ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 372

[OPPTS–400082; FRL–4645–6]

RIN 2070–AC47

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

AGENCY: Environmental Protection Agency (EPA).

ACTION: Proposed rule.

SUMMARY: EPA is proposing to add 313 chemicals and chemical categories to the list of toxic chemicals required to be reported on under section 313 of the Emergency Planning and Community Right-to-Know Act of 1986 and section 6607 of the Pollution Prevention Act of 1990. The proposed addition of these chemicals and chemical categories is based on their acute human health effects, carcinogenicity or other chronic human health effects, and/or their environmental effects. EPA believes that these chemicals and chemical categories meet the EPCRA section 313(d)(2) criteria for addition to the list of toxic chemicals.

DATES: Written comment on this proposed rule must be received on or before April 12, 1994. The public meeting will take place on March 2, 1994, at 1 p.m. and adjourn by 5 p.m.

ADDRESSES: Written comments should be submitted in triplicate to: OPPT Docket Clerk, TSCA Document Receipt Office (7407), Office of Pollution Prevention and Toxics, Environmental Protection Agency, Rm. E–G99, 401 M St., SW., Washington, DC 20460. Comments containing information claimed as confidential must be clearly marked as confidential business information (CBI). If CBI is claimed, three additional sanitized copies must also be submitted. Nonconfidential versions of comments on this proposed rule will be placed in the rulemaking record and will be available for public inspection. Comments should include the docket control number for this proposal, OPPTS–400082. Unit VI. of this preamble contains additional information on submitting comments containing information claimed as CBI.

The public meeting will be held at the: Environmental Protection Agency, Auditorium, Education Center, 401 M St., SW., Washington, DC.

FOR FURTHER INFORMATION CONTACT: Maria J. Doa, 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 or Toll free TDD: 800–553–7672, Attention: Docket Number OPPTS–400082.

SUPPLEMENTARY INFORMATION:

I. Introduction

A. Statutory Authority

This proposed rule is issued under sections 313(d) and (e)(1) of the Emergency Planning and Community Right-to-Know Act of 1986 (EPCRA), 42 U.S.C. 11023. 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. When enacted, section 313 established an initial list of toxic chemicals that was comprised of more than 300 chemicals and 20 chemical categories. Section 313(d) authorizes EPA to add chemicals to 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 has added to and deleted chemicals from the original statutory 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 submitting petitions. EPA must respond to petitions within 180 days either by initiating a rulemaking or by publishing an explanation of why the petition is denied. 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. Explanation for Expansion of the EPCRA Section 313 Chemical List

A. General Rationale

The Toxics Release Inventory (TRI), through the public access provisions of EPCRA, has proven to be one of the most powerful forces in empowering the Federal government, State governments, industry, environmental groups, and the general public, to fully participate in an informed dialogue about the environmental impacts of toxic chemicals in the United States.

A major section of EPCRA, which Congress passed in 1986, resulted in the creation of the Toxics Release Inventory. TRI is a publicly available data base that provides quantitative information on toxic chemical releases, transfers, recycling, and disposal. With the collection of this information for the first time in 1987, came the ability for the public, government, and the regulated community to understand the magnitude of chemical emissions in the United States; to compare chemical releases and transfers of chemical wastes among States, industries, facilities, and environmental media; and perhaps most importantly, to assess the need to reduce and where possible, eliminate these releases and transfers. TRI enables all interested in environmental progress to establish credible baselines, to set realistic goals, and to measure progress over time, in meeting those goals. The TRI system has become a neutral yardstick by which progress can be measured by all interested parties.

The original list of chemicals for which reporting was required consisted of 320 chemicals and chemical categories. The list was a combination of the Maryland Chemical Inventory Report List of Toxic or Hazardous Substances and the New Jersey Environmental Hazardous Substance List. The combination of these two lists provided a sound and logical starting point for the national TRI program. Recognizing however that the list would need to be a dynamic one, EPCRA specifically authorizes additions to and deletions from the list. To date, EPA has added 16 chemicals to the list and has deleted 12 chemicals from the list.

With 5 years experience behind the program, EPA, other federal agencies, Congress, and the public have recognized the need to expand the TRI list beyond the original chemicals and chemical categories and beyond the relatively limited reporting universe. (Currently reporting is only required from facilities that fall within the manufacturing Standard Industrial Classification (SIC) codes 20 through 39 that meet certain thresholds).

While the data on the chemicals that are covered have allowed the public and private sectors to be informed and involved in environmental decisionmaking as they never were before, it has become increasingly evident to those same constituents that they have access to information on a relatively small number of important chemicals. Congress has echoed this recognition in the Right-to-Know More

bills that were put forward in the 102nd Congress. EPA and State regulatory agencies have integrated TRI information as a critical component in their environmental decisionmaking and in many cases are constrained by the lack of similar information on chemicals of concern not covered by the TRI. While the TRI has been successful in focusing attention on the initial list of chemicals and in many cases fostering emissions reductions and prevention activities, that same focus has highlighted the need to expand beyond that initial list and to include additional chemicals that exhibit similar toxicity characteristics. This proposal is one of the first in a series of actions that EPA plans to use to expand the coverage of the TRI. This first phase will focus on adding chemicals, followed by a second phase that will identify additional facilities for inclusion. EPA is considering a third phase, which would look at modification of the data elements currently required by TRI.

In conjunction with these expansion activities EPA has been considering whether other adjustments are needed in the scope of the TRI program. EPA received petitions from the Small Business Administration and the American Feed Industry Association seeking an exemption for "small sources" (i.e, those facilities that file TRI forms with zero or small release estimates). EPA previously put those petitions out for public comment and, on review, believes there is substantial merit to the general concerns raised in the petitions.

The Agency's plan for proceeding on the small source issue would include the following steps. EPA is examining four options for establishing a small release exemption from the TRI reporting obligation: Cutoffs at zero, 500 pounds, 1,000 pounds, and 5,000 pounds. EPA will provide the public with a report on these four options by the end of January. This analysis will consider what data might not be available at both the national and community level, and the cost savings to the government and to industry of the four exemption levels. EPA plans to hold a public meeting in February for discussion of the report. Based on this feedback, EPA will then design a regulatory strategy that will align the small source issue with final action on today's proposal. The Agency's objective will be to minimize unnecessary data collection and reporting by facilities, including for the chemicals identified in today's proposal.

B. Development of the Chemical Addition Candidates

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: (1) 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 (f) 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).

In addition, EPA considered 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).

From this initial group of substances, EPA excluded chemicals that are already listed on section 313 or are already reportable under one of the EPCRA section 313 categories. For example, "cyanide, total" is listed under section 307(a) of the CWA. This listing is considered to be a subset of the EPCRA section 313 cyanide compounds category and the hydrogen cyanide listing. EPA decided not to propose listing these types of chemicals separately because they are already reportable under one of the existing section 313 categories. 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. The results of the toxicity screen for a subset of these chemicals were presented at a public meeting on May 29, 1992 (Ref. 4).

Other chemicals were also removed from consideration for this rulemaking because they are the subjects of two recently published EPCRA petition responses. On March 4, 1992, EPA received a petition from Governor Mario M. Cuomo of New York and the Natural Resources Defense Council (NRDC) to add 80 chemicals and 2 chemical categories to the list of toxic chemicals under section 313 of EPCRA. All of these chemicals and chemical categories appear on the RCRA list of hazardous wastes under 40 CFR 261.33(f) and as such are a subset of the chemicals screened by EPA. EPA responded to the petition in a proposed rulemaking on September 8, 1992 (57 FR 41020) and in a final rule adding 22 chemicals on November 30, 1993 (58 FR 63500).

On December 3, 1991, EPA received a petition from the NRDC, Friends of the Earth, and the Environmental Defense Fund to add hydrochlorofluorocarbons (HCFCs) to the list of toxic chemicals under section 313 of EPCRA. The HCFCs are listed under section 602(b) of the CAA as Class II ozone depleting substances and as such are a subset of the chemicals screened by EPA. EPA responded to the petition in a proposed rulemaking on June 24, 1992 (57 FR 28159) and in a final rule adding 11 HCFCs on November 30, 1993 (58 FR 63496). An additional 16 HCFCs not added to the TRI list by the November 30, 1993 final rule are proposed for addition in this rulemaking (See Unit IV.B.135. of this preamble).

1. *Toxicity screen.* A toxicity screen is a limited review of readily available toxicity data (e.g., information in data bases and other secondary sources) 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.B.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. 6). The numerical screening values reflected in

the *Draft Hazard Assessment Guidelines* were developed to capture, in the "sufficient for listing" screening category, the majority of chemicals already listed on various CERCLA and EPCRA lists, and thus known or suspected to be toxic and/or hazardous. These *Draft Hazard Assessment Guidelines* contain guidance for both the screening and hazard assessments of chemicals and are available for review in the docket associated with this rulemaking. This draft document was distributed at a public meeting on May 29, 1992. A final version of these guidelines has not yet been developed. Requests for further information about these draft guidelines should be addressed to the person identified under "FOR FURTHER INFORMATION CONTACT."

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*: "sufficient;" "may be sufficient;" or "insufficient." EPA received comment in response to the *Draft Hazard Assessment Guidelines* that objected to the Agency's use of the terms "sufficient," "may be sufficient," and "insufficient" as titles for the toxicity screening categories. The commenter claimed that these terms are appropriate only for the results of a hazard assessment. The commenter stated that these terms should not be used for screening categories because the toxicity screen only identifies chemicals for further consideration. EPA agrees that the screening categories only reflect a preliminary determination on each chemical, and therefore, to avoid further confusion, will refer to the screening categories as "high priority," "medium priority," and "low priority" to reflect the difference between a toxicity screen and a hazard assessment. These terms will be used throughout this document in reference to the toxicity screening categories. 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.

2. *Production volume screen.* EPCRA section 313(f) establishes reporting thresholds related to the amount of a chemical that is manufactured, processed, or otherwise used. [The EPCRA section 313 manufacture (includes import) and processing thresholds are 25,000 pounds per facility per year. The otherwise use threshold is 10,000 pounds per facility per year]. EPA anticipates that the addition of chemicals manufactured, imported, processed, or used in quantities less than the EPCRA section 313 volume thresholds would not result in the submission of TRI reports. Thus, EPA elected to initially focus its attention on chemicals likely to yield reports. Accordingly, EPA 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.

Production volume data on each of the chemicals were gathered primarily from two sources: (1) The TSCA Chemical Update System (1990); and (2) the FIFRA Section 7 Tracking System. On June 12, 1986 (51 FR 21438), EPA promulgated a rule pursuant to section 8(a) of TSCA which required manufacturers and importers to report every 4 years, subject to certain threshold production quantities and other exclusions, the quantities of chemicals they produced (40 CFR part 710). Among the exceptions to the inventory update rule (IUR) reporting were polymers, biological products, inorganic substances, and chemicals produced at less than 10,000 pounds, all with certain limitations. Data from the IUR is maintained in EPA's TSCA Chemical Update System (CUS).

Section 7 of FIFRA provides the Agency with annual production information on registered pesticides. EPA regulations implementing FIFRA section 7 (40 CFR part 167) require all manufacturers of pesticidal products (which includes formulated pesticides, active ingredients, and devices) to submit an annual report detailing the amount of each type of pesticidal product manufactured, sold and distributed during the past year, and estimated to be manufactured, imported, and processed during the current year (40 CFR 167.85).

For industrial inorganic compounds not subject to FIFRA or available on CUS, information from the public literature was used, supplemented with information from companies.

3. *Hazard evaluation.* EPA conducted a hazard evaluation 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 *Guidelines for Carcinogen Risk Assessment* (Ref. 2)). The guidelines that were used for each effect are Agency guidelines that are identified in the *Draft Hazard Assessment Guidelines* (Ref. 6). During this assessment the 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. On the other hand, where a review of the scientific data indicates that the chemical will cause various adverse effects at moderate dose levels, EPA believes, based on the total weight-of-the-evidence, that there is sufficient evidence for listing the chemical under EPCRA section 313.

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 requests comment on its approach in considering exposure as a part of its evaluation of these chemicals under sections 313(d)(2)(A) and (C).

Based on this analysis for each of the chemicals proposed for listing, EPA determined that one or more of the statutory criteria were met. A discussion of EPA's interpretation of the EPCRA section 313 criteria is given in Unit III.

of this preamble. A discussion of the evidence supporting EPA's proposal to add each of the chemicals to EPCRA section 313 is presented in Unit IV. of this preamble and in the record supporting this proposed rule.

4. *Other considerations.* EPA excluded certain chemicals and chemical categories from consideration for proposed listing under EPCRA section 313 in this rulemaking for a number of reasons. Some chemicals were identified only as environmental degradation products rather than chemicals that are manufactured, processed, or otherwise used by a facility. These chemicals will only be present in the environment as a result of the release into the environment of precursor chemicals. If the degradation product meets the toxicity criteria of EPCRA section 313, the precursor chemical may be considered for listing on EPCRA section 313. The degradation product would not be considered for listing on EPCRA section 313 because a facility subject to EPCRA section 313 is only required to file a TRI report for a chemical that it manufactures, processes, or otherwise uses, within the facility boundaries. Therefore, EPA does not believe that it is appropriate to consider listing such chemicals at this time.

Some of the lists reviewed by EPA included listings that represented waste streams from particular processes. These waste streams, such as coke oven emissions, are not discrete chemicals or chemical categories, but contain a wide range of chemicals, many of which are currently listed individually on EPCRA section 313. The focus of this rulemaking is on the addition of specific chemicals and chemical categories and, as such, EPA believes that these waste streams are inappropriate for listing under EPCRA section 313 at this time.

EPA also excluded chemicals whose only identified toxicity concern was a result of their status as a volatile organic compound (VOC). VOCs contribute to the formation of tropospheric ozone which causes a number of health-related and environmental problems. EPA continues to believe that VOCs meet the listing criteria of EPCRA section 313. However, EPA intends to address the issue of how VOCs should be listed on EPCRA section 313 separately. Therefore, chemicals whose only identified toxicity concern is due to their status as VOCs were excluded from consideration at this time.

EPA also identified chemicals that are routinely manufactured, processed, or otherwise used at levels far below the reporting thresholds of EPCRA section 313. These chemicals are not expected to ever be manufactured, processed, or otherwise used in quantities at or above these reporting thresholds. In this proposed rulemaking, EPA is attempting to add chemicals to EPCRA section 313 that are manufactured, processed, or otherwise used in quantities greater than the EPCRA section 313 volume thresholds and thus would result in the submission of TRI reports. Consequently, chemicals that are manufactured, processed, or otherwise used in quantities less than the EPCRA section 313 volume thresholds were excluded from further consideration at this time, because no reports would be filed under EPCRA section 313 for such chemicals.

Some of the chemicals that are manufactured, processed, or otherwise used below the EPCRA section 313 activity thresholds, particularly those 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. 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 may reconsider in the future the issue of listing such chemicals in a manner which would result in the submission of TRI reports. EPA requests comment on the following: Is it appropriate to list such chemicals on EPCRA section 313? 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 exemptions (40 CFR part 372.38), would be required to insure that release and transfer information would be collected?

III. EPCRA Section 313 Statutory Criteria

EPCRA section 313(d)(2) sets out criteria for adding chemicals to the list of chemicals subject to reporting under section 313(a). For a chemical (or category of chemicals) to be added to the EPCRA section 313(c) list of toxic chemicals, the Administrator must determine whether, in her judgement, there is sufficient evidence to establish any one of the following:

(A) The 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 as a result of continuous, or frequently recurring, releases.

(B) The chemical is known to cause or can reasonably be anticipated to cause in humans--

(i) cancer or teratogenic effects, or

(ii) serious or irreversible--

(I) reproductive dysfunctions,

(II) neurological disorders,

(III) heritable genetic mutations, or

(IV) other chronic health effects.

(C) The chemical is known to cause or can reasonably be anticipated to cause, because of--

(i) its toxicity,

(ii) its toxicity and persistence in the environment, or

(iii) its toxicity and tendency to bioaccumulate in the environment, a significant adverse effect on the environment of sufficient seriousness, in the judgement of the Administrator, to warrant reporting under this section.

To remove a chemical from the section 313(c) list, the Administrator must determine that there is not sufficient evidence to establish any of the criteria described above as required by EPCRA section 313(d)(3). Thus, the criteria for listing or delisting a chemical are identical. However, whereas EPA can add a chemical if only one of the criteria is met, it can only delete a chemical if none of the criteria are met.

To ascertain whether there is sufficient or insufficient evidence to determine that the statutory criteria are met for listing a chemical, EPA conducts a hazard assessment on the chemical and determines based on the weight-of-the-evidence, whether the chemical can reasonably be anticipated to cause any of the adverse effects specified in EPCRA section 313(d)(2). The hazard analysis is described above in Unit II.B.3. of this preamble. EPA's interpretation of the specific statutory criteria follows.

1. *Section 313(d)(2)(A) (acute human health effects).* To determine whether the section 313(d)(2)(A) "acute human health effects" criterion is met, EPA must examine the adverse effects associated with the chemical, the "concentration levels" which would cause acute human health effects, and the likelihood of such levels existing "beyond facility site boundaries as a result of continuous, or frequently recurring, releases." Such a determination may include, among other factors, consideration of production processes, workplace procedures, pollution controls, and the volume and pattern of production, use, and release, as well as other chemical-

specific factors. EPA believes that to make the section 313(d)(2)(A) determination it must demonstrate that a chemical can reasonably be anticipated to be released in quantities that result in concentration levels, or within a reasonable margin of exposure of the concentration levels, that would be expected to cause acute human health effects beyond the facility site boundary. The margin of exposure applied is dependent upon the type of hazard data (e.g., data in animals versus human) and the confidence in this hazard data base for acute effects (e.g., sufficiency of the hazard data). However, EPA is not required to make a facility-specific finding, nor is it necessary for EPA to demonstrate that these concentration levels or effects occur at or near any particular facility (Ref. 1). Furthermore, "EPA 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" (Ref. 1). Nor is EPA limited to considering concentration levels and potential acute human health effects at the "fenceline." Rather, the phrase "beyond facility site boundaries" reflects Congress' recognition that the "highest concentration to which persons outside the site boundary may be exposed" could occur at "any point outside the boundaries of the site on which the facility is located," including, for example, where an air emissions plume cools and settles to the ground (Ref. 1). Therefore, EPA believes that to make a finding under EPCRA section 313(d)(2)(A), the Agency may estimate concentrations at or beyond the facility site boundary through the use of estimated releases and modelling techniques. The term "continuous or frequently recurring releases" is included only to distinguish routine releases that are a normal consequence of the operation of a facility from the episodic and accidental releases that are subject to EPCRA section 304 (Ref. 1). As such, EPA believes that episodic and accidental releases are not pertinent in a determination that a chemical meets the section 313(d)(2)(A) criterion.

2. *Section 313(d)(2)(B) (chronic human health effects).* In contrast to the section 313(d)(2)(A) criterion, section 313(d)(2)(B) does not require consideration of either the nature and frequency of releases or concentration levels at facility site boundaries. Rather, section 313(d)(2)(B) is focused solely on whether the chemical is known or can reasonably be anticipated to cause cancer, teratogenicity, or other serious or irreversible chronic human health effects. Consequently, EPA believes that it is sufficient to consider only the toxicity of the subject chemical to make the section 313(d)(2)(B) determination.

3. *Section 313(d)(2)(C) (environmental effects).* The section 313(d)(2)(C) criterion requires EPA to consider a chemical's potential to cause significant adverse effects on the environment. The statute directs EPA to base its determination on a consideration of the toxicity of the chemical, either alone or in combination with the persistence of the chemical or the potential for the chemical to bioaccumulate. Congress intended that EPA consider a broad range of environmental effects when making a determination under section 313(d)(2)(C).

> In determining what constitutes a significant adverse effect on the environment...the Administrator should consider the extent to which the toxic chemical causes or can reasonably be anticipated to cause any of the following adverse reactions, even if restricted to the immediate vicinity adjacent to the site: (1) Gradual or sudden changes in the composition of animal life or plant life, including fungal or microbial organisms in an area. (2) Abnormal number of deaths of organisms (e.g. fish kills). (3) Reduction of the reproductive success or the vigor of a species. (4) Reduction in agricultural productivity, whether crops or livestock. (5) Alterations in the behavior or distribution of a species. (6) Long lasting or irreversible contamination of components of the physical environment, especially in the case of groundwater, and surface water and soil resources that have limited self-cleansing capability (Ref. 1).

EPA believes that the environmental effects criterion inherently contains a limited exposure component because of the statutory requirement for EPA 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. Unlike section 313(d)(2)(B), where EPA only has to determine whether certain kinds of effects are "known or reasonably anticipated" to occur, section 313(d)(2)(C) requires EPA to find the effect to be of sufficient seriousness to warrant reporting, which implies the possibility that under certain circumstances, a chemical that could theoretically cause a significant adverse effect on the environment is unlikely to cause one of a magnitude to warrant listing.

The extent to which exposure is factored into EPA's determination depends upon the inherent toxicity of a chemical, and a variety of other chemical-specific characteristics. EPA believes that when a chemical is inherently extremely toxic, that is, it is toxic at very low dose levels, an exposure assessment is not necessary because even minimal releases of such a chemical may reasonably be anticipated to result in significant adverse environmental effects. In such cases, EPA could rely on toxicity alone under section 313(d)(2)(C)(i) as a basis for listing.

However, for chemicals that exhibit adverse effects upon the environment solely based on toxicity at moderately low doses, EPA believes that consideration of potential exposure is warranted because minimal releases may not result in significant adverse effects upon the environment. These exposure considerations may include, among other factors, 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 modelling techniques.

EPCRA sections 313(d)(2)(C)(ii) and (iii) allow EPA to consider the impacts of other characteristics of a chemical. Where a chemical exhibits significant adverse effects in the environment based on toxicity and persistence or toxicity and bioaccumulation at very low to moderately low dose levels, EPA believes that exposure considerations are not required in addition to those considerations implicit in evaluation of the chemical's potential for persistence and bioaccumulation. This is because even minimal releases of the chemical 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. Therefore, evaluation of a chemical's persistence or bioaccumulation potential may be considered the functional equivalent of an exposure analysis.

In addition, for chemicals which induce well-established 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 affect changes across a whole ecosystem. EPA believes that these effects are of sufficient seriousness that additional

exposure considerations are not warranted because of the scope of their impact and the well-documented evidence supporting the adverse effects. EPA requests comment on its approach for considering exposure as a part of its evaluation for listing of these chemicals under section 313(d)(2)(C).

In Unit IV.B. of this preamble, EPA identifies each of the chemicals proposed for addition to EPCRA section 313 and the specific statutory criteria upon which the proposed addition is based.

IV. EPA's Technical Review

A. Introduction

Data on the chemicals and chemical categories were reviewed for evidence indicating adverse acute and chronic toxicity, carcinogenicity, mutagenicity, developmental and reproductive effects, neurotoxicity, and environmental effects. Information on the environmental fate was also reviewed.

For each chemical proposed for addition to EPCRA section 313 in this rulemaking, EPA conducted an extensive hazard assessment, and, where appropriate, an analysis of exposure, to determine whether the chemical met one or more of the EPCRA section 313(d)(2) listing criteria. This hazard assessment is discussed in detail in Unit II.B.3 of this preamble. Only after this careful review was a final determination made as to whether one of the EPCRA section 313(d)(2) listing criteria was met for each individual chemical or chemical category proposed for listing below. EPA need only show that one of the listing criteria is met in order to list a chemical or chemical category under EPCRA section 313. The information summarized below for each chemical or chemical category represents the key data elements that lead EPA to believe that there is sufficient evidence to establish that one of the section 313(d)(2) listing criteria is met. A more extensive review of the existing data base for each chemical or chemical category proposed for listing, which reflects the entire weight-of-the-evidence considered by EPA, is contained in following support documents: *Support Document for the Addition of Chemicals from Federal Insecticide, Fungicide, Rodenticide Act (FIFRA) Active Ingredients to EPCRA Section 313* (Ref 3); *Physical Properties and Environmental Fate of Some TRI Expansion Chemicals* (Ref. 5); *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. 7); and *Support Document for the Health and Ecological Toxicity Review of TRI Expansion Chemicals* (Ref. 8). These support documents contain a complete list of the references (which can be found in the public record for this proposed rulemaking) that were used in support of these proposed additions.

A list of the 313 chemicals and chemical categories and their Chemical Abstract Service (CAS) number, where appropriate, follows.

1. Abamectin (Avermectin B1) (CAS No. 071751-41-2)
2. Acephate (Acetylphosphoramidothioic acid O,S-dimethyl ester) (CAS No. 030560-19-1)
3. Acifluorfen sodium salt (5-(2-Chloro-4-(triflouromethyl)phenoxy)-2-nitro-benzoic acid, sodium salt) (CAS No. 062476-59-9)
4. Alachlor (CAS No. 015972-60-8)
5. Aldicarb (CAS No. 000116-06-3)
6. d-trans-Allethrin [d-trans-Chrysanthemic acid of d-allethrone] (CAS No. 028057-48-9)
7. Allylamine (CAS No. 000107-11-9)
8. Aluminum phosphide (CAS No. 020859-73-8)
9. Ametryn (N-Ethyl-N'-(1-methylethyl)-6-(methylthio)-1,3,5,triazine- 2,4 diamine) (CAS No. 000834-12-8)
10. Amitraz (CAS No. 033089-61-1)
11. Anilazine (4,6-Dichloro-N-(2-chlorophenyl)-1,3,5-triazin-2-amine) (CAS No. 000101-05-3)
12. Atrazine (6-Chloro-N-ethyl-N'-(1-methylethyl)-1,3,5,triazine-2,4-diamine) (CAS No. 001912-24-9)
13. Bendiocarb (2,2-Dimethyl-1,3-benzodioxol-4-ol methylcarbamate) (CAS No. 022781-23-3)
14. Benfluralin (N-Butyl-N-ethyl-2,6-dinitro-4-(trifluoromethyl) benzenamine) (CAS No. 001861-40-1)
15. Benomyl (CAS No. 017804-35-2)
16. o-Benzyl-p-chlorophenol (CAS No. 000120-32-1)
17. Bifenthrin (CAS No. 082657-04-3)
18. Bis(tributyltin) oxide (CAS No. 000056-35-9)
19. Boron trichloride (CAS No. 010294-34-5)
20. Boron trifluoride (CAS No. 007637-07-2)
21. Bromacil (5-Bromo-6-methyl-3-(1-methylpropyl)-2,4-(1H,3H)- pyrimidinedione) (CAS No. 000314-40-9)
22. Bromacil lithium salt (2,4-(1H,3H)-Pyrimidinedione, 5-bromo-6-methyl-3-(1-methylpropyl), lithium salt) (CAS No. 053404-19-6)
23. Bromine (CAS No. 007726-95-6)
24. 1-Bromo-1-(bromomethyl)-1,3-propanedicarbonitrile (CAS No. 035691-65-7)
25. 2-Bromo-2-nitropropane-1,3-diol (Bronopol) (CAS No. 000052-51-7)
26. Bromoxynil (3,5-Dibromo-4-hydroxybenzonitrile) (CAS No. 001689-84-5)
27. Bromoxynil octanoate (Octanoic acid, 2,6-dibromo-4-cyanophenyl ester) (CAS No. 001689-99-2)
28. Brucine (CAS No. 000357-57-3)
29. Butylate (Bis-2-methylpropyl)carbamothioic acid S-ethyl ester) (CAS No. 002008-41-5)
30. Butylated hydroxyanisole (CAS No. 025013-16-5)
31. C.I. Acid Red 114 (CAS No. 006459-94-5)
32. C.I. Direct Blue 218 (CAS No. 028407-37-6)
33. Calcium hypochlorite (CAS No. 007778-54-3)
34. Caprolactam (CAS No. 000105-60-2)
35. Carbofuran (CAS No. 001563-66-2)
36. Carbon monoxide (CAS No. 000630-08-0)
37. Carboxin (5,6-Dihydro-2-methyl-N-phenyl-1,4-oxathiin-3-carboxamide) (CAS No. 005234-68-4)
38. Chinomethionat (6-Methyl-1,3-dithiolo[4,5-b]quinoxalin-2-one) (CAS No. 002439-01-2)
39. Chlorendic acid (CAS No. 000115-28-6)
40. Chlorimuron ethyl (Ethyl-2-[[[(4-chloro-6-methoxyprimidin-2-yl)-carbonyl]-amino]sulfonyl]benzoate) (CAS No. 090982-32-4)
41. Chlorinated paraffins
42. 1-(3-Chloroallyl)-3,5,7-triaza-1-azoniaadamantane chloride (CAS No. 004080-31-3)
43. p-Chloroaniline (CAS No. 000106-47-8)
44. 5-Chloro-2-(2,4-dichlorophenoxy)phenol (CAS No. 003380-34-5)
45. 3-Chloro-2-methyl-1-propene (CAS No. 000563-47-3)
46. p-Chlorophenyl isocyanate (CAS No. 000104-12-1)
47. Chloropicrin (CAS No. 000076-06-2)
48. 3-Chloropropionitrile (CAS No. 000542-76-7)
49. p-Chloro-o-toluidine (CAS No. 000095-69-2)
50. Chlorotrifluoromethane (CFC-13) (CAS No. 000075-72-9)
51. Chlorpyrifos methyl (O,O-Dimethyl-O-(3,5,6-trichloro-2- pyridyl)phosphorothioate) (CAS No. 005598-13-0)
52. Chlorsulfuron (2-Chloro-N-[[(4-methoxy-6-methyl-1,3,5-triazin-2-yl)amino]carbonyl]benzenesulfonamide) (CAS No. 064902-72-3)
53. Clomazone (2-[(2-Chlorophenyl)methyl]-4,4-dimethyl-3-isoxazolidinone) (CAS No. 081777-89-1)
54. Crotonaldehyde (CAS No. 004170-30-3)
55. Cyanazine (CAS No. 021725-46-2)
56. Cycloate (CAS No. 001134-23-2)
57. Cyclohexanol (CAS No. 000108-93-0)
58. Cyfluthrin (3-(2,2-Dichloroethenyl)-2,2-dimethylcyclopropanecarboxylic acid, cyano(4-fluoro-3-phenoxyphenyl)methyl ester) (CAS No. 068359-37-5)
59. Cyhalothrin (3-(2-Chloro-3,3,3-trifluoro-1-propenyl)-2,2-dimethylcyclopropanecarboxylic acid cyano(3-phenoxyphenyl)methyl ester) (CAS No. 068085-85-8)
60. Cyromazine (N-Cyclopropyl-1,3,5-triazine-2,4,6-triamine) (CAS No. 066215-27-8)
61. Dazomet (Tetrahydro-3,5-dimethyl-2H-1,3,5-thiadiazine-2-thione) (CAS No. 000533-74-4)
62. Dazomet, sodium salt (2H-1,3,5-Thiadiazine-2-thione, tetrahydro-3,5-dimethyl-, ion(1-), sodium) (CAS No. 053404-60-7)
63. 2,4-DB (CAS No. 000094-82-6)
64. 2,4-D butoxyethyl ester (CAS No. 001929-73-3)

65. 2,4-D butyl ester (CAS No. 000094-80-4)
66. 2,4-D chlorocrotyl ester (CAS No. 002971-38-2)
67. Desmedipham (CAS No. 013684-56-5)
68. 2,4-D 2-ethylhexyl ester (CAS No. 001928-43-4)
69. 2,4-D 2-ethyl-4-methylpentyl ester (CAS No. 053404-37-8)
70. Diazinon (CAS No. 000333-41-5)
71. 2,2-Dibromo-3-nitrilopropionamide (CAS No. 010222-01-2)
72. Dicamba (3,6-Dichloro-2-methyoxybenzoic acid) (CAS No. 001918-00-9)
73. Dichloran (2,6-Dichloro-4-nitroaniline) (CAS No. 000099-30-9)
74. 3,3'-Dichlorobenzidine dihydrochloride (CAS No. 000612-83-9)
75. 3,3'-Dichlorobenzidine sulfate (CAS No. 064969-34-2)
76. trans-1,4-Dichloro-2-butene (CAS No. 000110-57-6)
77. Dichloromethylphenylsilane (CAS No. 000149-74-6)
78. Dichlorophene (2,2'-Methylenebis(4-chlorophenol) (CAS No. 000097-23-4)
79. trans-1,3-Dichloropropene (CAS No. 010061-02-6)
80. Diclofop methyl (2-[4-(2,4-Dichlorophenoxy) phenoxy]propanoic acid, methyl ester) (CAS No. 051338-27-3)
81. Dicyclopentadiene (CAS No. 000077-73-6)
82. Diethatyl ethyl (CAS No. 038727-55-8)
83. Diflubenzuron (CAS No. 035367-38-5)
84. Diglycidyl resorcinol ether (CAS No. 000101-90-6)
85. Dimethipin (2,3,-Dihydro-5,6-dimethyl-1,4-dithiin 1,1,4,4-tetraoxide) (CAS No. 055290-64-7)
86. Dimethoate (CAS No. 000060-51-5)
87. 3,3'-Dimethoxybenzidine dihydrochloride (o-Dianisidine dihydrochloride) (CAS No. 020325-40-0)
88. 3,3'-Dimethoxybenzidine hydrochloride (o-Dianisidine hydrochloride) (CAS No. 111984-09-9)
89. Dimethylamine (CAS No. 000124-40-3)
90. Dimethylamine dicamba (CAS No. 002300-66-5)
91. 3,3'-Dimethylbenzidine dihydrochloride (o-Tolidine dihydrochloride) (CAS No. 000612-82-8)
92. 3,3'-Dimethylbenzidine dihydrofluoride (o-Tolidine dihydrofluoride) (CAS No. 041766-75-0)
93. Dimethyl chlorothiophosphate (CAS. No. 002524-03-0)
94. Dimethyldichlorosilane (CAS No. 000075-78-5)
95. N,N-Dimethylformamide (CAS No. 000068-12-2)
96. 2,6-Dimethylphenol (CAS No. 000576-26-1)
97. Dinocap (CAS No. 039300-45-3)
98. Dinoseb (CAS No. 000088-85-7)
99. Diphenamid (CAS No. 000957-51-7)
100. Diphenylamine (CAS No. 000122-39-4)
101. Dipotassium endothall (7-Oxabicyclo(2.2.1)heptane-2,3-dicarboxylic acid, dipotassium salt) (CAS No. 002164-07-0)
102. Dipropyl isocinchomeronate (CAS No. 000136-45-8)
103. Disodium cyanodithioimidocarbonate (CAS No. 000138-93-2)
104. 2,4-D isopropyl ester (CAS No. 000094-11-1)
105. 2,4-Dithiobiuret (CAS No. 000541-53-7)
106. Dithiopyr (2-(Difluoromethyl)-4-(2-methylpropyl)-6-(trifluoromethyl)-3,5-pyridinedicarbothioic acid S,S-dimethyl ester) (CAS No. 097886-45-8)
107. Diuron (CAS No. 000330-54-1)
108. 2,4-D 2-octyl ester (CAS No. 001917-97-1)
109. Dodine (Dodecylguanidine monoacetate) (CAS No. 002439-10-3)
110. 2,4-DP (Dichlorprop) (CAS No. 000120-36-5)
111. 2,4-D propylene glycol butyl ether ester (CAS No. 001320-18-9)
112. 2,4-D sodium salt (CAS No. 002702-72-9)
113. Ethoprop (Phosphorodithioic acid O-ethyl S,S-dipropyl ester) (CAS No. 013194-48-4)
114. Ethyl dipropylthiocarbamate (EPTC) (CAS No. 000759-94-4)
115. Famphur (CAS No. 000052-85-7)
116. Fenarimol (.alpha.-(2-Chlorophenyl)-.alpha.-4-chlorophenyl)-5-pyrimidinemethanol) (CAS No. 060168-88-9)
117. Fenbutatin oxide (hexakis(2-methyl-2-phenylpropyl)distannoxane) (CAS No. 013356-08-6)
118. Fenoxaprop ethyl (2-(4-((6-Chloro-2 benzoxazolylen)oxy)phenoxy)propanoic acid, ethyl ester) (CAS No. 066441-23-4)
119. Fenoxycarb (2-(4-Phenoxyphenoxy)ethyl]carbamic acid ethyl ester) (CAS No. 072490-01-8)
120. Fenpropathrin (2,2,3,3-Tetramethylcyclopropane carboxylic acid cyano(3-phenoxyphenyl)methyl ester) (CAS No. 039515-41-8)
121. Fenthion (O,O-Dimethyl O-[3-methyl-4-(methylthio) phenyl] ester, phosphorothioic acid) (CAS No. 000055-38-9)
122. Fenvalerate (4-Chloro-alpha-(1-methylethyl)benzeneacetic acid cyano(3-phenoxyphenyl)methyl ester) (CAS No. 051630-58-1)
123. Ferbam (Tris(dimethylcarbamodithioato-S,S')iron) (CAS No. 014484-64-1)
124. Fluazifop butyl (2-[4-[[5-(Trifluoromethyl)-2-pyridinyl]oxy]-phenoxy]propanoic acid, butyl ester) (CAS No. 069806-50-4)
125. Flumetralin (2-Chloro-N-(2,6-dinitro-4-(trifluoromethyl)phenyl)-N-ethyl-6-fluorobenzenemethanamine) (CAS No. 062924-70-3)
126. Fluorine (CAS No. 007782-41-4)
127. Fluorouracil (5-Fluorouracil) (CAS No. 000051-21-8)
128. Fluvalinate (N-[2-Chloro-4-(trifluoromethyl)phenyl]-DL-valine(+)- cyano (3-phenoxyphenyl)methyl ester) (CAS No. 069409-94-5)
129. Folpet (CAS No. 000133-07-3)
130. Fomesafen (5-(2-Chloro-4-(trifluoromethyl)phenoxy)-N methylsulfonyl)-2-nitrobenzamide) (CAS No. 072178-02-0)
131. alpha-Hexachlorocyclohexane (CAS NO. 000319-84-6)
132. Hexamethylene-1,6-diisocyanate (CAS No. 000822-60-0)
133. n-Hexane (CAS No. 000110-54-3)
134. Hexazinone (CAS No. 051235-04-2)
135. Hydramethylnon (Tetrahydro-5,5-dimethyl-2(1H)pyrimidinone[3-[4-(trifluoromethyl)phenyl]-1-[2-[4(trifluoromethyl) phenyl]ethenyl]-2 propenylidene]hydrazone) (CAS No. 067485-29-4)
136—151. Hydrochlorofluorocarbons, specifically:
136. Dichloropentafluoropropane (CAS No. 127564-92-5)
137. 1,3-Dichloro-1,1,2,3,3-pentafluoropropane (HCFC-225ea) (CAS No. 136013-79-1)
138. 2,2-Dichloro-1,1,1,3,3-pentafluoropropane (HCFC-225aa) (CAS No. 128903-21-9)
139. 1,1-Dichloro-1,2,3,3,3-pentafluoropropane (HCFC-225eb) (CAS No. 111512-56-2)
140. 1,1-Dichloro-1,2,2,3,3-pentafluoropropane (HCFC-225cc) (CAS No. 13474-88-9)
141. 1,3-Dichloro-1,1,2,2,3-pentafluoropropane (HCFC-225cb) (CAS No. 000507-55-1)
142. 1,2-Dichloro-1,1,3,3,3-pentafluoropropane (HCFC-225da) (CAS No. 000431-86-7)
143. 3,3-Dichloro-1,1,1,2,2-pentafluoropropane (HCFC-225ca) (CAS No. 000422-56-0)
144. 2,3-Dichloro-1,1,1,2,3-pentafluoropropane (HCFC-225ba) (CAS No. 000422-48-0)
145. 1,2-Dichloro-1,1,2,3,3-pentafluoropropane (HCFC-225bb) (CAS No. 000422-44-6)
146. Dichlorofluoromethane (HCFC-21) (CAS No. 000075-43-4)
147. 1,1,1,2-Tetrachloro-2-fluoroethane (HCFC-121a) (CAS No. 000354-11-0)
148. 1,1,2,2-Tetrachloro-1-fluoroethane (HCFC-121) (CAS No. 000354-14- 3)
149. 1,2-Dichloro-1,1-difluoroethane (HCFC-132b) (CAS No. 001649-08-7)
150. 2-Chloro-1,1,1-trifluoroethane (HCFC-133a) (CAS No. 000075-88-7)
151. 3-Chloro-1,1,1-trifluoropropane (HCFC-253fb) (CAS No. 000460-35-5)
152. Imazalil (1-[2-(2,4-Dichlorophenyl)-2-(2-propenyloxy)ethyl]1H-imidazole) (CAS No. 035554-44-0)
153. 3-Iodo-2-propynyl butylcarbamate (CAS No. 055406-53-6)
154. Iprodione (3-(3,5-Dichlorophenyl)-N-(1-methylethyl)-2,4-dioxo-1-imidazolidinecarboxamide) (CAS No. 036734-19-7)
155. Iron pentacarbonyl (CAS No. 013463-40-6)
156. Isodrin (CAS No. 000465-73-6)
157. Isofenphos (2-[[Ethoxyl[(1-methylethyl) amino]phosphinothioyl]oxy]benzoic acid 1-methylethyl ester) (CAS No. 025311-71-1)
158. Isophorone (CAS No. 000078-59-1)
159. Isophorone diisocyanate (CAS No. 004098-71-9)
160. Lactofen (5-(2-Chloro-4-(trifluoromethyl)phenoxy)-2-nitro-2-ethoxy-1-methyl-2-oxoethyl ester) (CAS No. 077501-63-4)
161. Linuron (CAS No. 000330-55-2)
162. Lithium carbonate (CAS No. 000554-13-2)

163. Malathion (CAS No. 000121-75-5)
164. Man-made mineral fibers
165. Mecoprop (CAS No. 000093-65-2)
166. 2-Mercaptobenzothiazole (MBT) (CAS No. 000149-30-4)
167. Merphos (CAS No. 000150-50-5)
168. Metham sodium (Sodium methyldithiocarbamate) (CAS No. 000137-42-8)
169. Methazole (2-(3,4-Dichlorophenyl)-4-methyl-1,2,4-oxadiazolidine-3,5-dione) (CAS No. 020354-26-1)
170. Methiocarb (CAS No. 002032-65-7)
171. Methoxone ((4-Chloro-2-methylphenoxy) acetic acid) (MCPA) (CAS No. 000094-74-6)
172. Methoxone sodium salt ((4-Chloro-2-methylphenoxy) acetate sodium salt) (CAS No. 003653-48-3)
173. 1,1-Methylene bis(4-isocyanatocyclohexane) (CAS No. 005124-30-1)
174. Methylene bis(thiocyanate) (CAS No. 006317-18-6)
175. Methyl isothiocyanate (CAS No. 00556-61-6)
176. 2-Methyllactonitrile (CAS No. 000075-86-5)
177. N-Methylolacrylamide (CAS No. 000924-42-5)
178. Methyl parathion (CAS No. 000298-00-0)
179. N-Methyl-2-pyrrolidone (CAS No. 000872-50-4)
180. Methyltrichlorosilane (CAS No. 000075-79-6)
181. Metiram (CAS No. 009006-42-2)
182. Metribuzin (CAS No. 021087-64-5)
183. Mevinphos (CAS No. 007786-34-7)
184. Molinate (1H-Azepine-1-carbothioic acid, hexahydro-S-ethyl ester) (CAS No. 002212-67-1)
185. Monuron (CAS No. 000150-68-5)
186. Myclobutanil (.alpha.-Butyl-.alpha.-(4-chlorophenyl)-1H-1,2,4-triazole-1-propanenitrile) (CAS No. 088671-89-0)
187. Nabam (CAS No. 000142-59-6)
188. Naled (CAS No. 000300-76-5)
189. Nicotine and salts
190. Nitrapyrin (2-Chloro-6-(trichloromethyl) pyridine) (CAS No. 001929-82-4)
191. Nitrate ion (CAS No. 014797-55-8)
192. Nitric oxide (CAS No. 010102-43-9)
193. p-Nitroaniline (CAS No. 000100-01-6)
194. Nitrogen dioxide (CAS No. 010102-44-0)
195. Norflurazon (4-Chloro-5-(methylamino)-2-[3(trifluoromethyl)phenyl]-3(2H)-pyridazinone) (CAS No. 027314-13-2)
196. Oryzalin (4-(Dipropylamino)-3,5-dinitrobenzenesulfonamide) (CAS No. 019044-88-3)
197. Oxydemeton methyl (S-(2-(Ethylsulfinyl)ethyl) O,O-dimethyl ester phosphorothioic acid) (CAS No. 000301-12-2)
198. Oxydiazon (3-[2,4-Dichloro-5-(1-methylethoxy)phenyl]-5(1,1-dimethylethyl)-1,3,4-oxadiazol-2(3H)-one) (CAS No. 019666-30-9)
199. Oxyfluorfen (CAS No. 042874-03-3)
200. Ozone (CAS No. 010028-15-6)
201. Paraquat dichloride (CAS No. 001910-42-5)
202. Pebulate (Butylethylcarbamothioic acid S-propyl ester) (CAS No. 001114-71-2)
203. Pendimethalin (N-(1-Ethylpropyl)-3,4-dimethyl-2,6-dinitrobenzenamine) (CAS No. 040487-42-1)
204. Pentobarbital sodium (CAS No. 000057-33-0)
205. Perchloromethyl mercaptan (CAS No. 000594-42-3)
206. Permethrin (3-(2,2-Dichloroethenyl)-2,2-dimethylcyclopropanecarboxylic acid, (3-phenoxyphenyl)methyl ester) (CAS No. 052645-53-1)
207. Phenanthrene (CAS No. 000085-01-8)
208. Phenothrin (2,2-Dimethyl-3-(2-methyl-1-propenyl) cyclopropanecarboxylic acid (3-phenoxyphenyl)methyl ester) (CAS No. 026002-80-2)
209. 1,2-Phenylenediamine (CAS No. 000095-54-5)
210. 1,3-Phenylenediamine (CAS No. 000108-45-2)
211. 1,2-Phenylenediamine dihydrochloride (CAS No. 000615-28-1)
212. 1,4-Phenylenediamine dihydrochloride (CAS No. 000624-18-0)
213. Phenytoin (CAS No. 000057-41-0)
214. Phosphine (CAS No. 007803-51-2)
215. Phosphorus oxychloride (CAS No. 010025-87-3)
216. Phosphorus pentachloride (CAS No. 010026-13-8)
217. Phosphorus pentasulfide (CAS No. 001314-80-3)
218. Phosphorus pentoxide (CAS No. 001314-56-3)
219. Picloram (CAS No. 001918-02-1)
220. Piperonyl butoxide (CAS No. 000051-03-6)
221. Pirimiphos methyl (O-(2-(Diethylamino)-6-methyl-4-pyrimidinyl)-O,O-dimethyl phosphorothioate) (CAS No. 029232-93-7)
222— 249. Polycyclic aromatic compounds (PACs) including:
222. Benz(a)anthracene (CAS No. 000056-55-3)
223. Benzo(a)phenanthrene (CAS No. 000218-01-9)
224. Benzo(a)pyrene (CAS No. 000050-32-8)
225. Benzo(b)fluoranthene (CAS No. 000205-99-2)
226. Benzo(j)fluoranthene (CAS No. 000205-82-3)
227. Benzo(k)fluoranthene (CAS No. 000207-08-9)
228. Benzo(rst)pentaphene (CAS No. 000189-55-9)
229. Carbazole (CAS No. 000086-74-8)
230. Cyclopenta(cd)pyrene (CAS No. 027208-37-3)
231. Dibenz(a,h)acridine (CAS No. 000226-36-8)
232. Dibenz(a,j)acridine (CAS No. 000224-42-0)
233. Dibenz(a,c)anthracene (CAS No. 000215-58-7)
234. Dibenz(a,j)anthracene (CAS No. 000224-41-9)
235. Dibenzo(a,h)anthracene (CAS No. 000053-70-3)
236. Dibenzo(a,e)fluoranthene (CAS No. 005385-75-1)
237. Dibenzo(a,e)pyrene (CAS No. 000192-65-4)
238. Dibenzo(a,h)pyrene (CAS No. 000189-64-0)
239. Dibenzo(a,l)pyrene (CAS No. 000191-30-0)
240. 7H-Dibenzo(c,g)carbazole (CAS No. 000194-59-2)
241. 7,12-Dimethylbenz(a)anthracene (CAS No. 000057-976)
242. Indeno[1,2,3-cd]pyrene (CAS No. 000193-39-5)
243. 2-Methylchrysene (CAS No. 003351-32-4)
244. 3-Methylchrysene (CAS No. 003351-31-3)
245. 4-Methylchrysene (CAS No. 003351-30-2)
246. 5-Methylchrysene (CAS No. 003697-24-3)
247. 6-Methylchrysene (CAS No. 001705-85-7)
248. 2-Methylfluoranthene (CAS No. 033543-31-6)
249. 1-Nitropyrene (CAS No. 005522-43-0)
250. Potassium bromate (CAS No. 007758-01-2)
251. Potassium dimethyldithiocarbamate (CAS No. 000128-03-0)
252. Potassium N-methyldithiocarbamate (CAS No. 000137-41-7)
253. Primisulfuron (Methyl 2-[[[[[4,6-bis(difluoromethoxy)-2pyrimidinyl]-amino]carbonyl]amino]sulfonyl]benzoate) (CAS No. 086209-51-0)
254. Profenofos (O-(4-Bromo-2-chlorophenyl)-O-ethyl-S-propyl phosphorothioate) (CAS No. 041198-08-7)
255. Prometryn (N,N'-Bis(1-methylethyl)-6-methylthio-1,3,5-triazine-2,4-diamine) (CAS No. 007287-19-6)
256. Propachlor (2-Chloro-N-(1-methylethyl)-N-phenylacetamide) (CAS No. 001918-16-7)
257. Propanil (N-(3,4-Dichlorophenyl)propanamide) (CAS No. 000709-98-8)
258. Propargite (CAS No. 002312-35-8)
259. Propargyl alcohol (CAS No. 000107-19-7)
260. Propetamphos (3-[(Ethylamino)methoxyphosphinothioyl]oxy]-2-butenoic acid, 1-methylethyl ester) (CAS No. 031218-83-4)
261. Propiconazole (1-[2-(2,4-Dichlorophenyl)-4-propyl-1,3-dioxolan-2-yl]-methyl-1H-1,2,4,-triazole) (CAS No. 060207-90-1)
262. Quizalofop-ethyl (2-[4-[(6-Chloro-2-quinoxalinyl) oxy]phenoxy] propanoic acid ethyl ester) (CAS No. 076578-14-8)
263. Resmethrin ([5-(Phenylmethyl)-3-furanyl]methyl 2,2-dimethyl-3-(2-methyl-1-propenyl) cyclopropanecarboxylate]) (CAS No. 010453-86-8)
264. Sethoxydim (2-[1-(Ethoxyimino)butyl]-5-[2(ethylthio)propyl]-3-hydroxyl-2-cyclohexen-1-one) (CAS No.074051-80-2)
265. Simazine (CAS No. 000122-34-9)
266. Sodium azide (CAS No. 026628-22-8)
267. Sodium chlorite (CAS No. 007758-19-2)
268. Sodium dicamba (3,6-Dichloro-2-methoxybenzoic acid, sodium salt) (CAS No. 001982-69-0)
269. Sodium dimethyldithiocarbamate (CAS No. 000128-04-1)
270. Sodium fluoroacetate (CAS. No. 000062-74-8)

271. Sodium hypochlorite (CAS No. 007681-52-9)
272. Sodium nitrite (CAS No. 007632-00-0)
273. Sodium pentachlorophenate (CAS No. 000131-52-2)
274. Sodium o-phenylphenoxide (CAS No. 000132-27-4)
275. Sodium 2-pyridinethiol-1-oxide (CAS No. 015922-78-8)
276. Strychnine and salts
277. Sulfur dioxide (CAS No. 007446-09-5)
278. Sulfur trioxide (CAS No. 007446-11-9)
279. Sulfuryl fluoride (Vikane) (CAS No. 002699-79-8)
280. Sulprofos (O-Ethyl O-[4-(methylthio)phenyl]phosphorodithioic acid S- propyl ester) (CAS No. 035400-43-2)
281. Tebuthiuron (N-[5-(1,1-Dimethylethyl)-1,3,4-thiadiazol-2-yl)- N,N'-dimethylurea) (CAS No. 034014-18-1)
282. Tefluthrin (CAS No. 079538-32-2)
283. Temephos (CAS No. 003383-96-8)
284. Terbacil (5-Chloro-3-(1,1-dimethylethyl)-6-methyl- 2,4-(1H,3H)-pyrimidinedione) (CAS No. 005902-51-2)
285. Tetracycline hydrochloride (CAS No. 000064-75-5)
286. 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) (CAS No. 007696-12-0)
287. Tetrasodium ethylenediaminetetraacetate (CAS No. 000064-02-8)
288. Thiabendazole (2-(4-Thiazolyl)-1H-benzimidazole) (CAS No. 000148-79-8)
289. Thiabendazole, hypophosphite salt (2-(4-Thiazolyl) benzimidazole, hypophosphite salt) (CAS No. 028558-32-9)
290. Thiobencarb (Carbamic acid, diethylthio-, S-(p-chlorobenzyl)) (CAS No. 028249-77-6)
291. Thiodicarb (CAS No. 059669-26-0)
292. Thiophanate ethyl ([1,2-Phenylenebis (iminocarbonothioyl)] biscarbamic acid diethyl ester) (CAS No. 023564-06-9)
293. Thiophanate-methyl (CAS No. 023564-05-8)
294. Thiosemicarbazide (CAS No. 000079-19-6)
295. Triadimefon (1-(4-Chlorophenoxy)-3,3-dimethyl-1-(1H-1,2,4-triazol-1-yl)-2-butanone) (CAS No. 043121-43-3)
296. Triallate (CAS No. 002303-17-5)
297. Tribenuron methyl (2-(((((4-Methoxy-6-methyl-1,3,5-triazin-2-yl)-methylamino)carbonyl)amino)sulfonyl)-, methyl ester) (CAS No. 101200-48-0)
298. Tributyltin fluoride (CAS No. 001983-10-4)
299. Tributyltin methacrylate (CAS No. 002155-70-6)
300. S,S,S-Tributyltrithiophosphate (DEF) (CAS No. 000078-48-8)
301. Trichloroacetyl chloride (CAS No. 000076-02-8)
302. Trichloroethylsilane (CAS No. 000115-21-9)
303. Trichlorophenylsilane (CAS No. 000098-13-5)
304. 1,2,3-Trichloropropane (CAS No. 000096-18-4)
305. Triclopyr triethylammonium salt (CAS No. 057213-69-1)
306. Triethylamine (CAS No. 000121-44-8)
307. Triforine (N,N'-[1,4-Piperazinediylbis(2,2,2-trichloroethylidene)] bisformamide) (CAS No. 026644-46-2)
308. Trimethylchlorosilane (CAS No. 000075-77-4)
309. 2,3,5-Trimethylphenyl methylcarbamate (CAS No. 002655-15-4)
310. Triphenyltin chloride (CAS No. 000639-58-7)
311. Triphenyltin hydroxide (CAS No. 000076-87-9)
312. Vanadium pentoxide (CAS No. 001314-62-1)
313. Vinclozolin (3-(3,5-Dichlorophenyl)-5-ethenyl-5-methyl-2,4-oxazolidinedione) (CAS No. 050471-44-8)

A limited discussion of the health and environmental effects associated with each of the 313 chemicals and chemical categories is provided below in Unit IV.B. of this preamble. Each chemical is identified by chemical name, CAS No., and the list(s) from which the chemical originated. These lists are designated as follows:

CAA HAP: Clean Air Act section 112(b) "Hazardous Air Pollutants."
CAA OD: Clean Air Act section 602(b) Class II ozone depleters.
CAL: 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."
CERCLA: Comprehensive Environmental Response, Compensation, and Liability Act section 102.
CWA PPL: Clean Water Act section 307(a) "Priority Pollutant List."
EPCRA EHS: EPCRA section 302 "Extremely Hazardous Substances."
FIFRA AI: Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) "Active Ingredients."
FIFRA SR: FIFRA "Special Review, Canceled/Denied or Suspended, and Restricted Use Pesticides."
IARC: Monographs of the International Agency for Research on Cancer.
NTP: The 6th Annual Report on Carcinogens of the National Toxicology Program.
RCRA APP8: Resource Conservation and Recovery Act (RCRA) Chemicals listed at 40 CFR part 261 Appendix VIII.
RCRA P: RCRA Chemicals listed at 40 CFR part 261.33(e).
SDWA: Safe Drinking Water Act section 1412.
TSCA: Toxic Substances Control Act "Existing Chemicals."

