three additional sanitized copies must also be submitted. Nonconfidential versions of comments will be placed in the record for this action and will be available for public inspection. Comments should include the docket control number for the ANPR, OPPTS-400106 and the EPA contact. Unit II. of this document contains additional information on submitting comments containing information claimed as CBI.

Comments and data may also be submitted electronically by sending electronic mail (e-mail) to: oppt.ncic@epamail.epa.gov. 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 disks in WordPerfect 5.1 file format or ASCII file format. All comments and data in electronic form must be identified by the docket number OPPTS–400106. No CBI should be submitted through e-mail. Electronic comments on this ANPR may be filed online at many Federal Depository Libraries. Additional information on electronic submissions can be found in Unit IV. of this document.

FOR FURTHER INFORMATION CONTACT: Matt Gillen at 202-260-1801, e-mail: gillen.matthew@epamail.epa.gov for specific information regarding this Notice. For further information on EPCRA section 313 contact the Emergency Planning and Community Right-to-Know Hotline, Environmental Protection Agency, Mail Stop 5101, 401 M St., SW., Washington, DC 20460. Toll free: 1-800-535-0202, in Virginia and Alaska: 703-412-9877 or Toll free TDD: 800-553-7672.

SUPPLEMENTARY INFORMATION: Electronic Availability: An electronic copy of the documents listed in Unit I of this document are available from the EPA Public Access gopher (gopher.epa.gov) at the Environmental SubSet entry under ''Rules and Regulations.''

I. Introduction

In 1986, Congress enacted the Emergency Planning and Community Right-to-Know Act (EPCRA). Section 313 of EPCRA requires certain businesses to submit reports each year on the amounts of toxic chemicals their facilities release into the environment or otherwise manage. The information is placed in a publicly accessible data base known as the Toxics Release Inventory (TRI). The purpose of this requirement is to inform the public, government officials, and industry about the chemical management practices of specified toxic chemicals.

EPA is interested in expanding the information available via TRI to include chemical use information such as materials accounting data. The Agency began reviewing this issue in 1993 and held public meetings in 1994 and 1995. On August 8, 1995, President Clinton directed EPA to develop and implement, on an expedited schedule, a process for consideration of reporting use information under TRI. In response, EPA has begun the regulatory development process for additional review of chemical use reporting, which the Agency believes may provide a more detailed and comprehensive picture to the public about environmental performance and about toxic chemicals in their communities. EPA published the ANPR on October 1, 1996 (61 FR 51322) (FRL–5387–6), to give notice of EPA's consideration of this issue and to solicit comments on all aspects of chemical use and the collection of chemical use data. At the same time, the Agency also released ''Issues Paper No. 3'' which describes previous stakeholder comments on chemical use reporting. EPA also held three public meetings in October and December of 1996 to provide public forums for interested parties to provide input on the issues raised by the ANPR. This issues paper and ANPR can be obtained from the EPCRA hotline at the telephone numbers listed in the FOR FURTHER INFORMATION CONTACT unit of this document, or electronically via the EPA's TRI Homepage at: http://www.epa.gov/opptintr/tri.

The original comment period for the ANPR was due to expire on December 30, 1996. However, on November 26, 1996, the Department of Energy submitted a request for an extension of the comment period to allow time to gather and consolidate comments from various DOE facilities, and to account for DOE's need to comment on another EPA reporting initiative during the same period. EPA has decided to grant this request and to extend the comment period for an additional 60 days, or until February 28, 1997.

II. Rulemaking Record and Electronic Filing of Comments

A record has been established for the ANPR under docket number ''OPPTS-400106'' (including comments and data submitted electronically as described below). A public version of this record, including printed paper versions of electronic comments, which does not include any information claimed as CBI, is available for inspection from noon to 4 p.m., Monday through Friday, excluding legal holidays. The public record is located in the TSCA Nonconfidential Information Center, Room NE-B607, 401 M St., SW., Washington, DC 20460.

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

Electronic comments can be sent directly to EPA at oppt.ncic@epamail.epa.gov. Electronic comments must be submitted as an ASCII file avoiding the use of special characters and any form of encryption. The official record for this action, as well as the public version, as described above will be kept in paper form. Accordingly, EPA will transfer all comments received electronically into printed paper form as they are received and will place the paper copies in the official record which will also include all comments submitted directly in writing. The official record is the paper record maintained at the address in ADDRESSES at the beginning of this document.

List of Subjects in 40 CFR Part 372

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

Dated: December 23, 1996.

Lynn R. Goldman,

Assistant Administrator for Prevention, Pesticides and Toxic Substances.

