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ENVIRONMENTAL EFFECTS OF OZONE DEPLETION AND ITS INTERACTIONS WITH CLIMATE CHANGE:
2006 ASSESSMENT

Pursuant to Article 6 of the Montreal Protocol on Substances that Deplete the Ozone Layer under the Auspices of the United Nations Environment Programme (UNEP).

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Introduction

This assessment was prepared by the Environmental Effects Assessment Panel for the Parties to the Montreal Protocol. The assessment reports on some of the new findings since the last full assessment of 2002, again paying attention to the interactions between ozone depletion and climate change and their consequences for environmental and health issues. Simultaneous publication of the assessment in the scientific literature aims to show the scientific community how their data, modeling, and interpretations are playing a role in information dissemination to the Parties to the Montreal Protocol and other policy makers. It is also hoped that the publication will stimulate the scientific community to continue working on the gaps in knowledge that still exist.

The 2006 assessment will be published in the Journal Photochemical & Photobiological Sciences, 2007.

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TABLE OF CONTENTS

LIST OF ABBREVIATIONS

1,25(OH)2D	1,25-dihydroxyvitamin d
25(OH)D	25-hydroxyvitamin d
AK	Actinic keratosis
AO	Arctic Oscillation. A large-scale variation in Arctic wind patterns
APase	Alkaline phosphatase
APC	Antigen presenting cell
ASL	Above sea level
BCC	Basal cell carcinoma (s)
Br	Bromine (an ozone depleting chemical)
BrO	Bromine monoxide
BSWF	Biological spectral weighting functions
BWF	Biological weighting function
CAS	Chemical Abstracts Service
CC	Cortical cataract(s)
CDFA	Chlorodifluoroacetic acid
CDK	Climatic droplet keratopathy
CDOC	Colored dissolved organic carbon
CDOM	Colored (or chromophoric) dissolved organic matter
CPD	Cyclobutane pyrimidine dimmer
CFC	Chlorofluorocarbon. Ozone-damaging chemical (e.g., $CFC1_2$: dichlorodifluoromethane. CCl_2F_2), now controlled under the Montreal Protocol
CH	Contact hypersensitivity
CH_4	Methane (a greenhouse gas)
CIE	Commission Internationale de l'Eclairage (International Commission on Illumination)
Cl	Chlorine (an ozone depleting chemical)
CM	Cutaneous melanoma
CO	Carbon monoxide

CO_2	Carbon dioxide (a greenhouse gas)
COS	carbonyl sulfide
CPD	Cyclobutane pyrimidine dimer
Cu	Copper (Cu(I) and Cu(II) being different oxidation states)
DIC	Dissolved inorganic carbon
DMS	Dimethylsulfide
DMSP	Dimethylsulfoniopropionate
DNA	Deoxyribonucleic acid
DOC	Dissolved organic carbon
DOM	Dissolved organic matter
DON	Dissolved organic nitrogen
DSB	Double strand break
DTH	Delayed type hypersensitivity
DU	Dobson Unit (used for the measurement of total column ozone (1 DU=2.69 × 1016 molecule cm^{-2})
EAE	Experimental allergic encephalitis
EDUCE	European Database for Ultraviolet Radiation Climatology and Evaluation
EESC	Equivalent Effective Stratospheric Chlorine
ENSO	El Niño Southern Oscillation. A large-scale climate variability in the Pacific region
EP	Earth Probe (a NASA satellite)
EPA	Environmental Protection Agency
EV	Epidermodysplasia verruciformis
Fe	Iron (Fe(II) and Fe(III) being different oxidation states)
FMI	Finnish Meteorological Institute
GHG	Greenhouse gas
Glu I	A pathogenesis-related (PR) protein
HALS	Hindered Amine Light Stabilizer
HCFC	Hydrochlorofluorocarbon. Interim replacements for CFCs with small ozone depletion potential (e.g., R22: chlorodifluoromethane $CHClF_2$) to be phased out

HFC	Hydrofluorocarbon. Long-term replacements for CFCs, with zero ozone depletion potential
Hg	Mercury (Hg0aq and Hg(II) being different oxidation states)
HIV	Human immunodeficiency virus
HPV	Human papillomavirus
HSV	Herpes simplex virus
HY5	Transcription factor HY5, which is a key downstream effector of the UVR8 (UV-regulatory protein) pathway
IBD	Inflammatory bowel disease
IL	Interleukin
Ink4a	Murine inhibitor of kinase 4a protein (gene in italics)
IPCC	Intergovernmental Panel on Climate Change
IPF	Immune protection factor
kda	Kilodalton
KNMI	Dutch National Institute for Weather, Climate and Seismology (The Netherlands)
L·	Lipid radical
MAA	Mycosporine-like amino acids
Mb	Megabase, equal to 1 million base pairs
MC1R	Melanocortin 1 receptor
MHC	Major histocompatibility complex
MS	Multiple sclerosis
N_2O	Nitrous oxide (a greenhouse gas that is also a source of NO_2)
NAO	North Atlantic Oscillation. A large-scale variation and redistribution of atmospheric mass in the Atlantic region producing large changes in the NH dynamics
NASA	National Aeronautic and Space Administration (USA)
NaTFA	Sodium trifluoroacetate
NC	Nuclear cataract(s)
NCAR	National Centre for Atmospheric Research, USA
NH	Northern Hemisphere.

NIMBUS-7	A NASA satellite
NIVR	Netherlands Agency for Aerospace Programmes
NMHCs	Non-methane hydrocarbons
NMSC	Non-melanoma skin cancer
NO	Nitric oxide (an ozone depleting gas)
NO_2	Nitrogen dioxide (an ozone depleting gas)
NOAA	National Oceanic and Atmospheric Administration, USA
NOEC	No observed effect concentration
NOx	Nitrogen oxides
O_3	Ozone
OCS	Carbonyl sulfide (also COS)
ODS	Ozone depleting substance(s)
·OH	Hydroxyl radical (and important atmospheric cleaning agent)
OMI	Ozone Monitoring Instrument (on board the Aura satellite)
OTR	Organ transplant recipients
P	Phosphorous
PAH	Polycyclic aromatic hydrocarbon(s)
PAM	Pulse amplitude modulated (fluorescence)
PAR	Photosynthetically active radiation, 400-700 nm waveband
PAUR II	Photochemical Activity and solar Ultraviolet Radiation campaign 2
pCO_2	Partial pressure of carbon dioxide
PEC	Predicted environmental concentration
Pg	Peta gram (1x1012 grams)
PHR1	The gene encoding CPD photolyase
PNEC	Predicted no effect concentration
POC	Particulate organic carbon
POM	Particulate organic matter
PR	Pathogenesis-related proteins
PSC	Posterior subcapsular cataract(s)

PSC	Polar stratospheric cloud (ice crystals which form at high altitudes in Polar regions when the temperature is below a critical threshold)
Ptc	Murine patch protein (gene in italics)
PTCH	Human patch protein (gene in italics)
QBO	Quasi biennial oscillation (a shift in wind patterns - especially over the tropics - with a period of approximately 2.2 years)
RA	Rheumatoid arthritis
RAF	Radiation amplification factor (a measure of sensitivity to ozone change)
ROS	Reactive oxygen species (·OH, for example)
RT	Radiative transfer
SAGE	Stratospheric Aerosol and Gas Experiment, a satellite-based instrument
SCC	Squamous cell carcinoma
SCC	Squamous cell carcinoma
SH	Southern Hemisphere
SZA	Solar zenith angle (i.e. the angle between zenith and the centre of the solar disk)
TFA	Trifluoroacetic acid
Th1	T-helper 1
Th2	T-helper 2
TOMS	Total Ozone Mapping Spectrometer, a satellite-based instrument
Treg cell	T-regulatory cell
Troposphere	Lowest part of the earth's atmosphere (0-16 km)
UCA	Urocanic acid
UV	Ultraviolet. Wavelengths from 100 nm to 400 nm. Ozone and other atmospheric gases progressively absorb more and more of the radiation at wavelengths less than 320 nm. Only those greater than 290 nm are transmitted to the Earth's surface
UV index	A standardised unit for providing UV information to the public
UV-A	Electromagnetic radiation of wavelengths in the 315 to 400 nm range
UV-B	Wavelength range 280-315 nm, as defined by CIE
UV-B	Electromagnetic radiation of wavelengths in the 280 to 315 nm range
UV-C	Electromagnetic radiation of wavelengths in the 100 to 280 nm range

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UVEry	Erythemally-weighted UV irradiance, where the irradiance is weighted by the erythemal action spectrum.
UVI	A standard scale for reporting UV irradiance to the public. The UVI is a unitless number which is 40 times the erythemally-weighted irradiance, measured in units of W m^{-2}.
UVR	Ultraviolet radiation
UVR8	UV-regulatory protein
VDR	Vitamin d receptor
VOC	Volatile organic compound (s)
WMO	World Meteorological Organization
WOUDC	World Ozone and UV Data Centre
XP	Xeroderma pigmentosum

Environmental Effects of Ozone Depletion: 2006 Assessment

Interactions of Ozone Depletion and Climate Change

Executive Summary

Ozone and UV Changes

- **The Montreal Protocol is working. The concentrations of ozone depleting substances in the atmosphere are now decreasing.** Outside Polar Regions, the decline of ozone seen in the 1980s and 1990s has not continued. In Polar Regions, there is much higher variability. Each spring, large ozone holes continue to develop in Antarctica and less severe regions of depleted ozone continue to develop in the Arctic. There is evidence that some of these changes are driven by changes in atmospheric circulation rather than being solely attributable to reductions in ozone-depleting substances, which may indicate a linkage to climate change. Global ozone is still less than in the 1970s. Changes in ozone directly influence UV-B radiation, so elevated UV-B radiation due to reduced ozone is expected to continue.

- **The future evolution of atmospheric ozone remains uncertain. It is expected to increase slowly in the decades ahead, but it is not known whether it will return to higher, similar, or lower levels than those prior to the onset of ozone depletion.** Current chemical models are unable to reproduce accurately all of the observed ozone variability, the rates of future increases in greenhouse gases are not yet established, and interactions between ozone depletion and climate change are not yet fully understood. Current models predict that ozone will have recovered from the effects of man-made ozone-depleting gases by mid-century at mid-latitudes, and about 1-2 decades later at polar latitudes.

- **Long term responses in UV-B radiation caused by ozone changes have been observed.** Increases in UV-B irradiance have occurred over the period of ozone depletion. At unpolluted sites in the Southern Hemisphere, there is some evidence that UV-B irradiance has diminished since the late 1990s. Because of improvements in the availability and temporal extent of UV data we are now able to evaluate the changes in recent times compared with those estimated since the late 1920s, when ozone measurements first became available. The increases in UV-B radiation from about 1980 to the end of the 20th century have been larger than the long-term natural variability.

- **The effects of aerosols and air pollutants on long-term variations in UV-B irradiance may be comparable with those due to changes in ozone.** At some sites in the Northern Hemisphere, UV-B radiation may continue increasing because of the continuing reductions in the attenuation by aerosols since the 1990s despite the cessation of ozone depletion.

- **Interactions between ozone depletion and climate change are complex and can be mediated through changes in chemistry, radiation, and atmospheric circulation patterns.** The changes are in both directions: ozone changes affect climate, and climate changes affect ozone. Contrary to what was predicted from some models in previous assessments, more recent models and the observational evidence suggest that stratospheric ozone (and therefore UV-B radiation) has responded relatively quickly to changes in ozone depleting substances, implying that climate interactions have not delayed these responses.

- **There is greater uncertainty about future surface UV-B radiation than future ozone, since UV-B radiation will be additionally influenced by climate change.** Climate change can also affect UV-B radiation through changes in cloudiness, aerosols and surface reflectivity, without involving ozone. The rate of climate change is accelerating. Temperature changes over the 21st century are likely to be about 5 times greater than in the past century. This will affect future cloud, aerosol and surface reflectivity. Consequently, unless strong mitigation measures are undertaken with respect to climate change, profound effects on the biosphere and on the solar UV radiation received at the Earth's surface can be anticipated.

Health

- **In addition to cortical cataract, nuclear cataract has been found to be associated with polar UV radiation.** Numerous studies have implicated exposure to solar UV radiation as a causative factor in the development of cortical cataract. Several reports now confirm an association between nuclear cataract and UV exposure. In addition, higher ambient temperatures may increase the risk of nuclear cataract development. In contrast, there is insufficient evidence to infer a causative role for solar UV radiation in the induction of posterior subcapsular cataract.

- **Exposure to sunlight is a significant risk factor for pterygium on the surface of the eye.** Pterygium is an inflammatory, proliferative and invasive lesion of the human cornea that can severely impair vision. It is induced, in part, by the intracellular damage caused by UV-B exposure. Genetic factors and the degree of long-term exposure to sunlight are important parameters for the development of pterygia in populations of all skin colours.

- **Adverse photobiological effects of UV radiation on the eye can be enhanced by the presence of clouds and are thus affected by climate change.** Although direct sunlight does not play a major role in acute solar photokeratitis, sunburn of the eye, or in cataract formation, scattered and reflected UV-B radiation contribute to these disorders. Under conditions of cloud cover and with lower light levels, the natural defence mechanisms of the eye are relaxed, permitting greater exposure of the anterior surface of the eye and its internal structures. At the same time, the effective UV-B exposure of the eye can be increased during cloud cover due to scatter.

- **The incidence of squamous cell carcinoma (SCC), basal cell carcinoma (BCC) and melanoma continues to rise.** Approximate doublings in the incidence of all three types of skin cancer have been projected in the Netherlands for the years 2000 to 2015 and in many other countries with predominantly fair-skinned populations. The major increase in melanoma incidence has been for thin (early) melanomas that have high survival rates. In children, the incidence of melanoma is still rising and has been positively correlated with environmental UV radiation exposure.

- **Susceptibility to skin cancer is increasingly recognised as being linked with subtle variations in genes that code for proteins involved in prevention and repair of DNA**

damage. Such proteins function in defensive mechanisms that are crucial to the prevention of skin cancers. The relevance of certain gene variations differ between skin cancer types and these variations provide clues regarding the types of DNA damage and repair that are important in each of the skin cancer types. Thus, there is a wide range in the occult genetically determined susceptibility in a population. In the future, gene profiling may accurately identify high-risk individuals.

- **UV-induced immunosuppression is a crucial factor in the generation of skin cancers. In some subjects, this immunomodulation may lead to viral reactivation and a reduction in vaccine efficacy.** The lack of repair of UV-induced DNA changes decreases the resistance to skin cancers and is a significant factor in the generation of such tumours. By effects both on the virus itself and on suppression of immunity, solar UVR exposure can induce the reactivation of latent herpes simplex virus leading to the re-emergence of cold sores. The virus is a co-factor in the development of some skin cancers and conjunctival squamous cell carcinomas in association with human papillomavirus infection. Limited evidence indicates that UV radiation exposure can reduce the efficacy of vaccination, at least in genetically predisposed individuals.

- **Vitamin D, formed by exposure of the skin to UV-B (with subsequent hydroxylation to the active vitamin), may play a protective role against the development of several internal cancers, autoimmune and some other diseases.** A number of studies link low solar UV exposure with a higher risk of some internal cancers, such as colorectal and prostate, and autoimmune disease, such as multiple sclerosis and type 1 diabetes. As lack of exposure to the UV-B in sunlight leads to suboptimal vitamin D levels, vitamin D has been proposed as the protective factor in helping to prevent these diseases. The evidence to support the protective role of solar UV-B exposure and whether this is mediated through vitamin D is not definitive.

- **Personal strategies to protect the eye and skin from the adverse effects of high solar UVR exposure are being adopted increasingly by the general public.** Health campaigns in several countries such as Australia, Canada, UK, and USA have raised the awareness of the general public regarding protection from the sun. Broad-spectrum sunscreens, in widespread use in mid-latitudes by fair-skinned individuals, minimise the erythemal effects of high sun exposure. UV-absorbing soft contact lenses covering the entire cornea provide excellent protection from solar UV-B for the eye, and are superior to some tinted sunglasses as the soft contact lenses shield against UV radiation entering from the side or below.

- **It is not feasible to give a single recommendation for optimal solar UV-B exposure to allow sufficient vitamin D synthesis while not increasing the risk of skin cancer.** The solar UV-B dose experienced by an individual varies greatly depending on time of the day, latitude, altitude, season of the year, cloud cover, activity and type of clothing worn. Skin colour, age and genetic background are other critical factors in determining the positive or negative outcome of the exposure. Therefore the message regarding "safe" sun exposure depends on the individual and place of residence.

- **The interaction between ozone depletion and global climate change may adversely affect human health.** At present, it is impossible to predict how global warming might alter the behaviour of people, especially those living in mid-latitudes, with respect to the amount of time spent outdoors in sunlight. If temperatures rise, then personal solar UV radiation exposure might be greater than at present. This would then have detrimental effects on the incidence of skin cancer and cataract and on the immune system, although benefiting vitamin D status.

Terrestrial Ecosystems

- **Field studies, in which solar UV-B radiation is either augmented or attenuated, report many effects on higher plants and on bacteria, fungi and other microbes.** Although photosynthesis of higher plants and mosses is seldom affected in field studies by UV-B radiation, growth and morphology (form) of higher plants and mosses are often changed. This can lead to small reductions in shoot growth and changes in the competitive balance among species. Fungi and bacteria are generally more sensitive to damage by UV-B radiation than are higher plants. However, the species differ in their UV-B sensitivity to damage. This can lead to changes in species composition of microbial communities with subsequent influences on processes such as litter decomposition. Changes in plant chemical composition are commonly reported from experiments using enhancement or attenuation of UV-B radiation in sunlight.

- **Enhanced UV-B often leads to substantial reductions in consumption of plant tissues by insects.** In some cases this is because of altered insect behaviour, but changes in plant chemical and physical characteristics induced by UV-B radiation usually account for the reduced herbivory. Such modifications affect many interactions of plants with other organisms, both above and below ground. More is now understood about the mechanisms of these interactions.

- **Although sunlight does not penetrate significantly into soils, the biomass and morphology of plant root systems can be affected to a much greater degree than plant shoots.** Root mass can exhibit large declines with enhanced UV-B radiation. Also, UV-B-induced changes in soil microbial communities and biomass, as well as altered populations of small invertebrates have been reported and these changes have important implications for processing of mineral nutrients in the soil. Many of these ecosystem-level phenomena appear to be the result of systemic changes in chemical and physical properties of plants and in the nature of root exudates.

- **UV-B radiation and other environmental factors that are undergoing changes such as temperature, CO_2, moisture and available nitrogen over large areas may interact to produce a complex plant response.** In several studies, plant growth was augmented by higher CO2 levels, while on the other hand many of the effects of UV-B radiation were usually not ameliorated by the elevated CO_2. UV-B radiation often increases both plant frost tolerance and survival under extreme high temperature conditions. Conversely, extreme temperatures sometimes influence the UV-B sensitivity of plants. Plants that are drought tolerant are likely to be more tolerant of high UV-B flux. Furthermore, UV-B radiation has been reported to alleviate some symptoms of water stress. Biologically available nitrogen is exceeding historical levels in many regions due to human activities. Studies show that plants well supplied with nitrogen are generally more sensitive to UV-B radiation.

- **Many new developments in understanding the underlying mechanisms mediating plant response to UV-B radiation have emerged.** UV-B radiation results in an activation of as yet uncharacterised receptor molecules. These initial events engage signalling pathways that result in altered plant gene expression and response. Exposure to UV-B induces some signals that are UV-B-specific and some that have elements in common with those elicited by other environmental factors. The use of shared signalling elements generates overlapping patterns of gene expression and functional responses. This new information is helpful in understanding common responses of plants to UV-B radiation, such as diminished growth, acclimation to elevated UV radiation, and interactions of plants with plant consumer organisms. It also helps in interpreting the interaction of various environmental stresses on plant growth and function.

- **Technical issues concerning the use of biological spectral weighting functions (BSWFs) have been further elucidated.** The BSWFs are multiplication factors assigned to different wavelengths giving an indication of their relative biological effectiveness. They are critical to the proper conduct and interpretation of experiments in which organisms are exposed to UV radiation, both in the field and in controlled environment facilities. The characteristics of BSWFs vary considerably among different plant processes, such as growth, DNA damage, oxidative damage and induction of changes in secondary chemicals. Thus, use of a single BSWF for plant or ecosystem responses is not appropriate.

Aquatic Ecosystems

- **Recent field studies continue to show that even current solar UV-B radiation can adversely affect aquatic organisms.** Reductions in productivity and impaired reproduction and development have been shown for phytoplankton, fish eggs and larvae, zooplankton and other primary and secondary consumers exposed to UV-B radiation. UV-B-related decreases in biomass productivity can be transferred through all levels of the food web, as well as cause changes in species composition and structure and function of ecosystems. Decreases in primary production would result in reduced sink capacity for atmospheric carbon dioxide, with its related effect on climate change.

- **Experiments in large enclosures show that changes in community structure may be more ecologically important than effects of enhanced UV-B on overall algal biomass.** These mesocosm experiments allow the experimenter to control the level of UV radiation on plankton communities to simulate various levels of ozone depletion. Growth was inhibited by ambient UV radiation in fixed-depth experiments but not in mesocosms where vertical mixing exposed planktonic organisms to variable radiation regimes. A synthesis model simulating mesocosm experiments suggests that enhanced UV-B could cause a shift from primary producers to bacteria at the community level. Shifts in community structure could have important consequences for carbon dioxide concentration in oceanic surface waters.

- **Recent studies have expanded our understanding of UV-B protection mechanisms for aquatic organisms.** UV radiation impairs photosynthesis, nitrogen fixation and damage DNA, but most phytoplankton have developed mitigating measures including UV-absorbing substances, repair enzymes and reactive oxygen species scavenging systems. However, protection is not complete. Picoplankton cyanobacteria do not produce absorbing substances but rely on fast cell division; these organisms have recently been found to be ubiquitous and to contribute more than 50 % to the productivity in aquatic habitats. Solar UV controls the vertical position of macroalgae in the tidal zone. Organisms in the upper tidal zone have developed effective screening and repair mechanisms.

- **UV-B-related decreases in primary-producer biomass have a negative effect on the growth and survival of consumers, which form the higher levels in the aquatic food web.** Specific, direct UV B effects have been identified in a wide variety of consumers, including copepods and other zooplankton, corals and sea urchins.

- **In their natural habitat, zooplankton face conflicting selection pressures, including exposure to UV-B radiation and factors of global climate change.** Invertebrate predators cause an upward movement of the zooplankton during daylight hours, exposing them to high levels of UV radiation at the surface. Besides vertical migration and UV screening, zooplankton rely on photorepair of UV-B-induced DNA damage. Increases in water temperature resulting from climate change are expected to increase enzymatic activity, which would enhance photorepair.

- **Primary causes for a decline in fish populations are predation and poor food supply for larvae; however, exposure of the larvae to enhanced UV-B radiation may further contribute to this decline.** Other major factors are overfishing, increased water temperature due to global climate change, pollution, and disease. Imprecisely defined habitat characteristics and the naturally high mortality rates of fish larvae render quantitative assessment of specific UV-B effects difficult.

- **The concentration and chemical composition of dissolved organic matter in aquatic ecosystems govern the penetration of UV radiation in the water column.** UV radiation affects the species composition of plankton communities and thus the concentration of DOM. There is a strong link between early succession of zooplankton communities and terrestrial plant communities within watersheds, which in turn are affected by climate change. Consequently, climate change and UV radiation have the potential to affect species composition in lakes and also to increase the invasion potential by imported species.

Biogeochemical Cycles

- **Climate-related changes can alter the transfer of organic matter from terrestrial to freshwater and coastal ecosystems and thereby influence UV radiation penetration into water bodies, with major consequences for aquatic biogeochemical processes.** These changes are particularly prevalent in high latitude systems. Dissolved organic matter leaching from or running off terrestrial ecosystems enters streams, rivers, lakes and, ultimately the oceans. The coloured part of dissolved organic matter controls the penetration of UV radiation into water bodies, but is also photodegraded by solar UV to release small inorganic molecules, mainly CO_2.

- **Future increases in the temperature of surface waters will enhance stratification of lakes and the ocean, which will intensify effects of UV-B radiation on biogeochemistry in the surface layer.** This important effect is manifested by the extensive increase in transparency of the water to UV-B radiation in the upper layer of stratified aquatic environments. These effects of climate change increase the impacts of UV-B radiation on biogeochemical cycles in the upper layer of aquatic systems, thus partially offsetting the beneficial effects of an ozone recovery.

- **Climate change and changes in UV-B radiation influence the concentration of halogen-containing compounds that are involved in ozone chemistry in the atmosphere.** Emissions of halogen-containing compounds, for example, methyl bromide from higher plants, increase with increasing air temperature. Recent observations indicate that methyl bromide concentrations in the atmosphere are decreasing at a rate of 2.5 – 3.0 % per year but future global warming may reduce the current rate of decline. Bromine and other halogen radicals are also generated in UV-B radiation induced reactions of halogen-containing compounds both in atmospheric aerosols present in the marine boundary layer and in surface waters. These halogen-containing compounds may be transported by convection to the upper troposphere where the bromine radical participates in ozone destruction.

- **UV-B can alter the biological availability and toxicity of metals in aquatic environments.** Although many trace metals are essential trace nutrients, all metals are toxic above a certain concentration. In sunlit surface waters, however, they often exist in forms that are biologically not available. Increased UV-B can alter the chemical form of metals to produce forms that are available to aquatic organisms. For example, the UV-induced oxidation of elemental mercury results in the formation of precursors to methyl mercury that can adversely affect human health through bioaccumulation in aquatic food webs.

- **UV radiation drives photoreactions involved in cycling of marine sulphur, leading to the production of atmospheric aerosols and cloud formation.** Oceanic emissions of dimethylsulphide (DMS) produce atmospheric aerosols that influence atmospheric radiation and temperature. UV radiation induced transformation is an important sink of DMS in the upper ocean. Carbonyl sulphide, another important sulphur compound in the upper ocean, is produced in UV-B radiation induced reactions involving chromophoric DOM.

- **In terrestrial systems UV-B radiation can affect cycling of carbon and nutrients through changes in decomposition and soil biology.** Exposure to solar UV-B radiation causes direct photodegradation of dead plant material, especially in arid climates. When plants are exposed to UV-B radiation, changes in plant root exudation and/or the chemistry of dead plant material influence soil organisms and biogeochemistry. Changes in carbon and nutrient cycling induced by UV-B radiation can interact with responses to climate change and so may influence long-term ecosystem carbon budgets.

Air Quality

- **Models and measurements suggest that ozone transport from the stratosphere to the troposphere may have decreased by approximately 30% in the last 30 years.** Ozone concentrations near the ground are a key indicator of air quality. Tropospheric ozone concentrations are affected by UV-B radiation, local weather systems, and pollutant concentrations. Stratospheric ozone depletion has increased the rate of ozone production in the troposphere due to enhanced UV-B radiation but reduced the amount of ozone transported from the stratosphere to the troposphere.

- **The predicted future increase in stratospheric ozone may increase tropospheric temperature and concentrations of ozone in the atmospheric boundary layer.** Models predict that ozone concentrations in the atmospheric boundary layer will increase globally by 33 to 100% during the period 2000 to 2100 due to the combined effects of climate change, atmospheric pollution, and increases in stratospheric ozone. The impact of this increase on climate is difficult to quantify as tropospheric ozone concentrations are very variable, both in space and time.

- **Changes in the concentration of tropospheric hydroxyl radical caused by changes in UV-B radiation are now much better quantified**. Tropospheric hydroxyl radical (OH) is one of the major oxidizing agents in the atmosphere, destroying trace gases that are involved in ozone depletion, climate change, and urban air pollution. The globally averaged OH has been observed to change on short time scales (months – years) but not in the longer term. Recent measurements in a relatively clean location over 5 years showed that OH concentrations can be predicted by the intensity of solar ultraviolet radiation. If this relationship is confirmed by further observations, this approach could be used to characterize the oxidation efficiency of the troposphere in different chemical regimes using UV radiation measurements, thus simplifying assessment of air quality.

- **Confidence in models estimating the impact of ozone change on the oxidation capacity of the atmosphere has improved for unpolluted locations**. Measurements of UV radiation and chemical composition, including OH in the lower atmosphere, now normally agree with chemical models to within the measurement accuracy in unpolluted air both for clear skies and uniform cloud cover. However, in moderately and heavily polluted urban regions or forested environments, models and measurements disagree. These model uncertainties underline the importance of local measurements of tropospheric ozone, especially in areas where air may be polluted.

- **An analysis of surface-level ozone measurements in Antarctica suggests that there has been a significant change in the chemistry of the atmospheric boundary layer in this region as a result of stratospheric ozone depletion**. Measurements of ozone concentrations in the atmospheric boundary layer show a recent (since 1990) increase in surface ozone concentrations consistent with more UV radiation reaching the earth's surface during ozone hole episodes, and the enhanced production of nitrogen oxides from the ice. Thus, the Antarctic lower atmosphere is estimated to be more oxidizing now than before the development of the ozone hole, which may have adverse consequences through changing bioavailability of metals.

- **The tropospheric concentration of HFC-134a, a potent greenhouse gas and the main known anthropogenic source of trifluoroacetic acid, is increasing rapidly**. The increase is in agreement with the known usage and atmospheric loss processes. Observations in both hemispheres between 1998 and 2002 show that the concentration of HFC-134a has been increasing by up to 12% per year. The good agreement between observations and known sources and sinks gives increased confidence in predictions of the environmental build-up of trifluoroacetic acid. The increasing concentration of HFC-134a may contribute to an acceleration of climate change.

- **Risks to humans and the environment from substances produced by atmospheric degradation of hydrochlorofluorocarbons (HCFCs) and hydrofluorocarbons (HFCs) are considered minimal**. These include trifluoroacetic acid (TFA) and chlorodifluoroacetic acid. Recent studies reinforce the conclusion of small environmental and human health risks from current environmental loadings in fresh- and salt-water. Although the amounts of these compounds are expected to continue to increase in the future because of climate change and continued use of HCFCs and HFCs, current information suggests that this is not an issue of great importance.

- **Perfluoropolyethers, substances proposed as HCFC substitutes, have very large global warming potential and show great stability to chemical degradation in the atmosphere**. These compounds are commonly used as industrial heat transfer fluids. It is not known whether these substances will contribute significantly to global warming and its interaction with ozone depletion. Their risks should be further evaluated.

Materials Damage

- **Plastics and wood exposed to solar UV radiation undergo degradation losing their useful properties over a period of time.** This damage is dose-dependent and limits the outdoor lifetimes of most materials. The damage is exacerbated by higher ambient temperatures, higher humidity levels, and atmospheric pollutants. Light stabilizers and surface coatings are generally used to control the solar-UV induced damage to materials. Higher UV levels will require higher levels of stabilizers resulting in higher cost of materials used outdoors.

- **Several novel UV stabilizers and product fabrication techniques that improve UV-resistance have been reported.** New variants of effective light stabilizers, such as stabilizer compounds that bind to the polymer and are therefore less likely to be lost by leaching, have been reported recently. Mechanisms of synergistic effects of stabilizer blends have been further elucidated and will contribute to the design of new light-stabilizer blends. Continued research on this topic will facilitate the development of strategies that are better able to protect materials exposed to solar UV-B radiation.

- **An emerging trend towards the use of nanoscale fillers may improve the UV stability of plastics formulations.** These nanoscale fillers have smaller average particle sizes and often yield better mechanical properties than conventional fillers. Initial data suggest some of the nanoscale fillers may also act as good light stabilizers and extend the service life of products exposed to outdoor UV radiation. However, potential interference of these fillers with the effects of conventional light stabilizers or other additives such as antioxidants or flame retardants has not yet been fully evaluated.

- **Using powdered wood as a filler in plastics is continuing to be explored, and the effect of these fillers on UV-stability depends on the type of wood.** Powdered wood and other plant materials are used as low-cost natural fillers in some plastics products intended for outdoor use. Recent research indicates that several of these plant-derived fillers can either enhance the photodamage or act as a photostabilizer for the plastic material, depending on the source of the natural filler material and processing method used with the material. However, the lignin content in wood filler absorbs solar UV-B radiation and promotes photodamage of the polymer component. Identifying sources and processing technologies for these bio-based fillers without compromising light stability of filled polymers can lead to low-cost UV-stable plastics products for certain outdoor applications.

Chapter 1. Changes in biologically active ultraviolet radiation reaching the Earth's surface

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Summary

The Montreal Protocol is working. Concentrations of major ozone-depleting substances in the atmosphere are now decreasing, and the decline in total column amounts seen in the 1980s and 1990s at mid-latitudes has not continued. In polar regions, there is much greater natural variability. Each spring, large ozone holes continue to occur in Antarctica and less severe regions of depleted ozone continue to occur in the Arctic. There is evidence that some of these changes are driven by changes in atmospheric circulation rather than being solely attributable to reductions in ozone-depleting substances, which may indicate a linkage to climate change. Global ozone is still lower than in the 1970s and a return to that state is not expected for several decades. As changes in ozone impinge directly on UV radiation, elevated UV radiation due to reduced ozone is expected to continue over that period.

Long-term changes in UV-B due to ozone depletion are difficult to verify through direct measurement, but there is strong evidence that UV-B irradiance increased over the period of ozone depletion. At unpolluted sites in the southern hemisphere, there is some evidence that UV-B irradiance has diminished since the late 1990s. The availability and temporal extent of UV data have improved, and we are now able to evaluate the changes in recent times compared with those estimated since the late 1920s, when ozone measurements first became available. The increases in UV-B irradiance over the latter part of the 20th century have been larger than the natural variability.

There is increased evidence that aerosols have a larger effect on surface UV-B radiation than previously thought. At some sites in the Northern Hemisphere, UV-B irradiance may continue to increase because of continuing reductions in aerosol extinctions since the 1990s.

Interactions between ozone depletion and climate change are complex and can be mediated through changes in chemistry, radiation, and atmospheric circulation patterns. The changes can be in both directions: ozone changes can affect climate, and climate change can affect ozone. The observational evidence suggests that stratospheric ozone (and therefore UV-B) has responded relatively quickly to changes in ozone depleting substances, implying that climate interactions have not delayed this process. Model calculations predict that at mid-latitudes a return of ozone to pre-1980 levels is expected by mid 21st century. However, it may take a decade or two longer in polar regions. Climate change can also affect UV radiation through changes in cloudiness and albedo, without involving ozone and since tempera-

ture changes over the 21st century are likely to be about 5 times greater than in the past century. This is likely to have significant effects on future cloud, aerosol and surface reflectivity. Consequently, unless strong mitigation measures are undertaken with respect to climate change, profound effects on the biosphere and on the solar UV radiation received at the Earth's surface can be anticipated.

The future remains uncertain. Ozone is expected to increase slowly over the decades ahead, but it is not known whether ozone will return to higher levels, or lower levels, than those present prior to the onset of ozone depletion in the 1970s. There is even greater uncertainty about future UV radiation, since it will be additionally influenced by changes in aerosols and clouds.

Introduction

UV-B radiation (280-315 nm) has important influences on biological processes, and is strongly absorbed by atmospheric ozone (in both the stratosphere and the lower atmosphere). Reductions in stratospheric ozone are therefore important because of the corresponding increases in UV-B radiation reaching the Earth's surface. While some UV radiation is needed to synthesize vitamin D, which is necessary for human health, increases in UV-B radiation are also harmful for human health, for example, for melanoma and other health effects (see Chapter 2). Increases in UV-B radiation also increase damage to a wide range of organic molecules, including DNA molecules, and generally lead to increased harm to a diverse range of biological (see Chapters 3-5), and physical (see Chapters 6-7) processes. UV-B radiation is influenced by many factors other than ozone. These include changes in clouds, aerosols, air pollution, and surface reflection, all of which are influenced by climate change. Since the publication of the 2002 UNEP Effects Panel Assessment[1, 2] there has been continuing progress in research to understand the causes and effects of ozone change. There have been no changes in our understanding of basic principles since the previous assessment, but there have been significant improvements in our knowledge of past ozone and UV radiation, which put recent changes into a better historical context. Here we assess this new knowledge of ozone changes, their effects on UV radiation, and the interactions between ozone depletion and climate change.

Generally, the damaging effect of UV radiation increases towards shorter wavelengths. In this paper we focus on the erythemally-weighted UV radiation (UV_{Ery}),[3] for which a 1% reduction in ozone leads to a 1.2% increase in damaging radiation at high sun elevations.[4] However, other weighting functions with different sensitivities to ozone are more appropriate for other processes.[4] In some cases, the wavelength dependence of the effects is not yet well quantified. The continued availability of spectral measurements of solar radiation will therefore be crucial to quantifying these effects.

Ozone changes

The science of ozone depletion has recently been assessed by the WMO Scientific Assessment Panel.[5] In recent years, the geographic and seasonal extent of Antarctic ozone depletion has varied greatly from year to year (Figure 1-1), depending on the prevailing meteorological conditions. The springtime stratospheric ozone "hole" is expected to recur over the next decades, and there will continue to be a large year-to-year variability in its severity and environmental impact.

Ozone depletion is less severe in the Arctic, where there is also very large year-to-year variability (Figure 1-1), which is expected to continue, depending on the minimum temperatures reached. With global climate change, temperatures in the Arctic stratosphere are expected to continue to decrease, increasing the likelihood of severe ozone depletion due to heterogeneous chemistry on the surfaces of polar stratospheric clouds. For every degree of stratospheric cooling, a reduction in ozone of 15 Dobson Units can be expected.[6] This sensitivity is three times larger than had been estimated previously from model calculations. Therefore, future polar ozone depletion may be more susceptible to climate change than previous models had suggested.

Ozone depletion in polar regions has an impact on the ozone depletion at mid latitudes.[7-9] For example, it has been shown that approximately 50% of the ozone depletion at mid-southern latitudes is attributable to the export of ozone-poor air from Antarctica.[8]

Since the previous assessment,[1, 2] there has been increased evidence for a cessation of ozone reductions at mid-latitudes. A statistical analysis of satellite-derived ozone profiles indicated that the rate of ozone loss in the upper stratosphere (at 35-45 km altitude) has diminished globally, and that these changes are consistent with changes in total stratospheric chlorine.[11] In a follow-up study it was shown that the slow-down extended to the lower stratosphere.[12]

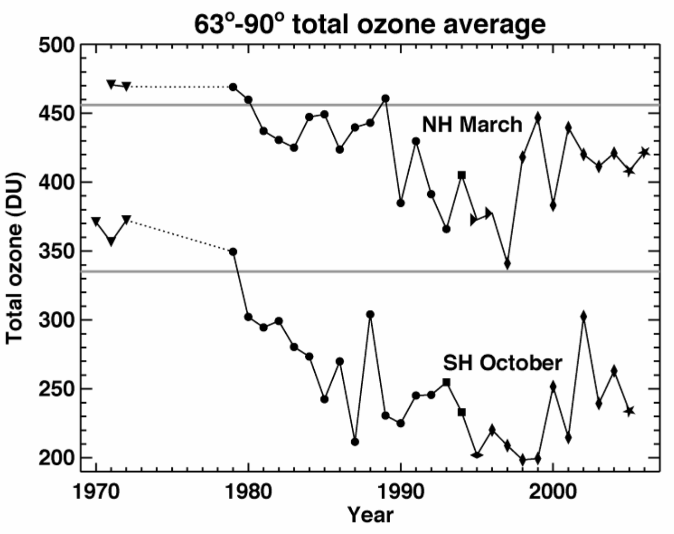

Figure 1-1. Total average column ozone poleward of latitude 63° in the springtime of each hemisphere (March for the Northern Hemisphere (NH) and October for the Southern Hemisphere (SH)), in Dobson Units (DU), based on data from various satellite instruments. The horizontal lines represent the average total ozone for the years prior to 1983 for the NH and SH. Based on Fig 4-7 in,[5] updated from.[10]

It had been predicted that detection of any recovery in the total column of ozone, which is most relevant for environmental impacts of UV radiation at the Earth's surface, would not be expected for several years,[13, 14] because of the natural inter-annual variation in several contributing dynamical factors,[15-17] which recent studies have shown may be larger than previously assumed.[18-20]

Statistical trend analyses have demonstrated that the total column amount of ozone at mid-latitudes reached a minimum in the late 1990s, and since then may have started to increase.[21] At mid-latitudes, the column amount of ozone in the 2002-2005 period was approximately 3% below 1980 levels in the Northern Hemisphere, and 6% below 1980 levels in the Southern Hemisphere,[5] which is similar to that in the period ending 1998-2001.[10, 22]

The observed changes in ozone are broadly consistent with those expected from changes in atmospheric chemistry that would result from the actions mandated by the Montreal Protocol. Changes in observed ozone are compared with estimated changes in effective equivalent stratospheric chlorine in Figure 1-2. However, changes in atmospheric circulation were also found to have a substantial influence on ozone variability, especially in the lower stratosphere,[23, 24] and have contributed to a significant portion of the observed increase in ozone in the Northern Hemisphere in recent years. These dynamical changes may be a consequence of climate change. If that is the case, these interactions may be important considerations for predicting future changes in ozone.

The future evolution of atmospheric ozone remains uncertain, firstly because current chemical models are unable to reproduce accurately all of the observed ozone variability,[5] secondly because the rates of future increases in greenhouse gases are not yet established,[25] and thirdly because interactions between ozone depletion and climate change are not yet fully understood, as discussed later in this document. A full recovery to ozone amounts present in the 1970s prior to the onset of ozone depletion is not expected for several decades at best.

Factors affecting UV radiation received at the Earth's surface

Solar ultraviolet radiation is attenuated by interaction with atmospheric constituents in the Earth's atmosphere through absorption and scattering processes. Furthermore, its variability is controlled by astronomical factors, including those that determine the angle at which solar radiation arrives at the Earth's atmosphere and the irradiance that is distributed per unit area at the top of the atmosphere. These astronomical factors and their roles are known very accurately.

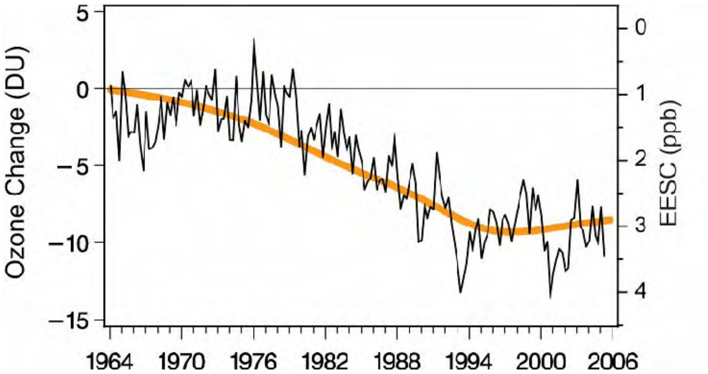

Figure 1-2. Area-weighted total ozone deviations for 60°S-60°N, estimated from ground-based measurements. Data have been deseasonalised and adjusted to remove the effects of solar-cycle and the quasi-biennial oscillation (QBO). The thick line represents the effective equivalent stratospheric chlorine (EESC), scaled to fit the ozone data from 1964-2005 (scale on right). Adapted from Fig 3-1 in.[5]

Absorption in the UV-B region is due to ozone primarily, to a lesser extent to SO_2, NO_2 and other minor species, and also to aerosols. Scattering occurs on molecules, aerosols, and clouds. Solar UV radiation is reflected by the clouds and the Earth's surface (land and water). All these processes have been extensively investigated. Although the physical interactions are well understood, the quantitative description is incomplete. Because of their complexity, the effects of clouds in particular remain difficult to calculate. As discussed later, clouds, aerosols, and surface albedo are factors that are likely to be affected by climate change, and may therefore impact the long-term variability of surface UV radiation. The effects of ozone on UV radiation are well understood. Since the previous assessment,[1, 2] new studies have quantified the effects of other factors on UV radiation and the related uncertainties, as discussed below.

Clouds constitute by far the most important factor controlling UV radiation for any given solar elevation angle, (e.g., [26]) and introduce a high variability in surface UV irradiance that limits the detection of influences from ozone.[27] Although clouds mainly attenuate radiation, new experimental data have confirmed results from previous studies by reporting large enhancements of UV radiation under partly cloudy skies.[28-31] Enhancements up to 40% have been observed at 420 nm (blue light) when the solar disk is visible, but are smaller in the UV-B region.[32, 33] The biological importance of enhancements of UV radiation under broken clouds, which may at times be sustained for hours, must be taken into consideration.

The most relevant radiation quantity for atmospheric chemistry is the actinic flux, which is the omni-directional flux passing through a sphere, rather than the cosine-weighted irradiance on a horizontal surface (see Chapter 6, Figure 6-2). Above clouds, localised increases of the UV actinic flux of between 60–100% have been observed.[34] Below the clouds, the actinic flux was found to be 55–65% smaller compared to clear skies. In both cases there is a direct influence on photochemistry, with consequences on tropospheric atmospheric composition. Although these effects can be adequately modelled for homogeneous and stratified clouds, it is extremely difficult to simulate broken cloud conditions adequately.

The interaction of clouds with other processes produces significant effects on radiation that either reaches the surface or is reflected to space. Increased scattering within the clouds interacts with molecular scattering, producing complex wavelength dependencies in the radiation scattered towards space and detected by satellite instruments.[35] Similarly, increased scattering by cloud droplets increases the possibility that these photons are absorbed by atmospheric constituents that are inside the cloud (e.g., ozone, NO_2, aerosols).[36, 37] A cloud layer over snow or ice covered surfaces may substantially increase the UV dose at the surface, through strengthening of multiple scattering. A twofold increase in UV radiance from the overhead sky has been observed under cloudy conditions compared with the clear sky in Antarctica, whereas the increases in the visible and in the infrared were much larger, with enhancements of up to factor of 100 in the near infrared.[38]

Aerosols and trace gases emitted near the surface of the Earth can also have large impacts on UV radiation.[39-41] Several studies have quantified aerosol radiative properties - particularly their UV absorbing efficiency - from ground based measurements of solar UV radiation.[42-48] Some satellite retrievals (e.g., TOMS) overestimate surface UV radiation by between 5 and 10% over areas with high aerosol load, especially in the presence of strongly absorbing aerosols. By combining measurements and radiative transfer (RT) modelling,[47-50] methods have been suggested for applying corrections to satellite retrievals.[51] These corrections will enable more accurate satellite estimates of UV irradiance in the future.

New evidence has shown that in urban areas, aerosols and air-pollutants such as ozone and nitrogen dioxide can significantly attenuate solar UV radiation.[40, 52-54] Recent studies have shown that aerosols and trace gases from biomass burning can penetrate into the stratosphere,[55] and consequently affect its chemistry. Increases in UV-B and UV-A solar radiation observed during the last two decades in Germany and Greece[56, 57] cannot be explained by changes in ozone amounts alone, and thus it is necessary to include diminishing influences from other factors such as pollution at these sites.[58-60]

Aerosols and trace gases that absorb UV radiation efficiently may provide protection to the ecosystem from UV radiation. On the contrary, aerosols that scatter UV radiation redistribute the incoming direct radiation to diffuse radiation, and therefore have little effect on the UV dose received at the surface. In the presence of such scattering aerosols, the total exposure to UV radiation is increased in locations shaded from direct sunlight. Moreover, changes in the distribution of surface radiation have important effects on the penetration of UV-B radiation into tree canopies (see Chapter 3) and aquatic environments (see Chapter 4).

UV irradiance variations can be affected by ozone changes caused by weather patterns. At middle latitudes, ozone variations, at time scales of 1-3 days, depend primarily on the scales of atmospheric motions related to weather systems, while ozone sources and sinks play a minor role. Several examples have been reported where dynamical mechanisms have led to the formation of short-lived episodes of extreme total ozone values.[61, 62]

Limitations in modelling UV radiation

The great complexity and heterogeneity of cloud structure cause difficulties for their accurate parameterization, despite the progress that has been recently achieved (e.g., [34, 63]). Similarly, the effects from topography, surface albedo for snow- or ice-covered terrains, have also been investigated. However, accurate representation of the actual radiation field is very difficult, requiring three-dimensional RT modelling, which is expensive and time consuming. Most

RT models do not yet account for polarization effects, which can be important for estimating the UV radiation, especially in unpolluted conditions.

Influences on satellite-derived UV radiation of aerosols and trace gases in the lower troposphere are well understood, but the implementation of procedures for eliminating these effects depends on the availability and spatial extent of additional information gathered near the surface (e.g., aerosol optical properties from measurements or from climatology).[48, 49] Information on aerosol optical properties is available at only a few sites and is derived either directly from measurements or indirectly with the aid of radiation measurements and model calculations. There is usually no information available about aerosol extinctions in the UV-B region. Tropospheric NO_2 may also affect satellite UV retrievals and retrieval of aerosol properties from ground-based radiation measurements.[64, 65] The variability of aerosol optical parameters (e.g., the single scattering albedo and phase function of the aerosols) with altitude, which are important in RT modelling, is known only from *in situ* measurements that are generally rare. New methodologies are required to derive these parameters at different altitudes by remote sensing techniques (e.g., [45]).

Air pollutants are generally efficient absorbers of UV radiation. Because they are extremely variable in time and space, their influence on the UV radiation received at the surface is important for determining the exposure of the biosphere in urban areas. Of particular importance is the role of tropospheric ozone which may increase as a consequence of stratospheric ozone reduction, which leads to increased photochemical production in the troposphere (the so called "self-healing" effect).[60]

The global distribution of UV radiation

UV data are available from satellite-borne sensors such as the new Ozone Monitoring Instrument (OMI) on NASA's Aura satellite, which supersedes the older Total Ozone Mapping Spectrometers (i.e., the TOMS instruments).[66] The retrievals of UV irradiances are from model calculations using derived ozone and cloud reflectances. Sample maps are shown in Figure 1-3, which show a strong peak in UV daily doses at the sub-solar latitudes, near 20ºN in June and near 20ºS in December. This is expected because solar elevation is a strong determinant of UV, and because ozone amounts are generally smaller in the tropics. Outside the tropics, the daily doses of UV radiation decrease markedly as one moves towards the poles, and there are large seasonal variations at mid to high latitudes. Although ozone depletion is most severe in Antarctica, the UV doses there are not particularly high during December (though still elevated substantially compared with the Arctic in June). However, in late spring, the peak UV irradiance, as well as the daily doses, can exceed even the peaks of the corresponding values observed at mid-latitudes. For example, in the spring of 1998, the daily doses of erythemally weighted UV irradiance measured by the National Science Foundation (NSF) network of spectrometers at several Antarctic stations, including the South Pole (where there are 24 hours of daylight in summer), greatly exceeded those that occurred in the summer at San Diego (see chapter 5 from[10]). The highest daily UV doses (and peak irradiance) occur in the tropics, and at high altitude sites, particularly when snow is present.

Recent ground-based measurements have confirmed that, in rural locations, the peak erythemally-weighted UV irradiance (or UVI values, where $UVI = 40 \times UV_{Ery}$ in Wm^{-2})[2, 22] are typically 40% greater in the southern hemisphere than at the corresponding latitude in the northern hemisphere.[67] Similar differences are also apparent in the mean summer UV irradiance from spectrometers. Such marked differences are not seen in UV data from satellite instruments that utilize backscattered ultraviolet radiation (e.g., TOMS, OMI) because these sensors do not adequately probe the lowermost regions of the atmosphere where aerosol and cloud extinctions are most important.[1] Previous studies have shown that altitudinal gradients in UV irradiance are largest in the lowest kilometer or two.[67, 68, 69]

Long-term changes in UV

Measured changes

The long-term variability of surface UV radiation has been studied using measurement records and model simulations of past UV irradiance based on proxy data, which are available for several decades back in time. In most cases, these proxies include total ozone, shortwave solar radiation, and cloudiness. Depending on the location, sunshine duration, snow depth, and aerosols may also be included, as discussed below.

