for the State to use voluntary consensus standards (VCS), EPA has no authority to disapprove a SIP submission for failure to use VCS. It would thus be inconsistent with applicable law for EPA, when it reviews a SIP submission, to use VCS in place of a SIP submission that otherwise satisfies the provisions of the Clean Air Act. Thus, the requirements of section 12(d) of the National Technology Transfer and Advancement Act of 1995 (15 U.S.C. 272 note) do not apply. This proposed rule does not impose an information collection burden under the provisions of the Paperwork Reduction Act of 1995 (44 U.S.C. 3501 *et seq.*).

List of Subjects in 40 CFR Part 52

Environmental protection, Air pollution control, Intergovernmental relations, Particulate matter, Reporting and recordkeeping requirements.

Authority: 42 U.S.C. 7401 *et seq.*

Dated: April, 20, 2005.

Wayne Nastri,

Regional Administrator, Region IX.

[FR Doc. 05–11718 Filed 6–13–05; 8:45 am]

BILLING CODE 6560–50–U

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 372

[TRI–2005–0004; FRL–7532–4]

RIN 2025–AA17

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

AGENCY: Environmental Protection Agency (EPA).

ACTION: Proposed rule, notice of data availability.

SUMMARY: On September 5, 2000, EPA issued a proposed rule, in response to a petition filed under section 313(e)(1) of the Emergency Planning and Community Right-to-Know Act (EPCRA), 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 proposed to add this chemical category to the EPCRA section 313 toxic chemical 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 purpose of today's action is to inform interested parties that, in an effort to ensure adequate opportunities for input from all affected parties, EPA is making available for public comment a revised hazard assessment for DINP.

DATES: Comments must be received on or before September 12, 2005.

ADDRESSES: Submit your comments, identified by Docket ID No. TRI–2005–0004, by one of the following methods:

- *Federal eRulemaking Portal: http://www.regulations.gov.* Follow the on-line instructions for submitting comments.
- *Agency Web site: http://www.epa.gov/edocket.* EDOCKET, EPA's electronic public docket and comment system, is EPA's preferred method for receiving comments. 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, Attention Docket ID No. TRI–2005–0004.
- *Hand Delivery:* EPA Docket Center, (EPA/DC) EPA West, Room B102, 1301 Constitution Ave., NW., Washington, DC, 20004, telephone: 202–566–1744, Attention Docket ID No. TRI–2005–0004. 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. TRI–2005–0004. 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.epa.gov/edocket,* 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 EDOCKET, regulations.gov, or e-mail. The EPA EDOCKET and the Federal regulations.gov Web sites are ''anonymous access'' systems, 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 EDOCKET or 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, any form of encryption, and be free of any defects or viruses.

Docket: All documents in the docket are listed in the EDOCKET index at: *http://www.epa.gov/edocket.* Although listed in the index, some information is not publicly available, *i.e.*, CBI or other information whose disclosure is restricted by statute. Certain other material, such as copyrighted material, is not placed on the Internet and will be publicly available only in hard copy form. Publicly available docket materials are available either electronically in EDOCKET or in hard copy at the OEI Docket, EPA/DC, EPA West, Room B102, 1301 Constitution Ave., NW., Washington, DC. The Public Reading Room 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, Toxics Release Inventory Program Division, Office of Information Analysis and Access (2844T), Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460; telephone number: 202–566–0743; fax number: 202–566–0741; e-mail: *bushman.daniel@epamail.epa.gov,* for specific information on this proposed rule, or for more information on EPCRA section 313, the Emergency Planning and Community Right-to-Know Hotline, Environmental Protection Agency, Mail Code 5101, 1200 Pennsylvania Ave., NW., Washington, DC 20460, Toll free: 1–800–424–9346, in Virginia and Alaska: 703–412–9810 or Toll free TDD: 1–800–553–7672.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does This Notice Apply To Me?

You may be potentially affected by this notice if you manufacture, process, or otherwise use DINP. Potentially affected categories and entities may include, but are not limited to:

Category	Examples of potentially affected entities
Industry	SIC major group codes 10 (except 1011, 1081, and 1094); 12 (except 1241); or 20 through 39; or industry codes 4911 (limited to facilities that combust coal and/or oil for the purpose of generating power for distribution in commerce); or 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 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 information that you consider to be CBI electronically through EPA's electronic public docket or by e-mail. Commenters wishing to submit proprietary information for consideration must clearly distinguish such information from other comments and clearly label it as CBI. Send submissions containing such proprietary information directly to the following address only, and not to the public docket, to ensure that proprietary information is not inadvertently placed in the docket: Attention: OEI Document Control Officer, Mail Code: 2822T, U.S. EPA, 1200 Pennsylvania Ave., NW., Washington, DC, 20460. You may claim information that you submit to EPA as CBI by marking any part or all of that information as CBI (if you submit CBI on disk or CD ROM, 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 CBI). The EPA will disclose information claimed as CBI only to the extent allowed by the procedures set forth in 40 CFR part 2.