EPA requests comment on the sufficiency of the evidence for each of the chemicals proposed for addition. In addition, EPA requests comment on any issues that may be specific to any of the individual chemicals or chemical categories. For example, should chemicals be listed on EPCRA section 313 that meet the EPCRA section 313 criteria but whose only use is as a drug product.

B. Chemicals Proposed for Addition to EPCRA Section 313

1. *Abamectin (avermectin B1)* (CAS No. 071751-41-2) (FIFRA AI) (Ref. 3). This compound induces developmental toxicity in several species with the mouse being the most sensitive species. Increased retinal folds in weanlings, decreased viability and lactation indices, and decreased body weight were noted in a two-generation rat reproduction study. The lowest-observed-effect level (LOEL) was 0.4 milligram per kilogram per day (mg/kg/day) and the no-observed-effect level (NOEL) was 0.12 mg/kg/day. Based on the NOEL, EPA derived a reference dose (RfD) of 0.0004 mg/kg/day. EPA believes that there is sufficient evidence for listing abamectin on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available developmental toxicity data.

Aquatic acute toxicity values for abamectin include a bluegill 96–hour LC_{50} of 9.6 parts per billion (ppb), a rainbow trout 96–hour LC_{50} of 3.6 ppb, and a daphnid 48–hour LC_{50} of 0.34 ppb. EPA believes that there is sufficient evidence for listing abamectin on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data.

2. *Acephate (acetylphosphoramidothioic acid O,S-dimethyl ester)* (CAS No. 030560-19-1) (FIFRA AI) (Ref. 3). In a 28–month feeding study in rats, inhibition of brain, plasma, and red blood cell cholinesterase activities was observed at 50 parts per million (ppm) (2.5 mg/kg/day). The NOEL for this study was 5 ppm (0.25 mg/kg/day). Similar findings were noted in a 2–year feeding study in dogs. The LOEL for this study was 100 ppm (2.5 mg/kg/day) and the NOEL was 30 ppm (0.75 mg/kg/day). EPA believes that there is sufficient evidence for listing acephate on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available neurotoxicity data for this chemical.

3. *Acifluorfen sodium salt (5-(2-chloro-4-(triflouromethyl)phenoxy)-2-nitro-benzoic acid, sodium salt)* (CAS No. 062476-59-9) (FIFRA AI) (Ref. 3). Acifluorfen is classified as a Group B2 compound, i.e., the chemical is a probable human carcinogen. Acifluorfen produced an increased incidence of combined malignant and benign liver tumors in two different strains of mice. The compound also displayed positive mutagenic activity in several non-mammalian test systems, and is structurally similar to four other diphenyl ether herbicide compounds which caused increased incidences of

liver tumors in two different strains of mice. EPA believes that there is sufficient evidence for listing acifluorfen sodium salt on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data.

4. *Alachlor* (CAS No. 015972-60-8) (FIFRA SR) (Ref. 8). Alachlor is an aniline-type herbicide. Dose-related hemolytic anemia with reductions in red blood cell counts, hematocrit and hemoglobin, as well as hemosiderosis in the liver, spleen and kidney occurred in male dogs orally exposed to alachlor for 1–year. The LOEL based on these effects was 3.0 mg/kg/day, and the NOEL was 1.0 mg/kg/day. Effects in female dogs in the same study were not demonstrated as clearly as in males but were considered suggestive of anemia. EPA derived an oral RfD of 0.01 mg/kg/day from this study.

In a three-generation reproduction study in rats, chronic nephritis and increased relative and absolute kidney weights were reported in F_2 adult males and F_3 pups. The LOEL was 10 mg/kg/day, and the NOEL was 3 mg/kg/day. Rabbits (Dutch Belted strain) that received alachlor via oral gavage during gestation days 6 to 27 had an increased rate of preimplantation loss (49 percent) and offspring with increased incidences of developmental malformations including major vessel variations, presacral vertebrae, and rudimentary and full 13th ribs. The increased incidence of rudimentary and full 13th ribs was dose-related, and a lowest-observed-adverse-effect level (LOAEL) of 10 mg/kg/day was determined based on this effect. The no-observed-adverse effect level (NOAEL) was not determined.

EPA has classified alachlor as a category Group B2 compound, i.e., the chemical is a probable human carcinogen. In a 2–year rat feeding study with Long-Evans rats, there were increased incidences of nasal turbinate tumors, malignant stomach tumors and thyroid follicular adenomas and carcinomas in both sexes at doses greater than or equal to 42 mg/kg/day. In an 18–month study in female CD-1 mice, bronchiolar tumors occurred at an increased incidence at 200 mg/kg/day.

EPA believes that there is sufficient evidence for listing alachlor on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the chronic toxicity and carcinogenicity data for this chemical.

5. *Aldicarb* (CAS No. 000116-06-3) (CERCLA; EPCRA EHS; FIFRA SR; RCRA APP8; RCRA P) (Ref. 8). Aquatic acute toxicity test data for aldicarb include a measured 96–hour LC_{50} of 50 ppb for bluegill and a measured 48–hour LC_{50} of 70 ppb for daphnid. In addition, the measured 48–hour EC_{50} for daphnid is 51 ppb. Measured terrestrial acute toxicity data for wildlife include an oral LD_{50} for female mallard ducks of 3.4 milligram per kilogram (mg/kg) and an oral LD_{50} for California quail of 2.58 mg/kg in males and 4.67 mg/kg in females. EPA believes that there is sufficient evidence for listing aldicarb on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the environmental toxicity data for this chemical.

6. *d-trans-Allethrin [d-trans-Chrysanthemic acid of dallethrone]* (CAS No. 028057-48-9) (FIFRA AI) (Ref. 3). Centrilobular hydropic degeneration of the liver (LOEL was 1,000 ppm or 25 mg/kg/day; the NOEL was 200 ppm or 5 mg/kg/day) was seen in dogs fed allethrin for 3 months. Increases in serum liver enzymes in female rats and increased liver weights in male and female rats (the LOEL was 250 mg/kg/day; the NOEL was 1,500 ppm or 75 mg/kg/day) were observed in rats fed allethrin for 3 months. Histopathology data were not presented in this study. Taken together, the results of these studies indicate hepatotoxic potential for d-trans-allethrin. EPA believes that there is sufficient evidence for listing d-trans-allethrin on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hepatic toxicity data.

7. *Allylamine* (CAS No. 000107-11-9) (EPCRA EHS) (Ref. 8). Repeated inhalation exposure to 5 ppm (0.011 mg/L) allylamine for 50 exposures of 7 hours caused liver and renal damage and myocarditis in rats. Congestion of the liver and kidney was observed in rats, rabbits, and dogs exposed to 5 or 20 ppm (0.011 or 0.044 milligram per liter (mg/L)) allylamine for 8 hours/day, 5 days/week, for 1–year. EPA believes that there is sufficient evidence for listing allylamine on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the hepatotoxicity and nephrotoxicity data for this chemical.

8. *Aluminum phosphide* (CAS No. 020859-73-8) (CERCLA; EPCRA EHS; RCRA APP8; RCRA P) (Ref. 8). The median lethal dose of aluminum phosphide in humans is 20 mg/kg. The acute inhalation toxicity of aluminum phosphide is attributed to phosphine gas resulting from decomposition of aluminum phosphide on contact with moisture in the air. Symptoms of phosphine poisoning include restlessness, headache, dizziness, fatigue, chest tightness, nausea, vomiting, lethargy, stupor, coma, convulsions, lowered blood pressure, pulmonary edema and respiratory failure; disorders of the kidney, liver, heart and brain can also occur. In female CFT-Wistar rats exposed to phosphine gas generated from aluminum phosphide pellets in distilled water, 100 percent mortality was observed after a 6–hour exposure to 40 ppm (0.1 mg/L), and exposure to 20 to 40 ppm (0.05 to 0.1 mg/L) for 6 hours resulted in 33 percent mortality. Symptoms of toxicity reported in these animals included dyspnea, loss of muscular coordination, polyuria, and paralysis.

EPA's exposure analysis indicates that aluminum phosphide concentrations are likely to exist beyond facility site boundaries, as a result of continuous, or frequently recurring releases, at levels that can reasonably be anticipated to cause significant adverse acute human health effects. EPA believes that there is sufficient evidence for listing aluminum phosphide on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(A) based on the available acute toxicity and exposure data for this chemical.

9. *Ametryn (N-Ethyl-N'-(1-methylethyl)-6-(methylthio)1,3,5,-triazine- 2,4-diamine)* (CAS No. 000834-12-8) (FIFRA AI) (Ref. 3). Fatty degeneration of the liver was observed in rats administered 100 mg/kg/day ametryn by gavage, 6 days per week for 13 weeks. The NOEL was 10 mg/kg/day (8.6 mg/kg/day adjusted for duration). In another study, hepatic effects (severe vascular congestion, centrilobular liver necrosis and fatty degeneration of individual liver cells) were observed in rats that died following gavage administration of 500 mg/kg/day ametryn for 6 days per week for 28 days. The NOEL was 250 mg/kg/day. EPA believes that there is sufficient evidence for listing ametryn on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hepatotoxicity data for this chemical.

The 72-hour EC_{50} for green algae is 14 ppb. Ametryn is a herbicide and may be expected to affect nontarget plants such as algae. EPA believes that there is sufficient evidence for listing ametryn on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data for this chemical.

10. *Amitraz* (CAS No. 033089-61-1) (FIFRA SR) (Ref. 8). Amitraz is an aniline-type insecticide. In a 2–year beagle dog feeding study, effects noted at the LOAEL dose (1.0 mg/kg/day) at various times during the study included significantly increased mean blood glucose concentration, slight hypothermia, and slight central nervous system depression (the latter effect occurred immediately after dosing on

days 1 and 2). The NOAEL in this study was 0.25 mg/kg/day and the oral RfD derived from the NOAEL was 0.0025 mg/kg/day. These findings were supported by similar results obtained in a 90–day feeding study in dogs. In studies with rats or mice exposed to amitraz from 90 days to 2 years, LOAELs less than or equal to 12 mg/kg/day were derived based on effects that included decreased body weight gain and changes in organ (brain or heart) weight (the NOELs were less than or equal to 3 mg/kg/day).

A three-generation reproduction study in rats demonstrated decreased litter size and increased mortality during suckling. The fetotoxic LOAEL in this study was 5 mg/kg/day and the NOAEL was 1.6 mg/kg/day. In a teratology study in rabbits, a fetotoxicity LOAEL of 5 mg/kg/day and NOAEL of 1 mg/kg/day were based on the incidences of cleft palate and meningocoele associated with small ears and displaced toes.

EPA believes that there is sufficient evidence for listing amitraz on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the chronic toxicity and developmental toxicity data for this chemical.

11. *Anilazine (4,6-dichloro-N-(2-chlorophenyl)-1,3,5-triazin-2-amine)* (CAS No. 000101-05-3) (FIFRA AI) (Ref. 3). When anilazine was administered to rats, maternal reproductive parameters were not affected. The systemic maternal NOEL was 150 mg/kg and the LOEL was 500 mg/kg, based on decreased body weight gain. The developmental NOEL was 1,500 mg/kg, which was the highest dose tested. In rabbits, the maternal toxicity NOEL was 15 mg/kg and the LOEL was 40 mg/kg, based on increased mortalities and decreased body weight gain (also decreased percentage of pregnant does at 75 mg/kg). The developmental NOEL was 40 mg/kg and the LOEL was 75 mg/kg, based on increased fetal mortality, decreased fetal weight, and increased postimplantation loss and inhibited ossification (phalanges). EPA believes that there is sufficient evidence for listing anilazine on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available developmental toxicity data.

Aquatic acute toxicity values for anilazine include a scud (Gammarus) 96–hour LC_{50} of 0.27 ppb and an oyster 96–hour EC_{50} (growth) of 46 ppb. EPA believes that there is sufficient evidence for listing anilazine on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data.

12. *Atrazine (6-chloro-N-ethyl-N'-(1-methylethyl)-1,3,5,-triazine-2,4-diamine)* (CAS No. 001912-24-9) (FIFRA AI) (Ref. 3). Based on sufficient evidence of carcinogenicity in animals, the International Agency for Research on Cancer (IARC) has classified atrazine as a Group 2B compound; i.e., the chemical is possibly carcinogenic to humans. Administration of atrazine to Sprague Dawley rats was associated with an increased incidence of mammary gland fibroadenomas and adenocarcinomas in female rats. A hormonal mechanism may be involved in the induction of mammary tumors by atrazine. Therefore there is sufficient evidence for listing atrazine on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for this chemical.

13. *Bendiocarb (2,2-dimethyl-1,3-benzodioxol-4-ol methylcarbamate)* (CAS No. 022781-23-3) (FIFRA AI) (Ref. 3). Depressed blood cholinesterase levels were reported in numerous species. In a developmental toxicity study in rats, cholinergic signs were observed in maternal animals at 4 mg/kg/day (LOEL). The maternal NOEL was 1 mg/kg/day; no adverse effects were observed in fetuses. A LOEL of 2.5 mg/kg/day for cholinesterase inhibition was reported in dogs in a 4–month dietary study. The NOEL was 0.5 mg/kg/day. Decreases in cholinesterase activity were observed in female rats fed 20, 30, or 40 mg/kg/day for 28 days. No NOEL was established in this study. However, no details regarding clinical signs or histopathological changes in neural tissue were reported. EPA believes that there is sufficient evidence for listing bendiocarb on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available neurological toxicity data for this chemical.

Aquatic acute toxicity values for bendiocarb include a mysid 96–hour EC_{50} of 6.7 ppb and a daphnid 48–hour EC_{50} of 29.2 ppb. Avian acute toxicity values include a mallard duck LD_{50} of 3.1 mg/kg. EPA believes that there is sufficient evidence for listing bendiocarb on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data.

14. *Benfluralin (N-butyl-N-ethyl-2,6-dinitro-4(trifluoromethyl) benzenamine)* (CAS No. 001861-40-1) (FIFRA AI) (Ref. 3). Increased relative liver weights, decreased red blood cell counts and decreased hematocrit and hemoglobin levels were observed in dogs orally administered benfluralin at a dose of 125 mg/kg/day for 2 years. The NOAEL was 25 mg/kg/day. Based on the NOAEL, EPA has established an oral RfD of 0.003 mg/kg/day. EPA believes that there is sufficient evidence for listing benfluralin on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hematological toxicity data for this chemical.

15. *Benomyl* (CAS No. 017804-35-2) (CAL; FIFRA SR) (Ref. 8). In a three-generation study in rats, a dietary level of 25 mg/kg/day of benomyl resulted in decreased weanling weights. The no-effect level was 5 mg/kg/day. Microphthalmia (the LOEL was 62.5 mg/kg/day; the NOEL was 30 mg/kg/day) was reported in a rat developmental toxicity study. Decreased fetal weight (the LOEL was 62.5 mg/kg/day; the NOEL was 30 mg/kg/day) was observed in another rat developmental toxicity study. The developmental effects were observed at doses that were not toxic to the maternal animal. Anomalies consisting of supra occipital scars, subnormal vertebral centrum, supernumary ribs, and cleft palate were reported in an oral developmental toxicity study in mice (the LOEL was 100 mg/kg/day; the NOEL was 50 mg/kg/day). An increase in the incidence of anomalies including encephalocele, hydrocephalus, microphthalmia, and anophthalmia was noted following administration of benomyl to rats by intubation during the first 20 days of pregnancy at doses of 125, 250, and 500 mg/kg. The developmental effects were always associated with death and were considered to be the cause of death. EPA believes that there is sufficient evidence for listing benomyl on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the developmental toxicity data for this chemical.

16. *o-Benzyl-p-chlorophenol* (CAS No. 000120-32-1) (FIFRA AI) (Ref. 3). In a 16–day oral rat study, dose-related increases in liver and kidney weights (absolute and relative) and nephrosis were observed at a dose level of greater than or equal to 62.5 mg/kg/day. A NOEL was not established. When the compound was administered by gavage for 13 weeks, rats developed multifocal dilation of renal tubules and increased liver weights (16 percent) at 240 mg/kg/day. The NOEL was 120 mg/kg/day. In a 90–day oral study, mice receiving 30 mg/kg/day developed kidney lesions. Increased liver weights were also noted. No NOEL was established in this study. EPA believes that there is sufficient evidence for listing o-benzyl-p-chlorophenol on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hepatic and renal toxicity data for this chemical.

17. *Bifenthrin* (CAS No. 082657-04-3) (FIFRA AI) (Ref. 3). Tremors or head and forelimb twitching were noted in dogs, rats and rabbits exposed to various

doses. NOEL values based on the appearance of tremors (often transient) ranged from 1 to 2.67 mg/kg/day. The oral RfD for bifenthrin was based on a 1year beagle dog feeding study, in which the LOEL, based on tremors observed during weeks 15 to 29, was 3.0 mg/kg/day and the NOEL was 1.5 mg/kg/day. The RfD based on this NOEL was 0.015 mg/kg/day.

In a rat teratology study, an increased incidence of hydroureter (without hydronephrosis) was noted in fetuses at 2 mg/kg/day (LOEL). The NOEL was 1 mg/kg/day.

EPA believes that there is sufficient evidence for listing bifenthrin on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available neurological and developmental toxicity data.

Aquatic acute toxicity values for bifenthrin include a bluegill 96–hour LC_{50} of 0.35 ppb, a rainbow trout 96–hour LC_{50} of 0.15 ppb, a sheepshead minnow LC_{50} of 17.5 ppb, and a daphnid 48–hour EC_{50} of 1.6 ppb. EPA believes that there is sufficient evidence for listing bifenthrin on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data.

18. *Bis(tributyltin) oxide* (CAS No. 000056-35-9) (FIFRA AI) (Ref. 3). Adverse effects on the immune system were reported in rats exposed to various doses of bis(tributyltin) oxide for a duration as short as 4 weeks. SPF-derived Wistar rats were fed the compound for 17 months. In this study, a LOEL of 0.25 mg/kg/day and a NOEL of 0.025 mg/kg/day were based on immunotoxicity manifested as decreased resistance to *Trichinella spiralis*, reduced natural killer (NK) cell activity in the spleen and reduced macrophage function. The RfD derived from this NOEL was 0.00003 mg/kg/day. Similar immunological effects were reported in 4- and 6-week rat feeding studies with 20 and 80 ppm (1 and 4 mg/kg/day; the LOEL was 1 mg/kg/day).

In rats that received dietary levels (of a range of doses that included 50 mg/kg/day) for 106 weeks, kidney function was decreased and serum levels of alanine aminotransferase, aspartate aminotransferase and alkaline phosphatase were increased. At the end of the 2–year study, nephrosis and vacuolization and pigmentation of the proximal tubular epithelium were reported in animals administered 50 mg/kg/day. On the basis of marginal effects at 5 mg/kg/day (LOEL), a NOEL of 0.5 mg/kg/day was established.

EPA believes that there is sufficient evidence for listing bis(tributyltin) oxide on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available immunological and renal toxicity data.

Aquatic acute toxicity values for bis(tributyltin) oxide include a bluegill 96–hour LC_{50} of 7.6 ppb, a rainbow trout 96–hour LC_{50} 6.9 ppb, a measured fathead minnow 96–hour LC_{50} of 2.7 ppb, and a daphnid 48–hour LC_{50} of 1.67 ppb. EPA believes that there is sufficient evidence for listing bis(tributyltin) oxide on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data.

19. *Boron trichloride* (CAS No. 010294-34-5) (EPCRA EHS) (Ref. 8). Boron trichloride is corrosive to the skin and mucosal tissue due to its rapid hydrolysis to hydrochloric acid and boric acid, the former acid being the corrosive species. Single, relatively large doses of boron administered through any route affects the central nervous system causing depressed circulation, diarrhea, vomiting, shock, and coma. The kidneys are the most severely affected organ. Symptoms of acute irritation of the upper airways were observed in humans at exposure levels of greater than or equal to 0.004 mg/L. Inhalation of 0.48 mg/L of boron trichloride proved fatal to certain laboratory animals. Inhalation of 0.096 mg/L of boron trichloride for 7 hours produced adverse effects on the respiratory tract, and weight loss.

EPA's exposure analysis indicates that boron trichloride concentrations are likely to exist beyond facility site boundaries, as a result of continuous, or frequently recurring releases, at levels that can reasonably be anticipated to cause significant adverse acute human health effects. EPA believes that there is sufficient evidence for listing boron trichloride on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(A) based on the available acute toxicity and exposure data for this chemical.

20. *Boron trifluoride* (CAS No. 007637-07-2) (EPCRA EHS) (Ref. 8). Boron trifluoride is a colorless gas that is corrosive to tissues due to its rapid hydrolysis to hydrofluoric acid and boric acid. The principal acute effect in animals is irritation of the mucous membranes of the respiratory tract and eyes; post mortem examination also revealed pneumonia and degenerative changes in renal tubules. The kidneys are most severely affected because boric acid concentrates in this organ. Exposure of six animal species to 0.28 mg/L of boron trifluoride for 4 to 7 hours a day, 5 days a week killed all animals within 30 days. Rats, rabbits, and guinea pigs were exposed to boron trifluoride via inhalation. Guinea pigs died of respiratory failure after being exposed to 0.036 mg/L for 19 days; rats experienced fluorosis of the teeth at this concentration. All three species were minimally affected at 0.004 mg/L. In a 2-week rat inhalation study, all animals died after 6 daily exposures to 0.18 mg/L. Rats exposed to 0.024 mg/L showed signs of respiratory irritation, increased lung weights, and depressed liver weights. Rats exposed to 0.17 mg/L of boron trifluoride 6 hours/day, 5 days a week for 13 weeks developed necrosis of the proximal tubular epithelium of the kidneys. Guinea pigs exposed to 0.035 mg/L, 7 hours/day, 5 days a week for 3 months developed severe pneumonitis and pulmonary changes indicating chemical irritation.

EPA believes that there is sufficient evidence for listing boron trifluoride on EPCRA section 313 pursuant to section 313(d)(2)(B) based on the available chronic toxicity data for this chemical.

21. *Bromacil (5-bromo-6-methyl-3-(1-methylpropyl)-2,4-(1H,3H)-pyrimidinedione)* (CAS No. 000314-40-9) (FIFRA AI) (Ref. 3). Increased thyroid activity was seen in male and female rats fed 5,000 ppm (250 mg/kg/day) bromacil for 90 days. In a 2–year dietary study, thyroid hyperplasia was seen in female rats fed 1,250 ppm (62.5 mg/kg/day). Thyroid follicular adenoma was observed in one female. EPA believes that there is sufficient evidence for listing bromacil on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available thyroid toxicity data for this chemical.

22. *Bromacil lithium salt (2,4-(1H,3H)-pyrimidinedione, 5-bromo-6-methyl-3-(1-methylpropyl), lithium salt)* (CAS No. 05340419-6) (FIFRA AI) (Ref. 3). Bromacil lithium salt will dissociate into bromacil, which is soluble in aqueous systems and lithium ion. Defects of the palate, eye, and external ear were reported in the offspring of rats administered 50 mg lithium chloride intraperitoneally on gestation days 1, 4, 7, and 9 followed by 20 mg/day until day 17. Cleft palates were also observed in mouse fetuses when mothers were gavaged with 300 to 465 mg/kg/day lithium carbonate on gestation day 6 to 15. An increase in Ebstein's anomaly was reported among offspring of women taking lithium; cardiovascular defects were found in 212 offspring exposed in utero to lithium therapy.

Increased thyroid activity was seen in male and female rats fed 5,000 ppm (250 mg/kg/day) bromacil for 90 days. In a 2–year dietary study, thyroid hyperplasia was seen in female rats fed 1,250 ppm (62.5 mg/kg/day). Thyroid follicular adenoma was observed in one female.

EPA believes that there is sufficient evidence for listing bromacil lithium salt on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available developmental and thyroid toxicity data.

23. *Bromine* (CAS No. 007726-95-6) (EPCRA EHS) (Ref. 8). Rats fed bromine at a dose of 0.01 mg/kg/day for 6 months experienced changes in their reflexes and blood indexes. Rats, mice, and rabbits inhaling 0.001 mg/kg/day for 4 months developed functional abnormalities of the respiratory, nervous, and endocrine systems. Data on the acute and chronic effects of bromine in humans are limited. Bromine is very corrosive to the eyes, skin, and mucous membranes in either the liquid or vapor form. A concentration of 10 ppm of bromine in air is intolerable in humans, and can cause severe irritation of the upper respiratory tract. Other clinical symptoms include neurologic, dermatologic, and gastrointestinal effects. The maximum concentration allowable in humans for a 0.5 to 1-hour exposure to bromine is 4 ppm. Bromine can cause lacrimation at concentrations less than 1 ppm. Chronic exposure to bromine (estimated concentration at 0.6 ppm) can result in eye irritation, upper respiratory irritation, coughing, and headache. Neurological symptoms have also been reported following chronic exposure to bromine.

EPA believes that there is sufficient evidence for listing bromine on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available chronic toxicity data for this chemical.

24. *1-Bromo-1-(bromomethyl)-1,3-propanedicarbonitrile* (CAS No. 035691-65-7) (FIFRA AI) (Ref. 3). In a 3–month dietary study where rats were administered 83.5, 500, and 3,000 ppm (4, 25, and 150 mg/kg/day) 1-bromo-1-(bromomethyl)-1,3-propanedicarbonitrile, a NOEL of 83.5 ppm (4 mg/kg/day) and a LOEL of 500 ppm (25 mg/kg/day) were established (based on neonatal splenic hematopoiesis, decreased parental body weight and food consumption, increased male urinary epithelial cells, amorphous casts, and crystals). At 3,000 ppm (150 mg/kg/day) there was decreased lactase dehydrogenase, increased total cholesterol, total protein, and albumin, elevated female organ-to-body weight ratio for thyroid, liver, spleen, ovaries, and pituitary. In a 13-week dietary study in beagle dogs (administered 167, 1,000, and 4,000 ppm; 4, 25, and 100 mg/kg/day) the LOEL was greater than 167 ppm (4 mg/kg/day) (increased male thyroid and female ovary organ to body weight ratio). At 1,000 ppm (25 mg/kg/day), the same signs were seen as at 167 ppm (4 mg/kg/day), plus diarrhea and increased organ to body weight ratio of thyroid, heart, liver, and adrenals. At 4,000 ppm (100 mg/kg/day), emesis and ataxia in males, decreased body weight gain/food consumption, decreased hematocrit, hemoglobin, immature red blood cells, and alkaline phosphatase, extramedullary hematopoiesis in the liver and spleen, thyroid enlargement with follicular cell hyperplasia, increased organ to body weight ratios for thyroid, adrenals, liver and spleen were seen. In a 13–week dietary study where beagle dogs were administered 167 ppm (4 mg/kg/day), thyroid stimulating hormone (TSH)-stimulated T3 and T4 increased in both sexes. Thyroids were enlarged (both sexes) with absolute weights and organ to body weight ratios increased in females.

EPA believes that there is sufficient evidence for listing 1-bromo-1-(bromomethyl)-1,3-propanedicarbonitrile on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available toxicity data for this chemical.

25. *2-Bromo-2-nitropropane-1,3-diol (bronopol)* (CAS No. 000052-51-7) (FIFRA AI) (Ref. 3). Severe irritation was reported in the gastrointestinal tracts of rats, mice or dogs administered single or multiple oral doses of 2-bromo-2-nitropropane-1,3-diol. In an acute oral study in mice, the LD_{50} of 374 mg/kg resulted in ulceration of the stomach and duodenum, thickening of the intestinal wall, and adhesions of the stomach to the liver. Severe gastric irritation was reported in dogs administered a single oral dose of 250 mg/kg. The NOEL was 100 mg/kg. Superficial ulceration with epithelial hyperplasia and hyperkeratosis, and congested vessels in the gastrointestinal mucosa, was observed in rats fed 80 mg/kg/day (LOEL) in their diet for 13 weeks. The NOEL was 20 mg/kg/day. Vomiting was noted in dogs fed 20 mg/kg/day in their diet for 13 weeks. The NOEL in this study was 8 mg/kg/day. In addition, blood was noted in the urine of these dogs. Mortality, irritation of the gastrointestinal tract, ulceration and stomach lesions were reported in a 2–year dietary study in rats fed 40 mg/kg/day. The NOEL was 10 mg/kg/day. EPA believes that there is sufficient evidence for listing 2-bromo-2-nitropropane-1,3-diol on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available toxicity data.

26. *Bromoxynil (3,5-dibromo-4-hydroxybenzonitrile)* (CAS No. 001689-84-5) (FIFRA AI) (Ref. 3). Developmental effects (hydrocephalus, microphthalmia, anophthalmia and severe defects in ossification of the skull) were observed in rabbits administered 60 mg/kg/day bromoxynil by gavage. The NOEL was 30 mg/kg/day. Developmental toxicity (increases in all forms of supernumerary ribs) was also observed in rats at 5 mg/kg/day. The NOEL was 1.5 mg/kg/day. The maternal LOEL (based on body weight loss) was 30 mg/kg/day. Several other developmental studies indicate potential developmental toxicity of bromoxynil. EPA believes that there is sufficient evidence for listing bromoxynil on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available developmental toxicity data for this chemical.

27. *Bromoxynil octanoate (octanoic acid, 2,6-dibromo-4-cyanophenyl ester)* (CAS No. 001689-99-2) (FIFRA AI) (Ref. 3). Bromoxynil octanoate hydrolyzes to yield bromoxynil and octanol. In a dermal developmental toxicity study, bromoxynil octanoate was developmentally toxic to rat fetuses (increased incidences of supernumerary ribs) at 15 mg/kg/day (LOEL). The NOEL was 10 mg/kg/day. The maternal LOEL for decreased body weight gain was 20 mg/kg/day. The NOEL was 15 mg/kg/day. Developmental effects (hydrocephalus, microphthalmia, anophthalmia and severe defects in ossification of the skull) were observed in rabbits administered 60 mg/kg/day bromoxynil by gavage. The NOEL was 30 mg/kg/day. Developmental toxicity (increases in all forms of supernumerary ribs) was also observed in rats at 5 mg/kg/day. The NOEL was 1.5 mg/kg/day. The maternal LOEL (based on body weight loss) was 30 mg/kg/day. EPA believes that there is sufficient evidence for listing bromoxynil octanoate on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available developmental toxicity data for bromoxynil and bromoxynil octanoate.

28. *Brucine* (CAS No. 000357-57-3) (CERCLA; RCRA APP8; RCRA P) (Ref. 8). Brucine is an alkaloid similar in structure to strychnine. It is capable of causing death or permanent injury due to exposures in normal use. In humans, brucine can cause central and peripheral paralysis, convulsions, and respiratory failure. A potentially lethal oral dose in small children is 5 to 10 mg. The lethal oral dose for an adult may be as low as 30 mg. The acute oral LD_{50} in rabbits is 4 mg/kg.

EPA's exposure analysis indicates that brucine concentrations are likely to exist beyond facility site boundaries, as a result of continuous, or frequently recurring releases, at levels that can

reasonably be anticipated to cause significant adverse acute human health effects. EPA believes that there is sufficient evidence for listing brucine on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(A) based on the available acute toxicity and exposure data for this chemical.

29. *Butylate (Bis-2-methylpropyl)carbamothioic acid S-ethyl ester)* (CAS No. 002008-41-5) (FIFRA AI) (Ref. 3). In a 2–year feeding study in mice, hepatic (cellular infiltrates, focal necrosis) and renal effects (amyloidosis, chronic nephritis, lymphocytic foci) were observed at 80 mg/kg/day. The NOEL was 20 mg/kg/day. In a separate study, liver pericholangitis was observed in rats fed 180 mg/kg/day for 56 weeks. The NOEL was 30 mg/kg/day. An increased relative liver weight was observed in male dogs fed 25 mg/kg/day for 1–year. The NOEL was 5 mg/kg/day. Based on the NOEL, EPA has established a chronic oral RfD of 0.05 mg/kg/day. EPA believes that there is sufficient evidence for listing butylate on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hepatic and renal toxicity data for this chemical.

30. *Butylated hydroxyanisole* (CAS No. 025013-16-5) (CAL; IARC; NTP) (Ref. 8). Butylated hydroxyanisole is classified by IARC as a Group 2B compound; i.e., the chemical is possibly carcinogenic to humans. Butylated hydroxyanisole has been shown to induce gastrointestinal tumors in rats and hamsters. EPA believes that there is sufficient evidence for listing butylated hydroxyanisole on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the carcinogenicity data for this chemical.

31. *C.I. Acid Red 114* (CAS No. 006459-94-5) (TSCA) (Ref. 8). In a 2–year bioassay conducted by the National Toxicology Program (NTP) in which F344 rats were exposed to C.I. Acid Red 114 via drinking water, hepatocellular carcinomas of the liver, tumors of the skin, and adenomas or carcinomas in the Zymbal's gland of both sexes were observed. In the same study, female rats also had increased incidences of adenoma or carcinoma in the clitoral gland, and squamous cell papilloma or carcinoma in the oral cavity. The exposure concentrations in this study ranged from 70 to 300 ppm (9.8 to 42 mg/kg/day) for males and from 150 to 600 ppm (21 to 84 mg/kg/day) for females. EPA believes that there is sufficient evidence for listing C.I. Acid Red 114 on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the carcinogenicity data for this chemical.

32. *C.I. Direct Blue 218* (CAS No. 028407-37-6) (NTP) (Ref. 8). In an NTP bioassay, there was clear evidence of carcinogenicity of C.I. Direct Blue 218 in male and female B6C3F1 mice based on significantly increased incidence of hepatocellular adenomas and carcinomas. In a 2–year NTP feeding study in rats, there was some evidence of carcinogenicity in male F344 rats based on a significant increase in the incidence of squamous cell papillomas of the pharynx in the high dose group (500 mg/kg/day). EPA believes that there is sufficient evidence for listing C.I. Direct Blue 218 on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the carcinogenicity data for this chemical.

33. *Calcium hypochlorite* (CAS No. 007778-54-3) (CERCLA) (Ref. 8). Aquatic acute toxicity data for calcium hypochlorite include a 96–hour measured LC_{50} for rainbow trout of 60 ppb and a 96–hour measured LC_{50} for the Atlantic silverside of 37 ppb. EPA believes that there is sufficient evidence for listing calcium hypochlorite on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available ecotoxicity data for this chemical.

34. *Caprolactam* (CAS No. 000105-60-2) (CAA HAP) (Ref. 7). Rats were administered caprolactam by oral gavage at doses of 0, 100, 500, and 1,000 mg/kg/day on gestation days 6 through 20. This resulted in a LOAEL of 1,000 mg/kg/day and a NOAEL of 500 mg/kg/day for fetal resorption. Rabbits were administered caprolactam by oral gavage at doses of 0, 50, 150, and 250 mg/kg/day on gestation days 6 through 28. This resulted in a LOAEL of 150 mg/kg/day for maternal and fetal body weight depression. In addition, a slight increase in the severity of spontaneous nephropathy (10,000 ppm) was observed in male rats of the first parental generation fed 10,000 ppm of caprolactam in a three-generation reproductive study, resulting in a NOAEL of 1,000 ppm (50 mg/kg/day). Mean body weights and food consumption were reduced in both parental generations at 5,000 and 10,000 ppm. Body weights of offspring were also reduced at these dietary concentrations (the LOAEL was 250 mg/kg/day). EPA believes that there is sufficient evidence for listing caprolactam on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available developmental toxicity data for this chemical.

35. *Carbofuran* (CAS No. 001563-66-2) (CERCLA; EPCRA EHS; FIFRA SR) (Ref. 8). Aquatic acute toxicity test data for carbofuran include a measured 96–hour LC_{50} for bluegill of 80 ppb. In addition, the measured 48–hour EC_{50} for daphnids is 35 ppb. Measured terrestrial acute toxicity data for wildlife include an oral LD_{50} for mallard ducks of 0.397 mg/kg for females and 0.480 mg/kg for males and an oral LD_{50} for female ring-necked pheasants of 4.15 mg/kg. EPA believes that there is sufficient evidence for listing carbofuran on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the environmental toxicity data for this chemical.

36. *Carbon monoxide* (CAS No. 000630-08-0) (CAL) (Ref. 8). Cardiovascular (e.g., electrocardiograph changes, atrial fibrillation, ventricular arrhythmias) and neurological (e.g., headache, dizziness, convulsions, and coma) effects were reported in humans exposed to carbon monoxide. In humans, histological effects in the brain include extensive demyelination of white matter, and necrosis. Neuropsychiatric disorders have also been reported. Persistant electrocardiograph changes, and degeneration of myocardial muscle fibers, hemorrhage and necrosis were observed following inhalation exposure of dogs to 100 ppm (0.11 mg/L) carbon monoxide, 5.5 hours/day, 6 days/week, for 11 weeks. Some of the dogs showed disturbances in gait and in postural and position reflexes. The toxicity of carbon monoxide results from its combination with hemoglobin in the blood to form carboxyhemoglobin which is a poor oxygen carrier. Thus, oxygen delivery by the blood is severely compromised, which leads to tissue hypoxia and possibly tissue poisoning, resulting in the toxic effects (including death) known for this substance.

Infants born to women who survive acute exposure to high concentrations of carbon monoxide during pregnancy often display neurological sequelae and gross brain damage. Exposure of pregnant rats to 150 ppm (0.17 mg/L) carbon monoxide caused reduced pup growth rate, and altered behavior (poor performance on negative geotaxis and homing tests) in pups.

EPA believes that there is sufficient evidence for listing carbon monoxide on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available chronic neurological, myocardial, and developmental toxicity data for this chemical.

Carbon monoxide is regulated under Title I of the CAA (Provisions for Attainment and Maintenance of National Ambient Air Quality Standards). In addition to this proposal to add carbon monoxide to EPCRA section 313, in Units IV.B.179. and 235, EPA is proposing to add two other

chemicals, nitrogen dioxide and sulfur dioxide, that are regulated under Title I of the CAA. Sulfur dioxide is also regulated under Title IV of the CAA (Acid Deposition Control). Extensive data, which are highly technical, are collected on these chemicals as required by the CAA. EPA requests comment on the following: (1) Is the information collected under the CAA sufficient for public right-to-know purposes; and (2) suggestions on how the data collected on these chemicals pursuant to CAA Titles I and IV could be used to meet the purposes of EPCRA section 313.

37. *Carboxin (5,6-dihydro-2-methyl-N-phenyl-1,4-oxathiin-3-carboxamide)* (CAS No. 005234-68-4) (FIFRA AI) (Ref. 3). Decreased body weight gain and food consumption, increased mortality, and reduced kidney, heart and spleen weights were observed in rats fed 600 ppm (30 mg/kg/day) carboxin for 2 years. The NOEL is 200 ppm (10 mg/kg/day). A similar NOEL was established in a three-generation rat reproduction study. Based on the NOEL, EPA established an oral RfD of 0.01 mg/kg/day. In a 90–day feeding study in rats, degeneration of the kidneys was seen at 600 ppm (30 mg/kg/day). The NOEL was 10 mg/kg/day. EPA believes that there is sufficient evidence for listing carboxin on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available renal toxicity data for this chemical.

38. *Chinomethionat (6-methyl-1,3-dithiolo[4,5-b]quinoxalin-2-one)* (CAS No. 002439-01-2) (FIFRA AI) (Ref. 3). Increases in liver weight, liver protein, and both total liver and microsomal RNA levels, as well as inhibition of mixed-function oxidase enzymes (e.g., N-demethylase, cytochrome P-450) were noted in rats administered 75 mg/kg/day by oral gavage for 4 days or in female rats administered 75 mg/kg/day in their diet for 21 days. Liver enlargement was reported in rats fed 10 mg/kg/day in their diet for 35 days. The increase in liver size was attributed to increased cellular protein and an increase in water content. Rats exposed orally to 2,700 mg/kg for 90 days (30 mg/kg/day) had changes in liver weight and effects on the hepatic microsomal oxidases as well as weight loss or decreased body weight gain. In a 1-year dog study, the NOEL was established at 0.6 mg/kg/day for the test material in the diet. The LOEL was 1.9 mg/kg/day as indicated by extra medullary hematopoietic nodules in the liver.

In a developmental toxicity study in rats, increased resorption and decreased fetal weight were reported at 37.5 mg/kg/day (the highest dose tested). The NOEL was 12.5 mg/kg/day. In another developmental study in rats given 30 mg/kg/day in carboxy methyl cellulose by gavage from gestation day 6 to 20, cleft palate, anasarca and micrognathia was observed.

EPA believes that there is sufficient evidence for listing chinomethionat on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hepatic and developmental toxicity data.

39. *Chlorendic acid* (CAS No. 000115-28-6) (NTP) (Ref. 8). Based on sufficient evidence of carcinogenicity in animals IARC classified chlorendic acid as a Group 2B compound; i.e., it is possibly carcinogenic in humans. In an NTP bioassay, there was clear evidence of liver carcinogenicity in both rats and mice. EPA believes that there is sufficient evidence for listing chlorendic acid on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the carcinogenicity data for this chemical.

40. *Chlorimuron ethyl (ethyl-2-[[[(4-chloro-6-methoxyprimidin-2-yl)-carbonyl]-amino]sulfonyl]benzoate)* (CAS No. 090982-32-4) (FIFRA AI) (Ref. 3). In a 1–year dog study, dietary administration of 37.5 mg/kg/day (LOEL) produced an increase in white blood cells in both sexes, a decrease in red blood cells, hematocrit, and hemoglobin in females, and an increase in alkaline phosphatase in males. The NOEL was 6.25 mg/kg/day. Based on the NOEL, an oral RfD of 0.02 mg/kg/day was derived. This study was given a high confidence rating. In a 2–year rat feeding study, changes in hematology parameters were observed at the LOEL of 125 mg/kg/day. The NOEL was 12.5 mg/kg/day. In an 18–month mouse feeding study, centrilobular hepatocellular hypertrophy was observed at 90 days at 187.5 mg/kg/day (LOEL). The NOEL was 18.75 mg/kg/day. EPA believes that there is sufficient evidence for listing chlorimuron ethyl on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hematological toxicity data.

41. *Chlorinated paraffins category* (CAA HAP) (Ref. 7). Chlorinated paraffins are defined as mixtures of linear saturated chlorinated hydrocarbons obtained through the partial chlorination of paraffin, olefin, or acetylene feedstocks which have an average chain length of 10 to 30 carbon atoms and contain average chlorine levels ranging from 40 to 70 percent by weight. Chlorinated paraffins can be described by the general formula: $C_xH_{2x-y+2}Cl_y$ where x ranges from 10 to 30 and y ranges from 3 to 26. Both 58 percent-chlorinated, short-chain (10 to 12 carbons) and 43 percent-chlorinated, long-chain (22 to 26 carbons) chlorinated paraffins were tested in rats and mice by gavage in a 2–year bioassay. The 58 percent-chlorinated, short-chain (10 to 12 carbons) chlorinated paraffins were carcinogenic in rats and mice: dosed male and female mice showed increased incidences of liver tumors, dosed male rats had increased incidences of kidney tubular cell hyperplasia and adenomas or adenocarcinomas (combined), and dosed female rats and mice showed increased thyroid gland follicular cell neoplasms, indicating an EPA Group B2 classification, i.e., a probable human carcinogen. The 43 percent-chlorinated, long-chain (22 to 26 carbons) chlorinated paraffins were carcinogenic in male mice showing an increased incidence of malignant lymphomas, and marginal increase in hepatocellular neoplasms in female mice and adrenal gland pheochromocytomas in female rats, indicating an EPA Group B2 category classification, i.e., the chemical is a probable human carcinogen. EPA believes that there is sufficient evidence for listing chlorinated paraffins on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for these chemicals.

The following ecotoxicity data (LC_{50}s followed by experiment duration in parenthesis) have been reported for short chain (10 to 13 carbons) and intermediate chlorination (59 percent chlorine) chlorinated paraffins: daphnid, 46 ppb (48–hour); mysid shrimp, 14 ppb (96–hour); marine algae, 42 ppb (96–hour); daphnid, 2 ppb and 9 ppb (21–day chronic study); and midge, 78 ppb (49–day chronic study). Ranges of chronic toxicity values are as follow: Freshwater invertebrates, 2 to 162 ppb; freshwater fish, 3 to 17.2 ppb; marine invertebrates, 2.4 to 24 ppb; and marine fish, 2.4 ppb to 620.5 ppm. Chlorinated paraffins are persistent with a half-life of greater than 30 days in the environment. EPA believes that there is sufficient evidence for listing the category chlorinated paraffins on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available ecotoxicity data for these chemicals and their persistence in the environment.

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. When filing reports for chemical categories the releases are determined in the same manner as the thresholds. One report if filed for the category and all releases are reported on this form.

42. *1-(3-Chloroallyl)-3,5,7-triaza-1-azoniaadamantane chloride* (CAS No. 004080-31-3) (FIFRA AI) (Ref. 3). Decrease in heart weight, obliterative vasculitis, and perivasculitis of the hepatic blood vessels were observed in dogs orally administered 1-(3-chloroallyl)-3,5,7-triaza-1-azoniaadamantane for 90 days. The NOEL was 7.5 mg/kg/day; the LOEL was 15 mg/kg/day. EPA believes that there is sufficient evidence for listing 1-(3-chloroallyl)-3,5,7-triaza-1-azoniaadamantane chloride on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available chronic toxicity data for 1-(3-chloroallyl)-3,5,7-triaza-1-azoniaadamantane.

43. *p-Chloroaniline* (CAS No. 000106-47-8) (CERCLA; RCRA APP8; RCRA P) (Ref. 8). In a 78–week study in which rats were fed p-chloroaniline, non-neoplastic proliferative lesions of the splenic capsule (focal fibrosis with subcapsular mesenchymal proliferation) were observed. The LOAEL was 12.5 mg/kg/day (the lowest dose tested) and the RfD derived from this data is 0.004 mg/kg/day. EPA believes that there is sufficient evidence for listing p-chloroaniline on EPCRA section 313 pursuant to section 313(d) (2)(B) based on the chronic toxicity data for this chemical.

44. *5-Chloro-2-(2,4-dichlorophenoxy)phenol* (CAS No. 003380-34-5) (FIFRA AI) (Ref. 3). In a 3–month dog feeding study, decreased red blood cell and hemoglobin values, increased serum alkaline phosphatase, jaundice, and increased liver weight were observed at 25 mg/kg/day (LOEL). No NOEL could be established. In another 3–month dog feeding study, the LOEL of 25 mg/kg/day produced morphologic changes in the liver (focal acidophilic granular degeneration of cytoplasm). The NOEL was 12.5 mg/kg/day. In a 3–month rat feeding study, 125 mg/kg/day (LOEL) produced increased liver weights in males. The NOEL was 50 mg/kg/day. At 150 mg/kg/day (LOEL), decrease in triglycerides, increase in creatinine, decrease in red blood cells, increase in spleen and heart weight, and cytomegaly were observed in another 3–month rat feeding study (NOEL was 50 mg/kg/day). In a 2–year study, dietary administration of 15 mg/kg/day produced decreases in red blood cells, hemoglobin concentration, and hematocrit as well as hepatic necrosis in males. At 50 mg/kg/day, there were decreases in red blood cells in females. EPA believes that there is sufficient evidence for listing 5-chloro-2-(2,4-dichlorophenoxy)phenol on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hematological toxicity data for this chemical.

45. *3-Chloro-2-methyl-1-propene* (CAS No. 000563-47-3) (NTP) (Ref. 8). In an NTP gavage bioassay there was clear evidence of carcinogenicity from 3-chloro-2-methyl-1-propene in rats and mice. The substance induced adrenal cortex, testicular and gastrointestinal tumors in rats and adrenal cortex and gastrointestinal tumors in mice. EPA believes that there is sufficient evidence for listing 3-chloro-2-methyl-1-propene on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the carcinogenicity data for this chemical.

46. *p-Chlorophenyl isocyanate* (CAS No. 000104-12-1) (TSCA) (Ref. 8). p-Chlorophenyl isocyanate is very lethal following inhalation. The 4–hour mouse inhalation LC_{50} value is 0.053 mg/L. In addition, isocyanates as a class are generally severe skin, eye, and respiratory irritants following acute exposure.

EPA's exposure analysis indicates that p-chlorophenyl isocyanate concentrations are likely to exist beyond facility site boundaries, as a result of continuous, or frequently recurring releases, at levels that can reasonably be anticipated to cause significant adverse acute human health effects. EPA believes that there is sufficient evidence for listing p-chlorophenyl isocyanate on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(A) based on the available acute toxicity and exposure data for this chemical.

47. *Chloropicrin* (CAS No. 000076-06-2) (FIFRA AI) (Ref. 3). Measured aquatic acute toxicity data for chloropicrin include a rainbow trout 96–hour LC_{50} of 16.5 ppb, a bluegill 96–hour LC_{50} of 105 ppb, and a 48–hour EC_{50} of 80 ppb. EPA believes that there is sufficient evidence for listing chloropicrin on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data for this chemical.

48. *3-Chloropropionitrile* (CAS No. 000542-76-7) (CERCLA; EPCRA EHS; RCRA APP8; RCRA P) (Ref. 8). 3-Chloropropionitrile is metabolized by hepatic cytochrome P450 enzymes to release cyanide. The substance is readily absorbed both dermally and orally. The mouse oral LD_{50} is 51.3 mg/kg.