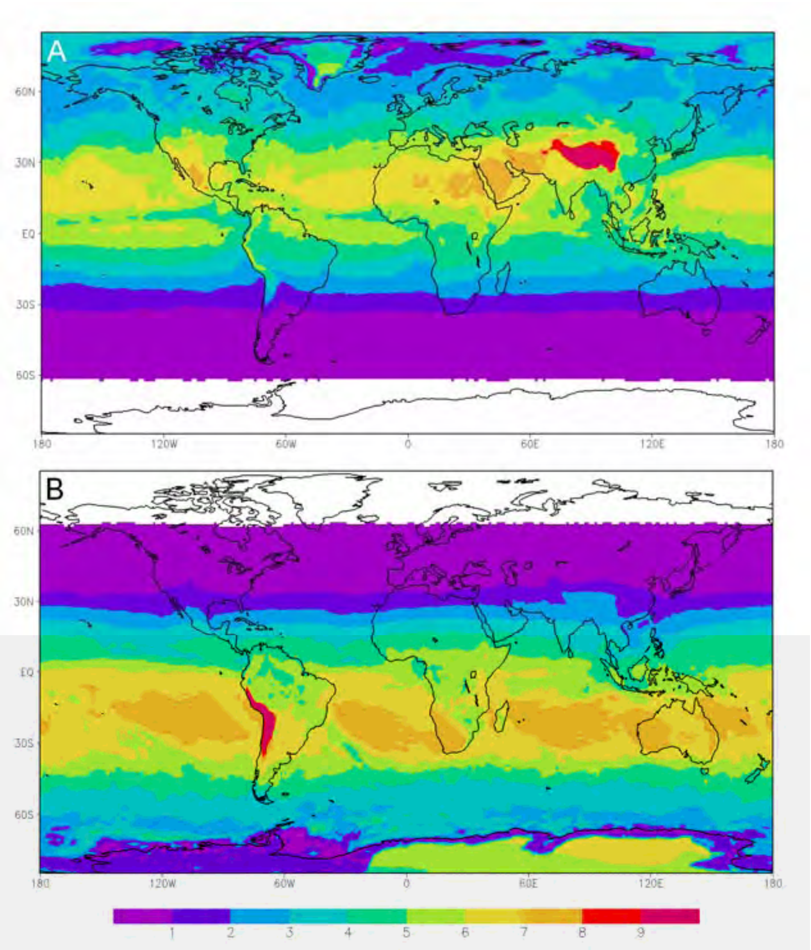

Figure 1-3. Global distribution of the average cloud corrected erythemal daily dose (in kJ m^{-2}) for June 2005 (left) and Dec 2005 (right) derived from OMI measurements. These Surface UV irradiance images were supplied by Aapo Tanskanen of the Meteorological Institute. OMI is a joint effort of KNMI, NASA, and FMI, and is managed by NIVR/Netherlands. The unshaded (white) areas are those with no UV data coverage.

Increases in surface UV irradiance in the 1990s have been observed from spectral measurements at a few stations in the northern hemisphere. This is evident, for example, in the updated record of Thessaloniki, Greece (up to end of 2005),[70, 71] at Hohenpeissenberg, Germany[72], and at Bilthoven, The Netherlands.[73] The continuous increase in surface UV irradiance, even at longer wavelengths, during the 1990s cannot be explained only by ozone depletion. It is attributed, in part, to reductions in atmospheric pollution leading to less aerosol extinctions,[74] and to decreases in cloudiness.[72] The period since the ozone stabilised at mid-latitudes is too short to reveal unequivocal changes in UV radiation. However, data since the late 1990s from one Southern Hemisphere site, where ozone does appear to have increased, indicate that surface UV radiation may be decreasing (see Figure 1-4).[75, 76] Note however that, in these data, there is an unexplained decrease around the turn of the century, which exaggerates the decreases in UVI since that time. At higher latitude southern hemisphere sites, the natural variability is generally much larger, masking any trends in UV.[77, 78]

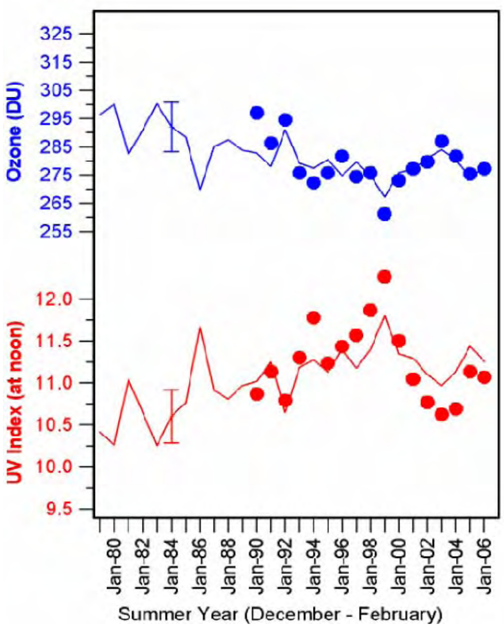

Figure 1-4. Long term changes in summertime ozone (upper panel) and in peak summertime UVI (lower panel) at Lauder, New Zealand. The symbols show the average ozone and corresponding noontime UV Index for the 5 highest UVI days in December, January and February of each summer derived from UV spectral irradiance measurements. The lines represent the average summertime ozone from satellite-derived ozone, and the corresponding UVI calculated from those ozone values.

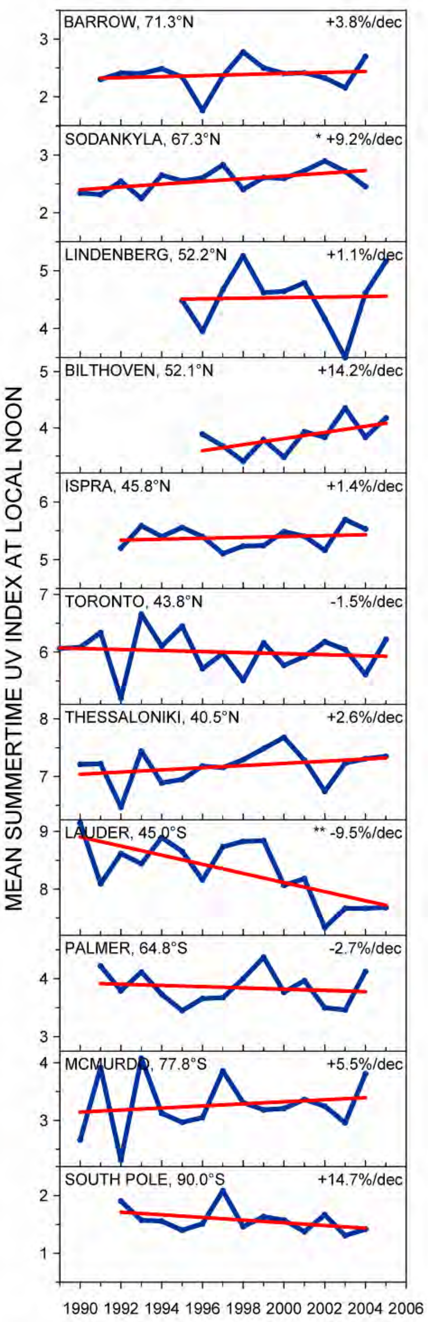

Figure 1-5. Long-term changes in UV Index averaged over the three summer months for all weather conditions within ±1 hour around local noon measured at 11 sites distributed worldwide. Linear regressions on the data (straight lines) were used to estimate the corresponding linear trends. This figure was prepared using updated series of published UV datasets. The significance level (** for 99%, and * for 95%) was calculated from the data variability only, neglecting the uncertainties in the measurements. Note that periods for the trends differ from site to site. Adapted from[5]

Examples of long-term changes in UV irradiance from high quality spectrometers are illustrated in Figure 1-5. The summertime UV irradiance generally increased over the observation period, but in some cases, especially in the Southern Hemisphere, there have been decreases in recent years. The larger gradient at Lauder can, in part, be attributed to the lower sampling frequency prior to 1994, when observations were made only during fair weather. Further, in the period between 1994 and September 1998, no data were taken during wet weather.

Long-term measurements of UV radiation with broadband radiometers were reported for Moscow, Russia (1968-2003)[53] and for Norrköping, Sweden (1983-2003),[79] which show an overall increase towards the late 1990s. Some of this increase is due to ozone reductions. However, decreases in aerosol optical thickness and effective cloud amount during this period[80, 81] have also occurred, and have led to increases in irradiance at longer wavelengths which are unaffected by ozone.[79]

Reconstructed time series of UV irradiance, extending several decades back from the start of the ozone depletion, are now available.[40, 73, 82, 83] For example, a method for estimating daily erythemal UV doses using total ozone, sunshine duration, and snow depth has been developed and applied at Sodankylä, Finland for the period 1950–1999.[84] The longest reconstructed time series of UV irradiance to date is from Davos, Switzerland, starting in 1920s when ozone measurements first became available.[85] This series demonstrates fluctuations in surface erythemal dose of similar magnitude to those observed since the late 1970s when the problem of human-induced ozone depletion began, and satellite derived ozone data became available (see Figure 1-6).

The changes deduced from reconstructed historical UV irradiance records, and from direct observations in recent decades, reflect those expected from changes in ozone and atmospheric transmission (since ozone changes are an input to the retrieval). Measurements of UV irradiance at various locations over northern mid-latitudes show that surface UV irradiance has increased in the 1980s and 1990s as a consequence of ozone depletion and increasing atmospheric transmission.[53, 58, 71, 73, 84-86]

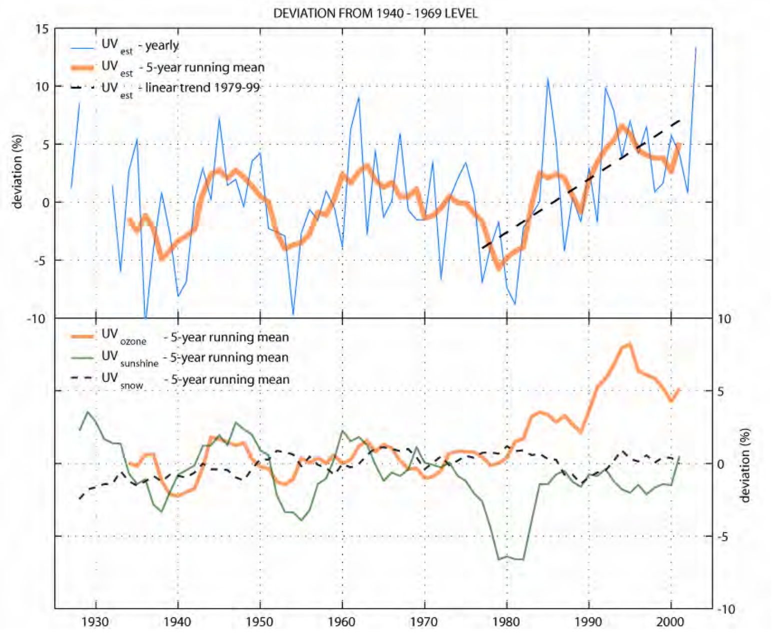

Figure 1-6. (Upper panel) Time series of reconstructed erythemal irradiance over Switzerland shown as deviation from the 1940-1969 mean. (Lower panel) Percentage contribution of ozone, sunshine duration and snow depth to the variability in UV irradiance. From [85].

Radiative transfer calculations have also been used to determine past UV irradiance from routine observations of total ozone, aerosol, albedo, and clouds at two locations in Central Europe, Würzburg, and Hohenpeissenberg.[87] The results show that, at these sites, UV-B radiation has increased over the period 1968 to 2000. Because there were supporting data records for cloud cover, aerosol and albedo in this study, the increase in UV-B irradiance could clearly be associated with a reduction in stratospheric ozone. Depending on the action spectra for specific effects, increases in the annual UV exposure are ~ +2 % to +5 % per decade. Regional differences have been found in the influence of clouds on UV radiation. For example, the UV-B irradiance increase due to ozone reduction is enhanced by clouds by about 1% per decade for Hohenpeissenberg and reduced by nearly the same amount for Würzburg.

From the above discussion it appears that UV radiation at the surface has been changing in the last three decades at rates (and even signs) which vary over time and between sites. The changes have been attributed in part to decreases in total ozone column, but other factors such as changes in clouds and aerosol are also important. Since the beginning of the 1990s, there have been indications that atmospheric transmission in the northern hemisphere has been increasing following reductions in cloudiness[88] and aerosols[86] which would have amplified any increases in UV irradiance attributable to ozone depletion.

Possible changes that precede the instrumented record

To assess more fully the present changes in ozone and UV-B radiation and their effects, it is desirable to gain knowledge about their historical long-term, non-anthropogenic changes. For example, it has been speculated that ozone depletion has been one of the factors contributing to large extinctions of organisms, such as that which took place 251 million years ago.[89, 90]

There have been speculations that damage to the ozone layer may have occurred 65 Myr ago, as a result of widespread combustion of biomass following a large meteoritic impact.[91] These authors estimate that the concentrations of organic chlorine and bromine may have been an order of magnitude greater than at present, resulting in ozone loss and increased UV radiation which could have damaged life on Earth. However, there is no proof that this contributed to any biospheric extinctions.

To assess possible changes, biological proxies for UV-B radiation have been considered using lake sediments[92, 93] (see Chapter 4). Analyses of plant pigments[94] have had limited success so far. It has been suggested that changes in UV-B radiation due to ozone depletion may have been responsible for a departure in the relationship between tree rings and temperature over the period since the 1970s.[95]. However, the changing behaviour seems to precede the period of most significant ozone depletion, and may be due to other factors. One possible factor is the decreasing irradiance due to increasing cloud and aerosols that occurred over this period, sometimes called "global dimming",[88] a trend which may have reversed to a "global brightening" since the late 1980s.[86] Further work on biological proxies for past UV-B radiation is currently being conducted by several groups, and reliable results may eventually be produced. Promising attempts include the chemical composition and structure of pollen grains and spores.[96, 97]

The UV-B irradiance depends not only on the ozone layer and other atmospheric properties (clouds, aerosol), but also on the variable emission of the sun[98] and the variable geometry of the solar system.[99] Although several proxies for past solar emission exist, and though modelling and comparison with other stars can provide further information, the uncertainties remain great, and published values must be used with caution.

In a very long-term perspective,[100] calculations based on the oxygen content of the atmosphere[101] can give some information about the historical development of the ozone layer, as can measurements of the isotope composition of certain minerals (reviewed by Rumble[102]). In turn, these provide information about past UV radiation. Quantitative information about the ozone layer in the early 20th century can be obtained from the study of historic spectrographic plates used in astronomical studies.[103] This offers the promise of more accurate estimation of UV radiation prior to the 1920s at a few sites.

Interactions between ozone depletion, UV radiation, and climate change

Interactions between ozone depletion and climate change have recently been assessed and summarized in the IPCC/TEAP report.[25] Further details regarding these interactions are given in Chapter 1 of the full IPCC Report.[104] A recent review has also been undertaken by the WMO.[105]

These interactions can be complex, and they can act either way, with ozone depletion impacting on climate change, or with climate change impacting on ozone depletion. The interactions can involve chemistry, radiation, and dynamics, as well as feedbacks between those three processes. Examples of climate change affecting ozone through dynamical feedbacks have been discussed already (see section on ozone changes). Some of these feedbacks are summarised in Figure 1-7. See Chapter 5 for further details on interactions that involve biogeochemical feedbacks.

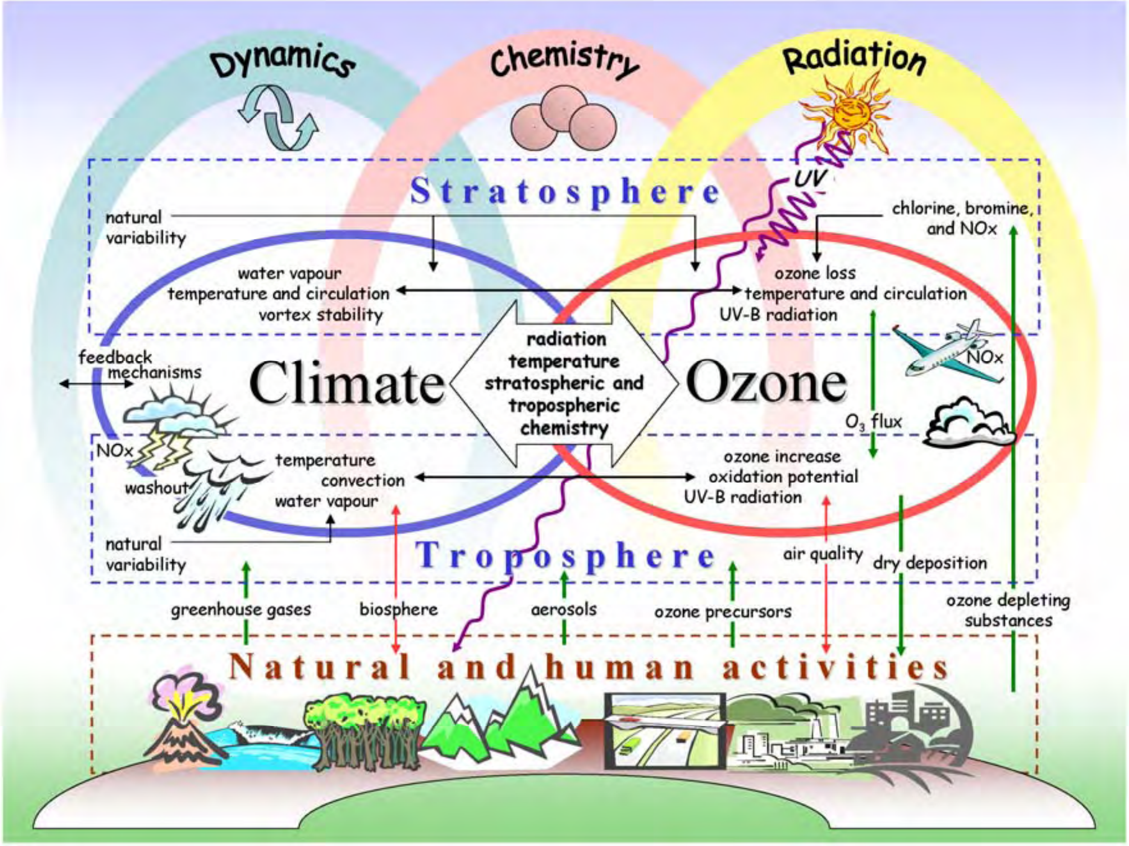

Figure 1-7. Update of ozone/climate feedback linkages. From [106].

Impacts of climate change on ozone depletion

Impacts of climate change on ozone depletion have been further explored, but there is not yet a consensus on whether the overall effect will be to delay or accelerate ozone recovery. Some processes would result in slowing of ozone recovery,[6, 107] while others would result in an acceleration.[108] Ozone-depleted air exported from polar latitudes comprises a significant portion of ozone losses at mid-latitudes[7-9]. This has negative implications for future ozone recovery at mid-latitudes: since further cooling of the polar stratosphere is expected as a consequence of global climate change, the cooling will be conducive to further rapid loss of ozone.

Changes in atmospheric circulation (dynamics) seem increasingly important for ozone variability.[109-112] For example, strong links have been found between stratosphere/troposphere exchange and the El Nino Southern-Oscillation (ENSO) climate pattern.[112] Model simulations show that much of the ozone increase seen in recent years at mid-northern latitudes can be explained by changes in dynamics, rather than being caused solely by reductions in atmospheric chlorine and bromine.[23] This has important implications for our confidence about future ozone recovery.

Ozone heats the stratosphere by absorbing incoming solar energy and outgoing infrared radiation from the Earth's surface. A significant component of the observed stratospheric cooling (-0.17 °C/decade) can be attributed to ozone depletion, rather than being solely a radiative effect of climate change.[113] Therefore, if ozone amounts were to increase in the future, this would tend to warm the stratosphere, diminishing the future cooling there due to increasing greenhouse gases (GHG). The warming effect would aid further recovery of the ozone layer in polar regions where heterogeneous chemistry on ice crystals dominates the ozone loss processes, but would have the opposite effect in other regions where gas-phase chemistry dominates. Consequently, the future impact of climate change may aid ozone recovery at mid-latitudes.

An analysis of four decades of ozone sonde data in Antarctica concluded that, while ozone depletion has been less severe in recent years, this cannot be necessarily linked to any recovery attributable to reductions in chlorine.[114] That result supported an earlier study,[115] which demonstrated that reduced Antarctic ozone loss during 2001 to 2004 is a consequence of warmer springtime temperatures at altitudes between 20 and 22 km in recent years.

Recent model calculations that include climate feedbacks suggest that, because of these interactions, ozone is expected to return to pre-1980 levels somewhat later in polar regions than at mid-latitudes. However, the observational evidence available so far suggests that these interactions have not yet introduced large delays in ozone recovery (e.g., see Figure 1-1 and 1-2). As discussed above, there is evidence that ozone has already started to recover at mid latitudes. In recent years, springtime ozone losses in Antarctica have generally been less severe,* but this has been attributed to increased temperatures in the lower stratosphere, which in turn may be attributed to changes in circulation patterns that have resulted from global warming.[114] In contrast, in the absence of changes in circulation patterns, climate change is expected to cause lower temperatures in the Arctic stratosphere, which will, in turn, lead to a greater probability of rapid ozone depletion on the surfaces of ice crystals.

The effects of climate change on ozone depletion may be most pronounced - yet least understood - at high latitudes,[116] where springtime ozone losses are expected to continue.[117] In polar regions especially, interactions with global warming complicate the recovery. Increases in water vapour and cooling of the stratosphere will be more important than elsewhere. Further, we have less confidence in the performance of models in this region, which tend to overestimate ozone concentrations and underestimate the ozone loss. The ozone loss depends critically on temperature. In polar regions, changes in climate (surface temperature) can trigger changes in circulation which affect ozone. Conversely, changes in ozone lead to changes in stratospheric temperature, which, in turn, may lead to changes in circulation which can trigger changes in climate.

Finally, one cannot simply assume that effects of climate change on ozone and UV radiation will continue at the present rate. According to recent assessments of climate change,[118] the average rate of surface temperature change over the 21st century is likely to be about 5 times that in the past century. Consequently, unless strong mitigation measures are undertaken with respect to climate change, profound effects on the ecosystem and on the solar UV radiation received at the Earth's surface could be anticipated.

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* The WMO reported that in 2006 the ozone depletion in Antarctica has been one of the most severe on record, with respect to the mass deficit of ozone. See Antarctic Ozone Bulletin 2006, No 4 at http://www.wmo.ch/web/arep/ozone.html

Impacts of ozone depletion on climate change

Changes in ozone and UV radiation can potentially influence climate through impacts on tropospheric photochemistry, as discussed in Chapter 6. Observational data and a new modelling study have both suggested that decreases in stratospheric ozone in Antarctica have led to climatic changes both in the stratosphere and at the Earth's surface. These changes in ozone have led to increased westerly winds at latitudes 50 to 60ºS. This, in turn, has resulted in a surface cooling in Antarctica and a warming at high latitudes outside the Antarctic continent.[119]

Changes in atmospheric temperatures lead to important changes in modes of atmospheric circulation, in particular the North Atlantic Oscillation (NAO) and the Arctic Oscillation (AO). These are responsible for large scale redistributions of atmospheric mass, which produce large scale variability in NH dynamics, and have a profound effect on winter climate variability around the Atlantic basin. Temperature increases in the troposphere as well as temperature decreases in the stratosphere both contribute to these changes.[110, 111] A potentially important impact of changing ozone on climate has been proposed recently to explain the strengthening of the NAO in recent decades.[120] This strengthening has altered the surface climate in these regions at a rate far in excess of global mean warming. Although weak NAO trends are reproduced in climate simulations of the 20th century, the unexplained strengthening of the NAO was fully simulated in a climate model by imposing observed ozone trends in the lower stratosphere. This implies that stratospheric variability needs to be reproduced in models to fully simulate surface climate variations in the North Atlantic sector.

As discussed further in Chapter 5, climate change can also be mediated through UV-induced changes[121] in dimethyl sulphide (DMS), a substance emitted from oceanic phytoplankton that can modify the reflectivity of the atmosphere.

Other factors that may affect ozone depletion and climate change

As discussed previously, volcanic eruptions, and long-term periodic variations in planetary motions can directly affect UV radiation, ozone, and climate. Changes in solar activity may also be important. Solar UV radiation arriving outside the Earth's atmosphere follows an 11-year cycle. Counter-intuitively, when the solar activity is highest, UV-B radiation at the surface has a minimum due to the increased production of ozone. Recent studies have demonstrated that the changes in solar UV radiation can also induce changes in some modes of atmospheric dynamics (e.g., the Southern Annular Mode (SAM), and the AO).[122, 123] Interactions with solar activity may also be more important for ozone depletion and UV irradiance increases than previously thought.[71, 122, 124-129]

Recent modelling studies have shown larger effects of increased CFCs, HCFCs and other halocarbons (and their effects on ozone) on tropical tropopause temperatures than had previously been calculated.[130] These temperatures are critical because they control the amount of water vapour entering the stratosphere, which in turn is converted to OH which then destroys ozone through catalytic cycles. The model indicates that the halocarbons have led to temperature increases in that region of about 0.4ºC over the last 50 years, which exceeds the cooling effect from the major GHGs. The fact that this region has actually cooled implies that other factors that are not included in the model must also be important. These could include increases in cirrus cloud, increases in water vapour (both of which have been observed), or increases in the strength of the mean atmospheric circulation. The model also shows that the indirect effect of ozone depletion in the stratosphere at mid to high latitudes has offset approximately half of the global surface warming to date. Consequently a slightly faster rate of surface warming is predicted in the future as ozone recovers.

Interactions between different regions of the stratosphere may also be important. Even in the event of a complete recovery in chlorine, ozone in the lower tropical stratosphere will not recover before ~2050, because of a reduction in UV irradiance due to more-rapidly increasing ozone at higher altitudes[131]. This can be thought of as a reverse example of the well-known "self-healing" effect for ozone, whereby ozone losses at high altitudes are partially compensated by ozone production from increased UV irradiance at lower altitudes. In some cases it is not obvious whether changes in ozone are driving changes in dynamics, or whether changes in dynamics are causing changes in ozone.[112]

Although there is speculation about the effects of climate change introducing lags to ozone recovery, the observational data so far suggests that global ozone may, in fact, be increasing faster than expected, given the estimated rate of decrease of Equivalent Effective Stratospheric Chlorine (EESC).[5]

Future changes in methyl bromide and methyl chloride emissions resulting from climate change may also be important. Methyl bromide constitutes the largest source of bromine atoms entering the stratosphere and therefore plays an important role in the depletion of stratospheric ozone,[132] and thus on UV radiation. The major source of methyl bromide is natural, but a significant fraction (about 20%, and reducing) is man made.[5] Methyl bromide emissions from rice paddies will increase appreciably with global warming,[133] (see Chapter 5).

Future expectations

The future pathway for UV radiation is uncertain because the pathway for ozone recovery is uncertain. As reported in recent assessments,[5, 25] there are wide variations between models that predict future ozone, and there are large discrepancies between past measurements, and model simulations of the past (see Figure 1-8).

A recent study of trends in total column ozone from several models and from satellite observations from the period 1979-2003 found large discrepancies between the models and measurements.[134] The observed positive trends in both hemispheres in the recent 7-year period are much larger than predicted by the models. Most models underestimate the past trends at mid- and high latitudes. Quantitatively, there is much disagreement among the models concerning future trends. However, the models agree that future ozone trends are expected to be positive and less than half the magnitude of the past downward trends.

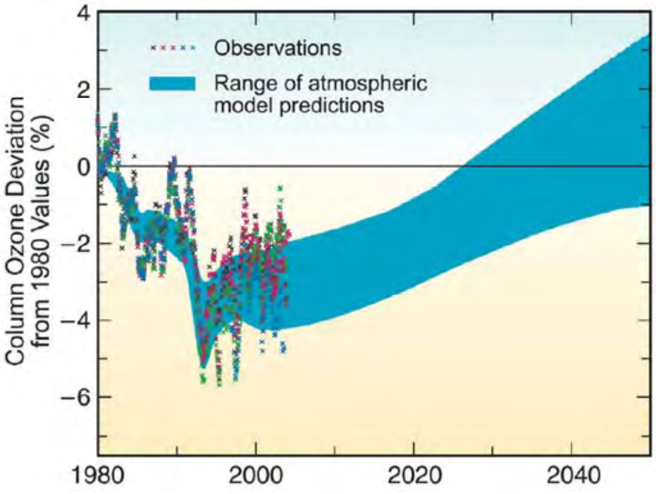

Figure 1-8. The future outlook for ozone recovery is uncertain. Observed and modelled column ozone amounts (60ºS–60ºN) as percent deviations from the1980 values. From [25].

Substantial research efforts in the 1980s and 1990s have advanced our knowledge about solar UV radiation and its interactions with the atmosphere and biosphere. In recent years, there have been significant improvements in instrumentation technology and radiative transfer modelling which have helped our understanding of the various processes relevant to UV radiation. There are still areas where further development is needed.

Important effects on UV radiation may be expected from long-term changes in ozone, clouds, and aerosols. According to the predictions of climate models, column ozone is expected to increase during the next decades. The response of surface UV radiation solely due to changes in ozone can be estimated from RT model calculations. Such estimates are shown in Figure 1-9, where any direct or indirect effects from clouds and aerosols are neglected. The ozone losses calculated by this model are larger than observed, especially in the Southern Hemisphere. Therefore, the corresponding increases in the calculated UV irradiance are overestimated. At mid-latitudes surface UV irradiance is predicted to peak between about 2000 and 2010 and is expected to return to the pre-1980 levels between 2040 and 2070, but the these phases occur later at the southern high latitudes. Figure 1-9 also shows that the predicted changes in UV irradiance are significantly larger in the southern hemisphere than in the northern hemisphere.

Despite the great progress that has been made in the last decade in understanding the relations and interactions of surface UV radiation with atmospheric composition and structure, there are still areas where scientific research is not sufficiently advanced. There is lack of widespread observational evidence, especially in the tropics and in urban environments, to quantify the influence of tropospheric ozone, aerosols, and other pollutants on surface UV radiation. Knowledge of interactions between climate change and UV radiation will improve as more modelling studies and observational evidence become available.

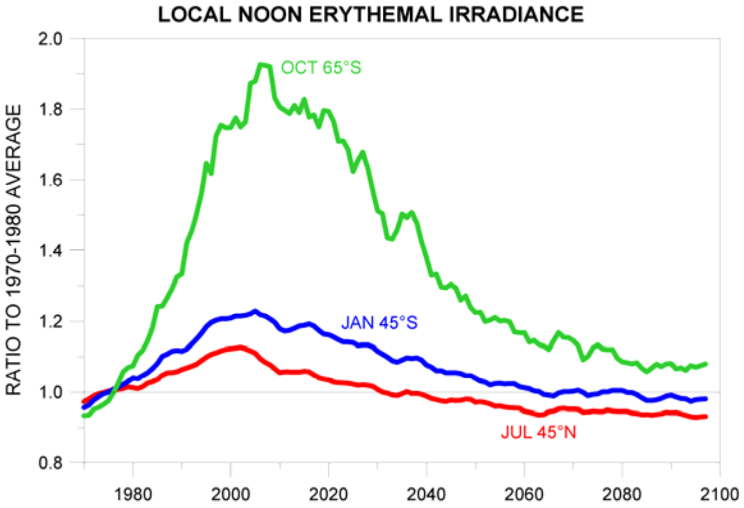

Figure 1-9. Estimated changes in erythemally weighted surface UV irradiance at local noon in response to projected changes in total column ozone for the period 1970-2099, using zonal-averages in total ozone in the latitude bands 35°N-60°N, 35°S-60°S, and 60°S-90°S, and the solar zenith angle corresponding to 45°N in July, 45°S in January and 65°S in October respectively. At each latitude, the irradiance is expressed as the ratio to the 1970-1980 average. The results have been smoothed with a 5 year running mean filter to remove some of the year to year variability in the ozone predictions in the model. From [5]

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Chapter 2. The effects on human health from stratospheric ozone depletion and its interactions with climate change

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Summary

Ozone depletion leads to an increase in the ultraviolet-B (UV-B) component (280-315 nm) of solar ultraviolet radiation (UVR) reaching the surface of the Earth with important consequences for human health. Solar UVR has many harmful and some beneficial effects on individuals and, in this review, information mainly published since the previous report in 2003[1] is discussed. The eye is exposed directly to sunlight and this can result in acute or long-term damage. Studying how UV-B interacts with the surface and internal structures of the eye has led to a further understanding of the location and pathogenesis of a number of ocular diseases, including pterygium and cataract. The skin is also exposed directly to solar UVR, and the development of skin cancer is the main adverse health outcome of excessive UVR exposure. Skin cancer is the most common form of malignancy amongst fair-skinned people, and its incidence has increased markedly in recent decades. Projections consistently indicate a further doubling in the next ten years. It is recognised that genetic factors in addition to those controlling pigment variation can modulate the response of an individual to UVR. Several of the genetic factors affecting susceptibility to the development of squamous cell carcinoma, basal cell carcinoma and melanoma have been identified. Exposure to solar UVR down-regulates immune responses, in the skin and systemically, by a combination of mechanisms including the generation of particularly potent subsets of T regulatory cells. Such immunosuppression is known to be a crucial factor in the generation of skin cancers. Apart from a detrimental effect on infections caused by some members of the herpesvirus and papillomavirus families, the impact of UV-induced immunosuppression on other microbial diseases and on vaccination efficacy is not clear. One important beneficial effect of solar UV-B is its contribution to the cutaneous synthesis of vitamin D, recognised to be a crucial hormone for bone health and for other aspects of general health. There is accumulating evidence that UVR exposure, either directly or via stimulation of vitamin D production, has protective effects on the development of some autoimmune diseases, including multiple sclerosis and type 1 diabetes. Adequate vitamin D may also be protective for the development of several internal cancers and infections. Difficulties associated with balancing the positive effects of vitamin D with the negative effects of too much exposure to solar UV-B are considered. Various strategies that can be adopted by the individual to protect against excessive exposure of the eye or the skin to sunlight are suggested. Finally, possible interactions between ozone depletion and climate warming are outlined briefly, as well as how these might influence human behaviour with regard to sun exposure.

Introduction

There are many harmful and some beneficial effects of solar ultraviolet radiation (UVR) on human health. Skin cancer and cataract are examples of the former category while the synthesis of vitamin D is one example of the latter category. With ozone depletion and the consequent increase in terrestrial UV-B, these effects may be enhanced. Various models predict increases in the number of skin cancers and cataracts that can be attributed to ozone depletion over the baseline that occurred before ozone depletion.[1, 2] However, as stated previously[1], human choice in determining where, when, how and for how long an individual is exposed to solar radiation is a major, if not the principal, factor that establishes the health outcomes. Assuming the same human exposure habits, ozone depletion with resulting increase in UV-B will increase the numbers of skin cancers and cataracts, while a positive effect could be a general improvement in vitamin D status.

In this report, discussion will centre first on interactions between solar UVR and the eye and, secondly, on interactions between solar UVR and the skin, concentrating on the risks of, and trends in, the incidence of skin cancers and the genetic factors involved in their development. A section on the immune effects of UVR comes next, followed by another on the UV-induced synthesis of vitamin D and its relationship with a range of diseases. Finally, strategies for responding to the problem of ozone depletion are considered, especially those that protect the individual. In most instances, only new information available since the previous full report in 2003 is included although in certain instances reference is made to earlier key publications. It should be noted that the topic of air pollution relating to ozone depletion is addressed in Chapter 6 of this report, this includes reference to aspects concerning human health.

The eye

The eye and the skin are the only organs of the body that are exposed to solar UVR. The effects of sunlight on the eye may be acute (usually after a latent period of several hours), long-term after an acute exposure, or long-term following chronic exposure to levels of UVR below those required for acute effects (Table 1). In our last report we focused on cataract, the UV-B related eye disease with the most serious public health implications.[1] This section of the report concentrates first on how UV-B reaches and interacts with the surface and internal structures of the eye, and then provides an update on chronic effects that may impair vision.

Interaction of solar UV-B with target tissues in the eye

At low solar zenith angles (high solar elevation angles), the UV-B photons most likely to fall on the cornea and other ocular tissues are those from indirect sunlight, i.e., those scattered by atmospheric components or reflected from surfaces. In contrast to its effects on the skin, direct sunlight plays a minor role in UV-B-related eye disorders due to a natural aversion to looking directly at the sun, and shadowing by the brows when the sun is high is the sky. Under conditions of cloud cover (with lower light levels), the natural defence mechanisms of the eye, for example squinting, are relaxed, permitting greater exposure of the outer surface and internal structures of the eye, such as the lens. At the same time, scattering and reflection by clouds increases the diffuse radiation incident on the eye.[3, 4] UV reflectance values vary considerably for different natural terrains and manufactured materials. Grass and other green vegetation are natural strong absorbers of UV-B and reflect this waveband poorly (2-3%), whereas fresh snow is an excellent reflector (more than 90%). These variations can result in significant errors in estimating UV-B exposure based solely on location, as was commonly done in early epidemiologic studies of the role of sunlight in eye disease.

Table 1. Potential acute and chronic effects of exposure to UV-B on the eye and adjacent tissues

Tissue	Acute Effect	Chronic Effect
Lid and peri-ocular skin	**Sunburn:** erythema (redness)	**Freckling**
	blistering	**Lentigines** (age spots)
	exfoliation (peeling)	**Hypomelanosis** (vitiligo)
	Tanning	**Non-melanoma skin cancer**
		Actinic keratosis
		Cutaneous melanoma
Conjunctiva	**Photoconjunctivitis**	**Pinguecula** (local degeneration)
	Chemosis (swelling)	**Dyskeratosis** (abnormal epithelial cell differentiation)
		Intraepithelial neoplasia
Cornea	**Photokeratitis**	**Climatic droplet keratopathy** (epithelial degeneration)
	Endothelial damage (swelling)	**Pterygium** (see text)
	Reactivation of latent herpes viruses	**Endothelial changes**
Lens	**Anterior subcapsular opacities**	**Age-related cataract** (see text)

Peripheral light focussing by the eye (see Figure 2-1). A factor that must be considered when assessing exposure of the internal structures of the eye to UV-B is that the various zones of the cornea direct the radiation to different locations within the eye. Coroneo et al.[5] suggested that light and UVR incoming from the side is focussed on specific areas of the cornea, resulting in a twenty-fold increase in exposure that may be important in the induction of pterygia and cataract. They also proposed that UVR was similarly concentrated in the lower nasal quadrant of the crystalline lens, the location where age-related cortical cataract is commonly first detected. Human[6] and mannequin[7] studies have confirmed that incoming temporal UVR from behind the coronal plane (100° to 135° to the sagittal plane, see Figure 2-1) was focussed into the anterior chamber angle. This is modified by corneal shape, anterior chamber depth, and location of the eye within the bony orbit, squinting, eyelashes, prominence of cheekbones and presence of lid skin folds on the temporal side of the eye.

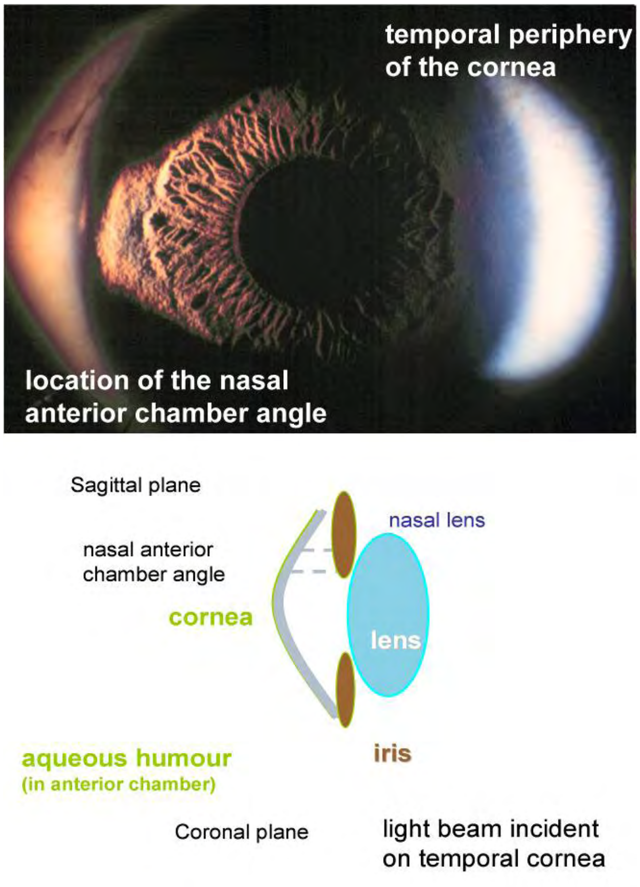

Figure 2-1. Peripheral light focussing. **Top:** Photograph showing how a beam directed towards the temporal side of the cornea is focussed into the anterior chamber angle. **Bottom:** A beam of light (or UVR), from behind the coronal plane, directed onto the temporal periphery of the cornea is refracted and focussed into the nasal angle of the anterior chamber of the eye, as shown by arrows. If the incident beam originates in front of the coronal plane, the focus shifts into the nasal part of the lens.

Transmittance of the ocular media. In order for UV-B incident on the surface of the eye to reach the crystalline lens, it must first pass through the cornea and the aqueous humour. Although the aqueous humour absorbs little environmental UV-B, the cornea has a significant role in preventing UV-B from reaching the lens, with some parts of the cornea being more effective than others. Kolozsvari *et el.*[8] have shown that UV-B absorption is about twice as high in the anterior layers (epithelium and Bowman layer) of the human cornea as in the posterior layers. Their data indicate that the whole cornea begins to transmit at 280 nm (<0.01%), increases to 1% at 295 nm and approaches 5% at 300 nm. Although the actual amount of UV-B transmitted is low, it should be noted that UV-B at 300 nm is about 600 times more biologically effective at damaging ocular tissue than UV-A at 325 nm.

At birth the human lens is colourless and allows both UV-B and UV-A to pass through to the retina. As the lens ages, there are significant changes in the lens proteins, including a decrease in their solubility, that result in increased, wavelength independent, scatter and consequent degradation of vision (clinically called nuclear sclerosis). Frequently there is also a yellowing which can eliminate the passage of UVR and limit the passage of light in the violet-blue end of the visible spectrum.

Chronic effects of UVR on the eye

Pterygium. This wing-shaped, inflammatory, proliferative and invasive growth occurs on the conjunctiva and cornea of the human eye (Figure 2-2). It is induced, in part, by intracellular damage caused by UV-B exposure[9] and most commonly occurs in the superficial layers of the nasal cornea. Pterygia grow towards the centre of the cornea and can severely impair vision. In their early stages, they appear as small opacities at the nasal edge of the cornea and then spread to become a fleshy raised area. A number of causal factors, other than UVR, have been proposed as important to pterygium development including mechanical irritation, heredity, heat, cold, and wind. None of these adequately explains the predominately nasal location of pterygia. This preferred location has been explained, however, on the basis of the peripheral light focussing effect discussed above.[10]

Pterygia are more prevalent and progress more rapidly in individuals living in regions near to the equator or at very high altitudes. Al Bdour and Latayfey[11] reported a strong correlation between pterygia and environmental UVR in Australian aborigines. In the more temperate climate of the northeastern U.S., a significant relationship was found between the cumulative dose of solar UV-A and UV-B and the prevalence of pterygium.[12] The higher prevalence of pterygium in outdoor occupations has been attributed to exposure to excessive amounts of sunlight. In a population-based sample of residents of the Australian state of Victoria who were aged 40 years and older, statistical modelling revealed that 43.6% of the risk of pterygium could be attributed to cumulative dose of sunlight.[13] This result was the same when cumulative dose of ocular UV-B was substituted in the model for cumulative dose of sunlight. Pterygium continues to be considered a significant public health problem in rural areas and occurs primarily as a result of ocular sun exposure.[13] In a study conducted in Perth, Western Australia[14], there were strong positive associations between pterygium and measures of potential and actual sun exposure. The strongest associations were seen for the estimated daily ocular solar radiation dose at any age, which in those in the highest quartile of exposure resulted in about a 7-fold greater risk. Although other agents may contribute to pterygium development[15-17], in most epidemiological studies the common factor is UVR exposure, thereby indicating that UVR can be considered a causal agent. Thus, the implication for prevention of pterygium is that ocular protection from sunlight is beneficial at all ages.

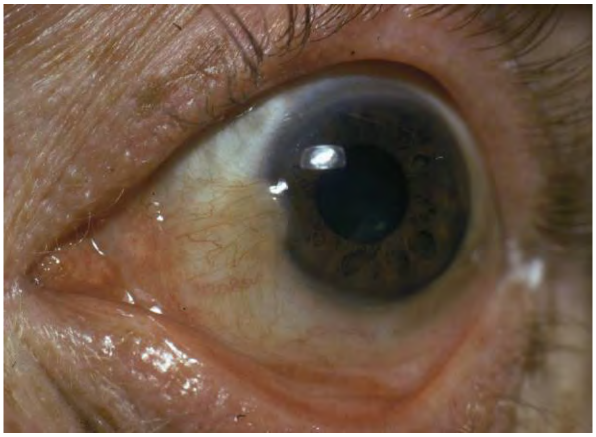

Figure 2-2. An early pterygium.

Cataract. Three main types of age-related cataract can be distinguished, based on their location: cortical cataract involving the anterior (and posterior) cortices of the lens; posterior subcapsular cataract at the extreme posterior cortex lining the lens capsule[1] and nuclear (sclerotic) cataract at the nucleus of the lens. However by the time the individual requires surgery, mixed categories are most commonly present. Cortical cataract arises from localised changes occurring in the cortex of the lens, where opaque radial spokes begin to develop on the periphery and extend towards the centre, eventually affecting vision.[18, 19] The second form of cataract, posterior subcapsular cataract, is thought to develop when the lens epithelial cells migrate to form a plaque of opacities and cysts at the posterior surface of the lens. These lesions are particularly detrimental to vision when the pupil constricts due to sunlight or other bright sources, or during near tasks. The third form of cataract, nuclear cataract, occurs as the crystalline lens of the eye ages and the nucleus loses its transparency, becoming more opalescent and sometimes turning yellowish to brown in hue.[20, 21]

A number of publications has reviewed the epidemiologic information linking UVR exposure to cataract.[22-24] Although earlier reviews concluded that the range and variability of the study designs precluded definitive conclusions, most of the more recent analyses suggest a role for UV-B in some types of age-related cataract, particularly cortical cataract. A frequently cited early estimate of risk from personal ocular exposure to solar UV-B is that of Taylor et al.[25] in the *Chesapeake Bay Waterman Study*. Watermen in the highest quartile had a three-fold increased risk for cortical cataract. It is important to note that the subjects in this cohort were only exposed to levels of solar UV encountered in mixed, often overcast, climate at intermediate latitude. The same group[26] that conducted the *Chesapeake Bay Waterman Study* also conducted a population-based epidemiologic study in Salisbury, Maryland. The increased risk of 10% of developing cortical cataract associated with UV exposure in this study (the Salisbury Eye Evaluation [SEE] project[27]) was more modest, but the population was considered more representative of the U.S. as a whole. West and her colleagues subsequently used these data from the SEE project as the basis from which to develop risk estimates for the entire U.S. population under conditions of ozone depletion.[26] These risk estimates, which were calculated for fixed levels of ozone depletion ranging between 5 and 20%, indicated that the number of cortical cataract cases seen by 2050 would increase between 1.3 and 6.9% respectively, with associated health costs for the U.S. of between about $0.6 and $3 billion respectively. There are, in addition, important social costs associated with cataract development.

A recent review found that there was insufficient evidence to conclude that UVR exposure played a causal role in the development of posterior subcapsular cataract.[28] However, a recent study in Japan[29] showed that the severity of nuclear cataract increased with UV-B exposure. Furthermore, lifetime cumulative UV-B exposure and particularly exposure in the teenage years correlated with the presence of nuclear cataract in females. Another report indicated that the association between nuclear cataract and occupational sun exposure was significant for exposure between the ages of 20 and 29 years.[30] Supporting evidence for such a difference in a period of age susceptibility is provided by an animal study in which the same dose of UVR induced more severe cataracts in young than in older animals.[31]

The skin

Sunburn is the effect most frequently experienced by the human population due to excessive solar UV-B exposure. It is an inflammatory reaction to a toxic assault on the skin. Although human skin is adapted to the ambient UVR, the sunburn reaction demonstrates that excessive exposures can stretch defensive mechanisms to the limit, or even exceed them (pain and blister formation). Despite the remembered discomfort from past episodes, about a third of US residents report at least one sunburn per year.[32] Fair-skinned people are most susceptible to sunburn, and they correspondingly run a higher risk of long term adverse effects, such as skin cancer. In the following sections, the relationship between various types of skin cancer and solar UVR is outlined.

Skin cancer types and trends

Skin cancer is the most common form of cancer among fair-skinned populations and its incidence has increased markedly over the last century. Many skin cancers are detected early, at a stage where they can be easily and effectively treated. This limits morbidity and mortality. In addition, for the majority of skin cancers, the 'non-melanoma' skin cancers (NMSC) consisting of basal cell carcinomas (BCC) and squamous cell carcinomas (SCC), the malignant potential is low which also reduces death from these diseases. This is not the case for the most malignant form of skin cancer, melanoma, that arises from pigment cells (melanocytes), and is responsible for most of the deaths from the skin cancer. Projections show an approximate doubling in all types of skin cancer from 2000 to 2015 in the Netherlands, but this is also due, in large part, to ageing of the population.[33]

Melanoma

As found in earlier epidemiological studies, cutaneous malignant melanomas are related to sun exposure in early life, to episodes of severe sunburn, and to the number of moles (nevi), which in turn is related to sun exposure in early life.[34] In the last decade, much progress has been made in identifying genetic changes in melanoma cells, and the functional importance of these genetic changes for melanoma development has been demonstrated in genetically modified mice. However, the precise mechanisms underlying nevus formation and progression to melanoma, and the role of solar UVR in this process, remain to be resolved.[35, 36]

Epidemiology of melanoma. Although rates of increase in melanoma incidence appear to be levelling off in countries with the highest number of cases[37-39], the absolute incidence is continuing to rise. Mortality, however, has risen much less or has even stabilized especially in females, and in younger age cohorts, although not in older males in countries such as the USA, Scotland and Australia. The major increase in incidence recently has been attributed to the thin melanomas that have high survival rates.[40] A thin melanoma is defined as being less than or equal to one mm thick. This predominance of the early stages of melanoma could be due to greater awareness in the general population regarding the dangers of suspicious-looking moles. Prompt diagnosis and treatment may then limit any increase in mortality.[41, 42] Melanomas with an attached nevus from which they apparently originated are on average thinner, of the more superficial spreading type and occur more often in irregularly exposed skin than melanomas that show no remnants of a precursor nevus.[43] Patients with the nevus-associated melanomas are younger and have more nevi.

