Dated: February 12, 1999.

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Acting Assistant Administrator for Prevention, Pesticides and Toxic Substances.

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ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 372

[OPPTS–400135; FRL–6050–3]

RIN 2070–AC00

Methyl Isobutyl Ketone; 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 methyl isobutyl ketone (MIBK) 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). EPA has reviewed the available data on this chemical and has determined that MIBK does 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 MIBK meets the listing criteria of EPCRA section 313(d)(2)(B) due to its contribution to the formation of ozone in the environment which causes adverse human health and environmental effects.

FOR FURTHER INFORMATION CONTACT: Daniel R. Bushman, Petitions Coordinator, 202–260–3882 or e-mail: bushman.daniel@epa.gov, for specific information regarding this document or for further information on EPCRA section 313, contact the Emergency Planning and Community Right-to-Know Information Hotline, Environmental Protection Agency, Mail Code 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. General Information

A. Does This Notice Apply To Me?

This document does not make any changes to existing regulations, however you may be interested in this document if you manufacture, process, or otherwise use MIBK. Potentially interested categories and entities may include, but are not limited to the following:

Category	Examples of Potentially Interested Entities
Chemical manufacturers	Chemical manufacturers that manufacture MIBK, use MIBK as a chemical intermediate, or use MIBK in the manufacture of protective coatings such as nitrocellulose lacquers and solvent-based vinyl and acrylic coatings
Chemical processors and users	Facilities that use MIBK as a process solvent

This table is not intended to be exhaustive, but rather provides a guide for readers regarding entities likely to be interested in this document. Other types of entities not listed in this table may also be interested in this document. Additional businesses that may be interested in this document are those covered under 40 CFR part 372, subpart B. If you have any questions regarding whether a particular entity is covered by this section of the CFR, consult the technical person listed in the ''FOR FURTHER INFORMATION CONTACT'' section.

B. How Can I Get Additional Information or Copies of This Document or Other Support Documents?

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

2. *In person or by phone*. If you have any questions or need additional information about this action, please contact the technical person identified in the ''FOR FURTHER INFORMATION CONTACT'' section. In addition, the official record for this document, including the public version, has been established under docket control number OPPTS–400135, (including the references in Unit VII. of this preamble). This record includes not only the documents physically contained in the docket, but all of the documents included as references in those documents. A public version of this record is available for inspection from 12 noon to 4 p.m., Monday through Friday, excluding legal holidays. The official record is located in the TSCA Nonconfidential Information Center, Rm. NE–B607, 401 M St., SW., Washington, DC.

II. Introduction

A. Statutory Authority

This action is taken under sections 313(d) and (e)(1) of 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 in amounts above reporting threshold levels, to report their environmental releases of such chemicals annually. Such facilities must also report pollution prevention and recycling data for such chemicals, pursuant to section 6607 of the PPA of 1990, 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. MIBK was included on the initial list. 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, EPCRA section 313(d)(3) requires EPA to find that none of the listing 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 compounds categories. EPA has also published in the **Federal Register** of November 30, 1994 (59 FR 61432) (FRL–4922–2) 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. Description of Petition and Regulatory Status of Methyl Isobutyl Ketone

MIBK is on the list of toxic chemicals subject to the annual release reporting requirements of EPCRA section 313 and PPA section 6607. MIBK was among the list of chemicals placed under EPCRA section 313 by Congress. MIBK is also subject to Clean Air Act Amendments (CAAA) and the Hazardous Waste Constituents List under the Resource Conservation and Recovery Act (RCRA). MIBK is considered a volatile organic compound (VOC) based on EPA's regulatory definition of a VOC (57 FR 3941, February 3, 1992).

On April 23, 1997, EPA received a petition from the Ketones Panel of the Chemical Manufacturers Association (CMA) to delete MIBK from the list of chemicals reportable under EPCRA section 313 and PPA section 6607. CMA had submitted petitions to delete methyl ethyl ketone (MEK) and MIBK from the EPCRA section 313 reporting requirements in September 1988, but these petitions were subsequently withdrawn because the petitioner became aware of the Agency's concerns for various toxicological effects of these chemicals. The petitioners state that since that time, EPA's concern for the toxicity of MIBK has decreased. Therefore, the petitioners argue that MIBK does not meet any of the listing criteria, and should be removed from the reporting requirements of EPCRA section 313.

