UNITED STATES COURT OF APPEALS
FOR THE DISTRICT OF COLUMBIA CIRCUIT

NATURAL RESOURCES DEFENSE COUNCIL Petitioner, v. UNITED STATES ENVIRONMENTAL PROTECTION AGENCY, *et al.*, Respondents.	)	Docket No. 04-1438

DECLARATION OF REVA RUBENSTEIN, PH.D. ON BEHALF OF RESPONDENT UNITED STATES ENVIRONMENTAL PROTECTION AGENCY

Pursuant to 28 U.S.C. § 1746, I, Reva Rubenstein, affirm and state as follows:

1. I am a Health Specialist in the Energy and Resources: Climate Change Science Group at ICF International, a contractor to the United States Environmental Protection Agency ("EPA"). ICF International (www.icfi.com) delivers consulting services and technology solutions in defense, energy, environment, homeland security, social programs, and transportation. ICF has extensive experience in providing technical support for the regulations governing the phase out of methyl bromide for EPA's Stratospheric Protection Division and analyzing the human health risks of substitutes for ozone-depleting substances ("ODS") over the past two decades. ICF also developed and maintains the Atmospheric Health Effects Framework model ("AHEF"), a peer-reviewed econometric model that estimates the skin cancers and other health effects resulting from ozone depletion.

2. I obtained my S.B. in Chemistry from Brooklyn College in Brooklyn, NY and my Ph.D. in Physical Chemistry from the Polytechnic Institute of Brooklyn (now Polytechnic University of New York) in Brooklyn, NY. Between 1992 and 1997, I taught a course entitled "Introduction to Environmental Management" as an Adjunct Professor for the Environmental Management Graduate Program at the University of Maryland, University College. In 1996, I taught "Risk Assessment in Decision Making" at EPA Headquarters.

3. I have been a Health Specialist for ICF since 2001. Before that, I was the Science Advisor to the Director of EPA's Stratospheric Protection Division from 1989 to 2001. My duties at EPA included analyzing toxicity and exposure reports submitted to EPA under the Significant New Alternatives Policy ("SNAP") program for both new and existing chemical alternatives, providing recommendations to the Director of the appropriate control measures for use, and evaluating new data concerning the effects of

ozone depletion. Risk assessments (hazard and exposure components) were completed for alternatives to chlorofluorcarbons ("CFCs") used in a variety of industry sectors. In 1987, I received the EPA Bronze Medal for Commendable Service for the risk assessment of hazardous waste constituents and, in 1997, I received the EPA Bronze Medal for Commendable Service for contributions to the research strategies for the protection of stratospheric ozone.

4. Beginning in the late 1980s and continuing throughout my tenure at EPA's Stratospheric Protection Division ("SPD"), I was responsible for developing the risk assessment to support EPA's 1988 Regulatory Impact Analysis: Protection of Stratospheric Ozone. This RIA formed the basis for all subsequent analyses of costs and benefits of SPD's programs. The health benefits analysis presented in these documents was based on the AHEF model. While at SPD, I reviewed the epidemiological and health effects literature that supported the AHEF, managed the grants that EPA had in place to establish the reaction kinetics that govern the atmospheric chemistry of ozone depletion, and managed the modeling tasks that were being performed by ICF to develop a robust modeling framework.

5. Since joining ICF in 2001, I have overseen the drafting of numerous risk screens for the SNAP program and have reviewed numerous toxicological peer-reviewed studies of substitutes for ODS. My role in the on-going AHEF development process is to provide guidance on updates to the model and to work with the team of epidemiologists and other health professionals, chemists, and economists that continue to support SPD's ozone protection initiatives. In 2005, I led a review of the EPA Office of Pesticide Program's (OPP) methyl bromide risk assessment for SPD.

6. I have authored three and co-authored five publications on the health risks of ozone depletion, as well as the toxicity of ODS and substitutes for ODS. One paper ("Regulatory aspects of hydrofluorocarbons") was published in the 9th volume of *Inhalation Toxicity*. I have also submitted papers for the Halon Options Technical Working Conferences (1995, 1998, 1999), the 11th International Conference on Carcinogenesis and Risk Assessment (1997), and the Earth Technologies Forum (1998). In addition, I have presented papers at the International Symposium of Solvent Substitutes (1997), the Annual Conference of the International Mobile Air Conditioning Association (1997), and the International Conference on Ozone Protection Technologies (1995, 1996, 1997). I was a member of the United Nations Environment Program's (UNEP) Halon Technical Option Committee and a member of the LEED™ (Leadership in Energy and Environmental Design) Technical and Scientific Advisory Committee (TSAC), which is part of the U.S. Green Building Council. I have also served as a member of the National Fire Protection Association Halon Alternative Protection Options HAO-AAA Technical Committee (1999 to 2002) and as a member of the Committee on Fire Suppression Substitutes and Alternatives to Halon (1996), which is a committee of the National Research Council's Naval Studies Board.

7. As requested by EPA staff, and in order to prepare this declaration, I have reviewed the Court's decision, *NRDC v. EPA*, 443 F.3d 476 (D.C. Cir. 2006), Petitioner's Petition for Rehearing or Rehearing En Banc, and the affidavits of Dr. Sasha Madronich ("Madronich Aff.") and Dr. Louis Anthony Cox, Jr ("Cox Aff."). I have also reviewed portions of the Economic Impact Analysis for Methyl Bromide Allocation Within the United States, ("EIA"), including Chapter 4: "Economic Options Discussion" (December 2, 2003 draft), which is included in the administrative record and is attached hereto as Attachment 1, and Chapter 8: "Benefits Analysis" (Oct. 9, 2003 draft), which was attached to Dr. Madronich's affidavit and is attached hereto as Attachment 2.

8. After reviewing these materials and based on my personal knowledge and experience regarding the AHEF model and the health risks of ODS, it is my understanding and opinion that (1) the calculations presented in Dr. Cox's affidavit do not accurately convey the incremental risk to a member of the U.S. population of death or illness as a result of the 2005 methyl bromide critical use exemption; (2) based on the analysis prepared for the EIA and the figures dervied by Dr. Madronich, a light-skinned member of the U.S. population alive in 2005 is likely to have approximately a 1 in 25,833,333 risk of premature death due to skin cancer and is likely to have approximately a 1 in 129,166 risk of contracting non-fatal skin cancer during his or her lifetime as a result of methyl bromide emissions attributable to the 2005 critical use exemption.[1]

9. These numbers can be calculated as follows. Out of the 10 premature deaths and 2000 non-fatal skin cancers estimated[2] to result from methyl bromide emissions attributable to the 2005 critical use exemption, I estimate that approximately 90% (or 9 premature deaths and 1800 non-fatal skin cancers) will occur in people alive in 2005, for the reasons given in paragraph 17, below. The AHEF projections of fatal and non-fatal skin cancers are calculated for the light-skinned portion of the U.S. population.[3] For 2005, the AHEF uses a figure of 232.5 million people to represent this population. Thus, to obtain a lifetime risk figure for a light-skinned member of the U.S. population alive in 2005, I use the following equations:

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[1] Risk calculations for cataracts are not discussed in detail here.

[2] Derived by Dr. Madronich by linear interpolation of the figures in Exhibit 8.3.1 of the EIA (Attach. 2).

[3] "Human Health Benefits of Stratospheric Ozone Protection: Peer Reviewed Report" (April 24, 2006), at 4 (Attach. 3). In contrast, the AHEF calculates cataracts cases in the U.S. population as a whole (293 million people in 2005).

- 9 premature deaths/232.5 million people = 1 premature death in 25,833,333 people or approximately 1 premature death in 25.8 million people

- 1800 non-fatal skin cancers/232.5 million people = 1 non-fatal skin cancer in 129,166 people or approximately 1 non-fatal skin cancer in 129,000 people

- Among the 490,274 people assumed to make up the NRDC membership in 2005, if we use the conservative assumption that all NRDC members are light-skinned, we would estimate 0.019 (or ~ 0.02) premature deaths and 3.795 (or ~3.8) non-fatal skin cancers.

10. The Atmospheric and Health Effects Framework (AHEF) model was developed by ICF in the mid-1980s. It estimates the skin cancers and other health effects resulting from ozone depletion. While the AHEF is capable of estimating world-wide impacts of various emission scenarios, its estimates are typically restricted to the U.S. population. Atmospheric lifetimes, chlorine/bromine composition, and other parameters related to an individual chemical's ozone depletion potential are used to estimate the impact of ODS on ozone concentrations, by month and by latitudinal band. Then, based on projected emissions of ozone depleting substances and the associated stratospheric ozone concentrations, the amount of ultraviolet (UV) radiation reaching the Earth's surface is estimated by latitude, month, year, and time of day using the Tropospheric Ultraviolet-Visible (TUV) radiation model.[4] When combined, the TUV model and the AHEF constitute a comprehensive exposure assessment.

11. Paragraph 6 of the Cox affidavit states: "the AHEF model is not a substitute for a human health risk assessment model." However, the AHEF contains all of the components necessary for a human health risk assessment, including hazard identification,[5] exposure

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[4] See Attach. 3 at 18.

[5] Hazard identification is the determination of whether a particular chemical is or is not causally linked to particular health effects.

assessment,[6] dose-response modeling,[7] characterization of risk,[8] and uncertainty.[9] In addition, peer reviewers have found that the AHEF's methodology represents a sound, state-of-the-art approach to assessing ozone-related health effects.[10] EPA's Office of Air and Radiation has used the model to assess the impacts of numerous regulatory programs under Title VI of the Clean Air Act.

12. The premature deaths and illnesses stated in Exhibit 8.3.1 of the EIA (attached to the Madronich affidavit and hereto as Attachment 2) are lifetime estimates and already take into account remaining years of exposure to the sun. Thus, in calculating risk to an individual using these figures, it is not necessary or appropriate to include a factor representing a person's lifetime or remaining years of exposure to the sun, as done in paragraph 12 of the Cox affidavit.

13. The AHEF, like any complex model, uses inputs and computational procedures that introduce uncertainty to the results. These include both quantified and un-quantified sources of uncertainty.[11] Paragraph 9 of the Cox affidavit states that the AHEF "does not make needed adjustments for confounders," including differences in sun exposure behavior. The AHEF assumes that sun exposure behavior is constant over time. Such sources of un-quantified uncertainty are inherent in most epidemiological health models of this kind. Most of these sources cannot be quantified because any assumptions or estimates would be speculative. Addressing variations in sun exposure behavior is well accepted to be beyond the ability of the current state of atmospheric and epidemiological science.

14. For the purposes of the EIA, the AHEF was used to calculate health effects for the full projected duration of EPA's critical-use exemption program, at declining levels up until

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[6] An exposure assessment is the determination of the extent of human exposure.

[7] Dose-response assessment is the determination of the relation between the magnitude of exposure and the probability of occurrence of the health effects in question.

[8] Risk characterization is defined as the description of the nature and often the magnitude of human risk, including attendant uncertainty.

[9] Uncertainty represents lack of knowledge about factors such as adverse effects or contaminant levels that may be reduced with additional study.

[10] Attach. 3 at (i).

[11] Attach. 3 at 33-43.

2018.[12] These estimates are for the U.S. population only. Dr. Madronich estimated the effects associated with the 2005 exemption alone, assuming a linear relationship between the methyl bromide emissions and the health effects shown in Ex. 8.3.1 of the EIA (Attach. 2). Dr. Cox stated that Dr. Madronich's "linearity assumption . . . is not valid for this purpose." Cox Aff. ¶ 10. I believe an assumption of linearity is a reasonable and appropriate approximation for several reasons. First, although some experts believe that the relationship between emissions of ODS and ozone depletion may be non-linear over the full range of ozone layer depletion, the relationship between current observations of ODS and ozone depletion can be characterized as linear. Moreover, because the 16.8 million pounds of additional emissions used in Dr. Madronich's calculations of incremental health effects is relatively small compared to total emissions of ODS used in the AHEF, the relationship between emissions and resulting health effects can be modeled as linear for methyl bromide scenarios. Also, in past analyses, incremental health effects estimated linearly have been compared to those estimated through actual runs of the AHEF, and for small changes in total emissions of ODS, the difference between health effects estimated by both methods has been shown to be negligible.

15. NRDC's rehearing petition, at 9, aggregates the projections of 10 premature deaths, 2000 non-fatal skin cancer cases, and 700 cataract cases. However, mortalities and morbidities are typically analyzed separately and valued differently in risk and economic analyses.

16. Methyl bromide does not remain in the stratosphere as long as certain other ODS, such as CFCs.[13] A single "pulse" of methyl bromide emissions contributes to ozone depletion over a relatively short time period compared to a single "pulse" of CFC emissions. However, in neither case is the ozone depletion a short-lived phenomenon. The ozone layer recovers slowly from changes in ozone levels, including changes caused by methyl bromide. Until recovery occurs, humans are exposed to higher UV radiation doses resulting from lower ozone levels.

