of the selection process follows, however, a detailed description of EPA's methodology and rationale for the proposed addition of these chemicals and chemical categories can be found in the proposed rule.

1. *Development of the chemical addition list.* As a starting point for screening candidates for addition to the toxic chemical list under EPCRA section 313, EPA chose to examine the lists of chemicals regulated or identified, as of concern, under various environmental statutes including: Section 112(b) of the Clean Air Act (CAA) as amended in 1990 (Hazardous Air Pollutants); (2) section 602(b) of the CAA (Class II ozone depleting substances); (3) section 307(a) of the Clean Water Act (CWA) (Priority Pollutant List); (4) Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) Active Ingredients, including Special Review, Canceled/Denied or Suspended, and Restricted Use Pesticides; (5) section 302 of EPCRA (Extremely Hazardous Substances); (6) section 102 of the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA); (7) section 3001 of the Resource Conservation and Recovery Act (RCRA) and chemicals listed at 40 CFR 261.33(e) and Appendix VIII; (8) section 1412 of the Safe Drinking Water Act as amended; (9) certain chemicals subject to the Toxics Substance Control Act (Existing Chemicals); and (10) the State of California Safe Drinking Water and Toxic Enforcement Act of 1986 (Proposition 65) (List of Chemicals Known to the State to Cause Reproductive Toxicity); and/or those chemicals designated as possible, probable, or known carcinogens in the Monographs of the International Agency for Research on Cancer (IARC) and the 6th Annual Report on Carcinogens of the National Toxicology Program (NTP), U.S. Department of Health and Human Services (DHHS).

2. *Screening of chemicals.* To prioritize chemicals for possible addition to EPCRA section 313, EPA applied a human health and ecotoxicity screen and a production volume screen, which are described below.

a. *Toxicity screen.* A toxicity screen is a limited review of readily available toxicity data that is used for a preliminary categorization of a chemical during the process of selecting candidates for possible listing under EPCRA section 313. The toxicity screen is used to identify chemicals for further consideration and does not reflect a final determination for listing a chemical under EPCRA section 313. Such a determination can only be made after a hazard assessment is conducted (See Unit II.3. of this preamble). The chemicals identified above were screened for four general effect categories: Acute human health effects, cancer, other chronic human health effects, and ecological effects.

The screening criteria associated with each of the effect areas used in the toxicity screen are discussed in detail in the *Revised Draft Hazard Assessment Guidelines for Listing Chemicals on the Toxic Release Inventory (Draft Hazard Assessment Guidelines)*, (Ref. 11). Based on the results of this screen, the chemicals were preliminarily placed in one of three screening categories defined in the *Draft Hazard Assessment Guidelines*: "high priority;" "medium priority;" or "low priority."

Chemicals that were categorized as "low priority" during the screening process were not considered further as candidates for addition to the EPCRA section 313 list in this rulemaking.

b. *Production volume screen.* EPCRA section 313(f) establishes reporting thresholds of either 25,000 or 10,000 pounds per facility per year related to the amount of a chemical that is manufactured, processed, or otherwise used. EPA anticipates that the addition of chemicals manufactured, imported, processed, or used in quantities less than the EPCRA section 313 activity thresholds would not result in the submission of Toxic Release Inventory (TRI) reports. Thus, EPA elected to focus its attention on chemicals likely to yield reports and also screened potential candidates for the likelihood of meeting the EPCRA section 313 volume thresholds. Chemicals for which there were no data to indicate that the chemical is likely to meet or exceed the EPCRA section 313 volume thresholds were not considered further as possible candidates for addition to the section 313 list at this time.

3. *Hazard evaluation.* After completing the screening phase, EPA conducted a thorough hazard assessment for each of the addition candidates that resulted from the above analyses and determined based on the weight-of-the-evidence if there was sufficient evidence to establish that the

candidate chemical met the statutory criteria for addition to EPCRA section 313. To make this determination, EPA senior scientists reviewed readily available toxicity information on each chemical for each of the following effect areas: acute human health effects; cancer; other chronic human effects; and environmental effects. In addition, EPA reviewed, where appropriate, information on the environmental fate of the chemical.

The hazard assessment was conducted in accordance with relevant EPA guidelines for each adverse human health or environmental effect (e.g., the appropriate guidelines for hazard evaluation of chemical carcinogens and for the type of evidence required to substantiate a determination of carcinogenicity are the *Assessment Guidelines for Carcinogen Risk* (Ref. 4)). During this assessment the number, severity, and significance of the effects induced by the chemical, the dose level causing the effect, and the quality and quantity of the available data, including the nature of the data (e.g., human epidemiological, laboratory animal, field or workplace studies) and confidence level in the existing data base, were all considered. Where a careful review of the scientific data for a particular chemical results in a high level of confidence that the chemical causes an adverse effect at relatively low dose levels, EPA believes that this evidence is sufficient for listing the chemical under section 313. EPA also believes that where a review of the scientific data indicates that the chemical will cause various adverse effects at moderate dose levels, the total weight-of-the-evidence indicates that there is sufficient evidence for listing the chemical under EPCRA section 313. EPA believes that both types of chemicals described above exhibit moderately high to high toxicity based on a hazard assessment.

EPA also conducted an analysis of exposure for each chemical or chemical category proposed for listing under EPCRA section 313(d)(2)(A) (i.e., based on adverse acute human health effects), and, where appropriate, under section 313(d)(2)(C) (i.e., based on adverse ecological effects). For chemicals listed under EPCRA section 313(d)(2)(A), this analysis included estimated concentrations of the chemical at or beyond the facility site boundary through the use of estimated releases and modelling techniques. EPA did not conduct an analysis of exposure for the chemicals proposed for listing under section 313(d)(2)(B) because these chemicals exhibit moderately high to high toxicity based on a hazard

assessment (see Unit IV.B. for a discussion of the use of exposure). As discussed more thoroughly in Unit IV.B. of this preamble, EPA does not believe that it is appropriate to factor exposure into the listing decisions for the chemicals being listed pursuant to section 313(d)(2)(B) in this rulemaking.

Following a review and analysis of the information available about each chemical in this final rule (including information provided through public comment) by senior Agency scientists, the Agency concludes that for each of the chemicals listed one or more of the EPCRA section 313 listing criteria are met. Moreover, the adverse effects associated with each of the chemicals being listed today are serious and significant. In some cases the effects are extreme, such as cancer or death. In others, the effects are serious and lasting, including, for example, impairment of a fetus' or an offspring's physical development, neurological effects inhibiting motor abilities or mental processes or impairing the ability to reproduce, or the sustainability of a fragile ecosystem such as an estuary. For a number of chemicals in the final rule, there is more than one adverse effect.

It is important to understand that although an adverse effect is known or can be reasonably anticipated to be caused by a chemical on the section 313 list, a release of a chemical into a community does not necessarily mean that the effect will occur. Exposure and dose are also important factors in determining whether an adverse effect occurs and how serious the manifestation will be. The listing of a chemical on the section 313 list does not mean that a particular community will experience these adverse effects. Instead the purpose for listing a chemical is to ensure that the public gets information about releases of such chemicals. Thus, EPA believes that for chemicals that typically do not affect solely one or two species but rather affect changes across a whole ecosystem and for which there is well-documented evidence supporting the adverse effects, that their addition to the EPCRA section 313 list is warranted even though the severity of the adverse effects that they induce will be dependent upon site-specific characteristics. Once EPA makes release data available through TRI, the community may then make its own determination on the importance of these releases (and their potential adverse effects).

The expansion of the EPCRA section 313 toxic chemical list is the first phase of the expansion of the TRI program. EPA plans to issue a proposed rule in

early 1995 expanding the scope of industry sectors that would be subject to EPCRA section 313. EPA's initial analysis for this effort is focused on industrial sectors which have activities related to manufacturing that result in significant releases of chemicals listed on EPCRA section 313. EPA is also considering further expanding right-to-know by investigating the feasibility of adding data on exposure to and use of chemicals at TRI facilities. The Agency believes that the collection of this type of data would provide a greater understanding of risk reduction and pollution prevention opportunities.

In conjunction with these expansion activities, the Agency is also considering situations where data of lesser value can be removed from the TRI system. Elsewhere in this issue of the **Federal Register**, EPA is promulgating a rule establishing an alternate threshold for facilities with low annual reportable amounts of listed toxic chemicals. This alternate threshold will provide considerable relief for facilities which generate "small" amounts of EPCRA section 313 chemicals in reportable amounts. This relief will offset the increased burden that this expansion rule may impose. The alternate threshold for manufacture, or process, or otherwise use for each of the chemicals meeting the facility category will be an amount greater than one million pounds per year. If a facility meets the alternate threshold criteria,

that facility will not be required to file a complete TRI report (Form R), but will be required to submit an annual certification statement for each chemical meeting these conditions for the reporting year for which these conditions were met and maintain records supporting calculations made to determine these conditions. EPA estimates that this alternate threshold provides the option to convert approximately 20,100 Form R reports to certification statements.

III. Summary of Final Rule

In this action, EPA is adding 286 chemicals and chemical categories, which includes 39 chemicals as part of two delineated categories, to the EPCRA section 313 list. EPA finds that each of these chemicals and chemical categories meets one or more of the EPCRA section 313(d)(2) criteria. Additionally, EPA believes that each of these chemicals can reasonably be anticipated to be manufactured or imported in quantities of at least 10,000 pounds (the EPCRA section 313 otherwise use reporting threshold) by at least one facility. Therefore, the Agency believes that the listing of these chemicals can reasonably be anticipated to generate EPCRA section 313 reports and that adding these chemicals to the toxic chemical list is appropriate.

The proposed rule and record supporting the rulemaking contain information on EPA's review of these

chemicals, including the toxicity evaluation. This background information will not be repeated here in the final rule. However, to the extent that comments were received on these issues, those comments are addressed in this document. In addition to general comment and comment addressing a broad number of chemicals, EPA received specific technical comments on 110 of the chemicals and chemical categories. Detailed responses to comments are contained in *Response to Comments Received on the January 12, 1994 Proposed Rule to Expand the EPCRA Section 313 List (Response to Comment Document, Ref. 14)*. Summaries of responses to comments on selected chemicals appear in units IV.F. and IV.G. of this preamble. Table 1 lists the chemicals that EPA has determined meet the statutory criteria of EPCRA section 313(d)(2) and are therefore being added to the toxic chemical list. Each of the chemicals and chemical categories listed below were found to meet the statutory criteria described in EPCRA section 313(d)(2)(A)-(C). This means that the Agency has made a finding that the chemical is known to cause an effect, or is reasonably anticipated to do so. It does not necessarily mean that the chemical is known to cause a given effect. The specific criterion or criteria that the chemical meets are also listed in Table 1 below.

TABLE 1.—CHEMICALS BEING ADDED TO THE EPCRA SECTION 313 LIST

| Chemical Name | CAS No. | Section 313(d)(2)(A) | Section 313(d)(2)(B) | Section 313(d)(2)(C) |
|-------------------------------------------------------------------------------------------------------|-------------|----------------------|----------------------|----------------------|
| Abamectin (Avermectin B1) | 071751-41-2 | | X | X |
| Acephate (Acetylphosphoramidothioic acid O,S-dimethyl ester) | 030560-19-1 | | X | |
| Acifluorfen sodium salt (5-(2-Chloro-4-(trifluoromethyl)phenoxy)-2-nitro-benzoic acid, sodium salt) | 062476-59-9 | | X | |
| Alachlor | 015972-60-8 | | X | |
| Aldicarb | 000116-06-3 | | | X |
| d-trans-Allethrin [d-trans-Chrysanthemic acid of d-allethrone] | 028057-48-9 | | X | |
| Allylamine | 000107-11-9 | | X | |
| Aluminum phosphide | 020859-73-8 | X | | |
| Ametryn (N-Ethyl-N'-(1-methylethyl)-6-(methylthio)-1,3,5,-triazine- 2,4 diamine) | 000834-12-8 | | X | X |
| Amitraz | 033089-61-1 | | X | |
| Anilazine (4,6-Dichloro-N-(2-chlorophenyl)-1,3,5-triazin-2-amine) | 000101-05-3 | | X | X |
| Atrazine (6-Chloro-N-ethyl-N'-(1-methylethyl)-1,3,5,-triazine-2,4-diamine) | 001912-24-9 | | X | |
| Bendiocarb (2,2-Dimethyl-1,3-benzodioxol-4-ol methylcarbamate) | 022781-23-3 | | X | X |
| Benfluralin (N-Butyl-N-ethyl-2,6-dinitro-4-(trifluoromethyl) benzenamine) | 001861-40-1 | | X | |
| Benomyl | 017804-35-2 | | X | |
| Bifenthrin | 082657-04-3 | | X | X |
| Bis(tributyltin) oxide | 000056-35-9 | | X | X |
| Boron trichloride | 010294-34-5 | X | | |
| Boron trifluoride | 007637-07-2 | | X | |
| Bromacil (5-Bromo-6-methyl-3-(1-methylpropyl)-2,4(1H,3H)-pyrimidinedione) | 000314-40-9 | | X | |
| Bromacil lithium salt (2,4(1H,3H)-Pyrimidinedione, 5-bromo-6-methyl-3 (1-methylpropyl), lithium salt) | 053404-19-6 | | X | |
| Bromine | 007726-95-6 | | X | |
| 1-Bromo-1-(bromomethyl)-1,3-propanedicarbonitrile | 035691-65-7 | | X | |
| 2-Bromo-2-nitropropane-1,3-diol (Bronopol) | 000052-51-7 | | X | |
| Bromoxynil (3,5-Dibromo-4-hydroxybenzotrile) | 001689-84-5 | | X | |
| Bromoxynil octanoate (Octanoic acid, 2,6-dibromo-4-cyanophenyl ester) | 001689-99-2 | | X | |

TABLE 1.—CHEMICALS BEING ADDED TO THE EPCRA SECTION 313 LIST—Continued

| Chemical Name | CAS No. | Section 313(d)(2)(A) | Section 313(d)(2)(B) | Section 313(d)(2)(C) |
|----------------------------------------------------------------------------------------------------------------------------------|-------------|----------------------|----------------------|----------------------|
| Brucine | | | | |
| C.I. Acid Red 114 | 000357-57-3 | X | | |
| C.I. Direct Blue 218 | 006459-94-5 | | X | |
| Carbofuran | 028407-37-6 | | X | |
| Carboxin (5,6-Dihydro-2-methyl-N-phenyl-1,4-oxathiin-3-carboxamide) | 001563-66-2 | | | X |
| Chinomethionat (6-Methyl-1,3-dithiolo[4,5-b]quinoxalin-2-one) | 005234-68-4 | | X | |
| Chlorendic acid | 002439-01-2 | | X | |
| Chlorimuron ethyl (Ethyl-2-[[[(4-chloro-6-methoxyimidin-2-yl)-carbonyl]-amino]sulfonyl]benzoate) | 000115-28-6 | | X | |
| 1-(3-Chloroallyl)-3,5,7-triaza-1-azoniaadamantane chloride | 090982-32-4 | | X | |
| p-Chloroaniline | 004080-31-3 | | X | |
| 3-Chloro-2-methyl-1-propene | 000106-47-8 | | X | |
| p-Chlorophenyl isocyanate | 000563-47-3 | | X | |
| Chloropicrin | 000104-12-1 | X | | |
| 3-Chloropropionitrile | 000076-06-2 | | | X |
| p-Chloro-o-toluidine | 000542-76-7 | X | | |
| 2-Chloro-1,1,1-trifluoroethane (HCFC-133a) | 000095-69-2 | | X | |
| Chlorotrifluoromethane (CFC-13) | 000075-88-7 | | X | X |
| 3-Chloro-1,1,1-trifluoropropane (HCFC-253fb) | 000075-72-9 | | X | X |
| Chlorpyrifos methyl (O,O-Dimethyl-O-(3,5,6-trichloro-2-pyridyl)phosphorothioate) | 000460-35-5 | | X | X |
| Chlorsulfuron (2-Chloro-N-[[[(4-methoxy-6-methyl-1,3,5-triazin-2-yl)amino]carbonyl]benzenesulfonamide) | 005598-13-0 | | X | X |
| Crotonaldehyde | 064902-72-3 | | X | |
| Cyanazine | 004170-30-3 | | X | |
| Cycloate | 021725-46-2 | | X | |
| Cyclohexanol | 001134-23-2 | | X | |
| Cyfluthrin (3-(2,2-Dichloroethenyl)-2,2-dimethylcyclopropanecarboxylic acid, cyano(4-fluoro-3-phenoxyphenyl)methylester) | 000108-93-0 | | X | |
| Cyhalothrin (3-(2-Chloro-3,3,3-trifluoro-1-propenyl)-2,2-Dimethylcyclopropanecarboxylic acid cyano(3-phenoxyphenyl)methyl ester) | 068359-37-5 | | X | X |
| Dazomet (Tetrahydro-3,5-dimethyl-2H-1,3,5-thiadiazine-2-thione) | 068085-85-8 | | X | |
| Dazomet sodium salt (2H-1,3,5-Thiadiazine-2-thione, tetrahydro-3,5-dimethyl-, ion(1-), sodium) | 000533-74-4 | | X | |
| 2,4-DB | 053404-60-7 | | X | |
| 2,4-D butoxyethyl ester | 000094-82-6 | | X | |
| 2,4-D butyl ester | 001929-73-3 | | X | |
| 2,4-D chlorocrotyl ester | 000094-80-4 | | X | |
| Desmedipham | 002971-38-2 | | X | |
| 2,4-D 2-ethylhexyl ester | 013684-56-5 | | X | |
| 2,4-D 2-ethyl-4-methylpentyl ester | 001928-43-4 | | X | |
| Diazinon | 053404-37-8 | | X | |
| 2,2-Dibromo-3-nitrilopropionamide | 000333-41-5 | | X | X |
| Dicamba (3,6-Dichloro-2-methoxybenzoic acid) | 010222-01-2 | | X | |
| Dichloran (2,6-Dichloro-4-nitroaniline) | 001918-00-9 | | X | |
| 3,3'-Dichlorobenzidine dihydrochloride | 000099-30-9 | | X | |
| 3,3'-Dichlorobenzidine sulfate | 000612-83-9 | | X | |
| trans-1,4-Dichloro-2-butene | 064969-34-2 | | X | |
| 1,2-Dichloro-1,1-difluoroethane (HCFC-132b) | 000110-57-6 | X | | |
| Dichlorofluoromethane (HCFC-21) | 001649-08-7 | | X | X |
| Dichloropentafluoropropane | 000075-43-4 | | X | X |
| 1,3-Dichloro-1,1,2,3,3-pentafluoropropane (HCFC-225ea) | 127564-92-5 | | X | X |
| 2,2-Dichloro-1,1,1,3,3-pentafluoropropane (HCFC-225aa) | 136013-79-1 | | X | X |
| 1,1-Dichloro-1,2,3,3,3-pentafluoropropane (HCFC-225eb) | 128903-21-9 | | X | X |
| 1,1-Dichloro-1,2,2,3,3-pentafluoropropane (HCFC-225cc) | 111512-56-2 | | X | X |
| 1,3-Dichloro-1,1,2,2,3-pentafluoropropane (HCFC-225cb) | 013474-88-9 | | X | X |
| 1,2-Dichloro-1,1,3,3,3-pentafluoropropane (HCFC-225da) | 000507-55-1 | | X | X |
| 3,3-Dichloro-1,1,1,2,2-pentafluoropropane (HCFC-225ca) | 000431-86-7 | | X | X |
| 2,3-Dichloro-1,1,1,2,3-pentafluoropropane (HCFC-225ba) | 000422-56-0 | | X | X |
| 1,2-Dichloro-1,1,2,3,3-pentafluoropropane (HCFC-225bb) | 000422-48-0 | | X | X |
| Dichlorophene (2,2'-Methylenebis(4-chlorophenol)) | 000422-44-6 | | X | X |
| trans-1,3-Dichloropropene | 000097-23-4 | | X | X |
| Diclofop methyl (2-[4-(2,4-Dichlorophenoxy) phenoxy]propanoic acid, methyl ester) | 010061-02-6 | | X | |
| Dicyclopentadiene | 051338-27-3 | | X | |
| Diethyl ethyl | 000077-73-6 | | X | |
| Diffubenzuron | 038727-55-8 | | X | |
| Diglycidyl resorcinol ether | 035367-38-5 | | X | X |
| Diisocyanates, consisting of: | 000101-90-6 | | X | |
| 1,3-Bis(methylisocyanate) cyclohexane | NA | | X | |
| 1,4-Bis(methylisocyanate) cyclohexane | 038661-72-2 | | | |
| 1,4-Cyclohexane diisocyanate | 010347-54-3 | | | |
| Diethyldiisocyanatobenzene | 002556-36-7 | | | |
| | 134190-37-7 | | | |

TABLE 1.—CHEMICALS BEING ADDED TO THE EPCRA SECTION 313 LIST—Continued

| Chemical Name | CAS No. | Section 313(d)(2)(A) | Section 313(d)(2)(B) | Section 313(d)(2)(C) |
|-------------------------------------------------------------------------------------------------------|-------------|----------------------|----------------------|----------------------|
| 4,4'-Diisocyanatodiphenyl ether | 004128-73-8 | | | |
| 2,4'-Diisocyanatodiphenyl sulfide | 075790-87-3 | | | |
| 3,3'-Dimethoxybenzidine-4,4'-diisocyanate | 000091-93-0 | | | |
| 3,3'-Dimethyl-4,4'-diphenylene diisocyanate | 000091-97-4 | | | |
| 3,3'-Dimethyl diphenylmethane-4,4'-diisocyanate | 000139-25-3 | | | |
| Hexamethylene-1,6-diisocyanate | 000822-06-0 | | | |
| Isophorone diisocyanate | 004098-71-0 | | | |
| Methylenebis(phenyl isocyanate) | 000101-68-8 | | | |
| 4-Methyldiphenylmethane-3,4-diisocyanate | 075790-84-0 | | | |
| 1,1-Methylene bis(4-isocyanatocyclohexane) | 005124-30-1 | | | |
| 1,5-Naphthalene diisocyanate | 003173-72-6 | | | |
| 1,3-Phenylene diisocyanate | 000123-61-5 | | | |
| 1,4-Phenylene diisocyanate | 000104-49-4 | | | |
| Polymeric diphenylmethane diisocyanate | 009016-87-9 | | | |
| 2,2,4-Trimethylhexamethylene diisocyanate | 016938-22-0 | | | |
| 2,4,4-Trimethylhexamethylene diisocyanate 015646-96-5 | | | | |
| Dimethipin (2,3,-Dihydro-5,6-dimethyl-1,4-dithiin 1,1,4,4-tetraoxide) | 055290-64-7 | | X | |
| Dimethoate | 000060-51-5 | | X | |
| 3,3'-Dimethoxybenzidine dihydrochloride (o-Dianisidine dihydrochloride) | 020325-40-0 | | X | |
| 3,3'-Dimethoxybenzidine hydrochloride (o-Dianisidine hydrochloride) | 111984-09-9 | | X | |
| Dimethylamine | 000124-40-3 | | X | |
| Dimethylamine dicamba | 002300-66-5 | | X | |
| 3,3'-Dimethylbenzidine dihydrochloride (o-Tolidine dihydrochloride) | 000612-82-8 | | X | |
| 3,3'-Dimethylbenzidine dihydrofluoride (o-Tolidine dihydrofluoride) | 041766-75-0 | | X | |
| Dimethyl chlorothiophosphate | 002524-03-0 | | X | |
| Dimethyldichlorosilane | 000075-78-5 | X | | |
| N,N-Dimethylformamide | 000068-12-2 | | X | |
| 2,6-Dimethylphenol | 000576-26-1 | | X | |
| Dinitrobutyl phenol (Dinoseb) | 000088-85-7 | | X | X |
| Dinocap | 039300-45-3 | | X | X |
| Diphenamid | 000957-51-7 | | X | |
| Diphenylamine | 000122-39-4 | | X | |
| Dipotassium endothall (7-Oxabicyclo(2.2.1)heptane-2,3-dicarboxylic acid, dipotassium salt) | 002164-07-0 | | X | |
| Dipropyl isocinchomeronate | 000136-45-8 | | X | |
| Disodium cyanodithioimidocarbonate | 000138-93-2 | | X | |
| 2,4-D isopropyl ester | 000094-11-1 | | X | |
| 2,4-Dithiobiuret | 000541-53-7 | | X | |
| Diuron | 000330-54-1 | | X | X |
| Dodine (Dodecylguanidine monoacetate) | 002439-10-3 | | | X |
| 2,4-DP (Dichlorprop) | 000120-36-5 | | X | |
| 2,4-D propylene glycol butyl ether ester | 001320-18-9 | | X | |
| 2,4-D sodium salt | 002702-72-9 | | X | |
| Ethoprop (Phosphorodithioic acid O-ethyl S,S-dipropyl ester) | 013194-48-4 | | X | X |
| Ethyl dipropylthiocarbamate (EPTC) | 000759-94-4 | | X | X |
| Famphur | 000052-85-7 | | X | X |
| Fenarimol (.alpha.-(2-Chlorophenyl)-.alpha.-4-chlorophenyl)-5-pyrimidinemethanol) | 060168-88-9 | | X | |
| Fenbutatin oxide (hexakis(2-methyl-2-phenylpropyl)distanoxane) | 013356-08-6 | | X | X |
| Fenoxaprop ethyl (2-(4-((6-Chloro-2-benzoxazolylen)oxy)phenoxy)propanoic acid,ethyl ester) | 066441-23-4 | | X | X |
| Fenoxycarb (2-(4-Phenoxyphenoxy)ethyl)carbamic acid ethyl ester) | 072490-01-8 | | X | |
| Fenpropathrin (2,2,3,3-Tetramethylcyclopropane carboxylic acid cyano(3-phenoxyphenyl)methyl ester) | 039515-41-8 | | X | X |
| Fenthion (O,O-Dimethyl O-[3-methyl-4-(methylthio) phenyl] ester, phosphorothioic acid) | 000055-38-9 | | X | X |
| Fenvalerate (4-Chloro-alpha-(1-methylethyl)benzeneacetic acid cyano(3-phenoxyphenyl)methyl ester) | 051630-58-1 | | X | X |
| Ferbam (Tris(dimethylcarbarnodithioato-S,S')iron) | 014484-64-1 | | X | X |
| Fluazifop butyl (2-[4-[[5-(Trifluoromethyl)-2-pyridinyl]oxy]-phenoxy]propanoic acid, butyl ester) | 069806-50-4 | | X | |
| Fluorine | 007782-41-4 | X | | |
| Fluorouracil (5-Fluorouracil) | 000051-21-8 | | X | |
| Fluvalinate (N-[2-Chloro-4-(trifluoromethyl)phenyl]-DL-valine(+)-cyano (3-phenoxyphenyl)methyl ester) | 069409-94-5 | | X | X |
| Folpet | 000133-07-3 | | X | X |
| Fomesafen (5-(2-Chloro-4-(trifluoromethyl)phenoxy)-N methylsulfonyl)-2-nitrobenzamide) | 072178-02-0 | | X | |
| alpha-Hexachlorocyclohexane | 000319-84-6 | | X | X |
| n-Hexane | 000110-54-3 | | X | |
| Hexazinone | 051235-04-2 | | X | X |

TABLE 1.—CHEMICALS BEING ADDED TO THE EPCRA SECTION 313 LIST—Continued

| Chemical Name | CAS No. | Section 313(d)(2)(A) | Section 313(d)(2)(B) | Section 313(d)(2)(C) |
|---------------------------------------------------------------------------------------------------------------------------------------------------------------|-------------|----------------------|----------------------|----------------------|
| Hydramethylnon (Tetrahydro-5,5-di-methyl-2(1H)-(trifluoromethyl)phenyl]-1-[2-[4-(trifluoromethyl)2propenyldiene]hydrazone) pyrimidinone[3-[4-phenyl]ethenyl]- | 067485-29-4 | | X | X |
| Imazail (1-[2-(2,4-Dichlorophenyl)-2-(2-propenyloxy)ethyl]-1H-imidazole) | 035554-44-0 | | X | |
| 3-Iodo-2-propynyl butylcarbamate | 055406-53-6 | | X | |
| Iron pentacarbonyl | 013463-40-6 | X | | |
| Isodrin | 000465-73-6 | | | X |
| Isofenphos (2-[[Ethoxyl[(1-methylethyl)amino]phosphinothioyl]oxy] benzoic acid 1-methylethyl ester) | 025311-71-1 | | X | X |
| Lactofen (5-(2-Chloro-4-(trifluoromethyl)phenoxy)-2-nitro-2-ethoxy-1-methyl-2-oxoethyl ester) | 077501-63-4 | | X | |
| Linuron | 000330-55-2 | | X | |
| Lithium carbonate | 000554-13-2 | | X | |
| Malathion | 000121-75-5 | | X | X |
| Mecoprop | 000093-65-2 | | X | |
| 2-Mercaptobenzothiazole (MBT) | 000149-30-4 | | | X |
| Merphos | 000150-50-5 | | X | |
| Metham sodium (Sodium methylthiocarbamate) | 000137-42-8 | | X | |
| Methazole (2-(3,4-Dichlorophenyl)-4-methyl-1,2,4-oxadiazolidine-3,5-dione) | 020354-26-1 | | | X |
| Methiocarb | 002032-65-7 | | X | |
| Methoxone ((4-Chloro-2-methylphenoxy) acetic acid) (MCPA) | 000094-74-6 | | X | |
| Methoxone sodium salt ((4-Chloro-2-methylphenoxy) acetate sodium salt) | 003653-48-3 | | X | |
| Methyl isothiocyanate | 00556-61-6 | | | X |
| 2-Methylacetonitrile | 000075-86-5 | | X | |
| N-Methylolacrylamide | 000924-42-5 | | X | |
| Methyl parathion | 000298-00-0 | | X | X |
| N-Methyl-2-pyrrolidone | 000872-50-4 | | X | |
| Methyltrichlorosilane | 000075-79-6 | X | | |
| Metiram | 009006-42-2 | | X | |
| Metribuzin | 021087-64-5 | | X | |
| Mevinphos | 007786-34-7 | | | X |
| Molinate (1H-Azepine-1 carbothioic acid, hexahydro-S-ethyl ester) | 002212-67-1 | | X | |
| Monuron | 000150-68-5 | | | X |
| Myclobutanil (.alpha.-Butyl-.alpha.-(4-chlorophenyl)-1H-1,2,4-triazole-1-propanenitrile) | 088671-89-0 | | X | |
| Nabam | 000142-59-6 | | X | |
| Naled | 000300-76-5 | | X | X |
| Nicotine and salts | NA | | X | |
| Nitrapyrin (2-Chloro-6-(trichloromethyl)pyridine) | 001929-82-4 | | X | |
| Nitrate compounds (water dissociable) | NA | | X | |
| p-Nitroaniline | 000100-01-6 | | X | |
| Norflurazon (4-Chloro-5-(methylamino)-2-[3-(trifluoromethyl)phenyl]-3(2H)-pyridazinone) | 027314-13-2 | | X | |
| Oryzalin (4-(Dipropylamino)-3,5-dinitrobenzenesulfonamide) | 019044-88-3 | | X | |
| Oxydemeton methyl (S-(2-(Ethylsulfinyl)ethyl) O,O-dimethyl ester phosphorothioic acid) | 000301-12-2 | | X | |
| Oxydiazon (3-[2,4-Dichloro-5-(1-methylethoxy)phenyl]-5-(1,1-dimethylethyl)-1,3,4-oxadiazol-2(3H)-one) | 019666-30-9 | | X | |
| Oxyfluorfen | 042874-03-3 | | X | X |
| Ozone | 010028-15-6 | | X | X |
| Paraquat dichloride | 001910-42-5 | | X | |
| Pebulate (Butylethylcarbamothioic acid S-propyl ester) | 001114-71-2 | | X | |
| Pendimethalin (N-(1-Ethylpropyl)-3,4-dimethyl-2,6-dinitrobenzenamine) | 040487-42-1 | | X | |
| Pentobarbital sodium | 000057-33-0 | | X | |
| Perchloromethyl mercaptan | 000594-42-3 | X | | |
| Permethrin (3-(2,2-Dichloroethenyl)-2,2-dimethylcyclopropanecarboxylic acid, (3-phenoxyphenyl)methyl ester) | 052645-53-1 | | X | X |
| Phenanthrene | 000085-01-8 | | | X |
| Phenothrin (2,2-Dimethyl-3-(2-methyl-1-propenyl) cyclopropanecarboxylic acid (3-phenoxyphenyl)methyl ester) | 026002-80-2 | | X | X |
| 1,2-Phenylenediamine | 000095-54-5 | | X | |
| 1,3-Phenylenediamine | 000108-45-2 | | X | |
| 1,2-Phenylenediamine dihydrochloride | 000615-28-1 | | X | |
| 1,4-Phenylenediamine dihydrochloride | 000624-18-0 | | | X |
| Phenytol | 000057-41-0 | | X | |
| Phosphine | 007803-51-2 | X | | |
| Picloram | 001918-02-1 | | X | |
| Piperonyl butoxide | 000051-03-6 | | X | X |
| Pirimiphos methyl (O-(2-(Diethylamino)-6-methyl-4-pyrimidinyl)-O,O-dimethyl phosphorothioate) | 029232-93-7 | | X | |
| Polychlorinated alkanes | NA | | X | X |

TABLE 1.—CHEMICALS BEING ADDED TO THE EPCRA SECTION 313 LIST—Continued

| Chemical Name | CAS No. | Section 313(d)(2)(A) | Section 313(d)(2)(B) | Section 313(d)(2)(C) |
|------------------------------------------------------------------------------------------------------------------------------------------------|-------------|----------------------|----------------------|----------------------|
| Polycyclic aromatic compounds (PACs) consisting of: | NA | | X | |
| Benz(a)anthracene | 000056-55-3 | | | |
| Benzo(a)phenanthrene | 000218-01-9 | | | |
| Benzo(a)pyrene | 000050-32-8 | | | |
| Benzo(b)fluoranthene | 000205-99-2 | | | |
| Benzo(j)fluoranthene | 000205-82-3 | | | |
| Benzo(k)fluoranthene | 000207-08-9 | | | |
| Benzo(rst)pentaphene | 000189-55-9 | | | |
| Dibenz(a,h)acridine | 000226-36-8 | | | |
| Dibenz(a,i)acridine | 000224-42-0 | | | |
| Dibenzo(a,h)anthracene | 000053-70-3 | | | |
| Dibenzo(a,e)fluoranthene | 005385-75-1 | | | |
| Dibenzo(a,e)pyrene | 000192-65-4 | | | |
| Dibenzo(a,h)pyrene | 000189-64-0 | | | |
| Dibenzo(a,l)pyrene | 000191-30-0 | | | |
| 7H-Dibenzo(c,g)carbazole | 00194-59-2 | | | |
| 7,12-Dimethyl benz(a)anthracene | 000057-97-6 | | | |
| Indeno[1,2,3-cd]pyrene | 000193-39-5 | | | |
| 5-Methylchrysene | 003697-24-3 | | | |
| 1-Nitropyrene | 005522-43-0 | | | |
| Potassium bromate | 007758-01-2 | | X | |
| Potassium dimethyldithiocarbamate | 000128-03-0 | | X | |
| Potassium N-methyldithiocarbamate | 000137-41-7 | | X | |
| Profenofos (O-(4-Bromo-2-chlorophenyl)-O-ethyl-S-propyl phosphorothioate) | 041198-08-7 | | X | |
| Prometryn (N,N'-Bis(1-methylethyl)-6-methylthio-1,3,5-triazine-2,4-diamine) | 007287-19-6 | | X | |
| Propachlor (2-Chloro-N-(1-methylethyl)-N-phenylacetamide) | 001918-16-7 | | X | |
| Propanil (N-(3,4-Dichlorophenyl)propanamide) | 000709-98-8 | | X | |
| Propargite | 002312-35-8 | | X | X |
| Propargyl alcohol | 000107-19-7 | | X | |
| Propetamphos (3-[(Ethylamino)methoxyphosphinothioyl]oxy)-2-butenic acid, 1-methylethyl ester) | 031218-83-4 | | X | |
| Propiconazole (1-[2-(2,4-Dichlorophenyl)-4-propyl-1,3-dioxolan-2-yl]-methyl-1H-1,2,4,-triazole) | 060207-90-1 | | X | |
| Quizalofop-ethyl (2-[4-[(6-Chloro-2-quinoxalinyloxy]phenoxy] propanoic acid ethyl ester) | 076578-14-8 | | X | |
| Resmethrin ([5-(Phenylmethyl)-3-furanyl]methyl 2,2-dimethyl-3-(2-methyl-1-propenyl)cyclopropanecarboxylate]) | 010453-86-8 | | X | X |
| Sethoxydim (2-[1-(Ethoxyimino)butyl]-5-[2-(ethylthio)propyl]-3-hydroxy-2-cyclohexen-1-one) | 074051-80-2 | | X | |
| Simazine | 000122-34-9 | | X | |
| Sodium azide | 026628-22-8 | | X | |
| Sodium dicamba (3,6-Dichloro-2-methoxybenzoic acid, sodium salt) | 001982-69-0 | | X | |
| Sodium dimethyldithiocarbamate | 000128-04-1 | | X | |
| Sodium fluoroacetate | 000062-74-8 | | X | X |
| Sodium nitrite | 007632-00-0 | | X | |
| Sodium pentachlorophenate | 000131-52-2 | | X | X |
| Sodium o-phenylphenoxide | 000132-27-4 | | X | |
| Strychnine and salts | NA | X | | |
| Sulfuryl fluoride (Vikane) | 002699-79-8 | | X | |
| Sulprofos (O-Ethyl O-[4-(methylthio)phenyl]phosphorodithioic acid S propyl ester) | 035400-43-2 | | X | X |
| Tebuthiuron (N-[5-(1,1-Dimethylethyl)-1,3,4-thiadiazol-2-yl]- N,N'-dimethylurea) | 034014-18-1 | | X | |
| Temephos | 003383-96-8 | | X | |
| Terbacil (5-Chloro-3-(1,1-dimethylethyl)-6-methyl- 2,4 (1H,3H)-pyrimidinedione) | 005902-51-2 | | X | |
| 1,1,1,2-Tetrachloro-2-fluoroethane (HCFC-121a) | 000354-11-0 | | X | X |
| 1,1,2,2-Tetrachloro-1-fluoroethane (HCFC-121) | 000354-14-3 | | X | X |
| Tetracycline hydrochloride | 000064-75-5 | | X | |
| Tetramethrin (2,2-Dimethyl-3-(2-methyl-1-propenyl) cyclopropanecarboxylic acid (1,3,4,5,6,7-hexahydro-1,3-dioxo-2H-isoindol-2-yl)methyl ester) | 007696-12-0 | | X | X |
| Thiabendazole (2-(4-Thiazoly)-1H-benzimidazole) | 000148-79-8 | | X | X |
| Thiobencarb (Carbamic acid, diethylthio-, S-(p-chlorobenzyl)) | 028249-77-6 | | X | X |
| Thiodicarb | 059669-26-0 | | X | X |
| Thiophanate ethyl ([1,2-Phenylenebis(iminocarbonothioyl)] biscarbamic acid diethyl ester) | 023564-06-9 | | X | |
| Thiophanate-methyl | 023564-05-8 | | X | |
| Thiosemicarbazide | 000079-19-6 | X | | |
| Triadimefon (1-(4-Chlorophenoxy)-3,3-dimethyl-1-(1H-1,2,4-triazol-1-yl)-2-butanone) | 043121-43-3 | | X | |
| Triallate | 002303-17-5 | | X | |
| Tribenuron methyl (2-(4-Methoxy-6-methyl-1,3,5-triazin-2-yl)-methylamino)carbonyl)amino)sulfonyl-, methyl ester) | 101200-48-0 | | X | |
| Tributyltin fluoride | 001983-10-4 | | | X |

TABLE 1.—CHEMICALS BEING ADDED TO THE EPCRA SECTION 313 LIST—Continued

| Chemical Name | CAS No. | Section 313(d)(2)(A) | Section 313(d)(2)(B) | Section 313(d)(2)(C) |
|---------------------------------------------------------------------------------|-------------|----------------------|----------------------|----------------------|
| Tributyltin methacrylate | 002155-70-6 | | X | |
| S,S,S-Tributyltrithiophosphate (DEF) | 000078-48-8 | | X | X |
| Trichloroacetyl chloride | 000076-02-8 | X | | |
| 1,2,3-Trichloropropane | 000096-18-4 | | X | |
| Triclopyr triethylammonium salt | 057213-69-1 | | X | |
| Triethylamine | 000121-44-8 | X | | |
| Triforine (N,N'-[1,4-Piperazinediylbis-2,2,2-trichloroethylidene]) bisformamide | 026644-46-2 | | X | |
| Trimethylchlorosilane | 000075-77-4 | X | | |
| 2,3,5-Trimethylphenyl methylcarbamate | 002655-15-4 | | X | |
| Triphenyltin chloride | 000639-58-7 | | X | X |
| Triphenyltin hydroxide | 000076-87-9 | | X | X |
| Vinclozolin (3-(3,5-Dichlorophenyl)-5-ethenyl-5-methyl-2,4-oxazolidinedione) | 050471-44-8 | | X | |

EPA is deferring final action on 40 chemicals and one chemical category until a later date. These chemicals and the comments received on them raised particularly difficult technical or policy issues which will require additional time to address. The Agency does not believe that it would be in the spirit of community right-to-know to delay final action on the remaining 286 chemicals and chemical categories, pending completion of work on the more limited group. In a future rulemaking, EPA will make a final determination as to whether these chemicals should be added to EPCRA section 313. The public comment that has been received specific to these deferred chemicals will be addressed as part of the future rulemaking discussed above. These chemicals follow:

- o-benzyl-p-chlorophenol butylate
- butylated hydroxyanisole (BHA)
- calcium hypochlorite
- caprolactam
- carbon monoxide
- cyromazine
- dichloromethylphenylsilane
- dithiopyr
- 2,4-D 2-octyl ester
- flumetralin
- iprodione
- isophorone
- man made mineral fibers
- methylene bis(thiocyanate)
- nitric oxide
- nitrogen dioxide
- nine polycyclic aromatic compounds, specifically:
 - carbazole
 - cyclopenta(cd)pyrene
 - dibenz(a,c)anthracene
 - dibenz(a,j)anthracene
 - 2-methylchrysene
 - 3-methylchrysene
 - 4-methylchrysene
 - 6-methylchrysene
 - 2-methylfluoranthene
- phosphorus oxychloride
- phosphorus pentachloride

- phosphorus pentasulfide
- phosphorus pentoxide
- primsulfuron
- sodium chlorite
- sodium hypochlorite
- sodium 2-pyridinethiol-1-oxide
- sulfur dioxide
- sulfur trioxide
- tefluthrin
- thiabenzazole, hypophosphite salt
- trichloroethylsilane
- trichlorophenylsilane
- vanadium pentoxide

Based on an evaluation of the public comments received and a reanalysis of the available data cited in the proposed rule, EPA has determined that three chemicals, clomazone, 5-chloro-2-(2,4-dichlorophenoxy)phenol, and tetrasodium ethylenediaminetetraacetate, that were proposed for listing do not have sufficient evidence of toxicity at this time to meet the statutory criteria of EPCRA section 313(d)(2) and thus are not listed in this final rule. Summaries of responses to chemical-specific comments for these chemicals appear in unit IV.G. of this preamble.