Specifically, the petitioners believe that MIBK is not known to cause, nor can it reasonably be anticipated to cause, significant adverse acute health effects at exposure levels that are likely to occur beyond industrial site boundaries as a result of continuous or frequently recurring releases. They also state that MIBK is not known to cause and cannot reasonably be anticipated to cause, significant chronic health effects in humans. The petitioners argue that MIBK also does not cause the type of adverse environmental effects that warrant reporting under EPCRA section 313.

Significant to the deliberations surrounding this petition review, is MIBK's status as a VOC. The petitioners argue for a revised interpretation of the EPCRA section 313 VOC policy. The basis for this argument is the petitioners contention that EPA does not have the statutory authority to list chemicals based upon indirect toxicity. The petitioners further contend that: (1) There are more effective ways to gather VOC emissions data; (2) EPA has other, more efficient, tools than the Toxics Release Inventory (TRI) for disseminating VOC emissions data; (3) TRI data are not used to support VOC emissions control programs; (4) the act of including non-toxic VOCs on the TRI may actually be counter productive, by providing disincentives for switching to these less toxic VOCs; and, (5) releases of MIBK in ozone non-attainment areas do not justify a nationwide reporting requirement (Ref. 1).

IV. EPA's Technical Review Of Methyl Isobutyl Ketone

The technical review of the petition to delete MIBK from the reporting requirements of EPCRA section 313 included an analysis of the available chemistry, health effects, ecological effects, environmental fate, exposure, and risk data for MIBK. Summaries of the technical reviews are provided in Unit IV.A. through E. The docket for this document contains additional information and more detailed discussions concerning the data available for MIBK. The reader should consult the support documents (Refs. 2, 3, 4, and 5) as well as the other studies contained or referenced in the docket.

A. Chemistry and Use

MIBK, also known as, MIK, 4-methyl-2-pentanone, 2-methyl-4-pentanone, and other names, is the second largest volume commercially produced ketone. It is a clear, colorless, stable, moderately low boiling, volatile, highly flammable liquid with a sweet, acetone-like odor. It is moderately soluble in water (17 grams per liter (g/l) at 20 °C, is miscible with most organic solvents, and forms azeotropes (i.e., mixtures that distill off in a fixed ratio) with water and many organic liquids. MIBK has strong solvent power and is a good solvent for many natural and synthetic resins (Ref. 2).

There were 163 million pounds of MIBK produced in the U.S. in 1996 and 25 million pounds were imported. Domestic production capacity is projected to hold steady at 210 million pounds through 1999. Domestic consumption was 148 million pounds in 1996. More than half of the MIBK consumed in the U.S. (62 percent) was used as a solvent for protective coatings. The next largest use of MIBK (18 percent) was as a chemical intermediate for rubber antioxidants and acetylenic surfactants (Refs. 2 and 3).

B. Metabolism and Absorption

MIBK is well-absorbed from the lung, gastrointestinal (GI) tract, and skin and is rapidly metabolized (Ref. 4).

C. Toxicological Evaluation

1. *Acute toxicity*. Available data indicate that MIBK has low acute toxicity. In humans, short-term inhalation exposures up to 30 minutes each day to concentrations as high as 500 parts per million (ppm) produced irritation of the eyes and upper and lower respiratory system, effects characteristic of solvent exposure (Ref. 4).

2. *Subchronic and chronic toxicity*. An assessment of direct exposure systemic toxicity from available subchronic toxicity studies on MIBK indicates that MIBK may cause liver and kidney toxicity. However, without additional chronic data, the effects seen were not considered to be serious or irreversible (Ref. 4).

i. *Carcinogenicity*. EPA was unable to identify any human or animal carcinogenicity data on MIBK. Although MIBK was weakly positive in the mouse lymphoma mutagenicity assay and in the mouse embryo cell transformation assay, there is insufficient evidence to reasonably extrapolate this information to anticipate that MIBK may cause cancer in humans (Refs. 4 and 6).

