B. Ultraviolet radiation effects on amphibians, coral, humans, and oceanic primary productivity

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Abstract:

Ultraviolet radiation (UVR) is a naturally occurring stressor to most forms of life. The sole relevant source of this stressor is the sun. The earth's stratospheric ozone layer reduces the amount of UVR that reaches the earth's surface. The potential for depletion of this ozone layer due to human activities and the subsequent increase in UVR at the earth's surface is a global environmental concern for both humans and ecosystems. An integrated risk assessment provides efficiency in data gathering, analysis and reporting by enabling risk assessors to use the combined knowledge from many disciplines to evaluate overall risk. This report describes the steps and example information that could be used for an integrated risk assessment but is not an actual risk assessment with all its associated calculations and conclusions. It is intended to be used as an example to stimulate discussion on the applicability of an integrated risk assessment process to a non-chemical stressor.

1. Background

There are very few ecosystems on the Earth that are unaffected by the daily incidence of ultraviolet radiation (UVR). There are fewer still that have not been shaped by exposure to UVR during the evolution of their biological components. Recent changes in UVR exposure at both the global and local level have, however, renewed concern regarding the potentially damaging effects of this ubiquitous stressor. Ozone depletion (Madronich et al., 1995; Kerr and McElroy, 1993), is the primary cause of changes in the dose of UVR received by aquatic and terrestrial species, including humans. Ozone depletion is the thinning (or eradication in the case of the polar ozone holes) of the UVR blocking ozone layer. This is caused by a host of chemicals, most notably chlorofluorocarbons (CFCs). While the 1987 Montreal Protocol and subsequent amendments have limited the production of these ozone-destroying chemicals, recovery of the protective ozone layer is expected to take several more decades (UNEP, 1998).

Although ozone depletion is the primary cause of altered UVR at the Earth's surface, other factors are also changing our contemporary environment. Global climate change, particularly global warming, and acidification both decrease the amount of dissolved organic carbon (DOC) which in turn increases UVR penetration into aquatic ecosystems (Schindler et al., 1996). Dissolved organic carbon is the primary mechanism for limiting UVR penetration into the water column (protecting aquatic organisms and establishing the lower limit of the photic zone); water itself has no UVR absorptive capacity. Climate warming primarily influences UVR penetration by decreasing the total amount of DOC, which is produced in terrestrial systems and then transported to aquatic systems. Climate warming can also act on DOC like acidification does, directly decreasing the amount of DOC already in the water body.

The overall effect of these changes is increased UV irradiance in both terrestrial and aquatic ecosystems. These changes in the relative amount of UVR reaching various habitats may be affecting ecosystems and human health, but research is only beginning to assess and quantify these effects. Humans, amphibians, coral, polar planktonic species, and crop plants are perhaps the five most widely studied biotic groups up to this point regarding UVR effects. Ozone depletion and other forms of global change that can influence UVR exposure are predicted to continue for several decades, if not longer. In addition, ecosystems impacted by increased UVR may take even longer to recover. It therefore is important to begin a comprehensive assessment of the risk of this stressor to living systems.

When considering UVR risks, it is important and unavoidable to note the other side of our long evolution with UVR, namely that organisms require sunlight. Vascular plants, algae and a whole host of organisms that contain autotrophic endosymbionts require light to fix carbon during photosynthesis. Sunlight is required for most vision systems. Exothermic organisms require the radiative heat of sunlight to maintain metabolic processes for cellular growth, development and reproduction. Vitamin D synthesis also requires full spectrum solar radiation. Conversely, it is important to note

that DNA, the genetic roadmap upon which all life relies, is damaged by UVR exposure. Through a number of adaptations (pigment, protective compounds, repair mechanisms, morphology, etc.) biota can exist with UVR exposure. There is a limit, however, to the amount of protection which can be provided and the amount of UVR against which adaptation can protect. Skin cancers are a classic example of the impact of UVR induced DNA damage.

This case study illustrates the advantages of the integrated approach to risk assessment because it capitalizes on the commonalities in stressor source, exposure pathways, and at least some of the mechanisms of effect to enhance the coherence, efficiency, and quality of an assessment of risks from a physical stressor such as UVR. Further, the selection of assessment endpoints emphasizes the interdependence of ecological and human health risks, as effected by the multiple cascading effects possible from exposure to increased levels of UVR. It also demonstrates the importance of considering how environmental regulation can influence the risks of a naturally occurring (albeit artificially modified) stressor.

