Commission (OTC) regulatory development effort which developed six model control programs. This rulemaking incorporates two of the OTC model control programs into the SIP: Solvent cleaning operations, and mobile equipment repair and refinishing operations. The emission reductions from these control measures will provide for achievement of a portion of the additional emission reductions needed to attain the 1-hour ozone standard.

V. What Are EPA's Conclusions?

EPA has evaluated the submitted revisions for consistency with its provisions, EPA regulations and EPA policy. The proposed control measures go beyond the reasonably available control technology (RACT) level controls that were previously approved for these source categories. These new control programs will strengthen the SIP by providing additional VOC emission reductions. Accordingly, EPA is proposing to approve the Subchapter 16 revisions as adopted on April 30, 2003.

VI. Statutory and Executive Order Reviews

Under Executive Order 12866 (58 FR 51735, October 4, 1993), this proposed action is not a ''significant regulatory action'' and therefore is not subject to review by the Office of Management and Budget. For this reason, this action is also not subject to Executive Order 13211, ''Actions Concerning Regulations That Significantly Affect Energy Supply, Distribution, or Use'' (66 FR 28355, May 22, 2001). This proposed action merely proposes to approve state law as meeting Federal requirements and imposes no additional requirements beyond those imposed by state law. Accordingly, the Administrator certifies that this proposed rule will not have a significant economic impact on a substantial number of small entities under the Regulatory Flexibility Act (5 U.S.C. 601 *et seq.*). Because this rule proposes to approve pre-existing requirements under state law and does not impose any additional enforceable duty beyond that required by state law, it does not contain any unfunded mandate or significantly or uniquely affect small governments, as described in the Unfunded Mandates Reform Act of 1995 (Pub. L. 104–4).

This proposed rule also does not have tribal implications because it will not have a substantial direct effect on one or more Indian tribes, on the relationship between the Federal Government and Indian tribes, or on the distribution of power and responsibilities between the Federal Government and Indian tribes, as specified by Executive Order 13175 (65 FR 67249, November 9, 2000). This action also does not have Federalism implications because it does not have substantial direct effects on the States, on the relationship between the national government and the States, or on the distribution of power and responsibilities among the various levels of government, as specified in Executive Order 13132 (64 FR 43255, August 10, 1999). This action merely proposes to approve a state rule implementing a Federal standard, and does not alter the relationship or the distribution of power and responsibilities established in the Clean Air Act. This proposed rule also is not subject to Executive Order 13045 ''Protection of Children from Environmental Health Risks and Safety Risks'' (62 FR 19885, April 23, 1997), because it is not economically significant.

In reviewing SIP submissions, EPA's role is to approve state choices, provided that they meet the criteria of the Clean Air Act. In this context, in the absence of a prior existing requirement for the State to use voluntary consensus standards (VCS), EPA has no authority to disapprove a SIP submission for failure to use VCS. It would thus be inconsistent with applicable law for EPA, when it reviews a SIP submission, to use VCS in place of a SIP submission that otherwise satisfies the provisions of the Clean Air Act. Thus, the requirements of section 12(d) of the National Technology Transfer and Advancement Act of 1995 (15 U.S.C. 272 note) do not apply. This proposed rule does not impose an information collection burden under the provisions of the Paperwork Reduction Act of 1995 (44 U.S.C. 3501 *et seq.*).

List of Subjects in 40 CFR Part 52

Environmental protection, Air pollution control, Ozone, Reporting and recordkeeping requirements, Volatile organic compounds.

Authority: 42 U.S.C. 7401 *et seq.*

Dated: November 5, 2003.

Jane M. Kenny,

Regional Administrator, Region 2.

[FR Doc. 03–29181 Filed 11–20–03; 8:45 am]

BILLING CODE 6560–50–P

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 63

[OAR–2003–0188; FRL–7587–5]

RIN A2060–0013

List of Hazardous Air Pollutants, Petition Process, Lesser Quantity Designations, Source Category List

AGENCY: Environmental Protection Agency (EPA).

ACTION: Proposed rule.

SUMMARY: The EPA proposes to amend the list of hazardous air pollutants (HAP) contained in section 112(b)(1) of the Clean Air Act (CAA) by removing the compound ethylene glycol monobutyl ether (EGBE) (2-Butoxyethanol) (Chemical Abstract Service (CAS) No. 111–76–2) from the group of glycol ethers. Today's action is being taken in response to a petition to delete EGBE from the HAP list submitted by the Ethylene Glycol Ethers Panel of the American Chemistry Council (formerly the Chemical Manufacturers Association) on behalf of EGBE producers and consumers. Petitions to delete a substance from the HAP list are permitted under section 112(b)(3) of the CAA.

The proposed rule is based on EPA's evaluation of the available information concerning the potential hazards and projected exposures to EGBE. We have made an initial determination that there are adequate data on the health and environmental effects of EGBE to determine that emissions, ambient concentrations, bioaccumulation, or deposition of EGBE may not reasonably be anticipated to cause adverse human health or environmental effects. Today's action includes a detailed rationale for removing EGBE from the glycol ethers group of HAP under section 112(b)(1) list of HAP.

DATES: *Comments.* Written comments on the proposed rule must be received by January 20, 2004.

Public Hearing. A public hearing will be held if requests to speak are received by the EPA on or before December 8, 2003. If requested, a public hearing will be held on December 19, 2003.

ADDRESSES: *Comments.* Comments may be submitted electronically, by mail, or through hand delivery/courier. Electronic comments may be submitted on-line at *http://www.epa.gov/edocket/.* Written comments sent by U.S. mail should be submitted (in duplicate if possible) to: Air and Radiation Docket and Information Center (Mail Code 6102T), Attention Docket ID Number

OAR–2003–0188, Room B108, U.S. EPA, 1200 Pennsylvania Avenue, NW., Washington, DC 20460. Written comments delivered in person or by courier should be submitted (in duplicate if possible) to: Air and Radiation Docket and Information Center (Mail Code 6102T), Attention Docket ID Number OAR–2003–0188, Room B102, U.S. EPA, 1301 Constitution Avenue, NW., Washington, DC 20460. The EPA requests a separate copy also be sent to the contact person listed below (*see* **FOR FURTHER INFORMATION CONTACT**).

Public Hearing. If a public hearing is requested by December 8, 2003 the public hearing will be held at the new EPA facility complex, Research Triangle Park, NC December 19, 2003. Persons interested in presenting oral testimony should contact Ms. Kelly A. Rimer, Risk and Exposure Assessment Group, Emission Standards Division (C404–01), U.S. EPA, Research Triangle Park, North Carolina 27711, telephone number (919) 541–2962 at least two days in advance of the hearing.

FOR FURTHER INFORMATION CONTACT: Ms. Kelly A. Rimer, Risk and Exposure Assessment Group, Emission Standards Division (C404–01), U.S. EPA, Research Triangle Park, NC 27711, telephone number (919) 541–2962, electronic mail address *rimer.kelly@epa.gov.*

SUPPLEMENTARY INFORMATION:

Regulated Entities. Entities potentially affected by today's action are those industrial facilities that manufacture or use EGBE. Today's action proposes to amend the list of HAP contained in section 112(b)(1) of the CAA by removing the compound EGBE.

Docket. The EPA has established an official public docket for this action under Docket ID Number A–99–24 and Electronic Docket ID Number OAR–2003–0188. The official public docket is the collection of materials that is available for public viewing at the EPA Docket Center (Air Docket), EPA West, Room B–108, 1301 Constitution Avenue, NW., Washington, DC 20004. The Docket Center is open from 8:30 a.m. to 4:30 p.m., Monday through Friday, excluding legal holidays. The telephone number for the Reading Room is (202) 566–1744, and the telephone number for the Air Docket is (202) 566–1742. All items may not be listed under both docket numbers, so interested parties should inspect both docket numbers to ensure that they have received all materials relevant to the proposed rule.

Electronic Access. An electronic version of the public docket is available through EPA's electronic public docket and comment system, EPA Dockets. You may use EPA Dockets at *http://www.epa.gov/edocket/* to submit or view public comments, access the index of the contents of the official public docket, and access those documents in the public docket that are available electronically. Once in the system, select ''search'' and key in the appropriate docket identification number.

Certain types of information will not be placed in the EPA dockets. Information claimed as confidential business information (CBI) and other information whose disclosure is restricted by statute, which is not included in the official public docket, will not be available for public viewing in EPA's electronic public docket. The EPA's policy is that copyrighted material will not be placed in EPA's electronic public docket but will be available only in printed paper form in the official public docket. Although not all docket materials may be available electronically, you may still access any of the publicly available docket materials through the EPA Docket Center.

For public commenters, it is important to note that EPA's policy is that public comments, whether submitted electronically or in paper, will be made available for public viewing in EPA's electronic public docket as EPA receives them and without change unless the comment contains copyrighted material, CBI, or other information whose disclosure is restricted by statute. When EPA identifies a comment containing copyrighted material, EPA will provide a reference to that material in the version of the comment that is placed in EPA's electronic public docket. The entire printed comment, including the copyrighted material, will be available in the public docket.

Public comments submitted on computer disks that are mailed or delivered to the docket will be transferred to EPA's electronic public docket. Public comments that are mailed or delivered to the docket will be scanned and placed in EPA's electronic public docket. Where practical, physical objects will be photographed, and the photograph will be placed in EPA's electronic public docket along with a brief description written by the docket staff.

Comments. You may submit comments electronically, by mail, by facsimile, or through hand delivery/courier. To ensure proper receipt by EPA, identify the appropriate docket identification number in the subject line on the first page of your comment. Please ensure that your comments are submitted within the specified comment period. Comments submitted after the close of the comment period will be marked ''late.'' The EPA is not required to consider these late comments.

Electronically. If you submit an electronic comment as prescribed below, EPA recommends that you include your name, mailing address, and an e-mail address or other contact information in the body of your comment. Also include this contact information on the outside of any disk or CD ROM you submit and in any cover letter accompanying the disk or CD ROM. This ensures that you can be identified as the submitter of the comment and allows EPA to contact you in case EPA cannot read your comment due to technical difficulties or needs further information on the substance of your comment. The EPA's policy is that EPA will not edit your comment and any identifying or contact information provided in the body of a comment will be included as part of the comment that is placed in the official public docket and made available in EPA's electronic public docket. If EPA cannot read your comment due to technical difficulties and cannot contact you for clarification, EPA may not be able to consider your comment.

Your use of EPA's electronic public docket to submit comments to EPA electronically is EPA's preferred method for receiving comments. Go directly to EPA Dockets at *http://www.epa.gov/edocket*, and follow the online instructions for submitting comments. Once in the system, select ''search'' and key in Docket ID No. OAR–2003–0188. The system is an ''anonymous access'' system, which means EPA will not know your identity, e-mail address, or other contact information unless you provide it in the body of your comment.

Comments may be sent by electronic mail (e-mail) to *a-and-r-docket@epa.gov*, Attention Docket ID No. OAR–2003–0188. In contrast to EPA's electronic public docket, EPA's e-mail system is not an ''anonymous access'' system. If you send an e-mail comment directly to the docket without going through EPA's electronic public docket, EPA's e-mail system automatically captures your e-mail address. E-mail addresses that are automatically captured by EPA's e-mail system are included as part of the comment that is placed in the official public docket and made available in EPA's electronic public docket.

You may submit comments on a disk or CD ROM that you mail to the mailing address identified in this document. These electronic submissions will be accepted in WordPerfect or ASCII file

format. Avoid the use of special characters and any form of encryption.

By Mail. Send your comments (in duplicate, if possible) to: EPA Docket Center (Air Docket), U.S. EPA West, (MD–6102T), Room B–108, 1200 Pennsylvania Avenue, NW., Washington, DC 20460, Attention Docket ID No. OAR–2003–0188.