Strouse et al.[44] found that the incidence of melanoma in children in the USA is rising rapidly but survival is improving. They showed that the incidence rate of melanoma was positively correlated with environmental UVR exposure. The chance of surviving a melanoma decreases with age and is lower for boys compared with girls. It is also lower if the primary tumour occurs on body sites other than the extremities and the torso (i.e., locations other than those exposed intermittently to the sun). The latter finding is in agreement with a study of adults by Berwick et al.[45] who found that survival from melanoma was higher in individuals with a history of increased intermittent sun exposure and episodes of sunburn. However, these authors also found improved survival with a history of increased skin awareness and increased solar elastosis (i.e., a skin 'aged' by chronic sun exposure).

Earlier reports regarding a seasonal variation in the diagnosis of melanoma were confirmed in recent European and Australian studies. These revealed maximum incidence for thin melanomas on extremities in the summer in females.[46, 47] This effect may be attributable to enhanced skin awareness in the hotter months or to stimulation of melanoma growth after (over-)exposure to the sun. Boniol et al.[47] found that survival from melanomas diagnosed in the summer was higher, as might be expected from the higher number of thin melanomas diagnosed at that time of the year, but, after correction for tumour thickness, the effect was still significant. The authors therefore suggest that patients in whom melanomas are diagnosed after recent sun exposure may show better survival.

Trends and changes in skin cancer incidence over recent decades clearly indicate the importance of human behaviour, particularly in relation to exposure to solar UVR.[37] For example, Gandini et al.[48] undertook a meta-analysis of 57 observational studies which showed that intermittent sun exposure and sunburn history played considerable roles as risk factors for melanoma, and Agredano et al.[49] found a very strong relationship between increasing access to air travel to leisure destinations and increasing melanoma incidence. However, case-control studies generally find that genetic factors carry more risk than behavioural aspects, such as moderating UV exposure.[50] It should be noted, however, that the genetic factors can be determined more accurately than personal UV exposure; the latter is assessed by very poor surrogates (e.g. recalled number of sunburns in youth or lifetime hours of sun exposure). This inaccuracy in determining past UV exposure will tend to lead to lower estimates of relative risk. Moreover, an individual’s behaviour with regard to UV exposure can be altered to reduce risk, very much in contrast to an individual’s genetic background.

Latitudinal and temporal trends in skin cancer, notably in melanomas, underline the major importance of UV exposure as an environmental risk factor. The integrity of the stratospheric ozone layer, as the prime atmospheric UV filter, therefore remains crucial in protection against melanoma.

Genetic risk factors for melanoma. There are well-established genetic factors conferring susceptibility to melanoma – notably inherited mutations in the cell-cycle control gene, *p16INK4a*, and in the “hair-colour” gene which codes for the melanocortin 1 receptor (MC1R). The MC1R gene contributes to the control of pigmentation in hair and skin[51] and is an important risk factor for all types of skin cancer, including melanoma.[52] Other additional genes are related to melanoma risk, e.g., the OCA2 gene which also controls skin and eye colour[53], and an as yet unknown gene located on chromosome 1.[54]

UVR causes DNA damage which can give rise to gene mutations which in turn can contribute to skin cancer formation (see below). Hence, repair of this damage is of crucial importance. The solar UV-B induced DNA damage (mainly cyclobutane pyrimidine dimers, CPDs) is removed by a ‘cut-and-paste’ type of DNA repair (‘nucleotide excision repair’). A complete dysfunction in one of the enzymes in this repair system results in a dramatic increase in risk of skin cancer, including melanoma. More subtle genetic variations (polymorphisms) in the repair enzymes can modify the efficacy of DNA repair, and thus affect skin cancer risk. Certain genetic variations in repair enzymes were indeed found to be associated with melanoma risk.[55-61]

UVR can generate reactive oxygen species and thus inflict damage to cell components, particularly DNA. Although melanin pigment is generally protective, it may also contribute to oxidative damage under certain conditions[62], especially its red variety, pheomelanin.[63] Certain inherited or acquired traits that increase oxidative stress appear to be associated with melanoma and its precursor lesion, dysplastic nevus.[64-66] Genetic variation in a protein (APE1) involved in the repair of oxidized DNA modifies melanoma risk.[67]

These inherited predispositions to develop melanoma will help to identify high risk groups who may be particularly susceptible to increases in ambient UV-B radiation.

Oncogenic alterations in melanomas. In terms of molecular mechanisms, melanomas from chronically exposed, from intermittently exposed and unexposed skin sites have different molecular signatures.[68] Notably, the melanomas from intermittently exposed skin have a high frequency of activating mutations in a critical signalling molecule, B-RAF. MC1R variants, which are associated with enhanced risk of melanoma, are strongly associated with *B-RAF* mutations.[51] Ten to 20% of melanomas from chronically exposed skin bear mutations in N-RAS, a protein preceding B-RAF in the signal cascade for cell proliferation.[69] Mutations in *B-RAF* and *N-RAS* genes are already present in some cells in nevi[70], but nevus cells do not proliferate and are kept in a 'senescent state'.[71, 72] The mutations in the *B-RAF* oncogene are not typical of UV-B radiation, but could be due to UV-induced oxidative damage.

The epidemiological finding that melanomas associated with intermittent sun exposure[45] show better survival may be linked to the specific molecular changes found in these tumours.

Animal experiments on UVR and melanoma. Because of the well-established role of UVR in NMSC and the known mutagenic and carcinogenic properties of UV-B radiation, it seems most likely that UV-B wavelengths are also contributing to the development of melanoma. However, human melanomas show no gene mutations that are typical of UV-B radiation. Animal models may serve to elucidate whether, and if so, how UV-B radiation contributes to the development of melanoma.

Experiments with transgenic mice confirmed the epidemiological finding that the neonatal period can be critical to the development of melanomas later in life.[73, 74] More specifically, a study in transgenic mice showed neonatal UV-B exposure to be highly effective.[75] These melanomas, which closely mimic the human disease, could not be evoked by neonatal UV-A exposure.[75] The latter finding is in accordance with earlier experiments in opposums[76], but differs from the results obtained with small *Xiphophorus* fish.[77] In the fish, both UV-B and UV-A neonatal exposure proved to be very effective in causing melanomas, and the variation in effectiveness with wavelength was recently found to closely follow the variation in the UV induction of oxidant radicals from melanin in the skin of the fish.[78] In the initial experiments with neonatal UV exposure, the HGF transgenic mice were albino, but recent experiments showed that these mice crossed into a pigmented background were also susceptible to melanoma induction by neonatal UV-B exposure (Noonan and De Fabo, personal communication). Further experimentation with this model may shed more light on the wavelength dependency of melanoma induction in mammals. In another model, melanomas were induced by massive doses of UV-B radiation delivered to repair-deficient transgenic mice.[79] However, the severe skin trauma inflicted may have caused non-specific tumour promotion.[80]

In support of the epidemiological finding that intermittent sunburning exposures increase the risk of melanoma, experiments in hairless mice have shown this type of exposure regimen to be considerably more effective in inducing nevi (potential precursors of melanoma) than a regimen in which the exposure was more evenly spread over time.[81] Thus, sunburning UV-B exposure of adults may indeed also contribute to melanoma development by stimulating the proliferation of melanocytes.[82]

Immunity and melanoma. There are indications that immune mechanisms against melanomas are present in humans as demonstrated by the occasional spontaneous regression of some pigmented skin lesions. Further, immune responses against melanoma antigens are readily detectable in patients and immunotherapy is actively used for melanoma treatment.[83, 84] As found earlier for BCC, melanoma risk appears to be related to the density of mast cells in unexposed skin[85] so that the higher the number of mast cells, the greater the chance of developing melanoma. Interestingly, children with eczema (atopic dermatitis) develop fewer melanocytic nevi than children without eczema[86], and the therapeutic effect of UV exposure on the eczema might be related to a possible effect of the radiation on the cytokine network in the skin, the products of which then stimulate melanocytic growth.

Whereas there is substantial evidence for a role for UV-induced immunosuppression in NMSC, it is not known as yet if this mechanism is a factor in melanoma progression; this is currently an area of intense investigation.

Non-melanoma skin carcinomas (NMSCs)

In epidemiological studies prior to 1980, the skin carcinomas, SCC and BCC, were not considered separately, and were commonly found in people who had accumulated excessive hours of solar (UV) exposure. In more recent studies, important differences between SCC and BCC have emerged. SCC is associated mainly with chronic and life-long accumulated sun exposure[87] whereas BCC, similar to melanoma, is more closely associated with early-life and intermittent exposures resulting in episodes of severe sunburn. In addition, while SCCs occur on body sites most regularly exposed to the sun such as the face, BCCs are also found frequently on sites exposed intermittently to sunlight. Also the genetic alterations identified in SCC and BCC show important differences.

Epidemiology of NMSC. Studies continue to show increases in the incidence of both SCC and BCC[33, 88, 89], with disproportionately high increases in BCC in young females on the lower limbs.[90, 91] Sunbathing is associated with a five-fold rise in the risk of BCC on the trunk.[57]

Although BCC is locally invasive, it is usually a slow growing and not very aggressive tumour; superficial BCC on the trunk is often misdiagnosed and confused with eczematous skin lesions. Detailed skin examination of subjects in a Queensland community established that the incidence of BCC on sites other than head, neck, hands and arms was 3-fold higher than actually treated[92]; a smaller study in Spain produced a similar result.[93] Hence, the large majority of the BCC on irregularly exposed sites appear to remain ‘sub-clinical’, i.e., cause no great discomfort, are never presented to a physician and remain essentially undetected.

Death due to NMSC in the USA has declined, and when it occurs, is often related to an excessively long delay before seeking medical care.[94]

Genetic risk factors for NMSC. UV-B radiation inflicts highly characteristic DNA damage (mainly CPDs), and the repair of this damage in human skin diminishes with age.[95] This type of DNA damage causes specific ‘point mutations’ which are found in the *P53* tumor suppressor gene in NMSC (see below). However, NMSC also show frequent crude chromosomal aberrations.[96] Such aberrations are already abundantly present in the benign precursor lesions of

SCC, the actinic keratoses (AKs). Complete double strand breaks (DSB) in the DNA cause these gross chromosomal losses and duplications. Interestingly, variants in genes involved in the repair of DSB in DNA appear to be related to NMSC risk, but not to melanoma risk[97] The association between NMSC and DSB repair ties in nicely with the recent finding that UV-B-exposed blood cells from patients with skin carcinomas are more prone to develop chromatid breaks than equivalent cells from melanoma patients and control subjects.[17]

Genetic variations in specific antioxidant proteins are associated with NMSC risk.[98] Variants of a repair enzyme, involved in excision of oxidized bases in DNA, affect SCC risk, but not the risk of BCC or melanoma.[97] Hence, there appear to be considerable differences in how oxidative DNA damage (such as induced by UVR) and its repair are related to the various types of skin cancer.

Oncogenic alterations in NMSC. Although considerably less efficient than UV-B, long-wave UV-A radiation can cause the same type of DNA damage as UV-B radiation, and thus give rise to 'UV-B-like' mutations in the *P53* tumor suppressor gene. However, oxidative damage contributes substantially at these longer wavelengths and causes different *P53* mutations from those induced by UV-B.[99, 100] Microscopic clusters of cell clones with strong expression of mutant-p53 protein in sun-exposed skin carry the same types of 'UV-B-like' *P53* mutations as skin carcinomas.[100, 101] Hence, all of these common microscopic clusters of cells with mutant-p53 in human skin could be potential precursors of skin carcinomas. In Swedish studies, microdissection of skin carcinomas showed consistent mutations in the *P53* gene throughout the tumour masses, i.e., most tumours appeared to be a clonal expansion from a founder cell with a particular 'UV-B-like' *P53* mutation.[101, 102] This conclusion is in agreement with earlier studies that found dominant 'UV-B-like' mutations in SCCs and BCCs.[103, 104] In contrast to these findings, a recent Australian study reported *P53* mutations to be very diverse, heterogeneous and disjunctive in SCCs and adjacent skin, i.e., every microdissected part of a tumour showed different *P53* mutations without any suggestion of a founder mutation or any clear overall indication of UVR as the cause.[105] By arguably separating out UV-B-like, UV-A-like, oxidative and 'other' *P53* mutations, the authors found the UV-B-like mutations to be located in the shallow parts of the tumours and the UV-A-like mutations in the deeper parts. This issue clearly needs to be investigated further.

Although both SCC and the precursor AK frequently carry various chromosomal aberration, the loss of a particular part of chromosome 18 appears to be related to the progression from AK to SCC.[96] The presence of multiple copies of parts of chromosomes may explain the amplification of the *H-RAS* oncogene frequently found in SCC.[106]

As reported in our previous review[1] and confirmed recently[107], nearly all BCCs display activation of the Hedgehog proliferative pathway, mostly through a defect in the PTCH protein in the cell membrane by mutations or loss of the coding gene. Some of these mutations are 'UV-B-like'. Further research has shown that certain variations in the *PTCH* gene may predispose towards BCCs[108], and that UV-B radiation can suppress PTCH function and thus potentially stimulate BCC development.[109]

Hence, the oncogenic alterations found in NMSC are attributable largely to UVR, and in some cases more specifically to UV-B radiation.

Animal experiments on UV and NMSC. Experiments in transgenic mice have identified the type of UV-B-induced damage (CPDs) that causes SCC and more immediate effects such as sunburn and thickening of the outer viable layer of skin (the epidermis).[110] Clones of cells with mutations in the *P53* gene – such as found in human skin – have been induced in well-controlled experiments in which mice were exposed to UV-B radiation.[111] Here these p53-mutant clones were tightly linked to the subsequent occurrences of SCC. In mice, UV-B-induced DNA damage gives rise to DSBs and strong signals for DSB repair.[112] Thus, UV-B radiation may induce the chromosomal aberrations present in human NMSC.

Immunity and NMSC. Organ transplant recipients (OTR) have a dramatically increased risk of developing SCC and, until recently, this was considered to be solely the result of taking immunosuppressive medication to prevent rejection of the transplant. Evidence is now accumulating to indicate that conventional immunosuppressive drugs can also adversely affect UV-induced DNA damage and repair in skin cells[113-115] and thus they may increase the risk of SCC. Immunosuppressed patients other than the OTR may be affected similarly.[116] A new generation of immunosuppressive drugs with a different mode of action may substantially reduce the risk of SCC.[117] Hence, the increased incidence of SCC in relation to immunosuppressive drugs may be due in large part to detrimental effects on UV defensive mechanisms in the skin, rather than to immunosuppression per se.

Immune effects of solar UVR

Mechanisms of UV-induced immunosuppression

When the skin is exposed to UVR, a complex cascade of events begins that ends in the suppression of certain types of immune responses, mainly those involving cell-mediated immunity. The main interactions affected are between three types of immunologically active white blood cells: antigen-presenting cells (APCs), T-helper (Th) lymphocytes and T-regulatory (T_{reg}) lymphocytes. The degree of suppression and the forms of cell-mediated immunity affected can vary depending on the quality, quantity and timing of the UVR, the frequency of the exposures, and the extent and location of the body surface irradiated.

One distinction commonly made is between local and systemic immunosuppression. Local immunosuppression occurs when an antigen (a “non-self” molecule that the host recognises as foreign and makes an immune response to) is applied directly to the irradiated body site soon after the UV exposure, resulting in a down-regulation of immunity to that antigen. In systemic immunosuppression, following UV exposure of one part of the body, the antigen is applied to a distant unirradiated body site, again leading to systemic down-regulation of immunity to that antigen. Certain steps of the two pathways differ such as whether the APCs have been directly exposed to the UVR or not.

The process for local immunosuppression is outlined in Figure 2-3, and details of both local and systemic mechanisms can be found in several excellent reviews.[118-121] In brief, at least three photoreceptors located at or near the skin surface are involved – DNA, *trans*-urocanic acid and membrane components. On absorption of photons, the respective structural changes include formation of thymine dimers in DNA, isomerisation of *trans* to *cis*-urocanic acid, and lipid peroxidation in membrane components. These alterations initiate the pathway and stimulate the local production of the large range of immune mediators shown in Figure 2-3. Such molecules have profound effects on various cell populations in the irradiated site and possibly elsewhere in the body. In particular, there are changes in the numbers and function of the APCs which lead to alterations in particular T lymphocyte subsets. For example, inhibition in the release of certain (type 1) cytokines from T helper 1 (Th1) cells occurs. This is significant as the type 1 cytokines are very important in the responses to simple chemicals, such as nickel, and in the immunological control of tumours and intracellular infections, such as those caused by viruses. At the same time, T_{reg} cells are stimulated to release immune mediators that are involved in the control of other T-cell subsets. Upon activation by a specific antigen, these T_{reg} cells are capable of down-regulating immunity by the production of immunosuppressive cytokines. There is much interest currently in trying to characterise populations of T_{reg} cells, particularly as they may have therapeutic value in the treatment of autoimmune and other diseases.[122]

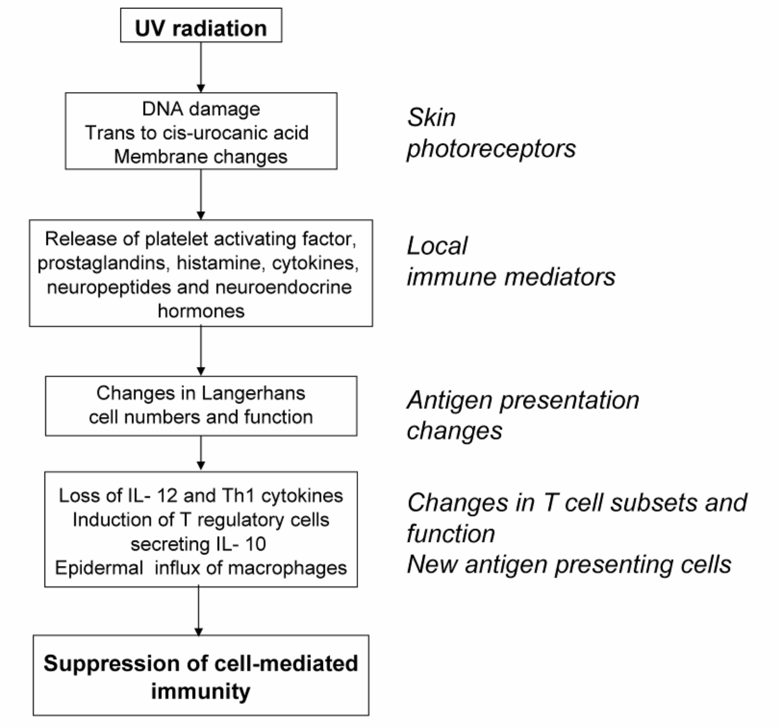

Figure 2-3. Outline of pathway leading to local immunosuppression (antigen applied to the irradiated skin site) following UV irradiation.

The majority of experimental systems to date have involved a single or a limited number of exposures to UVR, containing the UV-B waveband predominantly, and in doses sufficient to cause sunburn (erythema) followed by application of the test antigen. Under natural conditions, people are exposed to solar UVR in which the UVB represents less than 6% of the total UV spectrum[123] and they frequently receive suberythemal doses on a daily basis, especially during the summer months. Many respond to this chronic low level exposure by tanning and by skin thickening. These responses, which provide some protection against the burning effect of UVR, might lead to photoadaptation so that protection against UV-induced immunosuppression could also develop. This possibility has been tested recently in both mice and humans. For most immune responses, photoadaptation did not occur so that the immunosuppression continued throughout a period of repeated daily exposures to suberythemal solar simulated radiation.[124, 125]

The impact of UVR on infectious diseases and vaccination of human subjects

The 2003 UNEP report[1] summarized the evidence available at that time demonstrating that solar UVR exposure could adversely affect the pathogenesis of various infections. Information on this topic reported since 2003 is outlined below. The two cases in which UVR exposure definitely causes a detrimental change in the pathogenesis are herpes simplex virus (HSV) which causes cold sores and human papillomavirus (HPV) which commonly causes warts. In both cases, UV appears to have dual effects – both on the immune response and on the virus itself, and these mechanisms are outlined below. The apparent inability of UVR to alter the course of other human infections could be because the causative agents themselves do not contain any UV responsive elements, or that the human immune system is sufficiently robust so that if one aspect of it is suppressed, another can compensate. It should be noted, however, that only a limited number of human infections have been investigated thus far in the context of solar UVR.

Herpes simplex virus (HSV). Several epidemiological and experimental studies have indicated that exposure to solar UVR exposure is a common stimulus for the reactivation of HSV type 1 from latency in the nerve tissue. The virus then travels down the sensory nerve and replicates in the skin to induce a recurrent lesion (cold sore) at the same site as the initial infection had occurred. A large-scale study of 3678 infected patients, 2656 of whom suffered HSV recurrent lesions, was undertaken recently in a Prefecture of Japan to further evaluate the role of solar UVR exposure.[126] The self-reported cause of the recurrence of cold sores was the sun in 10.4% of individuals. In the summer months, this rose to 19.7% overall, and to 40% in subjects younger than 30 years. One mechanism likely to be important here is the suppression of local immune responses as a result of UV exposure: the virus arriving at the cutaneous site from the nerve will have time to replicate and to induce the clinical symptoms before effective immunological control is regained.[127, 128] Such a scenario has been shown to operate in mice infected cutaneously with HSV and then UV irradiated.[129] New studies indicate that a second mechanism involving a more direct interaction between HSV and UVR is probably required to reactivate the virus, in addition to the immune effects of the UVR. For example, UV-induced damage to nerve endings can lead to changes that result in the activation of HSV promoters, and hence to the reactivation of the virus from latency.[130]

Human papillomavirus (HPV). It has been recognised for several years that, in immunosuppressed subjects and those with epidermodysplasia verruciformis (EV; a rare genetic disease in which the APCs are defective), infections with certain cutaneous HPV types (EV-HPV) are associated with the development of SCCs but only on body sites most exposed naturally to sunlight, such as the face and backs of the hands. New information has now provided evidence that immunocompetent individuals can be similarly affected, i.e., UVR exposure and infection with certain cutanous HPV types can act as co-factors in the development of not only SCCs and but also of BCCs (reviewed in[131]). The interactions here are complex but, in brief, the HPV is able to stimulate cell proliferation and inhibit UV-induced programmed cell death (apoptosis) in the epidermis. These properties, together with the local immunosuppression and the additional genetic changes induced by the UVR exposure, may lead to tumour progression. Furthermore, on the basis of a lifetime-retrospective questionnaire on sun exposure, it has been suggested that sunburn episodes in the past lead to an increase in the risk of infection with particular HPV types in healthy subjects.[132]

The conjunctiva of the eye represents a further site where an association between HPV, SCC and sun exposure is probable. Conjunctival SCCs from subjects in Uganda, where the sunlight exposure is very high, were analysed for particular *P53* mutations (CC→TT) as a molecular signature of mutagenesis by solar UVR.[133] The prevalence of CC→TT transition (56%) was the highest observed in any of the cancer types evaluated and matched that of skin cancers in XP patients (see section below, The impact of UVR on tumour immunity). In addition EV HPV types were found in 86% of cases of SCCs of the conjunctiva.[134] It was suggested that these results confirm the causal role of solar UVR exposure in SCC of the conjunctiva and lead to the conclusion that the HPV infection could act as a co-factor in the mutagenesis process.

Recently an unexpected interaction between HPV types and solar UVR exposure has been revealed. Hrushesky and colleagues in the Netherlands observed a seasonal fluctuation in the frequency of cervical smears that were positive for the anogenital HPV types: it was twice as high in the summer months with a peak in August.[135, 136] There was a positive correlation between the monthly HPV detection rate and the monthly solar UVR exposure. Hrushesky et al. speculate that UV-induced systemic immunosuppression could be the main reason for the increase in active HPV infections in the cervix in the summer months. This finding could be of importance as the high-risk anogenital HPV types are recognised to be the primary cause of carcinoma of the cervix, a tumour that is estimated to kill about 500,000 women annually worldwide.

Vaccination. To date, only one large-scale experimental study, carried out in the Netherlands, has evaluated whether solar UVR exposure can affect the generation of immune responses to vaccines.[137] In brief, subjects were vaccinated with recombinant hepatitis B surface antigen following whole-body UV irradiation on five consecutive days in half of the individuals. While natural killer cell activity and contact hypersensitivity responses were suppressed in the irradiated subjects compared with the unirradiated subjects, there was no difference between the two groups in the hepatitis B-specific T cell or antibody responses. However, when the subjects were genotyped to characterise their cytokine polymorphisms (which can affect cytokine production or activity), it was found that individuals with a particular interleukin-1β polymorphism showed suppressed antibody responses to hepatitis B virus, if exposed to UVR prior to the vaccination.[138] Furthermore when skin samples were assessed for *cis*-urocanic acid concentration (which acts as major photoreceptor for UVR in the skin and can initiate the cascade resulting in immunosuppression), UV-irradiated subjects with higher *cis*-urocanic acid levels had suppressed T cell responses to the vaccine.[139] These results indicate that there are genetic and other differences in the way in which an individual might respond to vaccination in the context of UVR exposure. Therefore, it may not be appropriate to put all the irradiated or unirradiated subjects into single groups in order to make valid comparisons regarding UV-induced effects on immune responses during vaccination.

Three further studies are of interest. Sharma et al.[140] investigated an outbreak of measles in children in an Indian city and found that one-third of the cases had occurred in individuals who had been vaccinated previously against measles and who should have been protected as a result. They suggested that the virus-specific immunity could have waned due to solar UV-induced suppressive effects although experimental evidence is required to substantiate this idea. Snopov et al.[141] studied plasma cytokine levels following measles and poliovirus vaccination in infants in St Petersburg, Russia, some of whom had received ten daily suberythemal whole-body exposures to UV lamps (emitting predominantly UV-B) prior to the vaccination. This procedure was thought to improve the general health of such children. A shift towards a Th2 cytokine response occurred in the infants who had been UV exposed, but without the development of any clinical symptoms; antibody titres were not measured. Finally Ghoreishi and Dutz[142] demonstrated recently that if mice were immunised with a protein applied directly to UV-irradiated skin together with an adjuvant (the trancutaneous route), immune responses to that protein were not generated. This outcome was mediated by T_{reg} cells that function through the production of the immunosuppressive cytokine, interleukin-10. In the future, the trancutaneous route may become preferred to subcutaneous inoculation as it avoids the use of needles; thus this result is of considerable interest.

In conclusion, there is limited evidence that UVR exposure can reduce the efficacy of vaccination, at least in some individuals. Clearly, this issue requires further investigation, particularly with regard to the identification of UV-susceptible groups within a population.

The impact of UVR on tumour immunity

There is considerable evidence that UV-induced immunosuppression contributes significantly to the progression of both melanoma and non-melanoma skin cancers.[1] Recently Jans et al.[110] demonstrated that prevention of the formation of the most common UV lesion in the skin, CPDs, also prevented the vast majority of the acute responses in UV-exposed skin and increased the resistance to UV-induced tumour development. Furthermore, Kuchel et al.[143] found that CPD development is the initiating event for suppression of memory immune responses in human subjects. This study looked at the effect of UVR exposure on the memory immune response in individuals who were allergic to nickel. This means that they had already shown a cell-mediated immune response to nickel in the past and would therefore have an immunological memory of nickel. When irradiated with solar-simulated UVR and then challenged on the skin with nickel, the normal cell-mediated response (seen as reddening and inflammation of the skin) was suppressed. However, if liposomes containing a DNA repair enzyme were applied immediately after the exposure, the cell-mediated response was not suppressed.

Subjects with the genetic disease XP, in whom there are mutations affecting DNA repair, show enhanced UV-induced acute inflammation and a high incidence of UV-induced skin cancers, up to 5000 times that of the general population.[144] Application of liposomes containing a DNA repair enzyme to the exposed skin leads to a decrease in the rate of newly occurring actinic keratoses (precursors of SCC) and skin cancers compared with placebo[145] Mice with different genetic defects in nucleotide excision repair (used as animal models of XP disease) have been investigated to determine further the effect of DNA repair on UV-induced local immunosuppression.[146] In another example, transgenic mice with a defect in one form of nucleotide excision repair have been used to demonstrate that tumour cells, derived from a murine UV-B-induced SCC, first develop into tumours following subcutaneous injection, and then are subsequently rejected in exactly the same fashion as in the wild-type.[147] However, if the transgenic and wild-type mice were UV-B exposed prior to the tumour cell inoculation, the tumours were rejected in 40% of the transgenic mice, as compared to 96% rejection in the wild-type mice. It was concluded that this immune-mediated impairment in tumour rejection, induced by the lack of repair of the DNA damage following the UV exposure, could contribute significantly to skin cancer development in XP patients. This work on XP represents further compelling evidence that the immunosuppression caused by UVR can be a crucial factor in the generation of skin cancers.

Vitamin D

Although exposure to solar UVR has many adverse health effects in human populations, one very beneficial effect is its contribution to vitamin D status. The vitamin D status of an individual is based on measuring the serum or plasma concentration of 25-hydroxyvitamin D [25(OH)D]. The active form of vitamin D, 1,25-dihydroxyvitamin D [$1,25(OH)_2D$] is synthesised in the final step of the metabolic pathway. The levels of $1,25(OH)_2D$ are maintained even when 25(OH)D levels become sub-optimal. Currently the serum levels of 25(OH)D considered excessive, sufficient, insufficient and deficient are >250, 50-250, 25-50 and <25 nmol/L respectively.[148-150] These values are the topic of continuing discussion. For example, a recent report indicates that the most advantageous serum concentration of 25(OH)D for a number of health endpoints begins at 75 nmol/L with the optimum between 90-100 nmol/L.[151] For most people, more than 90% of their vitamin D requirement is acquired from exposure to solar UVR.

An action spectrum for vitamin D formation in human skin indicates that synthesis occurs most effectively following exposure to the UV-B waveband.[152] As solar UV-B is reduced to almost zero in the winter months at latitudes above 50^0 North or South[153], vitamin D status can vary greatly with season and location. Various surveys have provided evidence that many individuals, even those living in countries with high solar UVR[154], may have inadequate vitamin D status.[155-159] Because of its ability to absorb UV-B, melanin in the skin can also decrease vitamin D status although, with sufficient UV-B, adequate vitamin D status can be achieved.[160] As an example of the effect that skin pigmentation can have on vitamin D production, 42% of Black American women were considered 25(OH)D-deficient compared with 4.2% of white women in a recent survey.[161]

Vitamin D was identified almost one hundred years ago, and the link between sunlight exposure and childhood rickets proposed about four hundred years ago. Vitamin D is a very important hormone for many aspects of general health. It plays a major role in the growth, development and maintenance of bone, with deficits leading to low bone mineral density resulting in an increased risk of osteoporosis and fractures in adults and rickets in children. However adequate vitamin D status is now implicated in the prevention of an increasing list of non-skeletal disorders including several internal cancers and autoimmune diseases, and hypertension. $1,25(OH)_2D$ most commonly acts as a factor that stimulates cell differentiation and cell death.

Immune effects

Following the discovery of vitamin D receptors (VDRs) on several populations of immune cells, it is now known that vitamin D status can affect the immune system by suppressing T-cell proliferation, down-regulating antigen presentation, stimulating the generation of T_{reg} cells and Th2 cells, and activating macrophage function (reviewed in Mathieu et al.[162] Indeed, once it became known that $1,25(OH)_2D_3$ can be synthesised in the skin following UVR, it has been suggested as a mediator of UV-induced immunosuppression. One illustration of this aspect is its inhibitory effects on the ability of Langerhans cells (which form a dendritic cell network in the outermost layers of the skin and survey the skin for any foreign challenges) to present antigens.[163]

Cancer

The most persuasive evidence to date suggesting a protective role for vitamin D in human disease relates to some internal cancers. Most information is available for colon, breast, prostate and ovarian tumours. Recently Garland et al.[164] undertook a review of relevant epidemiological studies and concluded that 20 out of 30 studies on colon cancer, 9 out of 13 on breast cancer, 13 out of 26 on prostate cancer and 5 out of 7 on ovarian cancer reported a significant benefit of vitamin D, its serum metabolites, sunlight exposure or another marker of vitamin D status on cancer risk or mortality. The other studies demonstrated a favourable trend (of borderline significance) or no association with vitamin D or its markers. A second recent review found a significant inverse correlation between sunlight exposure and the incidence or mortality of prostate, ovary and colon cancers with the data on non-Hodgkin lymphoma giving conflicting results.[165] Vitamin D might provide a protective effect by controlling cell proliferation, inducing terminal differentiation of tumour cells and inhibiting angiogenesis. There are many VDR polymorphisms, and only particular genotypes of VDR in combination with low 25(OH)D levels may correlate with the increased risk of cancer or metastasis. Notably one large longitudinal case-control study in the Nordic countries found that the risk of prostate cancer was greatest in two groups: those men with a low serum 25(OH)D (below 19 nmol/L) and those with a high serum 25(OH)D (above 80 nmol/L).[166] In addition, the results of a very recent large randomised double-blind placebo-controlled trial taking place in post-menopausal women showed that the incidence of colorectal cancer in the 18,176 individuals assigned to receive calcium carbonate plus vitamin D_3 (400 IU) daily was no different from the 18,106 individuals assigned to the placebo group.[167] This finding has been criticised as the daily dose of vitamin D taken by the subjects was lower than that recommended by some experts.[168, 169] Furthermore a meta-analysis of 44 observational studies of either prospective (cohort) or retrospective (case-control) design concluded that individuals taking >1000 IU/day oral vitamin D or with >82 nmol/L serum 25(OH)D had 50% lower incidence of colorectal cancer compared with reference values.[170] In addition to vitamin D status, consideration of calcium status may be of crucial importance in the prevention of internal cancers, as has been demonstrated for colorectal adenomas.[171]

The majority of the epidemiological studies linking low UV exposures to higher incidence of internal cancers used latitude as a surrogate for exposure rather than measuring personal UV dose. However, several reports have now tried to include a personal estimation of sun exposure. In a case-control Australian survey, the risk of non-Hodgkin lymphoma fell with increasing solar irradiation, as assessed via a self-administered questionnaire and telephone interview.[172] As already noted in a previous section, Berwick et al.[45] reported that, following the diagnosis of early stage cutaneous melanoma, sun exposure was associated with increased survival rates over an average of a 5-year period. The irradiation was assessed by personal interview and a review of histopathological parameters, such as solar elastosis. Rukin et al.[173] assessed various parameters regarding past sun exposure, via a questionnaire, that might affect susceptibility to prostate cancer: in men with very low UV exposure, polymorphisms in particular subregions of the VDR gene were associated with risk. The difficulties of accurately estimating past personal UV exposure have already been indicated in this report. The finding of a significant protective effect is thus of some importance. Furthermore the inclusion of objective measures of past sun exposure such as solar elastosis provides further weight to this conclusion. No studies in animals have attempted to assess a protective role for solar UV exposure in internal cancer development, although several such studies have shown that vitamin D has activity against tumour proliferation and metastasis (reviewed in Giovannucci[174]).

Autoimmune diseases

As discussed above for the internal cancers, a protective role for vitamin D status is postulated for some autoimmune diseases, namely multiple sclerosis (MS), diabetes mellitus type 1, rheumatoid arthritis (RA) and inflammatory bowel diseases (IBDs). A brief overview of each is given below.

MS is an autoimmune disease in which an overactive Th1 cytokine response to an unidentified antigen stimulates an immune attack on myelin in the central nervous system. Initial epidemiological studies in human populations using latitude as a surrogate for solar UV exposure[175] and experimental studies in a mouse model of MS (experimental allergic encephalomyelitis, EAE)[176] support the view that there is a link between poor vitamin D status, due to low sunlight exposure, and MS incidence. New evidence has indicated that increased sun exposure during ages 6-15 years is associated with a decreased risk of MS.[177] In a prospective cohort study of almost 20,000 nurses in the USA, Munger et al.[178] revealed that vitamin D supplements (>400IU/day vs. nil) after the age of 25 was inversely associated with MS onset (40% decrease in risk). Also a record linkage study of skin cancer and MS has revealed that skin cancer incidence is significantly less common in MS patients than in those patients with other autoimmune or neurological diseases.[179] It is postulated that $1{,}25(OH)_2D$ could act by suppressing Th1 function while concurrently increasing T_{reg} and Th2 activities, thus helping to reduce the risk of MS development. However, in one of the few animal model studies to date in which UVR was incorporated, UV exposure induced progressive disease in some mice that had already developed the relapsing-remitting form of EAE.[180] It was shown that systemic immunosuppression had resulted from the UVR. These findings were explained by suggesting that Th1 responses contribute to disease onset while Th2 responses that are promoted by UVR may be more important in disease progression. It should be noted that in most of the mouse studies of EAE, $1{,}25(OH)_2D_3$ was added to the diet rather than vitamin D_3, the metabolite formed in the skin after UV exposure, and calcium supplementation was also provided. A recent paper reports that dietary vitamin D_3 provided protection from the development of EAE in female mice, but not in ovariectomised female mice or in male mice.[181] Thus a complex relationship between vitamin D and female hormones may be indicated.

For type 1 diabetes, epidemiological studies show increased incidence at higher latitude, the converse to skin cancer incidence. Added to this, convincing evidence from models of non-obese diabetic mice demonstrates that vitamin D deficiency in early life accelerates the appearance of the disease.[182] A birth-cohort study in Finland indicated that regular vitamin D intake in early childhood reduced the risk of type 1 diabetes development in later life.[183] Two other reports show the protective effects of vitamin D or cod-liver oil (rich in vitamin D) in type 1 diabetes.[184, 185] No studies have attempted to relate individual solar UVR dose with type 1 diabetes in humans or animal models thus far.

Unlike MS and type 1 diabetes, the incidence of rheumatoid arthritis (RA) does not correlate convincingly with latitude.[186] However, a prospective large-scale study has revealed an inverse association between vitamin D intake and RA.[187] As the symptoms of RA are largely due to the overactivity of the Th1 cytokines, especially tumour necrosis factor-α, low levels of $1{,}25(OH)_2D$ may not be sufficient to suppress this imbalance.

Inflammatory bowel diseases (IBDs) have an unknown aetiology but are immune-mediated and consist of at least two forms, ulcerative colitis and Crohn disease. A mouse model in which the VDRs are not expressed has been used to illustrate the importance of vitamin D for the maintenance of normal immune responses in the gastro-intestinal tract.[188] In another mouse model, $1{,}25(OH)_2D_3$ prevented and ameliorated the symptoms of IBD.[189] Therefore it is possible that a vitamin D deficiency may lead to a lack of suppression of the enhanced Th1 cytokine responses that are typical of IBDs in humans. The role of sunlight in IBD has not been examined experimentally, although it is known that IBDs have a complex aetiology involving environmental factors and are most prevalent in higher latitudes where exposure to solar UVR is reduced compared with lower latitudes.

In conclusion, for the human autoimmune diseases, MS, type 1 diabetes, RA and IBDs, there is growing, although still not definitive, evidence to associate low solar UVR exposure and/or vitamin D with occurrence. Recent cohort studies have indicated convincingly that poor vitamin D status can be prospectively associated with the onset of the first three of these diseases (reviewed in Ponsonby et al.[190]). However, it is possible that another factor, apart from vitamin D, which is also linked with sun exposure, may be involved in modulating immune responses.

Suggested factors include the UV-induced release of the neuropeptides, α-melanocytic-stimulating hormone and calcitonin-gene related peptide, or the light-induced suppression of melatonin levels.[190]

As a further indication of how complicated and confusing the links are between vitamin D deficiency and an increased risk of certain autoimmune diseases, there appear to be certain subsets of patient populations in whom the production of $1,25(OH)_2D_3$ is increased.[191-193] In those with Crohn disease, the elevated $1,25(OH)_2D_3$ is associated with low bone density and active disease which Abreu et al.[193] suggest may arise from inflammation occurring in the intestinal tract. In patients with sarcoidosis, elevated vitamin D was seen more frequently in those with extrathoracic involvement, a more serious form of the disease.[192]

Infectious diseases

Few studies to date have considered vitamin D in the context of infectious diseases, although Cantorna et al.[189] found that the susceptibility of mice to infection with HSV or the yeast *Candida albicans* was not affected by $1,25(OH)_2D_3$ given in the diet. However, historically vitamin D has been used to treat tuberculosis and there is more recent evidence that $1,25(OH)_2D_3$ can activate anti-mycobacterial activity in a murine model[194] and in cattle infected with *Mycobacterium bovis.*[195] An explanation of how this mechanism might operate has been provided using a mycobacterial model system. It has been shown that the activation of Toll-like receptors on human macrophages by mycobacterial lipopeptides leads to the up-regulation of the VDRs and vitamin D hydroxylase genes, resulting in the activation of the macrophages and the killing of the intracellular bacteria.[196] Several surveys have shown that, in temperate climates, the incidence of tuberculosis is higher in human subjects with low serum 25(OH)D levels[197], and a recent study involving foreign-born people living in London concluded that 25(OH)D deficiency correlated with tuberculosis amongst all ethnic groups, except white Europeans and Chinese/South Asians.[198] The lack of solar UVR exposure is likely to contribute to the low levels of vitamin D, but poor dietary intake may be important and particular VDR polymorphisms may provide a genetic risk factor for some ethnic groups. An interesting recent review suggests the hypothesis that the occurrence of epidemic influenza predominantly in the winter months might be explained by the seasonal deficiency in vitamin D, leading to a significant reduction in several anti-viral immune mechanisms.[199]

Safety of response strategies

Response strategies to deal with the problems arising from ozone depletion can be divided into those that are directed at restoring the appropriate level of ozone in the stratosphere by replacing ozone depleting substances (ODSs) with alternative chemicals, and those that are directed at protecting individuals from the increased solar UV-B arising from ozone depletion. Both strategies may have unintended consequences for human health. The sections below will summarize the safety aspects associated with the development and use of ODS replacement chemicals and then will discuss some of the issues associated with various personal protection strategies for the eye and the skin.

ODS replacement chemicals

Much of the safety testing of many of the substitutes for ODSs, for example HCFC-124, HFC-134a and HFC-227, continues to find low toxicity in humans and animals.[200, 201] However, there has been an increasing number of reports indicating that use or exposure to HCFC-123, in particular in occupationally exposed populations, can be associated with liver toxicity.[202-204] As the number of chemicals being proposed as replacements for ODSs is steadily increasing (EPA 2004, available at www.epa.gov/ozone/snap), it will be important to monitor their use for adverse events. This is particularly true for those chemicals that have seen limited use in the past and for which exposure and toxicity information is limited.

Personal protection strategies

Many of the protective strategies against excessive exposure to sunlight have been developed and advocated by those concerned about the effects of UVR on the skin. The first step towards protection from any toxic agent is to be aware that the hazard exists. The general advice to *seek shade* has become a keynote slogan for those involved in sun safety; this has been an effective addendum to the popular Australian *slip, slap, slop* campaign (now modified by New Zealand to be *slip, slap, slop* and *wrap*). The equivalent programme in the USA is called SunWise and it seeks to teach the public, especially children, how to protect themselves from overexposure to the sun (http://epa.gov.sunwise/).

Most public health pamphlets now include a reference to the need for hats and sunglasses. Wide brimmed (>10 cm) hats are recommended for head and eye protection and can reduce ocular exposure by up to 50%.[205] Protection from side-angles of UVR is often provided by the hood of a jacket and similar headwear. Although, as discussed above, there have been concerns that under-exposure to UV-B may impair vitamin D status, one recommendation suggests that 10-15 minutes per day in sunlight in the summer months should be sufficient to maintain adequate vitamin D status for most individuals.[164] This dose relates to white-skinned people living in countries such as north-west Europe and the USA, with exposure on unprotected skin. It should be modified considerably for those living at high or low latitude, for the season of the year and for immigrants with darker skin colour. In addition age, type of clothing, diet, whether the work place is in- or out-doors and the social environment are all important variables in determining how much ambient UVR exposure is optimal. One recent study illustrates the complexity of estimating recommended UV exposure times for the Australian population, and concludes that a single simple message for the general public is not possible.[206]

The skin and eye normally have some defences against oxidative and photo-induced damage. These include pigments such as melanin, antioxidant enzymes such as superoxide dismutase and catalase, and antioxidants such as vitamins C and E, lutein, β-carotene and other carotenoids, and glutathione. Many of these defences begin to diminish after 40 years of age resulting in less protection from radiation-induced damage to various structures of the eye.[207] The use of antioxidants, free radical scavengers and trace minerals, principally via the diet, appear to be effective in reducing the immunosuppressive effects of UVR as well as UV-B induced skin carcinogenesis[208]; no evidence was found of a similar effect for cataract or other UV-B-related ocular diseases. However, recent clinical and experimental data suggest that modifying a person’s antioxidant status via supplementation may require extreme caution as the antioxidant defence system is complex and intricately balanced, and altering it may actually make the carcinogenic impact of UV worse.[208]

Protection specific to the eye. The eye is located in the bony orbit, and the forehead, eyebrows, lids and eyelashes provide considerable protection from overhead solar irradiance.[209] This explains why solar exposure at levels that should produce corneal damage within minutes, if the exposure were directly onto the cornea, does not do so. The need to protect ocular tissues from excessive exposure to UVR using appropriate absorptive glass and plastic materials is generally accepted and well understood.[210, 211] Plastic lenses absorb up to about 350 nm and most high refractive index plastic (including polycarbonate) and glass lenses absorb even more UV-A. Thus, even clear spectacle lenses provide protection from UV-B. However, in the case of non-wrap around spectacles there is potential for ambient UVR to enter the eye from the side. This effect can be exacerbated by tinted sunglass lenses, which provoke a wider opening of the eye. This is particularly significant for the potential exposure of the crystalline lens from peripheral rays. Dose estimate factors have been proposed for the efficacy of a wide range of forms of eye protection, i.e., from ordinary glass spectacles to highly protective ski goggles.[210]

Most early contact lens materials, other than fluorosilicone acrylate, provided little protection from UVR. As a result, rigid and soft contact lenses have now been developed which offer various levels of protection from UVR. Consideration of the optical absorption characteristics of a given lens and the related protection factors may be used to predict the protection afforded by a given lens. This has been confirmed by Walsh et al.[212] using modelled and measured data under high levels of solar UVR in the summer months in Houston, Texas. Rigid contact lenses provide no protection for the peripheral cornea or from the effects of peripheral light focussing. On the other hand, soft contact lenses that cover the entire cornea will protect the eye from UVR entering from the side or below. Using model eye and mannequin studies, Kwok et al.[7] have demonstrated that UVR-blocking soft contact lenses effectively shield against peripheral corneal focussing of obliquely incident UVR in the anterior segment of the eye. They also re-emphasise that many sunglasses do not protect against these rays and that contact lenses would provide protection when sunglasses are not worn. Sliney[210] concluded that UVR-blocking soft contact lenses provide protection from UV-B equivalent to ski-goggles for the cornea and internal eye structures.

Protection specific to the skin. Broad spectrum sunscreens are being used increasingly by the general population to minimise the erythemal effect of high sun exposure. They are generally effective for that end-point but concerns have been expressed that regular sunscreen usage may impair cutaneous vitamin D synthesis, if the cream is applied at the correct concentration. While some reports indicate that sunscreens significantly decrease the production of 25(OH)D and 1,25(OH)$_2$D$_3$ [213], others found little effect on the levels of these two substances.[214, 215] Farrerons et al.[216] followed two groups of elderly subjects living in Barcelona, one treated with sunscreen and the other without treatment. The sunscreen users showed a minor decrease in serum 25(OH)D levels in both the summer and winter months compared with the controls, but this reduction was not sufficient to induce secondary hyperparathyroidism. It should be noted that, in practice, sunscreen application is frequently problematic with insufficient quantity being used to achieve the sun protection factor rated, or the spreading being non-uniform resulting in some skin sites getting little or no protection, or to some being washed or towelled off.[217, 218]

Efforts have been made to define sunscreens in terms of their ability to protect against UV-induced immunosuppression. The immune protection factor (IPF) has been developed in an attempt to compare one preparation with another[219] The IPF is defined as the ratio of UV doses influencing a particular immunological end-point in the presence or in the absence of the sunscreen. Using delayed hypersensitivity as an example, several reports indicate that sunscreens that absorb the UV-A waveband offer the most effective immunoprotection (reviewed in [220]). Of course, protection against the immune effects of solar UVR might not be beneficial if consideration of protection against selected internal cancers, autoimmune and infectious diseases is taken into consideration. Apart from sunscreens, there is considerable interest currently in identifying dietary constituents that could protect the skin's immune system against UV damage.[221, 222]

Some concerns have arisen about unintended consequences from the increased use of sunscreens to protect against UV-B. A number of the UV-B absorbing components in sunscreens have been shown to have weak estrogenic activity so may have adverse consequences for reproductive function in human and animal populations in the environment, lending strength to the recommendation that protection from UV-B should not rely solely on sunscreen use.[223-230] As discussed above, there are many protective strategies for the skin that do not have unintended consequences for the environment. These include staying indoors, wearing clothing that covers sun-exposed areas of the body during conditions of high ambient UVR or seeking shade during the middle hours of the day although this will provide partial protection only.

Possible interactions between climate change and ozone depletion

If the predicted higher ambient temperatures in summer due to global warming are combined with drier weather, people living in mid-latitudes may spend more time outdoors, thus increasing their solar UV exposure. Indeed, it has been shown, at least in schoolchildren in the UK, that climate and ambient temperature influence behaviour and hence sun exposure more than ambient solar UV.[217] While such behavioural adaptation may have benefits in terms of vitamin D synthesis, the impact on skin cancer incidence and other health aspects of solar UVR are predicted to be adverse. There is also the possibility that climate change may result in wetter weather with more individuals staying indoors. Also, there would be regional differences in behavioural responses to warming.

In the previous Report (UNEP 2002, published in de Gruijl et al.[1]) the possibility that rising temperatures due to global warming might enhance the induction of skin cancer by solar UVR was considered. This suggestion was based on experiments in mice performed many years ago.[231, 232] As the process of UV-carcinogenesis is similar in mice and humans, rising temperatures could have a similar impact on skin cancers in humans, but the effect might be quantitatively different. Data on the influence of temperature on UV-carcinogenesis in human populations are not available but it is possible to investigate skin cancer incidence in people of similar skin colour living at different altitudes.[233] An attempt was made to find some indication from existing results: the incidence of NMSC in fair-skinned males and females in 10 different, well distributed regions of continental USA, collected in the Third National Cancer Survey[234], has already been compared with UV-B measurements in the same region. In the new analysis, temperature data for these regions were added (van der Leun et al. personal communication). It was discovered that there was a similar trend to that in the mouse experiments towards a higher incidence of NMSC at relatively high temperatures compared with relatively lower temperatures. These preliminary results on human skin cancer reinforce the suggestion that the interaction of temperature and solar UV radiation may become an important health effect due to climate change. In addition it should be noted that, following the work of Sasaki et al.[235], higher ambient temperatures as a result of global climate change may interact with UVR exposure to further increase the risk of nuclear cataract development.

As temperatures increase, changes in the quality and quantity of pest infestations are likely to require the increased use of pesticides. There are recent reports that exposure to certain pesticides can result in immunosuppression, and, in the case of permethrin, that such immunosuppression[236] may be additive to that caused by exposure to UV-B.[237]

Conclusions and gaps in knowledge

In the four years since our last report, considerable progress has taken place regarding the impact of ozone depletion, and hence of increased solar UV-B, on human health. The mechanisms whereby UVR interacts with structures in the eye and causes a variety of ocular diseases are becoming clear, as are details regarding the genetic basis of skin cancers and the pathways leading to UV-induced immunosuppression. The suggested links between solar UVR exposure, vitamin D and protection against a variety of internal cancers, autoimmune diseases and infection require further confirmation. In Table 2, we indicate areas where crucial knowledge is lacking.

