the Rules section of this **Federal Register**.

FOR FURTHER INFORMATION CONTACT: Zach Hedgpeth, U.S. EPA, Region 10, 1200 Sixth Avenue, Mail stop WCM–122, Seattle, Washington 98101, e-mail: *hedgpeth.zach@epa.gov,* phone number (206) 553–1217.

SUPPLEMENTARY INFORMATION: In the "Rules and Regulations" section of this **Federal Register**, the EPA is codifying and incorporating by reference the State's hazardous waste program as an direct final rule. The EPA did not make a proposal prior to the direct final rule because we believe these actions are not controversial and do not expect comments that oppose them. We have explained the reasons for this codification and incorporation by reference in the preamble to the direct final rule. Unless we get written comments which oppose this incorporation by reference during the comment period, the direct final rule will become effective on the date it establishes, and we will not take further action on this proposal. If we get comments that oppose these actions, we will withdraw the direct final rule and it will not take effect. We will then respond to public comments in a later final rule based on this proposal. You may not have another opportunity for comment. If you want to comment on this action, you must do so at this time. For additional information, please see the direct final rule published in the "Rules and Regulations" section of this **Federal Register**.

Authority: This action is issued under the authority of sections 2002(a), 3006 and 7004(b) of the Solid Waste and Disposal Act, as amended, 42 U.S.C. 6912(a), 6926, 6974(b).

Dated: March 11, 2010.

Dennis J. McLerran,

Regional Administrator, EPA Region 10.

[FR Doc. 2010–7649 Filed 4–5–10; 8:45 am]

BILLING CODE 6560–50–P

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 372

[EPA–HQ–TRI–2010–0006; FRL–9134–1]

RIN 2025–AA28

Addition of National Toxicology Program Carcinogens; Community Right-to-Know Toxic Chemical Release Reporting

AGENCY: Environmental Protection Agency (EPA).

ACTION: Proposed rule.

SUMMARY: EPA is proposing to add sixteen chemicals to the list of toxic chemicals subject to reporting under section 313 of the Emergency Planning and Community Right-to-Know Act (EPCRA) of 1986 and section 6607 of the Pollution Prevention Act of 1990 (PPA). These sixteen chemicals have been classified by the National Toxicology Program (NTP) in their Report on Carcinogens (RoC) as "reasonably anticipated to be a human carcinogen." EPA believes that these sixteen chemicals meet the EPCRA section 313(d)(2)(B) criteria because they can reasonably be anticipated to cause cancer in humans. As in past chemical reviews, EPA adopted a production volume screen for the development of this proposed rule to screen out those chemicals for which no reports are expected to be submitted. Based on a review of the available production and use information, these sixteen chemicals are expected to be manufactured, processed, or otherwise used in quantities that would exceed the EPCRA section 313 reporting thresholds.

DATES: Comments must be received on or before June 7, 2010.

ADDRESSES: Submit your comments, identified by Docket ID No. EPA–HQ–TRI–2010–0006, by one of the following methods:

- *www.regulations.gov*: Follow the on-line instructions for submitting comments.
- E-mail: *oei.docket@epa.gov.*
- *Mail:* Office of Environmental Information (OEI) Docket, Environmental Protection Agency, Mail Code: 28221T, 1200 Pennsylvania Ave., NW., Washington, DC 20460
- *Hand Delivery:* EPA Docket Center (EPA/DC), EPA West, Room 3334, 1301 Constitution Ave., NW., Washington, DC 20460. Such deliveries are only accepted during the Docket's normal hours of operation, and special arrangements should be made for deliveries of boxed information.

Instructions: Direct your comments to Docket ID No. EPA–HQ–TRI–2010–0006. EPA's policy is that all comments received will be included in the public docket without change and may be made available online at http://*www.regulations.gov,* including any personal information provided, unless the comment includes information claimed to be Confidential Business Information (CBI) or other information whose disclosure is restricted by statute. Do not submit information that you consider to be CBI or otherwise protected through www.regulations.gov or e-mail. The *www.regulations.gov* Web site is an "anonymous access" system, which means EPA will not know your identity or contact information unless you provide it in the body of your comment. If you send an e-mail comment directly to EPA without going through www.regulations.gov, your e-mail address will be automatically captured and included as part of the comment that is placed in the public docket and made available on the Internet. If you submit an electronic comment, EPA recommends that you include your name and other contact information in the body of your comment and with any disk or CD–ROM you submit. If EPA cannot read your comment due to technical difficulties and cannot contact you for clarification, EPA may not be able to consider your comment. Electronic files should avoid the use of special characters, avoid any form of encryption, and be free of any defects or viruses.

Docket: All documents in the docket are listed in the *www.regulations.gov* index. Although listed in the index, some information is not publicly available, *e.g.,* CBI or other information whose disclosure is restricted by statute. Certain other material, such as copyrighted material, will be publicly available only in hard copy. Publicly available docket materials are available either electronically in *www.regulations.gov* or in hard copy at the OEI Docket, EPA/DC, EPA West, Room 3334, 1301 Constitution Ave., NW., Washington, DC. This Docket Facility is open from 8:30 a.m. to 4:30 p.m., Monday through Friday, excluding legal holidays. The telephone number for the Public Reading Room is (202) 566–1744, and the telephone number for the OEI Docket is (202) 566–1752.

FOR FURTHER INFORMATION CONTACT: Daniel R. Bushman, Environmental Analysis Division, Office of Information Analysis and Access (2842T), Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460; *telephone number:* 202–566–0743; *fax number:* 202–566–0677; *e-mail: bushman.daniel@epa.gov*, for specific information on this notice. For general information on EPCRA section 313, contact the Emergency Planning and Community Right-to-Know Hotline, toll free at (800) 424–9346 or (703) 412–9810 in Virginia and Alaska or toll free, TDD (800) 553–7672, *http://www.epa.gov/epaoswer/hotline/.*

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Notice Apply to Me?

You may be potentially affected by this action if you manufacture, process, or otherwise use any of the chemicals

included in this proposed rule. Potentially affected categories and entities may include, but are not limited to:

Category	Examples of potentially affected entities
Industry	Facilities included in the following NAICS manufacturing codes (corresponding to SIC codes 20 through 39): 311*, 312*, 313*, 314*, 315*, 316, 321, 322, 323*, 324, 325*, 326*, 327, 331, 332, 333, 334*, 335*, 336, 337*, 339*, 111998*, 211112*, 212324*, 212325*, 212393*, 212399*, 488390*, 511110, 511120, 511130, 511140*, 511191, 511199, 512220, 512230*, 519130*, 541712*, or 811490*. * Exceptions and/or limitations exist for these NAICS codes. Facilities included in the following NAICS codes (corresponding to SIC codes other than SIC codes 20 through 39): 212111, 212112, 212113 (correspond to SIC 12, Coal Mining (except 1241)); or 212221, 212222, 212231, 212234, 212299 (correspond to SIC 10, Metal Mining (except 1011, 1081, and 1094)); or 221111, 221112, 221113, 221119, 221121, 221122, 221330 (Limited to facilities that combust coal and/or oil for the purpose of generating power for distribution in commerce) (correspond to SIC 4911, 4931, and 4939, Electric Utilities); or 424690, 425110, 425120 (Limited to facilities previously classified in SIC 5169, Chemicals and Allied Products, Not Elsewhere Classified); or 424710 (corresponds to SIC 5171, Petroleum Bulk Terminals and Plants); or 562112 (Limited to facilities primarily engaged in solvent recovery services on a contract or fee basis (previously classified under SIC 7389, Business Services, NEC)); or 562211, 562212, 562213, 562219, 562920 (Limited to facilities regulated under the Resource Conservation and Recovery Act, subtitle C, 42 U.S.C. 6921 *et seq.*) (correspond to SIC 4953, Refuse Systems).
Federal Government	Federal facilities.

This table is not intended to be exhaustive, but rather provides a guide for readers regarding entities likely to be affected by this action. Some of the entities listed in the table have exemptions and/or limitations regarding coverage, and other types of entities not listed in the table could also be affected. To determine whether your facility would be affected by this action, you should carefully examine the applicability criteria in part 372 subpart B of Title 40 of the Code of Federal Regulations. If you have questions regarding the applicability of this action to a particular entity, consult the person listed in the preceding "**FOR FURTHER INFORMATION CONTACT**" section.

B. How Should I Submit CBI to the Agency?

Do not submit CBI information to EPA through *www.regulations.gov* or e-mail. Clearly mark the part or all of the information that you claim to be CBI. For CBI information in a disk or CD–ROM that you mail to EPA, mark the outside of the disk or CD–ROM as CBI and then identify electronically within the disk or CD–ROM the specific information that is claimed as CBI. In addition to one complete version of the comment that includes information claimed as CBI, a copy of the comment that does not contain the information claimed as CBI must be submitted for inclusion in the public docket. Information so marked will not be disclosed except in accordance with procedures set forth in 40 CFR part 2.

II. Introduction

Section 313 of EPCRA, 42 U.S.C. 11023, requires certain facilities that manufacture, process, or otherwise use listed toxic chemicals in amounts above reporting threshold levels to report their environmental releases and other waste management quantities of such chemicals annually. These facilities must also report pollution prevention and recycling data for such chemicals, pursuant to section 6607 of the PPA, 42 U.S.C. 13106. Congress established an initial list of toxic chemicals that comprised more than 300 chemicals and 20 chemical categories.

EPCRA section 313(d) authorizes EPA to add or delete chemicals from the list and sets criteria for these actions. EPCRA section 313(d)(2) states that EPA may add a chemical to the list if any of the listing criteria in Section 313(d)(2) are met. Therefore, to add a chemical, EPA must demonstrate that at least one criterion is met, but need not determine whether any other criterion is met. Conversely, to remove a chemical from the list, EPCRA section 313(d)(3) dictates that EPA must demonstrate that none of the listing criteria in Section 313(d)(2) are met. The EPCRA section 313(d)(2) criteria are:

(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 be reasonably 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 judgment of the Administrator, to warrant reporting under this section.

EPA often refers to the section 313(d)(2)(A) criterion as the "acute human health effects criterion;" the section 313(d)(2)(B) criterion as the "chronic human health effects criterion;" and the section 313(d)(2)(C) criterion as the "environmental effects criterion."

EPA has published in the **Federal Register** of November 30, 1994 (59 FR 61432) a statement clarifying its interpretation of the section 313(d)(2) and (d)(3) criteria for modifying the section 313 list of toxic chemicals.

III. Background Information

A. What is the NTP and the Report on Carcinogens?

The National Toxicology Program (NTP) is an interagency program within the Department of Health and Human Services (DHHS) headquartered at the National Institute of Environmental Health Sciences (NIEHS) of the National Institutes of Health (NIH). The mission of the NTP is to evaluate chemicals of public health concern by developing and applying tools of modern toxicology and molecular biology. The NTP

program maintains an objective, science-based approach in dealing with critical issues in toxicology and is committed to using the best science available to prioritize, design, conduct, and interpret its studies. The mission of the NTP includes the evaluation of chemicals for their potential to cause cancer in humans.

As part of their cancer evaluation work, the NTP periodically publishes a Report on Carcinogens (RoC) document. The RoC was mandated by the U.S. Congress, as part of the Public Health Service Act (Section 301(b)(4), as amended). The NTP describes the RoC as an informational scientific and public health document that identifies and discusses agents, substances, mixtures, or exposure circumstances that may pose a hazard to human health by virtue of their carcinogenicity. The NTP RoC serves as a meaningful and useful compilation of data on (1) the carcinogenicity (ability to cause cancer), genotoxicity (ability to damage genes), and biologic mechanisms (modes of action in the body) of the RoC-listed substances in humans and/or in animals, (2) the potential for human exposure to these substances, and (3) the regulations and guidelines promulgated by Federal agencies to limit exposures to RoC-listed substances. The NTP RoC is published periodically, with the most recently published 11th RoC having been released on January 31, 2005. The 11th RoC contains the NTP cancer classifications from the most recent chemical evaluations as well as the classifications from previous versions of the RoC.