[FR Doc. 97–57 Filed 1–2–97; 8:45 am]

BILLING CODE 6560–50–F

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40 CFR Part 372

[OPPTS–400107; FRL–5581–1]

RIN 2070–AC00

Barium Compounds; Toxic Chemical Release Reporting; Community Right-to-Know

AGENCY: Environmental Protection Agency (EPA).

ACTION: Denial of petition.

SUMMARY: EPA is denying a petition to remove the barium compounds category from the list of chemicals subject to the reporting requirements under section 313 of the Emergency Planning and

Community Right-to-Know Act of 1986 (EPCRA) and section 6607 of the Pollution Prevention Act of 1990 (PPA). This action is based on EPA's conclusion that barium compounds do not meet the deletion criterion of EPCRA section 313(d)(3). Specifically, EPA is denying this petition because EPA's review of the petition and available information resulted in the conclusion that barium ion (Ba^{+2}) can become available from the barium compounds subject to reporting and that barium ion can reasonably be anticipated to cause chronic toxicity. Therefore, barium compounds meet the criteria for inclusion on the list of chemicals subject to reporting under section 313 of EPCRA.

FOR FURTHER INFORMATION CONTACT: Daniel R. Bushman, Acting Petitions Coordinator, 202-260-3882 or e-mail: bushman.daniel@epamail.epa.gov, for specific information regarding this document. For further information on EPCRA section 313, contact the Emergency Planning and Community Right-to-Know Information Hotline, Environmental Protection Agency, Mail Stop 5101, 401 M St., SW., 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. Introduction

A. Statutory Authority

This action is taken under sections 313(d) and (e)(1) of the Emergency Planning and Community Right-to-Know Act of 1986 (EPCRA), 42 U.S.C. 11023. EPCRA is also referred to as Title III of the Superfund Amendments and Reauthorization Act of 1986 (SARA) (Pub. L. 99-499).

B. Background

Section 313 of EPCRA requires certain facilities manufacturing, processing, or otherwise using listed toxic chemicals to report their environmental releases of such chemicals annually. Beginning with the 1991 reporting year, such facilities also must report pollution prevention and recycling data for such chemicals, pursuant to section 6607 of the Pollution Prevention Act of 1990 (PPA), 42 U.S.C. 13106. Section 313 established an initial list of toxic chemicals that was comprised of more than 300 chemicals and 20 chemical categories. Barium-containing substances were included on the initial list, under the chemical category entitled ''barium compounds.'' Section 313(d) authorizes EPA to add or delete chemicals from the list, and sets forth criteria for these actions. EPA has added and deleted chemicals from the original statutory list. Under section 313(e)(1), any person may petition EPA to add chemicals to or delete chemicals from the 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 is denied.

EPCRA section 313(d)(2) states that a chemical may be listed if any of the listing criteria are met. Therefore, in order to add a chemical, EPA must demonstrate that at least one criterion is met, but does not need to examine whether all other criteria are also met. Conversely, in order to remove a chemical from the list, EPA must demonstrate that none of the criteria are met.

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 compound categories. EPA has also published a statement clarifying its interpretation of the section 313(d)(2) criteria for adding and deleting chemical substances from the section 313 list (59 FR 61439, November 30, 1994) (FRL-4922-2).

II. Description of Petition and Regulatory Status of Barium and Barium Compounds

Barium-containing substances are on the list of toxic chemicals subject to the annual reporting requirements of EPCRA section 313 and PPA section 6607. Barium-containing substances comprise the ''barium compounds'' category on the EPCRA section 313 list of toxic chemicals. The presence of barium in a compound defines its inclusion in the barium compounds category. As with all the metal compound categories on the EPCRA section 313 list of toxic chemicals, the basis for inclusion of the individual metal-containing substances within these categories is the toxicity which may be exhibited by the intact substance, or by the metal or metal ion which may be liberated from the intact substance within an organism, by biological fluids, or in the environment. EPA published a detailed discussion on the Agency's policies related to the metal compound categories on the EPCRA section 313 list of toxic chemicals in the Federal Register of May 23, 1991 (56 FR 23703).

EPA recently deleted barium sulfate (also known as barite) from the barium compounds category (59 FR 33205, June 28, 1994) (FRL–4767–5). EPA concluded that barium sulfate does not meet the toxicity criteria of EPCRA sections 313(d)(2)(A), (B) or (C), and that barium ion is available from barium sulfate only under low sulfate, anaerobic conditions in stagnant water bodies that are cut-off from surface and ground waters (i.e., conditions that cannot reasonably be anticipated to cause ecotoxicity or lead to human exposure to the ion). EPA believes that the low toxicity of barium sulfate can be mainly ascribed to the very low water solubility (2.4 milligrams per liter (mg/L) at 25 °C) of barium sulfate, barium ion's strong affinity for sulfate, and correspondingly, the low availability of barium ion.