Despite the distinct possibility that the ozone layer will repair itself in the coming decades, the general public will still require to maintain vigilance regarding their sunlight exposure. While it remains fashionable, for example, to have a tanned skin, to wear minimal clothing in hot weather and to experience holidays in the sun, the risk of overexposure of the white population is high. The projection of a doubling in the incidence of all three types of skin cancer in the next ten years, plus a large increase in the number of cataracts, due partly to an ageing population, mean that health campaigns that stress the harmful effects of solar UVR are required and justified. However, to maintain sufficient vitamin D levels, the protective measures employed by an individual should not go to the extreme of minimal or no solar UVR exposure in the summer months.

Table 2. Suggested current gaps in knowledge regarding solar UVR and human health

Subject	Key questions
The eye	What are the pathogenic mechanisms involved in the cataract types?
	What are the wavelength dependencies for cataract development?
	What are the associations between UVR and other environmental factors that contribute to the induction of nuclear cataract in residents of developing countries?
The skin	What is the action spectrum for induction of melanoma?
	Are there any interactions between UV-A and UV-B in the induction of non-melanoma skin cancer and melanoma?
	What are the pathogenic mechanisms underlying infant vs adult UVR exposure in skin carcinogenesis?
	What is the mechanism of the interactions between UV-B and UV-A with regard to effects on immunity?

	What is the action spectrum for the synthesis of vitamin D3 in pigmented and unpigmented skin?
	How much solar UVR exposure is required, and how should it be distributed over the year, to maintain adequate vitamin D levels in people of different skin phototypes living at different latitudes?
	Can valid estimates be given to the general public regarding optimal doses of solar UVR for vitamin D synthesis while reducing the risk of developing skin cancer?
	What is the effect of solar UVR in animal models of auto-immunity and internal cancers?
Protective measures	Should the immune protection factors of topical sunscreens be measured and publicised?
	Are there factors in the diet that could give significant protection against the harmful effects of solar UVR?
	Should the UV Index be used and analysed in developing countries, and should attempts be made to educate the general public regarding its meaning?
Climate change interactions	What are the combined effects of solar UVR and temperature on the skin and the eye?
	Will future changes in climate lead to people in mid-latitudes spending more time outdoors?

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Chapter 3. Terrestrial ecosystems, increased solar ultraviolet radiation, and interactions with other climate change factors

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Summary

There have been significant advances in our understanding of the effects of UV-B radiation on terrestrial ecosystems, especially in the description of mechanisms of plant response. A further area of highly interesting research emphasizes the importance of indirect UV radiation effects on plants, pathogens, herbivores, soil microbes and ecosystem processes below the surface. Although photosynthesis of higher plants and mosses is seldom affected by enhanced or reduced UV-B radiation in most field studies, effects on growth and morphology (form) of higher plants and mosses are often manifested. This can lead to small reductions in shoot production and changes in the competitive balance of different species. Fungi and bacteria are generally more sensitive to damage by UV-B radiation than are higher plants. However, the species differ in their UV-B radiation sensitivity to damage, some being affected while others may be very tolerant. This can lead to changes in species composition of microbial communities with subsequent influences on processes such as litter decomposition. Changes in plant chemical composition are commonly reported due to UV-B manipulations (either enhancement or attenuation of UV-B in sunlight) and may lead to substantial reductions in consumption of plant tissues by insects. Although sunlight does not penetrate significantly into soils, the biomass and morphology of plant root systems of plants can be modified to a much greater degree than plant shoots. Root mass can exhibit sizeable declines with more UV-B. Also, UV-B-induced changes in soil microbial communities and biomass, as well as altered populations of small invertebrates have been reported and these changes have important implications for mineral nutrient cycling in the soil. Many new developments in understanding the underlying mechanisms mediating plant response to UV-B radiation have emerged. This new information is helpful in understanding common responses of plants to UV-B radiation, such as diminished growth, acclimation responses of plants to UV-B radiation and interactions of plants with consumer organisms such as insects and plant pathogens. The response to UV-B radiation involves both the initial stimulus by solar radiation and transmission of signals within the plants. Resulting changes in gene expression induced by these signals may have elements in common with those elicited by other environmental factors, and generate overlapping functional (including acclimation) responses. Concurrent responses of terrestrial systems to the combination of enhanced UV-B radiation and other global change factors (increased temperature, CO_2, available nitrogen and altered precipitation) are less well understood. Studies of individual plant responses to combinations of factors indicate that plant growth can be augmented by higher CO_2 levels, yet many of the effects of UV-B radiation are usually not ameliorated by the elevated CO_2. UV-B radiation often increases both plant frost tolerance and survival under extreme high temperature conditions. Conversely, extreme temperatures sometimes influence the UV-B radiation sensitivity of plants directly. Plants that endure water deficit stress effectively are also likely to be tolerant of high UV-B flux. Biologically available nitrogen is exceeding historical levels in many regions due to human activities. Studies show that plants well supplied with nitrogen are generally more sensitive to UV-B radiation. Technical issues concerning the use of biological spectral weighting functions (BSWF) have been further elucidated. The BSWF, which are multiplication factors assigned to different wavelengths giving an indication of their relative biological effectiveness, are critical to the proper conduct and interpretation of experiments in which organisms are exposed to UV radiation, both in the field and in controlled environment facilities. The characteristics of BSWF vary considerably among different plant processes, such as growth, DNA damage, oxidative damage and induction of changes in secondary chemicals. Thus, use of a single BSWF for plant or ecosystem response is not appropriate. This brief review emphasizes progress since the previous report toward the understanding of solar ultraviolet radiation effects on terrestrial systems as it relates to ozone column reduction and the interaction of climate change factors.

Introduction

Terrestrial ecosystems are undergoing transitions in our changing climate and are likely to be in flux in the coming decades and beyond. Much of this may be attributed to direct and indirect effects of increasing temperature and CO_2. The influence of increased solar UV-B radiation is superimposed on these important drivers of our changing terrestrial ecosystems. Much has been learned about how plants and other organisms respond to UV radiation at the molecular and physiological levels and several studies have focused on the implications of interactions of vegetation with animals and microbes. Ecosystems are being confronted with several aspects of climate change simultaneously, resulting in interactive responses to environmental factors such as UV radiation, increasing temperature, CO_2, and changing precipitation patterns. Nitrogen is also considered, since biologically available nitrogen is increasing in more inhabited areas of the globe due to factors such as air pollution and agricultural application of nitrogen. Experiments have been conducted on agricultural and non-agricultural plants in various settings (Figure 3-1).

Ecosystem-level responses

Predictions of ecosystem consequences of enhanced UV-B radiation are necessarily complicated because of species interactions and the way in which living organisms affect and are affected by the abiotic components of the ecosystem, e.g., soils, water, mineral elements, etc. Ecosystem-level effects can best be examined by direct, manipulative experiments conducted in the field in intact ecosystems. Sometimes these experiments are confined to small sections of intact soil, vegetation and associated microorganisms, termed “mesocosms”. These field studies have either involved supplementation with UV-B-radiation-emitting lamps or attenuation of ambient solar UV-B radiation using special selective filters (Figure 3-1a and 3-1c). In both cases, there are appropriate control treatments to account for unintended side effects of the structures used to effect the UV-B radiation manipulations.

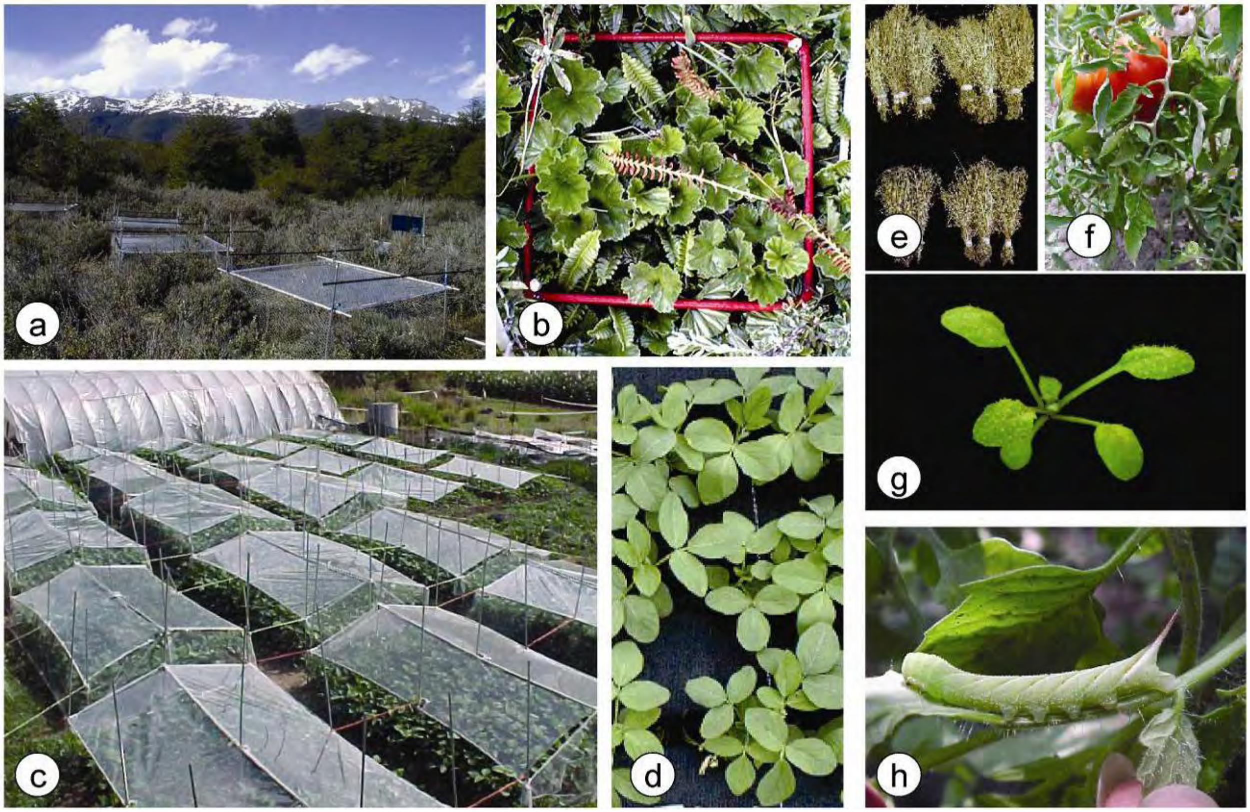

Figure 3-1. Examples of studies of the impacts of UV-B radiation on terrestrial ecosystems. The work described in this report is based on studies that addressed the effects of UV-B radiation manipulations on (a) intact natural ecosystems, (b) native plants, such as *Gunnera magellanica* and *Blechnum penna-marina*, (c) field crops, such as (d) soybean, (e) barley, (f) tomato, (g) the model plant *Arabidopsis thaliana*, and (h) several species of phytophagous insects, such as *Manduca sexta*.

Global distribution of climate change factors

Factors of climate change are being manifested across the globe in different patterns. Elevated CO_2 is fairly evenly distributed. Significant changes in the frequency and quantity of precipitation are also predicted at all latitudes because of climate change. With global warming the hydrological cycle will accelerate and general increases in precipitation are likely to occur.[1] However, there are large departures at regional scales, such that some areas will receive considerably more and some much less precipitation. Nitrogen deposition is increasingly a global issue and is particularly prominent in densely inhabited areas where agricultural nitrogen use and air pollution are the main contributors to the biologically available nitrogen in these ecosystems.[2] The relative increases in solar UV-B radiation and temperature are decidedly more pronounced at high latitudes and, accordingly, most of the more recent UV-B radiation experiments conducted at the ecosystem level have been at high latitudes. These have included locations in Antarctica and the southernmost tip of South America (Tierra del Fuego) and several

Arctic and sub-Arctic locations including northern Sweden, Svalbard, and Greenland. A few of the ecosystem-level experiments have involved manipulation of other climate change factors in addition to UV-B radiation, such as moisture.

Direct effects on organisms

Plants. Mosses, lichens, and higher plants have received most attention in assessing UV-B radiation effects on terrestrial systems. Photosynthesis of these plant groups at high-latitude sites is generally unaffected, although aboveground biomass is often reduced to a small extent by UV-B radiation in various supplementation and attenuation experiments.[3-6] Small effects on higher plant growth at high latitudes due to UV-B radiation concur with findings of many studies on plants at lower latitudes, e.g., Searls *et al.*[7] Occasionally, large growth reductions have been reported. For example, UV radiation attenuation studies with potted plants in Antarctica indicated that ambient UV-B radiation caused sizeable growth reductions.[8-11] Also, an endemic species of moss in Antarctica was found to have reduced levels of photoprotective pigments (compared to other widely distributed moss species), and current levels of UV radiation were reported to cause increased frequency of leaf morphological abnormalities.[12]

Among growth characteristics altered by UV-B radiation, reductions in plant height are often reported. Even subtle decreases in plant height due to UV-B radiation might be of eventual significance if different species are affected to different degrees as was the case in ecosystem plots of a peat bog in Tierra del Fuego.[5] In this study, when compared to the attenuated UV-B radiation treatment, higher plants were inhibited more by near-ambient solar UV-B radiation than was the moss layer. Thus, the moss could slowly engulf the higher plants; however, this would likely take several years to decades.

Both supplementation and attenuation of UV-B radiation in experimental studies may influence allocation of biomass to root systems. A few recent reports indicate large changes in root systems attributable to the UV-B radiation treatments aboveground, suggesting a systemic response to the radiation (Figure 3- 2). Root mass increased with solar UV-B radiation attenuation in experiments conducted in Greenland[13] and Tierra del Fuego.[14] In a similar vein, Ruhland *et al.*[11] reported substantial increases in root mass with reduced UV-B radiation in solar UV-B radiation attenuation experiments using potted plants in Antarctica. On the other hand, studies in Finland indicated an increase in root mass with UV-B radiation supplementation.[15] The underlying mechanisms behind these seemingly contradictory responses are not known but may be due in part to experimental differences. In all these cases, these were large relative changes — much larger than the relative changes in shoot mass generally reported as a result of UV-B radiation manipulations.

Animals. Most vertebrate animals and insects are assumed to be well protected by body coverings (fur, feathers, etc.) and pigmentation. However, amphibians, such as frogs and salamandars are much less well protected than other vertebrates and considerable controversy exists regarding how much they might be affected by exposure to ambient UV-B radiation in their natural habitats[16-18], see Chapter 4. The UV-B radiation sensitivity of soil insects such as the Arctic collembolan (springtail) species was investigated in laboratory tests.[19] The species varied considerably in pigmentation which corresponded to the degree to which they are normally exposed to sunlight. These ranged from soil-living forms that lacked apparent pigmentation to heavily pigmented surface-dwelling species often exposed to the sun. The UV-B radiation sensitivity was inversely correlated with pigmentation and well pigmented species were considered to be tolerant of UV-B radiation corresponding to solar UV-B radiation with substantially depleted ozone. Nevertheless, the author speculated that solar UV-B radiation (especially with ozone reduction) might influence the distribution of springtail species near the soil surface in heterogeneous environments. Behavioral responses to solar UV-B radiation have been reported for thrips, a leaf-eating insect that can cause severe damage to a variety of commercially important crops. Field studies demonstrated that these insects can detect and avoid exposure to the UV-B component of solar radiation, even though the natural background levels of solar UV-A and visible radiation contain much more energy, indicating that thrips have a sensory system with high UV-B radiation specificity.[20, 21] Whether or not this sensitivity is widespread among herbivorous insects is not known, but strong effects of UV-B radiation manipulations on levels of insect herbivory have been documented in a variety of ecosystems[22] and are described below.

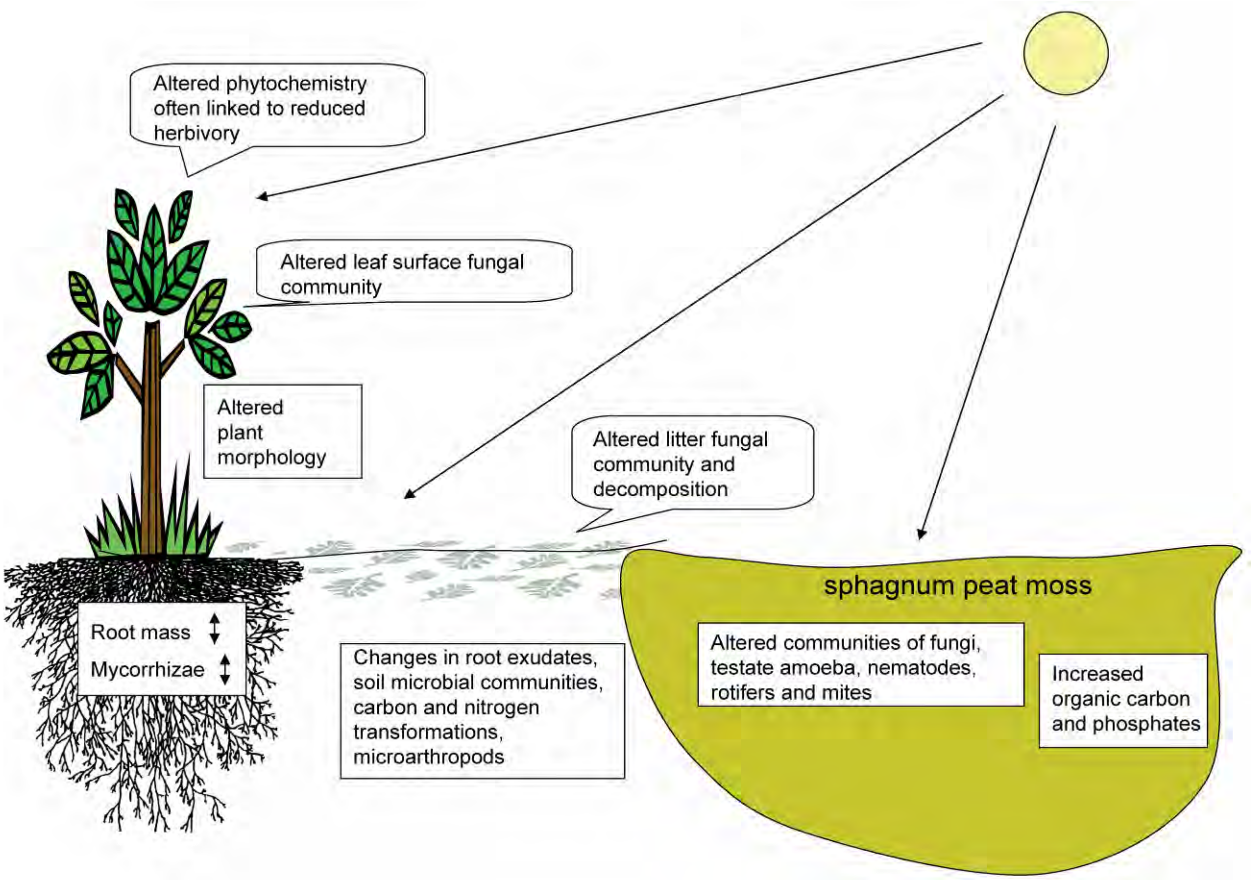

Figure 3-2. Ecosystem-level manifestations of changing the UV-B flux reaching above-ground vegetation and other surfaces. Above ground, higher plants are affected to a small degree in morphology and growth, while fungi and other microbes exposed to sunlight are often affected to a greater extent. Many effects occur below the surface of soils or peat where no sunlight penetrates. Root mass and mycorrhizal abundance can either increase or decrease with more UV-B radiation and these changes can be much larger than changes in the plant shoot system. Alterations in microbial communities, nutrient levels and fluxes as well as microinvertebrates have also resulted from UV-B radiation changes above ground. These are probably mediated by systemic changes in higher plants and in the *Sphagnum* peat.

Microbes. Unlike most photosynthetic plants (flowering plants, mosses and lichens), microbes can be quite sensitive to the UV-B radiation in direct sunlight.[23-26] However, this is usually not a uniform sensitivity among different species so that species composition changes are often observed on foliage and litter surfaces where these microbes are directly exposed to sunlight.[27-32] In the studies of Moody *et al.*[27] and Pancotto *et al.*[30], changes in microbial species composition were linked to differences in litter decomposition rates resulting from UV-B radiation manipulations. Solar UV radiation can also have direct effects promoting plant litter decomposition. This process is know as photodegradation and plays a significant role in carbon cycling in arid ecosystems[33] as discussed in Chapter 5.

Indirect effects of UV-B radiation

Though less expected, indirect effects of solar UV-B radiation are usually much more important, intricate and, indeed, fascinating than the direct effects of solar UV radiation. In addition, there is an increasing mechanistic understanding of these indirect effects that is developing as will be described later. In most cases, these effects are mediated through the plants, but can be manifested below ground as well as above ground.

Above ground

As discussed in the last report[22], UV irradiation of plants affects plant disease sensitivity and development. Although few phenomenological studies of UV radiation and plant disease have been published since the last report, there are important new developments in understanding the mechanisms of this action.

In the field, plants exposed to ambient UV-B radiation often suffer less herbivory by folivorous insects than plants cultivated under filters that specifically exclude the UV-B component of solar radiation. This effect of solar UV-B radiation on insect herbivory was reported to be very large in a number of earlier studies.[20, 34, 35] In some cases the negative influence of UV-B radiation on insect herbivory can be partially explained by direct effects of solar UV-B radiation on insect behavior.[20, 21] In most cases, however, the anti-herbivore effect of solar UV-B radiation appears to be indirect, i.e., mediated by UV-B-radiation-induced increases in plant defenses. Much has been learned concerning mechanisms of this indirect influence on susceptibility to herbivory, as discussed later.

Below ground

Sunlight scarcely penetrates the soil surface, yet manipulation of UV-B radiation has been reported to have several consequences belowground in ecosystems (Figure 3- 2). In the soil, specific microbial communities live in close association with roots. Relatively few field studies have probed soil microbial changes, but recently, reports indicate several potentially important changes. With supplemental UV-B radiation above ground[36] mycorrhizae, fungi that are associated with roots and important for plant mineral nutrition, were substantially decreased in quantity (by ca 20%). Zaller *et al.*[14] reported a reduction in mycorrhizae in a sedge fen with solar UV-B radiation attenuation, even though root production had increased.

Apart from mycorrhizae, other soil fungi, bacteria, and microfauna inhabit soils and participate in important ecosystem functions such as nutrient cycling (Chapter 5). UV-B radiation treatments above ground can be manifested in several changes in populations of these belowground organisms and also in some apparent changes in the soil environment. For example, in Greenland, although attenuating solar UV-B radiation above ground did not affect total microbial biomass, it did result in qualitative changes (indicated by lipid biomarkers that reflect composition of microbial groups) in the soil microbial communities.[13] In southern Finland, UV-B radiation supplementation led to changes in exudations from roots of two heath species growing in peatland microcosms. The two heath species reacted quite differently to the UV-B radiation supplementation treatment. The differences in root exudations were also reflected in the soil microbial biomass.[15] Similar findings of altered root exudations were reported from UV-B radiation supplementation experiments in a grassland in the United Kingdom, but only if the soil had been disturbed.[37] In Antarctica, solar UV-B radiation attenuation led to an altered soil microbial community and this was thought to be due to altered quantity and/or quality of root exudates.[38] Root exudations are quite important, since these provide significant amounts of the energy and carbon for soil microbial communities.[39] Changes in the quantity and quality of root exudations, if reflected in changes in the soil microbial function, can have important implications for soil nutrients and carbon (Chapter 5). In northern Sweden, UV-B radiation supplementation over a 5-year period with and without elevated CO_2 treatments were used to test soil microbial responses in field experiments.[40] The aboveground sub-Arctic heath vegetation was not greatly affected by these treatments. However, there were sizeable changes in bacterial community structure due to the supplemental UV-B radiation, such as alterations in the nature of carbon use profiles, decreased microbial carbon and increased microbial nitrogen. Other studies also suggest that UV-B-radiation-induced changes in plant chemistry have the potential to affect the interactions between plants and N-fixing symbiotic bacteria, as will be discussed later.

Convey *et al.*[41] reported that near ambient solar UV-B radiation decreased the prevalence of a prominent soil-dwelling microarthropod species in Antarctic soils where these field studies were performed. This was more pronounced in the springtails than the mites. Since these soil insects are not directly exposed to solar radiation, the effects were assumed to have been mediated by UV-B-induced changes in the vegetation.

As with soil, sunlight penetrates no more than a few millimeters into the surface of peat[42], yet many changes in the subsurface milieu of peatbogs have been found, including changes in the predominance of various microfauna (testate amoeba, rotifers, mites, and nematodes) and in the abundance and species diversity of microfungi (Figure 3- 2). Testate amoebae consistently increased with near-ambient UV-B radiation compared to the attenuated UV-B radiation treatment, although fungal species diversity decreased only to a small degree.[42-44] These changes were linked to increases in dissolved organic carbon and phosphates in the peat subsurface environment[44] (Figure 3-2). Although the significance of these changes in root mass, apparent root exudations, microbiological and abiotic chemical changes is not known, there are important potential implications for carbon sequestration and nutrient cycling in ecosystems (Chapter 5).

Interactions between UV-B radiation and other climate change factors

Several environmental factors have been shown to modify UV-B-induced responses of plants, either ameliorating or enhancing the UV-B radiation effect. Furthermore, UV-B radiation can alter the way in which plants respond to other factors such as temperature extremes.

On a global scale, CO_2 is clearly increasing rapidly and uniformly throughout the world. Other environmental variables such as temperature and UV-B radiation are increasing to various degrees at different latitudes, while water supply is changing in a less predictable manner. Additionally, biologically available nitrogen is increasing substantially in many regions. Tropospheric ozone is also elevated in many regions, and some of this increase is the result of enhanced UV-B radiation resulting from stratospheric ozone reduction (Chapter 6). A few studies of the interaction of elevated ozone and enhanced UV-B radiation have been conducted as discussed in the 2003 report,[22] but newer studies are not available.

Experiments designed to explore interactions of these factors with UV-B radiation are useful to determine potential effects in the future. However, those experiments manipulating two or, sometimes more factors simultaneously are necessarily more logistically difficult to conduct. Thus, most have been carried out under conditions that can depart from those in nature and therefore constrain how much they might represent plant responses in nature. Furthermore, most of these experiments have been conducted with isolated plants or plots of plants rather than in intact ecosystems, and most only analyzed direct UV-B radiation effects on plants aboveground.

Elevated CO_2, temperature and UV radiation

One of the most interesting questions to be answered by interaction studies is whether increases in CO_2 and temperature can counteract negative effects of UV-B radiation, or whether synergistic or additive negative effects might occur. In our previous report, we found these synergistic effects to be rare.[22] Several studies using sunlit controlled environment chambers to manipulate UV-B radiation, temperature and CO_2 have been reported recently. The chambers were very useful in controlling these factors, but may have compromised the realism in relation to field conditions due to the high UV levels employed and the removal of solar UV-A radiation by the chamber material. Koti *et al.*[45] reported that higher temperatures and UV-B radiation, either singly or in combination, had detrimental effects on soybean flower and pollen characteristics. Elevated CO_2 by itself had a small beneficial effect in some soybean varieties, but had no ameliorating effects on the decidedly detrimental effect of high temperature and high UV-B radiation on pollen morphology, production, or germination. The results further suggest that these combinations of environmental factors ultimately would negatively affect fruit set and soybean yield.[45]

In the same type of sunlit chambers, other factor interaction studies have been conducted with cotton.[46-49] Very high fluxes of UV-B radiation detrimentally affected leaf physiological activities such as photosynthesis and foliage development, though this did not occur with moderate UV-B radiation levels. Elevated CO_2 enhanced growth, but did not counteract the detrimental effects of the high UV-B flux.[47, 48] High temperature and UV-B radiation both reduced cotton fruit production.[49] Higher temperatures increased the fraction of leaves abscising, while UV-B radiation only had an effect on abscission in the high temperature treatment. Both higher temperatures and UV-B radiation increased fruit fall, especially in combination.

study involving the interaction of CO_2 and UV-B radiation conducted in conventional growth chambers, rather than sunlit chambers, tested potential ameliorating influence of elevated CO_2 on UV-B radiation effects for canola.[50] Several physiological and morphological characteristics were assessed. For some traits such as plant height, elevated CO_2 did counteract the tendency of UV-B radiation to decrease plant height. However, these studies were conducted in low-light conditions compared with natural sunlight so their results may be difficult to extrapolate to nature.

While in the above example higher temperatures could be considered detrimental, there are situations where higher temperatures facilitate physiological processes that are protective. Tropical legumes exposed to elevated UV-B radiation were less detrimentally affected if they were simultaneously exposed to higher temperatures. This has been attributed to the heat-stimulated synthesis of "heat shock" proteins by the plants.[51] In another study, UV-B radiation increased heat tolerance considerably in cucumber plants grown in growth chambers.[52]

In field studies, freezing tolerance in rhododendron was increased by UV-B radiation exposure[53] which corroborated earlier studies for this species.[54] Freezing tolerance of jack pine was similarly increased by UV-B radiation and this was linked to induction of secondary compounds (phenolics) in plant tissues.[55] This latter study was also conducted in growth chambers. The effect of UV-B radiation on frost hardiness in seedlings of several conifer species from southwestern Canada was investigated under greenhouse conditions.[56] Frost tolerance of four species decreased and the tolerance increased for another three species. Heat tolerance of two conifer species increased. These changes in frost and heat tolerance were only apparent if very high UV-B fluxes were employed so the applicability of these findings for conifer seedlings in nature may be limited.

Drought and UV radiation

In a controlled environment study, several clover varieties were compared under combined treatments of drought and high UV-B fluxes.[57] Drought and UV-B radiation interacted synergistically resulting in a substantial increase of UV-B radiation- absorbing compounds, including phenolics (flavonol glycosides), in drought-stressed plants. These changes were linked with somewhat improved water status of the plants. The authors suggested that clover varieties that are slow growing and adapted to other stresses such as drought are more likely to be UV-B radiation tolerant. In contrast to the interactions reported by this study with clover, Turtola *et al.*[58] conducted a greenhouse study with willows and found few significant interactions between water stress and UV-B radiation for various stress indicators. However, the visible light levels were exceedingly low in the greenhouse and may have compromised the realism of these results. Yang *et al.*[59] conducted a greenhouse study using UV-B radiation supplementation and water stress on two populations of an important shrub from the Tibetan Plateau of China used in land restoration efforts. The low elevation population of this shrub was more sensitive to UV-B radiation than to drought and the high elevation populations more sensitive to drought than UV-B radiation supplementation. There were also significant interactions between these two stress factors. For example, in the high elevation population, enhanced levels of UV-B radiation tended to somewhat alleviate the effects of drought on decreased plant dry mass, and increased the levels of a plant hormone (abscisic acid) that is known to mediate plant responses to water stress.

In a field study[60] in which solar UV radiation was manipulated by filters, productivity was decreased and forage quality (including nitrogen content and digestibility) increased with near-ambient UV radiation for the dominant grass species in a dry, but not in a wet year. Since foliage with higher forage quality decomposes more readily, solar UV radiation and drought could reduce both grass biomass and soil organic matter for this dominant species. In another grass species, productivity decreased due to near ambient UV radiation, but only in wet years.

Forage quality of this species did not change. The variable responses observed among species grown under changing conditions of soil moisture availability and UV radiation suggest that with current solar UV radiation, as drought becomes more prevalent with global change in the shortgrass-steppe ecosystem, the structure and function of this ecosystem may change substantially.[60] This is one of the few studies of climate change factors involving UV radiation that has been conducted in the field in an intact ecosystem. However, the UV radiation manipulations included a sizeable amount of UV-A as well as UV-B radiation removal, so the relevance for ozone depletion is limited. Current levels of solar UV radiation is playing an important role in this system, but it is not clear if an increase in the UV-B component due to ozone depletion would enhance the UV radiation effects on grass growth and foliage quality.

Nitrogen and UV-B radiation

Increasing nitrogen supply to plants may result in additional sensitivity to UV-B radiation. The interaction of nitrogen and UV-B radiation is relevant to areas of nitrogen deposition from pollution and in agricultural areas where nitrogen is intentionally applied.[2] In field experiments, supplemental UV-B radiation simulating ozone depletion caused reduced total biomass, nitrogen content and specific pigment concentrations for a South African legume species (*Cyclopia maculata*), but the UV-B radiation effect was much more pronounced when the plants were supplied with supplemental nitrate.[61] In another field study with enhanced UV-B radiation in Portugal, net photosynthesis of maize was less affected by UV-B radiation at low nitrogen supply than at high nitrogen levels. Furthermore, with elevated UV-B radiation, the responsiveness to plant nitrogen nutrition was decreased.[62] However, shoot length of a freshwater higher plant species was reduced by high fluxes of UV-B radiation and this radiation effect was exacerbated by low nitrogen levels in the plant.[63] A study with potted tree seedlings of two birch species conducted outdoors[64] showed effects of different levels of UV-B radiation and nitrogen. There was no apparent growth response to supplemented UV-B radiation, but a positive growth response to greater nitrogen supply. Supplemental UV-B radiation did affect the phenolic constituents of the birch species, but not in a uniform manner for the two species tested. The different levels of nitrogen supply yielded a diversity of responses in the two birch species for both phenolics and tannins. However, there were no interactions between UV-B radiation and nitrogen levels in these responses.

Symbiotic nitrogen fixation by micro-organisms in specialized nodules on plant roots is an important source of nitrogen for plants and also for neighboring plants that do not have the capacity to form nodules. Nodulation of bean plant roots was found to be stimulated substantially by UV-B radiation in outdoor experiments.[65] This positive effect of UV-B radiation on nodulation was accompanied by a large increase in UV-B-absorbing compounds in the root system. In contrast to the study of Pinto et al., elevated levels of UV-B radiation decreased nodulation in some species of grain legumes in a study by Rajendiran et al..[66] Additional studies by Chimphango *et al*.[61, 67] did not find an effect of elevated UV-B radiation on nodulation; however, they speculated that very high UV-B flux might inhibit nodulation. In summary, the available information suggests that increases in nitrogen supply can increase UV-B radiation sensitivity, at least in those species where higher nitrogen levels result in a reduced synthesis of protective UV-absorbing compounds.[61] On the other hand, moderate increases in UV-B radiation are likely to have only modest consequences on nitrogen fixation by symbiotic bacteria.

Where interactions between UV-B radiation and nitrogen supply occur, these can also modulate plant responses to other organisms. In a recent outdoor study using potted grapevines, application of high levels of nitrogen together with below-ambient UV radiation modified the susceptibility of some grapevines to powdery mildew disease. This was evident in a high incidence of infection when UV-B radiation was attenuated that was related, among other plant physiological and anatomical features, to low concentrations of specific phenolic compounds (flavonol glycosides and hydroxycinnamic acid derivatives) and less leaf wax.[68]

Mechanisms of plant responses to solar UV-B radiation

Since our last report,[22] there has been a substantial increase in understanding the mechanisms that mediate the responses of terrestrial plants to solar UV-B radiation. This progress is important because it improves our ability to understand the effects of UV-B radiation on organisms and their ecological interactions and to devise strategies for improving plant tolerance to UV-B radiation in species of economic interest. In addition, it also serves to highlight the importance of climate change interactions.

Three generalizations about ecophysiological impacts of UV-B radiation have emerged from work carried out under field conditions mostly using natural or moderately-enhanced levels of UV-B radiation.[3, 22] Firstly, solar UV-B radiation frequently has an inhibitory effect on plant growth, although this effect is generally small (<20%) and is more pronounced among herbaceous species than in woody perennials. Secondly, solar UV-B radiation elicits a variety of acclimation responses, which typically include increased activity of antioxidant enzymes, increased DNA repair capacity, and accumulation of phenolic compounds that serve as "sunscreens" or UV filters. Thirdly, plant exposure to solar UV-B radiation frequently has large effects on the interactions between plants and consumer organisms. In this section we will briefly describe some of the most recent advances in our understanding of the mechanisms underlying some of these plant responses.

Growth inhibition

Growth inhibition at the whole-plant level often correlates with reduced leaf expansion, which appears to be more sensitive to UV-B radiation than photosynthesis per unit leaf area.[34, 69, 70] CO_2 assimilation (net photosynthesis) per unit leaf area is largely unaffected by solar UV-B radiation,[7, 34, 70] and detailed studies have demonstrated that the integrity of part of the photosynthetic system (photosystem II) is not affected by ambient or moderately enhanced levels of UV-B radiation under field conditions.[10, 71] In a model system, using the primary leaf of wheat to study growth reduction, Hopkins *et al.*[72] found that leaves responded to UV-B radiation with changes in the rate and extent of cell division and elongation. This resulted in a decreased and retarded elongation leading to the reduced growth observed.

Reactive oxygen species. The inhibition of leaf growth by solar UV-B radiation may result from accumulation of UV-B-induced damage to key macromolecules and cellular structures. In living tissues, molecular oxygen may react to form reactive oxygen species (ROS), which include superoxide radicals, hydroxyl radicals, hydrogen peroxide, singlet oxygen and reactive nitrogen. All these molecules are potentially highly destructive, since they participate in many chemical reactions with lipids, proteins and nucleic acids resulting, for example, in damage to cell membrances due to lipid peroxidation.[73-75] However, recent evidence has indicated that in low concentrations they also function as signaling molecules (c.f. [75, 76, 77]) implicated in modulating normal plant development, including senescence (ageing) and many other physiological processes.[78] To date, much research has focused on these oxidant signals as well as their interrelationship with several key hormones and stress-induced proteins. Disease and climate change factors such as increased levels of UV-B radiation, temperature extremes, drought, and ozone can stimulate elevated amounts of ROS.[79-83]

UV-B radiation enhances the expression of several genes involved in natural senescence phenomena[84] where ROS are implicated.[85, 86] Apart from their role in accelerated ageing and other developmental processes, ROS, including nitric oxide,[87] also function in triggering defense mechanisms against a range of stress factors including UV-B radiation, temperature, drought, herbivore attack and disease.[88-91] The increased ROS levels are thought to activate intricate signaling networks that eventually result in internal regulation and plant acclimation to the altered environmental conditions. In addition to ROS, the defense-related growth regulators salicylic acid, jasmonic acid and ethylene have been implicated in the mediation of plant stress to changes in UV-B radiation.[92, 93]

DNA damage. It is also possible that solar UV-B radiation generates DNA damage at a rate that overburdens DNA repair mechanisms, leading to transient accumulation of toxic DNA photoproducts. Two basic photoproducts result from the absorption of UV-B photons by DNA: the cyclobutane pyrimidine dimer (CPD), which is thought to be quantitatively the most important (about 75% of the lesions), and the pyrimidine (6–4)-pyrimidone photoproduct (6–4 PP). Both of these lesions can have toxic and mutagenic effects and can impair DNA transcription and replication.[94] Mutants that are deficient in DNA repair mechanisms are more sensitive to UV-B radiation than wild-type plants.[95-97]

The relative importance of DNA photodamage in UV-B-induced growth inhibition has been tested recently in developing plants. A native herb from southern Patagonia, *Gunnera magellanica,* was exposed to a gradient of UV-B radiation from zero to moderate UV-B fluxes in a greenhouse study. Leaf expansion was measured as an indicator of plant growth and other techniques were used to detect damage to DNA (in the form of CPDs) and lipid peroxidation. Leaf expansion decreased and the CPD density increased with increasing UV-B radiation, although the degree of lipid peroxidation remained unaffected. The highest UV-B flux induced only a transient oxidative stress. These results suggested that at UV-B fluxes within the range that *G. magellanica* plants experience in their natural environment, DNA damage may be a factor in growth inhibition.[98] Field work in southern Patagonia has shown that the rates of CPD repair in *G. magellanica* plants were modest in comparison with other species and, under equivalent conditions, were about 50% lower than the repair rates of the model plant *Arabidopsis thaliana*.[99] This low DNA repair capacity may be one of the reasons why the midday CPD load in naturally occurring plants of *G. magellanica* was linearly correlated with UV-B fluxes in the hours before midday.[100]

Using relatively high UV-B radiation, studies in rice add further support to the idea that DNA damage in the form of CPDs may be one of the main determinants of UV-B-induced growth inhibition in plants grown under physiologically meaningful conditions.[101] This work demonstrated that there is natural variation among rice cultivars in DNA repair capacity, and that slight variations in photolyase (the enzyme involved in CPD repair) activity have consequences for the ability of rice plants to tolerate the growth-inhibitory effects of UV-B radiation.

Growth-inhibitory effects of ambient UV-B radiation have been detected in herbaceous species of the Tierra del Fuego National Park,[3] and also in the two species of vascular plants that occur in the Antarctic peninsula.[9] Low DNA repair capacity may be a general feature of these species of this high-latitude region, which have evolved under low levels of ambient UV-B radiation. Repair mechanisms may also be affected by other prevailing conditions when plants are exposed to UV-B radiation, for example by water availability and temperature. This was demonstrated in a lichen species (*Cladonia arbuscula)*, where exposure to UV-B radiation in a dried state resulted in deficient repair of DNA damage, evidenced by accumulation of CPDs. At low temperatures (2 °C) DNA damage was also not repaired even though the lichens were in a hydrated state.[102]

Acclimation response

Under field conditions, plants generally adapt to changes in UV-B radiation by activating an array of protective responses that include morphological changes,[34, 103] increased DNA repair capacity,[97] induction of protective compounds (sunscreens)[7, 104-107] and increased levels of antioxidants.[108]

Of all the acclimation responses, the best characterized is the accumulation of UV-absorbing sunscreens. These sunscreens include phenolic compounds derived from phenylalanine (flavonoids and other phenylpropanoid derivatives, such as sinapate esters) that accumulate in large quantities in the vacuoles of epidermal cells and effectively attenuate the UV component of sunlight with minimal change in the visible region of the spectrum. Various studies have suggested that certain flavonoid compounds serve not only a UV-screening/filtering function, but also an antioxidant function.[109-111] This has been further supported by recent work.[57, 112] Furthermore, those flavonoids with potential antioxidant properties have also been reported to increase differentially in response to drought stress.[113]

UV-B perception and signaling in UV-B radiation acclimation. A considerable body of work has continued to reveal details of how these protective responses are activated when plants are exposed to UV-B radiation (Figure 3- 3). However, our understanding of the mechanisms of UV-B response in higher plants is still fragmentary.

Potential pathways of UV-B perception and signaling have been partially characterized, largely under laboratory conditions.[85, 93, 114-120] UV-B-induced patterns of gene expression have been characterized under controlled environment conditions,[121, 122] and also in field and greenhouse experiments.[123-126] Changes in gene expression have been observed in tissues not directly exposed to UV-B, implying transmission of a signal from irradiated to non-irradiated tissues.[125]

One of the major gaps in our understanding of UV-B perception is that the nature of the primary UV-B photoreceptors is still not well defined. Data derived from the large number of research reports cited in this chapter, as well as work done in animal models, would indicate that DNA damage may act directly as a major trigger of molecular UV-B radiation response (Figure 3- 3). However, it has been known for some time that the action spectra for the induction of several UV-B responses do not match the action spectrum for DNA damage.[127] In addition, several responses to UV-B radiation cannot be mitigated by treatments that ameliorate DNA damage[117] and they are not exacerbated in mutant plants deficient in DNA damage repair.[128] In a study where plants were pre-acclimated to UV-B radiation during growth,[129] it was reported that the expression of a gene involved in UV screening pigment induction (chalcone synthase) did not occur, although high levels of DNA damage were observed. However, without the acclimation treatment, the chalcone synthase gene was strongly activated with only low levels of DNA damage occurring. Therefore, in these cases, additional mechanisms of UV-B radiation perception and signaling have to be considered.

There are several lines of evidence suggesting that plants have specific UV-B photoreceptors, analogous to the photoreceptors involved in the perception of visible light and far-red radiation (phytochromes, cryptochromes, and phototropins) (Figure 3- 3, for a review, see Ulm and Nagy[130]). However, attempts to identify these receptors using photophysiological techniques and, more recently, genetic approaches with the model plant *Arabidopsis thaliana*, have met with little success.

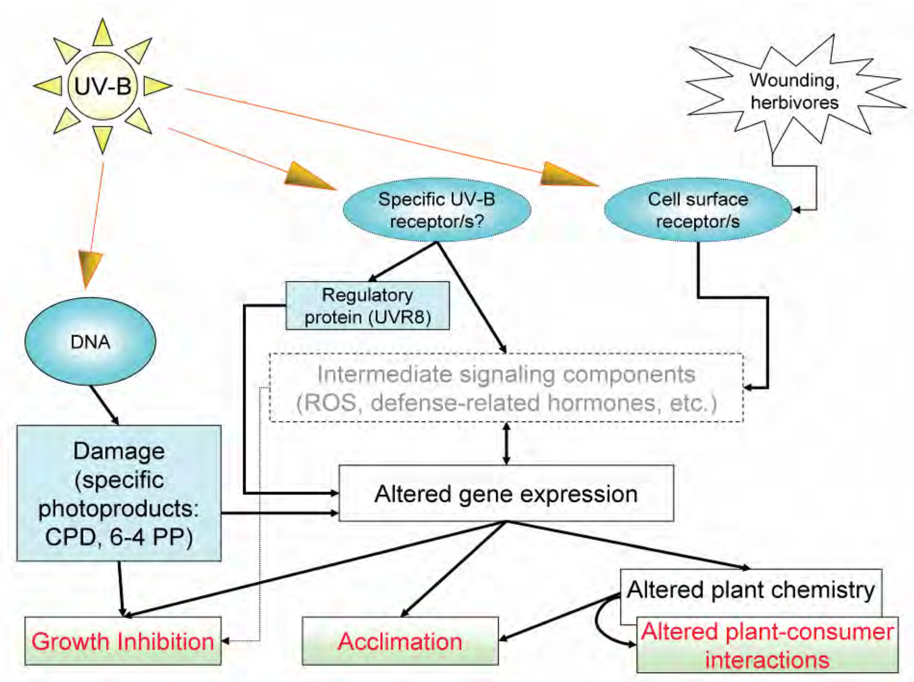

Figure 3-3. Mechanisms of plant response to solar UV-B radiation. Three general effects of solar UV-B on plants have been documented in field experiments: Growth inhibition (e.g., reduced leaf area expansion), acclimation (e.g. increased accumulation of UV-absorbing sunscreens), and alteration of plant interactions with organisms at other trophic levels (e.g., increased plant resistance to leaf-eating insects). Some of these responses can be directly linked to effects of solar UV-B on key cellular components (e.g., DNA lesions caused by UV-B photons may have inhibitory effects on plant growth). Other responses are triggered by the activation of intricate signaling networks that involve a number of signaling elements, in some instances including a UV-B-specific regulatory protein (UVR8), and in others, ROS and defense-related hormones, such as salicylic acid and jasmonic acid. Some of the signaling components appear to be shared by response circuits that are activated by a diverse group of environmental stressors (e.g., UV-B, drought, herbivory, etc.). Activation of overlapping signaling cascades by different stressors results in overlapping gene expression patterns and convergent physiological responses. Elucidation of these mechanisms will facilitate the understanding of the interactive effects of solar UV-B radiation and other environmental factors on plant growth and ecological relationships.

A recent study[119] led to the characterization of the first UV-B-specific signaling pathway, which is regulated by the protein UVR8 and controls the expression of numerous genes involved in UV-B protection and acclimation. The list of genes regulated by UVR8 includes most of the genes involved in the biosynthesis of flavonoids (protective pigments), the gene encoding CPD photolyase, PHR1 (which is essential for repair of UV-B-induced DNA damage), and genes concerned with protection against oxidative stress and photooxidative damage. A combination of molecular and genetic approaches has established that the transcription factor, HY5, is a key downstream effector of the UVR8 pathway. UVR8 regulates transcript levels of HY5 specifically in response to UV-B, and HY5 in turn regulates the expression of a substantial number of genes involved in UV protection. Previous work[122] has demonstrated that the effects of low levels of UV-B on the expression of a subset of genes required HY5, but was independent of previously described photoreceptors. At present there is growing consensus that UVR8 and HY5 orchestrate specific acclimation responses to UV-B radiation; however, the photoreceptor and early signaling components that are activated by UV-B radiation and eventually engage the UVR8 pathway remain to be elucidated.

For some effects of UV-B radiation on plants, a significant overlap has been documented between cellular responses activated by UV-B radiation and other environmental stress factors, such as tropospheric ozone, pathogen, and herbivore attack. This overlap has been evidenced by the occurrence of convergent patterns of gene expression (e.g.(Izaguirre, 2003 #68;Sävenstrand, 2002 #139}) activating of common signaling components (e.g., Holley *et al.*[131], reviewed in Stratmann[115]), and induction of similar defense molecules and secondary metabolites.[132] A possible explanation for this overlap is based on the activation of common signaling intermediates, such as ROS and defense-related growth regulators, as indicated in Figure 3-3. Another possibility, at least in the case of overlaps between responses elicited by UV-B radiation and attack by herbivorous insects, could be the activation of common cell surface receptors by UV-B radiation and insect elicitors, as hypothesized by Stratmann[115](Figure 3-3). Current experiments directed to testing this hypothesis will improve our understanding of the mechanisms of UV-B radiation effects on plants, and provide novel insights into the problem of increasing UV-radiation tolerance and resistance to insect pests in cultivated species. The existence of overlapping signaling components leading to a response may have important functional implications, because environmental stresses are seldom experienced by the plant in isolation.

Plant interactions with consumer organisms

UV-B radiation and plant-animal interactions. Plant exposure to solar UV-B radiation frequently results in a reduction in the level of insect herbivory (Figure 3-4 and reviewed in[22, 133, 134]). Recent studies show that this anti-herbivore effect induced by UV-B radiation correlates with a significant overlap between gene expression patterns induced by UV-B radiation and wounding under controlled environmental conditions.[121, 123] Components of signaling cascades that are activated by wounding and herbivory are also activated by UV-B radiation.[131] Furthermore, a number of plant products typically associated with anti-herbivore defenses, such as defense-related proteins (e.g., proteinase inhibitors), and phenolic compounds (e.g., chlorogenic acid), accumulate in larger quantities in plants exposed to UV-B radiation than in plants receiving no UV-B radiation.[118, 135] The increases in defense-related compounds in plants exposed to UV-B radiation correlate with a lower forage quality in tissues from these plants, as evaluated using insect growth bioassays (e.g.,[123, 136]). Behavioral bioassays have also shown that exposure to solar UV-B radiation can induce changes in the plants that reduce the attractiveness of the plants to female adults at the time of oviposition.[137] This effect of solar UV-B radiation may have important effects on insect herbivory under field conditions by controlling the distribution of plant-feeding larvae within the canopy.

UV-B radiation and pathogens. Severity of pathogen attack plays a determining role in plant productivity in the field and greenhouses, and studies have shown that many environmental factors modify both pathogen and plant response. Apart from precipitation patterns, temperature, and time of day, UV radiation can also be a modifying factor. Recent work from a field study indicated that species diversity was affected by solar UV-B radiation because of species-specific sensitivity to the radiation.[31] Besides the potential damaging effects of UV radiation, it also may play a role as a light signal for many processes of plant pathogen development (see [134]). UV-radiation effects may influence pathogen development either directly or indirectly, the latter as a consequence of UV-induced biochemical changes, which are reflected in interactions between pathogen and host (for reviews see [138, 139]). The initial events and signaling mechanisms underlying these interactions are complex, and will be briefly discussed below.

