AN ASSESSMENT OF THE RISKS OF STRATOSPHERIC MODIFICATION

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Chapter 7

CHAPTER 7

NONMELANOMA SKIN TUMORS

SUMMARY

Nonmelanoma skin tumors are the most common cancers occurring in white populations. The two major forms of nonmelanoma skin tumors are basal cell carcinoma (BCC) and squamous cell carcinoma (SCC). Although the incidence of BCC is generally several times greater than the incidence of SCC, SCCs account for as much as four-fifths of all nonmelanoma skin cancer deaths. Prolonged sunlight exposure is considered to be the dominant (but not only) risk factor for nonmelanoma skin tumors. As a result, it is believed that increased exposure to solar UV radiation (due to the depletion of the ozone layer) may increase the incidence of nonmelanoma skin tumors among susceptible populations. SCC would have a much larger percentage increase than BCC.

Individuals with predisposing genetic traits such as albinism and xeroderma pigmentosum (a genetically inherited inability to repair UV-induced DNA damage) are at the highest risk of developing nonmelanoma skin tumors. The much larger population consisting of light-skinned individuals is considered to be at high risk, expecially those with a susceptibility to sunburn, poor tanning ability, red or blond hair, blue or green eyes, and a Celtic heritage. Populations with pigmented skin, such as blacks, are at significantly lower (but not zero) risk.

FINDINGS

1. BASED ON THE SURVEYS WHICH HAVE BEEN CONDUCTED, PARTICULARLY IN THE UNITED STATES AND IN AUSTRALIA, PROLONGED SUN EXPOSURE IS CONSIDERED TO BE THE DOMINANT RISK FACTOR FOR NONMELANOMA SKIN TUMORS.

   1a. Nonmelanoma skin tumors tend to develop in sun-exposed sites (e.g., the head, face, and neck).

   1b. Higher incidence rates occur among occupational groups with outdoor exposures compared to those with indoor exposures.

   1c. A latitudinal and UV radiation gradient exists showing the highest incidence rates in geographic areas of relatively high UV radiation exposure.

   1d. Skin pigmentation provides a protective effect.

   1e. The risk of nonmelanoma skin tumors is highest among genetically predisposed individuals (e.g., those with xeroderma pigmentosum).

   1f. There is a predisposition for nonmelanoma skin tumors to develop among light-skinned individuals who are susceptible to sunburn and who have red/blond hair, blue/green eyes, and a Celtic heritage.

2. AVAILABLE EPIDEMIOLOGICAL EVIDENCE SUGGESTS THAT SCC AND BCC RESPOND DIFFERENTLY TO DIFFERENT LEVELS OF SOLAR EXPOSURE. IT HAS BEEN HYPOTHESIZED THAT CUMULATIVE UV RADIATION HAS A GREATER EFFECT ON THE DEVELOPMENT OF SCC THAN ON BCC.

   2a. The BCC/SCC incidence ratio decreases with decreasing latitude.

   2b. BCC is more likely to develop on regularly unexposed sites (e.g., the trunk) compared to SCC.

   2c. SCC is more likely to develop on sites receiving the highest cumulative UV radiation doses (e.g., the nose) than BCC.

   2d. For a given cumulative level of sunlight exposure, the risk of SCC may be greater than the risk of BCC.

3. THE RESULTS FROM SEVERAL EXPERIMENTAL STUDIES SUGGEST THAT UVB MAY BE THE MOST IMPORTANT COMPONENT OF SOLAR RADIATION THAT CAUSES VARIATIONS IN THE INCIDENCE OF NONMELANOMA SKIN TUMORS.

   3a. UV radiation produces malignant nonmelanoma skin tumors in animals. UVB wavelengths have been shown to be most effective in producing these tumors.

3b. UVB has been shown to cause a variety of DNA lesions, to induce neoplastic transformation in cells, and to be a mutagen in both animal and bacterial cells.

4. SEVERAL RESEARCHERS HAVE INVESTIGATED THE CHANGES IN THE INCIDENCE OF NONMELANOMA SKIN TUMORS THAT MAY RESULT FROM INCREASES IN EXPOSURE TO SOLAR UV RADIATION.

4a. Several studies have provided estimates of a biological amplification factor, BAF (also referred to as an amplication factor, AF), which is defined as the percent change in tumor incidence that results from a one percent change in UVB radiation. The results from six studies produced an overall AF range for all nonmelanoma skin tumors from 1.8-2.85, although AF estimates for one sex may be even larger (e.g., 1.4-3.2 for males from Fears and Scotto (1983)).

4b. AF estimates are generally higher for males than for females and increase with decreasing latitude. In addition, the AF estimates for SCC (e.g., 1.6-7.1) are higher than the AF estimates for BCC (e.g., 1.1-2.6). This finding is consistent with observations that the BCC/SCC ratio decreases with decreasing latitude and that BCC is more likely to develop on unexposed sites.

4c. There is, however, no general concensus on the best quantitative measure to use in estimating the effect of ozone depletion on UVB dosage. Although AF estimates appear to be relatively insensitive to the method used to measure UVB dosage, the estimated effect of ozone depletion on nonmelanoma skin cancer incidence is sensitive to the choice of an action spectrum. For example, using the DNA action spectrum, Scott (1981) estimated that a 5 percent ozone reduction would lead to an increase in BCC in Minneapolis-St. Paul of 7.7 percent for males and 5.8 percent for females. Using an action spectrum based on the RB meter, with the same functional model and cancer incidence data, Scotto et al. (1981) estimated corresponding increases to be 5.6 percent for males and 4.4 percent for females. The reason for this difference is that the optical amplification factor (percent change in weighting UVB wavelengths) is very sensitive to ozone depletion.

INTRODUCTION

There are two major types of nonmelanoma skin tumors: basal cell carcinoma (BCC) and squamous cell carcinoma (SCC). These tumors are among the most common cancers occurring in white populations. In the United States alone, nonmelanoma skin cancers develop in more than 400,000 white Americans each year and represent over half of all cancers occurring in U.S. whites.

Prolonged sunlight exposure is considered to be the dominant, but not only, risk factor for these tumors based on several observations. Nonmelanoma skin tumors tend to develop in sun-exposed sites (e.g., head and face) and occur at a higher rate among outdoor workers compared to indoor workers. Nonmelanoma incidence rates are higher in areas with high UV radiation exposure (e.g., closer to the equator) than in areas with low UV radiation exposure. Light-skinned individuals who are susceptible to sunburn and who have red/blond hair, blue/green eyes and a Celtic heritage are at high risk of developing nonmelanoma skin tumors.

To evaluate the role of solar radiation, and UVB in particular, in the development of nonmelanoma skin tumors, this chapter reviews the biology of the skin and nonmelanoma skin tumors focusing on their responses to solar radiation. Evidence from molecular and animal studies relating nonmelanoma skin tumors to ultraviolet radiation (UVR) is discussed. Epidemiological evidence linking exposure to solar radiation and development of nonmelanoma skin tumors among human populations is also reviewed. Finally, the available dose-response relationships that estimate potential changes in the incidence of nonmelanoma skin tumors due to changes in exposures to UVR are presented.

BIOLOGY OF NONMELANOMA SKIN TUMORS: LINKS TO UVB

Biology and Photobiology of the Skin

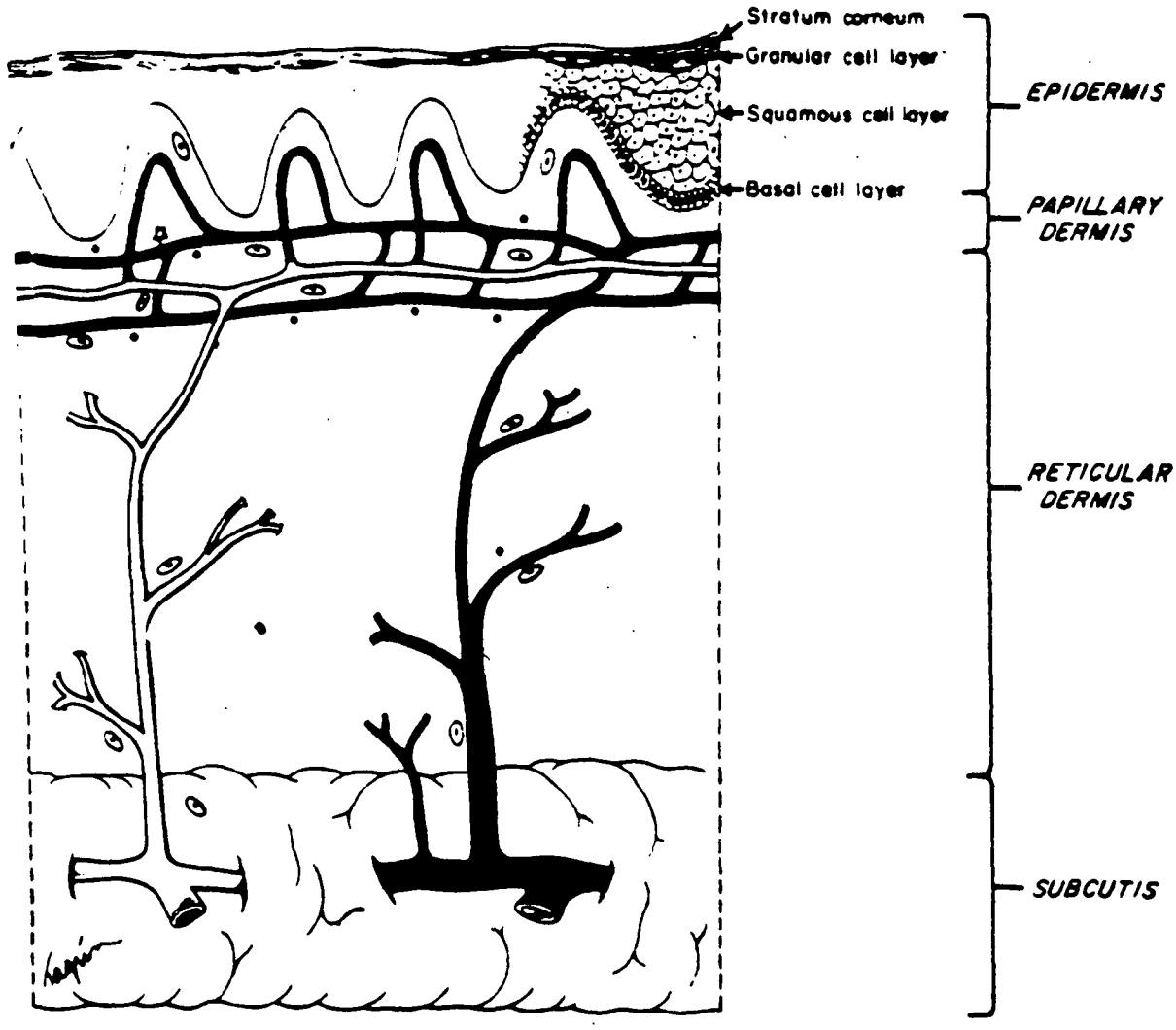
A general understanding of the biology and photobiology of the skin provides a useful background against which to examine the association between solar radiation and nonmelanoma skin tumors.

Skin Biology

There are three principal layers of skin: the epidermis, the dermis, and the panniculus adiposus (Exhibit 7-1). These layers vary in thickness depending on their location. The epidermis normally consists of about 6-10 cell layers and is typically 60 µm-100 µm thick. The thinner epidermis is found on the head, trunk, and upper limbs and the thicker epidermis is found on the lower limbs. The epidermis on the palms and soles is as much as ten times thicker than that on the head and trunk. The stratum corneum is the outermost, more dense layer of the epidermis and generally comprises between 8 µm-15 µm of the epidermis' thickness (Pearl 1984; Fitzpatrick and Soter 1985). The dermis is typically between 1700 µm and 2000 µm thick and the subcutaneous layer, the panniculus adiposus, is typically 4000 µm-9000 µm thick (Fitzpatrick and Soter 1985).

EXHIBIT 7-1

Organization of the Adult Skin



Source: Fitzpatrick and Soter (1985).

The epidermis is principally a mixture of three cell types of different embryonic origin and function; these are (in order of percent composition) the keratinocyte (80 percent), the melanocyte (5-10 percent) and the Langerhans cell (5-10 percent). The dermis is composed mainly of connective tissue fibers. These fibers, which are secreted by cells called fibroblasts, are responsible for the skin's resilience and elasticity. Many skin changes associated with aging are caused by the impact of solar radiation on these dermal fibers. The subcutaneous layer is a specialized layer of connective tissue that functions as a cushion between the bone and the epidermis and dermis. It consists primarily of fat cells.

Melanin is a pigment that absorbs light in the broad range of 250 nm-1200 nm; however, its absorption increases steadily towards the shorter wavelengths (Anderson 1983). It is synthesized in melanocytes from tyrosine and deposited in a protein matrix in organelles termed melanosomes (Romsdahl and Cox 1976). Melanosomes, in turn, are eventually destributed to keratinocytes which are vertically arranged in the skin. Melanin is a protein containing aromatic amino acids, urocanic acid, carotenoids (in the stratum corneum only), and nucleic acids. In humans and other mammals there are two predominant forms of melanin: eumelanin, which is brown or black, and phaeomelanin, which is yellow or auburn (the pigment responsible for red hair). The melanosomes containing these two types of melanin differ structurally in that those with phaeomelanin are round and have a protein matrix with a "tangled" appearance whereas eumelanin-containing melanosomes are round and have a characteristic lamellar structure (Norlund 1981).

It is the degree of melanosome aggregation and their number which determine skin color. Melanosomes in keratinocytes from light-skinned individuals are characteristically found as aggregates, whereas melanosomes in keratinocytes from dark-skinned individuals, because they are larger, generally occur singly. Extensive studies have indicated nonsignificant differences in the number of melanocytes in various racial groups, although there are variations by anatomic region. The highest concentration of melanocytes is in the cheek (2,310 $\pm$ 150 melanocytes/mm^2) and the lowest is in the back and thigh (890 $\pm$ 70/mm^2 and 1,000 $\pm$ 70/mm^2, respectively) (Briele and das Gupta 1979).

Photobiology

The interaction of sunlight with the skin is a complex process. It involves the transfer of energy from sunlight to various molecules in the skin layers and the subsequent cellular responses to this energy transfer. About 95 percent of incident radiation penetrates the skin while the remaining 5 percent is reflected by the stratum corneum. Two processes, scatter and absorption, determine the penetration of radiation into the skin. Measurements of the transmission of UVR through isolated epidermis tissue from medium complexion Caucasian skin (Bruls et al. 1984; Kaidbey et al. 1979) indicate that between 1 percent-20 percent of 295 nm UVB radiation would reach the basal layer. For 340 nm UVA radiation, as much as 20 percent-60 percent may penetrate to the basal layer (Kubitschek et al. 1986). Above 400 nm (in the visible range) transmission approaches 90 percent (Anderson 1983). The

amount of radiation reaching the basal layer is a function of the thickness of the epidermis and stratum corneum as well as their content of radiation-absorbing molecules called chromophores. There are a large number of chromophore types in the skin only one of which is melanin. Most of the optical absorbance within the skin is, however, attributable to melanin as graphically shown in Exhibit 7-2.

The skin has three response mechanisms for dealing with exposure to solar radiation: (1) dose-reduction through the production of increased melanin (tanning) or through keratinocyte hyperplasia; (2) damage repair in which the cell's DNA repair mechanisms remove photoinduced damage; and (3) cell removal.

Solar Radiation Dose Reduction

Dose-reduction via tanning may be either an immediate or a delayed response. The immediate response, sometimes referred to as immediate pigment darkening (IPD) (Pathak 1985), is due not to an increase in epidermal melanocytes or melanin but rather to changes in the melanin already present in the skin. Melanin in both melanocytes and keratinocytes evidently is oxidized. In addition, the specific distribution of melanosomes within the melanocyte is altered (Gange and Parrish 1983). The most effective wavelengths for producing IPD range from 320 nm-400 nm. The immediate response may begin within one minute following exposure, is maximal at about one hour, and is gone in about four hours.

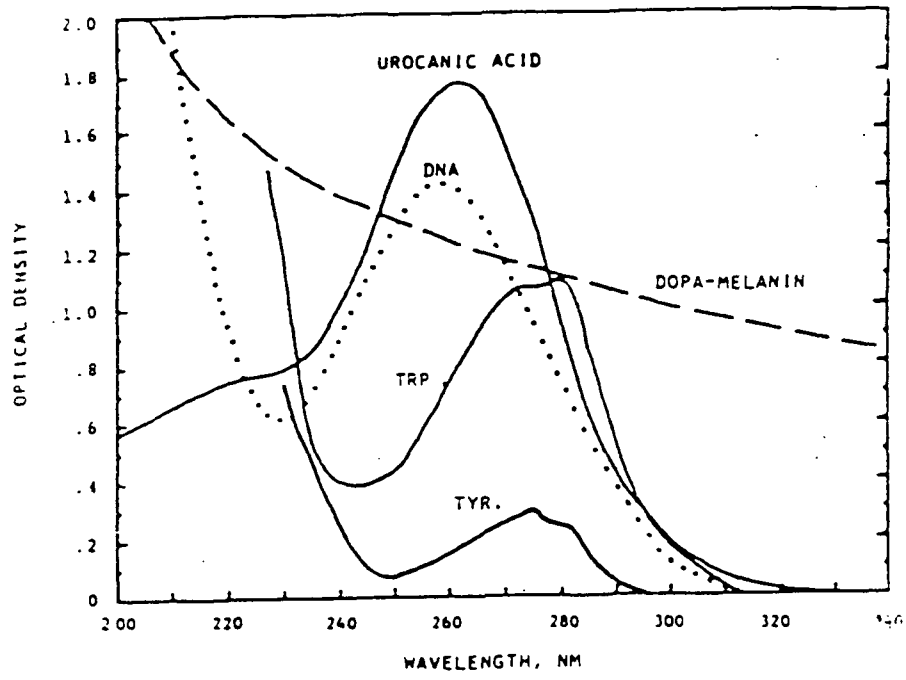
The delayed response involves the de novo synthesis of new melanosomes that are subsequently transported to keratinocytes where they produce a tan within about 10 hours after exposure. This response may continue for several days with the maximal tan being achieved in about one week. With time the tan wears off as the keratinocytes containing the extra pigment are sloughed off. In addition to the increased production of melanosomes, the number of active melanocytes in the skin also increases probably as a result of increased mitosis and recruitment of dormant melanocytes. The delayed tanning response is induced predominantly by wavelengths in the UVB range, although UVC, UVA and visible light can also induce the response.

There is a wide variation in the actual tanning response of individuals to sunlight exposure. Skin tanning responses have been generally classified into six skin types categories as outlined in Exhibit 7-3 (Pathak 1985).

The remaining dose-reduction response involves the increased production of keratinocytes and a subsequent thickening of the epidermis (keratinocyte hyperplasia). Studies in humans have shown that after a single exposure to UVB, a sustained increase in epidermal mitoses occurs leading to increases in the thickness of the epidermis and stratum corneum of between 1.5- and 3-fold over the course of one to three weeks. The UV dose first induces a transient depression in macromolecular synthesis in which DNA, RNA and protein synthesis are markedly reduced and then elevated. The response is maximal within 24 to 48 hours after irradiation but may continue for as long as one week. UVB and UVC are the most effective wavelengths for inducing this response. Keratinocyte hyperplasia protects individuals with a poor tanning ability

EXHIBIT 7-2

Ultraviolet Absorption Spectra of Major Epidermal Chromophores



Experimental Conditions:

DOPA-melanin, 1.5 mg% in H_2O;

urocanic acid, 10^{-4} M in H_2O;

calf thymus DNA, 10 mg% in H_2O (pH 4.5);

tryptophan (TRP), 2×10^{-4} M (pH 7);

tyrosine (TYR), 2×10^{-10} M (pH 7).

Source: Anderson (1983).

EXHIBIT 7-3

Skin Types and Skin Tanning Responses

Skin Type	Skin Tanning Response
I	Very sensitive. Tans little or not at all. Usually has red/blond hair, blue eyes, freckled skin. May develop severe sunburn after one hour of sun exposure in summer. MED: 15 mJ/cm^2-30 mJ/cm^2. No IPD response.
II	Sunburns frequently, tans poorly. MED: 20 mJ/cm^2-35 mJ/cm^2. Weak IPD response.
III	Burns first and tans later. Has IPD response.
IV	Lightly tanned skin. Dark eye and hair color. In summer, skin color changes from light brown to olive or medium brown. Strong IPD response.
V	Usually dark brown or black eye and hair color. Acquires deep tan. Intense IPD response.
VI	Darkly pigmented, e.g., African and American Blacks.

IPD = Immediate Pigment Darkening

MED = Minimal Erythemal Dose

because the disulfide rich keratin synthesized by the keratinocytes absorbs photons in the UVC and UVB ranges. Among lightly pigmented individuals, this skin thickening is probably the most important dose-reducing response whereas among dark-skinned individuals, tanning is the more important response (Gange and Parrish 1983).

DNA Repair Mechanisms

A different response mechanism for dealing with exposure to solar radiation is DNA repair. UV radiation can either directly or indirectly damage DNA. Direct damage results from the absorption of UVR by DNA and the subsequent formation of DNA lesions. The most significant form of direct damage is the development of cyclobutyl pyrimidine dimers (Spikes 1983). These dimers are formed between adjacent pyrimidines on the same DNA strand, and their presence renders the phosphodiester bond joining the deoxyribose moieties resistant to nuclease digestion. Indirect DNA damage is mediated by a reactive oxygen species, such as a superoxide radical, which absorbs radiative energy and transfers it to the DNA. UVB principally inflicts direct damage on DNA, but can also cause indirect damage at some wavelengths. UVA or UVC can inflict direct or indirect DNA damage on cells. DNA has an absorption maximum at 265 nm (UVC).

When DNA is damaged, the lesions may cause cell death or may merely disturb DNA transcription and replication. Based on the somatic mutation theory of carcinogenesis, these lesions may also result in mutations that subsequently result in neoplastic transformation. DNA damage that adversely affects cell function and survival needs to be repaired in order to assure continuation of a species.

The cell possesses three DNA repair mechanisms to respond to UV-induced damage: photoreactivation, excision repair, and postreplication gap repair. Repair mechanisms that result in unchanged DNA are called "error-free," whereas mechanisms that generate altered segments of DNA are generally called "error-prone." The inaccuracy of a repair mechanism is likely to result in secondary structural changes in DNA, some of which may lead to mutations.

Photoreactivation repair of pyrimidine dimers occurs when an enzyme binds to the dimer forming an enzyme-substrate complex. The enzyme-substrate complex absorbs photons of UVA light and the dimer is then monomerized (Spikes 1983). Photoreactivation is an error free, nonmutagenic repair pathway which offers a number of advantages to the cell. It uses UVA photons as an energy source. There is no incision into the DNA phosphodiester backbone and therefore no risk of DNA degradation. Additionally, there is no polymerization, and thus, no chance for the introduction of coding errors (Spikes 1983).

Another efficient DNA repair mechanism is called excision repair. Excision repair works more slowly than photoreactivation but may repair not only dimers but also other kinds of damage, e.g., methylations. In the case of dimers, excision repair involves the activity of three enzymes, an endonuclease specific for dimers, a polymerase, and a ligase. The

endonuclease attacks the DNA at or adjacent to the dimer and introduces a nick in the DNA. The DNA polymerase removes the damaged DNA segments while utilizing the opposite strand as a template for new synthesis, and finally a ligase joins the newly synthesized DNA to the preexisting strand. Other kinds of DNA damage such as methylations require different first enzymes but the activities of the polymerase and the ligase remain the same (Spikes 1983).

The third cell repair mechanism is termed post replication gap repair. This mechanism is invoked when, following DNA replication, there are gaps left in the DNA opposite the dimer. The cell invokes a DNA recombinational mechanism which provides a good copy of the DNA needed to repair the damage. Using this information, the cell can repair the damage with an excision repair mechanism (Spikes 1983). This mechanism is, however, fairly error prone.

Biology of Nonmelanoma Skin Tumors

Although most skin cancer statistics combine both BCC and SCC cell types, the limited data available from the U.S. and several other countries indicate differences in a number of characteristics. Basal cell carcinomas are neoplasms of the germinal layers of the epidermis and the appendages that differentiate toward glandular structures (Scotto and Fraumeni 1982). As a rule these tumors are slow growing and follow a relatively benign course, although on rare occasions they may result in extreme morbidity, mutilation, or if they metastasize, death (Pollack et al. 1982).

Basal cell carcinomas are believed to arise from a pluripotent epithelial cell present in the epidermis. It has been hypothesized that basal cell carcinomas arise because an abnormal interaction between these pluripotent stem cells and the surrounding connective tissue induces the cells to differentiate neoplastically (Pollack et al. 1982; Kent 1976). These tumors appear to be very stromal-dependent, however, and it has been suggested that they will rarely metastasize to a foreign tissue bed unless they take along a portion of their stroma (Pinkus 1953). This hypothesis has been confirmed in part by studies which have shown that basal cell tumor cells cultured in vitro in the absence of accompanying connective tissue convert to a keratinizing epithelium (Flaxman 1972; Kubilus et al. 1980). It has also been shown that autotransplants fail if the cells are transferred without stroma (Epstein et al. 1984).

Squamous cell carcinomas are neoplasms of the epidermis that differentiate towards keratin formation (Scotto and Fraumeni 1982). SCCs are far less fastidious in their growth requirements in vitro and will grow under a variety of conditions. They are generally more aggressive than BCCs and account for about three-fourths to four-fifths of the deaths attributable to nonmelanoma skin cancer (Dunn et al. 1965).

Cellular Studies

Solar radiation and its component UV and visible wavelengths have been observed to cause cellular changes, such as the induction of cell division and/or differentiation, the loss of specialized functions, mutation,

transformation, and death. Evidence from cellular and molecular studies clearly indicate that UVR can cause transformation in mammalian cells in the absence of any confounding immunological, hormonal, or physiological effects encountered in vivo. Shorter UVB wavelengths are generally more effective in transforming mammalian cells. In addition, the experiments conducted in vitro may be used to extrapolate effects in vivo.

As already described, a single in vivo exposure of human skin to UVB radiation can cause the epidermis to thicken, thereby increasing the tolerance of skin to subsequent radiation. UVB radiation exposure also induces melanocyte division (Rosdahl and Szabo 1978). Some investigators studying the response of melanocytes in trunk epidermis of black mice to UVR have concluded, however, that the increased number of active melanocytes results from both the proliferation and recruitment of amelanogenic melanocytes (Miyazaki et al. 1974).

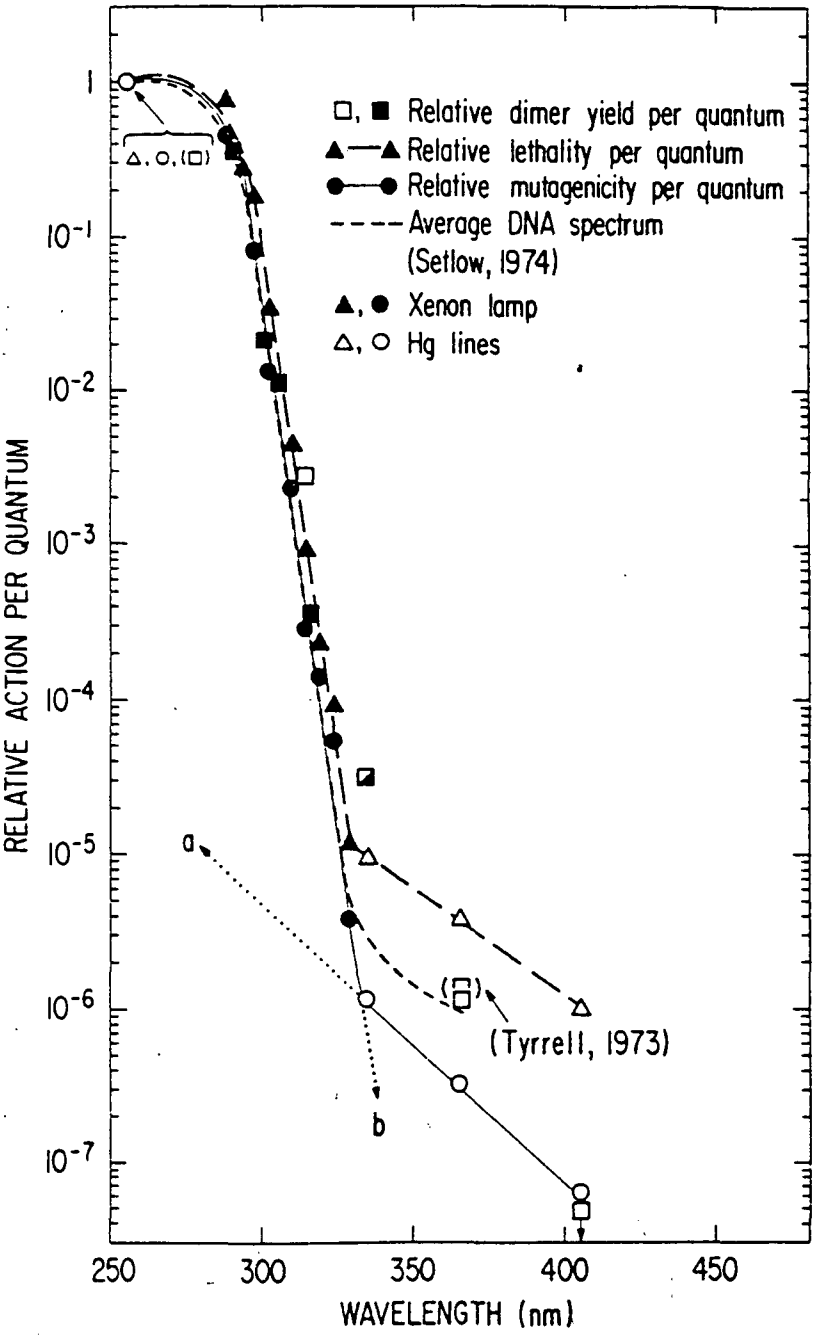
Exposure to UVR has also been found to result in the impairment of antigen presenting cell function in both the mouse and man (Greene et al. 1979). In animals with primary UVR induced tumors, this impairment results in the generation of UV-tumor-antigen specific suppressor lymphocytes (Kripke et al. 1977; Daynes et al. 1977).

Ultraviolet light is mutagenic in both bacterial and mammalian cells. The importance of mutation to an assessment of the role of UVB in nonmelanoma skin cancer development lies in a theory of carcinogenesis which suggests that somatic mutation in mammalian cells is the first step down a pathway which includes malignant transformation and ends in neoplasia and metastasis (Trosko and Chu 1975). In UVR-treated bacteria, researchers have concluded that pyrimidine dimers are responsible for much of the observed mutagenesis (Hall and Mount 1981). UVR treated cells, if subsequently treated with photoreactivating light, show a substantially decreased frequency of mutants per survivor compared to UVR-treated cells without subsequent photoreactivation treatment. Peak et al. (1984) derived action spectra for DNA dimer induction, lethality, and mutagenesis in E. coli over wavelengths from 254 nm-405 nm (Exhibit 7-4) and found that all three endpoints decreased in efficiency in a similar fashion as the wavelengths of radiation increased. From 300 nm-320 nm, all characteristics showed differences of about 2.5 orders of magnitude. Furthermore between about 250 nm and 320 nm, the values for the three endpoints either coincided with or closely paralleled Setlow's (1974) proposed average DNA action spectrum.

Experiments have also been performed using fibroblasts from xeroderma pigmentosum (XP) patients. These patients have a genetic inability to repair UV-induced pyrimidine dimers. Setlow et al. (1969) compared the responses of XP fibroblasts to normal human fibroblasts upon irradiation to UVR. They observed that normal human fibroblasts which were UVR irradiated, then stimulated to undergo cell division by immediate replating, showed much higher numbers of mutations than similarly irradiated normal cells which were incubated for seven days as nondividing monolayer cultures. These results indicated that normal human cells can repair the damage induced by UVR (presumably by excision repair). XP cells, by comparison, produced a

EXHIBIT 7-4

Ultraviolet Action Spectra for DNA Dimer Induction



Source: Peak et al. (1984).

comparable number of mutants whether replated immediately or given seven days of incubation. Since XP cells are unable to excise pyrimidine dimers, these experiments suggest that unexcised dimers present at the time of cell division may have been responsible for the production of mutations.

In vitro transformation is thought to be correlated, albeit imperfectly, to in vivo tumorigenesis (Heidelberg 1977). As such, it has been used extensively to characterize the potential carcinogenicity of a wide variety of chemicals and physical agents. Cells which have been induced in vitro to lose certain normal growth controls are frequently, although not always, tumorigenic in mice. A hierarchy of transformational changes is recognized and the ability of cells to grow without attachment to a solid substrate (loss of anchorage dependence) is generally accepted as the best correlate to tumorigenicity (Freedman and Shin 1974).

Doniger et al. (1981) developed action spectra for transformation, lethality, and thymine dimer formation using a monochromatic light source. In dose-response studies comparing pyrimidine dimer formation and transformation of Syrian hamster embryo cells, the slopes of the dose-response curves were not always parallel. The discordance was greatest at 290 nm. The lowest exposure required for equivalent cell transformation, lethality, and pyrimidine dimer formation was at 270 nm. Comparing results at 290 nm, 297 nm, and 302 nm, the respective doses required were 2.3 J/m^2, 8.7 J/m^2, and 25 J/m^2 for dimer formation and 7.4 J/m^2, 47 J/m^2, and 97 J/m^2 for transformation. A similar spectrum was found for induction of anchorage independent growth in human fibroblasts (Sutherland et al. 1981). These authors also found a maximum effectiveness at 265 nm and that transformation at 290 nm was six times more effective per photon than that at 297 nm.

Molecular Studies: DNA and UV Radiation

Chemical changes and biological damage induced by ultraviolet light require the absorption of light energy (photons) by molecules within the target. Each type of molecule is capable of absorbing radiation only in specific wavelength ranges. Examination of the absorption spectra of molecules in biological systems indicates that a number of biomolecules can absorb radiation in the 220 nm to 400 nm region and thus may be critical targets for detrimental UV effects (Spikes 1979). This section will review information only on the most important molecule, DNA, because of its putative role in mutagenesis, transformation and carcinogenesis.

As already mentioned, two possible types of mechanisms can induce DNA damage -- a direct mechanism resulting from absorption of energy by DNA and an indirect mechanism involving reactive oxygen species. A number of different lesions may be induced in DNA by UV irradiation. These include (1) pyrimidine dimers, (2) pyrimidine adducts, (3) single strand breaks, (4) double strand breaks and (5) protein-DNA crosslinks. Different wavelengths have different efficiencies for the production of these lesions.

Pyrimidine Dimers and Adducts

As described in an earlier section, pyrimidine dimers may be formed by direct absorption of photons in the UVB wavelength range. Adjacent pyrimidine molecules on the same strand of DNA become linked together by a cyclobutane ring between the 5 and 6 carbon atoms of each residue. Thymine dimers are the most likely pyrimidine dimers because the excited state of thymine is a much lower energy state than that of cytosine . Cytosine co-dimers are also possible, as are heterodimers between thymine and cytosine or uracil (Spikes 1982).

Action spectra for four-membered ring pyrimidine adduct formation between two consecutive bases on the same strand of DNA resemble the action spectra for pyrimidine dimer formation (Patrick and Rahn 1976). The efficiency of formation of these photoproducts is about two to ten times lower than for cytosine-cytosine and thymine-thymine dimers. The proportion of these adducts varies according to the base content of DNA and becomes much higher when the ratio of guanine-cytosine/adenine-thymine is greater than or equal to one.

Thymine glycols, another form of pyrimidine adduct, are defined as a group of ring saturated lesions of the 5,6-dihydroxydihydrothymine type and have been detected in the DNA of human cells after irradiation at 254 nm and 313 nm (Hariharan and Cerruti 1977). The formation of these lesions is probably caused by the action of hydroxyl radicals on the 5-6 double bond of thymine. In both the UVB and UVA range, thymine glycols may result from the action of reactive oxygen species produced by endogenous sensitizers.

Thymine glycol lesions occur with almost the same frequency as thymine dimers at 313 nm, indicating their possible significance in the UVB range (Cerruti and Netrawali 1979). Glycol lesions may undergo spontaneous decay to form apyrimidinic sites in a fashion similar to that described for gamma-radiation produced saturated thymine glycols (Dunlap and Cerrutti 1975). Only about one third of the thymine glycols are released from the DNA backbone, but there is little evidence to suggest that either the ring saturated thymine or the apyrimidinic decay products are lethal lesions in UV-irradiated DNA.

Brash and Haseltine (1982) have shown that there is a linear relationship between base damage incidence and mutation incidence. For shorter wavelengths (UVB) it seems clear that pyrimidine dimers and 64 photoadducts are involved in mutagenesis, but at 365 nm the correlation between dimers or adducts and mutagenesis is not known (Peak et al. 1984).

DNA Single-Strand Breaks

DNA single-strand breaks can be induced directly by UVB radiation or indirectly by UVA and visible light. Analysis of the relative efficiencies for the induction of single-strand breaks reveals an action spectrum that corresponds with nucleic acid absorption at or below 313 nm (Peak and Peak 1986a). Above 313 nm to wavelengths as long as 546 nm, single-strand breaks have been detected in human fibroblasts with an action spectrum in the visible range resembling that of riboflavin (Tyrell 1984). Relative to thymine

dimers, single-strand breaks are induced only to a small extent by 254 nm radiation, but as the wavelength is increased the proportion of single-strand breaks to thymine dimers increases. At 313 nm in E. coli, one single-strand break is induced for every nine thymine dimers (Cerrutti and Netrawalli 1979). Some single-strand breaks induced at 313 nm may be due to indirect effects from photosensitizers and oxygen dependent mechanisms (Miguel and Tyrell 1983), but the spectral analysis indicates that the majority of single-strand breaks induced at this wavelength are due to direct effects (Peak and Peak 1986a).

DNA Double-Strand Breaks

DNA double-strand breaks occur about 80 times less frequently than single-strand breaks at 313 nm in bacteria (Tyrrell 1984). Both single- and double-strand breaks are resealed within one hour in both prokauyotes and eukauyotes. Two repair processes, one slow and the other fast, repair damage induced in E. coli by ionizing radiation. There is some evidence that repair of breaks induced at 313 nm may proceed in a similar fashion (Tyrell 1984).

DNA-Protein Crosslinks

DNA-protein crosslinks can be induced by borderline UVB radiation (290 nm) via a direct photon-absorbing mechanism in human cellular DNA. Below 320 nm, there are approximately 40 DNA-protein crosslinks per lethal event. As cells can survive $2x10^3$ DNA-protein crosslinks induced at longer wavelengths (405 nm), it appears that such a small number of DNA-protein crosslinks is not important in UV-induced cell lethality. This assumes, however, that there are no interactions between dimers and DNA-protein crosslinks (Peak et al. 1985b).

DNA-protein crosslinking is demonstrable in normal human fibroblasts immediately after UV irradiation, but this crosslinking is partially reversed after about 12 hours. In fibroblasts from XP patients, crosslinking after UV-exposure was not reversed and actually progressed with time. This is possibly a secondary change due to severe cell damage (Fornace and Kohn 1976). The abnormal sensitivity of XP cells to UVR has generally been attributed to be a defective capacity to repair cyclobutylpyrimidine dimers in cellular DNA (Smith and Paterson 1981). The inability of XP cells to repair DNA crosslinks suggests, however, that the lethality of UVR to XP cells may not be fully explained by their inability to excise dimers but may also relate to the persistence of DNA-protein crosslinks.

Although the biological significance of DNA-protein crosslinks are not clear, it would seem that these lesions are not lethal to the cell. In normal cells the number of DNA-protein crosslinks per genome per lethal hit is greater than 900 (Peak and Peak 1986a). It can reasonably be concluded, however, that normal cells possess an ability to repair these lesions.

Animal Studies

Observations that outdoor workers had a greater tendency to develop skin cancer promoted Findlay (1928) to examine UV-induced carconogenesis in mice. Findlay reported that not only did UVR alone induce skin tumors in mice, but tumors induced by topically applied tar appeared more rapidly if those mice were subsequently exposed to UVR. Since Findlay's early works, researchers have found that tumors induced in mice by UVR are primarly squamous cell carcinomas and fibrosarcomas (Epstein and Epstein 1962; Hsu et al. 1975; Kligman and Kligman 1981; Kripke 1977; Spikes et al. 1977; Stenback 1975; Strickland et al. 1979; Winkelman et al. 1963) and are mosly monoclonal in origin (Burnham et al. 1986). There is no animal model for basal cell carcinomas.

A number of studies have attempted to identify specifically those UV wavelengths responsible for the observed carcinogenicity with varying results. Early studies used filters to remove shorter wavelengths from broadband UVR so that the effect of different wavelengths could be studied. In some of the original studies on the carcinogenic action spectrum of UVR, Rusch et al. (1943) reported that the carcinogenic wavelengths were between 290 nm and 334 nm; wavelenths greater than 334 nm and those at 254 nm were not found to be carcinogenic. Blum (1943), in contrast, concluded from his experiments that the most effective wavelengths for producing tumors in mice were between 260 nm and 300 nm.

Freeman (1975), using monochromatic UVR, exposed the ears of albino mice to a weekly dose of 420 J/m^2 at 290 nm, 600 J/m^2 at 300 nm, 7500 J/m^2 at 310 nm, and 49,500 J/m^2 at 320 nm. Freeman (1975) found that mice exposed to 290 nm developed no tumors, those exposed to 300 nm and 310 nm developed tumors with the same median latent period, and those exposed to 320 nm developed fewer tumors. When the mice were exposed to the same incident radiation at 300 nm and 310 nm, only the mice given radiation at 300 nm developed tumors. This result indicated that UVR at 300 nm is a more potent carcinogen than at 310 nm.

In a more recent study, Cole et al. (1986) investigated the ability of wavelength weighting schemes based on three different action spectra to predict the tumorigenicity of UVR. Use of a weighting scheme for an averaged spectrum based on DNA damage overestimated the importance of the shorter wavelengths in inducing tumors. Use of an action spectrum based on the sensitivity of the Robertson-Berger (RB) meter underestimated the contribution of the shorter wavelengths. These authors found that the best predictor of UVR effectiveness at inducing tumorigenesis in hairless mice was an action spectra based on the induction of mouse edema 48 hours after a single acute dose.

There has been a great deal of experimentation to determine if there is dose-rate reciprocity in UV carcinogenesis. Blum et al. (1942) studied the effect of the intensity of the dose on the tumor latency period and reported that for a 10-fold dose range of between 4.3 J/m^2/sec and 0.42 J/m^2/sec, no significant differences were found. In a second study using a wider range

of intensities and greater numbers of animals he reported that above 0.4 $J/m^2/sec$ the effectiveness of UVR in inducing tumors was almost independent of dose-rate, but below 0.4 $J/m^2/sec$ the effectiveness fell off rapidly with intensity.

To determine if the dose-rate affects tumors development, the amount of energy necessary to produce tumors in 50 percent the tested animals can also be calculated. De Gruijl et al. (1983) stated that the total dose delivered to hairless mice to induce tumors must be greater if a high daily dose is given, than if a low daily dose is given. The greater dose given to the mice was 9.4×10^3 $J/m^2/wk$. This same trend was reported by Spikes et al. (1977) in C3H mice.

Experiments on photocarcinogenesis in laboratory animals may shed light on the role of UVR in the development of human nonminanoma skin tumnors. Results from animal studies clearly show that UVR is carcinogenic and that UVB wavelengths (290 nm-320 nm) are most effective in inducing a response.

Immunosuppression

The risk of developing nonmelanoma skin tumors may be influenced by the immune status of a UVR-exposed animal or human. UVR itself is immunosuppressive (Kripke et al. 1977; Spellman et al. 1977), inducing the production of a class of lymphocytes, T suppressor cells, which depress the host's ability to respond to UVR-induced tumor antigens (Fisher and Kripke 1977, 1978; Spellman and Daynes 1977, 1978; Daynes et al. 1979).

The T-suppressor cell is thought to prevent the generation of the cytotoxic ("T-killer") lymphocytes (Romerdahl and Kripke 1986) which would normally kill UVR-induced tumor cells, having recognized them as foreign by the new antigens present on their surfaces (Kripke 1977). Other forms of immunosuppression, e.g., via administration of an antilymphocyte serum or 6-mercaptopurine, give conflicting results, sometimes enhancing and sometimes depressing photocarcinogenesis (Nathanson et al. 1976). (A more detailed discusion of this is presented in Chapter 9.)

Among systemically immunosuppressed individuals, such as kidney transplant patients, the incidence and aggressiveness of nonmelanoma skin cancer may be increased. The available data on the increased risk among immunosuppressed patients are generally more convincing for SCC than for BCC (Pollack et al. 1982).

EPIDEMIOLOGICAL EVIDENCE

Occurrence and Trends

Nonmelanoma skin tumors are among the most common malignant neoplasms occurring in white populations. Based on a one-year survey (1977-1978) conducted by the National Cancer Institute (NCI), it was determined that nonmelanoma skin cancers developed in approximately 400,000 white Americans each year (Scotto and Fraumeni 1982). The annual age-adjusted incidence rate

for this survey period was estimated to be $232.6/10^5$ among whites. For comparison, the estimated incidence rate for all other cancers among whites in the United States based on 1973-1976 data from the Surveillance, Epidemiology and End Results (SEER) Program was $318.9/10^5$ (NCI 1985). Among blacks, the annual age-adjusted incidence rates for nonmelanoma skin tumors and all other cancers (NCI 1985) were $3.4/10^5$ and $347.3/10^5$, respectively.

The incidence of BCC is generally several times greater than the incidence of SCC. For example, based on data collected for 62 skin cancer cases registered from 1956-1960 in three public hospitals in New Zealand, Eascott (1963) observed that 73 percent of the cases had BCC, 15 percent had SCC, and 7 percent had cutaneous malignant melanoma (CMM). Lee (1982) presented data on 2,019 skin cancer cases in Switzerland for 1974-1978 and showed that approximately 69 percent of the cases had BCC, 20 percent had SCC, and 11 percent had CMM. Among 2,000 individuals whose head, neck, forearms and dorsa of hands were examined in a survey in Alfred Hospital in Victoria from 1982-1983, 71 percent of the histologically confirmed skin tumors were BCC and 29 percent were SCC (Goodman et al. 1984). A similar distribution of SCC and BCC was observed in a one week study of 2,113 adults in 1982 in Maryborough, Australia whose head, neck, forearms, and dorsa of hands were examined (Marks et al. 1983).

These relative differences in BCC and SCC (and CMM) persist when males and females are examined separately even though the incidence in males generally exceeds that in females. Exhibit 7-5 presents age-adjusted BCC and SCC incidence data for white American males and females (Scotto and Fraumeni 1982). The incidence of BCC was approximately four to six times greater than the incidence of SCC.

The overall case fatality rate for nonmelanoma skin tumors is approximately 1 percent (Epstein et al. 1984). SCCs are, however, generally more aggresive than BCCs, accounting for about three-fourths to four-fifths of the deaths attributable to nonmelanoma skin cancer (Dunn et al. 1985; NAS 1982). SCCs may metastasize shortly after their appearance or even at the time of diagnosis (Epstein 1983). The rate of metastasis among SCC patients has been estimated by several researchers to range between 2 percent and 20 percent. Epstein (1983) reported that 25 percent of SCC patients with metastisis were alive five years after the diagnosis, 13 percent were alive 10 years after diagnosis, and only 8 percent were alive after 15 years. Approximatley two-thirds of metastasizing SCCs were observed to occur on the face and dorsa of the hands. BCCs, in contrast, rarely metastasize even when present for many years (Pollack et al. 1982) Their aggressiveness may be correlated with the site of tumor origin. According to Pollack et al. (1982), only about 100 cases of metastatic BCC have been reported.

Time Trends

Several researchers have observed that the incidence of BCC and SCC have been increasing over the past several decades (Lee 1982, NAS 1982). Epstein et al. (1984) has pointed out that the rate of increase of SCC is greater than that of BCC. Scotto and Fraumeni (1982), however, noted when comparing the

EXHIBIT 7-5

Comparison of Age-Adjusted Incidence Rates Per 100,000 for Squamous Cell Carcinoma (SCC) and Basal Cell Carcinoma (BCC) Among White Males and Females in the United States

Skin Tumor Type	Age-Adjusted Incidence Rate/10^5	
	Males	Females
SCC	65.4	23.6
BCC	246.6	150.1

Source: Scotto and Fraumeni (1982).

NCI 1971-1972 and 1977-1978 survey data that the observed incidence increases applied mainly to BCC. The authors noted that the incidence of BCC among United States whites increased by approximately 15 percent-20 percent over the six-year period between surveys.

In general, older individuals (e.g., over 60 years of age) are at higher risk of developing nonmelanoma skin tumors than are younger individuals. Vitaliano and Urbach (1980) observed that only three percent of a total 424 BCC and SCC cases from the Tumor Clinic of the Skin and Cancer Hospital in Philadelphia were under 40 years of age. However, Emmett (1982) and Harris (1982) have observed that SCC and BCC are no longer only diseases of old age since an increasing number of younger individuals have been presenting with nonmelanoma skin tumors. Harris (1982) suggested that the occurrence of nonmelanoma skin tumors among younger individuals was consistent with increased sunlight exposure among these age groups.

The age-specific incidence patterns for SCC and BCC are not completely identical, suggesting that different etiological mechanisms may exist. Although incidence rates for SCC and BCC have been reported to rise with age and level off at the oldest age groups (Scotto and Fraumeni 1982), the increase with age has been observed to be sharper for SCC than BCC. Laerum and Iversen (1981) summarized study results indicating that among a group of BCC cases, 15 percent were less than 50 years of age and 65 percent were less than 70 years of age. Among a group of SCC cases, in contrast, Laerum and Iversen (1981) observed that 70 percent were over 70 years of age.

Anatomical Distribution

The predominant anatomical sites for both SCC and BCC and for both males and females are generally the face, head, and neck. Exhibit 7-6 presents the distribution of BCC and SCC by sex for tumors occurring in whites in the United States (Scotto and Fraumeni 1982). The data were collected as part of the 1977-1978 NCI survey. The face, head, and neck accounted for 60 percent or more of the total nonmelanoma skin tumors. Most of the remaining BCCs occurred on the trunk whereas most of the remaining SCCs occurred on the upper extremities. Skin tumor data from 1974-1978 from Switzerland in Exhibit 7-7 shows similar tumor site distributions (Lee 1982). Scotto and Fraumeni (1982) noted that the tendency for nonmelanoma skin tumors to develop in exposed areas was consistent with the belief that solar radiation is a dominant risk factor for nonmelanoma.

There are, however, some general differences in the distributions of SCC and BCC which suggest that these nonmelanoma skin tumors may respond differently to different dosages of sun exposure. The available epidemiological data generally show that BCC is more likely to develop on regularly unexposed sites compared with SCC. Pollack et al. (1982) noted that although it is well-established that sun-exposed areas are more prone to BCC, in at least one study Urbach et al. (1972), approximately one-third of the BCCs occurred in light-protected regions. Laerum and Iversen (1981) observed that among a group of SCC and BCC cases, 90 percent of the BCCs and approximately 50 percent of the SCCs occurred on the head and neck. They

EXHIBIT 7-6

Percentage of Tumors by Anatomic Site for Nonmelanoma Skin Cancer Among White Males and Females in the United States (1977-1978 NCI Survey Data)

Anatomic Site	BCC		SCC	
	Male	Female	Male	Female
Face, Head and Neck	81.2	84.1	74.8	60.1
Trunk	12.0	8.9	4.5	5.3
Upper Extremities	4.9	3.4	18.1	25.8
Lower Extremities	1.3	2.9	1.3	5.7
Other Sites	0.5	0.7	1.4	3.2

Source: Scotto and Fraumeni (1982).

EXHIBIT 7-7

Distribution by Sex and Anatomic Site of Nonmelanoma Skin Tumors: Canton of Vaud, Switzerland (1974-1978)

Anatomic Site	BCC		SCC	
	Male	Female	Male	Female
Head and Neck	540 (71.2)	505 (78.7)	202 (80.5)	103 (72.5)
Trunk	130 (17.1)	76 (11.9)	8 (3.2)	11 (7.7)
Upper Limbs	14 (1.8)	17 (2.6)	26 (10.3)	19 (13.4)
Lower Limbs	15 (2.1)	18 (2.8)	5 (2.0)	7 (4.9)
Other	58 (7.6)	26 (4.0)	10 (4.0)	2 (1.4)
Total	758 (100.0)	642 (100.0)	251 (100.0)	142 (100.0)

Source: Levi and Chapallaz (1981) as cited in Lee (1982).

estimated that of these tumors, two-thirds of the BCCs occurred on sites of the head and neck receiving the highest UV radiation doses (e.g., the nose) whereas all the SCCs occurred at these sites. Hilstrom and Swanbeck (1970) examined only SCC cases and observed that about 80 percent of the SCCs occurred on the head. Facial SCCs occurred relatively equally between males and females, but of the SCCs on the external ear, 90 percent occurred among males and only 10 percent among females. Emmett (1982) examined only BCC cases and observed that 75.5 percent occurred on the head and neck, 16 percent on the limbs, and 8.4 percent on the trunk. Lee (1982) noted that although BCC and SCC tend to be concentrated on exposed sites, the distribution of BCC did not precisely correspond with sun-exposed areas.

Exposure Factors

Several exposure factors considered to be associated with nonmelanoma skin tumors have been identified in epidemiological studies including a latitudinal gradient, prolonged exposure to sunlight, and treatment with UV.

Prolonged sun exposure is considered to be the dominant risk factor for nonmelanoma skin tumors among light-skinned populations (NAS 1982; Scotto and Fraumeni 1982; Greene and O'Rourke 1985; Lee 1982; Beral and Robinson 1981). In its 1982 report, the National Academy of Sciences (NAS) wrote that experts agree that exposure to sunlight causes 90 percent or more of the BCCs and SCCs in the United States. Several observations supporting this hypothesis were cited in the NAS report (NAS 1982) and by other researchers (Scotto and Fraumeni 1982; Urbach 1983; Beral and Robinson 1981; Scotto et al. 1981; Laerum and Iversen 1982; Emmett 1982). These observations include:

- the tendency for nonmelanoma skin tumors to develop in sun-exposed sites (e.g., head, face, and neck);
- the higher incidence rates among occupational groups with outdoor exposures compared to those with indoor exposures;
- the latitudinal and UV radiation gradient showing the highest incidence rates in geographic areas of relatively high UV radiation exposure;
- the increase in incidence rates with increasing age;
- the inverse correlation between nonmelanoma skin tumor incidence and degree of skin pigmentation;
- the high risk among genetically predisposed individuals (e.g., those with xeroderma pigmentosum);
- the predisposition for nonmelanoma skin tumors to develop among light-skinned individuals who are susceptible to sunburn and who have red/blond hair, blue/light eyes, and a Celtic heritage;

- the increased incidence of SCC in individuals treated with high intensity UVB sources and oral methoxsalen or topical coal tar; and

- the capacity of UV radiation to induce nonmelanoma skin tumors in experimental animals.

The observed variations in nonmelanoma incidence by latitude in particular support an association with sun exposure. For example, Scotto and Fraumeni (1982) observed, based on the 1977-1978 NCI survey data, that the incidence of SCC and BCC in the United States showed a latitudinal gradient with higher rates in the south. These results are displayed in Exhibits 7-8 and 7-9 for white males and white females, respectively. (Also shown in these figures are 1973-1976 CMM data from the SEER program. The latitudinal gradients for CMM were least pronounced.) It should be noted, however, that only a small number of data points (as shown in Exhibits 7-8 and 7-9) were used to examine the latitudinal gradients of BCC and SCC.

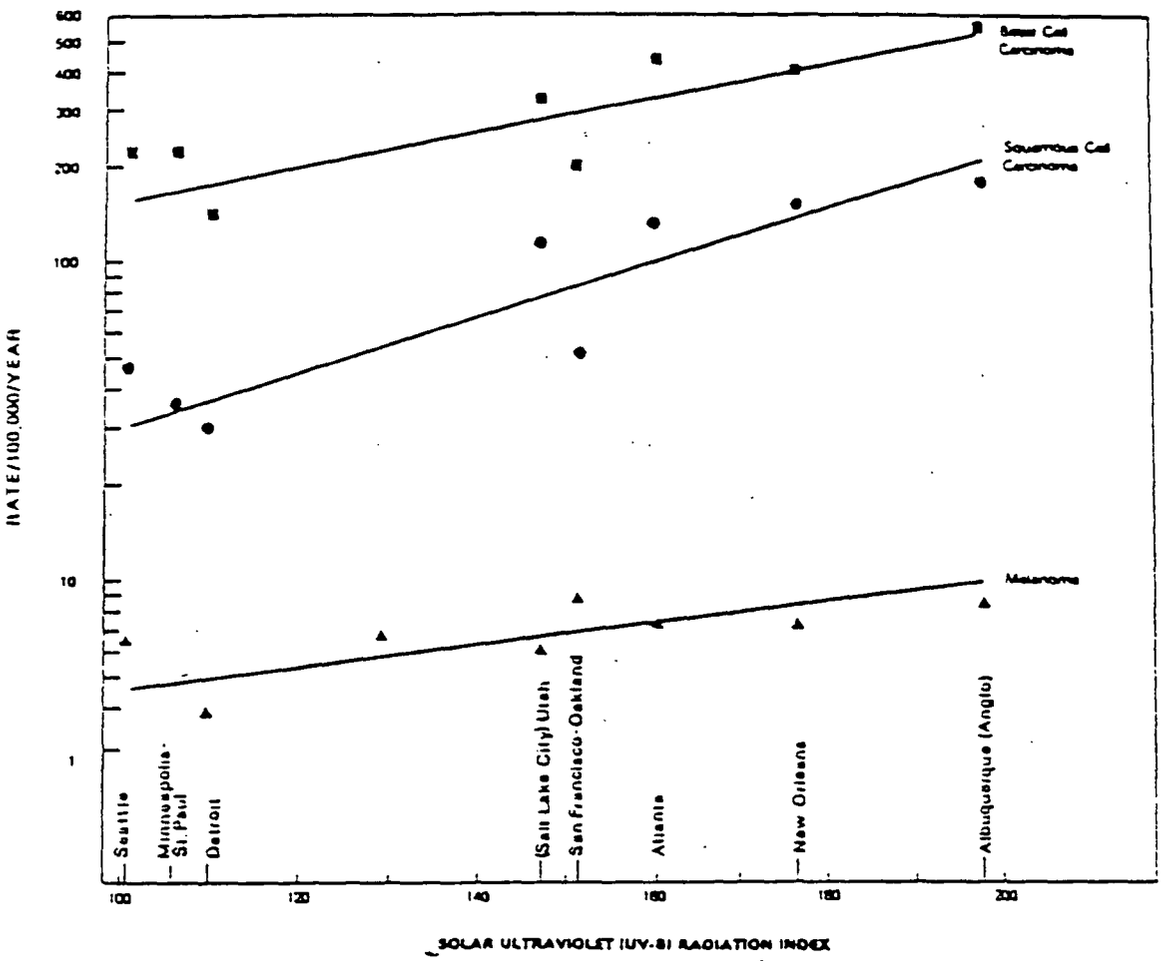
Variations in the incidence ratio of BCC to SCC by latitude again suggest that the two forms of nonmelanoma skin tumors respond differently to solar exposure. The results of MacDonald and Bubendorf (1964, as cited in Vitaliano and Urbach 1980) showed that the BCC/SCC ratio decreased from approximately 10:1 in northern United States cities to approximately 2:1 or 3:1 in southern rural areas. Similarly, Scotto and Fraumeni (1982) noted that the ratio of SCC incidence to BCC incidence increased with decreasing latitude and increasing sunlight exposure (see Exhibits 7-8 and 7-9). Pollack et al. (1982) commented that dosimetry studies revealed a poor correlation between BCC density in a site and UV radiation dose. They suggested that etiological factors for BCC other than UV radiation, such as the presence of areas of scarring or epidermal nevi, may exist. Among blacks, in whom nonmelanoma skin tumors occur rarely compared to whites, areas of trauma or scarring may be important sites for the development of SCC (Scotto and Fraumeni 1982).

Vitaliano and Urbach (1980) examined several risk factors for SCC and BCC in a case-control study. The study included 366 BCC and 58 SCC cases seen at the Tumor Clinic of the Skin and Cancer Hospital in Philadelphia (dates were not specified). A group of 294 white controls without carcinoma were selected from the skin and cancer outpatient department. Information compiled on each case and control included cumulative solar exposure based on vocational and military history, time spent sunbathing, and participation in outdoor sports (as a spectator or participant). Exposure was divided into four categories: E1=0-9,999 hours, E2=10,000-19,999 hours, E3=20,000-29,999 hours, and E4=30,000 or more hours. The host factors that were examined included complexion (pale or mild-dark), age (0-59 years or 60 and over), and ability to tan (tans or burn-sensitive).

Based on a statistical logistic regression of the case-control data, Vitaliano and Urbach (1980) identified the most important risk factors for BCC and SCC as follows:

EXHIBIT 7-8

Annual Age Adjusted Incidence Rates for Basal and Squamous Cell Carcinomas (1977-1978 NCI Survey Data) and Melanoma (1973-1976 SEER Data) Among White Males*

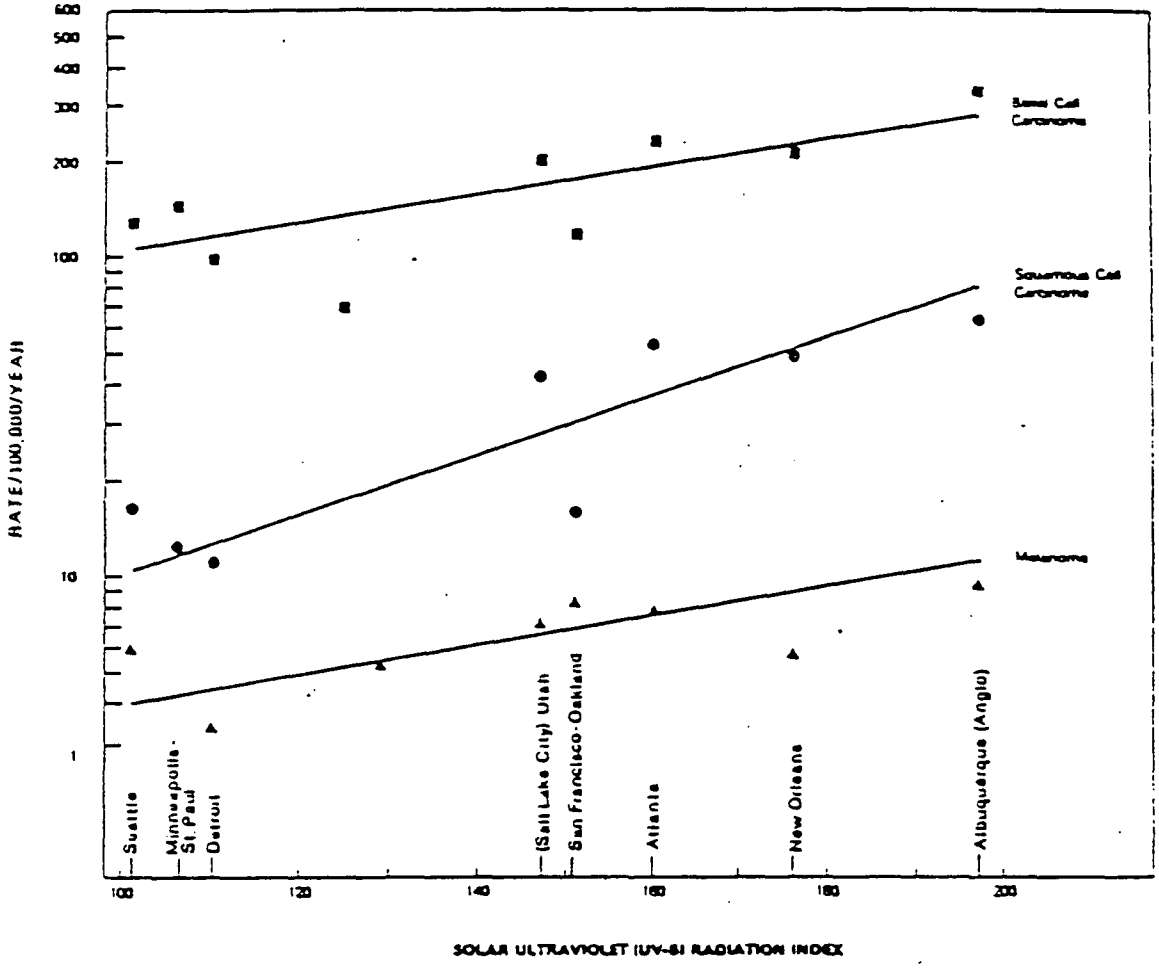


* According to annual UVB measurements at selected areas of the United States, with regression lines based on an exponential model.

Source: Scotto and Fraumeni (1982).

EXHIBIT 7-9

Annual Age Adjusted Incidence Rates for Basal and Squamous Cell Carcinomas (1977-1978 NCI Survey Data) and Melanoma (1973-1976 SEER Data) Among White Females*



* According to annual UVB measurements at selected areas of the United States, with regression lines based on an exponential model.

Source: Scotto and Fraumeni (1982).

BCC: cumulative solar exposure ($p<0.001$) >> ability to tan ($p<0.001$) >> age ($p<0.005$) >> complexion ($p<0.025$)

SCC: cumulative solar exposure ($p<0.001$) >> age ($p<0.001$) >> ability to tan ($p<0.005$).

Cumulative solar exposure was the most important risk factor for both SCC and BCC. Ability to tan was also important even at low levels of exposure. Complexion was a less important risk factor for SCC than for BCC.

Exhibit 7-10 presents the estimated relative risks (RRs) of BCC and SCC for the combinations of risk factors considered in the Vitaliano and Urbach (1980) study. Vitaliano and Urbach (1980) concluded that the most important difference between SCC and BCC was their relationship with cumulative exposure. As shown in Exhibit 7-10, a higher exposure level was required for BCC than for SCC to reach similar RRs. The authors noted that the maximum response of BCC to solar exposure occurred in exposure category E4 (30,000 or more hours) whereas for SCC it occurred in exposure category E3 (20,000-29,999 hours). They observed that the results were consistent with the belief that exposure to UV radiation has a greater effect on the development of SCC than on BCC although an association between BCC and sunlight does exist.

Additional evidence of an association between UV radiation and nonmelanoma skin tumors is provided by cohort studies of psoriasis patients treated with high intensity UVB radiation. High intensity UVB sources would be expected to damage DNA in the same way as 290 nm-300 nm solar wavelengths. The NAS (1982) report noted that in one cohort study, elevated risks were associated with UVB exposure and topical crude coal tar. Although treatment with photochemotherapy (oral methoxsalen and UVA) damages DNA in a different way than solar radiation, BCC and SCC rates in 1,373 patients were three times higher than expected (NAS 1982). In a more recent study by Stern et al. (1984) of 1,286 psoriasis patients followed-up for an average of 5.7 years, the risk of SCC 22 months after the first exposure to psoralen and UVA (PUVA) among those exposed to a high dose was 12.8 times that for those exposed to a low dose even after adjustment for exposure to ionizing radiation and topical tar preparations. No substantial dose related increase was observed for BCC. Stern et al. (1984) concluded that the results showed a substantial dose-dependent increase in the risk of SCC, even in those with neither a prior history of skin cancer nor substantial exposure to cutaneous carcinogens. The authors suggested that PUVA may act as both a co-carcinogen and an independent carcinogen in the development of SCC. In two European trials (Finland and Vienna-Innsbruck) no increased risk of skin cancer was observed among patients treated with PUVA (Stern 1984). The absence of a relationship may, however, have been due to different study methods, shorter follow-up periods, less sun or occupational exposure, and lower PUVA doses in the European patients.

Host Factors

As already mentioned, in addition to exposure factors, host factors play an important role in the risk of nonmelanoma skin tumors. Characteristics associated with pigmentation, including eye, hair, and skin color, ability to

EXHIBIT 7-10

Estimated Relative Risks of Basal and Squamous Cell Carcinomas for 32 Combinations of Risk Factors

Exposure Grade (Total Exposure, hr)	Tans				Burns-Sensitive			
	0-59 yrs		60+ yrs		0-59 yrs		60+ yrs	
	Dark	Pale	Dark	Pale	Dark	Pale	Dark	Pale
				Basal Cell Carcinoma				
E4 (30,000 or more)	3.19	4.94	4.99	7.76	6.10	9.43	9.57	14.80
E3 (20,000-29,999)	2.86	4.43	4.49	6.95	5.47	8.47	8.58	13.29
E2 (10,000-19,999)	1.77	2.75	2.79	4.32	3.39	5.26	5.32	8.25
E1 (0-9,999)	1.00	1.55	1.57	2.43	1.91	2.96	3.00	4.65
				Squamous Cell Carcinoma				
E4	7.09	22.79	28.61	90.12	26.61	84.66	107.70	347.08
E3	7.09	22.79	28.61	90.12	26.61	84.66	107.17	347.08
E2	4.42	5.72	17.94	23.08	16.60	21.41	66.99	86.52
E1	1.92	1.00	7.76	4.03	7.19	3.74	29.19	15.06

Source: Vitaliano and Urbach (1980).

tan, tendency to sunburn, and ethnic origin are particularly important. Evidence presented in the 1982 NAS report and by numerous other researchers has identified those with fair skin, blue/green eyes, red/blond hair, a tendency to burn and to rarely tan, and a Celtic heritage as being at high risk of developing nonmelanoma skin tumors (NAS 1982).

DOSE-RESPONSE RELATIONSHIPS

This section reviews the results of several important studies which have examined the quantitative relationship between exposure to UVR and the incidence of nonmelanoma skin cancer. The methodological issues which add uncertainty to these estimates are also discussed. Much of the discussion in this section is based on review papers by the National Research Council (1982), Van der Leun (1984), and Zeger et al. (undated).

The available evidence from experimental and epidemiological studies clearly indicates that UVR exposure is associated with the development of nonmelanoma skin tumors. In an effort to evaluate the potential impacts on nonmelanoma of increased UVR exposures due to a depletion of ozone layer, several researchers have attempted to quantify the relationship between UVR-dose and the incidence of nonmelanoma skin tumors. Dose-response relationships between UVR and the incidence of nonmelanoma skin tumors can then be combined with estimates of the effects of changing ozone levels on UVR to predict the potential effects of ozone changes on the incidence of nonmelanoma skin tumors.

Previous Analysis

Dose-response relationships for exposure to UVR and nonmelanoma skin tumors have been developed in both epidemiological and experimental studies. Five major dose-response analyses are reviewed below; three are based on epidemiological (ecological) studies, one is based on an animal experiment, and the last is based on a molecular experiment. In each of these studies, UVB exposure is estimated by weighting individual wavelenths in the UVB spectrum according to biological effectivenss. A single weighting approach is referred to as an action spectrum. Several action spectra based on the UVB measurement approach used are available including Robinson-Berger (RB) meter, human erythema and Setlow DNA. The erethyma and DNA action spectra weight those wavelengths thought to promote sunburn and damage to DNA more heavily than other wavelengths.

Early epidemiological research in dose-response estimates was conducted by the National Research Council (NRC 1975), Scott and Straf (1977) and Scotto et al. (1974). These studies all used nonmelanoma skin cancer incidence data from the 1971-1972 National Cancer Institute (NCI) survey and UV doses measured by RB meters at four locations in the United States. The NRC considered these early studies to provide only crude estimates of the dose-response relationship (NRC 1982).

Since these early works, additional epidemiological data were made available by NCI. A new survey of skin cancer incidence was conducted by NCI in 1977-1978 for eight locations across the U.S. This data consists of newly

diagnosed cases of nonmelanoma skin cancer, and are age-, sex-, and cell type-specific for whites only. In addition, new readings from RB meters at five locations in the U.S. were provided. These readings combined with the earlier RB readings provided UVB data for all eight locations included in the 1977-1978 NCI survey.

All three epidemidogical studies reviewed below use the 1977-1978 NCI survey data for nonmelanoma cancer incidence. The studies differ, however, in their choice of an action spectrum and in their specification of the model used to estimate the dose-response relationship.

The relationship between UVB and nonmelanoma skin tumors in the epidemioloigical studies is expressed as a biological amplification factor (BAF) which is defined as the percent change in tumor incidence that results from a one percent change in exposure to UVB radiation. Estimated BAF values can be combined with predictions of the changes in UVB radiation due to changes in ozone to obtain estimates of the effects of ozone changes on nonmelanoma skin tumor incidence rates.

Scotto et al. (1981) and Fears and Scotto (1983)

Scotto et al. (1981) estimated values for the BAF by correlating the incidence of nonmelanoma skin cancer with UVB dosage for eight locations in the U.S. Separate estimates are presented by location, sex, and cell type. The functional form of the model is

$$\ln (R) = \ln(a) + b\ U + e$$

where R is nonmelanoma skin cancer incidence, U is UVB radiation dosage, a and b are constants, and e is an error term. Incidence rates were obtained from the 1977-1978 NCI survey. UVB dosages were obtained from RB meters located at major metropolitan airports at each location. The coefficients were estimated using ordinary least squares regression.

Scotto et al. (1981) found that although incidence rates varied by age, BAFs did not vary significantly by age. Age-adjusted incidence rates were used for all estimates. Their estimates of the BAFs for BCC ranged from 1.32 to 2.59 for males and 1.06 to 2.07 for females. The estimates of the BAFs for SCC were slightly greater, from 2.08 to 7.09 for males and 2.18 to 4.30 for females. In both cases, higher estimates were obtained for locations at lower latitudes.

In a later study, Fears and Scotto (1983) again estimated BAFs by correlating nonmelanoma skin cancer incidence with UVB radiation dosage. In this study Fears and Scotto (1983) used two functional forms of the model. The first is the log-linear form used by Scotto et al. (1981) as described above. The second is the log-log form:

$$\ln (R) = \ln (a) + b \ln (U) + e$$

Separate estimates are presented by sex, location, and percent increase in UVB dosage but not by cell type. The data used were the same as those used in the earlier study.

Fears and Scotto (1983) concluded that the relationship between UVB radiation and nonmelannoma skin cancer incidence could be modeled using either the log-linear or the log-log form. They noted that there was no strong reason to prefer one form of the model over the other. The BAF estimates presented in this study ranged from 1.4 to 3.2 for males and 1.1 to 2.3 for females. Higher estimates were obtained for locations at lower latitudes and higher percent changes in UVB dosage.

Scott (1981)

Scott used the log-linear equation as specified in the Fears and Scotto (1983) study discussed above. Incidence data were also obtained from the 1977-1978 NCI survey. However, rather than using the RB meter data for UVB dosage, Scott (1981) used the action spectrum for DNA damage.

Scott (1981) concluded that the values of UVB dose obtained from the DNA damage weighting method were approximately linear with those obtained from the RB meter. Therefore, the estimates for the BAFs were approximately the same as the estimates made by Scott et al. (1981) using the RB meter data. Estimates of the change in UVB dosage associated with changes in ozone concentration, however, were found to be sensitive to the method used. Thus, even though the estimate of the BAFs were insensitive to the method used to measure UVB dosage, the estimated effect of ozone concentration on nonmelanoma skin cancer is sensitive to the choice of action spectrum.

For example, using the DNA action spectrum, Scott (1981) estimated that a 5 percent ozone reduction would lead to an increase in BCC in Minneapolis-St. Paul of 7.7 percent for males and 5.8 percent for females. Using the RB meter, with the same functional model and cancer incidence data, Scotto et al. (1981) estimated corresponding increases to be 5.6 percent for males and 4.4 percent for females. The reason for this difference lies in the differential sensitivity of different weighting functions to ozone depletion -- the optical amplification factor. Wavelengths at 295-300 nm change much more than at 315-320 nm. Thus the shape of the action spectrum significantly influences the effects of a given ozone depletion.

In an earlier paper, Scott and Straf (1977), using pooled data, pointed out that estimates of the BAF will be biased because actual doses vary by individual. Using survey data from one location only, they estimated that to correct this bias, the BAF's should be increased by 30 percent. More recent estimates of the bias indicate that BAF estimates should be increased by 70 percent (NRC 1982).

Rundell (1983)

The Rundell study is the third epidemiological study to use the 1977-1978 NCI survey data. Measurements from the RB meter were used to estimate UVB exposure. However, Rundell used a different dose-response model than that

used by Fears and Scotto (1983) or Scott (1981) to estimate the BAFs. The Rundell model used age as a proxy for cumulative sun exposure and estimated skin cancer incidence at a single location as a function of this proxy variable. Separate BAF estimates were made by sex, cell-type, and location.

The Rundell model was based on the assumption of reciprosity, i.e., the probability of developing nonmelanoma skin cancer depends only on the cumulative dose received and not on the time course of that dose. The assumption of reciprosity tends to bias the estimates of the BAFs upward. It should be recognized, however, that there is evidence that reciprosity may not be a valid assumption (Fears et al. 1978; Green 1978; de Gruijl 1983).

Rundell (1983) concluded that the probability of a nonmelanoma skin tumor becoming observable in an individual was well described by a log-normal distribution. Estimates of the BAF for males ranged from 1.8 to 2.2 for BCC and from 2.4 to 2.8 for SCC. The corresponding ranges for females were 1.1 to 1.5 for BCC and 1.6 to 2.1 for SCC. The higher values were obtained for lower latitudes.

Rundell (1983) estimated that a 1 percent depletion in ozone would lead to a 1.78 percent increase in BCC and a 2.3 percent increase in SCC. He concluded that these estimates were insensitive to the choice of action spectrum. Comparing dosage weighted by the RB meter with dosage weighted by the DNA damage spectrum, Rundell (1983) showed that with the RB meter the effect of ozone changes on UVB radiation would be small and the effect of UVB dosage on skin cancer incidence would be larger. Thus, the total effect of ozone changes on skin cancer incidence would be approximately the same.

de Gruijl et al. (1983)

The dose-response analysis presented by de Gruijl et al. (1983) was based on data from an animal experiment. Mice were exposed to radiation from a sunlamp for 3.25 hours per day at seven different levels ranging from 0 mJ/cm^2 to 190 mJ/cm^2. The response was measured by observing the initiation and growth of tumors on the mice. The results of this study showed that the initiation of tumors was dependent on the dose, but that the growth and yield of tumors were independent of dose. He also concluded that reciprocity was not supported, except at high dosage levels.

Kubitschek et al. (1986)

This study presents a method for calculating the effects of a reduction in atmospheric ozone upon the incidence of nonmelanoma skin tumors by calculating the incidence of mutations in *E. coli*. The model that was applied had the following form:

$$Sm = \int E(\theta,\lambda,w)\ T(\lambda)\ \varepsilon(\lambda)\ d\lambda$$

where

Sm	= Rate of induction of mutation
θ	= Solar zenith angle
λ	= Wavelength
W	= Amount of stratospheric ozone
$E(O,\lambda,W)$	= irradiation at earth's surface
$T(\lambda)$	= rate of transmission through skin
$\varepsilon(\lambda)$	= action spectrum from E. coli

The Kubitschek et al. (1986) study predicted that a one percent reduction in atmospheric ozone leads to a 1.7 percent increase in mutation rate. This estimate was constant for solar zenith angles up to 70 degrees, a range which includes most heavily populated areas of the globe. Their conclusions were based on the action spectrum for mutagenesis in E. coli, unpublished information in similar studies in mammalian cells, information on the epidermal transmission rate of the various UV wavelengths, and consideration of a BAF taken from van der Leun (1984). They estimated that a 3 percent-5 percent stratospheric ozone depletion rate would lead to an increase in nonmelanoma skin cancer rates of about 10 percent-20 percent.

Uncertainties in Estimates of Dose-Response

There are a number of issues related to the studies described above which add uncertainty to the estimates of the dose-response. The degree of uncertainty that is added has not been fully quantified.

A major issue affecting the uncertainty of the estimtes is the availability of epidemiological data. All of the epidemiological studies described above use the 1977-1978 NCI survey for nonmelanoma-tumor incidence data. However, data are not currently available for individual UVB dosage. Therefore, researchers have had to use a single measure of UVB dosage for all individuals in a given location. Actual dosages recieved will vary by individual because of behavioral factors such as time spent outdoors. The incidence of nonmelanoma skin cancer will also vary by individual because of genetic characteristics such as skin color. If these factors are correlated with location, the estimates could be biased. In addition, the issue of peak versus cumulative dose has not been addressed in these studies; although basal and squamous cell skin cancers clearly have somewhat different etiologics, suggesting somewhat different responses to UVB.

Furthermore, the three epidomological studies discussed above cannot be considered independent because they are all based on the same skin cancer incidence database. As more comprehensive data are collected for more locations, across longer time periods, and for individuals, estimates of dose-response will hopefully become more precise.

Another important issue is the choice of action spectrum for measuring UVB dosage. The estimate of the effect of ozone depletion on skin cancer incidence is sensitive to the choice of action spectrum. There is no current concensus on what is the best action spectrum to use to estimate the dose-response relationship.

There is also statistical uncertainty in the measurement of UVB flux. This uncertainty is not captured in the regression analysis, which considers the UVB values as certain. As a result, the dose-response coefficients are subject to a greater degree of statistical uncertainty than is indicated in the regressions. Incorporating statistical uncertainty in the estimates of UVB would probably result in different dose-response coefficients, but the direction and magnitude of this difference is not known (Zeger (no date)).

Another issue is whether the models used are valid for all scenarios of ozone change. For large levels of ozone depletion, for example, the amount of UVB radiation reaching the Earth's surface may be outside the range of UVB data used to estimate the dose-response relationship in the models, or may occur in a different pattern.

Because it is impossible to experiment on humans, experimental (e.g., animal) studies may be used to examine the association between UVB and nonmelanoma skin cancer. The extent to which the animal response to UVB resembles the human response is not well known. Furthermore, the intensity, duration, schedule, and range of the doses applied in an animal experiment is likely to differ from the exposures received by humans in the ambient environment.

In conclusion, while quantified dose-response estimates provide critical information relating skin cancer incidence rates to changes in the ozone level, they should be regarded with caution. As briefly described above, there are numerous uncertainties associated with such dose-response estimates.

Sensitivity of Dose-Response Estimates to Choice of Action Spectrum

As noted above, one important methodological issue which adds uncertainty to the estimates of dose-response is the choice of action spectrum. In comparing the DNA damage weighting method with the RB meter method used by Scotto et al. (1981), Scott (1981) found that estimates of the BAFs were approximately the same but that estimates of the effect of ozone depletion on UVB dosages were different. On the other hand, Rundell (1983) concluded that the effect of ozone depletion on skin cancer incidence was insensitive to the choice of action spectrum.

An independent analysis of the sensitivity of dose-response estimates to the choice of action spectrum was conducted and is summarized in Appendix A. The methodology used in this analysis follows that used by Fears and Scotto (1983). The dose-response is estimated using three alternative action spectra -- RB meter, human erethyma, and Setlow DNA.

The major result of this analysis was that the estimated BAF is not sensitive to the choice of action spectrum. The estimated coefficient using the human erethyma and Setlow DNA action spectra all fell well within one standard error of the coefficients estimated for the RB meter action spectrum (see Exhibit A-3 in Appendix A).

However, because the optical amplification factor is sensitive to ozone depletion, (that is, because UVB at 295-300 nm increases more than at 315 nm-320 nm) estimates of the effect of ozone depletion on UVB dosage were found to differ significantly by choice of action spectrum. Use of an action spectrum based on the effects on a biological target, of course, makes sense. Nevertheless, an issue still exists about whether DNA, erethyma, mouse edema, or some other biological action spectrum should be used. The Setlow DNA action spectrum was found to give the highest estimates of the effect of ozone depletion on UVB dosage of the three examined (see Exhibit A-4 in Appendix A). Obviously, the choice of an action spectrum will influence the estimates of the effect of ozone depletion on skin cancer incidence.

APPENDIX A

Sensitivity Analysis of the Selection of Action Spectra on Dose-Response Estimates Relating UVR Exposures to Nonmelanoma Skin Tumors

This appendix presents the results of an analysis of the relationship between changes in ozone levels and incidence rates of nonmelanoma skin tumors. Estimating the impact of ozone change on tumor incidence requires two pieces of information: (1) the response of UVB radiation to changes in ozone, and (2) the dose-response relationship between UVB exposure and the incidence of nonmelanoma skin tumors. This analysis focuses on the sensitivity of the selection of an action spectrum used to describe the relationship between UV exposure and the incidence of nonmelanoma skin cancer.

UVB Exposure and Nonmelanoma Skin Cancer

The relationship between UVB exposure and the incidence of nonmelanoma skin tumors was estimated from the power model used by Fears and Scotto (1983):

$$\ln (R) = a + b \ln (UV) + e$$

where R is age-adjusted incidence, e is an error term, and a and b are constants. The constant b is the BAF that describes the UVB dose-skin cancer incidence relationship.

The coefficients a and b were estimated using weighted ordinary least squares regression. Two sets of data were required to develop these estimates: (1) incidence rates for nonmelanoma skin tumors, and (2) measurements of UVB exposure for individuals developing these tumors. The sources of each data set are discussed below.

