open the discussion. All members of each panel, however, are encouraged to fully participate in the discussion. The members of each panel are listed below. The opening presenters for each panel are designated by an asterisk.

The schedule and composition of the panels are as follows:

Panel I 1:00–2:45 p.m.

Fred Moring, Pipeline Customer Coalition *
Peggy Heeg, Interstate Natural Gas Association of America *
Randall Rich, Independent Oil & Gas Association of West Virginia
Representative from Duke Energy Pipelines
David Sweet, Independent Petroleum Association of America
Katherine Edwards, Amoco Energy Trading Corporation, Amoco Production Company, Burlington Resources Oil & Gas Company, and Marathon Oil Company
Representative from the Public Service Commission of the State of New York
Representative from the Association of Oil Pipelines
D. Jane Drennan, Chevron Products Company

Panel II 3:15–5:00 p.m.

Representative of Electric Power Supply Association *
Representative of Edison Electric Institute *
Susan N. Kelly, National Rural Electric Cooperative Association
Representative from the American Public Power Association
Jeffrey D. Watkiss, Coalition for a Competitive Electric Market
Gordon Gooch, Travis & Gooch
Representative of the American Arbitration Association

The symposium will begin at 1:00 p.m. in the Commission Meeting Room, Room 2C, 888 First Street, NE., Washington, DC 20426. Speakers that have audio/visual requirements should contact Wanda Washington at (202) 208–1460, no later than March 26, 1998.

The Capitol Connection will broadcast live the audio from the public conference on its wireless cable system in the Washington, DC area. If there is sufficient interest from those outside the Washington, DC metropolitan area, the Capitol Connection may broadcast the conference live via satellite for a fee. Persons interested in receiving the audio broadcast, or who need more information, should contact Shirley Al-Jarnai or Julia Morelli at the Capitol Connection at (703) 993–3100, no later than noon on March 25, 1998.

In addition, National Narrowcast Network's Hearing-On-The-Line service covers all FERC meetings live by telephone. Call (202) 966–2211 for details. Billing is based on time on-line.

The Commission will also afford an opportunity for persons to file written comments in response to discussion at the symposium. Those wishing to file comments should do so by April 14, 1998.

FOR FURTHER INFORMATION CONTACT: David Faerberg, Office of the General Counsel, Federal Energy Regulatory Commission, 888 First Street, NE., Washington, DC 20426, (202) 208–1275.

By direction of the Commission.

David P. Boergers,

Acting Secretary.

[FR Doc. 98–8193 Filed 3–27–98; 8:45 am]

BILLING CODE 6717–01–M

ENVIRONMENTAL PROTECTION AGENCY

[FRL–5988–8]

National Advisory Council for Environmental Policy and Technology—Total Maximum Daily Load Committee: Public Meeting

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice of public meeting.

SUMMARY: Under the Federal Advisory Committee Act, PL 92463, EPA gives notice of a three day meeting of the National Advisory Council for Environmental Policy and Technology's (NACEPT) Total Maximum Daily Load (TMDL) Committee. NACEPT provides advice and recommendations to the Administrator of EPA on a broad range of environmental policy issues. The TMDL Committee has been charged to provide recommendations for actions which will lead to a substantially more effective TMDL program. This meeting is being held to enable the Committee and EPA to hear the views and obtain the advice of a widely diverse group of stakeholders in the national Water Program.

In conjunction with the three day meeting, the FACA Committee members and the EPA will host one meeting designed to afford the general public greater opportunity to express its views on TMDL and water related issues.

DATES: The three day public meeting will be held on May 4–6, 1998, at the Westin Atlanta North at Perimeter Hotel, Seven Concourse Parkway, Atlanta, Georgia 30328, (770) 395–3940. The full Committee meeting is scheduled to begin Monday, May 4, 1998, at 9 a.m. and conclude at 5:30 p.m. The meeting will reconvene at 8:30 a.m. on Tuesday, May 5, 1998, and is scheduled to adjourn at 5:00 p.m. On Wednesday, May 6, 1998, the meeting will reconvene at 8:30 a.m. and conclude at 3:00 p.m.

The public input session is scheduled in conjunction with the full Committee meeting and will also be held at the Westin Atlanta North at Perimeter. It will occur on Monday, May 4, 1998, from 7:30 p.m. until 9 p.m.

