EUP is effective from April 30, 2002 to October 30, 2005. (Dani Daniel; Rm. 211, Crystal Mall #2; telephone number: (703) 305–5409; e-mail address: *daniel.dani@epa.gov*).

100–EUP–RRN. Issuance. Syngenta Crop Protection, Inc., P.O. Box 18300, Greensboro, NC 27419. This EUP allows the use of 120.8 pounds of the insecticide thiamethoxam on 1,230 sq. ft. of 615 structures over a period of 3 years to evaluate the control of termites and other nuisance pests around homes. The program is authorized only in the States of Alabama, Arizona, California, Florida, Georgia, Hawaii, Kentucky, Louisiana, Mississippi, Nebraska, North Carolina, Oklahoma, South Carolina, Tennessee, Texas, and Virginia. The EUP is effective from April 30, 2002 to October 30, 2005. (Dani Daniel; Rm. 211, Crystal Mall #2; telephone number: (703) 305–5409; e-mail address: daniel.dani@epa.gov).

Persons wishing to review these EUPs are referred to the designated contact person. Inquiries concerning these permits should be directed to the persons cited above. It is suggested that interested persons call before visiting the EPA office, so that the appropriate file may be made available for inspection purposes from 8 a.m. to 4 p.m., Monday through Friday, excluding legal holidays.

Authority: 7 U.S.C. 136.

List of Subjects

Environmental protection, Experimental use permits.

Dated: February 6, 2002.

Donald R. Stubbs,

Acting Director, Registration Division, Office of Pesticide Programs.

[FR Doc. 02–3662 Filed 2–14–02; 8:45 am]

BILLING CODE 6560–50–S

ENVIRONMENTAL PROTECTION AGENCY

[OPPTS–00330; FRL–6815–8]

National Advisory Committee for Acute Exposure Guideline Levels (AEGLs) for Hazardous Substances; Proposed AEGL Values

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: The National Advisory Committee for Acute Exposure Guideline Levels for Hazardous Substances (NAC/AEGL Committee) is developing AEGLs on an ongoing basis to provide Federal, State, and local agencies with information on short-term exposures to hazardous chemicals. This notice provides AEGL values and Executive Summaries for eight chemicals for public review and comment. Comments are welcome on both the AEGL values in this notice and the technical support documents placed in the public version of the official docket for these eight chemicals.

DATES: Comments, identified by docket control number OPPTS–00330, must be received on or before March 18, 2002.

ADDRESSES: Comments may be submitted by mail, electronically, or in person. Please follow the detailed instructions for each method as provided in Unit I. of the **SUPPLEMENTARY INFORMATION**. To ensure proper receipt by EPA, it is imperative that you identify docket control number OPPTS–00330 in the subject line on the first page of your response.

FOR FURTHER INFORMATION CONTACT: *For general information contact*: Barbara Cunningham, Acting Director, Environmental Assistance Division (7408M), Office of Pollution Prevention and Toxics, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460; telephone number: (202) 554–1404; e-mail address: TSCA-Hotline@epa.gov.

For technical information contact: Paul S. Tobin, Designated Federal Officer (DFO), Office of Pollution Prevention and Toxics (7406M), Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460; telephone number: (202) 564–8557; e-mail address: tobin.paul@epa.gov.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

This action is directed to the general public to provide an opportunity for review and comment on ''Proposed'' AEGL values and their supporting scientific rationale. This action may be of particular interest to anyone who may be affected if the AEGL values are adopted by government agencies for emergency planning, prevention, or response programs, such as EPA's Risk Management Program under the Clean Air Act and Amendments Section 112r. It is possible that other Federal agencies besides EPA, as well as State and local agencies and private organizations, may adopt the AEGL values for their programs. As such, the Agency has not attempted to describe all the specific entities that may be affected by this action. If you have any questions regarding the applicability of this action to a particular entity, consult the DFO listed under **FOR FURTHER INFORMATION CONTACT**.

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

1. *Electronically*. You may obtain electronic copies of this document, and certain other related documents that might be available electronically, from the EPA Internet Home Page at http://www.epa.gov/. To access this document, on the Home Page select ''Laws and Regulations,'' ''Regulations and Proposed Rules,'' and then look up the entry for this document under the ''**Federal Register**—Environmental Documents.'' You can also go directly to the **Federal Register** listings at http://www.epa.gov/fedrgstr/.

2. *In person*. The Agency has established an official record for this action under docket control number OPPTS–00330. The official record consists of the documents specifically referenced in this action, any public comments received during an applicable comment period, and other information related to this action, including any information claimed as Confidential Business Information (CBI). This official record includes the documents that are physically located in the docket, as well as the documents that are referenced in those documents. The public version of the official record does not include any information claimed as CBI. The public version of the official record, which includes printed, paper versions of any electronic comments submitted during an applicable comment period, is available for inspection in the TSCA Nonconfidential Information Center, North East Mall Rm. B–607, Waterside Mall, 401 M St., SW., Washington, DC. The Center is open from noon to 4 p.m., Monday through Friday, excluding legal holidays. The telephone number for the Center is (202) 260–7099.

C. How and to Whom Do I Submit Comments?

You may submit comments through the mail, in person, or electronically. To ensure proper receipt by EPA, it is imperative that you identify docket control number OPPTS–00330 in the subject line on the first page of your response.

1. *By mail*. Submit your comments to: Document Control Office (7407), Office of Pollution Prevention and Toxics (OPPT), Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460.

2. *In person or by courier*. Deliver your comments to: OPPT Document Control Office (DCO) in EPA East

Building Rm. 6428, 1201 Constitution Ave., NW., Washington, DC. The DCO is open from 8 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The telephone number for the DCO is (202) 564–8930.

3. *Electronically*. You may submit your comments electronically by e-mail to: oppt.ncic@epa.gov, or mail or deliver your computer disk to the appropriate address identified in this unit. Do not submit any information electronically that you consider to be CBI. Electronic comments must be submitted as an ASCII file avoiding the use of special characters and any form of encryption. Comments and data will also be accepted on standard disks in WordPerfect 6.1/8.0 or ASCII file format. All comments in electronic form must be identified by docket control number OPPTS–00330. Electronic comments may also be filed online at many Federal Depository Libraries.

D. How Should I Handle CBI Information that I Want to Submit to the Agency?

Do not submit any information electronically that you consider to be CBI. You may claim information that you submit to EPA in response to this document as CBI by marking any part or all of that information as CBI. Information so marked will not be disclosed except in accordance with procedures set forth in 40 CFR part 2. In addition to one complete version of the comment that includes any information claimed as CBI, a copy of the comment that does not contain the information claimed as CBI must be submitted for inclusion in the public version of the official record. Information not marked confidential will be included in the public version of the official record without prior notice. If you have any questions about CBI or the procedures for claiming CBI, please consult the DFO listed under **FOR FURTHER INFORMATION CONTACT**.

E. What Should I Consider as I Prepare My Comments for EPA?

We invite you to provide your views on the various options we propose, new approaches we have not considered, the potential impacts of the various options (including possible unintended consequences), and any data or information that you would like the Agency to consider during the development of the final action. You may find the following suggestions helpful for preparing your comments:

1. Explain your views as clearly as possible.

2. Describe any assumptions that you used.

3. Provide copies of any technical information and/or data you used that support your views.

4. If you estimate potential burden or costs, explain how you arrived at the estimate that you provide.

5. Provide specific examples to illustrate your concerns.

6. Offer alternative ways to improve the notice or collection activity.

7. Make sure to submit your comments by the deadline in this notice.

8. To ensure proper receipt by EPA, be sure to identify the docket control number assigned to this action in the subject line on the first page of your response. You may also provide the name, date, and **Federal Register** citation.

II. Background

A. Introduction

EPA's Office of Prevention, Pesticides and Toxic Substances (OPPTS) provided notice on October 31, 1995 (60 FR 55376) (FRL–4987–3) of the establishment of the NAC/AEGL Committee with the stated charter objective as ''the efficient and effective development of AEGLs and the preparation of supplementary qualitative information on the hazardous substances for federal, state, and local agencies and organizations in the private sector concerned with [chemical] emergency planning, prevention, and response.'' The NAC/AEGL Committee is a discretionary Federal advisory committee formed with the intent to develop AEGLs for chemicals through the combined efforts of stakeholder members from both the public and private sectors in a cost-effective approach that avoids duplication of efforts and provides uniform values, while employing the most scientifically sound methods available. An initial priority list of 85 chemicals for AEGL development was published in the **Federal Register** of May 21, 1997 (62 FR 27734) (FRL–5718–9). This list is intended for expansion and modification as priorities of the stakeholder member organizations are further developed. While the development of AEGLs for chemicals are currently not statutorily based, at lease one rulemaking references their planned adoption. The Clean Air Act and Amendments Section 112(r) Risk Management Program states, ''EPA recognizes potential limitations associated with the Emergency Response Planning Guidelines and Level of Concern and is working with other agencies to develop AEGLs. When these values have been developed and peer-reviewed, EPA intends to adopt them, through rulemaking, as the toxic endpoint for substances under this rule (see 61 FR 31685).'' It is believed that other Federal, State and local agencies, and private organizations will also adopt AEGLs for chemical emergency programs in the future.