There have been several reports showing that UV radiation may either decrease or increase fungal attack (see [138]). These attacks depend on many interacting factors such as temperature, light, time of day and humidity as well as on plant morphology and genetic background.[31, 68, 134, 138]

UV-B radiation and pathogen infection trigger both specifically targeted defense responses as well as initiating crosstalk between signaling pathways.[140, 141] For example, the pathogen defense may result in repression of the UV protective flavonoid biosynthetic pathway.[140] In the latter study it was shown that specific genes were activated by UV-B radiation and subsequently repressed by the pathogen elicitor, demonstrating interaction between two specific signaling cascades, whereby the pathogen signal was perceived as the more important in plant defense and survival.[140] Thus, overlap between abiotic and biotic signaling suggests mutual interactions and response to different external pressures.

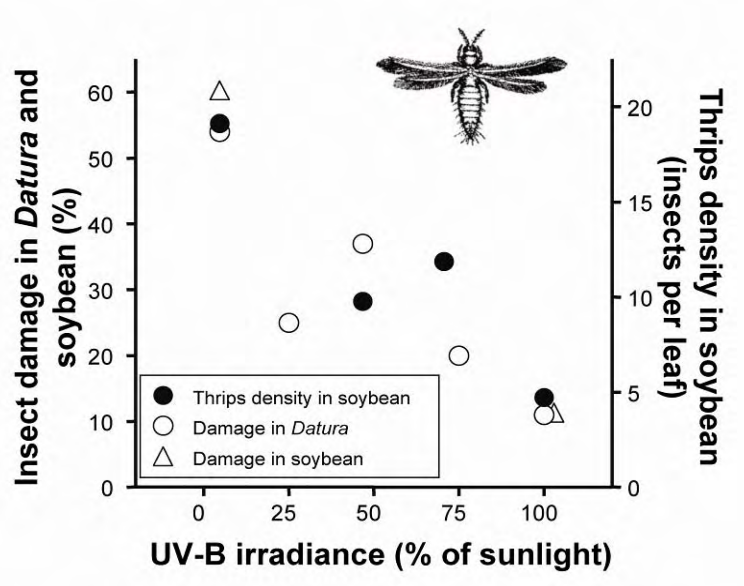

Figure 3-4. An example of effects of solar UV-B radiation on insect herbivory in the field. The graph shows the effect of attenuating the UV-B component of solar radiation on the intensity of insect herbivory by thrips (a piercing-sucking insect) in soybean crops, and leaf beetles (chewing insects) in plants of the common annual weed *Datura ferox* (original data in[21, 36]). The marked effects of solar UV-B radiation on leaf damage and insect population density are proportionally much larger than any reported effects of solar UV-B radiation on plant growth and morphology.

Induction of the so-called pathogenesis-related proteins (PR proteins) is another example of the complexity of plant response to UV-B radiation and other environmental factors. These proteins are found in many plants, not only in response to UV-B radiation[117, 142, 143], but also after fungal, bacterial, or viral attack as well as after exposure to certain organic chemicals or heavy metals.[144] The signal transduction pathway for the synthesis of these PR proteins induced by UV-B radiation is not yet fully elucidated and there appears to be crosstalk among different communication pathways when the plant is exposed to multiple stresses such as pathogens and UV-B radiation. A recent example of a PR protein induced by UV-B radiation is the enzyme, β-1,3-glucanase I (ßGlu I)[117], a protein widely found in seed plants, involved also in developmental plant processes.[145] A laboratory study conducted with beans (*Phaseolus vulgaris*) indicated that the induction of the PR protein, βGlu I, was correlated with photoreversible DNA damage, which has not been reported for other PR protein families. In addition, the results implicate the DNA damage (seen as CPD formation) as a key player in the signaling pathway resulting in the induction of βGlu I by UV-B radiation (Figure 3- 3). The plant response to UV-B radiation and subsequent induction of the PR protein, βGlu I, were found to be local rather than systemic (the latter where the whole plant rather than a localized area would be affected by a stress). Studies concerned with unraveling the complex internal plant pathways of communication are important for our understanding of the consequences of interactions between UV-B radiation and other stress factors, especially those related to climate change and modern agricultural practices.

Technical issues in conducting and evaluating UV-B radiation research

Biologically effective UV radiation

Biological spectral weighting functions (BSWFs) are factors assigned to different wavelengths indicating their relative biological effectiveness and these functions have been developed for a number of different plant processes. BSWFs have played a prominent role in the ozone reduction issue. Their uses have included predicting the relative increment of “biologically effective” UV radiation for a given increment in ozone depletion, evaluating latitudinal and elevational gradients of solar UV radiation, and calculating how much supplemental UV radiation to supply in experiments with UV-B-emitting lamps.

A single BSWF cannot be expected to apply to multiple plant responses or to be necessarily appropriate for all plant species, much less to generalize to other trophic level effects. In our report four years ago[22] we introduced new work showing a BSWF for plant growth / mass allocation effects which extended into the UV-A wavelengths with substantial effectiveness. This weighting function was developed in the laboratory with seedlings of cultivated oat[146] and was subsequently validated in the field for this species.[147] The importance of UV-A radiation in causing plant responses has been confirmed by other recent studies.[82, 148-150] For example, the production of singlet oxygen, one of the two major representatives of damaging ROS, is produced more effectively by UV-A than by UV-B radiation.[82] Specifically, UV-A wavelengths were twice as effective as UV-B wavelengths for inducing singlet oxygen. This is unique as BSWFs for damage to other plant processes typically show a marked decline toward longer wavelengths.

Evaluating weighting functions

There have been only a few attempts during the 1980's and 1990's to evaluate alternative BSWFs under polychromatic radiation conditions.[151] Many difficulties exist in evaluating BSWFs. In many locations, filtered-sunlight experiments produce insufficient effects to yield consistently measurable differences between different wavelength treatments. For example, even with the high UV fluxes of Arizona, USA (33 N), a dose of >70% of ambient UV-B radiation was typically needed before consistent statistically significant effects were seen in cotton and sorghum.[152] However, in most cases, it is likely that experiments combining modulated UV lamp supplementation with different treatments of UV-filtered sunlight will be necessary to continue testing BSWFs in the field. The total UV radiant flux removed in UV radiation-attenuation studies is considerably greater than the added UV radiation in lamp-supplementation studies. However, when treatments are weighted using BSWFs, the changes in biologically effective radiation in the lamp-supplementation studies may be greater or equal to that removed by the UV-B exclusion treatment. The relationship between filter and lamp treatments depends on the BSWF employed. Rousseaux *et al.*[106] compared treatments using filters and lamps for lettuce and oat in a field experiment where particular care was taken to insure similar microenvironmental conditions among all treatments. Overall, when comparing the two treatment methods, plant growth and allocation changes followed a pattern similar to a plant

DNA damage spectrum,[153] emphasizing the importance of radiation in the UV-B to UV-A transition zone. Responses of UV-absorbing compounds suggested less of an effect of these longer wavelengths, approaching instead a response predicted by the generalized plant response function.[154]

The spectral dependence for induction of UV-absorbing pigments was also examined in the cultivated oat in a lamp/filter experiment and sorghum, pepper and kochia in a UV-attenuation experiment.[155] Despite the very different experimental UV fluxes, a BSWF extending into the UV-A waveband with moderate effectiveness was most representative of the observed results for the four species. The plant DNA damage spectra[153] and a new flavonol induction BSWF with similar spectral response[148] were the most appropriate of the five BSWFs evaluated.[155]

Implications of using different BSWFs

This increasing evidence that plant BSWFs include significant UV-A radiation responses has substantial implications for the design and interpretation of experiments investigating plant response to ozone depletion. Best known among the uses of BSWFs is the calculation of radiation amplification factors (RAFs). They have been used to compare the effects of current levels of UV radiation with scenarios of ozone depletion. For small changes in ozone the RAF is the increment of effect (biological or chemical) resulting from a 1% ozone depletion increment. Thus a RAF of 1 would indicate a 1% increase in response. (Note in Chapter 1, only the erythemal spectrum is used as a weighting function. This spectrum has a RAF of 1.2 at high sun elevations. Erythema is the sunburn response of human skin.) The original generalized plant response function[154] has a RAF calculated at 1.6, while the RAF of the new growth / mass allocation BSWF is 0.2 for the same conditions.[156] This means that, if we accept the new plant growth BSWF in place of the generalized plant response, the effects anticipated under a given level of ozone reduction will be smaller than with the BSWF based on the generalized plant response. Spectra such as those of Quaite *et al.*[153] and Ibdah *et al.*[148] would be intermediate, as they have greater RAFs than the new plant growth / mass allocation BSWF but a lower RAF than the generalized plant response.

Choice of BSWF will also have a bearing on experimental simulation of ozone depletion using UV emitting lamps. This stems from several factors, but is primarily due to the substantial effect of short wavelengths from the lamps in providing biologically effective irradiance when the original generalized plant response[154] is used. The new BSWF weights these short-wavelength supplements proportionately less, and the lamps commonly used produce very little of the longer wavelengths in this BSWF. In fact, when simulating ozone depletion in the field, the racks of lamps may result in more shading of these longer wavelengths from the sun than they generate.[157] If we accept the new BSWF as appropriate, we have simulated a much lower level of ozone depletion in past experiments than was intended. These implications extend to controlled environments and greenhouses (or sunlit field chambers that do not transmit UV radiation). “Controls” in these situations, rather than receiving ambient UV radiation, often receive little UV, especially at shorter wavelengths. If the new BSWF is correct, these "controls" are even further removed from ambient UV radiation levels than had been previously assumed. In fact, even "treatment" UV levels, when weighted with this new BSWF in greenhouses or controlled environments, may be below ambient UV radiation levels! This issue is worthy of further quantitative evaluation.

The uncertainties in plant BSWFs remain significant, but may be addressed by new experimental approaches. Values of RAF are sensitive to the range of wavelengths considered in their calculation, especially the longest wavelength where the weighting function terminates, which is usually in the UV-A region.[158] It is often difficult to determine biological response to longer wavelengths, thus adding additional uncertainty. Equipment such as the free electron laser (FEL), has considerable potential to enhance our ability to resolve weighting functions in the UV-A waveband.[159]

In the field, plant characteristics that are influenced by UV-B radiation would be expected to change along latitudinal or elevation gradients in parallel with changes in biologically weighted irradiance. Theoretically, this would provide an opportunity to test different spectral weighting functions. An elevation gradient of UV-absorbing compound concentration in beech was described by Neitzke and Therburg.[160] There were higher concentrations with increasing elevation, and these concentrations correlated significantly with erythemally weighted UV radiation. However, they did not compare the relative increase in pigments with UV radiation weighted with other BSWFs. Also, for a discriminating test of BSWFs, it would be useful if the spectral distribution along the gradient changed appreciably.

These techniques provide routes to resolving plant BSWFs further. However, in the meantime, reporting of UV doses calculated from several different BSWFs is recommended.[161] This permits retrospective evaluation of experiments as more understanding of organism spectral responses develops.

UV dose-response relationships above and below ambient solar flux

A comparison of UV radiation-attenuation and supplementation approaches also begs the question of the nature of the plant response pattern to increasing UV-B radiation, i.e., the dose-response relationship. If biological responses exhibit an apparent saturation, the same increment of biologically effective UV radiation will exert less effect at higher UV fluxes, such as in supplementation experiments. Coleman and Day[152] tested the dose-response relationship in their UV radiation-attenuation experiments and did not see evidence of saturation as the UV radiation approached ambient levels. Newsham *et al.*[162] also did not find a saturation near ambient UV radiation in their Antarctic experiments. However, neither study combined the graded UV attenuation treatments with UV supplementation to test the dose-response relationship above ambient UV radiation. Thus, this issue remains to be resolved.

Concluding remarks and gaps in knowledge

Since our last report[22], there have been substantial advances in our understanding of the effects of UV-B radiation on terrestrial ecosystems, especially in the elucidation of mechanisms of plant response. A further area of highly interesting research emphasizes the importance of indirect UV radiation effects on plants, pathogens, herbivores, soil microbes and ecosystem processes below the surface. What was once considered the essence of understanding terrestrial ecosystem responses to ozone column reduction, namely reductions in plant photosynthesis and production, is now superseded by a much more complex picture of indirect UV radiation effects, trophic level interplay, and microbial participation in ecosystem function.

Enticing questions remain regarding the extent to which systemic chemical and physical changes in vegetation induced by UV-B radiation affect belowground systems. This includes roots and closely associated microoranisms and microbial communities beyond the immediate root microenvironment with ramifications for soil processes such as nutrient cycling. These changes in the belowground system are apparently mediated by the vegetation. It is recognized that well replicated field studies are highly important to realistically evaluate effects altered UV radiation. There is a particular paucity of field studies investigating the interactions of altered UV radiation combined with changes in other climate change factors, at least in part owing to the technical difficulties in conducting such studies.

Much has been learned about mechanisms of UV-B radiation action in plants and how resultant changes interact with ecologically important processes such acclimation, growth inhibition, plant-animal and plant-microbe interactions. A more detailed understanding of how UV-B radiation modifies gene expression and resultant systemic changes in plant characteristics is needed and will be useful in elucidating ecosystem-level changes. Research on the intricate and sometimes overlapping network of signaling pathways induced by UV radiation and other stimuli, such as pathogen attack, is important to understand why these sometimes overlap and sometimes counteract one another.

Seemingly arcane technical issues, such as which are the most relevant biological spectral weighting functions, count heavily in the conduct and evaluation of experiments designed to assess plant and ecosystem response to UV radiation changes. Thus, further refinement and testing of BSWF are of considerable priority.

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Chapter 4. Effects of solar UV radiation on aquatic ecosystems and interactions with climate change

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Summary

Recent results continue to show the general consensus that ozone-related increases in UV-B radiation can negatively influence many aquatic species and aquatic ecosystems (e.g., lakes, rivers, marshes, oceans). Solar UV radiation penetrates to ecological significant depths in aquatic systems and can affect both marine and freshwater systems from major biomass producers (phytoplankton) to consumers (e.g., zooplankton, fish, etc.) higher in the food web. Many factors influence the depth of penetration of radiation into natural waters including dissolved organic compounds whose concentration and chemical composition are likely to be influenced by future climate and UV radiation variability. There is also considerable evidence that aquatic species utilize many mechanisms for photoprotection against excessive radiation. Often, these protective mechanisms pose conflicting selection pressures on species making UV radiation an additional stressor on the organism. It is at the ecosystem level where assessments of anthropogenic climate change and UV-related effects are interrelated and where much recent research as has been directed. Several studies suggest that the influence of UV-B at the ecosystem level may be more pronounced on community and trophic level structure, and hence on subsequent biogeochemical cycles, than on biomass levels per se.

Introduction

Aquatic ecosystems are key components of the Earth's biosphere.[1] They produce more than 50 % of the biomass on our planet (Figure 4-1) and incorporate at least the same amount of atmospheric carbon dioxide as terrestrial ecosystems (cf. Chapter 5). The primary producers in freshwater and marine ecosystems constitute the basis of the intricate food webs, providing energy for the primary and secondary consumers and are thus important contributors for the production of the human staple diet in the form of crustaceans, fish, and mammals derived from the sea. Solar UV can negatively affect aquatic organisms.[2-4] The massive loss of stratospheric ozone over Antarctica over the past two decades as well as ozone depletion over the Arctic and high to mid latitudes have aroused concern about the effects of increased solar UV-B radiation on marine and freshwater ecosystems.[5] Clear lakes and oceans in alpine and polar regions, where UV penetrates deep into the water column, may be particularly vulnerable. The biological organisms in polar waters are even more at risk because of the limited repair capabilities under the inhibitory effects of low temperatures.[6]

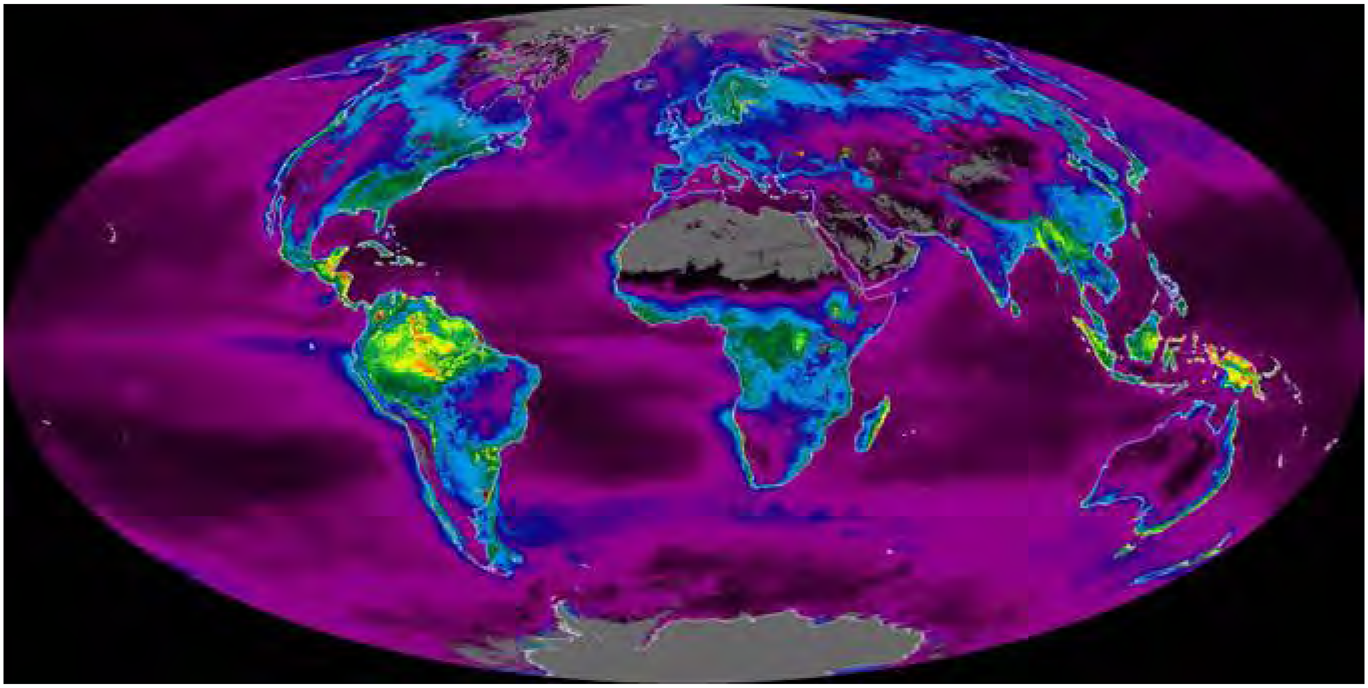

Figure 4-1. This false-color map represents the Earth's carbon "metabolism"— the rate at which plants absorbed carbon out of the atmosphere during the years 2001 and 2002. The map shows the global, annual average of the net productivity of vegetation on on land and in the ocean. The yellow and red areas show the highest rates, ranging from 2 to 3 kilograms of carbon taken in per square kilometer per year. The green areas are intermediate rates, while blue and purple shades show progressively lower productivity. In any given year, tropical rainforests are generally the most productive places on Earth. Still, the ongoing productivity near the sea's surface, over such a widespread area of the globe, makes the ocean more productive than the land. (Image courtesy of NASA, 2003).

Solar UV Radiation and Penetration in Aquatic Ecosystems

A growing number of stations and networks have shown that there has been an increase in solar UV-B radiation at the surface of and within aquatic systems[7-10] which corresponds with stratospheric ozone depletion.[11] Comparative measurements indicate continued increases in solar UV-B, which are masked by much larger seasonal changes and geographic differences (cf. Chapter 1).[12] Instrument accuracy has been improved in recent years and measurement deviations have been quantified.[13] In addition, biological and chemical actinometers have been developed to determine UV-B doses on site during experiments and exposure.[14-17]

Aquatic environments vary tremendously in their UV attenuation. Coastal areas and shallow continental shelf waters have a lower transparency than open ocean waters due to the runoff of silt and dissolved organic carbon (DOC) from shores. In open oceans the optical properties are largely determined by plankton and their degradation products,[18-20] with zooplankton being an additional source of DOC.[21] Owing to the high input of inorganic and decaying organic material, freshwater ecosystems usually have a high UV absorption which also depends on their level of eutrophication.[22]

Ozone and aerosols provide the primary filter in the atmosphere that reduces damaging UV radiation before it reaches the Earth's surface. While stratospheric ozone depletion has now stabilized and is beginning to return to pre-Montreal Protocol levels (see Chapter 1), the UV transparency of inland aquatic ecosystems remains highly variable and subject to increased UV exposure due to climate change.[23] Climate change alters the DOC concentration and hence the UV transparency of inland waters. Warmer, drier climates in particular will reduce the inundation and water saturation of soils within watersheds and hence reduce the inputs of DOC to adjacent lakes and streams.[23] In some cases a combination of acidification and climate change has led to dramatic increases in underwater UV penetration[23] (see Chapter 5). The impact of climate change may be particularly pronounced in freshwater ecosystems with low DOC concentrations due to the exponential increase in UV penetration at DOC concentrations below 2 mg L^{-1} (Figure 4-2). Such variable levels of DOC and hence UV exposure may be important factors in determining the distribution and abundance of planktonic and shallow benthic organisms as well as influence the spawning depth of vertebrates such as amphibians and fish that lay their eggs in shallow surface waters.

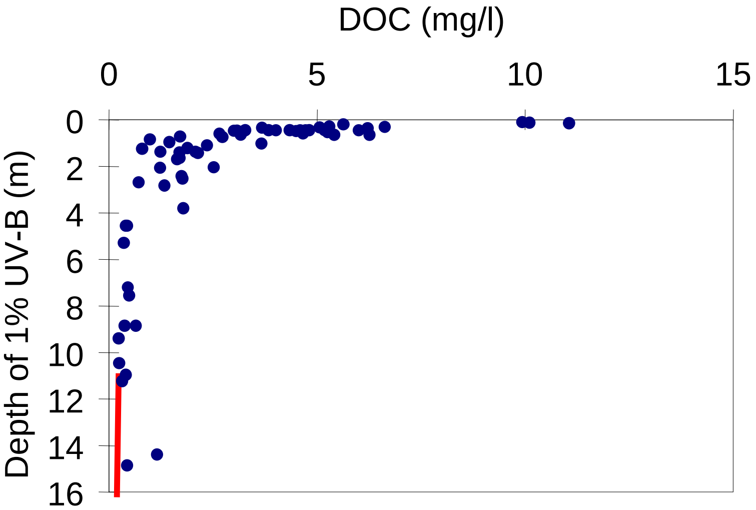

Figure 4-2. Relationship between the depth to which 1% of surface 320-nm UV radiation penetrates and concentration of dissolved organic carbon (DOC) in temperature lakes. Note that at low DOC concentrations (1 -2 mg L^{-1}) very small changes in the amount of DOC can cause large changes in the depth to which UV penetrates. Modified from.[23]

Climate warming has been found to increase eutrophication in boreal lakes.[24] In addition, the export of DOC from boreal peatlands increases with temperature. Since these areas cover about 15 % of the boreal and subarctic regions and climate warming is forecast to be most severe at high latitudes, the increasing temperatures are expected to have significant effects in boreal areas.[25] Phytoplankton abundance may vary by orders of magnitude driven by future climate-DOM-UV radiation interactions.[26] Other aquatic ecosystems also show that CDOM (colored dissolved organic material) is a mediator of climate-UV interactions.[26] Global warming has not only the potential to affect lake species compositions,[27] but also to increase the invasion potential by imported species.[28]

Besides inorganic particulate matter, dissolved and particulate organic carbon (DOC and POC) are the main attenuating substances in freshwater and coastal marine waters.[29] DOC concentrations often show large spatial and temporal variability.[30] Recent models analyzing the absorption of the components show that DOC mainly attenuates UV-B radiation while POC mainly decreases the UV-A radiation in the water column.[31] The optical effects of zooplankton and phytoplankton on UV attenuation in freshwater ecosystems are usually low,[32] but bacterioplankton plays a major role (cf. Chapter 5). While DOC is only slowly degraded in the water column, it is readily fragmented by solar UV to smaller subunits,[33] which are consumed by bacterioplankton.[34] This increases the UV transparency of the water column[35] where the resulting deeper UV-B penetration affects bacteria and other organisms.[36] In addition, photobleaching increases UV transparency. Increasing temperatures associated with global climate change are generally expected to decrease DOM concentrations and thus increase the penetration of UV-B radiation into the water.[37]

DOC is a source of dissolved CO_2 in the water,[38, 39] and pCO_2 is closely related to the DOC concentration in Swedish boreal lakes.[40] Acidification also decreases DOC concentrations.[41, 42] Depending on its concentration, DOC can have positive or negative effects on phytoplankton growth. Low concentrations contribute to nutrient recycling (N and P)[43] and availability, while higher concentrations negatively affect phytoplankton growth by shading.[44] Bacteria are the main agents for the mineralization of N and P from DOC. In addition to biomineralization, phototransformation alters biodegradation to a variable degree, depending on the source of DOC.[45]

Arctic and Antarctic marine and freshwater ecosystems are additionally affected by snow and ice cover. Even thin layers of snow or ice significantly decrease the penetration of solar UV.[46] Earlier ice melting due to increased temperature will expose phytoplankton blooms to higher solar UV radiation. The seasonal change in sea-ice cover is a major determinant of the Antarctic aquatic ecosystem. In addition, glacial meltwater plumes play a critical role near the ice edge and their influence extends more than 100 km into the open ocean and influences the biota by water column stratification, changes in turbidity, salinity and temperature.[47] Global warming at higher latitudes may lead to shallower mixed-layer depth, more intense seasonal stratification with shallower mixed layers and subsequent influence on UV impact on aquatic ecosystems.

Plankton

Plankton can be subdivided, based on physiological or taxonomic criteria into major groups of bacterioplankton, phytoplankton (including cyanobacteria and eukaryotes) and zooplankton.[48] In aquatic ecology, size (on a logarithmic scale) is used as a subdivision criterion: femtoplankton (0.02 – 0.2 μm), picoplankton (0.2 – 2 μm), nanoplankton (2 – 20 μm), microplankton (20 – 200 μm) and macroplankton (200 – 2000 μm). Even though the smallest organisms contribute a significant share to aquatic biomass productivity, these taxa have not yet been studied extensively in terms of UV sensitivity.

Bacterioplankton and Viruses

Although the bacteria are small in size, they contribute a significant biomass component in aquatic ecosystems and play a key role in biogeochemical processes.[49] Predation is the major mortality factor for planktonic bacteria.[50] Most bacterioplankton do not produce screening pigments but overcome solar radiation stress by fast cell division and effective repair mechanisms.[51] As long as the repair keeps up with the damage, the population is not threatened; but when CPDs (cyclobutane pyrimidine dimers) accumulate under high solar radiation, the population decreases. CPDs constitute by far the most frequent DNA damage induced by UV-B, followed by single- and double-strand breaks.[52, 53]

DNA damage correlates strongly with the penetration of UV radiation into the water column, and UV-B has a stronger effect than UV-A. When bacterioplankton was exposed in UV-transparent bags in tropical coastal waters, DNA damage was detectable down to 5 m. However, inhibition of leucine and thymidine incorporation, as markers for protein and DNA synthesis, respectively, occurred to a depth of 10 m.[54] Photorepair by the enzyme photolyase, using UV-A/blue light as an energy source,[55] is a major mechanism to reduce the CPD load.[4] Alternatively CPDs can be repaired by nucleotide excision repair.[56] Because of the path length of penetration, size seems to be a decisive factor for UV sensitivity: bacterioplankton from several boreal lakes in Canada were more sensitive to solar UV than the larger phytoplankton.[57]

Phytoplankton density significantly influences the depth distribution of bacterioplankton in the water column. During the summer, dense diatom phytoplankton populations develop in the Antarctic waters off the British Rothera Station, causing strong UV attenuation in the top layers.[58] At the surface, bacterioplankton incurred large UV-B-induced DNA damage (exceeding 100 CPDs per megabase pairs, Mbp), but it was protected from solar UV-B below the diatom population. This phenomenon was particularly prominent during January and February, when sea ice melting cause pronounced stabilization of the water column. Later in the season, this effect weakened and DNA damage was homogeneously distributed throughout the top 10 m in well-mixed waters.

Solar UV has a decisive role in bacterioplankton community structure in marine surface waters.[59] Large differences in sensitivity were found between different samples from the northern Adriatic Sea. When exposed to UV-B radiation, inhibition of amino acid incorporation varied substantially and there were even larger differences in the efficiency of recovery between species. In Antarctic marine bacteria UV-B and UV-A had similar negative effects on survival.[60] In contrast, in a high mountain lake (Spain) UV-A exerted the main effect.[61] In the upwelling zones of the Humboldt Current System, PAR induced a significant inhibition of bacterial productivity followed by UV-A and UV-B.[62]

Both in the Arctic and Antarctic, spores of *Bacillus subtilis* were inactivated by solar radiation within hours. However, a covering of ca. 500 µm of soil or dust or a retreat of ~1 mm into endolithic habitats prevented inactivation of the spores.[63] Snow covers of 5 – 15 cm thickness attenuated UV penetration by a factor of 10 and protected the spores from inactivation. Crust formation and biofilms are additional protective measures against environmental factors including desiccation, temperature changes and solar UV.[64] Halobacteria, being Archaea, show a much higher resistance to solar UV radiation than bacteria and even tolerate UV-C radiation,[65] reflecting the tolerance of shorter wavelengths penetrating through the atmosphere during early evolution of these organisms. At present UV-C does not reach the Earth surface - except high mountain locations - due to complete absorption in the atmosphere.

Another decisive factor for bacterial communities is the concentration of viruses.[66] Virus-to-bacteria ratios were found to be lowest in freshwater lakes and highest in saline lakes. The viral abundance was closely correlated with the concentration of DOC. Viruses have neither effective sunscreens nor photorepair capabilities[67] and are prone to solar UV damage.[68] This is supported by their seasonal abundance in central European lakes.[69] However, while being sensitive to solar UV, it is surprising that the presence of viruses can provide some protection from solar UV to their phytoplankton hosts such as *Phaeocystis*; the reason for this unexpected phenomenon is not known.[70] Anthropogenic pollutants such as cosmetic sun screens increase the abundance of viral particles in the water.[71]

Picoplankton

Unicellular picophytoplankton such as *Synechococcus* and *Prochlorococcus* are recognized as ubiquitous organisms of oceanic microbial loops and as the most abundant marine primary producers.[72] The effects of ambient levels of solar radiation on oceanic picoplankton were studied in the water column[73] using the range from unattenuated radiation to 23 % of the surface level. The radiation significantly increased cell death in *Prochlorococcus*, while the cyanobacterium *Synechococcus* had ten times the survival rate. Removal of UV radiation strongly reduced the cell death rate in the first species and eliminated it completely in *Synechococcus*. Natural solar radiation decreased the half-life times of the cells to a little over a day. A similar differential sensitivity of the two groups was found for Mediterranean ecotypes.[74] This generally high sensitivity of picoplankton to ambient solar radiation may act as a primary driver of species composition and population structure and govern the dynamics of the microbial food web in clear oceanic waters.[73]

Natural levels of solar UV-B have been determined in the Red Sea using a DNA biodosimeter.[75] In parallel, depth profiles of DNA damage were analyzed in plankton samples that had been collected from the water column down to 50 m. While the dosimeter did not show any response below 15 m, CPD DNA damage could be found in all plankton samples. CPD concentrations increased during the day and decreased over night, indicating DNA repair, but the dark repair processes did not remove all CPDs during the night. Exposure to UV-B increases the membrane permeability as shown in *Nannochloropsis*, which decreases the nitrogen uptake capability.[76]

Cyanobacteria

During the early Precambrian era, fluxes of solar UV-B and UV-C at the surface of the Earth were several-fold higher than today due to the lack of oxygen in the atmosphere and the consequent absence of ozone in the stratosphere (cf. Chapter 1). Early evolution was therefore limited to UV-protected aquatic habitats. Nonetheless, there was a strong selection for protective and mitigating strategies of early organisms against solar UV radiation.[77, 78] The early UV screens in aqueous environments may have been simple aromatic organic molecules, which later developed into specialized UV absorbers still found in cyanobacteria as well as in some eukaryotic photosynthetic organisms.[77]

Cyanobacteria are major biomass producers both in aquatic and terrestrial ecosystems and represent more than 50 % of the biomass in many aquatic ecosystems.[77] Because of their nitrogen-fixing capacity they serve as important fertilizers both in the sea and in terrestrial plant habitats such as tropical rice fields. Some cyanobacteria produce highly toxic substances, including neurotoxins and peptide hepatotoxins, which cause animal poisoning in many parts of the world[79] and pose considerable risks for human health by polluting drinking water reservoirs and recreational areas.[80] In the Baltic Sea the filamentous *Nodularia* forms extended blooms in late summer during calm weather.[81] These organisms are tolerant of ambient solar UV-B levels and outcompete more sensitive organisms even though solar UV-B has increased by 6 – 14 % over the last 20 years in this area.[82]

Recent studies show that UV-B radiation treatment results in a wide range of responses at the cellular level, including motility, protein biosynthesis, photosynthesis, nitrogen fixation and survival in cyanobacteria.[83, 84] The molecular targets include DNA and the photosynthetic apparatus.[85, 86] The phycobiliproteins, which serve as solar energy harvesting antennae, are specifically bleached by UV radiation.[87, 88] However, several studies have demonstrated an adaptation to UV stress and an increased resistance.[89, 90] Long-term exclusion of solar UV decreased the photosynthetic competence.[91] Adaptive mutagenesis, which has been found in cyanobacteria, increases their resistance to UV-B.[92] Additional stress by exposure to heavy metal ion pollutants adds to the UV-B effect.[93, 94]

Recent studies show that UV-B radiation treatment results in a wide range of responses at the level of the cell. On the molecular level UV exposure causes a wide range of responses. It induces an increased Ca^{2+} influx via L-type calcium channels.[95] The stress signal is subsequently amplified and transmitted using cyclic nucleotides as secondary messengers[96] followed by the production of shock proteins. UV-B treatment increased the concentration of 493 proteins out of 1350 at least threefold in the terrestrial species, *Nostoc commune*.[97] In addition to direct UV-B-induced damage to the DNA, oxidative stress (singlet oxygen and superoxide radicals) and damage were reported, causing lipid peroxidation and DNA strand breakage.[98] After prolonged UV-B exposures an adaptation to the reactive oxygen species (ROS) stress has been observed.[98] Typical ROS quenchers such as ascorbic acid, N-acetyl-L-cysteine or sodium pyruvate have protective effects.[99, 100]

Protective and mitigating strategies of cyanobacteria include mat or crust formation,[101] vertical migration of individuals within the mat, or shelf shading due to changes in morphology as observed in *Arthrospira platensis*.[102] In microbial mats the surface layer often serves as a protector for the organisms underneath. A mat in a high Arctic lake showed high concentrations of photosynthetic pigments in the lower part of the mat, while the black top layer was rich in scytonemins and MAAs.[103] By producing UV-absorbing substances including MAAs and/or scytonemins, many cyanobacteria are able to withstand excessive solar UV radiation.[104-106] MAAs are water-soluble compounds and have absorption maxima in the range from 310 to 360 nm.[77] Upon absorption of UV radiation MAAs form triplet states which thermally relax and thus render the radiation energy harmless.[107] MAAs are either constitutive elements within the cells or are induced by solar radiation.[108] In many cases action spectroscopy has shown that solar UV-B (which peaks around 300 nm) induces MAA synthesis in algae and phytoplankton, while visible radiation has no effect.[109] Biosynthesis of scytonemin is induced by exposure to UV-A radiation and can be enhanced by elevated temperatures and photooxidative conditions.[104] Scytonemins are exclusively synthesized by cyanobacteria and are chemically very stable. They can accumulate in sediments; their abundance in sediment cores has been utilized to reconstruct variations in the light regime over time.[110] Natural populations of the same species may vary in their concentration, indicating genetic differences.[111]

Phytoplankton

Phytoplankton are by far the major biomass producers in the oceans, and form the basis of the aquatic food webs. Their productivity rivals that of all combined terrestrial ecosystems. Another key ecological factor is that phytoplankton contribute significantly to the biological pump: atmospheric carbon dioxide is taken up by primary producers in the sea and is cycled through primary and secondary consumers. Most of this carbon dioxide returns to the atmosphere, but part of this sinks to the ocean floor as zooplankton fecal pellets and, to a larger extent, as dead phytoplankton.[112] In effect the biological pump removes about 3 – 4 Gt of carbon per year from the atmosphere and partially offsets anthropogenic input of carbon from fossil fuel burning and tropical deforestation.[113]

Phytoplankton are not evenly distributed in the oceans but dominate in the circumpolar regions and the upwelling waters over the continental shelves, as seen by satellite imaging.[114] Estimated cell density differences are in reasonable agreement with measurements in the field.[115] Marine phytoplankton are dominated by small-sized cells of <2 µm diameter.[116] A large number of recent studies points to a considerable sensitivity of phytoplankton communities to solar UV, ranging from polar to tropical habitats.[117]

Besides limitations in nutrients, light availability, pH and non-permissive temperatures, degree of adaptation and grazing pressure, high levels of solar radiation inhibits photosynthesis in species of different taxonomic groups.[118-121] The UV component adds more to photoinhibition than its energy share in solar radiation.[122] This inhibition can be monitored in terms of oxygen exchange,[123] carbon acquisition[124] or by measuring the quantum yield using pulse amplitude modulated (PAM) fluorescence.[125] Nutrient (mainly nitrogen and phosphorus) starvation often augments the UV effects on photosynthetic performance,[126] but may affect various species to a different degree causing changes in community structure.[127] This effect of nutrient deficiency may be caused by less efficient repair processes.[121] In addition, nutrient uptake, such as phosphorus, may be impaired by solar UV radiation.[128] Pollutants such as tributyltin, a constituent of antifouling paints, have a synergistic negative effect.[129, 130]

Photorepair is limited at low temperatures. While at 6° C solar UV radiation significantly inhibited growth in natural phytoplankton samples from a mountain lake in the U.S.A.; no such inhibition was observed at 14°C, indicating that the repair processes compensate the UV inhibition at the elevated temperature.[131]

Experimentally, ozone depletion has been mimicked by adding supplementary UV radiation from lamps to ambient solar radiation. This approach was tested at three locations in Southern Brazil, Canada and Patagonia.[132, 133]

Photoinhibition in terms of photosynthetic quantum yield is linked to the same mechanism as in other eukaryotic photosynthetic organisms from algae to higher plants: the photosynthetic electron transport chain is disrupted by photodegradation of the D1 protein in Photosystem II.[134, 135] Low visible radiation enhances the repair efficiency while high PAR enhances the damage.[136] Inhibition of protein synthesis results in retarded recovery. Nutrient starvation limits recovery also.[136] In contrast to photosynthesis, respiration is less affected by ambient levels of solar UV radiation.[137]

Exposure of natural Antarctic marine plankton to UV at depths from 1 m to less than 20 m showed that some phytoplankton species died, some flourished and others showed no effect.[138] These and other results suggest that ozone-related enhanced UV-B may change food web structure and function which in turn may affect biogeochemical cycles.[139] In Canadian Rocky Mountain lakes solar UV-A and UV-B were found to decrease algal density and alter community composition.[140] However, some studies indicated that after long-term exposure to solar UV, phytoplankton can adapt to the radiation.[141] UV-A had a higher impact than UV-B on hard-bottom shallow marine communities, but the effects on diversity and biomass disappeared during species succession within a few months.[5, 142] Also, in Patagonian oceanic plankton assemblages, UV-A had a stronger effect on photosynthesis during bloom periods than UV-B.[143] However, the relative sensitivity of phytoplankton to UV-A and UV-B may depend on the species composition and the nutrient state.[144] Mixing is an important factor in plankton survival. In contrast to marine habitats with high mixing, lakes often show stable thermal stratification. Consequently, lake plankton communities show vertical distribution[145] and populate certain horizontal bands of optimal light conditions[146] using buoyancy and active motility for niche selection. In the subtropical lake Tanganyika, phytoplankton were affected by solar UV radiation only in the top half-meter, reducing photosynthetic rates, damaging DNA (CPD formation) and inducing UV-absorbing compounds, indicating that vertical mixing decreases solar UV effects by transporting the cells to depth where active repair can take place. Fast vertical mixing within the upper mixing layer of tropical marine environments can enhance photosynthesis. Under cloudy conditions UV-A can be used as a source of energy, while under slow mixing and cloudless skies UV-A is inhibitory.[147] Other targets of UV-B damage are changes in ultrastructure and pigment concentration and composition.[148, 149] Besides direct effects on cellular targets, UV-B also operates via the production of ROS.[150] Phytoplankton defend themselves by activating antioxidant systems. However, UV-B decreases the activity of antioxidant enzymes and ROS scavengers.[151]

One mechanism of photoprotection against high solar radiation in many algal species (except red algae) is the xanthophyll cycle, which relies on the thermal dissipation of excess excitation energy thereby reducing the formation of singlet oxygen in the chloroplasts.[152] Zeaxanthin formation is also involved in increased non-photochemical quenching based on the migration of electronic excitation energy from Photosystem II chlorophyll to nearby carotenoids. UV exposure can enhance this process.[153]

MAAs are effective UV screens that protect phytoplankton from high solar UV radiation.[154] In the English channel MAAs are present on a year round basis with concentrations increasing rapidly during spring often coinciding with the appearance of algal blooms.[155] The action spectrum for MAA synthesis induction shows a clear maximum in the UV-B range.[156] In the dinoflagellate *Scrippsiella,* daily vertical migrations have been found to be related to circadian MAA biosynthesis.[157, 158] In dinoflagellates, MAAs seem to be packaged in certain organelles probably increasing the protective efficiency for specific cellular targets.[159] MAAs can operate both as UV absorbers and as quenchers for oxidative stressors.[160, 161] While MAAs are very stable molecules with respect to extreme temperatures, pH and UV radiation, they are easily destroyed in water in the presence of photosensitizers.[162]

Some freshwater yeasts represent a small group of planktonic organisms showing both a constitutive and a UV-inducible synthesis of photoprotective carotenoids and mycosporines.[163-165] The specific MAA is a compound linked to a glutaminol-glucoside,[74] which is also accumulated by copepods and ciliates from their diet.[165] Some green algae in extreme UV environments (snow algae) use sporopollenin as a UV-absorbing substance.[166] Others rely on massive accumulations of carotenoids such as astaxanthin[167] or β-carotene,[168] which provide protection against oxidative stress by scavenging singlet oxygen or peroxyl radicals.[159]

Some phytoplankton taxa including dinoflagellates and diatoms produce toxic substances, such as neurotoxins and domoic acid, and are a severe threat to animals and humans when they form blooms. Recent blooms of the toxic *Pseudo-nitzschia* have caused mass mortality among dolphins, sea lions and birds along the Californian coast.[169] These blooms seem to be increasing in frequency and geographical range. The organisms have a low sensitivity to solar UV radiation and escape damage of their photosynthetic apparatus by switching to heterotrophic growth.

Several taxa of marine phytoplankton such as Prymnesiophyceae and some dinoflagellates produce dimethylsulfoniopropionate (DMSP) which is converted into dimethylsulfide (DMS)(cf. Chapter 5). The latter is emitted into the atmosphere and forms cloud condensation nuclei, thereby affecting local climate over the ocean.[170] Cleavage of DMSP is induced by mechanical or dark stress, by grazing or viral attack.[171] This indicates that DMSP is involved in coping with oxidative stress.[172, 173] Because of the pronounced vertical migrations of the dinoflagellates, diurnal patterns were recorded in DMS production in the St. Lawrence Estuary. Recently, lakes and estuaries have also been found to be important sources of DMS.[174] A model has been developed to simulate the seasonal patterns of DMS production and validated against nutrient concentrations, biological standing stock and other parameters.[175] Marine biogenic iodocarbon emissions are also significant for marine aerosol formation and have a key effect on global radiative forcing.[176] Besides changes in stratospheric ozone, cloud cover is a major factor controlling the exposure of organisms to solar UV.[177]

The sea-ice ecosystems in the circumpolar oceans and water bodies of the Baltic and Caspian Seas constitute some of the largest biomes on Earth.[178] The semisolid ice matrix provides niches in which bacteria, phytoplankton algae, protists and invertebrates thrive.[179] Those organisms are strongly affected by temperature, salinity, nutrients, visible and ultraviolet solar radiation.[180] Sea-ice phytoplankton provide the fundamental energy and nutritional source for invertebrates such as krill in their early developmental stages which amount to about a quarter of the biomass production in ice-covered waters. The extreme conditions of their habitat force the organisms to adapt physiologically. The production of large concentrations of MAAs is also essential for the survival of primary consumers which ingest and incorporate the MAAs for their own protection. The expected loss of about 25 % of the sea ice due to global warming over the current century will certainly affect the productivity of the polar oceans.[180]

Anthropogenic acidification of boreal lakes decreases resistance of organisms to UV radiation and affects species composition with increasing trophic level. Therefore it is assumed that loss in species diversity will increase the susceptibility of acidified lakes to other stress factors. Ecosystem stability in boreal lakes is thus likely to decline as global change proceeds.[181]

Experiments in large (volume >1 m^3) outdoor enclosures, called mesocosms, are useful for the study of complex impacts on food-web structure and dynamics.[182-184] Mesocosms permit well-controlled experiments with natural phytoplankton communities in physical, chemical and light conditions mimicking those of the natural environment. In addition, UV radiation within mesocosms can be manipulated to simulate various levels of ozone depletion. Belzil and coworkers[182] find that while UV radiation increases can have subtle effects on bulk biomass (carbon and chlorophyll), changes in community structure may be a more significant ecological effect, because of differential sensitivity to UV radiation among planktonic organisms. These workers note that “planktonic communities do not suffer from the catastrophic negative impacts that might have been inferred from some laboratory experiments on individual components of the marine food web”. They note, in agreement with previous observations, that ambient levels of UV radiation already have significant effects. Mesocosm experiments, including both plankton and their grazers, also suggest that changes in community structure are potentially more important than effects on overall algal biomass.[184] Other workers found that phytoplankton growth was inhibited by UV radiation in fixed-depth experiments but not in mesocosms where vertical mixing exposed planktonic organisms to variable radiation regimes.[185] A synthesis model simulating mesocosm experiments suggests that enhanced UV-B could cause “a shift from primary producers to bacteria at the community level”.[186] Such a shift in community structure could have important consequences for CO_2 levels in oceanic surface waters. A mathematical model based on a predator-prey scheme considers sedimentation of phytoplankton, vertical mixing, and attenuation of PAR as well as UV radiation in the water column. Surprisingly, higher inhibition by UV radiation and longer mixing periods can induce strong fluctuations in the system and enhance plankton productivity due to the stronger effects on the predators.[187, 188]

Macroalgae and Aquatic Plants

Macroalgae are major biomass producers on rocky shores and continental shelves. The macroalgae canopies form habitats for larval fish, crustaceans, and other animals. Macroalgae are of commercial importance and are harvested on a large scale from natural vegetation and aquaculture for human consumption and industrial use.

Even without ozone depletion, UV-B radiation constitutes a significant stressor for macroalgae. Exposure to solar UV-B results in a host of biological effects on the molecular, cellular, individual and community levels.[189] Macroalgae are stressed by solar UV radiation to an extent which is genetically determined and results in a pronounced vertical stratification.[190] Even closely related species of the same genus may have significantly different UV sensitivity, causing them to grow in different habitats.[191] UV-tolerant species populate the tidal zone, while more sensitive species are found in deeper waters.[192] Seasonal changes in UV and visible radiation also result in a pronounced succession of species over the year in marine macrobenthic communities.[193] Besides changing salinity, temperature and desiccation in their habitats,[194] macroalgae are exposed to extreme variations in light intensity due to daily, seasonal and tidal cycles as well as changing turbidity in the water column.[195] Intertidal macroalgae of all major taxa can rapidly adapt to fast changes in radiation.[196, 197] Environmental conditions can be extreme in macroalgal habitats where, at polar growth sites, species have to survive in total darkness during several winter months.[198]

Young specimens were more prone to UV inhibition of photosynthesis, and species collected shortly after the winter were found to be affected more than those harvested later in the year, indicating an adaptive strategy to increasing natural short-wavelength radiation.[199] Both Arctic and Antarctic species showed pronounced effects of solar UV-B on photosynthesis, morphology and growth rates.[200, 201] Unfiltered solar radiation proved lethal to several Antarctic deep water algae. While tropical macroalgae are better adapted to higher solar UV and visible radiation than higher-latitude species, they are also affected by ambient solar UV.[202] Both UV-A and UV-B decrease growth rate, quantum yield of photosynthesis and cause accumulation of DNA damage. Since different species show different sensitivities, increases in solar UV-B radiation could influence species recruitment in the upper intertidal zone.[203]

Excessive solar radiation causes photoinhibition of photosynthesis;[204] elimination of total UV or UV-B alone reduces the severity of photoinhibition and shortens recovery time in many species.[152, 205, 206] Electron microscopy revealed pronounced damage of the thylakoid structure.[207] Enzymes involved in the photosynthetic CO_2 fixation and sugar formation are affected by UV radiation and the concentration of chlorophyll *a* decreases.[208, 209] The photosynthetic accessory phycobiliproteins operating as antenna pigments in red algae are even more sensitive to solar UV radiation.[210] UV-B is more effective than UV-A in decreasing growth rate.[203] In a laboratory study exposure to UV resulted in significant release of organohalogens from several polar macroalgae. These substances have ozone-depleting characteristics and so potentially enhance the incidence of solar UV.[203]

Most macroalgae have an efficient photorepair system of UV-induced CPDs.[211] Besides DNA repair mechanisms, efficient ROS scavenging enzymes were found in many macroalgae.[212] In several Arctic algae these enzymes vary significantly in activity over the growing season when algae have been collected before, during and after break-up of sea ice.[213] UV sensitivity decreases with age and developmental stage of macroalgae. The germination capacity of zoospores from five Laminariales species were found to decrease sharply after 16 h of exposure to visible and UV radiation.[214] Both zygotes and young germlings of brown algae show massive inhibition; UV-B radiation is more effective than UV-A.[215] Also juvenile stages of red and green algae showed a pronounced UV sensitivity.[216] Both UV-A and blue radiation reactivate spore germination after UV-B inhibition, indicating photolyase activity.[217] Motile gametes of brown algae use light-directed movement (phototaxis) to accumulate at the water surface improving the chances of finding a mating partner, but that phototactic response is drastically inhibited by solar UV. Enhanced levels of solar UV-B may affect this vital strategy and thus impair development of kelps.[218]

Many macroalgae of the tidal zone produce UV-absorbing compounds while subtidal species usually do not have this protection. However, deep-water algae are rarely exposed to significant levels of solar UV radiation.[219] Red algae have the highest percentage of species that synthesize MAAs,[220] followed by brown and green algae. The protective effect of MAAs was shown in the red alga *Porphyra*, commercially sold as Nori, where they block thymine dimer production. [221] MAAs are very stable against elevated temperatures and UV exposure.[222] The presence of ammonium increases the accumulation of MAAs. The blue component of visible radiation has the highest effect in inducing MAA biosynthesis in *Porphyra*.[223] Polychromatic action spectra of induction reveal the efficiency of short wavelength radiation in several species.[224, 225] Recently a new group of MAAs absorbing at 322 nm has been identified in green algae.[226] The common sea lettuce, *Ulva*, was found to produce a UV-B absorbing compound with a maximum at 292 nm.[227] In brown algae a novel group of UV absorbing pigments, phlorotannins, has been found.[228] Macroalgae can be classified according to their MAA production. Most deep water algae never produce MAAs even when transplanted to surface waters. Algae from the intertidal zone often show induction of MAAs, while species growing near the water surface normally have a high concentration of MAAs, which cannot be further induced.[229] Other defense mechanisms against photooxidative stress involve the induction of a wide range of antioxidant enzymes in brown, green and red algae[230] as well as biosynthesis of several carotenoids.[224]

Aquatic mosses and liverworts show UV-B-related responses similar to those of many macroalgae, including inhibition of photosynthesis, growth and pigmentation.[231, 232] PAM measurements show a pronounced photoinhibition during noon, from which the thalli recover when the UV stress decreases.[107] When exposed to high levels of solar UV-B radiation they produce UV-absorbing compounds, which seem to be hydroxycinnamic acid derivatives.[233] Aquatic flowering plants are also affected by solar UV. Sea grass meadows cover large areas of sandy bottom in shallow water[234] and contribute substantially to the aquatic biomass productivity.[235] Photosynthetic quantum yield dramatically decreases under unfiltered solar radiation. Removal of UV-B or total UV improves the photosynthetic activity.[236] Transfer experiments on plants growing at 15 m to 2.5 m water depth indicate an efficient adaptation of sea grasses to higher solar UV. Epiphytes growing on sea grass leaves has been considered detrimental since it reduces the photosynthetically available radiation, but as they strongly absorb UV-B radiation they exert a beneficial effect.[237] In a submersed aquatic angiosperm, UV-B exposure over 7 – 16 days caused an increase in several photosynthetic enzymes. Water transparency to visible and UV governs the distribution and abundance of submerged macrophytes in lakes in the Canadian Arctic.[238] Antioxidant enzymes were also activated by UV.[239] The common freshwater duckweed, *Lemna*, shows strong responses to simulated solar radiation, with a pronounced increase in ROS responses. This UV-induced stress response was augmented by exposure to copper, which alone also activates the ROS pathway.[240] Related species differ considerably in their UV-B sensitivity.[241]

Consumers

Consumers form the next higher level in the aquatic food webs after producers (Figure 4-3). In most cases several trophic levels follow each other, usually starting with zooplankton being the primary consumers. It is evident that a UV-related decrease in primary producer biomass has an effect on growth and survival of the consumers. In addition, specific UV effects have been identified in almost all consumers.[242]

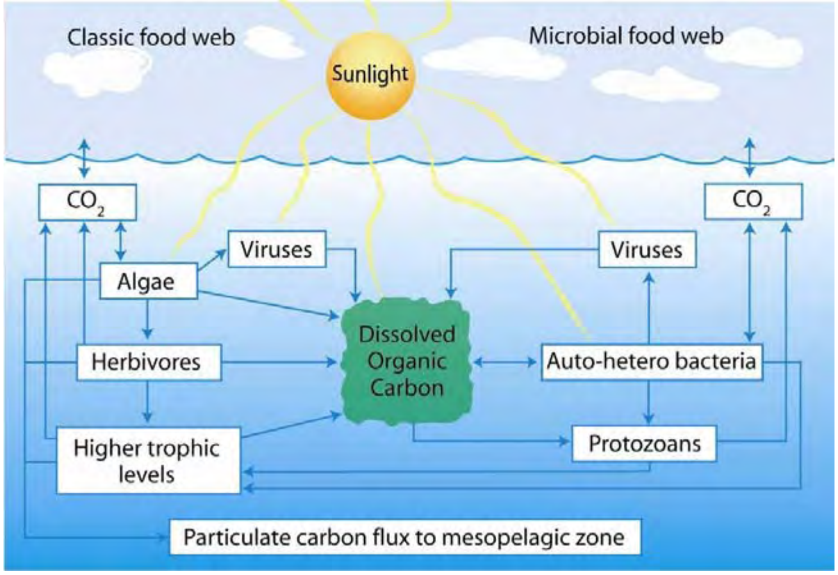

Figure 4-3. Schematic diagram of classic and microbial marine food webs illustrating the flow of carbon and energy through the systems. Adapted from DeLong and Karl, courtesy of the National Biological Information Infrastructure (NBII).[243]

Zooplankton

Zooplankton includes unicellular and multicellular life forms and can be classified in several size classes. It is also comprised of larval forms of fish, crustaceans, echinoderms, mollusks and other phyla. These forms will be discussed below.