Incidence of Nonmelanoma Skin Cancer

Age-specific incidence rates (new cases per 100,000 population) for nonmelanoma skin cancer among whites were obtained from Scotto et al. (1981) for eight locations in the United States (1977-1978 NCI survey data).

UVB Radiation

Although there is substantial agreement that wavelengths within the UVB band are primarily involved in the development of nonmelanoma skin cancers, there is uncertainty about which individual wavelengths are most important. In this analysis, the relative effect of using three alternative action spectra in regressions to quantify the coefficients relating UVB dose to skin cancer incidence relationship: Robinson-Berger (RB) meter, human erethyma, and Setlow DNA. The erethyma and DNA action spectra weight those wavelengths thought to promote sunburn and damage to DNA more heavily than other wavelengths.

Data on UVB radiation were obtained from the NASA UVB model (Serafino and Frederick 1986). This model uses mathematical relationships to estimate UV solar flux along different wavelengths (UVA and UVB) reaching the Earth's surface at any time of year and location on the globe. To estimate UV flux, the model uses information about global ozone abundance, terrain height, cloud cover, cloud transmission, and surface characteristics.

The NASA model provided estimates of UVB radiation for each of the eight U.S. locations. The data were provided for all three action spectra, and for each action spectrum, data were presented for three different time periods -- annual, a day in the month of June, and a clear day. In addition to UVB data at current ozone levels, the NASA model provided estimates of radiation reaching the Earth's surface at different ozone levels.

To analyze the significance of measuring UVB radiation using different action spectra and time periods, Exhibit A-1 shows correlations among the NASA UVB data for the three action spectra and for annual, month of June, and clear day time periods. The exhibit also shows correlations among actual UVB measurements (Scotto, Fears, and Fraumeni 1981) and the modeled data. The strong correlation among these UVB data suggests that dose-response coefficients are not highly sensitive to the different measures. Nonetheless, a regression analysis was performed using dose-response coefficients for the annual time period for all three action spectra. Before performing the regression analysis, two weights were applied to the data. First, the data were weighted by the population of each respective location. Exhibit A-2 shows the populations of these locations. Second, as a correction for heteroskedasticity, the data were weighted by the inverse of the estimated variance of the logarithm of the age-adjusted incidence rates.

Exhibit A-3 presents regression estimates of dose-response coefficients for basal and squamous cell skin cancers by sex. Separate estimates are given for all three action spectra (annual only). The exhibit shows a low, middle, and high estimate. The low value represents the estimated coefficient minus one standard error. The high coefficient adds one standard error. The dose-response coefficients for SCC are higher than those for BCC for each of the action spectra. In addition, the coefficients for SCC were found to be of greater statistical significance. These findings are consistent with the belief that cumulative UVB radiation has a greater effect on the development of SCC than on the development of BCC. As already mentioned, epidemiological observations indicate that the ratio of basal to squamous cell skin cancer decreases with decreasing latitude and that BCC is more likely to develop on regularly unexposed sites. In addition, the sensitivity of basal cell cancer incidence rates to UVB was higher for males than for females. The relative sensitivity by sex is reversed for SCC.

It is important to recognize that the NASA models UVB flux values are subject to statistical uncertainty. This uncertainty is not captured in the regression analysis which treats the UVB values as certain. As a result, the dose-response coefficients are subject to a greater degree of statistical uncertainty than is indicated in the regressions. Incorporating statistical

EXHIBIT A-1

Correlation of Alternative Measurements of UVB Radiation for Ten Locations in the United States[a]

	Annual[c]				Clear Day[c]			Month of June[c]		
	RB Actual[b]	RB	Erethyma	DNA	RB	Erethyma	DNA	RB	Erethyma	DNA
Annual[b]										
RB Actual	1.00	0.979	0.988	0.987	0.940	0.977	0.977	0.882	0.955	0.967
RB		1.00	0.990	0.984	0.964	0.975	0.970	0.935	0.975	0.989
Erethyma			1.00	0.999	0.941	0.985	0.986	0.883	0.959	0.971
DNA				1.00	0.932	0.984	0.987	0.866	0.949	0.963
Clear Day										
RB					1.00	0.986	0.955	0.938	0.967	0.968
Erethyma						1.00	0.999	0.869	0.947	0.960
DNA							1.00	0.850	0.936	0.951
Month of June										
RB								1.00	0.978	0.967
Erethyma									1.00	0.999
DNA										1.00

[a] The ten locations include: Seattle, Washington; Minneapolis, Minnesota; Detroit, Michigan; Des Moines, Iowa; Salt Lake City, Utah; San Francisco-Oakland, California; Atlanta, Georgia; Dallas-Ft. Worth, Texas; New Orleans, Louisiana; Albuquerque, New Mexico. Data for Des Moines and Dallas-Ft. Worth were included in the correlations; however, because nonmelanoma skin cancer rates were unavailable, these locations were excluded from the dose-response regression analysis.

[b] Annual UVB data from Scotto, Fears and Fraumeni (1981). These data are actual UVB measurements taken at National Weather Service stations.

[c] Model data from the NASA UV model.

EXHIBIT A-2

Population Weights for Ten Locations in the United States

Location	Weight
Seattle	1,607,469
Minneapolis-St. Paul	2,113,533
Detroit	4,353,413
Salt Lake City[a]	1,461,000
San Francisco-Oakland[b]	3,250,630
Atlanta	2,029,710
New Orleans	1,076,204
Albuquerque[c]	1,303,000

[a] State of Utah.

[b] San Francisco.

[c] State of New Mexico.

Source: State and Metropolitan Area Data Book, 1982, U.S. Department of Commerce, Bureau of the Census, U.S. Government Printing Office August 1982.

EXHIBIT A-3

Estimated Dose-Response Coefficients (and t-Statistics) for Basal and Squamous Cell Skin Cancers (UVB Dose-Skin Cancer Incidence)

	Robinson-Berger			Human Erethyma			Setlow DNA		
	a		b	a		b	a		b
	Low	Mid	High	Low	Mid	High	Low	Mid	High
Basal									
Male	1.300	1.810 (3.53)	2.330	1.020	1.410 (3.60)	1.800	0.932	1.290 (3.60)	1.650
Female	0.443	1.050 (1.73)	1.670	0.346	0.809 (1.75)	1.270	0.316	0.739 (1.75)	1.160
Squamous									
Male	1.940	2.840 (3.20)	3.730	1.540	2.210 (3.31)	2.880	1.420	2.030 (3.33)	2.640
Female	1.880	3.060 (2.60)	4.230	1.570	2.420 (2.87)	3.260	1.470	2.220 (2.94)	2.980

a
Estimated coefficient minus one standard error.

b
Estimated coefficient plus one standard error.

uncertainty into the estimates of UVB would probably result in different dose-response coefficients, but the direction and magnitude of this difference is not known.

Ozone Levels and Changes in UVB Exposure

The NASA UVB model was used to estimate changes in UVB radiation associated with changes in ozone levels. Exhibit A-4 shows representative estimates for San Francisco. In the exhibit, a two percent reduction in ozone resulted in an estimated 1.6 percent increase in UVB radiation as measured on a RB meter. The estimated percentage increase is greater using the human erethyma or Setlow DNA action spectra.

Relationship Between Ozone Changes and the Incidence of Nonmelanoma Skin Tumors

The NASA estimates of the sensitivity of UVB radiation to changes in ozone were combined with the UVB dose-skin cancer incidence coefficients. Exhibits A-5 and A-6 present the results of this analysis for an estimated 2 percent and 10 percent depletion in the ozone layer for San Francisco. For example, Exhibit A-5 indicates that the steady-state incidence rate of basal cell skin cancer could increase by 2.98 percent for males and 1.72 percent for females in response to a 2 percent depletion in ozone.

EXHIBIT A-4

Estimated Percentage Changes in UVB Radiation in San Francisco For a Two and Ten Percent Depletion in Ozone

	Ozone Depletion (percent)	
	2	10
Robinson-Berger	1.6	8.6
Human Erethyma	3.5	19.0
Setlow DNA	4.3	23.0

EXHIBIT A-5

Percentage Change in Incidence of Basal and Squamous Cell Skin Cancers for a Two Percent Depletion in Ozone for San Francisco

	Robinson-Berger			Human Erethyma			Setlow DNA		
	Low	Mid	High	Low	Mid	High	Low	Mid	High
Basal									
Male	2.13	2.98	3.86	3.55	4.95	6.36	3.99	5.56	7.17
Female	0.72	1.72	2.75	1.19	2.81	4.44	1.33	3.15	4.99
Squamous									
Male	3.20	4.72	6.24	5.41	7.86	10.36	6.14	8.89	11.72
Female	3.10	5.09	7.11	5.52	8.64	11.81	6.36	9.76	13.32

EXHIBIT A-6

Percentage Change in Incidence of Basal and Squamous Cell Skin Cancers for a Ten Percent Depletion in Ozone for San Francisco

	Robinson-Berger			Human Erethyma			Setlow DNA		
	Low	Mid	High	Low	Mid	High	Low	Mid	High
Basal									
Male	11.34	16.14	21.24	19.10	27.34	36.14	21.15	30.41	40.45
Female	3.73	9.07	14.80	6.11	14.87	24.32	6.72	16.43	26.97
Squamous									
Male	17.39	26.46	36.12	30.20	46.05	63.82	33.95	51.87	72.19
Female	16.81	28.78	41.86	30.88	51.40	74.84	35.34	57.93	84.68

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CHAPTER 8

CUTANEOUS MALIGNANT MELANOMA

SUMMARY

In 1985, there were 25,000 cases of cutaneous malignant melanoma (CMM) in the United States and 5,000 deaths. For more than a decade, there has been serious concern that CMM is at least partially caused by ultraviolet-B radiation (UVB). However, several aspects of the scientific information about CMM have puzzled researchers and have contributed to uncertainty about the relationship of CMM to solar radiation, in particular UVB. In the past several years, some progress has been made in understanding CMM and its possible relation to solar radiation, and there now exists an array of evidence which indicates that exposure to solar radiation, and UVB in particular, is a likely cause of CMM.

Supporting evidence includes the fact that people who lack the protection of pigmentation which blocks penetration of ultraviolet radiation (UVR) in the skin have higher CMM incidence rates; a correlation, in well-designed ecological studies, of higher CMM rates with decreasing latitude and increasing UVB levels; the association of freckling and nevus formation (risk factors for CMM) with solar exposure; the differences between natives and immigrants to sunny climates in CMM rates; high rates of CMM in patients who are genetically deficient at repairing DNA damage induced by UVB; and the indication, in controlled studies, that sun exposure at early ages and of an intermittent nature results in higher CMM risks.

Information that has created uncertainty about the relationship between solar radiation and CMM has included: a failure to find latitude gradients in some ecological studies; the fact that outdoor workers have lower CMM rates than indoor workers (but higher non-melanoma skin cancer rates); and the fact that anatomic sites with lower sun exposure have high CMM rates.

The latter evidence has made it clear that the relationship between solar radiation and CMM is not a simple one. In fact, this evidence can only be reconciled with the hypothesis that solar radiation is an etiological factor in CMM if one concludes that cumulative dose based on total number of hours in the sun is not the appropriate dose parameter to correlate with CMM development. This has led to the emergence of hypotheses that postulate the importance of intermittent exposure to high fluxes of UVB (resulting in large UVB doses) in the causation of CMM.

Unfortunately, at this time, there have been no studies which examine the correlation between CMM and actual measured UVB doses received by individuals, thus making it impossible to prove various hypotheses on CMM. The balance of available evidence appears to support the conclusion that solar radiation is at least a major cause of CMM. Other evidence appears to indicate that cumulative sun-exposed hours is not an appropriate measure of dose for CMM, and that CMM has a complicated etiology.

A variety of different kinds of evidence supports the hypothesis that UVB is a component of solar radiation that causes CMM. Most important is the fact that xeroderma pigmentosum patients, who are genetically deficient in repairing UVB-induced DNA damage, have significantly higher rates of CMM than the general population. Another fact that supports the hypothesis that UVB is a component of solar radiation that causes CMM is the fact that, in experimental studies, UVB is the most mutagenic and carcinogenic waveband as well as being the most active at causing immunosuppression. All of this information suggests that UVB is the primary component of solar radiation that causes CMM. Together, this evidence does not provide proof that UVB is the waveband of solar radiation that causes CMM; however, the preponderance of evidence makes UVB a likely cause of CMM.

Finally, although much remains uncertain about the relationships between solar radiation UVB and CMM, enough information exists to make reasonable estimates of the dose-response relationships which exist between UVB and CMM. These can then be used to estimate future cases and mortality of CMM if ozone depletion occurs. The possible relationships based on an improving but still deficient database in the United States indicate that each 1 percent ozone depletion will produce around a 1 to 2 percent increase in incidence and 0.8 to 1.5 percent increase in mortality. At this time, it is impossible to estimate how incidence and mortality of CMM will change in other countries whose populations have unknown skin pigmentation and behavior.

FINDINGS

1. CUTANEOUS MALIGNANT MELANOMA (CMM) IS A SERIOUS LIFE-THREATENING DISEASE THAT AFFECTS A LARGE NUMBER OF PEOPLE IN THE UNITED STATES.

   1a. CMM incidence and mortality is increasing among fair-skinned populations. These increases appear to be real and are not just the result of improved diagnosis and reporting.

   1b. In 1985, there were 25,000 cases of CMM and 5,000 fatalities related to melanoma in the United States. If current trends continue even without ozone depletion, the lifetime risk based on current data appears to be about 1 in 150 in the United States; compared to less than 1 in 66 currently observed in Australia.

2. LIMITATIONS IN THE DATABASE PREVENT ABSOLUTE CERTAINTY ABOUT THE RELATIONSHIP OF SOLAR RADIATION, UVB, AND CUTANEOUS MALIGNANT MELANOMA.

   2a. There is no animal model that uses UVB to induce melanomas experimentally.

   2b. There is no experimental *in vitro* model for malignant transformation of melanocytes.

   2c. No epidemiologic studies of CMM have been conducted in which individual human UVB exposures (and biologically effective doses of solar radiation) have been adequately assessed.

3. EVALUATION OF THE EPIDEMIOLOGICAL AND EXPERIMENTAL DATABASES REQUIRES CLOSE ATTENTION TO THE RELATIONSHIP OF WAVELENGTH AND DOSE AND TO THE VARIATIONS OF SOLAR RADIATION IN THE AMBIENT ENVIRONMENT.

   3a. Ozone differentially removes wavelengths of UVB between 295-320 nm; UVA (320-400 nm) is not removed above 350 nm and visible light (400-900 nm) is not removed. It totally removes all UV-C (i.e., wavelengths less than 295 nm).

   3b. Wavelengths between 295 nm and 300 nm are generally more biologically effective (i.e., damage target molecules in the skin including DNA) than other wavelengths in UVB and even more so than UVA radiation.

   3c. Latitudinal variations exist in solar radiation; model predictions indicate that the greatest variability is seen in cumulative UVB (e.g., monthly doses) followed by peak UVB (highest one-day doses) and then cumulative UVA. Peak UVA does not vary significantly across latitudes up to 60°N. Greater ambient variation also exists in UVB than UVA by time of day.

3d. The biologically effective dose of radiation that actually reaches target molecules depends on the duration of exposure at particular locations, time of day, time of year, behavior (i.e., in terms of clothes and sunscreens), pigmentation, and other characteristics of the skin including temporal variations (e.g., in tanning and in skin thickening).

3e. Cloudiness and albedo, although causing large variations in the amount of UVB and UVA, do not greatly change the ratio of UVB to UVA.

3f. Ozone depletion is predicted to cause the largest increases in radiation in the 295-299 nm UVB range, less in 300-320 nm UVB range, and UVA is virtually unaffected.

3g. Cutaneous malignant melanoma has a number of different histologic types which vary in their relationship to sunlight, site, and racial preference and possibly in their precursor lesions. Assessment of these types is not consistent among registries, thus confounding attempts to evaluate associations between CMM and solar radiation.

3h. Melanin is the principal pigment in skin that gives it color; melanin absorbs UV radiation very effectively, thus the darker the skin, the more the basal layer is protected from UV.

4. A LARGE ARRAY OF EVIDENCE SUPPORTS THE CONCLUSION THAT SOLAR RADIATION IS ONE OF THE CAUSES OF CUTANEOUS MALIGNANT MELANOMA.

4a. Whites have higher incidence and mortality rates of CMM than blacks.

4b. Light-skinned whites, including those who are unable to tan or who tan poorly, get more CMM than darker-skinned whites.

4c. Sun exposure leading to sunburn apparently induces melanocytic nevi.

4d. Individuals who have more melanocytic nevi develop more CMM; the greatest risk is associated with a particular type of nevus -- the dysplastic nevus.

4e. Sunlight induces freckling, and freckling is an important risk factor for CMM.

4f. Incidence has been increasing in cohorts in a manner consistent with changes in patterns of sun exposure, particularly with respect to increasing intermittent exposure of certain anatomical sites.

4g. Immigrants who move to sunnier climates have higher rates of CMM than populations who remain in their country of origin. Immigrants develop rates approaching those of the adopted country; this is particularly accentuated in individuals arriving before the age of puberty (10-14 years).

4h. CMM risk is associated with childhood sunburn; this may reflect an individuals pigmentary characteristics or may be related to nevus development.

4i. Most studies that have used latitude as a surrogate for sunlight or UVB exposure have found an increase in the incidence or mortality of CMM as one approaches the equator. A recent study on incidence that used measured UVB and survey data found a strong relationship between UVB and incidence. Another study that used modeled UVB data and an expanded database on mortality found a strong UVB-mortality relationship.

4j. Patients with xeroderma pigmentosum who cannot repair UVB-induced lesions in skin DNA have a 2,000-fold increase in CMM by the age of 20.

4k. One form of CMM, Hutchinson's melanotic freckle melanoma, appears almost invariably on the chronically sun damaged skin of older people.

5. SOME EVIDENCE CREATES UNCERTAINTY ABOUT THE RELATIONSHIP BETWEEN SOLAR RADIATION AND CUTANEOUS MALIGNANT MELANOMA.

5a. Some ecologic epidemiology studies have failed to find a latitudinal gradient for CMM.

5b. Outdoor workers generally have lower incidence and mortality rates for CMM than indoor workers, which appears incompatible with a hypothesis that cumulative dose from solar exposure causes CMM.

5c. Unlike basal cell and squamous cell carcinomas, most CMM occurs on sites that are not habitually exposed to sunlight; this contrast suggests that cumulative exposure to solar radiation or UVB is not solely responsible for variations in CMM.

6. ULTRAVIOLET-B RADIATION IS A LIKELY COMPONENT OF SOLAR RADIATION THAT CAUSES CUTANEOUS MALIGNANT MELANOMA (CMM).

6a. Xeroderma pigmentosum patients who fail to repair UVB induced pyrimidine dimers in their DNA have a 2,000-fold excess rate of CMM by the time they are 20.

6b. UVB is the most active part of the solar spectrum in the induction of mutagenesis and transformation in vitro.

6c. UVB is the most active part of the solar spectrum in the induction of carcinogenesis in experimental animals and is considered by most to be a causative agent of nonmelanoma skin cancer in humans.

6d. UVB is the most active portion of the solar spectrum in inducing immunosuppression which may have a role in melanoma development.

6e. The limitations in the epidemological and experimental database leave some doubt as to the effectiveness of UVB wavelengths in causing CMM.

7. WHILE SOME UNCERTAINTY EXISTS, INCREASES IN THE INCIDENCE AND MORTALITY OF CUTANEOUS MALIGNANT MELANOMA ARE LIKELY AS A RESULT OF OZONE MODIFICATION. ALTHOUGH IMPERFECT, INFORMATION EXISTS TO ESTIMATE CHANGES IN INCIDENCE AND MORTALITY IF THE OZONE LAYER IS MODIFIED.

7a. Well-designed ecological studies using survey data and measured UVB provide a reasonable estimate of dose response relationship for UVB and CMM. A one percent change in ozone is likely to increase incidence by between slightly less than 1% and 2%, depending on the choice of action spectrum.

7b. Using predictions of UVB from a NASA model and a database for CMM mortality developed by EPA/NIA, a dose-response relationship for CMM mortality has been developed. It is estimated that a one percent change in ozone would result in a 0.8 to 1.5 percent change in CMM mortality.

7c. Uncertainty exists about the appropriate action spectrum to be used in estimating dose, the best functional form for dose-response, and the best way to characterize dose (peak value, cumulative summer exposure, etc.). Histologically different CMM (or possibly CMM located at different anatomical sites) are likely to have different dose-response relationships. Current estimates of dose-response relationships fail to consider these histological or site differences.

7d. Additional problems for projecting future incidence and mortality of CMM in the U.S. include a lack of a good database with regard to variations in skin pigmentation and human sun-exposure behavior among different populations and estimates of how these relationships may change in the future.

7e. At this time, projections of CMM increases in other parts of the world are not possible due to insufficient access to necessary data.

INTRODUCTION

This chapter is designed to examine the current state of knowledge with regard to the relationship between human cutaneous malignant melanoma and solar radiation, in particular UVB. Two previous reviews examining this question came to the following somewhat contradictory conclusions:

> "The NCI [National Cancer Institute] is collecting skin melanoma incidence from ten locations in the United States. Earlier data analyses indicate that skin melanomas, which predominate in Caucasians, are related to UVB exposure. A preliminary finding is that the incidence is increasing and that the rate of increase may be greater than expected from earlier surveys. The dose-response relationship between skin melanoma and UVB appears to be more complicated than that observed for nonmelanomas of the skin." (NAS 1978)

> "...since 1976, the case for an association between UVB and melanoma has been weakened rather than strengthened by the results of additional clinical, pathological and epidemiological studies. Furthermore (with the exception of a single animal), it has not been possible to use UVB alone to induce melanomas in experimental animals." (NAS 1982)

These contrasting conclusions require that a closer examination be made of the relationship between melanoma and UVB.

Since publication of the second NAS document in 1982, more information has been gathered; this chapter reviews briefly both the old and the new information in order to ascertain what can be said at the current time about the relationship of UVB to melanoma. A much more detailed review of the relevant information is presented in Appendix A.

BACKGROUND INFORMATION

A number of factors can modify the amount of solar radiation which should be considered the "dose" received by a particular target. This section presents a brief overview of factors which can influence this "dose" -- a detailed discussion is provided in Chapter 2 of Appendix A.

Background on Solar Radiation and the Concept of Dose

The total amount of energy from ultraviolet radiation that any target receives in a given amount of time will depend, in part, on variations in ambient radiation that occur in the natural environment. One effect of the ozone layer and the earth's rotation and revolution is the modification of the quality and quantity of solar energy delivered from place to place over time. Ambient solar radiation incident on various sites on the earth's surface varies significantly with latitude, altitude, season (day of the year), time of day, cloudiness, reflectiveness of surfaces (albedo) and atmospheric aerosols. More important, these variations differ quantitatively for different wavelengths of UVR.

A location's latitude determines the average angle between the location and the sun. This angle, in turn, determines the thickness of the atmosphere that photons must pass through before reaching the location. The lower the angle, the longer the path through the ozone-containing stratosphere that photons must travel and the greater the amount of absorption that can occur. A longer path also allows longer exposure to aerosols and, thus, more scatter -- thereby additionally reducing the amount of energy reaching a location. Because ozone absorbs different wavebands differently, however, all photons are not retarded equally, instead differential gradients for various wavelengths occur. Exhibit 8-1 shows estimates from the UV Model, developed by Serafino and Frederick (1986), which predict that at 12 noon on March 21, the flux of UVB at 295-299 nm can be expected to vary from the equator to 70°N by a factor of over 100, while UVA at 335-339 nm can be expected to vary by a factor of 5.

Season also has a tremendous impact on the flux of solar radiation at a given location. Again, this occurs because the relationship of a location to the sun changes with season. Exhibit 8-2 shows model estimates of how the various wavelengths vary by month for Washington, D.C. Energy at 295 nm increases by about a factor of 10 from winter (December) to Spring (March) and by about another factor of 10 by midsummer (July). UVB at 305 nm shows much less variation and UVA very little, if any, variation.

Exhibit 8-3 shows the impact that varying the time of day has on estimates of UVB. Clearly, the time of day of exposure is a very important factor in determining the amount of UV energy received at the various wavelengths. Variations in cloud cover and surface albedo also cause variations in ambient solar energy levels, however, there is not much differential variation by wavelength (for additional details, see Chapter 2, Appendix A).

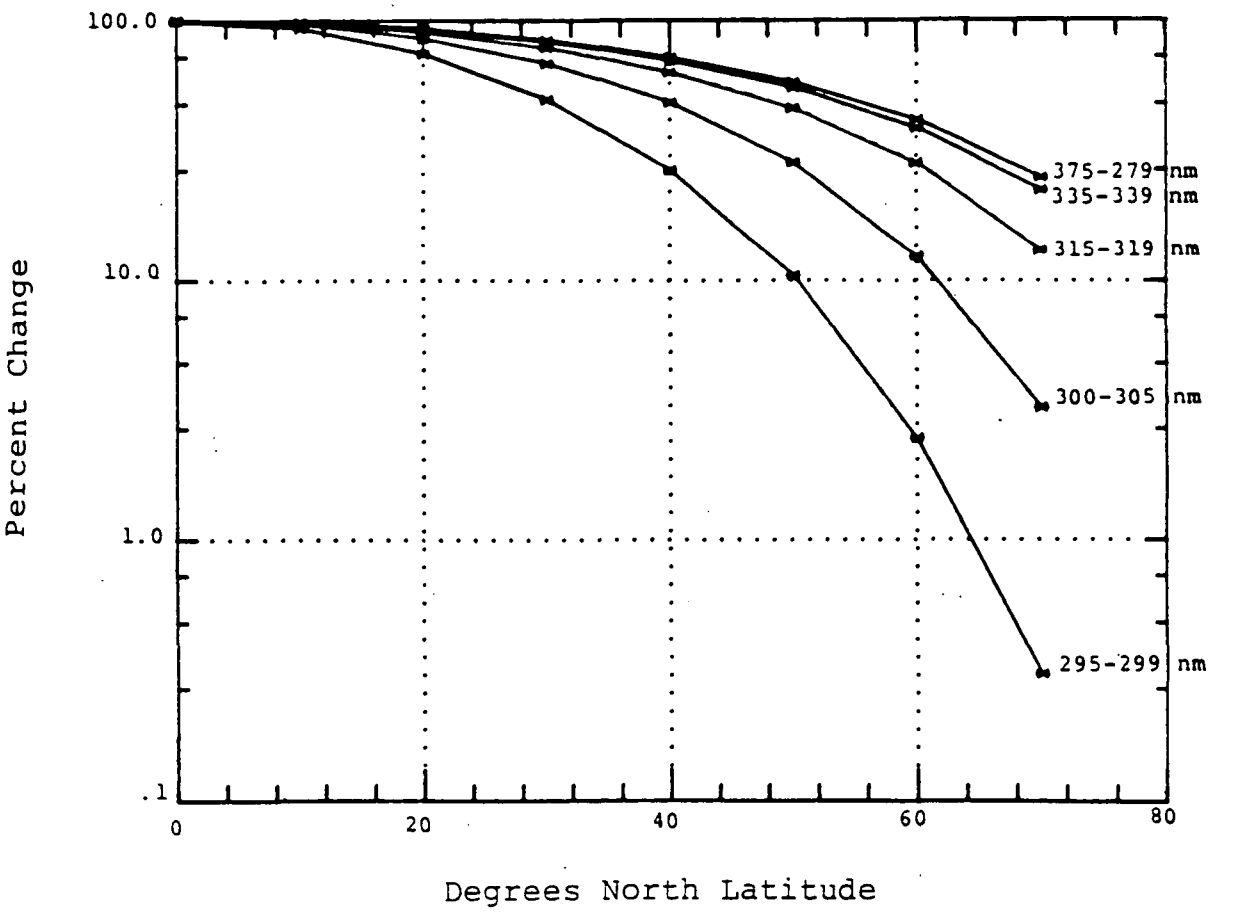
In addition to factors that modify the ambient levels of UV in the environment, there are a number of factors which can influence the biological effective dose (i.e., the amount of energy received by a target in the skin that results in the development of CMM). These include an individual's clothing and sun-exposure behavior habits, the degree of skin pigmentation and thickening and the action spectrum for the biologically important effect.

The amount of energy which is actually effective is determined by the action spectrum of the effect of concern. If, for instance, damage to DNA is the underlying effect linking solar radiation to cutaneous malignant melanoma, then not all of the photons delivered to the target molecules will be effective. Instead, the absolute amounts of energy delivered in the various wavelengths need to be weighted according to the action spectrum presented in Exhibit 8-4. There are a number of different effects which might underlie the relationship between solar radiation and CMM (details of which are presented in Chapters 2 and 3 of Appendix A); a key characteristic of them is that energy in the UVB wavelengths (295-320 nm) tends to be much more biologically active.

How much target cells can potentially receive depends on the time a person spends outside during certain periods of the day and days of the year. The total amount of energy delivered at a given location provides an upper bound

EXHIBIT 8-1

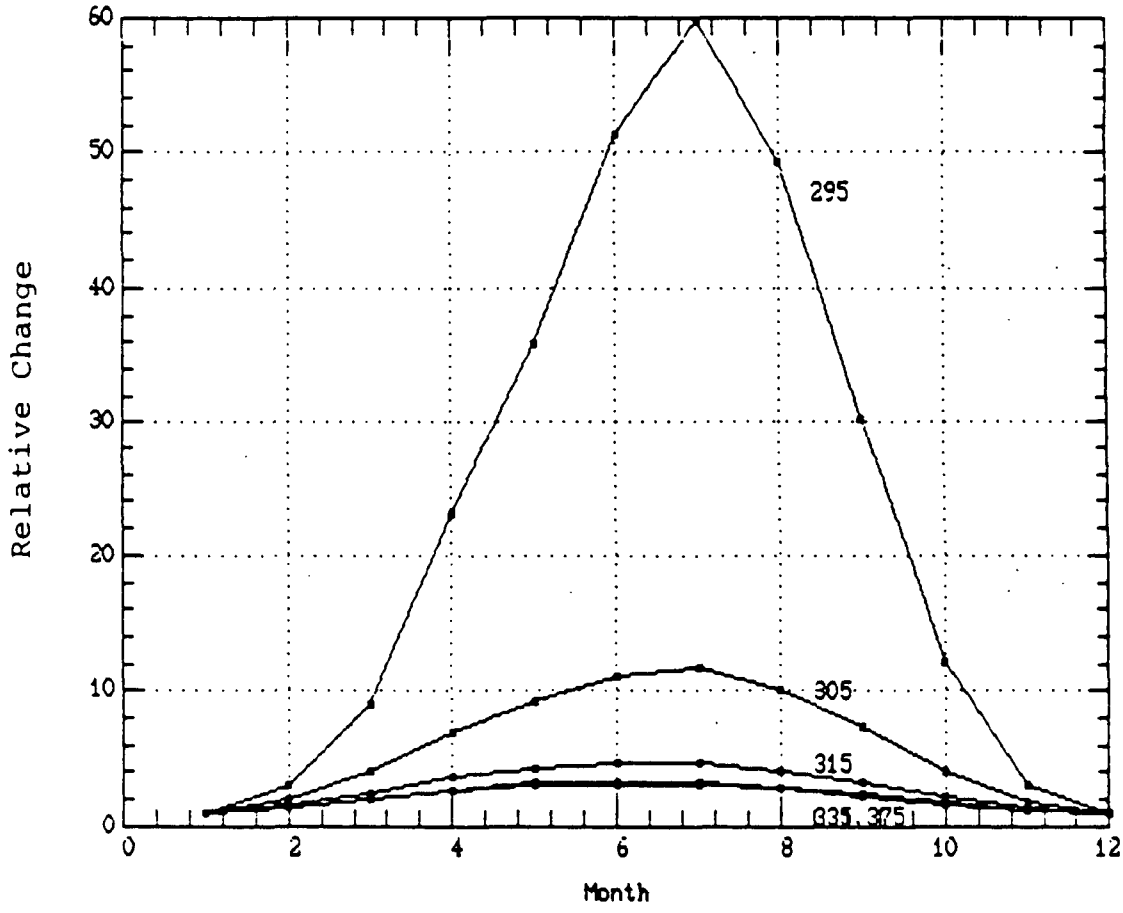
Variation in UV Radiation by Latitude as Percent of Levels at the Equator on March 21 at Noon



This graph shows the variation estimated by the NASA model for five wavebands of ultraviolet radiation for a single day, high noon, in March. The variation would be different for other days.

EXHIBIT 8-2

UV Radiation by Month in Washington, D.C.



This graph shows that the NASA model estimates that radiation at 295 nm has a much larger proportional gain than at 305 or higher wavelengths. Note that the 335 nm line is almost coincident with the x axis, indicating low monthly variation in Washington, D.C.

EXHIBIT 8-3

Relative Change in UV Flux by Hour in Washington, D.C., on June 21

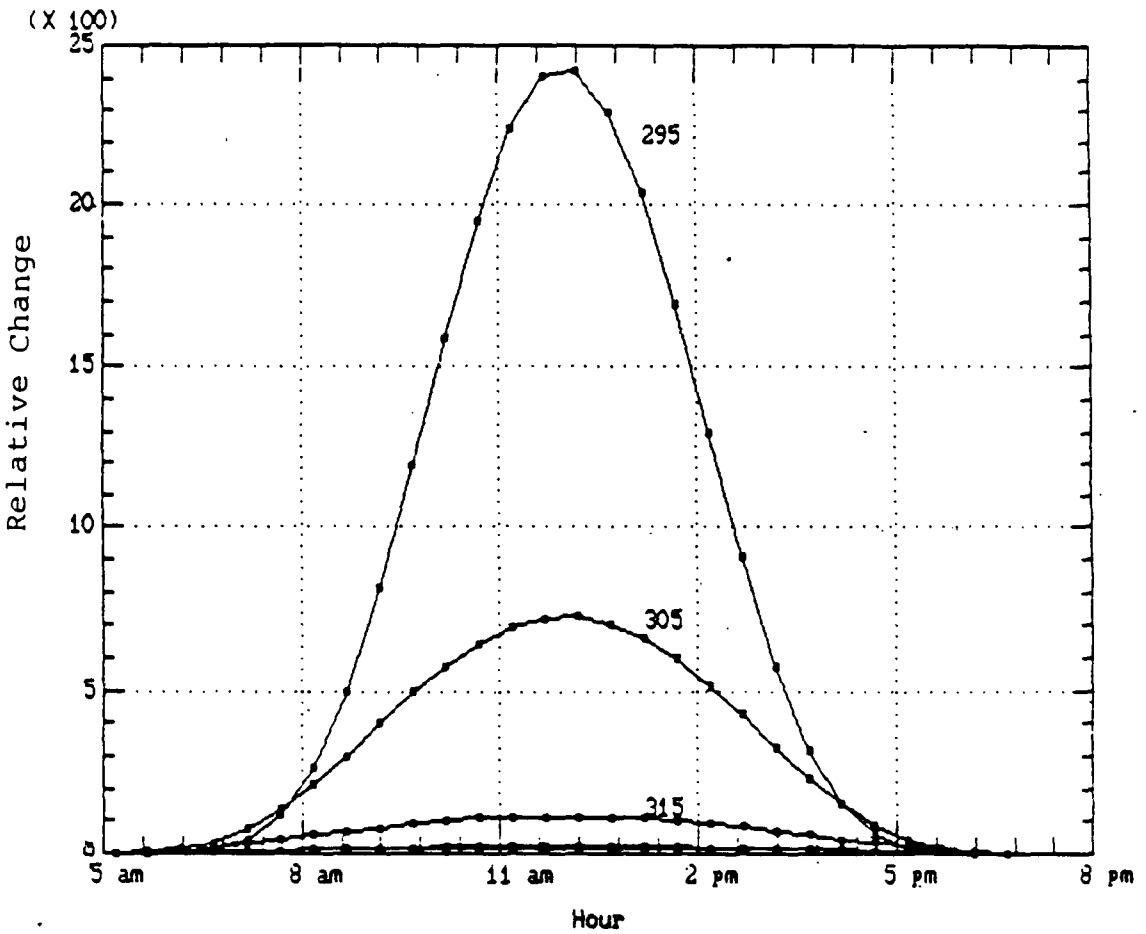
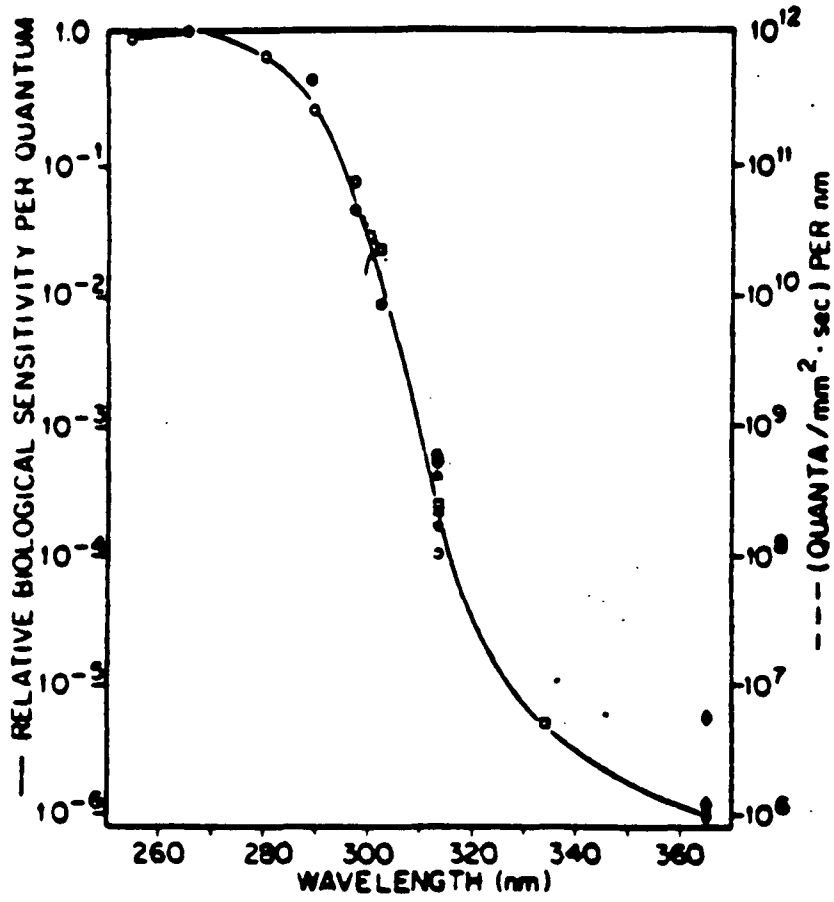


EXHIBIT 8-4

Average DNA Action Spectrum



Source: Adapted from Setlow 1974.

of exposure, not the actual exposure. Few individuals are out in the sun during all daylight hours, therefore actual exposure is correspondingly reduced. As indicated above, the seasonal and hourly variations in incident energy from solar radiation can be considerable particularly in those wavelengths that are the most biologically effective (295-299 nm) so all hours in the sun cannot be considered equal. In addition, people living in areas having the same number of sunlit hours may have additional behavioral differences that modify the amount of radiation reaching the skin (the actual exposure dose). For example, some people wear lots of clothing, others do not. Some people wear sunscreens, while others use sun reflectors to gather more solar radiation.

In addition, the transition from the amount of energy to which an individual is exposed to the amount of energy which is delivered to a target molecule (the biologically effective dose), involves the interplay of factors such as skin thickness and pigmentation. Keratin and melanin both absorb ultraviolet radiation. The greater the skin thickness and degree of pigmentation, the greater the amount of energy is prevented from penetrating the two potential targets. Thus earlier exposure to solar radiation and skin response can influence doses of solar radiation, by wavelength, reaching possible targets.

Background on Melanoma and the Concept of Response

Malignant melanoma is a rare tumor that arises as the result of the neoplastic transformation of a melanocyte. Melanocytes are pigment-producing cells. Exhibit 8-5 shows the relationship of the melanocyte to other cells in the epidermis and other skin layers.

The major function of the melanocyte is the production and distribution of melanin to keratinocytes -- the major cell population of the skin. Melanin is a pigment which absorbs UV light over a broad range of wavelengths (25 nm to 1200 nm); it is the principal chromophore responsible for the difference between black and white skin in the transmittance of UV light (shown in Exhibit 8-6).

The biology of cutaneous malignant melanoma is complex; there are several morphologic types of tumor that may have different pathways of histogenesis; they tend to behave differently in terms of age at appearance and characteristic location, and yet have common elements in their progression. Brief information is presented below; more detailed information is found in Appendix A.

Terminology for the four principal classes of melanomas was proposed by McGovern et al. (1973) and has been subsequently modified (Smith 1976; Elder et al. 1980). The four principal types of melanoma are given below:

(1) Melanoma arising in Hutchinson's melanotic freckle (HMF); it occurs predominantly on sites receiving the

EXHIBIT 8-5

Location of Melanocyte in the Epidermis

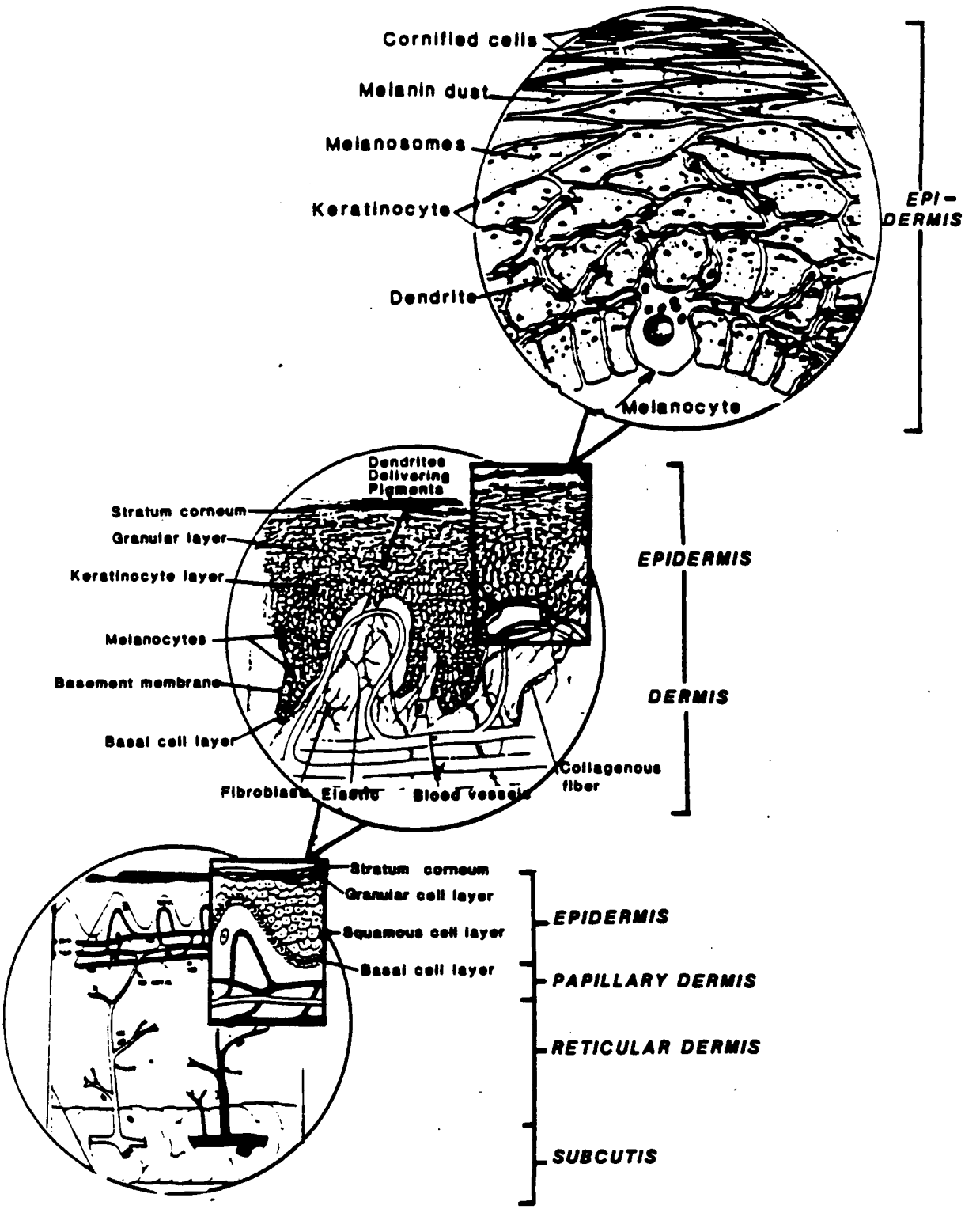
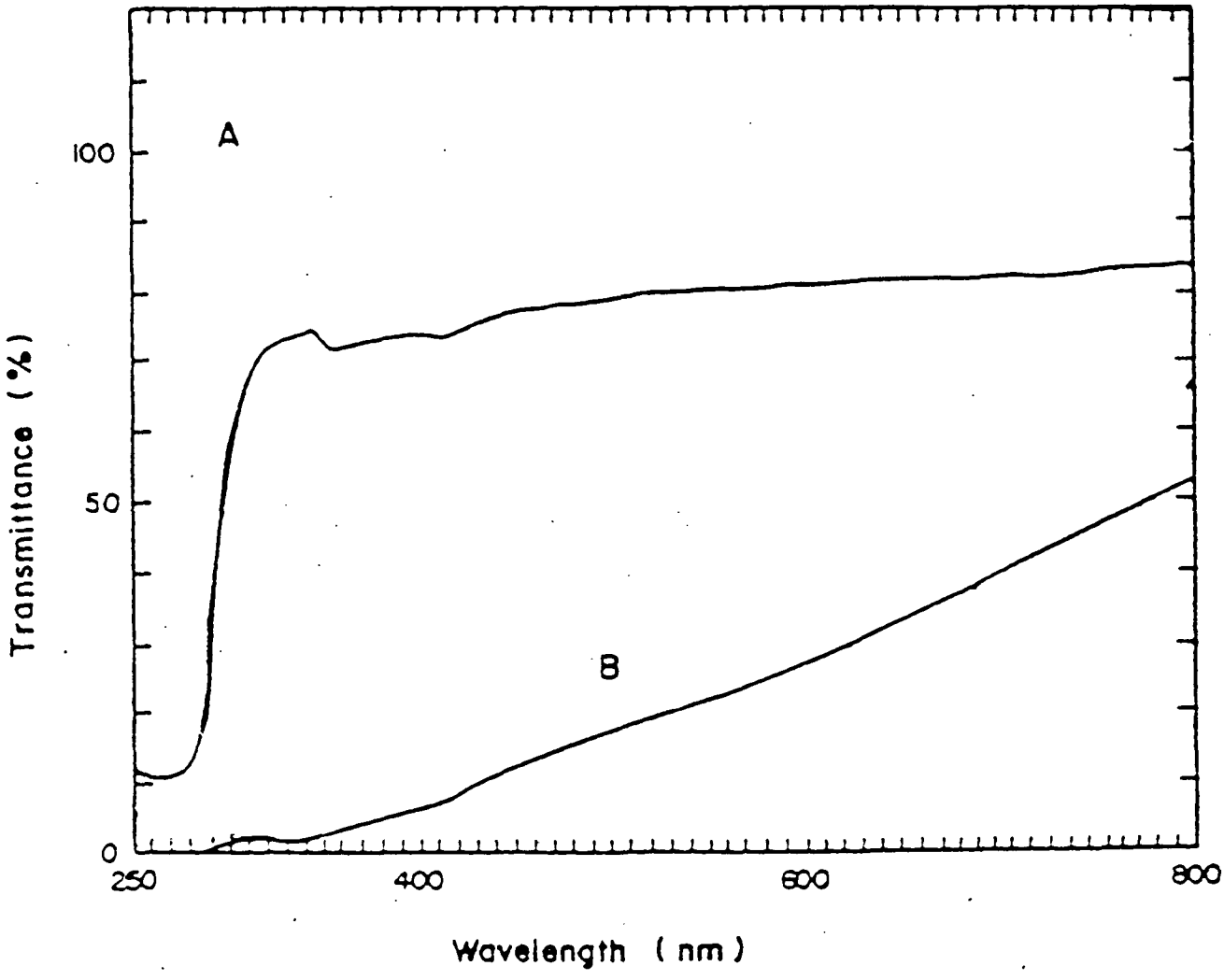


EXHIBIT 8-6

Comparative Transmittance of UV Radiation



Comparison of measured epidermal transmittance A, Caucasian; B, Dark Black.

Source: Wan et al. 1981.

greatest cumulative exposure and is thought to have the strongest relationship to cumulative solar exposure.

(2) Superficial spreading melanoma (SSM); it shows the strongest preference for sites that are intermittently exposed (trunks in males, lower extremities in females). It also shows a stronger association with nevi-possible precursor lesion.

(3) Nodular melanoma (NM); it has growth characteristics which differ from that of SSM and HMFM, although these types may progress to NM.

(4) Unclassifiable melanoma (UCM).

A fifth type of melanoma -- acral lentiginous melanoma (ALM) -- has been distinguished with a site preference for soles, palms, and subungual surfaces. It is virtually the only form observed in Blacks and in people who have never lived south of the Artic Circle (Clarke et al. 1986). Thus in characterizing response to dose, aggregation of all types of melanoma will tend to obfuscate real relationships.

EPIDEMIOLOGIC EVIDENCE

With the above background, this chapter will now review the epidemiologic and experimental evidence relevant to assessing the role of solar radiation and UVB in the development of CMM. Because of the extent of the information to be reviewed, the epidemiologic information has been described in five segments: time trends, anatomical site distribution, exposure factors, host factors, and miscellaneous factors. This is followed by a brief review of the experimental evidence and a discussion of possible dose-response relationships.

Time Trends

As indicated in Exhibit 8-7, there have been sharp increases in incidence and mortality due to CMM reported in Caucasian populations worldwide (Magnus 1982; Rousch et al. 1985b; Osterlind and Jensen 1986). Based on observational and analytical evidence, most experts agree that these trends in CMM incidence and mortality are genuine, and not due to increases in the registration and diagnosis of the disease (Magnus 1975; Pakkenen 1977; Malec et al. 1977).

Birth cohort analyses of CMM incidence rates across six nations show consistent increases in CMM for cohorts born between 1910-1930, whereas later birth cohorts (1940-1950) that show these increases do so to a lesser extent (Muir and Nectoux 1982). Most authors that have conducted cohort analyses of CMM incidence and mortality rates conclude that virtually all secular increases in CMM are due to cohort effects (Magnus 1981, 1982; Houghton et al. 1980; Muir and Nectoux 1982; Holman et al. 1980; Cooke et al. 1983).

EXHIBIT 8-7

Increases in Incidence and Mortality Rates From Malignant Melanoma in Different Countries

	Country	Sex	First Period of Observation		Second Period of Observation		Total Percent Increase	Number of Years	Average Annual Percent Increase*
			Time	Rate per 10^6	Time	Rate per 10^6			
Incidence	New York State	M	1941-1943	1.2	1967	3.4	176	25	7.0
		F	1941-1943	1.8	1967	2.9	65	25	2.6
Incidence	Norway	M	1955	1.8	1970	6.3	264	15	17.6
		F	1955	2.6	1970	6.8	195	15	13.0
Mortality	Norway	M	1956-1960	1.6	1966-1970	2.7	69	10	6.9
		F	1956-1960	1.3	1966-1970	1.8	36	10	3.6
Mortality	Canada	M	1951-1955	0.7	1966-1970	1.4	93	15	6.2
		F	1951-1955	0.6	1966-1970	1.2	107	15	7.1
Mortality	United Kingdom	Both	1950	0.5	1967	1.0	100	16	6.3
Mortality	Australia	M	1931-1940	1.0	1961-1970	3.6	267	30	8.9
		F	1931-1940	0.8	1961-1970	2.5	227	30	7.6
Mortality	Denmark	M	1956-1960	1.6	1966-1969	2.4	49	10	4.9
		F	1956-1960	1.6	1966-1969	2.1	32	10	3.2
Mortality	Sweden	M	1956-1960	1.7	1966-1968	2.1	30	9	3.3
		F	1956-1960	1.1	1966-1968	1.5	40	9	4.4
Incidence	Connecticut	M	1935-1939	1.1	1975-1979	8.2	645	40	16.1
		F	1935-1939	0.9	1975-1979	6.8	656	40	16.4
Incidence	U.S.A.	WM	1974	6.7	1983	9.6	43	10	4.3
		WF	1974	6.0	1983	8.3	38	10	3.8
Mortality	U.S.A.	WM	1950	1.0	1977	2.6	160	27	5.9
		WF	1950	0.8	1977	1.6	200	27	7.4

* Computed by dividing total percent increase in incidence or mortality rate by the number of years between the first and second periods of observations. The figures in this column do <u>not</u> represent compounded annual growth rates.

Adapted from: Elwood and Lee (1976); NCI (198); NCI (1985a); NCI (1985b).

In most countries, the first signs of increasing rates are seen in cohorts born around 1900, though increases in cohorts born as early as 1865 are observed in Australia and New Zealand (Holman et al. 1980; Cooke et al. 1983). In Norway and several other countries, there is a slight tendency for a slowing of increase in incidence in cohorts born around and after 1930 (Magnus 1981). Stabilization of mortality rates is also occurring in cohorts born 1925-1939 and later in countries such as Australia, New Zealand, England, Wales, and Finland (Lee and Carter 1970; Teppo 1978; Holman et al. 1980; Cooke et al. 1983).

Age-adjusted incidence rates of CMM of the head, neck, and face for birth cohorts born after approximately 1900 have not differed markedly; however, age-adjusted incidence rates of CMM of the lower extremities among females and the trunk among males have increased in successively younger birth cohorts born during the first half of this century (Magnus 1981; Houghton et al. 1980; Cooke 1984; Stevens and Mookjaukar 1984).

On the basis of the Connecticut Tumor Registry data, modeling of the CMM incidence rates observed in subsequent birth cohorts indicated that the incidence rate of CMM in the 1955 birth cohort will rival those for colon cancer, currently the third most common cancer in Connecticut (Rousch et al. 1985a).

Those anatomical sites that show the greatest amount of increase are not the sites that receive the greatest amount of cumulative solar exposure. This finding tends to lead to a conclusion that cumulative solar exposure is not causally associated with melanoma; as discussed in more detail below, cutaneous melanoma may be related to some other measure of exposure to solar radiation.

Anatomical Site Distribution

Cutaneous malignant melanoma has a unique anatomical distribution that has been the subject of numerous epidemiological investigations. Research efforts have focused on the site-specific trends in CMM incidence related to sex, age, race, histogenic type, birth cohort, and season. The most pronounced differences have been associated with gender, race, and birth cohort.

The overall site distribution is presented in Exhibit 8-8. There is wide variability among the studies presented in the exhibit, making it difficult to generalize about these data. However, as a rule, among white populations, the upper extremities have the lowest overall proportion of melanomas, while the lower extremities have the highest proportion. The variability is most probably due to differences among the study populations, such as their sex and racial distribution.

Gender is one of the factors associated with the most pronounced differences in CMM site distribution. Exhibit 8-9 lists data from 14 studies that reported CMM site distribution by sex. Data in the table indicate that most of the studies observed higher incidences of CMM on the lower extremities among females and on the trunk among males than on other parts of the body.

EXHIBIT 8-8

Anatomic Site Distribution of Cutaneous Malignant Melanoma
(Percentage of Total Tumors)

Location	Years	Sample Size	Head/Neck	Trunk	Extremities		Source
					Upper	Lower	
United States[a] (caucasian)	1978-1981	4,864	20	35	23	22	Scotto 1986
Texas	1944-1966	911	22	25	19	13	MacDonald 1976
Texas	1954-1970	510	25	16	21	37	Smith 1976
Alabama	1955-1980	537	27	28	19	23	Balch et al. 1982
New Mexico	1966-1977	403	27	29	19	25	Pathak et al. 1982
New South Wales	1955-1980	1,110	14	37	14	33	Balch et al. 1982
Queensland	1977	690	21	34	20	24	Little et al. 1980
New Zealand (Maori/Polynesian)	1963-1981	24	13	13	0	54	Moss 1984
Israel	1960-1972	966	16	25	21	38	Anaise et al. 1978
Norway	1955-1970	2,541	22[b]	43[c]	8	18	Magnus 1973
Finland	1953-1973	2,501	19	37	10	26	Teppo et al. 1978
Japan	1961-1982	546	15	20	13	46	Takahashi 1983
Hong Kong	1964-1982	43	7	7	21[d]	63[e]	Collins 1984
Uganda	1963-1966	152	8	11[f]	5	72[g]	Kiryabwire et al. 1968

[a] Based on data from Seattle, Detroit, Iowa, Utah, San Francisco/Oakland, Atlanta, and New Mexico.
[b] Face.
[c] Neck/truck.
[d] 17 percent of the total on the hands.
[e] 56 percent of the total on the feet.
[f] "Skin" and genitals.
[g] Feet and legs.

EXHIBIT 8-9

Anatomic Site Distribution of Cutaneous Malignant Melanoma by Gender

(Percentage of total tumors by gender[a])

Location (Ethnic Group)	Years	Sample Size	Sex	Head/Neck	Trunk	Extremities		Source
						Upper	Lower	
Predominantly Caucasian Study Populations								
United States	1980	4,545	F	15	31	19	35	Balch et al. 1984
			M	23	53	13	11	
United States[b]	1978-1981	4,864	F	16	25	26	33	Scotto and Fears (in press) 1986
			M	24	45	21	11	
North America (Caucasian)	-	-	F	16	29	18	36	Crombie 1981[c]
			M	27	44	17	12	
Europe (Caucasian)	-	-	F	16	23	13	47	Crombie 1981[c]
			M	23	42	13	23	
Texas (White)	1944-1966	1,252	F	17	18	25	28	MacDonald 1976
			M	23	25	20	11	
Hawaii (Caucasian)	1960-1977	262	F	9	28	28	35	Hinds and Kolonel 1980
			M	24	37	24	15	
New Mexico (Anglos)	1966-1977	403	F	22	20	21	37	Pathak et al. 1982
			M	33	41	16	10	
Israel	1960-1972	966	F	16	19	15	50	Anaise et al. 1978
			M	16	32	21	31	
Scotland	1961-1976	477	F	25	14	14	47	Pondes et al. 1981
			M	32	27	11	30	
Finland	1953-1973	2,501	F	18	28	11	36	Teppo et al. 1978
			M	19	48	9	17	
Denmark	1943-1957	1,204	F	24	19	14	33	Clemmesen 1965 (cited in Lee 1982)
			M	32	25	9	22	
Queensland	1977	713	F	19	21	22	35	Little et al. 1980
			M	23	45	17	13	
Texas	1954-1970	510	F	17	10	24	50	Smith 1976
			M	34	23	18	24	

EXHIBIT 8-9 (Continued)

Location (Ethnic Group)	Years	Sample Size	Sex	Head/Neck	Trunk	Extremities		Source
						Upper	Lower	
Mixed Study Populations								
Israel (Mixed Race)	1961-1967	368	F	15	21	15	42	Movshovitz and Modan 1973
			M	17	35	5	25	
Japan	1961-1982	546	F	20	19	18	42[d]	Takahashi 1983
			M	13	23	11	51[e]	
Texas (Spanish surname)	1944-1966	206	F	22	16	18	30	MacDonald 1976
			M	18	22	11	30	
Texas (Non-White)	1944-1946	30	F	6	6	18	53	MacDonald 1976
			M	31	--	8	54	
New Mexico (Hispanics)	1969-1977	35	F	23	36	32	9	Pathak et al. 1982
			M	23	46	23	8	
Hawaii (Non-Caucasian)	1960-1970	66	F	26	17	13	44	Hinds and Kolonel 1980
			M	16	19	19	47	
Hawaii (Non-Caucasian)	1960-1977	64	F	26	17	13	44[f]	Hinds 1979
			M	16	20	20	44[g]	
Uganda	1963-1966	152	F	6	11[h]	4	78[i]	Lewis 1967
			M	10	11[h]	5	74[j]	

[a] Percentages may not total 100 percent because of rounding errors and exclusion of "other sites" of MM.

[b] Based on data from Seattle, Detroit, Iowa, Utah, San Francisco/Oakland, Atlanta, and New Mexico.

[c] Truck includes scrotum and "unspecified" melanomas.

[d] 29 percent of the total were on the feet.

[e] 39 percent of the total were on the feet.

[f] 22 percent of the total were on the feet.

[g] 42 percent of the total were on the feet.

[h] "Skin" and genitals.

[i] 63 percent of the total were on the feet.

[j] 64 percent of the total were on the feet.

Most authors have concluded that the observed differences in site distribution by gender are not incompatible with the role of sunlight as a major etiologic factor (Pathak et al. 1982, Crombie 1981; Hinds and Kolonel 1980).

Race and gender also are associated with pronounced effects in site distribution of CMM. The predominant difference is a higher percentage of CMM occurring on the feet and, in some cases, on the hands of darker skinned ethnic groups. These groups have a much lower incidence than the light-skinned ethnic groups, so that the higher percentages on feet may not represent a higher incidence. Trauma has been suggested as a possible reason for the increased percentage of melanomas occurring on feet (Lewis 1967; Hinds 1979). Although in a recent review of the subject, Briggs (1984) concluded that there is no unequivocal evidence for a role of trauma in the vast majority of melanomas. One group has concluded that sunlight is not an important risk factor for melanoma (Hinds 1979; Hinds and Kolonel 1980) for any site among non-Caucasians.

There appear to be site-dependent differences in melanoma incidence by age, with melanomas of the face showing one pattern and melanoma of trunks and lower extremities, another. In several instances the differences have been identified as being between continuously exposed sites (e.g., head) and intermittently exposed sites (e.g., trunk and lower extremities). As a general rule the pattern observed for the exposed sites is characteristic of that observed for many other cancers -- a slow increase with age, up to the age of 40 or 50, followed by a rapid increase with age thereafter (Magnus 1981; Teppo 1982; Holman et al. 1980). For the intermittently-exposed sites, the pattern is quite different with a relatively steep increase which peaks between 40 and 60 (depending on the data set) and levels off or decreases thereafter (Magnus 1981a,b; Teppo 1982; Holman et al. 1980).

There are also differences in the anatomic distribution of melanomas associated with different histologic (histogenetic) types of CMM. HMFM and its precursor lesion, HMF, are observed predominantly on the face and other exposed sites, whereas SSM was most commonly observed on less exposed sites (trunk and lower extremities) (Holman et al. 1980, Smith 1976, Adler and Gaeta 1979, Pondes et al. 1981).

Very few studies have examined the relationship of geographic area to differences in a anatomic site distribution. In Norway, Magnus (1973) noted that the only site lacking a definite north/south gradient was the foot, and a comparison of Alabama patients to Australian patients noted that whereas melanomas of the trunk were the most common in both sets of patients, melanomas of the lower extremities were more common in Australians than in the Alabama patients, and head and neck tumors showed the reverse trend (Balch et al. 1982).

A final important parameter that has been associated with differences in site distribution of CMM is the temporal pattern of exposure. This is a complex parameter that is, as indicated earlier, difficult to separate from the observed age effects, but it is fairly clear that there are differences in the anatomic distribution of melanomas, which are associated with the year of

diagnosis and/or birth cohort. It is difficult to generalize, however, the conclusion drawn by Boyle et al. (1983) that there were cohort effects of melanoma incidence that differed by site and that the site effects differed by sex. The overall impression from the studies that looked at these cohort effects is, as well, that birth cohort-site differences are greater for sites that are intermittently exposed (e.g., trunk and lower extremities) than for those that are always exposed (e.g., face).

Exposure Factors

A number of studies have examined how various indicators or measures of exposure are related to melanoma incidence or mortality. This section reviews information from ecological studies that have looked at the correlation of melanoma incidence or mortality on the one hand, and geographical location and migration on the other. Studies that have examined the relationship between type of exposure (e.g., early, intermittent) and melanoma incidence are also discussed.

Geographic Location

Within predominantly Caucasian nations, most of the ecological studies of melanoma and latitude show increasing melanoma incidence and/or melanoma mortality with decreasing latitude, leading to the hypothesis that melanoma is associated with ultraviolet radiation because of the strong correlation between UV and latitude. As a general rule, those studies that failed to find this association did not adequately account for pigmentation differences (Crombie 1979) or had other serious methodological flaws (poor measurement of UVB) (Baker-Blocker 1980).

In general, melanoma rates (incidence and/or mortality) were found to be higher in areas closer to the equator, in coastal rather than inland areas, and in urban rather than rural areas within various nations (Holman and Armstrong 1984; Green and Siskind 1983; Magnus 1981a, 1981b).

Migrant Behavior

In an Australian study (Holman and Armstrong 1984), age at arrival in Australia was more important than duration of residence with respect to the risk of SSM, with arrival before age 10 incurring a risk approaching or exceeding the risk of SSM for those born in Australia. The odds ratio decreased when the age at arrival was 10-14 years and stabilized at a significantly lower level for those arriving at or after the age of 15.

Immigrants moving to sunnier climates in which the native CMM incidence rates exceed those of the immigrant's country of origin initially tend to have lower CMM risks than the native population. These risks increase, however, with increasing duration of residence or earlier age of arrival in the adopted homeland (Holman and Armstrong 1984).

Type of Sun Exposure

Early Exposures. CMM risk is associated with childhood sunburn (Green et al. 1985, Elwood et al. 1985b) but this association appears only to reflect an individual's pigmentary characteristics as related to poor sun tolerance. One study that evaluated the risk associated with outdoor work before college found a significantly elevated odds ratio for those who had worked outdoors compared with those who had not (Paffenbarger et al. 1978).

Intermittent ("Summer Sun") Exposure. One commonly examined surrogate for intermittent exposure has been a parameter related to a history of sunny vacations (Eklund and Malec 1978; Klepp and Magnus 1979; Adam et al. 1981). Different studies have used slightly different measures of this type of exposure behavior (e.g., one study evaluated those who "tanned themselves while on holiday abroad" (Adam et al. 1981), another evaluated those with a history of long (30 days or more), sunny vacations as a child (Lew et al. 1983). In general, however, there was a positive association between CMM risk and increasing number of "sunny vactions" taken.

The strongest evidence for an association between a measure of sunny vacation exposure comes from a case-control study conducted in Western Canada (Eldwood et al. 1985a, 1985b). Analysis of data from this study of 595 melanoma case-control pairs indicated that there was a significant ($p \leq 0.001$) trend of increasing risk of CMM which increasing numbers of sunny vacations per decade, with a relative risk of 1.8 associated with four or more sunny vacations per decade. In addition, this relative risk remained significant (RR = 1.7, 95% C.I.:1.2-2.3) even after controlling for significant host factors (hair color, skin color, history of freckles) and ethnic origin.

At least one study that evaluated a different measure of intermittent exposure found no significant association between intermittent exposure and an increased incidence of any histologic type of CMM (Holman and Armstrong 1984). This study of 507 pairs (matched according to sex, age, and residence) used as its surrogate for intermittent exposure a variable based on the ratio of recreational outdoor sun exposure in summer to total outdoor sun exposure in summer.

In an additional report of the same study (Holman et al. 1986), an increased SSM risk for some summer sun activities at early ages (from history or estimated) was observed for boating, fishing, and female sunbathing (on trunk). This study also found some increased risks for summer sun exposure by clothing habits. The primary CMM site was more frequently "sometimes exposed to sun" than usually exposed or usually covered, except for SSM, which showed increasing risk with increasing exposure. The risk of developing CMM of the trunk (especially SSM) was found to be greater for women whose bathing suits covered less of their bodies between 15 and 24 years of age.

A seasonal pattern with a summertime peak was found for CMM incidence rates (female in U.S. (Scotto and Nam 1980), both sexes in Hawaii (Hinds et al. 1981)). This seasonal pattern may be related to greater awareness of skin changes in summer months or could indicate promotional effects of UVB exposures.

Sunburn. A number of case-control studies that have examined the relationship of CMM to history of sunburn have found statistically significant associations. Most of these studies did not, however, adjust for pigmentary factors. One study that controlled for pigmentary factors found a significant association (Lew et al. 1983); however, in this study, biased selection of controls was likely, leaving this association in doubt. The conclusion drawn from studies that appeared to be subject to the fewest biases was that the presence or absence of factors associated with a tendency to sunburn, rather than the sunburn itself, determine the CMM risk.

A study from Western Australia (Holman et al. 1980) found significantly increased total CMM risk, SSM risk, and HMF risk with increased annual hours of bright sunlight at residence, with increasing actinic skin damage, and with previous nonmelanoma skin cancer.

Assessment of individual exposure through questionnaires. In Western Canada, an analysis of total sun exposure showed some increased risks in higher exposure groups compared with the lowest exposure group but none were statistically significant nor was there a significant trend of increasing risk (Holman et al. 1986).

In Queensland, Australia, elevated CMM risks were associated with increasing estimated total hours of sun exposure at ages 10 years and older after adjustment for exact age, presence of nevi on arms, hair color, and sunburn propensity. The confidence intervals for intermediate and higher levels of exposure include unity.

Indirect measures of cumulative sun exposure (i.e., assessment of numbers of hours exposed) show correlations with melanoma risk that were significant, borderline significant, and not significant. Several studies have evaluated a direct measure of received dose (i.e., actinic skin damage), which is also a measure of the skin's responsiveness to insult. In these studies, melanoma risk increased significantly with the degree of actinic skin damage.

Exposure During Sunspot Activity. Most studies that examined the relationship between CMM incidence rates and sunspot cycles found high correlations (cite studies). Different studies have observed differences in the lag time between the period of sunspot activity and changes in CMM incidence rates.

Most of the information presented above tends to support a relationship between melanoma and some function of increased exposure to solar radiation. What the exact function of sunlight exposure may be is unclear. There is some indication that early (childhood) exposure may be important, and other information implicates intensive exposure such as that which occurs during summertime and times of sunspot activity; however, no single kind of exposure clearly stands out. None of these studies considered UVB, which varies significantly with time of day. Thus, by using sun exposed hours as a dose variable, such studies may be inaccurately assessing dose, especially when they compare across occupations whose sun-exposure behavior may vary significantly.

Host Factors

There is clear evidence that certain host factors are associated with the risk of developing CMM. It is possible that the factors described below reflect the capability to modify the dose of solar radiation potentially received by a target cell or modify the ability of the cell to respond to insult.

Epidemiologic studies have shown that Caucasian populations have much higher rates of CMM incidence and mortality than other racial groups. In South Africa, CMM was approximately six times more frequent in whites than in blacks. Broken down by sex, the ratio of white to black CMM incidence was greater for males (13:1) than for females (4:1). Black/white differences in the United States population were even greater, although this may be due to differences in the quality of cancer registry data between South Africa and the United States. Based on 1983 SEER data, white:black ratios were 19:1 and 8:1 in males and females, respectively. In addition, whites in New Zealand experience much higher incidence rates than New Zealand Maoris and Polynesians. Likewise, American Indians experience much lower rates of CMM than American whites.

Within white populations, rates of CMM differ according to country of origin. CMM incidence rates for Hispanic whites in New Mexico, for example, are much lower than those for non-Hispanic whites; individuals from the Mediterranean countries in southern Europe tend to have lower rates than Caucasians from northern Europe; individuals of Celtic origin in Australia tend to have higher rates than non-Celtic individuals. Variation in the incidence of CMM within the Caucasian race is commonly thought to be a function of variation in genetically determined pigmentary traits across ethnic groups.

Numerous epidemiologic studies have focused on identification of important pigmentary characteristics in the etiology of CMM. Exhibit 8-10 shows a summary of findings from a number of these studies. The following associations between pigmentary traits and CMM risk have been found:

(a) Skin color -- in all studies reviewed fair complexions were associated with higher risks of CMM than were dark complexions.

(b) Hair and eye color -- in most studies red and blonde hair in childhood were associated with increased risk of CMM relative to dark hair. Blue eyes were an independent risk factor in only one of four well-controlled epidemiologic studies; however, this could be due to the homogeneous nature of most of the study populations.

(c) Freckling -- those who freckled readily were at consistently elevated CMM risk relative to other individuals.

EXHIBIT 8-10

Malignant Melanoma Risk Factors by Measures of Skin Pigmentation Within the Caucasian Population

Study Reference	Measures of Skin Pigmentation Within Caucasian Population					
	Skin Color	Hair Color	Eye Color	Freckling	Reaction to Sun (Tanning/ Sunburn)	Ethnicity
Lancaster and Nelson 1957	+	+	+		+	
Gellin et al. 1969	+	+	+		+/-	
Lane Brown et al. 1971						+
IARC 1976						+
MacDonald 1976						+
Klepp and Magnus 1979		-	-	+	+	
Mackie and Atchinson 1982					-	
Beral et al. 1983	+	+	-	+	+	
Hinds and Kolonel 1982						+
Lew et al. 1983		-	-		+	
Elwood et al. 1984	+	+		+	+	+
Holman and Armstrong 1984b	+	+	+	+	+	+
Graham et al. 1985	+	+	+		+	+

NOTE: + = Significant risk factor.
- = Not significant risk factor.
Blank = Not included in study.

(d) Reaction to sun exposure -- in most studies reviewed, individuals who usually burned and were unable to tan were at significantly higher risk of CMM than those who tanned easily.

Exposure to sunlight appears to encourage the appearance (and possibly the disappearance) of nevi (including dysplastic nevi) from the skin (Kopf et al. 1978, 1985; Armstrong et al. 1986). Dysplastic nevi are clearly a risk factor for melanoma independent of freckling or other pigmentary characteristics; it also appears that congenital nevi as well as acquired melanocytic nevi may also be risk factors (Elder et al. 1981).

Information from xeroderma pigmentosum (XP) patients indicates that individuals who have an inability to repair solar radiation-induced DNA damage also have a high incidence of CMM relative to the normal population (Kraemer et al. 1984). The best characterized defect in XP patients is an inability to excise pyrimidine dimers (Cleaver 1983) which suggests that the repair of such lesions can be important to prevention of CMM development.

Miscellaneous Factors

Although a number of possible etiologic agents for melanoma have been evaluated (e.g., exposure to chemicals or ionizing radiation), no strong candidate has emerged. It has been suggested that the risk of developing CMM may be elevated among individuals exposed to fluorescent lighting at work; however, several recent well-controlled studies have failed to find such an association. Indeed, one of these studies initially found a positive association based on personal interviews, but a self-administered postal survey of the same study subjects did not confirm the initial findings.

Total CMM incidence has been found to be higher among professional and administrative indoor office workers, but not among other indoor workers relative to outdoor workers; however, the implications of this finding may be confounded by differences in socio-economic status, melanoma site, and histologic type. There is a higher risk of indoor office workers developing CMM on an anatomical site that is usually covered (e.g., the trunk) than on a site that is usually exposed (e.g., the face). For usually covered parts of the body, the incidence of CMM among indoor office workers is higher than for outdoor workers. And the reverse is also true; for usually uncovered parts of the body, the incidence of CMM is higher among outdoor workers than among indoor office workers.

It is not clear why certain indoor workers are at greater risk for developing CMM than outdoor workers, nor why in these individuals the affected sites are those not normally exposed. One speculation is that indoor workers have more leisure time and that they spend that time under conditions of solar exposure that maximize intensive UVB exposures of areas that are not protected by a tan; however, there is no information currently available from a published study that would allow this hypothesis to be examined. From current data it is clear that for certain indoor workers, cumulative sun hours of

exposure that is not associated with melanoma risk. This information does not rule out that some other measure of exposure (e.g., number of high UVB exposures received at a time of low tan) may be associated with melanoma risk.

Weighing and Balancing the Epidemiologic Evidence

In summary, while there is no single piece of evidence which proves that solar radiation causes CMM, the weight of the available epidemiologic evidence clearly supports the conclusion that solar radiation plays a major role in the development of CMM for susceptible populations. Cumulative dose (i.e., total sun exposed hours) clearly does not explain important variations in incidence rates. Although, from existing evidence it is not possible to determine on the most accurate method of calculating dose, the collective evidence does suggest that exposure to solar radiation in some form produces cutaneous malignant melanomas in Caucasian populations.

EXPERIMENTAL EVIDENCE

Much of the experimental evidence relevant to the relationship of CMM and exposure to UVB has been reviewed on non-melanoma skin cancer. To briefly recapitulate, there are five important points:

(1) UVB is the most active portion of solar radiation responsible for the induction of adverse effects on mammalian systems; it has been shown in vivo and in vitro to induce transformation of mammalian epidermal cells and to be mutagenic to them as well. All of these effects are thought to occur by a mechanism that involves damage to the DNA (Doniger et al. 1981)

(2) UVB at 295-300 nm is the most active waveband of UVR for these effects, as well as for the induction of pyrimidine dimers.

(3) Patients with xeroderma pigmentosum, who cannot repair pyrimidine dimers induced in DNA by UVB (which are thought to be important to skin cancer development) have significantly higher rates of CMM than normal individuals.

(4) UVR is carcinogenic in animals and it is the UVB wavelengths that are the most effective at inducing cancer -- the shorter UVB wavelenths being more carcinogenic than the longer ones (Forbes et al. 1981); the tumors induced are principally fibrosarcomas and squamous cell carcinomas.

(5) UVR, and specifically UVB, can induce the generation of T suppressor cells, which specifically suppress the immune response to UV-induced tumors. The suppressor

cells have been shown to be capable of shortening the latest period of tumors induced by UVB, and UVB-treated animals also appear to have increased susceptibility to transplanted melanomas.

Weighing and Balancing the Experimental Evidence

The experimental data indicate that UVB irradiation damages DNA and induces carcinogenesis and immunosuppression in animals. CMM rates are elevated in patients who cannot repair UVR induced lesions in DNA.

DOSE-RESPONSE RELATIONSHIPS

Dose-response relationships between exposure to UVB and the incidence and mortality of CMM have been developed in several epidemiologic studies.

Animal studies are inadequate for estimating CMM dose-response because there is no animal model for melanoma induced by UVB. Because the limitations in animal studies appear insurmountable at this time, epidemiologic studies provide the best candidate dose-response relationships for risk estimation. This section reviews four such studies. Three of the studies -- Fears, Scotto, and Schneiderman (1976); Fears, Scotto, and Schneiderman (1977); and Scotto and Fears (1986) -- develop dose-response relationships associating UVB and melanoma incidence. Two studies -- Fears, Scotto, and Schneiderman (1976) and Pitcher (1986) -- estimate dose-response relationships for melanoma mortality.

Fears, Scotto, and Schneiderman (1976) present one epidemiologic analysis that is potentially useful for estimating dose-response. These authors use two types of data to represent UVB: (1) latitude -- latitude and UVB radiation weighted for erethyma effectiveness correlate at 0.97, and (2) monthly totals of erethyma-producing UV radiation expressed as Biologically Effective Units (BEU) and derived from Schultze (1974). Both types of data were correlated with CMM mortality and incidence data. Incidence information for four cities was obtained from the Third National Cancer Survey (TNCS) (1975), and mortality data were from the U.S. Cancer Mortality by County database (Mason and McKay (1973).

Exhibit 8-11 shows the results of a simple correlation model based on latitude:

$$\log R = \alpha + \beta L$$

where R is the age-adjusted rate of incidence or mortality, α and β are constants, and L is latitude. The estimated coefficients were statistically significant at $p \geq 0.01$. Exhibit 8-12 shows the results estimated using BEU's. These estimates were based on an exponential model and represent the percentage changes in incidence and mortality estimated to occur with increases in UV radiation dose of between 10 and 30 percent. Note that the use of an exponential model results in higher dose-response relationships for higher base exposures.

EXHIBIT 8-11

Summary Statistics for Regressions of Skin Cancer Incidence and Mortality on Latitude

	MALES			FEMALES		
	Correlation Coefficient*	Regression Slope±S.D.	Doubling Latitude	Correlation Coefficient*	Regression Slope±S.D.	Doubling Latitude
Melanoma Incidence	-.86	-.031 ± .007	- 9.8°	-.83	-.038 ± .007	-10.7°
Melanoma Mortality	-.81	-.017 ± .002	-19.9°	-.71	-.014 ± .002	-22.2°

*Simple correlation coefficient between log of incidence/mortality rate and latitude.

Source: Fears, Scotto, and Schneiderman (1976).

EXHIBIT 8-12

Estimated Relative Increases in Melanoma Skin Cancer Incidence and Mortality Associated with Changes in Ereythema Dose a/
Fears, Scotto, and Schneiderman (1976)
(Figures in Parentheses are 95 Percent Confidence Intervals)

	MALES				FEMALES		
	BASE BEU	Increase in Total Dose 10%	20%	30%	10%	20%	30%
Melanoma Incidence							
	650	15 (7-24)	32	52	13	29	46
	850	20	44 (18-75)	72	18	39	64
	1050	25	57	96 b/ (37-180)	22	50	84 b/
Melanoma Mortality							
	650	8 (6-10)	16	26	6	13	21
	850	10	22 (16-28)	35	9	18	28
	1050	13	28	45 (33-58)	11	22	36

a/ A sample computation is as follows:
A 10% increase in total dose at 48.25'N, where exposure is 650 BEU, equals 65 BEU.
A change of 65 BEU is equivalent to a reduction in latitude of 1.93'.
A change of 1.93' at 48.25'N is associated with an 18% increase in nonmelanoma incidence.

b/ These estimates require extrapolation well beyond the range of the data.

Source: Fears, Scotto, and Schneiderman (1976).

There are several important limitations in this work. In particular, only four cities were used for melanoma incidence. With a sample this small, variations in confounding factors such as behavior, skin pigmentation, and cloud cover may bias dose-response estimates. As a result, caution must be used in evaluating the error of estimate.

In another study, the same authors estimated a dose-response relationship for melanoma incidence using a power model (Fears, Scotto, and Schneiderman 1977):

$$P_{ij} = b\ (U_i)^c\ (A_i)^k$$

where:

P_{ij} = probability of developing melanoma for jth age group at location i;

U_i = annual UVB count at ith location;

A_j = midrange of jth age group; and

b, c, and k are constants.

This equation was estimated by fitting a log form of the model ($\ln R_{ij} = a + c \ln U_i + kLnA_i + E_{ij}$) to age-specific melanoma incidence rates for both males and females after weighting by the observed number of cases. Annual counts from Robertson-Berger (RB) meters were used for UVB; the RB meter attaches greater weight to longer wavelengths compared to the erythema action spectrum. Incidence data also were from the TNCS (1975).

Exhibit 8-13 presents summary regression coefficients and statistics for this age-specific analysis. Applying the results in this exhibit, a one percent increase in UV radiation results in an estimated $100*((1.01)^c-1)$ percentage increase in melanoma incidence, or 2.47 for females and 2.24 for males. The authors stress, however, that these estimates may be biased by the omission of location-specific demographic and environmental variables.

Scotto and Fears (1986) estimate a dose-response relationship for melanoma incidence using updated information about populations in a greater number of cities. The authors also introduce a new term (VAR) to adjust for the presence of some host or environmental characteristics:

$$Ln\ R_{ij} = a + b \ln (Age_i) + c \ln UVB + dVAR + e$$

EXHIBIT 8-13

Summary of Fears, Scotto, and Schneiderman (1977) Regression Analyses of Melanoma Incidence Dose-Response*

SEX	R-Squared	t-Statistic (UV Coefficient)	$\alpha \pm$ SD (constant)	$\kappa \pm$ SD (age coefficient)	$c \pm$ SD (UV coefficient)
Males	0.74	5.4	-35.6 + 6.5	.80 + .31	2.45 + .45
Females	0.62	4.6	-30.5 + 6.9	.29 + .34	2.23 + .48

* $\ln R\mu = \alpha + c \ln U_i + \kappa \ln A_j + \varepsilon\mu$

Source: Fears, Scotto, and Schneiderman (1977).

Annual counts obtained from Robertson-Berger meters were used for UV-B. Seven areas were included: Detroit, Seattle, Iowa, Utah, San Francisco, Atlanta, and New Mexico. Separate estimates were provided for: (1) anatomical sites of melanoma -- FHN (face, hands, neck), UE (upper extremity), LE (truck and lower extremity); and (2) age groups -- 20-39, 40-54, 55-64, 65-74.

Scotto and Fears (1986) found that after controlling for confounding variables each 1 percent increase in UVB causes melanoma incidence to increase by less than 1 percent. Exhibits 8-14 and 8-15 show results for different anatomical sites. The results are statistically significant ($p<0.1$). Exhibit 8-12 describes the effects of adding different constitutional and environmental variables derived using step-wise multiple regression. If the DNA action spectrum is used, Scotto's results indicate slightly less than a 2 percent increase in morbidity for each 1 percent ozone depletion.

Pitcher (1986) related data on melanoma deaths from the EPA/NCI data base (U.S. EPA, 1983) to exposure data obtained from a model developed by the National Atmospheric and Space Administration (NASA). The NASA model provided estimates of UV-B radiation for alternative locations measured at different times and using various biological action spectra.

Pitcher fit two different models for each sex. Defining DRM_{ijt} as the death rate for the ith cohort, the jth SMA (location), and the kth time period, he estimated:

$$DRM_{ijk} = \exp(b_0 + b_i AGE_{ik} + b_j EXP_j) + e$$

where AGE_{ik} is the age of ith cohort in the kth time period and EXP_j is the exposure in the jth SMA. In this model, the percentage change in the death rate that results from a given percentage change in exposure depends on the baseline exposure. The percentage change in death rates is higher the greater the baseline exposure.