IV. Summary of Public Comment

The public comment period for the proposed rule closed April 12, 1994. On March 9, 1994, EPA held a public meeting on the proposed addition of chemicals and chemical categories. Two hundred and sixty-six comments were received, including 136 from industry, 60 from trade associations, 32 from environmental groups, 15 from private citizens, 3 from Federal agencies, 7 from State agencies and 13 from other public interest groups, labor groups, universities, and associations. In addition to general comment and comment addressing a broad number of chemicals, EPA received specific technical comments on 110 of the chemicals and chemical categories. Detailed responses to all comments, except those comments specific to

chemicals for which final action is being deferred, are contained in the *Response to Comment Document* (Ref. 14).

In addition to a number of comments supporting the concept of chemical expansion, EPA received comments in the following major areas: EPA's screening process used to identify potential candidates and the Agency's use of the *Draft Hazard Assessment Guidelines* (Ref. 11); the use of exposure in determining if a chemical meets the statutory criteria of EPCRA section 313; listing of categories; the addition of chemicals that are regulated by the Food and Drug Administration (FDA); the addition of chemicals that are regulated under FIFRA; duplicative reporting; general technical comments; and chemical-specific comments.

A. Comments on EPA's Screening Process Used to Identify Potential Candidates for Addition to EPCRA Section 313 and on EPA's Use of the Draft Hazard Assessment Guidelines

1. *Screening based on toxicity.* Monsanto, Zeneca Incorporated, and the National Oilseed Processors Association contend that the use of minimum effective doses (MEDs) to screen chemicals as potential candidates for addition to the EPCRA section 313 list was unrealistic and overly broad as a screening tool. One of these commenters also contended that EPA based its proposed addition on toxicity screening only.

EPA believes that the commenter may have misunderstood the use of the MED screening criteria. The MED screen is not intended, and is not used by EPA, as a surrogate for the actual statutory listing criteria. The MED was used as a screening tool during the preliminary review of several thousand candidate chemicals, because MED values were available and they are based on experimental values. MEDs are not equivalent to lowest-observed-adverse-effect levels (LOAELs). MEDs are

generally derived from LOAELs from chronic toxicity studies using a log transformation and as such a MED is a single value based upon the best available study. Satisfying the MED screening criteria, however, does not mean that a chemical will necessarily be added to the list. In every case, the Agency determines that at least one of the section 313(d)(2) criteria is met before a chemical is listed. For example, isoprene, 1,3-dichloropropane, and dichlorodimethylmethane passed the toxicity screen, but upon a more detailed review, were determined not to meet the criteria of EPCRA section 313(d)(2) and thus were not proposed for addition.

EPA believes that MEDs are useful as a screening tool and that the methodology has been adequately reviewed both internal and external to the Agency. The MED system was first presented in a peer reviewed article by DeRosa, et. al (Ref. 2). The MED methodology has been used by EPA in programs other than EPCRA section 313. For example, the MED methodology is integral to the reportable quantity (RQ) scoring system as utilized by EPA in CERCLA section 102. The RQ scoring system scheme is described in several **Federal Register** documents (April 4, 1985, 50 FR 13456; September 29, 1986, 51 FR 34535; and March 16, 1987, 52 FR 8140). Further, the Superfund Amendments and Reauthorization Act of 1986 (SARA) required EPA and the Agency for Toxic Substances and Disease Registry (ATSDR) to develop a list of 275 hazardous substances most commonly found at facilities on the National Priorities List (NPL) and considered to present the most significant threat to human health at those sites or at other facilities where releases may occur. During development of criteria to select the first list of 100, the RQ methodology (as discussed in the *Draft Hazard Assessment Guidelines*, Ref. 11) was selected as one of the evaluation tools used to develop the initial list, and the annual updates. When the initial list was published (April 17, 1987, 52 FR 12866) a summary of the methodology used to develop the list was provided.

Monsanto believes that the use of an MED of 500 mg/kg/day as the upper limit of the "may be sufficient" category of the screening criteria required an unrealistically high dose to have been used for toxicity testing.

EPA agrees that the upper bound for the medium priority category may warrant reconsideration. EPA will address this issue and other comments received on the *Draft Hazard Assessment Guidelines* (Ref. 11), when

the Agency finalizes that document. However, none of the chemicals proposed for listing in the proposed rule had MEDs that approached this upper bound. Of the chemicals proposed for addition pursuant to EPCRA section 313(d)(2)(B), greater than 93 percent had MED values that were in the range for the high priority category; the remaining chemicals (less than 7 percent) had MEDs in the lowest fifth of the medium priority category range, i.e., MEDs only slightly greater than the high priority category range. EPA reiterates that the MED screen is not intended, and is not used by EPA, as a surrogate for the actual statutory listing criteria. Additions to EPCRA section 313 are based on a hazard assessment, and, where appropriate, an analysis of exposure, to determine whether the chemical meets one or more of the EPCRA section 313(d)(2) listing criteria.

The Natural Resources Defense Council supports the health and environmental effects screening criteria used by EPA as a reasonable basis to screen chemicals as candidates for possible addition to EPCRA section 313.

The Agency agrees with this commenter in its support of the use of the screening criteria and believes that the screening criteria provide a reasonable basis to make a preliminary evaluation of chemicals for possible addition to the EPCRA section 313 list. EPA also agrees with the commenter's statement that the specific screening values are consistent with established risk assessment procedures applied in other EPA programs.

2. Screening based on production volume. Eastman Chemical Company states that, in addition to the use of a production volume screen, the Agency should consider the number of TRI Form Rs that would likely be submitted subsequent to listing. If the number is considered to be minimal (perhaps 5, 10, 15 or more reports), then EPA should balance the public's right-to-know with the economic burden placed on an industry.

EPA adopted a production volume screen for the development of the proposed rule to screen out those chemicals for which no reports are expected to be submitted. The Agency believes that it has the discretion to not include such chemicals at this time. If chemicals that did not meet the production volume screen were listed, there would be an economic burden for firms that would have to determine that they did not exceed the reporting threshold, without providing any information to the public.

While the Agency has determined to not list chemicals for which no reports

would be submitted, EPA believes that it is appropriate to add chemicals to EPCRA section 313 for which even a small number of reports are likely to be submitted nationally. In such cases, the reporting facilities will still provide important information to the surrounding communities. Even though a particular chemical may only be manufactured, processed, or otherwise used at a relatively small number of facilities, the data provided in the TRI Form R reports by these facilities could represent significant information in the communities in which the facilities are located. The Agency believes that it would be inconsistent with the public's right-to-know not to list chemicals even if only a low number of reports is expected.

3. Use of the Draft Hazard Assessment Guidelines. Six industry trade organizations and three companies contend that EPA's use of the *Draft Hazard Assessment Guidelines* (Ref. 11) was inappropriate. The commenters state that the use of the term "draft guidelines" indicates that the document requires additional review. Therefore, they believe that EPA should refrain from using the document to support this rulemaking.

It is appropriate for EPA to use the *Draft Hazard Assessment Guidelines* (Ref. 11), as it did in this rule, in considering whether to list a chemical on the section 313 list. The *Draft Hazard Assessment Guidelines* are an embodiment of internal EPA practices that have been used in listing determinations that have evolved since the inception of the TRI program. The *Draft Hazard Assessment Guidelines* do not constitute a set of rules for adding or deleting chemicals to or from the list; the *Draft Hazard Assessment Guidelines* are an explanation of the process and general standards for evaluating chemicals against the EPCRA section 313 listing criteria. These *Draft Hazard Assessment Guidelines* notwithstanding, EPA has evaluated every chemical proposed for addition directly against the EPCRA section 313 statutory criteria, and has taken into consideration comments submitted by the public specific to those chemicals (responses to those chemical-specific comments are found in the *Response to Comment Document*, (Ref. 14); summaries of most significant chemical-specific comments are found in units IV.F. and IV.G. of this preamble).

B. Use of Exposure Assessments

One of the most significant issues raised by commenters relates to the Agency's consideration of hazard, exposure, and risk in interpreting the

section 313(d)(2) criteria. Specifically, a number of commenters believe that EPA's interpretation of the EPCRA section 313(d)(2)(B) criterion, chronic human health effects, and the section 313(d)(2)(C) criterion, ecological effects, has been overly restrictive. The commenters contend that EPA should conduct risk assessments and make a formal determination that a chemical poses a risk (i.e., a combination of exposure and hazard) before adding it to the EPCRA section 313 list. The commenters argue that the following factors support their contention: (1) The statutory criteria include an implicit exposure and thus risk component; (2) the legislative history illustrates Congress' intent that exposure considerations were to be an integral part of determining whether a chemical should be listed on the EPCRA section 313 list; and (3) EPA should consider exposure in conjunction with section 313(d)(2)(B), chronic human health effects, and for all listings pursuant to section 313(d)(2)(C), ecological effects, because there is precedent for the use of exposure in previous listing and delisting actions.

In light of the many comments received on this issue, EPA has reviewed its positions in this area, and agrees with many of the commenters that there are limited circumstances under which it is appropriate for EPA to consider exposure factors for listing decisions under section 313(d)(2). The Agency believes that exposure considerations are appropriate in making determinations (1) under section 313(d)(2)(A), (2) under section 313(d)(2)(B) for chemicals that exhibit low to moderately low toxicity based on a hazard assessment (i.e., those chemicals for which the value of listing on the EPCRA section 313 list on hazard alone is marginal), and (3) under section 313(d)(2)(C) for chemicals that are low or moderately ecotoxic but do not induce well-documented serious adverse effects as described below. The Agency believes that exposure considerations are not appropriate in making determinations (1) under section 313(d)(2)(B) for chemicals that exhibit moderately high to high human toxicity (These terms, which do not directly correlate to the numerical screening values reflected in the *Draft Hazard Assessment Guidelines*, are defined in unit II.) based on a hazard assessment, and (2) under section 313(d)(2)(C) for chemicals that are highly ecotoxic or induce well-established adverse environmental effects. For chemicals which induce well-established serious adverse effects, e.g.,

chlorofluorocarbons, which cause stratospheric ozone depletion, EPA believes that an exposure assessment is unnecessary. EPA believes that these chemicals typically do not affect solely one or two species but rather cause changes across a whole ecosystem. EPA believes that these effects are sufficiently serious because of the scope of their impact and the well-documented evidence supporting the adverse effects.

EPA, however, disagrees with those commenters who suggest that EPA must include a risk assessment component to EPCRA section 313 determinations. Specifically, EPA does not agree with the commenters about the extent to which exposure must be considered in making determinations under sections 313(d)(2)(B) and (C). This is primarily because EPA does not agree with the commenters' understanding of EPCRA section 313. Risk assessment may be pertinent and appropriate for use under statutes that control the manufacture, use, and/or disposal of a chemical, such as the Clean Air Act or the Toxic Substances Control Act. However, EPCRA section 313 is an information collection provision that is fundamentally different from other environmental statutes that control or restrict chemical activities.

EPCRA section 313 charges EPA with collecting and disseminating information on releases, among other waste management data, so that communities can estimate local exposure and local risks; risks which can be significantly different than those which would be assessed using generic exposure considerations. The intent of EPCRA section 313 is to move the determination of what risks are acceptable from EPA to the communities in which the releases occur. This basic local empowerment is a cornerstone of the right-to-know program.

EPCRA section 313 establishes an information collection and dissemination program, the burden it imposes is significantly less than the burden imposed by a statute which controls the manufacture, use, and/or disposal of a chemical. EPCRA section 313 requires that a facility use the best available information to prepare each chemical-specific TRI report. However, the statute does not require that the facility conduct monitoring or emissions measurements to determine these quantities. A facility must only estimate, to the best of its ability, the quantitative information it reports. This is in contrast to other environmental statutes that may require a facility to monitor releases, change its manufacturing

process, install specific waste treatment technology, or dispose of wastes in a certain manner. As such, the Agency believes that the standard that must be met to require information submission under EPCRA section 313 is less than that to regulate a chemical under a statute such as the Clean Air Act.

EPA believes that its position regarding the use of hazard, exposure, and risk in listing decisions is consistent with the purpose and legislative history of EPCRA section 313, as illustrated in the following passage from the Conference report:

The Administrator, in determining to list a chemical under any of the above criteria, may, but is not required to conduct new studies or risk assessments or perform site-specific analyses to establish actual ambient concentrations or to document adverse effects at any particular location. (H. Rep. 99-962, 99th Cong., 2nd Sess., p. 295 (Oct. 3, 1986)).

This passage indicates Congress did not intend to require EPA to conduct new studies, such as exposure studies, or perform risk assessments, and therefore did not consider these activities to be mandatory components of all section 313 decisions. EPA believes that this statement combined with the plain language of the statutory criteria clearly indicate that Congress intended that the decision of whether and how to consider exposure under EPCRA section 313(d)(2)(B) and (C) should be left to the Agency's discretion. EPA has carefully considered when and how to use exposure to fully implement the right-to-know provisions of EPCRA. The Agency believes that in this final rule, EPA has appropriately used the discretion provided to it to assure the addition of chemicals that meet the right-to-know objectives of EPCRA section 313 while not unduly burdening the regulated community.

EPCRA section 313 specifically requires that exposure be considered for listing a chemical pursuant to section 313(d)(2)(A). The statute mandates that EPA consider whether "a chemical is known to cause or can reasonably be anticipated to cause significant adverse acute human health effects at concentration levels that are reasonably likely to exist beyond facility site boundaries." EPA has, and will continue to look at exposures reasonably likely to exist beyond facility site boundaries when making a listing determination pursuant to EPCRA section 313(d)(2)(A).

The statute is silent on the issue of exposure considerations for the section 313(d)(2)(B) and (C) criteria. The language of section 313 does not prohibit EPA from considering exposure

factors when making a finding under either section 313(d)(2)(B) or section 313(d)(2)(C). However, the language of sections 313(d)(2)(B) and (C) does not require the type of exposure assessment and/or risk assessment argued by the commenters. EPA believes that it has the discretion under both section 313(d)(2)(B) and section 313(d)(2)(C) to consider, where appropriate, those exposure factors that may call into question the validity of listing of any specific chemical on TRI. In exercising this discretion, EPA considers it appropriate to employ exposure considerations to a limited extent in making determinations under EPCRA section 313(d)(2)(C) because this criterion requires the Agency to find a "significant adverse effect on the environment of sufficient seriousness, in the judgment of the Administrator to warrant reporting" under EPCRA section 313. This language recognizes the possibility that under certain circumstances, a chemical that could theoretically cause an adverse effect on the environment is unlikely to cause one of a magnitude sufficient to warrant listing. Moreover, because of the limitation on the number of chemicals listed pursuant to only section 313(d)(2)(C) that may be listed, EPA believes that it is appropriate to use both hazard and exposure factors as prioritizing considerations in these listing decisions. Therefore, to meet its obligation under section 313(d)(2)(C), in cases where a chemical is low or moderately ecotoxic, EPA may look at certain exposure factors (including pollution controls, the volume and pattern of production, use, and release, environmental fate, as well as other chemical specific factors, and the use of estimated releases and modeling techniques) to determine if listing is reasonable, i.e., could the chemical ever be present at high enough concentrations to cause a significant adverse effect upon the environment to warrant listing under section 313(d)(2)(C). Of the chemicals being added in today's action pursuant to section 313(d)(2)(C), all but one are highly ecotoxic. These highly ecotoxic chemicals are being added to the EPCRA section 313 list pursuant to section 313(d)(2)(C) based on their hazard. The other chemical, which is moderately ecotoxic, is being added to the EPCRA section 313 list pursuant to section 313(d)(2)(C) based on both its hazard and an exposure assessment for this chemical.

For listing determinations made pursuant to EPCRA section 313(d)(2)(B), in instances where the hazard

assessment indicates that the value of listing on EPCRA section 313 on hazard alone is marginal (i.e., a chemical is of low toxicity and unrealistic exposures would be necessary for it to pose a risk to communities), EPA may use exposure considerations in its listing decisions. Only chemicals for which the hazard assessments indicate moderately high to high toxicity are being added in today's action to the EPCRA section 313 list pursuant to section 313(d)(2)(B). None of these chemicals are chemicals for which the consideration of exposure factors would be appropriate.

Through this rulemaking, EPA is clarifying its position regarding the use of hazard, exposure, and risk in listing decisions under EPCRA section 313. EPA will consider exposure factors when making determinations under section 313(d)(2)(A) (acute human toxicity). In addition, EPA has discretion to consider exposure factors where appropriate for determinations under sections 313(d)(2)(B) (chronic human toxicity) and (C) (environmental toxicity), and that there is a broader range of circumstances in which exposure will be considered under section 313(d)(2)(C) than under (B).

EPA has reviewed its past listing decisions in light of this clarification, and believes that its prior listing determinations have been consistent in the consideration of exposure in 31 of the 32 listing/delisting determinations previous to this action, including a number of deletions of low toxicity chemicals that Congress placed on the initial EPCRA section 313 list. EPA is currently reviewing the one exception, inorganic fluorides, to determine if additional action is warranted. EPA will continue to evaluate petitions according to this clarification and will delete chemicals that do not meet the statutory criteria.

C. Addition of Categories

Six industry trade organizations, 7 companies, and the Department of Energy contend that section 313 does not provide EPA the statutory authority to list chemical categories. Some of the commenters contend that the intent of Congress was for EPA to review individual chemicals. Therefore, the commenters believe that EPA should list all chemicals individually. General Electric, American Iron and Steel Institute, and Eastman Chemical Company further contend that, based on legal precedent (citing *AFL-CIO vs. OSHA*, 965 F.2d 9262 (11th Cir. 1992)), EPA does not have the authority to list chemical categories or specific groups of chemicals.

EPA believes that the statutory authority to add "a chemical" to the list may be reasonably interpreted to include the authority to list groups or categories of chemicals. Indeed, this interpretation is supported by the initial list of chemicals and chemical categories adopted by Congress in section 313(c). In that initial list, Congress included 20 chemical categories, mainly metal compounds, but also categories of organic chemicals such as chlorophenols. Nothing in section 313 or its legislative history indicates or even suggests that Congress intended to preclude EPA from adding chemical categories to the list where the appropriate findings can be made.

Where, as with the categories being added in this final rule, EPA determines that the primary purpose of TRI--providing information to the community about the release of chemicals--is most appropriately served by listing a category of chemicals, EPA has the discretion to list a category rather than individual chemicals. Of course, in adding a category to the list, EPA must comply with the statutory criteria. The Agency believes it satisfies the statutory criteria to add a category to the list by identifying the toxic effect of concern for at least one member of the category and then showing why that effect may reasonably be expected to be caused by all other members of the category. A specific justification for each of the categories included in the final rule has been provided in the preamble of the January 12, 1994 proposed rule, in the docket supporting this rulemaking, and in the *Response to Comment Document* (Ref. 14).

Several commenters raised policy concerns and suggested that there would be regulatory difficulties associated with adding chemical categories. These are addressed below.

One commenter suggested that the regulated community would face uncertainty in deciding which chemicals belong in the category. In this final rule, EPA has described the categories in sufficient detail to alleviate uncertainty regarding their membership. Of course, the Agency will work with the public and the regulated community to develop, as appropriate, any interpretations and guidance the Agency determines are necessary to facilitate accurate reporting for these categories.

One commenter questions how to properly report a chemical which could be considered part of a category and which is also specifically, individually listed. Threshold determinations should be made for the individually-listed chemical rather than for the category. The current EPCRA section 313 list

contains some individually-listed chemicals that also meet the definition of an EPCRA section 313 listed category. For example, pentachlorophenol is listed individually on EPCRA section 313 but also meets the definition of the chlorophenol category. In these situations, threshold determinations should be made for the chemical as an individual entity rather than as a member of the category. A facility would not count the quantities manufactured, processed, or otherwise used toward threshold determinations for both the individual listing and the category listing, but rather only toward the individual chemical threshold.

One commenter contends that categories will lead to inadvertent non-compliance with reporting requirements. EPA does not believe that this is a significant concern. Because the categories being added to the EPCRA section 313 list today each consist of chemicals that are similar chemically and in effect, EPA believes that these categories will not be difficult for the public or industry to understand or for the Agency to administer. In addition, there are already categories on the current list, and EPA has not experienced a significant problem of the sort suggested by the commenter. The Congressional objective of providing information is outweighed by any possible problems that some facilities might have with inadvertent noncompliance.

One commenter states that the use of categories will artificially lower the thresholds for reporting chemicals within the category. The Agency believes that calculating the thresholds based on the category (i.e., a sum of the activities for each individual category member) is appropriate and not "artificially lower." As described above, categories are placed on the EPCRA section 313 list where each of the members can be expected to cause similar effects because all members of the category have a similar functional group or exhibit a similar characteristic. For each of the categories added in today's rule, EPA believes that because each member of the category has this similar functional group or exhibits a similar characteristic, each member of the category can be reasonably anticipated to cause similar adverse effects. The members of the category are not randomly selected, but are closely related and warrant being reported as a category. These chemicals in aggregate can reasonably be anticipated to cause an aggregate impact of the adverse effect associated with each member of the category. Thus, it is appropriate to apply the reporting thresholds to the category

regardless of whether the threshold amount is attributable to one member of the category or to individual members in aggregate.

One commenter believes that listing broad categories where the individual members have diverse properties and cause diverse effects does not constitute "good science." The Agency agrees that a category must be rationally constructed both in terms of similarity in the properties of the individual members and in terms of their effects. There is, of course, no requirement that the properties across category members be absolutely identical. EPA agrees that the members of a category be reasonably expected to elicit the same type of effect or related effects in order for a category to satisfy the statutory listing criteria. Furthermore, EPA agrees that determinations to list a category, as with listing an individual chemical are to be based on "good science." EPA has applied these principles to the categories being added in the final rule.

D. Policy Issues

There are several policy issues which were consistently raised in comments on specific chemicals and general comment on the entire proposed rule. For purposes of this final rule, EPA addresses these issues in this unit of the preamble and not in unit IV.F. of the preamble in the responses to chemical-specific comments. Detailed responses to comments on specific individual chemicals are available in the *Response to Comments Document* (Ref. 14).

1. *The addition of chemicals that may be released in small quantities.* Many commenters object to the addition of many of the chemicals to the EPCRA section 313 list because they do not believe that there will be significant releases of these chemicals. Therefore, they contend there will not be significant exposure to these chemicals and the associated risks will be low.

EPA believes that the chemicals added today meet the EPCRA section 313(d)(2) criteria and should be included on the EPCRA section 313 list. The quantity of a chemical released is not part of the statutory criteria. The purpose of EPCRA section 313 is to collect data on the quantity released so that local communities can make their own determinations about exposure.

Congress intended EPCRA section 313 to address the lack of information on toxic chemicals in communities by providing information on releases of toxic chemicals. The public can then use this release information with site-specific information and the appropriate attributes of a chemical to evaluate exposure. EPA considers it

inappropriate under the right-to-know program to supplant the public's power to make risk determinations on a community level by the Agency's use of specified levels of potential releases, exposure, or risk as screening criteria to exclude chemicals from the EPCRA section 313 list. By listing chemicals that present a hazard and providing TRI data on these chemicals to the public, EPA allows the public to make the determination as to whether there is a risk in their community. Furthermore, any exposure assessment conducted by EPA would be conducted from a national perspective and may not truly represent the risks to a specific community. (For a more detailed discussion on the Agency's use of exposure see Unit IV.B. of this preamble).

2. *The addition of chemicals that are regulated by FDA.* Eli Lilly and Company, National Agricultural Chemical Association, Pharmaceutical Manufacturers Association, and Hoffman-La Roche state that chemicals which are regulated by the FDA should not be added to EPCRA section 313. The commenters argue that the FDA approves a drug only after extensive testing and a determination that the benefits to the patients outweigh the risks. The commenters further state that access to these drugs is controlled because they can only be obtained through a medical doctor.

EPA agrees that the drug testing and approval process conducted by the FDA is extensive and necessary to protect the public health and well-being. However, as discussed above, the purpose of listing these chemicals under EPCRA section 313 is to provide information on the release, transfer, and waste management activities occurring in the community. This is a different function that addresses different issues than those addressed by FDA. Furthermore, while the main use of these chemicals is pharmaceutical in nature, that does not mean that they are not a hazard in other contexts. EPA agrees that in controlled situations (e.g., a doctor's prescription) ingestion of a drug is likely to have certain intended benefits. However, outside of this controlled situation, any adverse effects are not balanced by the benefits received from the use of the drug. Further, EPCRA section 313 will collect information on the release and disposal of these chemicals, which is not covered by the regulation of the use of a chemical as a drug.

3. *Chemicals regulated under FIFRA.* Several commenters do not support the addition of chemicals regulated under FIFRA to the EPCRA section 313 list of

toxic chemicals because, they contend, the major route of exposure, agricultural field use, has been addressed through FIFRA regulation which establishes safety factors and use directions allowing for safe use. They further contend that the use of these chemicals has been determined not to present an unreasonable risk and therefore, listing pesticides under EPCRA section 313 is unnecessary.

FIFRA regulations require that the Agency determine that pesticidal uses of a chemical do not cause "unreasonable adverse effects on the environment" which is defined in FIFRA section 2(bb) as "any unreasonable risk to man or the environment taking into account the economic, social, and environmental costs and benefits of the use of pesticides" (7 U.S.C. section 136(bb)). FIFRA is a regulatory statute, and the impacts of regulation can be immediate and direct (e.g., banning of a chemical), and as such EPA examines not only the hazards presented by the chemical, but also the specific exposure scenarios, and weighs the risks against the benefits of the chemical. The "unreasonable adverse effects" determination under FIFRA is specific to the intentional use of the chemical as a pesticide and does not address other uses or releases of the chemical that may result from manufacture, processing, or other use. Furthermore, a determination under FIFRA that the use of a chemical will not result in an "unreasonable adverse effect" is not a determination that the chemical is not hazardous or that the use of the chemical is without risk. Finally, EPCRA section 313 was not enacted to serve the same purpose as FIFRA. Listing on EPCRA section 313 provides communities with some of the information required to determine what risks may result from the manufacture, processing and non-pesticidal use of a chemical, information not generally provided through FIFRA.

4. *Duplicative reporting.* Many commenters believe that listing some of the chemicals proposed will result in duplicative regulation that will be unduly burdensome and of little benefit. One other commenter, Westinghouse Electric Corporation, states that EPA should utilize existing sources of information to avoid duplicative reporting.

Congress did not intend that the chemicals listed under EPCRA section 313 be limited to those that are *not* regulated under other environmental statutes and for which no information is collected pursuant to other requirements. The initial list of chemicals that Congress included in section 313 consisted of substances

regulated under RCRA, CWA, SDWA, CERCLA, FIFRA, and CAA. Further, as Representative Edgar stated in the House of Representatives debate on the Conference bill:

With respect to the contents of the toxic release form, estimates of releases into each environmental medium must be provided. This shall include any releases into the air, water, and land, as well as releases from waste treatment and storage facilities. This shall include all releases of toxic chemicals into surface waters whether or not such releases are pursuant to the Clean Water Act permits. (132 Cong. Rec. H9561, October 8, 1986)

EPA believes that the chemicals being added today meet the toxicity criteria of EPCRA section 313(d)(2) and, therefore, should be added to the EPCRA section 313 list. EPA further believes that the EPCRA section 313 requirements do not duplicate other regulatory program requirements. EPCRA was not enacted to serve the same purpose as other regulatory programs but to collect and disseminate information to the public. Nor is EPCRA section 313 intended to regulate how a chemical may be used, the amount of chemical a facility manufactures, processes, otherwise uses, and releases, what media the chemical is released to, or how the chemical is disposed. Therefore, TRI, as an information collection and dissemination program, is not designed to directly impose controls for the protection of human health or the environment in the same manner as other regulatory programs. The benefit of TRI is that it empowers the public, through access to release, transfer, and waste management data on toxic chemicals, to make determinations about risks in their communities based on TRI data, site-specific information, and the properties of the chemicals.

E. General Technical Comments

1. *Maternal toxicity.* A number of commenters argued that for certain chemicals in animal tests, the only evidence for developmental toxicity occurred at maternally toxic doses (that is, doses that were high enough to induce toxicity in the mother), and, therefore, developmental toxicity cannot be used as a basis for listing these chemicals under EPCRA section 313. EPA disagrees that fetal effects only in the presence of maternal toxicity demonstrate that a given substance does not present a developmental hazard. Although the developmental effects may have been seen in the presence of reversible maternal effects, the developmental effects may be more permanent and cannot be treated as only secondary to reversible maternal

toxicity. With regard to adverse effects in the presence of maternal toxicity, EPA believes that developmental effects at maternal toxicity are "... toxic manifestations and as such are generally considered a reasonable basis for Agency regulation and/or risk assessment" (Ref 6). This approach has particular relevance in situations where reversible maternal toxicity may occur in the presence of irreversible adverse fetal effects. The Agency does not distinguish between fetal effects observed in the presence of maternal toxicity or those observed without concomitant maternal toxicity. Both maternal and fetal toxicity are of concern to the Agency, and are within the criteria of EPCRA section 313(d)(2). Thus, EPA will use the effect, maternal or fetal, which is most sensitive to set LOAELs and no-observed-adverse-effect levels (NOAELs). If both occur at the same level, the LOAELs and NOAELs for both are the same. When the LOAEL is the same for the adult and developing organisms, it may simply indicate that both are sensitive to that dose level, rather than that the developmental effects result only from maternal toxicity. Moreover, whether developmental effects are secondary to maternal toxicity or not, the maternal effects may be reversible while effects on offspring may be permanent. There are several agents known to produce adverse developmental effects at minimally toxic doses in adult humans (e.g., tobacco smoking, alcohol, isotretinoin).

2. *Use of IRIS and other secondary sources.* Several commenters object to EPA's use of the Agency's Integrated Risk Information System (IRIS) data base, the Agency's Office of Pesticide Programs' 1988 TOX-One-Liners data base, Registry of Toxic Effects of Chemical Substances (RTECS) data base, and the Aquatic Information Retrieval (AQUIRE) data base. The commenters contend that in relying on these sources the Agency ignores other pertinent data that may be in its possession. They contend that EPA should have examined the primary sources, rather than relying on data bases which are summaries of studies. Specifically, some commenters claim that there are many studies in EPA's possession, but not included in the 1988 TOX-One-Liner data base, that appear not to have been considered in the review process, because they have not yet been reviewed by EPA's Office of Pesticide Programs. The commenters contend that reliance on IRIS or the 1988 TOX-One-Liner data base does not constitute a detailed analysis and careful

examination of the available data on a chemical.

EPA disagrees with the commenters. EPA's use of the Agency's IRIS data base for EPCRA section 313 purposes does constitute a hazard evaluation. That data base generally provides information against which EPA can evaluate the section 313(d)(2) criteria. The information contained in the IRIS data base represents the Agency's weight-of-evidence hazard assessment for chemicals contained in the data base. The information was developed after the Agency's thorough scientific review of the available data. Therefore, by relying on information in the IRIS data base in the review of chemicals for listing on EPCRA section 313, EPA made statutory determinations based on hazard assessments conducted by the Agency.

Although the 1988 TOX-One-Liners were used as part of the Agency's evaluation of the toxicity of a candidate chemical, a number of other sources were also used. These include decision documents from a number of Agency and EPA internal peer review groups, deliberations of the FIFRA Scientific Advisory Panel, and reference to data evaluation records for studies used in support of listing. Therefore, evaluations of the toxicity of individual chemicals has been made on the entire data base and did not rely only on the 1988 TOX-One-Liners data base. Furthermore, inclusion of all of the detailed studies in the docket was not possible, because of the proprietary nature of some of the information. However, in cases where relevant information was used in support of the listing decision, but was not included in the 1988 TOX One-Liners data base (which is the most recent *sanitized* version of the data base), sanitized versions of the additional sources were included in the docket. In those cases where only the 1988 TOX-One-Liners data base or other similar sources were cited, no additional data not described in the 1988 TOX-One-Liners, RTECS, or the AQUIRE data bases was considered to be relevant to this listing. For a few chemicals it has become apparent based on comments received that EPA's analysis did not include studies which are in EPA's possession but which EPA has not reviewed. The Agency is deferring the final action on these chemicals until such studies can be reviewed.

3. *Testing at toxic doses.* A number of commenters stated that pesticides which are registered under FIFRA should not be listed under EPCRA section 313 because the testing conducted to obtain a pesticide

registration under the FIFRA review process requires testing at dose levels "virtually guaranteed to produce a toxicological effect."