2. Problem Formulation

2.1 Impetus for the Assessment

Ultraviolet radiation is a ubiquitous stressor that may be impacting human and ecological systems on a global scale. It has been implicated in observed shifts in polar plankton community composition, local and global declines in amphibian population abundance and diversity, coral bleaching syndrome, and an increasing incidence of human skin cancer and other diseases. For example, excesses in human cataracts are estimated to peak at approximately 25 cases per million population under the UVR scenario associated with successful application of the 1997 Montreal amendments to the Montreal Protocol (reported in UNEP, 1998). Similarly, excess skin cancers are estimated to peak at around 100 per million (reported in UNEP, 1998). While quantitative data are limited, failure to understand the risks of changes in UVR has potentially far-reaching implications for the current state of life on this planet. Ecosystems may not be lost but they may be changed from what they are now. Human condition may not change dramatically as a sole result of UVR enhancement but human well-being will no doubt be influenced by the seemingly necessary changes in life style mandated by increased UVR. How the direct and indirect effects of a changing UVR regime influence ecosystem integrity and the health and status of human populations are areas of necessary exploration and assessment. The result of such an assessment should help to identify the extent of need for mitigation actions in both regulatory and personal behavior modification senses.

2.2 Assessment Questions

Put simply, the general question faced by regulatory agencies, public health organizations, and other stakeholder groups is:

> What are the risks to humans and nonhuman biota to the changes in exposure to UV radiation predicted over the next 50 years?

With the myriad defense and repair mechanisms that have evolved through time, there will likely be some populations and species that are better prepared to cope with enhanced UVR exposure. It must be kept in mind that UVR, unlike most traditionally considered environmental stressors, originates from a natural source: the sun. It is, however, enhanced by anthropogenic habitat and climatic alterations, such as ozone depletion, surface water warming, and water acidification.

Additional confounders to answering this question are the established interactive effects between UVR and xenobiotic chemicals (polycyclic aromatic hydrocarbons, pesticides, metals, pharmaceuticals), both in terms of photo-enhanced toxicity and photosensitization. While these factors increase the complexity of the assessment of risk from changes in UV radiation, they also increase the importance of assessing those risks in the context of multiple stressors (chemical pollution, nutrient enrichment and eutrophication, lake acidification) and seemingly unrelated regulatory actions (e.g., promulgation of water quality criteria).

Some additional assessment questions that may be specific to one or more assessment endpoints are:

> What is the relative change in dose at a given location due to all physical factors that influence exposure?
>
> What affects (both direct and indirect) does increased UVR have on specific biological receptors of concern?
>
> How do the interactions between UVR and xenobiotics modify the risk of UVR alone?
>
> Which biological effects are sensitive indicators of risks to broader systems that might serve as sentinels or early warning indicators of UVR risk?

2.3 Assessment Endpoints

Several issues shape the selection of endpoints in this stressor-driven assessment. They include observation of increased damage in important biotic systems that is linked plausibly to changes in UVR, as well as suspicion of increased likelihood of exposure (and subsequent effect) to elevated levels of UVR due to characteristics of the receptor. Although the risks of increases in UVR could be realized across a broad spectrum of living systems, this integrated risk assessment case study focuses on the following four (broadly defined) assessment endpoints. These endpoints are particularly good candidates due to the research already complete and data available.

1. Increased incidence of photo-damage related diseases in humans and wildlife, including skin cancer, ocular damage, immune suppression, and enhanced photosensitivity, that influence health and well being.

2. Disruption and lose of coral reef communities due to coral bleaching/disease, coral mortality, changes in reef persistence and formation dynamics, and cascading reef community interactions.

3. Declines in amphibian populations and lose of amphibian diversity locally and globally, due to increased mutation load, immune system suppression, photo-enhanced toxicity, and other mechanisms.

4. Decreases in oceanic primary productivity that result from impacts on photosynthesis and other UVR-induced damage, and the cascading effects on oceanic plankton community structure and function.