By Hand Delivery or Courier. Deliver your comments (in duplicate, if possible) to: EPA Docket Center, Room B–108, U.S. EPA West, 1301 Constitution Avenue, NW., Washington, DC 20004, Attention Docket ID No. OAR–2003–0188. Such deliveries are only accepted during the Docket Center's normal hours of operation.

By Facsimile. Fax your comments to: (202) 566–1741, Docket ID No. OAR–2003–0188.

CBI. Do not submit information that you consider to be CBI through EPA's electronic public docket or by e-mail. Send or deliver information identified as CBI only to the following address: Kelly Rimer, c/o Roberto Morales, Office of Air Quality Planning and Standards (OAQPS) Document Control Officer (C404–02), U.S. EPA, 109 TW Alexander Drive, Research Triangle Park, NC 27709, Attention Docket ID No. OAR–2003–0188. You may claim information that you submit to EPA as CBI by marking any part or all of that information as CBI (if you submit CBI on disk or CD ROM, mark the outside of the disk or CD ROM as CBI and then identify electronically within the disk or CD ROM the specific information that is CBI). Information so marked will not be disclosed except in accordance with procedures set forth in 40 CFR part 2.

Worldwide Web (WWW). In addition to being available in the docket, an electronic copy of today's proposed rule will also be available on the WWW through the Technology Transfer Network (TTN), on the TTN's policy and guidance page for newly proposed or promulgated rules at *http://www.epa.gov/ttn/oarpg.* The TTN provides information and technology exchange in various areas of air pollution control. If more information regarding the TTN is needed, call the TTN HELP line at (919) 541–5384.

Outline. This preamble is organized as follows:

I. Background

Section 112 of the CAA contains a mandate for EPA to evaluate and control emissions of HAP. Section 112(b)(1) includes a list of 188 specific chemical compounds and classes of compounds that Congress identified as HAP. The EPA must evaluate the emissions of substances on the HAP list to identify source categories for which the Agency must establish emission standards under section 112(d). We are required to periodically review the list of HAP and, where appropriate, revise the list by rule. In addition, under section 112(b)(3), any person may petition us to modify the list by adding or deleting one or more substances. A petitioner seeking to delete a substance must demonstrate that there are adequate data on the health and environmental effects of the substance to determine that emissions, ambient concentrations, bioaccumulation, or deposition of the substance may not reasonably be anticipated to cause any adverse effects to human health or the environment. A petitioner must provide a detailed evaluation of the available data concerning the substance's potential adverse health and environmental effects and estimate the potential exposures through inhalation or other routes resulting from emissions of the substance.

On August 29, 1997, the American Chemistry Council's Ethylene Glycol Ethers Panel submitted a petition to delete EGBE (CAS No. 111–76–2) from the HAP list in CAA section 112(b)(1), 42 U.S.C., 7412(b)(1). Following the receipt of the petition, we conducted a preliminary evaluation to determine whether the petition was complete according to Agency criteria. To be deemed complete, a petition must consider all available health and environmental effects data. A petition must also provide comprehensive emissions data, including peak and annual average emissions for each source or for an appropriately selected subset of sources, and must estimate the resulting exposures of people living in the vicinity of the sources. In addition, a petition must address the environmental impacts associated with emissions to the ambient air and impacts associated with the subsequent cross-media transport of those emissions. After receiving additional submittals through December 21, 1998, we determined the petition to delete EGBE to be complete. We published a notice of receipt of a complete petition in the **Federal Register** on August 3, 1999 and requested information to assist us in technically reviewing the petition.

We received eight submissions in response to our request for comment and information which would aid our technical review of the petition. The comments made general statements encouraging EPA to delist EGBE. None of the comments included technical information.

II. Criteria for Delisting

Section 112(b)(2) of the CAA requires us to make periodic revisions to the initial list of HAP set forth in section 112(b)(1) and outlines criteria to be applied in deciding whether to add or delete particular substances. Section 112(b)(2) identifies pollutants that should be listed as:

> * * * pollutants which present, or may present, through inhalation or other routes of exposure, a threat of adverse human health effects (including, but not limited to, substances which are known to be, or may reasonably be anticipated to be, carcinogenic, mutagenic, teratogenic, neurotoxic, which cause reproductive dysfunction, or which are acutely or chronically toxic) or adverse environmental effects whether through ambient concentrations, bioaccumulation, deposition, or otherwise * * *

Section 112(b)(3) of the CAA establishes general requirements for petitioning the Agency to modify the HAP list by adding or deleting a substance. Although the Administrator may add or delete a substance on his or her own initiative, the burden is on a petitioner to include sufficient information to support the requested addition or deletion under the substantive criteria set forth in section 112(b)(3)(B) and (C).

The Administrator must either grant or deny a petition to delist a HAP within 18 months of receipt of a complete petition. If the Administrator decides to deny a petition, the Agency publishes a written explanation of the basis for denial in the **Federal Register**.

A decision to deny a petition is final Agency action subject to review. If the Administrator decides to grant a petition, the Agency publishes a written explanation of the Administrator's decision, along with a proposed rule to add or delete the substance. The proposed rule is open to public comment and public hearing, and all additional substantive information received is considered prior to the issuance of a final rule.

To delete a substance from the HAP list, section 112(b)(3)(C) provides that the Administrator must determine that:

> * * * there is adequate data on the health and environmental effects of the substance to determine that emissions, ambient concentrations, bioaccumulation of deposition of the substance may not reasonably be anticipated to cause any adverse effects to the human health or adverse environmental effects.

We do not interpret CAA section 112(b)(3)(C) to require absolute certainty that a pollutant will not cause adverse effects on human health or the environment before it may be deleted from the list. The use of the terms ''adequate'' and ''reasonably'' indicate that the Agency must weigh the potential uncertainties and likely significance. Uncertainties concerning the risks of adverse health or environmental effects may be mitigated if we can determine that projected exposures are sufficiently low in relation to levels where adverse effects may occur to provide reasonable assurance that such adverse effects will not occur. Similarly, uncertainties concerning the magnitude of projected exposures may be mitigated if we can determine that the levels which might cause adverse health or environmental effects are sufficiently high to provide reasonable assurance that exposures will not reach harmful levels. However, the burden remains on a petitioner to demonstrate that the available data support an affirmative determination that emissions of a substance may not be reasonably anticipated to result in adverse effects on human health or the environment. The EPA will not remove a substance from the list of HAP based merely on the inability to conclude that emissions of the substance will cause adverse effects on human health or the environment. As a part of the requisite demonstration, a petitioner must resolve any critical uncertainties associated with missing information. We will not grant a petition to delete a substance if there are major uncertainties that need to be addressed before we would have sufficient information to make the requisite determination.

III. EPA Analysis of the Petition

A. Background

The broad category of glycol ethers (GE) are general solvents, also known as cellosolves. In 2000, ethylene glycol monobutyl ether made up an estimated 45 percent of the total GE production in the U.S. (or 325,000–350,000 tons). It is a colorless liquid with a mild, rancid odor. It is soluble in most organic solvents and mineral oil. It mixes with acetone, benzene, carbon tetrachloride, ethyl ether, n-heptane and water, and it is miscible with many ketones, ethers, alcohols, aromatic paraffin, and halogenated hydrocarbons.

Ethylene glycol monobutyl ether is used in hydraulic fluids and as a coupling agent for water-based coatings. It is used in vinyl and acrylic paints and varnishes and as a solvent for varnishes, enamels, spray lacquers, dry cleaning compounds, textiles, and cosmetics. Ethylene glycol monobutyl ether is a solvent for grease and grime in industrial cleaning. It is also used as a freeze-thaw agent in latex paints and emulsions, and as an intermediate in the production of esters, ethers, alkoxy alkyl halides, polyether alcohols, hemiacetals and acetals.

The petition states that EGBE released to the air has a half life of 3 to 33 hours. However, the California Air Resources Board (CARB) reports an EGBE half-life of 14 to 22 hours. The midpoint in these ranges of both these half-lives is 18 hours, and we used this value in our analysis as it represents a reasonable estimate of the half-life of EGBE. The petition identifies the principal oxidation products of EGBE as n-butyl formate, 2-hydroxyethyl formate, propionaldehyde, 3-hydroxybutyl formate, and several isomeric forms of an organic nitrate compound. Only one of these compounds (*i.e.*, propionaldehyde) is a listed HAP. However, the formate esters are known to transform in the atmosphere into formaldehyde, which is another listed HAP. In addition, propionaldehyde undergoes further transformation to formaldehyde and acetaldehyde (which is also a HAP).

The portion of EGBE that does not degrade to secondary products in the air, rapidly partitions to soil and water. Once in soil, EGBE is further decomposed through biotic processes, but it has been estimated that as much as 35 percent of the EGBE deposited on soil can eventually move to water. Due to its low volatility, high solubility, low vapor pressure, and minimal tendency to bind to sediments, once in surface water EGBE tends to remain dissolved until it biodegrades (half life = 1 to 4 weeks). It has a low bioconcentration factor, therefore, it is not anticipated to accumulate in the environment or in food stuffs.

Its relatively rapid biodegradation in water indicates that humans are unlikely to be exposed to significant amounts of EGBE in drinking water. However, the fact that EGBE released to the air preferentially partitions to water does raise a question concerning the risk from EGBE ingestion originating from air releases. Based on our review of the available information on EGBE, we have concluded that inhalation and ingestion are the important routes of nonoccupational exposures resulting from EGBE emissions, and consider these two routes of exposure in evaluating this petition.

B. Exposure Assessment

As a first step in evaluating the petition's inhalation risk assessment, we reviewed the petitioner's emissions inventory upon which the modeling was based. The petitioner used the 1993 Toxics Release Inventory (TRI) as a starting point to identify emissions of GE, including EGBE. To locate facilities emitting EGBE which were not included in the TRI, the petitioner searched EPA's TTN to identify regulatory documentation that might contain EGBE emissions data. This documentation includes information on recently promulgated maximum control technology (MACT) standards, information on area sources, and consumer and commercial product Volatile Organic Compounds (VOC) rules. The petitioner searched the National Air Toxics Clearinghouse which contains a database of State air toxic programs identifying those States with active air toxics programs and those that collected chemical specific data and contacted the State agencies for data. The petitioner also contacted 12 trade associations concerned with the use of EGBE to obtain data regarding industry use of EGBE and/or GE. Lastly, the petitioner contacted facilities known to be large EGBE emission sources to obtain specific modeling data, such as emission rates, stack height, distance to fence line.

After reviewing the petitioner's inventory, we have concluded that the methods used to identify sources of EGBE emissions are adequate and provide a reasonable representation of the EGBE emissions. To evaluate the overall completeness of the inventory, we compared the petition's list of EGBE emission sources to EPA's 1996 National Toxics Inventory (NTI), which is now called the National Emissions Inventory (NEI). We found the

petitioner's inventory to be comparable to the NTI. Therefore, we conclude that the petitioner's emissions inventory provides an adequate basis for dispersion modeling and the exposure assessment and is acceptable for that purpose.