It is not EPA's position that chemicals registered as pesticides under FIFRA should be precluded from listing simply because these chemicals were tested at doses which are designed to produce toxic effects. The commenters are correct that the FIFRA standard study design attempts to set the doses at levels which bracket the minimal toxic dose, and, therefore, the high dose(s) by design produces an effect. The purpose of this study design under FIFRA is to determine the potential for toxicity of the chemical, whether the responses are dose-related and, depending on the effects produced, the *degree* of toxicity. Because virtually any chemical substance can elicit a toxicological response at some dose level, the mere presence of the toxic response is not used in isolation in listing decisions under EPCRA section 313. Rather, it is the relative severity of the effect, the presence of a dose/response relationship, and whether the effect is manifested at relatively low doses which are considered in determining the hazard of the chemical, and in making listing determinations under EPCRA section 313.

4. *Precursor chemicals.* CRF AG Products Company, Monsanto, FMC Corporation, Eastman Chemical Company, and the Chemical Manufacturers Association question EPA's authority to list precursor chemicals (i.e., a chemical that reacts in vivo or in the environment to generate another chemical that produces the toxic effect supporting the listing) on the EPCRA section 313 list. The commenters believe that a chemical should only be added to the list based on the toxicity of the chemical itself. Further they contend that nowhere in the legislative history is there any indication that post-release transformation products, degradation products, or products of chemical reactions are legitimate bases for adding chemicals to the EPCRA section 313 list.

The EPCRA section 313(d)(2) listing criteria each state that EPA may list a chemical that it determines "causes or may reasonably be anticipated to cause" the relevant adverse human health or environmental effects. EPA believes that this language allows EPA to consider the effects caused by the degradation products of a listed chemical. Where it may reasonably be anticipated, based on available data, that the listed chemical would readily degrade into another chemical that would cause the adverse effect, EPA is acting reasonably and

within its grant of authority in listing the precursor to the toxic degradation product.

Furthermore, one could also view the effects caused by the degradation product as effects indirectly caused by the listed chemical. EPA believes it is within its authority to consider both the direct and indirect adverse human health and environmental effects of a chemical in making a listing determination. Based on the statutory language and legislative history, EPA interprets EPCRA section 313(d)(2) to include toxic effects indirectly caused by a listed chemical. The statute and the legislative history do not specifically preclude EPA from considering indirect effects in deciding whether a chemical meets the toxicity criteria under section 313. In the absence of specific congressional intent on the issue, it is reasonable for EPA to consider indirect effects in light of the broad statutory purpose to inform the public about releases of toxic chemicals to the environment. Were EPA to exclude indirect effects from consideration it would ill-serve the purpose of the statute by precluding public access to information about chemicals that, albeit, indirectly cause a wide range of adverse health and environmental effects.

There is precedent for the Agency to consider the "indirect" toxicity of a chemical being considered for listing. Indirect toxicity was the basis for the granting of two petitions, one to add seven chlorofluorocarbons and halons (August 30, 1990, 55 FR 31594) and a second to add hydrochlorofluorocarbons to the EPCRA section 313 list (December 1, 1993, 58 FR 64936). EPA also used indirect toxicity in support of its denial of petitions to delete certain volatile organic chemicals from the section 313 list, specifically, the ethylene and propylene petition (January 27, 1989, 54 FR 4072) and the cyclohexane petition (March 15, 1989, 54 FR 10668).

5. *Use of studies conducted by routes other than oral, inhalation, or dermal.* Several commenters maintain that intraperitoneal, intravenous, or subcutaneous injection (injection into the abdomen, a vein, or under the skin, respectively) has minimal relevance for evaluating potential human exposure from industrial situations and should not be used to support an EPCRA section 313 listing decision. One commenter contends that, if considered at all, intraperitoneal injection is a form of exposure that should be considered in establishing a section 313(d)(2)(A) finding of acute effects, not a section 313(d)(2)(B) finding of chronic effects.

EPA disagrees with the commenters. In making section 313 listing decisions,

the Agency cannot ignore the possible significance of any existing data, including data from intraperitoneal, intravenous, or subcutaneous injection studies. Although it is preferable to have toxicity data from the common routes of human exposure, EPA believes that for hazard assessment under EPCRA section 313, the Agency should use all available information to identify the hazard associated with a chemical. This comment relates to five chemicals (bromacil lithium salt, fluorouracil, pentobarbital sodium, tetracycline hydrochloride, and sodium nitrite) that are being added to the section 313 list today. For three of these chemicals, bromacil lithium salt, fluorouracil, and sodium nitrite, any data from intraperitoneal or other injection routes of exposure are supplemented by data from other, non-injection exposure routes. For example, in addition to chronic dog and rat injection studies to support the chronic hematological concerns of sodium nitrite, there are human oral data. For bromacil lithium salt, intraperitoneal injection studies in rats are supplemented by gavage studies in mice to support the developmental concerns for this chemical. In addition to the developmental effects observed in the offspring of women receiving fluorouracil intravenously, developmental abnormalities in mice, rats and hamsters receiving fluorouracil orally were used to support the developmental toxicity finding. For both pentobarbital sodium and tetracycline hydrochloride, the studies cited in the proposed rule in support of the developmental effects of these chemicals are either studies in which the chemical was administered via injection or studies in which the chemical was administered via another route. However, because both of these chemicals are commonly administered orally, and are efficacious by this route (orally), there is reason to extrapolate the effects observed in injection studies to effects by other routes. The proposed rule and the *Response to Comment Document* (Ref. 14) contain information on EPA's review of these chemicals, including the toxicity evaluation. This background information will not be repeated here in the final rule. Based on EPA's reanalysis of the available information in the proposed rule for these five chemicals, EPA has sufficient evidence to determine that bromacil lithium salt, fluorouracil, pentobarbital sodium, tetracycline hydrochloride, and sodium nitrite have sufficient evidence to meet the statutory listing criteria under EPCRA section 313(d)(2)(B).

6. *Use of acute studies to support a chronic finding.* Several commenters object to the use of data from acute studies to support a finding of chronic toxicity. The commenters contend that there is no correlation between transient acute impact and chronic toxicity that is appropriate to industrial chemicals as a whole. The commenters contend that, if a chemical exhibits transient acute but not chronic effects, it should not be listed based on chronic toxicity, unless additional data on chronic effects are also used in the determination to list the chemical.

EPA agrees with the commenter that if a chemical exhibits acute toxic effects, it should be listed based on acute effects unless additional data on adverse effects after long-term exposure are available. This comment relates to three of the chemicals (bromine, 2-bromo-2-nitropropane-1,3-diol, and sodium nitrite) that are being added to the section 313 list today. For these chemicals, any data from acute studies are supplemented by chronic toxicity information. In chronic toxicity studies, bromine produced upper respiratory irritation and neurological symptoms. In chronic toxicity studies, 2-bromo-2-nitropropane-1,3-diol produced various effects including lesions of the stomach mucosa, ulceration, raised areas and excrescences, inflammation, epithelial hyperplasia and hyperkeratosis, and congested vessels of the mucosa of the gastrointestinal (G.I.) tract. Sodium nitrite induced, in a chronic study in mice, reduced motor activity and major electroencephalogram (EEG) changes in treated animals. The proposed rule and the *Response to Comment Document* (Ref. 14) contain information on EPA's review of these chemicals, including the toxicity evaluation. This background information will not be repeated here in the final rule. A summary of the response to comments for these chemicals is provided in Unit IV.F. of this preamble.

7. *Use of cholinesterase inhibition as a measure of neurotoxicity.* Several commenters expressed concern that the Agency has used a chemical's effect of inhibiting plasma, red blood cell (RBC) or brain cholinesterase activity as a basis for listing chemicals on the EPCRA section 313 list. These commenters feel that this effect is not an adequate indicator of neurotoxicity.

The Agency believes that inhibition of plasma, RBC, or brain cholinesterase activity is an appropriate indicator to assess the toxicity of potential neurotoxicants (Ref. 7). In order for the normal activity of the nervous system to be altered by a toxic chemical, the chemical must enter the body, reach the

tissue target site(s), and be maintained at a sufficient concentration for a period of time in order for an adverse effect to occur. Biochemical changes precede the more overt, physiological changes associated with neurotoxicity, and are more easily detectable.

Acetylcholinesterase (AChE) is the enzyme that inactivates or terminates the effect of the neurotransmitter (acetylcholine) on its target. When this enzyme is inhibited, acetylcholine is built up in the body, and may result in loss of appetite, anxiety, muscle twitching, paralysis, or other neurotoxic effects. Thus, one can assess the signs and symptoms of systemic poisoning by many neurotoxins from their biochemical mechanism of action, such as the inhibition of AChE. Because of the severity of these effects, EPA takes a cautious approach by using a measure of cholinesterase activity as an indicator of neurotoxicity.

The comments concerning cholinesterase inhibition relate to six of the chemicals that are being added to the section 313 list today. The proposed rule and the *Response to Comment Document* (Ref. 14) contain information on EPA's review of these chemicals, including the toxicity evaluation. This background information will not be repeated here. Based on comments received and EPA's reanalysis of the available information in the proposed rule for these six chemicals, EPA has sufficient evidence to determine that acephate, cycloate, diazinon, ethyl dipropylthiocarbamate, pirimphos methyl, and profenofos meet the statutory listing criteria under EPCRA section 313(d)(2)(B) based on available neurotoxicity data for these chemicals.

8. *Use of certain studies for hazard assessment.* Several commenters argue that EPA should not use studies in support of listing a chemical on the EPCRA section 313 list, if these studies have been determined to be insufficient for use in risk assessments under FIFRA or TSCA. For example, the commenters point to studies EPA considered in this rulemaking in conducting hazard assessments even though the studies when submitted for use under FIFRA or TSCA were determined by EPA to be of "low confidence." EPA believes its use of these studies for section 313 purposes is appropriate. The "low confidence" determination under FIFRA or TSCA applies to the use of the studies for purposes of risk assessment associated with regulations that impose controls. The data base for a chemical may be rated low confidence because of shortcomings such as lack of experimental detail. Although these studies may be of limited value for

purposes of risk assessment in support of regulatory controls, when considered together, they present a sufficient weight-of-evidence as to the hazard associated with the chemical. As additions to EPCRA section 313 made pursuant to EPCRA section 313(d)(2)(B) are not based on the kind of risk assessment needed for regulatory controls, EPA believes that such studies can be used to support listing.

9. *Docket was incomplete for certain chemicals.* Several commenters contend that the docket information supporting the listing of certain chemicals is incomplete. Other commenters contend that, overall, the docket is too general and limited. Responses to comments about the evidence provided in the docket for specific chemicals are provided in the *Response to Comment Document* (Ref. 14).

In the public docket supporting this rulemaking, EPA included copies of EPA's support documents (Refs. 9, 12, and 13) for the proposed rule and copies of the main references cited in those documents. The primary references that are cited in these main reference documents were not themselves included. However, these reference documents are published material, readily accessible, and are in the public domain. EPA believes that the docket material for both the proposed and final rules contains the appropriate information to support the addition of these chemicals to the EPCRA section 313 list and to have provided the public an adequate basis on which to comment on the proposed rule.

F. Chemical-Specific Comments for Chemicals that Are Being Finalized in Today's Action

The Agency received comments on 110 of the 313 specific chemicals included in the proposed rule. This unit of the preamble summarizes the most significant of those comments and the Agency's responses. More detailed responses are included in the *Response to Comment Document* (Ref. 14). Neither this unit of the preamble nor the *Response to Comment Document* addresses comments specific to chemicals that have been deferred for final action. These comments will be addressed in a separate rulemaking specific to those chemicals.

1. *Abamectin.* One commenter, Merck, states that primates are less sensitive to the acute effects of abamectin and its analog, ivermectin, than rodents. The commenter implies that because humans are primates, abamectin should be less toxic in humans than in rodents. The commenter further contends that

ivermectin and abamectin have been used safely in animals and humans.

Abamectin interferes with gamma-aminobutyric acid (GABA) transmission and, as such, produces neurotoxic clinical signs such as tremors, ataxia, convulsions, or coma that are more severe in rodents and dogs than primates. EPA agrees that the available studies indicate that the sensitivity as well as doses required to produce neurotoxic effects vary from rodents to primates by a 20-fold factor. However, abamectin was proposed for addition to the EPCRA section 313 list based on developmental effects rather than neurotoxicity. There are no developmental studies with abamectin in primates. Therefore, EPA believes that the rodent studies cited in the proposed rule provide sufficient evidence that abamectin can reasonably be anticipated to cause developmental toxicity in humans.

When administered in therapeutic doses, the Agency does not dispute the animal and human safety and efficacy of ivermectin and abamectin, but the safety of a 0.2 to 0.3 mg/kg single therapeutic dose does not diminish the findings of the developmental, reproductive, neurotoxic, chronic, and carcinogenic animal studies with abamectin which in some cases demonstrate serious compound-related effects at higher than therapeutic doses in all species tested.

The same commenter states that although the aquatic toxicity data cited for the proposed listing of abamectin under EPCRA section 313 are accurate and valid, it may be inappropriate to list abamectin under EPCRA section 313 based on the environmental fate of this chemical, because of environmental fate factors which were not presented by EPA in the proposed rule.

EPA agrees with the commenter that the aquatic toxicity values presented in the proposed rule are accurate and valid. EPA disagrees that the environmental fate of abamectin will negate the chemical's ecological toxicity. EPA believes that the environmental fate factors presented by the commenter may reduce, but do not eliminate, the potential for adverse effects on aquatic organisms because the chemical is extremely acutely toxic to aquatic organisms.

EPA reaffirms that there is sufficient evidence for listing abamectin on the EPCRA section 313 list pursuant to EPCRA section 313(d)(2)(B) based on the available developmental toxicity data and pursuant to EPCRA section 313(d)(2)(C) based on the available ecotoxicity data. Therefore, EPA is finalizing the addition of abamectin on the EPCRA section 313 list.

2. *Alachlor.* Monsanto states that at the highest dose tested in the chronic mouse study cited in the proposed rule, EPA concluded there was an increase in lung tumors in females. Monsanto believes that other regulatory agencies have disagreed with this conclusion. The commenter contends that these tumors occur spontaneously in mice with a fairly high and variable frequency and a possible slight increase in a common rodent tumor at the highest dose tested does not represent a risk to humans receiving, at most, trace level exposure.

The Agency has concluded that there was statistically significant increase (the increase was greater than that which would be expected to occur spontaneously) in lung tumors in female CD-1 mice at 2 dose levels which were relevant to potential carcinogenicity to humans. The commenter provides no specifics to support its contention that "other regulatory agencies have disagreed with this conclusion" nor is the Agency aware of any.

The commenter further states that the *Support Document for the Health and Ecological Toxicity Review of TRI Expansion Chemicals* (Ref. 13) also incorrectly listed the dose levels ("[greater than] 42 mg/kg/day") producing tumors in rats in the 2-year rat feeding study cited in the proposed rule. The commenter argues that significant increases in thyroid and stomach tumors were observed only at 126 mg/kg/day, the highest dose tested; this dose level also produced severe, excessive toxicity. Thus, the commenter concludes that the dose-response curves for the stomach and thyroid tumors are exceptionally steep, with increased incidences observed only at a dose which exceeded the Maximum Tolerated Dose (MTD).

EPA believes that the *Support Document for the Health and Ecological Toxicity Review of TRI Expansion Chemicals* (Ref. 13) correctly states the toxic dose levels in the 2-year rat feeding study as being greater than or equal to 42 mg/kg/day. In this study, nasal tumors were significantly increased at 42 mg/kg/day and above and the stomach and thyroid follicular cell tumors at 126 mg/kg/day. The Agency agrees that the 126 mg/kg/day dose level probably exceeded the MTD; however, upon reconsideration of the carcinogenicity data, the Agency determined that the MTD is between 42 mg/kg/day and 126 mg/kg/day. Although the MTD was exceeded by the highest dose (126 mg/kg/day), significant effects were seen at 42 mg/kg/day, which does not exceed the MTD. Therefore, EPA believes that the

2-year chronic dog study cited in the proposed rule is a valid measure of the oncogenic potential of alachlor.

The commenter cites a chronic rat feeding study, not cited by EPA in the proposed rule, in which 5 to 6 months of alachlor administration followed by 19 months on control diet did not produce a significant increase in stomach or thyroid tumors in rats. The commenter believes that this information is consistent with the results of a study, not cited by EPA in the proposed rule, in which a close structural chloroacetanilide analog of alachlor has been shown to be a promoter but not an initiator of stomach tumors. The commenter did not further identify this study.

Although in the chronic rat feeding study referred to by Monsanto, the specific group which received alachlor in the diet for 5 to 6 months in this study, and then control diet as a recovery period did not develop stomach or thyroid tumors, the other groups on study which continued to receive alachlor in the diet developed both stomach and thyroid tumors as well as nasal turbinate tumors. Therefore, the failure to develop stomach tumors after 5 to 6 months treatment reflects the time frame required for tumor development rather than indicating a lack of carcinogenic response.

The commenter also discusses the mechanism of carcinogenicity for alachlor. The commenter states that the mechanism is nongenotoxic and hormonally mediated. The commenter argues that the mechanism exhibits a threshold and that nasal turbinate tumors in particular are not relevant to humans.

The Agency acknowledges the mechanism of carcinogenicity may be hormonally mediated. However, the mechanism does not alter the fact that the tumors are relevant to potential carcinogenesis in man. Mechanism of tumor development relates to the appropriate model by which cancer risk is calculated. However, mechanism has no impact on the determination of carcinogenicity hazard. In determining cancer classification, EPA does not assume that the specific types of tumors seen in animals will develop in humans. However, EPA believes that the development of tumors, such as nasal turbinates, in animals demonstrates the potential for tumor development in humans.

The same commenter states that two epidemiology studies, not cited by EPA in the proposed rule, have been conducted on alachlor manufacturing workers. The commenter contends that

neither study indicates an increase in tumors in humans due to exposure to alachlor. The commenter believes that these studies provide important additional evidence indicating that the tumors produced in rats by alachlor are not produced in humans and should have been considered by the Agency.

Epidemiological studies are used by the Agency in the overall evaluation of the carcinogenic potential of a chemical, along with other evidence. However, the studies cited by the commenter are based on a small sample size. Studies of this type cannot verify the levels and duration of exposure and represent results from a heterogeneous population. In addition, one of the two studies apparently only focused on tumors resulting in death of the study subjects and may reflect an under estimation of tumor incidence. Therefore, in the face of evidence of carcinogenicity in two adequately performed bioassays in two species, the epidemiology data, although pertinent, do not negate the importance of the animal data in the studies relied upon in the proposed rule.

EPA reaffirms that there is sufficient evidence for listing alachlor on the EPCRA section 313 list pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for this chemical. Therefore, EPA is finalizing the addition of alachlor on the EPCRA section 313 list.

3. *Ametryn*. Ciba-Geigy Corporation objects to listing ametryn under EPCRA section 313 on the basis of liver effects, stating that hepatotoxicity was observed only at high dose levels (100 and 500 mg/kg/day) in subchronic studies.

The Agency believes that the LOEL of 100 mg/kg/day is sufficiently low given the seriousness of the effect (hepatic toxicity) to justify listing on the EPCRA section 313 list. Thus, EPA reaffirms that there is sufficient evidence for listing ametryn on the EPCRA section 313 list pursuant to EPCRA section 313(d)(2)(B) based on the available hepatotoxicity data for this chemical, and pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data. Therefore, EPA is finalizing the addition of ametryn on the EPCRA section 313 list.

4. *Amitraz*. Nor-Am Chemical Company states that in the 2-year beagle dog feeding study cited in the proposed rule, contrary to EPA's conclusions, the only effects seen in the high dose (1.0 mg/kg/day) group were a small but insignificant increase in blood glucose and in one animal slight hypothermia during weeks 52 and 79.

EPA disagrees with the commenter. EPA has re-evaluated this study, and

determined that in this study amitraz induced significant changes in blood chemistry (increased blood glucose). Hypothermia occurred not only at the times noted by the commenter, but also on days 1 and 2, and in one dog 3 hours after dosing, which returned to normal within 24 hours, at the 1.0 mg/kg/day level, the LOEL. As noted in the proposed rule, these findings were supported by similar results obtained in a 90-day feeding study in dogs cited in the proposed rule.

Nor-Am disagrees with the Agency's conclusion that the NOAEL for fetotoxicity was 5 mg/kg/day in the 3-generation rat reproduction study cited in the proposed rule. The commenter believes that while there was a slight decrease in the mean litter size at birth in the 20 mg/kg/day dose group and decreased pup viability in the 5 and 20 mg/kg/day dose groups post partum, there was no direct evidence of fetotoxicity. Nor-Am states that the effect on litter size was only significant in the third generation animals at 5 mg/kg/day, and may have been due to an effect on lactation.

EPA's reanalysis of this data indicates that there was a decrease in litter size and pup survival at 5 mg/kg/day in all 3 generations and a slight reduction in pup weight in the F₁ and F₂ generations. Thus, there was direct evidence of fetotoxicity.

The commenter contends that the rabbit teratology study reported by the Agency in the proposed rule was considered by EPA to be invalid (i.e., significantly flawed) due to high abortion rates in all groups, inadequately small group sizes, and lack of assessment of fetuses. The commenter argues that the low incidence of anomalies upon which the NOAEL of 1 mg/kg/day was based were within historical control ranges and failed to show any clear dose-related effect. The commenter claims that a subsequent study, not cited by EPA in the proposed rule, revealed no effects on fetal morphology at doses up to 12 mg/kg/day while maternal toxicity was found at 3 mg/kg/day and above; no NOEL could be established. The commenter claims that this subsequent study, not cited by EPA in the proposed rule, should have been considered by EPA.

EPA disagrees. The rabbit teratology study cited by EPA in the proposed rule was never declared by EPA to be invalid (i.e., seriously flawed). Upon reanalysis of the rabbit teratology study, EPA determined that although this study does not fully satisfy the guidelines for study conduct under FIFRA, it is sufficient for the purposes of hazard assessment, with a NOEL and LOEL for

maternal and developmental toxicity of 5 and 25 mg/kg/day, respectively. As described in the proposed rule, at 25 mg/kg/day, the following effects were seen: Decreased litter size and increased pre and post-implantation losses, decreased maternal body weight gain, and increased abortions. The high abortion rate is indicative of maternal toxicity. Although the abortion rates were higher than the control, enough animals remained at sacrifice to evaluate the toxicity potential of this chemical, and to support the finding that amitraz can reasonably be anticipated to cause developmental toxicity.

The subsequent study cited by the commenter was also considered by EPA. This study also does not fully satisfy the guidelines for study conduct under FIFRA. Although the fetotoxic effects observed in the initial study (cited in the proposed rule) were not reproduced in the subsequent study referred to by the commenter and not cited in the proposed rule, this does not invalidate the results obtained in the initial study. Both studies were considered by EPA in determining the developmental toxicity of amitraz.

EPA reaffirms that there is sufficient evidence for listing amitraz on the EPCRA section 313 list pursuant to EPCRA section 313(d)(2)(B) based on the chronic toxicity and developmental toxicity data for this chemical. Therefore, EPA is finalizing the addition of amitraz on the EPCRA section 313 list.

5. *Atrazine*. Ciba-Geigy Corporation objects to the listing of atrazine under EPCRA section 313 based on increased incidence of mammary tumors in female Sprague-Dawley rats because the commenter contends that this tumor type is not indicative of potential carcinogenicity in humans. The commenter states that the effect is species (rat) and strain (Sprague-Dawley) specific. Further, the commenter states epidemiology data from Ciba-Geigy manufacturing and use indicate no evidence of carcinogenicity in a human population exposed for up to 30 years. Ciba-Geigy did not provide EPA with a copy of this study but did discuss the results in their comments.

While epidemiology data are considered in the weight of the evidence for carcinogenicity, the current classification is based upon a positive finding in a well conducted animal study as described in the *Risk Assessment Guidelines of 1986* (Ref. 5). Atrazine has been classified as a category C chemical by EPA's OPP Carcinogenicity Peer Review Committee and the Scientific Advisory Panel (EPA,

1988). The use of mammary tumor data for hazard assessment purposes, even when only one strain of test animal has been demonstrated to be positive, is consistent with current Agency policy. The Agency considers the cancer classification to be sufficient basis for listing of atrazine.

EPA reaffirms that there is sufficient evidence for listing atrazine on the EPCRA section 313 list pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data. Therefore, EPA is finalizing the addition of atrazine on the EPCRA section 313 list.

6. *Bendiocarb*. Nor-Am Chemical Company states that bendiocarb does not meet the criteria of EPCRA section 313(d)(2)(C) due to its environmental fate. The commenter alleges that it has been shown not to accumulate in soil, water, or plants and has a relatively short half-life (a few days). Nor-Am Chemical Company also contends that bendiocarb is rapidly broken down by hydrolysis to a biologically inactive product. As a result, the commenter states that there is no clear evidence of adverse effects on the environment associated with bendiocarb.

EPA disagrees that the environmental fate of bendiocarb will negate the chemical's ecological toxicity. EPA believes that the environmental fate factors presented by the commenter may reduce but do not eliminate the potential for adverse effects on aquatic organisms and birds because the chemical induces environmental toxicity at low dose levels. Thus, EPA believes that the chemical can reasonably be anticipated to cause a significant adverse effect on the environment.

EPA reaffirms that there is sufficient evidence for listing bendiocarb on the EPCRA section 313 list pursuant to EPCRA section 313(d)(2)(B) based on neurological toxicity data for this chemical, and pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data. Therefore, EPA is finalizing the addition of bendiocarb on the EPCRA section 313 list.

7. *Bifenthrin*. FMC Corporation does not support the addition of bifenthrin under EPCRA section 313 because "EPA overstates the neurological and [developmental effects] of bifenthrin. The neurological effects to which EPA referred were tremors or twitching, neurological signs that did not persist for the entire duration of the studies." EPA agrees with the commenter regarding the developmental toxicity potential or lack thereof, but disagrees with the commenter regarding the

neurological hazards. In addition to the tremors or twitching effects cited by the commenter, more severe symptoms, including clonic convulsions and death, occur in the studies referred to by the commenters that are cited in the proposed rule, at dose levels only slightly higher than those causing slight or occasional tremors and/or twitching. In a rat developmental toxicity study by gavage, cited in the proposed rule, the maternal LOEL based on tremors was 2 mg/kg/day; the NOEL was 1 mg/kg/day. The MTD of 2 mg/kg/day was established on the basis of findings in a rat pilot study (included as part of the chronic rat study cited in the proposed rule) in which there were 3 deaths out of 10 animals at 2.5 mg/kg/day. With regard to the comment concerning the transitory nature of the effects, although they may be transitory in nature, this does not diminish the significance of the adverse effects. In particular, neurotoxic effects leading to convulsion may result in more permanent, underlying damage which is not reversible upon cessation of immediate signs and symptoms. Therefore, the Agency concludes that the neurological effects due to bifenthrin are of sufficient seriousness to warrant listing.

EPA reaffirms that there is sufficient evidence for listing bifenthrin on the EPCRA section 313 list pursuant to EPCRA section 313(d)(2)(B) based on the available neurological toxicity data, and pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data. Therefore, EPA is finalizing the addition of bifenthrin on the EPCRA section 313 list.

8. *Bromine*. Great Lakes Chemical Corporation and Albemarle Corporation believe that bromine does not meet the listing criteria of EPCRA section 313. They contend that the Agency has failed to show that chronic exposure to bromine causes serious or irreversible effects. They also contend that the time-weighted average (TWA) of 0.1 part per million (ppm) established by the National Institute of Occupational Safety and Health (NIOSH) will protect against the acute effects of exposure. They believe, therefore, that the addition of bromine to the EPCRA section 313 list should not be finalized.

NIOSH established the TWA for bromine for acute effects. However, the Agency is not listing bromine on the EPCRA section 313 list on the basis of its acute effects but on the basis of the adverse effects it induces after chronic exposure. These effects include functional neurologic effects and abnormalities in respiratory and endocrine systems. In humans, chronic

exposure to bromine can cause severe irritation of the skin, mucous membranes and respiratory tract, gastroenteritis, and death. This severe irritation which can lead to death through either, or both, respiratory or gastroenteric irritation is the primary endpoint of concern although neurologic signs and symptoms which include dizziness, headache, and "feelings of oppression" along with other functional disturbances of the central nervous system (CNS) may also occur after exposure to bromine.

EPA reaffirms that there is sufficient evidence for listing bromine on the EPCRA section 313 list pursuant to EPCRA section 313(d)(2)(B) based on the available chronic toxicity data for this chemical. Therefore, EPA is finalizing the addition of bromine on the EPCRA section 313 list.

9. *2-Bromo-2-nitropropane-1,3-diol (Bronopol)*. Boots Microcheck contends that 2-bromo-2-nitropropane-1,3-diol presents only a moderate acute hazard, but does not present a chronic hazard. Therefore, the commenter concludes that the compound should not be listed under EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B).

Although the Agency agrees with the commenter that 2-bromo-2-nitropropane-1,3-diol presents a moderate acute hazard, EPA does not agree that the chemical is not a chronic toxicant. The effects noted in both acute and chronic studies, cited in the proposed rule, indicate irritation due to exposure to the compound. However, differing expressions of irritation are obtained depending upon the level of material to which the test animals were exposed and the duration of exposure. In the acute studies cited in the proposed rule, the acute gastric effects were seen at relatively high doses. In the chronic studies, cited in the proposed rule, the effects, described below, were noted following repeated oral exposure to lower doses of 2-bromo-2-nitropropane-1,3-diol. The NOEL for chronic oral exposure in rats was 10 mg/kg/day, with effects including lesions of the stomach mucosa, ulceration, raised areas and excrescences. In a 13-week study in rats cited in the proposed rule, effects included inflammation, epithelial hyperplasia and hyperkeratosis, and congested vessels of the mucosa of the G.I. tract. The chronic studies cited in the proposed rule show that irritation was caused by a repeated number of low doses. In these chronic studies multiple doses were required before irritation occurred. Further, the type of irritation caused by acute and chronic exposure are different. Therefore, the irritation due to chronic

exposure to 2-bromo-2-nitropropane-1,3-diol is distinguishable from that caused by acute exposure. EPA believes that the effects observed in the longer term studies are serious and potentially irreversible.

EPA reaffirms that there is sufficient evidence for listing 2-bromo-2-nitropropane-1,3-diol on the EPCRA section 313 list pursuant to EPCRA section 313(d)(2)(B) based on the available chronic toxicity data for this chemical. Therefore, EPA is finalizing the listing of 2-bromo-2-nitropropane-1,3-diol on the EPCRA section 313 list.

10. *Carboxin*. Zeneca Incorporated and Uniroyal Chemical oppose the listing of carboxin. The commenters claim that the effect of renal toxicity noted by EPA in the proposed rule was seen only in rat feeding studies and not in a chronic dog feeding study. Thus, they claim it appears to be a species-specific effect that may not be relevant to man.

EPA disagrees with the conclusions of the commenters. Because direct human testing is generally unavailable, animals are commonly accepted as surrogates for toxicity testing to predict potential hazard(s) to humans. Exceptions occur only in a few rare cases where effects have been determined to be species-specific (e.g., $\alpha 2\mu$ -globulin). It should be noted that the actual number of species tested with carboxin is limited and, therefore, it is premature to state that the renal toxicity of carboxin is species-specific. Significantly, the commenters did not provide any additional evidence to support their contention that the renal toxicity is species-specific. EPA uses information from the most sensitive species to evaluate potential human hazard(s), as a conservative assumption.

EPA reaffirms that there is sufficient evidence for adding carboxin on the EPCRA section 313 list pursuant to EPCRA section 313(d)(2)(B) based on the available renal toxicity data for this chemical. Therefore, EPA is finalizing the listing of carboxin on the EPCRA section 313 list.

11. *1-(3-Chloroallyl)-3,5,7-triaza-1-azoniaadamantane chloride*. Dow Chemical Company notes that, in the dog study cited in the proposed rule, the test material was administered in gelatin capsules due to problems with palatability. They argue that this mode of administration is unusual and introduces the confounding factor of what is in essence a bolus administration (given all at one time) of the chemical, and results in an artificially lowered NOEL.

The Agency does not agree that this mode of administration is unusual. EPA

frequently reviews dog studies in which the test material is administered by capsule. In addition, dog studies rarely permit ad libitum feeding as used in rat studies, even when dietary incorporation is the means of dose administration. Dogs generally receive a measured amount of food that they rapidly consume. Therefore, bolus administration closely approximates actual behavior in dogs. The concern that capsule administration produces an apparently altered response is not a confounding factor in the study cited in the proposed rule; and therefore the reported NOEL does not need to be raised as suggested by the commenter.

The same commenter contends that the effects used as a basis for listing occurred only in dogs and only in a single study, and, therefore, are not relevant to humans.

Because direct human testing is generally unavailable, animals are commonly accepted in the scientific and regulatory communities as surrogates for toxicity testing to predict potential hazard to humans, except in a few rare cases where effects have been determined to be species-specific (e.g., $\alpha 2\mu$ -globulin). In the interest of being protective, EPA uses information from the most sensitive species to evaluate potential human hazard. In addition, results demonstrated in a single well-conducted study are sufficient and can serve as a basis for listing on the section 313 list.

The same commenter states that the LOEL in the study was based upon a slight, reversible effect in the liver of a single animal. The study, the commenter argues, should have been considered in toto rather than relying on a single effect. The commenter implies that EPA should have set the LOEL at a higher dose.

The commenter is incorrect. The LOEL of 15 mg/kg/day is correct. This LOEL was based upon obliterative vasculitis and perivasculitis in one animal. However, these effects are not commonly seen in dogs, yet in the study cited in the proposed rule, they occurred in seven of eight dogs at 30 mg/kg/day, the dose next highest to the LOEL. EPA considers the effects seen in this study to be serious effects.

EPA reaffirms that there is sufficient evidence for listing 1-(3-chloroallyl)-3,5,7-triaza-1-azoniaadamantane chloride on the EPCRA section 313 list pursuant to EPCRA section 313(d)(2)(B) based on the available chronic toxicity data for this chemical. Therefore, EPA is finalizing the addition of 1-(3-chloroallyl)-3,5,7-triaza-1-azoniaadamantane chloride on the EPCRA section 313 list.

12. *Chlorosilanes*. Silicones Environmental Health and Safety Council and General Electric oppose the listing of the six chlorosilanes that were proposed for addition (dichloromethylphenylsilane, dimethyldichlorosilane, methyltrichlorosilane, trichloroethylsilane, trichlorophenylsilane, and trimethylchlorosilane) arguing that they undergo rapid hydrolysis and are not expected to be found in the atmosphere in appreciable concentrations. The commenters further state that EPA estimated conditions in its exposure assessment that greatly exceed actual conditions.

Based on these comments, EPA conducted revised exposure assessments for each of the chlorosilanes. These revisions support EPA's initial finding that dimethyldichlorosilane, methyltrichlorosilane, and trimethylchlorosilane can reasonably be anticipated to be present at facility boundaries in concentration levels that would cause a significant adverse effect. EPA believes that the exposure assessments were based on reasonable release estimates and reasonable worst-case concentration modeling. Details of this analysis are provided in the *Response to Comment Document* (Ref. 14). Thus EPA reaffirms that there is sufficient evidence to list dimethyldichlorosilane, methyltrichlorosilane, and trimethylchlorosilane on the EPCRA section 313 list pursuant to EPCRA section 313(d)(2)(A). Therefore, EPA is finalizing the listings for dimethyldichlorosilane, methyltrichlorosilane, and trimethylchlorosilane on the EPCRA section 313 list.

The revised exposure assessments for dichloromethylphenylsilane, trichloroethylsilane, and trichlorophenylsilane, however, indicate that these chemicals are not individually present at facility boundaries in concentration levels that would cause a significant adverse effect. However, two or more of these chemicals are usually produced together and as a category are reasonably anticipated to be present at facility boundaries in concentration levels that would cause a significant adverse effect. Therefore, EPA is deferring the individual listings of these three chemicals for consideration as a category possibly to be added at a later date.

13. *Crotonaldehyde*. Eastman Chemical and Monsanto believe that crotonaldehyde should not be added to

the EPCRA section 313 list because of inadequate data on human health. Furthermore, they contend that crotonaldehyde does not meet the criteria for listing as a carcinogen as put forth in the *Risk Assessment Guidelines for Carcinogen Risk* (Ref. 4) because it was tested in a single sex, single species experiment. The commenters further believe that EPA's statement that crotonaldehyde did not induce tumors at the high dose, because at that high dose crotonaldehyde is cytotoxic, is a contention which is not supported by scientific evidence. They believe that overall the weight of evidence for carcinogenicity, including reactivity and mutagenicity, is insufficient to support listing.

EPA agrees that the human carcinogenicity data are inadequate but feels that the available animal data are adequate to support a concern for carcinogenicity. The Agency accepts the single-sex, single species testing of crotonaldehyde as being sufficient for listing because these data are supported by strong evidence of mutagenicity in *Salmonella typhimurium*; a statistically significant increase in the number of both benign and malignant tumors in low dose animals and induced altered liver foci but not tumor formation in the high dose group. Crotonaldehyde is known to be severely cytotoxic with the capacity to induce cell death and alter cellular macromolecules. It caused gross degeneration, chromosome breakage and reciprocal translocations in *Drosophila melanogaster* and gross degeneration and polyploidy in all stages of spermatogenesis in mouse seminiferous tubules thus showing that it has ample ability to interact with cellular DNA and cause severe disruption in chromosome structure and cellular integrity. It is logical to assume that if crotonaldehyde is capable of such damage in the mammalian testis which is protected by the blood/testis barrier, it can also cause severe toxicity and cell death in the liver which has no such protection from toxic agents. Absent evidence to the contrary, which the commenter did not provide, EPA continues to believe that failure to observe tumor formation is due to cell death before tumors could develop. Based on these findings, the Agency believes that the weight of evidence for crotonaldehyde is sufficient for listing. EPA reaffirms that there is sufficient evidence for listing crotonaldehyde on the EPCRA section 313 list pursuant to EPCRA section 313(d)(2)(B) based on available carcinogenicity and mutagenicity data for this chemical. Therefore, EPA is finalizing the addition of

crotonaldehyde on the EPCRA section 313 list.