Zooplankton community structure in freshwater ecosystems is controlled by multiple factors, including DOC content and distribution throughout the water column, which regulates UV penetration (see Chapter 1). UV radiation is a potential driving force for zooplankton community structure in some lakes.[244] In shallow ponds of Finnish Lapland *Daphnia* only occurs when sufficient amounts of DOC are present.[245] Depending on the terrestrial succession in the watersheds of several Alaskan lakes, the UV attenuation depths (1% of surface irradiance at 320 nm) vary from 0.6 m to more than 14 m. This UV regime strongly controls the species composition of major macrozooplankton. When zooplankton from a UV-opaque lake was transplanted into the surface water (0.5 m depth) of a UV-transparent lake, it perished within only a few days, suggesting a strong link between early succession of zooplankton communities and terrestrial plant communities (a source of DOC) within the watershed. Large variations in UV sensitivity were also found in a study involving lakes of different UV transparencies.[246, 247] In response to high solar UV, *Daphnia* shows a pronounced avoidance response when observed in UV transmitting acrylic columns suspended in the surface waters. In contrast, when UV-B and short-wavelength UV-A are blocked, the animals prefer moving to the surface. In a low-UV lake, no such preferential behavior was seen. These results and those from a follow-up, open-lake experiment indicate that UV radiation may influence the vertical distribution and habitat partitioning of certain zooplankton in high-UV lakes, while predation, food availability and other factors may be more important in low-UV lakes.[248] Studies of sublethal effects of UV on the freshwater cladoceran *Daphnia* show increases in respiration rates at low levels of UV exposure and decreases at high levels.[249]

In their natural habitat, zooplankton face conflicting selection pressures. While invertebrate predators induce an upward movement during daylight hours, this exposes zooplankton to strong surface UV exposure.[250] Even though *Daphnia* and other zooplankton try to escape from surface UV radiation by vertical migration, the organisms cannot avoid excessive exposure. The copepod *Boeckella*, living in Lake Titicaca with very high solar UV levels, counters the detrimental effect by incorporating photoprotective MAAs.[251] Copepods cannot synthesize these substances but acquire them from their algal diet (e.g., dinoflagellates).[158]

In a study of Antarctic copepods, MAA concentration was strongly correlated with UV tolerance.[252] In an alpine lake there was a strong seasonality in MAA concentrations in phytoplankton and copepods with more than three times higher concentrations in the summer than in the winter.[253] Besides vertical migration and UV screening, copepods rely on photorepair of UV-B-induced DNA damage[254] as shown in species from Patagonia, Argentina.[255] Photoenzymatic repair contributes significantly towards UV-B tolerance in many cladocerans.[256] Some Antarctic copepods possess a less efficient photorepair mechanism, which has been attributed to the low temperatures typical of Antarctic lakes.[252] The implication is that at elevated temperatures (due to global warming) the enzymatic photorepair of UV-induced damage should be more efficient.[257] This hypothesis was tested in living *Daphnia* by extracting DNA at various temperatures. UV-induced DNA damage increased with temperature, but the light-dependent enzymatic repair more than offset the effect and the net DNA damage significantly decreased with increasing temperature.[258] This result was supported by a study of planktonic rotifers and crustaceans in Northern temperate lakes where UV had less detrimental effects on abundance and reproduction at higher temperatures.[259] However, one study found that mortality and DNA damage were as high as at low temperatures in freshwater ciliates, indicating that photolyase has an optimal temperature for its activity.[260] It is interesting to note that though elevated levels of solar UV induce mutations, there does not seem to be evolutionary selection toward UV protection in halophilic crustaceans.[261] Feeding experiments indicate that UV-B pretreated phytoplankton species negatively affect the life history of *Daphnia*.[262] Adults were smaller, and a smaller number of juveniles with lower fitness were produced under these conditions than in the controls, indicating that UV-B had a significant effect on food quality and impaired energy transfer to the next trophic level.[263, 264] The effect of climate warming on macro-zooplankton is subtle: Copepod populations were reduced in size but those of ostracods increased.[265] In contrast predation by fish has a major effect on population composition and density.

Several workers have reported results consistent with the hypothesis that UV influences zooplankton community structure and succession during early lake ontogeny. Engstrom and co-workers[266] studied the chemical and biological trends during lake evolution in recently deglaciated terrain near Glacier Bay, Alaska. They demonstrated that dissolved organic carbon (DOC) concentrations increased with lake age. Williamson et al.,[267] investigating changes in UV attenuation and macrozooplankton community structure in these same lakes, showed a strong dependence of UV radiation transparency on terrestrially derived DOC. They suggest a link between the development of terrestrial plant communities within these lake watersheds, changes in lake hydrology, and the early succession of zooplankton communities following deglaciation. These results suggest that UV radiation may be a more important factor than previously recognized in determining the distribution and abundance of zooplankton in lake ecosystems.

Corals and Sea Anemones

Recent accelerated catastrophic coral mortality has been linked with several environmental factors including bacterial and cyanobacterial infections,[268] increasing temperatures, [269-272] marine pollution[273] and human destruction of coral reefs. Many corals rely on the photosynthetic activity of dinoflagellates (zooxanthellae).[274] At temperatures exceeding a thermal threshold, corals are bleached. The underlying mechanism could be photoinhibition of photosynthesis in the zooxanthellae induced by the production of reactive oxygen species.[275, 276] However, recent results indicate that corals and their symbionts may be capable of adapting to higher temperatures.[277] Like corals, giant clams harbor symbiotic zooxanthellae. Clams also suffered mass bleaching on several reefs of the Great Barrier Reef.[278] Virus-like particles could also be associated with coral mortality.[279]

When symbiotic algae are exposed to solar radiation the host is also subjected to damaging solar UV radiation. Some stony corals expand their tentacles upon exposure to photosynthetically active radiation and contract them when encountering excessive radiation.[280] As a counter-measure to enhanced solar UV the algae produce MAAs, some of which are also transferred to the host.[274] Moreover, the host develops antioxidant defenses to protect itself from the photosynthetically produced oxygen. Herbicides also affect corals by impairing the photosynthetic symbiotic zooxanthellae.[281] Laboratory-kept colonies of the coral *Stylophora* maintained minimal amounts of MAAs, but the concentration of the UV-absorbing pigments increased rapidly upon exposure to broadband UV.[282] Four MAAs, produced by the zooxanthella *Symbiodinium*, increased first, followed by six additional ones which were synthesized at the expense of the primary MAAs.

Sea anemones occur in several color phenotypes. At the coast of Discovery Bay, Jamaica, pink morphs are more abundant in the lagoon and in deeper areas, while green individuals are found in the forereef (seaward and downward from the reef crest) and in shallower areas. Genetic analysis revealed two distinct variants with different UV absorbance and UV acclimatization capacities.[283] A comparison of sea anemones with dinoflagellates or green algae as symbionts or asymbiotic species showed that the MAAs mainly reflect phylogenetic differences among the anemones rather than the presence or kind of symbiont.[284]

Sea Urchins

Exposure to UV radiation causes apoptosis (cell self-destruction) in developing sea urchin embryos.[285] Embryos of three sea urchin species from different habitats ranging from the Gulf of Maine to the Antarctic indicated significant amounts of accumulated DNA damage in the form of cyclobutane pyrimidine dimers (CPD). Biological weighting functions for DNA damage indicated a high sensitivity for UV-A radiation, but the most sensitive species show an increased susceptibility to UV-B correlated with the lowest concentration of UV-absorbing compounds.[286] Larvae and embryos of these species dwell within 5 m of the ocean surface. UV-induced damage in the different larval stages was clearly correlated with the absence of MAAs. The absence of UV-screening substances strongly decreased survival.[287] Further, the observed delays in early cleavage and following development were closely related with UV-induced DNA damage. Reproduction in the circumpolar sea urchin *Sterechinus* occurs during austral spring when ozone concentrations during the past 25 years have declined by more than 50%. When the planktonic embryos were exposed in the top 1 m of the water column, nearly all exhibited DNA damage and 100% showed abnormal development.[288] UV-B removal prevented DNA damage. At depths below 3 m hardly any abnormal development or DNA damage occurred. The threshold for DNA damage from ambient solar UV-B was ≤ 25 kJm^{-2} (inducing ~17 CPDs mb^{-1}) and levels >80 kJm^{-2} precluded normal development.

The Antarctic sea ice has been thought to protect the benthic invertebrate fauna from solar UV-B radiation. However, recent investigations showed that short-wavelength UV-B (down to 304 nm) is transmitted through the austral spring annual ice of McMurdo Sound where it causes DNA damage and mortality during the early development in sea urchin embryos.[289] The degree of damage and mortality varies from year to year and depends on the thickness of the sea ice and on the total column ozone.

Amphibians

During the last decade amphibian populations have suffered widespread declines and even extinctions on a global scale.[290, 291] Many different factors, including habitat destruction[292, 293] and fragmentation,[294-297] global climate change,[298, 299] acid precipitation,[300, 301] environmental pollution,[302-305] including anthropogenic pesticides[304, 306] and fertilizers,[307] parasites,[308] introduction of exotic competitors and predators,[309-314] fungal diseases,[315, 316] and other pathogen outbreaks,[317-319] interannual variability in precipitation, as well as climate change-induced reductions in water depth at oviposition sites, have been suggested as responsible for those global declines.[320, 321] Since the 1990s, malformations have been noted in many parts of the United States[321] and in many other countries all over the globe.[322, 323]

Among other factors, solar UV-B radiation has been variously implicated as a possible contributing factor[324] involved in malformation and mortality, especially during the embryonic development. However, there are two conflicting views on the involvement of UV-B in amphibian declines.[325, 326] In a controlled laboratory study, leopard frogs (*Rana pipiens*) were exposed to unfiltered solar radiation or radiation without UV-B or total UV.[327] Unlike natural conditions, the larvae in the laboratory could not avoid exposure. Full sunlight caused ca. 50% mortality in early larval development, while filtered solar radiation had no effect. There was a clear correlation between solar UV doses and hindlimb malformation. In situ studies in the natural amphibian habitat showed a considerable protection from solar UV radiation by DOC and vegetation shading, especially during the sensitive development during spring.[328] When exposed to ambient solar radiation under controlled conditions and when natural shade and refuge were eliminated, embryos and larvae of several anuran species died.[329] A subsequent quantification of the outdoor UV exposure in Northern Minnesota and Wisconsin wetlands indicated that the risks for UV-induced malformations and mortality are low for both Northern leopard and mink frogs. The exposure of amphibian eggs and larvae to solar UV radiation strongly depends on the concentration of DOC in the water column.[330] One important factor is oviposition behavior: species which lay the eggs in UV-protected sites may be more sensitive to solar UV exposure than those which deposit their eggs at the water surface.[331] Amphibian species with the highest physiological sensitivity to UV-B are those with the lowest field exposures as a function of the location of embryos and the UV-B attenuations properties of water at each site. These results also suggest that conclusions made about vulnerability of species to UV-B in the absence of information on field exposures may often be misleading.[331]

Red-legged frog embryos (*Rana aurora*) appear to be tolerant to current ambient levels of UV-B, but radiation even slightly exceeding the ambient levels is lethal.[332] Although embryonic size is a complicated issue and small size at hatching can change very quickly after feeding, even at ambient levels, larvae exposed to UV-B as embryos tend to be smaller and less developed than non-exposed organisms. Amphibians use behavioral, physiological and molecular defenses against solar UV-B damage, but species-specific sensitivities may cause changes in community structure due to persistent UV-B level increases,[333] but because some species may be more successful than others, changes in species composition can result.[333]

Fishes

Although humans use about 8% of the productivity of the oceans, that fraction increases to more than 25% for upwelling areas and to 35% for temperate continental shelf systems. For about one-sixth of the world's population (primarily developing nations), the oceans provide at least 20% of their animal protein. 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, increased water temperature, pollution and disease, and/or exposure to increased UV-B radiation may contribute to that decline. The eggs and larvae of many fish are sensitive to UV-B exposure (Figure 4-4). 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 effects of ozone depletion on marine fish populations.

Visual predators, including most fish, are necessarily exposed to damaging levels of solar UV radiation. Skin and ocular components can be damaged by UV,[334] but large differences are found between different species.[335] Coral reef fishes can adapt to the UV stress by incorporating UV-absorbing substances, which they acquire through their diet, into their eyes and epidermal slime.[336] Exposure to solar radiation induced "suntanning" in red seabream. Histological, colorimetric and chemical assays showed that the sun-exposed fish had an up to five times higher concentration of melanin.[337] In addition to direct effects, including damage to biological molecules such as DNA and proteins and the generation of reactive oxygen species, photoactivation of organic pollutants and photosensitization may be detrimental. The damaging effects on eggs and larval stages may be enhanced by polycyclic aromatic hydrocarbons (PAHs) such as retene, which is a pollutant from pulp and paper mills.[338]

In goldfish, embryos are prone to UV effects during early development[339] and produce CPDs under UV radiation. These are more efficiently repaired in the presence of light.[340] Solar UV radiation has been shown to induce DNA damage in the eggs and larvae of the Atlantic cod,[294] where larvae were more sensitive than eggs. Artificial UV causes massive apoptosis in larval embryos of Japanese flounders.[341] Studies addressing biological weighting functions indicated a strong sensitivity towards solar UV-B. CPD loads as low as 10 per megabase DNA resulted in approximately 10% mortality. Use of video taping and measurement of oxygen consumption showed sublethal effects of UV radiation in juvenile rainbow trout[342]. Under worst-case scenarios (60% ozone loss, sunny weather and low water turbulence), solar UV-B eliminated buoyancy and caused mortality within 1 or 2 days.

Figure 4-4. Fish eggs and larvae are specifically prone to UV-B radiation. Salmon Alevin larva has grown around the remains of the yolk sac. In about 24 h it will be a fry without yolk sac (courtesy Uwe Kils)

Fish spawning depth strongly correlates with UV exposure. In-situ incubation experiments have shown that in a highly UV transparent lake 100% of yellow perch eggs (*Perca flavescens*) are killed before hatching when exposed to full solar UV.[343] In this same lake 92% of eggs are spawned at depths greater than 3 m, while in a nearby lake with low UV transparency 76% of eggs were spawned at depths shallower than 1 m. It is not known whether the fish are able to detect and avoid the high UV at shallower depths in the high UV lake or whether this spawning pattern is due simply to differential survival. In either case, the deeper spawning depths place the eggs in colder water where it takes them much longer to hatch compared to eggs spawned in the warm surface waters. A similar phenomenon has been observed in bluegill larvae (*Lepomis macrochirus*) in a UV-transparent lake where in 19 % of nests the estimated UV-induced mortality of larvae exceeds 25 %. Most nests are exposed to relatively low UV levels because they are either located at deeper depths or under overhanging branches.[344] In fish aquaculture, specific measures are introduced, such as installing UV sunscreens to avoid UV damage to larval fish in the usually shallow habitats.[345]

Other Aquatic Animals

Early life stages of marine organisms, particularly eggs and larvae, are vulnerable to solar UV-B radiation. Rocky shore mollusks show an increased mortality and retarded development upon UV exposure. These detrimental effects are synergistically enhanced in the presence of other stress factors such as high temperatures or salinity, pointing to strong underestimation of the ecological impacts of climate change by not accounting for the complex interactions among such environmental variables as temperature, salinity and oxygen availability.[346] Desiccation enhances mortality and negatively affects development in encapsulated embryos of rocky shore gastropods.[347]

The amphipod *Amphitoe valida* has high concentrations of MAAs and consequently low mortality while the isopod *Idothea baltica* has low MAA concentrations and shows high mortality. However, the latter species deposits all available MAAs into the eggs and embryos conferring protection to the progeny.[348]

Conclusions and Consequences

With the recognition of the importance of UV radiation effects on aquatic ecosystems, there has been a plethora of publications show that solar UV can adversely affects aquatic organisms. These studies document substantial impact on individual species yet considerable uncertainty remains with respect to assessing effects on ecosystems. Several studies indicate that the impact of increased UV radiation would be relatively low when considering overall biomass response while often, in contrast, the response is quite marked when the abundance, distribution and effects on individual species are considered. Ecosystem response to climate variability involves both synergistic and antagonistic influences with respect to UV radiation-related effects on aquatic ecosystems and these influences significantly complicate comprehension and prediction at the ecosystem level. With respect to assessing UV radiation-related effects, the influence of climate variability is often more important via indirect effects such as reduction in sea ice, changes in water column bio-optical characteristics and shifts in oceanographic biogeochemical provinces than through direct effects. Decreases in primary production would result in reduced sink capacity for atmospheric carbon dioxide, with its related effects on climate change.

The global decline of amphibian populations seems to be related to several complex, interacting causes. While one review clearly rejected any link between solar UV-B radiation and amphibian decline[326] evidence from more than 50 peer-reviewed publications from around the world shows that dozens of amphibian species are affected by UV-B.[325]

A number of new studies have both confirmed and strengthened evidence that UV-B has an important influence on community structure of various aquatic ecosystems. In lakes, phytoplankton abundance may vary by orders of magnitude depending upon future climate-DOM-UV interactions.[26] Also, lakes often show thermal stratification and as a consequence plankton communities show vertical distributions where the UV regime can strongly control species composition.[235] Other evidence supports the hypothesis that UV influences zooplankton and community structure and succession during early lake ontogeny.[267] Mesocosm studies, including both phytoplankton and their grazers, suggest that species composition and population structure may be more influenced by UV-B than overall algal biomass.[184] These results suggest that UV radiation may be a more important factor than previously recognized in determining community structure in aquatic systems.[349]

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Chapter 5. Interactive effects of solar UV radiation and climate change on biogeochemical cycling

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Summary

This report assesses research on the interactions of UV radiation (280-400 nm) and global climate change with global biogeochemical cycles at the Earth's surface. The effects of UV-B (280-315 nm), which are dependent on the stratospheric ozone layer, on biogeochemical cycles are often linked to concurrent exposure to UV-A radiation (315-400 nm), which is influenced by global climate change. These interactions involving UV radiation (the combination of UV-B and UV-A) are central to the prediction and evaluation of future Earth environmental conditions. There is increasing evidence that elevated UV-B radiation has significant effects on the terrestrial biosphere with implications for the cycling of carbon, nitrogen and other elements. The cycling of carbon and inorganic nutrients such as nitrogen can be affected by UV-B-mediated changes in communities of soil organisms, probably due to the effects of UV-B radiation on plant root exudation and/or the chemistry of dead plant material falling to the soil. In arid environments direct photodegradation can play a major role in the decay of plant litter, and UV-B radiation is responsible for a significant part of this photodegradation. UV-B radiation strongly influences aquatic carbon, nitrogen, sulfur and metals cycling that affect a wide range of life processes. UV-B radiation changes the biological availability of dissolved organic matter to microorganisms, and accelerates its transformation into dissolved inorganic carbon and nitrogen, including carbon dioxide and ammonium. The coloured part of dissolved organic matter (CDOM) controls the penetration of UV radiation into water bodies, but CDOM is also photodegraded by solar UV radiation. Changes in CDOM influence the penetration of UV radiation into water bodies with major consequences for aquatic biogeochemical processes. Changes in aquatic primary productivity and decomposition due to climate-related changes in circulation and nutrient supply occur concurrently with exposure to increased UV-B radiation, and have synergistic effects on the penetration of light into aquatic ecosystems. Future changes in climate will enhance stratification of lakes and the ocean, which will intensify photodegradation of CDOM by UV radiation. The resultant increase in the transparency of water bodies may increase UV-B effects on aquatic biogeochemistry in the surface layer. Changing solar UV radiation and climate also interact to influence exchanges of trace gases,

such as halocarbons (e.g., methyl bromide) which influence ozone depletion, and sulfur gases (e.g., dimethylsulfide) that oxidize to produce sulfate aerosols that cool the marine atmosphere. UV radiation affects the biological availability of iron, copper and other trace metals in aquatic environments thus potentially affecting metal toxicity and the growth of phytoplankton and other microorganisms that are involved in carbon and nitrogen cycling. Future changes in ecosystem distribution due to alterations in the physical and chemical climate interact with ozone-modulated changes in UV-B radiation. These interactions between the effects of climate change and UV-B radiation on biogeochemical cycles in terrestrial and aquatic systems may partially offset the beneficial effects of an ozone recovery.

Introduction

Global biogeochemistry plays a critical role in controlling life processes, climate and their interactions, including effects on atmospheric greenhouse gas concentrations. Changes in stratospheric ozone and hence in solar UV-B radiation (280-315nm) have many different effects on global biogeochemistry. Longer wavelength UV-A radiation (315- 400 nm) is little affected by ozone depletion, but can be affected by global climate change. UV radiation (280-400 nm), including both UV-B and UV-A, modifies carbon cycling through changes in its capture (photosynthesis), storage in biomass and non-living organic matter, and release (respiration and photochemical decomposition). The effects of UV radiation on carbon cycling are linked to effects on the cycling of metals and mineral nutrients such as nitrogen. Carbon and nutrient cycles are also substantially affected by other components of climate change, including warming, elevated CO_2 and altered patterns of precipitation, and there are also significant interactions between these factors and changes in UV radiation. Interactions between changing solar UV radiation and climate change occur through the effects of UV radiation on emissions of trace gases, such as carbon monoxide, carbonyl sulfide, methane, methyl bromide and dimethylsulfide. Climate change can alter the exposure of ecosystems to UV-B radiation by influencing the Earth system processes that affect ozone depletion (Chapter 1) as well as changes in aquatic UV-absorbing substances such as colored dissolved organic matter (CDOM). Biological responses of organisms to changing UV radiation and interactions with climate change are considered in detailed in Chapers 3 and 4. This chapter examines the responses of global biogeochemistry to interactions between stratospheric ozone depletion and co-occurring environmental changes in climate, land use, and atmospheric CO_2 (Figure 5-1). The primary focus is on new information obtained since our 2002 report[1] although in some instances reference is also made to earlier key publications.

Carbon Cycling

There is a consensus that terrestrial ecosystems are currently net sinks for carbon, i.e. that the uptake of CO_2 exceeds its release. The net accumulation of carbon by terrestrial ecosystems has been calculated to be around 0.7 petagrams (Pg) (1 Pg equals 1015 g, or 109 metric tons) carbon per year for the 1990s, although estimates vary widely.[2] This is substantially smaller than current estimates of oceanic CO_2 uptake (around 2.4 Pg carbon per year for the 1990s). However, the carbon balance of terrestrial systems is profoundly influenced by human activities such as land-use change, which may result in CO_2 emissions as great as 2.2 Pg carbon per year.[2, 3]

The exchange of carbon dioxide between the atmosphere and ecosystems is a balance between CO_2 uptake by photosynthesis, its storage in biomass and non-living organic matter, and its release through respiration, photodegradation, and burning. Changes in solar UV radiation have been shown to influence many of these processes.

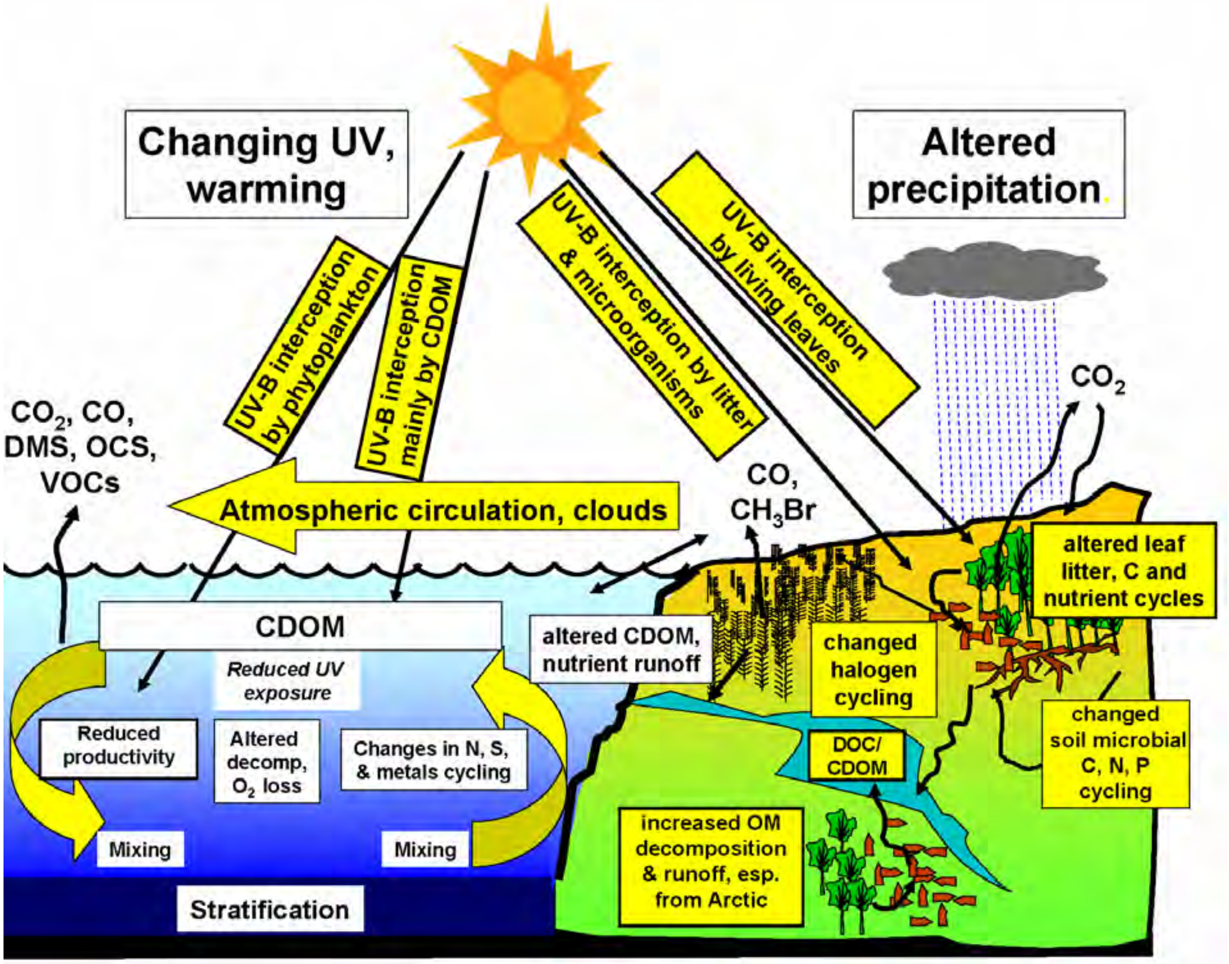

Figure 5-1. Conceptual model illustrating the potential effects of enhanced UV radiation and climate change on biogeochemical cycles : OM organic matter; DOM dissolved organic matter; CDOM colored (chromophoric) DOM; CO_2 carbon dioxide; CO carbon monoxide; DMS dimethylsulfide; OCS carbonyl sulfide; VOCs volatile organic hydrocarbons; CH_3Br methyl bromide.

Carbon Fixation

The balance of evidence continues to suggest that changes in UV-B radiation resulting from ozone depletion will have little effect on carbon fixation and growth in most terrestrial plants (see Chapter 3) and hence on large-scale carbon capture and storage by terrestrial vegetation. However, certain plant species or communities may be vulnerable to increased UV-B radiation and this may have significant ecological impacts in specific systems (see Chapter 3).

Whereas in terrestrial ecosystems changes in solar UV radiation influence carbon fixation directly through effects on plants, in aquatic systems, the primary producers are affected both directly and indirectly by solar UV radiation. The indirect effects are caused by changes in exposure to UV radiation due to variations in the concentration of CDOM and, to a lesser extent, suspended particles. CDOM is the main UV-absorbing constituent in aquatic systems and controls the penetration of UV radiation into water bodies. Organic matter produced on land and transported by rivers is an important source of CDOM to the coastal oceans.[4-7] However, CDOM undergoes UV-induced transformations, resulting in loss of colour and UV absorbance (referred to as photobleaching).[8-10] Photobleaching of CDOM increases the penetration depth of UV radiation into water bodies and enhances the exposure of aquatic ecosystems to UV radiation. This increased exposure enhances the negative, direct effects of UV on carbon fixation in aquatic systems (see Chapter 4). The effects of CDOM on UV penetration are determined by its chemical composition[11], and the optical properties and the mechanisms of photobleaching of CDOM are being intensively investigated.[8, 10-18]

Carbon Storage and Release

The storage of organic carbon in terrestrial ecosystems is estimated to be approximately 2000-3000 Pg, with about three times as much stored in soil as in vegetation.[19-21] Approximately 38,500 Pg of inorganic carbon are stored in the ocean and sediments, and approximately 750 Pg are stored as living and non-living organic carbon. The effects of changes in solar UV-B on long-term carbon storage remain unclear. However, UV-B influences short-term carbon turnover from plant litter (dead plant material) in terrestrial systems, and dissolved organic matter (DOM) and particulate organic matter (POM) in aquatic systems. The release of carbon can result not only from respiration but also from photodegradation of organic matter or phototransformation into forms more readily available to microbes. The photodegradation of wood is also discussed in Chapter 7.

Global respiration from terrestrial vegetation and from soils are both of the order of 50-60 Pg of carbon per year.[3, 22] The release of carbon differs among sources such as living plant material and dead organic matter and is highly dependent on factors such as temperature. Altered UV-B will influence carbon release from terrestrial ecosystems through changes in the efflux of CO_2 and/or CH_4.[23, 24] However, research over the past four years reinforces the conclusion of the previous UNEP report[1] that the effects of changing UV on carbon release from terrestrial ecosystems are complex and are likely to vary between species and ecosystems.

Carbon release from soils is a function of the activities of soil fauna and micro-organisms. Soil organisms may be vulnerable to UV-B damage (e.g.,[25]) but are rarely exposed to solar radiation in nature. Even so, plant responses to changing UV-B may indirectly affect soil organisms. Attenuation of solar UV-B modifies the communities of fungi and microfauna in the surface layer of *Sphagnum* wetland communities.[26, 27] These changes in soil communities are probably related to UV-induced changes in the quantity or chemistry of dead plant material or root exudates entering the soil, for example attenuation of solar UV-B reduced the leaching of DOC and P from *Sphagnum* moss[27] (Figure 5-2a). UV-B induced changes in leaf litter chemistry may also influence soil fauna, for example earthworms, although responses appear to be rather subtle and species-dependent.[28] Recent studies in the Antarctic[29], temperate grassland[30], and arctic heath[31] have also shown changes in soil microbial community structure in response to UV-B manipulations, rather than the changes in total microbial biomass that were evident in some previous studies.[32]

The effects of changed solar UV on the chemistry of leaf litter have been studied in a range of species. Changes in litter chemistry can occur in two ways: by the effects of UV exposure on decomposing litter (which are called direct effects), or the effects of UV on the chemistry of living tissues before litter-fall (so-called indirect effects). Direct effects occur when dead plant material decomposes on or above the soil surface, when directly exposed to solar UV. Recent data[33] highlights that direct photodegradation can be the dominant process in the decomposition of plant litter in ecosystems with high sunlight and limited rainfall, and that UV-B may account for 50% of such photodegradation (Figure 5-3a). In these dryland ecosystems changes in solar UV resulting either from variation in ozone or elements of climate change such as cloud cover may have substantial effects of carbon cycling. In other terrestrial ecosystems the effects of UV on litter decomposition are more complex. Exclusion of ambient solar UV-B during decomposition accelerated the break down of litter in some species (Figure 5-3b) but had only marginal effects in others (e.g., barley:[34]). Similarly, exclusion of ambient UV-B may increase litter colonization by fungi in some systems[35], but not others.[36] Overall, the direct effects of changing UV-B on litter decomposition appear to depend on species-specific differences in litter chemistry, and on absolute differences in UV dose and weather conditions between different sites.

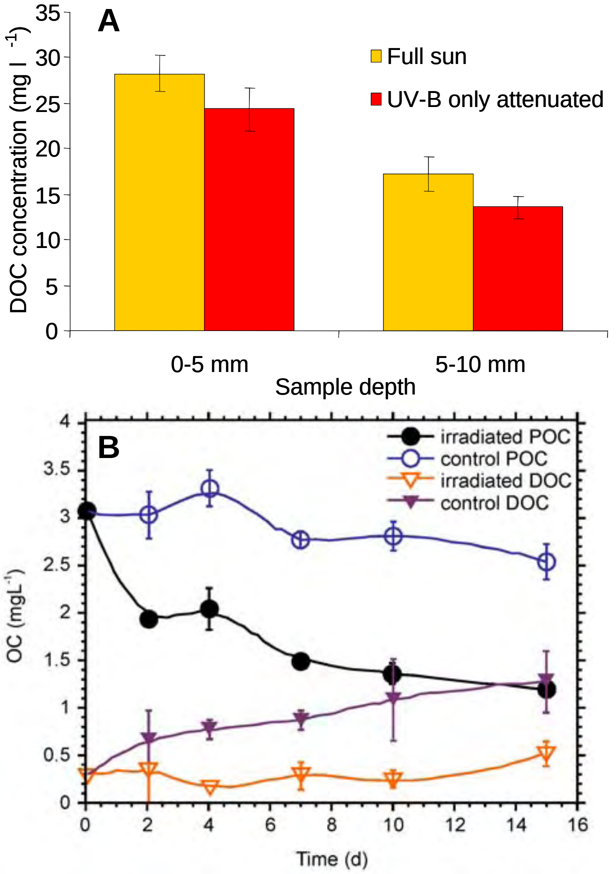

Figure 5-2. Figure 2. UV radiation influences the production of dissolved organic carbon in both terrestrial and aquatic ecosystems. In terrestrial ecosystems, recent evidence shows that UV-B exposure significantly increases the release of DOC from *Sphagnum* moss, the dominant plant in global peatland ecosystems (A, modified from Robson *et al.*[26]). This effect of UV-B was not confined to the surface layers exposed to UV-B, but occurs also in deeper layers (5-10 mm below the surface) where no UV-B penetrates. In aquatic systems, UV-B can result in the release of DOC from particulate organic carbon (POM). For example simulated solar radiation results in the loss of suspended POM and the production of DOC (B, from Mayer *et al.*[37]: data are means of triplicate experiments and the error bars are ± 1 standard deviation). Other studies using light filters showed that the POC to DOC conversions were mainly induced by UV radiation (Figure 2b, Reprinted with permission from Mayer et al.[37], Figure 1, p. 1066, Copyright 2006 by the American Society for Limnology and Oceanography, Inc.). The continual resuspension and exposure of deposited particulates to sunlight in shallow parts of systems such as the Missisippi Delta region provides an opportunity for cumulative photodissolution over time.

The indirect effects of changing UV on litter chemistry and consequent changes in decomposition are species specific. The previous observation that elevated UV-B during plant growth and development accelerated the decomposition of oak leaf litter[37] was shown recently to be associated with reductions in the extractability of some carbohydrate fractions in litter produced under elevated UV-B (Figure 5-3c), which stimulated colonization by some fungal decomposers.[38] In barley UV-B exposure during plant growth and development caused significant changes in litter chemistry, including nitrogen, and this was associated with a significant reduction in the loss of dry weight and nitrogen from litter during decomposition.[34] In the herbaceous plant *Gunnera magellanica* there were persistent differences in the fungal community colonizing litter produced under different UV environments[35], but this was not related to significant changes in litter chemistry or decomposition. In two grasses UV-B exposure during plant growth and development had no effect on litter quality or decomposition.[39]

Increased UV-B significantly modifies the chemistry of secondary metabolites produced by and released from plants (also see Chapter 3). The majority of studies of UV effects on plant chemistry have dealt with leaves, but increased UV may also induce accumulation of certain carbohydrates and UV-absorbing phenolics in the bark of birch saplings.[40] The chemistry of tissues not directly exposed to UV can also be affected, as in the roots of lupin (*Lupinus luteus)* plants grown under increased UV.[41] As with many UV responses, this effect on root chemistry appears to be species specific.[42, 43] Although these studies of UV effects on plant chemistry have not explicitly considered biogeochemical cycles, there is increasing evidence that plant-derived chemicals, especially phenolic compounds, have major consequences for soil processes.[44-46] It is notable that many of the plant-derived phenolic compounds considered to play vital roles in affecting soil processes have also been shown to be influenced by UV exposure.[40, 45, 47-49] Since UV-B effects on such phenolic compounds are specific both to plant species and individual compounds, the observed variation in effects on soil processes might be expected.

Since existing studies of the effects of increased UV-B on decomposition or soil processes have largely focused on individual species, the net effects of changed UV-B acting across areas of vegetation remain unclear. A recent modeling approach to the effects of climate change on decomposition suggests that the principal effect of elevated UV-B will be to increase litter accumulation and so contribute to carbon sequestration.[50] However, the conclusion of the previous assessment[1] that any such effect of UV on terrestrial carbon budgets is likely to be local and determined by species-specific responses in both plants and decomposers has not been changed by recent research.

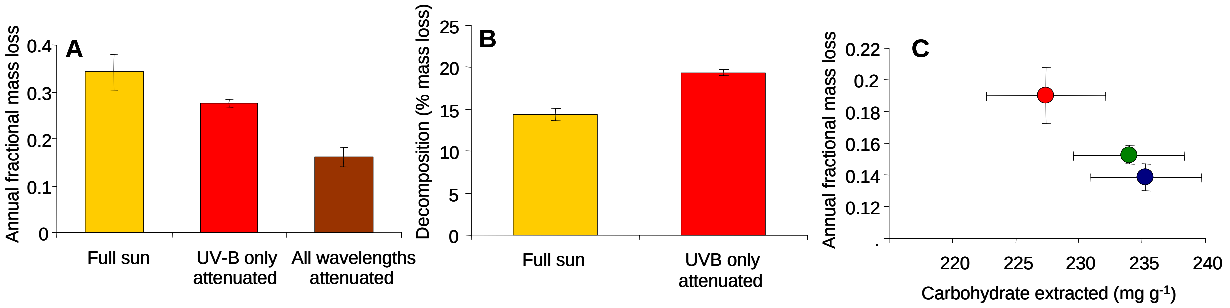

Figure 5-3. The effects of UV-B radiation of the decomposition of dead plant material in terrestrial ecosystems vary with factors such as climate and plant species. In high, light, arid ecosystems the break-down of dead plant material ("litter") may be largely due to photodegradation, and UV-B may accelerate this process. This is clear from a study of decomposition in the Patagonian steppe (A, modified from Austin and Vivanco[33]) where litter was decomposed either under full sunlight, sunlight in which only UV-B had been attenuated, or sunlight with all wavelengths attenuated by 90%. (Data are presented as the annual fractional mass loss obtained from the slope of the regression between ln(organic matter remaining/initial organic matter) against time. Error bars are ± standard error of the mean). In ecosystems less limited by water, the effects of UV-B on litter break-down are more variable, but there are many examples where UV-B suppresses decomposition, as in the herbaceous plant *Gunnera magellanica* grown in Tierra del Fuego (B, modified from Pancotto *et al.*[35]: data are means ± standard error of the mean). UV-B exposure during growth may also influence subsequent decomposition of leaf litter, as in oak (C, modified from McLeod *et al.*[39]) where the annual fractional mass loss is significantly increased in litter produced under elevated UV-B, and this is associated with reductions in the amount of carbohydrate released from this litter. (Data are means ± standard errors).

Carbon dioxide (CO_2) in aquatic systems is produced via microbial respiration, UV-induced mineralization of DOM into inorganic carbon, and/or shifts in the carbonate equilibrium.[1]

Whether photochemical mineralization or microbial respiration is the predominant process largely depends on the source and thus the chemical composition of DOM. A number of studies have shown that algal-derived DOM is more biodegradable and less photoreactive than terrestrial DOM.[5, 51-54] The extent of DOM photomineralization increases with decreasing pH[55] and also depends on the precipitation history.[56] Furthermore, photomineralization is catalyzed by iron[10] (see also the section "Metal Cycling").

Photochemical and microbial decomposition of DOM may interact since DOM is a key nutrient and energy source for consumers, including heterotrophic bacteria and metazooplankton. However, not all chemical forms of DOM are available to these organisms, and UV-induced transformations of DOM can alter its bioavailability.[1] UV-induced transformations of DOM may increase bioavailability of biorecalcitrant DOM and vice versa.[52, 57] The UV-induced increase in DOM bioavailability may enhance microbial respiration of DOM[58] or increase microbial biomass through the microbial loop.[59]

Transfers of carbon and nutrients between the water column and bottom sediments of freshwaters and the ocean are mediated by particles. Solar UV can affect the dynamics of such transfers by photooxidation of particulate organic carbon (POC).[1] UV also can induce the conversion of POC to dissolved organic carbon (DOC)[60](Figure 5-2b).

Nutrient Cycling

Nitrogen and phosphorus can limit productivity in terrestrial and aquatic ecosystems. UV radiation can affect nitrogen cycling in several ways, including changes in the decomposition of N-containing organic matter and through effects on nitrogen fixation. In addition, in aquatic environments UV interactions with inorganic nitrogen species such as nitrate and nitrite are an important source of reactive oxygen species, including the highly reactive hydroxyl radical.

Biologically labile nitrogen compounds such as nitrate, ammonium, and amino acids are rapidly recycled by the biota in aquatic systems, while N-containing substances that have structural features that inhibit assimilation, accumulate in the water column. Interactions of UV radiation and dissolved organic nitrogen (DON) provide a pathway for the conversion of persistent DON to compounds that are more easily assimilated by aquatic microorganisms, such as ammonium.[1] Ammonium photoproduction, i.e. "photoammonification", which is affected by solar UV-B radiation,[61] occurs in both freshwater lakes[62] and in coastal regions such as the Baltic Sea[61], where it can periodically be the largest source of new bioavailable nitrogen in the Baltic Sea.[61]

Solar UV can affect phosphorus cycling by photodegradation of enzymes such as alkaline phosphatase (APase) or release of APase sorbed to humic substances.[1] Additional evidence obtained in Canadian lakes confirmed that exposure to solar UV reduced APase activity.[63] Sorption of APase to clay particles protected the enzyme from UV-induced photodegradation, although enzymatic activity was reduced by sorption.[64]

Nitrogen inputs into soil originate from a number of sources, including atmospheric deposition, decomposition, biological nitrogen fixation and, in agricultural ecosystems, fertilizers.[65] Variation in responses of litter decomposition to changing UV-B affect the release of nitrogen from decaying plant litter.[34, 35, 39] The effects of changing UV on plant-derived phenolic compounds in soils may be especially important for nitrogen transformation processes.[44-46] Biological nitrogen fixation by free living cyanobacteria can be reduced by increased UV-B radiation[1, 66] (see Chapters 3 and 4). Increased UV-B may also inhibit nitrogen fixation by cyanobacteria within lichens[1] but recent data suggest that this does not occur in all species.[67] In recent studies of tropical legumes, elevated UV had no significant effects on symbiotic nitrogen fixation[41-43], in contrast to a number of previous studies.[1]

In terrestrial plants the uptake of nutrients such as phosphate and some forms of nitrogen is often achieved through the mycorrhizal symbiosis between plant roots and fungi. There is evidence that changing UV-B can influence root colonization by mycorrhizal fungi[1], but recent evidence shows that this response varies between sites, even within a single system[31], as well as between species.[30, 68] We are unaware of any studies that have explicitly measured mycorrhizal function rather than colonization.

Metal Cycling

Many trace metals are essential micronutrients for aquatic organisms[69-72], but most of them can be toxic to terrestrial and aquatic organisms, depending on their concentration and chemical speciation.[73-76] Solar UV radiation has significant effects on the chemical speciation of trace metals, for example, iron, manganese, copper, chromium, and mercury (Figure 5-4).

Iron

Iron is an essential micronutrient for phytoplankton and has been shown to co-limit phytoplankton growth in several marine environments.[77-79] The role of iron supply in stimulating phytoplankton blooms in high-nitrate, low-chlorophyll oceanic waters has been demonstrated[80, 81] and its effect on carbon sequestration is being intensively investigated.[82-84] Fe(III) is the most common form of iron in sunlit aquatic systems, but Fe(II) is the key form that determines the availability of iron for uptake by organisms.[85] In oxic, sunlit surface waters the maintenance of low Fe(II) concentrations is a result of the UV-induced reduction of Fe(III), both dissolved and colloidal,[86-89] and Fe(II) oxidation.[90-93] In surface waters of the Southern Ocean the photoreduction was primarily caused by UV-A radiation, although ozone depletion was estimated to increase the UV-B contribution to this photoreduction from 3.5 to 6.2%.[89] UV-induced iron chemistry will interact with large scale inputs of iron, such as dust deposition, that are influenced by climate change.[94] Dust deposition is the major source of iron in some

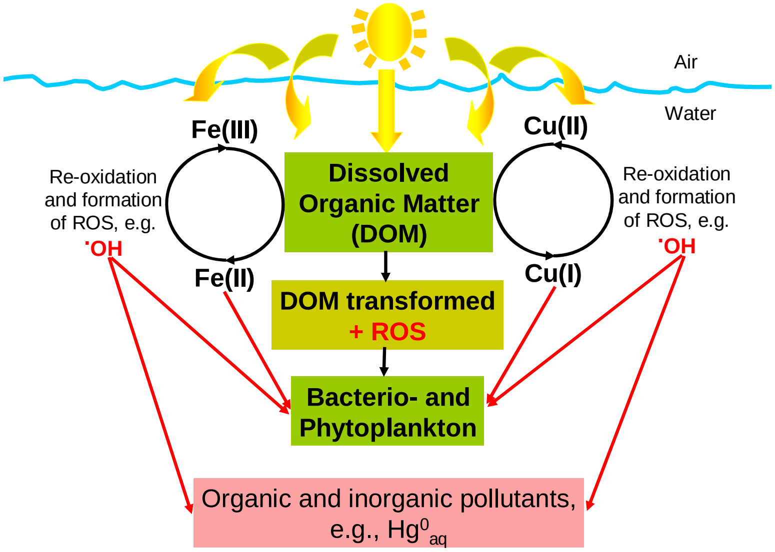

Figure 5-4. UV radiation is a key factor in the chemistry of iron and copper in aquatic systems, including its interactions with dissolved organic matter (DOM) and microorganisms. This schematic represents the UV-induced redox cycling of iron and copper, and the concomitant phototransformation of DOM and production of reactive oxygen species (ROS), for example the highly reactive hydroxyl radical (·OH), that can adversely affect bacterio- and phytoplankton and react with organic and inorganic pollutants, for example dissolved gaseous mercury, Hg_0aq.

remote regions of the ocean and UV exposure can affect the bioavailability of this iron. Enhanced iron bioavailability could affect both phytoplankton abundance and community structure particularly in these regions. Indeed, satellite observations of productivity in remote regions of the Southern Hemisphere correlate with dust deposition.[95] The regions in the Southern Hemisphere where oceanic productivity is strongly correlated with dust deposition (Figure 5-5) are those in which future changes in total ozone, and thus UV-B exposure, are likely to be most dynamic (see Chapter 1). Thus future changes in total ozone over this region could interact with dust deposition to alter the spatial distribution of primary and secondary pelagic production and may place additional stress on already depleted fish and mammal populations (also see Chapter 4).

