ACTION: Proposed rule.

SUMMARY: EPA is proposing to approve Maryland's 111(d)/129 plan (the ''plan'') for the control of air pollutant emissions from hospital/medical/infectious waste incinerators (HMIWIs). The plan was developed and submitted to EPA by the Maryland Department of the Environment, Air and Radiation Management Administration (MARMA), on April 14, 2000. EPA is publishing this approval action without prior proposal because we view this as a noncontroversial action and anticipate no adverse comments.

DATES: Comments must be received in writing by October 5, 2000.

ADDRESSES: Comments may be mailed to Denis M. Lohman, Acting Chief, Technical Assessment Branch, Mailcode 3AP22, Environmental Protection Agency, Region III, 1650 Arch Street, Philadelphia, Pennsylvania 19103–2029.

FOR FURTHER INFORMATION CONTACT: James B. Topsale at (215) 814–2190, or by e-mail at topsale.jim@epa.gov.

SUPPLEMENTARY INFORMATION: See the information provided in the direct final rule, of the same title, which is located in the rules section of the **Federal Register**.

Authority: 42 U.S.C. 7401–7671q.

Dated: August 21, 2000.

Bradley M. Campbell,

Regional Administrator, Region III.

[FR Doc. 00–22517 Filed 9–1–00; 8:45 am]

BILLING CODE 6560–50–P

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 372

[OEI-100004; FRL-6722-3]

RIN 2070-AC00

Addition of Diisononyl Phthalate Category; Community Right-to-Know Toxic Chemical Release Reporting

AGENCY: Environmental Protection Agency (EPA).

ACTION: Proposed rule.

SUMMARY: In response to a petition filed under section 313(e)(1) of the Emergency Planning and Community Right-to-Know Act (EPCRA), EPA is proposing to add a diisononyl phthalate (DINP) category to the list of toxic chemicals subject to the reporting requirements under EPCRA section 313 and section 6607 of the Pollution Prevention Act (PPA). EPA is proposing to add this chemical category to the EPCRA section 313 list pursuant to its authority to add chemicals and chemical categories because EPA believes this category meets the EPCRA section 313(d)(2)(B) toxicity criterion. The proposed addition of this category is based on DINP's carcinogenicity and liver, kidney, and developmental toxicity.

DATES: Comments, identified by the docket control number OEI-100004, must be received by EPA on or before December 4, 2000.

ADDRESSES: Comments may be submitted by mail, electronically, or in person. Please follow the detailed instructions for each method as provided in Unit I. of the **SUPPLEMENTARY INFORMATION** section of this document.

FOR FURTHER INFORMATION CONTACT: For technical information on this proposed rule contact: Daniel R. Bushman, Petitions Coordinator, Environmental Protection Agency, Mail Code 2844, 1200 Pennsylvania Ave., NW., Washington, DC 20460; telephone number (202) 260–3882, e-mail address: bushman.daniel@epa.gov. For general information on EPCRA section 313, contact the Emergency Planning and Community Right-to-Know Hotline, Environmental Protection Agency, Mail Code 5101, 1200 Pennsylvania Ave., NW., Washington, DC 20460, Toll free: 1–800–535–0202, in Virginia and Alaska: (703) 412–9877, or Toll free TDD: 1–800–553–7672.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

You may be affected by this action if you manufacture, process, or otherwise use any of the chemicals included in the proposed DINP category. Potentially affected categories and entities may include, but are not limited to:

Category	Examples of Potentially Interested Entities
Industry	SIC major group codes 10 (except 1011, 1081, and 1094), 12 (except 1241), or 20 through 39; industry codes 4911 (limited to facilities that combust coal and/or oil for the purpose of generating power for distribution in commerce); 4931 (limited to facilities that combust coal and/or oil for the purpose of generating power for distribution in commerce); or 4939 (limited to facilities that combust coal and/or oil for the purpose of generating power for distribution in commerce); or 4953 (limited to facilities regulated under the Resource Conservation and Recovery Act, subtitle C, 42 U.S.C. section 6921 *et seq.*), or 5169, or 5171, or 7389 (limited to facilities primarily engaged in solvent recovery services on a contract or fee basis)
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. Other types of entities not listed in the table could also be affected. To determine whether your facility would be affected by this proposed rule, 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 Can I Get Additional Information or Copies of this Document or Other Support Documents?

1. *Electronically.* You may obtain electronic copies of this document from the EPA Internet Home Page at http://www.epa.gov/. To access this document, on the Home Page select ''Laws and Regulations,'' ''Regulations and Proposed Rules,'' and then look up the entry for this document under the ''**Federal Register**—Environmental Documents.'' You can also go directly to the **Federal Register** listings at http://www.epa.gov/fedrgstr/.

2. *In person.* The Agency has established an official record for this proposal under docket control number OEI-100004. The official record consists of the documents specifically referenced in Unit VIII. of this proposal and other information related to this proposal,

including any information claimed as confidential business information (CBI). This official record includes the documents that are physically located in the docket, as well as the documents that are referenced in those documents. The public version of the official record does not include any information claimed as CBI. The public version of the official record is available for inspection in the TSCA Nonconfidential Information Center, North East Mall Rm. B-607, Waterside Mall, 401 M St., SW., Washington, DC. The Center is open from noon to 4 p.m., Monday through Friday, excluding legal holidays. The telephone number is (202) 260–7099.

C. How and to Whom Do I Submit Comments?

You may submit comments through the mail, in person, or electronically. Be sure to identify the appropriate docket control number (i.e., ''OEI-100004'') in your correspondence.

1.*By mail.* Submit written comments to: Document Control Office (7407), Office of Pollution Prevention and Toxics (OPPT), Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460.

2.*In person or by courier.* Deliver your comments to: OPPT Document Control Office (DCO) in East Tower Rm. G-099, Waterside Mall, 401 M St., SW., Washington, DC. The DCO is open from 8 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The telephone number for the DCO is: (202) 260–7093.

