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Environmental effects of ozone depletion: 1998 Assessment

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Environmental effects of ozone depletion: 1998 assessment

Introduction

The Montreal Protocol on Substances that Deplete the Ozone Layer requires periodic assessments of available scientific, environmental, technical and economic information. The assessments shall be made at least every four years. Assessments were made in 1989, 1991, 1994, and the present one, in 1998. The 1998 assessment focuses on new information since 1994, but it includes some background of prior information, so that it can be read without having the earlier reports at hand.

In 1994, the ozone layer was predicted to become thinner until about 1998, and to recover gradually thereafter. Taking into account new information, the Atmospheric Science Panel now expects that the most vulnerable period for ozone depletion will be extended into the coming two decades. Scientific studies are continuing on the most important effects, and on what can be done to prevent or mitigate these.

The present assessment deals with the results of such investigations. These repeatedly give reasons for concern for potential effects, but relatively little progress has been made in quantifying these effects. The more the investigators look into the problems, the more the complexity becomes apparent. Nevertheless, the knowledge is accumulating.

In comparison with the earlier assessments on effects of ozone depletion, the present report has a new section, Frequently Asked Questions.

J.C. van der Leun

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EXECUTIVE SUMMARY

Decreased quantities of total-column ozone are now observed over large parts of the globe, permitting increased penetration of solar UV-B radiation (280-315 nm) to the Earth´s surface. The present assessment deals with the possible consequences. The Atmospheric Science Panel predicts that the ozone layer will be in its most vulnerable state during the coming two decades. Some of the effects are expected to occur during most of the next century. Recent studies show that the effects of ozone depletion would have been dramatically worse without the protective measures taken under the Montreal Protocol.

The assessment is given in seven chapters, summarised as follows:

Changes in Ultraviolet Radiation

- **Stratospheric ozone levels are near their lowest point since measurements began, so current UV-B radiation levels are thought to be close to their maximum.** Total stratospheric content of ozone-depleting substances is expected to reach a maximum before the year 2000. All other things being equal, the current ozone losses and related UV-B increases should be close to their maximum. Increases in surface erythemal (sun-burning) UV radiation relative to the values in the 1970s are estimated to be:
  - about 7% at Northern Hemisphere mid-latitudes in winter/spring;
  - about 4% at Northern Hemisphere mid-latitudes in summer/fall;
  - about 6% at Southern Hemisphere mid-latitudes on a year-round basis;
  - about 130% in the Antarctic in the spring; and
  - about 22% in the Arctic in the spring.

- **The correlation between increases in surface UV-B radiation and decreases in overhead ozone has been further demonstrated and quantified by ground-based instruments under a wide range of conditions.** Improved measurements of UV-B radiation are now providing better geographical and temporal coverage. Surface UV-B radiation levels are highly variable because of sun angle, cloud cover, and also because of local effects including pollutants and surface reflections. With a few exceptions, the direct detection of UV-B trends at low and mid-latitudes remains problematic due to this high natural variability, the relatively small ozone changes, and the practical difficulties of maintaining long-term stability in networks of UV-measuring instruments. Few reliable UV-B radiation measurements are available from pre-ozone depletion days.

- **Satellite-based observations of atmospheric ozone and clouds are being used, together with models of atmospheric transmission, to provide global coverage and long-term estimates of surface UV-B radiation.** Estimates of long term (1979-1992) trends in zonally-averaged UV-irradiances that include cloud effects are nearly identical to those for clear-sky estimates, providing evidence that clouds have not influenced the UV-B trends. However, the limitations of satellite-derived UV estimates should be recognized. To assess uncertainties inherent in this approach, additional validations involving comparisons with ground-based observations are required.

- **Direct comparisons of ground-based UV-B radiation measurements between a few mid-latitude sites in the Northern and Southern Hemispheres have shown larger differences than those estimated using satellite data.** Ground-based measurements show that summertime erythemal UV irradiances in the Southern Hemisphere exceed those at comparable latitudes of the

Northern Hemisphere by up to 40%, whereas corresponding satellite-based estimates yield only 10 to 15% differences. Atmospheric pollution may be a factor in this discrepancy between ground-based measurements and satellite-derived estimates. UV-B measurements at more sites are required to determine whether the larger observed differences are globally representative.

- **High levels of UV-B radiation continue to be observed in Antarctica during the recurrent spring-time ozone hole.** For example, during ozone hole episodes, measured biologically-damaging radiation at Palmer Station, Antarctica (64°S) has been found to approach and occasionally even exceed maximum summer values at San Diego, USA (32°N).

- **Long term predictions of future UV-B levels are difficult and uncertain. Nevertheless, current best estimates suggest that a slow recovery to pre-ozone depletion levels may be expected during the next half-century.** Although the maximum ozone depletion, and hence maximum UV-B increase, is likely to occur in the current decade, the ozone layer will continue to be in its most vulnerable state into the next century. The peak depletion and the recovery phase could be delayed by decades because of interactions with other long-term atmospheric changes, e.g. increasing concentrations of greenhouse gases. Other factors that could influence the recovery include non-ratification and/or non-compliance with the Montreal Protocol and its Amendments and Adjustments, and future volcanic eruptions. The recovery phase for surface UV-B irradiances will probably not be detectable until many years after the ozone minimum.

Effects on Human and Animal Health

- **Recent estimates suggest that the increase in the risk of cataract and skin cancer due to ozone depletion would not have been adequately controlled by implementation of the Montreal Protocol (1987) alone but can be achieved through implementation of its later provisions.** Risk assessments for the US and the Northwestern Europe indicate large increases in cataracts and skin cancers under either the 'no Protocol' or the early Montreal Protocol scenarios. Under scenarios based on the later amendments, Copenhagen (1992) and Montreal (1997), increases in cataracts and skin cancer attributable to ozone depletion return almost to zero by the end of the next century.

- **The increases in UV-B radiation associated with ozone depletion are likely to lead to increases in the incidence and/or severity of a variety of short-term and long-term health effects, if current exposure practices are not modified by changes in behavior.**

- **Adverse effects on the eye will affect all populations irrespective of skin color.** Adverse impacts could include: more cases of acute reactions such as 'snowblindness'; increases in cataract incidence and/or severity (and thus the incidence of cataract-associated blindness); and increases in the incidence (and mortality) from ocular melanoma and squamous cell carcinoma of the eye.
- **Effects on the immune system will also affect all populations but may be both adverse and beneficial.** Adverse effects include depressed resistance to certain tumors and infectious diseases, potential impairment of vaccination responses, and possibly increased severity of some autoimmune and allergic responses. Beneficial effects could include decreases in the severity of certain immunologic diseases/conditions such as psoriasis and nickel allergy.
- **Effects on the skin could include increases in photoaging, and skin cancer with risk increasing with fairness of skin.** Increases in UV-B are likely to accelerate the rate of photoaging, as well

as increase the incidence (and associated mortality) of melanoma and the non-melanoma skin cancer, basal cell and squamous cell carcinoma.

- **Research is generating much new information that is being used to help reduce the uncertainties associated with the current risk estimates.** Evaluation of the impact of susceptibility genes is helping to identify highly susceptible populations so that their special risk can be assessed. Examination of the impacts of behavior changes such as consuming diets that are high in antioxidants, avoiding sun exposure during the four hours around solar noon, wearing covering apparel, e.g., hats, sunglasses, is beginning to identify important exposure patterns as well as possible mitigation strategies.

- **Quantitative risk assessments for a variety of other effects, such as UV-B induced immunosuppression of infectious diseases, are not yet possible.** New information continues to confirm the reasonableness of these concerns, but data adequate for quantitative risk assessment are not yet available.

Effects on Terrestrial Ecosystems

- **Increased UV-B can be damaging for terrestrial organisms including plants and microbes, but these organisms also have protective and repair processes.** The balance between damage and protection varies among species and even varieties of crop species; many species and varieties can accommodate increased UV-B. Tolerance of elevated UV-B by some species and crop varieties provides opportunities for genetic engineering and breeding to deal with potential crop yield reductions due to elevated UV-B in agricultural systems.

- **Research in the past few years indicates that increased UV-B exerts effects more often through altered patterns of gene activity rather than damage.** These UV-B effects on regulation manifest themselves in many ways including changes in life cycle timing, changes in plant form and production of plant chemicals not directly involved in primary metabolism. These plant chemicals play a role in protecting plants from pathogens and insect attack, and affect food quality for humans and grazing animals.

- **Terrestrial ecosystem responses to increased UV-B are evident primarily in interactions among species, rather than in the performance of individual species.** Much of the recent experimentation indicates that increased UV-B affects the balance of competition among higher plants, the degree to which higher plants are consumed by insects and susceptibility of plants to pathogens. These effects can be mediated in large part by changes in plant form and chemistry, but effects of UV-B on insects and microbes are also possible. The direction of these UV-B-mediated interactions among species is often difficult to predict based only on single-organism responses to increased UV-B.

- **Effects of increased UV-B radiation may accumulate from year to year in long-lived perennial plants and from generation to generation in annual plants.** This effect has been shown in a few recent studies, but the generality of this accumulation among species is not presently known. If this phenomenon is widespread, this would amplify otherwise subtle responses to UV-B seen in a single growing season, for example in forest trees.

- **Effects of increased UV-B must be taken into account together with other environmental factors including those associated with global change.** Responses of plants and other organisms to increased UV-B are modified by other environmental factors such as CO_2, water stress, mineral nutrient availability, heavy metals and temperature. Many of these factors also are changing as the global climate is altered.

Effects on Aquatic Ecosystems

- **Recent studies continue to demonstrate that solar UV-B and UV-A have adverse effects on the growth, photosynthesis, protein and pigment content, and reproduction of phytoplankton, thus affecting the food web.** These studies have determined biological weighting functions and exposure-response curves for phytoplankton, and have developed new models for the estimation of UV-related photoinhibition. In spite of this increased understanding and enhanced ability to model aquatic impacts, considerable uncertainty remains with respect to quantifying effects of ozone-related UV-B increases at the ecosystem level.

- **Macroalgae and seagrasses show a pronounced sensitivity to solar UV-B.** They are important biomass producers in aquatic ecosystems. Most of these organisms are attached and so cannot avoid being exposed to solar radiation at their growth site. Effects have been found throughout the top 10-15 m of the water column.

- **Zooplankton communities as well as other aquatic organisms including sea urchins, corals and amphibians are sensitive to UV-B.** There is evidence that for some of these populations even current levels of solar UV-B radiation, acting in conjunction with other environmental stresses, may be a limiting factor but quantitative evaluation of possible effects remains uncertain.

- **UV-B radiation is absorbed by and breaks down dissolved organic carbon (DOC) and particulate organic carbon (POC) and makes the products available for bacterial degradation and remineralization.** The degradation products are of importance in the cycling of carbon in aquatic ecosystems. Because UV-B breaks down DOC as it is absorbed, increases in UV-B can increase the penetration of both UV-B and UV-A radiation into the water column. As a consequence, the quantity of UV-B penetrating to a given depth both influences and is influenced by DOC. Warming and acidification result in faster degradation of these substances and thus enhance the penetration of UV radiation into the water column.

- **Polar marine ecosystems, where ozone-related UV-B increases are the greatest, are expected to be the oceanic ecosystems most influenced by ozone depletion.** Oceanic ecosystems are characterized by large spatial and temporal variabilities that make it difficult to select out UV-B specific effects on single species or whole phytoplankton communities. While estimates of reduction in both Arctic and Antarctic productivity are based upon measurable short-term effects, there remain considerable uncertainties in estimating long-term consequences, including possible shifts in community structure. Reduced productivity of fish and other marine crops could have an economic impact as well as affect natural predators; however quantitative estimation of the possible effects of reduced production remain controversial.

- **Potential consequences of enhanced levels of exposure of aquatic ecosystems to UV-B radiation include reduced uptake capacity for atmospheric carbon dioxide, resulting in the poten-**

tial augmentation of global warming. The oceans play a key role with respect to the budget of greenhouse gases. Marine phytoplankton are a major sink for atmospheric carbon dioxide and they have a decisive role in the development of future trends of carbon dioxide concentrations in the atmosphere. The relative importance of the net uptake of carbon dioxide by the biological pump and the possible role of increased UV-B in the ocean are still controversial.

Effects on Biogeochemical Cycles

- **Effects of increased UV-B on emissions of carbon dioxide and carbon monoxide (CO) and on mineral nutrient cycling in the terrestrial biosphere have been confirmed by recent studies of a range of species and ecosystems.** The effects, both in magnitude and direction, of UV-B on trace gas emissions and mineral nutrient cycling are species-specific and operate on a number of processes. These processes include changes in the chemical composition in living plant tissue, photodegradation (breakdown by light) of dead plant matter, including litter, release of carbon monoxide from vegetation previously charred by fire, changes in the communities of microbial decomposers and effects on nitrogen-fixing micro-organisms and plants. Long-term experiments are in place to examine UV-B effects on carbon capture and storage in biomass within natural terrestrial ecosystems.

- **Studies in natural aquatic ecosystems have indicated that organic matter is the primary regulator of UV-B penetration.** Enhanced UV-B can affect the balance between the biological processes that produce the organic matter and the chemical and microbial processes that degrade it**.** Changes in the balance have broad impacts on the effects of enhanced UV-B on biogeochemical cycles. These changes, which are reinforced by changes in climate and acidification, result from clarification of the water and changes in light quality.

- **Increased UV-B has positive and negative impacts on microbial activity in aquatic ecosystems that can affect carbon and mineral nutrient cycling as well as the uptake and release of greenhouse and chemically-reactive gases.** Photoinhibition of surface aquatic micro-organisms by UV-B can be partially offset by photodegradation of dissolved organic matter to produce substrates, such as organic acids and ammonium, that stimulate microbial activity.

- **Modeling and experimental approaches are being developed to predict and measure the interactions and feedbacks between climate change and UV-B induced changes in marine and terrestrial biogeochemical cycles.** These interactions include alterations in the oxidative environment in the upper ocean and in the marine boundary layer and oceanic production and release of CO, volatile organic compounds (VOC), and reactive oxygen species (such as hydrogen peroxide and hydroxyl radicals). Climate related changes in temperature and water supply in terrestrial ecosystems interact with UV-B radiation through biogeochemical processes operating on a wide range of time scales.

Effects on Air Quality

- **Increased UV-B will increase the chemical activity in the lower atmosphere (the troposphere).** Tropospheric ozone levels are sensitive to local concentrations of nitrogen oxides (NO_x) and hydrocarbons. Model studies suggest that additional UV-B radiation reduces tropospheric ozone in clean environments (low NO_x), and increases tropospheric ozone in polluted areas (high NO_x).

- **Assuming other factors remain constant, additional UV-B will increase the rate at which primary pollutants are removed from the troposphere**. Increased UV-B is expected to increase the concentration of hydroxyl radicals (OH) and result in faster removal of pollutants. Increased concentrations of oxidants such as hydrogen peroxide and organic peroxides are also expected. The effects of UV-B increases on tropospheric ozone, OH, methane, carbon monoxide, and possibly other tropospheric constituents, while not negligible, will be difficult to detect because the concentrations of these species are also influenced by many other variable factors (e.g., emissions).

- **No significant effects on humans or the environment have been identified from TFA produced by atmospheric degradation of HCFCs and HFCs.** Numerous studies have shown that TFA has, at most, moderate short-term toxicity. Insufficient information is available to assess potential chronic, developmental, or reproductive effects. The atmospheric degradation mechanisms of most substitutes for ozone depleting substances are well established. HCFCs and HFCs are two important classes of substitutes. Atmospheric degradation of HCFC-123 (CF_3CHCl_2), HCFC-124 (CF_3CHFCl), and HFC-134a (CF_3CH_2F) produces trifluoroacetic acid (TFA). Reported measurements of TFA in rain, rivers, lakes, and oceans show it to be a ubiquitous component of the hydrosphere, present at levels much higher than can be explained by currently reported sources. The levels of TFA currently produced by the atmospheric degradation of HFCs and HCFCs are estimated to be orders of magnitude below those of concern and make only a minor contribution to the current environmental burden of TFA.

Effects on Materials

- **Physical and mechanical properties of polymers are negatively affected by increased UV-B in sunlight.** Increased UV-B reduces the useful lifetimes of synthetic polymer products used outdoors and of biopolymer materials such as wood, paper, wool and cotton. The reduction in service life of materials depends on the synergistic effect of increased UV-B and other factors, especially the temperature of the material during exposure to sunlight. Even under harsh UV exposure conditions the higher temperatures largely determine the extent of increased UV-induced damage to photostabilized polyethylenes. However, accurate assessment of such damage to various materials is presently difficult to make due to limited availability of technical data, especially on the relationship between the dose of UV-B radiation and the resulting damage of the polymer or other material.

- **Conventional photostabilizers are likely to be able to mitigate the effects of increased UV levels in sunlight.** More effective photostabilizers for plastics have been commercialized in recent years. The use of these compounds allows plastic polymer products to be used in a wide range of different UV environments found worldwide. It is reasonable to expect existing photostabilizer technologies to be able to mitigate these effects of an increased UV-B on polymer materials. This, however, would increase the cost of the relevant polymer products, surface coatings, and treated biopolymer materials. However, the efficiencies of even the conventional photostabilizers under the unique exposure environments resulting from an increase in solar UV-B have not been well studied.

CHAPTER 1

CHANGES IN BIOLOGICALLY ACTIVE ULTRAVIOLET RADIATION REACHING THE EARTH'S SURFACE

S. Madronich (USA), R. L. McKenzie (New Zealand), L. O. Björn (Sweden), and M. M. Caldwell (USA)

Summary

Stratospheric ozone levels are near their lowest point since measurements began, so current UV-B radiation levels are thought to be close to their maximum**.** Total stratospheric content of ozone-depleting substances is expected to reach a maximum before the year 2000. All other things being equal, the current ozone losses and related UV-B increases should be close to their maximum. Increases in surface erythemal UV radiation relative to the values in the 1970s are estimated to be:
- about 7% at Northern Hemisphere mid-latitudes in winter/spring;
- about 4% at Northern Hemisphere mid-latitudes in summer/fall;
- about 6% at Southern Hemisphere mid-latitudes on a year-round basis;
- about 130% in the Antarctic in spring; and
- about 22% in the Arctic in spring.

Reductions in atmospheric ozone are expected to result in higher amounts of ultraviolet-B (UV-B) radiation reaching the Earth's surface. The expected correlation between increases in surface UV-B radiation and decreases in overhead ozone has been further demonstrated and quantified by ground-based instruments under a wide range of conditions. Improved measurements of UV-B radiation are now providing better geographical and temporal coverage. Surface UV-B radiation levels are highly variable because of cloud cover, and also because of local effects including pollutants and surface reflections. These factors usually decrease atmospheric transmission and therefore the surface irradiances at UV-B as well as other wavelengths. Occasional cloud-induced increases have also been reported.

With a few exceptions, the direct detection of UV-B trends at low and mid-latitudes remains problematic due to this high natural variability, the relatively small ozone changes, and the practical difficulties of maintaining long-term stability in networks of UV-measuring instruments. Few reliable UV-B radiation measurements are available from pre-ozone depletion days.

Satellite-based observations of atmospheric ozone and clouds are being used, together with models of atmospheric transmission, to provide global coverage and long-term estimates of surface UV-B radiation. Estimates of long term (1979-1992) trends in zonally-averaged UV-irradiances that include cloud effects are nearly identical to those for clear-sky estimates, providing evidence that clouds have not influenced the UV-B trends. However, the limitations of satellite-derived UV estimates should be recognized. To assess uncertainties inherent in this approach, additional validations involving comparisons with ground-based observations are required.

Direct comparisons of ground-based UV-B radiation measurements between a few mid-latitude sites in the Northern and Southern Hemispheres have shown larger differences than those estimated using satellite data. Ground-based measurements show that summertime erythemal UV irradiances in the Southern Hemisphere exceed those at comparable latitudes of the Northern Hemisphere by up to 40%, whereas corresponding satellite-based estimates yield only 10 to 15% differences. Atmospheric pollution may be a factor in this discrepancy between ground-based measurements and satellite-derived estimates. UV-B measurements at more sites are required to determine whether the larger observed differences are globally representative.

High levels of UV-B radiation continue to be observed in Antarctica during the recurrent springtime ozone hole. For example, during ozone hole episodes, measured biologically-damaging radiation at Palmer Station, Antarctica (64°S) has been found to approach and occasionally even exceed maximum summer values at San Diego, USA (32°N).

Long term predictions of future UV-B levels are difficult and uncertain. Nevertheless, current best estimates suggest that a slow recovery to pre-ozone depletion levels may be expected during the next half-century. Although the maximum ozone depletion, and hence maximum UV-B increase, is likely to occur in the current decade, the ozone layer will continue to be in its most vulnerable state into the next century. The peak depletion and the recovery phase could be delayed by decades because of interactions with other long-term atmospheric changes, e.g. increasing concentrations of greenhouse gases. Other factors that could influence the recovery include non-ratification and/or non-compliance with the Montreal Protocol and its Amendments and Adjustments, and future volcanic eruptions. The recovery phase for surface UV-B irradiances will probably not be detectable until many years after the ozone minimum.

Introduction

An important consequence of stratospheric ozone depletion is the increased transmission of solar ultraviolet (UV) radiation to the Earth's lower atmosphere and surface. UV radiation is known to affect many biological and chemical processes, and is largely detrimental to individual organisms. Specific concerns include increases in the incidence of skin cancer, ocular damage, and other health effects in humans and animals; damage to terrestrial and oceanic vegetation; damage to some outdoor materials; changes in the chemistry of the lower atmosphere (the troposphere), e.g. photochemical smog formation; and alterations of the biogeochemical cycles of non-living organic and inorganic matter whose degradation depends on the exposure to ambient solar radiation (UNEP, 1991; 1994; 1998).

Environmental UV radiation is highly variable. Some of these variations are easily quantified, such as those due to changes in the solar elevation with latitude, time of day and season. Variations in the atmospheric ozone column amount are of direct importance to surface UV radiation. Continuous ozone observations are available from ground-based stations since the 1950s for a number of locations, and near-global observations are available from satellite-based instruments for most of the period since 1979. Other factors, such as clouds, are much less predictable and their spatial and temporal distributions are still poorly characterized, especially on local scales and for short-term fluctuations. Additional localized perturbations may stem from surface elevation and reflections, and from variable atmospheric turbidity associated with air pollution.

In this report, the dependence of UV radiation on atmospheric ozone is emphasized. Systematic reductions in ozone have been observed in the last two decades, a likely result of human activities and in particular the emission of halogen-containing compounds of mostly anthropogenic origin (WMO,

1994a; 1998). Observations confirm that ozone reductions lead to increased UV radiation levels at the Earth's surface, if all other factors that influence atmospheric transmission (e.g., clouds, pollutants) are constant. However, it is also necessary to view these ozone-related UV increases in the context of the natural UV variability, and to consider the possibility of long-term changes in other factors such as cloud cover and air pollution.

Biologically Active UV Radiation

The solar radiation at the top of the Earth's atmosphere contains a significant amount of radiation of wavelength (λ) shorter - and therefore more energetic - than visible light (400-700 nm). Wavelengths in the range 100-400 nm constitute the ultraviolet (UV) spectral region. The shortest of these wavelengths (UV-C, 100-280 nm) are blocked (absorbed) essentially completely by atmospheric oxygen

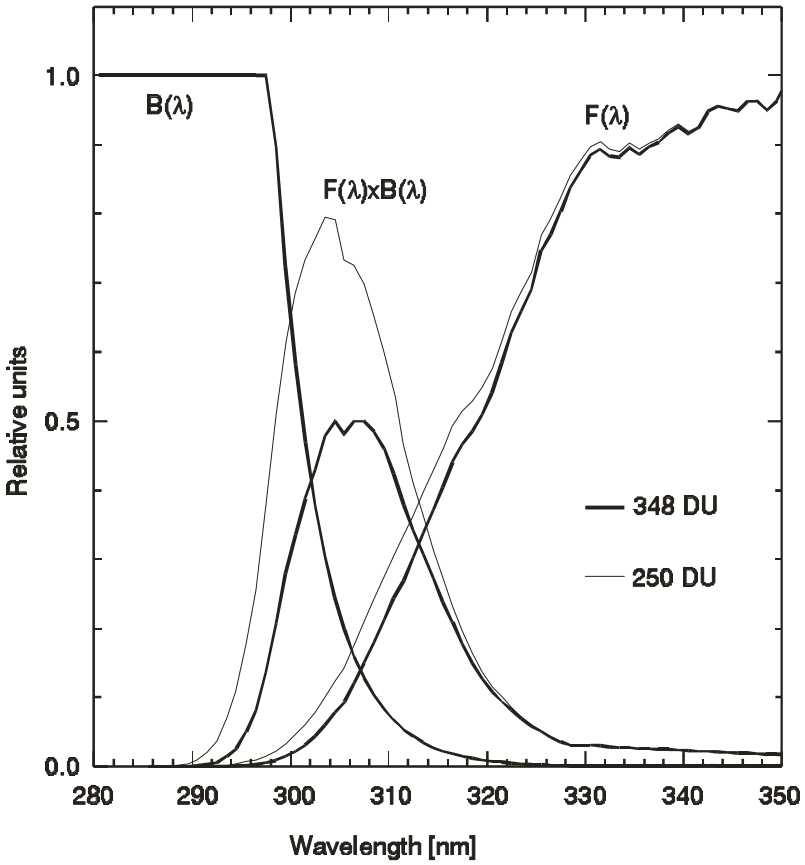

Fig. 1.1. Biologically active UV radiation. The overlap between the spectral irradiance F(λ) and the erythemal action spectrum B(λ) given by McKinlay and Diffey (1987) shows the spectrum of biologically active radiation, F(λ) x B(λ). The area under the product function F(λ) x B(λ) is the biologically active dose rate. Thick lines are for a total ozone column of 348 DU, thin lines for 250 DU (one Dobson Unit, or DU, is defined as the height in milli-centimeters that pure gaseous ozone would occupy if compressed to 1013 hPa at 0 °C, and thus equals 2.69×10^{16} molecules cm^{-2}) (from Madronich and Flocke, 1997).

(O_2) and ozone (O_3). Wavelengths in the UV-B range (280-315 nm*) are absorbed efficiently though not completely by O_3, while UV-A wavelengths (315-400 nm) are absorbed only weakly by O_3 and are therefore more easily transmitted to the Earth's surface.

Figure 1.1 illustrates the wavelength dependence of UV spectral irradiance at the Earth's surface. The strong decrease below about 330 nm is due to absorption by atmospheric ozone. Reductions in ozone lead to an increase at these wavelengths, as shown in the figure, with the largest fractional (percentage) increase occurring at progressively shorter wavelengths in the UV-A and UV-B ranges. Although UV-B irradiances are much smaller than those in the UV-A region, many biological responses to UV exposure are far greater at the shorter wavelengths. Thus even relatively small increments of UV-B radiation can lead to substantial biological effects.

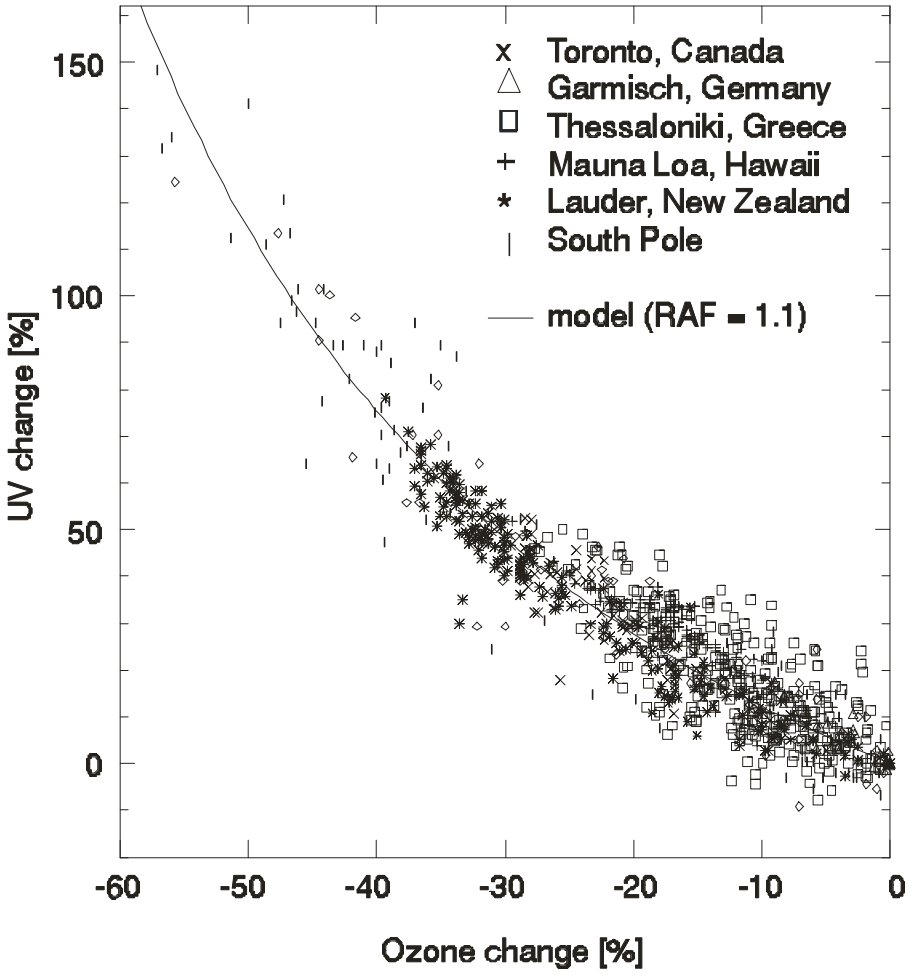

Fig. 1.2. Dependence of erythemal ultraviolet (UV) radiation at the Earth's surface on atmospheric ozone, measured on cloud-free days at various locations, at fixed solar zenith angles. Legend: South Pole (Booth and Madronich, 1994); Mauna Loa, Hawaii (Bodhaine et al., 1997); Lauder, New Zealand (McKenzie et al., 1998); Thessaloniki, Greece (updated from Zerefos et al., 1997); Garmisch, Germany (Mayer et al., 1997); and Toronto, Canada (updated from Fioletov et al., 1997). Solid curve shows model prediction with a power rule using RAF = 1.10.

* Some workers use 320 nm rather than 315 nm (C.I.E. definiton) as the boundary between UV-A and UV-B

To estimate the biological impacts of ozone-related UV increases, the wavelength dependence of the sensitivity to UV exposure must be known at least approximately. Spectral sensitivity functions (action spectra) have been determined in laboratory and field studies for a number of biological endpoints. Such action spectra allow the estimation of the effect of simultaneously changing radiation at different wavelengths by different amounts, as happens when ozone reductions occur. Figure 1.1 shows the action spectrum for erythema (skin-reddening) induction by UV radiation, and the spectral overlap between significant sensitivity (at shorter wavelengths) and significant spectral irradiance (at longer wavelengths). For this particular action, the overlap is greatest in the range 300-320 nm, and is quite sensitive to ozone amounts as shown in Figure 1.1. A useful measure of this overlap is the biologically active UV irradiance, or exposure UV_{bio}, defined as the area under the spectral overlap function,

$$UV_{bio} = \int F(\lambda)\, B(\lambda)\, d\lambda$$

where $F(\lambda)$ is the spectral irradiance, $B(\lambda)$ is the action spectrum for a particular biological effect, and the integral is carried out over all UV wavelengths.

The sensitivity of UV_{bio} to atmospheric ozone is frequently expressed with the Radiation Amplification Factor (RAF), defined as the percentage increase in UV_{bio} that would result from a 1% decrease in the column amount of atmospheric ozone. The radiation amplification factors are given in Table 1.1 for a number of different known effects. The RAFs can generally be used only to estimate effects of small ozone changes, e.g. of a few percent, because the relationship between ozone and UV_{bio} becomes non-linear for larger ozone changes. For action spectra that decrease approximately exponentially with increasing wavelength over 300-330 nm, the biologically active irradiances scale with larger ozone changes according to a power relationship (Madronich, 1993a,b; Booth and Madronich, 1994):

$$UV_{bio} \sim (Ozone)^{-\mathbf{RAF}}$$

The RAFs presented in Table 1.1 have been computed with a model of the propagation of spectral UV radiation through the atmosphere, combined with the appropriate action spectra for the different effects. RAFs may also be derived from spectral UV measurements made at the Earth's surface, when these are combined (numerically) with the appropriate action spectra. Generally good agreement is found between these two methods, within the combined uncertainties of the measurements and the models (McKenzie et al., 1991; Booth and Madronich, 1994; Blumthaler et al., 1995; Bodhaine et al., 1997).

RAFs are useful indicators of the sensitivity of a particular effect (i.e., a particular action spectrum) to ozone changes. Large RAF values indicate that the radiation associated with a particular effect is strongly sensitive to changes in atmospheric ozone, while small RAF values indicate that the relevant UV_{bio} is less sensitive to ozone changes. Values of RAF ~ 0 mean that the UV_{bio} for that particular effect is not dependent on ozone, as occurs in cases when an action spectrum shows strong sensitivity to longer UV-A and visible wavelengths, but not to UV-B radiation.

In many cases the full spectral sensitivity is not well known, and only estimates of the RAF value can be made. A particularly important consideration is the potential role of longer (UV-A and visible) wavelengths, where even relatively low sensitivity (per photon) may be of importance because the ambient radiation increases strongly with increasing wavelength (see curve marked $F(\lambda)$ in Figure 1.1). To show this sensitivity to longer wavelengths, the RAFs given in Table 1.1. were also calcu-

Table 1.1. Radiation Amplification Factors (RAFs) at 30°N.

Effect	RAF		Reference
	January (290 DU)	July (305 DU)	
Erythema*	1.6	1.5	Anders et al., 1995
Skin cancer in SKH-1 hairless mice (Utrecht)	1.5	1.4	de Gruijl et al., 1993
SKH-1 corrected for human skin transmission	1.2	1.1	de Gruijl and van der Leun, 1994
Elastosis	1.1	1.2	Kligman and Sayre, 1991
Photocarcinogenesis, skin edema	1.6	1.5	Cole et al., 1986
Photocarcinogenesis (based on STSL)	1.5	1.4	Kelfkens et al., 1990
Photocarcinogenesis (based on PTR)	1.6	1.5	Kelfkens et al., 1990
Melanogenesis	1.7	1.6	Parrish et al., 1982
Melanoma in fish	0.1	0.1	Setlow et al. 1993
DNA Related			
Generalized DNA damage*	2.2	2.1	Setlow, 1974
Mutagenicity and Fibroblast killing	2.2 [1.7]	2.0 [2.7]	Zölzer and Kiefer, 1984; Peak et al., 1984
Fibroblast killing	0.3	0.6	Keyse et al., 1983
Cyclobutane pyrimidine dimer formation	2.4 [2.0]	2.3 [2.1]	Chan et al., 1986
(6-4) photoproduct formation	2.7 [2.3]	2.5 [2.3]	Chan et al., 1986
HIV-1 activation	4.4 [0.1]	3.3 [0.1]	Stein et al., 1989
Eyes			
Damage to cornea	1.2	1.1	Pitts et al., 1977
Damage to lens (cataract)	0.8	0.7	Pitts et al., 1977
Other effects on animal cells			
Occupational exposure limit	1.4	1.5	ACGIH, 1991
Immune suppression	1.0 [0.4]	0.8 [0.4]	DeFabo and Noonan, 1983
Cell mortality in Chinese hamster	1.3	1.2	Banrud et al., 1993
Substrate binding in Chinese hamster	0.4	0.4	Banrud et al., 1993

Membrane Damage			
Membrane bound K^+-stimulated ATPase inactiv.	2.1 [0.3]	1.6 [0.3]	Imbrie and Murphy, 1982
Plants			
Isoflavonoid formation in bean	2.7 [0.1]	2.3 [0.1]	Wellmann, 1985
Photosynthetic electron transport	0.2	0.1	Jones and Kok, 1966
Overall photosynthesis in leaf of *Rumex patientia*	0.2	0.3	Rundel, 1983
DNA damage in Alfalfa	0.5	0.6	Quaite et al., 1992
Phytoplankton			
Inhibition of motility *(Euglena gracilis)*	1.9	1.5	Häder and Worrest, 1991
Inhibition of photosynthesis *(Phaeodactylum sp.)*	0.2	0.3	Cullen et al., 1992
Inhibition of photosynthesis *(Prorocentrum micans)*	0.3	0.4	Cullen et al., 1992
Inhibition of photosynthesis, in Antarctic community	0.8	0.8	Boucher and Prezelin, 1996
Inhibition of photosynthesis (*Nodularia spumigena*, cyanobacterium)	0.2	0.2	Häder et al., 1994
Tropospheric photolysis			
$O_3 + h\nu \rightarrow O(^1D) + O_2$*	1.7	1.5	de More et al., 1997
$O_3 + h\nu \rightarrow O(^3P) + O_2$	0.1	0.1	de More et al., 1997
$H_2O_2 + h\nu \rightarrow OH + OH$	0.4	0.4	de More et al., 1997
$HNO_3 + h\nu \rightarrow OH + NO_2$	1.1	1.0	de More et al., 1997
$NO_2 + h\nu \rightarrow O(^3P) + NO$	0.0	0.0	de More et al., 1997
$HCHO + h\nu \rightarrow H + CHO$	0.5	0.5	de More et al., 1997
$HCHO + h\nu \rightarrow H_2 + CO$	0.2	0.2	de More et al., 1997
Aqueous photochemistry			
CO production (Suwannee River)	0.3	0.3	Valentine and Zepp, 1993
CO production (Pacific Ocean)*	0.3	0.3	Zafirou, priv. comm., 1998
COS production (Gulf of Mexico)	0.2	0.2	Zepp and Andreae, 1994
COS production (North Sea)	0.6	0.6	Zepp and Andreae, 1994
Photodegradation of nitrate ions	1.1	1.0	Zepp et al., 1987

Photodegradation of HCHO (Biscayne Bay)	1.3	1.1	Kieber et al., 1990
Photoproduction of H_2O_2 in freshwater	0.1	0.1	Cooper et al., 1988
Materials damage			
Yellowness induction in polyvinyl chloride	0.2	0.2	Andrady et al., 1989
Yellowness induction in polycarbonate	0.4	0.4	Andrady et al., 1991
Other weighting functions			
Temple U. Robertson-Berger meter	0.8	0.7	Urbach et al., 1974
Solar Light Robertson-Berger meter (Model 501)	1.2	1.1	Manufacturer's literature, 1994
Ozone cross section (273 K)	0.8	0.8	de More et al., 1997
UV-A (315-400 nm)	0.03	0.02	
UV-B (280-315 nm)	1.25	0.99	
UV-B' (280-320 nm)	0.87	0.71	
Exponential decay, one decade per 14 nm	1.00	1.00	

Updated from UNEP (1994), with new entries denoted by (*). RAFs are computed on basis of daily integrals. Listed references are for the original action spectra used in the calculation of the RAFs. Measurements of action spectra often do not cover the full UV-A wavelength range. For such cases, the RAF calculations were repeated by extrapolating the action spectra to 400 nm; values shown in brackets show the RAF estimated from the extrapolated action spectra, for cases where the RAF changed by at least 0.2 units, and are indicative of the highly uncertain UV-A contribution.

lated by extrapolating the measured action spectra to 400 nm, and, for cases where such extrapolation lead to significant changes in the RAF, the re-calculated values are shown in brackets. The RAFs calculated from extrapolated action spectra are not necessarily more accurate than those without extrapolation, but rather show that such RAFs are quite uncertain, and more detailed measurements of the action spectra are need to assess the sensitivity to ozone.

It should be cautioned however that (i) neither the action spectra nor the resulting weighted irradiances (UV_{bio}) give a measure of the absolute damage to any particular organism; (ii) weighted irradiances computed from different action spectra cannot be compared directly to one another, because the action spectra usually specify only the spectral shape of the sensitivity, not the absolute value; (iii) even for a single action spectrum, increases in UV_{bio} do not necessarily imply a proportionate increase in effect, if dose-response relations for that effect are non-linear; (iv) any damage to a specific organism must be viewed in the context of its entire ecosystem including consideration of other stresses (e.g. nutrient availability, temperature) and interactions with other organisms (e.g. species competition); (v) action spectra are usually determined from short-term laboratory or field experiments, while

the effects of environmental UV increases may be felt on longer time scales; and (vi) considerable uncertainties are inherent in the experimental determinations of action spectra.

Measurements of UV Radiation

The last decade has seen a great increase in the number and general quality of solar UV measurements. Many new commercial and research-grade UV detectors have been developed, calibration procedures have been improved, and several national and international intercomparisons have been carried out (e.g. McKenzie et al., 1993; Gardiner et al., 1993; Seckmayer et al., 1994; Thompson et al., 1997; Kjeldstad et al., 1997; Webb, 1997; Leszczynski et al. 1998). Agreement among similarly calibrated spectro-radiometers is typically 5% or better in the UV-A range, and 5-10% in the UV-B range. Comparisons between different types of instruments (e.g. spectro-radiometers, broad band meters, filter radiometers) are more difficult, because of the need to put the different measured quantities on a similar basis, for example through the use of model interpolations (e.g. Slaper et al., 1995; Bernhard et al., 1996; Mayer and Seckmeyer, 1996; Booth, 1997).

Direct measurements of surface UV radiation confirm to a large extent the theoretical expectations, if allowances are made for local conditions (e.g. Booth and Madronich, 1994; Forster et al., 1995; Geogdzhaev et al., 1996; Gardiner and Martin, 1997; Mayer et al., 1997; Pachart et al., 1997; Pfister et al., 1997; Weihs and Webb, 1997). However the analysis, interpretation, and utilization of the measurements still lag behind the growing data archives. Some general patterns of temporal and geographical variations are also being identified (e.g. Seckmeyer et al., 1995; Bais et al., 1997; Bodhaine et al., 1997; Lu and Li, 1997; Orce et al., 1997; Qu et al., 1997; Sasaki et al., 1997; Zerefos et al., 1997; Bigelow et al. 1998). For example, ground-based measurements show that summertime erythemal UV irradiances in the Southern Hemisphere exceed those at comparable latitudes of the Northern Hemisphere by up to 40% (Seckmeyer et al., 1995), whereas corresponding satellite-based estimates yield only 10 to 15% differences (WMO, 1998). Atmospheric pollution may be a factor in this discrepancy between ground-based measurements and satellite-derived estimates. UV-B measurements at more sites are required to determine whether the larger observed differences are globally representative.

Ozone-Related UV Radiation Changes

The evidence is overwhelming that under cloud-free skies UV-B radiation is controlled largely by ozone (Bais et al., 1993; Kerr and McElroy, 1993; Booth and Madronich, 1994; Diaz et al., 1994; Frederick et al., 1994; Bojkov et al., 1995; Huber et al., 1995; Mims et al., 1995; Varatsos and Kondratyev, 1995; Björn and Holmgren, 1996; Hofmann et al., 1996; Bernhard et al., 1997; Bodhaine et al., 1997; Chubarova and Nezval, 1997; Kirchoff et al., 1997; Koskela et al. 1997; Mayer et al., 1997; Seckmeyer et al., 1997; Taalas et al., 1997; and references therein). The response of UV-B radiation to ozone changes is strongly dependent on wavelength because of the rapid increase of the ozone absorption cross section toward shorter wavelengths, with greater sensitivity at short wavelengths and low sun, where the slant ozone optical depth is greater [see for example the review by Madronich et al. (1995), the more recent measurements by Fioletov and Evans (1997), and references therein]. For biologically weighted radiation, measurements under cloud-free skies also show the theoretically expected dependence on ozone. Figure 1.2 shows the sensitivity of erythemal (skin-reddening) UV radiation to the ozone column amount, as measured at a number of different locations and for different solar zenith angles. When expressed on a relative (percentage) basis, the increases in erythemal UV

radiation are seen to correlate closely with ozone reductions, whether the latter stem from natural fluctuations and seasonal cycles, or from systematic long-term depletion. The largest percent UV increases are associated with the largest percent reductions in atmospheric ozone.

Current losses of stratospheric ozone are discussed in the Scientific Assessment of Ozone – 1998 (WMO, 1998). Relative to the values in the 1970s, these are estimated to be about 50% in the Antarctic spring (the ozone hole), about 15% in the Arctic spring, about 6% at Northern Hemisphere mid latitudes in winter/spring, about 3% at Northern Hemisphere mid latitudes in summer/fall, and about 5% at Southern Hemisphere mid latitudes year-round. The corresponding increases in erythemal UV radiation are estimated to be 130%, 22%, 7%, 4%, and 6%, respectively. No significant ozone trend has been found in equatorial regions. The geographical extent and severity of the Antarctic ozone hole have remained essentially unchanged since the early 1990s. Relatively little change in the mid latitude ozone losses has been observed in the last half-decade.

High levels of UV-B radiation have been observed directly in association with the Antarctic ozone hole (Frederick and Alberts, 1991; Booth et al., 1994; Seckmeyer et al., 1995, Frederick et al., 1998), and on occasion the measured DNA-damaging radiation at Palmer Station, Antarctica (64°S) has been found to exceed maximum summer values at San Diego, USA (32°N) (Booth et al., 1994). It should be noted that monitoring of UV-B irradiances in Antarctica began only in 1989, well after the appearance of the ozone hole, so that the UV-B levels in pre-ozone hole years can be only estimated.

The smaller increases of UV-B radiation at mid latitudes, while expected, have not yet been detected unambiguously. The record of mid-latitude UV-B measurements is not sufficient for the derivation of statistically significant trends. Little or no reliable historical information on the climatology of UV radiation is available from pre-ozone depletion days (e.g., pre-1980). The few available long-term UV measurement records have been hampered by the difficulty in maintaining stability of UV-measuring outdoor instruments over periods of decades, and by changes in atmospheric turbidity associated with local pollution. For example, measurements obtained with Robertson-Berger meters over 1974-85 suggested a decrease in UV radiation at 14 US locations (Scotto et al., 1988); however a recent re-analysis of these data has identified calibration shifts which, when removed, indicate that no significant trend can be derived from the data record (Weatherhead et al., 1997). Furthermore, increases in UV due to stratospheric ozone reductions may have been masked in some urban areas experiencing increasing levels of local air pollution (e.g. Garadzha and Nezval, 1987). Pronounced ozone losses have occurred for shorter periods of time, e.g. in the few years after the 1991 eruption of Mt. Pinatubo (Gleason et al., 1993) and over the Arctic during six of the past nine winters (Müller et al., 1997; Rex et al., 1997; Stolarski, 1997), with correspondingly higher measured UV-B radiation levels (e.g. Kerr and McElroy, 1993; Fioletov and Evans, 1997).

Tropospheric ozone is also an effective absorber of UV-B radiation (Brühl and Crutzen, 1989). In urban and industrialized regions, tropospheric ozone is formed by the photo-chemical reactions of some pollutants (nitrogen oxides and hydrocarbons), while in remote regions it stems from both downward transport from the stratosphere, and from in-situ photochemical production by both natural and anthropogenic precursor compounds transported from source regions (WMO, 1994a). Model-based estimates suggest that for industrialized regions of the Northern Hemisphere, the increases in tropospheric ozone since pre-industrial times may have reduced DNA-damaging UV radiation by 3-15% (Brühl and Crutzen, 1989; Madronich et al., 1991; Frederick et al., 1993; Blumthaler et al., 1997; Ma and Guicherit, 1997). Comparisons between spectral UV measurements in Germany and New Zealand also suggest that the lower UV radiation levels observed in Germany may be explained partly by higher tropospheric ozone levels (Seckmeyer and McKenzie, 1992). Recent changes in tropospheric ozone are estimated to be much smaller those since pre-industrial days, with both positive and

negative trends reported for different geographic locations (WMO, 1994a, 1998). Their contributions to the trend in the total ozone column are much smaller than those from changes in stratospheric ozone over the same time period (e.g., 1980 to present). Other gases such as sulfur dioxide (SO_2) and nitrogen dioxide (NO_2) can also reduce atmospheric UV transmission, however significant effects are limited to some extremely polluted urban environments.

Cloud-Related UV Radiation Changes

Clouds generally reduce surface UV irradiances, although the magnitude of this effect is highly variable depending on cloud amount and coverage, cloud cell morphology, particle size distributions and phase (water droplets and ice crystals), and possible in-cloud absorbers (esp. tropospheric ozone). It is useful to note that under some conditions, UV irradiances can be higher than for clear sky, as for example when both direct sunlight and light scattered by clouds (e.g. the sides of bright broken clouds) reaches the observer (Nack and Green, 1974; Mims and Frederick, 1994).

Numerous statistical correlations between UV transmission and cloud cover have been carried out (e.g. Paltridge and Barton, 1978; Cutchis, 1980; Josefsson, 1986; Ilyas, 1987; Ito, 1993; Björn and Holmgren, 1996; Estupinian et al., 1996; Schafer et al., 1996; Gillotay et al., 1997), but because of the high spatial and temporal variability of clouds, no single value can be given for their effects on surface UV levels. For example, analysis of the Robertson-Berger meter data record shows that monthly average UV levels are reduced by 10-50 percent, depending on season and location in the US (Frederick et al., 1989, 1993). An important aspect of clouds is that, by introducing strong variability in the UV intensities reaching the Earth's surface, they complicate the detection of long-term trends (Frederick and Erlick, 1995; Lubin and Jensen, 1995; Nunez et al., 1997).

Cloud transmission depends somewhat on wavelength. In the UV-A region, transmission increases slightly toward shorter wavelengths due to increased multiple reflections between cloud and the surrounding air molecules (Nack and Green 1974; Seckmeyer et al., 1996; Chubarova et al., 1997). At shorter wavelengths, in the UV-B range, long photon path lengths in clouds can increase absorption by tropospheric ozone, resulting in a sharp decrease in effective transmission (Frederick and Lubin, 1988; Mayer et al., 1998).

Aerosol-Related UV Radiation Changes

Small particles suspended in air (aerosols) can have a significant effect on the transmission of UV-B radiation to the surface. The magnitude of the effect is highly variable, depending on the number of particles and their physical and chemical make-up (e.g. sulfate haze, soot, dust, sea-salt aerosols). Such particles are frequently found in the lowest part of the troposphere (the boundary layer), and are often associated with pollution.

Liu et al. (1991) estimated that anthropogenic sulfate aerosols (associated primarily with fossil fuel combustion) have decreased surface UV-B irradiances by 5-18% in industrialized regions of the Northern Hemisphere. Additional evidence for the role of aerosols comes from simultaneous monitoring of UV irradiances and atmospheric turbidity in relatively polluted environments (Garadzha and Nezval, 1987; Varotsos et al., 1995; Estupinian et al., 1996; Mims, 1997), from differences between locations in the Northern (more polluted) and Southern (less polluted) hemispheres (Seckmeyer and McKenzie, 1992; Seckmeyer et al., 1995), and from the increases in UV irradiances with increasing surface elevation, in excess of those expected from pollution-free conditions (Cabrera et al., 1995; Madronich et al., 1995; Piazena 1996; Blumthaler et al., 1997). The measured effects on UV radiation are highly variable and specific to the various locations (e.g.,Wenny et al., 1998).

An important consideration is whether the aerosol particles are highly absorbing (e.g. soot) or simply scatter (re-direct) the incident radiation (e.g. sulfate aerosols). All particles tend to reduce the UV irradiance (defined as the radiation incident on a horizontal surface). However, scattering by non-absorbing aerosols can actually increase the UV exposure on non-horizontal surfaces due to the additional radiation incident from low angles (e.g. Blumthaler et al. 1997; Dickerson et al., 1997; Loxsom and Kunkel, 1997). The net effects on biota from such changes in direction of incidence are not well understood.

Stratospheric aerosols are usually too sparse to have any effect on atmospheric UV transmission. An exception arises following a major volcanic eruption, such as that of Mt. Pinatubo (Philippines) in June 1991 which injected large amounts of ash and sulfur dioxide (SO_2) into the stratosphere. The heavier ash sedimented out of the stratosphere relatively quickly and its optical effects were of limited geographical extent. Gaseous SO_2, on the other hand, was removed from the stratosphere mainly by chemical reactions to form H_2SO_4 molecules, which then readily nucleated into sulfate aerosol particles. Higher stratospheric sulfate aerosol loadings were observed for several years after the eruption, during which time these particles were distributed on global scales. Calculations indicate that the effects on biologically weighted UV irradiances were quite small, of order of a few percent (Madronich et al., 1991; Vogelmann et al., 1992; Tsitas and Yung, 1996), with even some possible enhancements at very short wavelengths and low sun when aerosols scatter some photons directly downward thus allowing a shorter crossing of the stratospheric ozone layer (Michelangeli et al., 1992; Davies, 1993). Ground-based measurements of UV irradiance after the Mt. Pinatubo eruption confirm the small decreases and also show a strong increase in diffuse/direct radiation at all wavelengths, in good agreement with theoretical models (Blumthaler and Ambach, 1994; Zeng et al., 1994, Lantz et al., 1996). A less direct but more important UV-related consequence of stratospheric aerosols is their effect on stratospheric ozone itself. Significant destruction of stratospheric ozone by heterogeneous chemical processes involving the aerosols was predicted (Hoffman and Solomon, 1989; Brasseur et al., 1990) and observed for several years after the Mt. Pinatubo eruption (Gleason et al., 1993; WMO, 1994a).

Model-Derived Surface UV Radiation

In view of the high spatial and temporal variability of surface UV radiation, and the difficulty of maintaining calibration within networks of instruments, it is unlikely that either a satisfactory global UV climatology or representative long-term UV trends can be derived from ground-based monitoring stations alone. Satellite-based observations of the atmosphere, on the other hand, provide the spatial (global or nearly global) coverage required for climatology development, as well as nearly continuous long-term monitoring. For example, the development of a climatology of UV radiation incident on the oceans will necessarily be based on such satellite-derived estimates. However, the derivation of surface UV irradiance from satellite-based observations is indirect, because satellite instruments see radiation reflected by the atmosphere and surface of the Earth. The determination of radiation transmitted to the surface requires the use of radiative transfer models to relate transmission, reflection, and atmospheric absorption.

Figure 1.3 shows the changes in UV radiation (at 310 nm) reaching the surface, computed for clear skies using satellite-based ozone measurements between 1979 and 1993. As expected from ozone trends (WMO, 1998), UV trends are not significant in the tropics, but increase pole-ward in both hemispheres. The largest changes (percentage and absolute) are seen in the Southern Hemisphere polar regions, but significant inter-annual and shorter variability should be noted at all latitudes, even after considering monthly and zonal averages. Patterns of long-term changes differ also between

hemispheres, e.g. with largest changes occurring in the early 1980s at southern mid-latitudes, while northern mid-latitudes show a more persistent long-term trend.

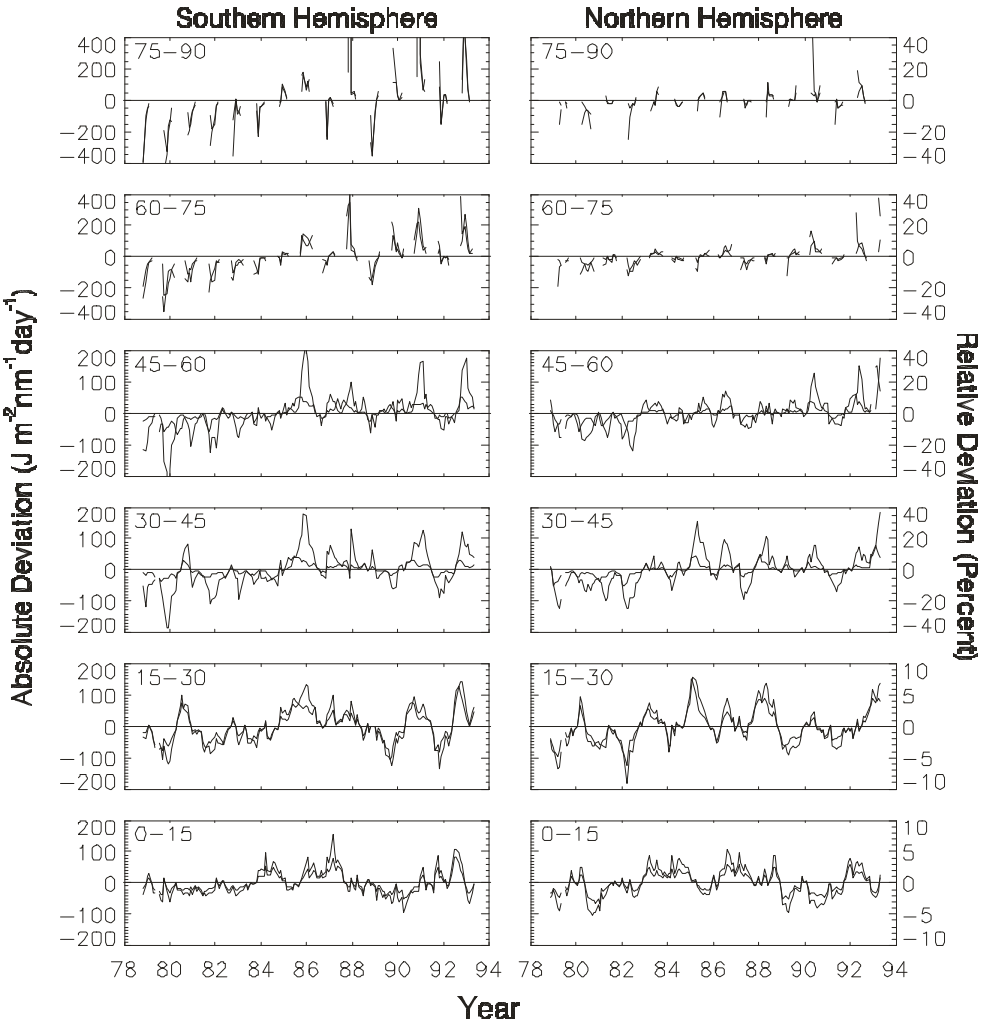

Figure 1.3: Changes in daily surface spectral irradiances at 310 nm, computed for cloud-free conditions from satellite-based ozone observations (TOMS, version 7, McPeters et al., 1996, monthly averages over different latitude bands indicated in each panel). Values given are deviations from the 1979-1993 means. Solid curves give absolute deviations (left scale), while dotted curves show percent changes (right scale). Note change of scales for different latitude bands.

Significant progress has been made in recent years, in utilizing satellite-based measurements of cloud cover as well as atmospheric ozone, to derive estimates of surface UV radiation levels (Eck et al., 1995; Herman et al., 1996; Meerkoetter et al., 1997). Recent work also suggests that it may be possible to derive tropospheric aerosol distributions from satellite-based observations (Herman et al., 1997; Krotkov et al., 1997; Hsu et al., 1997). Figure 1.4 shows the type of coverage and geographical detail

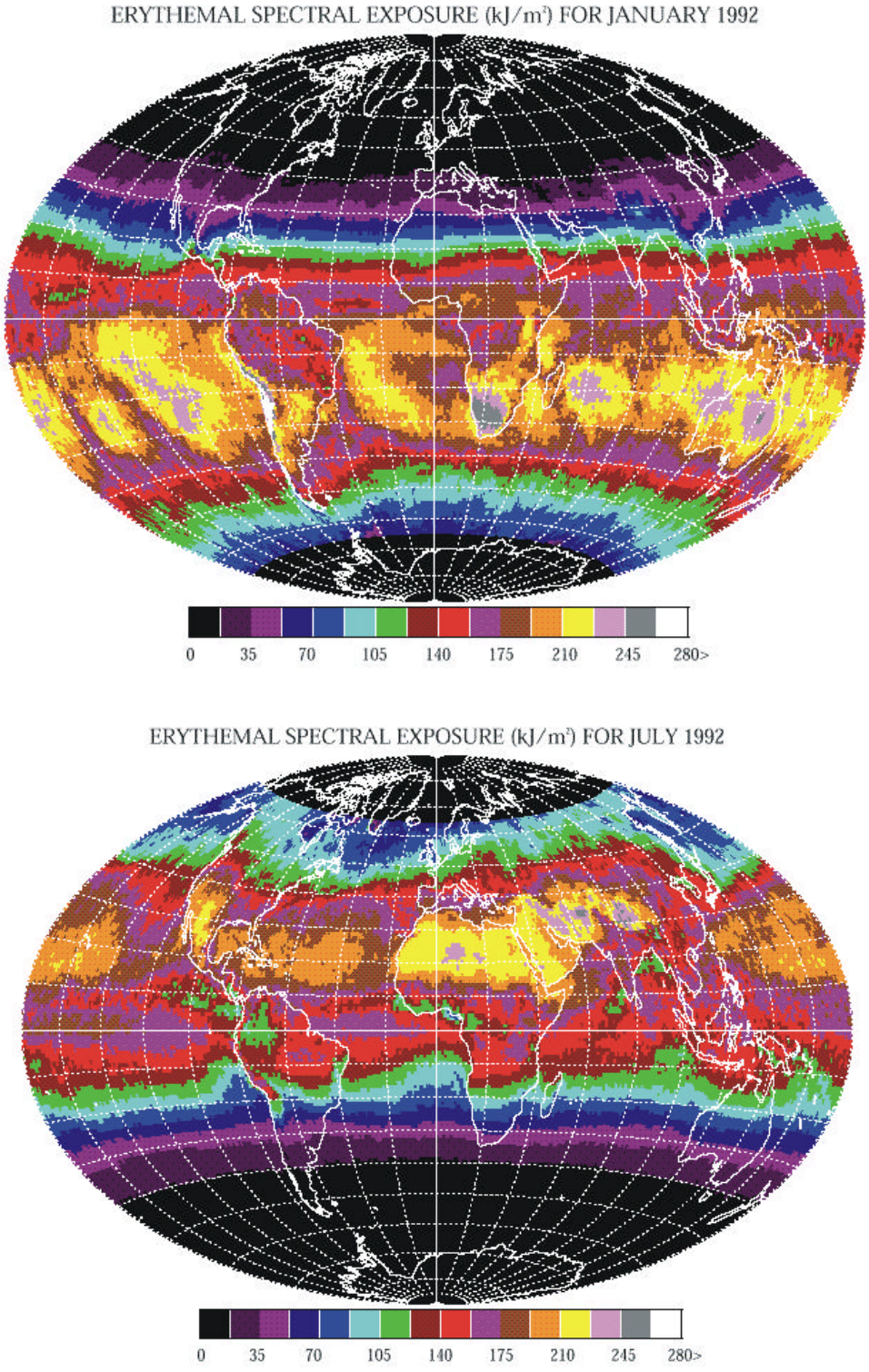

Fig. 1.4. Satellite-derived erythemal spectral irradiance at the Earth's surface. (a) For January 1992, (b) for July 1992. From Herman et al. (1998).

Table 1.2. Trends in biologically active radiation (weighted with the erythemal action spectrum of McKinlay and Diffey, 1987), derived from total ozone and cloud reflectivity measurements from the Total Ozone Mapping Spectrometer (TOMS, version 7) over 1979-1992. Adapted from Herman et al. (1996).

Latitude band	**Trend[(a)]**	**Uncertainty[(b)]**
degrees	**% decade^{-1}**	**± 2 sigma**
60°S – 70°S	4.5	5.5
60°S – 50°S	5.5	4.5
50°S – 40°S	3.5	3.5
40°S – 30°S	1.5	2.5
30°S – 20°S	2.0	2.5
20°S – 10°S	1.5	2.5
10°S – 0°	1.5	3.0
0° – 10°N	1.0	3.5
10°N – 20°N	0.0	2.5
20°N – 30°N	1.0	3.0
30°N – 40°N	3.0	3.0
40°N – 50°N	3.5	3.0
50°N – 60°N	5.0	4.0
60°N – 70°N	4.0	4.0

a) Zonally averaged trend over given latitude band, values rounded to nearest half percent.

b) As corrected by Herman et al. (1998) and includes combined instrumental error and variability of UV radiances.

Krotkov et al., 1997; Hsu et al., 1997). Figure 1.4 shows the type of coverage and geographical detail currently possible with the satellite-based approach. Long-term trends in cloud cover have partly offset or augmented UV trends resulting from ozone changes in some regions, but have been shown by Herman et al. (1996) to have little effect on long term changes when averaged over large geographical scales (zonal means). This type of analysis represents a considerable improvement over earlier analyses of satellite data that considered only ozone changes, with no consideration of clouds (e.g., Madronich, 1992). Table 1.2 shows the trends in surface UV radiation (erythemal weighting) over 1979-1992, derived from measurements of ozone and cloud reflectivity by the Total Ozone Mapping Spectrometer (TOMS, version 7) aboard the Nimbus 7 satellite. Positive trends are statistically significant at the two-standard deviation level over much of the mid-latitudes of both hemispheres. Extension to more recent years is complicated by the use of different instruments aboard different satellites, and analysis is still underway (WMO, 1998).

The limitations of these satellite-derived surface UV estimates should be recognized. The ozone and cloud determinations at any specific location are based on a single satellite overpass per day, and are estimated for other times by interpolation or, more simply, by assuming constancy over the spe-

cific day. Therefore it is essential that comparisons be made between ground-based UV monitoring and the satellite-derived UV levels, in order to have a more complete assessment of the uncertainties inherent in this method. Preliminary results of such comparisons are encouraging (e.g. Eck et al., 1995) but more ground-based validation is needed over longer periods of time and different geographical locations. Even so, comparisons to ground-based UV observations will not be able to account fully for some location-specific biases. For example, optical instruments borne by satellites have difficulty seeing the lower atmosphere (esp. in the presence of clouds) so local conditions (e.g. pollution) are not sampled accurately. Additional local factors, such as surface reflections and elevation gradients, are also problematic. Other promising approaches combine, as above, satellite data with radiative transfer calculations, but also include some ground-based observations by other instruments such as visible and total solar radiation detectors which are more accurate and much more widely deployed than UV detectors (Ito et al. 1994; Bodeker and McKenzie, 1996).

Future UV Radiation Levels

The prediction of future UV radiation levels must be considered according to the time scales of interest. On short time scales, of order of a few days or a week, UV radiation forecasts incur all of the difficulties of forecasting weather (especially clouds); of estimating atmospheric profiles of ozone and other gases and particles, some anthropogenic; and of accounting for a variety of possible other local factors including surfaces (elevation, orientation, reflectivity). These factors make accurate UV forecasts impractical beyond a few days. Next-day forecasts based on meteorological analyses are now being made with some success in a number of countries. In most cases, the results are disseminated to the public, with UV radiation levels expressed as a dimensionless UV index. International standardization was reached (WMO, 1994b; ICNIRP, 1995) on the method of calculation of the index, which is defined as the UV irradiance, in units of W m^{-2}, weighted by the erythemal action spectrum of McKinlay and Diffey (1987), then multiplied by 40. Using this scale, a UV index of 10 or more may be considered "extreme".

Long-term UV predictions (years, decades, or longer) are exceedingly difficult and uncertain, and therefore only appropriate in a statistical sense of averages, variabilities, and broad geographical patterns. Even then, many assumptions must be made not only about the future state of the ozone layer, but also about possible long-term changes in clouds, tropospheric pollutants, and changes in surface albedo. In considering future biological effects of UV changes, it is also necessary to allow for uncertain long-term changes in ecosystem size and composition and - specifically for humans - changes in behavior, migration and demographics.

Predictions of future ozone amounts are in themselves also very difficult. Natural perturbations such as major volcanic eruptions are unpredictable, though their importance to stratospheric ozone was clearly demonstrated in the aftermath of the 1991 Mt. Pinatubo eruption. Large uncertainties exist concerning the interactions of stratospheric chemistry with expanding human activities, e.g. the increasing emissions of so-called greenhouse gases and the associated changes in global climate, the effluents from growing fleets of subsonic and supersonic aircraft, and the changes in tropospheric air quality and self-cleaning (oxidizing) capacity. Their interactions with stratospheric ozone are current subjects of active research and are still not well quantified (WMO, 1998). A recent study, for example, suggests that the recovery of the ozone layer may be delayed significantly by interactions with increasing green-house gas concentrations (Schindell et al., 1998).

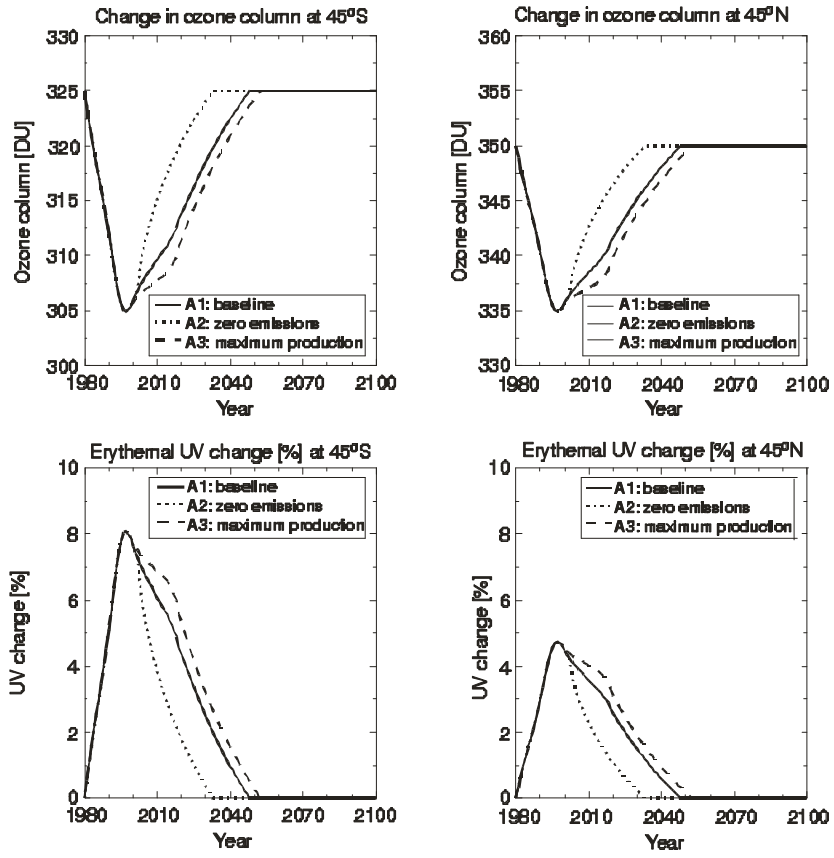

Fig. 1.5. Scenario for future changes in ozone and erythemally-weighted UV radiation at the Earth's surface, at 45° N and 45° S. UV radiation changes are estimated from ozone changes, which in turn are estimated from changes in atmospheric amounts of ozone-destroying substances (halocarbons). All other factors are assumed constant. Future scenarios shown are based on current control measures (Montreal 1997 Amendments), with scenario A1 (baseline, solid curves) accounting for the fact that production of some ozone-depleting substances is currently already below the allowed maximum, while in scenario A3 (dashed curves) production is at the maximum allowed level. Dotted curves are the zero-emission limit (starting in the year 2000) and only illustrate the minimum delay time imposed by atmospheric processes (from Madronich et al., 1998).

With a clear understanding of these uncertainties, it is nevertheless of interest to examine the implications of current international regulations to the future of the ozone layer, and consequently to the future of UV radiation. The 1987 Montreal Protocol and its subsequent adjustments and amendments limit the production and emission of ozone-destroying substances, primarily halocarbons. The atmospheric concentrations of these chemicals had been increasing throughout the 1970s and 1980s, but observations in the last few years (e.g. Montzka et al., 1996) show a marked slowing of growth and even decreases in many of these compounds as a result of implementation of the Protocol (WMO, 1998). Figure 1.5 shows the temporal change of ozone and surface UV radiation (at 45°N and 45°S) computed in correspondence to the halocarbon loading of the atmosphere. This calculation assumes that changes in UV radiation are due solely to ozone changes, which in turn are assumed to respond only to atmospheric halocarbon loading. The quantitative relation between ozone and halocarbon changes is based on the measured changes in both quantities through the 1980s (Daniel et al., 1996). The future scenarios shown in the figure are based on current control measures (Montreal 1997 Amendments), with scenario A1 accounting for the fact that production of some ozone-depleting substances

is currently already below the allowed maximum, while under scenario A3 production is at the maximum allowed level. In either case the UV radiation is expected to return to normal (pre-1980) levels by the middle of the next century. Scenario A2 shows the ozone/UV recovery if there is no emission after the year 2000; while this scenario is obviously unrealistic, it illustrates the natural time scale for the removal of the halocarbons already present in the atmosphere, and is therefore a fundamental limit to the rate of recovery.

Given the numerous uncertainties listed above, it is unlikely that future UV radiation changes will follow precisely any scenario presented in Figure 1.5. Two features of this figure are nonetheless noteworthy. First, the return to pre-ozone depletion levels will take several decades even under the most optimistic scenarios of compliance with international regulations of ozone-depleting substances. Second, and perhaps most important, is to note that in the present half-decade (1995-2000) ozone reductions are the largest since ozone observations began. The observed slowing and even turnover of the rate of growth of some atmospheric halocarbons is highly significant, but large uncertainties, stemming from both future human activities and the imperfect understanding of the complexity of the atmosphere, leave open the question of the extent and timing of the return to natural levels of stratospheric ozone and surface UV radiation.

References

ACGIH (American Conference of Governmental and Industrial Hygienists) 1992. *1991-1992 Threshold Limit Values*.

Anders, A., Altheide, H.-J., Knälmann, M. and Tronnier, H. 1995. Action spectrum for erythema in humans investigated with dye lasers. *Photochemistry and Photobiology* 61:200-205.

Andrady, A.L., Torikai, A. and Fueki, K. 1989. Photodegradation of rigid PVC formulations. III. Wavelength sensitivity to light-induce yellowing by monochromatic light. *Journal of Applied Polymer Science* 37:935-946.

Andrady, A.L., Fueki, K. and Torikai, A. 1991. Spectral sensitivity of polycarbonates to light-induced yellowing. *Journal of Applied Polymer Science* 42:2105-2107.

Bais, A.F., Zerefos, C.S., Meleti, C., Ziomas, I.C., Tourpali, K., Karaouza, V. and Balis, D. 1993. Variability of solar UV-B radiation at high and middle latitudes during EASOE 1991/92. *Journal of Geophysical Research* 98:5199-5204.

Bais, A.F., Blumthaler, M., Webb, A.R., Groeber, J., Kirsch, P.J., Gardiner, B.G., Zerefos, C.S., Svenoe, T. and Martin, T.J. 1997. Spectral UV measurements over Europe within the SESAME activities. *Journal of Geophysical Research* 102:8731-8736.

Banrud, H., Berg, K., Platou T. and Moan, J. 1993. An action spectrum for UV-induced attachment of V79 Chinese hamster cells to a substratum. *International Journal of Radiation Biology* 64:437-444.

Bernhard, G., Mayer, B. and Seckmeyer, G. 1996. All weather comparisons between spectral and broadband (Robertson-Berger) UV meters. *Photochemistry and Photobiology* 64:792-799.

Bernhard, G., Mayer, B. and Seckmeyer, G. 1997. Measurements of spectral solar UV irradiance in tropical Australia. *Journal of Geophysical Research* 102:8719-8730.

Bigelow, D.S., Slusser, J.R., Beaubien, A.F. and Gibson, J.H. 1998. The USDA Ultraviolet Radiation Monitoring Program. *Bulletin of the American Meteorological Society*, in press.

Björn, L.O. and Holmgren, B. 1996. Monitoring and modelling of the radiation climate at Abisko. *Ecological Bulletin* 45:204-209.

Blumthaler, M. and Ambach, W. 1994. Changes in solar radiation fluxes after the Pinatubo eruption. *Tellus* 46B:76-78.

Blumthaler, M., Salzgeber, M. and Ambach, W. 1995. Ozone and ultraviolet-B irradiances: experimental determination of the radiation amplification factor. *Photochemistry and Photobiology* 61:159-162.

Blumthaler, M., Ambach, W. and Ellinger, R. 1997. Increases in solar UV radiation with altitude. *Journal of Photochemistry and Photobiology B:Biology* 39:130-134.

Blumthaler, M., Schreder, J. and Gröbner, J. 1997. UV sky radiance influenced by aerosols and tropospheric ozone – measurements and modelling. In *IRS'96: Current Problems in Atmospheric Radiation*, Smith, W.L. and Stamnes, K. (eds.), Proceedings of the International Radiation Symposium, Fairbanks, Alaska 19-24 August 1996, Deepak Publishing, Hampton Virginia, pp. 853-856.

Bodeker, G.E. and McKenzie, R.L. 1996. An algorithm for inferring surface UV irradiance including cloud effects. *Journal of Applied Meteorology* 35:1860-1877.

Bodhaine, B.A., Dutton, E.G., McKenzie, R.L. and Johnston, P.V. 1997. Spectral UV measurements at Mauna Loa: July 1995-July 1996. *Journal of Geophysical Research* 23:2121-2124.

Bojkov, R.D., Fioletov, V.E. and Diaz, S.B. 1995. The relationship between solar UV irradiance and total ozone from observations over southern Argentina. *Geophysical Research Letters* 22:1249-1252.

Booth, C.R., Lucas, T., Morrow, J.H., Weiler, C.S. and Penhale, P.A. 1994. The United States National Science Foundation's polar network for monitoring ultraviolet radiation. In *Ultraviolet Radiation in Antarctica: Measurements and Biological Effects*, Weiler, C.S. and Penhale, P.A. (eds.), AGU Antarctic Research Series, volume 62, American Geophysical Union, Washington, DC, pp. 17-37.

Booth, C.R. and Madronich, S. 1994. Radiation amplification factors - improved formulation accounts for large increases in ultraviolet radiation associated with Antarctic ozone depletion. In *Ultraviolet Radiation in Antarctica: Measurements and Biological Effects*, Weiler, C.S. and Penhale, P.A. (eds.), AGU Antarctic Research Series, volume 62, American Geophysical Union, Washington, DC, pp. 39-42.

Booth, C.R. 1997. Synthetic UV spectroradiometry. In Smith, W.L. and Stamnes, K. (eds.), *IRS'96: Current Problems in Atmospheric Radiation*. Proceedings of the International Radiation Symposium, Fairbanks, Alaska 19-24 August 1996, Deepak Publishing, Hampton Virginia, pp. 849-852.

Boucher, N.P. and Prézelin, B.B. 1996. Biological weighting functions for the UV inhibition of carbon fixation in a natural Antarctic phytoplankton community. *Marine Ecology Progress Series* 144:233-236.

Brasseur, G.P., Granier, C. and Walters, S. 1990. Future changes in stratospheric ozone and the role of heterogeneous chemistry. *Nature* 348:626-628.

Brühl, C. and Crutzen, P.J. 1989. On the disproportionate role of tropospheric ozone as a filter against solar UV-B radiation. *Geophysical Research Letters* 16:703-706.

Cabrera, S., Bozzo, S. and Fuenzalida, H. 1995. Variations in UV radiation in Chile. *Journal of Photochemistry and Photobiology* 28:137-142.

Chan, G.L., Peak, M.J., Peak, J.G. and Haseltine, W.A. 1986. Action spectrum for the formation of endonuclease-sensitive sites and (6-4) photoproducts induced in a DNA fragment by ultraviolet radiation. *International Journal of Radiation Biology* 50:641-648.

Chubarova, N.Y. and Nezval, Y.I. 1997. Ozone, aerosol and cloudiness impacts on biologically effective radiation and UV radiation less 380 nm. In *IRS'96: Current Problems in Atmospheric Radiation*, Smith, W.L. and Stamnes, K. (eds.), Proceedings of the International Radiation Symposium, Fairbanks, Alaska 19-24 August 1996, Deepak Publishing, Hampton Virginia, pp. 886-889.

Chubarova, N.Y., Krotkov, N.A., Geogdzhaev, I.V., Kondranin, T.V. and Khattatov, V.U. 1997. Spectral irradiance: The effects of ozone, cloudiness and surface albedo. In *IRS'96: Current Problems in Atmospheric*

Radiation, Smith, W.L. and Stamnes, K. (eds.), Proceedings of the International Radiation Symposium, Fairbanks, Alaska 19-24 August 1996, Deepak Publishing, Hampton Virginia, pp. 881-885.

Cole, C.A., Forbes, D. and Davies, R.E. 1986. An action spectrum for UV photocarcinogenesis. *Photochemistry and Photobiology* 43:275-284.