17. Therefore, a portion of the health effects associated with the 2005 critical use exemption will occur in individuals not alive in 2005. However, of the estimated 10 mortalities and 2,000 non-fatal skin cancer incidences associated with incremental emissions of methyl bromide in 2005, I estimate that 90 percent[14] (about 9 mortalities and 1,800 incidences) will be associated with the 232.5 light-skinned individuals who were alive in 2005. These deaths and incidences are front-loaded on these individuals for three principal reasons. First, many individuals alive in 2005 will experience higher cumulative exposure to increased UV radiation than individuals born in later years. For example, an individual

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[12] Attach. 1, Exhibit 4.1.1; Attach. 2, Section 8.2.

[13] WMO (2002), Scientific Assessment of Ozone Depletion: 2002. World Meteorological Organization Global Ozone Research and Monitoring Project – Report No. 47 (Attach. 4), Table 1-3 (*compare* 0.7 year lifetime for methyl bromide *with* 100 year lifetime for CFC-12).

[14] The distribution of cataract cases may be somewhat different.

who is born in 2005 will be exposed to incremental UV radiation from 2005 until the ozone layer recovers to pre-depletion levels, whereas an individual born in 2006 will be exposed to one less year of incremental UV radiation, and so forth. Second, the U.S. population is relatively stable over time. Most of the people alive in 2006, for example, were also alive in 2005, and are already included in the 90% estimate. Third, the ozone layer is projected to recover around the middle to later part of this century; therefore, exposure to UV radiation will naturally decrease over time. As a result, individuals born in later years will be exposed to relatively lower cumulative UV radiation (compared to individuals born by 2005), despite the impacts on the ozone layer of incremental emissions of methyl bromide in 2005.

18. The AHEF model calculations typically extend to the year 2150 to be certain that all future deaths are captured in any given ODS emission scenario.[15] That time frame is not specific to methyl bromide or to a particular emissions year or years, but rather was developed for use with a variety of ODS and emissions years. The time frame is based on multiple considerations, including: exposure to UV radiation across future generations; the fact that recovery of the stratospheric ozone layer is projected for the middle to later part of this century (approximately 2050-2065 depending on global compliance with the Montreal Protocol); and the long lag between time of exposure and onset of skin cancer.

19. In order to calculate the risk to an individual of dying as a result of the 2005 critical use exemption, Dr. Cox divided the 10 deaths by the U.S. population multiplied by 145. Cox. Aff. ¶ 11. This does not yield an accurate statement of risk to an individual. The AHEF's calculations incorporate certain assumptions, including the size of the exposed population and the length of the exposure period. For example, the premature fatalities and illnesses presented in Attachment 2 are the AHEF's estimates for people who will be exposed to the incremental UV radiation in the U.S. (for skin cancer, this is limited to light-skinned individuals). Multiplying by 145 would overcount the exposed population and understate individual risk. In addition, the AHEF factors in the remaining years of UV radiation exposure in calculating lifetime risk for the exposed population. It is not necessary or appropriate to account separately for the remaining years of UV exposure.

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[15] Attach. 3, at 27 ("By approximately 2150, it is predicted that there will be no living population that experienced incremental exposure associated with depleted ozone levels, and hence, no additional health effects incidence or mortality above those expected to occur under 'normal' conditions.")

20. In paragraph 11 of his affidavit, Dr. Cox derives a "per person per year" excess fatality risk. This is not an accurate statement of annualized risk. Expressing the risk in annualized terms is not practical because the incremental risk to a population of developing skin cancer is not constant from year to year and instead increases over time as cumulative UV radiation exposure also increases. In other words, for each year that a population is exposed to incremental UV radiation resulting from methyl bromide emissions in 2005, that population's marginal risk of developing skin cancer increases. As a result, it is more appropriate to express the risk as a population's cumulative or lifetime risk.

I declare under penalty of perjury under the laws of the United States of America that the foregoing is true and correct to the best of my knowledge.

Executed this 16th day of June, 2006:

Reva Rubenstein

Reva Rubenstein
Senior Toxicologist
ICF International
1725 Eye Street, N.W.
Suite 1000
Washington, D.C. 20006

ATTACHMENT 1 TO EXHIBIT 1

Economic Impact Analysis for Methyl Bromide Allocation in the United States

Revised Draft Report

December 2, 2003

Prepared for

Hodayah Finman
Global Programs Division
U.S. Environmental Protection Agency
1200 Pennsylvania Avenue, NW (6205J)
Washington, DC 20460

Prepared by

ICF Consulting
1725 Eye Street NW, Suite 1000
Washington, DC 20006

4. Economic Options Discussion

This section provides an overview of the three broad regulatory options that are analyzed in this Economic Impact Analysis, and highlights salient features of the options that are important from the perspective of the economic analysis. Note that these are not the only options that EPA considered during the course of the proposed rulemaking process, but rather constitute a representative set of options that EPA initially identified as the basis of the economic analysis.

Section 4.1 below describes the criteria for a critical use exemption as described in the Montreal Protocol, the U.S. nomination for quantities in 2005 and 2006, and options for implementing the exemption. For purposes of this analysis, the section also describes the assumptions made about consumption in both the years of the nomination and beyond. Section 4.2 defines important terms used in this section. Sections 4.3, 4.4, and 4.5 describe three broad options that EPA could use to implement the CUE quantities that will be allocated to the U.S. by the Parties to the Montreal Protocol, including the relationship between the option and existing systems for allocating methyl bromide to end users. These sections also provide additional detail on the options analyzed in this document that is needed to develop quantitative cost estimates. Included in these sections is information on existing systems that provide a model for the system, the entities holding allowances or permits, the operation of the trading system, the method of allocation to end users, and recordkeeping and reporting requirements.

4.1 Overview of Phaseout Assumptions and Allocation Options

Critical use exemption language under Decision XI/6 of the Parties to the Montreal Protocol indicates that a use of methyl bromide will be considered critical only if, "(ii) There are no technically and economically feasible alternatives or substitutes available to the user that are acceptable from the standpoint of environment and health and are suitable to the crops and circumstances of the nomination;...(b)(i) All technically and economically feasible steps have been taken to minimize the critical use and any associated emissions of methyl bromide; (ii) Methyl bromide is not available in sufficient quantity and quality from existing stocks of banked or recycled methyl bromide, also bearing in the mind the developing countries' need for methyl bromide; (iii) [and] it is demonstrated that an appropriate effort is being made to evaluate, commercialize and secure national regulatory approval of alternatives and substitutes...Parties must demonstrate that research programs are in place to develop and deploy alternatives and substitutes..." In addition, the nominating party must determine that the lack of methyl bromide availability for that use would result in a significant market disruption (UNEP 2000 -- Montreal Protocol on Substances that Deplete the Ozone Layer Decision IX/6).

Based on the criteria indicated by the Montreal Protocol, the United States requested 39 percent of 1991 U.S. baseline consumption for 2005 and 37 percent for 2006 for CUE purposes from the Parties to the Montreal Protocol. This EIA assumes that methyl bromide quantities consumed in the United States in 2005 and 2006 will be equal to the quantities requested in the U.S. nomination. Beyond 2006,

the EIA assumes that consumption of methyl bromide for critical use will continue at 37 percent of baseline through 2010. Use then drops by 5 percent annually for 7 years through 2017, with a final drop of 2 percent and subsequent consumption of 0 percent in 2018 and beyond. Exhibit 4.1.1 summarizes this phaseout schedule.

Exhibit 4.1.1. Assumed Phaseout Schedule for U.S. Methyl Bromide Critical Use Exemption

Year	Percent consumption of 1991 baseline
2005	39
2006	37
2007	37
2008	37
2009	37
2010	37
2011	32
2012	27
2013	22
2014	17
2015	12
2016	7
2017	2
2018	0

These assumptions are used for strictly analytical purposes and do not represent an attempt to predict the actual course of a methyl bromide phaseout. The maximum amount of methyl bromide allowed for CUE each year will be determined by the Parties to the Montreal Protocol, and actual phaseout is likely to differ from these assumptions.

This lengthened period of methyl bromide availability and the need to distribute available amounts to end users necessitates analysis of various options for methyl bromide allocation to determine an economically fair system that will not unduly burden end users. The system must strike a balance between economic efficiency (i.e., methyl bromide is distributed in the most cost-efficient manner possible so that no individual could be made better off without causing another individual to be worse off) (Goodstein 1999), and equity (i.e., the avoidance of harming certain end users, such as small entities, even if efficiency must be somewhat compromised).

Implementing the longer period for the phaseout, and the increased availability of methyl bromide to end users eligible for a critical use exemption, requires developing and implementing a system for allocating or distributing the methyl bromide. The EPA considered a number of possible alternative allocation systems, and identified three systems for additional economic analysis. These allocation systems (also described as "models" or "options" in this EIA) are as follows:

Option 1: Producer/Importer Cap and Trade Allowance with Market Distribution of Methyl Bromide
Option 2: Producer/Importer Cap and Trade Allowance with End User Permit Trading
Option 3: Producer/Importer Cap and Trade Allowance with End User Permit Auction and Trading [initially considered as an option but not analyzed in the remainder of this document].

Under all three options, methyl bromide would be capped, and allowances would be allocated to producers and importers based on their historic levels of production or import. Allocation would be determined by historic production and trading of allowances between producers and importers would be allowed. Under Options 2 and 3 there would be additional regulations that would distribute rights of critical use methyl bromide to approved users. Under Option 2, EPA would provide permits to end users using a reconstructed baseline of historic methyl bromide consumption. These permits could then be traded, either within sectors or across sectors (depending on how the option is implemented). Option 3 involves the distribution of permits to end users at an auction where approved critical users may bid for the rights to buy methyl bromide. This option has four sub-options: auction to sectors ("sector auctions") or a universal auction, and post-auction trading within or among sectors.

EPA is proposing Option1 as the preferred regulatory option, based on a comparison of the total costs of the three options to EPA and to industry. The following sections outline the options in more depth, and Chapters 6, 7, and 9 provide a detailed comparison of administrative and total costs of the options.

4.2 Definition of Terms

Several terms are used frequently in descriptions of the three main methyl bromide allocation options:

- End users are individual business entities within sectors that use methyl bromide. For example, one hypothetical 25-acre tomato farm in Florida represents one end user.

- Methyl bromide allowances and permits refer to the unit of distribution of methyl bromide for critical use exemption (CUE allowances are held by importers and producers, and CUE permits are held by end users). An allowance or permit gives an allowance or permit holder the right to purchase, trade, or receive through allocation one kilogram of methyl bromide. Some of the assumptions made for the purpose of analysis were:

    - Allowances/permits expire after one year. For example, if an end user possesses permits to use 250 kilograms of methyl bromide in 2005 but only uses 200 kilograms by December 31, 2005, the end user cannot carry the 50 kilograms remaining in the 2005 permits over to 2006.

ATTACHMENT 2 TO EXHIBIT 1

8. Benefits Analysis

This section presents benefits of the CUE allocation phaseout as compared to the original methyl bromide Phaseout RIA.

8.1 Basic Methodology Framework

The benefits of the regulation were calculated using the Atmospheric and Health Effects Framework (AHEF) model. The AHEF, which consists of a series of FORTRAN modules, produces estimates of the likely increases in skin cancer mortality and incidence and cataract incidence resulting from past and future ODS emission scenarios, and compares the changes in health effects incidence and/or mortality to those that would otherwise exist under a the 1979-1980 baseline scenario of no-further-ozone-depletion scenario (i.e., no depletion beyond that which existed in the 1979-1980 time period). The AHEF compares these differences across policy and control scenarios to estimate the additional benefits of each scenario based on the degree of ODS control stringency (ICF 2000a).

8.2 Input Data

The primary input for the AHEF is methyl bromide emissions data (see Appendix B). The emissions model is based upon the assumption that 50 percent of methyl bromide consumed for treatment of agricultural soils is ultimately emitted to the atmosphere.[1] This analysis of benefits is for pre-plant and post-harvest methyl bromide uses for both the original and CUE phaseout scenarios. Consumption data are derived from figures reported to National Ozone Units and the Montreal Protocol Secretariat. In the baseline scenario, growth in future emissions was constrained to account for actual methyl bromide consumption, as well as the freeze required by the Montreal Protocol for both developed and developing countries. For purposes of this analysis, it is assumed that a CUE level equal to 39% of the 1991 baseline for 2005, 37% of the baseline for 2006, and further reduction as outlined in Chapter 4 (see Section 4.1) will be implemented.[2]

Other important inputs to the benefits assessment are unit values used to monetize reductions in health effects due to the phaseout. In addition to value of a saved life (VSL) estimates, other values include costs for hospitalization or treatment of skin cancer (e.g., surgery or therapy for melanoma and non-melanoma lesions), and medical treatment for cataracts. To monetize the avoided health effects the following input data were used:

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[1] Emissions rates have been reported to range from 32 to 87 percent as presented by UNEP (1998).

[2] As paraphrased by UNEP (1998), "For non-Article 5(1) Parties operating under the Protocol (developed countries) . . . A freeze on MB production and consumption [is] based on 1991 levels . . . For Parties operating under Article 5(1) of the Protocol (developing countries) a freeze on MB production and consumption [is] based on 1995-98 levels from 1 January 2002 . . ."