Copper

The bioavailability and thus toxicity of copper depends on its chemical speciation. It is likely that, by analogy to iron, reduced copper, Cu(I), is an important species with regard to the bioavailability and hence toxicity of copper. As with iron, reduction of Cu(II) to Cu(I) in oxic aquatic systems occurs to a large extent via UV-induced photochemical reactions of Cu(II) complexed by strong organic ligands.[96, 97] In the case of coastal estuaries, these strong Cu ligands are components of humic substances that can be photodegraded. Although solar UV radiation was primarily responsible for the photolysis of these strong Cu ligands, visible radiation also was involved.[98] Up to 25% of total dissolved copper detected in marine surface waters is Cu(I) which is stabilized by inorganic and organic ligands such as chloride and phytoplankton-derived organosulfur compounds.[75, 76] During its long-range transport in atmospheric waters, copper undergoes UV-induced redox cycling so that a large fraction of total copper in rainwater is Cu(I).[97] It was hypothesized that part of the strong Cu-complexing ligands found to be present in these rainwater samples were Cu(I)-complexing ligands that retard Cu(I) oxidation.

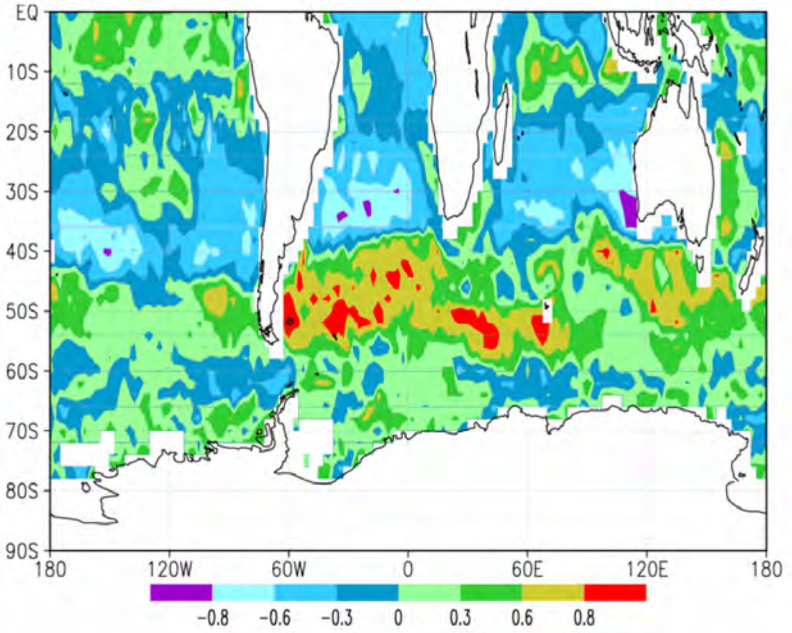

Figure 5-5. The productivity of marine ecosystems is highly correlated with inputs of dust derived from terrestrial ecosystems, which may be changing due to climate change. This map shows correlation coefficient observed between dust deposition and marine productivity as estimated by satellite estimates of chlorophyll.[95] The time series of dust deposition is estimated from a global transport model that has been verified against observations. The high correlations (>0.80) indicate that the input of atmospheric Fe in dust elicits a very quick and discernable response in some regions of the ocean. The strong covariance of dust input and biological activity is occurring in regions where productivity is Fe limited and the magnitude of the dust flux is quite small as this is occurring far from strong dust sources. These regions continue to experienced the greatest changes in stratospheric ozone concentration and, hence, in UV-B radiation (see Chapter 1).

Mercury

UV-induced redox processes also largely determine the bioavailability and thus toxicity of mercury. Mercury is a globally dispersed toxic pollutant that can be transported far from its emission sources.[99] Divalent mercury, Hg(II), can be methylated and bioaccumulated in the aquatic and terrestrial food chain[100] and methylmercury can adversely impact reproduction of wild fishes.[74] Reduction of Hg(II)[101, 102] and oxidation of dissolved gaseous mercury (Hg_0aq)[103-105] are both induced by UV radiation. Especially critical is the UV-induced oxidation of Hg_0aq since this process increases the pool of methylmercury. The photooxidation of Hg_0aq is chiefly mediated by UV radiation in natural brackish waters, and ·OH may be partly responsible for Hg_0aq oxidation in these systems.[103, 104] The photoproduction of methylmercury was dependent on DOM concentration and type in freshwater samples.[105] For example, water from lakes with logged watersheds generated methylmercury when exposed to sunlight whereas water from lakes with low levels of logging in the undisturbed watershed did not. Hence solar UV radiation can have significant and complex impacts on mercury volatilization, solubilization, and methylation.[106]

UV-Enhanced Oxidative Activity of Aquatic Systems

Reactive oxygen species (ROS), important in the processing of natural and anthropogenic compounds, are produced by UV-induced photoreactions of inorganic nitrogen, organic matter and/or metals.[10, 18, 58, 107] The most reactive ROS, ·OH, damages cellular components such as proteins, lipids, and nucleic acids[108] and inhibits bacterial carbon uptake.[57] UV-induced reactions involving iron or copper and hydrogen peroxide (Figure 5-4) can be an important source of ·OH in iron-rich, sunlit surface waters[18, 109] and can enhance the oxidative activity of aquatic systems.

The hydroxyl radical and other ROS also react with natural compounds and man-made chemicals such as pharmaceuticals[110-112], certain herbicides[113] and commercial dyes[114], as well as with naturally-occurring organic matter such as lignin[115] and DOM.[16-18, 58, 116-118] The oxidation of contaminants by ROS, however, is not always beneficial but may increase the toxicity of contaminants as in the case of mercury (see above subsection " Mercury").

Interactions Between UV Radiation and Other Co-Occurring Environmental Changes

Changes in the surface flux of UV will interact with co-occurring changes in climate, land use and atmospheric carbon dioxide all of which have the potential to influence the biogeochemical cycling in terrestrial and aquatic ecosystems. In the future, changes in temperature and rainfall patterns[119], cloud cover, forest fires[120], aquatic biological production, deforested land surfaces and aquatic circulation will interact with themselves and with changing surface UV radiation. These interactions will affect biogeochemical cycles, including carbon cycling, movement of CDOM from land to water, stratification of aquatic ecosystems, and trace gas exchange between the biosphere and atmosphere.

Interactions Involving Carbon Cycling

The effects of environmental change on carbon cycling may interact with those of solar UV radiation via changes in carbon fixation, storage and release, as discussed above. The greatest source of uncertainty in global carbon budgets of terrestrial ecosystems appears to be the tropical regions, where the balance between the high potential carbon fixation of tropical forests is balanced by substantial carbon release due to deforestation and other changes in land use.[2, 20] Since the effects of stratospheric ozone depletion on the UV climate of the tropics are minimal[121, 122], these uncertainties may be of little importance in assessing ecosystem responses to interactions between ozone depletion and climate change in this region. However, it is notable that several reports indicate that the main driver of increased carbon fixation in tropical forests is an increase in total solar radiation driven by changing patterns of clouds.[123, 124] The ecological effects of increased exposure to UV due to decreased clouds are unclear since, unlike stratospheric ozone depletion, variation in cloud cover will cause concurrent changes in both UV and photosynthetic radiation. While many tropical plants are considered tolerant of UV, significant UV responses have recently been reported in a range of tropical plants.[42, 43, 125] Thus, there is potential for a UV contribution to ecosystem responses to altered cloud cover even in the tropics, although this is likely to be species-specific. In addition, changes in aerosols, for example from combustion, affect UV-B radiation more than photosynthetic radiation[122] with unknown ecological consequences.

There is a clearer understanding of large-scale carbon budgets in temperate and high latitude regions, especially for the Northern Hemisphere. Analysis of atmospheric CO_2 trends in the Northern Hemisphere since 1994 provides evidence that warming may be increasing CO_2 fixation in spring, but this effect is offset by reductions in photosynthesis due to increasing summer drought.[126] While remote imaging shows greening at high latitudes over the last two decades[127, 128] and increases in the length of the growing season, such changes do not occur in all high-latitude ecosystems perhaps due to interactions with water supply.[129, 130] Carbon sequestration in high latitude forests due to warming may also be offset by increased respiration from burned forests.[131] Thus, while effects of changes in the growing season of carbon budgets may interact with changes in UV exposure,[1] these interactions remain hard to predict. Especially at high latitudes future climate changes are also likely to contribute strongly to changes in ecosystem distribution[132] (Figure 5-6), and since UV effects on both carbon fixation and release are specific to particular plants or ecosystems (see above and Chapter 3), this may significantly alter regional responses to changing UV climates. Increased temperatures generally increase decomposition rates, but the magnitude of temperature dependence varies substantially between different types of organic matter. While there is clearly a considerable variation in temperature dependence, carbon release from soils, reflecting microbial processes, appears to be more sensitive than that from living plants.[22] There is recent evidence for both soil organic matter[133-136] and leaf litters[137] that the temperature sensitivity of decomposition increases with decreasing litter quality. It is also notable that increased temperature may particularly favour those soil microbes best able to decompose recalcitrant organic material.[138] These effects of warming might interact with those caused by UV-B (see above), and this may be particularly pertinent for "high latitude" ecosystems where future changes are likely to be the most dynamic in the Earth system (see Chapter 1). Carbon release may also be influenced by many co-occurring environmental changes. Recent research shows that the effects of elevated CO_2 on carbon release from soil are complex.[139-142] The mechanisms involved include increased root growth[143, 144], altered litter quality[145], and changes in the soil microbial community[146-149], and so may interact with comparable responses caused by changing UV-B (see above). Interactions between UV and drought may occur through similar mechanisms, since drought substantially inhibits soil

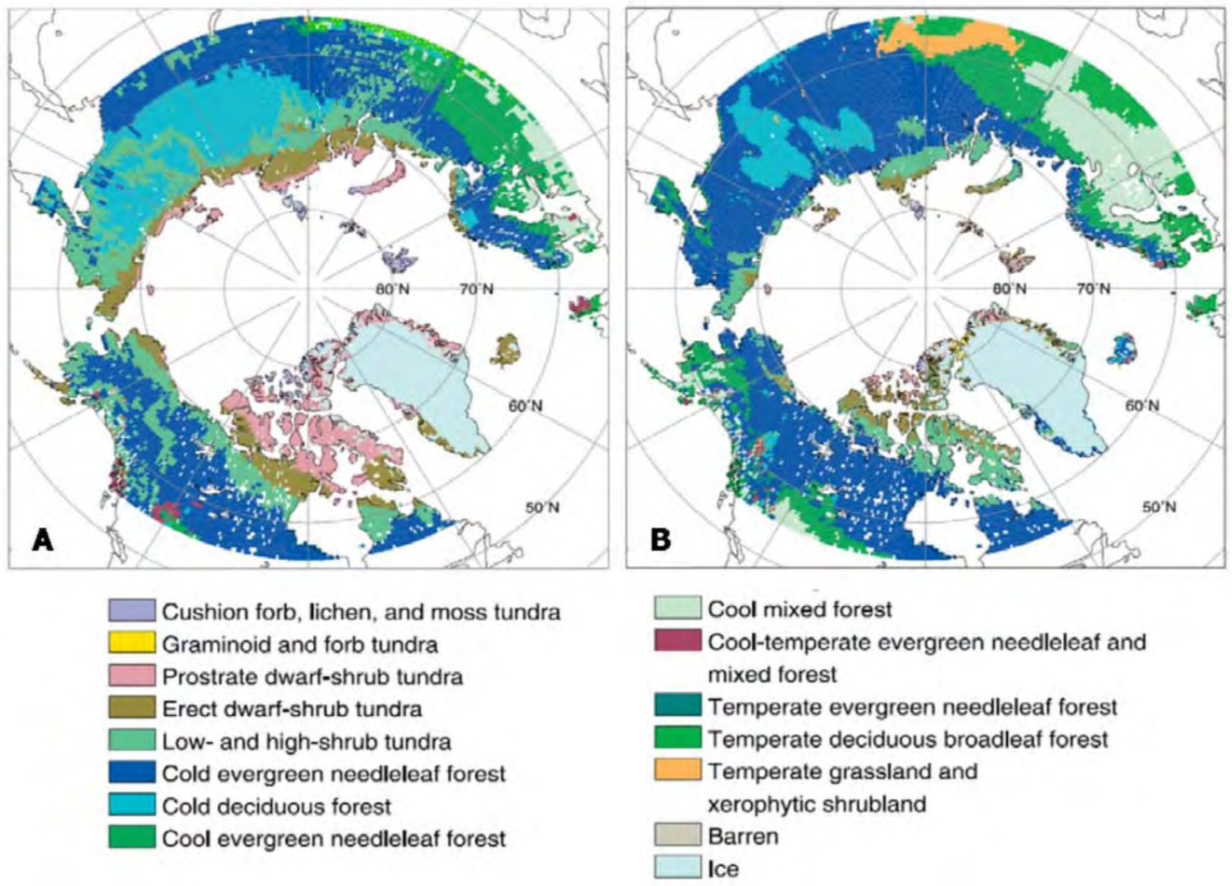

Figure 5-6. Climate change is expected to result in substantial shifts in the distribution of major ecosystems. Present and projected future geographic distributions of vegetation types north of 55ºN are illustrated here: A, Present-day potential natural vegetation; B, The potential vegetation driven by the mean climate of the decade 2090–2100 simulated by the HADCM2-SUL coupled model, using the IS92a scenario. The future vegetation types were predicted from climatology using a set of plant functional types embedded in the biogeochemistry-biogeography model BIOME4.[132]

respiration[150-152] and even mild water deficits can reduce the carbon flow from roots to the soil microbial community[153] and increase the abundance of phenolic degrading bacteria.[154]

Effects of Climate Change on Carbon Inputs from Terrestrial to Aquatic Systems and Implications for Aquatic Ecosystems

Deforestation and changes in land use have substantial effects on carbon losses from soils via leaching or run-off into water bodies where it is an important source of CDOM and DOM. Shifts in soil moisture content and related changes in oxygen content affect the microbial production of soil humic substances and thus can alter inputs of this important source of CDOM in freshwaters. UV-induced transformation of CDOM differs between aquatic and terrestrially derived DOM due to differences in composition, particularly phenolic content. Given the well-defined effects of elevated UV-B on the phenolic chemistry of litter and root exudates, it is possible that the composition of DOM leaching from or running-off terrestrial systems could be altered, with consequent effects in aquatic systems. That mechanism remains unexplored, but there are many ways by which climate change may enhance UV-induced CDOM bleaching. Warming affects the composition of CDOM produced by microbial decomposition of seagrass, rendering CDOM more susceptible to UV-induced bleaching.[155]

Climate-related changes in continental hydrology can alter the production and transport of UV-absorbing substances from land to the ocean. These changes involve alterations in discharge rates from rivers and the chemical content of the water transported and mixed into the ocean. Important factors influencing these changes include climate-related changes in the frequency and intensity of precipitation, extent of land use change, dams and anthropogenic and natural chemical inputs to the river basins. For example, in Arctic systems boreal wetlands are the major source of DOM in streams, rivers, lakes and the coastal ocean. Changes in temperature, precipitation and CO_2 affect the concentration and discharge of DOM from terrestrial ecosystems.[156, 157] UV-B radiation induces the degradation of CDOM in aquatic system and is especially significant at high latitudes.[1, 55] Degradation of terrestrially-derived DOM in the Arctic Ocean limits its movement into the deep ocean.[6] The effect of past climate changes on aquatic-terrestrial interactions also has been demonstrated.[158] Paleoecological techniques were used to demonstrate that periods of deforestation over the past 10,000 years were associated with rapid declines in the inputs of UV-absorbing CDOM, which in turn caused up to a ten-fold reduction in algal biomass. Climate-related changes in ice and snow cover will result in a greater area of the high latitude biosphere being exposed directly to UV-B (also see Figure 5-6).

Climate Change and Mixing in Aquatic Systems

UV-induced bleaching of CDOM in the surface layers of aquatic systems is intensified by stratification.[159] Climate models predict that global warming will result in reduced vertical mixing and increased stratification in the upper ocean.[160-162] Thus, the effects of elevated UV-B may interact with the effects of climate change on stratification, and the quantity and chemical composition of inputs from terrestrial systems. Observations in Europe during the 2003 heat-wave demonstrated that warming can enhance lake stratification.[163] Climate-related changes in atmospheric circulation and related changes in vertical mixing dynamics in the ocean and freshwaters can also affect UV exposure of aquatic organisms. For example, in the case of phytoplankton in the ocean, increased variability in the deep chlorophyll maximum[164] could result in more variable UV exposure, with possible consequences for primary production. Moreover, UV-B damage to microorganisms is strongly affected by changes in climate-induced mixing of the upper layers of aquatic environments and thus will affect carbon and nutrient cycling and the production and consumption of trace gases.

Satellite imagery has provided a first view of the global distribution of CDOM that controls the penetration of UV-B into the sea.[165] Photoreactions of CDOM and changes in ocean circulation patterns related to El Niño events strongly influence CDOM distributions. Remote sensing techniques also facilitate the global high resolution analysis of aquatic UV impacts on marine biogeochemical cycles. Relationships have been developed between remotely sensed ocean colour and UV attenuation in coastal regions of the ocean. The relationships were applied to determine changes in UV penetration into the Mid-Atlantic and South-Atlantic Bight near the eastern coast of the U.S.A.[166] These recent studies highlight the possibility of monitoring from space the interactions of climate-related mixing trends with the chemical compounds that are produced from UV interactions.

Interactions between Ozone Depletion and Climate Change Mediated Via Changes in Trace Gases Abundance and Chemistry

The budgets of trace gases such as methane, halocarbons, nitrogen oxides (NO_x) and carbon monoxide may be influenced by changing solar UV radiation, and through their effects on atmospheric chemistry some may also influence ozone (Chapter 6). Since climate change may influence the budgets of these trace gases, they are a key link between ozone depletion and other elements of environment change. There is increasing understanding of the responses of specific trace gases to climate change. As discussed in our previous report[1], at high latitudes ice and snow can play a key role in the budgets of many trace gases. Climate change is having major effects on ice and snow cover, and hence on the budgets of trace gases such as bromoform, NOx, alkenes, formaldehyde and CO.[1] These reactions have consequences for concentrations of tropospheric ozone at high latitudes (see Chapters 1 and 6).

New, high-resolution models of oceanic ecosystems have been developed which provide estimates of phytoplankton and trace gas distributions with high spatial resolution globally. Figure 5-7 illustrates the use of such Earth system models to estimate global distributions of surface ocean DMS based on a global ocean biogeochemistry physical circulation model. The ability to use high performance computing that enables future biogeochemical states to be estimated will allow assessments of UV stress on ocean ecosystems over the next 100 years.[167] The use of this type of global Earth system models can provide future estimates of O_3 and UV levels that contribute to trace gas exchange between the surface ocean and the atmosphere.

Methane, carbon monoxide and volatile organic compounds

Enhanced UV-B can reduce emissions of methane from peatland ecosystems and paddy fields, and this is partly explained by morphological changes in plant structure.[23] Emissions of methane from wetland ecosystems are highly dependent on the plant species present[23, 24, 168] and so could be affected by changes in ecosystem distribution (Figure 5-6). Recent research has demonstrated that plants themselves can be methane sources, even under aerobic conditions[169], although the contribution of such emissions to global methane budgets remains unclear. Changes in light and temperature affect plant production of methane but the role of UV-B is unknown. Thus, changes in UV might alter methane budgets through effects on the balance of different plant species, or on plant morphology. In addition, the soil can be a net sink for methane in some terrestrial ecosystems.[170, 171]

Other volatile organic compounds (VOCs) emitted from terrestrial vegetation (e.g., isoprene) are indirect sources of CO_2[172] and are important in the chemistry of the troposphere, which in turn may influence the UV climate (see Chapters 1 and 6). The production of VOCs is sensitive to elements of climate such as increased CO_2 concentration, drought and warming.[173, 174]

There are substantial emissions of carbon monoxide from both terrestrial and marine ecosystems.[1] In terrestrial ecosystems major peaks of CO are associated with periods of large-scale forest burning.[175, 176] CO emissions from terrestrial ecosystems are partly caused by photodegradation, in which solar UV plays a major role.[1, 177, 178] This is consistent with the observation that increases in CO emissions following burning are exacerbated by sunlight[177] and demonstrates the potential for interactions between burning and changing UV on CO emissions.

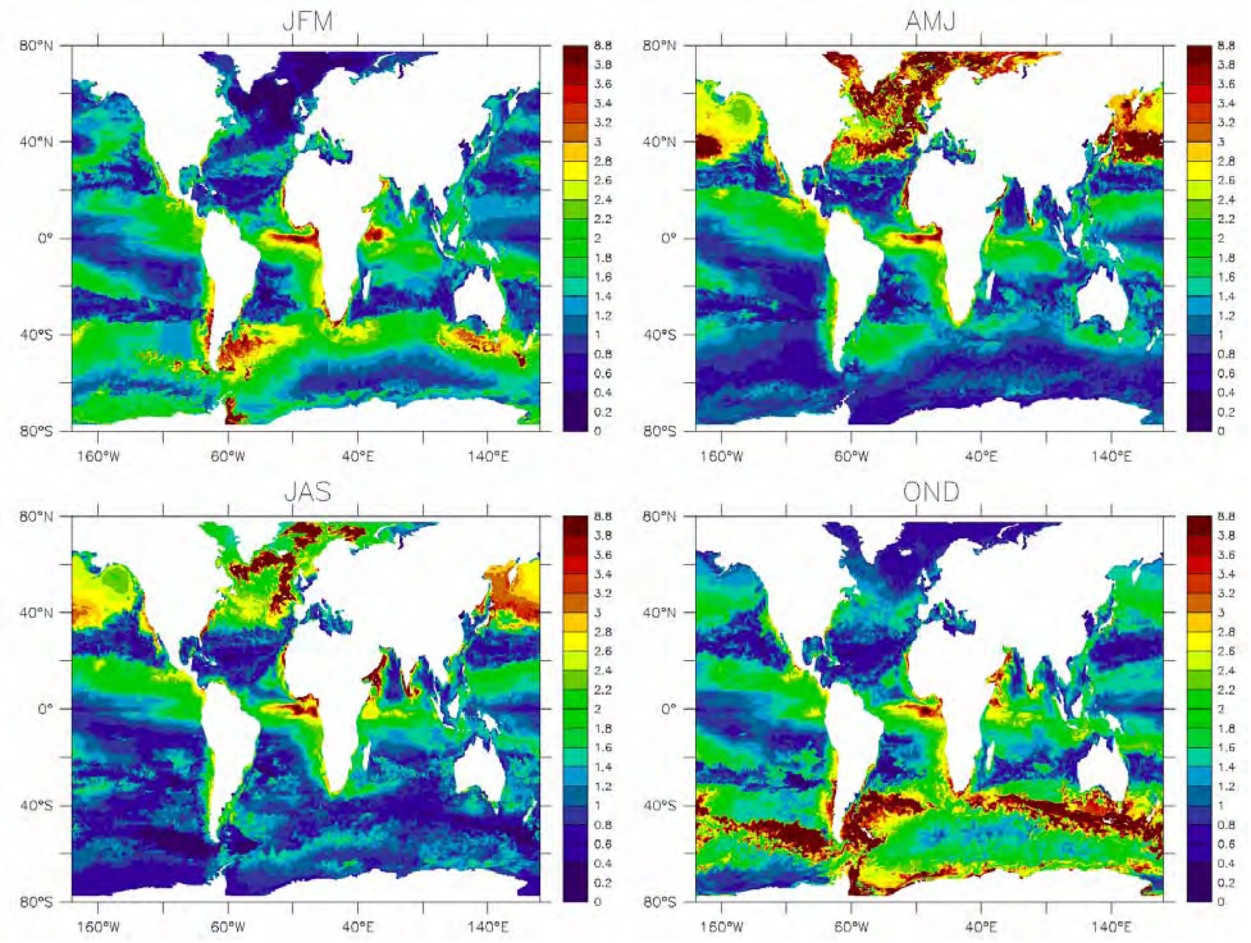

Figure 5-7. Dimethylsulfide plays a substantial role in the balance between incoming and outgoing radiation in the marine atmosphere. This figure shows the global distribution of the flux of DMS from the ocean to the atmosphere, based on a model created by the use of a detailed ecosystem model of oceanic nutrients, phytoplankton and DMS production.[167] As climate change occurs and ocean circulation changes this will alter the surface ocean distributions of DMS and change the exposure to surface UV-B. This is an example of how climate change alters the exposure of climate reactive chemicals to UV-B radiation that is dependent on atmospheric ozone distributions.

Halogen-Containing Compounds

Climate change and UV-B influence the budget of halogen-containing compounds that alter ozone chemistry in the atmosphere (see Chapters 1 and 6). The generation of halogen atoms in atmospheric aerosols can be driven by processes involving UV radiation in the marine boundary layer. Emissions of halomethanes related to climate change may interact with the flux of UV-B to the Earth's surface to modulate trends in atmospheric ozone concentrations.

Terrestrial ecosystems may be sources or sinks of methyl halides, the balance depending on a range of environmental variables.[179-182] Increased temperatures strongly influence emissions of methyl bromide and methyl iodide from plants[180, 182-184], but nothing is known about direct UV effects on these emissions. The observation that ectomycorrhizal fungi are significant sources of methyl halides[181] may be pertinent since increasing UV can affect the activity of these fungi in at least some systems.[185] Agricultural ecosystems remain a significant source of methyl bromide due to its continued use as a horticultural fumigant. The phase-out of methyl bromide fumigant has been constrained by the perceived lack of alternatives for the control of major soil-borne pests and pathogens, but recent research is pointing to economically viable alternatives, both chemical and biological.[186-189] Recent observations indicate that atmospheric methyl bromide concentrations are decreasing at a rate of 2.5 – 3.0 % per year.[190] However, because biogenic emissions of methyl bromide from terrestrial ecosystems generally respond positively to increased temperature, future global warming may change the current rate of decline of methyl bromide concentrations in the atmosphere.

Marine systems also have significant influences on concentrations of halogenated compounds in the atmosphere.[1] Photoreactions involving UV can degrade brominated and iodinated methanes in water[191, 192] and halocarbons also are hydrolyzed in the ocean.[1] Halogen chemistry as related to oceanic releases of organic and inorganic bromine and chlorine has impacts on atmospheric ozone.[193, 194] The production of bromocarbons by marine algae in tropical surface waters leads to significant emissions to the atmosphere.[193, 194] Since these emissions of halogen containing compounds occur in the same region as significant deep oceanic convection, there is evidence that this oceanic source of bromine influences tropospheric ozone concentrations. Photolysis of bromocarbons and reactions with ·OH create BrO that interacts with the ozone cycle. This is an example of where the physical climate system, through atmospheric dynamics and circulation, has feedbacks and interactions with the atmospheric chemical systems that influence ozone concentrations and surface UV-B fluxes (also see Chapter 6).

Sulfur Gases

Sulfur emissions from the ocean are affected by interactions between UV-B radiation and climate change. Of particular interest are dimethylsulfide (DMS) and carbonyl sulfide (COS). DMS emissions influence the balance between incoming and outgoing radiation in the marine atmosphere. Oceanic emissions of DMS produce particulates (i.e., sulfate aerosols) that directly and indirectly have a cooling effect on the marine atmosphere. Upper ocean mixing dynamics and the depth of the mixed layer can alter the effects of solar UV radiation on the biological production of DMS.[1] Several new studies have shown that UV and climate changes strongly influence sea to air exchange of DMS by interacting with biological and photochemical sinks of DMS in the upper ocean. Loss of DMS induced by UV-B is an important determinant of its concentrations in the surface ocean.[195-198] However, the effects of enhanced UV-B on DMS emissions are complex and can vary from one ocean region to another.[199-201] The results indicate that the influence of DMS-related atmospheric sulfate particles is modulated by the levels of surface UV radiation. As discussed previously UV may play a role in the production of carbonyl sulfide, the most concentrated sulfur gas in the troposphere.[1] New research has clarified this role.[202]

Nitrogen containing compounds

Emissions of nitrogen oxides (NOx) can participate in tropospheric reactions that affect air quality (see Chapter 6). One new finding has indicated that UV radiation induces NOx emissions from terrestrial plants.[203] NOx emissions can also be affected by elements of climate change[204] and especially by management practices in agroecosystems.[205, 206]

Concluding Remarks

Since our last report in 2002 there has been increasing international recognition of the interactions and feedbacks between climate change and surface UV radiation, especially in relation to biogeochemical cycles. It is in the nature of many of these interactions that they act over medium-long time scales and as a result, understanding of the consequences remains limited.

It is clear that climate related changes in the input of organic matter to water bodies influences the penetration of UV in to aquatic ecosystems. However, the role of such changes in specific regions, especially at high latitudes, remains uncertain.

The following have been identified as important areas of uncertainty:

- The interactions between trace gas generation and UV-B exposure.
- The role of UV-B radiation in the formation of bio-available metal species in aquatic systems.
- The link between the chemical composition and the bio- and photo- reactivity of DOM.
- The interactions of climate change induced ecosystem changes with changing surface UV-B radiation.
- The effects of the changing surface UV-B environment on future trends in climate and atmospheric chemistry.
- The role of UV-B in controlling biogeochemical cycling in terrestrial ecosystems, especially in the context of changing patterns of drought and cloud-cover resulting from climate change.
- The influence of climate-induced changes in aquatic mixing and stratification and continental runoff on biogeochemical cycling in freshwaters and the ocean.

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Chapter 6. Changes in tropospheric composition and air quality due to stratospheric ozone depletion and climate change

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Summary

It is well-understood that reductions in air quality play a significant role in both environmental and human health. Interactions between ozone depletion and global climate change will significantly alter atmospheric chemistry which, in turn, will cause changes in concentrations of natural and human-made gasses and aerosols. Models predict that tropospheric ozone near the surface will increase globally by up to 10 to 30 ppbv (33 to 100% increase) during the period 2000 to 2100. With the increase in the amount of the stratospheric ozone, increased transport from the stratosphere to the troposphere will result in different responses in polluted and unpolluted areas. In contrast, global changes in tropospheric hydroxyl radical (OH) are not predicted to be large, except where influenced by the presence of oxidizable organic matter, such as from large-scale forest fires. Recent measurements in a relatively clean location over 5 years showed that OH concentrations can be predicted by the intensity of solar ultraviolet radiation. If this relationship is confirmed by further observations, this approach could be used to simplify assessments of air quality. Analysis of surface-level ozone observations in Antarctica suggests that there has been a significant change in the chemistry of the boundary layer of the atmosphere in this region as a result of stratospheric ozone depletion. The oxidation potential of the Antarctic boundary layer is estimated to be greater now than before the development of the ozone hole.

Recent modeling studies have suggested that iodine and iodine-containing substances from natural sources, such as the ocean, may increase stratospheric ozone depletion significantly in polar regions during spring. Given the uncertainty of the fate of iodine in the stratosphere, the results may also be relevant for stratospheric ozone depletion and measurements of the influence of these substances on ozone depletion should be considered in the future.

In agreement with known usage and atmospheric loss processes, tropospheric concentrations of HFC-134a, the main human-made source of trifluoroacetic acid (TFA), is increasing rapidly. As HFC-134a is a potent greenhouse gas; this increasing concentration has implications for climate change. However, the risks to humans and the environment from substances, such as TFA, produced by atmospheric degradation of hydrochlorofluorocarbons (HCFCs) and hydrofluorocarbons (HFCs) are considered minimal. Perfluoropolyethers, commonly used as industrial heat transfer fluids and proposed as chloro-hydrofluorocarbon (CHFC) substitutes, show great stability to chemical degradation in the atmosphere. These substances have been suggested as substitutes for CHFCs but, as they are very persistent in the atmosphere, they may be important contributors to global warming. It is not known whether these substances will

contribute significantly to global warming and its interaction with ozone depletion but they should be considered for further evaluation.

Introduction

Reductions in air quality (from the presence of pollutants in the atmosphere) play a significant role in both environmental and human health. Poor air quality can lead to many adverse outcomes such as acid rain and respiratory disease. In the context of this assessment, it is recognized that increases in concentrations of tropospheric ozone and harmful substances generated from ozone have been shown to have significant impacts on human health as well as in the environment, particularly on plants. These effects have been widely reported and reviewed[1-7] and are not discussed further here. The quality of the air depends on a wide range of factors, including how rapidly chemicals are released and the reactions these substances undergo once they are released into the atmosphere. Solar UV-B radiation (280 – 315 nm) provides the energy for many of the chemical transformations that occur in the atmosphere. For example, the energy provided causes photolysis of a number of important atmospheric trace gases, such as sulfur dioxide (SO_2), formaldehyde (HCHO), and ozone (O_3). These processes will be altered by anything that changes the amount of UV-B radiation, including the elevation of the sun, clouds, and attenuation by some air pollutants.

The release of Ozone Depleting Substances (ODS), when transported to the stratosphere, reduces the amount of ozone. Decreases in stratospheric ozone lead to enhanced UV-B radiation in the lower atmosphere (troposphere), increasing the rate of the photolytic processes.[8, 9] As a result, there is a direct link between stratospheric ozone depletion and air quality.

There are other factors that change the reactive chemistry of the atmosphere, including increased air pollution and the emissions of climatically important greenhouse gases. Changes in climate can also dramatically change the chemistry of the atmosphere. Higher temperatures can lead to increases in the rates of chemical reactions, in the amount of water vapor present, and thus OH production[10] as well as enhance emissions of volatile organic compounds such as isoprene from biological sources.[11] All of these factors then potentially interact in determining the actual atmospheric condition.[12, 13] Thus, while change in atmospheric composition and circulation are observed, assigning cause and effect requires careful assessment.

The replacements for the original ozone depleting chemicals (chlorofluorocarbons (CFCs)), such as the hydrochlorofluorocarbons (HCFCs) and the hydrofluorocarbons HFCs, decompose primarily in the lower atmosphere. This decomposition can produce chemicals that impact air quality.[8]

Here we present a summary of recent work on understanding the impacts of ozone depletion, ozone depleting chemicals and their replacements on atmospheric composition and how these may interact with climate change. This is an update of the information in the previous report.[8]

Stratosphere – Troposphere Exchange

One direct impact of stratospheric ozone depletion is a potential reduction in the amount of ozone transported into the troposphere (Stratosphere Troposphere Exchange, STE). It has been estimated that there has been an approximate 30% reduction in the amount of ozone transported from the stratosphere to the troposphere due to stratospheric ozone depletion.[14] While there is agreement that ozone concentrations in the upper troposphere are very sensitive to change in stratospheric ozone, there remains a divergence of opinion on the sensitivity of ground-level ozone. Some calculations have predicted that up to 40% of ground-level ozone is due to transport of ozone from the stratosphere, depending on season and location,[15, 16] but other estimates are a factor of two lower.[14] All three models estimate that the contribution of vertically transported ozone to ground-level ozone concentrations is smallest in summer compared with other season in both hemispheres.

The mixing of stratospheric air (and hence ozone) into the troposphere is critically dependent on atmospheric circulation. A modeling study of the chemistry-climate interactions showed that the rate of ozone transport into the troposphere is affected by El Niño Southern Oscillation (ENSO) events.[13] An analysis of satellite ozone measurements also detected a dependence on ENSO, although they suggest it is due to the impact of circulation changes on *in situ* production.[17] Climatic variability can therefore mask changes in the contribution of stratospheric ozone to the troposphere due to stratospheric ozone depletion.

The situation is further complicated as stratospheric ozone depletion appears to explain most of the cooling observed in the lower stratosphere over the last two decades.[18] Such a cooling will modify STE, although it is not clear that this has been explicitly included in most current atmospheric models. We can conclude, however, that stratospheric ozone depletion will have a significant but small impact on tropospheric ozone amounts to date due to STE.

Significant longer-term increases in the amount of ozone brought into the troposphere from the stratosphere have been predicted due to climate change (80% by 2100).[19] This calculation ignored the increase in stratospheric ozone, which would increase the stratospheric-tropospheric transport further. Such changes in the upper troposphere are not directly convertible into ground-level concentrations due to possible changes in chemical processes in the troposphere. However, predictions indicate that ground-level ozone is likely to increase significantly in the next century,[14, 15] a trend enhanced by increases in stratospheric ozone. The predictions of these models could be verified against measured concentrations. However, the number of sites with long-term temporal and spatial measurements is few. This is an obvious data gap that could be addressed with collection of additional monitoring information.

Atmospheric photolysis

There has been ongoing work studying the fundamental atmospheric processes that are driven by solar radiation and, in particular, by UV-B radiation. Syntheses of data (photolysis cross sections and reaction rates) have recently been produced.[20-22] This section summarizes some of the key recent outcomes of this work.

Ozone in the troposphere. Ozone in the lower atmosphere plays a number of key roles. Due to its adverse impact upon both human and environmental health in many regions (e.g., European Union[2]), it is used as a key indicator of air quality.

Photolysis of ozone in the lower atmosphere is the primary source of the hydroxyl radical (OH) in unpolluted, humid environments, and OH initiates the removal process for most organic chemicals in the atmosphere, including methane and CO. It is estimated that OH initiates the destruction of 3 700 million tonnes of trace gases each year, including many gases involved in ozone depletion, the greenhouse effect, and urban air pollution.[23] These reactions lead to the formation of products which are subsequently taken up by cloud droplets and precipitation and washed out of the atmosphere. OH therefore plays a dominant role in the ability of the atmosphere to “cleanse” itself.

The formation of OH radical in clean environments is described by the following two reactions:

$$O_3 + h\nu(\lambda < 340nm) \rightarrow O_2 + O(^1\mathrm{D}) \tag{1}$$

$$O(^1\mathrm{D}) + H_2O \rightarrow 2OH \tag{2}$$

The key ozone photolysis product for the formation of OH is the $O(^1D)$ atom. $O(^1D)$ is an oxygen atom that has sufficient energy to react with H_2O. Another possible product of light absorption by ozone is the less energetic $O(^3P)$ oxygen atom which has insufficient energy to react with water (reaction (2)) and primarily reacts with oxygen again to form ozone. The chemical reactions have been discussed in more detail elsewhere.[9]

The production rate of $O(^1D)$ depends critically on the amount of UV radiation which, in turn, is dependent upon stratospheric ozone. As stratospheric ozone depletion increases solar radiation in the critical wavelength region, the loss of stratospheric ozone would be expected to lead to an increase in tropospheric $O(^1D)$, if nothing else changed. The total rate of production of $O(^1D)$ is determined by the concentration of ozone and the photodissociation constant J, which is given by

$$J = \int F(\lambda).\sigma(\lambda,T).\phi(\lambda,T)d\lambda\,, \tag{6.1}$$

where the integral is over wavelength (λ) at the temperature T. Here $F(\lambda)$ is the actinic flux, a measure of the amount of solar radiation available for initiating the chemical reaction, $\sigma(\lambda,T)$ is the strength of ozone absorption (cross section) and $\phi(\lambda,T)$ is the efficiency (quantum yield) of $O(^1D)$ production. The wavelength dependence of these is shown in Figure 6-1. With decreasing wavelength, the actinic flux ($F(\lambda)$) of solar radiation decreases dramatically due to the absorption by (mainly stratospheric) ozone, but the efficiency of production increases (given by the product $\sigma(\lambda,T)\phi(\lambda,T)$). This results in a small range of wavelengths (primarily in the UV-B region) where the maximum production of $O(^1D)$ occurs. The wavelength of maximum $O(^1D)$ production shifts depending on the amount of stratospheric ozone, the elevation of the sun above the horizon, and environmental parameters such as temperature.

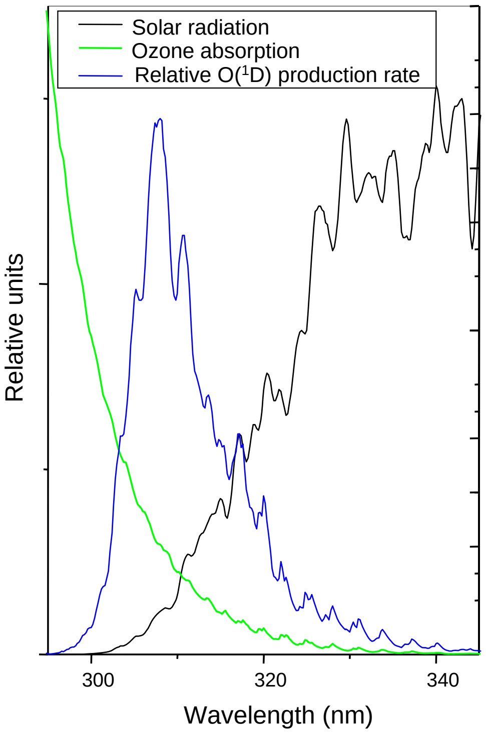
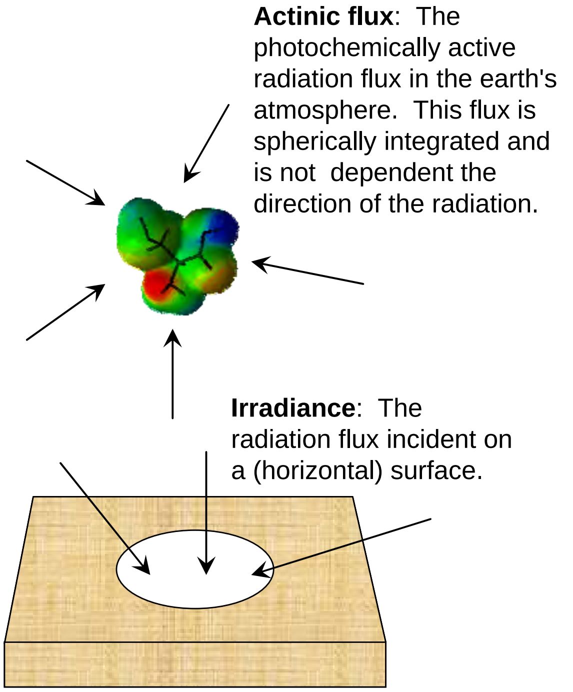


Figure 6-1. Production rate of $O(^1D)$ as a function of wavelength with a solar zenith angle of 60°

There have been significant advances in the determination of the actinic flux (see Chapter 1), which is more formally defined as the amount of solar radiation available at a point in the atmosphere. There are instruments designed to measure actinic flux directly.[24, 25] However, most measurements are of irradiance (the solar energy falling on a horizontal surface – see Figure 6-2). Methods have now been developed for converting irradiance to actinic flux.[26-31] The uncertainty in irradiance-derived actinic flux for $J(O^1D)$ is greater than for directly measured values. The uncertainty in the conversion of irradiance into actinic flux depends on the solar zenith angle, wavelength, and ambient conditions, but can be as low as 7% (95% confidence).[27]

The quantum yield ($\phi(\lambda, T)$) for production of $O(^1D)$ in the wavelength region around 308 nm is crucial. First, this is the wavelength of maximum production. Secondly, many of the estimates of the quantum yield at other wavelengths have been made relative to the quantum yield at 308 nm. Recent work has reduced the uncertainty in the quantum yield at 308 nm (298K = 25ºC) by nearly a factor of two.[32] These new values agree closely with previous best estimates based on the average of various laboratory measurements.[20]

It is now realized that the quantum yield for $O(^1D)$ production is non-zero up to at least 340 nm. While at these longer wavelengths the product $\sigma(\lambda, T)\phi(\lambda, T)$ is small (Figure 6-1), this longer wavelength dependence does reduce the dependence of $O(^1D)$ on the amount of stratospheric ozone.[20, 22] This has resulted in very good agreement between chemical and spectroradiometric measurements of the photolysis rate.[33] Measurements and models of UV actinic flux at the surface now show good agreement.[34] Similarly, measurements of OH and chemical model predictions of OH based on UV radiation measurements now normally agree to within measurement accuracy in remote, clean observation sites.[35, 36] As discussed in Chapter 1, the impact of clouds on actinic flux can be as large.[37] With such large variations, any future changes in cloud amount due to climate change could significantly change photochemically induced atmospheric processes.


Actinic flux: The photochemically active radiation flux in the earth's atmosphere. This flux is spherically integrated and is not dependent the direction of the radiation.

Irradiance: The radiation flux incident on a (horizontal) surface.

Figure 6-2. Two commonly used measures of the “intensity” of solar radiation from the sun. Both have units of $W \cdot m^{-2}$

In air substantially affected by land, other sources of OH have now been recognized as important, such as the photolysis of nitrous acid (HONO) (predominantly by UV-A radiation) in both urban environments[38] and in forests.[39] The wide variety of compounds present and variability of the atmospheric composition in continental air makes modeling assessments, like those described above for clean remote sites, very difficult.

Observed changes in OH. The OH radical has a very short atmospheric lifetime and is present in very small amounts, making direct detection of long term trends impossible. Two alternative methods have been used to determine global trends in OH indirectly.

The main method used for OH-trend detection involves interpretation of the long-term record of methylchloroform ($Cl_3C\text{-}CH_3$; 1,1,1-trichloroethane). Its only source is anthropogenic and the dominant sink is reaction with OH. With a good knowledge of the source, it is possible to infer the removal rate, and hence the amount of OH. Original estimates based on this technique suggested that there was little change in OH since 1980.[40] More recent analysis suggested substantial changes in the amount of OH occurred during the 1990s.[41] The issue of unaccounted emissions of methylchloroform has been raised. The magnitude of methylchloroform emissions from landfills has been debated as a result of measurements in Europe[42, 43] and the US.[44, 45] Consideration has also been given to the magnitude of ocean exchange,[46] which illustrates how difficult it is to account for the complexity of the atmosphere.[47] The most recent assessment, including a consideration of these factors, indicates that OH concentrations in 2003 are very similar to those of 1978, with the major alterations in the global OH concentration being driven by wildfires and climatic variations.[23] Global increases in the OH concentration due to stratospheric ozone depletion[48, 49] are therefore masked by other factors or are smaller than the uncertainties of the measurements themselves.

A second method of estimating OH has recently been developed using ^{14}CO.[50] No significant trend in OH for the Southern Hemisphere in the 1990s could be detected. Unfortunately, the data do not extend back far enough in time to be especially sensitive to changes induced by stratospheric ozone depletion.

A five-year study of OH at a European mountain site[51] found no detectable long term trend in OH concentration, and highlighted that, in this somewhat polluted site, there were clearly factors that were affecting OH concentration that were not part of existing atmospheric chemical models.[51, 52] Rohrer and Berresheim[51] concluded that OH concentration is essentially proportional to $J(O^1D)$ and that scaling of OH by $J(O^1D)$, which depends on solar UV radiation, eliminated most of the diurnal and seasonal variation, and transformed OH into a parameter with significantly reduced variability, provided that factors dependent on local conditions are considered.[51] Rohrer and Berresheim[51] proposed that regional or even global OH distributions could be characterized by a simple set of coefficients for timescales on the order of months or even years and suggested that this approach may be used to define an ‘OH index’ that characterizes the oxidation efficiency of the troposphere in different chemical regimes. This would establish a direct link between stratospheric ozone concentrations and OH concentrations on regional scales. An overarching issue is that most of these models are based on measured data from a limited number of locations, such as in Europe, Japan, North America, and over the Pacific. The troposphere still remains largely under-sampled with respect to OH measurements from the tropics (rain forests) or very large cities such as those in East Asia, key areas for the understanding of how climate change and air quality will influence each other in the near future.[53]

Global numerical models of OH in the atmosphere have advanced significantly. Three dimensional models that include the important known atmospheric chemistry processes[54] are now being augmented with models that include atmospheric transport.[55] Such models will be useful in assessing the impacts of climate change on OH concentrations. One such study has been carried out for the United States, which predicts an increase in OH of between 10 and 15%, and significant increases in ozone in the eastern states.[56]

Observed changes in tropospheric ozone. Hydroxyl radicals (OH) are believed to be one of the major reactive intermediates in the atmosphere. As stated earlier, the process of the production of hydroxyl radicals couples stratospheric ozone depletion directly to tropospheric chemistry. The chemistry of hydroxyl radicals with organic compounds can be simply summarized in two generalized reactions:

$$\text{organic compounds} \xrightarrow[O_3]{OH} \text{removal} \qquad (4)$$

or

$$\text{organic compounds} \xrightarrow[NO]{OH} \text{removal} + O_3 \qquad (5)$$

The distinction between the two routes (4) and (5) is the amount of nitric oxide (NO) in the atmosphere, with the first route occurring at low NO concentrations found in remote (clean) environments.

Estimates have been made of the impact of stratospheric ozone depletion on tropospheric chemistry, using ground-level ozone as the primary indicator. There are two competing processes. A decrease in stratospheric ozone will decrease the amount of ozone transported from the stratosphere to the troposphere (as discussed earlier). Secondly, the reduction in stratospheric ozone will lead to enhanced UV-B radiation, leading to a change in ozone due to the photolytically driven OH reactions shown above.