Cooper, W.J., Zika, R.C., Patasna, R.G. and Plane, J.M. 1988. Photochemical formation of hydrogen peroxide in natural waters. *Environmental Science and Technology* 22:1156-1160.

Cullen, J.J., Neale, P.J. and Lesser, M.P. 1992. Biological weighting function for the inhibition of phytoplankton photosynthesis by ultraviolet radiation. *Science* 258:646-649.

Cutchis, P. 1980. *A Formula for Comparing Annual Damaging Ultraviolet (DUV) Radiation Doses at Tropical and Mid-Latitude Sites*. Federal Aviation Administration Report-FAA-EE 80-21, U. S. Department of Transportation, Washington, DC.

Daniel, J.S., Solomon, S. and Albritton, D.L. 1995. On the evaluation of halocarbon radiative forcing and global warming potentials. *Journal of Geophysical Research* 100:1271-1285.

DeFabo, E.C. and Noonan, F.P. 1983. Mechanism of immune suppression by ultraviolet radiation in vivo. I. Evidence for the existence of a unique photoreceptor in skin and its role in photoimmunology. *Journal of Experimental Medicine* 158:84-98.

de Gruijl, F.R. and van der Leun, J.C. 1994. Estimate of the wavelength dependency of ultraviolet carcinogenesis and its relevance to the risk assessment of a stratospheric ozone depletion. *Health Physics* 4:317-323.

de Gruijl, F.R., Sterenborg, J.J.C.M., Forbes, P.D., Davies, R.E., Cole, C., Kelfkens, G., van Weelden, H., Slaper, H. and van der Leun, J.C. 1993. Wavelength dependence of skin cancer induction by ultraviolet irradiation of albino hairless mice. *Cancer Research* 53:53-60.

de More, W.P., Sander, S.P., Golden, D.M., Hamspon, R.F., Kurylo, M.J., Howard, C.J., Ravishankara, A.R., Kolb, C.E. and Molina, M.J. 1997. Chemical kinetics and photochemical data for use in stratospheric modeling. *Evaluation Number 12*. JPL Publication 97-4 Pasadena CA.

Diaz, S.B., Booth, C.R., Lucas, T.B. and Smolskaia, I. 1994. Effects of ozone depletion on irradiances and biological doses over Ushuaia. *Advances in Limnology* 43:115-122.

Dickerson, R.R., Kondragunta, S., Stenchikov, G., Civerolo, K.L., Doddridge, B.G. and Holben, B.N. 1997. The impact of aerosols on solar ultraviolet radiation and photochemical smog. *Science* 278:827-830.

Davies, R. 1993. Increased transmission of ultraviolet radiation to the surface due to stratospheric scattering. *Geophysical Research Letters* 98:7251-7253.

Eck,T.F., Bhartia, P.K. and Kerr, J.B. 1995. Satellite estimation of spectral UVB irradiance using TOMS derived total ozone and UV reflectivity. *Geophysical Research Letters* 22:611-614.

Estupinan, J.G., Raman, S., Crescenti, G.H., Streicher, J.J. and Barnard, W.F. 1996. The effects of clouds and haze on UV-B radiation. *Journal of Geophysical Research* 101:16807-16816.

Fioletov V.E. and Evans, W.F.J. 1997. The influence of ozone and other factors on surface radiation. In *Ozone Science: a Canadian Perspective on the changing ozone layer*, Wardle, D.I., Kerr, J.B., McElroy, C.T. and Francis, D.R. (eds.), University of Toronto Press, pp. 73-90.

Forster, P.M., Shine K.P. and Webb, A.R. 1995. Modelling ultraviolet radiation at the earth's surface. II. Model and instrument comparison. *Journal of Applied Meteorology* 34:2426-2439.

Frederick, J.E. and Alberts, A.D. 1991. Prolonged enhancement in surface ultraviolet radiation during the Antarctic spring of 1990. *Geophysical Research Letters* 18:1869-1871.

Frederick, J.E. and Erlick, C. 1995. Trends and interannual variations in erythemal sunlight, 1978-1993. *Photochemistry and Photobiology*, 62:476-484.

Frederick, J.E. and Lubin, D. 1988. The budget of biologically active ultraviolet radiation in the Earth-atmosphere system. *Journal of Geophysical Research* 93:3825-3832.

Frederick, J.E., Snell, H.E. and Haywood, E.K. 1989. Solar ultraviolet radiation at the Earth's surface. *Photochemistry and Photobiology* 51:443-450.

Frederick, J.E., Diaz, S.B., Smolskaia, I., Esposito, W., Lucas, T. and Booth, C.R. 1994. Ultraviolet solar radiation in the high latitudes of South America. *Photochemistry and Photobiology* 60:356-362.

Frederick, J.E., Koob, A.E., Alberts, A.D. and Weatherhead, E.C. 1993. Empirical studies of tropospheric transmission in the ultraviolet: Broadband measurements. *Journal of Applied Meteorology* 32:1883-1892.

Frederick, J.E., Qu, Z. and Booth, C.R. 1998. Ultraviolet radiation at sites on the Antarctic coast. *Photochemistry and Photobiology* 68:183-190.

Garadzha, M.P. and Nezval, Y.I. 1987. Ultraviolet radiation in large cities and possible ecological consequences of its changing flux due to anthropogenic impact. In *Proceedings of the Symposium on Climate and Human Health, World Climate Programme Applications*. WCPA Report No. 2, Leningrad, pp. 64-68.

Gardiner, B.G. and Martin, T.J. 1997. On measuring and modelling ultraviolet spectral irradiance. In *IRS'96: Current Problems in Atmospheric Radiation*, Smith, W.L. and Stamnes, K. (eds.), Proceedings of the International Radiation Symposium, Fairbanks, Alaska 19-24 August 1996, Deepak Publishing, Hampton Virginia, pp. 917-920.

Gardiner, B.G., Webb, A.R., Bais, A.F. et al. 1993. European intercomparison of ultraviolet spectroradiometers. *Environmental Technology* 14:24-43.

Geogdzhaev, I.V., Kondranin, T.V., Chuvarova, N.E. and Rublev, A.N. 1996. Comparison of UV measurements and modelling under broken cloudiness. In *IRS'96: Current Problems in Atmospheric Radiation*, Smith, W.L. and Stamnes, K. (eds.), Proceedings of the International Radiation Symposium, Fairbanks, Alaska 19-24 August 1996, Deepak Publishing, Hampton Virginia, pp. 865-868.

Gillotay, D., Müller, J.-F., Walravens, B. and Simon, P.C. 1997. The influence of didferent types of cloud layers on the UV climatology in Uccle, Belgium. In *IRS'96: Current Problems in Atmospheric Radiation*, Smith, W.L. and Stamnes, K. (eds.), Proceedings of the International Radiation Symposium, Fairbanks, Alaska 19-24 August 1996, Deepak Publishing, Hampton Virginia, pp. 921-924.

Gleason, J., Barthia, P.K., Herman, J.R., McPeters, R., Newman, P., Stolarski, R.S., Flynn, L., Labow, G., Larko, D., Seftor, C., Wellemeyer, C., Komhyr, W.D., Miller, A.J. and Planet, W. 1993. Record low global ozone in 1992. *Science* 260:523-526.

Häder, D.-P. and Worrest, R.C. 1991. Effects of enhanced solar ultraviolet radiation on aquatic ecosystems. *Photochemistry and Photobiology* 53:717-725.

Häder, D.-P., Worrest, R.C., Kumar, H.D. and Smith, R.C. 1994. Effects of increased solar UV-B irradiation on aquatic ecosystems. In *Environmental Effects of Stratospheric Ozone Depletion - 1994 Update*, van der Leun, J.C. and Tevini, M. (eds.), United Nations Environmental Programme, Nairobi, Kenya.

Herman, J.R., Bhartia, P.K., Kiemke, J., Ahmad, Z. and Larko, D. 1996. UV-B increases (1979-1992) from decreases in total ozone. *Geophysical Research Letters* 23:2117-2120.

Herman, J.R., Bhartia, P.K., Torres, O., Hsu, C., Seftor, C. and Celarier, E. 1997. Global Distribution of UV-Absorbing Aerosols From Nimbus-7/TOMS Data. *Journal of Geophysical Research* 102:16911-16922.

Herman, J.R., McKenzie, R.L., Diaz, S., Kerr, J., Madronich, S. and Seckmeyer, G. 1998. Ultraviolet Radiation at the Earth's Surface. In *Scientific Assessment of Ozone Depletion: 1998*, Albritton, D., Aucamp, P., Megie, G. and Watson, R. (eds.), World Meteorological Organization, Geneva, in press.

Hofmann, D.J., Oltmans, S.J., Bodhaine, B.A., Koenig, G.L., Harris, J.M., Lathrop, J.A., Schnell, R.C., Barnes, J., Chin, J., Kuniyuki, D., Ryan, S., Uchida, R., Yoshinaga, A., Neale, P.J., Hayes, D.R., Goodrich, R., Komhyr, W.D., Evans, R.D., Johnson, B.J., Quincy, D.M. and Clark, M. 1996. Record low ozone over

Mauna Loa observatory, Hawaii during the winter of 1994-1995. *Geophysical Research Letters* 23:1533-1536.

Hofmann, D.J. and Solomon, S. 1989. Ozone destruction through heterogeneous chemistry following the eruption of El Chichon. *Journal of Geophysical Research* 94:5029-5041.

Hsu, N.C., Seftor, C.J., Herman, J.R., Bhartia, P.K. and Thompson, A.M. 1997. Detection of biomass burning smoke and aerosols by TOMS. In *IRS'96: Current Problems in Atmospheric Radiation*, Smith, W.L. and Stamnes, K. (eds.), Proceedings of the International Radiation Symposium, Fairbanks, Alaska 19-24 August 1996, Deepak Publishing, Hampton Virginia, pp. 877-880.

Huber, M., Blumthaler, M. and Ambach, W. 1995. Total atmospheric ozone determined from spectral measurements of direct solar UV irradiance. *Geophysical Research Letters* 22:53-56.

ICNRP (International Commission on Non-Ionizing Radiation) 1995. UV-Index, 1995-1.

Ilyas, M. 1987. Effect of cloudiness on solar ultraviolet radiation reaching the surface. *Atmospheric Environment* 21:1483-1484.

Imbrie, C.W. and Murphy, T.M. 1982. UV-action spectrum (254-405 nm) for inhibition of a K^+-stimulated adenosine triphosphatase from a plasma membrane of Rosa damascena. *Photochemistry and Photobiology* 36:537-542.

Ito, T. 1993. UV-B observation network in the Japan Meteorological Agency. In *Frontiers of Photobiology*, Shima et al. (eds.), Elsevier Science Publishers.

Ito, T., Sakoda, Y., Uekubo, T., Naganuma, H., Fukuda, M. and Hayashi, M. 1994. Scientific application of UV-B observations from JMA Network. In *Proceedings of the 13th UOEH International Symposium and the 2nd Pan Pacific Cooperative Symposium on Human Health and Ecosystems*, Kodama, Y. and Lee, S.D. (eds.), Kitakyushu, Japan, pp. 107-125.

Josefsson, W. 1986. *Solar Ultraviolet Radiation in Sweden*. National Institute of Radiation Protection in Stockholm, SMHI Report-53, Norrköping, Sweden.

Kelfkens, G., de Gruijl, F.R. and van der Leun, J.C. 1990. Ozone depletion and increase in annual carcinogenic ultraviolet dose. *Photochemistry and Photobiology* 52:819-823.

Kerr, J.B. and McElroy, C.T. 1993. Evidence for large upward trends of ultraviolet-B radiation linked to ozone depletion. *Science* 262:1032-1034.

Keyse, S.M., Moses, S.H. and Davies, D.J.G. 1983. Action spectra for inactivation of normal and xeroderma pigmentosum human skin fibroblasts by ultraviolet radiation. *Photochemistry and Photobiology* 37:307-312.

Kieber, R.J., Zhou, X. and Moper, K. 1990. Formation of carbonyl compounds from UV-induced photodegradation of humic substances in natural waters: Fate of riverine carbon in the sea. *Limnology and Oceanography* 35:1503-1515.

Kirchoff, V.W.J.H., Zamorano, F. and Casiccia, C. 1997. UV-B enhancements at Punta Arenas, Chile. *Journal of Photochemistry and Photobiology B: Biology* 38:174-177.

Kjeldstad, B., Johnsen, B. and Koskela, T. 1997. *The Nordic Intercomparison of Ultraviolet and Total Ozone Instruments at Iana, October 1996.* Final report. Finnish Meteorological Institute Report No. 36, Helsinki.

Kligman, L.H. and Sayre, R.M. 1991. An action spectrum for ultraviolet induced elastosis in hairless mice: Quantification of elastosis by image analysis. *Photochemistry and Photobiology* 53:237-242.

Koskela, T., Keikkilä, A., Damski, J., Taalas, P. and Kylling, A. 1997. UV-B radiation at common optical air masses: geographical comaprison and model performance tests. In *IRS'96: Current Problems in Atmospheric Radiation*, Smith, W.L. and Stamnes, K. (eds.), Proceedings of the International Radiation Symposium, Fairbanks, Alaska 19-24 August 1996, Deepak Publishing, Hampton Virginia, pp. 925-929.

Krotkov, N.A., Barthia, P.K.,Herman, J., Celarier, E. and Eck, T. 1997. Estimates of spectral UVB irradiance from the TOMS instrument: Effects of clouds and aerosols. In *IRS'96: Current Problems in Atmospheric Radiation*, Smith, W.L. and Stamnes, K. (eds.), Proceedings of the International Radiation Symposium, Fairbanks, Alaska 19-24 August 1996, Deepak Publishing, Hampton Virginia, pp. 873-876.

Lantz, K.O., Shetter, R.E., Cantrell, C.A., Flocke, S.J., Calvert, J.G. and Madronich, S. 1996. Theoretical, actionometric, and radiometric determinations of the photolysis rate coefficient of NO_2 during MLOPEX II. *Journal of Geophysical Research* 101:14613-14629.

Leszczynski, K., Jokela, K., Ylianttila, L., Visuri, R. and Blumthaler, M. 1998. Erythemally weighted radiometers in solar UV monitoring: Results from WMO/STUK intercomparison. *Photochemistry and Photobiology* 67:212-221.

Liu, S., McKeen, S.A. and Madronich, S. 1991. Effect of anthropogenic aerosols on biologically active ultraviolet radiation. *Geophysical Research Letters* 18:2265-2268.

Loxom, F. and Kunkel C. 1997. The effect of aerosol optical depth on the measured diffuse fraction of UV spectral irradiance. In *IRS'96: Current Problems in Atmospheric Radiation*, Smith, W.L. and Stamnes, K. (eds.), Proceedings of the International Radiation Symposium, Fairbanks, Alaska 19-24 August 1996, Deepak Publishing, Hampton Virginia, pp. 930-933.

Lu, D. and Li, W. 1997. Observations and modeling of surface UV spectral radiances in China. In *IRS'96: Current Problems in Atmospheric Radiation*, Smith, W.L. and Stamnes, K. (eds.), Proceedings of the International Radiation Symposium, Fairbanks, Alaska 19-24 August 1996, Deepak Publishing, Hampton Virginia, pp. 934-937.

Lubin, D. and Jensen, E.H. 1995 Effects of clouds and stratospheric ozone depletion on ultraviolet radiation trends. *Nature* 377:710-713.

Ma, J. and Guicherit, R. 1997. Effects of stratospheric ozone depletion and tropospheric pollution on UVB radiation in the troposphere. *Photochemistry and Photobiology* 66:346-355.

Madronich, S. 1992. Implications of recent total atmospheric ozone measurements for biologically active ultraviolet radiation reaching the Earth's surface. *Geophysical Research Letters* 19:37-40.

Madronich, S. 1993. The atmosphere and UV-B radiation at ground level. In *Environmental UV Photobiology*, Björn, L.O. and Young, A.R. (eds.), Plenum Press, New York, pp. 1-39.

Madronich, S. 1993. UV radiation in the natural and perturbed atmosphere. In *Environmental Effects of UV (Ultraviolet) Radiation*, Tevini, M. (ed.), Lewis Publisher, Boca Raton, pp. 17-69.

Madronich, S. and Flocke, S. 1997. Theoretical estimation of biologically effective UV radiation at the Earth's surface. In *Solar Ultraviolet Radiation - Modeling, Measurements and Effects*, Zerefos, C. (ed.), NATO ASI Series Vol. I52, Springer-Verlag, Berlin.

Madronich, S., Björn, L.O., Ilyas, M. and Caldwell, M.M. 1991. Changes in biologically active ultraviolet radiation reaching the Earth's surface. In *Environmental Effects of Ozone Depletion: 1991 Update*, van der Leun, J.C., Tevini, M. and Worrest, R.C. (eds.), UNEP, Nairobi, 1991.

Madronich, S., McKenzie, R.L., Björn, L.O. and Caldwell, M.M. 1995. Changes in ultraviolet radiation reaching the Earth's surface. *Ambio* 24:143-152.

Madronich, S., Velders, G., Daniel, J., Lal, M., McCulloch, A. and Slaper, H. 1998. Halocarbon scenarios for the future ozone layer and related consequences. In *Scientific Assessment of Ozone Depletion: 1998*, Albritton, D., Aucamp, P., Megie, G. and Watson, R. (eds.), World Meteorological Organization, Geneva, in press.

Mayer, B. and Seckmeyer, G. 1996. All-weather comparison between spectral and broadband (Robertson-Berger) UV measurements. *Photochemistry and Photobiology* 64:792-799.

Mayer, B., Seckmeyer, G. and Kylling, A. 1997. Systematic long-term comparison of spectral UV measurements and UVSPEC modeling results. *Journal of Geophysical Research* 102:8755-8767.

Mayer, B., Kylling, A., Madronich, S. and Seckmeyer, G., 1998. Enhanced absorption of UV irradiance due to multiple scattering in clouds: experimental evidence and theoretical explanation. *Journal of Geophysical Research*, in press.

McKenzie, R.L., Matthews, W.A. and Johnston, P.V. 1991. The relationship between erythemal UV and ozone, derived from spectral irradiance measurements. *Geophysical Research Letters* 18:2269-2272.

McKenzie, R.L., Kotkamp, M., Seckmeyer, G., Erb, R., Roy, C.R., Gies, H.P. and Toomey, S.J. 1993. First southern hemisphere intercomparison of measured solar UV spectra. *Geophysical Research Letters* 20:2223-2226.

McKenzie, R.L., Johnston, P.V., Bodeker, G.E., Kotkamp, M., Bodhaine, B.A., Hofmann, D.J., Dutton, E.G., Schnell, R.C., Barnes, J.E. and Ryan, S.C. 1998. UV spectral irradiance comparison and contrast between Mauna Loa observatory, Hawaii, and Lauder, New Zealand. In *Proceedings of the XVIII Quadrennial Ozone Symposium*, Bojkov, R.D. and Visconti, G. (eds.), Parco Scientifico e Technologico d'Abruzzio, L'Aquila, Italy pp. 857-860.

McKinlay, A.F. and Diffey, B.L. 1987. A reference action spectrum for ultraviolet induced erythema in human skin. In *Human Exposure to Ultraviolet Radiation: Risks and Regulations*, Passchier, W.R. and Bosnjakovic, B.F.M. (eds.), Elsevier, Amsterdam.

McPeters, R.D., Hollandsworth, S.M., Flynn, L.E., Herman, J.R. and Seftor, C.J. 1996. Long-term ozone trends derived from the 16-year combined Nimbus 7/Meteor 3 TOMS Version 7 record. *Geophysical Research Letters* 23:3699-3702.

Meerkoetter, R, Wissinger, B. and Seckmeyer, G. 1997. Surface UV from ERS-2/GOME and NOAA/AVHRR data: A case study. *Geophysical Research Letters* 24:1939-1942.

Michelangeli, D.V., Allen, M., Yung, Y.L., Shia, R.-L., Crisp, D. and Eluszkiewicz, J. 1992. Enhancement of atmospheric radiation by an aerosol layer. *Journal of Geophysical Research* 97:865-874.

Mims, F.M.III. and Frederick, J.E. 1994. Cumulus clouds and UV-B. *Nature* 371:291.

Mims, F.M.III., Ladd, J.W. and Blaha, R.A. 1995. Increased solar ultraviolet-B associated with record low ozone over Texas. *Geophysical Research Letters* 22:227-230.

Mims, F.M.III. 1997. Biological effects of diminished UV and visible sunlight caused by severe air pollution. In *IRS'96: Current Problems in Atmospheric Radiation*, Smith, W.L. and Stamnes, K. (eds.), Proceedings of the International Radiation Symposium, Fairbanks, Alaska 19-24 August 1996, Deepak Publishing, Hampton Virginia, pp. 905-908.

Montzka, S.A., Butler, J.H., Myers, R.C., Thompson, T.M., Swanson, T.H., Clarke, A.D., Lock, L.T. and Elkins, J.W. 1996. Decline in the tropospheric abundance of halogen from halocarbons: implications for stratospheric ozone depletion. *Science* 272:1318-1322.

Müller, R., Crutzen, P.J., Gross, J.-U., Brühl, C., Russel, J.M.III., Gernandt, H., McKenna, D.S. and Tuck, A.F. 1997. Severe chemical ozone loss in the Arctic during the winter of 1995-96. *Nature* 389:709-712.

Nack, M.L. and Green, A.E.S. 1974. Influence of clouds, haze, and smog on the middle ultraviolet reaching the ground. *Applied Optics* 13:2405-2415.

Nunez, M., Michael, K., Turner, D., Wall, M. and Nilsson, C. 1997. A satellite-based climatology of UV-B irradiance for Antarctic coastal regions. *International Journal of Climatology* 17:1029-1054.

Orce, V.L., Paladini, A.A., San Roman, N.A., Labarga, J.C. and Helbing, E.W. 1997. Latitudinal UVR-PAR measurements in Argentina: extent of the 'ozone hole'. *Global and Planetary Change* 15:113-121.

Pachart, E., Lenoble, J., Brogniez, C., Bocquet, J.L. and Wang, P. 1997. Modelling of UV-B irradiance – complementarity with measurements. In *IRS'96: Current Problems in Atmospheric Radiation*, Smith, W.L. and Stamnes, K. (eds.), Proceedings of the International Radiation Symposium, Fairbanks, Alaska 19-24 August 1996, Deepak Publishing, Hampton Virginia, pp. 857-860.

Paltridge, G.W. and Barton, I.J. 1978. *Erythemal Ultraviolet Radiation Distribution over Australia - the Calculations, Detailed Results and Input Data Including Frequency Analysis of Observed Australian Cloud Cover*. Division of Atmospheric Physics. Technical Paper-33, Commonwealth Scientific and Industrial Research Organization, Australia.

Peak, M.J., Peak, J.G., Moehring, M.P. and Webb, R.B. 1984. Ultraviolet action spectra for DNA dimer induction, lethality, and mutagenesis in *Escherichia coli* with emphasis on the UVB region. *Photochemistry and Photobiology* 40:613-620.

Pfister, G., Puts, E., Bernhard, G., Seckemyer, G. and Mayer, B. 1997. Comparison of high resolution spectral UV –measurements with DISORT model using different extraterrestrial spectra. In *IRS'96: Current Problems in Atmospheric Radiation*, Smith W.L. and Stamnes, K. (eds.), Proceedings of the International Radiation Symposium, Fairbanks, Alaska 19-24 August 1996, Deepak Publishing, Hampton Virginia, pp. 938-941.

Piazena, H. 1996. The effect of altitude upon the solar UV-B and UV-A irradiance in the tropical Chilean Andes. *Solar Energy* 57:133-140.

Pitts, D.G., Cullen, A.P. and Hacker, P.D. 1977. Ocular effects of ultraviolet radiation from 295 to 365 nm. *Investigative Ophthalmology* 16:932-939.

Qu, J., Smith, F., Riebau, A., Sestak, M., Smith, L. and Coloff, S.G. 1997. Global solar ultraviolet-B radiation measurements and simulations in remote areas. In *IRS'96: Current Problems in Atmospheric Radiation*, Smith W.L. and Stamnes, K. (eds.), Proceedings of the International Radiation Symposium, Fairbanks, Alaska 19-24 August 1996, Deepak Publishing, Hampton Virginia, pp. 942-945.

Quaite, F.E., Sutherland, B.M. and Sutherland, J.C. 1992. Action spectrum for DNA damage in alfalfa lowers predicted impact of ozone depletion. *Nature* 358:576-578.

Rex, M. et al. 1997. Prolonged stratospheric ozone loss in the 1995-96 Arctic winter. *Nature* 389:835-838.

Rundel, R.D. 1983. Action spectra and the estimation of biologically effective UV radiation. *Journal of Investigative Opthalmology* 58:360-366.

Sasaki, M., Takeshita, S., Komatsu, H., Sugiura, M. and Sakata, T. 1997. Global solar ultraviolet-B irradiance at 35°N in Japan since 1990. In *IRS'96: Current Problems in Atmospheric Radiation*, Smith W.L. and Stamnes, K. (eds.), Proceedings of the International Radiation Symposium, Fairbanks, Alaska 19-24 August 1996, Deepak Publishing, Hampton Virginia, pp. 898-901.

Schafer, J.S., Saxena, V.K., Wenny, B.N., Barnard, W. and De Luisi, J.J. 1996. Observed influence of clouds on ultraviolet-B radiation. *Geophysical Research Letters* 23:2625-2628.

Schindell, R.T., Rind, D. and Lonergan, P. 1998. Increased polar stratospheric ozone losses and delayed eventual recovery owing to increasing greenhouse-gas concentrations. *Nature* 392:589-592.

Scotto, J., Cotton, G., Urbach, F., Berger, D. and Fears, T. 1988. Biologically effective ultraviolet radiation: Surface measurements in the United States, 1974 to 1985. *Science* 239:762-764.

Seckmeyer, G. and McKenzie, R.L. 1992. Elevated ultraviolet radiation in New Zealand (45°S) contrasted with Germany (48°N). *Nature* 359:135-137.

Seckmeyer, G., Erb, R. and Albold, A. 1996. Transmittance of a cloud is wavelength-dependent in the UV-range. *Geophysical Research Letters* 23:2753-2755.

Seckmeyer, G., Mayer, B., Bernhard, G., Erb, R., Albold, A., Jäger, H. and Stockwell, W.R. 1997. New maximum UV irradiance levels observed in Central Europe. *Atmospheric Environment* 31:2971-2976.

Seckmeyer, G., Mayer, B., Bernhard, G., McKenzie, R.L., Johnston, P.V., Kotkamp, M., Booth, C.R., Lucas, T. and Mestechikina, T. 1995. Geographical differences in the UV measured by intercompared spectroradiometers. *Geophysical Research Letters* 22:1889-1892.

Seckmeyer, G., Thiel, S., Blumthaler, M., Fabian, P., Gerber, S., et al. 1994. Intercomparison of spectral UV radiation measurement systems. *Applied Optics* 33:7805-7812.

Setlow, R.B. 1974. The wavelengths in sunlight effective in producing skin cancer: a theoretical analysis. *Proceedings of the National Academy of Science* 71:3363-3366.

Setlow, R.B., Grist, E., Thompson, K. and Woodhead, A.D. 1993. Wavelengths effective in induction of malignant melanoma. *Proceedings of the National Academy of Science* 90:6666-6670.

Slaper H., Reinen H.A.J.M, Blumthaler, M., Huber, M. and Kuik, F. 1995. Comparing ground-level spectrally resolved solar UV measurements using various instruments: A technique resolving effects of wavelength shift and slit width. *Geophysical Research Letters* 22:2721-2724.

Stein, B., Rahmsdorf, H.J., Steffen, A., Litfin, M. and Herrlich, P. 1989. UV-induced DNA damage is an intermediate step in UV-induced expression of human immunodeficiency virus type 1, collagenase, c-fos, and metallothionein. *Molecular and Cellular Biology* 9:5169-5181.

Stolarski, R. 1997. A bad winter for Arctic ozone. *Nature* 389:788-789.

Taalas, P., Damski, J., Kyro, E., Ginzberg, M. and Talamoni, G. 1997. Effect of stratospheric ozone variations on UV radiation and on tropospheric ozone at high latitudes. *Journal of Geophysical Research* 102:1533-1539.

Thompson, A., Early, E.A., DeLuisi, J., Disterhoff, P., Wardle, D., Kerr, J., Rives, J., Sun, Y., Lucas, T., et al. 1997. The 1994 North American interagency intercomparison of ultraviolet monitoring spectroradiometers. *Journal of the National Institute of Standards and Technology* 102:279-322.

Tsitas, S.R. and Yung, Y.L. 1996. The effect of volcanic aerosols on ultraviolet radiation in Antarctica. *Geophysical Research Letters* 23:157-160.

UNEP (United Nations Environment Programme) 1991. van der Leun, J.C., Tevini, M. and Worrest, R.C. (eds.), *Environmental Effects of Ozone Depletion: 1991 Update*. United Nations Environment Programme, Nairobi, 1991.

UNEP (United Nations Environment Programme) 1994. *Environmental Effects of Ozone Depletion: 1994 Update*, van der Leun, J.C., Tevini, M., Tang, X. and Worrest, R.C. (eds.), United Nations Environment Programme, Nairobi, 1994.

UNEP (United Nations Environment Programme) 1998. *Environmental Effects of Ozone Depletion: 1998 Update*, van der Leun, J.C., Tevini, M., Tang, X. and Worrest, R.C. (eds.), United Nations Environment Programme, Nairobi, this report.

Urbach, F., Berger, D. and Davies, R.E. 1974. Field measurements of biologically effective UV radiation and its relation to skin cancer in man. In *Proceedings of the Third Conference on Climatic Impact Assessment Program*, Broderick, A.J. and Hard T.M. (eds.), US Dept. of Transportation, Washington, DC.

Varotsos, C. and Kondratyev, K.Y. 1995. On the relationship between total ozone and solar ultraviolet radiation at St. Petersburg, Russia. *Geophysical Research Letters* 22:3481-3484.

Varotsos, C.A., Chronopoulos, G.J., Katsikis, S. and Sakellariou, N.K. 1995. Further evidence of the role of air pollution on solar ultraviolet radiation reaching the ground. *International Journal of Remote Sensing* 16:1883-1886.

Vogelmann, A.M., Ackerman, T.P. and Turco, R.P. 1992. Enhancements in biologically effective radiation following volcanic eruptions. *Nature* 359:47-49.

Weatherhead, E.C., Tiao, G.C., Reinsel, C., Frederick, J.E., DeLuisi, J.J., Choi, D. and Tam, W. 1997. Analysis of long-term behavior of ultraviolet radiation measured by Robertson-Berger meters at 14 sites in the United States. *Journal of Geophysical Research* 102:8737-8754.

Webb, A.R. 1997. *Advances in Solar Ultraviolet Spectroradiometry*. European Commission Air Pollution Research Report 63, Luxembourg.

Weihs, P. and Webb, A.R. 1997. Accuracy of spectral UV model calculations 2. Comparison of UV calculations with measurements. *Journal of Geophysical Research* 102:1551-1560.

Wellmann, E. 1985. UV-B-Signal/Response-Beziehungen unter natürlichen und artifiziellen Lichtbedingungen. *Berichte der Deutschen Botanischen Gesellschaft* 98:99-104.

Wenny, B.N., Schafer, J.S., DeLuisi, J.J., Saxena, V.K., Barnard, W.F., Petropavlovskikh, I.V. and Vergamini, A.J. 1998. A study of regional aerosol radiative properties and effects on ultraviolet-B radiation. *Journal of Geophysical Research* 103:17083-17097.

WMO (World Meteorological Organization) 1994a. Scientific Assessment of Ozone Depletion: 1994. In *Global Ozone Research and Monitoring Project*, Albritton, D.L, Watson, R.T. and Aucamp, P.J. (eds.), Report No. 37, World Meteorological Organization, Geneva.

WMO (World Meteorological Organization) 1994b. *Report of the WMO Meeting of Experts on UV-B Measurements, Data Quality and Standardization of UV Indices*. World Meteorological Organization Global Atmosphere Watch Report No. 95, Geneva.

WMO (World Meteorological Organization) 1998. Scientific Assessment of Ozone Depletion: 1998. In *Global Ozone Research and Monitoring Project*, Albritton, D.L, Aucamp, P.J., Megie, G. and Watson, R.T. (eds.), World Meteorological Organization, Geneva, in press.

Zeng, J., McKenzie, R.L., Stamnes, K., Wineland, M. and Rosen, J. 1994. Measured UV spectra compared with discrete ordinate method simulations. *Journal of Geophysical Research* 99:23019-23030.

Zepp, R.G. and Andreae, M.O. 1994. Factors affecting the photochemical formation of carbonyl sulfide in seawater. *Geophysical Research Letters* 21:2813-2816.

Zepp, R.G., Hoign, J. and Bader, H. 1987. Nitrate-induced photooxidation of trace organic chemicals in water. *Environmental Science and Technology* 21:443-450.

Zerefos, C.S., Balis, D.S., Bais, A.F., Gillotay, D., Simon, P.C., Mayer, B. and Seckmeyer, G. 1997. Variability of UV-B at four stations in Europe. *Geophysical Research Letters* 24:1363-1366.

Zölzer, F. and Kiefer, J. 1984. Wavelength dependence of inactivation and mutation induction to 6-thioguanine-resistance in V79 Chinese hamster fibroblasts. *Photochemistry and Photobiology* 40:9-53.

CHAPTER 2

HEALTH RISKS

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Summary

The health risks associated with ozone depletion will principally be those due to increased ultraviolet B radiation (UV-B) in environment, i.e., increased damage to the eyes, the immune system and the skin. Some new risks may also be introduced with the increased use of alternatives to the ozone-depleting substances (ODSs). Quantitative risk estimates are available for some of the UV-B-associated effects, e.g., cataract and skin cancer; however the data are insufficient to develop similar estimates for effects such as immunosuppression and the toxicity of alternatives.

Ocular damage from UV exposures includes effects on the cornea, lens, iris and associated epithelial and conjunctival tissues. The most common acute ocular effect of environmental ultraviolet radiation (UVR) is photokeratitis. Also known as snowblindness in skiers, this condition also occurs in other outdoor recreationists. Chronic eye conditions likely to increase with ozone depletion include cataract, squamous cell carcinoma, ocular melanoma and a variety of corneal/conjunctival effects, e.g., pterygium and pinguecula.

Suppression of local (at the site of UV exposure) and systemic (at a distant, unexposed site) immune responses to a variety of antigens has been demonstrated in both humans and animals exposed to UV-B. In experiments with animals these effects have been shown to worsen the course/outcome of some infectious diseases and cancers. There is reasonably good evidence that such immunosuppression plays a role in human carcinogenesis; however, the implications of such immunosuppression for human infectious diseases are still unknown.

In light-skinned populations, exposure to solar UVR appears to be the most important environmental risk factor for basal and squamous cell carcinomas and cutaneous melanoma. Originally it was believed that total accumulated exposure to UVR was the most important environmental factor in determining risk for these tumors. Recent information now suggests, that only squamous cell carcinoma risk is related to total exposure. In the cases of both basal cell carcinoma and melanoma, new information suggests that increases in risk are tied to early exposures (before about age 15), particularly those leading to severe sunburns.

Testing of a number of the CFC alternatives indicates that most of these chemicals have low acute toxicity, and low to moderate chronic toxicity. Some chemicals that were originally proposed as alternatives have been dropped from consideration because these tests raised concerns about toxicity and/or manufacturing difficulties. In one instance, high accidental occupational exposure was associated with liver damage, underlining the need for care in the use of these substitutes.

Recent quantitative risk estimates have been developed for cataract, melanoma and all skin cancers combined. These estimates indicate that under the Montreal adjustments cataract, and skin cancer incidence will peak mid-century at an additional incidence of just under 3 per 100,000 and about 7 per 100,000, respectively.

Introduction

As discussed in Chapter 1, nearly everyone and indeed every living thing is likely to be exposed to sunlight and the UV-B it contains for various periods during their life. In humans and animals, exposure is principally via the eyes and skin, with effects occurring as a result of the absorption of solar energy by molecules (termed chromophores) present in the tissues/cells present in these organs. As displayed in Fig. 2.1, the absorption of light energy leads to changes in these molecules that eventually can result in a biologic effect.

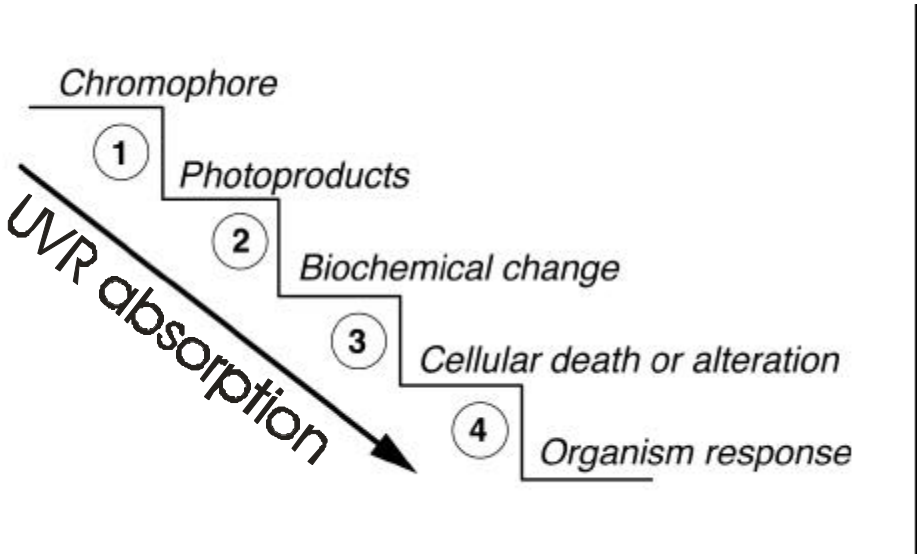

Fig. 2.1 Events in light-induced effects

Chromophores absorb light energy from the various wavelengths with differing efficiencies. This pattern of absorption is called an absorption spectrum and is characteristic of the type of molecule involved. Figure 2.2 shows absorption spectra for five of the chromophores present in skin and eye tissues that are thought to be important to the biologic effects of UV-B in humans and animals. These are DNA, tyrosine and tryptophan (two amino acids that are largely responsible for the UV absorbance of proteins), trans-urocanic acid (a molecule present in large amounts in the outermost layer of skin), and melanin (the principal pigment of the skin). The gray area in Fig. 2.2 marks that part of the UV spectrum, wavelengths under 290 nm, which is not present in terrestrial energy. Thus only those portions of these absorption spectra appearing in the white area (above 290 nm) are likely to be of any relevance to the effects associated with environmental exposures. As Fig. 2.2 indicates, for all of the molecules except melanin, absorption efficiency drops rapidly within the terrestrial UV-B spectral region with little or no absorbance in the UVA spectral region (above 320nm). Thus the increase in UV-B that accompanies ozone depletion will increase the amount of biologically active radiation present in ambient sunlight. As chapter 1 has discussed in more detail, while it is difficult to predict quantitatively exactly how these increases will be distributed globally, such increases have been observed in a variety of sites across the world. Because of the biologic activity of UV-B, such increases are likely to

have marked consequences for humans as well as other living creatures. Some of these consequences could be beneficial, e.g., a greater production of vitamin D in the skin of humans, but far more are likely to be detrimental.

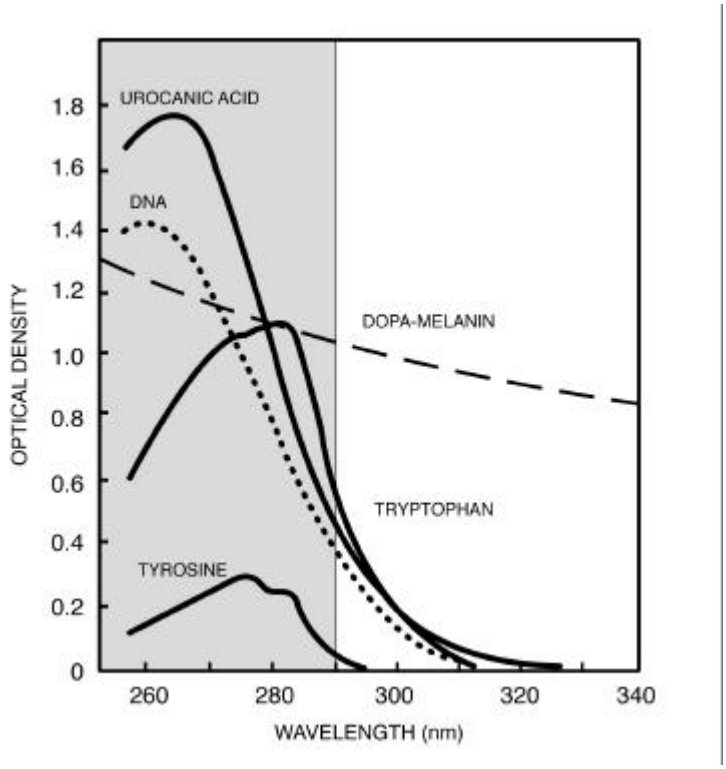

Fig. 2.2 UVR absorption spectra of molecules important to UV-induced health effects.

This chapter presents an overview of the consequences likely to accompany increases in UV-B. It will focus on the possible health risks and only briefly mention possible beneficial effects when these might offset adverse effects or when concerns about them might modify adaptive strategies. The chapter's design is adapted from a four-step risk assessment approach. It first identifies the hazards. Second, it discusses a variety of factors that can modify exposure or susceptibility. Third, it presents quantitative and qualitative estimates of risk with their attendant uncertainties, and fourth, it ends with a brief discussion of potential risks associated with several of the strategies being adopted to manage or mitigate risk.

Hazards for Humans

Humans have three major organ systems whose cells and tissues are commonly exposed to sunlight: the eye, the immune system and the skin, and it is in these three systems that the effects of sunlight on health have been documented. The cells/tissues exposed in the eye are principally those associated with the cornea; the iris and the lens, those of the skin include the outermost layer of the skin, the stratum corneum, and the epidermis; and those of the immune system are the Langerhans (or antigen-presenting) cells that reside in, or migrate through the epidermis.

Each of the different types of UV-exposed tissues contains a collection of chemical substances whose light-absorbing properties can contribute to the process shown in Fig. 2.1. Furthermore, the or-

gan systems are structured such that some tissues/cells will absorb part of the UV energy before it reaches others. Thus the spectrum of light which first hits the surface of an organ such as the skin, is not the same as that reaching tissues/cells located deeper, e.g., in the basal layer of the epidermis. As a result, the wavelength dependence, or action spectrum, of a particular end-point of concern rarely looks exactly like the absorption spectrum of a particular chromophore. Figure 2.3 shows action spectra for several of the more important effects, sunburn, DNA damage (dimers) and carcinogenesis, that will be discussed in detail below. Note again that only absorbance above 290 is relevant to environmental exposures

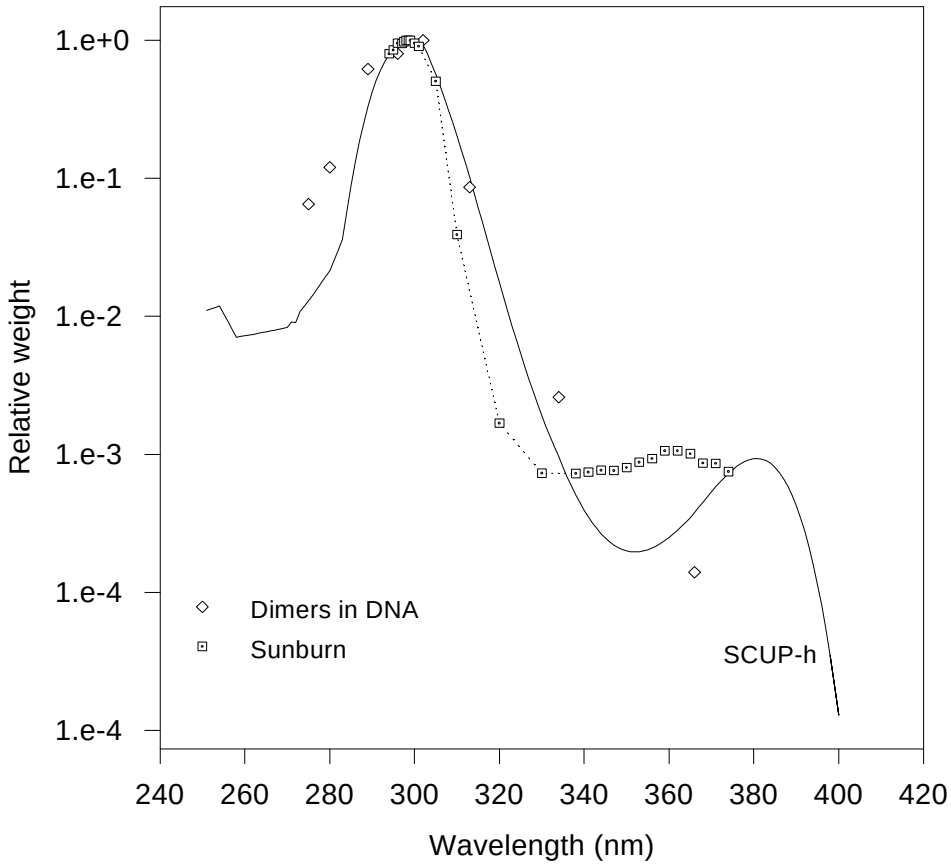

Fig. 2.3. Action spectra of key UV-associated health effects.

Effects on the Eye

As indicated above, the eyes are a principal route of exposure to UVR. As illustrated in Fig. 2.4, when sunlight (and the UVR it contains) impinges on the normal eye, the cornea is encountered first, then the lens, the vitreous humor, and the retina. Studies indicate that due to its absorption by various molecules in the cornea and the lens, most UVR never reaches the retina in the normal adult eye. In the case of ambient UVR (i.e., UV-B and UV-A), the shorter wavelengths are absorbed preferentially,

with the cornea absorbing most of the radiation below 300 nm, and the lens absorbing almost all of the rest of the UVR below about 370 nm (Merriam, 1996). Lens removal (as for the treatment of cataract) does place the retina at risk for UV damage; it is for this reason that many artificial replacement lenses are made with UV-absorbing materials.

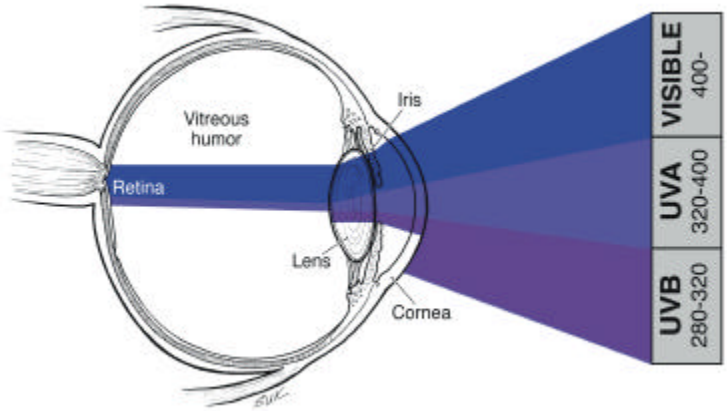

Fig. 2.4 Absorption of UVR by the eye

Effects on the Cornea/Conjunctiva

The ocular effect most directly attributable to environmental exposures to UVR is photokeratitis. The ocular equivalent of sunburn, this effect occurs after an acute, i.e., short term, exposure and is characterized by reddening (inflammation) of the eyeball, gritty feeling of severe pain, tearing, photophobia (avoidance of light) and blepharospasm (twitching). Frequently diagnosed in skiers as ”snowblindness”, photokeratitis is also seen in beach-goers and others involved in outdoor recreation. (McCarty and Taylor, 1996). Mechanistic studies have revealed that human corneal stromal cells when exposed to low doses (10-100 mJ/cm2) either in vitro or in situ show significant increases in the production of a number of biologically-active chemicals, i.e., cytokines. The cytokines detected, interleukin (IL)-1, IL-6, IL-8 and tumor necrosis factor alpha (TNFα), are proinflammatory and may be responsible for the inflammation which accompanies photokeratitis (Kennedy et al., 1997).

Additional ocular effects on the cornea/conjunctiva attributed to solar exposure are climatic droplet keratopathy (CDK), pinguecula, pterygium, and squamous cell carcinoma (SCC) of the cornea and conjunctiva. CDK is a degeneration of the fibrous layer of the cornea with the accumulation of droplet shaped deposits. Pterygium results from an outgrowth of the conjunctiva (outermost mucous layer) over the cornea, which results in the loss of transparency, and pinguecula is a raised opaque mass just adjacent to the cornea (Hollows, 1989) and SCC is a malignant neoplasm similar to those found on sun-exposed skin. Data supporting the relationship between solar exposure and disease are strongest with CDK and pterygium; epidemiologic studies indicate that chronic exposure to the sun, and, most probably UV-B, is an important factor in development of these diseases. Both are associated with outdoor living or working in environments with high surface reflectance, e.g., water, sand, concrete (Hollows, 1989; Taylor et al., 1989; Mackenzie et al., 1992). Interestingly, in a recent study of pterygium, a much-increased risk (36-fold) was found in people with early, intense exposures (residing at 30 degrees south latitude or less for the first 5 years of life). This was independent of the almost 40-fold increase in risk associated with a work environment below 30 degrees south between the ages of 20-29 (Mackenzie et al., 1992). Data linking pinguecula to solar exposure are largely anecdotal or

based on case reports (Bergmanson and Sheldon, 1997), although Taylor and his colleagues (1989) found a weak association between pinguecula and UVR exposure in the Maryland Watermen study.

A recent study of SCC of the eye (Newton et al., 1996) examined incidence data from across the world in order to assess whether solar UVR is a risk factor for this disease. The study focused on conjunctival and corneal lesions, excluding those on the eyelid, and developed estimated daily UV-B exposures weighted using the erythemal action spectrum. Because HIV infection increases the risk of conjunctival SCC, and two African centers have seen substantial increases in this tumor in the past 5-10 years, the analysis looked at two data sets, one including data from Africa and one excluding these data. With the African data, the study found an increase in incidence of SCC of the eye with UV-B exposure that is equivalent to almost a 50% increase in incidence for every 10° decrease in latitude. Without the African data, an equivalent 40% decline was found for each 10° increase in latitude.

Effects on the Uveal Tract

The uveal tract consists of the iris, ciliary body and choroid. Malignant melanoma of the uveal tract is the most commonly occurring primary ocular malignancy. Rare in Blacks, in white patients it most commonly occurs in the choroid (Berkow, 1992), but also occurs in the iris. Several epidemiologic studies (Holly et al., 1996), found an increased risk of intraocular melanomas associated with sensitivity to UV. In these studies, sensitivity to sunburn was associated with almost a two-fold increase in risk of intense UV exposures, e.g., prior history of a welding burn or snow blindness was associated with more than a seven-fold increase in risk (6et al., 1990). Others have found the evidence less than compelling (Dolin and Johnson, 1994; Schwartz et al., 1997). The latter analysis, however, examined only the hypothesis of a direct effect of UV on the affected cells (i.e., a DNA-damaging effect) and did not evaluate the role of indirect effects that might contribute to carcinogenesis such as immunosuppression associated with increased production of cytokines (Kennedy et al., 1997). That such processes can occur following UV exposure of the eyes is suggested by the recent finding that in mice, high ocular doses of UV-B can produce systemic immunosuppression equivalent to that obtained by skin irradiation. Furthermore, severing the optic nerve prevented this immunosuppression (Hiramoto et al., 1997).

Effects on the Lens

Of all of the ocular diseases associated with solar exposure, that which affects the lens, cataract, is by far the most important from a public health perspective. Characterized by a gradual loss in the transparency of the lens (due to the accumulation of oxidized lens proteins) (McCarty and Taylor, 1996), the end-result is frequently blindness, unless the affected lens is surgically removed.

Several different kinds of cataract are distinguished based on their location in the lens. Cortical cataracts develop in the outer layers of lens protein, commonly called the cortex of the lens. Nuclear cataracts occur in the inner layers of lens protein, i.e., the nucleus of the lens. And, posterior subcapsular cataracts (PSC) occur at the back (posterior) interface of the lens and its epithelial capsule. A fourth form of cataract is mixed, i.e., combining elements of two or more of the aforementioned forms. In a recent Italian case-control study of individuals aged 45-79 years old, pure cortical cataract accounted for slightly less than 50% of cases, pure nuclear cataract accounted for about 10%, pure PSC for less than 3%, and mixed for about 40% with the majority of the mixed having a cortical component (Italian American Cataract Study Group, 1991; 1994).

The epidemiologic evidence identifying exposure to UV-B as a risk factor for cataract suggests that the risk may be limited to pure or mixed cortical cataracts and PSC with the exception of the nuclear/cortical combination (Italian American Cataract Study Group, 1994). In the case of cortical

cataract, a number of studies have indicated that the relative risk associated with increased sun exposure is between about 1- and 3fold (Italian American Cataract Study Group, 1991; Klein et al., 1995; West and Valmadrid, 1995, West et al. 1998). In the Beaver Dam Eye Study, this places heavy sun exposure about on a par with diabetes or heavy drinking as a risk factor (Klein et al., 1995).

The economic and social importance of cataract is enormous. It is be leading cause of blindness in the world(West and Valmadrid, 1995), with public health care costs for cataract surgery in the U.S. exceeding $3 billion in 1992. With the prevalence of cataract after age 30 approximately doubling each decade, anything that accelerates onset by 10 years (e.g., the increase in UV achieved in moving from the northernmost to the southernmost regions of the US) would double the number of operations (Javitt and Taylor, 1995).

Effects on the Immune System

In humans, the skin is the principal barrier to the outside world, and thus the first line of defense against foreign agents that may threaten health. In order to fulfill this role, the skin hosts a number of cells from the immune system that can mount or modify immune responses against such 'foreign invaders' or against skin cells that have become 'strange', e.g., by virus infection or transformation into a cancer cell. However, to function optimally, the immune system needs to be able to discriminate between 'self' and ‘strange’ or 'non-self', and eliminate only the latter, especially if it is (potentially) harmful.

Immunosuppression

As mentioned above, the skin contains a wide range of molecules, including both proteins and DNA, which undergo photochemical reactions upon absorbing UVR. It is quite likely that a great many of the cell-surface proteins which are used to determine 'self' are modified in such photochemical reactions so that at certain UV doses, the skin becomes swamped with ‘non-self’ cells. Were the immune system to react to all of these cells, the resulting inflammatory response might compromise other important skin functions. For this reason, it is believed that the decreased immune responses observed after UV irradiation serve to prevent excessive inflammation and damage to the skin that has been exposed to the sun. The drawback of this postulated beneficial physiological response is that it may be detrimental when it coincides with the entry of an infectious agent, or the development of a cancer cell, against which a forceful immune reaction needs to be mounted. These immunosuppressive effects of UV exposure can thus result in adverse circumstances: i.e., implants of UV-induced tumors between genetically identical mice, are rejected in a naive, unexposed host, but they fail to be rejected in a UV exposed host (Fisher and Kripke, 1977). Such a UV-induced immune suppression has also been found for contact hypersensitivity (CH) reactions (a type of immune reaction seen following skin contact with certain reactive chemicals, e.g., poison ivy) (Kripke, 1984; Yoshikawa, et al., 1990; Cooper et al., 1992). It also is seen in delayed type hypersensitivity (DTH) reactions (the kind of immune response made against virus-infected cells and certain microorganisms) (Howie et al., 1986). Figure 2.5 shows the two phases of the contact allergy response. UV irradiation can decrease both the induction of new responses through immunization and the elicitation of established immunity. Furthermore, immune suppression can occur locally, within UV-irradiated skin, or systemically at distant sites, depending on the dose of UV and the type of immune response.

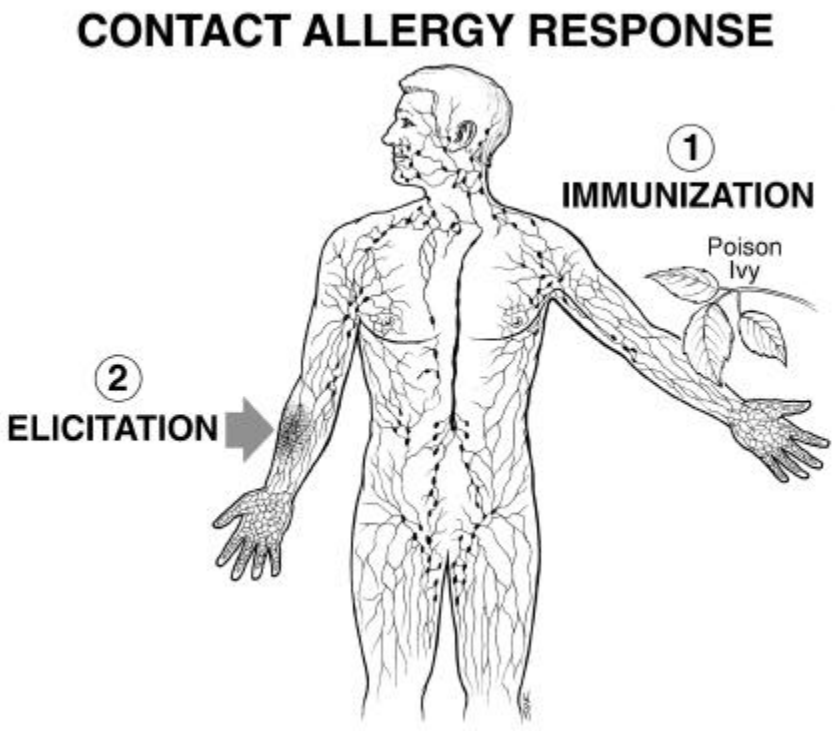

Fig. 2.5 Multiple phases of contact hypersensitivity responses.

Clearly, the switch from an immune reaction to UV-induced suppression of it needs to be well tuned; at one extreme, too much suppression could render an individual susceptible to infections, whereas at the other extreme, too little could result in skin-damaging inflammatory reactions upon UV exposure. The latter would resemble what has commonly been referred as an allergic reaction to sunlight (a 'sun allergy'), that a physician would diagnose as a photodermatosis, e.g. UV-B-induced polymorphic light eruption, PLE. This disease can often be treated successfully by subjecting the skin to a series of gradually increasing UV-B irradiations, which are thought to permit the skin to adapt slowly to the effects of UVB (van der Leun and van Weelden, 1986). Thus, PLE patients appear to suffer from a compromised adaptation response; their immune response can adapt to small changes in UV-B, but is overwhelmed by large ones. This could explain why PLE is more common toward the poles (van der Leun and De Gruijl, 1993), where the seasonal UV modulation is greatest. If, as projected, large decreases in ozone occur during the wintertime at higher latitudes, one would expect to see a decrease in the seasonal UV modulation at these locations and a lowered incidence of PLE.

Mechanisms

Immunological reactions tend to be rather complex because they involve multiple simultaneous processes that can act in concert or in opposition to one another. The impact of UV irradiation appears principally to be on cellular immune responses which are mediated through direct cell contact, and usually do not affect humoral immunity that is mediated through blood-borne proteins, e.g., so-called 'antibodies'. However, within the cellular immune response, there are multiple reactive sub-pathways that are affected differently by UV radiation. Current research on UV immunosuppression has had to recognize and account for these differences in order to understand the many, some of them seemingly contradictory, results. It is beyond the scope of this chapter to deal with this matter in any great detail. However, some of this information, in particular, the relevant action spectra, the role of antigen pre-

senting cells and the genetic factors that can modify these responses, are necessary to the development of risk management strategies and so is briefly summarized here.

A critical first step in understanding UV-induced immunosuppression is knowledge of the important chromophore(s). Unfortunately, recent research has increased rather than decreased the list of possibilities. Initially, there appeared to be two major chromophores of interest: urocanic acid (UCA; DeFabo and Noonan 1983), and DNA (Kripke et al., 1992), each playing a distinct role in the immune response. Finding from a number of different groups now contribute to the conclusion that both UCA and DNA are important to UV-induced systemic immunosuppression, and that under ambient exposures involving both UV-A and UV-B, a number of interacting events probably contribute to the final outcome. (For recent summaries see Streilein 1996 and Strickland and Kripke 1997). In addition, interest is now focused on chromophores that (perhaps through oxidation) alter cell membrane components (affecting internal cell signal transduction pathways (Devary et al., 1993), as well as on provitamin D_3 that, through its active metabolite [1,25-dihydroxivitamin D_3 (Müller and Bendtzen, 1996)] may become immunosuppressive.

Much of the interest in the roles of DNA damage and UCA isomerization in immune suppression relates to the fact that these events affect the production or expression of a variety of biologically active chemicals that can modify immune reactions. Some are released into circulation, i.e., cytokines, while others are displayed on the cell surface, e.g., cell-surface receptors; however, they are key candidates for the development of interventions.

The most prominent cellular target involved in the immunosuppressive action of UVR appears to be the Langerhans or antigen-presenting cell (APC). Large numbers of APCs reside in the epidermis and act as the skin's security force, catching and processing foreign intruders, e.g., antigens, microorganisms, then migrating to the draining lymph nodes to activate the T lymphocytes that will mount the final immune response. Like cis UCA, UV irradiation diminishes the number of Langerhans in the epidermis, and disturbs the proper priming of T cells, often leading to the generation of suppressor T cells that can specifically block the development of an effective immune response against the invading agent. These suppressive cells induce a lasting tolerance toward the invading agent (i.e. the immune system is rendered 'blind' for this specific agent). Recent research suggests that for these aspects of UV-induced immunosuppression: two cytokines, TNF-α and IL10 are responsible for the induction of the transient and persistent tolerances, respectively (Niizeki and Streilein, 1997)

Finally there is good evidence that susceptibility to UV-induced immunosuppression is under some degree of genetic control. Animal experiments initially demonstrated certain mouse strains to be resistant to UV-induced immunosuppression(Streilein and Bergstresser, 1988; Yoshikawa and Streilein, 1990); subsequent work indicated that this distinction was based on relatively high doses of antigen, with lower doses all animals became susceptible (Yamawaki et al., 1997). Humans also show differences in sensitivity to local suppression of the CH response (Cooper et al., 1992) which are largely independent of skin pigmentation. In fact, pigmentation provides surprisingly little protection against UV-induced immune suppression (Screibner et al., 1987) These findings suggest that vaccination programs carried out under conditions of high UV may want to evaluate their dosage regimens carefully in order to avoid providing doses that induce tolerance instead of immunity.

Infectious Diseases

Because of the experimental evidence that UV affected cellular immunity, concern arose about the implications of UV-induced immunosuppression for infectious diseases. Cellular immune responses are of paramount importance in the defense against a wide variety of infections. The 1903 Nobel laureate Niels Finsen first reported that UV radiation could heal skin tuberculosis (Lupus vulgaris) but

adversely affected smallpox (Finsen, 1901), and others later found that lung tuberculosis was also adversely affected. Much more recent work using animal models of human diseases has confirmed that UV-B can affect different infectious diseases (and even differing manifestations of the same diseases) differently, as well as indicated that certain diseases, e.g., schistosomiasis (Noonan and Lewis, 1995), are unaffected.

Human infectious diseases that in animal models have shown an effect of UV-B include herpes, tuberculosis, leprosy, trichinella, candidiasis, leishmaniasis, listeriosis, and Lyme disease. Reported effects have included suppression of immune responses to the organisms or their antigens, reactivation of latent infections, increased body burdens of organisms, decreased resistance to re-infection and reduced survival (Howie et al., 1986; Giannini and DeFabo, 1987; Denkins et al., 1989; Jeevan and Kripke, 1993; Goettsch et al., 1994).

UV-B has been shown to activate viruses such as herpes, HIV and HPV (Spruance, 1985; Perna et al,. 1987; Zmudzka and Beer, 1990) as well as affect the immune response to herpes (Norval and el-Ghorr, 1996). In animal models of human tuberculosis, leprosy, listeriosis, trichinosis and Lyme disease, UV-B treatments suppressed DTH responses to the organisms, depressed clearance of the organisms, and in certain instances, increased mortality (Jeevan and Kripke, 1993, Goettsch et al., 1994; Goettsch et al., 1996).

The information summarized above has raised concerns that UV-induced immune suppression could adversely affect the course of some infectious diseases in human populations. However, with the exception of HIV infection, there appear to be no recent published studies that have explored this issue through epidemiologic analysis. In the case of HIV infection, a recent report from the Multicenter AIDS Cohort Study (Saah et al. 1997) found no evidence that solar UV exposure exacerbated HIV infections in white homosexual males. Indeed, it was found that in men who were HIV positive at baseline, those who purposely sought sun exposure were less likely to have progressed to AIDS. As these authors noted, however, these findings need to be confirmed in a large, prospective study of HIV-infected individuals before any conclusions are made as to the beneficial effects of solar exposure on AIDS progression (Saah et al. 1997).

Lupus and other Autoimmune Diseases

The impact of UV-B exposures on APCs, and on the production and release of cytokines, opens up the possibility that increases in UV-B could either exacerbate or ameliorate autoimmune diseases. Data exist in support of both possibilities and seem to suggest that diseases likely to be exacerbated will be those involving aberrant humoral immunity, e.g., systemic lupus erythematosus (SLE), whereas those likely to be ameliorated will be those involving aberrant cellular immunity, e.g., multiple sclerosis (MS) and psoriasis. In the case of SLE it has long been known that UV-B exposures can exacerbate certain symptoms, probably in part due to the production of TNFα (McGrath et al., 1994).

More recent studies of SLE, however, also suggest that exposures to UV-B could not only exacerbate SLE, but might even serve as initiating events. First, studies of SLE have demonstrated a spectrum of APC and T-cell defects consistent with loss of the cytokines that help regulate cell mediated responses (Lucey et al., 1996) — exactly what is observed following UV exposures (Ullrich, 1995; Boonstra and Savelkoul, 1997). The loss of cytokines also removes a regulatory constraint on the cells important to humoral immunity, thereby leading to an increase in the production of antibodies of the type implicated in the pathogenesis of SLE (Ullrich, 1995). Second, although the SLE antigens are mostly derived from internal components of the cell (e.g., the nucleus and the cytoplasm), the autoimmune response that is made to them is of the type normally reserved for components found outside

the cell or on the cell membrane. This paradoxical finding may be explained by a recent observation that several of the nuclear and cytoplasmic autoantigens important to SLE pathogenesis are found on the surfaces of dying (apoptotic) skin cells, but not normal cells. If this means that these antigens are now presented to APC as if they were membrane components (Koh and Levine, 1997), this could explain the anomalous immune response seen in SLE. Since induction of apoptosis in keratinocytes is a consequence of UV exposure (Aragane et al.,1998), increases in UV could provide greater opportunities for encouraging an SLE type immune response.

In the case of MS, a degenerative disease of the nervous system with autoimmune characteristics, it has long been recognized that there is a latitude gradient for the disease, with incidence increasing with increasing distance from the equator (exactly the opposite of what is found for skin cancer.) More than a decade ago, it was proposed that this could be explained as a result of the immunosuppressive effect of sunlight, and cross-reactivity between melanocyte antigens and those of the nervous system important to MS development (Sharpe, 1986). Subsequent work has strengthened the hypothesis that a sunlight-mediated immunosuppression may explain the latitude gradient, although it is thought now that the important antigen(s) are viral proteins that are cross-reactive with myelin basic protein (McMichael and Hall, 1997).

Effects on the Skin

Sunburn

Sunburn is probably the most widely experienced form of acute solar damage to the skin. A reddening of the skin, which in severe cases leads to blistering, sunburn is a delayed erythema initially characterized by the appearance of ‘sunburn cells’, the depletion of APCs, and the infiltration of the epidermis and dermis by a variety of inflammatory cells, e.g., mast cells, monocytes and lymphocytes. Subsequently the skin responds with hyperproliferation. (Garmyn et al., 1995). Sensitivity to sunburn varies based on pigmentation, with heavily pigmented individuals generally being much less sensitive than lightly pigmented ones.

Sensitivity to sunburn, along with tanning ability, has been used to develop a classification system of six skin phototypes. The most sensitive individuals (skin type I) develop a moderate to severe sunburn after a short term (an hour or less) exposure in the summer, rarely tan even after repeated exposure and generally have very fair, often freckled skin, red or blond hair, and blue eyes. The most resistant individuals (skin type VI) are darkly pigmented without exposure and become even more deeply pigmented upon exposure (Pathak et al. 1985). These classes have been widely used to classify individuals within a population according to skin type; from such efforts have come the conclusion that sensitivity to sunburn is a risk factor for skin cancer (discussed in more detail below).

Photoaging

Aging of the skin is a well-documented consequence of exposure to sunlight. Characterized by wrinkles, altered pigmentation and loss of elasticity, such ‘photoaged’ skin is associated with an overgrowth of abnormal elastic fibers in the dermis, and a decrease of collagens (Kligman and Kligman, 1993). Reactive oxygen species (ROS) have been implicated in the etiology of photoaging (Scharffetter-Kochanek et al., 1997); however, mechanistic studies suggest that UV-B is much more effective than UV-A at inducing the over-production of precursor molecules (Kut et al., 1997). Furthermore, the combination of UV-B and UV-A, was slightly less effective than UV-B alone in the induction of

these effects (Kut et al., 1997). Since UV-A is generally considered more effective at ROS generation, these results suggest that the exact mechanism of these effects is yet to be determined. Given these uncertainties, photoaging must be considered one of the effects that may increase with stratospheric ozone depletion.

Skin Cancer

Among light-skinned populations, skin cancers associated with exposure to solar radiation are the most common kind of cancer. Three types of skin cancers comprise this group: basal cell carcinoma (BCC), squamous cell carcinoma (SCC), and cutaneous melanoma (CM). [The first two are often collectively referred to as non-melanoma skin cancer (NMSC).] BCC and SCC both result from the malignant transformation of keratinocytes, the major structural cell of the skin. CM on the other hand, results from the malignant transformation of melanocytes, which are the skin's pigment producing cells.

These tumors share some traits and differ in others. Shared traits include an increased risk of developing these tumors with increased exposure to sunlight, and a pigment-related variation in susceptibility, with darker skinned races and individuals being at less risk of developing skin cancer for a given amount of exposure than those with lighter skin. As discussed in detail below, although these tumors share certain features, their biologic behavior and relationships to solar exposure are qualitatively and quantitatively different; thus, they are discussed individually.

Epidemiological studies are a primary source of information for assessing the relevance of solar UV exposure to the etiology of diseases such as skin cancer. Even though there has been considerable improvement in study design, epidemiologic findings in this research area still suffer from two principal flaws. First, because of the lack of an independent metric of exposure, e.g., a biomarker of cumulative UV exposure, epidemiologic studies currently base their exposure estimates on patient and control recall of exposure much of which occurred decades ago, thus are subject to 'recall bias'. Second, there are inevitable correlations between what may be independent risk factors, e.g., sun sensitivity is directly related to the numbers of actual sunburns or the use of sunscreens. These in turn relate not only to intermittent exposure but also to sun seeking behavior that also increases the overall level of exposure.

Basal Cell Carcinoma

BCC is the predominant form of NMSC among white populations (Gallagher et al.,1995a); in the US it represents about 80% of the NMSC diagnosed (Epstein, 1996). Susceptibility varies according to skin type, with the most sensitive individuals being those with fair skin and a marginal to poor tanning ability (i.e., skin types I and II). In contrast to SCC and CM, BCC has no unique precursor lesion, although recent studies have identified a gene associated with nevoid BCC syndrome, which is associated with susceptibility to BCC in the general population. Alterations in this gene (termed 'patched') occur in 90% of BCC examined, and about 50% of these alterations are mutations caused by UV-B radiation, based on the pattern of base changes in this gene. This provides direct molecular evidence for a role of UVB in BCC.

The incidence rate of BCC among various white populations has been increasing recently, albeit more significantly in some locations than others. In Albuquerque, New Mexico, an evaluation by a major health care provider of the change in age-standardized rates of BCC between 1978 and 1991 found an increase of about 13% per year (Hoy, 1996)(SCC showed little change in rate.) In contrast, a survey done in Australia showed only about a 2% annual change between 1985 and 1990.

Because early studies in the U.S. showed that the majority of BCC (almost 80%) occurred on the most heavily exposed sites (head, neck and extremities), the risk of BCC was once thought to be directly related to life-time cumulative sunlight exposure (Scotto et al., 1981). However, recent epidemiologic data suggest that this conclusion is too simplistic. In two recent epidemiologic studies from Australia, Kricker and her colleagues (1995a, b), examined several interesting aspects of BCC. In the first report these authors found that the risks of BCC occurring on heavily exposed sites (head, neck and extremities) decreased with increasing total exposure, whereas the risks of BCC occurring on an intermittently exposed site (the trunk) showed the opposite pattern: increasing risk with increasing exposure. In the second report these authors concluded that intermittent exposure, especially in youth (between the ages of 15-19), may be important explaining BCC. These data are consistent with two hypotheses. Kricker et al.(1995a) suggest there is a plateau in BCC risk at higher levels of exposure, in accord with that postulated by others who had observed a similar pattern (Vitasa et al., 1990). Gallagher et al. (1995a) suggest that it is only childhood exposures that are important. Unfortunately distinguishing between these two hypotheses is difficult, since a majority of an individual's life time UV dose is accumulated by age 18 (Stern et al. 1986), and operationally they are the same: one represents a time threshold and the other a dose threshold.

Kricker and her colleagues (1995a, b) also looked at BCC risk based on skin type and found differing dose response relationships for those who tan well as compared to those who tan poorly. For good tanners, the risk of BCC increased with increasing sun exposure, whereas for the poor tanners the risk was initially flat and then fell with increasing exposure. Assuming the plateau hypothesis is correct, these observations suggest that good tanners get a lower effective dose per hour of solar exposure (probably due to the effect of tanning and skin thickening) than the poor tanners and thus their keratinocytes achieve this plateau later in their lifetime. The presence of a plateau also suggests that in order to reduce risk, particularly in those who tan poorly, substantial reductions in exposure will be required.

Squamous Cell Carcinoma

SCC, although much less common than BCC, is almost an order of magnitude more common than CM in the US. In the Netherlands, however, the ratio is almost 1:1. More than any other skin cancer, epidemiologic data on SCC implicate lifetime cumulative UV exposure as a critical risk factor.

In line with earlier studies, the multicenter Helios study in southern Europe showed that the risk of SCC goes up with lifetime exposure (Rosso et al., 1996). A smaller, but very thorough, Canadian study (Gallagher et al., 1995b) on 180 SCC patients treated in 1983 and 1984, did not find any significant increase in risk related to lifetime exposure, but rather, increased risk was associated with chronic occupational exposure over the decade prior to diagnosis of the tumor. As the authors point out, these findings may be hampered by random errors in recall and by a correction for body area exposed. Besides introducing a new source of error, the correction for exposed body area may not be appropriate for SCC since risk is primarily determined by the genotoxic damage at the site of occurrence. That persistent UV exposure in the final stages of tumor development is important can be inferred from the observation that avoiding sun exposure, including through the use of sunscreens, reduces formation of actinic keratoses, precursor lesions of SCC (Thompson et al. 1993). The Canadian study also found an increase in risk in relation to regular overexposure (sunburns) during childhood. The latter correlation with childhood sunburns has also been found in other studies (Kricker, 1992;1992, Grodstein, et al., 1995). However, as pointed out above and by Armstrong and his colleagues (1997), this may primarily imply that a high level of childhood exposure, rather than sunburns *per se,* is the dominant risk factor. Considering the presently available epidemiological data,

it appears prudent to conclude that UV exposure contributes to SCC risk both in early and late stages of tumor development.

Brash (1991) and his colleagues (Ziegler et al., 1993) found UV-related mutations in the *p53* tumor suppressor gene in the majority of SCC as well as BCC. In addition, this group found that about 60 % of actinic keratosis (AK, precursor lesions of SCC) bear such mutations (Ziegler et al., 1994). These mutations were also found in experimentally UV-B-induced murine skin cancers, first in low percentages (Kress et al., 1992) but later in the majority (Kanjilal et al., 1993;1993, Dumaz et al., 1997). In samples from normal skin of the shoulders of Australians who had skin cancers removed, cells with a specific UV-related *p53* mutation could be detected, while they were virtually absent in unexposed buttock skin (Nakazawa et al., 1994). In mouse experiments, microscopic clusters of cells with high levels of mutant p53 protein were observed long before the UV-induced macroscopic skin tumors appeared (Berg et al., 1996). Such microscopic clusters have also been found in human skin from surgical resections (Ren et al., 1996;1996, Brash et al., 1997). Hence, it appears that mutations in the *p53* gene occur at a very early stage of the development of SCC (in contrast to other tumors, like colon cancer, where it marks a late conversion to malignancy).

In addition to earlier experiments in which SCC were induced in hairless mouse skin by chronic UV-B exposure, it has recently been reported that SCC can be similarly induced in normal human skin grafted onto immune deficient mice (Nomura et al., 1997). These experiments clearly show that the human skin taken out of its own environment can develop AK and SCC within 1 to 2 years of daily UV-B exposure, although the yield of frank SCC was low. This may provide an important new model to investigate quantitative and wavelength relationships between skin cancer induction and UV exposure.

Cutaneous Melanoma

CM differs significantly from NMSC in terms of incidence, biologic behavior and relationship to UV-B. CM is a far rarer tumor and is generally much more aggressive than either BCC or SCC. Although in the US, CM probably accounts for only 2-3% of those skin cancers associated with solar exposure, it accounts for most of the mortality.

As with BCC, the risk of CM does not appear to be directly linked to cumulative, lifetime UV exposure. CM frequently occurs at anatomical locations that are not the most heavily sun-exposed. Furthermore, as noted in a recent review of almost 30 epidemiologic studies, an increased risk of melanoma is associated principally with an increase in intense exposures of the intermittent type, e.g., such as those received by areas exposed only during outdoor recreational activities (Elwood and Jopson, 1997).

There is also considerable evidence that exposures in early childhood area may be important. A number of epidemiologic studies have also found higher melanoma risk with increasing sun exposure in individuals who lived in sunny areas during their childhood (Holman et al., 1984; Autier and Dore; 1998). The latter authors also found that sun exposure during childhood may in some instances constitute a significant risk factor for melanoma only if there is substantial sun exposure during adult life, i.e., that childhood and adult exposures act interdependently.

An additional risk factor for CM revealed by epidemiologic studies is the presence of one or more forms of pigmented lesions on the skin - either freckles or moles (which are also known as melanocytic nevi). In a number of studies, prevalence of nevi was the single most important determinant of melanoma risk, a finding that was later confirmed by clinical studies (Gallagher and McLean, 1995). Pathological examination of melanomas frequently reveals histologic evidence of a

preexisting nevus (USEPA, 1987), leading to the suspicion that at least some nevi may be precursor lesions for melanoma. Since nevi exist in a gradient of premalignancy, with the common melanocytic nevus being the most benign and the dysplastic nevus being the least benign, some effort has been spent in evaluating the relative risk associated with various kinds of nevi. In a number of well-conducted epidemiologic studies, it has been found that having a large number of moles is associated with a higher risk of melanoma (Bataille et al., 1998; Berwick, 1998).

As shown in Fig. 2.3, a recently described action spectrum for melanoma in fish (Setlow et al., 1993), unlike that developed in mice for SCC (deGruijl et al., 1993) (and the rest of the action spectra in Fig. 2.3, appears to have a strong UV-A dependence. Work in the South American opossum provides some support to the notion that exposure to UV-A may be important to CM. In that animal model UV-A treatments alone induced melanocytic hyperplasia, a precursor to melanoma in these animals (Ley, 1997). To date, however, these lesions have not progressed to malignant melanoma. These findings suggest that the etiology of melanoma is probably complex and likely involves a multistep process of both UV-B and UV-A induced changes in a variety of different molecules (Longstreth 1998).

Hazards for Domestic Animals

SCC associated with ambient solar exposure has been reported in cattle, horses, cats, sheep, goats and dogs (Hargis, 1981; Mendez et al., 1997;Teifke and Lohr, 1996) These tumors occur principally in poorly pigmented skin unprotected by hair, and thus are frequently found on eyelids, nose, ears, tails, and the mucocutaneous junctions of the eyes and anogenital regions (Teifke and Lohr, 1996). The incidence of these tumors is generally very low, although occasionally herds of susceptible cattle or sheep can demonstrate incidences as high as 20 per cent (Mendez et al., 1997).