3.*Electronically.* Submit your comments electronically by e-mail to: ''oppt.ncic@epa.gov.'' Please note that you should not submit any information electronically that you consider to be CBI. Electronic comments must be submitted as an ASCII file avoiding the use of special characters and any form of encryption. Comments and data will also be accepted on standard computer disks in WordPerfect 6.1/8.0 or ASCII file format. All comments and data in electronic form must be identified by the docket control number OEI-100004. Electronic comments on this proposal may also be filed online at many Federal Depository Libraries.

D. How Should I Handle CBI Information that I Want to Submit to the Agency?

You may claim information that you submit in response to this proposal as CBI by marking any part or all of that information as CBI. Information so marked will not be disclosed except in accordance with procedures set forth in 40 CFR part 2. A copy of the comment that does not contain CBI must be submitted for inclusion in the public record. Information not marked confidential will be included in the public docket by EPA without prior notice. If you have any questions about CBI or the procedures for claiming CBI, please consult with the technical person identified in the **FOR FURTHER INFORMATION CONTACT** section.

II. Introduction

A. What is the Statutory Authority for this Proposed Action?

EPA is proposing this action under EPCRA section 313(d) and (e)(1), 42 U.S.C. 11023. EPCRA is also referred to as Title III of the Superfund Amendments and Reauthorization Act of 1986.

B. What is the General Background for this Proposed Action?

Section 313 of EPCRA 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. EPCRA section 313 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 forth criteria for these actions. Under EPCRA section 313(e)(1), any person may petition EPA to add chemicals to or delete chemicals from the list. EPA has added and deleted chemicals from the original statutory list. Pursuant to EPCRA section 313(e)(1), EPA must respond to petitions within 180 days either by initiating a rulemaking or by publishing an explanation of why the petition has been denied.

EPCRA section 313(d)(2) states that EPA may add a chemical to the list if any of the listing criteria 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, EPA must demonstrate that none of the criteria 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 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 issued a statement of petition policy and guidance in the **Federal Register** of February 4, 1987 (52 FR 3479) to provide guidance regarding the recommended content and format for submitting petitions. On May 23, 1991 (56 FR 23703), EPA issued guidance regarding the recommended content of petitions to delete individual members of the section 313 metal compounds categories. EPA has also 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. What is the Description of the Petition?

On February 29, 2000, EPA received a petition from the Washington Toxics Coalition (WTC) requesting EPA to add DINP to the list of toxic chemicals subject to reporting under EPCRA Section 313 and PPA section 6607. The WTC contends that DINP causes cancer, systemic toxicity, developmental toxicity, and endocrine disruption, and therefore should be added to the list of chemicals subject to reporting under EPCRA section 313 and PPA section 6607. The petitioner alleges that DINP is ''a dangerous phthalate ester that is used as the principal plasticizer in toys and many other products used by children and adults.'' WTC also claims that ''DINP has been shown to cause cancer

and other very serious toxic effects.'' The petitioner also asserts that ''in every study conducted to measure DINP exposure from children's use of plastic, DINP has been shown to migrate from the plastic into saliva when the plastic object is chewed or put into the child's mouth (Babich, 1998).''

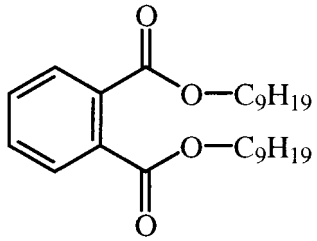
IV. What was EPA's Technical Review of Diisononyl Phthalate (DINP)?

In reviewing DINP for listing, EPA conducted a thorough hazard assessment and has preliminarily determined, based on the weight-of-the evidence, that there is sufficient evidence to establish that the DINP category met the statutory criteria for addition to EPCRA section 313. To make this determination, EPA senior scientists reviewed readily available toxicity information on the chemical for each of the following effect areas: acute human health effects; cancer and other chronic human health effects; and environmental effects. In addition, EPA reviewed information on the environmental fate of the chemical. The review is summarized in this proposal, and a more detailed discussion for each related topic can be found in EPA's technical report (Ref. 1). Referenced studies are contained in the public docket.

The hazard assessment was conducted in accordance with relevant EPA guidelines for each adverse human health or environmental effect (Refs. 14, 15, 16, 17, 18 and 21). During this assessment, the severity and significance of the effects induced by the chemical, the dose level causing the effect, and the quality and quantity of the available data, including the nature of the data (e.g., human epidemiological, laboratory animal, field or workplace studies) and confidence level in the existing data base, were all considered. EPA's assessment preliminarily concluded that the DINP category can reasonably be anticipated to cause carcinogenicity and liver, kidney, and developmental toxicity. In light of the continuous assessment of the developmental and reproductive toxicity potential of phthalates by the National Toxicology Program, the Agency may decide to evaluate potential hazards from other branched alkyl di-ester phthalates in the future (e.g., with eight or ten carbon alkyl chains).

A. What is the Chemistry and Use of DINP?

Diisononyl phthalates (DINP) are the branched alkyl di-esters of 1,2-benzenedicarboxylic acid in which the alkyl ester moieties contain a total of nine carbons. They constitute a family of di-ester phthalates widely used as plasticizers. They are colorless, oily liquids with high boiling points, low volatilities, and are poorly soluble in water (less than 10^{-4} milligrams per liter (mg/L)). Multiple CAS numbers are associated with DINP: 28553-12-0, 71549-78-5, 14103-61-8 and 68515-48-0. There is no single generic CAS number that represents all DINPs. The chemicals represented by CAS numbers 28553-12-0, 71549-78-5, and 14103-61-8 consist of a mixture of isomers (compounds which have the same molecular formula but differ in the arrangement of their atoms). The alkyl ester moieties of the DINP esters represented by the three CAS numbers stated above are branched and contain a total of nine carbons. These alkyl ester moieties are represented by the molecular formula C_9H_{19} (see structure below).



The molecular formulas of these nine-carbon alkyl ester moieties are the same for these DINP isomers. They differ in structure due to the arrangement of the carbons in the alkyl ester moieties. CAS number 68515-48-0 is also considered a DINP, but unlike the chemicals represented by the other three CAS numbers discussed above, 68515-48-0 consists of di-ester phthalates with nine-carbon alkyl ester moieties (approximately 70% by weight), mixed with lesser amounts of di-ester phthalates with eight- and ten-carbon alkyl ester moieties.