The second model differed from the first only by using the log of the exposure variable. In this model, a one percent increase in exposure generates approximately the same percentage increase in the mortality rate regardless of the baseline mortality.

Using UVB radiation data from the NASA model for a clear day in June and measured with the DNA action spectrum, Pitcher estimated constants on the exposure term in the second formulation to be 0.85 for males (standard error 0.067) and 0.58 for females (standard error 0.078). The clear day values of UV radiation can be considered peak estimates. With these constants, a 1 percent increase in UV radiation results in an estimated increase in melanoma mortality of about 0.85 percent for males and about 0.58 percent for females. Exhibit 8-16 extends the estimates to larger relative changes in UVB. Since the optical amplification factor for DNA is 2 percent increase in UVB for a 1 percent ozone depletion, the Pitcher data indicates that there would be a 1.70 increase for males and a 1.16 for females in melanoma mortality for a 1 percent depletion.

EXHIBIT 8-14

Biological Amplification Factors* for Skin Melanoma by Sex and Anatomical Site Groups, Adjusting for age and Selected Constitutional and Exposure Variables

VARIABLE	MALE		FEMALE	
	Trunk/LE	FHN/UE	Trunk/LE	FHN/UE
Age	6%	8%	5%	10%
Sunburn	4	8	5	10
Freckles	5	7	5	10
Scandinavian ancestry	5	9	5	10
Lt. hair color	5	8	5	10
Scot/Irish ancestry	6	6	4	8
Moles	6	7	5	11
Lt. eye color	6	8	4	9
Fair skin	6	8	4	9
Sunscreen use	4	7	4	10
Suntan lotion use	5	8	4	10
Radiation protection	5	8	6	10
Protective clothes	8	10	5	10
Hours outdoors during weekdays	7	9	5	10
Hours outdoors during weekends	5	7	8	13

*A biological amplification factor indicates the relative change in melanoma incidence associated with a 10 percent relative increase in UVB.

Source: Scotto and Fears (1986).

EXHIBIT 8-15

Biological Amplification Factors* for Melanoma Incidence by Sex and Anatomical Site Groups, Adjusting for Age and Combinations of Selected Constitutional and Exposure Variables

ANATOMICAL SITE	MALE	FEMALE
Trunk/LE	3 Percent	4 Percent
Variables included in model	Suntan lotion use Scandinavian Lt hair color UVB radiation index Hours outdoors during weekdays	Suntan lotion use Scandinavian Lt hair color UVB radiation index Hours outdoors during weekends
FHN/UE	5 Percent	6 Percent
Variables included in model	Scot/Irish Suntan lotion use Fair skin UVB radiation index Hours outdoors during weekends	Scot/Irish suntan lotion use fair skin UVB radiation index

*A biological amplification factor is a number that indicates the percentage increase in melanoma incidence for a 10 percent relative increase in UVB.

Source: Scotto and Fears (1986).

EXHIBIT 8-16

Estimated Percentage Change in Melanoma Mortality for Different Percentage Changes in UVB (DNA Action Spectrum for a Clear Day in June)

PERCENTAGE INCREASE IN UVB	MALES	FEMALES
1	0.85	0.58
2	1.7	1.2
5	4.2	2.9
10	8.4	5.7
20	17.0	11.0

Source: Derived from results of Pitcher (1986).

In the analyses done by Pitcher, the question was raised regarding which measure of UVB dose would be most appropriate for estimating mortality risks in the event of ozone depletion. The regression analysis showed a more significant statistical relationship using the DNA action spectrum for peak values (June 15th) as the exposure variable, as compared to using other action spectra (e.g., erythema and RB) and/or other time periods (e.g., annual cumulative values or cumulative values for June, which would be another way to measure peak values). It must be emphasized, however, that the statistical differences due to the different exposure measures were extremely small. Therefore, the decision regarding which dose measure to use is primarily judgmental, and should be based inter alia on hypotheses about disease etiology. While peak values were selected as the central case for analyses in Chapter 18, the question exists as to whether this approach overestimates the possible risks of ozone depletion. Using peak UVB values as the exposure measure may overstate these risks because the variability of peak values across locations is less than the variability of UVB measured over different time periods. The lower variability of the peak values results in larger estimates of the responsiveness of melanoma mortality to changes in UVB for this exposure measure. Consequently, the appropriate action spectrum for estimating dose-response for melanoma mortality is an area requiring future research and sensitivity analyses should be included in assessments of melanoma mortality.

Limitations of Epidemiologic Studies

A variety of problems exist in applying the results of the epidemiological studies to an analysis of melanoma risks associated with ozone depletion; many of these problems have been described in earlier sections. In particular, applying the dose-response relationships requires an assumption that certain key factors will not change. However, the proportion of individuals in the population who are most susceptible to UV radiation, such as certain ethnic groups, may change over time. Migration was not considered in the studies; if southern states experience large increases in population then the sensitivity of future populations to UVB will be underestimated. Individuals might also change their use of sunscreens or their propensity to work outside work habits.

The epidemiologic studies do not measure actual ambient UVB dose. Therefore, UVB data used to estimate dose-response relationships will differ from the actual potential ambient exposure of individuals at any location. Other factors will also affect actual ambient exposures, such as skin color, patterns of dress, work exposure, recreation, eating, and medical care and intervention. These factors can be expected, in varying degrees, to introduce unexplained variation (i.e., noise) into any analysis of incidence or mortality. Finally, few data are available to assess the significance of genetic factors that effect melanoma incidence but are not captured in epidemiologic studies.

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CHAPTER 9

EFFECTS OF SOLAR RADIATION ON THE IMMUNE SYSTEM AND RESISTANCE TO INFECTIONS

SUMMARY

Solar radiation has been found to have a detrimental effect on the immune system of both humans and experimental animals. In particular, UV radiation (UVR) reduces the ability of the cell-mediated arm of the immune response to respond adequately to antigens. UV irradiation of the skin results in the generation of cells which suppress the immune response to an antigen encountered via the skin. In a tumor bearing animal, this immunosuppression may result in the outgrowth of tumor cells which in normal animals would have been destroyed by the immune system. The cells in the skin that seem to be the most severely damaged are the Langerhans cells. These cells are necessary for the generation of an adequate immune response to those antigens which are introduced through the skin. Although it is not certain that the damage to the Langerhans cells is entirely responsible for the effect, it is clear that UV irradiation of skin reduces the immune response in that skin. Even more important, it is also clear that it is the UVB portion of the UV spectrum that is responsible for the depression of the immune response.

One important facet of this UVR-induced immunosuppression is the role it plays in UVB-induced carcinogenesis; it affects the host's ability to recognize tumor-specific antigens present on the UVR-induced tumor cells thereby increasing the risk for tumor development in animal models, probably by allowing the tumor to escape the normal immune surveillance mechanisms. Thus the tumor cells are allowed to divide and establish a large and growing tumor in the host.

The immunosuppressive effects of UVR also are very likely to have a deleterious effect on the immune response to those infectious diseases that enter through the skin, especially if the initial immune response to the agent takes place in the skin. As yet, little research has been done in this area. Preliminary evidence indicates, however, that UV irradiation during a first cutaneous infection with two very different organisms, the parasite Leishmania sp and Herpes simplex virus, may result in an impairment of the immune response of the host to subsequent infections. In the case of leishmaniasis, this could lead to the development of the more lethal form of the disease, visceral leishmaniasis.

Although there are no experimental data which specifically address this issue, all populations may be at risk from the immunosuppressive effects of UVR. In addition, individuals who are already immunosuppressed, e.g., transplant patients or individuals with immunodeficiency diseases, may be at greater risk. In developing countries, particularly along the equator, immunosuppressive effects occurring as the result of high solar insolation may exacerbate parasitic infections of the skin such as leishmaniasis.

The effects of UVB and solar radiation on the human immune system have not been studied in sufficient detail to allow estimation of dose-response relationships for these effects. Qualitatively, it is known from animal studies that the doses of UVB needed to induce immunosuppression are much lower than those required for carcinogenesis. This may mean that exposure to low doses of UVR, even doses that do not cause a sunburn, may decrease the ability of the human immune system to provide an effective defense against neoplastic skin cells or skin infections.

FINDINGS

1. UV-B SUPPRESSES THE IMMUNE SYSTEM IN ANIMAL EXPERIMENTS.

   1a. Acute high doses of UV radiation cause a panimmunosuppression which is short lived.

   1b. Low dose UV radiation also causes a depression in cell-mediated immunity which results in an inability to respond to an antigen which is presented to the animal through the UV irradiated skin, a depression of contact hypersensitivity.

   1c. Chronic UV radiation or high doses of UV radiation cause a depression in cell-mediated immunity, delayed-type hypersensitivity, that results in an inability of the animal to respond to an antigen which is presented ot the animal through unradiated skin.

   1d. Both the effects of the local depression of contact hypersensitivity and the systemic suppression of contact hypersensitivity are mediated by a T suppressor cell which prevents the development of active immunity to the antigen.

   1e. The immunosuppressive effects of UVR have been found to reside almost entirely in the UVB wavelengths.

2. SUPPRESSION OF THE IMMUNE SYSTEM MAY PLAY AN IMPORTANT ROLE IN CARCINOGENESIS.

   2a. Animals which are UV-irradiated also develop T suppressor cells which interfere with the immune response to UV-induced tumors in such a way that the animals are more susceptible to the growth of autochthonous UV-induced tumors.

3. LIMITED EXPERIMENTAL DATA INDICATES UV-B SUPPRESSES THE HUMAN IMMUNE SYSTEM.

   3a. Although there is not as much information on the effects of UVR on humans, there are several studies indicating that the immune response of humans is depressed by UVR and is depressed in UV-irradiated skin.

4. UVB-INDUCED SUPPRESSION OF THE HUMAN IMMUNE SYSTEM IS LIKELY TO HAVE A DELETERIOUS EFFECT IN MANY HUMAN DISEASES.

   4a. Preliminary studies indicate that UVR may interfere with the earliest immune response to microorganisms that infect via the skin and prevent an effective immune response, thus predisposing to reinfection or chronic infection.

   4b. Two human diseases that may be influenced by UVB-induced immune suppression are Herpes virus infections and leishmaniasis.

4c. Almost no research has been conducted on the influence of UVB on infectious diseases; additional investigation is clearly warranted.

4d. For at least one theory of the mechanisms of UVB suppression of the immune system (that involving uralonic acid), a possibility exists that non-whites, as well as whites, would be vulnerable toincreased human suppression caused by ozone depletion.

INTRODUCTION

As indicated in Chapter 7, it has been known for years that ultraviolet radiation is carcinogenic. In the past decade or so, studies designed to explore the mechanism of UVR-induced carcinogenesis revealed that UVR was a potent but specific immunosuppressive agent which could abrogate the ability of an animal to reject its own or a transplanted syngeneic UV-induced tumor. Subsequent work investigating the mechanism of UVR-induced immunosupression has indicated that UVR can have a number of effects on the immune system and that these effects may modify a variety of disease processes. It is the goal of this chapter to review information on the effects of UVR on the immune system, focusing first on what is known about the characteristics of UVR induced immunosuppression followed by a brief review of what is known about UVR's effects on the immune system as they relate to other cutaneous diseases and then finally by a more cursory review of the impact of UVR on a variety of other diseases, many having an allergic component.

EFFECTS OF ULTRAVIOLET RADIATION ON THE IMMUNE SYSTEM

Immediately after acute doses of UV irradiation, there is a suppression of some immune parameters but after chronic UV irradiation most immune functions return to normal. However, research has provided evidence that the effect of UVR on the immune response early in the course of UV treatment may lead to events that allow initiated cells to escape immune surveillance and grow into tumors in a sequence of events unique to UV carcinogenesis.

Several parameters of immune function have received the greatest attention, and they will be emphasized here; these are the effects of UVR on tumor growth, the suppression of contact hypersensitivity and delayed type hypersensitivity in vivo, and the effects on antigen presentation in vitro. However, other effects have been noted and will be briefly discussed. This chapter will discuss briefly the effects of UVR that can be seen immediately after treatment, i.e. the acute effects of UVR on the immune system and then the effects of chronic UV irradiation on the immune system. The latter two immune functions are not identical, but are very similar. In fact, all the immune functions that are depressed by chronic UV irradiation fall under the category of cell-mediated immunity, meaning that the effector mechanism involves a cell rather than an antibody. Contact hypersensitivity is the response that is generated when a person is sensitive to poison ivy. The cells responsible for the sensitivity are T cells, lymphocytes which require a period of residence in the thymus during development to gain functionality. The cells that actually produce the effect, such as the rash in poison ivy sensitivity, are called effector cells and, in this case, are T cells. Other T cells also take part in the immune response and can either act to suppress the immune response, suppressor T cells, or allow its expression, helper T cells.

Effects of UVR on Tumor Growth

The first indication that UVR might have an impact on the immune system came from studies by Kripke (1974) who reported that most murine tumors induced by UVR were rejected when transplanted into normal syngeneic

recipients whereas the same tumors grew progressively in immunosuppressed mice. This phenomenon was originally identified in a C3H mouse system in which tumors were induced using a high intensity mercury arc lamp (Kripke 1974), subsequent studies indicated that it held true for other inbred strains of mice irradiated using FS40 fluorescent sunlamps (Kripke 1977).

Experiments designed to determine if the mechanism of this effect was that UV irradiation of mice led to a generalized immunosuppression, evaluated a variety of immune parameters using a number of different exposure regimens (Kripke et al. 1977; Spellman et al. 1977; Norbury et al. 1977; Lynch and Daynes 1983). With a few exceptions, all of the functions examined (e.g., response to mitogens, allograft rejection, antibody production) remained normal. The exceptions included: a)when UV-irradiated mice were used as recipients in a graft-versus-host assay, there was a small but significant decrease in reactivity after 2 and 3 months of irradiation which returned to normal at 4 months (Kripke et al. 1977); b) Reactivity to dinitrochlorobenzene (DNCB) was depressed after 1 and 2 months of UVR but then also returned to normal (Kripke et al. 1977) and c) naturally occurring cell-mediated cytotoxicity in UV-irradiated mice was found to be transiently suppressed 6 days after UV radiation but was found to be normal after 5 to 10 weeks of UVR (Lynch and Daynes 1983).

It is clear from the above information that mice which have been chronically irradiated with UV light are not generally immunosuppressed but have normal immunity when tested over a very wide number of immune functions. However, they have a specific immune deficit and are unable to reject UV-induced tumors.

Further experiments revealed that the mechanism for the lack of immune reactivity to UV-induced tumors in UV-irradiated mice was a radiosensitive, Ia positive, Lyt-1+2- (Ullrich and Kripke 1984) T suppressor cell (Fisher and Kripke 1977, 1978, Spellman and Daynes, 1977, 1978, Daynes et al, 1979). These cells were specific for an antigen common to UV-induced tumors and did not affect the ability of immune mice to reject tumors as a function of immunity to tumor specific transplantation antigens (Roberts et al 1980) or to histocompatibility antigens (Kripke and Fisher 1976). Preliminary evidence indicates that UV-irradiated mice immunologically recognize UV-induced tumors (Romerdahl and Kripke, 1986), but that the suppressor cell acts to prevent the development of cytotoxic effector cells.

The suppressor T cells were found to be specific for UV-induced tumors, with one exception. The presence of UV-induced tumor specific suppressor cells was not found to interfere with rejection of transplanted syngeneic tumors (Kripke et al 1979) induced by methylcholanthrene and Moloney sarcoma virus as well as spontaneous tumors. Interestingly, growth of only one spontaneous tumor, B16 melanoma, was enhanced in UV-irradiated mice. The enhanced growth of B16 melanoma in UV-irradiated mice was confirmed by Bowen and Brody (1983) who reported an increased percentage of challenged mice which bore tumors and decreased tumor latency. It was also found that one can achieve the same effects on the transplantation immunity to UV-induced tumors with sunlight as with those artificial sources of UV radiation used in experimentation (Morrison and Kelley 1985).

Experiments in which plastic filters were used to remove wavelengths from the emission of Westinghouse FS40 sunlamps indicated that the ability of UVR to induce susceptibility to UV-induced tumors lay in wavelengths below 315 nm, for as the lower wavelengths were removed from the UVR, the effectiveness of the UVR in inducing susceptibility to UV-induced tumors decreased. Wavelengths above 315 nm were ineffective in inducing the susceptibility. Reducing the dose rate through neutral density filters while keeping the total dose constant did not change the proportion of mice which were tumor susceptible; therefore there was reciprocity with regards to dose-rate and time of UV exposure (De Fabo and Kripke, 1979, 1980).

It has also been shown that UV irradiation can affect the latent period and antigenic properties of tumors induced by other carcinogenic agents including benzo(a)pyrene and methylcholanthrene (Roberts and Daynes 1980). Ebbesen (1981) reported an enhanced incidence of lymphomas in BALB/c mice which had been UV-irradiated.

The role that UVR induced immunosuppression plays in tumorigenesis by UVR has been investigated by a number of laboratories (Fisher and Kripke 1982; De Gruijl and Van Der Leun 1982, 1983). In a series of experiments, it was found that UV-induced tumorigenesis was enhanced in those mice reconstituted with lymphoid cells from UV-irradiated mice as compared to mice reconstituted with cells from normal mice (Fisher and Kripke 1982). It was also found that preirradiation of mice enhanced subsequent UV tumorigenesis in previously unexposed skin (De Gruijl and Van Der Leun 1982, 1983), and that ventral UVB irradiation greatly enhanced the susceptibility of mice to dorsal two-stage carcinogenesis (Strickland et al 1985). Thus it is clear that UVR induced immunosuppression (and thus the presence of the T-suppressor cell) is of great importance in determining if a tumor will develop in response to the carcinogenic action of UVR. This implies that immune surveillance plays a role in retarding the growth of transformed cells and thus may at least partially account for the long latent period of those tumors.

Effects of UVR on Other Immune Parameters

Suppression of Contact Hypersensitivity

In subsequent investigations of the impact of UVR on immune responses, it was discovered that UVR treatment resulted in a decreased capacity to elicit contact hypersensitivity (CHS) responses to antigenic chemicals applied directly to the skin. Low doses of UVR result in a local suppression of contact hypersensitivity and animals may not be sensitized directly through UV-irradiated skin although they are normal in their responses when sensitized through skin not exposed to UVR. Larger doses of UVR result in a systemic suppression of contact hypersensitivity and animals are hyporesponsive regardless of the site of antigen application. In both cases antigen-specific T suppressor cells have been reported to develop.

The reason for the hyporesponsiveness of UV-irradiated animals is thought to be the fact that under certain circumstances UVR can suppress what is known as antigen presentation. It had been discovered earlier (Shevach and Rosenthal, 1973) in vitro studies that certain adherent cells in the spleen and lymph nodes were required to present antigen to T cells in order to elicit

a proliferative T cell response. T cells without the aid of antigen presentation by these adherent cells respond to antigen poorly or not at all. Antigen presenting cells were found to be adherent to plastic and were found in the spleen and among peritoneal exudate cells. In addition, it was discovered that certain cells in the epidermis, the Langerhans cells could also act to present antigen to lymphocytes. To examine the function of antigen presentation, one may chemically link (derivatization) a molecule to the antigen presenting cell. Frequently, haptens are used. A hapten is a molecule that, when present by itself, does not elicit an immune response. However, when linked to another, frequently larger molecule, a hapten can induce a specific immune response.

Suppression of Systemic Hypersensitivity

When adherent cells from the spleen or peritoneal exudate cells from UV-treated donor mice were derivatized with trinitrophenyl (TNP), they could not induce hapten-specific delayed-type hypersensitivity in UV irradiated mice; whereas adherent TNP-derivatized from normal mice were able to induce delayed-type hypersensitivity in UV-treated mice (Greene et al, 1979). The hyposensitivity could be transferred into a second recipient and a T suppressor cell was shown to be present in UV-irradiated mice immunized with splenic adherent cells from UV-irradiated mice. Thus, the potential for antigen presentation in UV-irradiated mice was changed in such a way that there was apparently preferential induction of suppressor cells rather than effector cells. This was a systemic effect in that it was unnecessary immunize through the UV-irradiated skin to show the suppressive effect. Other researchers also reported similar systemic suppression of antigen presentation (Noonan et al (1981b); Fox, 1981).

Mice given UVR for an extended time period (5 wks) no longer exhibited depressed antigen presentation (Gurish, 1982). However, after 6 days of UVR, although splenic adherent cells have depressed antigen presenting cells function, lymph nodes adherent cell antigen presenting cell function was elevated. This elevation was abrogated by splenectomy prior to UVR. Therefore decrease in splenic antigen presenting cell activity caused by acute doses of UVR may be due to migration of splenic antigen presenting cells to peripheral lymphoid tissues which drain the site of epidermal inflammation.

Suppression of Local Delayed Hypersensitivity

In addition, UVR also locally suppresses the response to antigen which is applied directly to the UV-irradiated skin. Researchers have studied the kinetics of the suppression. Following a dose of UVB from FS40 lamps of 32,480 J/m2 in a single exposure, depression of the ability to be sensitized to contact hypersensitivity in mice is observed immediately in UV-irradiated skin whereas immunization in nonexposed skin elicits a normal response (Lynch et al, 1983). However, 3 days later, mice are not able to be sensitized through non-UV-exposed skin (Noonan et al 1981a). At that time point, normal densities of ATPase-positive Langerhans cells with normal histology were found in the irradiated skin; yet normal contact hypersensitivity responses were not found when dinitrofluorobenzene (DNFB) was applied to the UV-irradiated skin.

In addition, epidermal cells harvested from skin which was UV irradiated immediately prior to the experiment were unable to function in inducing T cell proliferation in vitro. Epidermal cells from mice which had been exposed to UVR three days previously had an enhanced ability to induce T-cell proliferation whereas epidermal cells from non-irradiated skin were always able to so function. This was confirmed by the finding (Cooper et al 1985) that the initial depression of antigen presentation in UV-irradiated skin is quickly followed by the appearance of T6- Dr^{+} cells that enhance epidermal alloantigen presentation. The local suppressive effect of UVB radiation was also found to occur in guinea pigs (Haniszko and Suskind, 1963) (Morison and Kripke (1984) (Morison et al. 1981).

Application of DNCB to UVB-irradiated body wall skin of mice produces a state of unresponsiveness that is associated with a Lyt-1+ T suppressor cell that acts on the induction phase of immunity (Elmets 1983). It was suggested that UV impairs the antigen-presenting potential of Langerhans cells, in whose absence other hapten-derivatized cells are able to deliver a tolerogenic signal.

Effect of UVR on Skin Graft Rejection

The ability of UVR to depress antigen presentation in vivo was tested to determine if UV irradiation of skin grafts would be less immunogenic and thus if UV treatment could increase skin graft rejection time. In the first report, full thickness skin grafts were UV-irradiated and transplanted onto normal mice. However, there was no effect on first set or second set graft rejection times (Streilein et al 1980). However, when isolated mouse tail skin was UV-irradiated in vitro on both sides, transplantation across I/region differences was significantly prolonged and 50 percent of the grafts were not rejected at all (Claas et al 1985). The prolongation of the skin graft rejection time occurred over I-region differences but across H-2 differences, usually considered a stronger difference, there was no prolongation of the skin graft rejection time.

Effect of UVR on Lymphocyte Traffic

Milton (1981) also showed that UVR of lymphocytes in vitro had no effect on localization of lymphoid cells when those cells were injected into naive recipients 1 hr after radiation, but significantly depressed localization in lymph nodes of syngeneic and allogeneic recipients when lymphoid cells were cultured 24 hr after UVR and then injected. UVR of lymphocytes in vitro followed by a 4 hr incubation led to significant alterations in the localization of the lymphocytes to lymph nodes, gut, liver, and kidneys (Spangrude et al, 1983). UVC, but not UVB, depressed the localization in the spleen. UVR of mice resulted in a dramatic and long-lasting increase in the tropism of peripheral lymph nodes for circulating lymphoid cells. Termination of UVR did not reverse the tropism. The homing patterns of lymphocytes from long-term UV-irradiated mice did not vary from normal lymphocytes in UV irradiated mice. The alterations in tropism of lymphocytes from UV-irradiated mice may play a role in the induction of a long lasting suppression. In addition, dermal vasculature of the skin has been found to expand in terms of size and number of vessels (Spangrude et al 1983). However, very few inflammatory cells were detected in irradiated skin.

Effects of UVR on Lymphocyte Function In Vitro

In another measure of the effect UVR on immune functions, lymphocytes irradiated with UVC in vitro had nearly the same alloimmunogenicity in vivo when measured by primary antibody cytotoxic response (Milton, 1981). However, low doses of UVR in vitro from an FS40 sunlamp abrogated the differentiation events which led to the generation of functional cytotoxic T cells (Lynch et al 1981). T cells were found to be more sensitive to the effects than accessory cells. The effect seemed to be due to wavelengths in the UVB range since much higher amounts of UVA were not inhibitory.

Photobiology and Wavelength Dependence of Immune Suppression

The great majority of experimentation has been done with lamps which do not accurately simulate sunlight. (Details of the lamps used in the reserach presented in thie chapter are present in Appendix 9-A.) However, it was also shown that sunlight could suppress contact hypersensitivity in both mice and guinea pigs. Exposures occurred in an unshaded area on sunny days in September and early October at latitude 30, Frederick, MD. Exposure of mice on 3 consecutive days for 5 hours each resulted in a total dose of UVB of +37,000-48,000 J/m2 and a 77% suppression of contact hypersensitivity. Suppression was dose-dependent and could be transferred into naive recipients with lymphocytes. Mylar-filtered sunlight suppressed contact hypersensitivity, but less than that produced by unfiltered sunlight (Morison et al 1985).

In a study of the photobiology of the suppression of contact hypersensitivity by UVR, BALB/c mice were irradiated on the shaved dorsal skin with FS40 sunlamps at a total dose of about 32,000 J/m2 and sensitized 5 days later on the abdomen with dinitrofluorobenzene (DNFB) in acetone (Noonan et al, 1981a). Changes in dose fractionation over 5 days and changes in dose-rate over 10-fold range did not change the total dose required to induce systemic suppression. Since a Mylar filter, which removes wavelengths less than 315 nm, abrogated the effect, it was concluded that depression of contact hypersensitivity was due to UVB. However, the UVC emitted by the FS40 lamps may also play a role in depression of contact hypersensitivity. It has been shown to induce susceptibility to challenge with UV-induced tumors (Lill, 1984). Passive transfer of lymphocytes from mice which had been UV-irradiated and then treated with DNFB as above showed that such treatment resulted in the formation of antigen specific T suppressor cells.

Elmets et al (1983) studied the wavelength dependence of the contact hypersensitivity/suppressive effect of UVR and determined that the greatest efficiency of suppression was seen at 297 nm. Exposure to 400 J/m2 daily for 4 days resulted in 60 percent suppression whereas at wavelengths of 290 nm 700 J/m2 were required for equivalent suppression. Above 297 nm, a dose of 1000 J/m2 did not induce unresponsiveness. De Fabo and Noonan (1983) also constructed an action spectrum but examined 10 different narrow band wavelengths from 250 nm to 320 nm and found peak suppression of contact hypersensitivity at 270 nm. However, they also report that UVR at 295 nm is slightly more effective at inducing hyposensitivity than UVR at 290 nm which

agrees with the data of Elmets et al (1983). These wavelengths of UVR are those which will be increased if ozone in the upper atmosphere is depleted.

Mechanism of the Induction of Suppression

Daynes et al (1985) proposed the hypothesis that recirculating Ia+ cells have antigen presenting cell function. Exogenous inflammatory stimulation of the skin by UVR may result in a time-dependent redistribution of these cells to peripheral compartments resulting in reduced antigen presenting cell activity in the spleen and the enhanced antigen presenting cell activity in the skin and draining lymph nodes. They conclude that initially there may not be a systemic defect, but a redistribution of antigen presenting cells. However, the induction of suppressor cells in UV irradiated mice indicates that the potential for antigen presentation has been altered.

De Fabo and Noonan (1983) reported that their action spectrum of contact hypersensitivity suppression is congruent with the absorption spectrum of urocanic acid, a substance found in the epidermis. The majority of urocanic acid in the epidermis is found in the stratum corneum. When the skin of mice was stripped with adhesive tape four times before UVR, the stratum corneum is removed. If the mice are then treated with UVR, contact hypersensitivity suppression does not occur. They hypothesize that urocanic acid is a photoreceptor which is located primarily in the stratum corneum and which induces hyporesponsiveness upon UV irradiation.

It has also been possible to transfer suppression of contact hypersensitivity using serum from UV-irradiated mice (Swartz 1984). Serum taken immediately after UV-irradiation with FS40 sunlamps actually slightly enhanced contact hypersensitivity but serum taken 3 hours after UV irradiation suppressed contact hypersensitivity. More interestingly, spleen cells from mice that had received serum that had depressed contact hypersensitivity were able upon passage into normal mice to suppress contact hypersensitivity in those mice, indicating the probability that the transferred serum had played a role in the induction of a suppressor cell.

It has also been reported that prostaglandins play a role in the induction of hyposensitivity. Chung et al (1986) reported that both local and systemic suppression of contact hypersensitivity by UVR could be abrogated by treatment of the mice with indomethacin. Also, effector cells for contact hypersensitivity could be recovered from draining lymph nodes of all mice sensitized through non-irradiated skin sites and the spleen of both normal and UV-irradiated mice contained suppressor cells after hapten application. Therefore, the authors concluded that the effect of UV radiation on the immune system is not only through the induction of suppressor cells, but by modification of effector cell generation or by prostaglandin-mediated depression of contact hypersensitivity effector cell activity.

ANTIGEN PRESENTATION IN VITRO

In the experiments discussed above, the effect of UVR on immune reactivity in vivo has been assessed. The effect of UVR on antigen presentation has also been studied in a slightly different fashion using in vitro measures of immune function. It has been shown that there are epidermal cells that are capable

of inducing proliferation of T cells in vitro (Stingl et al 1978). There are two experimental systems used which will be discussed. In one case, the ability to stimulate the proliferation of T cells in an allogeneic (same species, different strain or histocompatibility antigens) lymphocyte proliferation assay is studied. Epidermal cells are mixed with allogeneic lymphocytes. The non-self, alloantigens present on the epidermal cells induce the lymphocytes to proliferate, if the antigens present on the epidermal cells are presented to the lymphocytes in a fashion to which the lymphocytes can respond. Thus this is an in vitro measure of antigen presentation. Epidermal cells can also stimulate the proliferation of syngeneic T cells; although the biologic significance is not understood. In the second experimental system, researchers take advantage of the fact that epidermal cells can also present exogenous antigen to immune T lymphocytes and induce proliferation in those cells (Faure et al 1984).

Langerhans cells are the only epidermal cells which express Ia antigen under normal circumstances. Pretreatment of epidermal cells with anti-Ia antibody was reported to abolish ability of epidermal cells to function in an allogeneic stimulation assay. Therefore, it was concluded that it was the Langerhans cells that were necessary for stimulation of lymphocyte proliferation (Aberer et al 1982). A dose of UVB irradiation (50 J/m2) in vitro was found to abolish the stimulation of allogeneic lymphocyte proliferation by Langerhans cells. This same dose of UVR did not affect epidermal cell viability and only marginally decreased the protein synthesizing ability of epidermal cells as measured by uptake of radiolabeled leucine. Thus it was not necessary to kill the Langerhans cell to reduce its ability to present antigen.

When one mixes lymphocytes from two different strains of mice, the lymphocytes recognize that there are histocompatibility differences and respond by proliferating. The relative roles of the two populations can be measured by treating one of the other with a drug such as mitomycin C that prevents division of that cell without killing it. Using this system, studies somewhat similar to those performed on epidermal cells were done with lymphocytes. Spangrude et al (1983) reported that in vitro UVR of lymphocytes showed that quite low doses of UVR were capable of depressing the ability of lymphocytes to act as stimulator or responder cells in a mixed lymphocyte culture. Doses of 20 J/m2 of UVB or UVC were effective in reducing the response whereas the maximum effect was seen at a dose of at 40 to 60 J/m2. UVC was slightly more effective than UVB in depressing proliferation. Therefore the ability of UVR to depress recognition of foreign antigens by lymphocytes is not limited to antigens presented by Langerhans cells.

There is a second system in which the antigen presenting capabilities of epidermal cells has been studied (Stingl et al, 1983). Mice were immunized in vivo with dinitrophenylated ovalbumin (DNP6-OVA). An epidermal cell suspension was prepared from normal mice and pulsed with antigen. The antigen-bearing epidermal cells were then washed and exposed to T cells from the immunized mice. If the epidermal cells are able to properly present the antigen, DNP6-OVA, to the immune T cells, the T cells will proliferate in response. Exposure of epidermal cells in vitro to UVB before pulsing with

DNP6-OVA led to a radiation dose-dependent reduction in their ability to stimulate antigen-specific proliferation in syngeneic immune T-cells. However, when epidermal cells were antigen-pulsed before UVR, their stimulatory capabilities were at least partly maintained.

A potential mechanism for the lack of stimulation of lymphocyte proliferation was reported by Sauder et al (1983) who reported that epidermal T-cell activating factor (ETAF), which has been found to be virtually identical to IL-1 (Luger et al 1983), could partially restore T cell stimulation which had been suppressed by UVR. In addition the differences in T-cell proliferation induced by epidermal cells which are UV irradiated prior to antigen pulse, compared to that induced by normal cells, correlated well with the amount of ETAF generated in epidermal cell cultures consisting of keratinocytes and Langerhans cells (Stingl et al 1983). Addition of ETAF could partially restore the decreased capacity of epidermal cells which are pulsed with antigen after UVR but not before UVR to stimulate antigen-specific T-cell proliferation. However, UVR was also shown to increase the production of ETAF in keratinocyte cultures (Ansel et al 1983) and epidermal cells from both normal and UV-irradiated mice were found to produce equivalent amount of ETAF (Gahring et al 1984). Therefore, the role of ETAF is far from clear.

LANGERHANS CELLS AND ANTIGEN PRESENTATION

As discussed above, Ia+ epidermal cells can present antigen in vitro, this capability is depressed by UVR, and the epidermal cell which has been implicated in antigen presentation is a Langerhans cell. Further research has focused on the relationship of Langerhans cells to the induction of contact hypersensitivity and effects of UV on Langerhans cells.

To determine the relationship between antigen presentation and the presence of Langerhans cells, Toews et al (1980) took advantage of the fact reported earlier (Bergstresser et al, 1980) that murine tail skin has significantly fewer Langerhans cells than body wall skin. Sensitization could only be effected through the body wall skin. When a short course of UV light at a dose of 100 J/m2 day for 4 days from FS20 lamps was used to deplete Langerhans cells from body wall skin, sensitization could also not be effected through UV-irradiated skin. In addition, mice whose first exposure to DNFB was through Langerhans cell-deficient skin (tail skin or UV-treated body wall skin) could not subsequently be sensitized through body wall skin. There was no hyporesponsiveness in UV-treated mice if the first antigen contact was through unirradiated skin. This work was confirmed by Semma and Sagami (1981) who in addition showed that the hyporesponsiveness was due to active suppression of contact hypersensitivity by hapten-specific T suppressor cells. Interestingly, it was shown that when tail skin is transplanted to the body, attempts to sensitize the animal through the tail skin are unsuccessful (Streilein et al 1980). Therefore, the inability to induce contact hypersensitivity through the tail skin is due to a peculiarity of the skin and not its anatomic location.

UVR can deplete Langerhans cells from skin as measured both by ATPase staining, expression of Ia antigen, or morphologic criteria. The depletion of Langerhans cells in both murine and human skin by UVB and UVA had very similar

dose responses in both mice and humans (Aberer et al 1981) upon exposure to either single or cumulative doses of UVA, UVB, or UVA containing small amounts of UVB.. Doses of UVR which completely abrogated the Langerhans cell markers of ATPase staining and Ia antigens also caused cellular damage to some Langerhans cells, as seen by electron microscopy. However, the vast majority of the Langerhans cells seen ultrastructurally appeared normal. At lower doses of UVR, such as 150-300 J/m2 of UVB and 4000-10,000 J/m2 of UVA, Langerhans cells were the only epidermal cells to show damage at the ultrastructural level whereas higher doses affected all epidermal cells. Depression of Ia antigens and ATPase staining occurs at lower doses of UVR than cellular damage. There was a rough correlation between staining for Ia antigens and ATPase staining, but staining for ATPase persisted in intracellular areas at UVR doses which completely abolished staining for Ia antigens. Gilchrest et al (1982) reported that although young adult (22-26 year-old) humans have more Langerhans cells than old (62 to 68 year-old) humans, the Langerhans cells in young adults were much more sensitive to UVR. Obata and Tagami (1985) studied the number of Ia+ cells which remained after UVR. After a single dose of UVB radiation (2000 J/m2 at 300 nm) they determined that although some Langerhans cells simply lost Ia antigen, the majority disappeared due to cell damage. From 4 to 14 days after UVR, enlargement of the remaining Langerhans cells occurred and very large Langerhans cells were seen. The recovery rate of Langerhans cell number after UVR was slower than for keratinocytes.

Action spectra for depletion of Langerhans cells and keratinocyte damage were reported by Obata and Tagami (1985) to be similar and to also be similar for what has been reported for the induction of unscheduled DNA synthesis and UVR-induced erythema. To reduce the number of Ia+ cells to less than 20 percent of normal numbers, UVR at 260 nm was more effective than 280 nm and 300 nm, which gave similar results. UVR at 320 nm and greater was much less effective. For example at 340 nm and 360 nm, 10,000 J/m2 did not reduce the number of Ia+ cells and at 327 nm, 10,000 J/m2 only gave a 40 percent reduction whereas 2000 J/m2 at 280 nm and 300 nm reduced the number of Ia+ cells to less than 20 percent of control. This may play a role in the suppression of local contact hypersensitivity.

However, Noonan et al (1984) reported that the depletion of Langerhans cells by UVR and the suppression of systemic contact hypersensitivity by UVR are not causally related. Langerhans cells were identified by ATPase staining of EDTA-separated epidermal sheets. Electron microscopy was used to confirm the staining and it was reported that there was no evidence for Langerhans cells which did not stain for ATPase. Mice were irradiated with 270 nm, 290 nm, or 320 nm UVR produced by interference filtration of a Xenon-arc light source. UV radiation at 270 nm or 290 nm caused a loss of dendrites and a decrease in the total number of Langerhans cells in a dose dependent fashion. The dose to yield a 50 percent reduction in the number of Langerhans cells was about 250 J/m2 for 270 nm and 290 J/m2 for 290 nm. Doses of 270 nm and 290 nm UVR which decreased the number of Langerhans cells were insufficient to suppress contact hypersensitivity to TNCB applied to unexposed skin. A dose of 320 nm (8200 J/m2) which caused 50 percent suppression of contact hypersensitivity had no effect on the number or morphology of Langerhans cells. In addition, the number and morphology of Langerhans cells on the

ventral epidermis was not affected following a dose of UV to the dorsal skin even though that UVR resulted in systemic suppression of contact hypersensitivity when there was an attempt to sensitize mice though the ventral epidermis.

The same conclusion was reached by Morison et al (1984) using a different approach. Mice were exposed to UVB on the dorsum with a single 3 hour exposure. Mice were also subjected to the trauma of photosensitization with rose bengal or eosin and visible light filtered to cut off below 390 nm. Finally, mice were also treated with UVA continuously for 72 hr. The Langerhans cells in the skin were examined 3 days after UVR by staining for ATPase and by electron microscopy. The number of normal-appearing Langerhans cells 3 days after UVB was only 28 percent whereas the number and morphology in unexposed skin in the same mice were normal. UVA radiation resulted in the complete destruction of normal Langerhans cells. Photosensitization with rose bengal or eosin and visible radiation did not reduce Langerhans cells. However, when contact hypersensitivity was tested, photosensitization with rose bengal and visible light did suppress TNCB reactivity whereas photosensitization with eosin and visible light or treatment with UVA did not. Thus the absence of Langerhans cells in the epidermis did not correlate with suppression of contact sensitivity. Also, the presence of the UVB damaged Langerhans cells was not the cause of hyporesponsiveness since depletion of Langerhans cells by UVA (which did not decrease contact hypersensitivity) followed by UVB radiation also depressed contact hypersensitivity. This is important because it implies that the mechanism of the depression of immune reactivity by UVB is more specific than simple tissue damage.

A possible answer to the conflicting reports on the role of epidermal cells in UV-induced suppression of contact hypersensitivity has been proposed. In agreement with a larger number of previous studies, Granstein (1985a, 1985b) has reported that when mice are immunized subcutaneously with hapten-coupled UV-irradiated epidermal cells, the mice are hyporesponsive when delayed-type hypersensitivity is measured with a footpad assay and in addition hapten-specific T suppressor cells are present. However, depletion of I-J bearing cells with anti-I-J antibody and complement from the UV irradiated epidermal cells (prior to UVR and haptenation) prevents the appearance of these suppressor cells. Non-UVR treated epidermal cells which are I-J depleted and haptenated can induce delayed-type hypersensitivity. Therefore, there seems to be an additional cell in the epidermis, other than the Langerhans cell and the keratinocyte, which is UVR resistant and which apparently presents antigen in a tolerogenic fashion and thus preferentially induces suppressor cells. Thus hypothetically, when antigen is presented in the skin in the usual fashion, Langerhans cells present the antigen and effector cells are generated. However, when skin is UV-irradiated, the function of the Langerhans cell is depressed and antigen can then be presented by the I-J cell, resulting in the generation of suppressor cells.

However, one needs to explain the relationship between induction of tolerogenic presentation of antigen by UVR and the induction of suppressor cells which are specific for antigens present on UV-induced tumors. Spellman

and Daynes (1984) have reported that there are antigens present on UV-irradiated skin that are cross-reactive with UV-induced tumors. Mice were irradiated 30 minutes per day for 6 weeks, which is well under the time of radiation required to produce tumors. Skin from these animals was then transplanted as 1 cm grafts to normal syngeneic mice. Twenty days later a second graft of UV-irradiated skin was made. When mice were challenged with a UV-induced progressor tumor 15 days later, animals were protected from the challenge. Since grafting of UV-irradiated skin protected a mouse from challenge with a UV-induced tumor, the conclusion was drawn that similar antigens were present on both the UV-irradiated skin and the UV-induced tumors. Therefore, if UVR causes the expression of neoantigens on skin and at the same time facilitates the induction of suppressor cells to antigens, then the immune response against those tumors might take the form of induction of suppression rather than induction of cytotoxic cells.

HUMAN STUDIES

There have been fewer studies of the effects of UVR on the immune response in humans. Morison et al (1979) irradiated human volunteers with 1.5 minimal erythemal doses (MED) to produce what they termed asymptomatic erythema or 3 MED to produce what they termed symptomatic erythema. Post irradiation, all subjects showed a significant increase in the proportion of circulating polymorphonuclear leukocytes and a corresponding increase in the proportion of circulating lymphocytes. Subjects who received 3 MED also had a significant decrease in the proportion of circulating E-rosette forming cells, i.e. T cells, and an increase in the proportion of null cells. The changes in the absolute numbers of these cells were not significant. Thirty minutes and 4 hours after UV-irradiation, the response of lymphocytes to PHA was increased, then decreased, reaching a minimum 12 hours after UVR, and returning to normal 72 hours post exposure.

Hersey et al (1983a) reported on the effects of solarium exposure on normal human subjects. Normal volunteers were given a standard course of treatment in a solarium to acquire a suntan; twelve 1/2 hr exposures. The energy delivered was 10 J/m2 for UVA and about 1% of total irradiation was UVB (which was not quantitated). Immune functions were carried out before, on completion, and 2 weeks after the end of irradiation. The test subjects had reduced skin test responses to DNCB and slightly reduced numbers of blood lymphocytes. There was a relative increase in T (OKT3+) cells which was attributable to an increase of OKT8+ (suppressor) cells. There was a significant increase in suppressor T-cell activity against pokeweed mitogen induced IgG production in vitro and a depression of NK activity. The changes were still present in some subjects 2 weeks after solarium exposure. The NK data is a little uncertain since the control subjects had lower levels of NK activity throughout the studies than the UV-irradiated subjects. Some patients had increased NK activity after treatment and some did not change. In a second study (Hersey, 1983b), normal human volunteers were exposed to sunlight for 12 days in a 2 week period. Tests of immune function were made before, on completion and 2 weeks after the end of the UVR. There were 15 UV-irradiated subjects and 13 age- and sex-matched controls. There were no significant changes in hemoglobin levels, total leukocyte count, and lymphocyte, or neutrophil counts. In test subjects there was a small but

significant drop in T cell numbers which returned to normal by 2 weeks. There was, however, a marked increase in the ratio of suppressor cells to cytotoxic helper cells which had not returned to normal at 2 weeks after UV-irradiation. After UVR there was an increase in the activity of pokeweed mitogen-induced gamma radiation-sensitive suppressor T cell activity against IgG and IgM production. Although immunoglobulin production in vitro has too wide a range for statistical analysis of the small numbers of patients to be of value, gamma irradiation of T cells prior to culture increased the amount of immunoglobulin produced in patients exposed to sunlight but not in controls. There were 5 persons who did not show these changes, but they were largely accounted for by persons who did not receive the full dose of UVR. Immunoglobulin levels in vivo did not change nor did NK activity change significantly in this study. O'Dell et al (1980) reported that there was a diminished immune response in sun-damaged skin. The concentration of DNCB required to elicit a positive patch test was greater in sun-damaged skin than in skin which was normal. In addition, the delayed-type hypersensitivity to intradermal injection of Candida, mumps, and PPD antigens was decreased in sun-damaged skin so that the differences were not due to a difference in percutaneous absorption of antigen through sun-damaged skin. The response to a primary irritant was the same in both sun-damaged skin and normal skin and there was no difference in the two tested sites (back of the neck and the back) in volunteers without sun-damaged skin at the back of the neck. Therefore, there is apparently a local suppression of contact hypersensitivity in sun damaged skin.

There is also evidence that the inability to repair UV-induced damage may also play a role in impairment of immune function. A syndrome xeroderma pigmentosum has been defined in which the patients, who suffer from sun sensitivity and increased numbers of sunlight-induced cancers, also have been shown to have impaired ability to remove and repair lesions in their DNA caused by UVR. Morison et al (1985) reported that xeroderma pigmentosum patients had impaired ability to develop contact hypersensitivity to DNCB in sun-exposed skin when compared to normal control subjects. The numbers and morphology of Langerhans cells in the skin were the same in both groups. Unfortunately, the study did not include an assessment of ability to develop contact hypersensitivity in skin not usually exposed to sunlight.

EFFECTS OF ULTRAVIOLET RADIATION ON INFECTIOUS DISEASES

Upon the realization that UVB can suppress antigen presentation of antigens applied through both irradiated and unirradiated skin, concern developed that the immune response to diseases in which the first contact of the immune system with the invading organism was though the skin might be adversely affected by UV irradiation. This was especially true since only low doses of UV irradiation had been found to be necessary to suppress an immune response the allergen was applied through the UV-irradiated skin. As yet, not a great deal of research has been published on this problem, although there are at least two infectious diseases, Herpes virus infections and cutaneous leishmaniasis in which research has begun to indicate that the impact of UVR-induced immunosupression may play a role..

It has been reported for some time that exacerbation of Herpes simplex virus infections can be caused by sun exposure. The following generalizations have been suggested (Harber and Bickers, 1981). Each patient's photosensitivity is site-specific and the action spectrum may be unique to a patient. Some have been reported to react to UVC and some to a source emitting UVC and UVB. Other patients may exacerbate with heat exposure. An exposure of 5 to 6 MED has been reported to cause exacerbation of oral herpes (Spruance 1985). It is common to report exacerbation following sun exposure or UVB phototherapy although the mechanism is unknown (Morison 1984). In fact, exacerbation of genital herpes has also been reported to occur following exposure to the penis to experimental UVR (Klein and Linnemann, Jr. 1986) and there are anecdotal reports of recurrence following sun exposure which did not include the genitalia (Oates 1986).

Although, the mechanism of the exacerbation is unknown, recent investigations on the effects of UV radiation on primary Herpes infection in mice are yielding very interesting information. Low doses of UVR from sunlamps given 7 days prior to infection of mice with Herpes simplex virus suppressed the delayed-type hypersensitivity to the virus (Howie et al 1986a). Once it was induced, immune suppression to the virus persisted for at least 3 months after the UVR. The suppression has been shown to be due to two different subsets of T lymphocytes (Howie et al 1986b). Thus in this animal model, exposure to UV radiation at an appropriate time prior to infection, suppressed the immune response made to that organism for some time and may have profound effects on the outcome of the infection.

There have been a few results with regard to the potential role of UVR-induced immunosuppression in infectious disease progression from studies using an animal model of an important tropical and subtropical cutaneous disease, leishmaniasis, . Giannini (1986) has reported that suberythematous doses of UVB irradiation to mice blocked the development of the initial skin lesions when Leishmania major promastigotes were injected. However, the UVB also abrogated the development of delayed type hypersensitivity to the leishmanial antigens. Protective immunity in leishmaniasis is known to be cell mediated and delayed type hypersensitivity to leishmanial antigens is considered indicative of healing. Therefore, UV irradiation of mice infected with leishmanial parasites profoundly interferes with immunity. The possibility exists that in human leishmaniasis UV irradiation might also play a role in the pathogenesis of the disease. Infection of humans through recently UV-irradiated skin might suppress the immune response to leishmanial antigens and thus predispose to a more chronic, more severe disease.

UVR and Cutaneous Conditions Other Than Cancer

There are a number of cutaneous conditions in which UVB is thought to play a crucial role. Although the incidences of these diseases seem small, their actual impact on human health is magnified because almost all of them are chronic diseases for which there is no known cure. Some of them, such as lupus erythematosus and solar urticaria, encompass a spectrum of severity and may indeed be several diseases of different etiology but labeled with the same name, thus studies indicating a broad action spectrum may be misleading.

Because variations in thickness of the ozone layer will have the largest effects on UVB fluxes, we address primarily the role of these wavelengths in diseases affect by UVR, even though the greater magnitude of responses may be elicited by the UVA wavelengths for some of these diseases.

It is not the intent of this section to be a complete discussion of those diseases in which UVR may play a role, but to only briefly discuss those diseases in which there is evidence that UVB plays a role.

Lupus erythematosus

Lupus erythematosus (LE) occurs in two main forms, discoid lupus erythematosus (DLE) which is a limited disease with lesions primarily limited to the skin, and systemic lupus erythematosus (SLE). SLE is a very heterogeneous disease with an autoimmune etiology. Included in the disease symptomatology is an abnormal reactivity to sunlight, which has been reported in as many as 58 per cent of patients. Exposure to sunlight can cause new skin lesion, exacerbate old skin lesions, and can also induce systemic exacerbation of the disease (Zamansky 1985). The patients show a variety of serum antibodies reactive to many self antigens. However, the characteristic antibodies are those to DNA. Patients with SLE have been reported to have antibodies in their serum that react with UV/irradiated DNA. Some of the antibodies will react quite specifically only with UV-irradiated DNA (Davis et al 1976). Patients with LE possibly have increased sensitivity to UVB. There have been suggestions, although unconfirmed, that the minimal erythemal dose is at the lower limit of normal (reviewed in Morison, 1983). It appears that the skin lesions of lupus were induced more efficiently by UVB than by UVA but the studies are not definitive. The fact that many patients are on immunosuppressive therapy probably complicates results and may be the reason that not all patients develop skin lesions following ultraviolet radiation. Eight patients with LE were phototested by irradiation of involved and uninvolved skin sites with a high pressure mercury arc source. In 3 or 8 patients the involved skin showed extension of the lesions whereas the uninvolved skin did not react (Everett and Olson, 1965). Freeman et al (1969) studied 15 patients testing the action spectrum of photosensitivity. Although not all patients reacted positively, the peak sensitivity for the for the abnormal photosensitivity was 300 nm, in the UVB range. Skin lesions were produced in patients with DLE by UVB radiation. In disseminated DLE, the sensitivity to ultraviolet radiation extended to 330 nm. Cripps and Rankin (1973) showed that UVR at 300 nm caused a long/lasting erythema in the six patients tested. Four of those patients had histologic evidence of lupus erythematosus in those sites from 7 weeks to 6 months after testing. It was nessary to administer 8 MEDs to achieve this result; lesser doses had no effect on the skin. Sunlight is considered to be a factor in exacerbation of both DLE and SLE, although all skin lesions may not be on exposed skin (reviewed in Cripps, 1983). Cripps (1983) stressed that although photosensitivity occurs in SLE, under routine clinical conditions, most patients with DLE do not exhibit photosensitivity. Lymphocytes from patients with SLE were found to be more sensitive to the toxic effects of UVC that lymphocytes from normal patients (Beiglie and Teplitz 1975)

Also apparent is that exposure to sunlight may on occasion exacerbate not only the skin lesions of SLE but the systemic disease. In some patients symptoms usually flare up following sun exposure. Other patients may show sensitivity at one time and not another. Exacerbation of SLE for any reason requires additional immunosuppression for treatment and may be life threatening (Steinberg, 1985). However, because of the normal cycles of exacerbation and remission of SLE, it is extremely difficult to study the effects of UVR on systemic lesions of SLE. Murine models of SLE have been used to study the exacerbation of the human disease after exposure to sunlight. One strain, male BxSB mice died prematurely and had acceleration of their disease after both acute and chronic treatment with FS40/sunlamps. However, filtering the radiation through Mylar filters, which removes UVB wavelengths, abrogated the effect. Therefore, the accelerated autoimmunity reported in these mice was due to a sensitivity to UVB (Ansel et al 1984, 1985). Not all strains of mice which are used as models for autoimmune disease are sensitive to sunlamps (Ansel et al 1985, Strickland 1984) but the male BxSB mouse should prove to be a valuable model to study the problem. Therefore, although the exact interaction between UVB and systemic and skin lesions of SLE is not clear, there is ample evidence that UVB can play a role in the disease.

Phototoxicity

Phototoxic reactions occur when drugs or natural substances interact with UV radiation or light and result in toxicity. Action spectra of phototoxic reactions in humans show that the greatest magnitude of responses are found in the UVA wavelengths. However, the data also suggest that wavelengths below UVA may also contribute to the reaction (Harber and Baer, 1972).

Phototoxicity associated with chlorpromazine has been reported to occur primarily with UVA (Epstein S, 1978, Hunter et al 1970), but UVB has also been implicated (Hunter et al 1970). When chlorpromazine is UVB-irradiated and then injected subcutaneously in experimental animals, it produced toxic responses. Therefore, it is possible that at least some part of the phototoxic responses to chlorpromazine are due to stable photoproducts which are toxic (Reviewed in Harber and Bickers, 1981, pg. 131).

Sulfanilamides were the first effective antibacterial drugs to be/developed. Sulfanilamides were also the first drugs recognized as being capable of inducing both phototoxic and photoallergic reactions in humans (Epstein, S 1939). The action spectrum has been measured following irradiation of intradermally injected sites in humans and has been found to be in the UVB range (reviewed in Harber and Bickers, 1981, pg. 137). Blum (1941) reported that the skin reaction of a patient to irradiation from a mercury arc source could be prevented by filtering the radiation though window glass, which removes UVB. In addition, testing of sulfanilamide for phototoxicity in vitro using toxicity to human lymphocytes showed that the phototoxocity was induced by UVB but not UVA (Morison et al 1982). The same in vitro testing demonstrated that phototoxocity of hexachlorophene is also due to UVB and not UVA.

Photoallergy

Photoallergy differs from phototoxicity in that an immune response in the cause of the lesions induced by UV radiation or light. Induction of photoallergy is generally considered to be due to UVA rather than UVB. In fact, low doses of UVB to the site of induction of photoallergy have been shown to suppress the photoallergic response in a manner analogous to suppression of contact hypersensitivity in that antigen-specific suppressor T cells were generated (Morison, 1984). Also systemic suppression of photoallergy, not associated with suppressor cells can also be caused by UVB radiation of cyclophosphamide-treated mice.

Solar Urticaria

Solar urticaria can be induced by UVB, UVA, and visible light. It can be passively transferred by an intradermal injection of serum from a patient into previously irradiated skin of a/normal volunteer (Epstein, S 1949) or can be transferred when serum from a patient is injected into the skin of a normal volunteer and then UV-irradiated (Sulzberg and Baer 1945). Patients can be classified into 7 groups on the basis of action spectra and immunologic parameters. In Type I patients the active wavelengths are found in the UVB range and it is apparently mediated by IgE whereas in Type V patients the action spectrum crosses UVB, UVA, and visible light and it is not transferable by the patients serum (Harber et al 1963, Ive et al, 1965). It is a rare disease with somewhat more than 100 patients having been reported in the literature (Harber and Bickers, 1981).

Polymorphous Light Eruption

Polymorphous light eruption is a disease of unknown etiology. Phototesting has led to the realization that it can be induced by UVA, UVB, or visible light. However, the papular and plaque-like varieties have an action spectrum primarily in the UVB range (Harber and Bickers 1981). Some patients seem to respond abnormally to UVA (Epstein J 1983). There is disagreement on the action spectrum which probably arises at least in part from tests on a heterogeneous group of patients with a heterogenous group of disorders. However, most investigators believe that the primary action spectrum which induces papules is in the UVB range (reviewed in Harber and Bickers, Ch. 14, 1983). It has also been reported (reviewed in Cripps, 1983) that patients have a normal minimal erythemal dose in the UVB range with prolonged erythema. However, UVA was reported to be more effective than UVB in induction of delayed erythema (reported in Cripps, 1983).

Actinic Reticuloid and Photosensitive Eczema

These two disorders are similar in that they occur in elderly males. Photopatch testing with known photosensitizers has yielded negative results. In patients with photosensitive eczema, the action spectrum is limited to UVB. In patients with actinic reticuloid, a chronic dermatosis, the

sensitivity is first seen in the UVB range but later extends into the UVA range as the disease becomes chronic. Patients show a decreased minimal erythemal dose, sometimes as low as 30 times less than normal (reviewed in Harber and Bickers, 1981, Cripps, 1983). It has been suggested that the two diseases may actually be two phases of the same disease (Morison, 1984).

Bloom's Disease

The skin lesions of Bloom's disease may exacerbate with sun exposure and is thought to be due to UVB (Harber and Bickers, 1983).

Pellagra

The cutaneous lesions of pellagra, erythema followed by desquamation and hyperpigmentation, may be precipitated by sunlight. Harber and Bickers (1983) speculate that lack of NADP and NADP-H may render the patients unable to repair damage caused by the skin by UVB.

CONCLUSIONS

Ultraviolet radiation has been shown to have a variety of effects on the functioning of the immune system. Acute doses of UVR suppress some immune parameters in a nonspecific fashion. Thus this may be similar to the immune suppression caused by thermal burns. In addition, UVR can suppress the induction of contact hypersensitivity and affect the immune response in such a way that suppressor cells are generated to an antigen to which the animal is exposed during that time. One of the more interesting effects of UVR is the generation of T suppressor cells which specifically suppress the immune response to UV-induced tumors. The suppressor cells have been shown to be capable of shortening the latent period of tumors induced by UVR. This is a unique tumor system in which the carcinogen also affects the immune system such that the growth of the tumors is facilitated in a specific fashion. Two recent pieces of information offer a possible explanation as to the mechanism by which UVR induces such a facilitation. UVR of mouse skin causes the appearance of antigens which cross react with those of UV-induced tumors. This occurs at a time when the mice are susceptible to suppression of contact hypersensitivity. Therefore, it is possible that mice which are UV irradiated and whose ability to mount a contact hypersensitivity reaction is suppressed in a way which facilitates the induction of tolerance at the same time during which neoantigens are expressed on irradiated skin. Therefore the mice develop tolerance to those neoantigens which are expressed on UV-irradiated skin as well as UV-induced tumors and are unable to mount an immune response to the incipient tumors. This interferes with immune surveillance and the growth of the tumors occurs unchecked.

We have no direct evidence that UVR of man also affects the immune response in such a way that the growth of tumors is facilitated by a suppressor cell. However, we do know that UVR of humans does cause suppression of the immune response and, more important, that induction of contact hypersensitivity is depressed in human skin which has been

sun/damaged. Thus increasing dose of UVR may act to increase the incidence of skin tumors in man both by acting as a carcinogen and also, in an indirect manner, facilitating the growth of those tumors by interfering with immune surveillance.

There is at present only preliminary experimental evidence from animal models that UVR can detrimentally affect the immune response to infectious diseases. However, it is well to note that these effects were successfully predicted from the earlier studies on the effects of UVR on the immune response and the probability is great that this is only the tip of the iceberg. There are a wide variety of diseases in which the primary immune response to the offending organism occurs in the skin. In some diseases, the infection remains confined to the skin whereas in others the infection may become widespread throughout the body. In evaluating the possible effects of increased UVR on the populations of the world that we recognize the potential for increased morbidity that can accrue on a world wide scale.

The spread and cure of many cutaneous diseases are controlled at least partially by the immune response. Some of those diseases, such as leishmaniasis, may become systemic if they are uncontrolled. The majority may cause significant morbidity and loss of function if allowed to progress without intervention from the immune system or medication. If UVR of skin depresses the immune reactivity in the skin/by facilitating induction of suppressor cells, this could have major impact on the health and welfare of a great number of people in the world, in particular the citizens of the third world countries. If such mechanisms do occur, then increasing doses of UVR could cause suffering, losses of productivity, and increased health care costs in the ensuing years. At present, there is little research in this area.

APPENDIX 9A

RADIATION SOURCES

It is appropriate to discuss briefly the sources of UVR used in experimentation since the characteristics of these lamps play a great role in the interpretation of results of experiments conducted with them. A more complete discussion is made by Spikes (1983). Solar radiation consists of broad spectrum irradiation with the lowest wavelengths reaching the earth being approximately 290 nm. There is proportionally greater energy in the UVA range than UVB. However, it is impractical to use sunlight for experimentation due to both short and long term variability in intensity and wavelength. Therefore, in order to estimate the effects of solar radiation investigators have used a variety of UV-emitting lamps. These lamps are good and usually inexpensive sources of UV radiation; but it is important to realize the limitations in the precision and comparability of data generated by use of each light source. The two major types of light sources are germicidal lamps and sunlamps. Germicidal lamps emit about 90 percent of their radiation at 254 nm with approximately 1 percent at 313 nm, 1 percent at 365 nm, 2 percent at 405 nm, 3 percent at 436 nm, and 0.4 percent at 577 nm. There are major limitations in the use of such lamps in studies to evaluate the impact that solar radiation may have on various systems as the UV radiation produced by these lamps is not principally at 254 nm a wavelength which does not penetrate the earth's atmosphere. In addition, these lamps are not a monochromatic sources since small but detectable amounts of radiation are emitted in the UVB, the UVA, and the visible range.

Fluorescent sunlamps emit most of their radiation in the UVB range and so are better, yet still not perfect surrogates for solar radiation. A small amount of the radiation is in the UVC range and there is radiation in the UVA and visible range also. The proportion of radiation in the various wavelengths is not the same as in sunlight because of the relationship of UVB to UVA and visible light. Therefore, although the bulbs are called sunlamps, they are not actually a solar simulator and there is significant irradiation at wavelengths other than UVB so that when experiments are performed using sunlamps, such as the Westinghouse FS40 or FS20, one must be careful in interpreting the data. These are not pure UVB sources. Solar simulators and sources of monochromatic light are available but are quite expensive. It is difficult to perform experiments requiring monochromatic light since high intensity monochromaters usually use a narrow band of light and diffraction grating to select the desired wavelengths so that only small areas can be irradiated. Filters can also be used to control wavelengths; however filters which allow only narrow wavelengths to pass are expensive, usually small, and decrease the intensity of the emission significantly. Plastics can also be used to remove shorter wavelengths of light; however the transmission characteristics of the plastic must be periodically monitored since UV irradiation will change the absorbency of the plastic. Therefore, in interpreting the results of experimentation done with UV radiation, it is always necessary to be aware of the characteristics of the lamp and the filters used.

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Chapter 10

CHAPTER 10

CATARACTS AND OTHER EYE DISORDERS

SUMMARY

Cataracts are opacities that develop in the lens of the eye and impair vision. In the United States and other developed countries, cataract operations prevent most cataracts from causing blindness. However, in the U.S. cataract remains the third leading cause of legal blindness. In developing countries where such operations are not always available, cataracts often result in blindness.

Scientific understanding of the physical mechanisms which cause cataracts is incomplete; it is likely that more than one mechanism operates. Epidemiological studies, laboratory animal studies, and biochemical analysis support the belief that some cataracts are caused by ultraviolet radiation B (UV-B). Ultraviolet radiation A and other causes are also likely. A change in the amount of UV-B radiation is reasonably likely to alter the incidence of cataracts. UV-B may also play a role in causing or exacerbating other eye disorders. Ozone modification that alters the amount of UV-B reaching the earth's surface is likely to change the prevalence (and incidence) of cataracts.

FINDINGS

1. THERE APPEARS TO BE A REASONABLE PROBABILITY THAT CATARACT INCIDENCE WILL CHANGE WITH ALTERATIONS IN THE FLUX OF UV-B CAUSED BY OZONE MODIFICATION.

   1a) Many possible mechanisms exist for formation of cataracts. UV-B may play an important role in some mechanisms.

   1b) Although the cornea and aqueous of the human eye screen out significant amounts of UV-A and UV-B radiation, nearly 50 percent of radiation at 320 nm is transmitted through to the lens. Transmittance declines substantially below 320 nm, so that less than one percent is transmitted below approximately 290 to 300 nm. However, the results of laboratory experiments on animals indicate that short wavelength UV-B (i.e., below 290 nm) is perhaps 250 times more effective than long wavelength UV-B (i.e., 320 nm) in inducing cataract.

   1c) In laboratory animal experiments, the action spectrum for cataracts is weighted heavily in the UV-B range.

   1d) Human cataract prevalence appears to vary with latitude and UV radiation; brunescent nuclear cataracts show the strongest relationship.

2. INCREASES IN THE AMOUNT OF UV-B THAT CAN REACH THE RETINA APPEAR CAPABLE OF CAUSING STABLE RETINAL DISORDERS AND RETINAL DEGENERATION, TWO CAUSES OF BLINDNESS.

3. UV-B MAY PLAY A ROLE IN DISORDERS OF THE EYES AS WELL AS IN DEVELOPMENTAL DISORDERS.

CATARACTS[1]

The longest standing hypothesis which may account for the development of senile cataracts is that radiant energy, particularly sunlight, is a major factor in the etiology of the disease. This concept apparently originated from observations reported by a number of individuals indicating that cataracts occurred more frequently or earlier in persons whose occupations kept them outdoors or that populations living in areas with longer hours of sunshine have a higher frequency of cataract than populations from areas where there is less sunshine. While the early studies were severely flawed by their failure to consider adequately the possible effects of a variety of socioeconomic and other variables, Duke-Elder (1926, 1972) proposed that "the fundamental cause of cataract in all its forms may be traced to the incidence of radiant energy directly on the lens itself."

Definition of Cataract

Cataract is defined as an opacity in the normally transparent lens of the eye which produces an impairment of vision. Cataracts may occur as a result of a wide variety of factors including metabolic disorders, exposure to toxic agents, trauma, exposure to radiation, and hereditary factors. The great majority of cataracts, however, are the so-called senile cataracts which occur in older individuals and for which no specific causative factor can be identified. Cataract is a major cause of visual impairment and blindness particularly in developing countries where access to modern surgical facilities is limited. Even in the United States, cataract is the third leading cause of legal blindness. Some 60 percent of people aged 60 to 74 have at least some cataractous changes in their lenses. The only efficacious treatment for cataract at present is the surgical removal of the opaque lens and nearly 660,000 such operations were performed in 1982 in the U.S.

It is clear therefore that senile cataract is a very significant health problem, both in terms of its impact on the affected individuals and on society at large. While considerable progress has been made in elucidating the biochemical etiology of certain specific cataract types, such as sugar cataracts, there is little conclusive data on the causes of senile cataracts. It is likely that there are a variety of potential risk factors and that, in general, senile cataracts have a multifactorial etiology. This conclusion is supported by the great variability observed clinically in the time of onset, the rate of maturation, and the morphological appearance and location within the lens of these opacities. It appears that many, if not all, of the processes contributing to senile cataractogenesis are normal aging processes which for whatever reason are accelerated in certain individuals.

Cataract Classification

There are now rather sophisticated systems for the classification of cataracts. Chylack et al. (1978, 1983) have devised an in vitro system which

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[1]This section in boldface type is taken from Pitts, D.G. et al. (in press).

is based on photographic documentation of opacification and nuclear color. Almost 2500 cataracts have been studied and classified since the original methods were adopted (Chylack et al., 1983). In addition, Marcantonio et al. (1980) have suggested a system of classification of human senile cataracts by using photography for in vivo and in vitro analysis and determining the sodium and protein content of the extracted lens. The photographic method gave consistent involvement of the lens nucleus but it was not always possible to relate sodium changes to light scattering. Marcantonio et al. (1980) argue that a dual classification system is necessary because most cataracts are mixed in nature and the osmotic and nuclear mechanisms provide quite different changes in protein distribution.

The present state of knowledge concerning the human senile cataract probably justifies a classification into only two major types in spite of the elegant classification systems mentioned above. The first and most common type of human senile cataract is the cortical cataract. Cortical cataracts are characterized by imbalances in cation levels within the lens cells which produce osmotic swelling and ultimate opacity in the lens cortex. Altered cation balance may result from damage to the Na^+, K^+-ATPase or from compromise of the normal permeability characteristics of the lens membranes. Any of a great variety of potential insults could be the ultimate cause of such cataracts. The second major type of senile cataract is the nuclear cataract which is characterized primarily by very marked modifications to the structural proteins of the lens, the crystalline, in the central region of the lens. These two types of cataracts are not at all mutually exclusive; in many instances, senile cataracts contain both cortical and nuclear opacities. It is advisable for those who are involved in cataract research to become knowledgeable and use a recommended classification system.

Epidemiological Studies

A few general statements should be made relative to the cataract epidemiological studies. Most epidemiological studies have reported an increase in the prevalence of cataracts with an increase in age and at 65 years of age and above there is an acceleration in the prevalence of cataracts. Women have shown a larger prevalence of cataracts than men; however, this difference has not been shown to be statistically significant.

In recent years there have been a number of epidemiological studies reported which have attempted to establish that there is an association between cataract prevalence and exposure to sunlight or the ultraviolet component of sunlight. Hiller, Giacometti and Yuen (1977) utilized data on the populations sampled in two independent health surveys, the Model Reporting Area for Blindness Statistics (MRA) and the National Health and Nutrition Examination Survey (HANES) to compare the prevalence of cataract in geographical areas with varying total annual hours of sunlight. The total annual hours of sunlight was obtained from U.S. Weather Bureau statistics and ranged from 1800 to 3800 hours. For each health survey ocular diseases other than cataract were used as control and the data reported as age-specific ratios of cataract cases to control cases for areas of differing total annual insolation. The findings suggest that in the youngest age group studies (20-44 yrs) there is no greater incidence of cataract in areas of high annual sunlight, but that with increasing age there appears to develop an increasing

association of cataract with sunlight exposure. In the 65-74 yr sample population, there was at least a doubling of the ratio of cataract to each control disease between the lowest and highest sunshine area and in persons 75 years and older this trend was even more pronounced. The study did not consider the possible effects of genetic or socioeconomic differences among the populations from different areas nor did it consider the actual sunlight exposure of the individuals (e.g. indoor vs outdoor occupation).

Zigman, Datiles, and Torczynski (1979) have studied populations from three widely separated areas (Manila, the Philippines; Tampa, Florida; and Rochester, New York) which differ considerably in the yearly levels of ultraviolet radiation in sunlight. This study considered not only the age of the individuals studied, but also considered separately those with indoor and outdoor occupations. The findings which were based on study of extracted cataracts included analysis of the cataract type for each lens. The results indicated that no correlation existed between geographic location and distribution of cataracts except for the brunescent cataracts; i.e., those nuclear cataracts with significantly increased pigmentation levels. In Manila, the area with greatest UV, such cataracts accounted for 43% of total cataracts while in Tampa 20% of cataracts were brunescent and in Rochester, the area of least UV, only 9%. In all three populations, the percentage of brunescent cataracts extracted in persons with outdoor occupations was markedly higher than in those persons working indoors. Thus, both the latitudinal variation and the individual differences within geographic regions suggested a strong relationship between UV exposure and brunescent cataract. These data are consistent with numerous reports that tropical areas have higher cataract incidence than areas at higher latitudes and that the percentage of brunescent cataracts is higher in tropical areas (Pirie 1972 and references in Taylor 1980). The confounding factors which were pointed out by Zigman, Datiles, and Torczynski (1979) as not being controlled in their study included the economic, nutritional and genetic backgrounds of the individuals in the respective populations. While these differences would probably be substantial, particularly between Manila and the two U.S. sites, this study is significant in that it involved some biochemical evaluation of the cataracts as well as the epidemiological data.

Two recent epidemiological studies have concerned cataracts in Australian aborigines, a rural population exposed to relatively intense solar radiation. Taylor (1980) studied 350 individuals from which detailed personal histories were obtained. The possible role of a variety of personal and environmental factors in cataractogenesis were investigated. Among the 350 individuals, all of whom were over 30 years of age, 116 had lens opacities as determined by slit-lamp examination. The major findings relative to the possible influence of radiation exposure to cataractogenesis were a trend toward association of cataracts with increased hours of sunlight and with higher annual mean UV-B levels. These trends were reflected by a strong association of cataract with lower latitudes. No other environmental factors studies appeared to be associated with cataract nor did any personal factors other than age. No correlation would found between occupation and cataract; it is not clear whether this finding is inconsistent with Zigman, Datiles, and Torczynski (1979) since it is not reported whether any of the individuals studied had

indoor occupations. Unfortunately, the types of cataracts present were not reported in this study (Taylor 1980).

In a second study involving a much greater geographical area and over 50% (64,307) of the total Aborigine population, Hollows and Moran (1981) also found a statistically significant correlation between environmental UV irradiation and the prevalence of cataract (p 0.005). Additionally cataracts occurred much more frequently in the younger age group (40-59 yrs) in geographical zones of high UV radiation than in zones of low UV irradiation. The authors suggest that the Aborigines from remote rural Australia are a suitable population for such studies since their lifestyle tends to be highly uniform throughout the country. In contrast to these results, a large sample of non-Aboriginal people from the same areas showed much lower levels of cataract, particularly at younger ages, and there was no correlation between cataract prevalence and sunlight in this group. This was attributed to the much higher standard of living and the greater likelihood of indoor occupations in this group.

The data of Hollows and Moran (1981) may be used to estimate the number of senile cataracts which are caused by sunlight if we can accept the confounding factors mentioned above. Exhibit 10-1 presents the prevalence of cataracts found in aborigines by UV zone for three different age groups. Exhibit 10-2 compares the prevalence of cataracts found in non-aborigines and aborigines who live in the same regions.

Exhibit 10-1 shows that 13.6% of the aborigines in the lowest solar region develop cataracts while about 30% in the more solar intense zones develop cataracts. These data appear to indicate that about 15% of the senile cataracts are due to solar exposure. If the aborigine and non-aborigine are compared (Exhibit 10-2) there was about a 13% increase in cataracts which are probably due to solar radiation. More recently, Weale (1982) has presented a method of estimating the risk attributed to light for the incidence of senile cataracts and reported a risk factor of 5. If the risk factor may be expressed in percent form or 20%, the results are not too different from the above. It is interesting that the two sets of diverse data present such interesting results. Incidentally, the 18.3% prevalence for cataracts for the non-aborigine compared quite favorably to the Framingham Study (Kahn et al. 1977a and b) and the Gisborne Study (Martinez et al. 1982).

Thus, there appears to be a consensus from these epidemiological studies in support of the notion that senile cataract or at least a particular segment of the heterogeneous mixture of opacity types which comprise senile cataracts, is associated with higher exposure to sunlight. Substantive questions could certainly be raised concerning each of the studies cited above; however, taken in aggregate they represent a variety of approaches, using different types of populations, different criteria for cataract, different sampling and statistical methods, and different variables to test the same hypothesis. It is striking that the general conclusions of each study are so similar.

EXHIBIT 10-1

Cataract Prevalence by UV Zone

UV Zone	Age 0-39	40-59	60+
1	0	1.7%	13.6%
2	0	2.6%	24.2%
3	0.1%	3.7%	29.5%
4	0.1%	3.8%	30.5%
5	0.2%	5.1%	29.8%

Source: after Hollows and Moran 1981.

EXHIBIT 10-2

Comparison of Cataract Prevalence for Aborigines and Non-Aborigines

Ethnic Group	Age 50-59	60+
Non-aborigine	0.8%	18.3%
Aborigine	4.4%	29.3%

Source: after Hollows and Moran 1981.

Ocular Transmittance

The effect of nonionizing radiation on a cell depends on the specific chemical composition within the cell, that is, on the presence of absorbing molecules or chromophores (Lerman 1980a). This type of radiation must be absorbed in order to cause a change in the molecule since absorbed energy is required to promote a chemical change. Molecules in excited electronic states have different chemical and physical properties than their counterparts in the ground state (prior to absorption of energy). Thus cells that do not contain chemical compounds absorbing at certain wavelengths will transmit these wavelengths. For example, the nucleic acids and most proteins in a cell are essentially transparent to and completely transmit visible light but absorb certain wavelengths in the UV region (between 250 and 295 nm) and can be damaged by this form of radiation, while other macromolecules in a cell such as rhodopsin, which absorbs at 498 nm, and hemoglobin, which has absorption peaks in the UV and visible region (275, 400, and 540 to 576 nm), appear colored since they absorb visible light. These latter macromolecules can be damaged by high intensities of visible radiation at their specific absorption wavelengths.

The transmittance data for the rabbit, primate and human eye and ocular media for the wavelength range of 200nm to 2500nm are given in Exhibits 10-3, 10-4 and 10-5. Exhibits 10-6 and 10-7 present transmittance data for the 200 to 400 wavelength range (Kinsey 1948; Boettner and Wolter 1962; Maher 1978; Barker 1979).

The Action Spectrum

Since the absorption of nonionizing radiation is determined by the chemical composition of the tissue being exposed, the more radiation that the molecules absorb the greater will be the effect of the radiation. The term "action spectrum" is used as a measure of the relative effect of different wavelengths of radiation on a chemical compound, macromolecule, cell, or entire organism. The action spectrum is a plot of the dose or radiant exposure necessary to produce the defined effect versus the wavelength. For example, the maximum efficiency for experimental photokeratitis has been shown to occur at approximately 300 nm, with a smaller peak at 295 and 320 nm (Pitts 1978 and Pitts and Cullen 1981). The action spectra for photokeratitis, cataracts and retinal lesions for the rabbit, primate and human are presented in Exhibits 10-8 and 10-9.

The eye is the only organ or tissue in the body (aside from the skin) that is particularly sensitive to the non-ionizing wavelengths of radiation (longer than 280 nm) normally present in our environment. In addition to infrared and visible radiation, we are constantly exposed to ultraviolet radiation (solar and man-made) throughout life. It is estimated that approximately 8% (11 mW/cm^2) of solar radiation above the atmosphere is in the ultraviolet region (280-400 nm). At sea level this is decreased to 2-5 mW/cm^2, depending on geographic location and season (Lerman 1980b).