2.4 Conceptual Model

Figure 1 conceptualizes the relationships between the source of UV radiation (the Sun), exposure pathways, and effects on both humans and wildlife to be included in the integrated risk assessment of UVR. The conceptual model communicates hypothesized commonalities in exposure pathways, suggesting efficiencies in data collection, modeling efforts, and characterization of expected direct effects. It further describes suspected linkages among assessment endpoints (sometimes through intermediate biological components) that help to identify potential indirect effects resulting from changes in UVR. This integrated conceptual model helps to define the exposure and effects characterization activities described in subsequent sections. Relationships hypothesized in the model are further described in those sections.

2.5 Analysis Plan

The analysis plan for this assessment takes advantage of commonalities in source and exposure pathways, as well as similarities in some of the mechanisms of action of

UVR damage in all species. The following primary steps would be followed to assess risks to all assessment endpoints:

- Predict changes in levels of UVR impacting earth's surface based on modeling efforts and trends analysis (sources and emissions). Acknowledge uncertainty in future trends projections.

- Estimate local dosimetry based on near-field influences and site-dependent characteristics using data from surface monitoring networks; evaluate exposure confounders for media specific to each of the four assessment endpoints (e.g., water quality), developing relationships between UVR intensity spectra and environmental modulators.

- Translate exposure to expected effect based on action spectra for effect endpoints. Some spectra may be generic for all taxa (e.g., DNA damage); others may be specific for particular assessment endpoints (e.g., photo-inhibition of photosynthesis). Some relevant action spectra exist in the literature; others may need to be developed.

- Cascade direct effects through species interactions and food web dynamics to secondary, indirect effects (relevant only to coral reef and oceanic productivity assessment endpoints) using ecological models.

- Evaluate mitigating results of homeostasis (including repair mechanisms) and adaptation/acclimation on predicted effects using understanding gained through past experimental evidence, natural "experiments" (e.g., the Australian situation described later), and analogy.

- Explore the potential for ecosystems to recover from UVR impacts as the ozone layer and UVR levels return to near-normal conditions.

2.6 Summary

Commonalities in stressor source, exposure pathways, and mechanisms of damage and repair facilitate development of analogous assessment endpoints and holistic conceptual model(s). These conceptual models communicate the direct and indirect pathways of the primary (UVR) and secondary (e.g., loss of food source) stressors to relevant biotic components, taking advantage of the full level of current understanding of UVR physics and exposure issues. Although the UVR action spectrum may be dependent on the specific assessment endpoint, knowing the nature and mechanisms of effect in humans informs our conceptualization of possible direct effects of UVR on amphibians, coral, and phytoplankton, and probably vice versa. Problem Formulation has identified common data needs to evaluate risks from the direct effects of UVR on a variety of receptor organisms and target systems. The integrated assessment benefits substantially from the resource efficiencies gained through use of common models and

flux measurements (networks) in predicting outcomes, and the enhanced insights to possible effects to receptor organisms gained through analogy to other species.

3. Characterization of Exposure

3.1 Sources and Emissions

The only environmentally relevant source of UVR is the Sun. Irradiance from this source is expected to remain fairly constant relative to the temporal scale of this assessment, although significant modification of the amount of UVR reaching Earth's surface are projected as a result of decreased concentrations of ozone in the stratosphere (see below).

3.2 Distribution Pathways

The amount of UVR reaching specific receptors is affected by a number of processes and conditions, including ozone depletion, global change (particularly increased sea surface temperature and altered cloud cover), aquatic acidification (by alteration of concentration of dissolved organic matter), and changing nutrient profiles all can influence the amount of UVR reaching specific receptors.