Of the chemicals represented by the four CAS numbers stated above, two (68515-48-0 and 28553-12-0) were reported by industry to EPA under the Inventory Reporting Regulations at 40 CFR part 710 of having production volumes of greater than 10,000 pounds per year. Actual production volumes for the chemicals represented by these two CAS numbers ranged in the millions of pounds per year. While reviewing data for the hazard assessments, it was noted that only a limited number of studies reported the CAS numbers for the DINP test chemical base stocks. When reported, the CAS numbers were either 68515-48-0 or 28553-12-0. These two CAS numbers represent the primary DINP products manufactured commercially in the United States. Again, these two CAS numbers represent a mixture of DINP isomers and not any one single specific DINP isomer. There was no literature available for review which identified a single specific DINP isomer as the test chemical. Please refer to EPA technical report (Ref. 1) for the full report on chemistry and environmental fate.

The principle use of DINP is as a plasticizer, particularly in the production of polyvinyl chloride (PVC). The treatment of plastics with DINP provides greater flexibility and softness to the final product. Some of the uses of DINP treated plastics are the production of coated fabrics, plastic toys, electrical insulation, and vinyl flooring. In 1999, at the request of the U.S. Consumer Product Safety Commission (CPSC), manufacturers voluntarily removed DINP from toys intended to be mouthed and intended to be used by children under age 3, due to health concerns. The voluntary action has had little impact on the demand for DINP as DINP is used in other types of toys (e.g., squeeze toys) and in other products (Refs. 1 and 15).

Approximately 2 billion pounds of phthalate plasticizers are produced in the United States each year. Of this total, production of DINP represents approximately 10% to 15% of the market, or 200 to 300 million pounds per year (Ref. 1). This figure is supported by the *Chemical Economics Handbook* (CEH), published by SRI, a proprietary source of information on the chemical industry which estimates a 1999 U.S. production of DINP of 250 million pounds (Ref. 20). Domestic consumption is approximately equal to production (Ref. 1).

B. What are the Environmental Fate Data for DINP?

Due to the limited available information specific to DINP, some of the information presented is based upon other di-ester phthalates, in particular di-octyl phthalates. Because of the close similarity in structure and physical-chemical properties of DINP to other di-ester phthalates, appropriate environmental fate analogies can be deduced for DINP (Ref. 1).

In water, hydrolysis is not considered a major mechanism for the degradation of phthalate esters under typical environmental conditions. At a neutral pH, phthalate esters are hydrolyzed at slow rates. The hydrolysis of DINP can be characterized as a two-step process, with the first step resulting in the creation of a monoester phthalate and one free nine-carbon alcohol molecule, and the second hydrolysis step resulting in the formation of phthalic acid and the

creation of a second nine-carbon alcohol (Ref. 1).

Due to its physical-chemical properties, DINP can be expected to partition in the environment to soils and sediments. Modeling results indicate that DINP would reside in the sediment fraction of rivers, ponds, and eutrophic lakes where they would be susceptible to biological degradation (Ref. 1). Microorganisms from diverse environments have been shown to degrade phthalate esters and associated degradation products. The microbial metabolism of phthalates under both aerobic and anaerobic conditions begins by ester hydrolysis resulting in the formation of the monoester and the corresponding alcohol. The rate of degradation is dependent upon chemical concentration and test matrix. Half-life values range from weeks to months. Biological degradation of the phthalates appears to be the dominant loss mechanism in the environment.

Very limited experimental data are available on the bioaccumulation potential of DINP. In general it can be stated that phthalates that are readily biotransformed have limited potential to bioaccumulate in most aquatic and terrestrial food animals. The Estimation Programs Interface for Windows (EPIWIN) model estimates a BCF value of 3.162 for DINP, indicating low bioaccumulation potential (Ref. 1).

Based on the available environmental fate data and model estimates, DINP is not expected to persist in most waters and soils or to bioaccumulate in aquatic or terrestrial organisms.

C. What are the Absorption and Metabolism Data for DINP?

DINP is well absorbed in the gastrointestinal (GI) tract of the rat and readily distributed to major tissues, particularly the liver, within 1 hour of administration; studies have shown that the majority of an oral dose of ^{14}C-DINP is excreted in the urine with the majority of the radiolabeled species appearing within 24 to 48 hours. DINP is poorly absorbed through the skin. In a dermal absorption study in rats, only 3% of the applied dose was recovered by the end the 7 days (Ref. 1). DINP is de-esterified to the monoester in the GI tract which is further metabolized by side chain oxidation to the oxidation products (ketones, diacids, aldehydes/alcohols) or by hydrolysis to phthalic acid occurring primarily in the liver. A major sex difference is demonstrated in the recovery of low amounts of the monoester oxidation products such as in the GI tract of female rats. This may suggest that intestinal hydrolysis of the diester is more limited in female rats. Livers had the highest concentration of radioactivity, followed by kidney, blood, muscle, and fat. There was no evidence of accumulation of DINP or metabolites in blood or tissue following repeated dosing and all metabolic products were completely eliminated by 72 hours.

D. What is EPA's Toxicity Evaluation for DINP?

1.*What is EPA's evaluation of the chronic toxicity of DINP?*

a.*What developmental toxicity data were found for DINP?* DINP has been shown to cause developmental toxicity in rats exposed during gestation to doses as low as 250 milligrams per kilogram per day (mg/kg/day). Developmental effects were observed in a two-generation reproductive study in rats, where the mean pup body weights in males and females of the first generation (F_1) were significantly reduced at all doses including 250 mg/kg/day, the lowest dose tested, by postnatal day (PND) 21. In the second generation (F_2), the mean female pup weight was significantly reduced at 250 mg/kg at PND 7 and male and female pup body weights were reduced on PND 7, 14 and 21 at 290 mg/kg/day. The significant decreases in the mean body weight of pups from two generations may result in serious developmental delays in growth throughout the lifetime of the rat. In a recent meeting conducted by the Consumer Product Safety Commission's Chronic Hazard Advisory Panel on DINP (Ref. 2) the data from the two-generation reproductive toxicity study on DINP (Ref. 3) were analyzed using benchmark dose analysis to more precisely define the dose at which developmental effects would be expected. The serious effect noted was a reduction in offspring (both F_1 and F_2) body weight at all dietary levels during the lactational phase. The estimated point of departure from the data was 200 to 260 mg/kg/day, which was consistent with the experimental dose of 250 mg/kg/day.