The petitioner used a modification of the air dispersion modeling approach described in EPA's ''Tiered Modeling Approach for Assessing Risk due to Sources of Hazardous Air Pollutants'' (EPA–450/4–92–001) (Tiered Approach) to develop predictions of the maximum annual concentrations for the EGBE emission sources identified in its inventory. The petitioner's modifications of the Tiered Approach first consisted of conducting an ''inverted tier 1'' assessment before the petitioner conducted a standard tier 1 analysis. The EPA's tier 1 conservatively predicts the air concentration from a facility when few data are available. The required inputs are: Estimates of annual emission rate, distance to fence line and whether the release is from a point or area source. The result of tier 1 is a maximum annual concentration for the pollutant assessed. The petitioner used the inverted tier 1 approach in order to identify an emission rate that would result in a specified maximum annual concentration. The petitioner could then estimate, for a large number of facilities, what emission rates would result in the specified maximum concentration. All facilities who emitted EGBE in amounts that resulted in the specified maximum concentration would then be brought forth to the next level of analysis. In our review of this approach, we have determined that it is reasonable, and would tend to overestimate rather than underestimate maximum annual ambient average concentrations. This is because the petitioner used a combination of a ground level emission release and a 50 meter distance to fence line, which are assumptions that would tend to overstate impacts. Also, the petitioner chose to use a maximum annual ambient average concentration of 3 milligrams per cubic meter (mg/m^3) as the cut-off for a facility to be brought forward to a more detailed analysis. The value the petitioner chose as a cut-off is far below the EPA inhalation reference concentration, which is a peer-reviewed value defined as an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily inhalation exposure to the human population (including sensitive subgroups) that is likely to be without appreciable risk of deleterious noncancer effects during a life time. Given that the current EPA Inhalation Reference Concentration (RfC) is 13 mg/m^3, using 3 mg/m^3 as a cutoff resulted in a greater number of facilities being brought into the more detailed analysis. This increases our confidence that the exposure assessment will likely over-rather than under-estimate the actual maximum annual ambient average concentrations of EGBE.

All 3,439 sources in the inventory went through the inverted tier 1 analysis. Of those, 286 showed maximum annual ambient average concentrations of EGBE of 3 mg/m^3 or greater. The petitioner included these 286 sources in the next level of analysis, the standard tier 1 analysis described above.

Upon review, we determined the petitioner appropriately applied the tier 1 analysis and correctly identified 64 sources as showing a maximum annual ambient average concentration of 3 mg/m^3 or greater. These sources moved on to the next phase of the analysis.

This next phase is the petitioner's second modification to the standard EPA Tiered Approach. It includes a probabilistic modeling exercise along with a decision analysis method (CARTSCREEN). The petitioner employed these methods as an additional screening tool for sources whose maximum annual average ambient concentrations of EGBE that, according to the tier 2 analysis, are predicted to exceed 3 mg/m^3, but that may not warrant a tier 2 or 3 analysis. The petitioner first constructed a distribution of values of additional source parameters, for example, stack diameter, exit temperature and velocity. The model randomly selected a value for each input from that distribution of values, constructing a hypothetical facility, before running SCREEN3. This procedure was repeated a total of 25,000 times. The results of this probabilistic modeling exercise were imported into the decision tool CARTSCREEN along with data from actual facilities, in order to complete the data set. The results of CARTSCREEN showed which facilities would emit EGBE in amounts that result in maximum annual average ambient concentrations of 3 mg/m^3 or greater. Of the 64 facilities for which this analysis was conducted, 41 sources moved on to the tier 2 analysis.

We have determined that the assumptions and parameter selection underlying this modification are consistent with the objectives of the EPA tiered approach. The modeling component of this approach used SCREEN3, which is a regulatory model developed and used by the EPA. In addition, we have determined that CARTSCREEN uses well established decision tree methods which are appropriately applied here.

The petitioner brought forth the 41 sources from the previous iteration, and added 29 sources back into the tier 2 analysis because there were enough data to do so. The petitioner added these 29 facilities back into the analysis in order to be conservative, even though these facilities produce hazards below the 3 mg/m^3 cutoff established by the petitioner. The petitioner used EPA's SCREEN3 model and followed EPA's Guidance on Air Quality models (40 CFR part 51, appendix W), the EPA's Tiered Modeling Guidance, and SCREEN3 documentation. The tier 2 analysis required the following information for each facility: annual EGBE emission rate; release type (point, area, volume) release height; inside stack diameter; stack gas exit velocity and temperature; horizontal distance across area or volume sources; terrain, land use (urban or rural); and building dimensions. The petitioner included the raw data for the dispersion model analysis and the model outputs. The results showed that maximum predicted annual average ambient concentration of EGBE ranged from near 0 mg/m^3 to 37 mg/m^3.

We reviewed the data, verified the appropriateness of the model and facility input parameters, and evaluated the model outputs for several emissions sources selected at random. Our evaluation confirmed that the petitioner applied appropriate EPA guidelines in the dispersion modeling analysis and that the predicted maximum annual EGBE concentrations were consistent with the objective of the tier 2 analysis.

Two sources had predicted concentrations over 3 mg/m^3. However, the petitioner included five facilities in the tier 3 analysis, in order to include the two largest EGBE emissions sources identified in the inventory. The analysis used EPA's Industrial Source Complex Short Term Model, Version 3 (ISCST3) model and followed EPA's Guidance on Air Quality models, the EPA's Tiered Modeling Guidance, and ISCST3 documentation. In addition to the release inputs used in tier 2, the ISCST3 model requires emissions information for all emission points, (SCREEN3 makes the simplifying assumption that all emissions come out of 1 stack), fence line data, 5 years of meteorological data, and a receptor grid. The petitioner used the regulatory default mode. The results showed that the maximum annual average ambient concentration (regardless of fence line) resulting from a single major source's emissions of EGBE is 0.3 mg/m^3. (A major source is a source that emits greater than 10 tons

per year (tpy) of EGBE or 25 tpy of EGBE combined with other HAP.)

We have determined that the petitioner performed the dispersion modeling analysis following appropriate modeling guidance. Based on our technical review of the various emission modeling components, we have confirmed that the highest predicted maximum annual average off-site concentration (*i.e.*, the maximum annual level occurring over 5 years) of EGBE for any individual major source facility does not exceed 0.3 mg/m^3. We judge that these estimates are more likely to over predict than under predict actual exposures due to the health-protective assumptions made in the analysis. Based on the information provided in the petition on EGBE emissions, we evaluated the potential impact of emission sources within close proximity to each other. First, we looked at the emissions from closely located major sources. Based on our evaluation, we concur with the petitioner that the maximum annual EGBE concentration from closely located major sources is expected to be no greater than 0.07 mg/m^3.

Next, we evaluated the petitioner's modeling approach for closely located area sources (*i.e.*, sources emitting less than 10 tpy EGBE located 500 meters from each other). We determined that the assumptions underlying the petitioner's model were conservative, and that the maximum estimated annual concentration of EGBE from area sources is likely to be no greater than 0.5 mg/m^3. We note that this concentration is higher than the maximum annual ambient average concentration predicted from either a major source or a group of closely located major sources. This is not unexpected as smaller sources can have emission release characteristics that can result in higher impacts to the surrounding communities. For example, while smaller sources may emit less EGBE, they may also have shorter stack heights, or fence lines that are closer to the emission points. Also, people may live closer to a smaller facility.

We reviewed the literature and various EPA databases to assess the potential contribution of the ambient background EGBE to the maximum annual concentration of EGBE. Subsequently, we determined that EGBE monitoring data that could be used to determine the background EGBE level are not available. We, therefore, proceeded to evaluate the petitioner's background estimation approaches. Based on our evaluation, we have determined that both approaches provide acceptable, yet conservative estimates. Therefore, we have concluded that the ambient background concentration of EGBE is not likely to have a significant influence on maximum annual exposures to EGBE.

To summarize the air quality modeling component of the inhalation exposure assessment, the petitioner provided a tiered modeling analysis of EGBE emissions using EPA guidelines and models. The analysis was performed following acceptable modeling guidance. Based on a detailed technical review of the analyses, it is our conclusion that model inputs, assumptions, and results provide a conservative representation of EGBE sources. The modeling analysis demonstrated that the maximum annual concentration of EGBE was no greater than 0.3 mg/m^3 from a single major source, 0.07 mg/m^3 from a cluster of major sources, and 0.5 mg/m^3 from a cluster of area sources.

We judge the petition's overall approach to exposure assessment to be acceptable. The use of the maximum annual average ambient concentration for each emission source to characterize the exposed population provides a conservative approach to chronic exposure modeling. Furthermore, based on our experience, we judge that a refined exposure assessment estimating exposures for actual people living near these facilities would result in maximum individual exposures significantly lower than the maximum annual average ambient approach. Given the likely proximity of inhabitable areas and the variability of human activity patterns over an annualized time period, it is our expectation that actual maximum individual exposure would be at least a factor of 2 less than predicted by the models and at least an order of magnitude below EPA's RfC.

After evaluating the petitioner's ingestion exposure scenarios, we determined that the scenarios were acceptable and that the human exposure parameters used to calculate a person's average daily intake were conservative. However, as a part of our assessment of potential ecological risk due to EGBE emissions, we had previously derived an independent estimate of the concentration of EGBE in a water body situated at the point of the maximum annual average EGBE concentration from the largest emission source in the petitioner's inventory. This estimate was approximately 28 times greater than that presented in the petition. Therefore, based on this estimate, we were concerned that the petitioner's estimation method was not sufficiently conservative, and we carried out the following analysis described below.

Our estimation of EGBE in surface water was a worst-case estimate. It was derived using a Mackay Level III fugacity model to estimate the steady state equilibrium concentration of a known volume (*i.e.*, 1,000 kilograms per hour (kg/h) of EGBE released to the atmosphere in each of four environmental media: Air, soil, sediment, and water. The EGBE concentration predicted in air was then ratioed with the maximum concentration predicted for a single major source from the petitioner's ISCST3 model (*i.e.*, 0.3 mg/m^3) of the largest emission source to develop a scaling factor. The EGBE concentration in water as predicted by the Mackay model was then multiplied by the scaling factor to predict EGBE concentrations in a water body situated at the point of the maximum annual average EGBE concentration. The results yielded an estimated concentration of 3.6 milligrams per liter (mg/L) of EGBE in the water body.

We consider these results to be very conservative (*i.e.*, worst case) because numerous variables were not taken into consideration that, if considered, were likely to reduce estimates of EGBE in water. For example, we did not consider degradation in the water, nor did we consider that the body of water would have to be continuously exposed at the fence line concentration across its entire surface to approach this predicted concentration. Therefore, we do not anticipate surface water concentrations greater that 3.6 mg/L to occur as a result of airborne deposition of EGBE.

Even though we do not feel that surface water concentrations would approach 3.6. mg/L, we used this worst case estimate, to recalculate the average daily intake for each of the age groups in each exposure scenario. For the Residential Scenario involving the ingestion of EGBE in drinking water, we calculated an average daily intake of 0.1 milligram per kilogram per day (mg/kg/day) for adults and 0.2 mg/kg/day for children of both age groups. For the Residential Scenario involving dermal contact with EGBE during bathing and showering, we determined an average daily intake of 0.00003 mg/kg/day for adults, 0.0004 mg/kg/day for older children, and 0.0005 mg/kg/day for younger children. For the Recreational Scenario involving incidental ingestion of EGBE in surface water while swimming, we calculated an average daily intake of 0.0007 mg/kg/day for adults, 0.04 mg/kg/day for older children, and 0.03 mg/kg/day for younger children. Lastly, for the

Recreational Scenario involving dermal contact with EGBE in surface water, we calculated an average daily intake of 0.0003 mg/kg/day for adults, 0.0002 mg/kg/day for older children, and 0.0006 g/kg/day for younger children.

Combining the Residential and Recreational Scenarios for each of the age groups provided a worst-case exposure scenario. The average daily intake for the combined worst case are: Adults 0.1 mg/kg/day, older children 0.3 mg/kg/day, and younger children 0.3 mg/kg/day. Based on this analysis, we have concluded that exposures to EGBE arising from the ingestion of surface water exposed may not reasonably be anticipated to exceed 0.3 mg/kg/day, and would be significantly less.

C. Human Health Effects of EGBE

The petitioner used the 1997 draft Integrated Risk Information System (IRIS) assessment as the basis for their human health effects evaluation of EGBE. Since then, the IRIS assessment has been completed (in 1999) and more recent toxicological information on EGBE has become available. Therefore, rather than evaluating the information presented in the petition, we focus our evaluation of EGBE's health effects on the more recent data.

