Dakota Department of Agriculture and the Minnesota Department of Agriculture (hereafter referred to as the ''Applicants'') to use the pesticide benomyl (CAS 17804–35–2) (formulated as ''Benlate Fungicide'') for the control of *Sclerotinia* stem rot in canola. A maximum of 60,000 acres in North Dakota, and a maximum of 10,500 acres in Minnesota could be treated. The Applicants propose the use of a pesticide which contains an active ingredient which has been the subject of a Special Review, and is intended for a use that could pose similar risks to the risks posed by the uses that were the subject of the Special Review. In accordance with 40 CFR 166.24, EPA is soliciting public comment before making the decision whether or not to grant the exemptions.

DATES: Comments must be received on or before May 22, 1997.

ADDRESSES: Three copies of written comments, bearing the identification notation ''OPP–181045,'' should be submitted by mail to: Public Response and Program Resource Branch, Field Operations Division (7506C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In person, bring comments to: Rm. 1132, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA.

Comments and data may also be submitted electronically by following the instructions under ''SUPPLEMENTARY INFORMATION.'' No Confidential Business Information (CBI) should be submitted through e-mail.

Information submitted in any comment concerning this notice may be claimed confidential by marking any part or all of that information as CBI. Information so marked will not be disclosed except in accordance with procedures set forth in 40 CFR part 2. A copy of the comment that does not contain CBI must be provided by the submitter for inclusion in the public record. Information not marked confidential may be disclosed publicly by EPA without prior notice. All written comments filed pursuant to this notice will be available for public inspection in Room 1132, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA, from 8:30 a.m. to 4 p.m., Monday through Friday, except legal holidays.

FOR FURTHER INFORMATION CONTACT: By mail: Olga Odiott, Registration Division (7505W), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460; Office location, telephone number and e-mail: Sixth floor, Crystal Station #1, 2800 Jefferson Davis Highway, Arlington, VA, (703) 308–6418; e-mail: odiott.olga@epamail.epa.gov.

SUPPLEMENTARY INFORMATION: Pursuant to section 18 of the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) (7 U.S.C. 136p), the Administrator may, at her discretion, exempt a state agency from any registration provision of FIFRA if she determines that emergency conditions exist which require such exemption. The Applicants have requested the Administrator to issue specific exemptions for the use of benomyl on canola to control the *Sclerotinia* stem rot. Information in accordance with 40 CFR part 166 was submitted as part of the requests.

The Applicants state that the last 4 years have been favorable to the buildup of *Sclerotinia* in the soil, and that experience with other crops indicates the *Sclerotinia* levels are sufficiently high to place the canola crop in a highly vulnerable position if a rainy period occurs when the crop is flowering. The Applicants state that canola growers will likely suffer severe economic losses since there are no registered alternative pesticides available and the fungus has become sufficiently widespread that crop rotation will be of limited effectiveness in the major canola producing areas.

The Applicants propose to make a single aerial application of benomyl at a rate of 0.5 lbs. active ingredient (a.i.) per acre during the 20 to 30 percent bloom stage. The need for application of the fungicide will be determined by the weather in the weeks prior to bloom and the yield potential. The proposed use is for up to 60,000 acres of canola in North Dakota, and 10,500 acres of canola in Minnesota. Therefore, use under these exemptions could potentially amount to a maximum total of 35,250 lbs. of the active ingredient, benomyl (30,000 in North Dakota and 5,250 in Minnesota). Emergency exemptions for this use were granted to North Dakota in 1989 thru 1992.

This notice does not constitute a decision by EPA on the application itself. The regulations governing section 18 require publication of a notice of receipt in the **Federal Register** for an application for a specific exemption proposing the use of a pesticide which contains an active ingredient which has been the subject of a Special Review, and is intended for a use that could pose similar risks to the risks posed by the uses that were the subject of the Special Review. Such notice provides for opportunity for public comment on the application.

The official record for this notice, as well as the public version, has been established for this notice under docket number [OPP–181045] (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 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The official notice record is located at the address in ''ADDRESSES'' at the beginning of this document.

Electronic comments can be sent directly to EPA at:

opp-docket@epamail.epa.gov

Electronic comments must be submitted as an ASCII file avoiding the use of special characters and any form of encryption. Comment 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 [OPP–181045]. Electronic comments on this notice may be filed online at many Federal Depository Libraries.