In addition to one complete version of the comment that includes any 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 and EPA's electronic public docket. If you submit the copy that does not contain CBI on disk or CD ROM, mark the outside of the disk or CD ROM clearly that it does not contain CBI. Information not marked as CBI will be included in the public docket and EPA's electronic public docket without prior notice. If you have any questions about CBI or the procedures for claiming CBI, please consult the person identified in the **FOR FURTHER INFORMATION CONTACT** section.

II. What Did EPA Propose and What Is the Purpose of This Notice?

In response to a petition to add diisononyl phthalate (DINP) to the EPCRA section 313 list of toxic chemicals, EPA published a proposed rule to add a DINP category to the EPCRA section 313 list (65 FR 53681, September 5, 2000). The proposed rule was based on information contained in the hazard assessment for DINP that was developed in response to the petition (Ref. 1). In response to comments on the proposal, EPA revised its hazard assessment for DINP. The purpose of this notice is to allow the public an opportunity to comment on a revised hazard assessment that EPA has developed for DINP (Ref. 2).

A. What Preliminary Determinations Did EPA Reach in the Proposed Rule?

After a review of the data available at the time, the Agency preliminarily determined that there was sufficient evidence to support the conclusion that DINP can reasonably be anticipated to cause carcinogenicity and liver, kidney, and developmental toxicity. The preliminary findings were summarized as follows.

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. (65 FR 53686, September 5, 2000).

EPA EAPA also stated that:

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. (65 FR 53686, September 5, 2000).

B. Why Has EPA Issued This Notice?

EPA received significant comments on its original hazard assessment for DINP both during the comment period for the proposed rule and in later submissions to the Agency. Based on the comments EPA received, the Agency decided to revise the DINP hazard assessment and subject the document to a peer review process. The revised DINP hazard assessment was reviewed by experts within EPA and then by a group of external peer reviewers. Based on these reviews EPA made additional revisions to the DINP hazard assessment. The comments received from the external peer reviewers and EPA's responses to those comments have been placed in the docket for this notice (Ref. 3). The revisions to the hazard assessment have an impact on the preliminary conclusions that EPA reached in the proposed rule. Therefore, EPA is making the revised DINP hazard assessment available for public comment through the publication of this notice.

III. How Has the Hazard Assessment Changed?

The original DINP hazard assessment has been updated to include the most recent data available on the toxicity of DINP and has been revised based on public comments and on feedback from internal and external peer reviewers. Based on public comment, the revised hazard assessment has been expanded

to include a more extensive discussion of each human health endpoint of concern. The significant substantive changes to the hazard assessment are discussed in the following sections.

A. What Changes Have Been Made to the Discussion of Carcinogenicity?

1. *Liver Cancer.* In the original DINP hazard assessment (Ref. 1), EPA stated that DINP was a liver carcinogen in rats and mice and could reasonably be anticipated to cause cancer in humans. Liver tumors were demonstrated in three independent chronic studies in rats and mice (Refs. 4, 5, and 6). At that time, EPA acknowledged that there was an ongoing scientific discussion regarding the role of peroxisome proliferation and peroxisome proliferation activating receptor-÷ (PPAR÷) activation in rodent liver tumors and its relevance to human cancer.

In accordance with EPA's cancer guidelines (Ref. 7), however, the data were considered insufficient to demonstrate that a response in animals was not relevant to any human situation. Therefore, the default assumption that positive effects in animal studies indicate that DINP could have carcinogenic potential in humans was warranted. Based on this default assumption, EPA's position in the original hazard assessment was that DINP could reasonably be anticipated to cause cancer in humans.

Recently, the Office of Prevention, Pesticides and Toxics Substances (OPPTS) presented a draft of a proposed OPPTS science policy on PPARαmediated hepatocarcinogenicity to the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) Science Advisory Panel (SAP), a Federal advisory committee. The SAP serves as the primary scientific peer review mechanism of OPPTS under Section 25(d) of FIFRA. The guidance document described an approach OPPTS proposed to use to evaluate the scientific information regarding the mode of action of PPAR÷ activating chemicals, such as DINP, in rodent liver tumors and the relevance of this mode of action for human liver cancer. EPA is currently reviewing the responses from the FIFRA SAP and will notify the public on a science policy decision. EPA therefore reserves judgment on whether DINP can reasonably be anticipated to cause liver cancer in humans, pending the results of this EPA review. The revised DINP hazard assessment has been updated to include a discussion of EPA's ongoing review.