We used the IRIS toxicological database to evaluate the human health effects associated with exposures to EGBE, and to identify an appropriate human health criterion for the risk characterization (IRIS, 1999). Specifically, we used the toxicological data presented in support of the IRIS RfC and Inhalation Reference Concentration and reference dose (RfD) which is contained in *The Toxicological Review of Ethylene Glycol Monobutyl Ether (EGBE).* This document is electronically available via EPA's IRIS Page at *http://www.epa.gov/iris.* The IRIS is the Agency's official repository of consensus human health risk information. It was created and is maintained by the Agency to provide assistance to Agency decision makers on the potential adverse human health effects of particular substances. In addition, EPA scientists have investigated and analyzed information on the human carcinogenic potential of EGBE that was published after the IRIS assessment was final. We had our evaluation of the new information peer reviewed by experts external to the agency, and we use this evaluation to help us draw conclusions about the potential for EGBE to cause cancer in humans (see docket for EPA's August, 2003 Interim Final Report, ''An evaluation of the Human Carcinogenic Potential of Ethylene Glycol Butyl Ether''). Based on these reviews, we have determined that adequate data concerning the potential health effects of EGBE are available and are of sufficient quality to use as the basis for deciding whether or not to delete EGBE.

The IRIS reports that the reproductive toxicity of EGBE has been studied in a variety of well conducted oral and inhalation studies using rats, mice, and rabbits. In addition, several developmental studies have addressed EGBE toxicity from conception to sexual maturity including toxicity to the embryo and fetus, following oral and dermal exposures to rats, mice, and rabbits. Ethylene glycol monobutyl ether was not found to cause adverse effects in any reproductive organs in any study. In a two generational reproductive toxicity study, fertility was reduced in mice only at very high (maternally toxic) doses. Maternal toxicity related to the adverse effects on red blood cells (called hematologic effects) due to exposure to EGBE and relatively minor developmental effects have been reported in developmental studies. We conclude from these studies that EGBE is not significantly toxic to reproductive organs of parents, male or female. In addition, no teratogenic toxicities were noted in any of the studies. Therefore, we also conclude that EGBE is not significantly toxic to developing fetuses of laboratory animals.

Our review of the IRIS assessment confirmed that hemotologic effects is the primary response in sensitive species following inhalation, oral, or dermal administration of EGBE. The reported sensitivities range from that of the guinea pig which displays no hemolytic effects from EGBE at exposures levels as high as 1,000 mg/kg (oral) or 2,000 mg/kg (dermally) to the rat which displays increased sensitivity at single-inhalation exposures below 100 parts per million (ppm) (483 mg/m^3) and single oral exposures below 100 mg/kg. No hemolysis has been observed in controlled laboratory acute inhalation exposures of human volunteers up to 195 ppm (941.9 mg/m^3) and reversible hemolytic effects have been observed in a case where humans consumed single oral doses of 400 to 1,500 mg/kg of EGBE.

Data considered in the IRIS toxicological review, primarily from acute and in vitro studies, indicate that humans are significantly less sensitive to the hemolytic toxicity of EGBE than typical laboratory species such as mice, rats, or rabbits. While studies of chronically exposed humans are lacking, several laboratory animal studies have demonstrated this, as have in vitro studies using either whole blood or washed red blood cells. In addition, blood from potentially sensitive individuals, including the elderly and those persons with congenital hemolytic disorder such as sickle-cell anemia or hereditary spherocytosis, does not show an increased hemolytic response when incubated with EGBE's active metabolite, 2-butoxyacetic acid (BAA).

The principal study used to determine the EGBE RfC is a 2-year bioassay that involved groups of F344 rats exposed to 0, 31, 125, and 500 ppm EGBE in air for 12 months (6 hours/day, 5 days/week). Female rats exposed to the three highest concentrations at all exposure durations developed clinical signs consistent with hemolytic effects associated with EGBE exposures. A Lowest Observed Adverse Effects Level (LOAEL) of 31 ppm (149.7 mg/m^3) was identified in this study for hematologic and histopathologic effects in female rats.

The human equivalent concentration (HEC) was calculated using the standard RfC approach, a physiologically based pharmacokinetic (PBPK) approach, a benchmark concentration (BMC) approach, and a PBPK/BMC approaches combined. The PBPK/BMC approach was determined by the IRIS Peer Review Panel to provide the best estimate of a HEC because it incorporated much of the mechanistic information available for EGBE, best characterized the dose-response relationship for EGBE-induced hematologic effects, and reduced the potential uncertainties to the greatest extent. The HEC as determined by the PBPK/BMC method was then reduced by a series of uncertainty factors to derive the RfC. An overall uncertainty factor (UF) of 30 was applied to account for extrapolation from an adverse effect (UF = 3) and to account for the variation in the sensitivity within the human population (UF = 10).

The principal study for the ingestion Rfd involved groups of 10 female F344 rats exposed to 750, 1,500, 3,000, 4,500, and 6,000 ppm of EGBE via drinking water for 13 weeks. Decreases in body weight were observed in female rats exposed to the two highest dose levels. The study results show hematologic changes at all dose levels after 13 weeks that were indicative of mild to moderate anemia. Using this study, EPA calculated human equivalent doses (HED) using all four approaches. We selected the PBPK/BMD approach for the derivation of the RfD because it incorporated much of the mechanistic information available for EGBE, best characterized the dose-response relationships for EGBE-induced hematologic effects, and reduced the potential uncertainties to the greatest extent. Using the HED from the PBPK/

BMC model, and a total UF of 10 to account for variation in sensitivity within the human population (UF = 10), the EPA determined that the IRIS RfD was 0.5 mg/kg/day.

The IRIS review states that EGBE has been adequately tested in conventional genotoxicity tests for its potential to induce gene mutations in in vitro systems and cytogenetic damage in both in vitro and in vivo systems. The available data do not support a mutagenic or clastogenic potential for EGBE. The EPA's Toxicological Review of EGBE, available at *http://www.epa.gov/iris/toxreviews/0500-tr.pdf#page=68*, states that one laboratory has reported weak genotoxicity responses at toxic doses, though these data are considered to be questionable, may be a result of impurities in the test material.

In addition, the 1999 IRIS describes structure-activity relationship (SAR) analyses that have been conducted to provide insight into EGBE's potential carcinogenicity to humans. These analyses have been found to be useful for agents that are believed to initiate carcinogenesis through Deoxyribonucleic Acid (DNA) reactive mechanisms. Based on chemical structure, EGBE does not resemble any known chemical human carcinogens and is not expected to have electrophilic or DNA reactive activity. The IRIS review states that there are no reliable epidemiologic studies available that address the potential carcinogenicity of EGBE.

The IRIS review utilized a draft report of the results of a 2-year inhalation bioassay performed by the National Toxicology Program (NTP, 1998) using rats and mice that had recently become available. The NTP (1998) report indicates no evidence of carcinogenic activity in male F344/N rats, and equivocal evidence of carcinogenic activity in female F344/N rats based on increased combined incidences of benign and malignant pheochromocytoma (mainly benign) of the adrenal medulla. They also reported some evidence of carcinogenic activity in male B6C3F1 mice based on increased incidences of hemangiosarcoma of the liver, and some evidence of carcinogenic activity in female B6C3F1 mice based on increased incidences of forestomach squamous cell papilloma or carcinoma (mainly papilloma).

The IRIS discusses the relevance of these tumors to humans. For example, the phenochromocytoma in the female rats were indicated as only a marginally significant trend. Further, these types of tumors are difficult to distinguish from non-neoplastic adrenal medullary hyperplasia, and therefore need to be interpreted with caution. The hemangiosarcoma in livers of male mice appear to be exposure related. However, the increases were slight and, like the forestomach lesions in female mice, were not observed in any other sex or species. There is also evidence to suggest that these cancer lesions in mice are associated with unique aspects of mouse physiology (*i.e.*, the known increased sensitivity of mice to oxidative stress and the existence of a forestomach), and are secondary to noncancer (*i.e.*, hemolysis and forestomach irritation) effects.

The IRIS concludes that because of the uncertain relevance of these tumor increases to humans, the fact that EGBE is generally negative in genotoxic tests, and the lack of human data to support the findings in rodents, the human carcinogenic potential of EGBE, in accordance with the recently proposed Guidelines for Carcinogen Risk Assessment (U.S. EPA, 1996a), cannot be determined at this time, but suggestive evidence exists from rodent studies. Therefore, under existing EPA guidelines, EGBE is judged to be a possible human carcinogen.

Since the publication of NTP's draft report (NTP, 1998) on their 2-year inhalation bioassay of EGBE, and since the IRIS update of December 1999, there has been continued discussion among scientists from government, industry, and academia concerning the human carcinogenic potential of EGBE. The NTP (2000a) finalized their study results without changing their original determination of equivocal evidence of carcinogenic activity in female rats, some evidence of carcinogenic activity in male mice, and some evidence of carcinogenic activity in female mice. These findings by NTP, along with the EPA's conclusion in the 1999 IRIS assessment that the carcinogenic potential of EGBE ''cannot be determined at this time, but suggestive evidence exists from rodent studies'', prompted scientists from academia and industry to design research projects aimed at determining the mode of action for the formation of the forestomach and liver tumors observed in mice. We report here on recent findings in scientific publications, from scientific meetings and in the EPA (1999b) draft cancer guidelines, to provide an up-to-date evaluation of the mode of action involved in the origin of these tumors in mice and their human relevance.

Establishing the mode of action is critical for determining an effect's relevance to humans and for choosing the approach most appropriate for dose-response modeling (*i.e.*, whether to use a linear or nonlinear approach). As is extensively discussed in the Agency's interim and draft cancer guidelines (U.S. EPA. 1999b; 2003), in order to determine a chemical's mode of action, one must consider the full range of key influences a chemical or its metabolites might have as an initiator or promoter of the complex carcinogenic process. With this in mind, we evaluated EGBE's role in the formation of female mouse forestomach and male mouse liver tumors that were observed following two-years of inhalation exposure (National Toxicology Program, 2000a). Our August 2003 interim final report provides details of this evaluation.

With regard to forestomach papillomas and carcinoma in female mice, the NTP study (NTP 2000a) shows that at the highest exposure level, 250 ppm, the 10 percent incidence of squamous papilloma and 12 percent combined incidence of squamous cell papillomas or carcinomas were significantly increased over study controls and exceeded the ranges for historical controls of 0–2 percent and 0–3 percent, respectively. This study reports that 8 percent is the highest incidence of forestomach neoplasms that has been observed in contemporary historical controls. NTP (2000a) did not observe significant increases in forestomach papillomas and carcinomas at any other exposure levels in female mice, nor at any exposure level in male mice or either sex of rats.

Recent reviews of available in vitro and in vivo genotoxicity assays are in agreement that EGBE is not likely to be genotoxic (Commonwealth of Australia, 1996; Elliot and Ashby, 1997; U.S. EPA, 1999a; NTP, 2000a). The NTP (2000a)) suggested that EGBE caused chronic irritation leading to forestomach injury including penetrating ulcers and that the observed ''neoplasia (papillomas and one carcinoma) was associated with a continuation of the injury/degeneration process.''