Other effects in domestic animals that may increase under ozone depletion include exacerbation of infectious bovine keratoconjuntivitis in cattle (Kopecky et al., 1980), and skin lesions and cataract in farm-raised fish (Mayer 1992), both of which have been associated with significant economic losses. Concerns have also been raised with regard to cataract and eye infections in sheep herds raised under the ozone hole in Chile; however, an investigation into this issue found no evidence to support such a concern (Schein et al., 1995)

Factors Modifying Exposure and Susceptibility

As intimated above, the risks to humans and domestic animals of developing the effects described above depend on a number of factors besides the ambient UV exposures, including such things as the degree of skin pigmentation, sun-seeking behavior, age at exposure, etc. This section briefly reviews what is known about a number of these factors because of the importance of this information to the development of risk management strategies.

Genetics

A number of genetic differences have been described that influence susceptibility to the adverse effects of solar exposure. These include variations in genes determining: 1) quantitative and qualitative differences in pigmentation, 2) the repair of UV-induced damage to DNA and other molecules, 3) the ability to make an immune response to certain types of antigens, and 4) the expression of oncogenes

or growth promoting substances, e.g., cytokines. In some cases, these variations may lead to differences in the dose of UV-B delivered to the target cell, whereas others may influence the kinds and amounts of damage, and still others, the repair or the consequences of damage. To date, no single genetic change has been identified that confers absolute susceptibility to these effects, rather each of these genes appears either to demonstrate a number of different alleles or a variety of different mutations which are associated with greater or lesser responses to the effects of UV-B. Furthermore, it is becoming clear, at least in the case of skin cancer, that multiple processes must be compromised in order for adverse effects to occur.

One of the most overarching sets of such genetic differences is that conferring high or low degrees of pigmentation. Numerous studies have indicated that, in general, those of Negroid ancestry show a very much lower incidence of skin cancer (100 fold for NMSC, 10 fold for CM) than those of Caucasoid ancestry (Scotto et al., 1981). A more recent series of reports from Hawaii extended this observation to other races and found that Caucasians on the island of Kauai have about a 10 times higher incidence of NMSC compared to those of Japanese ancestry, who in turn have a five fold higher incidence than those of Philippine or Hawaiian ancestry (Reizner et al., 1993; Chuang et al., 1995). Interestingly, a similar degree of protection is not conferred by pigmentation for either for cataract (Hiller et al., 1983) or the immunological effects of solar exposure (Screibner et al., 1987).

Among the light-skinned races, certain qualitative differences in pigment also appear to be important to sun sensitivity, as well as to skin cancer risk. In particular, those with higher ratios of pheomelanin (the yellow/orange melanin found in red hair) to eumelanin (the brown/black melanin of brown/black hair) who rarely tan and almost always burn, appear to be at greatest risk (Gallagher et al., 1995a, 1995b, Holman et al., 1984b). Pheomelanin is known to generate reactive oxygen species (ROS) when irradiated with UV-B, whereas eumelanin appears to act protectively against ROS; it has been postulated that this difference may be the reason for the increased susceptibility to skin cancer of those with fair skin and red hair (USEPA 1987). Recent information suggests however, that multiple factors are important to susceptibility, and that different genes may be important to poor tanning ability and UV sensitivity. In the case of poor tanning ability, it has been shown that people with a poor tanning response show variations in a gene important to eumelanin synthesis in melanocytes (Valverde et al., 1995). Furthermore, some of these genetic variations may be associated with increased risk of melanoma (Valverde et al., 1996). In the case of sensitivity to the erythemal effects of UV-B, it has recently been shown that individuals showing the greatest inflammatory response to UV-B exposures lack a key detoxification enzyme for ROS (Kerb et al., 1997).

Another set of genes important to understanding susceptibility to the effects of solar exposures are those associated with repair of UV-B-induced alterations in DNA. Patients with xeroderma pigmentosum (XP), a rare genetic disease characterized by poor repair of UV-induced DNA damage, have a 2000-fold increased risk of developing skin cancer before the age of 20 (Kraemer et al., 1984). It has been suggested that many skin cancer patients suffer from similar albeit much less severe defects in DNA repair (Alcalay et al.1990; Wei et al., 1993); however, these findings have not been universal (Hall et al., 1994) and require additional investigation.

One interesting finding with regard to repair deficiency syndromes and skin cancer is the discrepancy observed between two such syndromes in the development of skin cancer. Trichothiodystrophy (TDD) is a DNA repair deficiency syndrome with many similarities to XP; indeed one form of XP, XP-D, has mutations in the same gene as that affected in TDD. However, only XP-D individuals are at increased risk of skin cancer. An explanation for this anomaly may be that cells from XP-D patients, but not from TDD patients, are more susceptible to one of the steps in UV-B induced immunosuppression (Ahrens et al., 1997). Thus, in addition to a faulty DNA repair process,

XP-D patients also have a compromised immune response. The conclusion from this study is that faulty DNA repair alone may not be sufficient to cause the observed increase in skin cancer, it may need to be accompanied by a compromised ability to respond to UV-induced tumorigenesis (Ahrens et al., 1997).

Two additional sets of genes which appear to be able to influence the development of UV-B induced neoplastic responses are 1) those which affect the immune response and 2) those which act as growth regulators, either stimulating uncontrolled growth, e.g., proto-oncogenes, or restraining such growth, e.g., suppressor genes. In the case of those that influence the immune response, as demonstrated by the finding with XP-D patients, compromising the immune response to UV-induced tumors. In the case of growth regulators, a host of genes have been identified which when mutated (by UV or another insult), can result in the development of a tumor. Chief among these is the gene for p53 that has been discussed above in some detail. Mutations in p53 appear to be key to the development of both BCC and SCC, but not CM (Brash et al., 1996). Another gene, that which codes for cyclin dependent kinase inhibitor 2 (CDKN2) or p16, has been found to be very important in CM. Recent information suggests that CDKN2 is a melanoma tumor suppressor gene located on chromosome 9 which is particularly important to familial melanoma but may also have a role in sporadic melanoma (Naylor an Everett, 1996; Fountain 1998).

Behavior

There are a number of behavioral choices that can significantly affect the risks associated with ozone depletion. The largest of these is undoubtedly ‘sun-seeking’ behavior. Numerous epidemiologic studies have demonstrated the importance of various exposure patterns. Thus high cumulative exposure is a risk for SCC and many of the ocular effects, most notably cataract (Gallagher et al. 1995a ; Hodge et al., 1995). Childhood and intermittent exposures, particularly those leading to sunburns, appear to be important to BCC and CM (Gallagher et al. 1995a; Holman and Armstrong, 1984a,b; Berwick, 1998), and intense exposures appear to be important for sunburn, melanoma, BCC, snowblindness, pinguecula, CDK, and pterygia (Longstreth, 1998, Kricker et al., 1995a, b;Mackenzie et al., 1992). Clearly those who avoid such behaviors will reduce their risk. Such avoidance can be achieved a number of different ways, e.g. modifying time of exposure, avoiding exposure during the peak solar hours (10 am to 2 pm in the Northern hemisphere); wearing protective clothing such as hats, sunglasses and densely woven materials; staying in the shade and off of highly reflective surfaces, foregoing sunny vacations.

Diet

Numerous studies have explored the impact of various nutritional variable on the expression UV-associated adverse effects. Information has come from experimental as well as epidemiologic studies, however, for the most part what appears to be clear cut in experimental systems is found to be far from clear-cut in epidemiologic studies. In the case of cataract for example, Varma et al. (1995) indicate that the experimental evidence for the protective effect of antioxidants for cataract is quite compelling, In contrast, a parallel review of the epidemiologic evidence by Hodge et al. (1995), indicates that the information is difficult “to unravel.” These latter authors conclude that nutrition is clearly important in the case of nutritionally deprived communities, but also conclude that these findings are difficult to generalize to more affluent communities because the relevant studies provided conflicting results. Even in nutritionally deprived populations, however, the protective role of adequate nutrition in the form of adequate protein consumption or additional nutritional supplements (only the ribofla-

vin/niacin complex demonstrated any effect) did not apply to cortical cataracts, the major form of UV-induced cataract (Hodge et al., 1995).

In the case of skin cancer, the epidemiologic evidence also appears to be somewhat conflicting. A summary of the information on NMSC presented in one recent review indicates that while one study found a protective effect of dietary factors, other studies found no significant benefit (Strom and Yamamura, 1997). In the case of melanoma, a number of different studies have examined either the consumption or serum levels of vitamin E, α-tocopherol, or β-carotene consumption and related them to risk. The results have been highly variable; e.g., in the case of consumption, vitamin E in foods, but not in supplements, was protective (Berwick, 1998). In the case of serum levels, some results suggest that low serum concentrations of α-tocopherol, or β-carotene were associated with higher risk (Armstrong and Kricker, 1995) whereas others studies found that plasma levels of α-tocopherol were not related to risk. Taken collectively, these results suggest that dietary interventions may be of little help in preventing or managing the risks of cataract and skin cancer from UV-B.

Medical Treatment/Status

A number of factors related to health status have shown an association with increased risks from UV exposures. From a risk assessment perspective, these factors often identify sensitive subpopulations whose reactions may occur earlier or to a higher degree than a normal population thus providing information helpful to the understanding of mechanisms needed for risk management. From a risk management perspective, such populations may require special handling in the development of appropriate management strategies.

Given the importance of the immune response to the development of skin cancer, it was hypothesized early on that immunosuppression would have an impact on tumor development. Studies in renal transplant patients (whose immune response are suppressed in order to prevent rejection of a kidney transplant) confirmed this hypothesis by revealing a dramatic increase in warts and SCC on sun-exposed skin of these patients (Harteveld et al.,1990). The warts are known to be associated with human papilloma viruses (HPV), but the carcinomas and precursor lesions in these patients were also found to bear a great variety of HPV (Tieben et al., 1994), not generally found in SCC (Kawashima et al.,1990). It has, however, recently been found that HPV are commonly detectable in hair plucked from eye brows (Boxman et al., 1997). The question now is whether the HPV is merely a hitchhiker in proliferating carcinoma cells or whether it really plays a causal role in the development of these tumors. The skin carcinomas in people with renal transplants were also found to contain UV-related p53 gene mutations (McGregor et. al., 2997). These findings clearly show that a good immune system prevents the development of potential carcinomas on sun-exposed skin. As discussed above, the UV radiation from the sun can also exert immunosuppressive action and thus enhance the development of skin carcinomas.

Besides SCC, non-Hodgkin's lymphoma occurs much more frequently in people on immunosuppressive medication (Deeg et al., 1993). The risks of non-Hodgkin's lymphoma and skin cancer appear to be associated; people who were treated for skin carcinomas have an increased risk of non-Hodgkin's lymphoma (Frisch and Melbye, 1995; Frisch et al., 1994) and vice versa, i.e., people treated for non-Hodgkin's lymphoma have an increased risk of skin cancer (Adami et al., 1995). The persistent increase in non-Hodgkin's lymphoma over the last 4 to 5 decades parallel increases in skin cancer incidence and it is hypothesized that both these trends are due to increased exposure to sunlight (Cartwright et al., 1994). It has been speculated that the common factor in the etiologies of these cancers is the UV-induced immunosuppression (Goldberg, 1997) or more specifically, that cytokines

(Vos et al., 1994) released upon UV exposure may stimulate the outgrowth of precursor cells of B lymphocytes to develop into a non-Hodgkin's lymphoma (Hirayama and Ogawa,1994). Non-Hodgkin's lymphomas have been reported to occur more frequently in the sunniest parts of Great Britain (Bentham et al., 1996), but in contrast to skin carcinomas, there is no increase in non-Hodgkin lymphomas toward the south in North America (Hartge et al.,1996). Interestingly, mice developed lymphomas as a consequence of exposure to UV radiation and a chemical carcinogen (7,12-dimethylbenz (a) anthracene), whereas treatment with each of these factors separately did not induce lymphomas (Husain et al.,1991). In sum, there appears to be an association between the risk of skin carcinomas and non-Hodgkin's lymphomas, but whether UV radiation is a risk factor for non-Hodgkin's lymphomas is not clear. More data are required to negate or confirm a direct relationship between UV radiation and non-Hodgkin's lymphoma.

The immune system can be compromised in many different ways, e.g. through medication or infections. An additional UV-induced suppression might then have more devastating impact than normal. A very timelytopical example of an affliction that cripples the immune system is AIDS (cause by HIV), and these patients could run an increased risk from UV exposures. However, AIDS patients with psoriasis have been treated with UV radiation and no aggravation of the AIDS was found (Adams et al., 1996; Meola et al., 1993). This result could be offset by the fact that psoriasis itself activates the immune system on which UV radiation then exerts a first dampening effect without further diminishing the resistance to HIV.

Certain medical treatments may add to the cancer risk from (solar) UV exposures, e.g., immunosuppressionradiation therapies for cancer (Berwick, 1998), PUVA, (a combination of UVA and oral dosing with 8-methoxypsoralen) treatments for psoriasis (Stern and Laird, 1994).

The PUVA treatment of psoriasis has become very widely used but has been found to be associated with a substantial increase in skin cancer risk in a long-term follow-up. Recently, it has been reported that in the long run the risk of melanoma is also significantly increased (Stern et al., 1997). The squamous cell carcinomas found on the PUVA-treated individuals occurred frequently on skin areas that are not regularly exposed to the sun but are exposed in the PUVA treatment: e.g., like the legs, where they do not commonly occur in the general public. Curiously enough, many of the skin carcinomas taken from PUVA-treated patients were found to have mutations in the *p53* gene that pointed at UVB radiation instead of the PUVA treatment as the direct cause (Nataraj et al. 1997). This would indicate that UVB radiation may even have contributed to the development of these clearly PUVA-related skin tumors. Clearly, there are a number of factors that can amplify the risks from UV exposure, and vice versa. Identifying high-risk populations will open up the possibility for well-targeted mitigating strategies.

Risk Assessment

Quantitative risk assessment of the effects of ozone depletion is a data intensive process which until recently has only been done for a few effects, most notably those affecting human health for which there are adequate data, i.e., cataract and skin cancer. With the exception of the 1989 USEPA risk assessment (1987) and regulatory impact assessment (RIA) (USEPA, 1988), the impact of increases in ambient UV-B on these diseases has previously been quantified principally by comparing two stationary situations: one with an ozone concentration constantly at a normal level and the other constantly at a lower level (UNEP report 1994, Longstreth et al., 1995, Madronich and De Gruijl, 1993). The corresponding disease incidences were estimated in two steps from the constant level of increased ambient

UV radiation to the increased incidences: the first step is represented by the radiation amplification factor, RAF= (percentage increase in carcinogenic UV) per (percent decrease in ozone)(see chapter 1), and the second step by the biological amplification factor, BAF= (percentage increase in incidence) per percent increase in carcinogenic UV radiation (De Gruijl and van der Leun, 1994). The overall increase in incidence per percent ozone depletion is then represented by the amplification factor, AF= RAFxBAF.

The future projections of ozone depleting substances in the atmosphere made in recent years have invited scenario studies on future ozone density and corresponding levels of ambient UVR. These in turn are now being translated into assessments of the risks to the biosphere in order to assess the importance of such atmospheric changes. It cannot be over-emphasized (see Chapter 1), however, that these scenario studies should not be taken as genuine forecasts. They are, at best, idealized computations on the effects of the changes in a small subset of factors leaving all other relevant modifying factors undisturbed. In the real world many of the other relevant factors may change and diminish or aggravate the effects (e.g. increased or decreased cloudiness). Nevertheless, these scenario studies serve the purpose of quantifying and comparing the potential effects of certain policies.

As indicated above it is not currently possible to develop quantitative risk assessments for all of the health effects expected from ozone depletion. Presented below, therefore, is a mixture of quantitative and qualitative information that assesses to the extent possible, the likely impacts of ozone depletion on human health.

Cataract

In the Chapter 2 of the 1989 UNEP Environmental Effects Panel Report (van der Leun et al. 1989), a static estimate was developed of the cataract risk of ozone depletion. In that effort it was estimated that the world’s population, if subjected to a sustained 1% decrease in the ozone layer, would develop between 100,000 to 150,000 additional cases annually. More recently, the USEPA has updated the work developed for their earlier RIA (USEPA 1988), using a quantitative model that incorporates the ozone depletion scenarios developed by the Scientific Assessment Panel (UNEP 1998). Presented in Fig. 2.6 are the results of that effort (R. Rubenstein, personal communication). Although these estimates were developed on the basis of U S data, they should be applicable to similar populations, i.e., those that are adequately nourished, worldwide. As discussed above, under-nourished populations may be a greater risk.

Sunburn

Exposure to sunlight may lead to a reddened and painful skin. This 'sunburn' is mainly caused by the UV-B radiation in sunlight. Exposures to more UV-B give more severe sunburns. An increase of sunburns by ozone depletion would be more than a nuisance; sunburn is also considered to be a risk factor for more serious effects, such as melanoma.

Analysis of available knowledge leads to the conclusion that sunburns will not appreciably increase under a decreasing ozone layer; this is due to a powerful adaptation of the skin (van der Leun and de Gruijl, 1993). A gradual thinning of the ozone layer would, for instance, lead to 20 percent more UV-B in 10 years' time. The skin is equipped with an adaptation that can even cope with the changes in UV-B with the seasons. These are much more drastic; in mid-latitudes, the UV-B irradiance in summer is typically 10 times larger than in winter.

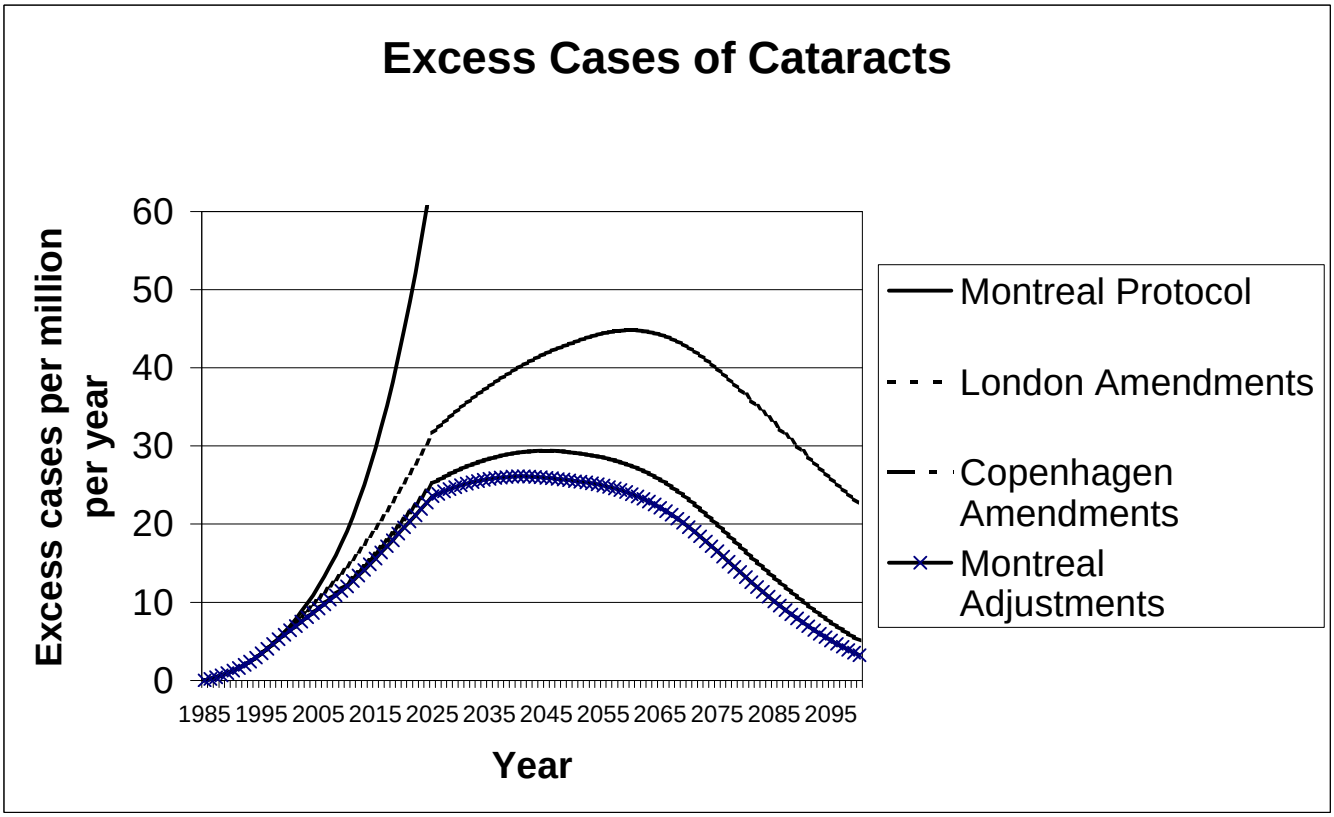

Fig. 2.6 Cataract risk estimates based on various scenarios.

Experience with phototherapy of skin diseases shows that one UV-B exposure, sufficient to cause a slight reddening, decreases the sensitivity of the skin by about 20 percent. In a series of exposures, this can be repeated many times. That is how the skin adapts to the UV-B changes with the seasons. A calculation shows that adaptation from winter to summer irradiance requires 13 such steps of 20 percent each. This will not change much under a UV-B irradiance increased by 20 percent due to ozone depletion. It will in fact become a bit easier, as the winter irradiance increases more than that in summer, so that the difference becomes a bit smaller.

It is certainly possible to think of situations where adaptation cannot work in this way. For instance, if a totally unadapted skin is suddenly exposed to full sunlight, more UV-B in the sunlight will increase the likelihood of sunburn. Persons going on an expedition to the Antarctic ozone hole have reported experiences in this line. But such conditions are quite exceptional. By far the most sunburns arise from lack of care in going through the adaptation process. Such sunburns will not increase.

Skin Cancer

Using the process described above, the amplification factors for SCC and BCC have been determined to be 3, and 1.7, respectively. As discussed above in the section on BCC, the AF for SCC has a greater degree of certainty than that for BCC. Melanoma, because of uncertainties in its action spectrum could have an RAF equivalent to that of the carcinomas (1.2) or be closer to 0.1 (if the process is mainly UV-A-driven). A third possibility is that the development of melanoma can involve at least two different UV-driven processes, each with a substantially different wavelength dependence: e.g. UVA-driven initiation of transformed cells and UVB-driven immune suppressive episodes that promote the development of the tumor, or vice versa. Thus any quantitative model for the UV induction of mela-

noma will have significantly more uncertainty than that for SCC and probably more than that for BCC as well.

Recent risk assessment efforts with a quantitative model that incorporate ozone depletion scenarios from the Scientific Assessment Panel, provides estimates of the additional cancer risks in populations annually based on the estimated changes in UV-B over time (Slaper et al. 1996; Arnold et al. 1998). It should be noted, however, that such efforts are not just a matter of including information on the changing concentration of ozone (and UV-B) with time. There are also a number of issues that need to be addressed with regard to the assumptions chosen for the dose-response models used to approximate the relationship between exposure and effect. The process of disease development has to be dissected in phases (steps) that are either UV-driven or not, and it should be known at which stage in the development (early or late, or both) UV is important. From experimental data and epidemiology, it can be inferred that chronic accumulation of UV exposures is important throughout the development of SCC. In contrast, for BCC and CM, acute intense exposures, particularly those acquired in childhood, may be the critical dose metric, although as discussed above, this may be true in the case of CM only if adulthood exposures are also substantial (Autier and Doré, 1998).

Several groups are developing risk estimates using such scenario-based approaches; unpublished data from two of them developed for this assessment are presented here. Given below in Fig. 2.7 is a summary graphic from the Dutch group (Slaper et al., 1996) Calculations for skin cancer risks are performed for five scenarios applying the UV-chain methodology developed by Slaper et al. 1996, and assuming full worldwide compliance with the agreed protocols within the Vienna Convention.

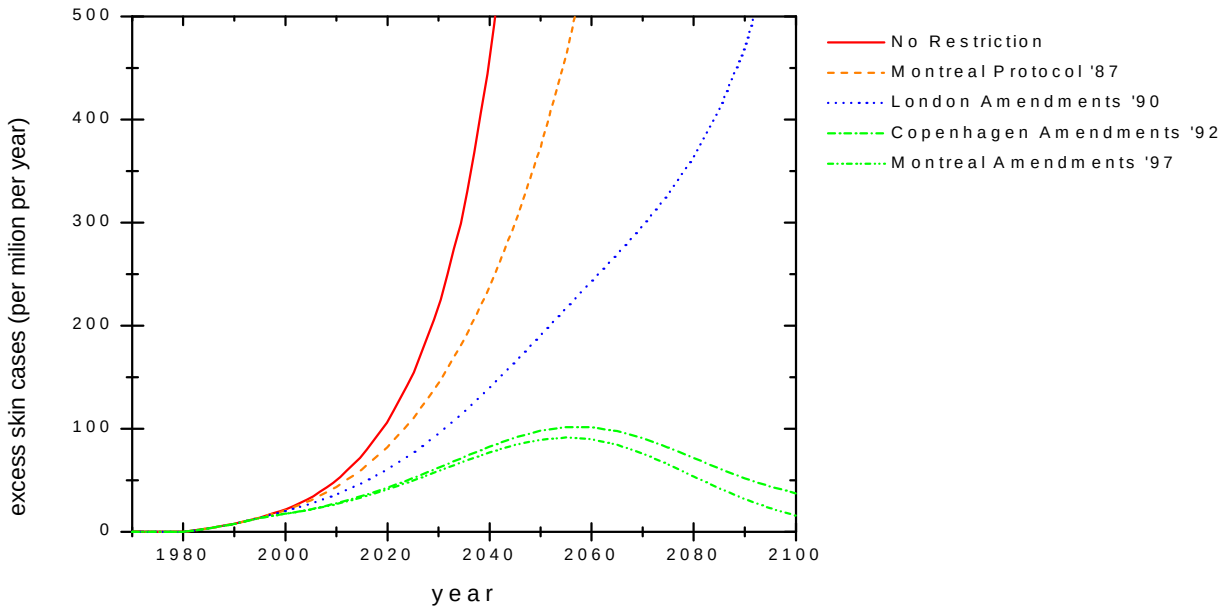

Fig. 2.7 All cancer risk estimates based on various scenarios

The calculations are based on the production and depletion scenarios used in the WMO/UNEP scientific assessment of ozone depletion (WMO, 1998). Skin cancer risks are calculated for the zonal average ozone depletion observed at 45 degrees N (as reported in the 1998 ozone assessment), assuming a population with the sensitivity and age distribution as in the USA (risk in 1980 estimated at 2000 skin

cancer cases per million per year). Excess cases refer to additional cases due to ozone depletion. The majority of the excess cases are nonmelanoma, and the lethality is approximately 2% of the incidence. The risks are probably conservative estimates, because:

- full compliance with restrictions are assumed, throughout the world
- aging of the population will probably increase the excess risks with 60%
- probable increases in exposure due to changes in behavior are not accounted for.

It should also be noted that certain risk-groups (outdoor workers with a fair complexion) probably have much higher excess risks for the non-melanoma skin cancers, and also that in certain areas depletion can be larger than the zonal average used in this evaluation.

Infections

Although it is now adequately documented that UV radiation can modulate immune reactions in rodents as well as in humans, the impact of current levels of ambient solar UV radiation on infections in human populations is still unknown. Currently available epidemiological are unsuited to ascertain and quantify any such effect (De Gruijl, 1997), and given the fact that scientists have been aware of this lack of data for decades, a well-designed epidemiological study that addresses this issue is long overdue. Consequently, we are still completely ignorant when it comes to quantifying possible effects on infections of ozone depletion.

In developing animal models for the effects of UV radiation on infections, investigators have been measuring changes in fundamental immune reactions that are associated with the course of the infection and that may also be measured in humans. Thus, the aim is to predict UV-induced effects on human resistance to infection by measuring the relevant changes in basic immune responses after UV exposure (Goettsch et al.,1998), a so-called 'parallelogram' approach. This approach is in its infancy and requires a thorough and detailed knowledge of the immunological responses that play a role in any particular infection under consideration, in order to identify the relevant measurements. This approach also has certain limitations in that the outcome of such analysis only evaluates host resistance and does not provide complete information on the spread and course of an infection in a population.

The first conjectural calculations demonstrate that physiologically relevant exposures to solar UV radiation (e.g. 90 minutes around noon in July at 40^{o} N) may significant hamper cellular immunity against a bacterial infection (*Listeria monocytogenes*) in the 5 % most sensitive individuals in a population of white Caucasians. This result is in reasonable agreement with direct measurements of the UV-induced suppression immune reactions against simple chemicals (Yosikawa et al., 1990; Cooper et al., 1992), where UVB exposures of the same order of magnitude as those calculated were found to affect a high percentage of people. In spite of these promising developments in indirect methods for assessing UV-related risks of infection, a more direct quantitative assessment of UV-induced enhanced infection remains desirable. A reliable assessment of the magnitude and breadth of effects of current ambient UV levels on infections and on success rates of vaccinations appears to be a long way off, and an expansion to include the effects of an ozone depletion delves even deeper into realm of human ignorance.

Offsetting Risks of Mitigation Strategies

CFC Substitutes

A large number of chemical substances are now being used (or proposed) as substitutes for the ODSs which are being phased-out under the Montreal Protocol and its amendments. Increased usage of such substances will increase human (and environmental) exposures to them and may also increase risks from these compounds to human (and environmental) populations. A complete assessment of the risks of ozone depletion thus needs to include not only the risks associated with increases in UVB, but also the risks from the replacement substances.

It is however beyond the scope of this document to do a complete life cycle (from production, through use and release) risk assessment for these chemicals. First, the list of possible substitutes is growing, second the production and use information for many of these compounds is constantly changing and not generally available and finally with the exception of the more or less well-known substitutes such as the hydrochlorofluorocarbons and other chlorofluorocarbons, the toxicology database for is inadequate. Thus in this section, we present only a qualitative assessment of the toxicity information on some of the better characterized substitutes and indicate where readers can find more information.

All three HFAs, -132b, -133a, and -142b, demonstrate a low acute inhalation toxicity. HFA-132b showed a moderate level of toxicity upon repeated exposure in standard toxicity tests, but demonstrated maternal and fetal toxicity at all concentrations in a reproductive toxicity study. For technical and toxicological reasons it is thus not being actively pursued as an ODS substitute. HFA-133a demonstrated moderately high toxicity to most systems upon repeated exposures with a No Observed Adverse Effect Level (NOAEL) of 24,000 mg/m^3. However, adverse reproductive effects were observed in several studies with a NOAEL recommended as 485 mg/m^3. Company occupational exposure limits have been established between 5 and 24 mg/m^3. HFA-142b demonstrates a moderate level of toxicity upon repeated exposure with a NOAEL for chronic exposure of 82,000 mg/m^3. There is some evidence of cardiovascular sensitization potential but only at very high levels (205,000 mg/m^3) and no evidence of carcinogenic, reproductive or developmental effects. An 8-h occupational exposure levels of 1000 ppm has been proposed.

All three HFCs, -32, -125, and -134a, demonstrated low toxicity upon acute and repeated inhalation exposure in standard toxicologic testing protocols. None demonstrated any reproductive toxicity and only one, -134a demonstrated any fetotoxicity (NOAEL 10,000 ppm in one study and 100,000 ppm in a second which was also the dose at which maternal toxicity was observed.)

All three HCFCs, HCFC-21, HCFC-124, and HCFC-141b demonstrate low acute inhalation toxicity, and HCFC-124 and -141 demonstrate generally low toxicity overall. The recommended occupational standard for these two HCFCs is 1000 ppm. HCFC-21 demonstrates greater toxicity than the other two HCFCs, with liver toxicity and cardiac sensitization at relatively low concentrations (15 ppm and 1000 ppm, respectively.) The recommended occupational standard for HCFC-21 is 10 ppm.

Table 2.1. CFC Substitutes

Common	Chemical name	CAS No	Reference
FC-3-1-10	Perfluorobutane	355-35-9	
FC-5-1-14	Perfluorohexane	355-42-0	
FM-100	Bromodifluoromethane	1511-62-2	
HCFC-21	Dichlorofluoromethane	75-43-4	ECETOC 1990a
HCFC-22	Chlorodifluoromethane	75-45-6	ECETOC 1989
HCFC-124	1-Chloro-1,2,2,2-tetrafluoroethane	2837-89-0	ECETOC 1994a
HCFC-141b	1,1-Dichloro-1-fluoroethane	1717-00-6	ECETOC 1994b
HCFC-142b	1-Chloro-1,1-difluoroethane	75-68-3	
HCFC-143a	1,1,1-Trifluoromethane	420-46-2	
HCFC-152a	1,1-Difluoroethane	75-37-6	EPA 1994
HCFC-225ca	1,1-Dichloro-1,1,2,2,3-pentafluoropropane	505-55-1	
HCFC-225cb	1,3-Dichloro-1,1,2,2,3-pentafluoropropane	507-55-1	
HFA-132b*	1,2-Dichloro-1,1-difluoroethane	1649-08-7	ECETOC 1990b
HFA-133a	1-Chloro-2,2,2-trifluoroethane	75-88-7	ECETOC 1990c
HFA-142b	Chlorodifluoroethane	75-68-3	ECETOC 1991
HFC-23	Trifluoromethane	75-46-4	
HFC-32	Difluoromethane	75-10-5	ECETOC 1995a
HFC-125	Pentafluoroethane	354-33-6	ECETOC 1994c
HFC-134a	1,1,1,2-Tetrafluoroethane	811-97-2	ECETOC 1995b
HFC-227ea	1,1,1,2,3,3,3-heptafluoropropane	43-89-0	
	d-Limonene	5989-27-5	
	Methylene chloride	75-09-2	ECETOC 1984

References

Adami, J., Frisch, M., Yuen, J., Glimelius, B. and Melbye, M. 1995. Evidence of an association between non-Hodgkin's lymphoma and skin cancer. *British Medical Journal* 310:1491-1495.

Adams, M.L., Houpt, K.R. and Cruz, P.D. Jr. 1996. Is phototherapy safe for HIV-infected individuals? *Photochem Photobiol* 64:234-237.

Ahrens, C., Grewe, M., Berneburg, M., Grether-Beck, S, Quilliet, X., Mezzina, M., Sarasin, A., Lehmann, A.R., Arlett, C.F. and Krutmann, J. 1997. Photocarcinogenesis and inhibition of intercellular adhesion molecule 1 expression in cells of DNA-repair-defective individuals. *Proceedings National Academy Sciences USA* 94:6837-6841.

Alcalay, J., Freeman, E.E., Goldberg, L.H. and Wolf, J.E. 1990. Excision repair of pyrimidine dimers induced by simulated solar radiation in the skin of patients with basal cell carcinoma. *Journal of Investigative Dermatology* 95:506-509.

Aragane, Y., Kulms, D., Metze, D., Wilkes, G., Poppelmann, B., Luger, T.A. and Schwartz, T. 1998. Ultraviolet light induced apoptosis via direct activation of CD95 (Fas/APO-1) independently of its ligand CD95L. *Journal of Cell Biology* 140:171-182.

Armstrong, B.K., Kricker A. and English, D.R. 1997. Aetiology and pathogenesis of skin cancer. *Australian Journal Dermatology* 58 (Suppl.):S1-S6.

Armstrong, B.K. and Kricker, A. 1995. Skin cancer. *Dermatologic Clinics* 13:583-594

Arnold, F., Abseck, S., Longstreth, J., Pitcher, H. and Rubenstein, R. 1998. Stratospheric ozone depletion and future U.S. melanoma mortality. Manuscript in preparation.

Autier, P. and Doré, J.-F. 1998. Influence of sun exposures during childhood and during adulthood on melanoma risk. *International Journal of Cancer* 77:533-537.

Bataille, V., Grulich, A., Sasieni, P., Swerdlow, A., Newton Bishop, J., McCarthy, W., Hersey, P., Cuzick, J. 1998. The association between naevi and melanoma in populations with different levels of sun exposure: a joint case-control study of melanoma in the UK and Australia. *British Jounal of Cancer* 77:505-510.

Bentham, G. 1996. Association between incidence of non-Hodgkin's lymphoma and solar ultraviolet radiation in England and Wales. *British Medical Journal* 312:1128-1131.

Berg, R.J.W., Van Kranen, H.J., Rebel, H.G., De Vries, A., Van Vloten, W.A., Van Kreijl, C.F., Van der Leun, J.C. and De Gruijl, F.R. 1996. Early p53 alterations in mouse skin carcinogenesis by UVB radiation: immunohistochemical detection of mutant p53 protein in clusters of preneoplastic epidermal cells. *Proceedings National Academy Sciences USA* 93:14025-14029.

Bergmanson, J.P.G. and Sheldon, T.M. 1997. Ultraviolet radiation revisited. *CLAO* 23:196-204.

Berkow, R. 1992. Ophthalmologic disorders, chapter 17. In *The Merck Manual*, Berkow, R. (ed.), Merck Research Laboratories, Rahway, NJ.

Berwick, M.C. 1998. Epidemiology: current trends, risk factors and environmental concerns. In *Cutaneous Melanoma*. Balch, C.M, Houghton, A.N., Sober, A.J. and Soong, S.-J. (eds.), Quality Medical Publishing, St. Louis, pp. 551-571.

Boonstra, A. and Savelkoul, H.F.J. 1997. The role of cytokines in ultraviolet B induced immunosuppression. *European Cytokine Network* 8:117-123.

Boxman, I.L., Berkhout, R.J., Mulder, L.H., Wolkers, M.C., Bouwes Bavinck, J.N., Vermeer, B.J. and ter Schegget, J. 1997. Detection of human papillomavirus DNA in plucked hairs from renal transplant recipients and healthy volunteers. *Journal Investigative Dermatology* 108:712-715.

Brash, D.E., Ziegler, A., Jonason, A.S., Simon, J.A., Kunala, S. and Leffell, D.J. 1996. Sunlight and sunburn in human skin cancer: p53, apoptosis, and tumor promotion. *Journal of Investigative Dermatology Symposium Proceedings* 1:136-142.

Brash, D.E., Rudolph, J.A., Simon, J.A., Lin, A., McKenna, G.J., Baden, H.P., Halparin, A.J. and Ponten, J. 1991. A role for sunlight in skin cancer: UV-induced *p53* mutations in squamous cell carcinomas. *Proceedings. National Academy of Sciences USA* 88:10124-10128.

Cartwright, R., McNally, R. and Staines, A. 1994. The increasing incidence of non-Hodgkin's lymphoma (NHL): the possible role of sunlight. *Leukemia Lymphoma* 14:387-394.

Cooper, K.D., Oberhelman, L., Hamilton, T.A., Baardsgaard, O., Terhune, M., LeVee, G., Anderson, T. and Koren, H. 1992. UV exposure reduces immunization rates and promotes tolerance to epicutaneous antigens in humans: relationship to dose, CD1a-DR+ epidermal macrophage induction and Langerhans cell deletion. *Proceedings of the National Academy of Science USA* 89:8497-8501.

Chuan, T.-Y., Reizner, G.T., Elpern, D.J., Stone, J.L. and Farmer, E.R. 1995. Non-melanoma skin cancer in Japanese ethnic Hawaiians in Kauai, Hawaii: An incidence report. *Journal of the American Academy of Dermatology* 33:422-426.

Deeg, H.J. and Witherspoon, R.P.1993. Risk factors for the development of secondary malignancies after marrow transplantation. *Hematology/Oncology Clinics North America* 7:417-429.

DeFabo, E.C. and Noonan, F.P. 1983. Mechanism of immune suppression by ultraviolet irradiation in vivo. I. Evidence for existence of a unique photoprotector in skin and its role in photoimmunology. *Journal of Experimental Medicine* 158:84-89.

de Gruijl, F.R. 1997. Health effects from solar UV radiation. *Radiation Protection Dosimetry* 72:177-196.

de Gruijl, F.R. and van der Leun, J.C. 1994. Estimate of the wavelength dependency of ultraviolet carcinogenesis in humans and its relevance to the risk assessment of a stratospheric ozone depletion. *Health Physics* 67:319-325.

de Gruijl, F.R., Sterenborg, H.J.C.M., Forbes, P.D., Davies, R.E., Cole, C., Kelfkens, G., van Weelden, H., Slaper, H. and van der Leun, J.C. 1993. Wavelength dependence of skin cancer induction by ultraviolet radiation of albino hairless mice. *Cancer Research* 53:53-60.

Denkins, Y., Fidler, I.J. and Kripke, M.L. 1989. Exposure of mice to UVB radiation suppresses delayed hypersensitivity to *Candida albicans*. *Photochemistry and Photobiology* 49:615-619.

Devary, Y., Rosette, C., DiDonata, J.A. and Karin, M. 1993. NF-κB activation by ultraviolet light is not dependent on a nuclear signal. *Science* 261:1442-1445.

Dumaz, N., Van Kranen, H.J., De Vries, A., Berg, R.J.W., Wester, P.W., Van Kreijl, C.F., Sarasin, A., Daya-Grosjean, L. and De Gruijl, F.R. 1997. The role of UVB light in skin carcinogenesis through the analysis of *p53* mutations in squamous cell carcinomas of hairless mice. *Carcinogenesis* 18:897-904.

ECETOC 1984. *Joint Assessment of Commodity Chemicals No.4. Methylene chloride, CAS: 75-09-2*, Turner, L. (ed.), European Center for Ecotoxicology and Toxicology of Chemicals, Brussels, Belgium.

ECETOC 1989. *Joint Assessment of Commodity Chemicals No. 9. Chlorodifluoromethane CAS: 75-45-6*, Carpanini, F.M. (ed.), European Center for Ecotoxicology and Toxicology of Chemicals, Brussels, Belgium.

ECETOC 1990a. *Joint Assessment of Commodity Chemicals No.16. Dichlorofluoromethane (HCFC-21) CAS:75-43-4*, Stringer, D.A. (ed.), European Center for Ecotoxicology and Toxicology of Chemicals, Brussels, Belgium.

ECETOC 1990b. *Joint Assessment of Commodity Chemicals No.11. 1,2,-Dichloro-1,1-difluoroethane (HFA-132b) CAS:1649-08-7*, Carpanini, F.M. (ed.), European Center for Ecotoxicology and Toxicology of Chemicals, Brussels, Belgium.

ECETOC 1990c. *Joint Assessment of Commodity Chemicals No.14. 1-Chloro-2,2,2-trifluoroethane (HFA-133a)*, Stringer, D.A. (ed.), European Center for Ecotoxicology and Toxicology of Chemicals, Brussels, Belgium.

ECETOC 1991. *Joint Assessment of Commodity Chemicals No.17. Chlorodifluorethane (1-chloro-1,1,difluoroethane; HFA 142b) CAS: 75-68-3*, Stringer, D.A. (ed.), European Center for Ecotoxicology and Toxicology of Chemicals, Brussels, Belgium.

ECETOC 1994a. *Joint Assessment of Commodity Chemicals No. 25. 1-Chloro-1,2,2,2-tetrafluoroethane (HCFC 124) CAS No. 2873-89-0*, Stringer, D.A. (ed.),, European Center for Ecotoxicology and Toxicology of Chemicals, Brussels, Belgium.

ECETOC 1994b. *Joint Assessment of Commodity Chemicals No.29. 1,1-Dichloro-1-Fluoroethane (HCFC-141b).CAS No 1717-00-6*, Stringer, D.A. (ed.), European Center for Ecotoxicology and Toxicology of Chemicals, Brussels, Belgium.

ECETOC 1994c. *Joint Assessment of Commodity Chemicals No.24. Pentafluoroethane (HFC 125) CAS No. 354-33-6*, Stringer, D.A. (ed.), European Center for Ecotoxicology and Toxicology of Chemicals, Brussels, Belgium.

ECETOC 1995a. *Joint Assessment of Commodity Chemicals No.32. Difluoromethane (HFC-32) CAS No. 75-10-5*, Stringer, D.A. (ed.), European Center for Ecotoxicology and Toxicology of Chemicals, Brussels, Belgium.

ECETOC 1995b. *Joint Assessment of Commodity Chemicals No.31. 1,1,1,2-Tetrafluoroethane (HFC-124a) CAS No. 811-97-2*, Stringer, D.A. (ed.), European Center for Ecotoxicology and Toxicology of Chemicals, Brussels, Belgium.

Elwood, M. and Jopson, J. 1997. Melanoma and sun exposure: an overview of published studies. *Journal of Cancer* 73:198-203.

EPA, 1994

Epstein, J.H. 1996. Nonmelanoma skin cancer. *Comprehensive Therapeutics* 22:179-182.

Finsen, N.R. (1901) Phototherapy. J.H. Sequira (transl.), Edward Arnold, London, pp. 79.

Fisher, M.S. and Kripke, M.L. 1977. Systemic alteration induced in mice by ultraviolet light irradiation and its relationship to ultraviolet carcinogenesis. *Proceedings of the National Sciences USA* 74:1688-1692.

Fountain, J.W. 1998. Genetic factors. In *Cutaneous Melanoma*, Balch, C.M, Houghton, A.N., Sober, A.J. and Soong, S.-J. (eds.), Quality Medical Publishing, St. Louis, pp.475-492.

Frisch, M., Hjalgrim, H., Olsen, J.H. and Melbye, M. 1994. Risk of subsequent cancer after diagnosis of basal cell carcinoma: a population-based epidemiological study. *Annals of Internal Medicine* 125:815-821.

Frisch, M. and Melbye, M. 1995. New primary cancers after squamous cell skin cancer. *American Journal of Epidemiology* 141:916-922.

Gallagher, R.P and McLean, D.I. 1995. The epidemiology of acquired melanocytic nevi - a brief review. *Dermatology Clinics* 13:595-603.

Gallagher, R.P., Hill, G.B., Bajdik, C.D., Fincham, S., Coldman, A.J., McLean, D.I. and Trefall, W.J. 1995a. Sunlight exposure, pigmentary factors and the risk of nonmelanocytic skin cancer. I: basal cell carcinoma *Archives Dermatology* 131:157-163

Gallagher, R.P., Hill, G.B., Bajdik, C.D., Fincham, S., Coldman, A.J., McLean, D.I. and Trefall, W.J. 1995b. Sunlight exposure, pigmentary factors and the risk of nonmelanocytic skin cancer. II: squamous cell carcinoma. *Archives Dermatology* 131:164-169.

Garmyn, M., Degreef, H. and Gilcrest, B.A. 1995. The effect of acute and chronic photodamage on gene expression in human keratinocytes. *Dermatology* 190:305-308.

Giannini, M.S.H. and DeFabo, E.C. 1987 Abrogation of skin lesions in cutanteous leishmaniasis. In *Leishmaniasis: The First Centenary (1885-1985) New Strategies for Control*, Hart, D.T. (ed.), NATO ASI Series A: Life Sciences, Plenum Publishing Company, London.

Goettsch, W., Garssen, J., De Gruijl, F.R. and Van Loveren, H. 1996. UVB-induced decreased resistance to *Trichinella spiralis* in the rat is related to impaired cellular immunity. *Photochemistry Photobiology* 54:581-585.

Goettsch, W., Garssen, J., Deijns, A., de Gruijl, F.R. and van Loveren, H. 1994. UV-B exposure impairs resistance to infection by *Trichinella spiralis. Environmental Health Perspectives* 102:298-301.

Goettsch, W., Garssen, J., Slob, W., De Gruijl, F.R. and Van Loveren, H. 1998. Risk assessment for the harmful effects of UVB radiation on the immunological resistance to infectious diseases. *Environmental Health Perspectives* 106:71-77.

Goldberg, L.H. 1997. Basal cell carcinoma as a predictor of other cancers. *The Lancet* 349:664-665.

Grodstein, F., Speizer, F.S. and Hunter, D.J. 1995. A prospective study of incident squamous cell carcinoma of the skin in the nurses' health study. *Journal of the National Cancer Institute* 87:1061-1066.

Hall, J., English, D.R., Artuso, M., Armstrong, B.K. and Winter, M. 1994. DNA repair capacity as a risk factor for non-melanocytic skin cancer--a molecular epidemiological study. *International Journal Cancer* 58:179-184.

Hargis, A.M. 1981 A review of solar induced lesions in domestic animals. *The Compendium on Continuing Education* 3:287-300.

Harteveld, M.M., Bouwes Bavinck, J.N., Kootte, A.M.M., Vermeer, B.J. and Vandenbroucke, J.P. 1990. Incidence of skin cancer after renal transplantation in the Netherlands. *Transplantation* 49:506-509.

Hartge, P., Devesa, S.S., Grauman, D., Fears, T.R. and Fraumeni, J.F. Jr. 1996. Non-Hodgkin's lymphoma and sunlight. *Journal of the National Cancer Institute* 88:298-300.

Hiller, R., Sperduto, R.D. and Ederer, F. 1983. Epidemiologic associations with cataract in the 1971-1972 National Health and Nutrition Examination Survey. *American Journal of Epidemiology* 118:239-249.

Hiramoto, K., Mashita, Y., Katada, T., Konishi, H., Sugiura, M. and Hayakawa, R. 1997. Immunosuppression by ultraviolet B rays via eyes in mice. *Archives Dermatological Research* 289:709-711.

Hirayama, F. and Ogawa, M. 1994. Cytokine regulation of early B-lymphopoiesis assessed in culture. *Blood Cells* 20:341-346.

Hodge, W.G., Whitcher, J.P. and Satariano, W. 1995. Risk factors for age-related cataracts. *Epidemiologic Reviews*: 17:336-346.

Hollows, F.C. 1989. Ultraviolet radiation and eye diseases. *Transactions Menzies Foundation* 15:113-117.

Holly, E.A., Aston, D.A., Ahn, D.K. and Smith, A.H. 1996. Intraocular melanoma linked to occupations and chemical exposures. Epidemiology 7:55-61.

Holman, C.D.J. and Armstrong, B.K. 1984a. Cutaneous malignant melanoma and indicators of total accumulated exposure to the sun: an analysis separating histogenetic types. *Journal of the National Cancer Institute* 73:75-82.

Holman, C.D.J. and Armstrong, B.K. 1984b. Pigmentary traits, ethnic origin, benign nevi, and family history as risk factors for cutaneous malignant melanoma. *Journal of the National Cancer Institute* 73:75-82.

Howie, S., Norval, M. and Maingay, J. 1986. Exposure to low-dose ultraviolet radiation suppressed delayed-type hypersensitivity to herpes simplex virus in mice. *Journal of Investigative Dermatology* 86:125-128.

Hoy, W.E. 1996. Nonmelanoma skin carcinoma in Albuquerque, New Mexico - experience of a major health care provider. *Cancer* 77:2489-2495.

Husain, Z., Pathak, M.A., Flotte, T. and Wick, M.M. 1991. Role of ultraviolet radiation in the induction of melanocytic tumors in hairless mice following 7,12-dimethylbenz (a) anthracene application and ultraviolet irradiation. *Cancer Research* 51:4964-4970.

Italian-American Cataract Study Group 1991. Risk Factors for Age-related cortical, nuclear and posterior subcapsular cataracts. *American Journal Epidemiology* 133:542-553.

Italian-American Cataract Study Group 1994. Incidence and progression of cortical, nuclear and posterior subcapsular cataract. *American Journal Ophthalmology* 118:623-631.

Jeevan, A. and Kripke, M.L. 1993. Impact of ozone depletion on immune function. *World Resources Review* 5:141-155.

Javitt, J.C. and Taylor, H.R. 1995. Cataract and latitude. *Documenta Ophthalmologica* 88:307-325.

Kanjilal, S., Pierceall, W.E., Cummings, K.K., Kripke, M.L. and Ananthaswamy, H.N. 1993. High frequency of *p53* mutations in ultraviolet radiation-induced murine skin tumors: evidence for strand bias and tumor heterogenicity. *Cancer Research* 53:2961-2964.

Kawashima, M., Favre, M., Obalak, S., Jablonska, S. and Orth, G. 1990. Premalignant lesions and cancers of the skin in the general population: evaluation of the role of human papillomaviruses. *Journal Investigative Dermatology* 95:537-542.

Kennedy, M., Kim, K.H., Harten, B., Brown, J., Planck, S., Meshul, C., Edelhauser, H., Rosenbaum, J.T., Armstrong, C.A. and Ansel, J.C. 1997. Ultraviolet irradiation induces the production of multiple cytokines by human corneal cells. *Investigations Ophthalmology & Visual Science* 38:2483-2491.

Kerb, R., Brockmöller, J., Reum, T. and Roots, I. 1997. Deficiency of glutathione s-transferases T1 and M1 as heritable factors of increased cutaneous UV sensitivity. *Jounal of Investigative Dermatology* 108:229-232.

Klein, B.E.K., Cruickshanks, K.J. and Klein, R. 1995. Leisure time, sunlight exposure and cataracts. *Documenta Ophthalmologica* 88:295-305.

Kligman, A.M. and Kligman, L.H. 1993. Photoaging. In *Dermatology in General Medicine, 4th. Ed.*, Fitzpatrick, T.B., Eisen, A.Z., Wolff, K., Freedberg, I.M. and Austen, K.F. (eds.), McGraw-Hill, New York, pp. 2972-2979.

Koh, J.S. and Levine, J.S. 1997. Apoptosis and autoimmunity. *Current Opinion in Nephrology and Hypertension* 16:259-266.

Kopecky, K.E., Pugh, G.W. and Hughes, D.E. 1980. Wavelength of ultraviolet radiation that enhances onset of critical infectious bovine keratoconjunctivitis. *American Journal of Veterinary Research*. 41:1412-15.

Kraemer, K.H., Lee, M.M. and Scotto, J. 1984. DNA repair protects against cutaneous and internal neoplasia: evidence from xeroderma pigmentosum. *Carcinogenesis* 5:11-514.

Kress, S., Sutter, C., Strickland, P.T., Mukhtar, H., Schweizer, J. and Schwartz, M. 1992. Carcinogen-specific mutational pattern in the *p53* gene in ultraviolet B radiation-induced squamous cell carcinomas of mouse skin. *Cancer Research* 52:6400-6403.

Kricker, A. 1992. *Sun Exposure and Non-Melanocytic Skin Cancer*. Dissertation. Perth, University of Western Australia.

Kricker, A., Armstrong, B.K., English, D.R. and Heenan, P.J. 1995a. A dose-response curve for sun exposure and basal cell carcinoma. *International Journal Cancer* 60:482-488.

Kricker, A., Armstrong, B.K., English, D.R. and Heenan, P.J. 1995b. Does intermittent sun exposure cause basal cell carcinoma? *International Journal Cancer* 60:489-494.

Kripke, M.L. 1984. Immunological unresponsiveness induced by ultraviolet radiation. *Immunological Reviews* 80:87-102.

Kripke, M.L., Cox, P.A., Alas, L.G. and Yarosh, D.B. 1992. Pyrimidine dimers in DNA initiate systemic suppression in UV-irradiated mice. *Proceedings of the National Academy of Sciences USA* 89:7516-7520.

Kut, C., Hornebeck, W., Groult, N., Redziniack, G. Godeau, G. and Pellat, B. 1997. Influence of successive and combined ultraviolet A and B on matrix metalloelastases produced by human dermal fibroblasts in culture. *Biology International* 21:347-352.

Ley, R.D. 1997. Ultraviolet radiation A-induced precursors of cutaneous melanoma in *Mondelphis domestica*. *Cancer Research* 57:3682-3684.

Longstreth, J.D. 1998. Melanoma genesis: putative causes and possible mechanisms. In *Cutaneous Melanoma*, Balch, C.M, Houghton, A.N., Sober, A.J. and Soong, S.-J. (eds), Quality Medical Publishing, St. Louis, pp. 535-550.

Longstreth, J.D., De Gruijl, F.R., Kripke, M.L., Takizawa, Y. and Van der Leun, J.C. 1995. Effects of increased solar ultraviolet radiation on human health. *Ambio* 24:153-165.

Lucey, D.R., Clerici, M. and Shearer, G.M. 1996. Type 1 and type 2 cytokine dysregulation in human infectious, neoplastic and inflammatory diseases. *Clinical Microbiology Reviews* 9:532-562.

Mackenzie, F.D., Hirst, L.W., Battistutta, D. and Green, A. 1992. Risk analysis in the development of pterygia. *Ophthalmology* 99:1056-1061.

Madronich, S. and De Gruijl, F.R. 1993. Skin cancer and UV radiation. *Nature* 366:23.

Mayer, S.J. 1992. Stratospheric ozone depletion and animal health. *Veterinary Record* 131:120-122.

McCarty, C.A. and Taylor, H.R. 1996. Recent developments in vision research. *Investigations Ophthalmology & Visual Science* 37:1720-1723.

McGrath, H., Bell, J.M. and Haycock, J.W. 1994. Fluorescent light activates the immunomodulator cis-urocanic acid in vitro: implications for patients with systemic lupus erythematosus. *Annals of the Rheumatic Diseases* 53:396-399.

McGregor, J.M., Berkhout, R.J.M., Rozycka, M., ter Schegget, J., Bouwes-Bavinck, J.N., Brooks, L. and Crook, T. 1997. p53 mutations implicate sunlight in post-transplant skin cancer irrespective of human papillomavirus status. *Oncogene* 15:1737-1740.

McMichael, A.J. and Hall, A.J. 1997. Does immunosuppressive ultraviolet radiation explain the latitude gradient for multiple sclerosis? *Epidemiology* 8:642-645.

Méndez, A., Pérez, J., Ruiz-Villamor, E., Martin, M.P. and Mozos, E. 1997. Clinicopathological study of an outbreak of squamous cell carcinoma in sheep. *Veterinary Record* 141:597-600.

Meola, T., Soter, N.A., Ostreicher, R., Sanchez, M. and Moy, J.A. 1993. The safety of UVB phototherapy in patients with HIV infection. *Journal American Academy Dermatology* 29:216-220.

Merriam, J.C. 1996. The concentration of light in the human lens. *Transactions American Ophthalmological Society* 94:804-918.

Müller, K. and Bendtzen, K. 1996. 1,25-dihydroxivitamin D_3 as a natural regulator of human immune functions. *Journal of Investigative Dermatology Symposium Proceedings* 1:68-71.

Nakazawa, H.D., English, D., Randell, P.L., Nakazawa, K., Martel, N., Armstrong, B.K. and Yamasaki, H. 1994. UV and skin cancer: specific *p53* gene mutation in normal skin as a biologically relevant exposure measurement. *Proceedings National Academy Sciences USA* 91:360-363.

Nataraj, A.J., Wolf, P., Cerroni, L. and Ananthaswamy, H.N. 1997. p53 mutation in squamous cell carcinomas from psoriasis patients treated with psoralen + UVA (PUVA). *Journal Investigative Dermatology* 109:238-243.

Naylor, M.F. and Everett, M.A. 1996. Involvement of the p16INK4 (CDKN2) gene in familial melanoma. *Melanoma Research* 6:139-145.

Newton, R., Ferlay, J., Reeves, G., Beral, V. and Parkin, D.M. 1996. Effect of ambient solar ultraviolet radiation on incidence of squamous-cell carcinoma of the eye. *Lancet* 347:1450-1451.

Niizeki, H. and Streilein, J.W. 1997. Hapten-specific tolerance induced by acute low-dose ultraviolet B radiation of the skin is mediated via IL-10. *Journal of Investigative Dermatology* 109:25-30.

Nomura, T., Nakajima, H., Hongyo, T., Taniguchi, E., Fukuda, K., Li, L.Y., Kurooka, M., Sutoh, K., Hande, P.M., Kawaguchi, T., Ueda, M. and Takatera, H. 1997. Induction of cancer, actinic keratosis and specific *p53* mutations by UVB light in human skin maintained in severe combined immunodeficient mice. *Cancer Research* 57:2081-2084.

Noonan, F.P. and Lewis, F.A. 1995 UVB-induced immune suppression and infection with Schistosoma mansoni. *Photochemistry and Photobiology* 61:99-105.

Norval, M. and el Ghorr, A.A. 1996. UV radiation and mouse models of herpes simplex virus infection. *Photochemistry and Photobiology* 64:242-245.

Pathak, M.A. 1985. Activation of the melanocyte system by ultraviolet radiation and cell transformation. *Annals of the New York Academy of Sciences* 453:328-339.

Perna, J.J., Manix, M.L., Rooney, J.E., Notkins, A.D. and Straus, S.E. 1987. Reactivation of latent herpes simplex virus infection by ultraviolet radiation: a human model. *Journal American Academy of Dermatology* 17:197-212.

Pitcher, H.M. and Longstreth, J.D. 1991. Melanoma mortality and exposure to ultraviolet radiation: An empirical relationship. *Environment International* 17:7-21.

Ren, Z.P., Pontén, F., Nistér, M. and Pontén, J. 1996. Two distinct p53 immunohistochemical patterns in human squamous cell skin cancer, precursors and normal epidermis. *International Journal Cancer* 69:174-179.

Rosso, S., Zanetti, R., Martinez, C., Tormo, M.J., Schraub, S., Sancho-Garnier, H., Franceschi, S., Gafa, L., Perea, E., Navarro, C., Laurent, R., Schrameck, C., Talamini, R., Tumino, R. and Wechsler, J. 1996. The multicentre south European study 'Helios'. II: different sun exposure patterns in the aetiology of basal cell and squamous cell carcinomas of the skin. *British Journal Cancer* 73:1447-1454.

Saah, A.J., Horn, T.D., Hoover, D.R., Chen, C., Whitmore, S.E., Flynn, C., Wesch, J., Detels, R. and Anderson, R. 1997. Solar ultraviolet radiation exposure does not appear to exacerbate HIV infection in homosexual men. *AIDS* 11:1773-1778.

Schein, O.D., Vicencio, C., Munoz, B., Gelatt, K.N., Duncan, D.D., Nethercott, J., Honeyman, J., Koren, H.S. and West, S. 1995. Ocular and dermatologic health effects of ultraviolet radiation exposure from the ozone hole in southern Chile. *American Journal Public Health* 85:546-550.

Scharffetter-Kochanek, K., Wiaschek, M., Brenneisen, P., Schauen, M., Blaudschun, R. and Wenk, J. 1997. UV-induced reactive oxygen species in photocarcinogenesis and photoaging. *Biological Chemistry* 378:1247-1257.

Scotto, J., Pitcher, H. and Lee, J.A.H. 1991. Indications of future decreasing trends in skin-melanoma mortality among whites in the United States. *International Journal of Cancer* 49:490-497.

Scotto, J., Fears, T.R. and Fraumeni, J.F. Jr. 1981. Incidence of nonmelanoma skin cancer in the United States. National Cancer Institute, U.S. Department of Health and Human Services, NIH 82-2433.

Screibner, A., Hollis, D.E., Murray, E., McCarthy, W.H. and Milton, G.W. 1987. Effects of exposure to ultraviolet light on epidermal Langerhans cells and melanocytes in Australians of Aboriginal, Asian and Celtic descent. *Photodermatology* 3:15-25.

Setlow, R.B., Grist, E., Thompson, K. and Woodhead, A.D. 1993. Wavelengths effective in the induction of malignant melanoma. *Proceedings of the National Academy of Sciences USA* 71:3363-3366.

Sharpe, R.J. 1986. The low incidence of multiple sclerosis near the equator may be due to ultraviolet light induced suppressor cells to melanocyte antigens. *Medical Hypotheses* 19:319-323.

Slaper, H., Velders, G.J.M., Daniel, J.S., De Gruijl, F.R. and Van der Leun, J.C. 1996. Estimates of ozone depletion and skin cancer incidence to examine the Vienna Convention achievements. *Nature* 384:256-258.

Spruance, S.L. 1985. Pathogenesis of herpes simplex labialis: experiment induction of lesions with UV light. *Journal Clinical Microbiology* 22:366-468.

Stern, R.S., Weinstein, M.C. and Baker, S.G. 1986. Risk reduction for nonmelanoma skin cancer with childhood sunscreen use. *Archives Dermatology* 122:537-545.

Stern, R.S. and Laird, N. 1994. The carcinogenic risk of treatments for severe psoraisis. *Cancer* 73:2759-2764.

Stern, R.S., Nichols, K.T. and Vakeva, L.H. 1997. Malignant melanoma in patients treated for psoriasis with methoxsalen (psoralen) and ultraviolet A radiation (PUVA). The PUVA Follow-Up Study. *New England Journal Medicine* 336:1041-1045.

Streilein, J.W. 1996. Photoimmunology of non-melanoma skin cancer. *Cancer Surveys* 26:207-217.

Streilein, J.W. and Bergstresser, P.R. 1988. Genetic basis of ultraviolet-B effects on contact hypersensitivity. *Immunogenetics* 27:398-405.

Stickland, F.M. and Kripke, M.L. 1997. Immune response associated with nonmelanoma skin cancer. *Clinics in Plastic Surgery* 24:637-647.

Strom, S.S. and Yamamura, Y. 1997. Epidemiology of nonmelanoma skin cancer. *Clinics in Plastic Surgery* 24:627-636.

Taylor, H.R., West, S.K., Rosenthal, F.S., Munoz, B., Newland, H.S. and Emmett, E.A. 1989. Corneal changes associated with chronic UV irradiation. *Archives Ophthalmology* 107:1481-1484.

Teifke, J.P. and Lohr, C.V. 1996. Immunohistochemical detection of p53 overexpression in paraffin wax-embedded squamous cell carcinomas of cattle, horses, cats and dogs. *Journal of Comparative Pathology* 114:205-210.

Tieben, L.M., Berkhout, R.J.M., Smits, H.L., Bouwes Bavinck, J.N., Vermeer, B.J., Bruijn, J.A., Van der Woude, F.J. and Ter Schegget, J. 1994. Detection of epidermodysplasia verruciformis-like human papillomavirus types in malignant and premalignant skin lesions of renal transplant recipients. *British Journal of Dermatology* 131:226-230.

Thompson, S.C., Jolley, D. and Marks R. 1993. Reduction in solar keratoses by regular sunscreen use. *New England Journal of Medicine* 329:1147-1151.

Ullrich, S.E. 1995. The role of epidermal cytokines in the generation of cutaneous immune reactions and ultraviolet radiation-induced immune suppression. Photochemistry and Photobiology 62:389-401.

UNEP (United Nations Environment Programme) 1994. *Environmental Effects of Ozone Depletion: 1994 Update*, van der Leun, J.C., Tevini, M., Tang, X. and Worrest, R.C. (eds.), United Nations Environment Programme, Nairobi, 1994.

UNEP (United Nations Environment Programme) 1998. *Environmental Effects of Ozone Depletion: 1998 Update*, van der Leun, J.C., Tevini, M., Tang, X. and Worrest, R.C. (eds.), United Nations Environment Programme, Nairobi, this report.

USEPA (U.S.Environmental Protection Agency) 1987a. Chapters on: Nonmelanoma skin cancer, Cutaneous malignant melanoma, and Cataract, In *Assessing the Risks of Trace Gases That Can Modify the Stratosphere*, EPA 400/1-87/001C. Pp 7-1 to 7-66, 8-1 to 8-51, 10-1 to 10-48.

USEPA (U.S.Environmental Protection Agency) 1988. *Regulatory Impact Analysis: Protection of Stratospheric Ozone*, Stratospheric Protection Program, Office of Air and Radiation, U.S. Environmental Protection Agency.

Valverde, P., Healy, E, Jackson, I, Rees J.L. and Thody, A.J. 1995. Variants of the melanocyte stimulating hormone receptor gene are associated with red hair and fair skin in humans. *Nature Genetics* 11:328-330.

Valverde, P., Healy, E, Sikkink, S., Haldane, F., Thody, A.J., Carothers, A., Jackson, I.J. and Rees, J.L. 1996. The Asp84glu variant of the melanocortin 1 receptor (MC1R) is associated with melanoma. *Human Molecular Genetics* 5:1663-1666.

van der Leun, J.C. and Van Weelden, H. 1986. UVB phototherapeutic principles, radiation sources, regimens. In *Therapeutic Photomedicine*, H Hoenigsmann, G Stingl (eds.), Karger, Basel, pp. 39-51.

van der Leun, J.C., Takizawa, Y. and Longstreth, J.D. 1989. Human Health, Chapter 2. In Environmental Effects Panel Report, United Nations Environment Programme, Nairobi.

van der Leun, J.C., Takizawa, Y. and Longstreth, J.D. 1989. Human health. In Environmental Effects Panel Report, van der Leun, J.C., Tevini, M, and Worrest, R.C. (eds.), UNEP, Nairobi, 1991.

van der Leun, J.C. and De Gruijl, F.R. 1993. Influences of ozone depletion on human and animal health. In *UVB Radiation and Ozone Depletion*, Tevini, M. (ed.), Lewis Publishing, Boca Raton, pp 95-123.

Varma, S.D., Devamanoharan, P.S. and Morris, S.M. 1995. Prevention of cataracts by nutritional and metabolic antioxidants. *Critical Reviews of Food Science and Nutrition* 35:111-29.

Vitasa, B.C., Taylor, H.R., Strickland, P.T., Rosenthal, F.S., West, S., Abbey, H., Ng, S.K., Munoz, B. and Emmett, E.A. 1990. Association of nonmelanoma skin cancer and actinic keratosis with cumulative solar ultraviolet exposure in Maryland watermen. C*ancer* 65:2811-2817.

WMO (World Meteorological Organization) 1998. Scientific Assessment of Ozone Depletion: 1998. In *Global Ozone Research and Monitoring Project*, Albritton, D.L, Aucamp, P.J., Megie, G. and Watson, R.T. (eds.), World Meteorological Organization, Geneva, in press.

Wei, Q., Matanoski, G.M., Farmer, E.R., Hedayati, M.A. and Grossman, L. 1993. DNA repair and aging in basal cell carcinoma: a molecular epidemiology study. *Proceedings National Academy Sciences USA* 90:1614-1618.

West, S.K., Duncan, D.D., Munoz, B, Rubin, G.S., Fried, L.P., Bandeen-Roche, K. and Schein, O.D. 1998. Sunlight exposure and the risk of lens opacities in a population-based study: the Salisbury eye evaluation project. *JAMA* 280:714-718

West, S.K. and Valmadrid, C.T. 1995. Epidemiology of risk factors for age-related cataract *Surveys in Ophthalmology* 39:323-335.

Yamawaki, M., Katiyar, S.K., Anderson, C.Y., Tubesing, K.A., Mukhtar, H. and Elmets, C.A. 1997. Genetic variation in low-dose UV-induced suppression of contact hypersensitivity and in skin photocarcinogenesis. *Journal Investigative Dermatology* 109:716-721.

Yoshikawa, T. and Streilein, J.W. 1990. Genetic basis of the effects of ultraviolet light B on cutanteous immunity. Evidence that polymorphism at the *Trfa* and *LPS* loci governs susceptibility. *Immunogenetics* 32:398-405.

Yoshikawa, T., Rae, V., Bruin-Slot, W., Van den Berg, W., Taylor, J.R. and Streilein J.W. 1990. Susceptibility to effects of UV-B radiation on the induction of contact hypersensitivity as a risk for skin cancer in humans. *Journal of Investigative Dermatology* 95:530-536.

Ziegler, A., Leffel, D.J., Kunala, S., Sharma, H.W., Gailani, M., Simon, J.A., Halperin, A.J., Shapiro, P.E., Bale, A.E. and Brash, D.E. 1993. Mutation hotspots due to sunlight in the p53 gene of nonmelanoma skin cancers. *Proceedings National Academy Sciences USA* 90:4216-4220.

Zmudzka, B.Z. and Beer, J.B. 1990. Activation of human immunodeficiency virus by ultraviolet radiation. *Photochemistry and Photobiology* 6:1153-1162.

Ziegler, A., Jonason, A.S., Leffel, D.J., Simon, A., Sharma, H.W., Kimmelman, J., Remington, L., Jacks,T. and Brash, D.E. 1994. Sunburn and p53 in the onset of skin cancer. *Nature* 372:773-776.

CHAPTER 3

EFFECTS OF INCREASED SOLAR ULTRAVIOLET RADIATION ON TERRESTRIAL ECOSYSTEMS

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Summary

Elevated solar UV-B radiation associated with stratospheric ozone reduction may exert effects on terrestrial ecosystems through actions on plants, microbes, and perhaps on some animals. At the ecosystem level, the effects are less well understood than at the molecular and organismal levels. Many of the most important, yet less predictable, consequences will be indirect effects of elevated UV-B acting through changes in the chemical composition and form of plants and through changes in the abiotic environment. These indirect effects include changes in the susceptibility of plants to attack by insects and pathogens in both agricultural and natural ecosystems; the direction of these changes can result in either a decrease or an increase in susceptibility. Other indirect effects of elevated UV-B include changes in competitive balance of plants and nutrient cycling. The direct UV-B action on plants that results in changes in form or function of plants appears to occur more often through altered gene activity rather than damage. The yield of some crop varieties can be decreased by elevated UV-B, but other varieties are not affected. Plant breeding and genetic engineering efforts should be able to cope with the potential threats to crop productivity due to elevated UV-B. For forest trees, this may be more difficult if effects of elevated UV-B accumulate over several years. All effects of elevated UV-B radiation must be considered in the context of other climate changes such as increased temperature and levels of carbon dioxide, which may alter the UV-B responses, especially for plants. The actions of elevated carbon dioxide and UV-B appear to be largely independent, but interactions occur between changes in UV-B and other factors. Other ecosystem-level consequences of elevated UV-B radiation are emerging and their magnitude and direction will not be easily predicted.

Introduction

Terrestrial ecosystems include agricultural lands, agroecosystems, and less intensively managed lands such as forests, grasslands, savannahs, deserts, tundra, etc. In any of these environments, ecosystem function includes many attributes that could potentially be affected by increased solar UV-B radiation including plant biomass production, seed production, plant consumption by herbivores including insects, disease incidence of plants and animals, population fluctuations of plants and animals, and changes in species composition and mineral nutrient cycling. Treatment of some aspects of ecosystem function, e.g., nutrient cycling, overlap with Chapter 5, and effects on amphibians in aquatic components of terrestrial systems overlap with Chapter 4.

Study of ecosystem-level effects of solar UV-B increase has only been undertaken in the past few years. However, much attention has been directed to effects of UV-B radiation on higher plants since the first reports of potential stratospheric ozone reduction over 25 years ago (e.g., Johnston 1971, Crutzen 1972). Approximately 600 papers have appeared, but the majority of these deal with herbaceous, agricultural plants under laboratory or glasshouse conditions. Fewer than 5% of the studies have been conducted under field conditions, and fewer still with plants from forests and other nonagricultural systems.

Numerous reviews of this literature dealing primarily with effects on terrestrial plants have appeared in the last decade (Bornman, 1989; Caldwell et al., 1989; Krupa and Kickert, 1989; Tevini and Teramura, 1989; Stapleton, 1992; Bornman and Teramura, 1993; Caldwell and Flint, 1993, 1994a,b; Strid et al., 1994; Tevini, 1993, 1994, 1996; Teramura and Sullivan, 1994; Manning and Tiedemann, 1995; Bornman and Sundby-Emanuelsson, 1995; Björn, 1996; Jordan, 1996; Panten et al., 1996; Sullivan, 1997; Rozema et al., 1997a,b). Rather than a review of the literature, this chapter provides an overview with interpretation of the results for both agriculture and of other ecosystems such as forests, grasslands, etc.

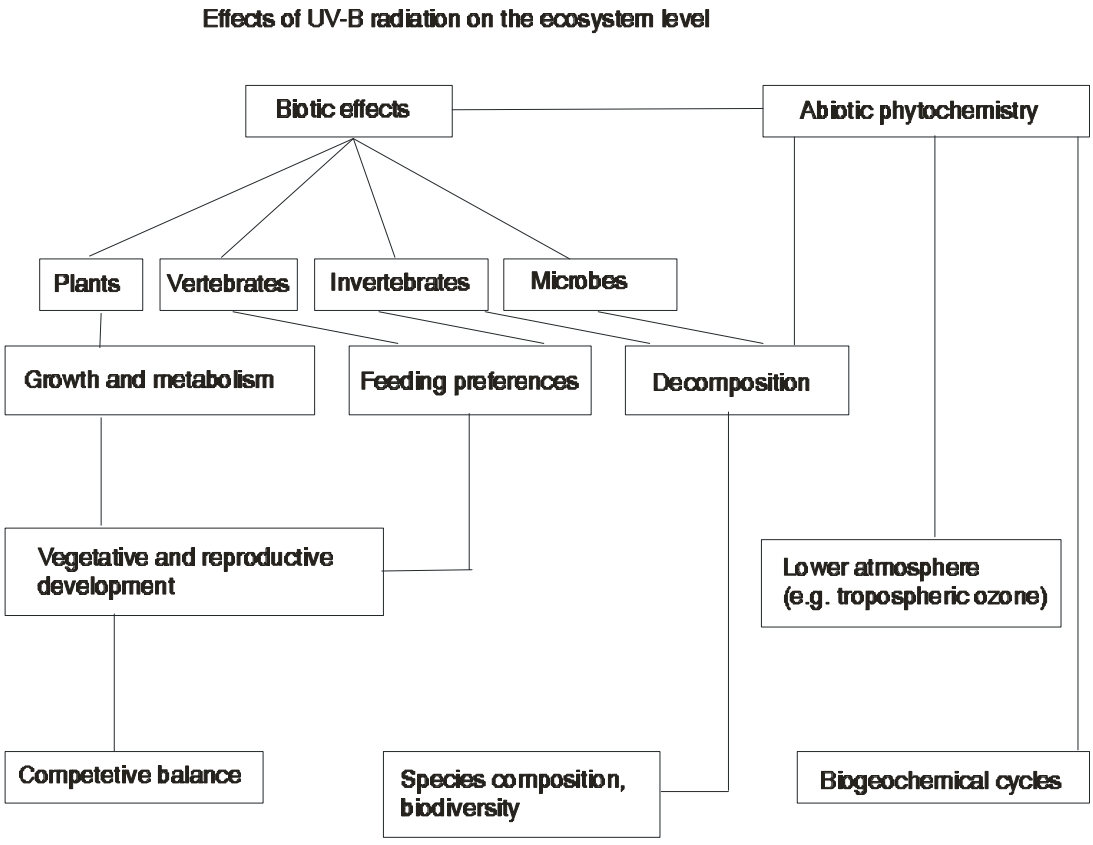

Fig. 3.1. Scheme of direct photochemical effects of elevated solar UV-B radiation on plants, microbes and animals and indirect ecosystem effects mediated through changes in plant competition, microbial population changes, secondary chemistry, plant litter decomposition and air quality. The arrows indicate some of the potential interactions and consequences of UV-B radiation. See also Fig. 5.2 for related ecosystem-level effects and processes.

In terrestrial ecosystems, organisms apart from higher plants have received comparatively little attention with respect to direct effects of solar UV-B radiation increases. Some direct effects on microbes

and animal life have been demonstrated (e.g., Blaustein et al., 1994; Gehrke et al., 1995; see also Chapters 2 and 4). Microbes perform many important ecosystem functions including litter decomposition, cycling of mineral nutrients, pathogenic action, and symbiotic interaction with both plants and animals. Direct UV-B effects on microbes have been extensively studied (Jagger, 1981), but the ecological relevance is not well understood. Animals are often thought to be generally well shielded from solar UV-B radiation by pigments such as melanin, body coverings such as feathers, fur, etc. In some animals, the eyes may be at risk if there is prolonged UV-B exposure (see Chapter 2). However, microbes exposed to sunlight are usually not so well shielded.

