

b. How does a crew monitor and respond to changing weather conditions, including storms?

20. Fatigue: OSHA believes that fatigue can affect communication tower workers in several ways. Climbing a communication tower is physically demanding, and OSHA is concerned that fatigue due to exertion can be hazardous for tower workers. Accelerated work timelines can also result in tower workers working very long hours. And OSHA understands that communication tower workers may travel long distances to reach remote worksites, which can result in workers being fatigued before they even begin work.

a. What hazards are faced by a worker who finds it physically challenging to perform expected tasks, such as climbing a tower or performing a self-rescue? What impact can this have on other crew members?

b. What are the common causes of worker fatigue at communication tower worksites?

c. What are the effects of fatigue on tower worker safety, and what types of incidents occur as a result of worker fatigue?

21. Other common hazards:

a. What other hazards are present in communication tower work, and what types of incidents are resulting from those hazards? What can be done to protect employees from those hazards?

b. What are some health and safety considerations involved in working with communications equipment installed on non-dedicated tower structures, such as water towers, buildings, silos, electrical transmission towers, etc.?

### Contracting and Work Oversight

22. Describe your role in the contract chain and the key safety-related provisions typically included in your contracts. How do contracting parties oversee or enforce those provisions? What are the consequences if a party fails to fulfill those contractual requirements?

23. What characteristics of past safety performance does your company use in selecting potential contractors and subcontractors? What safety-related criteria does your company use in this selection process?

24. Are safety-related factors considered in determining whether to remove a contractor/subcontractor from an ongoing project or from future selection processes? If so, what specific factors are considered?

25. What are the ways in which the multi-leveled contracting environment (*i.e.*, where entities such as the carrier,

tower owner, turfing vendor, subcontractor, and contractors hired by the subcontractor all have some role in the project) impacts employee safety at communication tower worksites?

26. What practices might companies in the contracting chain adopt to encourage communication and coordination among employers at tower work sites? What obstacles stand in the way of communication and coordination between different parties in the contracting chain?

### Economic Issues

27. The Agency seeks information on the number and size of firms that are engaged in communication tower work and on the number of employees employed by those firms.

28. The Agency seeks information about wage and turnover rates for employees who work on communication towers. The Agency is also interested in information about the experience possessed by workers currently doing communication tower work. Are they usually experienced in this type of work? Are there many new or inexperienced employees working on communication towers?

29. What types of equipment are used in tower work and how often is this equipment repaired and/or replaced?

30. The Agency seeks information from all employers in the contracting chain about the extent to which employees directly engaged in tower work are covered by workers' compensation and/or an employer liability insurance policy.

### Tower Design

31. Can towers be designed and built with elevators for lifting personnel or materials? Can towers be built with booms or davits aloft to aid in hoisting materials?

32. How would elevators or davits affect productivity/efficiency, *e.g.*, the amount of time spent on the tower? How would elevators or davits address or cause any safety hazards at the site? For example, would elevators or davits address hazards related to employee fatigue?

33. What are the industry standards for providing fall protection anchor points on new towers?

### Regulatory/Non-Regulatory Approaches

34. What would be the advantages and disadvantages of an OSHA standard that covers both construction and maintenance activities on communication towers?

35. What effects have the North Carolina and Michigan regulatory

approaches had on work practices and climber safety in those states?

36. Should an OSHA standard be limited to work performed on communication towers, or should it also cover towers used for other purposes?

37. If OSHA does not initiate a dedicated rulemaking for work on communication towers, what other types of regulatory actions might be necessary and appropriate?

38. What non-regulatory approaches could OSHA take to address hazards faced by employees working on communication towers?