14. *Cycloate*. Zeneca Incorporated contends that in the 3-generation rat feeding study, cited in the proposed rule as being of unknown duration, the distended myelin sheath demyelination and nerve fiber loss at the LOEL of 3.0 mg/kg/day occurred only after extensive exposure and as such would not be relevant to a toxic release type of short exposure.

The effects described in this study are considered to be both serious and irreversible. Adverse effects that are induced by a chemical after repeated long-term exposures and are a valid basis for listing under EPCRA section 313.

The same commenter states that the 3-generation rat reproduction study cited in the proposed rule was replaced by a more recent (1990) 2-generation rat reproduction study, also cited in the proposed rule, in which the toxic effects on pup survival (LOEL of 50 mg/kg/day) and pup body weight (LOEL of 20 mg/kg/day) occurred at doses which were maternally toxic as well.

EPA considered both studies in its evaluation of cycloate. As described in unit IV.E. of this preamble, developmental effects seen in developing organisms are considered to be adverse whether or not they occur at doses that are also maternally toxic.

EPA reaffirms that there is sufficient evidence for listing cycloate on the EPCRA section 313 list pursuant to EPCRA section 313(d)(2)(B) based on the available neurological and developmental toxicity data. Therefore, EPA is finalizing the addition of cycloate on the EPCRA section 313 list.

15. *Cyclohexanol*. Monsanto opposes the listing of cyclohexanol because concentrations that led to tremors, central nervous system depression, lethargy, or hypothermia in rabbits, as cited in the proposed rule, are above the level of MED that EPA identified in the *Draft Hazard Assessment Guidelines* (Ref. 11) as high priority or moderate priority. Furthermore, the concentrations that led to reproductive impacts in rats were above the MED level of high priority. In addition, Monsanto states that the Industrial Health Foundation submitted to EPA's TSCA office the results of a 2-generation reproduction study demonstrating a NOEL of 500 ppm in air which should have been considered. The commenter claims that EPA has also not demonstrated that the effects mentioned, or concentrations at which they occurred, were serious or irreversible.

EPA agrees that the concentrations that led to tremors, central nervous system depression, lethargy, or hypothermia in rabbits are above the level of MED that EPA identifies in the *Draft Hazard Assessment Guidelines* (Ref. 11) as high priority for listing. However, while the 2,500 mg/kg/day dermal exposure is above the moderate priority MED guideline, the 997 ppm (438 mg/kg/day) is within this category. In addition to the neurotoxicity effects, as cited in the proposed rule, cyclohexanol also induces renal, hepatic, and myocardial effects at moderate dose levels (for example, inhalation of 0.59 mg/L of cyclohexanol induced degenerative changes in the livers and kidneys of rabbits). EPA considers these effects to be serious. In this case, based on a weight-of-evidence approach, EPA believes that cyclohexanol presents a sufficient hazard to warrant listing under EPCRA section 313 even though the reported values for neurotoxicity effects are in excess of the MEDs placing a chemical in the high priority grouping.

EPA disagrees that the concentrations that led to reproductive impacts in rats and gerbils (15 mg/kg) as described in the proposed rule are above the MED range for high priority listing. EPA reiterates the overall reproductive toxicity of this chemical, based on a weight-of-evidence, supports the addition of cyclohexanol to the EPCRA section 313 list.

The chemical tested in the 2-generation reproduction study submitted to the Agency by the Industrial Health Foundation, cited by the commenter, was cyclohexanone not cyclohexanol as claimed by the commenter.

EPA reaffirms that there is sufficient evidence for listing cyclohexanol pursuant to EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available chronic neurological, hepatic, renal, myocardial, and reproductive toxicity data for this chemical. Therefore, EPA is finalizing the addition of cyclohexanol on the EPCRA section 313 list.

16. *Cyhalothrin*. Zeneca Incorporated contends that the neurotoxicity signs observed in the 6-month and 1-year dog studies cited in the proposed rule occurred at doses that were "otherwise toxic as well" and do not provide any evidence of a specific neurotoxicity. Zeneca Incorporated implies that the presence of "otherwise toxic" signs reduces the significance of the neurotoxicity observed in the cited study.

The phrase "otherwise toxic as well" was not defined by the commenter. The

clinical signs of neurotoxicity observed in the dogs at 3.5 mg/kg/day (ataxia, muscle tremors, and convulsions in the 1-year study cited in the proposed rule) and at 10 mg/kg/day (unsteadiness and trembling in the 6-month study cited in the proposed rule) are considered by EPA to be evidence of physiological neurotoxicity. Although there were no pathologic changes in the nervous tissue, EPA considers these effects to be serious because they often precede pathologic neurotoxicity. With the exception of liquid feces, there were no reported toxic findings other than those related to neurotoxicity.

EPA reaffirms that there is sufficient evidence for listing cyhalothrin on the EPCRA section 313 list pursuant to EPCRA section 313(d)(2)(B) based on the available neurological toxicity data. Therefore, EPA is finalizing the addition of cyhalothrin on the EPCRA section 313 list.

17. *Desmedipham*. Nor-Am Chemical states that methemoglobin formation, which is cited by EPA as the basis for listing, is an entirely reversible effect which occurs only after prolonged and consistent exposure. Therefore, the commenter concludes that this finding, by itself, should not be used.

Based on the 90-day dog study, cited in the proposed rule, EPA considers 150 ppm to be a NOAEL. Methemoglobin values were only minimally higher than control levels and were not associated with an increase in Heinz bodies. In the 1-year dog feeding study, after 13 weeks treatment at 300 ppm, methemoglobin was seen associated with histopathological changes (hemosiderin and hemopoiesis). While methemoglobinemia may be a reversible effect, it is nevertheless a serious effect, and in some cases irreversible damage may occur as a result of methemoglobinemia. Methemoglobinemia interferes with the oxygenating capacity of blood resulting in an undersupply of oxygen to the tissues. Therefore, methemoglobinemia is a toxic effect and not simply an indicator of exposure to desmedipham as concluded by the commenter.

Therefore, EPA reaffirms that there is sufficient evidence for listing desmedipham on the EPCRA section 313 list pursuant to EPCRA section 313(d)(2)(B) based on the available hematological toxicity data. Therefore, EPA is finalizing the addition of desmedipham on the EPCRA section 313 list.

18. *2,2-Dibromo-3-nitropropionamide*. Dow Chemical Company and Rohm Haas state that the corrosivity and irritancy of the 2,2-dibromo-3-nitropropionamide

(DBNPA) solutions to the esophagus, pharynx, trachea, and lungs led to development of dyspnea in rats. The commenters imply that the dyspnea in rats should be discounted because it was caused by the method of administration rather than the toxicity of the chemical.

The Agency agrees that the dyspnea observed in the 4-week and 13-week rat gavage studies cited in the proposed rule may have been due to severe irritation of the trachea and lungs from accidental or incidental delivery of small amounts of the DBNPA dosing solutions into the larynx, pharynx, trachea, and/or lungs during the procedure. However, this suggestion of possible cause can be neither refuted nor confirmed based upon the available data. Dyspnea is the basis for the LOEL in the study. One of the commenters agrees that DBNPA is corrosive, particularly to the eyes and, at the least, is severely irritating to the respiratory tract. This is consistent with the effects observed in the two subject studies. The Agency considers the finding of dyspnea in the 4- and 13-week studies to be of sufficient seriousness to warrant listing on the EPCRA section 313 list.

EPA reaffirms that there is sufficient evidence for listing 2,2-dibromo-3-nitropropionamide on the EPCRA section 313 list pursuant to EPCRA section 313(d)(2)(B) based on the available chronic respiratory toxicity data. Therefore, EPA is finalizing the addition of 2,2-dibromo-3-nitropropionamide on the EPCRA section 313 list.

19. *Diclofop-methyl*. Nor-Am Chemical and Hoechst-Celanese contend that EPA interpreted the doses administered by gavage as diet concentrations (ppm) in the rat teratology study cited in the proposed rule. One commenter states the Agency should provide clarifications concerning "mortality" of the pups and the calculation of the actual test substance intake at different stages during the in-life phases during development in the 3-generation rat reproduction study.

The commenter is correct in stating that the Agency erred in interpreting gavage doses as ppm in the rat teratology study. However, EPA still believes that the doses at which adverse effects occur are sufficiently low and the adverse effects reported are of sufficient seriousness to warrant listing. The developmental NOEL is 10 mg/kg/day and the LOEL is 32 mg/kg/day based on an increased incidence of a number of variations and malformations, as described in the proposed rule. While the maternal effects on body weight and food consumption at 32 mg/kg/day are

transient and reversible, some of the developmental effects at this dose are irreversible. In the 3-generation rat reproduction study cited in the proposed rule, a decrease in pups born alive in the F_{1a} , reduced pup weights (F_{1a} and $2a$) and general retardation of physical development (F_{1a} and $2a$) was noted in offspring at 100 ppm (5 mg/kg/day). The commenter considers the LOEL for this study to be 6.7 mg/kg/day. This dose resulted in decreased parental food consumption and body weight and there were no post partum pup mortalities. Additionally, there were no effects on fertility at the LOEL at any time during the three generations.

The commenters further stated that EPA should "consider the validity" of the 30-day rat study cited in the proposed rule because heart, kidney, and adrenal weights were increased only at doses with no histopathological correlates and were due to the pharmacodynamic lipid metabolism of the test material by the liver.

The increased relative heart, liver, and kidney weights at 80 ppm (4 mg/kg/day) in the 30-day rat feeding study is further substantiated by a recent 90-day rat feeding study cited in the proposed rule with a LOEL of 80 ppm and a NOEL of 20 ppm (1 mg/kg/day). In the recent 90-day study cited in the proposed rule, absolute and relative liver and kidney weight was increased in males and relative liver and kidney weight was increased in females at 80 ppm. These increased organ weights are evidence of a compound-related effect. The Agency interprets Hoechst-Celanese's own statements regarding the 30-day rat feeding study that "increased liver weights and centrilobular enlargement of hepatic cells at dietary concentrations of 80 ppm and higher" as evidence of toxicity.

Hoechst-Celanese also contends that the effects in the renal cortex observed in the 90-day dog study cited in the proposed rule at 250 ppm (15 and 13.4 mg/kg/day in males and females, respectively) did not occur at the highest concentration tested in the 1-year dog study (80 ppm, 4-5 mg/kg/day) indicating that the finding in the 90-day study was not test substance related.

EPA believes that the effects occurring in the renal cortex in the 90-day dog study at 13 to 15 mg/kg/day may not have appeared in the 1-year dog study, since the highest dose tested was 4-5 mg/kg/day. If higher doses were employed in the 1-year study, then renal effects could possibly have occurred. However, the results of the 1-year study do not negate the 90-day results, since the dose levels used in the 90-day study were so much higher.

Hoechst-Celanese also states that the Agency used an invalid (flawed) reproductive toxicity study to support the listing. The commenter indicates that the study was compromised by infection of the rat colony with RCV/SDA virus. They further state that another reproductive toxicity study, which EPA did not cite in the proposed rule, should have been evaluated in which the fetotoxic NOEL was 30 mg/kg/day instead of greater than 5 mg/kg/day as in the original study.

The Agency does not find the original study to be invalid. The data were considered to be valid for regulatory purposes. In addition, the Agency found the fetotoxic NOEL in the study referred to by the commenter, not cited by EPA in the proposed rule, to be 5 mg/kg/day, not 30 mg/kg/day as stated by the commenter.

EPA reaffirms that there is sufficient evidence for listing diclofop-methyl on the EPCRA section 313 list pursuant to EPCRA section 313(d)(2)(B) based on the available developmental, hepatic, and renal toxicity data. Therefore, EPA is finalizing the addition of diclofop-methyl on the EPCRA section 313 list.

20. *Diisocyanates*. EPA originally proposed to list three diisocyanates (hexamethylene-1,6-diisocyanate, isophorone diisocyanate, and 1,1-methylene bis(4-isocyanatocyclohexane)) on the basis of acute toxicity pursuant to EPCRA section 313(d)(2)(A). As an alternative, EPA proposed to create a delimited diisocyanates category containing these 3 diisocyanates and 17 other diisocyanates based on chronic pulmonary irritation pursuant to EPCRA section 313(d)(2)(B). EPA is finalizing addition of the delimited diisocyanate category based on chronic pulmonary toxicity and therefore has not addressed comments concerning the acute toxicity of any of the diisocyanates. EPA believes that diisocyanates are best added as a category because the members of this category are structurally similar (i.e., each contains the diisocyanate functionality), they induce a similar toxic effect (chronic pulmonary irritation), and their toxicity is due to the diisocyanate portion of the molecule common to all members.

Chemical Manufacturers Association Hexamethylene-1,6-Diisocyanate Panel, Dow Chemical Company, Monsanto, Olin Chemicals, Sealed Air Corporation, Huls America Incorporated, and the Diisocyanates Panel of the Chemical Manufacturers Association oppose EPA's alternative proposal to create a diisocyanate category and believe that individual diisocyanates should be evaluated and included on the EPCRA section 313 list only if the diisocyanate

independently satisfies the statutory listing criteria. The commenters state that in adding a broad category of diisocyanates, EPA ignores its statutory mandate to evaluate the individual toxicity of each chemical and to evaluate the exposure potential to the EPCRA community by each individual chemical. The commenters contend that the category would mislead the public as to the amount and type of toxic chemicals to which communities may be exposed. The commenters contend that data collected in aggregate is confusing and difficult to use or interpret. Commenters state that adding a category of diisocyanates based upon the isocyanate functionality is based on the chronic effects associated with exposures to a limited number of diisocyanates and that this method unjustifiably equates toxicity across an entire class of chemicals that have different properties and effects. Commenters state that diisocyanates encompass a diverse group of chemicals which vary significantly in physical and chemical properties and in potential toxicity. Commenters state that the available evidence on the pulmonary effects or toxicity of individual diisocyanates (toluene diisocyanate, methylenebis(phenylisocyanate), and isophorone diisocyanate) does not support the addition of a diisocyanates category. The commenters also state that EPA has not cited any data to support the assertions that diisocyanates cause these effects. Commenters state that individual diisocyanates have been shown to respond differently in mutagenicity studies and that other toxicological differences would be expected among individual diisocyanates, because of differences in their ability to penetrate membranes, the capacity of organisms to metabolize them, the specific reactivity of the diisocyanate groups, etc. Commenters state that in the proposed rule EPA recognized these differences by stating that some diisocyanates are classified as probable carcinogens and others are not. The Wisconsin Department of Natural Resources supports EPA's alternative proposal to create a diisocyanate category and would prefer this manner of listing to listing each diisocyanate separately.

As discussed in unit IV.C. of the preamble, EPA believes that it is acting reasonably within its discretion in listing a category of chemicals by showing that at least one member of the category meets the listing criteria of EPCRA section 313 and that the other members can reasonably be expected to exhibit the same or similar toxic effect.

EPA believes that the available data on the chronic pulmonary toxicity for several members of the diisocyanates category are sufficient for listing under EPCRA section 313(d)(2)(B). EPA also believes that the diisocyanate moiety, common to all members of the category, is responsible for the observed chronic pulmonary toxicity. Therefore, EPA believes that it is reasonable to anticipate that all members of the diisocyanate category will exhibit chronic pulmonary toxicity and that creating a category of diisocyanates is the most appropriate way to list this class of chemicals. As stated in Unit IV.B. of the preamble, EPA does not believe that it is required to consider exposure for chemicals that are moderately high to highly toxic based on a hazard assessment when determining if a chemical can be added for chronic effects pursuant to EPCRA section 313(d)(2)(B); therefore, EPA is not required to evaluate the exposure potential for the members of the diisocyanates category. EPA believes that, because each member of the diisocyanates category has the same functional groups and can reasonably be anticipated to cause similar toxic effects, the diisocyanates category will not mislead the public as to the amounts and type of chemicals released and will not be confusing to use or interpret.

EPA agrees that the diisocyanates are a diverse group of chemicals which vary in physical and chemical properties. However, EPA also believes that the reactive portion of diisocyanate chemicals is the diisocyanate moiety itself and that the rest of the molecule does not affect the reactivity of this portion of the molecule. EPA stands by its interpretation of the literature, as cited in the proposed rule and background material, on the adverse pulmonary effects of diisocyanates and believes that this information supports the addition of a diisocyanates category. The Agency agrees that structural differences among individual diisocyanates may indeed affect their absorption and metabolism. However, since absorption and metabolism are not necessary for chronic pulmonary irritation to occur, the effect of structural differences upon either absorption or metabolism is not an issue in this case. The Agency agrees with the commenter that there are differences in the carcinogenicity/mutagenicity of toluene diisocyanate, methylenebis(phenylisocyanate), and isophorone diisocyanate and that these differences are most likely the result of the differences in absorption and metabolism. However, since neither of

these endpoints is the basis for listing diisocyanates as a category and since chronic pulmonary irritation can occur without absorption and metabolism taking place, these issues do not affect the Agency's overall concern for diisocyanates or its decision to list them as a category on the EPCRA section 313.

As EPA discussed in the proposed rule, there currently are four other diisocyanates listed on the EPCRA section 313 list, these are:

Toluene-2,4-diisocyanate (000584-84-9)

Toluene-2,6-diisocyanate (000091-08-7)

Toluene diisocyanate (mixed isomers) (026471-62-5)

Methylenebis(phenylisocyanate) (000101-68-8)

EPA is leaving the toluene diisocyanate compounds listed individually. In addition to the effects discussed above, these compounds have been shown to be carcinogenic. EPA believes it is appropriate to continue to individually list carcinogenic diisocyanates because they exhibit a different toxic endpoint than other members of the category.

Methylenebis(phenylisocyanate) has not been shown to be a carcinogen and as EPA discussed in the proposed rule it is being moved into the diisocyanate category.

EPA reaffirms its determination that diisocyanates meet the criteria of EPCRA section 313(d)(2)(B). Therefore, EPA is finalizing the addition of the diisocyanates category that consists of the following chemicals:

1,3-Bis(methylisocyanate)cyclohexane (CAS No. 038661-72-2)

1,4-Bis(methylisocyanate)cyclohexane (CAS No. 010347-54-3)

1,4-Cyclohexane diisocyanate (CAS No. 002556-36-7)

Diethyl-diisocyanatobenzene (CAS No. 134190-37-7)

4,4'-Diisocyanatodiphenyl ether (CAS No. 004128-73-8)

2,4'-Diisocyanatodiphenyl sulfide (CAS No. 075790-87-3)

3,3'-Dimethoxybenzidine-4,4'-diisocyanate (CAS No. 000091-93-0)

3,3'-Dimethyl-4,4'-diphenylene diisocyanate (CAS No. 000091-97-4)

3,3'-Dimethyldiphenylmethane-4,4'-diisocyanate (CAS No. 000139-25-3)

Hexamethylene-1,6-diisocyanate (CAS No. 000822-06-0)

Isophorone diisocyanate (CAS No. 004098-71-0)

Methylenebis(phenylisocyanate) (CAS No. 000101-68-8)

4-Methyldiphenylmethane-3,4-diisocyanate (CAS No. 075790-84-0)

1,1-Methylene bis(4-isocyanatocyclohexane) (CAS No. 005124-30-1)

1,5-Naphthalene diisocyanate (CAS No. 003173-72-6)

1,3-Phenylene diisocyanate (CAS No. 000123-61-5)

1,4-Phenylene diisocyanate (CAS No. 000104-49-4)

Polymeric diphenylmethane diisocyanate (CAS No. 009016-87-9)

2,2,4-Trimethylhexamethylene diisocyanate (CAS No. 016938-22-0)

2,4,4-Trimethylhexamethylene diisocyanate (CAS No. 015646-96-5)

In reassessing the Agency's proposal in light of comments received and other information, it has become clear to EPA that the effect of concern (chronic pulmonary toxicity) is related to the diisocyanate moiety and therefore common to all diisocyanate compounds not just those included in the delimited category finalized in this rule. EPA believes that many other diisocyanates not covered by the category may meet the EPCRA section 313 criteria. Therefore, EPA believes that it may be more appropriate to create a diisocyanates category based on a molecular formula description rather than a more limited category comprised of certain named diisocyanates. However, EPA did not include a molecular formula category option in its proposal and therefore has not given the public an opportunity to comment on such a category. Accordingly, in this action EPA is finalizing the addition of a delimited category consisting of the 20 diisocyanates on which the Agency has received comment and for which the Agency has made a final determination that the chemicals meet the EPCRA section 313 criteria for listing. EPA believes that the chemicals covered by this category represent the majority of diisocyanates in production and that listing the delimited category will provide meaningful data to the public. At a later date, EPA will consider whether a more broad diisocyanates category is warranted and appropriate.

21. *Dimethylamine*. Olin Chemicals does not believe that the data cited by EPA are sufficient to prove that dimethylamine causes "... significant adverse acute human health effects at concentration levels that are reasonably likely to exist beyond facility site boundaries. . . ." The commenter did not substantiate this contention. The commenter requests a more rigorous review of the available data before adding dimethylamine to the EPCRA section 313 list.

The Agency is not proposing to list dimethylamine pursuant to section 313(d)(2)(A), therefore the Agency does not have to show that the chemical causes "... significant adverse acute

human health effects at concentration levels that are reasonably likely to exist beyond facility site boundaries. . . .” EPA disagrees that the data cited are insufficient to prove that dimethylamine is likely to cause significant human health effects. As articulated in the proposed rule, dimethylamine is corrosive to mucous membranes, the eyes and respiratory tract. Chronic exposure results in dose-related lesions in the respiratory and olfactory epithelium and is associated with centrilobular fatty degeneration and necrosis of parenchymal cells after inhalation exposure for 18 to 20 weeks. Rats exposed to oral doses as low as 0.035 mg/kg for 8 months showed neurological effects including changes in conditioned reflexes while single doses of 240 to 260 mg/kg caused excitement and muscle weakness in mice, rats, guinea pigs, and rabbits. Dimethylamine is corrosive to the respiratory tract, exhibits hepatotoxicity and is neurotoxic. EPA reaffirms that there is sufficient evidence to list dimethylamine on the EPCRA section 313 list pursuant to EPCRA section 313(d)(2)(B) based on the available chronic respiratory, hepatic, and neurological toxicity data for this chemical. Therefore, EPA is finalizing the addition of dimethylamine on the EPCRA section 313 list.

22. *2,6-Dimethylphenol*. One commenter, General Electric, states that the proposed addition of 2,6-dimethylphenol to EPCRA section 313 is based upon a “low confidence” study and a 10-week subchronic study which the ITC found insufficient to evaluate.

The commenter is concerned that EPA is using the same data set in two rule makings; TSCA section 4 and the decision to list on the EPCRA section 313 list. The commenter quotes the ITC finding that the studies are of “low confidence.” The Agency used these data to derive an oral RfD of 0.0006 mg/kg/day. IRIS confidence in the studies is low because of lack of experimental detail. EPA also concedes that the ITC had low confidence in these studies for its purposes which are *risk assessment*. However, EPA believes that these data are sufficient for the purposes of hazard assessment and concludes that these studies when considered together present a sufficient weight of the evidence determination for listing 2,6-dimethylphenol on EPCRA section 313 because 2,6-dimethylphenol causes hepatotoxicity and nephrotoxicity at relatively low dose levels. EPA reaffirms that there is sufficient evidence to list 2,6-dimethylphenol on the EPCRA section 313 list pursuant to EPCRA section 313(d)(2)(B) based on the

available hepatotoxicity and nephrotoxicity data for this chemical. Therefore, EPA is finalizing the addition of 2,6-dimethylphenol on the EPCRA section 313 list.

23. *Dinoseb*. Uniroyal Chemical Company objects to the listing of dinoseb (dinoseb is the trade name for dinitrobutyl phenol) because the sale of dinoseb as a herbicide or insecticide is prohibited and remaining inventories have been used up or disposed. However, the commenter notes that dinitrobutyl phenol continues to be produced and sold for uses not subject to FIFRA (e.g. as an inhibitor in the polymer industry).

EPA believes that the chemical is more properly listed by its common chemical name, dinitrobutyl phenol, rather than its trade name. However, EPA also recognizes that this chemical is well known as dinoseb. Therefore, EPA is finalizing the addition of this chemical as dinitrobutyl phenol (dinoseb).

24. *Disodium cyanodithioimidocarbamate*. Buckman Laboratories International, Incorporated contends that the compound was not teratogenic in either the rat or rabbit studies cited in the proposed rule. Specifically, they contend that skeletal variations and increased resorptions should be considered an artifact (i.e., occurring by chance rather than as a result of treatment), and should not be considered as evidence of developmental toxicity.

EPA disagrees with the commenter. In both the rabbit and rat teratology studies cited in the proposed rule, developmental effects were observed at levels that were above the maternally toxic level (greater than 3 mg/kg for rabbits and greater than 6 mg/kg for rats). Furthermore, the effects observed cannot be considered an artifact, because in rabbits receiving 30 mg/kg, there is a continuation of the effects observed at 10 mg/kg, with an accompanying increase in the severity of the developmental findings. This shows that the effects are related to the dose received and do not occur by chance.

EPA reaffirms that there is sufficient evidence for listing disodium cyanodithioimidocarbonate on the EPCRA section 313 list pursuant to EPCRA section 313(d)(2)(B) based on the available developmental toxicity data. Therefore, EPA is finalizing the listing of disodium cyanodithioimidocarbamate on the EPCRA section 313 list.

25. *Ethyl dipropylthiocarbamate (EPTC)*. Zeneca Incorporated states that in the study cited by EPA in the

proposed rule in which rats were orally administered the test compound for 2 years, brain cholinesterase reductions were slight and only occurred at 120 mg/kg/day, not 15 mg/kg/day. The commenter claims that neuromuscular changes occurred only after extended exposure, and are not relevant to listing on the EPCRA section 313 list.

The commenter is referring to two studies cited in the proposed rule. A 2-year rat feeding study established a NOEL of 5 mg/kg/day and a systemic LOEL of 25 mg/kg/day with neuromuscular atrophy/degeneration and decreased body weight gains as the findings. At 125 mg/kg/day, the effects included chronic myocarditis, cataracts, increased SGOT and decreased RBC cholinesterase (ChE) activity. The neuromuscular and cardiac changes are serious and potentially irreversible effects. The second study is a 3-month feeding study in Sprague-Dawley rats. Although this study was not identified in the proposed rule, the results of the study were described. The systemic NOEL in this study was 3 mg/kg/day, and the LOEL was 15 mg/kg/day. The effects seen included increase in cardiomyopathy and decreased weight gain and food consumption. As noted by the commenter, brain ChE depression in this study in females was slight and occurred at 120 mg/kg/day and, taken by itself, is not sufficient for listing, however, when considered with other effects in a weight-of-evidence approach, this endpoint is supportive of listing. The commenter further states that the 2-year dietary rat study is old and has been superseded by another study (Ref. 8), in which the NOEL was 25 mg/kg/day.

The Agency agrees with the commenter's comment regarding the replacement of the older study with a newer study, but disagrees with the commenter's NOEL. The Agency's NOEL for this study is 5 mg/kg/day and not 25 mg/kg/day. However, the results of the older study demonstrate that heart effects of EPTC are seen in more than one study.

The commenter further states that in the 2-generation rat reproduction study, cardiomyopathy was observed only in the F^{1A} females and was incidental to treatment. EPA disagrees with this contention. The investigators did not look for this effect in other generations. Thus, there is no reason to conclude that this effect was not manifested in other generations. In addition, this type of adverse effect has been seen in other studies, such as the 2-year rat study and the 3-month rat study discussed above and cited in the proposed rule.

The Agency believes that the cardiopathic finding at 10 mg/kg/day, degenerative cardiomyopathy, is the pivotal finding of toxicological significance for EPTC. EPA believes that this is a serious effect. Therefore, this effect cannot be considered incidental to treatment.

The commenter further states that the neurological effects seen in the study are not relevant to the EPCRA section 313 due to prolonged exposure and the cardiovascular observations occurred at the highest dose tested in the studies cited.

The cardiovascular effects occur after relatively short exposures at doses of 9 mg/kg/day in male rats and 18 mg/kg/day in female rats. These dose levels are sufficiently low and the adverse effects are serious and potentially irreversible. The Agency considers the neurotoxicity due to prolonged exposure to be relevant for purposes of listing on the EPCRA section 313 list. Releases of chemicals that induce adverse effects after prolonged exposure is among the type of information that Congress intended TRI to include.

EPA reaffirms that there is sufficient evidence for listing EPTC on the EPCRA section 313 list pursuant to EPCRA section 313(d)(2)(B) based on the available neurological, cardiovascular, and reproductive toxicity data for this chemical. Therefore, EPA is finalizing the addition of EPTC on the EPCRA section 313 list.

26. *Fenoxaprop-ethyl*. Hoechst-Celanese and Nor-Am Chemical indicate that the chronic interstitial nephritis reported at 80 ppm in the 3-month dog study cited in the proposed rule "was [a] non substance-related, incidental finding since 12/24-months chronic treatment produced no comparable pathogenesis" and that "liver and kidney were not the target organs in dogs; effects were confined to reduced body weight gains at the highest concentration (75 ppm)."

EPA disagrees with the commenters. The dietary levels of fenoxaprop-ethyl in the studies compared by the commenter were 0, 16, 80, and 400 ppm and 0, 3, 15, and 75 ppm for the 3-month and 24-month studies, respectively; both studies are cited in the proposed rule. The microscopic findings in the 3-month study indicated that there was a dose response for chronic interstitial nephritis with inflammatory changes in the medulla and inner cortex. One half of the dogs were affected at 400 ppm, which is much higher than the highest dietary level in the 24-month study (75 ppm). Therefore, the inflammatory changes in the kidneys of treated dogs at 80 and

400 ppm in the 3-month study appear to be related to the ingestion of fenoxaprop-ethyl and, therefore, the kidney appears to be a target organ. The Agency did not cite liver effects in dogs as a basis for listing.

EPA reaffirms that there is sufficient evidence for listing fenoxaprop-ethyl on the EPCRA section 313 list pursuant to EPCRA section 313(d)(2)(B) based on the available renal and developmental toxicity data for this chemical, and pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data. Therefore, EPA is finalizing the addition of fenoxaprop-ethyl on the EPCRA section 313 list.

27. *Fenoxycarb*. Ciba-Geigy Corporation and Miles Incorporated disagree with the listing of fenoxycarb on the EPCRA section 313 list because they believe that the adverse hepatic effects observed in mice and rats (3-month dietary study and 2-year oncogenicity study, both cited in the proposed rule) are not sufficiently serious to support listing. They note that no evidence of neoplastic lesions was reported in the chronic studies. They further state that delayed pinna unfolding in the reproductive toxicity study in rats cited in the proposed rule is a reflection of slower growth only, and therefore should not be used to support listing.

The Agency disagrees that the evidence does not support a finding that section 313(d)(2)(B) are satisfied. The effects in the chronic studies include focal necrosis, changes which are considered by the Agency to be serious and potentially irreversible in nature. The liver effects in the subchronic study demonstrate the progression of changes leading to necrosis in the chronic study. The Agency considers these to be serious adverse effects.

The developmental effects (slight delays in pinna unfolding) were said by the commenter not to reflect developmental effects since development was complete and function apparently unaffected, and that these effects were considered a reflection of slower growth (reduced body weights) as were the differences in relative organ weights. The Agency considers reduced rat pup body weight and slower growth with resulting differences in organ weight to be effects that are indicators of potential hazard. The Agency's *Developmental Risk Assessment Guidelines* (Ref. 6) state "A change in offspring body weight is a sensitive indicator of developmental toxicity, in part because it is a continuous variable. In some cases, offspring weight reduction may be the only indicator of developmental

toxicity. While there is always a question as to whether weight reduction is a permanent or transitory effect, little is known about the long-term consequences of short-term fetal or neonatal weight changes. Therefore, when significant weight reduction effects are noted, they are used as a basis to establish the NOAEL." EPA, therefore, considers evidence of delayed development, including delayed pinna unfolding and reduced body weight gain, to be significant signs of developmental toxicity.

EPA reaffirms that there is sufficient evidence for listing fenoxycarb on the EPCRA section 313 list pursuant to EPCRA section 313(d)(2)(B) based on the available hepatic and developmental toxicity data for this chemical. Therefore, EPA is finalizing the addition of fenoxycarb on the EPCRA section 313 list.

28. *Fomesafen*. Zeneca Incorporated states that clear species differences are evident which would suggest that peroxisome proliferation and consequential liver toxicity is not relevant to man.

Zeneca Incorporated did not provide any new evidence which supports the lack of relevance of these effects to man. In the absence of evidence to the contrary, the Agency believes that liver toxicity, which is associated with peroxisome proliferation is relevant to the assessment of potential human health effects. As there is evidence of hepatic toxicity in three different rat studies at varying dosages and durations and one dog study, EPA reaffirms that there is sufficient evidence for listing fomesafen on the EPCRA section 313 list pursuant to EPCRA section 313(d)(2)(B) based on the available hepatic toxicity data for this chemical. Therefore, EPA is finalizing the addition of fomesafen on the EPCRA section 313 list.

29. *n-Hexane*. The National Oilseed Processors Association and The National Cotton Council of America contend that EPA failed to perform a detailed hazard evaluation that culminated in a weight-of-evidence determination regarding the toxicity of n-hexane as required under the Agency's *Draft Hazard Assessment Guidelines* (Ref. 11). Commenters state that portions of EPA's support document were taken almost verbatim from the Agency's IRIS data base and that the Agency appears to have relied extensively on the IRIS summary previously prepared for n-hexane. Commenters state that EPA should have evaluated the merits and conclusions of each study separately.

The IRIS data base that EPA cited in support of the listing of n-hexane

represents the Agency's weight-of-evidence hazard determination for chemicals contained in the data base. The information contained in the IRIS data base was developed after the Agency's thorough review of the available data on n-hexane. Therefore, by relying on the IRIS data base EPA did not fail to perform a detailed hazard evaluation of n-hexane as required by the *Draft Hazard Assessment Guidelines* (Ref. 11).

The same commenters state that based on EPA's screening criteria included in the *Draft Hazard Assessment Guidelines* (Ref. 11) if a substance produces neurotoxic effects at doses that are greater than 500 mg/kg/day (i.e., if the lowest observable adverse effect level is 500 mg/kg/day), then the substance would be placed in the "insufficient for listing" category. Commenters went on to state that most of the studies that EPA-cited in support of the listing of n-hexane indicated that n-hexane produces neurotoxic effects only at very high dose levels and in many cases significant effects are only seen at doses that exceed 500 mg/kg/day.

EPA believes that the commenters have misinterpreted the screening criteria contained in the *Draft Hazard Assessment Guidelines* (Ref. 11). The criteria are based on the MED levels which are not LOAELs. These MED values are derived from the LOAELs and, therefore, the direct comparison of the screening criteria with LOAELs is inappropriate. However, EPA notes that significant effects from n-hexane are seen in quantities significantly less than 500 mg/kg/day, for example, a LOAEL of 204 mg/m³ (58 ppm, LOAEL(ADJ) of 73 mg/m³) was established for certain electrophysiological alterations in humans.

These commenters made numerous specific comments concerning the adequacy of the studies summarized in IRIS used to support a chronic neurotoxicity finding for n-hexane. Commenters state that n-hexane is only toxic at very high levels if at all and that the data are not sufficient to support listing under EPCRA section 313. The commenters state that EPA failed to show how the data contained in the *Support Document for the Addition of Chemicals from Section 112(b) of the Clean Air Act Amendments and Chlorinated Paraffins to EPCRA Section 313* (Ref. 12) and the IRIS data base compare with the criteria for adding substances to the EPCRA section 313 list and how the Agency evaluated these studies in light of such criteria.

In the *Response to Comment Document* (Ref. 14), EPA has addressed the specific comments concerning the

adequacy of the studies used to support a chronic neurotoxicity finding for n-hexane. EPA agrees with the commenters that some of the studies included in the data base for n-hexane have limitations. However, the review of the comments and data have not changed EPA's position that the weight-of-evidence supports a finding of chronic neurotoxicity for n-hexane. The weight-of-evidence determination contained in the Agency's IRIS data base is the basis for determining that n-hexane can reasonably be anticipated to cause neurotoxicity in humans. EPA reaffirms that there is sufficient evidence for listing n-hexane on the EPCRA section 313 list pursuant to EPCRA section 313(d)(2)(B) based on the available neurotoxicity data for this chemical. Therefore, EPA reaffirms its determination that n-hexane meets the listing requirements of EPCRA section 313.

DuPont states that if EPA adds n-hexane (CAS No. 110-54-3) to the EPCRA section 313 list the Agency should clarify that isomers of n-hexane are not included in the addition of n-hexane.

EPA notes that the listing of n-hexane is for the straight chain (i.e., "n") isomer of hexane only as the chemical name and CAS number indicate. EPA does not believe that any special qualifier is needed to make the distinction between n-hexane and other isomers of hexane that are not included in this listing.

30. *3-Iodo-2-propynyl butylcarbamate*. Troy Corporation disagrees with the Agency's conclusion that no NOEL was achieved in the rat chronic toxicity/carcinogenicity study cited in the proposed rule. The commenter states that the non-neoplastic changes observed at the 40 mg/kg/day and 80 mg/kg/day dose levels, while also present at the 20 mg/kg/day dose level (lowest dose tested), were not statistically significant and therefore a NOEL was clearly established at this dose. The commenter also contends that non-neoplastic lesions of the stomach in rats are not relevant to humans.

The Agency agrees with the commenter that the increases in nonneoplastic changes reported at 20 mg/kg/day were not statistically significant. However, the lack of a study NOEL was based upon decreased body-weight gain in males at 20 mg/kg/day (the lowest dose tested), not the nonneoplastic lesions. The non-neoplastic lesions reported at 40 and 80 mg/kg/day are still considered serious enough to support a listing on the EPCRA section 313 list.