ii. *Mutagenicity*. Studies indicate that MIBK is not a gene mutagen in *Salmonella typhimurium* strains TA98, TA100, TA1535, and TA 1538 either with or without metabolic activation. MIBK is weakly positive in mouse lymphoma cells *in vitro* without but not with activation, is not a chromosome mutagen *in vitro* in Chinese hamster ovary and rat RL4 cells, nor does it induce micronuclei *in vivo* in the mouse micronucleus assay by intraperitoneal injection. MIBK does not induce DNA effects in the *Saccharomyces cerevisiae* homozygosis and recombination assay, and it is equivocal in the unscheduled DNA synthesis assay in rat hepatocytes *in vitro*. MIBK induces morphological cell transformation in BALB/c 3T3 cell in culture without and possibly with metabolic activation. Thus, in general, MIBK exposure does not appear to be associated with genotoxicity *in vitro* or *in vivo* (Refs. 4 and 7).

iii. *Developmental toxicity*. MIBK was subject to testing under section 4 of the Toxic Substances Control Act (TSCA). As part of the testing requirements for MIBK, a developmental toxicity study in rats and mice (Ref. 8) was previously submitted and reviewed by EPA (Ref. 4). EPA's 1985 review of the data concluded that MIBK caused significant developmental toxicity (fetal death, reduced fetal body weight, and delayed ossification) only at the high-dose of 3,000 ppm (Ref. 9). No effects were observed at lower doses and a No

Observed Adverse Effect Level (NOAEL) of 1,000 ppm for both rats and mice was derived. A Lowest Observed Adverse Effect Level (LOAEL) of 3,000 ppm was derived based on fetotoxicity in rats.

EPA's 1988 review of the same study concluded that in the rat study there were statistically significant decreases in fetal body weight (Ref. 10). In addition, it was noted that marginal decreases in fetal body weight at the mid-dose of 1,000 ppm were observed when compared to controls but they were not statistically significantly different and were slightly higher than those in the low-dose group. It was concluded in that review that MIBK induced developmental effects in rats with a LOAEL of 300 ppm (the lowest dose tested). However, a statistical evaluation of fetal body weight over the dose range tested concluded that the significant reduction in fetal body weight per litter seen in small litters at the low-dose group of 300 ppm was actually an artifact of exceptionally heavy fetuses in two small litters in the control group and therefore not treatment-related. The results of that evaluation, coupled with the absence of effects at the mid-dose group of 1000 ppm, argued against a dose-related decrease in fetal body weight. Therefore, the LOAEL of 3,000 ppm and a NOAEL of 1,000 ppm appear to be the more appropriate toxicity levels (Ref. 4).

iv. *Reproductive toxicity*. No reproductive/fertility studies conducted with MIBK have been identified. The only information available is from the 90-day inhalation toxicity study on MIBK (Ref. 11). In that study, organ weight and histological data in high-dose rats and mice were comparable to controls for the ovaries, uterus, oviducts, vagina, cervix, testis, epididymis, prostate, and seminal vesicles. However, this is not sufficient information to characterize the potential for reproductive toxicity of MIBK (Ref. 4).

v. *Neurotoxicity*. While MIBK alone appears to produce only transient neurological effects at high doses, there is evidence that MIBK enhances the neurotoxic effects of other compounds (Ref. 4). It has been reported that simultaneous subchronic (90-days) exposure to vapors of 1,000 ppm *n*-hexane and 100, 250, 500, or 1,000 ppm MIBK markedly increased the neurotoxic action of *n*-hexane in hens (Ref. 12). Another study also supports the suggestion that MIBK synergizes the neurotoxic action of *n*-hexane by enhancing its metabolic activation through induction of cytochrome P-450 enzymes (Ref. 13).

vi. *Toxicity related to ozone formation*. MIBK is a volatile organic compound and, as such, has the potential to contribute to the formation of ozone in the troposphere (i.e., the lower atmosphere). As EPA has previously stated (59 FR 1788, January 12, 1994), ozone can affect structure, function, metabolism, pulmonary defense against bacterial infection, and extrapulmonary effects. Among these extrapulmonary effects are: (1) Cardiovascular effects; (2) reproductive and teratological effects; (3) central nervous system effects; (4) alterations in red blood cell morphology; (5) enzymatic activity; and (6) cytogenetic effects on circulating lymphocytes.