B. What are the NTP cancer classifications and criteria?

The NTP RoC classifies chemicals as either "known to be a human carcinogen" or "reasonably anticipated to be a human carcinogen." The criteria that the NTP uses to list an agent, substance, mixture, or exposure circumstance under each classification in the RoC (Ref. 1) are as follows:

"*Known To Be Human Carcinogen:*
There is sufficient evidence of carcinogenicity from studies in humans*, which indicates a causal relationship between exposure to the agent, substance, or mixture, and human cancer.

Reasonably Anticipated To Be Human Carcinogen:
There is limited evidence of carcinogenicity from studies in humans*, which indicates that causal interpretation is credible, but that alternative explanations, such as chance, bias, or confounding factors, could not adequately be excluded,
or
there is sufficient evidence of carcinogenicity from studies in experimental animals, which indicates there is an increased incidence of malignant and/or a combination of malignant and benign tumors (1) in multiple species or at multiple tissue sites, or (2) by multiple routes of exposure, or (3) to an unusual degree with regard to incidence, site, or type of tumor, or age at onset,
or
there is less than sufficient evidence of carcinogenicity in humans or laboratory animals; however, the agent, substance, or mixture belongs to a well-defined, structurally related class of substances whose members are listed in a previous Report on Carcinogens as either known to be a human carcinogen or reasonably anticipated to be a human carcinogen, or there is convincing relevant information that the agent acts through mechanisms indicating it would likely cause cancer in humans. Conclusions regarding carcinogenicity in humans or experimental animals are based on scientific judgment, with consideration given to all relevant information. Relevant information includes, but is not limited to, dose response, route of exposure, chemical structure, metabolism, pharmacokinetics, sensitive sub-populations, genetic effects, or other data relating to mechanism of action or factors that may be unique to a given substance. For example, there may be substances for which there is evidence of carcinogenicity in laboratory animals, but there are compelling data indicating that the agent acts through mechanisms which do not operate in humans and would therefore not reasonably be anticipated to cause cancer in humans.

* This evidence can include traditional cancer epidemiology studies, data from clinical studies, and/or data derived from the study of tissues or cells from humans exposed to the substance in question that can be useful for evaluating whether a relevant cancer mechanism is operating in people."

The NTP classifications for the potential for a chemical to cause cancer are very similar to the EPCRA section 313(d)(2)(B) statutory criteria for listing a chemical on the list of toxic chemicals subject to reporting under EPCRA section 313: "(B) The chemical is known to cause or can reasonably be anticipated to cause in humans— (i) cancer * * * " The specific data used by the NTP to classify a chemical as "Known To Be Human Carcinogen" or "Reasonably Anticipated To Be Human Carcinogen" are consistent with data used by EPA to evaluate chemicals for their potential to cause cancer and classify chemicals as either "Carcinogenic to Humans" or "Likely to Be Carcinogenic to Humans" (Ref. 2).

C. What is the review process for the RoC?

Specific details of the nomination and review process for the development of the 11th RoC are described in the introduction to the 11th RoC (Ref. 1). In general, the RoC review process includes evaluations by scientists from the NTP, other Federal health research and regulatory agencies (including EPA), and nongovernmental institutions. The RoC review process includes external peer review and several opportunities for public comment. For the 11th RoC, two Federal scientific review groups, the NIEHS/NTP Review Committee for the Report on Carcinogens RG1 and the NTP Executive Committee Interagency Working Group for the Report on Carcinogens RG2, evaluated the classification recommendations. An EPA representative was a member of the RG2 committee. These reviews were followed by a third independent external scientific peer review by a standing subcommittee of the NTP Board of Scientific Counselors (the RoC Subcommittee). During the entire process there were three opportunities for public comment. The Director of the NTP received for review all of the recommendations of the review groups, the opinion of the NTP Executive Committee, and all public comments. After evaluating this information and any other relevant information the NTP Director developed recommendations to the Secretary, DHHS regarding whether and/or how to classify nominations in the RoC. The final draft of the RoC was prepared by the NTP based on the NTP Director's recommendations and was submitted it to the Secretary, DHHS, for review and approval. Once approved, the Secretary submitted RoC to the U. S. Congress as a final document. Submittal of the RoC to Congress constituted publication of the report, at which time it became available to the public.

IV. EPA's Review of the 11th RoC

A. How did EPA select the NTP RoC chemicals being proposed for addition?

The most recent version of the NTP RoC that EPA previously reviewed for

possible additions to the EPCRA section 313 list was the 6th RoC (January 12, 1994, 59 FR 1788). Each new version of the RoC adds newly classified chemicals to the existing list. EPA's present review of the 11th RoC identified 81 chemicals that are not on the EPCRA section list, 54 of which were previously reviewed for listing when EPA reviewed the 6th RoC. Those previous reviews concluded that the 54 chemicals that were not proposed for addition would not be manufactured, processed, or otherwise used at levels that exceed the EPCRA section 313 reporting thresholds. For this review EPA only considered the 27 chemicals that had been added to the RoC since the 6th RoC was published and thus had not been previously reviewed for listing. Of the 27 chemicals, EPA determined that 12 are manufactured, processed, or otherwise used in quantities sufficient to exceed reporting thresholds for at least one facility (Ref. 3). In addition, 4 chemicals are included for addition to the polycyclic aromatic compounds category.

Section 313(d)(2) of EPCRA provides EPA the discretion to add chemicals to the TRI list when there is sufficient evidence to establish any of the listing criteria. EPA can add a chemical that meets one criterion regardless of its production volume. But as in past chemical reviews (*e.g.,* January 12, 1994, 59 FR 1788), EPA adopted a production volume screen for the development of this proposed rule to screen out those chemicals for which no reports are expected to be submitted. If chemicals that did not meet the production volume screen were listed, there would be an economic burden for firms that would have to determine that they did not exceed the reporting threshold. Yet as no reports would be filed, there would be no information to the public on these chemicals. EPA feels it is appropriate at this time to focus on chemicals for which reports are likely to be filed.

EPA reviewed the NTP 11th RoC chemical profiles and supporting materials for each chemical being proposed for listing in this rule (Ref. 4). Given the extensive scientific reviews conducted by the NTP for their RoC documents, EPA's review focused on ensuring that there were no inconsistencies with how the Agency would consider the available data. EPA found no inconsistencies and agrees with the hazard conclusions of the NTP 11th RoC for each of the chemicals included in this proposed rule.

B. What technical data supports the NTP RoC classifications and EPA's proposed additions to the EPCRA section 313 list?

This section presents the data that supported the NTP 11th RoC classifications of each chemical now being proposed for inclusion on the EPCRA section 313 list and why EPA believes the data support the addition of these chemicals to the EPCRA section 313 list. The NTP chemical profiles, the NTP chemical background documents, and the references cited within each of the portions of the NTP 11th RoC chemical profiles quoted here, are all included in the docket for this rulemaking. While they are contained in the docket and are part of the rulemaking record, the references within the quotations cited from the NTP 11th RoC profile documents in this section are not included in the list of references in Unit VI. of this Federal Register notice. The full citations for the references contained in the quotations can be found in the NTP 11th RoC profile documents cited for each chemical.

1. *1-Amino-2,4-Dibromoanthraquinone* (CAS No. 81–49–2) (Refs. NTP Profile/Background document (Refs. 5 and 6)). The NTP has classified 1-amino-2,4-dibromoanthraquinone as "reasonably anticipated to be a human carcinogen." The classification is based on sufficient evidence of carcinogenicity in experimental animals. The NTP substance profile for 1-amino-2,4-dibromoanthraquinone (Ref. 5) included the following summary information of the evidence of carcinogenicity:

"Carcinogenicity

1-Amino-2,4-dibromoanthraquinone (ADBAQ) is *reasonably anticipated to be a human carcinogen* based on sufficient evidence from studies in experimental animals. Orally administered ADBAQ significantly increased the incidences of benign and/or malignant tumors at multiple tissue sites in two species of animals. ADBAQ caused benign and malignant liver tumors in rats and mice of both sexes; tumors of the large intestine, kidney, and urinary bladder in male and female rats; and tumors of the forestomach and lung in male and female mice (NTP 1996).

Two cohort studies evaluated the risk of cancer among workers in plants manufacturing anthraquinone dyes; however, it is not known whether workers were exposed specifically to ADBAQ (Gardiner *et al.* 1982, Delzell *et al.* 1989). Some evidence suggests that anthraquinone dye workers may have an increased risk of cancer. Significant excesses of esophageal and prostate cancer occurred among workers in some areas of a Scottish anthraquinone dyestuffs plant, and excesses of lung and central nervous system cancer occurred among workers at a New Jersey anthraquinone dye and epichlorohydrin plant (Barbone *et al.* 1992, 1994, Sathiakumar and Delzell 2000). Nevertheless, estimates of risk in all studies were based on small numbers of cancer deaths, and workers may have been exposed to other carcinogens.

Additional Information Relevant to Carcinogenicity

Evaluation of ADBAQ's genetic effects has been hindered by ADBAQ's limited solubility. ADBAQ caused mutations in some strains of bacteria but not in rodent cells, which were tested at lower concentrations (Haworth *et al.* 1983, NTP 1996). In mammalian cells, ADBAQ induced chromosomal aberrations (changes in chromosome structure or number) and sister chromatid exchange; however, the results varied between laboratories and between trials at the same laboratory (Loveday *et al.* 1990, NTP 1996). Point mutations in the *ras* proto-oncogene (a gene potentially associated with cancer) occurred at a higher frequency in forestomach and lung tumors from the two-year carcinogenicity study of ADBAQ-exposed mice than in spontaneous tumors from control mice not exposed to ADBAQ. The predominant types of mutations were A to T transversions and A to G transitions, suggesting that ADBAQ or its metabolites target adenine bases in the ras proto-oncogene (Hayashi *et al.* 2001).

ADBAQ is rapidly absorbed from the gastrointestinal tract and distributed to most soft tissues. The majority of ADBAQ is metabolized, and both ADBAQ and its metabolites are excreted in the feces and urine. However, the metabolites of ADBAQ have not been identified (NTP 1996). The mechanism by which ADBAQ causes cancer is not known; however, there is no evidence to suggest that mechanisms of tumor induction observed in experimental animals would not occur in humans. Four other anthraquinones (2-aminoanthraquinone, 1-amino-2-methylanthraquinone, danthron [1,8-dihydroxyanthraquinone], and disperse blue 1) are listed in the Report on Carcinogens as *reasonably anticipated to be human carcinogens.*"

EPA has reviewed the NTP assessment for 1-amino-2,4-dibromoanthraquinone and agrees that

1-amino-2,4-dibromoanthraquinone can reasonably be anticipated to cause cancer in humans. EPA believes that the evidence is sufficient for listing 1-amino-2,4-dibromoanthraquinone on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for this chemical.

2. 2,2-*bis(Bromomethyl)-1,3-propanediol* (CAS No. 3296–90–0) (Refs. NTP Profile/Background document (Refs. 7 and 8)). The NTP has classified 2,2-bis(bromomethyl)-1,3-propanediol as "reasonably anticipated to be a human carcinogen." The classification is based on sufficient evidence of carcinogenicity in experimental animals. The NTP substance profile for 2,2-bis(bromomethyl)-1,3-propanediol (Ref. 7) included the following summary information of the evidence of carcinogenicity:

Carcinogenicity

The flame retardant 2,2-bis(bromomethyl)-1,3-propanediol, technical grade (BBMP), is *reasonably anticipated to be a human carcinogen* based on sufficient evidence of carcinogenicity from studies in experimental animals which indicates there is increased incidence of malignant tumor formation at multiple tissue sites in rats and mice. Two year dietary studies of BBMP in F344 rats showed significantly increased incidences of neoplasms of the skin, subcutaneous tissue, mammary gland, Zymbal gland, oral cavity, esophagus, forestomach, small and large intestines, mesothelium, urinary bladder, lung, thyroid gland, and seminal vesicle and in the incidence of mononuclear cell leukemia in males, and an increase in the incidence of neoplasms of the oral cavity, esophagus, mammary gland, and thyroid gland in females. Similar studies in B6C3F1 mice found increased incidences of neoplasms of the harderian gland, lung, and kidney in males and neoplasms of the harderian gland, lung, and subcutaneous tissue in females (NTP 1996, Dunnick *et al.* 1997).