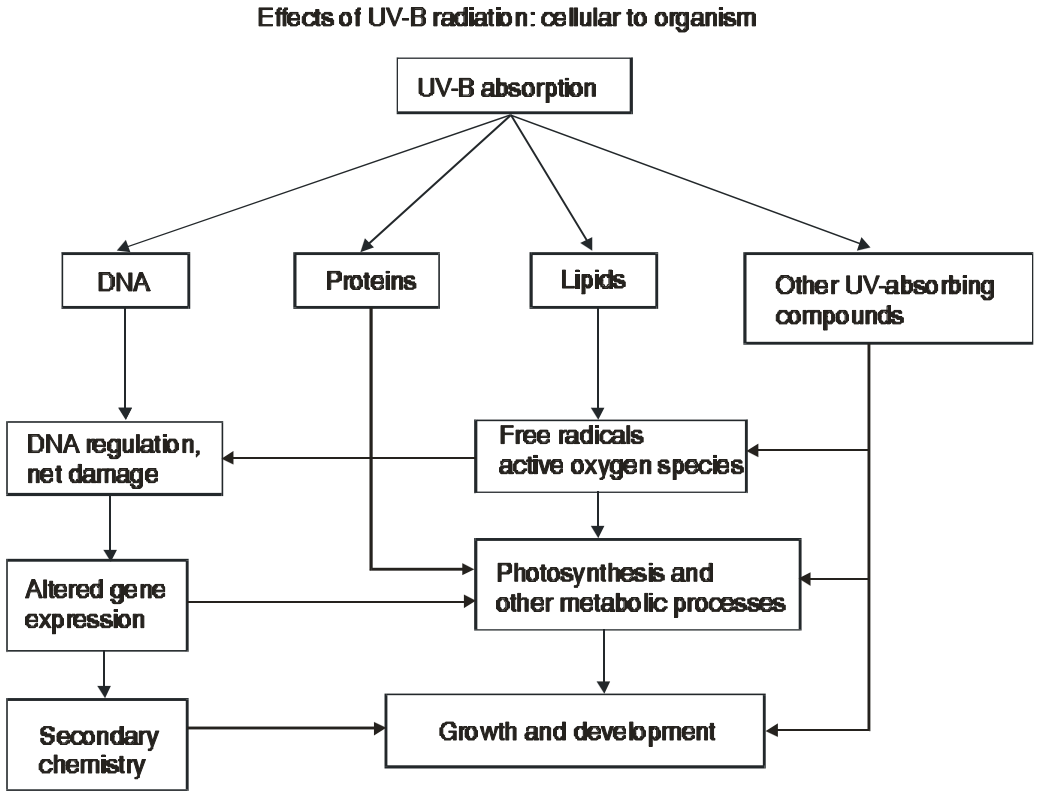

Figure 3.2. The influence of UV-B radiation on several damage and regulatory processes in organisms. Much of the initial damage can be repaired, (e.g., DNA damage). Damage and regulatory changes in plants and other organisms alter metabolism and ultimately growth, reproduction and survival. The arrows indicate some of the potential interactions and consequences of UV-B radiation.

The major anticipated effects of increased solar UV-B on agricultural and nonagricultural ecosystems (such as forests, grasslands, savannahs, deserts, tundra, etc.) may result from direct UV-B radiation effects on plants, insects and microbes, or indirect effects of UV-B on these organisms that mediate other effects (Fig. 3.1). Non-biological UV-B effects such as direct photochemical reactions in plant litter during decay (Chapter 5) or effects on the ambient air quality (Chapter 6) can influence other processes in ecosystems. Although the principal processes may be the same in highly managed agroecosystems (e.g., agronomic crops) and in nonagricultural ecosystems, their importance may differ. For example, effects on litter decomposition or plant competitive balance may be less important in annually cultivated crop fields.

For individual organisms, there are several potential pathways of UV-B action in damage and regulatory processes that affect whole organism performance, such as growth and reproduction (Fig. 3.2).

General Effects on Organisms

Basic Effects of UV-B Radiation on Organisms and their Protective Responses

Enhanced UV-B radiation can have many direct and indirect effects on organisms. However, organisms have developed mechanisms of protection and mitigation of UV-B radiation damage. General deleterious effects include production of active oxygen species and free radicals, DNA damage and, for plants, partial inhibition of photosynthesis. Protective responses include radiation shielding due to structural or pigment changes and specific damage repair systems. Although photochemical lesions of DNA and proteins and damage as a result of active oxygen species and free radicals may occur, many of the effects of UV-B radiation may be expressed through increased regulation rather than sustained damage,

In order for UV radiation to be effective in most organisms, it must effectively penetrate into the tissues and be absorbed. Structural and biochemical changes induced by enhanced levels of UV-B radiation ultimately modify the penetration of UV radiation into plants and other organisms. The UV shielding in most animals is thought to be quite effective in minimizing UV-B damage, but this should be further examined (see Chapter 2). For example, different stages of insect larvae may be less well protected by UV-absorbing pigments. In plants, a certain amount of UV-screening pigments may be constitutive, and additional UV-absorbing compounds (usually phenolic compounds) can be synthesized when plants are exposed to increased levels of UV radiation. This will naturally be important in reducing the penetration of UV-B radiation to underlying tissues. Experimental mutant plants that lack these pigments are very sensitive to natural sunlight UV-B (Li et al., 1993; Reuber et al., 1996). Other adjustments in plant leaves after exposure to increased UV-B radiation may also contribute to a heightened UV defense. At the structural level, increased leaf thickness is often induced by UV-B radiation that reduces UV-B penetration to internal leaf tissues (Bornman and Vogelmann, 1991). Ultraviolet radiation penetration varies among different plant species and this may be reflected in the sensitivity of these species. Penetration of UV-B was found to be greatest in herbaceous dicotyledons (broad-leaved plants) and was progressively less in woody dicotyledons, grasses and conifers (Day et al., 1992). The UV penetration also changes with leaf age; younger leaves attenuate UV-B radiation less than do the more mature leaves, as was shown for some conifers (DeLucia et al., 1991, 1992).

Of the different kinds of molecular damage, radiation damage to DNA is potentially dangerous to cells, because a single photon hit in a single molecule may have dramatic, sometimes even lethal effects. Many different types of DNA damage are known that result from free radicals and reactive oxygen species formed in various photochemical processes. The two most common UV-B induced DNA lesions are the cyclobutane pyrimidine dimers and (6-4) photoproducts which are pyrimidine adducts. These two types of lesions differ from other DNA lesions in that many organisms living in sunlit habitats possess special enzymes (photolyases) that can effectively repair many of these lesions in the presence of visible light and favorable temperatures. Some DNA repair systems can also operate without light (Britt, 1996; Taylor et al., 1996). Much of the research in this area has been conducted under laboratory conditions, but the level of DNA lesions in intact plants has also been measured under field conditions (e.g., Quaite et al., 1992b; Ballaré et al., 1996; Stapleton et al., 1997). While these

studies indicate effective repair of DNA damage (Stapleton et al., 1997), the UV component of sunlight is still sufficient to result in some level of persistent damage. Low temperature can slow this enzymatic repair of DNA damage (Britt, 1996; Takeuchi et al., 1996). Therefore, plants, cold-blooded animals and microbes in cold environments may suffer from a less favorable balance between damage and repair than others. Unfortunately, these environments overlap with those exposed to the greatest ozone depletion.

When exposure to increased UV radiation leads to stimulation of UV-absorbing compounds in plant tissues, another protective effect can result from the antioxidant properties that certain of the compounds confer. Enhanced levels of UV-B radiation appear to selectively stimulate those flavonoids (a type of phenolic) with potential antioxidant properties (Cen et al., 1993; Liu et al., 1995; Reuber et al., 1996; Olsson et al., 1998). This selective enhancement can be up to 500% (Reuber et al., 1996). At present, it is not known how extensive this selective induction is within the plant kingdom.

Many genes in plants, animals and microorganisms are regulated by UV-B, and changes in UV-B may have important consequences by altered gene action (Strid et al., 1994; Jordan, 1996; Bender et al., 1997). The mechanisms of how the organism perceives UV-B radiation and how signals are transduced are not yet well understood. Active oxygen can be one trigger for altered gene activity (Mackerness et al. 1998). No matter what the triggering agent, altered gene activity is important, since UV-B radiation is involved in changes of gene expression which are reflected in many aspects of plant function. For example, an increased amount of UV-B radiation results in enhanced synthesis of UV-screening pigments and is due to the expression of particular genes (Jenkins et al., 1997). It appears that the effects of UV-B radiation on photosynthesis, growth and development of plants are caused by altered gene action. This is currently a topic of intensive research.

Decreased elongation also may be due to UV-induced destruction of the plant hormone auxin, that absorbs in the UV-B range and could be photodegraded by high levels of UV-B radiation. Oxidative enzymes, such as the peroxidases, the activity of which is increased by enhanced UV-B radiation, also may be involved in plant hormone-regulated growth responses, as shown in sunflower and rice plants (Ros and Tevini, 1995; Huang et al., 1997). The levels of another plant hormone, ethylene, which causes greater radial growth and less elongation, are increased after UV-B irradiation in sunflower seedlings (Ros and Tevini, 1995) and cultured shoots of pear seedlings (Predieri et al., 1993). Changes in hormone levels ultimately may be due to UV-B-induced gene expression, but this remains to be demonstrated.

The Biological Effectiveness of Changes in Sunlight

As explained in Chapter 1, the biological effectiveness of solar UV-B radiation needs to be taken into account in assessing what ozone reduction, and the resulting changes in solar radiation, may mean for biological systems and processes. The biological weighting functions used for this purpose often come from action spectra. Action spectra assumed to be relevant for organisms, especially plants (Fig. 3.3) all indicate that the shorter UV-B wavelengths are the most important. However, the relative importance of shorter vs longer UV-B wavelengths (the slopes in Fig. 3.3) varies considerably. Depending on these slopes and the tails of the spectra extending into UV-A, the Radiation Amplification Factors (discussed in Chapter 1) vary enormously. Only the weighting functions with steep slopes result in RAF values suggesting that ozone reduction is potentially important. Thus, the evaluation of weighting functions (and therefore action spectra) is critical. Although there is evidence that action spectra for some plant functions are steep, indicating that ozone reduction translates into large increases in ef-

fective solar UV-B (Caldwell, 1971; Setlow, 1974), some more recent spectra developed specifically for evaluating the ozone reduction problem show somewhat flatter slopes (and therefore somewhat lower RAF values) than the earlier work (Caldwell et al., 1986; Steinmüller, 1986; Quaite et al., 1992a; Cen and Björn, 1994). Still, many of these spectra are sufficiently steep so that ozone reduction must be taken seriously (Flint and Caldwell, 1996; Caldwell and Flint, 1997). Biological weighting functions also are needed to relate solar UV to UV from lamps used in many experiments.

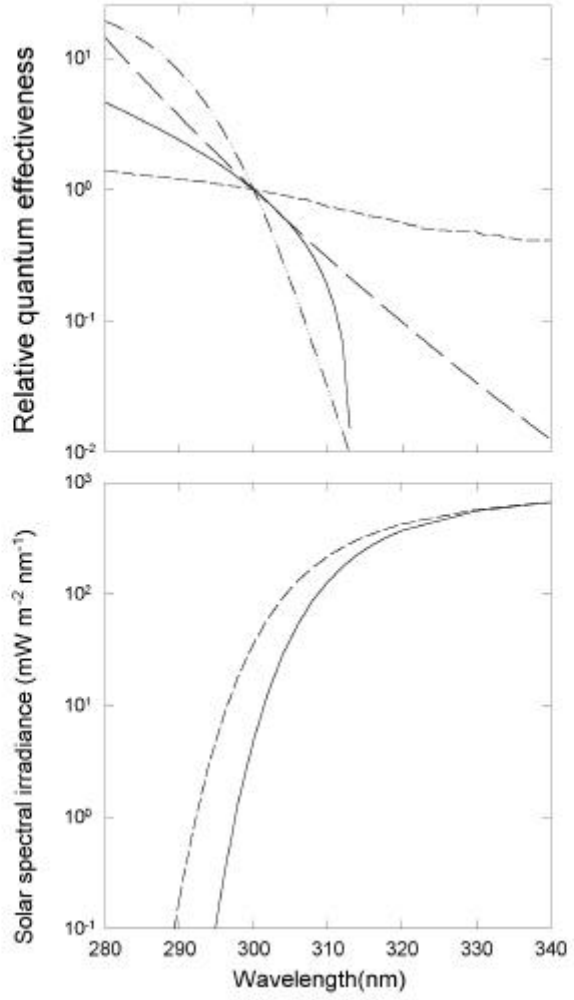

Figure 3.3. (upper panel) Action spectra for “naked” DNA damage (Setlow, 1974) (lines with dashes and dots), DNA dimer formation (a type of DNA damage) in intact alfalfa seedlings (Quaite et al., 1992a)(line with long dashes), a generalized plant action spectrum compiled from various plant spectra (Caldwell, 1971)(continuous line) and a spectrum for putative lipid damage based on a luminescence indicator (Cen and Björn, 1994)(line with short dashes). The lower panel shows solar spectral irradiance at 360 (continuous line) and 180 (dashed line) Dobson Units (DU) of total atmospheric ozone (Dobson Units are an expression used for describing thickness of the ozone layer at standard temperature and pressure (0° and 101.3 Pa); 1 mm ozone layer thickness is equivalent to 100 DU). The solar irradiation is calculated for latitude 49 N at solar noon at the summer solstice (June 21) using the model of Green et al. (1980).

Plant Growth Responses

In many plant species reduced leaf area and/or stem growth have been found in studies carried out in growth chambers, greenhouses and in the field (Tevini and Teramura, 1989; Johanson et al., 1995; Mepsted et al., 1996; Tevini, 1996; Rozema et al., 1997a). These studies have traditionally been conducted with specially filtered UV lamps. It is important in such experiments to maintain a realistic balance between different spectral regions since both UV-A (315-400 nm) and visible (400-700 nm) radiation can have strong ameliorating effects on responses of plants to UV-B (Caldwell et al., 1994). In growth chambers and greenhouses, the radiation conditions are usually quite different from those in nature. For example, the visible radiation which is used in photosynthesis (400 to 700 nm, photosynthetically active radiation, PAR) and the UV-B/UV-A/PAR ratios are different from those in the field. If UV-A and PAR are low, the effects of UV-B may be much more severe. Thus, even if realistic levels of UV-B are used in simulating ozone reduction, the plant response may be exaggerated relative to that in the field. In addition, other factors, such as temperature, water and nutrients differ from conditions in the field and this can alter response to UV-B radiation. It is, however, important that these studies conducted under controlled conditions be verified as much as possible under field conditions. Even under field conditions, if applied UV-B is not adjusted downward during cloudy periods, the UV-B sensitivity may be unduly pronounced (Fiscus and Booker, 1995). Unfortunately, the most expensive and difficult experiments, i.e., those conducted in the field with UV-B supplements adjusted for cloudiness and other atmospheric conditions, are seldom undertaken. In the last few years more field experiments have been conducted and many of these employ lamp systems with controls to make continual adjustments according to prevailing sunlight conditions.

Also, there are several studies in which the UV component of existing sunlight has been altered by special filters in the field or in special small greenhouses or growth chambers located outdoors. The filters have involved special glass, plastics, or in one series of studies, ozone gas in a UV-transparent plexiglass envelope (Tevini et al., 1990; Mark and Tevini, 1997). Many of these studies involving filtered sunlight have shown that normal ambient solar UV-B can cause somewhat reduced leaf area, smaller seedlings, etc. (Searles et al., 1995; Ballaré et al., 1996; Mark et al., 1996; Saile-Mark and Tevini, 1997).

Plant species vary considerably in their response to UV-B in both controlled-environment and field studies. Also, varieties of the same species can vary in their response. For example, in the field, sizeable differences in response to UV-B were found among varieties of soybean (Teramura et al., 1990) and rice (Dai et al., 1992, 1997; Kulandaivelu et al., 1997). Experiments in greenhouses covered by different materials that transmitted different amounts of UV indicated that varieties of bean (*Phaseolus vulgaris*) from lower latitudes were less affected than those from higher latitudes under higher UV-B radiation (Saile-Mark and Tevini 1997).

Plant Reproductive Processes

Ultraviolet-B radiation can alter both the timing of flowering (Caldwell, 1968; Ziska et al., 1992; Staxén and Bornman, 1994; Mark et al., 1996; Tevini, 1996) as well as the number of flowers in certain species (Musil, 1995; Klaper et al., 1996; Saile-Mark and Tevini, 1997). Differences in timing of flowering may have important consequences for the availability of pollinators. Such effects may be due to regulatory alterations in the plant rather than damage *per se*. Poorly protected reproductive organs might, however, be susceptible to damaging effects. Most of the reproductive parts of plants, such as pollen and ovules, are rather well shielded from solar UV-B radiation. For example, anther walls can absorb more than 98% of incident UV-B radiation (Flint and Caldwell, 1983). In addition,

the pollen wall contains UV-B absorbing compounds affording protection during pollination as do the other flower parts such as sepals, petals and walls of the ovaries (Day and Demchik, 1996). Only after transfer to the stigma might pollen be susceptible to solar UV-B radiation. In vitro experiments have shown that germinating pollen can be sensitive at this time to UV-B radiation in some cases (Flint and Caldwell, 1984). However, often pollen germination itself is not affected, but pollen tube growth of many species can be retarded as shown in a survey of 34 plant species or varieties (Torabinejad et al., 1998).

Carry-over Effects of UV-B Irradiation in Subsequent Generations

In sexually reproducing populations of an annual desert plant, effects of UV-B irradiation on growth and allocation of biomass appeared to accumulate as subsequent generations were exposed to UV-B irradiation (Musil, 1996). Furthermore, after four generations of UV-B irradiation, the effects persisted in a fifth generation that was not exposed to UV-B treatment (Musil et al., 1998). If this phenomenon is common, it could amplify the effects of UV-B radiation changes. This is somewhat analogous to apparent accumulated effects of UV-B irradiation over several growing seasons in long-lived woody plants discussed later.

Ecosystem-level UV-B Radiation Effects Involving Higher Plants

Competitive Balance

In forests, grasslands, etc., overall primary plant productivity may not be greatly affected by ozone reduction even if the growth of some plants is diminished. However, since plant species differ greatly in growth responsivity to UV-B, it is anticipated that a productivity reduction of one species will probably lead to increased productivity of another, more UV-tolerant species. This is likely because more resources (e.g., light, moisture and nutrients) will be available to the tolerant species. Thus, the overall productivity of the system may well remain about the same while species composition may change. However, a change in the balance of species could have far-reaching consequences for the character of many ecosystems.

Another mechanism whereby the competitive balance of plant species can be changed by increased UV-B is through changes in plant form. Even if plant production *per se* is not affected by increased UV-B, changes in plant form can result in changes in which species can more effectively compete for sunlight. This phenomenon has been demonstrated in several experiments. For example, in a six-year field study using modulated UV-B lamp systems, the competitive balance of two species (wheat and a common weed, wild oat) could be changed even though the increased UV-B radiation had no effect on production and growth of these species if grown by themselves (Barnes et al., 1988). A quantitative analysis of competition for sunlight in the mixed stands with and without supplemental UV-B showed that subtle changes in plant form of the two species were sufficient to change the balance of competition for sunlight that is necessary for photosynthesis (Barnes et al., 1995). Therefore, one species can achieve some advantage over the other because one captures more sunlight for photosynthesis. In these experiments, the wheat benefited from increased UV-B and the weed suffered. However, in other mixtures of crop and weeds, the situation might be reversed. Also, other changes in plant form, such as greater allocation of biomass to roots, might change competitive effectiveness of individual species for soil moisture and nutrients. In grasslands and forests that are not managed intensively, similar changes in species composition may be experienced.

Ecosystem-level experiments with nonagricultural systems are only beginning. Early reports of one experiment in a subarctic heath ecosystem suggest that species composition changes may result from UV-B supplementation (Johanson et al., 1995.)

Plant Susceptibility to Pathogens and Insects

The extent to which plant tissues are consumed by insects or the degree to which pathogens attack plants is regulated by several properties of the plant host tissues. Experiments in which solar UV-B radiation has been modified by selective filters show that present-day solar UV-B radiation can substantially reduce insect herbivory of agricultural and native plant foliage (Ballaré et al., 1996; Mazza et al., submitted; Rousseaux et al., 1998). Field studies involving supplementation of solar UV-B radiation with lamp systems indicated a substantial reduction in populations of a herbivorous insect on a heathland plant (Salt et al., 1998). The reasons for these changes are not always clear, but they may be mediated through changes in plant secondary chemistry or alterations in plant nitrogen or sugar content. Studies involving UV lamps indicated decreased herbivory by a moth caterpillar under elevated UV-B radiation and this was attributed to increases of host pea plant tissue nitrogen content (Hatcher and Paul, 1994). Mulberry plants previously irradiated with UV from lamps suffered less herbivory by silkworms (*Bombyx mori*) and the lower consumption was attributed to lower sucrose content of the foliage (Yazawa et al., 1992). McCloud and Berenbaum (1994) have shown in laboratory studies that UV-B radiation can increase furanocoumarin content of plant tissue which, in turn, results in slower development of certain insect larvae during early life stages of the larvae. Although the foregoing would suggest that insect herbivory may always be decreased by UV-B radiation, another study shows that herbivory can be increased three-fold (e.g., Buck and Callaghan, submitted).

The results of most of these studies indicate that the effects on insect herbivory are all due to changes in the host plant tissues. However, there are some indications that some insects may respond directly to solar UV-B radiation. Thrips on soybeans were found to consume less foliage if the foliage had been previously exposed to ambient solar UV-B. Furthermore, the thrips appeared to directly sense and avoid solar UV-B radiation even though they were mildly attracted to UV-A radiation (Mazza et al., submitted).

Plant fungal and viral diseases react in several different ways to UV-B radiation in several experiments, conducted primarily in laboratory and greenhouse conditions. In four of ten studies, UV-B was found to counteract disease severity and in the other six studies, it promoted disease development (Manning and Tiedemann, 1995). The direction of the UV-B radiation effect on disease severity can also vary with the variety of the host. In a rust-resistant variety of wheat, additional UV-B radiation had little effect, but it promoted the rust infection in a rust-sensitive wheat variety (Manning and Tiedemann, 1995). It is not clear in many of these experiments whether the changes in disease severity were due simply to changes caused by UV-B radiation in the host plant, or whether direct UV-B radiation effects on the fungal or viral pathogens was involved. Cucumber plants first exposed to UV-B radiation were more susceptible to subsequent infection by two fungal pathogens if the host plants were exposed to UV-B radiation prior to infection; but UV-B irradiation after infection had no effect on disease severity (Orth et al., 1990). Such an experiment suggests the effect of UV-B radiation was mediated through changes in the host plant tissues. There is also evidence from solar UV-B exclusion studies showing increased incidence of fungal disease when UV-B is removed (Gunasekera et al., 1997).

These changes in insect herbivory and disease severity caused by alterations of solar UV-B can be sizeable; they can operate in different directions and have very important implications for both agri-

cultural and nonagricultural ecosystems. They may be much more important than known influences of UV-B radiation on plant production based on realistic field studies.

Even roots of plants whose shoots are exposed to elevated UV-B radiation can be affected as indicated by root interactions with microorganisms. For example, the nature of microorganism assemblages that were associated with roots of sugar maple trees (*Acer saccharum*) was altered by exposure of the tree shoots to elevated UV-B radiation (Klironomos and Allen, 1995). This was obviously a systemic effect of UV-B expressed in the roots of the host plant.

Timing of Life Phases

The timing of life phases of plants is a combination of response to environmental factors and the genetic constitution of the plant. For example, as mentioned earlier, UV-B exposure can alter the timing of flowering. This timing of events such as flowering, entering and breaking of dormancy, and even senescence is important not only to the individual plant, but also in how plants interact with other plants and animals. For example, a shift in the timing of flowering can mean that a plant species might not have sufficient insect pollinators available at the new time of flowering either because the insects are not present or because other plant species are attracting these pollinators. Such changes could also conceivably be important in agricultural systems, but intervention with management options may make these changes less important. As indicated earlier in this chapter, increased UV-B has been shown to advance or delay (depending on species) the time of flowering in plants. There is little work at present on flowering responses and virtually nothing on other potential effects of UV-B on life phase timing of plants or other terrestrial organisms.

Other Effects due to Changes in Higher Plant Tissues

In higher plants, secondary compounds, such as lignin, are important as structural materials. These are related to phenolic compounds and may change in composition with elevated UV-B radiation (e.g., Gehrke et al. 1995). If the ratio of lignin to cellulose in plant tissues changes, it can alter the rate of decomposition. This has very important implications for biogeochemical cycles as discussed fully in Chapter 5.

Ecosystem Effects of Solar UV-B not Mediated by Higher Plants

Although the considerable emphasis on UV-B effects on plants and plant-mediated ecosystem effects is deserved, elevated solar UV-B radiation may affect ecosystem function through other mechanisms (Fig. 3.1). Direct effects of solar UV-B on terrestrial animal life, microbes and the lower atmosphere (Chapter 6) all can have important ecosystem-level implications. Comparatively little study has been directed to effects on animal life apart from humans (Chapter 2).

It has been experimentally established that animals, from fish to mammals, can acquire skin cancer under laboratory conditions and some domestic animals exhibit such symptoms in poorly pigmented body areas (Chapter 2). However, in nature, protection by fur and plumage or behavioral patterns make it unlikely that there will be sufficient UV-B radiation exposure for skin cancer to be a hazard for most animals. There is a possibility of eye damage in animals, but that has not been investigated under field conditions.

Microbes exposed to sunlight play important roles in many ecosystem functions including decomposition of plant litter, diseases of plants and animals, biogeochemical transformations (Chapter 5),

etc. Microbes in several habitats are exposed to sunlight including those that are airborne, or live on soil, litter and foliage surfaces. Gehrke et al. (1995) found that among fungi in plant litter, *Mucor hiemalis* and *Truncatella truncata* were reduced in abundance by UV-B radiation corresponding to pronounced ozone depletion, while *Penicillium brevicompactum* was unaffected by increased UV-B radiation exposure. Cyanobacteria growing on soil surfaces may be important for nitrogen fixation from the air. It is known that their sensitivity to UV-B radiation also varies considerably among species (see Chapters 4 and 5). Leaf surface microorganism assemblages have also been shown to be altered by increased UV-B radiation (Newsham et al., 1997).

When the stratospheric ozone is depleted, solar UV-B penetrates more effectively into the lower atmosphere and can alter local air pollution (Chapter 6), which, in turn can influence terrestrial ecosystems. For example, under conditions of substantial ambient NO_x more ozone can be formed in the lower atmosphere due to ozone reduction in the stratosphere (Chapter 6). It is well known that ambient ozone can adversely affect higher plants (Manning and Tiedemann, 1995). Other nonbiological effects of elevated solar UV-B radiation include accelerated photodestruction of plant litter exposed to sunlight (Chapter 5).

Which Terrestrial Ecosystems Might be Most Affected by Increased UV-B Radiation?

Much of the experimentation has been designed to simulate UV-B levels expected on clear days with unobstructed sunlight, whereas many areas have persistent cloud cover and, correspondingly, lower UV-B flux rates. However, there is some suggestion that plant responsiveness to UV-B may be influenced by the ratio of UV-B:visible sunlight as much as by the absolute level of UV-B radiation (Deckmyn et al., 1994; Deckmyn and Impens, 1997). Certain clouds tend to transmit more radiation at shorter wavelengths than at longer wavelengths (Bordewijk et al., 1995); therefore, the ratio of UV-B:PAR would be greater than under clear-sky conditions. Yet, this has not been documented over extended time periods in different environments. The potential importance of plant responsiveness to greater UV-B:PAR ratios during cloudy periods deserves further attention and ecosystems that occur in cloudy environments should not necessarily be dismissed from consideration for the ozone reduction problem.

Overall, the consequences of increased solar UV-B in forests, grasslands and other nonagricultural ecosystems may involve several complex pathways (Fig. 3.1) rather than simply a reduction in overall ecosystem primary productivity. However, the effects of these more involved pathways are difficult to predict without conducting experiments with assemblages of plant species and long-term study of ecosystem responses. This has, thus far, received very little attention in experimental research.

Where ecosystem-level studies of terrestrial responses to increased solar UV-B have been initiated, high-latitude ecosystems have been emphasized since the relative ozone reduction is more pronounced at high latitudes. Yet, the absolute UV-B flux is greater at low latitudes where ozone reduction is not very pronounced.

Further discussion of implications for specific types of ecosystems follows later in this chapter.

Interaction of UV-B and other Factors

Plants and other organisms in nature seldom are affected by only a single stress factor, such as UV-B radiation. Instead, they typically respond to several factors acting in concert, such as water stress, increased atmospheric CO_2, mineral nutrient availability, heavy metals, tropospheric air pollutants and temperature. Therefore, it is important to keep in mind that the effectiveness of UV-B radiation can be greatly increased or decreased by such factors. Visible radiation is an important ameliorating factor and, thus, as natural levels as possible should be applied in laboratory experiments for attaining more realistic results, as discussed earlier.

Among the most common of factors in nature is water stress. In a field study, Sullivan and Teramura (1990) demonstrated that UV-B mediated reductions in photosynthesis and growth were observed only in well-watered soybeans. When soybeans were water stressed, there was no significant effect of the UV-B radiation on either photosynthesis or growth. The interpretation was that water stress resulted in a large reduction in photosynthesis and growth that masked the UV-B effect. Furthermore, water stressed plants resulted in a higher concentration of leaf flavonoids, which in turn, provided greater UV-B protection. Other interactions between UV-B radiation and water status of plants also occur. Elevated UV-B radiation in field experiments tended to alleviate drought symptoms in two Mediterranean pine species (Petropoulou et al., 1995; Manetas et al., 1997). In a moss species, UV-B radiation inhibited growth when the moss was under water stress, but stimulated growth when the moss was well hydrated (Gehrke, 1998).

Increases of atmospheric CO_2 are a certain element of global climate change and atmospheric CO_2 concentration will likely double by the middle of the next century (Mitchell et al., 1990). Many experiments with elevated CO_2 employ a twice-ambient CO_2 concentration as a treatment condition. Such a doubling often results in more pronounced plant responses than are evident in many elevated UV-B radiation lamp experiments designed to simulate up to 20% ozone column reduction under field conditions. However, responses to CO_2 are small in semi-natural ecosystems where nutrient or water availability may strongly constrain plant growth. For example, Gwynn-Jones et al. (1997) showed that growth responses to elevated CO_2 and enhanced UV-B (both alone and in combination) were small during the first three years of experimentation in a sub-arctic heath. Also, most ecosystem-level effects of elevated CO_2 are mediated through changes in plant tissues. When studied independently, plant growth responses to changes in UV-B radiation and atmospheric CO_2 concentration generally are thought to be in opposite directions. Usually, however, in most experiments employing both elevated CO_2 and UV-B radiation, these factors do not yield interactions, with some exceptions (see reviews by Björn et al., 1997; Sullivan, 1997). Elevated CO_2 sometimes appears to provide some protection against elevated UV-B radiation for some species; yet, elevated UV-B radiation can limit the ability of some species to take advantage of elevated CO_2 in photosynthesis. Allocation of biomass in plants can also change in a complicated fashion with the combination of CO_2 and UV-B radiation treatments (reviewed by Sullivan, 1997). Increased temperature is also a predicted element of global climate change. In a study combining two levels of UV-B radiation with two levels of CO_2 and two temperatures, the results indicated that either elevated CO_2 or somewhat higher temperature had similar effects in reducing the growth-inhibiting effects of elevated UV-B radiation on sunflower and maize seedlings (Mark and Tevini 1997).

Plant uptake and translocation of mineral nutrients within the plant can be affected by elevated UV-B radiation, but the mineral nutrient status of plants also can affect plant responsiveness to UV-B radiation (Murali and Teramura, 1985; Ros, 1995; Musil and Wand, 1994). Nitrogen concentration in plant tissues can increase under elevated UV-B which has been linked with reduced insect herbivory

(Hatcher and Paul, 1994; Rousseaux et al., 1998). The uptake of certain nutrients may also be modified by UV-B radiation and cadmium. In oilseed rape (*Brassica napus)* plants grown under additional enhanced UV-B radiation and simultaneously exposed to different concentrations of cadmium, the manganese content in the shoots decreased in plants exposed to cadmium and UV-B radiation, while significant increases in magnesium, calcium, phosphate, copper and potassium occurred only in those plants exposed to cadmium and UV-B radiation. Cadmium uptake was not affected by UV-B radiation. The UV-B had no additional influence on the nutrient content of the roots (Larsson et al., 1998). An earlier study showed that both cadmium and UV-B radiation negatively influenced photosynthetic efficiency in spruce seedlings (Dubé and Bornman, 1992).

Interaction of UV-B radiation with tropospheric air pollutants is also of concern although little work thus far has been conducted in this area. One field study of soybean plants showed them to be sensitive to ozone in the air, but not sensitive to UV-B supplements from lamps under the particular test conditions. There were no significant interactions of supplemental UV-B and ozone (Miller et al., 1994). However, in pine seedlings grown in a growth cabinet with a simulated solar UV radiation, increasing the ozone concentration increased the sensitivity of the pine seedlings to UV-B radiation since the ozone reduced the levels of UV-B-absorbing pigments in the plant tissues. In another experiment with tobacco, UV-B radiation increased the level of ozone-induced foliage lesions (Thalmair et al., 1996).

Implications for Agriculture, Forests and other Ecosystems

Crops

One of the primary concerns of future increases in solar UV-B radiation is its potential effect on global agriculture. Despite the obvious potential consequences of the issue, we cannot make quantitative predictions of anticipated effects resulting from stratospheric ozone depletion. This is due to the limitation in controlled-environment studies as discussed earlier and the overall paucity of well-replicated experiments performed in the field. Even in comparisons of field studies, there are large differences in temperature, precipitation, soil types, etc. from year to year and in different locations. This adds to the difficulty in making generalizations about the effects. Also, a common finding is that different varieties of the same crop species often react differently to elevated UV-B radiation (Fig. 3.4).

The general procedure in such field experiments is to supplement ambient sunlight with special fluorescent UV lamps filtered to supply either extra UV-B radiation (treatment) or with the UV-B removed (control). The methodology has continuously been improved, e.g., by introduction of automatic systems that change the lamp output to more realistically simulate the UV-B supplement with proper balance with the existing sunlight. Therefore, older experiments, and especially those performed in glasshouse or growth chamber conditions, are presently considered to be less reliable.

The compilation of harvestable yield in field experiments in Figure 3.4 indicates how variably different varieties responded and also that many varieties did not respond in a significant manner (statistically speaking) and a very few were even stimulated in production. From the entire population of studies, there is a tendency toward more negative effects.

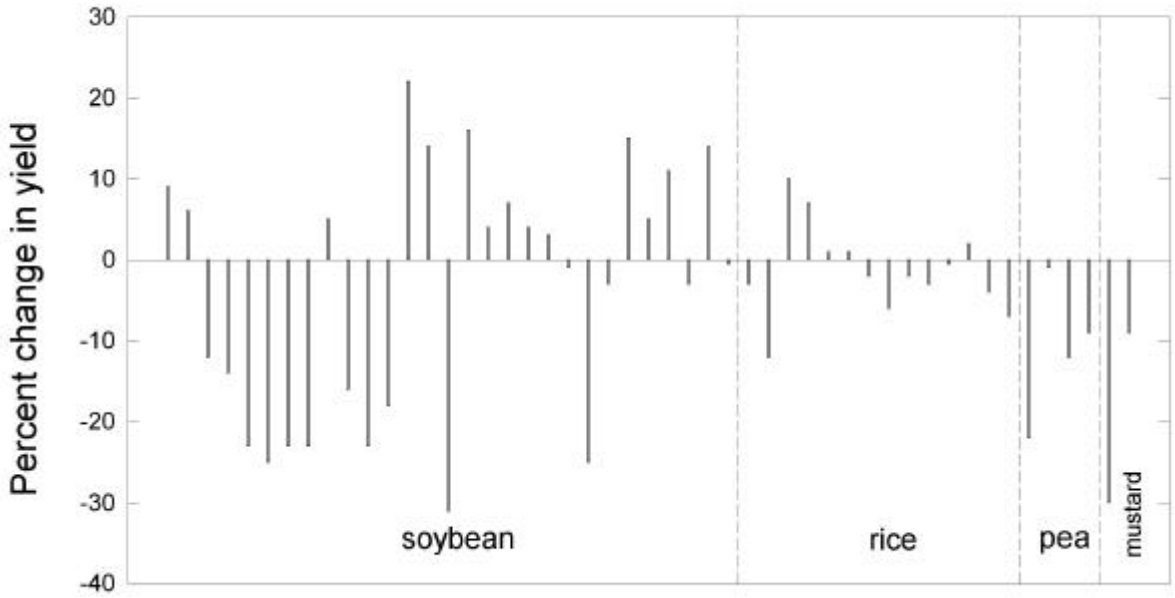

Fig. 3.4. Relative changes in yield (seed production) of four crops evaluated for UV-B radiation responsiveness in 49 field trials with UV-B supplementation from lamps. Each bar represents results obtained with one variety in one field experiment in which ozone depletion was simulated (usually ca. 20% depletion). Soybean data from Teramura and Murali (1986), Sinclair et al. (1990), Teramura et al. (1990), D'Surney et al. (1993), and Miller et al. (1994); rice data from Nouchi et al. (1997) and Olszyk et al. (1996); pea data from Mepsted et al. (1996); and mustard data from Conner and Zangori (1997). Most effects smaller than 10% were not statistically significant, but small sample sizes and other environmental factors may have obscured differences.

In addition to quantitative changes in crop yield, evidence exists for qualitative changes as well. For instance, in the study mentioned above, UV-B radiation also resulted in small changes on the order of 1 to 5% in the protein and oil content of the soybean seed (Teramura et al., 1990).

Because of the broad range of response patterns in crop species, plant breeding and genetic engineering for UV tolerance is an important aspect to consider in order to avoid significant crop production losses. There may, however, be some qualitative changes in seed or foliage characteristics that accompany the development or use of more UV-B-tolerant varieties. This remains to be explored. Other agroecosystem consequences of elevated UV-B radiation are likely to be more important, such as changes in insect or pathogen susceptibility of crops.

Forests

Relatively little information exists on the effects of UV-B radiation on forest tree species. Tropical forests, though representing nearly one half of global productivity and much of the total tree species diversity, have received very little attention with respect to the ozone reduction problem. Although little, or no, ozone reduction has thus far occurred in the tropics, only a small decrease of ozone at these latitudes would result in a very sizeable absolute increase of UV-B radiation since solar UV-B radiation is already very intense in these regions (see Chapter 1). One study has shown that excluding existing solar UV-B radiation with filters can result in increased growth of some tropical tree species (Searles et al., 1995). Otherwise, the effects of UV-B radiation on tropical tree species have not received much attention.

Fortunately, there is some information for mid-temperate latitude tree species. Because they are long-lived, trees present the opportunity to observe the longer-term cumulative effects of UV-B exposure over several years for the same individuals. These cannot be explored in annual crop species. In a field study using loblolly pine (Sullivan and Teramura, 1992), seedlings from several different geographic regions were grown for three consecutive years under UV-B lamps in a field experiment. Seedlings were exposed to either ambient solar UV-B or ambient levels supplemented with the UV-B from lamps, similar to studies with soybean yield (Teramura et al., 1990). After the first year of UV-B exposure, reductions were observed in biomass of seedlings derived from several geographic areas. By the end of the third year, these biomass reductions were several-fold larger in one variety. These overall growth reductions were generally associated with small decreases in both roots and shoots, but not necessarily accompanied by reductions in photosynthesis. This may be due to changes in needle growth or shifts in allocation of biomass as has been found for some crop species. These results suggested that the effects of UV-B radiation may accumulate in long-lived plants such as trees.

The fact that decreases in conifer needle biomass and needle length and leaf area of broadleaf trees were not accompanied by sizeable reductions of photosynthesis (Sullivan and Teramura, 1992; Dillenburg et al., 1995; Sullivan et al., 1996) may be due to the very low penetration of UV-B radiation into older foliage. It appears that the decreased growth of leaves and conifer needles upon exposure to enhanced levels of UV-B radiation may be due, in part, to epidermal cell wall thickening. This might prevent cell wall extension and, thereby, growth of these cells (Liu and McClure, 1995; Sullivan et al., 1996). Thus, changes at the level of the epidermis, the first leaf cell layer to receive the incident radiation, can have other important consequences.

Other Ecosystems at Mid and High Latitudes

Although absolute UV-B irradiance is naturally very low in high-latitude ecosystems, such as tundra and subarctic areas, there is experimental evidence that the plants in such systems react to increases in UV-B associated with realistic levels of ozone depletion. Some plant species exhibit growth inhibitions and others do not, thus, eventually altered community composition may be expected (Johanson et al. 1995, Rousseaux et al., 1998, Searles et al., 1998). Longer-term observations of species composition are being pursued in high-latitude subarctic systems in Sweden, a high arctic site on Spitzbergen Island and in southernmost Argentina (Tierra del Fuego). In the latter system, attenuating the naturally occurring solar UV-B radiation increased insect herbivory, decreased plant tissue nitrogen concentrations, and increased populations of some microfauna (amoeba and rotifers) that inhabit peat bogs (Rousseaux et al., 1998; Searles et al., 1998). The subarctic studies in Sweden have been underway for several years and these show several effects including decreased litter decomposition (Gehrke et al. 1995), increased fruit formation and greatly increased insect herbivory (Gwynn-Jones et al., 1997; Buck and Callaghan, submitted). Similar ecosystem studies are underway in a mid-latitude site in the Netherlands where dune grasslands are important (Rozema, et al. 1997a).

Although terrestrial ecosystems at high latitudes are not highly productive for grazing, timber production, etc., the influence of ozone reduction on these systems may be important for several reasons. Carbon sequestration is generally quite high in these ecosystems, including the extensive peat formations which are also being studied in the Swedish subarctic and southern Argentinean systems. Compared with other locations, these ecosystems are under the greatest ozone depletion, especially in the Southern Hemisphere, and they also experience the greatest warming as the global greenhouse effect intensifies. Thus, they are sensitive indicators of several features of climate change. These high latitude ecosystems are also very important for the survival of indigenous ethnic groups in the Northern Hemisphere.

References

Ballaré, C.L., Scopel, A.L., Stapleton, A.E. and Yanovsky, M.J. 1996. Solar ultraviolet-B radiation affects seedling emergence, DNA integrity, plant morphology, growth rate, and attractiveness to herbivore insects in *Datura ferox. Plant Physiology* 112:161-170.

Barnes, P.W., Jordan, P.W., Gold, W.G., Flint, S.D. and Caldwell, M.M. 1988. Competition, morphology and canopy structure in wheat (*Triticum aestivum* L.) and wild oat (*Avena fatua* L.) exposed to enhanced ultraviolet-B radiation. *Functional Ecology* 2:319-330.

Barnes, P.W., Flint, S.D. and Caldwell, M.M. 1995. Early-season effects of supplemented solar UV-B radiation on seedling emergence, canopy structure, simulated stand photosynthesis and competition for light. *Global Change Biology* 1:43-53.

Bender, K., Blattner, C., Knebel, A., Lordanov, M., Herrlich, P. and Rahmsdorf, H.J. 1997. UV-induced signal transduction. *Journal of Photochemistry and Photobiology B* 37:1-17.

Björn, L.O. 1996. Effects of ozone depletion and increased UV-B on terrestrial ecosystems. *International Journal of Environmental Studies* 51:217-243.

Björn, L.O., Callaghan, T.V., Johnsen, I., Lee, J.A., Manetas, Y., Paul, N.D., Sonesson, M., Wellburn, A.R., Coop, D., Heide-Jørgensen, H.S., Gehrke, C., Gwynn-Jones, D., Johanson, U., Kyparissis, A., Levizou, E., Nikolopoulos, D., Petropoulou, Y. and Stephanou, M. 1997. The effects of UV-B radiation on European heathland species. *Plant Ecology* 128:252-264.

Blaustein, A.R., Hoffman, P.D., Hokit, D.G., Kiesecker, J.M., Walls, S.C. and Hays, J.B. 1994. UV repair and resistance to solar UV-B in amphibian eggs: A link to population declines? *Proceedings of the National Academy of Science* 91:1791-1795.

Bordewijk, J., Slaper, H., Reinen, H.A.J.M. and Schlamann, E. 1995. Total solar radiation and the influence of clouds and aerosols on the biologically effective UV. *Geophysical Research Letters* 22:2151-2154.

Bornman, J.F. 1989. Target sites of UV-B radiation in photosynthesis of higher plants. *Journal of Photochemistry and Photobiology B: Biology* 4:145-158.

Bornman, J.F. and Sundby-Emanuelsson, C. 1995. Response of plants to UV-B radiation: some biochemical and physiological effects. In *Environment and Plant Metabolism*, Smirnoff, N. (ed.), BioScientific, Oxford, pp. 245-262.

Bornman, J.F. and Teramura, A.H. 1993. Effects of ultraviolet-B radiation on terrestrial plants. In *Environmental UV-Photobiology*, Young, A.R., Björn, L.O., Moan, J. and Nultsch, W. (eds.), Plenum Press, New York, pp. 427-471.

Bornman, J.F. and Vogelmann, T.C. 1991. Effect of UV-B radiation on leaf optical properties measured with fibre optics. *Journal of Experimental Botany* 42:547-554.

Britt, A.B. 1996. DNA damage and repair in plants. *Annual Review of Plant Physiology and Plant Molecular Biology* 47:75-100.

Buck, N. and Callaghan, T.V. (submitted). The direct and indirect effects of enhanced UV-B on the moth caterpillar *Epirrita autumnata*. *Ecological Bulletins.*

Caldwell, M.M. 1968. Solar ultraviolet radiation as an ecological factor for alpine plants. *Ecological Monographs* 38:243-268.

Caldwell, M.M. 1971. Solar ultraviolet radiation and the growth and development of higher plants. In *Photophysiology*, Giese, A.C. (ed.), volume 6, Academic Press, New York, pp. 131-177.

Caldwell, M.M. and Flint, S.D. 1993. Implications of increased solar UV-B for terrestrial vegetation. In The Role of the Stratosphere in Global Change, Chanin, M.L. (eds.), Springer-Verlag, Heidelberg, pp. 495-516.

Caldwell, M.M. and Flint, S.D. 1994a. Solar ultraviolet radiation and ozone layer change: Implications for crop plants. In *Physiology and Determination of Crop Yield*, Boote, K., Bennett, J.M., Sinclair, T.R. and Paulsen, G.M. (eds.), ASA-CSSA-SSSA, Madison, WI, pp. 487-507.

Caldwell, M.M. and Flint, S.D. 1994b. Stratospheric ozone reduction, solar UV-B radiation and terrestrial ecosystems. Climatic Change 28:375-394.

Caldwell, M.M. and Flint, S.D. 1997. Uses of biological spectral weighting functions and the need for scaling for the ozone reduction problem. *Plant Ecology* 128:66-76.

Caldwell, M.M., Camp, L.B., Warner, C.W. and Flint, S.D. 1986. Action spectra and their key role in assessing biological consequences of solar UV-B radiation change. In *Stratospheric Ozone Reduction, Solar Ultraviolet Radiation and Plant Life*, Worrest, R.C. and Caldwell, M.M. (eds.), Springer, Berlin, pp. 87-111.

Caldwell, M.M., Flint, S.D. and Searles, P.S. 1994. Spectral balance and UV-B sensitivity of soybean: a field experiment. *Plant, Cell and Environment* 17:267-276.

Caldwell, M.M., Teramura, A.H. and Tevini, M. 1989. The changing solar ultraviolet climate and the ecological consequences for higher plants. *Trends in Ecology and Evolution* 4:363-367.

Cen, Y.-P. and Björn, L.O. 1994. Action spectra for ultraweak luminescence by ultraviolet radiation (270-340 nm) in leaves of *Brassica napus. Journal of Photochemistry and Photobiology, B. Biology* 22:125-129.

Cen Y-P., Weissenböck G. and Bornman J.F. 1993. The effects of UV-B radiation on phenolic compounds and photosynthesis in leaves of *Brassica napus*. In *Physical, Biochemical and Physiological Effects of Ultraviolet Radiation on Brassica napus and Phaseolus vulgaris*, PhD thesis, Lund, Sweden.

Conner, J.K. and Zaingori, L.A. 1997. A garden study of the effects of ultraviolet-B radiation on pollination success and lifetime female fitness in *Brassica. Oecologia* 111:388-395.

Crutzen, P.J. 1972. SSTs--a threat to the earth's ozone shield. *Ambio* 1:41-51.

Dai, Q.J., Coronel, V.P., Vergara, B.S., Barnes, P.W. and Quintos, A.T. 1992. Ultraviolet-B radiation effects on growth and physiology of four rice cultivars. *Crop Science* 32:1269-1274.

Dai, Q., Peng, S., Chavez, A.Q., Miranda, M.L.L., Vergara, B.S. and Olszyk, D.M. 1997. Supplemental ultraviolet-B radiation does not reduce growth or grain yield in rice. *Agronomy Journal* 89:793-799.

Day, T.A. and Demchik, S.M. 1996. Influence of enhanced UV-B radiation on biomass allocation and pigment concentrations in leaves and reproductive structures of greenhouse-grown *Brassica rapa*. *Vegetatio* 127:109-116.

Day, T.A., Vogelmann, T.C. and DeLucia, E.H. 1992. Are some plant life forms more effective than others in screening out ultraviolet-B radiation? *Oecologia* 92:513-519.

Deckmyn, G., Martens, C. and Impens, I. 1994. The importance of the ratio UV-B/photosynthetic active radiation (PAR) during leaf development as determining factor of plant sensitivity to increased UV-B irradiance: effects on growth, gas exchange and pigmentation of bean plants (*Phaseolus vulgaris* cv. Label). *Plant, Cell and Environment* 17:295-301.

Deckmyn, G. and Impens, I. 1997. The ratio UV-B photosynthetically active radiation (PAR) determines the sensitivity of rye to increased UV-B radiation. *Environmental and Experimental Botany* 37:3-12.

DeLucia, E.H., Day, T.A. and Vogelmann, T.C. 1991. Ultraviolet-B radiation and the Rocky Mountain environment: measurement of incident light and penetration into foliage. *Current Topics in Plant Biochemistry and Physiology* 10:32-48.

DeLucia, E.H., Day, T.A. and Vogelmann, T.C. 1992. Ultraviolet-B and visible light penetration into needles of two species of subalpine conifers during foliar development. *Plant, Cell and Environment* 15:921-929.

Dillenburg, L.R., Sullivan, J.H. and Teramura, A.H. 1995. Leaf expansion and development of photosynthetic capacity and pigments in *Liquidambar styraciflua*- effects of UV-B radiation. *American Journal of Botany* 82:878-885.

D'Surney, S.J., Tschaplinski, T.J., Edwards, N.T. and Shugart, L.R. 1993. Biological responses of two soybean cultivars exposed to enhanced UV-B radiation. *Environmental and Experimental Botany* 33:347-356.

Dubé, S.L. and Bornman, J.F. 1992. Response of spruce seedlings to simultaneous exposure to ultraviolet-B radiation and cadmium.. *Plant Physiology and Biochemistry* 30:761-767.

Fiscus, E.L. and Booker, F.L. 1995. Is increased UV-B a threat to crop photosynthesis and productivity? *Photosynthesis Research* 43:81-92.

Flint, S.D. and Caldwell, M.M. 1983. Influence of floral optical properties on the ultraviolet radiation environment of pollen. *American Journal of Botany* 70:1416-1419.

Flint, S.D. and Caldwell, M.M. 1984. Partial inhibition of in vitro pollen germination by simulated solar ultraviolet-B radiation. *Ecology* 65:792-795.

Flint, S. D. and Caldwell, M.M. 1996. Scaling plant ultraviolet spectral responses from laboratory action spectra to field spectral weighting factors. *Journal of Plant Physiology* 148:107-114.

Gehrke, C. 1998. Impacts of enhanced ultraviolet-B radiation on mosses with contrasting morphology and growth patterns in a subarctic heath ecosystem. *Ecology* (in press).

Gehrke, C., Johanson, U., Callaghan, T., Chadwick, D. and Robinson, C.H. 1995. The impact of enhanced ultraviolet-B radiation on litter quality and decomposition processes in *Vaccinium* leaves from the Subarctic. *Oikos* 72:213-222.

Green, A.E.S., Cross, K.R. and Smith, L.A. 1980. Improved analytic characterization of ultraviolet skylight. *Photochemistry and Photobiology* 31:59-65.

Gunasekera, T.S., Paul, N.D. and Ayres, P.G. 1997. The effects of ultraviolet-B (UV-B: 290-320 nm) radiation on blister blight disease of tea (*Camellia sinensis*). *Plant Pathology* 46:179-185.

Gwynn-Jones, D., Lee, J.A. and Callaghan, T.V. 1997. Effects of enhanced UV-B radiation and elevated carbon dioxide concentrations on a sub-arctic forest heath ecosystem. *Plant Ecology* 128:242-249.

Hatcher, P.E. and Paul, N.D. 1994. The effect of elevated UV-B radiation on herbivory of pea by *Autographa gamma*. *Entomologia Experimentalis et Applicata* 71:227-233.

Huang, S., Dai, Q., Peng, S., Chavez, A.Q., Miranda, M.L.L., Visperas, R.M. and Vergara, B.S. 1997. Influence of supplemental ultraviolet-B on indoleacetic acid and calmodulin in the leaves of rice (*Oryza sativa* L.). *Plant Growth Regulation* 21:59-64.

Jagger, J. 1981. Near-UV radiation effects on microorganisms. *Photochemistry and Photobiology* 34:761-768.

Jenkins, G.I., Fuglevand, G. and Christie, J.M. 1997. UV-B perception and signal transduction. In *Plants and UV-B: Responses to Environmental Change*, P. Lumsden (ed.), Cambridge Univ. Press, UK, pp. 135-156.

Johanson, U., Gehrke, C., Björn, L.O., Callaghan, T.V. and Sonesson, M. 1995. The effects of enhanced UV-B radiation on a subarctic heath system. *Ambio* 24:106-111.

Johnston, H.S. 1971. Reduction of stratospheric ozone by nitrogen oxide catalysts from supersonic transport exhaust. *Science* 173:517-522.

Jordan, B.R. 1996. The effects of ultraviolet-B radiation on plants: A molecular perspective. *Advances in Botanical Research* 22:97-162.

Klaper, R., Frankel, S. and Berenbaum, M.R. 1996. Anthocyanin content and UVB sensitivity in *Brassica rapa*. *Photochemistry and Photobiology* 63:811-813.

Klironomos, J.N. and Allen, M.F. 1995. UV-B-mediated changes in below-ground communities associated with the roots of *Acer saccharum. Functional Ecology* 9:923-930.

Krupa, S.V. and Kickert, R.N. 1989. The greenhouse effect - impacts of ultraviolet-B (UV-B) radiation, carbon dioxide (CO_2), and ozone (O_3) on vegetation. *Environmental Pollution* 61:263-393.

Kulandaivelu, G., Lingakumar, K. and Premkumar, A. 1997. UV-B radiation. In *Plant Ecophysiology*, Prasad, M.N.V. (ed.), John Wiley & Sons, Inc, pp. 41-60.

Larsson, E.H., Bornman, J.F. and Asp, H. 1998. Influence of UV-B radiation and Cd^{2+} on chlorophyll fluorescence, growth and nutrient content in *Brassica napus*. *Journal of Experimental Botany* 49:1031-1039.

Li, J., Ou-Lee, T-M., Raba, R., Amundson, R.G. and Last, R.L. 1993. *Arabidopsis* flavonoid mutants are hypersensitive to UV-B irradiation. *Plant Cell* 5:171-179.

Liu, L. and McClure, J.W. 1995. Effects of UV-B on activities of enzymes of secondary phenolic metabolism in barley primary leaves. *Physiologia Plantarum* 93:734-739.

Liu, L., Gitz, D.C. and McClure, J. W. 1995. Effects of UV-B on flavonoids, ferulic acid, growth and photosynthesis in barley primary leaves. *Physiologia Plantarum* 93:725-733.

McCloud, E.S. and Berenbaum, M.R. 1994. Stratospheric ozone depletion and plant-insect interactions: Effects of UVB radiation on foliage quality of *Citrus jambhiri* for *Trichoplusia ni*. *Journal of Chemical Ecology* 20:525-539.

Mackerness, S., A.-H., Surplus, S.L., Jordan, B.R. and Thomas, B. 1998. Effects of supplementary ultraviolet-B radiation on photosynthetic transcripts at different stages of leaf development and light levels in pea (*Pisum sativum* L.): Role of active oxygen species and antioxidant enzymes. *Photochemistry and Photobiology* 68:88-96.

Manetas, Y., Petropoulou, Y., Stamatakis, K., Nikolopoulos, D., Levizou, E., Psaras, G. and Karabournioutis, G. 1997. Beneficial effects of enhanced UV-B under field conditions: improvement of needle water relations and surviving capacity of *Pinus pinea* L. seedlings during the dry Mediterranean summer. *Plant Ecology* 128:100-108.

Manning, W.J. and Tiedemann, A.V. 1995. Climate change: Potential effects of increased atmospheric carbon dioxide (CO_2), ozone (O_3), and ultraviolet-B (UV-B) radiation on plant diseases. *Environmental Pollution* 88:219-245.

Mark, U. and Tevini, M. 1997. Effects of solar ultraviolet-B radiation, temperature and CO_2 on growth and physiology of sunflower and maize seedlings. *Plant Ecology* 128:224-234.

Mark, U., Saile-Mark, M. and Tevini, M. 1996. Effects of solar UVB radiation on growth, flowering and yield of central and southern European maize cultivars (*Zea mays* L.). *Photochemistry and Photobiology* 64:457-463.

Mazza, C., Zavala, J., Scopel, A. and Ballaré, C.L. 1998. Perception of solar ultraviolet-B radiation by photophagous insects: Mechanisms and ecosystem implications. *Proceedings of the National Academy of Sciences*, (in press).

Mepsted, R., Paul, N.D., Stephen, J., Corlett, J.E., Nogués, S., Jones, N.R. and Ayres, P.G. 1996. Effects of enhanced UV-B radiation on pea (*Pisum sativum* L.) grown under field conditions in the UK. *Global Change Biology* 2:325-334.

Miller, J.E., Booker, F.L., Fiscus, E.L., Heagle, A.S., Pursley, W.A., Vozzo, S.F. and Heck, W.W. 1994. Ultraviolet-B radiation and ozone effects on growth, yield, and photosynthesis of soybean. *Journal of Environmental Quality* 23:83-91.

Mitchell, J.F.B., Mananbe, S., Meleshko, V. and Tokioka, T. 1990. Equilibrium climate change and its implications for the future. In *Climate Change. The IPCC Scientific Assessment*, Houghton, J.T., Jenkins, G.J. and Ephraums, J.J. (eds.), Cambridge University Press, Cambridge.

Murali, N.S. and Teramura, A.H. 1985. Effects of ultraviolet-B irradiance on soybean. VII. Biomass and concentration and uptake of nutrients at varying P supply. *Journal of Plant Nutrition* 8:177-192.

Musil, C.F. 1995. Differential effects of elevated ultraviolet-B radiation on the photochemical and reproductive performances of dicotyledonous and monocotyledonous arid-environment ephemerals. *Plant, Cell and Environment* 18:844-854.

Musil, C.F. 1996. Accumulated effect of elevated ultraviolet-B radiation over multiple generations of the arid-environment annual *Dimorphotheca sinuata* DC. (Asteraceae). *Plant, Cell and Environment* 19:1017-1027.

Musil, C.F. and Wand, S.J.E. 1994. Differential stimulation of an arid-environment winter ephemeral *Dimorphotheca pluvialis* (L.) Moench by ultraviolet-B radiation under nutrient limitation. *Plant, Cell and Environment* 17:245-255.

Musil, C.F., Midgley, G.F. and Wand, S.J.E. 1998. Carryover of enhanced ultraviolet-B exposure effects to successive generations of a desert annual: interaction with atmospheric CO_2 and nutrient supply. *Global Change Biology* (in press).

Nouchi, I., Kobayashi, K. and Kim, H.Y. 1997. Effects of enhanced UV-B radiation on growth and yield of rice in the field. *Journal of Agricultural Meteorology* 52:867-870.

Newsham, K.K., Low, M.N.R., McLeod, A.R., Greenslade, P.D. and Emmett, B.A. 1997. Ultraviolet-B radiation influences the abundance and distribution of phylloplane fungi on pedunculate oak (*Quercus robur*). *New Phytologist* 136:287-297.

Olsson, L.C., Veit, M., Weissenböck, G. and Bornman, J.F. 1998. Differential flavonoid response to enhanced UV-B radiation in *Brassica napus*. *Phytochemistry* (in press).

Olszyk, D., Dai, Q., Teng, P., Leung, H., Luo, Y. and Peng, S. 1996. UV-B effects on crops: response of the irrigated rice ecosystem. *Journal of Plant Physiology* 148:26-34.

Orth, A.B., Teramura, A.H. and Sisler, H.D. 1990. Effects of ultraviolet-B radiation on fungal disease development in *Cucumis sativus*. *American Journal of Botany* 77:1188-1192.

Panten, H., Schnitzler, J.-P. and Steinbrecher, R. 1996. Wirkung von Ultraviolettstrahlung auf Pflanzen. *Naturwissenschaftlicher Rundschau* 9:343-348.

Petropoulou, Y., Kyparissis, A., Nikolopoulos, D. and Manetas, Y. 1995. Enhanced UV-B radiation alleviates the adverse effects of summer drought in two Medieterranean pines under field conditions. *Physiologia Plantarum* 94:37-44.

Predieri, S., Krizek, D.T., Wang, C.Y., Mirecki, R.M. and Zimmerman, R.H. 1993. Influence of UV-B radiation on developmental changes, ethylene, CO_2 flux and polyamines in cv. doyenne d'hiver pear shoots grown *in vitro*. *Physiologia Plantarum* 87:109-117.

Quaite, F.E., Sutherland, B.M. and Sutherland, J.C. 1992a. Action spectrum for DNA damage in alfalfa lowers predicted impact of ozone depletion. *Nature* 358:576-578.

Quaite, F.E., Sutherland, B.M. and Sutherland, J.C. 1992b. Quantitation of pyrimidine dimers in DNA from UV-B-irradiated alfalfa (*Medicago sativa* L.) seedlings. *Applied and Theoretical Electrophoresis* 2:171-175.

Reuber, S., Bornman, J.F. and Weissenböck, G. 1996. A flavonoid mutant of barley *Hordeum vulgare* L.) exhibits increased sensitivity to UV radiation in the primary leaf. *Plant Cell and Environment* 19:593-601.

Ros, J. 1995. Synergistic and/or antagonistic effects of enhanced (reduced) UV-B radiation (artificial, solar) at various nitrogen levels in crop plants. In *First European Symposium on the Effects of Environmental UV-B Radiation*, Bauer, H. and Nolan, C. (eds.), Brussels, Luxembourg, pp. 245-252.

Ros, J. and Tevini, M. 1995. UV-radiation and indole-3-acetic acid: Interactions during growth of seedlings and hypocotyl segments of sunflower. *Journal of Plant Physiology* 146:295-305.

Rousseaux, M.C., Ballaré, C.L., Scopel, A.L., Searles, P.S. and Caldwell, M.M. 1998. Solar ultraviolet-B radiation affects plant-insect interactions in a natural ecosystem of Tierra del Fuego (southern Argentina). *Oecologia* (in press).

Rozema, J., van de Staaij, J.W.M. and Tosserams, M. 1997a. Effects of UV-B radiation on plants from agro- and natural ecosystems. In *Plants and UV-B: Responses to Environmental Change*, Lumsden, P.J. (ed.), Cambridge University Press, Cambridge, pp 213-232.

Rozema, J., van de Staaij, J., Björn, L.O. and Caldwell, M.M. 1997b. UV-B as an environmental factor in plant life: stress and regulation. *Trends in Ecology and Evolution* 12:22-28.

Saile-Mark, M. and Tevini, M. 1997. Effects of solar UV-B radiation on growth, flowering and yield of central and southern European bush bean cultivars (*Phaseolus vulgaris* L.). *Plant Ecology* 128:114-125.

Salt, D.T., Moody, S.A., Whittaker, J.B. and Paul, N.D. 1998. Effects of enhanced UVB on populations of the phloem feeding insect *Strophingia ericae* (Homoptera: Psylloidea) on heather (*Calluna vulgaris*). *Global Change Biology* 4:91-96.

Searles, P.S., Caldwell, M.M. and Winter, K. 1995. Response of five tropical dicotyledon species to natural solar ultraviolet-B radiation. *American Journal of Botany* 82:445-453.

Searles, P.S., Flint, S.D., Diaz, S.B., Rousseaux, M.C., Ballaré, C.L. and Caldwell, M.M. 1998. Solar ultraviolet-B radiation influence on *Sphagnum* bog and *Carex* fen ecosystems: first field season findings in Tierra del Fuego, Argentina. *Global Change Biology* (in press).

Setlow, R.B. 1974. The wavelengths in sunlight effective in producing skin cancer: a theoretical analysis. *Proceedings of the National Academy of Science USA* 71:3363-3366.

Sinclair, T. R., N'Diaye, O. and Biggs, R.H. 1990. Growth and yield of field-grown soybean in response to enhanced exposure to ultraviolet-B radiation. *Journal of Environmental Quality* 19:478-481.

Stapleton, A.E., Thornber, C.S. and Walbot, V. 1997. UV-B component of sunlight causes measurable damage in field-grown maize (*Zea mays* L): Developmental and cellular heterogeneity of damage and repair. *Plant, Cell and Environment* 20:279-290.

Stapleton, A.E. 1992. Ultraviolet radiation and plants: burning questions. *The Plant Cell* 4:1353-1358.

Staxén, I. and Bornman, J.F. 1994. A morphological and cytological study of *Petunia hybrida* exposed to UV-B radiation. *Physiologia Plantarum* 91:735-740.

Steinmüller, D. 1986. On the effect of ultraviolet radiation (UV-B) on leaf surface structure and on the mode of action of cuticular lipid biosynthesis in some crop plants. *Karlsruher Beiträge zur Entwicklungs- und Ökophysiologie der Pflanzen* 6:1-174.

Strid, A., Chow, W.S. and Anderson, J.M. 1994. UV-B damage and protection at the molecular level in plants. *Photosynthesis Research* 39:475-489.

Sullivan, J.H. 1997. Effects of increasing UV-B radiation and atmospheric CO_2 on photosynthesis and growth: implications for terrestrial ecosystems. *Plant Ecology* 128:194-206.

Sullivan, J.H. and Teramura, A.H. 1990. Field study of the interaction between solar ultraviolet-B radiation and drought on photosynthesis and growth in soybean. Plant Physiology 92:141-146.

Sullivan, J.H. and Teramura, A.H. 1992. The effects of ultraviolet-B radiation on loblolly pine. 2. Growth of field-grown seedlings. *Trees* 6:115-120.

Sullivan, J. H., Howells, B.W., Ruhland, C.T. and Day, T.A. 1996. Changes in leaf expansion and epidermal screening effectiveness in *Liquidambar styraciflua* and *Pinus taeda* in response to UV-B radiation. *Physiologia Plantarum* 98:349-357.

Takeuchi, Y., Murakami, M., Nakajima, N., Kondo, N. and Nikaido, O. 1996. Induction and repair of damage to DNA in cucumber cotyledons irradiated with UV-B. *Plant and Cell Physiology* 37:181-187.

Taylor, R.M., Nikaido, O., Jordan, B.R., Rosamond, J., Bray, C.M. and Tobin, A.K. 1996.

Ultraviolet-B-induced DNA lesions and their removal in wheat (*Triticum aestivum* L.) leaves

Plant, Cell and Environment 19:171-181.

Teramura, A.H. and Murali, N.S. 1986. Intraspecific differences in growth and yield of soybean exposed to ultraviolet-B radiation under greenhouse and field conditions. *Environmental and Experimental Botany* 26:89-95.

Teramura, A.H. and Sullivan, J.H. 1994. Effects of UV-B radiation on photosynthesis and growth of terrestrial plants. *Photosynthesis Research* 39:463-473.

Teramura, A.H., Sullivan, J.H. and Lydon, J. 1990. Effects of UV-B radiation on soybean yield and seed quality: A six-year field study. *Physiologia Plantarum* 80:5-11.

Tevini, M. 1993. Effects of enhanced UV-B radiation on terrestrial plants. In UV-B Radiation and Ozone Depletion: Effects on Humans, Animals, Plants, Microorganisms, and Materials, Tevini, M. (ed.), Lewis Publishers, Boca Raton, Florida, pp. 125-153.

Tevini, M. 1994. UV-B effects on terrestrial plants and aquatic organisms. Progress in Botany 55:174-190.

Tevini, M. 1996. Erhöhte UV-B-Strahlung: Ein Risiko für Nutzpflanzen? *Biologie in unserer Zeit* 4:246-254.

Tevini, M. and Teramura, A.H. 1989. UV-B effects on terrestrial plants. *Photochemistry and Photobiology* 50:479-487.

Tevini, M., Mark, U. and Saile, M. 1990. Plant experiments in growth chambers illuminated with natural sunlight. In Environmental Research with Plants in Closed Chambers. Air Pollution Research Report 26, Payer, H.D., Pfirrman, T. and Mathy, P. (eds.), Commission of the European Communities, Belgium, pp. 240-251.

Thalmair, M., Bauw, G., Thiel, S., Döhring, T., Langebartels, C. and Sandermann, H. 1996. Ozone and ultraviolet-B effects on the defense-related proteins B-1,3-glucanase and chitinase in tobacco. *Journal of Plant Physiology* 148:222-228.

Torabinejad, J., Caldwell, M.M., Flint, S.D. and Durham, S. 1998. Susceptibility of pollen to UV-B radiation: an assay of 34 taxa. *American Journal of Botany* 85:360-369.

Yazawa, M., Shimizu, T. and Hirao, T. 1992. Feeding response of the silkworm, *Bombyx mori*, to UV irradiation of mulberry leaves. *Journal of Chemical Ecology* 18:561-569.

Ziska, L.H., Teramura, A.H. and Sullivan, J.H. 1992. Physiological sensitivity of plants along an elevational gradient to UV-B radiation. *American Journal of Botany* 79:863-871.

CHAPTER 4

EFFECTS ON AQUATIC ECOSYSTEMS

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Summary

Regarding the effects of UV-B radiation on aquatic ecosystems, recent scientific and public interest has focused on marine primary producers and on the aquatic web, which has resulted in a multitude of studies indicating mostly detrimental effects of UV-B radiation on aquatic organisms. The interest has expanded to include ecologically significant groups and major biomass producers using mesocosm studies, emphasizing species interactions. This chapter provides an assessment of the effects of UV-B radiation on dissolved organic matter, decomposers, primary and secondary producers, and briefly summarizies recent studies in freshwater and marine systems.

Dissolved organic carbon (DOC) and particulate organic carbon (POC) are degradation products of living organisms. These substances are of importance in the cycling of carbon in aquatic ecosystems. UV-B radiation has been found to break down high molecular weight substances and make them available to bacterial degradation. In addition, DOC is responsible for short-wavelength absorption in the water column. Especially in coastal areas and freshwater ecosystems, penetration of solar radiation is limited by high concentrations of dissolved and particulate matter. On the other hand, climate warming and acidification result in faster degradation of these substances and thus enhance the penetration of UV radiation into the water column.

Several research groups have investigated light penetration into the water column. Past studies on UV penetration into the water column were based on temporally and spatially scattered measurements. The process of spectral attenuation of radiant energy in natural waters is well understood and straight forward to model. Less known is the spatial and temporal variability of in-water optical properties influencing UV attenuation and there are few long-term observations. In Europe, this deficiency of measurements is being corrected by a project involving the development of a monitoring system (ELDONET) for solar radiation using three-channel dosimeters (UV-A, UV-B, PAR) that are being installed from Abisko (North Sweden, 68°N, 19°E) to Tenerife (Canary Islands, 27°N, 17°W). Some of the instruments have been installed in the water column (North Sea, Baltic Sea, Kattegat, East and Western Mediterranean, North Atlantic), establishing the first network of underwater dosimeters for continuous monitoring.

Bacteria play a vital role in mineralization of organic matter and provide a trophic link to higher organisms. New techniques have substantially changed our perception of the role of bacteria in aquatic ecosystems over the recent past and bacterioplankton productivity is far greater than previously thought, having high division and turnover rates. It has been shown that bacterioplankton plays a central role in the carbon flux in aquatic ecosystems by taking up dissolved organic carbon (DOC) and remineralizing the carbon. Bacterioplankton are more prone to UV-B stress than larger eukaryotic or-

ganisms and, based on one study, produce about double the amount of caeyclobutane dimers. Recently, the mechanism of nitrogen fixation by cyanobacteria has been shown to be affected by UV-B stress. Wetlands constitute important ecosystems both in the tropics and at temperate latitudes. In these areas, cyanobacteria form major constituents in microbial mats. The organisms optimize their position in the community by vertical migration in the mat which is controlled by both visible radiation and UV-B. Cyanobacteria are also important in tropical and subtropical rice paddy fields where they contribute significantly to the availability of nitrogen. Solar UV affects growth, development, and several physiological responses of these organisms.

On a global basis, phytoplankton is the most important biomass producer in aquatic ecosystems. The organisms populate the top layers of the oceans and freshwater habitats where they receive sufficient solar radiation for photosynthetic processes. New research strengthens previous evidence that solar UV affects growth and reproduction, photosynthetic energy harvesting enzymes, and other cellular proteins, as well as photosynthetic pigment contents. The uptake of ammonium and nitrate is affected by solar radiation in phytoplankton, as well as in macroalgae. Damage to phytoplankton at the molecular, cellular, population and community levels has been demonstrated. In contrast, at the ecosystem level there are few convincing data with respect to effects of ozone-related UV-B increases and considerable uncertainty remains. Following UV-B irradiation, shifts in phytoplankton community structure have been demonstrated, which may have consequences for the food web.

Macroalgae and seagrasses are important biomass producers in aquatic ecosystems (but considerably smaller than phytoplankton). In contrast to phytoplankton most of these organisms are sessile and can thus not avoid the exposure to solar radiation at their growth site. Recent investigations showed a pronounced sensitivity to solar UV-B, and effects have been found throughout the top 10-15 m of the water column. Photoinhibition can be quantified by oxygen exchange or by PAM (pulse amplitude modulated) fluorescence. Surface-adapted macroalgae, such as several brown and green algae, show a maximum of oxygen production at or close to the surface; whereas algae adapted to lower irradiances usually thrive best when exposed deeper in the water column. Mechanisms of protection and repair are being investigated.

UV effects on aquatic animals is of increased interest. Evidence for UV effects have been demonstrated in zooplankton activity. Other UV-B sensitive aquatic organisms include sea urchins, corals, and amphibians. Solar UV radiation has been known to affect corals directly. In addition, photosynthesis in their symbiotic algae is impaired, resulting in reduced organic carbon supply. Amphibian populations are in serious decline in many areas of the world, and scientists are seeking explanations for this phenomenon. Most amphibian population declines are probably due to habitat destruction or habitat alteration. Some declines are probably the result of natural population fluctuations. Other explanations for the population declines and reductions in range include disease, pollution, atmospheric changes and introduced competitors and predators. UV-B radiation is one agent that may act in conjunction with other stresses to adversely affect amphibian populations.

The succession of algal communities is controlled by a complex array of external conditions, stress factors and interspecies influences. Freshwater ecosystems have a high turnover and the success of an individual species is difficult to predict but the development of general patterns of community structure follows defined routes. There is a strong predictive relationship between DOC concentration and the depth to which UV radiation penetrates in lakes. Since DOC varies widely, freshwater systems display a wide range of sensitivity to UV penetration. In these systems, increased solar UV-B is an additional stress factor that may change species composition and biomass productivity.

The Arctic aquatic ecosystem is one of the most productive ecosystems on earth and is a source of fish and crustaceans for human consumption. Both endemic and migratory species breed and reproduce in this ocean in spring and early summer, at a time when recorded increases in UV-B radiation are maximal. Productivity in the Arctic ocean has been reported to be higher and more heterogeneous than in the Antarctic ocean. In the Bering Sea, the sea-edge communities contribute about 40–50% of the total productivity. Because of the shallow water and the prominent stratification of the water layer, the phytoplankton is more exposed and affected by solar UV-B radiation. In addition, many economically important fish (e.g., herring, pollock, cod and salmon) spawn in shallow waters where they are exposed to increased solar UV-B radiation. Many of the eggs and early larval stages are found at or near the surface. Consequently, reduced productivity of fish and other marine crops are possible but has not been demonstrated.

There is increased consensus, covering a wide range of aquatic ecosystems, that environmental UV-B, independent of ozone related increases, is an important ecological stress that influences the growth, survival and distribution of phytoplankton. Polar ecosystems, where ozone-related UV-B increases are the greatest and which are globally significant ecosystems, are of particular concern. However, these ecosystems are characterized by large spatial and temporal variability which makes it difficult to separate out UV-B specific effects on single species or whole phytoplankton communities. There is clear evidence for short-term effects. In one study a 4 to 23% photoinhibition of photosystem II activity was measured under the ozone hole. However, extrapolation of short-term effects to long-term ecological consequences requires accounting for various complex effects and quantitative evaluation remains uncertain.

Introduction

Solar short-wavelength radiation has been shown to reach ecologically significant depths in many freshwater and marine ecosystems (USEPA, 1987; Smith et al., 1992; Scully and Lean, 1994; Häder, 1995a; Booth et al., 1997; Coohill et al., 1996). Drastic stratospheric ozone depletion over both the Antarctic and Arctic, as well as moderate decreases in total ozone column over high and mid-latitude waters, have been reported. There is strong evidence that these trends increase the amount of solar UV-B which penetrates within the euphotic zone, where phytoplankton productivity takes place. In addition, there is evidence that it alters the ratio of UV-B:UV-A:PAR radiation which may impair the delicate light-dependent responses of aquatic organisms, including photosynthesis, photoorientation, photoinhibition and photoprotection (Smith et al., 1992; Häder et al., 1995; Gerber et al., 1996; Jiménez et al., 1996; Häder, 1997a,b). Changes in the spectral composition exceeding those experienced during the evolution of exposed organisms may pose significant stress for the diverse aquatic ecosystems (IASC, 1995). Both UV-B and UV-A affect growth and productivity by a number of mechanisms involving several molecular targets within the exposed cells. While most organisms possess effective protective and repair mechanisms, excessive exposure to solar UV radiation may overload their capabilities.

Significant changes of solar UV on aquatic ecosystems may result in decreased biomass productivity. The impact of this decrease would be reflected through all levels of the intricate food web, resulting in reduced food production for humans (Häder et al., 1995; Häder, 1997e; Häder and Worrest, 1997), reduced sink capacity for atmospheric carbon dioxide (Ducklow et al., 1995; Takahashi et al., 1995, 1997), as well as changes in species composition and ecosystem integrity. The role of oceanic carbon dioxide uptake in global warming is of high significance (Sarmiento and Le Quéré, 1996; Thomson, 1997). However, the potential impact of ozone depletion on atmospheric carbon dioxide,

mediated through inhibition of marine primary production, is uncertain and a more rigorous and detailed analysis is urgently needed. Research has been intensified over the last few decades to evaluate UV-B related damage of aquatic ecosystems (Nolan and Amanatidis, 1995). Important reviews on various aspects of UV effects on aquatic ecosystems include: aquatic ecosystes in general (Häder 1997c; Häder and Worrest, 1997); the role of MAA‘s in marine organisms (Dunlap and Shick, 1998); phytoplankton (Cullen and Neale, 1997a,b; Häder, 1997a); microalgae (Franklin and Foster, 1997; Häder and Figueroa, 1997); corals and coral bleaching (Shick et al., 1996; Lesser, 1996); lake acidification and UV penetration (Williamson, 1995, 1996).

Dissolved Organic Matter and Solar UV Radiation

Solar UV has been shown to degrade dissolved organic carbon (DOC) photolytically, most of the DOC being of terrestrial origin and relatively resistant to other forces (Naganuma et al., 1996). Humic substances are fairly resistant to bacterial degradation, but after photolytic activity the products (e.g., formaldehyde, acetaldehyde, glyoxylate, and pyruvate) are readily taken up by bacterioplankton (Wetzel et al., 1995). Humic substances strongly absorb ultraviolet radiation. Thus, increased breakdown of DOC and subsequent consumption by bacteria increases the UV-B penetration into the water column. Close to the surface, solar UV radiation inhibits bacterioplankton activity and rapidly photolyzes DOC. However, the uptake of the fragments is hampered by the inhibition of the bacterial ectoenzymes. Only when both bacteria and the photolyzed DOC circulate to deeper layers does the uptake rate increase. Another aspect of DOC photolysis is the generation of photosensitizers, which upon absorption of UV radiation produce reactive oxygen species or free radicals. DMS is released from the water at a rate that is closely correlated with the concentration of DOC (Herndl, 1997). DMSP is considered an osmoregulator in phytoplankton.