The Agency will review and consider all comments received during the comment period in determining whether to issue the emergency exemptions requested by the North Dakota Department of Agriculture and the Minnesota Department of Agriculture.

List of Subjects

Environmental protection, Pesticides and pests, Emergency exemptions.

Dated: April 23, 1997.

Stephen L. Johnson,

Director, Registration Division, Office of Pesticide Programs.

[FR Doc. 97–11634 Filed 5–6–97; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[OPPTS–400110; FRL–5598–8]

Ethylene Glycol; Toxic Chemical Release Reporting; Community Right-to-Know

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: EPA is issuing the results of its technical review and evaluation of a petition to delete ethylene glycol from the list of toxic chemicals subject to the reporting requirements under section 313 of the Emergency Planning and

Community Right-to-Know Act (EPCRA) and section 6607 of the Pollution Prevention Act of 1990 (PPA). Since the petition to delete ethylene glycol was withdrawn on October 28, 1996, there is no need for final action by the Agency. However, the Agency has decided to issue its findings in order to make publicly available the technical review and subsequent scientific conclusion.

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

Section 313 of the Emergency Planning and Community Right-to-Know Act (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. Ethylene glycol was included on the initial EPCRA section 313 list of toxic chemicals. 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 61432, November 30, 1994) (FRL–4922–2).

II. Description of the Petition

On March 21, 1994, Bonded Products, Inc. petitioned the Agency to delist ethylene glycol from the list of toxic chemicals subject to reporting under section 313 of EPCRA and section 6607 of PPA. The Bonded Products petition was based on the contention that: ethylene glycol is biodegradable, rapidly loses its toxicity and, therefore, is not expected to cause adverse environmental, or acute or chronic health effects; and, that releases from the consumer use of ethylene glycol are likely to be significantly higher compared to releases from manufacturing facilities. The petitioners argued that ethylene glycol does not meet any of the EPCRA section 313(d)(2) criteria for listing. EPA staff reviewed the petition based on information and data that the Agency retrieved from its own review of the literature, as well as information supplied by other interested parties. On October 28, 1996, Bonded Products withdrew their petition.

The review of Bonded Products, Inc.'s petition was complete prior to their request for withdrawal, and the Agency has determined that it is in the public's best interest and clearly in keeping with the Community-Right-to-Know ethic to provide a summary of the chemical review and conclusion. Bonded Products, Inc. or any other party may re-petition the Agency on ethylene glycol at any time. The Agency remains open to receiving and reviewing new information and re-evaluating its position on this chemical as it relates to section 313 of EPCRA.

III. Technical Review of Ethylene Glycol

The technical review of the petition to delete ethylene glycol from the EPCRA section 313 list of toxic chemicals included an analysis of the relevant chemistry, metabolism and absorption, toxicity, and exposure data available to the Agency for ethylene glycol. Summaries of the analysis of each of these areas is provided in Units III.A. through III.F. of this preamble, and a more complete discussion of this information can be found in the EPA documents prepared for this assessment (Refs. 1-14), which have been placed in the public docket for this petition (Docket OPPTS-400110).

A. Chemistry, Use, and Production Profile

Ethylene glycol is a colorless, odorless, syrupy liquid with a sweet taste. It has a relatively high boiling point (197.6 °C), flash point (116 °C), autoignition temperature (412.93 °C), and is relatively non-volatile at room temperature (Ref. 1). Ethylene glycol absorbs water and can take up twice its weight of water at 100 percent relative humidity. Additionally, the substance reduces the freezing point of water and is widely used as an antifreeze and deicer.

Ethylene glycol is generally produced by the noncatalytic, liquid phase hydration of ethylene oxide (Ref. 1). Diethylene glycol, triethylene glycol and tetraethylene glycol are co-products. Other processes have been patented such as: (1) oxidation of ethylene in an aqueous medium using an iron-copper catalyst; and (2) rhodium-catalyzed production of ethylene glycol from synthesis gas (a mixture of carbon monoxide and hydrogen from coal gasification) instead of ethylene.