### Authority and Signature

This document was prepared under the direction of David Michaels, Ph.D., MPH, Assistant Secretary of Labor for Occupational Safety and Health, U.S. Department of Labor. It is issued pursuant to sections 3704 *et seq.*, Public Law 107-217, 116 STAT. 1062 (40 U.S.C. 3704 *et seq.*); sections 4, 6, and 8, Public Law 91-596, 84 STAT. 1590 (29 U.S.C. 653, 655, 657); 29 CFR part 1911; and Secretary of Labor's Order No. 1-2012 (77 FR 3912 (Jan. 25, 2012)).

Signed at Washington, DC, on March 27, 2015.

**David Michaels,**

*Assistant Secretary of Labor for Occupational Safety and Health.*

[FR Doc. 2015-08633 Filed 4-14-15; 8:45 am]

BILLING CODE 4510-26-P

## ENVIRONMENTAL PROTECTION AGENCY

### 40 CFR Part 372

[EPA-HQ-TRI-2015-0011; FRL-9925-29-OEI]

RIN 2025-AA41

### Addition of 1-Bromopropane; Community Right-To-Know Toxic Chemical Release Reporting

**AGENCY:** Environmental Protection Agency (EPA).

**ACTION:** Proposed rule.

**SUMMARY:** The Environmental Protection Agency (EPA) is proposing to add 1-bromopropane 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 (PPA) of 1990. 1-Bromopropane has been classified by the National Toxicology Program in their 13th Report on Carcinogens as "reasonably anticipated to be a human carcinogen." EPA believes that 1-bromopropane meets the EPCRA section

313(d)(2)(B) criteria because it can reasonably be anticipated to cause cancer in humans. Based on a review of the available production and use information, 1-bromopropane is 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 15, 2015.

**ADDRESSES:** Submit your comments, identified by Docket ID No. EPA-HQ-TRI-2015-0011, by one of the following methods:

- *www.regulations.gov:* Follow the on-line instructions for submitting comments.

- *Email:* [oei.docket@epa.gov](mailto: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-2015-0011. EPA's policy is that all comments received will be included in the public docket without change and may be made available online at [www.regulations.gov](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](http://www.regulations.gov) or email. The [www.regulations.gov](http://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 email comment directly to EPA without going through [www.regulations.gov](http://www.regulations.gov), your email 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](http://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](http://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; email: [bushman.daniel@epa.gov](mailto: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 (select menu option 3) or (703) 412-9810 in Virginia and Alaska or toll free, TDD (800) 553-7672, <http://www.epa.gov/superfund/contacts/infocenter/>.

**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 1-bromopropane. Potentially affected categories and entities may include, but are not limited to:

Category	Examples of potentially affected entities
Industry .....	<p>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*.</p> <p>*Exceptions and/or limitations exist for these NAICS codes.</p> <p>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) (corresponds 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.) (corresponds to SIC 4953, Refuse Systems).</p> <p>Federal facilities.</p>
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.

## II. Introduction

### A. What is the statutory authority for this proposed rule?

This rule is issued under EPCRA section 313(d) and section 328, 42 U.S.C. 11023 *et seq.* EPCRA is also referred to as Title III of the Superfund Amendments and Reauthorization Act of 1986.

### B. What is the background for this action?

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 308 individually listed 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)(A) through (C) are met. The EPCRA section 313(d)(2)(A) through (C) criteria are:

- 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.
- The chemical is known to cause or can reasonably be anticipated to cause in humans:
  - Cancer or teratogenic effects; or
  - Serious or irreversible—
    - Reproductive dysfunctions,
    - Neurological disorders,
    - Heritable genetic mutations; or
    - Other chronic health effects.
- The chemical is known to cause or can be reasonably anticipated to cause, because of:
  - Its toxicity;
  - Its toxicity and persistence in the environment; or
  - 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 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 13th RoC having been released on October 2, 2014 (79 FR 60169, October 6, 2014). The 13th RoC contains the NTP cancer classifications from the most recent chemical evaluations, as well as the classifications from previous versions of the RoC (Reference (Ref.) 1).

### 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. 2) 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, which can be useful for evaluating whether a relevant cancer mechanism is operating in humans.