- Value of a life saved is based on EPA (1999b). The value used for this analysis is $5.8 million.
- Value of avoided non-fatal melanoma skin cancer is based on ICF (1999a). The value used for this analysis is $12,500.
- Value of avoided non-melanoma skin cancer is based on ICF (1999b). The value used for this analysis is $1,250.
- Value of avoided cataract is based on ICF (1999c). The value used for this analysis is $2,500.

These values were used in the Phaseout RIA and are used here to maintain consistency between the monetized estimates of benefits presented in that analysis and the estimates presented here.

8.3 Results of the Benefits Analysis for the CUE Scenario

Exhibit 8.3.1 presents preliminary estimates of the increases in human health effects expected from the CUE scenario, compared to the methyl bromide phaseout, as estimated by the AHEF. As stated previously, benefits were monetized by multiplying the reduced morbidity and mortality estimates by their respective unit value. Note that the monetized values are based on the central incremental case value in Exhibit 8.3.1. Monetized values are not provided for the uncertainty range of incremental cases. As shown, the benefits of the proposed CUE allocations in the United States are estimated to have decreased by $783.8 million (undiscounted relative to the original 2005 phaseout). Benefits were also assessed at discount rates of 7, 3, and 1 percent. As shown by Exhibit 8.3.2, discounted benefits decreased $60 million and $209 million at 7 and 3 percent, respectively. On an annualized basis, the decrease in benefits range from $4.4 million to $8.0 million at 7 and 3 percent discount rates, respectively.

Exhibit 8.3.1. Decreased Human Health Benefits from CUE allocations compared to the Methyl Bromide Phaseout in the United States in 2005 (1999-2150)

	Cutaneous Malignant Melanoma		Non-melanoma Skin Cancer			Cataract
	Incidence	*Mortality*	*Basal Cell Incidence*	*Squamous Cell Incidence*	*Mortality*	*Incidence*
Incremental Cases [a] (Uncertainty Range)	660 (264 – 1,056)	83 (33 – 133)	15,809 (6,324 - 25,294)	7,752 (3,101 – 12,403)	42 (17 – 67)	8,105 (3,242 – 12,968)
Unit Value (1999$)	$12,500	$5.8 million	$1,250	$1,250	$5.8 million	$2,500
Monetized Benefit (undiscounted)	-$8.3 million	-$481.5 million	-$19.8 million	-$9.7 million	-$244.2 million	-$20.3 million

[a] Values in parentheses represent an uncertainty range of approximately 60% (0.6), based on health effects uncertainties for the following factors: 0.50 for action spectrum values, 0.05 for the UV radiative transfer modeling step, 0.30 for the biological amplification factor (BAF), and 0.10 for the choice of dose metric used in the AHEF. The value of 0.6 is the square root of the sum of the squared uncertainty terms.

Exhibit 8.3.2. Decreased Human Health Benefits from CUE allocations compared to the Methyl Bromide Phaseout in the United States in 2005 (1999-2150)

Scenario	Benefits (1997$)
Undiscounted	
NPV	($783.8 million)
Annualized	$15.1 million)
Discount Rate: 7 percent	
NPV	($60.4 million)
Annualized	($4.4 million)
Discount Rate: 3 percent	
NPV	($208.6 million)
Annualized	($8.0 million)
Discount Rate: 1 percent	
NPV	($479.8 million)
Annualized	($11.9 million)

8.4 Unquantified Benefits

Changes in the incidence and mortality for the numbers of skin cancers and incidence for cataracts are not the only indicators of the damage to human health and the environment that result from increases in UV radiation due to ozone depletion. Increased UV radiation can cause a wide variety of additional human health problems, including actinic keratosis (a skin disease) and immune system disorders. Increased UV levels also lead to higher concentrations of tropospheric ozone (smog) that can adversely impact human respiratory and pulmonary systems. Furthermore, the impact of ozone depletion is not limited to humans; plants and animals can also suffer serious consequences from UV radiation. Overall, in addition to fewer skin cancers and

cataracts, the following endpoints are expected to change due to the phaseout modifications.

Increase in:

- mortality from acute exposure;[3]
- immune system suppression;
- aquatic and terrestrial ecosystem disruption, including reproductive/developmental effects, immune system suppression;
- impacts on agriculture such as decreased plant productivity, slowed metabolism, hastened plant disease;
- impacts on materials (i.e., accelerated breakdown of plastics and other synthetics); and
- lost productivity and evacuations.

Therefore, negative unquantified impacts will follow in each of these areas as a result of the CUE.

[3] Incremental human health effects due to acute exposure expected from the CUE scenario were examined for this analysis. Between 2005 and 2018, 5.4 fatalities are expected due to acute methyl bromide exposure and 106.3 cases of acute methyl bromide exposure are expected in California. Benefits (or lost benefits) associated with acute exposure to methyl bromide are not examined further in this document, as analysis of these exposures fall under the purview of the Office of Pesticide Programs (OPP) and any re-registration requirements under FIFRA. (CADPR 2000, EPA 1999b, ICF 1999).

ATTACHMENT 3 TO EXHIBIT 1

HUMAN HEALTH BENEFITS OF STRATOSPHERIC OZONE PROTECTION

Peer Reviewed Report

Prepared for:

Global Programs Division
Office of Air and Radiation
U.S. Environmental Protection Agency
Washington, DC 20460

Prepared by:

ICF Consulting
1725 Eye Street NW, Suite 1000
Washington, DC 20006

April 24, 2006

Preface and Peer Review Summary

This report was prepared by the U.S. Environmental Protection Agency (EPA) with the support of its contractor, ICF Consulting, Inc. (ICF). This report describes the analytical and empirical methodologies used by the Atmospheric Health Effects Framework (AHEF), a model used to predict changes in human health effects that result from changes in the use and release of ozone-depleting substances (ODS).

The authors of this report consulted with experts from government, industry, and academia in the fields of atmospheric chemistry and dynamics, health effects of ultraviolet radiation atmospheric modeling, and health effects modeling (see Acknowledgments section). In August and September of 2003, the draft final document was peer reviewed for its technical content by Dr. Edward De Fabo of The George Washington University in Washington, DC, and by Mr. Archie McCulloch of Marbury Technical Consulting in Cheshire, United Kingdom, and visiting research fellow at the School of Chemistry, University of Bristol. The peer reviewers were asked to draw upon their expertise in ultraviolet (UV) radiation biological effects assessment and atmospheric science, respectively, to comment on whether the methods, tools, and approach used in the study reflect sound scientific practice and adequately address the questions at hand.

Written comments were received from peer reviewers. In these comments, the reviewers stated that the methodology used in this model represents a sound, state-of-the-art approach to assessing ozone-related health effects. A number of comments identified areas for clarification of specific technical items, all of which have been considered by the authors. The reviewers stated that the report provides solid analysis and discussion of results, given the scope of the work and the uncertainties that currently exist in the areas of ozone depletion and UV radiation health impacts estimation.

Several areas were highlighted during peer review of this report. Dr. De Fabo highlighted the fact that one of the greatest sources of uncertainty in estimating UV radiation-induced health impacts is the lack of adequate experimental data from which a biological action spectrum for cutaneous malignant melanoma (CMM) can be developed. Due to this lack of information, the AHEF predicts cases of malignant melanoma based on the SCUP-h action spectrum for squamous cell carcinoma (SCC). Dr. De Fabo agreed that the SCUP-h spectrum is the most appropriate action spectrum available to model CMM at this time. He noted that the action spectrum for CMM still remains to be determined, and that use of the SCUP-h in modeling CMM should be reconsidered if future research reveals that the shape of the action spectrum for CMM is not congruent with the SCUP-h action spectrum. EPA acknowledges that further scientific research in these and other areas could complement and significantly enhance the information presented in this report.

Dr. De Fabo also agreed that the removal of cataract incidence from the AHEF's health effects modeling reflects a sound decision, in light of recent analyses that suggest a weak correlation between UV exposure and cataract incidence in the United States. Dr. De Fabo also affirmed that the paper's discussion on immunosuppression accurately reflects the current state of the science.

Mr. McCulloch suggested several revisions to the original text to remove ambiguity, and provided additional information on the methodologies and assumptions used by WMO in their 1999 and 2003 reports, to allow for a more accurate and thorough comparison of the projected ozone concentrations predicted by WMO and by the AHEF. Mr. McCulloch also commented on the need to clearly justify the selection of 55 as the bromine efficiency factor—or *alpha factor*—for use in the AHEF instead of 45, which is the value recommended by WMO (WMO 2003). The selection of an alpha factor of 55 is based on the results of state-of-the-art atmospheric models, and is also the value used in a recent report prepared for the U.S. Department of Defense (Independent Review Panel 2002, Wuebbles 2003). In general, Mr. McCulloch affirmed that the atmospheric science module of the paper provides clear descriptions of the methodology and model parameters used, which allow the reader to reach conclusions about the way the methods have been applied and how they relate to "mainstream" atmospheric science (e.g., WMO Ozone Assessments).

All comments of the reviewers were considered, and the document was modified appropriately.

EPA wishes to acknowledge everyone involved in this report and thank reviewers for their extensive time, effort, and expert guidance. The involvement of peer reviewers and other scientific contacts greatly enhanced the technical soundness of this report. EPA accepts responsibility for all information presented and any errors contained in this document.

Global Programs Division (6205J)
Office of Atmospheric Programs
U.S. Environmental Protection Agency
Washington, DC 20460

Executive Summary

Stratospheric ozone protects the biosphere from potentially damaging doses of ultraviolet (UV) radiation. Depletion of stratospheric ozone, caused by the release of man-made ozone-depleting substances (ODS)—such as chlorofluorocarbons (CFCs), halons, methyl bromide, and hydrochlorofluorocarbons (HCFCs)—could lead to significant increases in UV radiation reaching the Earth's surface, which could in turn lead to adverse human and animal health effects, as well as ecosystem impacts.

The Montreal Protocol on Substances That Deplete the Ozone Layer (Montreal Protocol) is a landmark international agreement designed to protect the stratospheric ozone layer. The treaty was originally signed in 1987 and substantially amended in 1990, 1992, and 1997. The Montreal Protocol stipulates phaseout schedules for the production and consumption of compounds that deplete ozone in the stratosphere.

The United States Environmental Protection Agency (EPA) uses its Atmospheric and Health Effects Framework (AHEF) to evaluate certain human health impacts associated with reduced emissions of ODS under the Montreal Protocol and associated amendments. Specifically, the AHEF estimates the probable increases in skin cancer mortality and incidence in the United States that result from ODS emission scenarios relative to the baseline. The baseline is defined as the health effects that would have occurred if ozone concentrations that existed in 1979-1980 had been maintained through the time period modeled. The 1979-1980 concentrations of ozone are used as the baseline because at this date minimal ozone depletion had occurred. Differences in health effects can be compared across broad policy scenarios to estimate potential benefits of alternative ODS controls.

The accuracy of the AHEF's predictions depends upon continual updating of its inputs and methodologies to reflect on-going scientific advances since the AHEF's creation in the mid 1980s. Significant new research results that have been incorporated into the revised version of the AHEF include the following:

- Recalibration and refinement of stratospheric ozone concentration measurements;
- Updated ODS emission data;
- Improved forecasts of the impact of emissions of ODS on stratospheric ozone concentrations;
- New predictions of the impact of changing ozone concentrations on UV radiation intensity at the Earth's surface;
- Updated information on the biological effects of UV radiation of different wavelengths (action spectra), and how age and year of birth affect the induction of skin cancers and other human health effects;
- Improved estimation of projected skin cancer mortality rates, based on more recent and reliable epidemiological data;
- Revised health effects modeled by the AHEF, to more accurately predict only those health effects for which an agreed upon dose-response relationship is available; and
- Updated population data.

While each of these model updates has affected the AHEF output to varying degrees—either slightly or significantly increasing or decreasing total projected health effects—each has contributed to more accurate modeling results. In addition to these model updates, several other changes have been made to enhance model resolution and flexibility. Appendix A details all of the model updates and changes that

have been made to the AHEF since its inception, and provides explanations and justifications for why each one was performed, and its implication on modeling results.

Despite these model updates, no model or set of results quantifying health effects impacts can be considered final, given that research on the atmospherics of ozone depletion and health effects of UV exposure is ongoing. Many important issues must continue to be investigated and, as significant new findings are incorporated into the AHEF, the accuracy of predictions and the implications for protecting stratospheric ozone will be enhanced. For example:

- Additional research on the effects of UV radiation on darker-skinned populations would enable the AHEF to predict the incremental health effects for all populations;
- Further disaggregation of cataract incidence data by state, and the generation of a population-weighted, geographically distributed dose-response relationship for cataract incidence and UV exposure would allow for appropriate modeling of cataract incidence changes in the AHEF;
- Additional scientific research into the impacts of UV exposure on immune suppression would allow for the inclusion of this health endpoint into the model;
- Improved ground-level UV monitoring would allow the AHEF to incorporate the effects of cloud-cover and pollution on UV radiation at ground-level; and
- Additional research on the effects of UV radiation on non-human endpoints (e.g., aquatic systems, agriculture) would allow the AHEF to predict the broader impacts associated with ODS emission scenarios.

The AHEF is a living model, designed with the ability to accept changes in any model input or assumption based on new scientific findings, and/or to incorporate any new information as it becomes available. As the science on stratospheric ozone depletion and its associated impacts continues to evolve, so too will the AHEF.