A study in which BBMP was administered in the feed to male F344 rats for three months, followed by maintenance on a control diet for up to two years, found neoplasms at the same sites as in the two-year study of male F344 rats described above. However, this study found higher incidences of neoplasms of the oral cavity, forestomach, small intestine, large intestine, lung, Zymbal gland, thyroid gland, and mesothelium than did the two-year study; these neoplasms were considered to be related to BBMP exposure (NTP 1996, Dunnick *et al.* 1997).

No published case reports or epidemiological studies of human cancer and exposure to BBMP were found (IARC 2000).

Additional Information Relevant to Carcinogenicity

BBMP has been shown to be mutagenic in bacterial and mammalian test systems, under special conditions. BBMP is mutagenic in *Salmonella typhimurium* strains TA100 and TA1535 only when tested in the presence of metabolic activation (30% S9 liver homogenate from induced hamsters) (Zeiger *et al.* 1992). In cultured Chinese hamster ovary cells, BBMP induces chromosomal aberrations only in the presence of metabolic activation, and it does not induce sister chromatid exchange with or without activation. Male and female mice exposed to BBMP under various conditions showed significant increases in the frequency of micronucleated erythrocytes (NTP 1996).

No available data suggest that mechanisms thought to account for BBMP's induction of tumors in experimental animals would not also operate in humans."

EPA has reviewed the NTP assessment for 2,2-bis(bromomethyl)-1,3-propanediol and agrees that 2,2-bis(bromomethyl)-1,3-propanediol can reasonably be anticipated to cause cancer in humans. EPA believes that the evidence is sufficient for listing 2,2-bis(bromomethyl)-1,3-propanediol on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for this chemical.

3. *Furan* (CAS No. 110–00–9) (Refs. NTP Profile/Background document (Refs. 9 and 10)). The NTP has classified furan as "reasonably anticipated to be a human carcinogen." The classification is based on sufficient evidence of carcinogenicity in experimental animals. The NTP substance profile for furan (Ref. 9) included the following summary information of the evidence of carcinogenicity:

"Carcinogenicity

Furan is *reasonably anticipated to be a human carcinogen* based on sufficient evidence of malignant tumor formation at multiple tissue sites in multiple species of experimental animals (IARC 1995).

When administered by gavage, furan induced an increase in the incidence of hepatic cholangiocarcinoma, hepatocellular adenoma, hepatocellular carcinoma, and mononuclear cell leukemia in male and female F344/N rats treated for up to 2 years (NTP 1993). Gavage administration of furan to male F344 rats for 9, 12, or 13 months resulted in high incidences of cholangiocarcinoma by 16 months after cessation of treatment (Maronpot *et al.* 1991, Elmore and Sirica 1993). When administered by gavage, furan induced a dose-dependent increase in the incidence of hepatocellular adenoma and carcinoma and benign pheochromocytoma in male and female $B6C3F_1$ mice treated up to 2 years (NTP 1993).

No adequate human studies of the relationship between exposure to furan and human cancer have been reported.

Additional Information Relevant to Carcinogenicity

In bacteria, furan induced gene mutations in *Salmonella typhimurium* strain TA100 (Lee *et al.* 1994) and in *E. coli* containing bacteriophage T7 (Ronto *et al.* 1992), but not in S. *typhimurium strains* TA98 (Lee *et al.* 1994), TA1535, or TA1537 (Mortelmans *et al.* 1986). In *Drosophila melanogaster,* it did not induce gene mutations (Foureman *et al.* 1994). In mammalian *in vitro* systems, it induced gene mutations in mouse lymphoma cells (McGregor *et al.* 1988), DNA damage in Chinese hamster ovary (CHO) cells (NTP 1993), and chromosomal damage in CHO cells with an exogenous metabolic activation system (NTP 1993, IARC 1995), but it did not induce DNA damage in mouse or rat hepatocytes (Wilson *et al.* 1992, NTP 1993). In mammalian *in vivo* systems, furan induced chromosomal aberrations in bone marrow of $B6C3F_1$ mice (NTP 1993), but did not induce DNA damage in bone marrow or hepatocytes of $B6C3F_1$ mice (Wilson *et al.* 1992, NTP 1993) or hepatocytes of F344/CrIBr rats (Wilson *et al.* 1992).

A current hypothesis for the mechanism of furan-induced carcinogenesis is metabolic activation of furan by cytochrome P450 to a reactive and cytotoxic intermediate that stimulates cell replication, increasing the likelihood of tumor induction (Chen *et al.* 1995, Kedderis *et al.* 1993). The postulated reactive metabolite is *cis*-2-butene-1,4-dial, which was recently characterized as a furan metabolite by Chen *et al.* (1995). This reactive metabolite probably explains furan's binding reactivity with proteins both *in vitro* (uninduced and induced F344 male rat liver microsomes) and in vivo (F344 male rat liver protein) in biological systems (Burka *et al.* 1991, Parmar and Burka 1993). Furan metabolites may react with DNA, but Burka *et al.* (1991) did not detect any

radiotracer in DNA from livers of rats treated with [^{14}C]furan.

No data were available that would suggest that the mechanisms thought to account for tumor induction by furan in experimental animals would not also operate in humans."

EPA has reviewed the NTP cancer assessment for furan and agrees that furan can reasonably be anticipated to cause cancer in humans. EPA believes that the evidence is sufficient for listing furan on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for this chemical.

4. *Glycidol* (CAS No. 556–52–5) (Ref. NTP Profile/NTP study (Refs. 11 and 12)). The NTP has classified glycidol as "reasonably anticipated to be a human carcinogen." The classification is based on sufficient evidence of carcinogenicity in experimental animals. The NTP substance profile for glycidol (Ref. 11) included the following summary information of the evidence of carcinogenicity:

"Carcinogenicity

Glycidol is *reasonably anticipated to be a human carcinogen* based on sufficient evidence of carcinogenicity in experimental animals (NTP 1990, IARC 2000). Two-year studies were conducted with mice and rats that were administered glycidol by gavage. Male rats showed increased incidences of mesotheliomas of the tunica vaginalis, fibroadenomas of the mammary gland, gliomas of the brain, and neoplasms of the forestomach, intestine, skin, Zymbal gland, and thyroid gland. Female rats had increased incidences of fibroadenomas and adenocarcinomas of the mammary gland, gliomas of the brain, neoplasms of the oral mucosa, forestomach, clitoral gland, and thyroid gland, and leukemia. Male B6C3F$_1$ mice had increased incidences of neoplasms of the harderian gland, forestomach, skin, liver, and lung. Female B6C3F$_1$ mice had increased incidences of neoplasms of the harderian gland, mammary gland, uterus, subcutaneous tissue, and skin. Other neoplasms that may be related to the administration of glycidol were fibrosarcomas of the glandular stomach in female rats and carcinomas of the urinary bladder and sarcomas of the epididymis in male mice (NTP 1990).

No adequate human studies of the relationship between exposure to glycidol and human cancer have been reported (IARC 2000)."

EPA has reviewed the NTP cancer assessment for glycidol and agrees that glycidol can reasonably be anticipated to cause cancer in humans. EPA believes that the evidence is sufficient for listing glycidol on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for this chemical.

5. *Isoprene* (CAS No. 78–79–5) (Refs. NTP Profile/Background document (Refs. 13 and 14)). The NTP has classified isoprene as "reasonably anticipated to be a human carcinogen." The classification is based on sufficient evidence of carcinogenicity in experimental animals. The NTP substance profile for isoprene (Ref. 13) included the following summary information of the evidence of carcinogenicity:

Carcinogenicity

Isoprene is *reasonably anticipated to be a human carcinogen* based on sufficient evidence of tumor formation at multiple organ sites in multiple species of experimental animals (Melnick *et al.* 1994, NTP 1995, 199[9], Placke *et al.* 1996). Inhalation exposure of mice to isoprene vapors induced increased incidences of neoplasms of the lung, liver, harderian gland, forestomach, hematopoietic system, and circulatory system. Inhalation exposure of rats to isoprene vapors induced increased incidences of neoplasms of the mammary gland, kidney, and testis (IARC 1999).

No adequate human studies of the relationship between exposure to isoprene and human cancer have been reported.

Additional Information Relevant to Carcinogenicity

Isoprene is the 2-methyl analog of 1,3-butadiene, an industrial chemical that has been identified as an animal and human carcinogen. Isoprene and butadiene are metabolized to monoepoxide and diepoxide intermediates by liver microsomal cytochrome P450-dependent monooxygenases from several species, including humans. Detoxification of these intermediates may occur by hydrolysis catalyzed by epoxide hydrolase or conjugation with glutathione catalyzed by glutathione-*S*-transferase. The diepoxide intermediates of isoprene and butadiene are mutagenic in *Salmonella typhimurium,* whereas the parent compounds are inactive (Gervasi *et al.* 1985). In mice, isoprene and 1,3-butadiene induced sister chromatid exchanges in bone marrow cells and increased the frequency of micronucleated erythrocytes in peripheral blood (Tice *et al.* 1987, Tice *et al.* 1988). Common sites of neoplasm induction by isoprene and butadiene include the mammary gland and testis in rats, and the liver, lung, harderian gland, forestomach, and circulatory system in mice (NTP 199[9]). Lung and harderian gland neoplasms induced by isoprene in mice had a high frequency of unique K-*ras* mutations (A to T transversions at codon 61) (Hong *et al.* 1997).

No data were available that would suggest that mechanisms thought to account for tumor induction by isoprene in experimental animals would not also operate in humans.

EPA has reviewed the NTP cancer assessment for isoprene and agrees that isoprene can reasonably be anticipated to cause cancer in humans. EPA believes that the evidence is sufficient for listing isoprene on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for this chemical.

6. *Methyleugenol* (CAS No. 93–15–2) (Refs. NTP Profile/Background document (Refs. 15 and 16)). The NTP has classified methyleugenol as "reasonably anticipated to be a human carcinogen." The classification is based on sufficient evidence of carcinogenicity in experimental animals. The NTP substance profile for methyleugenol (Ref. 15) included the following summary information of the evidence of carcinogenicity:

Carcinogenicity

Methyleugenol is *reasonably anticipated to be a human carcinogen* based on sufficient evidence of carcinogenicity from studies in experimental animals, which indicates there is an increased incidence of malignant and/or combination of malignant and benign tumors at multiple tissue sites in multiple species of experimental animals. In animal studies, methyleugenol given orally to rats induced liver and stomach tumors in both sexes and kidney, mammary gland, and skin tumors in males. Methyleugenol given orally to mice induced benign and malignant tumors of the liver. Tumors of the stomach in male mice also were considered related to exposure to methyleugenol (NTP [2000]). Earlier studies found that methyleugenol and two similar compounds, the structurally related allylbenzenes, safrole and estragole, induced liver tumors in mice after intraperitoneal injection (IARC 1976, Miller *et al.* 1983). Safrole is listed in the Report on Carcinogens as *reasonably anticipated to be a human carcinogen* and by IARC as *possibly carcinogenic to humans* (Group 2B).

No adequate human studies of the relationship between exposure to

methyleugenol and human cancer were found.