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. 3).

#### C. What is the review process for the RoC?

Specific details of the nomination and review process for the development of the 13th RoC are described in the Process for Preparation of the Report on Carcinogens section of the 13th RoC (Ref. 4). 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 13th RoC, during the entire nomination, selection, and review process there were seven opportunities for public comment. For each candidate substance, an expert panel was convened to peer review the NTP monograph document prepared for each candidate substance. The RoC Monograph on 1-Bromopropane consists of the following components: (Part 1) the cancer evaluation component that reviews the relevant scientific information, assesses its quality, applies the RoC listing criteria to the scientific information, and gives the RoC listing status for 1-bromopropane, and (Part 2) the RoC monograph’s substance profile containing the NTP’s listing status decision, a summary of the scientific evidence considered key to reaching that decision, and data on properties, use, production, exposure, and Federal regulations and guidelines to reduce exposure to 1-bromopropane. The expert panel members had the following major responsibilities in reviewing the draft RoC monograph:

(1) to comment on the draft cancer evaluation components for 1-bromopropane,

specifically, whether they are technically correct and clearly stated, whether the NTP has objectively presented and assessed the scientific evidence, and whether the scientific evidence is adequate for applying the RoC listing criteria, and (2) to comment on the draft substance profile for 1-bromopropane, specifically, whether the scientific justification presented in the substance profile supports the NTP’s preliminary policy decision on the RoC listing status of 1-bromopropane. The panel was also asked to vote on the following questions: (1) Whether the scientific evidence supports the NTP’s conclusion on the level of evidence for carcinogenicity from experimental animal studies on 1-bromopropane and (2) whether the scientific evidence supports the NTP’s preliminary listing decision for 1-bromopropane in the RoC. The panel agreed with the NTP conclusions that 1-bromopropane should be listed in the RoC based on sufficient evidence of carcinogenicity from studies in experimental animals, which found skin tumors in male rats, large intestine tumors in female and male rats, and lung tumors in female mice.” (Ref. 5)

Based upon the peer-review comments, the Office of the Report on Carcinogens (ORoC) prepared a revised draft RoC Monograph, which was then reviewed by the NTP Board of Scientific Counselors. The ORoC, in concert with the NTP Director, then finalized the RoC monographs and submitted the newly reviewed substances to the NTP Executive Committee for consultation. The final draft of the 13th RoC was then submitted to the Secretary of Health and Human Services (HHS) for review and approval. Once approved, the Secretary submitted the 13th RoC to the U.S. Congress as a final document and published the document on the RoC Web site (<http://ntp.niehs.nih.gov/pubhealth/roc/roc13/index.html>).

#### IV. EPA’s Review of the 13th RoC

##### A. How did EPA select the NTP RoC chemical 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 12th RoC (March 13, 2013, 78 FR 15913). Each new version of the RoC adds newly classified chemicals to the existing list. EPA’s present review of the 13th RoC identified 1-bromopropane as the only newly listed chemical that is not on the EPCRA section 313 list.

EPA reviewed the NTP 13th RoC chemical profile and supporting materials for 1-bromopropane (Ref. 6). 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’s review of the 1-bromopropane chemical profile and supporting material found no inconsistencies between how the data were interpreted by the NTP and how that same data would be interpreted under EPA’s Guidelines for Carcinogen Risk Assessment (Ref. 3). Therefore, EPA agrees with the hazard conclusions of the NTP 13th RoC for 1-bromopropane.

##### B. What technical data supports the NTP RoC classification and EPA’s proposed addition of 1-bromopropane to the EPCRA section 313 list?

This section presents the data that supported the NTP 13th RoC classification of 1-bromopropane and why EPA believes the data support the addition of this chemical to the EPCRA section 313 list. The RoC 1-Bromopropane Profile document (Ref. 7), the RoC Monograph on 1-Bromopropane (Ref. 5), and the available references cited within the portion of the 13th RoC chemical profile 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 quotation cited below from the 13th RoC 1-Bromopropane Profile document 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 quotation can be found in the NTP 13th RoC 1-Bromopropane Profile document (Ref. 7).