2. Model Overview

The AHEF has five main computational steps that lead to estimated changes in incidence and mortality for various UV-related health effects for a given ODS emission scenario. These computational steps are as follows:

1. Projecting baseline incidence and mortality of health effects;
2. Projecting impacts of future ODS emissions on stratospheric ozone;
3. Modeling the resulting changes in ground-level UV radiation;
4. Deriving dose-response relationships for health effect incidence and mortality; and
5. Projecting future health effects incidence and mortality.

These steps are described in detail below.

Step 1. Projections of baseline incidence and mortality are computed based on historical rates assuming column ozone concentrations remained constant at 1979-1980 levels.

The AHEF defines the "baseline" incidence and/or mortality for skin cancer as what would be expected to occur in the future if the concentration of stratospheric ozone remained fixed at 1979-1980 levels. This baseline provides a standard against which to evaluate increases in mortality and/or incidence for these health effects from future ODS emissions and ozone depletion and, under most scenarios, future recovery of the ozone layer to 1979-1980 levels.[3] The following data and calculations form the baseline estimate of current and future incidence and mortality:

- Historical data on skin cancer incidence and mortality were used to derive rates (per 100,000 people) for UV-related health effects in the U.S. population. Rates are based on age, sex, and in some cases, birth year.
- Historical U.S. population estimates (up to 1990) were obtained from the U.S. Census Bureau, and national population estimates for 1991-2050 were derived by age and sex groupings from U.S. Census Bureau projections. Population projections by state, age, sex, and race—based on national population projections for year 2050 and state population projections through 2025[4]—were grouped by latitude-based regions. (Population was assumed to be constant from 2050 to 2100.)
- The number of individuals in each age and sex group was multiplied by the appropriate historical incidence and/or mortality rate to produce an estimated baseline number of future skin cancer cases and deaths per year.

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[3] The AHEF assumes that changes in behavior that might confound the establishment of an accurate baseline do not occur. For example, a population that becomes less sun-seeking could theoretically have a lower baseline risk than the earlier cohort that provided the baseline data, and an increase in cloudiness or rainfall could reduce the number of hours spent outdoors, thereby reducing baseline exposures.

[4] State population projections through 2025 were computed as the sum of the totals for the states in each region, and then regional populations (by age, sex, and race) were projected to 2050 based on the national Census projections for 2050—under the assumption that the 2025 regional age, sex, and race proportions of the total U.S. population will remain unchanged through 2050. In this way, population estimates for 1990-2025 were based on state population projections, while population estimates for 2025-2050 were based on national population projections. See Section 3.5 for more details.

Because skin cancer and solar UV irradiance vary by latitude, the baseline U.S. health effects data were stratified into three latitude regions (i.e., 20 to 30°N, 30 to 40°N, and 40 to 50°N), to correspond with satellite data on ozone concentrations. Because skin cancer incidence and mortality among darker-skinned populations are not well understood in terms of rates of responsiveness to increased UV exposures, these health effects are only modeled for light-skinned populations. Once the required information becomes available, data for darker-skinned U.S. populations may be included.

Step 2. Impacts of future emissions of ODS on stratospheric ozone concentrations are modeled.

Since 1978, satellites have provided measurements of stratospheric ozone concentrations using a latitudinal grid. Data from the first of these satellites, the Nimbus-7, indicate that during the satellite's lifespan from 1978 to 1993, ozone concentrations declined in a manner that corresponds to an increase in the concentration of stratospheric chlorine and bromine released from the dissociation of ODS molecules. Using this relationship, the AHEF can use estimated ODS emissions to predict future decreases in stratospheric ozone. First, the framework uses regression coefficients to quantify the relationship between past ODS emissions and past changes in ozone concentrations. These regression coefficients were derived as follows:

- Historical information on the concentrations of stratospheric ozone by latitude and month was obtained from satellite data.

- Estimates of emissions of ODS were obtained for past time periods that could affect ozone during the years for which satellite data were available. These ODS emissions estimates were then combined with information on each ODS species' degree of dissociation and rate of transport to the stratosphere. Using this information, total ODS emissions were converted to equivalent effective stratospheric chlorine (EESC) for each year and month for which ozone measurements were available from the Nimbus-7 satellite.

- Statistical linear regressions were performed using the 1978-1993 annual EESC estimates and stratospheric ozone concentrations, as measured by the Nimbus-7, to estimate the impact of ODS on ozone concentrations. These regressions were estimated by month and by latitudinal band.[5]

- Future changes in ozone associated with projected emissions for each ODS emission scenario were converted to EESC estimates which were then multiplied by the estimated regression coefficients to predict future ozone concentrations by month and latitude band.

Step 3. Changes in ground-level UV radiation are estimated.

Based on projections of stratospheric ozone concentration, UV radiation intensities at the Earth's surface were estimated by latitude, month, year, and time of day using the Tropospheric Ultraviolet-Visible radiation model (TUV, v3.9a, as described in Madronich 1993a, Madronich 1993b). The TUV model generates look-up tables[6] (see Section 5.1 for more detail) of weighted solar UV irradiance at sea level as a function of solar zenith angle and projected total column ozone based on the following assumptions: obstruction-free and cloud-free skies; standard profiles of air density, temperature, and tropospheric ozone (USSA 1976); typical continental aerosols (Elterman 1968); and 10 percent isotropic ground reflectivity.

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[5] A similar procedure has been used in WMO assessments, which also use the Nimbus-7 satellite data (WMO 1995, WMO 1999). See Appendix D: Comparison of AHEF and WMO Predicted Ozone Concentrations for more information on how AHEF and WMO column ozone estimates compare.

[6] The axes of these look-up tables are solar zenith angle and column ozone concentrations.

Once solar UV irradiance at the Earth's surface is calculated, estimates of UV exposure experienced by humans can be computed. Peak hour or daily dose on any day of the year, or cumulative doses for a set of months or for an entire year are examples of possible dose metrics. The AHEF estimates UV exposures for both the entire day of June 21st (i.e., peak day) and the cumulative dose for the entire year (calculated as the dose on the 15th day of each month multiplied by 30 days per month summed across months) for selected action spectra.[7]

Step 4. Dose-response relationships for skin cancer incidence and mortality are selected.

Determining the health effects caused by UV exposure first requires information on the relative weights to be placed on each discrete UV wavelength to reflect the degree to which each wavelength causes biologic damage. Such a weighting function is called an action spectrum—an experimentally derived function that describes the relative effectiveness of each UV wavelength in the induction of skin cancers. Action spectra are normally developed by scientists by exposing a test animal to different UV wavelengths and then verifying the effectiveness of each wavelength at inducing a specific health effect. For each health effect, an available action spectrum must be selected for use in the AHEF.

Once the action spectrum for each health effect is selected, it is then possible to explore the relationship between those health effects and the intensity of UV exposure. These dose-response relationships are typically derived by correlating measurements or estimates of UV exposure received for a specific action spectrum and given health effect at various locations, and the level of incidence or mortality for that health effect at those same locations.

For example, the incidence of SCC decreases with distance from the equator (i.e., increasing latitude). It is also the case that UV irradiance decreases with distance from the equator. A dose-response relationship can thus be derived statistically by correlating the incidence of SCC measured at various locations at a variety of latitudes with the UV radiation doses measured or estimated for those same locations, as shown in Figure 1.

Step 5. All inputs are combined to project future skin cancer incidence and mortality.

The final step in the modeling framework incorporates the inputs from Steps 1-4 to project future additional skin cancers generated under a particular emission scenario compared to 1979-1980 baseline ozone conditions. This includes two calculations by the AHEF. First, the AHEF projects future baseline skin cancer incidence and mortality. Then the AHEF calculates the future annual percentage change in UV radiation dose for a given action spectrum across the three latitudinal bands of the United States for the specific ODS emission scenario. Multiplying the percentage change in UV exposure in a future year by the appropriate dose-response relationship yields the percentage change in future skin cancer incidence/mortality attributable to the future change in ozone concentrations. These percentages are then multiplied by the baseline incidence and/or mortality for that health effect to compute the absolute number of additional future cases or deaths attributable to ozone depletion under various ODS emission scenarios relative to the 1979-1980 baseline ozone levels.[8]

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[7] It is important to note that this analysis does not include a comprehensive listing of all published action spectra that may be applicable to the prediction of skin care and cataracts in humans. For example, the derivation of new action spectra for UV-mediated health effects not considered in this report (e.g., immunosuppression) is an active field of research. The AHEF's modular structure, described in detail below, enables new action spectra or new information on other UV-mediated human health endpoints to be easily incorporated into the modeling framework.

[8] This method of multiplying the changes in UV exposure by the BAF and the underlying baseline incidence or mortality is the same as that used by other researchers to estimate changes in health effects based on changes in ozone concentrations (e.g., Madronich and de Gruijl 1994, Pitcher and Longstreth 1991).

7. Modeling Results

This section presents the projected changes in incidence and/or mortality for each of the health effects and policy scenarios examined.

7.1 Results Presented by Policy Scenario and by Health Effect

Table 7 presents the incremental number of skin cancer cases/deaths in excess of the baseline (i.e., those associated with changes in column ozone concentrations from levels observed in 1979-1980) that are projected to occur under each ODS control scenario. Decreasing incidences/mortalities that result as more stringent control scenarios are implemented illustrates the benefits of each further amendment and/or adjustment to the Montreal Protocol. Table 8 presents the *avoided* health effects realized in moving from one ODS policy scenario to the next (e.g., from the Montreal Protocol to the London Amendments). Figure 4 illustrates that as ODS controls are tightened, additional incidence and mortality estimates for each health effect relative to baseline move closer to zero on the y-axis (i.e., closer to the incidence and mortality that would be expected if 1979-1980 ozone concentrations had been maintained throughout the time period modeled).

Table 7. Summary of Incremental Skin Cancer Incidence/Mortality for ODS Policy Scenarios Relative to Baseline

Cohort Group/ Scenario	CMM Incidence (Cases)	CMM Mortality (Deaths)	BCC Incidence (Cases)	SCC Incidence (Cases)	NMSC Mortality (Deaths)
Montreal Protocol					
1890-1980	301,687	44,582	8,814,835	5,050,875	30,859
1985-2010	794,121	109,352	21,250,450	11,517,066	66,829
2015-2050	2,042,358	265,759	50,978,569	26,627,765	147,554
2055-2100	3,228,517	409,876	78,708,574	40,793,037	220,452
London Amendments					
1890-1980	101,523	13,774	2,785,732	1,514,657	7,960
1985-2010	113,885	13,854	2,688,789	1,375,322	6,926
2015-2050	80,379	9,527	1,830,867	924,516	4,602
2055-2100	31,569	3,831	734,634	377,381	1,946
Copenhagen Amendments					
1890-1980	76,048	10,118	2,047,391	1,096,153	5,593
1985-2010	66,922	7,815	1,495,278	743,682	3,634
2015-2050	18,026	2,023	379,285	186,009	906
2055-2100	0	0	0	0	0
Montreal Adjustments					
1890-1980	68,816	9,076	1,834,142	974,827	4,923
1985-2010	54,940	6,356	1,210,046	599,467	2,925
2015-2050	10,308	1,155	216,245	105,993	517
2055-2100	0	0	0	0	0

Note: The numbers presented above indicate the number of cases in excess of the baseline (1979-1980) for each scenario.

Table 8. Incremental Number of Avoided Skin Cancer Incidence/Mortality Under ODS Policy Scenarios with Increasingly Stringent Controls

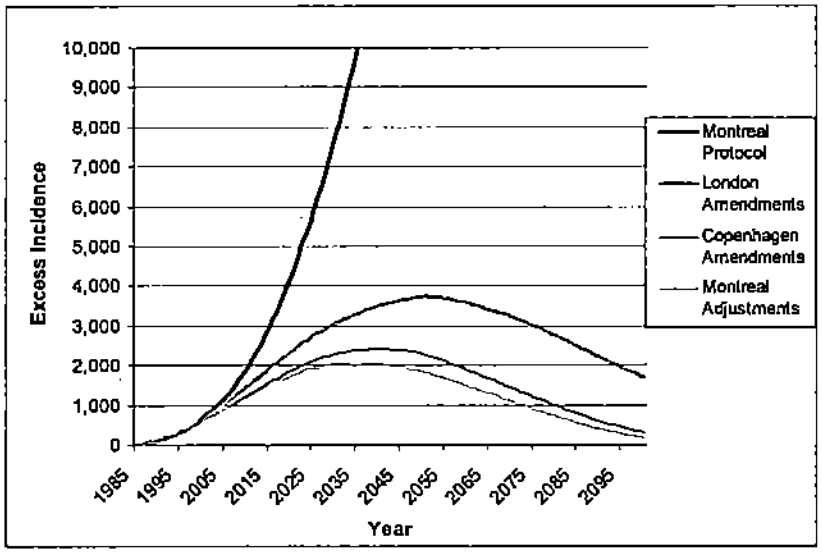
Cohort Group/Scenario	CMM Incidence (Cases)	CMM Mortality (Deaths)	BCC Incidence (Cases)	SCC Incidence (Cases)	NMSC Mortality (Deaths)
Incremental Number of Avoided Cases/Deaths: From Montreal Protocol to London Amendments					
1890-1980	200,164	30,809	6,029,103	3,536,217	22,898
1985-2010	680,236	95,498	18,561,661	10,141,745	59,903
2015-2050	1,961,979	256,232	49,147,703	25,703,249	142,952
2055-2100	3,196,948	406,045	77,973,940	40,415,656	218,506
Total	**6,039,327**	**788,584**	**151,712,406**	**79,796,866**	**444,258**
Incremental Number of Avoided Cases/Deaths: From London Amendments to Copenhagen Amendments					
1890-1980	25,475	3,655	738,342	418,504	2,368
1985-2010	46,963	6,038	1,193,511	631,640	3,292
2015-2050	62,353	7,504	1,451,582	738,507	3,697
2055-2100	31,569	3,831	734,634	377,381	1,946
Total	**166,360**	**21,028**	**4,118,068**	**2,166,033**	**11,303**
Incremental Number of Avoided Cases/Deaths: From Copenhagen Amendments to Montreal Adjustment					
1890-1980	7,232	1,042	213,249	121,326	670
1985-2010	11,982	1,459	285,232	144,215	708
2015-2050	7,718	868	163,040	80,017	389
2055-2100	0	0	0	0	0
Total	**26,932**	**3,369**	**661,520**	**345,557**	**1,767**

Note: The numbers presented above indicate the number of avoided cases from one policy scenario to another.