2. *Mononuclear Cell Leukemia.* In the original DINP hazard assessment (Ref. 1), EPA stated that there were clear, statistically significant increases in the incidences of mononuclear cell leukemia (MNCL) in two independent chronic oral studies using Fischer rats. Fischer rats (F–344) had an increased mortality due to MNCL in DINP-treated rats suggesting that DINP is associated with the elevated incidence, progression and severity of MNCL. The tumor findings may be biologically significant because the time to onset of tumor was shorter and the disease was more severe in treated animals compared to the timing of onset and severity in control animals. EPA believes that it is therefore highly unlikely that these findings were unrelated to treatment. EPA notes, however, that there are several sources of uncertainty in the interpretation of the MNCL data. These include a high and variable background rate of MNCL and a lack of information on the mode of action for induction of MNCL. Furthermore, some scientists believe that MNCL may be a rodent-specific cancer of unclear relevance to human cancer. EPA is reviewing the scientific question of the biological relevance of MNCL and human cancer. As a result of these scientific uncertainties, EPA reserves judgment on the human significance of MNCL and whether DINP can reasonably be anticipated to cause cancer in humans.

Because the data currently available do not permit a clear conclusion on the relevance of DINP-induced liver tumors and MNCL at this time, EPA reserves judgment on whether DINP can reasonably be anticipated to cause cancer in humans.

B. What Changes Have Been Made to the Discussion of Chronic Liver Toxicity?

In the original DINP hazard assessment (Ref. 1), EPA stated that DINP could reasonably be anticipated to cause chronic liver toxicity based on increased liver weight, increased liver enzyme activities, and chronic liver lesions based on two chronic studies in rats (Refs. 4 and 5). In the revised hazard assessment, the chronic liver lesion is described in more detail and identified as spongiosis hepatis. The incidence of spongiosis hepatis was dose-related and significantly elevated in male rats chronically treated with DINP in two independent studies conducted by different laboratories. The incidence of spongiosis hepatis was not elevated in female rats or in male or female mice. Based on the available data, the Agency has identified a No Observed Adverse Effect Level (NOAEL) of 15 milligrams per kilogram per day (mg/kg/day) and a Lowest Observed Adverse Effect Level (LOAEL) of 152 mg/kg/day for the Lington *et al.* study (Ref. 4) and a NOAEL of 88 mg/kg/day and a LOAEL of 359 mg/kg/day for the Moore study (Ref. 5), based on indications of serious liver damage (*i.e.*, a statistically significant increased incidence of spongiosis hepatis and increased liver weight and liver enzyme activities) in male rats chronically exposed to DINP for two years.

The Agency believes that the existing data support the conclusion that the increased incidence of spongiosis hepatis in dosed rats is clearly related to DINP treatment. In further evaluating the data for hepatic spongiosis, the Agency considered (1) The possibility that the occurrence of spongiosis hepatis and induction of peroxisome proliferation were related; (2) the possibility that the occurrence of spongiosis hepatis was a consequence of MNCL; (3) the relationship of spongiosis hepatis to hepatocellular cancer; and (4) the human relevance of hepatis spongiosis.

EPA believes that the occurrence of spongiosis hepatis is not likely to be related to the occurrence of peroxisome proliferation, hepatocellular cancer, or MNCL. Spongiosis hepatis is a lesion of the perisinusoidal cells of the liver (the Ito cells) whereas the carcinogenic and other toxic effects of DINP on the liver involved hepatocytes, which are the predominant cell type in the liver. This lesion has been found in livers of rodents and fish following exposure to substances (*e.g.*, nitrosamines) that induce cancer in these species (Ref. 8). Both chronic studies of DINP (Refs. 4 and 5) reported increased incidences of treated male rats with spongiosis hepatis. EPA has determined that spongiosis hepatis is relevant to human health and that determination is discussed in detail in the revised hazard assessment (Ref. 2, section II.E.1).

Spongiosis hepatis was also considered to be a significant finding by the authoritative scientific panel, the U.S. Consumer Product Safety Commission's (CPSC) Chronic Hazard Advisory Panel (CHAP) on DINP in their final report (Ref. 9). In addition to the CPSC panel report, a Histopathology Peer Review and Pathology Working Group convened by Experimental Pathology Laboratories (ELP) (Ref. 10) independently evaluated the liver slides from rats chronically treated with DINP and confirmed that the spongiosis hepatitis was increased in male rats of both chronic rodent studies. These findings support EPA's position that chronic liver toxicity is an endpoint of concern for DINP. At the time of the development of the original DINP hazard assessment, EPA had not

reviewed the ELP report (Ref. 10), or seen the CHAP report (Ref. 9) that discusses the ELP report conclusions because the final CHAP report was not available.