1. *1-Bromopropane* (Chemical Abstracts Service Registry Number 106–94–5) (Refs. RoC Monograph and Profile documents (Refs. 5 and 7)). The NTP has classified 1-bromopropane as “reasonably anticipated to be a human carcinogen.” The classification is based on sufficient evidence of carcinogenicity in experimental animals and supporting data on mechanisms of carcinogenesis. The RoC substance profile for 1-bromopropane (Ref. 7) included the following summary information of the evidence of carcinogenicity:

##### Carcinogenicity

1-Bromopropane is *reasonably anticipated to be a human carcinogen* based on sufficient evidence of carcinogenicity from studies in experimental animals. 1-Bromopropane, either directly or via reactive metabolites, causes molecular alterations that typically are associated with carcinogenesis, including genotoxicity, oxidative stress, and glutathione depletion. These alterations, observed mainly *in vitro* and in toxicity studies in rodents, are relevant to possible mechanisms of human carcinogenicity and support the relevance of the cancer studies in experimental animals to human carcinogenicity.

### Cancer Studies in Experimental Animals

Inhalation exposure to 1-bromopropane caused tumors in two rodent species and at several different tissue sites, including one tissue site in rats at which tumors are rare (NTP 2011).

In male rats, 1-bromopropane caused significant dose-related increases in the incidences of several types of benign and/or malignant skin tumors (keratoacanthoma; keratoacanthoma and squamous-cell carcinoma combined; and keratoacanthoma, squamous-cell carcinoma, basal-cell adenoma, and basal-cell carcinoma combined). Both female and male rats showed an increased incidence of large-intestine tumors (adenoma of the colon and rectum), which are rare tumors in rats. In females, the incidence was dose-related and statistically significantly higher than in concurrent controls, and it exceeded the historical control range for all routes of exposure used in studies, including inhalation exposure. In males, the incidence of large-intestine adenoma was not significantly increased, but exceeded the historical control range for inhalation-exposure studies, and its occurrence was considered to be biologically significant because of the rarity of these tumors (which occurred in less than 0.2% of the historical controls). Although no carcinoma of the large intestine was observed in male or female rats in this study, adenoma of the large intestine has been shown to progress to carcinoma in other studies, and forms a morphologic continuum with carcinoma (Deschner 1983, Chang 1984, Nigro 1985).

In female mice, 1-bromopropane caused significant dose-related increases in the incidence of benign and malignant lung tumors combined (alveolar/bronchiolar adenoma and carcinoma).

These findings are supported by the observation of additional tumors in rats that may have been related to 1-bromopropane exposure, including malignant mesothelioma of the abdominal cavity and pancreatic islet tumors in males and skin tumors (squamous-cell papilloma, keratoacanthoma, and basal-cell adenoma or carcinoma) in females.

### Other Relevant Data

1-Bromopropane is well absorbed following ingestion, inhalation, or dermal exposure. Occupational exposure occurs primarily by inhalation and dermal contact. Unmetabolized 1-bromopropane has been detected in the urine of exposed workers at levels significantly correlated with exposure to 1-bromopropane in air (Kawai *et al.* 2001, Ichihara *et al.* 2004).