Additional Information Relevant to Carcinogenicity

Mechanistic data indicate that liver tumors induced by methyleugenol and structurally related allylbenzenes result from metabolism of these compounds to DNA-reactive intermediates. Methyleugenol may be bioactivated by three different pathways: (1) Hydroxylation at the 1′ position of the allylic side chain to yield 1′-hydroxymethyleugenol, followed by sulfation of this intermediate to form 1′-hydroxymethyleugenol sulfate, (2) oxidation of the 2′,3′-double bond of the allylic side chain to form methyleugenol-2,3-oxide, and (3) *O*-demethylation followed by spontaneous rearrangement to form eugenol quinone methide. Formation of protein adducts and DNA adducts in the livers of animals (and in cultured human hepatocytes) exposed to allylbenzenes and induction of liver tumors by these compounds in animals have been attributed to activation via the hydroxylation pathway, because similar effects were produced by the 1′-hydroxy metabolites and because these effects were inhibited by pretreatment with sulfotransferase inhibitors (Miller *et al.* 1983, Boberg *et al.* 1983, Randerath *et al.* 1984, Gardner *et al.* 1996, NTP [2000]).

Methyleugenol, safrole, and estragole induce unscheduled DNA synthesis in rat hepatocytes, and their corresponding 1′-hydroxy metabolites are more potent genotoxic agents than are the parent compounds (Howes *et al.* 1990, Chan and Caldwell 1992). Methyleugenol induces morphological transformations in Syrian hamster embryo cells (Kerckaert *et al.* 1996), sister chromatid exchange in Chinese hamster ovary (CHO) cells (NTP [2000]), intrachromosomal recombination in yeast (Schiestl *et al.* 1989), and DNA repair in *Bacillus subtilis* (Sekizawa and Shibamoto 1982). Methyleugenol does not induce mutations in *Salmonella typhimurium* (NTP [2000]) or *Escherichia coli* (Sekizawa and Shibamoto 1982), chromosomal aberrations in CHO cells (NTP [2000]), or micronucleated erythrocytes in peripheral blood of mice (NTP [2000]). A higher frequency of β-*catenin* mutations was observed in liver tumors from mice treated with methyleugenol than in spontaneous liver tumors from control mice (Devereux *et al.* 1999). Methyleugenol's lack of mutagenicity in bacteria may be due to the need for sulfation in the metabolic activation of methyleugenol to its ultimate mutagenic or carcinogenic form.

EPA has reviewed the NTP cancer assessment for methyleugenol and agrees that methyleugenol can reasonably be anticipated to cause cancer in humans. EPA believes that the evidence is sufficient for listing methyleugenol on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for this chemical.

7. *Nitroarenes (selected)* (Refs. NTP Profile. (Ref. 17)). The NTP has classified five nitroarenes as "reasonably anticipated to be a human carcinogen." The five nitroarenes are: 1,6-Dinitropyrene, 1,8-Dinitropyrene, 6-Nitrochrysene, 1-Nitropyrene, and 4-Nitropyrene. 1-Nitropyrene is already on the EPCRA section 313 list under the polycyclic aromatic compounds (PACs) category (November 30, 1994, 59 FR 61485). All of the members of the PACs category are listed based on concerns for their carcinogenicity and were listed as a category because they are structurally similar and induce a similar toxic effect (cancer) (November 30, 1994, 59 FR 61463). Since the four other nitroarenes are PACs and are being proposed for listing based on a concern for carcinogenicity they are being proposed for addition to the PACs category, and not for individual listing.

The PACs category is one of several categories of chemicals of special concern for which reporting is triggered at lowered thresholds. 40 CFR 372.28(a)(2). The special concern for the PACs category members is that they are persistent, bioaccumulative, and toxic (PBT) chemicals. More specifically, it is the persistence and bioaccumulative properties of these chemicals that led EPA to lower reporting thresholds (October 29, 1999, 64 FR 58666). The persistence and bioaccumulation data for the four nitroarenes addressed in this proposal follows the individual summaries of the cancer data for each chemical. In addition to the data for the nitroarenes, there is a discussion of the PBT criteria and how it was applied to the PACs category.

a. *1,6-Dinitropyrene* (CAS No. 42397–64–8) (Refs. NTP Profile/Background document (Refs. 17 and 18)). The NTP has classified 1,6-dinitropyrene as "reasonably anticipated to be a human carcinogen." The classification is based on sufficient evidence of carcinogenicity in experimental animals. The NTP substance profile for 1,6-dinitropyrene (Ref. 17) included the following summary information of the evidence of carcinogenicity:

"Carcinogenicity

1,6-Dinitropyrene is *reasonably anticipated to be a human carcinogen* based on sufficient evidence of malignant tumor formation in multiple species of experimental animals, at multiple sites and by multiple routes of exposure (IARC 1989).

When administered by subcutaneous injections, 1,6-dinitropyrene induced injection-site sarcomas in male mice and male and female rats, and leukemia in female rats (Tokiwa *et al.* 1984, Ohgaki *et al.* 1985, Imaida *et al.* 1995). Intraperitoneal injections of 1,6-dinitropyrene caused an increased incidence of liver-cell tumors in male mice (Wislocki *et al.* 1986) and induced sarcomas of the peritoneal cavity in female rats (Imaida *et al.* 1991). In two studies, squamous cell carcinomas of the lung were induced in male rats receiving 1,6-dinitropyrene by intrapulmonary injection (Maeda *et al.* 1986, Iwagawa *et al.* 1989). The incidences of myeloid leukemia and lung adenocarcinomas were significantly increased in male and female hamsters receiving 1,6-dinitropyrene by intratracheal instillation (Takayama *et al.* 1985). 1,6-Dinitropyrene induced carcinoma of the pituitary gland in an oral study of short-term duration in rats (Imaida *et al.* 1991).

No adequate data were available to evaluate the carcinogenicity of 1,6-dinitropyrene in humans.

Additional Information Relevant to Carcinogenicity

Intratracheal administration of 1,6-dinitropyrene to rats previously inoculated to de-epithelialized trachea with an immortalized bronchial cell line, caused tumors when the tracheas were then implanted subcutaneously into nude mice (Iizasa *et al.* 1993). 1,6-Dinitropyrene is genotoxic in a wide variety of assays in bacteria and mammalian cells including human cells. 1,6-Dinitropyrene also demonstrates evidence of cell transformation activity *in vitro* in rat tracheal epithelial cells. Metabolic pathways leading to mutagenic and clastogenic metabolites and DNA adducts of 1,6-dinitropyrene have been described (IARC 1989).

No data were available that would suggest that the mechanisms thought to account for tumor induction by 1,6-dinitropyrene in experimental animals would not also operate in humans."

EPA has reviewed the NTP cancer assessment for 1,6-dinitropyrene and agrees that 1,6-dinitropyrene can reasonably be anticipated to cause

cancer in humans. EPA believes that the evidence is sufficient for listing 1,6-dinitropyrene in the PACs category on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for this chemical.

b. *1,8-Dinitropyrene* (CAS No. 42397–65–9) (Refs. NTP Profile/Background document (Refs. 17 and 18)). The National Toxicology Program has classified 1,8-dinitropyrene as "reasonably anticipated to be a human carcinogen." The classification is based on sufficient evidence of carcinogenicity in experimental animals. The NTP substance profile for 1,8-dinitropyrene (Ref. 17) included the following summary information of the evidence of carcinogenicity:

"Carcinogenicity

1,8-Dinitropyrene is *reasonably anticipated to be a human carcinogen* based on sufficient evidence of malignant tumor formation in multiple species of experimental animals, at multiple sites, and by multiple routes of exposure (IARC 1989). When administered by subcutaneous injections, 1,8-dinitropyrene induced injection-site sarcomas in male mice and male and female rats, and leukemia in female rats (Imaida *et al.* 1995, Ohgaki *et al.* 1984, 1985, Otofuji *et al.* 1987). Intraperitoneal injections of 1,8-dinitropyrene induced sarcomas of the peritoneal cavity, leukemia, and mammary adenocarcinoma in female rats (Imaida *et al.* 1991, 1995). The incidences of mammary tumors, including adenocarcinomas, were increased in female rats receiving 1,8-dinitropyrene by gavage (Imaida *et al.* 1991, IARC 1989).

No adequate data were available to evaluate the carcinogenicity of 1,8-dinitropyrene in humans.

Additional Information Relevant to Carcinogenicity

1,8-Dinitropyrene is genotoxic in a wide variety of assays in bacteria and mammalian cells demonstrating evidence of cell transformation activity *in vitro,* and metabolic pathways leading to mutagenic and clastogenic metabolites and DNA adducts have been described (IARC 1989).

No data were available that would suggest that the mechanisms thought to account for tumor induction of 1,8-dinitropyrene in experimental animals would not also operate in humans."

EPA has reviewed the NTP cancer assessment for 1,8-dinitropyrene and agrees that 1,8-dinitropyrene can reasonably be anticipated to cause cancer in humans. EPA believes that the evidence is sufficient for listing 1,8-dinitropyrene in the PACs category on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for this chemical.

c. *6-Nitrochrysene* (CAS No. 7496–02–8) (Refs. NTP Profile/Background document (Refs. 17 and 19)). The National Toxicology Program has classified 6-nitrochrysene as "reasonably anticipated to be a human carcinogen." The classification is based on sufficient evidence of carcinogenicity in experimental animals. The NTP substance profile for 6-nitrochrysene (Ref. 17) included the following summary information of the evidence of carcinogenicity:

"Carcinogenicity

6-Nitrochrysene is *reasonably anticipated to be a human carcinogen* based on sufficient evidence of carcinogenicity at multiple sites in multiple species of experimental animals (IARC 1989). In seven studies, when administered by intraperitoneal injection, 6-nitrochrysene caused lung tumors in male and female mice and also induced liver tumors in female and/or male mice in three of these studies and malignant lymphoma in one study (Busby *et al.* 1985, 1989, El-Bayoumy *et al.* 1992, Li *et al.* 1994, Fu *et al.* 1994, Imaida *et al.* 1992, Wislocki *et al.* 1986). Dysplastic and/or adenomatous lesions of the colon were increased in male and female rats, and colon adenocarcinomas were increased in male rats receiving 6-nitrochrysene by intraperitoneal injection (Imaida *et al.* 1992). Mammary fibroadenoma, adenocarcinoma, and spindle cell sarcomas were increased in female rats receiving 6-nitrochrysene by injection into the mammary gland (El-Bayoumy *et al.* 1993).

No data were available to evaluate the carcinogenicity of 6-nitrochrysene in humans.

Additional Information Relevant to Carcinogenicity

6-Nitrochrysene induced skin tumors, mainly papillomas, in a dermal initiation-promotion study in which 6-nitrochrysene was used as the initiator, followed by promotion with a phorbol ester (El-Bayoumy *et al.* 1982). It also caused lung and forestomach tumors when given by intraperitoneal injection to transgenic mice carrying a human hybrid c- Ha-*ras* gene (Ogawa *et al.* 1996). 6-Nitrochrysene is genotoxic in several assays in bacteria and mammalian cells and induces cell transformation in finite lifespan cells *in vitro.* Metabolic pathways leading to mutagenic and clastogenic metabolites and DNA adducts have been described (IARC 1989). The presence of 6-nitrochrysene- DNA adducts in tumor target tissue supports the possibility that tumors induced by this chemical are at least in part a result of chemical-induced DNA damage. No data were available that would suggest that the mechanisms thought to account for tumor induction by 6-nitrochrysene in experimental animals would not also operate in humans."