Specific factors that will influence exposure to receptors include:

- location of the receptor on Earth's surface, because the angle of the sun and altitude both influence the intensity and wavelength spectrum of the radiation
- thickness of the Stratospheric ozone layer as influenced by natural distribution phenomenon and global changes (depletion, hole formation)
- natural habitat protection (e.g., shade, water depth)
- physiological/morphological adaptations or characteristics of receptor organisms (e.g., pigmentation, fur/feather)
- behavioral adaptations of receptor organisms (including use of screens and block by humans and changes in activity patterns by all organisms)
- water quality and clarity

As a result of current efforts to control ozone-destroying chemicals, the maximum ozone depletion and accompanying UVR increase is expected to occur within the next decade (UNEP, 1998). The rate of ozone recovery is difficult to predict, however, due to complicated interactions with changes in the atmosphere such as the expected increase in greenhouse gases. Madronich et al. (1998) estimated that if the various Montreal Protocol controls are met, UVR would be expected to return to normal levels by the middle of this century. These estimates also assume changes in UVR are solely influenced by changes in ozone and that ozone changes are the direct result of halocarbon inputs. Deviations from adherence to the protocols and uncertainties in our understanding of atmospheric chemistry could impact these predictions significantly.

3.3 Transport and Fate Models

Transport (except in the form of radiance) and fate models are not applicable to this stressor.

3.4 External and Internal Exposure Models

Estimates of actual exposure experienced by receptors would be made using existing models of radiance, ozone layer protective effects, and light transmission and penetration through the lower atmosphere and into local habitats and setting. Models of global change and trends analysis based on monitoring data from surface networks would be used to project plausible UVR change scenarios relevant to the temporal bounds of the assessment. "Local" modifications of intensity spectra would be made as appropriate to receptor habitat (e.g., depth in water column) using appropriate models, empirical relationships, and estimated exposure at the molecular level estimated.

Similar considerations are needed for secondary stressors (xenobiotics) and other confounders. Secondary stressors of concern include polycyclic aromatic hydrocarbons, or PAHs (Arfsten et al., 1996; Fernandez and l'Haridon, 1994; Huang et al., 1993), pesticides (Zaga et al., 1998), and metals (Rossman, 1981). Interactions between these stressors and UVR have been found in both marine and freshwater systems, in plants and animals. Effects may only be seen in the combined exposure of UVR and these compounds when exposure to ambient levels of the chemical are insufficient to cause effects. Additional confounders include environmental changes such as acid rain (Wright and Schindler, 1995) and global climate change, especially altered sea surface and lake temperature (Leavitt et al., 1997; Morris and Hargreaves, 1997; Vodacek et al., 1997; Siegel and Michaels, 1996; Schindler et al., 1996).

3.5 Measures of Exposure Related Parameters

The important step in assessing the current and predicted risk of enhanced UVR is to have reliable and comparable dosimetry. This needs to be done on both global and local scales. Global/regional monitoring systems are currently being employed and expanded in North America and Europe, as well as at several global hot spots. Examples of such systems in the United States include networks run by the U.S. Environmental Protection Agency and the Department of Agriculture. On a local level, individual experiment and field monitoring needs to include full spectrum, calibrated dosimetry. Additionally water quality parameters (DOC concentration, turbidity, reactive chemical concentrations) also need to be monitored during research evaluating UVR effects on aquatic ecosystems.

3.6 Analytical Tools

While there still is some debate regarding the best instrumentation at both the local and the global scale, every effort should be made to provide such data so that results

are comparable. Models will be used to extrapolate broad-based monitoring data to dose. Because biological damage and effects are functions of both wavelength frequency and intensity, UVR exposure would be quantified as frequency spectra. Modifiers and other confounders would be quantified as appropriate to those factors.

3.7 Summary

The source of UV radiation and many of the exposure pathways are identical for both human and ecological assessments, allowing common information on UVR intensity obtained from monitoring networks and tools to be used as the initial measure of exposure. Models and methods for estimating or measuring dose can also be shared. The benefits of an integrated approach thus include cost efficiencies and minimization of data collection needs.

4. Characterization of Effects

4.1 Reported effects and modes of action

The range of effects of UVR on humans includes skin cancer, immunosuppression, and ocular damage (de Gruijl and Van der Leun, 1993; Noonan and De Fabo, 1993; Zigman, 1993). Enhanced photosensitivity also has been reported in conjunction with use of some pharmaceuticals. Reported direct effects on amphibians include developmental damage, mortality, and possible immunocompromise (Carey, 2000; Blaustein et al., 1997; Worrest and Kimeldorf, 1976; Cummins et al., 1999). UVR has been implicated as a factor in coral bleaching but the mechanism is unknown (Fitt and Warner, 1995; Gleason and Wellington, 1993; Lesser et al., 1990). Enhanced UVR also has been reported to decrease photosynthetic rate of marine algae. Many of these effects are predicated on DNA damage although the mechanisms are mostly speculative. The role of behavioral, morphological and physiological protections increase the variability in these responses among species, populations and individuals.