The same commenter states that the simple findings of increased liver-to-body weight ratio found in the subchronic oral toxicity study in rats cited in the proposed rule as well as the incidence of non-neoplastic stomach irritation found in the chronic toxicity/carcinogenicity study cited in the proposed rule in rats is not of sufficient seriousness to warrant EPCRA section 313 listing. The commenter claims that neither study cited demonstrates a sufficiently known or reasonably anticipated adverse health effect in humans to warrant section 313 listing.

EPA disagrees. The commenter provides no basis for the contention that the nonneoplastic lesions of the stomach in rats are not relevant to humans. The incidence of these lesions was dose dependent, and was apparent at both sacrifices. Moreover, incidence increased with duration of treatment. The Agency considers this effect to be serious in nature and not readily reversible, and therefore the chemical warrants listing. The liver-to-body weight ratio from the subchronic study is not in itself sufficient to warrant listing on the EPCRA section 313 list but is provided as corroborating information.

EPA reaffirms that there is sufficient evidence for listing 3-iodo-2-propynyl butylcarbamate on the EPCRA section 313 list pursuant to EPCRA section 313(d)(2)(B) based on the available chronic toxicity data for this chemical. Therefore, EPA is finalizing the addition of 3-iodo-2-propynyl butylcarbamate on the EPCRA section 313 list.

31. *Iron pentacarbonyl*. International Specialty Products contends that iron pentacarbonyl cannot reasonably be anticipated to cause significant adverse human health effects at concentration levels that are likely to exist beyond facility site boundaries because it rapidly decomposes.

EPA disagrees. The Agency considered the instability of iron pentacarbonyl under certain conditions, such as high temperature and/or prolonged exposure to direct sunlight, in its original modeling of exposure to iron pentacarbonyl cited in the proposed rule. EPA believes that its modeling was based on reasonable reactivity data. EPA reiterates that its exposure assessment indicates that iron pentacarbonyl can reasonably be anticipated to be present at facility boundaries at concentration levels that would cause an adverse effect. EPA reaffirms that iron pentacarbonyl is sufficient for listing on the EPCRA section 313 list pursuant to EPCRA section 313(d)(2)(A) based on available acute toxicity and exposure data for this

chemical. Therefore, EPA is finalizing the addition of iron pentacarbonyl on the EPCRA section 313 list.

32. *Lithium carbonate.* FMC Corporation contends that although lithium is toxic at therapeutic levels, naturally occurring levels are below the toxic range and therefore, lithium carbonate poses no threat to the general population. The commenter also contends that there is no evidence that lithium carbonate is "known to cause or can reasonably be anticipated to cause significant adverse acute human health effects at concentration levels that are reasonably likely to exist beyond facility site boundaries as a result of continuous, or frequently recurring, releases." Thus, the commenter feels that there is no basis for the listing of lithium carbonate on the EPCRA section 313 list.

The Agency is not proposing to list lithium carbonate on the basis of acute effects but on the basis of developmental effects. Therefore, the Agency does not need to determine that lithium carbonate is "known to cause or can reasonably be anticipated to cause significant adverse acute human health effects at concentration levels that are reasonably likely to exist beyond facility site boundaries as a result of continuous, or frequently recurring, releases," but rather must satisfy the section 313(d)(2)(B) criteria.

As the commenter noted, lithium carbonate is a well-known developmental toxicant in both animals and humans at therapeutic levels and can cause life-threatening cardiac abnormalities in the developing human fetus. The commenter argues that lithium is toxic at therapeutic levels but not at naturally "occurring levels." The Agency agrees that lithium may be toxic at therapeutic levels but also recognizes that use of lithium in a therapeutic setting is carefully controlled. Levels observed in a therapeutic setting may have little or no relationship to levels seen in an uncontrolled release setting. Furthermore, both the efficacy of lithium and its associated toxicity in humans is dependent upon individual sensitivity and can vary widely from individual to individual making uncontrolled release even more problematic. EPA reaffirms that there is sufficient evidence to list lithium carbonate under EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available developmental toxicity data for this chemical. Therefore, EPA is finalizing the addition of lithium carbonate on the EPCRA section 313 list.

33. *Metam sodium.* Buckman Laboratories International, Incorporated

and Zeneca Incorporated state that the developmental toxicity studies cited in support of listing for metam sodium were rejected by the Agency to support the registration of a pesticide under FIFRA, and therefore should not be used. Further, they state that these data have been superseded by two new studies that have been accepted by the Agency, and that only the new studies should be considered for listing of metam sodium.

The two earlier studies referred to by the commenters and cited in the proposed rule were submitted to the Agency under FIFRA. EPA's evaluation of those studies for purposes of FIFRA indicated that they did not fully satisfy the guidelines for developmental toxicity studies (Ref. 6); however, EPA did not reject these studies. EPA considers them sufficient as part of a weight-of-evidence evaluation, in determining the developmental toxicity of this chemical. The two new studies cited by the commenter have been reviewed by the Agency. The Agency found these studies to fully satisfy the guidelines (Ref. 6). However, these new studies do not supersede the previous studies nor did the Agency ignore them. Rather, all four studies were used together as part of the Agency's weight-of-evidence to evaluate the chemical. EPA does not ignore indications of potential toxicity simply because of study design flaws. A full discussion of these studies is contained in the *Response to Comment Document* (Ref. 14).

Zeneca Incorporated further stated that the rat teratology study was a gavage study and not a dietary study. The commenter claims that this is an unrealistic route of human exposure.

The commenter is correct in stating that the rat teratology study was a gavage study and not a dietary study. This does not diminish its appropriateness for consideration in the hazard assessment for listing. In fact, EPA requests that developmental toxicity studies be conducted by gavage, because this route allows for a more accurate assessment of the dose the animal actually receives.

EPA reaffirms that there is sufficient evidence for listing metam sodium on the EPCRA section 313 list pursuant to EPCRA section 313(d)(2)(B) based on the available developmental toxicity data for this chemical and its metabolite, carbon disulfide. Therefore, EPA is finalizing the addition of metam sodium on the EPCRA section 313 list.

34. *N-Methyl-2-pyrrolidone.* IBM, American Automobile Manufacturers Association, and N-Methylpyrrolidone Producers Group object to the listing of

N-methyl-2-pyrrolidone (NMP) on the EPCRA section 313 list. NMP Producers Group contends that the 2-generation reproductive study and the rabbit gavage developmental study cited in the proposed rule are flawed. NMP Producers Group further contends that the author of the 2-generation rat reproductive study and an independent reviewer have reached similar conclusions.

In reviewing the material submitted by the commenter, EPA failed to find a statement from the author that the study was flawed. The review of the 2-generation rat reproductive study by an independent reviewer did not find fault with the entire study but stated that it should not be used for *risk assessment* purposes. EPA agrees with this judgement but is not using this study for risk assessment purposes but rather as an indication of human health hazard. The Agency believes that the adverse effects seen in these studies are of sufficient seriousness to warrant listing under EPCRA section 313(d)(2)(B).

NMP Producers Group also states that when the 2-generation reproductive study is evaluated taking into account the genetic fertility problem in the strain of male rats, the study establishes a NOAEL of 160 mg/kg rather than the NOAEL of 50 mg/kg cited in the proposed rule. The commenters also believe the study should not be considered because the variability in male fertility was not dose-dependent.

EPA does not agree that the NOAEL should be adjusted for the fertility problem of the strain of male rats used in the study. During the first mating on which EPA based its concern level (F_{2a}) the high-dose male group exhibited a 24 percent reduction in the mating index. In addition, there was a statistically significant, dose-related reduction in the male fertility index; thus, the index was 93.1, 72.4, 72.4, and 46.7 in the control, low-, mid-, and high-dose groups, respectively. The control value in this study is 93.1 percent, well within acceptable limits for any reproductive effects study and as seen the reduction in mating index is dose-related being 72.4 percent in the low- and mid-dose groups and 46.7 percent in the high-dose group. With a control value of 93.1 percent and using the concurrent controls as an index of mating performance for the males in this study, the Agency feels that there is no reason to adjust the NOAEL of 50 mg/kg to account for reduced fertility in the test animals. During the second mating (F_{2b}), the male high-dose group exhibited a 31 percent reduction in the mating index, and again, there was a statistically significant, dose-related reduction in the

male fertility index (83.3, 69.0, 60.0, and 34.5 in the control, low-, mid-, and high-dose groups, respectively). The female high-dose group exhibited a 28 percent reduction in the fertility index, and again, there was a statistically significant, dose-related reduction in the fecundity index (92.6, 74.1, 64.3, and 50.0 in the control, low-, mid-, and high-dose groups, respectively). Again, the Agency does not feel that a control value of 83.3 percent fertility index in the control animals is abnormal and is more concerned with the dose-related decrease in fertility as an indication that NMP is a reproductive toxicant. EPA is also concerned with the decrease in fecundity index in the females and does not feel that the control value of 92.6 percent warrants any adjustment of NOAEL for reduced fertility or mating ability among males in the study.

The Agency also disagrees with NMP Producers Group's contention that decreases in male fertility observed are not dose dependent. The data presented above clearly show a correlation between dose and decreased fertility.

NMP Producers Group claims that the effects of NMP administration manifested only in the second generation of animals.

EPA disagrees and believes that effects were manifested in the first generation. There was a reduction in fertility in the F1 generation, histological evidence of reproductive effects including hypospermia and significant systemic toxicity in the F1 generation. In addition, EPA does not believe that it is unusual to see increased severity in the second generation since animals have either been treated for 2 generations or are the offspring of treated animals and cumulative effects or effects on the reproductive system of the first generation animals may manifest in the second generation.

NMP Producers Group further believes that NMP is not a developmental hazard because EPA's conclusion is based on observations from what the commenter claims is a flawed reproductive study. The commenter adds that a considerable body of evidence supports the conclusion that NMP is not uniquely toxic to a developing fetus.

EPA's conclusions about the developmental toxicity of NMP are based upon a rabbit gavage study and the developmental portion of the 2-generation reproductive study referred to above and cited in the proposed rule. The rabbit gavage study showed a significant increase in resorptions and malformations (misshapen skull bone and cardiovascular malformations). The

LOAEL for developmental toxicity in this study was 540 mg/kg and the NOAEL was 175 mg/kg. The developmental portion of the 2-generation reproductive effects study showed evidence of developmental toxicity in both generations after exposure to 500 mg/kg as demonstrated by reduced litter size, reduced postnatal survival, and reduced pup body weight. The Agency believes that despite the flaws in the study, the data described above clearly show evidence of developmental toxicity. In addition, EPA believes that the body of evidence supports the finding that NMP is uniquely toxic to the developing fetus and the information available to the Agency both from the rat developmental study and rabbit gavage study is sufficient to list NMP on the EPCRA section 313 list.

EPA reaffirms that there is sufficient evidence to list N-methyl-2-pyrrolidone under EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on available developmental and reproductive toxicity data for this chemical. Therefore, EPA is finalizing the addition of N-methyl-2-pyrrolidone on the EPCRA section 313 list.

35. *Molinate*. Zeneca Incorporated contends that the observations attributed to the 35 mg/kg/day dose level in the rat developmental toxicity study "in fact occurred at 140 mg/kg/day, the highest dose tested and were thus a consequence of maternal toxicity." The commenter states that the NOEL for that study was 35 mg/kg/day.

The Agency does not agree that the NOEL for this study was 35 mg/kg/day. The NOEL for developmental toxicity was 2.2 mg/kg/day based on an increase in runting at the next highest doses, 35 and 140 mg/kg/day. The other adverse effects listed in the comments for this study occurred only at the highest dose tested (140 mg/kg/day). The NOEL for maternal toxicity was 35 mg/kg/day and that the effects on the pups (runting) occurred at a dose level lower than the dose level found to be maternally toxic.

The same commenter stated that the issue of whether molinate is a reproductive toxin on the basis of its adverse effect on fertility in rodents has been very extensively investigated with studies in rabbits, dogs, monkeys, and man, and these studies have shown "conclusively that the effects seen in rodents is [sic] not relevant to man."

While EPA agrees that there has been extensive testing of molinate with respect to fertility, the data on the rabbit and dog do not support the commenter's contention that the effects seen in rodents are specific only to rodents. For example, in each of the fertility studies

in rabbits, both an increase in pre-implantation loss and abnormal sperm were observed. These two consistent [reproducible] observations are suggestive of fertility effects, are two of the same observations found in rats and, although not as dramatic as observed in rats, cannot be negated. In the chronic dog study, lesions in male reproduction organs and effects on sperm were observed, which demonstrate that, at least in the males, the gonads are target organs for molinate. The lack of any effect on the limited parameters assessed in the male monkey studies lends little credence to the argument since only male monkeys were exposed to molinate, and no reproduction studies have been performed to assess reproductive performance. Since molinate is reaching the gonads in all species, not only in rodents as the commenter claims, molinate can reasonably be anticipated to cause fertility/reproductive effects in humans. Further, animals are accepted as surrogates for toxicity testing to predict potential hazard to humans, except in a few rare cases where effects have been determined to be species-specific [e.g., $\alpha 2\mu$ -globulin].

The same commenter further contends that a NOEL of 2 mg/kg/day was established in the rat 2-year study, and that this study should not be used to evaluate the neurotoxicity of molinate because the study was not designed to evaluate that effect. Rather, the commenter contends that the "definitive position on neurotoxicity has been determined by specific [neurotoxicity] studies." Zeneca Incorporated did not provide a reference for these "specific studies."

EPA agrees that the NOEL for effects other than neurotoxic effects is 2 mg/kg/day in the chronic rat study. No NOEL for neurotoxic effects was established in that study. The LOEL for neurotoxicity in this study is 0.35 mg/kg/day. Although this study was not specifically designed to evaluate the neurotoxic effects of molinate, adverse neurological effects were reported. Further, they were substantiated by the findings from a 1-year study in dogs.

The same commenter stated that the effects observed in the dog study were found at the highest dose administered for 1-year and were "largely a consequence of extended exposure" and as such should not form a part of the EPCRA listing. The commenter implies that because this is a chronic adverse effect, the effect is not relevant to the EPCRA section 313 criteria.

As specified in section 313(d)(2)(B), a chemical may be listed if it causes

chronic toxicity. Thus, the comment is not relevant.

EPA reaffirms that there is sufficient evidence for listing molinate on the EPCRA section 313 list pursuant to EPCRA section 313(d)(2)(B) based on the available developmental, reproductive, and neurological toxicity data for this chemical. Therefore, EPA is finalizing the addition of molinate on the EPCRA section 313 list.

36. *Nitrate compounds (proposed as nitrate ion)*. American Automobile Manufacturers Association, Merck, and the Department of Energy disagree with EPA's proposal to list nitrate ion because an ion is not a chemical. Merck further states that nitrate ion "exists only in aqueous media." The Chevron Companies, the Department of Energy, Chemical Manufacturers Association, and Air Products and Chemicals, Incorporated contend that in proposing to add nitrate ion to EPCRA section 313 the Agency actually proposed to add a category of chemicals that dissociate to generate nitrate ion. EPA agrees with the commenters that an ion does not meet the definition of a chemical for purposes of listing on the EPCRA section 313 list and that by proposing nitrate ion the Agency had, in effect, proposed the addition of a category of nitrate compounds that dissociate in water that are reportable only when in aqueous solution. Thus based on the comments provided by the commenters, the Agency is finalizing the addition of the following category: water dissociable nitrate compounds (reportable only when in aqueous solution). Qualifiers of this sort have been used to define the form of a chemical for which reports should be submitted, e.g., zinc (fume or dust). The qualifier following this listing indicates that only water dissociable nitrate compounds that are manufactured, processed, or otherwise used as an aqueous solution at a facility are subject to reporting. As with all other aspects of EPCRA section 313 reporting, only the weight of the listed chemical is subject to threshold determinations. That determination does not include, for example the weight of the water or any other constituent in the solution other than the nitrate compound. Beyond the threshold determination, the amounts reportable on Form R should only include the mass of the nitrate portion of the compound in solution. This approach is consistent with guidance given for determining threshold and release amounts for metal compounds. EPA recognizes that most monitoring data available measure only the dissociated nitrate ion released and not the amount of total nitrate compounds

from which the nitrate ion dissociated. Reporting of the amount of the total water dissociable nitrate compound in wastes would be complicated when more than one substance contributes to the nitrate ion content of the waste and when the nitrate compound is converted to a different substance due to waste treatment or other processes. It is therefore reasonable to require reporting of only the nitrate ion released in order to avoid confusion over the meaning of total compound released.

EPCRA section 313 requires threshold determinations for chemical categories to be based on the total of all chemicals in the category manufactured, processed, or otherwise used. For example, a facility that manufactures three members of a chemical category would count the total amount of all three chemicals manufactured towards the manufacturing threshold for that category. One report is filed for the category and all releases are reported on this form.

In the proposed rule, EPA discussed both the human health and environmental adverse effects attributable to nitrates. EPA continues to be concerned about the potential environmental impacts of nitrates. In today's action, EPA is adding nitrate compounds based on the adverse human health effects that the nitrate moiety causes. Nitrate causes methemoglobinemia. Methemoglobinemia, like carbon monoxide, interferes with the oxygenating capacity of the blood resulting in an under supply of oxygen to the tissues. In adults, cyanosis to lips and mucous membranes occurs at a level of 1.5 g/dL (10 percent saturation in an adult with normal hemoglobin levels). Levels between 30 percent and 50 percent saturation in adults produce depression of the cardiovascular and central nervous systems; levels between 50 percent and 70 percent cause stupor, convulsions and respiratory depression and levels above 70 percent are usually fatal. Because of increased requirement for oxygen in growing tissue and because of decreased blood volume in infants, they are much more sensitive to nitrate ion toxicity than adults. Infants have a lower activity of methemoglobin reductase and thus are more susceptible. Consequently adverse effects are seen at much lower levels in infants than in adults. Irreversible damage to organs such as the heart or brain, and the development of coronary artery disease or pulmonary disease are more likely to develop in infants because the anoxia caused by methemoglobinemia can occur more rapidly and have more devastating effects in growing tissue

than in the "static" tissue of the adult body. EPA believes that these are serious adverse effects that satisfy the criteria of EPCRA section 313(d)(2)(B).

37. *Ozone*. Many commenters opposed the addition of the CAA criteria pollutants (sulfur dioxide, sulfur trioxide (SO_x), nitric oxide and nitrogen dioxide (NO_x), carbon monoxide (CO), and ozone) to the EPCRA section 313 list since extensive data on these chemicals is already collected under the CAA.

EPA agrees with the commenters that there are many complex issues associated with the extensive collection of data on these chemicals under the Clean Air Act. Therefore, EPA is deferring the listing of these chemicals for possible addition at a later time to address some of the issues involving the availability of data collected under the CAA. The Agency does not believe, however, that the listing of ozone should also be deferred. Emissions of ozone, also a criteria pollutant, are not captured under the CAA. The CAA mandates the collection of data on the releases of VOCs (VOCs react in the troposphere to generate ozone and other air pollutants), which are regulated to maintain the ambient air quality standard for ozone. EPA believes there are many other significant uses of ozone (e.g., wastewater treatment, bottled water purification, and chemical intermediate) that would be captured by EPCRA section 313 reporting. Accordingly, EPA does not believe that the finalization of ozone should be deferred. EPA reaffirms that ozone meets the EPCRA section 313(d)(2) criteria pursuant to EPCRA section 313(d)(2)(B) and 313(d)(2)(C) based on the available toxicity data for this chemical. Therefore, EPA is finalizing the addition of ozone on the EPCRA section 313 list.

38. *Pebulate*. Zeneca Incorporated comments that the neurological effects noted in the 1-year feeding study in dogs cited in the proposed rule occurred at the highest dose level (100 mg/kg/day), which was, by design, a toxic dose. Thus, the commenter claims that there is no reasonable hazard.

The Agency disagrees. Although the highest dose tested is designed to elicit toxicity in the dogs, the presence of Wallerian type degeneration of the white matter of the spinal cord at the 100 mg/kg/day dose level in dogs of both sexes is of considerable seriousness and cannot be dismissed only because it occurred at the highest dose tested. Although the dose eliciting degeneration of the spinal cord was the highest dose tested, 100 mg/kg/day, these adverse effects are of sufficient

seriousness to warrant listing based upon the potential for similar effects in humans.

In this study, the NOEL for the Wallerian type neurological lesions is 50 mg/kg/day. However, the NOEL in males is less than 5 mg/kg/day (LOEL based on findings of abnormal behavior, ataxia, severe convulsions, and congestion in both kidneys in one dog). In females, the NOEL was 5 mg/kg/day and the LOEL was 25 mg/kg/day with occurrence of blood in feces, increased absolute and relative liver weight, increase in severity of lipofuscin deposition in kidneys, and hemosiderin deposition in liver and spleen.

EPA reaffirms that there is sufficient evidence for listing pebulate on the EPCRA section 313 list pursuant to EPCRA section 313(d)(2)(B) based on the available neurological toxicity data for this chemical. Therefore, EPA is finalizing the addition of pebulate on the EPCRA section 313 list.

39. *Permethrin*. Zeneca Incorporated states that the hepatic effects noted in the liver of rats and dogs are adaptive rather than toxic responses to the pyrethroid. The commenter further claims that there were no changes in liver weight relative to body weight.

EPA does not agree that the incidence of liver weight increase is not a significant effect, or that there were no changes in liver weight relative to body weight. The liver weights, relative to bodyweight, were increased in all treated groups in the 2-year rat study. EPA believes that the hepatic changes noted in these studies represent a significant adverse effect.

The same commenter contends that in the 2-year rat study cited in the proposed rule, "the NOEL is also incorrectly stated as 5 mg/kg/day, where EPA states a LOEL of 100 mg/kg/day."

The Agency disagrees with the commenter. The NOEL and LOEL should be 5 and 100 mg/kg/day, respectively. At 100 mg/kg/day there was an increase in alkaline phosphatase, liver weight and cellular swelling of the liver (indicative of typical smooth endoplasmic reticulum proliferation), and one male in the low dose group was affected, focal inflammation with degenerative change in the zona fasciculata and swelling and vacuolation of cells in the zona reticularis of the adrenals and reduced body weight in females. EPA considers these to be serious adverse effects.

EPA reaffirms that there is sufficient evidence for listing permethrin on the EPCRA section 313 list pursuant to EPCRA section 313(d)(2)(B) based on the available hepatic toxicity data for this chemical, and pursuant to EPCRA

section 313(d)(2)(C) based on the available environmental toxicity data. Therefore, EPA is finalizing the addition of permethrin on the EPCRA section 313 list.

40. *Phosphine*. Coors Brewing Company states that only liquid phosphine can cause the health effects necessary to support a listing on the EPCRA section 313 list.

Phosphine is a gas (the boiling point is negative 87.4 °C); it only occurs as a liquid when placed under reduced temperature and/or pressure. The acute toxicity data used to support the listing of phosphine is based on exposure to phosphine in the air (i.e., phosphine gas). Thus, EPA does not agree that only liquid phosphine could cause the health effects necessary to support listing under EPCRA section 313. EPA reaffirms that there is sufficient evidence for listing phosphine on the EPCRA section 313 list pursuant to EPCRA section 313(d)(2)(A). Therefore, EPA is finalizing the addition of phosphine on the EPCRA section 313 list.

41. *Polychlorinated alkanes (proposed as chlorinated paraffins)*. EPA proposed the addition of chlorinated paraffins that consisted of polychlorinated alkanes. Occidental Chemical Corporation and the Chlorinated Paraffins Industry Association state that the proposed chlorinated paraffins category is really a category of chlorinated hydrocarbons since it covers a broad range of chlorinated hydrocarbons including chlorinated paraffins and chlorinated α -olefins.

EPA believes that there may be confusion over what is covered by the chlorinated paraffins category, as named, because the name chlorinated paraffins identifies a particular feedstock used to make this class of compounds. However, as discussed below, EPA believes that the chlorination of paraffins and α -olefins results in products that do not differ significantly structurally, physically, or toxicologically. EPA believes that, rather than name the category based on one of the feedstocks used to make these compounds, a more appropriate name for the category is one that describes the actual members of the category. Therefore, because the members of this category are alkanes containing three or more chlorines, EPA is renaming this category polychlorinated alkanes. The new category name will not expand the scope of the category. EPA believes that the toxicity data for chlorinated paraffins is sufficient for all polychlorinated alkanes that fall within the same carbon number and chlorine

content regardless of what materials where used to manufacture them.

Courtlands Aerospace, ELF Lubricants North America, Incorporated, Tower Oil & Technology Company, National Oil Products, Incorporated, OxyChem, the American Automobile Manufacturers Association, the Association of International Automobile Manufacturers, Incorporated, the Specialty Steel Industry of the United States, the Independent Lubricant Manufacturers Association, Engineered Lubricants, Sealed Air Corporation, American Federation of Labor and Congress of Industrial Organizations, Chlorinated Paraffins Industry Association, and Far West Oil Company, Incorporated contend that the available toxicity data is insufficient to support the addition of chlorinated paraffins to EPCRA section 313. Some of these commenters state that they do not believe that EPA should create chemical categories such as that proposed for chlorinated paraffins on the EPCRA section 313 list. Some of these commenters state that the long-chain chlorinated paraffins have not been classified as "probable human carcinogens" by NTP or IARC. Some of these commenters made numerous specific comments concerning the adequacy of the studies used to support EPA's listing of chlorinated paraffins and pointed out flaws in the data for both long and short-chain chlorinated paraffins. Some of the flaws that the commenters allege concern the studies used to support the listing of the short-chain species and included: (1) Kidney tumors in male rats may be due to binding to $\alpha 2\mu$ -globin, a male rat-specific protein; (2) route of administration (forced gavage feeding); (3) corn oil as a vehicle; (4) use of the B6C3F1 mouse; (5) short-chain chlorinated paraffins are non-genotoxic in a variety of short-term assays; (6) peroxisome proliferation, liver growth in male and female rats and mice and stimulation of replicative DNA in rodents have not been shown to occur in guinea pigs indicating that they are mouse and rat specific and have no relation to tumor formation in humans; and (7) thyroid tumors may be a consequence of a perturbation in the metabolism of thyroxine. Some of the commenters contend that only the data on short-chain chlorinated paraffins are sufficient to justify a listing on the EPCRA section 313 list and that EPA should limit the category to just the short-chain species.

a. *Long-chain chlorinated paraffins*. IARC has not classified the long-chain chlorinated paraffins because there is insufficient evidence that they cause

cancer in treated animals. The NTP found no evidence of cancer in male rats treated with 1,875 or 3,750 mg/kg/day for 24 months with long-chain chlorinated paraffins. Female rats treated with 900 mg/kg/day showed marginal increases in adrenal gland tumors; female rats treated with 5,000 mg/kg/day had marginal increases in liver tumors. The only significant increase in tumor formation occurred in male mice which had a significant increase in malignant lymphomas. After further evaluation of the available data and considering the available statistics, the high background rate of lymphoma in the strain of mice used in the NTP bioassay and the statements made by the NTP Working Group and the Quality Assessment Narrative, which was submitted by the commenters, EPA agrees that there is insufficient evidence to conclude that the malignant lymphomas observed in male mice were treatment related and that long-chain chlorinated paraffins should not be classified as potential carcinogens. Therefore, the Agency concludes that there is insufficient evidence to list long-chain chlorinated paraffins on the EPCRA section 313 list.

b. *Short-chain chlorinated paraffins.* IARC has classified the short-chain chlorinated paraffins as Group 2B, i.e., sufficient evidence for carcinogenicity in animals and probably carcinogenic in humans. Detailed responses to all of the comments concerning the toxicity of the short-chain species are contained in the *Response to Comment Document* (Ref. 14). Summaries of the responses to the most significant comments concerning flaws in the studies used to support the listing of the short-chain species are provided below.

(1) The Agency agrees that the kidney tumors observed in rats are most likely not relevant to tumor formation in man because the male rat kidney possess a unique protein, $\alpha 2\mu$ -globulin which has been shown to be responsible for the development of rat liver kidney tumors, not only after administration of short-chain chlorinated paraffins but after administration of many other chemicals also. However, to state that chlorinated paraffins bind to a protein which is similar to $\alpha 2\mu$ -globulin and that this binding is not seen in guinea pigs as evidence that kidney tumor formation is not relevant to human tumor formation in this instance is not a convincing argument.

(2) The Agency agrees that forced gavage feeding may not be a relevant route of administration when one is using the data for human risk assessment. In this instance, the data are being used as an indication of potential

human hazard and EPA accepts the data as being indicative of such potential.

(3) EPA believes that corn oil is an accepted vehicle of administration for many *in vivo* studies because it is relatively inert and has not been shown to interact with test agents.

(4) The B6C3F1 mouse is the accepted test species of the NTP and EPA has no reason to question the NTP's choice of test species nor to discount results of cancer bioassays using this species.

(5) EPA does not believe that non-genotoxicity is a sufficient reason to dismiss the relevance to man of tumor formation by the short-chain chlorinated paraffins. Non-genotoxicity may be a factor in selecting a model to use for dose response estimation, once tumor formation has been established, but it is not a reason to disregard the significance of tumors which are formed by agents which are non-genotoxic in short-term tests.

(6) The Agency is not convinced that failure to observe liver growth, peroxisome proliferation in hepatocytes and stimulation of replicative DNA in guinea pigs is proof that these effects are specific to rats and mice and have no bearing on tumor formation in humans.

(7) The Agency agrees that there was a perturbation of thyroxine levels in treated animals but does not agree that the observed tumors are therefore irrelevant.

Therefore, the Agency finds that there is sufficient evidence for listing short-chain chlorinated paraffins on the EPCRA section 313 list pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for these chemicals.

i. *Ecotoxicity data.* Courtaulds Aerospace, Occidental Chemical Corporation, and the American Automobile Manufacturers Association contend that ecotoxicity data are only available for the short-chain (10 to 13 carbons, 59 percent chlorine) chlorinated paraffins. The commenters object to EPA assuming the same ecotoxicity for all members of the chlorinated paraffin category because of the potential difference in effects related to chain length and chlorine content.

Although it was stated as such in the proposed rule, EPA did not intend to equate the ecotoxicity of the short-chain chlorinated paraffins with the ecotoxicity of other members of the category. The ecotoxicity data on the short-chain chemicals was provided as further support for the listing of the short-chain chemicals. However, as EPA is not finalizing the addition of the long-chained species, this issue is no longer relevant.

ii. *Chlorinated paraffins versus chlorinated α -olefins.* OxyChem, the American Automobile Manufacturers Association, the Association of International Automobile Manufacturers, the Independent Lubricant Manufacturers Association, and the Chlorinated Paraffins Industry Association correctly state that EPA's proposed definition of chlorinated paraffins does not exclude chlorinated α -olefins. The commenters further contend that chlorinated paraffins and chlorinated α -olefins are distinctly different chemicals with different physical, chemical, and biological properties.

The information provided by the commenters does not substantiate their claim that chlorinated paraffins and chlorinated α -olefins are distinctly different chemicals with different physical/chemical properties. The main difference between chlorinated paraffins and chlorinated α -olefins that EPA is aware of is that chlorinated paraffins, typically manufactured from paraffin mixtures, are also mixtures whereas individual chlorinated α -olefins can be manufactured in moderate to high purity. The issue is whether a pure chlorinated α -olefin falls within the range of structural characteristics that vary in a chlorinated paraffin mixture. In this case, EPA believes that there are no significant structural differences between chlorinated paraffins and chlorinated α -olefins. Both are primarily linear hydrochlorocarbons, and the degree of chlorination of both groups of substances can be controlled. Sixty percent chlorination of 1-dodecene, for example, would yield a product with the formula $C_{12}H_{19}Cl_7$ and a molecular weight of approximately 411. Sixty percent chlorination of the short-chain grade paraffin mixture would yield a mixture of products with an average formula of $C_{12}H_{19}Cl_7$ and an average molecular weight of approximately 411.

The commenters claim that the chlorine positions in chlorinated α -olefins differ significantly compared to the chlorine positions in chlorinated paraffins. EPA does not believe that chlorination at carbons 1 and 2 of the α -olefins makes a significant difference in the majority of the isomers formed by both reactions and even if it did, there are no data that indicate that having two of the chlorines at carbons 1 and 2 is significant from a toxicity standpoint. The commenters do not substantiate their claim (mass spectral data submitted by one commenter is inconclusive and cannot be used in support, for or against, the commenter's position); EPA is not aware of experimental evidence that suggests that

the possible variations in chlorine positions between the chlorinated paraffins and the chlorinated α -olefins differ significantly from the variations possible within these two groups of substances. Since for the α -olefins the first two chlorines are added at carbons 1 and 2, the relative amounts rather than type of each isomer formed may differ between the chlorination of paraffins and α -olefins, especially as the degree of chlorination decreases.

The commenters' claim that chlorinated α -olefins are distinctly different from chlorinated paraffins because their physical properties are very different is unjustifiable. As discussed in detail in the *Response to Comment Document* (Ref. 14), the physical properties of discreet chemicals cannot be compared to those of chemical mixtures. The commenters do not discuss specific differences between chlorinated paraffins and chlorinated α -olefins and therefore do not substantiate their claim. They do, however, elaborate on the differences between structures within the chlorinated paraffins group, particularly those structures that represent the extremes in the C_{10} to C_{30} range. This discussion is therefore more relevant to the issue of listing categories versus individual chemicals discussed subsequently and does not address the issue of differences in the physical properties between chlorinated paraffins and chlorinated α -olefins, discussed previously. Furthermore, EPA believes that the specific differences between structural extremes within the chlorinated paraffins group that the commenters elaborate on are trends that are also observed between structural extremes within the chlorinated α -olefins group.

A valid comparison of physical property data can only be made between two discreet substances of known purity or, in some cases, between two mixtures of chemicals with well defined compositions. EPA believes that an α -olefin and a paraffin, both with the same chain length and both with the same degree of chlorination, are essentially identical structurally (especially if the degree of chlorination is high); the same isomers can be predicted for the chlorination of an α -olefin and a paraffin of the same chain length. The physical properties of chlorinated α -olefins and the corresponding chlorinated paraffins are therefore expected to be very similar. The differences in the chemical and physical properties that the commenters refer to are largely or completely due to the fact that the chlorinated paraffins are mixtures of different chain lengths

while the chlorinated α -olefins typically are composed of a single chain length.

iii. *Category definition.* Since EPA has determined that only the short-chain species meet the listing requirements of EPCRA section 313, the polychlorinated alkanes category will be defined by the following formula and description: $C_xH_{2x-y}Cl_y$; where $x = 10$ to 13 and $y = 3$ to 12 and where the average chlorine content ranges from 40 to 70 percent with the limiting molecular formulas set at $C_{10}H_{19}Cl_3$ and $C_{13}H_{16}Cl_{12}$.

EPCRA section 313 requires threshold determinations for chemical categories to be based on the total of all chemicals in the category manufactured, processed, or otherwise used. For example, a facility that manufactures three members of a chemical category would count the total amount of all three chemicals manufactured towards the manufacturing threshold for that category. One report is filed for the category and all releases are reported on this form.

42. *Polycyclic aromatic compounds.* In the proposed rule, EPA proposed the addition of a delineated polycyclic aromatic compounds (PAC) category that consisted of 28 polycyclic aromatic compounds. Alternatively, EPA proposed the addition of a PAC category based on the following broad definition: "includes all chemical species from the polycyclic aromatic hydrocarbon, aza-polycyclic, thio-polycyclic, or nitroarene families where polycyclic means three or more fused rings. More specifically, it means any combination of three or more fused six or five membered hydrocarbon rings with at least two or more rings being aromatic. The structure may contain fused non-aromatic 5-membered rings, a ring nitrogen, a ring sulfur, one or more attached nitro groups, or one or more attached alkyl groups" (January 12, 1994, 59 FR 1832).

Monsanto, The Chevron Companies, Amoco Corporation, Armco Steel Company, Mobil Oil Corporation, UNOCAL, Pennzoil, Phillips Petroleum Company, American Petroleum Institute, and the Department of Energy object to listing polycyclic aromatic compounds as a category and recommend that EPA list them separately as individual chemicals. American Coke and Coal Chemicals Institute and Mobil Oil Corporation state that if the chemicals are not individually listed then the proposed delineated category should be used. Koch Industries Incorporated, Texaco Incorporated, and the Wisconsin Department of Natural Resources object to the alternative proposal for a PAC category with the broad definition and

recommend that EPA implement the delineated category approach. The Natural Resources Defense Council recommends that EPA use the broad category definition.

EPA believes that polycyclic aromatic compounds should be listed as a delineated category rather than listed individually or defined under the broad category definition. Most if not all of the polycyclic aromatic compounds included in the category are not intentionally manufactured, they are byproducts and impurities from various industrial processes. As such, they occur as complex mixtures that are typically released or transferred together. EPA believes that for this class of compounds a category listing is the most appropriate way to track releases and transfers under EPCRA section 313 because members of this category are structurally similar and induce a similar toxic effect.

The American Petroleum Institute, Mobil Oil Corporation, American Coke and Coal Chemicals Institute, Koch Industries Incorporated, Monsanto, The Chevron Companies, and Amoco Corporation state that analytical methodologies do exist to identify specific chemicals such as those proposed for the delimited PAC category; however, these analytical methodologies require a chemical-by-chemical analysis. They add that since a chemical-by-chemical analysis is required, there would be no reduction in the reporting burden for either a category based on the broad definition or for the proposed delimited category.

EPA proposed the broad category definition approach as a possible way to reduce the reporting burden for a PAC category. However, the majority of the industries that would have to report do not agree that this will result in a reduction of their reporting burden, they believe that it will cause confusion over what chemicals are covered by this category, and do not believe that analytical methodologies exist to identify all of the thousands of chemicals that would be covered by a PAC category based on the alternative broad definition. EPA is therefore not finalizing the alternative proposal to create a PAC category based on the broad definition but is finalizing the proposed delimited PAC category as explained above.

EPA believes that although it may be necessary to perform a chemical-by-chemical analysis for members of this delimited category, the most appropriate way to track releases and transfers under EPCRA section 313 is by creating this category as explained above.