EPA's exposure analysis indicates that 3-chloropropionitrile concentrations are likely to exist beyond facility site boundaries, as a result of continuous, or frequently recurring releases, at levels that can reasonably be anticipated to cause significant adverse acute human health effects. EPA believes that there is sufficient evidence for listing 3-chloropropionitrile on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(A) based on the available acute toxicity and exposure data for this chemical.

49. *p-Chloro-o-toluidine* (CAS No. 000095-69-2) (IARC; NTP) (Ref. 8). p-Chloro-o-toluidine is classified as a Group B2 carcinogen by EPA; i.e., the compound is a probable human carcinogen. It is classified as a Group 2B carcinogen by IARC; i.e., a possible human carcinogen. Epidemiology studies are inadequate in evaluating the carcinogenic potential of 4-chloro-o-toluidine hydrochloride in humans. In a long-term feeding study by NCI, p-chloro-o-toluidine hydrochloride induced hemangiomas, hemangiosarcomas, and vascular tumors in mice. An increase in the incidence of pituitary chromophobe adenomas was observed in female rats following dietary administration. EPA believes that there is sufficient evidence for listing p-chloro-o-toluidine on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for this chemical.

50. *Chlorotrifluoromethane (CFC-13)* (CAS No. 000075-72-9) (CAA OD) (Ref. 8). Chlorofluorocarbons, including chlorotrifluoromethane (CFC-13) are known to release chlorine radicals into the stratosphere. Chlorine radicals act as catalysts to reduce the net amount of stratospheric ozone.

Stratospheric ozone shields the earth from ultraviolet-B (UV-B) radiation (i.e., 290 to 320 nanometers). Decreases in total column ozone will increase the percentage of UV-B radiation, especially at its most harmful wavelengths, reaching the earth's surface.

Exposure to UV-B radiation has been implicated by laboratory and epidemiologic studies as a cause of two types of nonmelanoma skin cancers: squamous cell cancer and basal cell cancer. Studies predict that for every 1 percent increase in UV-B radiation, nonmelanoma skin cancer cases would increase by about 1 to 3 percent.

Recent epidemiological studies, including large case control studies, suggest that UV-B radiation plays an important role in causing malignant melanoma skin cancer. Recent studies predict that for each 1 percent change in UV-B intensity, the incidence of

melanoma could increase from 0.5 to 1 percent.

Studies have demonstrated that UV-B radiation can suppress the immune response system in animals, and, possibly, in humans. Increases in exposure to UV-B radiation are likely to increase the incidence of cataracts and could adversely affect the retina.

Aquatic organisms, particularly phytoplankton, zooplankton, and the larvae of many fishes, appear to be susceptible to harm from increased exposure to UV-B radiation because they spend at least part of their time at or near the surface of waters they inhabit.

Increased UV-B penetration has been shown to result in adverse impacts on plants. Field studies on soybeans suggest that yield reductions could occur in some cultivars of soybeans, while evidence from laboratory studies suggest that two out of three cultivars are sensitive to UV-B. Because this increased UV-B radiation can be reasonably anticipated to lead to cancer and other chronic human health effects and significant adverse environmental effects, there is sufficient evidence for listing chlorotrifluoromethane (CFC-13) on EPCRA section 313 pursuant to EPCRA sections 313(d)(2)(B) and (C).

51. *Chlorpyrifos methyl (O,O-dimethyl-O-(3,5,6-trichloro-2-pyridyl)phosphorothioate)* (CAS No. 005598-13-0) (FIFRA AI) (Ref. 3). Humans experienced a 10 percent reduction in plasma cholinesterase activity after 10 dermal exposures to 10 mg/kg/day and a 47 percent reduction after 4 dermal exposures to 25 mg/kg/day (exposures were for 12 hours per day). Rabbits experienced a 97 to 100 percent reduction in plasma cholinesterase activity after 5 dermal exposures to 10 mg/kg/day for 12 hours a day or 2 dermal exposures to 25 mg/kg/day for 12 hours a day. In a 2–year rat feeding study, red blood cell and plasma cholinesterase inhibition were observed at 1 mg/kg/day (LOEL). The NOEL was 0.1 mg/kg/day. In a 2–year dog feeding study, plasma cholinesterase inhibition was observed at 1 mg/kg/day (LOEL). The NOEL was 0.1 mg/kg/day. The oral rat LD_{50} is between 1,159 mg/kg and 3,833 mg/kg. Lethargy, ataxia, diarrhea, salivation, and tremors were observed in these studies. EPA believes that there is sufficient evidence for listing chlorpyrifos methyl on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available neurological toxicity data.

Aquatic acute toxicity values for chlorpyrifos methyl include a daphnid 48–hour LC_{50} of 1.11 ppb and a rainbow trout 96–hour LC_{50} of 12.6 ppb. EPA believes that there is sufficient evidence for listing chlorpyrifos methyl on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data.

52. *Chlorsulfuron (2-chloro-N-[[(4-methoxy-6-methyl-1,3,5-triazin-2-yl)amino]carbonyl]benzenesulfonamide)* (CAS No. 064902-72-3) (FIFRA AI) (Ref. 3). In a rabbit developmental study, an increased incidence of fetal resorptions was observed at the LOEL of 75 mg/kg/day. The NOEL was 25 mg/kg/day.

In a 3-generation rat reproduction study, a decrease in fertility index was observed at 125 mg/kg/day (LOEL). The NOEL was 25 mg/kg/day. EPA believes that there is sufficient evidence for listing chlorsulfuron on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available developmental and reproductive toxicity data for this chemical.

53. *Clomazone (2-[(2-chlorophenyl)methyl]-4,4-dimethyl-3-isoxazolidinone)* (CAS No. 081777-89-1) (FIFRA AI) (Ref. 3). In a 90–day dog feeding study, increased cholesterol and increased absolute and relative liver weights were observed at 62.5 mg/kg/day (LOEL). The NOEL was 12.5 mg/kg/day. Dietary administration of 62.5 mg/kg/day (LOEL) to dogs for 1–year also produced increased cholesterol and increased liver weights. The NOEL was 12.5 mg/kg/day. In a 90–day mouse feeding study, megalocytosis of the liver cells was seen at 2.6 mg/kg/day (LOEL). No NOEL was established. In a 2–year rat feeding study, elevated cholesterol levels and liver-to-body weight ratios were observed at 21.5 mg/kg/day (LOEL). The NOEL was 4.3 mg/kg/day. Dietary administration of 62.5 mg/kg/day (LOEL) to dogs for 1–year increased cholesterol and liver weights. The NOEL was 12.5 mg/kg/day.

In a two-generation reproduction study, decreased pup viability, reduced survival, decreased body weight, and nonfunctional limbs were observed in the offspring of rats that were orally administered 50 mg/kg/day (LOEL). The NOEL was 5 mg/kg/day.

EPA believes that there is sufficient evidence for listing clomazone on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hepatic and developmental toxicity data.

54. *Crotonaldehyde* (CAS No. 004170-30-3) (RCRA APP8) (Ref. 8). Crotonaldehyde has been tested for carcinogenicity in one animal study. When crotonaldehyde was administered to male F344 rats at 0, 42, or 421 mg/L for 113 weeks, there was a statistically significant increase in the incidence of hepatocellular neoplasms (benign and malignant combined) in the low dose group. The lack of tumorigenic effects at the high-dose group is believed to be due to the hepatotoxicity observed in this group. At high dose, crotonaldehyde is cytotoxic; cells died before neoplasms are manifested. Crotonaldehyde and other alpha, beta-unsaturated carbonyls are chemically reactive compounds which can readily react with cellular macromolecules such as DNA and proteins. Mutagenicity studies in a slightly modified preincubation Ames test have clearly shown that crotonaldehyde is mutagenic. EPA believes that there is sufficient evidence for listing crotonaldehyde on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity and mutagenicity data for this chemical.

55. *Cyanazine* (CAS No. 021725-46-2) (CAL; FIFRA SR) (Ref. 8). Cyanazine is a triazine-type herbicide. In a three-generation reproduction study in Long-Evans rats, F_3b female weanlings had increased relative brain weights and decreased relative kidney weights. The LOAEL was 4.05 mg/kg/day and the NOAEL was 1.35 mg/kg/day. In rabbits that received cyanazine in gelatin capsules during gestation days 6 to 18, there was increased postimplantation loss, decreased litter size, and alterations in ossification. In addition, there were increased malformations in the offspring, including anophthalmia/microphthalmia, dilated brain ventricles, dome cranium and thoracoschisis (the LOAEL was 2 mg/kg/day; the NOAEL was 1 mg/kg/day). Similar developmental effects were reported in Fischer 344 rats administered cyanazine during gestation days 6 to 15 (the LOAEL was 25 mg/kg/day; the NOAEL 5 was mg/kg/day). EPA believes that there is sufficient evidence for listing cyanazine on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the developmental toxicity data for this chemical.

56. *Cycloate* (CAS No. 001134-23-2) (FIFRA AI) (Ref. 3). Cycloate, a carbamate pesticide, is a cholinesterase inhibitor. Symptoms of poisoning include salivation, lacrimation, convulsions, and death. Depressed plasma cholinesterase was observed in a 9–week rat inhalation study at 0.0025 mg/L. The NOEL was less than 0.0025 mg/L. Decreased serum cholinesterase (in males and females) and Wallerian degeneration of nerve fibers in spinal cord and sciatic nerve (females) were observed at 0.12 mg/L in a 10-week rat inhalation study (cholinesterase NOEL is 0.012 mg/L). In both inhalation studies, animals were exposed for 6

hours/day, 5 days/week. Plasma, red blood cell, and brain cholinesterase inhibition was reported in rats fed 8 mg/kg/day for 2 years. The NOEL was less than 8 mg/kg/day. Dose-related neuropathy and muscle myopathy were observed. In a 2–year rat feeding study, distended myelin sheath demyelination and nerve fiber loss occurred at 3 mg/kg/day (LOEL). The NOEL was 0.5 mg/kg/day.

Decreased weight and survival were observed in the offspring of rats orally administered 24 mg/kg/day (LOEL) and 72 mg/kg/day of cycloate, respectively (duration and frequency of dosing not reported). The reproductive NOEL was 8 mg/kg/day. Decreased pup weight was observed at 20 mg/kg/day and decreased pup survival was observed at 50 mg/kg/day in a 2-generation rat reproduction study. The NOEL values for these endpoints were 2.5 mg/kg/day and 20 mg/kg/day, respectively.

EPA believes that there is sufficient evidence for listing cycloate on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available neurological and developmental toxicity data.

57. *Cyclohexanol* (CAS No. 000108-93-0) (TSCA) (Ref. 8). Four rabbits exposed to 997 ppm (4 mg/L) for 11 days (6 hours/day, 5 days/week) and a rabbit receiving dermal applications of approximately 2,500 mg/kg/day for 10 days (1 hour/day) developed tremors, central nervous system depression, lethargy or hypothermia.

Microscopic or degenerative changes were observed in the livers and kidneys of rabbits inhaling 145 ppm (0.59 mg/L) of cyclohexanol for 50 days (6 hours/day, 5 days/week), or repeated doses at 272 ppm (1.1 mg/L). In addition, degenerative myocardial effects were observed at this exposure level. Repeated inhalation exposure to higher doses (997 to 1,229 ppm; 4 to 5 mg/L) in rabbits resulted in degenerative changes in the brain and heart as well as liver and kidneys.

Reproductive effects including testicular atrophy, loss of Type A spermatogonia, spermatocytes and spermatozoa, "shrinkage" of seminiferous tubules and Leydig cells, reductions in RNA protein, sialic acid, and glycogen in testes, epididymis and seminal vesicles and increased testicular cholesterol and alkaline phosphatase were observed in male rats or gerbils exposed to 15 mg/kg of cyclohexanol for 21 to 37 days. These changes were accompanied with decreased fertility, and occurred at exposure levels which had no effect on the liver or kidney.

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

58. *Cyfluthrin (3-(2,2-Dichloroethenyl)-2,2-dimethylcyclopropanecarboxylic acid, cyano(4-fluoro-3-phenoxyphenyl)methyl ester)* (CAS No. 068359-37-5) (FIFRA AI) (Ref. 3). In a 14–day rat study, oral administration of 60 mg/kg/day produced tremors, uncoordinated gait, salivation, slight brain hemorrhages, necrosis of the skeletal muscle fibers, and death. The NOEL was not defined. In another study, salivation, straddled gait, axonal degeneration of sciatic nerve, microtubular dilation, and mitochondria degeneration in the sciatic and femoral nerves were observed in rats administered 80 mg/kg/day orally for 5 days and 40 mg/kg/day for the following 9 days. No NOEL was established.

Liver and adrenal weight increases were observed in rats orally administered 40 to 80 mg/kg/day for 28 days. The highest dose of 80 mg/kg/day was reduced to 40 mg/kg/day. The NOEL was 20 mg/kg/day. Liver weight changes and urobilinogen and ketone bodies in the urine were observed in rats fed 15 mg/kg/day for 28 days. No NOEL was established. In a 28–day mouse feeding study, increased liver weight was observed at 50 mg/kg/day (LOEL). The NOEL was 15 mg/kg/day. Inflammatory foci in the kidneys of females were observed at 7.5 mg/kg/day in a 2–year rat feeding study. The NOEL was 2.5 mg/kg/day. Based on the NOEL of the study, an oral RfD of 0.025 mg/kg/day was determined. Increased alkaline phosphatase activity was observed in males at 7.5 mg/kg/day in a 23–month mouse feeding study.

EPA believes that there is sufficient evidence for listing cyfluthrin on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available neurological, hepatic, and renal toxicity data.

Aquatic acute toxicity values for cyfluthrin include a rainbow trout 96–hour LC_{50} of 0.68 ppb, a bluegill 96–hour LC_{50} of 1.5 ppb, and a daphnid 48–hour EC_{50} of 0.14 ppb. EPA believes that there is sufficient evidence for listing cyfluthrin on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data.

59. *Cyhalothrin (3-(2-chloro-3,3,3-trifluoro-1-propenyl)-2,2-dimethylcyclopropanecarboxylic acid cyano(3-phenoxyphenyl)methyl ester)* (CAS No. 068085-85-8) (FIFRA AI) (Ref. 3). Cyhalothrin administered orally (in capsules) to dogs at 10 mg/kg/day for 26 weeks produced occasional disturbances of the nervous system (unsteadiness and/or muscular trembling). The NOEL for these effects was not defined. In a 1–year dog study, ataxia, muscle tremors, and convulsions were observed following oral administration at 3.5 mg/kg/day. Abnormal gait and convulsions were observed at 0.5 mg/kg/day. The LOEL of the study was 0.5 mg/kg/day and the NOEL was 0.1 mg/kg/day. EPA believes that there is sufficient evidence for listing cyhalothrin on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available neurological toxicity data.

60. *Cyromazine (N-cyclopropyl-1,3,5-triazine-2,4,6-triamine)* (CAS No. 066215-27-8) (FIFRA AI) (Ref. 3). In a 6–month dog feeding study, 7.5 mg/kg/day (LOEL) produced changes in hematocrit and hemoglobin levels. The NOEL was 0.75 mg/kg/day. Based on the NOEL, an oral RfD of 0.0075 mg/kg/day was derived. In a 90–day dog feeding study, the LOEL of 25 mg/kg/day produced an increase in relative liver weights in males. The NOEL was 7.5 mg/kg/day. In a 90–day rat feeding study, the LOEL of 15 mg/kg/day produced a decrease in relative liver weights in males. The NOEL was 1.5 mg/kg/day. EPA believes that there is sufficient evidence for listing cyromazine on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hematological toxicity data.

61. *Dazomet (tetrahydro-3,5-dimethyl-2H-1,3,5-thiadiazine-2-thione)* (CAS No. 000533-74-4) (FIFRA AI) (Ref. 3). Animals fed dazomet at a dietary dose of 40 ppm for 2 years showed focal necrosis and fatty metamorphosis of the liver. Rats fed 30.3 mg/kg/day experienced decreased weight gain and changes in liver weight. Renal focal tubular necrosis was seen in rats fed 10 ppm (0.5 mg/kg/day) for 2 years. EPA believes that there is sufficient evidence for listing dazomet on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hepatic and renal toxicity data for this chemical.

62. *Dazomet sodium salt (tetrahydro-3,5-dimethyl-2H-1,3,5-thiadiazine-2-thione, ion(1-), sodium)* (CAS No. 053404-60-7) (FIFRA AI) (Ref. 3). The available toxicity data is on dazomet. Rats fed 80 ppm for 2 years (4 mg/kg/day) showed focal necrosis and fatty metamorphosis of the liver. Rats fed 30.3 mg/kg/day experienced decreased weight gain and changes in liver weight. Renal focal tubular necrosis was seen in

rats fed 10 ppm (0.5 mg/kg/day) for 2 years. EPA believes that there is sufficient evidence for listing dazomet sodium on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available renal toxicity data for its free acid, dazomet.

63. *2,4-DB* (CAS No. 000094-82-6) (FIFRA SR) (Ref. 8). 2,4-DB (4-(2,4-dichlorophenoxy)butanoic acid) is a 2,4-dichlorophenoxy-type herbicide. In a study involving beagle dogs fed a diet containing 2,4-DB for 90 days, a LOAEL of 25 mg/kg/day was determined, based on internal hemorrhaging and mortality observed during the first 3 to 9 weeks of treatment. The NOAEL in this study was 8 mg/kg/day. At this dose level, slight increases in liver weights were observed, but unaccompanied by any gross or histopathologic lesions. EPA has derived an oral RfD of 0.008 mg/kg/day from the LOAEL. In a subchronic rat feeding study, the LOAEL and NOAEL values determined were higher (the LOAEL was approximately 80 to 100 mg/kg/day; the NOAEL was approximately 25 to 30 mg/kg/day), and were based on severe liver and kidney damage.

In the above-mentioned subchronic (90–day) dog feeding study, it was observed that the animals exposed to doses of 2,4-DB at 25 mg/kg/day (the LOAEL) and higher exhibited aspermatogenesis within the first 3 to 9 weeks of treatment. The offspring of rats orally exposed to 17 mg/kg of 2,4-DB during days 1 to 7 of gestation developed abnormalities. There was also an increase in stillbirths at this dose level. In a separate study, offspring of rats orally exposed to 416 mg/kg on days 5 or 9 of gestation exhibited increased preimplantation loss and/or developmental toxicity.

EPA believes that there is sufficient evidence for listing 2,4-DB on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the hepatic, reproductive, and developmental toxicity data for this chemical.

64. *2,4-D butoxyethyl ester* (CAS No. 001929-73-3) (CERCLA; FIFRA AI; IARC) (Ref. 8). 2,4-D butoxyethyl ester is a 2,4-dichlorophenoxy-type herbicide. In mammals, the butoxyethyl ester of 2,4-D is hydrolyzed to yield the free acid, 2,4-D. Therefore, the toxicity of 2,4-D butoxyethyl ester is expected to be similar to that of 2,4-D, in which the kidney, liver, and nervous system are the primary targets of injury. EPA believes that there is sufficient evidence for listing 2,4-D butoxyethyl ester on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the known chronic effects of its metabolite 2,4-D.

65. *2,4-D butyl ester* (CAS No. 000094-80-4) (CERCLA; FIFRA AI; IARC) (Ref. 8). 2,4-D butyl ester is a 2,4-dichlorophenoxytype herbicide. In mammals, the butyl ester of 2,4-D is hydrolyzed to yield the free acid, 2,4-D. Therefore, the toxicity of 2,4-D butyl ester is expected to be similar to that of 2,4-D, in which the kidney, liver, and nervous system are the primary targets of injury. EPA believes that there is sufficient evidence for listing 2,4-D butyl ester on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the known toxic effects of its metabolite 2,4-D.

66. *2,4-D chlorocrotyl ester* (CAS No. 002971-38-2) (CERCLA; FIFRA AI; IARC) (Ref. 8). 2,4-D chlorocrotyl ester is a 2,4-dichlorophenoxy-type herbicide. In mammals, the chlorocrotyl ester of 2,4-D is hydrolyzed to yield the free acid, 2,4-D. Therefore, the toxicity of 2,4-D chlorocrotyl ester is expected to be similar to that of 2,4-D, in which the kidney, liver and nervous system are the primary targets of injury. EPA believes that there is sufficient evidence for listing 2,4-D chlorocrotyl ester on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the known toxic effects of its metabolite 2,4-D.

67. *Desmedipham* (CAS No. 013684-56-5) (FIFRA AI) (Ref. 3). In a 90–day dog study, groups of four beagles/sex were fed diets containing 0 to 5.24 mg/kg/day. This caused increased methemoglobin at 5.24 mg/kg/day (LOEL). EPA believes that there is sufficient evidence for listing desmedipham on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hematological toxicity data.

68. *2,4-D 2-ethylhexyl ester* (CAS No. 001928-43-4) (CERCLA; FIFRA AI; IARC) (Ref. 8). 2,4-D 2-ethylhexyl ester is a 2,4-dichlorophenoxy-type herbicide. The 2-ethylhexyl moiety contains eight carbons and, therefore, is an isooctyl group. Developmental toxicity following maternal exposure to 2,4-D isooctyl esters has been demonstrated in the rat and mouse. Fetotoxicity occurred in offspring of rats exposed to 528 mg/kg during gestation days 8 through 11. Rats orally exposed to doses as low as 302 mg/kg during gestation days 9 through 12 had musculoskeletal abnormalities. Exposure to a lower dose (188 mg/kg) for a longer period during gestation (days 6 through 15) caused developmental effects on homeostasis and effects on newborn growth statistics. In mice, 438 mg/kg administered orally during gestation days 8 to 12 also caused effects on newborn growth statistics.

EPA believes that there is sufficient evidence for listing 2,4-D 2-ethylhexyl ester on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the developmental toxicity data for 2,4-D isooctyl esters, and on the toxic effects of its metabolite 2,4-D.

The aquatic acute toxicity data for 2,4-D isooctyl esters include a measured 48–hour LC_{50} of 8.8 ppm for bluegill. In addition, 2,4-D isooctyl esters are expected to bioaccumulate based on the estimated log K_{ow} of 6.6. EPA believes that there is sufficient evidence for listing 2,4-D 2-ethylhexyl ester on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data and the potential for bioaccumulation.

69. *2,4-D 2-ethyl-4-methylpentyl ester* (CAS No. 053404-37-8) (CERCLA; FIFRA AI; IARC) (Ref. 8). 2,4-D 2-ethyl-4-methylpentyl ester is a 2,4-dichlorophenoxy-type herbicide. The 2-ethyl-4-methylpentyl ester moiety contains eight carbons and, therefore, is an isooctyl group. Developmental toxicity following maternal exposure to 2,4-D isooctyl esters has been demonstrated in the rat and mouse. Fetotoxicity occurred in offspring of rats exposed to 528 mg/kg during gestation days 8 through 11. Rats orally exposed to doses as low as 302 mg/kg during gestation days 9 through 12 had musculoskeletal abnormalities. Exposure to a lower dose (188 mg/kg) for a longer period during gestation (days 6 through 15) caused developmental effects on homeostasis and effects on newborn growth statistics. In mice, 438 mg/kg administered orally during gestation days 8 through 12 also caused effects on newborn growth statistics.

EPA believes that there is sufficient evidence for listing 2,4-D 2-ethyl-4-methylpentyl ester on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the developmental toxicity data for 2,4-D isooctyl esters, the toxic effects of its metabolite 2,4-D. The aquatic acute toxicity data for 2,4-D isooctyl esters include a measured 48–hour LC_{50} of 8.8 ppm for bluegill. In addition, 2,4-D isooctyl esters are expected to bioaccumulate based on the estimated log K_{ow} of 6.6. EPA believes that there is sufficient evidence for listing 2,4-D 2-ethyl-4-methylpentyl ester on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data and the potential for bioaccumulation.

70. *Diazinon* (CAS No. 000333-41-5) (CERCLA; FIFRA SR) (Ref. 8). Diazinon, an organophosphate insecticide, causes plasma cholinesterase inhibition and

central nervous system depression. Significant inhibition of plasma cholinesterase was observed in two men administered five doses of 0.025 mg/kg/day. Diazinon administered to men at doses of 0.05 mg/kg/day for 28 days caused a 35 to 40 percent reduction in plasma cholinesterase. A NOEL for cholinesterase inhibition of 0.02 mg/kg/day was identified from several controlled studies in humans. Clinical symptoms of diazinon poisoning include headache, nausea, sweating, vomiting, and diarrhea all of which are indicative of neurotoxicity. Plasma cholinesterase inhibition (93 percent) and red blood cell inhibition (90 percent) occurred in monkeys orally exposed to diazinon in doses of 5 mg/kg/day for 52 weeks. The NOEL for inhibition of cholinesterase in this study was 0.05 mg/kg/day and the LOEL was 0.5 mg/kg/day.

Urogenital defects in the offspring of female rats orally administered diazinon at doses of 26.4 mg/kg on days 12 to 15 of gestation has been reported. Diazinon also induced musculoskeletal abnormalities in offspring when administered orally to mothers at doses of 45 mg/kg on days 8 to 12 of gestation. Post-implantation mortality was increased in female rats administered 63.5 mg/kg on day 10 of gestation. Similar reproductive and developmental effects were observed in mice. Oral administration of 3.96 mg/kg of diazinon (days 1 to 22 of gestation) caused decreased litter size and delayed behavioral effects in the newborn. Doses of 0.210 mg/kg and 3.78 mg/kg administered orally on days 1 to 21 of gestation caused abnormalities in the immune and reticuloendothelial system and biochemical and metabolic abnormalities of the offspring, respectively.

EPA believes that there is sufficient evidence for listing diazinon on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the developmental and chronic neurotoxicity data for this chemical.

Measured aquatic acute toxicity data for diazinon include a 96–hour LC_{50} for rainbow trout of 90 ppb and a daphnid 96–hour LC_{50} of 0.90 ppb. In addition, measured terrestrial wildlife acute toxicity data for diazinon include an oral LD_{50} for male mallard ducks of 3.54 mg/kg and an oral LD_{50} for male pheasants of 4.33 mg/kg. EPA believes that there is sufficient evidence for listing diazinon on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the environmental toxicity data for this chemical.

71. *2,2-Dibromo-3-nitrilopropionamide* (CAS No. 010222-01-2) (FIFRA AI) (Ref. 3). Oral administration of 50 mg/kg/day (LOEL) to rats for 4 weeks produced dyspnea and weight loss. The NOEL was 25 mg/kg/day. Oral administration of 30 mg/kg/day to rats for 13 weeks produced dyspnea. The NOEL was 13 mg/kg/day. These data may be indicative of direct effects of the compound on the respiratory system. EPA believes that there is sufficient evidence for listing 2,2-dibromo-3-nitrilopropionamide on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available chronic respiratory data.

72. *Dicamba (3,6-Dichloro-2-methyoxybenzoic acid)* (CAS No. 001918-00-9) (FIFRA AI) (Ref. 3). Decreased fetal body weights and increased post-implantation loss was observed in the offspring of rabbits receiving 10 mg/kg/day of dicamba on days 6 through 18 of gestation. The LOEL was 10 mg/kg/day and NOEL was 3 mg/kg/day. Based on the NOEL, EPA derived an oral RfD value of 0.03 mg/kg/day. In a separate study, disorders of oxidative phosphorylation and focal necrosis in the heart were observed in newborn rats following transplacental exposure to dicamba. In a developmental toxicity study, an increase in skeletal malformations was seen in the offspring of rats orally administered 64 mg/kg/day on days 6 through 19 of gestation. EPA believes that there is sufficient evidence for listing dicamba on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available developmental toxicity data for this chemical.

73. *Dichloran (2,6-Dichloro-4-nitroaniline)* (CAS No. 000099-30-9) (FIFRA AI) (Ref. 3). Dichloran, an aniline, is a potential inducer of methemoglobinemia. Either single or repeated oral doses of dichloran produced enlarged livers and induction of microsomal enzymes in the rat. Dogs fed 21 mg/kg/day had increases in serum transaminases. In Rhesus monkeys, where dichloran does not induce hepatic enzymes, 160 mg/kg/day for 3 months caused hepatic centrilobular fatty infiltration and death. Inhalation exposure to 0.17 mg/L produced elevated cholesterol levels and increased liver weight in a 3–month rabbit study and increased liver weight in a 21–day rat study. In a 2–year mouse study, dietary administration of 102.7 mg/kg/day (LOEL) produced centrilobular hepatocyte enlargement, focal necrosis, acute inflammatory cell infiltration, vacuolization of centrilobular hepatocytes, increased weight of the liver and increased incidence of erythropoiesis in males. The NOEL was 30 mg/kg/day. EPA believes that there is sufficient evidence for listing dichloran on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hepatic toxicity data.

74. *3,3'-Dichlorobenzidine dihydrochloride* (CAS No. 000612-83-9) (TSCA) (Ref. 8). IARC has classified 3,3'dichlorobenzidine (o-dichlorobenzidine) as a group 2B compound, i.e. this chemical is possibly carcinogenic in humans. IARC uses the generic name 3,3'-dichlorobenzidine interchangeably with 3,3'-dichlorobenzidine dihydrochloride. The dihydrochloride salt of 3,3'-dichlorobenzidine is expected to be equally as toxic as the free base (3,3'-dichlorobenzidine). EPA believes that there is sufficient evidence for listing 3,3'-dichlorobenzidine dihydrochloride on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on its potential to cause cancer in humans.

75. *3,3'-Dichlorobenzidine sulfate* (CAS No. 064969-34-2) (TSCA) (Ref. 8). IARC has classified 3,3'-dichlorobenzidine (o-dichlorobenzidine) as a group 2B compound, i.e. this chemical is possibly carcinogenic in humans. The sulfate salt of 3,3'dichlorobenzidine is expected to be equally as toxic as the free base (3,3'-dichlorobenzidine). EPA believes that there is sufficient evidence for listing 3,3'-dichlorobenzidine sulfate on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on its potential to cause cancer in humans.

76. *trans-1,4-Dichloro-2-butene* (CAS No. 000110-57-6) (EPCRA EHS) (Ref. 8). Mortality in two of six rats was observed following inhalational exposure to 62 ppm (0.34 mg/L) for 4 hours. An acute inhalation LC_{50} in rats was 86 ppm (0.44 mg/L). EPA's exposure analysis indicates that trans-1,4-dichloro-2-butene concentrations are likely to exist beyond facility site boundaries, as a result of continuous, or frequently recurring releases, at levels that can reasonably be anticipated to cause significant adverse acute human health effects. EPA believes that there is sufficient evidence for listing trans-1,4-dichloro-2-butene on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(A) based on the available acute toxicity and exposure data for this chemical.

77. *Dichloromethylphenylsilane* (CAS No. 000149-74-6) (EPCRA EHS) (Ref. 8). As a class, chlorinated silanes are very corrosive to the skin and mucous membranes and liberate hydrochloric acid in the presence of water. The 2–hour mouse inhalation LC_{50} value for dichloromethylphenylsilane is 0.17 mg/L. EPA's exposure analysis indicates

that dichloromethylphenylsilane concentrations are likely to exist beyond facility site boundaries, as a result of continuous, or frequently recurring releases, at levels that can reasonably be anticipated to cause significant adverse acute human health effects. EPA believes that there is sufficient evidence for listing dichloromethylphenylsilane on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(A) based on the available acute toxicity and exposure data for this chemical.

78. *Dichlorophene (2,2'-methylenebis(4-chlorophenol)* (CAS No. 000097-23-4) (FIFRA AI) (Ref. 3). Increased incidence of microphthalmia was observed in the offspring of rats administered 25 mg/kg/day (teratogenic LOEL). The NOEL was 5.0 mg/kg/day. A dose of 75 mg/kg/day (fetotoxic LOEL) produced delayed ossification of vertebral centra and sternaebrae, reduced body weight and length, and increased resorptions in rat fetuses. The fetotoxic NOEL was 5.0 mg/kg/day. No other developmental studies were available. EPA believes that there is sufficient evidence for listing dichlorophene on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available developmental toxicity data.

Aquatic acute toxicity values for dichlorophene include a measured 48–hour LC_{50} of 50 ppb for Spicodioptomus (calanoid copipod). EPA believes that there is sufficient evidence for listing dichlorophene on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data.

79. *trans-1,3-Dichloropropene* (CAS No. 010061-02-6) (CERCLA; CWA PPL) (Ref. 8). Clinical reports have documented the occurrence of histiocytic lymphoma in two firemen and acute myelomonocytic leukemia in a farmer exposed accidently to 1,3-dichloropropene. Information on the isomer or isomer mixture (i.e., trans/cis isomers) was not specified. The lymphoma and leukemia were refractory to treatment, and all three men died. There is evidence that 1,3-dichloropropene may cause cancer in rats and mice after oral exposure. In a 2–year gavage study, rats treated with 25 or 50 mg/kg/day 1,3-dichloropropene (53 percent cis isomer, 45 percent trans isomer, 1 percent epichlorhydrin) developed squamous cell papillomas and carcinomas of the forestomach. Male rats also developed neoplastic nodules of the liver. Female mice that received 50 or 100 mg/kg/day developed squamous cell papillomas and carcinomas of the forestomach, transitional cell carcinomas of the urinary bladder, and an increased incidence of alveolar/bronchiolar adenomas. A statistically significant increase in bronchioalveolar adenomas was noted in male mice exposed to 60 ppm (272 mg/L) 1,3-dichloropropene vapors (50 percent cis isomer, 43 percent trans isomer). This benign lung tumor was not seen in female mice or in male or female rats. IARC assigned 1,3-dichloropropene to Group 2B, i.e., possibly carcinogenic in humans. EPA believes that there is sufficient evidence for listing trans-1,3-dichloropropene on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for 1,3-dichloropropene (unspecified isomer).

80. *Diclofop methyl (2-[4-(2,4-dichlorophenoxy) phenoxy]propanoic acid, methyl ester)* (CAS No. 051338-27-3) (FIFRA AI) (Ref. 3). In a rat teratology study, increased resorptions, reduced body weights, and dilation of the renal pelvis or distension of the ureter in offspring were reported in rats fed 1.6 mg/kg/day (LOEL). The NOEL was 0.5 mg/kg/day. Increased pup mortality was observed at 5 mg/kg/day (LOEL) in a 3-generation rat reproduction study. The NOEL was 1.5 mg/kg/day.

In a 30–day rat feeding study, increased relative heart, liver, and kidney weights were observed at the LOEL of 4 mg/kg/day. No NOEL was established. Jaundice, increased bilirubin, increased serum glutamic-pyruvic transaminase and serum glutamic-oxaloacetic transaminase, and increased liver and kidney weights were observed in a 30–day dog feeding study at 50 mg/kg/day. The NOEL was 12.5 mg/kg/day. In a 90–day rat feeding study, elevated liver weights and centrilobular enlargement of hepatic cells were observed at 4 mg/kg/day. The NOEL was 1.6 mg/kg/day. Dogs fed 6.25 mg/kg/day for 90 days had increased lipid content and focal changes in the renal cortex. The NOEL was 2 mg/kg/day. EPA believes that there is sufficient evidence for listing diclofop methyl on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available developmental, hepatic, and renal toxicity data.

81. *Dicyclopentadiene* (CAS No. 000077-73-6) (TSCA) (Ref. 8). Convulsions were reported in rats or mice following inhalation of dicyclopentadiene at dosage levels of 332 or 145 ppm (1.8 or 0.78 mg/L), respectively, for 1 or 2 days. The reported acute oral LD_{50} in rats is 353 mg/kg. Animals at this dose level had convulsions and muscle weakness. In a 90–day inhalation study in dogs, neurotoxic symptoms observed included diarrhea, excessive salivation and lack of control of hind quarters. The NOAEL in this study was 8.9 ppm (0.048 mg/L); no LOEL was reported. EPA believes that there is sufficient evidence for listing dicyclopentadiene on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the chronic neurotoxicity data for this chemical.

82. *Diethatyl ethyl* (CAS No. 038727-55-8) (FIFRA AI) (Ref. 3). In a 2–year study, groups of six beagles/sex were given doses orally from 0 to 31.25 mg/kg/day. The lowest dose (0.25 mg/kg/day) produced a positive Coombs test. EPA believes that there is sufficient evidence for listing diethatyl ethyl on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hematological toxicity data for this chemical.

83. *Diflubenzuron* (CAS No. 035367-38-5) (FIFRA SR) (Ref. 8). In a 2–year study in which beagle dogs received diflubenzuron daily in gelatin capsules, the LOAEL for increases in sulfhemoglobin and methemoglobin was 10 mg/kg/day and the NOAEL was 2 mg/kg/day. EPA has derived an oral RfD of 0.02 mg/kg/day for this chemical from this study. Similar effects were noted in two separate 2–year rat feeding studies (the LOAEL was 7.8 to 8 mg/kg/day; the NOAEL was 2 mg/kg/day), and in a lifetime oral study in mice (the LOAEL was 12 mg/kg/day; the NOAEL was 2.4 mg/kg/day). EPA believes that there is sufficient evidence for listing diflubenzuron on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hematological toxicity data.

Measured aquatic acute toxicity data for diflubenzuron include a 48–hour LC_{50} of 4.55 ppb for daphnids. EPA believes that there is sufficient evidence for listing diflubenzuron on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the environmental toxicity data for this chemical.

84. *Diglycidyl resorcinol ether* (CAS No. 000101-90-6) (IARC; NTP) (Ref. 8). Diglycidyl resorcinol ether is classified by IARC as a Group 2B compound, i.e., it is possibly carcinogenic in humans. In an NTP bioassay, rats orally administered 12 mg/kg of diglycidyl resorcinol ether 5 days a week for 103 weeks developed squamous cell papillomas and squamous cell carcinomas of the stomach. Mice orally administered 50 mg/kg 5 days a week for 103 weeks developed squamous cell carcinomas and squamous cell papillomas of the stomach. Mice orally administered 70.5 mg/kg/day of diglycidyl resorcinol ether for 2 years developed blood lymphomas and Hodgkin's disease. Mice receiving

dermal applications of diglycidyl resorcinol ether for 1–year developed skin tumors. EPA believes that there is sufficient evidence for listing diglycidyl resorcinol ether on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the carcinogenicity data for this chemical.

85. *Dimethipin (2,3,-Dihydro-5,6-dimethyl-1,4-dithiin 1,1,4,4-tetraoxide)* (CAS No. 055290-64-7) (FIFRA AI) (Ref. 3). In a 1–year dog feeding study, decreased erythrocyte, hemoglobin, and hematocrit levels as well as increased platelet levels were observed at 75 mg/kg/day. The LOEL for systemic toxicity based on decreased body weight was 7.5 mg/kg/day. No NOEL could be established. In a 2–year rat feeding study, increased absolute and relative liver weights were observed at 10 mg/kg/day (LOEL). The NOEL was 2 mg/kg/day. Based on the NOEL in the study, EPA established an oral RfD of 0.02 mg/kg/day. EPA believes that there is sufficient evidence for listing dimethipin on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hematological and hepatic toxicity data.

86. *Dimethoate* (CAS No. 000060-51-5) (CERCLA; EPCRA EHS; FIFRA SR; RCRA APP8; RCRA P) (Ref. 8). Dimethoate is an organophosphate insecticide. In humans, dimethoate causes typical symptoms of cholinesterase inhibition (sweating, diarrhea, salivation, headache, difficulty in breathing, etc.). In a controlled human study, subjects were administered dimethoate for 57 days. Whole blood and erythrocyte cholinesterase inhibition was observed from day 20 on. The NOEL was 0.202 mg/kg/day, and the LOEL was 0.434 mg/kg/day. In another study in which humans were administered dimethoate for 57 days, the NOEL for cholinesterase inhibition was 15 mg/day (0.2 mg/kg based on a 70 kg person). The LOEL was not specified. Cholinergic symptoms reflective of cholinesterase inhibition following dimethoate administration have also been observed in laboratory animals. A 2–year feeding study in rats determined the NOEL and LOEL for plasma and brain cholinesterase inhibition to be 0.05 and 0.5 mg/kg/day, respectively.

Dimethoate was tested for developmental effects in Wistar rats. Cygon 4E (47.3 percent dimethoate, 52.7 percent unspecified constituents) was administered to pregnant females on days 6 to 15 of gestation. The NOEL for developmental effects was 6 mg/kg/day. At a LOEL of 12 mg/kg/day, an increase in the incidence of wavy ribs was observed in the fetuses. An increase in offspring mortality occurred in a five-generation chronic feeding study (actual doses were 9.5 to 10.5 mg/kg/day) in male and female CD-1 mice. At 12 mg/kg/day (120 mg/kg, gestation days 6 to 15), musculoskeletal abnormalities were observed in the rat offspring. EPA believes that there is sufficient evidence for listing dimethoate on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available developmental and neurotoxicity data for this chemical.

87. *3,3'-Dimethoxybenzidine dihydrochloride (o-Dianisidine dihydrochloride)* (CAS No. 020325-40-0) (TSCA) (Ref. 8). IARC has classified 3,3'-dimethoxybenzidine (o-dianisidine) as a Group 2B compound, i.e., this chemical is possibly carcinogenic. In an NTP carcinogenicity bioassay, increases in neoplasms of the skin, oral cavity, large intestine, liver, uterus, and cervix were noted in rats administered this chemical in drinking water at dose levels of 6, 12, or 21 mg/kg/day in males and 7, 14, or 23 mg/kg/day in females. The dihydrochloride salt of o-dianisidine is expected to be equally as toxic as the free base (o-dianisidine). EPA believes that there is sufficient evidence for listing 3,3'-dimethoxybenzidine dihydrochloride on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on its potential to cause cancer in humans.

88. *3,3'-Dimethoxybenzidine hydrochloride (o-Dianisidine hydrochloride)* (CAS No. 111984-09-9) (TSCA) (Ref. 8). IARC has classified 3,3'-dimethoxybenzidine (o-dianisidine) as a Group 2B compound, i.e., this chemical is possibly carcinogenic. In an NTP carcinogenicity bioassay, increases in neoplasms of the skin, oral cavity, large intestine, liver, uterus and cervix were noted in rats administered this chemical in drinking water at dose levels of 6, 12, or 21 mg/kg/day in males and 7, 14, or 23 mg/kg/day in females. The hydrochloride salt of o-dianisidine is expected to be equally as toxic as the free base (o-dianisidine). EPA believes that there is sufficient evidence for listing 3,3'dimethoxybenzidine hydrochloride on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on its potential to cause cancer in humans.

89. *Dimethylamine* (CAS No. 000124-40-3) (TSCA) (Ref. 8). Dimethylamine is corrosive to the mucous membranes, respiratory tract and eyes of treated animals. B6C3F1 mice and F344 rats exposed to 10 to 175 ppm (0.018 to 0.32 mg/L) dimethylamine via inhalation for 6 to 12 months developed dose-related lesions in the respiratory and olfactory epithelium. Significant decreases in body weight occurred in high-dose (175 ppm; 0.32 mg/L) animals of both species, and some of the high-dose mice died following exposure.

Centrilobular fatty degeneration and necrosis of parenchymal cells were reported in mice, rats, rabbits or guinea pigs administered 97 or 183 ppm (0.18 or 0.34 mg/L) dimethylamine via inhalation for 18 to 20 weeks. Increased liver weight without any histopathological changes were reported following 8–month oral exposure of rats to 0.35 mg/kg/day and guinea pigs exposed to 3.5 mg/kg/day.

Rats administered oral doses of dimethylamine as low as 0.035 mg/kg for 8 months exhibited changes in conditional reflexes including marked attenuation of the excitation process and speedier extinction of the positive reflex.

EPA believes that there is sufficient evidence for listing dimethylamine on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the chronic respiratory, hepatic, and neurological toxicity of this chemical.

90. *Dimethylamine dicamba* (CAS No. 002300-66-5) (FIFRA AI) (Ref. 3). In a pilot rabbit developmental toxicity study, an increase in early and late fetal resorptions was observed in animals receiving the LOEL of 1.0 mg/kg/day. The NOEL was 0.5 mg/kg/day (oral doses, days 6 to 18 of gestation). In another study, increased post-implantation loss was observed in rabbits receiving the LOEL of 10 mg/kg/day (oral doses, days 6 to 18 of gestation). Developmental toxicity was also observed at doses of 10 mg/kg/day in studies with dicamba. EPA believes that there is sufficient evidence for listing dimethylamine dicamba on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available developmental toxicity data for this chemical.

91. *3,3'-Dimethylbenzidine dihydrochloride* (o-Tolidine dihydrochloride) (CAS No. 000612-82-8) (TSCA) (Ref. 8). In a bioassay conducted by NTP, 3,3'-dimethylbenzidine dihydrochloride was found to be carcinogenic in both mice and rats. Male and female mice exposed to concentrations of 5 to 140 ppm (0.95 to 26.6 mg/kg/day) in drinking water for 112 weeks developed lung alveolar cell adenoma and adenocarcinoma. Male and female F344 rats exposed to concentrations of 30 to 150 ppm (4.2 to 21 mg/kg/day) in drinking water for 60 to 61 weeks developed tumors in the gastrointestinal tract, liver, lung and oral cavity. Tumors in the skin, Zymbal's gland, preputial gland in males, clitoral gland and mammary

gland in females, and leukemia in females were also noted in this study. EPA believes that there is sufficient evidence for listing 3,3'-dimethylbenzidine dihydrochloride on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on its potential to cause cancer in humans.

92. *3,3'-Dimethylbenzidine dihydrofluoride (o-Tolidine dihydrofluoride)* (CAS No. 041766-75-0) (TSCA) (Ref. 8). Neither IARC or EPA has classified 3,3'-dimethylbenzidine dihydrofluoride with respect to carcinogenicity. In a bioassay conducted by NTP, however, 3,3'-dimethylbenzidine dihydrochloride was found to be carcinogenic in both mice and rats. Male and female mice exposed to concentrations of 5 to 140 ppm (0.952 to 6.6 mg/kg/day) in drinking water for 112 weeks developed lung alveolar cell adenoma and adenocarcinoma. Male and female F344 rats exposed to concentrations of 30 to 150 ppm (4.2 to 21 mg/kg/day) in drinking water for 60 to 61 weeks developed tumors in the gastrointestinal tract, liver, lung, and oral cavity. Tumors in the skin, Zymbal's gland, preputial gland in males, clitoral gland and mammary gland in females, and leukemia in females were also noted in this study. EPA believes that there is sufficient evidence for listing 3,3'-dimethylbenzidine dihydrofluoride on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on its potential to cause cancer in humans and on the carcinogenicity data for 3,3'-dimethylbenzidine dihydrochloride.

93. *Dimethyl chlorothiophosphate* (CAS. No. 002524-03-0) (EPCRA EHS) (Ref. 8). In a dominant lethal study, male rats were administered dimethyl chlorothiophosphate by gavage for 5 consecutive days and mated to untreated females. The LOEL of 7.5 mg/kg/day was determined based on an increase in preimplantation losses and dead implants. No NOEL for dimethyl chlorothiophosphate was determined from this study. EPA believes that there is sufficient evidence for listing dimethyl chlorothiophosphate on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the developmental toxicity data for this chemical.

94. *Dimethyldichlorosilane* (CAS No. 000075-78-5) (CERCLA; EPCRA EHS) (Ref. 8). As a class, however, chlorinated silanes are very corrosive to the skin and mucous membranes and liberate hydrochloric acid in the presence of water. Dimethyldichlorosilane causes severe burns and the vapor is harmful to humans. The 2-hour mouse inhalation LC_{50} value is 0.30 mg/L. EPA's exposure analysis indicates that dimethyldichlorosilane concentrations are likely to exist beyond facility site boundaries, as a result of continuous, or frequently recurring releases, at levels that can reasonably be anticipated to cause significant adverse acute human health effects. EPA believes that there is sufficient evidence for listing dimethyldichlorosilane on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(A) based on the available acute toxicity and exposure data for this chemical.

95. *N,N-Dimethylformamide* (CAS No. 000068-12-2) (CAA HAP) (Ref. 7). In humans, N,N-dimethylformamide (DMF) produced an increase in subjective symptoms suggestive of mild liver dysfunction in workers and changes in objective measurements of liver damage (serum enzymes and liver enlargement) via inhalation exposure, resulting in a LOAEL of 22 mg/m3 (adjusted LOAEL of 7.9 mg/m3)). Although there are several additional studies which are generally inadequate when considered individually, taken together, these studies demonstrate that DMF exposure is associated with hepatic toxicity in humans. Several animal inhalation studies further support the hepatotoxic effects of DMF. EPA believes that there is sufficient evidence for listing N,N-dimethylformamide on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based upon the available hepatotoxicity data for this chemical.

96. *2,6-Dimethylphenol* (000576-26-1) (TSCA) (Ref. 8). Oral administration of 2,6-dimethylphenol to rats for 8 months produced histologic lesions (the LOEL was 6.0 mg/kg/day; the NOEL was 0.6 mg/kg/day) in the liver, kidneys, and spleen. Another supporting oral study in rats that also reported histological lesions in the liver and kidneys (the LOEL was 6.0 mg/kg/day; the NOEL was 0.06 mg/kg/day) of rats following subchronic oral administration of 2,6-dimethylphenol. EPA believes that there is sufficient evidence for listing 2,6-dimethylphenol on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the hepatotoxicity and nephrotoxicity data for this chemical.

97. *Dinocap* (CAS No. 039300-45-3) (CAL; FIFRA SR) (Ref. 8). Dinocap is a dinitrophenyl-type fungicide. In mice, oral administration of 25 mg/kg/day of dinocap on days 7 to 16 of gestation has been shown to increase post-implantation mortality and reduce newborn viability. Oral administration of 5.0 mg/kg/day to pregnant mice produced developmental toxicity in the offspring (administration of 10 mg/kg/day resulted in abnormalities of the musculoskeletal and hepatobiliary system in the offspring). In the same study, oral administration of 20 mg/kg/day on days 7 to 16 of gestation produced craniofacial abnormalities in offspring. In the same study, behavioral abnormalities and delayed growth were observed in offspring of mice receiving 12 mg/kg/day on days 7 to 16 of gestation. EPA believes that there is sufficient evidence for listing dinocap on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the developmental toxicity data for this chemical.

Measured aquatic acute toxicity data for dinocap indicate that the LC_{50} for rainbow trout is 15 ppb and the LC_{50} for bluegill is 20 ppb. EPA believes that there is sufficient evidence for listing dinocap on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the environmental toxicity data for this chemical.

98. *Dinoseb* (CAS No. 000088-85-7) (CAL; EPCRA EHS; FIFRA SR; RCRA APP8; RCRA P; SDWA) (Ref. 8). Dinoseb is a dinitrophenyl-type herbicide and insecticide. In a three generation reproduction study dinoseb produced decreased pup weights (the LOEL was 1 mg/kg/day; the NOEL was not determined) in the F_{1b}, F_{2a}, and F_{3a} pups. The F_{1b} pup weights diminished (combined sexes) by day 21 at dose levels greater than 1 mg/kg/day. Other studies have shown biologically and statistically significant increases in developmental malformations and/or anomalies (the LOEL was 10 mg/kg/day; the NOEL was 3 mg/kg/day), and an increased incidence of an absence of ossification for a number of skeletal sites and supernumerary ribs (the LOEL was not specified; the NOEL was 3 mg/kg/day). Dinoseb administered by gavage to rabbits from days 6 to 18 of gestation produced neural tube defects (the LOEL was 10 mg/kg/day; the NOEL was 3 mg/kg/day).

The fertility index in male rats was reduced in a reproductive study in animals fed dinoseb at dose levels of 15.6 mg/kg/day or 22.2 mg/kg/day over an 11-week period. Decreased seminal vesicle weight, decreased sperm count and increased incidence of abnormal sperm were noted at dose levels of 9.1 mg/kg/day and higher. The NOEL was 3.8 mg/kg/day.

EPA believes that there is sufficient evidence for listing dinoseb on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the developmental and reproductive toxicity data for this chemical.

Aquatic acute toxicity data for dinoseb include a measured fat-head minnow 96-hour LC_{50} of 88 ppb. EPA

believes that there is sufficient evidence for listing dinoseb on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the environmental toxicity data for this chemical.

99. *Diphenamid* (CAS No. 000957-51-7) (FIFRA SR) (Ref. 8). Diphenamid is a diphenylacetamide-type herbicide. In a 2–year study in dogs fed diphenamid, an increase in liver weight and an increase in portal macrophages and fibroblasts were seen at the LOEL of 10 mg/kg/day. The NOEL was 3 mg/kg/day. Based on the NOEL, an RfD of 0.03 mg/kg/day was derived. In a 2–year study in rats fed diphenamid, an increase in liver weight was seen at the LOEL of 30 mg/kg/day; the NOEL was 10 mg/kg/day. Although, no histopathological changes were reported in these studies, biochemical changes accompanied by histo-pathological changes were observed in a 2–generation study in rat pups. EPA believes that there is sufficient evidence for listing diphenamid on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hepatotoxicity data for this chemical.