ADDRESSES: Materials or written comments may be transmitted to the Committee through Hazel Groman , Designated Federal Officer, NACEPT/TMDL, U.S. EPA, Office of Water, Office of Wetlands, Oceans, and Watersheds, Assessment and Watershed Protection Division (4503F), 401 M Street, SW, Washington, DC 20460.

FOR FURTHER INFORMATION CONTACT: Hazel Groman, Designated Federal Officer for the Total Maximum Daily Load Committee at 202–260–8798.

Dated: March 17, 1998.

Hazel Groman,

Designated Federal Officer.

[FR Doc. 98–8217 Filed 3–27–98; 8:45 am]

BILLING CODE 6560–50–U

ENVIRONMENTAL PROTECTION AGENCY

[OPPTS–400119; FRL–5752–6]

Methyl Ethyl 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 ethyl ketone (MEK) 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 MEK 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 MEK meets the listing criteria of EPCRA section 313(d)(2)(B) and (C) 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@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 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 in amounts above reporting threshold levels, 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. MEK 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, 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 compounds categories. EPA has also published a statement clarifying its interpretation of the section 313(d)(2) and (3) criteria for adding and deleting chemical substances from the section 313 list (59 FR 61432, November 30, 1994) (FRL–4922–2).

II. Description of Petition and Regulatory Status of Methyl Ethyl Ketone

MEK is on the list of toxic chemicals subject to the annual release reporting requirements of EPCRA section 313 and PPA section 6607. MEK was among the list of chemicals placed under EPCRA section 313 by Congress. MEK is subject to the Clean Air Act (CAA) as a volatile organic compound (VOC) and a hazardous air pollutant. MEK is also on the Hazardous Waste Constituents List under the Resource Conservation and Recovery Act (RCRA).

On November 26, 1996, EPA received a petition from the Ketones Panel of the Chemical Manufacturers Association (CMA), to delete MEK from the list of chemicals reportable under EPCRA section 313 and PPA section 6607. CMA had submitted a petition to delete MEK and methyl isobutyl ketone (MIBK) from the EPCRA section 313 reporting requirements in September 1988, but this petition was subsequently withdrawn because the petitioner became aware of the Agency's concerns for developmental toxicity and neurotoxicity. The current petitioner states that since that time, EPA's concern for these effects has decreased. Therefore, the petitioner argues that MEK does not meet any of the listing criteria, and should be removed from the reporting requirements of EPCRA section 313.

Specifically, the Panel believes that MEK 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 MEK ''is not known to cause and cannot reasonably be anticipated to cause, significant chronic health effects in humans.'' They state that EPA's Integrated Risk Information System (IRIS) data base recognizes that MEK ''has little if any neurotoxic potential.'' In addition, the Panel discusses in the petition that based upon several developmental toxicity studies that have been conducted, EPA should use a revised reference concentration (RfC), based upon EPA modified guidance for conducting risk assessments. The petitioner argues that MEK also does not cause the type of adverse environmental effects that warrant reporting under section 313.

Significant to the deliberations surrounding this petition review, is MEK's status as a VOC. The petitioner argues for a revised interpretation of the EPCRA section 313 VOC policy, contending that EPA does not have the statutory authority to list chemicals based upon ''indirect'' toxicity. The petitioner further contends 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 MEK in ozone non-attainment areas do not justify a nationwide reporting requirement (Ref. 1).

III. EPA's Technical Review of Methyl Ethyl Ketone

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

A. Chemistry and Use

MEK, also known as 2-butanone, ethyl methyl ketone, and methyl acetone, is the largest volume commercially produced ketone other than acetone. It is a clear, colorless, stable, low-boiling (79.6 °C), highly volatile (vapor pressure 90.6 torr at 25 °C) and highly flammable (flash point 1 °C, autoignition temperature 515 °C) liquid with an acetone-like odor. It is very soluble in water (240 grams per liter (g/l) at 20 °C), miscible with organic solvents, and forms azeotropes with water and many organic liquids. MEK has exceptionally high solvent power and is a good solvent for many natural and synthetic resins. It is used as a solvent in the surface coatings industry, specifically in vinyl lacquers, nitrocellulose lacquers, and acrylics. It is used mainly in surface coatings and is also used as a chemical intermediate. It is also used as a solvent for adhesives, printing inks, degreasing and cleaning fluids, smokeless powder,

and as an intermediate in the production of antioxidants, perfumes, and catalysts (Ref. 2).