EPA has reviewed the NTP cancer assessment for 6-nitrochrysene and agrees that 6-nitrochrysene can reasonably be anticipated to cause cancer in humans. EPA believes that the evidence is sufficient for listing 6-nitrochrysene in the PACs category on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for this chemical.

d. *4-Nitropyrene* (CAS No. 57835–92–4) (Refs. NTP Profile/Background document (Refs. 17 and 20)). The National Toxicology Program has classified 4-nitropyrene as "reasonably anticipated to be a human carcinogen." The classification is based on sufficient evidence of carcinogenicity in experimental animals. The NTP substance profile for 4-nitropyrene (Ref. 17) included the following summary information of the evidence of carcinogenicity:

"Carcinogenicity

4-Nitropyrene is *reasonably anticipated to be a human carcinogen* based on sufficient evidence of malignant tumor formation at multiple tissue sites in multiple species of experimental animals (IARC 1989). Intraperitoneal injections of 4-nitropyrene caused an increased incidence of liver tumors in male mice, lung tumors in male and female mice (Wislocki *et al.* 1986), and mammary adenocarcinomas in female rats (Imaida *et al.* 1991). When administered by subcutaneous injections, 4-nitropyrene induced sarcomas at the injection site, and increased incidences of mammary adenocarcinomas, leukemia, and tumors of the Zymbal gland in female rats (Imaida *et al.* 1995, IARC 1989). In two studies, female rats receiving mammary gland injections of 4-nitropyrene showed an increased incidence of mammary tumors (Imaida *et al.* 1991, El-Bayoumy *et al.* 1993).

No data were available to evaluate the carcinogenicity of 4-nitropyrene in humans.

Additional Information Relevant to Carcinogenicity

Although not as reactive or potent as some of the mononitro- or dinitropyrenes, 4-nitropyrene is genotoxic in bacterial cells and induces cell transformation in BALB cells *in vitro.* Metabolic pathways for 4-nitropyrene, leading to mutagenic and likely DNA adducts, have also been described (IARC 1989).

No data were available that would suggest that the mechanisms thought to account for tumor induction by 4-nitropyrene in experimental animals would not also operate in humans."

EPA has reviewed the NTP cancer assessment for 4-nitropyrene and agrees that 4-nitropyrene can reasonably be anticipated to cause cancer in humans. EPA believes that the evidence is sufficient for listing 4-nitropyrene in the PACs category on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for this chemical.

e. *Nitroarene persistence and bioaccumulation data.* The above four nitroarenes are being proposed for addition to the PACs category, the members of which have been classified as PBT chemicals with lower reporting thresholds (October 29, 1999, 64 FR 58666). For purposes of EPCRA section 313 reporting, EPA established persistence half-life criteria for PBT chemicals of 2 months in water/sediment and soil and 2-days in air, and established bioaccumulation criteria for PBT chemicals as a bioconcentration factor (BCF) or bioaccumulation factor (BAF) of 1,000 or higher. Chemicals meeting the PBT criteria were assigned 100 pound reporting thresholds. With regards to setting the EPCRA section 313 reporting thresholds, EPA set lower reporting thresholds (10 pounds) for those PBT chemicals with persistence half-lifes of 6 months or more in water/sediment or soil and with BCF or BAF values of 5,000 or higher, these chemicals were considered highly PBT chemicals. At the time of the lowering of the thresholds for the PACs category, the persistence and bioaccumulation data for the current members in the category showed variation in these characteristics (October 29, 1999, 64 FR 58713). The PACs persistence data included air half-lifes of 2 hours to 4 days, surface water half-lifes of 79 days to 44 years, and soil half-lifes of 20 days to 14.6 years. The PACs bioaccumulation data ranged from BCFs of 800 to 31,440. EPA determined that while there was variation in the persistence and bioaccumulation data for the members of the PACs category, the best way to report these chemicals was as one single category (October 29, 1999, 64 FR 58725). While much of the persistence and bioaccumulation data for the PACs chemicals exceeded what EPA classified as highly persistent and bioaccumulative for setting reporting thresholds, EPA decided not to assign the PACs category the lower 10 pound reporting threshold because of the variability of the persistence and bioaccumulation data across members of the category (October 29, 1999, 64 FR 58726).

Since little data is available on the persistence of the four nitroarenes being proposed for listing, the data for 1-nitropyrene, a member of the PACs category, was used to estimate the persistence properties of the four nitroarenes (Ref. 21). 1-nitropyrene is a structural isomer of 4-nitropyrene and very close chemical analog of the other nitroarenes. The persistence data for 1-nitropyrene cited in the PBT chemical rule included air half lives of 10 hours to 4 days and surface water half lives of 16 to 44 years (October 29, 1999, 64 FR 58713). Based on EPA's assessment (Ref. 21), the four nitroarenes are expected to have similar persistence properties due to structural similarities and comparability of the available data.

Most of the bioaccumulation data for the members of the PACs category were calculated using a regression-derived equation (Ref. 22). The regression equation used to estimate the BCF values for the PACs category members for PBT chemical rule was: log BCF = 0.77 log Kow − 0.70 + correction factor. The estimated BCF value for 1-nitropyrene cited in the PBT rule was 908 (Ref. 22). The most recent equations for BCF calculations use the equation: log BCF = 0.6598 log Kow − 0.333 + correction factor (Ref. 21). The results using results both equations to calculate BCF values for the four nitroarenes are as follows: The calculated BCF values for 1,6- and 1,8-dinitropyrene ranged from 480–660, for 6-nitrochrysene they ranged from 1600 to 2600, and for 4-nitropyrene they ranged from 630–910 (Ref. 21).

EPA believes that the persistence and bioaccumulation data for the four nitroarenes is sufficiently similar to that for the current members of the PACs category that they should be included in the PACs category with the current 100 pound category reporting threshold.

8. *o-Nitroanisole* (CAS No. 91–23–6) (Refs. NTP Profile/Background document (Refs. 23 and 24)). The National Toxicology Program has classified o-nitroanisole as "reasonably anticipated to be a human carcinogen." The classification is based on sufficient evidence of carcinogenicity in experimental animals. The NTP substance profile for o-nitroanisole (Ref. 23) included the following summary information of the evidence of carcinogenicity:

"Carcinogenicity

o-Nitroanisole is *reasonably anticipated to be a human carcinogen* based on sufficient evidence of malignant tumor formation at multiple tissue sites in multiple species of experimental animals (NTP 1993).

When administered in the diet to male and female rats, o-nitroanisole induced increased incidences of mononuclear cell leukemia and neoplasms of the urinary bladder, kidney, and large intestine. When administered in the diet to mice, *o*-nitroanisole induced increased incidences of benign and malignant hepatocellular neoplasms in males and increased incidences of hepatocellular adenomas in females.

No adequate human studies of the relationship between exposure to o-nitroanisole and human cancer have been reported (IARC 1996).

Additional Information Relevant to Carcinogenicity

o-Nitroanisole is genotoxic in a wide variety of bacteria and mammalian cellular assays, and mutagenic and carcinogenic metabolites have been described (NTP 1993, IARC 1996).

No data were available that would suggest that the mechanisms thought to account for tumor induction by *o*-nitroanisole in experimental animals would not also operate in humans."

EPA has reviewed the NTP cancer assessment for o-nitroanisole and agrees that o-nitroanisole can reasonably be anticipated to cause cancer in humans. EPA believes that the evidence is sufficient for listing o-nitroanisole on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for this chemical.

9. *Nitromethane* (CAS No. 75–52–5) (Refs. NTP Profile/Background document (Refs. 25 and 26)). The National Toxicology Program has classified nitromethane as "reasonably anticipated to be a human carcinogen." The classification is based on sufficient evidence of carcinogenicity in experimental animals. The NTP substance profile for nitromethane (Ref. 25) included the following summary information of the evidence of carcinogenicity:

"Carcinogenicity

Nitromethane is *reasonably anticipated to be a human carcinogen* based on sufficient evidence of carcinogenicity in experimental animals. When administered by inhalation, nitromethane significantly increased the combined incidences of benign and malignant tumors at three tissue sites in mice and at a different tissue site in rats. In mice, nitromethane caused harderian gland and lung tumors in both sexes and liver tumors in females. In rats, nitromethane caused mammary gland tumors in female F344/N rats but did not cause any increased tumors in Long-Evans rats (exposed to lower levels) (NTP 1997). The International Agency for Research on Cancer (2000) also has concluded that there was sufficient evidence for the carcinogenicity of nitromethane in experimental animals.

No studies evaluating the carcinogenicity of nitromethane in humans were found in the published literature.

Additional Information Relevant to Carcinogenicity

The mechanism by which nitromethane causes cancer is not known. Nitromethane did not cause mutations in bacteria and does not appear to cause genetic damage in mammalian test systems. In cultured mammalian cells, nitromethane did not cause chromosomal aberrations (changes in chromosome structure or number), sister chromatid exchange, or micronucleus formation (a sign of chromosome damage or loss). Inhalation exposure of mice to nitromethane did not cause micronucleus formation in the erythrocytes (red blood cells), in either bone marrow or peripheral (circulating) blood (IARC 2000). In cultured Syrian hamster embryo cells, nitromethane induced cell transformation (a step in tumor formation) (Kerckaert *et al.* 1996, NTP 2002).

Nitromethane appears to be absorbed by inhalation; the available data suggest that dermal absorption is negligible. Metabolism of nitromethane by experimental animals *in vivo* has not been characterized. Metabolism of nitromethane by liver microsomes from Fischer 344 rats resulted in formation of only trace amounts of formaldehyde (IARC 2000)."

EPA has reviewed the NTP cancer assessment for nitromethane and agrees that nitromethane can reasonably be anticipated to cause cancer in humans. EPA believes that the evidence is sufficient for listing nitromethane on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for this chemical.

10. *Phenolphthalein* (CAS No. 77–09–8) (Refs. NTP Profile/Background document (Refs. 27 and 28)). The National Toxicology Program has classified phenolphthalein as "reasonably anticipated to be a human carcinogen." The classification is based on sufficient evidence of carcinogenicity in experimental animals. The NTP substance profile for phenolphthalein (Ref. 27) included the following summary information of the evidence of carcinogenicity:

"Carcinogenicity

Phenolphthalein is *reasonably anticipated to be a human carcinogen* based on sufficient evidence of increased incidence of malignant and/or combination of malignant and benign tumors in multiple tissue sites and in multiple species (IARC 2000). In a two-year $B6C3F_1$ mouse carcinogenicity study, NTP (1996) concluded that phenolphthalein, administered in feed, induced significant increases in the incidence of histiocytic sarcoma and lymphomas of thymic origin in males and females and malignant lymphoma (all types) and benign ovarian sex cord stromal tumors in females. In the corresponding Fischer 344 rat dietary carcinogenicity study, phenolphthalein induced significant increases in the incidence of benign pheochromocytoma of the adrenal medulla in males and females and renal tubule adenoma in males (NTP 1996). In a 6-month dietary study with female heterozygous *p*53-deficient transgenic mice, phenolphthalein induced a significant increase in the incidence of malignant lymphoma of thymic origin (Dunnick *et al.* 1997).

A few epidemiological studies have investigated the association between the use of phenolphthalein-containing laxatives and colon cancer or adenomatous colorectal polyps. No consistent association was found. Cancers at other sites have not been investigated in humans (IARC 2000).

Additional Information Relevant to Carcinogenicity

The malignant thymic lymphomas induced by phenolphthalein in female heterozygous *p*53-deficient transgenic mice exhibited a loss of the normal *p*53 allele, suggesting the involvement of a mutagenic mechanism in tumor induction and/or progression (Dunnick *et al.* 1997).

Phenolphthalein causes enhanced oxygen radical production in *in vitro* systems. *In vivo,* reduction of phenoxyl radicals could allow reformation of phenolphthalein, establishing a futile cycle of oxidation and reduction, thereby generating more free radical species. Thus, phenolphthalein may be a significant source of oxidative stress in physiological systems.