3. *Ecotoxicity*. MIBK is of low concern with respect to aquatic toxicity based on measured toxicity data and quantitative structure activity relationship (QSAR) analysis (Refs. 4 and 14). Measured toxicity values include a fish 96-hour lethal concentration for 50 percent of the testing sample (LC_{50}) of 780 milligrams per liter (mg/L), a daphnid 24-hour LC_{50} of 4,300 mg/L and a green algal 48-hour effective concentration for 50 percent of the population (EC_{50}) of 980 mg/L. Consistent with the measured values, QSAR predicted acute toxicity resulted in a 96-hour LC_{50} of 420 mg/L for fish and a 96-hour EC_{50} of 250 mg/L for green algal. The QSAR predicted chronic toxicity value for fish is 47 mg/L, the daphnid chronic value is 15 mg/L, and the chronic algal value is 16 mg/L. In addition, the 28-day bioconcentration factor (BCF) of 0.5 is low.

As a VOC, MIBK contributes to the formation of ozone in the environment. As EPA has previously stated (59 FR 1788, January 12, 1994), ozone's effects on green plants include injury to foliage, reductions in growth, losses in yield, alterations in reproductive capacity, and alterations in susceptibility to pests and pathogens. Based on known interrelationships of different components of ecosystems, such effects, if of sufficient magnitude, may potentially lead to irreversible changes of sweeping nature to ecosystems.

D. Toxicological Summary

The only toxicological studies that provide sufficient evidence that MIBK can be reasonably anticipated to cause serious or irreversible health effects from direct exposure are the developmental toxicity studies. According to the EPA guidelines for developmental toxicity risk assessment (1991), evidence of developmental toxicity in a single animal study is sufficient to assume a potential hazard to humans. These developmental studies indicate that MIBK has the potential to cause developmental effects at moderately high to high doses. Other types of health effects from direct exposure are not considered either because the available data do not support a concern that is consistent with the criteria, or the data are lacking. However, as a VOC, MIBK contributes to the formation of tropospheric ozone which can cause significant adverse effects to human health and the environment.

E. Exposure Review

The available data indicate that MIBK can cause chronic developmental toxicity at moderately high to high doses (i.e., MIBK has low to moderately low toxicity). Because MIBK has low to moderately low toxicity EPA believes it is appropriate to conduct an exposure assessment. Since there is a possibility that the chronic developmental effects associated with exposures to relatively high concentrations of MIBK could be caused by short-term exposures, a short-term (i.e., acute type) exposure assessment was conducted (Ref. 5). The exposure assessment was conducted only to determine the potential for adverse chronic developmental effects to occur as a result of concentrations of MIBK that are reasonably likely to exist beyond facility site boundaries. For a discussion of the use of exposure considerations in modifying the EPCRA section 313 list of toxic chemicals, refer to the **Federal Register** of November 30, 1994 (59 FR 61432).

1. *Exposure assessment*. Two exposure scenarios were considered, ambient air exposures at or beyond the facility site boundary and drinking water exposures due to releases to the surface water. The estimates were derived through the use of 1994 annual release information submitted under TRI and standard modeling techniques.

Releases reported for MIBK during 1994 were retrieved from the Toxic Release Inventory System (TRIS) data base. According to TRIS, more than 25,500,000 pounds of MIBK were released in 1994 from 1,031 sources nationwide. Of this amount, 27 percent was from fugitive or nonpoint source emissions and 72 percent originated from stack or point source emissions to the atmosphere (Ref. 5). In addition, lesser amounts of MIBK (less than 1 percent) were released to surface waters, underground injection of wastes, and the land.

The SCREEN3 and the Industrial Source Complex Short Term (ISCST3) models were used to derive estimates of acute MIBK air concentrations (Ref. 5). These acute models provided estimates

of concentrations of MIBK in the air for both 1 and 24 hours. The ReachScan model was used to derive estimates of acute MIBK water concentrations. These concentrations were used to calculate exposures resulting from surface water releases to drinking water sources (Ref. 5).

The ambient air concentrations estimated are based on the assumption that releases take place continuously over 365 days per year; releases occurring over shorter periods will result in higher concentrations. Ninety-nine percent of all MIBK released into the environment is through stack (point) and fugitive (area) emissions into the atmosphere (Ref. 5). The remaining one percent of releases go to surface waters, landfill, and deep well injections.