EXHIBIT 10-3

Composite Transmittance Curves for the Rabbit With Representative Data for Boettner and Wolter (human, 1962), Weisinger (rabbit, 1956) and Kinsey (rabbit, 1948) for Comparison (after Barker, 1979)

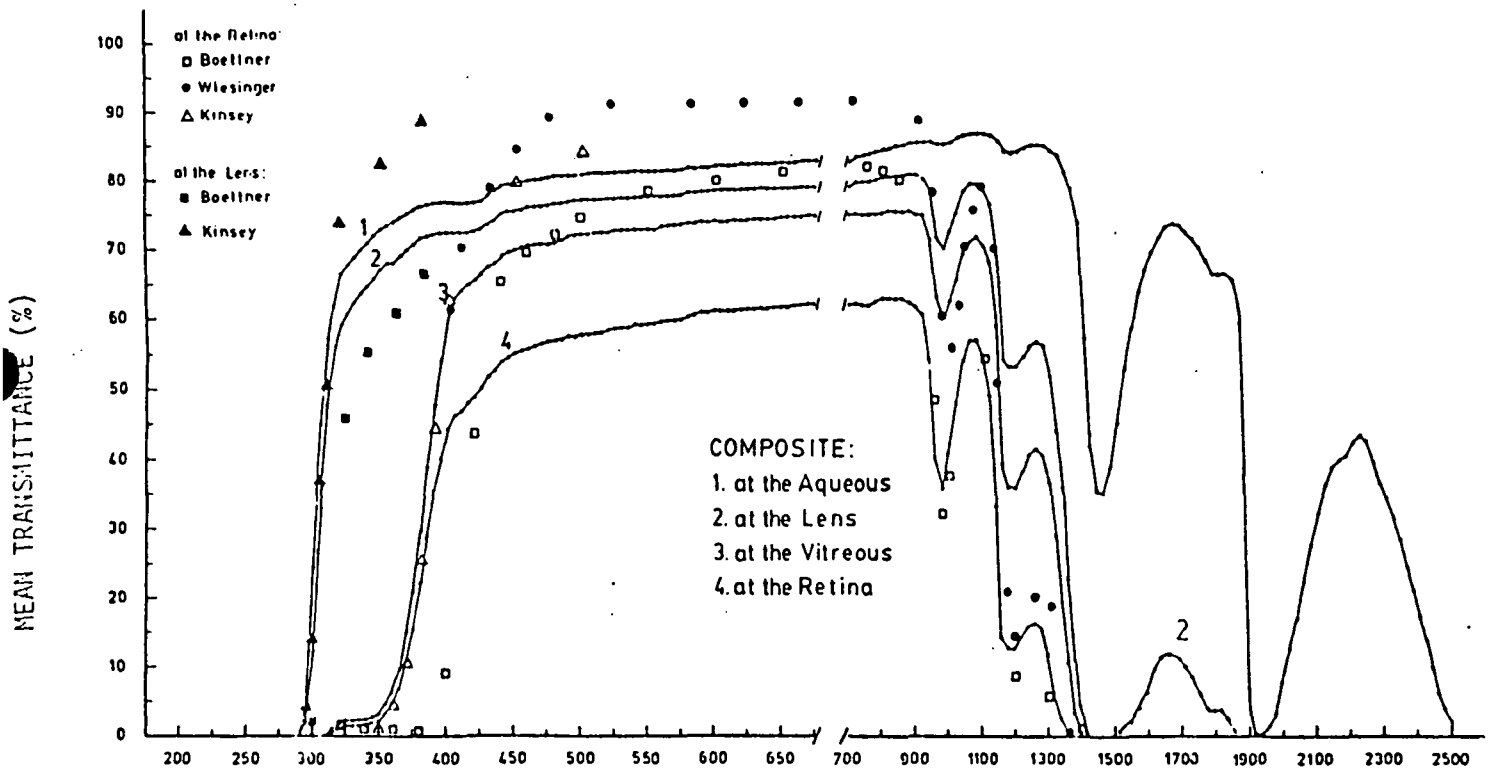


EXHIBIT 10-4

Calculated Total Transmittance of the Human Eye
(after Boettner and Wolter 1962)

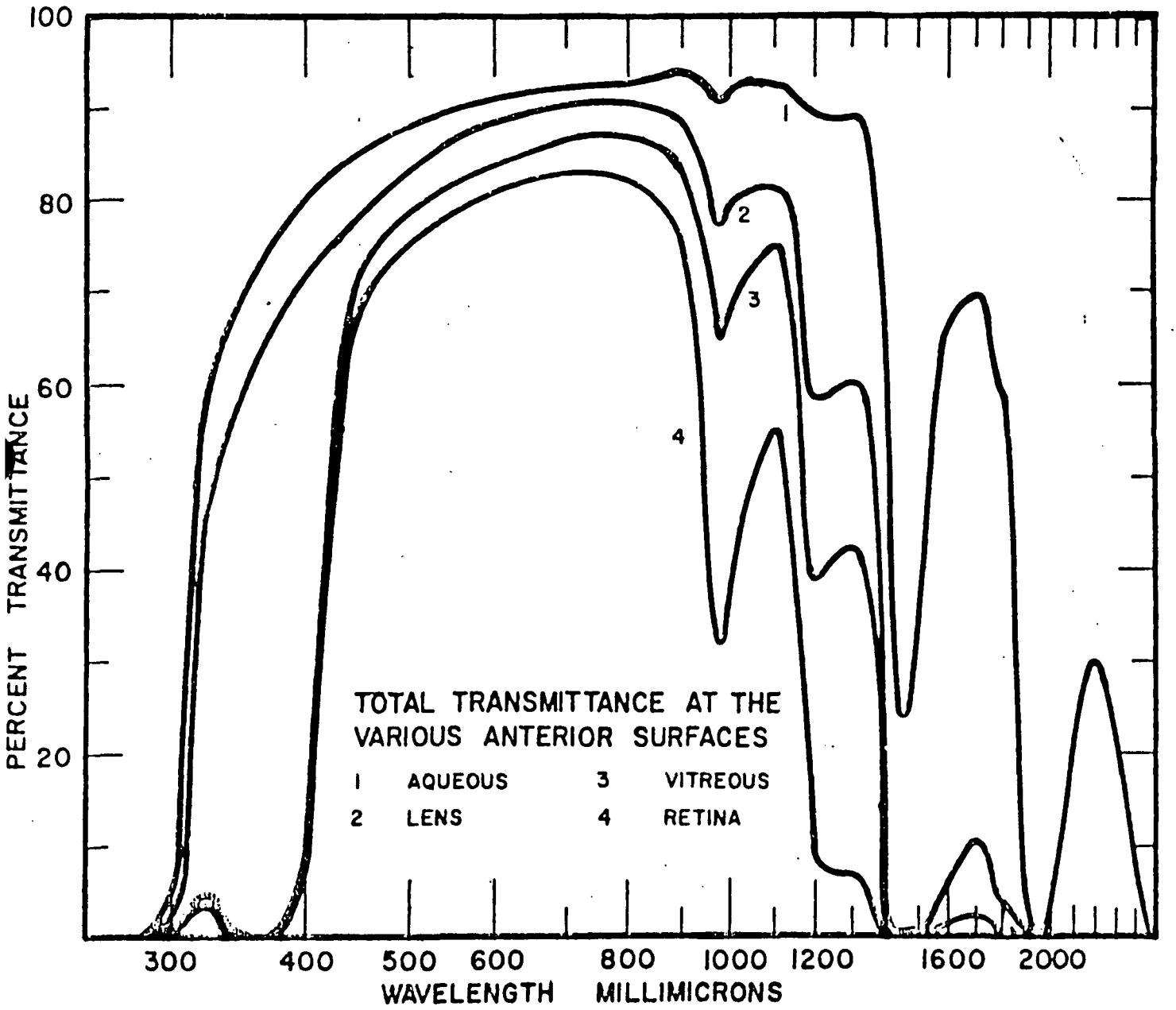


EXHIBIT 10-5

Percent Transmissivity Through the Entire Rhesus Eye
(after Maher 1978)

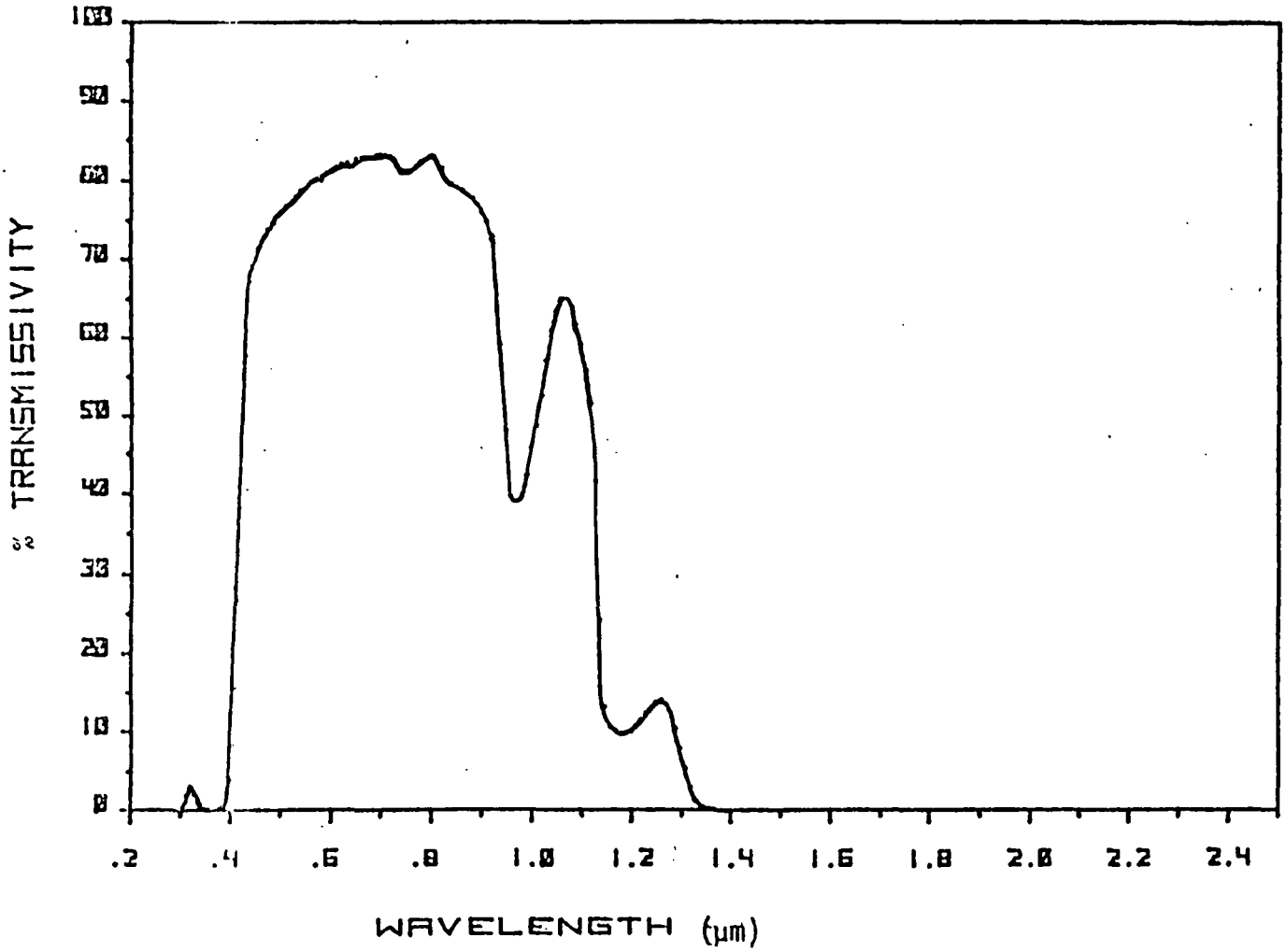


EXHIBIT 10-6

Transmittance of the Total Rabbit Cornea, the Total Human Cornea, and the Rabbit Corneal Epithelium (after Kinsey [1948] and Boettner and Wolter [1962])

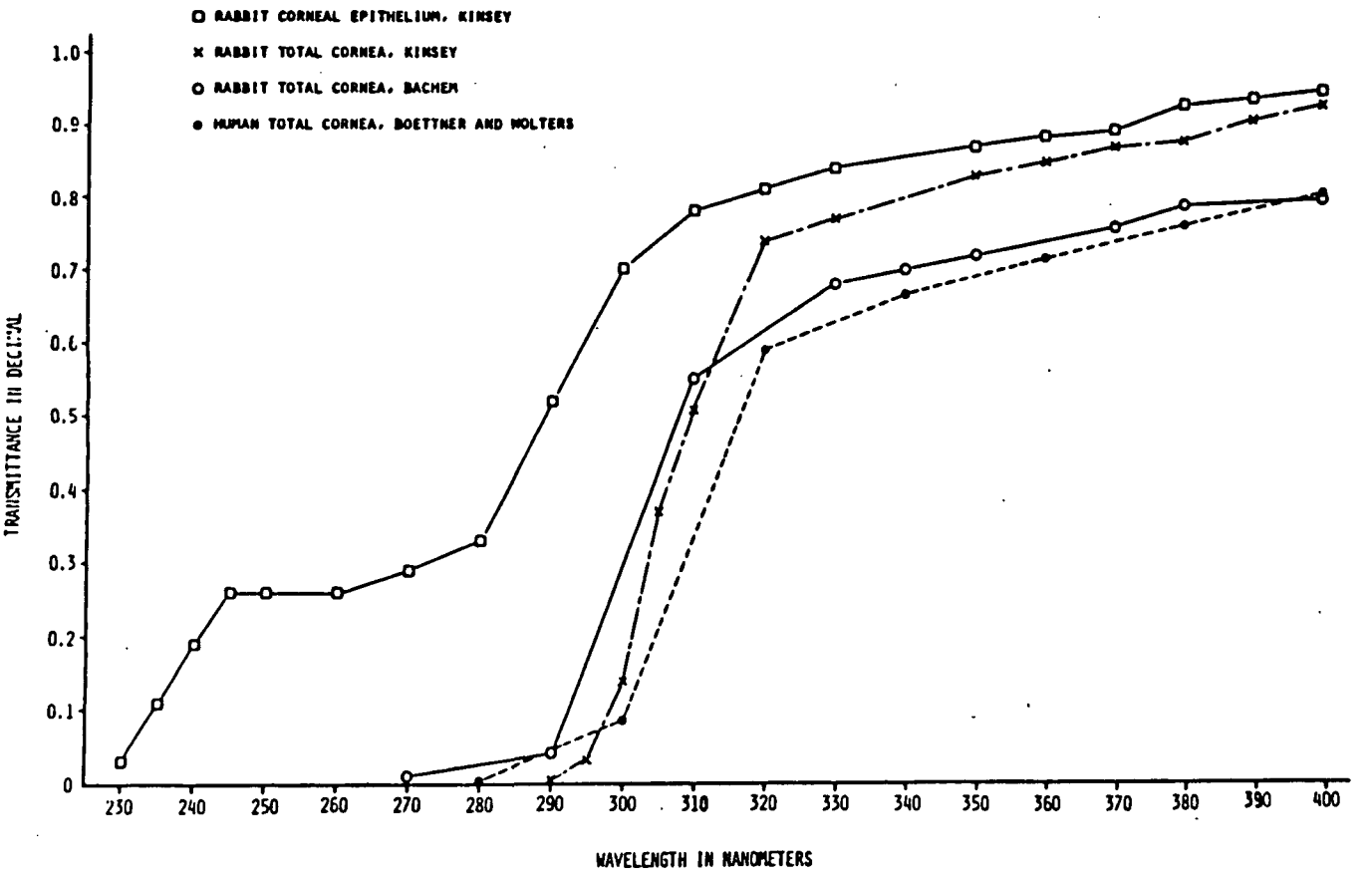


EXHIBIT 10-7

Transmittance of the Anterior Ocular Structures of the Human and Rabbit Eyes. The Symbols Indicate the Percentage of Radiant Energy Incident On a Certain Structure (after Pitts, Hacker, and Parr 1977)

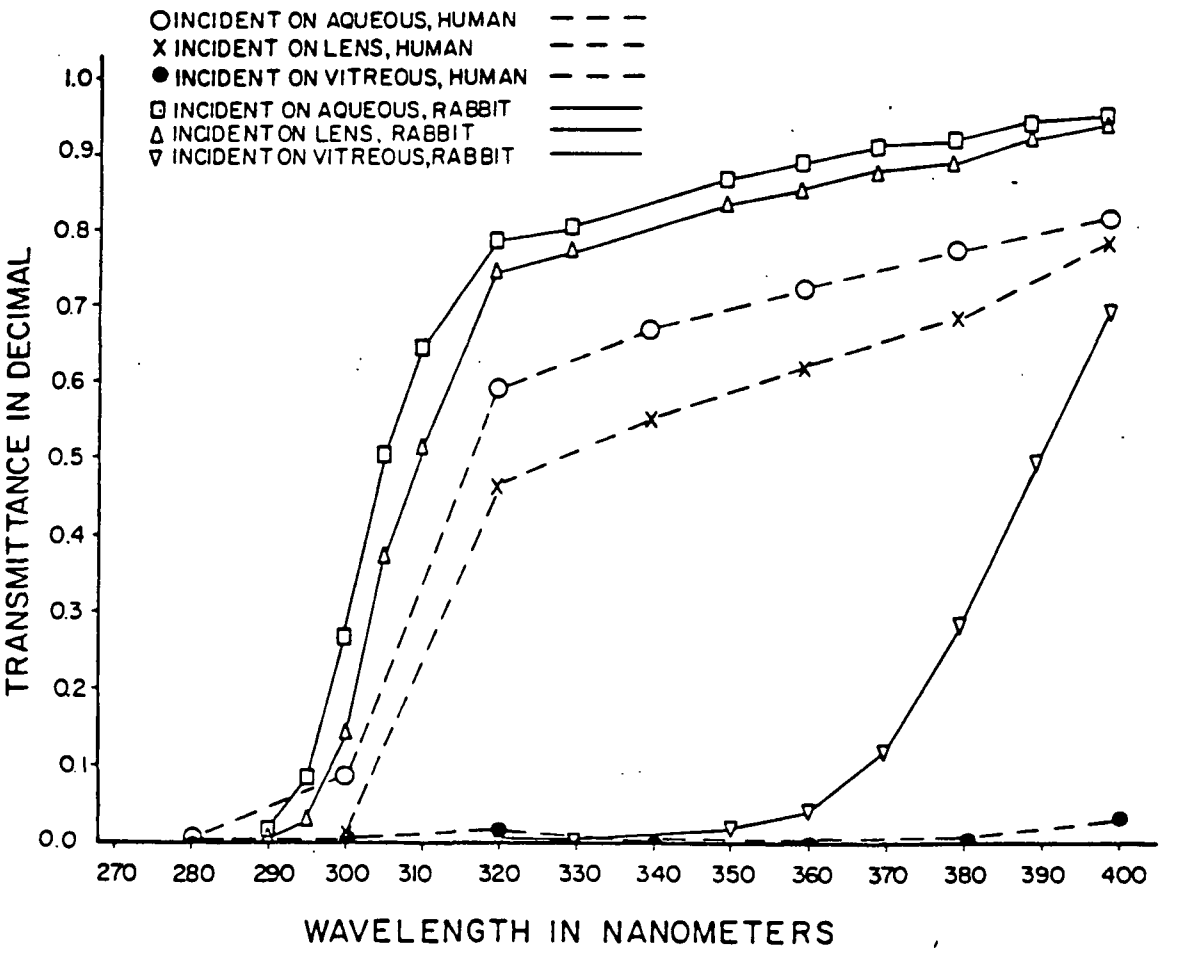


EXHIBIT 10-8

UV Radiant Exposure Threshold Data for the Cornea H_c, Lens H_L Cataracts, and Retina H_R for the Rabbit and Primate

WAVELENGTH	CORNEAL Radiant Exposure H_c in J/cm²		LENS Radiant Exposure H_L in J/cm²		RETINA Radiant Exposure H_R in J/cm²	
nm	Rabbit	Primate	Rabbit	Primate	+Rabbit	*Primate
210	0.17	0.33				
220	0.046	0.021				
230	0.03	0.022				
240	0.033	0.012				
250	0.041	0.020				
260	0.018	0.011				
270	0.005	0.004				
280	0.11	0.006				
290	0.012	0.007	3.00			
295			0.75			
300	0.022	0.01	0.15	0.12	0.225	0.12
305	0.07	--	0.30			
310	0.05	0.02	0.75			
315	7.25	--	4.50			
320	7.5	9.6	12.60			5.0
325	18.0	--	50.00			
330	30.0	41.1				
335	30.0	--				
340	--	58.3				
345	--	--				
350	--	61.5				5.4
355	50.0	--				
360	--	88.4				
365	65.0	--				
370	--	130.				
375	--	--				
380	--	170.				8.1
385	--	--				
390	--	258.				
395	--	--				
405	--	--				15.0
441	--	--				30.0

* for 100s exposure duration in aphakic primate producing retinal lesion.

+ for 650s exposure duration in phakic rabbit eye producing changes in the retina.

EXHIBIT 10-9

The Action Spectra for Photokeratitis and Cataracts for the Primate and Rabbit (after Pitts 1981)

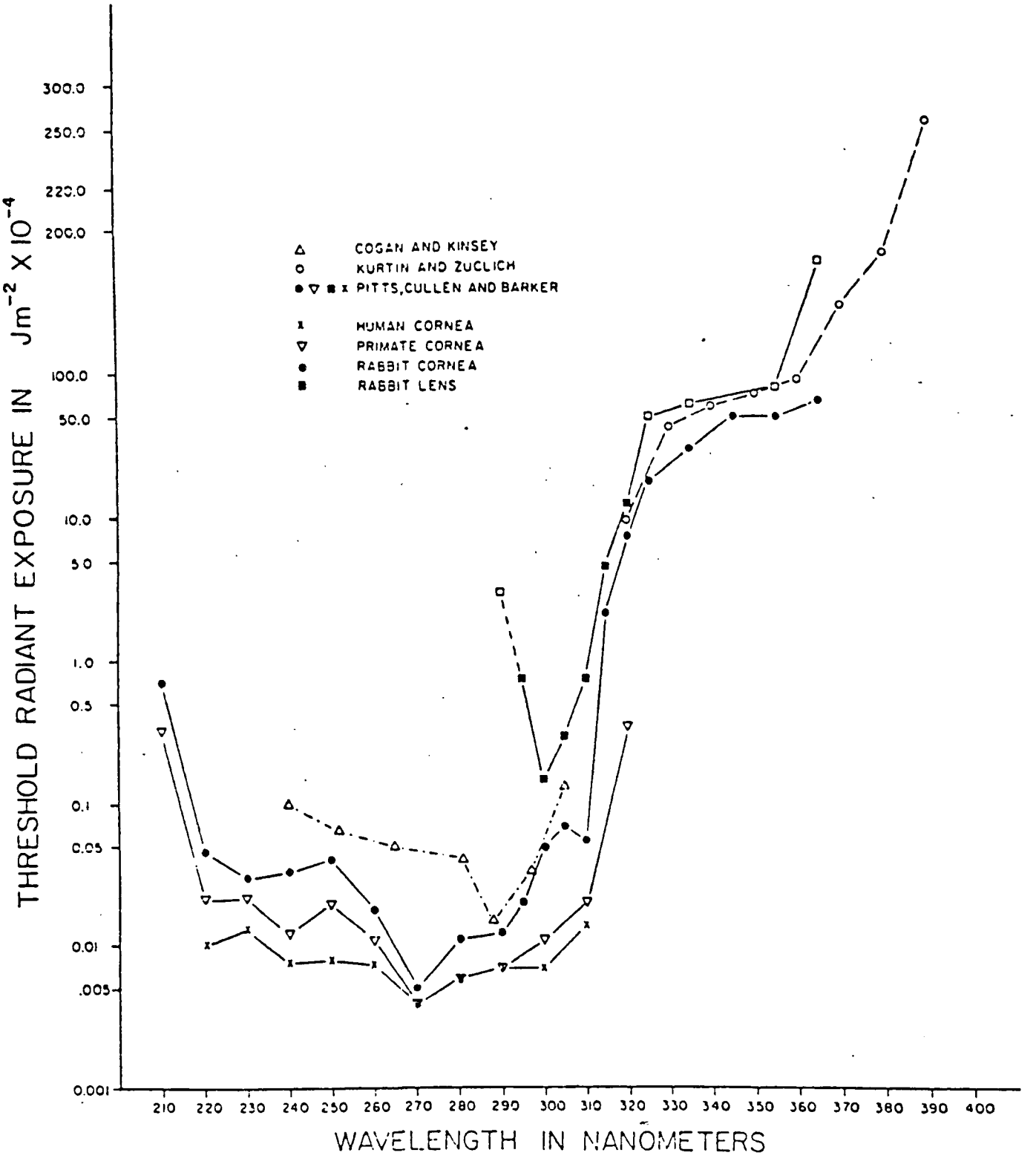
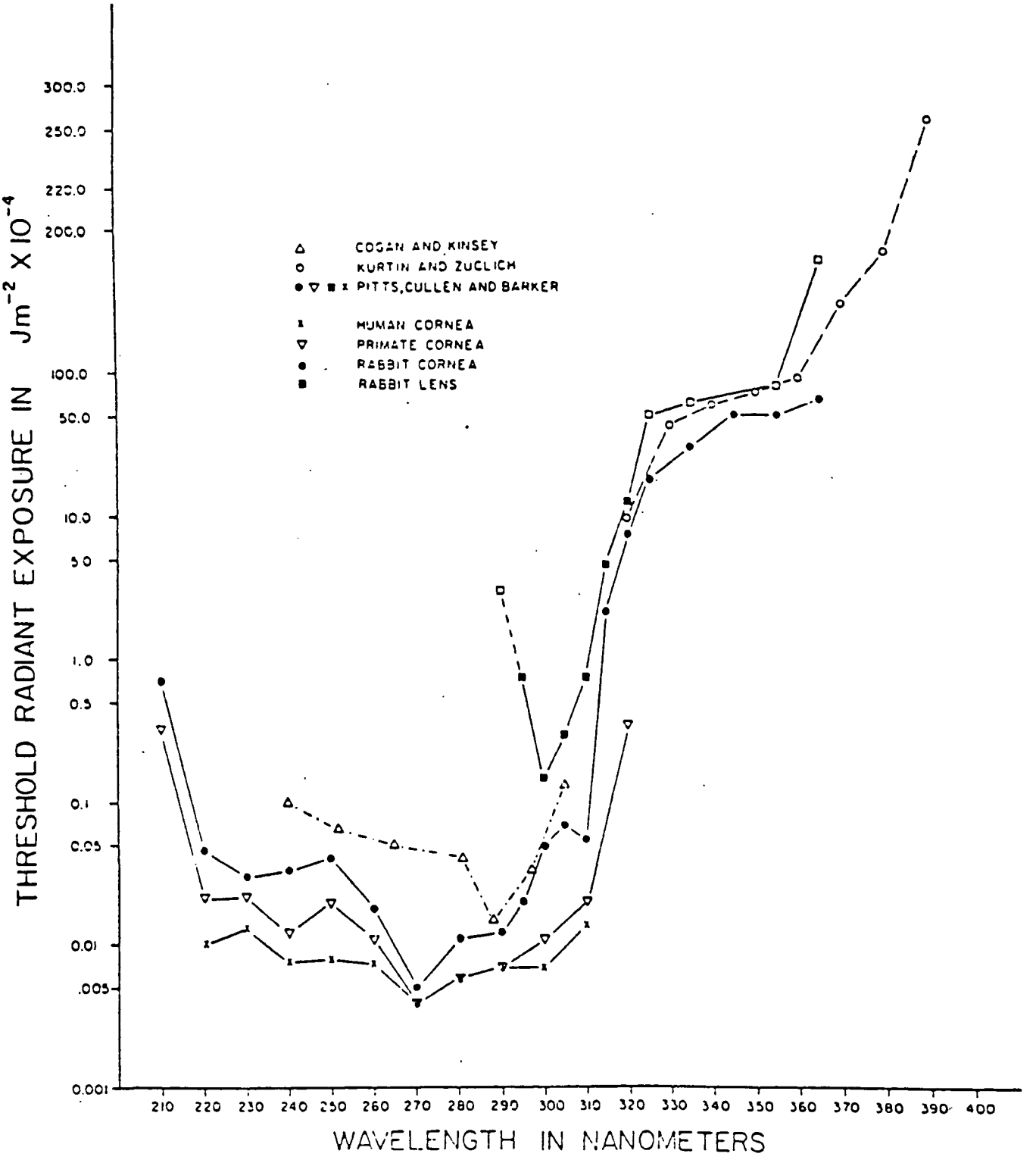


EXHIBIT 10-9

The Action Spectra for Photokeratitis and Cataracts for the Primate and Rabbit (after Pitts 1981)



Nature has provided us with transparent ocular media which are essentially avascular and contain very few visible wavelength absorbing chromophores in order to effectively transmit (as well as refract) the specific wavelengths required to initiate the visual process by photochemical reactions. However, these tissues do have the ability to absorb varying amounts of ultraviolet radiation (particularly the ocular lens). The shorter the wavelengths of radiation absorbed the greater the potential for photic damage since there is an inverse relationship between a wavelength and the photon energy association with it. Thus, UV radiation is the non-ionizing portion of the electromagnetic spectrum which could cause the most damage, provided that it is absorbed. This axiom applies to all the ocular tissues including the retina in the very young eye where the lens has not as yet become as effective UV filter but, in particular, the ocular lens sustains the greatest amount of photochemical change during a lifetime of exposure to ambient UV radiation.

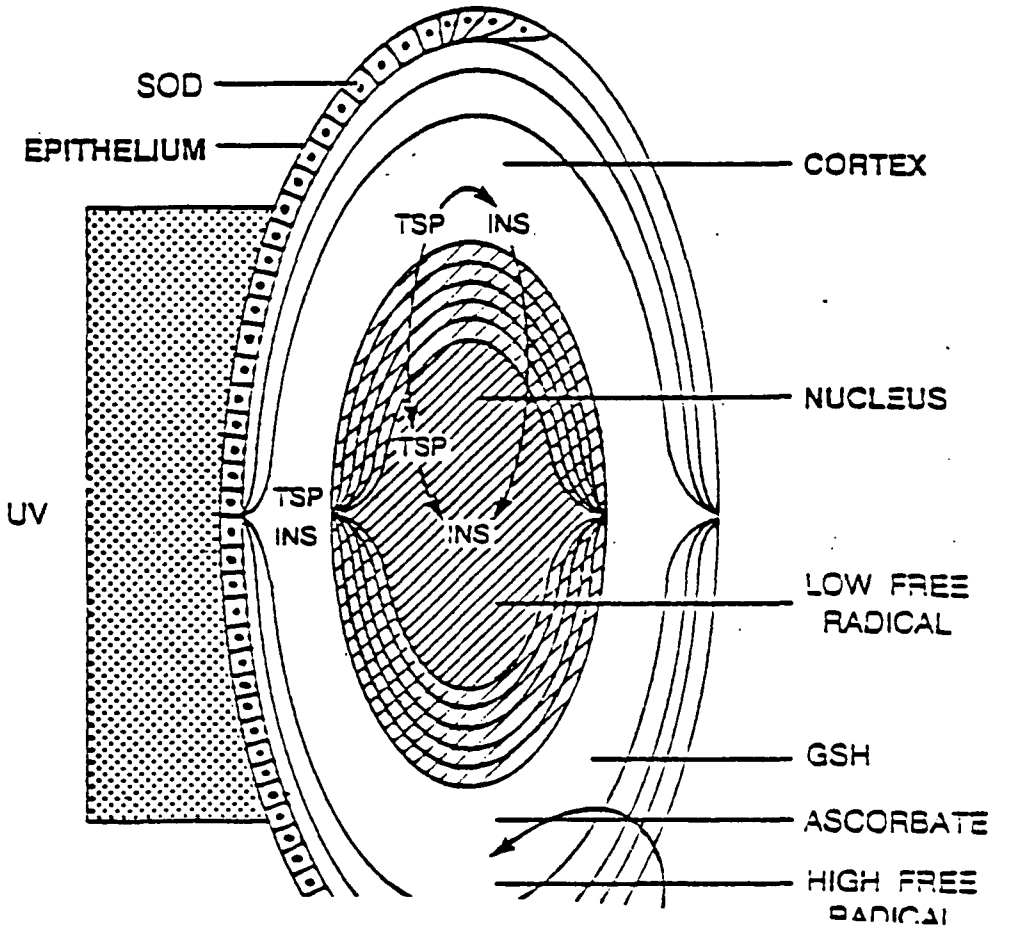
Some Biochemical Mechanisms

There are several mechanisms which are biochemically related to radiation damage to the eye and the lens in particular. These mechanisms include photo-oxidation of free and protein bound tryptophan, photosynthesis processes involving the activated species of oxygen, disruption of the cation transport system and damage to the nucleic acids (DNA) of the lens epithelium. Some of these mechanisms have only been studied only in cultures, some in vitro and others in vivo. It is important to be able to project the mechanism to a "real live" situation in order to be able to evaluate the relative importance of the factors involved in inducing the senile cataract. Prior to discussing these mechanisms it may be desirable to review some of the basic concepts in the biochemical mechanisms of radiation induced damage; therefore, some current interpretations of lens free radicals and oxidation reduction reactions relative to cataract follows. Exposure to UV radiation initiates enzymatic activity involved in cellular protection from oxidative processes which may be due to both light and metabolism (Exhibit 10-10). Catalase destroys hydrogen peroxide, which is produced in all cells by metabolism; superoxide dismutase (SOD) has the responsibility of destroying the superoxide radical, a very toxic radical due to its powerful oxidative nature. In the lens cortex, there is very little SOD or catalase, but they are highly concentrated in the epithelial cells. Of all the ocular tissues, the greatest concentrations of these enzymes are present in the retina. In the lens cortex there are other agents to protect against oxidation such as glutathione (GSH) and asorbic acid. There is only a small amount of vitamin E in the lens, but attempting to protect animals from ocular tissue oxidative damage by feeding them high levels of vitamin E or other anti-oxidants has not succeeded. The sum total of all of the anti-oxidants that are present in the ocular tissues still does not protect against the formation of free radical oxidative reactions to near-UV radiation. Another toxic oxidant that can form is singlet oxygen.

The content of the stable free radicals previously described is highest in the cortex and seems to decrease in the nucleus, of the lens. The process of aggregation of soluble proteins (TSP) occurs both in the cortex and in the nucleus. Because of the conservative growth process of the lens, much of the

EXHIBIT 10-10

Free Radials and Oxidation: Reduction Systems



SOD represents superoxide dismutase, GSH represents glutathione. Epithelial SOD and catalase protect these cells against oxidation from excessive hydrogen peroxide and superoxide anion. High levels of ascorbic acid and glutathione protect cortical fibers against oxidation. TSP represents soluble and INS insoluble protein. TSP converts to INS protein both in the cortex and in the nucleus. But INS accumulates more in the nucleus than in the cortex. Free radicals are quenched in nucleus due to binding to proteins.

aggregated material remains in the nucleus indefinitely and often in a form that is associated with the fiber cell membranes. The lens fraction called the insoluble fraction contains both membranes and the aggregated proteins. There is a chemical reaction between the free radicals and proteins so that the protein binds to free radicals chemically, which quenches the ESR (electron spin resonance) signal. Multiple molecular species of proteins can be associated through new crosslinks leading to aggregation and, therefore, light scattering. In the nucleus of the lens, the free radicals are quenched by their reactions with proteins. Since all enzymes are proteins, this may illustrate a universal process whereby enzyme activity is inhibited by near-UV radiation. These changes would eventually cause malfunction of all cells and tissues.

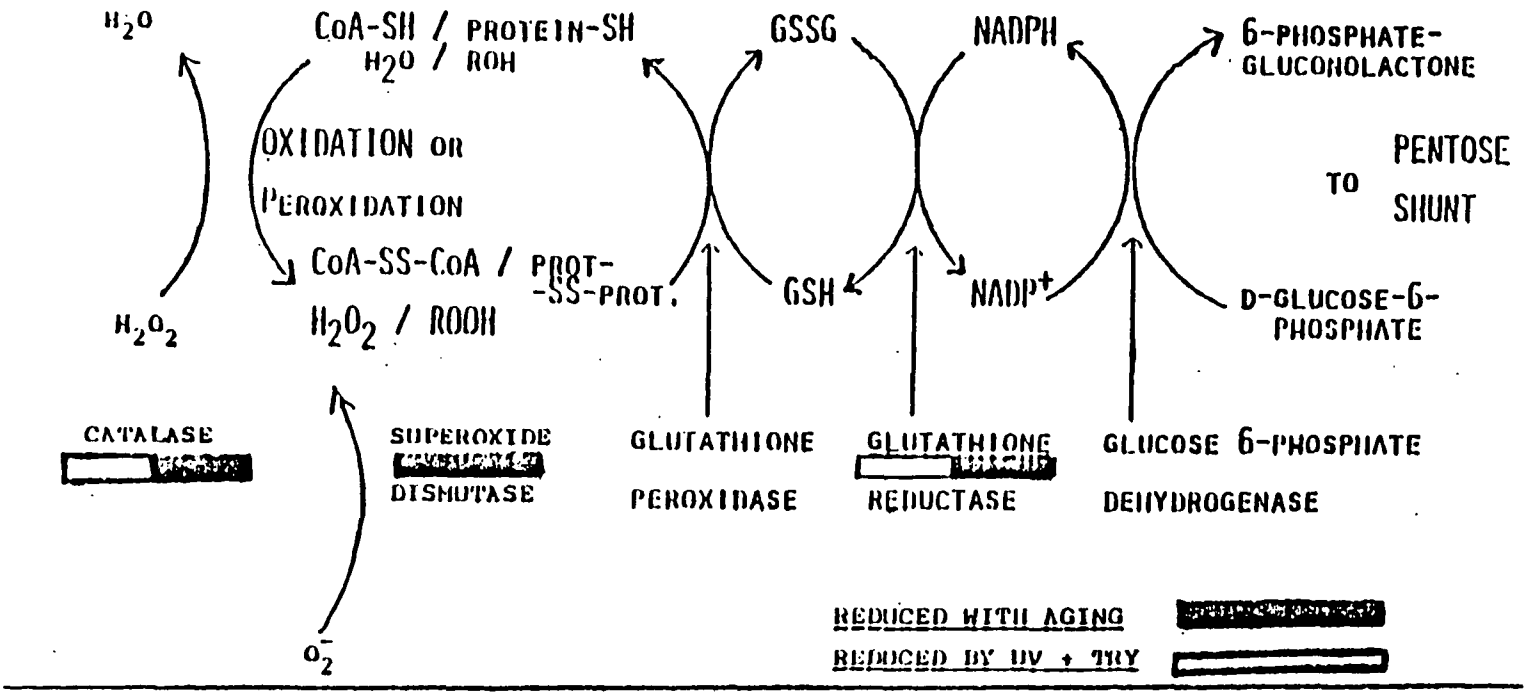
Exhibit 10-11 shows a scheme of a series of enzymatic oxidation: reduction reactions going on in all cells that are influenced by aging and exposure to near-ultraviolet radiation. These reactions are all linked together. The co-factor NADH/NADP system is linked to the oxidation and reduction of glutathione (GSH). When oxidation occurs, reduced GSH becomes oxidized GSSG, changes which also relate to protein oxidation involving SS-crosslinks. Many enzymes, in order to remain active, must maintain sulfhydril groups (SH) of cysteine in reduced form in order for the protein portion to maintain its activities as an enzyme. Many enzymes require free-SH groups in their active sites to retain their activity. If these enzymes are oxidized so that disulfides form, they are inactivated. For example, if high concentrations of hydrogen peroxide are generated, protein SH will convert to SS thereby inhibiting the activity. Several of the enzymes referred to in Exhibit 10-11 are known to be reduced with enhanced aging in most living cells. In many cases the cells turn over, so that cells' enzymatic activities are maintained, but where there are populations of cells that do not turn over very actively, a cumulative process can lead to a great loss of enzyme activities. This may occur in the lens.

One enzyme that is diminished with aging is glutathione reductase and it also diminishes under in vitro circumstances in living tissues when near-UV radiation is provided in the presence of excess tryptophan. There has not yet been an aging or photoinactivation study of glutathione peroxidase. The SOD is very important as a protective agent because it destroys the very toxic superoxide radical. SOD activity does diminish with aging, but is not sensitive to destruction from near-UV radiation (with tryptophan as a sensitizer). Because the lens substance contains little oxygen to serve as a superoxide source, the site of lens damage would be the epithelium if SOD activity were to be diminished drastically. Damage to the lens epithelium would certainly lead to both physiological and developmental anomalies in the lens and cataract. The SOD activity must be maintained in the retina, however, due to its high oxygen tension and the potential for oxidation to occur more readily.

Hydrogen peroxide (H202) is another very powerful oxidant that is present in ocular tissues and fluids by virtue of the action of SOD on superoxide and other metabolic systems, such as the glutathione and ascorbic acid cycles. Recently, high levels of H202 (mM concentrations) have been

EXHIBIT 10-11

Enzyme Systems Involved in Oxidation: Reduction



Enzyme activities that are decreased with aging are shown by solid bars and those decreased by light exposure (artificially induced) are shown by the open bars.

observed in the human aqueous humor by Garner and Spector (1980). This concentration of H202 is suspected of being capable of causing cataracts by poisoning important enzymes and by crosslinking proteins (Spector and Garner 1982). In eyes without lenses, H202 from the aqueous humor could more easily diffuse even to the retina (Zigman 1981, Kramer 1980), a highly hazardous circumstance. Ocular tissues utilize the enzyme catalase to destroy and detoxify H202. Since this enzyme is diminished with aging and has been shown to be inhibited by near-UV radiation plus tryptophan (Zigman, Yulo, and Griess 1976), its activity loss would allow heightened levels of H202 to damage ocular tissues. A large imbalance in favor of H202 accumulation would lead to extensively altered ocular tissue proteins and much enzyme inactivation.

Glutathione reductase is another enzyme of great functional importance to the lens and retina, and it is diminished with aging and UV exposure (Kalustian, Sun, and Zigman 1978). Its loss of activity would estimate oxidation of small molecules and proteins in most ocular tissues.

Another enzyme (not involved in oxidation: reduction, but significant with regard to its photoinactivation) is Na^+K^+ ATPase. This enzyme is also sensitive to near-UV radiation in the presence of tryptophan and loses its activity accordingly. In the lens, it maintains the osmotic balance by controlling the sodium and potassium cation exchange and prevents water inhibition and swelling. In some genetic cataracts in mice, it has been found that the cause of cataract is the high concentration of an inhibitor of ATPase in the lens (Kinoshita 1974). It is likely then that a strong inhibition of lens epithelial cell ATPase by light-sensitized action could be a major factor in cataract formation osmotically. In the retina, the ATPase activity is very important in terms of the chemical reactions that support the visual process. Should this enzyme be photochemically inhibited in the retina, the functioning of the visual process would be markedly reduced. Retinal Na^+K^+ ATPase is also sensitive to inhibition by near-UV plus tryptophan.

Before discussing the biochemical studies which pertain to the possible cataractogenic effects of optical radiation, it may be pertinent to review some contrasts between human lenses and those of the most commonly used laboratory animals. All vertebrate lenses are composed essentially of a single cell type, the lens fiber, which differentiates from a single layer of epithelial cells present only at the bow or equator of the lens. New fibers are continually laid down at the periphery; thus, the oldest tissue is located at the center or nucleus of the lens and the cells become progressively younger as one moves from the center toward the lens capsule in the cortex. Cells are never sloughed from the lens and this makes the cells in the lens nucleus as old as the animal. Furthermore, differentiated lens fibers gradually lose virtually all cell organelles, including nuclei, and lose the capacity to synthesize protein as they age and are forced toward the nucleus. Therefore, the proteins, primarily lens crystallins, present in the aging human lens may be the longest-lived proteins in the organism. This means that unlike most other tissues the central portion of the lens does not have the ability to replace damaged proteins with newly synthesize proteins. This may be an important factor in the presumptive long-term effects of chronic

exposure to near UV radiation. There are very clear differences between the lens nucleus and cortex in terms of their biochemistry and the loss of the capacity to repair or replace damaged proteins probably accounts for much of the difference.

There is at least one very significant difference between human lenses and the common laboratory animals and that is the presence of pigmented compounds in the human lens. While the lenses of most non-primate mammals have no pigment, the lenses of diurnal primates and a few other strongly diurnal species are yellow. Cooper and Robson (1969) demonstrated that in the human lens there are two classes of pigmented compounds. One group that is present even before birth is of low molecular weight, is water soluble and absorbs maximally at about 365 nm. A second class of colored compounds appears later, increases with age, is bound to the lens proteins and absorbs maximally near 320 nm. This latter class of chromophores is localized primarily in the lens nucleus and may be responsible for the age-related increase in pigmentation in human lenses. Since these chromophores absorb in the near UV, they may be major determinants of the effects of such radiation on the human lens.

Studies at the Biochemical Level

Studies at the biochemical level have generally been concerned with the structural modifications which crystallins undergo during aging and cataractogenesis and have attempted to explain these reactions in terms of photo-oxidative mechanisms. It is well-documented that crystallins, particularly in the lens nucleus, accumulate a variety of modifications. These include the formation of disulfides and other covalent crosslinks, the development of a novel blue fluorescence, progressive pigmentation, oxidation of methionine, racemization of aspartate residues, polypeptide chain cleavages, deamidations, aggregation and ultimate insolubilization. The lack of turnover of protein in the lens nucleus accounts for the accumulation of these modifications and they have been the subject of several recent reviews (Zigler and Goosey 1981; Hoenders and Bloemendal 1981; Harding 1981). It is clear that most of the protein changes are the result of oxidative stress, and the possible role of radiation in that stress has been the subject of much study over the last 15 years.

Pirie (1968, 1972) studied the effects of sunlight on solutions of lens proteins and of other proteins. The proteins became brown following irradiation and analysis of absorbance changes indicated similarity to those found in lens proteins from cataracts. Pirie also found decreased levels of the oxidation sensitive amino acids, histidine and tryptophan in protein from cataracts relative to normal lenses. Although the data for tryptophan was subsequently retracted as an artifact, Dilley and Pirie (1974) suggested that photooxidation of tryptophan residues, perhaps with formation of N'-formyl kynurenine, was a primary step in cataractogenesis. These studies stimulated a number of other investigators.

Kurzel (1973) performed fluorescence and phosphorescence measurements and Weiter and Finch (1975) ESR studies on human lenses and found signals which they believed to be due to tryptophan free radical species in lens proteins.

Van Heyningen (1971) was able to identify several of the components contributing to the color of human lenses as kynurenine derivatives, species which can be derived from tryptophan either metabolically or photooxidatively. These were part of the low molecular weight colored material present in human lenses and Van Heyningen (1973) subsequently showed that lens proteins exposed to sunlight in the presence of these compounds were photo-oxidized more extensively than in their absence. The mechanisms of this accelerated photo-oxidation was not determined.

In addition to oxidation of protein bound tryptophan other possible mechanisms were explored. Zigman et al. (1973) and Zigman and Vaughan (1974) demonstrated that photo-oxidation of free tryptophan yielded pigmented and fluorescent species which would bind to lens crystallins in vitro. This raised the possibility that the target of photo-oxidation could be either free or protein-bound tryptophan. Numerous investigators turned to the study of brunescent nuclear cataracts since these lenses have the greatest concentration of the pigment and of the non-tryptophan fluorescence. The search for clear decreases in the levels of tryptophan in the proteins of such lenses or in their free amino acid pool has not been successful to date (Dilley and Pirie 1974; Pirie and Dilley 1974; Zigler et al. 1976). It should be noted however that tryptophan comprises less than 2% of total amino acid in crystallins and, thus, small changes would be difficult to detect especially in view of the inherent problems of tryptophan analysis.

Lerman (1980b) suggests that in the normal lens less than 20% of the protein tryptophan is susceptible to photo-oxidative damage. Studies on the novel blue fluroescence of aging crystallins, particularly the insoluble fraction from brunescent lens nuclei suggest the presence of a number of related species (Lerman 1980b). The species with emissions in the visible are generally more concentrated in nuclear cataracts. There is some evidence that this fluorescence may be concentrated in certain crystallin polypeptides (Zigman 1981). It has also been demonstrated that the formation of non-disulfide covalent crosslinks between crystallin polypeptides is associated with the heavily pigmented protein fraction and can be generated in vitro by irradiation (Buckingham and Pirie 1972). Additional photoproducts of tryptophan, including B-carbolines (Dillon, Spector and Nakanishi 1976) and anthranilic acid (Truscott, Faull, and Augusteyn 1977) have been identified from the crystallins of nuclear cataracts. Dillon and Spector (1980), Dillion et al. (1982) and Borkman, Tassin, and Lerman (1981) have studied the photolysis of free tryptophan, or tryptophan containing peptides, and of isolated lens crystallins. Analysis of these data suggests that a variety of products are possible and that the microenvironment of individual tryptophan residues is of paramount importance. Additionally, photolysis is much faster in the presence of oxygen.

Harding and Dilley (1976), however, raised two objections to the idea that sunlight caused brown nuclear cataracts. First, they pointed out that the lens damage is in the nucleus whereas the shortest wavelengths reaching the lens; i.e., those which might be absorbed by tryptophan are probably absorbed in the anterior lens cortex. Indeed as noted above cataracts induced in animals by UV-B are located in the anterior cortex. Secondly, the lack of

evidence for loss of tryptophan in brown cataracts was cited. While these arguments are difficult to rebut in terms of mechanisms in which UV oxidation of tryptophan is the central event, there is another mechanism of photo-oxidation for which these objections may be less significant.

Recently, there has been increasing interest in the possible role of photosensitized processes, particularly with the involvement of activated species of oxygen, in the oxidative damage observed in the human lens. This work was spurred by data demonstrating light-mediated lens damage with such photosensitizing drugs as 8-methoxypsoralen, phenothiazines, and tetracycline and by the studies cited above by Pirie and Van Heyningen showing accelerated UV effects on crystallins in the presence of kynurenines and related compounds. Zigler and Goosey (1981) have demonstrated that several of these compounds endogenous to human lenses are capable of generating singlet molecular oxygen, a highly reactive species capable of damaging proteins as well as other biological molecules and structures. The ability of this oxidant to induce the oxidative changes characteristic of human crystallins has been established in vitro. It has also been demonstrated that such photosensitizing activity is present in the heavily altered insoluble lens crystallins from brunescent cataracts and to a lesser extent in the soluble crystallins of normal human lenses as well. Based on experiments in vitro, such a photosensitized process could account for the generation of each of the oxidative modifications presently known to occur in lens crystallins. Furthermore such a process seems consistent with nuclear localization of damage. In the lens nucleus there is no repair or replacement of altered molecules, thus allowing progressive accumulation of crystallins with oxidized residues some of which are photodynamic sensitizers. The UV-A which these species absorb will readily penetrate to the nucleus. Additionally while the lens cortex is protected by a battery of antioxidant defenses it is known that these defenses are markedly reduced in the nucleus (Hata and Hockwin 1977; Fecondo and Augusteyn 1983).

One could envision a system in which there was a slow rate of photo-oxidation ongoing in the lens controlled by antioxidant defenses and by the greatly reduced oxygen tension in the lens nucleus. The oxidative stress might have several components including direct UV oxidation of tryptophan (primarily UV-B), the high levels of H202 in the aqueous humor (Spector and Gardner 1982) and photosensitized oxidation involving activated species of oxygen. It has been recently demonstrated that even fetal human lenses contain low molecular weight chromophores which can generate singlet oxygen when irradiated with UV-A. The initial photochemical event could be absorption by such chromophores or direct photo-oxidation of tryptophan to produce N-formyl kynurenine (NFK) a known photodynamic sensitizer. In either case, it seems likely that the continued build-up of oxidized products in the nucleus is likely due primarily to a sensitized process, since such a process would not require large-scale tryptophan loss nor would it require penetration of UV-B into the nucleus. A relatively small number of stable sensitizing species such as NFK, bound within the long-lived nuclear crystallins, could continue to generate singlet oxygen indefinitely with the gradual accumulation of the various oxidative protein changes outlined above culminating in aggregation and insolubilization of much of the nuclear crystallins in advanced brunescent cataracts.

Several suggestions have been made that attempt to reconcile the finding that tryptophan is not reduced in sinile cataracts (as compared to normal lenses) with the hypothesis that it acts as an important endogenous photosensitizer. For example, it has been suggested that tryptophan is oxidized to a reactive molecule that simply transfers its energy to some other cell component from the exicted state. It then returns to the ground state without being destroyed in the process, yet photosensitizes damage to other lens macromolecules (Weiter and Finch 1975; Weiter and Subramanian 1978). Another explanation is that free tryptophan rather than protein bound tryptophan may be destroyed when it acts as a photosensitizer, whereas tryptophan incorporated within lens proteins is unaffected. The level of free tryptophan may be reduced in cataract (Zigler et al. 1976). The failure of protein-incorporated tryptophans to be photo-oxidized might be explained by the fact that they lie in very different micro-environments that render them less susceptible to photo-oxidation (Lerman, 1980b). A third suggestion to explain the finding that tryptophan is not decreased in cataractous lenses is that only a small percent of the protein-incorporated tryptophan may be susceptible to photo-oxidation and small losses in these susceptible tryptophan might not be detectable with current analytical capabilities. Tryptophan and its photoproducts have become the most frequent culprits implicated in causing photo-oxidative changes in lens proteins, changes that are hypothesized to be responsible for senile cataractogenesis.

Photo-oxidation of lens proteins has become a major topic in lens research and has been shown to be induced by photosensitizers that are either endogenous (Zigler and Goosey 1981) or externally applied (Goosey, Zigler, and Kinoshita 1980) to the lens. One effect frequently found to occur as a direct effect of UV or as an effect of UV plus a photosensitizer is crosslinking of lens proteins (Buckingham and Pirie 1972; Goosey, Zigler, and Kinoshita 1980).

Interest in agents that can cross-link or otherwise aggregate lens proteins has been high since Benedek (1971) first proposed that the mechanisms underlying senile cataract formation was aggregation and insolubilization of lens crystallins. They suggested that aggregated lens proteins would serve as light scattering particles in the lens. The search was then underway for these high molecular weight aggregates, but unfortunately, the research produced very inconsistent and conflicting results. Some research groups found an increase in the amount of high molecular weight proteins in senile cataractous lenses while other groups found no difference in the amount of high molecular weight proteins in cataractous lenses compared to that present in lenses in age matched controls (Harding and Dilley 1976). It was also suggested that aggregation was an artifact resulting from the extraction procedure (Harding 1972). Analogous studies have more recently been carried out on lenses in which the cataracts were separated according to the nature of the changes present in the lens. The results of those studies have suggested that nuclear brunescent, but not cortical cataract, is associated with formation of increasing amounts of water insoluble proteins (Truscott and Augusteyn 1977). Some researchers have attributed this increase to cross-linking of lens proteins induced by photo-oxidation. It has been further suggested that the reason these changes occur more readily in the lens nucleus than in the cortex is that the cortex has a higher concentration of protective anti-oxidants.

If Benedek's (1971) original hypothesis that the mechanism underlying senile cataract formation was aggregation and insolubilization of lens crystallins is correct, then one would expect to find an increase in protein cross-linking and aggregation in lenses that show light scattering. To the contrary, lenses with light scattering opacities in the cortex do not show an increase in high molecular weight proteins or crosslinks (Truscott and Augusteyn 1977; Anderson, Wright, and Spector 1979). Instead, reports in the literature suggest that there may be an increase in protein aggregates in lenses with substantial nuclear brunescence. This is rather a surprise because nuclear brunescence per se is not a light scattering change in the lens; rather, the lens turns a yellow or a brown color that absorbs rather than scatters light, reducing its intensity on the retina (Lerman and Borkman 1976). The effect of brunescence is, therefore, analogous to placing a transparent filter before the eye.

Unfortunately, only man and a very few other diurnal species have been found to develop lens browning so that it has been difficult to find an animal model in which to test the hypothesis that browning can be induced in vivo by exposure to UV radiation. Therefore, those studies that have examined the effects of chronic UV exposure in experimental animals have not demonstrated that UV induces lens browning. They have, however, consistently demonstrated that UV exposure induces light-scattering cortical opacities (Bachem 1956; Zigman and Vaughan 1974; Pitts, Hacker, and Parr 1977). Therefore, though we may lack a suitable model for nuclear brunescence, there are animal models that can be used to study the induction of cortical opacities by UV radiation. These models mimic the common and considerably more visually disturbing senile cortical cataracts in man.

In those studies on UV-induced lens changes in which histology of the lenses were examined, epithelial cell changes were a uniform finding (Zigman and Vaughan 1974; Pitts, Hacker, and Parr 1977). In their study of chronically exposed mice, Zigman and Vaughan (1974) noted the similarity of the lens changes to changes induced by X-irradiation. For example, lens cells appeared to have lost their capacity to differentiate and were found to have migrated to the posterior pole, a situation that has also been found to be associated with senile cortical cataracts (Streeten and Eshaghian 1978).

While biochemical approaches have generally concentrated on nuclear cataracts with respect to UV-mediated effects, there has been recent interest in photosensitized reactions in the aqueous humor as sources of damage to lens membranes and, hence, as a possible initiator of cortical (osmotic) cataracts. Varma, Kumar, and Richards (1979) have been investigating the possibility that a photochemical conversion of molecular oxygen present in the aqueous humor and lens into superoxide and subsequent derivatization of superoxide to other potent oxidants such as hydrogen peroxide, hydroxyl radical and singlet oxygen may be involved in initiating a cascade of toxic biochemical reactions leading to the formation of cataracts. Thus, according to this hypothesis, cataractogenic influence of light is mediated by a photochemical generation of superoxide from the ambient oxygen. Spin restriction offered by the molecular oxygen makes the formation of superoxide a necessary event in most oxidation reactions involving oxygen. It is

commonly understood that, like many other free radicals, superoxide and its derivatives, if allowed to remain unscavenged for any length of time in a biological mileu, will initiate many nonspecific and deleterious reactions such as an upsetting of the normal redox chain, oxidation of vitally important protein and nonprotein - SH, peroxidation of membrane and cytosolar lipids, and polymerization and depolymerization of macromolecules such as proteins and hyaluronic acids. The cataractogenic influence of these oxidants is likely to be modulated by certain endogenous protective mechanisms. Superoxide dismutase (SOD), catalase and perioxidase constitute the first line of defense against the toxic effects of those oxygen species. Jernigan et al. (1981) showed that singlet oxygen generated in the medium had similar effects. Varma, Beachy, and Richards (1982) have demonstrated lipid peroxidation in cultured lenses irradiated with fluorescent light. While such studies are of great interest in terms of the effects of these oxidants on the lens, at the present time there is no real evidence that they are produced insignificant quantities in the ocular humors by light mediated processes.

Considerable effort has been directed at elucidating mechanisms by which UV radiation (alone or in combination with exogeneous photosensitizers) might induce cataract formation. As previously mentioned, it has been shown that UV radiation can induce cross-linking and aggregation of proteins. As has also been mentioned, protein aggregation may be associated with nuclear changes but it is not a characteristic associated with cortical cataracts. It may therefore be more relevant to consider the possibility that UV damage to lens proteins induces local perturbations in macromolecular structure and function. This might in turn result in localized changes in lens structure of function. For example, transport systems and ATPase (Varma, Kumar, and Richards 1979) in the lens have been shown to be inhibited by UV radiation. If the lens transport systems are damaged by UV radiation, the osmotic balance will be disrupted which, in turn, would produce major changes in lens morphology.

The UV effects on transport systems may occur due to direct UV absorption by and damage to the related enzymes; however, other mechanisms may also contributed to this inhibition. For example, the activity of transport enzymes could be dramatically altered secondary to UV induced disruption of lipids in lens membranes; and, lipid peroxidation has been proposed as a mechanisms of cataract formation (Goosey, Allison, and Garcia 1983). The resulting alterations in lipid structure could of themselves make the membranes leaky or, as just mentioned, such alterations could disrupt the structure and function of membrane proteins. If one can extrapolate from the large amount of research that has been done on other cell systems, then it may be predicted that fairly long wavelengths of UV will similarly be able to produce membrane damaging effects in the lens (Moss and Smith 1981; Imbrie and Murphy 1982; Sprott, Martin, and Schneider 1976).

If we are to determine the role of lipid peroxidation or inactivation of specific enzyme systems in the development of UV cataracts, it is critically important that lenses be sampled several intervals prior to the onset and during the development of lens opacities. Once the lens is fully opacified,

retrospectively, it is difficult to state with any assurance that some observed biochemical change was the specific change that initiated the cataract.

For example, if an ATPase were found to be inactivated after a particular UV-induced cataract was fully developed, one might conclude that this inactivation had caused cell swelling, membrane lysis, and an "osmotic cataract." But it is also possible that the primary cause of the cataract was direct membrane damage or inhibition of protein synthesis and, that, only secondarily was ATPase activity involved. Therefore, to show that ATPase inactivation is of any real significance in the etiology of the cataract, it is necessary to demonstrate that ATPase inactivation precedes development of the lens opacities. In the case of UV-induced cataracts, one also needs to demonstrate that those wavelengths that inactivate the ATPase are those same wavelengths that most readily induce cataract formation.

It may be misleading to examine effects of UV (with or without added photosensitizers) on lens macromolecules isolated in test tubes because UV effects in vitro may be very different from the effects of UV on the intact lens. For example, it is possible to cause tryptophan destruction in lens proteins irradiated at a concentration of 2.5 mg/ml in a test tube, but the same type of irradiation was not found to produce such effects at the concentration of protein in the lens (Dillon et al. 1982). Also, the action spectrum may differ considerably from the absorption spectrum (Turro and Lamola 1977). The action spectrum shifts markedly in situ in the case of the photosensitizer, psoralen. Psoralen, riboflavin, methylene blue and rose bengal have all been found to induce photo-oxidative changes in isolated lens proteins when these proteins are irradiated with UV. However, each of these photosensitizers requires the presence of oxygen, otherwise lens proteins are not damaged. The amount of oxygen in the lens is not high (Kwan, Ninikoski, and Hunt 1972; Barr and Roetman 1974). Thus, these findings must be extrapolated to the in vivo lens with considerable discretion and it remains to be determined just how much photodynamic damage (that is damage where oxygen is involved as an intermediate) can be produced in the lens in situ. This is a very important research question because any effect that is of significance as a mechanisms of cataract formation must be inducible in a lens that is still in the eye. For example, it might be interesting to use 254 nm radiation as a toll in vitro to demonstrate that the lens epithelium has the capacity to repair damaged DNA (Jose and Yielding 1977). However, 254 nm radiation does not penetrate the cornea, so it will not be hazard for the in vivo lens and it will not cause cataracts. Therefore, it is a far more significant finding that 300 nm radiation can induce DNA repair synthesis in the lens epithelium when the lens is exposed in situ through the intact cornea (Jose, Kock, and Respondek 1982; Brenner and Grabner 1982). This finding shows that 300nm UV radiation penetrates through the cornea and demonstrates that epithelial cell DNA is a target of its effects.

Therefore, if we are to establish mechanisms of UV damage in the lens, we must be able to show that a given damaging reaction can be produced by UV in the in situ lens. An effect that can be produced on lens macromolecules isolated in a test tube may be interesting and stimulate further research, but

it must not be taken as sufficient proof that such an effect can occur in vitro. This is especially true for those reactions that require oxygen.

One photosensitizer that may provide important clues as to mechanisms involved in cataract formation is the drug psoralen which in combination with UV radiation is used in treating psoriasis. Two different mechanisms have been proposed to underly the psoralen cataract. According to one hypothesis, the cataracts occur as a consequence of psoralen binding covalently to lens proteins which results in formation of additional damaging photosensitizers in the lens (Lerman, Megaw, and Willis 1980). The second hypothesis is that the cataracts occur as a consequence of psoralen induced damage to lens nucleic acids (Jose and Yielding 1979).

It has been found that intense exposure to UV radiation will cause psoralen to bind to isolated lens proteins, specifically the tryptophan moieites (Lerman, Megaw, and Willis 1980); however, the reaction requires oxygen (Megaw, Lee, and Lerman 1980). Therefore, it is important to determine whether such protein binding occurs in the intact lens. Although spectra have been presented that demonstrate that UV radiation induces psoralen binding in the in vivo lens, it is not clear that the binding observed represents protein specific binding or whether the binding is to some other lens macromolecule. Lens protein fractions have been extracted from psoralen-treated humans or laboratory animals and fluorescence spectra taken from these. These spectra were interpreted to demonstrate photobinding of psoralen to lens proteins; however, no effort was made in these studies to extract nucleic acids from these "protein" preparations. It is very possible that a significant amount of the binding that was observed is accounted for by psoralen binding to RNA or DNA present in the degenerating nuclei of the lens cortex. The possibility that the spectra represented binding to nucleic acids in the differentiating fibers was discounted by the researchers because there is such an overwhelming concentration of protein compared to DNA in lens fibers (Lerman, Megaw, and Gardner 1982a). This is a reasonable assumption if a significant percentage of the psoralen were actually bound to the protein. However, the argument would be nullified if the majority of the psoralen were found to be bound to the small amount of nucleic acids in the fibers. Autoradiograms showing psoralen binding in the lens suggest that the latter is in fact the case (Lerman et al, 1981). Examination of those autoradiograms shows pronounced binding of psoralen in the epithelium plus discrete binding to the degenerating fiber nuclei. In comparison, binding to any other portions of the fibers is not distinguishable from background grains.

Autoradiography has also demonstrated psoralen binding in the nucleated layers of the retina and cornea but no binding to the non-nucleated layers of these tissues (Lerman et al, 1981). It is rather difficult to reconcile the failure of psoralen to bind to the non-nucleated regions of the retina and cornea with the suggestion that it binds significantly to cortical fiber proteins. A trivial explanation for this difference is that lens proteins are somehow "different" from corneal and retinal proteins, making the former susceptible to psoralen binding and the latter not. This argument is rather difficult to reconcile with the requirement of oxygen for photo-induction of

psoralen linking to proteins, a requirement that would lead one to expect greater binding to the proteins in the highly oxygenated retina and cornea compared to the relatively anoxic lens.

If lens proteins are the major target of psoralen's action on the lens, then one would expect that the cortical fibers would be disrupted as a primary event. In fact, however, histologic observations have shown that the first target of psoralen's effects are the epithelial cells (Jose, Kock, and Respondek 1982). Only after very considerable damage is observable in the epithelium is any damage detectable in the lens cortex which is consistent with DNA as a primary target of psoralen damage. That psoralen plus UVB can induce DNA damage in the lens is shown by the finding that the combination induces DNA repair in lens epithelial cells (Jose and Yielding 1979). It will be interesting to resolve the question as to the relative roles of specific macromolecular targets in development of psoralen-induced cataracts.

As with psoralen, most other studies of UV effects on the lens have directed their major emphasis toward examinations of changes in lens proteins. But, as with the psoralen study, there are compelling reasons to consider other macromolecules a targets of UV damage. Lens lipids have been mentioned previously and interest in lipids peroxidizing effects is of current interest in many laboratories. However, interest in the effect of UV and photosensitizers on lens DNA is currently very limited. The role of DNA damage in development of lens cortical opacities is generally overlooked or rationalized away. Some have assumed that there is so little DNA in the lens that even if it were a target, any effects on it would be overwhelmed by effects on lens proteins. There are, however, good reasons to consider the lens to be similar to other biological systems in which DNA is a significant target of UV damage.

If one considers the action spectrum that has been determined in those studies in which specific wavelengths of UV were isolated to examine their cataractogenic potential, it is noted that these lie in the range of 290 to 320 nm (Bachem 1956; Pitts, Hacker, and Parr 1977). These are the same wavelengths that most readily produce thymidine dimers in skin (Pathak, Kramer, and Guengerich 1972). Shorter wavelengths are blocked from deeper layers of the skin by the stratum corneum, quite analogous to corneal UV absorption which protects the lens. These are the same wavelengths which most readily induce malignant transformation and skin cancer (Freeman 1975). UV radiation up to 320 nm also can induce DNA repair synthesis in cultured fibroblasts (Ichihashi and Ramsey, 1976). Longer wavelengths of UV do not induce skin cancer, although they may, if applied very intensely, induce strand breaks and other damage in DNA (Webb and Peak 1981, Harm 1978). Endogenous photosensitizers are likely to be involved since DNA itself does not absorb appreciably at that wavelength. Long wave UV may potentiate the action of short wave UV in cancer induction and it can also be noted that 364 nm has been found to inactivate DNA repair mechanisms (Tyrell 1976).

With all the interest in tryptophan as an endogenous photosensitizer of protein damage in the lens, it is interesting to point out parenthetically that photo-oxidation of tryptophan has also been found to induce DNA damage in

lower organisms as a consequence of production of H202 (Ananthaswamy and Eisenstark, 1976). The possibility that those free radical species that have been found to be generated from UV excitation of tryptophan in the lens may also act on lens DNA as a target should not be overlooked. Furthermore, tryptophan photoproducts have also been found to bind to DNA (Glazer, Rincon, and Eisenstark 1976) and to inhibit strand rejoining in damaged DNA (Yoakum et al. 1974).

Another reason to consider that DNA is a target of UV damage in the lens is the finding that 300 nm radiation can induce DNA repair synthesis in the lens epithelium, even when the irradiation is applied through the intact cornea (Jose, Kock, and Respondek 1982; Brenner and Grabner 1982) and DNA repair synthesis is an indication that DNA was damaged. This point is often misunderstood, repair is not a perfect process and it should not be assumed that lens DNA will be absolutely protected. Mutational and lethal events can occur any time that DNA is damaged and repair is undertaken; thus, anything that can induce DNA repair must be recognized for its primary damaging effects. Errors induced in the genome of lens cells may manifest themselves as mutational events in the target epithelial cell. Such mutational events would be cumulative over an individual's lifetime. Those cells that have undergone mutations might loose their capacity to differentiate into normal lens fibers and, for example, pile up at the posterior pole. The later is seen in the senile cataract (Streeten and Eshaghian 1978) as well as in animals exposed to near UV radiation (Zigman and Vaughan 1974). The cells may also not carry out their normal functions such as maintaining less osmolarity. Then we might develop what appeared to be an "osmotic" cataract, when in fact, the underlying mechanism was genetic.

POTENTIAL CHANGES IN SENILE CATARACT PREVALENCE FOR CHANGES IN UV-B

Epidemiological studies have identified a correlation between the prevalence of various types of cataracts in humans and the flux of sunlight or ultraviolet radiation reaching the earth's surface (Hiller, Giacometti and Yuen 1977, Zigman, Datiler, and Torczynski 1979; Taylor 1980, Hollows and Moran 1981). Hiller, Sperduto and Ederer (1983) developed a multivariate logistic risk function that describes the correlation found between the prevalence of senile cataracts and the flux of UV-B and other risk factors. The results of this study may indicate the magnitude of change in the prevalence of senile cataracts that could be associated with changes in UV-B flux due to ozone depletion.

Hiller, Sperduto and Ederer based their analysis on the 1971-1972 National Health and Nutrition Examination Survey (HANES) general medical and opthalmological examinations of over 10,000 persons, ages 1-74 years. Using the HANES data, persons were assigned to one of two mutually exclusive groups: (1) cataract or aphakia (either eye), and (2) neither cataract nor aphakia (either eye). Cataract was defined as "senile lens changes (cortical, nuclear, posterior-subcapsular, or other) consistent with best corrected visual acuity of 20/30 (6/9) or worse" (Hiller, Sperduto and Ederer (1983) p. 240). This definition differs from the HANES survey, which used a visual

acuity of 20/25 (6/7.5) or worse. Aphakia was diagnosed when "the lens had been surgically removed and there was no history of congenital, traumatic or secondary cataract" (Hiller, Sperduto and Ederer (1983) p. 240). The study by Hiller, Sperduto and Ederer included HANES data on a total of 2,225 persons between the ages of 45 and 74 years who had resided at least one half of their life in the state where the HANES examination took place. Of these 2,225 people, 413 (18.6 percent) were placed in the cataract or aphakia outcome category.

The UV-B data were developed by NOAA for the 35 HANES locations used in the study based on a statistical analysis of UV-B data collected at 10 locations using Robertson-Berger meters (RB-meters). The statistical analysis incorporates season, latitude, elevation, weather (clouds), and haze. Subsequent validation of the estimates at six locations indicated that the differences between the estimated and observed mean daily flux average about seven percent.

These data on UV-B and outcome (i.e., cataract) were used in conjunction with demographic and medical history data to estimate the following multivariate logistic risk function:

$$P = \frac{1}{1 + \exp(-a - b_i X_i - \ldots - b_k X_k)},$$

where P is the probability (or risk) of having a cataract, and X_i are risk factors. In addition to UV-B, the following risk factors were analyzed: age; race; sex; education; diabetis; systolic blood pressure; and residence (urban, rural).

Exhibit 10-12 displays the standardized regression coefficients estimated for each of the risk factors. Positive coefficients indicate factors that are correlated with increased risk, negative coefficients indicate factors that are correlated with decreased risk. The coefficients presented in the exhibit are "standardized," meaning that they represent the expected change in the logit of P (equal to ln (P/1-P)) for a one standard deviation change in the risk factor. Standardization of the coefficients allows the relative importance of the risk factors to be identified by the relative size of the standardized coefficients.

As shown in Exhibit 10-12, three risk functions were estimated: (1) univariate (outcomes as a function of the risk factor); (2) bivariate (outcome as a function of the risk factor and age); and (3) outcome as a function of all the risk factors simultaneously. For all three formulations, UV-B is statistically significant, and positively correlated with the increased risk of being in the cataract outcome category.

Using the multivariate risk function coefficients, and the mean values for all the risk factors other than UV-B, the change in the prevalence of cataract for each 1.0 percent change in UV-B is estimated to be approximately 0.5 percent. This relationship holds for changes in UV-B as large as minus 20

EXHIBIT 10-12

Standardized Regression Coefficients for Cataract

Risk Factor [a]	Mean [b]	Univariate [c]	Bivariate [d]	Multivariate [e]
Age	61.97	1.22[f]	-	1.20[f]
Race	1.22	0.18[f]	0.20[f]	0.13[g]
Sex	1.52	0.07	0.08	0.08
Education	2.87	-0.43[f]	-0.25[f]	-0.14[g]
Diabetes	0.08	0.25[f]	0.23[f]	0.21[f]
Systolic Blood Pressure	146.3	0.33[f]	0.15[g]	0.08
UV-B	3.59	0.19[f]	0.20[f]	0.13[g]
Residence	1.37	0.18[f]	0.21[f]	0.19[f]

a/ Values for categorial risk factors: race: 1 = white, 2 = black; sex: 1 = male, 2 = female; education: 1 = ≤5 grades; 2 = 5-8 grades; 3 = 9-11 grades; 4 = 12 grades; 5 = college; diabetes: 0= absent, 1 = present; residence: 1 = urban; 2 = rural.

b/ Mean value for the risk factor in the 2,225 persons in the study.

c/ Each risk factor analyzed separately.

d/ Each risk factor analyzed with age only.

e/ All risk factors analyzed simultaneously.

f/ p(two sided) ≤0.005.

g/ p(two sided) ≤0.05.

Source: Hiller, R., R. Sperduto and F. Ederer (1983), "Epidemiologic Associations with Cataract in the 1971-1972 National Health and Nutrition Examination Survey," Americal Journal of Epidemiology, Vol. 118, No. 2, pp. 239-249.

percent to plus 30 percent. Outside of this range, reductions in UV-B are associated with less of a reduction in cataract prevalence, and increases in UV-B are associated with larger increases.

Of note is that this estimated relationship between UV-B and cataract prevalence varies with age; UV-B has a larger effect on prevalence (on a percentage basis) among younger individuals. Exhibit 10-13 displays the percent increase in cataract prevalence expected due to increases in UV-B, for peoples of different ages. As shown in the exhibit, the percentage increase in prevalence due to changes in UV-B are estimated to be larger for 50 year olds than for 70 year olds.

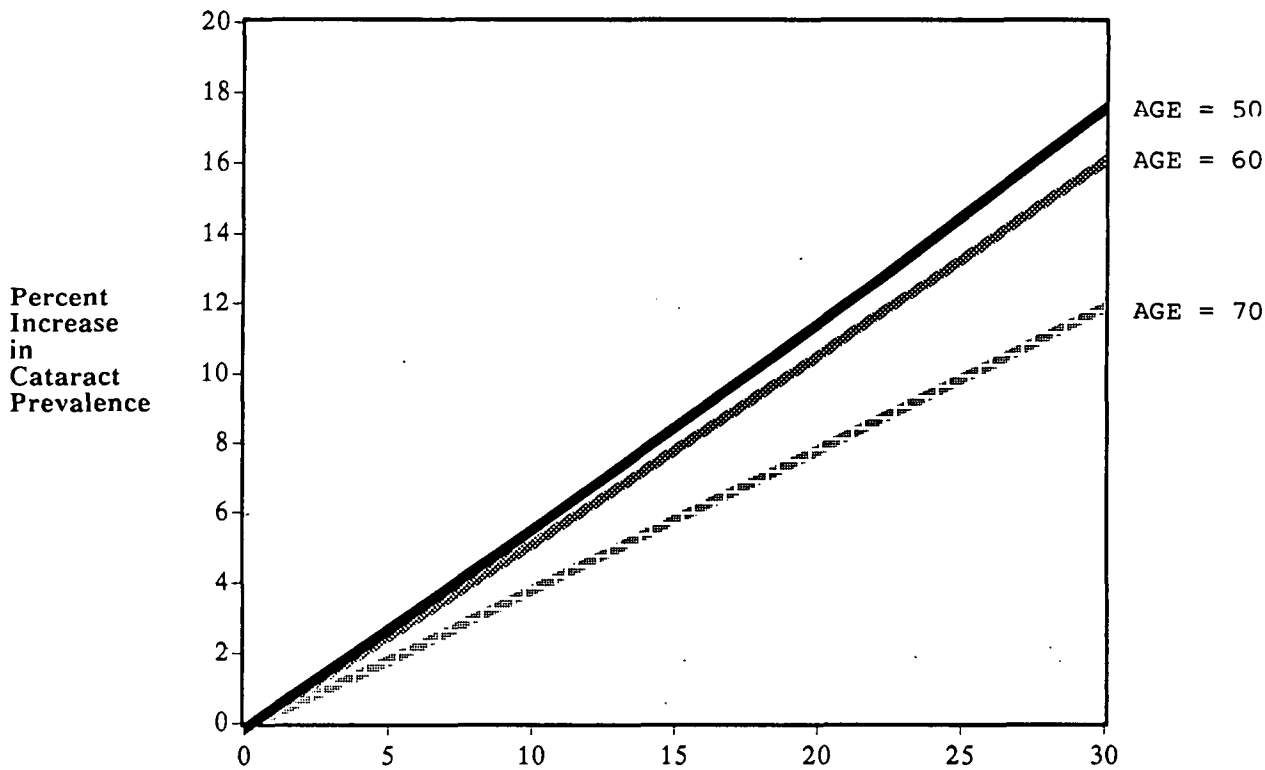
Although the effect of UV-B on prevalence is estimated to be larger at younger ages (on a percentage basis) using the multivariate risk function, the prevalence of senile cataracts is known to increase substantially with age. Leske and Sperduto (1983) report the prevalence of senile cataracts in both sexes found in the Framingham Eye Study to be as follows: 52 to 64 years old -- 4.5 percent; 65 to 74 years old -- 18.0 percent; 75 to 85 years old -- 45.9 percent. These prevalence estimates use the same definition of cataracts as used by Hiller, Sperduto, and Ederer. (Larger prevalence rates are reported by Leske and Sperduto based on HANES data. These estimates, however, use a definition of cataract that includes a decrease in vision to 20/25 (6/7.5), instead of the 20/30 (6/9) used in both the Framingham study and the Hiller, Sperduto, and Ederer risk study.) Because cataracts are more prevalent in older individuals, increases in the actual number of cases of cataracts would likely be larger for older individuals, even though the percentage increase in risk has been estimated to be larger for younger individuals.

Using the prevalence data cited above, the prevalence of cataracts in the U.S. population is on the order of 9.3 million. Using the multivariate risk function (with all values set to their means except age and UV-B) the hypothetical increased prevalence for 1985 that would have occurred had the entire population experienced 1.0 percent ozone depletion can be estimated. For a 1.0 percent depletion, the annual UV-B flux measured on the RB-meter has been estimated to increase by approximately 0.83 percent (see Serafino and Frederick (in press) and Chapter 17 for a discussion of the relationship between ozone depletion and UV flux). A 0.83 percent increase in UV-B is associated with increases in cataract prevalence that varies by age. Across all the ages, prevalence would be expected to be about 0.26 percent higher, or about 24,000 cases, had ozone been depleted by 1.0 percent.

Of note is that the RB-meter measure of UV radiation may not be the appropriate action spectrum to use to evaluate the potential biological effects of increased UV-B such as cataract. For example, the DNA action spectrum may be preferred. Even though the RB-meter and DNA action spectra are highly correlated in the range of current observations, because the DNA action spectrum is more heavily weighted toward shorter wavelengths, it increases more rapidly with decreases in ozone levels; a 1.0 percent depletion would lead to approximately a 2.0 percent increase in UV-B. Using the 2.0 percent increase in UV-B would yield about a 0.6 percent increase in prevalence, or about 57,000 cases.

EXHIBIT 10-13

Estimated Relationship Between Risk of Cataract and UV-B Flux



Increased UV-B flux (measured with an RB-meter) is associated with increased prevalence of cataract. The percent change in prevalence varies by age.

Source: Developed from data presented in Hiller R., R. Sperduto, and F. Ederer (1983), "Epidemiologic Associations with Cataract in the 1971-1972 National Health and Nutrition Examination Survey," American Journal of Epidemiology, Vol. 118, No. 2, pp. 239-249.

There are various important limitations in the use of these estimates and data. The correlation between UV-B and cataracts reported by Hiller, Sperduto and Ederer does not prove a causal connection -- other (unknown) factors could be playing a role. These (unknown) factors would have to be correlated with UV-B flux. Also, the study does not have estimates of individual lifetime UV-B exposure, thereby limiting the strength of the evidence for the association between UV-B exposure and cataracts. Additionally, the sample population analyzed may not be representative of the entire U.S. population. Finally, the outcome category used in the study does not differentiate between different types of cataracts, some of which may be more strongly related to UV-B exposure than others.

Confidence in the estimates developed here are strengthened by several considerations. The correlation between UV-B flux and sunlight flux is high, and a correlation between sunlight and cataracts has also been found in Australia (Taylor 1980) and in China (Mao and Hu 1982). An association between UV-B exposure and cataract has also been demonstrated in laboratory animals. Therefore, although considerable investigation remains to be performed, indications are that the association between UV-B and cataract is a reasonable basis for evaluating potential impacts due to increased UV-B flux associated with ozone depletion.

OTHER EYE DISORDERS[2]

Stable Retinal Disorders

Evidence has been adduced which provides a chain of evidence on which to postulate that optical radiation may contribute to human retinal disorders which are not progressive, that is, stable retinal problems. This chain starts with the knowledge that UV-B, UV-A and light are absorbed by various retinal tissues (Wolbarsht 1976; Warner 1982). Moreover, the amount of radiant energy incident on the retina is dependent on the transparency of the cornea and lens, and on pupil diameter. Next a large number of bioeffects of these wavelengths have been identified which are hazardous to the retina (Marshal 1970; Williams and Baker 1980). Further, empirical estimates have been made of the domain of time, intensity, and wavelength which can induce damage to the pigment epithelium, the photoreceptors and the inner retina. These estimates provide sufficient quantitative evidence to establish safety standards to protect people from the short-term retinal damage which could be induced by lasers and other intense sources of optical radiation (Sliney and Wolbarsht 1980; Pitts 1973). There is, moreover, sufficient evidence of additive and persistent changes in the monkey and human retina to hypothesize that some stable long-term visual impairments in humans may be related to optical radiation. There are reports that optical radiation produces retinal damage hours, days, months and years after irradiation. Although sparse, this evidence is instructive. For example, sungazing by humans has been reported

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[2]This section in boldface type is taken from Waxler, M. (in press).

to produce persistent, evolving and delayed alterations in the visual system. Cavonius, Elgin, and Robbins (1974) reported that aniseikonia may persist for years. Pigmentary patches and blind spots have been reported to evolve in a complicated fashion over many months after solar retinopathy (Lowenstein and Steel 1941). In addition, late complications resulting in reduced visual acuity have been described (McFaul 1969). Furthermore there are several reports in humans of prolonged, and even cumulative, impairments in dark adaptation following extended viewing by humans of skylight (Hecht et al. 1948; Clark, Johnson, and Dreher 1946) or bright blue, violet (Brindley 1953) and ultraviolet (Wolfe 1949) radiation. Also there is at least one report of persistent visual impairment resulting from the accidental exposure to ultraviolet radiation from a welding arc (Naidoff and Sliney 1974). Even more important is the description by Tso and Woodford (1983) of sub-RPE neovascularization and late impairment in fluid transport after several years of evolving changes in retinal pathology following excessive exposure to optical radiation.

Some of these effects are the result of injury to the retina which was ophthalmoscopically visible, some of these effects resulted from exposure to optical radiation which did not produce ophthalmoscopically visible damage. Most important are two demonstrations that the addition of subthreshold exposures separated by several days induces retinal damage (Greiss and Blankenstein 1981; Kuwabara and Okisake 1976). The data strongly suggest that retinal damage induced by optical radiation can cumulate over days. This is especially important since the concern about long-term visual health problems necessarily involves consideration of the intermittent nature of exposure to emissions from multiple sources over an extended period of time.

Retinal Degeneration

There are some reasons to suspect that retinal damage induced by ultraviolet radiation can be unstable, that is, produce progressive damage. The retina of monkeys can be damaged by ultraviolet radiation (Ham 1984; Sykes et al. 1981). This damage to the retinal pigment epithelium, to the photoreceptors and the neurons is dose dependent, photochemically mediated and cumulative over hours and days. Although very few long-term observations have been made on the primate retina following excessive exposure to optical radiation, there are theoretical reasons to suspect that a retina compromised by disease or age would be more vulnerable to any degenerative changes which could be induced by optical radiation (Young 1981). The combination of short-wavelength optical radiation, oxygen, chromophores, and photosensitizers in the retina is potent with possibilities for producing retinal degeneration, although the only direct evidence of the exacerbation of retinal degeneration by optical radiation has been obtained in rodents (LaVail and Battelle 1975). In addition, the higher retinal doses of UV received by aphakes and pseudophakes make these individuals especially vulnerable to cystoid macular edema (Kraff et al. 1985). Fluorescein leakage occurs following exposure of the retina of the aphakic monkey eye to UV-A radiation, but comparable studies using UV-B radiation have not been conducted. Therefore, retinal degeneration should be considered a risk of excessive exposure to ultraviolet radiation.

Very little research has been conducted on primates, on diurnal animal models of human retinal degenerations, or on retinas compromised by chemicals, age or other agents. The striking evidence of long-delayed and persistent changes in the monkey retina following optical radiation, coupled with some clinical evidence in humans, suggests that research on primates needs emphasis.

Aging Disorders

Deterioration of the visual system as individuals age is a fact (Owsley et al. 1986). The likelihood that optical radiation contributes to this deterioration is high for several reasons. First, the cellular pathology observed in older individuals is similar in appearance to radiation induced damage (Kuwabara 1978). Second, the ophthalmoscopic appearance of the aged retina is sometimes similar to that induced by optical radiation (Klein 1958). Third, some of the kinds of vision loss described in the aged are consistent with the losses which could be produced by optical radiation damage (Jaffe, de Monestario, and Podgor 1982; Harweth and Sperling 1975). Fourth, the accumulation of lipofushin materials in the RPE and other changes in the retina suggest that the aged retina has a lessened capability of repair (Feeney-Burns, Berman, and Rothman 1980).

Optical radiation may have quite different visual health consequences for eyes with and without ocular disease. Optical radiation may accelerate the deterioration of central vision in older individuals with central retinal disease (Hyman et al. 1983), whereas in those free of ocular disease optical radiation may impair paramacular and peripheral vision more than foveal vision. This interpretation is offered to reconcile the facts of visual loss outside the macular in disease-free aged eyes (Owsley et al. 1986) with Young's prediction that the central retina will exhibit the major effects of lifetime retinal exposure to optical radiation (Young 1981). This ad hoc explanation is not very satisfactory but it does indicate the need to temporize theory with data. Other interpretations also are possible, e.g., those individuals with central retinal disease may have had a different exposure history or some genetic vulnerability.

Given these facts and theoretical considerations, exposure of the aged retina to ultraviolet radiation in excess of amounts permitted by the phakic eye probably is hazardous. The retina of the aged individual is exposed to more radiation than phakics receive when the crystalline lens is extracted without placing a comparable filter in front of the retina, even when ordinary sunglasses are used.

Research is needed to determine the extent to which optical radiation is a factor in the deterioration of the vision of the elderly. In the meantime, we should take practical steps to protect our eyes from excessive optical radiation, and thus prolong our visual lifetime.

Development Disorders

While there are several reasons to suspect hazardous effects of optical radiation on visual development, almost no research has been conducted on the

developing primate retina. In fact, there is very little experimental exposure data on the developing retina of any species. Glass et al. (1985) have reported that the probability of retinopathy of prematurity (ROP) is highest in infants exposed to more light during their hospital stay. This evidence confirms a prediction by Wolbarsht et al. (1983) about ROP and light and is consistent with the evidence for the interaction of oxygen and light in retinal damage (Ruffolo et al. 1984). The action spectrum for this effect is unknown but UV-B and UV-A probably play a role. Furthermore, there are a large variety of sources and situations in which infants are exposed to optical radiation. Some of the same evidence which gave rise to suspicions about the role of optical radiation in retinal degeneration and visual aging is relevant to the potential hazards to visual development: short-wavelength radiant energy incident on the retina, short-wavelength chromophores in the retina, dose-dependence of retinal damage, cumulative effects, long duration effects, and delayed effects. In addition, there are special properties of the developing visual system which might decrease injury thresholds, such as more transparent ocular media and different densities of chromophores and screening pigments, or which might increase the vulnerability of infants, such as the long period of post-natal retinal development (especially foveal) and early dependence on non-foveal visual fields. Thus, the long periods required for many visual processes to mature suggests a window-of-vulnerability during which time optical radiation might alter later visual performance, especially if optical radiation alters the spatial and temporal summation properties of the neural retina.

Even though there is little experimental data on the effects of optical radiation on the developing retina, the photobiological and visual science evidence is sufficient to postulate that disorders of visual development are risks of optical radiation.