Based on data presented in Table 7, Figure 4 through Figure 7 graphically present the incremental health benefits for successively more stringent scenarios for CMM incidence, CMM mortality, NMSC incidence, and NMSC mortality.[23] As shown in Figure 4, the curve representing impacts associated with the Montreal Adjustments most closely approaches the baseline (1979-1980) ozone concentration (at zero on the y-axis) after a number of years, but there remain significant opportunities for further reducing health effects. Because the recovery of ozone (i.e., the return to pre-depletion levels prevalent in the 1979-1980 timeframe) is projected to occur around 2050, no exposures attributable to ozone depletion will accrue for people born after this recovery date. Incremental UV exposures for people born before 2050, however, will continue to result in health effects incidence and mortality after 2050, albeit at a lower rate than in earlier years. By approximately 2150, it is predicted that there will be no living population that experienced incremental exposure associated with depleted ozone levels, and hence, no additional health effects incidence or mortality above those expected to occur under "normal" conditions (i.e., 1979-1980 ozone levels).

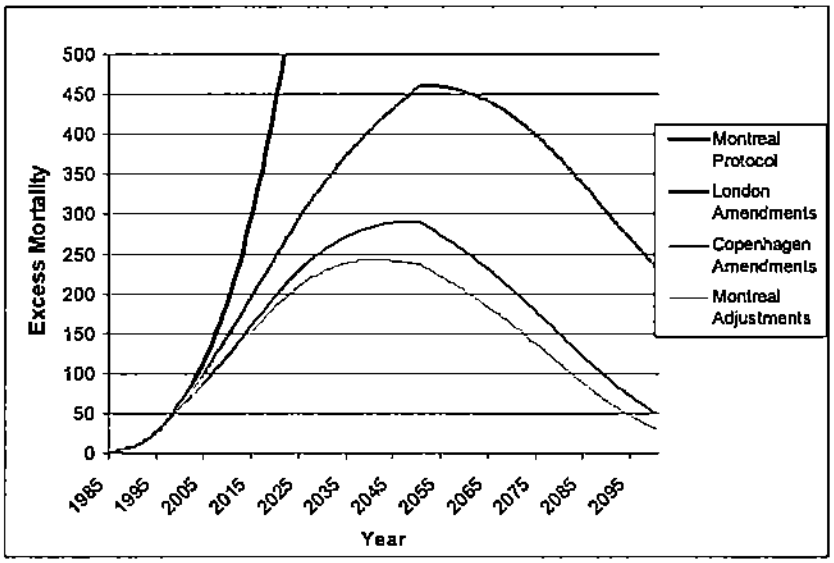
[23] These estimates do not include effects on ozone from climate variation and other factors. How climate may ultimately affect the recovery of stratospheric ozone is unclear and beyond the scope of the AHEF.

Figure 4. Annual Incremental U.S. CMM Incidence through 2100 Under Different ODS Control Policies (SCUP-h Action Spectrum)



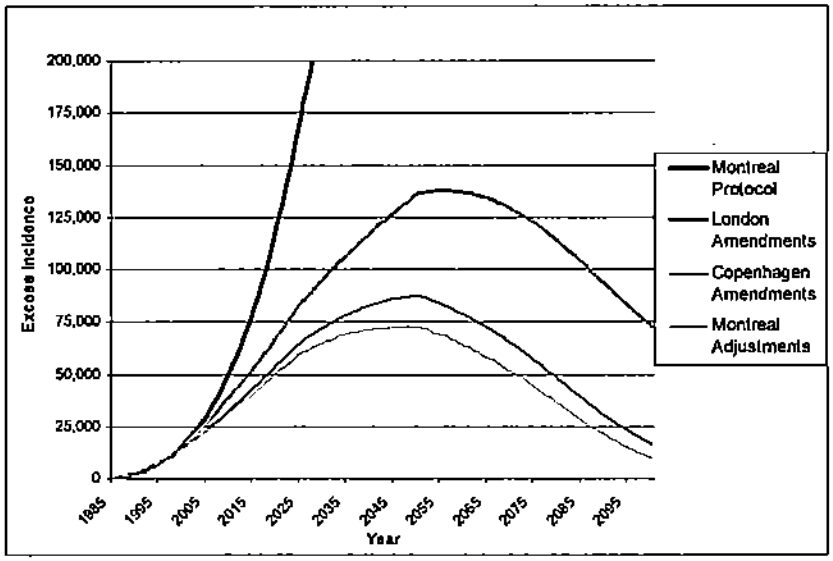
Note: Because this graph shows the incremental CMM incidence relative to the 1979-1980 baseline, the level of CMM incidence in the baseline is represented by zero on the y-axis.

Figure 5. Annual Incremental U.S. CMM Mortality through 2100 Under Different ODS Control Policies (SCUP-h Action Spectrum)



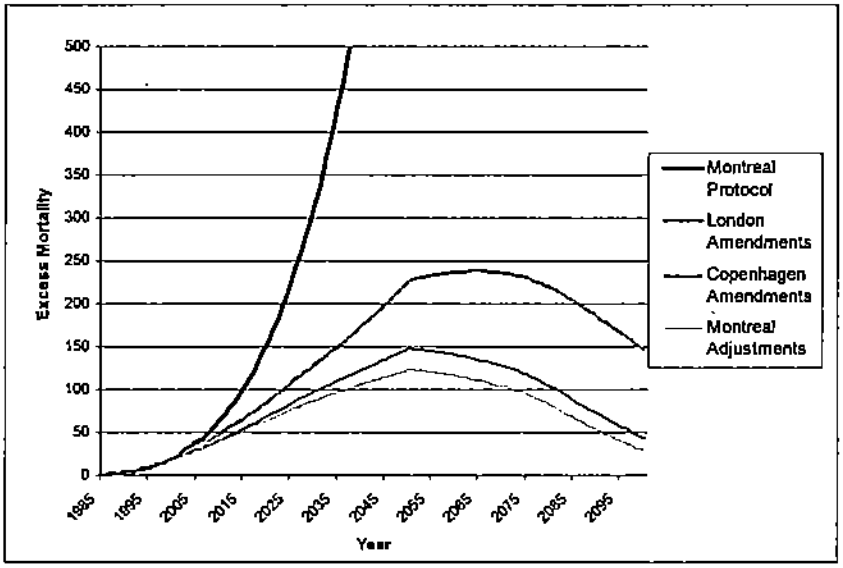
Note: Because this graph shows the incremental CMM mortality relative to the 1979-1980 baseline, the level of CMM mortality in the baseline is represented by zero on the y-axis.

Figure 6. Annual Incremental U.S. NMSC Incidence through 2100 Under Different ODS Control Policies (SCUP-h Action Spectrum)



Note: Because this graph shows the incremental NMSC incidence relative to the 1979-1980 baseline, the level of NMSC incidence in the baseline is represented by zero on the y-axis.

Figure 7. Annual Incremental U.S. NMSC Mortality through 2100 Under Different ODS Control Policies (SCUP-h Action Spectrum)



Note: Because this graph shows the incremental NMSC mortality relative to the 1979-1980 baseline, the level of NMSC mortality in the baseline is represented by zero on the y-axis.

9. Uncertainty Analysis

The AHEF, like any complex modeling framework, uses inputs and computational procedures that introduce uncertainty to the results. These inputs come from various existing sources and are combined with other inputs and procedures derived specifically for this analytical framework. Proper interpretation and use of the human health effects results generated by the AHEF requires some understanding of the nature and magnitudes of the major sources of uncertainty involved. This section uses a combination of empirical analyses and theoretical reasoning to roughly characterize the quantifiable and unquantifiable uncertainties associated with the AHEF's incidence and mortality predictions.

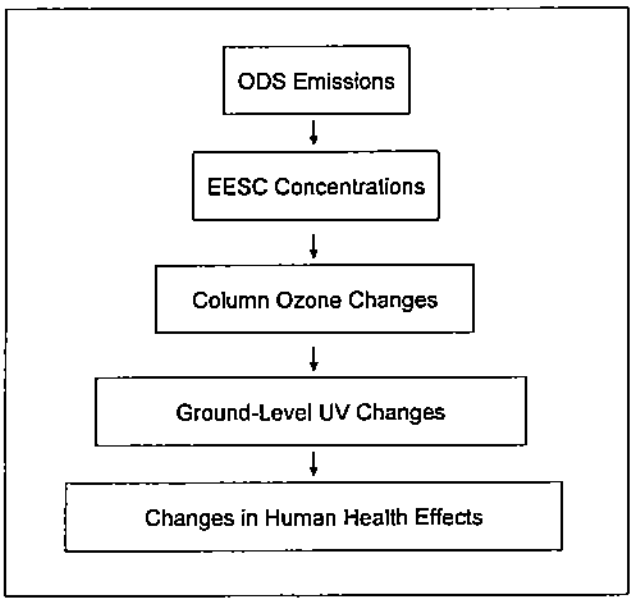
The remainder of this section is organized as follows:

- Section 9.1 focuses on four major sources of uncertainty in the AHEF's estimates of health effects that are considered to be central to its structure, and that have been quantified to the extent possible;
- Section 9.2 presents a discussion of other unquantified sources of uncertainty that affect the AHEF's results, but that are not considered to be central to its structure; and
- Section 9.3 summarizes the quantified and unquantified sources of uncertainty.

9.1 Major Sources of Uncertainty

The AHEF uses past and future ODS emissions to generate equivalent effective stratospheric chlorine (EESC) concentrations, which in turn are used to estimate stratospheric column ozone changes. These column ozone changes then are used to compute changes in ground-level UV radiation, from which estimated changes in human health effects can be calculated. Figure 9 illustrates these model inputs.

Figure 9. Central Computational Procedures Associated with Uncertainty in the AHEF



Each of the linkages identified in the figure is the source of some degree of uncertainty. Although some might attempt to combine these different sources using statistical techniques, it is best to consider each source separately for two reasons. First, the quantitative estimates of the levels of uncertainty of the AHEF's many inputs and modeling components were derived using different techniques of varying levels of precision. Second, and perhaps more important, is that uncertainties concerning some of the inputs and computations might be inversely related. For example, if the TUV's estimated ground-level UV radiation is biased upward, so that variations in UV exposures are too high, then the estimated BAFs (which are derived based on correlation with ground-level UV radiation variation) will be biased downward.

From a purely statistical standpoint, the largest source of uncertainty in the AHEF is the EESC-to-column ozone component, with standard errors around the mean effects ranging from about 25 to over 100 percent. However, as will be discussed in Section 9.1.2, this is a product of the limited data available for the regression analysis and likely does not reflect the true uncertainty that would be revealed with substantially more data.

By contrast, the TUV's estimates of changes in ground-level UV radiation due to changes in column ozone impart statistical uncertainty of up to 10 percent. Similarly, the choice of action spectrum for each health endpoint yields very small variations in the health effects results, with the exception of the DNA-h action spectrum (which is not used in the AHEF), as explained in more detail in Section 9.1.4. The last two sources of quantified uncertainty—the age-weighted exposure scenario assumption and the estimated BAFs—also introduce relatively modest variation in the estimated health effects, of about 11 percent and up to 30 percent, respectively.

Thus, as is true of any complex modeling framework with multiple inputs and computational procedures, the AHEF does contain uncertainties. Perhaps over time, these can be reduced as additional data and research become available. At present, however, the AHEF embodies the best inputs, assumptions, and computational procedures that are known. The remainder of this section discusses the five major areas of uncertainty in greater detail.