The Chevron Companies, Amoco Corporation, Mobil Oil, UNOCAL, Pennzoil, and the American Petroleum Institute state that polycyclic aromatic compounds share some physical and chemical properties but that this does not necessarily imply similar toxicities. These commenters state that the toxicity potentials vary widely among the polycyclic aromatic compounds but that the public tends to associate all members of a category with the most toxic chemical in the category.

EPA recognizes that similarities in physical and chemical properties do not necessarily indicate that the ability to induce carcinogenic effect among the members of the polycyclic aromatic compounds category are identical. However, these compounds are chemically similar, induce the same toxicological effect (carcinogenicity), and typically are produced, released, and transferred as complex mixtures rather than individual chemicals. EPA therefore believes that it is appropriate to consider these compounds as a category.

Mobil Oil Corporation contends that 11 of the PACs proposed for listing have been reviewed by IARC and found to have insufficient data in animals and no data in humans making the overall evaluation IARC-3 or inadequate evidence of carcinogenicity.

The 11 chemicals the commenter cites as being classified by IARC as a group 3 chemical, i.e., the chemical is not classifiable as to its carcinogenicity, are: carbazole (CAS No. 86-74-8); cyclopenta(cd)pyrene (CAS No. 27208-37-3); dibenz(a,c)anthracene (CAS No. 215-58-7); dibenz(a,i)anthracene (CAS No. 224-41-9); dibenzo(a,e)fluoranthene (CAS No. 5385-75-1); 2-methylchrysene (CAS No. 3351-32-4); 3-methylchrysene (CAS No. 3351-31-3); 4-methylchrysene (CAS No. 3351-30-2); 6-methylchrysene (CAS No. 1705-85-7); 2-methylfluoranthene (CAS No. 33543-31-6); and 1-nitropyrene (CAS No. 5522-43-0). The commenter is correct in that 10 of these 11 compounds have been classified as IARC group 3 chemicals. The 11th compound, 1-nitropyrene (CAS No. 5522-43-0), was classified by IARC as a Group 2B chemical, i.e., a possible human carcinogen. The IARC classification and a review of the data indicate that the data is sufficient to support the listing of this chemical on the EPCRA section 313 list pursuant to EPCRA section 313(d)(2)(B). A second compound, dibenzo(a,e)fluoranthene (CAS No. 5385-75-1) was classified by EPA as a B2 category chemical, the chemical is a probable human carcinogen, which justifies its addition

to EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B). Upon further review of the other 9 IARC group C chemicals, the Agency believes that a more detailed review of their relationship to the 19 PACs for which cancer data is available and is sufficient is necessary before they can be placed on the list on the basis of structure alone. Therefore, EPA will not add these 9 chemicals to the EPCRA section 313 list at this time and the delineated category will consist of the other 19 chemicals proposed for this category.

EPCRA section 313 requires threshold determinations for chemical categories to be based on the total of all chemicals in the category manufactured, processed, or otherwise used. For example, a facility that manufactures three members of a chemical category would count the total amount of all three chemicals manufactured towards the manufacturing threshold for that category. One report is filed for the category and all releases are reported on this form.

43. *Potassium dimethyldithiocarbamate*. Buckman Laboratories International, Incorporated states that the proposed listing of the chemical was based on the results of the rat and rabbit teratology studies, cited in the proposed rule, although neither study demonstrates evidence of developmental toxicity. They contend that the findings in the developmental studies should be considered an artifact.

The findings in rabbits cannot be considered artifacts because there is a dose-related increase in the severity of developmental effects at 38 and 77 mg/kg. At 38 mg/kg, developmental toxicity was characterized by increased post implantation loss, malformations, and sternebral malalignments. At 77 mg/kg, there were reports of severe fetal and embryo lethality. EPA did not cite a rat study in the proposed rule as the commenter claims.

EPA reaffirms that there is sufficient evidence for listing potassium dimethyldithiocarbamate on the EPCRA section 313 list pursuant to EPCRA section 313(d)(2)(B) based on the available neurological toxicity data for this chemical. Therefore, EPA is finalizing the addition of potassium dimethyldithiocarbamate on the EPCRA section 313 list.

44. *Prometryn*. Ciba-Geigy Corporation states that marked renal and hepatic degenerative changes were noted in the high-dose dogs only in the 2-year dog study cited in the proposed rule. The commenter further claims that although minor liver effects were seen in rats in the 28-day study cited in the proposed rule, there were no liver

effects in rats after 90 days at dose levels up to 5,000 ppm. This 90-day study that the commenter cited was not cited by EPA in the proposed rule. Thus, the commenter does not believe that the data support the addition of this chemical to the EPCRA section 313 list.

EPA disagrees with the commenter. In the 2-year dog feeding study, prometryn induced degenerative changes in liver and kidney and bone marrow atrophy at 37.5 mg/kg/day (LOEL, the NOEL is 3.75 mg/kg/day). Although the dose eliciting degenerative changes in liver and kidney and bone marrow atrophy was the highest dose tested, these adverse effects are of sufficient seriousness to warrant listing based upon the potential for similar effects in humans. Further, the findings of the 2-year dog study and the 28-day rat study, cannot be discounted based solely on the results of the 90-day study referred to by the commenter. Rather EPA has considered all of the data in concluding that prometryn meets the criteria for addition to the EPCRA section 313 list.

The commenter questions the use of the rabbit developmental toxicity study because only a slight effect (if real) was noted at the highest dose tested, and was not statistically significant. Although the use of the rabbit developmental toxicity study may not be justified, and the potential for developmental effects therefore not supported, EPA reaffirms that there is sufficient evidence for listing prometryn on the EPCRA section 313 list pursuant to EPCRA section 313(d)(2)(B) based on available hepatic, renal, and bone marrow toxicity data. Therefore, EPA is finalizing the addition of prometryn on the EPCRA section 313 list.

45. *Propachlor*. Monsanto contends that the developmental toxicity study in rabbits cited in the proposed rule was a study that was rejected by the Agency. Monsanto further stated that in this study "a slight decrease in viable fetuses, slight increase in post-implantation loss, and slight decrease in mean fetal weight was noted at the highest dose tested (116.7 mg/kg/day) which caused severe treatment-related maternal toxicity including death. An equivocal increase in post-implantation loss on a percent basis was noted in the mid-dose (58.3 mg/kg/day) level. Marginal developmental effects that were seen were not statistically significant and were within the historical control limits. Propachlor does not produce any observable maternal or fetal toxicity at 5.8 or 58.3 mg/kg/day. In addition, propachlor does not cause developmental toxicity in rats." Monsanto concluded that, based on the "weight of evidence from the rat

and rabbit studies, there does not appear to be any developmental risk to humans."

The Agency does not concur with the commenter's statement that "propachlor does not produce any observable maternal or fetal toxicity at 5.8 or 58.3 mg/kg/day dose levels," nor with the statement that "the marginal developmental effects that were seen were within the historical control limits." The Agency's rationale for the disagreements are as follows:

In a developmental toxicity study in rabbits, oral administration of propachlor at 116.7 mg/kg/day caused maternal toxicity as evidenced by death, clinical signs [salivation and reduced defecation], decreased body weight gain and food consumption, and gross pathological lesions of the stomach. Developmental toxicity at 58.3 and 116.7 mg/kg/day included dose-related increases in the total number of resorptions/litter and post-implantation losses compared to concurrent and/or historical controls.

Based on these findings, it is apparent that the developmental effects seen at 58.3 and 116.7 mg/kg/day levels are attributable to propachlor; the NOEL was 5.8 mg/kg/day.

EPA reaffirms that there is sufficient evidence for listing propachlor on the EPCRA section 313 list pursuant to EPCRA section 313(d)(2)(B) based on the available developmental toxicity data for this chemical. Therefore, EPA is finalizing the addition of propachlor on the EPCRA section 313 list.

46. *Propargyl alcohol*. International Specialty Products is opposed to the listing of propargyl alcohol apparently because an uncertainty factor of 3,000 was used by EPA in setting the RfD. The commenter feels that an uncertainty factor of 100 would have been more appropriate and cites instances where such an uncertainty factor has been used by IRIS in setting reference doses. The commenter does not question the renal or hepatotoxicity cited in IRIS as a basis of its concern.

The commenter is correct in stating that EPA has used uncertainty factors of 100 for other chemicals. However, that was not deemed appropriate in this instance for reasons which are set out by EPA in the IRIS data base. EPA continues to support the listing of propargyl alcohol under EPCRA section 313 on the basis of chronic toxicity which may pose a significant health hazard as manifested by renal and hepatic effects. The uncertainty factor plays no part in this decision. EPA reaffirms that there is sufficient evidence for listing propargyl alcohol on the EPCRA section 313 list pursuant to

EPCRA section 313(d)(2)(B) based on the available hepatotoxicity and nephrotoxicity data for this chemical. Therefore, EPA is finalizing the addition of propargyl alcohol on the EPCRA section 313 list.

47. *Propiconazole*. Ciba-Geigy Corporation states that the increased incidence of liver tumors in the oncogenicity study on propiconazole was noted only in male mice in the high dose (2,500 ppm), which exceeded the MTD, based on decreased survival and body weight gain.

EPA believes that the study high dose (2,500 ppm, equivalent to 325 mg/kg/day) was excessively toxic; however, the Agency also determined that the mid dose (500 ppm, equivalent to 65 mg/kg/day) was not considered sufficiently high to evaluate the carcinogenic potential of propiconazole. The Agency believes that a supplementary study should be conducted in male mice at doses selected to sufficiently evaluate carcinogenic potential without excessive toxicity. At this time however, based on the currently available evidence, propiconazole remains classified as a Group C, possible human carcinogen, with the RfD approach recommended for quantification of human risk.

The commenter further states that relatively minor gastrointestinal effects were noted in dogs at the high dose only (250 ppm).

EPA believes that the data in both the 3-month and 1-year dog studies demonstrate gastrointestinal effects at the high-dose (250 ppm, equivalent to 6.25 mg/kg/day). These effects are considered severe, and, therefore, are of sufficient seriousness to warrant listing propiconazole on the EPCRA section 313 list.

EPA reaffirms that there is sufficient evidence for listing propiconazole on the EPCRA section 313 list pursuant to EPCRA section 313(d)(2)(B) based on the available hepatic and gastrointestinal toxicity data for this chemical. Therefore, EPA is finalizing the addition of propiconazole on the EPCRA section 313 list.

48. *Simazine*. Ciba-Geigy Corporation objects to the listing of simazine under EPCRA section 313 based on reports of liver, kidney, testicular and neural pathology in sheep and increases in liver enzymes in a dog 2-year study. The commenter maintains that the sheep study was conducted to investigate the possible effects that would result if large amounts of simazine were ingested by this species. The commenter also states that in a 1-year study there were some indications of effects on the hematopoietic system but not the

hepatic system at the high dose of 1,500 ppm.

In a 1-year study, NOEL and LOELs of 0.68 and 3.41 mg/kg/day, respectively, were established based on decreased body weight gain, and decreased RBC, HGB, HCT in females. Although the sheep study was conducted for a purpose other than to investigate the overall toxicity of simazine, this does not negate the relevance of its results. EPA reaffirms that there is sufficient evidence for listing simazine on the EPCRA section 313 list pursuant to EPCRA section 313(d)(2)(B) based on the available hepatic, renal, neurological, and reproductive toxicity data for this chemical. Therefore, EPA is finalizing the addition of simazine on the EPCRA section 313 list.

49. *Sodium nitrite*. American Automobile Manufacturers Association contends that EPA has proposed listing on the basis of chronic toxicity but the support document cites studies based on high dose, acute exposures. High dose gestational studies in rats and mice were also cited as the basis for developmental (fetal) toxicity.

EPA agrees that the human studies cited in the proposed rule are acute studies. However, these studies in conjunction with the chronic study in mice, which showed reduced motor activity and major EEG changes in treated animals, support the basis for concern for chronic neurological effects. EPA thus considers sufficient indication of a potential chronic neurologic hazard to list this chemical on the EPCRA section 313.

There were two developmental studies in mice and one reproductive study in rats cited in the proposed rule which showed effects on the fetal development whether sodium nitrite was administered during gestation or lactation. The doses used in the studies with mice, 30 and 80 mg/kg/day, respectively, are not abnormally high for this type of study; the dose range reported for the rat reproductive study, 26 to 256 mg/kg/day is also not abnormally high. The results from all three studies indicate that sodium nitrite induces developmental effects in animals and are sufficient to make a determination that the chemical is a potential health hazard in man.

EPA reaffirms that there is sufficient evidence for listing sodium nitrite on the EPCRA section 313 list pursuant to section 313(d)(2)(B) based on the chronic hematological and developmental toxicity data for this chemical. Therefore, EPA is finalizing the addition of sodium nitrite on the EPCRA section 313 list.

50. *Triallate*. Monsanto contends that the hepatic health effects listed in the proposed rule for triallate are trivial effects that do not provide sufficient evidence that triallate causes hepatic toxicity. In addition, Monsanto claims that pregnant rats exhibited abnormal behavioral signs at 90 but not at 30 mg/kg/day.

Although the Agency agrees that there is not sufficient evidence for hepatotoxic potential of triallate, the Agency does not concur with the commenter that "pregnant rats exhibited abnormal behavioral signs at 90 but not at 30 mg/kg/day." Head bobbing and circling, clear signs of neurotoxicity, were observed in pregnant females at 30 mg/kg/day. Males and non-pregnant females did not exhibit these clinical signs. These data suggest that pregnant rats are more susceptible to the neurologic potential of triallate than the general population.

The commenter noted the existence of a subchronic neurotoxicity study in rats and indicated that this study provides a better estimation of the neurotoxic potential of triallate than the 2-generation reproduction study.

The Agency agrees that the subchronic neurotoxicity study in rats (Ref. 10) provides a clearer picture of the neurotoxic potential of triallate. The Agency has reviewed this study and concludes that the results indicate the neurotoxic potential of triallate and further corroborates the findings cited by EPA in the proposed rule.

Thus, the Agency reaffirms that there is sufficient evidence for listing triallate on the EPCRA section 313 list pursuant to EPCRA section 313(d)(2)(B) based on the available chronic neurotoxicity data for this chemical. Therefore, EPA is finalizing the addition of triallate on the EPCRA section 313 list.

G. Chemicals Not Being Added to EPCRA Section 313

1. *5-Chloro-2-(2,4-dichlorophenoxy)phenol*. Ciba-Geigy Corporation and The Dial Corporation contend that insufficient evidence is available to support the conclusion that 5-chloro-2-(2,4-dichlorophenoxy)phenol poses a risk of hematological toxicity to humans based on handling and uses of the product.

Based on these comments and EPA's reanalysis of the data, the Agency has concluded that the information presented in the proposed rule is not sufficient to justify adding 5-chloro-2-(2,4-dichlorophenoxy)phenol to the EPCRA section 313 list based upon potential human hazard. Therefore, EPA is not finalizing the addition of this chemical on the EPCRA section 313 list.

2. *Clomazone*. FMC Corporation claims that clomazone induces adverse effects only at high dose levels.

The Agency agrees with the commenter. Based on these comments and EPA's reanalysis of the data, the Agency has concluded that the information presented in the proposed rule is not sufficient to justify adding clomazone to the EPCRA section 313 list based upon potential human hazard. Therefore, EPA is not finalizing the addition of this chemical to EPCRA section 313.

3. *Tetrasodium ethylenediaminetetraacetate*. Eight commenters contend that the proposed listing for this chemical was based solely on a single, unreliable developmental toxicity study which used a mixture that contained tetrasodium ethylenediaminetetraacetate along with several other chemicals.

EPA concedes that the effects cannot be attributed solely to tetrasodium ethylenediaminetetraacetate. Therefore, the Agency is not finalizing the addition of tetrasodium ethylenediaminetetraacetate on the EPCRA section 313 list.

H. Miscellaneous Comments

1. *Year-to-year comparisons of the TRI data*. BP America, Texaco, and American Automobile Manufacturers Association contend that the proposed expansion of the EPCRA section 313 list will eliminate any consistency with earlier TRI data and make tracking environmental progress impossible. American Automobile Manufacturers Association further states that EPA needs to ensure that the "total TRI releases and transfers" measurement system allows accurate interpretation of the data, allowing the public to realistically assess progress in pollution prevention. Also, the commenters add that considerable confusion results in trying to explain the different data sets to the public. Mobil Oil Corporation states that EPA should divide the list into three sub-groups so that a facility's history can be tracked on a more common basis.

EPA recognizes that changes in the EPCRA section 313 list and in the reporting requirements have an effect on the characterization of the TRI data. In fact, some change has occurred for every reporting year. In an attempt to provide useful year-to-year comparisons, EPA has presented the TRI data annually on a normalized list of chemicals, i.e., the list of chemicals used for year-to-year comparisons is the same for every year in the comparison. EPA further recognizes the effect that expansion of

the EPCRA section 313 list will have on the TRI data and will continue to work to find ways to make the data useable for cross-year comparisons. EPA will use the 1995 reporting year as the base year for comparisons that include the chemicals added today. Facilities should still be able to track pollution prevention progress for those chemicals previously listed (using 1988 as the base year) and have a new base year for the additional chemicals which can be used to track future pollution prevention progress.

2. *Public perceptions*. Russell Uclaf Corporation, National Paint and Coatings Association, Association of International Automobile Manufacturers, and American Frozen Food Institute oppose the listing of these chemicals under EPCRA section 313 because of the public's misperception of the associated dangers. American Automobile Manufacturers Association (AAMA) states that since the public considers all chemicals on the EPCRA section 313 list to be toxic, any chemical on the list is subject to adverse scrutiny, regardless of the actual risks associated with the chemical. While recognizing past efforts by EPA towards public education, AAMA believes that misunderstanding and misinterpretation of the data still exists which makes it more critical that EPA not expand the list with low risk chemicals. Texaco and AAMA believe that before the Agency expands the EPCRA section 313 list, resources should be committed to provide public education on actual risks portrayed by the data and educate the public on viable means of chemical risk reduction and chemical management.

The chemicals that are listed under EPCRA section 313 exhibit a wide range of effects at various dose levels. While EPA attempts to communicate the TRI data in the most accurate manner, the Agency recognizes that there exists the perception that the TRI data may sometimes be mischaracterized, but that does not justify not adding a chemical for which the statutory criteria are clearly met. EPA agrees that the better approach to such a problem is improving public information on the chemical, which, combined with the release, transfer, and waste management data will enable the public to participate in informed environmental decision-making. EPA continues to attempt to provide the public with means for interpreting the TRI data.

3. *Persistent bioaccumulative chemicals*. In the proposed rule, EPA requested comment on whether chemicals that are manufactured in trace amounts in waste streams, are

highly toxic at very low dose levels and have physical, chemical, or biological properties that make the chemicals persist for extended periods in the environment, and bioaccumulate through the food chain should be listed on the EPCRA section 313 list (January 12, 1994, 59 FR 1791). EPA noted that persistent bioaccumulative toxic chemicals, such as dioxins, are of particular concern in ecosystems such as the Great Lakes Basin due to the long retention time of the individual lakes and the cycling of the chemical from one component of the ecosystem to another. EPA also requested comment on the following: If EPA were to add this type of chemical to EPCRA section 313, what modifications to EPCRA section 313, such as lowering the reporting thresholds and modifying the *de minimis* in mixture exemption (40 CFR 372.38), would be required to ensure that release and transfer information would be collected? In addition to two comments opposed to the addition of this type of chemical, EPA received 35 comments supporting the addition of toxic persistent bioaccumulative chemicals. The majority of these commenters also supported lowering the reporting thresholds for this type of chemical.

Monsanto and Dow Chemical Company object to the addition of persistent bioaccumulative chemicals that are produced in quantities less than the EPCRA section 313 reporting thresholds. The commenters state that many of the persistent, bioaccumulative toxic compounds which are of concern are no longer manufactured in the United States, and are merely present in the environment due to historical activities and not current activities.

EPA disagrees with this contention. EPA's request for comment focused on chemicals that are generated in small quantities. This is not limited to chemicals produced as a product, but includes chemicals that are generated in waste streams. Many persistent, bioaccumulative toxic chemicals are produced in waste streams. Further, EPCRA section 313 requires the reporting of chemicals manufactured in waste streams if the quantity produced exceeds the appropriate reporting threshold.

Monsanto further claims that "the amounts of these particularly dangerous substances coming from industrial facilities are so small that they can have no measurable impact on health or the environment."

EPA disagrees that releases of these chemicals are so low that they will not have an adverse effect upon human health or the environment. The

persistent bioaccumulative aspects of these toxic chemicals are such that even very small quantities released can reasonably be anticipated to cause adverse effects upon human health and the environment.

Monsanto also states that the concept of different reporting thresholds suggests that this threshold would be proportional to the relative hazard. Thresholds for practically non-toxic chemicals may be very high using this concept.

EPA requested comment on lowering the thresholds for these chemicals not so that the reporting thresholds for chemicals listed on the EPCRA section 313 list would be proportional to the relative hazard of the chemicals. Rather, EPA requested comment on lowering the threshold for persistent bioaccumulative chemicals because even minimal releases of these chemicals may result in elevated concentrations in the environment or in an organism that can reasonably be anticipated to result in significant adverse effects. This reflects the increased likelihood that there will be exposure to a chemical that persists due to its longer residence time in the environment. Repeated minimal releases of a persistent chemical may result in elevated concentrations in the environment. For a chemical that bioaccumulates, even low levels of the chemical in the environment may result in increased concentrations in an organism. Thus, lower thresholds for these substances would be considered due to the persistent and bioaccumulative nature of the substances, rather than the direct hazard.

In its next action to add chemicals to the EPCRA section 313 list, the Agency intends to consider the addition of chemicals that are persistent and bioaccumulate. EPA also intends to consider lowering the reporting thresholds for these additional chemicals and those chemicals that are persistent and bioaccumulate that are now on the EPCRA section 313 list. Accordingly, comments received in response to EPA's request for comment on the potential addition of persistent bioaccumulators will be addressed in the future rulemaking if these chemicals are proposed for addition.

4. *Additional chemicals.* The Wisconsin Department of Natural Resources states that the EPCRA section 313 chemical list should be expanded to include the six chemicals listed in section 112(b) of the CAA not currently included on the EPCRA section 313 list or as part of the January 12, 1994 proposal.

EPA has reviewed all of the chemicals listed under section 112(b) of the CAA not currently on the EPCRA section 313 list and has determined that the remaining chemicals either do not meet the current listing criteria or no reports would be received since their production volumes are below reporting thresholds.

5. *Hormone mimics.* The National Wildlife Federation recommends that EPA add to the section 313 list all chemicals with estrogenic or other hormone-mimicking qualities, and the reporting thresholds and *de minimis* exemption for mixtures eliminated for these chemicals.

EPA agrees that the deleterious effects of hormone mimicking chemicals may warrant their future review for listing on the EPCRA section 313 list. Although the effects of these chemicals are difficult to predict, and it is often impossible to establish a clear cause/effect relationship, still it is clear from the available evidence that these chemicals warrant consideration. Wide scale changes in wildlife and human populations have been noted by some researchers. Population decreases and reproductive effects have been linked to these chemicals in a number of wildlife species, including but not limited to bears, Florida panthers, songbirds, and bald eagles, to list just a few. Possibly of greater concern are the effects of these chemicals in humans. In addition to the carcinogenic potential of many of these chemicals, effects on fertility, immune system damages, and many childhood problems have been attributed to hormone-mimics. A number of the chemicals with widespread distribution in the environment reported to have reproductive and endocrine-disrupting effects (Ref. 1) are either already on the EPCRA section 313 list, or are being added as a result of this action. EPA may consider reviewing the remaining chemicals on this list as part of a future action.

As to removing the reporting thresholds and *de minimis* exemption for the hormone-mimicking chemicals already on the section 313 list, these possibilities will be examined at the time that modifying the reporting thresholds and *de minimis* exemption for persistent and bioaccumulative chemicals is addressed. As many of the hormone mimicking chemicals are also either persistent or bioaccumulative they could be included as part of such a review.

I. Comment on EPA's Regulatory Impact Assessment

Comments that are specific to individual chemicals or chemical

categories are addressed in the *Response to Comments Document* (Ref. 14).

Many commenters state that EPA's Regulatory Impact Analysis (RIA) failed to meet the requirements of Executive Order 12866, which mandates regulatory planning and review. The commenters state that: (1) The RIA for the proposed rule did not analyze any alternatives other than adding the 313 chemicals and chemical categories to the EPCRA section 313 list, (2) that it excluded the economic effects due to complying with state, local and other federal requirements that are triggered when a chemical is listed under EPCRA section 313, and (3) that it did not analyze the benefits of the rule. Many commenters also contend that small business impacts were understated in the RIA, and that the time required for compliance is higher than estimated in the RIA. These comments and the Agency's responses are discussed below.

1. *Alternatives.* The commenters believe that the RIA should have included alternatives to adding all of the proposed chemicals and chemical categories to the EPCRA section 313 list. In response to these comments, EPA has revised the RIA to include a variety of alternatives, such as adding the CAA criteria air pollutants, not adding chemicals regulated under FIFRA, not adding the water dissociable nitrate compounds category, and adding the proposed chemicals in conjunction with an alternate reporting threshold for facilities with low-levels of TRI chemicals in wastes. The commenters requested that EPA present the costs for adding each individual chemical. EPA cannot provide the costs on an individual chemical basis because the estimates for most of the chemicals were derived from confidential business information. Displaying the costs for each chemical could disclose this confidential information.

2. *Linked requirements.* Numerous commenters state that the RIA excludes the costs of compliance with state, local and other federal requirements that are triggered when a chemical is listed under EPCRA section 313. The linked requirements that the commenters raise include state taxes and fees, state pollution prevention planning requirements, special requirements for certain National Pollutant Discharge Elimination System (NPDES) storm water permits, and requirements for federal facilities under Executive Order 12856. EPA has revised the RIA to discuss state and federal requirements that are linked to reporting under EPCRA section 313. However, EPA has not quantified the costs of such

requirements. In some cases, this is because there is insufficient data to make a reasonable estimate. In other cases, EPA does not believe that the requirements represent a social cost. The requirements that may be linked to listing under EPCRA section 313 are discussed below.

a. *State fees.* Thirteen states place a tax or fee on facilities filing TRI Form R reports. These states are Colorado, Florida, Iowa, Kansas, Maine, Massachusetts, Minnesota, Mississippi, Nevada, Ohio, Pennsylvania, South Dakota, and Texas. Many commenters estimate that the costs resulting from state fees and taxes linked to EPCRA section 313 reporting are up to 50 percent of the direct cost of filing the forms, and state that any tax is likely to induce a reduction in economic welfare. EPA has revised the RIA to discuss state fees and taxes that are linked to reporting under EPCRA section 313. However, the taxes and fees are not direct social costs, and EPA does not believe that there is sufficient information to estimate the net social costs or benefits of these requirements.

The commenters treat state taxes and fees on the EPCRA section 313 reports as costs, but these are transfer payments and not economic costs to society. Specifically, the standard definition of a cost in economics is the consumption of a resource (e.g., labor, equipment, natural resources, etc.). A tax or fee is a transfer payment from one party to another. While the fee is a cost to the firm (and/or its customers), it is income to the state. No resources are consumed, except for transaction costs, so the amount of the fee is not a cost to society.

EPA disagrees with the commenters' contention that any tax or fee is likely to induce a reduction in economic welfare. EPA believes that taxes or fees on toxic chemicals may resolve a market failure and increase social welfare. The use of toxic chemicals often creates a negative externality. For instance, releases of a chemical may cause health and environmental effects in the surrounding community. In such cases, it is likely that private costs are below true social costs, because private markets do not provide an adequate incentive for firms to internalize these externalities. In such cases, taxes may be the optimal method to correct the market failure. EPA believes that if the commenters feel that the fees are not set at the level that optimizes social welfare, their remedy lies with the appropriate state agency, and not EPA.

EPA does not feel that it is feasible to estimate the size of the transfer payment resulting from state fees and taxes linked to EPCRA section 313, or the net

social costs or benefits of these payments. The commenters made their estimates by applying the maximum state fees to all facilities nation-wide. EPA does not feel that such a calculation is appropriate. Most states have no fees or taxes linked to EPCRA section 313 reporting, and the level of the fees or taxes (and how they are assessed) is different in each of the rest of the states, varying from \$25 to \$50,000. Many of the state requirements are not flat fees, but are graduated depending on the level of releases that a facility reports. An accurate representation of the size of the transfer payments would require estimating the geographic distribution of new reports, and the level of releases and transfers for each report. EPA feels that it is not possible to predict with a reasonable degree of accuracy the location and level of releases for facilities that will report on the chemicals being added to the EPCRA section 313 list.

Nor is it feasible to accurately estimate the net social costs or benefits of the state fees and taxes. To do so would require knowing not only the size of the transfer payments, but the damages caused by the use and release of the chemicals, and the change in behavior that would result from the fees and taxes. EPA does not have adequate information on the facilities that would be affected by the rule to make such estimates. As a result, the RIA has been revised to qualitatively discuss state fees and taxes linked to EPCRA section 313 reporting, but does not estimate the size of the resulting transfer payments, or the net social costs or benefits.

b. *State pollution prevention programs.* Seven states (Arizona, Maine, Massachusetts, Minnesota, Mississippi, New Jersey, and Texas) mandate pollution prevention plans from facilities reporting under EPCRA section 313. Facilities in these states that are reporting to TRI for the first time because of the additions to the chemical list will have to prepare pollution prevention plans. Although the development of pollution prevention plans imposes a cost on facilities, the RIA did not analyze the costs of these requirements. Many commenters contend that there are significant costs for preparing such plans, and that the RIA should have included these costs.

Quantifying the impacts of state pollution prevention requirements would require predicting which facilities reporting for the additional chemicals would be located in these seven states. As stated above, it is not possible to accurately predict the geographic location of new reporters.

Thus, no costs are estimated for state pollution prevention plans in the RIA.

Nor does the RIA quantify the benefits derived from these pollution prevention planning requirements. None of the commenters submitted any evidence comparing the social benefits of such requirements to the costs. Therefore, EPA has no information from which to conclude that the linked requirements for state pollution prevention plans would reduce the net social benefits of adding chemicals to EPCRA section 313.

EPA has not quantitatively estimated either the costs of state pollution prevention planning requirements or the benefits of such programs in the RIA. However, the RIA has been revised to qualitatively discuss requirements that are linked to EPCRA section 313 reporting, including pollution prevention plan preparation.

c. *NPDES storm water permits.* EPA issued National Pollutant Discharge Elimination System (NPDES) "baseline" general permits for storm water discharges associated with industrial activity on September 9, 1992 (57 FR 41236). EPA subsequently proposed a multi-sector storm water industrial permit covering 29 industrial sectors (November 19, 1993, 58 FR 61147). The "baseline" general and multi-sector general permits have special pollution prevention requirements for certain EPCRA section 313 facilities, and the "baseline" permits also contain special monitoring requirements. Many commenters assert that the RIA underestimates the costs of the rule by a factor of up to 5.6 by not including the costs of NPDES storm water permit requirements that are triggered by adding chemicals to the EPCRA section 313 list.

EPA believes that the commenters' estimates are based on a cost scenario that is not applicable to the typical facility affected by the proposal to add chemicals under EPCRA section 313. There are four reasons that the commenters' estimates are not generally appropriate. Any of these reasons alone demonstrate that the commenters have overestimated the number of facilities that are affected and the size of the impact. Because there may also be a significant overlap among the four, the commenters' estimates are likely to apply to few, if any, facilities. The commenters' estimates would not apply to all facilities affected by the rule, as the commenters contend.

First, only a fraction of the facilities that would report under EPCRA section 313 for the additional chemicals would be affected by the NPDES storm water permits. A facility that submits TRI Form R is only subject to storm water

permitting requirements if industrial materials or activities are exposed to storm water, and if the facility is reporting to TRI for one of the section 313 water priority chemicals. Only about two dozen of the chemicals being added to the EPCRA section 313 list qualify as section 313 water priority chemicals, and thus would be covered by the NPDES requirements. About half of these are pesticides, which would not be manufactured or processed at many facilities.

Second, EPA expects the majority of facilities to have existing containment systems that meet most of the requirements of the NPDES permits. Third, many of the costs for the storm water requirements are likely to apply at the facility level. In such cases, facilities that installed systems for the current EPCRA section 313 chemicals will not face incremental costs for the additional chemicals. Fourth, the special requirements of the NPDES storm water permits are based on the coverage of EPCRA section 313 at the time the permits were issued. The NPDES requirements do not apply to chemicals that are added to the EPCRA section 313 list until the time of permit renewal (which occurs every 5 years), and may not apply in subsequent permits, depending on the Agency's decisions at the time those permits are issued.

In addition, the commenters based their estimates solely on the upper bound of EPA's estimates for the NPDES permits, and have ignored the mix of low-cost and high-cost facilities that is likely to exist. EPA believes that the commenters' estimate is a hypothetical "worst-case" scenario that does not apply to the typical facility and may not apply to any facilities. EPA believes that the costs of the storm water requirements for the proposed chemicals will be relatively minor. Again, EPA has revised the RIA to qualitatively discuss the linkage between EPCRA section 313 reporting and the NPDES storm water, but it has not made any quantitative estimates of these costs.

d. *Executive order 12856.* Executive Order 12856, signed by the President in August 1993, extends the coverage of EPCRA to federal facilities. In addition, section 3-303(a) of the Executive Order states that "Each federal agency shall establish a plan and goals for eliminating or reducing the unnecessary acquisition by that agency of products containing extremely hazardous substances or toxic chemicals" (emphasis added). The Executive Order defines "toxic chemical" as a substance on the list described in section 313 of EPCRA. Many commenters contend that

the cost to the federal government and the private sector of complying with Executive Order 12856 for the chemicals being added to EPCRA section 313 will be \$1.5 billion per year.

EPA does not believe that the effects of Executive Order 12856 should have a bearing on the decision-making regarding the addition of toxic chemicals to EPCRA section 313. EPA believes that following the commenters line of reasoning would discourage the federal government from ever making any changes in procurement, for whatever reason, because doing so might have an impact on a supplier. Furthermore, EPA believes that there is insufficient data to make any estimate of the effects of the Executive Order, and that the resources required to make such an estimate would exceed the value of the information.

EPA notes that section 3-303(a) of the Executive Order does not require the elimination of toxic chemicals in federal procurement. If the performance characteristics of a toxic chemical or product containing a toxic chemical are critical in the required tasks, federal agencies may continue to purchase it. Each federal agency must make its own determination whether a particular toxic chemical is necessary in a particular use.

The Executive Order requires that federal agencies eliminate or reduce the unnecessary procurement of extremely hazardous substances or toxic chemicals. None of the commenters identify which toxic chemicals are being unnecessarily purchased by the federal government, and which federal agencies are making these unnecessary purchases. Without such information, EPA cannot verify the commenters' claim that the addition of chemicals to EPCRA section 313 will create significant impacts as a result of the Executive Order. If these chemicals are not being purchased by the federal government, or are not being purchased unnecessarily, there will not be an impact.

The commenters' estimate of \$1.5 billion in costs is based solely on a series of assumptions, which are not supported by data. EPA does not believe that the commenter's analysis was based on a careful analysis of any factual information. EPA has no data with which to replace these assumptions. Given EPA's belief that effect of the Executive Order should not have a bearing on the rulemaking (as well as the limitations of the Executive Order, the small amount of procurement that would be affected, and the ability of producers to sell to private sector clients or manufacture substitutes), EPA does

not believe that there is a need to develop any data on these factors.

The Executive Order states that "the environmental, energy and economic benefits of energy and water use reductions are very significant," and that "the federal government has the opportunity to realize significant economic as well as environmental benefits of pollution prevention." The Executive Order provided a mandate for the federal government to reduce its unnecessary use of toxic chemicals. EPA believes that the proposal to add chemicals to the section 313 list complements this mandate.

Furthermore, EPA hopes that federal agencies will comply with the spirit of the Order, and reduce their unnecessary use of toxic and hazardous chemicals, whether or not these chemicals are listed on the EPCRA section 313 list. EPA believes that, by definition, the social benefits cannot exceed the social costs for an unnecessary toxic chemical, and social welfare can be improved by switching to a substitute product. Therefore, EPA believes that any actions federal agencies take to meet their obligations under Executive Order 12856 will have a positive net benefit.

3. *Benefits.* Many commenters assert that the RIA did not show any benefits to adding chemicals to the EPCRA section 313 list of chemicals. The commenters appear to have made these statements because EPA did not make a quantitative estimate of the benefits associated with the rule.

There are two types of benefits associated with EPCRA section 313. The first type of benefit is due to improvements in understanding, awareness, and decision-making related to the provision and distribution of information. The second type of benefits derive from changes in behavior that result from the information reported to TRI. These benefits include reduced environmental and health risks, and reduced treatment and disposal costs. These changes in behavior come at some cost to society. Because the current state of knowledge about the economics of information is not highly developed, EPA has not attempted to quantify the pure information benefits of adding chemicals to the EPCRA section 313 list. Because of the inherent uncertainty in the chain of events, EPA has also not attempted to quantify the benefits or the costs of the changes in behavior that result from the information. EPA does not believe that there are adequate methodologies to make reasonable quantitative estimates of either type of benefits. However, EPA believes that its qualitative discussion of the effects of the current TRI program show that such

benefits do exist. The information on the additional chemicals is expected to improve scientific understanding of the environment and health risks, foster greater community awareness of industrial activities, and allow Federal, state, and local authorities to make better informed decisions on acceptable levels of toxic chemicals in communities.

Instead, EPA has drawn its conclusions about the net benefits of adding chemicals to EPCRA section 313 by inference. In enacting EPCRA and the PPA, Congress implicitly determined that the net benefits of reporting was positive for the original list of 320 chemicals and categories. EPA's interpretation of the statutory toxicity criteria is more stringent than Congress' original determination because EPA has deleted 12 chemicals from the original list of 320 chemicals and categories developed by Congress. EPA believes that all of the chemicals being finalized meet the statutory toxicity criteria of section 313, and are at least as toxic as some of the chemicals for which Congress believed there were net benefits due to reporting. Thus, by inference, the net benefits of reporting for the chemicals added in this rulemaking should be positive as well.