Modeling data was used to estimate Average Potential Dose Rates (APDRs) for MIBK. The inhalation APDRs range from 0.2 to 3.3 milligrams/kilogram/day (mg/kg/day) and the drinking water exposure from the five facilities that result in the highest concentration in surface waters ranged from 0.92 to 47 micrograms per liter (ug/L). The resulting drinking water APDRs from these same sites ranged from 2.8×10^{-5} to 1.4×10^{-3} mg/kg/day.

2. *Exposure evaluation*. A margin of exposure (MOE) approach was used in this assessment to describe potential risks associated with exposure to MIBK (Ref. 4). The MOE is calculated as the ratio of the NOAEL for developmental toxicity to the estimated exposure level. The MOE does not provide an estimate of population risk, but simply describes the relative distance between the exposuere level and the NOAEL. The value of the MOE that is associated with a concern for toxic effects is generally expressed as the product of the applicable uncertainty and modifying factors; uncertainty factors that the Agency considers for non-cancer effects are described in the Integrated Risk Information System (IRIS) (1998). For consideration of developmental toxicity, the applicable uncertainty factors are described in the developmental toxicity guidelines (1991). These include two uncertainty factors, one for consideration of intraspecies variation, and another for interspecies variation. In accordance with EPA science policy, each of these uncertainty factors is given a value of 10. Thus, for developmental effects, an MOE greater than 100 would generally indicate a low level of concern, whereas a value less than 100 is judged to be of concern.

The rat NOAEL of 1,000 ppm from the inhalation developmental toxicity study (6 hour exposures) was converted to an average daily dose of 1,152 mg/kg/day. The NOAEL was then adjusted to a 24 hour exposure duration (to achieve consistency with the exposure estimates, which represent daily averages) and MOEs were calculated by dividing the inhalation developmental toxicity NOAEL by the APDR estimates for each of the top discharging facilities. MOEs for the highest single hour of the year were not derived since the animal dose from the inhalation developmental toxicity study was defined on a daily basis and since there were uncertainties in the relevance of this scenario as a descriptor of anticipated exposures. The relevant exposure scenario for the pregnant female was defined in the exposure assessment as time spent at home, 23.7 hours/day at exposures resulting from releases from MIBK to air (stack and fugitive) for the highest single day of the year. However, an exposure scenario duration of 23.7 hours/day spent inside a residence may not characterize the target population. To complement the analysis, an exposure duration of 16.4 hours spent inside a residence was also evaluated. In addition, there were concerns about the uncertainty introduced by comparing time spent indoors to outdoor ambient air concentrations of MIBK. Therefore, the recommended value of 2 hours/day spent outdoors at a residence was also evaluated (Ref. 15). The MOEs for the exposure durations depicted were greater than 100 for all of the top discharging facilities for exposure estimates derived with the ISCST3 model, while corresponding MOEs based on estimates obtained with the SCREEN3 model were lower than 100. The ISCST3 model allows for the use of more site-specific data, in this case wind speed, and therefore estimates of exposure obtained using this model provide more relevant information.

The APDR estimates for acute exposures resulting from surface water releases for the top five discharging facilities range from 2.8×10^{-5} to 1.4×10^{-3} mg/kg/day; the MOE values for these estimates range from 1.7×10^{8} to 3.3×10^{6}. Therefore, the MOE is greater than 100 for acute exposures resulting from surface water releases for all of the top discharging facilities (Ref. 4).

In summary, based on the concentrations likely to exist beyond facility site boundaries and the resulting MOE calculations from the exposure conditions described here, there is low concern for a potential for developmental effects for the general population following acute inhalation exposures to MIBK (Ref. 4).

V. Summary of Technical Review

The hazard assessment indicates that, except for VOC concerns, MIBK has low acute and chronic (systemic) toxicity in that effects occur only at high doses (3,000 ppm.). Based on information currently available, all toxicity endpoints examined, except for developmental toxicity, did not appear to meet the listing criteria for EPCRA section 313. A screening level risk assessment for developmental toxicity indicated low risk based on modeled potential acute exposures to women living in communities near release sites. Thus, based on EPA's modeling, TRI reported releases of MIBK are not expected to be sufficient to cause the type of high dose developmental effects associated with MIBK. The available data do indicate that MIBK can enhance the neurotoxicity of other solvents such as *n*-hexane; however, at this time EPA has not made a final determination as to the significance of this effect with regard to the EPCRA section 313(d)(2) criteria. MIBK has low direct environmental toxicity. MIBK is however a high volume VOC that contributes to the formation of tropospheric ozone which can cause significant adverse effects to human health and the environment.