In the case of humans, cumulative life-long dose seems to play a role in susceptibility, with early life exposure having the most impact, yet effects often are not seen until later in life (WHO, 1995). Early life-stages of other organisms are also likely to be the most sensitive (Longstreth et al., 1998). Frogs, for example, are more sensitive in the early larval stages than as adults (Hansen, 1998).

Another important aspect of UVR effects, due to the evolutionary time frame of exposure, is the variability of effects among populations. Some populations are simply better adapted to higher levels of UVR. In humans, the most obvious example includes the observation that human populations which have historically existed in areas near the equator, where UVR is naturally more intense, have greater concentrations of melanophores and melanin in their skin. The effectiveness of this phenology as protection is demonstrated in the adverse impacts of UVR on individuals from higher latitudes when they immigrate to equatorial regions. For example, Australians of British

extraction suffer a higher rate of skin cancer compared to their aboriginal neighbors (Green and Williams, 1993). In coral, such variation is seen between shallow and deep-water populations of the same species (Gleason, 1993; Siebeck, 1981, 1988). Variability has also been seen in amphibian populations, with high elevation populations often being better adapted to high UVR intensity (Hansen, 1998). Phenologic variation among populations will influence the degree to which effects are experienced even at similar exposure levels.

4.2 Biomarkers and Indicators

DNA is often referred to as one of, if not the, most sensitive targets of UV-B (a limited range of the UVR spectrum) damage (Setlow, 1997), with exposure resulting in mutations or cell death (Mitchell and Karentz, 1993). DNA lesions can be linked directly to UV-B radiation (Buma et al., 1997; Malloy et al., 1997). Lesions produced by UV-B include pyrimidine dimers and (6-4) photoproducts. Biomarkers of DNA damage might be both useful indicators of exposure to UVR and predictors of biological effect. Commonalities among species in causal pathways leading from DNA damage to biological effect could increase the efficiency of data collection and use in the integrated risk assessment.

4.3 Exposure-Response Modeling

UV radiation is composed of a band of wavelengths with varying influence of environmental factors on exposure and effects. As a result, measures of biological responses to UVR need to be based on exposure to specific wavelengths, i.e., action spectra. The response of DNA to UVR has been quantified by its action spectra for damage (Setlow, 1974). Other endpoints have also been quantified with their action spectra, including erythemal response (McKinlay and Diffey, 1987) and plant damage (Rundel, 1983). Characterization of direct effects of UVR on the assessment endpoints would rely on action spectra as the description of exposure-response relationships. Action spectra may be transportable across assessment endpoints if the mechanisms of effect are similar.

4.4 Extrapolation

Because data on the effects of UVR will always be limited, the ability to extrapolate existing data to other organisms, systems, and situations is a necessary activity of the integrated risk assessment. Many uncertainties result from our inadequate knowledge of the factors influencing the accuracy of such extrapolation. Extrapolations of effects between tested and untested species (e.g., mice and humans, frogs, and salamanders) can be aided by the knowledge of common mechanisms and action spectra. These data can assist in estimating responses of sensitive populations as well as understanding the influence of changes in behavior patterns on exposure. Extrapolations from individuals to populations require models of population responses that incorporate

measures of survival, fecundity, productivity, and other factors that influence population dynamics. These models are often unavailable. Our ability to extrapolate to ecosystems from individual species responses must be based on an understanding of the interactions among biotic and abiotic components, and depend on models of ecosystem structure and function at a variety of spatial scales.