Two different chemical transport models have been used, with Isaksen *et al.*[57] calculating the impact of a uniform 10% decrease in ozone column, and Fusco and Logan[14] using the measured stratospheric ozone amounts from 1979 and 1993. The results from Isaksen *et al.*[57] are shown in Figure 6-3 for both the ozone amount (left hand panels) and the change in ground-level ozone (right hand panels). While the details of the models are quite different, the broad conclusions they draw are similar. In clean Southern Hemispheric air, the ground-level ozone amount is predicted to decrease with decreased stratospheric ozone (Equation (4)). In the northern hemisphere, a slight increase in ozone amounts is predicted with decreasing stratospheric ozone

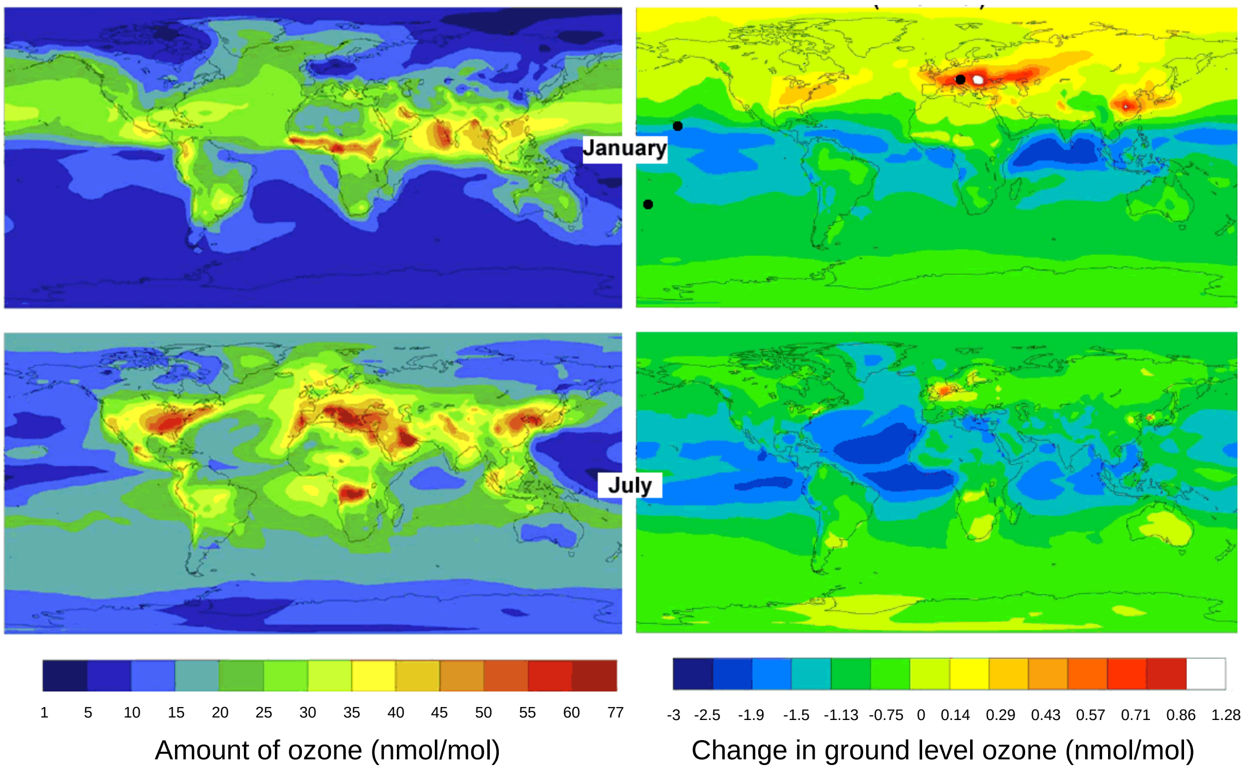


Figure 6-3. Illustration of modeled ground level ozone and the change in ground level ozone resulting from changes in stratospheric ozone. The left hand panels show the modeled monthly average amount of ozone in January and July. The right hand panel shows the changes calculated using a 10% decrease in total ozone column. Figure adapted from Isaksen *et al.*[58]

(Equation (5)). This difference between the hemispheres is caused by the concentration of NO_X from air pollution. Some features of the modeled results differ, and the swap between ozone removal and production would be expected at lower nitrogen oxide concentrations (e.g.[58]), however, the changes estimated by the models indicate that the magnitude of the induced change in tropospheric ozone is small in comparison with the impacts of air pollution.[14, 59]

The impact of likely future changes in atmospheric composition has been assessed using eleven different atmospheric climate models, and they predicted that tropospheric ozone will increase globally by 10 to 30 ppbv from 2000 to 2100.[60] These models have ignored the impact of changes in atmospheric circulation, and so this increase is driven by the increase in anthropogenic emissions of gases like NO/NO_2 and hydrocarbons. Stratospheric ozone recovery is predicted to increase tropospheric ozone by a further 3 ppbv.[60]

Other relevant photolytic processes. Ketones such as acetone ($(CH_3)_2CO$), are present in the atmosphere either due to direct release at the ground or as a product of the decomposition of a range of organic compounds. They are important atmospheric trace gases,[61] playing a key role in the formation and transport of pollutants. For example, acetone can decompose via direct photolysis or via OH, which can be abbreviated as follows:

$$(CH_3)_2CO + OH \rightarrow\rightarrow CH_3COO \tag{6}$$

$$CH_3COO + NO_2 \rightleftarrows CH_3COONO_2 \tag{7}$$

The product CH_3COONO_2 (PAN) is a well known urban pollutant but is also stable at upper troposphere temperatures, and has been recognized as a means of long-range transport of pollutants, such as reactive nitrogen.[62]

A reassessment of the direct photolysis reaction for acetone[63] and for some other ketones[64] has significantly decreased estimates of their overall photolytic sensitivity (quantum yield) in the UV-B region. The changes increase the relative importance of the UV-B region, but decreases the calculated rate of photolysis by 80 – 90%.[65] This indicates that acetone will be quite widely distributed in the upper troposphere in both hemispheres and hence available for reaction to produce PAN. Consequently, the reaction with OH is now understood to be the dominant loss mechanism for acetone. When included in calculations of the global atmosphere, these changes improve the agreement between the theoretical estimates and observations.[65]

The photolysis rate for formaldehyde, one of the key products of the atmospheric decomposition of organic molecules, has been investigated. There are two different routes for decay, and the rate of both routes will depend on stratospheric ozone. Methods for deriving the photolysis rate from irradiance have been proposed,[31] although to date such estimates have not been widely used.

Antarctic atmosphere. Surface ozone at the South Pole is of particular interest as it lies underneath the region of greatest stratospheric ozone depletion. An analysis of measurement records has shown that, during November – December, there was a decrease in ground-level ozone over the period 1970 – 1990.[66] Since 1990, there has been a detectable increase in surface ozone (up to 20%).[67]

Photolysis of ozone and NO in the Antarctic

The importance of photolysis was elegantly demonstrated by measurements of a second peroxide, methylhydroperoxide (CH_3OOH). In this clean environment, the following two reactions represent a significant fraction of the OH reactions:

$$CO + OH + O_2 \rightarrow \ \rightarrow CO_2 + HO_2 \qquad (8)$$

$$CH_4 + OH + O_2 \rightarrow \ \rightarrow H_2O + CH_3OO \qquad (9)$$

It should be noted that there are other reactions which produce these radicals. HO_2 can then react to form H_2O_2 or other peroxides:

$$2HO_2 \rightarrow H_2O_2 + O_2 \qquad (10)$$

$$HO_2 + CH_3O_2 \rightarrow CH_3OOH + O_2 \qquad (11)$$

Additional NO can permit the following reactions to become significant:

$$NO + CH_3OO + O_2 \rightarrow \ \rightarrow NO_2 + HCHO + HO_2 \qquad (12)$$

$$NO + HO_2 \rightarrow NO_2 + OH \qquad (13)$$

The NO removes methylperoxide and produces HO_2 (Reaction (13)). So, when UV was enhanced the increased H_2O_2 was accompanied by a 20% decrease in CH_3OOH.

In the clean Antarctic atmosphere, the NO concentrations would be expected to be low enough that enhanced radical chemistry should result in a decrease in ozone (reaction (4)), as observed in the period before 1990. The change after 1990 is attributed to the impact of additional UV radiation on the snowpack, enhancing nitrogen oxide release.[67] The extra NO produced then alters the atmospheric reactions so that additional UV radiation enhances ozone production. This effect is most noticeable during November when the largest absolute increase in UV radiation is experienced.

Measurements made on the West Antarctic Ice Sheet in 2000 – 2002 showed strong evidence of a negative correlation between stratospheric ozone and ground-level hydrogen peroxide (H_2O_2). A 70% increase in surface H_2O_2 concentration under conditions of low stratospheric ozone (less than 220DU compared to around 320 DU) has been observed.[68] The increase in hydrogen peroxide was explained by an enhanced photolysis of ozone and NO production (see box).

If the impact of UV radiation on NO was not included in a theoretical model, it failed to capture the magnitude of the observed H_2O_2 changes.[68] These changes imply that the atmospheric boundary layer in Antarctica has become more oxidizing due to stratospheric ozone depletion. The ecological impact of this has not been studied.

Impact of air pollution on photolysis by UV-B radiation. The transmission of UV-B radiation to the Earth's surface is increased by removal of stratospheric ozone. The reduction in UV-B radiation by aerosols, and primarily carbon black from combustion has been found to be significant.[38] A global chemical transport model[69] has been used to estimate the impact of aerosols on tropospheric chemistry. Calculations of the impact of aerosols indicate that they typically cause a 1 – 5% reduction in radiation driving $O(^1D)$ formation (UV-B radiation) in general (see Figure 6-4). However, there are regions, notably in Africa, Europe and Asia where impacts of the order of 30% or more are predicted due to increased aerosol release. The net effects on ozone concentrations are more modest (< 5%) and are somewhat smaller than the direct effect of reactions on the aerosol surface.

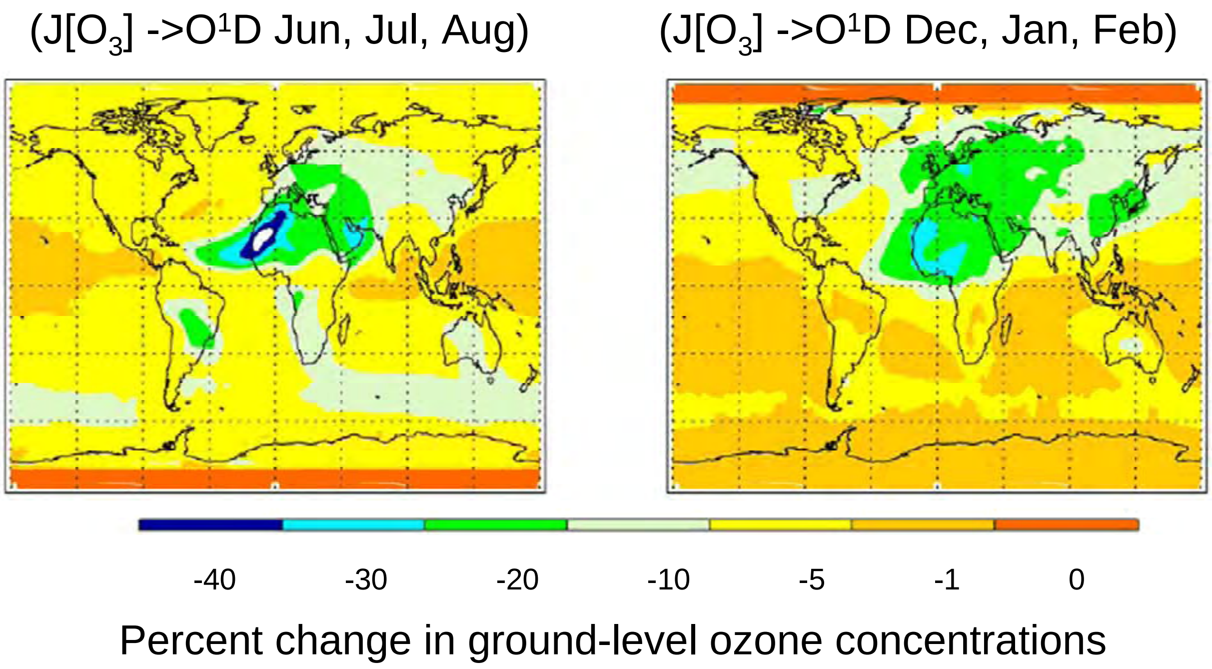


Figure 6-4. Calculated change in ground-level ozone concentration due to the effect of aerosols on (UV-B) photolysis. From Tie *et al.* [70].

Fluorinated substances

Volatile halogenated substances have an important role in ozone depletion and global warming. Since several of these substances are used as refrigerants, there is a potential for interaction between their use, their effects on ozone, and climate change. A number of highly fluorinated compounds (PFCs) undergo degradation and transformation in the atmosphere and several of these have relevance to interactions between global warming and stratospheric ozone depletion. The ultimate degradation products are perfluorinated acids with varying chain lengths, depending on the starting material. The mechanisms of the atmospheric transport and breakdown of long- and short-chain hydrochlorofluorocarbons (HCFCs) have been well-characterized[70, 71] and the results indicate that they are not likely to form the short-chain perfluorocarboxylic acids (PFCAs), such as trifluoroacetic acid (TFA). Perfluorinated aldehydes formed during the atmospheric oxidation of HFCs, HCFCs, and fluorinated alcohols are transported in the atmosphere dissolved in cloud water and react and are subsequently transported to the surface in precipitation. This mechanism has been identified as an additional source of longer chain PFCAs (such as of perfluorooctanoic acid), from the breakdown of fluorotelomer alcohols and possibly explains the presence of perfluorooctanoic acid in remote regions such as the Arctic.[71] These longer chain acids and their parent materials are very resistant to breakdown and have been observed to become more concentrated up the food chain in mammals and birds,[72, 73] where they may have harmful effects.[74]

In contrast to the longer chain PFCs discussed above, several of the HCFCs and HFCs can break down into trifluoroacetic acid (TFA Figure 6-5). These are halothane, isoflurane, HCFC 123, HCFC-124, HFC-134a, and HFC-143a. Observations in both hemispheres (Mace Head, Ireland and Cape Grim, Tasmania) between 1998 and 2002 showed that the concentration of HFC-134a had increased rapidly (3 picomol mol^{-1} year^{-1}) equivalent to 12% year^{-1} based on measured concentrations of approximately 25 picomol mol^{-1} year^{-1} measured in the troposphere at Mace Head, Ireland in 2002.[75] This increase is in agreement with the known usage and atmospheric loss processes. As HFC-134a is a potent greenhouse gas, this increasing concentration has implications for climate change[76] as well as the production of TFA.

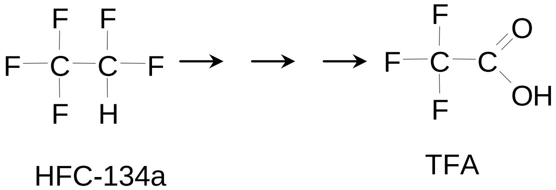


Figure 6-5. Formation of trifluoroacetic acid (TFA) from an HFC.

The final environmental sink for TFA is in the oceans and landlocked lakes. Concentrations of TFA in rainwater range from <0.5 to 350 ng L^{-1}, depending on location and distance from anthropogenic activity.[77] The predominant source of TFA in non-oceanic surface waters is likely anthropogenic as concentrations in surface water samples >2 000 years-old obtained from groundwater and ice cores in Greenland were not detectable (< 2 ng L^{-1}).[78] There are probably natural sources of TFA in seawater; relatively large concentrations have occasionally been detected in close proximity to undersea volcanic vents.[79] Concentrations up to 350 ng L^{-1} in flowing surface waters have been reported from several locations.[80-82] However, in landlocked lakes, they may be as large as 40 000 ng L^{-1}.[80] Reports of TFA concentrations in oceans are generally less than or equal to 200 ng L^{-1}[79, 80, 82] and much of this appears to have pre-industrial natural origins.[83] Based on historical production[84] of HFCs and HCFCs that are potential sources of TFA as well as projections of future uses,[76] an estimate of total production was made (Table 6-1). A worst-case estimate of TFA release from complete conversion of HFCs and HCFCs to TFA produced a total of 22 x 10^6 tonnes of TFA. After dilution and complete mixing in the volume of the oceans (1.34 x 10^{21} L), the increase in concentration above the nominal base level of 200 ng L^{-1} reported by Frank *et al.*[83] would be small (0.016 ng L^{-1}). Even if mixing were slow, concentrations in receiving zones would be less than those in flowing fresh waters or less than double the nominal base concentration. Even considering other sources of TFA, the added inputs from anthropogenic activity will be insignificant.

Table 6-1. Historical and projected production of HFCs and HCFCs in tonnes

	HCFC-124	**HFC-134a**	**HFC-143a**
Total production as of 2003[a]	32 253	1 172 891	39 615
Estimated annual production in 2015[b]	1 000	446 000	72 000
Estimated total production by 2020/2040[c]	93 681	19 402 446	3 351 615

[a] From[84]. [b] From[76]. [c] Phaseout projected for 2020 in developed countries and 2040 in developing countries, half of the total use attributed to developed countries and half to developing countries.

TFA is a strong acid (dissociation constant, pKa, = 0.3) and is completely ionised at normal environmental pHs and is present in the environment as a salt form. It is also highly stable under normal environmental conditions. Its stability in the environment is a direct result of the great strength of the C-F bond and the lack of functional groups on the molecule that are susceptible to chemical or biological degradation (Figure 6-5).

Laboratory and microcosm studies with TFA and related substances[81, 85, 86] have suggested no additional environmental hazards from current environmental loadings in fresh or salt water. The smallest effect concentrations (EC50s) for TFA in sensitive species such as aquatic plants ranged from 0.222×10^6 to 10×10^6 ng L^{-1}.[86] Estimates of more sensitive responses (assay endpoints) suggested a toxic benchmark concentration[87] of 0.046×10^6 ng L^{-1},[81] which is much greater than current[81] or projected concentrations in fresh and saltwater environments. The projected future increased loadings to the oceans from fresh water due to climate change and continued use of HCFCs and HFCs, are judged to present negligible risks for aquatic organisms and humans.

There is evidence that the perfluoropolyethers, substances which are proposed as chlorohydrofluorocarbon (CHFC) substitutes, have great stability to chemical degradation in the atmosphere as well as very large global warming potential.[88] Perfluoropolyethers (PFPEs) are commonly used industrial heat transfer fluids that may be released to the atmosphere. In smog chamber studies on a distilled fraction of a commercial mixture containing perfluoropolymethylisopropyl ethers (PFPMIEs, Figure 6-6), reactivity of PFPMIE with Cl was less than 2×10^{-17} cm^3 $molecule^{-1}$ s^{-1}, while reactivity with OH was less than 6.8×10^{-16} cm^3 $molecule^{-1} s^{-1}$, indicating low reactivity in the troposphere. Using half-life data from perfluorinated alkanes, a lower limit for the total atmospheric lifetime of PFPMIE was calculated to be 800 years. PFPMIE was shown to have instantaneous radiative forcing of 0.65 W m^{-2} ppb^{-1}.[88] This corresponds to a global warming potential (GWP) on a 100 year time-frame of 9000 relative to carbon dioxide (GWP of one) and 1.95 relative to CFC-11, a value exceeded by only a few hydrofluoro ethers.[88] PFPMIE has a longer atmospheric lifetime than CFC-11, and hence, the GWP of PFPMIE increases with the time horizon. For example, the GWP of PFPMIE over a 500 year horizon was estimated at 6.89 relative CFC-11.[88] These substances have been suggested as substitutes for CHFCs[89] but, as they are very persistent in the atmosphere, they may be important contributors to global warming. It is not known whether these substances will contribute significantly to global warming and its interaction with ozone depletion but they should be considered for further evaluation.

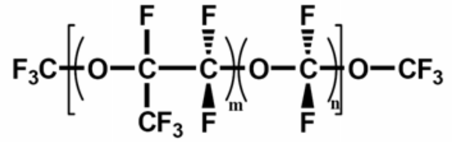


Figure 6-6. Structure of a perfluoropolymethylisopropyl ether.

Conclusions, uncertainties and data needs

Research has now uncovered a distinct signature in air quality due to changes in stratospheric ozone. This is most noticeable in the Antarctic, where the change in stratospheric ozone is the largest and there are relatively few sources of atmospheric contamination. Elsewhere, changes in stratospheric ozone have apparent impacts on air quality, in particular ground level ozone concentrations, which depend on the composition of the atmosphere at that location. These conclusions are in agreement with current understanding of the chemistry of the atmosphere. Calibration and verification data for models are frequently taken from less polluted areas in temperate regions. Additional spatial and temporal measurements of air pollutants, OH, and ozone from other regions such the tropics, forested areas, and highly polluted areas would allow models of interactions between UV-B radiation, climate change, and air quality to be improved.

The decomposition products of CFC's and their replacements do not appear to have a significant environmental impact at this time. Trifluoroacetic acid is very persistent in water, but unlikely to be found at concentrations which present a significant environmental risk. Some other highly fluorinated compounds do have significant global warming potentials and long environmental lifetimes, so their climate impact will need to be considered carefully.

Future impacts of variations in stratospheric ozone on air quality will depend heavily on the magnitude of other changes to the atmosphere, driven by effects such as climate change and increasing industrialization. The projected increase in ground level ozone due to human activities is likely to be somewhat enhanced by recovering stratospheric ozone in the next century.

Our understanding of the atmosphere and the changes that are occurring within it are focused on relatively small regions of the globe. In particular little is known about the tropics, partially because of a lack of measurement but also because of the complexity of the environment. This lack will need to be redressed.

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Chapter 7. Effects of stratospheric ozone depletion and climate change on materials damage.

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Summary

Nanoscale inorganic fillers with average particle sizes smaller by an order of magnitude or more compared to those of conventional fillers are becoming commercially available. The efficacy of these fillers used in polymer formulations and particularly their effect as photostabilizers are beginning to be investigated. These may enhance or retard photodegradation depending on the surface coating of the particles or their chemical nature. Some recent data indicate their use as effective photostabilizers in some common polymers. However, the potential deleterious interaction of the nanoscale fillers with other additives in the formulation has also been pointed out. Depending on the efficiency of stabilization and the economics of their use nanofillers may provide a useful route to UV-stabilization of plastics and rubber used outdoors. Insufficient data are available at this time to assess their potential impact on material and coatings stabilization.

Organic fillers such as lignocellulose continue to be investigated for outdoor applications. Their cost advantage makes them attractive despite the somewhat reduced engineering properties of their composites. Recent reports, however, suggest the photostability of these composites to depend on the source of fiber as well as the processing techniques employed in fabricating products from them. Identification of the key determinants in terms of species, isolation and processing of polymer/wood composites is critical to developing them for long-term outdoor use.

Efforts are continuing on the synthesis of new light stabilizers, particularly those based on a hindered amine light stabilizers (HALS), and on identifying synergistic combinations of known stabilizers for common thermoplastics. Variants of HALS-type stabilizers that reduce the loss of stabilizer via leaching or migration were recently reported. Studies on the permanence of the stabilizers themselves when exposed to solar UV wavelengths have also been reported in recent work. Identification of relevant mechanisms is important not only to understand the interactions of climate changes and higher UV solar environments with materials damage, but also to guide future design of light-stabilizers.

Introduction

Most of the naturally-occurring biopolymers such as wood, hair, wool, and proteins as well as synthetic organic polymers (plastics and rubber) absorb solar UV radiation and consequently undergo photodegradation. About a third of the plastic produced in North America and in Europe is used in building applications. These products such as siding, exposed pipes, glazing, cable coverings, extruded window frames or doors, and in organic protective coatings (See Table

7-1). A majority of these polymers (mainly PE, PP, PVC, PC) are inherently photolabile materials, slowly losing their desirable physical and mechanical properties on routine exposure to solar UV radiation.

Initially, the photodegradation may result in uneven surface discoloration of the product, but extended exposure invariably leads to loss in key mechanical properties such as strength or impact resistance of the material. A large body of research data exists on the types and rates of light-induced damage suffered by different classes of polymers and their numerous formulations. Plastics products are almost never made from pure polymer resin; a number of additives including inorganic reinforcing fillers, thermal stabilizers, antioxidants, flame retardants, colorants, lubricants, processing aids and even biocides are mixed with the pure polymer to obtain a formulation used to fabricate a product. The chemical make-up of the formulation is dictated by the engineering requirements of the product and the environment in which the product is expected to function. As each of the additives can potentially alter the photo-susceptibility of base polymer markedly, the database on the effects of UV radiation on various polymer materials and their formulations is voluminous. The natural biopolymers, on the other hand, have fixed chemical compositions and their photodegradation chemistry is less variable with the source of material.

Table 7-1. Plastics Commonly Used in Outdoor Applications.

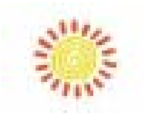
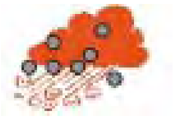
	Plastic Type	**Abbreviation**	**Outdoor Applications**
1.	Polyethylene (High-density and Low-density)	HDPE, LDPE	Irrigation pipes, water storage tanks, greenhouse film, outdoor furniture, artificial turf.
2.	Polypropylene	PP	Artificial turf, outdoor carpet, stadium seating, outdoor furniture, cable covering, toys.
3.	Poly(vinyl chloride) and Chlorinated poly(vinyl chloride)	PVC, CPVC	Rigid pipes for potable and waste water, Extruded window and door frames, Siding and gutters, Conduits and cable covering, Roofing membrane, Roofing membrane, coated fabric for tents.
4.	Polycarbonate	PC	Glazing, outdoor lighting applications.
5.	Copolymers of ethylene and propylene	EPDM	Single-ply membrane roofing.
6.	Acrylic polymer	PMMA	Glazing material.
7.	Fiber-reinforced plastics	FRP	Roofing panels, water tanks, pipes.

Outdoor applications of most common plastics are possible only because of the commercial availability of efficient light stabilizers that can be incorporated in their formulations. Light stabilizers remain the fastest growing segment of the plastics additives market (growing at 6-11% per year). The most used of these are the hindered amine light stabilizers (HALS), a class of remarkably efficient radical scavengers that suppress light-induced degradation in polymers.

A second important group consists of UV absorbers that absorb the damaging UV-B radiation reaching the surface of material and convert it into thermal energy. Either of these stabilizer compounds is typically used at very low concentrations (0.5-1.0% w/w) in the polymer formulation and delivers a defined minimum service life for products regularly exposed to sunlight during use. As it is often the most expensive component in the polymer formulation, R&D effort by industry is focused on improving stabilizer effectiveness and on discovering more potent stabilizer varieties.

An inevitable consequence of stratospheric ozone depletion has been an increase in the UV-B component in solar radiation reaching the earth's surface. This would be expected to accelerate the light-induced degradation reactions in some materials, especially plastics used outdoors routinely, reducing their service life. The projected average increase in ambient temperatures from global warming is only 1.5-4.0 °C over the period 1990-2002[1], too small to cause a significant increase in materials degradation rates; however, a much larger increase in temperature that can significantly increase degradation rates is expected in some geographic regions. In those regions where both higher ambient temperatures and higher UV-B levels occur together, the degradation rates for materials will be considerably accelerated, further shortening the useful life of materials. The relationship between the rate of reaction and the absolute temperature is exponential; a small change in temperature can therefore result in a large increase in the rate of degradation reactions, for reactions of low activation energy. Other climatic factors such as rainfall, humidity, tropospheric ozone, and air pollutants further exacerbate the situation contributing to an even faster rate of degradation of materials exposed outdoors. Some of these worst-affected geographic regions are likely to include developing countries that rely heavily on low-cost plastic products in their economy (for instance China is the second largest producer of plastics in the world). This will also be true of developing countries where wood is extensively used in housing construction. Table 7-2 summarizes the qualitative impact of these factors on the light-induced damage to plastics and wood materials.

Table 7-2. The effect of climatic variables on light-induced degradation of materials.

	 Increase in Solar UV	 Increase in Temperature	 Increase in Humidity	 Increase in Pollutants**
Polymer	++++	+++	+	+
Wood	+++	++	+++	+

** Particularly sulfur and nitrogen oxides and ground level ozone.

The number of + symbols indicate the availability of publications supporting the effect of the particular climatic variable in increasing the light-induced degradation of the material. ++++ =Very High. +++ = High ++ = Moderate + = Low

The challenge to the plastics industry will then be to devise strategies and innovative technologies that allow the service life of the UV-sensitive materials to be maintained at the currently accepted levels despite a potential increase in the solar UV-B component. A simple and possibly effective solution assumes that HALS and other stabilizers in use today used at higher concentrations will turn out to be effective in UV-B-rich solar environments.

Thus, increased concentration of these same stabilizers in polymer formulation or in wood coatings is likely a practical mitigation strategy. While it is a reasonable strategy, the efficacy of the existing light stabilizers with UV-rich solar radiation resulting from ozone layer depletion has not been fully demonstrated. A second feasible response might be to substitute photo-susceptible materials currently in use with better UV-resistant polymers or surface-modified wood products. Either response will add to the cost of the material and the products. Estimates of the global costs associated with either of these strategies are not available at this time. Such estimates need to be based on realistic assessments of materials damage and require the use of advanced damage estimate methodologies as well as the likely efficacy of the various mitigation options available. The research data that allows a rigorous assessment of either of these is unfortunately not available at the present time.

Techniques for assessing light-induced degradation

Quantifying the photodamage associated with the exposure of a polymer material to solar radiation generally requires a knowledge of; a) the wavelength dependence of sensitivity of the material to the particular type of damage of interest; b) a dose-response relationship that is applicable to the exposure and deviations from the reciprocity law applicable to that photodegradation processes; and c) the validity of assumptions of additivity of damage at different wavelengths and the validity of the reciprocity law for the system of interest. Data for wavelength dependence of sensitivity (in the form of either plots of sensitivity vs. wavelength[2] or as activation spectra [3] for several key polymer materials have been published. Only very limited information is available on dose-response relationships of relevant materials for modes of photodamage of interest. New analyses and findings on any of these critical topics therefore contribute to better and more complete damage estimates. Recent literature, however, continues to be sparse on new data on wavelength or dose-response relationships for hitherto uninvestigated systems.

A critical review was recently published on the reciprocity relationships in materials photodegradation.[4] The reciprocity law is the relationship between irradiance I (photons.cm^{-2}), exposure duration t (hours) and the resulting photodamage obtained in an exposure experiment. In instances where the law holds, the quantity (I.t) is directly proportional to extent of damage, implying that a short exposure at high intensity and a longer exposure at a correspondingly low intensity to yield the same amount of materials damage. Even where the reciprocity law generally applies, deviations are expected at both very high and very low irradiance. When deviation is observed at moderate irradiance, a modified form of the reciprocity rule, extent of photodamage is proportional to(I^p.t). A compilation of available data on materials photodegradation reported the value of p~0.9 to 1.0 for a majority of polymers for which data were available[4], confirming that reciprocity is a reasonable assumption for most materials.

Over the years, research on light-induced damage to polymers and wood have consistently relied on fairly modest analytical techniques, mainly conventional spectroscopy for functional group analysis, surface color measurements, and mechanical property measurements.[5] However, these continue to provide new insights as illustrated by the Fourier Transform Infrared Spectroscopy (FTIR) study on acrylic coatings[6] for which material in which a square root dependence of damage on UV irradiance was recently reported.[7] Saron and Felsiberti[8] demonstrated the use of dynamic mechanical analysis (DMA) and FTIR in comparing the surface versus bulk degradation of polymer blends. While providing valuable information, these techniques have limited sensitivity, especially in monitoring early stages of photodegradation.

Recent years have seen a trend towards the use of sophisticated techniques for monitoring light-induced damage. The application of these will allow more accurate damage estimates to be identified and also help in designing more potent light stabilizers. Several interesting examples are discussed below; it is the novel application of the techniques for photodamage studies rather than individual finding from a particular study that is more relevant to the present purpose.

Laser scanning confocal microscopy (LSCM), an ideal technique to study changes in bulk as well as surface of samples was used in a recent study on photodegradation of acrylic coatings[9]. The LSCM technique is likely to be used more commonly in future studies and may allow non-destructive and accurate degradation profile studies via optical sectioning. Infrared attenuated total reflection methods continue to be used over the years in studying depth profiles resulting from degradation of polymers. Work by Nagai et al.[10] and others[11-15] illustrate the value of this conventional technique in establishing the existence of a layered degradation profiles in plastic materials. A highly degraded layer just below the surface of the polymer was observed in their studies.

A second new technique used in analysis of photodegraded materials is atomic force microscopy (AFM) and the related probe microscopic techniques. AFM nanoindentation technique was recently used to study changes in surface hardness of fiber-reinforced vinyl ester composites.[12] This technique has particular merit in studying the newer nanocomposite materials with nanoscale fillers that are below the size limit amenable to optical microscopy. Positron annihilation spectroscopy (PAS), a sensitive technique of analysis for physical defects at a microscale, has been previously employed to study the photodamge to polymers. In the recent study on epoxy polymer[16] a correlation between physical defects and chemical defects (from electron spin resonance (ESR) spectroscopy) in the degraded polymer was demonstrated for the first time. ESR is the more common technique used in recent degradation studies[17, 18] and the observed correlation with PAS suggests that ESR data may have additional value in shedding some light on degradation induced changes in polymer morphology. Advances are also being made on ESR methods; 2-D ESR imaging was used by [18] in and other[11] studying the depth profile of degradation in HALS-stabilized polymers.

Also reported recently was a study on surface photooxidation of poly(ether sulfone) under UV-B irradiation using x-ray photon correlation spectroscopy (XPS) to study the evolution of $–SO_3H$ groups on the polymer surface.[19] In general, use of advanced surface spectroscopic methods (such as XPS) is important as photo-induced changes initiate at the outer surface of the polymer and propagate inwards. Increased use of these highly sensitive techniques to probe photodegraded polymer surfaces is an encouraging development.

Effect of fillers on photodegradation

In fabricating building materials and other products where superior and durable mechanical properties are important, plastic materials are generally compounded with reinforcing fillers such as carbon, or glass fiber. These polymer composites with inorganic inclusions in the polymer matrix have dramatically improved properties such as the modulus and compressive strength. Even with other categories of plastics products, non-reinforcing fillers such as calcium carbonate or rutile titanium dioxide (titania) are typically used either as an opacifier or as a lower-cost filler (relative to polymer) to reduce overall cost of the product. Fillers account for more than 50 percent of the global plastics additives market. Invariably, it is the UV stability of the plastic formulation that determines the service lifetime or the cost of maintaining a useful lifetime of a product used outdoors under increased UV-B levels. Therefore, it is important to review the impact of novel additives including fillers on the service life of plastic compounds under routine exposure to solar UV-B radiation.

The use of fillers can significantly affect the UV-induced degradation of a polymer material; for instance, in rigid PVC compounds, it is the titania (TiO_2) added to the formulation that is responsible for its UV resistance and its outdoor lifetime. Titania is the most widely used white pigment in coating formulations. A compounded polymer is a mixture of a number of chemical additives and interaction of fillers with any of these may lead to unexpected effects. Non-polymer components of the formulation may also be degraded by interaction with an additive. For instance, with plasticized PVC/TiO_2 system exposed to solar radiation, the titania protected the PVC from degradation but promoted the photodegradation of the phthalate plasticizer in the formulation. Outdoor lifetime of the formulation was therefore significantly reduced.[20] Inert fillers that do not chemically participate in the photodegradation reactions, tend to improve the photostability of polymers by shielding underlying polymer from UV exposure.

Experimental data that are in line with the already-established detrimental effect of common additives such as flame retardants[21], recycled plastics [22], copolymers[23], and crosslinking agents[24] were reported in recent studies. Of particular interest is the better elucidation of the mechanism involved in the deactivation of hindered amine type light stabilizers (HALS) in polyolefins due to the presence of an aromatic brominated flame-retardant additive in the same formulation.[25] Photodegradation of the retardant liberates hydrogen bromide that, in turn, reacts with the HALS to convert the active stabilizer into its inactive ammonium salt reducing the service life of the polymer.

Lignocellulose fillers: The use of wood- or fiber-filled polyolefin composites in outdoor applications is not a new technology but is presently increasing in popularity[26]. In 2006 an estimated 500,000 tonnes of wood-plastic composites will be used in building products in North American markets.[27] Wood fibers in general have mechanical properties that allow the design of composites for less-demanding applications and often provide a cost advantage compared to the common inorganic fillers. Recent studies on lingo-cellulose natural fiber fillers suggest that these fillers can either increase or decrease the photostability of polymer composites containing them, depending on the origin of the fiber and the weight fraction of filler used. An important variable in these studies is the processing technique used to fabricate the composite product (i.e., injection molding or extrusion) that very significantly influences its weatherability.[28] For instance, wood shavings and kenaf fibers (49% by weight) were found to enhance the photostability of polyethylene composites.[29] A similar result was reported for the polypropylene/palm-derived fiber composites.[30] Lignin filler in natural rubber formulations was found to exert a stabilizing effect.[31] The palm-derived fibers at 10-40% by weight in unplasticized PVC were reported to increase the light-induced discoloration of the composite but left the mechanical properties unchanged on extended exposure to laboratory accelerated weathering.[32]

Deglise and Beatrice[33], however, reported the wood filler to reduce the photostability of HDPE composites with the acceleration of photodamage proportional to the volume fraction of wood used in the composite material. Studies on injection-molded samples of a similar composite also showed a similar result increasing photodegradability as measured in terms of discoloration as well as loss in mechanical properties.[34] Data reported[35] for PP composites with 25 and 50% w/w wood fiber are also in agreement with the above. This discrepancy in the findings is likely due to differences in the composition of the fibers (and therefore on source of fiber) used in the composites. The chromophores in lignin component of wood fibers absorb of solar UV-B wavelengths making the composites susceptible to photodegradation.[36] Photodegradation of lignin results in the formation of deep yellow degradation products, mainly paraquinone chromophores, via a series of reactions called the phenoxy quinone redox cycle. The brightness reversion of mechanical pulps that contain a lignin fraction is well documented[37, 38] and recently reported for bleached chemi-mechanical pulps.[39] The energy absorption and transfer characteristics depend on the chemical make-up of the fiber (especially the lignin and extractives content. The fraction of fiber in the composite determines the fraction of wood exposed at the surface layers of polymer where UV-B absorption can take place and may also play a role in determining photodegradability of a molded product. Composites of PP/cellulose, already photodegraded by exposure to UV-B was found to enhance the subsequent biodegradability of the material. This confirms earlier findings of similar effects for other composites and is possibly due to increased surface hydrophilicity of the photodegraded composites.[40]

Both wood and wood-plastic composites can be protected from photodegradation by using HALS and UV absorber additives.[41] Muasher and Sain[42] studied the efficacy of various such additives for hardwood powder/HDPE composites and concluded that high molecular weight diester HALS (used either alone or with a benzotriazole UV absorber for synergistic protective effect[43]) was the most effective in controlling fading and yellowing of the material on exposure to UV radiation.

Nano-scale fillers*:* There is growing trend in using nanostructured versions of conventional inorganic fillers in plastic composites. While of similar chemical composition, the nanoscale fillers have an average particle size of only a several tens of nanometers compared to the particle size of hundreds of nm in conventional fillers. The surface area of the nanofillers, however, may be as high a hundred $m^2\ g^{-1}$ of material. The higher surface area allows more extensive interface interactions between the polymer matrix and nanoparticles, resulting in better mechanical properties in the nanocomposite at much lower filler content (therefore at a lower cost as well). Therefore, nanopowdered pigments such as rutile TiO_2, monmorillonite clays, fullerenes, and single-walled carbon nanotubes (SWCN) are being explored as the next generation of reinforcing filler materials. The use of nanofillers in composite applications is expected to grow rapidly in the coming years. Nanoclay masterbatches (or concentrates) designed for automotive applications are already on the market and provide significant cost-savings when used in place of conventional fillers. Nanoclays are also useful in making polymer alloys of normally incompatible polymers (e.g. PP and PS); these are already being used in automotive interior applications. In some plastics, such as copolymers of ethyl vinyl acetate or PP compounds, as little as 3-5% of nanoclay can impart acceptable flame retardancy. This avoids the compounding problems, environmental issues, and interference with HALS components, encountered with the use of brominated flame retardant additives referred to earlier. Recent data also suggest nanoscale pigments may act synergistically with conventional light stabilizers.[44] The paucity of research literature on UV-induced degradation of nanocomposite at this time allows only an initial limited assessment of their potential impact on UV-induced degradation of the polymer composites.

Several studies illustrate the effect of nanofillers in increasing the photodegradability of polymers. Larger surface area interacting with substrate results in increased generation of free radical initiator species that in turn increase rates of photoreactions. Qin et al.[45] working with PE/nanoclay MMT composites found the nanofiller to enhance photodegradation of PE as assessed by spectroscopy. However, the effect depends upon the ionic composition of the filler; and the ammonium ion associated with the exfoliated nanoclay, and may not be generic to the nanomaterial itself.[46] By selecting a photocatalytic nanofiller a rapidly photodegradable plastic formulation can be obtained; mixing nanoscale anatase titania (a photocatalyst) with polystyrene was reported to increase the rate of photodegradation of the polymer.[47]

Nanofillers as light stabilizers: In general opacifiers such as coated titania filler to enhance the light stability of polymer such as PVC by shielding the polymer from UV radiation[48]. Nano-scale fillers now becoming commercially available have much smaller particle sizes and therefore considerably larger surface area available for interaction with radiation. Nano-sized TiO_2, for instance, has an average particle size of about 15 nm and an average surface area of ~100 m^2g^{-1} as opposed to conventional TiO_2 of particle size of size ~300 nm and surface area of 8 m^2 g^{-1}. Light shielding capability at the same volume fraction of filler increases as the particle size decreases (See Figure 7-1) provided the filler is well dispersed n the matrix. With nano-anatase form of titania, large surface area per unit mass lead to rapid catalytic oxidation of coating formulations (based on alkyd and acrylic polymers) compared to conventional fillers; with the coated rutile nanoparticles, however, effective light stabilization in acrylic and alkyd paint media compared to conventional light stabilizers was observed.[49]

The opacifier nanoparticle structure of rutile form of titania consisting of a rutile core, aluminum oxide surface coating, and organic top coating was similar to that for conventional rutile filler. Composite of polypropylene with ZnO nanoparticles also showed stabilization against UV-induced degradation with stabilizer effectiveness increasing with filler content[50]. Model epoxy polymer films with nano-rutile titania was found to show UV-stability in accelerated laboratory exposure[46]; the protective effect varied with the volume fraction of nanofiller in the composition. A particularly efficient photoprotective layer for use as a coating on high-performance textile fibers was made by dispersing 25-70 nm nanoparticles of ZnO or TiO_2 in an acrylic polymer.[51] The same can be achieved with thermoset polymers with ~5 wt percent of ZnO nanoparticles to obtain a coating that is curable, as recently disclosed in a US patent application. The protective base coat or a surface clear coat of the polymer containing metal oxide nanoparticles can be used as a protective coating on a variety of products.[52]

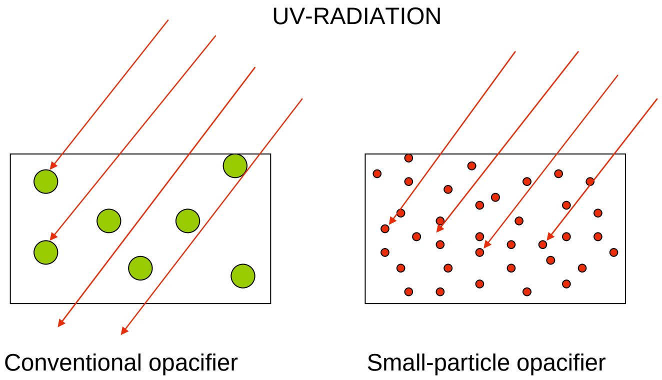


Figure 7-1. Improved light shielding by the same volume fraction small particles compared to large particles of an opacifier in a polymer matrix

Substituting of nanoscale opacifying fillers for conventional fillers in commercial plastics formulation should in theory lead to more efficient photostabilization. Adequate data are not available as yet on the performance of these in commercially relevant formulations. Preliminary data on weathering of LDPE plastic films by Commonwealth Scientific and Research Organization, CSIRO , shows synergistic photostabilization by ZnO (1-2 phr) nanoparticles and HALS (0.3-0.4 phr) in LDPE films.[44] This is not unexpected as similar synergism between HALS and carbon black is known for polyethylenes weathered outdoors. A recent report claimed ZnO nanoparticlulate fillers can in polyolefins display photostabilizer activity exceeding that of HALS compounds.[53] For model liquid systems (e.g. cumene) undergoing thermal as opposed to photo-oxidation, however, Zeynalov et.al.[54] found the effectiveness of a common phenolic antioxidant to decrease when nanosized filler particles were used. The mechanism of increased initiation via catalysis of hydroperoxide decomposition is common in photooxidation of polymers. Therefore it is not clear whether the nanofiller materials will consistently act as stabilizers in the presence of other plastics additives. More data are needed before a final assessment of the value of nanomaterials as photostabilizers can be made.

The preliminary research findings suggest that in some polymer formulations nanoscale filler materials can yield superior mechanical performance as well as UV-B protection in polymer composites are particularly relevant to this discussion. Assuming the availability of low-cost nanofillers with volume production, assuming more efficient photostabilization, this emerging technology may provide a lower-cost route to maintaining service lifetimes unchanged despite any possible increase in the solar UV-B fraction.

Recent Developments in HALS

The most effective (and expensive) light stabilizers for common plastics intended for outdoor are the HALS additives used in concentrations of less that 0.6% (of the polymer). Figure 7-2 illustrates the role of HALS in reacting and removal of free radicals from the oxidizing polymer. Efficient UV light absorbers are also used where the molecule is able to absorb the high-energy UV-B wavelengths and convert these into thermal energy. In addition to the light-stabilizers, plastic compounds generally include a thermal stabilizer package that protects the material from thermal and thermooxidative degradation primarily during processing.

Potential new light stabilizers continue to be reported from time to time.[55] Synergistic combinations of different HALS compounds as well as HALS/UV absorber combinations have been reported to obtain even higher levels of photostabilization[43, 56] and consequently longer service lifetimes. Mixed filler system of titania with conducting carbon black was also reported to enhance the photostability of injection molded poly(propylene-co-ethylene) polymer.[57] As they act via different mechanisms and this is to be expected. The search for better and lower cost light

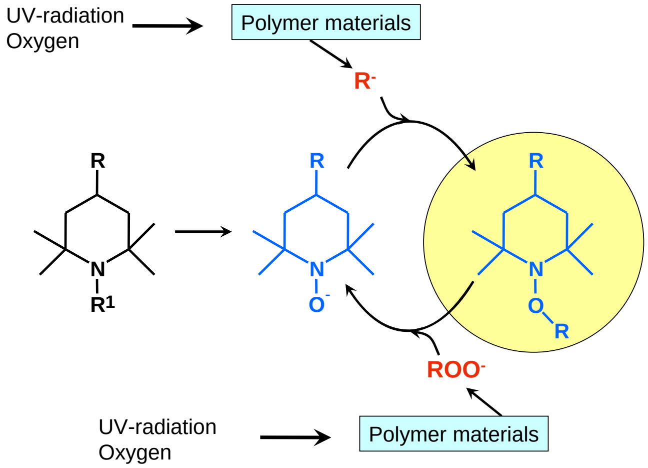


Figure 7-2. Mechanism of HALS action in removing free radicals from photodegrading plastic materials.

stabilizers and polymer types that are inherently photostable in sunlight is an on-going process,[58-60]

A particularly important consideration in the selection of light stabilizers for wood or plastic materials is the permanence of the stabilizer itself especially under higher intensities of UV-B irradiation. Conceptually, the light stabilizer is slowly depleted on exposure either due to slow leaching out of the plastic or due to photochemical breakdown by UV-B. Leaching losses are significant and was recently modeled.[61] As light initiated oxidation begins the mechanical properties of interest deteriorate with further exposure (see Figure 7-3).

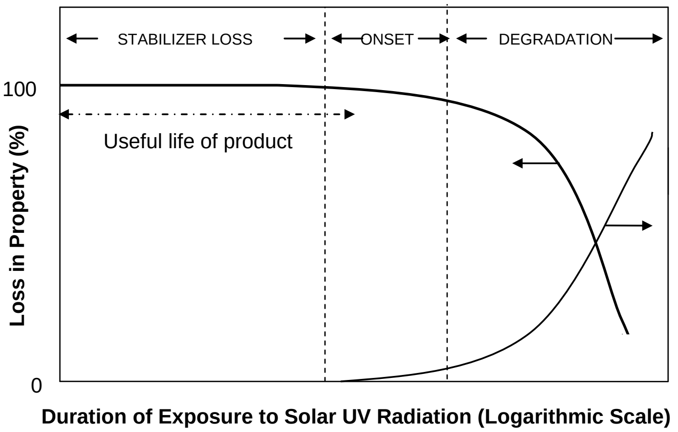


Figure 7-3. Conceptual representation of the onset of degradation reaction with the loss of light stabilizer in a photostabilized plastic material.

The most popular stabilizers belonging to HALS category are not used up in the stabilization reactions but are regenerated within the polymer, and an important route to their loss is via their own degradation. Structural changes undergone by HALS exposed to UV radiation in a PP matrix were investigated using a new reactive thermal desorption gas chromatographic (RTD-GC) method.[21] The kinetics of HALS loss and the build up of nitroxyl radical during exposure to UV were reported and the data help in better modeling of the kinetics of HALS depletion or deactivation on exposure. Chemically affixing the light stabilizer entity on to the polymer chains to improve their permanence by reducing losses via migration to surface[62] has been reported. An interesting HALS type molecule that is also linked to a blue-emitting fluorophore molecule was recently claimed to be a ‘one-step’ fluorescent brightner and a light stabilizer compound[63] In specialized applications where brightening is important, as in textile or paper substrates, the additive may have considerable advantages over conventional technology. Singh et.al.[64] Reported the synthesis of a novel HALS-type polymeric stabilizer and demonstrated its effectiveness on high-impact polystyrene to be superior to that of conventional light stabilizers. This improvement is likely a result of reduced leaching of stabilizer from the matrix.

An interesting HALS molecule also containing a UV absorber moiety was recently synthesized.[65] In clear-coat formulations this stabilizer was shown to photograft itself onto the polymer on exposure to UV and result in reduced migration. Pickett et al[66] discussed a kinetic scheme for delamination of coatings stabilized with UV absorbers that allows better prediction of their service life on outdoor exposure. The approach is superior to the simpler approach of using zero- or first-order kinetic expressions hitherto used. When used in conjunction with HALS in UV-cured polyurethane coatings, Decker[67] found UV absorbers to maintain their effectiveness after 4800 h of accelerated exposure in weather-Ometer.

Processing for photostability

A protective cap layer has been used in the past with PVC polymers as an approach to protection from light-induced damage. A thin adhering surface layer containing high levels of the opacifier coextruded on the surface of a PVC product to protect the underlying less-stabilized polymer from photodamage was reported decades ago. Most of the 23 million tonnes of vinyl plastics produced globally each year is used in building or other outdoor applications. Any reduction in the levels of opacifier, rutile titania, used to stabilize the rigid PVC formulations used in building products is therefore of particular interest. .

A recent patent application[68] proposes the use of a cap layer of weather resistant polyethyleneterphthalate glycol (PETG) coextruded on PVC, CPVC, high-impact polystyrene or other common plastics. Decker et.al. reported a similar technique for PVC, where a UV-cured highly-photostabilized protective acrylic layer was adhered to the surface of a vinyl product to improve its weatherability.[69] The acrylic clear coat carried UV absorbers as well as HALS and the composition showed improved surface properties such as abrasion resistance. The approach is similar to the use of acrylic surface coatings (stabilized with nanofillers) to coat high-value industrial fibers to photostabilize them better.[51] The same concept, but using a polymerizable surface coating with a high level of UV absorber (benzotriazoles) intended for protection products from light-induced damage was disclosed in a recent patent application.[70]

Conclusions and gaps in knowledge

The recent research findings reviewed here focus primarily on the effects of plastics additives, particularly the fillers and flame retardants, on the UV stability of the polymers routinely exposed outdoors during use. Natural fiber-based fillers (lignocelluloses) in polyolefin composites may either increase or decrease photostability of the resin depending on the origin of fiber and the processing technique used. As the mechanical properties of these composites appear to be adequate for some applications, information on which sources of wood fiber enhances polymer photostability will be useful to the industry, particularly in developing countries. With future availability of such information, wood-fibers may contribute to low-cost photostable composites that can function in high-UV solar radiation environments with minimal help from conventional stabilizers.

Nanoscale fillers where the particle size tends to be <100 nm is seen to be emerging as a replacement for the conventional fillers. They are effective as reinforcing fillers at lower concentrations and are therefore cheaper to use. In addition, in early studies some nanoscale fillers appear to impart antioxidant effects, photostability, and flame retardancy to the composite. The very high surface area of UV-absorbing nanoparticle oxides, for instance, also delivers superior photostability to the composites. Only a few publications of preliminary data are available on the topic at this time, but these novel fillers can evolve into an important class of polymer additive in the short term. Their superior light stability suggests widespread availability of nanofillers able to mitigate the effects of increased solar UV at relatively lower levels of use in plastics formulations (or in wood coatings). However, the interaction of nanoparticle fillers with plastics additives used in the formulations remain to be elucidated.

Numerous studies using very sensitive analytical techniques (some for the first time) have contributed to better mechanistic understanding of the chemical and physical changes in surface and bulk of photodegrading plastics and wood materials. These contribute towards a better understanding of light stabilizer – additive interactions as well as the mechanisms of antioxidation in polymers. Such information is invaluable in synthesizing novel photostabilizers and in designing UV-resistant plastics formulations.

The chemistry of interactions between various constituents (including the polymer itself) in plastic formulations exposed to solar UV radiation is poorly understood. This lack of quantitative information makes it difficult to predict the photodamage to a given formulation exposed to solar UV radiation, and therefore does not permit designing new formulations for specified lifetimes under different UV scenarios. A better understanding of how the different climatic factors interact with UV environments in modifying photodamage to materials is needed. Particular important is extending existing mechanistic and predictive models on polymer photodamage to include the effect of changes in ambient temperature, humidity, and air pollutants, all known to impact the photodamage process in biopolymers (especially wood) and plastics.

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