Skeletal variations including extra cervical and accessory (14th) ribs were significantly increased in two developmental studies in two different strains of rats. There were statistically significant increases in the percentage of litters with dilated kidney pelvises in both studies (Refs. 4 and 5). Developmental toxicity with the kidney and skeletal system as target organs was evident in the study conducted in Wistar dams given 1,000 mg/kg/day (Ref. 4). There were statistically significant increases in the number of affected fetuses per litter that had rudimentary cervical ribs and accessory 14th ribs in the high dose group. Skeletal malformations (i.e., shortened and bent long bones) were observed in the high dose group. There were increased incidences of dilated kidney pelvises at the high dose; three fetuses also had a total absence of kidney and ureter development. The same skeletal variations were demonstrated in offspring in Sprague-Dawley dams given 500 mg/kg/day while the kidney effects were observed at 1,000 mg/kg/day (Ref. 5). These skeletal variations and kidney effects occurred in the absence of or at minimal maternal toxicity (decreased body weight gain or increased organ weight). While the effect of extra lumbar ribs may not be considered serious malformations, the effect on cervical ribs is of great toxicological concern. Cervical ribs are an uncommon finding and their presence may indicate a disruption of gene expression leading to this structural anomaly (Ref. 22). In addition, there is concern that cervical ribs may interfere with normal nerve function and blood flow. The kidney effects in fetuses might lead to progressive kidney damage and impaired kidney function and therefore are considered to be serious.

b. *What other chronic toxicity effects data were found for DINP?* Increased liver weight and liver enzyme activities occurred at doses of DINP as low as 152 mg/kg in rats and chronic liver lesions were noted at 307 mg/kg/day. These liver effects are indicators of serious liver damage produced by DINP. In addition, these effects are early indicators of the tissue damage which leads to DINP-induced liver tumors (Ref. 7).

In addition to chronic liver toxicity, biochemical indicators of chronic kidney toxicity were evident in male rats given DINP at 307 mg/kg/day and female rats given DINP at 885 mg/kg/day. Also, chronic progressive irreversible kidney damage (nephropathy) occurred in female mice exposed to DINP at 1,888 mg/kg/day which lead to early mortality (Refs. 6 and 7).

c. *What carcinogenicity data were found for DINP?* DINP is a liver carcinogen in rats and mice. Liver tumors have been demonstrated in male F-344 rats exposed to dietary DINP at 733 mg/kg/day and female and male B6C3F1 mice exposed to 335 and 741 mg/kg/day, respectively, for 2 years (Ref. 6). Based on these data, EPA currently believes that DINP is a carcinogen.

One issue that has been raised with respect to other phthalate esters, such as di-(ethylhexyl) phthalate (DEHP), is the mechanism of the tumor production in

rodents (peroxisome proliferation-induced hepatocarcinogenicity) and its relevance to human cancer risk. DEHP's cancer classification is currently being reviewed by the Agency. As with DEHP, in the DINP studies the liver tumor production in rodents is associated with peroxisome proliferation. Several subchronic and chronic studies in rats (21-day, 13 week) demonstrate biochemical evidence of dose-related peroxisome proliferation in liver. Studies in rat hepatocytes indicate that the monoester (MINP) is the active form of DINP which stimulates peroxisomes. It has been suggested that liver tumors induced by chronic peroxisome proliferation are unique to rodents in that rats and mice are particularly responsive to peroxisome proliferators whereas other species (hamsters, guinea pigs, primates and humans) are relatively resistant. However, the Agency believes that there are still questions regarding the relationship between liver tumors and peroxisome proliferation. In accordance with EPA's cancer guidelines (Refs. 14 and 21), in the event that the data are insufficient to demonstrate that a response in animals is not relevant to any human situation, the default assumption is that positive effects in animal studies indicate that the agent under study can have carcinogenic potential in humans. Therefore, at this time, EPA's belief that DINP can reasonably be anticipated to cause cancer in humans is unchanged.

DINP has been shown to induce kidney tumors in male F-344 rats after prolonged exposure (2 year) to high doses (733 mg/kg/day) of dietary DINP (Ref. 7). These tumors occurred in male rats at high doses and a male rat-specific mechanism involving alpha $_{2u}$-globulin accumulation in the kidney has been postulated. However, in the same study, indicators of kidney toxicity occurred in females rats given 885 mg/kg/day as evidenced as a high urine creatinine clearance, suggesting a compromised ability to concentrate in the kidney tubules and a high blood urea nitrogen (biomarker of kidney damage). Also, in a chronic toxicity study in mice, female mice exposed to 1,888 mg/kg/day had a statistically significant increase in the incidence and severity of chronic progressive nephropathy which lead to early mortality (Ref. 6). The kidney toxicity in female rats and the chronic progressive kidney toxicity in female mice argues against a male rat-specific mechanism (i.e. alpha $_{2u}$-globulin accumulation). The tumors in male rats could be the result of a response to kidney damage induced by chronic DINP administration and not solely a consequence of the alpha $_{2u}$-globulin mechanism (Ref. 8).