Barium is regulated under the Safe Drinking Water Act, (42 U.S.C. 300f-300j-26); the current maximum contaminant level (MCL) is 2 mg/L (2 parts per million (ppm)) (40 CFR 141.62(b)(3)).

On June 28, 1996, EPA received a petition from the Chemical Products Corporation (CPC) to delete the entire barium compounds category from the EPCRA section 313 list of toxic chemicals. With this action, CPC petitioned EPA to delete all barium compounds from the list of toxic chemicals subject to the annual reporting requirements of EPCRA section 313 and PPA section 6607. In the petition, data are presented from various toxicity studies on a limited number of barium compounds. The petitioner contends that all barium compounds should be deleted because the available toxicity data show that barium ion does not meet the criteria for inclusion on the list of EPCRA section 313 chemicals. The petitioner also asserts that under environmental conditions barium ion is largely unavailable from barium compounds because of the presence of sulfate ion in the environment; sulfate ion will react quickly with barium ion to form barium sulfate.

III. EPA's Technical Review of Barium Compounds

The technical review of the petition to delete barium compounds from the reporting requirements of EPCRA section 313 and PPA section 6607 included an analysis of the chemistry, health effects, ecological effects, and environmental fate data available for barium compounds.

A. Chemistry and Use

Barium is a metallic substance that occurs in nature as its divalent cation

(ion), Ba^{+2}. Barium compounds are those substances that contain barium as part of their molecular formula. EPA has published a discussion on the chemistry of barium ion (Ref. 1). Barium ion is highly electropositive, and reacts readily with anions (sulfate (SO_4^{-2}), chloride (Cl^{-1}), carbonate (CO_3^{-2}), nitrate (NO_3^{-2}), etc.) to form the corresponding barium salt. The water solubility of the salt and, therewith, its ability to dissociate to barium ion is largely dependent on the affinity between barium ion and the anion. Barium chloride is highly water soluble (317 grams per liter (g/L)), whereas barium carbonate and barium sulfate are considerably less soluble, having water solubilities of 24 mg/L and 2.4 mg/L, respectively (Ref. 2). Barium carbonate is soluble in diluted solutions of hydrochloric, nitric or acetic acid. These acids react with barium carbonate to form barium chloride, barium nitrate, and barium acetate, respectively, which are all freely soluble in water (Ref. 2).

Another important factor controlling the availability of barium ion from a barium compound is the presence of sulfate ion. In waters, the availability of barium ion from a barium compound is governed largely by the concentration of sulfate ion present in solution. The availability of barium ion is inversely related to the concentration of sulfate; barium ion availability is suppressed in the presence of sulfate, and enhanced when sulfate concentration is low. This is because sulfate has a high affinity for barium ion and will form barium sulfate which precipitates out of solution (Ref 1). A more detailed discussion of factors that control barium ion availability in waters is provided below in Unit III.C. of this notice ''Environmental Fate of Barium Compounds.''

The most common natural form of barium is barium sulfate (barite). The greater natural occurrence of barium sulfate with respect to other barium salts is likely to be due to the relatively stronger affinity between Ba^{+2} and SO_4^{-2}, when compared to the affinity between Ba^{+2} and other naturally occurring anions.

Barium carbonate is another naturally occurring barium compound. It is also produced commercially from barium sulfate. Barium carbonate is often added to brick and clay products to precipitate sulfates. Barium carbonate is used also in the production of ceramic materials and glass products, and to produce other barium compounds. Barium compounds produced from barium carbonate include: barium acetate; barium bromide; barium chloride; barium 2-ethylhexanoate; barium hydroxide; barium hydrosulfide; barium iodide; barium metaborate; barium nitrate; barium nitrite; barium oxide; barium peroxide; barium sodium niobium oxide; barium sulfide; barium titanate; and higher purity grades of barium sulfate (Ref. 3). The uses of most of these barium compounds are summarized in Ref. 3.

B. Toxicological Evaluation

EPA's toxicological evaluation of barium compounds consisted of an analysis of health and environmental data pertaining to barium-containing substances included on the EPCRA section list of toxic chemicals as part of the barium compounds category. Data were obtained from: studies found in the literature (Refs. 4-12); the Hazardous Substances Data Bank (Ref. 13); EPA's Integrated Risk Information System (IRIS) (Ref. 14); a previous Federal Register Notice on barium sulfate (Ref. 15); a 1992 report published by the U.S. Department of Health and Human Services' Agency for Toxic Substances and Disease Registry entitled *Toxicological Profile for Barium* (Ref. 16); a 1993 report published by the U.S. Department of Health and Human Services' National Toxicology Program entitled *Toxicology and Carcinogenesis Studies of Barium Chloride Dihydrate in F4344 Rats and B6C3F1 Mice* (Ref. 17); and a 1990 EPA document entitled *The Drinking Water Criteria Document for Barium* (Ref. 18). The health and environmental portions of these reference sources are summarized below. Detailed discussions can be found in the publications and in the technical reports (Refs. 19-22) prepared by the EPA scientists who reviewed the publications. EPA's toxicological evaluation of barium compounds also included a review of the analysis of health and environmental data stated in the petition and the petitioner's interpretation of such data.