There were 2.3 billion kilograms of ethylene glycol produced in 1992 and production has been fairly steady since the early 1980's (Ref. 2). Domestic consumption was 2.1 billion kilograms. The major end use of ethylene glycol is in the production of polyethylene terephthalate (PET), with 30 percent used for fibers and 22 percent used for films, bottles, and other molded plastics, laminates, and castings (Ref. 2). An additional 38 percent of ethylene glycol production is used in antifreeze application, such as the principle ingredient of all-weather automobile cooling system fluids, deicing solutions for aircraft and pavement, and in fire extinguishers and sprinkler systems. The remaining 10 percent of demand is in miscellaneous applications such as a diluent and coupler in cutting fluids, as a solvent or coupling agent for stains, dyes, resins, inks, soluble oils, and hydraulic fluids. It is also used as a component in the manufacture of polyester laminating resins and other plastics.

B. Metabolism and Absorption

Ethylene glycol itself appears to have relatively low toxicity, but it is oxidized to a variety of more toxic metabolites such as glycolaldehyde, glycolic acid, glyoxalic acid and oxalic acid (Ref. 6). In general, the accumulation of these acids leads to acidosis (the state that is characterized by actual or relative decrease of alkali in body fluids in relation to the acid content). Present information suggests that glycolic acid is the major toxic metabolite contributing to metabolic acidosis, which is the underlying cause of systemic toxicity following exposure to ethylene glycol.

Based on a comparison of metabolism studies, ethylene glycol appears to be less well absorbed following dermal application than following administration via oral gavage (Ref. 10). In addition, even when an ethylene glycol aerosol is generated to maximize the amount available for inhalation, the body burden remains fairly low. In the study by Frantz et al. (Ref. 15), ethylene glycol and its metabolites (glycolic acid and oxalic acid) were excreted in the urine of animals dosed both orally and dermally. In contrast, the study by Marshall and Cheng (Ref. 16) showed that after inhalation exposure to ^{14}C-labeled ethylene glycol, the only ^{14}C-containing material identified in the plasma and urine (both for the aerosol and vapor) was unmetabolized ethylene glycol.

C. Human Toxicity Evaluation

The inherent toxicity of ethylene glycol is low relative to several of its metabolites. The evidence for this comes from clinical studies and laboratory investigations (Ref. 4). Ethanol is a competitive inhibitor of alcohol dehydrogenase (ADH), the first enzyme in the ethylene glycol metabolic pathway, and is very effective in treating animal and human ethylene glycol poisonings. If treatment is started early enough, the metabolic acidosis and renal failure discussed below can be prevented.

1. *Inhalation toxicity*. Two inhalation developmental toxicity studies have been conducted by the same group (Refs. 17 and 18). In a whole body exposure study (Ref. 17), mice and rats were exposed to ethylene glycol aerosols of 150, 1,000 or 2,500 milligrams per cubic meter (mg/m^3) for 6 hours/day on gestational days 6 through 15. The actual measured concentrations were 119, 888, or 2,090 mg/m^3. In rats, maternal toxicity occurred only at the highest concentration and was indicated by a significant increase in absolute and relative liver weight. In rats, evidence of prenatal developmental toxicity (reduced ossification in the humerus, zygomatic arch, and the metatarsals and proximal phalanges of the hindlimb) was observed at the two higher concentrations. In mice, incidences of prenatal developmental toxicity were increased at the two highest concentrations and included malformations in the head (exencephaly), face (cleft palate, foreshortened and abnormal face, and abnormal facial bones), and skeleton (vertebral fusions, and fused, forked, and missing ribs). The No Observed Adverse Effect Level (NOAEL) for maternal toxicity in rats was 888 mg/m^3 and in mice was 119 mg/m^3. The NOAEL for developmental toxicity in rats was 119 mg/m^3 and in mice was below this concentration.

A major confounding factor in this study was the deposition of a detectable quantity of ethylene glycol upon the animals during exposure. The animals could have received the chemical via the oral route by preening or by dermal absorption, although much less would be taken in via the skin. Analysis of the chemical on the fur of rats and mice after the exposure period at the highest concentration indicated that much of the chemical dose (65-95 percent) was potentially derived from ingestion after grooming.