There is also a dose-related statistically significant increase in the incidence of mononuclear cell leukemia (MNCL) with associated anemia (decreased hemoglobin levels and red blood cell numbers) and decreased body weight gain in male and female Fisher rats exposed to doses of 152/307 mg/kg/day and higher (Ref. 7). It is known that MNCL is life threatening in Fisher rats and results in a decreased life span. In addition, although MNCL is recognized as a common neoplasm in Fisher rats, the mechanism of producing MNCL is not completely understood. Therefore, the significance of MNCL and its biological relevance for human cancer risk remains uncertain and cannot be discounted.

d. *What genotoxicity data were found for DINP?* DINP has been evaluated for gene mutations, cytogenetic effects, cell transformation ability and unscheduled DNA synthesis and none of the data evaluated indicate that DINP is mutagenic or genotoxic (Ref. 1).

e. *What reproductive toxicity data were found for DINP?* No reproductive toxicity was observed in a one- and two-generation reproductive study in rats at doses as high as 1,000 mg/kg (Ref. 3). However, in this study, landmarks of sexual maturation (i.e., preputial separation, anogenital distance, nipple retention, biochemical and structure of the developing reproductive system) were not examined. These landmarks of sexual maturation are used to assess the effects of a chemical on reproductive tract development. Other phthalates, such as DEHP and dibutyl phthalate, have been shown to have an effect on reproductive tract development. At this time, therefore, EPA has preliminarily determined that the data are insufficient to indicate whether or not DINP exposures are associated with detectable effects on reproductive function.

f. *What endocrine disruption data were found for DINP?* There are reports that phthalates may have endocrine modulating effects. Early reports suggested that DINP was very weakly estrogenic in *in vitro* screening assays using a recombinant yeast screen and estrogen-responsive human breast cancer cell lines (Ref. 9). Although these screening assays are highly specific for estrogen, later *in vivo* studies have shown that neither DINP nor any other phthalate was positive in screening assays such as vaginal certification and uterotrophic assays in mice (Ref. 10). Therefore, EPA has preliminarily determined that there is insufficient evidence, at this time, to demonstrate whether or not DINP causes hormone disruption.

2. *What acute toxicity data were found for DINP?* Acute toxicity studies in rats and rabbits indicate that DINP, like other long chain phthalate esters, has low oral (rat oral LD_{50} >10 grams/kilogram (g/kg)) (LD_{50}, i.e., the dose that is lethal to 50% of test organisms) and dermal (rabbit dermal LD_{50} >3 g/kg) acute toxicity. Acute inhalation toxicity (LC_{50}) (i.e., the concentration that is lethal to 50% of test organisms) data are not available because information on measurements of test-chamber atmospheric levels were generally inadequately reported or the generating and monitoring concentrations were not described. However, the low vapor pressure of DINP usually precludes inhalation of any significant amount except perhaps as an aerosol adsorbed to airborne particulates. DINP is only minimally irritating to eyes and skin. In human adults, it is estimated that the probable lethal oral dose is between .5 and 5 g/kg (1 ounce—1 quart/adult). DINP does not penetrate the skin very well (3% dermal absorption) and is not a dermal sensitizer (Ref. 1). Based on the available data, EPA has preliminarily determined that DINP does not cause acute toxic effects.

3. *What ecotoxicity data were found for DINP?* Based on available ecotoxicity data, this group of chemicals has not been tested at levels high enough to cause 50% mortality in fish or invertebrates. In one study, insufficient mortality was observed at the highest concentrations tested (Ref. 12) to calculate acute toxicity values. Technically, the acute no observed effect concentrations (NOECs) for these chemicals are greater than the highest concentrations tested. The lowest effect level for assessment purposes is <0.06 mg/L for Daphnia magna, and 0.10 mg/L for fathead minnows.

The only reported studies with actual effects were embryo larval studies with Channel catfish, Fowler's toad, and Leopard frog, with reported effects noted between 1 and 100 parts per million. The water solubility of DINP must be considered as a factor in these studies. Since this compound is sparingly soluble (water solubility is approximately 10^{-4} mg/L), it would be difficult to conduct aquatic toxicity studies at concentrations high enough to cause mortality. All of the published aquatic toxicity studies have unsuitable test designs for these poorly water soluble compounds.

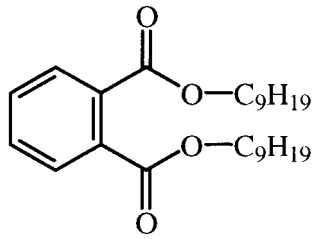
The reported maximum acceptable toxicant concentration of 0.055 mg/L (Ref. 13), actually was due to physical entrapment of Daphnids at the surface

of the test vessel, rather than due to direct toxicity.

Based on the available data, EPA cannot preliminarily determine whether or not DINP can cause or reasonably be anticipated to cause, because of its toxicity, a significant adverse effect on the environment.

E. What is the Basis for a DINP Category?

In this proposal, the Agency has classified DINP as a category consisting of any branched alkyl di-ester of 1,2-benzenedicarboxylic acid in which the alkyl ester moieties contain a total of nine carbons. The molecular formula for DINP is $C_{26}H_{42}O_4$. The structure of DINP is shown below with the nine carbon alkyl ester moieties represented by the molecular formula -C_9H_{19}.



EPA is proposing to create the DINP category for several reasons. There is no single CAS number which encompasses all DINP isomers. The human health hazard assessment included the review of studies conducted with chemical test base stocks which consisted of solely diisononyl phthalates isomers or test stocks composed of mostly DINP (approximately 70% by weight). Of the studies reviewed, all were found to show serious adverse human health effects (liver, kidney, or developmental toxicity or carcinogenicity) regardless of the test base stock that was used. The common component of the tested materials are the branched alkyl di-esters of 1,2-benzenedicarboxylic acid with nine carbon alkyl ester moieties. EPA believes that the available data on the carcinogenicity and liver, kidney, and developmental toxicity for certain members of the DINP category for which EPA has data are sufficient for listing those members under EPCRA section 313(d)(2)(B). EPA also believes that there is sufficient information to conclude that based on structural and physical/chemical property similarities to those members of the category for which data are available it is reasonable to anticipate that all members of the DINP category will exhibit carcinogenicity and/or liver, kidney, and developmental toxicity in humans. For these reasons and because no one CAS number adequately covers all diisononyl phthalate isomers, EPA is proposing a DINP category.