1. *Acute mammalian toxicity*. In humans, symptoms of acute barium toxicity after accidental or intentional oral ingestion of 1-15 grams of soluble barium salts include: muscular paralysis; respiratory failure; arterial hypertension; cardiac arrhythmias; profound hypokalemia and death (Refs. 5 and 18). The threshold of a toxic oral dose in adults has been estimated to be 200-500 milligrams (mg) or 2.86 - 7.14 milligrams per kilogram (mg/kg) of body weight. This quantity applies to the equivalent weight of the barium ion absorbed from the gut from the barium compound. The digestive system is extremely permeable to the barium ion. Acute lethal oral doses for barium in adults have been estimated to be 3-4 grams (calculated 43 - 57 mg/kg) (Refs. 13 and 18). Animal studies support similar cardiotoxic effects following acute exposure.

Ogen, et al. summarized the results of two large outbreaks of food poisoning that occurred following consumption of sausage that contained barium carbonate which was accidentally substituted for potato starch during sausage preparation (Ref. 12). The authors estimate that the amount of barium carbonate ingested in most of the affected individuals was 2-3 grams per person. The characteristic symptoms occurred within 8 hours after ingestion of the contaminated sausage, and included: vomiting, diarrhea, general weakness, paresthesia, difficulty in breathing, and, in the more severe cases, paralysis of the limbs and respiratory muscles. Most of the 144 affected individuals received treatment and recovered within a few days, however, 19 individuals required hospitalization, and one patient died. The authors of the study attribute the observed toxicity of barium carbonate to its reaction with hydrochloric acid in the stomach to yield barium chloride, which dissociates readily to barium ion and is absorbed systemically. These authors cite other studies involving food poisoning from barium carbonate.

The acute oral lethality of barium in animals has been well documented. There is a wide variability in the lethal dose of barium among species and age, as well as between strains of the same species. Nevertheless, the acute lethality of various barium salts is a function of their solubility in water or acid. In rats, acute oral toxicities of barium chloride, fluoride, nitrate and acetate have median lethal dose (LD_{50}) values of 118, 250, 355 and 921 mg barium/kg, respectively (Refs. 4, 13, and 17).

2. *Subchronic and chronic mammalian toxicity*. EPA's review of the available toxicity data for barium compounds identified kidney toxicity as the toxicological endpoint of concern. There are also varying reports on cardiovascular effects in humans and test animals from subchronic and chronic exposure to barium.

The U.S. Department of Health and Human Services' National Toxicology Program (NTP) conducted toxicology and carcinogenicity studies in F344/N rats and B6C3F1 mice by administering barium chloride dihydrate (99 percent pure) in drinking water for 15 days, 13 weeks, and 2 years (Ref. 17). Under the conditions of the study, there was no evidence of carcinogenic activity in any of the test animals. There were chemical-related increased incidences of kidney toxicity (nephropathy) in male and female mice. The Lowest Observed Adverse Effect Level (LOAEL) for

kidney toxicity in mice is approximately 180 milligrams per kilogram per day (mg/kg/day) (Refs. 19 and 20). Kidney toxicity was observed in rats, but the data are conflicting (kidney effects were seen in the 13-week study, but not in the 2-year study). Test animals and their offspring were not observed for reproductive or developmental effects. The results of the NTP study are summarized below. A more detailed summary is provided in Ref. 19.

In groups of 60 male and 60 female mice receiving 0, 500, 1,250, or 2,500 mg/L barium chloride dihydrate in drinking water for 2 years, dose-related nephropathy was observed. The incidence of nephropathy was significantly increased in mice of both genders that received 2,500 mg/L. The nephropathy consisted of extensive regeneration of cortical and medullary renal tubule epithelium, tubule dilatation, hyaline cast formation, multifocal interstitial fibrosis and in some kidneys, glomerulosclerosis. These lesions were accompanied by brown crystals (barium precipitated salts) located within the kidney's tubules lumen and interstitium throughout the cortex and medulla. The kidney lesions were considered the cause of death in most animals. The absolute and relative spleen weights in female rats in the highest dose were lower compared to controls. Based on the renal toxicity, the LOAEL is 160 mg/kg/day for male mice and 200 mg/kg/day for female mice. The No Observed Adverse Effect Level (NOAEL) is 75 mg/kg/day for male mice and 90 mg/kg/day for female mice.