1-Bromopropane is metabolized via several pathways; 16 urinary metabolites have been detected in rodents, and several other metabolites have been proposed (Jones and Walsh 1979, Ishidao *et al.* 2002, Garner *et al.* 2006). The primary metabolic pathways in rodents are oxidation reactions catalyzed by cytochrome P450 (primarily CYP2E1) and glutathione conjugation. The available data on human metabolism of 1-bromopropane, although limited, suggest that some of its metabolic pathways in humans are similar to those observed in rodents. Four mercapturic conjugates identified in the urine of rodents

were also identified in the urine of workers exposed to 1-bromopropane (Hanley *et al.* 2009). The major metabolite, *N*-acetyl-S-(*n*-propyl)-L-cysteine, has been detected in the urine of exposed workers at levels that increased with increasing levels of 1-bromopropane in ambient air (Hanley and Dunn 2006, Valentine *et al.* 2007, Hanley *et al.* 2009, 2010). This metabolite is produced in humans by conjugation of 1-bromopropane with glutathione, and that reaction also releases free bromide ions, another useful biomarker for human exposure to 1-bromopropane (Jones and Walsh 1979, Hanley *et al.* 2006). No studies were identified that tested for the occurrence in humans of the oxidative metabolites that are obligate intermediates to the measured conjugates.

### Studies on Mechanisms of Carcinogenesis

The mechanism(s) by which 1-bromopropane causes cancer is not known. However, exposure to 1-bromopropane has been shown to cause molecular alterations related to carcinogenicity, including genotoxicity (mutations and DNA damage), oxidative stress, glutathione depletion, and immunomodulation.

Studies have shown that 1-bromopropane can bind to macromolecules; it formed S-propylcysteine-globin adducts in exposed animals and humans (Valentine *et al.* 2007). Although 1-bromopropane did not induce mutations in bacteria under standard assay conditions, it did induce mutations in bacteria both with and without exogenous mammalian metabolic activation in the only reported study whose design was appropriate for testing a highly volatile chemical (Barber *et al.* 1981). It also caused mutations in cultured mammalian cells with or without mammalian metabolic activation (Elf Atochem 1996, as reviewed in NTP 2003) and DNA damage in cultured human cells without metabolic activation (Toraason *et al.* 2006). In addition, there is limited evidence of DNA damage in leukocytes from 1-bromopropane-exposed workers (Toraason *et al.* 2006). In rodents exposed *in vivo*, 1-bromopropane did not increase micronucleus formation in bone marrow (Kim *et al.* 1998, as reviewed in NTP 2003) or peripheral blood erythrocytes (Elf Atochem 1996, cited in NTP 2003, NTP 2011) or cause dominant lethal mutations. However, the dominant lethal mutation assay is generally regarded as relatively insensitive for the detection of mutagenic agents (Saito-Suzuki *et al.* 1982, Yu *et al.* 2008).

There is evidence that metabolic activation plays a role in the genotoxicity and toxicity of 1-bromopropane. Several reactive metabolites (or intermediates) of 1-bromopropane have been identified in rodents, including glycidol and  $\alpha$ -bromohydrin, and propylene oxide has been proposed as a metabolite (Garner *et al.* 2006). These compounds cause genotoxic effects *in vitro*, including DNA adduct formation, mutations, and DNA or chromosome damage (Stolzenberg and Hine 1979, IARC 1994, 2000). Glycidol and propylene oxide cause cytogenetic effects *in vivo* and are carcinogenic in experimental animals, and both substances are listed in the Report on

Carcinogens as *reasonably anticipated to be human carcinogens*. These reactive and genotoxic metabolites may be responsible for at least some of the carcinogenic effects of 1-bromopropane. As with 1-bromopropane, oral exposure to glycidol caused rare tumors of the large intestine in rats, as did oral exposure to two halogenated alkane analogues of 1-bromopropane, tribromomethane and bromodichloromethane (NTP 1987, 1989, 1990).

Chronic exposure to 1-bromopropane may produce levels of oxidative metabolites that exceed the glutathione-conjugating capacity or may inhibit enzymes required for glutathione synthesis. Because glutathione is an important cellular defense mechanism, reduced levels can lead to oxidative stress, increased toxicity, and carcinogenicity. Numerous studies have shown that 1-bromopropane induces both oxidative stress and glutathione depletion (Lee *et al.* 2005, 2007, 2010a, Liu *et al.* 2009, 2010, Huang *et al.* 2011). Studies with Cyp2e1<sup>-/-</sup> knockout mice, cytochrome P450 inhibitors, or a glutathione synthesis inhibitor showed that this metabolic activation pathway is involved in 1-bromopropane-induced toxicity, including neurological and reproductive effects, hepatotoxicity, and immunosuppression (NTP 2003, 2011, Lee *et al.* 2007, 2010a,b). Neurological effects of 1-bromopropane exposure have also been reported in humans (Li *et al.* 2010, Ichihara *et al.* 2012).