To address the potential confounding factor of multiple exposure routes cited above, a further study used nose-only exposure of mice to 500, 1,000, and 2,500 mg/m^3 of ethylene glycol aerosol for 6 hours/day on gestational days 6 through 15 (Ref. 18). Results from the positive control (whole body exposure to 2,100 mg/m^3) confirmed the results from the previous study. In the nose-only portion, the two higher concentrations produced increased kidney weights in the dams. At the highest concentration, fetal weights were reduced and fetal skeletal variations and one fetal skeletal malformation (fused ribs) were increased. The developmental NOAEL for nose-only inhalation exposure was 1,000 mg/m^3; the maternal NOAEL was 500 mg/m^3. The developmental NOAEL in this study was at least 10 times the whole body value since a NOAEL was not established in the previous whole body inhalation study but was less than 119 mg/m^3. The maternal NOAEL was approximately five times the previous value. This nose-only exposure study indicates that most of the adverse effects seen in the whole-body exposure study were due to systemic exposure from noninhalation routes; however, as discussed above, adverse effects were seen in the nose-only exposure study.

The toxicity data strongly indicate that ethylene glycol is much less toxic than its metabolites; however, it is not known if ethylene glycol might act directly on embryos. The available literature does not provide adequate data to allow definitive conclusions concerning ethylene glycol's toxicity to embryos (Ref. 4).

2. *Oral toxicity*. Ethylene glycol is expected to be absorbed through the skin and from the lung and the gastrointestinal tract. After absorption, it is expected to enzymatically oxidize to oxalic acid, glycolic acid, glycolaldehyde and carbon dioxide. The aldehyde metabolites are believed to be responsible for neurotoxicity and the oxalic acid metabolites for renal toxicity (Ref. 8).

a. *Renal toxicity*. The oral reference dose (RfD) for ethylene glycol as established by the Agency's RfD/RfC (reference concentration) working group is 2 milligrams per kilogram per day (mg/kg/day). An RfD reflects the Agency's estimate of a level of daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime (Ref. 19). The RfD for ethylene glycol is based on a feeding study by DePass et al. (1986, as cited in EPA's Integrated Risk Information System (IRIS), 1994; Ref. 20) in which the critical effect was kidney toxicity. Groups of male and female rats (30/sex/group) and male and female mice (20/sex/group) were fed diets containing ethylene glycol at doses of 0, 40, 200, or 1,000 mg/kg/day for 2 years. Urinary calcium oxalate crystals and increased kidney weight were seen in all high-dose rats. Histopathologic changes in high-dose male rats included tubular cell hyperplasia, tubular dilation, peritubular nephritis, parathyroid hyperplasia, and generalized soft tissue mineralization. No adverse effects were seen in rats of either sex at the mid or the low doses. There were no adverse effects seen in mice of either sex at any dose tested. The Lowest Observed Adverse Effect Level (LOAEL) was determined to be 1,000 mg/kg/day and the NOAEL was 200 mg/kg/day. The RfD was set with an uncertainty factor of 100, 10 for interspecies extrapolation and 10 for differences in human sensitivity. Confidence in the study, the uncertainty factor and the RfD was high.

b. *Developmental/reproductive toxicity*. IRIS includes a review of several developmental reproductive studies with LOAELs at or near that seen in the DePass study which was

used to set the oral RfD. These studies were not chosen as the basis for the RfD since the LOAEL from the DePass study was somewhat lower and the RfD was deemed protective of developmental effects. In a 3-generation reproduction study, Lamb, as cited in IRIS (Ref. 20), treated rats with 0, 40, 200, or 1,000 milligrams per kilogram (mg/kg) in the diet and found no treatment related effects. In another study cited in IRIS (Ref. 20), ethylene glycol was administered by gavage at 0, 50, 150, 500 or 1,500 mg/kg to 30 pregnant female CD-1 mice/group on gestation days 6-15. Animals were sacrificed on gestation day 18 and examined for signs of maternal and developmental toxicity. There was an increase in skeletal abnormalities at both 500, and 1,500 mg/kg. A No Observed Effect Level (NOEL) was established at 150 mg/kg for developmental toxicity with a Lowest Observed Effect Level (LOEL) of 500 mg/kg.

c. *Oncogenicity/carcinogenicity/mutagenicity*. There is no evidence that ethylene glycol is oncogenic or that it is a mutagen (Ref. 8).

d. *Acute toxicity*. Ethylene glycol is acutely toxic to humans; the minimum lethal ingested dose for adults is approximately 1.4 milliliters per kilogram (ml/kg) or 100 ml for a 70 kg person (Ref. 8). Signs of ethylene glycol poisoning can be divided into three stages. Stage one includes central nervous system (CNS) disturbances and gastrointestinal symptoms. Stage two includes signs of cardiovascular, pulmonary, and metabolic irregularities and stage three includes renal failure brought on by the precipitation of calcium oxalate crystals in renal tubules and from the direct toxic action of oxalic and glycolic acids upon the kidneys (Ref. 8).