4.5 Direct and indirect effects

As was mentioned earlier, UVR can impact biological systems on a number of levels in ways that are not limited to the direct effects on the exposed individuals themselves. In addition to DNA and cellular damage, morphological changes induced by UVR exposure can affect trophic dynamics (Zellmer, 2000). In the case of a coral reef ecosystem, UVR effects can occur at a number of trophic levels. The heart of the coral reef ecosystem is the coral/zooxanthellae symbiosis. Both species in the symbiotic relationship may be affected independently by UVR and by the resultant adverse reaction of the other. For example, changes in dinoflagellate pigmentation can alter the protection afforded to the coral, and changes in coral cell condition may alter the suitability of the environment in which their endosymbionts live. As coral rely on zooxanthellae to some extent for energy production through photosynthesis, any alteration in zooxanthellae condition or location could adversely effect coral condition. Additionally, other coral reef inhabitants, including algae (macro- and micro-), invertebrates, and fish, all can be affected directly by UVR exposure and indirectly by changes in coral condition. The cascading effects of poor coral condition can include a shift in dominance in the reef community to algal species that changes nutrient profiles and habitat suitability for other reef inhabitants.

The effects of UVR on oceanic primary production could also extend beyond the direct effects on phytoplankton. The biomass production of nearly every fisheries is limited by food supply (Cushing, 1982; Nixon, 1988). An UVR-induced decrease in primary production could cascade through the food web to affect larval fish that feed on phytoplankton or, more often, on zooplankton whose abundance is tied to primary production. These effects include reduced growth rates of larval stages of fish and invertebrates. In turn, reduced growth rates can increase larval mortality rates through intense size-specific predation and survival. An extended larval stage also increases the probability that currents will transport the larvae to an unsuitable habitat. Changes in oceanic productivity likely would have indirect effects on humans and other consumers through impacts on food supplies.

However, the specific mechanisms by which UVR effects cascade through the food web are likely to vary in different portions of the ocean. Biologically damaging UV-B appears to be limited to approximately the top 10-15% of the euphotic zone in ocean waters (Behrenfeld et al., 1995). Hardy et al. (1996) concluded that the most pronounced inhibitory effect on oceanic primary productivity would occur in the sub-Antarctic (40-50 degrees south) and not in the Antarctic or the tropics. Measurable decreases in phytoplankton-specific growth rates and biomass from UVR are also most

likely to occur in nutrient-rich areas of the ocean. In other areas, nutrient limitation rather than UVR may well be the limiting factor (Behrenfeld et al., 1994).

4.6 Summary

Several endpoints have common mechanisms of effect and similar biomarkers of effect, a situation that promotes characterization efficiencies by permitting extrapolation of effects across species. An extrapolation approach to developing action spectra models (wavelength specific exposure-response models) may be possible depending on the similarity in mechanisms and exposure pathways. Recognition of linkages among components of the integrated conceptual model enhances understanding of the indirect effects of UVR exposure that can influence risks to the assessment endpoints in (sometimes) unexpected ways. Similarly, information about potential effects to some assessment endpoints may suggest previously unexpected effects to other endpoints for which information is lacking.

5. Risk Characterization

5.1 Combining Exposure and Effects

As described in section 2, the assessment question being addressed is "what are the risks to humans and nonhuman biota to changes in exposure to UV radiation over the next 50 years?". To evaluate these risks requires projection(s) of specific stratospheric ozone depletion scenarios and the resultant increases in UV radiation projected to reach the earth's surfaces. Profiles of UVR exposure at various points on the earth can be coupled with exposure-response relationships for individual assessment endpoints to estimate expected direct effects on humans and ecological systems. When combined with modeled estimates of indirect effects resulting from ecosystem interactions and linkages among assessment endpoints, the exposure profiles and exposure-response relationships provide holistic, integrated estimates of risk to the assessment endpoints.

5.2 Determining Causation

Establishing causation between UVR and apparent effects on assessment endpoints depends upon two primary types of information: correlative (associative) and mechanistic. Correlative relationships are associations of changes in human or ecosystem condition with broad measures of variation in UV radiation. These associations might include, for example, incidence of skin cancer and cataracts as a function of latitude and longitude, or effects on plankton productivity as a function of water quality and behavioral patterns. Mechanistic relationships provide understanding of the causal linkages between exposure and the direct and indirect effects on specific receptors. Mechanistic relationships often are deduced experimentally by manipulating UVR wavelengths in the laboratory (using artificial lights or specific wavelength filters) or in limited field manipulations (using specific wavelength filters of natural sunlight). Conclusions regarding causation are strengthened as similarities in associative and

mechanistic relationships increase among the various pathways described in the integrated conceptual model.