Groups of 10 male and 10 female F344/N rats received barium chloride dihydrate in drinking water at doses of 0, 125, 500, 1,000, 2,000 or 4,000 mg/L, 7 days a week for 13 weeks (Ref. 17). Drinking water levels were estimated to deliver daily doses of 10, 30, 65, 110, or 200 mg/kg for male rats and 10, 35, 65, 115, or 180 mg/kg body weight to females. Three male rats and one female rat that received 4,000 mg/L died during the last week of the study. A significant decrease in motor activity was observed in rats that received the highest dose.

The absolute and relative kidney weights of female rats that received 2,000 and 4,000 mg/L and the relative kidney weight of male rats in the 4,000 mg/L groups were greater than controls and were associated with barium-induced renal lesions. Barium-induced renal lesions occurred in three male and three female rats in the highest dose groups. Gross pathology revealed kidneys that were pale and had roughened surfaces. Microscopically, the kidney lesion appeared as a minimal to mild focal to multifocal dilatation of the proximal convoluted tubules in the outer medulla and the renal cortex. Tubule dilatation observed in this study was different from the common spontaneous lesions observed in the kidney of rats.

In a similar 13–week study on mice (Ref. 17), barium-induced nephropathy was observed in 10 male and 9 female mice in the highest dose group. Gross pathology revealed kidneys that were pale and had roughened surfaces. The nephropathy consisted of mild to moderate multifocal tubule dilatation, regeneration and atrophy with crystals in the lumens of the atrophic tubules. An increased amount of fibrous connective tissue was present in the affected kidneys. The LOAEL in male mice was 450 mg/kg/day and in female mice was 495 mg/kg/day based on the mortality, lower final mean body weights and water consumption, presence of renal, thymic and splenic lesions. The NOAEL was 205 mg/kg/day for male mice and 200 mg/kg/day for female mice.

In a 13-week drinking water study (Ref. 11), barium chloride dihydrate was given to groups of 10 male and 10 female F344/N rats and B6C3F1 mice at levels of 0, 125, 500, 1,000, 2,000, and 4,000 mg/L (ppm). The estimated average barium doses for rats were 0, 5.1, 20.0, 39.0, 70.0, and 128 mg/kg/day and for mice were 0, 12.0, 45.0, 83.0, 165, and 399 mg/kg/day. Mortality ranged from 60 to 70 percent in mice and from 10 to 30 percent in rats in the 4,000 mg/L groups. Deaths in mice were associated with barium-induced renal toxicity. Renal lesions in rats were much less severe than in mice and did not contribute to the barium-induced deaths seen in the high dose group. In both species the highest dose produced marginal decreases in motor activity, grip strength, and thermal sensitivity. The authors attributed these effects to secondary changes resulting from barium chloride toxicity at this dose. In mating trials, no anatomical effects on offspring of rats or mice were noted. Rats given 4,000 mg/L had marginal reductions in pup weights. No effects were noted on reproductive indices. Based on the mortality and renal toxicity at 4,000 mg/L in both rats and mice, the NOAEL was 70 mg/kg/day in rats and 165 mg/kg/day in mice.

Reports on the cardiovascular effects of subchronic and chronic exposure to barium in humans and animals vary. Brenniman et al. (Ref. 7) conducted an epidemiological study in which death-rates (established from death certificates) in communities with high levels of barium in their drinking water (2 -10 mg/L) were compared to communities that were exposed to low levels of barium in water (0.0 - 0.2 mg/L). While an initial analysis of the data indicated statistical differences in blood pressure between the communities, extensive analysis did not. No statistically significant differences were found in blood pressure between individuals in the two cities even when adjustments for duration of exposure, use of water softeners and the use of antihypertensive drugs were made (Ref. 17) .

In a human study conducted by Wones et al. (Ref. 8), 11 healthy men were enrolled in a 10-week barium drinking water dose-response protocol. Diet and lifestyle were controlled and the barium content of the drinking water was varied from 0 mg/L (first 2 weeks) to 5 mg/L (next 4 weeks) to 10 mg/L (last 4 weeks). There were no changes in morning or evening systolic or diastolic blood pressures, plasma cholesterol or lipoprotein, serum potassium or calcium or glucose levels. There were no arrythmias related to barium exposure. Consumption of barium in drinking water at a dose of 0.21 mg barium/kg/day did not appear to affect any of the cardiovascular parameters monitored in this study (Ref. 17). This study was considered limited by the EPA's Office of Drinking Water due to its small study population and short duration of exposure (4 weeks) and because there was no lowest effect dose.