Most MEK is produced by a two-step process from petroleum derived butene/butane mixtures (Ref. 3). MEK is also available as a by-product from liquid phase oxidation of butane to acetic acid and is produced by direct oxidation of n-butenes.

There were 545 million pounds of MEK produced in the U.S. in 1994 and 16 million pounds were imported. Domestic production capacity is projected to increase to 595 million pounds in 1997. Three producers, Exxon Chemical, Hoechst-Celanese, and Shell Chemical, have been identified. Domestic consumption was 388 million pounds in 1994. More than half of the MEK consumed in the U.S. (60 percent) was used as a solvent for protective coatings, as virtually all natural and synthetic resins used in lacquers are soluble in MEK. The next largest use of MEK (14 percent) was in solvent-based adhesives, such as rubber cement. MEK was employed as a solvent in the manufacture of magnetic tapes (10 percent), and as a dewaxing agent in the refining of lubricating oil (5 percent). As a chemical intermediate (5 percent), MEK was used to produce perfumes, antioxidants, catalysts, peroxides, and diacetal. Three percent of the MEK consumed domestically was for printing ink, while another three percent was used for miscellaneous purposes, such as paint removal (Refs. 1 and 4).

Substitutes for MEK have been investigated by coating formulators with mixed success. Alternative technologies include 100 percent solvent products, water-based resins systems, and reformulated solvent blends. Ethyl acetate in some cases is a drop-in substitute for MEK with no significant change in properties. Butyl acetate and isobutyl acetate can be used in many formulations as partial or full substitutes for MEK. A blend of acetone and MIBK is also used as a MEK substitute. Water-based and 100 percent solid coating systems may also be substituted for MEK solvents. MEK is likely to remain in use, particularly in high quality applications, unless alternative systems are further developed (Ref. 4).

B. Metabolism and Absorption

MEK is well absorbed from the lung, gastrointestinal (GI) tract, and skin. Pulmonary uptake in humans ranged from 41 percent to 56 percent. Case reports in humans and/or studies in rats demonstrate that MEK is absorbed from the GI tract and the skin (Ref. 5).

C. Toxicological Evaluation

1. *Acute toxicity*. Available data indicate that MEK has low acute toxicity. In humans, inhalation of high doses produces irritation of the eyes and upper and lower respiratory system, effects characteristic of solvent exposure (Ref. 6).

2. *Subchronic and chronic toxicity*. Available data indicate that MEK has low chronic toxicity. Although no chronic exposure studies have been found, several well-designed repeated-dose oral and inhalation studies in laboratory animals demonstrate low systemic toxicity with MEK. The Occupational Safety and Health Administration (OSHA) Permissible Exposure Level (PEL) for MEK is 200 parts per million (ppm), or about 589 milligrams per cubic meter (mg/m^3). EPA's current RfC of 1.0 mg/m^3 (or approximately 968 milligrams per kilogram per day (mg/kg/day)) for MEK is based on a developmental toxicity study in mice (Refs. 6 and 7).

a. *Carcinogenicity*. MEK is classified in EPA's IRIS data base (Ref. 8) as category D, not classifiable as to human carcinogenicity, based on no human carcinogenicity data and inadequate animal data (Ref. 6).

b. *Mutagenicity*. There is a wealth of mutagenicity information on MEK submitted pursuant to section 4 of the Toxic Substances Control Act (TSCA). MEK was negative in the Ames assay with and without activation. It induced chromosome mutations (aneuploidy) in yeast cells. It also induced cell transformation in BALB/c cells. It was also negative in the UDS assay, for sister chromatid exchange (SCE's) in Chinese Hamster Ovary (CHO) cells, in the mouse micronucleus assay, for gene mutations in *E. coli*, in the mouse lymphoma assay, and for chromosome aberrations in CHO cells (Ref. 6).

c. *Developmental toxicity*. Not available at the time of the first petition on MEK, is an inhalation developmental toxicity study in Swiss mice. This is the key study, on which the RfC is based (Ref. 7). In the study, four groups of 10 virgin and 33 pregnant mice were exposed to 0, 398, 1,010, or 3,020 ppm (0, 1,174, 2,978, or 8,906 mg/m^3) MEK for 7 hours per day (hr/day) during gestation days 6-15. Neither maternal nor developmental toxicity was observed at the low or mid doses. At 3,020 ppm, there was a decrease in fetal body weight that was significant only in males and a significant trend in the incidence of misaligned sternebrae when measured on a fetus, but not litter basis. At this dose there was also an increase in maternal relative liver and kidney weight, but the biological significance of this effect is not known.