The Agency believes that EGBE is not genotoxic and that a nonlinear mode of action is principally responsible for the increased forestomach tumor incidence reported by NTP (2000a). However, reports of weak positive effects by EGBE at high concentrations in some in vitro assays (see discussion in full report located in the docket under ''Other Possible Modes of Action for Forestomach Tumor Development in Female Mice'') indicate the potential for contribution from direct interaction of butoxyacetaldehyde (BAL), an EGBE metabolite, with DNA. While these weak positive findings may be due to study design artifacts (*e.g.*, changes in

pH or osmolarity associated with high EGBE concentrations), they may indicate contribution from BAL which has caused clastogenic changes in Chinese hamster lung (v79) and human lymphocyte cells (Elliot and Ashby, 1997). As we discuss in the full report, available evidence from a published EGBE PBPK model that has been modified to include kinetics for the metabolism of the BAL intermediate (Corley, 2003) suggests that the conditions of these in vitro assays (*e.g.*, no metabolic activation; high, cytotoxic concentrations of BAL) are of little relevance to expected target organ (forestomach) environment (*e.g.*, high metabolic activity; low concentrations of BAL). However, additional research (*e.g.*, verification of these PBPK modeling results and further genotoxicity research using more appropriate assays and currently accepted test protocols) would be beneficial to provide a more definitive determination regarding the role of BAL in the formation of forestomach tumors in female mice.

We conclude that the available data establish a plausible nonlinear, nongenotoxic mode of action for the moderate increase observed by NTP (2000a) in the incidence of forestomach tumors in female mice following chronic inhalation exposure to EGBE. Forestomach tissue irritation caused by constant exposure to EGBE and its metabolites and subsequent cell proliferation appear to be key precursor events in the mode of action for these tumors. While certain dosimetric processes and morphological aspects of the forestomach make rodents particularly susceptible to these events, we judge this mode of action to be of qualitative relevance to humans. However, due to the lack of a comparable organ for storage and the long term retention of EGBE, the exposure concentrations that would be necessary to cause hyperplastic effects and tumors in humans, if attainable, are likely to be much higher than the concentrations necessary to cause forestomach effects in mice. In fact, our analysis indicates that the exposure concentrations necessary to cause hyperplastic effects in humans would be much higher than the existing RfD and RfC for EGBE. Given that humans, including potentially sensitive subpopulations such as children, have no known organ for the retention of a comparable target dose of EGBE or its metabolites, we feel it is reasonable to conclude that the RfC and RfD developed for EGBE (EPA, 1999a) are sufficient for the prevention of hyperplasia and associate tumors in humans.

With respect to liver tumors in male mice, scientists have placed particular focus on hemangiosarcomas of the liver reported by NTP (2000a) because this was the only tumor type that was increased over both concurrent and historical controls, and because one study proposed a mode of action involving EGBE for this tumor (Sascha *et al.*, 2002).

A metabolite of EGBE, butoxyacetic acid, has long been known to cause hemolysis in rodents (Carpenter *et al*, 1956). This hemolysis leads to the accumulation of hemosiderin (iron) in phagocytic Kupffer cells of the liver of both rats and mice (NTP, 2000a). Recent research in mice and rats indicates that the increased iron levels associated with EGBE-induced hemolysis can produce oxygen radicals which produce oxidative damage in the liver that is more severe in mice than in other species, and increased DNA synthesis in both cells that line blood vessels and liver cells that is unique to mice (Sascha *et al.*, 2002). This research hypothesizes that these events can contribute to the transformation of the endothelial cells to hemangiosarcomas (and hepatocytes to hepatocellular carcinomas) in male mice. Given the high background rate of these tumors in male mice relative to female mice and rats (NTP, 2000b; Klaunig, 2002), we feel it is reasonable to hypothesize that the endothelial cells and hepatocytes in the livers of male mice are more susceptible to oxidative stress resulting from iron buildup in local Kupffer cells. While additional research would be informative with respect to mechanistic issues such as the relative susceptibility of endothelial cells and hepatocytes to oxidative stress caused by the hemolytic effects of EGBE and the apparent resistance of female mice to the development of hemangiosarcomas despite experiencing similar hemolytic effects, there is enough evidence at this time to support an EPA determination that events associated with hemolysis could have contributed to the increased incidence of these tumors in male mice exposed to EGBE.

Available data establish a plausible nonlinear, nongenotoxic mode of action for the moderate increase observed by NTP (2000a) in the incidence of liver tumors in male mice following chronic inhalation exposure to EGBE. The proposed mode of action suggests that the endothelial cells and hepatocytes of male mice are sensitive to the formation of the subject neoplasms (as evidenced by the relatively high background rate of these tumors in male mice) and that excess iron from EGBE-induced hemolysis can result in sufficient iron-induced oxidative stress to cause the observed, marginal increase in the incidence of liver hemangiosarcomas and hepatocellular carcinomas in these animals (NTP, 2000a). Given the relatively low sensitivity of humans, including subpopulations such as children, to the hemolytic effects of EGBE, we feel it is reasonable to conclude that the EGBE RfC and RfD (EPA, 1999a) are sufficient for the prevention of hemolysis and associate tumors in humans.

We anticipate additional research may be completed in the near term. We will review those results and peer review our findings at the earliest opportunity.

D. Human Health Risk Characterization and Conclusions

We used a Hazard Quotient (HQ) approach to characterize the noncancer risk associated with the exposures to EGBE. In this case, the HQ is developed by comparing the level of exposure to the IRIS RfC or RfD for EGBE. If the HQ is less than 1, the reference level is not exceeded, and the adverse health effects are unlikely.

Based on our assessment of the information provided in the petition, it is possible to derive a quantitative evaluation of an inhalation HQ for EGBE. Based on our evaluation of the modeling data, we judge that maximum ambient annual average exposures to EGBE are not likely to exceed 0.3 mg/m^3 for a single major source, or 0.5 mg/m^3 for a group of closely located area sources. The reference level to be used in the determination of EGBE's HQ is the RfC of 13 mg/m^3. This criterion addresses the health effect of concern due to chronic inhalation exposures to EGBE. In addition, the criterion includes the margins of safety built into the IRIS RfC (*i.e.*, any needed uncertainty factors to address sensitive subpopulations and other factors) and is, therefore, protective of sensitive subpopulations.

Using this approach, we calculate an HQ for the maximum annual ambient concentration of EGBE from a single major source to be 0.02. In other words, the EGBE air concentration is 2 percent of the RfC. For closely located area sources, the HQ is 0.04, or 4 percent of the RfC. To be extremely conservative, we might assume that the single major source is located among the group of area sources. In this case, the maximum annual ambient average concentration would be 0.8 mg/m^3 and the HQ would be 0.06, or 6 percent of the RfC. All HQ are well below the health criterion of an HQ of 1. Further, we judge that the

exposures to EGBE of actual persons living in the immediate vicinity of EGBE emission sources would be significantly less than the concentrations estimated by the model. Considering such things as human activity patterns and that predicted ambient concentrations fall significantly from those predicted by the models, we expect that the HQ for most of the surrounding population would be several orders of magnitude less than one.

We also use a Hazard Index (HI) approach to characterize the potential for EGBE exposures to cause adverse effects when combined with typical exposures to pollutants that also affect the circulatory system. In this case, we rely on the 1996 National Air Toxics Assessment (NATA) which estimates risks to certain HAP by census blocks. The NATA results indicate that more than 99 percent of the census blocks have circulatory system HI below 0.1. As such, even when combined with other exposures to circulatory system toxicants, EGBE exposures would results in HI that are well below 1.0 and, therefore, would not be associated with risk of adverse effects.

The reference level we used to determine EGBE's ingestion HQ is the IRIS RfD of 0.5 (mg/kg/day). Based on our analysis, we judge that maximum exposures to EGBE via ingestion of water contaminated with EGBE from air releases is not likely to exceed 0.28 mg/kg/day. The resulting HQ is 0.6. In other words the concentration in the environment is 60 percent of the RfD. Given the conservative nature of the parameters used to derive the average daily intake, we conclude that the actual HQ will be significantly less than 0.6.

Therefore, based on information presented in the petition, EPA's evaluation of data made available after the submission of the petition, and our own supplemental analyses, we have made an initial determination that emissions, ambient concentrations, bioaccumulation or deposition of EGBE may not reasonably be anticipated to cause any adverse effects to human health.

E. Ecological Risk Characterization and Conclusions

We developed an independent ecological risk assessment (ERA) to evaluate the potential environmental impacts of EGBE emissions. We organized our analysis according to EPA's framework for ecological risk assessment and followed a two tiered approach. Under this approach, the tier 1 analysis used conservative point estimates of exposure (maximum possible concentration in the environment) and effect (*e.g.*, national ambient water quality criterion). If the tier 1 analysis indicated that a conservative estimate of exposure would not exceed a very sensitive effects threshold (*i.e.*, quotient <1), the analysis was terminated. If the tier 1 analysis indicated the potential for effect (*i.e*, quotient >1), the analysis proceeded to tier 2. In tier 2, more realistic assumptions were made about exposure and effects. If the tier 2 quotients were less than one, the analysis was terminated. However, if one or more of the tier 2 quotients were greater than one, the risk assessment would proceed to a probabilistic risk assessment.

Because EGBE concentrations will be the highest close to the emission source and because it is unlikely to be transported widely due to its short half-life in air and its propensity to partition from air to soil and water, we decided that the appropriate spatial modeling scale for the analysis was local. Using the petitioner's dispersion modeling analysis, we selected the single facility from the inventory that was the source of the largest maximum predicted annual concentration of EGBE as predicted by the ISCST3 model. This maximum annual average concentration was then used in conjunction with a Mackay Level I fugacity model to determine a steady state equilibrium concentration of EGBE in soil, water, and sediment in a simulated environment situated at the fence line. (Due to the relatively short distance from the source to the fence line, we assumed EGBE to disperse in the atmosphere as a passive tracer, not subject to removal through deposition or chemical reaction during transport.)

We developed exposure scenarios for small mammals and aquatic species and derived a quotient to characterize the potential ecological risk. The tier 1 ERA suggested that EGBE may have the potential to cause adverse effects to small mammals and to sensitive aquatic biota residing close to and downwind of the largest emitting source. This determination was, at least in part, due to the conservatism of tier 1 analysis, and the fact the decision criterion for these quotients were derived from very minor effects which were unlikely to be ecologically significant at the population level of ecological organization.

The tier 2 analysis combined a Level III Mackay Model and the ISCST3 outputs for the largest source. The Level III fugacity model takes into account reaction, advection and intermedia exchange after emission to the atmosphere. Based on the fugacity/ISCST3 approach, the estimated EGBE concentrations in air, soil, and water were determined to be 0.3 mg/m^3, 0.07 mg/kg, and 3.64 mg/L, respectively.

The lowest aquatic acute toxicity value available was for the protozoan *Endosiphon sulcatum* which experienced a 5 percent inhibition of cell multiplication at 91 mg/L following a 72-hour exposure. Due to the relatively minor effect reported and because the protozoa were exposed over several generations during the 72-hour period, we applied an acute/chronic adjustment factor of 10 to derive a safe level (*i.e.*, toxicity reference value (TRV)) of 9 mg/L for aquatic biota in water.

The TRV for small mammals was based on the critical mammalian studies identified by IRIS for inhalation and oral exposure. Hemolysis was the critical endpoint of concern. A TRV of 20 mg/kg/day was derived by dividing the most sensitive LOAEL for female rats (59 mg/kg/day) by an uncertainty factor of three to adjust for the absence of a NOAEL.

Exposure scenarios were developed for each species and a quotient was calculated. In both cases, the quotient for aquatic invertebrates and small mammals was determined to be less than one. This suggested that both aquatic organisms and small mammals are not likely to be adversely affected by EGBE emissions to the atmosphere.

Based on our review of these data supplemented by additional environmental modeling, we have made an initial determination that there are adequate data on environmental effects of EGBE to determine that ambient concentrations, bioaccumulation, or deposition of EGBE are not reasonably anticipated to cause adverse environmental effects.