9.1.1 Translating ODS Emissions into EESC Concentrations

One source of uncertainty in the AHEF methodology is that the magnitude of ozone depletion and recovery based on ODS emissions could be different from those predicted under the international controls in place now or scheduled for the future. This could occur because ODS use might be less than allowed under the various current and future phaseout requirements, or ODS use could be higher in the future if ODS use exceeds allowable amounts due to non-compliance with the phaseout targets. However, to date, countries have reportedly tended to over-comply with Montreal Protocol obligations (i.e., they have generally undertaken ODS phaseout efforts before the limits imposed by the Protocol take effect), as described in WMO (2003). For example, in 1999, reports of CFC production indicated that production of CFCs was 20,000 ODP-tons less than allowable consumption in that year (WMO 2003).[24] Thus, the scenario of total compliance used in the AHEF may potentially represent the maximum ODS emissions scenario.

Similarly, the parameters that characterize the process of how ODS emissions translate into EESC are also taken to be given, despite the fact that the reaction kinetics of these transitions and the composition

[24] Although scientific measurements of actual CFC-11 and CFC-12 emissions have indicated that mixing ratios were 5 to 10 percent higher than ratios that would have been expected if production levels were identical to those reported, the discrepancy between measurements and reported values could be related to differences in measured and reported values that have occurred throughout the entire measurement period for CFC-11 and CFC-12, rather than as a result of under-reporting in 1999. Supporting this hypothesis, measurement and production values have been closer in recent years (WMO 2003).

of the future atmosphere are also subject to uncertainty (as discussed in more detail in Section 9.2). These inputs are as up-to-date as the available complex atmospheric models can provide. Moreover, undertaking a sensitivity analysis for all of the relevant parameters that translate ODS emissions at the ground into EESC would be prohibitively resource intensive. Hence, the uncertainties in ODS use/emissions to EESC portion of the AHEF's structure are noted, but not quantitatively examined.

9.1.2 Translating EESC Concentrations into Stratospheric Column Ozone

From a statistical standpoint, the largest source of uncertainty in the AHEF is introduced by the limited data points available for use in predicting changes in column ozone resulting from changes in EESC. The reason these factors are statistically uncertain is that they are estimated from a very limited data set of satellite-measured stratospheric ozone concentrations and estimated EESC for the years that stratospheric ozone data are available from NASA's Nimbus-7 Total Ozone Mapping Spectrometer (TOMS) (i.e., 1978 to 1993). Relatively few observations in a data set can lead to large standard errors in any statistical analysis.

Furthermore, UV radiation changes resulting from ozone depletion and ground level pollution (i.e., tropospheric ozone generation) are more accurately measured by spectrally resolved ground-based monitors than by satellite measurements on which the AHEF currently relies (this is discussed further in Section 10.5). Satellite data are not as accurate for measuring ozone concentrations at ground-level as they are at higher altitudes because of the coupling between UV absorption by ozone and UV scattering by aerosols and particulate matter. These considerations are important when the ozone perturbations occur in the lower-to-middle troposphere, where soot and other aerosols are prevalent. When ozone perturbations occur in the stratosphere (i.e., well above the region where scattering occurs), absorption predominates. Thus, the altitude at which ozone perturbations occur can affect UV radiation at the ground level. These effects are not well captured by satellite data and hence, ground level UV monitoring data could help to improve modeling estimates, particularly in urban areas.

Table 11 presents the estimated mean impacts of EESC on column ozone, along with the standard errors, for four different months and for each of the three latitude bands modeled by the AHEF. Because the AHEF estimates EESC by year and then estimates column ozone by month and latitude based on regression analyses using TOMS data, the variation in the AHEF's predicted ozone by month and latitude is attributable in large part to the data source and not the regressions that estimate the impact of EESC on column ozone. EESC is measured in parts per billion and column ozone is measured in Dobson units. Table 11 illustrates that an EESC increase of 1,000 parts per billion (ppb) results in an estimated reduction of 16 Dobson Units of column ozone in January in the 30°N to 20°N latitude band.

Table 11. Means and Standard Errors of EESC to Column Ozone Coefficients for Select Months and Latitudes (Change in Dobson Units for a 1 ppb Change in EESC)

Month	30°N-20°N Latitude Band		40°N-30°N Latitude Band		50°N-40°N Latitude Band	
	Mean	Standard Error	Mean	Standard Error	Mean	Standard Error
January	-0.0160	0.0104 (65%)	-0.0344	0.0124 (36%)	-0.0431	0.0122 (28%)
April	-0.0142	0.0096 (68%)	-0.0268	0.0108 (40%)	-0.0400	0.0107 (27%)
July	-0.0032	0.0055 (172%)	-0.0080	0.0060 (75%)	-0.0103	0.0074 (72%)
October	-0.0077	0.0053 (69%)	-0.0076	0.0045 (59%)	-0.0122	0.0045 (37%)

Standard errors of roughly 25 to over 150 percent indicate large statistical uncertainty of the column ozone coefficients. Until additional data on column ozone from satellite or ground-level measurements are obtained to refine these estimates, such uncertainty cannot be reduced. For additional discussion on the uncertainty associated with the AHEF's column ozone estimates, see Appendix D, which compares AHEF and WMO (1999) predicted ozone concentrations.

9.1.3 Translating Column Ozone into Ground-Level UV Radiation

Uncertainty in the estimation of weighted UV exposure at the Earth's surface was not explicitly quantified. Experts generally agree that the uncertainty contributed by the column ozone-to-UV calculations is relatively small compared to those introduced by other inputs and components of the analysis. Uncertainties in translating column ozone to ground-level UV radiation are dominated by uncertainties in the following:

- *Clear sky radiation model.* The accuracy of the TUV model has been evaluated extensively by comparisons with other models (e.g., Koepke et al. 1998) and with direct measurements of UV radiation (e.g., Shetter et al. 1992, 1996; Kirk et al. 1994; Lantz et al. 1996; Gao et al. 2001; Bais et al. 2003). For spectrally resolved radiation, the agreement is 10% or better for all wavelengths of biological relevance (e.g., Kirk et al. 1994, Bais et al. 2003). For integrated quantities (e.g. biologically effective UV and atmospheric photolysis coefficients), agreement improves to roughly 5% or better due to averaging over the relevant wavelength ranges. These small errors are believed to result primarily from uncertainties in the extraterrestrial irradiance (approximately 5-10% in the UV-B band), the ozone absorption cross-section (less than 2% in the UV-B, De More et al. 1997), and from incomplete knowledge of the atmosphere (e.g., exact aerosol amount) at the time of the measurements.

- *UV perturbations due to clouds and air pollutants.* Clouds and air pollutants generally reduce the UV radiation incident at the Earth's surface. However, as long as cloud cover and pollutant levels remain constant, the relative (percent) changes in UV radiation due to changes in stratospheric ozone are expected to be identical to those computed for cloud-free, pollution-free conditions (WMO 1990). This is because the absorption of photons by stratospheric ozone occurs at altitudes far above those of clouds and air pollutants. Any future systematic changes in cloud cover (e.g., related to climate change) or air pollutants are highly uncertain and speculative, and are not included in the AHEF at the present time. It is recognized, however, that such putative changes could either increase or decrease the average UV radiation levels incident at the Earth's surface.

9.1.4 Translating UV Exposures into Human Health Effects

The final major modeling step in the AHEF's structure that introduces some uncertainty to the estimated health effects is the translation of changes in ground-level UV exposure into incremental skin cancers. This step involves multiplying the percentage change in estimated UV exposure by the BAF for a particular action spectrum, exposure scenario (discussed in Section 5), and health effect. Specifically, three sources of uncertainty come into play: (i) uncertainty associated with choice of action spectrum, (ii) uncertainty regarding exposure period, and (iii) uncertainty in the BAF. Each of these sources of uncertainty is explored further below.

9.1.4.1 Uncertainty associated with choice of action spectrum

An important source of uncertainty in the AHEF's estimates of UV-related health effects is related to a lack of complete understanding regarding the correct weighting for the portions of the UV spectrum that are most effective in causing health effects. Several candidate action spectra have been developed based on both human observations (e.g., erythema) and from laboratory experiments on animals (e.g., SCUP-h), but precisely which spectrum weighting causes particular human health effects remains unknown.

Despite some uncertainty regarding selection of an appropriate action spectrum for each health effect, it is possible to choose among the available spectra based on certain parameters. For example, as Table 12 illustrates for various health effects endpoints under the Montreal Adjustments ODS control scenario,

there is a range of expected incidence and mortality estimates for CMM and NMSC based on which action spectrum is selected. Both the SCUP-h and erythema spectrum have good correlation (within a few percentage points) for the examined health effects, while the DNA-h spectrum has wider variability. This divergence is because the DNA-h action spectrum is more tightly focused on the UV-B portion of the spectrum. Furthermore, there is a poor understanding of the correction factors needed to adjust between viral/bacterial DNA (for which the spectrum was originally developed) and human DNA (i.e., DNA-h).

Table 12. Incremental CMM Incidence and Mortality and NMSC Mortality for Three Action Spectra

Action Spectrum/ All Cohorts	CMM Incidence		CMM Mortality		NMSC Mortality	
	Excess Incidence	Difference from SCUP-h	Excess Mortality	Difference from SCUP-h	Excess Mortality	Difference from SCUP-h
DNA-h	192,494	41.5%	23,767	41.3%	12,457	79.8%
Erythema	133,199	-1.2%	16,421	-1.4%	8267	-2.7%
SCUP-h	134,064	-	16,587	-	8,365	-

As additional data become available on the dose-response relationship for CMM and NMSC, use of the SCUP-h action spectrum may be re-evaluated.

9.1.4.2 Uncertainty regarding exposure period

Another source of uncertainty in the AHEF's health effects estimates is associated with the exposure period over a person's lifetime that is most likely to be the cause of UV-related health effects. This is especially relevant for CMM, since it has been hypothesized that CMM is largely the product of intense exposures early in life (e.g., through age 20) rather than cumulative lifetime exposure. As discussed on page 32 (see Table 10), CMM mortality changes by ±11 percent when the exposure assumptions are changed, with uncertainty concerning the appropriate exposure dose manifesting itself less in the total incremental risks predicted, than in *when* those incremental effects are predicted to occur, and *who* will bear them (i.e., shifting the risk to children born after 1980).

9.1.4.3 Uncertainty in the BAFs

Uncertainty in the BAFs is associated with (1) the accuracy of the BAFs themselves, as measured by the uncertainty ranges, and (2) whether or not the BAF can be appropriately calculated for the health effect of concern, which depends on the selection of the action spectrum. As described in detail below, the uncertainty in the AHEF's predicted excess UV-related human health effects is 6 percent for CMM mortality, 5 percent for NMSC mortality, and 30 percent for NMSC incidence. These uncertainty ranges are small and not significant compared to the levels of uncertainty that are common in health effects assessments for other hazards.

CMM Incidence/Mortality

The BAFs used by the AHEF for CMM incidence and mortality were estimated econometrically by correlating data on latitudinal variations in UV exposure and skin cancer mortality. As with any statistical estimate, these estimated BAFs have standard errors. The estimated BAFs for CMM mortality and their standard errors for the SCUP-h UV action spectrum using the cumulative lifetime UV exposure assumption are shown in Table 13. At a 95 percent confidence interval, the BAF for light-skinned males based on annual exposures ranges from 0.55 to 0.62. This yields an uncertainty range of approximately ±6 percent around the central value (median).

Table 13. Estimated Mean, Standard Errors, and Confidence Intervals for the BAFs for CMM Mortality for the SCUP-h Action Spectrum and Exposure Scenarios, by Sex

	Annual With Clouds				Peak Clear Day (June 21)			
	Light-Skinned Males		Light-Skinned Females		Light-Skinned Males		Light-Skinned Females	
Mean	0.5846		0.5047		1.444		1.310	
Standard Error	0.02		0.02		0.05		0.06	
95% Confidence Limit	*Upper Bound*	*Lower Bound*	*Upper Bound*	*Lower Bound*	*Upper Bound*	*Lower Bound*	*Upper Bound*	*Lower Bound*
	0.62	0.55	0.55	0.46	1.55	1.34	1.43	1.19
97.5% Confidence Limit	*Upper Bound*	*Lower Bound*	*Upper Bound*	*Lower Bound*	*Upper Bound*	*Lower Bound*	*Upper Bound*	*Lower Bound*
	0.63	0.54	0.56	0.45	1.56	1.33	1.45	1.17

Although researchers' understanding of the biology and pathogenesis of CMM tumors has improved in recent years (Nesbit et al. 1998, Fidler 1998), uncertainty remains about the etiology and mechanism(s) of induction of these tumors (Longstreth 1998). While most researchers agree that the primary environmental risk factor for CMM is exposure to sunlight, there is uncertainty about three important aspects of this relationship:

- *Effects of Early Life Exposure.* Some studies indicate that exposures early in life could increase the risk of adult cases of CMM, although preliminary results suggest that high childhood exposures are only important in the context of high adult exposures (Harrison *et al.* 1994, Zanetti *et al.* 1992, Autier and Dore 1998). It has also been hypothesized that chronic low-level UV exposure may even be protective (Holman and Armstrong 1984). Depending on how and if early childhood exposure does indeed influence CMM incidence, and on whether chronic low-level UV exposure may be protective, CMM incidence rates may be under- or over-projected in the AHEF. However, the overall impacts on results are not expected to be great (i.e., up to 11 percent, as explained in Section 8.2).