Based on the dose level at which these effects were observed, the concern for developmental toxicity appears to be low. The Lowest Observed Adverse Effect Level (LOAEL) is 3,020 ppm (approximately 2,898 mg/kg/day) and the No Observed Adverse Effect Level (NOAEL) is 1,010 ppm (968 mg/kg/day).

The two inhalation studies in rats that formed the basis of concern at the time of the first petition were both conducted by the same group of researchers and in the same laboratory. In the first study (Ref. 7), animals were exposed to MEK at 0, 1,126, or 2,618 ppm (0, 3,320, or 7,720 mg/m^3). At the low dose, there was a decrease in fetal body weight and crown:rump length; these effects were not seen at the high dose. There was also a significant increase in total number of litters containing fetuses with skeletal anomalies. At the high dose, there was a significant increase in number of fetuses and litters having gross anomalies. Maternal toxicity was not observed. The LOAEL from this study is 1,126 ppm.

The second study (Ref. 9) was conducted to determine the repeatability of the above findings. Exposures to MEK were 0, 412, 1,002, or 3,005 ppm (0, 1,215, 2,955, or 8,861 mg/m^3). No effects were seen at the low or mid dose. At the high dose, there was delayed ossification of bones in the skull and cervical centra and an increase in the incidence of extralumbar ribs. There was also decreased maternal body weight gain and increased water consumption at the high dose. The NOAEL from this study is 1,002 ppm, and the LOAEL is 3,005 ppm (Ref. 6).

d. *Reproductive toxicity*. Reproductive toxicity data on MEK could not be found. There is a two-generation rat study with 2-butanol (a metabolic precursor to MEK) in which Wistar rats (30/sex/group) were given 0, 0.3 percent, 1.0 percent, or 3.0 percent in drinking water (Ref. 10). Because of significant toxicity seen in the high-dose group, treatment of high-dose parents and offspring was reduced to 2.0 percent. The critical effect was decreased fetal birth weight at the 2.0 percent dose.

Based on the dose level at which these effects were observed, the concern for reproductive toxicity appears to be low. The LOAEL for 2-butanol is 2.0 percent (3,122 mg/kg/day) and the NOAEL is 1.0 percent (1,771 mg/kg/day) (Ref. 6).

e. *Neurotoxicity*. According to the latest IRIS report on MEK, which was updated in June 1993, ''at present, there is no convincing experimental evidence

that MEK is neurotoxic. . .other than possibly inducing central nervous system depression at high exposure levels'' (Ref. 8). The prior neurotoxicity concerns identified for MEK were based on enhancement of the neurotoxicity of other solvents, such as n-hexane, by MEK (Ref. 11).

f. *Toxicity related to ozone formation*. MEK 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, ozone can affect structure, function, metabolism, pulmonary defense against bacterial infection, and extrapulmonary effects (Ref. 12). 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*. MEK is toxic to aquatic organisms at relatively high concentrations. The fish 96–hour lethal concentration for 50 percent of the testing sample (LC_{50}) range from 2,300 to 3,220 ppm; the daphnid 48–hour LC_{50}s range from 2,200 to 5,091 ppm, and the green algal 96–hour effective concentration for 50 percent of the population (EC_{50}) is 1,200 ppm. The fish chronic values range from 220 to 300 ppm, the daphnid chronic value is 52 ppm, and the algal chronic value is 45 ppm. MEK's calculated bioconcentration factor, 0.640, is low (Ref. 13).