Retinal Problems

The only human data from which one could derive an estimate of the radiant exposure of UV which could damage the retina is Hecht et al. (1948) and Clark, Johnson, and Dreher (1946). These data showed that staring at skylight for several hours induced an abnormal retardation of dark adaptation. Since rhodopsin has an absorption band in the UVA (Kurzel, Wolbarsht, and Yananashi 1977), this deficit could have been due to the ambient outdoor UVA. If the ambient level of UVA at the cornea was about 9×10^{-2} Jcm^{-2}, as suggested by Ham and Mueller (1982), then the retinal radiant exposure might have been about 9×10^{-3} Jcm^{-2} in the Hecht and Clark studies. Experimental estimates of UV retinal damage in monkeys have been few. Schmidt and Zuclich (1980) found the threshold at 325 nm to be 10 Jcm^{-2}. Recent evidence in phakic rabbits (Pitts, Bergmanson, and Chu 1983) and rats (Rapp, Jose, and Pitts 1985) suggest that the radiant exposure necessary to damage the retina is even lower at 300 nm. The threshold for 300 nm damage to the monkey retina currently is approximately 0.6 Jcm^{-2}. Ham and Mueller (1982) found the aphakic monkey has thresholds of 5.0 Jcm^{-2} at 325 nm, 5.4 Jcm^{-2} at 350 and 8.1 Jcm^{-2} at 380 nm.

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Chapter 11

CHAPTER 11

RISKS TO CROPS AND TERRESTRIAL ECOSYSTEMS FROM ENHANCED UV-B RADIATION

SUMMARY

In making an assessment of the risk to crops and ecosystems of increased ultraviolet-B radiation (UV-B), it must be recognized that existing knowledge is in many ways deficient. The effects of enhanced levels of UV-B have been studied in only four of the ten major terrestrial plant ecosystems. Most of our knowledge is derived from studies focused upon agricultural crops and conducted at mid-latitudes, not in the tropics or at more poleward latitudes. Trees have not been subject to experimentation. Experimental protocols often have had flaws; too often, single year studies have been done, rather than long term ones and too much of the existing data comes from growth chambers, in which plants grow under unrealistic conditions, rather than from field studies. Therefore, the full extent of the potential impacts of enhanced levels of UV-B radiation on a global basis cannot be adequately assessed.

Despite these limitations, a broad range of experimental results demonstrated that, in nearly half of the plant species examined, UV-B radiation deleteriously affected crop yield and quality. Data exist that indicate that it may be reasonably anticipated that if UV-B increases, crop yield and quality will decline for at least some cutivars. Existing data also suggest that increased UV-B will alter the distribution and abundance of plants and potentially disrupt ecosystems. Unfortunately, a qualitative prediction of how these ecosystems would be altered cannot be determined from the current knowledge base.

FINDINGS

1. OF ALL CULTIVARS TESTED, APPROXIMATELY 70% WERE SENSITIVE TO UV-B; THE REMAINING 30% WERE GENERALLY NOT AFFECTED.

   1a. The large intraspecific variation in response to UV-B suggests that tolerant genotypes exist.

   1b. Selective breeding techniques for resistance to UV-B may be useful in reducing the impact of increased UV-B radiation if it does not product deleterious effects for other characteristics desired in plants. Presently, the genetic bases for these differences between sensitive and insensitive plants are not well understood.

2. THE EFFECTS OF UV-B RADIATION HAVE BEEN EXAMINED FOR ONLY FOUR OF THE TEN MAJOR TERRESTRIAL ECOSYSTEMS AND FOR ONLY A THIRD OF THE PLANT GROWTH FORMS.

   2a. Little or no data exist for trees, woody shrubs, vines, or lower vascular plants.

   2b. Because of a lack of data it is impossible to adequately address the question of the effects of UV-B changes on plants on a truly global basis, since one would have to assume that plant groups not studied respond in an analogous fashion to the herbaceous agricultural species which have been studied.

3. DESPITE THE LARGE UNCERTAINTIES THAT RESULT FROM IMPERFECT EXPERIMENTAL DESIGN OR DOSIMETRY, THE OVERRIDING TREND OF EXPERIMENTAL DATA SUGGESTS A POTENTIAL REDUCTION IN CROP YIELD DUE TO ENHANCED UV-B RADIATION.

   3a. Enhanced levels of UV-B simulating between 16 and 25% ozone depletions caused crop yield reductions of up to 25% in 3/4 of the soybean cultivars tested.

   3b. Unlike drought or other geographically isolated stresses, increases in UV-B due to changes in the ozone column, could effect all areas of the world simultaneously. Even small reductions in crop yield on a global basis could lead to considerable economic consequences. Therefore, increases in UV-B constitute a large, but difficult to quantify, risk to global agricultural productivity.

4. THE INTERACTION BETWEEN OTHER MICROCLIMATIC FACTORS AND UV-B ARE IMPORTANT IN DETERMINING CROP YIELD.

   4a. Soybean (CV Essex) yield could be accurately predicted when total UV-B dose, daily maximum temperature, and number of days of precipitation are included in a regression model.

5. CROP QUALITY MAY BE REDUCED BY INCREASES IN UV-B, BUT THE RESPONSE MAY NOT OCCUR IN A LINEAR, DOSE-DEPENDENT MANNER.

   5a. The lipid and protein content of soybeam was reduced up to 10%; however higher UV-B doses did not consistently result in the largest reductions.

6. THE EFFECTS OF UV-B ON FUNGAL OR VIRAL PATHOGENS VARY WITH PATHOGEN, PLANT SPECIES, AND CULTIVAR.

   6a. Reduced vigor in UV-sensitive plants could render the plants more susceptible to pest or disease damage and thus result in considerable reductions in crop yield.

   6b. Current evidence on the issue of pathogens is very limited.

7. CHANGES IN UV-B LEVELS MAY INDUCE SHIFTS IN BOTH INTER- AND INTRASPECIFIC COMPETITION.

   7a. If these shifts favor weeds over crops, enhanced UV-B radiation could have severe consequences, such as the increased economic cost of increased tilling and herbicide application.

   7b. Although competition exists in natural ecosystems, increases in UV-B could alter the results of the competition.

8. UV-B RADIATION INHIBITS AND STIMULATES FLOWERING, DEPENDING ON THE SPECIES AND GROWTH CONDITIONS.

   8a. The timing of flowering may also be influenced by UV-B radiation, and pollen may be susceptible to UV damage upon germination.

   8b. Reproductive structures enclosed within the ovary or another wall appear to be weel-protected from UV-B radiation.

9. INTERACTIONS BETWEEN UV-B RADIATION AND OTHER ENVIRONMENTAL FACTORS ARE IMPORTANT IN DETERMINING POTENTIAL UV-B EFFECTS ON PLANTS.

   9a. UV-B effects tend to be exaggerated under low light regimes and suppressed under conditions of limited nutrients or water.

   9b. Interactions with other environmental effects make extrapolation of data from growth chambers or greenhouses to field conditions difficult and often unreliable.

INTRODUCTION

This chapter examines the published and unpublished material currently available to assess the likely impact of projected increases in UV-B radiation upon global crop productivity and the distribution and abundance of plants in natural ecosystems. The limitations to this assessment are formidable because of the paucity of experimental data and the slow development of appropriate experimental technology. Therefore, actual risks may be far greater or somewhat less than current knowledge suggests.

ISSUES AND UNCERTAINTIES IN ASSESSING THE EFFECTS OF UV-B RADIATION ON PLANTS

Ideally, experiments should be designed to develop a data base which perfectly simulates future conditions for all plant species. These conditions should include all direct effects of enhanced UV-B in addition to all of the possible significant combinations with other effects (interactions). Such interactions should include, but not be limited to, the effects of increased atmospheric levels of carbon dioxides (CO2), drought, mineral deficiency, etc. A perfect simulation of all possible future environments would make it possible to accurately assess the potential impacts of global UV-B radiation increases with high precision. Unfortunately, such ideal circumstances do not exist. In reality, we must make assessments based upon imperfect experimental designs, which include only very selective and sometimes unrealistic growing conditions and the testing of only a few representative plant species. Therefore, the existing data base for this assessment will allow us to examine only some of the potential effects of UV-B changes and cannot be regarded as conclusive.

ISSUES CONCERNING UV DOSE AND CURRENT ACTION SPECTRA FOR UV-B IMPACT ASSESSMENT

Total global UV-B irradiance is dependent on a number of factors including solar angle, latitude and altitude, stratospheric ozone concentration, atmospheric turbidity, and cloud cover. The earth-sun distance and minor solar fluctuations also contribute to annual variations in irradiance (Caldwell 1971). Because of diurnal and seasonal variations in many of these factors, the spectral composition of solar radiation also varies substantially. On a daily basis, solar UV-B irradiance is sinusoidal, peaking at solar noon. Annually, UV-B irradiance is maximum during summer and minimum during winter. Experiments evaluating the effectiveness of UV-B radiation on plants typically do not account for such changes because of practical difficulties in monitoring and supplementing UV-B radiation. Generally, supplemental UV-B radiation is provided using filtered sunlamps as a squarewave function by using timers. This system provides a proportionately greater UV irradiance during morning and late afternoons and under cloudy skies than would be anticipated outdoors. Caldwell, Gold et al. (1983) have designed a modulated system to monitor ambient UV-B and provide the desired supplemental UV-B dose. This system provides a more realistic simulation of anticipated ozone depletion because it modulates lamp output in accordance

with actual levels of incoming solar UV radiation. Despite substantial expenses, it is highly recommended that such a system be utilized to improve field simulations and sensitivity of validation studies.

The source of UV-B radiation most commonly used in plant effects research is the fluorescent sunlamp which emits radiation principally in the UV-B region. This is a low-pressure mercury vapor lamp containing a phosphor that fluoresces primarily in the UV-B region, but includes some UV-C and UV-A radiation. Although the energy emitted principally comes from the fluorescing phosphor, some emission from mercury vapor is superimposed upon this, producing distinct lines in the spectrum. A weighting functions is absolutely essential to determine the biological effectiveness of the spectral energy emitted from various types of lamps. The biologically effective irradiance (I_{BE}) is given by the following relationship:

$$I_{BE} = I_{\lambda} \, E_{\lambda} \, d_{\lambda}$$

where I_{λ} is the lamp spectral irradiance and E_{λ} is the relative effectiveness of the energy to produce a response at wavelength λ. Thus, the biologically effective irradiance is the product of the action spectrum and the spectral irradiance at each wavelength.

Several UV action spectra have been developed that share the common feature of decreasing effectiveness as wavelength increases, but have considerable variation in the rate of this decrease. UV-B effectiveness in an isolated organelle can differ considerably from an intact plant because of the cellular shielding effects in the plant and inherent repair mechanisms. Caldwell (1971) has developed a generalized plant damage spectrum based upon the combined responses of a number of different plant species. Although this represents a step forward in understanding plant responses, an action spectrum developed on intact plants under polychromatic radiation would be preferable. Caldwell et al. (1986) have recently attempted to develop such an action spectrum and although there are experimental limitations, they were able to show that it is technologically possible.

Another essential reason for developing appropriate action spectra is the evaluation of radiation amplification factors (RAF), i.e., the relative increase in biologically effective UV-B radiation associated with a specific ozone reduction. RAF is a complex function taking initial ozone layer thickness, % ozone layer reduction, latitude, season, and biological weighting function into consideration (NAS 1979). The increase in solar UV-B radiation as a result of ozone reduction becomes appropriate only when the biological effectiveness of this radiation is known. Without an RAF, the absolute increase of total solar UV-B radiation resulting from even an appreciable ozone reduction is insignificant.

Since solar spectral irradiance increases by orders of magnitude with increasing wavelength in the UV-B region, the tails of the action spectra have a profound effect on the net RAF. The RAF values are much higher for those tails with steep slopes than those with shallow slopes. Thus, the computed

biological effectiveness of solar radiation could either be under- or overestimated if the action spectra are not realistic of true plant responses.

It is evident from the above discussion that a more realistic action spectrum is needed for a proper assessment of the possible consequences of ozone depletion.

ISSUES CONCERNING NATURAL PLANT ADAPTATIONS TO UV

Tremendous variability exists in species sensitivity to UV-B radiation (Krizek 1978; Van, Gerrard, and West 1976; Hashimoto and Tajima 1980; Tevini, Iwanzik, and Thoma 1981 and 1982; Tevini and Iwanzik 1982; Teramura 1983) (Exhibit 11-1). Some plants show sensitivity to ambient levels of UV-B radiation (Teramura, Biggs, and Kossuth 1980; Bogenrieder and Klein 1978; Sisson and Caldwell 1976), and others are apparently unaffected by rather massive UV enhancements (Becwar, Moore, and Burke 1982; Ambler, Rowland, and Maher 1978). Similarly, large differences have been reported among cultivars of a given species (Biggs, Kossuth, and Teramura 1981; Dumpert and Boscher 1982; Murali and Teramura 1986a,b; Murali, Teramura, and Randall 1986). Presently the mechanisms for these inherent differences have not been well documented.

Three main categories of natural UV protective mechanisms may be considered (Beggs, Schneider-Ziebert, and Wellmann 1986). The first includes repair mechanisms such as photoreactivation, a light-activated enzyme-mediated process whereby pyrimidine dimers produced by UV absorption are split (Rupert 1984). Indirect evidence (Beggs, Schneider-Ziebert, and Wellman 1986; Tanada and Hendricks 1953; Bridge and Klarman 1973) suggests that photoreactivation is a widespread phenomenon in plants. Excision repair, the replacement of deleterious photoproducts by new, correct DNA sequences, has also been documented in plant tissues (Howland, Hart, and Yette 1975, Soyfer 1983). Finally, quenching and free radical scavenging of oxygen singlets produced by photo-oxidation also alleviate some types of UV-induced damage.

The second category of protective mechanisms include those that tend to minimize the damaging effects of UV-B radiation. Probably the most important of these mechanisms in plants is growth delay. If cell division stops or is slowed upon UV irradiation, other repair mechanisms could help ameliorate the damage before it becomes lethal. Although many plants show a growth inhibition upon UV-B radiation exposure (Teramura 1983), it is not clear whether this inhibition is the direct effect of the damage or due to this protective mechanism.

The third category of protective mechanism involves those which effectively reduce the amount of UV radiation actually reaching sensitive plant targets. Structural attenuation by the cuticle and cell wall may play only a minor role since they offer little UV absorption (Caldwell, Robberecht, and Flint 1983; Steinmuller and Tevini 1985). The principal mechanism is probably the production of UV-absorbing pigments in outer tissue layers. Flavonoids and other related polyphenolic compounds, which occur in epidermal

EXHIBIT 11-1

A Summary of Studies Examining Cultivars Differences in UV-B Radiation Sensitivity

Crop	Number of Cultivars	Growth Condition a/	Conclusion b/	Basis of Comparison c/	Reference
Glycine max	19	G.C.	20% tolerant 60% intermediate 20% sensitive	d.w.	Biggs et al. 1981
	2	G.H.	Cultivar Altona more sensitive than Bragg	d.w.	Vu et al. 1978
	23	G.H.	8% tolerant 33% unaffected 59% sensitive	d.w.	Teramura and Murali 1986
	5	F	20% sensitive 80% unaffected	seed d.w.	
Phaseolus vulgaris	2	G.H. & G.C.	BBL 290 more sensitive than Astro	leaf resistance	Bennett (1981)
	3	G.H.	Maxidor sensitiv Saxa, Favorit tolerant		Dumpert and Boscher (1982)
Brassica oleracea	2	G.H. & G.C.	no difference	p.s. & d.w.	Van et al. (1976)
	2	G.C.	(?)		Garrard et al. (1976)
Cucumis sativus	5	G.H.	20% tolerant 80% sensitive	d.w.	Murali and Teramura (1986a)
	2	G.H.	Poinsett extremely sensitive, Ashley slightly sensitive	d.w.	Krizek (1978)
Tritium aestivum	4	G.H.	no difference (?)	d.w.	Dumpert and Boscher (1982)
	7	G.C.	no difference	d.w.	Biggs and Kossuth (1978)
	2	F	no difference	d.w.	Biggs et al. (1984)
Zea mays	4	G.C.	25% extremely sensitive 75% sensitive	d.w.	Biggs and Kossuth (1978)
Oryza sativa	5	G.C.	60% tolerant 40% sensitive	d.w.	Biggs and Kossuth (1978)
Hordeum vulgare	4	G.H.	25% tolerant 75% sensitive	d.w.	Dumpert and Boscher (1982)
	3	G.C.	no difference	d.w.	Biggs and Kossuth (1978)
Spinacia oleracea	2	G.H.	both sensitive	d.w.	Dumpert and Boscher (1982)

* * * DRAFT FINAL * * *

EXHIBIT 11-1

A Summary of Studies Examining Cultivars Differences in UV-B Radiation Sensitivity
(continued)

Crop	Number of Cultivars	Growth Condition a/	Conclusion b/	Basis of Comparison c/	Reference
Gossypium hirsutum	2	G.H.	no difference	d.w.	Ambler et al. (1978)
Pennisetum glaucum	2	G.C.	no difference	d.w.	Biggs and Kossuth (1978)
Cucurbita pepo	3	G.C.	no difference	d.w.	Biggs and Kossuth (1978)

a/ G.H. = greenhouse; G.C. = growth chamber; F = field

b/ If data presented, sensitive means UV-B radiation reduced d.w. by at least 10% over control plants. Tolerant indicates that UV-B resulted in less than 10% reduction in growth. In some cases, tolerant plants were even stimulated by UV-B radiation.

c/ d.w. = total plant dry wt; p.s. = net photosynthesis.

cells and have high UV absorption coefficients (Caldwell, Robberecht, and Flint 1983), are likely candidates. It has been shown that flavonoid concentrations in plant leaves substantially increase upon UV exposure (Wellman 1982; Murali and Teramura 1985a and 1986a; Robberecht and Caldwell 1978; Tevini, Iwanzik, and Teramura 1981 and 1983; Flint, Jordan, and Caldwell 1985), but it has not been established whether such an increase can completely attenuate the damaging effects of UV. Some studies suggest that despite large increases in flavonoid concentration, metabolic processes, such as photosynthesis, are still affected (Sisson 1981, Teramura et al. 1984, Mirecki and Teramura 1984). Also, total leaf flavonoid concentrations alone do not account for the range of responses observed in species sensitivity. For example, total leaf flavonoid levels found in UV-B irradiated soybeans were less than those found in cucumber, yet cucumber is much more sensitive to UV (Murali and Teramura 1986a,c). Therefore, species sensitivity to UV-B radiation is probably the product of a number of UV-protective mechanisms acting in concert within the plant. We currently need more specific information concerning plant adaptations to UV-B radiation before we can further refine our estimates of the ability of natural plant protective mechanisms to compensate for the projected increases in solar UV-B radiation.

ISSUES ASSOCIATED WITH THE EXTRAPOLATION OF DATA FROM CONTROLLED ENVIRONMENTS TO THE FIELD

Plant responses in growth chambers or greenhouses may neither quantitatively nor qualitatively resemble field responses because the environmental conditions are unlike those found in nature. Plants are more sensitive to a given UV dose when grown in growth chambers compared with field-grown plants (Caldwell 1981, Bennett 1981, Teramura 1982, Mirecki and Teramura 1984) because in artificial environments a single factor is generally manipulated, while all other factors are kept constant or are optimized for growth. Plants outdoors commonly experience simultaneous, multiple stresses including water or nutrient limitation. In addition to these differences in physical factors, artificial environments lack biotic factors such as the interactions between other plants, insects, diseases, etc.

However, enormous complexities are inherent in field studies. Daily changes in the environment are superimposed upon longer scale seasonal and annual changes. Both temporal and spatial variability often result in inconsistencies in plant responses between one year and the next (for examples see Biggs et al. 1984; Gold and Caldwell 1983; Teramura 1981b; Lydon, Teramura, and Summers 1986).

Therefore, one useful approach in understanding the effects of UV-B radiation on plants under more realistic conditions has been the study of the interactions between UV and other, commonly experienced plant stresses. These studies revealed that exposure to enhanced levels of UV radiation may affect the susceptibility of some plants to water stress, and alter the sensitivity to UV by inducing flavonoid biosynthesis (Teramura, Tevini, and Iwanzik 1983; Tevini, Thoma, and Iwanzik 1983; Teramura et al. 1984a; Teramura, Forseth, and Lydon 1984). The results of field experiments (Murali and Teramura 1986c)

revealed no additional deleterious effects of UV-B radiation when combined with water stress. It was hypothesized that changes in leaf anatomy, increased flavonoid production, and reduced growth induced by water stress masked the UV effects.

Plant productivity (accumulation of dry matter) became more sensitive to UV-B radiation as total mineral supply decreased (Bogenrieder and Doute 1982). Murali and Teramura (1985a, b) found that plant sensitivity to UV-B radiation decreased as the phosphorus level decreased. This suggests that the greatest impact of UV-B enhancement might appear in well-fertilized (agricultural) regions, rather than in areas of low fertility.

In most growth chambers, visible irradiances range from 10 to 40% of average midday irradiances. This is a cause for concern, since many of the deleterious effects of UV radiation may be ameliorated by exposures to longer wavelengths. Therefore, growth chamber experiments could overestimate the impact of UV effectiveness in the field. Despite the wide range of plant species and growth conditions, a clear trend emerges: plants grown in higher levels of visible radiation are less sensitive to UV-B radiation. A corollary to this conclusion is that plant sensitivity to UV-B radiation is strongly influenced by the level of visible radiation available during growth and development, and that shaded environments maximize this sensitivity.

Lydon, Teramura, and Summers (1986) and Teramura and Murali (1986) have specifically examined the differences in UV-B radiation response between field and greenhouse-grown soybean. Six soybean cultivars were grown in an unshaded greenhouse and in the field under a similar UV enhancement. Cultivar sensitivity was ranked based upon a combined plant response including changes in total plant dry weight, leaf area, and plant height. The relative ranking for UV-B sensitivity in the greenhouse was quite similar to that found in the field. The major difference was that UV-B radiation produced a substantially larger (2- to 10-fold) effect on greenhouse plants compared with field-grown plants. Also, in specific instances, different conclusions could be drawn from the individual data sets. For example, cultivar James was sensitive and York resistant to UV-B based upon greenhouse data. In the field, however, these cultivars demonstrated the opposite response. Therefore, if controlled environment-to-field extrapolations are necessary, they must be done with the utmost caution. At best, general trends may be implied, but specific or quantitative extrapolations do not yet seem plausible.

Despite a great range of experimental growth conditions and UV doses, most cultivars show large individual variation in response to UV-B radiation (Exhibit 11-1). Therefore, the potential for ameliorating the impacts of projected increases in solar UV radiation may be present in our current crop germplasm by selecting for UV tolerance. However, we have little understanding of the mechanisms responsible for these cultivar differences and the relationship of genetic factors that provide UV tolerance to other desirable plant characteristics. If tolerance to UV-B radiation were linked (in a genetic sense) to some undesirable characteristics, crop breeding may not ameliorate the impacts of UV-B damage.

UNCERTAINTIES IN OUR CURRENT KNOWLEDGE OF UV-B EFFECTS ON TERRESTRIAL ECOSYSTEMS AND PLANT GROWTH FORMS

Most of our knowledge of the biological effects of increasing solar UV radiation stems from research focused upon agricultural crops. Plants from only four of ten ecosystems, representing only about 27% of global net primary productivity (NPP) have been examined (Exhibit 11-2). In temperate forests and temperate grasslands, only very limited preliminary data are available. Also of 25 categories of major plant growth forms, the effectiveness of UV-B radiation has only been examined in eight (32%). Only very limited data exist for trees, vines, small woody shrubs, epiphytes, or lower vascular plants. Thus the experimental data base represents only 19 of the 314 plant families in the world (Cronquest 1981). Of those families tested, only seven include representative species in which harvestable yield was examined. Therefore, we have very little information from which to calculate the potential impacts of increasing levels of solar UV radiation on global terrestrial productivity of ecosystem dynamics.

Ecosystem composition and function are dependent upon the influence of numerous biotic and abiotic factors. Within populations, plants may possess protective mechanisms to UV-B, but the ability of higher plants to select for those mechanisms within the timeframe of expected changes in UV-B radiation is unclear as to the effect of that selection. Differential sensitivity could result in subtle shifts in species interactions such as competition. These shifts as well as changes in abiotic factors (soil nutrients, climate, etc.) could produce significant changes in ecosystem composition.

UNCERTAINTIES WITH THE ABILITY TO EXTRAPOLATE KNOWLEDGE TO HIGHER AMBIENT CO2 ENVIRONMENT AND OTHER ATMOSPHERIC POLLUTANTS

Global atmospheric carbon dioxide (CO2) concentration has been gradually increasing from 205 ppm some 20,000 years ago to approximately 340 ppm today (Keelings 1978, Neftel et al. 1982). It is anticipated that sometime between 2075 and 2100, the atmospheric CO2 concentration will reach 600 ppm (Gates 1983).

At present we have no direct experimental evidence on the effects of UV-B radiation under increased levels of atmospheric CO2. However, in general it is believed that increased CO2 increases photosynthesis and water use efficiency in most plants (which are reduced by UV-B radiation), especially in C3 plants. Exhibit 11-3 summarizes some of that research.

Recently, however, some confounding evidence has been found for perennials, in which high levels of CO2 led to a decrease in biomass production (Fried, 1986). Furthermore, most CO2 work has been done in growth chambers, which differs from realistic field conditions. The net effect of an interaction between UV-B radiation and increased level of CO2 cannot, at this time, be predicted. Whether the deleterious effects of UV-B will be proportionally higher or lower in a high CO2 world is uncertain. Similarly, the combined impacts of higher CO2 and UV-B on inter- and intraspecific competitive balance is impossible to ascertain. Other changes in the environment, such as those

EXHIBIT 11-2

Survey of UV Studies by Major Terrestrial Plant Ecosystems (after Whittaker 1975)

Ecosystem	Global NPP (10^9 ton/yr)	Total Area (10^6 km^2)	Included in UV Study [a]
Tropical forest	49.4	24.5	no
Temperate forest	14.9	12.0	yes
Savanna	13.5	15.0	no
Boreal forest	9.6	12.0	no
Agricultural	9.1	14.0	yes
Woodland and scrubland	6.0	8.5	no
Temperate grassland	5.4	9.0	yes
Swamp and marsh	4.0	2.0	no
Desert and semidesert	1.7	42.0	no
Tundra and alpine	1.1	8.0	yes

[a] Only studies examining some aspect of growth.

EXHIBIT 11-3

Summary of UV-B and CO2 Effects on Plants (Lemon 1983; Teramura 1983)

Plant Characteristic	Enhanced UV-B	Doubling of CO2
Photosynthesis	Decreases in many C3 and C4 plants	In C3 plants increases up to 100% but in C4 plant only a small increase
Leaf conductance	No effect in many plants	Decreases both in C3 and C4 plants
Water use efficiency	Decreases in most plants	Increases in both C3 and C4 plants
Dry matter production and yield	Decreases in many plants	In C3 plants almost doubles but in C4 plants only a small increase
Leaf area	Decreases in many plants	Increases more in C3 than C4 plants
Specific leaf weight	Increases in many plants	Increases
Crop maturity	No effect	Accelerated
Flowering	May inhibit or stimulate flowering in some plants	Flowers produced earlier
Interspecific differences	Species may vary in degree of response	Major differences occur between C3 and C4 plants
Intraspecific differences	Response varies among cultivars	Response may vary among cultivars
Drought stress	Plants become less sensitive to UV-B but not tolerant to drought	Plants become more drought tolerant

due to global warming and climatic changes, may also play important roles in determing effects, but cannot be assessed. Further uncertainty stems from the absence of knowledge on the interactions between increased UV-B levels and that of other pollutants in the lower atmosphere. Primarily anthropogenic in origin, the major pollutants include ozone, sulfur oxides, and nitrogen oxides. According to the National Crop Loss Assessment Network, farm crop losses in the United States in 1981 due to air pollution were between 1 and 2 billion dollars. Many studies have demonstrated that in combination, the deleterious effects of air pollutants were additive and in a few cases multiplicative (Reinert, Heagle, and Heck 1975). At present, no data are available on the interaction between UV-B radiation and various air pollutants.

RISKS TO CROP YIELD RESULTING FROM AN INCREASE IN SOLAR UV-B RADIATION

During the past 10 years, only nine field studies have examined the effects of UV-B radiation on crop yield (Exhibits 11-4 and 11-5). These nine studies included 22 crop species. Corn (Zea mays) was included in six of the nine studies, soybean (Glycine max) found in four, and tomato (Lycopersicon esculentum), bean (Phaseolus vulgaris), and potato (Solanum tuberosum) were each found in three studies. Yield was reduced by UV-B in about half of these species .

Ambler, Rowland, and Maher (1978) grew eight species of crops in a field at Beltsville, Maryland (30°N). Plants were grown under a lamp irradiation system maintained 1.6 m above them in a linear arrangement of unfiltered Westinghouse BZS-CLG and FS-40 sunlamps. A 2-dimensional gradient was established: one parallel to the lamps and another at right angles. Only broccoli showed a significant UV effect although the authors suggested that sorghum and corn also were affected. The use of unfiltered lamps, which emit both UV-B and UV-C, make interpretation of the results difficult, since UV-C radiation produces qualitatively and quantitatively different effects from UV-B (Nachtway 1975).

Bartholic, Halsey, and Garrard (1975) conducted field exclusion studies in Gainesville, Florida (29° 36'N) in which ambient UV-B was filtered through plastic films of Mylar Type S or polyethylene. Since plants were grown under panels covered with these films, only the direct beam component of ambient UV was removed (approximately one-half the total ambient UV present). Compared with uncovered plots, total yield decreased in beans and corn growing under both Mylar and polyethylene. Tomato matured significantly earlier under Mylar, resulting in an apparent decrease in fruit weight in plants growing in uncovered plots. There may have been a shading problem associated with the panel framework, or a difference may have been maintained in leaf temperatures or soil moisture as a direct result of the exclosure themselves.

Becwar, Moore, and Burke (1982) also conducted an exclusion study located at 3000 m in the Colorado Rocky Mountains (39° 11'N). Mylar, Aclar, and cellulose acetate filters were used. The only UV effect reported was a

EXHIBIT 11-4

Summary of Field Studies Examining the Effects of UV-B Radiation on Crop Yields.
Values represent percent changes from controls

	Ambler et al. (1978) (1)	Bartholic et al. (1975) (2)	Becwar et al. (1982) (3)	Biggs and Kossuth (1978) (4)	Biggs et al. (1984) (4)	Eisenstark et al. (1985) (4)	Esser (1980) (5)	Hart et al. (1975) (1)
Cucurbita maxima	0							
Cucurbita pepo				-14 to -90				
Phaseolus vulgaris	0	+12 to +15					+53 to -75	
Triticum aestivum					-5			
Zea mays	0	+29 to +39		0	0	-79 to -87		0
Spinacia oleracea							+11 to -56	
Sorghum bicolor	0							
Capsicum annum								-(?)
Glycine max	0							0
Cynodon dactylon								
Beta vulgaris	0							
Brassica oleracea var. capitata							+19 to -49	
Brassica oleracea var. botrytis	-24 to -45%							
Lycopersicon esculentum		-5 to -26		-11 to -39				0
Nicotiana tobaccum								0
Raphanus sativus			0					
Pennisetum glaucum								0
Solanum tuberosum			0	0			-2 to -41	
Brassica juncea				-9 to -43				
Vigna unguiculata				-18 to -38				
Oryza sativa					0			
Arachis hypogaea				0				0

(1) Unfiltered Westinghouse BZS-CLG and FS-40 sunlamps
(2) Ambient UV filtered with Mylar Type S or poleyethylene
(3) Ambient UV filtered with cellulose acetate, Aclar or Mylar
(4) Westinghouses FS-40 sunlamps filtered with cellulose acetate or Mylar
(5) Unfiltered Philips TL 40/12 sunlamps and lamps filtered with Schott WG 305 (2 and 3 mm) filters

EXHIBIT 11-5

Details of Field Study by Teramura (1981-1985) a/

CULTIVAR b/	YEAR: 1981		1982		1983 c/		1984 c/		1985	
	SIMULATED OZONE CHANGE (%): -16	-25	-16	-25	-16	-25	-16	-25	-16	-25
Bay	-10	NE d/	+6	-8						
Essex	-25 e/	NE	-12	-23	+39 e/	+6 e/	+14 e/	-7	+6	-20 e/
James	-14 e/	NE	-22	-25						
Williams	+22 e/	NE	+9	+14	+13 e/	-11 e/	+15 e/	+10 e/	+18 e/	+4 e
York	-12	NE	-25 e/	-8						
Forrest	NE	NE	-14	+27						

a/ Values reported are percent changes in seed yields for given simulated ozone changes (-16% and -25%) compared to controls (yields measured on a dry weight basis). Irradiance by either mylar (control) or cellulose acetate (UV-B supplemented) filtered FS-40.

b/ After initially screening 23 cultivars (16 of which were sensitive to UV-B; >10% yield reduction) in the greenhouse, these six were chosen for field experimentation. The six represent the full range of UV sensitivity found in the greenhouse; including very sensitive and very tolerant cultivars. Beginning in 1983 only Essex (very sensitive) and Williams (very tolerant) were planted in the field to increase the experimental sample size to 200. As shown in the table, yields for Essex were generally reduced at the higher UV level (ozone change of -25%), but were mixed (relative to controls) at the other UV level. Yields for Williams were generally enhanced by increased UV. Of note is that Essex is currently replacing other older cultivars (including Williams) and is becoming one of the most widely planned soybeans in the U.S. In a UV enriched environment, Essex will be deleteriously affected. Therefore superior cultivars being developed today by crop breeders may not be suitable for the future should the UV environment change.

c/ Drought year. Low yields for both controls and experimental (i.e., dosed) plants.

d/ NE = Not evaluated.

e/ Significantly different at p=0.05 level.

decrease in wheat height (between 8 and 19% depending upon plant age), with no corresponding change in total plant dry weight. A second study was conducted using filtered FS-40 sunlamps to supplement ambient levels of solar UV-B. No significant effect on crop yield was produced despite a calculated 52% UV enhancement compared with sea level irradiance simulating the dose plants received at sea level as control to make actual comparisons.

Biggs and Kossuth (1978) reported yields for seven crops grown in beds filled with a synthetic soil mix in Gainesville, Florida (29° 36'). UV-B radiation was supplemented with a linear arrangement of six FS-40 sunlamps at a 12° angle from horizontal, producing a gradient in UV-B irradiances. Control plants were grown without lamps above them and were adjacent to those receiving the highest UV dose. Although some "significant" UV effects were reported, no indication of statistical tests nor descriptive statistics were given. The greatest reductions in yield were often found in plants receiving the lowest UV irradiance and the largest UV effects were found in plants growing adjacent to ones showing little or no effect. Although each plant along the gradient received a uniquely different UV dose, plants and treatments were pooled for analysis. These manipulations undoubtedly added a great deal of experimental variability and resulted in difficulty in interpretation.

In another field experiment, Biggs et al. (1984) conducted a two-year evaluation of crop yield in rice, wheat, corn and soybean. Plants were again grown in beds filled with synthetic soils, but UV irradiation was supplied by FS-40 sunlamps filtered with either Mylar or 3, 5, or 10 ml cellulose acetate providing 0, 32, 23 or 16% UV enhancements. The only significant yield reduction reported was for wheat (5% reduction) and only for one of the two experimental years. The data for rice, corn and soybean were highly variable and therefore no statistical differences could be detected.

Eisenstark et al. (1985) grew corn in large pots for three growing seasons in Columbia, Missouri (38° 57'N). Two levels of UV radiation were supplied by cellulose acetate-filtered FS-40 sunlamps simulating a 7 and 21% ozone depletion. Controls included lamps filtered with Mylar and no overhead lamps. Plants were found to be particularly susceptible to UV-induced effects during tassel development (Eisenstark and Perrot 1985). Plants irradiated as seedlings produced total grain yield reductions of 23 and 32% respectively when compared with Mylar control plants. Even larger differences were found when these plants were compared with those grown without overhead lamps; yield was reduced by 80% in plants filtered by Mylar and 87% in those grown under cellulose acetate. The authors suggested that this large lamp effect could be due to additional UV-A emitted from the sunlamps, but this UV-A supplement was very small relative to UV-A present in midday solar radiation (Caldwell et al. 1986). The implications of this study for field crops (as opposed to potted plants) is doubtful, however, since the pots allowed root structures to reach higher temperatures than in fields.

Esser (1980) conducted field studies in Frankfurt, Federal Republic of Germany (50°N) on bean, cabbage, spinach, and potato. He used linearly arranged Philips TL 40/12 sunlamps suspended 3 m above the plants and produced four UV enhancements using a combination of reflectors, Schott WG305 filters, and unfiltered lamps. Significant yield reductions were found only under unfiltered lamps in the four crops tested. Yields for spinach, cabbage, and bean increased under filtered lamps.

Hart et al. (1975) grew plants under linearly arranged unfiltered FS-40 sunlamps at Beltsville, Maryland (39°N). The absence of experimental data and details on this study limit its usefulness. Also, the presence of UV-C radiation from these unfiltered lamps greatly decreases the validity of these data.

Teramura has grown soybeans for five seasons at Beltsville, Maryland (39°N) (Exhibit 11-5). During the first two years, six soybean cultivars were grown and two cultivars grown in subsequent years under filtered FS-40 sunlamps oriented perpendicularly to the soybean rows. This arrangement avoids the large variation in UV irradiance along the length of the bulb which is a major problem with linearly arranged (end-to-end) lamps. Control plants were grown under Mylar-filtered lamps. Field experiments simulated 0, 16, and 25% ozone depletions. Seed yield was consistently reduced at a simulated 25% ozone depletion in cultivar Essex. Yield reductions of up to 20% were reported in three of the five years. The "UV-tolerant" cultivar Williams was less affected but did show an 11% reduction in yield in 1983. Also in 1983, a substantially higher relative yield (6% increase) was found in cultivar Essex, which was opposite to that observed in the previous two years. One critical caveat which must be included with this observation is that 1983 was an extremely dry year. Actual seed weights of control plants during 1983 were only 20-30% of those harvested in 1981 and 1982. Therefore, it is questionable whether these 1983 data are representative of true (normal) field trends. This graphically illustrates the importance of multiyear studies and emphasizes the need to monitor other environmental variables in addition to UV radiation. If the results on Essex in drought years was repeated, it would indicate that higher UV radiation actually helped plants in severe drought years, reducing the crop loss by a few percent.

Soybean yield is influenced by other microclimatic factors as well as total UV-B dose. Yield appears to be strongly influenced by the number of days of precipitation and the number of days where the maximum temperature exceeds 35°C. In general, yield decreases as the number of hot days increases and increases as the number of days with precipitation approaches 25 in the growing season. However, further increases in the number of days with precipitation deceases yield. The relative importance of UV-B dose is a function of the cultivar and other prevailing microclimatic factors. For example, in Essex, total seed yield can be predicted, within 95% confidence intervals, by including total UV-B does, number of precipitation events, and the number of days where air temperature exceeds 35°C in a regression model.

Despite the broad range of experimental protocols and dosimetry used by various investigators, it appears that increases in solar UV radiation could potentially have a deleterious impact upon global crop yields. Even discounting the data from the three unfiltered lamp studies (Ambler, Rowland, and Maher 1978, Esser 1980, Hart et al. 1975), there are still more instances of significant reductions in yield than reports of no effect. While uncertainty exists, all of these studies were imperfect validations, suffering from one problem or another in experimental design or dosimetry, and only three studies (Biggs et al. 1984, Eisenstark et al. 1984, Teramura 1981-1985) include multiyear observations in which longer-scale environmental variability has been taken into consideration and only mid-latitude regions have been analyzed. It is reasonably likely that increases in solar UV will decrease global crop yields.

RISKS TO YIELD DUE TO A DECREASE IN QUALITY

Biggs and Kossuth (1978) reported that the number of abnormally shaped tomato fruit decreased 11 to 41% under enhanced levels of UV-B radiation in the field. They also reported a 6 to 23% reduction in the number of culls (rots, cracks, sunscald, etc.) in plants receiving a moderate UV-B dose, while those receiving a low dose produced a 10 to 34% increase in culls. Mean potato weight of grade A large potatoes increased 3 to 13% under enhanced levels of UV-B radiation, but not in any other grade categories. In neither case was a clear, linear relationship between UV dose and plant response demonstrated.

In a field study conducted in Beltsville, Maryland, Ambler, Rowland, and Maher (1978) reported that the sugar content in sugar beets significantly increased between 17-21% with increasing UV-B irradiance. However, they only observed these significant increases in plants directly under the lamp irradiation system and not in plants receiving a comparable UV-B dose at some distance away. Possibly some other uncontrolled factor (perhaps shading) was inadvertently introduced into the experiment.

Teramura (1982-1985) examined the effects of UV-B radiation on seed protein and lipid concentrations of soybean grown in the field at Beltsville, Maryland (Exhibit 11-6). Overall, the effects of UV radiation were relatively small but some were nonetheless significant. Seed protein concentrations declined under elevated levels of UV-B radiation in two of the four years (up to 5% reduction) in cultivar Essex. Seed lipid concentrations were reduced by 3 to 5% in cultivar Forrest in 1982, the only year in which this character was measured in Forrest. Williams showed a slight reduction in seed lipid content, with a maximum reduction of 10% reported in 1984.

The extent of UV-mediated alterations in yield quality cannot presently be estimated with any degree of confidence due to the paucity of experimental data at hand. Furthermore, conclusions drawn from a single growing season may be unreliable because of the annual variation in responses reported. Nonetheless the consistent reductions in yield quality in soybean suggest that the risk may be quite high in some crops.

EXHIBIT 11-6

Summary of changes in Yield Quality in Soybean Between the 1982 and 1985 Growing Seasons
(Teramura 1982-1985)

Crop	Yield Character	1982 % Change	Signifi-cance	1983 % Change	Signifi-cance	1984 % Change	Signifi-cance	1985 % Change	Signifi-cance
Glycine max									
cv Bay	% protein	0 to -5	ns						
	% lipid	-1.4 to +3	ns						
Essex	% protein	-3 to -5	P=0.05	+1 to -5	P=0.05	+1 to 0	ns	-0.7 to 0	ns
	% lipid	-2 to +.5	ns	+2 to -2	ns	-2 to 0	ns	0	ns
James	% protein	+.2 to +3	ns						
	% lipid	-1 to -2	ns						
Williams	% protein	0 to -.2	ns	+.4 to -.2	ns	+3 to +5	P=0.05	0.5 to -1	P=0.05
	% lipid	+5 to +8	P=0.05	-1 to +6	ns	-5 to -10	P=0.05	0	ns
Forrest	% protein	+.5 to +2	ns						
	% lipid	-3 to -5	P=0.05						
York	% protein	-2 to -3	ns						
	% lipid	0 to +3	ns						

* * * DRAFT FINAL * * *

RISKS TO YIELD DUE TO POSSIBLE INCREASES IN DISEASE OR PEST ATTACK

Esser (1980) reported a significant decrease in the number of aphids per plant but no significant difference in the spidermite population with increased UV-B radiation. Although the results suggest that UV-B radiation can have potentially beneficial effects on pest control, these conclusions must be judged with caution since the observation period was less than two weeks.

Growth chamber and in vitro studies of fungal pathogens showed that hyaline spores are more sensitive to UV-B radiation than pigmented spores (Carns, Grahm, and Ravitz 1978). Owens and Krizek (1980), however, found that survival of a pigmented spore (Cladosporium cucumerinum) was decreased with UV-B radiation due to a delay in germ tube emergence. Other studies (Esser 1980, Biggs et al. 1984) indicate that there is no clear relationship between spore coloration and UV-B radiation effectiveness.

Three leaf fungal pathogens showed a significant decrease in disease severity with UV-B radiation in a growth chamber study (Esser 1980), and a field study by Biggs et al. (1984) showed no significant difference in disease severity on leaves and seeds under increased UV-B radiation. Biggs et al. (1984) and Biggs (1985) found that in a leaf rust-sensitive cultivar of wheat, disease severity increased with UV-B radiation. In a rust-resistant cultivar, however, there were no differences in disease severity.

Semeniuk and Goth (1980) found significant UV-B mediated reductions in potato virus infection on Chenopodium quinoa. At irradiances over 86 m^{-2} $UV\text{-}B_{BE}$, no infection occurred. In this study, virus extract was exposed to UV-B radiation immediately upon its application over the leaf surface. Intuitively, viruses should be highly susceptible to UV-B radiation, since they consist of nucleic acids encased in proteins, both of which have high UV absorption properties. However, virus sensitivity must be viewed in light of screening offered by the host tissues.

One of the plant defense mechanisms that inhibits final development is the production of a class of chemicals known as phytoalexins (Bell 1981). Phytoalexin production in plants can be induced artificially through mechanical injury, high temperature, application of fungicides and antibiotics, and by UV radiation (Bridge and Klarman 1973, Reilly 1975). UV-B radiation can induce isoflavonoid phytoalexin synthesis (Bakker et al. 1983), but excess production can be toxic due to free radical formation (Beggs et al. 1984).

At present, it is difficult to forecast the consequences of enhanced UV-B radiation in terms of pest and disease damage. From the limited information available, it appears that in some cases UV-B radiation might decrease disease severity while in others it might aggrevate it. The effects vary with pathogen, plant species, and cultivar. Further studies are obviously needed to develop a better understanding of the consequences of increased levels of UV-B radiation on pests and plant diseases.

RISKS TO YIELD DUE TO COMPETITION WITH OTHER PLANTS

Many plants have been shown to exhibit a wide range of sensitivity to enhanced UV-B radiation (Teramura 1983), which could lead to changes in competitive ability within plant communities through differential UV-B resistance (Caldwell 1977). Solar UV exclusion studies (cited in Gold and Caldwell 1983) using natural competing species show large differences in response to present levels of UV radiation. This suggests that current levels of UV radiation may be partly responsible for interspecific competition among various native plant species.

Gold and Caldwell (1983) studied intraspecific competition in wheat (Triticum aestivum L.), wild oats (Avena fatua L.), and goat grass (Aegilops cylindrica Host) at various planting densities in Logan, Utah, under ambient and UV-B levels simulating a 16% ozone reduction. No significant differences in shoot biomass production were observed. Therefore, enhanced levels of UV-B radiation may be of little consequence in terms of intraspecific plant competition. Preliminary results (Caldwell personal communication; 1985, unpublished manuscript) however, indicate that interspecific competition may be affected. UV-B appeared to alter growth by the inhibition of internode elongation in wild oat but not in wheat, thus favoring wheat in competition for available light.

Fox and Caldwell (1978) and Gold and Caldwell (1983) examined the effects of UV-B radiation on the competitive interaction of field-grown competing pairs from three plant associations: agricultural crops and associated weeds, montane forage crops, and disturbed weedy associates. To measure competitive ability, relative crowding coefficients (RCC) were used. RCC based upon total above ground biomass indicate that there were significant shifts in the competitive balance of some agricultural crop and weed associations. For example, pigweed (Amaranthus) was more competitive under ambient UV conditions and alfalfa (Medicago) exhibited the competitive advantage under enhanced UV-B radiation. Under a 40% ozone reduction, wheat (Triticum) had a competitive advantage over wild oats (Avena) and goat grass (Aegilops) but no significant differences were observed with a 16% reduction. In the previous year, the competitive balance was found to be in favor of Avena. It was suggested that this difference was due to a late planting resulting in large water and temperature stresses during seedling development (Gold and Caldwell 1983). Similarly, the weedy Geum had the competitive advantage over Poa grass under ambient conditions, but the balance was shifted under enhanced UV-B radiation. Preliminary field data suggest that the competitive ability of wheat, based on seed biomass, increased relative to wild oats under enhanced UV-B radiation.

These results demonstrate that enhanced levels of UV-B radiation can alter the competitive interactions of some species. Total harvestable yield, as well as its quality, is altered by the presence of weeds (Bell and Nalewaja 1968, McWhorter and Patterson 1980). Since there is a large number of weeds typically associated with various crop plants, the impact of increasing levels of UV-B radiation upon agricultural systems could potentially have serious

consequences if weeds gain a competitive advantage over crops. Even very subtle differences in sensitivity could result in large changes in species composition over time and possibly affect ecosystem function. At this time, however, it is impossible to determine the extent of the risk.

RISK TO YIELD DUE TO CHANGES IN POLLINATION AND FLOWERING

In plants in which pollination and subsequent fertilization take place during the day with flowers fully open, reproductive tissues would seemingly receive an appreciable UV-B dose. However, ovules enclosed in the ovary are well-protected against UV-B radiation. Flint and Caldwell (1983) have shown that the anther wall filters out over 98% of the incident UV-B radiation in six plant species. Therefore, before another dehiscence, pollen is also well-protected.

Southworth (1969) found UV absorbing compounds with a maximum absorbance in the UV-B range in the wall of pollen . Additional studies on the UV absorption profiles of stigmatic surfaces and exudates of many species show one or more peaks in the UV-B region (Martin 1970, Martin and Brewbaker 1971). Therefore, under natural conditions, the effectiveness of UV-B may be minimal because of UV-absorbing pigments in the anther and pollen walls and in stigmatic surface exudate.

Results of numerous studies during the early part of this century on the effects of solar UV radiation using window glass filters indicated an inhibition of flowering by solar UV radiation (see reviews by Popp and Brown 1936, Caldwell 1971). However, these experiments were generally executed with insufficient sample sizes and failed to isolate UV irradiation as the single contributing factor causing the difference in flowering. For instance, Caldwell (1968) has shown that leaf temperatures are increased sufficiently under window glass to alter the induction of flowering (Zeevaart 1976).

Results of Kasperbauer and Loomis (1965) studying Melilotus and Caldwell (1968) with Trifolium dasyphyllum show an increase in flowering with the exclusion of solar UV radiation. A growth chamber study by Klein, Edsall, and Gentile (1965) incorporating primarily UV-A radiation also shows an increase in the number of flowers produced in marigold by the exclusion of UV radiation. Similarly, greenhouse trials on beans and peas also show an inhibition of flowering and decrease in flower number due to UV-B radiation (Biggs and Basiouny 1975). However, field studies using unfiltered sunlamps (which emit both UV-B and UV-C) produced no significant effects on flower, number, or date of flowering in marigold and tomatoes, tasseling in maize, or heading in sorghum (Hart et al. 1975). Biggs and Kossuth (1978), on the other hand, found an increase in flower number but a decrease in the flowering duration in potatoes, and a decrease in flower number in tomato at peak flowering in UV-B irradiated plants. Apparently, some changes in flowering are suggested that correlate with UV-B radiation, but whether this would lead to an appreciable affect in harvestable yield has not been fully investigated.

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Chapter 12

CHAPTER 12

AN ASSESSMENT OF THE EFFECTS OF ULTRAVIOLET-B RADIATION EFFECTS ON AQUATIC ORGANISMS

SUMMARY

Various experiments have demonstrated that UV-B radiation causes damage to fish larvae and juveniles, shrimp larvae, crab larvae, copepods, and plants essential to the aquatic food web. These damaging effects include decreases in fecundity, growth, survival, and other functions of these organisms. In natural marine plant communities a change in species composition rather than a decrease in net production is the probable result of enhanced UV-B exposure. The change in community composition may introduce instablilities to ecosystems and would likely have an influence on higher trophic levels. A decrease in column ozone could diminish the near-surface season of invertebrate zooplankton populations. Whether the population could endure a significant shortening of the surface season is unknown.

The direct effect of UV-B radiation on food-fish larvae closely parallels the effect on invertebrate zooplankton. Information is required on seasonal abundances and vertical distributions of fish larvae, vertical mixing, and penetration of UV-B radiation into appropriate water columns before effects of incident or increased levels of exposure to UV-B radiation can be predicted. However, in one study involving anchovy larvae, a 20% increase in incident UV-B radiation (which would accompany about a 9% decrease in the atmospheric ozone column) would result in all of the larvae within a 10-meter mixed layer in April and August being killed after 15 days. It was calculated that about 8% of the annual larval population throughout the entire water column would be directly killed by a 9% decrease in column ozone.

Effects induced by solar UV-B radiation have been measured to depths of more than 20 m in clear waters and more than 5 m in unclear water. The euphotic zone (i.e, those depths with levels of light sufficient for positive net photosynthesis) is frequently taken as the water column that reaches down to the depth at which surface of the photosynthetically active radiation is reduced 99%. In marine ecosystems, UV-B radiation penetrates approximately the upper 10% of the marine euphotic zone before it is reduced to 1% of its surface irradiance. Pentration of UV-B radiation into natural waters is a key variable in assessing the potential impact of this radiation on any aquatic ecosystem.

FINDINGS

1. REDUCTION IN STRATOSPHERIC OZONE WILL INCREASE SOLAR ULTRAVIOLET RADIATION THAT HAS THE POTENTIAL TO HARM AQUATIC LIFE.

   1a. Increases in energy in the 290-320 nm wavelengths that would reach the ground if the ozone layer depleted could harm aquatic life.

   1b. There is no single "correct" weighting function for determining the importance of different wavelengths to the damage UV-B radiation can cause; a somewhat different weighting function exists for each biological endpoint.

   1c. Based on the DNA response, a 10% ozone decrease would result in a 28% increase in biologically effective radiation, the radiation that appears most relevant to aquatic organisms.

   1d. A 10% decrease in column ozone would produce an increase in biologically effective radiation comparable to migrating over 30° toward the equator, a change of ecological significance.

2. EXPERIMENTAL DATA DEMONSTRATES A RISK TO AQUATIC LIFE.

   2a. Various experiments have shown that UV-B radiation damages fish larvae and juveniles, shrimp larvae, crab larvae, copepods, and plants essential to the marine food web.

   2b. Up to some threshold level of exposure, most zooplankton show no effect of exposure to UV-B radiation, but exposure above the dose/dose-rate threshold elicits significant and irreversible effects.

   2c. While the exact limits of tolerance and exposure have not been precisely determined for any organism, estimates of these two properties for a wide variety of aquatic organisms show them to be essentially equal.

   2d. The equality of tolerance and exposure suggests that solar UV-B radiation is currently an important ecological factor and the sunlight-exposed organisms sacrifice potential resources to avoid increased UV-B exposure to live in their particular niche. Thus, even small increases of UV-B exposure would be likely to further injure the species currently under the most stress.

   2e. A decrease in column ozone is reasonably likely to diminish the near-surface season of zooplankton. Whether the population could endure a significant shortening of the surface season is unknown.

   2f. Sublethal exposure of copepods produces a reduction in fecundity.

2g. Of the animals tested, no zooplankton possess a sensory mechanism for directly detecting UV-B radiation; therefore it would be unlikely that they would actively avoid enhanced levels of exposure resulting from a reduction in column ozone.

2h. Exposure of a community to UV-B stress in controlled experiments has resulted in a decrease in species diversity, and therefore a probable reduction in ecosystem resilience and flexibility.

2i. There is a predicted 8% annual loss of the larval anchovy population for a 9% reduction in column ozone in a marine system with a 10-meter mixed layer.

3. SIGNIFICANT LIMITATIONS EXIST IN PROJECTING DAMAGE TO AQUATIC SYSTEMS FROM CHANGES IN ULTRAVIOLET RADIATION

3a. Because aquatic organisms are small and do not usually have fixed locations, it is very difficult to obtain accurate data needed to model the systems and verify results. Understanding of the lifecycle of organisms is very limited.

3b. In common with all other living organisms, the aquatic biota cope with solar UV-B radiation by avoidance, shielding, and repair mechanisms. Uncertainty exists as to how much such behavior could reduce damage.

3c. Determination of UV-B exposure in aquatic systems is more complex than for terrestrial ecosystems because of the variable attenuation of UV-B radiation in the water column.

3d. Current data only indicate probable trends and are based on incomplete and limited observations. Consequently they must be regarded as very tentative. The possibility exists that the risks from UV-B radiation are smaller or larger than current experimental evidence appears to indicate.

INTRODUCTION

As explained in Chapter 2, it is ozone, though in very small concentrations in the atmosphere, that is almost entirely responsible for the absorption of solar ultraviolet (UV) radiation in the 290-320 nm waveband (Exhibit 12-1).

If Exhibit 12-1 were modified to account for a 10% ozone layer reduction, given the scale, one would scarcely notice the difference. The additional UV radiation reaching the earth's surface as a result of a 10% ozone reduction would amount to less than a 0.05% increase in the total sunlight energy. At the very margin of the diagram, however, the percentage increases would be much larger because the base is so small and it is in those wavelengths that biological responses to ultraviolet radiation received from the sun appear substantially greater.

Based on models of depletion (see Chapter 19, Appendix A), a 10% ozone reduction at 45°N latitude would result in a 28% increase for biologically effective radiation for DNA damage (Setlow 1975). The generalized plant response would increase by 21% (plant damage) (Caldwell 1974). Exhibit 12-2 shows the relationship between ozone depletion and action spectra. Because of the relatively high increases in UV-B at the lower ends of this part of the spectrum and its relative biological effect, ozone depletion constitutes a threat to aquatic life.

BACKGROUND ON MARINE ORGANISMS AND SOLAR ULTRAVIOLET RADIATION

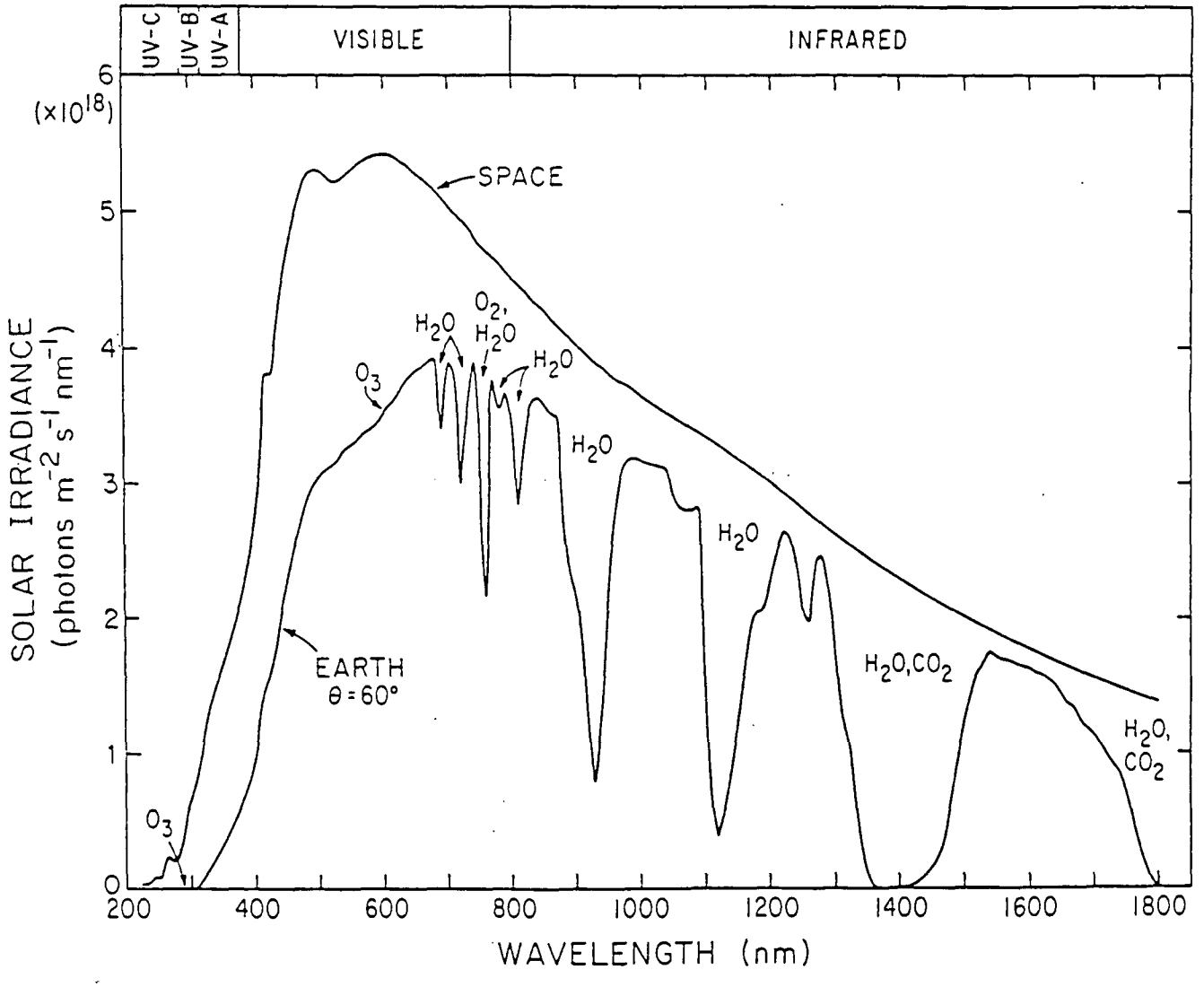
Organisms in fresh water or the oceans are either swimmers (nekton), bottom-dwellers (benthos), or drifters (plankton). Plankton can be either plants (phytoplankton) or animals (zooplankton). An important zooplankton group, the ichthyoplankton, is comprised of the drifting eggs and larvae of many species of fish.

Phytoplankton provide essentially all of the chemical energy required by the marine food web through the photosynthetic process (primary production). Zooplankton are consumers (grazers). Zooplankton are usually less than about 5 cm in diameter or length, most often less than 1-2 mm. Nearly all groups of aquatic animals, at least for some phase in their life-history, are considered zooplankton -- for some species, in the egg and/or larval stage, for others, throughout their life cycle. Zooplankton are critical components in typical aquatic feed webs (nutrient pathways) that lead to larger animals, including those comprising the fisheries (finfish and shellfish) and therefore affect man himself.

In marine ecosystems, UV-B radiation penetrates approximately the upper 10 meter of the marine euphotic zone before it is reduced to 1% of its surface irradiance. Penetration of UV-B radiation into natural waters is a key variable in assessing the potential impact of this radiation on any aquatic ecosystem (Exhibit 12-3).

EXHIBIT 12-1

Solar Irradiance Outside the Earth's Atmosphere and at the Surface of the Earth for a Solar Zenith Angle of 60°



Adapted from Forsythe 1954. Regions of significant absorption by atmospheric ozone, oxygen, water vapor, and carbon dioxide are shown.

EXHIBIT 12-2

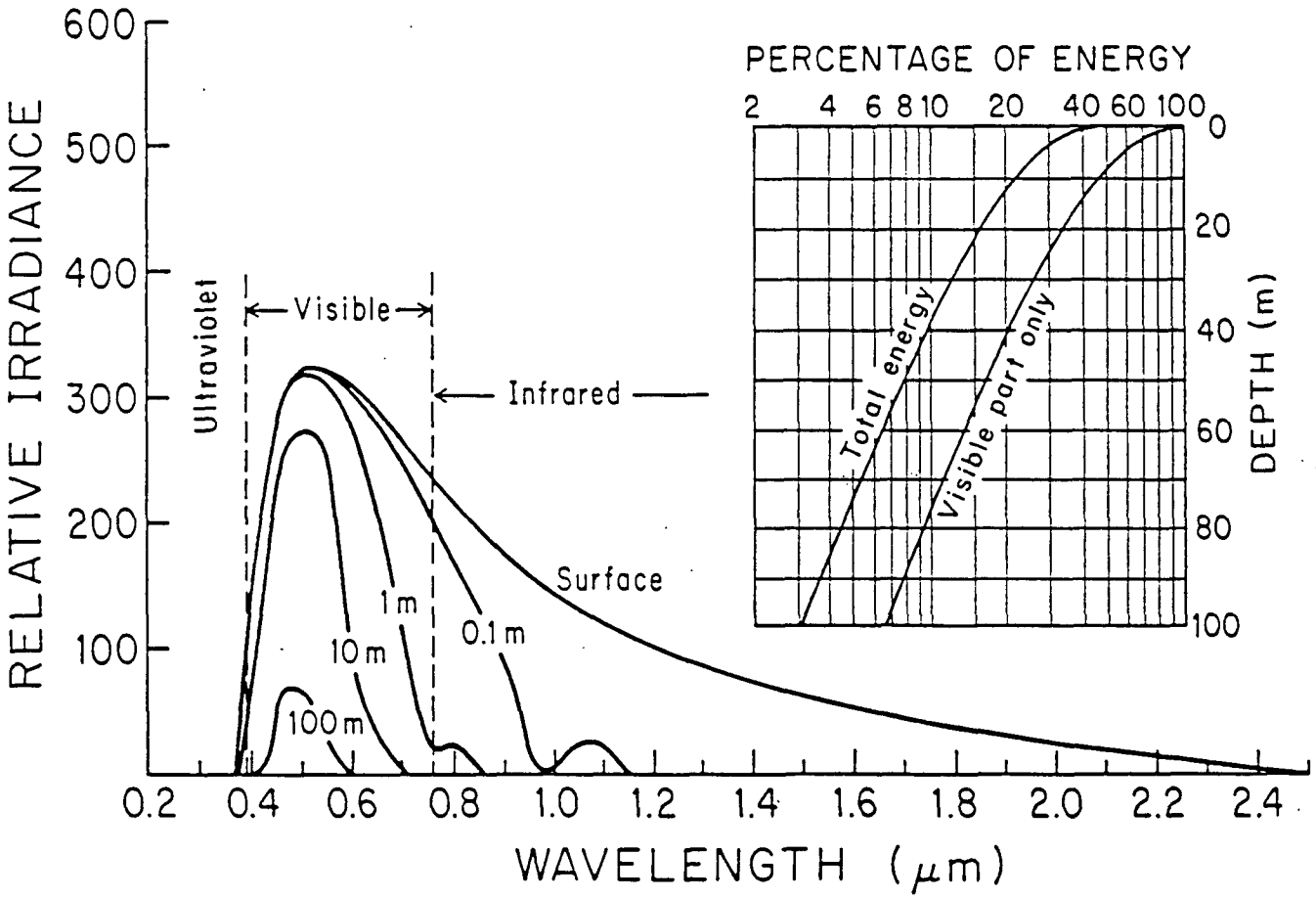
Relationship between Ozone Depletion and Biological Effectiveness of Increased UV-B Radiation (Based on model by Green, Cross, and Smith)

Ozone Decrease	Increase in UV Radiation		Increase in Biological[a] Effectiveness	
	290-320 nm	290-360 nm	DNA	Plant
10%	8%	1.1%	28%	21%
20%	17%	2.4%	67%	49%
30%	27%	3.7%	125%	85%
40%	38%	5.2%	213%	132%

[a] Action spectra are referenced to 300 nm = 1.00.

EXHIBIT 12-3

Solar Spectral Irradiance at the Surface of the Ocean
and at Four Depths



Adapted from Sverdrup, Johnson, and Fleming 1942. Insert illustrates percentage of energy transmitted at depth in oceanic waters.

Photosynthesis requires light, but adequate sunlight seldom penetrates to the bottom of natural waters. Thus, aquatic plants are largely confined to a relatively thin layer at the surface of the water, which is termed the euphotic zone, the surface layer where plants create more chemical energy by photosynthesis than they use for their own metabolism. This zone is where UV-B can penetrate. Zooplankton are found at all depths, depending on species and season, but are most abundant in the sunlit upper 100 meter where the bulk of their food, including phytoplankton, is found. Many species normally live very close to the surface, even in daylight, while others occupy the near-surface layer during only part of their life cycle. The near-surface layer is a very important zone in the interactions of the physical/chemical/biological components of aquatic systems.

Zooplankton have apparently evolved mechanisms and behavior by which they have adjusted to current levels of UV radiation (Damkaer 1982), but they may not be able to adjust to relatively rapid increases in total UV exposure. If there are changes in abundance of zooplankton species, those changes would have an impact far beyond any direct effects because of the critical role zooplankton play in energy transfer within the ecosystem.

Limitations of Pioneer Investigations

As early as 1925, scientists were aware of damaging effects from the ultraviolet component of sunlight on aquatic organisms (Huntsman 1925; Klugh 1929, 1930; Harvey 1930; ZoBell and McEwen 1935; Giese 1938; Bell and Hoar 1950; Dunbar 1959; Marinaro and Bernard 1966). These reports are, for the most part, of historical interest only; they cannot be strictly related to present investigations because of a lack of precise or absolute measurements of UV dose-rates and doses. A lack of proper instrumentation for UV irradiance measurements in aquatic research including laboratory studies, persisted into at least the late 1960's.

Limitations of Recent Investigations

There are only a few reports that were based on experimental methods appropriate to the problem of enhanced solar UV-B radiation. This meager body of knowledge does not compare well to the enormous literature available on, for example, chemical pollution. Even for chemical pollution there are still great uncertainties in predicting ecosystem responses. It must be recognized that attempts to predict ecosystem responses to enhanced UV-B radiation are in the earliest stages. A number of reports are difficult to apply because the experiments used extraordinarily high UV-B dose-rates and/or doses. Frequently investigators increased radiation in damaging wavelengths far beyond the amount that can be expected from ozone depletion.

Another problem in predicting UV-effects is that we are not yet very close to being able to extrapolate the effects on one species to those on another. Nor can we say with much assurance how the same species might react to UV radiation in a different environment. There is simply too little data of an acceptable quality to go beyond a general assessment of risk. All that follows must start off with these caveats.

EFFECTS OF UV-B ON RADIATION PHYTOPLANKTON

Direct Impact on Primary Productivity

The amount of UV-B radiation reaching the ocean's surface has long been suspected as a factor influencing primary production. Research shows convincingly that ultraviolet radiation, at levels currently incident at the surface of natural bodies of water, has an influence on phytoplankton productivity (Steemann Nielsen 1964; Jitts et al. 1976; Hobson and Hartley 1983). It has been calculated that, near the surface of the ocean, a 25% reduction in ozone concentration would cause a decrease in primary productivity by about 35% (Smith et al. 1980). The estimated reduction in production for the whole euphotic zone would be about 10%. Because these calculations were based on attenuation lengths, (i.e., the product of depth in the water column and the diffuse attenuation coefficient of water), waters of various turbidities and absorption characteristics can be compared.

If one assumes that present phytoplankton populations sense and control their average vertical position in such a way as to limit UV-B exposure to a tolerable level, then a 10% increase in solar UV-B radiation would necessitate a downward movement of the average position, thereby reducing the average UV-B exposure by 10%. There would be a corresponding reduction of light for photosynthesis. The loss of photosynthetically active radiation in many locations might not be significant. However, in some very productive areas, especially high latitude ocean areas, photosynthetically active radiation is the primary limiting factor for marine productivity (Russell-Hunter 1970). The loss of photosynthetically active radiation from optical measurements has been estimated to be in the range of 2.5% to 5% for a 10% increase in UV-B radiation (Calkins 1982). If the photosynthetic base of aquatic ecosystems were perturbed, one would expect ramifications to extend up through the food web through predator-prey relationships.

Experiments with marine diatoms have shown significant reductions in biomass, protein, and chlorophyll at UV-B irradiances equivalent to ozone reductions that would occur for a 5%-15% ozone depletion. Laboratory studies on chain-forming diatoms and other phytoplankton show that growth increased when UV-B radiation was filtered out of the incident solar radiation, indicating that existing levels of UV-B radiation depress productivity (Thomson, Worrest, and Van Dyke 1980; Worrest 1982).

Effects Because of Changes in Motility

An additional effect is that UV-B radiation may endanger the survival of microorganisms (Euglena, slime mold, some blue-green algae) by decreasing their motility and by inhibiting phototactic and photophobic responses (Haeder 1986). The inability of a population to move into favorable environments, could result in damage that may impair development.

Effects on Photosynthesis

Jones and Kok (1966) observed a direct inhibitory effect of UV radiation on photosynthesis in chloroplasts. This form of inhibition is a short-term response and shows a different UV-B wavelength dependence from most other forms of UV-B injury. Smith et al. (1980) found the short-term depression of photosynthesis by natural solar UV radiation measured in the ocean resembles the direct photoinhibition described by Jones and Kok. If direct photoinhibition were the only pathway for reduction of photosynthesis by marine phytoplankton, the effect of ozone reduction would be minor; however, Smith and Baker (1980) point out that there could be long-term effects that might show a different action spectrum and be of more consequence.

In Hawaii long-term photoinhibition of growth of six algal species under natural sunlight has been measured (Jokiel and York 1984). Two strains could not grow at all at the levels of irradiance for full natural surface sunlight. Of the species tested, those collected from tropical surface water showed the greatest adaptive power, but it is reasonable to conclude that resistance to solar UV radiation is achieved through expenditure of resources that can be better applied to other needs in less exposed species.

Worrest et al. (1981) found a short-term (4-5 h) depression of photosynthesis in seven strains of algae upon exposure to enhanced levels of UV radiation. There was a large difference in strain sensitivity. The observed depression appeared to linearly correlate with UV-B dose using the DNA (Setlow 1974) action spectrum. This is in contrast to the results of Smith et al. (1980) who found the Jones and Kok weighting function to be appropriate.

Multi-Day Effects

Measurements of photosynthesis that span only a single day could underestimate the overall action of solar UV exposure by failing to account for effects on the next day's population level. The effects of UV-B radiation could result in delaying growth or direct killing. Thus, subsequent population could fall below the numbers that an unexposed population would attain. Prolonged delays (about two days) in growth of the survivors have been observed in two strains of the diatom Thalassiosira, which had been irradiated with simulated solar UV-B radiation at doses below lethal levels. If unicellular organisms are in a rapid growth phase, a growth delay equalling the time of one growth cycle produces the same effect on the subsequent population as would be produced by a 50% killing (without growth delay).

Shifts in Community Composition and Possible Implications

Shifts in species diversity and community composition of phytoplankton communities have been observed in a simulated marine ecosystem exposed to UV-B equivalent to ozone decreases of 15%, (Worrest et al. 1981). Natural communities appear to show changes in species composition rather than a decreasing net production. Experiments in simulated marine ecosystems seem

to indicate that higher UV-B radiation will not decrease biomass and chlorophyll accumulation, (Worrest et al. 1981). However, a change in community composition might result in a more unstable ecosystem and might have influence on higher trophic levels (Kelly 1986). For example, one effect of enhanced levels of UV-B radiation would be to alter the size distribution of the component producers in a marine ecosystem. Increasing or decreasing the size of the representative primary producers could alter the energy allotment that grazers use for finding and consuming food. This could reduce the feeding efficiency of the consumer, which would have a variety of effects. In addition, the food quality of the producers is altered by exposure to UV-B radiation. It has been demonstrated that the protein content, dry weight, and pigment concentration are all depreseed or the composition altered by enhanced levels of UV-B radiation (Doehler 1984, 1985). Consequently, a UV-induced shift in the distribution and abundance of different kinds of aquatic plant organisms could have important ramifications throughout the ecosystem.

EFFECTS ON INVERTEBRATE ZOOPLANKTON

Background

There have been several studies on the effects of UV-B radiation on a variety of zooplankton. Investigators have reported that the effect of increased UV-B radiation on some marine zooplankton (e.g., copepods, shrimp larvae, crab larvae) is to increase the mortality of the organisms and to decrease the fecundity of the survivors. Regardless of the species investigated, the potential exists for a disruption of the food web, which would indirectly affect fish populations that are important as food. Of course, studies that consider economically important zooplankton species, such as larval stages of certain shrimp, crab, and fish, would have importance to man because damage to these species would directly reduce the amount of food for human consumption.

Methodological Issues

How well photorepair mechanisms might mitigate damage is an important issue. It appears that up to some level (daily-dose threshold), photorepair will mitigate damage. However, it also appears that near-surface exposure levels that could accompany a significant reduction in column ozone would exceed threshold levels.

The issue of thresholds is important. It appears that regardless of the cellular-level responses to UV irradiation, it is usually noted that up to some level of UV exposure, the organism appears to suffer no harm. Extrapolating survival curves for different UV exposure levels would indicate that organisms can cope with the damage produced by some levels of additional UV radiation without negative effects. At greater doses, where the survival curve becomes steep, it appears that the repair systems may become inactivated by the radiation, or that the damage to the general tissues has gone beyond the capacity of the repair systems.

A study by Damkaer et al. (1980) illustrates the importance of threshold effect. The authors found no alterations in activity, growth-rate, developmental rate, and survival in a number of shrimp and crab larvae exposed to low levels of artificial UV-B radiation. However, when a certain combination of UV dose-rate and total dose was exceeded, negative effects became quite sharply noticeable.

Lethal Effects of Enhanced UV-B Radiation

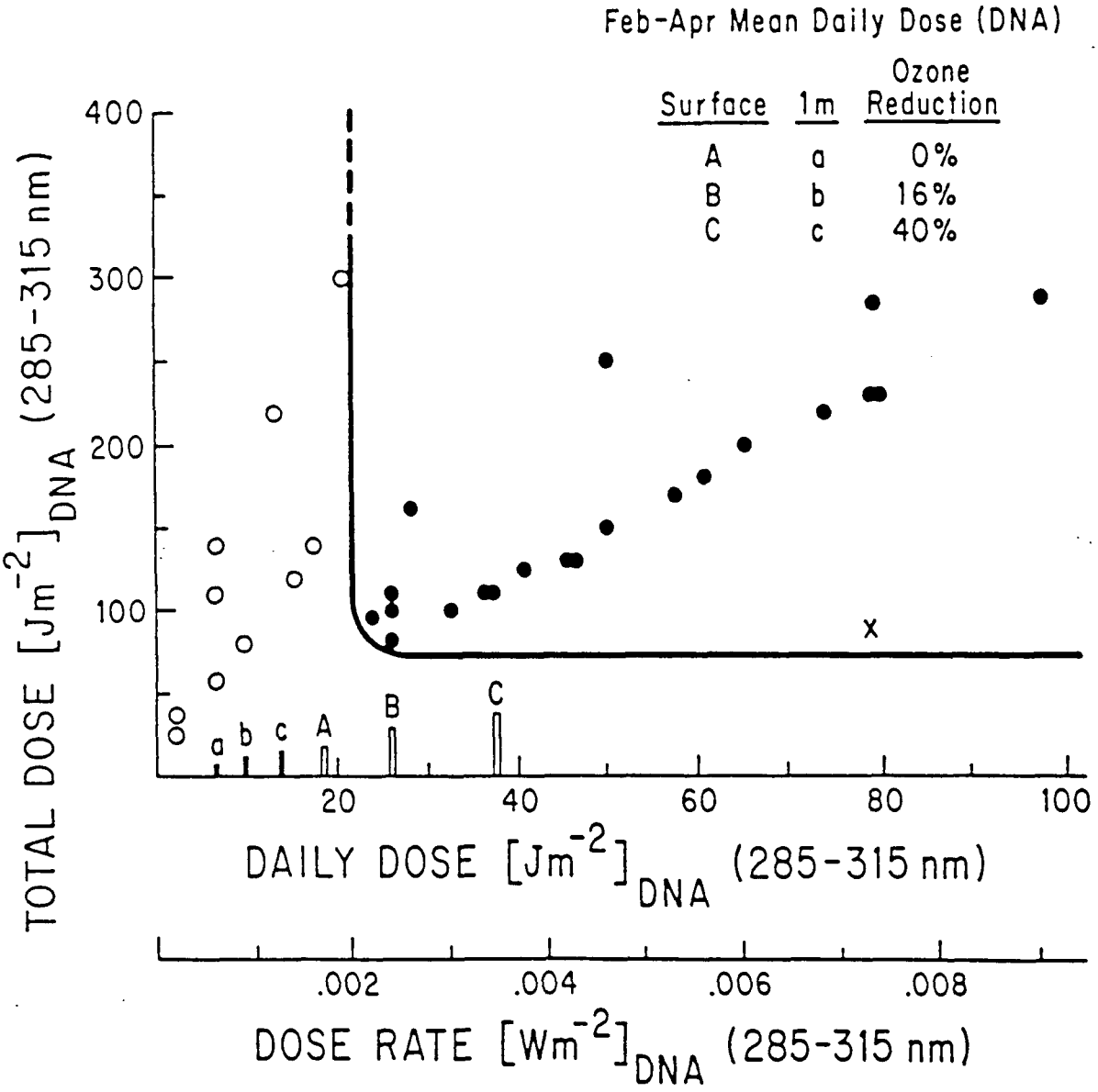
Damkaer et al. (1980) have done a number of studies in which enhanced UV-B radiation produced irreversible damage and death. UV thresholds, based on the lowest level or irradiance where statistically significant (exceeding the 95% confidence limits of five replicates). Irreversible effects occurred compared to the controls. Damkaer et al. (1980) have compared the effective UV thresholds with the maximum effective solar daily dose rates at an experimental site in Washington state (Exhibit 12-4). The results indicated that the maximum solar levels are currently exceeded for all tested organisms. Obviously, these animals can survive threshold values for maximum solar UV levels. To be effective, UV thresholds probably must be exceeded during several consecutive days. A more realistic approach would be to compare the threshold levels with median solar UV levels (Exhibit 5). The threshold for all groups would then appear to be above the present median solar incident UV levels, at least until late in the time span of natural occurrence near the surface. The data seem to suggest that UV levels have exerted a considerable influence in the long-term adjustments of these populations to those specific seasons. Of course, many other physical as well as biological factors operate concurrently, and the organism's life cycle is a compromise with the total environment. Late in the surface season, it is possible that natural UV levels exert a detrimental effect, particularly on the shrimp larvae, which have a lower total dose tolerance than crab larvae.

Damkaer et al. (1980) compared the estimated effective UV daily doses simulating various ozone reductions at the experimental site with the UV survival thresholds (Exhibit 12-5). Obviously, the probability that UV thresholds would be exceeded increases with diminishing ozone. With a 40% ozone reduction, there appears only to be a short "window" of safety at the beginning of each group's surface season. Even a 20% ozone reduction significantly shortens the season. Whether or not the populations could endure with a drastically reduced time of near-surface occurrence is not known. Success of any year-class depends on the timing of a great number of other events besides UV level. Under current stress conditions, early larvae may do well one year, whereas only late larvae may survive in a subsequent year. However, an additional stress like enhanced levels of UV-B radiation is not likely to be beneficial. Combined with other stresses that make early year survival impossible, it could devastate populations.

That larval stage development appeared to be unaffected below survival threshold UV levels is an important observation, since the survival of the unharmed larvae is critical to their proper and timely development. Beyond the threshold levels, however, development and survival rapidly declined.

EXHIBIT 12-4

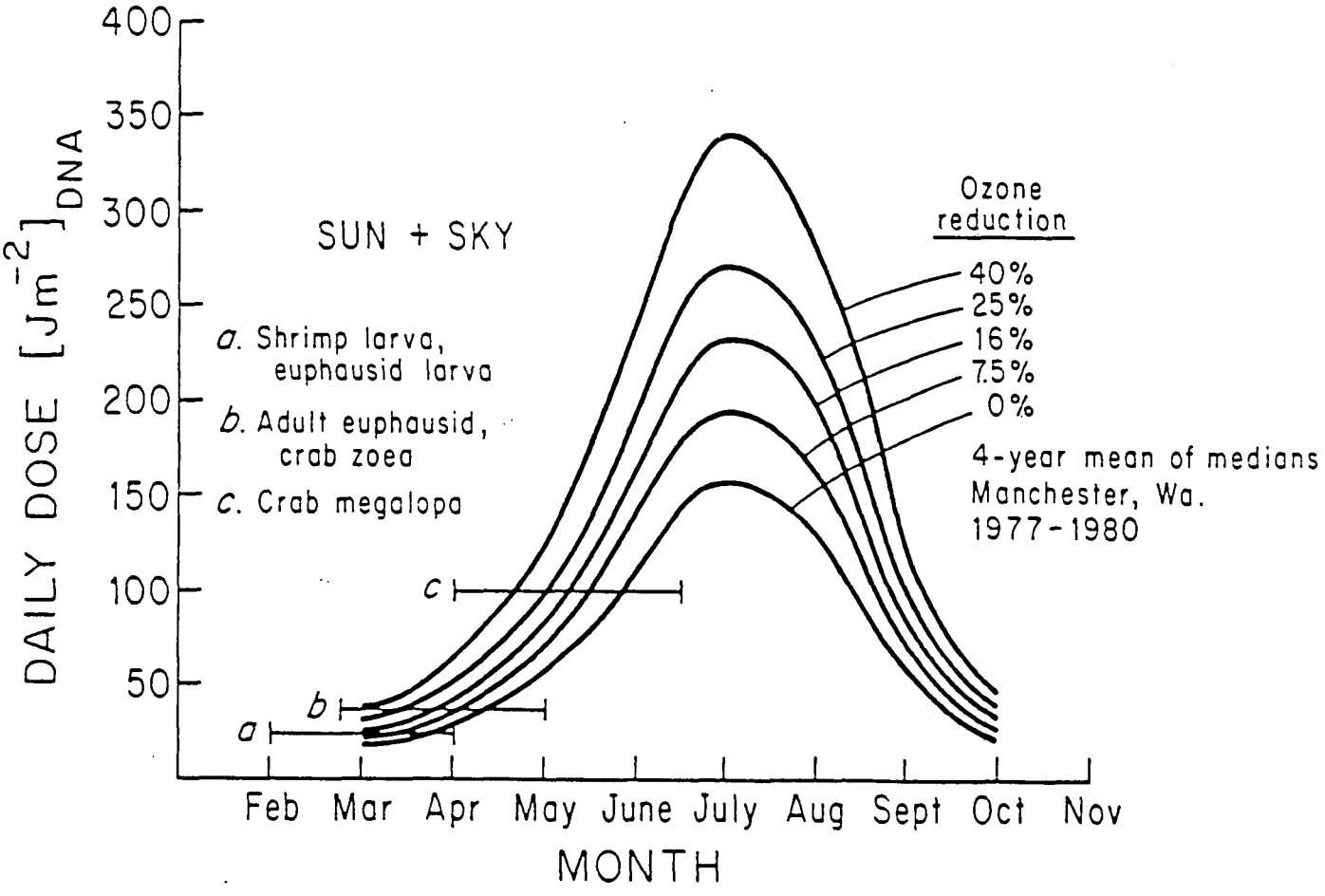
Lethal Effects on Shrimp Larvae for Various Combinations of UV-B Dose-Rate and Total Dose



Open circles indicate maximum observed combinations for survival; closed circles indicate minimum observed combinations for mortality. Open vertical bars indicate mean February-April surface solar UV-B daily dose-rates at various atmospheric ozone concentrations; closed vertical bars indicate mean daily dose-rates at 1 m (adapted from Damkaer et al. 1980).