B. Characterization of the AEGLs

The AEGLs represent threshold exposure limits for the general public and are applicable to emergency exposure periods ranging from 10 minutes to 8 hours. AEGL-1, AEGL-2, and AEGL-3 levels, as appropriate, will be developed for each of five-exposure periods (10 and 30 minutes, 1 hour, 4 hours, and 8 hours) and will be distinguished by varying degrees of severity of toxic effects. It is believed that the recommended exposure levels are applicable to the general population including infants and children, and other individuals who may be sensitive and susceptible. The AEGLs have been defined as follows:

AEGL-1 is the airborne concentration (expressed as parts per million (ppm) or milligrams/meter cubed (mg/m^3) of a substance above which it is predicted that the general population, including susceptible individuals, could experience notable discomfort, irritation, or certain asymptomatic, non-sensory effects. However, the effects are not disabling and are transient and reversible upon cessation of exposure.

AEGL-2 is the airborne concentration (expressed as ppm or mg/m^3) of a substance above which it is predicted that the general population, including susceptible individuals, could experience irreversible or other serious, long-lasting adverse health effects, or an impaired ability to escape.

AEGL-3 is the airborne concentration (expressed as ppm or mg/m^3) of a substance above which it is predicted that the general population, including susceptible individuals, could experience life-threatening health effects or death.

Airborne concentrations below the AEGL-1 represent exposure levels that could produce mild and progressively increasing odor, taste, and sensory irritation or certain non-symptomatic, non-sensory effects. With increasing airborne concentrations above each AEGL level, there is a progressive increase in the likelihood of occurrence and the severity of effects described for each corresponding AEGL level. Although the AEGL values represent threshold levels for the general public, including sensitive subpopulations, it is recognized that certain individuals, subject to unique or idiosyncratic

responses, could experience the effects described at concentrations below the corresponding AEGL level.

C. Development of the AEGLs

The NAC/AEGL Committee develops the AEGL values on a chemical-by-chemical basis. Relevant data and information are gathered from all known sources including published scientific literature, State and Federal agency publications, private industry, public data bases, and individual experts in both the public and private sectors. All key data and information are summarized for the NAC/AEGL Committee in draft form by Oak Ridge National Laboratories together with ''draft'' AEGL values prepared in conjunction with NAC/AEGL Committee members. Both the ''draft'' AEGLs and ''draft'' technical support documents are reviewed and revised as necessary by the NAC/AEGL Committee members prior to formal NAC/AEGL Committee meetings. Following deliberations on the AEGL values and the relevant data and information for each chemical, the NAC/AEGL Committee attempts to reach a consensus. Once the NAC/AEGL Committee reaches a consensus, the values are considered ''Proposed'' AEGLs. The Proposed AEGL values and the accompanying scientific rationale for their development are the subject of this notice.

In this notice, the NAC/AEGL Committee publishes proposed AEGL values and the accompanying scientific rationale for their development for eight hazardous substances. These values represent the sixth set of exposure levels proposed and published by the NAC/AEGL Committee. EPA published the first ''Proposed'' AEGLs for 12 chemicals from the initial priority list in the **Federal Register** of October 30, 1997 (62 FR 58840–58851) (FRL–5737–3); for 10 chemicals in the **Federal Register** of March 15, 2000 (65FR 14186–14196) (FRL–6492–4); for 14 chemicals in the **Federal Register** of June 23, 2000 (65 FR 39263–39277) (FRL–6492–4); for 7 chemicals in the **Federal Register** of December 13, 2000 (65 FR 77866–77874) (FRL–6752–5); and for 18 chemicals in the **Federal Register** of May 2, 2001 (66 FR 21940–21964) (FRL–6776–3) in order to provide an opportunity for public review and comment. In developing the proposed AEGL values, the NAC/AEGL Committee has followed the methodology guidance entitled ''Guidelines for Developing Community Emergency Exposure Levels for Hazardous Substances,'' published by the National Research Council of the National Academy of Sciences (NAS) in 1993. The term Community Emergency Exposure Levels (CELLS) is synonymous with AEGLs in every way. The NAC/AEGL Committee has adopted the term acute exposure guideline levels to better connote the broad application of the values to the population defined by the NAS and addressed by the NAC/AEGL Committee. The NAC/AEGL Committee invites public comment on the proposed AEGL values and the scientific rationale used as the basis for their development.

Following public review and comment, the NAC/AEGL Committee will reconvene to consider relevant comments, data, and information that may have an impact on the NAC/AEGL Committee's position and will again seek consensus for the establishment of Interim AEGL values. Although the Interim AEGL values will be available to Federal, State, and local agencies and to organizations in the private sector as biological reference values, it is intended to have them reviewed by a subcommittee of the NAS. The NAS subcommittee will serve as a peer review of the Interim AEGL values and as the final arbiter in the resolution of issues regarding the AEGL values, and the data and basic methodology used for setting AEGLs. Following concurrence, ''Final'' AEGL values will be published under the auspices of the NAS.

III. List of Chemicals

On behalf of the NAC/AEGL Committee, EPA is providing an opportunity for public comment on the AEGLs for the eight chemicals identified in the following table. This table also provides the fax-on-demand item number for the chemical-specific documents, which may be obtained as described in Unit I.B.

A. Fax-On-Demand Table

TABLE 1.—FAX-ON-DEMAND NUMBER

CAS No.	Chemical name	Fax-On-Demand Item No.
56–23–5	Carbon tetrachloride	4851
75–56–9	Propylene oxide	4864
7637–07–2	Boron trifluoride-dimethyl ether	4892
7782–50–5	Chlorine	4916
7783–81–5	Uranium hexafluoride	4919
10049–04–4	Chlorine dioxide	4926
163702–07–6	Methyl nonafluorobutyl ether (HFE-7100 component)	4933
163702–08–7	Methyl nonafluoroisobutyl ether (HFE-7100 component)	4934

B. Executive Summaries

The following are executive summaries from the chemical-specific technical support documents (which may be obtained as described in Unit I.B. and III.) that support the NAC/AEGL Committee's development of AEGL values for each chemical substance. This information provides the following: A general description of each chemical, including its properties and principle uses; a summary of the rationale supporting the AEGL-1, 2, and 3 concentration levels; a summary table of the AEGL values; and a listing of key references that were used to develop the AEGL values. More extensive toxicological information and additional references for each chemical may be found in the complete technical support documents. Risk managers may be interested to review the complete technical support document for a chemical when deciding issues related

to use of the AEGL values within various programs.

1. *Carbon tetrachloride*—i. *Description*. Carbon tetrachloride (CAS No. 56–23–5) is a colorless, nonflammable, heavy liquid only slightly soluble in water that is used as a laboratory and industrial solvent, an intermediate in the synthesis of trichlorofluoromethane and dichlorodifluoromethane, and was formerly used as a dry-cleaning agent, grain fumigant, anthelmintic, and fire suppressant.

Numerous case reports were available regarding acute inhalation exposure of humans to carbon tetrachloride although most lacked definitive-exposure terms. These reports, however, affirmed the hepatotoxic and renal toxicity of carbon tetrachloride as well as a delayed response for serious and fatal effects. Additionally, data from controlled exposures of humans to carbon tetrachloride were also available.

Animal toxicity data for inhaled carbon tetrachloride indicate hepatotoxic and renal effects, as well as anesthetic-like effects, as primary endpoints. The most sensitive endpoint for evaluating the toxicity of carbon tetrachloride in animals appears to be measurement of serum enzyme activities that reflect hepatic damage. Several studies provided lethality data for various concentrations and exposure durations but data regarding nonlethal effects were limited or available only from long-term exposure studies.

Studies in animals have shown the metabolism and disposition of carbon tetrachloride to be complex and varied among species. Although the precise mechanism of toxicity is equivocal, the biotransformation of carbon tetrachloride by the monooxygenase enzymes (specifically CYP2E1) to reactive intermediates is critical for expression of toxicity. It is this activation process that is critical in modifying the toxic response to carbon tetrachloride.

The AEGL-1 values were based upon a controlled exposure of human subjects to 158 ppm for 30 minutes (Davis, 1934). The exposure resulted in a feeling of nervousness and slight nausea. Development of AEGL values for the various exposure periods was based upon the exponential function, $C^n \times t = k$ (ten Berge et al., 1986), where n = 2.5 as determined by the lethal response of rats to various exposures of carbon tetrachloride. The AEGL-1 values were adjusted by an uncertainty factor of 10 to account for the protection of sensitive individuals (such as users of alcohol) who, due to metabolism and disposition factors, are known to be more susceptible to the toxic effects of carbon tetrachloride.

The AEGL-2 was also based upon human data from controlled exposure experiments in which subjects experienced headache, nausea, and vomiting following 15-minute exposure to 1,191 ppm carbon tetrachloride (Davis, 1934). It is believed that these effects may impair escape. The AEGL-2 values were derived with temporal scaling based upon the exponential function where n = 2.5. The AEGL values were further adjusted by the application of an uncertainty factor of 10 to account for individuals who may be more susceptible to the toxic effects of carbon tetrachloride due to variability in metabolism and disposition of the chemical.