As a VOC, MEK contributes to the formation of ozone in the environment. As EPA has previously stated, 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 (Ref. 12). Based on the 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. Exposure Review

1. *Exposure assessment*. The available data indicate that MEK can cause chronic developmental toxicity at moderately high to high doses. Because there is a possibility that the developmental effects associated with exposures to relatively high concentrations of MEK could be caused by short-term exposures, an exposure assessment was conducted. The exposure assessment was conducted only to determine the potential for adverse chronic developmental effects to occur as a result of concentrations of MEK that are reasonably likely to exist beyond facility site boundaries as a result of continuous, or frequently recurring, releases from facility sites (Ref. 14). For a discussion of the use of exposure in EPCRA section 313 listing and delisting decisions, refer to the **Federal Register** of November 30, 1994 (Ref. 12).

MEK releases were retrieved from the Toxics Release Inventory System (TRIS) data base. There were 2,389 TRI reports submitted for MEK in 1994. Most of the industrial releases are to air. Total quantities released to air, water, and land in 1994 were 78,624,939 pounds, 108,163 pounds, and 51,794 pounds, respectively. Thus, since most releases of MEK are to air, only airborne exposures were considered. Furthermore, because the critical effect is developmental toxicity, which can be initiated upon acute exposure, acute ambient concentrations estimated by the Point Plume (PTPLU) model were the exposure concentrations examined.

This procedure generates estimates of concentrations and exposures under three different scenarios that include a variety of wind conditions, one of which is a relatively stagnant situation. These three scenarios have been labeled: (1) The typical scenario, (2) the stagnant scenario, and (3) the maximum scenario. The model does not consider decay of the chemical in the environment.

A combination of both conservative and non-conservative assumptions were used to generate the exposure estimates with the PTPLU model. The conservative assumptions include the use of weather station data known to generate the highest concentrations and therefore potential exposures, as well as the use of a 24–hour exposure duration. Non-conservative assumptions include the assumption that TRI releases are spread over 365 days per year, 24 hours a day, and a 24–hour averaging time for concentration estimates. Given a shorter release period, estimated exposures could be significantly higher.

Estimates of acute ambient concentrations resulting from stack releases from five discharging facilities range from 3.0 to 9.0 mg/m^3 for a ''typical'' scenario; 6.0 to 17.0 mg/m^3 for a ''stagnant'' (no wind) scenario; and, 37 to 103 mg/m^3 for the maximum scenario. Acute ambient concentrations resulting from fugitive releases from five discharging facilities range from 5.0 to 12 mg/m^3 for a typical scenario; 40.0 to 110 mg/m^3 for a stagnant scenario; and, 100 to 240 mg/m^3 for the maximum scenario (Ref. 14).

2. *Exposure evaluation*. The exposure estimates illustrated in this assessment utilize release information submitted under TRI and standard modeling techniques to derive ambient air concentrations of MEK under three release scenarios (typical, stagnant, and maximum or peak) for the top releasing facilities for each type of release, fugitive and stack. Release estimate data are evaluated as to whether they exceed an Agency accepted RfC or reference dose (RfD), respectively, or when appropriate, a Margin of Exposure (MOE).

The IRIS RfC for MEK is based on mild, but significant developmental toxicity (decreased fetal body weight and misaligned sternebrae). An RfC represents an estimate of a daily inhalation exposure of the human population that is likely to be without appreciable risk of deleterious effects during a lifetime. The RfC makes adjustments to account for uncertainties about portal of entry and long-term exposure effects. Because developmental effects are an endpoint of concern for this chemical, it would not be appropriate to use the RfC for assessing the potential risk of developmental toxicity associated with acute exposure to MEK because the RfC is set for long-term exposures. It would be appropriate to derive an RfC_{DT} and compare it to the estimated human exposure concentration; however, there is no official Agency RfC_{DT}. Therefore, a MOE approach was used. The rationale for following this approach is that developmental toxicity requires assessment of short-term exposures (Ref. 6).

A MOE calculation is used in instances of non-cancer endpoints and is essentially a ratio of the NOAEL and the estimated exposure to the particular chemical, including any modifying factors on the exposure. The resultant value is then compared to the product of the uncertainty factors which are selected for the chemical of interest. Uncertainty factors are generally factors of 10 with each factor representing a specific area of uncertainty in the available data. For MEK, a factor of 10 was used to account for the possible differences in responsiveness between humans and animals in prolonged exposure studies, and a second factor of 10 was used to account for variation in susceptibility among individuals in the human population. The resultant uncertainty factor of 100 was therefore used in this assessment (Ref. 6).