VI. Rationale for Denial

EPA is denying the petition submitted by the Ketones Panel of the Chemical Manufacturers Association to delete MIBK from the EPCRA section 313 list of toxic chemicals. This denial is based on EPA's conclusion that VOCs, such as MIBK, contribute to the formation of tropospheric ozone which is known to cause significant adverse effects to human health and the environment. Therefore, EPA has concluded that MIBK meets the listing criteria of EPCRA section 313(d)(2)(B) and (d)(2)(C) because MIBK contributes to the formation of ozone, which causes serious adverse human health and environmental effects at relatively low doses. EPA has previously stated that ozone meets the listing criteria of EPCRA section 313(d)(2)(B) and (d)(2)(C) (59 FR 61432, November 30, 1994), and that because VOCs contribute to the formation of tropospheric ozone they meet the criteria for listing under EPCRA section 313 (54 FR 4072, January 27, 1989; 54 FR 10668, March 15, 1989; 59 FR 49888, September 30, 1994; 60 FR 31643, FRL–4952–7, June 16, 1995; and 63 FR 15195, FRL–5752–6, March 30, 1998). EPA has also stated (54 FR 4072, January 27, 1989 and 54 FR 10668, March 15, 1989) that while it is not EPA's intention to include all VOC

chemicals on the EPCRA section 313 list, those VOCs whose volume of use or emissions are large enough to raise substantial VOC concerns would be retained on the EPCRA section 313 list. MIBK is a VOC with both a high production volume and high air emissions, therefore, EPA has determined that MIBK should remain on the EPCRA section 313 list of toxic chemicals.

EPA has previously determined (59 FR 61432, November 30, 1994) that ozone has moderately high to high chronic toxicity and high environmental toxicity. Therefore, in accordance with EPA's stated policy on the use of exposure assessments (59 FR 61432, November 30, 1994), EPA does not believe that an exposure assessment is appropriate for determining that MIBK meets the toxicity criteria of EPCRA section 313(d)(2)(B) and (d)(2)(C) based on its contribution to the formation of ozone.

EPA disagrees with the petitioner's contention that ''indirect toxicity'', such as that caused by VOCs, does not meet the EPCRA section 313 listing criteria. The EPCRA section 313(d)(2) listing criteria each state that EPA may list a chemical that it determines ''is known to cause or can reasonably be anticipated to cause'' the relevant adverse human health or environmental effect. It further provides that ''[a] determination under this paragraph shall be based on generally accepted scientific principles.'' Ultimately, the crux of the issue the petitioner raises lies in interpreting the phrase ''cause or can reasonably be anticipated to cause'', which Congress chose not to define. In arguing that EPA lacks the statutory authority to base its listing decisions on ''indirect toxicity'', the petitioner would have the Agency adopt an artificially narrow view of causation that would require a single-step path between exposure to the toxic chemical and the effect. Such a mechanistic approach confuses the mode or mechanism of the chemical's action (i.e., the chain of causation) with the fundamental question of whether, regardless of the number of intervening steps, there is a natural and continuous line, unbroken by any intervening causes, between exposure to the chemical and the toxic effect. By contrast, EPA believes that Congress granted the Agency broad discretion in making listing decisions and directed EPA to rely on generally accepted scientific principles in making determinations to implement this section of EPCRA.

It is a generally accepted scientific principle that causality need not be linear, i.e., a one-step process (e.g., Proposed Guidelines for Ecological Risk Assessment, September 9, 1996, 61 FR 47552 and 47586; Proposed Guidelines for Carcinogen Risk Assessment, April 23, 1996, 61 FR 17960 and 17981). And for purposes of EPCRA section 313, the distinction between direct and indirect effects is technically an artificial one. Whether the toxic effect is caused directly by a chemical by a one-step process, or indirectly by a degradation product of the chemical or by a second chemical that is created through chemical reactions involving the first chemical, the toxic effect still occurs as a result of the presence of the chemical in the environment. It makes no difference to the affected organism whether the toxic agent was a result of chemical reactions. Fundamentally, EPCRA section 313 is concerned with adverse effects on humans and the environment, not the chain of causation by which such effects occur. In fact, this type of ''indirect'' toxicity is not unlike the effects of certain nonlinear carcinogens. Some carcinogens induce cancer through a multiple-step mechanism in which the chemical causes an intervening pathological change, and this pathological change is the direct cause of the cancer, but this does not mean that the chemical is not known or reasonably anticipated to cause cancer. It is therefore reasonable for EPA to consider such effects in light of the broad statutory purpose to inform the public about releases to the environment. Were EPA to exclude indirect effects from consideration, it would dilute the purpose of the statute by precluding public access to information about chemicals that cause a wide range of adverse health and environmental effects.