EPA believes that the experience of the past 5 years shows that reporting under EPCRA section 313 has produced real gains in understanding about exposure to toxic chemicals. EPA sees no reason why the information on the additional chemicals will provide less understanding than the currently reported chemicals have provided.

4. *Small business.* Under the Regulatory Flexibility Act (5 U.S.C. sections 601 - 612), agencies must prepare an analysis of small business impacts for proposed rules. Many commenters contend that small business impacts were understated in the RIA, and they question EPA's conclusion that the rule will not have a significant impact on a substantial number of small entities. EPA believes that the commenters have significantly overestimated the costs of the rule, and that the commenters' estimates of small business impacts are not valid. EPA has provided additional analysis in the RIA for the final rule that demonstrates that the rule will not have significant cost impacts on small entities.

EPA believes that, whether or not the proposed rule would have had significant cost impacts on small entities, the Agency has subsequently met its obligations under the Regulatory Flexibility Act. Where a proposed rule would have significant impacts on small entities, the Act requires EPA to identify

and consider (but not necessarily adopt) alternatives that minimize the impact on these entities, while accomplishing the stated objectives of the applicable statute.

Elsewhere in this issue of the **Federal Register**, EPA is finalizing a rule establishing an alternate threshold for low-levels of TRI chemicals in waste that would otherwise meet the reporting requirements under EPCRA section 313. Such facilities can submit an annual certification statement in lieu of a TRI Form R. EPA estimates that facilities will require an average of 34 hours to comply with the requirements for a certification statement, compared to 53 hours for a TRI Form R. The alternate reporting threshold will apply to the chemicals being added under EPCRA section 313 by this rule as well as chemicals currently listed under EPCRA section 313.

EPA's guidelines for implementing the Regulatory Flexibility Act state that "The alternatives considered for the purpose of fulfilling the Act's requirements need not be restricted in applicability to small entities. Regulatory alternatives that prove to be more cost-effective for small entities often will be more cost-effective for larger entities as well. For example, alternatives that place lesser burden on facilities with lower emission levels, lower production levels, etc., should be analyzed in conjunction with fulfilling the Act's requirements even though such alternatives may not ease the burden on all (or even most) small entities and may benefit large entities as well as small ones."

Because EPA has considered, and adopted, an alternative that places lesser burden on facilities with lower emission levels, EPA believes that it has met the requirements of the Regulatory Flexibility Act. The alternate threshold will provide significant relief for small businesses that will report for the proposed chemicals, which is the intent of the Act.

5. *Reporting burden.* Many commenters report that the time required for compliance with EPCRA section 313 is higher than that estimated in the RIA. Commenters estimates of the time required to prepare a TRI Form R and perform the necessary recordkeeping vary from 91 to 2,000 hours, compared with EPA's estimate of 53 hours.

The unit time estimates used by EPA are average values. EPA recognizes that large multi-divisional, multi-departmental facilities may require more than the average time to comply. As with any average, some facilities will be above the average and others will be

below it. However, there are many other facilities subject to the rule that are not large, multi-divisional or multi-departmental. These facilities will typically have a simpler compliance process.

The variability among facilities is evident in comments on the rule submitted by a large chemical manufacturing company, that provided estimates showing that it spends an average of 28 hours for each TRI Form R that is submitted to compile information, perform calculations, prepare the TRI Form R and maintain records. This includes the time spent on compliance determination for chemicals that are below threshold levels. This is less than EPA's estimate of 53 hours for the same activities.

While some of the commenters may require more time than average to comply with the rule, other companies require less time than average. EPA believes that its time estimates are a reasonable average for the manufacturing sector as a whole.

V. Rulemaking Record

The record supporting this final rule is contained in the docket number OPPTS-400082B. All documents, including an index of the docket, are available in the TSCA Nonconfidential Information Center (NCIC), also known as the TSCA Public Document Office, from noon to 4 p.m., Monday through Friday, excluding legal holidays. TSCA NCIC is located at EPA Headquarters, Rm. NE-B607, 401 M St., SW., Washington, DC 20460.

VI. References

- (1) Colborn, T., F.S. fom Saal A.M. Soto. *Developmental Effects of Endocrine-Disrupting Chemicals in Wildlife and Humans*. Environmental Health Perspectives 101:378-384.
- (2) DeRosa, Stara and Durkin *Ranking Chemicals Based on Chronic Toxicity Data, Tox. and Ind. Health*, Vol. 1, No.4, (1985)
- (3) NAS/NRC. *Risk Assessment in the Federal Government: Managing the Process*. National Academy Press, Washington, DC (1983)

(4) USEPA/OHEA. *Risk Assessment Guidelines for Carcinogen Risk*. U.S. Environmental Protection Agency, Cincinnati, OH. (1987).

(5) USEPA/OHEA. *The Risk Assessment Guidelines of 1986*. U.S. Environmental Protection Agency, Washington, DC (1987)

(6) USEPA/OHEA. *Guidelines for Developmental Toxicity Risk Assessment*. U.S. Environmental Protection Agency, Washington, DC (1991) [56 FR 63805]

(7) USEPA/OHEA. *Draft Report: Principles of Neurotoxicity Risk Assessment; Notice*. U.S. Environmental Protection Agency, Washington, DC (1993) [58 FR 41556]

(8) USEPA/OPP. *EPTC-RS-DCI. Evaluation of Two-Year Chronic Rat Study (Accession Nos. 254335, 254336, 254337, 254338, and Addendum to Final Report (Accession Nos. 258076, 260057)*. U. S. Environmental Protection Agency, Washington, DC (1986)

(9) USEPA/OPP. *Support Document for the Addition of Chemicals from Federal Insecticide, Fungicide, Rodenticide Act (FIFRA) Active Ingredients to EPCRA Section 313*. U. S. Environmental Protection Agency, Washington, DC (1993).

(10) USEPA/OPP. *Triallate: Subchronic Neurotoxicity Study in Rats*. U.S. Environmental Protection Agency, Washington, DC (1994).

(11) USEPA/OPPT. *Revised Draft Hazard Assessment Guidelines for Listing Chemicals on the Toxic Release Inventory*. U. S. Environmental Protection Agency, Washington, DC (1992).

(12) USEPA/OPPT. *Support Document for the Addition of Chemicals from Section 112(b) of the Clean Air Act Amendments and Chlorinated Paraffins to EPCRA section 313*. U. S. Environmental Protection Agency, Washington, DC (1993).

(13) USEPA/OPPT. *Support Document for the Health and Ecological Toxicity Review of TRI Expansion Chemicals*. U. S. Environmental Protection Agency, Washington, DC (1993).

(14) USEPA/OPPT. *Response to Comments Received on the January 12, 1994 Proposed Rule to Expand the EPCRA Section 313 List*. U. S. Environmental Protection Agency, Washington, DC (1994).

VII. Regulatory Assessment Requirements

A. Executive Order 12866

Under Executive Order 12866 (58 FR 51735, October 4, 1993) the Agency must determine whether the regulatory action is "significant" and therefore subject to review by the Office of Management and Budget (OMB) and the requirements of the Executive Order. Under section 3(f), the order defines as "significant" those regulatory actions likely to lead to a rule (1) Having an annual effect on the economy of \$100 million or more, or adversely and materially affecting a sector of the economy, productivity, competition, jobs, the environment, public health or safety, or State, local or tribal governments or communities (also referred to as "economically significant"); (2) creating serious inconsistency or otherwise interfering with an action taken or planned by another agency; (3) materially altering the budgetary impacts of entitlements, grants, user fees, or loan programs; or (4) raising novel legal or policy issues arising out of legal mandates, the President's priorities, or the principles set forth in this Executive Order.

EPA has prepared a Regulatory Impact Analysis (RIA) in conjunction with this rulemaking. A copy of this document (titled "Regulatory Impact Analysis of the Final Rule to Add Various Chemicals and Chemical Categories to the EPCRA Section 313 List of Toxic Chemicals") is available in the TSCA NCIC (See Unit V. of this preamble), for review and copying.

EPA has estimated that the total costs to industry of adding the new chemicals to the EPCRA section 313 list is approximately \$99 million in the first year and \$49 million each year thereafter. Costs to EPA are approximately \$1 million per year.

TABLE 2.-SUMMARY OF COST COMPARISON BETWEEN PROPOSED AND FINAL RULE

| | Proposed Rule | Final Rule | Final Rule with Alternate Threshold |
|---------------------------------------------|-----------------|------------------|-------------------------------------|
| Number of chemicals and chemical categories | 313 | 286 ¹ | 286 |
| Number of new facilities | 2,404 | 1,225 | 1,225 |
| Total number of facilities | 7,049 | 3,509 | 3,509 |
| Number of TRI Form Rs submitted | 28,196 | 14,036 | 10,548 |
| Number of annual certifications submitted | 0 | 0 | 3,488 |
| First year industry costs | \$160.4 million | \$99 million | \$92.8 million |

TABLE 2.—SUMMARY OF COST COMPARISON BETWEEN PROPOSED AND FINAL RULE—Continued

| | Proposed Rule | Final Rule | Final Rule with Alternate Threshold |
|--------------------------------|----------------|----------------|-------------------------------------|
| Subsequent year industry costs | \$88.5 million | \$48.8 million | \$44.3 million |
| EPA Costs | \$2.1 million | \$1.1 million | \$0.9 million |

Source-RIA. The results for the alternate threshold are based on a 500 pound level of total waste.

This includes 39 chemicals as part of two delineated categories.

The costs for the final rule are different from the costs for the proposed rule, as shown in Table 2. There are two reasons for this change. First, the number of chemicals and chemical categories added has decreased from 313 to 286, which reduced the number of reports that would be submitted. Second, the number of reports estimated for one chemical, water dissociable nitrate compounds (reportable only when in aqueous solution), was increased from 2,146 to 3,066 to account for facilities that create water dissociable nitrate compounds in aqueous solution through on-site biological treatment of wastewater.

Elsewhere in this issue of the **Federal Register**, EPA is finalizing a rule establishing an alternate threshold for facilities with low amounts of a listed toxic chemical in waste (see Unit II. of this preamble). Qualifying facilities would be eligible to submit an annual certification statement instead of a TRI Form R. Because the time required for the alternate threshold is less than the time required for a TRI Form R, the cost of compliance with this rule will be lowered as a result. The effect of the alternate threshold on the chemicals being added by this rule is demonstrated in Table 2. Further information on the effect of the alternate threshold is presented elsewhere in this issue of the **Federal Register**.

The costs described in Table 2 represent only those actions that are required by this rule. There are other requirements that are linked to reporting under EPCRA section 313, but which are not required by this rule. There are 13 states that place a fee or tax on facilities that file a TRI Form R or report to EPA under EPCRA section 313, and 7 states that mandate pollution prevention plans from such facilities. EPA has also created special requirements for certain facilities with NPDES storm water permits that report under EPCRA section 313.

Adding chemicals and chemical categories to the EPCRA section 313 list may cause some facilities to incur additional costs through these linked

requirements. These costs have not been monetized, but they should not be significant. The linked fees and taxes are transfers, and not social costs, and many of the reporting facilities will not be located in the 13 states with fees and taxes. Also, the NPDES and pollution prevention planning requirements are most likely to create costs for facilities that are new reporters. There will be approximately 1,225 new reporters as a result of this rule, although not all of these will be subject to the NPDES requirements, or be located in states with pollution prevention planning requirements. The linkage to the NPDES requirements is limited to about two dozen of the new chemicals, not all 286 chemicals and chemical categories being added.

The market failure that this rule is intended to correct is the externality created by the lack of information available to citizens about the releases and transfers of toxic chemicals in their communities. Taking no action would allow this externality (and the resultant social costs) to continue. It is expected that this rulemaking will generate benefits by providing citizens with access to information that otherwise would not be available to them. The benefits of the rule itself are limited to improvements in understanding, awareness and decision-making related to the provision and distribution of information.

EPA believes that the rulemaking can reasonably be anticipated to indirectly yield health and environmental benefits by leading to reductions in the releases and transfers of toxic chemicals. These changes in behavior come at some cost to industry. The net benefits of the follow-on activities are the difference between the benefits of decreased chemical releases and transfers, and the costs of the actions needed to achieve them. As noted above, EPA has not quantified the benefits of this rule or the follow-on activities.

This action was submitted to OMB for review, as required by Executive Order 12866, and any comments or changes made in response to OMB suggestions or recommendations have been documented in the public record.

B. Regulatory Flexibility Act

The Regulatory Flexibility Act of 1980 requires each Federal agency to perform a Regulatory Flexibility Analysis for all rules that are likely to have a "significant impact on a substantial number of small entities." EPA investigated the potential impact of the proposed rule on small businesses, and has prepared a Final Regulatory Flexibility Analysis (FRFA). This assessment has been included as part of the RIA and is summarized below.

In assessing small business impacts, EPA calculated the costs incurred by two hypothetical facilities that are supplier notification facilities reporting to the TRI for the first time. Facilities were assumed to file only Form Rs, instead of any annual certification statements. Thus, the results are based on conservative assumptions. The first facility files a report for a single new chemical, while the other files reports for four new chemicals. For each hypothetical facility, annual regulatory costs were calculated and compared to average annual sales.

The cost impact ratios were calculated based on the average annual sales of those facilities currently reporting under EPCRA section 313 for which annual sales and employee figures could be obtained from Dun Bradstreet. The Dun Bradstreet data base was used instead of Census data on the assumption that facilities that report under EPCRA section 313 are not uniformly distributed throughout the entire population of facilities in each size category. EPA believes that it is reasonable to assume that facilities reporting under EPCRA section 313 have, on average, larger annual sales than the typical facility in an industry. Therefore, the annual sales of current reporters should be a more appropriate measure than the sales of all facilities in an industry.

A small business was defined as having fewer than 50 employees. Although a more detailed break-down of size categories would have allowed for a closer examination of the potential impact on even smaller facilities, the total number of observations in the matched data base was too small to allow for additional categories.

EPA often uses a cost impact percentage of one percent as a threshold

measure below which facilities are not considered to be significantly impacted as a result of a regulation. Under the scenario in which facilities are assumed to submit one TRI Form R, the cost impact percentages are well below one percent for all employee size classes in all SICs. The highest cost impact percentage is 0.4 percent for small facilities in Standard Industrial Classification (SIC) codes 25 (Furniture) and 31 (Leather) in the first year of reporting.

Under the scenario in which facilities are assumed to submit four TRI Form Rs, cost impact percentages in the first year of reporting are above one percent only for small facilities in SIC codes 25 (1.2 percent) and 31 (1.1 percent). Cost impact percentages are below 0.8 percent for all industries in subsequent years.

The higher impact rates for the hypothetical facilities occur in industry sectors where there have historically been a relatively small number of establishments reporting. Approximately 8 percent of all facilities in SIC code 25 (Furniture) and 10 percent of all facilities in SIC code 31 (Leather) currently report to EPCRA section 313 (compared to 57 percent of all facilities in the chemical industry). It is reasonable that large and medium businesses are more highly represented in these percentages than small businesses, because they would be more likely to exceed the EPCRA section 313 thresholds. In addition, facilities in SIC 25 and 31 have typically submitted fewer than four reports each, and would be less likely to submit four reports for the new chemicals than facilities in other industries.

Thus, cost impacts for facilities potentially affected by the rule were not found to be of sufficient magnitude to cause significant impacts. Although

EPA has found that the rule does not result in significant impacts on small facilities, EPA has separately developed alternatives to meet the goals of the Regulatory Flexibility Act (i.e., to accomplish the objectives of EPCRA section 313 while minimizing the economic impact on small entities). EPA proposed a rule establishing an alternative reporting threshold for low-level releases and transfers (July 28, 1994, 59 FR 38524). The proposal requested comment on five different levels for the alternate reporting threshold. This rule is being finalized elsewhere in today's issue of the **Federal Register**.

C. Paperwork Reduction Act

The collection of information and other requirements under section 313 of EPCRA and section 6607 of the PPA are covered under OMB approval number 2070-0093, which was issued on May 14, 1992. While this approval normally would have expired on November 30, 1992, it remains in effect pursuant to the 1993 Department of Veteran Affairs and Housing and Urban Development and Independent Agencies Appropriations Act, Pub. L. 102-389, signed October 6, 1992, which states that:

Notwithstanding the Paperwork Reduction Act of 1980 or any requirements thereunder the Environmental Protection Agency Toxic Chemical Release Inventory TRI Form R and instructions, revised 1991 version issued May 19, 1992, and related requirements (OMB No. 2070-0093), shall be effective for reporting under section 6607 of the Pollution Prevention Act of 1990 (Public Law 101-508) and section 313 of the Superfund Amendments and Reauthorization Act of 1986 (Public Law 99-499) until such time as revisions are promulgated pursuant to law.

This final rule adds chemicals to the list of toxic chemicals subject to reporting under section 313 of EPCRA and section 6607 of the PPA and does

not change the elements of the TRI reporting form, its instructions, or related requirements. Accordingly, the TRI Form R and instructions and related requirements remain in effect, as provided by Pub. L. 102-389.

The industry reporting burden for collecting this information is estimated to average 53 hours per respondent annually, including time for reviewing instructions, searching existing data sources, gathering and maintaining the data needed, and completing and reviewing the collection of information. The actual burden to a specific facility may deviate from this estimate depending on the complexity of the facility's operations and the profile of the release.

List of Subjects in 40 CFR Part 372

Environmental protection, Community right-to-know, Reporting and recordkeeping requirements, Toxic chemicals.

Dated: November 22, 1994.

Carol M. Browner,
Administrator.

Therefore, 40 CFR part 372 is amended to read as follows:

Part 372—[AMENDED]

1. The authority citation for part 372 continues to read as follows:

Authority: 42 U.S.C. 11013 and 11028.

2. In § 372.65 by adding chemicals to paragraph (a) alphabetically, to paragraph (b) by CAS no. sequence, and to paragraph (c) by alphabetically adding six categories to read as follows:

§ 372.65 Chemicals and chemical categories to which the part applies.

* * * * *
(a) * * *

| Chemical Name | CAS No. | Effective Date |
|------------------------------------------------------------------------------------------------------|------------|----------------|
| Abamectin [Avermectin B1] | 71751-41-2 | 1/1/95 |
| Acephate (Acetylphosphoramidothioic acid O,S-dimethyl ester) | 30560-19-1 | 1/1/95 |
| Acifluorfen, sodium salt [5-(2-Chloro-4-(trifluoromethyl)phenoxy)-2-nitro-benzoic acid, sodium salt] | 62476-59-9 | 1/1/95 |
| Alachlor | 15972-60-8 | 1/1/95 |
| Aldicarb | 116-06-3 | 1/1/95 |
| d-trans-Allethrin [d-trans-Chrysanthemic acid of d-allethrine] | 28057-48-9 | 1/1/95 |
| Allylamine | 107-11-9 | 1/1/95 |
| Aluminum phosphide | 20859-73-8 | 1/1/95 |
| Ametryn (N-Ethyl-N'-(1-methylethyl)-6-(methylthio)-1,3,5,-triazine-2,4-diamine) | 834-12-8 | 1/1/95 |
| Amitraz | 33089-61-1 | 1/1/95 |

| Chemical Name | CAS No. | Effective Date |
|----------------------------------------------------------------------------------------------------------------------------------|------------|----------------|
| Anilazine [4,6-dichloro-N-(2-chlorophenyl)-1,3,5-triazin-2-amine] | 101-05-3 | 1/1/95 |
| Atrazine (6-Chloro-N-ethyl-N'-(1-methylethyl)-1,3,5,-triazine-2,4-diamine) | 1912-24-9 | 1/1/95 |
| Bendiocarb [2,2-Dimethyl-1,3-benzodioxol-4-ol methylcarbamate] | 22781-23-3 | 1/1/95 |
| Benfluralin (N-Butyl-N-ethyl-2,6-dinitro-4-(trifluoromethyl)benzenamine) | 1861-40-1 | 1/1/95 |
| Benomyl | 17804-35-2 | 1/1/95 |
| Bifenthrin | 82657-04-3 | 1/1/95 |
| Bis(tributyltin) oxide | 56-35-9 | 1/1/95 |
| Boron trichloride | 10294-34-5 | 1/1/95 |
| Boron trifluoride | 7637-07-2 | 1/1/95 |
| Bromacil (5-Bromo-6-methyl-3-(1-methylpropyl)-2,4-(1H,3H)-pyrimidinedione) | 314-40-9 | 1/1/95 |
| Bromacil, lithium salt [2,4-(1H,3H)-Pyrimidinedione, 5-bromo-6-methyl-3-(1-methylpropyl), lithium salt] | 53404-19-6 | 1/1/95 |
| Bromine | 7726-95-6 | 1/1/95 |
| 1-Bromo-1-(bromomethyl)-1,3-propanedicarbonitrile | 35691-65-7 | 1/1/95 |
| 2-Bromo-2-nitropropane-1,3-diol (Bronopol) | 52-51-7 | 1/1/95 |
| Bromoxynil (3,5-Dibromo-4-hydroxybenzoxynitrile) | 1689-84-5 | 1/1/95 |
| Bromoxynil octanoate (Octanoic acid, 2,6-dibromo-4-cyanophenyl ester) | 1689-99-2 | 1/1/95 |
| Brucine | 357-57-3 | 1/1/95 |
| C.I. Acid Red 114 | 6459-94-5 | 1/1/95 |
| C.I. Direct Blue 218 | 28407-37-6 | 1/1/95 |
| Carbofuran | 1563-66-2 | 1/1/95 |
| Carboxin (5,6-Dihydro-2-methyl-N-phenyl-1,4-oxathiin-3-carboxamide) | 5234-68-4 | 1/1/95 |
| Chinomethionat [6-Methyl-1,3-dithiolo[4,5-b]quinoxalin-2-one] | 2439-01-2 | 1/1/95 |
| Chlorendic acid | 115-28-6 | 1/1/95 |
| Chlorimuron ethyl [Ethyl-2-[[[(4-chloro-6-methoxyprimidin-2-yl)-carbonyl]-amino]sulfonyl]benzoate] | 90982-32-4 | 1/1/95 |
| 1-(3-Chloroallyl)-3,5,7-triaza-1-azoniaadamantane chloride | 4080-31-3 | 1/1/95 |
| p-Chloroaniline | 106-47-8 | 1/1/95 |
| 3-Chloro-2-methyl-1-propene | 563-47-3 | 1/1/95 |
| p-Chlorophenyl isocyanate | 104-12-1 | 1/1/95 |
| Chloropicrin | 76-06-2 | 1/1/95 |
| 3-Chloropropionitrile | 542-76-7 | 1/1/95 |
| p-Chloro-o-toluidine | 95-69-2 | 1/1/95 |
| 2-Chloro-1,1,1-trifluoro-ethane (HCFC-133a) | 75-88-7 | 1/1/95 |
| Chlorotrifluoromethane (CFC-13) | 75-72-9 | 1/1/95 |
| 3-Chloro-1,1,1-trifluoro-propane (HCFC-253fb) | 460-35-5 | 1/1/95 |
| Chlorpyrifos methyl [O,O-dimethyl-O-(3,5,6-trichloro-2-pyridyl)phosphorothioate] | 5598-13-0 | 1/1/95 |
| Chlorsulfuron [2-chloro-N-[[4-methoxy-6-methyl-1,3,5-triazin-2-yl]amino]carbonyl]benzenesulfonamide] | 64902-72-3 | 1/1/95 |
| Crotonaldehyde | 4170-30-3 | 1/1/95 |
| Cyanazine | 21725-46-2 | 1/1/95 |
| Cycloate | 1134-23-2 | 1/1/95 |
| Cyclohexanol | 108-93-0 | 1/1/95 |
| Cyfluthrin [3-(2,2-Dichloroethenyl)-2,2-dimethylcyclopropanecarboxylic acid, cyano(4-fluoro-3-phenoxyphenyl)methyl ester] | 68359-37-5 | 1/1/95 |
| Cyhalothrin [3-(2-Chloro-3,3,3-trifluoro-1-propenyl)-2,2-dimethylcyclopropanecarboxylic acid cyano(3-phenoxyphenyl)methyl ester] | 68085-85-8 | 1/1/95 |

| Chemical Name | CAS No. | Effective Date |
|-----------------------------------------------------------------------------------------------|-------------|----------------|
| Dazomet(Tetrahydro-3,5-dimethyl-2H-1,3,5-thiadiazine-2-thione) | 533-74-4 | 1/1/95 |
| Dazomet, sodium salt [Tetrahydro-3,5-dimethyl-2H-1,3,5-thiadiazine-2-thione, ion(1-), sodium] | 53404-60-7 | 1/1/95 |
| 2,4,-DB | 94-82-6 | 1/1/95 |
| 2,4-D butoxyethyl ester | 1929-73-3 | 1/1/95 |
| 2,4-D butyl ester | 94-80-4 | 1/1/95 |
| 2,4-D chlorocrotyl ester | 2971-38-2 | 1/1/95 |
| Desmedipham | 13684-56-5 | 1/1/95 |
| 2,4-D 2-ethylhexyl ester | 1928-43-4 | 1/1/95 |
| 2,4-D 2-ethyl-4-methylpentyl ester | 53404-37-8 | 1/1/95 |
| Diazinon | 333-41-5 | 1/1/95 |
| 2,2-Dibromo-3-nitropropionamide | 10222-01-2 | 1/1/95 |
| Dicamba (3,6-Dichloro-2-methoxybenzoic acid) | 1918-00-9 | 1/1/95 |
| Dichloran [2,6-Dichloro-4-nitroaniline] | 99-30-9 | 1/1/95 |
| 3,3'-Dichlorobenzidine dihydrochloride | 612-83-9 | 1/1/95 |
| 3,3'-Dichlorobenzidine sulfate | 64969-34-2 | 1/1/95 |
| trans-1,4-Dichloro-2-butene | 110-57-6 | 1/1/95 |
| 1,2-Dichloro-1,1-difluoroethane (HCFC-132b) | 1649-08-7 | 1/1/95 |
| Dichlorofluoromethane (HCFC-21) | 75-43-4 | 1/1/95 |
| Dichloropentafluoropropane | 127564-92-5 | 1/1/95 |
| 1,1-dichloro-1,2,2,3,3-pentafluoropropane (HCFC-225cc) | 13474-88-9 | 1/1/95 |
| 1,1-dichloro-1,2,3,3,3-pentafluoropropane (HCFC-225eb) | 111512-56-2 | 1/1/95 |
| 1,2-dichloro-1,1,2,3,3-pentafluoropropane (HCFC-225bb) | 422-44-6 | 1/1/95 |
| 1,2-dichloro-1,1,3,3,3-pentafluoropropane (HCFC-225da) | 431-86-7 | 1/1/95 |
| 1,3-dichloro-1,1,2,2,3-pentafluoropropane (HCFC-225cb) | 507-55-1 | 1/1/95 |
| 1,3-dichloro-1,1,2,3,3-pentafluoropropane (HCFC-225ea) | 136013-79-1 | 1/1/95 |
| 2,2-dichloro-1,1,1,3,3-pentafluoropropane (HCFC-225aa) | 128903-21-9 | 1/1/95 |
| 2,3-dichloro-1,1,1,2,3-pentafluoropropane (HCFC-225ba) | 422-48-0 | 1/1/95 |
| 3,3-dichloro-1,1,1,2,2-pentafluoropropane (HCFC-225ca) | 422-56-0 | 1/1/95 |
| Dichlorophene [2,2'-Methylene-bis(4-chlorophenol)] | 97-23-4 | 1/1/95 |
| trans-1,3-Dichloropropene | 10061-02-6 | 1/1/95 |
| Diclofop methyl [2-[4-(2,4-Dichlorophenoxy)phenoxy]propanoic acid, methyl ester] | 51338-27-3 | 1/1/95 |
| Dicyclopentadiene | 77-73-6 | 1/1/95 |
| Diethyl ethyl | 38727-55-8 | 1/1/95 |
| Diflubenzuron | 35367-38-5 | 1/1/95 |
| Diglycidyl resorcinol ether | 101-90-6 | 1/1/95 |
| Dimethipin [2,3,-Dihydro-5,6-dimethyl-1,4-dithiin-1,1,4,4-tetraoxide] | 55290-64-7 | 1/1/95 |
| Dimethoate | 60-51-5 | 1/1/95 |
| 3,3'-Dimethoxybenzidine dihydrochloride (o-Dianisidine dihydrochloride) | 20325-40-0 | 1/1/95 |
| 3,3'-Dimethoxybenzidine hydrochloride (o-Dianisidine hydrochloride) | 111984-09-9 | 1/1/95 |
| Dimethylamine | 124-40-3 | 1/1/95 |
| Dimethylamine dicamba | 2300-66-5 | 1/1/95 |
| 3,3'-Dimethylbenzidine dihydrochloride (o-Tolidine dihydrochloride) | 612-82-8 | 1/1/95 |
| 3,3'-Dimethylbenzidine dihydrofluoride (o-Tolidine dihydrofluoride) | 41766-75-0 | 1/1/95 |
| Dimethyl chlorothiophosphate | 2524-03-0 | 1/1/95 |
| Dimethyldichlorosilane | 75-78-5 | 1/1/95 |
| N,N-Dimethylformamide | 68-12-2 | 1/1/95 |
| 2,6-Dimethylphenol | 576-26-1 | 1/1/95 |

| Chemical Name | CAS No. | Effective Date |
|-----------------------------------------------------------------------------------------------------------------------------|------------|----------------|
| Dinitrobutyl phenol (Dinoseb) | 88-85-7 | 1/1/95 |
| Dinocap | 39300-45-3 | 1/1/95 |
| Diphenamid | 957-51-7 | 1/1/95 |
| Diphenylamine | 122-39-4 | 1/1/95 |
| Dipotassium endothall [7-Oxabicyclo(2.2.1)heptane-2,3-dicarboxylic acid, dipotassium salt] | 2164-07-0 | 1/1/95 |
| Dipropyl isocinchomerate | 136-45-8 | 1/1/95 |
| Disodium cyanodithioimidocarbonate | 138-93-2 | 1/1/95 |
| 2,4-D isopropyl ester | 94-11-1 | 1/1/95 |
| 2,4-Dithiobiuret | 541-53-7 | 1/1/95 |
| Diuron | 330-54-1 | 1/1/95 |
| Dodine [Dodecylguanidine monoacetate] | 2439-10-3 | 1/1/95 |
| 2,4,-DP | 120-36-5 | 1/1/95 |
| 2,4-D propylene glycol butyl ether ester | 1320-18-9 | 1/1/95 |
| 2,4-D sodium salt | 2702-72-9 | 1/1/95 |
| Ethoprop [Phosphorodithioic acid O-ethyl S,S-dipropyl ester] | 13194-48-4 | 1/1/95 |
| Ethyl dipropylthiocarbamate [EPTC] | 759-94-4 | 1/1/95 |
| Famphur | 52-85-7 | 1/1/95 |
| Fenarimol [.alpha.-(2-Chlorophenyl)-.alpha.-4-chlorophenyl]-5-pyrimidinemethanol] | 60168-88-9 | 1/1/95 |
| Fenbutatin oxide (Hexakis(2-methyl-2-phenyl-propyl)distannoxane) | 13356-08-6 | 1/1/95 |
| Fenoxaprop ethyl [2-(4-((6-Chloro-2-benzoxazolyl)oxy)phenoxy)propanoic acid,ethyl ester] | 66441-23-4 | 1/1/95 |
| Fenoxycarb [2-(4-Phenoxyphenoxy)ethyl]carbamic acid ethyl ester] | 72490-01-8 | 1/1/95 |
| Fenpropathrin [2,2,3,3-Tetramethylcyclopropane carboxylic acid cyano(3-phenoxy-phenyl)methyl ester] | 39515-41-8 | 1/1/95 |
| Fenthion [O,O-Dimethyl O-[3-methyl-4-(methylthio)phenyl]ester, phosphorothioic acid] | 55-38-9 | 1/1/95 |
| Fenvalerate [4-Chloro-alpha-(1-methylethyl)benzeneacetic acid cyano(3-phenoxyphenyl)methyl ester] | 51630-58-1 | 1/1/95 |
| Ferbam [Tris(dimethylcarbamio-dithioato-S,S')iron] | 14484-64-1 | 1/1/95 |
| Fluazifop-butyl [2-[4-[[5-(Trifluoromethyl)-2-pyridinyl]oxy]-phenoxy]propanoic acid, butyl ester] | 69806-50-4 | 1/1/95 |
| Fluorine | 7782-41-4 | 1/1/95 |
| Fluorouracil (5-Fluorouracil) | 51-21-8 | 1/1/95 |
| Fluvalinate [N-[2-Chloro-4-(trifluoromethyl)phenyl]-DL-valine(+)-cyano (3-phenoxyphenyl)methyl ester] | 69409-94-5 | 1/1/95 |
| Folpet | 133-07-3 | 1/1/95 |
| Fomesafen [5-(2-Chloro-4-(trifluoromethyl)phenoxy)-N-methylsulfonyl]-2-nitrobenzamide] | 72178-02-0 | 1/1/95 |
| alpha-Hexachlorocyclohexane | 319-84-6 | 1/1/95 |
| n-Hexane | 110-54-3 | 1/1/95 |
| Hexazinone | 51235-04-2 | 1/1/95 |
| Hydramethylnon [Tetrahydro-5,5-dimethyl-2(1H)-pyrimidinone[3-[4-(trifluoromethyl)phenyl]ethenyl]-2-propenylidene]hydrazone] | 67485-29-4 | 1/1/95 |
| Imazalil [1-[2-(2,4-Dichlorophenyl)-2-(2-propenyloxy)ethyl]-1H-imidazole] | 35554-44-0 | 1/1/95 |
| 3-Iodo-2-propynyl butylcarbamate | 55406-53-6 | 1/1/95 |
| Iron pentacarbonyl | 13463-40-6 | 1/1/95 |
| Isodrin | 465-73-6 | 1/1/95 |
| Isofenphos [2-[[Ethoxy[(1-methylethyl)amino]phosphinothioyl]oxy]benzoic acid 1-methylethyl ester] | 25311-71-1 | 1/1/95 |
| Lactofen [5-(2-Chloro-4-(trifluoromethyl)phenoxy)-2-nitro-2-ethoxy-1-methyl-2-oxoethyl ester] | 77501-63-4 | 1/1/95 |
| Linuron | 330-55-2 | 1/1/95 |
| Lithium carbonate | 554-13-2 | 1/1/95 |
| Malathion | 121-75-5 | 1/1/95 |
| Mecoprop | 93-65-2 | 1/1/95 |
| 2-Mercaptobenzothiazole (MBT) | 149-30-4 | 1/1/95 |
| Merphos | 150-50-5 | 1/1/95 |
| Metham sodium (Sodium methylthiocarbamate) | 137-42-8 | 1/1/95 |
| Methazole [2-(3,4-Dichlorophenyl)-4-methyl-1,2,4-oxadiazolidine-3,5-dione] | 20354-26-1 | 1/1/95 |
| Methiocarb | 2032-65-7 | 1/1/95 |
| Methoxone (4-Chloro-2-methylphenoxy) acetic acid (MCPA) | 94-74-6 | 1/1/95 |

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| Methoxone-sodium salt ((4-chloro-2-methylphenoxy) acetate sodium salt) | 3653-48-3 | 1/1/95 |
| Methyl isothiocyanate [isothiocyanatomethane] | 556-61-6 | 1/1/95 |
| 2-Methylacetonitrile | 75-86-5 | 1/1/95 |
| N-Methylolacrylamide | 924-42-5 | 1/1/95 |
| Methyl parathion | 298-00-0 | 1/1/95 |
| N-Methyl-2-pyrrolidone | 872-50-4 | 1/1/95 |
| Methyltrichlorosilane | 75-79-6 | 1/1/95 |
| Metiram | 9006-42-2 | 1/1/95 |
| Metribuzin | 21087-64-5 | 1/1/95 |
| Mevinphos | 7786-34-7 | 1/1/95 |
| Molinate (1H-Azepine-1-carbothioic acid, hexahydro-S-ethyl ester) | 2212-67-1 | 1/1/95 |
| Monuron | 150-68-5 | 1/1/95 |
| Myclobutanil [.alpha.-Butyl-.alpha.-(4-chlorophenyl)-1H-1,2,4-triazole-1-propanenitrile] | 88671-89-0 | 1/1/95 |
| Nabam | 142-59-6 | 1/1/95 |
| Naled | 300-76-5 | 1/1/95 |
| Nitrapyrin (2-Chloro-6-(trichloromethyl) pyridine) | 1929-82-4 | 1/1/95 |
| p-Nitroaniline | 100-01-6 | 1/1/95 |
| Norflurazon [4-Chloro-5-(methylamino)-2-(3-(trifluoromethyl)phenyl)-3(2H)-pyridazinone] | 27314-13-2 | 1/1/95 |
| Oryzalin [4-(Dipropylamino)-3,5-dinitrobenzenesulfonamide] | 19044-88-3 | 1/1/95 |
| Oxydemeton methyl [S-(2-(ethylsulfanyl)ethyl) o,o-dimethyl ester phosphorothioic acid] | 301-12-2 | 1/1/95 |
| Oxydiazon [3-[2,4-Dichloro-5-(1-methylethoxy)phenyl]-5-(1,1-dimethylethyl)-1,3,4-oxadiazol-2(3H)-one] | 19666-30-9 | 1/1/95 |
| Oxyfluorfen | 42874-03-3 | 1/1/95 |
| Ozone | 10028-15-6 | 1/1/95 |
| Paraquat dichloride | 1910-42-5 | 1/1/95 |
| Pebulate [Butylethylcarbamothioic acid S-propyl ester] | 1114-71-2 | 1/1/95 |
| Pendimethalin [N-(1-Ethylpropyl)-3,4-dimethyl-2,6-dinitrobenzenamine] | 40487-42-1 | 1/1/95 |
| Pentobarbital sodium | 57-33-0 | 1/1/95 |
| Perchloromethyl mercaptan | 594-42-3 | 1/1/95 |
| Permethrin [3-(2,2-Dichloroethenyl)-2,2-dimethylcyclopropanecarboxylic acid, (3-phenoxyphenyl)methyl ester] | 52645-53-1 | 1/1/95 |
| Phenanthrene | 85-01-8 | 1/1/95 |
| Phenothrin [2,2-Dimethyl-3-(2-methyl-1-propenyl)cyclopropanecarboxylic acid (3-phenoxyphenyl)methyl ester] | 26002-80-2 | 1/1/95 |
| 1,2-Phenylenediamine | 95-54-5 | 1/1/95 |
| 1,3-Phenylenediamine | 108-45-2 | 1/1/95 |
| 1,2-Phenylenediamine dihydrochloride | 615-28-1 | 1/1/95 |
| 1,4-Phenylenediamine dihydrochloride | 624-18-0 | 1/1/95 |
| Phenytoin | 57-41-0 | 1/1/95 |
| Phosphine | 7803-51-2 | 1/1/95 |
| Picloram | 1918-02-1 | 1/1/95 |
| Piperonyl butoxide | 51-03-6 | 1/1/95 |
| Pirimiphos methyl [O-(2-(Diethylamino)-6-methyl-4-pyrimidinyl)-O,O-dimethylphosphorothioate] | 29232-93-7 | 1/1/95 |
| Potassium bromate | 7758-01-2 | 1/1/95 |
| Potassium dimethyldithiocarbamate | 128-03-0 | 1/1/95 |