100. *Diphenylamine* (CAS No. 000122-39-4) (RCRA APP8) (Ref. 8). Increased liver and kidney weights were noted in dogs that received 25 mg/kg/day (the LOAEL) of diphenylamine in their feed for 2 years. The NOAEL in this study was 2.5 mg/kg/day and the oral RfD was 0.025 mg/kg/day. Pronounced anemia and decreased body weight gain were also noted in these animals. The hepatotoxicity induced by diphenylamine is manifested by peripherolobular fat changes and increased lipids. Vacuolar degeneration and hepatocyte necrosis were reported in rats or guinea pigs that received 2 or 4 percent (i.e., 1,000 or 2,000 mg/kg/day for rats and 800 to 1,600 mg/kg/day for guinea pigs) of diphenylamine in the diet for 6 months. In another 2–year rat study, changes reported in the kidney in diphenylamine-fed animals included epithelial necrosis in the proximal tubule, cystic dilatation of tubules, and interstitial inflammation.

EPA believes that there is sufficient evidence for listing diphenylamine on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the chronic hepatic and renal toxicity data for this chemical.

101. *Dipotassium endothall (7-oxabicyclo(2.2.1)heptane-2,3-dicarboxylic acid, dipotassium salt)* (CAS No. 002164-07-0) (FIFRA AI) (Ref. 3). In a 2–year dog feeding study, increased absolute and relative weight of the stomach and small intestine was observed at 6 mg/kg/day (LOEL). The NOEL was 2 mg/kg/day. An oral RfD of 0.02 mg/kg/day was derived based on the NOEL. EPA believes that there is sufficient evidence for listing dipossium endothall on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available chronic toxicity data for this chemical.

102. *Dipropyl isocinchomeronate* (CAS No. 000136-45-8) (FIFRA AI) (Ref. 3). Dipropyl isocinchomeronate has been classified by EPA as a Group B2 compound, i.e., a probable human carcinogen. This classification is based on the findings of multiple malignant and benign tumors in the rat (liver adenomas and carcinomas in both sexes, kidney carcinomas in both sexes, benign testes tumors in males and uterine tumors in females), and multiple malignant tumors in the mouse (liver adenomas and carcinomas in both sexes and lung/bronchiolar adenomas and carcinomas in males). EPA believes that there is sufficient evidence for listing dipropyl isocinchomeronate on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity toxicity data.

103. *Disodium cyanodithioimidocarbonate* (CAS No. 000138-93-2) (FIFRA AI) (Ref. 3). Rats administered disodium cyanodithioimidocarbonate by gavage on gestation days 6 to 15 demonstrated increased skeletal variations in offspring. The NOEL is 6 mg/kg, and the LOEL is 18 mg/kg. In a rabbit teratology study, increased resorptions were observed in rabbits administered the compound by gavage on gestation days 6 to 18. The NOEL is 3 mg/kg, and the LOEL is 10 mg/kg. EPA believes that there is sufficient evidence for listing disodium cyanodithioimidocarbonate on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available developmental toxicity data.

104. *2,4-D isopropyl ester* (CAS No. 000094-11-1) (CERCLA; FIFRA AI: IARC) (Ref. 8). 2,4-D isopropyl ester is a 2,4-dichlorophenoxy-type herbicide. In mammals, the isopropyl ester of 2,4-D is hydrolyzed to yield the free acid, 2,4-D. Therefore, the toxicity of 2,4-D isopropyl ester is expected to be similar to that of 2,4-D, in which the kidney, liver, and nervous system are the primary targets of injury. 2,4-D is presently included in the EPCRA section 313 list of toxic chemicals. EPA believes that there is sufficient evidence for listing 2,4-D isopropyl ester on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the known toxic effects of its metabolite 2,4-D.

105. *2,4-Dithiobiuret* (CAS No. 000541-53-7) (CERCLA; EPCRA EHS; RCRA APP8; RCRA P) (Ref. 8). In experimental animals, 2,4-dithiobiuret is a highly toxic substance that causes death through respiratory depression and respiratory failure. Rats receiving 1 mg/kg/day for 6 days suffered from delayed onset of neuromuscular depression. Rats given 2,4-dithiobiuret for 52 days showed signs of muscle weakness after a latency period of 3 to 4 days. The NOEL was determined to be 0.125 mg/kg/day. The LOEL was 0.25 mg/kg/day. The cause of the muscle weakness was depressed neuromuscular transmission. EPA believes that there is sufficient evidence for listing 2,4-dithiobiuret on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the chronic neurotoxicity data for this chemical.

106. *Dithiopyr (2-(difluoromethyl)-4-(2-methylpropyl)-6-(trifluoromethyl)-3,5-pyridinedicarbothioic acid S,S-dimethyl ester)* (CAS No. 097886-45-8) (FIFRA AI) (Ref. 3). In a 2-generation rat reproduction study, decreased body weight, diffuse hepatocellular swelling, and "white spots" on the livers were observed in the offspring of rats administered greater than or equal to 16.4 mg/kg/day. The NOEL values were 1.7 mg/kg/day. In a 13–week rat feeding study, the LOEL of 6.62 mg/kg/day produced diffuse hepatocellular swelling. The NOEL was 0.662 mg/kg/day. In a 13-week dog feeding study, increased alkaline phosphatase, discolored livers, and cholestasis was observed at 10 mg/kg/day (LOEL). The NOEL was 1 mg/kg/day. In addition, at 30 mg/kg/day, increased serum glutamic-pyruvic transaminase and serum glutamic oxaloacetic transaminase, increased liver and kidney weights, and decreased cholesterol and albumin were observed. EPA believes that there is sufficient evidence for listing dithiopyr on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hepatic and renal toxicity data.

107. *Diuron* (CAS No. 000330-54-1) (CERCLA) (Ref. 8). In a 2–year study in dogs administered diuron, sulfhemoglobin (an abnormal blood pigment) was detected following doses as low as 3.125 mg/kg/day (LOAEL). The NOAEL was 0.625 mg/kg/day. Higher doses (6.25 and 31.25 mg/kg/day) caused decreased red blood cell, hemoglobin, and hematocrit values. The highest dose tested (31.25 mg/kg/day) also caused an increase in erythrogenic activity in the bone marrow, hemosiderosis in the spleen, increased liver weight, and body weight loss. EPA has derived an oral RfD of 0.002 mg/kg/day for this chemical from this study. Similar effects (anemia, increased erythrogenic activity in the bone

marrow, and abnormal pigments in the blood) were also observed in rats exposed orally to doses as low as 6.25 mg/kg/day for 2 years, or to 250 mg/kg/day for 90 days. In a 7–week study, rats receiving diuron doses of greater than or equal to 10 mg/kg/day had decreased red blood cells and significantly increased methemoglobinemia.

Offspring of Wistar rats fed diuron during days 6 to 15 of gestation showed developmental toxicity, that included malformed ribs, extra ribs, and delayed ossification. The developmental LOAEL in this study was 100 mg/kg/day. No NOAEL was determined. Maternal and fetal body weights decreased at 400 mg/kg/day. In a three-generation reproduction study in rats fed diuron at 6.25 mg/kg/day, decreased body weights were reported in the F_{2b} and F_{3a} litters.

EPA believes that there is sufficient evidence for listing diuron on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hematological and developmental toxicity data for this chemical.

The measured aquatic toxicity data for diuron includes a 1.5–hour EC_{50} of 0.010 ppm (10 ppb) for marine green algae. EPA believes that there is sufficient evidence for listing diuron on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the environmental toxicity data for this chemical.

108. *2,4-D 2-octyl ester* (CAS No. 001917-97-1) (CERCLA; FIFRA AI; IARC) (Ref. 8). 2,4-D 2-octyl ester is a 2,4-dichlorophenoxy-type herbicide. The 2-octyl moiety contains eight carbons and, therefore, is an isooctyl group.

Developmental toxicity following maternal exposure to 2,4-D isooctyl esters has been demonstrated in the rat and mouse. Fetotoxicity occurred in offspring of rats exposed to 528 mg/kg during gestation days 8 to 11. Rats orally exposed to doses as low as 302 mg/kg during gestation days 9 through 12 had musculoskeletal abnormalities. Exposure to a lower dose (188 mg/kg) for a longer period during gestation (days 6 through 15) caused developmental effects on homeostasis and effects on newborn growth statistics. In mice, 438 mg/kg administered orally during gestation days 8 through 12 also caused effects on newborn growth statistics.

EPA believes that there is sufficient evidence for listing 2,4-D 2-octyl ester on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the developmental toxicity data for 2,4-D isooctyl esters, and the toxic effects of its metabolite 2,4-D.

The aquatic acute toxicity data for 2,4-D isooctyl esters include a measured 48–hour LC_{50} of 8.8 ppm for bluegill. In addition, 2,4-D isooctyl esters are expected to bioaccumulate based on the estimated log K_{ow} of 6.6. EPA believes that there is sufficient evidence for listing 2,4-D isooctyl esters on EPCRA section 313 pursuant to section EPCRA 313(d)(2)(C) based on the available environmental toxicity data and the potential for bioaccumulation.

109. *Dodine (dodecylguanidine monoacetate)* (CAS No. 002439-10-3) (FIFRA AI) (Ref. 3). Aquatic acute toxicity values for dodine include a daphnid 48–hour EC_{50} of 17.8 ppb. EPA believes that there is sufficient evidence for listing dodine on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data.

110. *2,4-DP (dichlorprop)* (CAS No. 000120-36-5) (FIFRA SR; IARC) (Ref. 8). 2,4-DP (2-(2,4-dichlorophenoxy)propionic acid) is a 2,4-dichlorophenoxy-type herbicide. Developmental toxicity has been reported in rats and mice administered oral doses of 2,4-DP as low as 20 mg/kg during gestation days 4 through 18. Behavioral changes and physical effects were observed in newborn rats, while increased post-implantation loss was observed in the mothers. Exposure of mice to much higher doses (3,000 and 4,000 mg/kg) for shorter durations (i.e., gestation days 6 through 15) caused musculoskeletal abnormalities and fetotoxicity.

EPA believes that there is sufficient evidence for listing 2,4-DP on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available developmental toxicity data for this chemical.

111. *2,4-D propylene glycol butyl ether ester* (CAS No. 001320-18-9) (CERCLA; FIFRA AI; IARC) (Ref. 8). 2,4-D propylene glycol butyl ether ester is a 2,4-dichlorophenoxy-type herbicide. In mammals, the propylene glycol butyl ether ester is expected to hydrolyze to yield the free acid, 2,4-D. Therefore, the toxicity of 2,4-D propylene glycol butyl ether ester is expected to be similar to that of 2,4-D, in which the kidney, liver, and nervous system are the primary targets of injury. EPA believes that there is sufficient evidence for listing 2,4-D propylene glycol butyl ether ester on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the chronic toxicity data for this chemical.

112. *2,4-D sodium salt* (CAS No. 002702-72-9) (CERCLA; FIFRA AI; IARC) (Ref. 8). 2,4-D sodium salt is a 2,4-dichlorophenoxy-type herbicide. In mammals, the sodium salt is expected to hydrolyze to yield the free acid, 2,4-D. Therefore, the toxicity of 2,4-D sodium salt is expected to be similar to that of 2,4-D, in which the kidney, liver, and nervous system are the primary targets of injury. 2,4-D is presently included in the EPCRA section 313 list of toxic chemicals. EPA believes that there is sufficient evidence for listing 2,4-D sodium salt ester on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the systemic toxicity data for this chemical.

113. *Ethoprop (phosphorodithioic acid O-ethyl S,S-dipropyl ester)* (CAS No. 013194-48-4) (FIFRA AI) (Ref. 3). Ethoprop is acutely toxic to animals. The acute oral LD_{50} in rats is 5.62 mg/kg/day. Clinical signs of toxicity observed in animals at this dose level included depression, salivation, inactivity, convulsions and prostration. Similar signs were reported at the 4–hour inhalation LC_{50} of 0.12 mg/L in rats. In a 2–year rat chronic feeding study, plasma, red blood cell, and brain cholinesterase inhibition were observed in both sexes at 0.5 mg/kg/day. The NOEL was 0.05 mg/kg/day. Similar results were reported in a chronic dietary study in mice at 0.1 mg/kg/day. The NOEL was 0.01 mg/kg/day. The two chronic studies together with the results of acute studies indicate the potential neurotoxicity of ethoprop. EPA believes that there is sufficient evidence for listing ethoprop on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available neurological toxicity data.

Aquatic acute toxicity values for ethoprop include a mysid 96–hour LC_{50} of 7.5 ppb, a shrimp 96–hour LC_{50} of 13 ppb, and a daphnid 48–hour EC_{50} of 93 ppb. Avian acute and dietary toxicity data include a ring-necked pheasant 14–day LD_{50} of 4.2 mg/kg and a mallard duck 14–day LD_{50} of 12.6 mg/kg. EPA believes that there is sufficient evidence for listing ethoprop on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data for this chemical.

114. *Ethyl dipropylthiocarbamate (EPTC)* (CAS No. 000759-94-4) (FIFRA AI) (Ref. 3). EPTC is a cholinesterase inhibitor. Workers exposed to EPTC complained of headache, malaise, nausea, and impaired working ability. Poisoned animals exhibited salivation, lacrimation, blepharospasm, and depression. Neuropathy was observed in rats orally administered 25 mg/kg/day for 2 years. The LOEL was 25 mg/kg/day and the NOEL was 5 mg/kg/day. Decreased brain cholinesterase activity was observed in female rats orally administered 15 mg/kg/day (LOEL). The

NOEL was 3 mg/kg/day. The 4-hour inhalation rat and cat lowest-lethal-concentration values are 0.2 mg/L and 0.4 mg/L, respectively. Somnolence and salivation were observed in exposed animals. The dermal rabbit LD_{50} is 10,000 mg/kg. Depressed righting reflexes, prostration, and clonic convulsions were observed.

In a 2–year dietary rat study, degenerative cardiomyopathy was observed in males receiving 9 mg/kg/day of EPTC. No NOEL was established. This effect was observed in females at 36 mg/kg/day. The NOEL was 18 mg/kg/day. In a 2-generation rat reproduction study, parental toxicity included cardiomyopathy observed in rats orally administered 10 mg/kg/day. Based on the NOEL of 2.5 mg/kg/day, EPA derived an oral RfD of 0.025 mg/kg/day. In a 2–year dietary rat study, chronic myocarditis was observed at the LOEL of 125 mg/kg/day. The NOEL was 25 mg/kg/day.

An increased incidence of fetal resorptions, increased incidence of fetal retardations, and decreased fetal body weights were observed in rats receiving 300 mg/kg/day of EPTC on days 6 to 15 of gestation. The LOEL was 300 mg/kg/day and the NOEL was 100 mg/kg/day. The NOEL was 10 mg/kg/day. In a 2-generation rat reproduction study, decreased pup weight was observed in both generations at 40 mg/kg/day. The NOEL was 10 mg/kg/day.

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

115. *Famphur* (CAS No. 000052-85-7) (CERCLA; FIFRA AI; RCRA APP8; RCRA P) (Ref. 8). Famphur is a thiophosphate-type cholinesterase inhibitor. In a 90–day feeding study, rats given diets supplemented with famphur showed decreased plasma and brain cholinesterase activity at 1.25 mg/kg/day, and decreased whole blood cholinesterase activity at 0.15 mg/kg/day. A bull was treated with famphur for 43 days before signs of neurotoxicity appeared. The symptoms, including paresis of all four limbs, were attributed to focal cervical or diffuse spinal cord lesions. Calves receiving 60.75 mg/kg showed marked inhibition of whole blood cholinesterase. EPA believes that there is sufficient evidence for listing famphur on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the chronic neurotoxicity known for this chemical.

Measured terrestrial wildlife acute toxicity data for famphur indicate that the oral LD_{50} values for the redwinged blackbird and the starling are 1.78 mg/kg and 4.22 mg/kg, respectively. In addition, the measured oral LD_{50} for mallard ducks is 3.45 mg/kg (based on 35 percent active ingredient). EPA believes that there is sufficient evidence for listing famphur on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the environmental toxicity data for this chemical.

116. *Fenarimol (.alpha.-(2-chlorophenyl)-.alpha.-4-chlorophenyl)-5-pyrimidinemethanol)* (CAS No. 060168-88-9) (FIFRA AI) (Ref. 3). In a 3–month mouse feeding study, liver weights were increased in males at levels greater than or equal to 620 ppm (80.6 mg/kg/day) and in females at levels greater than 1,100 ppm (143 mg/kg/day). At higher doses (143 to 260 mg/kg/day), decreased total bilirubin, hepatomegaly, and/or periportal fatty liver changes were observed. Mice exposed to dietary levels of 78 mg/kg/day for 1–year had increased liver weight and slight fatty changes. One year feeding studies in Wistar rats also resulted in increased liver weights (the LOEL was 17.5 mg/kg/day; the NOEL was 6.5 mg/kg/day). In a 2–year feeding study with Wistar rats, fatty changes in the liver were observed at 17.5 mg/kg/day (LOEL). The NOEL was 6.5 mg/kg/day. A 2–year feeding study in mice resulted in fatty liver changes. The LOEL was 78 mg/kg/day and the NOEL was 22.1 mg/kg/day. EPA believes that there is sufficient evidence for listing fenarimol on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hepatic toxicity data.

117. *Fenbutatin oxide (hexakis(2-methyl-2-phenylpropyl)distannoxane)* (CAS No. 013356-08-6) (FIFRA AI) (Ref. 3). In a rat teratology study, the LOEL for developmental toxicity (toxic to zygote) was 60 mg/kg/day and the NOEL was 30 mg/kg/day. In a rabbit teratology study, oral administration of 5 mg/kg/day produced intrauterine lethality and was also toxic to maternal animals. The NOEL was 1 mg/kg/day. In a 3-generation rat reproduction study, administration of 15 mg/kg/day (LOEL) produced decreased viability index. The NOEL was 5 mg/kg/day. EPA believes that there is sufficient evidence for listing fenbutatin oxide on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available developmental toxicity data for this chemical.

Aquatic acute toxicity values for fenbutatin oxide include a rainbow trout 96–hour LC_{50} of 1.7 ppb, a fathead minnow 96–hour LC_{50} of 1.9 ppb, a daphnid 48–hour EC_{50} of 3.1 ppb, a bluegill sunfish 96–hour of LC_{50} of 4.8 ppb, and a sheepshead minnow 96–hour LC_{50} of 20.8 ppb. Avian acute toxicity values include a quail oral LD_{50} of 0.007 mg/kg. EPA believes that there is sufficient evidence for listing fenbutatin oxide on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data for this chemical.

118. *Fenoxaprop ethyl (2-(4-((6-chloro-2-benzoxazolylen)oxy)phenoxy)propanoic acid,ethyl ester)* (CAS No. 066441-23-4) (FIFRA AI) (Ref. 3). In a 30–day mouse feeding study, liver weight increases were observed (LOEL 20 ppm or 2.6 mg/kg/day and NOEL 10 ppm or 1.3 mg/kg/day). In a 32–day rat feeding study, changes in the liver and kidney as well as altered lipid metabolism and decreased cholesterol were observed. The LOEL in the rat study was 80 ppm (4 mg/kg/day). The NOEL was 20 ppm (1 mg/kg/day). Inflammatory changes in the kidney (chronic interstitial nephritis) were reported in dogs that received a 3–month feeding of 80 ppm (2 mg/kg/day, the LOEL). The NOEL was 16 ppm or 0.4 mg/kg/day. Decreased serum lipids and cholesterol were reported in rats exposed for 2 years to dietary levels greater than or equal to 180 ppm (9 mg/kg/day, the LOEL). The NOEL in this study was 30 ppm (1.5 mg/kg/day).

In a developmental toxicity study, fetotoxic effects (slightly impaired growth and delayed ossification) were reported at 100 mg/kg/day. The NOEL was 32 mg/kg/day. These effects were observed at doses that were also toxic to maternal animals. In a 2–generation reproductive toxicity feeding study in rats, decreased survival, decreased body weight at study termination, and significant changes in kidney and liver weights were reported in the F_{2a} and F_{2b} litters. The fetotoxic LOEL in this study was 5 ppm (0.25 mg/kg/day, the lowest dose tested). The LOEL and NOEL for maternal toxicity (increased kidney and liver weights) were 80 ppm (4 mg/kg/day) and 30 ppm (1.5 mg/kg/day), respectively. Thus, the fetotoxic effects were observed at doses lower than those that produced maternal toxicity.

EPA believes that there is sufficient evidence for listing fenoxaprop ethyl on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available renal and developmental toxicity data for this chemical.

Aquatic acute toxicity values for fenoxaprop ethyl include a mysid 96–hour EC_{50} of 98 ppb. EPA believes that there is sufficient evidence for listing fenoxaprop ethyl on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C)

based on the available environmental toxicity data.

119. *Fenoxycarb (2-(4-phenoxyphenoxy)ethyl]carbamic acid ethyl ester)* (CAS No. 072490-01-8) (FIFRA AI) (Ref. 3). Liver changes (including fatty changes, glycogen depletion, hepatocyte hypertrophy and multinucleated hepatocytes) were reported in mice (the LOEL was 80 mg/kg/day; the NOEL was not determined) and rats (the LOEL was 300 mg/kg/day; the NOEL was 100 mg/kg/day) following 3–month dietary exposures. Dose-related changes in the liver of male rats, including increased relative liver weight, focal necrosis, centrilobular hypertrophy and pigmented histiocytes, were reported after the first year of a 2–year oncogenicity study. The LOEL for these effects was 600 ppm (30 mg/kg/day) and the NOEL was 200 ppm (10 mg/kg/day). Male and female rats exposed to a higher dose (1,800 ppm or 90 mg/kg/day) in this study had increased alkaline phosphatase and reduced platelets and white blood cells, and fibrosis was present in the hepatic lesions in the males.

In a reproduction study in rats, delays in pinna unfolding and eye opening were reported at 10 mg/kg/day.

EPA believes that there is sufficient evidence for listing fenoxycarb on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hepatic and developmental toxicity data for this chemical.

120. *Fenpropathrin (2,2,3,3-tetramethylcyclopropane carboxylic acid cyano(3-phenoxyphenyl)methyl ester)* (CAS No. 039515-41-8) (FIFRA AI) (Ref. 3). In a 1–year feeding study, tremors were noted in dogs exposed to 6.25 mg/kg/day. The NOEL was 2.5 mg/kg/day. In a developmental toxicity study in rats, signs of neurotoxicity reported in the pregnant dams included ataxia, tremors, convulsions, lacrimation, prostration of death. The LOEL for maternal toxicity was 10 mg/kg/day and the NOEL was 6 mg/kg/day. In 2–year dietary studies in rats and mice, body tremors and increased mortality were observed in male rats (the LOEL was 30 mg/kg/day; the NOEL was 22.5 mg/kg/day), whereas only marginally increased hyperactivity was noted in female mice (the LOEL was 65.2 mg/kg/day; the NOEL was 16.2 mg/kg/day). EPA believes that there is sufficient evidence for listing fenpropathrin on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available neurological toxicity data for this chemical.

Aquatic acute toxicity values for fenpropathrin include a rainbow trout 96–hour LC_{50} of 2.3 ppb, a bluegill 96–hour LC_{50} of 2.2 ppb, a sheepshead minnow 96–hour LC_{50} of 3.1 ppb, and a daphnid 48–hour EC_{50} of 0.53 ppb. EPA believes that there is sufficient evidence for listing fenpropathrin on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data for this chemical.

121. *Fenthion (O,O-dimethyl O-[3-methyl-4-(methylthio) phenyl] ester, phosphorothioic acid)* (CAS No. 000055-38-9) (FIFRA AI) (Ref. 3). In cases of human poisonings from fenthion exposure, reported cholinergic manifestations included the following: A man who ingested 257 mg/kg had an increased pulse rate (no effect on blood pressure) and gastrointestinal symptoms including diarrhea and nausea or vomiting; a woman that ingested 525 mg/kg experienced muscle contraction or spasticity, respiratory depression, and miosis; a woman that ingested an unspecified amount of fenthion did not exhibit the initial cholinergic crisis until 5 days postexposure, and symptoms (primarily psychosis) recurred 24 days later. Similar signs of toxicity, characteristic of organophosphate poisoning, were observed in rats that were fed 300 ppm (15 mg/kg/day). Symptoms reported in these rats included spasms, nervousness, salivation and diarrhea as well as ophthalmological symptoms such as eyeball protrusion and corneal turbidity. LOEL and NOEL values for cholinesterase inhibition from animal studies of various durations include the following: In a 28–day feeding study in rats, the LOEL was 10 ppm (0.5 mg/kg/day) and the NOEL was 5 ppm (0.65 mg/kg/day) for brain cholinesterase inhibition; in another 28–day rat feeding study, plasma and erythrocyte cholinesterase recovered 2 weeks postexposure. The LOEL for cholinesterase inhibition in a 30–day inhalation study in rats was 0.163 mg/L. In a 63–day rat feeding study, significant cholinesterase inhibition occurred by day 3 at 25 mg/kg/day. In a 16–week feeding study in rats, the LOEL for cholinesterase inhibition was 5 ppm in females (0.65 mg/kg/day) and the NOEL was 3 ppm (0.15 mg/kg/day). EPA believes that there is sufficient evidence for listing fenthion on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available neurological toxicity data for this chemical.

Aquatic acute toxicity values for fenthion include a daphnid 48–hour LC_{50} of 0.62 ppb for immobilization. Acute toxicity values for other non-standard aquatic invertebrates range from a 48–hour EC_{50} of 0.024 ppb for brown shrimp to a 96–hour EC_{50} of 110 ppb for scud. Avian acute toxicity values include a male mallard duck oral LD_{50} of 5.94 mg/kg, a male bobwhite quail LD_{50} of 4 mg/kg, and a mourning dove oral LD_{50} of 4.63 mg/kg. EPA believes that there is sufficient evidence for listing fenthion on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data for this chemical.

122. *Fenvalerate (4-chloro-alpha-(1-methylethyl)benzeneacetic acid cyano(3-phenoxyphenyl)methyl ester)* (CAS No. 051630-58-1) (FIFRA AI) (Ref. 3). Excitement and ataxia were observed in rats administered fenvalerate at the oral LD_{50} dose of 70.2 mg/kg. The oral mouse LD_{50} for fenvalerate is 185 mg/kg. Tremor, convulsions, and ataxia were observed in this study. Neurological dysfunctions consisting of jerky leg movements, exaggerated flexion of the hind limb, and unsteady gait were observed in rats fed 7.5 mg/kg/day (LOEL) of fenvalerate for 13 weeks. The NOEL was 2.5 mg/kg/day. Based on the NOEL of the study, EPA derived an oral RfD of 0.0025 mg/kg/day. Peripheral nerve and spinal cord lesions were observed in rats orally administered 360 mg/kg.

In a 6–month dog feeding study, normocytic anemia, increased serum cholesterol levels, and hepatic microgranulomatosis were observed in animals administered fenvalerate at 6.25 mg/kg/day (LOEL). No NOEL was defined. In a 2–year mouse feeding study, multifocal granulomata in the liver was observed in males and females fed fenvalerate at 7.5 and 37.5 mg/kg/day, respectively. The male NOEL was 1.5 mg/kg/day and the female NOEL was 7.5 mg/kg/day. In a 20–month mouse feeding study, decreased erythrocyte count, increased mean cell volume of the blood, and granulomatous changes in the liver were observed at 15 mg/kg/day (LOEL). The NOEL was 4.5 mg/kg/day.

EPA believes that there is sufficient evidence for listing fenvalerate on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available neurological, hepatic, and hematological toxicity data for this chemical.

Measured aquatic acute toxicity data for fenvalerate include a bluegill 96–hour LC_{50} of 0.26 ppb, a fathead minnow 96–hour LC_{50} of 0.33 ppb, a rainbow trout 96–hour LC_{50} of 1.2 ppb, an Atlantic salmon 96–hour LC_{50} of 1.2 ppb, and a sheepshead minnow 96–hour LC_{50} of 4.4 ppb. In addition, the 48–hour LC_{50} for daphnids is 0.05 ppb. EPA believes that there is sufficient evidence

for listing fenvalerate on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the environmental toxicity data for this chemical.

123. *Ferbam (tris(dimethylcarbamodithioato-S,S')iron)* (CAS No. 014484-64-1) (FIFRA AI) (Ref. 3). In an 80–week feeding study in rats, females fed 96 mg/kg/day had ataxia that progressed to hind limb paralysis. The NOEL was not determined. Symptoms of neurotoxicity reported in mice following acute oral exposure included somnolence, excitement and ataxia, although the doses at which these signs occurred were much higher (the LD_{50} in this study was 3,400 mg/kg). EPA believes that there is sufficient evidence for listing ferbam on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available neurological toxicity data.

Aquatic acute toxicity values for ferbam include a daphnid 48–hour LC_{50} of 90 ppb, a 96–hour LC_{50} of 52 ppb for the eastern oyster, and a guppy 96–hour LC_{50} of 90 ppb. EPA believes that there is sufficient evidence for listing ferbam on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data for this chemical.

124. *Fluazifop butyl (2-[4-[[5-(trifluoromethyl)-2-pyridinyl]oxy]-phenoxy]propanoic acid, butyl ester)* (CAS No. 069806-50-4) (FIFRA AI) (Ref. 3). A 3–month rat feeding study demonstrated hepatocyte hypertrophy in males (the LOEL was 5 mg/kg/day; the NOEL was 0.5 mg/kg/day). In a 1–year feeding study, dogs had changes in serum alkaline phosphatase and alanine aminotransferase and/or alanine sulfatransferase (the LOEL was 25 mg/kg/day; the NOEL was 5 mg/kg/day). Similar changes were also reported in dogs following 3 months exposure in their diet (the LOEL was 125 mg/kg/day). In a carcinogenicity study, male mice fed 20 ppm (2.6 mg/kg/day, the LOEL) had an increased incidence of hepatocyte hypertrophy. The NOEL was 5 ppm or 0.65 mg/kg/day. Male and female mice exposed to a higher dose of 80 ppm (10.4 mg/kg/day) had increased liver weight (relative and absolute) and hypertrophy of periacinal hepatocytes. Males in this dose group also had increased pigmentation in hepatocytes and Kupffer cells.

In a teratogenicity study in Sprague-Dawley rats exposed via oral gavage, delayed ossification and an increased incidence of hydroureter were observed in fetuses (the fetotoxic LOEL was 5 mg/kg/day; the NOEL 1 mg/kg/day) and a teratogenic LOEL of 200 mg/kg/day (the NOEL was 10 mg/kg/day) was determined based on the incidence of diaphragmatic hernia. Maternal toxicity was observed in this study at doses higher than those causing fetotoxicity and included reduced body weight gain and decreased gravid uterus (the maternal LOEL was 200 mg/kg/day; the NOEL was 10 mg/kg/day). In a 2-generation reproductive toxicity dietary study in Wistar rats, the reproductive LOEL of 250 ppm (12.5 mg/kg/day; the NOEL was 80 ppm or 4 mg/kg/day) was based on reduced litter sizes, reduced viability, reduced testis and epididymis weights and tubular atrophy in offspring. Fetotoxicity (delayed ossification and eye opacities) was also demonstrated in New Zealand White rabbits (the LOEL was 30 mg/kg/day; the NOEL was 10 mg/kg/day). EPA believes that there is sufficient evidence for listing fluazifop butyl on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hepatic and developmental toxicity data for this chemical.

125. *Flumetralin (2-chloro-N-(2,6-dinitro-4-(trifluoromethyl)- phenyl)-N-ethyl-6-fluorobenzenemethanamine)* (CAS No. 062924-70-3) (FIFRA AI) (Ref. 3). Aquatic acute toxicity values for flumetralin include a daphnid 48–hour EC_{50} of greater than 2.8 ppb, a bluegill sunfish 96–hour LC_{50} of greater than 3.2 ppb, and a rainbow trout 96–hour LC_{50} of greater than 3.2 ppb. EPA believes that there is sufficient evidence for listing flumetralin on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data for this chemical.

126. *Fluorine* (CAS No. 007782-41-4) (CERCLA; EPCRA EHS; RCRA APP8; RCRA P) (Ref. 8). Inhalation of fluorine causes initial coughing, choking and chills, which is followed 1 or 2 days later with pulmonary edema. Fluorine has a strong caustic action on mucous membranes, eyes and skin. In human volunteers exposed to 100 ppm (0.16 mg/L) for 30 seconds, much irritation to the nose and eyes was reported. In acute inhalation studies in animals, lethality occurs at a fairly uniform level and is the result of pulmonary edema. Following 1 hour exposures in mice, rats or guinea pigs, the inhalation LC_{50} values ranged from 150 to 185 ppm (0.23 to 0.29 mg/L). The LC_{50} for rabbits following a 30-minute exposure was 270 ppm (0.42 mg/L). EPA's exposure analysis indicates that fluorine concentrations are likely to exist beyond facility site boundaries, as a result of continuous, or frequently recurring releases, at levels that can reasonably be anticipated to cause significant adverse acute human health effects. EPA believes that there is sufficient evidence for listing fluorine on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(A) based on the available acute toxicity and exposure data for this chemical.

127. *Fluorouracil (5-Fluorouracil)* (CAS No. 000051-21-8) (CAL; EPCRA EHS) (Ref. 8). A major use of fluorouracil is in the palliative treatment of carcinoma of the colon, rectum, breast, stomach, and pancreas that is not amenable to surgery or irradiation. The major toxic effects of fluorouracil are on the normal, rapidly proliferating tissues particularly of the bone marrow and lining of the gastrointestinal tract. Leukopenia, predominantly of the granulocytopenic type, thrombocytopenia, and anemia occur commonly with intravenous fluorouracil therapy at doses ranging from 6 to 12 mg/kg. Pancytopenia and agranulocytosis also have occurred.

Developmental abnormalities or other effects on newborns were reported in offspring of women receiving 150 or 240 mg/kg fluorouracil intravenously during weeks 11 to 14 or 20 to 31 of pregnancy. In addition, maternal toxicity to the reproductive organs, toxicity to the fetus, and developmental abnormalities have been reported in mice, rats, and hamsters receiving oral, intraperitoneal, or intramuscular doses of fluorouracil ranging from 10 to 700 mg/kg.

Chronic neurotoxic effects were noted in dogs fed fluorouracil at a dietary dose of 2 mg/kg/day for 6 months. In this study, animals were examined at the end of 3 months and 6 months. At the end of the experiment, or at death, the brain was removed and examined (only one dog survived the entire 6–month period). Histological sections of the brain showed the presence large multiple monolocular vacuoles in the wall of the fornix of the third ventricle.

EPA believes that there is sufficient evidence for listing fluorouracil on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the toxicity of this substance to bone marrow, and on the developmental and chronic neurotoxicity data for this chemical.

128. *Fluvalinate (N-[2-chloro-4-(trifluoromethyl)phenyl]DL-valine(+)-cyano (3-phenoxyphenyl)methyl ester)* (CAS No. 069409-94-5) (FIFRA AI) (Ref. 3). Delayed ossification and decreased weight and length of fetuses were observed in offspring of rats orally administered 50 mg/kg/day (LOEL) on days 6 to 15 of gestation. The NOEL was 10 mg/kg/day. These effects were observed at doses that produced maternal toxicity. Curved tibia and fibula were observed in the offspring of

rabbits orally administered 125 mg/kg/day (LOEL). The NOEL was 25 mg/kg/day. In a 2–generation reproduction study, a decrease in pup weight and growth were observed in offspring of rats orally administered 5 mg/kg/day (LOEL). The NOEL was 1 mg/kg/day. Significantly decreased weight and survival were observed in offspring of rats orally administered 25 mg/kg/day.

In a range finding study, dietary administration of 50 mg/kg/day for 30 days produced skin lesions in rats. The NOEL was not determined. A 2–year rat feeding study was terminated at 64 weeks due to dermal lesions produced in animals at 15 mg/kg/day. The NOEL was 2 mg/kg/day. Dietary administration of 10 mg/kg/day (LOEL for effect) to mice for 2 years produced scabbing and dermal abrasion. No NOEL for these effects was established. An increase in plantar ulcers was observed in rats fed 2.5 mg/kg/day (LOEL) for 2 years. The NOEL was 1 mg/kg/day. Decreases in body weight gain were also observed in this study. Based on the NOEL of the study, an oral RfD of 0.01 mg/kg/day was derived. In a 2–generation rat reproduction study, dietary administration of 5 mg/kg/day produced decreased body weight gain and skin lesions in parents and offspring.

Dietary administration of 2.5 mg/kg/day to rats for 13 weeks produced anemia in blood parameters (decreased hematocrit, hemaglobin, and red blood cells). The NOEL was 1.0 mg/kg/day. Dietary administration of 30 mg/kg/day (LOEL) to rats for 3 months produced decreased hemoglobin, hematocrit, and red blood cell count in rats. The NOEL was 3 mg/kg/day.

EPA believes that there is sufficient evidence for listing fluvinate on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available developmental, dermal, and hematological toxicity data for this chemical.

Aquatic acute toxicity values for fluvalinate include a daphnid 48–hour EC_{50} of 0.40 ppb, a bluegill sunfish 96–hour LC_{50} of 0.9 ppb, a rainbow trout 96–hour LC_{50} of 2.9 ppb, and a sheepshead minnow 96–hour LC_{50} of 0.8 ppb. EPA believes that there is sufficient evidence for listing fluvinate on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data for this chemical.

129. *Folpet* (CAS No. 000133-07-3) (CAL) (Ref. 8). Folpet is classified as a Group B2 compound by EPA; i.e., the substance is a probable human carcinogen. Folpet has been shown to induce carcinoma and adenoma of the duodenum in both sexes of CD-1 and B6C3F1 mice. EPA believes that there is sufficient evidence for listing folpet on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for this chemical.

Aquatic acute toxicity test data for folpet include a measured 96–hour LC_{50} of 39 ppb for rainbow trout, and a measured 96–hour LC_{50} of 72 ppb (0.072 ppm) for bluegill. EPA believes that there is sufficient evidence for listing folpet on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the environmental toxicity data for this chemical.

130. *Fomesafen (5-(2-chloro-4-(trifluoromethyl)phenoxy)-N methylsulfonyl)-2-nitrobenzamide)* (CAS No. 072178-02-0) (FIFRA AI) (Ref. 3). Decreased plasma cholesterol and triglycerides and increased liver weights (reversible at 7 days post-treatment) were observed at 50 mg/kg/day (only dose tested) when administered in the diet of rats for 4 weeks. In a 90–day rat study, dietary administration of 5 mg/kg/day (LOEL) produced alterations in lipid metabolism and increases in liver weight. The NOEL was 0.25 mg/kg/day. In a 26-week dog study, dietary administration of 25 mg/kg/day (LOEL) produced alterations in lipid metabolism and liver changes (changes not defined). The NOEL was 1 mg/kg/day. Liver toxicity (increased liver masses, discolored hepatocytes, and pigmented Kupffer cells) was observed in a 2–year rat feeding study at 50 mg/kg/day (LOEL). The NOEL was 5 mg/kg/day. Metabolism studies have shown that fomesafen accumulates in the liver. EPA believes that there is sufficient evidence for listing fomesafen on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hepatic toxicity data for this chemical.

131. *alpha-Hexachlorocyclohexane* (CAS No. 000319-84-6) (CERCLA; CWA PPL; FIFRA SR) (Ref. 8). alpha-Hexachlorocyclohexane is classified by EPA as a Group B2 compound; i.e., the substance is a probable human carcinogen. Although human data are limited, there is a case report of acute leukemia in a Japanese sanitation employee following occupational exposure to alpha-hexachlorocyclohexane and DDT. alpha-Hexachlorocyclohexane has been shown in dietary studies to cause an increase in the incidence of liver tumors in five mouse strains and in Wistar rats. EPA believes that there is sufficient evidence for listing alpha-hexachlorocyclohexane on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the carcinogenicity data for this chemical.

Measured aquatic acute toxicity test data for alpha-hexachlorocyclohexane include a 48–hour EC_{50} of 800 ppb for daphnids. This chemical is expected to bioaccumulate in aquatic systems because the measured bioconcentration factor (BCF) for rainbow trout is 1950. EPA believes that there is sufficient evidence for listing alpha-hexachlorocyclohexane on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the environmental toxicity data for this chemical and its potential for bioaccumulation.

132. *Hexamethylene-1,6-diisocyanate or Diisocyanates category* (CAS No. 000822-60-0) (CAA HAP) (Ref. 7). Hexamethylene-1,6-diisocyanate (HDI) is extremely toxic via the inhalation route. The rat LC_{50} for HDI ranges from 56 (385 mg/m^3) to 45 ppm (310 mg/m^3). The mouse LC_{50} for HDI is 4 ppm (30 mg/m^3). HDI also induces irritation of the upper respiratory tract in mice after acute exposure. The mouse LOAEL was 0.062 ppm (0.43 mg/m^3) for a 3–hour exposure. A NOAEL was not established. Acute exposures to HDI vapors may induce pulmonary irritation in the rat at 60 mg/m^3, but data were insufficient to generate a LOAEL or NOAEL for this effect.

Although the data are insufficient to evaluate the potential for HDI to produce pulmonary hypersensitivity, indirect evidence suggests that inhalation of monomeric HDI may cause pulmonary sensitivity. In addition, data are insufficient to evaluate the potential for HDI to elicit an allergic reaction in previously sensitized animals or people; however, indirect evidence suggests that inhalation of monomeric HDI may elicit allergic responses (i.e., asthma, alveolitis) in isocyanate-sensitized individuals.

EPA's exposure analysis indicates that HDI concentrations are likely to exist beyond facility site boundaries, as a result of continuous, or frequently recurring releases, at levels that can reasonably be anticipated to cause significant adverse acute human health effects. EPA believes that there is sufficient evidence for listing hexamethylene-1,6-diisocyanate on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(A) based on the available acute toxicity and exposure data for this chemical.

EPA is proposing to list HDI as an individual chemical on EPCRA section 313. In addition, in Units IV.B.144. and 158. of this preamble, EPA is proposing to individually list isophorone diisocyanate and 1,1-methylene bis(4-isocyanatocyclohexane) on EPCRA

section 313. As an alternative proposal to the individual listing of HDI, isophorone diisocyanate, and 1,1-methylene bis(4-isocyanatocyclohexane), EPA is proposing to create a diisocyanates category that includes HDI, isophorone diisocyanate, 1,1-methylene bis(4-isocyanatocyclohexane), and 16 other diisocyanates.

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. When filing reports for chemical categories, the releases are determined in the same manner as the thresholds. One report is filed for the category and all releases are reported on this form.

The chemicals selected for this proposed category are members of the diisocyanates category under review by EPA's Office of Pollution Prevention and Toxics. This category has been defined as monomeric diisocyanates of molecular weight less than or equal to 300, plus polymeric diphenylmethane diisocyanate (which is only 40 to 60 percent polymerized). Chemicals were included in this category based on similar chronic and acute adverse respiratory effects. The following chemicals are the proposed members of the EPCRA section 313 diisocyanates category:

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)

Diethyldiisocyanatobenzene (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. 004098-71-9)

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)

These diisocyanates represent a category of chemicals that may effect many organ systems. However, the primary toxicity target for diisocyanates is the upper and lower respiratory tract resulting in chronic pulmonary irritation. Diisocyanates are also known respiratory and dermal sensitizing agents. Both acute and chronic effects may result from acute or chronic exposures. These effects may be immune- or non-immune mediated. EPA believes that diisocyanates should be listed as a category because it is the isocyanate functionality that is responsible for the observed chronic pulmonary irritation associated with exposures to members of this category. The other part of the molecule does not mitigate to any large degree the observed toxic effects. EPA believes that there is sufficient evidence for listing diisocyanates as a category on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available toxicity data for members of the category.

Currently there are four other diisocyanates listed on EPCRA section 313, these are:

Toluene-2,4-diisocyanate (CAS No. 000584-84-9)

Toluene-2,6-diisocyanate (CAS No. 000091-08-7)

Toluenediisocyanate (mixed isomers) (CAS No. 026471-62-5)

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

EPA intends to maintain the individual listings for the three toluene diisocyanate compounds. In addition to the effects discussed above, these compounds have been classified as probable carcinogens. EPA intends to continue to individually list diisocyanates that are possible of probable carcinogens. Methylenebis(phenylisocyanate) has not been shown to be a carcinogen and EPA is proposing to remove it as an individually listed chemical, and add it to the diisocyanates category if the alternative proposal for creation of the category is finalized.

EPA requests comment on the alternative proposal to create a diisocyanates category and what other diisocyanates should be included in such a category.

133. *n-Hexane* (CAS No. 000110-54-3) (CAA HAP) (Ref. 7). In an epidemiology study, no neurological abnormalities were noted in workers. However, neurophysiological tests showed that the mean motor nerve conduction velocities of the exposed group was significantly decreased over the values for the control group. Also, the residual latency of motor nerve conduction of the posterior tibial nerve in the exposed group was significantly slowed when compared with the nonexposed group. A LOAEL of 204 mg/m^3 (58 ppm, LOAEL(ADJ) of 73 mg/m^3) was established for these electrophysiological alterations in humans. The alterations observed are consistent with n-hexane-induced peripheral neuropathy observed in other studies in humans and in animals. EPA believes that there is sufficient evidence for listing n-hexane on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based upon the available neurotoxicity data for this chemical.

134. *Hexazinone* (CAS No. 051235-04-2) (FIFRA AI) (Ref. 3). In a 2-year mouse feeding study, liver hypertrophy, hyperplastic nodules and focal necrosis were observed at 375 mg/kg/day (LOEL). The NOEL was 30 mg/kg/day. In a 90-day feeding study in dogs, decreased body weight, increased alkaline phosphatase activity, decreased albumin/globulin ratio and increased absolute and relative liver weights were noted in both sexes at 5,000 ppm (125 mg/kg/day; LOEL). The NOEL was 1,000 ppm (25 mg/kg/day). EPA believes that there is sufficient evidence for listing hexazinone on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available toxicity data for this chemical.

Measured aquatic acute toxicity test data for hexazinone include an EC_{50} of 7 ppb for *S. capricornutum*. EPA believes that there is sufficient evidence for listing hexazinone on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data for this chemical.

135. *Hydramethylnon (tetrahydro-5,5 dimethyl-2(1H)-pyrimidinone[3-[4-(trifluoromethyl)phenyl]-1-[2-[4(trifluoromethyl) phenyl]ethenyl]-2-propenylidene]hydrazone)* (CAS No. 067485-29-4) (FIFRA AI) (Ref. 3). In a 90-day dog feeding study, testicular atrophy was observed at 6 mg/kg/day (LOEL). The NOEL was 3 mg/kg/day. In a 90-day rat study, dietary administration of 5 mg/kg/day (LOEL) produced testicular atrophy. The NOEL was 2.5 mg/kg/day. Dietary

administration of 6.5 mg/kg/day for 18 months produced testicular lesions in mice. The NOEL was 2.75 mg/kg/day. In a 2–year rat study, dietary administration of 5 mg/kg/day produced decreased testicular weight and testicular atrophy. The NOEL was 2.5 mg/kg/day. In a 3-generation rat reproduction study, oral administration of 5 mg/kg/day produced male infertility. The NOEL was 2.5 mg/kg/day.

Decreased fetal weight was observed in the offspring of rats administered 30 mg/kg/day (LOEL). The NOEL was 10 mg/kg/day. Increased post implantation loss and decreased fetal viability were observed in the offspring of rabbits administered 15 mg/kg/day (LOEL). The NOEL was 5 mg/kg/day. Vertebral anomalies were seen in the offspring of rabbits administered 10 mg/kg/day (LOEL). The NOEL was 5 mg/kg/day.

Dietary administration of 1 mg/kg/day (LOEL) for 6 months to dogs produced increased absolute and relative liver weights. The NOEL was 0.33 mg/kg/day. Based on the NOEL of the study, an oral RfD of 0.0003 mg/kg/day was derived.

EPA believes that there is sufficient evidence for listing hydramethylnon on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available reproductive, developmental, and hepatic toxicity data for this chemical.

The 96–hour LC_{50} in the Chanel Catfish was 90 ppb. Bioaccumulation factors in bluegill sunfish are 1300 for the whole fish, 780 for the fillet, and 1900 for viscera. EPA believes that there is sufficient evidence for listing hydramethylon on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data and the potential for bioaccumulation.

136. *Hydrochlorofluorocarbons* (CAA OD) (Ref. 8). Hydrochlorofluorocarbons are known to release chlorine radicals into the stratosphere. Chlorine radicals act as catalysts to reduce the net amount of stratospheric ozone.

Stratospheric ozone shields the earth from ultraviolet-B (UV-B) radiation (i.e., 290 to 320 nanometers). Decreases in total column ozone will increase the percentage of UV-B radiation, especially at its most harmful wavelengths, reaching the earth's surface.

Exposure to UV-B radiation has been implicated by laboratory and epidemiologic studies as a cause of two types of nonmelanoma skin cancers: squamous cell cancer and basal cell cancer. Studies predict that for every 1 percent increase in UV-B radiation, nonmelanoma skin cancer cases would increase by about 1 to 3 percent.

Recent epidemiological studies, including large case control studies, suggest that UV-B radiation plays an important role in causing malignant melanoma skin cancer. Recent studies predict that for each 1 percent change in UV-B intensity, the incidence of melanoma could increase from 0.5 to 1 percent.

Studies have demonstrated that UV-B radiation can suppress the immune response system in animals, and, possibly, in humans. Increases in exposure to UV-B radiation are likely to increase the incidence of cataracts and could adversely affect the retina.

Aquatic organisms, particularly phytoplankton, zooplankton, and the larvae of many fishes, appear to be susceptible to harm from increased exposure to UV-B radiation because they spend at least part of their time at or near the surface of waters they inhabit.

Increased UV-B penetration has been shown to result in adverse impacts on plants. Field studies on soybeans suggest that yield reductions could occur in some cultivars of soybeans, while evidence from laboratory studies suggest that two out of three cultivars are sensitive to UV-B.

Because this increased UV-B radiation can be reasonably anticipated to lead to cancer and other chronic human health effects and significant adverse environmental effects, EPA believes there is sufficient evidence for listing the following HCFCs that are commercially viable on EPCRA section 313 pursuant to EPCRA sections 313(d)(2)(B) and (C). EPA is proposing that the following HCFCs be added individually to EPCRA section 313:

Dichloropentafluoropropane (CAS No. 127564-92-5)

1,3-Dichloro-1,1,2,3,3-pentafluoropropane (HCFC-225ea) (CAS No. 136013-79-1)

2,2-Dichloro-1,1,1,3,3-pentafluoropropane (HCFC-225aa) (CAS No. 128903-21-9)

1,1-Dichloro-1,2,3,3,3-pentafluoropropane (HCFC-225eb) (CAS No. 111512-56-2)

1,1-Dichloro-1,2,2,3,3-pentafluoropropane (HCFC-225cc) (CAS No. 13474-88-9)

1,3-Dichloro-1,1,2,2,3-pentafluoropropane (HCFC-225cb) (CAS No. 000507-55-1)

1,2-Dichloro-1,1,3,3,3-pentafluoropropane (HCFC-225da) (CAS No. 000431-86-7)

3,3-Dichloro-1,1,1,2,2-pentafluoropropane (HCFC-225ca) (CAS No. 000422-56-0)

2,3-Dichloro-1,1,1,2,3-pentafluoropropane (HCFC-225ba) (CAS No. 000422-48-0)

1,2-Dichloro-1,1,2,3,3-pentafluoropropane (HCFC-225bb) (CAS No. 000422-44-6)

Dichlorofluoromethane (HCFC-21) (CAS No. 000075-43-4)

1,1,1,2-Tetrachloro-2-fluoroethane (HCFC-121a) (CAS No. 000354-11-0)

1,1,2,2-Tetrachloro-1-fluoroethane (HCFC-121) (CAS No. 000354-14-3)

1,2-Dichloro-1,1-difluoroethane (HCFC-132b) (CAS No. 001649-08-7)

2-Chloro-1,1,1-trifluoroethane (HCFC-133a) (CAS No. 000075-88-7)

3-Chloro-1,1,1-trifluoropropane (HCFC-253fb) (CAS No. 000460-35-5).

137. *Imazalil (1-[2-(2,4-dichlorophenyl)-2-(2-propenyloxy)ethyl]-1H-imidazole)* (CAS No. 035554-44-0) (FIFRA AI) (Ref. 3). In a rat teratology study, increased maternal mortality, decreased litter size, and increased number of dead fetuses were observed in animals administered 40 mg/kg/day (LOEL). The NOEL was 10 mg/kg/day. Stillbirths and altered live birth index were observed in rats orally administered 80 mg/kg/day days 16 through 22 of gestation and 21 days post gestation. Altered lactation index was observed in rats orally administered 20 mg/kg/day on days 16 through 22 of gestation and 21 days post gestation. Post-implantation loss was observed in rabbits orally administered 0.63 mg/kg/day on days 6 through 18 of gestation. Altered viability index was observed in rabbits orally administered 2.5 mg/kg/day on days 6 through 18 of gestation. EPA believes that there is sufficient evidence for listing imazalil on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available developmental toxicity data for this chemical.