VII. References

1. CMA, 1996. Petition of the Chemical Manufacturers Association Ketones Panel to Delist Methyl Isobutyl Ketone Under Section 313 of the Emergency Planning and Community Right-to-Know Act of 1986. Chemical Manufacturers Association. (April 27, 1997).

2. USEPA, OPPT. Tou, Jenny; ''Chemistry Report on Methyl Isobutyl Ketone, EPCRA 313 Delisting Petition.'' (August 10, 1997).

3. USEPA, OPPT. Rice, Cody, ''Economic Analysis of the Proposed Deletion of Methyl Isobutyl Ketone from the EPCRA 313 List of Toxic Chemicals.'' (May 20, 1997).

4. USEPA, OPPT. Anitole, Katherine; ''Hazard/Risk Assessment of Methyl Isobutyl Ketone'' (November 24, 1997).

5. USEPA, OPPT. Brennan,Tom and Cinalli, Christina, ''Exposure Assessment for Methyl Isobutyl Ketone.'' (August 14, 1997). Docket control number OPPTS–400110 contains the references cited in this document.

6. USEPA, OPPT. Memorandum from David Lai, Ph.D., Existing Chemicals Assessment Branch, Risk Assessment Division, to Katherine Anitole, Ph.D., Existing Chemicals Assessment Branch, Risk Assessment Division. Subject: Hazard Assessment of Methyl Isobutyl Ketone (MIBK) in Response to Petition for Delisting in TRI: Carcinogenicity. (June 27, 1997).

7. USEPA, OPPT. Memorandum from Michael C. Cimino, Ph.D., Science Support Branch, Risk Assessment Division, to Katherine Anitole, Ph.D., Existing Chemicals Assessment Branch, Risk Assessment Division. Subject: Delisting Petition for Methyl Isobutyl Ketone (MIBK): Mutagenicity Hazard. (June 23, 1997).

8. Tyl, R.W., et al., ''Developmental Toxicity Evaluation of Inhaled Methyl Isobutyl Ketone in Fischer 344 Rats and CD-1 Mice.'' *Fund. Appl. Toxicol.* v. 8, (1987), p. 310.

9. USEPA, OTS. Memorandum from Myron S. Ottley, Ph.D., Toxic Effects Branch, Health and Environmental Review Division, to Jim Kariya, Chemical Review and Evaluation Branch, Health and Environmental Review Division. Subject: Review of Developmental Toxicity Data on Methyl Isobutyl Ketone. (January 18, 1985).

10. USEPA, OTS. Memorandum from Marlissa Campbell, Toxic Effects Branch, Health and Environmental Review Division, to Elbert Dage, Chemical Review and Evaluation Branch, Health and Environmental Review Division. Subject: Review of the Potential Developmental Toxicity of methyl Isobutyl Ketone (MIBK). (1988).

11. Phillips, R.O., et al., ''A 14-Week Vapor Inhalation Study of Methyl Isobutyl Ketone.'' *Fund. Appl. Toxicol.* v. 9, (1987), p.380.

12. Abou-Donia, M. et al., ''The Joint Neurotoxic Action of Inhaled Methyl Butyl Ketone Vapor and Dermally Applied o-Ethyl-o-4-Nitrophenyl Phenylphosphonothioate in Hens: Potentiating Effect.'' *Toxicol. Appl. Pharmacol.* v. 79, (1985), pp. 69–82.

13. Habig, C., Abou-Donia, M., Lapadula, D., ''Cytochrome P-450 Induction in Chickens Exposed Simultaneously to *N*-Hexane and Methyl IsoButyl Ketone.'' *The Toxicologist* v. 9, (1989), p. 194.