EXHIBIT 12-5

Estimated Effective UV-B Solar Daily Dose at Varoius Atmospheric Ozone Concentrations Based on 4-Year Mean of Medians, Manchester, Washington, 1977-1980



Dose increments are from Gerstl, Zardeki, and Wiser 1986. Also shown are approximate thresholds of UV-B daily dose for principal experimental zooplankton groups, in natural seasonal position (adapted from Damkaer et al. 1981).

The apparent UV thresholds are near-current incident surface UV levels. Nevertheless, the implications of these experiments for zooplankton in nature remains in some doubt because of lack of information on the subsurface UV spectrum, the effects of vertical mixing, unknown in situ behavior of irradiated zooplankton, and unknown sublethal and pre-lethal effects.

The degree to which other zooplankton groups would be affected by these levels of UV-B irradiation cannot be predicted. Although, in crustaceans the same basic tissues would likely be involved, and therefore the response would be qualitatively similar; species vary substantially insensitivity to UV-B radiation. If the accumulated level of UV-B radiation is the most important factor in damage, then increased UV radiation through ozone depletion may significantly affect even species that are moderately resistance to short exposures of high UV intensities.

Copepods are the most important zooplankton from the perspective of ecosystems. Karanas, Worrest, and Van Dyke (1979) reported on the survival of developmental stages of the estuarine copepod Acartia clausii exposed to daily UV-B dose approaching natural (Oregon coast) daily doses. Although the total doses in this experiment were relevant, the dose-rates exceeded natural dose-rates by about a factor of two.

It is known that organisms are protected to some degree by their own pigmentation, and this suggests that organisms may have evolved pigmentation as a consequence of the damaging effects of solar radiation. Damkaer (1982) reviewed several possibilities for this in regard to zooplankton. Siebeck (1978) found a direct relationship between pigmentation in cladocerans of mountain and lowland lakes and their apparent natural UV exposure. In field and laboratory experiments, the more-pigmented cladocerans tolerated the greater exposures. Siebeck also noted a marked photorepair potential.

Hairston (1980) has drawn attention to the relationship between vertical distribution and pigmentation in some freshwater copepods. Typically the most pigmented forms are closest to the surface. Because the pressure of predation should be against the conspicuous pigmented forms, Hairston concluded that "photodamage may be a more important selective force than previously supposed."

That these kinds of copepods do have a UV-tolerance related to their pigmentation has been verified in laboratory experiments. Ringelberg, Keyser, and Flik (1984) studied the UV-B tolerance of a red-pigmented and a translucent variety of the same copepod species from separate, though nearby, lakes in France. The red copepod tolerated higher artificial UV-B doses. The experimental doses were obtained by exposure to a single dose-rate for varying amounts of time. Sharp mortality increases began in the translucent form at about half the total dose as for the pigmented form.

An interesting aspect of these experiments is that in all cases where death occurs, the dose-rate needed to reach the threshold effect for shrimp (Damkaer et al. 1980). The total doses tolerated by the two copepod color-forms bracket the total dose threshold of shrimp larvae. The tolerance

limit appears to be the same in vulnerable zooplankton, suggesting perhaps a universal tolerance limit in near-surface organisms of the structure and size of shrimp larvae and copepods. It would be useful to conduct additional experiments with such copepod color-forms and include exposures to lower and higher dose-rates at more natural periodicites. Also, Ringelberg et al. observed no photorepair, but indicated that the intensity of visible radiation in their experiments was extremely low (~0.4 W m^{-2}).

Thomson (personal communication) has demonstrated that current levels of UV-B radiation are of significance in the developmental life of several species of shellfish. For some species a 10% decrease in column ozone could lead to as much as an 18% increase in the number of abnormal larvae produced. Exhibit 12-6 is a compilation by Thomson of data denoting the estimated biologically effective UV-B doses leading to significant effects in major zooplankton groups.

Sublethal Effects of UV-B

There are many examples of sublethal stresses (of all kinds) that can affect natural populations. If stresses alter the reproductive capacity of organisms, effects can be significant and rapid. Karanas, Worrest, and Van Dyke (1981) reported a possible mechanism through which enhanced solar UV-B radiation could have a large impact on copepod populations without direct killing. The estuarine copepod Acartia clausii was irradiated with UV comparable to mid-summer surface irradiance at 45°N. After five days, the surviving copepods were mated. If either (but not both) parent had been irradiated, the egg production and subsequent hatch of the female was significantly reduced. If both parents had been irradiated, the production of viable larvae was halved. The subsequent survival of the hatched larvae from irradiated crosses did not appear to be significantly affected.

Even though Karanas, Worrest, and Van Dyke (1981) applied a natural-level surface dose-rate of artificial UV-B radiation, this dose-rate was still rather high and probably represents an extreme situation. To obtain reasonable total doses, the copepods were exposed for only 1-2 hours. Real dose-rates, especially at depth, would be considerably less. These copepods are found at the surface, as well as some distance below, but their distribution on the required scale of 1 m or less is not well known. Acartia clausii are taken in a wide variety of water types, but generally this is a coastal/estuarine species. In its usual habitat, effective UV-B irradiation may not extend beyond a few meters depth. In view of the apparently profound sublethal, reproductive response to UV-B radiation, it would be of great interest to have additional multi-generation observations.

Possible Photorepair of UV-B Damage and Its Implications on Using Experimental Data

The photochemistry and photobiology of UV-B-induced damage, particularly nucleic acid disruption, as well as the several molecular repair mechanisms that are consistently found in plants and animals, have been reviewed by Caldwell and Nachtwey (1975). Among the repair mechanisms, the quantitative

EXHIBIT 12-6

Estimated Biologically Effective UV-B Doses Leading to Significant Effects in Major Marine Zooplankton Groups

Zooplankton Group	Dose Rate W m^{-2} (DNA eff)[a]	Total Dose J m^{-2} (DNA eff)	Time For Effect
Shrimp/Euphausiid larvae	6.0×10^{-2}	2.6×10^{3}	4 days
Euphausiid adults	9.9×10^{-2}	6.5×10^{3}	6 days
Copepod larvae	3.4×10^{-1}	1.4×10^{3}	1.0 hour
Copepod postlarvae	1.6×10^{-1}	2.7×10^{3}	4.5 hours
Copepod adults	3.4×10^{-1}	3.0×10^{3}	2.5 hours
Crab larvae	9.9×10^{-2}	6.5×10^{3}	6 days
Crab postlarvae	2.8×10^{-1}	6.0×10^{4}	20 days
Anchovy/Mackerel	6.0×10^{-2}	2.5×10^{4}	12 days
Oyster/Mussel larvae	1.2×10^{-1}	2.2×10^{3}	5.0 hours

[a] DNA action spectrum is referenced to 300 nm = 1.00.

Complication by Thomson.

EXHIBIT 12-7

Annual Fish Production in Coastal Waters
Baseline Data for Coastal Waters

	Baseline	-5% P^1	-5% e
Primary production, kJ m^{-2} yr^{-1}, (P^1)	4200	4000	4200
Ecological efficiency per trophic level, (e)	0.15	0.15	0.14
Number of trophic levels from plant production to fish production, (n)	3	3	3
Fish production, kJ m^{-2} yr^{-1}, (P^1e^n)	14	13	12
Total fish production, 109 kg yr^{-1} (using Winberg's transformation, 4.2 kJ = 1 g fish flesh)	121(100%)	115(95%)	103(86%)

Adapted from Ryther (1969).

capacity of the dark-repair systems is quite different from those systems that require light. Under typical experimental conditions, it is presumed that the dark-repair mechanisms could function freely. Dark-repair systems appear to offset a very high proportion of the damage caused by relatively low doses of irradiation. However, photorepair mechanisms often appear capable of repairing only so much damage, even over a wide UV-B dose range (Murphy 1975). Consequently, the restorative effects of photoreactivation may be regarded as particularly critical in situations where organisms are subjected to UV-B irradiation near and above tolerance limits. There has been an implication in some review papers (e.g., NAS 1984) that UV-B damage will be overcome by the maximally functioning photorepair mechanism. Up to some level (daily dose threshold) this is probably true, but beyond that threshold there will be some residual, irreversible damage.

Experiments with artificial sources of UV-B radiation usually are conducted in the laboratory under light regimes using more than an order of magnitude less visible light than found in nature. Near UV and/or visible light is needed for photoreactivation, and the accuracy of some observations may have been influenced by less-than-maximum photorepair in the laboratory. Experiments with shrimp larvae and adult euphausiids (Damkaer and Dey 1983), comparing survival at various UV-B doses and dose-rates combined with different levels of near-UV and visible irradiance, suggest that photorepair occurs in these organisms. The dramatic sensitivity of both shrimp larvae and adult euphausiids when deprived of photoreactivating light clearly demonstrates the relative importance of photorepair under UV stress. However, this apparent photorepair reaches maximum levels at relatively low levels of visible irradiance. Consequently, it is doubtful that photorepair and related survival would have been greater if experimental levels had been equal to solar levels of irradiance.

A similar relationship has been reported between survival in UV-damaged anchovy larvae and level of irradiance of photoreactivating light needed for repair (Kaupp and Hunter 1981). The level needed for maximum photorepair is far less than ambient solar irradiation. The photoreactivation experiments on shrimp larvae and euphausiids suggest that the previously derived dose/dose-rate thresholds are not altered with additional photoreactivating light. Clearly, a significant lack of photoreactivating light, which normally would be concurrent with solar UV-B irradiance, would certainly depress threshold levels. Clearly, past experiments still have not approached the intensity or dose of local solar photoreactivating light. However, for UV-B exposures below threshold levels maximum photorepair potential has been reached at the low levels (2-9 W m^{-2}) of photoreactivating light. Nearly doubling the dose-rate and dose of photoreactivating light did not increase the survival of euphausiids. With anchovy larvae, Kaupp and Hunter (1981) reported that the photorepair mechanisms appeared to be "fully activated" at a photoreactivating light (320-500 nm) irradiance of 7 W m^{-2} and a daily dose of 77.5 kJ m^{-2} available over four days. At greater irradiance levels, up to levels equivalent to solar photoreactivating light at 45°N latitude, Kaupp and Hunter observed no further increase in anchovy survival. There is extraordinary close agreement between the relatively low dose-rates and doses

necessary for apparent maximum photoreactivation in anchovy larvae and euphausiids. Thus, most of the experimental data cannot be eliminated on the basis of failing to account for photorepair.

Another important point is that from these observations, it is clear that in nature there would always be sufficient photoreactivating light to provide maximum photorepair potentials in shrimp larvae and euphausiids (and also in anchovy larvae). But it must be emphasized that even maximum photorepair may not be sufficient to reverse all potential injuries caused by UV-B radiation.

Possible Vertical Distribution and Its Implications on Using Experimental Data

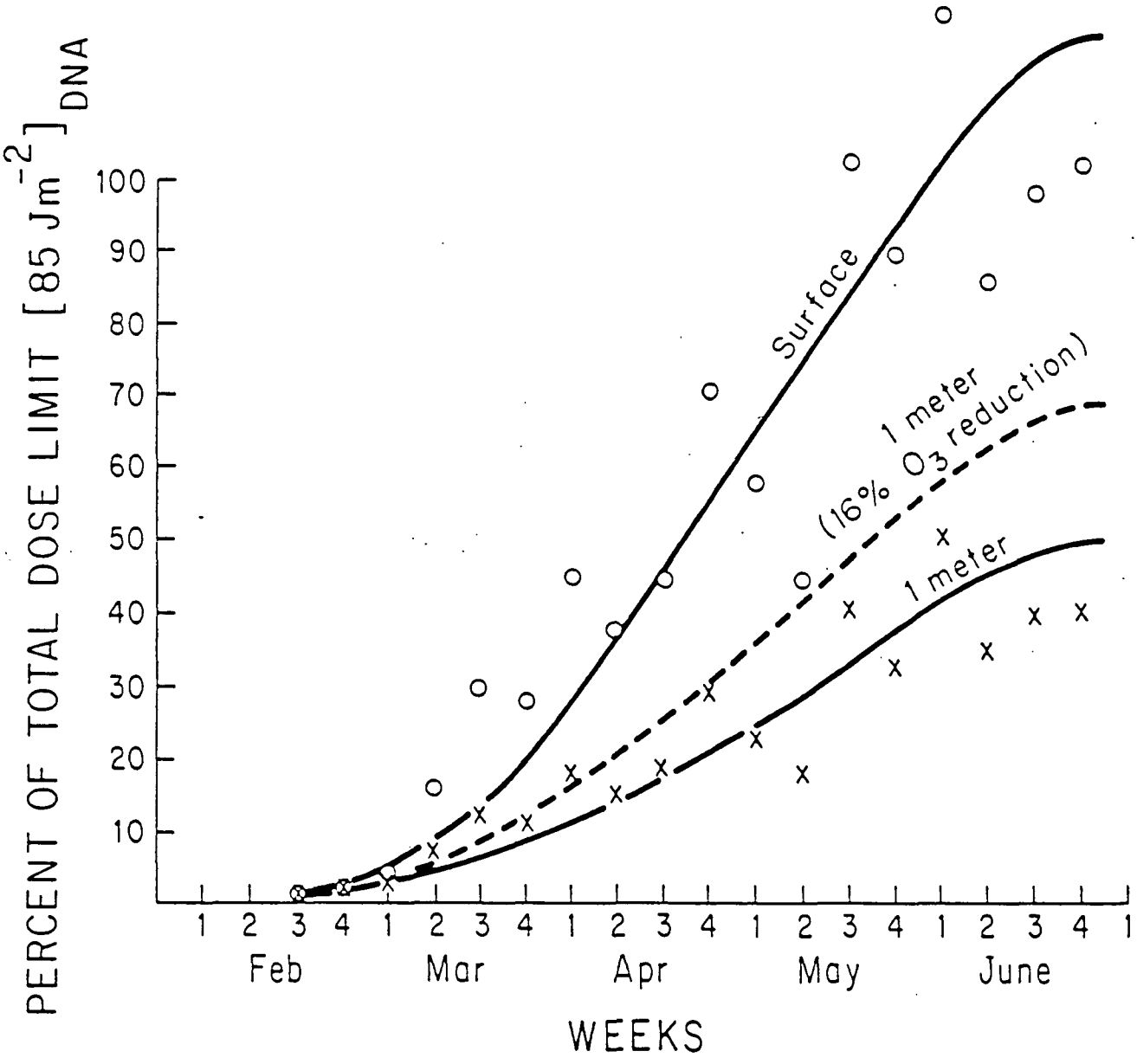
The actual solar radiation to which surface-living zooplankton are actually exposed in nature is very important in assessing risks to higher ambient UV-B. Given the large effects UV radiation had on zooplankton and the apparent existance of threshold levels, understanding UV-B in the natural environment is critical to assessing risks. To assess the potential dangers to zooplankton populations from increasing solar UV-B irradiaton requires consideration of the subsurface irradiance levels. The data for the shrimp larvae shown in Exhibit 12-4 show that the mean of the observed daily UV-B surface doses (current ozone thickness) between mid-February and mid-April is within the experimentally determined safe zone. Daily doses calculated for a 1-m depth are therefore even farther within the safe zone, so that presumably the shrimp larvae could tolerate these mean dose-rates for long periods. With a 16% or 40% ozone reduction, the total-dose thresholds would be exceeded at the surface only if the mean dose-rates are attained for two or three consecutive days (two sunny days). Even then, however, the calculated mean UV-B levels at 1 m would remain in the safe zone. Understanding the implications of higher UV-B requires knowing where the shrimp larvae spend their time.

The actual vertical distributions of the shrimp in nature are not known. In the laboratory, young larvae are attracted to light and definitely prefer the near surface. The larvae do not appear to avoid high UV-B irradiance (Damkaer and Dey 1982), It is not likely they can sense this radiation in sufficient time to prevent lethal damage. Damkaer and Dey assumed that the younger shrimp larvae are constantly moving toward the surface, and that during the day the majority of the population is in the upper meter.

Vertical motion makes it difficult to determine the probability that particular shrimp larvae would receive lethal exposures of UV-B radiation; in Exhibit 12-8, an approximate estimate is made. The probability is obviously time-dependent. At the surface, shrimp larvae are unlikely to encounter lethal daily or bi-daily UV-B exposures until the end of the surface season (mid-April). This is also about the time that the mean noon dose-rate exceeds the dose-rate threshold. Before then, the shrimp larvae would be exposed daily to a one-third total dose limit for three consecutive days. This level is in the safe exposure zone because the dose-rate would be low (Exhibit 12-8). At 1 m, the mean dose-rates in mid-April are still less than the shrimp's threshold dose-rates, yet the surface probabilities for lethal

EXHIBIT 12-8

Percentage of Total Dose Limit Likely to be Reached on Any Particular Day; Lethal Doses Accumulated Only After Dose-Rate Threshold is Exceeded



Circles are 4-year weekly means of surface daily UV-B doses; x's are these doses calculated at 1 m (adapted from Damkaer et al. 1981).

exposure may be sufficiently high at that time to limit the seasonal occurrence of the larvae. With a 16% ozone reduction, the current mid-April UV-B conditions would be reached near the end of March. If UV-B radiation is a contributing factor in regulating the seasonal occurrence of shrimp larvae, ozone reductions may act to shorten the surface time available to the larvae. The seasonal relationships in the UV-B tolerance of different zooplankton groups (Exhibit 12-8) suggest that UV irradiation is an important environmental factor. Hunter, Kaupp, and Taylor (1982) postulate that the surface occurrence of eggs and larvae of a number of clupeid fishes are already limited by UV-B.

Knowing that shrimp larvae are limited by UV-B already, and could be further limited, does not provide us with an assessment of damage. Data on population dynamics and recruitment of shrimp stocks are insufficient to estimate the impacts of shortened surface seasons. It is possible that ordinarily the greatest number of successful recruits are normally derived from early-season larvae and that increased UV-B irradiance would then have no appreciable direct effect on shrimp population stability. It is also possible that late-season larvae are essential to maintaining local populations. In fact, it is possible that the situation varies from year to year with other environmental stresses, so that having a long season is critical to sustaining the population in the long term. Thus ozone reductons may lead to no change, to a loss in productivity, or to extinction of these shrimp in particular locations.

It is clear that increases in UV radiation through depeletion of ozone would create an additional stress to the seasonal near-surface stages of these species. It is quite possible that natural UV levels have already had a selective role in the seasonal adaptation of these species. The question remains: how much of their surface-living time could these species forego and still maintain survivor-level populations?

Avoidance and Its Implications In Assessing Risk

Investigation of the biological effects of UV-B radiation on aquatic organisms have included a variety of approaches, ranging from histological analyses to simple survival studies. Most of this work has been concerned with the direct effects of natural or enhanced UV-B radiation. The assumption implicit in some of the conclusions is that the organisms that live in the near-surface layer will be passive recipients of ambient UV-B radiation, regardless of its irradiance level.

Because some of these near-surface organisms appear to be living near their UV tolerance limits (Damkaer et al. 1980) it is possible that they have evolved a sensitivity to fluctuations in UV-B radiation. In addition to photorepair mechanisms, they may possess an ability to avoid harmful levels of UV-B radiation by appropriate defensive behavior. In moderately prdouctive ocean waters, the DNA-weighted irradiance at 1 m is only about 40% incident on the surface (Smith and Baker 1979). Under these conditions, the capacity to sense harmful UV-B radiation and to avoid it by simply swimming or sinking a meter below the surface would provide considerable protection.

However, even if sensory mechanisms exist to warn an organism of dangerous UV-B radiation, the particular stimulus that evokes the avoidance response may not be effective under a new spectral distribution caused by ozone depletion. A reduction in the ozone layer would not increase UV-A and visible radiation. If the avoidance response is linked to high levels of visible irradiance, for example, the avoidance response would not be activated by increased depletion. It seems likely that only a sensory system that directly detects irradiance in the UV-B range and does so before irreparable damage occurs will afford surface-living organisms a safety potential.

Experiments by Damkaer and Dey (1982) suggest that shrimp possess no organ to sense UV-B. They observed differences in behavior between irradiated and non-irradiated shrimp larvae (Pandalus platyceros) or copepods (Epilabidocera longipedata). The irradiated specimens maintained their near-surface positions in visible light (as did the controls) until the time of decreased activity and death at about four days. Adult euphausiids, on the other hand, seemed to avoid this same level of visible light (with or without UV-B radiation) but were attracted to low levels of visible light with or without extremely high levels of UV-B radiation (Damkaer and Dey 1983). These investigators also found that the zoea stage of the shore crab (Hemigrapsus nudus) is extremely attracted to strong visible light. No difference in response-time (seconds) was noted between control larvae and larvae receiving lethal UV-B doses until decreased activity and death of the irradiated larvae at about 10 days. There was no apparent reluctance of the crab larvae to swim toward and to hold themselves within a zone producing a lethal dose of UV-B radiation.

These experiments suggested that there were no differences in behavior between UV-B irradiated organisms and the control organisms until lethal doses of radiation reduced their activity. This is not considered to be an active response to UV radiation, and it would have no value in avoidance. The apparent inability to perceive potential danger from UV-B exposure occurred at dose-rates well above established laboratory thresholds and at exposures to total doses that, in most experiments, surpassed lethal total-dose thresholds for similar animals (Damkaer et al. 1980). If the animals tested in these studies possess a behavioral mechanisms for protection from dangerous levels of irradiation it seems unlikely that it could be based on the direct sensing of UV-B radiation. The zooplankton tested seemed to be attracted to wavelengths of radiation longer than those in the UV-B range, and the additional exposure to high levels of UV-B irradiance did not alter their short-term behavior. Within the limits examined, these animals generally positioned themselves as near the light source as possible. That organisms continue to seek out a strong light source even while doomed from past UV-B exposures demonstrates not only the strength of this photo-positive response but also, probably, their inability to independently discriminate between safe and dangerous levels of UV-B irradiance. Since reductions in atmospheric ozone would allow more incident UV-B (and create no change in visible irradiation), the currently evolved response of some organisms to future solar irradiation is unlikely to be optimal.

Effects of UV-B on Biological Diversity and Subsequent Impact on Food Web

Almost all life ultimately depends on plants, and in the ocean most plant life is planktonic (phytoplankton). To function, plants must receive sunlight, and plants have evolved to require (and tolerate) more or less sunlight, depending on the species. For plants, their balance with current levels of solar radiation is therefore critical. Ultimately, animal life exists because it obtains fixed chemical energy from plants. The interaction of life forms in obtaining energy makes them interdependent, forming food chains, or more frequent complex food webs. Animals feeding directly on plants also have evolved receptor mechanisms for solar light. These adaptations typically involved movements and secondary responses to visually feeding predators.

Even if UV-B caused an adverse effect in only a few marine organisms, others are likely to be affected. There have been several studies indicating that reduced UV irradiation leads to increased productivity in phytoplankton. Only a very few experiments have subjected phytoplankton to reasonable enhancements of UV radiation. Some of these experiments (e.g., Thomson, Worrest, and Van Dyke 1980) suggest that phytoplankton have resistance to above-ambient UV levels. Also, Worrest (1982) cites a study by Wolniakowski in which growth rates of a flagellated phytoplankton, initially depressed by exposure to enhanced UV radiation, returned to the base rate after a few days. Finally, in field observations, Hobson and Hartley (1983) found that summer UV levels did not depress phytoplankton photosynthesis. This is not unexpected since it may have resulted from a combination of seasonal succession of UV-resistant species and adaptation to increased UV radiation.

The most likely direct negative effects of higher UV radiation on phytoplankton would be a change in community composition (Worrest et al. 1981); primary production may not suffer any decrease. The species composition that follows is likely to have differences in size and/or nutritional value. The impact of this kind of change, at the lowest level of the food web, could be marked and severe. One quite likely effect is a loss of diversity. Diversity is associated with stability in ecosystems, allowing alternate routes or choices within food webs. In nature, stresses are often best measured not by changes in productivity or population size, but by changes in species diversity. With loss of species, an ecosystem loses some of its natural resiliency and flexibility. Other "shocks" to the system become potentially more damaging. Unfortunately, our knowledge base is insufficient to make any definitive assessments. All that can be done is to argue by analogy. Experience has shown that a loss in diversity leads to instability in ecosystems, which can cascade to create significant change.

EFFECTS ON ICHTHYOPLANKTON (FISHERIES)

As explained with invertebrate zooplankters, regardless of the cellular-level responses to UV irradiation, it is usually noted that up to some UV level there is no apparent effect on ichthyoplankton. At greater

doses either the repair systems themselves may become inactivated by the radiation, or the damage to the general tissues is beyond the capacity of the repair systems. To be effective, these threshold levels probably must be exceeded during several consecutive days. The apparent UV thresholds are near current-incident UV levels. In one experiment the thresholds for all groups appeared to be above the present median solar incident UV levels at the test location, at least until late in the time span of natural occurrence near the surface. An ozone depletion of 20% would significantly shorten this season. The effects of this shortening on annual productivity is not known. Whether or not the population could endure with a drastically reduced time of near-surface occurrence is not known. Success of any year-class depends on the timing of a great number of other events in addition to levels of exposure to UV-B radiation.

Hunter, Kaupp, and Taylor (1979) exposed anchovy eggs and larvae and mackerel larvae to high doses of UV-B radiation in small closed containers. They reported that the dose that killed one-half of the test organisms (LD_{50}) is clearly not an acceptable criterion for biological effect. They recognized that "it seems unlikely that any of the damaged survivors, regardless of dosage, would be able to feed successfully." Hunter, Kaupp, and Taylor (1982) recognized that "none of the larvae surviving LD_{50} doses at age 4-days would survive to age 12-days."

Apart from the fact that laboratory experiments can never duplicate natural responses, the subsequent experiments on UV effects by Hunter, Kaupp, and Taylor (1981, 1982) involved some additional shortcomings that must be kept in mind while evaluating the results. First, it is often difficult to maintain test organisms in good condition. For some common animals, it is not yet possible to keep them alive in the laboratory, much less experiment meaningfully with them. For most of the other animals the laboratory conditions themselves exert a powerful negative influence, so that a true and isolated measurement of UV stress is not attainable. This can be seen in the high 12-day mortalities (average 61%) in the controls for the anchovy larvae. These larvae were kept in small closed containers, a poor but perhaps necessary approximation of the open ocean. There can be no doubt that these larvae were under significant stress even without UV radiation. Hunter et al. refer to wide fluctuations in temperature in the solar-light experiments; oxygen was probably also a limiting factor. Perhaps a flow-through seawater system or another species of fish larvae would eliminate this serious problem. As in some agricultural field tests (Teramura 1986), actual field tolerance of UV radiation is greater than that determined in the laboratory.

The second confounding factor in the experimental design of Hunter, Kaupp, and Taylor is that the tests apparently ended at Day 12. Hunter, Kaupp, and Taylor (1982) reported that at age 20-days, anchovy larvae begin daily vertical migrations. This larval movement into deeper water during the day, and into shallow water at night, would coincidentally avoid significant UV-B exposure. After 20-days survival, then, the gauntlet of UV exposure would have been successfully negotiated. A more meaningful experimental

criterion would have been survival to at least age 20-days. However, from the reported mortality rates, perhaps even the control larvae would not have survived this long.

Related to the test endpoint is the third major problem in the interpretation of these experiments. When experiments are conducted to an appropriate developmental endpoint (e.g., to the age when larvae avoid the daytime near-surface layer), it is usually apparent that below a certain irradiation level the organisms are not seriously affected. On the other hand, beyond that irradiation level there seems to be fairly general and irreversible damage. In experiments with planktonic invertebrates (Damkaer et al. 1980, 1981, 1983), it can be seen that a very great range of UV-B dose-rates and doses could lead to the same LD_{50}; this depends solely on the time chosen for the observation. In every case, however, at the developmental endpoint, the groups that had exhibited an LD_{50} were essentially all dead.

Hunter, Kaupp, and Taylor (1982) stated that none of the larvae surviving the reported LD_{50} daily dose to age 4-days would have survived to age 12-days. The LD_{50} criterion, then, was clearly inappropriate. Hunter, Kaupp, and Taylor admit that their LD_{50} daily dose to age 12-days may also have underestimated the effect of UV on survival. In their 1981 study they predicted the effects on natural populations by assuming that all organisms receiving less that this LD_{50} value will survive, but this is not an appropriate use of the LD_{50}. To the extent that organisms receiving just under LD_{50} doses will probably all die, and that LD_{50} values here suggest only that a lower dose might also lead to death, the lethal daily dose estimated by Hunter, Kaupp and Taylor could be an underestimate of UV effects. However, in a reinterpretation of the data considering threshold dose-rate and dose effects (Damkaer, personal communication), the critical daily dose can be estimated.

Hunter, Kaupp, and Taylor (1981) noted that the lengths of anchovy larvae in all UV treatments, even with the lowest dose, were significantly less than the length of control larvae. Length, then, could be a sensitive criterion for UV-B effects, and this, and other criteria, should be explored. A judgement would be necessary as to what diminished length the individual larvae could tolerate before there is a significant effect on survival.

Hunter, Kaupp, and Taylor (1982) realized that information is required on seasonal abundance and vertical distributions of anchovy larvae, vertical mixing, and penetration of UV-B radiation into anchovy-populated seawater before effects of current and increased levels of UV-B radiation can be predicted. There are fairly good data on seasonal abundances and vertical distributions of anchovy larvae, as well as good calculations on average UV-B penetration into known water types. To detect a mortality due to UV-B radiation, against the high natural mortality, would be a difficult problem. But this is not the same as predicting the effect due to this factor alone. For this, mortality rates from other causes are not required because we are

concerned only with how UV-B radiation would act on those that survive the other perils of their existence.

Hunter, Kaupp, and Taylor assumed that all larvae at depths where UV-B exceeds the LD_{50} daily dose will die. They also assumed a static, unmixed vertical distribution. Finally, these authors provided data that indicated that anchovy larvae off southern California are typically centered in moderately productive ocean waters that have about 0.5 mg Chl *a* m^{-3}. Baker, Smith, and Green (1981) calculated surface DNA-effective UV-B irradiance levels expected for this area, and Smith and Baker (1979) calculated the penetration of UV-B radiation into moderately productive ocean water.

While Hunter, Kaupp, and Taylor calculated that 13.4% of the annual larval anchovy population would die (presumably after 12-day exposures during certain critical times) in a static situation, an analysis by Damkaer (personal communication) indicates that somewhat more would die (14.7%) and in shorter times (4 to 10 days), if there were no vertical mixing. However, static situations are not probable because of physical vertical mixing of water (through the action of wind, density currents, and tides) and the active and passive movement of fish larvae. If some vertical mixing did not offer relief from the effects of UV-B radiation, it is likely that there would be no anchovy larvae above a few meters depth; this is clearly not the case. Probably the surface mixed layer extends to at least 25 m from December to February (Sverdrup, Johnson, and Fleming 1942). A mixed layer of at least 10 m can be assumed for the rest of the year.

For the mixed situation in February, the larvae would spend less than one hour each day in the surface 1-m layer, resulting in exposures where the threshold doses would never be reached in the larval stage. In the February model, with mixing to 25 m, there would have to be a six-fold increase in incident UV-B radiation before threshold doses would be reached. For the months March-October, with mixing to 10 m, the threshold doses are also not reached within the 20 days available before the larvae become vertically migrating. Only in April and August, with the highest surface irradiances, are these doses approached within this available time (~ 22 days).

For March-October, with 10-m mixing, a 10% increase in incident UV-B radiation would still not lead to threshold doses in less than 20 days. With a 20% increase in incident UV-B radiation (a result of about a 9% decrease in the atmospheric ozone column) the depth of the threshold dose-rate is increased. With the 20% increase in incident irradiance, in the dose/dose-rate threshold model, all of the anchovy larvae within the 10-m mixed layer in April and August would be killed, the threshold doses being achieved after 15 days.

Apparently at all months about 36% of the larval anchovy population is above 10 m (Hunter, Kaupp, and Taylor 1981). The greatest numbers of anchovy larvae are found in April (20% of annual population), so that 7.2% of the annual population would be eliminated with a 9% ozone decrease. Only 1.9% of

the annual population is present in August, so the loss of those above 10 m would amount to 0.7% of the annual population. The total predicted loss, then, due to a 9% decrease in total ozone column, would be about 8% of the larval anchovy population (Exhibit 9).

In the static model of Hunter, Kaupp, and Taylor 13.4% of the larval anchovy population dies through the current effects of solar UV-B radiation. They predict only an additional 4.2% decrease if UV-B levels increase 60+%. Presumably, in that model, a 20% increase in UV-B irradiance would lead to only a 1.4% additional loss, so the total loss under a 20% increase would be 14.8%. In contrast, the dose/dose-rate threshold and vertical mixing model presented by Damkaer would predict no current solar UV-B damage, but an 8% total larval population loss at a 20% increase in UV-B irradiance (10% depletion) (Exhibit 9). Because of complex interactions between mixing-depths, vertical distribution of larval anchovy, seasonal changes in solar irradiance and the penetration of UV into seawater, and seasonal change in anchovy abundance, there is not a linear relationship between mixing-depth and predicted annual loss of anchovy larvae. However, within the range of values represented in Exhibit 9, there is a maximum predicted loss at current UV levels with a 5-m mixed layer, and a maximum predicted loss at a 60% increase in UV-B irradiance with a 10-m mixed layer.

Perhaps changes in lower levels of the food web, if they occur with higher UV-B are likely to be more important than the direct effects upon the fisheries changes in the composition of primary production of organic biomass could after the mortality experienced by larval fish. And perhaps there would be a synergistic effect on mortality. Some fish die from direct exposure, some die from lack of food, and some die from the combination of a reduction in food and the weakening from exposure (or are weakened and become outcompeted by other fauna for limited food reserves).

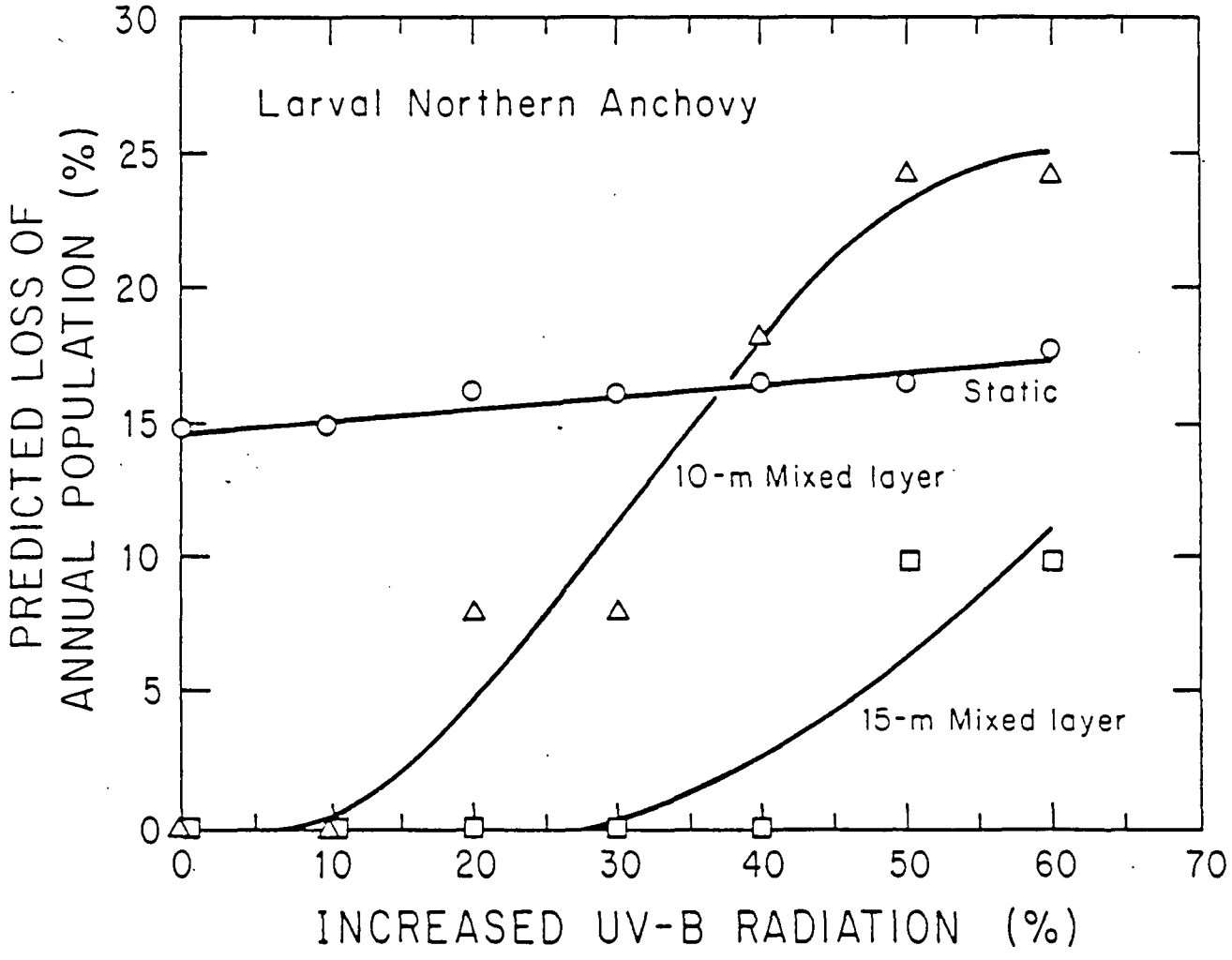
The impact on marine fisheries as a food supply to humans would be significant if the species of phyto- and zooplankton that adapted to enhanced UV-B radiation were of different nutritional value (i.e., if they altered growth and fecundity of the consumers or different accessibility to humans). If the indirect impact of suppression upon consumers were linear, a 5% reduction of primary production would result in a 5% reduction in fish production (Exhibit 12-9). A question still under investigation is whether the trophodynamic relationships might be nonlinear. For example, there may be an amplification factor involved that results in a relatively greater impact at higher trophic levels than at the primary-producer level.

Levels of UV-B irradiance range latitudinally, with the highest exposures in the tropics. The current difference between the extremes of exposures is about 3- to 6-fold, but biota are presently adapted to the levels that are normally experienced at their current locations. An effect of ozone depletion of ecological significance is that a 10% decrease in the ozone concentration would produce a 25% increase of biologically effective ultraviolet radiation (DNA, Plant; Exhibit 2), which would correspond to migrating over 30° toward the equator. Continued investigations concerning

the range of natural ecological uncertainties, which are much larger than the uncertainties in the particular photobiological effects, will be required to permit assessment of the possible consequences for the many complex ecological interactions as well as for the productivity of fisheries.

EXHIBIT 12-9

Effect of Increased Levels of Solar UV-B Radiation on the Predicted Loss of Larval Northern Anchovy from Annual Populations, Considering the Dose/Dose-Rate Threshold and Three Vertical Mixing Models



Based on data of Hunter, Kaupp, and Taylor 1982.

CONCLUSIONS

At a minimum, evidence exists that both small and large changes in the stratospheric ozone layer would affect aquatic ecosystems. It will be very difficult to determine the biological effects of small changes in UV-B irradiance at the ocean surface because aquatic ecosystems have a huge physical and biological "noise" level. Storms, clouds, and global currents make dramatic changes in the status of life in the ocean that are currently unpredictable. One should not, however, conclude that changes that are lost in the "noise" of the system are automatically insignificant simply because we cannot measure and define exactly what is taking place. Changes that occur because of small systemic events, such as higher UV-B, could accumulate in time to produce much more significant change in aquatic organisms that appear very sensitive to current UV-B radiation. The possibility thus exists that changes outside the historical range of UV-B could have implications far greater than we are currently able to predict with confidence. We ought not to mistake our ignorance about what will happen if UV-B increases with the conclusion that all is well. At this time we can place almost no limit on what will happen. An increase in UV-B might have small effects, or it might be much more significant.

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CHAPTER 13

EFFECTS OF UV-B ON POLYMERS

SUMMARY

Several polymers (and largely the impurities present in the polymers) readily absorb the ultraviolet (UV) radiation. One effect of increased UV-B radiation, resulting from possible future depletion of the ozone layer in the stratosphere, will be accelerated degradation of the polymeric materials. Energies associated with the UV radiation are large enough to initiate reactions in polymers, which lead to their degradation by affecting their mechanical and optical properties and thus reducing their service life. Several methods are used to stabilize the polymers to maintain a useful service life in their various important applications.

While much is known about the physical processes of degradation, little research has focused to date on the potential costs and response to such degradation. Initial studies suggest that one likely response to increased UV-B induced damage is to increase the amount of light stabilizer in the polymers. Due to lack of relevant data, only approximate estimation methods are available to determine the extent of light-induced damage and the degree of stabilization required to minimize it. The effect of increased UV-B radiation is manifested in increased costs of production, including raw material costs, energy costs, and maintenance costs.

FINDINGS

1. DEPLETION OF THE OZONE LAYER (STRATOSHPERE) WILL INCREASE THE AMOUNT OF ULTRAVIOLET-B (290-315 nm) RADIATION REACHING THE EARTH'S SURFACE.

2. INCREASED UV-B RADIATION WILL ACCELERATE THE DEGRADATION OF POLYMERS.

   2a. Several commercial polymers (e.g., polyethylene, polypropylene, poly(vinylchloride)), although theoretically UV transparent, contain sufficient chromophore impurities that absorb significant amounts of light in the UV-B region of the spectrum. Other polymers (e.g., polycarbonate) have structural features in the molecule which result in strong UV-B light absorption.

   2b. Several polymers have important outdoor applications (e.g., building industry -- siding, window profiles, glazing; packaging -- film, containers; housewares and toys; and paints and protective coatings). Such polymers are likely to be exposed to significant amounts of UV-B radiation.

   2c. Absorption of UV-B radiation in polymers causes photo-induced reactions and alters important mechanical and optical properties of the polymers (e.g., yellowing, brittleness) and thus degrade (reduce the useful life) the polymers.

3. UV-STABILIZERS ARE USED IN POLYMERS FOR PROTECTION AGINAST UV RADIATION. INCREASED UV-B RADIATION MAY REQUIRE INCREASED USAGE OF STABILIZERS TO MAINTAIN A PRODUCT'S USEFUL LIFE.

   3a. Increased amounts of stabilizers might adversely affect the processing and use properties of some polymers (e.g., hardness, thermal conductivity, flow characteristics). For example, increased amounts of titanium dioxide in poly(vinylchloride) might affect its processing properties.

   3b. Changes in the amount of stabilizer (and other additives) would require development of new formulations.

4. DAMAGES FOR GLOBAL STRATOSPHERIC AND CLIMATE CHANGE ARE DIFFICULT TO ESTIMATE.

   4a. Due to lack of relevant experimental data, only approximate estimation methods are available to determine the extent of light-induced damage.

   4b. Depending upon the chemical nature of polymer, the components of the compound and the weathering factors, both temperature and humidity tend to increase the rate of degradation.

5. INCREASED UV-B DUE TO OZONE DEPLETION WILL HAVE ADVERSE ECONOMIC EFFECTS.

5a. Change in polymer processing properties can result in more equipment shutdown, higher maintenance costs, increased utility costs.

5b. Increased operating costs and material costs (stabilizers, lubricants, and other additives) would have adverse economic impact on the polymer/plastic industry and related industries.

5c. In a case study, based on preliminary methodology, for a given scenario of ozone depletion (26% depletion by 2075), undiscounted, cumulative (year 1984-2075) economic damage for poly(vinylchloride) is estimated at $4.7 billion (USA only). Due to the lack of data, damage in other polymers is not assessed. Like PVC, it is expected to be significant.

PHOTODEGRADATION OF POLYMERS

Structure of Polymers

Macromolecules in the polymers are composed of several monomeric units joined by chemical bonds to each other. The monomeric units contain chemical bonds that are either in the main chain of the macromolecule or connect various atoms or side groups to it. Side groups, if present, contain additional chemical bonds. All of these bonds may be reaction sites in polymer degradation, and various energy sources may be effective in supplying the energy necessary to break the bonds. The dissociation energies of chemical bonds in common polymers range from about 65 kcal/mole (C-Cl) to 108 kcal/mole (C-F) with carbon-carbon bonds in the middle (75-85 kcal/mole). The most important types of energy that cause polymer degradation are heat, mechanical energy, and radiation. Thermal and mechanical degradation of polymers may occur during thermomechanical processing. The most common form of radiant energy which causes degradation is that of the UV component of sunlight; the energy of a photon with wavelength λ = 300 nm is about 95 kcal/mole, which is higher than most bond dissociation energies in polymers, and therefore capable of breaking these bonds.

Wavelengths of UV radiation at which various commercial polymers have maximum sensitivity and their corresponding photon energies are given in Exhibit 13-1 below.

EXHIBIT 13-1

Wavelengths of UV Radiation and Polymers With Maximum Sensitivity and Corresponding Photon Energies

Polymer	Wavelength (nm)	Energy kcal/mole
Styrene-acrylonitrile copolymer	290,325	99,88
Polycarbonate	295,345	97,83
Polyethylene	300	96
Polystyrene	318	90
Polyvinylchloride	320	89
Polyester	325	88
Vinyl chloride-vinyl acetate copolymer	327,364	87,79
Polypropylene	370	77

Source: Kelen, T., Polymer Degradation, Van Nostrand Reinhold Company, Inc., New York, 1983.

Light Absorption

A prerequisite for light-induced damage in materials is their ability to absorb the radiation (this observation is sometimes referred to as the Grotthus-Draper Law of Photochemistry) (Andrady 1986). Very few materials absorb light at all wavelengths across the spectrum. Pure samples of polyethylene, polypropylene, and poly(vinylchloride), for instance, are theoretically UV transparent and are not expected to absorb light in the range 290-315 nm, the UV-B region of the spectrum. However, completely "pure" polymers do not exist. During synthesis, processing, and storage, various amounts of carbonyl and hydroperoxy groups accumulate in the commercial polymers. These chromophores readily absorb UV light in the 290-315 nm wavelengths. Initiation of degradation consists mainly of the decomposition of these chromophores.

Photophysical and Photochemical Processes

Degradation of the polymers involves a series of photophysical and photochemical processes. The physical processes involved in photodegradation include absorption of light by the material, electronic excitation of the molecules, and deactivation energy transfer to some acceptor. When the lifetime of the excited state is sufficiently long, the species can participate in various chemical transformations. This is especially true when the photoinduced degradation reactions are carried out in the presence of oxygen (see equations 1-4).

$$\text{Polymer (RH)} \xrightarrow{\text{light}} \text{polymer excited state (RH*)} \quad (1)$$

$$\text{RH*} \xrightarrow{\text{degradation}} \text{polymer radicals or broken (scission) chain end (R•)} \quad (2)$$

$$\text{R•} + \text{air} \longrightarrow \text{polymer peroxy radicals (RO2•)} \quad (3)$$

$$\text{RO2•} + \text{RH} \xrightarrow{\text{light}} \text{ROOH} + \text{R•} \text{ (further polymer degradation)} \quad (4)$$

As mentioned earlier, hydroperoxy and carbonyl groups are the two most common impurities present in the polymers. In the case of hydroperoxy groups (ROOH), the energy of UV light is sufficient to cause both of the following decompositions (Kelen 1983):

$$\text{ROOH} \xrightarrow{\text{light}} \text{RO•} + \text{•OH} \quad (\sim 42 \text{ kcal/mole}) \quad (5)$$

$$\text{ROOH} \xrightarrow{\text{light}} \text{R•} + \text{•OOH} \quad (\sim 70 \text{ kcal/mole}) \quad (6)$$

Dissociation of the O-H bond is less facile:

$$ROOH \xrightarrow{light} ROO\bullet + \bullet H \quad (\sim 90 \text{ kcal/mole}) \qquad (7)$$

Because of the low bond dissociation energy (42 kcal/mole), decomposition according to reaction (5) is predominant in photooxidation of polymers containing hydroperoxy groups.

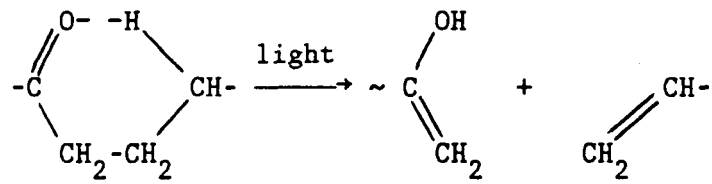
The carbonyl group absorbs UV light readily and hence is easily excited. The excited carbonyl group decomposes via Norrish reactions of Types I, II, and III (Kelen 1983). The Norrish-I reaction (8) is a radical cleavage of the bond between the carbonyl group and the α-carbon atom (α - scission).

$$-CH_2-\overset{\overset{O}{\|}}{C}-CH_2- \xrightarrow{light} -CH_2-\overset{\overset{O}{\|}}{C}\bullet + \bullet CH_2 \qquad (8)$$

$$\downarrow$$

$$CH_2\bullet + CO$$

The Norrish-II reaction (9) is a nonradical scission that occurs through the formation of a six-membered cyclic intermediate to form an olefin and an alcohol or ketone.



(9)

The Norrish-III reaction (10) is also a nonradical chain scission and leads to the formation of an olefin and an aldehyde.

$$\begin{matrix} O\ CH_3 & & O & CH_2 \\ \| \ | & & \| & \| \\ -C-CH- & \xrightarrow{light} & -CH & + \ CH- \end{matrix} \qquad (10)$$

In the case of PVC, the photolytic scission takes place as follows:

$$-CH_2-\underset{\underset{Cl}{\|}}{CH}- \xrightarrow{light} -CH_2-\overset{\bullet}{C}H- + \bullet Cl \qquad (11)$$

POLYMERS IN OUTDOOR USES AND POTENTIAL DEGRADATION

A variety of polymeric materials are used in applications where the material is routinely exposed to solar radiation. These include mainly the use of plastics in the building industry and in paints/coatings. In other applications such as packaging, outdoor furnishing, housewares, and toys, exposure occurs from extended to more limited, possibly intermittent, periods of time.

According to recent estimates, of the 45,506 million pounds of U.S. plastic sales in 1985, the building industry above accounted for 9,428 million pounds, 21% of the total consumption. Packaging applications were the largest market consuming 10,846 million pounds (Horst, et al. 1986).

However, bulk of the consumption represents that of a limited number of different plastics. These include the so-called "commodity" plastics and a few other types selected on the basis of their unique physical-chemical properties. Exhibit 13-2 gives a breakdown of the types of plastics used in each application along with the likelihood of exposure to sunlight in normal use. Some plastics/products, not experiencing direct sunlight, can still be exposed to UV light since the UV light is easily reflected and scattered by terrain and buildings, resulting in significant irradiation levels at surfaces illuminated only by diffused or reflected light (Andrady 1986). Thus, vulnerability to degradation is a function of a polymer's normal lifetime and the percent of time it is exposed to direct or indirect sunlight.

Light-Induced Damage

Exposure to light and several physical-chemical processes (described earlier) occurring simultaneously result in deterioration of polymeric materials.

Properties most likely to be affected by the degradation of the polymer include both mechanical and optical properties. Mechanical properties such as tensile strength, elongation, modulus, and impact strength can all be affected by UV degradation of polymeric materials. Optical properties that have been observed to change upon exposure to UV radiation include transparency, color, chalking or cracking of surfaces, and yellowing. These effects are most noticeable on the surface of plastic materials because of the limited penetration of UV light through the materials. Thus, the material may show cosmetic changes in appearance long before any degradation in its bulk mechanical property is noted.

The mechanical weakening of plastic material occurs because of a reduction in the molecular weight of the polymer. Essentially all of the mechanical properties of polymers are related to the molecular weight of the material. Thus, for every bond in the polymer molecule that breaks due to UV radiation, the average molecular weight will be reduced, and the corresponding mechanical properties will be reduced in some proportion to this molecular weight reduction (Andrady 1986).

EXHIBIT 13-2

Plastics Used in Applications Where Exposure of the Material to Sunlight Might be Expected

Type of Plastic	Application	Usage 1000 metric tons	Exposure
	Building Industry		
Poly(vinylchloride) (rigid)	siding	410	high
	door/window	284	high
	other	33	high
	conduit	493	moderate
	Irrig. pipe	247	high
	other pipe	1912	low
Poly(vinylchloride) (plasticized)	roofing	22	high
	liners	25	moderate
	wire-cable	410	N/A
	weatherstrip	38	moderate
	garden hose	48	high
Unsaturated Polyester	glazing	41	high
	panels/siding	122	high
	pipe	237	low
Polycarbonate	glazing	90	high
	fixtures	9	low
Acrylic	glazing	84	high
	Packaging		
Polyethylene	film	696	N/A
	containers	2990	N/A
Polypropylene	film	375	N/A
	containers	325	N/A
Thermoplastic Polyester	containers	635	N/A

EXHIBIT 13-2
(Continued)

Type of Plastic	Application	Usage 1000 metric tons	Exposure
	Housewares and Toys		
Polyethylene		954	N/A
Polystyrene		378	N/A
Polypropylene		176	N/A

Note: Exhibit 2 is based on 1985 estimates as reported in Modern Plastics, (1986) 63(1): 69.

N/A indicates exposure information not available. However, based on the application, it is expected to be low to moderate.

Discoloration of polymeric materials is also related to bond breaking, but in these cases, bonds that are broken may have little effect on mechanical properties. The best example of a polymer discoloring by yellowing is in transparent flexible polyvinyl chloride (PVC) window materials used for rear windows of convertible automobiles. The PVC darkens from weathering because of impurities in the resin initiating a dehydrochlorination reaction as outlined below:

$$-CH_2-\underset{\displaystyle Cl}{\underset{|}{CH}}- \xrightarrow[\text{impurities}]{\text{light}} -\overset{\displaystyle H}{\overset{|}{C}}=CH- + HCl \qquad (12)$$

The color is caused by the multiple double bonds (~9 repeat units in sequence) created between the carbon atoms, resulting in a polymer that absorbs the light in the visible region of the spectrum and thus appears yellow. Often this discoloration is evident long before mechanical properties of the polymer material begin to change. This, again, is because mechanical properties are related to the molecular weight of the polymer more than to the chemical structure.

Other changes in polymeric materials from UV exposure, similar to yellowing, also occur mostly at the surface. Such changes as surface cracking, chalking or crazing (i.e., when a clear surface becomes translucent because of formation of microcracks) are all evident in polyolefins, polycarbonates, vinyl, acrylic, and styrenic polymers when exposed to UV. These surface effects drastically reduce the life expectancy of materials that require maximum transmission of visible light, such as Plexiglas and greenhouse windows, solar energy collectors, and streetlamp globes.

Several different modes of damage occur concurrently. However, depending on the application of the polymer, one or more of such properties may be important. Exhibit 13-3 gives the critical mode of damage and one or more secondary modes of damage identified for selected applications of polymers. The critical mode of damage is defined as the degree of damage which renders the material "unacceptable" at the shortest duration of exposre, thus reducing the polymer's service life.

Plastic Compounds

Polymer resins are rarely, if ever, used by themselves in any application. To optimize the properties demanded by a given application and to enable the product to be easily fabricated, a number of other "ingredients" are added to the polymer to obtain a "compound." The nature of the non-polymer ingredients in a compound and the ratio in which they are mixed will depend on the processing technique, the grade of polymer, and the desired properties in the end product.

EXHIBIT 13-3

Modes of Damage Experienced by Polymers Used in Outdoor Applications

Polymer	Application	Damage
Poly(vinylchloride)	siding, window frames	[+ yellowing] + chalking - impact properties - tensile properties + surface distortion
	pipes	[- burst pressure] - impact properties + yellowing
	roofing materials	[+ brittleness]
	automobile upholstery	+ discoloration
Unsaturated polyethylene	outdoor surfaces	[+ surface erosion] + discoloration - strength
Polyethylene/polypropylene	irrigation pipe, outdoor furniture	[+ brittleness] - tensile properties
	synthetic turf, stadium seats, packaging	- electrical properties
Polycarbonates	glazing material	[+ yellowing] + loss of transparency

\+ = increase
\- = decrease
[ ] = parentheses indicate critical mode of damage

Source: Andrady, Anthony, "Analysis of Technical Issues Related to the Effect of UV-B on Polymers," Research Triangle Institure, Rsearch Triangle Park, N.C., March 1986.

An example of a rigid PVC siding compound is given in Exhibit 13-4.

EXHIBIT 13-4

PVC Siding Compound Composition

Component	Concentration (pph)*
PVC resin	100
Heat stabilizer	1.5
Lubricant	1
Titanium dioxide	12
Impact modifier	6
Calcium carbonate	3
Processing aid	1

* pph = parts per hundred parts resin

Source: Andrady, Anthony, "Analysis of Technical Issues Related to the Effect of UV-B on Polymers," Research Triangle Institure, Rsearch Triangle Park, N.C., March 1986.

The fabrication of the siding involves feeding the melted compound through a slit die, a process where the polymer is heated and sheared. Therefore a heat stabilizer is usually used in the compound. The lubricant prevents sticking of melt to metal parts in the processing machinery. A suitable rubber is added as an impact modifier to reduce the brittleness of PVC resin. The processing aid is added to improve the melt processing characteristics while the chalk is added to cheapen the compound. Titanium dioxide is the light stabilizer. As explained in the previous sections, several polymers when exposed to sunlight (even under current UV conditions), would degrade, some of them rapidly. Therefore, stabilizers such as titanium dioxide are used to reduce the damage from exposure to light. Any depletion in the stratospheric ozone with potential increase in the UV-B radiation would accelerate the degradation process.

Light Stabilizers in Polymer Products

Light stability in polymeric materials is achieved by the inclusion of an appropriate "light stabilizer" in a polymer compound. Light stabilizers are substances that absorb the light in the UV-B range of the spectrum very

efficiently. When incorporated into a transparent polymer matrix, the stabilizer molecules compete with the polymer molecules for the available light. Light absorbed by the UV-absorber molecules is harmlessly dissipated and does not lead to any damage of the polymer matrix. For best results the light absorber should be soluble in polymer, absorb light in the UV-B range of the spectrum, and have a very high molar extinction coefficient (so that it is effective at relatively low concentrations).

Three different methods are used to achieve light stability. The first method involves use of UV-absorbing compounds such as 2-hydroxy-benzophenones, 2-hydroxyphenylbenzotriazoles, 2-hydroxyphenyl-S-triazines and derivatives of phenyl salicylates (Heller 1969). These compounds absorb and dissipate the light without degrading the polymer.

A second approach is to prevent light from reaching the bulk of the polymer (light shielding) by opacification of the matrix. Inclusion of a light absorbing (or light reflecting) insoluble pigment in the matrix prevents the light from penetrating the bulk of the polymer. However, a fraction of surface layer is unprotected and is therefore affected by light. Titanium dioxide absorbs UV-B radiation very effectively and is used in PVC compositions as a light screener. Other pigments used as light screeners include zinc oxide, magnesium oxide, lead carbonate, and barium sulfate. One of the most effective light screeners is carbon black. Used in appropriate concentrations and adequately dispersed in the matrix, the small-sized carbon black particles impart excellent light stability in polyethylene formulations. Exhibit 13-5 gives the UV screening effectiveness of several selected pigments at concentrations of 2% in polypropylene films of 10-mil thickness (Uzelmeir 1970). The screening power is given as the ratio of the weathering stability (lifetime) of the pigmented to unpigmented materials (Andrady 1986). Higher ratio indicates longer stability of the polymer. For example, a polymer that is compatible with carbon black would have a lifetime 12 times longer when pigmented with carbon black and exposed to sunlight than without carbon black. On the other hand, a polymer that is compatible with cadmium yellow would have a lifetime twice as long when pigmented with cadmium yellow (at the same concentration as carbon black in the previous polymer) than without cadmium yellow.

The UV absorbance at specific wavelengths is also given in Exhibit 13-5. This is an indication of how well the pigment is able to absorb the light. Higher absorbance for titanium dioxide (0.92 at 300 nm) than for iron oxide (0.29 at 300 nm) means that at the same pigment concentration (2%), titanium dioxide is able to absorb more light than iron oxide.

The third approach to achieve light stability does not prevent or control the interaction of light with the polymer. Instead, the light stabilizer additive either seeks to deactivate or quench the excited state chromophores before the degradation process occurs, or acts as a free radical scavenger, which mops up the damaging free radicals as soon as they are formed. Complex chelates of nickel, for example, have been used as light stabilizers in polyolefins and are believed to act as quenchers and peroxide decomposers.

EXHIBIT 13-5

UV Screening Effectiveness of Selected Pigments

Pigment	Screening Power	Absorbance 300nm	325nm	350nm
Titanium Dioxide	2.25	0.92	0.97	1.01
Iron Oxide	3.25	0.29	0.30	0.32
Cadmium Yellow	2.0	0.36	0.36	0.36
Chromium Oxide	2.75	0.05	0.06	0.07
Carbon Black (Channel Black)	12.0	1.56	1.56	1.56

Note: Chemical interactions limit the use of some of the above pigments in several polymers. For instance, iron oxide can be effectively used with polypropylene but not with PVC, as it catalyses the photooxidation of the latter polymer (King 1968).

In recent years the use of radical scavenger molecules, particularly the very effective Hindered Amine Light Stabilizers (HALS) has gained wide industrial acceptance. Exhibit 13-6 gives the current U.S. consumption of light stabilizers in various types of plastics.

DAMAGE FUNCTIONS AND RESPONSE TO DAMAGE

Depletion of the stratospheric ozone would change the spectral distribution as well as the solar radiation pattern, with a potential increase in the UV-B radiation striking the earth's surface. The magnitude of such changes on the weathering of plastics and potential damage that might occur are difficult to assess due to lack of experimental data. However, various theoretical approaches are considered to estimate the damage.

There are three possible responses to potential damage from increased UV-B radiation:

1. Add greater amount of light stabilizers to prevent further degradation of polymers.

2. Develop new or improved performance stabilizers to absorb the additional radiation.

3. Switch from polymers to alternative materials (e.g. ceramics, metal, etc.)

EXHIBIT 13-6

Domestic Consumption of Light Stabilizers, 1984-85

Plastic Type	Domestic Consumption (1000 lb) (of UV Stabilizers) 1984	1985
Polyolefins	4290	4600
Polycarbonates	352	400
PVC and related copolymers	171	180
Acrylic	165	170
Polyester	165	171

Note: Above figures do not include light shielders such as titanium dioxide

The relevant statistics for pigments (colorants) are given below.

Pigment	Domestic Consumption (1000 lb) (of Pigments/Colorants) 1984	1985
Titanium Dioxide	271	272
Carbon Black	80	85
Iron Oxide	9	10
Cadmium Compounds	5	5
Chromium Compounds	5	5

Source: Andrady, Anthony, "Analysis of Technical Issues Related to the Effect of UV-B on Polymers," Research Triangle Institute, Research Triangle Park, North Carolina, March 1986.

Several attempts have been made to estimate the increased damage to materials from potential increase in UV-B radiation. Because of the complexity of the analysis, each approach has focused on only the first response. Each approach analyzes how much additional light stabilizer would be required for a given ozone depletion (function of UV light intensity) scenario, to maintain the current level of useful life for various polymers. Thus, each approach has attempted to relate intensity of the UV light and concentration of the light stabilizer in the polymer.

Transparent Polymers (CIAP Approach)

Schultz et al. in a research study (Climatic Impact Assessment Program (CIAP)) funded by the U.S. Department of Transportation were the first to examine the potential adverse effects of the ozone depletion on materials (Schultz et al. 1974). Although their concern was depletion of ozone from SST flights and not from discharge of chlorofluorocarbons, their approach is nonetheless intuitive.

The CIAP approach employed the Beer-Lambert law to calculate approximate increases in UV stabilizer required to offset the effects of increased UV light (Andrady 1986). According to this law, for a homogeneous material with no scattering losses at the surface or in the bulk of the polymer, the monochromatic light absorption by the system is described as follows:

The fraction of incident monochromatic light energy absorbed is proportional to the concentration of the chromophores and to the path length of the light within the substrate.

$$\text{Log } I_t/I_o = -ec\ell \qquad (13)$$

I_t = intensity of the transmitted light

I_o = intensity of the incident light

e = molar extinction coefficient (proportionality constant)

c = molar concentration of the light absorbing moiety

ℓ = thickness of the film

An increase in the incident light intensity will result in a higher light flux in the polymer; therefore, to maintain the same level of protection, the concentration of the UV absorber has to be increased. If an increase in the light intensity by a factor x required a corresponding increase in the UV absorber concentration by a factor y, the Beer-Lambert Law for the new system gives

$$\text{Log}(I_t/I_o \cdot x) = -eyc\ell \quad (14)$$

The two above equations can be combined to give

$$y = \text{Log}(x)/H + 1 \quad (15)$$

where $H = ec\ell$, a constant

This simple equation predicts the variation of the factor increase in the UV absorber concentration, y, with the factor increase in the intensity of light, x. An empirical observation made is that an increase in the UV light intensity by a factor of 1.5 generally requires an increase in the UV absorber concentration by a factor of 2.0.

Based on this empirical observation and the damage function (equation 15), CIAP developed the following relationship:

$$y = 1 + (\log_{10}x)/(\log_{10}1.5) \quad (16)$$

Using the above relationship and using a brash extrapolation of the world market, CIAP developed increased stabilizer requirements for various levels of ozone depletion. A market period of 50 years (1970-2020) was considered in the CIAP study to estimate the future market of plastics. The results are summarized in Exhibit 13-7.

EXHIBIT 13-7

Increased Stabilization Market (1970-2020)

Ozone Depletion Percent	By 1990 million	By 2020 1974 dollars *
50	1,040	5,040
20	370	1,800
10	172	840
5	84	408

* Values at zero discount rate.

Source: Schultz, A., D. Gordon, and W. Hawkins, "Economic and Social Measures of Biologic and Climatic Change," CIAP Monograph 6, September 1974.

Battelle Study

This study was a follow-up to the CIAP study. The basic assumptions remained the same, but all economic market data used for analysis was updated using more current information. Also the different ozone depletion scenario was used in the Battelle study. Exhibit 13-8 below shows the ozone depletion estimates used in the study.

EXHIBIT 13-8

Ozone Depletion Estimates

Year	Ozone Depletion (percent)
1985	0.0
1995	0.15
2005	0.62
2015	1.50
2025	2.84
2035	4.66
2045	7.08
2055	10.41
2065	15.44
2075	26.08

Source: EPA estimates based on emission scenario developed by EPA and modeled by Lawrence Livermore Lab.

For the projection of the polymer demand to the year 2075, Battelle used a simplified assumption that the growth rate ratio of UV stabilized plastics to GDP (Gross Domestic Product) for the United States will remain constant at 1.7 into the 21st century.

Historical U.S. market share of the world plastic market and projection of the U.S. plastic market were used to project the world plastic market through the year 2075.

Battelle used Cutchis' formula to estimate the effect of ozone depletion on the average UV light intensity (Cutchis 1974):

$$\log x = 0.00954D \qquad (17)$$

where x is UV light intensity factor and D is the percentage of ozone depletion.

To describe the relationship between light intensity (x) and the stabilizer concentration (y), Battelle used a mathematical formula developed by Shultz, Garden, and Hawkins (1974).

$$\log x = 0.176\ (y-1) \qquad (18)$$

The Combination of the Cutchis and Shultz formuli, gave the following relationship:

$$y-1 = 0.0542D \qquad (19)$$

This formula was used in the economic impact model.

Exhibit 13-9 gives cumulative added cost for the U.S. and the world in the presence of ozone depletion.

EXHIBIT 13-9

Cumulative Added Cost

Year	Ozone Depletion (percent)	U.S. (1983 $ billion)*	World (1983 $ billion)*
1985	0.0	0.0	0.0
2015	1.50	0.13	0.60
2045	7.08	2.3	14
2075	26.08	19.4	120

* Values at zero discount rate.

Source: Hattery, G., V. McGinniss, and P. Taussig "Costs Associated with Increased Ultraviolet Degradation of Polymers." BATTELLE Columbus Laboratories, Columbus, Ohio, April 1985.

Extension to Filled Polymers (Andrady/Shultz)

While the CIAP approach is sound and simple to use, its applicability is limited to the transparent, homogeneous polymers. With the exception of window glazing, polymers used outdoors are generally opaque and are often stabilized using light screeners. Therefore a parallel model based on light stabilization, applicable to opaque materials (filled polymer), was developed by the Research Triangle Institute (Andrady 1986).

A filled polymer can be described by a homogeneous resin matrix in which essentially spherical filler particles are randomly dispersed. The filler can be a pigment with high light absorptivity, so that all the light reaching the filler particle is absorbed. For simplicity, the random distribution can be described as spheres of radius r, in two dimensions, within a series of layers of thickness 2r (see Exhibit 13-10). If the pigment volume fraction is V, the number of spherical pigment particles within a lamina volume element of $y \cdot y \cdot 2r$ cm^3 is $N = 2ry^2V/(4\pi/3)r^3$.

The fraction of light obliterated in passing through the laminar is the cumulative cross-sectional area of the spheres per unit laminar area, $N\pi r^2/y^2$, which is equal to 3V/2. The surface intensity, I_o, can then be related to I_ℓ, intensity at depth ℓ, as

$$I_\ell = I_o(1-3V/2)^{\ell/2r} \tag{20}$$

For light intensity increased by a factor x and a corresponding increase in the light stabilizer by a factor y, the above equation can be modified as

$$I_\ell = x \cdot I_o(1-3Vy/2)^{\ell/2r} \tag{21}$$

Using the assumption of equal residual fractional intensity at a characteristic depth of ℓ units, it can be shown that

$$\ln x = \frac{\ell}{2r} \cdot \ln((1-3V/2)/(1-3Vy/2)) \tag{22}$$

This equation has the same predictive feature as the CIAP equation but requires a knowledge of ℓ and r for its application.

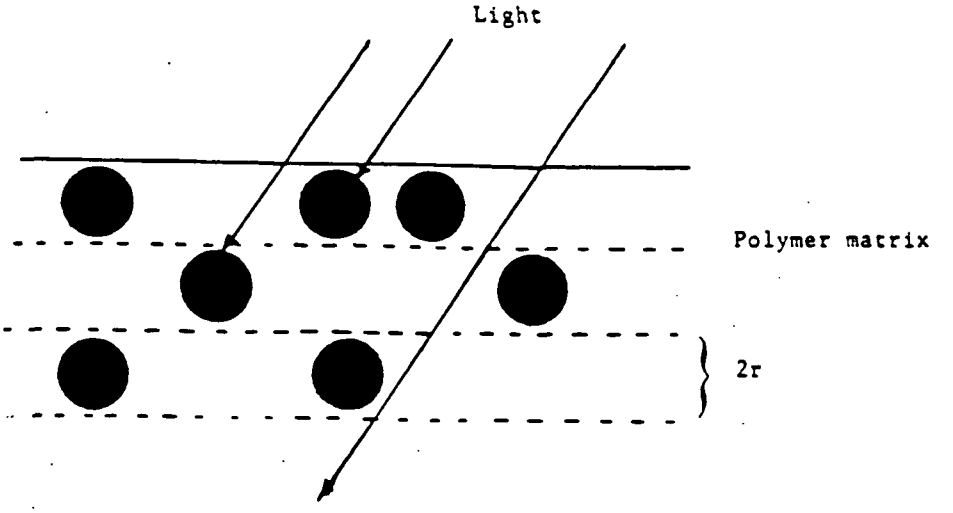
An empirical observation relating x to y is needed in this case as well; however, such experimental data are not presently available for the polymers of interest. No economic damage estimates have been developed using this approach.

Comprehensive Approach (Andrady 1986)

While the two approaches described previously can be used for clear or filled polymers to determine the amount of additional stabilizer requirements

EXHIBIT 13-10

Diagrammatic Representation of the Effect of Pigment/Fillers as Light Shield. (Monodisperse Spherical Filler)



corresponding to an increase in the light intensity, neither approach takes into account the activation spectra for degradation.

The extent (and even the nature) of damage suffered by a polymer on exposure to sunlight is markedly wavelength dependent. More appropriate methodology to obtain the damage function should include (1) source spectral distribution, (2) activation spectra, and (3) the functional relationship between the ozone concentration in the stratosphere and the spectral irradiance.

Atmospheric attenuation of sunlight is a result of several processes. The following are the three main processes: (1) Rayleigh scattering - scattering of light due to gas molecules in the atmosphere; (2) aerosol scattering or Mie scattering caused by fine particles and fluid droplets suspended in the atmosphere; and (3) absorption by ozone. These factors should be considered in calculating the spectral distribution and intensity of light at a given location.

An activation spectra should be available for each specific polymer to assess the damage. A normalized activation spectra then can be used as a continuous weighting function describing the spectral sensitivity of a specific damage process in a given polymer.

The total damage function (index), D, for a polymer exposed to sunlight can be presented as

$$D = \int_{\lambda_0}^{\lambda_1} F(\lambda)H(\lambda)d\lambda \qquad (23)$$

where $F(\lambda)$ = adjusted normalized activation spectrum (ANAS)
$H(\lambda)$ = spectral distribution of sunlight for natural weathering
λ_0, λ_1 = wavelength range of the ANAS

It is assumed that the effect of each wavelength in the range is additive, with no synergism or mutual inhibition.

If $H(\lambda)$ represents an actual measured terrestrial spectrum, then D will be a measure of the damage under current ozone layer conditions, arbitrarily defined as being deteriorated to zero extent.

The above approach of using the actual measured spectrum of the solar irradiance as the source of spectral distribution limits the usefulness of the analysis to specific localities and possibly to certain times of the year (the time when $H(\lambda)$ is fair representation of the solar spectrum). This approach avoids various scattering coefficients and their effects. If we assume that the depletion of the ozone layer has the sole effect on the spectral distribution (with other effects being small), the damage index can be modified (D*), which will relate to damage under reduced ozone level conditions.

This requires a knowledge of the absorption coefficient of ozone. Reliable data have been published for the absorption coefficient, $A(\lambda)$ (Griggs 1968):

$$A(\lambda) = \exp(38.54 - 0.1237\lambda) \qquad (24)$$

The effect of reduced ozone concentration on the terrestrial solar irradiance at a given wavelength can be expressed as

$$I(\lambda,c',\theta) = I_0\exp(-A(\lambda)c'\sec\theta-\tau(\lambda)\sec\theta) \qquad (25)$$

where

$A(\lambda)$ = ozone absorption coefficient, equation (20)

c' = ozone concentration at reduced level

$\sec\theta$ = approximation term for path length of light through the air mass

τ = Rayleigh scattering thickness

θ = solar zenith angle at the location

If we neglect the Rayleigh scattering, the factor increase in the UV radiation due to ozone loss can be given by

$$I(\lambda, c', \theta)/I_o = \exp(A(\lambda)c(1-c'/c)\sec\theta) \quad (26)$$

where (1-c'/c) is the fractional decrease (f) in the ozone concentration.

The damage index under the conditions of ozone layer deterioration can be expressed as

$$D(f,\theta) = \int_{\lambda_0}^{\lambda_1} F(\lambda)H(\lambda)\exp(A(\lambda)\cdot c\cdot f\cdot\sec\theta)d\lambda \quad (27)$$

The seasonal variation of ozone concentrations, the seasonal variation of the solar zenith angle and the diurnal variation of the sun angle (time angle) should be considered while estimating the damage index for different time periods (i.e. daily, annual, seasonal, etc.) For instance, the daily damage index at a given level of ozone depletion is given by

$$D(f,\theta)_{24} = 2\ Z \iint F(\lambda)H(\lambda)\Delta(\lambda)d\lambda dt \quad (28)$$

where $\Delta(\lambda)$ is the factor increase defined in equation (26). The value of ozone concentration c is no longer a constant but is time dependent c(t). Z is a constant term used to convert the time angle to seconds.

Illustration of Comprehensive Approach for Polyvinylchloride: A Case Study

Using the solar spectrum as observed at Miami, Florida, on March 23, 1985, at the solar noon, spectral distribution $H(\lambda)$ was calculated for various wavelengths. An adjusted normalized action spectrum , $F(\lambda)$, was developed using the Miami sunlight spectrum. The integral in equation (28) was evaluated numerically using a computer program. Damage function and the

factor increase in the damage were calculated for several solar zenith angles at different fractional ozone depletion. The results are shown in Exhibit 13-11.

As might be expected, the factor increase in damage is higher at larger solar zenith angles and hence will be greater for higher latitudes. However, the solar irradiation is also lower at these latitudes.

The value of the damage index is very sensitive to (a) the action spectra used in its calculation, (b) the zenith angle of the sun at the location in question, and (c) the thickness of the polymer used.

The thickness of the polymer is an important factor. In the case of a film of the material, a fraction of the incident light is transmitted through it. Often, the transmitted light is deficient in UV radiation. In the case of a thick polymer, the light is totally absorbed but the absorption is limited to a thin surface layer of the polymer. Thus in those modes of damage involving the surface integrity, thick samples undergo as much damage as the thin films. This is true, for instance, where "yellowing" is the critical mode of damage.

Mathtech Study

This is the most recent study of estimating the economic damage from potential depletion of ozone. The study utilizes the comprehensive approach developed by the Research Triangle Institute (Andrady 1986).

According to the comprehensive approach, the total damage D is given by

$$D = \lambda\, H(\lambda)\, \Delta\,(\lambda)\, F(\lambda)\, d\lambda \qquad \text{(equation (23))}$$

The integral might be evaluated numerically with $\Delta(\lambda)=1$ to represent current baseline conditions. However, the lack of activation spectrum for polymers limits the usefulness of the approach. The only action spectrum available in literature relates to transparent PVC films and to type of damage other than yellowing. However, a very approximate estimate might be made using the activation spectra for polyene formation based on Reinisch et al. 1970. Yellowing in PVC is a direct result of the generation of long polyene sequences.

EXHIBIT 13-11

Relative Damage Indices for Yellowing of PVC Under Miami (March 22nd) Conditions, at Different Extents of Ozone Layer Deterioration

Fractional Loss of Ozone	Relative Damage Index* at Different Solar Zenith Angles		
	0°	30°	60°
0	1	1	1
0.05	1.01	1.01	1.02
0.10	1.02	1.02	1.04
0.15	1.03	1.03	1.06
0.20	1.04	1.05	1.09
0.25	1.05	1.06	1.13
0.30	1.06	1.08	1.18
0.35	1.08	1.09	1.25
0.40	1.09	1.11	1.36
0.45	1.11	1.14	1.55
0.50	1.13	1.17	1.93

Note: Solar irradiance values used were for a surface facing south at 45°.

* Relative damage index = D/D_o where D_o is the damage index calculated for current levels of ozone.

Source: Andrady, Anthony, "Analysis of Technical Issues Related to the Effect of UV-B on Polymers," Research Tringle Institute, Research Triangle Park, North Carolina, March 1986.

Based on the data, the damage (D) can be estimated as

$$\mathrm{Ln}(D) = a + g(S) \qquad (29)$$

where a, g = constants
S = stabilizer concentration

For reduced ozone level, this equation can be modified further to the following form:

$$\frac{S'}{S} = \left(\frac{1}{g(S)} \cdot \ln \frac{D'}{D}\right) + 1 \qquad (30)$$

where $\frac{D'}{D}$ = factor increase in damage

$\frac{S'}{S}$ = factor increase in titanium dioxide stabilizer

S', D' = stabilizer concentration and damage at reduced ozone level

This equation allows the determination of the required concentration of the stabilizer of increased extent of damage. Applying the data available for yellowing (damage) of rigid PVC versus various stabilizer concentrations to the above equation, ranges of factor increase in damage and stabilizer concentration are calculated for PVC as illustrated in Exhibit 13-12. The same approach can be extended to other polymers and stabilizers.

To calculate the economic damage, Mathtech has maintained a hypothesis similar to the other studies, which is that producers of the PVC products will change the amount of stabilizer (titanium dioxide) in the resin compound in order to maintain quality and lifetime characteristics of their products. However, the earlier studies (Schwartz, Gordon, and Hawkins 1974; Battelle 1985) did not consider how changes in compound formulation would affect production costs of PVC--which can occur in several ways. First, resource costs will be increased as more titanium dioxide is used per pound of plastic produced. Second, the change in formulation will lead to more frequent replacements of screws and barrels since titanium dioxide is abrasive in nature and its higher concentration will increase wear and tear of the machinery. Third, the change in formulation will lead to increased energy requirements for PVC processing equipment since the melt viscosity of the extrudate increases with titanium dioxide increase and will require higher energy to operate the screw. Also increased viscosity may require more lubricant and processing for successful extrusion.

EXHIBIT 13-12

Estimated Ranges of Factor Increase in Damage and the Factor Increase in Stabilizer Needed to Counter the Change for Yellowing of Rigid PVC Compositions

	Zenith Angle 30° Spring ~ Noontime		Zenith Angle 60° Fall ~ Noontime	
Percent Loss of Ozone	D'/D	S'/S	D'/D	S'/S
0-5	~ 1.01	1.01-1.02	1.01-1.02	1.01-1.05
5-10	1.01-1.02	1.01-1.05	1.03-1.04	1.03-1.09
10-20	1.02-1.05	1.03-1.11	1.04-1.09	1.05-1.20
20-30	1.03-1.08	1.03-1.18	1.07-1.18	1.08-1.38

Note: D'/D = factor increase in damage
S'/S = factor increase in titanium dioxide stabilizer

Zenith angles selected to reflect North American locations.

Source: Horst, R., K. Brown, R. Black, and M. Kianka, "The Economic Impact of Increased UV-B Radiation on Polymer Materials: A Case Study of Rigid PVC," Mathtech, Inc., Princeton, New Jersey, June 1986.

In order to estimate the cumulative damage, demand for PVC was estimated using historical data and related to construction activity since PVC is used heavily in several outdoor construction activities. The supply side of the market for PVC products was determined through a model plant analysis. The information from demand and supply analyses is used to determine aggregate price indices for PVC products and subsequent economic damage.

The ozone depletion estimates were the same as shown in Exhibit 13-8. The total PVC damage associated with ozone depletion is given in Exhibit 13-13.

The results are made for the following set of circumstances: 1) All estimates are reported in millions of 1984 dollars; 2) the factor increase in titanium dioxide concentration is computed for a zenith angle of 60°; and 3) polymer producers do not respond to the hypothesized decreases in stratospheric ozone until 10 years after the impact is observed.

As indicated so far, due to the lack of data, the Mathtech analysis is limited to PVC only. In order to provide broader coverage of potential

damages, it is appropriate to consider other polymers that may be adversely impacted by increased UV-B radiation.

EXHIBIT 13-13

PVC Damage with Ozone Depletion
(Millions of 1984 dollars)

	Discount Rate			
	0%	2%	5%	10%
Low Estimate of PVC Consumption	2,440	603	97	10
Middle Estimate of PVC Consumption	4,716	1,137	174	17
High Estimate of PVC Consumption	9,158	2,159	315	27

Source: Horst, R., K. Brown, R. Black, and M. Kianka, "The Economic Impact of Increased UV-B Radiation on Polymer Materials: A Case Study of Rigid PVC," Mathtech, Inc., Princeton, New Jersey, June 1986.

The following equations, developed by Mathtech, Inc., provide future demand projections for each polymer:

$$Q = A\ (1 - \exp\ (-kT)) \qquad (31)$$

For small values of parameter k, the above equation can be linearlized as follows:

$$Q_T = a+b\ (T-T_0) + c(T-T_0)^2 \qquad (32)$$

where Q = consumption per person in year T

T_0 = year for which the projection is made

a, b, c = parameters to be estimated.

Projections of future demands for selected polymers and selected years are provided in Exhibit 13-14. Damage to these polymers is difficult to estimate without any activation spectra. However, the magnitude of the damage is expected to be similar to that for PVC.

EXHIBIT 13-14

Projections of Future Demand for Selected Years
(Thousands of Metric Tons)

Year	Acrylics	Polyester
1980	75.0	644.0
2000	148.5	1130.6
2025	179.4	1400.0
2050	186.7	1472.8
2075	190.0	1504.8

Source: Horst, R., K. Brown, R. Black, and M. Kianka, "The Economic Impact of Increased UV-B Radiation on Polymer Materials: A Case Study of Rigid PVC," Mathtech, Inc., Princeton, New Jersey, June 1986.

EFFECT OF TEMPERATURE AND HUMIDITY ON PHOTODEGRADATION

A necessary element in the discussion of damage functions missing so far is the possible role of temperature and humidity. Increasing the temperature almost always results in an increase in the rate of a chemical reaction. This is certainly true of the chemical reactions responsible for photodegradation. The extent of such an increase depends upon the energy of activation for the particular reaction. The activation energy for photodegradation of polypropylene in air, for instance, is 77-80 kcal/mole. That for PVC (based on polyene formation) determined by Reinisch et al. (1970) was 18 kcal/mole. These values allow the estimation of approximate factor increase in the weathering reaction due to increased temperature.