IV. How Does the Revised Hazard Assessment Impact EPA's Previous Conclusions About the Toxicity of DINP?

In the previous DINP hazard assessment (Ref. 1), carcinogenicity was determined to be a significant concern for DINP. However, based on the revised hazard assessment (Ref. 2), at this time EPA reserves judgment on whether cancer is an endpoint of concern for DINP. This conclusion may change in the future depending on how EPA resolves the issues concerning the human relevance of the types of tumors that DINP has been shown to cause. The revised hazard assessment also provides a more detailed and specific discussion about the ability of DINP to cause chronic liver toxicity than that contained in the previous hazard assessment. The revised discussion on chronic liver toxicity provides additional support to the previous conclusion that DINP causes chronic liver toxicity.

The revised DINP hazard assessment does not affect the previous conclusions from the original DINP hazard assessment that developmental and chronic kidney toxicity are endpoints of concern for DINP. In addition, nothing in the revised DINP hazard assessment affects EPA's previous conclusion that the addition of DINP as a category of chemicals, rather than individual chemical listings, would be appropriate.

V. What Type of Comments Is EPA Interested In Receiving?

EPA is requesting comments on all parts of the revised hazard assessment. However, EPA is specifically interested in comments on the sections of the revised hazard assessment that deal with carcinogenicity and chronic liver toxicity. EPA also requests commenters to provide any additional data or information on the human relevance of any of the adverse effects discussed in the revised hazard assessment.

VI. References

EPA has established an official public docket for this action under Docket ID No. TRI–2005–0004. The previous docket number for the proposed rule was OEI–100004 and was assigned prior to the development of EPA's electronic public docket and comment system. Therefore, EPA is creating a new electronic docket number for this action. All the materials submitted to docket number OEI–100004 have been transferred to docket number TRI–2005–0004. The public docket includes information considered by EPA in developing this proposed rule, 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. USEPA, OEI. 2000. Technical Review of Diisononyl Phthalate. Office of Environmental Information, Becki Madison, Christine Augustyniak, Ron Bloom, Candace Brassard, Ossi Meyn, Nicole Paquette, Pam Russell, and John Scalera.

2. USEPA, OEI. 2005. Revised Technical Review of Diisononyl Phthalate, March 4, 2005. Office of Environmental Information, Environmental Analysis Division, Analytical Support Branch. Amy Newman, Candace Brassard, Kathryn Montague, Nicole Paquette, Ph.D., and John Scalera.

3. USEPA, OEI. 2005. Response to External Peer Review Comments on the Revised Technical Review for Diisononyl Phthalate, April 2005.

4. Lington, A.W.; Bird M.G.; Plutnick, R.T.; Stubblefield, W.A.; and Scala, RA. 1997. Chronic toxicity and carcinogenic evaluation of diisononyl phthalate in rats. Fundam. Appl. Toxicol 36: 79–89.

5. Moore, M.R., 1998. Oncogenicity study in rats with di(isononyl)phthalate including ancillary hepatocellular proliferation and biochemical analyses. TSCATS Doc# 89980000308. Old Doc 8EHQ099813083. Fiche # OTS05562832. Submitted by Aristech Chemical Corporation. Produced by Covance Laboratories 2598–104.

6. Moore M.R., 1998. Oncogenicity study in mice with di(isononyl)phthalate including ancillary hepatocellular proliferation and biochemical analyses. TSCATS Doc# 89990000046. Old Doc 8EHQ119813083. Fiche # OTS05562833. Submitted by Aristech Chemical Corp. Produced by Covance 2598–105.

7. USEPA. 1999. Draft Revised Guidelines for Carcinogen Risk Assessment. NCEA-F–0644, July 1999.

8. Stoebel, P.; Mayer, F.; Zerban, H., Bannasch, P. 1995. Spongiotic pericytoma: a benign neoplasm deriving from the perisinusoidal cells in rat liver. Am. J. Pathol. 146:903–913.

9. Chronic Hazard Advisory Panel on Diisononyl Phthalate. Report to the U.S. Consumer Product Safety Commission. June 2001. U.S. Consumer Product Safety Commission, Bethesda, MD.

10. Experimental Pathology Laboratories, 1999. Histology peer review and pathology working group review of selected lesions of the liver and spleen in male and female F344 rats exposed to DINP. EPL Project number 303–013, Pathology Report.

List of Subjects in 40 CFR Part 372

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

Dated: June 7, 2005.

Kimberly T. Nelson,

Assistant Administrator for Office of Environmental Information and Chief Information Officer.

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