Perry et al. (Ref. 9) studied the effect of barium in drinking water on blood pressure in rats. A total of 195 female weanling Long-Evans rats were subdivided into a control group of 26 animals (0 mg/L) and 3 exposure groups of 13 rats. Each group was provided drinking water containing 1, 10, or 100 mg/L of barium chloride for 1, 4, or 16 months. There were significant increases in mean systolic blood pressure in rats receiving the highest dose at 1 and 4 months (7.1 and 6.3 mg/kg/day, respectively). In the 16-month study, rats exposed to 0.51 and 5.1 mg/kg/day had significant increases in blood pressure as well. Also at the highest dose, there was a decrease in contractility and excitability of cardiac muscle fiber. The LOAEL for the 16-month study was 0.51 mg/kg/day as evidenced by increase in blood pressure and the NOAEL was 0.051 mg/kg/day. However, the test animals were maintained on a special contaminant-free diet that restricted their intake of certain beneficial trace metals, such as calcium and potassium. This restriction may have contributed to the observed hypertensive effects. Several other studies with rats and mice lasting from

13 weeks to 2 years show no increase in blood pressure or any other cardiovascular effects.

3. *Ecotoxicity*. Barium compounds have low toxicity to aquatic organisms and plants (Refs. 15 and 22). The low toxicity of barium compounds to aquatic species is attributable to the presence of sulfate in waters; barium ion liberated from a barium compound reacts with sulfate to form barium sulfate, which precipitates from solution.

C. Environmental Fate of Barium Compounds

EPA's environmental fate evaluation of barium compounds consisted of an analysis of environmental fate data pertaining to barium-containing substances included on the EPCRA section 313 list of toxic chemicals as part of the barium compounds category. Data were obtained from studies found in the literature (Refs. 23, 26-29, 31, and 32) and several government documents (Refs. 24, 25, and 30). The portions of these reference sources that are relevant to EPA's review of the environmental fate of barium compounds are summarized below. Detailed discussions can be found in the publications and in Ref. 33, EPA's technical review of these publications.

1. *Air*. Most barium compounds released to the environment from industrial sources are in forms that do not become widely dispersed (Ref. 23). In the atmosphere, barium compounds are likely to be present in particulate form. Although chemical reactions may cause changes in speciation of barium in air, the main mechanisms for the removal of barium compounds from the atmosphere are likely to be wet and dry deposition (Ref. 24).

Elemental barium is oxidized readily in moist air (Refs. 25 and 26). The residence time of barium in the atmosphere may be several days, depending on the size of the particulate formed, the chemical nature of the particulate, and environmental factors such as rainfall (Ref. 24).

2. *Water*. In aquatic media, barium compounds are likely to precipitate out of solution as barium sulfate ($BaSO_4$) or barium carbonate ($BaCO_3$). Waterborne barium may also adsorb to suspended particulate matter (Refs. 24, 27, and 28). Precipitation of barium sulfate is accelerated when rivers enter ocean waters. This is due to the higher sulfate content in ocean waters (Ref. 33). Sedimentation removes a large portion of barium compounds that are suspended in surface waters (Ref. 29).

Appreciable quantities of barium sulfate or carbonate precipitate may occur in aquatic environments. This is because natural waters usually contain sulfate or carbonate concentrations that are sufficient to react with barium ion to form barium sulfate or carbonate, which precipitates from solution (Ref. 30). In natural waters at pH levels of 9.3 or below, barium ion will react to form barium sulfate (Ref. 27). At pH above 9.3 formation of barium carbonate is favored.

3. *Soil*. Barium is not very mobile in most soils. The rate of transportation of barium in soils is dependent on soil characteristics. Soil properties that influence the transportation of barium to groundwaters are cation exchange capacity and calcium carbonate ($CaCO_3$) content. In soils with a high cation exchange capacity (e.g., fine textured mineral soils or soils with high organic matter content), barium mobility will be limited by adsorption (Ref. 28). High calcium carbonate content limits mobility by precipitation of the element as barium carbonate. In soils, barium will also precipitate as barium sulfate in the presence of sulfate ions (Refs. 27 and 28). Barium is more mobile and is more likely to be leached from soils in the presence of chloride due to the increased solubility of barium chloride as compared to other chemical compounds of barium (Ref. 28). Barium can form compounds with fatty acids (e.g., in acidic landfill leachate) with enhanced mobility in soils due to the lower charge of these compounds and subsequent reduction in adsorption capacity (Ref. 28). The significance of these mobility enhancing processes is thought to be minor overall, and it is likely that in the presence of sulfate or carbonate in soils, barium ion will react to form a solid (barium sulfate or barium carbonate) with relatively low mobility.