Measurements and Modeling

To determine the effects of solar radiation on marine ecosystems the penetration of UV and PAR into the water column needs to be measured (Montecino and Pizarro, 1995; Björn et al., 1996). Marine waters show large temporal and regional differences in their concentrations of dissolved and particulate absorbing substances. Jerlov (1968) classified marine waters into nine types of coastal and five types of open ocean waters depending on their transmission. The ratio between the 0.1% depths for UV-B and PAR can be used to calculate the detrimental effects on algae by solar UV-B radiation that hits the organisms in the euphotic zone (Piazena and Häder, 1997). Recent developments allow an accurate measurement of the underwater light field (Morrow and Booth, 1997). Yellow substances, chlorophyll *a* and other photosynthetic pigments, as well as organic and inorganic particulate material mainly cause the spectral attenuation of UV-B radiation in the water column. The modulation of open ocean underwater light field by phytoplankton has been extensively studied (Figueroa et al., 1994), but reports on the underwater light field in coastal habitats colonized by macroalgae are scarce, particularly in the UV-B range. Algal canopies modify the light quality by absorption and scattering of the incident light.

Recently, a network of dosimeters (ELDONET) was installed in Europe ranging from Abisko in Northern Sweden to Gran Canaria with a total of 26 instruments (Santas et al., 1997). Two of the instruments are located at high altitudes and six are located under water where they operate in conjunction with a terrestrial counterpart (Fig. 4.1). These instruments record solar radiation fully automatically in three channels (UV-B, UV-A, PAR). The data are transmitted to a server in Pisa and are

available to the public on the Internet in graphical and numerical form (http://power.ib.pi.cnr.it:80/eldonet/). Other networks have been installed, e.g., Biospherical Instruments Inc. (San Diego, California) is responsible for obtaining and distributing irradiance data from the U.S. National Science Foundation UV Spectroradiometer Monitoring Network (http://www.biospherical.com). Data is available from stations in San Diego (California), Ushuaia (Argentina), Barrow (Alaska) and three Antarctic stations (South Pole, Palmer and McMurdo). Great care is necessary to guarantee quality control of the light measurements (Seckmeyer et al., 1994).

Fig. 4.1 Locations of the terrestrial, aquatic and high altitude instruments in the ELDONET network of solar dosimeters. Circles terrestrial instruments, squares underwater instruments, triangles high altitude instruments.

Underwater UV irradiance has been measured at Northern latitudes (79°N, Spitsbergen, Norway). The measurements showed significant short term increases due to local ozone holes as that reported in winter 1994-1995 in the European SESAME campaign (Second European Stratospheric Arctic and

Midlatitude Experiment) in which a depletion of 20-30% of the stratospheric ozone was observed (Ott and Amanatides, 1994).

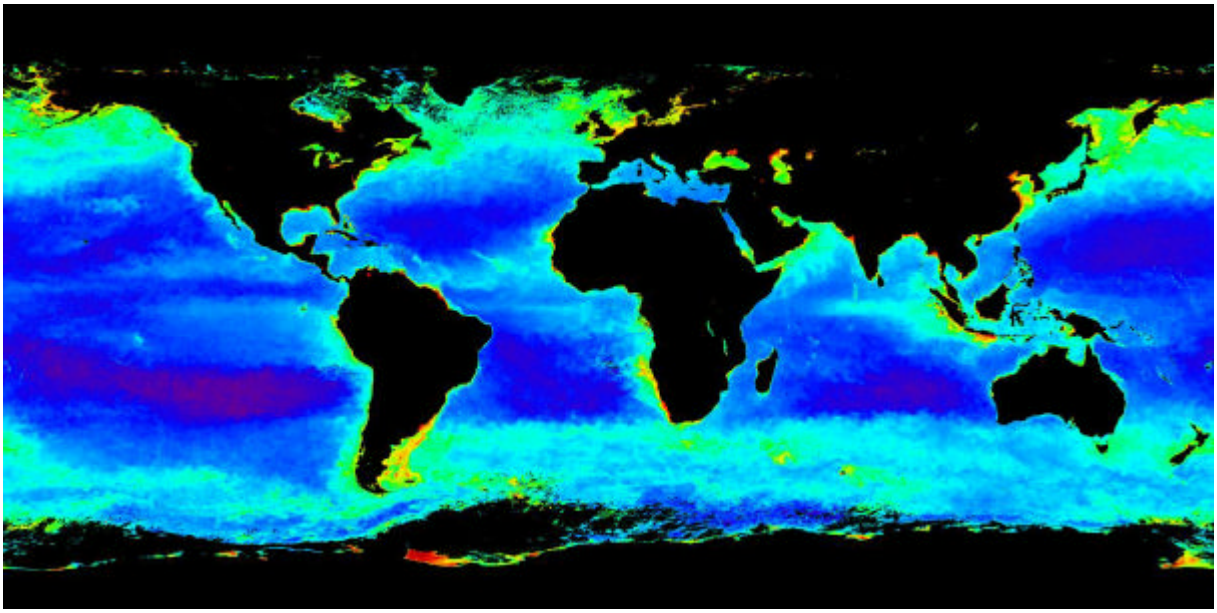


Fig. 4.2 Chlorophyll concentration in the ocean. Provided by the SeaWiFS Project, NASA/Goddard Space Flight Center

Another approach to quantify the underwater light climate is modeling (Zeng and Stamnes, 1994). Within limits the optical characteristics of the water column can be obtained from satellite data (e.g., CZCS and SeaWiFS, Fig. 4.2). These instruments cover only the visible range, but attempts have been and are being made to extrapolate the data into the UV range so as to make these remotely sensed data relevent to UV studies.

There have been great efforts to develop techniques for measuring algal biomass by using remote sensors. Most work has focused on quantifying chlorophyll from phytoplankton in surface oceanic waters (Brown et al., 1995). Piazena and Häder (1997) discussed the applicability of remote sensing to detect and quantify phytoplankton in the water. One major obstacle for remote monitoring is the fact that overflying instruments mainly determine the surface signal. Therefore, profound knowledge of the vertical distribution of phytoplankton, as well as the distribution of algal groups is necessary to derive a quantitative analysis of biomass productivity (Piazena and Häder, 1997). Mooring of optical instruments has been used to determine phytoplankton production in the oligotrophic waters of the Sargasso Sea (Waters and Smith, 1994). Meinesz et al. (1991) have studied macroalgal biomass and distribution on the bottom of clear waters in Polynesia.

Recently Behrenfeld and Falkowski (1997a) have evaluated models used to estimate photosynthetic rates derived from satellite-based chlorophyll concentration. In addition, they (Behrenfeld and Falkowski, 1997b) have evaluated various primary productivity models, provided a classification scheme for these productivity models and show that many of these, apparently different, models show fundamental synonymy. If equivalent parameterizations are used for satellite derived chlorophyll and the maximum chlorophyll-specific carbon fixation rate, then estimates of global annual primary production were found to be due primarily to these variables.

Bacterioplankton and Picoplankton

Use of modern epifluorescence microscopy techniques has substantially changed our perception of the role of bacteria in aquatic ecosystems over the recent past. Bacterioplankton productivity is far greater than previously thought, having high division and turnover rates (Fuhrman and Noble, 1995). The productivity is comparable to or exceeds phytoplankton primary productivity (Herndl, 1997) Bacterioplankton is no longer regarded solely as a final decomposer of organic material (Fig. 4.3). According to the "microbial loop hypothesis," bacterioplankton is seen in the center of a food web, having a similar function to phytoplankton and protists (Pomeroy and Wiebe, 1988). It has been shown that bacterioplankton plays a central role in the carbon flux in aquatic ecosystems by taking up dissolved organic carbon (DOC) and remineralizing the carbon.

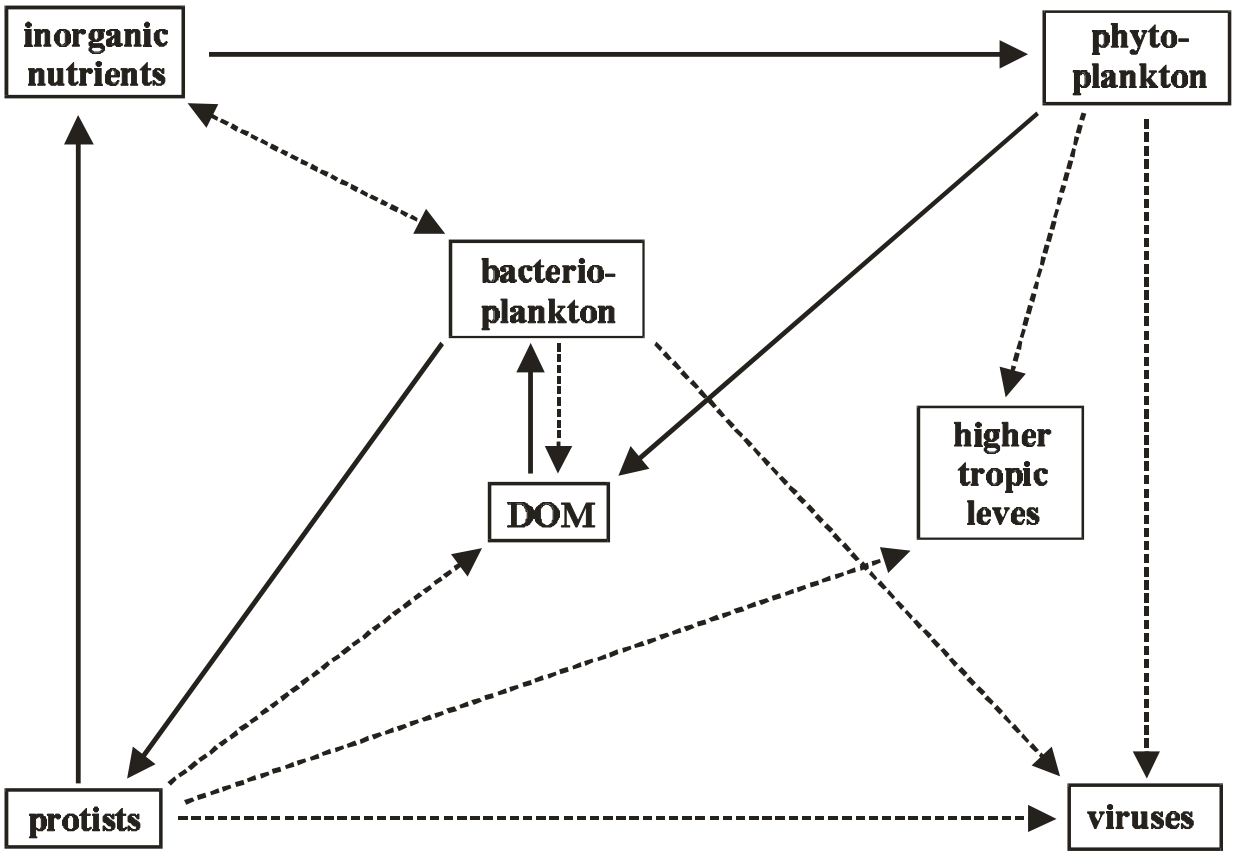


Fig. 4.3 Microbial loop (continuous arrows) in an aquatic habitat.

The effect of solar UV on bacterioplankton depends on the spectral attenuation coefficients in the water column and the time pattern of exposure and protection for the organisms as they are passively moved in the mixing layer. Bacterioplankton seems to lack UV screening pigments such as mycosporines or scytonemins, possibly because of their small size (Karentz et al., 1994; Garcia-Pichel, 1994). As a consequence, bacterioplankton are more prone to UV-B stress than larger eukaryotic organisms and exposure produces about double the amount of cyclobutane dimers as shown in a case study in the Gulf of Mexico (Jeffrey et al., 1996 a,b). This damage is at least partially offset by photoreactivation (Nicholson, 1995). The equilibrium between UV damage and photorepair is governed by the passive movement of the cells within the mixing layer where they are alternately exposed to high levels of damaging solar UV near the surface and beneficial UV-A/blue at greater depths. Other

macromolecular components of the bacterial cells, as well as ectoenzymes responsible for the cleavage of external organic matter, are affected by solar UV-B (Müller-Niklas et al., 1995). The bacterioplankton serves as food for heterotrophic flagellate picoplankton (<1 µm). The bacterial plankton population is limited by UV damage, viruses and heterotrophic flagellates (Aas et al., 1996; Sommaruga et al., 1995). This effect is partially offset by an effective repair mechanism using the photolyase enzyme. UV/blue radiation (360-430 nm) is most effective in the induction of the activity. It should be mentioned that also the viruses and nanoflagellates show a high sensitivity to solar UV radiation (Sommaruga et al., 1996).

Cyanobacteria

Cyanobacteria are a group of prokaryotes that possess a higher plant-type oxygenic photosynthesis. In addition to being a key player in aquatic productivity, several of these organisms is capable of fixing atmospheric nitrogen either as free-living organisms or in symbiosis with many other species including protists, animals and plants (Sinha and Häder, 1997). They use the enzyme nitrogenase to reduce atmospheric nitrogen into ammonium ions (NH_4^+), which they make available for aquatic eukaryotic phytoplankton as well as higher plants (Kashyap et al., 1991; Sinha et al., 1996; Sinha and Häder, 1996; Kumar et al., 1996a). The agricultural potential of cyanobacteria has been recognized as a biological fertilizer for wet soils such as in rice paddies (Banerjee and Häder, 1996). Cyanobacteria are cosmopolitan and must possess a high potential of adaptation to diverse environmental factors. However UV-B is known to affect processes such as growth, survival, pigmentation, motility, as well as the enzymes of nitrogen metabolism and CO_2 fixation (Donkor and Häder, 1996; Donkor and Häder, 1997). Depending on the species, growth and survival decrease within a few hours of UV-B irradiation. Cyanobacteria also make nitrogen in mid latitude agricultural systems, though not as massive as in paddy rice fields; therefore UV-B effects on these organisms could also be relevant on a global scale.

In addition to DNA, which is a highly susceptible cellular target, the photosynthetic pigments are affected. The phycobiliproteins, especially, are readily bleached and cleaved (Sinha et al., 1995, 1996; Aráoz and Häder, 1997). Bleaching of these accessory pigments is far more efficient than that of chlorophyll *a* or carotenoids (Sinha et al., 1995). At lower doses the energy transfer to the reaction center of photosystem II is impaired (Sinha et al., 1996). Simultaneously with destruction, an increased synthesis of phycobiliproteins has been observed under mild UV-B stress. The fact that these pigments strongly absorb in the UV-B range and that they form a peripheral layer around the sensitive central part containing the DNA might indicate that phycobilins are effective screening pigments, as well (Aráoz and Häder, 1997). They are capable of intercepting more than 99% of UV-B radiation before it penetrates to the genetic material.

UV-B induced inhibition of photosynthetic activity has been demonstrated in a number of marine and freshwater cyanobacteria. Sinha et al. (1996) reported that, in addition to the bleaching of the photosynthetic pigments, RuBisCO (ribulose-1,5-bis-phosphate carboxylase/oxygenase) activity was severely affected by UV-B treatment. Ammonium uptake was reduced by 10% in cultures exposed to solar radiation.

The nitrogen fixing enzyme nitrogenase is inhibited by UV-B even after a few minutes of in-vivo exposure. A complete loss of activity was found within 35-55 minutes depending upon the species (Kumar et al., 1996b). The inactivation may possibly be due to the inhibition of ATP synthesis by UV-B. In contrast to the effect on nitrogenase, a stimulation of nitrate reductase by UV-B was found

in all nitrogen fixing cyanobacterial strains studied so far (Sinha et al., 1995), while the ammonia-assimilating enzyme glutamine synthetase (GS) is inhibited.

Many cyanobacteria have developed a number of adaptive strategies to reduce the negative effects of excessive radiation, including the avoidance of brightly irradiated habitats, the synthesis of UV screening pigments, and the production of chemical scavengers that detoxify the highly reactive oxidants produced photochemically (Vincent and Roy, 1993). Screening pigments include scytonemin and mycosporine-like amino acids (MAAs), as well as a number of spectroscopically characterized but chemically unidentified, water-soluble pigments (e.g., a brown-colored pigment from *Scytonema hofmanii* and a pink extract from *Nostoc spongiaeforme*) (Kumar et al., 1996b; Donkor and Häder, 1995). Cyanobacteria such as *Scytonema* and *Nostoc* form filaments that are embedded in a mucilaginous sheath. The screening pigment from *Scytonema hofmannii* shows an absorption maximum at 314 nm and is released into the medium during the late stationary phase of growth. These organisms are more tolerant of UV-B irradiation than those that do not contain such covering (Sinha et al., 1995). For example, other species of *Scytonema* that do not produce this pigment are unable to survive 2 h of UV-B (2.5 Wm^{-2}). Karsten and Garcia-Pichel (1996) showed that screening pigments such as scytonemins, carotenoids and mycosporine-like amino acids are incorporated into the cytoplasm or the outer slime sheath, efficiently protecting the organisms from solar short-wavelength radiation.

Phytoplankton

Considerable recent work, covering a wide range of aquatic ecosystems, have contributed to an increased consensus that environmental UV-B, independent of ozone related increases, is an important ecological stress that influences the growth, survival and distributions of phytoplankton. On a global scale, phytoplankton is the most important biomass producer in aquatic ecosystems. The organisms populate the top layers of the oceans and freshwater habitats where they receive sufficient solar radiation (photosynthetic available radiation, PAR) for photosynthetic processes. This layer is called the euphotic zone, the base of which is defined as the depth where gross daily photosynthetic carbon fixation balances phytoplankton respiratory losses over a day (typically a depth to which 1% to 0.1% of PAR penetrates). Within this zone, phytoplankton is simultaneously exposed to solar UV radiation, in addition to longer-wavelength radiation. Information required to quantitatively estimate ozone-related UV-B damage to phytoplankton include: the spectral characteristics of solar radiation penetrating to depth and its space-time variability; a biological weighting function (BFW) of the biological effect; an exposure-response curve (ERC); a quantitative model of phytoplannkton inhibition by UV-B; and an assessment of how vertical mixing influences variable irradiance exposures of phytoplankton and their physiological response. We are far from a full quantitative understanding but advances in each of these areas have been made during the past few years and recent reviews include (Smith and Cullen, 1996; Hader, 1997c; Cullen and Neale, 1997; Vernet and Smith, 1997).

While it has long been known that DOC influences the penetration of UV-B, recent work has provided quantitative data which permits a more accurate estimated of DOC breakdown by UV-B, the resultant reduction of absorption in the UV region, and consequent increased penetration to depth. Among other things, this recent work permits a more accurate estimation of penetration of UV-B to depth based upon knowledge of in-water DOC concentrations. This may be especially important in assessing the potential influence of increased UV-B on freshwater ecosystems.

Biologically weighting functions, describing the spectrally weighted sensitivity of phytoplankton photosynthesis to UV and visible irradiance, have recently been determined by a number of

workers (Cullen and Neale, 1996; Boucher and Prezelin, 1996; Neale et al., 1998a). There is general agreement that the biological weighting, while highest in the UV-B, also contains a significant UV-A component. However, in spite of this broad agreement, biological weighting functions have been shown to vary by species, region, mixing characteristics of the water column and, perhaps, other environmental variability. As a consequence, it is now recognized that a single, or even a few, BWFs may be inadequate for a complete description of an ecosystem thus making quantitative analysis more complex.

Exposure-response curves (Cullen and Neale, 1996, 1997a,b) have recently been determined. To estimate the ERC one must determine if the measured damage is a function solely of cumulative exposure or whether it is a function of exposure rate. As noted by these authors, this difference is fundamental, and it has an important impact on both the design and interpretation of experiments and on the extrapolation of experimental results to real-world predictions. Further, the shape of the ERC influences model accuracy. In addition, the ERC which is important for accurate modeling, shows a range of experimental variability (Neale et al., 1998a), likely dependent on the balance between damage and repair and, thus, on the time-scale considered. Recent work has shown both forms of ERC in phytoplankton from different hydrographic environments thus making accurate modeling of ozone-related impacts more complex.

Models have been developed (Arrigo, 1994; Cullen and Neale, 1994; Boucher and Prezelin, 1996; Cullen and Neale, 1996a; Neale et al., 1998a) in an effort to estimate the impact of ozone depletion. These efforts represent important advances in our effort to quantify possible impacts, identifying the most significant processes and unknowns and evaluating uncertainities. However these recent advances continue to underscore the difficulty of using short-term observations to estimate longer-term (days to years) ecological response (Smith et al., 1992; Vincent and Roy, 1993; Bothwell et al., 1994; Cullen and Neale, 1994; Holm-Hanson, 1997; Neale et al. 1998a).

Solar UV affects growth and reproduction, photosynthetic energy harvesting enzymes (Vassiliev et al., 1994; Herrmann et al., 1995a, 1996, 1997; Giacometti et al., 1996; Figueroa et al., 1997; Gieskes and Buma, 1997), and other cellular proteins, as well as photosynthetic pigment contents (Gerber and Häder, 1995a,b; Buma et al., 1996a; Peletier et al., 1996; Häder, 1997a). The uptake of ammonium and nitrate is affected by solar radiation in phytoplankton (Behrenfeld, 1995; Döhler,1996, 1997; Döhler and Hagmeier, 1997), as well as in macroalgae (Döhler et al., 1995a). Phytoplankton responds with the production of heat-shock proteins, as well as changes in the cellular amino acid pools. One of the major targets is the DNA, which strongly absorbs in the short-wavelength range of solar radiation. Solar UV-B has been found to induce DNA damage and DNA synthesis delay in many organisms (Scheuerlein et al., 1995; Buma et al., 1995, 1996b, 1997). UV-B effects have also been studied on the ecosystem level using mesocosms (Santas et al., 1996; Wängberg and Selmer, 1997).

Macroalgae and Seagrasses

While phytoplankton are motile in the water column (Häder et al., 1995), most macroalgae are sessile and therefore restricted to their growth site (Lüning, 1990). Macroalgae show a distinct and fixed pattern of vertical distribution in their habitat. Some of these plants inhabit the supralittoral (coast above high water mark) exposed only to the spray from the surf, whereas others populate the eulittoral (intertidal zone), which is characterized by the regular temporal change in the tides (Häder, 1997d). Still others are never exposed to air since they are restricted to the sublittoral zone. The range in exposure can be substantial, from over 1000 Wm^{-2} (total solar radiation) at the surface to less than 0.01% of

that which reaches the understory of a kelp habitat (Markager and Sand-Jensen, 1994). Macroalgae have developed mechanisms to regulate their photosynthetic activity to adapt to the changing light regime and protect themselves from excessive radiation (Foster and Franklin, 1997). They use the same mechanism of photoinhibition as higher plants to decrease the photosynthetic electron transport during periods of excessive radiation. This phenomenon facilitates thermal dissipation of excessive excitation. Different algal species occupy different depth niches and are adapted to different solar exposure (Häder and Figueroa, 1997). They also differ in their ability to cope with enhanced UV radiation (Dring et al., 1996). A broad survey was carried out to understand photosynthesis in aquatic ecosystems and the different adaptation strategies to solar radiation of ecologically important species of green, red and brown algae from the North Sea, Baltic Sea, Mediterranean, Atlantic, polar and tropical oceans (Markager and Sand-Jensen, 1994; Wiencke et al., 1994; Figueroa et al., 1996; Beach and Smith, 1996a,b; Kirst and Wiencke, 1996; Häder and Figueroa, 1997; Porst et al., 1997).

Photoinhibition can be quantified by oxygen exchange (Häder and Schäfer, 1994) or by PAM (pulse amplitude modulated) fluorescence measurements developed by Schreiber et al. (1986) and based on transient changes of chlorophyll fluorescence. Surface-adapted macroalgae, such as several brown (*Cystoseira, Padina, Fucus*) and green algae (*Ulva, Enteromorpha*), show a maximum of oxygen production at or close to the surface (Herrmann et al., 1995b; Häder and Figueroa, 1997); whereas algae adapted to lower irradiances usually thrive best when exposed deeper in the water column (the green algae *Cladophora, Caulerpa,* most red algae) (Häder and Figueroa, 1997). It is interesting to note that respiration is inhibited to a far smaller degree than photosynthesis.

PAM fluorescence allows the determination of the photochemical and non-photochemical quenching (Büchel and Wilhelm, 1993). Recently, an underwater PAM instrument was developed for in situ measuring the quantum yield of fluorescence, which promises advances in the knowledge on ecophysiology of macroalgae. The increase in nonphotochemical quenching is related to the violaxanthin cycle, which is believed to quench excess excitation energy both in algae and in higher plants (Demmig-Adams and Adams, 1992; Häder and Figueroa, 1997). Even algae harvested from rock pools, where they are exposed to extreme irradiances, show signs of photoinhibition after extended periods of exposure (Fig. 4.4). Deep-water algae and those adapted to shaded conditions are inhibited even faster when exposed to direct solar radiation. Large differences were also found in the recovery between high light-adapted and protected species. A considerable proportion of photoinhibition is due to PAR (400-700 nm). Exclusion studies were carried out to determine the effects of solar UV-B and UV-A (Herrmann et al., 1995a). Increasing exposure to solar radiation resulted in a shift of the compensation point to higher irradiances. The compentation point defines the irradiance at which photosynthetic oxygen production and respiratory oxygen consumption balance each other. Exclusion of UV-B partially reduced the effects. This trend increased when about half or all of the UV-A radiation was excluded (Schott filters WG 360 and 395).

Chronic photoinhibition occurs when algae are exposed to excessive irradiance. The inhibition is characterized by photodamage of PS II reaction centers and subsequent proteolysis of the D1 protein (Critchley and Russell, 1994). In contrast, dynamic photoinhibition is readily reversible and follows a diurnal pattern with the lowest quantum yield around or soon after noon (Hanelt et al., 1994; Häder and Figueroa, 1997). The lowest light compensation point for photosynthesis has been reported in Arctic and Antarctic algae (Gómez et al., 1995; Wiencke, 1996; Gómez and Wiencke, 1996).

The long-term effects of solar UV on the primary productivity of macroalgae still need to be evaluated. Shallow water specimens in coral reefs undergo a 50% reduction in photosynthetic efficiency during the middle of the day and show a complete recovery by late afternoon. Both UV-A and

UV-B cause depression of the photosynthetic rate in the brown alga *Laminaria digitata* (Foster and Lüning, 1996).

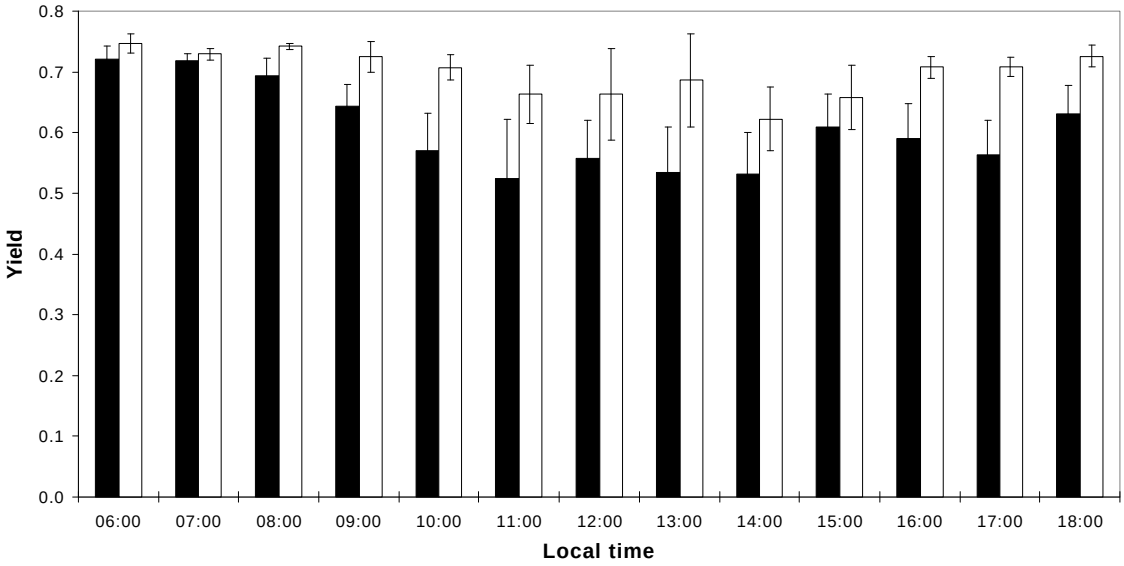


Fig. 4.4 Photosynthetic quantum yield measured on site using a PAM fluorimeter in the Mediterranean brown alga *Padina pavonica* harvested from 0 m (closed bars) and 6 m depth (open bars) at 1-h intervals (from Häder, 1997c).

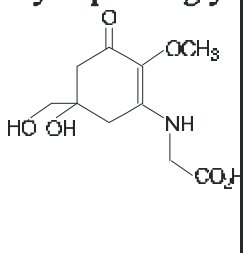
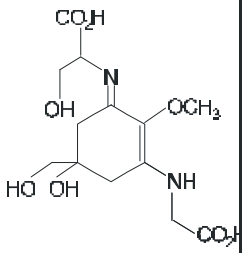
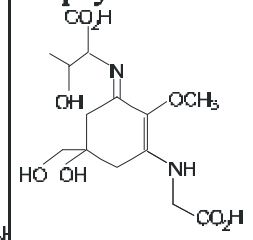
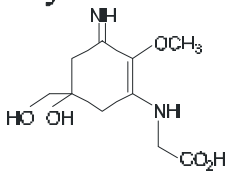
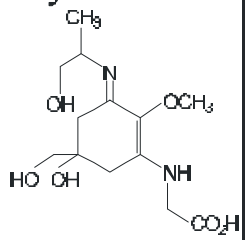
Recently, different methods for measuring light absorption in macroalgae have been compared (Mercado et al., 1996). The absorption determined by using an integrating sphere and by the opal-glass technique in a spectrophotometer in thin macroalgae was intercalibrated. García-Pichel (1995) has developed a scalar irradiance fiber-optic microprobe for the measurement of ultraviolet radiation at high spatial resolution.

The photoprotective mechanism of the xanthophyll cycle has been investigated mostly in microalgae (Schubert et al., 1994) and to less extent in macroalgae, e.g., the green alga *Ulva lactuca* (Grevby, 1996) and the brown algae *Dictyota dichotoma* (Uhrmacher et al., 1995) and *Lobophora variegata* (Franklin et al., 1996a). Red algae did not show the xanthophyll cycle.

Another mechanism for protection against UV radiation (UV-A and UV-B) is the production of screening pigments such as carotenoids or UV-absorbing mycosporine-like amino acids (MAAs, Tab. 4.1). MAAs have been found in green, red and brown algae from tropical, temperate and polar regions. Since these substances are chemically very stable they accumulate in the sediment of lakes and can be used of a permanent record for past ultraviolet radiation environments (Leavitt et al., 1997). In tropical algae, enhanced levels of carotenoids and UV-absorbing compounds were detected in tissues from the canopy compared to tissues from understory locations in turf-forming rhodophytes (Beach and Smith, 1996a,b). Current research indicates that solar UV-B is a stress factor for macroalgae and seagrasses even at current levels; therefore further increases in UV-B may reduce biomass production and changes in species composition in macroalgae ecosystems.

Zooplankton

Phytoplankton concentrations depend not only on nutrient availability, light, temperature and UV stress but also strongly on the grazing losses due to zooplankton activity (Banse, 1995). The zooplankton communities in turn not only depend on phytoplankton availability but also on grazing pressure as well as solar UV and temperature. Even at current levels, solar UV-B radiation can be a limiting factor, and small increases in UV-B exposure could result in significant reductions in the size of the consumer community (Damkaer, 1982; Kouwenberg et al., in press a). However, variability in cloud cover, water quality, and vertical distribution and displacement within the water column can all have an impact on the magnitude of the UV-B effect. Also, related to temperature effects, the macrozooplankton biomass in the California Current decreased by 80% since 1951 due to climatic warming by more than 1.5°C in some places (Roemmich and McGowan, 1995). As in phytoplankton, also in zooplankton UV-B induced DNA damage and photoenzymatic DNA repair have been demonstrated (Malloy et al., 1997). In planktonic embryos of copepods photoreactivation of UV induced damage was found to be an efficient repair mechanism (Naganuma et al., 1997). However, UV severely affects survival, fecundity and sex ratio in several intertidal copepods while others remained largely unaffected (Chalker-Scott, 1995).

Mycosporine-gly	Shinorine	Porphyra-334	Palythine	Palythinol
				
310 nm	334 nm	334 nm	320 nm	332 nm
5.32	2.79	5.22	4.48	8.85

Tab. 4.1 Structure, absorption maximum and retention time (HPLC) of some common mycosporines (Karentz et al., 1991)

Secondary Consumers

Other UV-B sensitive marine organisms include sea urchins and corals (Brown et al., 1994; Shick et al., 1996). However, many organisms seem to have adapted to solar UV by different strategies. For example, the planula larvae of the coral *Agaricia agaricites* show a pronounced variation in UV-B sensitivity along a depth gradient (Gleason and Wellington, 1995) and the green sea urchin *Strongylocentrotus droebachiensis* uses mycosporine-like amino acids for UV absorption that it derives from its diet. This latter adaptation was determined by feeding a MAA-rich red alga, *Mastocarpus stellatus*, and a MAA-deficient brown alga, *Laminaria saccharina,* to sea urchins (Carroll and Shick, 1996).

Although humans use about 8% of the primary productivity of the oceans, that fraction increases to more than 25% for upwelling areas and to 35% for temperate continental shelf systems (Vitousek et al., 1997). For about one-sixth of the world's population (primarily developing nations), the oceans provide more than one-third of their animal protein (FAO, 1995). Many of the fisheries that depend upon the oceanic primary productivity are unsustainable. Although the primary causes for a decline in fish populations are predation and poor food supply for larvae, overfishing of adults, water tempera-

ture, pollution and disease (Holmes, 1994; Rothchild, 1996), exposure to increased UV-B radiation may contribute to that decline. The eggs and larvae of many fish are sensitive to UV-B exposure (Hunter et al., 1982; Little and Fabacher, 1994; Kouwenberg et al., in press b). However, imprecisely defined habitat characteristics and the unknown effect of small increases in UV-B exposure on the naturally high mortality rates of fish larvae are major barriers to a more accurate assessment of ozone depletion on marine fish populations.Actual in-lake experiments have demonstrated that ambient UV levels in the surface waters of temperate lakes are adequate to induce 100% mortality of yellow perch eggs in low DOC lakes but not in lakes with higher DOC levels (Williamson et al., 1997).

Amphibian populations are in serious decline in many areas of the world (Wake, 1991), and scientists are seeking explanations for this phenomenon (Hays et al., 1996; Blaustein and Kiesecker, 1997). Worrest and Kimeldorf (1976) noted several adverse effects of increased exposure to UV-B radiation on the systemic development of boreal toad (*Bufo boreas boreas*) tadpoles in the laboratory. They questioned whether an increased exposure to UV-B radiation in nature could have an adverse impact on amphibian development. Vetter and colleagues (submitted), using a newly developed chemiluminescent immunoblot assay capable of measuring thymine-thymine pyrimidine dimers (TT dimers) in DNA, have investigated DNA damage and repair in pelagic fish eggs and larvae. Since the typical method of thymine dimer repair is photoenzymatic repair, the observed amount of DNA damage at any time of day is the net result of damage rates and repair rates. They find that over a day the typical diel pattern of DNA damage, at least for northern anchovy, resembles a dose-rate meter rather than a cumulative dose meter, i.e. DNA damage increases as the sun rises, reaches a peak level of damage near solar noon, and is followed by a period of rapid repair in the afternoon when UV-B is decreasing but the visible light utilized for repair is still abundant. An understanding of this diel cycle of damage and repair is essential for the correct interpretation of relationships between solar irradiance and levels of DNA damage in field samples.

As reported by several authors (Blaustein et al., 1997; Licht and Grant, 1997; Ovaska et al., 1997; Corn, 1998; Ankley et al., in press), field studies in which amphibian embryos were exposed to natural sunlight or to sunlight with UV-B radiation removed have shown conflicting results. Some studies resulted in increased embryonic mortality after UV-B exposure; whereas others show that current levels of UV-B radiation are not detrimental. Abiotic factors, such as water depth, water color, and dissolved organic content at the egg-laying sites, effectively reduce UV-B penetration through the water and reduce exposure to UV-B radiation at all life history stages. Biotic factors, such as jelly capsules around eggs, melanin pigmentation of eggs, and color of larvae and metamorphosed forms, further reduce the effectiveness of UV-B penetration.

Most amphibian population declines are probably due to habitat destruction or habitat alteration. Some declines are probably the result of natural population fluctuations. Other explanations for the population declines and reductions in range include disease, pollution, atmospheric changes and introduced competitors and predators. UV-B radiation is one agent that may act in conjunction with other stresses to adversely affect amphibian populations.

Ecosystems

Freshwater

The succession of periphytic and limnic algal communities is controlled by a complex array of external conditions, stress factors and interspecies influences (Rai et al., 1996). Freshwater ecosystems

have a high turnover and the success of an individual species is difficult to predict but the development of general patterns of community structure follows defined routes (Biggs, 1996). Even though transparency for solar UV-B is considerably lower than in oceanic waters, increased solar UV-B is an additional stress factor which may change species composition and biomass productivity (Williamson, 1995, 1996; Häder and Häder, 1997; Piazena and Häder, 1997). The interaction of UV-B and heavy-metal concentrations resulted in synergistic inhibition of nutrient uptake, enzyme activity, carbon fixation, ATP synthesis, and oxygen evolution in a number of phytoplankton species (Rai et al., 1996; Rai and Rai, 1997). Sixty-seven freshwater species of algae (Chlorophyta and Chromophyta) were screened in an experiment to determine their UV-B sensitivity (Xiong et al., 1996). The algae were selected to represent different ecosystems ranging from high-altitude lakes to thermal springs. The most sensitive species lost 30–50% of their oxygen-evolving capacity during a 2-h UV-B exposure (2 Wm^{-2}). Many UV-B resistant species were found in high mountain locations. They often have solid cell walls encrusted with sporopollenin. In another experiment the effects of solar UV-B on growth and species composition were studied in an exclusion experiment in a high-altitude mountain lake (Halac et al., 1997). In this study no significant differences were found between the control (full sunlight) and the UV-B depleted enclosure. However, it should be mentioned that UV-A also has been found to affect growth and photosynthesis (Kim and Watanabe, 1994). In other organisms UV-A had a beneficial effect, partially counteracting UV-B inhibition (Quesada, 1995). In addition to the primary producers, the significance of heterotrophic picoplankton in freshwater ecosystems needs to be taken into account (Sommaruga and Robarts, 1997).

The results of an experiment by Bothwell et al. (1994) reinforce the view that predictions of responses by ecosystems to elevated UV-B exposure should not be based solely on single-species assessments. As reported, greater algal growth occurred in an artificial stream under UV-B exposure than in the control, after some lag time. The explanation of this surprising (at that time) result was that the grazers, larval chironomids, were more sensitive to UV-B radiation than their food, the algae.

The Antarctic Aquatic Ecosystem.

Productivity in the Southern Ocean is characterized by large scale spatial and temporal variability (Sullivan et al., 1993; Arrigo, 1994: Smith et al., 1998). This makes it difficult to filter out UV-B specific effects from other variable environmental effects (Neale et al., 1998a), or to estimate the impact on single species or whole phytoplankton communities (Karentz and Spero, 1995; Davidson et al., 1996). Especially at high latitudes, variability in solar elevation, cloud cover, deep vertical mixing and the cover of ice and snow significantly confound field results of UV-B effects on phytoplankton and the consequent interpretation of these results. With increasingly complete observations, recent estimates of the effect of 50% ozone reduction on integral water column productivity are relatively consistent, <5% (Boucher and Prezelin, 1996) and 0.7-8.5% (depending on BWF, assumed mixing regime and cloudiness, Neale et al., 1998b), with earlier estimates (6%, Smith et al., 1992).

Observations by many workers, which vary greatly in both time and space, show convincing evidence of UV-B damage to phytoplankton, but in order to determine long-term effects acclimation and adaptation phenomena (Villafane et al., 1995; Lesser, 1996; Helbling et al., 1996) as well as other factors (Neale at al., 1998a) need to be assessed. Several models have been developed (Arrigo, 1994; Behrenfeld et al., 1994; Broucher and Prezelin, 1996; Neale at al., 1998a) to permit estimate of ecosystem productivity loss based on short-term observations. While it has long been known that vertical mixing is a major complication in attempting to quantify UV-B effects on phytoplankton, only recently have the interactive effects of ozone depletion and vertical mixing on photosynthesis of Antarctic phytoplankton been modeled (Neale et al., 1998a). Field results of these workers (Neale et al.,

1998b), in agreement with others (Smith et al., 1992; Helbling et al., 1994; Vernet et al., 1994), clearly demonstrate that photosynthesis of Antarctic phytoplankton is inhibited by ambient UV during incubation in fixed containers. The difficulty comes in the generalization of these experimental results to Antarctic waters where mixing significantly alters the exposure of phytoplankton to UV-B. To estimate this environmental influence, Neale and coworkers (Neale et al., 1998a) have developed a model of UV-influenced phytoplankton during vertical mixing. They find that near-surface UV strongly inhibits photosynthesis under all modeled conditions and that inhibition of photosynthesis can be enhanced or decreased by vertical mixing, dependent upon the depth of the mixed layer. Further, they show that an abrupt 50% reduction in stratospheric ozone could, as a worst case, lower daily integrated water column photosynthesis by as much a 8.5%. Note, that this modeling result is consistent with the results of Smith and coworkers who specifically targeted the marginal ice zone (MIZ), where meltwater provides stability and minimizes vertical mixing, for their studies. However, Neale and coworkers also note that inhibition associated with realistic environmental variability can have a stronger influence on integrated water column photosynthesis than UV-B effects: vertical mixing by about ±37%, measured variable sensitivity of phytoplankton to UV about ±46%, and cloudiness about ±15%. These workers conclude "that ozone depletion can inhibit primary productivity in open waters of the Antarctic, but that natural variability in exposures of phytoplankton to UV, associated with vertical mixing and cloud cover, has a major role in either enhancing or diminishing the impact on water column photosynthesis". They also note that "regardless of these natural interactions, UV is a significant environmental stressor, and its effects are enhanced by ozone depletion".

The Arctic Aquatic Ecosystem

Though being in a similar situation of increasing UV-B stress as the Antarctic aquatic ecosystems, the Arctic differs in many respects from its antipode (Weiler and Penhale, 1994; Wängberg et al., 1996). The Arctic ocean is a nearly closed water mass with limited water exchange with the Atlantic and Pacific oceans. It represents 25% of the global continental shelf and receives about 10% of the world river discharge. This considerable freshwater inflow causes pronounced stratification year round and is responsible for high concentrations of particulate and dissolved organic carbon (POC and DOC), which strongly affect the penetration of solar UV into the water column. The plumes of major rivers can be traced several hundred kilometers (Burenkov, 1993). Another difference between the Arctic and the Antarctic is the greater importance of macroalgae in the Arctic. The Arctic aquatic ecosystem is one of the most productive ecosystems on earth and is a source of fish and crustaceans for human consumption. Both endemic and migratory species breed and reproduce in this ocean in spring and early summer, at a time when recorded increases in UV-B radiation are maximal. Productivity in the Arctic ocean has been reported to be higher and more heterogeneous than in the Antarctic ocean (Springer and McRoy, 1993). In the Bering Sea, the sea edge communities contribute about 40–50% of the total productivity. Because of the shallow water and the prominent stratification of the water layer the phytoplankton may experience relatively high levels of solar UV-B. In addition, many economically important fish (e.g., herring, pollock, cod and salmon) spawn in shallow waters where they are exposed to this increased solar UV-B radiation when ozone is depleted. Many of the eggs and early larval stages are found at or near the surface. It is possible, given the general relationships between primary and fish production, that reduced productivity of fish and other marine crops would afffect no only humans in the region but also natural predators (orrers, seals, foxes, ice bears). However, further careful analysis is necessary to quantify UV-B related phytoplankton inhibitation and possible affects on the flow of energy to higher trophic levels. Currently we cannot accurately estimate if ozone-related impacts will, or will not, influence fish and other important marine crops.

The high concentrations of humic substances, which tend to be strong absorbers of UV-B radiation, may alter the underwater light penetration significantly (Wängberg et al., 1996). On the other hand, UV-B is known to photochemically attack humic substances altering the absorptive nature of the water column and leading to faster uptake by bacteria and heterotrophic nanoflagellates (Wängberg et al., 1996). The problem is more complicated and not well understood since UV-B has been found to be more detrimental for small phytoplankton organisms (Karentz et al., 1994) and even more so for the bacterioplankton (Herndl, 1997). In contrast, a recent study of size fractionated phytoplankton in a lake indicated that cells larger than 2 µm were twice as sensitive to solar UV-B than smaller cells (Milot-Roy and Vincent, 1994). The Arctic ocean is often nutrient limited, especially with respect to the inorganic nutrients such as nitrogen and phosphorus. The nitrogen cycle governs the primary productivity of the marine ecosystems. The same is true for the oligotrophic lakes and streams. Nitrogen and phosphorus uptake are UV-B sensitive (Döhler, 1992) which may augment the UV-B sensitivity of Arctic phytoplankton communities. Low doses of UV-B increase the uptake of phosphate, which is probably used for DNA repair, while it impairs the uptake at higher doses. All these effects have an impact on the biogeochemical cycles.

Conclusions and Consequences

Potential consequences of enhanced levels of exposure to UV-B radiation include loss of biomass, such as food sources for humans; changes in species composition (; decrease in availability of nitrogen compounds; and reduced uptake capacity for atmospheric carbon dioxide, resulting in the potential augmentation of global warming. Although there is significant evidence that increased UV-B exposure is harmful to aquatic organisms, damage to ecosystems is still uncertain. One of the most important concepts for assessing the impacts of enhanced levels of UV-B exposure on aquatic ecosystems is that complex rather than simple responses are likely to be the rule. Responses will not be limited to simple decreases in primary production. In fact, shifts in community structure may initially be more common and result in little detectable differences in ecosystem biomass.

References

Aas, P., Lyons, M., Pledger, R, Mitchell, D.L. and Jeffrey, W.H. 1996. Inhibition of bacterial activities by solar radiation in nearshore waters and the Gulf of Mexico. *Aquatic Microbial Ecology* 11:229-238.

Ankley, G.T., Tiegte, J.E., DeFoe, D.L., Jensen, K.M., Holcombe, G.W., Durhan, E.S. and Diamond, S.A. 1998. Effects of ultraviolet radiation and methoprene on survival and development of *Rana pipiens*. *Environmental Toxicology and Chemistry* 17: in press.

Aráoz, R. and Häder, D.-P. 1997. Ultraviolet radiation induces both degradation and synthesis of phycobilisomes in *Nostoc* sp.: a spectroscopic and biochemical approach. *FEMS Microbiology Ecology* 23:301-313.

Arrigo, K.R. 1994. Impact of ozone depletion on phytoplankton growth in the Southern Ocean: large-scale spatial and temporal variability. *Marine Ecology Progress Series* 114:1-12.

Banerjee, M. and Häder, D.-P. 1996. Effects of UV radiation on the rice field cyanobacterium, *Aulosira fertilissima*. *Environmental and Experimental Botany* 36:281-291.

Banse, K. 1995. Zooplankton: Pivotal role in the control of ocean production. *ICES Journal of marine Science* 52:265-277.

Beach, K.S. and Smith, C.M. 1996a. Ecophysiology of tropical rhodophytes. I. Microscale acclimation in pigmentation. *Journal of Phycology* 32:701-710.

Beach, K.S. and Smith, C.M. 1996b. Ecophysiology of tropical rhodophytes. II. Microscale acclimation in photosynthesis. *Journal of Phycology* 32:710-718.

Behrenfeld, M.J., Lee II, H. and Small, L.F. 1994. Interactions between nutritional status and long-term responses to ultraviolet-B radiation stress in a marine diatom. *Marine Biology* 118:523-530.

Behrenfeld, M.J. 1995. Ultraviolet-B radiation effects on inorganic nitrogen uptake by natural assemblages of oceanic plankton. *Journal of Phycology* 31:25-36.

Behrenfeld, M.J. and Falkowski, P.G. 1997a. Photosynthetic rates derived from satellite-based chlorophyll concentration. *Limnology and Oceanography* 42:1-20.

Behrenfeld, M.J. and Falkowski, P.G. 1997b. A consumer's guide to phytoplankton primary productivity models. *Limnology and Oceanography* 42:1479-1491.

Biggs, B.J.F. 1996. Patterns in benthic algae of streams. In *Algal Ecology*, Stevenson, R.J. (ed.), pp. 31-56.

Björn, L.O., Cunningham, A., Dubinsky, Z., Estrada, M., Figueroa, F.L., Garcia-Pichel, F. Häder, D.-P., Hanelt, D., Levavasseur, G. and Lüning, K. 1996. Technical discussion. I: Underwater light measurements and light absorption by algae. In *Underwater Light and Algal Photobiology*. *Scientia Marina* 60 (Suppl. 1). Figueroa, F.L., Jiménez, C., Pérez-Lloréns, J.L. and Niell, F.X. (eds.), Barcelona, Spain, pp. 59-63.

Blaustein, A.R. and Kiesecker, J.M. 1997 The effects of ultraviolet-B radiation on amphibians in natural ecosystems. In *The Effects of Ozone Depletion on Aquatic Ecosystems*, Häder, D.-P. (ed.), R.G. Landes Company, pp. 175-188.

Blaustein, A.R., Kiesecker, J.M., Chivers, D.P. and Anthony, R.G. 1997. Ambient UV-B radiation causes deformities in amphibian embryos. *Proceedings of the National Academy of Sciences (USA)* 94:13735-13737

Booth, C.R., Morrow, J.H., Coohill, T.P., Frederick, J.E., Häder, D.-P., Holm-Hansen, O., Jeffrey, W.H., Mitchell, D.L., Neale, P.J., Sobolev, I., van der Leun, J. and Worrest, R.C. 1997. Impacts of solar UVR on aquatic microorganisms. *Photochemistry and Photobiology* 65:252-269.

Bothwell, M.L., Sherbot, D.M.J. and Pollock, C.M. 1994. Ecosystem response to solar ultraviolet-B radiation: Influence of trophic level interactions. *Science* 256:97-100.

Boucher, N.P. and Prezelin, B.B. 1996. An in situ biological weighting function for UV inhibition of phytoplankton carbon fixation in the Southern Ocean. *Marine Ecology Progress Series* 114:223-236.

Brown, B.E., Dunne, R.P., Scoffin, T.P. and le Tissier, M.D.A. 1994. Solar Damage in intertidal corals. *Marine Ecology Progress Series* 105:219-230.

Brown, C.W., Esaias, W.E. and Thompson, A.M. 1995. Predicting phytoplankton composition from space – Using the ratio of euphotic depth to mixed-layer depth: An evaluation. *Remote Sensing Environment* 53:172-176.

Büchel, C. and Wilhelm, C. 1993. In vivo analysis of slow chlorophyll fluorescence induction kinetics in algae: progress, problems and perspectives. *Photochemistry and Photobiology* 58:137-148.

Buma, A.G.J., Engelen, A.H. and Gieskes, W.W.C. 1997. Wavelength-dependent induction of thymine dimers and growth rate reduction in the marine diatom *Cyclotella* sp. exposed to ultraviolet radiation. *Marine Ecology Progress Series* 153:91-97.

Buma, A.G.J., van Hannen, E.J., Roza, L., Veldhuis, M.J.W. and Gieskes, W.W.C. 1995. Monitoring ultraviolet-B-induced DNA damage in individual diatom cells by immunofluorescent thymine dimer detection. *Journal of Phycology* 51:314-321.

Buma, A.G.J., van Hannen, E.J., Veldhuis, M.J.W. and Gieskes, W.W.C. 1996b. UV-B induces DNA damage and DNA synthesis delay in the marine diatom *Cyclotella* sp. *Scientia Marina* 60 (Suppl. 1). Barcelona, Spain, pp. 101-106.

Buma, A.G.J., Zemmelink, H.J., Sjollema, K. and Gieskes, W.W.C. 1996a. UVB radiation modifies protein and photosynthetic pigment content, volume and ultrastructure of marine diatoms. *Marine Ecology Progress Series* 147:47-54.

Burenkov, V.I. 1993. Optical properties of the Laptev Sea near the Lena River delta. *Proc. Symp. on High Latitude Optics*. Tromso, Norway, 28 June – 2 July 1993.

Caroll, A.K. and Shick, J.M. 1996. Dietary accumulation of UV-Absorbing mycosporine-like amino acids (MAAs) by the green sea urchin *(Strongylocentrotus droebachiensis). Marine Biology* 124:561-569.

Chalker-Scott, L. 1995. Survival and sex ratios of the intertidal copepod, *Tigriopus californicus,* following ultraviolet-B (290-320 nm) radiation exposure. *Marine Biology* 123:799-804.

Coohill, T.P., Häder, D.-P. and Mitchell, D.L. 1996. Environmental ultraviolet photobiology: Introduction. *Photochemistry and Photobiology* 64:401-402.

Corn, P.S. 1998. Effects of ultraviolet radiation on boreal toads in Colorado. *Ecological Applications* 8:18-26.

Critchley, C. and Russell, A.W. 1994. Photoinhibition of photosynthesis *in vivo*: the role of protein turnover in photosystem II. *Physiologia Plantarum* 92:188-196.

Cullen, J.J. and Neale, P.J. 1994. Ultraviolet radiation, ozone depletion, and marine photosynthesis. *Photosynthesis Research* 39:303-320.

Cullen, J.J. and Neale, P.J. 1996. Effects of ultraviolet radiation on short-term photosynthesis of natural phytoplankton. *Photochemistry and Photobiology* - in press.

Cullen, J.J. and Neale, P.J. 1997a. Biological weighting functions for describing the effects of ultraviolet radiation on aquatic systems. In *The effects of Ozone Depletion on Aquatic Ecosystems. Ch. 6*, Häder, D-P. (ed.), Environmental Intelligence Unit, Academic Press and R.G. Landes Company, Austin, pp. 97-118.

Cullen, J.J. and Neale, P.J. 1997b. Effects of ultraviolet radiation on short-term photosynthesis of natural phytoplankton. *Photochemistry and Photobiology* 65:264-266.

Damkaer, D.M. 1982. Possible influence of solar UV radiation in the evolution of marine zooplankton. In *The Role of Solar Ultraviolet Radiation in Marine Ecosystems*, Calkins, J. (ed.), Plenum Press, New York, pp. 701-706.

Davidson, A.T., Marchant, H. J. and de la Mare, W. K. 1996. Natural UV exposure changes the species composition of Antarctic phytoplankton in mixed culture. *Aquatic Microbial Ecology* 10:299-305.

Demmig-Adams, B. and Adams III., W. 1992. Photoprotection and other responses of plants to high light stress. *Annual Reviews of Plant Physiology and Plant Molecular Biology* 43:599-626.

Döhler, G. 1996. Effect of UV irradiance on utilization of inorganic nitrogen by the Antarctic diatom *Odontella weissflogii* (Janisch) Grunow. *Botanica Acta* 109:35-42.

Döhler, G., 1992. Impact of UV-B radiation (290–320 nm) on uptake of ^{15}N-ammonia and ^{15}N-nitrate by phytoplankton of the Wadden Sea. *Marine Biology* 112:485-489.

Döhler, G., 1997. Impact of UV radiation of different wavebands on pigments and assimilation of ^{15}N-ammonium and ^{15}N-nitrate by natural phytoplankton and ice algae in Antarctica. *Journal of Plant Physiology* 151:550-555.

Döhler, G. and Hagmeier, E. 1997. UV-Effects on Pigments and Assimilation of ^{15}N-Ammonium and ^{15}N-Nitrate by Natural Marine Phytoplankton of the North Sea. *Botanica Acta* 110:481-488.

Döhler, G., Hagmeier, E. and David, C. 1995a. Effects of solar and artificial UV irradiation on pigments and assimilation of ^{15}N ammonium and ^{15}N nitrate by macroalgae. *Journal of Photochemistry and Photobiology B*: *Biology* 30:179-187.

Donkor, V.A. and Häder, D.-P. 1995. Protective strategies of several cyanobacteria against solar radiation. *Journal of Plant Physiology* 145:750-755.

Donkor, V.A. and Häder, D.-P. 1996. Effects of ultraviolet irradiation on photosynthetic pigments in some filamentous cyanobacteria. *Aquatic Microbial Ecology* 11:143-149.

Donkor, V.A. and Häder, D.-P. 1997. Ultraviolet radiation effects on pigmentation in the cyanobacterium *Phormidium uncinatum*. *Acta Protozoologica* 36:49-55.

Ducklow, H.W., Carlson, C.A., Bates, N.R., Knap, A.H. and Michaels, A.F. 1995. Dissolved organic carbon as a component of the biological pump in the North Atlantic Ocean. *Philosophical Transactions of the Royal Society of London B: Biological Sciences* 348:161-167.

Dunlap, W.C. and Shick, J.M. 1998. Ultraviolet radiation-absorbing mycosporine-like amino acids in coral reef organisms: a biochemical and environmental perspective. *Journal of Phycology* 34:418-430.

FAO 1995. *FAO Yearbook of Fishery Statistics – Catches and Landings,* http://www.fao.org/waicent/faoinfo/fishery/trends/catch/globalsf.htm

Figueroa, F.L., Jiménez, C., Lubián, L.M., Montero, O., Lebert, M. and Häder, D.-P. 1997. Effects of high irradiance and temperature on photosynthesis and photoinhibition in *Nannochloropsis gaditana* Lubián (Eustigmatophyceae). *Journal of Plant Physiology* 151:6-15.

Figueroa, F.L., Jiménez, C., Pérez-Lloréns, J.L. and Niell, F.X. 1996. *Underwater light and algal photobiology*. *Scientia Marina* 60 (Suppl. 1). Barcelona, Spain, pp. 343.

Foster, R. and Lüning, K. 1996. Photosynthetic response of *Laminaria digitata* to ultraviolet A and B radiation. In *Underwater light and algal photobiology*. *Scientia Marina* 60 (Suppl. 1), Figueroa, F.L., Jiménez, C. and Pérez-Lloréns, J.L. (eds.), Barcelona, Spain, pp. 65-71.

Franklin, L.A. and Foster, R.M. 1997. The changing irradiance environment: consequences for marine macrophyte physiology, productivity and ecology. *European Journal of Phycology* 32:207-232.

Fuhrman J.A. and Noble R.T. 1995. Viruses and protists cause similar bacterial mortality in coastal seawater. *Limnology and Oceanography* 40:1236-1242.

Garcia-Pichel, F. 1994. A model for the internal self-shading in planktonic organisms and its implications for the usefulness of ultraviolet sunscreens. *Limnology and Oceanography* 39:1704-1717.

Garcia-Pichel, F. 1995. A scalar irradiance fiber-optic microprobe for the measurement of ultraviolet radiation at high spatial resolution. *Photochemistry and Photobiology* 61:248-254.

Gerber, S. and Häder, D.-P. 1995a. Effects of enhanced solar irradiance on chlorophyll fluorescence and photosynthetic oxygen production of five species of phytoplankton. *FEMS Microbiology Ecology* 16:33-42.

Gerber, S. and Häder, D.-P. 1995b. Effects of artificial and simulated solar radiation on the flagellate *Euglena gracilis*: physiological, spectroscopical and biochemical investigations. *Acta Protozoologica* 34:13-20.

Gerber, S., Biggs, A. and Häder, D.-P. 1996. A polychromatic action spectrum for the inhibition of motility in the flagellate *Euglena gracilis*. *Acta Protozoologica* 35:161-165.

Giacometti, G.M., Barbato, R., Chiaramonte, S., Friso, G. and Rigoni, F. 1996. Effects of ultraviolet-B radiation on photosystem II of the cyanobacterium *Synechocystis* sp. PCC 6083. *European Journal of Biochemistry* 242:799-806.

Gieskes, W.W.C. and Buma, A.G.J. 1997. UV damage to plant life in a photobiologically dynamic environment: the case of marine phytoplankton. *Plant Ecology* 128:16-25.

Gleason, D.F. and Wellington, G.M. 1995. Variation in UVB sensitivity of planula larvae of the coral *Agaricia agaricites* along a depth gradient. *Marine Biology* 123:693-703.

Gómez, I. and Wiencke, C. 1996. Photosynthesis, dark respiration and pigment contents of gametophytes and sporophytes of the Antarctic brown alga *Desmarestia menziessi*. *Botanica Marina* 39:149-157.

Gómez, I., Wiencke, C. and Weykman, G. 1995. Seasonal photosynthetic characteristics of the brown alga *Ascoseira mirabilis* from King George Island (Antarctica). *Marine Biology* 123:167-172.

Grevby, C. 1996. Organisation of the light harvesting complex in fucoxanthin containing algae. PhD Thesis, Göteborg University, Sweden.

Häder, D.-P. 1995a. Photo-ecology and environmental photobiology. In *CRC Handbook of Organic Photochemistry and Photobiology*, Horspool, W.M. and Song, P.-S. (eds.), CRC Press, Boca Raton, New York, London, Tokyo, pp. 1392-1401.

Häder, D.-P. 1997a. Effects of UV radiation on phytoplankton. In *Advances in Microbial Ecology*, Jones, J.G. (ed.), Vol. 15. Plenum Press, New York, London, pp. 1-26.

Häder, D.-P. 1997b. Effects of solar UV-B radiation on aquatic ecosystems. In *Plants and UV-B: Responses to Environmental Change*, Lumsden, P.J. (ed.), Cambridge University Press, Cambridge, pp. 171-193.

Häder, D.-P. 1997c. UV-B and aquatic ecosystems. In *UV-B and Biosphere*, Rozema, J., Gieskes, W.W.C., van de Geijn, S.C., Nolan, C. and de Boois, H. (eds.), Kluwer Academic Publishers, Dordrecht, Boston and London, pp. 4–13.

Häder, D.-P. 1997d. Penetration and effects of solar UV-B on phytoplankton and macroalgae. *Plant Ecology* 128:4-13.

Häder, D.-P. 1997e. Stratospheric ozone depletion and increase in ultraviolet radiation. In *The Effects of Ozone Depletion on Aquatic Ecosystems. Ch. 1*, Häder, D.-P. (ed.), Environmental Intelligence Unit, Academic Press and R.G. Landes Company, Austin, pp. 1-4.

Häder, D.-P. and Figueroa, F.L. 1997. Photoecophysiology of marine macroalgae. *Photochemistry and Photobiology* 66:1-14.

Häder, D.-P. and Schäfer, J. 1994. *In-situ* measurement of photosynthetic oxygen production in the water column. *Environmental Monitoring and Assessment* 32:259-268.

Häder, D.-P. and Worrest, R.C. 1997. Consequences of the effects of increased solar ultraviolet radiation on aquatic ecosystems. In *The Effects of Ozone Depletion on Aquatic Ecosystems. Ch. 3*, Häder, D.-P. (ed.), Environmental Intelligence Unit, Academic Press and R.G. Landes Company, Austin, pp. 11-30.

Häder, D.-P., Worrest, R.C., Kumar, H.D. and Smith, R.C. 1995. Effects of increased solar ultraviolet radiation on aquatic ecosystems. *Ambio* 24:174-180.

Häder, M. and Häder, D.-P. 1997. Optical properties and phytoplankton composition in a freshwater ecosystem (Main-Donau-Canal). In *The Effects of Ozone Depletion on Aquatic Ecosystems. Ch. 9*, Häder, D.-P. (ed.), Environmental Intelligence Unit, Academic Press and R.G. Landes Company, Austin, pp. 155-174.

Halac, S., Felip, M., Camarero, L., Sommaruga-Wögrath, S., Psenner, R., Catalan, J. and Sommaragu, R. 1997. An *in situ* enclosure experiment to test the solar UVB impact on plankton in a high-altitude mountain lake. I. Lack of effect on phytoplankton species composition and growth. *Journal of Plankton Research* 19:1671-1686.

Hanelt, D., Jaramillo, M.J., Nultsch, W., Senger, S. and Westermeir, R. 1994. Photoinhibition as a regulatory mechanism of photosynthesis in marine algae of Antarctica. *Serie Cient. Inst. Antarct., Chil.* 44:67-77.

Hays, J.B., Blaustein, A.R., Kiesecker, J.M., Hoffman, P.D., Pandelova, I., Coyle, D. and Richardson, T. 1996. Developmental responses of amphibians to solar and artificial UVB sources: A comparative study. *Photochemistry and Photobiology* 64:449-456.

Helbling, E.B., Chalker, B.E., Dunlap, W.C., Holm-Hansen, O. and Villafane, V.E. 1996. Photoacclimation of antarctic marine diatoms to solar ultraviolet radiation. *Journal of Experimental Marine Biology and Ecology* 204:85-101.

Helbling, E.W., Villafane, V. and Holm-Hansen, O. 1994. Effects of ultraviolet radiation on Antarctic marine phytoplankton photosynthesis with particular attention to the influence of mixing. In *Ultraviolet Radiation in Antarctica: Mesurements and Biological Effects*, Weiler, C.S. and Penhale, P.A. (eds.), Antarctic Research Searies # 62, American Geophysical Union, Washington, DC, pp. 207-227.

Herndl, G.J. 1997. Role of ultraviolet radiation on bacterioplankton activity. In *The Effects of Ozone Depletion on Aquatic Ecosystems. Ch. 8*, Häder, D.-P. (ed.), Environmental Intelligence Unit, Academic Press and R.G. Landes Company, Austin, pp. 143-154.

Herrmann, H., Ghetti, F., Scheuerlein, R. and Häder, D.-P. 1995b. Photosynthetic oxygen and fluorescence measurements in *Ulva laetevirens* affected by solar irradiation. *Journal of Plant Physiology* 145:221-227.

Herrmann, H., Häder, D.-P. and Ghetti, F. 1997. Inhibition of photosynthesis by solar radiation in *Dunaliella salina*: relative efficiencies of UV-B, UV-A and PAR. *Plant, Cell and Environment* 20:359-365.

Herrmann, H., Häder, D.-P., Köfferlein, M., Seidlitz, H.K. and Ghetti, F. 1995a. Study on the effects of UV radiation on phytoplankton photosynthetic efficiency by means of a sunlight simulator. *Med. Biol. Environ*. 23:36-40.

Herrmann, H., Häder, D.-P., Köfferlein, M., Seidlitz, H.K. and Ghetti, F. 1996. Effects of UV radiation on photosynthesis of phytoplankton exposed to solar simulator light. *Journal of Photochemistry and Photobiology B: Biology* 34:21-28.

Holmes, R. 1994. Biologists sort the lessons of fisheries collapse. *Science* 264:1252-1253.

Holm-Hansen, O. 1997. Short- and long-term effects of UVA and UVB on marine phytoplankton productivity. *Photochemistry and Photobiology* 65:266-268.

Hunter, J.R., Kaupp, S.E. and Taylor, J.H. 1982. Assessment of effects of UV radiation on marine fish larvae. In *The Role of Solar Ultraviolet Radiation in Marine Ecosystems*, Calkins, J. (ed.), Plenum Press, New York, pp. 459-497.

IASC. 1995. Effects of increased ultraviolet radiation in the Arctic. IASC Report No. 2, IASC Secretariat. Journal of Plant Physiology 148:42-48.

Jeffrey, W.H., Aas, P., Maille Lyons, M., Coffin, R.B., Pledger, R.J. and Mitchell, D.L. 1996a. Ambient solar radiation-induced photodamage in marine bacterioplankton. *Photochemistry and Photobiology* 64:419-427.

Jeffrey, W.H., Pledger, R.J., Aas, P., Hager, S., Coffin, R.B., Haven, R.V. and Mitchell, D.L. 1996b. Diel and depth profiles of DNA photodamage in bacterioplankton exposed to ambient solar ultraviolet radiation. *Marine Ecology Progress Series* 137:283-291.

Jerlov, N.G. 1968. *Optical Oceanography*. Elsevier Publications Company, Amsterdam.

Jiménez, C., Figueroa, F.L., Aguilera, J., Lebert, M. and Häder, D.-P. 1996. Phototaxis and gravitaxis in *Dunaliella bardawil*: Influence of UV radiation. *Acta Protozoologica* 35:287-295.

Karentz, D. and Spero, H.J. 1995. Response of a natural *Phaeocystis* population to ambient fluctuations of UVB radiation caused by Antarctic ozone depletion. *Journal of Plankton Research* 17:1771-1789.

Karentz, D., Bothwell, M.L., Coffin, R.B., Hanson, A., Herndl, G.J., Kilham, S.S., Lesser, M.P., Lindell, M., Moeller, R.E., Morris, D.P., Neale, P.J., Sanders, R.W., Weiler, C.S. and Wetzel, R.G. 1994. Report of working group on bacteria and phytoplankton. In *Impact of UV-B Radiation on Pelagic Freshwater Ecosystems. Archiv für Hydrobiologie Beiheft* 43 (Special issue), Williamson, C.E. and Zagarese, H.E. (eds.), pp. 31-69.

Karsten, U. and Garcia-Pichel, F. 1996. Carotenoids and mycosporine-like amino acid compounds in members of the Genus *Microcoleus* (Cyanobacteria): A chemosystematic study. *Systematic and Applied Microbiology* 19:285-294.

Kashyap, A.K., Pandey, K.D. and Gupta, R.K. 1991. Nitrogenase Activity of the Antarctic cyanobacterium *Nostoc co*mmune: influence of temperature. *Folia Microbiologica* 36:557-560.

Kim, D.S. and Watanabe, Y., 1994. Inhibition of growth and photosynthesis of freshwater phytoplankton by ultraviolet A (UVA) radiation and subsequent recovery from stress. *Journal of Plankton Research* 16:1645-1654.

Kirst, G. O. and Wiencke, C. 1996. Ecophysiology of algae. *Journal of Phycology* 31:181-199.

Kouwenberg, J.H.M., Browman, H.I., Runge, J.A., Cullen, J.J., Davis, R.F. and St-Pierre, J.-F. 1998 a. Biological weighting of ultraviolet (280-400 nm) induced mortality in marine zooplankton and fish. II. *Calanus finmarchicus* G. (Copepoda) eggs. *Marine Biology*, in press.

Kouwenberg, J.H.M., Browman, H.I., Cullen, J.J., Davis, R.F., St-Pierre, J.-F. and Runge, J.A. 1998b. Biological weighting of ultraviolet (280-400 nm) induced mortality in marine zooplankton and fish. I. Atlantic cod (*Gadus morhua* L) eggs. *Marine Biology*, in press.

Kumar, A., Sinha, R.P. and Häder, D.-P. 1996b. Effect of UV-B on enzymes of nitrogen metabolism in the cyanobacteria *Nostoc calcicola*. *Journal of Plant Physiology* 148:86-91.

Kumar, A., Tyagi, M.B., Srinivas, G., Singh, N., Kumar, H.D., Sinha, R.P.and Häder, D.-P. 1996a. UVB shielding role of $FeCl_3$ and certain cyanobacterial pigments. *Photochemistry and Photobiology* 64:321-325.

Leavitt, P.R., Vinebrooke, R.D., Donald, D.B., Smol, J.P. and Schindler, D.W. 1997. Past ultraviolet radiation environments in lakes derived from fossil pigments. *Nature* 388:457-459.

Lesser, M. 1996. Acclimation of phytoplankton to UV-B radiation: oxidative stress and photoinhibition of photosynthesis are not prevented by UV-Absorbing compounds in the dinoflagellate *Prorocentrum micans*. *Marine Ecology Progress Series* 132:287-297 (correction: *Marine Ecology Progress Series* 141:312).

Licht, L.E.and Grant, K.P. 1997 The effects of ultraviolet radiation on the biology of amphibians. *American Zoologist* 37:137-145.

Little, E.E. and Fabacher, D.L. 1994. Comparative sensitivity of rainbow trout and two threatened salmonids, Appache Trout and Lahontan Cutthroat Trout, to UV-B radiation. *Archiv für Hydrobiologie Beiheft* 43:217-226.

Lüning, K. 1990. *Seaweeds. Their Environment, Biogeography and Ecophysiology*. Wiley, New York.

Malloy, K.D., Holman, M.A., Mitchell, D. and Detrich III, H.W. 1997. Solar UVB-induced DNA damage and photoenzymatic DNA repair in Antarctic zooplankton. *Proceedings of the National Academy of Sciences (USA)* 94:1258-1263.

Markager, S. and Sand-Jensen, K. 1994. The physiology and ecology of light-grown relationship in macroalgae. In *Progress in Phycological Research* Vol. 10, Round, F.E. and Chapman, D.J. (ed.), Biopress Ltd., Bristol, pp. 209-298.

Meinesz, A., Belscher, T., Bouderesque, C.-F. and Lefevre, J.-R. 1991. Premiére évaluation des potentialés du satellite Spot pour la cartographie des peuplements benthiques superficieles de Méditerranée occidentale. *Oceanologica Acta* 14:299-307.

Mercado, J. M., Jiménez, C., Niell, F.C.X. and Figueroa, F.L. 1996. Comparisons of methods for measuring light absorption by algae and their application to the estimation of the package effect. In *Underwater light and algal photobiology. Scientia Marina* 60 (Suppl. 1), Figueroa, F.L., Jiménez, C. and Pérez-Lloréns, J.L.(eds.), Barcelona, Spain, pp. 39-45.

Milot-Roy, V. and Vincent, W.F. 1994. UV radiation effects on photosynthesis: the importance of near-surface thermoclines in a subarctic lake. In *Advances in Limnology: Impact of UV-B radiation on Pelagic Ecosystems. Archiv für Hydrobiologie Beiheft* 43, Williamson, C.E. and Zagarese, H.E. (eds.), pp. 171-184.

Montecino, V. and Pizarro, G. 1995. Phytoplankton acclimation and spectral penetration of UV irradiance off the central Chilean coast. *Marine Ecology Progress Series* 121:261-269.

Morrow, J.H. and Booth, C.R. 1997. Instrumentation and methodology for ultraviolet readiation measurements in aquatic environments. In *The Effects of Ozone Depletion on Aquatic Ecosystems. Ch. 4*, Häder, D.-P. (ed.), Environmental Intelligence Unit, Academic Press and R.G. Landes Company, Austin, pp. 31-44.

Müller-Niklas, G., Heissenberger, A., Puskaric, S. and Herndl, G.J. 1995. Ultraviolet-B radiation and bacterial metabolism in coastal waters. *Aquatic Microbial Ecology* 9:111-116.

Naganuma, T., Inoue, T. and Uye, S. 1997. Photoreactivation of UV-induced damage to embryos of a planktonic copepod. *Journal of Plankton Research* 19:783-787.

Naganuma, T., Konishi, S., Inoue, T., Nakane, T. and Sukizaki, S. 1996. Photodegradation or photoalteration? Microbial assay of the effect of UV-B on dissolved organic matter. *Marine Ecology Progress Series* 135:309-310.

Neale, P.J., Cullen, J.J. and Davis, R.F. 1998a. Inhibition of marine photosynthesis by ultraviolet radiation: Variable sensitivity of phytoplankton in the Weddell-Scotia Sea during austral spring. *Limnology and Oceanography* 43:433-488.

Neale, P.J., Davis, R.F. and Cullen, J.J. 1998b. Interactive effects of ozone depletion and vertical mixing on photosynthesis of Antarctic phytoplankton. *Nature* 392:585-589.

Nicholson, W.L. 1995. Photoreactivation in the genus *Bacillus. Current Microbiology* 31:361-365.

Nolan, C.V. and Amanatidis, G.T. 1995. European Commission research on the fluxes and effects of environmental UVB radiation. *Journal of Photochemistry and Photobiology B: Biology* 31:3-7.

Ott, H. and Amanatides, G.T. 1994. *Sesame 1994-1995: a European contribution to the stratospheric ozone issue*. DG XII, EC, Brussels, pp. 16.

Ovaska, K., Davis, T.M. and Flamarique, I.N. 1997. Hatching success and larval survival of the frogs *Hyla regilla* and *Rana aurora* under ambient and artificially enhanced solar ultraviolet radiation. *Canadian Journal of Zoology* 75:1081-1088.

Peletier, H., Gieskes, W.W.C. and Buma, A.G.J. 1996. Ultraviolet-B radiation resistance of benthic diatoms isolated from tidal flats in the Dutch Wadden Sea. *Marine Ecology Progress Series* 135:163-168.

Piazena, H. and Häder, D.-P. 1997. Penetration of solar UV and PAR into different waters of the Baltic Sea and remote sensing of phytoplankton. In *The Effects of Ozone Depletion on Aquatic Ecosystems. Ch. 5*, Häder, D.-P. (ed.), Environmental Intelligence Unit, Academic Press and R.G. Landes Company, Austin, pp. 45-96.

Pomeroy, L.R. and Wiebe, W.J. 1988. Energetics of microbial food webs. *Hydrobiologia* 159:7-18.

Porst, M., Herrmann, H., Schäfer, J., Santas, R. and Häder, D.-P. 1997. Photoinhibition in the Mediterranean green alga *Acetabularia mediterranea* measured in the field under solar irradiation. *Journal of Plant Physiology* 151:25-32.

Quesada, A. 1995. Growth of Antarctica cyanobacteria under ultraviolet radiation: UVA counteracts UVB inhibition. *Journal of Phycology* 31:242-248.

Rai, L.C., Tyagi, B. and Mallick, N. 1996. Alternation in photosynthetic characteristics of *Anabaena dolium* following exposure to UV-B and Pb. *Photochemistry and Photobiology* 64:658-663.

Rai, P.K. and Rai L.C. 1997. Interactive effects of UV-B and Cu on photosynthesis, uptake and metabolism of nutrients in green alga *Chlorella vulgaris* and simulated ozone column. *Journal of General and Applied Microbiology* 43:(in press).

Roemmich, D. and McGowan, J. 1995. Climatic warming and the decline of zooplankton in the California Current. *Science* 267:1324-1326.

Rothchild, B.J. 1996. How bountiful are ocean fisheries? *Consequences* 2:26-36.

Santas, R., Häder, D.-P. and Lianou, C. 1996. Effects of solar UV radiation on diatom assemblages of the Mediterranean. *Photochemistry and Photobiology* 64:435-439.

Santas, R., Koussoulaki, A. and Häder, D.-P. 1997. In assessing biological UV-B effects, natural fluctuations of solar radiation should be taken into account. *Plant Ecology* 128:93-97.

Sarmiento, J.L. and Le Quéré, C. 1996. Oceanic carbon dioxide uptake in a model of century-scale global warming. *Science* 274:1346-1350.

Scheuerlein, R., Treml, S., Thar, B., Tirlapur, U.K. and Häder, D.-P. 1995. Evidence for UV-B-induced DNA degradation in *Euglena gracilis* mediated by activation of metal-dependent nucleases. *Journal of Photochemistry and Photobiology B*: *Biology* 31:113-123.

Schreiber, U., Schliwa, U. and Bilger, W. 1986. Continuous recording of photochemical and non-photochemical chlorophyll fluorescence quenching with a new type of modulation fluorometer. *Photosynthesis Research* 10:51-62.

Schubert, H., Kroon, B.M.A. and Matthijs, H.C.P. 1994. In vivo manipulation of the xanthophyll cycle and the role of the zeaxanthin cycle in the photoprotection against photodamage in the green alga *Chlorella pyrenoidosa*. *Journal of Biological Chemistry* 269:7267-7272.

Scully, N.M. and Lean, D.R.S. 1994. The attenuation of ultraviolet radiation in temperate lakes. In *Advances in Limnology: Impact of UV-B radiation on pelagic ecosystems. Archiv für Hydrobiologie Beiheft* 43, Williamson, C.E. and Zagarese (eds.), pp. 135-144.

Seckmeyer, G., Thiel, S., Blumthaler, M., Fabian, P., Gerber, S., Gugg-Helminger, A., Häder, D.-P., Huber, M., Kettner, C., Köhler, U., Köpke, P., Maier, H., Schäfer, J., Suppan, P., Tamm, E. and Thomalla, E. 1994. Intercomparison of spectral-UV-radiation measurement systems. *Applied Optics* 33:7805-7812.

Shick, J.M., Lesser, M.P. and Jokiel, P.L. 1996. Effects of ultraviolet radiation on corals and other coral reef organisms. *Global Change Biology* 2:527-545.

Sinha, R.P. and Häder, D.-P. 1996. Photobiology and ecophysiology of rice field cyanobacteria. *Photochemistry and Photobiology* 64:887-896.