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| Potassium N-methyldithiocarbamate | 137-41-7 | 1/1/95 |
| Profenofos [O-(4-Bromo-2-chlorophenyl)-O-ethyl-S-propyl phosphorothioate] | 41198-08-7 | 1/1/95 |
| Prometryn [N,N'-Bis(1-methylethyl)-6-methylthio-1,3,5-triazine-2,4-diamine] | 7287-19-6 | 1/1/95 |
| Propachlor [2-Chloro-N-(1-methylethyl)-N-phenylacetamide] | 1918-16-7 | 1/1/95 |
| Propanil [N-(3,4-Dichlorophenyl)propanamide] | 709-98-8 | 1/1/95 |
| Propargyl alcohol | 2312-35-8 | 1/1/95 |
| Propetamphos [3-[[[(Ethylamino)methoxyphosphinothioyl]oxy]-2-butenic acid, 1-methylethyl ester] | 107-19-7 | 1/1/95 |
| Propiconazole [1-[2-(2,4-Dichlorophenyl)-4-propyl-1,3-dioxolan-2-yl]-methyl-1H-1,2,4-triazole] | 31218-83-4 | 1/1/95 |
| Quizalofop-ethyl [2-[4-[(6-Chloro-2-quinoxalyl)oxy]phenoxy]propanoic acid ethyl ester] | 76578-14-8 | 1/1/95 |
| Resmethrin [[5-(Phenylmethyl)-3-furanyl]methyl 2,2-dimethyl-3-(2-methyl-1-propenyl)cyclopropanecarboxylate] | 10453-86-8 | 1/1/95 |
| Sethoxydim [2-[1-(Ethoxyimino)butyl]-5-[2-(ethylthio)propyl]-3-hydroxy-2-cyclohexen-1-one] | 74051-80-2 | 1/1/95 |
| Simazine | 122-34-9 | 1/1/95 |
| Sodium azide | 26628-22-8 | 1/1/95 |
| Sodium dicamba [3,6-Dichloro-2-methoxybenzoic acid, sodium salt] | 1982-69-0 | 1/1/95 |
| Sodium dimethyldithiocarbamate | 128-04-1 | 1/1/95 |
| Sodium fluoroacetate | 62-74-8 | 1/1/95 |
| Sodium nitrite | 7632-00-0 | 1/1/95 |
| Sodium pentachlorophenate | 131-52-2 | 1/1/95 |
| Sodium o-phenylphenoxide | 132-27-4 | 1/1/95 |
| Sulfuryl fluoride [Vikane] | 2699-79-8 | 1/1/95 |
| Sulprofos [O-Ethyl O-[4-(methylthio)phenyl]phosphorodithioic acid S-propyl ester] | 35400-43-2 | 1/1/95 |
| Tebuthiuron [N-[5-(1,1-Dimethylethyl)-1,3,4-thiadiazol-2-yl]-N,N'-dimethylurea] | 34014-18-1 | 1/1/95 |
| Temphos | 3383-96-8 | 1/1/95 |
| Terbacil [5-Chloro-3-(1,1-dimethylethyl)-6-methyl-2,4(1H,3H)-pyrimidinedione] | 5902-51-2 | 1/1/95 |
| 1,1,1,2-Tetrachloro-2-fluoroethane (HCFC-121a) | 354-11-0 | 1/1/95 |
| 1,1,2,2-Tetrachloro-1-fluoroethane (HCFC-121) | 354-14-3 | 1/1/95 |
| Tetracycline hydrochloride | 64-75-5 | 1/1/95 |
| Tetramethrin [2,2-Dimethyl-3-(2-methyl-1-propenyl)cyclopropanecarboxylic acid (1,3,4,5,6,7-hexahydro-1,3-dioxo-2H-isoindol-2-yl)methyl ester] | 7696-12-0 | 1/1/95 |
| Thiabendazole [2-(4-Thiazolyl)-1H-benzimidazole] | 148-79-8 | 1/1/95 |
| Thiobencarb [Carbamic acid, diethylthio-, s-(p-chlorobenzyl)] | 28249-77-6 | 1/1/95 |
| Thiodicarb | 59669-26-0 | 1/1/95 |
| Thiophanate ethyl [[1,2-Phenylenebis(iminocarbonothioyl)]biscarbamic acid diethyl ester] | 23564-06-9 | 1/1/95 |
| Thiophanate-methyl | 23564-05-8 | 1/1/95 |
| Thiosemicarbazide | 79-19-6 | 1/1/95 |
| Triadimefon [1-(4-Chlorophenoxy)-3,3-dimethyl-1-(1H-1,2,4-triazol-1-yl)-2-butanone] | 43121-43-3 | 1/1/95 |
| Triallate | 2303-17-5 | 1/1/95 |
| Tribenuron methyl [2-((((4-Methoxy-6-methyl-1,3,5-triazin-2-yl)-methylamino)carbonyl)amino)sulfonyl)-methyl ester] | 101200-48-0 | 1/1/95 |
| Tributyltin fluoride | 1983-10-4 | 1/1/95 |
| Tributyltin methacrylate | 2155-70-6 | 1/1/95 |
| S,S,S-Tributyltrithiophosphate (DEF) | 78-48-8 | 1/1/95 |
| Trichloroacetyl chloride | 76-02-8 | 1/1/95 |
| 1,2,3-Trichloropropane | 96-18-4 | 1/1/95 |
| Triclopyr, triethylammonium salt | 57213-69-1 | 1/1/95 |
| Triethylamine | 121-44-8 | 1/1/95 |
| Triflorine [N,N'-[1,4-Piperazinediyl-bis(2,2,2-trichloroethylidene)] bisformamide] | 26644-46-2 | 1/1/95 |
| Trimethylchlorosilane | 75-77-4 | 1/1/95 |
| 2,3,5-Trimethylphenyl methylcarbamate | 2655-15-4 | 1/1/95 |
| Triphenyltin chloride | 639-58-7 | 1/1/95 |

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| Triphenyltin hydroxide | 76-87-9 | 1/1/95 |
| Vinclozolin [3-(3,5-Dichlorophenyl)-5-ethenyl-5-methyl-2,4-oxazolidinedione] | 50471-44-8 | 1/1/95 |

(b) * * *

| CAS No. | Chemical Name | Effective Date |
|--------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|------------------------------------------------------------------------------|
| 51-03-6 51-21-8 | Piperonyl butoxide Fluorouracil (5-Fluorouracil) | 1/1/95 1/1/95 |
| 52-51-7 | 2-Bromo-2-nitropropane-1,3-diol (Bronopol) | 1/1/95 |
| 52-85-7 | Famphur | 1/1/95 |
| 55-38-9 | Fenthion [O,O-Dimethyl O-[3-methyl-4-(methylthio)phenyl] ester, phosphorothioic acid] | 1/1/95 |
| 56-35-9 | Bis(tributyltin) oxide | 1/1/95 |
| 57-33-0 57-41-0 | Pentobarbital sodium Phenytoin | 1/1/95 1/1/95 |
| 60-51-5 | Dimethoate | 1/1/95 |
| 62-74-8 | Sodium fluoroacetate | 1/1/95 |
| 64-75-5 | Tetracycline hydrochloride | 1/1/95 |
| 68-12-2 | N,N-Dimethylformamide | 1/1/95 |
| 75-43-4 | Dichlorofluoromethane (HCFC-21) | 1/1/95 |
| 75-72-9 75-77-4 75-78-5 75-79-6 75-86-5 75-88-7 76-02-8 76-06-2 | Chlorotrifluoromethane (CFC-13) Trimethylchlorosilane Dimethyldichlorosilane Methyltrichlorosilane 2-Methylacetonitrile 2-Chloro-1,1,1-trifluoroethane (HCFC-133a) Trichloroacetyl chloride Chloropicrin | 1/1/95 1/1/95 1/1/95 1/1/95 1/1/95 1/1/95 1/1/95 1/1/95 |
| 76-87-9 | Triphenyltin hydroxide | 1/1/95 |
| 77-73-6 | Dicyclopentadiene | 1/1/95 |
| 78-48-8 | S,S,S-Tributyltrithiophosphate (DEF) | 1/1/95 |
| 79-19-6 | Thiosemicarbazide | 1/1/95 |
| 85-01-8 | Phenanthrene | 1/1/95 |
| 88-85-7 | Dinitrobutyl phenol (Dinoseb) | 1/1/95 |
| 93-65-2 94-11-1 | Mecoprop 2,4-D isopropyl ester | 1/1/95 1/1/95 |

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| 94-74-6 | Methoxone (4-Chloro-2-methylphenoxy) acetic acid (MCPA) | 1/1/95 |
| 94-80-4 | 2,4-D butyl ester | 1/1/95 |
| 94-82-6 | 2,4-DB | 1/1/95 |
| 95-54-5 | 1,2-Phenylenediamine | 1/1/95 |
| 95-69-2 | p-Chloro-o-toluidine | 1/1/95 |
| 96-18-4 | 1,2,3-Trichloropropane | 1/1/95 |
| 97-23-4 | Dichlorophene [2,2'-Methylene-bis(4-chlorophenol)] | 1/1/95 |
| 99-30-9 | Dichloran [2,6-Dichloro-4-nitroaniline] | 1/1/95 |
| 100-01-6 | p-Nitroaniline | 1/1/95 |
| 101-05-3 | Anilazine [4,6-dichloro-N-(2-chlorophenyl)-1,3,5-triazin-2-amine] | 1/1/95 |
| 101-90-6 | Diglycidyl resorcinol ether | 1/1/95 |
| 104-12-1 | p-Chlorophenyl isocyanate | 1/1/95 |
| 106-47-8 | p-Chloroaniline | 1/1/95 |
| 107-11-9 | Allylamine | 1/1/95 |
| 107-19-7 | Propargyl alcohol | 1/1/95 |
| 108-45-2 | 1,3-Phenylenediamine | 1/1/95 |
| 108-93-0 | Cyclohexanol | 1/1/95 |
| 110-54-3 | n-Hexane | 1/1/95 |
| 110-57-6 | trans-1,4-Dichloro-2-butene | 1/1/95 |
| 115-28-6 | Chlorendic acid | 1/1/95 |
| 116-06-3 | Aldicarb | 1/1/95 |
| 120-36-5 | 2,4-DP | 1/1/95 |
| 121-44-8 | Triethylamine | 1/1/95 |
| 121-75-5 | Malathion | 1/1/95 |
| 122-34-9 | Simazine | 1/1/95 |
| 122-39-4 | Diphenylamine | 1/1/95 |
| 124-40-3 | Dimethylamine | 1/1/95 |
| 128-03-0 | Potassium dimethyldithiocarbamate | 1/1/95 |
| 128-04-1 | Sodium dimethyldithiocarbamate | 1/1/95 |
| 131-52-2 | Sodium pentachlorophenate | 1/1/95 |
| 132-27-4 | Sodium o-phenylphenoxide | 1/1/95 |
| 133-07-3 | Folpet | 1/1/95 |

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| 136-45-8 | Dipropyl isocinchomerate | 1/1/95 |
| 137-41-7 | Potassium n-methyldithiocarbamate | 1/1/95 |
| 137-42-8 | Metham Sodium | 1/1/95 |
| 138-93-2 | Disodium cyanodithioimidocarbonate | 1/1/95 |
| 142-59-6 | Nabam | 1/1/95 |
| 148-79-8 | Thiabendazole [2-(4-Thiazolyl)-1H-benzimidazole] | 1/1/95 |
| 149-30-4 | 2-Mercaptobenzothiazole | 1/1/95 |
| 150-50-5 | Merphos | 1/1/95 |
| 150-68-5 | Monuron | 1/1/95 |
| 298-00-0 | Methyl parathion | 1/1/95 |
| 300-76-5 | Naled | 1/1/95 |
| 301-12-2 | Oxydemeton methyl [s-(2-(Ethylsulfanyl)ethyl)o,o-dimethyl ester phosphorothioic acid] | 1/1/95 |
| 314-40-9 | Bromacil (5-Bromo-6-methyl-3-(1-methylpropyl)-2,4-(1H,3H)-pyrimidinedione) | 1/1/95 |
| 319-84-6 | alpha-Hexachlorocyclohexane | 1/1/95 |
| 330-54-1 | Diuron | 1/1/95 |
| 330-55-2 | Linuron | 1/1/95 |
| 333-41-5 | Diazinon | 1/1/95 |
| 354-11-0 | 1,1,1,2-Tetrachloro-2-fluoroethane (HCFC-121a) | 1/1/95 |
| 354-14-3 | 1,1,2,2-Tetrachloro-1-fluoroethane (HCFC-121) | 1/1/95 |
| 357-57-3 | Brucine | 1/1/95 |
| 422-44-6 | 1,2-dichloro-1,1,2,3,3-pentafluoropropane (HCFC-225bb) | 1/1/95 |
| 422-48-0 | 2,3-dichloro-1,1,1,2,3-pentafluoropropane (HCFC-225ba) | 1/1/95 |
| 422-56-0 | 3,3-dichloro-1,1,1,2,2-pentafluoropropane (HCFC-225ca) | 1/1/95 |
| 431-86-7 | 1,2-dichloro-1,1,3,3,3-pentafluoropropane (HCFC-225da) | 1/1/95 |
| 460-35-5 | 3-chloro-1,1,1-trifluoropropane (HCFC-253fb) | 1/1/95 |
| 465-73-6 | Isodrin | 1/1/95 |
| 507-55-1 | 1,3-dichloro-1,1,2,2,3-pentafluoropropane (HCFC-225cb) | 1/1/95 |
| 533-74-4 | Dazomet (Tetrahydro-3,5-dimethyl-2H-1,3,5-thiadiazine-2-thione) | 1/1/95 |
| 541-53-7 | 2,4-Dithiobiuret | 1/1/95 |
| 542-76-7 | 3-Chloropropionitrile | 1/1/95 |
| 554-13-2 | Lithium carbonate | 1/1/95 |
| 556-61-6 | Methyl isothiocyanate [Isothiocyanatomethane] | 1/1/95 |
| 563-47-3 | 3-Chloro-2-methyl-1-propene | 1/1/95 |
| 576-26-1 | 2,6-Dimethylphenol | 1/1/95 |
| 594-42-3 | Perchloromethyl mercaptan | 1/1/95 |
| 612-82-8 | 3,3'-Dimethylbenzidine dihydrochloride (o-Tolidine dihydrochloride) | 1/1/95 |
| 612-83-9 | 3,3'-Dichlorobenzidine dihydrochloride | 1/1/95 |
| 615-28-1 | 1,2-Phenylenediamine dihydrochloride | 1/1/95 |
| 624-18-0 | 1,4-Phenylenediamine dihydrochloride | 1/1/95 |
| 639-58-7 | Triphenyltin chloride | 1/1/95 |
| 709-98-8 | Propanil [N-(3,4-Dichlorophenyl)propanamide] | 1/1/95 |
| 759-94-4 | Ethyl dipropylthiocarbamate (EPTC) | 1/1/95 |
| 834-12-8 | Ametryn (N-Ethyl-N'-(1-methylethyl)-6-(methylthio)-1,3,5,-triazine-2,4-diamine) | 1/1/95 |
| 872-50-4 | N-Methyl-2-pyrrolidone | 1/1/95 |

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| 924-42-5 957-51-7 | N-Methylacrylamide Diphenamid | 1/1/95 1/1/95 |
| 1114-71-2 | Pebulate [Butylethylcarbamo-thioic acid S-propyl ester] | 1/1/95 |
| 1134-23-2 | Cycloate | 1/1/95 |
| 1320-18-9 | 2,4-D propylene glycol butyl ether ester | 1/1/95 |
| 1563-66-2 1649-08-7 1689-84-5 1689-99-2 | Carbofuran 1,2-dichloro-1,1-difluoroethane (HCFC-132b) Bromoxynil (3,5-Dibromo-4-hydroxybenzotrile) Bromoxynil octanoate (Octanoic acid, 2,6-dibromo-4-cyanophenyl ester) | 1/1/95 1/1/95 1/1/95 1/1/95 |
| 1861-40-1 | Benfluralin(N-Butyl-N-ethyl-2,6-dinitro-4-(trifluoromethyl)benzenamine) | 1/1/95 |
| 1910-42-5 1912-24-9 1918-00-9 1918-02-1 1918-16-7 1928-43-4 1929-73-3 1929-82-4 | Paraquat dichloride Atrazine (6-Chloro-N-ethyl-N'-(1-methylethyl)-1,3,5,-triazine-2,4-diamine) Dicamba (3,6-Dichloro-2-methoxybenzoic acid) Picloram Propachlor [2-Chloro-N-(1-methylethyl)-N-phenylacetamide] 2,4-D 2-ethylhexyl ester 2,4-D butoxyethyl ester Nitrapyrin (2-Chloro-6-(trichloromethyl)pyridine) | 1/1/95 1/1/95 1/1/95 1/1/95 1/1/95 1/1/95 1/1/95 1/1/95 |
| 1982-69-0 1983-10-4 2032-65-7 2155-70-6 2164-07-0 | Sodium dicamba [3,6-Dichloro-2-methoxybenzoic acid, sodium salt] Tributyltin fluoride Methiocarb Tributyltin methacrylate Dipotassium endothall [7-Oxabicyclo(2.2.1)heptane-2,3-dicarboxylic acid, dipotassium salt] | 1/1/95 1/1/95 1/1/95 1/1/95 1/1/95 |
| 2212-67-1 | Molinat (1H-Azepine-1-carbothioic acid, hexahydro-S-ethyl ester) | 1/1/95 |
| 2300-66-5 | Dimethylamine dicamba | 1/1/95 |
| 2303-17-5 2312-35-8 2439-01-2 2439-10-3 2524-03-0 | Triallate Propargite Chinomethionat [6-Methyl-1,3-dithiolo(4,5-b)quinoxalin-2-one] Dodine [Dodecylguanidine monoacetate] Dimethyl chlorothiophosphate | 1/1/95 1/1/95 1/1/95 1/1/95 1/1/95 |
| 2655-15-4 2699-79-8 2702-72-9 | 2,3,5-Trimethylphenyl methylcarbamate Sulfuryl Fluoride [Vikane] 2,4-D sodium salt | 1/1/95 1/1/95 1/1/95 |
| 2971-38-2 | 2,4-D chlorocrotyl ester | 1/1/95 |
| 3383-96-8 3653-48-3 | Temephos Methoxone - sodium salt (4-Chloro-2-methylphenoxy acetate sodium salt) | 1/1/95 1/1/95 |
| 4080-31-3 4170-30-3 | 1-(3-Chloroallyl)-3,5,7-triaza-1-azoniaadamantane chloride Crotonaldehyde | 1/1/95 1/1/95 |
| 5234-68-4 5598-13-0 5902-51-2 6459-94-5 | Carboxin (5,6-Dihydro-2-methyl-N-phenyl-1,4-oxathiin-3-carboxamide) Chlorpyrifos methyl [O,O-dimethyl-O-(3,5,6-trichloro-2-pyridyl)phosphorothioate] Terbacil [5-Chloro-3-(1,1-dimethylethyl)-6-methyl-2,4-(1H,3H)-pyrimidinedione] C.I. Acid Red 114 | 1/1/95 1/1/95 1/1/95 1/1/95 |
| 7287-19-6 | Prometryn [N,N'-Bis(1-methylethyl)-6-methylthio-1,3,5-triazine-2,4-diamine] | 1/1/95 |
| 7632-00-0 7637-07-2 | Sodium nitrite Boron trifluoride | 1/1/95 1/1/95 |

| CAS No. | Chemical Name | Effective Date |
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| 7696-12-0 | Tetramethrin [2,2-Dimethyl-3-(2-methyl-1-propenyl)cyclopropane-carboxylic acid (1,3,4,5,6,7-hexahydro-1,3-dioxo-2H-isoindol-2-yl)methyl ester] | 1/1/95 |
| 7726-95-6 | Bromine | 1/1/95 |
| 7758-01-2 | Potassium bromate | 1/1/95 |
| 7782-41-4 | Fluorine | 1/1/95 |
| 7786-34-7 | Mevinphos | 1/1/95 |
| 7803-51-2 | Phosphine | 1/1/95 |
| 9006-42-2 | Metiram | 1/1/95 |
| 10028-15-6 | Ozone | 1/1/95 |
| 10061-02-6 | trans-1,3-Dichloropropene | 1/1/95 |
| 10222-01-2 | 2,2-Dibromo-3-nitrilopropionamide | 1/1/95 |
| 10294-34-5 | Boron trichloride | 1/1/95 |
| 10453-86-8 | Resmethrin [[5-(Phenylmethyl)-3-furanyl]methyl phenyl]cyclopropanecarboxylate]] 2,2-dimethyl-3-(2-methyl-1-pro- | 1/1/95 |
| 13194-48-4 | Ethoprop [Phosphorodithioic acid O-ethyl S,S-dipropyl ester] | 1/1/95 |
| 13356-08-6 | Fenbutatin oxide (hexakis(2-methyl-2-phenylpropyl)distannoxane) | 1/1/95 |
| 13463-40-6 | Iron pentacarbonyl | 1/1/95 |
| 13474-88-9 | 1,1-Dichloro-1,2,2,3,3-pentafluoropropane (HCFC-225cc) | 1/1/95 |
| 13684-56-5 | Desmedipham | 1/1/95 |
| 14484-64-1 | Ferbam [Tris(dimethylcarbamio-dithioato-S,S')iron] | 1/1/95 |
| 15972-60-8 | Alachlor | 1/1/95 |
| 17804-35-2 | Benomyl | 1/1/95 |
| 19044-88-3 | Oryzalin [4-(Dipropylamino)-3,5-dinitrobenzene-sulfonamide] | 1/1/95 |
| 19666-30-9 | Oxydiazon [3-[2,4-Dichloro-5-(1-methylethoxy)phenyl]-5-(1,1-dimethylethyl)-1,3,4-oxadiazol-2(3H)-one] | 1/1/95 |
| 20325-40-0 | 3,3'-Dimethoxybenzidine dihydrochloride (Dianisidine dihydrochloride) | 1/1/95 |
| 20354-26-1 | Methazole [2-(3,4-Dichlorophenyl)-4-methyl-1,2,4-oxadiazolidine-3,5-dione] | 1/1/95 |
| 20859-73-8 | Aluminum phosphide | 1/1/95 |
| 21087-64-5 | Metribuzin | 1/1/95 |
| 21725-46-2 | Cyanazine | 1/1/95 |
| 22781-23-3 | Bendiocarb [2,2-Dimethyl-1,3-benzodioxol-4-yl methylcarbamate] | 1/1/95 |
| 23564-05-8 | Thiophanate methyl | 1/1/95 |
| 23564-06-9 | Thiophanate ethyl [[1,2-Phenylenebis(iminocarbonothioyl)]biscarbamic acid diethyl ester] | 1/1/95 |
| 25311-71-1 | Isafenphos [2-[[Ethoxy]((1-methylethyl)amino)phosphinothioyl]oxy]benzoic acid 1-methylethyl ester] | 1/1/95 |
| 26002-80-2 | Phenothrin [2,2-Dimethyl-3-(2-methyl-1-propenyl)cyclopropanecarboxylic acid (3-phenoxyphenyl)methyl ester] | 1/1/95 |
| 26628-22-8 | Sodium azide | 1/1/95 |
| 26644-46-2 | Triflorine [N,N'-[1,4-Piperazinediylbis(2,2,2-trichloroethylidene)] bisformamide] | 1/1/95 |
| 27314-13-2 | Norflurazon [4-Chloro-5-(methylamino)-2-[3-(trifluoromethyl)phenyl]-3(2H)-pyridazinone] | 1/1/95 |
| 28057-48-9 | d-trans-Allethrin [d-trans-Chrysanthemic acid of d-allethrine] | 1/1/95 |
| 28249-77-6 | Thiobencarb [Carbamic acid, diethylthio-, s-(p-chlorobenzyl)] | 1/1/95 |
| 28407-37-6 | C.I. Direct Blue 218 | 1/1/95 |
| 29232-93-7 | Pirimiphos methyl [O-(2-(Diethylamino)-6-methyl-4-pyrimidinyl)-O,O-dimethyl phosphorothioate] | 1/1/95 |
| 30560-19-1 | Acephate (Acetylphosphoramidothioic acid O,S-dimethyl ester) | 1/1/95 |
| 31218-83-4 | Propetamphos [3-[[[(Ethylamino)methoxyphosphino-thioyl]oxy]-2-butenic acid, 1-methylethyl ester] | 1/1/95 |
| 33089-61-1 | Amitraz | 1/1/95 |
| 34014-18-1 | Terbutiuron [N-[5-(1,1-Dimethylethyl)-1,3,4-thiadiazol-2-yl]-N,N'- dimethylurea] | 1/1/95 |
| 35367-38-5 | Diffubenzuron | 1/1/95 |
| 35400-43-2 | Sulprofos [O-Ethyl O-[4-(methylthio)phenyl]phosphorodithioic acid S-propyl ester] | 1/1/95 |
| 35554-44-0 | Imazalil [1-[2-(2,4-Dichlorophenyl)-2-(2-propenyloxy)ethyl]-1H-imidazole] | 1/1/95 |
| 35691-65-7 | 1-Bromo-1-(bromomethyl)-1,3-propanedicarbonitrile | 1/1/95 |
| 38727-55-8 | Diethyl ethyl | 1/1/95 |
| 39300-45-3 | Dinocap | 1/1/95 |
| 39515-41-8 | Fenproprathrin [2,2,3,3-Tetramethylcyclopropane carboxylic acid cyano(3-phenoxyphenyl)methyl ester] | 1/1/95 |
| 40487-42-1 | Pendimethalin [N-(1-Ethylpropyl)-3,4-dimethyl-2,6-dinitrobenzen-amine] | 1/1/95 |
| 41198-08-7 | Profenofos [O-(4-Bromo-2-chlorophenyl)-O-ethyl-S-propyl phosphorothioate] | 1/1/95 |
| 41766-75-0 | 3,3'-Dimethylbenzidine dihydrofluoride (ortho-Tolidine dihydrofluoride) | 1/1/95 |
| 42874-03-3 | Oxyfluorfen | 1/1/95 |
| 43121-43-3 | Triadimefon [1-(4-Chlorophenoxy)-3,3-dimethyl-1-(1H-1,2,4-triazol-1-yl)-2-butanone] | 1/1/95 |
| 50471-44-8 | Vinclozolin [3-(3,5-Dichlorophenyl)-5-ethenyl-5-methyl-2,4- oxazolidinedione] | 1/1/95 |
| 51235-04-2 | Hexazinone | 1/1/95 |
| 51338-27-3 | Diclofop methyl [2-[4-(2,4-Dichlorophenoxy)phenoxy]propanoic acid, methyl ester] | 1/1/95 |

| CAS No. | Chemical Name | Effective Date |
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| 51630-58-1 | Fenvalerate | 1/1/95 |
| 52645-53-1 | Permethrin [3-(2,2-Dichloroethyl)-2,2-dimethylcyclopropanecarboxylic acid, (3-phenoxyphenyl)methyl ester] | 1/1/95 |
| 53404-19-6 | Bromacil, lithium salt [2,4-(1H,3H)-Pyrimidinedione, 5-bromo-6-methyl-3-(1-methylpropyl), lithium salt] | 1/1/95 |
| 53404-37-8 | 2,4-D 2-ethyl-4-methylpentyl ester | 1/1/95 |
| 53404-60-7 | Dazomet, sodium salt [Tetrahydro-3,5-dimethyl-2H-1,3,5-thiadiazine-2-thione, ion(1-), sodium] | 1/1/95 |
| 55290-64-7 | Dimethipin [2,3,-Dihydro-5,6-dimethyl-1,4-dithiin 1,1,4,4-tetraoxide] | 1/1/95 |
| 55406-53-6 | 3-Iodo-2-propynyl butylcarbamate | 1/1/95 |
| 57213-69-1 | Triclopyr, triethylammonium salt | 1/1/95 |
| 59669-26-0 | Thiodicarb | 1/1/95 |
| 60168-88-9 | Fenarimol [.alpha.-(2-Chlorophenyl)-.alpha.-4-chlorophenyl]-5-pyrimidine- methanol] | 1/1/95 |
| 60207-90-1 | Propiconazole [1-[2-(2,4-Dichlorophenyl)-4-propyl-1,3-dioxolan-2-yl]-methyl-1H-1,2,4,-triazole] | 1/1/95 |
| 62476-59-9 | Acifluorfen, sodium salt [5-(2-Chloro-4-(trifluoromethyl)phenoxy)-2-nitro-benzoic acid, sodium salt] | 1/1/95 |
| 62924-70-3 | Flumetralin [2-Chloro-N-(2,6-dinitro-4-(trifluoromethyl)-phenyl)-N-ethyl-6-fluorobenzenemethanamine] | 1/1/95 |
| 64902-72-3 | Chlorsulfuron [2-chloro-N-[[4-methoxy-6-methyl-1,3,5-triazin-2-yl]amino] carbonyl]benzenesulfonamide] | 1/1/95 |
| 64969-34-2 | 3,3'-Dichlorobenzidine.sulfate | 1/1/95 |
| 66441-23-4 | Fenoxaprop ethyl [2-(4-[(6-Chloro-2-benzoxazolylen)oxy]phenoxy) propanoic acid, ethyl ester] | 1/1/95 |
| 67485-29-4 | Hydramethylnon [Tetrahydro-5,5-dimethyl-2(1H)-pyrimidinone[3-[4-(trifluoromethyl)phenyl]-1-[2-[4-(trifluoromethyl)phenyl]ethenyl]-2- propenylidene]hydrazone] | 1/1/95 |
| 68085-85-8 | Cyhalothrin [3-(2-Chloro-3,3,3-trifluoro-1-propenyl)-2,2- dimethylcyclopropanecarboxylic acid cyano(3-phenoxyphenyl)methyl ester] | 1/1/95 |
| 68359-37-5 | Cyfluthrin [3-(2,2-Dichloro-ethyl)-2,2-dimethylcyclo-propanecarboxylic acid, cyano(4-fluoro-3-phenoxyphenyl)methyl ester] | 1/1/95 |
| 69409-94-5 | Fluvalinate [N-[2-Chloro-4-(trifluoromethyl)phenyl]-DL-valine(+)-cyano(3-phenoxyphenyl)methylester] | 1/1/95 |
| 69806-50-4 | Fluazifop-butyl [2-[4-[[5-(Trifluoromethyl)-2-pyridinyl]oxy]-phenoxy]propanoic acid, butyl ester] | 1/1/95 |
| 71751-41-2 | Abamectin [Avermectin B1] | 1/1/95 |
| 72178-02-0 | Fomesafen [5-(2-Chloro-4-(trifluoromethyl)phenoxy)-N-methylsulfonyl]-2- nitrobenzamide] | 1/1/95 |
| 72490-01-8 | Fenoxycarb [2-(4-Phenoxyphenoxy)ethyl]carbamic acid ethyl ester] | 1/1/95 |
| 74051-80-2 | Sethoxydim [2-[1-(Ethoxyimino)butyl]-5-[2-(ethylthio)propyl]-3-hydroxy-2-cyclohexen-1-one] | 1/1/95 |
| 76578-14-8 | Quizalofop-ethyl [2-[4-[(6-Chloro-2-quinoxalinyloxy]phenoxy] propanoic acid ethyl ester] | 1/1/95 |
| 77501-63-4 | Lactofen [5-(2-Chloro-4-(trifluoromethyl)phenoxy)-2-nitro-2-ethoxy-1-methyl-2-oxoethyl ester] | 1/1/95 |
| 82657-04-3 | Bifenthrin | 1/1/95 |
| 88671-89-0 | Myclobutanil [.alpha.-Butyl-.alpha.-(4-chlorophenyl)-1H-1,2,4-triazole- 1-propanenitrile] | 1/1/95 |
| 90982-32-4 | Chlorimuron ethyl [Ethyl-2-[[[(4-chloro-6-methoxyprimidin-2-yl)-carbonyl]-amino]sulfonyl]benzoate] | 1/1/95 |
| 101200-48-0 | Tribenuron methyl [2-(((4-Methoxy-6-methyl-1,3,5-triazin-2-yl)- methylamino)carbonyl)amino)sulfonyl]-, methyl ester] | 1/1/95 |
| 111512-56-2 | 1,1-dichloro-1,2,3,3,3-pentafluoropropane (HCFC-225eb) | 1/1/95 |
| 111984-09-9 | 3,3'-Dimethoxybenzidine hydrochloride (Dianisidine dihydrochloride) | 1/1/95 |
| 127564-92-5 | Dichloropentafluoropropane | 1/1/95 |
| 128903-21-9 | 2,2-Dichloro-1,1,1,3,3-pentafluoropropane (HCFC-225aa) | 1/1/95 |
| 136013-79-1 | 1,3-Dichloro-1,1,2,3,3-pentafluoropropane (HCFC-225ea) | 1/1/95 |

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| Category Name | Effective Date |
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| Diisocyanates (This category includes only those chemicals listed below) | 1/1/95 |
| 038661-72-2 1,3-Bis(methylisocyanate)cyclohexane | |
| 010347-54-3 1,4-Bis(methylisocyanate)cyclohexane | |
| 002556-36-7 1,4-Cyclohexane diisocyanate | |
| 134190-37-7 Diethyldiisocyanatobenzene | |
| 004128-73-8 4,4'-Diisocyanatodiphenyl ether | |
| 075790-87-3 2,4'-Diisocyanatodiphenyl sulfide | |
| 000091-93-0 3,3'-Dimethoxybenzidine-4,4'-diisocyanate | |
| 000091-97-4 3,3'-Dimethyl-4,4'-diphenylene diisocyanate | |
| 000139-25-3 3,3'-Dimethyldiphenylmethane-4,4'-diisocyanate | |
| 000822-06-0 Hexamethylene-1,6-diisocyanate | |
| 004098-71-0 Isophorone diisocyanate | |
| 075790-84-0 4-Methyldiphenylmethane-3,4-diisocyanate | |
| 005124-30-1 1,1-Methylene bis(4-isocyanatocyclohexane) | |
| 000101-68-8 Methylenebis(phenylisocyanate) (MDI) | |
| 003173-72-6 1,5-Naphthalene diisocyanate | |
| 000123-61-5 1,3-Phenylene diisocyanate | |
| 000104-49-4 1,4-Phenylene diisocyanate | |
| 009016-87-9 Polymeric diphenylmethane diisocyanate | |
| 016938-22-0 2,2,4-Trimethylhexamethylene diisocyanate | |
| 015646-96-5 2,4,4-Trimethylhexamethylene diisocyanate | |
| Nicotine and salts | 1/1/95 |
| Nitrate compounds (water dissociable; reportable only when in aqueous solution) | 1/1/95 |

| Category Name | Effective Date |
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| <p style="text-align: center;">* * * * *</p> <p>Polychlorinated alkanes: Includes those chemicals defined by the following formula: $C_xH_{2x-y}Cl_y$ where x = 10 to 13; y = 3 to 12; and where the average chlorine content ranges from 40–70% with the limiting molecular formulas $C_{10}H_{19}Cl_3$ and $C_{13}H_{16}Cl_{12}$.</p> | 1/1/95 |
| <p style="text-align: center;">* * * * *</p> <p>Polycyclic aromatic compounds (PACs): (This category includes only those chemicals listed below)</p> <ul style="list-style-type: none"> 00056-55-3 Benz(a)anthracene 00218-01-9 Benzo(a)phenanthrene 00050-32-8 Benzo(a)pyrene 00205-99-2 Benzo(b)fluoranthene 00205-82-3 Benzo(j)fluoranthene 00207-08-9 Benzo(k)fluoranthene 00189-55-9 Benzo(rst)pentaphene 00226-36-8 Dibenz(a,h)acridine 00224-42-0 Dibenz(a,j)acridine 00053-70-3 Dibenzo(a,h)anthracene 05385-75-1 Dibenzo(a,e)fluoranthene 00192-65-4 Dibenzo(a,e)pyrene 00189-64-0 Dibenzo(a,h)pyrene 00191-30-0 Dibenzo(a,l)pyrene 00194-59-2 7H-Dibenzo(c,g)carbazole 00057-97-6 7,12-Dimethylbenz(a)anthracene 00193-39-5 Indeno[1,2,3-cd]pyrene 03697-24-3 5-Methylchrysene 05522-43-0 1-Nitropyrene | 1/1/95 |
| <p style="text-align: center;">* * * * *</p> <p>Strychnine and salts</p> <p style="text-align: center;">* * * * *</p> | 1/1/95 |

[FR Doc. 94-29376 Filed 11-23-94; 4:03 pm]

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