Although negative for mutagenicity and DNA damage in bacteria, phenolphthalein exhibits genetic activity in several *in vitro* and *in vivo* mammalian assays. Phenolphthalein was positive for the induction of chromosomal aberrations in cultured Chinese hamster ovary cells in the presence of metabolic activation and induced *hprt* gene mutations, chromosomal aberrations, and morphological transformation in Syrian hamster embryo cells. Phenolphthalein was also positive for the induction of micronucleated erythrocytes in mice following multiple, but not single, treatments administered by gavage or dosed feed. Phenolphthalein also induced micronuclei in female heterozygous *p*53-deficient transgenic mice exposed via dosed feed for 26 weeks. Abnormal sperm were induced in male mice, but not male rats, treated with phenolphthalein via dosed feed for 13 weeks. Phenolphthalein was negative for Na/K ATPase gene mutations and aneuploidy in Syrian hamster embryo cells.

No data were available that would suggest that the mechanisms thought to account for tumor induction by phenolphthalein in experimental animals would not also operate in humans. Phenolphthalein causes oxidative stress and also demonstrates the capability to alter tumor suppressor gene pathways, which are both mechanisms believed to be involved in human cancer."

EPA has reviewed the NTP cancer assessment for phenolphthalein and agrees that phenolphthalein can reasonably be anticipated to cause cancer in humans. EPA believes that the evidence is sufficient for listing phenolphthalein on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for this chemical.

11. *Tetrafluoroethylene* (CAS No. 116–14–3) (Refs. NTP Profile/Background document (Refs. 29 and 30)). The National Toxicology Program has classified tetrafluoroethylene as "reasonably anticipated to be a human carcinogen." The classification is based on sufficient evidence of carcinogenicity in experimental animals. The NTP substance profile for tetrafluoroethylene (Ref. 29) included the following summary information of the evidence of carcinogenicity:

"Carcinogenicity

Tetrafluoroethylene (TFE) is *reasonably anticipated to be a human carcinogen* based on sufficient evidence of malignant tumor formation at multiple sites in multiple species of experimental animals (NTP 1997). When administered by inhalation to F344 rats, TFE induced renal tubule neoplasms, hepatocellular neoplasms, liver hemangiosarcoma, and mononuclear cell leukemia. When administered by inhalation to $B6C3F_1$ mice, TFE induced liver hemangiomas and hemangiosarcomas, hepatocellular neoplasms, and histiocytic sarcomas.

No adequate human studies of the relationship between exposure to TFE and human cancer have been reported (IARC 1999).

Additional Information Relevant to Carcinogenicity

In prokaryotic systems, TFE was negative for the induction of gene mutations in *Salmonella typhimurium* with and without S9 activation. In mammalian systems *in vitro,* TFE was also negative for the induction of gene mutations in Chinese hamster ovary cells (HSDB 2001). No increases in the frequency of micronucleated erythrocytes were observed in peripheral blood samples obtained from TFE-exposed mice (NTP 1997).

The frequency of H-ras codon 61 mutations observed in TFE-induced hepatocellular neoplasms (15%) was significantly less than the corresponding frequency (56 to 59%) in spontaneous liver neoplasms of $B6C3F_1$ mice, suggesting that TFE induces liver neoplasms via a *ras*-independent pathway (NTP 1997).

The kidney-specific toxicity and carcinogenicity of TFE is most likely related to the selective uptake and subsequent processing of TFE-glutathione conjugates by renal β-lyase (Miller and Surh 1994, Anders *et al.* 1988). In rats, a TFE cysteine conjugate is bioactivated in the kidney to a difluorothionacetyl fluoride, the putative reactive metabolite for TFE-induced nephrotoxicity (NTP 1997).

No data were available that would suggest that the mechanisms thought to account for tumor induction by TFE in experimental animals would not also operate in humans."

EPA has reviewed the NTP cancer assessment for tetrafluoroethylene and agrees that tetrafluoroethylene can reasonably be anticipated to cause cancer in humans. EPA believes that the evidence is sufficient for listing tetrafluoroethylene on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for this chemical.

12. *Tetranitromethane* (CAS No. 509–14–8) (Refs. NTP Profile/NTP study (Refs. 31 and 32)). The National Toxicology Program has classified tetranitromethane as "reasonably anticipated to be a human carcinogen." The classification is based on sufficient evidence of carcinogenicity in experimental animals. The NTP substance profile for tetranitromethane (Ref. 31) included the following summary information of the evidence of carcinogenicity:

"Carcinogenicity

Tetranitromethane is *reasonably anticipated to be a human carcinogen* based on sufficient evidence of carcinogenicity in experimental animals. Exposure to tetranitromethane in a two-year inhalation bioassay caused a dose-related increase in alveolar/bronchiolar neoplasms to nearly all mice and rats exposed to concentrations of 2 and 5 ppm respectively. The incidences of these neoplasms in lower exposure concentration groups (2 ppm for rats and 0.5 ppm for mice) were 66% and 44% in male and female rats, respectively, and 54% and 48% in male and female mice, respectively (NTP 1990). The majority of animals with alveolar/bronchiolar neoplasms had neoplasms diagnosed as carcinomas, and these neoplasms frequently metastasized to a variety of organs. Squamous cell carcinomas of the lung were also markedly increased in rats exposed to 5 ppm. This particular type of neoplasm has been found in only 3 of approximately 1,600 untreated control male rats and in none of a similar number of untreated female controls (NTP 1990).

No adequate human studies of the relationship between exposure to tetranitromethane and human cancer have been reported (IARC 1996)."

EPA has reviewed the NTP cancer assessment for tetranitromethane and agrees that tetranitromethane can reasonably be anticipated to cause cancer in humans. EPA believes that the evidence is sufficient for listing tetranitromethane on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for this chemical.

13. *Vinyl Fluoride* (CAS No. 75–02–5) (Refs. NTP Profile/Background document (Refs. 33 and 34)). The National Toxicology Program has classified vinyl fluoride as "reasonably anticipated to be a human carcinogen." The classification is based on sufficient evidence of carcinogenicity in experimental animals. The NTP substance profile for vinyl fluoride (Ref. 33) included the following summary information of the evidence of carcinogenicity:

"Carcinogenicity

Vinyl fluoride is *reasonably anticipated to be a human carcinogen* based on sufficient evidence of carcinogenicity in experimental animals. Both male and female rats exposed to vinyl fluoride by inhalation showed increased incidences of hepatic hemangiosarcoma, hepatocellular adenoma or carcinoma, and Zymbal gland carcinoma. Both male and female mice exposed to vinyl fluoride by inhalation showed increased incidences of hepatic hemangiosarcoma, bronchiolar-alveolar adenoma or adenocarcinoma, hepatocellular adenoma, and harderian gland adenoma. Female mice also showed an increased incidence of mammary gland adenocarcinoma (Bogdanffy *et al.* 1995, IARC 1995).

The tumor responses of laboratory animals to vinyl fluoride are similar to their responses to vinyl chloride, a known human carcinogen (IARC 1987), and to vinyl bromide, a probable human carcinogen (IARC 1986). A unique feature of vinyl chloride carcinogenicity is that vinyl chloride induces rare hepatic hemangiosarcomas in experimental animals and is causally associated with excess risk of liver hemangiosarcoma in epidemiological studies of exposed workers. The fact that vinyl fluoride, vinyl chloride, and vinyl bromide all induce rare hemangiosarcomas of the liver in experimental animals and induce the formation of similar DNA adducts suggests a possible common mechanism of carcinogenicity for all three of these chemicals.

No adequate human studies of the relationship between exposure to vinyl fluoride and human cancer were found.

Additional Information Relevant to Carcinogenicity

Vinyl fluoride is mutagenic in *Salmonella typhimurium* with the addition of a rat liver homogenate metabolic activation system. In addition, vinyl fluoride induces gene mutations and chromosomal aberrations in Chinese hamster ovary cells (with metabolic activation), sex-linked recessive lethal mutations in *Drosophila melanogaster,* and micronuclei in bone marrow cells of female mice (IARC 1995).

Vinyl fluoride likely is metabolized in a manner similar to vinyl chloride: Oxidation via cytochrome P450 to fluoroethylene oxide, followed by

rearrangement to 2-fluoroacetaldehyde, which is oxidized to fluoroacetic acid. Human, rat, and mouse liver microsomes metabolize vinyl fluoride at similar rates (Cantoreggi and Keller 1997). Vinyl fluoride metabolites form covalent DNA adducts. Inhalation exposure of rats and mice to vinyl fluoride produced a dose-related increase in the formation of the promutagenic adduct N^2,3-ethenoguanine in their liver DNA (Swenberg *et al.* 1995).

No available data suggest that mechanisms by which vinyl fluoride induces tumors in experimental animals would not also operate in humans."

EPA has reviewed the NTP cancer assessment for vinyl fluoride and agrees that vinyl fluoride can reasonably be anticipated to cause cancer in humans. EPA believes that the evidence is sufficient for listing vinyl fluoride on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for this chemical.

V. Rationale for Listing

The NTP RoC document undergoes significant scientific review and public comment. The NTP review mirrors the review EPA has historically done to assess chemicals for listing under EPCRA section 313 on the basis of carcinogenicity. The conclusions regarding the potential for chemicals in the NTP RoC to cause cancer in humans are based on established sound scientific principles. EPA believes that the NTP RoC is an excellent and reliable source of information on the potential for chemicals covered in the NTP RoC to cause cancer in humans. Based on EPA's review of the data contained in the 11th NTP RoC, EPA has determined that the chemicals in this proposed rule can reasonably be anticipated to cause cancer. Therefore, EPA believes that the evidence is sufficient for listing all of the chemicals in this proposed rule on the EPCRA section 313 toxic chemical list pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data for these chemicals as presented in the 11th RoC.

EPA considers chemicals that can reasonably be anticipated to cause cancer to have moderately high to high chronic toxicity. EPA does not believe that it is appropriate to consider exposure for chemicals that are moderately high to highly toxic based on a hazard assessment when determining if a chemical can be added for chronic effects pursuant to EPCRA section 313(d)(2)(B) (*see* 59 FR 61440–61442). Therefore, in accordance with EPA's standard policy on the use of exposure assessments (59 FR 61432), EPA does not believe that an exposure assessment is necessary or appropriate for determining whether any of the chemicals in this proposed rule meet the criteria of EPCRA section 313(d)(2)(B).

VI. References

EPA has established an official public docket for this action under Docket ID No. EPA–HQ–TRI–2010–0006. The public docket includes information considered by EPA in developing this action, including the documents listed below, which are electronically or physically located in the docket. In addition, interested parties should consult documents that are referenced in the documents that EPA has placed in the docket, regardless of whether these referenced documents are electronically or physically located in the docket. For assistance in locating documents that are referenced in documents that EPA has placed in the docket, but that are not electronically or physically located in the docket, please consult the person listed in the above **FOR FURTHER INFORMATION CONTACT** section.

1. NTP, 2005. National Toxicology Program. Introduction: Report on Carcinogens, Eleventh Edition. Released January 31, 2005. U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program, Research Triangle Park, NC 27709.

2. USEPA. Guidelines for Carcinogen Risk Assessment. Risk Assessment Forum, U.S. Environmental Protection Agency, Washington, DC, March 2005.

3. USEPA, OEI. Economic Analysis of the Proposed Rule to add 16 Chemicals to the EPCRA Section 313 List of Toxic Chemicals. February 16, 2010.

4. USEPA, OEI. Memorandum from Mark Miller, PhD, Toxicologist, Analytical Support Branch to Nicole Paquette, PhD, Chief, Analytical Support Branch. January 28, 2010. Subject: Review of National Toxicology Program (NTP) Cancer Classification Data for Sixteen Chemicals.

5. NTP, 2005. National Toxicology Program. 11th Report on Carcinogens—1-Amino-2,4-dibromoanthraquinone Substance Profile. Released January 31, 2005. U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program, Research Triangle Park, NC 27709.