It is unclear whether induction of immunotoxicity by 1-bromopropane plays a role in tumor development. Recent studies have shown that 1-bromopropane causes immunosuppression in rodents (Lee *et al.* 2007, Anderson *et al.* 2010). In particular, it reduced the numbers of T cells and T-cell subpopulations. In addition, there is evidence that 1-bromopropane causes an inflammatory response. It induced dose-related increases in gene expression and production of proinflammatory cytokines in mouse macrophages (Han *et al.* 2008) and an inflammatory response in rats (NTP 2011). However, chronic respiratory inflammation and lung tumors were not associated in rodents; respiratory inflammation occurred in rats but not mice, whereas lung tumors occurred in mice but not rats.

### Cancer Studies in Humans

No epidemiological studies or case reports were identified that evaluated the relationship between human cancer and exposure specifically to 1-bromopropane.

EPA has reviewed the NTP assessment for 1-bromopropane and agrees that 1-bromopropane can reasonably be anticipated to cause cancer in humans. EPA believes that the evidence is sufficient for listing 1-bromopropane 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 (see Unit III). Based on EPA's review of the data contained in the NTP 13th RoC, EPA has determined that 1-bromopropane can reasonably be anticipated to cause cancer (Ref. 6). Therefore, EPA believes that the evidence is sufficient for listing 1-bromopropane on the EPCRA section 313 toxic chemical list pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data presented in the NTP 13th 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 1-bromopropane meets 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-2015-0011. 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. For convenience, the docket also includes all of the **Federal Register** documents cited in this action.

1. NTP, 2014. National Toxicology Program. Report on Carcinogens, Thirteenth Edition. Released October 2, 2014. U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program, Research Triangle Park, NC 27709. (<http://ntp.niehs.nih.gov/pubhealth/roc/roc13/index.html>)
2. NTP, 2014. National Toxicology Program. Report on Carcinogens, Thirteenth Edition, Introduction section. Released October 2, 2014. U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program, Research Triangle Park, NC 27709.
3. USEPA. Guidelines for Carcinogen Risk Assessment. Risk Assessment Forum, U.S. Environmental Protection Agency, Washington, DC, March 2005.
4. NTP, 2014. National Toxicology Program. Report on Carcinogens, Thirteenth Edition, Process for Preparation of the Report on Carcinogens section. Released October 2, 2014. U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program, Research Triangle Park, NC 27709.
5. NTP, 2013. Report on Carcinogens Monograph on 1-Bromopropane. Office of the Report on Carcinogens, Division of the National Toxicology Program, National Institute of Environmental Health Sciences, U.S. Department of Health and Human Services. NIH Publication No. 13-5982, September 25, 2013
6. USEPA, OEI. Memorandum from Jocelyn Hospital, Toxicologist, Analytical Support Branch to Sandra Gaona, Acting Chief, Analytical Support Branch. November 3, 2014. Subject: Review of National Toxicology Program (NTP) Cancer Classification Data for 1-bromopropane.
7. NTP, 2014. National Toxicology Program. Report on Carcinogens, Thirteenth Edition, Profile for 1-Bromopropane. Released October 2, 2014. U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program, Research Triangle Park, NC 27709.
8. USEPA, OEI. Economic Analysis of the Proposed Rule to add 1-Bromopropane to the EPCRA Section 313 List of Toxic Chemicals. February 17, 2015.