The AEGL-3 was based upon an estimated lethality threshold (1-hour LC_{01} of 5,135.5 ppm) using data from multiple studies on laboratory rats (Adams et al., 1952; Dow Chemical, 1986). Temporal scaling using the exponential function where n = 2.5 was derived from lethality data and used to develop values for AEGL-specific exposure durations. An uncertainty factor of 10 was again applied to account for individuals who may be more susceptible to the toxic effects of carbon tetrachloride (e.g., P-450 induction by ethanol consumption and overall variability in metabolism and disposition of the chemical). Because animal data were used, an uncertainty factor of 3 was applied to account for possible variability in metabolism and the toxic response among species, bringing the total uncertainty factor adjustment to 30. Application of additional uncertainty factors did not appear to be warranted because animal data showed that long-term exposures to carbon tetrachloride above the AEGL-3 values did not result in notable toxic effects.

Although a carcinogenic response following oral exposure of laboratory species has been demonstrated, quantitative data for inhalation exposures were unavailable. However, a unit risk of 1.5E-5 per µg (gram)/m^3 has been established based upon route-to-route extrapolation from carcinogenicity data for oral exposures in various laboratory species. An estimation of AEGLs based upon carcinogenic potential was conducted but the assessment revealed that AEGLs derived from noncarcinogenic toxicity endpoints were more applicable for human health protection relative to adverse effects following acute inhalation exposures.

The AEGL values developed for carbon tetrachloride did not incorporate the possibility of dermal exposure. If the potential for dermal absorption exists, the AEGL values may not be appropriate. Additionally, for AEGL-2 and AEGL-3 exposures, the possibility exists for long-term hepatotoxic effects possibly requiring the need for antioxidant therapy.

The calculated values are listed in Table 2 below:

TABLE 2.—SUMMARY OF PROPOSED AEGL VALUES FOR CARBON TETRACHLORIDE [PPM (MG/M^3)]

Classification	10-minutes	30-minutes	1-hour	4-hour	8-hour	Endpoint (Reference)
AEGL-1 (Nondisabling)	25 (157)	16 (101)	12 (75)	6.9 (43)	5.2 (33)	Nervousness and slight nausea in human subjects exposed for 30 minutes to 158 ppm (Davis, 1934)
AEGL-2 (Disabling)	140 (881)	90 (566)	68 (428)	39 (245)	30 (189)	Nausea, vomiting, headache in human subjects exposed to 1,191 ppm for 15 minutes (Davis, 1934)
AEGL-3 (Lethal)	350 (2,202)	230 (1,447)	170 (1,069)	99 (623)	75 (472)	Lethality in rats; estimated LC_{01} (Adams et al., 1952; Dow Chemical, 1986)

ii. *References*. a. Adams, E.M.; Spencer, H.C.; Rowe, V.K.; McCollister, D.D.; and Irish, D.D. 1952. Vapor toxicity of carbon tetrachloride determined by experiments on laboratory animals. *Archives of Industrial Hygiene and Occupational Medicine*. 6:50–66.

b. Davis, P. A. 1934. Carbon tetrachloride as an industrial hazard. *Journal of the American Medical Association*. 103:962–966.

c. Dow Chemical. 1986. Comparison of the result of exposure of rats and cavies to the vapors of carbon tetrachloride and bromochloromethane. Dated: 7/11/60. EPA-OTS 86-870002363.

d. ten Berge, W.F. 1986. Concentration-time mortality response relationship of irritant and systemically acting vapours and gases. *Journal of Hazardous Materials*. 13:301–309.

2. *Propylene oxide*—i. *Description*. Propylene oxide (CAS No. 75–56–9) is an extremely flammable, highly volatile, colorless liquid. The odor of propylene oxide has been described as sweet and alcoholic in nature, and has reported odor thresholds ranging from 10 ppm to 200 ppm. The primary industrial uses of propylene oxide include its use in the production of polyurethane foams and resins, propylene glycol, functional fluids (such as hydraulic fluids, heat transfer fluids, and lubricants), and propylene oxide-based surfactants. It is also used as a food fumigant, soil sterilizer, and acid scavenger.

Data addressing inhalation toxicity of propylene oxide in humans were limited to one case report, general environmental work surveys, and molecular biomonitoring studies. Studies addressing lethal and nonlethal inhalation toxicity of propylene oxide were available in monkeys, dogs, rats, mice, and guinea pigs. General signs of toxicity following acute exposure to propylene oxide vapor included nasal discharge, lacrimation, salivation, gasping, lethargy/hypoactivity, weakness, and incoordination. Repeated exposures resulted in similar but generally reversible signs of toxicity.

Propylene oxide is a direct alkylating agent that will covalently bind to DNA and proteins. Consequently, it has tested positive in a number of *in vitro* tests, but has produced equivocal results in *in vivo* test systems. Data addressing the potential carcinogenicity of propylene oxide in animals is considered adequate for establishing propylene oxide as a carcinogen in experimental animals.

The proposed AEGL-1 values for propylene oxide were based on an environmental health survey in which 8-hour time weighted averages (TWA) were determined from a 3-day sampling period during which no worker complaints were noted (Chemical Manufacturers Association (CMA), 1998). The highest 8-hour TWA value of 31.8 ppm was chosen for the derivation. An interspecies uncertainty factor was not needed, since the data were from human exposures. An intraspecies uncertainty factor of 3 was applied because the toxic effects (no complaints noted) were less severe than those defined for the AEGL-1 tier. Therefore, a total uncertainty factor of 3 was applied. These values are supported by mouse data from the National Toxicology Program (NTP) (1985) study. Mice were the most sensitive species tested, and dyspnea was the most sensitive endpoint of toxicity following exposure to propylene oxide. Dyspnea was observed in mice exposed for 4 hours to 387 ppm propylene oxide vapor, the lowest concentration tested, but not in mice exposed to 98.5 ppm propylene oxide vapor or less for 6 hours/day, 5 days/week for 2 weeks (NTP, 1985). Therefore, an AEGL-1 can be derived using the exposure concentration of 98.5 ppm for 6 hours (a no-observed-effect level (NOEL) for dyspnea). Following application of a total uncertainty factor of 3 (interspecies uncertainty factor of 1 because mice were the most sensitive laboratory species tested, and available data indicate that mice are equally or slightly more sensitive than humans; an intraspecies uncertainty factor of 3 because the toxic effect (NOEL for dyspnea) was less severe than that defined for the AEGL-1 tier), one obtains AEGL-1 values approximately two-fold greater than those generated using the human data.

The proposed AEGL-2 values are based on the average of AEGL-2 values derived using four propylene oxide exposure concentrations measured in the breathing zone of three workers (380 ppm for 177 minutes, 525 ppm for 121 minutes, 392 ppm for 135 minutes, and 460 ppm for 116 minutes) (CMA, 1998). The industrial hygienist noted that ''the odor was quite strong during the sampling; however, the irritation was not intolerable.'' The exact nature of the irritation, other than the strong odor, was not provided, but occasional eye irritation was noted in the report as the reason for the monitoring program. When deriving AEGL-2 values, an interspecies uncertainty factor was not applicable. An intraspecies uncertainty factor of 3 was applied because the toxic effects (occasional eye irritation; strong odor) were less severe than those defined for the AEGL-2 tier. Therefore, a total uncertainty factor of 3 was applied. The AEGL-2 values are supported by the data from the NTP study in which mice exposed to 387 ppm for 4 hours exhibited dyspnea. Although a NOEL was not established for dyspnea at this concentration, no other effects were noted. In addition, when compared to other studies investigating propylene oxide toxicity in mice, the NTP study reported toxic effects occurring at much lower concentrations than those observed in other studies. Following application of a total uncertainty factor of 3 (interspecies uncertainty factor of 1 because mice were the most sensitive laboratory species tested, and available data indicate that mice are equally or slightly more sensitive than humans; an intraspecies uncertainty factor of 3 because the toxic effect was less severe than that defined for the AEGL-2 tier), one obtains AEGL-2 values approximately 1.4-fold greater than those generated using the human data.

The highest nonlethal concentration in humans was chosen for the AEGL-3 derivation (CMA, 1998). A worker exposed to 1,520 ppm propylene oxide for 171 minutes did not experience mortality; in fact, exposure to this concentration did not cause the worker to cease working. The notation was made by the industrial hygienist that ''the odor was quite strong during the sampling; however, the irritation was not intolerable.'' In deriving AEGL-3 levels, an interspecies uncertainty factor is not needed. An intraspecies uncertainty factor of 3 was applied because the toxic effects (strong odor) were less severe than those defined for the AEGL-3 tier. A modifying factor of 2 was applied to account for the sparse data set (one sample measurement from one worker; old survey from 1968). That these values should be protective of human health is supported by the mouse data. The highest nonlethal concentration in mice was 859 ppm for 4 hours (NTP, 1985). Following application of a total uncertainty factor of 3 (an interspecies uncertainty factor of 1 because mice were the most sensitive laboratory species tested, and available data indicate that mice are equally or slightly more sensitive than humans; an intraspecies uncertainty factor of 3 because the mechanism of toxicity is not expected to differ greatly between individuals), one obtains AEGL-3 values approximately 1.4-fold greater than those generated using the human data.