The general effect of humidity or water is to increase the rate of degradation of the polymer. It is generally believed to cause slight plasticization (or softening) of the polymer matrix. This is hardly likely to be the only mechanism. Several experimental studies strongly suggest water/pigment interactions. The effect of irradiation of titanium dioxide in

the presence of water was shown to generate hydrogen peroxide (Hoffmann et al. 1971). The deleterious effect of peroxides on polymers is well known. Thus depending upon the chemical nature of polymer, the components of the compound and the weathering factors, both temperature and humidity tend to increase the rate of degradation. Generalized quantitative data are not available on the polymers of interest to provide potential damage estimates.

FUTURE RESEARCH

As evident in the discussion of this chapter, there are no experimental data available on several polymers of interest to provide a good estimate of damage in polymers under increased light intensity. The EPA is currently funding a research study to evaluate the available scientific and technical information and conduct experimental as well as field studies to evaluate effects of increased UV radiation on polymers.

Poly(vinylchloride) (PVC) polymers are selected for the experimental study since PVC is the most widely used thermoplastic for outdoor applications. Two main categories of PVC formulations, (a) rigid PVCs such as those used in siding and window frames and (b) plasticized PVCs such as those used in flexible roofing membranes and cable coatings, will be studied. Typical formulations and formulations with varying amounts of titanium dioxide will be evaluated in a simulated solar radiation using a Xenon lamp source.

The field research will study the question of UV-induced material damage from a global point of view. The types of UV radiation (in terms of spectral quality) which North Americans would receive at 10-20% ozone depletion is typical of the solar flux received at some equatorial locations at current levels of ozone. Studying the material behavior at these locations would be invaluable in estimating the effects of increased UV light at northern latitudes.

The yellowing of PVC will be the main criterion used to assess damage, although other tests may be employed as necessary. Data obtained from this research will be used to assess damage to PVCs and to estimate additional costs to stabilize PVC in the event of partial ozone depletion.

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CHAPTER 14

POTENTIAL EFFECTS OF STRATOSPHERIC OZONE DEPLETION ON TROPOSPHERE OZONE (SMOG)

SUMMARY

Tropospheric ozone, 03(T), (smog) is an air pollutant formed near the earth's surface as a result of photochemical reactions involving ultraviolet radiation, hydrocarbons, nitrogen oxides, oxygen and sunlight. At high concentrations, often found during warmer months, smog can adversely affect human health, agricultural crops, forests and materials. The U.S. Environmental Protection Agency (EPA) has established a primary standard to protect public health of 0.12 ppm (one-hour average) not to be exceeded more than one day per year. In 1979, EPA also determined that a secondary welfare standard, more stringent than the primary standard, was unnecessary for the protection of vegetation. Currently, EPA is reviewing available scientific and technical information to determine whether these standards are adequate to protect health and welfare.

This chapter reviews preliminary scientific information which suggests that increases in UV-B radiation may effect rate of tropospheric ozone (smog) formation in urban areas. The results from these analyses suggest that increased ultraviolet radiation increases the rate of smog production and acid rain precursors. Moreover, global warming, associated with ozone depleting substances may enhance these reactions. Further analyses of additional cities are being conducted. If these analyses confirm these results, it would appear likely that in the future more cities and regions would violate the ambient air standards and that more restrictive measures to control hydrocarbons and nitrogen oxides may be required in order to comply with current standards.

FINDINGS

1. PRELIMINARY RESULTS FROM ONE STUDY SUGGEST THAT INCREASED ULTRAVIOLET RADIATION FROM OZONE DEPLETION WOULD INCREASE THE RATE OF TROPOSPHERIC OZONE FORMATION.

   1a. Increases in UV-B associated with ozone depletion would increase the quantity of increase in ground-based ozone associated with various hydrocarbon and nitrogen oxides emission levels depending on the chemistry of each city.

   1b. Global warming would enhance the effects of increased UV-B radiation on the formation of ground-based ozone.

   1c. Ground-based ozone is predicted to form earlier in the day. This may cause larger populations to be exposed to peak values in some cities.

2. PRELIMINARY RESULTS ALSO PREDICT LARGE INCREASES IN HYDROGEN PEROXIDE.

   2a. If hydrogen peroxide increases as predicted, the oxidation potential of the atmosphere, including the formation of acid rain, would be influenced.

3. INCREASES IN GROUND BASED OZONE WOULD ADVERSELY AFFECT PUBLIC HEALTH AND WELFARE.

   3a. There is a potential for more cities to be unable to meet health-based standards.

   3b. Crops, ecosystems, and materials could be adversely affected.

INTRODUCTION

Tropospheric ozone, O3, (smog) is an air pollutant formed in the ambient air as a result of a series of complex photochemical reactions involving ultraviolet radiation, hydrocarbons, and nitrogen oxides emitted from mobile and stationary sources, atmospheric oxygen, and sunlight. At ambient concentrations, often measured during warmer months, O3 can adversely affect human health, agricultural crops, forests, ecosystems, and materials. Interactions of O3 with nitrogen oxides and sulfur oxides may also contribute to the formation of acidic precipitation. Typical short-term (1-hour) O3 levels range from 0.01 ppm in some isolated rural areas to as high as 0.35 ppm in one of the nation's most heavily populated metropolitan areas. Daily daylight seasonal averages in some rural areas have been reported to be 0.06 ppm and higher.

Using 1982-1984 data -- the most recent available -- 216 metropolitan statistical areas (MSAs) out of 319 MSAs (68%) have enough O3 air quality data to ascertain attainment status. 119 (MSAs) (55%) have more than one expected exceedance per year of the current EPA health-based O3 standard of 0.12 ppm. Thus, more than one-half of the MSAs with sufficient data violate the standard. Approximately 115 million people, i.e., over one-half of the total U.S. population, live in areas which exceed the standard. However, this does not mean that everyone in these areas is exposed to O3 concentrations at or above the standard (EPA 1986).

Of MSAs with sufficient data, about 14% (34) have a characteristic highest concentrations (CHC) of 0.16 ppm O3 or higher and over 4% (11) of MSAs have a CHC above 0.20 ppm O3. There is no clear temporal trend in O3 concentration levels in MSAs around the country, although 1982 -- and to a lesser extent, 1981 -- had generally lower levels than other years during the 1979-1984 time period.

Generally, third quarter (July-September) and seasonal (April-September) average of 8:00 a.m. - 4:00 p.m. daily daylight values are both in the range of 0.043-0.050 ppm O3. Statistically significant, but modest, relationships exist between peak and longer-term mean indices of O3 air quality in urban areas.

Recently, EPA (1986) reviewed scientific and technical information on the known and potential health effects of ozone. The information includes respiratory tract absorption and deposition of ozone, studies of mechanisms of O3 toxicity, effects of exposure to O3 reported in controlled human exposure, field, epidemiological and animal toxicology studies, as well as air quality information. The results of that review suggest that:

(1) The mechanisms by which inhaled O3 may pose health risks involve (a) penetration into and absorption of O3 in various regions of the respiratory tract, (b) pulmonary response resulting from chemical interactions

of O3 along the respiratory tract, and (c) extrapulmonary effects caused indirectly by effects of O3 in the lungs.

(2) The risks of adverse effects associated with absorption of O3 in the tracheobronchial and alveolar regions of the respiratory tract are much greater than for absorption in the extrathoracic region (head). Increased exercise levels are generally associated with higher ventilation rates and increased oronasal or oral (mouth) breathing. Thus, maximum penetration and exposure of sensitive lung tissue occurs when heavily exercising individuals are exposed to O3.

(3) Factors which affect susceptibility to O3 exposure are activity level and environmental stress (e.g., humidity, high temperature).

(4) The major subgroups of the population at greatest risk to the effects of O3 include: (a) individuals with preexisting respiratory disease (e.g., asthmatics and persons with chronic obstructive lung disease or allergies), (b) "responders" who are otherwise healthy individuals, both adults and children, but experience significantly greater group mean lung function response to O3 exposure, and (c) any individual exercising heavily during exposure to O3.

(5) The major health concerns associated with exposure to O3, in approximate order of strength of the data base include:

(a) alterations in pulmonary function;

(b) symptomatic effects;

(c) effects on work performance;

(d) aggravation of preexisting respiratory disease;

(e) morphological effects (lung structure damage);

(f) altered host defense systems (i.e., increased susceptibility to respiratory infection); and

(g) extrapulmonary effects (e.g., effects on blood enzymes, central nervous system, liver, endocrine system).

(6) The most useful exposure-response information is from controlled human exposure and field studies which provide a quantitative relationship between alterations in pulmonary function and O3 exposure concentrations.

The EPA (1986) document came to the following conclusions on vegetation impacts:

(a) The mechanisms by which O3 may injure plants and plant communities include (1) absorption of O3 into leaf through stomata followed by diffusion through the cell wall and membrane, (2) alteration of cell structure and function, as well as critical plant processes, resulting from the chemical interaction of O3 with cellular components, (3) occurrence of secondary effects including reduced growth and yield and altered carbon allocation;

(b) The magnitude of the O3 induced effects depends upon the physical and chemical environment of the plant, as well as varous biological factors (including genetic potential, developmental age of plant and interaction with plant pests);

(c) Effects of O3 on vegetation and ecosystems have been demonstrated to occur from both short-term and long-term exposures. Although there are a limited number of studies in which short-term (1-2 hour) exposures have resulted in growth and yield reduction, there is a growing body of evidence that repeated peaks above a given level are important in eliciting plant response;

(d) Concerning long-term exposures, the bulk of the evidence indicates that growth and yield losses occur in several plant species exposed to seasonal concentrations of O3, typically characterized as the daily daylight mean over the growing season. In addition, evidence indicates that forests experience cumulative stress as a result of chronic exposure to O3. Exhibit 14-1 summarizes the range of O3 levels and exposure times required to induce 5% and 20% foliar injury. Exhibit 14-2 (EPA 1986) provides a more complete survey; and

(e) Damage to materials is another effect of O3. There appears to be no threshold level below which material damage will not occur; the slight acceleration of the aging processes of materials occurs at the level of the proposed standard. The materials known to be most susceptible to ozone attack are elastomers, textile fibers and dyes, and certain types of paint.

EXHIBIT 14-1

Ozone Concentrations for Short-Term Exposure That Produce 5% or 20% Injury to Vegetation Growth Under Sensitive Conditions[1]

Exposure Time, Hour	Ozone Concentrations (ppm) that may Produce 5% (20%) Injury: Sensitive Plants	Intermediate Plants	Less Sensitive Plants
0.5	0.35 to 0.50 (0.45 to 0.60)	0.55 to 0.70 (0.65 to 0.85)	≥0.70 (0.85)
1.0	0.15 to 0.25 (0.20 to 0.35)	0.25 to 0.40 (0.35 to 0.55)	≥0.40 (0.55)
2.0	0.09 to 0.15 (0.12 to 0.25)	0.15 to 0.25 (0.25 to 0.35)	≥0.30 (0.40)
4.0	0.04 to 0.09 (0.10 to 0.15)	0.10 to 0.15 (0.15 to 0.30)	≥0.25 (0.35)
8.0	0.02 to 0.04	0.07 to 0.12	≥0.20 (0.30)

[1] The concentrations in parenthesis are for the 20% injury level. Exhibit from U.S. Environmental Protection Agency (1985, p. 7-225).

EXHIBIT 14-2

Ozone Concentrations at Which Significant Yield Losses Have Been Noted for a Variety of Plant Species Exposed Under Various Experimental Conditions

Plant Species	Exposure Duration	Yield Reduction, % of Control	O_3 Concentration, ppm	Reference
Alfalfa	7 hr/day, 70 days	51, top dry wt	0.10	Neely et al., 1977
Alfalfa	2 hr/day, 21 day	16, top dry wt	0.10	Hoffman et al., 1975
Pasture grass	4 hr/day, 5 days/wk, 5 wk	20, top dry wt	0.09	Horsman et al., 1980
Ladino clover	6 hr/day, 5 days	20, shoot dry wt	0.10	Blum et al., 1982
Soybean	6 hr/day, 133 days	55, seed wt/plant	0.10	Heagle et al., 1974
Sweet corn	6 hr/day, 64 days	45, seed wt/plant	0.10	Heagle et al., 1972
Sweet corn	3 hr/day, 3 days/wk, 8 wk	13, ear fresh wt	0.20	Oshima, 1973
Wheat	4 hr/day, 7 day	30, seed yield	0.20	Shannon and Mulchi, 1974
Radish	3 hr	33, root dry wt	0.25	Adedipe and Ormrod, 1974
Beet	2 hr/day, 38 days	40, storage root dry wt	0.20	Ogata and Maas, 1973
Potato	3 hr/day, every 2 wk, 120 days	25, tuber wt	0.20	Pell et al., 1980
Pepper	3 hr/day, 3 days/wk, 11 wk	19, fruit dry wt	0.12	Bennett et al., 1979
Cotton	6 hr/day, 2 days/wk, 13 wk	62, fiber dry wt	0.25	Oshima et al., 1979
Carnation	24 hr/day, 12 days	74, no. of flower buds	0.05-0.09	Feder and Campbell, 1968
Coleus	2 hr	20, flower no.	0.20	Adedipe et al., 1972
Begonia	4 hr/day, once every 6 days for a total of 4 times	55, flower wt	0.25	Reinert and Nelson, 1979
Ponderosa pine	6 hr/day, 126 days	21, stem dry wt	0.10	Wilhour and Neely, 1977
Western white pine	6 hr/day, 126 days	9, stem dry wt	0.10	Wilhour and Neely, 1977
Loblolly pine	6 hr/day, 28 days	18, height growth	0.05	Wilhour and Neely, 1977
Pitch pine	6 hr/day, 28 days	13, height growth	0.10	Wilhour and Neely, 1977
Poplar	12 hr/day, 5 mo	+1333, leaf abscission	0.041	Wilhour and Neely, 1977
Hybrid poplar	12 hr/day, 102 days	58, height growth	0.15	Patton, 1981
Hybrid poplar	8 hr/day, 5 day/wk, 6 wk	50, shoot dry wt	0.15	Patton, 1981
Red maple	8 hr/day, 6 wk	37, height growth	0.25	Dochinger and Townsend, 1979
American sycamore	6 hr/day, 28 days	9, height growth	0.05	Kress and Skelly, 1982
Sweetgum	6 hr/day, 28 days	29, height growth	0.10	Kress and Skelly, 1982
White ash	6 hr/day, 28 days	17, total dry weight	0.15	Kress and Skelly, 1982
Green ash	6 hr/day, 28 days	24, height growth	0.10	Kress and Skelly, 1982
Willow oak	6 hr/day, 28 days	19, height growth	0.15	Kress and Skelly, 1982
Sugar maple	6 hr/day, 28 days	12, height growth	0.15	Kress and Skelly, 1982

POTENTIAL EFFECTS OF ULTRAVIOLET RADIATION AND INCREASED TEMPERATURES ON URBAN SMOG

Recently, Whitten (1986) conducted a preliminary investigation into the potential changes in urban ozone levels because of an increase in solar ultraviolet (UV) radiation resulting from reductions in the stratospheric ozone layer. Estimates were also made of the effects on urban ozone chemistry resulting from a general warming of the lower atmosphere. The focus of this study was the effect of reductions in stratospheric ozone by as much as 30 percent of (1) peak ozone concentrations in urban areas and (2) the attendant control requirements predicted by a modified version of the Empirical Kinetics Modeling Approach (EKMA). The model used in this study was the simple trajectory model used in the EKMA. This model was developed by the EPA and is widely used to assess the effectiveness of emissions control scenarios to abate urban ozone.

Whitten began by investigating how the ozone formation processes in the lower troposphere would be affected by increased UV transmission through the atmosphere. From preliminary work, it became evident that the photolysis channel of formaldehyde that leads to radical products is selectively enhanced at a higher UV flux. Formaldehyde emissions are products of incomplete combustion, and formaldehyde is a major intermediate oxidation product from virtually all organic molecules. Radicals from formaldehyde photolysis provide the main source of radicals needed to drive the chain reactions which generate photochemical smog (Whitten 1983), therefore, an increase in rate of photolysis will have a bearing on the design of future control strategies.

The photolysis of ozone to electronically excited oxygen atoms is thought to be the second most important source of the radicals that drive smog formation. However, the role of ozone photolysis is different from formaldehyde because ozone is the principal ingredient of photochemical smog. At low levels of oxidation potential, the excited oxygen atoms tend to accelerate smog reactions, making the atmospheric chemistry more efficient in generating ozone from minimal precursor emissions. However, at the higher or more severe ozone levels, the excess radicals can partially suppress the ozone peak, making the precursors seem less efficient in generating ozone. Other photolysis rates can also be affected, but their contribution to smog formation is less important.

A specific increase in formaldehyde photolysis to radical products is difficult to accurately determine for a number of reasons. Atmospheric photolysis rates have often been calculted in 10 nm wavelength intervals. However, the accurate calculation of formaldehyde photolysis requires fine spectral resolution for both the formaldehyde absorption cross-section and the surface solar flux (related to the ozone absorption cross-section). Only in this manner can a comparison of the respective fine structures be performed. Near-ground solar flux data of high resolution and known stratospheric ozone abundance have not been readily available. Hence, Whitten (1986) estimated surface solar fluxes using low-resolution information provided by the National Aeronautics and Space Administration.

Using the spectral data, Whitten (1986) estimated formaldehyde and ozone photolysis rates as a function of zenith angle and ultraviolet flux changes due to stratospheric ozone depletion. The estimated photolysis rates for formaldehyde, coupled with the information compiled for surface ozone photolysis, provide the inputs needed to calculate diurnal photolysis rates for projected future ozone column densities. This information is used to evaluate the future impact of stratospheric ozone changes on near-surface smog formation.

The preliminary results of the possible effects of increased ultraviolet radiation on urban smog are based on simulation of atmospheric conditions for three urban cities:

(1) Nashville -- because it is nearly in compliance with the 0.12 ppm federal ozone standard.

(2) Philadelphia -- to represent cities that require moderate control (30%-50% reduction in organic precursors) to achieve the 0.12 ppm standard.

(3) Los Angeles -- because of the severity of the exceedance of the ozone standard in that region.

Present-day estimates assume a total ozone column of 300 DU and future predictions assume a total ozone column of 250 and 200 DU. A 33% decrease in total ozone column would increase ozone photolysis by approximately a factor of two, and increase formaldehyde photolysis to radical products by nearly 20%. Both of these increases are approximations because of uncertainties regarding the spectral fine structure and other factors.

The effects of these photolysis rate increases are given in Exhibit 14-3 for background temperatures of 298K and 302K. For all three cities, the model predicts higher ozone concentrations resulting either from increases in temperature or decreases in the Dobson number. The magnitude of the increases in ozone is apparently a function of local hydrocarbon-to-nitrogen oxide ratio, reactivity, meteorology, and emission distribution. The linearity of the response to stratospheric ozone depletion is not general. For the Los Angeles case, the increases in ozone are moderate and quite linear with decreasing Dobson number. For the Philadelphia case, ozone is predicted to increase progressively as the Dobson number declines; that is, only a modest increase in ozone is seen for a decline in Dobson number from 300 to 250 units, but a more dramatic increase in ozone is seen when the Dobson number declines from 250 to 200 units. For the Nashville case, the increases are all very dramatic and the linearity exists only if temperature does not increase. At the "current" temperature of 298K, the simulated urban ozone tends to increase linearly with Dobson number decrease; but at the warmer 302K temperature, the simulated ozone increases more for the first 50-unit Dobson change than for the second 50-unit change, down to the 200 unit limit studies. Preliminary trajectory model results suggest that peak smog levels are reached earlier in the day, which would expose larger populations to peak values. Hydrogen peroxide increased from approximately 1.7 ppb to 3.3 ppb

EXHIBIT 14-3

Ozone Concentrations (ppm) Predicted for Changes in Dobson Number and Temperature for Three Cities

	Ozone Concentrations					
Temperature (K)	298K			302K		
Dobson Number	300	250	200	300	250	200
City						
Los Angeles*	0.288	0.301	0.315	0.306	0.318	0.331
Philadelphia	0.112	0.127	0.149	0.122	0.134	0.159
Nashville	0.130	0.161	0.195	0.146	0.184	0.215

* For Los Angeles, values under 298K used actual hourly temperatures and values under 302K are from simulations using those temperatures increased by 4K.

in Los Angeles and from .19 ppb to approximately 3.0 ppb in Philadelphia for a 100 unit change in Dobson. These later changes suggest significant potential impacts for ozone depletion on acid rain formation.

The effects of a 4K increase in temperature are most pronounced in the Los Angeles and Nashville simulations where 14 to 23 ppb increases in simulated ozone peaks are predicted. The effects of a combined temperature increase and reduce Dobson number appear to be additive, if not synergistic, especially for the Nashville simulations. When a 4K temperature increase is combined with only a 50-unit Dobson reduction, the simulated ozone peak jumps from a base value of 0.13 ppm to over 0.18 ppm ozone. See Exhibit 14-4.

CONCLUSIONS AND FUTURE RESEARCH DIRECTIONS

The preliminary study (Whitten 1986) was limited in several ways because the primary focus was on the atmospheric chemistry of ozone formation e.g., potential changes in acid rain were not addressed. (Although the effects of local warming and increased ultraviolet radiation were addressed in the preliminary study, increases in air pollution such as smog and acid rain can only be addressed through more extensive modeling using more complex atmospheric models). However, the simple, moving box model remains applicable in the more extensive modeling.

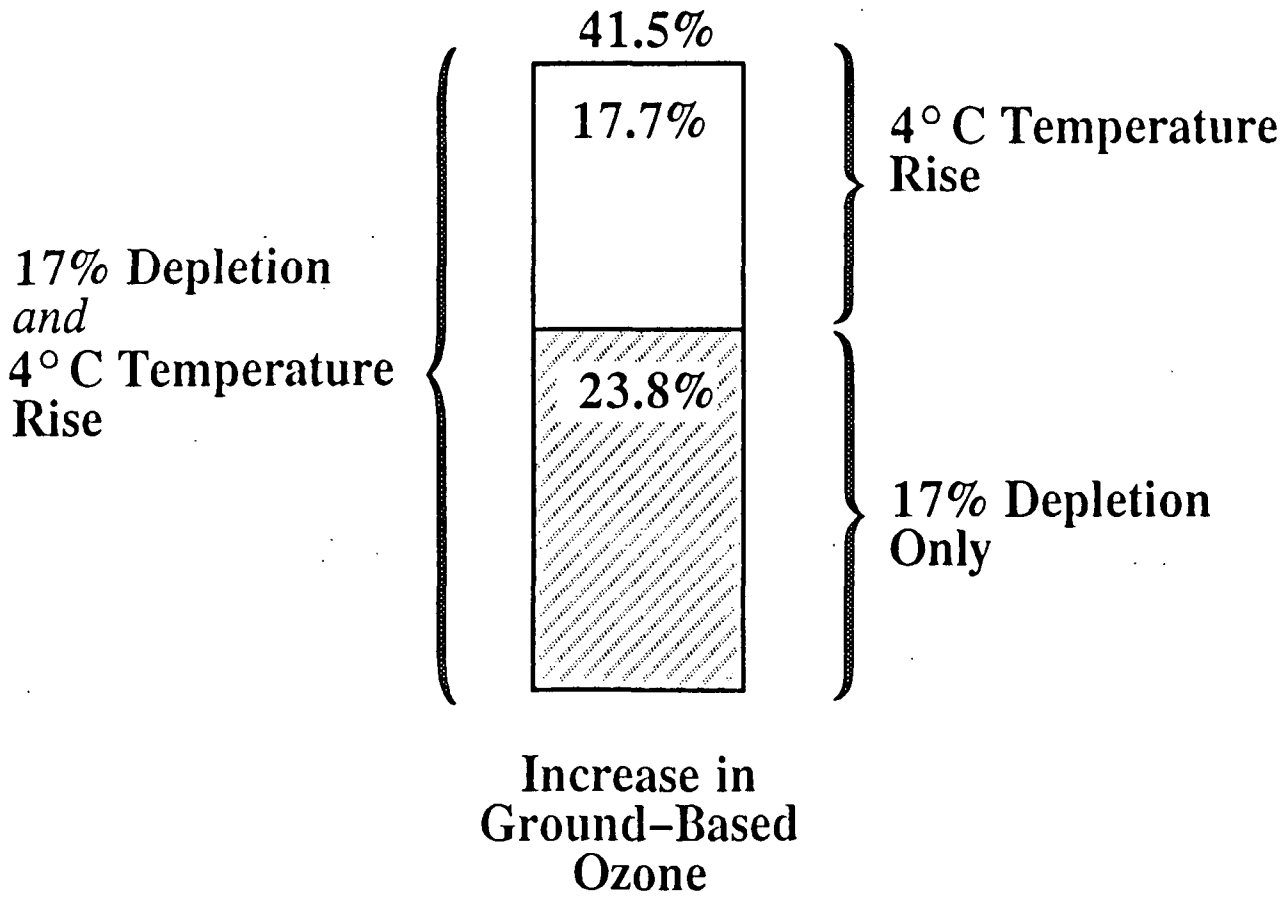
Although the initial study involved the use of only one atmospheric chemical mechanism, the Carbon Bond Mechanism (CBM), the key reactions are virtually identical in all currently accepted atmospheric mechanisms. Hence, it can be expected that other mechanisms would show the same or similar responses to the global effects. Thus, the use of the CBM is not considered a serious limitation of the current work. However, the increased radiation effects studied could not utilize high resolution spectral data even though the preliminary study seemed to show a need to use such data.

Thus far, atmospheric modeling has addressed only three trajectories, one each in three cities. Three cities is not an adequate sample/size to characterize the potential range of conditions affected by these global effects. Although the model is sensitive to initial and boundary conditions, these conditions were not varied, even though the hypothesized global effects would affect such conditions. The data for initial and boundary conditions were primarily composed of air from upwind cities or air recirculated from the day before. Emissions were not varied to account for local warming, even though many emissions increase with temperature. Climate and other meteorological changes would affect important factors such as mixing height and wind flow patterns. These possible changes were also not addressed in the preliminary study.

A very important finding of the preliminary study is the increased rate of smog formation due to increased ultraviolet radiation. Even if the peak smog concentration is not affected, the increased rate of formation will move the high concentrations of smog closer to the precursor emissions where the population density is invariably higher. Hence the number of people exposed

EXHIBIT 14-4

Global Warming Would Exacerbate Effects of
Depletion on Ground-Based Ozone in Nashville



Source: Adapted from Whitten (1986).

to high smog concentrations would increase. This exposure effect is not addressed in the preliminary study. A grid type model is more appropriate for such exposure studies.

In summary, the following limitations of the preliminary study are noted:

- Effects on pollutants other than urban smog or ozone were not addressed; acid rain may be affected by global effects.
- The chemical effects were calculated using low resolution spectral data; high resolution data appear to be needed.
- Since only three cities were studied, the range of conditions most affected by global effects could not be elucidated.
- Multi-day effects such as inter- or intracity carryover of pollutants were not addressed.
- Temperature effects on emissions rates or changes in wind patterns, mixing heights, or frequency of episodic meteorological conditions were not addressed.
- Possible increases in population exposure due to increased smog levels and increased rates of smog formation were not addressed.

Current studies attempt to address these limitations by (1) increasing the number of trajectory cases, (2) using high resolution spectral data, (3) studying the range of conditions which can possibly affect carryover of pollutants, (4) varying the emissions with temperature, (5) studying climate change effects, and (6) calculating population exposure changes. The use of an acid rain model and a grid model will be incorporated in the on-going investigations.

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Chapter 15

CHAPTER 15

CAUSES AND EFFECTS OF SEA LEVEL RISE

SUMMARY

One of the most widely examined impacts of the projected global warming is the possible rise in sea level. Researchers have identified at least three mechanisms that might cause a significant rise: the warming and resulting expansion of the upper layers of the ocean, the melting of alpine glaciers, and the melting and disintegration of polar ice sheets in Greenland and Antarctica. Estimates of the rise through the year 2100 range, in the absence of efforts to limit the greenhouse warming, range from 50 cm to over 2 m. Even the most conservative estimate implies a substantial acceleration over the 10 to 15 cm rise of the last century.

A rise in sea level in that range would permanently inundate wetlands and lowlands, accelerate coastal erosion, exacerbate coastal flooding, and increase the salinity of estuaries and aquifers. Although wetlands have kept pace with sea level rise in the last several thousand years, a 1-2 m rise would destroy a majority of U.S. coastal marshes and swamps. River deltas such as those of the Mississipi, Ganges, and Nile rivers appear to be particularly vulnerable.

Along the open coast, beach erosion could reach 1 to 2 m for every 1 cm rise in sea level, in addition to whatever erosion might be caused by other factors. Because buildings are generally found within 50 m of the shore, even the 30 cm rise projected for the next 40 years could threaten coastal property and the recreational use of beaches, unless additional remedial measures are implemented.

Sea level rise would also increase the vulnerability of coastal areas to flooding from storm surges and rainwater. In the area of Charleston, S.C., for example, the area now flooded by a 100-year storm would be flooded by a 10-year storm if sea level rises 1.6 m. Protecting against increased flooding would require improvement or construction of levees, seawalls, and drainage facilities.

Higher water levels wold also increase the salinity of estuaries and aquifers. For example, Philadelphia's drinking water intake on the Delaware River would be threatened by a 73-cm rise, as would adjacent aquifers in New Jersey that are recharged by the (currently) fresh part of the river. Construction of additional reservoirs might be necessary to offset salinity increases.

Few studies have estimated the economic significance of future sea level rise. One study suggests that the impacts of a 0.9 to 2.4 m rise by 2075 could be as great as 17 to 35 percent of total economic activity in the Charleston, South Carolina area and 5 to 16 percent of the activity around Galveston, Texas. No one has yet estimated the potential nationwide cost of defending shorelines and other resources from the projected rise in sea level.

FINDINGS

1. GLOBAL AVERAGE SEA LEVEL HAS RISEN 10 TO 15 CM OVER THE LAST CENTURY.

   1a. Ocean and glacial studies suggest that the rise is higher than what models of thermal expansion and alpine meltwater would project given the 0.6 C warming of the past century.

   1b. However, no cause-and-effect between warming and past sea level has been conclusively demonstrated.

2. THE PROJECTED GLOBAL WARMING WOULD ACCELERATE THE CURRENT RATE OF SEA LEVEL RISE BY EXPANDING OCEAN WATER, MELTING ALPINE GLACIERS, AND EVENTUALLY INCREASING THE RATE AT WHICH POLAR ICE SHEETS MELT OR DISCHARGE ICE INTO THE OCEANS.

3. ESTIMATES OF THE RISE IN SEA LEVEL THAT COULD TAKE PLACE IF MEASURES TO LIMIT THE GLOBAL WARMING ARE NOT UNDERTAKEN RANGE FROM 10 TO 20 CM BY THE YEAR 2025, AND 50 TO 200 CM BY 2100.

   3a. Greenland and alpine glaciers could each contribute 10 to 30 cm through 2100.

   3b. The contribution of Antarctic deglaciation is likely to be between 0 and 100 CM; however,the possibilities cannot be ruled out that (1) increased snowfall could increase the size of the Antarctic ice sheet and thereby offset part of the sea level rise from other sources: or (2) meltwater and enhanced calving of the ice sheet could increase the contribution of Antarctica to as much as 2 m.

   3c. When added to estimated 40-cm rise due to thermal expansion, the 10 to 160 cm estimated glacial contribution estimated by the NAS Polar Research Board is consistent with the conclusion stated here.

4. DISINTEGRATION OF THE WEST ANTARCTIC ICE SHEET MIGHT RAISE SEA LEVEL 6 M OVER THE NEXT FEW CENTURIES, IN ADDITION TO THE SMALLER CONTRIBUTIONS FROM THERMAL EXPANSION, ALPINE GLACIERS, AND ICE SHEETS IN GREENLAND AND EAST ANTARCTICA.

   4a. Glaciologists generally believe that such a disintegration would take at least 300 years, and probably at least 500 years.

   4b. A global warming might result in sufficient thinning of the Ross and Filcher-Ronne Ice Shelves in the next century to make the process irreversible.

5. LOCAL TRENDS IN SUBSIDENCE AND EMERGENCE MUST BE ADDED OR SUBTRACTED TO ESTIMATE THE RISE AT PARTICULAR LOCATIONS.

   5a. Most of the Atlantic and Gulf Coasts of the United States--as well as the Southern Pacific coast--are subsiding 10-20 cm per century.

   5b. Louisiana is subsiding 1 m per century and parts of Alaska are emerging 10-100 cm per century.

6. A SUBSTANTIAL RISE IN SEA LEVEL WOULD PERMANENTLY INUNDATE WETLANDS AND LOWLANDS, ACCELERATE COASTAL EROSION, EXACERBATE COASTAL FLOODING, AND INCREASE THE SALINITY OF ESTUARIES AND AQUIFERS.

7. LOUISIANA IS THE STATE MOST VULNERABLE TO A RISE IN SEA LEVEL. IMPORTANT IMPACTS WOULD ALSO OCCUR IN FLORIDA, MARYLAND, DELAWARE, NEW JERSEY, AND IN THE COASTAL REGIONS OF OTHER STATES.

8. A RISE IN SEA LEVEL OF 1 TO 2 M BY THE YEAR 2100 COULD DESTROY 50% TO 80% OF U.S. COASTAL WETLANDS.

9. EROSION CAUSED BY SEA LEVEL RISE COULD THREATEN U.S. RECREATIONAL BEACHES.

   9a. Case studies of beaches in New Jersey, Maryland, California, South Carolina, and Florida have concluded that a 30-cm rise in sea level would result in beaches eroding 20-60 m or more.

   9b. Because the first row of houses is generally less than 20 m from the shore at high tide, and if available studies are representative, recreational beaches throughout the nation would be threatened by a 30-cm rise unless major beach preservation efforts are undertaken.

10. SEA LEVEL RISE WOULD INCREASE THE DAMAGES FROM FLOODING, FLOOD PROTECTION, AND FLOOD INSURANCE IN COASTAL AREAS.

   10a. Flood damages would increase because higher water levels would provide a higher base for storm surges.

   10b. Erosion would increase the vulnerability to storm waves,and decreased natural and artificial drainage would increase flooding duirng rainstorms.

11. INCREASED SALINITY FROM SEA LEVEL RISE WOULD CONVERT CYPRESS SWAMPS TO OPEN WATER AND THREATEN DRINKING WATER SUPPLIES IN LOUISIANA, PHILADELPHIA, AND NEW JERSEY. OTHER AREAS, SUCH AS SOUTHERN FLORIDA, MAY ALSO BE VULNERABLE BUT HAVE NOT BEEN INVESTIGATED.

12. ESTIMATES OF DAMAGE FROM SEA LEVEL RISE MUST CONSIDER HUMAN RESPONSES.

12a. The adverse impacts of sea level rise could be ameliorated through anticipatory land use planning and structural design changes.

12b. In two case studies in Charleston and Galveston, accelerated anticipatory planning was estimated to reduce net damages by 20 to 60%.

13. OTHER IMPACTS OF A GLOBAL WARMING MIGHT OFFSET OR EXACERBATE THE IMPACTS OF SEA LEVEL RISE.

13a. Increased droughts might amplify the salinity impacts of sea level rise.

13b. Increased hurricanes and increased rainfall in coastal areas could amplify flooding from sea level rise.

13c. Warmer temperatures might impair peat formation of salt marshes and would enable mangrove swamps to take over areas that are presently salt marsh.

13d. Decreased northeasterners might reduce damage.

14. RIVER DELTAS THROUGHOUT THE WORLD WOULD BE VULNERABLE TO A RISE IN SEA LEVEL, PARTICULARLY THOSE WHOSE RIVERS ARE DAMMED OR LEVEED.

15. ECONOMIC STUDIES OF THE CHARLESTON, SOUTH CAROLINA AND GALVESTON, TEXAS AREAS SUGGEST THAT SEA LEVEL RISE COULD BE ECONOMICALLY IMPORTANT TO COASTAL AREAS.

CAUSES OF SEA LEVEL RISE

The worldwide average sea level depends primarily on (1) the shape and size of ocean basins, (2) the amount of water in the oceans, and (3) the average density of seawater. Only the latter two factors are influenced by climate. Subsidence and emergence caused by natural factors such as isostatic and tectonic adjustments of the land surface, as well as human-induced factors such as oil and water extraction, can cause trends in relative sea level at particular locations to differ from trends in global sea level.

Past Trends in Sea Level

Hays and Pitman (1973) analyzed fossil records and concluded that over the last 100 million years, changes in mid-ocean ridge systems have caused the sea level to rise and fall over 300 m. However, Clark, Farrell, and Peltier (1978) have pointed out that these changes have accounted for sea level changes of less than 1 mm per century. No published study has indicated that this determinant of sea level is likely to have a significant impact in the next century.

The impact of climate on sea level has been more pronounced. During ice ages the glaciation of substantial portions of the northern hemisphere has removed enough water from the oceans to lower sea level 100 m below the present level during the last (18,000 years ago) and earlier ice ages. (Donn, Farrand, and Ewing 1962; Kennett 1982; Oldale 1985).

Although the glaciers that once covered much of the northern hemisphere have retreated, the world's remaining ice cover contains enough water to raise sea level over 75 m (Hollin and Barry 1979). As Exhibit 15-1 shows, Hollin and Barry (1979) and Flint (1971) estimate that existing alpine glaciers contain enough water to raise sea level 30 or 60 cm, respectively. The Greenland and West Antarctic Ice Sheets each contain enough water to raise sea level about 8 m, and East Antarctica has enough ice to raise sea level over 60 m.

There is no evidence that either the Greenland or East Antarctic Ice sheets have completely disintegrated in the last two million years. However, it is generally recognized that sea level was about 7 m higher than today during the last interglacial (Moore 1982; Mercer 1968; Hollin 1962), which was 1° to 2° warmer than today. Because the West Antarctic Ice Sheet is marine-based and thought by some to be vulnerable to climatic warming, attention has focused on this source for the higher sea level. Mercer (1968) found that lake sediments and other evidence suggested that summer temperatures in Antarctica have been 7° to 10°C higher than today at some point in the last two million years, probably during the last interglacial 125,000 years ago, and that such temperatures could have caused a disintegration of the West Antarctic Ice Sheet. However, others are not certain that marine based glaciers are more vulnerable to climate change than

EXHIBIT 15-1

Snow and Ice Components

	Area (10^6 km^2)	Ice Volume (10^6 km^3)	Sea-Level Equivalent a/ (m)
Land ice: East Antarctica b/	9.86	25.92	64.8
West Antarctica c/	2.34	3.40	8.5
Greenland	1.7	3.0	7.6
Small ice caps and mountain glaciers d/ (Flint 1971, Hollin and Barry 1979)	0.54	0.12	0.3 0.6
Permafrost (excluding Antarctica):			
Continuous	7.6	0.03 to	0.08 to
Discontinuous	17.3	0.7	0.17
Sea ice: Arctic e/			
Late February	14.0	0.05	
Late August	7.0	0.02	
Antarctic f/			
September	18.4	0.06	
February	3.6	0.01	
Land snow cover g/			
N. Hemisphere			
Early February	46.3	0.002	
Late August	3.7		
S. Hemisphere			
Late July	0.85		
Early May	0.07		

EXHIBIT 15-1 (continued)

Snow and Ice Components
Footnotes

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a/ 400,000 km^3 of ice is equivalent to 1 m global sea level.

b/ Grounded ice sheet, excluding peripheral, floating ice shelves (which do not affect sea level). The shelves have a total area of 1.62×10^6 km^2 and a volume of 0.79×10^6 km^3 (Drewry and Heim 1983).

c/ Including the Antarctic Peninsula.

d/ Flint, Hollin, and Barry (1971).

e/ Excluding the Sea of Okhotsk, the Baltic Sea, and the Gulf of St. Lawrence (Walsh and Johnson 1979). Maximum ice extents in these areas are 0.7 million, 0.4 million, and 0.2 million km^2, respectively.

f/ Actual ice area excluding open water (Zwally et al. 1983). Ice extent ranges between 4 million and 20 million km^2.

g/ Snow cover includes land ice but excludes snow-covered sea ice (Dewey and Heim 1981).

Source: Glaciers, Ice Sheets, and Sea Level. National Academy Press, p. 242. Modified from J.T. Hollin and R.G. Barry, "Empirical and Theoretical Evidence Concerning the Response of the Earth's Ice and Snow Cover to a Global Temperature Increase," Environment International, 1979, 2:437-444.

land based glaciers;[1] Robin (1985) suggests that the higher sea level during the last interglacial period may have resulted from changes in the East Antarctic Ice Sheet.

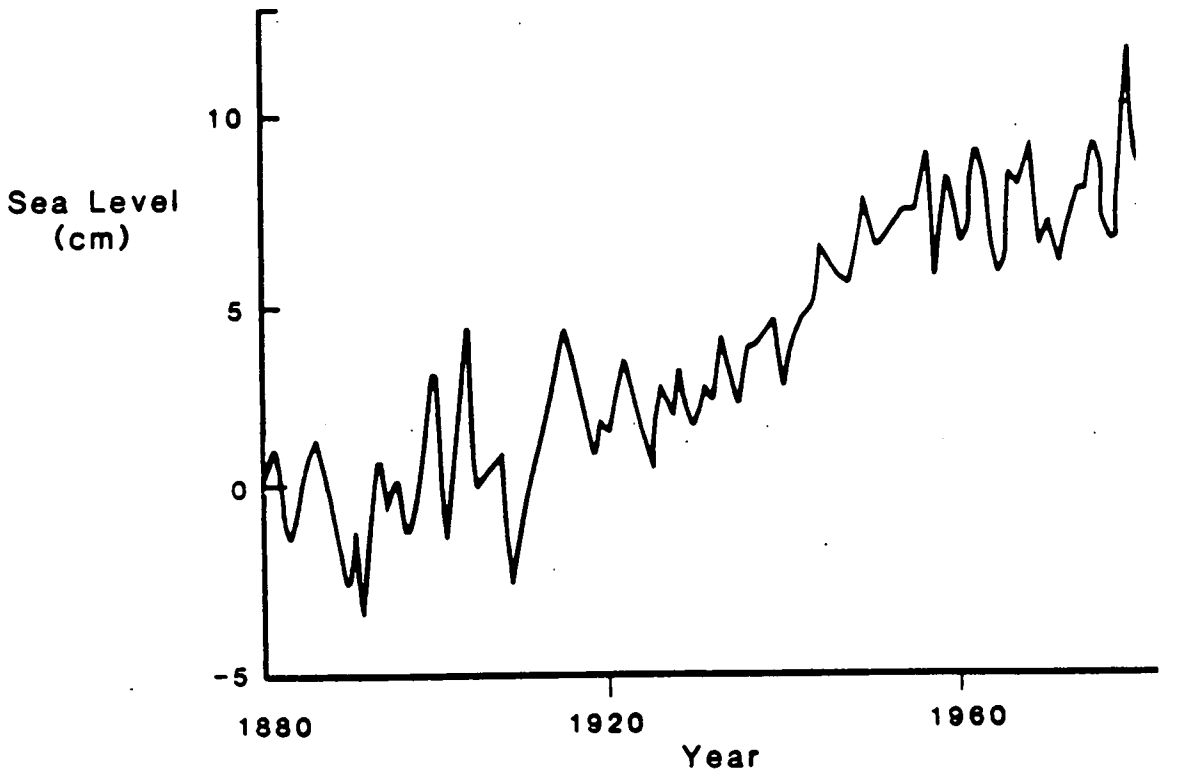
Tidal gauges have been available to measure the change in relative sea level at particular locations over the last century.[2] Studies combining these measurements to estimate global trends have concluded that sea level has risen 10-15 cm during the last century (Barnett 1984; Gornitz, Lebedeff, and Hansen 1982; Fairbridge and Krebs 1962; Hicks et al. 1983 for U.S. Coast); Exhibit 15-2 shows the sea level curve estimated by Gornitz et al. Barnett (1984) found that the rate of sea level rise over the last 50 years had been about 2.0 mm/year, while in the previous 50 years there had been little change; however, the acceleration in the rate of sea level rise was not statistically significant. Emery and Aubrey (1985 and n.d.) have accounted for estimated land surface movements in their analyses of tidal gauge records in Northern Europe and western North America and have found an acceleration in the rate of sea level rise over the last century.[3] Braatz and Aubrey (n.d.) have found that the rate of relative sea level rise on the east coast of North America accelerated after 1934.

Several researchers have investiaged possible causes of current trends in sea level. Barnett (1984) and Gornitz, Lebedeff, and Hansen (1982) estimate that thermal expansion of the upper layers of the oceans resulting from the observed global warming of 0.4°C in the last century could be responsible for a rise of 0.4 to 0.5 mm/year. Recent analysis suggests that the warming has been .6°C, which would account for thermal expansion of .6-.7 mm/year (Jones, Wigley, and Wright 1986). Roemmich and Wunsch (1984) examined temperature and salinity measurements at Bermuda and concluded that the 4°C isotherm had migrated 100 m downward, and that the resulting expansion of ocean water could be responsible for some or all of the observed rise in relative sea level. Roemmich (1985) showed that the warming trend 700 m below the surface was statistically significant. However, Barnett (1984) found no significant trend based on an examination of the upper layers of the ocean. Neverthless, Braatz and Aubrey (n.d.) note that long-term steric changes in the ocean are not confined to the upper layers of the oceans, implying that the Barnett analysis does not necessarily contradict the Roemmich and Wunsch conclusion. Because one-dimensional models that can not capture the complexities of the ocean have been used to estimate the past expansion of ocean water, these authors all caution that their results should be viewed as first approximations.

Meier (1984) estimates that retreat of alpine glaciers and small ice caps could currently contribute between 0.2 and 0.72 mm/year to sea level. The National Academy of Sciences Polar Research Board (Meier et al. 1985) concluded that existing information is insufficient to determine whether the impacts of Greenland and Antarctica are positive or zero. Thus, thermal expansion and alpine melting would explain a rise of .8 to 1.4 mm/yr, compared with the observed rise of 1 to 1.5 mm/yr. Although the estimated global warming of the last century appears at least partly responsible for the last century's rise in sea level, no study has demonstrated that global warming might be responsible for an acceleration in the rate of sea level rise.

EXHIBIT 15-2

Worldwide Sea Level in the Last Century



Sources: V. Gornitz, S. Lebedeff, and J. Hansen, "Global Sea Level Trend in the Past Century," Science, 1982, p. 1611-1614.

Impact of Future Global Warming on Sea Level

Concern about a substantial rise in sea level as a result of the projected global warming stemmed originally from Mercer (1968), who suggested that the Ross and Filchner-Ronne ice shelves might disintegrate, causing a deglaciation of the the West Antarctic Ice Sheet and a resulting 6- to 7-m rise in sea level, possibly over a period as short as 40 years (see also Mercer 1978).

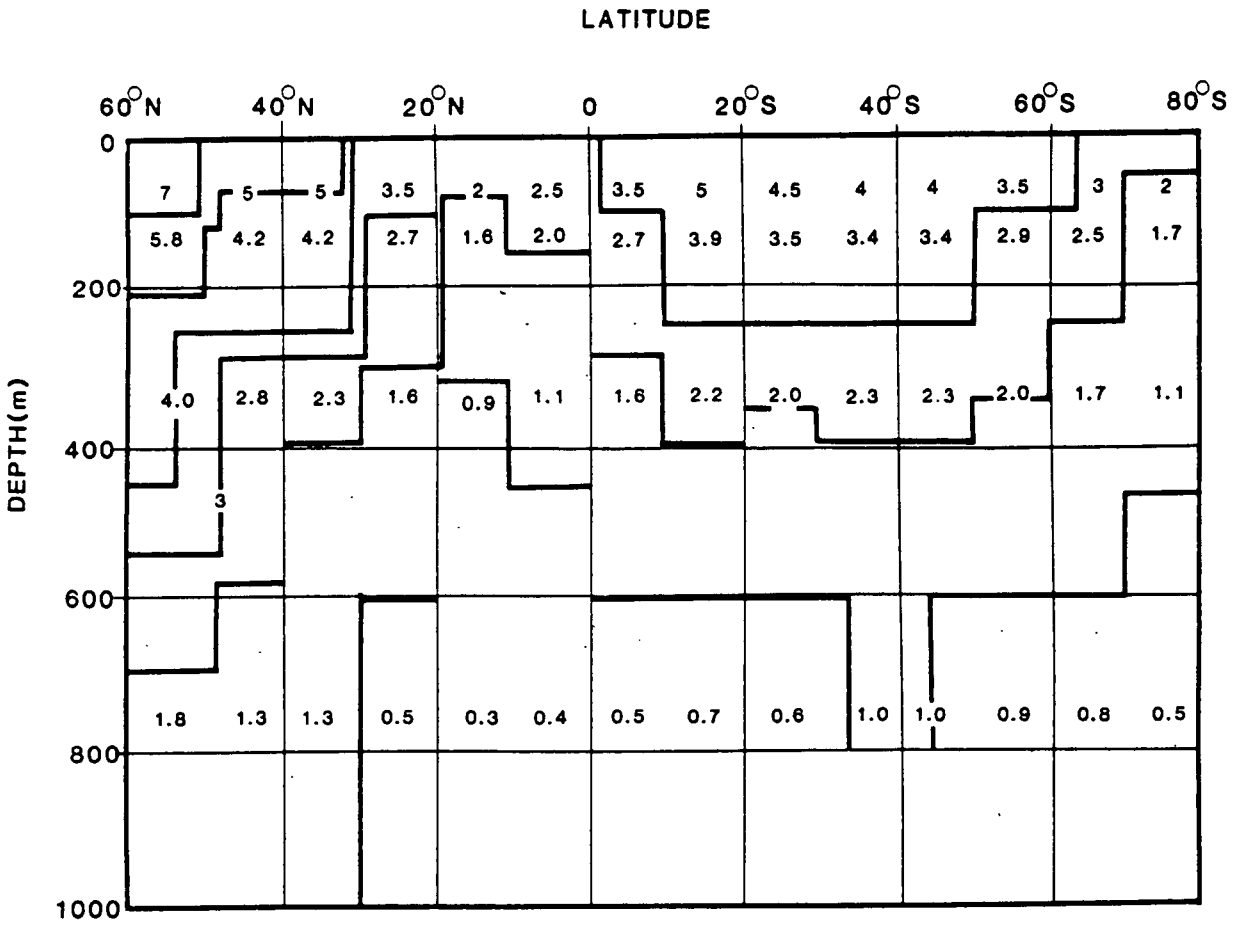
Subsequent investigations have concluded that such a rapid rise is unlikely. Hughes (1983) and Bentley (1983) estimated that such a disintegration would take at least 200 or 500 years, respectively. Other researchers have estimated that this process would take considerably longer (Fastook 1985; Lingle 1985).

Researchers have turned their attention to the magnitude of sea level rise that might occur in the next century. The best-understood factors are the thermal expansion of ocean water and the melting of alpine glaciers. In the National Academy of Sciences report "Changing Climate," Revelle (1983) used the model of Cess and Goldenberg (1981) to estimate temperature increases at various depths and latitudes resulting from a 4.2°C warming by the years 2050-2060 (Exhibit 15-3). Although his assumed time constant of 33 years probably resulted in a conservatively low estimate, he estimated that thermal expansion would result in an expansion of the upper ocean sufficient to raise sea level 30 cm.

Using a model of the oceans developed by Lacis et al. (1981), Hoffman, Wells and Titus (1986) examined a variety of possible scenarios of future emissions of greenhouse gases and global warming. They estimated that a warming of between 1° and 2.6°C could result in a thermal expansion contribution to sea level of between 12 and 26 cm by the year 2050. They also estimated that a global warming of 2.3 to 7.0°C by 2100 would result in a thermal expansion of 28 to 83 cm.

Revelle (1983) suggested that although he could not estimate the future contribution of alpine glaciers to sea level rise, a contribution of 12 cm through 2080 would be reasonable. Meier (1984) used glacier balance and volume change data for 25 glaciers for which the available record exceeded 50 years to estimate the relationship between historic temperature increases and the resulting negative mass balances of the glaciers. He estimated that a 28-mm rise had resulted from a warming of 0.5°C and concluded that a 1.5° to 4.5°C warming would result in a rise of 8 to 25 cm in the next century. Using these results, the Meier et al. (1985) concluded that the contribution of glaciers and small ice caps to a rise in sea level through 2100 is likely to be 10 to 30 cm. They noted that the gradual depletion of remaining ice cover might reduce the contribution of sea level rise somewhat. However, the contribution might also be greater because the historic rise took place over a sixty-year period, while the forecast period is over 100 years. Using Meier's estimated relationship between global warming and the alpine contribution, Hoffman, Wells, and Titus (1986) estimated alpine contributions through the year 2100 at 12 to 38 cm for a global warming of 2.3° to 7.0°C.

EXHIBIT 15-3



Computed average increase in ocean temperatures at particular depths and latitudes for a doubling of atmospheric carbon dioxide and probable increase in other greenhouse gases by the year 2080, based on Flohn's (1982) prognosis. Solid lines are isoplethis for temperature changes. Note that the top 100 m warms 2°-7°, while at depths of 800-1000 m the warming is mostly less than 1°.

Source: Revelle (1983).

The first published estimate of the contribution of Greenland meltwater to future sea level rise was Revelle's (1983) estimate of 12 cm through the year 2080. Using estimates by Ambach (1980 and 1982) that the equilibrium line (between snowfall accumulation and melting) rises 100 m for each 0.6°C rise in air temperature, Revelle concluded that the projected 6°C warming would be likely to raise the equilibrium line 1000 m. He estimated that such a change in the equilibrium line would result in a 12-cm contribution to sea level rise for the next century.

Meier et al. (1985) noted that the large ablation area makes Greenland a "significant potential contributor of meltwater to the ocean if climatic warming causes an increase in the rate of ablation and an upward shift of the equilibrium line." They found that a 1000-m rise in the equilibrium line would result in a contribution of 30 cm through the year 2100. However, because Ambach (1985) found the relationship between the equilibrium line and temperature to be 77 m per degree (C), the panel concluded that a 500 m shift in the equilibrium line would be more likely. Based on the assumption that Greenland will warm 6.5°C by the year 2050 and that temperatures will remain constant thereafter, they estimated that such a change would contribute about 10 cm to sea level through 2100, but also noted that "for an extreme but highly unlikely case, with the equilibrium line raised 1000 m, the total rise would be 26 centimeters." Bindschadler (1985) treated the two cases as equally plausible. However, his analysis was conducted before the results of Ambach (1985) were known and he has since indicated agreement with the findings of Meier et al. (1985).[4]

Available estimates of the Greenland contribution assume that all meltwater flows into the oceans and that the ice dynamics of the glaciers do not change. Meier et al. (1985) suggested that much of the water would refreeze and therefore decrease the contribution to sea level rise. Although a change in ice dynamics might imply additional deglaciation and eventually increase the rate of sea level rise, they concluded that such changes were unlikely to occur in the next century.

The potential impact of a global warming on Antarctica in the next century is the least certain of all the factors by which a global warming might contribute to sea level rise. Meltwater from East Antarctica might make a significant contribution by the year 2100. This source could be significant since global warming is expected to be amplified in polar regions, and large parts of East Antarctica are far enough from the poles to possibly warm up so that meltwater runoff is significant.. Unfortunately no one has estimated the likely contribution.[5] Several studies have examined "deglaciation," which also includes the contribution of ice sliding into the oceans. Bentley (1983) examined the processes by which a deglaciation of West Antarctica might occur. The first step in the process would be accelerated melting of the undersides of the Ross and Filchner-Ronne ice shelves caused by warmer water circulating underneath them. The thinning of these ice shelves could cause them to become unpinned and cause their grounding lines to retreat. Revelle (1983) concluded that the available literature suggests that the ice shelves might disappear in 100 years; the Antarctic ice streams would then flow directly into the oceans, without the

back pressure of the ice shelves. Hughes (1983) and Bentley (1983) estimate that a complete disintegration of the West Antarctic Ice Sheet could take an additional 200 and 500 years, respectively.

Although a West Antarctic deglaciation would occur over a period of centuries, it is possible that an irreversible deglaciation could commence before 2050. Thomas, Sanderson, and Rose (1979) suggested that if the ice shelves thinned more than about 1 m per year, the ice would move into the sea at a sufficient speed such that even a cooling back to the temperatures of today would not be sufficient to result in a reformation of the ice shelf.

To estimate the likely Antarctic contribution for the next century, Thomas (1985) developed four scenarios of the impact of a 3°C global warming by the year 2050:

(1) For a shelf melting rate of 1 m/year with seaward ice fronts remaining at present locations--implies a rise of 28 cm by the year 2100.

(2) For a shelf melting rate of 1 m/year with ice fronts calving back to a line linking the areas where the shelf is grounded, during the 2050s--implies a rise of 1.6 m by 2100.

(3) For a scenario similar to case 1 but with a melt rate of 3 m/year--implies a rise of 1 m by 2100.

(4) For a scenario similar to case 2 but with a melt rate of 3 m/year--implies a rise of 2.2 meters by 2100.

Thomas concluded that the 28-cm rise implied by case 1 would be most likely to occur. He also stated that even if enhanced calving did occur, it would be likely to occur after 2050, "suggesting that probably associated sea-level rise would be closer to the 1 m of case 3 than the 2.2 m of case 4."

Meier et al. (1985) evaluated the Thomas study and papers by Lingle (1985) and Fastook (1985). Although Lingle estimated that the contribution to sea level rise of West Antarctica through 2100 would be 3 to 5 cm, he did not evaluate East Antarctica; Fastook made no estimate for the year 2100. Thus, the panel concluded that "imposing reasonable limits" on the Thomas model yields a range of 20 to 80 cm by the year 2100 for the Antarctic contribution. However, they also noted several factors that would reduce the amount of ice discharged into the sea: removal of the warmest ice from the ice shelves, retreat of grounding lines, and increased lateral shear stress. They also concluded that increased precipitation over Antarctica might increase the size of the polar ice sheets there. Thus, they concluded that the contribution from Antarctica could cause a rise in sea level up to 1 m or a drop of 10 cm; a rise of between 0 and 30 cm is most likely.

Exhibit 15-4 summarizes the various estimates of future global sea level rise for specific years. Revelle (1983) estimated that the rise was likely to be 70 cm, ignoring the impact of a global warming on Antarctica. He also noted that the latter contribution was likely to be 1 to 2 m per century after 2050, but declined to add that to his estimate. Meier et al. (1985) projected that the contribution of glaciers would be sufficient to raise sea level 20 to 160 cm, with a rise of "several tenths of a meter" most likely. Thus, if one extrapolates from the Revelle (1983) estimate of thermal expansion through the year 2100, the Meier et al. report predicts a rise of between 50 and 200 cm. Using a range of estimates for future concentrations of greenhouse gases, the climate's sensitivity to such increases, oceanic heat uptake, and the behavior of glaciers, Hoffman, Wells, and Titus (1986) estimated that the rise would be 10 to 21 cm by 2025 and 57 to 368 cm by 2100.

Future Trends in Local Sea Level

Although most attention has focussed on projections of global sea level, impacts on particular areas would depend on local relative sea level. As Exhibit 15-5 shows, tidal guage measurements suggest that relative sea level has risen ten to twenty centimeters more rapidly along much of the U.S. coast than the worldwide average (Hicks, Debaugh, and Hickman 1983). Important exceptions include Louisiana, which is subsiding close to 1 m per century, and Alaska, which is emerging 10 to 100 cm per century.

Local subsidence and emergence are caused by a variety of factors. Rebound from the retreat of glaciers after the last ice age has resulted in the uplift of northern Canada, New England, and parts of Scandinavia; emergence in Alaksa is due more to tectonic adjustments. The uplift in polar latitudes has resulted in subsidence in other areas, notably the U.S. Atlantic and Gulf coasts. Groundwater pumping has caused rapid subsidence around Houston, Texas; Taipai, Taiwan; and Bangkok, Thailand, among other areas (Leatherman 1984). River deltas and other newly created land subside as the unconsolidated materials compact. Although subsidence and emergence trends may change in the future, particularly where anthropogenic causes are curtailed, no one has linked these causes to future climate change in the next century.

However, the removal of ice from Greenland and Antarctica would deform the ocean floor. Clark and Lingle have (1977) calculated the impact of a uniform 1 m contribution from West Antarctica. They concluded that relative sea level at Hawaii would rise 125 cm, and that along much of the U.S. Atlantic and Gulf Coasts, the rise would be 15 cm. On the other hand, sea level would drop at Cape Horn by close to 10 cm, and the rise along the southern half of the Argentine and Chilean coasts would be less than 75 cm.

Other contributors to local sea level that might change as a result of a global warming include currents, winds, and freshwater flow into estuaries. None of these impacts, however, has been estimated.

EXHIBIT 15-4

Estimates of Future Sea Level Rise (centimeters)

Year 2100 by Cause (Year 2085 for Revelle 1983):

	Thermal Expansion	Alpine Glaciers	Greenland	Antarctica	Total
Revelle (1983)	30	12	12	2	70
Hoffman et al. (1983)	28-115	b/	b/	b/	56-345
Meier et al. (1985) c/	-	10-30	10-30	-10-+100	50-200
Thomas (1985)	-	-	-	0-200	-
Hoffman et al. (1986)	28-83	12-37	6-27	12-220	57-368

Total Rise in Specific Years: d/

	2000	2025	2050	2075	2085	2100
Hoffman et al. (1983)						
Low	4.8	13	23	38	-	56.0
Mid-range low	8.8	26	53	91	-	144.4
Mid-range high	13.2	39	79	137	-	216.6
High	17.1	55	117	212	-	345
Hoffman et al. 1986						
Low	3.5	10	20	36	44	57
High	5.5	21	55	191	258	368

a/ Revelle attributes 16 cm to other factors.

b/ Hoffman et al. (1983) assumed that the glacial contribution would be one to two times the contribution of thermal expansion.

c/ NAS (1985) estimate includes extrapolation of thermal expansion from Revelle (1983).

d/ Only Hoffman et al. reports made year-by-year projections for the next century.

Sources: Hoffman et al. (1986); Meier et al. (1985);
Hoffman et al. (1983): Revelle (1983); Thomas (1985).

EXHIBIT 15-5

Local Sea Level Rise

	Historic a/ Relative Sea Level Trend (mm/yr)	Historic b/ Subsidence Rate (mm/yr)	Local Rise for c/ 1 Meter Global Rise by 2100 (cm)
Portland, Maine	2.3	1.1	112.6
Boston, Massachusetts	2.3	1.1	112.6
Newport, Rhode Island	2.6	1.4	116.1
New London, Connecticut	2.2	1.0	111.5
New York, New York	2.8	1.6	118.4
Sandy Hook, New Jersey	4.2	3.0	134.5
Atlantic City, New Jersey	4.0	2.8	132.2
Philadelphia, Pennsylvania	2.6	1.4	116.1
Baltimore Maryland	3.2	2.0	123.0
Annapolis, Maryland	3.7	2.5	128.8
Hampton Roads, Virginia	4.3	3.1	135.7
Charleston, South Carolina	3.4	2.2	125.3
Fernandina, Florida	1.7	0.5	105.8
Miami Beach, Florida	2.3	1.1	112.6
Cedar Key, Florida	2.0	0.8	109.2
Pensacola, Florida	2.4	1.2	113.8
Eugene Island, Louisiana	10.	8.8	201.2
Galveston, Texas	6.3	5.1	158.7
San Diego, California	1.9	0.7	108.0
Los Angeles, California	0.6	-0.6	93.1
San Francisco, Califonia	1.2	0	100.0
Astoria, Oregon	-0.5	-1.7	80.5
Seattle, Washington	1.9	0.7	108.5
Juneau, Alaska	-12.9	-14.1	-62.2
Sitke, Alaska	-2.4	-3.6	58.6
Worldwide	1.2	0	100.0

a/ Based on assumed global rise of 12 cm/century. From Hicks, Debaugh, and Hickman (1983).

b/ Relative sea level trend minus consensus estimate of 12 cm/century for global sea level rise.

c/ One meter plus extrapolation of historic subsidence rate.

Source: Hicks, Debaugh, and Hickman 1983.

EFFECTS OF SEA LEVEL RISE

A rise in sea level of 1 or 2 m would permanently innundate wetlands and lowlands, accelerate coastal erosion, exacerbate coastal flooding, threaten coastal structures, and increase the salinity of estuaries and aquifers. Substantial research has been done on the implications of sea level rise for coastal erosion and wetlands and relatively little work has been done in the other areas.

The remaining sections of this chapter summarize research on the impacts of sea level rise. The level of damage would depend in large measure on when and how people respond. Much of the research has been conducted to assist agencies that might have to respond to the impacts of sea level rise, and has attempted to estimate impacts for three alternative responses: (1) no countermeasures are taken, (2) measures are taken in response to sea level rise as it occurs, and (3) planning for sea level rise before it occurs. We follow this convention and examine potential impacts, possible responses, and the relative merits of anticipation.

Submergence of Coastal Wetlands

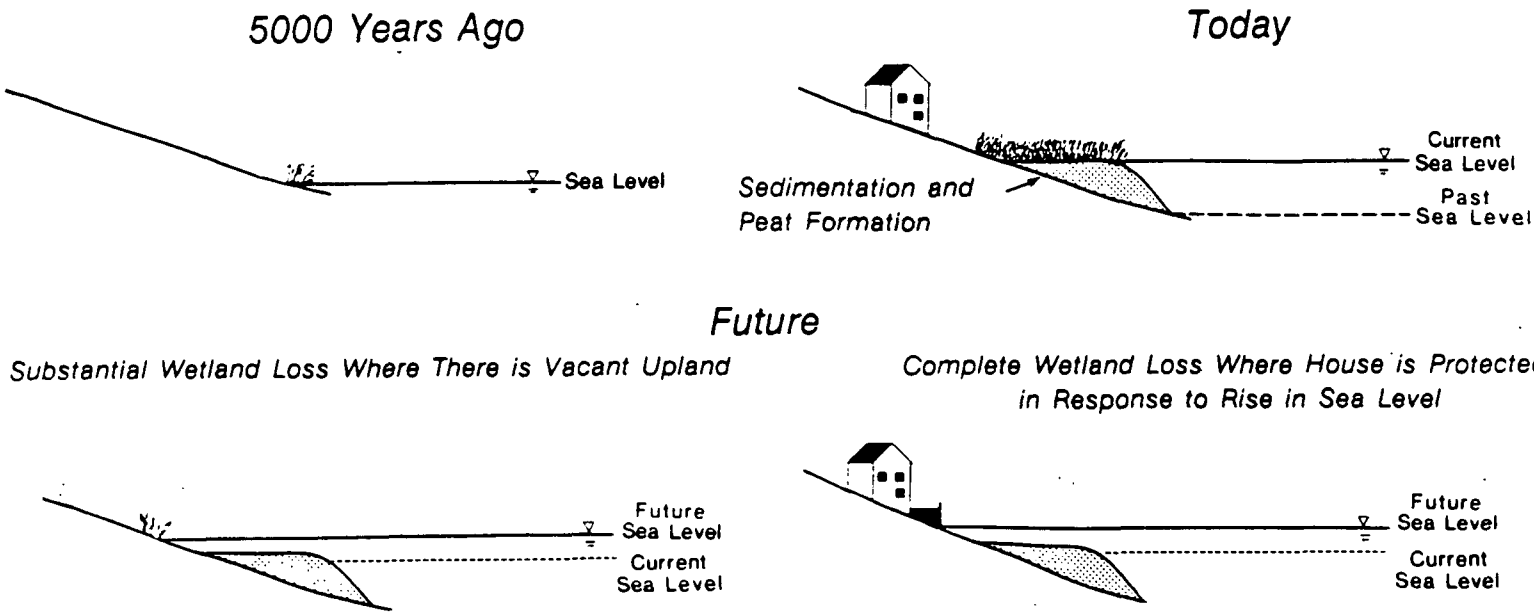
The most direct impact of a rise in sea level is the innundation of areas that had been just above the water level before the sea rose. Coastal wetlands are generally found at elevations below the highest tide of the year and above mean sea level. Thus, wetlands account for most of the land less than 1 m above sea level.

Because a common means of estimating past sea level rise has been the analysis of marsh peats, the impact of sea level rise on wetlands is fairly well-understood. For the last several thousand years, marshes have generally kept pace with the rate of sea level rise through sedimentation and peat formation (Emery and Uchupi 1972; Redfield 1972 and 1967; Davis 1985). As sea level rose, new wetlands formed inland while the seaward boundary was maintained. Because the wetland area has expanded, Titus, Henderson, and Teal (1984) hypothesized that one would expect a concave marsh profile, i.e., that there is more marsh area than the area found immediately above the marsh. Thus, if sea level rose more rapidly than the marsh's ability to keep pace, there would be a net loss of wetlands. Moreover, a complete loss might occur if protection of developed areas prevented the inland formation of new wetlands. (See Exhibit 15-6a.)

Kana et al. (1986 and n.d.) surveyed marsh transects in the areas of Charleston, South Carolina and two sites near Long Beach Island, New Jersey, to evaluate the concavity of wetland profiles and the vulnerability of wetlands to a rise in sea level. Their data showed that in the Charleston area, all of the marsh was between 30 and 110 cm above current sea level, an elevation range of 80 cm (see Exhibit 15-6b). The area with a similar elevation range just above the marsh was only 20% as large. Thus, a rise in sea level exceeding vertical marsh accretion by 80 cm would result in an 80% loss of wetlands. In the New Jersey sites, the marsh was also found within an elevation range of 80 cm; a rise in sea level 80 cm in excess of marsh accretion would result in 67% to 90% losses.

EXHIBIT 15-6a

Evolution of Marsh as Sea Level Rises



Coastal marshes have kept pace with the slow rate of sea level rise that has characterized the last several thousand years. Thus, the area of marsh has expanded over time as new lands were inundated. If in the future, sea level rises faster than the ability of the marsh to keep pace, the marsh area will contract. Construction of bulkheads to protect economic development may prevent new marsh from forming and result in a total loss of marsh in some areas.

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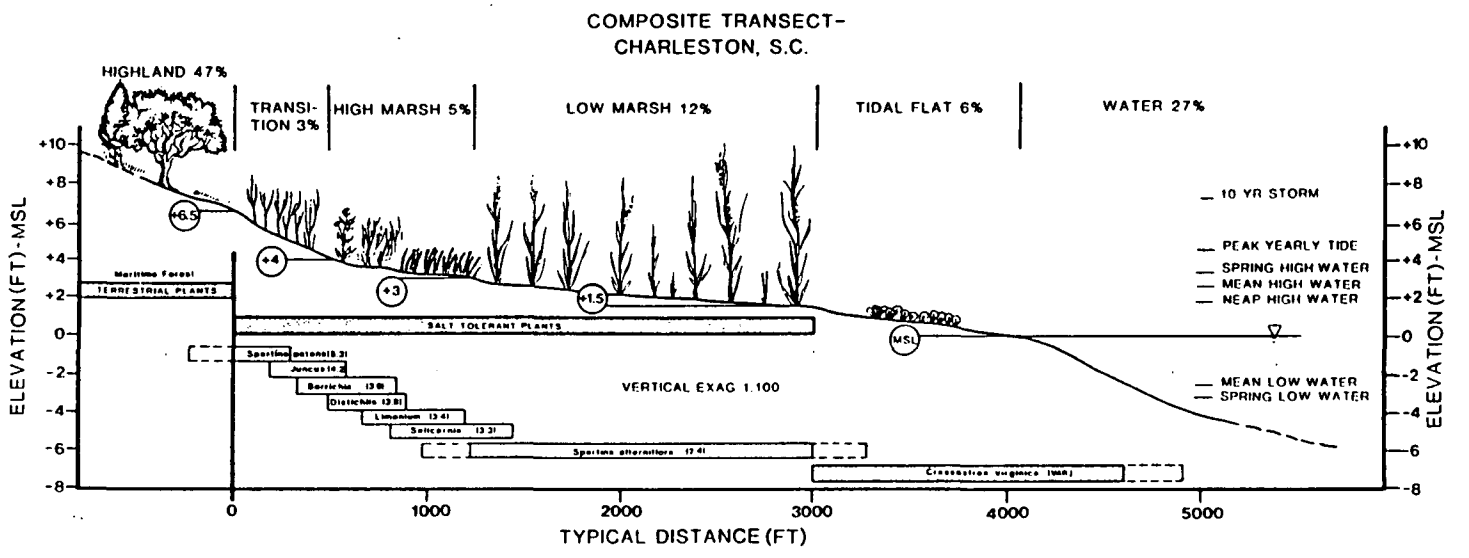
EXHIBIT 15-6b

Composite Transect -- Charleston, S.C.



Composite wetlands transect for Charleston area. Area above the marsh is much steeper than the marsh.

Source: Kana, Baca and William, 1986.

The future ability of marshes to accrete vertically is uncertain. Based on field studies by Ward & Domeracki (1978), Hatton et al. (1983), Meyerson (1972), Sterans & MacCreary (1957), and Kana et al. (1986) concluded that current vertical accretion rates are approximately 4-6 mm/year in the two case study areas, greater than the current rate of sea level rise but less than the rates of rise projected for the next century. If current accretion trends continue, rises of 87 and 160 cm by 2075 would imply 50% and 80% losses of wetlands, respectively, in the Charleston area. Kana et al. (n.d.) also estimated 80% losses in the New Jersey sites for a 160-cm rise through 2075. However, because the high marsh dominates in that area, they concluded that the principal impact of an 87-cm rise by 2075 would be the conversion of high to low marsh.

In both cases, the losses of marsh could be greater if inland areas are developed and protected with bulkheads or levees. Because there is a buffer zone between developed areas and the marsh in South Carolina, protecting development from a 160-cm rise would increase the loss from 80% to 90%. Without the buffer, the loss would be close to 100%.

The marshes and swamps of Louisiana, which account for 40% of the coastal wetlands in the United States (excluding Alaska), would be particularly vulnerable to an accelerated rise in sea level. The wetlands in this state are mostly less than 1 m above sea level, and are generally subsiding approximately 1 m per century as its deltaic sediments compact (Boesch 1982). Until the last century, the wetlands were able to keep pace with this rate of relative sea level rise because of the sediment conveyed to the wetlands by the Mississippi River.

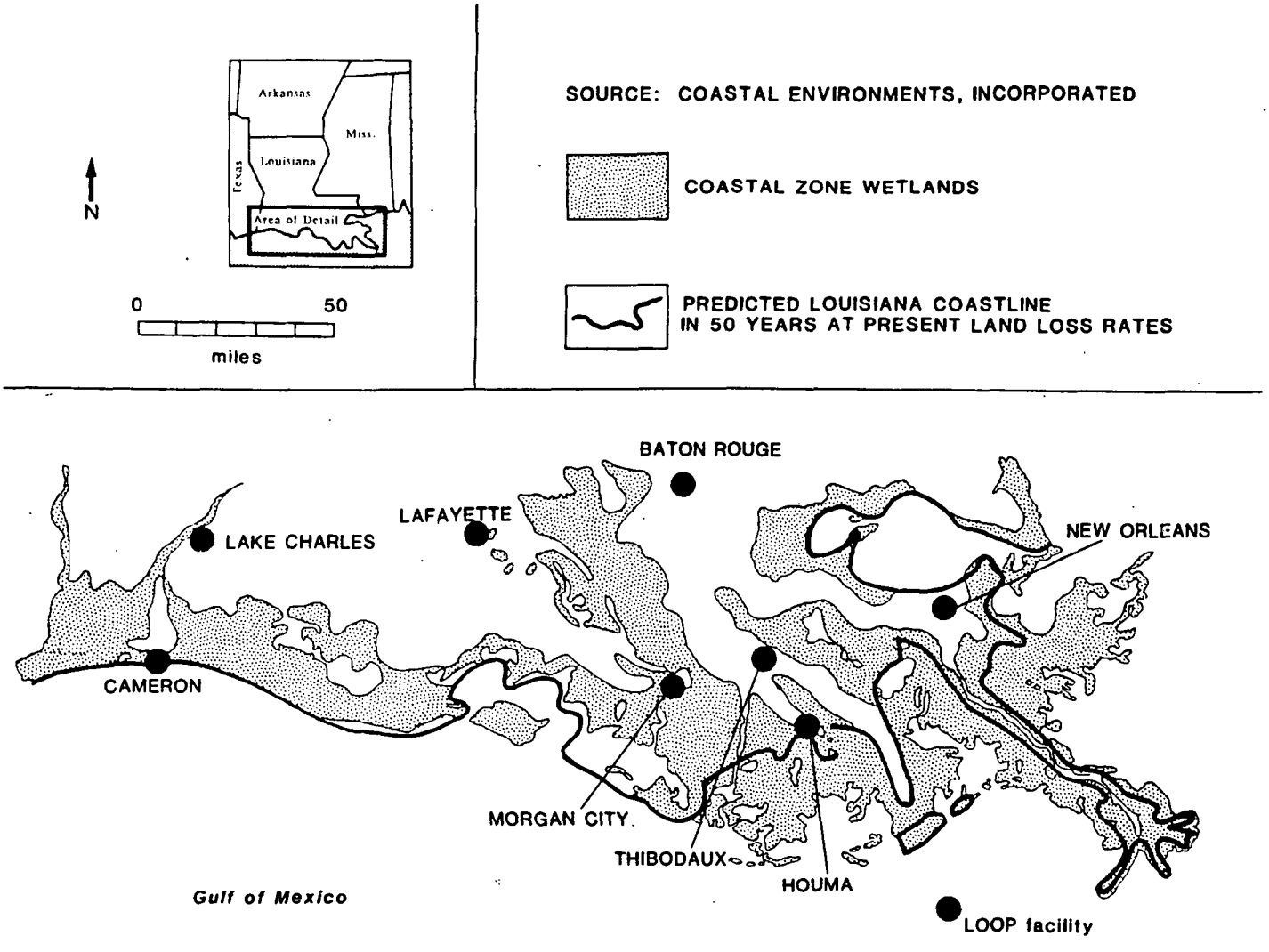
Human activities, however, have largely disabled the natural processes by which coastal Louisiana might keep pace with sea level rise. Dams, navigation channels, canals, and flood protection levees have interrupted the flow of sediment, freshwater, and nutrients to the wetlands. As a result, over 100 square kilometers of wetlands convert to open water every year (Gagliano, Meyer-Arendt, and Wicker 1981). A substantial rise in sea level would further accelerate the process of wetland loss in Louisiana (see Exhibit 15-7).

Throughout the world, people have dammed, leveed, and channelized major rivers, curtailing the amount of sediment that reaches river deltas. Even at today's rate of sea level rise, substantial amounts of land are converting to open water in Egypt and Mexico (Milliman and Meade 1983). Other deltas, such as the Ganges in Bangladesh and India, are currently expanding seaward. These areas would require increased sediment, however, to keep pace with an accelerated rise in sea level. Additional projects to divert the natural flow of river water would increase the vulnerability of these areas to a rise in sea level.

Broadhus et al. (1986) examined the possible impacts of future sea level rise on Egypt and Bangladesh, the inhabited areas of which are in the deltas of the Nile and Ganges Rivers. They estimated that 50 and 200 cm rises by 2100 would flood 0.35% and 0.7% of Egypt's land area, respectively. However, because the nation's population is concentrated in the low-lying areas, 16 and

EXHIBIT 15-7

Louisiana Shoreline in the Year 2030



The solid line shows the predicted shoreline for 2030 if current trends continue. The entire shaded area could be lostby 2085 if sea level rises 1 m. However, a 20 cm rise by 2020 would result in the shore retreating to this point by 2020 or sooner.

Source: Coastal Environments, Incorporated.

21 percent of the people currently reside in the areas that would be lost. Broadhus et al. (1986) also estimate that Bangladesh could lose 12% and 28% of its land, which currently houses 9% and 27% of its population (see Exhibit 15-8).

To develop an understanding of the potential nationwide impact of sea level rise on coastal wetlands in the United States, Park et al. (1986) used topographic maps to characterize wetland elevations at 52 sites comprising 4800 square kilometers (1.2 million acres) of wetlands, over 17% of all U.S. coastal wetlands. Using published vertical accretion rates, they estimated the impact of 1.4 m and 2.1 m rises in sea level through the year 2100 for each of the sites. Weighing their results according to the coastal wetland inventory by Alexander, Broutman, and Field (1986), Titus (n.d.) estimated a loss of wetlands between 47% and 82%, which could be reduced to between 31% and 70% if new wetlands are not prevented from forming inland.

Inundation

Although coastal wetlands are found at the lowest elevations, innundation of low land could also be important in some areas, particularly if sea level rises at least 1 m. Unfortunately, the convention of 10-ft contours in the mapping of most coastal areas has prevented a general assessment of land loss. Nevertheless, a few case studies have been conducted.

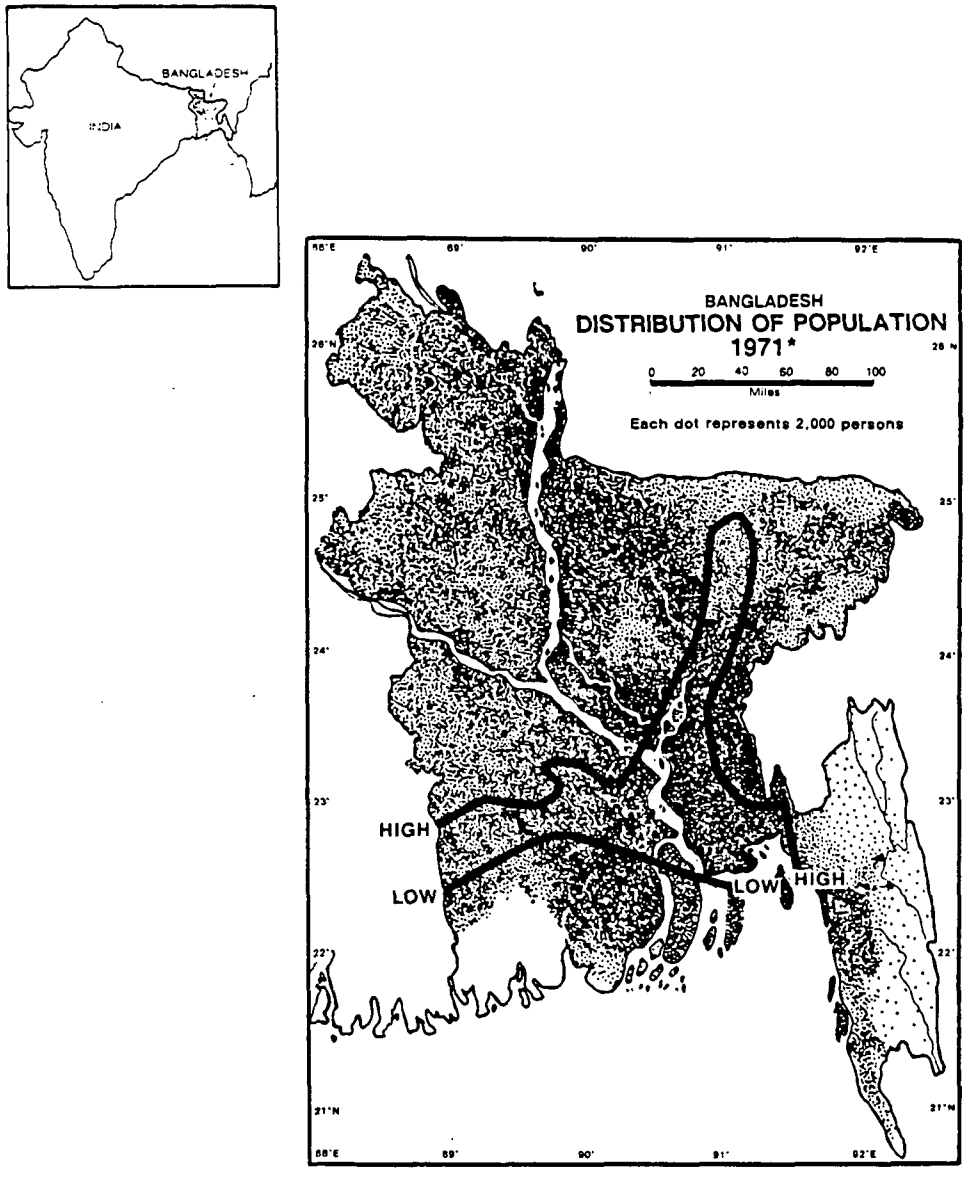
Kana et al. 1984 used data from aerial photographs to assess elevations in the area around Charleston. They concluded that 160 and 230 cm rises would result in 30% and 46% losses of the area's dry land, respectively. Leatherman (1984) estimated that such rises would result in 9% and 12% losses of the land in the area of Galveston and Texas City, Texas, assuming that the elaborate network of seawalls and levees were maintained.

The only nationwide assessment of the innundation from projected sea level rise was conducted by Schneider and Chen (1980). Unfortunately, the smallest rise in sea level they considered was a 4.5-m (15-ft) rise, in part because smaller contours are not generally available in topographic maps. Nevertheless, their findings suggest which coastal states would be most vulnerable: Louisiana (which would lose 28% of its land and 51% of its assets); Florida (24% and 52%); Delaware (16% and 18%); Washington, D.C. (15% and 15%); Maryland (12% and 5%); and New Jersey (10% and 9%).