- *Choice of Appropriate Action Spectrum.* There are no studies on CMM induction in test animals and, as such, an action spectrum specific to CMM has not yet been developed. However, recent studies suggest that the appropriate action spectrum to predict tumor induction may be more dependent on UV-A radiation than previously suspected (Setlow et al. 1993, Ley 1997). The lack of adequate experimental data from which to derive an action spectrum for CMM is one of the greatest sources of uncertainty in estimating UV-induced health impacts. Due to this lack of information, the AHEF predicts CMM cases and deaths based on the SCUP-h action spectrum for SCC. However, this analysis should be reconsidered if future studies aimed at developing an action spectrum for CMM reveal that its shape is not similar to the SCUP-h action spectrum for SCC (DeFabo 2001).

- *Effects of UV-B on Tumor Suppression.* One important variable confounding the dose-response relationship is the effect of UV-B on human tumor suppression genes. It is hypothesized that UV-B may inactivate tumor suppression genes (i.e., the p21 gene), making humans more susceptible to UV-related cancers. More specifically, research indicates that UV light targets the retinoblastoma (RB) pathway of the p21 genetic locus, which contains genes that encode kinase inhibitors and act as tumor suppressors (Kannan *et al.* 2003, Chin *et al.* 1997, Hutchinson 2003). This introduces uncertainty into the AHEF, as the model does not consider how UV independently affects tumor suppression genes and how this may lead to increased UV-related health impacts. Thus, because it is not possible to separate the effects of UV radiation on DNA and the p21 gene, there is some uncertainty regarding the dose-response relationship derived from incidence and

mortality data. Although the degree of uncertainty is not quantified, it is not expected to be significant.

NMSC Mortality

The BAFs used by the AHEF for NMSC mortality were estimated econometrically by correlating data on latitudinal variations in UV exposure and skin cancer mortality. The estimated BAFs for NMSC mortality and their standard errors for the SCUP-h UV action spectrum using the cumulative lifetime UV exposure assumption are shown in Table 14. At a 95 percent confidence interval, the BAF for light-skinned males based on annual exposures ranges from 0.65 to 0.77. This yields an uncertainty range of approximately ±5 percent around the central value (median).

Table 14. Estimated Mean, Standard Errors, and Confidence Intervals for the BAFs for NMSC Mortality for the SCUP-h Action Spectrum and Exposure Scenarios, by Sex

	Annual With Clouds				Peak Clear Day (June 21)			
	Light-Skinned Males		Light-Skinned Females		Light-Skinned Males		Light-Skinned Females	
Mean	0.7094		0.4574					
Standard Error	0.03		0.03		0.07		0.09	
95% Confidence Limit	*Upper Bound*	*Lower Bound*	*Upper Bound*	*Lower Bound*	*Upper Bound*	*Lower Bound*	*Upper Bound*	*Lower Bound*
	0.77	0.65	0.52	0.40	2.21	1.93	1.74	1.39
97.5% Confidence Limit	*Upper Bound*	*Lower Bound*	*Upper Bound*	*Lower Bound*	*Upper Bound*	*Lower Bound*	*Upper Bound*	*Lower Bound*
	0.78	0.64	0.53	0.39	2.22	1.91	1.77	1.36

SCC and BCC Incidence

Table 15 presents the mean BAF values and associated standard errors for SCC and BCC incidence, which were derived by de Gruijl and Forbes (1995) using similar statistical techniques. Sources of uncertainty associated with the BAFs calculated by de Gruijl and Forbes include confounding factors, such as migration, patient reporting delay, high early life exposure, and potential exposure to other carcinogens. Relative error for carcinogenicity caused by wavelengths over 340 nm was still very substantial in 1995. In addition, the model was unable to account for epidermal thickening and pigmentation that alter spectral sensitivity of the skin, although corrections for thicker human epidermises could be applied. Also, differences between mice and humans (e.g., better adaptation of humans to increases in UV exposure) may have influenced the results of applying the hairless mouse model to humans. This yields an upper uncertainty range of approximately 30 percent for the BCC and SCC incidence AHEF estimates.

Table 15. BAFs and Standard Errors for BCC and SCC Incidence

SCC		BCC	
U.S. Males	*U.S. Females*	*U.S. Males*	*U.S. Females*
2.6 ± 0.7	2.6 ± 0.8	1.5 ± 0.5	1.3 ± 0.4

Source: de Gruijl and Forbes 1995.

9.2 Other Unquantified Sources of Uncertainty

There are a number of other sources of uncertainty in the AHEF's health effects predictions. Some of these sources of uncertainty are possible to quantify, but are not central to the structure of the AHEF. Others cannot be quantified because any assumptions or estimates would be simply speculative. These other sources of uncertainty include:

- Composition of the future atmosphere;
- Future conditions of the ozone layer;
- Effect of climate change on ozone depletion;
- Global compliance with modeled policy scenarios;
- Laboratory techniques and instrumentation for deriving action spectra;
- Demographic and human behavioral changes; and
- Baseline information.

Accurate prediction of future changes in human health effects would require consideration of the net effect of all of the factors described above. Although this challenge is beyond the ability of the current state of atmospheric and epidemiological science, these uncertainties are described qualitatively in more detail below. This section concludes with a summary of these uncertainties.

9.2.1 Composition of the Future Atmosphere

The exact composition of the future atmosphere as a result of compliance with different ODS phaseout policies is unknown. As levels of atmospheric chlorine are reduced, the impact of ozone depletion from chlorine and bromine radical species generated from ODS would change. In addition, long-term systematic changes in atmospheric opacity (e.g., clouds, aerosols, other pollutants) will also impact the AHEF's ability to model changes in ozone. Likewise, future changes in climate could result in changes in the atmospheric circulation patterns and therefore could change cloud cover. The impacts of such changes on the predicted recovery of the ozone layer are unknown. All of these uncertainties could influence the AHEF's ability to model atmospheric processes accurately.

9.2.2 Future Conditions of the Ozone Layer

Uncertainties also can be contributed by assumptions regarding the future conditions of the ozone layer in response to the phaseout of ODS. Some computer models predict that the phaseout of ODS will slow and eventually stop the rate of ozone depletion, and suggest that natural ozone-making processes will enable stratospheric ozone to return to 1979-1980 ozone conditions. These models also predict that the recovery will eventually result in increased concentrations beyond 1979-1980 levels[25] (see Chapter 12 in WMO 1999 for more detail). Because there is incomplete knowledge about the behavior of ozone prior to the satellite measurements taken in 1979-1980, the AHEF imposes a limit on future ozone recovery to the conditions observed in 1979-1980.

9.2.3 Effect of Climate Variations on Ozone Depletion

The effects of global climate variations on stratospheric temperature and, in turn, on ozone depletion, are not well understood, and have therefore not been modeled in the AHEF. While this effect is not incorporated into any other international models used to assess future global ozone depletion, it does represent a modeling constraint that should be noted.

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[25] Whether this recovery scenario, called "ozone superabundance," is likely to occur is open to debate, particularly because of the potential for complex interactions between global climate change and stratospheric ozone dynamics. Model computations have predicted both higher and lower amounts of ozone in the future.

9.2.4 Global Compliance with Modeled Policy Scenarios

This analysis assumes global compliance with each of the modeled policy scenarios. To the extent that these limitations are not adhered to, future ODS emissions could be different in both composition and quantity.

9.2.5 Laboratory Techniques and Instrumentation

Additional uncertainty can be contributed by the laboratory techniques and instrumentation used for deriving the action spectra used to weight UV exposure. Discrepancies between the wavelengths of UV radiation intended to be administered and the wavelengths actually received by the test organism can result in orders of magnitude differences in the measured response. In addition, many action spectra are derived using monochromatic light sources that do not fully simulate the polychromatic light received directly from the sun.

9.2.6 Demographic and Behavior Changes

Future demographic and behavior changes that could affect the accuracy of the AHEF include:

- *Changes in human UV exposure behavior*: This evaluation assumes that human exposure behavior remains constant through time, and does not take into account innovations in sun protection technology (e.g., improved sunglasses and sunscreens), increased public awareness of the effects of overexposure to UV, and increased sensitization to the need for early treatment of suspicious lesions.

- *Improvements in medical care/increased longevity*: Improvements in medical care and predictions of increased longevity for many population subgroups could affect estimates of future skin cancer incidence and mortality significantly.

- *Changes in socioeconomic profiles*: Socioeconomic profiles can impact a variety of factors, ranging from demand for air travel to areas where high UV exposure is expected (i.e., the beach), to the types of skin cancer most commonly observed.

- *Changes in population composition and size*: Population composition changes such as the expected increase in Hispanic populations, whose more pigmented skin is thought to decrease skin cancer risk, could have significant effects on future U.S. skin cancer rates.

The above factors are either not easily quantified (e.g., human behavior), or they are not central to the analysis (e.g., improvements in medical care), and are therefore not addressed further in this evaluation.

9.2.7 Accuracy of Baseline Information

It is possible that error is introduced to the AHEF's results through misreporting of skin cancer incidence and mortality data (i.e., the AHEF's baseline estimates). With disease data, under-, over-, and mis-reporting are not uncommon. For example, a recent study revealed that the incidence of CMM has been systematically under-reported in the SEER data (Clegg *et al.* 2002).[26] The original SEER data indicated that CMM rates in white males were relatively flat or even falling (ranging from -11.1 percent to 3.3 percent annually after 1996). However, after adjusting for underreporting, CMM rates were actually found

[26] There is little reason to believe that the SEER CMM incidence under-reporting extends to the NCI-based CMM mortality input information.

to have increased between 3.8 to 4.4 percent annually since 1981 (Clegg *et al.* 2002). Underreporting of CMM incidence is largely attributable to diagnosis in doctors' offices, as opposed to hospitals and other treatment centers with better reporting accuracy. However, the AHEF results are not significantly affected by this underreporting because CMM incidence estimates in the AHEF are not based directly on SEER incidence data. Rather, because the AHEF estimates CMM incidence based on the *ratio* of SEER incidence data to projected annual mortality estimates, and because underreporting would affect both baseline and scenario estimates, the effects on *incremental* changes in CMM incidence would be second order.

9.3 Summary of Quantified and Unquantified Sources of Uncertainty

Of the major sources of uncertainty associated with the AHEF, the total quantified uncertainty is roughly 60 percent, as summarized in Table 16:

Table 16. Major Sources of Quantified Uncertainty

Source of Uncertainty	Quantified Uncertainty
Translating column ozone to ground-level UV	
TUV Model	≈ 5%
Translating UV exposure to human health effects	
Uncertainty in the BAFs	≤ 30%
• CMM mortality (6%)	
• NMSC mortality (5%)	
• NMSC incidence (30%)	
Uncertainty with choice of action spectrum	≈ 50%
Early life exposure vs. whole life exposure	≈ 10%
Total $\sqrt{(5^2 + 30^2 + 50^2 + 10^2)}$	**≈ 60%**

In addition to the major quantified sources of uncertainty listed above, the atmospheric component of the AHEF (i.e., translation of ODS emissions into (a) EESC concentrations and (b) changes in column ozone concentrations) is also a source of uncertainty, though not quantitatively examined in this analysis. It should be noted, however, that this uncertainty associated with the atmospheric parameters used in the AHEF is inherent in all atmospheric models, including those used by WMO in its Scientific Assessment of Ozone Depletion reports (WMO 1990, 1992, 1995, 1999, 2003).

Other unquantified sources of uncertainty discussed above relate to different parts of the AHEF that estimate changes in ozone, changes in UV radiation, and changes in health effects. Table 17 summarizes these unquantified uncertainties.

Table 17. Factors with Unknown Contributions to Uncertainty

Factor	Parameter
Change in Ozone Estimates	▪ Composition of Future Atmosphere ▪ Ability to Model Atmospheric Processes Accurately ▪ Response of Ozone Layer to Changing ODS Concentrations ▪ Effect of Climate Change on Ozone Depletion ▪ Global Compliance with Modeled Policy Scenarios ▪ Changes in Composition and Quantity of ODS Emissions
Change in UV Radiation Estimates	▪ Long-term Systematic Changes in Atmospheric Opacity (e.g., clouds, aerosols, other pollutants)
Change in Health Effect Estimates	▪ Changes in Human UV Exposure Behavior ▪ Laboratory Techniques and Instrumentation for Deriving an Action Spectrum ▪ Improvements in Medical Care/Increased Longevity ▪ Changes in Socioeconomic Factors (e.g., demographics and human behavioral changes) ▪ Baseline Information (e.g., misreporting of skin cancer incidence and mortality data) ▪ Changes in Population Composition and Size

ATTACHMENT 4 TO EXHIBIT 1

World Meteorological Organization
Global Ozone Research and Monitoring Project—Report No. 47

SCIENTIFIC ASSESSMENT OF OZONE DEPLETION: 2002

Pursuant to Article 6 of the Montreal Protocol on Substances that Deplete the Ozone Layer

National Oceanic and Atmospheric Administration
National Aeronautics and Space Administration
United Nations Environment Programme
World Meteorological Organization
European Commission

Table 1-3. Halogenated trace gas lifetimes.