The experimentally derived exposure values were then scaled to AEGL time frames using the concentration-time relationship given by the equation $C^n \times t = k$, where c = concentration, t = time, k is a constant, and n generally ranges from 1 to 3.5 (ten Berge, 1986). Data appropriate for the derivation of n were extremely limited. Because of the lack of data for empirical derivation of n for propylene oxide, and based on the

similar mechanism of action of propylene oxide as compared to ethylene oxide, the derived value of n for ethylene oxide will be used in the scaling of propylene oxide AEGL values across time. The value of n = 1.2 for ethylene oxide was derived empirically from 1- and 4-hour LC_{50} values for rats. An n value of approximately 1 is further supported by propylene oxide guinea pig data that also suggest a linear relationship. The 10-minute AEGL-1 value was set equal to the 30-minute AEGL value because the NAC considers it inappropriate to extrapolate from the exposure duration of 8 hours to 10 minutes.

A carcinogenic risk assessment of propylene oxide resulted in values that exceed the values based on acute toxicity. Therefore, they are not proposed for AEGL-3. Additionally, while long-term inhalation exposure studies have demonstrated that propylene oxide is carcinogenic in mice and rats, no tumors were observed when 12-week-old male Sprague-Dawley rats were exposed to 433 or 864 ppm propylene oxide for 30 days or 1,724 ppm for 8 days (exposures were for 6 hours/day, 5 days/week) and allowed to die naturally (Sellakumar et al., 1987). This shorter-term exposure suggests a lack of carcinogenic effect following acute exposures.

The calculated values are listed in Table 3 below:

TABLE 3.—SUMMARY OF PROPOSED AEGL VALUES FOR PROPYLENE OXIDE [PPM (MG/M^3)]

Classification	10-minutes	30-minutes	1-hour	4-hour	8-hour	Endpoint (Reference)
AEGL-1 (Nondisabling)	110 (260)	110 (260)	60 (140)	19 (45)	11 (26)	8-hour TWA of 31.8 ppm resulted in no worker complaints (CMA, 1998)
AEGL-2 (Disabling)	1,300 (3,100)	510 (1,200)	290 (690)	91 (220)	51 (120)	Humans: Strong odor and irritation noted in monitoring study; average of AEGL-2 values using 4 exposure concentrations and durations: 380 ppm for 177 minutes, 525 ppm for 121 minutes, 392 ppm for 135 minutes, 460 ppm for 116 minutes (CMA, 1998)
AEGL-3 (Lethal)	2,700 (6,400)	1,100 (2,600)	610 (1,400)	190 (450)	110 (260)	Humans: Highest recorded nonlethal concentration of 1,520 ppm for 171 minutes (CMA, 1998)

ii. *References*. a. CMA. 1998. Chemical Manufacturers Association to National Advisory Committee, (NAC)/AEGLs, Human Experience with Propylene Oxide. Dated: October 16, 1998.

b. NTP. 1985. Toxicology and Carcinogenesis Studies of Propylene Oxide (CAS No. 75–56–9) in F344/N Rats and B6C3F_1 Mice (Inhalation Studies). NTP TR 267, National Institutes of Health (NIH) Publication No. 85–2527, U.S. Department of Health and Human Services, Research Triangle Park, NC.

c. Sellakumar, A.R.; Snyder, C.A.; and Albert, R.E. 1987. Inhalation carcinogenesis of various alkylating agents. *Journal of the National Cancer Institute*. 79:285–289.

d. ten Berge, W.F. 1986. Concentration-time mortality response relationship of irritant and systemically acting vapours and gases. *Journal of Hazardous Materials*. 13:301–309.

3. *Boron trifluoride-dimethyl ether*—i. *Description*. Boron trifluoride-dimethyl ether (CAS No. 7637–07–2) is one of several different complexes that can be formed with boron trifluoride. The complexes are generally formed for ease of handling boron trifluoride. The ether complexes consist of a 1:1 molar ratio of boron trifluoride and the dimethyl or diethyl ether and can dissociate under the proper temperature and pressure conditions. A single study was found that addressed the toxicity of boron trifluoride-dimethyl ether, but it reported only nominal concentrations. Because the complex can dissociate to form boron trifluoride, the AEGL derivations are based upon this one chemical species alone.

Boron trifluoride is a colorless gas with an odor that has been described both as pungent and suffocating or as pleasant. Although the gas is stable in dry air, it immediately forms a dense white mist or cloud when exposed to moist air. It has been reported that upon exposure to even low levels of moisture in the air, boron trifluoride reacts to form the dihydrate, BF3 $2H_2O$. It has been demonstrated that boron trifluoride dihydrate is strongly corrosive to the eyes and skin of rabbits. Boron trifluoride is an excellent catalyst, and has fire retardant and antioxidant properties, nuclear applications, and insecticidal properties.

No definitive data were available addressing the toxicity of boron trifluoride in humans. A statement was made in one study that a worker could detect the odor of boron trifluoride at a concentration of 1.5 ppm (4.1 mg/m^3) (Torkelson et al., 1961). Acute toxicity data were available in dogs, rats, mice, and guinea pigs, but exposure concentrations were generally expressed only in terms of nominal concentrations. Studies which actually measured the exposure concentrations and compared them to nominal concentrations found actual concentrations ranged from 25–56% of nominal (Rusch et al., 1986; Torkelson et al., 1961). Studies identifying endpoints other than those of mortality were limited. No data were available to evaluate the potential for boron trifluoride to cause developmental/reproductive toxicity or carcinogenicity in animals. Boron trifluoride was not mutagenic to several strains of *Salmonella typhimurium*.

The AEGL-1 derivation is based upon lacrimation noted in some rats starting at week 2 of exposure to 6 mg/m^3 boron trifluoride for 6 hours/day, 5 days/week for 13 weeks (exposures were to liquid aerosols of boron trifluoride dihydrate; concentrations reported are based on boron trifluoride) (Rusch et al., 1986; Hoffman and Rusch, 1982). This essentially represents a no-effect level for irritation for an acute exposure. Lacrimation was also reported in some rats exposed to 2 mg/m^3 for 6 hours/day, 5 days/week for 13 weeks, but the observation did not occur until week 10, which is even less relevant to an acute exposure scenario. A total uncertainty factor of 10 was applied. Because the AEGL-1 is based upon essentially a no-effect level for an acute exposure

scenario, an interspecies uncertainty factor of 3 was applied. An intraspecies uncertainty factor of 3 was applied based upon the following reasoning. At higher exposure levels boron trifluoride is an irritant, while at lower levels of exposure it is a renal toxicant. In both cases, the dose response curve is very steep. An example of the steepness of the dose-response curve is seen in the Rusch et al. (1986) study, in which all animals died from renal toxicity as a result of five, 6-hour exposures at 180 mg/m^3, while none even showed signs of renal effects following 10 exposures at 66mg/m^3. Also, none of the animals that died from the exposures at 180 mg/m^3 showed signs of pulmonary irritation even though this exposure was only 16th of the LC_{50} and was for a longer daily duration of 6 hours compared to 4 hours. For these reasons, it was judged that an intra-species uncertainty factor of 3 would protect even the sensitive members of the exposed population. The derived value was set equal to all AEGL time points because the endpoint is a no-effect level for an irritant.

The key study chosen for derivation of the AEGL-2 is the Rusch et al. (1986) study in which five male and five female rats were exposed to 180 mg/m^3 of boron trifluoride for 6 hours/day for 5 days (exposures were to liquid aerosols of boron trifluoride dihydrate; concentrations reported are based on boron trifluoride). Although all rats died from renal toxicity at the end of 5 days of exposure, the only signs observed after 1 day of exposure were those of irritation. It is possible that there may have been some renal toxicity as a consequence of the first day of exposure. The AEGL-2 value was developed by dividing the 180 mg/m^3 exposure level by 2 as a modifying factor since no pathology was conducted after the first exposure; therefore, renal effects could not be characterized or quantified. The resulting value of 90 mg/m^3 is divided by a total uncertainty factor of 10:3 for intraspecies and 3 for interspecies. This provides a starting value of 9 mg/m^3 for a 6-hour exposure. An interspecies uncertainty factor of 3 was used because no effects were seen in rats exposed to 66 mg/m^3 for 6 hours/day for 10 days (Rusch et al., 1986); 1 dog exposed to boron trifluoride at 1,380–2,760 mg/m^3 for 2 hours exhibited only breathing sounds and on necropsy visible signs of irritation to the respiratory tract (DuPont Company, 1948); another group of 2 rats, exposed to 2,760 mg/m^3 for 1 hour exhibited similar necropsy signs (DuPont Company, 1948); and while 1/10 mice died when exposed to 2,100 mg/m^3 for 5.5 hours, none died or even lost body weight when exposed to 350 mg/m^3 for 5.5 hours (Stokinger and Spiegl, 1953). An intraspecies uncertainty factor of 3 was chosen based on the same reasoning provided for the AEGL-1: The dose-response curve was steep for boron trifluoride's actions as both an irritant and renal toxicant. The AEGL-2 starting value of 9 mg/m^3 is in between the 6 hours/day, 5 days/week, 13-week exposure to 17 mg/m^3, which resulted in irritation in rats and renal toxicity in 2/40 rats (one of the rats died of renal toxicity at week 12), and the 6 hours/day, 5 days/week, 13-week exposure to 6 mg/m^3 which resulted only in minimal irritation (lacrimation starting at week 2) (Rusch et al., 1986; Hoffman and Rusch, 1982).