5.3 Combining Lines of Evidence

Conclusions about the risks of UVR, and confidence in those conclusions, can also be strengthened by accumulating evidence that verifies and quantifies the relationships hypothesized in the integrated conceptual model. Evidence of biologically meaningful changes in UVR (as modeled by exposure scenarios) derives from the aforementioned monitoring network data and observations of stratospheric ozone depletion. The evidence of effects of UVR on assessment endpoints includes a combination of direct experimental determinations of UVR action spectra relevant to the assessment endpoints coupled with associative descriptions relating differences in UVR intensity with direct biological effects as observed in natural manipulations of UVR. The weight of evidence supporting risk conclusions increases as the information about expected effects across assessment endpoints converges onto a common picture. Thus, evidence of effects of increased UVR on amphibians, when combined with evidence of analogous effects on humans, helps to define and strengthens confidence in the risk characterization.

5.4 Uncertainties

The primary uncertainties in the assessment reflect those associated with future UVR changes and the specific adaptations individuals and organisms use that influence dose and effect. Estimating the future risk of UVR depends in part on predictions of future ozone levels as part of overall global change. There also is limited quantitative data on the relationship between environmental variables and UVR transmittance/penetration in specific systems (particularly aquatic). Because UVR is a naturally occurring stressor, numerous adaptations, behaviors and repair mechanisms exist. Predicting risk to individuals and populations generally includes only a subset of these adaptations. Additional uncertainties in understanding and predicting effects on humans and ecological systems include: (1) confirming the mechanism of effects on a broad spectrum of organisms and endpoints; (2) extrapolating the effects from tested to untested species; (3) understanding the significance of predicted effects to populations and system functions; and (4) the inadequacy of our knowledge of the global nature of interactions impacting large-scale ecological systems.

5.5 Presentation of results

Results of the integrated risk assessment of changes in UVR would be presented as a coherent package that presents risks to endpoints in connected fashion based on a common scenario for UVR intensity and distribution. Results would be communicated as probabilities of direct effects on individual humans (increases in cancer rates and ocular damage) in the context of human welfare, well being, and social systems coupled with probabilities of extinction and projected losses of biodiversity and productivity of aquatic

ecosystems. The relationships between loss of ecosystem productivity to quality of human condition will be described to the extent possible.

5.6 Summary

The risk characterization would use future UV radiation scenarios, action spectra and other exposure-response relationships to characterize direct effects, and ecological models to characterize indirect effects on assessment endpoints. Understanding of the relative risks among assessment endpoints is enhanced by using exposure scenarios, models, and data shared in common, and by drawing analogies among mechanisms of effect when possible. Causation would be determined through evaluation of a combination of correlative and mechanistic relationships linking UVR to biological effect, with risk conclusions being further defined and strengthen as multiple lines of evidence about those relationships converge. The overall confidence in risk conclusions would be enhanced as similarities among risk estimates for individual assessment endpoints emerge.

6. Risk Communication

To date, communication of risks of UVR has focused primarily on those to human health. Some regions of the world are establishing and communicating policy to respond to elevated UVR levels and human sensitivity. Many federal governments, including the US, have adopted the UVR Index (developed by the World Health Organization, World Meteorological Organization, United Nations Environmental Program and the International Commission on Non-Ionizing Radiation Protection) to explain daily dose and risk to the public. Many countries are also offering standardized guidelines on use of sunscreens and acceptable daily UVR exposure. For example, the Australian Radiation Protection and Nuclear Safety Agency monitors UVR levels, assesses effects, and offers "personal protection strategies." In 1996, Australia started to standardize UVR protection rating of clothing, and several countries (including Australia, the UK, Germany, France, and the US) have developed such standards for sunglasses. Further, various governmental (US EPA SunWise School Program, Australian Radiation Protection and Nuclear Safety Agency, and others) and non-governmental (national Cancer Societies, World Health Organization, United Nations Ozone Sectretariat, and others) groups have developed aggressive public health communication efforts about risks and personal protection approaches. Because of this attention to public health, and despite implications that UVR may be a contributor to observed impacts in selected ecological systems such as coral reef communities, phytoplankton communities, and amphibian populations, public perception of UVR risks has been shaped largely by human health effects.