V. What is the Summary of EPA's Technical Review?

After a review of the available data in response to this petition, the Agency has preliminarily determined that there is sufficient evidence for listing this category of DINP on EPCRA section 313 pursuant to EPCRA section 313 (d)(2)(B) because the DINP category can reasonably be anticipated to cause carcinogenicity and liver, kidney, and developmental toxicity. The following is a summary of the findings.

DINP has been shown to cause developmental toxicity in prenatal rats. This developmental toxicity included significant decreases in the mean body weight of pups from two generations which may result in serious developmental delays in growth throughout the lifetime. In addition, skeletal variations were observed which may interfere with normal nerve function and blood flow. Kidney effects in fetuses were observed which might lead to progressive kidney damage and impaired kidney function.

DINP has been shown to cause chronic liver and kidney toxicity in rats and mice. The liver effects are indicators of the serious liver damage produced by DINP and are early indicators of the tissue damage which leads to DINP-induced tumors. In addition to chronic liver toxicity, biochemical indicators of chronic kidney toxicity were evident in male and female rats. Also, chronic progressive irreversible kidney damage (nephropathy) occurred in female mice which lead to early mortality.

DINP has been shown to be a liver carcinogen in rats and mice, to induce kidney tumors in male rats, and to increase the incidence of mononuclear cell leukemia.

VI. What is EPA's Explanation of the Petition Response and Rationale for Listing?

EPA is proposing to grant the petition to add DINP to the EPCRA section 313 list of toxic chemicals. In light of the discussion in Unit IV.E., EPA is proposing to add a chemical category entitiled ''Diisononyl Phthalate (DINP) category,'' to the EPCRA section 313 list of toxic chemicals. This category will include the four CAS numbers that represent the DINP esters identified by name and CAS number in Unit IV.A., as well as any other branched alkyl di-ester of 1,2-benzenedicarboxylic acid in which the alkyl ester moieties contain a total of nine carbons. As EPA has explained in the past (see 59 FR 61442-61443 November 30, 1994), the Agency believes that EPCRA allows the Agency, in its discretion, to add a chemical category to the list, where EPA identifies the toxic effect of concern for at least one member of the category and then shows why that effect can reasonably be expected to be caused by all other members of the category. Here, individual toxicity data do not exist for all members of the proposed category; however, as discussed in Unit IV.E. of this preamble, EPA believes that the available data on the carcinogenicity and liver, kidney, and developmental toxicity for certain members of the DINP category are sufficient for listing those members under EPCRA section 313(d)(2)(B). EPA currently believes that it is reasonable to anticipate that all members of the DINP category as described will exhibit carcinogenicity and liver, kidney, and developmental toxicity in humans and that creating a category of DINP is the most appropriate way to list this class of chemicals.

EPA does not believe that it is required to consider exposure for chemicals that are moderately high to highly toxic based on a hazard assessment when determining if a chemical can be added for chronic effects pursuant to EPCRA section 313(d)(2)(B) (59 FR 61432, 61433, 61440-61442). The technical review of the toxicity data clearly indicates that DINP is known to cause or can reasonably be anticipated to cause cancer and other serious or irreversible chronic liver, kidney, and developmental toxicity in humans. EPA has preliminarily determined that DINP can reasonably be anticipated to cause cancer and that the observed liver, kidney, and developmental toxicity occur at relatively low doses, and thus the Agency believes DINP to have moderately high to high chronic toxicity for each of these effects. EPA also believes that there is sufficient information to conclude that all of the members of the DINP category are moderately high to highly toxic based on structural and physical/chemical property similarities to those members of the category for which data are available. EPA, therefore, does not believe that an exposure assessment is required or appropriate for determining whether the DINP category (or its members) proposed for listing in this rulemaking meet the criteria of EPCRA section 313(d)(2)(B).

In sum, EPA believes that there is sufficient evidence to show that the DINP category is known to cause or can reasonably be anticipated to cause cancer and other serious or irreversible chronic liver, kidney, and

developmental toxicity in humans. EPA believes it has the authority to list the DINP category under EPCRA section 313 based on any one of these effects. Therefore, EPA believes that this chemical category meets the EPCRA section 313(d)(2)(B) criteria for listing.

For purposes of EPCRA section 313, threshold determinations for chemical categories must be based on the total of all chemicals in the category (see 40 CFR 372.25(d)). For example, a facility that manufactures three members of a chemical category would count the total amount of all three chemicals manufactured towards the manufacturing threshold for that category. When filing reports for the DINP category, the releases are determined in the same manner as the thresholds. One report is filed for the category and all releases are reported on one Form R (the form for filing reports under EPCRA section 313 and PPA section 6607). With regard to mixtures of chemicals, facilities only need to report releases and other waste management activities for the portion of the mixture that is covered by the category. For example, CAS number 68515-48-0 represents a mixture of phthalate esters which includes alkyl ester moieties containing eight, nine, and ten carbons. For such a mixture only the percentage of the mixture that contains phthalate esters that have nine carbons in the alkyl ester moiety would be reportable under the DINP category.

VII. What Issues is EPA Requesting Comment On?

EPA requests public comment on this proposal to add a DINP category to the EPCRA section 313 list of toxic chemicals. Specifically, EPA requests comment on its technical review of DINP, including its environmental fate, absorption and metabolism, toxicity, and carcinogenicity, and on EPA's preliminary determination that there is sufficient evidence to establish that the DINP category meets the statutory criteria for addition to EPCRA Section 313. EPA also requests that commenters provide any additional data they may have on the environmental fate, absorption and metabolism, toxic effects and carcinogenicity of DINP. Finally, EPA requests comment on alternative methods for adding DINP instead of by category.

VIII. What are the References Cited in this Proposed Rule?

1. USEPA, OEI. 2000. Office of Environmental Information, Becki Madison, Christine Augustyniak, Ron Bloom, Candace Brassard, Ossi Meyn, Nicole Paquette, Pam Russell, and John Scalera. Technical Review of Diisononyl Phthalate.