The AEGL-3 derivation is based upon a 4-hour LC_{01} value of 736 mg/m^3 calculated using rat mortality data from Rusch et al. (1986) (exposures were to liquid aerosols of boron trifluoride dihydrate; concentrations reported are based on boron trifluoride). Although other LC_{50} values were available (1-hour LC_{50}s of 1,000 and 1,100 mg/m^3 in rats [Vernot et al, 1977]; 2-hour LC_{50} of 3,460 mg/m^3 in mice [Kasparova and Kirii, 1972], and 4-hour LC_{50} of 109 mg/m^3 in guinea pigs [Stokinger and Spiegl, 1953]), the Rusch et al. (1986) rat study was chosen for the AEGL-3 derivation because it was the best characterized study and the actual exposure concentrations of boron trifluoride were measured. An interspecies uncertainty factor of 10 was applied because the LC_{50} values indicated variability among species in their sensitivity to boron trifluoride. An intraspecies uncertainty factor of 3 was chosen based on the same reasoning provided for the AEGL-1 and AEGL-2: The dose-response curve was steep for boron trifluoride's actions as both an irritant and renal toxicant.

Experimentally derived exposure values are scaled to AEGL time frames using the concentration-time relationship given by the equation $C^n \times t = k$, where C = concentration, t = time, k is a constant, and n generally ranges from 1 to 3.5 (ten Berge, 1986). The value of n could not be empirically derived due to the inadequate data. Therefore, the default value of n = 1 was used for extrapolating from shorter to longer exposure periods and a value of n = 3 was used to extrapolate from longer to shorter exposure periods for the AEGL-2 and AEGL-3. The 10-minute value was set equal to the 30-minute value for the AEGL-2 and AEGL-3 because it is not considered appropriate to extrapolate from a 6-hour or 4-hour exposure duration, respectively, to a 10-minute exposure duration.

The calculated values are listed in Table 4 below:

AEGL values are given in terms of mg/m^3 because exposures were to liquid aerosols of boron trifluoride dihydrate and boron trifluoride gas becomes an aerosol upon contact with moisture in the air.

TABLE 4.—SUMMARY OF PROPOSED AEGL VALUES FOR BORON TRIFLUORIDE (MG/M^3)

Classification	10-minute	30-minute	1-hour	4-hour	8-hour	Endpoint (Reference)
AEGL-1 (Nondisabling)	0.60 mg/m^3	0.60 mg/m^3	0.60 mg/m^3	0.60 mg/m^3	0.60 mg/m^3	Value representing a no-effect level for irritancy following an acute exposure; exposures were to 6 mg/m^3 for 6 hour/day, 5 day/week, for 13 week (Rusch et al., 1986; Hoffman and Rusch, 1982a)
AEGL-2 (Disabling)	21 mg/m^3	21 mg/m^3	16 mg/m^3	10 mg/m^3	6.8 mg/m^3	Signs of irritation and renal toxicity (resulting in death) following exposure to 180 mg/m^3 for 6 hour/day for 5 days (Rusch et al., 1986; Hoffman and Rusch, 1982b)

TABLE 4.—SUMMARY OF PROPOSED AEGL VALUES FOR BORON TRIFLUORIDE (MG/M^3)—Continued

Classification	10-minute	30-minute	1-hour	4-hour	8-hour	Endpoint (Reference)
AEGL-3 (Lethal)	49 mg/m^3	49 mg/m^3	39 mg/m^3	25 mg/m^3	12 mg/m^3	Calculated 4-hour LC_{01} in male and female rats of 736 mg/m^3; based upon analytical concentrations (Rusch et al., 1986; Hoffman, 1981)

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4. *Chlorine*—i. *Description*. Chlorine (CAS No. 7782–50–5) is a greenish-yellow, highly reactive halogen gas with a pungent, suffocating odor. The vapor is heavier than air and will form a cloud in the vicinity of a spill. Like other halogens, chlorine does not occur in the elemental state in nature; it rapidly combines with both inorganic and organic substances. Chlorine is used in the manufacture of a wide variety of chemicals, as a bleaching agent in industry and household products, and as a biocide in water and waste treatment plants.

Chlorine is an irritant to the eyes and respiratory tract; reaction with moist surfaces produces hydrochloric and hypochlorous acids. Its irritant properties have been studied in human volunteers and its acute inhalation toxicity has been studied in several laboratory animal species. The data from the human and laboratory animal studies were sufficient for development of three AEGLs for 5-time periods (i.e., 10 and 30 minutes and 1, 4, and 8 hours). Regression analysis of human data on nuisance irritation responses (itching or burning of the eyes, nose, or throat) for exposure durations of 30–120 minutes and during exposures to 0–2 ppm of chlorine determined that the relationship between concentration and time is approximately $C^2 \times t = k$ (ten Berge and Vis van Heemst, 1983).

The AEGL-1 was based on the observation that exposure of adult human volunteers, including an atopic individual with allergic rhinitis, to 0.5 ppm for 4 hours produced no sensory irritation but did result in transient changes in some pulmonary function parameters for the atopic individual (Rotman et al., 1983). Because both sexes were tested, subjects were undergoing light exercise during exposures on a treadmill or step test that increased the heart rate to 100 beats/minute, making them more vulnerable to sensory irritation, and an exercising susceptible individual did not exhibit adverse effects, no uncertainty factor to account for differences in human sensitivity was applied. The intraspecies uncertainty factor of 1 is supported by another study in which a concentration of 0.4 ppm for 1 hour was a no-effect concentration for changes in pulmonary function parameters in individuals with airway hyperreactivity/asthma (D'Alessandro et al., 1996). Chlorine is a highly irritating and corrosive gas that reacts directly with the tissues of the respiratory tract with no pharmacokinetic component involved in toxicity; therefore, effects are not expected to vary greatly among other susceptible populations. Because the 0.5 ppm concentration appeared to be a threshold concentration for more severe effects in susceptible individuals, regardless of the exposure duration, the 0.5 ppm concentration was applied across all AEGL-1 exposure durations. The 0.5 ppm concentration was considered appropriate for the 8-hour AEGL-1 because effects were not increased in the susceptible individual following a second 4-hour exposure on the same day.

The AEGL-2 values were based on the same study in which healthy human subjects experienced some sensory irritation and transient changes in pulmonary function measurements and a susceptible individual experienced an asthmatic-like attack (shortness of breath and wheezing) at a concentration of 1 ppm after 4 hours of exposure (Rotman et al., 1983). The susceptible individual remained in the exposure chamber for the full 4 hours before the symptoms occurred. Because both sexes were tested, subjects were undergoing light exercise during the exposures, making them more vulnerable to sensory irritation, and an exercising susceptible individual exhibited effects consistent with the definition of the AEGL-2, no uncertainty factor to account for differences in human sensitivity was applied. The intraspecies uncertainty factor of 1 is supported by another study in which a concentration of 1.0 ppm for 1 hour resulted in significant changes in pulmonary function parameters for all five tested individuals who had a history of airway hyperreactivity/asthma; two of the five subjects experienced undefined respiratory symptoms following exposure (D'Alessandro et al., 1996). Chlorine is a highly irritating and corrosive gas that reacts directly with the tissues of the respiratory tract with no pharmacokinetic component involved in toxicity; therefore, effects are not expected to vary greatly among other susceptible populations. Time-scaling was considered appropriate for the AEGL-2 as the AEGL-2 is defined as the threshold for irreversible effects which in the case of irritants generally involves tissue damage. Although the endpoint used in this case, wheezing and a significant increase in airways resistance, may be below the AEGL-2

definition, it is assumed that some biomarkers of tissue irritation would be present in the airways and lungs. The 4-hour 1 ppm concentration was scaled to the other time periods using the $C^2 \times t = k$ relationship. The scaling factor was based on regression analyses of concentrations and exposure durations that attained nuisance levels of irritation in human subjects. The 10-minute value was set equal to the 30-minute value in order to not exceed the highest exposure of 4.0 ppm in controlled human studies.