4. *Barium solubility in anaerobic environments*. Although the formation of barium sulfate precipitate is thought to be the major fate pathway for barium ion in aqueous environments containing adequate levels of sulfate, there is evidence indicating that under anaerobic, low sulfate conditions, enhanced barium solubility from barium sulfate can occur. Barium ion concentrations greater than those expected based on the solubility of barium sulfate can result through a series of steps in which available sulfate is reduced to sulfide by anaerobic bacteria (Ref. 31).

The existence of anaerobic, sulfate poor aquatic environments where enhanced barium solubility may occur has been documented (Ref. 32). However, these environments are often found in northern glaciated regions in water bodies that are isolated from flowing surface waters and groundwaters. As these areas tend to be remote, the likelihood of releases of barium compounds entering these environments with subsequent attainment of barium ion concentrations of environmental significance is low.

D. Acute Exposure

Because barium compounds have been associated with acute effects in humans, EPA conducted a limited exposure analysis. (See discussion of use of exposure in listing decisions, 59 FR 61440, November 30, 1994.) Based on the TRI data, EPA has determined that the concentration levels of barium compounds likely to exist beyond facility site boundaries are low compared to the levels that would be required to induce the acute toxicities discussed above. Therefore, EPA does not believe that adverse acute human health effects are reasonably likely to occur as a result of continuous, or frequently recurring releases of barium compounds from facilities (Ref. 33).

IV. Technical Summary

EPA's technical review shows that many barium compounds are known to produce toxic effects in humans and experimental animals with the main target organ being the kidneys. Several barium compounds are acutely toxic to humans; however, EPA's exposure analysis indicates that the concentrations required to produce these acute toxicities are not reasonably likely to exist beyond facility site boundaries as a result of continuous, or frequently recurring releases of barium compounds from facilities. With regard to chronic toxicity, the data from animal studies support a LOAEL of approximately 180 mg/kg/day for renal toxicity. Based on these data, EPA considers barium ion to have moderately high chronic toxicity. From its technical review EPA concludes that: barium ion is bioavailable from barium compounds, including some compounds with low water solubility (e.g, barium carbonate); and that barium ion is responsible for the toxic effects produced by barium compounds. Available data indicate that barium compounds are not ecotoxic. EPA's previous determination (59 FR 33205, June 28, 1994) (FRL-4767-5) that barium sulfate is essentially non-toxic to humans and the environment, and thus does not meet the EPCRA section 313(d)(2) criteria for listing remains unchanged.

V. Rationale for Denial

With the exception of barium sulfate, barium-containing substances are chemicals subject to EPCRA section 313

(listed under the category of ''barium compounds'') and PPA section 6607 reporting requirements. The petition to delist barium compounds is based on the petitioner's contention that barium compounds are not toxic and do not meet any of the statutory criteria under section 313(d)(2). In addition, the petitioner contends that due to an abundance of sulfate in the environment, barium ion is not available from barium compounds released into the environment because environmental sulfate will combine with barium ion to form barium sulfate.

EPA's review of available data has led the Agency to conclude that in experimental animals and humans: (1) Barium ion is available from barium compounds, including some compounds that have low water solubility; and (2) barium ion causes moderately high toxicity to the kidney.

Based on available data, EPA concludes that barium compounds can reasonably be anticipated to cause chronic toxicity in humans because of their ability to liberate barium ion, which in turn causes adverse chronic health effects. Therefore, barium compounds meet the criteria of EPCRA section 313(d)(2)(B). EPA concludes that barium compounds should not be deleted from the section 313 list of toxic chemicals, and the petition should be denied. Because barium compounds can reasonably be anticipated to cause moderately high chronic toxicity, EPA does not believe that an exposure assessment is necessary to conclude that barium compounds meet the toxicity criterion of EPCRA section 313(d)(2)(B). For a discussion of the use of exposure in EPCRA section 313 listing/delisting decisions, see 59 FR 61440, November 30, 1994.

EPA agrees with the petitioner that sulfate is a ubiquitous substance in the environment, and that sulfate reacts with barium ion to form barium sulfate. EPA also agrees that barium sulfate does not meet the criteria for listing on the section 313 list of toxic chemicals. EPA does not agree, however, that the presence of sulfate in the environment ensures that barium compounds cannot be toxic to humans. In its review of the toxicity of barium compounds, EPA concludes that environmental presence of barium ion is not a necessary prerequisite for toxicity from a barium compound. In the technical review portion of this notice, EPA describes studies in which adverse effects were observed following exposure to an intact barium compound. The toxicity occurs as a consequence of barium ion release *in vivo*. Therefore, exposure to an intact barium compound can reasonably be anticipated to cause toxicity as a result of the release of barium ion in the body.