2. McKee, RH. 2000. Presented at Meeting of the Consumer Product Safety Commission's Chronic Hazard Advisory Panel on Diisononyl Phthalate (DINP). Bethesda, MD. Exxon Mobil Biomedical Sciences, Inc.

3. Waterman, SJ, Keller LH, Trimmer, GW, Freeman, JJ, Nikiforov, AI, Harris, SB, McKee, RH. 2000. Two generation reproduction study in rats given di-isononyl phthalate in the diet. *Reprod Toxicol* Jan-Feb;14(1):21-36.

4. Hellwig, J, Freudenberger, H, Jack, R. 1997. Differential prenatal toxicity of branched phthalate esters in rats. *Food and Chem Toxicol* 35: 501-512.

5. Waterman, SJ, Ambroso, JL, Keller, LH, Trimmer, GW, Nikiforov, AI, Harris, SB. 1999. Developmental toxicity of di-isodecyl and di-isononyl phthalates in rats. *Repro Toxicol* 13(2):131-136.

6. TSCATS Doc 89980000046 Moore MRCL.1998 Oncogenicity study in mice with di(isononyl)phthalate including ancillary hepatocellular proliferation and biochemical analyses. Fiche 1B OTS05562833 Old Doc 8EHQ0119813083. Aristech Chem Corp/ Covance 2598-105.

7. TSCATS Doc 89980000308, Moore MRCL.1998. Oncogenicity study in rats with di(isononyl)phthalate including ancillary hepatocellular proliferation and biochemical analyses. Fiche 1B OTS05562832 Old Doc 8EHQ099813083 Aristech Chem Corp/Covance 2598-104.

8. Lington, AW, Bird, MG, Plutnick, RT, Stubblefield, WA, Scala RA. 1997. Chronic toxicity a carcinogenic evaluation of diisononyl phthalate in rats. *Fundam Appl. Toxicol* 36: 79-89.

9. Harris, CA, Henttu, P, Parker, MG, Sumpter, JP. 1997. The estrogenic activity of phthalate esters in vitro. *Environ Health Perspect* 105: 802-811.

10. Zacharewski, TR, Meek, MD, Clemons, JH, Wu, ZF, Fielden, MR, Matthews, JB. 1998. Examination of the *in vitro* and *in vivo* estrogenic activities of eight commercial phthalate esters. *Toxicol Sci* 46: 282-293.

11. Staples, CA, Peterson, DR, Parkerton, TF, Adams, WJ. 1997. The Environmental Fate of Phthalate Esters: A Literature Review. *Chemosphere*, 35(4): 667-749.

12. Adams, WJ, Biddinger, GR, Robillard, KA, and Gorsuch, JW. 1995. A summary of the acute toxicity of 14 phthalate esters to representative aquatic organisms. *Journal of Environmental Toxicology and Chemistry*. 14(9): 1569-1574.

13. Rhodes, JE, Adams, WJ, Biddinger, GR, Robillard, KA, and Gorsuch, JW. 1995. Chronic Toxicity of 14 phthalate esters to *Daphnia magna* and rainbow trout (*Oncorhynchus mykiss*). *Journal of Environmental Toxicology and Chemistry*. 14(11): 1967-1976.

14. USEPA. 1996. *Proposed Guidelines for Carcinogen Risk Assessment*. EPA/600/P-92/003c, April 1996.

15. USEPA. 1996. *Reproductive Toxicity Risk Assessment Guidelines*. EPA/630/R-96/009, September 1996.

16. USEPA. 1991. *Guidelines for Developmental Toxicity Risk Assessments*. 56 **Federal Register** 63789 (December 5, 1991).

17. USEPA. 1989. *Reference Dose (RfD): Description and Use in Health Risk Assessments*. Appendix A to the Integrated Risk Information System (IRIS). http://www.epa.gov/iris/cfd.htm.

18. USEPA. 1998. *Office of Prevention Pesticides and Toxic Substances Harmonized Test Guidelines*. Series 870. Health Effects. Guidelines OPPTS 870.3700, 870.3800, 870.4300. http://www.epa.gov/docs/OPPTS_Harmonized/870_Health_Effects_Test_Guidelines/.

19. Babich, M.A. 1998. The risk of chronic toxicity associated with exposure to diisononyl phthalate (DINP) in children's products. Consumer Product Safety Commission. Bethesda, MD.

20. Chemical Economics Handbook. 2000. Stanford Research International, Menlo Park, CA.

21. USEPA. 1987. *Guidelines for Carcinogenic Risk Assessment*. EPA/600/8-87/045, August 1987.

22. National Toxicology Program. 2000. CERHR Expert Panel Review of Phthalates. Center for the Evaluation of Risks to Human Reproduction. May 24, 2000. http://cerhr.niehs.nih.gov/news/ms7_18_00.html.

IX. What are the Regulatory Assessment Requirements for this Proposed Action?

A. Executive Order 12866

Under Executive Order 12866 (58 FR 51735, October 4, 1993), the Agency must determine whether the regulatory action is subject to review by the Office of Management and Budget (OMB). Pursuant to the terms of this Executive Order, it has been determined that this proposed rule is not a ''significant regulatory action'' and is therefore not subject to OMB review.

B. Regulatory Flexibility Act

Pursuant to section 605(b) of the Regulatory Flexibility Act, 5 U.S.C. 601 *et. seq.*, the Agency hereby certifies that this proposed action does not have a significant adverse economic impact on a substantial number of small entities.

Based on what EPA currently knows about DINP EPA believes that, under current EPCRA reporting thresholds, between 35 and 100 additional TRI reports would be filed and no facility will file more than 1 additional TRI report. EPA estimates a first year time burden on reporting facilities to be 78 hours, or less, for a cost of $5,640 per affected facility or less. These costs are approximately $5,640 per report in the first year (for a total first year cost of between $195,000 and $565,000). In subsequent years this cost falls to $4,000 per report (for a total cost of $140,000 to $400,000). These estimates include the time needed to review instructions; search existing data sources; gather and maintain the data needed; complete and review the collection of information; and transmit or otherwise disclose the information. The actual burden on any specific 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.