138. *3-Iodo-2-propynyl butylcarbamate* (CAS No. 055406-53-6) (FIFRA AI) (Ref. 3). In a 90–day rat study, oral administration of 50 mg/kg/day (LOEL) produced increased liver-to-body-weight ratios. The NOEL was 20 mg/kg/day. In a 2–year rat study, dietary administration of 40 and 80 mg/kg/day produced significant non-neoplastic pathological changes in the stomach. No NOEL was established; the LOEL was 20 mg/kg/day. Based on this study, EPA derived an oral RfD of 0.07 mg/kg/day. EPA believes that there is sufficient evidence for listing 3-iodo-2-propynyl butylcarbamate on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available chronic toxicity data for this chemical.

139. *Iprodione (3-(3,5-dichlorophenyl)-N-(1-methylethyl)-2,4-dioxo-1-imidazolidinecarboxamide)*

(CAS No. 036734-19-7) (FIFRA AI) (Ref. 3). Increased red blood cell Heinz bodies and decreased prostate weight (the LOEL was 15 mg/kg/day; the NOEL was 4.2 mg/kg/day) were observed in dogs fed iprodione for 1–year. Increased Heinz bodies were also seen in females at 15 mg/kg/day. At 90 mg/kg/day, increased liver weight was noted in male and female dogs. Based on the NOEL, an oral RfD of 0.04 mg/kg/day was derived. In another 1–year feeding study in dogs, decreased red blood cell counts and hemoglobin and hematocrit levels (the LOEL was 600 ppm or 15 mg/kg/day; the NOEL was 100 ppm or 2.5 mg/kg/day) were observed. At 3,600 ppm (90 mg/kg/day), increased absolute and relative liver weights and increase liver alkaline phosphatase, serum glutamic-pyruvic transaminase, serum glutamic-oxaloacetic transaminase, and lactate dehydrogenase activities were noted. Decreased red blood cell count and decreased hemoglobin and hematocrit levels (the LOEL was 24.6 mg/kg/day in males, 26.4 mg/kg/day in females; the NOEL was 17.5 mg/kg/day in males, 18.4 mg/kg/day in females). EPA believes that there is sufficient evidence for listing iprodione on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hematological and hepatic toxicity data for this chemical.

Acute aquatic toxicity data include a green algae 120–hour EC_{50} of 21 ppb. EPA believes that there is sufficient evidence for listing iprodione on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data for this chemical.

140. *Iron pentacarbonyl* (CAS No. 013463-40-6) (EPCRA EHS) (Ref. 8). Humans exposed to high concentrations of iron pentacarbonyl immediately experience headache and dizziness. These effects are followed 12 to 36 hours after exposure by symptoms such as fever, cyanosis, cough, and shortness of breath. In humans, iron pentacarbonyl has also been known to cause adverse effects on the respiratory and central nervous system, liver, and kidney. The rat oral LD_{50} is 25 mg/kg and the rat inhalation LC_{50} value is 0.044 mg/L. The 4–hour inhalation LC_{100} in mice is 0.007 mg/L. The rabbit oral LD_{50} is 12 mg/kg. EPA's exposure analysis indicates that iron pentacarbonyl concentrations are likely to exist beyond facility site boundaries, as a result of continuous, or frequently recurring releases, at levels that can reasonably be anticipated to cause significant adverse acute human health effects. EPA believes that there is sufficient evidence for listing iron pentacarbonyl on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(A) based on the available acute toxicity and exposure data for this chemical.

141. *Isodrin* (CAS No. 000465-73-6) (CERCLA; EPCRA EHS; RCRA APP8; RCRA P) (Ref. 8). Measured aquatic acute toxicity data for isodrin include a 24–hour LC_{50} of 12 ppb for bluegills and a 24–hour LC_{50} of 6 ppb for minnows. EPA believes that there is sufficient evidence for listing isodrin on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the environmental toxicity data for this chemical.

142. *Isofenphos (2-[[ethoxyl[(1-methylethyl] amino] phosphinothioyl] oxy] benzoic acid 1-methylethyl ester)* (CAS No. 025311-71-1) (FIFRA AI) (Ref. 3). In a 108–week feeding study in mice, inhibition of brain cholinesterase (the LOEL was 100 ppm or 13 mg/kg/day; the NOEL was 10 ppm or 1.3 mg/kg/day) and plasma cholinesterase (the LOEL was 10 ppm or 1.3 mg/kg/day; the NOEL was 0.13 mg/kg/day) was observed. Inhibition of red blood cell cholinesterase (the LOEL was 10 ppm or 0.5 mg/kg/day; the NOEL was 1 ppm or 0.05 mg/kg/day) was seen in a 2–year feeding study in rats. Other studies (14– and 90–day feeding studies in dogs, 30– and 90–day studies in rats, and a 3–week inhalation study in rats) also demonstrate cholinesterase (plasma, red blood cell or brain) inhibition in rats and dogs. EPA believes that there is sufficient evidence for listing isophenphos on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available neurological toxicity data for this chemical.

Aquatic acute toxicity values for isofenphos include a daphnid 48–hour EC_{50} of 1.6 ppb and a mysid 96–hour EC_{50} of 1.7 ppb. EPA believes that there is sufficient evidence for listing isofenphos on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data.

143. *Isophorone* (CAS No. 000078-59-1) (CAA HAP) (Ref. 7). Isophorone has been shown to cause neurotoxic effects in humans exposed to atmospheric concentrations of 5 to 8 ppm. After being exposed for 1 month, workers complained of fatigue and malaise. Neurotoxicity was also observed in humans following acute exposure. At 40 to 85 ppm, effects included nausea, headache, dizziness, faintness, inebriation, and a feeling of suffocation. Increasing exposure concentrations resulted in increasing severity of symptoms. Irritation and central nervous system (CNS) depression were observed at concentrations of 200 to 400 ppm. EPA believes that there is sufficient evidence for listing isophorone on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available neurotoxicity data for this chemical.

144. *Isophorone diisocyanate* (CAS No. 004098-71-9) (TSCA) (Ref. 8). The 4–hour inhalation LC_{50} value of isophorone diisocyanate in rats is 0.123 mg/L. The rat and mouse 3–hour inhalation RD_{50} (50 percent reduction in respiratory rate) values are 0.0046 mg/L and 0.0019 mg/L, respectively. A 50–year old man developed severe asthma after exposure to an unspecified amount of paint containing isophorone diisocyanate. A 1–hour exposure to an unspecified amount of the compound caused eczema in three out of four workers. In addition, isocyanates as a class are generally severe skin, eye and respiratory irritants. EPA's exposure analysis indicates that isophorone diisocyanate concentrations are likely to exist beyond facility site boundaries, as a result of continuous, or frequently recurring releases, at levels that can reasonably be anticipated to cause significant adverse acute human health effects. EPA believes that there is sufficient evidence for listing isophorone diisocyanate on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(A) based on the available acute toxicity and exposure data for this chemical.

As detailed in Unit IV.B.132. of this preamble, as an alternative proposal to the individual listing of HDI, isophorone diisocyanate, and 1,1-methylene bis(4-isocyanatocyclohexane), EPA is proposing to create a diisocyanates category that includes HDI, isophorone diisocyanate, 1,1-methylene bis(4-isocyanatocyclohexane), and 16 other diisocyanates.

145. *Lactofen (5-(2-chloro-4-(trifluoromethyl)phenoxy)-2-nitro-2-ethoxy-1-methyl-2-oxoethyl ester)* (CAS No. 077501-63-4) (FIFRA AI) (Ref. 3). Lactofen meets the criteria of an EPA Group B2 compound, i.e., a probable human carcinogen. This conclusion was based on an increased incidence of hepatocellular carcinomas in males and combined incidence of hepatocellular adenomas and carcinomas in both sexes of CD-1 mice following dietary administration of lactofen. In CD rats, there was increased incidence of liver neoplastic nodules in both sexes. Four structurally similar chemicals, acifluorfen, nitrofen, oxyfluorfen, and fomesafen, all produced hepatocellular tumors in rodents.

Results of several subchronic and chronic studies indicated the liver and kidney as target organs for lactofen.

Increased absolute and relative liver weight and hepatocytomegaly (the LOEL was 1.5 mg/kg/day; the NOEL was not determined) were observed in male mice fed lactofen for 78 weeks. At 37.5 mg/kg/day, there was also an increased incidence of cataracts and renal pigmentation. Based on the LOEL, an oral RfD of 0.002 mg/kg/day was derived. Renal dysfunction and decreased hemoglobin and hematocrit levels and red blood cell counts (the LOEL was 25/75 mg/kg/day; the NOEL was 5 mg/kg/day) were observed in a 1–year feeding study in dogs. Increased renal and hepatic pigmentation (the LOEL was 50 mg/kg/day; the NOEL was 25 mg/kg/day) were noted in a 2–year feeding study in rats. In a 90–day mouse study, increased alkaline phosphatase, serum glutamate oxaloacetate transaminase (SGOT), and serum gleutanic pyruvic transaminase (SGPT) activities, increased liver weight, hepatic necrosis, biliary hyperplasia, decreased hematocrit and hemoglobin levels and red blood cell counts, extramedullary hematopoiesis, and kidney nephrosis and fibrosis (the LOEL was 26 mg/kg/day; the NOEL was not determined) were seen. Decreased hemoglobin and hematocrit levels, decreased red blood cell counts, and brown pigment in the kidney and liver (the LOEL was 50 mg/kg/day) were noted in a 90–day feeding study in rats.

EPA believes that there is sufficient evidence for listing lactofen on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data and hepatic, renal, and hematological toxicity data for this chemical.

146. *Linuron* (CAS No. 000330-55-2) (FIFRA SR) (Ref. 8). The appearance of sulfhemoglobin in the blood of dogs, rats, or mice exposed to linuron has been reported. In fact, available animal data from feeding studies of various durations (30 days to 2 years) with linuron as well as from studies with structurally similar urea-based herbicides indicate that the presence of sulfhemoglobin (abnormal blood pigment) and morphological changes in red blood cells provide the most sensitive indicator of exposure to linuron. In a 2–year feeding study with beagle dogs, the LOAEL, based on the presence of the sulfhemoglobin, was 0.625 mg/kg/day. This was the lowest dose tested. Red blood cell counts were decreased in dogs exposed to higher doses of linuron. EPA has derived an oral RfD of 0.002 mg/kg/day for linuron from this study. Similar findings were reported in two separate 2–year rat feeding studies. In one of these studies, the LOAEL was 31.25 mg/kg/day and the NOAEL was 6.25 mg/kg/day. These values were based on spleen and bone marrow changes indicative of hemolysis, and an increase in mortality and growth retardation. In the other 2–year rat study, a LOAEL of 2.5 mg/kg/day (the lowest dose tested) was based on decreased red blood cell counts and reticulocytosis. Elevated sulfhemoglobin levels were reported in rats exposed for as little as 30 days to 150 mg/kg/day. This exposure level also caused severe growth retardation and increased mortality. The LOAEL for decreased body weight gain was 15 mg/kg/day and the NOAEL was 3 mg/kg/day. Chronic administration of linuron at 4 mg/kg/day to rats caused hypochromic anemia, decreased cholinesterase and peroxidase activities in the blood.

A LOAEL of 31.25 mg/kg/day was established in a 3–generation reproductive toxicity study in which linuron (in the diet) caused reduced weanling weights, reduced liver and kidney weights, liver atrophy, and reduced pup survival. In a separate developmental toxicity study in rats administered linuron orally, a LOAEL of 31.25 mg/kg/day was based on an increased incidence of fetal resorptions. The LOAEL for maternal toxicity in this study was 6.25 mg/kg/day (NOAEL 2.50 mg/kg/day), and was based on decreased food consumption and decreased body weight gain. An oral teratology study in rabbits indicated a LOAEL of 5 mg/kg/day (lowest dose tested) based on decreased fetal body weight, decreased litter size and an increase in skull malformations.

EPA believes that there is sufficient evidence for listing linuron on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the hematological and developmental toxicity data for this chemical.

147. *Lithium carbonate* (CAS No. 000554-13-2) (CAL) (Ref. 8). A major use of lithium carbonate is in the treatment of manic episodes of manic-depressive illness. Decreases in the number of implantations, number of live fetuses and fetal body weight, and increases in resorptions and various limb/skeletal anomalies were reported in the offspring of Wistar rats that received 100 mg/kg (the fetotoxic LOEL; the fetotoxic NOEL was 50 mg/kg) during gestation days 6 through 15. Offspring of mice that received 465 mg/kg/day during gestation days 6 through 15 had increased craniofacial abnormalities. Fetal death and reductions in litter size were also noted. EPA believes that there is sufficient evidence for listing lithium carbonate on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available developmental toxicity data for this chemical.

148. *Malathion* (CAS No. 000121-75-5) (CERCLA) (Ref. 8). Malathion is a phosphorothioate insecticide. Its insecticidal properties are due to cholinesterase inhibition. A 42–year old woman ingested a minimum of 120 ml of a 50 percent solution (approximately 850 mg/kg). She quickly became comatose, cyanotic, flaccid, devoid of tendon reflexes, and miotic. Her serum cholinesterase activity was 22 percent of normal for 9 days and her red blood cell cholinesterase activity was 10 to 25 percent of normal for 45 days. Thirty-five cases of poisoning by ingestion were reported in India. The symptoms observed were cyanosis, excess salivation, pinpoint pupils, pulmonary edema, and electrocardiographic abnormalities; all of which are indicative of cholinesterase inhibition. Autopsy of the fatalities indicated damage to the myocardium. In a 56–day study in which men were orally administered malathion, the NOEL for neurotoxic effects was 0.23 mg/kg/day and the LOEL was 0.34 mg/kg/day. Plasma and red blood cell cholinesterase inhibition was observed at 0.34 mg/kg/day; however, no clinical signs of overt toxicity were noted at this dose. Based on the NOEL, EPA has derived an oral RfD of 0.02 mg/kg/day for this chemical. Cholinesterase inhibition symptoms have also been observed in experimental animals exposed to malathion. EPA believes that there is sufficient evidence for listing malathion on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the chronic neurotoxicity data for this chemical.

Measured aquatic acute toxicity data for malathion include a 96–hour LC_{50} of 68 ppb for rainbow trout, a 96–hour LC_{50} of 51 ppb for sheepshead minnow, and a 96–hour LC_{50} of 76 ppb for lake trout. In addition, the measured 48–hour EC_{50} for daphnids is 0.9 ppb. EPA believes that there is sufficient evidence for listing malathion on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the environmental toxicity data for this chemical.

149. *Man-made mineral fibers category* (CAA HAP) (Ref. 7). Man-made mineral fibers are synthetic, amorphous (noncrystalline) fibers which consist of three major groups: Glass fibers; mineral wool fibers (which includes mainly rock wool and slag wool); and refractory ceramic fibers. Health concerns for these fibers are based on the morphological and toxicologic similarities with asbestos, a known human carcinogen, causing lung cancer and mesotheliomas in humans and non-malignant

respiratory diseases (e.g. lung fibrosis). Injection studies, in which glass wool and glass microfibers were directly placed into the respiratory airways, the pleural or abdominal cavities of laboratory animals, have shown consistent evidence of carcinogenesis. Experimental studies have shown evidence of carcinogenesis by injection of rock wool and slag wool. IARC has classified glass wool, rock wool, and slag wool fibers as Group 2B compounds, i.e., possible human carcinogens. EPA has classified refractory ceramic fibers as Group B2 compounds, i.e., probable human carcinogen. EPA believes that there is sufficient evidence for listing man-made mineral fibers as a category on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for these fibers.

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. When filing reports for chemical categories, the releases are determined in the same manner as the thresholds. One report is filed for the category and all releases are reported on this form.

EPA considered a number of options for listing man-made mineral fibers on EPCRA section 313. In 1977, the National Institute for Occupational Safety and Health (NIOSH) recommended that exposures to fibers be limited to 3 fibers per cubic centimeters (f/cc) for fibers that are less than 3.5 micrometers in diameter and longer than 10 micrometers in length. NIOSH has since commented that in order to protect workers from lung cancer it will be necessary to lower the exposure to 0.2 f/cc for fibrous glass. In 1992, the Occupational Safety and Health Administration (OSHA) proposed a 1 f/cc 8–hour time-weighted average (TWA) limit for respirable fibers of fibrous glass, including refractory ceramic fibers. Respirable fibers are generally defined as fibers with a diameter of less than 3.5 micrometers whose length is at least 3 times the diameter (i.e., an aspect ratio (fiber length divided by fiber diameter) of 3 or greater). In order to ease the burden of reporting, EPA considered listing fibers based on an aspect ratio that simply discriminates between particles and fibers. This, however, seemed to be overly inclusive in that it would cover nonrespirable as well as respirable fibers. EPA also considered using a diameter criteria without an aspect ratio but this option also appears to be too inclusive since it may include particles as well as fibers. EPA is proposing to list man-made mineral fibers as a category that includes glass microfibers, glass wool fibers, rock wool fibers, slag wool fibers, and refractory ceramic fibers that have a diameter less than 3.5 micrometers and an aspect ratio greater than 3. This definition is consistent with both the NIOSH and OSHA recommendations and is limited to fibers that are respirable. EPA requests comment on this definition of man-made mineral fibers and any other options for defining a fibers category.

150. *Mecoprop* (CAS No. 000093-65-2) (IARC) (Ref. 8). Mecoprop is a mono-chloro, mono-methylphenoxy isopropanoic acid type herbicide. IARC has assigned mecoprop to Group 2B, i.e., it is possibly carcinogenic to humans.

In several animal studies, changes in liver or kidney weights were the most sensitive indicators of mecoprop toxicity. In a 90–day rat feeding study, the LOAEL was 9 mg/kg/day and the NOAEL was 3 mg/kg/day. At 26 mg/kg/day, the changes in organ weights were accompanied by decreased glucose levels in males and increased creatinine levels in females. EPA has derived an oral RfD of 0.001 mg/kg/day from this study.

EPA believes that there is sufficient evidence for listing mecoprop on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the hepatic and renal toxicity data for this chemical.

151. *2-Mercaptobenzothiazole (MBT)* (CAS No. 000149-30-4) (TSCA) (Ref. 8). The 21–day maximum acceptable toxicant concentration (MATC) for daphnids range from 240 to 470 ppb. The 60-day MATC for rainbow trout range from 41 to 78 ppb. EPA's exposure analysis indicates that releases of 2-mercaptobenzothiazole will result in concentration levels that can reasonably be anticipated to cause significant adverse environmental effects. EPA believes that there is sufficient evidence for listing 2-mercaptobenzothiazole on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data and exposure data for this chemical.

152. *Merphos* (CAS No. 000150-50-5) (FIFRA SR) (Ref. 8). Merphos is a thiophosphate-type cholinesterase inhibitor. Delayed neurotoxic effects have been reported in a 28-year old man following accidental exposure to the chemical over a period of 3 days. Fourteen days later, he developed complete facial diplegia and decreased conduction velocity in his nerve fibers. He recovered completely. Both immediate and delayed neurotoxic effects following exposure to merphos have been reported in experimental animals. In a 3–month hen feeding study the NOEL for neurotoxic effects was 0.1 mg/kg/day and the LOEL was 0.5 mg/kg/day. At 0.5 mg/kg, hens showed delayed neurotoxicity, ataxia, and equivocal changes in the spinal cord and peripheral nerves. Based on the NOEL, EPA derived an oral RfD of 0.00003 mg/kg/day for this chemical. In a 112-day rat feeding study, females showed red blood cell cholinesterase inhibition at the LOEL of 0.25 mg/kg/day. The NOEL was 0.1 mg/kg/day. In a 90–day rat feeding study, animals showed reduced brain cholinesterase activity at the LOEL of 3.8 mg/kg/day. The NOEL was 1.8 mg/kg/day. In a 90–day dog feeding study, plasma cholinesterase inhibition was observed at the LOEL of 2.5 mg/kg/day. The NOEL was 0.75 mg/kg/day. Fourteen cattle and 20 sheep administered single doses of merphos (25 to 200 mg/kg) or 10 daily doses of merphos (2.5 mg/kg/day) showed emaciation, diarrhea, and depression of blood cholinesterase. Ingested merphos is rapidly metabolized to n-butyl mercaptan within the gastrointestinal tract. n-Butyl mercaptan has been shown to be responsible for the acute neurotoxic effects of merphos. Thus, oral exposure to merphos is expected to cause acute neurotoxic symptoms while dermal exposure to merphos is expected to cause delayed neurotoxic symptoms. EPA believes that there is sufficient evidence for listing merphos on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the chronic neurotoxicity data for this chemical.

153. *Metham sodium (sodium methyldithiocarbamate)* (CAS No. 000137-42-8) (FIFRA AI) (Ref. 3). Postimplanatation loss was observed in rabbits administered metham sodium at 30 mg/kg/day (LOEL) on days 6 to 18 of gestation. The NOEL was 10 mg/kg/day (4.2 mg/kg/day based on active ingredient). In rats fed metham sodium, increased variations, retardations, and anomalies were reported at doses of 10 mg/kg/day (LOEL) administered on days 6 to 15. The NOEL was less than or equal to 10 mg/kg/day (less than or equal to 4.2 mg/kg/day based on active ingredient). Although neither study was considered to be fully adequate due to study design and reporting deficiencies, the weight of evidence indicates that metham sodium induces developmental

toxicity. In addition, metham sodium is metabolized to carbon disulfide, a potent developmental toxicant. EPA believes that there is sufficient evidence for listing metham sodium on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available developmental toxicity data for this chemical and its metabolite, carbon disulfide.

154. *Methazole (2-(3,4-dichlorophenyl)-4-methyl-1,2,4-oxadiazolidine-3,5-dione)* (CAS No. 020354-26-1) (FIFRA AI) (Ref. 3). Rabbits receiving 30 or 60 mg/kg/day by gavage on days 6 to 18 of gestation exhibited increased embryolethality. The NOEL was 10 mg/kg/day. EPA believes that there is sufficient evidence for listing methazole on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available developmental toxicity data for this chemical.

155. *Methiocarb* (CAS No. 002032-65-7) (CERCLA; EPCRA EHS) (Ref. 8). Measured terrestrial acute toxicity data for wildlife include an oral LD_{50} of 4.6 mg/kg for red-winged blackbirds. EPA believes that there is sufficient evidence for listing methiocarb on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the environmental toxicity data for this chemical.

156. *Methoxone ((4-Chloro-2-methylphenoxy) acetic acid) (MCPA)* (CAS No. 000094-74-6) (FIFRA SR; IARC) (Ref. 8). Methoxone is a chlorophenoxy-type herbicide. Animal studies indicate that the kidney and liver are the primary target organs of methoxone toxicity. Beagle dogs fed diets containing methoxone for 1-year developed liver toxicity, which was demonstrated by increased liver weights associated with alterations in serum glutamate-pyruvate transaminase, serum glutamate-oxaloacetate transaminase, bilirubin, triglyceride and cholesterol levels. These effects occurred at doses of 0.75 mg/kg/day (LOAEL) and higher. The NOAEL was 0.15 mg/kg/day. Kidney changes in the treated animals included deposition of kidney pigment in the proximal tubular epithelium (the LOAEL was 0.75 mg/kg/day; the NOAEL was 0.15 mg/kg/day), and was accompanied by alterations in creatinine, urea, and potassium levels. EPA derived an oral RfD of 0.0005 mg/kg/day from this study. Similar changes suggesting liver and kidney toxicity were reported in another 90-day dog feeding study (the LOAEL was 3 mg/kg/day; the NOAEL was 1 mg/kg/day) and in rats in a 90-day feeding study (the LOAEL was 7.5 mg/kg/day; the NOAEL was 2.5 mg/kg/day).

EPA believes that there is sufficient evidence for listing methoxone on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hepatic and renal toxicity data for this chemical.

157. *Methoxone sodium salt ((4-chloro-2-methylphenoxy) acetate sodium salt)* (CAS No. 003653-48-3) (FIFRA SR; IARC) (Ref. 8). Methoxone sodium salt is a chlorophenoxy-type herbicide. Animal studies indicate that the kidney and liver are the primary target organs of methoxone toxicity. Beagle dogs fed diets containing methoxone for 1-year developed liver toxicity, which was demonstrated by increased liver weights associated with alterations in serum glutamate-pyruvate transaminase, serum glutamate-oxaloacetate transaminase, bilirubin, triglyceride and cholesterol levels. These effects occurred at doses of 0.75 mg/kg/day (LOAEL) and higher. The NOAEL was 0.15 mg/kg/day. Kidney changes in the treated animals included deposition of kidney pigment in the proximal tubular epithelium (the LOAEL was 0.75 mg/kg/day; the NOAEL was 0.15 mg/kg/day), and was accompanied by alterations in creatinine, urea, and potassium levels. EPA derived an oral RfD of 0.0005 mg/kg/day from this study. Similar changes suggesting liver and kidney toxicity were reported in another 90-day dog feeding study (the LOAEL was 3 mg/kg/day; the NOAEL was 1 mg/kg/day) and in rats in a 90-day feeding study (the LOAEL was 7.5 mg/kg/day; the NOAEL was 2.5 mg/kg/day).

EPA believes that there is sufficient evidence for listing methoxone sodium salt on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on its potential to cause cancer and on the available hepatic and renal toxicity data for this chemical.

158. *1,1-Methylene bis(4-isocyanatocyclohexane)* (CAS No. 005124-30-1) (TSCA) (Ref. 8). The 5-hour rat inhalation LC_{50} value for 1,1-methylenebis(4-isocyanatocyclohexane) is 0.21 mg/L. The 3-hour mouse inhalation RD_{50} (50 percent reduction in respiratory rate) value is 0.027 mg/L. In addition, isocyanates as a class are generally severe skin, eye, and respiratory irritants. EPA's exposure analysis indicates that 1,1-methylenebis(4-isocyanatocyclohexane) concentrations are likely to exist beyond facility site boundaries, as a result of continuous, or frequently recurring releases, at levels that can reasonably be anticipated to cause significant adverse acute human health effects. EPA believes that there is sufficient evidence for listing 1,1-methylenebis(4-isocyanatocyclohexane) on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(A) based on the available acute toxicity and exposure data for this chemical.

As detailed in Unit IV.B.132. of this preamble, as an alternative proposal to the individual listing of HDI, isophorone diisocyanate, and 1,1-methylene bis(4-isocyanatocyclohexane), EPA is proposing to create a diisocyanates category that includes HDI, isophorone diisocyanate, 1,1-methylene bis(4-isocyanatocyclohexane), and 16 other diisocyanates.

159. *Methylene bis(thiocyanate)* (CAS No. 006317-18-6) (FIFRA AI) (Ref. 3). The minimal human lethal dose for methylene bis(thiocyanate) is 15 to 30 g (214 to 429 mg/kg), although fatalities have been reported at 300 mg (4.3 mg/kg). Clinical effects may include decreased blood pressure, apnea, cerebral excitation, convulsions, coma, vomiting, diarrhea, abdominal cramping, albuminuria, skin rashes, exfoliative dermatitis, muscle weakness, goiter, and toxic psychosis. The intravenous mouse LD_{50} is 3.6 mg/kg. The subcutaneous rabbit DLo is 20 mg/kg; convulsions and lowered blood pressure were observed in this study. EPA believes that there is sufficient evidence for listing methylene bis(thiocyanate) on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available neurological toxicity data for this chemical.

160. *Methyl isothiocyanate* (CAS No. 00556-61-6) (FIFRA AI) (Ref. 3). Aquatic acute toxicity values for methyl isothiocyanate include a fish 96-hour LC_{50} of 94 ppb, a 96-hour LC_{50} of 130 ppb for bluegills, and a daphnid 48-hour LC_{50} of 55 ppb. EPA believes that there is sufficient evidence for listing methyl isothiocyanate on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data.

161. *2-Methyllactonitrile* (CAS No. 000075-86-5) (CERCLA; EPCRA EHS; RCRA APP8; RCRA P) (Ref. 8). 2-Methyllactonitrile belongs to a class of substances known as the cyanohydrins. Cyanohydrins are generally quite toxic because they can release hydrogen cyanide. An oral dose of 5 mg/rat (approximately 14 mg/kg) of 2-methyllactonitrile administered twice weekly for 3 to 8 months produced liver and kidney lesions. Inhalation of 10.2 mg/L twice weekly for 3 to 8 months (duration of each individual exposure not reported) produced kidney lesions, desquamation of the bronchial epithelium, and bronchial ulcerations. EPA believes that there is sufficient

evidence for listing 2-methyllactonitrile on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the chronic toxic effects to the liver, kidney, and bronchi caused by this chemical.

162. *N-Methylolacrylamide* (CAS No. 000924-42-5) (CAL) (Ref. 8). There was clear evidence of carcinogenicity from N-methylolacrylamide in a 2–year study using B6C3F1 mice administered the substance by oral gavage. In both sexes, there were increased incidences of Harderian gland adenomas or carcinomas, hepatocellular adenomas or carcinomas, and alveolar or bronchiolar adenomas and carcinomas. There was also an increase in ovarian granulosa cell tumors. EPA believes that there is sufficient evidence for listing N-methylolacrylamide on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the carcinogenicity data for this chemical.

163. *Methyl parathion* (CAS No. 000298-00-0) (CERCLA; FIFRA SR; RCRA APP8; RCRA P) (Ref. 8). Methyl parathion is a thiophosphate-type cholinesterase inhibitor. Methyl parathion is highly toxic when administered to experimental animals at low doses. The rat and mouse oral LD_{50} values are reported to be 6.01 mg/kg and 18 mg/kg, respectively. The rat and mouse 4-hour inhalation LC_{50} values are reported to be 0.034 mg/L and 0.12 mg/L, respectively, at which symptoms of cholinesterase inhibition were observed.

Human volunteers showed a 37 percent decrease in red blood cell cholinesterase activity following oral administration of 0.43 mg/kg/day of methyl parathion for 10 days. The LOEL was 0.43 mg/kg/day and the NOEL was 0.31 mg/kg/day. In a 90–day dog feeding study, brain, red blood cell, and plasma cholinesterase inhibition was observed at the LOEL of 1.0 mg/kg/day. The NOEL was 0.3 mg/kg/day. In a chronic rat feeding study, plasma and erythrocyte cholinesterase were inhibited throughout the study and brain cholinesterase was depressed at the termination of the study at 2.5 mg/kg/day. The NOEL for systemic toxicity was 0.025 mg/kg/day. An adequate NOEL for neurologic changes was not defined. Overt signs of cholinergic toxicity (tremors, abnormal gait, alopecia) were observed in the animals at a dose of 2.5 mg/kg/day. Histologic examination revealed evidence of peripheral neuropathy in animals administered this dose. EPA has derived an oral RfD of 0.00025 mg/kg/day based on the systemic NOEL for this chemical.

Hepatocellular swelling, degeneration, and fatty change have been observed in humans acutely intoxicated with methyl parathion. Hepatocellular changes were observed in patients that survived for 28 hours to 9 days after intoxication. Methyl parathion was orally administered to rats in increasing doses for 36 days (starting with 0.37 mg/kg/day and increasing by a factor of 1.5 on every 4th day). Weight loss, hyperglycemia, and macrocytic anemia, all secondary to hepatotoxicity, were observed.

EPA believes that there is sufficient evidence for listing methyl parathion on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the chronic neurotoxicity and hepatic toxicity data for this chemical.

Measured aquatic acute toxicity data for methyl parathion include a 48–hour EC_{50} of 0.14 ppb for daphnids and a 96–hour LC_{50} of 15 ppb for crayfish. EPA believes that there is sufficient evidence for listing methyl parathion on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data for this chemical.

164. *N-Methyl-2-pyrrolidone* (CAS No. 000872-50-4) (TSCA) (Ref. 8). In a 2–generation reproductive study, there was evidence of reproductive toxicity in the F_1 generation after exposure to 50 mg/kg/day (LOAEL; no NOAEL was established). Exposure to 50 mg/kg/day or more resulted in significant reductions in the male fertility index and in the female fecundity index. In addition, exposure to 500 mg/kg/day resulted in an increased incidence of dams with decreased corpora lutea. There was also evidence of developmental toxicity in both generations after exposure to 500 mg/kg/day as demonstrated by reduced litter size, reduced postnatal survival, and reduced pup weight.

Maternal toxicity (significant reduction in mean body weight gain) was observed in rabbits receiving 175 mg/kg by gavage on days 6 through 18 of gestation (The NOAEL was 55 mg/kg/day). Exposure to 540 mg/kg/day (LOAEL) resulted in developmental toxicity as demonstrated by a significant increase in resorptions, and malformations (misshapen skull bone and cardiovascular malformations). The NOAEL for developmental toxicity was 175 mg/kg/day.

EPA believes that there is sufficient evidence for listing N-methylpyrrolidone on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available developmental and reproductive toxicity data for this chemical.

165. *Methyltrichlorosilane* (CAS No. 000075-79-6) (EPCRA EHS) (Ref. 8). As a class, chlorinated silanes are very corrosive to the skin and mucous membranes and liberate hydrochloric acid in the presence of water. Methyltrichlorosilane causes severe burns and the vapor is harmful to humans. The 2–hour mouse inhalation LC_{50} value is 0.180 mg/L. EPA's exposure analysis indicates that methyltrichlorosilane concentrations are likely to exist beyond facility site boundaries, as a result of continuous, or frequently recurring releases, at levels that can reasonably be anticipated to cause significant adverse acute human health effects. EPA believes that there is sufficient evidence for listing methyltrichlorosilane on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(A) based on the available acute toxicity and exposure data for this chemical.

166. *Metiram* (CAS No. 009006-42-2) (FIFRA SR) (Ref. 8). Metiram is an ethylene bisdithiocarbamate (EBDC) fungicide. Evidence suggests that ethylene bisthiocarbamate fungicides and ethylenethiourea (a common contaminant, metabolite, and degradation product of these fungicides) cause cancer and adverse developmental effects in experimental animals. In a 2–year diet study, ethylenethiourea caused liver adenomas and carcinomas in mice, and thyroid follicular cell adenomas and carcinomas in mice and rats. A NOAEL of less than or equal to 5 mg/kg has been reported for ethylenethiourea, based on a rat developmental toxicity study. Ethylenethiourea caused delayed ossification or hardening of the parietal bone in pups. EPA believes that there is sufficient evidence for listing metiram on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the carcinogenicity and developmental toxicity data for ethylenethiourea, a metabolite and degradation product of metiram.

In Unit IV.B.172. of this preamble, EPA is proposing to add another ethylene bisdithiocarbamate (EBDC), nabam. An additional two EBDCs, zineb and maneb, are currently individually listed on the EPCRA section 313 list of toxic chemicals. The category of EBDCs has recently been added to EPCRA section 313 (December 1, 1993, 58 FR 63500). EPA requests comment on the following: (1) Should the individual EBDCs, metiram and nabam, be added individually to EPCRA section 313 even though they are members of the EBDC category, which is listed on EPCRA section 313; and (2) should the individual listings for two EBDCs, zineb and maneb, be deleted and added as members of the newly created EBDC category.

167. *Metribuzin* (CAS No. 021087-64-5) (FIFRA AI) (Ref. 3). In a rabbit teratology study, the NOEL for maternal and fetotoxicity was 15 mg/kg/day, and the LOEL was 45 mg/kg/day. Developmental effects including irregular spinus process and decreased pup body weight were observed in rats treated with metribuzin (Sencor) during gestation day 7 to 19 at 85 mg/kg/day (LOEL). The NOEL for developmental toxicity was 30 mg/kg/day. The LOEL and NOEL for maternal toxicity were 30 and 10 mg/kg/day, respectively.

In a 2–year dog feeding study, adverse effects observed at 1,500 ppm (37.5 mg/kg/day; LOEL) included weight reduction, increased mortality, hematologic changes, and liver/kidney damage. The systemic NOEL was 100 ppm (2.5 mg/kg/day). In a 2–year rat feeding study, decreased weight gain, mortality, and pathological changes in the liver and kidney were observed at 300 ppm (15 mg/kg/day). The NOEL was 100 ppm (5 mg/kg/day).

EPA believes that there is sufficient evidence for listing metribuzin on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hepatic, renal, and developmental toxicity data for this chemical.

168. *Mevinphos* (CAS No. 007786-34-7) (CERCLA; EPCRA EHS) (Ref. 8). Measured aquatic acute toxicity values for mevinphos include a 96–hour LC_{50} of 70 ppb for bluegills, and a 96–hour LC_{50} of 0.16 ppb for daphnids. Measured acute avian toxicity data include a pheasant oral LD_{50} of 1.37 mg/kg, a mallard duck oral LD_{50} of 4.63 mg/kg, and a sharp-tailed grouse oral LD_{50} of 1.34 mg/kg. EPA believes that there is sufficient evidence for listing mevinphos on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the environmental toxicity data for this chemical.

169. *Molinate (1H-azepine-1-carbothioic acid, hexahydro-S-ethyl ester)* (CAS No. 002212-67-1) (FIFRA AI) (Ref 3). In a rat developmental toxicity study, adverse effects observed following administration of molinate at 35 mg/kg/day (LOEL) included increased post-implantation loss, lower fetal body weight, increased incidence of runts, and external/soft tissue/skeletal variants; the NOEL was 2.2 mg/kg. In a rabbit developmental study, adverse effects such as an increase in the number of abortions, and a decrease in the number of females with live fetuses were noted at 200 mg/kg/day. The NOEL was 20 mg/kg/day. The developmental effects were observed at levels which were toxic to maternal animals.

In a rat fertility test, reductions in fertility, dose-related altered sperm morphology, and a reduction in the number of viable fetuses were observed following administration of molinate. The NOEL was 0.2 mg/kg/day and the LOEL was 4 mg/kg/day. Based on the NOEL of the study, an oral RfD of 0.002 mg/kg/day was derived. In a 90–day study in male rats, the lowest toxic oral dose of 324 mg/kg produced adverse effects on spermatogenesis, male fertility, and viability index. The 20–day inhalation male rat lowest-toxic contentration (TCLo) is 0.0006 mg/L. At this exposure level adverse effects on spermatogenesis and male fertility index were reported. In a 2-generation rat reproduction study, the reproductive NOEL was 0.3 mg/kg/day, and the LOEL was 2.5 mg/kg/day based on reduced fecundity and increased incidence of ovarian vacuolation/hypertrophy. In a 3–month rat inhalation study, testicular degeneration and abnormal spermatozoa were observed at 0.002 mg/L (LOEL). No NOEL was determined.

In a 2–year study in rats fed molinate, adverse effects seen at 0.35 mg/kg/day included degeneration and demyelination of the sciatic nerve and skeletal muscle atrophy/reserve cell hyperplasia; no NOEL was determined. In a 1–year study in dogs administered molinate orally, adverse effects observed at 50 mg/kg/day included anemia, loss of ability to bark, ataxia, splayed hind limbs, vacuolation of the medulla, demyelination of the pons and spinal cord, tremors, and eosinophilic bodies in the nervous system.

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

170. *Monuron* (CAS No. 000150-68-5) (FIFRA SR) (Ref. 8). The measured aquatic toxicity data for monuron include a 1.5-hour EC_{50} of 90 ppb and a 10-day EC_{50} of 100 ppb for marine algae. EPA believes that there is sufficient evidence for listing monuron on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the environmental toxicity data for this chemical.

171. *Myclobutanil (alpha-butyl-alpha-(4-chlorophenyl)-1H-1,2,4-triazole-1-propanenitrile)* (CAS No. 088671-89-0) (FIFRA AI) (Ref. 3). Hepatocellular hypertrophy (the LOEL was 5.9 mg/kg/day; the NOEL was 0.3 mg/kg/day) was seen in a 90–day feeding study in dogs. In another 90–day feeding study, hepatocellular necrosis and hypertrophy (the LOEL was 147.2 mg/kg/day; the NOEL was 49.1 mg/kg/day) were observed in rats. Hepatocellular hypertrophy (the LOEL was 14.3 mg/kg/day in males and 15.7 mg/kg/day in females; the NOEL was 3.1 mg/kg/day in males and 3.83 mg/kg/day in females) was noted in a 1–year feeding study in dogs. Hepatic effects (centrilobular hepatocytic hypertrophy, kupffer cell pigmentation, periportal vacuolation and altered foci) were observed in mice fed 75 mg/kg/day myclobutanil for 2 years. At 15 mg/kg/day, increased liver mixed function oxidase (the NOEL was 3 mg/kg/day) was also seen.

Testicular atrophy (the LOEL was 9.84 mg/kg/day; the NOEL was 2.49 mg/kg/day) was observed in a 2–year chronic feeding study in rats. The seminiferous tubules were frequently devoid of spermatid formation and germinal epithelial cells. Based on the NOEL, an oral RfD of 0.025 mg/kg/day was derived. Testicular atrophy (the LOEL was 46.4 mg/kg/day; the NOEL was 9.28 mg/kg/day) was also noted in a 2-generation reproduction study.

In a developmental toxicity study in rats, increased resorption and decreased viability were observed at 93.8 mg/kg/day (LOEL). The NOEL was 31.3 mg/kg/day. In a developmental toxicity study in rabbits, an increased number of resorptions per litter, reduced viability index, and reduced litter size were observed at 200 mg/kg/day (LOEL). The NOEL was 60 mg/kg/day.

EPA believes that there is sufficient evidence for listing myclobutanil on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hepatic, reproductive, and developmental toxicity data for this chemical.

172. *Nabam* (CAS No. 000142-59-6) (FIFRA SR) (Ref. 8). Nabam is an ethylene bisthiocarbamate fungicide. Evidence suggests that ethylene bisthiocarbamate fungicides and ethylenethiourea (a common contaminant, metabolite, and degradation product of these fungicides) cause cancer and adverse developmental effects in experimental animals. In a 2–year diet study ethylenethiourea caused liver adenomas and carcinomas in mice, and thyroid follicular cell adenomas and carcinomas in mice and rats. A NOAEL of less than or equal to 5 mg/kg has been reported for ethylenethiourea, based on a rat developmental toxicity study. Ethylenethiourea caused delayed ossification or hardening of the parietal bone in pups. EPA believes that there is sufficient evidence for listing nabam on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the carcinogenicity and developmental

toxicity data for ethylenethiourea, a metabolite and degradation product of nabam.

173. *Naled* (CAS No. 000300-76-5) (CERCLA; FIFRA SR) (Ref. 8). Naled is an organophosphate-type cholinesterase inhibitor. In a human acute poisoning case, toxic symptoms included abdominal cramps, hypersecretion, emesis, perspiration, anxiety, vertigo and horizontal nystagmus, and persisted for 4 months. In a 2–year rat feeding study the NOEL for neurotoxic effects was 0.2 mg/kg/day and the LOEL was 2.0 mg/kg/day. It was observed in this study that, at 2.0 mg/kg/day, brain cholinesterase activity was inhibited by approximately 24 percent. At 10.0 mg/kg/day, brain cholinesterase activity was inhibited by approximately 60 percent, and both plasma and red blood cell cholinesterase were also inhibited. Based on the NOEL, EPA has an oral RfD of 0.002 mg/kg/day for this chemical. In a 1-year feeding study using dogs as the test species, plasma and red blood cell cholinesterase activity were inhibited at 2.0 mg/kg/day. The NOEL was 0.2 mg/kg/day and the LOEL was 2.0 mg/kg/day.

In a 2-generation reproduction study of naled in rats, the NOEL was 6 mg/kg/day. At 18 mg/kg/day, decreased litter size, survival, and pup body weight were observed.

EPA believes that there is sufficient evidence for listing naled on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the chronic neurotoxicity and reproductive toxicity data for this chemical.

Measured aquatic acute toxicity values for naled include a 48–hour EC_{50} of 0.35 ppb for daphnids and a 96–hour LC_{50} of 87 ppb for lake trout. EPA believes that there is sufficient evidence for listing naled on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the environmental toxicity data for this chemical.

174. *Nicotine and salts* (CAL; CERCLA; EPCRA EHS; FIFRA AI; RCRA APP8; RCRA P) (Ref. 8). Nicotine salts will dissociate in aqueous solutions to yield soluble nicotine. Nicotine is highly toxic in humans. The estimated lethal oral dose in adults is approximately 40 to 60 mg. The onset of toxicity is rapid. Symptoms include nausea, salivation, abdominal pain, vomiting, diarrhea, headache, weakness, sweating, and confusion. Nicotine markedly stimulates the central nervous system, causing tremors and convulsions. The stimulation is followed by depression, and death resulting from paralysis of respiratory muscles. Nicotine can also activate parasympathetic ganglia and cholinergic nerve endings resulting in gastrointestinal hyperactivity.

Skeletal defects and occasional cleft palates were observed in mice injected with 25 mg/kg nicotine on gestation days 9 to 11. Reduced size in the newborn of rats and limb deformities in the offspring of swine were reported in swine and rats following oral exposure to 1,058 ppm nicotine (approximately 53 mg/kg/day). Deformities were found in some rabbit fetuses when dams were administered nicotine at a dose of 20 mg/kg 5 times during pregnancy. Pregnant swine fed aqueous leaf extracts of tobacco at the rate of 16 and 32 mg/kg nicotine produced arthrogrypotic newborn pigs.

EPA believes that there is sufficient evidence for listing nicotine and its salts as a category on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the developmental toxicity data for these substances.

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. When filing reports for chemical categories, the releases are determined in the same manner as the thresholds. One report if filed for the category and all releases are reported on this form.

175. *Nitrapyrin (2-chloro-6-(trichloromethyl) pyridine)* (CAS No. 001929-82-4) (FIFRA AI) (Ref. 3). In a 1–year study in dogs fed nitrapyrin adverse effects noted included increased cholesterol and alkaline phosphatase, increased absolute and relative liver weight and panlobular/centrilobular hepatocellular hypertrophy. The NOEL was 3 mg/kg/day and the LOEL was 15 mg/kg/day. In a 10–week reproductive rat study, adverse effects observed included increased incidence of fetal liver hypertrophy and vacuolization at 75 mg/kg/day (LOEL). The NOEL was 20 mg/kg/day. In a 90–day rat feeding study, hepatocellular fatty change and necrosis, renal tubule epithelial cell swelling and increasingly severe interstitial nephritis were observed at 50 mg/kg/day. The NOEL was 15 mg/kg/day. In a 2–year rat feeding study, an increase in glomerulonephropathy was observed in males dosed with 60 mg/kg/day and an increase in hepatic hypertrophy and vacuolization was observed in males and females dosed with 60 mg/kg/day. The NOEL was 20 mg/kg/day.

Increased incidence of crooked hyoid bone and craniofacial abnormalities were observed in the offspring of rabbits orally administered nitrapyrin at 30 mg/kg/day (LOEL) on days 6 through 18 of gestation. The NOEL was 10 mg/kg/day. Decreased weight and hypertrophy and vacuolization of the liver were observed in offspring of rats dosed with 75 mg/kg/day (LOEL) for 10 weeks prior to mating. The NOEL was 20 mg/kg/day. EPA believes that there is sufficient evidence for listing nitrapyrin on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available renal, hepatic, and developmental toxicity data for this chemical.

176. *Nitrate ion* (CAS No. 014797-55-8) (SDWA) (Ref. 8). Nitrate refers to the nitrate ion (NO_3-). Infantile methemoglobinemia occurs in human infants exposed to aqueous solutions of nitrate ion and can progress to cyanosis and death. Based on numerous epidemiological and clinical studies, EPA has determined a LOAEL of 1.8 to 3.2 mg/kg/day and a NOAEL and RfD of 1.6 mg/kg/day, corresponding to 10 mg/L nitrate-nitrogen or 44 mg/L nitrate ion in drinking water. Infants weighing an average of 4 kg (0 to 3 months of age) are the most sensitive population to nitrate-induced methemoglobinemia. This is primarily due to their higher stomach pH which favors the growth of nitrate-reducing bacteria, the immaturity of their metabolic enzyme systems, and reduced capacity of their erythrocytes to reduce methemoglobin to hemoglobin. EPA believes that there is sufficient evidence for listing nitrate ion on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hematological toxicity data for this chemical.

In nitrogen-limited waters, nitrates have the potential to cause increased algal growth leading to eutrophication in the aquatic environment. (Nitrate-nitrogen is the form of nitrogen most available to plants.) Studies of estuarine water at several locations along the eastern coast of the United States have indicated that low concentrations of dissolved nitrogen (e.g., nitrate) limit primary production of plants.

Additions of nitrate to such estuarine systems stimulate primary production of plants and can produce changes in the dominant species of plants, leading to cultural eutrophication and ultimately to deterioration of water quality, including algal blooms.

It has been determined that lakes with a spring maximum concentration of more than 300 ug/L of inorganic nitrogen (e.g., nitrates) could be

expected to have algal nuisances in the summer.

Toxic effects result from oxygen depletion as the algae die and decay. Toxic effects have also been related to the release of decay products or direct excretion of toxic substances from sources such as blue-green algae.

EPA believes that there is sufficient evidence for listing nitrate ion on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data.

177. *Nitric oxide* (CAS No. 010102-43-9) (CERCLA; EPCRA EHS; RCRA APP8; RCRA P) (Ref. 8). The acute toxicity of nitric oxide has been rated high. Nitric oxide causes death or permanent injury after very short exposure to small quantities. Exposure to nitric oxide can result in acute and chronic changes of the pulmonary system including pulmonary edema, pneumonitis, bronchitis, bronchiolitis, emphysema, and methemoglobinemia. Neurologic effects (fatigue, restlessness, anxiety, mental confusion, lethargy, loss of consciousness) have also been reported. The effects of nitric oxide may be related to the formation of methemoglobin. EPA believes that there is sufficient evidence for listing nitric oxide on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available neurological and hematological toxicity data for this chemical.

178. *p-Nitroaniline* (CAS No. 000100-01-6) (CERCLA; RCRA APP8; RCRA P) (Ref. 8). In a 14–day study in mice fed p-nitroaniline in doses as low as 10 mg/kg, 5 days per week, methemoglobin concentrations were found to be significantly higher than those in control animals. In the same study, hematocrit values in mice that received 300 mg/kg, and total erythrocyte counts in mice that received 100 or 300 mg/kg, were significantly lower than those of control animals. Similar effects were observed in 13–week and 2–year mouse studies. In the 2–year study, lesions related to the administration of p-nitroaniline occurred in the spleen, liver, and bone marrow (primarily in mice receiving 30 or 100 mg/kg) and were observed at 9 and 15 months. In addition, increases in the incidence or severity of splenic congestion, hematopoiesis, pigment (hemosiderin) accumulation, Kupffer cell pigmentation in the liver, and bone marrow hypercellularity (hyperplasia). EPA believes that there is sufficient evidence for listing p-nitroaniline on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the chronic toxicity data for this chemical.

179. *Nitrogen dioxide* (CAS No. 010102-44-0) (CERCLA; EPCRA EHS; RCRA APP8; RCRA P) (Ref. 8). Acid precipitation occurs in large regions of the Eastern United States and Canada, Europe, and Japan. This widespread occurrence of acid precipitation and dry deposition results in large part from man-made emissions of oxides of sulfur and nitrogen (e.g., nitrogen dioxide). These substances are transformed in the atmosphere into sulfuric acid and nitric acid, transported over great distances and deposited on vegetation, soils, surface waters, and materials. These substances are transferred from the atmosphere into ecosystems by the absorption of gases, the impaction and gravitational settling of fine aerosols and coarse particles, and precipitation.

Acids contained in polluted snow are released as contaminated meltwater. The resulting release of pollutants can cause major or rapid changes in the acidity of streams and lake waters. Interference with normal reproduction in fish populations is induced by acidity of lake and stream waters. Reproduction of frogs and salamanders is also inhibited by atmospheric acidification of surface waters.

Atmospheric deposition of sulfuric acid and nitric acid can cause serious damage to crops and forests. Biological effects include induction of necrotic lesions, loss of nutrients due to leaching from foliar organs, accelerated erosion of waxes and leaf surfaces, and interference with normal reproductive processes. Acidification also decreases the rate of many soil processes such as nitrogen fixation and the breakdown of organic matter.

EPA believes that there is sufficient evidence for listing nitrogen dioxide on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data for this chemical.

Nitrogen dioxide is regulated under Title I of the CAA (Provisions for Attainment and Maintenance of National Ambient Air Quality Standards). In addition to this proposal to add nitrogen dioxide to EPCRA section 313, in Units IV.B.36. and 235, EPA is proposing to add two other chemicals, carbon monoxide and sulfur dioxide, that are regulated by Title I of the CAA. Sulfur dioxide is also regulated by Title IV of the CAA (Acid Deposition Control). Extensive data, which are highly technical, are collected on these chemicals as required by the CAA. EPA requests comment on the following: (1) Is the information collected under the CAA sufficient for public right-to-know purposes; and (2) suggestions on how the data collected on these chemicals pursuant to CAA Titles I and IV could be used to meet the purposes of EPCRA section 313.