D. Environmental Toxicity

Ethylene glycol appears to represent a low hazard to the environment (Refs. 8 and 11). The freshwater aquatic toxicity data range from a median effective concentration (EC_{50}) of 4.4 grams per milliliter (g/ml) (duckweed) to a median lethal concentration (LC_{50}) of 111 g/ml (bluegill sunfish). Terrestrial toxicity data range from a median lethal dose (LD_{50}) of 1.65 grams per kilogram (g/kg) for cats to 5.5 g/kg for dogs and 12 g/kg for mice.

Reports of animal poisonings that were reviewed, were the results of accidental or intentional releases during consumer use. They were not the result of environmental exposures that may result from releases of ethylene glycol that are reasonably likely to come from TRI reporting facilities under normal operating conditions.

E. Exposure Assessment

Ethylene glycol can be acutely toxic to humans. Therefore, an assessment was conducted of the potential for adverse acute human health effects to occur as a result of concentrations of ethylene glycol that are reasonably likely to exist beyond facility site boundaries as a result of continuous, or frequently recurring, releases from facility sites (Refs. 5, 6, and 13). As discussed above in Unit III.C. of this preamble, ethylene glycol produces adverse chronic health effects only at relatively high doses and thus has low chronic toxicity. Therefore, an exposure assessment was also conducted for chronic health effects (Refs. 5, 6, and 21). For a discussion of the use of exposure in EPCRA section 313 listing/delisting decisions, refer to the **Federal Register** of November 30, 1994.

Ethylene glycol releases reported for 1992 were retrieved from the Toxic Release Inventory System (TRIS) data base. The TRIAIR model, the Office of Pollution Prevention and Toxics' (OPPT) program for assessing releases of TRI chemicals to the atmosphere, was used to estimate chronic concentrations and exposures resulting from releases of ethylene glycol. The Point Plume (PTPLU) model was used to derive estimates of acute concentrations and exposures resulting from atmospheric releases. The TRIAIR model assumes a 99.9 percent destruction efficiency for all releases that are reported as sent to incinerations. A half-life of 22.6 hours in the atmosphere was used for ethylene glycol in the assessment. Ethylene glycol is quite biodegradable, but is not readily sorbed, volatilized, or hydrolyzed (Ref. 6).

According to the 1992 releases obtained from TRIS, over 11.7 million pounds of ethylene glycol are released per year by about 940 facilities nationwide. Data from the Aerometric Information Retrieval System (AIRS) Facility Subsystem were also considered. Based on review of AIRS and the type of data available for ethylene glycol, it was determined that the data for ethylene glycol are not adequate to support an exposure assessment.

Eighteen states each discharging over 100,000 pounds per year accounted for 93 percent of the total reported releases of ethylene glycol to the atmosphere. These releases were used for chronic exposure estimations. Each of the highest per-site discharges were used to estimate concentrations and exposures under acute conditions.

Concentrations modeled with the PTPLU model can be expected to occur up to 250 meters from the source, which may be beyond the facility fenceline. The PTPLU model provides ground-level concentrations which are hourly average values. Incorporating wind conditions, three scenarios were generated: (1) The typical situation; (2) the stagnation situation; and (3) the maximum situation. The maximum scenario is anticipate to last for only 2 hours, as compared with the 24-hour duration of the typical and stagnation scenarios. As the name implies, the stagnation scenario incorporates relatively little air movement. Each scenario was run for stack releases and for fugitive releases. Assumptions made were conservative on the whole. However, the assumption that releases occur over 365 days and 24 hours a day is not conservative. If, for example, releases occurred over only 1 month, even with 24-hour a day discharge, the resulting exposure estimates would increase by a factor of 12 or one order of magnitude.