6. NTP, 2002. Report on Carcinogens Background Document for 1-Amino-2,4-dibromoanthraquinone. September 19, 2002. Prepared for, U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program, Research Triangle Park, NC 27709. Prepared by, Technology Planning and Management Corporation Canterbury Hall, Suite 310, 4815 Emperor Blvd., Durham, NC 27703. Contract Number N01–ES–85421.

7. NTP, 2005. National Toxicology Program. 11th Report on Carcinogens—2,2-bis(Bromomethyl)-1,3-propanediol Substance Profile. Released January 31, 2005. U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program, Research Triangle Park, NC 27709.

8. NTP. Report on Carcinogens Background Document for 2,2-bis(Bromomethyl)-1,3-propanediol (Technical Grade). Prepared for, U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program, Research Triangle Park, NC 27709. Prepared by, Technology Planning and Management Corporation, Canterbury Hall, Suite 310, 4815 Emperor Blvd., Durham, NC 27703. Contract Number N01–ES–85421.

9. NTP, 2005. National Toxicology Program. 11th Report on Carcinogens—Furan Substance Profile. Released January 31, 2005. U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program, Research Triangle Park, NC 27709.

10. NTP, 1999. National Toxicology Program Report on Carcinogens Background Document for Furan. March 1999. Prepared for, November 18–19, 1996, Meeting of the Report on Carcinogens Subcommittee of the Board of Scientific Counselors. Prepared by, Integrated Laboratory Systems, Research Triangle Park, NC 27709. NIEHS Contract No. N01–ES–25346.

11. NTP, 2005. National Toxicology Program. 11th Report on Carcinogens—Glycidol Substance Profile. Released January 31, 2005. U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program, Research Triangle Park, NC 27709.

12. NTP, 1990. Toxicology and Carcinogenesis Studies of Glycidol (CAS No. 556–52–5) In F344/N Rats and B6C3F1 Mice (Gavage Studies). Technical Report Series No. 374. NIH Publication No. 90–2829, March 1990. National Toxicology Program, Research Triangle Park, NC. 229 pp.

13. NTP, 2005. National Toxicology Program. 11th Report on Carcinogens—Isoprene Substance Profile. Released January 31, 2005. U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program, Research Triangle Park, NC 27709.

14. NTP, 1999. National Toxicology Program Report on Carcinogens Background Document for Isoprene. March 1999. Prepared for, December 2–3, 1998, Meeting of the Report on Carcinogens Subcommittee of the NTP Board of Scientific Counselors. Prepared by, Integrated Laboratory Systems, Research Triangle Park, NC 27709. NIEHS Contract No. N01–ES–25346.

15. NTP, 2005. National Toxicology Program. 11th Report on Carcinogens—Methyleugenol Substance Profile. Released January 31, 2005. U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program, Research Triangle Park, NC 27709.

16. NTP, 2000. Report on Carcinogens Background Document for Methyleugenol. December 13–14, 2000, Meeting of the NTP Board of Scientific Counselors Report on Carcinogens Subcommittee. Prepared for, U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program, Research Triangle Park, NC 27709. Prepared by, Technology Planning and Management Corporation, Canterbury Hall, Suite 310, 4815 Emperor Blvd., Durham, NC 27703. Contract Number N01–ES–85421.

17. NTP, 2005. National Toxicology Program. 11th Report on Carcinogens—Nitroarenes (Selected) Substance Profile. Released January 31, 2005. U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program, Research Triangle Park, NC 27709.

18. NTP, 1999. National Toxicology Program Report on Carcinogens Background Document for 1,6-Dinitropyrene and 1,8-Dinitropyrene. Final March 1999. Prepared for, November 18–19, 1996, Meeting of the Report on Carcinogens Subcommittee of the NTP Board of Scientific Counselors. Prepared by, Integrated Laboratory Systems, Research Triangle Park, NC 27709. NIEHS Contract No. N01–ES–25346.

19. NTP, 1999. National Toxicology Program Report on Carcinogens Background Document for 6-Nitrochrysene. Final March 1999. Prepared for, November 18–19, 1996, Meeting of the Report on Carcinogens Subcommittee of the NTP Board of Scientific Counselors. Prepared by, Integrated Laboratory Systems, Research Triangle Park, NC 27709. NIEHS Contract No. N01–ES–25346.

20. NTP, 1999. National Toxicology Program Report on Carcinogens Background Document for 4-Nitropyrene. Final March 1999. Prepared for, November 18–19, 1996, Meeting of the Report on Carcinogens Subcommittee of the NTP Board of Scientific Counselors. Prepared by, Integrated Laboratory Systems, Research Triangle Park, NC 27709. NIEHS Contract No. N01–ES–25346.

21. USEPA/OEI. Technical Support Document: Bioaccumulation and Persistence Data for Selected Nitroarenes. Office of Environmental Information, Environmental Analysis Division, Analytical Support Branch, November 2009.

22. USEPA/OPPT. Technical Support Document for Determination of Bioaccumulation (BAF) and Bioconcentration (BCF) Values for Persistent Bioaccumulative Toxic (PBT) Chemicals and for Identification of PBT Chemicals. Jerry Smrchek, PhD, Biologist, Existing Chemicals Assessment Branch, Risk Assessment Division. September 1998.

23. NTP, 2005. National Toxicology Program. 11th Report on Carcinogens—o-Nitroanisole Substance Profile. Released January 31, 2005. U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program, Research Triangle Park, NC 27709.

24. NTP, 1999. National Toxicology Program Report on Carcinogens Background Document for o-Nitroanisole. Final March 1999. Prepared for, November 18–19, 1996, Meeting of the Report on Carcinogens Subcommittee of the NTP Board of Scientific Counselors. Prepared by, Integrated Laboratory Systems, Research Triangle Park, NC 27709. NIEHS Contract No. N01–ES–25346.

25. NTP, 2005. National Toxicology Program. 11th Report on Carcinogens—Nitromethane Substance Profile. Released January 31, 2005. U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program, Research Triangle Park, NC 27709.

26. NTP, 2002. Final Report on Carcinogens Background Document for Nitromethane. March 25, 2002. Prepared for, U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program, Research Triangle Park, NC 27709. Prepared by, Technology Planning and Management Corporation, Canterbury Hall, Suite 310, 4815 Emperor Blvd., Durham, NC 27703. Contract Number N01–ES–85421.

27. NTP, 2005. National Toxicology Program. 11th Report on Carcinogens—Phenolphthalein Substance Profile. Released January 31, 2005. U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program, Research Triangle Park, NC 27709.

28. NTP, 1999. National Toxicology Program Report on Carcinogens Background Document for Phenolphthalein. Final March 1999. Prepared for, October 30–31, 1997, Meeting of the Report on Carcinogens Subcommittee of the NTP Board of Scientific Counselors. Prepared by, Integrated Laboratory Systems, Research Triangle Park, NC 27709. NIEHS Contract No. N01–ES–25346.

29. NTP, 2005. National Toxicology Program. 11th Report on Carcinogens—Tetrafluoroethylene Substance Profile. Released January 31, 2005. U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program, Research Triangle Park, NC 27709.

30. NTP, 1999. National Toxicology Program Report on Carcinogens Background Document for Tetrafluoroethylene. Final March 1999. Prepared for, October 30–31, 1997, Meeting of the Report on Carcinogens Subcommittee of the NTP Board of Scientific Counselors. Prepared by, Integrated Laboratory Systems, Research Triangle Park, NC 27709. NIEHS Contract No. N01–ES–25346.

31. NTP, 2005. National Toxicology Program. 11th Report on Carcinogens—Tetranitromethane Substance Profile. Released January 31, 2005. U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program, Research Triangle Park, NC 27709.

32. NTP, 1990. Toxicology and Carcinogenesis Studies of Tetranitromethane (CAS No. 509–14–8) in F344/N Rats and B6C3F1 Mice (Inhalation Studies). Technical Report Series No. 386. NIH Publication No. 90–2841. Research Triangle Park, NC and Bethesda, NC: National Toxicology Program. 207 pp.

33. NTP, 2005. National Toxicology Program. 11th Report on Carcinogens—Vinyl Fluoride Substance Profile. Released January 31, 2005. U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program, Research Triangle Park, NC 27709.

34. NTP. Final Report on Carcinogens Background Document for Vinyl Fluoride. Meeting of the NTP Board of Scientific Counselors Report on Carcinogens Subcommittee. Prepared for, U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program, Research Triangle Park, NC 27709. Prepared by, Technology Planning and Management Corporation, Canterbury Hall, Suite 310,

4815 Emperor Blvd, Durham, NC 27703. Contract Number N01–ES–85421.

VIII. Statutory and Executive Order Reviews Associated With This Action?

A. Executive Order 12866, Regulatory Planning and Review

This action is not a "significant regulatory action" under the terms of Executive Order (EO) 12866 (58 FR 51735, October 4, 1993) and is therefore not subject to review under the EO.

B. Paperwork Reduction Act

This proposed rule does not contain any new information collection requirements that require additional approval by the Office of Management and Budget (OMB) under the Paperwork Reduction Act (PRA), 44 U.S.C. 3501 *et seq.* Currently, the facilities subject to the reporting requirements under EPCRA 313 and PPA 6607 may use either the EPA Toxic Chemicals Release Inventory Form R (EPA Form 1B9350–1), or the EPA Toxic Chemicals Release Inventory Form A (EPA Form 1B9350–2). The Form R must be completed if a facility manufactures, processes, or otherwise uses any listed chemical above threshold quantities and meets certain other criteria. For the Form A, EPA established an alternative threshold for facilities with low annual reportable amounts of a listed toxic chemical. A facility that meets the appropriate reporting thresholds, but estimates that the total annual reportable amount of the chemical does not exceed 500 pounds per year, can take advantage of an alternative manufacture, process, or otherwise use threshold of 1 million pounds per year of the chemical, provided that certain conditions are met, and submit the Form A instead of the Form R. In addition, respondents may designate the specific chemical identity of a substance as a trade secret pursuant to EPCRA section 322 42 U.S.C. 11042: 40 CFR part 350.

OMB has approved the reporting and recordkeeping requirements related to Form R, supplier notification, and petitions under OMB Control number 2070–0093 (EPA Information Collection Request (ICR) No. 1363.15); those related to Form A under OMB Control number 2070–0143 (EPA ICR No. 1704.09); and those related to trade secret designations under OMB Control number 2070–0078 (EPA ICR No. 1428). As provided in 5 CFR 1320.5(b) and 1320.6(a), an Agency may not conduct or sponsor, and a person is not required to respond to, a collection of information unless it displays a currently valid OMB control number. The OMB Control numbers relevant to EPA's regulations are listed in 40 CFR part 9, 48 CFR chapter 15, and displayed on the information collection instruments (*e.g.,* forms, instructions).

For Form R, EPA estimates the industry reporting and recordkeeping burden for collecting this information to average, in the first year, approximately $4,615 per Form R (for a total first year cost of $858,299 based on 16,069 total burden hours). In subsequent years, the burden for collecting this information is estimated to average $1,553 per Form R (for a total cost of $288,902 based on 5,517 total burden hours). These estimates include the time needed to become familiar with the requirement (first year only); review instructions; search existing data sources; gather and maintain the data needed; complete and review the collection information; and transmit or otherwise disclose the information. The actual burden on any facility may be different from this estimate depending on the complexity of the facility's operations and the profile of the releases at the facility. Upon promulgation of a final rule, the Agency may determine that the existing burden estimates in the ICRs need to be amended in order to account for an increase in burden associated with the final action. If so, the Agency will submit an information collection worksheet (ICW) to OMB requesting that the total burden in each ICR be amended, as appropriate.