Industrial Designation or Common Name	Chemical Formula	Lifetime (years)	Notes
Halogen-substituted methanes			
HFC-41	CH_3F	2.4	1
HFC-32	CH_2F_2	4.9	1
HFC-23	CHF_3	270	1
FC-14 (carbon tetrafluoride)	CF_4	50 000	2
Methyl chloride	CH_3Cl	1.3	1, 3
Dichloromethane	CH_2Cl_2	0.38	15, 16
Chloroform	$CHCl_3$	0.41	15, 16
Carbon tetrachloride	CCl_4	26	5
HCFC-31	CH_2ClF	1.3	4
HCFC-22	$CHClF_2$	12.0	1, 6
HCFC-21	$CHCl_2F$	1.7	1, 6
CFC-13	$CClF_3$	640	2
CFC-12	CCl_2F_2	100	2, 7
CFC-11	CCl_3F	45	2, 7
Methyl bromide	CH_3Br	0.7	3
Dibromomethane	CH_2Br_2	0.33	8, 15, 16
Bromoform	$CHBr_3$	0.07	8, 15, 16
Bromodifluoromethane	$CHBrF_2$	5.8	4
Bromochloromethane	CH_2BrCl	0.37	8, 15, 16
Bromodichloromethane	$CHBrCl_2$	0.21	7, 15, 1
Dibromochloromethane	$CHBr_2Cl$	0.19	7, 15, 16
Halon-1301	$CBrF_3$	65	2, 7
Halon-1211	$CBrClF_2$	16	9
Halon-1202	CBr_2F_2	2.9	17
Methyl iodide	CH_3I	0.02	7, 15, 16
Diiodomethane	CH_2I_2	Minutes	7, 15, 16
Chloroiodomethane	CH_2ClI	Hours	7, 15, 16
Trifluoroiodomethane	CF_3I	<0.005	2, 15
Halogen-substituted ethanes			
HFC-161	CH_3CH_2F	0.21	2, 15
HFC-152	CH_2FCH_2F	0.60	2, 15
HFC-152a	CH_3CHF_2	1.4	1, 6
HFC-143	CH_2FCHF_2	3.5	1
HFC-143a	CH_3CF_3	52	1
HFC-134	CHF_2CHF_2	9.6	1
HFC-134a	CH_2FCF_3	14.0	1, 6
HFC-125	CHF_2CF_3	29	1, 6
FC-116 (perfluoroethane)	CF_3CF_3	10 000	2
chloroethane	CH_3CH_2Cl	0.11	15
1,1 dichloroethane	CH_2ClCH_2Cl	0.19	10, 15
Methyl chloroform	CH_3CCl_3	5.0	9

Table 1-3, continued.

Industrial Designation or Common Name	Chemical Formula	Lifetime (years)	Notes
HCFC-142b	CH_3CClF_2	17.9	1, 6
HCFC-141b	CH_3CCl_2F	9.3	1, 6
HCFC-123	$CHCl_2CF_3$	1.3	6, 11
HCFC-124	$CHClFCF_3$	5.8	6, 11
CFC-113	CCl_2FCClF_2	85	2
CFC-113a	CCl_3CF_3	NA	NA
CFC-114	$CClF_2CClF_2$	300	2
CFC-115	$CClF_2CF_3$	1700	2
Halon-2402	$CBrF_2CBrF_2$	20	17
Halogen-substituted propanes			
HFC-281ea	CH_3CHFCH_3	0.06	15
HFC-263fb	$CH_3CH_2CF_3$	1.6	2
HFC-245ca	$CH_2FCF_2CHF_2$	6.2	1
HFC-245ea	$CHF_2CHFCHF_2$	4.0	2
HFC-245eb	$CH_2FCHFCF_3$	4.0	12
HFC-245fa	$CHF_2CH_2CF_3$	7.6	1
HFC-236cb	$CH_2FCF_2CF_3$	13.6	1
HFC-236ea	CHF_2CHFCF_3	10.7	1
HFC-236fa	$CF_3CH_2CF_3$	240	1
HFC-227ea	CF_3CHFCF_3	34.2	1
FC-218 (perfluoropropane)	$CF_3CF_2CF_3$	2600	2
n-propyl chloride	$CH_3CH_2CH_2Cl$	0.06	13, 15
HCFC-243cc	$CH_3CF_2CFCl_2$	26.4	4
HCFC-225ca	$CHCl_2CF_2CF_3$	1.9	11
HCFC-225cb	$CHClFCF_2CClF_2$	5.8	11
n-propyl bromide	$CH_3CH_2CH_2Br$	0.04	15, 16
n-propyl iodide	$CH_3CH_2CH_2I$	0.003	7, 15, 16
isopropyl iodide	CH_3CHICH_3	0.002	7, 15
Halogen-substituted higher alkanes			
HFC-365mfc	$CH_3CF_2CH_2CF_3$	8.6	1
HFC-356mcf	$CH_2FCH_2CF_2CF_3$	1.2	1
HFC-356mff	$CF_3CH_2CH_2CF_3$	8.1	1
HFC-338pcc	$CHF_2CF_2CF_2CHF_2$	12.3	1
FC-318 (perfluorocyclobutane)	$c\text{-}C_4F_8$	3200	1
FC-31-10 (perfluorobutane)	C_4F_{10}	2600	2
HFC-43-10mee	$CF_3CHFCHFCF_2CF_3$	15.9	1
HFC-458mfcf	$CF_3CH_2CF_2CH_2CF_3$	23.2	1
FC-41-12 (perfluoropentane)	C_5F_{12}	4100	2
HFC-55-10mcff	$CF_3CF_2CH_2CH_2CF_2CF_3$	7.7	2
FC-51-14 (perfluorohexane)	C_6F_{14}	3200	2

Table 1-3, continued.

Industrial Designation or Common Name	Chemical Formula	Lifetime (years)	Notes
Fluorinated alcohols			
	CF_3CH_2OH	0.41	15
	$CF_3CF_2CH_2OH$	0.39	15
	$(CF_3)_2CHOH$	2.0	4
Fluorinated ethers			
HFE-152a	CH_3OCHF_2	1.6	1
HFE-143a	CH_3OCF_3	4.3	1
HFE-134	CHF_2OCHF_2	26	1
HFE-125	CHF_2OCF_3	136	1
HFE-227ea	$CF_3OCHFCF_3$	11	2
HFCE-235da2	$CHF_2OCHClCF_3$	2.6	2
HFE-236ea2	$CHF_2OCHFCF_3$	5.8	2
HFE-236fa	$CF_3OCH_2CF_3$	3.7	2
HFE-245fa1	$CHF_2CH_2OCF_3$	2.2	2
HFE-245fa2	$CHF_2OCH_2CF_3$	4.9	1
HFE-245cb2	$CH_3OCF_2CF_3$	5.1	4
HFE-254cb2	$CH_3OCF_2CHF_2$	2.6	4
HFE-263fb2	$CH_3OCH_2CF_3$	0.1	2
HFE-329mcc2	$CF_3CF_2OCF_2CHF_2$	6.8	2
HFE-338mcf2	$CF_3CF_2OCH_2CF_3$	4.3	2
HFE-347mcc3	$CH_3OCF_2CF_2CF_3$	5.2	4
HFE-347mcf2	$CF_3CF_2OCH_2CHF_2$	2.8	2
HFE-347pf3	$CHF_2OCH_2CF_2CF_3$	5.9	4
HFE-347sy2	$CF_3CF(OCH_3)CF_3$	3.7	4
HFE-356mec3	$CH_3OCF_2CHFCF_3$	0.94	2
HFE-356mff2	$CF_3CH_2OCH_2CF_3$	0.4	15
HFE-356pcc3	$CH_3OCF_2CF_2CHF_2$	0.93	2
HFE-356pcf3	$CHF_2OCH_2CF_2CHF_2$	3.6	4
HFE-356pcf2	$CHF_2CH_2OCF_2CHF_2$	2	2
HFE-365mcf3	$CH_3OCH_2CF_2CF_3$	0.11	2, 15
HFE-374pc2 [a]	$CH_3CH_2OCF_2CHF_2$	5	2
	$CF_3CH(OCF_3)CHF_2$	3.1	2
	$(CF_3)_2CFOCH_3$	3.4	2
HFE-7100	$C_4F_9OCH_3$	5	2
HFE-7200	$C_4F_9OC_2H_5$	0.77	2
H-Galden 1040x [b]	$CHF_2OCF_2OCF_2CF_2OCHF_2$	6.3	2
HFE-236ca12	$CHF_2OCF_2OCHF_2$	12.1	2
HFE-338pcc13	$CHF_2OCF_2CF_2OCHF_2$	6.2	2
Other fluorinated species			
Trifluoromethylsulfurpentafluoride	SF_5CF_3	800	18
Sulfur hexafluoride	SF_6	3200	2

Table 1-3, continued.

Footnotes

For completeness, estimates for local lifetimes for some very short-lived ($\tau < 0.5$ years) species are included. As discussed in Chapter 2, the atmospheric lifetimes for these species (defined as the ratio of burden to emission; see Prather and Ehhalt et al., 2001) depend on the location and time of emission. Thus, these local lifetimes should not be used in estimation of semiempirical ODP, GWP, or EESC calculations for these gases.

Notes:

1. Global lifetime estimated from a process lifetime with respect to tropospheric OH calculated relative to 6.1 years for CH_3CCl_3, assuming an average temperature of 272 K (Spivakovsky et al., 2000; Prather and Ehhalt et al., 2001); OH rate constants from Sander et al. (2002); and stratospheric loss lifetimes inferred from IPCC (2001).
2. Prather and Ehhalt et al. (2001) and Ramaswamy et al. (2001).
3. See Section 1.5 for further discussion related to methyl halide global lifetimes.
4. Lifetime calculated as in Note 1 except that no estimate of a stratospheric loss lifetime was available to include in the lifetime estimate listed. Hence this is an upper bound to the global lifetime estimate.
5. IPCC (2001) and including an oceanic loss term with 94-year lifetime observed in saturation data and ascribed to an unidentified process (Yvon-Lewis and Butler, 2002).
6. Including oceanic loss term from Yvon-Lewis and Butler (2002). The contribution of oceanic loss to the lifetime of HCFC-21, HCFC-22, HCFC-123, HCFC-124, HCFC-141b, HFC-125, and HFC-152a is small; for HFC-134a and HCFC-142b it is negligibly small at the reported precision.
7. WMO (1999).
8. Lifetimes listed include local tropospheric photolysis lifetimes from Table 2-9 in Kurylo and Rodriguez et al. (1999). Consideration of only tropospheric OH loss results in local lifetimes of 0.34 years for CH_2Br_2, 0.21 years for $CHBr_3$, and 0.37 years for CH_2BrCl.
9. See Section 1.4 text for discussion.
10. OH rate constant from Qui et al. (1992).
11. Lifetime calculated as in Note 1, but with stratospheric loss from Naik et al. (2000).
12. Lifetime calculated as in Note 1, but with OH rate constant and stratospheric loss from Naik et al. (2000).
13. Markert and Nielsen (1992).
14. OH rate constant from DeMore and Bayes (1999).
15. The values estimated correspond to local lifetimes in the free troposphere. For species that react with OH, the process lifetime due to OH reaction is calculated using the rate constant at 275 K (for lifetimes greater than 10 days) or 300 K (for lifetimes less than 10 days) and OH concentration of 1×10^6 molec cm^{-3}. These should not be used in estimating ODP, GWP, or EESC because the atmospheric burden for these short-lived gases ($\tau <$ 0.5 years) depends on the location and time of emissions.
16. See Chapter 2.
17. From the 2-D model calculation in Fraser et al. (1999).
18. Takahashi et al. (2002).

[a] Referred to as HFE-374pcf2 in past Assessments.

[b] Also known as HFE-43-10pccc124.

Previous lifetime estimates of carbon tetrachloride were derived from observations and modeling of photolytic loss rates in the stratosphere (Volk et al., 1997; Prinn and Zander et al., 1999). Reports of widespread observations of undersaturation of carbon tetrachloride in the ocean (Huhn et al., 2001; Wallace et al., 1994) suggest an additional, significant loss for this gas (Yvon-Lewis and Butler, 2002). When combined with the other loss processes, a revised global lifetime of 26 (17-36) years is now calculated for this gas (Yvon-Lewis and Butler, 2002).

1.4.3 Fractional Release Factors

In considering the effect of halogen source gases on ozone, it is first necessary to determine the composition of tropospheric air entering the stratosphere, mostly through the tropical tropopause. For compounds with lifetimes measured in years, the amount of halogen entering the stratosphere for a specific level of emission is inversely related to the lifetime at steady state. Once a halogen source gas is in the stratosphere, release of a halogen atom from the source gas through photolysis or chemical reaction is a function of stratospheric local lifetime or loss frequency and differs greatly from compound to compound. More complete release of the halogen atom catalyst in the stratosphere produces a greater extent of local photochemical loss of ozone, for a given source gas or in comparing source gases, all else being equal.

A point measurement of a given halogen source molecule in the stratosphere can be recast as a fractional release (FR), defined as

$$FR = \frac{\rho_{entry} - \rho_{point}}{\rho_{entry}} \qquad (1\text{-}5)$$

where ρ is the source compound mixing ratio or mole frac-