In the absence of human data, the AEGL-3 values were based on animal lethality data. The mouse was not chosen as an appropriate model for lethality because mice often showed delayed deaths which several authors attributed to bronchopneumonia. Because the mouse was shown to be more sensitive than other mammals (dog and rat) to irritant gases including chlorine and does not provide an appropriate basis for quantitatively predicting mortality in humans, a value below that resulting in no deaths in the rat (213 and 322 ppm in two studies) and above that resulting in no deaths in the mouse (150 ppm) for a period of 1 hour was chosen (MacEwen and Vernot, 1972; Zwart and Woutersen, 1988). The AEGL-3 values were derived from a 1-hour concentration of 200 ppm. This value was divided by a total uncertainty factor of 10:3 to extrapolate from rats to humans (interspecies values for the same endpoint differed by a factor of approximately 2 within each of several studies), and by an uncertainty factor of 3 to account for differences in human sensitivity. The susceptibility of asthmatics relative to healthy subjects when considering lethality is unknown, but the data from two studies with human subjects showed that doubling a no-effect concentration for irritation and bronchial constriction resulted in potentially serious effects in the asthmatics but not in the normal individuals. Time-scaling was considered appropriate for the AEGL-3 because tissue damage is involved (data in animal studies clearly indicate that time-scaling is appropriate when lung damage is involved). The AEGL-3 values for the other exposure times were calculated based on the $C^2 \times t = k$ relationship which was derived based on the endpoint of irritation from a study with humans.

The calculated values are listed in Table 5 below:

TABLE 5.—SUMMARY OF PROPOSED AEGL VALUES FOR CHLORINE [PPM (MG/M^3)]

Classification	10-minute	30-minute	1-hour	4-hour	8-hour	Endpoint (Reference)
AEGL-1[a] (Nondisabling)	0.50 (1.5)	0.50 (1.5)	0.50 (1.5)	0.50 (1.5)	0.50[b] (1.5)	No to slight changes in pulmonary function parameters in humans (Rotman et al., 1983; D'Alessandro et al., 1996)
AEGL-2 (Disabling)	2.8 (8.1)	2.8 (8.1)	2.0 (5.8)	1.0 (2.9)	0.70 (2.0)	Asthmatic-like attack in human subjects (Rotman et al., 1983; D'Alessandro et al., 1996)
AEGL-3 (Lethal)	50 (145)	28 (81)	20 (58)	10 (29)	7.1 (21)	Lethality—rat (MacEwen and Vernot, 1972; Zwart and Woutersen, 1988)

[a] The distinctive, pungent odor of chlorine will be noticeable to most individuals at these concentrations.
[b] Because effects were not increased following an interrupted 8-hour exposure of anatopic individual to 0.5 ppm, the 8-hour AEGL-1 was set equal to 0.5 ppm.

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e. Zwart, A. and Woutersen, R.A. 1988. Acute inhalation toxicity of chlorine in rats and mice: time-concentration-mortality relationships and effects on respiration. *Journal of Hazardous Materials*. 19:195–208.

5. *Uranium hexafluoride*—i. *Description.* Uranium hexafluoride (CAS No. 7783–81–5) is a volatile solid. It is one of the most highly soluble industrial uranium compounds and, when airborne, hydrolyzes rapidly on contact with moisture to form hydrofluoric acid (HF) and uranyl fluoride (UO_2F_2) as follows:

$$UF_6 + 2H_2O \rightarrow UO_2F_2 + 4HF$$

Thus, an inhalation exposure to uranium hexafluoride is actually an inhalation exposure to a mixture of both fluorides. Pulmonary irritation, corrosion, or edema may occur from the hydrofluoric acid component and/or renal injury may be observed from the uranium component. As concentration is decreased and duration is increased, the effects of hydrogen fluoride are reduced, and the effects of the uranium component may be increased (Spiegel, 1949).

In the absence of relevant chemical-specific data for derivation of AEGL-1 values for uranium hexafluoride, a modification of the AEGL-1 values for hydrogen fluoride was used to derive AEGL-1 values for uranium hexafluoride. The use of hydrogen fluoride as a surrogate for uranium hexafluoride was deemed appropriate since it is likely that it is the hydrolysis product, HF, that is responsible for adverse effects. The hydrogen fluoride AEGL-1 values were based on the threshold for pulmonary inflammation in healthy human adults (Lund et al., 1999). Since a maximum of four moles of hydrogen fluoride are produced for every mole of uranium hexafluoride hydrolyzed, a stoichiometric adjustment factor of 4 was applied to the hydrogen fluoride AEGL-1 values to approximate AEGL-1 values for uranium hexafluoride. AEGL-1 values were derived only for the 10-minute, 30-minute, and 1-hour time points since it is likely that renal toxicity may be more relevant at the longer time points and no data exist for renal toxicity consistent with the definition of AEGL-1.

The AEGL-2 was based on renal pathology in dogs exposed to 192 mg/m^3 UF_6 for 30 minutes (Morrow et al., 1982). An uncertainty factor of 3 was used to extrapolate from animals to humans, and an uncertainty factor of 3 was also applied to account for sensitive individuals (total uncertainty factor = 10). This total uncertainty factor is considered sufficient since the observed renal pathology is generally considered reversible and thus this effect may be below the definition of AEGL-2. Furthermore, the use of a larger total uncertainty factor would yield AEGL-2 values below or approaching the AEGL-1 values. The concentration-exposure time relationship for many irritant and systemically acting vapors and gases may be described by $C^n \times t = k$, where the exponent, n, ranges from 0.8 to 3.5 (ten Berge et al., 1986). To obtain conservative and protective AEGL values in the absence of an empirically derived chemical-specific scaling exponent, temporal scaling was performed using n = 3 when extrapolating to shorter time points and n = 1 when extrapolating to longer time points using the $C^n \times t = k$ equation. (Although a chemical-specific exponent of 0.66 was derived from rat lethality data in which the endpoint was pulmonary edema, the default values were utilized for time-scaling AEGL-2 values since the endpoints for AEGL-2 (renal toxicity) and death (pulmonary edema) involve different mechanisms of action).

The AEGL-3 was based on an estimated 1-hour threshold for death in rats ($_{13}$ LC_{50} of 365 mg/m^3) (Leach et al, 1984). This approach is considered appropriate due to the steepness of the concentration-response curve for lethality in rats after exposure to UF_6. An uncertainty factor of 3 was used to extrapolate from animals to humans; the interspecies uncertainty factor of 3 is considered sufficient since the cause of death (pulmonary edema) is due to the hydrogen fluoride hydrolysis product, and lethality studies of hydrogen fluoride suggest that the rat was approximately 3-times less sensitive than the most sensitive (hypersusceptible) species (mouse) (EPA, 2001). An uncertainty factor of 3 was also applied to account for sensitive individuals since death is due to severe tissue damage resulting in pulmonary edema from the HF hydrolysis product (total uncertainty factor = 10). Furthermore, the total uncertainty factor of 10 is considered sufficient in light of the steep concentration-response curve. The value was then scaled to the 10-minute, 30-minute, 4-hour, and 8-hour time points, using $C^{0.66} \times t = k$. The exponent of 0.66 was derived from rat lethality data ranging from 2 minutes to 1 hour exposure duration in the key study.

The calculated values are listed in Table 6 below:

TABLE 6.—SUMMARY OF PROPOSED AEGL VALUES FOR URANIUM HEXAFLUORIDE (MG/M^3)

Classification	10-minute	30-minute	1-hour	4-hour	8-hour	Endpoint (Reference)
AEGL-1 (Nondisabling)	3.6 mg/m^3	3.6 mg/m^3	3.6 mg/m^3	NR	NR	Modification of hydrogen fluoride AEGL-1 values (EPA, 2001)
AEGL-2 (Disabling)	28 mg/m^3	19 mg/m^3	9.6 mg/m^3	2.4 mg/m^3	1.2 mg/m^3	Renal tubular pathology in dogs (Morrow et al., 1982)
AEGL-3 (Lethality)	550 mg/m^3	100 mg/m^3	36 mg/m^3	4.4 mg/m^3	1.6 mg/m^3	Estimated 1-hour NOEL for death in the rat (Leach et al., 1984)

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e. ten Berge, W.F.; Zwart, A.; and Appelman, L.M. 1986. Concentration-time mortality response relationship of irritant and systemically acting vapours and gases. *Journal of Hazardous Materials*. 13:301–309.

f. EPA. 2001. Acute exposure guideline levels for hydrogen fluoride. (Interim Draft 2:7/2001).

6. *Chlorine dioxide*—i. *Description*. Chlorine dioxide (CAS No. 10049–04–4) is a yellow to reddish-yellow gas at room temperature. It has an unpleasant odor, similar to the odor of chlorine and reminiscent of nitric acid. It is a respiratory irritant. Pure chlorine dioxide is stable in the dark and unstable in light. Chlorine dioxide dissociates in water into chlorite and chloride, and to a lesser extent into chlorate. The major use of chloride dioxide is that of a drinking water disinfectant. Other uses include the bleaching of textiles, paper pulp, flour, cellulose, leather, fats, oils, and beeswax; taste and odor control of water; an oxidizing agent; and the manufacture of chlorite salts. The acute inhalation data base for chlorine dioxide is quite sparse for both human and animal exposures.

The AEGL-1 was based on slight salivation, slight lacrimation, and slight red-ocular discharge in rats exposed to 3 ppm chlorine dioxide for 6 hours (DuPont, 1955). A total combined uncertainty factor of 10 was applied to account for interspecies and intraspecies variability, and a modifying factor of two was applied to account for the sparse data base and the resulting uncertainty about the most sensitive species. Thus, the total uncertainty/modifying factor is 20. Chlorine dioxide is a highly reactive chemical. The clinical signs of minor irritation are likely caused by a direct chemical effect on external tissue. This minor irritation is not likely to vary greatly among species or among individuals. The AEGL-1 value was held constant across all time points since minor irritation is not likely to be time dependent.