To support a more holistic understanding of UVR risks, and thereby facilitate more effective risk management (including personal choice) decisions, coherent messages should be developed about risks to all assessment endpoints. Such messages should explain the current knowledge of UVR effects, including expected impacts on

sensitive ecosystems as well as projected changes in human disease incident attributable to UVR exposure. Although continued emphasis on health effects may be needed in certain venues (for example, public health programs) to promote effective personal protection, communication of risks to regulatory agencies and international governance bodies needs to be consistent and integrated with respect to all assessment endpoints. Coherent expression of the risks of changing UVR will help these groups establish priorities for risk management action.

6.1 Summary

An emphasis on communicating risks of UVR exposure to human health, although necessary to facilitate effective choices about personal protection, has under-represented possible adverse effects on ecological systems. The resulting lack of appreciation of ecological risks can hinder identification of risk management priorities. An integrated approach to risk communication would provide consistent, coherent, and simultaneous expressions of risks to all assessment endpoints, thereby facilitating selection among potential mitigation efforts that minimize risk to humans and non-humans alike, and promoting understand of why various risk management actions are taken.

7. Risk Management and Stakeholders

Due to the global nature of UVR exposure and the causes of UVR enhancement, effective risk management and stakeholder involvement will require creative solutions. Although the integrated risk assessment can provide improved understanding of the likely adverse effects that would be experienced by all assessment endpoints under various exposure scenarios, the decisions made by the regulatory community and the public should be integrative as well to be effective in mitigating risk.

The Montreal Protocol, along with its subsequent amendments (see UN Ozone Secretariat for details), currently sets down control limits on known causes of ozone depletion. It will be at least a decade before their effectiveness can be assessed with respect to reduced UVR exposure. In the interim, the public health community will need to continue programs to encourage human behavioral changes to minimize exposure. The most aggressive these have been in Australia, but many other countries (including the United States) are increasing public awareness both through government agencies and non-government organizations, such as national cancer societies. Additionally, there may be need for enhanced regulatory approaches for evaluating and rating protection, such as creating testing and guidelines to standardize SPF and sunscreen ingredients, sunglasses, and clothing. As mentioned above, a group of international organizations have created the UV Index for informing the public about UVR exposure effects and personal exposure decision making.

In contrast, facilitated behavioral adjustments and personal protection strategies are not possible or feasible for non-human receptors. Protection of stratospheric ozone

layer seemingly is the principle option for minimizing risks to ecosystems. Ideally, management decisions directed towards source reduction (that is, minimization of ozone stratospheric depletion through control of ozone-depleting substances) would consider the potential for both direct and indirect effects on ecological systems and direct effects on humans simultaneously. In evaluating relationships between source reduction and risk, attention should be paid to alterations in UVR penetration due to changing sea surface temperature, DOC content, and acidification, and to introduction of photoreactive anthropogenic contaminants in sensitive ecosystems.

In addition to direct and indirect effects of increased UVR on ecosystems and ecological processes, risk management decisions should consider the impact of ecosystem degradation on human well-being. Potential indirect ecological impacts with respect to humans include: changes in food production (availability and success of crops and fisheries), forest health (crucial in limiting the effects of greenhouse gases), and other ecosystem services (decreased oxygen production by phytoplankton, loss of recreational resources). Loss or degradation of any of these ecosystem functions likely would be detrimental to humans and other assessment endpoints, as well as difficult to correct once they are altered. The connections between ecological and human well-being are, perhaps, among the strongest arguments for integrated assessment and management of UVR risks.

7.1 Summary

Risk management informed by more holistic understanding of risks of changing UVR to humans and ecological systems will result in management and personal decisions that are most effective in optimizing risk mitigation strategies. Integrated risk assessment characterizes the adverse effects of enhanced UVR on all assessment endpoints in a consistent and coherent manner, allowing relative risks among assessment endpoints to be compared and understood, and the tradeoffs inherent in various risk management options to be transparent and recognized. Integrated risk assessment also facilitates a deeper understanding of the dependence of human well-being on ecological functions and services, thereby aiding in the identification of management approaches that optimize risk reduction from both human health and ecological perspectives, and avoiding unintended consequences.

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Figure 1. Conceptual model of risk of UV to humans and ecosystems (modified from U.S. EPA, 1998)