In addition, EPA does not agree that the presence of sulfate in the environment automatically ensures that barium ion availability will not result from barium compounds released into the environment. EPA feels that continuous releases of a barium compound (particularly a highly soluble one) to a given area could deplete sulfate in that area. Once sulfate depletion takes place, continued release of the barium compound could lead to availability of barium ion.

EPA's denial of this petition is consistent with the Agency's published policy and guidance on metal compound categories under section 313 of EPCRA (56 FR 23703, May 23, 1991). This policy and guidance articulated EPA's determination that the toxicity of a metal-containing compound that dissociates or reacts to generate the metal ion can be expressed as a function of the toxicity induced by the intact species and the availability of the metal ion. Thus, EPA stated that for petitions to exempt individual metal-containing compounds from the EPCRA section 313 list of toxic chemicals, EPA bases its decisions on the evaluation of all chemical and biological processes that may lead to metal ion availability, as well as on the toxicity exhibited by the intact species. EPA stated that the Agency will deny petitions for chemicals that dissociate or react to generate the metal ion at levels which can reasonably be anticipated to cause adverse effects to human health or the environment and for which the metal ion availability cannot be properly characterized.

In summary, EPA's review of information pertaining to barium compounds resulted in the conclusion that in mammals: (1) Barium ion is available from barium compounds (including some compounds that have low water solubility); and (2) barium ion causes chronic toxic effects. Thus, barium compounds can reasonably be anticipated to cause chronic toxicity in humans because of their ability to liberate barium ion. EPA believes that the available data satisfy the criterion in EPCRA section 313(d)(2)(B). Accordingly, EPA is denying the petition.

VI. References

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17. U.S. Department of Health and Human Services. 1993 NTP Technical Report 432. Toxicology and Carcinogenesis Studies of Barium Chloride Dihydrate in F4344 Rats and B6C3F1 mice. NIH Publication # 93-3163.

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VIII. Administrative Record

The record supporting this decision is contained in docket control number OPPTS–400107. All documents, including the references listed in Unit VI. above and an index of the docket, are available to the public in the TSCA Non-Confidential Information Center (NCIC), also known as the Public Docket Office, from noon to 4 p.m., Monday through Friday, excluding legal holidays. The TSCA NCIC is located at EPA Headquarters, Rm. NE-B607, 401 M St., SW., Washington, DC 20460.

List of Subjects in 40 CFR Part 372

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

Dated: December 23, 1996.

Lynn R. Goldman,

Assistant Administrator, for Prevention, Pesticides and Toxic Substances.

[FR Doc. 97–56 Filed 1–2–97; 8:45 am]

BILLING CODE 6560–50–F

FEDERAL COMMUNICATIONS COMMISSION

47 CFR Part 73

[MM Docket No. 96–259; RM–8970]

Radio Broadcasting Services; Moscow, ID

AGENCY: Federal Communications Commission.

ACTION: Proposed rule.

SUMMARY: This document requests comments on a petition for rule making filed by Darin L. Siebert requesting the allotment of Channel 277A to Moscow, Idaho, as that community's second local commercial FM service. Coordinates used for Channel 277A at Moscow are 46–42–24 and 116–55–08. As Moscow, Idaho, is located within 320 kilometers (199 miles) of the Canadian border, the Commission must obtain the concurrence of the Canadian government to this proposal.

DATES: Comments must be filed on or before February 18, 1997, and reply comments on or before March 5, 1997.

ADDRESSES: Secretary, Federal Communications Commission, Washington, D.C. 20554. In addition to filing comments with the FCC, interested parties should serve the petitioner, as follows: Darin L. Siebert, S. 605 Grand Ave., Pullman, WA 99163.

FOR FURTHER INFORMATION CONTACT: Nancy Joyner, Mass Media Bureau, (202) 418–2180.

SUPPLEMENTARY INFORMATION: This is a synopsis of the Commission's Notice of Proposed Rule Making, MM Docket No. 96–259, adopted December 20, 1996, and released December 27, 1996. The full text of this Commission decision is available for inspection and copying during normal business hours in the FCC's Reference Center (Room 239), 1919 M Street, NW, Washington, DC The complete text of this decision may also be purchased from the Commission's copy contractors, International Transcription Service, Inc., (202) 857–3800, 2100 M Street, NW., Suite 140,Washington, DC 20037.

Provisions of the Regulatory Flexibility Act of l980 do not apply to this proceeding.

Members of the public should note that from the time a Notice of Proposed Rule Making is issued until the matter is no longer subject to Commission consideration or court review, all *ex parte* contacts are prohibited in Commission proceedings, such as this one, which involve channel allotments. See 47 CFR 1.1204(b) for rules governing permissible *ex parte* contacts.