The AEGL-2 was based on lacrimation, salivation, dyspnea, weakness, and pallor in rats exposed to

12 ppm chlorine dioxide for 6 hours (DuPont, 1955). A total combined uncertainty factor of 10 was applied to account for interspecies and intraspecies variability, and a modifying factor of 2 was applied to account for the sparse data base and the resulting uncertainty about the most sensitive species. Thus, the total uncertainty/modifying factor is 20. This total adjustment factor of 20 is reasonable since the derived 4 hour AEGL-2 value is 0.69 ppm yet rats repeatedly exposed to 3 ppm chlorine dioxide (Dupont, 1955), 6 hours/day for 10 days showed only minor irritation (slight salivation, slight lacrimation, and slight red-ocular discharge on the first day of the study). Even allowing for differences in response between species and individuals, this comparison indicates that the derived AEGL-2 values are reasonable and the threshold for disabling susceptible humans should be above this level. The use of a higher combined uncertainty factor/modifying factor of 200 would give a 4 hour AEGL value of 0.069 ppm yet when rats were exposed to 0.1 ppm of chlorine dioxide for 5 hours/day for 10 weeks, no clinical signs were observed during treatment and at necropsy (Dalhamn, 1957). This comparison shows that a combined uncertainty/modifying factor of 200 is excessively large. The concentration-exposure time relationship for many irritant and systemically acting vapors and gases may be described by $C^n \times t = k$, where the exponent, n, ranges from 0.8 to 3.5 (ten Berge et al., 1986). To obtain conservative and protective AEGL values in the absence of an empirically derived chemical-specific scaling exponent, temporal scaling was performed using n = 3 when extrapolating to shorter time points (30-minutes, 1-hour, and 4-hours) and n = 1 (8-hours) when extrapolating to longer time points using the $C^n \times t = k$ equation. The 30-minute AEGL-2 value was also adopted as the 10-minute AEGL-2 value due to the added uncertainty of extrapolating from a 6-hour time point to 10-minutes.

The AEGL-3 was based on a study showing no deaths in rats exposed to 26 ppm chlorine dioxide for 6 hours (DuPont, 1955). A total combined uncertainty factor of 10 was applied to account for interspecies and intraspecies variability, and a modifying factor of 2 was applied to account for the sparse data base and the resulting uncertainty about the most sensitive species. Thus, the total uncertainty/modifying factor is 20. The total factor of 20 is considered adequate. Using a larger combined uncertainty/modifying factor of 200 would give a 4 hour AEGL-3 value of 0.15 ppm. The value of 0.15 ppm is too low, because rats exposed to 0.1 ppm of chlorine dioxide for 5 hours/day for 10 weeks showed no clinical signs during treatment or at necropsy (Dalhamn, 1957). This comparison shows that a combined uncertainty/modifying factor of 200 is excessively large. The concentration-exposure time relationship for many irritant and systemically acting vapors and gases may be described by $C^n \times t = k$, where the exponent, n, ranges from 0.8 to 3.5 (ten Berge et al., 1986). To obtain conservative and protective AEGL values in the absence of an empirically derived chemical-specific scaling exponent, temporal scaling was performed using n = 3 when extrapolating to shorter time points (30-minutes, 1-hour, and 4-hours) and n = 1 (8-hours) when extrapolating to longer time points using the $C^n \times t = k$ equation. The 30-minute AEGL-3 value was also adopted as the 10-minute AEGL-3 value due to the added uncertainty of extrapolating from a 6-hour time point to 10-minutes.

The calculated values are listed in Table 7 below:

TABLE 7.—SUMMARY OF PROPOSED AEGL VALUES FOR CHLORINE DIOXIDE [PPM (MG/M^3)]

Classification	10-minute	30-minute	1-hour	4-hour	8-hour	Endpoint (Reference)
AEGL-1 (Nondisabling)	0.15 (0.41)	0.15 (0.41)	0.15 (0.41)	0.15 (0.41)	0.15 (0.41)	Slight salivation, slight lacrimation, and slight red-ocular discharge in rats exposed to 3 ppm for 6 hours (DuPont, 1955)
AEGL-2 (Disabling)	1.4 (3.9)	1.4 (3.9)	1.1 (3.0)	0.69 (1.9)	0.45 (1.2)	Lacrimation, salivation, dyspnea, weakness, and pallor in rats exposed to 12 ppm for 6 hours (DuPont, 1955)
AEGL-3 (Lethal)	3.0 (8.3)	3.0 (8.3)	2.4 (6.6)	1.5 (4.1)	0.98 (2.7)	No lethality in rats exposed to 26 ppm for 6 hour (DuPont, 1955)

ii. *References.* a. Dalhamn, T. 1957. Chlorine Dioxide: Toxicity in animal experiments and industrial risks. *Archives of Industrial Health.* 15:101–107.

b. DuPont. 1955. Summary of Toxicological Evaluations of Chlorine Dioxide. Haskell Laboratory for Toxicology and Industrial Medicine. Haskell Lab Report No. 80-55. E.I. du Pont de Nemours and Company, Inc. Wilmington, DE.

c. ten Berge, W.F.; Zwart, A.; and Appleman, L.M. 1986. Concentration-time mortality response relationship of irritant and systemically acting vapors and gases. *Journal of Hazardous Materials.* 13:301–310.

7. and 8. *Methyl nonafluorobutyl ether and Methyl nonafluoroisobutyl ether*—i. *Description.* HFE-7100 is a mixture of methyl nonafluorobutyl (CAS No. 163702–07–6) and methyl nonafluoroisobutyl (CAS No. 163702–08–7) ethers in ratios of 30–50 and 50–70%, respectively. This mixture has been developed as a replacement for presently used chlorofluorocarbons and other ozone-depleting chemicals. It is used in industrial situations as a cleaning agent, lubricant carrier, drying agent, specialty solvent, and heat-transfer medium. It is a volatile liquid with a slight ethereal odor. No information on production was located.

Except for a single monitoring study conducted by 3M Company and reported by AIHA (1999) in which exposures were noted to be below 50 ppm, no information on human exposures was located. Animal data using the rat as the model addressed anesthetic properties, toxicity, neurotoxicity, and genotoxicity. A study with the beagle addressed cardiac

sensitization. HFE-7100 is practically nontoxic; it does not have anesthetic properties and is not a cardiac sensitizer. No information useful for time-scaling across the AEGL exposure durations was available.

The AEGL-1 value is based on a subchronic study with the rat (Coombs et al., 1996b). In this study, rats were exposed to concentrations up to 15,159 ppm for 6 hours/day, 5 days/week for 13 weeks. This concentration was not neurotoxic. Only reversible organ weight increases were observed and these were attributed to the repeated nature of the exposure. Because the concentration was basically a NOAEL, the exposures were repeated, and uptake is greater in the rodent than in primates, based on the higher respiratory rate and cardiac output of rodents compared with primates, an interspecies uncertainty factor of 1 was applied. Studies addressing neurotoxicity and cardiac sensitization and studies with pregnant rats failed to identify significant toxicological endpoints. Therefore, an intraspecies uncertainty factor of 3 was applied. Because human data are very limited and because some of the key studies used limited numbers of animals, a modifying factor of 2 was applied. The resultant value is 2,500 ppm. Time-scaling may not be relevant for anesthetics and halogenated hydrocarbons as blood concentrations of these chemicals rapidly reach equilibrium and do not greatly increase as exposure duration is increased. The presence of the perfluoro group of HFE-7100 limits its solubility in biological fluids. Furthermore, the repeated nature of the exposures of the key study support the use of the same value across all time points. Therefore, the 2,500 ppm concentration is applicable for all AEGL-1 time points.

The AEGL-2 value is based on a 10-minute cardiac sensitization test with beagles (Kenny et al., 1996) and is supported by a 4-week repeat exposure study with the rat (Coombs et al., 1996a). Six male beagles exposed to 48,900 ppm for 10 minutes and challenged with an adrenaline dose of 1–12 μg/kilogram (kg) (individualized for each dog) did not show cardiac sensitization. However, all of the beagles exhibited signs of restlessness, agitation, tremors, and muscle rigidity. These signs were described following the second challenge, but may have been present pre-challenge. All beagles recovered fully and were used for subsequent studies. The cardiac sensitization test is very conservative as the levels of adrenaline administered represent an approximate 10-fold excess over blood concentrations that would be achieved endogenously in dogs or humans, even in highly stressful situations. Because this is a conservative endpoint (the dogs exhibited clinical signs but fully recovered), the test addresses the stress that might be experienced in an escape situation, and the dog heart is considered an appropriate model for the human heart, an interspecies uncertainty factor of 1 was applied. Heart patients would not be at extra risk because HFE-7100 is not a cardiac sensitizer and studies with pregnant rats failed to identify significant toxicological endpoints. Therefore, an intraspecies uncertainty factor of 3 was applied to protect potentially susceptible individuals. Because human data are very limited and because some of the key studies used limited numbers of animals, a modifying factor of 2 was applied. The resulting value is 8,200 ppm. Time-scaling may not be relevant for anesthetics and halogenated hydrocarbons as blood concentrations of these chemicals rapidly reach equilibrium and do not greatly increase as exposure duration is increased. Furthermore the presence of the perfluoro group of HFE-7100 limits its solubility in biological fluids. Therefore, the 8,200 ppm concentration is applicable for all AEGL-2 time points. The values are supported by a study in which rats were exposed to concentrations up to 30,000 ppm for 6 hours/day, 5 days/week for 4 weeks. These rats exhibited reversible liver hypertrophy which is attributable to the repeated nature of the exposures (Coombs et al., 1996a). The repeated nature of this study supports using a single value across the AEGL-2 time points.