F. Transformation Assessment

Ethylene glycol monobutyl ether is one of many VOC that transform into other HAP after emission into the ambient air. The petition identifies the principal oxidation products of EGBE as n-butyl formate, 2-hydroxyethyl formate, propionaldehyde, 3-hydroxybutyl formate, and several isomeric forms of an organic nitrate compound. Only one of these compounds (*i.e.*, propionaldehyde) is a listed HAP. However, the formate esters are known to transform in the atmosphere into formaldehyde, which is another listed HAP. In addition, propionaldehyde undergoes further transformation to formaldehyde and acetaldehyde (which is also a HAP). Both formaldehyde and acetaldehyde are probable human carcinogens and

have been identified by the EPA as among the 33 HAP of greatest concern under the Integrated Urban Air Toxics Strategy published in the **Federal Register** on July 19, 1999 (64 FR 38706).

The petitioner concluded that insignificant amounts of these compounds are formed as a result of secondary transformation of EGBE. After reviewing the petitioner's analysis, we concluded that it was a reasonable effort to determine whether EGBE transformation products are likely to be of concern. However, there were data gaps and additional questions which we judged to need further attention. Consequently, we undertook an independent analysis to estimate typical urban ambient air concentrations of formaldehyde, acetaldehyde, and propionaldehyde due to EGBE transformation. Our evaluation, summarized below, indicates that atmospheric transformation of EGBE emissions may not reasonably be anticipated to cause adverse effects to human health. The full transformation assessment is contained in the docket.

A large percentage of ambient formaldehyde and acetaldehyde is due to atmospheric transformation of VOC. In fact, the State of California has estimated that as much as 88 percent of the ambient formaldehyde and 41 to 67 percent of the ambient acetaldehyde arise from atmospheric transformation from VOC. The remainder is attributed to direct emissions. A previous analyses carried out as part of the EPA's Cumulative Exposure Project (CEP) in the mid-1990s suggests that EGBE transformation is not among the most significant contributors to ambient formaldehyde and acetaldehyde. The CEP analysis identified two pollutants (propene and ethene) as major contributors to ambient concentrations of formaldehyde, and two pollutants (propene and 2-butene) as the major contributors to acetaldehyde. Several other VOCs including EGBE were considered only minor precursors to formaldehyde and acetaldehyde in the CEP analysis.

Secondary formaldehyde is formed from EGBE via a two step process. First, EGBE with an average half-life of approximately 18 hours and a life time of about 25 hours transforms into intermediate compounds, such as formate esters and proprionaldehyde. Second, these compounds transform into formaldehyde. Based on the information contained in the petition, formate esters have half-lives ranging from 21 hours to 55 hours. Proprionaldehyde has a half-life of about 12 hours. Due to the relatively long time required to complete the process, and the resulting large dilution of the EGBE reaction products in the atmosphere, we do not anticipate elevated concentrations of formaldehyde formation due to EGBE transformation near EGBE emissions points that will cause adverse effects to human health.

We have estimated that the half-life for EGBE to convert to formaldehyde through the two step process is approximately 37 hours. Assuming the average wind speed is about 3 miles per hour (mph), a plume from any given EGBE emission will travel about 111 miles in a 37-hour period. A conservative dispersion calculation at this point in time indicates that the plume is well dispersed such that EGBE concentrations are decreased by at least 300-fold from the predicted maximum fence line concentrations. Considering dispersion alone and the maximum fence line concentration for the largest EGBE emission source presented in the petition of approximately 330 micrograms per meter cube (ug/m^3) (*i.e.*, 0.3 mg/m^3), we can conservatively estimate that EGBE levels in typical urban areas might be as high as 1 ug/m^3. Concurrent with this dispersion, EGBE emissions transform relatively slowly into formaldehyde which, in turn, decomposes much more quickly. We estimate that the concentrations of formaldehyde due to EGBE transformation at this point would be roughly 0.06 ug/m^3.

Based on available ambient monitoring data for 82 urban area monitoring sites in 17 States, we determined that the ambient average concentration of formaldehyde in urban areas is about 2.8 ug/m^3. Therefore, we estimate that roughly 2 percent (*i.e.*, 0.06 ug/m^3) of the ambient formaldehyde could be due to EGBE transformation. However, due to the conservatism built into the estimation procedure, we feel this is an overestimate. We feel that the actual contribution of EGBE to formaldehyde levels is much less than 2 percent.

We also considered the risk to human health posed by ambient formaldehyde. Using EPA default exposure and risk assumptions (such as the assumption that there is no threshold for the carcinogenic effect and that the dose-response relationship is linear at low doses), the increased risk of cancer for people assumed to be exposed for a lifetime to the ambient concentration can be calculated by multiplying the ambient concentration by the cancer Unit Risk Estimate (URE). The URE is an upper bound estimate of the increased risk of cancer per unit of exposure for a lifetime. (The IRIS glossary defines upper-bound as ''a plausible upper limit to the value of a quantity. This is usually not a true statistical confidence limit''.) The current URE for formaldehyde, as listed by IRIS, is 1.3×10^{-5} per microgram per cubic meter (per ug/m^3). (**Note:** The EPA periodically reviews and updates the toxicological information for chemicals on IRIS. Currently we are reviewing formaldehyde. As such, the URE may change, but based on currently available information, it is not likely to become higher than what is currently on IRIS.) This means that if people are exposed to 1 microgram of formaldehyde per cubic meter of air (1 ug/m^3) for a lifetime, we estimate that they would have an estimated upper bound increased risk of cancer of 1.3×10^{-5} or 13 in a million. Therefore, if we assume people are exposed to the average ambient concentration of formaldehyde (*i.e.*, 2.8 ug/m^3) for a lifetime, we calculate the upper bound increased cancer risk for these people to be about 30 in a million, or 3×10^{-5}. Thus, while the total level of risk from ambient levels of formaldehyde is greater than one in a million (or 1×10^{-6}), a relatively small portion of these ambient levels is likely to be attributable to EGBE transformation.

Given the level of risk from formaldehyde generally, and because EGBE is likely to contribute less than 2 percent to the total ambient concentration of formaldehyde, we do not anticipate that formaldehyde from EGBE transformation will have an adverse impact on human health.

We also assessed the potential for adverse health effects other than cancer. No EPA RfC is available for formaldehyde for an assessment of noncancer risks. Therefore, we compared ambient levels to the minimal risk level (MRL) for formaldehyde, produced by the Agency for Toxics Substances and Disease Registry. The MRL for formaldehyde is 10 ug/m^3. The ambient outdoor levels of formaldehyde used for this analysis are less than the MRL, which suggests that adverse noncancer effects are not likely to result from exposures to these ambient outdoor concentrations.

Propionaldehyde is also produced by the secondary transformation of EGBE. The half-life of propionaldehyde is about 1.4 times shorter than the half-life of EGBE, which indicates that propionaldehyde degrades about 1.4 times faster than it is formed from EGBE. Assuming steady state, we have determined that the concentration of propionaldehyde (in ug/m^3) is expected to be roughly 2.8 times lower than the concentration of EGBE. Assuming that 1

ug/m^3 is representative of the ambient EGBE concentrations expected in typical urban areas, based on monitoring data, we estimate that propionaldehyde concentrations resulting from degradation of these EGBE levels would be roughly 0.4 ug/m^3.

Based on available monitoring data from 23 sites, the mean ambient air concentration of propionaldehyde is 0.94 ug/m^3. The 95th percentile of the ambient monitoring data is 2.3 ug/m^3. Since the ambient average concentration of propionaldehyde in urban areas is about 0.94 ug/m^e, we estimated that as much as 40 percent (*i.e.*, 0.4 ug/m^3) of the ambient propionaldehyde could be due to EGBE transformation.

Propionaldehyde is not classified as a carcinogen, and we were not able to locate data that indicated carcinogenic properties. Consequently, cancer risks due to the ambient levels of propionaldehyde were not evaluated. There are, however, very limited data on noncancer effects of propionaldehyde; but there are no RfCs or MRLs available.

The only noncancer benchmark found on propionaldehyde is a draft Preliminary Evaluation Concentration (PEC) of 9 ug/m^3, developed in 1994 and presented in a draft EPA report titled: *Non-Cancer Benchmarks for Screening Hazardous Air Pollutants for the Urban Area Source Program. Draft for Peer Review*. (April 1994). The draft PEC is an interim screening level value and has not undergone peer review. It is based on the assumption that propionaldehyde exhibits toxic effects similar to acetaldehyde, but is less toxic than acetaldehyde. In deriving the PEC, several uncertainty factors were applied to account for various uncertainties and data limitations. Based on the approach to derivation, we believe that the PEC is probably protective, and that exposures to propionaldehyde at levels below 9 ug/m^3 are not likely to pose significant risk of adverse noncancer health effects.

Using the PEC as a decision criterion, the mean ambient concentrations for propionaldehyde (about 0.94 ug/m^3) and the 95th percentile (about 2.3 ug/m^3) are well below the PEC of 9 ug/m^3. Although we estimate EGBE transformation to contribute as much as 40 percent of the ambient concentration of propionaldehyde, we judge that adverse noncancer health effects are not likely to result due to transformation of EGBE to propionaldehyde.

Acetaldehyde is also formed from EGBE via a two step process. In this process, EGBE transforms to propionaldehyde which then further converts to one of 3 compounds: formaldehyde, acetaldehyde or peroxypropionly nitrate. As described previously in this section, we assumed that each EGBE molecule is converted to one propionaldehyde molecule in 25 hours and that half of the propionaldehyde converts into acetaldehyde in 12 hours. Based on these assumptions, we estimated that in approximately 37 hours, one half of the available EGBE molecules in the ambient air is convert to acetaldehyde molecules. The half-life of acetaldehyde is about 2.5 times shorter than the half-life of EGBE's conversion to acetaldehyde through the two step process, which indicates that acetaldehyde degrades about 2.5 times faster than it is formed from EGBE. Therefore, assuming steady state, the concentration of acetaldehyde is predicted to be roughly 6.7 times lower than the concentration of EGBE. Assuming that 1 ug/m^3 is representative of the ambient EGBE concentrations that would be expected in typical urban areas, we estimate that acetaldehyde concentrations resulting from degradation of these EGBE levels would be roughly 6.7 times lower, or 0.15 ug/m^3.

Since the ambient average concentration of acetaldehyde in urban areas is about 2.5 ug/m^3 (based on available ambient monitoring data for urban areas), we estimated that roughly 6 percent or 0.15 ug/m^3 of the ambient acetaldehyde could be due to EGBE transformation. We think this is a conservative estimate, and that the actual contribution of EGBE to acetaldehyde levels in typical urban areas is likely to be less than 6 percent.

To evaluate the potential risks for public health, the increased cancer risks can be estimated. The URE for acetaldehyde is 2×10^{-6} per ug/m^3. (**Note:** As with formaldehyde, the URE for acetaldehyde is currently being reviewed by EPA and is likely to change. However, based on currently available information, the URE for acetaldehyde is not likely to become significantly higher, and may be much lower than the current value.) This means that if people are exposed to 1 microgram of acetaldehyde per cubic meter of air (1 ug/m^3) for a lifetime, we estimate that they would have an estimated upper bound increased risk of cancer of 2.2×10^{-6}, or 2.2 in 1 million. Therefore, if we assume people are exposed to the average ambient concentration of acetaldehyde (*i.e.*, 2.5 ug/m^3) for a lifetime, we calculated the upper bound increased cancer risk for these people to be about 6 in 1 million, or 6×10^{-6}. As with formaldehyde, the total risk level from ambient levels of acetaldehyde is greater than 1 in 1 million. However, only a relatively small portion of these ambient levels is attributable to EGBE transformation. Because EGBE is likely to contribute less than 6 percent of the total ambient concentration of acetaldehyde, we do not anticipate that acetaldehyde from EGBE transformation will have an adverse impact on human health.

We also evaluated the potential for noncancer hazards. The RfC for acetaldehyde is 9 ug/m^3, which is higher than the reported ambient concentrations, therefore, we do not expect adverse noncancer effects to occur due to exposures to these outdoor ambient concentrations.