F. Exposure Evaluation

1. *Chronic inhalation exposure*. In evaluating chronic inhalation exposures, ideally, exposure estimates would be compared to an RfC. However, in this case chronic inhalation information is neither readily available nor abundant, so an RfC has not been derived for ethylene glycol. In general, the oral RfD should not be used to evaluate inhalation exposures to ethylene glycol because it appears that the metabolism via the two routes is different. Specifically, this is demonstrated by the lack of toxic metabolites of ethylene glycol found in the urine and plasma of animals dosed via inhalation. Additionally, it is believed that the proximate cause for the toxicity seen from ethylene glycol is not attributed to the chemical itself but rather to its metabolites. Therefore use of the oral RfD would tend to be overly protective for inhalation effects from exposure to ethylene glycol. If, however, the evaluation of the chronic exposure data indicates that concentrations are below the RfD value, then the likelihood of concentrations of concern existing for inhalation effects is greatly diminished. For these reasons, the chronic exposures predicted were compared to the oral RfD of 2 mg/kg/day. The comparison showed that even the highest chronic exposures predicted for the chemical are, at a minimum, an order of magnitude below the RfD. Therefore, it is not predicted that concentrations of concern will exist for chronic inhalation exposures to ethylene glycol as a result

of releases from TRI reporting facilities (Ref. 6).

2. *Acute inhalation exposure.* Although the oral RfD was used to assess chronic inhalation exposures it was not used to assess acute inhalation exposures. This is because oral RfDs are based on the assumption of lifetime exposure (i.e., long-term exposure) and in most cases are not appropriately applied to less-than-lifetime exposure situations such as acute inhalation exposures. In addition, as discussed above, it appears that ethylene glycol metabolism is different via the oral and inhalation routes of exposure. Therefore, instead of using the RfD, the acute inhalation assessment focused on the generation of Margin of Exposure (MOE) calculations for inhalation exposures. A MOE calculation is used in instances of non-cancer endpoints and is essentially a ratio of the NOAEL or LOAEL and the estimated exposure to the particular chemical, including any modifying factors on the exposure (absorption, etc.). 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 ethylene glycol, a factor of 10 was introduced 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. This assessment focused on maternal and developmental toxicity, which EPA believes are the most significant adverse chronic effects caused by ethylene glycol. For the generation of MOEs used in this assessment the NOAELs from the Tyl study (Ref. 18) were utilized.

MOEs calculated from estimated stack emissions were below the relevant uncertainty factors for the top two releasers for all exposure scenarios for maternal toxicity. For developmental toxicity, MOEs below the relevant uncertainty factors were calculated for the stagnant and maximum exposure scenarios. MOEs calculated from fugitive releases under the stagnant condition were also below the relevant uncertainty factors for the top five releasers for both maternal and developmental toxicity. A similar situation was observed under the maximum scenario for maternal toxicity. Two things should be noted about the calculated MOEs. The first is that all exposure estimates were driven by facility specific data reported as required under EPCRA section 313. These estimates are considered within the realm of possibility, although are characterized as ''what if'' scenarios. These ''what if'' scenarios provide a possible exposure level, without probability and are not based on bounding or worst-case conditions which fall outside the exposure curve. Second, there is limited information to suggest that no metabolites are formed when ethylene glycol is inhaled. Since the toxicity data indicates that the metabolites of ethylene glycol are much more toxic than ethylene glycol itself, this normally would greatly reduce the concern for inhalation exposure to this chemical. However, adverse effects were noted in the 1995 Tyl study (Ref. 18) with nose-only exposure in rodents, which indicates that ethylene glycol is toxic via the inhalation route of exposure. Therefore, the resultant NOAELs from that study were utilized in this acute inhalation exposure assessment. Further, 100 percent of the inhaled dose of ethylene glycol is assumed to be absorbed.

In summary, based on the concentrations likely to exist beyond facility site boundaries and the resulting MOE calculations, there is a potential for chronic maternal and developmental effects for the general population following acute inhalation exposures to ethylene glycol (Ref. 6).

3. *Acute and chronic oral exposures.* The potential dose rates predicted for surface water driven oral exposures are identified as bounding estimates and are, therefore, likely to be much higher than actual exposures. Using the highest potential dose rate identified in the exposure assessment of 80 mg/day and dividing by 70 kg (standard assumption for body weight), a modified dose of 1.143 mg/kg/day was calculated. This dose is below the RfD of 2 mg/kg/day indicating that the exposure estimated is not likely to be associated with adverse chronic health risks (Refs. 6 and 21).

None of the exposure data indicates that ethylene glycol will be present beyond facility site boundaries at concentrations that can reasonably be anticipated to cause the adverse acute human health effects discussed under Unit III.C.2.d. of this preamble (Refs. 6 and 13). Therefore, it is unlikely that adverse acute human health effects are reasonably likely to occur as a result of concentrations likely to exist beyond facility site boundaries as a result of continuous, or frequently recurring, releases of ethylene glycol.