The AEGL-3 value is based on the same cardiac sensitization study with beagles (Kenny et al., 1996) and is supported by an acute inhalation study with the rat (3M Company, 1995). Prior to the second challenge dose of adrenaline during a cardiac sensitization test, one of two dogs exposed to 89,300 ppm for 10 minutes exhibited severe clinical signs including restlessness, cold extremities, limb rigidity, head and whole-body tremors, head shaking, arched back, agitation, and salivation. The second dog survived the second challenge dose of adrenaline but exhibited similar adverse clinical signs. The cardiac sensitization test is very conservative as the levels of adrenaline administered represent an approximate 10-fold excess over blood concentrations that would be achieved endogenously in dogs or humans, even in highly stressful situations. Because this is a conservative endpoint (the dogs exhibited clinical signs but fully recovered), the test addresses the stress that might be experienced in an escape situation, and the dog heart is considered an appropriate model for the human heart, an interspecies uncertainty factor of 1 was applied. Heart patients would not be at extra risk because HFE-7100 is not a cardiac sensitizer and studies with pregnant rats failed to identify significant toxicological endpoints. Therefore, an intraspecies uncertainty factor of 3 was applied to protect potentially susceptible individuals. Because human data are very limited and because some of the key studies used limited numbers of animals, a modifying factor of 2 was applied. Time-scaling may not be relevant for anesthetics and halogenated hydrocarbons as blood concentrations of these chemicals rapidly reach equilibrium and do not greatly increase as exposure duration is increased. Therefore, the resulting 15,000 ppm concentration is applicable for all AEGL-3 time points. The 89,300 ppm concentration may be a conservative estimate of the threshold for lethality as rats survived a 4-hour exposure to 100,000 ppm (3M Company, 1995).

The calculated values are listed in Table 8 below:

TABLE 8.—SUMMARY OF PROPOSED AEGL VALUES FOR HFE-7100 [PPM (MG/M^3]

Classification	10-minute	30-minute	1-hour	4-hour	8-hour	Endpoint (Reference)
AEGL-1 (Nondisabling)	2,500 (25,550)	2,500 (25,550)	2,500 (25,550)	2,500 (25,550)	2,500 (25,550)	Reversible organ weight changes, repeated exposures, rat (Coombs et al., 1996b)

TABLE 8.—SUMMARY OF PROPOSED AEGL VALUES FOR HFE-7100 [PPM (MG/M^3]—Continued

Classification	10-minute	30-minute	1-hour	4-hour	8-hour	Endpoint (Reference)
AEGL-2 (Disabling)	8,200 (84,000)	8,200 (84,000)	8,200 (84,000)	8,200 (84,000)	8,200 (84,000)	Clinical signs, cardiac sensitization test, dog (Kenny et al., 1996)
AEGL-3 (Lethal)	15,000 (150,000)	15,000 (150,000)	15,000 (150,000)	15,000 (150,000)	15,000 (150,000)	Severe clinical signs, cardiac sensitization test, dog (Kenney et al., 1996)

ii. *References.* a. 3M Company. 1995. Acute inhalation toxicity for HFE-7100 in the rat. Memo, 3M Company, Toxicology Services. 3M Center, St. Paul, MN.

b. AIHA. 1999. Workplace Environmental Exposure Levels: HFE-7100. American Industrial Hygiene Association, Fairfax, VA.

c. Coombs, D.W.; Shepherd, C.K.; Bannerman, M.; Hardy, C.J.; Crook, D.; Hall, M.; Hughes, E.W.; and Gopinath, C. 1996a. T-6334: 28-Day repeat dose inhalation toxicity study in rats. MIN 181/952688. Huntingdon Life Sciences, Huntingdon, Cambridgeshire, England.

d. Coombs, D.W.; Shepherd, C.K.; Bannerman, M.; Hardy, C.J.; Crook, D.; Hall, M.; and Healey, G.F. 1996b. T-6334: 13-Week repeat dose inhalation toxicity study in rats. MIN 196/961181. Huntingdon Life Sciences, Huntingdon, Cambridgeshire, England.

e. Kenny, T.J.; Shepherd, C.K.; Bannerman, M.; Hardy, C.J.; and Gilkison, I.S. 1996. T-6334: Assessment of cardiac sensitization potential in dogs. MIN 182/953117. Huntingdon Life Sciences, Limited.

IV. Next Steps

The NAC/AEGL Committee plans to publish ''Proposed'' AEGL values for five-exposure periods for other chemicals on the priority list of 85 in groups of approximately 10 to 20 chemicals in future **Federal Register** notices during the calendar year 2002.

The NAC/AEGL Committee will review and consider all public comments received on this notice, with revisions to the ''Proposed'' AEGL values as appropriate. The resulting AEGL values will be established as ''Interim'' AEGLs and will be forwarded to the National Research Council, National Academy of Sciences (NRC/NAS), for review and comment. The ''Final'' AEGLs will be published under the auspices of the NRC/NAS following concurrence on the values and the scientific rationale used in their development.

List of Subjects

Environmental protection, Acute exposure guideline levels, Hazardous substances.

Dated: February 1, 2002.

Susan B. Hazen,

Acting Assistant Administrator for Prevention, Pesticides and Toxic Substances.

[FR Doc. 02–3774 Filed 2–14–02; 8:45 am]

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ENVIRONMENTAL PROTECTION AGENCY

[FRL–7146–3]

42 U.S.C. 122(I), Proposed Administrative Agreement for Collection of CERCLA Response and Oversight Costs

AGENCY: Environmental Protection Agency (USEPA).

ACTION: Proposed CERCLA 122(h) Administrative Agreement.

SUMMARY: USEPA is proposing to execute an Administrative Agreement (Agreement) under Section 122 of CERCLA for collection of a percentage of response and oversight costs at the Chippewa Avenue Area Groundwater Contamination Superfund Site. The Respondent has agreed to pay $65,000 out of total unrecovered response and oversight costs of approximately $695,582,81, and in return will receive a covenant not to sue and contribution protection from USEPA. USEPA today is proposing to execute this Agreement because the settlement, in combination with $1,000,000 received for the Site pursuant to an April 15, 1993, settlement of a USEPA claim in the LTV Steel bankruptcy proceedings, achieves collection of approximately 63% of total Site costs.

DATES: Comments on this proposed settlement must be received on or before March 18, 2001.

ADDRESSES: Copies of the proposed settlement are available at the following address for review: (It is recommended that you telephone Fouad Dababneh at (312) 353–3944 before visiting the Region V Office). Fouad Dababneh, U.S. Environmental Protection Agency, Region V, 77 W. Jackson Boulevard, (SR–6J), Chicago, Illinois 60604–3590, (312) 353–3944.

Comments on this proposed settlement should be addressed to: (Please submit an original and three copies, if possible) Fouad Dababneh, U.S. Environmental Protection Agency, Region V, 77 W Jackson Boulevard, (SR–6J), Chicago, Illinois 60604–3590, (312) 353–3944.

FOR FURTHER INFORMATION CONTACT: Fouad Dababneh at (312) 353–3944.

SUPPLEMENTARY INFORMATION: The Chippewa Site is an approximately four square mile area centered on the intersection of Chippewa and Main streets in South Bend, Indiana. Located within the Chippewa Avenue Area Groundwater contamination Site are the Rum Village and South Well Field municipal drinking water supply wells. In 1997, the City of South Bend and the Indiana Department of Environmental Management (IDEM) requested EPA assistance in investigating dissolved solvents contamination in the South and Rum Village well fields. Accordingly, in response to the release or threatened release of hazardous substances at or from the Site, EPA undertook response actions at the Site pursuant to Section 104 of CERCLA, 42 U.S.C. 9604.

In October 1997, Region 5 initiated a groundwater investigation to identify the types and concentrations of groundwater contaminants in the vicinity of the South Well Field. Data from the groundwater investigation was intended to be used in the design of an interim treatment system for the South Well Field. In 1998, however, IDEM entered into an agreement with certain parties, including The Toro Corporation, for installation of an air stripper treatment system for the two well fields. The air stripper system has since been installed. As a result, Region 5 does not expect to incur additional costs for the Site.

As a part of an April 15, 1993, bankruptcy settlement with LTV Steel, AM General (a subsidiary of LTV Steel)