The estimated time burden for the first year of reporting is 0.4% of the labor hours of the firms with exactly ten full-time employees, which have the smallest number of total labor hours of any firm subject to this rule. 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. Thus this rule is not expected to have a significant adverse economic impact on a substantial number of small entities. A more detailed economic analysis is located in EPA's technical report (Ref. 1).

C. 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 1B 2070-0093 (EPA ICR 1B1363): those related to Form A under OMB control 2070-0143 (EPA ICR 1B 1704): and those related to trade secret designations under OMB Control 2070-0078 (EPA ICR1B 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 of 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 78 hours per report in the first year, at an estimated cost of $5,640 per Form R (for a total first year cost of between $195,000 and $565,000). In subsequent years, the burden for collecting this information is estimated to average 55 hours per report, at an estimated cost of $4,000 per report (for a total cost of $140,000 to $400,000). 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 both 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 accuracy of the Agency's estimate of the burden of the propose 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 Sandy Farmer, Office of Environmental Information (2822), 1200 Pennsylvania Avenue, NW., Washington, DC 20460, by calling (202) 260-2740, or electronically by sending an e-mail message to ''farmer.sandy@epa.gov''.

D. Unfunded Mandates Reform Act and Executive Orders 13084 and 13132

Pursuant to Title II of the Unfunded Mandates Reform Act of 1995 (UMRA) (Public Law 104-4), EPA has determined that this proposed 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 1 year. It is estimated that the total cost of the rule, which is summarized in Unit IX.B. of this preamble, is $195,000 to $565,000 in the first year of reporting. In addition, today's proposal would not create a mandate on State, local or tribal governments, nor would it significantly or uniquely affect the communities of Indian tribal governments; therefore, it is not subject to the requirement for prior consultation with Indian tribal governments as specified in Executive Order 13084, entitled *Consultation and Coordination with Indian Tribal Governments* (63 FR 27655, May 19, 1998). Nor would this action have a substantial direct effect on 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, entitled *Federalism* (64 FR 43255, August 10, 1999).

E. Executive Order 12898

Pursuant to Executive Order 12898 (59 FR 7629, February 16 1994), entitled *Federal Actions to Address Environmental Justice in Minority Populations and Low Income Populations,* the Agency has considered environmental justice related issues with regard to the potential impacts of this action on environmental and health conditions in low-income and minority populations. By adding a DINP category to the list of toxic chemicals subject to reporting under section 313 of EPCRA, EPA would be providing communities across the United States (including low-income populations and minority populations) with access to data that may assist them in lowering exposures and consequently reducing 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 appropriates 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.

F. Executive Order 13045

Pursuant to Executive Order 13045, entitled *Protection of Children from Environmental Health Risks and Safety Risks* (62 FR 19885, April 23, 1997), if an action is economically significant under Executive Order 12866, the Agency must, to the extent permitted by law and consistent with the Agency's mission, identify and assess the environmental health risks and safety risks that may disproportionately affect children. Since this action would not be economically significant under Executive Order 12866, this action is not subject to Executive Order 13045.

G. National Technology Transfer and Advancement Act

Section 12(d) of the National Technology Transfer and Advancement Act of 1995 (NTTAA) (15 U.S.C. 272 note) directs EPA to use voluntary consensus standards in its regulatory activities unless doing so would be inconsistent with applicable law or impractical. Voluntary consensus standards are technical standards (e.g., materials specifications, test methods, and sampling procedures) that are developed or adopted by voluntary consensus standards bodies. The NTTAA directs EPA to provide Congress, through OMB, explanations when the Agency decides not to use available and applicable voluntary consensus standards. This action does not involve technical standards, nor did EPA consider the use of any voluntary consensus standards. In general, EPCRA does not prescribe technical standards to be used for threshold determinations or completion of EPCRA section 313 reports. EPCRA section 313(g)(2) states that ''In order to provide the information required under this section, the owner or operator of a facility may use readily available data (including monitoring data) collected pursuant to other provisions of law, or, where such data are not readily available, reasonable estimates of the amounts involved. Nothing in this section requires the monitoring or measurement of the quantities, concentration, or frequency of any toxic chemical released into the environment beyond that monitoring and measurement required under other provisions of law or regulation.''

List of Subjects in 40 CFR Part 372

Environmental protection, Chemicals, Community right-to-know, Hazardous substances, Intergovernmental relations, Reporting and recordkeeping requirements, Superfund, Toxic chemicals.

Dated: August 25, 2000.

Elaine G. Stanley,

Director, Office of Information Analysis and Access.

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

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

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

2. In § 372.65 by adding alphabetically one chemical category to paragraph (c) to read as follows:

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

* * * * *

(c) * * *

Category name	Effective date
* * * * * * *	
Diisononyl Phthalates (DINP): Includes all branched alkyl di-esters of 1,2 benzenedicarboxylic acid in which alkyl ester moieties contain a total of nine carbons.	1/1/01
* * * * * * *	

[FR Doc. 00–22656 File 9–1–00; 8:45 am]

BILLING CODE 6560–09–F

FEDERAL COMMUNICATIONS COMMISSION

47 CFR Part 73

[DA 00–1905; MM Docket No. 00–146, RM–9937; MM Docket No. 00–147, RM–9938; MM Docket No. 00–148, RM–9939; MM Docket No. 00–149, RM–9940]

Radio Broadcasting Services; Marietta, MS; Lake City, CO; Quanah, TX; Smiley, TX

AGENCY: Federal Communications Commission.

ACTION: Proposed rule.

SUMMARY: This document proposes four new allotments to Marietta, MS; Lake City, CO; Quanah, TX; and Smiley, TX. The Commission requests comments on a petition filed by Robert Sanders proposing the allotment of Channel 250A at Marietta, Mississippi, as the community's first local aural transmission service. Channel 250A can be allotted to Marietta in compliance with the Commission's minimum distance separation requirements with a site restriction of 1.3 kilometers (0.8 miles) east to avoid a short-spacing the licensed sites of Station WWMS(FM), Channel 248C1, Oxford, Mississippi,