180. *Norflurazon (4-Chloro-5-(methylamino)-2-[3(trifluoromethyl)phenyl]-3(2H)-pyridazinone)* (CAS No. 027314-13-2) (FIFRA AI) (Ref. 3). Congestion of the liver, hepatocyte swelling and increased liver weights, and increase in colloid vacuole in the thyroid were observed in dogs fed 450 ppm (10.25 mg/kg/day) norflurazon for 6 months. The NOEL was 150 ppm (3.75 mg/kg/day). An oral RfD of 0.04 mg/kg/day has been determined. Increased relative liver weight and hypertrophy of the thyroid with depletion of colloid were seen in rats fed 2,500 ppm (125 mg/kg/day) norflurazon for 90 days. The NOEL was 500 ppm (25 mg/kg/day). Hepatic hyperplasia and hypertrophy and increased relative liver weight were noted in a 28–day feeding study in rats. The LOEL was 1,000 ppm (50 mg/kg/day) and the NOEL was 500 ppm (25 mg/kg/day). Increased relative liver weight and diffuse and smooth granular livers were seen in a 28–day feeding study in mice. The LOEL was 2,520 ppm (328 mg/kg/day) and the NOEL was 420 ppm (55 mg/kg/day). EPA believes that there is sufficient evidence for listing norflurazon on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hepatic and thyroid toxicity data.

181. *Oryzalin (4-(Dipropylamino)-3,5-dinitrobenzene sulfonamide)* (CAS No. 019044-88-3) (FIFRA AI) (Ref. 3). Reduced hemoglobin and hematocrit levels, decreased red blood cell count, increased blood urea nitrogen (BUN) and alkaline phosphatase and SGPT, anemia, hepatic changes, splenic hematopoiesis and hyperplastic bone marrow were observed in dogs fed 56.25 mg/kg/day (the NOEL was 18.75 mg/kg/day) for 3 months. Increases in serum cholesterol levels, alkaline phosphatase activity, and relative liver and kidney weights and decreases in alanine transaminase (the LOEL was 50 mg/kg/day; the NOEL was 5 mg/kg/day) were observed in dogs fed oryzalin for 1–year. Decreased red blood cell count and hematocrit and hemoglobin levels (LOEL was 45 mg/kg/day; NOEL was 15 mg/kg/day) were noted in a 1-year feeding study in rats. In a 2–year feeding study in rats, decreased red blood cell count and hematocrit and hemoglobin levels, and increased BUN and liver and kidney weights (the LOEL was 45 mg/kg/day; the NOEL was 15 mg/kg/day) were observed. EPA believes that there is sufficient evidence for listing oryzalin on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on

the available hepatic and hematological toxicity data for this chemical.

182. *Oxydemeton methyl (S-(2-(Ethylsulfinyl)ethyl) O,O-dimethyl ester phosphorothioic acid)* (CAS No. 000301-12-2) (FIFRA AI) (Ref. 3). Two multigeneration reproduction studies indicate a variety of reproductive effects at 2.1 to 2.5 mg/kg/day. These effects include decreased litter size and viability, decreased weight of the testes and ovaries, and increased epididymal vacuolation. The NOELs were 0.38 and 0.5 mg/kg/day. A NOEL of 0.9 mg/kg/day was determined in a 5–day study in the rat. The LOEL for decreased fertility and epididymal sperm motility was 5 mg/kg/day.

Oxydemeton methyl can cause inhibition of brain, plasma, and red blood cell cholinesterase. In a 2-generation reproduction study, statistically significant inhibition of red blood cell and brain cholinesterase activity (the NOEL was less than 0.043 mg/kg/day) was observed in adult males and females of the F^0 and F^1 generations. In a 5–day feeding (dominant lethal plus) study, inhibition of plasma cholinesterase activity (the LOEL was 1.5 mg/kg/day; the NOEL was 0.45 mg/kg/day) was observed. EPA believes that there is sufficient evidence for listing oxydemeton methyl on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available reproductive and neurological toxicity data for this chemical.

183. *Oxydiazon (3-[2,4-Dichloro-5-(1-methylethoxy)phenyl]5- (1,1-dimethylethyl)-1,3,4-oxadiazol-2(3H)-one)* (CAS No. 019666-30-9) (FIFRA AI) (Ref. 3). Rats given 40 mg/kg/day by gavage on days 6 to 15 of gestation exhibited increased fetal resorptions. The NOEL was 12 mg/kg/day.

Increased liver and kidney weight (associated with no pathology) and increased alkaline phosphatase activity were observed in rats fed 100 mg/kg/day (the NOEL was 25 mg/kg/day) for 90 days. Increased levels of SGPT and alkaline phosphatase activities and increased liver weight (the LOEL was 5 mg/kg/day; the NOEL was 0.5 mg/kg/day) were observed in a 2–year feeding study in rats. Effects noted at 150 mg/kg/day included liver pathology, hemolytic anemia, increased kidney weight, and pigment nephrosis. Based on the NOEL, an oral RfD of 0.005 mg/kg/day was derived. EPA believes that there is sufficient evidence for listing oxydiazon on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available developmental, hepatic, and renal toxicity data for this chemical.

184. *Oxyfluorfen* (CAS No. 042874-03-3) (FIFRA SR) (Ref. 8). Oxyfluorfen is a phenoxyphenyl-type herbicide. Several chronic oral toxicity studies suggest that oxyfluorfen may be hepatotoxic. Hepatic effects (e.g. increased absolute liver weight, necrosis, regeneration, and hyperplastic nodules) were observed in mice fed diets containing greater than 3 mg/kg/day oxyfluorfen for 20 months (the NOEL was 0.3 mg/kg/day). Based on these findings, an oral RfD value of 0.003 mg/kg/day was derived. This study was supported by other chronic feeding studies that demonstrated increases in liver weight, alkaline phosphatase activity, and bile pigmented hepatocytes (the LOEL was 15 mg/kg/day; the NOEL was 2.5 mg/kg/day) in dogs, and minimal hypertrophy of centrilobular hepatocytes (the LOEL was 40 mg/kg/day; the NOEL was 2 mg/kg/day) in rats. EPA believes that there is sufficient evidence for listing oxyfluorfen on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the hepatotoxic effects of this chemical.

The estimated chronic MATC values for fish and daphnids are 9 ppb and 20 ppb oxyfluorfen, respectively. The estimated log K_{ow} is 6.1. EPA believes that there is sufficient evidence for listing oxyfluorfen on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the environmental toxicity data and potential for bioaccumulation for this chemical.

185. *Ozone* (CAS No. 010028-15-6) (EPCRA EHS) (Ref. 8). Information from a large number of studies of both humans and animals indicate that ozone can affect structure, function, metabolism, pulmonary defense against bacterial infection, and extrapulmonary effects. Among these extrapulmonary effects are: (1) Cardiovascular effects; (2) reproductive and teratological effects; (3) central nervous system effects; (4) alterations in red blood cell morphology; (5) enzymatic activity; and (6) cytogenetic effects on circulating lymphocytes. EPA believes that there is sufficient evidence for listing ozone on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available toxicity data for this chemical.

Effects of ozone on green plants include injury to foliage, reductions in growth, losses in yield, alterations in reproductive capacity, and alterations in susceptibility to pests and pathogens. Based on the known interrelationships of different components of ecosystems, such effects, if of sufficient magnitude, may potentially lead to irreversible changes of sweeping nature to ecosystems.

Measured aquatic acute toxicity values for ozone include a 96–hour LC_{50} of 80 ppb for striped bass, a 96–hour LC_{50} of 30 ppb for channel catfish, and a 96–hour LC_{50} of 9.3 ppb for rainbow trout. EPA believes that there is sufficient evidence for listing ozone on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available ecotoxicity data for this chemical.

186. *Paraquat dichloride* (CAS No. 001910-42-5) (EPCRA EHS; FIFRA SR) (Ref. 8). Paraquat can cause death in humans as a consequence of severe injury to the lungs, or as a result of kidney, liver, or heart failure. Following exposure, death may occur in 24 hours or less. The acute oral LD_{50} values for paraquat are reported as 57, 120, 25, 50 and 35 mg/kg in the rat, mouse, dog, monkey, and cat, respectively. Chronic pneumonitis (the LOEL was 0.93 mg/kg/day; the NOEL was 0.45 mg/kg/day) was reported in dogs fed diets containing paraquat dichloride for 52 weeks. These results are supported by the results of a 2–year feeding study in rats (the LOEL was 3.75 mg/kg/day based on nonneoplastic lung lesions; the NOEL was 1.25 mg/kg/day) and a 90–day feeding study in dogs (the LOEL was 1.5 mg/kg/day based on increased lung weight, alveolitis, and alveolar collapse; the NOEL was 0.5 mg/kg/day).

EPA believes that there is sufficient evidence for listing paraquat dichloride on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the chronic toxicity data for this chemical.

187. *Pebulate (Butylethylcarbamothioic acid S-propyl ester)* (CAS No. 001114-71-2) (FIFRA AI) (Ref. 3). In a 1-year dog feeding study, a NOEL of greater than 5 mg/kg/day was established due to abnormal behavior, ataxia, convulsions, and neurological effects in the brain and spinal cord at 100 mg/kg/day. EPA believes that there is sufficient evidence for listing pebulate on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available neurological toxicity data.

188. *Pendimethalin (N-(1-Ethylpropyl)-3,4-dimethyl-2,6-dinitrobenzenamine)* (CAS No. 040487-42-1) (FIFRA AI) (Ref. 3). Increased liver weights and alkaline phosphatase activity and hepatic lesions (the LOEL was 50 mg/kg/day; the NOEL was 12.5 mg/kg/day) were observed in dogs fed pendimethalin for 2 years. EPA derived an oral RfD of 0.04 mg/kg/day. Hypertrophy of the liver and increased liver weights were observed in rats fed 5,000 ppm (250 mg/kg/day) for 3 months. The NOEL was 25 mg/kg/day.

EPA believes that there is sufficient evidence for listing pendimethalin on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hepatic toxicity data.

189. *Pentobarbital sodium* (CAS No. 000057-33-0) (CAL) (Ref. 8). Pentobarbital sodium is commonly used as a sedative hypnotic. The average adult sedative dose is 20 to 40 mg orally. The average adult hypnotic dose is 100 to 200 mg orally. Pentobarbital is also used parenterally or rectally to provide basal hypnosis for general, spinal, or regional anesthesia. Like other barbiturates, a common adverse effect to using pentobabital sodium is central nervous system depression. Chronic exposure to pentobarbital sodium may lead to psychological and physical dependence.

Intraperitoneal injection of 20 mg/kg on day 1 of pregnancy produced adverse effects on fertility in rats. Intraperitoneal injections of 80 mg/kg to rats on day 1 of pregnancy caused preimplantation loss. Intraperitoneal injection of 94.5 mg/kg on day 2 of pregnancy decreased fertility and caused fetal death in rats. Intraperitoneal injection of 22 mg/kg on day 10 of pregnancy caused adverse effects in rat fetuses (details of study not reported). Subcutaneous injection of 520 mg/kg of pentobarbital sodium on days 9 to 21, or administration of 30 mg/kg on day 19 of pregnancy produced abnormal behavioral effects in rat offspring. Exposure to pentobarbital sodium during pregnancy can cause fetal addiction to the substance.

EPA believes that there is sufficient evidence for listing pentobarbital sodium on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the developmental, reproductive, and chronic neurological toxicity data for this chemical.

190. *Perchloromethyl mercaptan* (CAS No. 000594-42-3) (CERCLA; EPCRA EHS) (Ref. 8). The rat oral LD_{50} and 4–hour rat inhalation LC_{50} values for perchloromethyl mercaptan are 8.26 mg/kg and 0.26 mg/L, respectively. The 2–hour mouse inhalation LC_{50} value is reported to be 0.296 mg/L. In an eye irritation test, 50 micrograms (μg) (0.13 mg/kg/day) placed in a rabbit's eye for 24 hours produced a severe reaction. EPA's exposure analysis indicates that perchloromethyl mercaptan concentrations are likely to exist beyond facility site boundaries, as a result of continuous, or frequently recurring releases, at levels that can reasonably be anticipated to cause significant adverse acute human health effects. EPA believes that there is sufficient evidence for listing perchloromethyl mercaptan on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(A) based on the available acute toxicity and exposure data for this chemical.

191. *Permethrin (3-(2,2-Dichloroethenyl)-2,2-dimethylcyclopropanecarboxylic acid, (3-phenoxyphenyl)methyl ester)* (CAS No. 052645-53-1) (FIFRA AI) (Ref. 3). Increased liver weights (the LOEL was 500 ppm or 25 mg/kg/day; the NOEL was 100 ppm or 5 mg/kg/day) were observed in rats fed permethrin for 2 years. Based on the NOEL, EPA derived an oral RfD of 0.05 mg/kg/day. Decreased alkaline phosphatase activity, hepatocellular swelling, and increased liver weight (the LOEL was 100 mg/kg/day; the NOEL was 5 mg/kg/day) were observed in dogs orally administered (in capsules) permethrin for 1–year. Tremors, excessive salivation, convulsions, and incoordination were noted at 1,000 mg/kg/day. EPA believes that there is sufficient evidence for listing permethrin on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hepatic toxicity data.

Aquatic acute toxicity values for permethrin include a fathead minnow 96–hour LC_{50} of 3.5 ppb, a rainbow trout 96–hour measured LC_{50} of 0.62 ppb, a bluegill 96–hour LC_{50} of 2.52 ppb, an Atlantic silverside 96–hour measured LC_{50} 2.2 ppb, and a daphnid 48–hour LC_{50} of 0.32 ppb. EPA believes that there is sufficient evidence for listing permethrin on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data.

192. *Phenanthrene* (CAS No. 000085-01-8) (CERCLA; CWA PP) (Ref. 8). Measured aquatic acute toxicity data for phenanthrene include a 48–hour LC_{50} of 700 ppb for daphnids. The measured 28–day LC_{50} for rainbow trout is 40 ppb, and teratogenetic effects were noted. The measured bioconcentration factor (BCF) values include a fathead minnow 28–day BCF of 5,100 and a daphnid 24–hour BCF of 1,165. EPA believes that there is sufficient evidence for listing phenanthrene on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data for this chemical and its potential to bioaccumulate.

193. *Phenothrin (2,2-dimethyl-3-(2-methyl-1-propenyl) cyclopropanecarboxylic acid (3-phenoxyphenyl)methyl ester)* (CAS No. 026002-80-2) (FIFRA AI) (Ref. 3). Hepatocellular enlargement and increased absolute and relative liver weights were observed in a chronic feeding study in dogs. The LOEL was 27.7 mg/kg/day in males and 26.8 mg/kg/day in females. The NOEL was 8.2 mg/kg/day in males and 7.1 mg/kg/day in females. Hepatocellular hypertrophy and increased relative liver weight (the LOEL was 150 mg/kg/day; the NOEL was 50 mg/kg/day) were observed in a chronic oncogenicity study in rats. Increased liver weight (the LOEL was 150 mg/kg/day, the NOEL was 45 mg/kg/day) was noted in another chronic oncogenicity feeding study in mice. EPA believes that there is sufficient evidence for listing phenothrin on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hepatic toxicity data for this chemical.

Aquatic acute toxicity values for phenothrin include a rainbow trout 96–hour LC_{50} of 16.7 ppb and a goldfish 48–hour LC_{50} of 100 ppb. EPA believes that there is sufficient evidence for listing phenothrin on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data for this chemical.

194. *1,2-Phenylenediamine* (CAS No. 000095-54-5) (RCRA APP8) (Ref. 8). EPA has classified 1,2-phenylenediamine as a Group B2 compound, i.e., a probable human carcinogen. 1,2–Phenylenediamine dihydrochloride appeared to be carcinogenic in both rats and mice, as evidenced by an increased incidence of hepatocellular carcinomas in both species. A significantly increased incidence of hepatocellular carcinomas was observed in high dose group male rats and mice, and female mice of both treated groups. EPA believes that there is sufficient evidence for listing 1,2-phenylenediamine on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the carcinogenicity data for 1,2–phenylenediamine dihydrochloride.

195. *1,3-Phenylenediamine* (CAS No. 000108-45-2) (RCRA APP8) (Ref. 8). Increased absolute and relative liver weights and degenerative liver lesions (the LOEL was 18 mg/kg/day; the NOEL was 6.0 mg/kg/day) were noted in a 90–day oral study in rats exposed to 1,3-phenylenediamine. EPA believes that there is sufficient evidence for listing 1,3-phenylenediamine on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the hepatotoxicity data for this chemical.

196. *1,2-Phenylenediamine dihydrochloride* (CAS No. 000615-28-1) (RCRA APP8) (Ref. 8). EPA has classified 1,2–phenylenediamine as a Group B2 compound, i.e., a probable human carcinogen. 1,2-Phenylenediamine dihydrochloride appeared to be carcinogenic in both rats and mice, as evidenced by an increased incidence of hepatocellular carcinomas in both species. A significantly increased incidence of hepatocellular

carcinomas was observed in high dose group male rats and mice, and female mice of both treated groups. EPA believes that there is sufficient evidence for listing 1,2-phenylenediamine dihydrochloride on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the carcinogenicity data for this chemical.

197. *1,4-Phenylenediamine dihydrochloride* (CAS No. 000624-18-0) (RCRA APP8) (Ref. 8). Measured aquatic acute toxicity for 1,4-phenylenediamine include a fish 96–hour LC_{50} of 60 ppb. EPA believes that there is sufficient evidence for listing 1,4-phenylenediamine dihydrochloride on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data for 1,4-phenylenediamine.

198. *Phenytoin* (CAS No. 000057-41-0) (CAL; IARC; NTP) (Ref. 8). Phenytoin is a hydantoin-type anticonvulsant, and is used mainly in the prophylactic management of tonic-clonic (grand mal) seizures and partial seizures with complex symptomatology. In doses used to treat seizure disorders (i.e., 300 mg/day in adults, 5 mg/kg/day in children) phenytoin can cause adverse effects such as constipation, dysphagia, nausea, vomiting, anorexia and weight loss. Ingestion of 4.5 g (64 mg/kg/day) by adults and 0.6 g (60 mg/kg/day) by children has produced transient coma with motor restlessness. Ingestion of 11 mg/kg/day produced changes in motor activity in a child (duration of study not reported). Oral administration of 7.8 mg/kg/day for 4 days produced encephalitis, hallucinations, and irritability in a man. Ingestion of 7.6 mg/kg/day for 2 weeks caused encephalitis, hallucinations, and ataxia in a woman.

Phenytoin is classified as a Group 2B compound by IARC; i.e., possible human carcinogen. Ingestion of 16.5 mg/kg/day for 1–year produced lymphoma including Hodgkin's disease and skin tumors in a child. Oral exposure to phenytoin produced lymphoma in mice (doses and duration of study not reported).

Oral administration of 5.9 mg/kg/day to a woman for the first 39 weeks of pregnancy induced kidney tumors in the offspring. In another study, oral administration of 5.9 mg/kg/day to a woman for the first 39 weeks of pregnancy induced brain tumors in the offspring. Oral administration of 2 mg/kg/day to a woman for 1–year produced lymphoma including Hodgkin's disease. Congenital malformation was reported in 6.12 percent of births to 98 epileptic mothers receiving phenytoin regularly during the first 4 months of pregnancy. Hypothrombinemia and hemorrhage has occurred in newborns of mothers who received phenytoin during pregnancy. Oral doses of 4.0 to 5.9 mg/kg/day administered to women for the first 39 weeks of pregnancy produced craniofacial abnormalities, nervous system disorders, and delayed physical effects in their children. Doses of 2.0 mg/kg/day given to a woman for the first 39 weeks of pregnancy produced abnormalities of skin, appendages, and musculoskeletal system in her child as well as other developmental abnormalities. Oral doses of 5.0 mg/kg/day produced biochemical and metabolic abnormalities in the offspring. Higher doses of phenytoin (130 mg/kg/day) orally administered to rats produced behavioral, growth, musculoskeletal, and nervous system abnormalities in the offspring.

EPA believes that there is sufficient evidence for listing phenytoin on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the chronic neurological and developmental toxicity data and on the carcinogenicity data for this chemical.

199. *Phosphine* (CAS No. 007803-51-2) (CAA HAP) (Ref. 7). Available data on phosphine indicate that its inhalation LC_{50} for rats is between 4 and 40 ppm (the exposure time was 4 hours). Phosphine is a highly-toxic gas with a probable oral lethal dose of 5 mg/kg. An air concentration of 3 ppm is safe for long-term exposure, 500 ppm is lethal in 30 minutes, and a concentration of 1,000 ppm is lethal after a few breaths.

EPA's exposure analysis indicates that phosphine concentrations are likely to exist beyond facility site boundaries, as a result of continuous, or frequently recurring releases, at levels that can reasonably be anticipated to cause significant adverse acute human health effects. EPA believes that there is sufficient evidence for listing phosphine on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(A) based on the available acute toxicity and exposure data for this chemical.

200. *Phosphorus oxychloride* (CAS No. 010025-87-3) (CERCLA; EPCRA EHS) (Refs. 5 and 8). Phosphorus oxychloride reacts with water to yield phosphoric acid and hydrochloric acid.

Phosphoric acid, as well as other phosphates, have the potential to cause increased algal growth leading to eutrophication in the aquatic environment.

Eutrophication may result when nutrients, especially phosphates, enter into an aquatic ecosystem in the presence of sunlight and nitrogen. The phosphate ion is a plant nutrient, which can be a major limiting factor for plant growth in freshwater environments. In excess, phosphates can cause algal blooms. Toxic effects result from oxygen depletion as the algae die and decay. Toxic effects have also been related to the release of decay products or direct excretion of toxic substances from sources such as blue-green algae.

Laboratory studies indicate that eutrophication may occur at phosphate concentrations as low as 50 ppb in lakes. The resulting oxygen depletion and toxic decay products (e.g., hydrogen sulfide) kill many invertebrates and fish.

Although green algae are more sensitive to growth stimulation by phosphates in fresh water, blue-green algal blooms may cause greater damage. At least three species of blue-green algae are known to excrete toxins. Secretion by cyanobacteria of dyalyzable metabolites have inhibited the growth of other species of algae and may result in algal monoculture. When algal blooms of these toxic species occur in a reservoir, lake, slough, or pond, the cells and toxins can become sufficiently concentrated to cause illness or death in invertebrates and vertebrates. Major losses have been reported for cattle, sheep, hogs, birds (domestic or wild) and fishes, minor losses for dogs, horses, small wild animals, amphibians, and invertebrates.

Eutrophication may occur in slow moving rivers, but is less likely in swift rivers where rapid mixing occurs. Light is the most important limiting factor because rivers are murkier than lakes thus, the chances of eutrophication in swift rivers are slight. However, lakes and reservoirs collect phosphates from influent streams and store a fraction of them within consolidated sediments, thus serving as a phosphate sink.

The available information derived from animal and controlled human studies clearly indicates that exposure to acid aerosols can produce health effects of concern, particularly in sensitive subgroups of the population and after chronic exposure. The bulk of these studies, however, have examined sulfuric acid exposures. Data for other acid species and mixtures are extremely limited. However, as the effects appear to be due to the acidity of the species, this data should pertain to acid aerosols consisting of other mineral acids, such as hydrochloric acid. The effects seen range from mild and transient changes, such as small, reversible functional effects in exercising asthmatics, to more substantial effects that may have acute or chronic health consequences, such as persistently altered clearance and structural changes that may be suggestive of chronic lung disease. In addition, there are some notable consistencies in the health effects

information across various studies and disciplines.

EPA believes that there is sufficient evidence for listing phosphorous oxychloride on EPCRA section 313 pursuant to EPCRA sections 313(d)(2)(B) and (C) based on the available chronic human and environmental toxicity data for its degradation products phoshoric acid and hydrochloric acid.

201. *Phosphorus pentachloride* (CAS No. 010026-13-8) (EPCRA EHS) (Refs. 5 and 8). Phosphorus pentachloride reacts with water to yield phosphoric acid and hydrochloric acid. As described in Unit IV.B.200. of this preamble, phosphates, including phosphoric acid, have the potential to cause increased algal growth leading to eutrophication and fish kills in the aquatic environment.

The available information derived from animal and controlled human studies clearly indicates that exposure to acid aerosols can produce health effects of concern, particularly in sensitive subgroups of the population and after chronic exposure. The bulk of these studies, however, have examined sulfuric acid exposures. Data for other acid species and mixtures are extremely limited. However, as the effects appear to be due to the acidity of the species, this data should pertain to acid aerosols consisting of other mineral acids, such as hydrochloric acid. The effects seen range from mild and transient changes, such as small, reversible functional effects in exercising asthmatics, to more substantial effects that may have acute or chronic health consequences, such as persistently altered clearance and structural changes that may be suggestive of chronic lung disease. In addition, there are some notable consistencies in the health effects information across various studies and disciplines.

EPA believes that there is sufficient evidence for listing phosphorus pentachloride on EPCRA section 313 pursuant to EPCRA sections 313(d)(2)(B) and (C) based on the available chronic human and environmental toxicity data for its degradation products phoshoric acid and hydrochloric acid.

202. *Phosphorus pentasulfide* (CAS No. 001314-80-3) (CERCLA) (Refs. 5 and 8). Phosphorus pentasulfide reacts in water to yield phosphoric acid and hydrogen sulfide.

As described in Unit IV.B.200. of this preamble, phosphates, including phosphoric acid, have the potential to cause increased algal growth leading to eutrophication and fish kills in the aquatic environment.

Acute exposures to large amounts of hydrogen sulfide (approximately 250 ppm or more) have produced pulmonary edema, unconsciousness, respiratory paralysis, asphyxiation, and/or death in some individuals. Similar effects are also noted in animals. In a subchronic study, inflammation of the nasal mucosa occurred in mice following 90–day inhalation of hydrogen sulfide, resulting in a NOAEL of 42.5 mg/m^3 (30.5 ppm; Human Equivalent Concentration (HEC) is 0.93 mg/m^3) and a LOAEL of 110 mg/m^3 (80 ppm; HEC is 2.4 mg/m^3). Other respiratory effects, such as alveolar edema, infiltrates in the bronchioles, cellular necrosis, hyperplasia, and exfoliation in various respiratory tissues, have been reported in rats.

Aquatic toxicity test data for hydrogen sulfide show that measured fish 96–hour LC_{50} values range from 7 to 776 ppb.

EPA believes that there is sufficient evidence for listing phosphorus pentasulfide on EPCRA section 313 pursuant to EPCRA sections 313(d)(2)(B) and (C) based on the available chronic human and environmental toxicity data for its degradation products, phosphoric acid and hydrogen sulfide.

203. *Phosphorus pentoxide* (CAS No. 001314-56-3) (EPCRA EHS) (Refs. 5 and 8). Phosphorus pentoxide rapidly hydrolyzes in the presence of water to yield phosphoric acid.

As described in Unit IV.B.200. of this preamble, phosphates, including phosphoric acid, have the potential to cause increased algal growth leading to eutrophication and fish kills in the aquatic environment. EPA believes that there is sufficient evidence for listing phosphorous pentoxide on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data for its degradation product phosphoric acid.

204. *Picloram* (CAS No. 001918-02-1) (FIFRA AI; SDWA) (Ref. 8). Animal studies in dogs, rats, or mice for various durations (2 weeks to 2 years) have indicated the liver as the primary target of picloram toxicity. In a 6–month feeding study in beagle dogs, a LOAEL of 35 mg/kg/day and a NOAEL of 7 mg/kg/day were determined for increased liver weights (relative and absolute). At a higher dose (175 mg/kg/day), there were increases in serum alkaline phosphatase concomitant with the increases in liver weight. Other toxic effects in the higher dosed animals included reduced food consumption and body weight. EPA has derived an oral RfD of 0.07 mg/kg/day for this chemical based on the findings of this study. Hepatotoxicity has also been reported in a 2–year rat feeding study. The LOAEL was 60 mg/kg/day based on changes in liver histopathology. The NOAEL was 20 mg/kg/day. Hepatotoxicity was also observed in a 90–day rat feeding study. The LOAEL was 150 mg/kg/day based on changes in liver histopathology, necrosis, and bile duct proliferation. The NOAEL was 50 mg/kg/day. Increased liver weights were also reported in mice following dietary exposure to picloram for 13 weeks. The LOAEL was 1,000 mg/kg/day. Liver swelling was reported in rats administered picloram in feed for 13 weeks. The LOAEL was 150 mg/kg/day. EPA believes that there is sufficient evidence for listing picloram on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hepatotoxicity data for this chemical.

205. *Piperonyl butoxide* (CAS No. 000051-03-6) (FIFRA SR) (Ref. 8). Measured aquatic acute toxicity data for piperonyl butoxide include a 96–hour LC_{50} of 3.4 ppb for rainbow trout and a 96–hour LC_{50} of 4.2 ppb for bluegill. EPA believes that there is sufficient evidence for listing piperonyl butoxide on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the environmental toxicity data for this chemical.

206. *Pirimiphos methyl (O-(2-(diethylamino)-6-methyl-4-pyrimidinyl)-O,O-dimethyl phosphorothioate)* (CAS No. 029232-93-7) (FIFRA AI) (Ref. 3). Pirimiphos methyl is a cholinesterase inhibitor in humans and other mammalian species. A mild and transient decrease in plasma cholinesterase activity was observed in 2 of 4 female humans given pirimiphos methyl daily in a capsule at dose levels of 0.25 mg/kg/day for 56 days. This effect was not seen in 3 of 3 males. The dose level of 0.25 mg/kg/day was considered a NOEL for plasma cholinesterase inhibition. Based on the NOEL, an oral RfD of 0.01 mg/kg/day was derived. The findings of the 56–day study were corroborated by the 28–day feeding study (capsule) with 5 male human volunteers where 1 individual showed borderline cholinesterase depression. Inhibition of brain cholinesterase (LOEL was 0.5 mg/kg/day, the NOEL for cholinesterase inhibition was not determined) was observed in a 2–year feeding study in dogs. Inhibition of plasma cholinesterase activity (the LOEL was 2.5 mg/kg/day; the NOEL was 0.5 mg/kg/day) was seen in a 2–year feeding study in rats. No clinical signs were reported for the above studies. EPA believes that there is sufficient evidence for listing pirimiphos methyl on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available neurological toxicity data for this chemical.

207. *Polycyclic aromatic compounds (PACs)* (CAS No. NA) (CAA HAP) (Ref. 7). Polycyclic aromatic compounds are a class of chemicals that include polycyclic aromatic hydrocarbons, azapolycyclic aromatic hydrocarbons, thio-polycyclic aromatic hydrocarbons, nitroarenes, and others. PACs can be formed in any combustion process that involves the burning of fuels or, more generally, materials containing carbon and hydrogen. Some industrial sources include coke ovens, catalytic cracking of crude oil, carbon black production, and iron and steel processes.

Materials containing mixtures of PACs have been shown to be carcinogenic. Several epidemiology studies have shown increased mortality due to lung cancer in humans exposed to coke-oven emissions, roofing-tar emissions, and cigarette smoke. Each of these mixtures contains benzo(a)pyrene, benzo(a)anthracene, benzo(b)fluoranthene, benzo(a)phenanthrene, and dibenzo(a,h)anthracene as well as other potentially carcinogenic PACs and other carcinogenic and potentially carcinogenic chemicals, tumor promotors, initiators, and co-carcinogens such as nitrosoamines, coal tar pitch, and creosote. Although it is impossible to evaluate the contribution of any individual PAC to the total carcinogenicity of these mixtures to humans, reports of this nature provide qualitative evidence of the potential for mixtures containing PACs to cause cancer in humans. In addition, several PACs caused cancer in animals when orally (e.g., benz(a)anthracene, benzo(a)pyrene, dibenz(a,h)anthracene), dermally (e.g., benz(a)anthracene, benzo(a)phenanthrene, benzo(b)fluoranthene, benzo(a)pyrene, dibenz(a,h)anthracene, and indeno (1,2,3-cd)pyrene) or inhalationally (e.g., benzo(a)pyrene) exposed. EPA believes that there is sufficient evidence for listing these PACs on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for these chemicals. EPA is proposing to create a delimited category for PACs that includes the chemicals discussed below.

a. *Benzo(b)fluoranthene* (CAS No. 000205-99-2). Benzo(b)fluoranthene is classified as a Group B2 compound by EPA, i.e., the compound is a probable human carcinogen. It is classified as a Group 2B compound by IARC, i.e., the compound is a possible human carcinogen. Benzo(b)fluoranthene produced tumors in mice after lung implantation, intraperitoneal or subcutaneous injection and skin painting. EPA believes that there is sufficient evidence for listing benzo(b)fluoranthene on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for this chemical.

b. *Benzo(j)fluoranthene* (CAS No. 000205-82-3). Benzo(j)fluoranthene is classified as a Group 2B compound by IARC, i.e., the compound is a possible human carcinogen. In multiple skin painting assays and in a mouse-skin initiation-promotion assay, benzo(j)fluoranthene produced tumors in female mice. EPA believes that there is sufficient evidence for listing benzo(j)fluoranthene on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for this chemical.

c. *Benzo(k)fluoranthene* (CAS No. 000207-08-9). Benzo(k)fluoranthene is classified as a Group B2 compound by EPA, i.e., the compound is a probable human carcinogen. It is also classified as a Group 2B compound by IARC, i.e., the compound is a possible human carcinogen. Benzo(k)fluoranthene produced tumors after lung implantation in mice and when administered with a promoting agent in skin painting studies. Equivocal results have been found in a lung adenoma assay in mice. Benzo(k)fluoranthene is mutagenic in bacteria. EPA believes that there is sufficient evidence for listing benzo(k)fluoranthene on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for this chemical.

d. *Carbazole* (CAS No. 000086-74-8). Mice fed a basal diet containing carbazole showed a dose-related increase in liver nodules and hepatocellular carcinomas after oral administration. EPA believes that there is sufficient evidence for listing carbazole on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for this chemical.

e. *Cyclopenta(cd)pyrene* (CAS No. 027208-37-3). In a skin painting assay and in several mouse-skin initiation-promotion assays, cyclopenta(cd)pyrene produced tumors in female mice. Cyclopenta(cd)pyrene is also mutagenic to Salmonella and mammalian cells in vitro and induces morphologic transformation in C3H10T1/2 cells in vitro. EPA believes that there is sufficient evidence for listing cyclopenta(cd)pyrene on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for this chemical.

f. *Dibenz(a,c)anthracene (CAS No. 000215-58-7).* In a skin painting assay and in several mouse-skin initiation-promotion assays, dibenz(a,c)anthracene produced tumors in female mice. EPA believes that there is sufficient evidence for listing dibenz(a,c)-anthracene on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for this chemical.

g. *Dibenz(a,h)acridine* (CAS No. 000226-36-8). Dibenz(a,h)acridine is classified as a Group 2A compound by IARC, i.e., the compound is a probable human carcinogen. Dibenz(a,h)acridine has been shown to be carcinogenic in animals. EPA believes that there is sufficient evidence for listing dibenz(a,h)acridine on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for this chemical.

h. *Dibenz(a,j)acridine* (CAS No. 000224-42-0). Dibenz(a,j)acridine is classified as a Group 2B compound by IARC, i.e., the compound is a possible human carcinogen. Dibenz(a,j)acridine has been shown to be carcinogenic in animals. EPA believes that there is sufficient evidence for listing dibenz(a,j)acridine on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for this chemical.

i. *Dibenz(a,j)anthracene* (CAS No. 000224-41-9). Dibenz(a,j)anthracene produced tumors after subcutaneous injection and after skin painting in female mice. EPA believes that there is sufficient evidence for listing dibenz(a;j)anthracene on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for this chemical.

j. *Dibenzo(a,e)fluoranthene* (CAS No. 005385-75-1). Dibenzo(a,e)fluoranthene produced tumors in female mice after mouse-skin initiation-promotion assay and skin painting. Dibenzo(a,e)fluoranthene also produced tumors in both male and female mice after subcutaneous injection. EPA believes that there is sufficient evidence for listing dibenzo(a,e)fluoranthene on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for this chemical.

k. *Dibenzo(a,e)pyrene* (CAS No. 000192-65-4). Dibenzo(a,e)pyrene is classified as a Group 2B compound by IARC, i.e., the compound is a possible human carcinogen. Dibenzo(a,e)pyrene has been shown to be carcinogenic in animals. EPA believes that there is sufficient evidence for listing dibenzo(a,e)pyrene on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for this chemical.

l. *Dibenzo(a,h)pyrene* (CAS No. 000189-64-0). Dibenzo(a,h)pyrene is

classified as a Group 2B compound by IARC, i.e., the compound is a possible human carcinogen. Dibenzo(a,h)pyrene has been shown to be carcinogenic in animals. EPA believes that there is sufficient evidence for listing dibenzo(a,h)pyrene on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for this chemical.

m. *Dibenzo(a,l)pyrene* (CAS No. 000191-30-0). Dibenzo(a,l)pyrene is classified as a Group 2B compound by IARC, i.e., the compound is a possible human carcinogen. Dibenzo(a,l)pyrene produced tumors in both male and female mice after subcutaneous (s.c.) injection and tumors in female mice after skin painting. EPA believes that there is sufficient evidence for listing dibenzo-(a,l)pyrene on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for this chemical.

n. *7H-Dibenzo(c,g)carbazole* (CAS No. 000194-59-2). 7H-Dibenzo(c,g)carbazole is classified as a Group 2B compound by IARC, i.e., the compound is a possible human carcinogen. 7H-Dibenzo(c,g)carbazole has been shown to be carcinogenic in animals. EPA believes that there is sufficient evidence for listing 7H-dibenzo(c,g)carbazole on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for this chemical.

o. *2-Methylchrysene* (CAS No. 003351-32-4). In a skin painting assay and in a mouse-skin initiation-promotion assay, 2-methylchrysene produced tumors in female mice. EPA believes that there is sufficient evidence for listing 2-methylchrysene on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for this chemical.

p. *3-Methylchrysene* (CAS No. 003351-31-3). In a skin painting assay and in a mouse-skin initiation-promotion assay, 3-methylchrysene produced tumors in female mice. EPA believes that there is sufficient evidence for listing 3-methylchrysene on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for this chemical.

q. *4-Methylchrysene* (CAS No. 003351-30-2). In a skin painting assay and in a mouse-skin initiation-promotion assay, 4-methylchrysene produced tumors in female mice. EPA believes that there is sufficient evidence for listing 4-methylchrysene on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for this chemical.

r. *5-Methylchrysene* (CAS No. 003697-24-3). 5-Methylchrysene is classified as a Group 2B compound by IARC, i.e., the compound is a possible human carcinogen. In a skin-painting assay and in a mouse-skin initiation-promotion assay, 5-methylchrysene produced tumors in female mice. EPA believes that there is sufficient evidence for listing 5-methylchrysene on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for this chemical.

s. *6-Methylchrysene* (CAS No. 001705-85-7). In a skin painting assay and in a mouse-skin initiation-promotion assay, 6-methylchrysene produced tumors in female mice. EPA believes that there is sufficient evidence for listing 6-methylchrysene on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for this chemical.

t. *2-Methylfluoranthene* (CAS No. 033543-31-6). In a skin painting assay, 2-methylfluoranthene produced benign and malignant skin tumors in female mice. In a female mouse-skin initiation-promotion assay, 2-methylfluoranthene produced skin papillomas. EPA believes that there is sufficient evidence for listing 2-methylfluoranthene on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for this chemical.

u. *1-Nitropyrene* (CAS No. 005522-43-0). 1-Nitropyrene is classified as a Group 2B compound by IARC, i.e., the compound is a possible human carcinogen. 1-Nitropyrene produced mammary adenocarcinomas and squamous-cell carcinomas in a dose-dependent manner by oral administration in rats, papillomas (not statistically significant) by skin application in mice, and lung adenomas by intratracheal instillation in hamsters. In a s.c. injection study, 1-nitropyrene produced tumors (i.e., one extraskeletal osteosarcoma and seven malignant fibrous histiocytomas) at the injection site in male Fisher rats. In another s.c. injection study, 1-nitropyrene produced tumors at the injection site in both male and female CD rats and mammary tumors in females. EPA believes that there is sufficient evidence for listing 1-nitropyrene on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for this chemical.

In addition to the above compounds, EPA proposes that the PAC category also include the following seven PACs:

Benz(a)anthracene (CAS No. 000056-55-3)

Benzo(a)phenanthrene (CAS No. 000218-01-9)

Benzo(a)pyrene (CAS No. 000050-32-8)

Benzo(rst)pentaphene (CAS No. 000189-55-9)

Dibenzo(a,h)anthracene (CAS No. 000053-70-3)

7,12-Dimethylbenz(a)anthracene (CAS No. 000057-97-6)

Indeno[1,2,3-cd]pyrene (CAS No. 000193-39-5)

These PACs were proposed for listing individually in EPA's response to a petition to add certain chemicals that appear on the RCRA list of toxic wastes under 40 CFR 261.33(f) to EPCRA section 313 (57 FR 41020, September 8, 1992). These chemicals were proposed for addition based on the available carcinogenicity data. Due to the similarities of these seven PACs to the chemicals listed in Unit IV.B.207.a. through IV.B.207.u. of this preamble, EPA believes that these chemicals should be added to EPCRA section 313 as part of the delineated PAC category rather than listed individually.

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. When filing reports for chemical categories the releases are determined in the same manner as the thresholds. One report is filed for the category and all releases are reported on this form. In the case of the delimited PAC category, only the 28 chemicals listed above would be included for purposes of making the threshold determinations and in filing reports on releases.

The Clean Air Act Amendments section 112(b) Hazardous Air Pollutants list includes a listing for polycyclic organic matter (POM) that includes PACs. The definition given for the POM category is broad and chemically non-specific and may be delineated by test method. For the purpose of listing under EPCRA section 313, EPA considered the following more chemically-specific definition for a PAC category: "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 five-membered rings, a ring nitrogen, a ring sulfur, one or more attached nitro groups, or one or more attached alkyl groups." As an

alternative to the delimited category, EPA is proposing to add a PAC category based on this broad definition. Although this definition may include chemicals of low or no concern, it may be less of a burden for facilities to report their total PACs rather than trying to determine which and how much of the specific PACs covered by the delimited category they are producing and releasing. EPA requests comment on the addition of the delimited PACs category versus the alternative PAC category based on the broader definition.

208. *Potassium bromate* (CAS No. 007758-01-2) (IARC) (Ref. 8). IARC has assigned potassium bromate to Group 2B, i.e., it is possibly carcinogenic to humans. Male and female rats orally exposed to 250 or 500 ppm (35 to 70 mg/kg/day) potassium bromate in drinking water for 110 weeks had an increased incidence of renal cell adenomas and adenocarcinomas and, in males, there was also an increased incidence of mesothelioma in the peritoneal cavity. EPA believes that there is sufficient evidence for listing potassium bromate on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for this chemical.

209. *Potassium dimethyldithiocarbamate* (CAS No. 000128-03-0) (FIFRA AI) (Ref. 3). New Zealand White rabbits given 38 mg/kg/day by gavage on days 6 to 18 of gestation exhibited malalignment of sternebrae, total postimplantation loss, and fetal weight decrement. Also at this dose level, various malformations including adactyly, gastroschisis, short tail, anal atresia, spina bifida, atelectasis, costal cartilage anomaly, vertebral anomaly with/without rib, caudal vertebrea anomaly, and severe sternebrae malalignment were observed in 6 of 52 fetuses from 5 of 11 litters. At the 77 mg/kg/day dose level, there was severe fetal/embryo lethality. The NOEL was 12.8 mg/kg/day. EPA believes that there is sufficient evidence for listing potassium dimethyldithiocarbamate on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available developmental toxicity data for this chemical.

210. *Potassium N-methyldithiocarbamate* (CAS No. 000137-41-7) (FIFRA AI) (Ref. 3). By analogy to the analogue, potassium dimethyldithiocarbamate, potassium N-methyldithiocarbamate can reasonably be anticipated to cause fetotoxicity, postimplantation loss and malformations. Data on potassium dimethyldithiocarbamate follows. New Zealand White rabbits given 38 mg/kg/day by gavage on days 6 to 18 of gestation exhibited malalignment of sternebrae, total postimplantation loss, and fetal weight decrement. Also at this dose level various possible malformations including adactyly, gastroschisis, short tail, anal atresia, spina bifida, atelectasis, costal cartilage anomaly, vertebral anomaly with/without rib, caudal vertebrea anomaly, and severe sternebrae malalignment in 6 of 52 fetuses from 5 of 11 litters. At the 77 mg/kg/day dose level, there was severe fetal/embryo lethality. The NOEL was 12.8 mg/kg/day. EPA believes that there is sufficient evidence for listing potassium N-methyldithiocarbamate on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available developmental toxicity data for potassium dimethyldithiocarbamate.

211. *Primisulfuron (methyl 2-[[[[[4,6-bis(difluoromethoxy)2-pyrimidinyl]-amino] carbonyl] amino]sulfonyl] benzoate)* (CAS No. 086209-51-0) (FIFRA AI) (Ref. 3). In a 90–day dog feeding study, reduced thyroid weights accompanied by colloid depletion and parafollicular hyperplasia and anemia were observed at the LOEL of 25 mg/kg/day. The NOEL was 0.625 mg/kg/day. In a 1-year dog study, dietary administration of 250/125 mg/kg/day (LOEL: the dose was changed after week 10 in the study) produced thyroid hyperplasia, anemia, increased platelet levels, vacuolar changes, and increased absolute and relative liver weights. The NOEL was 25 mg/kg/day. In an 18–month study in mice, dietary administration of 1.7 mg/kg/day produced increased absolute and relative liver weights in females. No NOEL was established. Based on this study, an oral RfD of 0.006 mg/kg/day was derived. In a 2–year mouse study, increases in absolute and relative liver weights were observed at 408 mg/kg/day in males and 1.7 mg/kg/day in females. The systemic LOEL and NOEL in males was 408 mg/kg/day and 40.2 mg/kg/day, respectively. The systemic LOEL in females was 1.7 mg/kg/day and a NOEL could not be established. EPA believes that there is sufficient evidence for listing primisulfuron on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available thyroid and liver toxicity data for this chemical.

Plant toxicity values include a duckweed 14–day EC_{50} of 0.27 ppb and an algae 7–day EC_{50} of 24 ppb. EPA believes that there is sufficient evidence for listing primisulfuron on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data for this chemical.

212. *Profenofos (O-(4-bromo-2-chlorophenyl)-O-ethyl-Spropyl phosphorothioate)* (CAS No. 041198-08-7) (FIFRA AI) (Ref. 3). In a 6–month feeding study in dogs, inhibition of plasma and red blood cell cholinesterase activities were observed at 2 ppm (0.05 mg/kg/day). The NOEL was 0.2 ppm (0.005 mg/kg/day). Based on the NOEL, EPA derived an oral RfD of 0.00005 mg/kg/day. Other studies (21, 28, and 90–day studies in rat, rabbit and dog) also demonstrate cholinesterase (plasma, red blood cell or brain) inhibition in rats and mice. EPA believes that there is sufficient evidence for listing profenofos on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available neurological toxicity data.

213. *Prometryn (N,N'-bis(1-methylethyl)-6-methylthio-1,3,5-triazine-2,4-diamine)* (CAS No. 007287-19-6) (FIFRA AI) (Ref. 3). Degenerative changes in the liver and kidney, and bone marrow atrophy (the LOEL was 37.5 mg/kg/day; the NOEL was 3.75 mg/kg/day) were observed in dogs fed prometryn for 2 years. Based on the NOEL, EPA derived an oral RfD of 0.004 mg/kg/day. Fatty liver degeneration (the LOEL was 500 mg/kg; the NOEL was 250 mg/kg) was observed in rats fed prometryn for 28 days.

In a teratology study in rabbits, test material was administered by gavage from gestation day 7 to 19. Increased abortions and late resorptions occurred at 72 mg/kg/day. The NOEL was 12 mg/kg/day.

EPA believes that there is sufficient evidence for listing prometryn on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hepatic, renal, bone marrow, and developmental toxicity data.

214. *Propachlor (2-chloro-N-(1-methylethyl)-N-phenylacetamide)* (CAS No. 001918-16-7) (FIFRA AI) (Ref. 3). No evidence of maternal toxicity was seen in rabbits administered propachlor by gavage at 0, 5, 15, or 50 mg/kg/day on days 7 to 19 of gestation. Statistically significant increases in mean resorptions/postimplantation loss with corresponding decreases in the mean number of viable fetuses were reported at 15 and 50 mg/kg/day when compared to controls. EPA believes that there is sufficient evidence for listing propachlor on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available developmental toxicity data.

215. *Propanil (N-(3,4-dichlorophenyl)propanamide)* (CAS No. 000709-98-8) (FIFRA AI) (Ref. 3). Results of several subchronic and chronic toxicity studies indicated the

liver and spleen as the target organs for propanil. Increased relative spleen weight (the LOEL was 20 mg/kg/day; the NOEL was 5 mg/kg/day) was noted in female rats fed propanil for 2 years. Based on the NOEL, EPA derived an oral RfD of 0.005 mg/kg/day. Histopathological changes (the LOEL was 30 mg/kg/day; the NOEL was 25 mg/kg/day) in the liver and spleen were observed in mice orally administered propanil for 90 days. At higher dose levels (i.e., 240 and 1,920 mg/kg/day) cyanosis, methemoglobinemia, and increased liver and spleen weight were noted. In a 90–day rat study, increased spleen weight (the LOEL was 50 mg/kg/day; the NOEL was 16.5 mg/kg/day) was seen in females. Decreased hemoglobin levels was seen in males. Increased SGOT and SAP activities (the LOEL was 100 mg/kg/day; the NOEL was 15 mg/kg/day) were observed in dogs orally administered propanil for 2 years. EPA believes that there is sufficient evidence for listing propanil on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hepatic toxicity data.

216. *Propargite* (CAS No. 002312-35-8) (CERCLA) (Ref. 8). In a developmental toxicity study in which rabbits were exposed via oral gavage to doses greater than or equal to 6 mg/kg/day (fetotoxic LOAEL) of propargite during gestation days 6 to 18, delayed ossification, increased fetal resorption, decreased fetal viability and reductions in fetal body weight were noted. The maternal LOAEL in this study was also 6 mg/kg/day and was based on body weight reductions. The NOEL for maternal and fetal toxicity was 2 mg/kg/day. Developmental effects (increased incidence of missing sternebrae) were also reported in offspring of rats exposed orally during gestation days 6 to 15. The fetotoxicity LOAEL was 25 mg/kg/day and the NOAEL was 6 mg/kg/day. EPA believes that there is sufficient evidence for listing propargite on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the developmental toxicity data for this chemical.

Measured aquatic acute toxicity data for propargite include a bluegill sunfish LC_{50} of 31 ppb. EPA believes that there is sufficient evidence for listing propargite on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the environmental toxicity data for this chemical.

217. *Propargyl alcohol* (CAS No. 000107-19-7) (CERCLA; RCRA APP8; RCRA P) (Ref. 8). Histopathological changes in the liver and kidney were reported in a subchronic rat feeding study following exposure to propargyl alcohol in the diet for as little as 4 weeks. The liver changes included increased organ weight, hepatocytic megalocytosis with proliferation of bile ducts and cytoplasmic vacuolization of hepatocytes, as well as hematological and serum enzyme changes indicative of liver damage. The kidney weights were increased in females only, and both sexes had karyomegaly of the renal tubular epithelial cells. The LOAEL for these changes was 15 mg/kg/day and the NOAEL was 5 mg/kg/day. EPA derived an oral RfD of 0.002 mg/kg/day from this study. EPA believes that there is sufficient evidence for listing propargyl alcohol on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the hepatotoxicity and nephrotoxicity data for this chemical.

218. *Propetamphos (3-[[(Ethylamino)methoxyphosphinothioyl] oxy]-2-butenoic acid, 1-methylethyl ester)* (CAS No. 031218-83-4) (FIFRA AI) (Ref. 3). Purebred beagle dogs were given propetamphos for 52 weeks in feed. A dose of 2.5 mg/kg/day caused increased relative liver weight and increased liver enzymes. Dogs given 12.5 mg/kg/day developed hepatocellular necrosis. The NOEL was 0.5 mg/kg/day.