G. Summary of Technical Review

The data indicate that, based on the doses required to cause adverse effects, ethylene glycol has low chronic and acute toxicity to humans both orally and by inhalation. The exposure analysis indicates that ethylene glycol cannot reasonably be anticipated to cause significant adverse acute human health effects at concentration levels that are reasonably likely to exist beyond facility site boundaries as a result of continuous, or frequently recurring, releases from facility sites. The analysis of ethylene glycol's chronic toxicity concluded that ethylene glycol can reasonably be anticipated to cause chronic maternal and developmental effects in humans at relatively high doses. It was also determined that concentrations of ethylene glycol that are reasonably likely to exist beyond facility site boundaries as a result of acute exposure scenarios are reasonably likely to be sufficient to cause these chronic maternal and developmental effects. Based on available literature, ethylene glycol represents a low hazard to the environment and is not anticipated to cause environmental toxicity as a result of reported releases of ethylene glycol from facility sites.

IV. Explanation

Since the petition to delete ethylene glycol has been withdrawn by Bonded Products, Inc. EPA has no statutory responsibility to deny or grant the initial request. However, because the technical review and evaluation of the petition are complete, EPA determined that it is in the public's best interest, and clearly in keeping with the Community Right-to-Know ethic, to provide the public with a summary of EPA's review and conclusion. Based on the technical review discussed above, EPA concluded that this petition be denied based on concerns for chronic maternal and developmental effects for the general population following acute inhalation exposure from reported air releases of ethylene glycol. EPA believes that ethylene glycol meets the toxicity criteria of EPCRA section 313(d)(2)(B) based on the available chronic maternal and developmental toxicity data and the exposure analysis.

V. References

1. USEPA, OPPT. Tou, Jenny; ''Chemistry Report on Ethylene Glycol, EPCRA Section 313 Delisting Petition.'' (June 1, 1994).

2. USEPA, OPPT. Krueger, Susan; ''Economic Analysis of the Proposed Delisting of Ethylene Glycol from the

EPCRA Section 313 Toxic Release Inventory.'' (May 16, 1994).

3. USEPA, OPPT. Memorandum from Pat Jennings, Exposure Assessment Branch, Economics, Exposure, and Technology Division. Subject: Summary of the Environmental Fate of Ethylene Glycol. (May 20, 1994).

4. USEPA, OPPT. Memorandum from Mary Henry, Health Effects Branch, Health and Environmental Review Division. Subject: Ethylene Glycol Petition. (August 3, 1995).

5. USEPA, OPPT. Memorandum from Patricia Harrigan, Exposure Assessment Branch, Economics, Exposure, and Technology Division. Subject: Expanded Exposure Assessment for Ethylene Glycol. (June 19, 1995).

6. USEPA, OPPT. Memorandum from Linda M. Rusak, Hazard Integrator, Analysis and Information Management Branch, Chemical Screening and Risk Assessment Division. Subject: Petition to Delist Ethylene Glycol from TRI. (September 6, 1995).

7. USEPA, OPPT. Memorandum from Leonard C. Keifer, Chemist, Health Effects Branch, Health and Environmental Review Division. Subject: Metabolism of Ethylene Glycol. (March 29, 1995).

8. USEPA, OPPT. Memorandum from Angela Auletta, Chief, Health Effects Branch, Health and Environmental Review Division. Subject: Petition to Delist Ethylene Glycol from the Toxic Chemical Release Inventory. (May 23, 1994).

9. USEPA, OPPT. Memorandum from Mary Henry, Health Effects Branch, Health and Environmental Review Division. Subject: Review of Developmental Toxicity Studies with Ethylene Glycol. (March 24, 1995).

10. USEPA, OPPT. Memorandum from Leonard C. Keifer, Chemist, Health Effects Branch, Health and Environmental Review Division. Subject: Review of Absorption/ Metabolism Study for Ethylene Glycol Administered via Inhalation and Comparison with Results from Dosing via Oral Gavage and Dermal Administration. (August 1, 1995).

11. USEPA, OPPT. Memorandum from J. V. Nabholz, Health and Environmental Review Division. Subject: Ethylene Glycol [107-21-1]: Wildlife Poisoning. (December 5, 1995).