14. USEPA, OPPT. Memorandum from Jerry Smrcheck, Existing Chemicals Assessment Branch, Risk Assessment Division, to Katherine Anitole, Existing Chemicals Assessment

Branch, Risk Assessment Division. Subject: Ecological Hazard of MIBK. (June 26, 1997).

15. USEPA, ORD. 1997. Exposure Factors Handbook, Office of Research and Development, National Center for Environmental Assessment, U.S. Environmental Protection Agency, Washington, DC, (1997): EPA/600/P-95/002(Fa-Fc).

List of Subjects in 40 CFR Part 372

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

Dated: February 12, 1999.

Susan H. Wayland,

Acting Assistant Administrator for Prevention, Pesticides and Toxic Substances.

[FR Doc. 99–4320 Filed 2–22–99; 8:45 am]

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ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 372

[OPPTS–400134; FRL–6030–6]

RIN 2070–AC00

Chromite Ore from the Transvaal Region of South Africa; Toxic Chemical Release Reporting; Community Right-to-Know

AGENCY: Environmental Protection Agency (EPA).

ACTION: Proposed rule.

SUMMARY: EPA is granting a petition by proposing to exempt both chromite ore mined in the Transvaal Region of South Africa and the unreacted ore component of the chromite ore processing residue (COPR) from 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). These chemicals are currently reported as part of the category ''chromium compounds'' on the list of toxic chemicals in section 313(c) of EPCRA. The proposal is based on EPA's preliminary conclusion that this particular chromite ore from the Transvaal Region and the unreacted ore component of the COPR (in the case of this delisting decision, chromite ore processing residue, or COPR, includes the solid waste remaining after the aqueous extraction of oxidized chromite ore that has been combined with soda ash and kiln roasted at approximately 2,000 °F) meet the deletion criterion under EPCRA section 313(d)(3).

DATES: Written comments, identified by the docket control number OPPTS–400134, must be received by EPA on or before April 26, 1999.

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

FOR FURTHER INFORMATION CONTACT: Daniel R. Bushman, Petitions Coordinator, 202–260–3882 or e-mail: bushman.daniel@epamail.epa.gov, for specific information regarding this document or for further information on EPCRA section 313, the Emergency Planning and Community Right-to-Know Information Hotline, Environmental Protection Agency, Mail Code 7408, 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. General Information

A. Does this Proposal Apply to Me?

You may be potentially affected by this proposal if you kiln roast chromite ore in the production of chromium chemicals or if you process chromite ore (e.g., metal finishers, leather tanning, etc.). Potentially affected categories and entities may include, but are not limited to:

Category	Examples of Potentially Affected Entities
Chemical Manufacturers	Chemical manufacturers that kiln roast chromite ore in the production of chromium chemicals (e.g., sodium dichromate, sodium chromate, etc.)
Metal Manufacturers	Metal manufacturers that kiln roast chromite ore in the production of chromium chemicals (e.g., chromic acid, chromic oxide, potassium dichromate, chromic sulfate, calcium chromate, etc.)
Smelting Refractories	Smelting refractories that kiln roast chromite ore in the production of chromium chemicals (e.g., sodium dichromate, sodium chromate, etc.)

This table is not intended to be exhaustive, but rather provides a guide for readers regarding entities likely to be affected by this action. Other types of entities not listed in this table could also be affected. To determine whether you or your business is affected by this action, you should carefully examine the applicability provisions in part 372, subpart B of Title 40 of the Code of Federal Regulations (CFR). If you have any questions regarding the applicability of this action to a particular entity, consult the technical person listed in the ''FOR FURTHER INFORMATION CONTACT'' section.

B. How Can I Get Additional Information or Copies of this Document or Other Support Documents?

1. *Electronically*. You may obtain electronic copies of this document and various support documents from the EPA Internet Home Page at http://www.epa.gov/. On the Home Page select ''Laws and Regulations'' and then look up the entry for this document under the ''Federal Register - Environmental Documents.'' You can also go directly to the ''Federal Register'' listings at http://www.epa.gov/homepage/fedrgstr/.

2. *In person or by phone*. If you have any questions or need additional information about this action, please contact the technical person identified in the ''FOR FURTHER INFORMATION CONTACT'' section. In addition, the official rulemaking record for this proposal, including the public version, has been established under docket control number OPPTS–400134, (including the references in Unit VII. of