Sinha, R.P. and Häder, D.-P. 1997. Impacts of UV-B irradiation on rice-field cyanobacteria. In *The Effects of Ozone Depletion on Aquatic Ecosystems. Ch. 9*, Häder, D.-P. (ed.), Environmental Intelligence Unit, Academic Press and R.G. Landes Company, Austin, pp. 189-198.

Sinha, R.P., Kumar, H.D., Kumar, A. and Häder, D.-P. 1995. Effects of UV-B irradiation on growth, survival, pigmentation and nitrogen metabolism enzymes in cyanobacteria. *Acta Protozoologica* 34:187-192.

Sinha, R.P., Singh, N., Kumar, A., Kumar, H.D., Häder, M. and Häder, D.-P. 1996. Effects of UV irradiation on certain physiological and biochemical processes in cyanobacteria. *Journal of Photochemistry and Photobiology, B: Biology* 32:107-113.

Smith, R.C., Baker, K.S., Byers, M.L. and Stammerjohn, S.E. 1998. Primary productivity of the Palmer Long-Term Ecological Research Area and the Southern Ocean. *Journal of Marine Systems* 529:in press.

Smith, R.C., Prézelin, B.B., Baker, K.S., Bidigare, R.R., Boucher, N.P., Coley, T., Karentz, D., MacIntyre, S., Matlick, H.A., Menzies, D., Ondrusek, M., Wan, Z. and Waters, K.J. 1992. Ozone depletion: Ultraviolet radiation and phytoplankton biology in Antarctic waters. *Science* 255:952-959.

Sommaruga, R. and Robarts, R.D. 1997. The significance of autotrophic and heterotrophic picoplankton in hypertrophic ecosystems. *FEMS Microbiology Ecology* 24:187-200.

Sommaruga, R., Krössbacher, M., Salvenmoser, W., Catalan, J. and Psenner, R. 1995. Presence of large virus-like particles in a eutrophic reservoir. *Aquatic Microbial Ecology* 9:305-308.

Sommaruga, R., Oberleiter, A. and Psenner, R. 1996. Effect of UV radiation on the bacterivory of a heterotrophic nanoflagellate. *Applied and Environmental Microbiology* 62:4395-4400.

Springer, A.M. and McRoy, C.P. 1993. The paradox of pelagic food webs in the northern Bering Sea. III. Patterns of primary production. *Continental Shelf Research* 13:575-599.

Sullivan, C.W., Arrigo, K.R., McClain, C.R., Comiso, J.C. and Firestone, J. 1993. Distributions of phytoplankton blooms in the Southern Ocean. *Science* 262:1832-1837.

Takahashi, T., Feely, R.A., Weiss, R.F., Wanninkhof, R.H., Chipman, D.W., Sutherland, S.C. and Takahashi, T. 1997. Global air-sea flux of CO_2: An estimate based on measurements of sea-air pCO_2 difference. *Proceedings of the National Academy of Sciences (USA)* 94:8282-8299.

Takahashi, T., Takahashi, T.T. and Sutherland S. 1995. An assessment of the role of the North Atlantic as a CO_2 sink. *Philosophical Transactions of the Royal Society of London B: Biological Sciences* 348:143-152.

Thomson, D.J. 1997. Dependence of global temperatures on atmospheric CO_2 and solar irradiance. *Proceedings of the National Academy of Sciences (USA)* 94:8370-8377.

Uhrmacher, S., Hanelt, D. and Nultsch, W. 1995. Zeaxanthin content and the degree of photoinhibition are linearly correlated in the brown alga *Dictyota dichotoma*. *Marine Biology* 123:159-165.

USEPA (U. S. Environmental Protection Agency) 1987. An assessment of the effects of ultraviolet-B radiation on aquatic organisms. In *Assessing the Risks of Trace Gases That Can Modify the Stratosphere*. EPA 400/1-87/001C, pp. 1-33.

Vassiliev, I.R., Prasil, O., Wyman, K.D., Kolber, Z.K., Hanson, A. K., Prentice, J.E. and Falkowski, P.G. 1994. Inhibition of PS II photochemistry by PAR and UV radiation in natural phytoplankton communities. *Photosynthesis Research* 42:51-64.

Vernet, M. and Smith, R.C. 1997. Effects of ultraviolet radiation on the pelagic Antarctic ecosystem. In *The effects of Ozone Depletion on Aquatic Ecosystems. Ch. 16*, Häder, D-P. (ed.), Environmental Intelligence Unit, Academic Press and R.G. Landes Company, Austin, pp. 247-265.

Vernet, M., Brody, E.A., Holm-Hansen, O. and Mitchell, B.G. 1994. The response of Antarctic phytoplankton to ultraviolet radiation: absorption, photosynthesis, and taxonomic composition. In *Ultraviolet Radiation in Antarctica: Mesurements and Biological Effects*, Weiler, C.S. and Penhale, P.A. (eds.), Antarctic Research Searies # 62, American Geophysical Union, Washington, DC, pp. 207-227.

Villafane, V.E., Helbling, W.W., Holm-Hansen, O. and Chalker, B.E. 1995. Acclimatization of Antarctic natural phytoplankton assemblages when exposed to solar ultraviolet radiation. *Journal of Plankton Research* 17:2295-2306.

Vincent, W.F. and Roy, S. 1993. Solar ultraviolet radiation and aquatic primary production: damage, protection and recovery. *Environmental Reviews* 1:1-12.

Vitousek, P.M., Mooney, H.A., Lubchenco, J. and Melillo, J.M. 1997. Human domination of Earth's ecosystems. *Science* 277:494-499.

Wake, D.B. 1991. Declining amphibian populations. *Science* 253:860.

Wängberg, S.-Å, and Selmer, J.-S. 1997. Studies of effects of UV-B radiation on aquatic model ecosystems. In *The Effects of Ozone Depletion on Aquatic Ecosystems. Ch. 12*, Häder, D.-P. (ed.), Environmental Intelligence Unit, Academic Press and R.G. Landes Company, Austin, pp. 199-214.

Wängberg, S.-Å, Selmer, J.-S., Ekelund, N.G.A. and Gustavson, K. 1996. UV-B effects on Nordic marine ecosystems. *TemaNord, Nordic Council of Ministers, Denmark* 1996:515.

Waters, K.J. and Smith, R.C. 1994. Phytoplankton production in the Sargasso Sea as determined using optical mooring data. *Journal of Geophysical Research* 99:18,385-18,402

Weiler, C.S. and Penhale, P.A. 1994. *Ultraviolet Radiation in Antarctica: Measurements and Biological Effects*. American Geophysical Union, Washington, DC.

Wetzel, R.G., Hatcher, P.G. and Bianchi, T.S. 1995. Natural photolysis by ultraviolet irradiance of recalcitrant dissolved organic matter to simple substrates for rapid bacterial metabolism. *Limnology and Oceanography* 40:1369-1380.

Wiencke, C. 1996. Recent advances in the investigation of Antarctic macroalgae. *Polar Biology* 16:231-240.

Wiencke, C., Bartsch, I., Bischoff, B., Peters, A.F. and Breeman, A.M. 1994. Temperature requirements and biogeography of Antarctic, Arctic and amphiequatorial seaweeds. *Botanica Marina* 37:247-259.

Williamson, C.E. Metzgar, S.L., Lovera, P.A. and Moeller, R.E. 1997. Solar ultraviolet radiation and the spawning habitat of yellow perch, *Perca flavescens. Ecological Applications* 7:1017-1023.

Williamson, C.E. 1995. What role does UV-B radiation play in freshwater ecosystems? *Limnology and Oceanography* 40:386-392.

Williamson, C.E. 1996. Effects of UV radiation on freshwater ecosystems. *International Journal of Environmental Studies* 51:245-256.

Worrest, R.C. and Kimeldorf, D.J. 1976. Distortions in amphibian development induced by ultraviolet-B enhancement (290-315 nm) of a simulated solar spectrum. *Photochemistry and Photobiology* 24:377-382.

Xiong, F., Lederer, F., Lukavsky, J. and Nedbal, L. 1996. Screening of freshwater algae (*Chlorophyta, Chromophyta*) for ultraviolet-B sensitivity of the photosynthetic apparatus. *Journal of Plant Physiology* 148:42-48.

Zeng, J. and Stamnes, K. 1994. Impact of stratospheric ozone depletion on UV penetration into the ocean. 1994 Ocean Sciences Meeting, February 21-25, San Diego, California.

CHAPTER 5

EFFECTS OF ENHANCED SOLAR ULTRAVIOLET RADIATION ON BIOGEOCHEMICAL CYCLES

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Summary

Effects of increased UV-B on emissions of carbon dioxide and carbon monoxide (CO) and on mineral nutrient cycling in the terrestrial biosphere have been confirmed by recent studies of a range of species and ecosystems. The effects, both in magnitude and direction, of UV-B on trace gas emissions and mineral nutrient cycling are species-specific and operate on a number of processes. These processes include changes in the chemical composition in living plant tissue, photodegradation (breakdown by light) of dead plant matter, including litter, release of carbon monoxide from vegetation previously charred by fire, changes in the communities of microbial decomposers and effects on nitrogen-fixing microorganisms and plants. Long-term experiments are in place to examine UV-B effects on carbon capture and storage in biomass within natural terrestrial ecosystems. Studies in natural aquatic ecosystems have indicated that organic matter is the primary regulator of UV-B penetration. Changes in the organic matter, caused by enhanced UV-B reinforced by changes in climate and acidification, result in clarification of the water and changes in light quality that have broad impacts on the effects of enhanced UV-B on aquatic biogeochemical cycles. Increased UV-B has positive and negative impacts on microbial activity in aquatic ecosystems that can affect carbon and mineral nutrient cycling as well as the uptake and release of greenhouse and chemically-reactive gases. Photoinhibition of surface aquatic microorganisms by UV-B can be partially offset by photodegradation of dissolved organic matter to produce substrates, such as organic acids and ammonium, that stimulate microbial activity. Modeling and experimental approaches are being developed to predict and measure the interactions and feedbacks between climate change and UV-B induced changes in marine and terrestrial biogeochemical cycles. These interactions include alterations in the oxidative environment in the upper ocean and in the marine boundary layer and oceanic production and release of CO, volatile organic compounds (VOC), and reactive oxygen species (such as hydrogen peroxide and hydroxyl radicals). Climate related changes in temperature and water supply in terrestrial ecosystems interact with UV-B radiation through biogeochemical processes operating on a wide range of time scales.

Introduction

This chapter reviews recent scientific information concerning the impacts of increasing UV-B exposure on biogeochemical cycles. This is a broad area of research that deals with the complex and often long term interaction of biological, chemical, and physical processes that control the exchange and recycling of matter and energy at and near the Earth's surface.Various controlled laboratory and field studies over the past four years have provided further evidence that enhanced UV-B radiation can af-

fect biogeochemical cycles. However, there have been no appropriate in situ studies addressing the extent to which biogeochemical cycles have already been affected by ozone depletion. This assessment therefore emphasizes potential effects should ozone continue to be depleted. Our focus is on the effects of enhanced UV on carbon and mineral nutrient cycling in terrestrial and aquatic ecosystems (Figure 1) and on the exchange of radiatively- and chemically-important gases between the biosphere and the atmosphere. More detailed treatments of UV-B effects on terrestrial and aquatic ecosystems appear in Chapters 3 and 4.

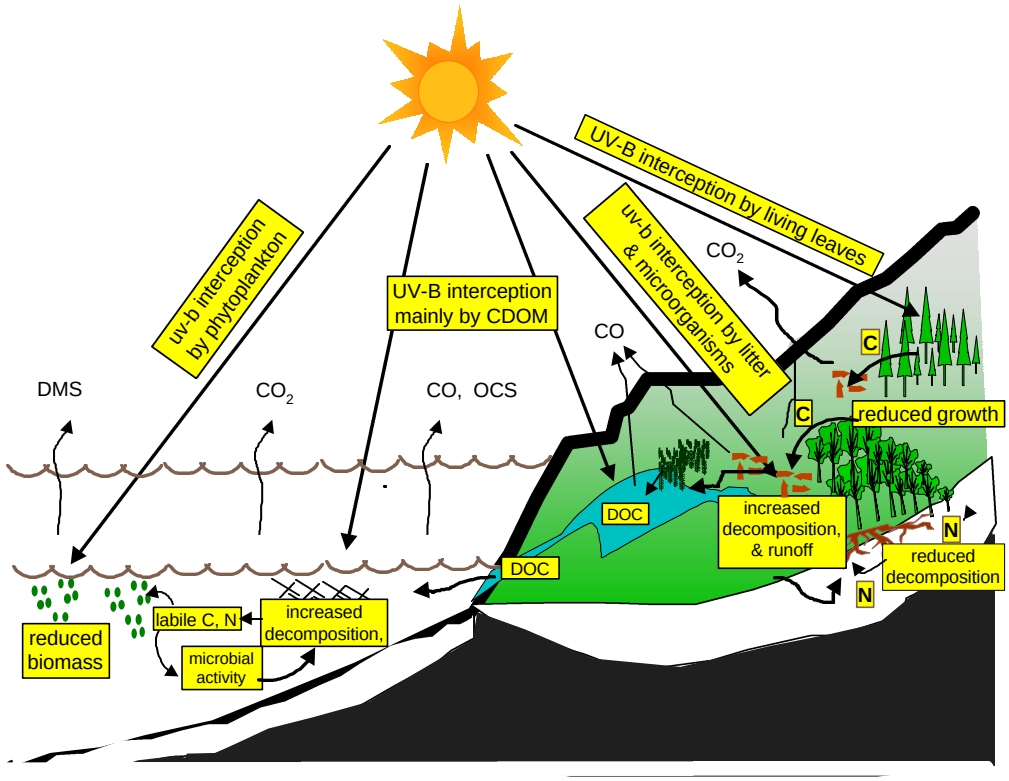

Fig. 5.1 Conceptual model illustrating the potential effects of enhanced UV radiation on biogeochemical cycles in freshwater, marine and terrestrial ecosystems. The effects involving living organisms, e.g. reduced plant growth, are species- and/or exposure-dependent.

Terrestrial Ecosystems

Two linked processes are of particular potential significance in biogeochemical cycling in terrestrial ecosystems: 1) the cycling of carbon including its capture (photosynthesis), storage (biomass and soil organic matter content) and release (plant and soil organism respiration) and 2) the cycling of mineral nutrients such as nitrogen upon which plant production and ecosystem productivity are dependent. The exchange of trace carbon and nitrogen gases between terrestrial systems and the air also is dependent on these processes. This section on terrestrial ecosystems reviews actual and potential effects of UV-B (direct and indirect) on carbon and mineral nutrient cycling and trace gas exchange.

Since the 1994 UNEP report (Zepp et al. 1994, 1995), there has been a considerable increase in the number of studies on biogeochemical cycling in terrestrial ecosystems. Current studies include a

program in which standard litter (birch leaves) is being decomposed under standard conditions in UV-B enhancement experiments in natural heathlands from the Mediterranean to the high Arctic (Paul and Moody personal. comm.), in collaboration with UV-B exclusion experiments in southern Argentinean heathlands (Caldwell and Balares pers comm). Other on-going projects include UV-B regulation of *Sphagnum fuscum* production and decomposition in the sub Arctic (Björn et al. 1998), decomposition in Dutch coastal ecosystems (Rozema et al. 1997a) and photodegradation of plant litter in high Arctic ecosystems (Johanson et al. pers comm). Current studies on UV-B impacts, and in combination with other treatments such as drought, fertilization, and perturbation on the growth and community structure of limestone grassland will also give information on some aspects of biogeochemical cycling (C.Thorpe and J.P. Grime pers. comm.)

Carbon Capture and Storage

Although many studies, but not all (Tosserams and Rozema 1995, Gehrke et al. 1996), suggest that photosynthesis and plant growth can be reduced by elevated levels of UV-B radiation, data on plant biomass and rates of net primary productivity, i.e. the standing stock of living matter expressed as g dry weight per unit ground area, and growth expressed as g dry weight per unit ground area per unit time, are difficult to find for natural ecosystems and forests. However, a review (Rozema et al. 1997a) suggests that there is little impact of UV-B radiation on the primary production of terrestrial ecosystems. Although the length of individual *Sphagnum fuscum* shoots was reduced by 20% within one

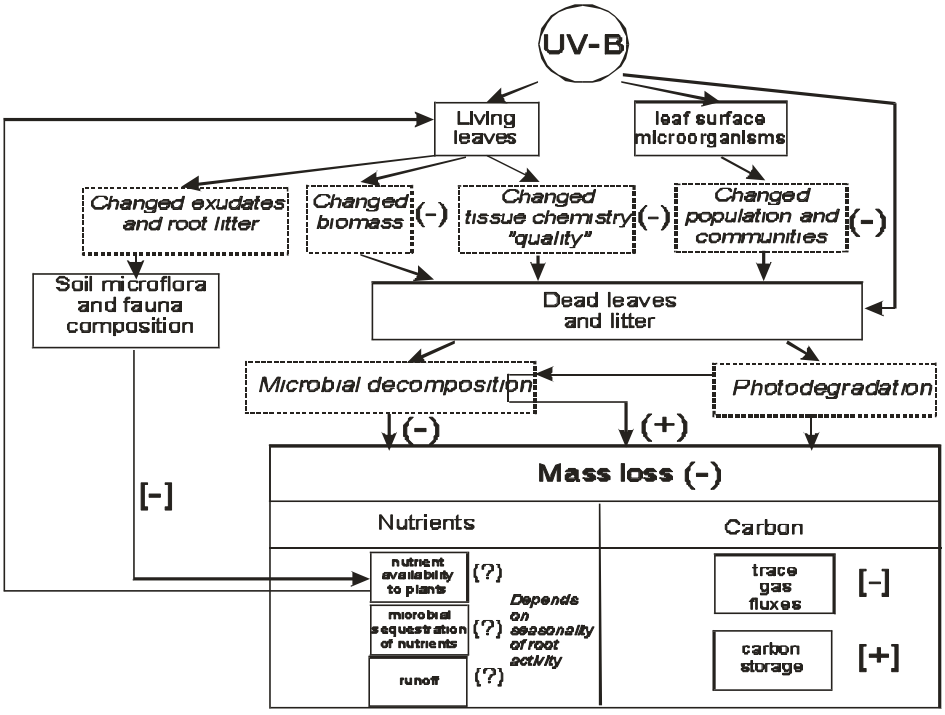

Fig. 5.2 Summarized relationships between UV-B, plant development, decomposition and carbon and mineral nutrient cycling. “+” denotes measured overall increases in processes, “-“ denotes measured overall decreases. Signs are concensuses of never more than 4 studies while those in square brackets are inferences only. Data from Gehrke et al. 1995; Klironomos and Allen, 1995; Newsham et al. 1997a, b; Rozema et al. 1997 a; Gwynn-Jones et al. pers. comm.; Johanson et al. pers. comm.

growing season (Gehrke et al. 1996), changes in biomass or primary productivity per unit ground area were not detected (C. Gehrke pers. comm.).

Data for carbon storage in soils and plant litter are not available. This is primarily due to the practical difficulties of running experiments over time scales sufficiently long to allow high UV-B treated plant material to senesce naturally and be decomposed under experimental conditions. However, it is clear that the impact of increased UV-B on carbon storage will result from a balance between the impacts on plant productivity and decomposition of litter.

The latter process will be determined by direct photodegradation of litter exposed to elevated UV-B, indirect UV-B generated changes in the tissue quality of living plant tissues exposed to elevated UV-B and direct UV-B impacts on the mortality and growth of fungal decomposers and other soil decomposer organisms (Moorhead and Callaghan 1994)(Figure 2). The outcome of these processes is likely to be ecosystem and even species specific, although similar trends may occur within functional plant groups.

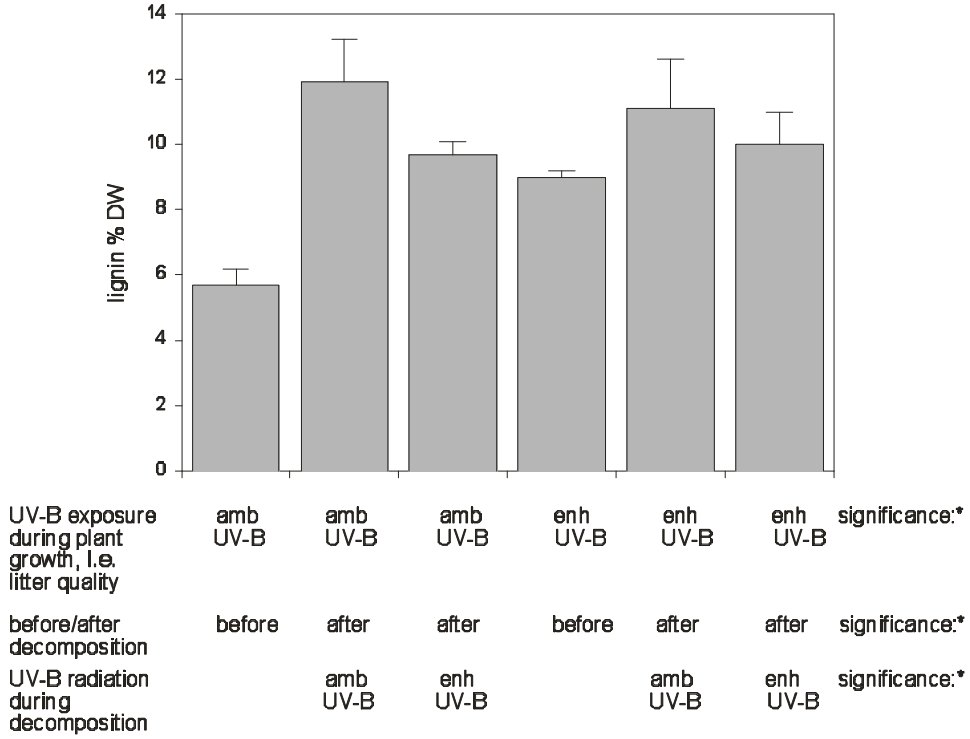

Fig. 5.3 Impacts of UV-B radiation on photodegradation and microbial decomposition of dune grassland litter. “amb” = ambient, “enh” = enhanced. The row labelled "UV-B exposure during plant growth" describes the conditions under which the grass was grown, *i.e.* under ambient or enhanced UV-B. The row "before/after decomposition" denotes whether the litter had not ("before") or had ("after") been subjected to decomposition in litter bags on a dune grassland under ambient or enhanced UV-B. The row "UV-B radiation during decomposition" describes the conditions under which the litter was decomposed, *i.e.* under ambient or enhanced UV-B. For example, the last bar indicates the lignin content of the litter from grass grown under enhanced UV-B and then subjected to enhanced UV-B during the period of decomposition. The primary effect observed was an increase in lignin content and a reduction in decomposition rates for the leaf litter of plants grown under enhanced UV-B. Exposure of the decomposing leaf litter to enhanced UV-B decreased the increase in lignin content, indicating that lignin photodegradation was stimulated. From Rozema et al. 1997a.

Indirect Effects of UV-B on Decomposition. Litter chemistry. Chemical changes in leaves (e.g. increased lignin) of the dune grassland species Calamagrostis epigeios, a grass, were induced by elevated UV-B and resulted in reduced decomposition (Figure 5.3) (Rozema et al. 1997b). (Here and throughout section 5.2 the term 'elevated UV-B' corresponds to a 15% reduction in the total ozone column at the location of the experiment or source material). In contrast, UV-B supplementation over three growing seasons had no significant effects on plant chemistry (nitrogen concentration (g N per g dry weight), C:N ratio and in some cases water soluble phenolic compounds) in Rubus chamaemorus, a perennial herb, or Calluna vulgaris, heather, (Moody et al., submitted). Nevertheless, chemical analysis at the end of the experiment revealed an indirect UV-B effect on nitrogen concentration, where nitrogen loss was significantly greater from litter of plants grown at enhanced UV-B (Moody et al., submitted), even though there were no significant differences in mass loss.

Such differences in type and magnitude of litter chemistry response among plant species or functional types may be a function of the protective barrier in leaves (cuticle, epidermis), leaf angles or relative amount of self-shading. All these factors can modify the exposure of internal leaf tissues to UV-B.

It is commonly accepted that exposure to enhanced UV-B leads to accumulation of flavonoids in leaf tissue (e.g. Gwynn-Jones and Johanson 1996). Such accumulation results from up-regulation of phenylpropanoid metabolism, which may also lead to an increase in a wide range of phenolic compounds. We have limited knowledge about the role of these compounds in decomposition although some are known to inhibit this process.

Rate of Decomposition and Mass Loss. Elevated UV-B reduced the rate of decomposition of Vaccinium uliginosum leaf litter by changing the chemical composition of the litter (Gehrke et al. 1995). Similarly a 3% lower mass loss of oak litter at enhanced UV-B was attributable to altered leaf chemistry as carbon content was 5% less compared to ambient arrays (Newsham et al. 1997b) and the mass loss of High-Arctic Salix polaris leaf litter was retarded by field exposure to enhanced UV-B (Johanson et al. pers. comm.).

More recently, no differences have been shown in the decomposition rates of *Rubus chamaemorus* leaf material field- grown for 12 months and decomposed at enhanced UV-B for over 24 months (Moody et al. submitted). However, when litter was produced under enhanced UV-B for two seasons and then decomposed in the field under these same conditions, there was a small (5.5%) but significant increase in mass loss compared with controls. Although, when present, the impacts of increased UV-B on decomposition and mass loss are small, long periods of decomposition and responses of geographically widespread ecosystems will amplify the importance of the impacts.

Direct Effects of UV-B on Decomposition

Microbial Diversity. Several researchers have shown changes in microbial communities associated with decomposing litter following exposure to enhanced UV-B (Gehrke et al. 1995, Newsham et al. 1997a, Newsham et al. 1997b, Moody et al. submitted). It could be hypothesized that observed changes in microbial respiration measured during the controlled decomposition of litter in microcosms are a result of UV-B induced changes in the microbial decomposer community. Evidence has been provided in support of this hypothesis (Gehrke et al. 1995; Moody et al. submitted). In the latter study the fungal decomposer community isolated from litter decomposing under ambient UV-B was signifi-

cantly different from that of litter from elevated UV-B. Of the two most frequently isolated species, *Aspergillus fumigatus* was found to dominate at ambient UV-B while at elevated UV-B *Penicillium purpurogenum* was dominant.

Photodegradation. Research has shown evidence of increased litter photodegradation at enhanced UV-B (Gehrke et al. 1995; Rozema et al. 1997b; Fig. 5.3). Nevertheless the impacts of such effects may sometimes be off-set by UV-B induced chemical changes in leaves (e.g. increased lignin), and the balance between the opposing processes might be difficult to relate to the causes.

However, no significant *direct* effects of UV-B (*i.e.* effects of UV-B treatments imposed during decomposition) were found on decomposition measured as mass loss (Moody et al. submitted) of *Rubus* litter. This lack of direct effects was consistent in *Rubus* litter taken from plants grown under ambient and elevated UV-B for two growing seasons and subsequently decomposed in the field for 12 months, and in *Rubus* litter collected at Moor House (the site of origin of the plant material) and decomposed under ambient and elevated UV-B for 24 months.

In practice, canopy structure and phenology (seasonal development of leaf expansion, etc.) determine whether the UV-B influences decomposition by changes in leaf chemistry or by direct photodegradation. Evergreen and wintergreen canopies in which senescent leaves and litter are shaded by living leaves would be expected to show indirect UV-B impacts as the living leaves attenuate UV-B. In contrast, simple seasonal canopies in which litter is exposed during Spring e.g. bracken fern, would be expected to show photodegradation.

Mineral Nutrient Cycling

In many natural ecosystems, mineral nutrient cycling is critical in supporting plant production. In agricultural and forest ecosystems, any constraint of increased UV-B radiation on mineral nutrient cycling can conceptually be modified by increasing fertilizer application and the outcome will rely on economics. Any change in mineral nutrient cycling could affect biodiversity of organisms which is often relatively high when mineral nutrient supply is limited.

Mineral Nutrient Capture. In some natural ecosystems, nitrogen input via nitrogen fixation in free-living and symbiotic cyanobacteria is an important process. It has been shown that UV-B irradiation of soil in field experiments stimulated nitrogen fixation by 17 and 19% at irradiances of 0.34 and 0.49 W m^{-2} while irradiances of 0.77 W m^{-2} decreased nitrogen fixation by 13% when compared with untreated controls (Uralets 1991). In the High Arctic, recent preliminary results suggest that field exposure to enhanced UV-B significantly reduces cyanobacterial nitrogen fixation (B.Solheim pers. comm.).

Studies on the nitrogen fixing cyanobacterium *Nostoc* showed that survival and growth were virtually arrested after 120 min of UV irradiation due to impairment of energy transfer from phycobiliproteins to the photosynthetic reaction centers (Sinha et al. 1995). Such results suggest that increased UV-B could reduce nitrogen availability to plants in natural ecosystems (e.g. polar and alpine and those of moist soils during early successional stages), and some agro-ecosystems (e.g. rice paddy fields). See also Chapter 4.

There is evidence that UV-B radiation can also affect mineral nutrient capture by plants (Weih et al. in press). It is believed that this is mediated through the modification of the soil organism community. UV-B can affect rhizosphere organisms that are fed primarily by root-derived substances. In experiments on *Acer saccharum* mycorrhizae associated with the tree seedlings were inhibited by UV-B irradiation of the plants and there was a stimulation of the saprobe/pathogen soil organisms (Kilrono-

mos and Allen 1996). Although implications for mineral nutrient capture by the plants was not apparently investigated, it can be assumed that reduced mycorrhizal activity would reduce **mineral** nutrient uptake by the plant while increased saprobe activity could result in **mineral** nutrient immobilization and competition between microbes and plant roots.

Mineral Nutrient Allocation within Plants. There is much, and increasing, evidence that the exposure of green leaves to elevated UV-B radiation changes the allocation of plant **mineral** nutrients between various organic molecules (Rozema et al 1997a). Most commonly, UV-B induced pigment production and changes in phenols and tannins diverts nitrogen and carbon to refractory compounds which can affect herbivory (digestibility) and microbial decomposition of plant tissues. In recent decomposition experiments on dwarf shrub litters from a sub arctic heathland, elevated UV-B decreased tannin content, but did not significantly change the contents of nitrogen, phosphorus and the C:N ratio in contrast to CO_2 at 660 ppm which changed all of these chemical components of the litters (Gwynn-Jones et al, unpublished). However, exposure of other plants to elevated UV-B increased tannin and also the refractory material lignin (Gehrke et al. 1995; Rozema et al. 1997a).

Interactions With Other Co-Occurring Environmental Change Variables

Enhanced UV-B is only one of many aspects of global environmental change. Other factors that are currently changing are well documented, including changes in atmospheric CO_2 concentrations, surface temperatures (both cooling and warming trends have been recorded), and nitrogen deposition. Both high temperatures and elevated CO_2 (660 ppm) concentrations can affect plant tissue chemical composition ("quality") (see Gwynn-Jones et al. 1996) while increased temperatures can increase decomposition rates thereby enhancing mineral nutrient turnover but reducing carbon storage. If the main effect of increased temperature is to reduce the moisture content of soil and plant litter, then the opposite effects occur. Possible synergism between increased temperatures, elevated CO_2 and increased UV-B radiation could have significant consequences for biogeochemical cycling. Recent unpublished work. has shown that there is no combined effect of elevated CO_2 (660 ppm) and elevated UV-B radiation on the CO_2 respired from litters of the sub arctic dwarf shrubs *Vaccinium myrtillus* and *V. uliginosum* (Gwynn Jones et al., personal communication). However, both treatments applied singly affected decomposition in counter intuitive ways: both increased CO_2 (660 ppm) and elevated UV-B increased decomposition. There are precedents for elevated CO_2 increasing decomposition (e.g. Robinson et al. 1997) but the result for the UV-B treatment is opposite to that obtained for just one of the species, *V. uliginosum.* (Gehrke et al. 1995)

Interactions between drought and elevated UV-B treatments in a limestone grassland community apparently increase competitive vigor under field conditions in those species which adopt a more compact, xerophytic growth form (C.Thorpe and J.P.Grime pers. comm.). Indeed, increased UV-B may pre-dispose certain plants to become drought tolerant and therefore more competitive during drought periods.

UV-B and Trace Gas Exchange

In addition to uptake of CO_2 by photosynthesis and release of CO_2 by respiration, living plants and litter release the chemically important gases CO and volatile organic compounds (VOC), such as isoprene, to the atmosphere (IPCC, 1992). VOC and CO react in the troposphere to influence ozone, other oxidants and aerosols. Isoprene, in particular, plays a key role in such reactions. In addition, CO and VOC emissions affect the oxidizing capacity of the troposphere (Chapter 6) thus possibly affecting the concentration of the greenhouse gases, methane and CFCs.

Changes in UV-B have been shown to have no detectable effects on the release of isoprene from three temperate plant species (Harley et al. 1996). However, although direct effects of UV-B radiation have little effect, UV-B induced changes in the competitive balance of plants in terrestrial ecosystems could result in changes in species composition that could affect net fluxes of these chemically-important gases.

Additional results have demonstrated that plant leaves produce CO on exposure to solar radiation and that senescent and dead leaves from temperate deciduous plants and tropical grasses produce CO more rapidly than living plant leaves (Tarr et al.,1995; Zepp et al., 1996; Schade et al., 1997). Estimates of the CO source from photodegradation of non-living plant matter indicate that 50 to 200 Tg of CO may be produced annually on a global basis. This source is sufficiently large that it should not be neglected when considering the global CO budget. Wavelength studies (>300 nm) indicate that the action spectra are species dependent and that UV-B radiation produces CO from the leaves with the highest efficiency, although UV-A radiation also induces CO formation. The floor of boreal forests that have experienced fire also have been shown to be net sources of CO to the atmosphere and the production of CO is in part attributable to UV-induced photodegradation of the charred organic matter (Zepp et al., 1997)

Although terrestrial plants are believed to be an important sink of carbonyl sulfide, (OCS), the most concentrated sulfur gas in the lower atmosphere, no data are available on the effects of enhanced UV-B on plant uptake of OCS. Likewise, no results have become available on the UV-B mediated uptake of nitrogen oxides by vegetation during the past four years.

Aquatic Ecosystems

As in the case of terrestrial ecosystems, considerable new field and laboratory research has been conducted on the effects of UV radiation on aquatic biogeochemical cycles since the last report. Research has continued on UV effects on aquatic carbon capture and storage, mineral nutrient cycling, and water-air trace gas exchange.

Unlike terrestrial systems, the penetration of UV-B as well as photorepairing wavelengths of solar radiation into water is predominantly controlled by non-living organic matter, mainly the colored (chromophoric) component of dissolved organic matter (CDOM) (Figure 5.4). Enhanced UV-B can affect the balance between the biological processes that produce the organic matter and the chemical and microbial processes that degrade it**.** Observations in lakes and the sea have shown that, in stratified waters, exposure to solar UV radiation helps cause net decreases in CDOM concentrations and increases in UV-B penetration. These changes, which are reinforced by changes in climate and acidification, potentially have broad impacts on the effects of enhanced UV-B on biogeochemical cycles. See following discussion on decomposition and UV-B penetration and related considerations in Chapter 4.

Much recent research has focused on the effects of UV-B radiation on the interactions of UV-B and dissolved organic carbon (DOC). In particular, evidence is accumulating that microbial activity is enhanced by the UV-induced decomposition of polymeric components of DOC to biologically-available organic compounds and mineral nutrients. Direct photodegradation of DOC to carbon dioxide has be shown to be an important decomposition pathway in freshwater and coastal waters. UV-B effects on aquatic biogeochemical cycles, coupled with the influences of other global changes that are occurring in climate and other factors, result in changes in UV penetration and trace gas exchange.

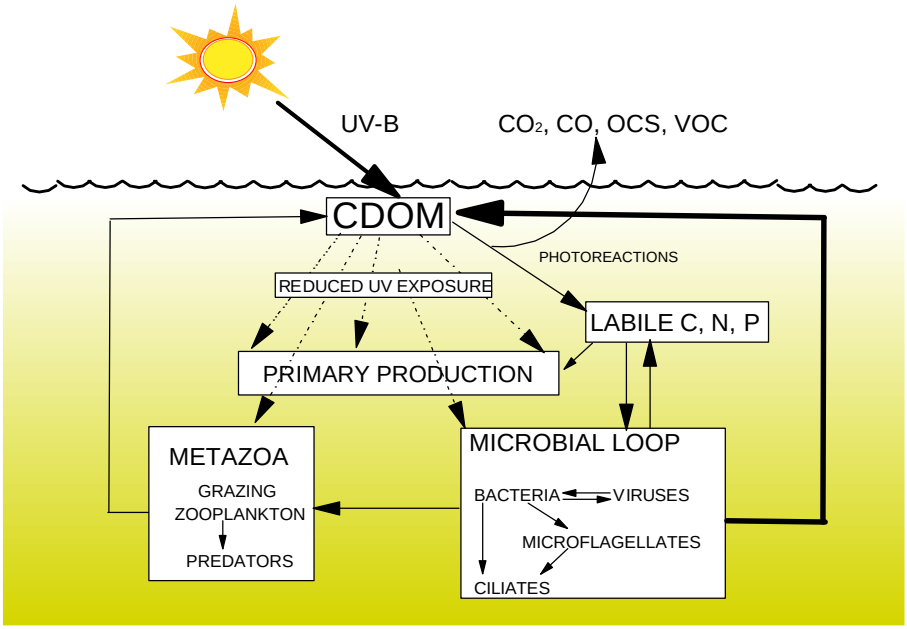

Fig 5.4 Conceptual model of the interactions of UV-B radiation with biogeochemical cycles in aquatic ecosystems.The colored dissolved organic matter (CDOM) controls the penetration of UV-B into the water. UV-B can affect the concentrations of CDOM by inducing photobleaching that is reinforced by changes in climate and acidification. Photoreactions driven by UV-B can enhance the biological availability of the CDOM and can produce various greenhouse and chemically-reactive trace gases. Adapted from Pomeroy and Wiebe (1988).

Carbon Capture and Storage

Phytoplankton communities play a central role in the creation of biomass in large lakes and the ocean and submerged and partially-submerged aquatic vegetation is the primary creator of biomass in many freshwater systems (Wetzel, 1992*)*. Freshwaters also receive inputs of allochthonous (externally produced) organic matter derived from terrestrial plants, and rivers transport large amounts (~400 teragrams (Tg) C yr^{-1}); 1 Tg=10^{12} g) of terrestrial organic matter into coastal waters. The effects of UV-B on terrestrial plant productivity which could affect freshwater and coastal systems are discussed in Section 5.2.1 and in Chapter 3 and recent research on submerged plants is considered in Chapter 4.

Photosynthesis and growth of phytoplankton can be enhanced by reducing exposure to current levels of UV-B and inhibited by enhanced UV-B such as the levels observed under the ozone hole in Antarctica (Smith et al., 1992; Karentz et al., 1994; Cullen and Neale, 1994*)*. Ozone depletion may influence the ability of the ocean to take up atmospheric CO_2 (Peng, 1992), but the net impact of a reduction in primary production on the ocean sink for atmospheric CO_2 is uncertain. In addition to photoinhibition and repair mechanisms, diurnal and depth-related changes in solar spectral irradiance, vertical mixing dynamics, microbial cycling (see below) and other factors affect net carbon storage. The net impact clearly is not a linear function of increased UV-B exposure. Recent papers have

discussed these factors in more detail (e.g. Smith and Cullen 1995; Moran and Zepp 1997; Prézelin et al. 1998, Neale et al 1998).

Although it is well-established that the chemical composition of phytoplankton, and presumably of submerged vegetation, is altered on exposure to UV-B radiation (Rozema et al., 1997b), *e.g.* through formation of UV-protecting compounds such as mycosporin-like amino acids, and that similar changes reduce the decomposability of the litter from terrestrial plants (see Section 5.2.2), there are no reports on the effects of such changes on the decomposability of the detritus derived from exposed primary producers.

Carbon storage in aquatic ecosystems reflects the balance between biomass production and decomposition processes. Research on interactions between UV-B radiation and decomposition, which has been particularly active since our last report, is considered in the following section.

Decomposition

The overall impact of UV-B on carbon storage depends on the flux of carbon and mineral nutrients through the microbial food web, which involves transfers of biomass from the primary producers to metazoa (grazing zooplankton and predators) and heterotrophic microorganisms. The latter consume a significant portion of primary production via a food web, components of which may "loop" (Figure 4.2) (Pomeroy and Wiebe, 1988). Although a large part of the biomass in aquatic ecosystems can be readily assimilated and decomposed, the decomposition of non-living terrestrial plant matter in part results in the production of colored polymers that are somewhat resistant to further microbial assimilation and breakdown. This colored organic matter, hydrophobic components of which have been referred to as "humic substances," occurs in soils and also abounds in wetlands. It contains readily-detectable lignocellulose components that have been used as indicators of the terrestrial contribution to marine organic matter (Moran and Hodson, 1994; Opsahl and Benner, 1997). The water soluble portion of the colored organic matter, i.e. the colored dissolved organic matter (CDOM), and particles of decayed organic matter find their way into streams, rivers, lakes and the coastal ocean where they have a variety of interactions with UV-B that are further discussed in this section. Algal-derived biomass similarly can be converted by microorganisms into CDOM, but the spectroscopic properties, and possibly the photochemical properties, of this autochthonous (internally-generated) CDOM differ from that of terrestrial CDOM. Recent studies in marine ecosystems indicate that the CDOM in the open ocean occurs mainly by production at depth (Siegel and Michaels, 1996). Organic matter accumulates at the air-sea interface and potential effects of UV on the sea surface microlayer have been recently reviewed (Liss and Duce 1997).

Decomposition of the DOC and particulate organic carbon (POC) in the euphotic zone (the layer of the sea in which most photosynthesis occurs) can be affected by UV-B through inhibition of microbial activity, by direct photodegradation of the CDOM and POC to CO_2 and other gases, and by UV-induced photodegradation of the persistent, polymeric components of the DOC to readily decomposable compounds.

Microbial Decomposition. Research has continued on the effects of enhanced UV radiation on bacterial activity in the sea and in freshwaters. UV-B radiation inhibits the activity of bacteria (Karentz et al., 1994; Jeffrey et al., 1996a, 1996b; Herndl et al., 1993, 1997; Reitner et al., 1997) and direct DNA damage (pyrimidine dimerization) has been demonstrated in field studies (Jeffrey et al., 1996a; 1996b). The greatest damage is observed in poorly-mixed, stratified waters. However, several recent studies in lakes, coastal waters, and the Gulf of Mexico showed that the reduction in microbial activity is attenuated with increased winds and surface layer mixing (Jeffrey et al., 1996a, 1996b) and the ac-

tivity is rapidly restored in the dark (within a few hours) via repair and regrowth (Mullerniklas et al., 1995; Jeffrey et al., 1996a, 1996b; Herndl et al., 1997; Kaiser and Herndl, 1997). Thus, adverse effects of UV light on microbial activity can change the timing and location of microbial decomposition of labile organic matter in the upper ocean. Very recent studies have shown that the amount and distribution of marine viruses also are affected by UV-B radiation in the sea (Weinbauer et al. 1997; Wilhelm et al., 1998). Viruses can influence microbial diversity and activity, including decomposition. Research on light-induced repair of sunlight-damaged viruses in the presence of bacteria, probably by photoreactivation, has recently appeared (Wilhelm et al. 1998).

Photochemical Degradation. In addition to its effects on microbial activity, UV-B has direct effects on decomposition. Research on these effects has expanded since the last report in 1994. There is substantial evidence now that UV-B plays an important role in the photodegradation of CDOM by solar radiation, based on a number of action spectra that have been reported (De Haan 1993; Frimmel 1994; Hongve 1994; Moran and Zepp 1997). The radiation amplification factors for photodegradation of the DOM from a variety of marine and freshwater environments fall in the same range as those calculated for most environmental effects considered in this report (see Chapter 1). When sunlight is absorbed by the polymeric CDOM of either freshwater or marine origin, it is cleaved to a variety of photoproducts and its average molecular weight is reduced (for review see Moran and Zepp 1997). Major processes include the direct photochemical mineralization of the CDOM and POC to inorganic compounds, *e.g.*, carbon monoxide, carbon dioxide, and other forms of dissolved inorganic carbon (DIC) (Salonen and Vähätalo 1994; Miller and Zepp 1995; Granéli et al. 1996; Amon and Benner 1996). Direct photodegradation can account for a large fraction of the mineralization of DOC in certain freshwaters and coastal regions (Miller and Zepp 1995; Granéli et al. 1996; Amon and Benner 1996).

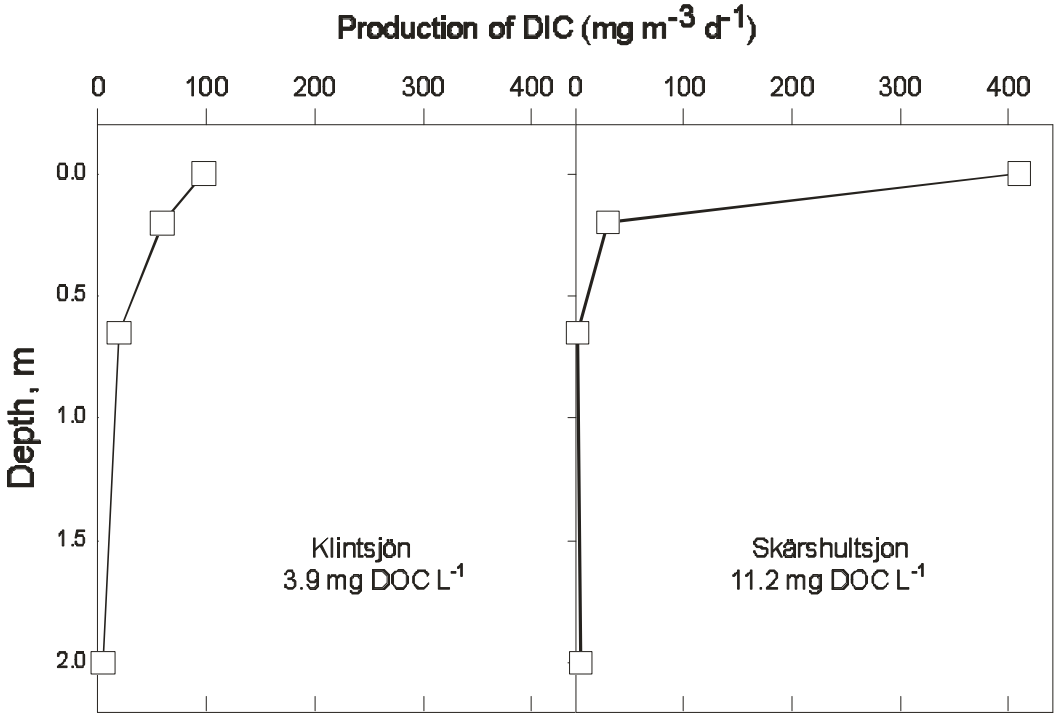

Fig. 5.5 Photodegradation rates of DOM to DIC versus depth in two Swedish lakes. The samples were filtered to remove algae and most bacteria prior to their exposure in quartz containers placed at the depths indicated. Depth-integrated production of DIC by photodegradation was about the same in both lakes (Granéli et al., 1996).

The photodegradation processes involve, in part, reactive oxygen species such as superoxide ions and hydroxyl radicals, that are produced on absorption of UV radiation (Blough and Zepp, 1995; Zafiriou et al., 1998; Vaughan and Blough 1998). Complexation of freshwater DOC with iron affects its reaction with these reactive transients and stimulates its photodegradation (Voelker and Sulzberger, 1996; Voelker et al. 1997; Gao and Zepp, 1998). These recent studies have important implications for marine biogeochemistry. Marine scientists have long puzzled over the fate of riverine organic matter on entry to the ocean. Isotopic studies indicate that the DOC in the open ocean is primarily of marine origin although some terrestrial character would have been expected. The recent studies of DOC oxidation induced by sunlight support the hypothesis that DOC photodegradation likely accounts for the observed loss of riverine DOC in the coastal ocean (Miller and Zepp 1995). This hypothesis is further supported by observations that the lignocellulose component of DOC is substantially reduced in coastal regions (Opsahl and Benner 1997).

Photochemically-mediated Bacterial Decomposition. Other organic photoproducts are formed that can be significantly more biologically active than the parent DOM from which they were formed (Wetzel et al. 1995; Lindell et al. 1995, 1996; Mullerniklas et al., 1995; Corin et al., 1996; Dahlen et al., 1996; Miller and Moran, 1997; Moran and Zepp 1997; Kaiser and Herndl, 1997; Jørgensen et al. 1998). Photochemically-mediated bacterial degradation (via photochemical modification of otherwise refractory CDOM into biologically labile forms) also can be an important pathway for decomposition of CDOM, although the relative importance of this pathway compared to direct photodegradation seems to strongly depend on the DOC source. In the few previous studies that have quantitatively compared rates of photochemical carbon gas formation to rates of photochemical formation of identifiable, low molecular weight biologically labile photoproducts, carbon gases have been found to be produced at rates many-fold higher (see Moran and Zepp 1997). A recent study, however, indicated that alternating exposure of CDOM to bacterial degradation and sunlight results in the production of a large pool of unidentified substrates that are readily assimilated by microorganisms. These substrates are believed to be comprised of low molecular weight carbonyl compounds that have not yet been identified and higher molecular weight compounds and humic substances that have been modified by exposure to sunlight although not transformed into small, chemically-identifiable compounds (Miller and Moran 1997).

A few recent studies have indicated that photoreactions driven by UV-B can reduce the microbial availability of certain organic substrates such as peptone and algal exudates (Thomas and Lara, 1995; Naganuma et al. 1996; Tranvik and Kokalj 1998). In at least one study, it was demonstrated that this phenomenon involves light-induced cross-linking between the CDOM and algal exudates (Tranvik and Kokalj 1998).

Decomposition and UV-B Penetration. Recent research has shown that CDOM controls the penetration and spectral distribution of solar radiation in freshwater (Williamson et al., 1996; Morris and Hargreaves, 1997) and marine systems, especially in regions close to the coast (Blough and Green, 1994; Kirk, 1994; Nelson and Guarda, 1995; Siegel and Michaels, 1996; Siegel and Dickey, 1987; Degrandpre et al., 1996; Vodacek et al, 1997). The presence of CDOM can reduce UV exposure, but CDOM also attenuates visible radiation required for photosynthesis. Efforts to model these competing spectral effects of CDOM on primary production in the sea have recently appeared (Arrigo et al. 1996). Spectral changes accompanying the decomposition of CDOM can influence the extent of UV-B damage and photorepair. The photochemical degradation and/or photochemically-mediated bacte-

rial decomposition of CDOM are accompanied by loss of absorbance, *i.e.* photobleaching, in the UV and visible spectral regions. The spectral changes observed on irradiation of freshwater CDOM involve fractional decreases in the UV region that exceed those observed in the visible region (Morris and Hargreaves 1997; Miller and Zepp unpublished results). Seasonal and depth changes in UV attenuation have been attributed to the photochemical degradation of CDOM, coupled with climatic factors such as stratification of the water caused by temperature gradients (Siegel and Dickey, 1987; Morris and Hargreaves, 1997; Vodacek et al., 1997*)*. Such spectral changes, which affect the penetration of damaging UV-B as well as that of photorepairing radiation, are discussed in more detail in Section 5.3.4 and in Chapter 4. Photochemical changes in CDOM absorbance can affect both UV-B penetration as well as photorepair of UV-B damage (see Chapter 4 for more details).

Mineral Nutrient Cycling

Research has continued on the effects of UV-B on nitrogen-fixing organisms in aquatic environments. This research has demonstrated that UV-B generally photoinhibits nitrogen fixation (see Chapter 4).

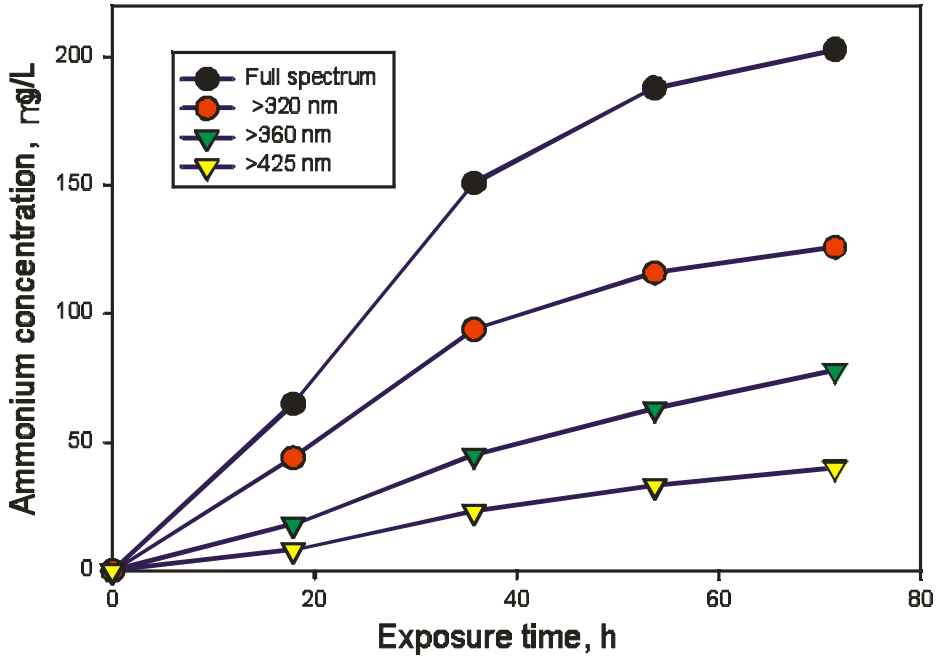

Fig. 5.6. Photoproduction of ammonium from dissolved organic nitrogen on exposure of water from a coastal Florida river to simulated solar radiation with and without filters in place to block various wavelengths of UV radiation. The results show that exclusion of UV-B radiation (<320 nm) reduces the photo-ammonification rate (Bushaw et al., 1996).

Interactions of UV-B radiation and CDOM provide a pathway for the release of N-containing nutrients. The photodegradation of DOM by sunlight is accompanied by the formation of readily assimilable nitrogen compounds such as ammonium (Bushaw et al. 1996) and amino acids (Bushaw et al. 1996, Jørgensen et al. 1998) that stimulate microbial activity. Under N-limiting conditions, the release of nitrogenous photoproducts from DOM photodegradation was found to significantly increase rates of bacterial growth (Bushaw et al. 1996). This photodegradation process occurs most efficiently with UV-B radiation (Figure 5). Nitrogen-rich photoproducts are likely to be of greatest biological interest

in coastal regions and other ecosystems where plankton are nitrogen limited and concentrations of light-absorbing DOM are high (Bushaw et al. 1996).

Enhanced UV-B can indirectly affect phosphorus cycling as well. Dissolved organic matter forms complexes with extracellular phosphatase enzymes that inhibit their activity. Exposure to UV-B degrades these complexes thus restoring their activity (Wetzel 1992).

Interactions with Other Co-Occurring Environmental Change Variables

Changes in UV-B interact with other global changes in precipitation chemistry (e.g. acidification) and climate (e.g. precipitation and temperature changes). Recent research has provided examples of such interactions in aquatic ecosystems. Reductions of dissolved organic carbon in Canadian lakes during the past two decades reportedly have resulted in increased penetration of UV-B (Schindler et al., 1996). The DOC reductions are attributable in part to photodegradation of the DOC by the increased solar UV radiation over this region, coupled with acid deposition and climatic variation. The latter appear to involve a combination of changes in input of DOC by runoff associated with precipitation changes coupled with stratification caused by warming. Stratification reduces transport of UV-absorbing compounds from deeper water into the euphotic zone, thus enhancing the effects of CDOM photodegradation on UV-B penetration. A study of the algal-derived pigments preserved in Canadian lake sediments has revealed a possible link between climate-related precipitation changes and UV exposure (Leavitt et al. 1997). Sedimentary profiles of the occurrence of UV-induced pigments indicate that the greatest UV penetration correlated with periods of drought, when levels of UV-absorbing CDOM in the lakes were minimal. As noted above, UV-B damage to microorganisms is strongly affected by changes in wind-induced mixing of the upper layers of aquatic environments.

Increased pollution of aquatic environments by substances derived from the usage of fossil fuels, such as polycyclic aromatic hydrocarbons (PAH), also may interact with increased UV-B to affect biogeochemical cycles. Studies conducted since the 1994 report have provided extensive evidence that ultraviolet light greatly enhances the toxicity of PAH to aquatic organisms (Ankley et al. 1994; Ankley et al. 1995).

UV-B and Trace Gas Exchange

Volatile Organic Compounds. A variety of VOC are emitted to the atmosphere from the ocean. These compounds participate in chemical reactions in the boundary layer that influence oxidative capacity and concentrations of oxidants and carbonyl compounds such as formaldehyde. Emissions of isoprene, in particular, can affect boundary layer processes. Recent research indicates that VOC production in the upper ocean is driven by a combination of DOM photoreactions and of biological activity in seawater. For example, a recent report indicates that emissions of isoprene and other VOC vary seasonally in the North Sea and Southern Ocean, with the highest emissions during summer (Broadgate et al., 1997). Isoprene emissions were well-correlated with chlorophyll concentrations, indicating that phytoplankton or reactive DOM released by phytoplankton are the source (Broadgate et al. 1997). Presumably, then, adverse effects of increased UV-B on phytoplankton are altering the emissions of isoprene.

Emissions of other VOC, however, did not correlate well with chlorophyll concentrations, indicating that other natural processes must account for VOC production as well. VOC emissions via these other processes are stimulated by enhanced UV-B (Riemer et al., submitted). Factors influencing the photoproduction of VOC from DOM have been investigated (Riemer et al., submitted). The photoproduction rate of DOM to form VOC in filtered seawater exposed to sunlight was approximately

proportional to the absorbance of the water. Action spectra showed that solar UV-B radiation is predominantly responsible for VOC photoproduction in sunlight. The formation rates of these compounds were decreased with increasing oxygen concentration. Other results indicated that high concentrations of alkenes are present in surface regions of Antarctic waters under the ozone hole (Atlas et al, 1994). These studies of VOC production provide additional evidence of linkage between ozone depletion, changes in surface UV-B irradiance, chemical exchange at the air-sea interface and the oxidative capacity of the marine boundary layer.

Carbon Monoxide. CDOM photoreactions are believed to be the main source of CO in seawater; CO loss has been ascribed primarily to exchange to the atmosphere and microbial metabolism. As a result of these processes, CO emissions from the sea follow a diurnal pattern with maximum near surface ocean concentrations greatly exceeding saturation during daylight. Strong gradients of CO were observed in the lowest 10 meters of the atmosphere over the Atlantic Ocean (Springer-Young et al. 1996) in the boundary layer near the Azores during the Marine Aerosol and Gas Exchange (MAGE) program. The samples nearest the ocean surface were some 50 ppb higher than at the 10 meter altitude sampling inlet. These data showed that that the ocean is a source of CO to the atmosphere and that this source influences the marine boundary layer concentration of CO.

New research results have shown that the photoproduction of CO in the ocean is induced mainly by the UV component of solar radiation (Atlas et al., 1994; Zafiriou, unpublished results). Quantum yields (the quantum yield is the fraction of absorbed radiation that results in photoreaction) for CO production at wavelengths >297 nm were highest in the UV-B region. (Zafiriou, unpublished results). These results are similar to those previously reported for CO photoproduction in freshwaters.

Recent estimates of the global oceanic CO source range from 13 Tg/yr up to 1200 Tg/yr (Erickson 1989; Zuo and Jones 1995; Bates et al. 1995). The estimate of 1200 Tg/yr is based on the assumption that most of the CO that is produced in the upper ocean escapes to the atmosphere. This assumption is supported by observations that CO uptake by chemoautotrophic bacteria are quite slow under environmental conditions and by observations of CO oxidation in the Sargasso Sea, a region of the ocean that is very oligotrophic with low microbial biomass. On the other hand, the much-lower estimate of 13 Tg/yr was derived using extensive data on CO concentrations in near-surface seawater and the air above the Pacific Ocean. This flux estimate was computed by air-sea exchange equations that use measured CO concentrations and exchange coefficients that are related to wind speed. The large difference between CO flux estimates derived from these two approaches is not understood at this point. One possibility is that there must be a major oceanic CO sink that has not been previously identified. If so, the magnitude of that sink must be approximately as large as the atmospheric sink for CO that involves reaction with the hydroxyl radical. Alternatively, the CO concentrations determined on the cruises may not have been measured at depths and locations optimal for computing realistic global fluxes.

Sulfur Gases. Atmospheric sulfur plays an important role in the radiative balance of the atmosphere. Dimethyl sulfide (DMS) is strongly related to the amount, activity and species assemblage of surface ocean primary producers. DMS is related to certain species of phytoplankton (Bates et al., 1987, 1992; Keller et al., 1989). The transfer of DMS from the ocean to the atmosphere and the subsequent transformation into sulfate particles may influence the atmospheric radiative balance (Twomey, 1977; Charlson et al., 1987; Andreae et al., 1988; Atkinson, 1989; Ayers and Gras, 1991; Boers et al., 1994). To a first approximation, one may expect that since DMS is closely related to primary productivity of certain phytoplankton species, the DMS concentration may decrease during ozone depletion episodes when primary productivity decreases. Stimulation of microbial activity by enhanced UV-B (discussed above) possibly may reinforce the decrease in DMS concentration, since bacterial decomposition of

DMS is its major fate in the upper ocean (Kieber et al., 1996). This simplistic view must, however, be tempered by the observation that DMS concentrations increase when the phytoplankton are stressed by zooplankton grazing (Dacey and Wakeham, 1986). Should increased UV-B at the ocean surface stress the phytoplankton in similar ways as zooplankton grazing, then the DMS input may actually increase, at least for a short period of time.

In addition to possible effects of solar UV radiation on the biological production of DMS, DMS emissions to the atmosphere also are affected by competing marine biological and photochemical decomposition of DMS in the upper ocean. DMS photoreactions accounted for 7% to 40% of the total turnover of DMS in the surface mixed layer of the equatorial Pacific Ocean (Kieber et al., 1996). The photoreaction involved conversion to dimethylsulfoxide, but the yield of the conversion was only 14%.

One of the best examples that demonstrates the link between the natural atmospheric sulfur cycle and the physical climate system is the observational evidence that links the satellite derived stratus cloud optical depth and observed DMS derived cloud condensation nuclei (CCN) concentrations at Cape Grim, Australia (Boers et al. 1994). Statistical evidence indicates that the optical depth of the clouds is correlated with the number of CCN in the atmosphere. Thus, any UV-B related changes at the surface of the ocean that results in the alteration in DMS flux to the atmosphere and the subsequent formation of particles would also alter the atmospheric radiation budget for the affected region.

Carbonyl sulfide (OCS) is produced in surface seawater by the photochemical degradation of dissolved organic matter (Uher and Andreae 1997; Flöck and Andreae 1996) and the ocean is a net source of OCS to the atmosphere. The photoproduction of OCS and CO in the sea appear to be linked by competitive reactions involving free radical species (Pos et al., 1998). Recent research has confirmed that estuaries are a major source of marine OCS with sea-to-air fluxes over 50 times more than those from the open ocean (Zhang et al. 1998); sedimentary processes also contribute to estuarine production of OCS. Observed seasonal variations of OCS at a coastal site in Australia were consistent with an ocean source in the summer and a land-based sink during the winter (Griffith et al. 1998). The action spectra for the production of OCS in seawater indicate that this gas is produced most efficiently by the UV-B component of solar radiation (Erickson and Eaton, 1993**;** Zepp and Andreae 1994, Weiss et al. 1995a, Weiss et al. 1995b). There is a competition between UV-B induced OCS production, OCS loss by hydrolysis and vertical mixing which leads to temporal and spatial changes in OCS air-sea fluxes (Najjar et al. 1995). The strong influence of vertical mixing in the upper ocean may also influence the near surface pool of OCS available for air-sea exchange (Najjar et al. 1995). For example, recent data indicate that certain regions of the ocean can act as a net sink for OCS, especially during the winter when UV-B irradiance is low or in low productivity, warm regions of the sea where CDOM and reactive organosulfur concentrations are low and hydrolysis rates are high (Ulshofer et al., 1995; Weiss et al, 1995b). The hydrolysis of OCS, although it reduces the efficiency of its escape to the atmosphere, results in the production of sulfide in the upper ocean (Elliot et al., 1989) where it can affect various metals cycles.

OCS is very resistant to oxidation and/or removal by processes in the troposphere, although uptake by terrestrial plants and soils can reduce its concentration (see earlier discussion in Section 5.2.6). On transport of OCS to the stratosphere it is converted to background stratospheric sulfate. Until recently it was believed that OCS was the major source of background sulfate in the stratosphere, but recent studies indicates that other sources are more significant (Chin and Davis 1995; Kjellstrom 1998). Much of the OCS that enters the stratosphere returns to the troposphere and there is increasing evidence that other tropospheric sulfur gases, such as SO2, may be contributing to the background stratospheric sulfate layer.

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References

Amon, R.M.W. and Benner, R. 1996. Photochemical and microbial consumption of dissolved organic carbon and dissolved oxygen in the Amazon River system. *Geochimica et Cosmochimica Acta* 60:1783-1792.

Andreae, M.O., Berresheim, H., Andreae, T.W., Kritz, M.A., Bates, T.S. and Merrill, J.T. 1988. Vertical distribution of dimethyl sulfide, sulfur dioxide, aerosol ions and radon over the north-east Pacific Ocean. *Journal of Atmospheric Chemistry* 6:149-173.

Ankley, G.T., Collyard, S.A., Monson, P.D. and Kosian, P.A. 1994. Influence of ultraviolet light on the toxicity of sediments contaminated with polycyclic aromatic hydrocarbons. *Environmental Toxicology and Chemistry* 11:1791-1796.

Ankley, G.T., Erickson, R.J., Phipps, G.L., Mattson, V.R., Kosian, P.A., Sheedy, B.R. and Cox, J.S. 1995. Effects of light intensity on the phototoxicity of fluoranthene to a benthic macroinvertebrate. *Environmental Science and Technology* 29:2828-2833.

Arrigo, K.R. and Brown, C.W. 1996. Impact of chromophoric dissolved organic matter on UV inhibition of primary productivity in the sea. *Marine Ecology Progress Series* 140:207-216.

Atkinson, R. 1989. Kinetics and mechanisms of the gas-phase reactions of the hydroxyl radical with organic compounds. *Journal of Physical & Chemical Reference Data, Monograph* 1:171-187.

Atlas, E., Lueb, R., Madronich, S., Prezelin, B. and Smith, R. 1994. Dissolved trace gas measurements and UV effects near the Antarctic Peninsula during ICECOLORS ’93. *EOS Transactions of the American Geophysical Union* 75:377.

Ayers, G.P. and Gras, J.L. 1991. Seasonal relationships between cloud condensation nuclei and aerosol methane sulphonate in marine air. *Nature* 353:834-835.

Bates, T.S., Charlson, R.J. and Gammon, R.H. 1987. Evidence for the climatic role of marine biogenic sulphur. *Nature* 329:319-321.

Bates, T.S., Kelly, K.C., Johnson, J.E. and Gammon, R.H. 1995. Regional and seasonal variations in the flux of oceanic carbon monoxide to the atmosphere. *Journal of Geophysical Research* 100:23093-23102.

Bates, T.S., Lamb, B.K., Guenther, A., Dignon, J., and Stoiber, R.E. 1992. Sulfur emissions to the atmosphere from natural sources. *Journal of Atmospheric Chemistry* 14:315-337.

Björn, L.O., Callaghan, T.V., Gehrke, C., Johanson, U., Sonesson, M. and Gwynn-Jones, D. 1998. The problem of ozone depletion in northern Europe. *Ambio* (in press).

Blough, N.V. and Green, S.A. 1994. Spectroscopic characterization and remote sensing of non-living organic matter. In *Role Of Non-Living Organic Matter in the Earth’s Carbon Cycle*, Zepp, R. G. and Sonntag, C. (eds.), Wiley, New York, pp. 42-57.

Blough, N.V. and Zepp, R.G. 1995. Reactive oxygen species in natural waters. In *Active oxygen: Reactive oxygen species in chemistry*, Foote, C.S. et al. (eds), Chapman and Hall, pp. 280-333.

Boers, R., Ayers, P. and Gras, J.L. 1994. Coherence between seasonal variation in satellite-derived cloud optical depth and boundary layer CCN concentrations at a mid-latitude Southern Hemisphere station. *Tellus Series B-Chemical and Physical Meteorology* 46(N2):123-131.

Broadgate, W.J., Liss, P.S, and Penkett, S.A. 1997. Seasonal emissions of isoprene and other reactive hydrocarbon gases from the ocean. *Geophysical Research Letters* 24:2675-2678.

Bushaw, K.L., Zepp, R.G., Tarr, M.A., Schulz-Jander, D., Bourbonniere, R.A., Hodson, R.E., Miller, W.L., Bronk, D.A., Moran, M.A. 1996. Photochemical release of biologically available nitrogen from dissolved organic matter. *Nature* 381:404-407.

Charlson, R.J., Lovelock, J.E., Andreae, M.O., Warren S.G. 1987. Oceanic phytoplankton, atmospheric sulfur, cloud albedo and climate. *Nature* 326:655-661.

Chin, M. and Davis, D.D. 1995. A reanalysis of carbonyl sulfide as a source of statospheric background sulfur aerosols. Journal of Geophysical Research 100(D):8993-9005.

Corin, N., Backlund, P. and Kulovaara, M. 1996. Degradation products formed during UV-irradiation of humic waters. Chemosphere 33:245-255.

Cullen, J.J. and Neale, P.J. 1994. Ultraviolet radiation, ozone depletion, and marine photosynthesis, *Photosynthesis Research* 39:303-320.

Dacey, J.W. and Wakeham, S.G. 1986.Oceanic dimethylsulfide: Production during zooplankton grazing on phytoplankton. *Science* 233:1314-1316.

Dahlén, J., Bertilsson, S. and Pettersson, C. 1996. Effects of UV-A irradiation on dissolved organic matter in humic surface waters. Environment International. 22:501-506.

Degrandpre, M.D., Vodacek, A., Nelson, R.K., Bruce, E.J. and Blough, N.V. 1996. Seasonal seawater optical properties of the U.S. Middle Atlantic Bight. *Journal of Geophysical Research* 101:22727-22736.

Elliot, S., E. Lu, E. and Rowland, F.S. 1989. Rates and mechanisms for hydrolysis of carbonyl sulfide in natural waters. *Environmental Science and Technology* 23:458- 461.

Erickson, D.J. III. 1989. Ocean to atmosphere carbon monoxide flux: Global inventory and climate implications. *Global Biogeochemical Cycles* 3:305-314.

Erickson, D.J. III and Eaton, B. E. 1993. Global biogeochemical cycling estimates with CZCS satellite data and general circulation models. *Geophysical Research. Letters* 20:683-686.

Flöck, O.R. and Andreae, M.O. 1996. Photochemical and non-photochemical formation and destruction of carbonyl sulfide and methyl sulfide in ocean waters. *Marine Chemistry* 54:11-26. Flöck

Frimmel, F. H. 1994. Photochemical aspects related to humic substances. Environment International 20:373-385.

Gao, H. and Zepp, R.G. 1998. Factors influencing photoreactions of dissolved organic matter in a coastal river of the southeastern United States. *Environmental Science and Technology* 32:2940-2946.

Gehrke C., Johanson, U., Callaghan, T.V., Chadwick, D. and Robinson, C.H. 1995. The impact of enhanced ultraviolet-B radiation on litter quality and decomposition processes in Vaccinium leaves from the Subarctic. *Oikos* 72:213-222.

Gehrke C., Johanson U., Gwynn-Jones D., Björn, L.O., Lee, J.A., Callaghan T.V, and Sonesson, M. 1996. Single and interactive effects of enhanced ultraviolet-B radiation and increased atmospheric CO_2 on terrestrial sub-arctic ecosystems. In *Plant Ecology in the Sub-arctic Swedish Lapland. Ecological Bulletins*, Karlsson, P.S. and Callaghan, T.V. (eds.), 45:192-203.

Granéli, W., Lindell, M. and Tranvik, L. 1996. Photooxidative production of dissolved inorganic carbon in lakes of different humic content. *Limnology and Oceanography* 41:698-706.

Griffith, D.W.T., Jones, N.B. and Matthews, W.A. 1998. Interhemispheric ratio and annual cycle of carbonyl sulfide (OCS) total column fro ground-based solar FTIR spectra. *Journal of Geophysical Research* D 103:8447-8454.

Gwynn-Jones, D. and Johanson, U. 1996. Growth and pigment production in two subarctic grass species under four different UV-B irradiation levels. *Physiologia Plantarum* 97:701-707.

Gwynn-Jones, D., Björn, L.O., Callaghan, T.V., Gehrke, C., Johanson, U., Lee, J.A. and Sonesson, M. 1996. Effects of enhanced UV-B radiation and elevated concentrations of CO_2 on a Subarctic heathland. In *Carbon Dioxide Populations, and Communities*, Körner, C. and FA Bazzaz, F. A. (eds.), Academic Press, pp. 197-207.

Harley, P., Deem, G., Flint, S. and Caldwell, M. 1996. Effects of growth under elevated UV-B on photosynthesis and isoprene emission in *Quercus gambelii* and *Mucuna pruriens. Global Change Biology* 2:149-154.

Herndl, G., Müller-Niklas, G. and Frick, J. 1993. Major role of ultraviolet-B in controlling bacterioplankton growth in the surface layer of the ocean. *Nature* 361:717-719.

Herndl, G.J., Brugger, A., Hager, S., Kaiser, E., Obernosterer, I., Reitner B. and Slezak, D. 1997. Role of ultraviolet-B radiation on bacterioplankton and the availability of dissolved organic matter. *Plant Ecology* 128:42-51.

Hongve, D. 1994. Sunlight degradation of aquatic humic substances. *Acta Hydrochimica Hydrobiologie* 3:117-120.

IPCC, Watson, R., Meira-Filho, L.G., Sanhueza, E. and Janetos, A. 1992. Greenhouse gases: Sources and sinks. Chapter A1, In *Climate Change 1992: Supplementary Report to the IPCC Scientific Assessment*, Houghton, J.T. Callander, B.A. and Varney, S.K. (eds.), pp. 28-46.

Jeffrey, W.H., Pledger, R.J., Aas P., Hager, S., Coffin, R.B., Vonhaven, R. and Mitchell, D.L. 1996a. Diel and depth profiles of DNA photodamage in bacterioplankton exposed to ambient solar ultraviolet radiation. *Marine Ecology-Progress Series* 137:283-291.

Jeffrey, W.H., Aas P., Lyons, M.M., Coffin, R.B., Pledger, R.J. and Mitchell, D.L. 1996b. Ambient solar-radiation induced photodamage in marine bacterioplankton. *Photochemistry and Photobiology* 64:419-427.

Jørgensen, N.O.G., Tranvik, L., Edling, H., Granéli, W. and Lindell, M. 1998. Effects of sunlight on occurrence and bacterial turnover of specific carbon and nitrogen compounds in lake water. *FEMS Microbology Ecology* 25:217-227.

Kaiser, E. and Herndl, G.J. 1997. Rapid recovery of marine bacterioplankton activity after inhibition by UV radiation in coastal waters. *Applied and Environmental Microbiology* 63:4026-4031.

Karentz, D., Bothwell, M.L., Coffin, R.B., Hanson, A., Herndl, G.J., Kilham, S.S., Lesser, M.P., Lindell, M., Moeller, R.E., Morris, D.P., Neale, P.J., Sanders, R.W.,Weiler, C.S. and Wetzel,

R.G. 1994. Impact of UV-B radiation on pelagic freshwater ecosystems: report of working group on bacteria and phytoplankton. *Archive fur Hydrobiologie* 43:31-69.

Keller, M.D., Bellows, W.K. and Guillard, R.R.L. 1989. A survey of dimethyl sulfide production in 12 classes of marine phytoplankton, In E. Saltzman and W. Cooper *(eds.) Biogenic Sulfur in the Environment*, American Chemical Society, Washington, D.C., pp. 167-182.

Kieber, D.J., Jiao, J., Kiene, R.P. and Bates, T.S. 1996. Impact of dimethylsulfide photochemistry on methyl sulfur cycling in the equatorial Pacific Ocean. *Journal of Geophysical Research* 101:3715-3722.

Kilronomos, J.N. and Allen, M.F., 1996. UV-B mediated changes on below-ground communities associated with the roots of Acer saccharum. *Functional Ecology* 9:923-930.

Kirk, J.T.O. 1994. Optics of UV-B radiation in natural waters. *Archiv fur Hydrobiologie* 43:1-16.

Kirk, J.T.O. 1994. *Light and Photosynthesis in Aquatic Ecosystems*, Cambridge University Press, New York, pp. 46-84.

Kjellstrvm, E. 1998. A three-dimensional global model study of carbonyl sulfide in the troposphere and lower stratosphere. *Journal of Atmospheric Chemistry* 29:151-177.

Leavitt, P.R., R.D. Vinebrooke, R.D. Donald, D.R., Smol, J.P. and Schindler, D.W. 1997. Past ultraviolet radiation environments in lakes derived from fossil pigments. *Nature* 388:457-459.

Lindell, J.J., Granéli, W. and Tranvik, L.J. 1995. Enhanced bacterial growth in response to photochemical transformation of dissolved organic matter. *Limnology and Oceanography* 40:195-199.

Lindell, J.J., Granéli, W. and Tranvik, L.J. 1996. Effects of sunlight on bacterial growth in lakes of different humic content. *Aquatic Microbial Ecology* 11:135-141.

Liss, P and Duce, R. 1996. *The Sea Surface and Global Change*, Cambridge University Press, Cambridge.

Miller, W.L. and Zepp, R.G. 1995. Photochemical production of dissolved inorganic carbon from terrestrial organic matter: significance to the oceanic organic carbon cycle. *Geophysical Research Letters* 22:417-420.

Miller, W.L. and Moran, M.A. 1997. Interaction of photochemical and microbial processes in the degradation of refractory dissolved organic matter from a coastal marine environment. *Limnology and Oceanography* 42:1317-1324.

Moody, S.A., Ineson, P., Ayres, P.G. and Paul, N.D.1998. Effect of elevated ultraviolet-B radiation (290-320 nm) on the chemical quality and decomposition of Rubus chamaemorus and Calluna vulgaris. submitted

Moorhead, D. and Callaghan, T.V. 1994. Effects of increasing UV-B radiation on decomposition and soil organic matter dynamics: a synthesis and modeling study. *Biology and Fertility of Soils* 18:19-26.

Moran, M.A. and Hodson, R.E. 1994. Dissolved humic substances of vascular plant origin in a coastal marine environment. *Limnology & Oceanography* 39:762-771.

Moran, M.A. and Zepp, R.G. 1997. Role of photoreactions in the formation of biologically labile compounds from dissolved organic matter. *Limnology and Oceanography* 42:1307-1316.

Morris, D.P. and Hargreaves, B.R. 1997. The role of photochemical degradation of dissolved organic matter in regulating UV transparency of three lakes on the Pocono Plateau. *Limnology and Oceanography* 42:239-249.

Mullerniklas, G., Heissenberger, A., Puskaric, S. and Herndl, G.J. 1995. Ultraviolet-B radiation and bacterial metabolism in coastal waters. *Aquatic Microbial Ecology* 9:111-116.

Naganuma, T., Konishi, T., Inoue, T., Nakane, T. and Sukizaki, S. 1996. Photodegradation or photo-alteration? Microbial assay of dissolved organic matter. *Marine Ecology Progress Series* 135:309-310.

Najjar, R.G., Erickson, D.J. III, and Madronich, S. 1995. Modeling the air-sea fluxes of gases formed from the decomposition of dissolved organic matter: carbonyl sulfide and carbon monoxide, In *Role Of Non-Living Organic Matter in the Earth's Carbon Cycle*, Zepp, R.G. and Sonntag, C. (eds.), Wiley, pp. 107-132.

Neale, P.J., Davis, R.F. and Cullen, J.J. 1998b. Interactive effects of ozone depletion and vertical mixing on photosynthesis of Antarctic phytoplankton. *Nature* 392:585-589.

Nelson, J.R. and Guarda, S. 1995. Particulate and dissolved spectral absorption on the continental shelf of the southeastern United States. *Journal of Geophysical Research* 100:8715-8732.