The Agency would appreciate any comments or information that could be used to: (1) Evaluate whether the proposed collection of information is necessary for the proper performance of the functions of the Agency, including whether the information will have practical utility; (2) evaluate the reasonableness of the Agency's estimate of the burden of the proposed collection of information, including the validity of the methodology and assumptions used; (3) enhance the quality, utility, and clarity of the information to be collected; and (4) minimize the burden of the collection of information on those who are to respond, including through the use of appropriate automated electronic, mechanical, or other technological collection techniques or other forms of information technology, e.g., permitting electronic submission of responses. Please submit your comments within 90 days as specified at the beginning of this proposal. Copies of the existing ICRs may be obtained from Rick Westlund, Collection Strategies Division, U.S. Environmental Protection Agency (2822T), 1200 Pennsylvania Ave., NW., Washington, DC 20460 or by calling (202) 566–1672.

C. Regulatory Flexibility Act (RFA), as Amended by the Small Business Regulatory Enforcement Fairness Act of 1996 (SBREFA), 5 U.S.C. 601 et seq.

The RFA generally requires an agency to prepare a regulatory flexibility analysis of any rule subject to notice and comment rulemaking requirements under the Administrative Procedure Act or any other statute unless the agency certifies that the rule will not have a significant economic impact on a substantial number of small entities. Small entities include small businesses, small organizations, and small governmental jurisdictions. For purposes of assessing the impacts of today's rule on small entities, small entity is defined as: (1) A business that is classified as a "small business" by the Small Business Administration at 13 CFR 121.201; (2) a small governmental jurisdiction that is a government of a city, county, town, school district or special district with a population of less than 50,000; and (3) a small organization that is any not-for-profit enterprise which is independently owned and operated and is not dominant in its field.

Of the 109 entities estimated to be impacted by this proposed rule, 41 are small businesses. Of the affected small businesses, all 41 have cost impacts of less than 1% in both the first and subsequent years of the rulemaking. No small businesses are projected to have a cost impact of 1% or greater. In the first year, of the 41 estimated cost impacts, there is a maximum impact of 0.616% and a minimum impact of less than 0.001%. Facilities eligible to use Form A (those meeting the appropriate activity threshold which have 500 pounds per year or less of reportable amounts of the chemical) will have a lower burden. No small governments or small organizations are expected to be affected by this action. Thus this rule is not expected to have a significant adverse economic impact on a substantial number of small entities. A more detailed analysis of the impacts on small entities is located in EPA's economic analysis support document (Ref. 3).

After considering the economic impacts of today's rule on small entities, I certify that this action will not have a significant economic impact on a substantial number of small entities. We continue to be interested in the potential impacts of the proposed rule on small entities and welcome comments on issues related to such impacts.

D. Unfunded Mandates Reform Act

This rule does not contain a Federal mandate that may result in expenditures of $100 million or more for State, local, and tribal governments, in the aggregate, or the private sector in any one year. EPA's economic analysis indicates that the total cost of this rule is estimated to be $859,072 in the first year of reporting. Thus, this rule is not subject to the requirements of sections 202 or 205 of UMRA.

This rule is also not subject to the requirements of section 203 of UMRA because it contains no regulatory requirements that might significantly or uniquely affect small governments. Small governments are not subject to the EPCRA section 313 reporting requirements.

E. Executive Order 13132 (Federalism)

This action does not have federalism implications. It will not have substantial direct effects on the States, on the relationship between the national government and the States, or on the distribution of power and responsibilities among the various levels of government, as specified in Executive Order 13132. This action relates to toxic chemical reporting under EPCRA section 313, which primarily affects private sector facilities. Thus, Executive Order 13132 does not apply to this action.

In the spirit of Executive Order 13132, and consistent with EPA policy to promote communications between EPA and State and local governments, EPA specifically solicits comment on this proposed action from State and local officials.

F. Executive Order 13175: Consultation and Coordination With Indian Tribal Governments

This action does not have tribal implications, as specified in Executive Order 13175 (65 FR 67249, November 9, 2000). This action relates to toxic chemical reporting under EPCRA section 313, which primarily affects private sector facilities. Thus, Executive Order 13175 does not apply to this action. In the spirit of Executive Order 13175, and consistent with EPA policy to promote communications between EPA and Indian Tribal Governments, EPA specifically solicits additional comment on this proposed action from tribal officials.

G. Executive Order 13045: Protection of Children From Environmental Health Risks and Safety Risks

This action is not subject to EO 13045 (62 FR 19885, April 23, 1997) because it is not economically significant as defined in EO 12866, and because the Agency does not believe the environmental health or safety risks addressed by this action present a disproportionate risk to children. This action relates to toxic chemical reporting under EPCRA section 313, which primarily affects private sector facilities.

H. Executive Order 13211: Actions Concerning Regulations That Significantly Affect Energy Supply, Distribution, or Use

This action is not subject to Executive Order 13211 (66 FR 28355 (May 22, 2001)), because it is not a significant regulatory action under Executive Order 12866.

I. National Technology Transfer and Advancement Act

Section 12(d) of the National Technology Transfer and Advancement Act of 1995 ("NTTAA"), Public Law 104–113, 12(d) (15 U.S.C. 272 note) directs EPA to use voluntary consensus standards in its regulatory activities unless to do so would be inconsistent with applicable law or otherwise impractical. Voluntary consensus standards are technical standards (e.g., materials specifications, test methods, sampling procedures, and business practices) that are developed or adopted by voluntary consensus standards bodies. NTTAA directs EPA to provide Congress, through OMB, explanations when the Agency decides not to use available and applicable voluntary consensus standards.

This proposed rulemaking does not involve technical standards. Therefore, EPA is not considering the use of any voluntary consensus standards.

J. Executive Order 12898: Federal Actions To Address Environmental Justice in Minority Populations and Low-Income Populations

Executive Order (EO) 12898 (59 FR 7629 (Feb. 16, 1994)) establishes Federal executive policy on environmental justice. Its main provision directs Federal agencies, to the greatest extent practicable and permitted by law, to make environmental justice part of their mission by identifying and addressing, as appropriate, disproportionately high and adverse human health or environmental effects of their programs, policies, and activities on minority populations and low-income populations in the United States.

EPA has determined that this proposed rule will not have disproportionately high and adverse human health or environmental effects on minority or low-income populations because it does not affect the level of protection provided to human health or the environment. This proposed rule adds additional chemicals to the EPCRA section 313 reporting requirements. By adding chemicals to the list of toxic chemicals subject to reporting under section 313 of EPCRA, EPA would be providing communities across the United States (including minority populations and low income populations) with access to data which they may use to seek lower exposures and consequently reductions in chemical risks for themselves and their children. This information can also be used by government agencies and others to identify potential problems, set priorities, and take appropriate steps to reduce any potential risks to human health and the environment. Therefore, the informational benefits of the proposed rule will have a positive impact on the human health and environmental impacts of minority populations, low-income populations, and children.

List of Subjects in 40 CFR Part 372

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

Dated: March 31, 2010.

Lisa P. Jackson,

Administrator.

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

PART 372—[AMENDED]

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

Authority: 42 U.S.C. 11023 and 11048.

§ 372.28 [Amended]

2. In § 372.28, the table in paragraph (a)(2) under the heading "Polycyclic aromatic compounds (PACs): (This category includes only those chemicals listed below)" is amended by adding four new entries in alphabetical order to read as follows:

§ 372.28 Lower thresholds for chemicals of special concern.

(a) * * *

(2) * * *

Category name	Reporting threshold
* * * * *	
Polycyclic aromatic compounds (PACs): (This category includes only those chemicals listed below)	100
* * * * *	
42397–64–8 1,6-Dinitropyrene.	
42397–65–9 1,8-Dinitropyrene.	
* * * * *	
07496–02–8 6-Nitrochrysene.	
* * * * *	
57835–92–4 4-Nitropyrene.	

§ 372.65 [Amended]

3. Section 372.65 is amended as follows:

a. In the table to paragraph (a) by adding new entries in alphabetical order.

b. In the table to paragraph (b) by adding new entries in numerical order.

c. In the table to paragraph (c) under the heading "Polycyclic aromatic compounds (PACs): (This category includes only those chemicals listed below)" by adding four entries in alphabetical order.

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

* * * * *

(a) * * *

Chemical name	CAS No.	Effective date
* * * * * * *		
1-Amino-2,4-dibromoanthraquinone	00081–49–2	1/11
* * * * * * *		
2,2-bis(Bromomethyl)-1,3-propanediol	003296–90–0	1/11
* * * * * * *		
Furan	00110–00–9	1/11
* * * * * * *		
Glycidol	00556–52–5	1/11
* * * * * * *		
Isoprene	00078–79–5	1/11
* * * * * * *		
Methyleugenol	00093–15–2	1/11
* * * * * * *		
o-Nitroanisole	00091–23–6	1/11
* * * * * * *		
Nitromethane	00075–52–5	1/11
* * * * * * *		
Phenolphthalein	00077–09–8	1/11
* * * * * * *		
Tetrafluoroethylene	00116–14–3	1/11
* * * * * * *		
Tetranitromethane	00509–14–8	1/11
* * * * * * *		
Vinyl Fluoride	00075–02–5	1/11
* * * * * * *		

(b) * * *

CAS No.	Chemical name	Effective date
* *	* * * *	
00075–02–5	Vinyl Fluoride	1/11
* *	* * * *	
00075–52–5	Nitromethane	1/11
* *	* * * *	
00077–09–8	Phenolphthalein	1/11

CAS No.	Chemical name	Effective date
* * *	* * *	
00078–79–5	Isoprene	1/11
* * *	* * *	
00081–49–2	1-Amino-2,4-dibromoanthraquinone	1/11
* * *	* * *	
00091–23–6	o-Nitroanisole	1/11
* * *	* * *	
00093–15–2	Methyleugenol	1/11
* * *	* * *	
00110–00–9	Furan	1/11
* * *	* * *	
00116–14–3	Tetrafluoroethylene	1/11
* * *	* * *	
00509–14–8	Tetranitromethane	1/11
* * *	* * *	
00556–52–5	Glycidol	1/11
* * *	* * *	
03296–90–0	2,2-bis(Bromomethyl)-1,3-propanediol	1/11
* * *	* * *	

(c) * * *

Category name	Effective date
* * *	
Polycyclic aromatic compounds (PACs): (This category includes only those chemicals listed below).	
* * *	
42397–64–8 1,6-Dinitropyrene	1/11
42397–65–9 1,8-Dinitropyrene	1/11
* * *	
07496–02–8 6-Nitrochrysene	1/11
* * *	
57835–92–4 4-Nitropyrene	1/11

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FEDERAL COMMUNICATIONS COMMISSION

47 CFR Part 27

[WTB Docket No. 07–293; FCC 10–46]

Operations of Wireless Communications Services in the 2.3 GHz Band

AGENCY: Federal Communications Commission.

ACTION: Proposed rule.

SUMMARY: The Federal Communications Commission (Commission) seeks comment on revising the performance requirements for the 2.3 GHz Wireless Communications Service (WCS) band. The Commission is seeking comment on possible revision of the performance requirements (also known as buildout or construction requirements) for the 2.3 GHz WCS band to ensure that that the spectrum is used intensively in the public interest.

DATES: Interested parties may file comments on or before April 21, 2010, and reply comments on or before May 3, 2010. Written comments on the Paperwork Reduction Act proposed information collection requirements must be submitted by the public, Office of Management and Budget (OMB), and other interested parties on or before June 7, 2010.

ADDRESSES: You may submit comments, identified by WTB Docket No. 07–293, by any of the following methods:

- *Federal eRulemaking Portal: http://www.regulations.gov.* Follow the instructions for submitting comments.
- *Federal Communications Commission Web site: http://www.fcc.gov/cgb/ecfs.* Follow the instructions for submitting comments.
- *E-mail: ecfs@fcc.gov,* and include the following words in the body of the message, "get form." A sample form and directions will be sent in response.