Based on our analyses, as well as information presented in the petition, we feel that EGBE transformation to HAP is not a significant concern for public health. Since EGBE transformation products are likely to pose relatively low risks in typical urban ambient air, and since EGBE emissions are not expected to result in elevated levels of formaldehyde, proprionaldehyde, or acetaldehyde near EGBE emission sources that pose significant risks to human health, we have made an initial determination that the available data indicate that atmospheric transformation of EGBE emissions to other HAP is not reasonably anticipated to cause significant human health risks.

The quantitative estimates and the associated risk estimates presented above have some uncertainty associated with the estimates. This is due to the simplified approach, assumptions made, and incomplete knowledge of the atmospheric chemistry, and toxicity of the chemicals. However, we generally used conservative assumptions including: lifetime exposures; linear non-threshold dose-response relationship; conservative estimate of formaldehyde that would be formed per mole of EGBE transformed; and that the EGBE concentrations are 1 ug/m^3. Therefore, we judge that the estimates of risk due to the transformation of EGBE to formaldehyde, proprionaldehyde, and acetaldehyde as presented in this analysis are more likely to be overestimated rather than underestimated. Overall, this analysis suggests that the fractions of formaldehyde, proprionaldehyde, and acetaldehyde in typical urban ambient air resulting from transformation of EGBE emissions are not likely to pose significant risks to human health.

The EPA also recognizes that EGBE is a potential tropospheric ozone precursor. However, we feel that it is inappropriate to include a substance on the HAP list under CAA section 112(b)

due entirely to its tendency to form ozone. Section 112(b)(2) of the CAA provides that no air pollutant which is listed under CAA section 108(a), such as ozone, may be added to the HAP list. It further provides that a pollutant that is a precursor to a pollutant listed under section 108(a), such as EGBE, may not be included on the HAP list unless it ''independently meets'' the HAP list criteria. As explained in this preamble, we feel that the petitioner has demonstrated that EGBE does not independently meet the criteria for listing as a HAP under section 112 of the CAA.

The CAA established requirements for reducing the emission of air pollutants, and deals separately with HAP (which are to be listed and regulated under CAA section 112) and criteria air pollutants (which are to be listed under CAA section 108 and regulate under various other sections of the CAA). Precursors of criteria air pollutants, such as VOC, are regulated for their contribution to ambient levels of criteria pollutants under statutory provisions that do not apply to HAP. This structure would lose its significance if EPA were to include substances on the HAP list solely as a result of their contribution to concentrations of criteria air pollutants.

G. Public Comments

We requested public comment as a part of the **Federal Register** notice announcing the receipt of a complete petition to delist EGBE (64 FR 42125–27). The comments contained no technical information or data which was relevant to our review of this petition. Copies of the comments have been included in the docket for the proposed rule.

H. Conclusions

Uncertainty is an inherent part of risk assessment. It arises because risk assessment is a complex process, requiring the integration of multiple factors, and because it involves predictions of risk that are not directly observable. In the analysis, uncertainty arises for the following reasons. The IRIS database, used as the source of the human health effects decision criteria, is imperfect and leads to uncertainty in the RfC. We also recognize that there is uncertainty in the computer models used to predict the fate and transport of EGBE in the environment. These models are simplifications of reality and some variables are excluded.

For decisions which are based largely on risk assessments, some degree of uncertainty is acceptable. Such is the case for this proposed delisting decision. We do not interpret CAA section 112(b)(3)(C) to require absolute certainty that a pollutant will not cause adverse effects on human health or the environment before it may be deleted from the list. The use of the terms ''adequate'' and ''reasonably'' indicate that the Agency must weigh the potential uncertainties and their likely significance. To this end, the assessment applies conservative assumptions to bias potential error toward overstating human and ecological health effects. Thus, EPA is confident that even when we consider the uncertainties in the petition's initial assessment and in the additional analyses, the results are more likely to over-estimate rather than under-estimate true exposures and risks.

Based on our evaluation of the petition and the subsequent analyses, we judge that the potential for adverse human health and environmental effects to occur from projected exposures is sufficiently low to provide reasonable assurance that such adverse effects will not occur. For example, the petitioner appropriately applied EPA's model guidelines and EPA's tiered dispersion modeling approach which we designed to be conservative. Also, the petitioner used sound analytic principles in modifying the standard assessments described in the Tiered Approach, the inverted tier 1 and the CARTSCREEN analyses. In addition, the petition did not apply a formal exposure assessment to the predicted ambient air concentrations. Instead, the petition used the maximum annual ambient average air concentrations alone as a surrogate for exposure. Based upon the likely proximity of inhabitable areas and knowledge of human activity patterns, we feel that actual exposures will be far less than predicted exposures that were derived from the dispersion analysis. Further, when modeling clusters of EGBE sources, the petition showed that concentrations resulting from both closely located major and area sources are not likely to adversely affect health. Finally, the petition's analysis using available data from monitors suggest that ambient concentrations of EGBE in urban areas are over two orders of magnitude lower than the modeled maximum concentrations.

With regard to toxicity, the information available to the Agency at this time indicates that nonlinear modes of action are likely responsible for the increased incidence of tumors observed by the NTP (2000) in mice following chronic EGBE exposure. Application of nonlinear quantitative assessment methods indicate that the noncancer RfD of 0.5 mg/kg/day and the RfC of 13 mg/m^3, which EPA developed for EGBE, are adequately protective of these carcinogenic effects. This determination assumes a nonlinear mechanism that requires exposure levels to be high enough to cause certain lesions that are precancerous. Information is currently inadequate to dismiss the potential contribution of a linear mechanism associated with the possible mutagenic metabolite BAL. Additional research (*e.g.*, verification of existing physiologically based pharmaco kinetic modeling results and improved genotoxicity assays) would assist the Agency in making a more certain decision concerning the potential for BAL to contribute to the adverse effects seen in animals following EGBE exposure and use of the proposed nonlinear assessment approach. If additional information on BAL becomes available between the proposal and the final action on the delisting decision, EPA will evaluate and peer review such information. We may or may not determine that any new information would be relevant to our analysis of EGBE emissions.

As described above, EPA's proposed decision to remove EGBE from the list of HAP is based on the results of a risk assessment demonstrating that emissions of EGBE may not reasonably be anticipated to result in adverse human health or environmental effects. In addition to the analyses presented and the uncertainties inherent in risk assessment, we have considered other information related to EGBE in making this decision, namely the transformation of EGBE into other HAP as it decomposes in the ambient air. We conclude that ambient concentrations of the transformed HAP are very small, and that they decompose rapidly. Therefore, we do not anticipate that EGBE transformation will be significant enough to have an adverse impact on human health.

We also considered the fact that EGBE is reported to the Toxics Release Inventory (TRI) as part of the group of glycol ethers. The 2000 TRI shows the air emissions of the class of chemicals ''Certain Glycol Ethers'' to be ranked number 12 by volume. Under the proposed rule, it would no longer be regulated as a HAP, but it will continue to be reported in the TRI, as part of the group ''Certain Glycol Ethers'' and regulated under EPA's criteria pollutant (ozone) program.

In conclusion, EPA has made an initial determination, after careful consideration of the petition and after completing additional analyses, that there are adequate data on the health and environmental effects of EGBE to determine that emissions, ambient concentrations, bioaccumulation of

deposition of EGBE may not reasonably be anticipated to cause any adverse effects to the human health or adverse environmental effects.

IV. Statutory and Executive Order Reviews

A. Executive Order 12866: Regulatory Planning and Review

Under Executive Order 12866 (58 FR 51735, October 4, 1993), EPA must determine whether the regulatory action is ''significant'' and, therefore, subject to Office of Management and Budget (OMB) review and the requirements of the Executive Order. The Executive Order defines ''significant regulatory action'' as one that is likely to result in a rule that may:

(1) Have an annual effect on the economy of $100 million or more or adverse affect in a material way the economy, a sector to the economy, productivity, competition, jobs, the environment, public health or safety, or state, local or tribal governments or communities;

(2) Create a serious inconsistency or otherwise interfere with an action taken or planned by another agency;

(3) Materially alter the budgetary impact of entitlements, grants, user fees, or loan programs, or the rights and obligation of recipients thereof; or

(4) Raise novel legal or policy issues arising out of legal mandates, the President's priorities, or the principles set forth in the Executive Order.

Pursuant to the terms of Executive Order 12866, it has been determined that the proposed action does not constitute a ''significant regulatory action'' and is, therefore, not subject to OMB review.

B. Paperwork Reduction Act

This action does not impose an information collection burden under the provisions of the Paperwork Reduction Act, 44 U.S.C. 3501 *et seq.* The proposed action will remove EGBE from the CAA section 112(b)(1) HAP list and, therefore, eliminate the need for information collection under the CAA. Burden means the total time, effort, or financial resources expended by persons to generate, maintain, retain, or disclose or provide information to or for a Federal agency. This includes the time needed to review instructions; develop, acquire, install, and utilize technology and systems for the purposes of collecting, validating, and verifying information, processing and maintaining information, and disclosing and providing information; adjust the existing ways to comply with any previously applicable instructions and requirements; train personnel to be able to respond to a collection of information; search data sources; complete and review the collection of information; and transmit or otherwise disclose the information. An Agency may not conduct or sponsor, and a person is not required to respond to a collection of information unless it displays a currently valid OMB control number. The OMB control numbers for EPA's regulations are listed in 40 CFR part 9 and 48 CFR chapter 15.

C. Regulatory Flexibility Act

The Regulatory Flexibility Act (RFA) generally requires an agency to prepare a regulatory flexibility analysis of any rule subject to notice and comment rulemaking requirements under the Administrative Procedure Act or any other statute unless the agency certifies that the rule will not have a significant economic impact on a substantial number of small entities. Small entities include small business, small organizations, and small governmental jurisdictions. For the purposes of assessing the impacts of today's proposed rule on small entities, small entity is defined as: (1) A small business that meets the definitions for small business based on the Small Business Association (SBA) size standards which, for this proposed action, can include manufacturing (NAICS 3999–03) and air transportation (NAICS 4522–98 and 4512–98) operations that employ less 1,000 people and engineering services (NAICS 8711–98) operations that earn less than $20 million annually; (2) a small governmental jurisdiction that is a government of a city, county, town, school district or special district with a population of less than 50,000; and (3) a small organization that is any not-for-profit enterprise which is independently owned and operated and is not dominant in its field.

After considering the economic impact of today's proposed rule on small entities, I certify that this proposed action will not have a significant economic impact on a substantial number of small entities. In determining whether a rule has significant economic impact on a substantial number of small entities, the impact of concern is any significant adverse economic impact on small entities, since the primary purpose of the regulatory flexibility analysis is to identify and address regulatory alternatives ''which minimize any significant economic impact of the proposed rule on small entities.'' (5 U.S.C. 603 and 604). Thus, an agency may certify that a rule will not have a significant economic impact on a substantial number of small entities if the rule relieves regulatory burden, or otherwise has a positive economic effect on all of the small entities subject to the rule. The proposed rule will eliminate the burden of additional controls necessary to reduce EGBE emissions and the associated operating, monitoring and reporting requirements. We have, therefore, concluded that today's proposed rule will relieve regulatory burden for all small entities. We continue to be interested in the potential impacts of the proposed rule on small entities and welcome comments on issues related to such impacts.