Newsham, K.K., Low, M.N.R., McLeod, A.R., Greenslade, P.D. and Emmett, B.A. 1997a. Ultraviolet-B radiation influences the abundance and distribution of phylloplane fungi on pedunculate oak (Quercus robur). *New Phytologist* 136:287-297.

Newsham, K.K., McLeod, A.R., Roberts, J.D., Greenslade, P.D. and Emmett, B.A. 1997b. Direct effects of elevated UV-B radiation on the decomposition of Quercus robur leaf litter. *Oikos* 79:592-602.

Opsahl, S. and Benner, R. 1997. Distribution and cycling of terrigenous dissolved organic matter in the ocean. *Nature* 386:480-482.

Peng, T.-H. 1992. Possible effects of ozone depletion on the global carbon cycle. *Radiocarbon* 34:772-779.

Pomeroy, L.R. and Wiebe, W. J. 1988. Energetics of microbial food webs. *Hydrobiologia* 159:7-18.

Pos, W.H., Riemer, D.H. and Zika, R.G. 1998. Carbonyl sulfide (OCS) and carbon monoxide (CO) in natural waters:evidence of a coupled production pathway. *Marine Chemistry* 62:89-101.

Prézelin, B.P., Moline, M.A. and Matlick, H.A. 1998. ICECOLORS '93: Spectral UV radiation effects on Antarctic frazil ice algae. In *Antarctic Sea Ice Biological Processes,Interactions, and Variability, Antarctic Research Series 73*, Lizotte, M.P. and Arrigo, K.R. (eds.), American Geophysical Union, pp. 45-83.

Reitner, B., Herndl, G.J. and Herzig, A. 1997. Role of ultraviolet-B radiation on photochemical and microbial oxygen consumption in a humic rich shallow lake. *Limnology and Oceanography* 42:950-960.

Riemer, D.D., Pos, W.H., Milne, P. J. and Zika, R.G., Photochemical production rates on non-methane hydrocarbons (NMHCs) in seawater: Relation to dissolved organic matter (DOM). *Journal of Geophysical Research*, submitted.

Robinson, C., Michelsen, A., Lee, J.A., Whitehead, S.J., Callaghan, T.V., Press, M.C. and Jonasson, S. 1997. Elevated CO_2 affects decomposition of Festuca Vivipara (L) sM. litter and roots in

experiments simulating environmental change in two contrasting arctic ecosystems. *Global Change Biology* 3:37-49.

Rozema, J., Tosserams, M., Nelissen, H.J.M., van Heerwaarden, I., Broekman, R.A. and Flierman, N. 1997a. Stratospheric ozone reduction and ecosystem processes: enhanced UV-B radiation affects chemical quality and decomposition of leaves of the dune grassland species Calamagrsotis epigeios. *Plant Ecology* 128:284-94.

Rozema, J., van de Staaij, J., Björn, L.O. and Caldwell, M. 1997b. UV-B as an environmental factor in plant life: stress and regulation. *Trends in Ecology and Evolution* 12:22-28.

Salonen, K. and Vähätalo, A. 1994. Photochemical mineralization of dissolved organic matter in Lake Skjervatjern. *Environment International* 20:307-312.

Schade, G.W., Hofmann, R.M. and Crutzen, P.J., 1997. Emissions from degrading grass biomass in a South African savanna, and estimate of a global CO source strength of photochemically induced CO emissions from degrading plant matter. *EOS Transactions of the American Geophysical Union* 78:1023.

Schindler, D.W., Curtis, J.P., Parker, B.R. and Stainton, M.P. 1996. Consequences of climate warming and lake acidification for UV-B penetration in North American boreal lakes. *Nature* 379:705-708.

Siegel, D.A. and Dickey, T.D. 1987. Observations of the vertical structure of the diffuse attenuation coefficient spectrum. *Deep Sea Research* 34:547-563.

Siegel, D.A. and Michaels, A.F. 1996. Quantification of non-algal light attenuation in the Sargasso Sea: Implications for biogeochemistry and remote sensing, *Deep-Sea Research II* 43:321-346.

Sinha, R.P., Lebert, M., Kumar, H.D. and Haeder, D.P. 1995. Disintegration of phycobilisomes in a rice field cyanobacterium Nostoc sp. following UV irradiation. *Biochemistry and Molecular Biology International* 37:697-706.

Smith, R.C., Prezelin, B.B., Baker, K.S., Bidigare, R.R., Boucher, N.P., Coley, T., Karentz, D., MacIntyre, S., Matlick, H.A., Menzies, D., Onderusek, M., Wan Z., and Waters K.J. 1992. Ozone depletion: Ultraviolet radiation and phytoplankton biology in Antarctic waters. *Science* 255:952-959.

Smith, R.C. and Cullen, J.J. 1995. Effects of UV radiation on phytoplankton. *Reviews of Geophysics* Supplement:1211-1223.

Springer-Young, M., Erickson, D.J. III, and Carsey, T. 1996. Carbon monoxide gradients in the marine boundary layer of the Atlantic Ocean. *Journal of Geophysical Research* 101(D):4479-4484.

Tarr, M.A., Miller, W.L. and Zepp, R.G. 1995. Direct photoproduction of carbon monoxide from plant matter. *Journal of Geophysical Research* 100(D):11,403-11,413.

Thomas, D.N. and Lara, R.J. 1995. Photodegradation of algal derived dissolved organic carbon. *Marine Ecology Progress Series* 116:309-310.

Tosserams, M. and Rozema, J. 1995. Effects of ultraviolet-B radiation (UV-B) on growth and physiology of the dune grassland species *Calamagrostis epigeios*. *Environmental Pollution* 89:209-214.

Tranvik, L. and Kokalj, S. 1998. Decreased biodegradability of algal DOC due to interactive effects of UV radiation and humic matter. *Aquatic Microbial Ecology* 14:301-307.

Twomey, S. 1977. *Atmospheric Aerosols*. Elsevier. New York. 301p.

Uher, G. and Andreae, M.O. 1997. Photochemical production of carbonyl sulfide in North Sea water: a process study. *Limnology and Oceanography* 42:432-442.

Ulshofer, U.S., Uher, G., Andreae, M.O. 1995. Evidence for a winter sink of atmospheric carbonyl sulfide in the northeast Atlantic Ocean. *Geophysical Research Letters* 22:2601-2604.

Uralets, T.I. 1991. Field study of the effect of long term UV-B irradiation on the nitrogen-fixing ability of soil. *Kosmicheskaya Biologiya 1 Aviakosmicheskaya Meditsina* 25:15-17.

Vaughan, P. and N.V. Blough, N.V. 1998. Photoproduction of hydroxyl radicals from dissolved organic carbon. *Environmental Science and Technology* accepted.

Vodacek, A., Degrandpre, M.D., Peltzer, E.D., Nelson, R.K. and Blough, N.V. 1997. Seasonal variation of CDOM and DOC in the Middle Atlantic Bight: terrestrial inputs and photooxidation. *Limnology and Oceanography* 42:674-686.

Voelker, B.M. and Sulzberger, B. 1996. Effects of fulvic acid on Fe(II) oxidation by hydrogen peroxdie. *Environmental Science and Technology* 30:1106-1114.

Voelker, B.M., Morel, F.M.N., Sulzberger, B. 1997. Iron redox cycling in surface waters: Effects of humic substances and light. *Environmental Science and Technology* 31:104-1011.

Weih, M., Johanson, U. and Gwynn-Jones, D., 1998. Growth and nitrogen utilization in seedlings of mountain birch (*Betula pubescens* ssp. *tortuosa*) as affected by ultraviolet radiation (UV-A and UV-B) under laboratory and outdoor conditions. *Tree, Function and Structure* (in press).

Weinbauer, M.G., Wilhelm, S.W., Suttle, C.A., Garza, D.R. 1997. Photoreactivation of ultraviolet-radiation induced DNA damage is a major process controlling viral infectivity in the sea. *Applied and Environmental Microbiology* 63:2200-2205.

Weiss, P.S., Andrews, S.S., Johnson, J.E., Zafiriou, O.C. 1995a. Photoproduction of carbonyl sulfide in South Pacific Ocean waters as a function of irradiation wavelength. *Geophysical Research Letters* 22:215-218.

Weiss, P.S.; Johnson, J.E.; Gammon, R.H.; Bates, T.S. 1995b. Reevaluation of the open ocean source of carbonyl sulfide to the atmosphere. *Journal of Geophysical Research* 100(D):23,083-23,092.

Wetzel, R.G., 1992. Gradient-dominated ecosystems: sources and regulatory functions of dissolved organic matter in freshwater ecosystems. *Hydrobiologia* 229:181-198.

Wetzel, R.G., Hatcher, P.G. and Bianchi, T.S. 1995. Natural photolysis by ultraviolet irradiance of recalcitrant dissolved organic matter to simple substrates for rapid bacterial metabolism. *Limnology and Oceanography* 40:1369-1380.

Wilhelm, S.W.,Weinbauer, M.G., Suttle, C.A., Jeffrey W.H. 1998. The role of sunlight in the removal and repair of viruses in the sea. *Limnology and Oceanography* 43:586-592.

Williamson, C.E., Stemberger, R.S., Morris, D.P., Frost, T.M. and Paulsen, S.G. 1996. Ultraviolet radiation in North American lakes: attenuation estimates from DOC measurements and implication for plankton communities. *Limnology and Oceanography* 41:1024-1034.

Zafiriou, O.C., Voelker, B.M. and Sedlak, D.L. 1998. Chemistry of the superoxide radical (O2-) in seawater: Reactions with inorganic copper complexes. *Journal of Physical Chemistry A* 102:5693-5700.

Zepp, R.G. and Andreae, M.O. 1994. Factors affecting the photochemical formation of carbonyl sulfide in seawater. *Geophysical Research Letters* 21:2812-2816.

Zepp, R.G., Callaghan, T.V. and Erickson, D. 1994. Effects of increased solar ultraviolet radiation on biogeochemical cycles. In *Environmental Effects of Ozone Depletion*, van der Leun, J.C., Tevini, M., Teramura, A.H. (eds), United Nations Environment Programme Publication, ISBN 92 807 1245 4, pp. 79-93.

Zepp, R.G., Callaghan, T.V. and Erickson, D.J. 1995. Effects of increased solar ultraviolet radiation on biogeochemical cycles. *Ambio* 24(3):181-187.

Zepp, R.G., Miller, W.L., Burke, R.A., Parsons, D.A.B. and Scholes, M.C. 1996. Effects of moisture and burning on soil-atmosphere exchange of trace carbon gases in a southern African savanna. *Journal of Geophysical Research* 101(D):23,699-23,706.

Zepp, R.G., Miller, W.L., Tarr, M.A. and Burke, R. A. 1997. Soil-atmosphere fluxes of carbon monoxide during early stages of post-fire succession in upland Canadian boreal forests. *Journal of Geophysical Research* 102(D26), 25,665-25,674.

Zhang, L., Walsh, R.S. and Cutter, G.A. 1998. Estuarine cycling of carbonyl sulfide: production and sea-air flux. *Marine Chemistry* 61:127-142.

Zuo, Y. and Jones, R.D. 1995. Formation of carbon monoxide by photolysis of dissolved marine organic material and its significance in the carbon cycling of the oceans. *Naturwissenschaften* 82:472-474.

CHAPTER 6

Changes in Tropospheric Composition and Air Quality

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Summary

Reductions in stratospheric ozone (O_3) cause increased penetration of ultraviolet-B (UV-B) radiation to the troposphere, and therefore increases in the chemical activity in the lower atmosphere (the troposphere). Tropospheric ozone levels are sensitive to local concentrations of nitrogen oxides (NOx) and hydrocarbons. Model studies suggest that additional UV-B radiation reduces tropospheric ozone in clean environments (low NOx), and increases tropospheric ozone in polluted areas (high NOx).

Assuming other factors remain constant, additional UV-B will increase the rate at which primary pollutants are removed from the troposphere. Increased UV-B is expected to increase the concentration of hydroxyl radicals (OH) and result in faster removal of pollutants such as carbon monoxide (CO), methane (CH_4), non-methane hydrocarbons (NMHCs), sulfur and nitrogen oxides, hydrochlorofluorocarbons (HCFCs), and hydrofluorocarbons (HFCs). Concentrations of peroxy radicals (both inorganic and organic) are expected to increase, leading to higher atmospheric levels of hydrogen peroxide (H_2O_2) and organic peroxides. The effects of UV-B increases on tropospheric O_3, OH, methane, CO, and possibly other tropospheric constituents, while not negligible, will be difficult to detect because the concentrations of these species are also influenced by many other variable factors (e.g., emissions).

Trifluoroacetic acid (TFA, CF_3COOH) is produced in the atmosphere by the degradation of HCFC-123 (CF_3CHCl_2), HCFC-124 (CF_3CHFCl), and HFC-134a (CF_3CH_2F), which are used as substitutes for ozone depleting substances. The atmospheric oxidation mechanisms of these replacement compounds are well established. Reported measurements of TFA in rain, rivers, lakes, and oceans show it to be a ubiquitous component of the hydrosphere, present at levels much higher than can be explained by reported sources. The levels of TFA produced by the atmospheric degradation of HFCs and HCFCs emitted up to the year 2020 are estimated to be orders of magnitude below those of concern, and to make only a minor contribution to the current environmental burden of TFA.

No significant effects on humans or the environment have been identified from TFA produced by atmospheric degradation of HCFCs and HFCs. Numerous standard short-term studies have shown that TFA has, at most, moderate toxicity.

Introduction

Ultraviolet-B radiation (280-315 nm) is sufficiently energetic to cause photolysis of atmospheric trace gases such as ozone (O_3), nitrogen dioxide (NO_2), hydrogen peroxide (H_2O_2), formaldehyde (HCHO), and nitric acid (HNO_3) (e.g.: Finlayson-Pitts and Pitts, 1986; Graedel and Crutzen, 1993). The prod-

ucts of photolysis: O, NO, OH, H, HCO, and eventually HO_2 and organic peroxy radicals, are highly reactive. The oxidizing capacity of the troposphere is controlled by these photolytic products, especially hydroxyl radicals (OH), which originate from the photolysis of ozone in the presence of water vapor:

$$O_3 + h\nu\ (\lambda \leq 330nm) \rightarrow O(^1D) + O_2$$

$$O(^1D) + H_2O \rightarrow OH + OH$$

Ozone photolysis in the troposphere is strongly dependent on the available UV-B radiation, and therefore is sensitive to absorption by stratospheric ozone. Figure 6.1 shows the rate coefficients for this process, measured at Mauna Loa (Hawaii), as a function of the amount of ozone in the path of the solar beam.

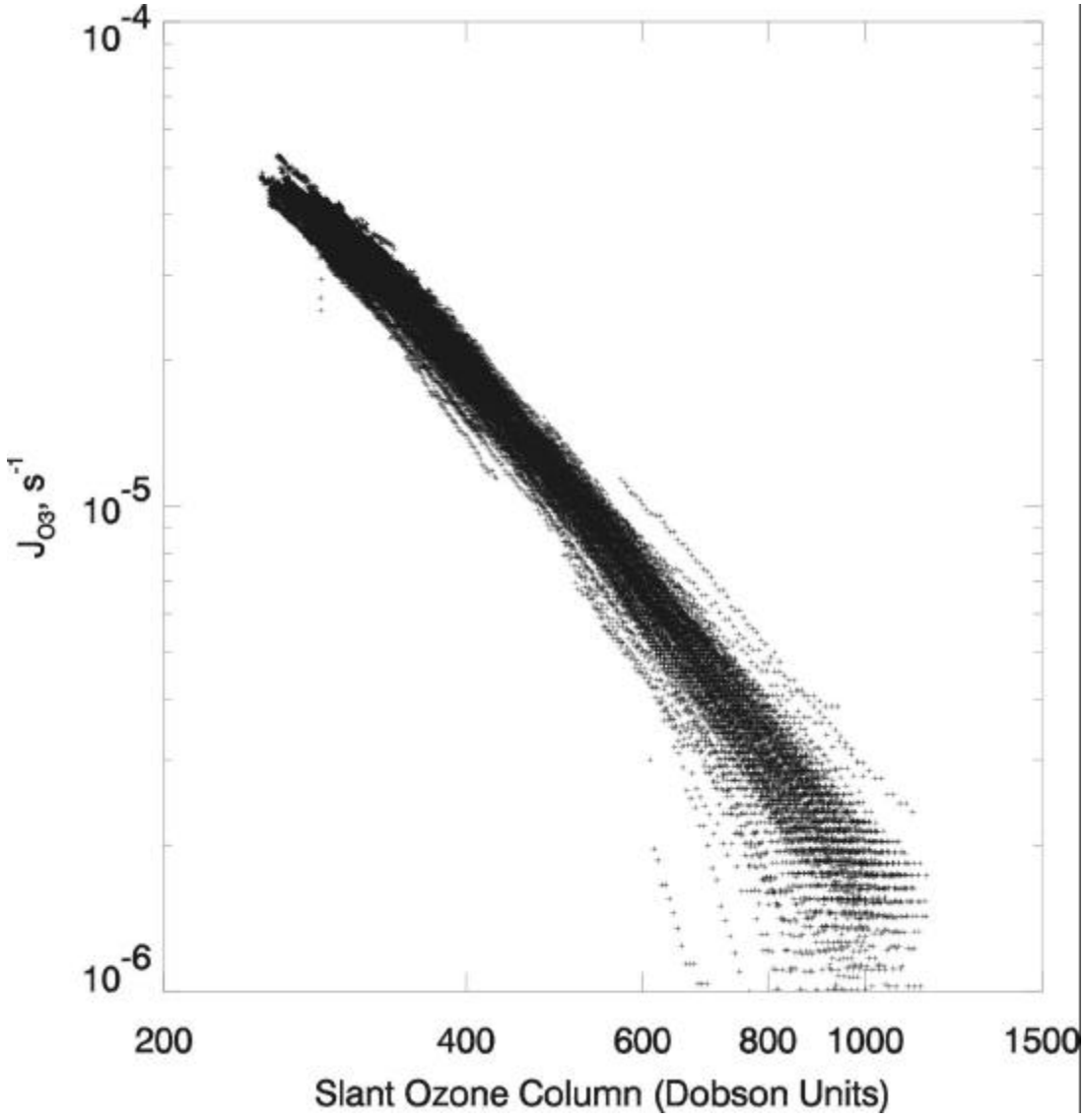

Fig. 6.1. Dependence of the rate coefficient J_{O3} for the reaction $O_3 + h\nu\ (\lambda \leq 330\ nm) \rightarrow O(^1D) + O_2$ on the amount of ozone in the path of the solar beam (the slant ozone column). The points give over 33,000 direct measurements of J_{O3} obtained with a chemical actinometer at Mauna Loa Hawaii, during 1991-1992 by Shetter et al. (1996).

Important atmospheric trace gases, such as CH_4 and other hydrocarbons, several halocarbons, NO_2, as well as sulfur containing species are primarily removed by OH. In addition, OH plays an important role in the production of tropospheric ozone. UV-B radiation is a key environmental factor controlling tropospheric chemistry. Stratospheric O_3 reduction and its consequence of increased surfaced UV-B radiation on a globe scale have been confirmed (WMO, 1994;1998). A general increase in tropospheric photochemistry and perturbations to concentrations of O_3 and OH radicals are expected. These changes may affect tropospheric composition and air quality. The magnitude of these effects is uncertain because of the complexity and non-linear nature of tropospheric chemistry and is the subject of active research. An assessment of the state of knowledge is presented below.

Hydrochlorofluorocarbons (HCFCs) and hydrofluorocarbons (HFCs), are substitutes for ozone-depleting substances. HCFCs and HFCs are degraded in the atmosphere, and some of these compounds produce trifluoroacetic acid (TFA), which has no known significant atmospheric degradation mechanism. TFA formed in the atmosphere is expected to enter the hydrosphere (mostly via precipitation), and may have detrimental effects on biota if accumulated in sufficient quantities. Production of TFA during the atmospheric degradation of HFCs and HCFCs is assessed below.

Increases in UV-B Photodissociation Rate Coefficients

Photodissociation reactions are of the general form:

$$AB + h\nu \rightarrow A + B.$$

The photodissociation rate coefficient J of species x in the troposphere is calculated by evaluating the integral equation:

$$J_x = \int F(\lambda)\, \sigma_x(\lambda,T)\, \phi_x(\lambda,T)\, d\lambda$$

In the above equation, $F(\lambda)$ represents the actinic flux and is independent of species, σ_x and ϕ_x denote the molecular absorption cross section and quantum yield (both of dependent on the species x), T is the temperature of the air parcel, and λ is the wavelength of the radiation. The sensitivity of the response of J to increases of UV-B radiation varies significantly for different species (Madronich and Granier, 1994; Krol and Van Weele, 1997). To quantify the response to ozone change, Madronich and Granier (1994) defined the sensitivity factor S_i:

$$S_i = \ln(J_i^*/J_i) / \ln(O_3/O_3^*)$$

where J_i^* and J_i are the photodissociation rate coefficients of a specific photolysis reaction corresponding to ozone column amounts O_3^* and O_3, respectively. In essence, the value of S_i gives the percent increase in J_i resulting from a 1% reduction of stratospheric ozone. Calculated values of S_i are given in Table 6.1 for selected species of tropospheric importance. As shown in the table, the S_i for O_3 is the largest, while the S_i for NO_2 is very small. The response of J_{O3} to stratospheric ozone depletion is significant while that of J_{NO2} is negligible. Values given in Table 6.1 are similar to those computed by Madronich and Granier (1994), Madronich et al. (1995; 1998), and Granier et al. (1998). Small differences stem from difference in conditions (e.g. latitudes, solar zenith angles) as well as some model differences.

Fuglestvedt et al. (1995) calculated monthly J values (for the 15th of each month) of 16 photolytic reactions from 1979 to 1993. Figure 6.2 shows the changes in global total ozone observed by satellite-based instruments, and the corresponding calculated changes in globally averaged tropospheric J_{O3}, the dissociation rate coefficient for O_3 yielding $O(^1D)$. Both are given as annual averages and normal-

ized to 1979 levels. As shown in figure 6.2, the global total ozone column densities decreased by 8 percent while the J_{O3} increased by 12 percent from 1979 to 1993.

Table 6.1. Sensitivity (S_i) of photodissociation coefficients of several molecules, to changes in total column ozone. Calculations for upper troposphere, total ozone column of 328 Dobson Units. From Ma, 1996.

Chemical Formula	Name	S_i
O_3	Ozone	1.45
HNO_3	Nitric acid	0.89
CH_3CHO	Acetaldehyde	0.73
CH_3COCH_3	Acetone	0.60
HCHO	Formaldehyde	0.38
H_2O_2	Hydrogen peroxide	0.31
CH_3OOH	Methyl hydroperoxide	0.31
N_2O_5	Dinitrogen pentoxide	0.30
NO_2	Nitrogen dioxide	0.02
HNO_2	Nitrous acid	0.01

Changes in Tropospheric Chemical Composition

Changes in O_3

Tropospheric ozone plays an important role in the atmosphere because its photolysis in the presence of water vapor is the primary source for hydroxyl radical (OH), which is responsible for the removal of many important trace gases. Moreover, it is an effective greenhouse gas (especially in the upper troposphere), is toxic to organisms, and contributes to global biogeochemistry because of its UV absorption (see Chapter 5). Ozone also causes damage to many organic materials, particularly if carbon-carbon unsaturation is present, such as in rubber products.

Tropospheric ozone stems from two processes, downward transport from the stratosphere, and in-situ photochemical production from the oxidation of hydrocarbons or carbon monoxide (CO) in the presence of NOx. Ozone is removed from the troposphere by in-situ chemistry and by deposition at the Earth's surface. The effect of decreased stratospheric ozone on the influx of stratospheric ozone into the troposphere is unknown and is not discussed here. The general effect of increased UV-B radiation on the chemical budget of ozone is discussed below.

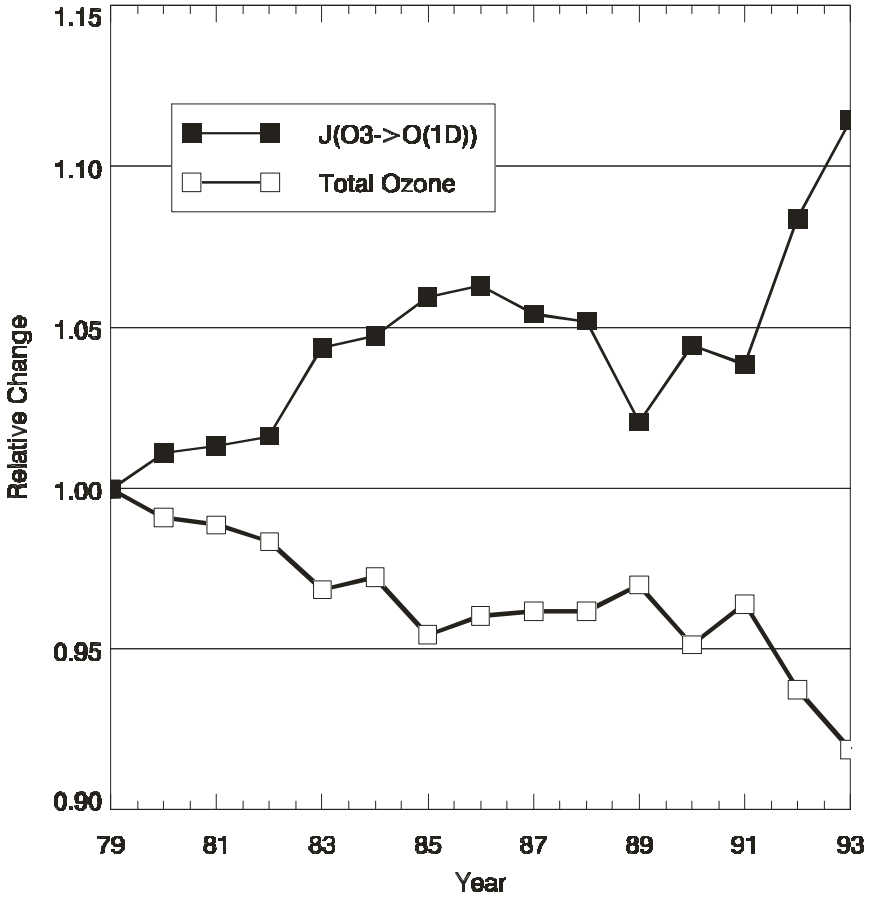

Fig. 6.2. Development of observed global total ozone densities and calculated averages of J_{O3} given as annual averages normalized to 1979 levels (from Fuglestvedt et al., 1995).

The photolysis of ozone followed by reaction of $O(^1D)$ atoms with water vapor is a major photochemical sink for O_3, and the main source of OH radicals:

$$O_3 + h\nu\ (\lambda \leq 330nm) \rightarrow O(^1D) + O_2 \qquad (R1)$$

$$O(^1D) + H_2O \rightarrow OH + OH \qquad (R2)$$

Only a small fraction of $O(^1D)$ atoms react with water vapor; most $O(^1D)$ atoms undergo relaxation to ground state $O(^3P)$ via collisions with molecular oxygen or nitrogen:

$$O(^1D) + M(N_2, O_2) \rightarrow O(^3P) + M \qquad (R3)$$

Ground state $O(^3P)$ atoms react mainly with molecular oxygen forming ozone again. The reaction of $O(^1D)$ atoms with water vapor, R2, is the rate-determining step in the photochemical loss of tropospheric ozone through the photolysis of O_3 itself. Additional losses of O_3 occur through reaction with HO_2 radicals via R4 and (to a lesser extent) through reaction of OH radicals with O_3 via R5:

$$HO_2 + O_3 \rightarrow OH + 2O_2 \qquad (R4)$$

$$OH + O_3 \rightarrow HO_2 + O_2 \qquad (R5)$$

The photochemical loss rate of ozone is therefore approximately:

$$L_{O3} = [O_3]\{ F J_{O3} + k_4[HO_2] + k_5[OH]\}$$

where F is the fraction of $O(^1D)$ that reacts with water vapor (typically a few percent).

The production of ozone in the troposphere occurs through the conversion of NO to NO_2 by peroxy radicals (HO_2 and RO_2), followed by photolysis of NO_2:

$$RO_2 + NO \rightarrow RO + NO_2 \qquad \text{(R6)}$$

$$HO_2 + NO \rightarrow OH + NO_2 \qquad \text{(R7)}$$

$$NO_2 + h\nu\ (\lambda \leq 420\ nm) \rightarrow NO + O(^3P) \qquad \text{(R8)}$$

$$O(^3P) + O_2 + M \rightarrow O_3 + M \qquad \text{(R9)}$$

Hydrocarbons and carbon monoxide (CO) provide the peroxy radicals through their oxidation and are consumed in the process, while NO_x is conserved and thus acts as catalyst in ozone formation. The conversion of NO to NO_2 by peroxy radicals is the rate-limiting step, since the photolysis of NO_2 (R8) and the subsequent reaction (R9) are rapid. The ozone formation rate is given approximately by:

$$P_{O3} = [NO]\{ k_6 [RO_2] + k_7 [HO_2]\}$$

The chemical budget of ozone in a given region is governed by the rate of net ozone production, P_{O3} - L_{O3}. The effect of increased UV-B radiation on the chemical budget of ozone, i.e. the change of P_{O3} - L_{O3}, is complex. An increase in UV-B radiation will increase J_{O3} and hence increase the rate of ozone destruction L_{O3}. Considering the production of ozone, J_{NO2} is quite insensitive to increasing UV-B radiation (see Table 6.1), but the increase of UV-B radiation will lead to increased concentrations of hydroxyl radicals and thus to increased concentrations of HO_2 and RO_2 radicals, which will enhance the production of ozone if NOx is available. An increase in UV-B radiation will enhance both destruction and production process for tropospheric ozone. Since P_{O3} is dependent on the NOx abundance and L_{O3} is, to first approximation, insensitive to NOx, the sign and magnitude of trends in tropospheric O_3 (from UV-B radiation increases) are dependent on the NOx concentration.

Several model studies have addressed the question of how tropospheric ozone may respond to UV-B increases. Liu and Trainer (1988) used a zero-dimensional model (chemistry only, no transport) to show that the sign (increase or decrease) of tropospheric ozone changes depends sensitively on the local NOx concentrations. At the high NOx levels typical of urban conditions, increases in UV-B are expected to result in higher levels of surface ozone, so that exceedances of air quality standards may be more frequent (De Leeuw and Van Rheineck Leyssius, 1991), or alternatively more stringent regulations may be required to meet current standards (Gery et al., 1987). Qualitatively similar results were found using one-dimensional models (chemistry plus vertical transport) by Thompson et al. (1990) and by Ma (1996), but with quantitative results shown to depend also on other chemical constituents (e.g. hydrocarbons, CO). The study by Ma (1996) further suggests that in the upper troposphere the changes in ozone are probably rather small, due to the usually very low concentrations of water vapor which therefore reduce the importance of reactions R1-R3 (and thus of J_{O3}). Increases in J_{O3} become more important in the middle and lower troposphere, where higher levels of water vapor

are found, and especially in the continental boundary layer if significant NOx concentrations are present. The results of these studies are sensitive to whether the calculations are carried out over polluted areas (e.g., the troposphere above industrialized regions) or more pristine regions (e.g., over remote ocean regions). Fuglestvedt et al. (1994; 1995) used a two-dimensional model of the global troposphere (i.e., allowing for variations with both altitude and latitude). Calculations suggest that at high latitudes of the Southern Hemisphere there is a large reduction in tropospheric O_3 in response to stratospheric ozone depletion in the spring, but the maximum effect, a 16 % reduction in tropospheric O_3 at 2.5 km altitude over 60-80°S, is delayed relative to the October minimum in stratospheric ozone by about 2 months, to early summer (December) when solar UV-B actinic fluxes are highest. At middle and high latitudes of the Northern Hemisphere, increased UV-B radiation is predicted to increase tropospheric O_3 in mid-spring (April) because of high levels of NOx and other ozone precursors (CO and hydrocarbons), but some reductions were also noted for high northern latitudes in late spring (May and June).

Very little observational evidence exists in support of these model calculations, due mostly to the sparcity of tropospheric ozone measurements (especially vertical profiles) and to the difficulty of attributing any observed trends to UV-B increases rather than any other of the numerous factors that also contribute to the tropospheric ozone budget, e.g., trends in emissions of the precursors NOx and hydrocarbons. The most relevant observations are those obtained in polar regions, because the changes in stratospheric ozone (and therefore tropospheric UV-B levels) are larger than those found for mid-latitudes and the tropics. Long-term observations at the South Pole indicate that some enhanced net destruction of surface O_3 may already have occurred in association with the large stratospheric ozone losses in that region (Schnell et al., 1991). More recently, Taalas et al. (1997) have studied vertical profiles obtained from balloon-borne ozone sondes over 1988-1994 at Marambio, Antarctica (64° S), and at Sodankyla, Finland (67° N). During months with substantial stratospheric ozone loss, ozone levels were found to be reduced also in the upper troposphere (6-8 km), averaging 12.8% reductions in the Antarctic and 10.0% in the Arctic. It was proposed that both reductions in the ozone flux from the stratosphere to the troposphere and the changes in the photochemistry of the upper troposphere are possible reasons for this correlation between upper tropospheric and lower stratospheric ozone concentrations. In the middle (3-5 km) and lower (0-2 km) troposphere, tropospheric ozone deviations were found to be negative over Antarctica but positive in the Arctic, in qualitative agreement with the model-derived expectations given the more polluted conditions present in the high latitudes of the Northern Hemisphere (relative to Antarctica).

Tropospheric ozone concentrations are sensitive not only to UV radiation, but also to many other factors including concentrations of nitrogen oxides, water vapor, carbon monoxide, methane and non-methane hydrocarbons. Long-term changes in the emission sources of these compounds are known to be occurring, and their effects on tropospheric ozone, relative to the effects of increased UV radiation levels, are at present highly uncertain.

Changes in HOx

The odd-hydrogen radicals ($HOx = OH + HO_2$) play a key role in tropospheric chemistry, as already illustrated in reactions R1-R9. Important sources of these radicals include the photolysis of ozone, hydrogen peroxide (H_2O_2), formaldehyde (HCHO), and several other inorganic and organic species. Table 6.1 shows that many of the corresponding photolysis rate coefficients are sensitive to the amount of stratospheric ozone.

Model calculations consistently show that increased UV-B actinic fluxes (associated with stratospheric ozone reductions) yield higher tropospheric concentrations of the HOx radicals (Liu and Trainer, 1988; Thompson et al., 1990; Madronich and Granier, 1992; Fuglestvedt et al., 1994; 1995; Ma, 1996). For example, Fuglestvedt et al. (1995) calculated that the increases in UV-B radiation over 1979-1993 have led to an increase in OH on the global scale of about 8%, with the largest fractional increases (about 40 %) found for high southern latitudes in October. The modeling study by Ma (1996) investigated the potential changes in the concentrations of OH, HO_2, and CH_3O_2 in the troposphere for a 10 percent reduction of stratospheric ozone. The concentration of OH is predicted to increase by 8 to 9 percent in the lower troposphere over global scales, due mainly to the increase in J_{O3}. The concentrations of HO_2 and CH_3O_2 are predicted to increase by about 4 to 6 percent respectively, due to the increased oxidation rate of CO and hydrocarbons by OH.

There is little direct observational evidence in support of the model-predicted increases in HOx. Several studies have attempted to estimate recent global trends in OH concentrations based on measurements of methyl chloroform concentrations and the requirement that its sources (which are fairly well known) be balanced by its atmospheric sink, which is believed to be primarily reaction with OH. The study by Prinn et al. (1995), which updates the earlier study by Prinn et al. (1992), found no statistically significant trend on OH over 1978-1990. More recently, a re-analysis of the methyl chloroform data (Krol et al., 1998) suggests a slight positive OH trend, ca. 0.4-0.5 % yr^{-1} over 1978-1993 at the five monitoring stations considered. However, such OH trends can result from many different atmospheric changes, including from increases in O_3 and H_2O_2 (precursors to OH) especially in the northern hemisphere as a result of changing emissions of NOx, CO, and hydrocarbons; from increases in water vapor concentrations above tropical oceans (possibly associated with climate warming); and from changes in temporal and geographical patterns of other human activities (e.g., biomass burning). It is at present difficult to assess how much of any observed trends in global OH concentrations can be attributed to UV-B increases stemming from stratospheric ozone depletion.

Changes in CH_4 and CO

The atmospheric concentrations of methane (CH_4) and carbon monoxide (CO) are determined by the balance between emission sources (many of which are related to human activities) and atmospheric removal, mostly by OH radicals. For CO an important additional source is the atmospheric oxidation of CH_4 and other hydrocarbons. Short-term and long-term variations in atmospheric CH_4 and CO concentrations stem from the superposition of these sources and sinks, and, when local concentration measurements are considered, from transport processes that re-distribute the gases to regions far from the original emission sources. The effect of possible tropospheric OH increases (from stratospheric ozone depletion) on the average concentrations of CO and CH_4 is estimated in many cases to be smaller than, or comparable to, the effects of changes in emission sources, and is therefore difficult to detect by inspection of the available long-term CO and CH_4 monitoring data records. The estimated effects of the tropospheric UV-B increases are not negligible (at least based on model estimates), and must therefore be given some consideration as contributing to the overall trends (Liu and Trainer, 1988; Thompson et al., 1990; Madronich and Granier, 1992, 1994; Bekki et al., 1994; Fuglesvedt et al., 1994, 1995).

Analyses of polar ice cores (Etheridge et al., 1998) indicate that pre-industrial atmospheric CH_4 concentrations were about 650 ±40 ppb. Atmospheric methane concentrations started to increase about a hundred years ago, probably because of human activities linked to rising population. The rate of increase has not been constant. In the late 1970s the rate of increase was approximately 16 ppb/yr, while in the later part of the 1980s the increase was about 9 ppb/yr. To the extent that increased UV-B

radiation is expected to lead to higher levels of OH, a correspondingly faster loss of CH_4 is expected. Figure 6.3 shows the change of CH_4 concentrations calculated with a two-dimensional model (Fuglesvedt et al., 1995), that are expected to result from increased UV-B radiation alone. The changes in the global annual growth rate are also shown. The figure shows that the increased UV-B radiation may have reduced the global methane concentration by as much as 30 ppb from 1980 to 1993, and thus may have contributed partly to the overall trend declines observed since 1980.

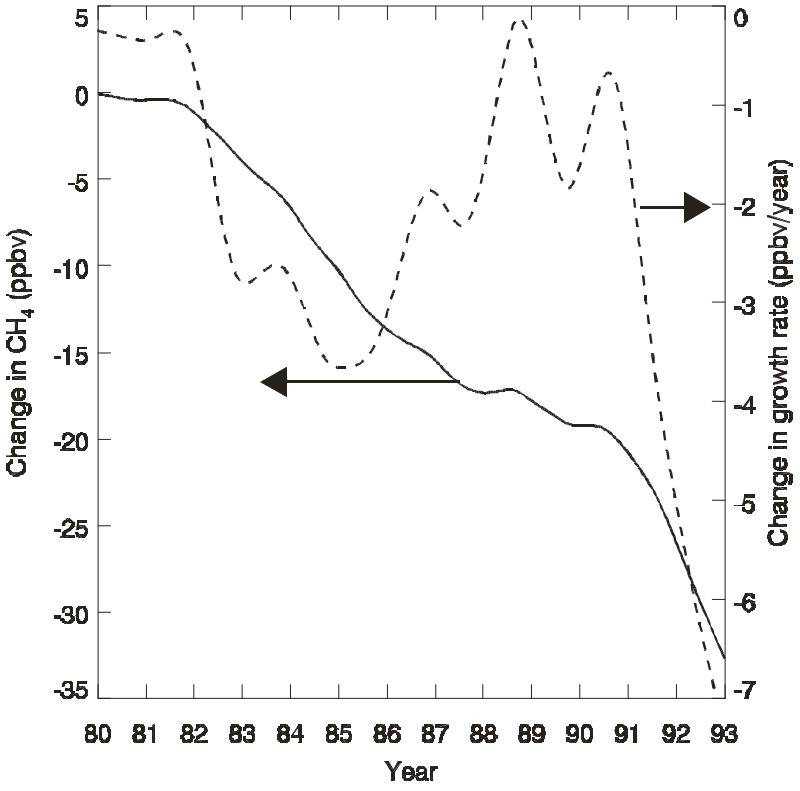

Fig. 6.3. Predicted change in the global level and growth rate of CH_4 due to UV increases (from Fuglestvedt et al., 1995).

CO concentration measurements suggest an increase in the CO abundance of 1 percent per year over the past 40 years in the Northern Hemisphere (Zander et al., 1989), while no significant trend was found in the Southern Hemisphere (Brunke et al., 1990). A large decrease in the CO concentrations was observed between 71°N and 41°S during June 1990 - June 1993, with a decrease of 15 to 18 ppbv at most stations located north of 25°N, and of 8 to 12 ppbv between 41°S and 20°N (Novelli et al., 1994). To the extent that OH increases are expected in association with stratospheric ozone depletion, global decreases in CO concentrations are expected. Granier et al. (1996) used a global three-dimensional chemical transport model to investigate possible causes for the observed CO decrease, and concluded that changes in total ozone abundance may be responsible for a global decrease in CO of about 3.5 ppbv and 1.7 ppbv in the Northern and Southern Hemispheres, respectively, accounting for about 20% of the observed CO decreases. The large remaining decreases were attributed to decreased industrial and transportation CO sources, to decreased biomass burning, and to a global decrease in surface air temperatures for several years following the eruption of Mt. Pinatubo.

An additional study, though not directly related to stratospheric ozone depletion, illustrates the sensitivity of tropospheric CH_4 and CO to stratospheric transmission of solar UV-B wavelengths. Observa-

tions show sharp increases in the growth rates of CH_4 and CO in the tropics and middle southern latitudes for several months following the eruption of Mt. Pinatubo on June 15, 1991. This volcanic eruption emitted approximately 20 Mtons of SO_2 and 3-5 km^3 of ash into the upper troposphere and lower stratosphere. Calculations using a radiative transfer model showed that the tropospheric UV-B actinic flux in the tropics was attenuated by about 12% immediately after the eruption due to direct absorption by SO_2, and was perturbed for up to 1 year after the eruption due to scattering by sulfate aerosols (Dlugokencky et al., 1996). This study suggested that the decreased UV-B flux caused a decrease of OH concentrations and therefore lead to the observed anomalously large growth rates for CH_4 and CO during late 1991 and early 1992. Methyl chloroform, whose atmospheric removal is also initiated by OH, showed a small positive anomaly in Cape Grim, Australia, consistent with observations for CH_4 at comparable latitudes. The increased growth rates were short-lived, as CH_4 and CO growth rates showed strong decreases during late 1992 and 1993 (Dlugokencky et al., 1994; Novelli et al., 1994). Bekki et al. (1994) suggested that for several years after the Mt. Pinatubo eruption, faster rates of removal of CO and CH_4 may have resulted lower stratospheric ozone (and therefore higher tropospheric UV and OH); however measurements of the isotopic composition of CH_4 are more consistent with decreased emissions, possibly from biomass burning (Lowe et al., 1997).

In summary, the potential perturbations to CO and CH_4 concentrations from increased tropospheric UV-B levels are only one of many factors contributing to the observed trends and variations. Changes in emission sources are well-recognized, although not fully quantified. Changes in OH concentrations have also likely occurred due to many factors, in addition to UV-B changes, including trends in precursor species such as hydrocarbons and NOx (and therefore tropospheric ozone), changes in emissions of biogenic (natural) hydrocarbons from changes in land use, climatic changes in temperature and water vapor, and possibly even other changes in actinic fluxes due to trends and variabilities of clouds and aerosols. The net response of atmospheric CO and CH_4 concentration is, to first order, a result of the superposition of multiple driving factors, but it remains exceedingly difficult to separate and quantify the importance of each of these contributions

Changes in H_2O_2

Hydrogen peroxide (H_2O_2) is one the principal oxidants in the troposphere, and plays an important role in the aqueous phase oxidation of SO_2 to $SO_4^=$. Model calculations (e.g., Fuglesvedt et al., 1994, 1995) suggest that tropospheric H_2O_2 concentrations should increase in response to enhanced tropospheric UV-B actinic fluxes, in parallel to the expected increases in HOx (especially HO_2). The calculated H_2O_2 increases are greatest in low NOx regions, where the formation of peroxides is the predominant fate of HOx radicals. Measurements at Eurocore (central Greenland, 72°N) showed a 50% increase of the H_2O_2 concentration in the firn/ice during last 200 years, with most of the increase having occurred between 1960 and 1988 (Sigg and Neftel, 1991). Recent studies at Summit, Greenland (72°N, in 1995) confirmed the H_2O_2 increases found in ice cores, and showed a further increase of the H_2O_2 concentration since 1988, leading to an overall increase of 60±12% during the last 150 years (Anklin and Bales, 1997). Photochemical model calculations for Summit (Neftel et al., 1995) indicate that ozone depletion over 1980-1990 (from 395 to 360 Dobson Units) gives an atmospheric H_2O_2 increases of about 7% for summer, which could account for about one third of the increase observed over that time period. Anklin and Bales proposed that the recent increase could be partly due to increasing UV-B radiation caused by the stratospheric ozone depletion and a combination of changes in tropospheric chemistry from other causes such as increased emissions of hydrocarbons and CO.

Atmospheric Production and Fate of Trifluoroacetic Acid

Hydrochlorofluorocarbons (HCFCs) and hydrofluorocarbons (HFCs) have been identified as alternatives to CFCs. Among this class of products, HCFC-123, HCFC-124 and HFC-134a are expected to degrade to give trifluoroacetic acid (TFA). Environmental TFA can also be produced during the oxidation of other organofluorine compounds released to the atmosphere by human activities, e.g. halothane and isoflurane anaesthetics. Trifluoroacetic acid is widely used in the chemical industry in processes where it is either consumed or becomes part of a chemical waste stream.

Hydrochlorofluorocarbons (HCFCs) and hydrofluorocarbons (HFCs) are used as CFC substitutes in a variety of applications. A substantial body of experimental and theoretical work has been undertaken to determine the atmospheric chemistry and environmental impact of these compounds. While some minor uncertainties exist, our current understanding of the atmospheric chemistry of the commercially important HFCs and HCFCs is now well established (Wallington et al., 1994; 1995). A generalized scheme for the atmospheric oxidation of a haloalkane that could degrade to give trifluoroacetic acid is given in Figure 6.4.

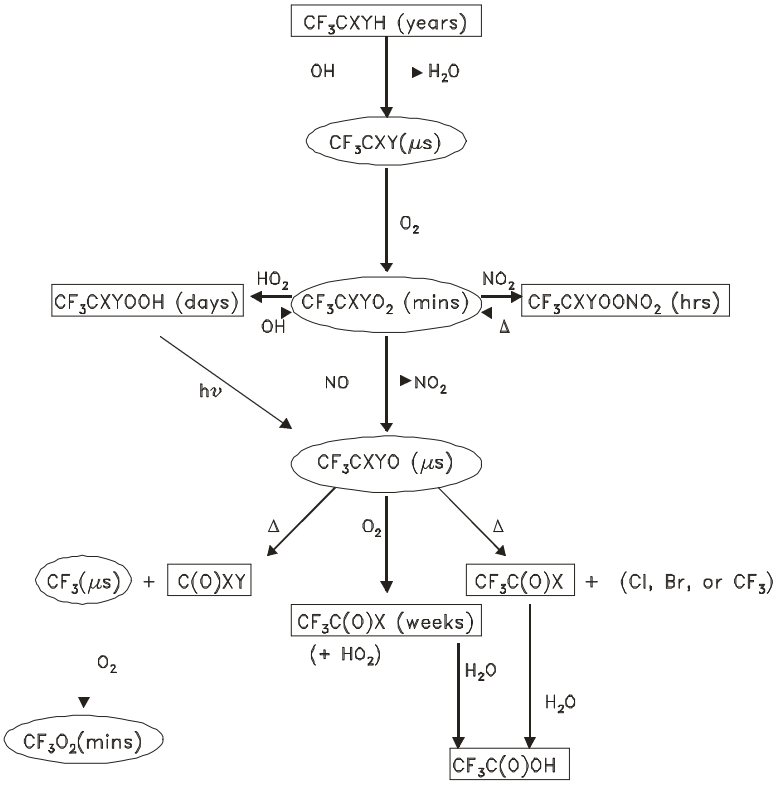

Fig. 6.4. Generalized scheme for the atmospheric oxidation of a halogenated organic compound, CF_3CXYH (X = Cl or F, and Y = Cl, H, Br, or CF_3). Radical intermediates are enclosed in ellipses. Typical lifetime estimates are given in parenthesis.

Oxidation is initiated by reaction with OH radicals giving a halogenated alkyl radical which adds O_2 to give the corresponding peroxy radical (RO_2). Peroxy radicals react with three important trace species in the atmosphere: NO, NO_2, and HO_2 radicals. Reactions with HO_2 and NO_2 delay, but do not prevent, the conversion of peroxy (CF_3CXYO_2) into alkoxy (CF_3CXYO) radicals. Reactions of haloperoxy radicals with NO are rapid and give the alkoxy radical with essentially 100% yield. The at-

mospheric fate of the alkoxy radical, CF_3CXYO, is either decomposition or reaction with O_2. Decomposition occurs by C-C bond fission, or by the elimination of a Br, Cl, or CF_3 group. The atmospheric fate of $CF_3C(O)X$ (X=F or Cl) is dominated by incorporation into rain-cloud-sea water followed by rapid hydrolysis to trifluoroacetic acid. Photolysis is a competing loss mechanism for $CF_3C(O)Cl$ and limits its conversion into $CF_3C(O)OH$ to 60% (Cox et al., 1995). There are no competing loss processes for $CF_3C(O)F$; it is converted entirely into $CF_3C(O)OH$. Although $CF_3C(O)OH$ is produced in aqueous phase chemistry, is highly soluble and partitions into the water phase (Bowden et al., 1996), the evaporation of cloud droplets can transfer $CF_3C(O)OH$ to the gas phase where it can react with OH radicals. However, this reaction is slow (Carr et al., 1994; Møgelberg et al., 1994) and is only a minor (<5%, Kanakidou et al., 1995) loss of $CF_3C(O)OH$. The main atmospheric fate of $CF_3C(O)OH$ is rain-out to the surface.

To assess the potential for a halocarbon to produce trifluoroacetic acid, it is necessary to quantify the yield of trifluoroacetyl halide in its gas phase oxidation mechanism. This has been established for all the commercially significant halocarbons. Six compounds, listed in Table 6.2, have been identified that degrade to give trifluoroacetyl halide. With the exception of HFC-134a, the atmospheric oxidation mechanisms of the compounds given in Table 6.2 are relatively simple. Atmospheric oxidation of halothane (Bilde et al., 1998), isoflurane, and HCFC-123 (Edney et al., 1991; Tuazon and Atkinson, 1993a; Hayman et al., 1994) gives $CF_3C(O)Cl$ of which approximately 60% undergoes hydrolysis to give $CF_3C(O)OH$. Oxidation of HCFC-124 (Tuazon and Atkinson, 1993a; Edney and Driscoll, 1992) and HFC-227ea (Zellner et al., 1994; Møgelberg et al., 1996) gives $CF_3C(O)F$ which is then converted into $CF_3C(O)OH$. Current and projected use of HFC-227ea is minor. The oxidation mechanism of HFC-134a is complicated by two factors. First, vibrationally excited CF_3CFHO^* radicals are formed in the $CF_3CFHO_2 + NO$ reaction and two thirds of these vibrationally excited radicals undergo decomposition via C-C bond scission on a time scale short compared to that needed for chemical reactions (Wallington et al., 1996). Second, reaction with O_2 competes with decomposition via C-C bond scission for the available thermalized CF_3CFHO radicals (Wallington et al., 1992; Tuazon and Atkinson, 1993b; Rattigan et al., 1994). The net effect is that the molar yield of $CF_3C(O)F$, and hence $CF_3C(O)OH$, is approximately 0.13 (Wallington et al., 1996).

An estimate for the concentration of $CF_3C(O)OH$ in rain water can be obtained as follows. Halothane and isoflurane have been used as anesthetics for many years. It is estimated that the current emissions of halothane and isoflurane are 1500 and 750 tonnes/yr, respectively (Boutonnet et al., 1998). The atmospheric lifetimes of halothane and isoflurane are short (see Table 6.2) compared to the time scale over which they have been emitted into the atmosphere. Assuming that these compounds are in steady state then their degradation gives a combined yield of 1540 tonnes/year of $CF_3C(O)Cl$. Accounting for photolysis, this results in a global deposition rate of 800 tonnes/year of $CF_3C(O)OH$. Current atmospheric concentrations of HCFC-123 and HCFC-124 are at, or below the detection limit of 0.1 ppt (Boutonnet et al., 1998). Thus, upper limits for atmospheric burdens of these gases are 2500 tonnes for HCFC-123 and 2300 tonnes for HCFC-124. Combining these burdens with the lifetime values given in Table 6.2 and accounting for photolysis of $CF_3C(O)Cl$ it can be concluded that upper limits for the flux of $CF_3C(O)OH$ from HCFC-123 and HCFC-124 are 760 and 320 tonnes/year, respectively. HFC-134a is present at a concentration of 2.5 pptv in the Northern Hemisphere and 1.2 pptv in the Southern Hemisphere (Montzka et al., 1996; Oram et al., 1996) from which it can be estimated that the present atmospheric burden of HFC-134a is 31,000 tonnes. Combining this burden with the lifetime and yield values in Table 6.2 gives a $CF_3C(O)OH$ flux of 300 tonnes/year from the atmospheric oxidation of HFC-134a. Combining the contributions from these known sources gives an upper limit for the estimated contemporary TFA formation rate of 800 + 760 + 320 + 300 = 2180 tonnes/year. The known TFA precursors are relatively long lived and therefore distributed on a global

scale. The annual global rainfall is 4.9×10^{17} liters (Erchel, 1975) and the global average TFA concentration in rainwater is expected to be less than 5 ng/l. Detailed computer modeling studies using appropriate global OH fields, halocarbon emissions, and rainfall patterns have confirmed that the simple approach adopted above provides a reasonable estimate of expected TFA concentrations in rainwater (Rodriguez et al., 1993; Kanakidou et al., 1995; Wild et al., 1996).

To assess the future contribution of HFCs and HCFCs to levels of TFA in rainwater we need to consider future emission scenarios for these compounds which are reviewed in detail elsewhere (Boutonnet et al., 1998). Use of halothane and isoflurane anesthetics is anticipated to remain constant, or perhaps decline. Production of HCFC-123 and HCFC-124 is regulated under the Montreal Protocol and will be phased out. Production of HFC-134a is not regulated and anticipated to increase significantly. Using an emission scenario from the US Environmental Protection Agency (EPA) combined with a three-dimensional atmospheric model, Rodriguez et al. (1994) calculated that in the year 2020 the atmospheric decomposition fluxes of HFC-134a, HCFC-123, and HCFC-124 will be 115, 20, and 20 kTonnes/yr. Multiplying by the appropriate $CF_3C(O)OH$ molar yields in Table 6.2 and making the necessary adjustments for the molecular weights involved, a flux of 16.7 + 8.9 + 16.7 = 42.3 kTonnes/yr is estimated and translates to a global average concentration in rainwater of 86 ng/l. Kotamarti et al. (1998) estimated the global average TFA concentration in rain water for 2010 to be in the range 100-160 ng/l.

Table 6.2: Compounds known to produce TFA ($CF_3C(O)OH$) in the atmosphere.

Compound	Molecular weight	Common name	Molar $CF_3C(O)OH$ yield	Atmospheric lifetime
$CF_3CHClBr$	197.5	Halothane	0.6	1.2 years [1]
$CF_3CHClOCHF_2$	184.5	Isoflurane	0.6	5 years [2]
CF_3CHCl_2	153	HCFC-123	0.6	1.5 years [3]
CF_3CHFCl	136.5	HCFC-124	1.0	6.0 years [3]
CF_3CH_2F	102	HFC-134a	0.13	14.6 years [4]
CF_3CHFCF_3	170	HFC-227ea	1.0	36.5 years [4]

[1] Orkin and Khamagonov (1993); [2] Brown et al. (1989); [3] WMO (1989); [4] IPCC (1996).

Water bodies characterized by little or no outflow and high evaporation rates may have the potential to accumulate TFA. Tromp et al. (1995) developed a concentration-dependent model and estimated that TFA concentrations of 100 μg/l could be achieved in this type of water body in as few as 30 years, with rainfall concentration only 1 μg/l. In contrast, a recent analysis by Boutonnet et al. (1998) questioned the validity of the assumptions inherent in the Tromp et al. study, concluding that accumulation of TFA in seasonal wetlands "appears to be highly improbable". The potential for accumulation of TFA remains unclear.

High concentrations of TFA have been observed in contemporary water and air samples, suggesting the existence of one or more large unknown sources. Samples of rain and surface waters (oceans, rivers, lakes, and springs) have been obtained from many geographical areas (USA, Canada, Australia, South Africa, Germany, Israel, Ireland, France, Switzerland, Finland) and show that TFA is a

ubiquitous contaminant of the hydrosphere (Frank et al., 1996; Zehavi and Seiber, 1996; Grimvall et al., 1997; Wujcik et al., 1998), with values up to 40900 ng/l (Zehavi and Seiber, 1996). The average TFA concentration in rain water observed in Bayreuth during 1995 was 100 ng/l (Frank et al., 1996). The source of the currently observed levels is unknown and puzzling. The observed TFA concentrations are orders of magnitude larger than those predicted to result from the atmospheric degradation of the replacement HCFCs and HFCs.

TFA in the Biosphere

Environmental Distribution

Trifluoroacetic acid, $CF_3C(O)OH$ or TFA, is a strong organic acid with pKa of 0.23 and it is miscible with water (solubility over 10000 g/l). The vapor pressure is relatively high, 105.7 hPa at 20°C, the partition coefficient n-octanol/water (log) is -0.2. Laboratory studies have been performed to assess the strength of binding of TFA to a variety of soil types (van Dijk, 1992; Richey et al., 1997). Some studies showed that TFA did not adsorb to soil, others showed that TFA generally interacted weakly with most soils but was strongly adsorbed by some soils containing high levels of organic matter. The data are not necessarily contradictory but may reflect the heterogeneity of soils tested.

Degradation

TFA is a stable ion in the aqueous phase and no significant loss process such as hydrolysis, photolysis, or formation of insoluble salts has been identified. There have been two reports of TFA degradation under anaerobic conditions. In the first study, natural sediments reduced TFA (Visscher et al., 1994). However, even though this work was done in replicate, the experimenters and others were unable to reproduce it in subsequent studies (Matheson et al., 1996; Emptage et al., 1997). In the second study (Chauhan et al., 1995), labelled TFA was removed from a mixed anaerobic in vitro microcosm. Limited evidence of decarboxylation has also been reported for two strains of bacteria grown under highly specific conditions (Chauhan et al., 1995**).** A field study indicated that TFA was retained in vegetation and soil of a temperate North American forest, especially in the case of wetlands with organic soils (Richey et al., 1997; Likens et al., 1997).

Bioaccumulation in Animals and Plants

Potential for bioaccumulation in animals is highly unlikely due to the extremely low log K_{ow} (-0.2). Due to its high solubility TFA can accumulate in plants via roots uptake of water. This phenomenon is supported by experimental data which demonstrate that TFA is a xylem mobile herbicide, transported through the stem and accumulated in leaves (Rollins et al., 1989). The bioaccumulation in vegetation calculated according the following equation: log CF= 5.943 – 2.385 log MW gives a value of 10.89 as concentration factor (CF), of the same order of magnitude of those experimentally observed.

TFA is not concentrated in lower aquatic-life forms such as bacteria, small invertebrates, oligochaete worms and some aquatic plants including *Lemna gibba* (duckweed). In terrestrial higher plants such as sunflower and wheat some bioaccumulation was seen (Thompson et al., 1994). This appeared to be related to uptake with water and then concentration due to transpiration water loss. When transferred to clean hydroponic media, some elimination of TFA was seen. Also, over 80% of the TFA in

leaves was found to be water extractable, suggesting that no significant metabolism of TFA had occurred.

Ecotoxicology

Effect of TFA on Activated Sludge: The semi-continuous activated sludge test indicated that TFA had no discernible effect on the performance of the sludge for the biodegradation of organic carbon (van Ginkel and Kroon, 1992).

Effect of TFA on metabolism of microbial communities: The mineralization of acetate to carbon dioxide is a key link in the biogeochemical carbon cycle. Therefore it is essential to know whether TFA, which is structurally close to acetate, could interfere with acetate metabolism. Experimental results (Bott and Standley, 1998) suggest that TFA at concentrations several orders of magnitude higher than those anticipated in the environment did not impact acetate mineralization to carbon dioxide nor did it affect incorporation of acetate into cellular material. The effect of TFA has been investigated in free-living nitrogen-fixing bacteria (Nagel and Odom, 1997). The experiments were designed to determine whether TFA was specifically toxic to nitrogen fixation. No effect of TFA on growth or N_2 fixation with ammonium ion as nitrogen source was noted at concentrations as high as 100 ppm TFA. It has been shown that TFA has no effect on methanogenic environments where acetate is an important intermediate (Emptage et al., 1997).

Aquatic toxicity: In acute toxicity tests no effects of NaTFA on water fleas (*Daphnia magna*) and zebra fish (*Brachydanio rerio*) were found at a concentration of 1200 mg/l (Rhone-Poulenc, 1995). A 7-day study with duckweed (*Lemna gibba*) revealed a Predicted No Effect Concentration (NOEC) of 300 mg/l (Smyth et al., 1993). Based on the results of five toxicity tests with *Selenastrum capricornutum* a NOEC of 0.12 mg/l was found. However, algal toxicity tests with NaTFA and *Chlorella vulgaris, Scenedesmus subspicatus, Chlamydomonas reinhardtii, Dunaliella tertiolecta, Euglena gracilis, Phaeodactylum tricornutum, Navicula pelliculosa, Skeletonema costatum, Anabaena flos-aquae and Microcystis aeruginosa* resulted in NOEC values which were all higher than 100 mg/l (Smyth et al., 1994a,b,c). Recovery of the growth of *S. capricornutum* was found when TFA was removed from the test solutions and therefore TFA should be considered algistatic and not algicidal for *S. capricornutum*. The reason for the unique sensitivity of this strain is unknown, but a recovery of the growth rate was seen when citric acid was added, suggesting a competitive inhibition of the citric acid cycle.

One semi-field study (Bott and Standley, 1995) with mesocosm streams has been conducted with NaTFA to study the potential effects of TFA on freshwater algal communities and primary productivity. Long-term exposure to a mean NaTFA concentration of 31-32 µg/l had no effect on the primary productivity of the diatom-dominated algal flora. Effects on organic carbon excretion which were related to high levels of TFA were noted in some experiments. TFA did not alter the algal species composition in the stream mesocosm.

Terrestrial plants: Application of NaTFA at 1000 mg/l to seeds of sunflower, cabbage, lettuce, tomato, mungbean, soybean, wheat, corn oats and rice did not affect germination (Thompson and Windeatt, 1994; Emerich, 1997). Foliar application of a solution of 100 mg/l of NaTFA to field grown plants did not affect growth of sunflower, soya, wheat, maize, oilseed rape, rice and plantain (Davison and Pearson, 1997). When plantain, wheat and soya were grown in hydroponic systems containing NaTFA, no effects were seen on plantain at 32 mg/l, on Triticum and soya at 1 mg/l, and on wheat at 10 mg/l (Thompson, 1995; Thompson et al., 1995; Davison and Pearson, 1997).

Mammalian Toxicity

TFA is not metabolised in mammalian systems. The half-life of TFA in humans is 16 hours. As expected, the free acid is more acutely toxic than the sodium salt. In one study, 2 of 5 mice died from a dose of 150 mg/kg of HTFA, an effect comparable to that seen with an equimolar dose of HCl (Blake et al., 1969). In contrast, no deaths were seen when mice were given an oral dose of 5.000 g/kg of NaTFA. In studies involving single intraperitoneal injections of doses up to 2 g/kg, only mild liver toxicity was seen (Rosenberg, 1971; Rosenberg and Wahlstrom, 1971). There was only one report of an acute inhalation toxicity study (Kheilo and Kremneva, 1966). This study involved single two-hour exposures of both rats and mice. The LC 50 for mice was 13.5 mg/l (2,900 ppm) and for rats it was 10 mg/l (2,140 ppm). This would classify TFA as having low inhalation toxicity. These same authors reported that the irritation threshold for humans was 54 ppm.

As one would expect of a strong acid, HTFA is a severe irritant to the skin (Patty, 1963; Kheilo and Kremneva, 1966). Concentrations as low as 2% were moderate skin irritants. It would be expected to be a severe eye irritant. When conjugated with protein, it has been shown to elicit an immunological reaction (Mathieu et al., 1974; Reves and McCracken, 1976; Ford et al., 1984; Satoh et al., 1985;1989), however it is unlikely that TFA itself would elicit a sensitisation response (Waldon and Ratra, 1972; Reves and McCracken, 1976; Ford et al., 1984). Repeated administrations of aqueous solutions have shown that TFA can cause increased liver weight and induction of peroxisomes (Just et al., 1989; Warheit, 1993). Relative to the doses (0.5% in diet or 150 mg/kg/day) the effects are mild.

In a series of Ames assays, TFA was reported to be non-mutagenetic (Blake et al., 1981). Its carcinogenic potential has not been evaluated. Although TFA was shown to accumulate in amniotic fluid following exposure of pregnant animals to high levels of halothane (1,200 ppm), no foetal effects were seen. Given the high levels of halothane exposure, it is unlikely that environmental TFA is a reproductive or developmental hazard.

TFA Risk Assessment

The environmental risk from TFA as degradation product of CFCs substitutes can be deduced from the value of the ratio of exposure to effect, or of Predicted Environmental Concentrations to Predicted No Effect Concentrations (PEC/PNEC). To derive a PEC, production and releases of parent compounds, along with rates of transformation into TFA, have been modeled. A range of concentrations in rain water has been calculated, taking into account geographical variations (OH radical concentrations, amount of rain, regional releases of parent compound). A rough average concentration of 0.1 µg/l in rainwater, by the year 2020, is taken as a global PEC. But an important question remains concerning the origin of the large current levels of TFA which have been measured in the environment (fresh and marine surface waters, rain and air) and which cannot be explained by the known industrial sources.

The physico-chemical properties of TFA allow a prediction of no bioaccumulation in animals. In terrestrial plants, accumulation can take place but is transient and effects on vegetation can be observed only in experiments using non environmentally relevant concentrations (102-106 mg/l).

TFA is not retained in soils, with the exception of those having high organic content; in this case, it is uncertain if it can still be bioavailable. Generally speaking, TFA remains associated with water and has been shown to be not effective on several types of metabolism related to basic biogeochemical cycles.

Given the fact that deposited TFA would remain in water, a number of studies have been done aiming to derive a PNEC in the aquatic compartment. Standard acute tests on fish and daphnia carried out with NaTFA show no effects on animals at large concentrations (up to 1 g/l TFA). This is rein-

forced by toxicological data on mammals. On the other hand, the NOEC for the standard algal species *Selenastrum capricornutum* is around 0.10 mg/l (as TFA). To see if this sensitivity was general among algae, 10 other species belonging to 4 different classes have been tested: no one was sensitive to NaTFA (NOEC> 100 mg/l). It is proposed to use as a $PNEC_{aqua}$ (the NOEC of this species of algae) as a worst case, without any further safety factor. So, $PNEC_{aqua}$ =0.1 mg/l.

A large amount of research has been devoted to effects on higher plants, as they could be exposed to TFA in rain water through leaves and stems and TFA in pore water through roots. A few tested species (selected among those having an important role in feeding people and cattle) show a significant sensitivity to TFA (as NaTFA). The threshold for effects on wheat and soya is around 5 mg/l with a NOEC=1 mg/l. Other studies show that nitrogen fixation is not affected up to 1 mg/kg soil. Therefore, a conservative figure could be $PNEC_{soil}$ = 0.1 mg/l.

No significant risk is anticipated from TFA produced by atmospheric degradation of the present and future production of HCFCs and HFCs, as there is a 1000-fold difference between the PNEC and the PECs.

References

Anklin, M. and Bales, R.C. 1997. Recent increase in H_2O_2 concentration at Summit, Greenland. *Journal of Geophysical Research* 102:19099-19104.

Bekki, S., Law, K.S. and Pyle, J. 1994. Effects of ozone depletion of atmospheric CH_4 and CO concentrations. *Nature* 371:595-597.

Bilde, M., Wallington, T.J., Ferronato, C., Orlando, J.J., Tyndall, G.S., Estupinan, E. and Haberkorn, S. 1998. Atmospheric Chemistry of CH_2BrCl, $CHBrCl_2$, $CHBr_2Cl$, $CF_3CHBrCl$ and CBr_2Cl_2. *Journal of Physical Chemistry A* 102:1976-1986.

Blake, D.A., Cascorbi, H.F., Rozman, R.S. and Meyer, F.J. 1969. Animal toxicity of 2,2,2-trifluoroethanol. *Toxicology and Applied Pharmacology* 15:83-91.

Blake, D.A., DiBlasi, M.C. and Gordon, G.B. 1981. Absence of mutagenic activity of trifluoroethanol and its metabolites in *Salmonella typhimurium*. *Fundamental and Applied Toxicology* 1:415-418.

Boutonnet, J-C., Bingham, P., Calamari, D., de Rooij, C., Franklin, J., Kawano, T., Libre, J.-M., McCulloch, A., Malinverno, G., Odom, J.M., Rusch, G.M., Smythe, K. and Sobolev, I. 1998. Human and Ecological Risk Assessment, in press.

Bott, T.L. and Standley, L.J. 1995. *The Interaction of trifluoroacetate with microbial communities.* Abstract for platform presentation for the annual meeting for the American Society for Microbiology, Washington, DC, USA.

Bott, T.L. and Standley, L.J. 1998. Trifluoroacetate, an atmospheric breakdown product of hydrofluorocarbon refrigerants, and freshwater benthic microbial communities: incorporation and effects on acetate metabolism. *Environmental Science and Technology* 32:469-475.

Bowden, D.J., Clegg, S.L. and Brimblecombe, P. 1996. The Henry's law constant of trifluoroacetic acid and its partitioning into liquid water in the atmosphere. *Chemosphere* 32:405-420.

Brown, A.C., Canosa-Mas, C.E., Parr, A.D., Pierce, J.M.T. and Wayne, R.P. 1989. Tropospheric lifetimes of halogenated anaesthetics. *Nature* 341:635-637.

Brunke, E., Scheel, H. and Seiler, W. 1990. Trends in tropospheric CO, N_2O, and CH_4 observed at Cape Point, South Africa. *Atmospheric Environment* 24A:585-595.

Carr, S., Treacy, J.J., Sidebottom, H.W., Connell, R.K., Canosa-Mas, C.E., Wayne, R.P. and Franklin, J. 1994. Kinetics and mechanisms for the reaction of hydroxyl radicals with trifluoroacetic acid under atmospheric conditions. *Chemical Physics Letters* 227:39-44.

Cox, R.A., Rattigan, O., Hayman, G.D., Howard, C.J., Nielsen, O.J., Sidebottom, H.W. and Zellner, R. 1995. *Atmospheric Degradation Mechanisms - Summary of Discussions*, Chapter 2 of NASA/NOAA/AFEAS Workshop on HFC/HCFC Atmospheric Chemistry, Boulder, Colorado.

Chauhan, S., Criddle, C. and Tiedje, J. 1995. *Potential for microbial transformation of trifluoroacetic acid.* Final Report to AFEAS SP91-18.11/BP93-15.

Davison, A.W. and Pearson, S. 1997. *Toxicity of TFA to plants.* AFEAS SP91-1823/ BP96-31 University of Newcastle Report.

De Leeuw, F.A.A. and Van Rheineck Leyssius, H.J. 1991. Sensitivity of oxidant concentrations on changes in U.V. radiation and temperature. *Atmospheric Environment* 25A:1025-1032.

Dlugokencky, E.J., Steele, L.P., Lang, P.M. and Masarie, K.A. 1994. The growth rate and distribution of atmospheric methane. *Journal of Geophysical Research* 99:17021-17043.

Dlugokencky, E.J., Dutton, E.G., Novelli, P.C., Tans, P.P., Masarie, K.A., Lantz, K.O. and Madronich, S. 1996. Changes in CH_4 and CO growth rates after the eruption of Mt. Pinatubo and their link with changes in tropical tropospheric UV flux. *Geophysical Research Letters* 23:2761-2764.

Edney, E.O. and Driscoll, D.J. 1992. Chlorine initiated photooxidation studies of hydrochlorofluorocarbons (HCFCs) and hydrofluorocarbons (HFCs): results for HCFC-22 ($CHClF_2$); HDC-41 (CH_3F); HCFC-124 ($CClFHCF_3$); HFC-125 (CF_3CHF_2); HFC-134a (CF_3CH_2F); HCFC-142b ($CClF_2CH_3$); and HFC-152a (CHF_2CH_3). *International Journal of Chemical Kinetics* 24:1067-1081.

Edney, E.O., Gay, B.W.Jr. and Driscoll, D.J. 1991. Chlorine initiated oxidation studies of hydrochlorofluorocarbons: results for HCFC-123 (CF_3CHCl_2) and HCFC-141b ($CFCl_2CH_3$). *Journal of Atmospheric Chemistry* 12:105-120.

Emerich, D. 1997. *Effects of TFA on symbiotic nitrogen fixation by soybean*. Final Report to AFEAS SP91-18.24/BP95-32 University of Missouri.

Emptage, M., Tabinowski, J.A. and Odom, J.M. 1997. The effect of fluoroacetates on methanogenesis in samples from selected methanogenic environments. *Environmental Science and Technology* 31:732-734.

Erchel, E. 1975. *World water balance*. Elsevier, New York.

Etheridge, D.M., Steele, L.P., Francey, R.J. and Langenfelds, R.L. 1998. Atmospheric methane between 1000A.D. and present: evidence of anthropogenic emissions and climatic variability. *Journal of Geophysical Research* 103:15979-15993.

Finlayson-Pitts, B.J. and Pitts, J.N.Jr. 1986. *Atmospheric Chemistry*. Wiley-Interscience, New York pp.1098.

Ford, D.J., Coyle, D.E. and Harrington, J.F. 1984. Effects of hypersensitivity to a halothane metabolite on halothane-induced liver damage. *Anesthesiology* 60(2):141-143.

Frank, H., Klein, A. and Renschen, D. 1996. Environmental trifluoroacetate. *Nature* 382:34.

Fuglestvedt, J.S., Jonson, J.E. and Isaksen, I.S.A. 1994. Effects of reductions in stratospheric ozone on tropospheric chemistry through changes in photolysis rates. *Tellus* 46B:172-192.

Fuglestvedt, J.S., Jonson, J.E., Wang, W.C. and Isaksen, I.S.A. 1995. Responses in tropospheric chemistry to changes in UV fluxes, temperatures and water vapor densities. *In Atmospheric Ozone as a Climate Gas*, Wang, W.C. and Isaksen, I.S.A. (eds.), NATO ASI Series, Vol. I 32, Springer-Verlag, Berlin, pp.145-163.

Gery, M.W., Edmond, R.D. and Whitten G.Z. 1987. *Tropospheric ultraviolet radiation*. U.S. Environmental Protection Agency report EPA/600/3-87/097, Washington D.C.

Graedel, T.E. and Crutzen, P.J. 1993. *Atmospheric Change*. Freeman and Co., New York.

Granier, C., Müller, J.-F., Madronich, S. and Brasseur, G.P. 1996. Possible causes for the 1991-1993 decrease in the global tropospheric CO abundance: a three-dimensional study. *Atmospheric Environment* 30:1673-1682.

Granier, C., Shine, K., Hansen, J., Lal, S. and Stordal, F. 1998. Climate effects of ozone and halocarbon changes. In *Scientific Assessment of Ozone Depletion: 1998. Global Ozone Research and Monitoring Project World Meteorological Organization*, Albritton, D.L, Aucamp, P.J., Megie, G. and Watson, R.T. (eds.), Geneva, in press.

Grimvall A., Borén, H., von Sydow, L. and Laniewski, K. 1997. *Analysis of TFA in Samples of Rain, Snow and Ice Collected at Remote Sites*, presentation to AFEAS.

Hayman, G.D., Jenkin, M.E., Murrells, T.P. and Johnson, C.E. 1994. Tropospheric degradation chemistry of HCHC-123 (CF3CHCl2): a proposed replacement chlorofluorocarbon. Atmospheric Environment 28:421-437.

IPCC (Intergovernmental Panel on Climate Change) 1996. Climate change 1995: The science of climate change, contribution of Working Group 1 to the Second Assessment Report of the IPCC, J.T. Houghton, L.G. Meira Filho, B.A. Callander, N. Harris, A. Kattenberg and K. Maskell (eds.), The University Press, Cambridge, UK.

Kanakidou, M., Dentener, F.J. and Crutzen, P.J. 1995. A global three-dimensional study of the fate of HCFCs and HFC-134a in the troposphere. *Journal of Geophysical Research* 100:18781-18801.

Just, W.W., Gorgas, K., Hartl, F.U., Heinemann, P., Salzer, M. and Schimassek, H. 1989. Biochemical effects of zonal heterogeneity of peroxisome proliferation induced by perfluorocarboxylic acids in rat liver. *Hepatology (Baltimore)* 9(4):570-581.

Kheilo, L.S. and Kremneva, S.N. 1966. Comparative assessment of the toxicity of trifluoroacetic acid and pentafluoropropionic acids. *Gigiena Truda I Professionalnye Zabolevaniia* 10(3):13-17.

Kotamarthi, V.R., Rodriguez, J.M., Ko, M.K.W., Sze, N.D. and Prather, M.J. 1998. Production of trifluoroacetic acid from HCFCs and HFCs: a three-dimensional modeling study. *Journal of Geophysical Research* 103:5745-5758.

Krol, M.C. and van Weele, M. 1997. Implications of variations in photodissociation rates for global tropospheric chemistry. *Atmospheric Environment* 31:1257-1273.

Krol, M., van Leeuwen, P.J. and Lelieveld, J. 1998. Global OH trend inferred from methylchloroform measurements. *Journal of Geophysical Research* 103:10697-10711.

Likens, G.E., Tartowski, S.L., Berger, T.W., Richey, D.G., Driscoll, C.T., Frank, H.G. and Klein, A. 1997. *Transport and fate of trifluoroacetate in upland forest and wetland ecosystems*. Proceedings of the National Academy of Sciences of the United States of America, 94(N9):4499-4503.

Liu, S.C. and Tranier, M. 1988. Response of the tropospheric ozone and odd hydrogen radicals to column ozone change. *Journal of Atmospheric Chemistry* 6:221-233.

Lowe, D.C., Manning, M.R., Brailsfort, G.W. and Bromley, A.M. 1997. The 1991-1992 atmospheric methane anomaly: southern hemisphere ^{13}C decrease and growth rate fluctuations. *Geophysical Research Letters* 24:857-860.

Ma, J. 1996. *Effects of clouds, stratospheric ozone depletion and tropospheric pollution on the chemical budget of ozone in the troposphere through changes in photolysis rates*, TNO Institute of Environmental Sciences Report R 96/406, The Netherlands..

Madronich, S. and Granier, C. 1992. Impact of recent total ozone changes on tropospheric ozone photodissociation, hydroxyl radicals, and methane trends. *Geophysical Research Letters* 19:465-467.

Madronich, S. and Granier, C. 1994. Tropospheric chemistry changes due to increases in UV-B radiation. In *Stratospheric Ozone Depletion/UV-B Radiation in the Biosphere*, Biggs, H.R. and Joyner, M.E.B. (eds.), NATO ASI vol. I18, Springer-Verlag, Berlin, pp. 3-10.

Madronich, S., McKenzie, R.L., Björn, L.O. and Caldwell, M.M. 1995. Changes in ultraviolet radiation reaching the Earth's surface. *Ambio* 24:143-152.

Madronich, S., McKenzie, R.L., Björn, L.O. and Caldwell, M.M. 1998. Chapter 1: Changes in ultraviolet radiation reaching the Earth's surface. *In Environmental Effects of Ozone Depletion: 1998 Update*, van der Leun, J.C., Tevini, M., X. Tang, and Worrest, R.C. (eds.), United Nations Environment Programme, Nairobi, this report.