12. USEPA, OPPT. Memorandum from Patricia Harrigan, Exposure Assessment Branch, Economics, Exposure, and Technology Division. Subject: Comparison of 1993 Releases of Ethylene Glycol. (August 24, 1995).

13. USEPA, OPPT. Memorandum from Linda M. Rusak, Hazard Integrator, Analysis and Information Management Branch, Chemical Screening and Risk Assessment Division. Subject: Ethylene Glycol, Acute Risk Assessment. (December 16, 1994).

14. USEPA, ORD. Memorandum from Carole Kimmel, National Center for Environmental Assessment. Subject: Review of Ethylene Glycol Risk Assessment for EPCRA Section 313 Delisting Petition. (November 2, 1995).

15. Frantz, S.W. et al., ''Ethylene Glycol: Comparison of Pharmacokinetics and Material Balance Following Single Intravenous, Oral and Cutaneous Administration to Male and Female Sprague-Dawley Rats.'' Bushy Run Research Center, Export, PA. Project Report 51-543. (March 24, 1989).

16. Marshall, Thomas C. and Yung Sung Cheng. ''Deposition and Fate of Inhaled Ethylene Glycol Vapor and Condensation Aerosol in the Rat.'' Fundamental and Applied Toxicology. v. 3, (1983), pp. 175-181.

17. Tyl, R.W. et al., ''Evaluation of the Developmental Toxicity of Ethylene Glycol Aerosol in the CD Rat and CD-1 Mouse by Whole-Body Exposure.'' Fundamental and Applied Toxicology. v. 24, (1995), pp. 57-75.

18. Tyl R.W. et al., ''Evaluation of the Developmental Toxicity of Ethylene Glycol Aerosol in CD-1 Mice by Nose-Only Exposure.'' Fundamental and Applied Toxicology. v. 27, (1995), pp. 49-62.

19. IRIS. 1994. ''Glossary of Risk Assessment-Related Terms.'' U.S. Environmental Protection Agency's Integrated Risk Information System. (February 1, 1994).

20. IRIS. 1994. U.S. Environmental Protection Agency's Integrated Risk Information System file pertaining to Ethylene Glycol. (March 8, 1994).

21. USEPA, OPPT. Memorandum from Linda M. Rusak, Hazard Integrator, Analysis and Information Branch, Chemical Screening and Risk Assessment Division. Subject: Ethylene Glycol, Chronic Risk Assessment. (August 19, 1996).

VI. Administrative Record

The record supporting this notice is contained in docket control number OPPTS-400110. All documents, including the references listed in Unit V. 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

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

Dated: April 28, 1997.

Lynn R. Goldman,

Assistant Administrator for Prevention, Pesticides and Toxic Substances.

[FR Doc. 97–11902 Filed 5–7–97; 8:45 am]

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ENVIRONMENTAL PROTECTION AGENCY

[FRL–5822–2]

Proposed Administrative Settlement Under the Comprehensive Environmental Response, Compensation, and Liability Act; Indian Line Farm Superfund Site

AGENCY: Environmental Protection Agency.

ACTION: Notice of proposed settlement agreement and request for public comment.

SUMMARY: The U.S. Environmental Protection Agency (EPA) is proposing to enter into settlement agreements to address claims under the Comprehensive Environmental Response, Compensation and Liability Act of 1980, as amended (CERCLA), 42 U.S.C. 9601 *et seq.* Notice is being published to inform the public of the proposed settlements and of the opportunity to comment. The settlements are intended to resolve the liability under CERCLA of the Metropolitan District Commission (''MDC''), the Commonwealth of Massachusetts, and TDL, Inc., for past costs incurred by EPA in connection with an emergency removal action conducted in 1992 and 1993, at the Indian Line Farm Superfund Site in Canton, Massachusetts.

DATES: Comments must be provided on or before June 6, 1997.

ADDRESSES: Comments should be addressed to the Docket Clerk, U.S. Environmental Protection Agency, Region I, JFK Federal Building, Mailcode RCG, Boston, Massachusetts 02203, and should refer to: Proposed Administrative Agreement under 122(h) of the Comprehensive Environmental Response, Compensation, and Liability Act; RE: Indian Line Farm Superfund Site Canton, Massachusetts.

FOR FURTHER INFORMATION CONTACT: Sandra Dupuy, U.S. Environmental Protection Agency, J.F.K. Federal