## VII. What are the Statutory and Executive Order reviews associated with this action?

*A. Executive Order 12866: Regulatory Planning and Review and Executive Order 13563: Improving Regulation and Regulatory Review*

This action is not a significant regulatory action and was therefore not submitted to the Office of Management and Budget (OMB) for review.

### *B. Paperwork Reduction Act*

This action 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). OMB has previously approved the information collection activities contained in the existing regulations and has assigned OMB control numbers 2025-0009 and 2050-0078. 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 Forms A and R, supplier notification, and petitions under OMB Control number 2025-0009 (EPA Information Collection Request (ICR) No. 1363) and those related to trade secret designations under OMB Control 2050-0078 (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).

*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.*

I certify that this action will not have a significant economic impact on a substantial number of small entities under the RFA. The Agency has determined that of the 140 entities estimated to be impacted by this action, 136 are small businesses; no small

governments or small organizations are expected to be affected by this action. All 136 small businesses affected by this action are estimated to incur annualized cost impacts of less than 1%. 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 action 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. 8).

*D. Unfunded Mandates Reform Act*

This action does not contain an unfunded mandate of \$100 million or more as described in UMRA, 2 U.S.C. 1531 through 1538, and does not significantly or uniquely affect small governments. This action is not subject to the requirements 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. EPA's economic analysis indicates that the total cost of this action is estimated to be \$531,002 in the first year of reporting (Ref. 8).

*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.

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

This action does not have tribal implications, as specified in Executive Order 13175. 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.

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

The EPA interprets Executive Order 13045 as applying only to those regulatory actions that concern environmental health or safety risks that the EPA has reason to believe may disproportionately affect children, per the definition of "covered regulatory action" in section 2-202 of the Executive Order. This action is not

subject to Executive Order 13045 because it does not concern an environmental health risk or safety risk.

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

This action is not subject to Executive Order 13211, because it is not a significant regulatory action under Executive Order 12866.

*I. National Technology Transfer and Advancement Act*

This rulemaking does not involve technical standards.

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

The EPA believes the human health or environmental risk addressed by this action will not have potential disproportionately high and adverse human health or environmental effects on minority, low-income or indigenous populations. The results of this evaluation are contained below.

This action does not address any human health or environmental risks and does not affect the level of protection provided to human health or the environment. This action adds an additional chemical to the EPCRA section 313 reporting requirements. By adding a chemical 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 action 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: April 8, 2015.  
**Gina McCarthy,**  
*Administrator.*

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

**PART 372—TOXIC CHEMICAL RELEASE REPORTING: COMMUNITY RIGHT-TO-KNOW**

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

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

■ 2. In § 372.65, paragraph (a) is amended by adding in the table the entry for "1-Bromopropane" in alphabetical order and in paragraph (b) by adding in the table the entry for "106-94-5" in numerical order to read as follows:

**§ 372.65 Chemicals and chemical categories to which this part applies.**

\* \* \* \* \*  
 (a) \* \* \*

Chemical name	CAS No.	Effective date
* * * * *		
1-Bromopropane .....	106-94-5	1/1/16
* * * * *		

(b) \* \* \*

CAS No.	Chemical name	Effective date
* * * * *		
106-94-5 ..	1-Bromopropane	1/1/16
* * * * *		

\* \* \* \* \*  
 [FR Doc. 2015-08664 Filed 4-14-15; 8:45 am]  
**BILLING CODE 6560-50-P**

**FEDERAL COMMUNICATIONS COMMISSION**

**47 CFR Part 73**

[MB Docket No. 15-88, RM-11747; DA 15-444]

**Television Broadcasting Services; Bend, Oregon**

**AGENCY:** Federal Communications Commission.

**ACTION:** Proposed rule.

**SUMMARY:** The Commission has before it a petition for rulemaking filed by TDS Broadcasting LLC ("TDS"), the licensee of KOHD, channel 51, Bend, Oregon, requesting the substitution of channel