Matheson, L.J., Guidetti, J.R., Visscher, P.T., Schaefer, J.K. and Oremland, R.S. 1996. *Summary of research results on bacterial degradation of trifluoroacetate (TFA). October 1993-October 1995.* USGS Open file Report 96-219. Prepared in cooperation with the Alternative Fluorocarbons Environmental Assessment Study (AFEAS). US Geological survey, Information Services, Box 25286, Mail Stop 417, Denver Federal Center, Denver CO 80225-0046.

Mathieu, A., DiPadua, D., Mills, J. and Kahan B. 1974. Experimental immunity to a metabolite of halothane and fluroxene. *Anaesthesiology* 40(4):385-390.

Møgelberg, T.E., Nielsen, O.J., Sehested, J., Wallington, T.J. and Hurley, M.D. 1994. Atmospheric chemistry of CF_3COOH: kinetics of the reaction with OH radicals. *Chemical Physics Letters* 226:171-177.

Møgelberg, T.E., Sehested, J., Bilde, M., Wallington, T.J. and Nielsen, O.J. 1996. Atmospheric chemistry of HFC-227ea: spectrokinetic investigation of the $CF_3CFO_2(\cdot)CF_3$ radical, its reactions with NO and NO_2 and the atmospheric fate of the $CF_3CFO(\cdot)CF_3$ radical. *Journal of Physical Chemistry* 100:8882-8889.

Montzka, S.A., Myers, R.C., Butler, J.H., Elkins, J.W., Lock, L.T. and Clarke, A.D. 1996. Observations of HFC-134a in the remote troposphere. *Geophysical Research Letters* 23:169-172.

Nagel E. and Odom, J.M. 1997. *The effect of TFA on growth and nitrogen fixation by nitrogen fixing bacteria.* DuPont Report submitted to AFEAS.

Neftel, A., Bales, R.C. and Jacob, D.J. 1995. *H_2O_2 and HCHO in polar snow and their relation to atmospheric chemistry.* NATO ASI Series, Vol. I30, Springer-Verlag, Berlin, pp.249-264.

Novelli, P.C., Masarie, K.A. and Lang, P.M. 1994. Recent changes in atmospheric carbon monoxide. *Science* 263:1587-1580.

Oram, D.E., Reeves, C.E., Sturges, W.T., Penkett, S.A., Fraser, P.J. and Langenfelds, R.L. 1996. Recent tropospheric growth rate and distribution of HFC-134a (CF_3CH_2F). *Geophysical Research Letters* 23:1949-1952.

Orkin, V.L. and Khamaganov, V.G. 1993. Rate constants for reactions of OH radicals with some Br-containing haloalkanes. *Journal of Atmospheric Chemistry* 16:169-178.

Patty, F.A. 1963. *Industrial Hygiene and Toxicology*, Vol. II, 2nd revised edit., pp. 1801-1803.

Prinn, R.G., Cunnold, D.M., Simmonds, P.G., Alyea, F.N., Boldi, R., Crawford, A.J., Fraser, P.J., Gutzler, D., Hartley, D.E., Rosen, R.D. and Rasmussen, R. 1992. Global average concentration and trend for hydroxyl radicals deduced from ALE/GAGE trichloroethane (methyl chloroform) data for 1978-1990. *Journal of Geophysical Research* 97:2445-2461.

Prinn, R.G., Weiss, R.F., Miller, B.R., Huang, J., Alyea, F.N., Cunnold, D.M., Fraser, P.J., Hartley, D.E. and Simmonds, P.G. 1995. Atmospheric trends and lifetime of CH_3CCl_3 and global OH concentrations. *Science* 269:187-192.

Rattigan, O.V., Rowley, D.M., Wild, O., Jones, R.L. and Cox, R.A. 1994. Mechanism of atmospheric oxidation of 1,1,1,2-tetrafluoroethane (HFC 134a). *Journal of the Chemical Society, Faraday Transactions* 90:1819-1829.

Reves, J.G. and McCracken, L.E. 1976. Hepatic pathology and skin test reactions to trifluoroacetylated autologous protein after repeated halothane anesthesia in the guinea pig. *British Journal of Anaesthesia* 48(5):419-424.

Rhône-Poulenc 1995. *Material Safety Data Sheet*, v. 6 Dec. 95.

Richey, D.G., Driscoll, C.T. and Likens, G.E. 1997. Soil retention of trifluoroacetate. *Environmental Science and Technology* 31:1723-1727.

Rodriguez, J.M., Ko, M.K.W., Sze, N.D. and Heisey, C.W. 1993. *Proceedings of the STEP-HALOCSIDE/AFEAS Workshop*, University College Dublin, Ireland, p. 104.

Rodriguez, J.M., Ko, M.K.W., Sze, N.D., Heisey, C.W., Tromp, T.K. and Sze, N.D. 1994. *Modeling dosage projections for trifluoroacetic acid, Workshop on Decomposition of TFA in the environment, 8-9 Feb 1994*, Washington D.C., available from AFEAS, 1333 H Street NW, Washington D.C., 20005.

Rollins, A., Barber, J., Elliott, R. and Wood, B. 1989. Xenobiotic monitoring in Plants by 19F and 1H nuclear magnetic resonance imaging and spectroscopy: Uptake of trifluoroacetic acid in *Lycopersicum esculentum*. *Plant Physiology* 91:1243-1246.

Rosenberg, P.H. 1971. Decrease in reduced glutathione and NADPH and inhibition of glucose-6-phosphate dehydrogenase activity caused by metabolites of fluoroxene and halothane. *Ann. Med. Exp. Biol. Fenn.* 49(2):84-88.

Rosenberg, P.H. and Wahlstrom, T. 1971. Hepatotoxicity of halothane metabolites *in vivo* and inhibition of fibroblast growth *in vitro*. *Acta Pharmacologica et Toxico*logica 29(1): 9-19.

Satoh, H., Fukuda, Y., Anderson, D.K., Ferrans, V.J., Gillette J.R. and Pohl, L.R. 1985. Immunological studies on the mechanism of halothane-induced hepatotoxicity: immunohistochemical evidence of trifluoroacetylated hepatocytes. *Journal of Pharmacology and Experimental Therapeutics* 233(3): 857-862.

Satoh, H., Martin, B.M., Schulick, A.H., Christ, D.D., Kenna, J.G. and Pohl, L.R. 1989. Human anti-endoplasmic reticulum antibodies in sera of patients with halothane-induced hepatitis are directed against a trifluoroacetylated carboxylesterase. *Proceedings of the National Academy of Science* 86:322-326.

Schnell, R.C., Liu, S.C., Oltmans, S.J., Stone, R.S., Hoffman, D.J., Dutton, E.G., Deshler, T., Sturges, W.T., Harder, J.W., Sewell, S.D., Trainer, M. and Harris, J.M. 1991. Decrease of summer tropospheric ozone concentrations in Antarctica. *Nature* 351:726-729.

Shetter, R.E., Cantrell, C.A., Lantz, K.O., Flocke, S.J., Orlando, J.J., Tyndall, G.S., Gilpin, T.M., Madronich, S. and Calvert, J.G. 1996. Measurements and calculations of the photodissociation rate coefficient for $O_3 + h\nu \rightarrow O(^1D) + O_2$ during the MLOPEX II field campaign. *Journal of Geophysical Research* 101:14631-14641.

Sigg, A. and Neftel, A. 1991. Evidence for a 50 percent increase in H_2O_2 over the past 200 years from a Greenland ice core. *Nature* 351:557-559.

Smyth, D.V., Thompson, R.S. and Gillings, E. 1993. Sodium trifluoroacetate: Toxicity to the duckweed (*Lemna gibba*). *Brixham Environmental Laboratory Report* BL4872/B.

Smyth D.V., Thompson, R.S. and Gillings, E. 1994a Sodium trifluoroacetate: Toxicity to the freshwater diatom *Navicula pelliculosa*. *Brixham Environmental Laboratory Report* BL4968/B.

Smyth D.V., Thompson, R.S. and Gillings, E. 1994b Sodium trifluoroacetate: Toxicity to the marine alga *Skeletonema costatum*. *Brixham Environmental Laboratory Report* BL4980/B.

Smyth D.V., Thompson, R.S. and Gillings, E. 1994c Sodium trifluoroacetate: Toxicity to the blue-green alga *Anabaena flos-aquae*. *Brixham Environmental Laboratory Report* BL4967/B.

Taalas, P., Damski, J., Kyro, E., Ginzberg, M. and Talamoni, G. 1997. Effect of stratospheric ozone variations on UV radiation and on tropospheric ozone at high latitudes. *Journal of Geophysical Research* 102:1533-1539.

Thompson, A.M., Huntley, M.A. and R.W. Stewart. 1990. Perturbations to tropospheric oxidants. *Journal of Geophysical Re*search 95:9829-944.

Thompson, R.S. 1995. Sodium trifluoroacetate: Toxicity to Plantago major, by aqueous exposure of the roots. *Brixham Environmental Laboratory Report* BL5474/B.

Thompson, R.S. and Windeatt, A.J. 1994. Sodium trifluoroacetate: Effects on seed germination (ten species, by aqueous exposure). *Brixham Environmental Laboratory Report* BL5197/B.

Thompson R.S., Stewart, K.M. and Gillings, E. 1994. Trifluoroacetic acid: Accumulation from aqueous solution by the roots of Sunflower (*Helianthus annuus). Brixham Environmental Laboratory Report* BL5042/B.

Thompson R.S., Stewart, K.M. and Gillings, E. 1995. Sodium trifluoroacetate: Toxicity to wheat (Triticum aestivum) in relation to bioaccumulation (by aqueous exposure of the roots*). Brixham Environmental Laboratory Report* BL5473/B.

Tromp, T.K., Ko, M.K.W., Rodriguez, J.M. and Sze, N.D. 1995. Potential accumulation of a CFC-replacement degradation product in seasonal wetlands. *Nature* 376:327-330.

Tuazon, E.C. and Atkinson, R. 1993a. Tropospheric transformation products of a series of hydrofluorocarbons and hydrochlorofluorcarbons. *Journal of Atmospheric Chemistry* 17:179-199.

Tuazon, E.C. and Atkinson, R. 1993b. Tropospheric degradation products of CH_2FCF_3 (HFC-134a). *Journal of Atmospheric Chemistry* 16:301-312.

van Dijk, N.R.M. 1992. Adsorption of sodium trifluoroacetate (NaTFA) to three different soils. *Solvay-Duphar Report* 56635/91/91.

van Ginkel, C.G. and Kroon A.G.M.1992. *Biodegradability of trifluoroacetic acid sodium salt in a modified SCAS test. Report to AKZO Chemicals International.* AKZO Research Laboratories Arnhem Corporate Research, Analytical and Environmental Chemistry Department, Valperweg 76, 6824 BM Arnhem, The Netherlands.

Visscher, P.T., Culbertson, C.W. and Oremland, R.S. 1994. Degradation of trifluoroacetate in oxic and anoxic sediments. *Nature* 69:729-731.

Waldron, B.A. and Ratra, C.K. 1972. Response of lymphocytes cultured after halothane anesthesia to halothane and trifluoroacetic acid. *British Journal of Anaesthesiology* 44:1150-1154.

Wallington, T.J., Hurley, M.D., Ball, J.C. and Kaiser, E.W. 1992. Atmospheric chemistry of hydrofluorocarbon 134a; fate of the alkoxy radical CF_3CFHO. *Environmental Science and Technology* 26:1318-1324.

Wallington, T.J., Schneider, W.F., Worsnop, D.R., Nielsen, O.J., Sehested, J., DeBruyn, W. and Shorter, J.A. 1994. The environmental impact of CFC replacements – HFCs and HCFCs. *Environmental Science and Technology* 28:320A-326A.

Wallington, T.J., Schneider, W.F., Nielsen, O.J., Sehested, J., Worsnop, D.R., DeBruyn, W.J. and Shorter, J.A. 1995. Atmospheric chemistry and environmental impact of hydrofluorocarbons and hydrochlorofluorocarbons. In *Halon Replacements: Science and Technology*, Miziolek, A.W. and Tsang, W. (eds.), American Chemical Society Symposium Series 611, pp.16-30.

Wallington, T.J., Hurley, M.D., Fracheboud, J.M., Orlando, J.J., Tyndall, G.S., Sehested, J., Møgelberg, T.E. and Nielsen, O.J. 1996. Role of excited CF_3CFHO radicals in the atmospheric chemistry of HFC-134a. *Journal of Physical Chemistry* 100:18116-18122.

Warheit, D.B. 1993. *Mechanistic studies with HCFC 123*. Dupont Company, Haskell Laboratory Report HLR 828-92.

Wild, O., Rattigan, O.V., Jones, R.L., Pyle, J.A. and Cox, R.A. 1996. Two-dimensional modeling of some CFC replacement compounds. *Journal of Atmospheric Chemistry* 25:167-199.

WMO (World Meteorological Organization) 1989. Scientific Assessment of Stratospheric Ozone: 1989. In *Global Ozone Research and Monitoring Project, Report No. 20, Vol. 1*, Albritton, D.L. and Watson, R.T. (eds.), World Meteorological Organization, Geneva.

WMO (World Meteorological Organization) 1994. Scientific Assessment of Ozone Depletion: 1994. In *Global Ozone Research and Monitoring Project - Report No. 37*, Albritton, D.L, Watson, R.T. and Aucamp, P.J. (eds.), World Meteorological Organization, Geneva.

WMO (World Meteorological Organization) 1998. Scientific Assessment of Ozone Depletion: 1998. In *Global Ozone Research and Monitoring Project*, Albritton, D.L, Aucamp, P.J., Megie, G. and Watson, R.T. (eds.), World Meteorological Organization, Geneva, in press.

Wujcik, C.E., Zehavi, D. and Seiber, J.N. 1998. Trifluoroacetic acid levels in 1994-1996 fog, rain, snow and surface waters from California and Nevada. *Chemosphere* 36:1233-1245.

Zander, R., Demoulin, P., Ehhalt, D., Scmidt, U. and Rinsland, C. 1989. Secular increase of the total vertical column abundance of carbon monoxide over central Europe. *Journal of Geophysical Research* 94:11021-11028.

Zehavi, D. and Seiber, J.N. 1996. An analytical method for trifluoroacetic acid in water and air samples using headspace gas chromatographic determination of the methyl ester. *Analytical Chemistry* 68:3450-3459.

Zellner, R., Bednarek, G., Hoffmann, A., Kohlmann, J.P., Mörs, V. and Saathoff, H. 1994. Rate and mechanism of the atmospheric degradation of 2H-heptafluorpropane (HFC-227). *Berichte. Bunsengesellschaft für Physikalische Chemie* 98:141-146.

CHAPTER 7

EFFECTS OF INCREASED SOLAR ULTRAVIOLET RADIATION ON MATERIALS

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Summary

Synthetic polymers such as plastics, as well as naturally-occurring polymer materials such as wood, are extensively used in building construction and other outdoor applications where they are routinely exposed to sunlight. The UV-B content in sunlight is well known to adversely affect the mechanical properties of these materials limiting their useful life. Presently their outdoor lifetimes depend on the use of photostabilizers in the case of plastics and on protective surface coatings in the case of wood. Any increase in the solar UV-B content due to a partial ozone depletion would therefore tend to decrease their outdoor service life. It is the synergistic effect of increased UV radiation with other factors such as the temperature that would determine the extent of such reduction in service life. The increased cost associated with such a change would be felt unevenly across the globe. Those developing countries that depend on plastics as a prime material of construction and experience high ambient temperatures are likely to be particularly affected in spite of the relatively small fractional decrease in ozone at those locations.

Assessment of the damage to materials, associated with ozone depletion, requires a knowledge of the wavelength dependence as well as the dose-response characteristics of the polymer degradation processes of interest. While the recent literature includes some reliable spectral sensitivity data little dose-response information has been reported making it difficult to make such assessments reliably at the present time. This is particularly true for the naturally-occurring materials popularly used in construction applicastions.

To maintain polymers at the same useful lifetime in spite of increased solar UV-B content the amount of photostabilizers used in the formulations might be increased. This strategy assumes that conventional stabilizers will continue to be effective with the spectrally altered UV-B-enhanced solar radiation. While the present understanding of the degradation chemistry suggests the strategy to have merit, its effectiveness, in an altered solar radiation environment, has not been demonstrated for common polymers. The availability of this data is crucial to reliably estimate the cost of mitigating the increased damage to materials as a result of a possible partial depletion of the ozone layer using this approach.

Introduction

A wide variety of synthetic and naturally-occurring high polymers absorb solar ultraviolet radiation and undergo photolytic, photooxidative, and thermo-oxidative reactions that result in the degradation of the material (Rabek,1995: Scott, 1990: Wypych, 1990). The degradation suffered by these materials can range from mere surface discoloration affecting the aesthetic appeal of a product to extensive loss

of mechanical properties that severely limit its performance. The deleterious effects of solar UV-B radiation in particular, on wood, paper, biopolymers and polymers (plastics and rubber), are well known. The phenomenon is of special interest to the building industry which relies on polymer building products that are routinely exposed to sunlight during use. Most of the common polymers used in such applications contain photostabilizers to control photodamage and to ensure acceptable lifetimes under outdoor exposure conditions. In both the U.S. and Western Europe about 20-30 percent of the annual production of plastics resin is used by the building sector (Mader, 1992). The use of plastics in building applications is popular in the developing world as well because of the low cost and the ease of use of plastics components compared to the conventional metal, glass, mortar, wood and other materials. Plastics are used in other products such as outdoor furniture, fishing gear, and marine crafts, that are also routinely used outdoors. Table 7.1 illustrates the diversity of products that fall into this category. A large body of research literature on polymers deals directly with the issue of increasing the useful lifetimes of such products under routine outdoor exposure conditions (Shlyapnikov et al., 1996: Scott, 1985: Burn, 1994: Wypych, 1990).

Table 7.1 Plastics materials routinely exposed to solar UV radiation

Building applications	Plastic window and door frames, siding, mobile home skirting, gutters and downspouts, conduits, cable covering, flooring, outdoor furniture. Exterior fascia and soffit [rigid PVC formulations]
	Membrane roofing, geomembranes, weatherstripping [plasticized PVC, EPDM rubber, other rubbers]
	Glazing, covers for lighting fixtures [polycarbonate and acrylics]
	Varnishes and coatings used to protect surfaces. Highway marking paints. Resins used in the repair of monuments
Agricultural Applications	Irrigation hoses, pipes, netting [Polyethylene and PVC] Tanks for storage of water [Unsaturated polyester, and PE]
	Mulch films and greenhouse films [PE and PVC]
Transportation	Automobile tires [rubber]. Plastics used in automobile, aircraft, and marine vessel construction [composite]
Other	Fishing nets, sails, outdoor temporary housing, outdoor furniture, fibers and textiles
Biopolymers	Wool, human hair, wood, chitinaceous materials

PE: Polyethylene, PP: Polypropylene, PVC: Poly(vinyl chloride), EPDM: Ethylene -propylene- diene monomer

It is mainly the ultraviolet radiation in sunlight that presently determines the useful lifetime of even adequately photostabilized plastic products in outdoor applications. Any increase in the UV-B content in terrestrial solar radiation due to a partial depletion of the stratospheric ozone layer is therefore ex-

pected to have an impact on the outdoor lifetimes of this category of materials. The damage to polymers under exposure to UV-B radiation is generally intensity-dependent. While the incremental increase in UV-B in solar radiation due to ozone depletion is expected to be small, the efficiency of polymer degradation processes at these wavelengths is generally high. Marginal increases in solar UV levels can therefore translate into a noticeable decrease in the service life of polymer products. In applications such as organic protective coatings, electrical cable jackets and plastic fishing gear, premature failure of the material can involve significant indirect economic losses that greatly exceed the replacement cost of the polymer material. With these applications, it is not easy to estimate the magnitude of the losses consequent to increased UV in sunlight.

The severity of the ozone-layer depletion and the consequent enhancement of UV-B in terrestrial solar radiation is latitude dependent. Most depletion of the ozone layer occur at the higher latitudes where the largest increases in the UV levels is expected. While the solar radiation environment at these locations may become harsher due to additional UV-B, most of them enjoy relatively moderate temperatures that slow down the degradation reactions of materials. The change in the ozone column at low latitudes is relatively small, but these regions presently experience high ambient temperatures as well as high solar UV-B insolation. The service life of plastics under such harsh conditions is reported to be dramatically reduced. For instance, the tensile strength of white poly(vinyl chloride), PVC, pipes exposed for 24 months in Dhahran (Saudi Arabia) decreased by 43 percent while an exposure of the same duration in Florida resulted in only a 26 percent decrease in the property (Hussein et al., 1995). The combination of high ambient temperatures as responsible for the reduced lifetime of the product.. Even a small increase in solar UV-B levels can dramatically accelerate the deterioration processes in locations where the ambient temperature is high. In the tropical developing countries housing construction mostly relies on lumber and other plant materials while the use of plastics is also on the increase. Plastics are also extensively used in irrigation, water distribution and run-off applications, water storage tank construction, fishing nets, and agricultural films. A decrease in the service lives of these items can have a serious socio-economic impact on the populations in these countries.

The crucial role of temperature on the weathering of polyethylenes was illustrated in a recent study on desert exposure of polyethylene films. Two sets of polyethylene film samples one maintained at 25 C at all times in an air-cooled, UV-transparent enclosure, and the other left under the much higher ambient temperature, were exposed to sunlight outdoors. The air temperature varied in the range of 26 C to 36 C during the period of exposure. However, the surface temperature of plastics exposed to sunlight can be much higher (by as much as 60 C for common plastics depending on color and thickness) than that of the surrounding air due to heat build-up (Rabonavitch et al., 1983). Figure 7.1 shows the change in extensibility of the films obtained for each set of samples. Samples kept at the lower temperature deteriorated much slower relative to those at ambient temperature although both were exposed to the same dose of solar UV radiation. It is the synergistic effect of high temperature and solar UV radiaion that is responsible for the rapid degradation of the polyethylene films under these conditions. The findings are consistent with the observation that weathering rates of common plastics are very much slower when exposed floating in sea water in marine environment, compared to those exposed on land (Andrady, 1990). Water acting as a heat sink is able to maintain low sample temperatures, retarding deterioration.

For a given material, the decrease in service life due to increased UV levels in will invariably be determined by a) the spectral irradiance distribution of sunlight and environmental factors such as the ambient temperature ; b) the spectral sensitivity and the dose-response characteristics of the material; and c) the efficacy of the available light-stabilizers under spectrally-altered light conditions (Andrady, 1997). Any serious assessment of the extent of anticipated photodamage requires quantitative infor-

mation pertaining to each of these. The solar radiation data and the temperatures for most locations of interest are reliably known. A growing body of data on the spectral sensitivity of common polymers as well as some compounded systems used in specific applications (such as in PVC plastic siding) are becoming available (Andrady, 1997). However, the information on the dose-response characteristics of the degradation in plastics and natural materials, as well as on the effect of temperature on the UV-induced degradation, remain scant. Perhaps the least amount of information is available on the effectiveness of the conventional light-stabilizers when used under UV-enhanced sunlight conditions. Figure 7.2 shows the basic steps involved in photodamage and illustrates the various strategies commonly used to mitigate light-induced degradation of polymers.

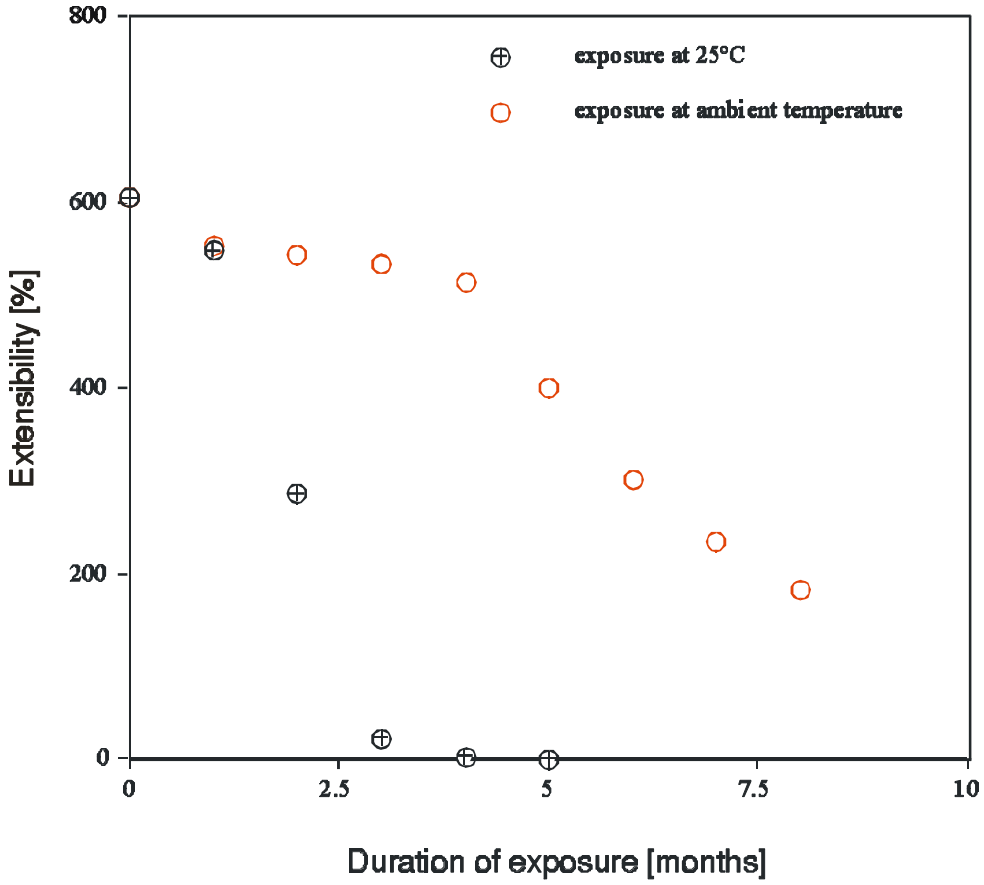


Fig. 7.1. Change in the extensibility of polyethylene film samples exposed in Dhahran, Saudi Arabia. The open symbols are for samples maintained at 45 C during the exposure, and the filled symbols are samples exposed under ambient conditions [Hamid 1998].

Effective light absorbers such as benzotriazoles, benzophenones and phenyl esters, as well as hindered amine light stabilizers (HALS) are presently used in plastics formulations intended for outdoor use (usually at a 0.05 -2.0 wt percent level). Improved stabilizers are introduced into the market periodically. Reliance on increased concentrations of these conventional light-stabilizers to maintain present service lifetimes of plastics products is the most likely response of the plastics industry to counter the effects of increased solar UV B levels. Reported data, such as those for harsh desert weathering experiments suggest that HALS and titania opacifier (Summers et al., 1983) used at higher levels can increase the service life of common plastics, considerably. However, the potential of the conventional photostabilizers to breakdown under exposure to enhanced UV solar radiation, possibly decreasing their effectiveness, is a concern. Even some commercial HALS compounds are reported to be photo-

lyzed by UV radiation (Pan et al, 1993) but no action spectra for the breakdown of even the common photostabilizers are available. Novel and more effective light-stabilizers might be developed to supplement the existing compounds.

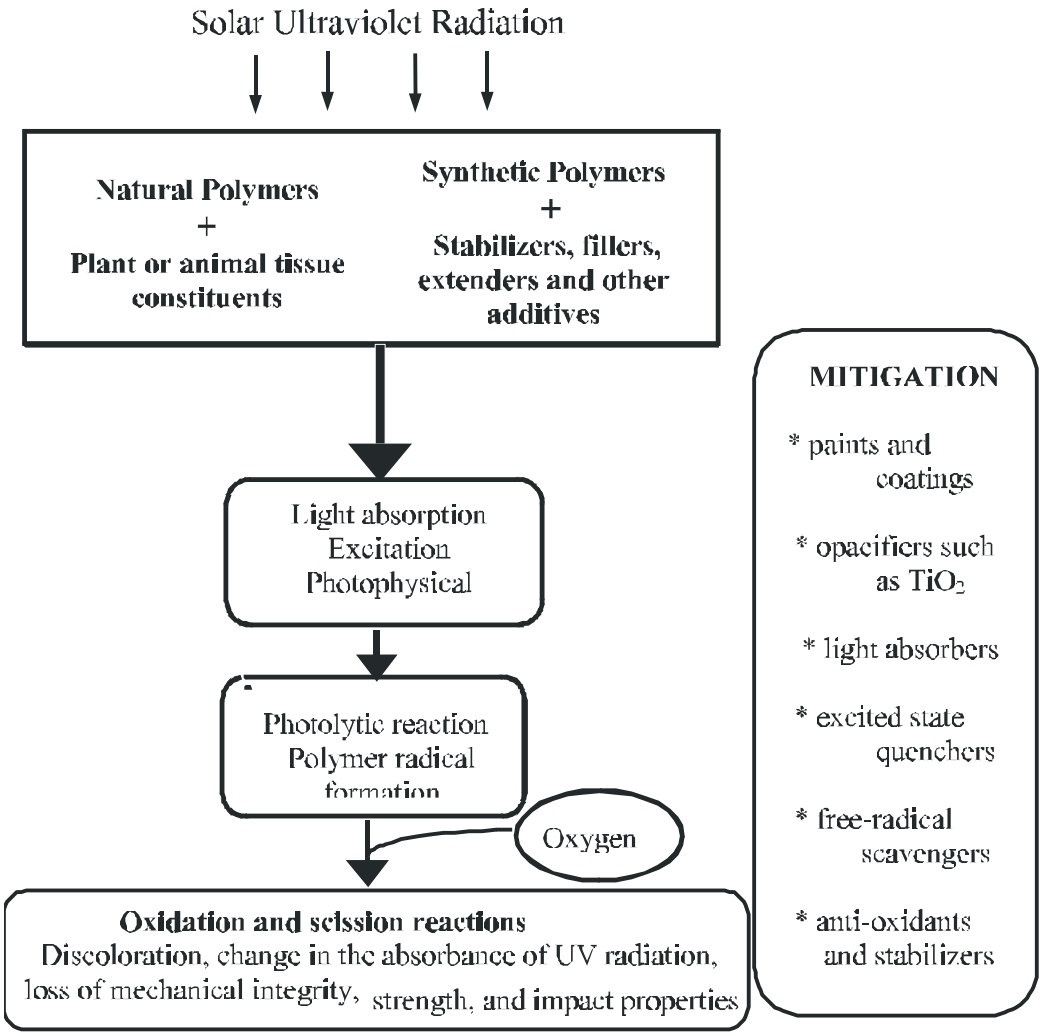


Fig. 7.2. A schematic diagram of the various stages of light-induced damage in polymers and its mitigation.

With organic light stabilizers such as hindered amines, increasing the stabilizer level in the composition will have little or no impact on processibility of the resin. The cost, however, will be significantly affected as the contribution of the stabilizer cost to the total cost of a product such as greenhouse films can be as much as 30 percent. With inorganic opacifiers such as titania or carbon used with resins such as PVC for instance, higher levels will affect processibility, power consumption, and even the lifetime of processing equipment, due to increased melt viscosity. Capstock technology (Moore, 1994) where a photolabile polymer is covered by a surface cap layer of the same material rich in light stabilizer, or a photoresistant polymer is also a promising approach As the incremental cost of these efforts rise, it is possible that other weather-resistant polymers will become more cost-effective for particular high-value applications.

In the case of high-value wood, protective surface coating will remain the primary means of controlling light-induced damage. With low-quality material (that is generally left unprotected) used in dwelling construction in developing countries a decrease in useful lifetime is expected. Use of wood in building might involve additional costs for more frequent painting or other maintenance.

UV Damage to Polymers

The chemical pathways by which common polymers photodegrade are fairly well known, but various aspects of the mechanisms involved remain unelucidated. However, it is important to take into account very significant influence of compounding additives in modifying these pathways (Gugumus, 1993). Typically, these are pigments, extenders, photostabilizers and thermal stabilizers. For instance, the effect of flame-retardant additives on the photodegradation of several common polymer compositions was reported recently (Torikai et al. 1998, 1993a-c). Virtually all plastics products are manufactured using extrusion, injection molding, or extrusion blowing. The processing of polymers using heat and high shear into useful end products introduces impurities and reaction products that make them susceptible to photodegradation. Because of these complications, the extrapolation of research findings on UV-induced degradation of pure polymer resins to compounded and processed products of the same polymer, is often unreliable. Photodegradation data generated on the actual polymer formulations used in practice, processed in the conventional manner are the most useful for assessment of damage.

The many concurrent chemical processes taking place in polymers exposed to UV radiation result in several different modes of damage, each progressing at a different rate. It is usually the critical first-observed damage process that determines the useful service life of the product. For instance, poly(vinyl chloride), PVC, window frame exposed to sunlight undergoes discoloration, chalking, loss of impact strength, and a reduction in tensile properties as well as a host of other chemical changes. It is, however, the discoloration (or the uneven yellowing) of the window frame that generally determines its service life [Ho, 1984]. The consumer may demand its replacement based on this criterion alone. In most developing countries, however, these products often continue to be used despite changes in appearance or evenfal stages of damage becomes apparent. With continued use, however, other damage such as chalking and eventually loss of impact resistance (leading to cracking) can occur making the product even more unacceptable. The two critical modes of photodamage applicable to most natural and synthetic materials are yellowing discoloration and loss in mechanical integrity.

Yellowing Discoloration. Both natural biopolymer materials and synthetic polymers undergo UV induced discoloration, usually an increase in the yellowness on exposure. Lignocellulosic materials such as wood and paper readily undergo light-induced yellowing (Hon et al., 1991). While both cellulose and lignin constituents of wood can photoyellow, it is the latter that is mostly responsible for the phenomenon. Lignin, which comprises 29-33% by weight of softwood, contains numerous chromophores that efficiently absorb UV radiation (Heitner, 1993). As much as 80-95% of the absorption coefficient of wood can be ascribed to the lignin fraction. The complex photochemistry of yellowing in lignin-containing materials is not completely understood; the present understanding of the process was succinctly summarized recently (Forsskåhl et al., 1993) and at least four pathways of photodamage have been recently discussed. The practical interest in discoloration relates specially to newsprint paper made of groundwood pulp that yellows rapidly on exposure to sunlight. Action spectra for photoyellowing of these pulps have been reported, and a recent study (Andrady et al., 1991) confirms the solar UV wavelengths to cause yellowing while the wavelengths in the region of 500 nm to 600 nm was

shown to photobleach the pulp. The cellulose fraction in wood also undergoes a free radical mediated degradation on exposure to wavelengths < 340 nm.

The photodamage to wool has serious economic implications in large producer countries. Exposure of wool keratins to sunlight is well known to cause yellowing, bleaching, and main-chain scission of the proteins (Lennox et al., 1971). Launer (Launer, 1965) established that visible radiation in sunlight causes photobleaching of wool while the UV wavelength causes photoyellowing. Based on Lennox data (Lennox et al., 1971), the most effective yellowing wavelengths were in the UV-A region (340 -420 nm). As ozone layer depletion results in an increase in both UV B as well as UV A content of sunlight, wool appears to be a material that might be particularly affected.

Preliminary data on the photostability of Chitosan, another commonly found biopolymer, were recently reported (Andrady et al., 1996). While not used commercially in high volume, the biopolymer occurs widely in nature in fungal cell walls, crustacean exoskelton and in insect tissue. Ultraviolet radiation in the wavelength range 250 nm to about 340 nm was reported to cause changes in the average molecular weight as determined by solution viscosity as well as the absorbance (at 310 nm) in chitosan derived from crab shells. The damaging role of UV-B in creating free+radicals in human hair has also been reported (Jahan et al., 1987) but no quantitative spectral sensitivity data are available.

Of the synthetic polymers, poly(vinyl chloride), PVC, is best-known for its tendency to undergo photoyellowing. The photothermal mechanisms leading to the formation of conjugated polyenes that causes yellowing, is well understood (Decker, 1984: Gardette et al., 1991). An opacifier (generally rutile titania) is used to slow down the rate of yellowing in white profiles widely used in siding, window frames and pipes (Titow, 1984). The reaction is localized in the surface layers of the polymer specially in opaque formulations used in building applications. The activation energy for dehydrochlorination is reported to have a temperature coefficient of 8-18 kJ mol^{-1} suggesting this process to be readily enhanced at high temperatures (Owen, 1984). As with wool and paper, while the UV-wavelengths cause yellowing of PVC the visible radiation >400 nm tend to cause photobleaching. Several possible photobleaching mechanisms are reported in the literature but the process is little understood.

A second polymer used in building applications, mainly as glazing, is polycarbonate. When irradiated with short wavelength UV-B or UV-C radiation polycarbonates undergo a rearrangement reaction (referred to as a photo-Fries rearrangement). At low oxygen levels this reaction can yield yellow-colored products such as o-dihydroxy-benzophenones (Rivaton et al., 1988). But when irradiated at longer wavelengths (including solar visible wavelengths) in the presence of air, polycarbonates undergo oxidative reactions that result in the formation of other yellow products (Factor et al., 1987). However, neither the detailed mechanisms nor the specific compounds responsible for the yellow coloration have been fully identified (Factor, 1995). Monochromatic exposure experiments on the wavelength sensitivity of several degradation processes of bis-phenol A polycarbonates have been reported recently (see Table 7.2).

Polystyrene, widely used in both building and packaging as expanded foam, also undergoes light-induced yellowing. The presence of air retards the process and the origin of the coloration is again not clear. It is variously attributed to conjugated polyene, various oxygenated species, or products of ring-opening reactions (Rabek et al., 1995).

Table 7.2 Spectral sensitivity data from monochromatic exposure experiments.

Material Type	Damage Studied	B	r^2	Ref
1. Poly(vinyl chloride)				
1.1 rigid compound - 0% TiO2	Yellowing	-0.035	0.95	1
- 0% TiO2		-0.048	0.99	
- 2.5% TiO2		-0.058	0.98	
- 5.0% TiO2		-0.073	0.99	
1.2 plasticized compound	Stiffness change	-0.02	0.83	2
2. Polycarbonate				
2.1 rigid sheets	Yellowing	-0.082	0.99	3
2.2 films	Quantum Yield of chain scission	-0.044	0.99	4
	Change in Absorbance	-0.059	0.88	5
3. Poly(methyl methacrylate)	Quantum Yield of chain scission	non-linear		6
4. Lignocellulose				
3.1 mechanical pulp	Yellowing	-0.011	0.99	7
5. Chitosan				
5.1 Chitosan films	Absorbance at 310 mm. (260 - 320nm)	-0.017	0.89	8
	Viscosity	non linear		
6. Wool	Yellowing	-0.025	0.95	9

Note: r is the correlation coefficient

References 1- [Andrady, 1989] 2- [Warner et.al., 1966] 3- {Andrady et.al., 1992]

4- [Torikai et.al., 1993a] 5- [Fukuda et.al. 1991] 6- [Mitsuoka et.al., 1993]

7- [Andrady et.al. 1991] 8- [Andrady et.al., 1996] 9- [Lennox et.al 1971]

Loss of Mechanical Integrity. The loss of strength, impact resistance, and mechanical integrity of plastics exposed to UV radiation is well known. These changes in bulk mechanical properties reflect polymer chain scission (and/or cross linking) as a result of photodegradation. Changes in solution viscosity and the gel permeation characteristics of polymers have been used (Torikai et.al., 1993) to establish molecular changes during photodegradation.

With polyethylene and polypropylene, the loss of useful tensile properties on exposure to solar radiation is a particular concern. These are used extensively in agricultural mulch films, greenhouse films, plastic pipes, and outdoor furniture. Polyethylene films exposed to solar UV-B radiation readily

lose their extensibility and strength (Hamid et al. 1991, 1995) as well as their average molecular weight (Andrady et. al., 1993). General features of the mechanism of photodegradation in both polyethylene and polypropylene is fairly well understood (Allen, 1983: Rabek, 1995). The mechanism is one of thermooxidative or photooxidative degradation rather than of direct photolysis, and is catalyzed by the presence of metal compounds. The free radical pathways that lead to hydroperoxidation and consequent chain scission are fairly well understood (Shlyapnikov et al., 1996). Of the polymers used worldwide, polyethylene enjoys the largest annual volume. Research interest in understanding and controlling the photdegradation process of this polymer is therefore continuing. Efficient classes of light stabilizers such as the hindered amine light stabilizers (HALS) are used to ensure that adequate lifetimes are obtained in polyolefin products intended for outdoor use under a wide range of UV environments.

Poly(vinyl chloride) PVC, is used widely in building applications where the impact strength of the material is an important requirement. The projected consumption of PVC in the near future (1995 - 2010) is much higher in the developing world and cin countries in transition. Estimated demand for Asia alone is more than that for the US, Canada and the European community combined(Gappert, 1996). Exposure to solar UV radiation is well known to decrease the impact strength of the polymer (Decker, 1984). As the surface layers of the plastic material degrades the titanium dioxide powder used as an opacifier is gradually released and may even form a surface layer loose enough to be rubbed off. This is responsible for “chalking” of extensively exposed PVC siding materials. Both the tensile strength and the extensibility of rigid PVC samples also decrease with the duration of exposure to solar UV radiation and the material finally embrittles (Decker, 1984). Similar changes also take place on exposure of plasticized PVC formulations used in membrane roofing applications and cable coverings (Matsumoto et al., 1984).

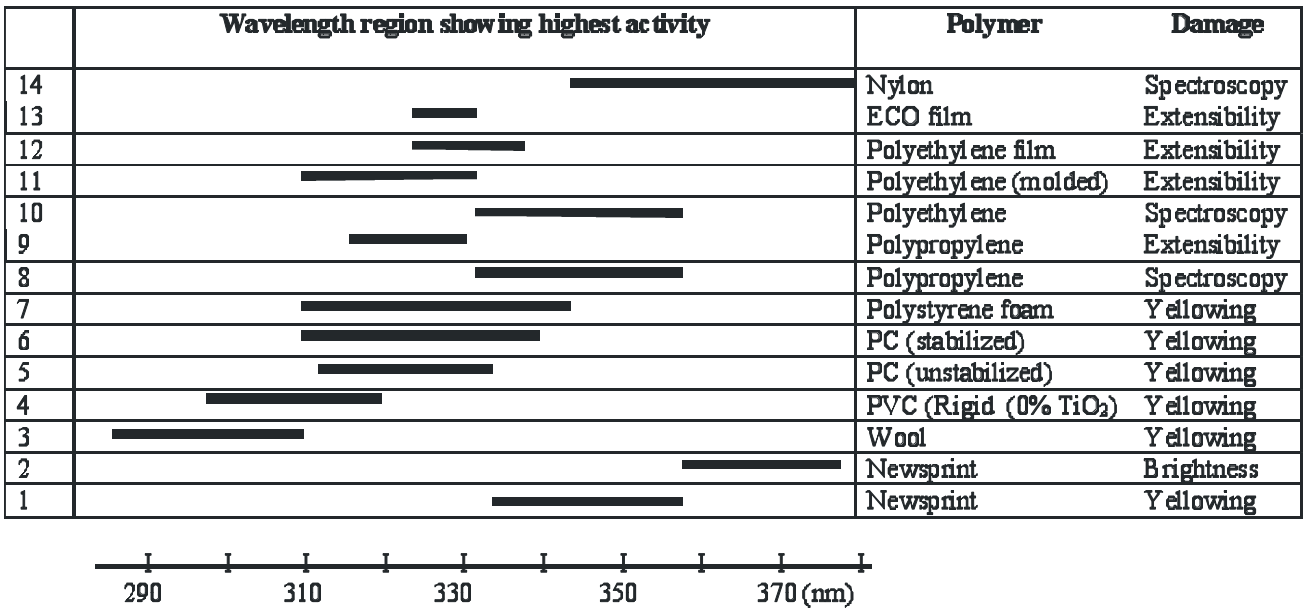
Other common polymers shown in Table 7.1 also undergo a loss in mechanical strength on photodegradation. A rapid change in the mechanical integrity of polystyrene caused by extensive chain scission during the photodegradation has been reported (Ghaffar et al.,1976).

Wavelength Sensitivity of Photodegradation

Assessing the damage to materials from exposure to UV-enhanced sunlight requires an understanding of their spectral sensitivity. Spectral sensitivity data for polymers are typically generated using a source of monochromatic radiation or a white light source such as a filtered xenon source (one whose spectral irradiance distribution is designed to closely approximate that of terrestrial sunlight at unit air mass). With experiments based on exposure of materials to monochromatic radiation, the effectiveness $\Im(\lambda)$ in units of damage per incident photon (defined as the ratio of the measured change in the property of interest ΔP to the number of incident photons), is obtained for several irradiation wavelength. In most instances a linear relationship exists between the logarithm of the effectiveness of damage and the wavelength of exposure, with higher damage per incident photon obtained at the shorter UV wavelengths. The gradient B of a plot of the natural logarithm of $\Im(\lambda)$ versus λ is a measure of the monochromatic wavelength sensitivity. The Table 7.2 lists the values of B that quantifies the wavelength sensitivity of polymeric materials reported in the literature. All data in the table are based on laboratory exposure experiments using monochromatic radiation. The spectral sensitivity of a material assessed by ths approach depends upon the type of damage process investigated.

When using a white-light source, a series of cut-on filters is used to isolate different bands of the source spectrum (Andrady 1997). The increment of radiation between two adjacent filters in the series depends on their transmission spectra T(λ) and the spectral irradiance distribution of the source H(λ).

$$\text{Increment} = H(\lambda)\,(\,T_i(\lambda) - T_{i+1}(\lambda)\,)$$



a) All data reported were generated using cut-on filter technique in combination with a filtered xenon source. The wavelength ranges shown are the intervals within which maximum photodamage was obtained. b) Abbreviations for plastics. ECO - {ethylene-carbon monoxide (1%) } copolymer: PC - Bisphenol A polycarbonate sheet: PVC - poly(vinyl chloride). c) 1 and 2. Newsprint paper made of mechanical pulp from Southern Pine. Photodamage measured is the increase in yellowness index and the optical brightness of the paper (Andrady et al., 1991). 3. Data estimated from the publication (Launer, 1965). 4. Rigid PVC sample similar to that used in vinyl siding applications except that no opacifier (titanium dioxide) was used. The absence of the opacifier allowed photodamage to be obtained within a reasonable duration of laboratory exposure. The reference source includes data for samples formulated with opacifier as well. (Andrady et al., 1989). 5. Data is for yellowing of bisphenol A polycarbonate sheet. The study also found crosslinking damage at wavelengths below 315 nm (Pyrde, 1985). 6 Commercial bisphenol A polycarbonate containing a light absorber as a photostabilizer was used to obtain these data using natural sunlight as a source of radiation (Andrady et al., 1991). 7 Data for expanded extruded polytyrene sheets designed to photodegrade faster than regular resin (Andrady unpublished data) 8. Data for polypropylene films (Zhang et al., 1996). 9. Injection molded polypropylene (Andrady unpublished data) 10-13. Data on polyethylene films (Andrady, 1996). 14. Nylon fibers (Hu, 1998).

Fig. 7.3. Summary of activation spectra for common polymers exposed to solar-simulated radiation (filtered xenon source radiation) published in the literature showing the wavelength interval in which most photodamage was obtained.

Typically, the change in a property of interest (Δ Damage), before and after exposure of identical samples exposed behind filters i and i+1 is obtain from the experiment. This is plotted as a function of the bandpass ($T_i(\lambda) - T_{i+1}(\lambda)$) at half bandwidth for each pair of filters used. The resulting bar dia-

gram has been referred to as an activation spectrum in the materials research literature. Wavelength sensitivity studies using a xenon source (that provides radiation spectrally similar to solar radiation, Hirt et al., 1967) are particularly valuable in assessing the potential effects of enhanced UV-B radiation in sunlight (Fig. 7.3). A qualitative estimate of the relative significance of various ultraviolet wavelengths in the terrestrial solar spectrum in causing specific photodamage in polymers can be discerned from the published activation spectra. Figure 7.4 shows a compilation of these regions from published data on various polymers. With activation spectra for most polymers, it is the less efficient UV-A wavelengths rather than the UV-B wavelengths that yield the largest relative amount of photodamage. This is expected because of the relatively low UV-B content in the solar spectrum. However, in activation spectra for yellowing of PVC (rigid formulations), polycarbonate, and polyamide, the maximum damage is obtained in a spectral region that includes UV B wavelengths as well. It is clear from the figure that most of the polymers will undergo considerable degradation when exposed to the UV-B and/or UV-A spectral regions, suggesting that any increase in the solar UV will result in an increase in damage.

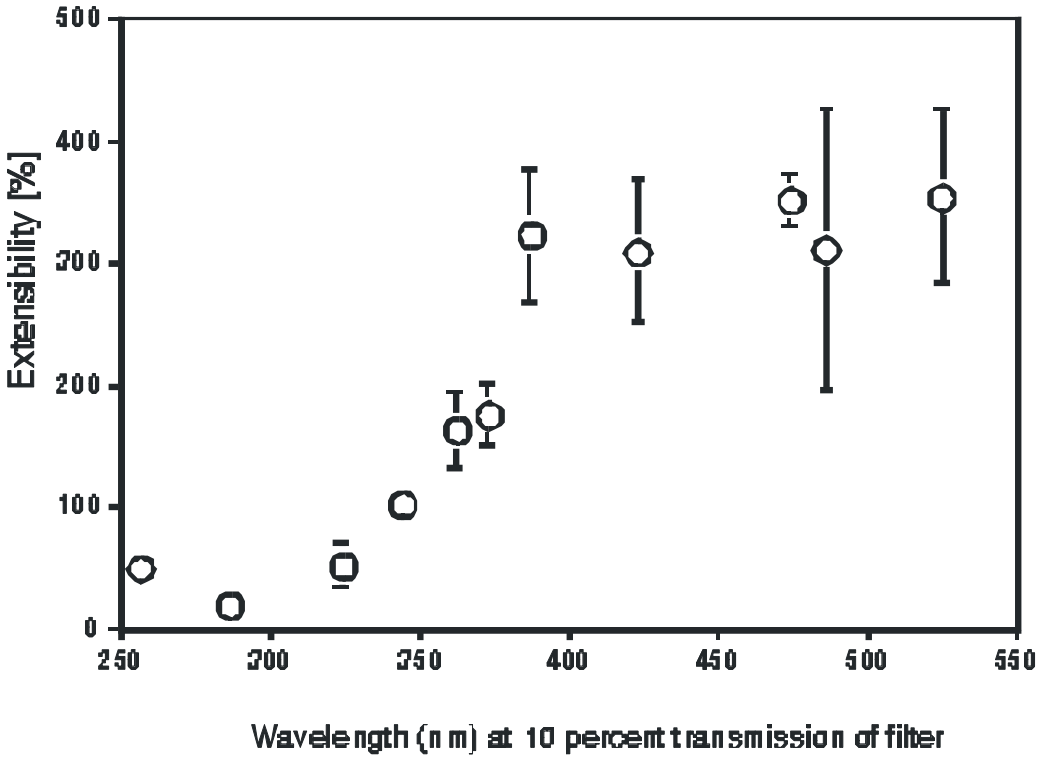


Fig. 7.4. Cumulative wavelength sensitivity curve for change in extensibility of low-density polyethylene film exposed to a filtered xenon source for 469 hours at ambient temperature (Hu, 1997).

The change in property of interest may also be plotted as a function of the 10 percent transmittance value of filters to yield a cumulative spectral sensitivity curve. Data on wavelength sensitivity of polyethylene film samples xposed to filtered xenon-source radiation is shown in Figure 7.4. The data shows that wavelengths shorter than about 400 nm affect the mechanical properties of the film. Using a full white light spectrum as opposed to narrow near-monochromatic bands of radiation in wavelength sensitivity experiment has the advantage that synergism (or antagonism) at different wavelengths can contribute to the results.

The spectral sensitivity data from the two experimental approaches (using near monochromatic radiation and using a white light source) are interconvertible provided several assumptions are made. i) the law of reciprocity applies ; ii) effects of different wavelengths are additive (the lack of synergistic or antagonistic effects); and iii) the light absorbance characteristics of the polymer are not time-dependent within the durations of exposure. For even the common polymers not enough data is often available to validate these assumptions. However, in the case of poly(vinyl chloride), PVC, and mechanical pulp materials, the activation spectra have been used to successfully derive monochromatic spectral sensitivity curves in the UV-B and UV-A wavelength range (Andrady, 1997).

Conclusions

The lack of reliable wavelength sensitivity data and dose-response information has always been a serious limitation in efforts to assess the increased damage to materials from enhanced UV levels resulting from ozone layer depletion. In recent years, however, this need has been partly addressed with several relevant action spectra for at least the common polymeric materials reported in the literature. More importantly some of this data pertains to formulations typically used in outdoor applications in the building industry. Also significant is the availability of spectral sensitivity data for several biopolymers during the recent years.

However, very limited dose-response data is available for these same compositions and the information reported is somewhat inconsistent. Furthermore, the photostabilizer effectiveness (or even their own photostability under exposure to UV-enhanced solar radiation) still remains unclear. These limitations do not allow reliable damage estimates for polymers typically used in building applications to be reliably estimated. The various assumptions typically employed in damage assessment for materials have not yet been validated for a great majority of polymers.

An important aspect of the problem is the role of temperature in exacerbating the effects of an increase in solar UV radiation. In regions with high ambient temperatures the assessment process must take into account the very high bulk temperatures polymers are subjected to during their service life. To do so effectively a better understanding of UV induced degradation processes at the lower temperatures will be particularly useful. Again the data available on this topic are very limited.

While the relevent information is begining to appear in the research literature, there is as yet inadequate dtata to conduct reliable damage estimates for most common polymers.

References

Allen, N.S 1983. *Degradation and Stabilization of Polyolefins*, Applied Science Publishers, London, pp. 373.

Andrady, A.L., Fueki, K. and Torikai, A. 1989. Photodegradation of rigid PVC formulations. I. Wavelength sensitivity of light induced yellowing by monochromatic light. *Journal of Applied Polymer Science* 37:935-946.

Andrady, A.L. and Pegram, J.E 1990. Weathering of polyethylene (LDPE) and enhanced photodegradable polyethylene in the marine environment, *Journal of Applied Polymer Science* 39:363-370.

Andrady, A.L., Pegram, J.E Tropsha, Y. 1993 Changes in carbonyl index and average molecular weight on embrittlement of enhanced photo-degradable polyethylene. *Journal of Environmental Degradation of Polymers* 1, 171-179.

Andrady, A.L., Fueki, K. and Torikai, A. 1991. Spectral sensitivity of polycarbonate to light-induced yellowing. *Journal of Applied Polymer Science* 42:2105-2107.

Andrady, A.L., Searle, N.D. and Crewdson, L.F. 1992. Wavelength sensitivity of stabilized and unstabilized polycarbonate to solar-simulated radiation. *Polymer Degradation.and Stability* 35:235-247.

Andrady, A.L., Pegram, J.E. and Tropsha, Y. 1993. Changes in carbonyl index and average molecular weight on embrittlement of enhanced photo-degradable polyethylene. *Journal of Environmental Degradation of Polymers* 1:171-179.

Andrady, A.L., Torikai, A. and Kobatake, T. 1996. Spectral sensitivity of chitosan photodegradation. *Journal Applied Polymer Science* 62:1465-1471.

Andrady, A.L. 1997. Wavelength sensitivity in polymer photodegradation. *Advances in Polymer Sciences* 128:49-94.

Burn, L.S. 1994. Lifetime prediction of uPVC pipes - experimental and theoretical comparisons. *Plastics Rubber and Composites Processing and Applications* 21:99-108.

Decker, C. 1984. Photodegradation of PVC. In *Degradation and Stabilization of PVC*, Owen, E.D. (ed.), Elsevier Applied Science Publishers, London, p. 81-136.

Ghaffar, A., Scott, G.A. and Scott, G. 1976. The chemical and physical changes occurring during U.V. degradation of high impact polystyrene. *European Polymer Journal* 11:271-275.

Factor, A., Ligon, W.V. May, R.J. and Greenberg, F.H. 1987. Recent advances in polycarbonate photodegradation. In *Advances in Stabilization and Controlled Degradation of Polymers II*, Patsis, A.V. (ed.), Technomic Publishing Company. Lancaster Pa., pp. 45 - 58.

Factor, A. 1995. Search for the sources of color in thermally aged, weathered, and gamma-ray irradiated Bisphenol A Polycarbonate. *Die Angewandte Makromolekulare Chemie* 232:27-43.

Forsskåhl, I. and Tylli, H. 1993. Light-induced yellowing of wood-containing papers. In *Photochemistry of Lignocellulosic Materials*, Heitner, C. and Sciano, J.C. (eds.), ACS Symposium Ser. 531 American Chemical Society, p. 45-59.

Fukuda, Y. and Osawa, Z. 1991. Wavelength effect on the photodegradation of polycarbonate and poly(methyl methacrylate). *Polymer Degradation and Stability* 34:75-80.

Gappert, G.1996. *Vinyl 2020: Progress, Challenges, Prospects for the next Quarter Centur,* The Vinyl Institute, Morristown New Jersey 07960.

Gardette, J., Lemaire, J. 1991. Photothermal and thermal oxidations of rigid, plasticized and pigmented poly(vinyl chloride). *Polymer Degradation and Stability* 34:135-167.

Gugumus, F. 1993. Re-evaluation of the stabilization mechanisms of various light stabilizer classes. *Polymer Degradation and Stability* 39:117-135.

Hamid, H. 1998. Unreported data. Personal communication.

Hamid, S.H., Amin, M.B. and Maadhah A.G. 1995. Weathering degradation of polyrethylene. In *Handbook of Polymer Degradation*, Hamid, S.H., Amin, M.B. and Maadhah A.G. (eds), Marcel Dekker Inc. New York.

Hamid, S.H., Pritchard, W. 1991. Mathematical modelling of weather-induced degradation of polymer properties. *Journal of Applied Polymer Science* 43:651-678.

Heitner, C. 1993. Light-induced yellowing of wood-containing papers. In *Photochemistry of Lignocellulosic Materials*. Heitner, C. and Sciano, J.C. (eds.), ACS Symposium Ser. 531 American Chemical Society. p. 3-25.

Hirt, R.C. and Searle, N.D. 1967. Energy characteristics of outdoor and indoor exposure sources and their relation to the weatherability of plastics. *Applied Polymer Symposia* 4:61-83.

Ho, B.Y.K 1984. Development of light-stable PVC stabilizer systems for rigid weatherable applications. *Journal of Vinyl Technology* 6:162-166.

Hon, D.N.-S. 1991. Photochemistry of wood. In *Wood and Cellulosic Chemistry*. Hon, D.N.-S. and Shiraishi, N. (eds.), Marcel Dekker, Inc. New York, pp. 525-556.

Hu, X. 1997. Wavelength sensitivity of photooxidation of polyethylene. *Polymer Degradation and Stability* 55:131-134.

Hu, X. 1998. Wavelength sensitivity of photooxidation of polyamide 6. (Manuscript accepted for publication by *Polymer Degradation and Stability*).

Hussein, I., Hamid, S.H. and Khan, J.H. 1995. Poly(vinyl chloride) pipe degradation studies in natural environments. *Journal of Vinyl and Additive Technology* 1:137-141.

Jahan, M.S., Drouin, T.R. and Sayre, R.M. 1987. Effect of humidity on photoinduced ESR signal from human hair. *Photochemistry and Photobiology* 45:543-546.

Launer, H.F. 1965. Effect of light upon wool. part IV: Bleaching and yellowing by sunlight. *Textile Research Journal* 35:395-400.

Lennox, F.G., King, M.G., Leaver, I.H., Ramsay, G.C., Savige, W.E. 1971. Mechanisms of prevention and correction of wool photoyellowing. *Applied Polymer Symposium* 18:353-369.

Mader, F.W. 1992. Plastics waste management in Europe. *Die Makromolekulare Chemie, Macromolecular Symposia* 57:15-31.

Matsumoto, S., Oshima, H., Hasuda, Y. 1984. Natural and induced weathering of plasticized poly(vinyl chloride). *Journal of Polymer Science, Polymer Chmirty Edition* 22:869-871.

Mitsuoka, T., A. Torikai, K. Fueki, 1993. Wavelength sensitivity of the photodegradation of poly(methyl methacrylate). *Journal of Applied Polymer Science* 47:1027-1032.

Moore, S. 1994. Construction offers window of opportunity to plastics. *Modern Plastics* April:46-48.

Owen, E.D. 1984. Degradation and Stabilization of PVC, Degradation and Stabilization of PVC, Elsevier Applied Science Publishers, New York, p. 320.

Pyrde, C.A. 1985. Photoaging of polycarbonate: effects of selected variables on degradation pathways. In *Polymer Stabilization and Degradation*, Klemchuk, P.P. (ed.), Symp. Ser. 280 American Chemical Society, p. 329-353.

Rabek, J.F. 1995. *Polymer Photodegradation*, Chapman and Hall, London, England.

Pan, J. and Cui, S. 1993. Study of the photolysis of a commercial hindered amine light stabilizer. *Polymer Degradation and Stability* 40:375-378.

Rabek, J.F. 1995. *Polymer Photodegradation*, Chapman and Hall, London, pp. 655.

Rabonavitch, E.B., Queensberry, J.G. and Summers, J.W. 1983. Predicting heat buildup due to sun's energy. *Journal of Vinyl Technology* 5:110-115.

Rivaton, A. and Lemaire, J. 1988. Photooxidation and thermooxidation of tetramethyl-bisphenol-A polycarbonate. *Polymer Degradation and Stability* 23:51-73.

Scott, G. 1990. *Polymer Degradation and Stabilization*, Elsevier Applied Science Publishers, London.

Scott, G. 1985. *New Trends in the Photochemistry of Polymers* Elsevier Applied Science Publishers, London.

Shlyapnikov, Y.A., Kiryushkin, S.G. and Mar'in, A.P 1996. *Antioxidative Stabilization of Polymers*, Taylor and Francis Publishers Ltd, London, England.

Summers, J.W. 1983. Formulations for vinyl housing siding: History, present, future. *Journal of Vinyl Technology* 5:43-46.

Titow, W.V. 1984. *PVC Technology.* Fourth Edition. Elsevier Applied Science Publishers, New York. pp. 1233

Torikai, A., Kobatake, T. and Okisaki, F. 1998. Photodegradation of polystyrene containing flame retardants. *Journal of Applied Polymer Science* 67:1293-1300.

Torikai, A., Mitsuoka, T. and Fueki, K. 1993a. Wavelength sensitivity of the photo-induced reactions in polycarbonate. *Journal of Polymer Science. A: Chemistry E*dition 31:2785-2788.

Torikai, A., Kato, H. Fueki, K. Suzuki, Y. Okisaki, A. and Nagata, M. 1993b. Photodegradation of polymer materials containing flame-cut agents. *Journal of Applied Polymer Science* 50:2185-2190.

Torikai, A., Chigata, K. and Fueki, K. 1993c. Photodegradation of incombustible polymer materials. *Polymer Preprints. Japan* 42:2045-2047.

Warner, W.C., Gruber, E.E. 1966. Light aging of a polyblend film under interference filters, *I&EC Product Research and Development* 5:219-229.

Wypych, J. 1990. *Weathering Handbook.* Chemtec Publishing, Toronto, Canada, pp. 512.

Zhang, Z., Hu, X. and Luo Z. 1996. Wavelength sensitivity of photooxidation of polypropylene. *Polymer Degradation. and Stability* 51:93-97.

FREQUENTLY ASKED QUESTIONS (FAQ)

co-ordinated by

Janet F. Bornman and Jan C. van der Leun

Health Effects

1) How do we balance the good and bad effects of sunlight on human health?

In general, moderate exposure to sunlight in the course of everyday life is not detrimental. This basic exposure evidently allows us to function normally, and it proves to be sufficient to maintain an adequate level of vitamin D (in combination with our dietary intake). While sunlight is important for physical health it also causes various adverse health effects such as skin cancer, ageing of the skin, eye disorders and suppression of the immune system. It is clear that excessive UV exposure should be avoided to minimise the risk of development of such disorders.

2) How strong is the evidence that UV-B radiation causes skin cancer in humans?

The evidence is strong. The earliest experimental evidence that UV-B radiation causes skin cancer was acquired with animals; in humans there was a clear association between sun exposure and skin cancer, but that did not point specifically to UV-B. In recent years the advancement of molecular biology has provided us with analyses that produce direct evidence that genetic alterations found in human skin carcinomas are indeed caused by UV-B radiation.

3) Should one have all moles removed to decrease the risk of skin cancer?

No, there is no evidence to suggest that removing all of the moles would reduce the risk of skin cancer. However, it is important to be alert to atypical moles, especially those exhibiting changes in appearance (in colour or at the edges), and to screen those individuals that are known to run a high risk, either from a family history of melanoma mortality or of atypical moles.

4) Do sunglasses protect against cataracts?

Sunglasses that markedly reduce the UV-exposure of the eyes will reduce UV damage, such as cataracts. The best protection is achieved by a combination of UV-absorbing glasses and a shielding against light coming into the eyes from the sides. However, some sunglasses may not effectively block UV radiation and eye damage may occur.

Duration of Exposure to UV-B Radiation

5) Is the UV amount one receives as a child important even in later years?

Yes. Children should not be overexposed to UV radiation: sunbathing should be strongly discouraged. UV exposure, and especially sunburns, in early life can substantially increase the skin cancer risk later in life (especially the risk of basal cell carcinoma and melanoma).

Even if the risk is related to total accumulated exposure, as appears to be the case for a part of the non-melanocytic skin cancers (SCC), exposures early in life still may carry a greater risk. There is a long lag time, typically of several decades, between exposure and the development of a tumour. Therefore, early exposures have a greater probability in resulting in a tumour.

Are Animals at Risk?

6) Are hair-covered animals at any risk?

Yes. Skin cancer is found in almost all animals that have been studied in the long-term, for example, cattle, goats, sheep, cats, dogs, guinea pigs, rats, and mice. Direct effects of UV-B radiation on body parts which are covered by thick hair are negligible. However, even furred animals usually have exposed skin around mouth and nostrils, and sometimes on some other parts of the body. These parts, unless they are heavily pigmented, can be damaged by radiation.

7) Will penguins be affected by the ozone hole?

To our knowledge there are no studies concerning UV-B effects on penguins. As their eyes are exposed to a lot of UV due to the high reflectivity of snow and a marked enhancement during the ozone hole, investigation into the impact on penguins is desirable. The fact that penguins are visual predators, eating krill or fish in the water column, would make any eye damage an important issue for survival.

8) Is UV-B radiation a factor in the decline of frogs and other amphibians?

Possibly. Amphibian populations are in serious decline in many areas of the world, and scientists are seeking explanations for this. Most amphibian population declines are probably due to habitat destruction or habitat alteration. Some declines are probably the result of natural population fluctuations. Other explanations for the population declines, as well as the reductions in range of habitation, include disease, pollution, atmospheric changes and introduced competitors and predators. UV-B radiation is one agent that may act in conjunction with other stresses to adversely affect amphibian populations. Field studies in which embryos of frogs, toads, and salamanders were exposed to natural sunlight or to sunlight with UV-B radiation removed have shown conflicting results. Some studies resulted in increased embryonic mortality after UV-B exposure, whereas others show that current levels of UV-B radiation are not detrimental. Factors such as water depth, water colour, and the dissolved organic content of the water at the sites of egg deposition effectively reduce UV-B penetration through the water and reduce exposure to UV-B radiation at all life history stages. Biotic factors, such as jelly capsules around eggs, melanin pigmentation of eggs, and colour of larvae and metamorphosed forms, further reduce the effects of UV-B exposure.

Aquatic Life

9) Does water effectively shield aquatic organisms from UV exposure?

No. Pure water is quite transparent to UV radiation; a beam of UV-B radiation must travel over one-half kilometre through pure water in order to be completely absorbed. Natural waters do contain UV-absorbing substances, such as dissolved organic matter, that partly shields aquatic organisms from UV-B, but the degree of shielding varies widely from one water body to another. In clear ocean and

lake waters ecologically-significant levels of UV-B can penetrate to several tens of meters; in contrast, in turbid rivers and wetlands UV-B may be completely absorbed within the top few decimetres. Most organisms in aquatic ecosystems, such as phytoplankton, live in the illuminated euphotic zone close to the water surface where exposure to UV-B can occur. In particular, UV-B radiation may damage those organisms that live at the surface of the water during their early life stages.

Terrestrial Plant Life

10) What will be the effects of an increased UV-B radiation on crop and forest yields?

There are some UV-B-sensitive varieties of crops that experience reductions in yield. However, there are also UV-B-tolerant varieties, providing the opportunity to breed and genetically engineer UV-B tolerant varieties. For commercial forests, tree breeding and genetic engineering may be used to improve UV-B tolerance. For unmanaged or natural forests, these methods are not an option. While many forest tree species appear to be UV-B tolerant, there is some evidence that UV-B effects, sometimes detrimental, can slowly accumulate from year to year. If this finding is a general phenomenon, this would be cause for concern since it would greatly complicate breeding efforts in commercial forests and negatively affect natural forests.

11) Can plants protect themselves against increased UV-B?

Yes, partly. Plants already have reasonable UV shielding; for most plants only a small proportion of the UV-B radiation striking a leaf actually penetrates very far into the inner tissues. Also, when exposed to an enhanced UV-B level, many species of plants can increase the UV-absorbing pigments in their tissues. Other adaptations include increased thickness of leaves which reduces the proportion of inner tissues exposed to UV-B radiation. Several repair mechanisms also exist in plants, as is the case for other organisms. This includes repair systems for DNA damage or oxidant injury. The net damage a plant experiences is the result of the balance among damage, protection and repair processes. For many plants, the net damage is negligible.

Location-specific Issues

12) Is the increase in UV-B radiation caused by ozone depletion equivalent to that incurred by moving several hundred kilometres towards the equator?

Yes, but this comparison does not nullify the serious impact of an ozone depletion, as is sometimes suggested by questions like this. The suggestion is based on a fallacy, namely, comparing a personal risk perception with the effect on a population. An elevation of say 10% in risk would not be noticeable for the person involved. For a population it is quite different. With regard to skin cancer such an increase could mean 100-200 extra cases a year per million people. This would be an important public health effect. However, movements of entire populations, or even ecosystems, do not usually occur in a human lifetime, and the comparison is therefore inappropriate.

13) Can organisms adjust to a changed UV environment?

Yes, many organisms can respond physiologically with changes such as development of UV screening compounds and additional layers of protective tissues. However, there are genetic limitations to the degree to which these physiological adjustments can take place for each organism. Some can adjust more effectively than others. Over long periods of time and several generations of populations, there

is the possibility that genetic adaptation can develop as well. However, in organisms with moderately long life spans and small population sizes, the genetic adaptation is likely to be very slow.

14) Does ozone depletion pose any danger in the tropics?

Probably not. Increases in UV-B radiation are unlikely, since no significant trend in stratospheric ozone has been observed in the tropics. However, viewing the biosphere as a unit, there may be indirect effects of ozone depletion at other latitudes on tropical ecosystems. If ozone were to be depleted in the tropics, this would constitute a serious danger because of the naturally occurring high levels of UV-B radiation due to the high solar angles and already relatively low normal stratospheric ozone levels.

15) Do we need to worry about relatively small increases in UV-B due to ozone depletion, when natural variability is so much larger?

Yes. The change in UV-B from ozone depletion is systematically upward. The natural variability (e.g., from time of day, or clouds) can be larger, but goes in both directions, up and down. While the evidence for ozone depletion is very strong, there is little evidence for long-term changes in cloud cover.

Many detrimental effects of UV-B are proportional to the cumulative UV-B exposure. For example, skin cancer results from the total exposure accumulated over many years under both sunny and cloudy conditions. Any systematic increase in UV-B radiation will increase incidence among a population (as well as individual risk) regardless of the natural variability of the UV-B radiation.

16) Does one get higher UV exposures at higher elevations?

Yes. Higher elevations have less atmosphere overhead, as evidenced by the thinner air and lower atmospheric pressure. The increase in sun-burning UV radiation is typically about 5-10% for each kilometre of elevation, the exact number depending on the specific wavelength, solar angle, reflections, and other local conditions. Frequently, other factors besides thickness of the atmosphere cause even larger differences in UV radiation between elevations. Snow is more common at higher elevations, and reflections from it can lead to very large increases in exposure.

Lower locations tend to have more haze and more polluted atmosphere which can block some UV radiation.

17) Does air pollution protect one from UV-B radiation?

Yes, but at a high price. Air pollution is generally undesirable due to the numerous other serious problems associated with it, including respiratory illness, eye irritation, and damage to vegetation. While most of the atmospheric ozone resides in the stratosphere, some ozone is also made in the troposphere by the chemical interactions of pollutants such as nitrogen oxides and hydrocarbons. This tropospheric ozone is a component of the photochemical smog found in many polluted areas. Airborne particles (smoke, dust, sulphate aerosols) can also block UV radiation, but they can also increase the amount of scattered light (haze) and therefore increase the UV exposure of side-facing surfaces (e.g., face, eyes).

No single value can be given for the amount of UV-B reduction by pollution, because pollution events tend to be highly variable and local. Comparisons of measurements made in industrialised regions of the Northern Hemisphere (e.g., central Europe) and in very clean locations at similar latitudes in the Southern Hemisphere (e.g., New Zealand) suggest pollution-related UV-B reductions can be important.

Clear Skies vs. Cloud Cover

18) Can changes in cloudiness cause larger UV changes than ozone depletion?

Long-term trends in cloud type and amount are largely unknown due to the relatively short data record of comprehensive cloud observations, and the high variability of clouds on inter-annual and longer time scales. Some evidence exists showing that, at least over the time span of satellite-based ozone measurements, changes in cloud cover have been much less important than stratospheric ozone reductions in causing surface UV changes.

19) Are the risks of ultraviolet (UV) exposure at the beach less on a cloudy day?

Not necessarily. The effect of clouds on UV radiation is as varied as the clouds themselves. Fully overcast skies lead to reductions in surface UV irradiance. On average, scattered or broken clouds also cause reductions, but short-term or localised UV levels can be larger than for cloud-free skies if direct sunlight is also present. Clouds tend to randomise the directions of the incoming radiation (because of scattering) so that a hat may provide less protection on a cloudy day relative to a clear day.

Furthermore, people often change their behaviour on cloudy days. If they spend more time out in the open, or forego the use of sunscreen, they may end up with a very bad sunburn. In general, less UV radiation is received per hour under an overcast sky than under a clear sky, but extending one's stay at the beach may easily compensate for this effect. A completely cloud-covered sky may still transmit substantial amounts of UV-B radiation. In principle, any amount of UV-B radiation exposure contributes to the skin cancer risk.

Sunbathing

20) Will sunscreens protect one from harmful effects of increased UV-B radiation?

Not always. Sunscreens applied to human skin limit the penetration of UV radiation into the skin, and thus sunburn can be prevented. Sunscreens were primarily developed for this purpose. The effectiveness of sunscreens in protecting against skin cancer and immune suppressions is under debate. Any effectiveness in these respects may well be lost if the sunscreen is used to stay out in the sunlight longer than would be done without the sunscreen. It should also be kept in mind that there are other ways to protect the skin. These include staying out of the sunlight during the hours when the UV-B is maximal around solar noon, seeking the shade, wearing clothes, and especially hats.

21) Will getting a suntan help prevent skin cancer?

No. There is no evidence that getting a suntan will help prevent skin cancer. The UV exposure needed to acquire the tan adds to the skin cancer risk. The fact that one is able to tan well does, however, signify that the personal risk is lower (by a factor of 2 to 3) than for people who do not tan. Naturally dark-skinned people have a built-in protection of their skin against sunlight.

22) Is tanning with UV lamps safer than with sunlight?

No. The risks are approximately equal. For some time it was hoped that UV lamps could be made safer by making more use of long-wavelength (UV-A) radiation. That type of radiation is much less carcinogenic than the shorter-wavelength UV-B radiation, but one needs more UV-A than UV-B for acquiring a tan.

Economic Consequences

23) Has the benefit of the Montreal Protocol been worth the cost?

Yes. Several attempts have been made to investigate the economic impacts of the problem of a depleted ozone layer. Such attempts meet with many problems. There are good reasons for concern for effects on humans, animals, plants and materials, but most of these cannot be estimated in quantitative terms. Calculating the economic impact of such effects is uncertain. Moreover, economic terms are applicable only to some of the effects, such as the cost of medical treatments, and the loss of production in fisheries and agriculture, and damage to materials; but what is the cost equivalent of suffering, of a person becoming blind or dying, or the loss of a rare plant or animal species?

In spite of all these difficulties, attempts have been made. The most comprehensive example is a study initiated by Environment Canada for the 10th anniversary of the Montreal Protocol on Substances that Deplete the Ozone Layer. In this study, 'Global Costs and Benefits of the Montreal Protocol' (1997), the costs were calculated for all measures taken internationally to protect the ozone layer, such as replacement of technologies using ozone-depleting substances. The benefits are the total value of the damaging effects avoided in this way. The total costs of the measures taken to protect the ozone layer were calculated to be 235 billion US (1997) dollars. The effects avoided world-wide, though far less quantifiable, were estimated to be almost twice that amount. This latter estimate included only reduced damage to fisheries, agriculture and materials. The cataracts and skin cancers, as well as the potential associated fatalities avoided, were listed as additional benefits, and not expressed in economic terms.

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List of Abbreviations

AF amplification factor
AK actinic keratosis
APC antigen presenting cell
BAF biological amplification factor
BCC basal cell carcinoma
CDK climatic droplet keratopathy
CDKN2 cyclin dependent kinase inhibitor 2
CDOM colored (or chromophoric) dissolved organic matter
CFC chlorofluorocarbons
CFC chlorofluorocarbons
CH contact hypersensitivity
CM cutanteous melanoma
DIC dissolved inorganic carbon.
DMS dimethyl sulfide
DOC dissolved organic carbon
DOM dissolved organic
DTH delayed type hypersensitivity
DU Dobson unit
EPA Environmental Protection Agency
g DW grams per dry weight
HCFC hydrochlorofluorocarbon
HCFC hydrochlorofluorocarbon
HFC hydrofluorocarbon
HIV human immunodeficiency virus
HPV human papilloma virus
IL Interleukin
MS multiple sclerosis
N_2O nitrous oxide
NaTFA sodium trifluoro acetate
NMHCs non-methane hydrocarbons
NMSC non-melanoma skin cancer
NO nitric oxide
NOEC no effect concentration
NO_x nitrogen oxides
OCS carbonyl sulfide
PAR photosynthetically active radiation
PEC predicted environmental concentration
PLE polymorphic light eruption
POC particulate organic carbon
PSC posterior subcapsular cataracts
PUVA psoralen + UVA
RAF radiation amplification factor
ROS reactive oxygen species
SCC squamous cell carcinoma
SLE systemic lupus erythematosus
TDD trichothiodystrophy
TFA trifluoroacetic acid
TNFα tumor necrosis factor alpha
TOMS Total Ozone Mapping Spectrometer
UCA urocanic acid
UV-A electromagnetic radiation of wavelengths in the 315 to 390 nm range
UV-B electromagnetic radiation of wavelengths in the 280 to 315 nm range
UV ultraviolet
VOC volatile organic compounds
XP xeroderma pigmentosum

Hintere Umschlagseite:

The Vienna Convention for the Protection of the Ozone Layer (1985) and the Montreal Protocol on Substances that Deplete the Ozone Layer (1987) are now recognized as very successful in preventing a global environmental catastrophe, which could have been caused by stratospheric ozone depletion.

Scientific assessment reports have long stimulated the environmental policy process for the protection of the ozone layer. The Montreal Protocol provides for the following scientific assessment process: beginning in 1990, and at least every four years thereafter, the Parties shall assess the control measures provided for in Article 2 and Articles 2A to 2H on the basis of available scientific, environmental, technical and economic information.

This procedure has been followed since then and, in this publication, the Environmental Effects Assessment Panel is presenting herewith the latest of its assessments. This assessment is the product of the joint efforts of scientists from developed and developing countries from all regions of the world.

Readers will find information on the effects of increased ultraviolet radiation (UV-B) on human health, terrestrial ecosystems, aquatic ecosystems, biogeochemical cycles, tropospheric composition and air quality and materials, and a section also entitled “Frequently Asked Questions”.

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