The calculated MOE includes the NOAEL (ca. 1,380 mg/kg/day) from the mouse developmental study divided by the acute estimated Average Potential

Dose Rates (APDRs). The MOE is greater than 100 for stack releases under all three scenarios typical, stagnant, and maximum. The MOE is greater than 100 for fugitive releases in all three scenarios except one discharging facility under stagnant scenarios. It should be noted that the exposure estimates are based on facility release estimates, which generally are not the result of monitoring studies. Also, the APDRs assume that the target population is exposed to ambient (outdoor) air continuously. Thus, the exposure characterization reflects potential concerns engendered by estimated high exposures. Using these assumptions, the assessment illustrated that exposure concentrations do not exceed the MOE, except for one scenario (Ref. 6).

In summary, based on the concentrations likely to exist beyond facility site boundaries and the resulting MOE calculations, there is low concern for a potential for developmental effects for the general population as a result of direct toxicity following acute inhalation exposures to MEK. Furthermore, based on the developmental effects observed, if the MOE were calculated on the basis of a benchmark dose instead of the apparent NOAEL from the developmental toxicity study, the concern for potential developmental effects would be further weakened, if not eliminated. Therefore, under the exposure conditions described here, there appears to be low potential for developmental effects associated with exposure to MEK (Ref. 6).

IV. Summary of Technical Review

The hazard assessment strongly indicates that, except for VOC concerns, MEK has low acute and chronic (systemic) toxicity in that effects occur only at high doses. Specifically, developmental toxicity for MEK is characterized by high dose effects and lack of consistency between studies for one species. The exposure assessment, conducted only for developmental effects, indicates a low potential for these effects to occur from reported releases of MEK from TRI facilities under the conditions modeled. Thus, based on EPA's modeling, TRI reported releases of MEK are not expected to be sufficient to cause the type of high dose developmental effects associated with MEK. The available data do indicate that MEK 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) criterion. MEK has low direct environmental toxicity. MEK 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.

V. Rationale for Denial

EPA is denying the petition submitted by the Ketones Panel of the CMA to delete MEK from the EPCRA section 313 list of toxic chemicals. This denial is based on EPA's conclusion that VOCs, such as MEK, 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 MEK meets the listing criteria of EPCRA section 313(d)(2)(B) and (C) because MEK 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 (C) (59 FR 61432, November 30, 1994). EPA has stated in prior **Federal Register** notices (54 FR 4072, January 27, 1989; 54 FR 10668, March 15, 1989; 59 FR 49888, September 30, 1994; and 60 FR 31643, June 16, 1995) that because VOCs contribute to the formation of tropospheric ozone they meet the criteria for listing under EPCRA section 313. 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. MEK is a VOC with both a high production volume and high air emissions. Therefore, EPA has determined that MEK should remain on the EPCRA section 313 list of toxic chemicals. EPA intends to provide further clarification of its EPCRA section 313 VOC policy in a future **Federal Register** notice.

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 necessary to conclude that MEK, since it contributes to the formation of ozone, meets the toxicity criteria of EPCRA section 313(d)(2)(B) and (C).

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). 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 two-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.

VI. References

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

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

3. Kirk-Othmer Encyclopedia of Chemical Technology, 3rd. Edition, Vol. 13 (1981), Vol. 21 (1983), and 4th Edition, Vol. 4 (1992), John Wiley Sons, New York.

4. USEPA, OPPT. Wise, Sherry; ''Economic Analysis of the Proposed Deletion of Methyl Ethyl Ketone from the EPCRA 313 List of Toxic Chemicals.'' (February 10, 1997).

5. USEPA, OPPT. Keifer, Leonard; ''Absorption Review for Methyl Ethyl Ketone (MEK).'' (January 22, 1997).

6. USEPA, OPPT. Hernandez, Oscar; ''Health Hazard Assessment: Delist Petition for MEK.'' (June 1, 1997).

7. Schwetz, B.A., et al., ''Developmental Toxicity of Inhaled Methyl Ethyl Ketone in Swiss Mice.'' Fund. and Appl. Toxicol. v. 16 (1991), pp. 742-748.

8. IRIS, 1993. U.S. Environmental Protection Agency's Integrated Risk Information System file pertaining to methyl ethyl ketone.