Red blood cell and plasma cholinesterase inhibition were seen in a 2-week rat inhalation study at 1 mg/kg/day (LOEL). No NOEL could be established. Cholinesterase inhibition was observed at 0.4 mg/kg/day in a 13–week rat dietary study. The NOEL was 0.2 mg/kg/day. Cholinesterase inhibition was also observed at 0.1 mg/kg/day (LOEL) in a 6–month dog dietary study. The NOEL was 0.05 mg/kg/day. In a 92–week mouse feeding study, red blood cell, brain, and plasma cholinesterase were inhibited at 1.0 mg/kg/day (LOEL). The NOEL was 0.5 mg/kg/day. Based on this study, an oral RfD of 0.005 mg/kg/day was derived. In a 2–year dietary rat study, plasma cholinesterase depression was observed at 0.6 mg/kg/day (LOEL). The cholinesterase NOEL was 0.3 mg/kg/day. Alopecia and hyperflexia were observed at 6 mg/kg/day (systemic LOEL). The systemic NOEL was 0.6 mg/kg/day. In a lifetime mouse study, dietary administration of 1 mg/kg/day produced plasma, red blood cell, liver, and brain cholinesterase depression. The NOEL was 0.05 mg/kg/day.

EPA believes that there is sufficient evidence for listing propetamphos on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hepatic and neurological toxicity data for this chemical.

219. *Propiconazole (1-[2-(2,4-dichlorophenyl)-4-propyl-1,3-dioxolan-2-yl]- methyl-1H-1,2,4,-triazole)* (CAS No. 060207-90-1) (FIFRA AI) (Ref. 3). In a 2-generation rat reproduction study, dietary administration of 25 mg/kg/day produced an increased incidence of hepatic clear cell change in parental animals and administration of 125 mg/kg/day produced an increased incidence of hepatic lesions in offspring. The parental NOEL was 5 mg/kg/day and the developmental NOEL was 25 mg/kg/day. In a 2–year mouse study, dietary administration of 65 mg/kg/day (LOEL) produced increased liver lesions and liver weight in males, whereas, administration of 325 mg/kg/day produced increased liver tumors, increased SGPT and SGOT levels, increased liver weight, hepatocyte enlargement, and vacuolation and fat deposition in the liver of both sexes. The NOEL was 13 mg/kg/day.

In a 3–month dog dietary study, lymphoid follicles were observed in the mucous membranes of the pyloric part of the stomach at 6.25 mg/kg/day. The NOEL was 1.25 mg/kg/day. In a 1–year dog study, dietary administration of 6.25 mg/kg/day produced mild gastric mucosal irritation. The NOEL was 1.25 mg/kg/day. Based on the NOEL of the study, an oral RfD of 0.013 mg/kg/day was derived.

EPA believes that there is sufficient evidence for listing propiconazole on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hepatic and gastrointestinal toxicity data for this chemical.

220. *Quizalofop-ethyl (2-[4-[(6-chloro-2-quinoxalinyl) oxy]phenoxy] propanoic acid ethyl ester)* (CAS No. 076578-14-8) (FIFRA AI) (Ref. 3). In a 3–month rat study, dietary administration of 6.4 mg/kg/day produced changes in liver weight and liver lesions. The NOEL was 2 mg/kg/day. In a 6–month dietary dog study, 10 mg/kg/day produced testicular atrophy in males. The NOEL was 2.5 mg/kg/day. Liver cell enlargement was observed at 3.7 mg/kg/day in males and 4.6 mg/kg/day in females (LOELs) in a 2–year rat dietary study. The NOELs for males and females were 0.9 mg/kg/day and 1.1 mg/kg/day, respectively. Based on the study, an oral RfD of 0.009 mg/kg/day was derived. Increased liver weights were observed in pregnant rats in a teratology study. The maternal LOEL was 100 mg/kg/day and the NOEL was 30 mg/kg/day. No teratogenic NOEL could be established. In a 2-generation rat reproduction study, increased liver weights and increased incidence of eosinophilic changes in the liver were observed in the offspring at 5 mg/kg/day (LOEL). The NOEL was 1.25 mg/kg/day.

EPA believes that there is sufficient evidence for listing quizalofop-ethyl on

EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available reproductive and hepatic toxicity data for this chemical.

221. *Resmethrin ([5-(phenylmethyl)-3-furanyl]methyl 2,2-dimethyl-3-(2-methyl-1-propenyl) cyclopropanecarboxylate)* (CAS No. 010453-86-8) (FIFRA AI) (Ref. 3). Oral administration of 30 mg/kg/day (LOEL) in capsules for 6 months produced increases in liver weights in female dogs. The NOEL was 10 mg/kg/day. In a 2–year rat study, dietary administration of 125 mg/kg/day produced increases in liver weight and pathological lesions. The NOEL was 25 mg/kg/day.

In a one-generation reproduction rat study, administration of 25 mg/kg/day (LOEL) in the diet produced an increase in dead pups and lower pup weight among survivors. No NOEL could be established. In a 3-generation reproduction rat study, dietary administration of 25 mg/kg/day (LOEL) produced an increase in pups cast dead and lower pup weight among survivors. No NOEL could be established. Based on the NOEL of the study, an oral RfD of 0.03 mg/kg/day was derived.

Signs of neurotoxicity, including piloerection, ataxia, sensory changes in peripheral nerves, changes in locomotor activity, salivation, tremors, and convulsions were observed in rats, dogs, mice, and rabbits given acute oral, intravenous or intraperitoneal injections greater than or equal to 160 mg/kg. In a 3–month rat inhalation study, 0.1 mg/L (LOEL) produced behavioral effects and 1 mg/L produced decreased locomotor activity, tremors, and other behavioral changes. No NOEL could be established.

EPA believes that there is sufficient evidence for listing resmethrin on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hepatic, reproductive, and neurological toxicity data for this chemical.

Aquatic acute toxicity values for resmethrin include a rainbow trout 96–hour LC_{50} of 0.275 ppb (89 percent a.i.), a bluegill sunfish 96–hour LC_{50} of 0.750 ppb (89 percent a.i.), a lake trout 96–hour LC_{50} of 1.7 ppb (84.5 percent a.i.), and a fathead minnow 96–hour LC_{50} of 3.0 ppb. EPA believes that there is sufficient evidence for listing resmethrin on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data for this chemical.

222. *Sethoxydim (2-[1-(ethoxyimino)butyl]-5-[2(ethylthio)propyl]-3-hydroxy-2-cyclohexen-1-one)* (CAS No. 074051-80-2) (FIFRA AI) (Ref. 3). Mild anemia (the LOEL was 17.5 mg/kg/day; the NOEL was 8.9 mg/kg/day) was observed in male dogs fed sethoxydim for 1–year. Based on the NOEL, EPA derived an oral RfD of 0.09 mg/kg/day. Swollen liver cells (the LOEL was 117 mg/kg/day; the NOEL was 45 mg/kg/day) were seen in mice fed sethoxydim for 14 weeks. Pathological effects in the liver (the LOEL was 45 mg/kg/day; the NOEL was 15 mg/kg/day) were noted in rats fed sethoxydim for 14 weeks. Nonneoplastic liver lesions (the LOEL was 54 mg/kg/day; the NOEL was 18 mg/kg/day) were observed in mice fed sethoxydim for 2 years. Decreased phenosulfophthalein (PSP) clearance (the NOEL was greater than 3 mg/kg/day; the LOEL not determined) was noted in dogs given sethoxydim in the diet for 26 weeks. Decreased PSP clearance (the LOEL was 20 mg/kg/day; the NOEL was 2 mg/kg/day) was also noted in a 6–month feeding study in dogs. EPA believes that there is sufficient evidence for listing sethoxydim on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hematological, hepatic, and renal toxicity data.

223. *Simazine* (CAS No. 000122-34-9) (FIFRA SR; SDWA) (Ref. 8). Simazine is a triazine-type herbicide. Chronic exposure of sheep to low doses (approximately 1.4 to 6 mg/kg/day) of simazine caused fatty and granular degeneration in the liver, and increased SGOT and alkaline phosphatase. Neuronophagia, diffuse kidney degeneration, diffuse glial proliferation and degeneration of ganglion cells in the cerebrum and medulla were also reported in these animals. Dogs that received 1,500 ppm (37.5 mg/kg/day) simazine in a 2–year feeding study also had slight increases in serum alkaline phosphatase and SGOT, indicative of liver damage.

Sheep that received 1.4 mg/kg/day simazine for 37 to 111 days had necrotic changes in the germinal epithelium of the testis and disturbances in spermatogenesis.

EPA believes that there is sufficient evidence for listing simazine on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the hepatic, renal, neurological, and reproductive toxicity of this chemical.

224. *Sodium azide* (CAS No. 026628-22-8) (CERCLA; EPCRA EHS; RCRA P) (Ref. 8). Although not used clinically, sodium azide is a direct acting vasodilator. A reduction in blood pressure was noted in hypertensive patients orally exposed to sodium azide during an investigation of the substance in treating cancer. Reductions in blood pressure were also reported in animals following acute exposure. The minimal hypotensive dose in humans has been estimated to be approximately 0.2 to 0.4 µg/kg (0.0002 to 0.0004 mg/kg). EPA believes that there is sufficient evidence for listing sodium azide on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the ability of this substance to lower blood pressure.

225. *Sodium chlorite* (CAS No. 007758-19-2) (FIFRA AI) (Ref. 3). A decrease in erythrocyte half-life (the LOEL was 100 ppm or 7.3 mg/kg/day; the NOEL was 50 ppm or 3.65 mg/kg/day) was observed in cats administered sodium chlorite in the drinking water for 90 days. Increase in glucose-6-phosphatase dehydrogenase activity, mean corpuscular volume (MCV), osmotic fragility, and acanthocytes were observed in mice administered 100 ppm (19 mg/kg/day) in the drinking water for 30 days. In another 30–day drinking water study, increased glucose-6-phosphatase dehydrogenase activity, MCV, and osmotic fragility were noted in mice administered 100 ppm (19 mg/kg/day). The NOEL was 1.9 mg/kg/day. The results of in vitro studies show that sodium chlorite can result in oxidative damage to erythrocytes. EPA believes that there is sufficient evidence for listing sodium chlorite on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hematological toxicity data.

226. *Sodium dicamba (3,6-Dichloro-2-methoxybenzoic acid, sodium salt)* (CAS No. 001982-69-0) (FIFRA AI) (Ref. 3). No toxicity data are available for sodium dicamba. However, data are available on dicamba as discussed below. In solution, sodium dicamba will dissociate into sodium ion and the dicamba anion. Decreased fetal body weights and increased postimplantation loss were observed in the offspring of rabbits receiving 10 mg/kg/day on days 6 through 18 of gestation. The LOEL was 10 mg/kg/day and NOEL was 3 mg/kg/day. Based on the NOEL, EPA derived an oral RfD value of 0.03 mg/kg/day. In a separate study, disorders of oxidative phosphorylation and focal necrosis in the heart were observed in newborn rats following transplacental exposure to dicamba. In a developmental toxicity study, an increase in skeletal malformations was seen in the offspring of rats orally administered 64 mg/kg/day on days 6 through 19 of gestation. EPA believes that there is sufficient evidence for listing sodium dicamba on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available developmental toxicity data for dicamba.

227. *Sodium dimethyldithiocarbamate* (CAS No. 000128-04-1) (FIFRA AI) (Ref. 3). By analogy to potassium dimethyldithiocarbamate, sodium dimethyldithiocarbamate can reasonably be anticipated to cause fetotoxicity, postimplantation loss and malformations. Data on potassium dimethyldithiocarbamate follows. New Zealand white rabbits given 38 mg/kg/day by gavage on days 6 to 18 of gestation exhibited malalignment of sternebrae, total postimplantation loss, and fetal weight decrement. Also at this dose level, various possible malformations including adactyly, gastroschisis, short tail, anal atresia, spina bifida, atelectasis, costal cartilage anomaly, vertebral anomaly with/without rib, caudal vertebrea anomaly, and severe sternebrae malalignment in 6 of 52 fetuses from 5 of 11 litters. At the 77 mg/kg/day dose level, there was severe fetal/embryo lethality. The NOEL was 12.8 mg/kg/day. EPA believes that there is sufficient evidence for listing sodium dimethyldithiocarbamate on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available developmental toxicity data for the analogue potassium dimethyldithiocarbamate.

228. *Sodium fluoroacetate* (CAS. No. 000062-74-8) (CERCLA; EPCRA EHS; FIFRA SR; RCRA APP8; RCRA P) (Ref. 8). In a 13–week oral study in rats, gavage administration of sodium fluoroacetate (0.02 mg/kg/day) resulted in decreased testis weight and altered spermatogenesis in males (the NOAEL was 0.05 mg/kg/day). In addition, increased heart weight was noted in females and males administered 0.20 mg/kg/day of sodium fluoroacetate. The increase in heart weight, however, was only accompanied by subacute, minimal inflammation (not dose-related). Also, fluorocitrate levels were significantly increased after 4 weeks in males administered 0.50 mg/kg/day and after 13 weeks in both male and female rats administered 0.20 or 0.50 mg/kg/day. The testicular and cardiac effects were reported to be consistent with those noted in the literature.

A case study reported a deliberate ingestion of an unspecified dose of sodium fluroacetate by a healthy female. The woman experienced nausea, vomiting, and abdominal pain 30 minutes after ingestion, with subsequent seizures occurring 60 minutes after the initial onset of symptoms. Neurological examination after 2 weeks revealed severe cerebellar dysfunction. By 18 months, memory disturbances and depressive behavior persisted. Inhalation exposure to unspecified levels of sodium fluoroacetate caused salivation, loss of speech, violent convulsions, and coma in a male worker. The patient ultimately recovered. Neurological effects have also been reported in rats in a 13–week oral study. Four of 20 female rats treated with 0.50 mg/kg/day (the highest dose tested) exhibited convulsions at day 79, with no recurrences for the remainder of the study. An estimated lethal dose of sodium fluoroacetate in humans ranges from 5 to 10 mg/kg.

EPA believes that there is sufficient evidence for listing sodium fluoroacetate on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the neurologic, reproductive, and myocardial toxicity data for this chemical.

Measured oral LD_{50} values of fluoroacetate in the house sparrow, redwinged blackbird, starling and golden eagle are 3.0, 4.22, 2.37, and 1.25 to 5 mg/kg, respectively. In addition, measured acute toxicity data for mammalian wildlife include an oral LD_{50} of 0.22 to 0.44 mg/kg for mule deer, an oral LD_{50} of 1.41 mg/kg for male ferrets, and an oral LD_{50} of 0.5 to 1.0 mg/kg for bears. EPA believes that there is sufficient evidence for listing sodium fluoroacetate on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the environmental toxicity data for this chemical.

229. *Sodium hypochlorite* (CAS No. 007681-52-9) (CERCLA) (Ref. 8). Aquatic acute toxicity data for sodium hypochlorite include a 96–hour measured LC_{50} of 100 ppb for bluegill and a 96–hour measured LC_{50} of 80 ppb for fathead minnow. In addition, the 96–hour measured LC_{50} values for non-standard test species range from 32 ppb for coho salmon to 82 ppb for Pacific sand lance. EPA believes that there is sufficient evidence for listing sodium hypochlorite on EPCRA section 313(d)(2)(C) based on the available ecotoxicity data for this chemical.

230. *Sodium nitrite* (CAS No. 007632-00-0) (CERCLA) (Ref. 8). Sodium nitrite causes conversion (oxidation) of hemoglobin to methemoglobin. Methemoglobin cannot combine reversibly with oxygen and its formation can cause anemic hypoxia which may lead to intense cyanosis. Infants are particularly susceptible to this effect because of their higher stomach pH, immature enzyme systems, the reduced capacity of newborn erythrocytes to reduce methemoglobin to hemoglobin, and the increased rate of nitrite-induced oxidation of fetal hemoglobin to methemoglobin (approximately twice the rate of adult hemoglobin oxidation). Coma and methemoglobinemia/carboxyhemoglobinemia were reported in a human that received sodium nitrite (71 mg/kg) orally. In animal studies, methemoglobinemia was reported in dogs that received an intravenous dose of 30 mg/kg sodium nitrite and in rats administered a 10 mg/kg dose of sodium nitrite subcutaneously.

Fetotoxicity (fetal death) was reported following oral exposure of pregnant rats to sodium nitrite (30 mg/kg/day) during gestation days 1 through 22. In mice exposed orally to 80 mg/kg/day during gestation days 6 to 15 there was increased preimplantation loss and fetal death, and in mice exposed to a lower dose (20 mg/kg/day) during gestation days 1 to 14, abnormalities of the blood or lymphatic system were reported in offspring. In offspring of rats orally exposed to 26 to 256 mg/kg/day during pregnancy (gestation days 1 through 22) and/or during lactation (20 to 21 days after birth), effects on growth including biochemical and/or metabolic changes were noted.

EPA believes that there is sufficient evidence for listing sodium nitrite on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available chronic hematological and developmental toxicity data for this chemical.

231. *Sodium pentachlorophenate* (CAS No. 000131-52-2) (FIFRA AI) (Ref. 3). Pentachlorophenol has been classified by EPA as a Group B2 compound, i.e., a probable human carcinogen. This was based on occurrence of increased combined incidence of hemangiosarcomas, liver tumors, and pheochromocytomas in female mice. EPA believes that there is sufficient evidence for listing sodium pentachlorophenate on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for its parent compound, pentachlorophenol.

Aquatic acute toxicity values for sodium pentachlorophenate include a rainbow trout 96–hour LC_{50} of 55 ppb, a bluegill 96–hour LC_{50} of 44 ppb, a fathead minnow 96–hour LC_{50} of 20 ppb, and a shrimp 96–hour LC_{50} of 84 ppb. EPA believes that there is sufficient evidence for listing sodium pentachlorophenate on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data for this chemical.

232. *Sodium o-phenylphenoxide* (CAS No. 000132-27-4) (CERCLA; IARC) (Ref. 8). Sodium o-phenylphenoxide has been classified by IARC as a Group 2B compound; i.e., the substance is possibly carcinogenic in humans. EPA believes that there is sufficient evidence

for listing sodium o-phenylphenoxide on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the carcinogenicity data for this chemical.

233. *Sodium 2-pyridinethiol-1-oxide* (CAS No. 015922-78-8) (FIFRA AI) (Ref. 3). New Zealand white rabbits were tested with test material dermally on days 6 to 18 of gestation. At 0.5 mg/kg/day, pups exhibited missing or defective vertebrae, ribs and sternebrae. No NOEL was established. EPA believes that there is sufficient evidence for listing sodium 2-pyridinethiol-1-oxide on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available developmental toxicity data for this chemical.

234. *Strychnine and salts* (CERCLA; EPCRA EHS; FIFRA SR; RCRA APP8; RCRA P) (Ref. 8). Strychnine salts will dissociate in aqueous solutions to yield soluble strychnine. Strychnine, an alkaloid, can cause violent convulsions in humans. Other effects include agitation, hypertonicity of muscles, and painful muscle spasms. Renal failure and respiratory paralysis generally ensues, from severe or prolonged convulsions. A potentially lethal oral dose in a small child is 5 to 10 mg. The lethal oral dose for an adult may be as low as 30 mg. Similar effects have also been reported in animals exposed at lethal doses ranging from 0.25 to 2.35 mg/kg via oral and parenteral routes of exposure. EPA's exposure analysis indicates that strychnine and strychnine salts concentrations are likely to exist beyond facility site boundaries, as a result of continuous, or frequently recurring releases, at levels that can reasonably be anticipated to cause significant adverse acute human health effects. EPA believes that there is sufficient evidence for listing strychnine and salts as a category on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(A) based on the available acute toxicity and exposure data for this chemical.

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. When filing reports for chemical categories, the releases are determined in the same manner as the thresholds. One report if filed for the category and all releases are reported on this form.

235. *Sulfur dioxide* (CAS No. 007446-09-5) (CERCLA; EPCRA EHS) (Ref. 8). Acid precipitation occurs in large regions of the Eastern United States and Canada, Europe, and Japan. This widespread occurrence of acid precipitation and dry deposition results in large part from man-made emissions of oxides of sulfur (e.g., sulfur dioxide) and oxides of nitrogen. These substances are transformed in the atmosphere into sulfuric acid and nitric acid, transported over great distances and deposited on vegetation, soils, surface waters, and materials. These substances are transferred from the atmosphere into ecosystems by the absorption of gases, the impaction and gravitational settling of fine aerosols and coarse particles and, precipitation.

Acids contained in polluted snow are released as contaminated meltwater. The resulting release of pollutants can cause major or rapid changes in the acidity of streams and lake waters. Interference with normal reproduction in fish populations is induced by acidity of lake and stream waters. Reproduction of frogs and salamanders is also inhibited by atmospheric acidification of surface waters.

Atmospheric deposition of sulfuric acid and nitric acid can cause serious damage to crops and forests. Biological effects include induction of necrotic lesions, loss of nutrients due to leaching from foliar organs, accelerated erosion of waxes and leaf surfaces, and interference with normal reproductive processes. Acidification decreases the rate of many soil processes such as nitrogen fixation and the breakdown of organic matter.

EPA believes that there is sufficient evidence for listing sulfur dioxide on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data for this chemical.

Limited data on long-term human exposure to sulfuric acid with respect to occupational settings are available. Recent studies suggest that sulfuric acid aerosols at levels as low as 0.02 to 0.04 mg/m^3 may cause significant effects on lung function in humans. Effects noted include increased risk of chronic bronchitis in smokers and reduced tracheobronchial clearance rate. Other studies suggest that sulfuric acid at concentrations as low as 0.04 mg/m^3 may act synergistically with copollutants such as ozone, NO_2, and metal particulates in causing decreased pulmonary diffusing capacity and bronchial hypersensitivity. These effects are presumably attributable to the acidic and oxidative properties of sulfuric acid, and are therefore pH and concentration dependent. EPA believes that there is sufficient evidence for listing sulfur dioxide on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available chronic toxicity data for sulfuric acid, the hydrolysis product of sulfur dioxide.

Sulfur dioxide is regulated under Title I of the CAA (Provisions for Attainment and Maintenance of National Ambient Air Quality Standards) and Title IV of the CAA (Acid Deposition Control). In addition to this proposal to add sulfur dioxide to EPCRA section 313, in Units IV.B.36. and 179, EPA is proposing to add two other chemicals, carbon monoxide and nitrogen dioxide, that are regulated under Title I of the CAA. Extensive data, which are highly technical, are collected on these chemicals as required by the CAA. EPA requests comment on the following: (1) Is the information collected under the CAA sufficient for public right-to-know purposes; and (2) suggestions on how the data collected on these chemicals pursuant to CAA Titles I and IV could be used to meet the purposes of EPCRA section 313.

236. *Sulfur trioxide* (CAS No. 007446-11-9) (EPCRA EHS) (Ref. 8). IARC has classified sulfur trioxide in Group 1, i.e., the chemical is carcinogenic to humans based on sufficient evidence of carcinogenicity in humans. EPA believes that there is sufficient evidence for listing sulfur trioxide on EPCRA section 313 pursuant to section 313(d)(2)(B) based on the carcinogenicity data for this chemical.

Acid precipitation occurs in large regions of the Eastern United States and Canada, Europe, and Japan. This widespread occurrence of acid precipitation and dry deposition results in large part from man-made emissions of oxides of sulfur (e.g., sulfur trioxide) and oxides of nitrogen. These substances are transformed in the atmosphere into sulfuric acid and nitric acid, transported over great distances and deposited on vegetation, soils, surface water, and materials. These substances are transferred from the atmosphere into ecosystems by the absorption of gases, the impaction and gravitational settling of fine aerosols and coarse particles and, precipitation.

Acids contained in polluted snow are released as contaminated meltwater. The resulting release of pollutants can cause major or rapid changes in the acidity of streams and lake waters. Interference with normal reproduction in fish populations is induced by acidity of lake and stream waters. Reproduction of frogs and salamanders is also inhibited by atmospheric acidification of surface waters.

Atmospheric deposition of sulfuric acid and nitric acid can cause serious damage to crops and forests. Biological effects include induction of necrotic lesions, loss of nutrients due to leaching from foliar organs, accelerated erosion of waxes and leaf surfaces, and interference with normal reproductive processes. Acidification decreases the rate of many soil processes such as nitrogen fixation and the breakdown of organic matter.

EPA believes that there is sufficient evidence for listing sulfur trioxide on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data for this chemical.

Limited data on long-term human exposure to sulfuric acid with respect to occupational settings are available. Recent studies suggest that sulfuric acid aerosols at levels as low as 0.02 to 0.04 mg/m^3 may cause significant effects on lung function in humans. Effects noted include increased risk of chronic bronchitis in smokers and reduced tracheobronchial clearance rate. Other studies suggest that sulfuric acid at concentrations as low as 0.04 mg/m^3 may act synergistically with copollutants such as ozone, NO_2, and metal particulates in causing decreased pulmonary diffusing capacity and bronchial hypersensitivity. These effects are presumably attributable to the acidic and oxidative properties of sulfuric acid, and are therefore pH and concentration dependent. EPA believes that there is sufficient evidence for listing sulfur trioxide on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available chronic toxicity data for sulfuric acid, the hydrolysis product of sulfur trioxide.

237. *Sulfuryl fluoride (Vikane)* (CAS No. 002699-79-8) (FIFRA AI) (Ref. 3). The primary effects of sulfuryl fluoride in humans are respiratory irritation and central nervous system depression, followed by excitation and possibly convulsions. Rabbits exposed via inhalation (6 hours/day, 5 days/week, for 2 weeks) to sulfuryl fluoride showed hyperactivity, convulsions and vacuolation of the cerebrum at 600 ppm (2.5 mg/L). Renal lesions were present in all rats exposed by inhalation (6 hours/day, 5 days/week, for 2 weeks) to 600 ppm (2.5 mg/L) sulfuryl fluoride. Minimal renal changes were noted in rats exposed to 300 ppm (1252 mg/L), whereas no effects occurred at 100 ppm (4.2 mg/L). Convulsions at near lethal concentrations were reported in rabbits, mice, and rats. In a 30-day inhalation study, loss of control, tremors of the hind quarters, and histopathological changes in the lung, liver, and kidney were reported in rabbits exposed to 400 ppm (1.6 mg/L) for 7 hours/day, 5 days/week for 5 weeks. The NOEL was 200 ppm (0.83 mg/L). Cerebral vacuolation and/or malacia and inflammation of nasal tissues were observed in rabbits exposed by inhalation to 100 or 300 ppm (0.4 or 1.25 mg/L) for 13 weeks. The NOEL was 30 ppm (0.125 mg/L). Rats exposed by inhalation to 100 to 600 ppm (0.4 to 0.25 mg/L) sulfuryl fluoride for 13 weeks developed mottled teeth (indicative of fluoride toxicity), renal and respiratory effects, and cerebral vacuolation. EPA believes that there is sufficient evidence for listing sulfuryl fluoride on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available neurological, renal, and respiratory toxicity data for this chemical.

238. *Sulprofos (O-Ethyl O-[4-(methylthio)phenyl] phosphorodithioic acid S-propyl ester)* (CAS No. 035400-43-2) (FIFRA AI) (Ref. 3). The acute dermal rabbit LD_{50} is between 745 mg/kg and 994 mg/kg. Ataxia, tremors, and diarrhea were observed. In a 28–day dietary study, administration of 1 mg/kg/day produced decreased red blood cell and brain cholinesterase activity. The NOEL was 0.1 mg/kg. Dietary administration of 15 mg/kg/day for 3 months produced hyperactivity in female rats. The NOEL was 5 mg/kg/day. In the same study, 5 mg/kg/day produced red blood cell and brain cholinesterase inhibition in both sexes. The cholinesterase NOEL was 1.5 mg/kg/day. Red blood cell and brain cholinesterase inhibition, diarrhea, vomiting, and some hind limb paralysis were seen in dogs orally administered 5 mg/kg/day (LOEL) for 3 months. The NOEL was 0.5 mg/kg/day. In a 22–month dietary mouse study, plasma and red blood cell cholinesterase were inhibited at 3.25 mg/kg/day. The NOEL was 0.325 mg/kg/day. Plasma, red blood cell, and brain cholinesterase inhibition were seen at a dietary administration of 2.5 mg/kg/day (LOEL) in a 2–year dog study. The NOEL was 0.25 mg/kg/day. Based on this study, an oral RfD of 0.003 mg/kg/day was derived. Dietary administration of 3 mg/kg/day (LOEL) produced plasma and red blood cell cholinesterase depression in a 2–year rat study. The NOEL was 0.3 mg/kg/day.

Increased unossified sternebrae were observed in the offspring of rats given 10 mg/kg/day (LOEL) by gavage during days 6 to 15 of gestation. No NOEL was established.

EPA believes that there is sufficient evidence for listing sulprofos on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available neurological and developmental toxicity data for this chemical.

The aquatic acute values for sulprofos include bluegill 96–hour LC_{50} value of 1.03 ppm and 11 ppm (technical product). The channel catfish bioconcentration factor for whole fish is 704 to 1006. EPA believes that there is sufficient evidence for listing sulprofos on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data and the potential for bioconcentration.

239. *Tebuthiuron (N-[5-(1,1-Dimethylethyl)-1,3,4-thiadiazol-2-yl)-N,N'-dimethylurea)* (CAS No. 034014-18-1) (FIFRA AI) (Ref. 3). Administration of 25 mg/kg/day (LOEL) on days 6 through 18 of gestation produced reduced body weights in offspring of rabbits. The NOEL was 10 mg/kg/day. In a 3–month rat study, dietary administration of 125 mg/kg/day (LOEL) produced growth suppression and pancreatic lesions. The NOEL was 50 mg/kg/day. In a 2-generation rat reproduction study, depressed body weight gain was observed in the female parental generation at 14 mg/kg/day. The NOEL was 7 mg/kg/day. Based on the NOEL of the study, an oral RfD of 0.07 mg/kg/day was derived. In a 3-generation rat reproduction study, decreased body weight was observed in the offspring of animals administered 20 mg/kg/day (LOEL). No NOEL was established. Dietary administration of 40 mg/kg/day to rats for 2 years produced growth suppression. The NOEL was 20 mg/kg/day. EPA believes that there is sufficient evidence for listing tebuthiuron on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available developmental toxicity data for this chemical.

240. *Tefluthrin* (CAS No. 079538-32-2) (FIFRA AI) (Ref. 3). Delayed ossification was seen in the offspring of rats administered 5 mg/kg/day (LOEL) orally on days 7 through 16 of gestation. The NOEL was 3 mg/kg/day.

In a 3–month rat study, dietary administration of 10 mg/kg/day produced plasma, red blood cell, and brain cholinesterase inhibition. The NOEL was 5 mg/kg/day. In a 6–month dog study, dietary administration of 10 mg/kg/day (LOEL) produced plasma cholinesterase inhibition. The NOEL was 1 mg/kg/day.

In a 21-day rat dietary study, administration of 20 mg/kg/day (LOEL for females) produced decreased platelet counts, increased white blood cell, lymphocyte, and neutrophil counts in males and females. The NOEL for females was 5 mg/kg/day. Increased absolute and relative liver weights were

observed at 5 mg/kg/day in males, thus no NOEL could be established for males. Dietary administration of 10 mg/kg/day (LOEL) for 3 months to rats produced increased absolute liver weights, decreased bilirubin levels, and hepatocellular hypertrophy. The NOEL was 5 mg/kg/day. In a 6–month dog study, dietary administration of 10 mg/kg/day (LOEL) produced hepatotoxicity (effects not reported). The NOEL was 1 mg/kg/day. In a 2–year mouse study, dietary administration of 13.5 mg/kg/day produced liver necrosis. The NOEL was 3.4 mg/kg/day.

EPA believes that there is sufficient evidence for listing tefluthrin on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available developmental, neurological, hepatic, and hematological toxicity data for this chemical.

Aquatic acute toxicity values for tefluthrin include a rainbow trout 96–hour LC_{50} of 0.06 ppb, a bluegill 96–hour LC_{50} of 0.13 ppb, a sheepshead minnow 96–hour LC_{50} of 0.13 ppb, a daphnid 48–hour EC_{50} of 0.07 ppb, and a mysid 96–hour EC_{50} of 0.053 ppb. EPA believes that there is sufficient evidence for listing teflurin on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data for this chemical.

241. *Temephos* (CAS No. 003383-96-8) (FIFRA AI) (Ref. 3). Temephos is a cholinesterase inhibitor in many mammalian species. The LOELs at which the cholinesterase inhibition was observed ranged from 0.3 to 10 mg/kg/day. However, human subjects that ingested 256 mg/day for 5 days or 64 mg/day for 4 weeks showed no clinical signs or effects on plasma or red blood cell cholinesterase activities. Dietary exposure of rats to 350 mg/kg/day for 90 days resulted in cholinesterase inhibition only; no clinical signs were reported. Rabbits and guinea pigs tolerated 10 mg/kg/day for extended periods without clinical effects, and dogs tolerated 3 to 4 mg/kg/day, the highest dose tested. EPA believes that there is sufficient evidence for listing temephos on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available neurological toxicity data.

242. *Terbacil (5-chloro-3-(1,1-dimethylethyl)-6-methyl- 2,4-(1H,3H)-pyrimidinedione)* (CAS No. 005902-51-2) (FIFRA AI) (Ref. 3). Decreases in the number of implantations and live fetuses, were observed in rats administered 62.5 mg/kg/day (LOEL) orally for days 6 to 15. The NOEL was 12.5 mg/kg/day. Significantly reduced body weights were observed in the offspring of rabbits orally administered 600 mg/kg/day (LOEL) orally on days 6 to 18 of gestation. The NOEL was 200 mg/kg/day.

In a 2–week rat dietary study, administration of 1,000 mg/kg/day produced increased absolute and relative liver weights. In a 3–month rat dietary study, administration of 25 mg/kg/day (LOEL) produced increased liver weights and vacuolization and hypertophy of hepatocytes. The NOEL was 5 mg/kg/day. In a 1-year dog study, dietary administration of 48 mg/kg/day to males and 12 (LOEL) and 48 mg/kg/day to females produced increased alkaline phosphatase and alanine transaminase levels. The NOEL was 3 mg/kg/day. In a 2–year dog study, dietary administration of 6.25 mg/kg/day (LOEL) produced slight increases in liver weights, elevated alkaline phosphatase levels, and increased thyroid-to-body-weight ratios. The NOEL was 1.25 mg/kg/day. Based on the NOEL, an oral RfD of 0.013 mg/kg/day was established. Hypertrophy of centrilobular hepatocytes was observed in male mice administered 162.5 mg/kg/day (LOEL) in the diet. The NOEL was 6.5 mg/kg/day.

EPA believes that there is sufficient evidence for listing terbacil on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hepatic and developmental toxicity data for this chemical.

243. *Tetracycline hydrochloride* (CAS No. 000064-75-5) (CAL) (Ref. 8). Tetracycline hydrochloride is widely used as an antibiotic for the treatment of many common infections. The average oral adult dose for most infections is 1 to 2 grams per day in equally divided doses. The most frequent adverse reactions to orally administered tetracycline hydrochloride are gastrointestinal effects including nausea, vomiting, diarrhea, bulky loose stools, and abdominal discomfort. Photosensitivity, manifested as an exaggerated sunburn reaction on sun-exposed areas of the body has occurred following oral therapy with tetracycline hydrochloride. Photosensitivity reactions of this type generally develop within a few minutes to several hours after sun exposure and usually persist 1 to 2 days after discontinuance of tetracycline hydrochloride.

Manufacturers of tetracycline hydrochloride state that this substance should not be used in women during the last half of pregnancy or in children younger than 8 years of age unless other appropriate drugs are ineffective or contraindicated. The American Academy of Pediatrics recommends that tetracycline hydrochloride be used only in children who are 9 years of age or older, except under unusual circumstances. Use of tetracycline hydrochloride in pregnant women or infants has resulted in retardation of skeletal development and bone growth in the fetus or child. Because tetracycline hydrochloride localizes in the dentin and enamel of developing teeth, use of this substance during tooth development may cause enamel hypoplasia and permanent yellow-gray to brown discoloration of the teeth. Use of tetracycline hydrochloride may result in discoloration of the deciduous teeth of children if the substance is used during pregnancy or in children up to 4 to 6 months of age. These effects are most common following long-term use of tetracycline hydrochloride but have occured following repeated short-term use. Premature infants treated with tetracycline have demonstrated a 40 percent depression of bone growth. This effect is readily reversible if exposure to the substance is short.

Intraperitoneal injection of 85 mg/kg/day on days 14 to 18 of gestation has resulted in abortion and extra embryonic structures in rat offspring. Subcutaneous injection of 48 mg/kg/day on days 16 through 20 of gestation and intramuscular injection of 40 mg/kg/day to rats on days 10 through 15 of gestation resulted in embryo/fetotoxicity. Exposure to 50 mg/kg/day on days 7 to 15 of pregnancy resulted in postimplantation loss and fetotoxicity in rats. Exposure to 85 mg/kg/day on days 7 to 15 of pregnancy resulted in abortion in rats. Fetotoxicity was observed in mice receiving 86 mg/kg/day of tetracycline hydrochloride on days 8 to 13 of gestation.

EPA believes that there is sufficient evidence for listing tetracycline hydrochloride on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available developmental toxicity data and other chronic toxicity data for this chemical.

244. *Tetramethrin (2,2-Dimethyl-3-(2-methyl-1-propenyl) cyclopropanecarboxylic acid (1,3,4,5,6,7-hexahydro-1,3-dioxo-2-H-isoindol-2-yl)methyl ester* (CAS No. 007696-12-0) (FIFRA AI) (Ref. 3). Depression, salivation, ataxia, lethargy, and convulsions were observed in acute rat studies in which the oral LD_{50} values were greater than or equal to 4,400 mg/kg. Tremors, excitement, and increased urine volume were observed in an acute dermal rat study in which the LD_{50} was greater than 2,500 mg/kg. Tremors, ataxia, dyspnea, gastointestinal hypermotility, and diarrhea were observed in rats and mice administered tetramethrin subcutaneously or

intraperitonealy. The LD_{50} was greater than 500 mg/kg. In a 6–month dog dietary study, administration of 62.5 mg/kg/day produced nervouseness and tremors. The NOEL was 31.25 mg/kg/day.

EPA believes that there is sufficient evidence for listing tetramethrin on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available neurological toxicity data for this chemical.

Aquatic acute toxicity values for tetramethrin include a bluegill 96–hour LC_{50} of 21 ppb (mixed isomers, technical product) and 69 ppb. EPA believes that there is sufficient evidence for listing tetramethrin on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data for this chemical.

245. *Tetrasodium ethylenediaminetetraacetate* (CAS No. 000064-02-8) (FIFRA AI) (Ref. 3). Increased occurrence of 13th rudimentary ribs was observed in the offspring of rats orally administered 5 mg/kg/day (LOEL). No NOEL was established and the dosing duration was not reported. EPA believes that there is sufficient evidence for listing tetrasodium ethylenediaminetetraacetate on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available developmental toxicity data for this chemical.

246. *Thiabendazole (2-(4-Thiazolyl)-1H-benzimidazole)* (CAS No. 000148-79-8) (FIFRA AI) (Ref. 3). Oral administration of 600 mg/kg/day (LOEL) to rats on days 6 through 15 of gestation produced cleft palate and open eyes. Musculoskeletal abnormalities were observed in the offspring of mice orally administered 240 mg/kg on day 9 of gestation. Musculoskeletal abnormalities were also observed in the offspring of rats orally administered 296 mg/kg/day on days 8 through 15 of gestation. Decreased litter size, and skin abnormalities were observed in the offspring of rats orally administered 667 mg/kg/day on days 8 through 15 of gestation. Oral administration of 1,300 mg/kg/day produced musculoskeletal abnormalities and fetal death in the offspring of mice. Oral administration of 2,400 mg/kg/day on day 11 of gestation produced craniofacial abnormalities in the offspring of mice. EPA believes there is sufficient evidence for listing thiabendazole on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available developmental toxicity data for this chemical.

Aquatic acute toxicity values for thiabendazole include a rainbow trout 96–hour LC_{50} of 560 ppb, a daphnid 48–hour EC_{50} of 0.31 ppb, and a mysid 96–hour LC_{50} of 340 ppb. EPA believes that there is sufficient evidence for listing thiabendazole on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data.

247. *Thiabendazole, hypophosphite salt (2-(4-thiazolyl) benzimidazole, hypophosphite salt)* (CAS No. 028558-32-9) (FIFRA AI) (Ref. 3). Few toxicity data are available on thiabendazole, hypophosphite salt. However, data are available on the parent compound, thiabendazole, as discussed below.

Oral administration of 600 mg/kg/day (LOEL) to rats on days 6 through 15 of gestation produced cleft palate and open eyes. Musculoskeletal abnormalities were observed in the offspring of mice orally administered 240 mg/kg on day 9 of gestation. Musculoskeletal abnormalities were also observed in the offspring of rats orally administered 296 mg/kg/day on days 8 through 15 of gestation. Decreased litter size and skin abnormalities were observed in the offspring of rats orally administered 667 mg/kg/day on days 8 through 15 of gestation. Oral administration of 1,300 mg/kg/day produced musculoskeletal abnormalities and fetal death in the offspring of mice. Oral administration of 2,400 mg/kg/day on day 11 of gestation produced craniofacial abnormalities in the offspring of mice. EPA believes that there is sufficient evidence for listing thiabendazole hypophosphite salt on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available developmental toxicity data for this chemical.

No laboratory data are available for thiabendazole hypophosphite salt. Ecotoxicity data are available for the parent compound thiabendazole. Aquatic acute toxicity values for thiabendazole include a rainbow trout 96–hour LC_{50} of 560 ppb, a daphnid 48–hour EC_{50} of 0.31 ppb, and a mysid 96–hour LC_{50} of 340 ppb. EPA believes that there is sufficient evidence for listing thiabendazole hypophosphite salt on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data for this chemical.

248. *Thiobencarb (carbamic acid, diethylthio-, s-(p-chlorobenzyl))* (CAS No. 028249-77-6) (FIFRA AI) (Ref. 3). Measured aquatic acute toxicity values for thiobencarb include a chinook salmon 96–hour LC_{50} of 760 ppb, a striped bass 96–hour LC_{50} of 760 ppb, a rainbow trout 96–hour LC_{50} of 790 ppb, and a green algae 72–hour EC_{50} of 30 ppb (population reduction). EPA believes that there is sufficient evidence for listing thiobencarb on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data.

249. *Thiodicarb* (CAS No. 059669-26-0) (FIFRA AI) (Ref. 3). Thiodicarb is a cholinesterase inhibitor in mammalian species. In addition, hematological effects have been observed in various species. Tremors and pinpoint pupils (the NOEL was less than 0.043 mg/L) were noted in rats exposed by inhalation to thiodicarb for 9 days. Macrocytic anemia (the LOEL was 2 g/kg; the NOEL was 1 g/kg) was observed in a 21–day dermal study in rabbits. In another 21–day dermal study, decreased red blood cell counts and decreased hemoglobin levels (the LOEL was 4 g/kg/day; the NOEL was 1 g/kg/day) were reported. Decreased plasma and red blood cell cholinesterase activities (the LOEL was 30 mg/kg/day; the NOEL was 10 mg/kg/day) were observed in rats fed thiodicarb for 28 days. Decreased red blood cell cholinesterase activity and decreased hemoglobin levels (the LOEL was 10 mg/kg/day; the NOEL was 3 mg/kg/day) were seen in a 13–week feeding study in rats. Inhibition of plasma and red blood cell cholinesterase activities (the LOEL was 45 mg/kg/day; the NOEL was 15 mg/kg/day) was noted in dogs fed thiodicarb for 6 months. Significant hematological and clinical chemistry values were also seen at 45 mg/kg/day (the NOEL was 15 mg/kg/day). Reductions in red blood cell cholinesterase activity (the LOEL was 12.8 mg/kg/day; the NOEL was 4.5 mg/kg/day) were also seen in a 1-year feeding study in dogs. In addition, decreased red blood cell and decreased hemoglobin and hematocrit levels, and increased relative spleen and liver weights (the LOEL was 38.3 mg/kg/day; the NOEL was 12.8 mg/kg/day) were reported. EPA believes that there is sufficient evidence for listing thiodicarb on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available neurological and hematological toxicity data.

Aquatic acute toxicity values for thiodicarb include a bluegill 96–hour LC_{50} of 1.47 ppm , a rainbow trout 96–hour LC_{50} of 2.65 ppm, a sheepshead minnow 96–hour LC_{50} of 530 ppb, a daphnid 48–hour EC_{50} of 27 ppb, a mysid 96–hour LC_{50} of 29.3 ppb, an eastern oyster 96–hour LC_{50} of 1.0 ppb, and an algae 96–hour EC_{50} of 450 ppb. EPA believes that there is sufficient evidence for listing thiodicarb on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data.

250. *Thiophanate ethyl ([1,2-phenylenebis (iminocarbonothioyl)]*

biscarbamic acid diethyl ester) (CAS No. 023564-06-9) (FIFRA AI) (Ref. 3). In a 6–month dog study, dietary administration of 500 mg/kg/day (LOEL) produced thyroid changes. The NOEL was 50 mg/kg/day. Thyroid follicular hypertrophy was observed at 50 mg/kg/day (LOEL) in a rat 2–year dietary study. The NOEL was 10 mg/kg/day. EPA believes that there is sufficient evidence for listing thiophanate ethyl on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available thyroid toxicity data for this chemical.

251. *Thiophanate-methyl* (CAS No. 023564-05-8) (FIFRA SR) (Ref. 8). Decreased spermatogenesis was observed in male rats fed 32 mg/kg/day thiophanate-methyl. The NOEL was 8 mg/kg/day. Other effects noted at the 32 mg/kg/day dose level included decreased body weight and histological evidence of hyperthyroidism.

In a 3-generation reproductive study in rats, reduced litter weights were seen at a daily dietary dose of 32 mg/kg thiophanate-methyl. The NOEL was 8 mg/kg/day. A decrease in the number of implantations was observed in mice administered a limit dose of 1,000 mg/kg/day.

EPA believes that there is sufficient evidence for listing thiophanate-methyl on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the reproductive toxicity data for this chemical.

252. *Thiosemicarbazide* (CAS No. 000079-19-6) (CERCLA; EPCRA EHS; RCRA APP8; RCRA P) (Ref. 8). The oral LD_{50}s for thiosemicarbazide in rats and dogs are 9.16 and 10 mg/kg, respectively. The LDLo in the mouse is 94 mg/kg. Cats orally administered thiosemicarbazide experienced convulsions, salivation, and vomiting; the LD_{50} was 20 mg/kg. Intraperitoneal injection of 2.5 mg/kg of thiosemicarbazide produced restlessness, running fits, and convulsions in rabbits. EPA's exposure analysis indicates that thiosemicarbazide concentrations are likely to exist beyond facility site boundaries, as a result of continuous, or frequently recurring releases, at levels that can reasonably be anticipated to cause significant adverse acute human health effects. EPA believes that there is sufficient evidence for listing thiosemicarbazide on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(A) based on the available acute toxicity and exposure data for this chemical.

253. *Triadimefon (1-(4-chlorophenoxy)-3,3-dimethyl-1-(1H-1,2,4-triazol-1-yl)-2-butanone)* (CAS No. 043121-43-3) (FIFRA AI) (Ref. 3). Decreased hematocrit, red blood cell count, and hemoglobin volume were observed in dogs orally administered 60 mg/kg/day (LOEL) for 13 weeks. No NOEL was established. In a 2–year dietary rat study, decreased hemoglobin and erythrocyte counts were observed at 25 mg/kg/day (LOEL). The NOEL was 2.5 mg/kg/day. Based on the NOEL of the study, an oral RfD of 0.03 mg/kg/day was derived. Dietary administration of 25 mg/kg/day (LOEL) for 2 years to dogs produced increased serum alkaline phosphatase and N-demethylase activity and increased liver weight. The NOEL was 2.5 mg/kg/day. Increased erythrocyte count, thrombocyte count, hemoglobin, and hematocrit levels in females and increased serum alkaline phosphatase, serum glutamic-pyruvic transaminase, serum glutamic-oxaloacetic transaminase, liver weights, and hyperplastic nodules in both sexes were observed at 234 mg/kg/day in a 2–year mouse dietary study. The NOEL was 6.5 mg/kg/day and the LOEL was 39 mg/kg/day. In another 2–year mouse dietary study, administration of 234 mg/kg/day produced hepatocellular adenomas. Doses of 39 mg/kg/day in males (LOEL) and 6.5 mg/kg/day in females (LOEL) produced nonneoplastic and preneoplastic changes in the liver, increased liver weights with correlating effects on serum enzymes, and hepatocellular hypertrophy. The NOEL in males was 6.5 mg/kg/day and no NOEL in females could be established.

Cleft palates were observed in the offspring of rats orally administered 75 mg/kg/day (LOEL). The NOEL was 30 mg/kg/day. Increased incidence of abnormal ribs, extra ribs, and distended urinary bladders were observed in the offspring of rats orally administered 90 mg/kg/day (LOEL). The NOEL was 30 mg/kg/day. Increases in fetal resorptions were observed in rabbits given 100 mg/kg/day by gavage (LOEL). The NOEL was 30 mg/kg/day. Increased incidences of incomplete ossification of pelvic pubes and phalanges, and irregular spinous processes were observed in the offspring of rabbits orally administered 50 mg/kg/day (LOEL) on days 6 through 18 of gestation. The NOEL was 20 mg/kg/day.

In a 3-generation rat reproduction study, decreased fertility and decreased litter size were observed at 90 mg/kg/day (LOEL). The NOEL was 15 mg/kg/day. In a 2-generation reproduction study in rats, decreased pup weights, decreased litter size, and decreased pup viability were observed at 90 mg/kg/day (LOEL). The NOEL was 2.5 mg/kg/day.

EPA believes that there is sufficient evidence for listing triadimefon on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hepatic, hematological, developmental, and reproductive toxicity data for this chemical.

254. *Triallate* (CAS No. 002303-17-5) (FIFRA SR) (Ref. 8). Triallate, a dithiocarbamate insecticide, is a cholinesterase inhibitor. When triallate was administered to rats at a dose of 147.1 mg/kg/day orally and to cats at a dose of 0.028 mg/L/day (via aerosol) for 2 months, the animals developed fatal morphological changes in neurons of the cerebral cortex, subcortical area, cerebellum, and spinal cord. Doses of 30 mg/kg/day caused head bobbing and circling in pregnant rats. The NOEL was 7.5 mg/kg/day. The LOEL and NOEL for liver effects in a 2–year study in dogs fed diets containing triallate were 4.25 mg/kg/day and 1.28 mg/kg/day, respectively. At 4.25 mg/kg/day an increase in hemosiderin deposition and serum alkaline phosphatase was observed in both sexes, and an increase in liver weight was observed in females. Based on the NOEL, an oral RfD of 0.013 mg/kg/day was derived. In a hamster chronic feeding study, decreased triglycerides were seen in males at the LOEL of 30 mg/kg/day. The NOEL was 5 mg/kg/day. Ninety-day feeding studies in rats (10 mg/kg/day) and dogs (5 mg/kg/day) showed no treatment related adverse effects except for increased liver-to-body-weight ratios in the dogs. EPA believes that there is sufficient evidence for listing triallate on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the chronic neurological and hepatic toxicity data for this chemical.

255. *Tribenuron methyl (2-(((((4-methoxy-6-methyl-1,3,5-triazin-2-yl)-methylamino)carbonyl)amino)sulfonyl)-, methyl ester)* (CAS No. 101200-48-0) (FIFRA AI) (Ref. 3). In a 1-year feeding study in dogs, elevated serum bilirubin and aspartate aminotransferase (AST) levels and increased urinary volume were reported in males receiving 8.16 mg/kg/day (LOEL). The NOEL for males was 0.79 mg/kg/day. The LOEL for females was 52.02 mg/kg/day (the highest dose tested) and was based on increased serum creatinine and transient increases in AST, globulin, and serum bilirubin. These females had an 18.2 percent decrease in body weight gain. The NOEL for females was 8.18 mg/kg/day. The highest dose in males (51.46 mg/kg/day) caused increases in serum creatinine and a 20 percent decrease in body weight gain. The oral RfD, derived from the NOEL for males, was 0.008 mg/kg/day. In a 90–day feeding study, decreased absolute and relative liver and kidney weights, serum glucose, globulin and cholesterol levels

were observed in rats at 87.5 mg/kg/day (LOEL). The NOEL was 5 mg/kg/day. EPA believes that there is sufficient evidence for listing tribenuron methyl on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hepatic and renal toxicity data for this chemical.

256. *Tributyltin fluoride* (CAS No. 001983-10-4) (FIFRA AI) (Ref. 3). Aquatic acute toxicity values for tributyltin fluoride include a bleak fish 96–hour LC_{50} of 2.3 ppb, an algae 72–hour EC_{50} of 9.3 ppb, and a Harpacticoid copepod 96–hour LC_{50} of 0.8 ppb. EPA believes that there is sufficient evidence for listing tributyltin fluoride on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data.

257. *Tributyltin methacrylate* (CAS No. 002155-70-6) (FIFRA AI) (Ref. 3). Pregnant rats were given tributyltin methacrylate by gavage on days 6 to 19 of gestation. Mean fetal weight and maternal body weight gain were decreased at 18 mg/kg/day. Fetal resorptions were also significantly increased. The fetotoxic NOEL for this study was 9 mg/kg/day. EPA believes that there is sufficient evidence for listing tributyltin methacrylate on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available developmental toxicity data for this chemical.