D. Unfunded Mandates Reform Act

Title II of the Unfunded Mandates Reform Act of 1995 (UMRA), Public Law 1044, establishes requirements for Federal agencies to assess the effects of their regulatory actions on State, local, and tribal governments and the private sector. Under section 202 of the UMRA, EPA generally must prepare a written statement, including a cost-benefit analysis, for proposed and final rules with ''Federal mandates'' that may result in expenditures to State, local, and tribal governments, in the aggregate, or to the private sector, of $100 million or more in any 1 year. Before promulgating an EPA rule for which a written statement is needed, section 205 of the UMRA generally requires EPA to identify and consider a reasonable number of regulatory alternatives and adopt the least costly, most cost-effective or least burdensome alternative that achieves the objectives of the rule. The provisions of section 205 do not apply when they are inconsistent with applicable law. Moreover, section 205 allows EPA to adopt an alternative other than the least costly, most cost-effective or least burdensome alternative if the Administrator publishes with the final rule an explanation why that alternative was not adopted. Before EPA establishes any regulatory requirements that may significantly or uniquely affect small governments, including tribal governments, it must have developed under section 203 of the UMRA a small government agency plan. The plan must provide for notifying potentially affected small governments, enabling officials of affected small governments to have meaningful and timely input in the development of EPA regulatory proposals with significant Federal intergovernmental mandates, and informing, educating, and advising small governments on compliance with the regulatory requirements.

Today's proposed rule contains no Federal mandates for State, local, or

tribal governments or the private sector. The proposed rule imposes no enforceable duty on any State, local or tribal governments or the private sector. In any event, EPA has determined that the proposed rule does not contain a Federal mandate that may result in expenditures of $100 million or more for State, local, and tribal governments, in the aggregate, or the private sector in any 1 year. Because the proposed rule removes a compound previously labeled in the CAA as a HAP, it actually reduces the burden established under the CAA. Thus, today's proposed rule is not subject to the requirements of sections 202 and 205 of the UMRA.

E. Executive Order 13132: Federalism

Executive Order 13132 (64 FR 43255, August 10, 1999) requires EPA to develop an accountable process to ensure ''meaningful and timely input by State and local officials in the development of regulatory policies that have federalism implications.'' ''Policies that have federalism implications'' is defined in the Executive Order to include regulations that have ''substantial direct effects on the States, on the relationship between the national government and the States, or on the distribution of power and responsibilities among the various levels of government.''

Under Executive Order 13132, EPA may not issue a regulation that has federalism implications, that imposes substantial direct compliance costs, and that is not required by statute, unless the Federal government provides the funds necessary to pay the direct compliance costs incurred by State and local governments, or EPA consults with State and local officials early in the process of developing the proposed regulation. The EPA also may not issue a regulation that has federalism implications and that preempts State law unless the Agency consults with State and local officials early in the process of developing the proposed regulation.

Today's proposed rule removes the substance EGBE from the list of HAP contained under section 112(b)(1) of the CAA. It does not impose any additional requirements on the States and does not affect the balance of power between the States and the Federal government. Thus, the requirements of section 6 of the Executive Order do not apply to the proposed rule.

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

Executive Order 13175 (65 FR 67249, November 9, 2000) requires EPA to develop an accountable process to ensure ''meaningful and timely input by tribal officials in the development of regulatory policies that have tribal implications.'' The proposed rule does not have tribal implications, as specified in Executive Order 13175.

A review of the available emission inventory does not indicate that tribal EGBE emissions sources are subject to control under the CAA, therefore, the proposed rule is not anticipated to have tribal implications. In addition, the proposed action will eliminate control requirements for EGBE and, therefore, reduces control costs and reporting requirements for any tribal entity operating a EGBE source subject to control under the CAA which we might have missed. Thus, Executive Order 13175 does not apply to the proposed rule.

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

Executive Order 13045 (62 FR 19885, April 23, 1997) applies to any rule that: (1) Is determined to be ''economically significant'' as defined under Executive Order 12866, and (2) concerns an environmental health or safety risk that EPA has reason to believe may have a disproportionate effect on children. If the regulatory action meets both criteria, the Agency must evaluate the environmental health or safety effects of the planned rule on children, and explain why the planned regulation is preferable to other potentially effective and reasonably feasible alternatives considered by the Agency.

The EPA interprets Executive Order 13045 as applying only to those regulatory actions that are based on health or safety risks, such that the analysis required under section 5–501 of the Executive Order has the potential to influence the regulation. The proposed rule is not subject to Executive Order 13045 because it is not economically significant as defined in Executive Order 12866, and because the Agency does not have reason to believe the environmental health or safety risks addressed by this action present a disproportionate risk to children. This determination is based on the fact that the RfC is determined to be protective of sensitive sub-populations, including children.

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

The proposed rule is not subject to Executive Order 13211 (66 FR 28355, May 22, 2001) because it is not a significant regulatory action under Executive Order 12866.

I. National Technology Transfer and Advancement Act

Section 112(d) of the National Technology Transfer and Advancement Act of 1995 (NTTAA), Public Law No. 104–113, section 12(d) 915 U.S.C. 272 note, directs all Federal agencies to use voluntary consensus standards instead of government-unique standards in their regulatory activities unless to do so would be inconsistent with applicable law or otherwise impractical. Voluntary consensus standards are technical standards (*e.g.*, material specifications, test method, sampling and analytical procedures, business practices, *etc.*) that are developed or adopted by one or more voluntary consensus standards bodies. Examples of organizations generally regarded as voluntary consensus standards bodies include the American Society for Testing and Materials (ASTM), the National Fire Protection Association (NFPA), and the Society of Automotive Engineers (SAE). The NTTAA requires Federal agencies like EPA to provide Congress, through OMB, with explanations when an agency decides not to use available and applicable voluntary consensus standards. The proposed rule does not involve technical standards. Therefore, EPA is not considering the use of any voluntary consensus standards.

List of Subjects in 40 CFR Part 63

Environmental protection, Air pollution control, Hazardous substances, Reporting and recordkeeping requirements.

Dated: November 4, 2003.

Marianne Lamont Horinko,

Acting Administrator.

For the reasons set out in the preamble, title 40, chapter 1, part 63, of the Code of Federal Regulations is proposed to be amended as follows:

PART 63—[AMENDED]

1. The authority citation for part 63 continues to read as follows:

Authority: 42 U.S.C. 7401, *et seq.*

Subpart C—[Amended]

2. Subpart C is amended by adding § 63.61 to read as follows:

§ 63.61 Deletion of ethylene glycol monobutyl ether (CAS number 111–76–2) from the list of hazardous air pollutants.

The substance ethylene glycol monobutyl ether (EGBE) (2-Butoxyethanol) (CAS No. 111–76–2) is deleted from the list of hazardous air

pollutants established by 42 U.S.C. 7412(b)(1).

[FR Doc. 03–28787 Filed 11–20–03; 8:45 am]
BILLING CODE 6560–50–P

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Parts 123 and 501

[FRL–7589–7]

Water Pollution Control; State Program Requirements; Program Modification Application by Arizona To Administer the Sewage Sludge Management (Biosolids) Program

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice of application and public comment period.

SUMMARY: The State of Arizona has submitted a program modification application to EPA, Region 9 to administer the sewage sludge (biosolids) management program. According to the State's application, this program would be administered by the Arizona Department of Environmental Quality (ADEQ). The application from Arizona is complete and is available for inspection and copying.

DATES: The public comment period on the State's request for approval to administer the proposed AZPDES biosolids program will be from the date of publication until January 5, 2004. Comments postmarked after this date may not be considered.

ADDRESSES: *Viewing/Obtaining Copies of Documents.* You can view Arizona's application for modification from 8 a.m. until 5 p.m. Monday through Friday, excluding holidays, at the Arizona Department of Environment Quality, Records Management Center, 1110 W. Washington St., Phoenix, AZ 85007. Please call (602) 771–4378 to set up an appointment. A copy of Arizona's application is also available for viewing from 9 am to 4 pm, Monday through Friday, excluding legal holidays, at EPA Region 9, 12th floor, Water Division, 75 Hawthorne St., San Francisco, CA. Part or all of the State's application may be copied, for a minimal cost per page, at ADEQ's office in Phoenix or EPA's office in San Francisco. ADEQ's submission documents are also available on the Internet at: *http://www.adeq.state.az.us/environ/water/compliance/assurance.html#bio.*

Comments. Electronic comments are encouraged and should be submitted to mitchell.*matthew@epa.gov.* Please send a copy to *varga.chris@ev.state.az.us.* Written comments may be sent to Matthew Mitchell (WTR–5), EPA, Region 9, 75 Hawthorne Street, San Francisco, CA 94105. Please send an additional copy to Chris Varga, Surface Water Permits Unit, Arizona Department of Environmental Quality, 1110 W. Washington, Phoenix, AZ 85007. Public comments may be sent in either electronic or paper format. EPA requests that electronic comments include the commentor's postal mailing address. No Confidential Business Information (CBI) should be submitted through e-mail. Comments and data will also be accepted on disks in WordPerfect 8.0 format or ASCII file format. If submitting comments in paper format, please submit the original and three copies of your comments and enclosures. Commentors who want EPA to acknowledge receipt of their comments should enclose a self-addressed stamped envelope.

FOR FURTHER INFORMATION CONTACT: Matthew Mitchell at the above address by phone at (415) 972–3508, or by e-mail at *mitchell.matthew@epa.gov.*

SUPPLEMENTARY INFORMATION:

Background

Under section 402 of the Clean Water Act (CWA), 33 U.S.C. 1342, the EPA may issue permits allowing discharges of pollutants from point sources into waters of the United States, subject to various requirements of the CWA. These permits are known as National Pollutant Discharge Elimination System (NPDES) permits. Section 402(b) of the CWA, 33 U.S.C. 1342(b), allows states to apply to the EPA for authorization to administer their own NPDES permit programs.

Section 405 of the Clean Water Act (CWA), 33 U.S.C. 1345, created the sewage sludge management program, requiring EPA to set standards for the use and disposal of sewage sludge and requiring EPA to include sewage sludge conditions in some of the NPDES permits which it issues. The rules developed under section 405(d) are also self-implementing, and the standards are enforceable whether or not a permit has been issued. Section 405(c) of the CWA provides that a state may submit an application to EPA for administering its own sewage sludge program within its jurisdiction. EPA is required to approve each such submitted state program unless EPA determines that the program does not meet the requirements of sections 304(i) and/or 402(b) and 405 of the CWA or the EPA regulations implementing those sections.

On June 11, 2002, Arizona submitted an application to EPA for approval of a state-administered NPDES permit program pursuant to CWA section 402(b). The Arizona NPDES program (known as AZPDES) was approved by EPA on December 5, 2002. Prior to its submission of the AZPDES program application, Arizona determined that it would submit a separate application for the CWA Section 405 biosolids program at a later date. EPA received the biosolids program submittal from Arizona on November 29, 2002. Arizona's application for the biosolids management program approval contains a letter from the Governor requesting program approval, an Attorney General's Statement, copies of pertinent State statutes and regulations, a Program Description, and a Memorandum of Agreement (MOA) to be executed by the Regional Administrator of EPA, Region 9 and the Director of ADEQ. The State submitted a modification of its Attorney General's Statement, which EPA received on October 10, 2003.

Biosolids and the State Biosolids Management Program

Biosolids, or sewage sludge, are the solids separated from liquids during treatment at a domestic or municipal wastewater treatment plant and treated to stabilize and reduce pathogens. EPA in 1993 adopted standards for management of biosolids generated during the process of treating municipal wastewater. 40 CFR part 503. The part 503 rules establishes standards under which biosolids may be land applied as a soil amendment, disposed in a surface disposal site, or incinerated, and requirements for compliance with 40 CFR part 258 if placed in a municipal landfill. The standards, designed to protect public health and the environment, include pollutant limits, pathogen reduction requirements, vector attraction reduction requirements, and management practices specific to the use or disposal option selected.

The Arizona biosolids management program imposes requirements on wastewater treatment plants, biosolids appliers, and surface disposal site operators. It also provides for the issuance of permits under certain conditions, enforcing the standards as necessary, and providing guidance and technical assistance to members of the regulated community. The program also includes a state-specific feature requiring a land applier to register an application site with ADEQ before biosolids is applied to the site.

Indian Country

Arizona is not authorized to carry out its biosolids management program in Indian Country, as defined in 18 U.S.C. 1151.