9. Deacon, M.M., M.D. Pilny, J.A. John, et al., ''Embryo- and Fetotoxicity of Inhaled Methyl Ethyl Ketone in Rats.'' Toxicol. Appl. Pharmacol. v. 59. (1981), pp. 620-622.

10. Cox et al, 1975. ''Toxicity Studies in Rats with 2-Butanol Including Growth, Reproduction and Teretologic Observations.'' Food and Drug Research Laboratories, Inc. Rpt. 91MR-R 1673.

11. USEPA, OPPT. Memorandum from Lois Dicker, Ph.D., Chief, Existing Chemicals Assessment Branch, Risk Assessment Division. Subject: Review of the Interactive Effects of Methyl Ethyl Ketone (MEK) with Neurotoxic Solvents: Response to OSHA/NIOSH Comments. (October 6, 1997).

12. USEPA. ''Addition of Certain Chemicals.'' Proposed rule, (59 FR 1788, January 12, 1994).

13. USEPA, OPPT. Nabholtz, J.V.; ''Delisting Petition for Methyl Ethyl Ketone: Environmental Toxicity.'' (December 10, 1996).

14. USEPA, OPPT. Powers, Mary; ''Exposure Assessment for Methyl Ethyl Ketone.'' (June 2, 1997).

VII. Administrative Record

The record supporting this decision is contained in docket control number OPPTS–400119. All documents, including the references listed in Unit VI. of this document 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

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

Dated: March 19, 1998.

Lynn R. Goldman,

Assistant Administrator for Prevention, Pesticides and Toxic Substances.

[FR Doc. 98–8208 Filed 3–27–98; 8:45 am]

BILLING CODE 6560–50–F

EXPORT-IMPORT BANK OF THE UNITED STATES

Notice of Open Special Meeting of the Advisory Committee of the Export-Import Bank of the United States (Export-Import Bank).

SUMMARY: The Advisory Committee was established by Public Law 98–181, November 30, 1983, to advise Export-Import Bank on its programs and to provide comments for inclusion in the reports of the Export-Import Bank of the United States to Congress.

TIME AND PLACE: Tuesday, April 14, 1998, at 9:30 a.m. to 3:15 p.m. The meeting will be held at the Export-Import Bank in room 1143, 811 Vermont Avenue, NW, Washington, D.C. 20571.

AGENDA: The meeting will include a discussion of the following: the capacity of commercial banks to step up to some risk in the medium term in order to set the stage for the use of delegated authority; the availability of information from the exporter community on the net employment impact of a change in the foreign content policy; the ability of financial intermediaries in project finance cases to take on operational and risk-sharing roles that neutralize the administrative and program budget implications of offering pre-completion comprehensive cover; and the adequacy of short- and medium-term export credit availability for small and medium sized exporters and what additional delivery mechanisms might expand the availability of such support.

PUBLIC PARTICIPATION: The meeting will be open to public participation, and the last 10 minutes will be set aside for oral questions or comments. Members of the public may also file written statement(s) before or after the meeting. In order to permit the Export-Import Bank to arrange suitable accommodations, members of the public who plan to attend the meeting should notify Megan Becher, Room 1284, 811 Vermont Ave., NW, Washington, DC 20571, (202) 565–3507, no later than April 6, 1998. If any person wishes auxiliary aids (such as a sign language interpreter) or other special accommodations, please contact, prior to April 6, 1998, Megan Becher Room 1284, Vermont Avenue, NW, Washington, DC 20571, Voice: (202) 565–3955 or TDD (202) 565–3377.

FURTHER INFORMATION: For further information, contact Megan Becher, Room 1284, 811 Vermont Ave., NW, Washington, DC 20571, (202) 565–3507.

Kenneth Hansen,

General Counsel.

[FR Doc. 98–8225 Filed 3–27–98; 8:45 am]

BILLING CODE 6690–01–M

FEDERAL COMMUNICATIONS COMMISSION

Notice of Public Information Collection(s) Submitted to OMB for Review and Approval

March 23, 1998.

SUMMARY: The Federal Communications Commission, as part of its continuing effort to reduce paperwork burden invites the general public and other Federal agencies to take this opportunity to comment on the following information collection(s), as required by the Paperwork Reduction Act of 1995, Pub. L. 104–13. An agency may not conduct or sponsor a collection of information unless it displays a currently valid control number. No person shall be subject to any penalty