258. *S,S,S-Tributyltrithiophosphate (DEF)* (CAS No. 000078-48-8) (FIFRA AI) (Ref. 8). S,S,S-Tributyltrithiophosphate (DEF) is a cholinesterase inhibitor. Both immediate and delayed neurotoxic effects have been reported in humans following exposure to DEF. The exposure levels at which these effects occurred, however, were not reported. In a 3–month hen feeding study, the NOEL for neurotoxic effects was 0.1 mg/kg/day and the LOEL was 0.5 mg/kg/day. At 0.5 mg/kg day, hens showed delayed neurotoxicity, ataxia, and equivocal changes in the spinal cord and peripheral nerves. Based on the NOEL, EPA has derived an oral RfD of 0.00003 mg/kg/day for this chemical. In a 12–week dog feeding study, animals showed over sensitivity to stimuli at 0.62 mg/kg/day; the NOEL was 0.12 mg/kg/day. In the same study, the LOEL for cholinesterase inhibition was 0.12 mg/kg/day and a NOEL was not established. Brain cholinesterase inhibition was observed in a chronic rat feeding study at 1.25 mg/kg/day. The NOEL was 0.25 mg/kg/day. EPA believes that there is sufficient evidence for listing S,S,S-tributyltrithiophosphate on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the chronic neurotoxicity data for this chemical.

Measured acute aquatic toxicity data for S,S,S–tributyltrithiophosphate include a rainbow trout 96–hour LC_{50} of 660 ppb (0.660 ppm) and a bluegill 96–hour LC_{50} of 620 ppb (0.620 ppm). The measured log K_{ow} is 5.7. EPA believes that there is sufficient evidence for listing S,S,S-tributyltrithiophosphate on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the environmental toxicity data for this chemical and its potential for bioaccumulation.

259. *Trichloroacetyl chloride* (CAS No. 000076-02-8) (EPCRA EHS) (Ref. 8). Trichloroacetyl chloride is highly toxic in humans by the oral and inhalation routes of exposure. Numerous cases of strong irritation of the eyes, skin, and respiratory tract and fever, nausea, and vomiting following exposure to trichloroacetyl chloride have been reported. The acute inhalation LC_{50} values for mice and rats are 0.445 mg/L and 0.475 mg/L, respectively, indicating that trichloroacetyl chloride is highly toxic by inhalation in these species. EPA's exposure analysis indicates that trichloroacetyl chloride concentrations are likely to exist beyond facility site boundaries, as a result of continuous, or frequently recurring releases, at levels that can reasonably be anticipated to cause significant adverse acute human health effects. EPA believes that there is sufficient evidence for listing trichloroacetyl chloride on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(A) based on the available acute toxicity and exposure data for this chemical.

260. *Trichloroethylsilane* (CAS No. 000115-21-9) (EPCRA EHS) (Ref. 8). Chlorinated silanes are very corrosive to the skin and mucous membranes and liberate hydrochloric acid in the presence of water. Trichloroethylsilane causes severe burns and the vapor is harmful to humans. The mouse 2–hour inhalation LC_{50} value is 0.30 mg/L. EPA's exposure analysis indicates that trichloroethylsilane concentrations are likely to exist beyond facility site boundaries, as a result of continuous, or frequently recurring releases, at levels that can reasonably be anticipated to cause significant adverse acute human health effects. EPA believes that there is sufficient evidence for listing trichloroethylsilane on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(A) based on the available acute toxicity and exposure data for this chemical.

261. *Trichlorophenylsilane* (CAS No. 000098-13-5) (EPCRA EHS) (Ref. 8). Chlorinated silanes are very corrosive to the skin and mucous membranes and liberate hydrochloric acid in the presence of water. Trichlorophenylsilane causes severe burns and the vapor is harmful to humans (concentration not specified). The 2–hour mouse inhalation LC_{50} value is 0.33 mg/L. EPA's exposure analysis indicates that trichlorophenylsilane concentrations are likely to exist beyond facility site boundaries, as a result of continuous, or frequently recurring releases, at levels that can reasonably be anticipated to cause significant adverse acute human health effects. EPA believes that there is sufficient evidence for listing trichlorophenylsilane on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(A) based on the available acute toxicity and exposure data for this chemical.

262. *1,2,3-Trichloropropane* (CAS No. 000096-18-4) (RCRA APP8) (Ref. 8). Results of a subchronic oral toxicity study in rats and mice reveal that the primary target organs for 1,2,3-trichloropropane are the liver and kidney. Renal and hepatic necrosis were observed in rats administered 1,2,3-trichloropropane by gavage for 17 weeks. The LOAEL was 16 mg/kg/day and the NOAEL was 8 mg/kg/day for hepatic effects. The LOAEL was 32 mg/kg/day and the NOAEL was 16 mg/kg/day for renal effects. Hepatic necrosis in mice occurred at 125 mg/kg/day. The NOAEL was 63 mg/kg/day. Less severe renal necrotic changes were seen at 250 mg/kg/day. The NOAEL was 125 mg/kg/day. The renal and hepatic lesions were accompanied by increases in organ weights and alterations in serum enzymes that were indicative of hepatic and renal toxicity. At lower dose levels (the LOAEL was 16 mg/kg/day), nonregenerative anemia (decreased hematocrit, hemoglobin, and erythrocyte count) was observed in rats. The NOAEL was 8 mg/kg/day. Nonregenerative anemia is considered to be one of the most sensitive effects of 1,2,3-trichloropropane.

The respiratory tract is a principal target of inhaled 1,2,3-trichloropropane in humans and animals. Irritation of the eyes and throat has been reported in humans acutely exposed (15 minutes) to 100 ppm (0.602 mg/L) of 1,2,3-trichloropropane via inhalation. Irritative effects on the olfactory epithelium have been observed in rats exposed by inhalation to 3 ppm (the LOAEL was 0.018 mg/L; the NOAEL was 0.006 mg/L) of 1,2,3-trichloropropane for 11 days. Histological effects have also been seen in the nasal cavity (the LOEL was 125 mg/kg/day; the NOAEL was 63 mg/kg/

day) in rats and in the bronchiolar epithelium (the LOEL was 63 mg/kg/day; the NOAEL was 32 mg/kg/day) in mice that were exposed to 1,2,3-trichloropropane by oral intubation for 17 weeks.

EPA believes that there is sufficient evidence for listing 1,2,3-trichloropropane on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the hematological, respiratory, hepatic, and renal toxicity data for this chemical.

263. *Triclopyr triethylammonium salt* (CAS No. 057213-69-1) (FIFRA AI) (Ref. 3). Degeneration of proximal tubules (the LOEL was 20 mg/kg/day; the NOEL was 5 mg/kg/day) was noted in male and female rats fed triclopyr for 3 months. A LOEL of 2.5 mg/kg/day, based on phenosulfophthalein (PSP) excretion, was reported in dogs fed triclopyr for 6 months. A similar effect was also noted at the LOEL of 5 mg/kg/day, determined in dogs fed triclopyr for 8 months. The NOEL was greater than 5 mg/kg/day. Significant increases in absolute and relative kidney weights were observed in rats fed 36 mg/kg/day for 2 years. The NOEL was 12 mg/kg/day. In a pharmacokinetic study, reduced PSP excretion was seen in dogs administered 5 mg/kg/day, whereas no effect on PSP excretion was seen in monkeys administered 20 mg/kg/day. No details on the route and length of exposure were provided. EPA believes that there is sufficient evidence for listing triclopyr triethylammonium salt on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available renal toxicity data.

264. *Triethylamine* (CAS No. 000121-44-8) (CAA HAP) (Ref. 7). Triethylamine is an acute irritant which causes eye and nasal irritation and pulmonary toxicity in mice and rats and is an acute eye toxicant in man.

In a survey of workers exposed to triethylamine, none of the workers reported effects at 5 ppm. Slight to mild effects were noted at concentrations between 5 and 10 ppm and above 10 ppm workers reported visual disturbances which included halo vision and irritation of the eyes, nose, and throat. In a separate report, eye irritation and visual disturbances consisting of foggy vision, blue haze or halo vision (halo around lights) was reported in 19 workers exposed to triethylamine. Exact exposure levels were not determined. The TWA in the work place of those individuals who complained of "blue haze" was 11 mg/m^3 with a range of 4 to 24 mg/m^3. The American Council of Government and Industrial Hygienists (ACGIH) has set a threshold limit value-time weighted average (TLV-TWA) of 10 ppm and a threshold limit value-short-term exposure limit (TLV-STEL) of 15 ppm based upon inhalation toxicity in guinea pigs and rats and skin irritation and eye injury in rabbits.

EPA's exposure analysis indicates that triethylamine concentrations are likely to exist beyond facility site boundaries, as a result of continuous, or frequently recurring releases, at levels that can reasonably be anticipated to cause significant adverse acute human health effects. EPA believes that there is sufficient evidence for listing triethylamine on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(A) based on the available acute toxicity and exposure data for this chemical.

265. *Triforine (N,N'-[1,4-piperazinediylbis(2,2,2-trichloroethylidene)] bisformamide)* (CAS No. 026644-46-2) (FIFRA AI) (Ref. 3). In a 2-year feeding study in rats, anemia was reported. The LOEL, based on this effect, was 3,125 ppm (156 mg/kg/day) and the NOEL was 625 ppm (31.25 mg/kg/day). Siderosis of Kupffer cells and bone marrow cells was reported in dogs exposed to triforine in their diet for 2 years. The LOEL in this study was 1,000 ppm (25 mg/kg/day) and the NOEL was 100 ppm (2.5 mg/kg/day). Effects on red blood cells, hematocrit or hemoglobin were also noted in dogs or rats in several 13-week feeding studies. For example, dogs exposed to a 20.6 percent a.i. formulation of the compound for 13 weeks at dose levels that included 600 ppm (the LOEL, equivalent to 15 mg/kg/day or 3.1 mg a.i./kg/day) and 100 ppm (the NOEL, equivalent to 2.5 mg/kg/day or 0.5 mg a.i./kg/day) had siderosis in the liver, spleen, and bone marrow.

A decrease in mean relative weight of offspring was observed in rabbits exposed to 25 mg/kg triforine (the fetotoxicity LOEL). The fetotoxicity NOEL was 5 mg/kg. The LOEL and NOEL for maternal toxicity in this developmental toxicity study were also 25 mg/kg and 5 mg/kg, respectively, and were based on reduced food intake and body weight loss. Fetotoxicity (decreased number of fetuses and increased resorptions) was also reported in the offspring of rats fed 1,600 mg/kg (the fetotoxicity LOEL) for an unspecified duration. The fetotoxicity NOEL was 800 mg/kg.

EPA believes that there is sufficient evidence for listing triforine on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available hematological and developmental toxicity data for this chemical.

266. *Trimethylchlorosilane* (CAS No. 000075-77-4) (EPCRA EHS) (Ref. 8). Chlorinated silanes are very corrosive to the skin and mucous membranes and liberate hydrochloric acid in the presence of water. Trimethylchlorosilane causes severe burns and the vapor is harmful to humans. The mouse inhalation LCLo value is 0.10 mg/L. EPA's exposure analysis indicates that trimethylchlorosilane concentrations are likely to exist beyond facility site boundaries, as a result of continuous, or frequently recurring releases, at levels that can reasonably be anticipated to cause significant adverse acute human health effects. EPA believes that there is sufficient evidence for listing trimethylchlorosilane on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(A) based on the available acute toxicity and exposure data for this chemical.

267. *2,3,5-Trimethylphenyl methylcarbamate* (CAS No. 002655-15-4) (FIFRA AI) (Ref. 3). Cholinesterase inhibition was reported in a series of studies for this carbamate pesticide. In dogs that received 2,000 ppm (50 mg/kg/day) 2,3,5-trimethylphenyl methylcarbamate in their diet for 14 days, there was inhibition of plasma and red blood cell cholinesterases and also weight loss. Brain cholinesterase was slightly decreased in rats in a 2-year feeding study at 200 ppm (10 mg/kg/day). At 800 ppm (40 mg/kg/day), there were fatty changes in the liver which disappeared after 7.5 months. EPA believes that there is sufficient evidence for listing 2,3,5-trimethylphenyl methylcarbamate on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available neurological toxicity data for this chemical.

268. *Triphenyltin chloride* (CAS No. 000639-58-7) (EPCRA EHS) (Ref. 8). Oral exposure of male rats to 380 mg/kg triphenyltin chloride over 19 days caused adverse effects on the testes, epididymis, sperm duct, prostate gland, seminal vesicle, Cowper's gland, and accessory glands. EPA believes that there is sufficient evidence for listing triphenyltin chloride on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the reproductive toxicity data for this chemical.

Measured aquatic acute toxicity data for triphenyltin chloride include a 48-hour LC_{50} for carp of 55 ppb and a 72-hour EC_{50} (growth) for marine green algae of 0.92 ppb. In addition, the measured aquatic toxicity information indicates a freshwater green algae 8-day EC_{50} (growth) of 2 ppb. EPA believes that there is sufficient evidence for listing triphenyltin chloride on EPCRA section 313 pursuant to EPCRA section

313(d)(2)(C) based on the environmental toxicity data for this chemical.

269. *Triphenyltin hydroxide* (CAS No. 000076-87-9) (FIFRA SR) (Ref. 8). Triphenyltin hydroxide has been classified by EPA as a Group B2 compound, i.e., a probable carcinogen. This was based on the significant increases in fetal pituitary gland adenomas in female Wistar rats and Leydig cell tumors in male Wistar rats fed 1 or 4 mg/kg/day triphenyltin hydroxide for 2 years. There were significant increases of hepatocellular adenomas and combined hepatocellular (adenoma and/or carcinoma) tumars in male and female NMRI mice fed 0.65, 2.6, or 10.4 mg/kg/day for 80 weeks.

In a developmental toxicity study in rats, oral doses of 15 mg/kg of triphenyltin hydroxide during gestation days 1 to 7 prevented implantation. When administered from day 8 and onwards, the compound was fetolethal.

EPA believes that there is sufficient evidence for listing triphenyltin hydroxide on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the carcinogenicity data and the developmental toxicity data for this chemical.

Measured aquatic acute toxicity data for triphenyltin hydroxide include a fathead minnow 96–hour LC_{50} of 5.4 ppb, a bluegill 96–hour LC_{50} of 23 ppb, a rainbow trout 96–hour LC_{50} of 15 ppb, and a marine green algae 72–hour LC_{50} of 13.9 ppb. EPA believes that there is sufficient evidence for listing triphenyltin hydroxide on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the environmental toxicity data for this chemical.

270. *Vanadium pentoxide* (CAS No. 001314-62-1) (CERCLA; EPCRA EHS; RCRA APP8) (Ref. 8). Eighteen workers exposed to vanadium pentoxide dusts at concentration in excess of 0.5 mg/m^3 (0.0005 mg/L) for a period of up to 2 weeks developed respiratory symptoms that persisted for nearly 2 weeks after removal from exposure. Inhalation of unspecified levels of vanadium pentoxide for 1 to 5 years produced asthma in 3 of 20 workers. Mice and rats exposed to 1 to 3 mg/m^3 (0.001 to 0.003 mg/L) vanadium pentoxide 6 hours/day for 3 months developed histopathologic changes in their lungs and had a decrease in growth rate. EPA believes that there is sufficient evidence for listing vanadium pentoxide on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available chronic respiratory toxicity data for this chemical.

271. *Vinclozolin (3-(3,5-dichlorophenyl)-5-ethenyl-5-methyl-2,4-oxazolidinedione)* (CAS No. 050471-44-8) (FIFRA AI) (Ref. 3). The results of a 3–month feeding study in Wistar rats administered 4,500 ppm (225 mg/kg/day; the only dose tested) indicate that vinclozolin interacts with numerous steroid hormones in male and female animals. A broad spectrum of steroid hormones were affected in these animals, including increases in adrenocorticotropic hormone, lutenizing hormone, follicle stimulating hormone, testosterone, corticosterone, aldosterone, and dehydroepiandrosterone and slight decreases in estradiol levels in males. Female rats had elevated adrenocorticotropic hormone and luteinizing levels and depressed corticosterone and aldosterone levels, while follicle stimulating hormone, testosterone, dehydroepiandrosterone and estradiol levels were comparable to controls. After a 2–month recovery period postdosing, all male hormone levels were normal except for a slight elevation in FSH, and all female hormone levels were normal except for a slight elevation in estradiol. The endocrine changes also were reported in developmental, subchronic, and chronic toxicity studies.

A broad spectrum of organ changes occurred in dogs exposed to vinclozolin in a 1-year feeding study. Males administered 4.8 mg/kg/day (the LOEL for males) had increases in testes weights, increased bilirubin, and prostate atrophy. The NOEL in males was 2.4 mg/kg/day. Females in this study had increased adrenal weights, lipid accumulation in the adrenal glands, and marginally increased hemosiderin in the liver at 5.1 mg/kg/day (the LOEL for females). The NOEL for females was 2.5 mg/kg/day. At higher doses (47 mg/kg/day in males and 53 mg/kg/day in females), there were increases in weights of the liver, spleen, testes, adrenal, and thyroid. Other effects included increased diffuse hyperplasia of the Leydig cells, lipid accumulation in the adrenal cortex, and increased platelets in males, and in females, slight increases in mean corpuscular volume and mean corpuscular hemoglobin concentration. The oral RfD for this compound, 0.025 mg/kg/day, was based on the findings of a 6–month feeding study with beagle dogs, in which adrenal weights (absolute and relative) were significantly increased at 7.5 mg/kg/day (the LOEL). The NOEL was 2.5 mg/kg/day. Both males and females exposed to higher doses (600 and 2,000 ppm, or 15 and 50 mg/kg/day) had histological changes in the adrenal glands, including vacuolation of the zona fasciculata. In addition to effects on the adrenal gland, males exposed to the LOEL dose and higher had decreased absolute kidney weights, and at 600 ppm, fat droplets in the distal tubule were observed.

Pseudohermaphroditism (a decrease in anal-genital distance) occurred in male offspring of rats administered doses of 50 mg/kg (the LOEL) and higher by gavage. The developmental NOEL was 15 mg/kg. The same effect was noted in the offspring of rats that received dermal applications of 180 mg/kg/day (LOEL; the developmental NOEL was 60 mg/kg/day) during gestation, and also in a 2-generation reproduction study in rats (the LOEL was 86 mg/kg/day, the NOEL was 25 mg/kg/day). Other developmental effects observed in the latter study included developmental delays, reduced male and female pup weight, increased stillbirths and increased pup mortality throughout lactation.

EPA believes that there is sufficient evidence for listing vinclozolin on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available endocrine, adrenal, renal, hepatic, and developmental toxicity data.

V. Rationale for Listing

EPA is proposing to add the chemical substances identified in Unit IV.B. of this preamble because EPA believes that these chemicals meet the statutory criteria for listing under section 313(d)(2) of EPCRA. The bases for these determinations and the specific toxic effects are summarized in Unit IV.B. of this preamble and set forth in more detail in the rulemaking record.

EPA intends to evaluate public comment on this proposed rule and issue a final rule by November 30, 1994. Reporting for the chemicals identified in the final rule would be required for activities during the 1995 calendar year. Such reports would have to be submitted to EPA and States by July 1, 1996.

VI. Rulemaking Record

The record supporting this proposed rule is contained in docket number OPPTS–400082. Nonconfidential documents, including an index of the docket, are available to the public in the TSCA Nonconfidential Information Center (NCIC), also known as the TSCA Public Docket Office from 12 noon to 4 p.m., Monday through Friday, excluding legal holidays. The TSCA Public Docket Office is located at EPA Headquarters, Rm. E–G102, 401 M St., SW., Washington, DC 20460.

Any person who submits comments claimed as CBI must mark the

comments as "confidential," "CBI," or other appropriate designation. Comments not claimed as confidential at the time of submission will be placed in the public file. Any comments marked as confidential will be treated in accordance with the procedures in 40 CFR part 2. Any person submitting comments claimed to be confidential must prepare and submit a nonconfidential public version of the comments in triplicate that EPA can place in the public file.

VII. Request for Public Comment

EPA requests comment on any aspect of this proposal. EPA requests specific comment as detailed in the following paragraphs.

EPA requests comment on the sufficiency of the evidence for each of the chemicals proposed for addition in Unit IV.B. of this preamble. In addition, EPA requests comment on any issues that may be specific to any of the individual chemicals or chemical categories.

EPA requests comment on whether it would be appropriate to list persistent bioaccumulative toxic chemicals that are manufactured, processed, or otherwise used below the current reporting thresholds on EPCRA section 313. 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 exemptions, would be required to insure that release and transfer information would be collected?

In Units IV.B.132., IV.B.144., and IV.B.158., of this preamble, EPA is proposing to add individually three diisocyanates: hexamethylene-1,6-diisocyante; isophorone diisocyanate; and 1,1-methylene bis(4-isocyanatocyclohexane). EPA requests comment on its alternative proposal in Unit IV.B.132. of this preamble to create a diisocyanates category rather than adding diisocyanates individually to EPCRA section 313. EPA also requests comment on what diisocyanates, other than those listed in IV.B.132. of this preamble, should be included in such a category.

EPA requests comment on its proposed definition of man-made mineral fibers, given in Unit IV.B.149. of this preamble, and any other options for defining a fibers category.

In Unit IV.B.166. and 172. of this preamble, EPA is proposing to add two ethylene bisdithiocarbamates (EBDCs): metiram; and nabam. An additional two EBDCs, zineb and maneb, are currently listed on the EPCRA section 313 list of toxic chemicals. The category of EBDCs has recently been added to EPCRA section 313 (December 1, 1993; 58 FR 63500). EPA requests comment on the following: (1) Should the individual EBDCs, metiram and nabam, be added individually to EPCRA section 313 even though they are members of the EBDC category, which is listed on EPCRA section 313; and (2) should the individual listings for two EBDCs, zineb and maneb, be deleted and added as members of the newly created EBDC category?

EPA requests comment on whether polycyclic aromatic compounds (PACs) should be added as a delineated category consisting of the PACs listed in Unit IV.B.207. of this preamble or as a category with the definition given in Unit IV.B.207. of this preamble.

EPA requests comment on its approach in considering exposure as a part of its evaluation of certain chemicals under sections 313(d)(2)(A) and (C).

In Units IV.B.36, 179., and 235. of this preamble, EPA is proposing to add three chemicals (sulfur dioxide, nitrogen dioxide, and carbon monoxide) that are regulated by Title I of the CAA (Provisions for Attainment and Maintenance of National Ambient Air Quality Standards). In addition, sulfur dioxide is regulated under Title IV of the CAA (Acid Deposition Control). Extensive data, which are highly technical, are collected on these chemicals as required by the CAA. EPA requests comment on the following: (1) Is the information collected under the CAA sufficient for public right-to-know purposes; and (2) suggestions on how the data collected on these chemicals pursuant to CAA Titles I and IV could be used to meet the purposes of EPCRA section 313.

Comments should be submitted to the address listed under the ADDRESSES unit. All comments must be received on or before April 12, 1994.

VIII. Public Meeting

EPA will hold a 1–day public meeting to discuss the issues presented above. The tentative agenda for this public meeting will include a discussion of the issues presented in Unit VII. of this preamable.

Scheduling of oral statements will be on a first come first served basis by calling the telephone number listed under FOR FURTHER INFORMATION CONTACT. All statements will be made part of the public record and will be considered in the development of the final rule.

IX. References

(1) U.S. Congress, House of Representatives. "Conference Report No. 962," 99th Cong., 2nd Session. 294 (1986).

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

(3) 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).

(4) USEPA/OPPT. *Issue Paper Prepared for the Public Meeting on Expansion of the Toxic Release Inventory.* U. S. Environmental Protection Agency, Washington, DC (1993).

(5) USEPA/OPPT. *Physical Properties and Environmental Fate of Some TRI Expansion Chemicals.* U. S. Environmental Protection Agency, Washington, DC (1993).

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

(7) 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, D.C. (1993).

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

X. 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 the Office of Management and Budget (OMB) and the requirements of the Executive Order. Under section 3(f), the order defines a "significant regulatory action" as an action likely to result in 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.

Pursuant to the terms of this Executive Order, it has been determined that this proposed rule is a "significant regulatory action." As such, this action was submitted to OMB for review, 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." The analysis supporting this proposed rule estimated the maximum cost that a small business might incur, and calculated the cost impact percentage (reporting costs divided by average value of shipments) for each employee size, class, and SIC code.

Reporting costs are estimated at less than one percent of the average value of shipments per report in the first year, and less than one-half of one percent of the value of shipments per report in subsequent years. The precise impacts depend on how many reports an individual small business submits. However, experience with current reporters indicates that small businesses generally submit fewer reports per facility than larger ones. Most of the reports are anticipated to be submitted from industries with the lowest impacts. Because of this, no segment of the manufacturing sector is likely to suffer significant adverse effects due to this rule. Therefore, EPA certifies that this proposed rule will not have a significant impact on a substantial number of small entities.

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 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 101508) 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 proposed 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 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.

Send comments regarding this collection of information, including suggestions for reducing this burden, to Chief, Information Policy Branch, 2131, U.S. Environmental Protection Agency, 401 M St., SW., Washington, DC 20460; and to the Office of Information and Regulatory Affairs, Office of Management and Budget, 726 Jackson Place NW., Washington, DC 20503, marked "Attention: Desk Officer for EPA." The final rule will respond to any OMB or public comments on this collection of information.

List of Subjects in 40 CFR Part 372

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

Dated: January 6, 1994.

Carol M. Browner,

Administrator.

Therefore it is proposed that 40 CFR part 372 be amended to read as follows:

Part 372—[AMENDED]

1. The authority citation for part 372 would continue 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 four 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-allethrone]	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
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Amitraz	33089-61-1	1/1/95

Chemical Name	CAS No.	Effective Date
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Anilazine [4,6-dichloro-N-(2-chlorophenyl)-1,3,5-triazin-2-amine]	101-05-3	1/1/95
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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
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o-Benzyl-p-chlorophenol	120-32-1	1/1/95
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Bifenthrin	82657-04-3	1/1/95
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Bis(tributylin) 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
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2-Bromo-2-nitropropane-1,3-diol (Bronopol)	52-51-7	1/1/95
* * * * * * *		
Bromoxynil (3,5-Dibromo-4-hydroxybenzonitrile)	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
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Butylate (Bis-2-methylpropyl)carbamothioic acid S-ethyl ester)	2008-41-5	1/1/95
Butylated hydroxyanisole	25013-16-5	1/1/95
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C.I. Acid Red 114	6459-94-5	1/1/95
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C.I. Direct Blue 218	28407-37-6	1/1/95
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Calcium hypochlorite	7778-54-3	1/1/95
Caprolactam	105-60-2	1/1/95
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Carbofuran	1563-66-2	1/1/95
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Carbon monoxide	630-08-0	1/1/95
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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
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1-(3-Chloroallyl)-3,5,7-triaza-1-azoniaadamantane chloride	4080-31-3	1/1/95
p-Chloroaniline	106-47-8	1/1/95
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5-Chloro-2-(2,4-dichlorophenoxy)phenol	3380-34-5	1/1/95
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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
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3-Chloropropionitrile	542-76-7	1/1/95
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p-Chloro-o-toluidine	95-69-2	1/1/95

Chemical Name	CAS No.	Effective Date
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
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Clomazone [2-[(2-Chlorophenyl)methyl]-4,4-dimethyl-3-isoxazolidinone]	81777-89-1	1/1/95
Crotonaldehyde	4170-30-3	1/1/95
Cyanazine	21725-46-2	1/1/95
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Cycloate	1134-23-2	1/1/95
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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
Cyromazine [N-Cyclopropyl-1,3,5-triazine-2,4,6-triamine]	66215-27-8	1/1/95
• • • • • • •		
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
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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
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Diazinon	333-41-5	1/1/95
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2,2-Dibromo-3-nitrilopropionamide	10222-01-2	1/1/95
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Dicamba (3,6-Dichloro-2-methyoxybenzoic 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
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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
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Dichlorofluoromethane (HCFC-21)	75-43-4	1/1/95
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Dichloromethylphenylsilane	149-74-6	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
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trans-1,3-Dichloropropene	10061-02-6	1/1/95
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Diclofop methyl [2-[4-(2,4-Dichlorophenoxy)phenoxy]propanoic acid, methyl ester]	51338-27-3	1/1/95
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Dicyclopentadiene	77-73-6	1/1/95
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Diethatyl ethyl	38727-55-8	1/1/95

Chemical Name	CAS No.	Effective Date
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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
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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
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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
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Dimethyl chlorothiophosphate	2524-03-0	1/1/95
Dimethyldichlorosilane	75-78-5	1/1/95
N,N-Dimethylformamide	68-12-2	1/1/95
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2,6-Dimethylphenol	576-26-1	1/1/95
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Dinocap	39300-45-3	1/1/95
Dinoseb	88-85-7	1/1/95
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Diphenamid	957-51-7	1/1/95
Diphenylamine	122-39-4	1/1/95
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Dipotassium endothall [7-Oxabicyclo(2.2.1)heptane-2,3-dicarboxylic acid, dipotassium salt]	2164-07-0	1/1/95
Dipropyl isocinchomeronate	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
Dithiopyr [2-(Difluoromethyl)-4-(2-methylpropyl)-6-(trifluoromethyl)-3,5-pyridinedicarbothioic acid S,S-dimethyl ester]	97886-45-8	1/1/95
Diuron	330-54-1	1/1/95
2,4-D 2-octyl ester	1917-97-1	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
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Ethoprop [Phosphorodithioic acid O-ethyl S,S-dipropyl ester]	13194-48-4	1/1/95
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Ethyl dipropylthiocarbamate [EPTC]	759-94-4	1/1/95
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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-benzoxazolylen)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(dimethylcarbamo-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
Flumetralin [2-Chloro-N-(2,6-dinitro-4-(trifluoromethyl)phenyl)-N-ethyl-6-fluorobenzenemethanamine]	62924-70-3	1/1/95
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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
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alpha-Hexachlorocyclohexane	319-84-6	1/1/95
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Hexamethylene-1,6-diisocyanate	822-60-0	1/1/95
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n-Hexane	110-54-3	1/1/95
Hexazinone	51235-04-2	1/1/95

Chemical Name	CAS No.	Effective Date
Hydramethylnon [Tetrahydro-5,5-dimethyl-2(1H)-pyrimidinone[3-[4-(trifluoromethyl)phenyl]-1-[2-[4-(trifluoromethyl)phenyl]ethenyl]-2-propenylidene]hydrazone]	67485-29-4	1/1/95
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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
Iprodione [3-(3,5-Dichlorophenyl)-N-(1-methylethyl)-2,4-dioxo-1-imidazolidinecarboxamide]	36734-19-7	1/1/95
Iron pentacarbonyl	13463-40-6	1/1/95
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Isodrin	465-73-6	1/1/95
Isofenphos [2-[[Ethoxyl[(1-methylethyl)amino]phosphinothioyl]oxy]benzoic acid 1-methylethyl ester]	25311-71-1	1/1/95
Isophorone	78-59-1	1/1/95
Isophorone diisocyanate	4098-71-9	1/1/95
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Lactofen [5-(2-Chloro-4-(trifluoromethyl)phenoxy)-2-nitro-2-ethoxy-1- methyl-2-oxoethyl ester]	77501-63-4	1/1/95
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Linuron	330-55-2	1/1/95
Lithium carbonate	554-13-2	1/1/95
Malathion	121-75-5	1/1/95
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Mecoprop	93-65-2	1/1/95
2-Mercaptobenzothiazole (MBT)	149-30-4	1/1/95
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Merphos	150-50-5	1/1/95
Metham sodium (Sodium methyldithiocarbamate)	137-42-8	1/1/95
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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
Methoxone-sodium salt ((4-chloro-2-methylphenoxy) acetate sodium salt)	3653-48-3	1/1/95
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1,1'-Methylene bis(4-isocyanatocyclohexane)	5124-30-1	1/1/95
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Methylene bis(thiocyanate)	6317-18-6	1/1/95
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Methyl isothiocyanate [Isothiocyanatomethane]	556-61-6	1/1/95
2-Methyllactonitrile	75-86-5	1/1/95
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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
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Molinate (1H-Azepine-1-carbothioic acid, hexahydro-S-ethyl ester)	2212-67-1	1/1/95
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Monuron	150-68-5	1/1/95
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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
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Nitrapyrin (2-Chloro-6-(trichloromethyl) pyridine)	1929-82-4	1/1/95
Nitrate ion	14797-55-8	1/1/95
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Nitric oxide	10102-43-9	1/1/95
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p-Nitroaniline	100-01-6	1/1/95
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Nitrogen dioxide	10102-44-0	1/1/95
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Norflurazon [4-Chloro-5-(methylamino)-2-[3-(trifluoromethyl)phenyl]-3(2H)-pyridazinone]	27314-13-2	1/1/95

Chemical Name	CAS No.	Effective Date
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Oryzalin [4-(Dipropylamino)-3,5-dinitrobenzenesulfonamide]	19044-88-3	1/1/95
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Oxydemeton methyl [S-(2-(ethylsulfinyl)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
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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
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Pentobarbital sodium	57-33-0	1/1/95
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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
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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
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Phenytoin	57-41-0	1/1/95
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Phosphine	7803-51-2	1/1/95
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Phosphorous oxychloride	10025-87-3	1/1/95
Phosphorous pentachloride	10026-13-8	1/1/95
Phosphorous pentasulfide	1314-80-3	1/1/95
Phosphorous pentoxide	1314-56-3	1/1/95
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Picloram	1918-02-1	1/1/95
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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
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Potassium bromate	7758-01-2	1/1/95
Potassium dimethyldithiocarbamate	128-03-0	1/1/95
Potassium N-methyldithiocarbamate	137-41-7	1/1/95
Primisulfuron [Methyl 2-[[[[[4,6-bis(difluoromethoxy)-2-pyrimidinyl]- amino]carbonyl]amino]sulfonyl]benzoate]	86209-51-0	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
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Propanil [N-(3,4-Dichlorophenyl)propanamide]	709-98-8	1/1/95
Propargite	2312-35-8	1/1/95
Propargyl alcohol	107-19-7	1/1/95
Propetamphos [3-[[(Ethylamino)methoxyphosphinothioyl]oxy]-2-butenoic acid, 1-methylethyl ester]	31218-83-4	1/1/95
Propiconazole [1-[2-(2,4-Dichlorophenyl)-4-propyl-1,3-dioxolan-2-yl]- methyl-1H-1,2,4,-triazole]	60207-90-1	1/1/95
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Quizalofop-ethyl [2-[4-[(6-Chloro-2-quinoxalinyl)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
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Sethoxydim [2-[1-(Ethoxyimino)butyl]-5-[2-(ethylthio)propyl]-3-hydroxy-2-cyclohexen-1-one]	74051-80-2	1/1/95
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Simazine	122-34-9	1/1/95
Sodium azide	26628-22-8	1/1/95
Sodium chlorite	7758-19-2	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

Chemical Name	CAS No.	Effective Date
Sodium hypochlorite	7681-52-9	1/1/95
Sodium nitrite	7632-00-0	1/1/95
Sodium pentachlorophenate	131-52-2	
Sodium o-phenylphenoxide	132-27-4	1/1/95
Sodium 2-pyridinethiol-1-oxide	15922-78-8	1/1/95
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Sulfur dioxide	7446-09-5	1/1/95
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Sulfur trioxide	7446-11-9	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
Tefluthrin	79538-32-2	1/1/95
Temephos	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
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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
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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
Tetrasodium ethylenediaminetetraacetate	64-02-8	1/1/95
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Thiabendazole [2-(4-Thiazolyl)-1H-benzimidazole]	148-79-8	1/1/95
Thiabendazole, hypophosphite salt [2-(4-Thiazolyl)benzimidazole, hypophosphite salt]	28558-32-9	1/1/95
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Thiobencarb [Carbamic acid, diethylthio-, s-(p-chlorobenzyl)]	28249-77-6	1/1/95
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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
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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
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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
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Trichloroacetyl chloride	76-02-8	1/1/95
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Trichloroethylsilane	115-21-9	1/1/95
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Trichlorophenylsilane	98-13-5	
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
Triforine [N,N'-[1,4-Piperazinediyl-bis(2,2,2-trichloroethylidene)] bisformamide]	26644-46-2	1/1/95
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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
Triphenyltin hydroxide	76-87-9	1/1/95
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Vanadium pentoxide	1314-62-1	1/1/95
Vinclozolin [3-(3,5-Dichlorophenyl)-5-ethenyl-5-methyl-2,4- oxazolidinedione]	50471-44-8	1/1/95
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(b) * * *

CAS No.	Chemical Name	Effective Date
	* * * * * * *	
51-03-6	Piperonyl butoxide	1/1/95
51-21-8	Fluorouracil (5-Fluorouracil)	1/1/95
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52-51-7	2-Bromo-2-nitropropane-1,3-diol (Bronopol)	1/1/95
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52-85-7	Famphur	1/1/95
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55-38-9	Fenthion [O,O-Dimethyl O-[3-methyl-4-(methylthio)phenyl] ester, phosphorothioic acid]	1/1/95
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56-35-9	Bis(tributyltin) oxide	1/1/95
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57-33-0	Pentobarbital sodium	1/1/95
57-41-0	Phenytoin	1/1/95
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60-51-5	Dimethoate	1/1/95
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62-74-8	Sodium fluoroacetate	1/1/95
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64-02-8	Tetrasodium ethylenediaminetetraacetate	1/1/95
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64-75-5	Tetracycline hydrochloride	1/1/95
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68-12-2	N,N-Dimethylformamide	1/1/95
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75-43-4	Dichlorofluoromethane (HCFC-21)	1/1/95
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75-72-9	Chlorotrifluoromethane (CFC-13)	1/1/95
75-77-4	Trimethylchlorosilane	1/1/95
75-78-5	Dimethyldichlorosilane	1/1/95
75-79-6	Methyltrichlorosilane	1/1/95
75-86-5	2-Methyllactonitrile	1/1/95
75-88-7	2-Chloro-1,1,1-trifluoroethane (HCFC-133a)	1/1/95
76-02-8	Trichloroacetyl chloride	1/1/95
76-06-2	Chloropicrin	1/1/95
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76-87-9	Triphenyltin hydroxide	1/1/95
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77-73-6	Dicyclopentadiene	1/1/95
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78-48-8	S,S,S-Tributyltrithiophosphate (DEF)	1/1/95
78-59-1	Isophorone	1/1/95
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79-19-6	Thiosemicarbazide	1/1/95
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85-01-8	Phenanthrene	1/1/95
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88-85-7	Dinoseb	1/1/95
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93-65-2	Mecoprop	1/1/95
94-11-1	2,4-D isopropyl ester	1/1/95
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94-74-6	Methoxone (4-Chloro-2-methylphenoxy) acetic acid (MCPA)	1/1/95
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94-80-4	2,4-D butyl ester	1/1/95
94-82-6	2,4-DB	1/1/95
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95-54-5	1,2-Phenylenediamine	1/1/95

CAS No.	Chemical Name	Effective Date
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95-69-2	p-Chloro-o-toluidine	1/1/95
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96-18-4	1,2,3-Trichloropropane	1/1/95
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97-23-4	Dichlorophene [2,2'-Methylene-bis(4-chlorophenol)]	1/1/95
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98-13-5	Trichlorophenylsilane	1/1/95
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99-30-9	Dichloran [2,6-Dichloro-4-nitroaniline]	1/1/95
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100-01-6	p-Nitroaniline	1/1/95
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101-05-3	Anilazine [4,6-dichloro-N-(2-chlorophenyl)-1,3,5-triazin-2-amine]	1/1/95
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101-90-6	Diglycidyl resorcinol ether	1/1/95
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104-12-1	p-Chlorophenyl isocyanate	1/1/95
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105-60-2	Caprolactam	1/1/95
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106-47-8	p-Chloroaniline	1/1/95
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107-11-9	Allylamine	1/1/95
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107-19-7	Propargyl alcohol	1/1/95
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108-45-2	1,3-Phenylenediamine	1/1/95
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108-93-0	Cyclohexanol	1/1/95
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110-54-3	n-Hexane	1/1/95
110-57-6	trans-1,4-Dichloro-2-butene	1/1/95
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115-21-9	Trichloroethylsilane	1/1/95
115-28-6	Chlorendic acid	1/1/95
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116-06-3	Aldicarb	1/1/95
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120-32-1	o-Benzyl-p-chlorophenol	1/1/95
120-36-5	2,4-DP	1/1/95
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121-44-8	Triethylamine	1/1/95
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121-75-5	Malathion	1/1/95
122-34-9	Simazine	1/1/95
122-39-4	Diphenylamine	1/1/95
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124-40-3	Dimethylamine	1/1/95
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128-03-0	Potassium dimethyldithiocarbamate	1/1/95
128-04-1	Sodium dimethyldithiocarbamate	1/1/95
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131-52-2	Sodium pentachlorophenate	1/1/95
132-27-4	Sodium o-phenylphenoxide	1/1/95
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133-07-3	Folpet	1/1/95

CAS No.	Chemical Name	Effective Date
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136-45-8	Dipropyl isocinchomeronate	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
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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
149-74-6	Dichloromethylphenylsilane	1/1/95
150-50-5	Merphos	1/1/95
150-68-5	Monuron	1/1/95
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298-00-0	Methyl parathion	1/1/95
300-76-5	Naled	1/1/95
301-12-2	Oxydemeton methyl [s-(2-(Ethylsulfinyl)ethyl)o,o-dimethyl ester phosphorothioic acid]	1/1/95
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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
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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
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465-73-6	Isodrin	1/1/95
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507-55-1	1,3-dichloro-1,1,2,2,3-pentafluoropropane (HCFC-225cb)	1/1/95
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533-74-4	Dazomet (Tetrahydro-3,5-dimethyl-2H-1,3,5-thiadiazine-2-thione)	1/1/95
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541-53-7	2,4-Dithiobiuret	1/1/95
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542-76-7	3-Chloropropionitrile	1/1/95
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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
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576-26-1	2,6-Dimethylphenol	1/1/95
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594-42-3	Perchloromethyl mercaptan	1/1/95
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612-82-8	3,3'-Dimethylbenzidine dihydrochloride (o-Tolidine dihydrochloride)	1/1/95
612-83-9	3,3'-Dichlorobenzidine dihydrochloride	1/1/95
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615-28-1	1,2-Phenylenediamine dihydrochloride	1/1/95
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624-18-0	1,4-Phenylenediamine dihydrochloride	1/1/95
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630-08-0	Carbon monoxide	1/1/95
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639-58-7	Triphenyltin chloride	1/1/95
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709-98-8	Propanil [N-(3,4-Dichlorophenyl)propanamide]	1/1/95
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759-94-4	Ethyl dipropylthiocarbamate (EPTC)	1/1/95

CAS No.	Chemical Name	Effective Date
822-60-0	Hexamethylene-1,6-diisocyanate	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	N-Methylolacrylamide	1/1/95
957-51-7	Diphenamid	1/1/95
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1114-71-2	Pebulate [Butylethylcarbamo-thioic acid S-propyl ester]	1/1/95
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1134-23-2	Cycloate	1/1/95
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1314-56-3	Phosphorous pentoxide	1/1/95
1314-62-1	Vanadium pentoxide	1/1/95
1314-80-3	Phosphorous pentasulfide	1/1/95
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1320-18-9	2,4-D propylene glycol butyl ether ester	1/1/95
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1563-66-2	Carbofuran	1/1/95
1649-08-7	1,2-dichloro-1,1-difluoroethane (HCFC-132b)	1/1/95
1689-84-5	Bromoxynil (3,5-Dibromo-4-hydroxybenzonitrile)	1/1/95
1689-99-2	Bromoxynil octanoate (Octanoic acid, 2,6-dibromo-4-cyanophenyl ester)	1/1/95
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1861-40-1	Benfluralin(N-Butyl-N-ethyl-2,6-dinitro-4-(trifluoromethyl)benzenamine)	1/1/95
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1910-42-5	Paraquat dichloride	1/1/95
1912-24-9	Atrazine (6-Chloro-N-ethyl-N'-(1-methylethyl)-1,3,5,-triazine-2,4-diamine)	1/1/95
1917-97-1	2,4-D 2-octyl ester	1/1/95
1918-00-9	Dicamba (3,6-Dichloro-2-methyoxybenzoic acid)	1/1/95
1918-02-1	Picloram	1/1/95
1918-16-7	Propachlor [2-Chloro-N-(1-methylethyl)-N-phenylacetamide]	1/1/95
1928-43-4	2,4-D 2-ethylhexyl ester	1/1/95
1929-73-3	2,4-D butoxyethyl ester	1/1/95
1929-82-4	Nitrapyrin (2-Chloro-6-(trichloromethyl)pyridine)	1/1/95
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1982-69-0	Sodium dicamba [3,6-Dichloro-2-methoxybenzoic acid, sodium salt]	1/1/95
1983-10-4	Tributyltin fluoride	1/1/95
2008-41-5	Butylate (Bis-2-methylpropyl) carbamothioic acid S-ethyl ester)	1/1/95
2032-65-7	Methiocarb	1/1/95
2155-70-6	Tributyltin methacrylate	1/1/95
2164-07-0	Dipotassium endothall [7-Oxabicyclo(2.2.1)heptane-2,3-dicarboxylic acid, dipotassium salt]	1/1/95
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2212-67-1	Molinate (1H-Azepine-1-carbothioic acid, hexahydro-S-ethyl ester)	1/1/95
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2300-66-5	Dimethylamine dicamba	1/1/95
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2303-17-5	Triallate	1/1/95
2312-35-8	Propargite	1/1/95
2439-01-2	Chinomethionat [6-Methyl-1,3-dithiolo[4,5-b]quinoxalin-2-one]	1/1/95
2439-10-3	Dodine [Dodecylguanidine monoacetate]	1/1/95
2524-03-0	Dimethyl chlorothiophosphate	1/1/95
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2655-15-4	2,3,5-Trimethylphenyl methylcarbamate	1/1/95
2699-79-8	Sulfuryl Fluoride [Vikane]	1/1/95
2702-72-9	2,4-D sodium salt	1/1/95
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2971-38-2	2,4-D chlorocrotyl ester	1/1/95
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3380-34-5	5-Chloro-2-(2,4-dichlorophenoxy)phenol	1/1/95
3383-96-8	Temephos	1/1/95
3653-48-3	Methoxone - sodium salt (4-Chloro-2-methylphenoxy acetate sodium salt)	1/1/95
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4080-31-3	1-(3-Chloroallyl)-3,5,7-triaza-1-azoniaadamantane chloride	1/1/95
4098-71-9	Isophorone diisocyanate	1/1/95
4170-30-3	Crotonaldehyde	1/1/95
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5124-30-1	1,1'-Methylene bis(4-isocyanatocyclohexane)	1/1/95

CAS No.	Chemical Name	Effective Date
5234-68-4	Carboxin (5,6-Dihydro-2-methyl-N-phenyl-1,4-oxathiin-3-carboxamide)	1/1/95
5598-13-0	Chlorpyrifos methyl [O,O-dimethyl-O-(3,5,6-trichloro-2-pyridyl)phosphorothioate]	1/1/95
5902-51-2	Terbacil [5-Chloro-3-(1,1-dimethylethyl)-6-methyl-2,4-(1H,3H)-pyrimidinedione]	1/1/95
6317-18-6	Methylene bis(thiocyanate)	1/1/95
6459-94-5	C.I. Acid Red 114	1/1/95
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7287-19-6	Prometryn [N,N'-Bis(1-methylethyl)-6-methylthio-1,3,5-triazine-2,4-diamine]	1/1/95
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7446-09-5	Sulfur dioxide	1/1/95
7446-11-9	Sulfur trioxide	1/1/95
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7632-00-0	Sodium nitrite	1/1/95
7637-07-2	Boron trifluoride	1/1/95
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7681-52-9	Sodium hypochlorite	1/1/95
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
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7726-95-6	Bromine	1/1/95
7758-01-2	Potassium bromate	1/1/95
7758-19-2	Sodium chlorite	1/1/95
7778-54-3	Calcium hypochlorite	1/1/95
7782-41-4	Fluorine	1/1/95
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7786-34-7	Mevinphos	1/1/95
7803-51-2	Phosphine	1/1/95
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9006-42-2	Metiram	1/1/95
10025-87-3	Phosphorous oxychloride	1/1/95
10026-13-8	Phosphorous pentachloride	1/1/95
10028-15-6	Ozone	1/1/95
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10061-02-6	trans-1,3-Dichloropropene	1/1/95
10222-01-2	2,2-Dibromo-3-nitrilopropionamide	1/1/95
10102-43-9	Nitric oxide	1/1/95
10102-44-0	Nitrogen dioxide	1/1/95
10294-34-5	Boron trichloride	1/1/95
10453-86-8	Resmethrin [[5-(Phenylmethyl)-3-furanyl]methyl 2,2-dimethyl-3-(2-methyl-1-propenyl)cyclopropanecarboxylate]	1/1/95
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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(dimethylcarbamo-dithioato-S,S')iron]	1/1/95
14797-55-8	Nitrate ion	1/1/95
15922-78-8	Sodium 2-pyridinethiol-1-oxide	1/1/95
15972-60-8	Alachlor	1/1/95
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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
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20859-73-8	Aluminum phosphide	1/1/95
21087-64-9	Metribuzin	1/1/95
21725-46-2	Cyanazine	1/1/95
22781-23-3	Bendiocarb [2,2-Dimethyl-1,3-benzodioxol-4-ol 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
25013-16-5	Butylated hydroxyanisole	1/1/95
25311-71-1	Isofenphos [2-[[Ethoxyl[(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
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26628-22-8	Sodium azide	1/1/95
26644-46-2	Triforine [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

CAS No.	Chemical Name	Effective Date
28057-48-9	d-trans-Allethrin [d-trans-Chrysanthemic acid of d-allethrone]	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
28558-32-9	Thiabendazole, hypophosphite salt [2-(4-Thiazolyl)benzimidazole, hypophosphite salt]	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-butenoic acid, 1-methylethyl ester]	1/1/95
33089-61-1	Amitraz	1/1/95
34014-18-1	Terbuthiuron [N-[5-(1,1-Dimethylethyl)-1,3,4-thiadiazol-2-yl)-N,N'- dimethylurea]	1/1/95
35367-38-5	Diflubenzuron	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
36734-19-7	Iprodione [3-(3,5-Dichlorophenyl)-N-(1-methylethyl)-2,4-dioxo-1-imidazolidine-carboxamide]	1/1/95
38727-55-8	Diethatyl ethyl	1/1/95
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39300-45-3	Dinocap	1/1/95
39515-41-8	Fenpropathrin [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
51630-58-1	Fenvalerate	1/1/95
52645-53-1	Permethrin [3-(2,2-Dichloroethenyl)-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
66215-27-8	Cyromazine [N-Cyclopropyl-1,3,5-triazine-2,4,6-triamine]	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-ethenyl)-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-quinoxalinyl)oxy]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
79538-32-2	Tefluthrin	
81777-89-1	Clomazone [2-[(2-Chlorophenyl)methyl]-4,4-dimethyl-3-isoxazolidinone]	1/1/95
82657-04-3	Bifenthrin	1/1/95
86209-51-0	Primisulfuron [Methyl 2-[[[[[4,6-bis(difluoromethoxy)-2-pyrimidinyl]- amino]carbonyl]amino]sulfonyl]benzoate]	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
97886-45-8	Dithiopyr [2-(Difluoromethyl)-4-(2-methylpropyl)-6-(trifluoro-methyl)-3,5-pyridinedicarbothioic acid S,S-dimethyl ester]	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

(c) * * *

Category Name	Effective Date
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Chlorinated paraffins: Includes those chemicals defined by the following formula: CxH2x-y+2Cly where x = 10 to 30 and y = 3 to 26	1/1/95
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Man-made mineral fibers: Includes glass microfibers, glass wool fibers, rock wool fibers, slag wool fibers, and refractory ceramic fibers that have a diameter less than 3.5 micrometers and an aspect ratio greater than 3.	1/1/95
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Nicotine and salts	1/1/95
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Polycyclic Aromatic Compounds (PACs): (This category includes only those chemicals listed below) 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 00086-74-8 Carbazole 27208-37-3 Cyclopenta(cd)pyrene 00226-36-8 Dibenz(a,h)acridine 00224-42-0 Dibenz(a,j)acridine 00215-58-7 Dibenz(a,c)anthracene 00224-41-9 Dibenz(a,j)anthracene 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 03351-32-4 2-Methylchrysene 03351-31-3 3-Methylchrysene 03351-30-2 4-Methylchrysene 03697-24-3 5-Methylchrysene 01705-85-7 6-Methylchrysene 33543-31-6 2-Methylfluoranthene 05522-43-0 1-Nitropyrene	1/1/95
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Strychnine and salts	1/1/95
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