As with wetland loss, the responses to innundation broadly fall into the categories of retreat and holding back the sea. Levees are used extensively in the Netherlands and New Orleans to prevent areas that are below sea level from being flooded and could be similarly constructed around other major cities. In lightly developed areas, however, the cost of a levee might be greater than the value of the property being protected. Moreover, even where levees prove to be cost-effective, the environmental implications of replacing natural shorelines with manmade structures would need to be considered.

EXHIBIT 15-8

Distribution of Population in Bangladesh



The high scenario represents 2 to 2.5 m rise with sedimentation disrupted by human activities. Low scenario represents 50 cm rise and natural sedimentation maintained.

Source: Broadhus et al. (1986).

Coastal Erosion

Sea level rise can also result in the loss of land above sea level through erosion. Bruun (1962) showed that the erosion resulting from a rise in sea level would depend upon the average slope of the entire beach profile extending from the dunes out to the point where the water is too deep for waves to have a significant impact on the bottom (generally a depth of about 10 m). (See Exhibit 15-9):

$$r = s * p/(h+d)$$

where r = shorelines retreat; s = sea level rise; p = horizontal length of the beach profile from dune to the offshore limit of significant wave action; h = height of the dune crest; and d = depth of the offshore limit of the beach profile. By comparison, innundation depends only on the slope immediately above the original sea level. Because beach profiles are generally flatter than the the portion of the beach just above sea level, the "Bruun Rule" generally implies that the erosion from a rise in sea level is several times greater than the amount of land directly inundated.

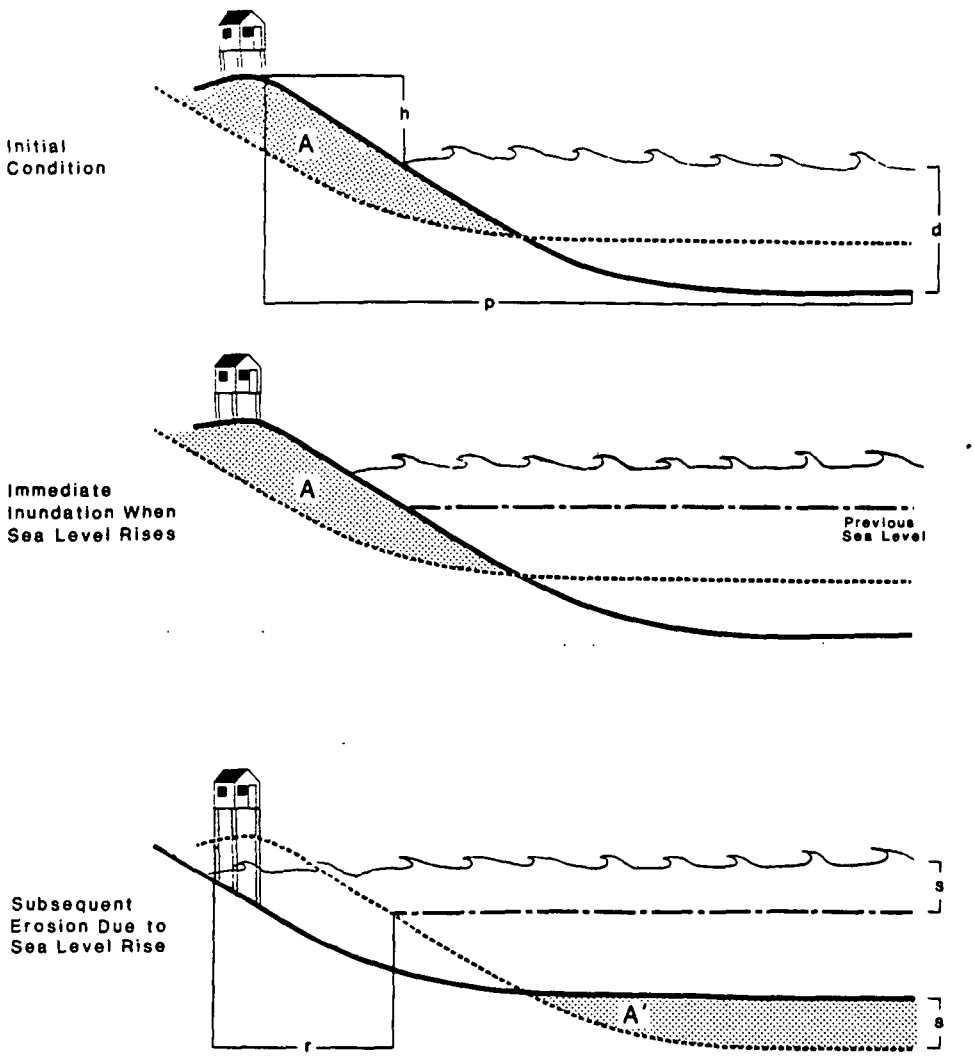
Processes other than sea level rise also contribute to erosion. These include storms, structures, currents, and alongshore transport. Because sea level has risen slowly in recent centuries, verification of the Bruun Rule on the open coast has been difficult; it has been difficult to distinguish erosion due to sea level rise from erosion caused by other processes. However, water levels along the Great Lakes can fluctuate over 1 m in a decade. Hands (1976, 1979, and 1980) and Weishar and Wood (1983) have demonstrated that the Bruun Rule generally predicts the erosion resulting from rises in water levels there.

The Bruun Rule has been applied to project erosion due to sea level rise for several areas. Bruun (1962) found that a 1 cm rise in sea level would generally result in a 1 m shoreline retreat, but that the retreat could be as great as 10 m along some parts of the Florida coast. Everts (1985) and Kyper and Sorensen (1985) however, found that along the coasts of Ocean City, Maryland and Sandy Hook, New Jersey, respectively, the shoreline retreat implied by the Bruun Rule would be only about 75 cm. Kana et al. (1984) found that along the coast of South Carolina, the retreat could be 2 m. The U.S. Army Corps of Engineers (1979) indicated that along the coast of San Franciso, where waves are generally larger than those found along the Atlantic Coast, the shore might retreat 2 to 4 m for a 1-cm rise in sea level.

Dean and Maurmeyer (1983) generalized the "Bruun Rule" approach to consider the "overwash" of barrier islands. Coastal geologists generally believe that coastal barriers can maintain themselves in the face of slowly rising sea level through the landward transport of sand, which washes over the island during storms, building the island upward and landard. Because this formulation of the Bruun Rule extends the beach profile horizonatally to include the entire islands as well as the active surf zone, it always predicts greater erosion than the Bruun Rule. However, the formulation may not be applicable to developed barrier islands, where the common practice of public officials is to bulldoze sand back onto the beach after a major storm.

EXHIBIT 15-9

The Bruun Rule



A rise in sea level of S causes immediate inundation. However it would eventually require the offshore bottom to rise by S. The necessary S and A' would be supplied from the upper part of the beach A. Total shoreline retreat r is equal to S * p/(h+d).

The potential erosion from a rise in sea level could be particularly important to recreational beach resorts, which include some of the nation's most economically valuable and intensely used land. Although nationwide statistics are not available, information on particular locations is instructive. Every weekend in the summer, approximately 250,000 people visit Ocean City, Maryland to sunbathe on its 15-km (9-mile) beach.[6] Along most of the Ocean City shoreline, the beach is less than 15 m wide during high tide--a width that is typical of the most extensively used beach resorts. Relatively few of the most intensely developed resorts along the Atlantic Coast have beaches wider than about 30 m at high tide. Thus, the rise in relative sea level of 30 cm projected in the next 40 to 50 years could erode most recreational beaches in developed areas, unless additional erosion response measures are taken.

Flooding and Storm Damage

A rise in sea level could increase flooding and storm damages in coastal areas for three reasons: erosion caused by sea level rise would increase the vulnerability of communities; higher water levels would provide storm surges with a higher base to build upon; and higher water levels would decrease natural and artificial drainage.

The impact of erosion on vulnerability to storms is generally a major consideration in proposed projects to control erosion, most of which have historically been funded through the U.S. Army Corps of Engineers. The impact of sea level rise, however, has not generally been considered separately from other causes of erosion.

The impact of higher base water levels on flooding has been investigated for the areas around Charleston, South Carolina and Galveston, Texas (Barth and Titus 1984). Kana et al. (1984) found that around Charleston, the area within the 10-year flood plain would increase from 33% in 1980, to 48%, 62%, and 74% for rises in sea level of 88, 160, and 230 cm, respectively, and that the area within the 100-year flood plain would increase from 63% to 76%, 84%, and 90% for the three scenarios.[9] Gibbs (1984) estimated that a rise of about 90 cm would double the average annual flood damages in the Charleston area (but that flood losses would not increase substantially for higher rises in sea level because shoreline retreat would result in a large part of the community being completely abandoned).[9]

Leatherman (1984) conducted a similar analysis of Galveston Island, Texas. He estimated that the area within the 100-year flood plain would increase from 58% to 94% for an 88 cm rise in sea level,[9] and that for a rise greater than 1 m, the Galveston seawall would be overtopped during a 100-year storm. Gibbs estimated that the damage from a 100-year storm would be tripled for a rise of about 90 cm.[9]

A wide variety of shore protection measures would be available for communities to protect themselves from increased storm surge and wave damage due to sea level rise (Sorensen, Weisman, and Lennon 1984). Many of the measures used to address erosion and inundation, including seawalls,

breakwaters, levees, and rebuilding beaches also provide protection against storms. In Galveston, which is already protected on the ocean side by the seawall, Gibbs hypothesized that it might be necessary to completely encircle the developed areas with a levee to prevent flooding from the bay side; upgrading the existing seawall might also be necessary.

Kyper and Sorensen (1985 and n.d.) examined the implications of sea level rise for the design of coastal protection works at Sea Bright, New Jersey, a coastal community that is currently protected by a seawall and has no beach. Because the seawall is vulnerable to even a 10-year storm, the Corps of Engineers and the State of New Jersey have been considering a possible upgrade. Kyper and Sorensen estimated that the cost of upgrading the seawall for current conditions would be 3.5 to 6 million dollars per kilometer of shoreline, noting that if designed properly, the seawall would be useful throughout the next century. However, they estimated that a rise in relative sea level of 30-40 cm would be likely to result in serious damage to the seawall during a major storm because of higher water levels and the increased wave heights resulting from the erosion of submerged sand in front of the seawall. To upgrade the seawall to withstand a 1-m rise in relative sea level would cost 5.7 to 9 million dollars per kilometer (50% more). They concluded that policy makers would have to weigh the tradeoff between the cost of designing the wall to withstand projected sea level rise and the cost of subsequent repairs and a second overhaul.

In addition to community-wide engineering approaches, measures can also be taken by individual property owners to prevent increased flooding. In 1968, the U.S. Congress created the National Flood Insurance Program to encourage communities to avoid risky construction in flood-prone areas. In return for requiring new construction to be elevated above expected flood levels, the federal government provides flood insurance, which is not available from the private sector. If sea level rises, flood risks will increase. In response, local ordinances will automatically require new construction to be further elevated and insurance rates on existing properties will rise unless those properties are further elevated. As currently organized, the National Flood Insurance Program would react to sea level rise as it occurred. Various measures to enable the program to anticpate sea level rise have been proposed, including warning policy holders that rates may increase in the future if sea level rises, denying coverage to new construction in areas that are expected to be lost to erosion within the next 30 years, and setting premiums according to the average risk expected over the lifetime of the mortgage (Howard, Pilkey, and Kaufman 1985; Titus 1984).

Case studies in Charleston, South Carolina and Fort Walton Beach, Florida have examined the implications of sea level rise for rainwater flooding and the design of coastal drainage systems. Waddell and Blaylock (n.d.) estimated that a 25-year rainstorm (with no storm surge) would result in no damage for the Gap Creek watershed in Fort Walton Beach. However, a rise in sea level of 30 to 45 cm would result in damages of 1.1-1.3 million dollars in this community of 4,000 residents during a 25-year storm. An upgrade costing $550,000, however, would prevent such damages.

LaRoche and Webb (n.d.) had previously developed the master drainage plan for Charleston, South Carolina and later evaluated the implications of sea level rise for the Grove Street watershed in that community. They estimated that the cost of upgrading the system for current conditions would be $4.8 million and the cost of upgrading the system for a 30-cm rise would be $5.1 million. If the system is designed for current conditions and sea level rises, the system would be deficient and the city would face a retrofit cost of $2.4 million. Thus, for the additional $300,000 necessary to upgrade for a 30-cm rise, the city could ensure that it would not have to spend an additional $2.4 million later. Noting that the decision whether to design now for a rise in sea level depends on the probability that sea level would rise, they concluded that a 3% real social discount rate would imply that designing for sea level rise is worthwhile if the probability of a 30-cm rise by 2025 is greater than 30%. At a discount rate of 10%, they concluded, designing for future conditions is not worthwhile.

Increased Salinity in Estuaries and Aquifers

Although most researchers and the general public have focused on the increased flooding and shoreline retreat associated with a rise in sea level, the inland penetration of saltwater could be important in some areas.

A rise in sea level increases the salinity of an estuary by altering the balance between freshwater and saltwater forces. The salinity of an estuary represents the outcome of (1) the tendency for the ocean salt water to completely mix with the estuarine water and (2) the tendency of freshwater flowing into the estuary to dilute the saline water and push it back toward the ocean. During droughts, the salt water penetrates upstream; during the rainy season, low salinity levels prevail. A rise in sea level has an impact similar to decreasing the freshwater inflow. By widening and deepening the estuary, sea level rise increases the ability of saltwater to penetrate upstream.

The implications of sea level rise for increased salinity have only been examined in detail for Louisiana and the Delaware Estuary. In Louisiana, saltwater intrusion is currently resulting in the conversion of cypress swamps--which can not tolerate saltwater--to open water lakes, as well as the conversion of fresh and intermediate marsh to marsh with higher salinity levels. In response to these trends, numerous projects have been proposed to divert freshwater from the Mississippi River to these wetlands (Roberts et al. 1983; U.S. Army Corps of Engineers 1982; van Beek et al. 1982). Although the cause of the saltwater intrusion has been primarily the dredging of canals and the sealing off of river distributaries that once provided the wetlands with fresh water, relative sea level rise is gradually increasing the saltwater force in Louisiana's wetlands; a further rise in sea level would accelerate this process. Haydl (1984) also concluded that subsidence may be resulting in increased salinity of drinking and irrigation water supplies in some parts of Louisiana.

The impact of current sea level trends on salinity has been considered in the long-range plan of the Delaware River Basin Commission since 1981 (DRBC 1981). The drought of the 1960s resulted in salinity levels that almost contaminated the water supply of Philadelphia and surrounding areas. Hull and Tortoriello (1979) found that the 13-cm rise projected between 1965 and 2000 would result in the "salt front" migrating 2 to 4 km farther upstream during a similar drought. They found that a moderately sized reservoir (57 million cubic meters) to augment river flows would be needed to offset the resulting salinity increases.

Hull, Thatcher, and Tortoriello (1986) examined the potential impacts of an accelerated rise in sea level due to the greenhouse warming. They estimated that 73-cm and 250-cm rises would result in the salt front migrating an additional 15 and 40 kilometers, respectively, during a repeat of the 1960s drought. They also found that the health-based 50 ppm sodium standard (equivalent to 73 ppm chloride) adopted by New Jersey would be exceeded 15% and 50% of the time, respectively, and that the EPA drinking water 250-ppm chloride standard would be exceeded over 35% of the time in the high scenario (see Exhibit 15-10).

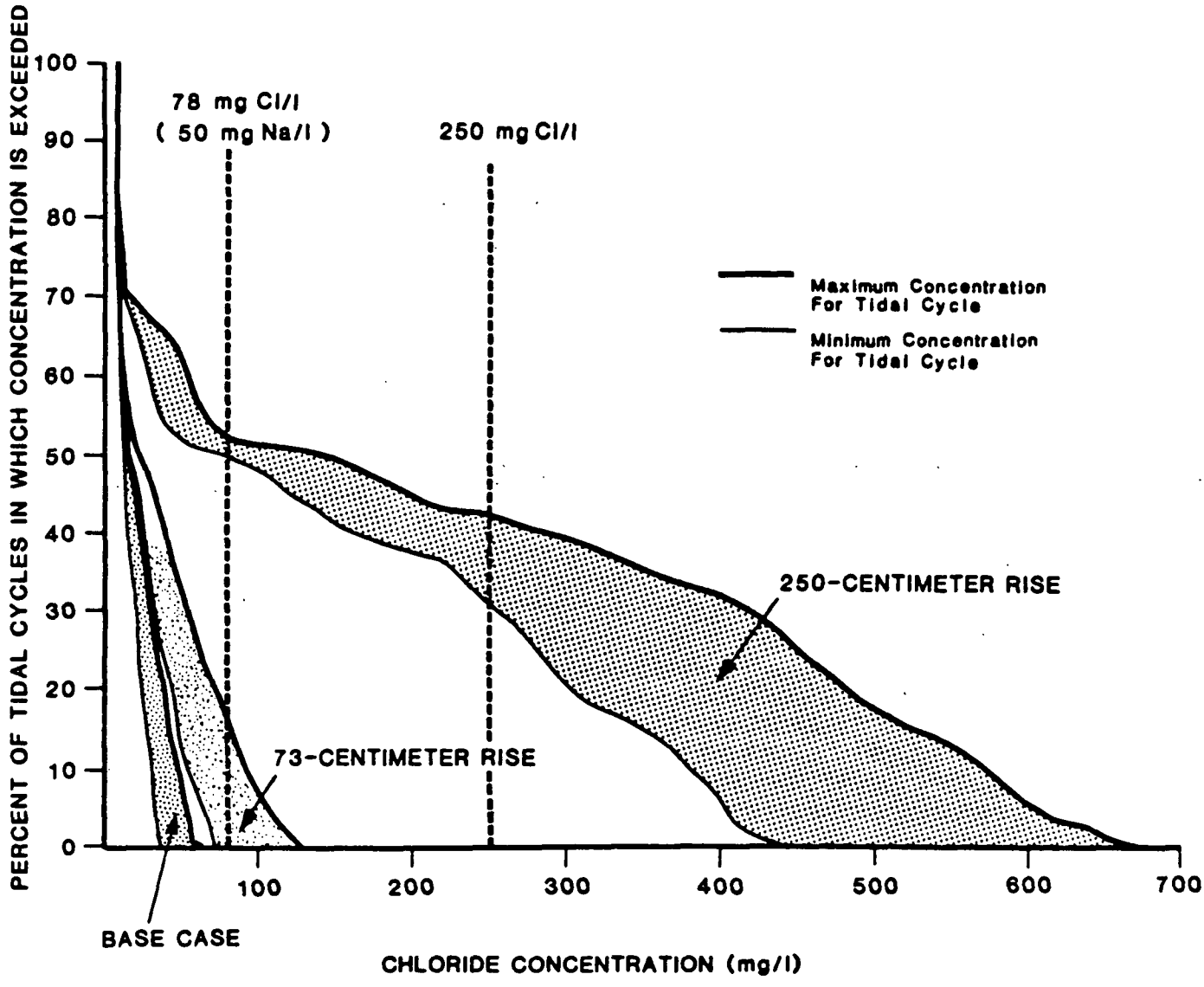
Lennon, Wisniewski, and Yoshioka (1986) examined the implications of increased estuarine salinity for the Potomac-Raritan-Magothy aquifer system, which is recharged by the (currently fresh) Delaware River and serves the New Jersey suburbs of Philadelphia. During the 1960s drought, river water with chloride concentrations as high as 150 ppm recharged these aquifers. Lennon et al. estimated that a repeat of the 1960s drought with a 73-cm rise in sea level would result in river water with concentrations as high as 350 ppm recharging the aquifer, and that during the worst month of the drought, over one-half of the water recharging the aquifer system would have concentrations greater than 250 ppm. With a 250-cm rise, 98% of the recharge during the worst month of the drought would exceed 250 ppm, and 75% of the recharge would be greater than 1000 ppm.

Hull and Titus (1986) examined the options by which various agencies might respond to increased salinity in the Delaware estuary. They concluded that planned but unscheduled reservoirs would be more than enough to offset the salinity increased from a 1-ft rise in sea level, although those reservoirs had originally been intended to meet increased consumption. They noted that construction of the reservoirs would not be necessary until the rise became more imminent. However, they also suggested that, given the uncertainties, it might be advisable today to identify additional reservoir sites to ensure that future generations retained the option of building additional reservoirs if necessary.

A rise in sea level could increase salinities in other areas, although the importance of those impacts has not been investigated. Kana et al. (1984) and Leatherman (1984) made preliminary inquiries into the potential impacts on coastal aquifers around Charleston and Galveston, respectively. However, they concluded that in-depth assessments were not worthwhile because the aquifers around Charleston are already salt-contaminated because of overpumping, and pumping of groundwater has been prohibited in the Galveston

EXHIBIT 15-10

Percent of Tidal Cycles in Which Specified Concentration is Exceeded at Torresdale During a Recurrence of the 1960's Drought for Three Sea Level Scenarios



The 250 mg Cl/l level is the EPA drinking water standard for chloride. The 50 mg/l Na level is the New Jersey sodium standard.

Source: Hill et al. 1981.

area due to the resulting land subsidence. The potential impacts on Florida's Everglades and the shallow aquifers around Miami might be significant, but they have not been investigated.

Economic Significance of Sea Level Rise

No one has estimated a dollar value of the likely impacts of sea level rise for the nation or any coastal state. Schneider and Chen (1980) estimated the economic impact of what was once (but is no longer) thought to be a plausible scenario: rises of 4.6 to 7.6 m (15 to 25 ft) occurring with little or no warning during the early part of the twenty-first century. They estimated that this scenario would result in real property losses of 100 to 150 billion dollars, representing 6.2% to 8.4% of all real property in the nation.

The only comprehensive attempt to place a dollar value on the impacts of sea level rise for particular communties was the study by Gibbs (1984) of the Charleston and Galveston areas. In addition to considering more realistic scenarios ranging from 0.9 to 2.4 m through 2075, Gibbs made several improvements on the approach of Schneider and Chen, differentiating between the economic impact if sea level rise was expected and the impact resulting from reacting to it as it occurs; modeling how investment decisions might respond to floods and erosion; and explicitly considering community-wide strategies to limit losses, including shore protection and abandonment.

In the Charleston study, Gibbs evaluated the implications of (1) efforts to avoid development of some vacant suburban areas likely to be flooded in the future; (2) a partial abandonment; and (3) elevating the existing seawalls protecting Charleston to provide additional protection. For a rise of 28-64 cm through 2025, Gibbs estimated that present value of the cumulative impact would be $280-1065 million (5% to 19% of economic activity in the area for the period), which could be reduced to $160-420 million if sea level rise was anticipated. Most of this impact would result from a 10% to 100% increase in expected storm damages, although Gibbs also estimated losses to erosion at $7-35 million.[9] For the period between 1980 and 2075, Gibbs estimated that the economic impacts would be $1250-2510 million (17% to 35%) and could be reduced $440-1100 million through anticipatory measures. If accurate, these estimates suggest that Charleston would be extremely vulnerable to a rise in sea level. However, these estimates were necessarily based on a number of simplifying assumptions regarding storm damage and current elevation of structures.

Gibbs performed a similar analysis of the Galveston area and concluded that the impacts of sea level rise through 2025 would represent $115-360 million (1.1%-3.6%) if not anticipated, and $90-140 million if anticipated. The impacts through 2075 were estimated at $555-1840 million (4.9%-16%) if not anticipated, and $310-730 million if anticipated. The assumptions Gibbs used suggest that a 100-year storm today would result in damages equal to about 7% of the property in the area, and that average annual storm damages are equal to $6 million, slightly less than 0.2% of the value of property in the study area.

Other studies can be used to gain an understanding of the economic significance of particular classes of impacts. As discussed above, a 30-cm rise in sea level would erode most recreational beaches back to the first row of houses. The studies cited in the section on erosion indicate that the typical beach profile extends out about 1000 m, which suggests that 300,000 cubic yards of sand per kilometer of shoreline are required to raise the beach profile 30 cm. If sand costs are typically $3-10/cubic meter, the beach rebuilding costs of a 30 cm rise in sea level would be $1 to 3 million per kilometer. If the United States has a few thousand kilometers of recreational beaches, it would cost billions and perhaps tens of billions of dollars to rebuild the beach in response to a 30-cm rise in sea level. This estimate considers only the beaches themselves; raising people's lots to avoid inundation would further increase the costs.

The U.S. Army Corps of Engineers (1971) estimated that in 1971, 25,000 km of shoreline (exclusive of Alaska, Great Lakes, and Hawaii) were eroding, of which 17% were "critically eroding," and would require engineering solutions. If 17% of all shorelines require erosion control, that would imply protection of close to 10,000 km of shoreline. Sorensen (1986) describes dozens of engineering options for preventing erosion, the least expensive of which costs $300,000 per kilometer, which would imply a cost of at least three billion dollars for protecting all shorelines.

Relationship to Other Impacts of the Greenhouse Warming

The impacts of sea level rise on coastal areas, as well as their importance, is likely to depend in part on other impacts of the greenhouse warming. Although future sea level is uncertain, there is a general consensus that a global warming would cause sea level to rise; by contrast, the direction of most hydrological and storm event changes at particular locations is unknown.

Warmer temperatures might change the ability of wetlands to keep pace with sea level rise. Mangrove swamps would replace salt marshes in some areas that are currently too cold for mangroves. No one has assessed the impact of this vegetation change on vertical accretion. Peat formation is generally greater in New England marshes than at lower latitudes. Warmer temperatures might reduce that rate of vertical accretion for these wetlands.

Changing climate could alter the frequency and tracks of storms. Because hurricane formation requires water temperatures of 27°C or higher (Wendland 1977), a global warming might result in an extension of the hurricane season and in the formation of hurricanes at higher latitudes. Besides increasing the amount of storm damage, increased frequency of severe storms would tend to flatten the typical beach profile, causing substantial shoreline retreat unless additional sand was placed on the beach. A decreased frequency of severe winter storms might have the opposite impact at higher latitudes.

Changes in precipitation could also affect the impacts of sea level rise. Because warmer temperatures would intensify the hydrologic cycle, it is generally recognized that a global warming would increase rainfall

worldwide. If rainfall in maritime environments increases, rainwater flooding could be increased because of decreased drainage and increased precipitation. The impact of sea level rise on saltwater intrusion could be offset by decreased drought frequency or exacerbated by increased drought frequency (Rind and Lebedeff 1984).

CONCLUSION

Researchers have identified a wide variety of impacts that could result from a 50 to 200 cm rise in sea level. These impacts include both economic losses resulting from land loss, flooding, shore protection, and increased costs for drinking water, as well as environmental costs such as the disruption of coastal ecosystems resulting from wetland loss and increased salinities. Although there are substantial uncertainties regarding the magnitude of both the causes and effects of future sea level rise, even conservative estimates suggest that important impacts are likely to result.

A high priority for future research should be the use of existing case studies to derive estimates of the nationwide and worldwide magnitude of the various impacts that have been identified. Although preliminary estimates of the potential loss of U.S. coastal wetlands exist, the implications of those losses have not been assessed. For most other impacts, no natioinwide estimates are available. That type of information will be important for evaluating the benefits of possible actions to mitigate the greenhouse warming and resulting rise in sea level.

NOTES

1. Mark Meier, University of Colorado (personal communication).

2. The only true measure of absolute sea level would be the rise of sea level relative to the center of the earth. Unfortunately, no such measurements are yet available. Therefore, researchers have had to combine tidal guage measurements of relative sea level trends at various locations, filter out known movements of the land surface, and take weighted averages to arrive at estimates of global sea level trends.

3. This result was reported in the North America study. The data also show this to be true in the Northern Europe study, but the result was not reported. David Aubrey, Woods Hole Oceanographic Institute, Woods Hole, Massachusetts (personal communication).

4. Robert Bindschadler, Goddard Space Flight Center, Greenbelt, Maryland (personal communication).

5. James Hansen, Goddard Insitute for Space Studies, New York (personal communication).

6. Sandy Coyman, Department of Planning and Community Development, Town of Ocean City, Maryland (personal communication).

7. North Carolina Administrative Code, Chapter 7H, 1983. Raleigh, North Carolina: Office of Coastal Management.

8. Fred Michaud, Office of Floodplain Management, State of Maine (personal communication).

9. These results are reported in an Appendix by Titus et al. 1984

10. See Note 9.

APPENDIX A

The sea level rise damages reported for Charleston and Galveston in Chapter 18 were derived from the following data taken from Gibbs (1984).

Scenario	Global Sea Level Rise by 2075 (cm)	Charleston Impact a/		Galveston Impact a/	
		With Anticipation	Without Anticipation	With Anticipation	Without Anticipation
Trend	11.4	b/	b/	b/	b/
Low	75.2	440	1250	310	555
Medium	146.8	730	1910	415	965
High	219.2	1100	2510	730	1890

a/ Through 2075 in bilions of dollars discounted at 3%.

b/ All estimates are relative to the trend scenario.

The damage estimates consist of a low estimate and a high estimate. The low estimate is derived by linearly interpolating the "with anticipation" estimates for Charleston and Galveston according to the amount of global sea level rise. The high estimates are derived by linearly interpolating the "without anticipation" estimates.

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Chapter 16

CHAPTER 16

POTENTIAL EFFECTS OF FUTURE CLIMATE CHANGES ON FORESTS AND VEGETATION, AGRICULTURE, WATER RESOURCES, AND HUMAN HEALTH

SUMMARY

The greenhouse effect resulting from increased levels of CO2, chloroflurocarbons, methane, N2O, and other trace gases in the atmosphere has been recognized by the scientific community for several decades as a potential cause of future climate change. In the last few years, estimates of the rate of change of these gases in the atmosphere has heightened concern about global warming and associated climate and environmental change. Chapter 6 -- Global Warming--presents a review of recent chemical and physical evidence supporting the greenhouse phenomenon. From this evidence it is generally concluded that in the relatively short period of time of the next 50-100 years the earth's climate will undergo important changes. These include: potential increases in temperatures, changes in precipitation, humidity, windfields, ocean currents, and the frequency of extreme events such as hurricanes. Furthermore, these climate parameters will induce still other shifts in sea levels, ice margins, the hydrologic cycle, air pollution episodes and other phenomenon.

Recently the World Meterological Organization, the United Nation's Environmental Programme and the International Council of Scientific Union summarized current scientific data on global climate change. These findings are presented in Exhibit 16-1. Similar findings have been reported by NAS (1979 and 1982).

EXHIBIT 16-1

SUMMARY OF FINDINGS FROM THE WMO/UNEP/ICSU CONFERENCE ON GLOBAL CLIMATE HELD IN VILLACH, AUSTRIA, OCTOBER 1985

- Many important economic and social decisions are being made today on long-term projects -- major water resource management activities such as irrigation and hydro-power; drought relief; agricultural land use; structural designs and coastal engineering projects; and energy planning -- all based on the assumption that past climatic data, without modification, are a reliable guide to the future climate conditions. This is no longer a valid assumption, since the increasing concentrations of greenhouse gases are expected to cause a significant warming of the global climate in the next century. It is a matter of urgency to refine estimates of future climate conditions to improve these decisions.

- The amounts of some trace gases in the troposhere, notably carbon dioxide (CO2), nitrous oxide (N2O), methane (CH4), ozone (O3), and chloro-fluorocarbons (CFC), are increasing. These gases are essentially transparent to incoming short-wave solar radiation, but absorb and emit longwave radiation and are thus able to influence the earth's climate.

- The role of greenhouse gases other than CO2 in changing the climate is already about as important as that of CO2. If present trends continue, the combined concentrations of atmospheric CO2 and other greenhouse gases would be (radiatively) equivalent to a doubling of CO2 from pre-industrial levels, possibly as early as the 2030's.

- The most advanced experiments with general circulation models of the climatic system show increases of the global mean equilibrium surface temperature of between 1.5°C and 4.5°C for a doubling of the atmospheric CO2 concentration or equivalent. Because of the complexity of the climatic system and the imperfections of the models, particularly with respect to ocean-atmosphere interactions and clouds, values outside of this range cannot be excluded. The realization of such changes will be slowed by the inertia of the oceans; the delay in reaching the mean equilibrium temperatures corresponding to doubled greenhouse gas concentrations is expected to be a matter of decades.

- While other factors such as aerosol concentrations, changes in solar energy input, and changes in vegetation may also influence climate, the increased amounts of greenhouse gases are likely to be the most important cause of climate change over the next century.

- Regional scale changes in climate have not yet been modelled with confidence. However, regional differences from the global averages show that warming may be greater in high latitudes during late autumn and winter than in the tropics; annual mean runoff may increase in high latitudes; and summer dryness may become more frequent over the continents at middle latitude in the Northern Hemisphere. In tropical regions, temperature increases are expected to be smaller than the average global rise, but the effects on ecosystems and humans could have far reaching consequences. Potential evapotranspiration probably will increase throughout the tropics, whereas in moist, tropical regions, convective rainfall could increase.

- Based on the observed changes since the beginning of this century, it is estimated that global warming of 1.5°C to 4.5°C would lead to a sea-level rise of 20 to 140 cm. A sea-level rise in the upper portion of this range would have major direct effects on coastal areas and estuaries. A significant melting of the West Antarctic ice sheet leading to a much larger rise in sea level, although possible at some future date, is not expected during the next century.

- Based on analyses of observational data, the estimated increase in global mean temperature of between 0.3°C and 0.7°C during the last 100 years is consistent with the projected temperature increase attributable to the observed increase in CO2 and other greenhouse gases, although it cannot be ascribed in a scientifically vigorous manner to these factors alone.

- Based on evidence of the effects of past climate changes, there is little doubt that a future change in climate of the magnitude obtained from climate models could have a profound effect on global ecosystems, agriculture, water resources, and sea ice.

As noted in the WMO/UNEP/ICSU report the projected changes in climate will have important impacts on all aspects of society. Agriculture, forests, human health, water resources, energy planning, and recreation are among the sectors likely to be affected. Moreover, all these sectors are likely to be affected simultaneously throughout the world, but to different extents. Today we know a great deal from palioclimatic records about how past shifts in climate affected the growth and development of forest systems, the location of lakes, and development of agriculture. But the changes that occurred to these systems in the past took 18 to 20 thousand years to unfold as the earth warmed approximately 4°C-5°C. During that time, forest composition shifted, some lake systems were lost and new ones were formed. Most importantly, the changes took place during a period when the earth's population was small and civilizations were in formative stages.

Today modern society is much more complex, but still vulnerable to climatic changes. Our industrial society relies on a sustained climate to replenish natural resources as a source of raw materials, for transport of goods and for the food we eat. We assume that the climate that supports our society, while variable and difficult to predict over short periods, will not shift appreciably. Indeed, most decisions made by farmers, forest managers, state and local water management officials, utility executives and government policy makers assume that climate will be constant. However, if current predictions from global climate models prove to be correct, some increase in global temperatures may be inevitable simply because of the presence of trace gases which have already been emitted to the atmosphere.

Our current understanding of the effects of global climate change on the environment is incomplete. Moreover, several features of the greenhouse phenomen make it unique, different from other environmental issues and difficult to analyze. Among these features are the following:

- The effects will not take place immediately, but over several decades.
- The effects will be virtually irreversible over several centuries.
- All nations of the world still experience the effects at the same time.
- There is no historical analog for the amount of global warming likely to occur in a relatively short period of the next 50 to 100 years.

Scientists have only begun to analyze the potential impacts from global warming and changes in other climate variables. Insights are available from historical data and from the application of predictive models. In most cases however, our understanding of the consequences both beneficial and detrimental, is in a formative stage. Historical analogs provide qualitative information about likely effects, but they cannot predict the future because the anticipated rate and increase in temperature are beyond the range of previous warm periods. Predictive models of both the climate system and potential effects often do not include complete parameterizations of important system variables. Thus, more advanced global climate models capable of providing regional predictions are needed, and more comprehensive and sophisticated analyses of environmental effects are necessary to understand fully the implications throughout the world.

Recognizing these limitations, the following section summarizes what is known about climate impacts on the environment with emphasis on forests, agriculture, water resources and human health. Perhaps of equal importance are potential impacts which have not been reported or analyzed, including potential impact on ports, electricity, demand and supply, population shifts, work place absenteeism rates, hurricane frequency, air pollution emissions, wildlife management and national security. These potential impact areas and many others represent the challenges to be investigated as the science supporting predictions of global climate change improves.

FINDINGS

The following findings are summarized from Appendix B, which provides a comprehensive review of potential impacts of global climate change.

1. CLIMATE CHANGE HAS HAD A SIGNIFICANT IMPACT ON FORESTS IN THE PAST. IF CURRENT PREDICTIONS PROVE ACCURATE, THERE IS A POTENTIAL FOR DRAMATIC SHIFTS IN FORESTS AND VEGETATION OVER THE NEXT 100 YEARS.

   1a. Climate models predict that a global warming of approximately 1.5°C to 4.5°C will be induced by a doubling of atmospheric CO2 and other Trace gases during the next 50 to 100 years. The period 18,000 to 0 years B.P. is the only general analog for a global climate change of this magnitude. The geological record from this glacial to inter- glacial interval provides a basis for qualitatively understanding how vegetation may change in response to large climatic change.

   1b. The paleovegetational record shows that climatic change as large as that expected to occur in response to CO2-doubling is likely to induce significant changes in the composition and patterns of the world's biomes. Changes of 2°C to 4°C have been significant enough to alter the composition of biomes, and to cause new biomes to appear and others to disappear. At 18,000 B.P., the vegetation in Eastern North America was quite distinct from that of the present day. The cold/dry climate of that time seems to have precluded the widespread growth of birch, hemlock, beech, alder, hornbeam, ash, elm and chestnut, all of which are fairly abundant in present-day deciduous forest. Southern pines were limited to grow with oak and hickory in Florida.

   1c. Available paleoecological and paleoclimatological records do not provide an analog for the high rate of climate change and unprecedented global warming predicted to occur over the next century. Previous changes in vegetation have been associated with a climates that were nearly 5°C to 7°C cooler and which took thousands of years to evolve rather than the predicted decadal changes. Insufficient temporal resolution e.g., via radiocarbon dates limits our ability to analyze the decadal-scale rates of change that occurred prior to the present millennium.

   1d. Limited experiments conducted with dynamic vegetation models for North America suggest that decreases in net biomass may occur and that significant changes in species composition are likely. Experiments with one model suggest that Eastern North American biomass may be reduced by 11 megagrams per hectare (10% of live biomass) given the equivalent of a doubled CO2 environment. Plant taxa will respond individualistically rather than as whole communities to regional changes in climate variables. At this time such analyses must be treated as only suggestive of the kinds of change that could occur. Many critical processes are simplified or omitted and the actual situation could be worse or better.

1e. Future forest management decisions in major timber-growing regions are likely to be affected by changes in natural growing conditions. For example, one study suggests that loblolly pine populations are likely to move north and northeast into Pennsylvania and New Jersey while its range shrinks in the west. The total geographic range of the species may increase, but a net loss in productivity may result because of shifts to less accessible and productive sites. While the extent of such changes is unclear, adjustments will be needed in forest technology, resource allocation, planning, tree breeding programs, and decision-making to maintain and increase productivity.

1f. Dynamic vegetation models based on theoretical descriptions of all factors that could influence plant growth must be improved and/or developed for all major kinds of vegetation. In order to make more accurate future predictions, these models must be validated using the geological record and empirical ecological response surfaces. In particular, the geological record can be used to test the ability of vegetation models to simulate vegetation that grew under climate conditions unlike any of the modern day conditions.

1g. Dynamic vegetation models should incorporate direct effects of atmospheric CO2 increases on plant growth and other air pollution effects. Improved estimates of future regional climates are also required in order to make accurate predictions of future vegetation changes.

2. AGRICULTURE IS VERY DEPENDENT ON CLIMATE. LIMITED ASSESSMENTS SUGGEST THAT IMPORTANT CHANGES IN AGRICULTURE AND FARM PRODUCTIVITY ARE LIKELY THROUGHOUT THE WORLD, IF CLIMATE CHANGE OCCURS AS PREDICTED.

2a. Climate has had a significant impact on farm productivity and geographical distribution of crops. Examples include the 1983 drought which contributed to a nearly 30% reduction in corn yields in the U.S., the persistent Great Plains drought between 1932-1937 which contributed to nearly 200,000 farm bankruptcies, and the climate shift of the Little Ice Age (1500-1800) which led to the abandonment of agricultural settlements in Scotland and Norway.

2b. World agriculture is likely to undergo significant shifts if trace gas-induced climate warming in the range of 1.5°C to 4.5°C occurs over the next 50 to 100 years. Climate effects on agriculture will extend from local to regional and international levels. However, modern agriculture is very dynamic and is constantly responding to changes in production, marketing and government programs.

2c. The main effects likely to occur at the field level will be physical impacts of changes in thermal regimes, water conditions, and pest infestations. High temperatures have caused direct damage to crops such as wheat and corn; moisture stress, often associated with elevated temperatures, is harmful to corn, soybean and wheat during flowering and grain fill; and increased pests are associated with higher, more favorable temperatures.

2d. Even relatively small increases in the mean temperature can increase the probability of harmful effects in some regions. Analysis of historical data has shown that an increase of 1.7°C (3°F) in mean temperature changes the likelihood of a five consecutive daily maximum temperature event of at least 35°C (95°F) by about a factor of three for a city like Des Moines. In regions where crops are grown close to their maximum tolerance limits, changes extreme temperature events may have significant harmful effects on crop growth and yield.

2e. Limited experiments using climate scenarios and agricultural productivity models have demonstrated the sensitivity of agricultural systems to climate change. Future farm yields are likely to be effected by climate because of changes in the length of the growing season, heating units, extreme winter temperatures, precipitation, and evaporative demand. In addition, field evaluations show that total productivity is a function of the drought tolerance of the land and the moisture reserve, the availability of land, the ability of farmers to shift to different crops, and other factors.

2f. The transition costs associated with adjusting to global climatic change are not easily calculated, but are likely to be very large. Accommodating to climate change may require shifting to new lands and crops, creating support services and industries, improving and relocating irrigation systems, developing new soil management and pest control programs, and breeding and introducing new heat or drought tolerant species. The consequences of these decisions on the total quantity, quality, and cost of food are difficult to predict.

2g. Current projections of the effects of climate change on agriculture are limited because of uncertainties in predicting local temperature and precipitation patterns using global climate models, and because of the need for improved research studies using controlled atmospheres, statistical regression models, dynamic crop models and integrated modeling approaches.

3. WATER RESOURCE SYSTEMS HAVE UNDERGONE IMPORTANT CHANGES AS THE EARTH'S CLIMATE HAS SHIFTED IN THE PAST. CURRENT ANALYSES SUGGEST AN INTENSIFIED HYDROLOGIC CYCLE, IF CLIMATE CHANGE OCCURS AS PREDICTED.

3a. There is evidence that climate change since the last ice age (18,000 years B.P.) has significantly altered the location of lakes although the extent of present day lakes is broadly comparable with 18,000 years B.P. For example, there is evidence indicating the existence of many tropical lakes and swamps in the Sahara, Arabian, and Thor Deserts around 9,000 to 8,000 years B.P.

3b. The inextricable linkages between the water cycle and climate ensure that potential future climate change will significantly alter hydrological processes throughout the world. All natural hydrological processes -- precipitation, infiltration, storage and movement of soil moisture, surface and subsurface runoff, recharge of groundwater, and evapotranspiration -- will be affected if climate changes.

3c. As a result of changes in key hydrological variables such as precipitation, evaporation, soil moisture, and runoff, climate change is expected to have significant effects on water availability. Early hydrological impact studies provide evidence that relatively small changes in precipitation and evaporation patterns might result in significant, perhaps critical, changes in water availability. For many aspects of water resources, including haman consumption, agricultural water supply, flooding and drought management, groundwater use and recharge, and reservoir design and operation, these hydrologic changes will have serious implications.

3d. Despite significant differences among climate-change scenarios, a consistent finding among hydrological impact studies is the prediction of a reduction in summer soil moisture and changes in the timing and magnitude of runoff. Winter runoff is expected to increase and summer runoff will decrease. These results appear to be robust across a range of climate change scenarios.

3e. Future directions for research and analyses suggest that improved estimates of climate variables are needed from large-scale climate models; innovative techniques are needed for regional assessments; increased numbers of assessments are necessary to broaden our knowledge of effects on different users; and increased analyses of the impacts of changes in water resources on the economy and society are necessary.

4. WEATHER IS CLOSELY ASSOCIATED WITH MORBIDITY AND MORTALITY RATES IN OUR SOCIETY.

4a. Weather has a profound effect on human health and well being. It has been demonstrated that weather impacts are associated with changes in birth rates, outbreaks of pneumonia, influenza, and bronchitis, and related to other morbidity effects and linked to pollen concentrations and high pollution levels.

4b. Large increases in mortality have occurred during previous heat and cold waves. It is estimated that 1,327 fatalities occurred in the United States as a result of the 1980 heat wave and Missouri alone accounted for over 25% of that total.

4c. Hot weather extremes appear to have a more substantial impact on mortality than cold wave episodes. Most research indicates that mortality during extreme heat events varies with age, sex, and race. Factors associated with increased risk include alcoholism, living on higher floors of buildings, and the use of tranquilizers. Factors associated with decreased risk include the use of air conditioning, frequent exercising, consumption of fluids, and living in shaded residences. Acclimatization may moderate the impact of successive heat waves over the short-term.

4d. Threshold temperatures for cities have been determined which represent maximum and minimum temperatures associated with increases in total mortality. These threshold temperatures vary regionally, i.e., the threshold temperature for winter mortality in mild southern cities such as Atlanta is 0°C and for more northerly cities such as Philadelphia, threshold temperature is -5°C.

4e. Humidity has an important impact on mortality, since it contributes to the body's ability to cool itself by evaporation of perspiration. It also has an important influence on morbidity in the winter because cold, dry air leads to excessive dehydration of nasal passages and the upper respiratory tract and increased chance of microbial and viral infection.

4f. Precipitation in the form of rainfall and snow is also associated with changes in mortality. In New York City, upward trends in mortality were noted the day after snowfalls which had accumulated 2 in. or more. In Detroit where snowfall is more frequent, mortality increases were noted after the snowfall accumulation exceeded 6 in.

4g. If future global warming induced by increased concentrations of trace gases does occur, it has the potential to significantly affect human mortality. In one study, total summertime mortality in New York City was estimated to increase by over 3,200 deaths per year for a 7°F trace gas-induced warming without acclimatization. If New Yorkers fully acclimatize, the number of additional deaths is estimated to be no different than today. It is hypothesized that, if climate warming occurs, some additional deaths is likely to occur because economic conditions and the basic infrastructure of the city will prohibit full acclimatization even if behavior changes.

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CHAPTER 17

MODELS FOR INTEGRATING THE ANALYSES OF HEALTH AND ENVIRONMENTAL RISKS ASSOCIATED WITH OZONE MODIFICATION

SUMMARY

Analyses of the potential risks due to emissions of ozone-modifying substances and greenhouse gases must assess a variety of effects on human health and the environment. To assess risks numerous factors must be examined together, including: economic and population growth; chemical use and emissions; ozone modification; changes in ultraviolet (UV) radiation flux; climate change; and effects on human health and the environment.

To evaluate risks the Integrating Analysis Model has been developed to provide a framework for systematically evaluating the risks and costs associated with alternative assumptions about the ozone modification issue and policies for limiting the emissions of ozone-modifying substances. This chapter describes the manner in which the Integrating Analysis Model (the model) draws from the analyses described in earlier chapters.

The model begins with assumptions about the potential future use of ozone-modifying substances and concentrations of other trace gases, including chlorofluorocarbons, methyl chloroform, carbon tetrachloride, Halons (bromine-containing compounds), carbon dioxide, methane, and nitrous oxide. This potential future use and emissions may be modified by control strategies. The resulting global emissions of the substances are computed, followed by the atmospheric science module, which assesses the impacts of these emissions on ozone. The risks of ozone modification for human health and the environment are evaluated using specified dose-response relationships where available for each of the areas of interest.

Using this overall framework, the model can reflect a wide range of assumptions and the joint implications of all the assumptions can be identified. Of note is that the model is not a substitute for choosing the assumptions, relationships, and data to describe the system being modeled. It merely reflects these assumptions and data in a consistent framework.[1]

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[1] When used for performing policy analysis, the model does not make decisions, for example, about which, if any, control strategies are preferred or about which, if any, ozone depletion estimates are correct. Decisions about the need to protect stratospheric ozone or the best way to do so must be based not only on model results, but also on considerations and judgments either not reflected in the model or reflected implicitly in the assumptions provided as input to the model.

INTRODUCTION

Analyses of the potential risks due to emissions of ozone-modifying substances and greenhouse gases must assess a variety of effects on human health and the environment. As described earlier in this document, numerous factors influence ozone modification and its effects. To assess risks, the following must be characterized, and their joint implications (including their uncertainties) examined together: economic and population growth; chemical use and emissions; ozone modification; changes in ultraviolet (UV) radiation flux; climate change; and effects on human health and the environment.

Analyzing and combining these factors is a complex task. To make this task tractable, two models have been developed:

1. Integrating Analysis Model. The primary role of the Integrating Analysis Model is to provide a framework for systematically evaluating the risks and costs associated with alternative assumptions about the ozone modification issue and policies for limiting the emissions of ozone-modifying substances.

2. Transient Temperature and Sea Level Rise Model. The primary role of this model is to assess the implications of emissions of greenhouse gases in terms of increases in global average temperature and sea level over time.

Together, these two models provide a comprehensive framework for evaluating the potential risks of emissions of potential ozone-modifying substances and greenhouse gases. This chapter describes the manner in which the Integrating Analysis Model (the model) draws from the analyses described in earlier chapters.[2] The Transient Temperature and Sea Level Rise Model is described in Appendix A of Chapter 6.

This chapter first describes the framework that the model provides for the analysis. Then, an overview of the model's analysis procedure is presented. The third section of this chapter presents the major limitations of the model. The chapter concludes with a series of appendices that describe the model's design and operation in detail.

THE MODEL AS A FRAMEWORK

The objective of the model is to provide a framework within which the implications of alternative assumptions and policies can be identified. The framework reflects the order and structure of the analysis, and the user may define the assumptions and data to be used in the model for any given run. Exhibit 17-1 displays the major parts or "modules" of the model. The model

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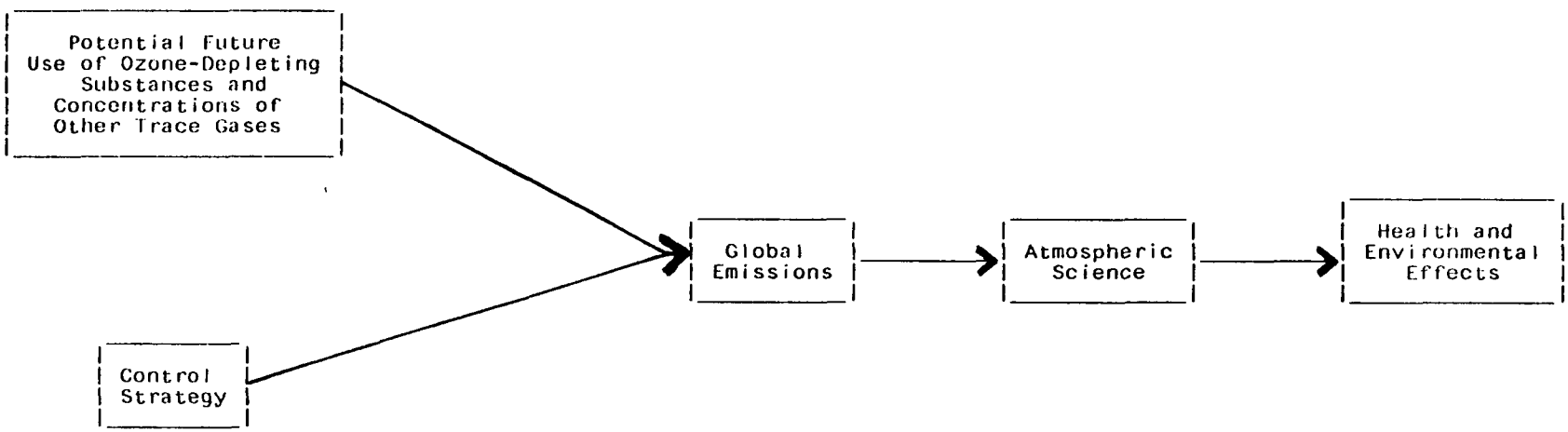
[2] The Integrating Analysis Model was originally described in Gibbs and Hoffman (1986).

EXHIBIT 17-1

MODULAR STRUCTURE



begins with assumptions about the potential future use of ozone-modifying substances and concentrations of other trace gases, including chlorofluorocarbons, methyl chloroform, carbon tetrachloride, Halons (bromine-containing compounds), carbon dioxide, methane, and nitrous oxide. This potential future use and emissions may be modified by control strategies. The resulting global emissions of the substances are computed, followed by the atmospheric science module, which assesses the impacts of these emissions on ozone.[3] The risks of ozone modification for human health and the environment are evaluated using specified dose-response relationships where available for each of the areas of interest.

Using this overall framework, the model can reflect a wide range of assumptions within each of the modules. When the model is run, the joint implications of all the assumptions are identified. Of note is that the model is not a substitute for choosing the assumptions, relationships, and data to describe the system being modeled. It merely reflects these assumptions and data in a consistent framework. In its simplest terms, the model is a calculator that is needed to assist in computing multiple estimates of possible values. Decisions about best assumptions or the need to protect stratospheric ozone or the best way to do so may use the model and its results, but must also be based on considerations and judgments either not reflected in the model or reflect implicitly in the assumptions provided as input to the model.

ANALYSIS PROCEDURE

A model "run" is comprised of:

- inputs provided by the user that define the data and assumptions to use in the analysis;
- algorithms that are carried out according to the user's inputs and instructions contained within the model; and
- outputs prepared by the model that describe the results of the algorithms that are carried out.

The model is "deterministic," meaning that a unique output or result is produced by a specified input; there are no random (i.e., stochastic) elements within the model.

To initiate a model run, the user must specify inputs for each module he wishes to use. (The user may choose to run only a portion of the model.)

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[3] These emissions rates are also used in the Transient Temperature and Sea Level Rise Model to evaluate climate change effects.

Exhibit 17-2 provides an overview of the input choices for each of the following modules:

- Potential Use. The user may choose one of the five scenarios of future use that have been incorporated into the model. The compounds included are: chlorofluorocarbons (CFC-11; CFC-12; CFC-22; CFC-113); carbon-tetrachloride (CCL4); methyl chloroform (CH3CCL3); Halon-1211; and Halon-1301. Each compound can be analyzed for up to six end uses, in 10 regions of the world. The five scenarios reflect a wide range of potential future use, and are based on historical trends and projections.[4] Alternatively, the user may specify his own scenario of future use.

- Control Strategy. The user may specify a variety of controls on future use, such as kilogram limits, use limits, technology requirements, or fees. The potential costs and benefits of controls are not evaluated in this document because the objective of this risk assessment is to evalute risks in the absence of regulation.

- Global Emissions. Emissions of the chlorofluorocarbons, CCL4, CH3CCL3, Halon-1211 and Halon-1301 are computed from the production and use estimates based on the release rate algorithm data described in Chapter 3. The release rates reflect the fact that these chemicals are stored in certain products for various periods of time. The effects of control strategies are reflected in the release estimates, and the user may specify alternative release rates as desired. Scenarios of emissions or atmospheric concentrations are also specified for carbon dioxide (CO2), methane (CH4) and nitrous oxide (N2O).

- Atmospheric Science. A parameterized numerical fit to a one-dimensional (1-D) atmospheric model is used to compute global ozone depletion as a function of global emissions of chlorofluorocarbons, CC14, CH3CC13, and Halons, and concentrations of CO2, N2O and CH4.[5] The uncertainty in the estimates of global depletion may be modeled.[6] Alternatively, the user may bypass this

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[4] These five scenarios are described in Chapter 3.

[5] See Connell (1986) for a description of this method.

[6] The range of uncertainty identified in Stolarski and Douglass (1985) is currently incorporated into the model. The user may provide alternative uncertainty estimates.

EXHIBIT 17-2

MAJOR MODEL INPUT CHOICES

MODULE	CHOICES	COMMENTS	SOURCES
Potential Future Use of Ozone-Modifying Substances	Future Use: -- Scenarios based on studies of historical trends and projections of economic growth and population growth -- Any user-supplied estimates of future use can be incorporated	Future Use: -- Scenarios reflect a wide range of potential future use.	Based on: UNEP (1986).
Control Strategies	Strategies can be defined in terms of limits to production in various ways, including: -- limits in kilograms; -- limits in kilograms per capita; -- bans of specified uses; -- requirements to implement specified technologies; and -- fees or taxes.	Data on control options are based on U.S. data. Information for other parts of the world may be entered by the user, or the U.S. data can be applied to the rest of the world as a default is no data are available.	Estimates of the costs and effectiveness of controls are based on data presented in Camm (1986). Analyses are underway to update these estimates.
Global Emissions	Several estimates of the rate of release after use have been developed based on analyses of historical data. Emissions reflect the effectiveness of the control strategy in limiting emissions. The user may choose several scenarios of effectiveness, which reflects the level of reductions expected to occur as the result of the control strategy identified.	Data on the effectiveness of control options are based on U.S. data. Information on other parts of the world may be entered by the user.	The rates of emissions following use are based on data presented in Quinn (1986).

EXHIBIT 17-2 (continued)

MAJOR MODEL INPUT CHOICES

MODULE	CHOICES	COMMENTS	SOURCES
Atmospheric Science	Ozone Modification: -- Parameterized relationship between global emissions and global ozone depletion. -- The user may specify his own estimate of ozone depletion for use in the model. If this choice is made, the global emissions estimate is not used.	Ozone Modification: -- Global estimates based on analysis of a one-dimensional atmospehric model. -- Estimates that vary by latitude are based on an analysis of a two-dimensional atmospheric model.	Based on: Connell (1986) and Isaksen (1980).
Health and Environmental Effects	Ranges of relationships with UV radiation are developed for each effect. The user may specify for each effect low, medium, or high estimates.	Ranges based on published estimates.	Based on analyses by: Scotto, Fears, and Fraumeni (1981), Scotto and Fears (in press), Hiller, Sperduto, and Ederer (1983), Horst (1986), Andrady (1986), and Pitcher (in press).

algorithm and use a different relationship or independent estimates of global ozone depletion. Using the parameterization estimate of global depletion, estimates of ozone modification by latitude are then made based on an analysis of the results of a two-dimensional model (Isaksen 1986). It should be noted that this two step method of producing latitudinal depletion estimates results in lower estimates of depletion than Isaksen (1986) because the global estimates based on the parameterization are lower than the global estimates produced by Isaksen (1986).

- Health and Environmental Effects. The association between ozone depletion, ultraviolet (UV) radiation, and effects may be specified by the user. Ranges of dose-response relationships may be identified for melanoma and non-melanoma skin cancers, and cataracts. Population projections (by race, age, and sex) are incorporated into the model for purposes of computing future incidence of human health effects. Alternative population estimates may be identified by the model user. The effects on PVC polymers are also reported. Estimates of effects are made for the U.S. only.

Based on these inputs, the model performs calculations on a year-by-year basis. For example, at the beginning of year t, the model first computes the potential use of each of the ozone-modifying substances, in each of its applications, in each portion of the world being analyzed. This estimate of use reflects the user's choice of scenario (for the first module) as well as the effects of any controls initiated according to the user's instructions prior to and including year t.[7] Based on this estimate of use in year t, and estimates for all years prior to year t, the global emissions in year t are computed for each potential ozone-depleting substance.

The atmospheric science module computes an estimate of global ozone depletion for year t based on the emissions estimates for year t, atmospheric conditions due to emissions prior to year t, and user-specified assumptions regarding the concentrations of carbon dioxide, methane, and nitrous oxide.[8] Global ozone depletion is translated into estimates that vary by latitude. The latitudinal estimates of ozone depletion are then translated into associated changes in UV flux, using three action spectra for weighting

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[7] The effects of controls include the direct reduction in use due to the application of control technologies, as well as the potential reduction in use associated with the influence that increased production costs and prices may have on demand.

[8] Appropriate ranges of assumptions for these gases are described in Chapter 4.

the different UV wavelengths: DNA; Robertson-Berger Meter (RB-Meter) and erythema. The changes in UV flux in year t (and prior years) are used in dose-response relationships to estimate increased risks of melanoma and non-melanoma skin cancers and cataracts. Other impacts, such as polymer degradation, are also computed.

After the computations for year t are performed, the model repeats the computations for year t+1. This annual series of computations is consequently repeated for each year of the model run. After all the years have been analyzed, the following summary outputs are produced:

- future production of ozone-depleting substances;
- future emissions of ozone-depleting substances;
- future global ozone depletion;
- future ozone depletion by latitude;
- future increases in UV flux; and
- estimates of effects, including:
  - -- melanoma skin cancers (incidence and mortality);
  - -- non-melanoma skin cancers (incidence and mortality);
  - -- cataracts (incidence); and
  - -- costs of damages to polymers (PVC used outdoors).

The effects estimates are currently based primarily on data that describe the U.S. Consequently, only U.S. effects estimates are currently produced.

MODEL LIMITATIONS

In using any model, it is important to understand its limitations. The model is being developed in a neutral fashion. Its flexible design allows users to evaluate wide ranges of inputs and assumptions. The model cannot incorporate, however, numerous factors that are as yet undefined or poorly understood. Consequently, the model excludes a variety of potential factors and analyses that may be important for making decisions regarding stratospheric ozone protection.

The most important exclusions from the model are a variety of effects of ozone modification. Many effects that increased UV radiation will have on human health and the environment are not well quantified. For example, only limited data are available that describe the effects of UV radiation on plants and aquatic organisms. These data cannot be extrapolated to all crops and ecosystems to evaluate the net effects of ozone modification and global warming. The potential for effects is substantial, but current data do not allow quantitative estimates at this time. Similarly, the effects of UV radiation on ground oxidants are not well quantified at this time (see Whitten (1986) for case studies of effects on oxidants).

Similarly, too little knowledge currently exists to allow immune suppression effects of increased UV radiation to be quantified. These effects may be particularly important because they may affect all individuals in the world in important ways that are not currently anticipated.

Exhibit 17-3 summarizes the excluded effects. The omission of these known effects, which cannot be quantified, is a clear bias reflected in the estimates of the risks associated with ozone depletion. In addition, there may be other links among UV radiation, global warming, and human health and the environment that have not yet been identified. The potential bias of the results due to lack of information on these effects is unknown.

The model is limited in its ability to address issues related to risk management and policy analysis.[9] This document does not address these issues because it is focused on risk assessment.

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[9]The model currently does not address a variety of risk management issues, including:

- the feasibility of alternative control strategies;
- potential trade of ozone-modifying substances among regions of the world; and
- the number of countries likely to participate in international agreements to limit the use and emissions of ozone-depleting substances.

Decision makers must use their own judgments regarding these factors. The model may be used to test the implications of these judgments (e.g., the implications of only half the countries participating in a control strategy may be identified by specifying the expected participation as a model input). However, the model is not meant to be used in place of these types of judgments, or any others, that are required of decision makers.

EXHIBIT 17-3

Effects Not Quantified

Effect	Comments
Impact on plants	Preliminary experiment data indicate risks of decreased yields. See Chapter 11.
Impact on aquatic organisms	Laboratory data indicate increase UV radiation may adversely affect survivability and breeding. See Chapter 12.
Impact on urban ground-based ozone formation (smog)	Case studies of three cities indicate potential increases in smog formation, and formation earlier in the day. See Chapter 14.
Increased incidence of cutaneous diseases as a result of immune suppression	See Chapter 9.
Increased incidence of serious cases of non-melanoma skin cancer	Relationship with UV-B radiation not quantified.
Impacts of climate change on human health, water resources, forestry, and agriculture	See Chapter 16.
Impacts of sea level rise on wetlands	See Chapter 15.
Impacts on polymers other than PVC	See Chapter 13.

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APPENDIX A

MODEL DESIGN AND MODEL FLOW

INTRODUCTION

This appendix describes the design and flow of the CFC Policy Model (the model). The model is deterministic -- it computes a series of outputs from data supplied by the model user and from data that reside in files associated with the model. A model "run" is comprised of (1) a set of instructions (i.e., inputs) supplied by the model user; (2) a set of computations performed within the model; and (3) a set of output tables that present the results of the computations performed by the model. The model is designed with a modular structure, with each "module" performing a designated function. The modules are independent entities that communicate with each other.

The modular structure facilitates the maintenance and development of the model by:

- allowing desired modifications to the model to be localized to individual modules;
- allowing communication among modules to be identified easily (communication among modules is particularly important for modeling feedbacks from one portion of the model to another portion); and
- minimizing memory requirements and execution time.

The remainder of this appendix is divided into the following sections:

- Level of Aggregation;
- Model Flow;
- Specifying a Model Run; and
- Limitations.

LEVEL OF AGGREGATION

The smallest unit of analysis in the model includes compound, region, end use, and time, as follows:

- Compound -- Up to ten compounds may be analyzed separately:
  - -- CFC-11;
  - -- CFC-12;
  - -- CFC-22;
  - -- CFC-113;
  - -- CFC-114;
  - -- CFC-115;
  - -- Carbon tetrachloride (CCl_4);

-- Methyl chloroform (CH_3CCl_3);
-- Halon-1211; and
-- Halon-1301.

Data are currently not analyzed for CFC-114 and CFC-115.

- Region -- The world is divided into 10 separate regions (the regions can be analyzed individually or in groups):

  -- USA;
  -- Canada;
  -- Western Europe;
  -- Japan, Australia, and New Zealand;
  -- USSR and East Bloc;
  -- Central Planned Asia;
  -- Middle East;
  -- Africa;
  -- Latin America; and
  -- South and East Asia.

- End Use -- Each compound may be identified with up to six applications or "end uses." Each end use can be analyzed separately. End uses currently incorporated into the model include:

  -- aerosol propellant;
  -- flexible foam;
  -- rigid polyurethane foam;
  -- rigid nonurethane foam;
  -- refrigeration;
  -- solvent;
  -- fire extinguisher; and
  -- miscellaneous.

- Time -- a year is the smallest unit of time in this model. The current range is 1931-2165.

Analysis is performed at these levels of information. Major revisions to the model are required to modify this level of disaggregation.

MODEL FLOW

The model is divided into two main parts: (1) the Analysis Program reads input data, performs computations, and creates output files; (2) the Report Writer Program reads output files from the Analysis Program, and creates formatted reports as specified by the user. This section discusses each part of the model in turn.

Analysis Program

The Analysis Program begins by reading an input file that describes the configuration of the model run desired by the user, including:

- which years will be analyzed;
- the portions of the model (i.e., modules) that will be used in the analysis;
- the data files that will be used as inputs; and
- the years for which output values should be computed.

Based on these user-supplied data, a series of variables are initialized and the model run specified by the user then proceeds.

The flow of a model run can be described in terms of the following five primary parts or "modules" of the Analysis Program:

1. Production Scenarios Module;
2. Policy Alternatives Module;
3. Emissions Module;
4. Atmospheric Science Module; and
5. Effects Module.

As shown in Exhibit A-1, these five modules are all executed within a "year loop," i.e., all the information from all the modules is computed for one year, then the model moves on to the next year. This structure has several benefits, including:

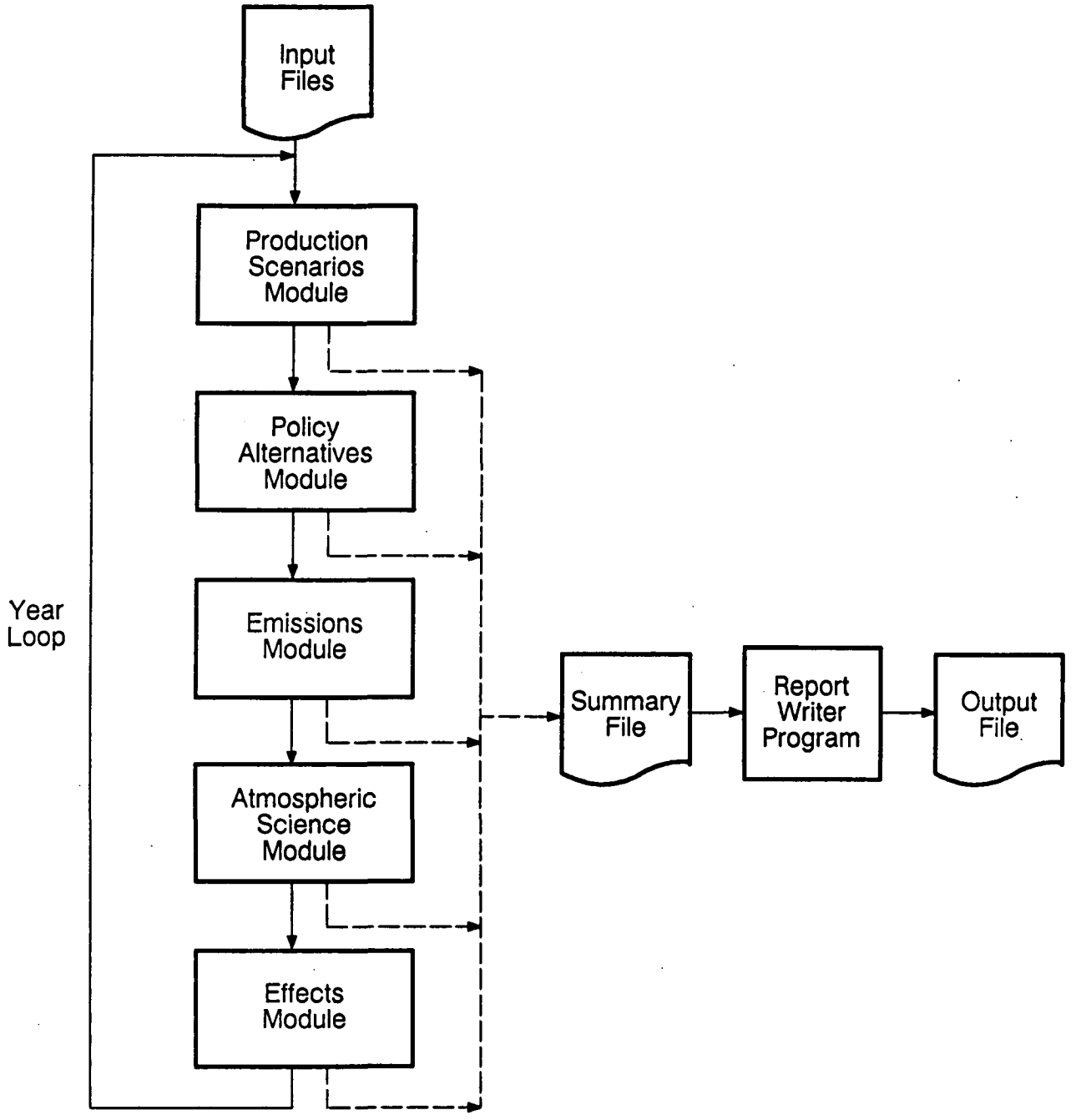
- the results of year "t" can be used to indicate what would happen in year "t+1" (in other words, feedbacks between modules and across years can be modeled);
- the information required to be stored is limited to the current year being analyzed.

Appendices B through F describe each of the modules in turn. Briefly, each module performs the following computations:

- Appendix B: Production Scenarios Module -- This module computes a scenario of production over time for each compound. The scenario for each compound is disaggregated by region and end use.
- Appendix C: Policy Alternatives Module -- This module identifies the implications of alternative control policies specified by the user. The module's input includes policy specifications (from the user), production scenarios (from the previous module), and

EXHIBIT A-1

Flow of Analysis Program



candidate technical options for controlling the use of potential ozone-depleting substances (identified in an input file). The final results of this module are (1) the incurred costs of control attributable to the policy; and (2) the reductions in production realized as a consequence of control.

- Appendix D: Emissions Module -- This module calculates global emissions of each compound by applying a release algorithm to the production scenarios (which have been modified by the Policy Alternatives Module). The release algorithm specifies the time it takes (after production) for the compound to be released for each end use of each compound. The algorithm reflects the fact that the chemicals are stored in products for many years.

- Appendix E: Atmospheric Science Module -- This module uses the global emissions estimates from the Emissions Module and estimates of atmospheric concentrations of carbon dioxide (CO2), methane (CH4) and nitrous oxide (N2O) to calculate global and latitudinal ozone depletion. Ozone depletion is calculated using parameterized equations that represent one-dimensional and two-dimensional models of the atmosphere. Uncertainty is also modeled based on investigations of uncertainty propagation through an atmospheric model.

- Appendix F: Effects Module -- This module projects the effects that ozone depletion may have on human health and the environment. Human health effects are focused on melanoma and non-melanoma skin cancers and cataracts. Other effects include degradation of materials.

The results of each module are written to a "Summary File." This Summary File is used by the Report Writer Program to create detailed tables as specified by the user.

Report Writer Program

The Report Writer Program reads in the data stored in the Summary File, and creates reports for the model run as specified by the user. The reports for a given model run may show results disaggregated by region, compound, end use, and time. Results across runs may also be displayed and compared. Of note is that once a Summary File is created by the Analysis Program, the Report Writer Program can be run several times to generate reports at various levels of aggregation without re-running the Analysis Program.

SPECIFYING A MODEL RUN

To specify a model run the user fills out a set of data files that describe the assumptions and data to be used. The names of these data files, and their purposes, are listed in Exhibit A-2. Each is described in turn.

- The Run File identifies the overall model flow, including which modules are to be executed. This file includes:

  -- the range of years for the model run;

  -- the names of modules to be executed;

  -- the names of the data files to be used;

  -- the years for which summary results will be retained; and

  -- the discount rates for cost/benefit analysis.

- The Production Scenarios File specifies the scenario of future production of potential ozone-depleting compounds to use in the analysis. The user may choose one of a set of defined scenarios, or may create his own. The user also chooses (or creates) a scenario of population and economic growth.

- The Policy Alternatives File defines the control strategy (if any) that the user is evaluating. In addition to defining the level of controls or the policy objective, the user must choose among alternative scenarios that define the technical options available for reducing the use of potential ozone-depleting compounds.

- The Atmospheric Science File allows the user to choose among alternative methods for computing ozone-depletion. The user may specify a range of uncertainty surrounding the ozone-depletion estimate. The assumptions regarding the concentrations of other trace gases (carbon dioxide, methane, and nitrous oxide) are also specified in this file.

- The Effects File identifies the assumptions used to (1) estimate effects (in their natural units); and (2) evaluate the effects in monetary terms. The user chooses (or supplies) dose-response coefficients for each effect. The value (in dollars) of each effect may also be specified.

EXHIBIT A-2

Files Required to Specify a Run

File Name	Purpose
Run File	To identify modules included in the run and data files to be used, specify years for which outputs are desired, and define the discount rates used to calculate costs and benefits.
Production Scenarios File	To choose a scenario of future production for each compound and of future population and economic growth.
Policy Alternatives File	To define the control strategy to be evaluated and the scenario of available technical controls.
Atmospheric Science File	To choose a method for estimating ozone depletion and specify a range of uncertainty surrounding the method. Also to specify scenarios of concentrations of other trace gases.
Effects File	To specify the dose-response coefficients for each human health effect, the action spectrum to be used for each human health effect, and the economic value to be placed on each human health effect. Also, to specify the values used to estimate polymer degradation.
Report Writer File	To identify runs for which reports are to be written and the reports desired.

- The Report Writer File identifies the runs for which reports are to be written and the reports desired.

Of note is that once these files have been specified, and the model is run, the entire model need not be re-run to perform additional analyses. For example, to analyze an alternative set of effects assumptions, only the Effects File needs to be filled out, and only the Effects Module needs to be executed. The results from the previous modules can be stored for future use. This flexibility in the modular design of the model helps to facilitate numerous comparison runs and reduces the time and expense of the analysis.

LIMITATIONS

Although the model was implemented in a modular fashion to allow for flexibility in development and use, it has several important limitations.

- Size. The size of the model was kept manageable by limiting the units of analysis to 10 regions, 10 compounds, and six end uses per compound. More regions (e.g., for analyses of individual countries) cannot be accomodated easily. The number of compounds and end uses per compound are also fixed.

- Feedback between compounds. Each compound is analyzed independently. The control of one compound (e.g., CFC-12) does not influence the use of other compounds. These feedbacks may be analyzed by specifying alternative production scenarios that reflect the expected substitutions.

- Feedback between years. Each year is analyzed independently, and feedbacks are handled by making previous years' data available through special data structures. To minimize memory requirements, special data structures are established only for those data needed to model feedbacks. Consequently, the addition of new feedbacks between years would likely require modifications to the data structure.

APPENDIX B

SCENARIOS OF CHEMICAL PRODUCTION, POPULATION, AND GNP

INTRODUCTION

This portion of the model generates scenarios of (1) historical and future production of potential ozone depleting substances, (2) population, and (3) economic activity (i.e., gross national product, or GNP). Ten potential ozone-depleting compounds may be specified for analysis: CFC-11; CFC-12; CFC-22; CFC-113; CFC-114; CFC-115; Carbon Tetrachloride (CC14); Methyl Chloroform (CH3CC13); Halon-1211; and Halon-1301. The production of each compound is disaggregated by up to 10 regions of the world, and up to six different end uses or applications. The population and GNP scenarios may also be disaggregated by up to 10 regions of the world.

The remainder of this appendix is organized as follows:

- Production Scenarios describes the standard procedure for specifying scenarios of production (historical and future);
- Population and GNP Scenarios describes the specification of these scenarios;
- User-Modified Scenarios describes how the user may override portions of the standard procedures for specifying the production, population, and GNP scenarios; and
- Limitations presents the limitations of this portion of the model.

PRODUCTION SCENARIOS

Production scenarios may cover any period from 1931 to 2100. Historical data are used through 1984, and a variety of scenarios are specified to reflect potential future production from 1985 onward. To run the model, the user may (1) choose an existing scenario included in the Production Scenario File,[1] (2) create a new scenario in the Production Scenario File, (3) modify an existing scenario using the procedure outlined below in the section on user-modified scenarios, or (4) omit the specification of production from the model by specifying emissions directly (see Appendix D) or ozone depletion directly (see Appendix E). This section describes the format of the existing

[1] The five scenarios included in the Production Scenario File are described in Chapter 3.

scenarios specified in the Production Scenarios File. These scenarios are defined in three parts: global production; regional shares; and end use shares. Each part is described in turn.

Global Production

World production for each of the 10 potential ozone-depleting compounds is reported in units of millions of kilograms per year. Exhibit B-1 shows the middle production scenario for all eight compounds currently analyzed. Intermediate values between the years reported are calculated by the model (using either linear or exponential interpolation, as specified by the user). Production is assumed to be constant following 2050.

Regional Shares

Regional shares are used to divide global production into the following 10 regions:

- U.S.;
- Canada (CAN);
- Western Europe (WEUR);
- Japan, Australia, and New Zealand (JANZ);
- USSR and East Bloc (EUSSR);
- Centrally Planned Asia (ASCEN);
- Middle East (MIDE);
- Africa (AFR);
- Latin America (LA); and
- South and East Asia (SEASIA).

Exhibit B-2 shows a scenario of regional shares for CFC-11. The regional shares are multiplied by the global production to produce regional production estimates. For example, to compute 2000 U.S. CFC-11 production, the model multiplies CFC-11 global production for that year, 543.0 million kg, by the regional share for the U.S. for that year, 0.212, to calculate 115.1 million kg of production. For each year, the shares add to one, indicating that all global production is accounted for when disaggregating across regions. Also of note is that the regional shares may change over time (e.g., in this scenario the U.S. share of world CFC-11 production shrinks from 73.7 percent [0.737] in 1958 to 19.9 percent [0.199] by 1980).

End Use Shares

An "end use share" is the fraction of regional production that is allocated to a particular application or end use. Each compound may have up to six different end uses. End use shares may vary by the 10 regions of the world and may change over time. The model currently includes the following end uses:

- aerosol propellant;
- flexible urethane foam;
- rigid polyurethane foam;

EXHIBIT B-1

Future Global Production Scenarios: Middle Scenario (millions of kilograms)

	CFC-11	CFC-12	CFC-22	CFC-113	CCl4	CH3CCl3	Halon-1211	Halon-1301
1985	375.0	475.0	111.0	163.0	71.2	545.0	10.8	10.8
2000	543.0	688.0	161.0	283.0	103.0	848.0	19.7	19.7
2025	1,006.9	1,275.6	298.1	525.0	191.0	1,534.6	36.5	32.3
2050	1,867.0	2,365.0	552.0	974.0	354.2	2,777.0	67.8	53.1
2075	1,867.0	2,365.0	552.0	974.0	354.2	2,777.0	67.8	53.1

EXHIBIT B-2

Regional Use Shares for CFC-11

Year	U.S.	CAN	WEUR	JANZ	EUSSR	ASCEN	MIDE	AFR	LA	SEASIA	TOTAL
Historical Data											
1931	1.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	1.000
1958	0.737	0.023	0.165	0.075	0.000	0.000	0.000	0.000	0.000	0.000	1.000
1965	0.577	0.037	0.263	0.120	0.001	0.000	0.000	0.000	0.000	0.000	1.000
1970	0.410	0.052	0.364	0.166	0.003	0.001	0.001	0.001	0.001	0.001	1.000
1975	0.333	0.057	0.406	0.185	0.007	0.003	0.002	0.002	0.003	0.002	1.000
1980	0.199	0.067	0.470	0.215	0.019	0.007	0.004	0.004	0.009	0.006	1.000
Scenario Of Future Regional Shares											
1985	0.211	0.035	0.251	0.114	0.149	0.057	0.034	0.034	0.069	0.046	1.000
2000	0.212	0.033	0.231	0.118	0.133	0.066	0.037	0.037	0.081	0.052	1.000
2025	0.212	0.030	0.214	0.116	0.113	0.069	0.046	0.042	0.097	0.061	1.000
2050	0.212	0.028	0.197	0.115	0.093	0.072	0.054	0.046	0.113	0.070	1.000
2100	0.212	0.028	0.197	0.115	0.093	0.072	0.054	0.046	0.113	0.070	1.000

- rigid nonurethane foam;
- refrigeration;
- solvent;
- fire extinguisher; and
- miscellaneous.

Exhibit B-3 shows an example of end use shares for CFC-11 in the U.S.[2] All end use shares for a year add to one (i.e., all production is assigned to some type of end use). In the module, end use shares are multiplied by estimated regional production to estimate the amount of production going into each end use. For example, U.S. 2000 CFC-11 production used to make rigid polyurethane foam would be calculated as follows:

543.0 (2000 CFC-11 Global Production)	x	0.212 (2000 CFC-11 U.S. Regional Share)	x	0.588 (2000 CFC-11 U.S. Rigid Polyurethane Foam Share)	=	67.7 million kg (2000 CFC-11 U.S. Rigid Polyurethane Foam Use)

Using these three factors (global production, regional shares, and end use shares), the historical and potential future production of each of the 10 compounds can be specified in detail. For ease of use, the model incorporates a range of scenarios that reflect a large range of potential future growth in production (0 to 5 percent annual growth). The user may choose one of these scenarios, create a new scenario, or modify an existing scenario as described below in the section entitled User-Modified Scenarios.

POPULATION AND GNP SCENARIOS

A series of population and GNP scenarios are provided in the model.[3] The user has the option of choosing one of these scenarios, or creating his own scenario. Existing scenarios reflect a wide range of projected growth, ranging from 0.5 percent to 1.1 percent growth per year (population from 1985 to 2075) and 1.0 percent to 3.6 percent growth per year for GNP (1985 to 2075). The model does not use historical population or GNP in its analysis.

The population scenario is specified for each of 10 regions in terms of a base year value (1975) and a list of growth indices. Each index value is multiplied by the base year value to produce a population projection for a given year for a region. Exhibit B-4 presents the middle scenario for the U.S. population. To compute the U.S. population projection for 2050, the base year population for the U.S. (213,925,000) is multiplied by the index for 2050 (1.373), which results in a U.S. population projection of 293,719 in 2050. This method allows for the rates of growth of population to vary by region, and to change over time.

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[2] The sources for the end use shares are described in Chapter 3.

[3] Scenarios were presented in Gibbs (1986).

EXHIBIT B-3

U.S. End Use Shares for CFC-11

	Year	Aerosol Propellant	Flexible Urethane Foam	Rigid Polyurethane Foam	Rigid Nonurethane Foam	Refrigeration	Miscellaneous	Total
HISTORICAL DATA	1931	0.000	0.000	0.000	0.000	1.000	0.000	1.000
	1958	0.869	0.045	0.046	0.003	0.011	0.026	1.000
	1970	0.542	0.157	0.162	0.009	0.037	0.093	1.000
	1980	0.132	0.284	0.371	0.020	0.064	0.129	1.000
SCENARIO OF FUTURE END USE SHARES	1985	0.055	0.290	0.490	0.026	0.061	0.078	1.000
	2000	0.038	0.275	0.588	0.031	0.053	0.015	1.000
	2050	0.011	0.283	0.604	0.031	0.055	0.016	1.000

EXHIBIT B-4

Middle U.S. Population Scneario

Year	Population Index	Population (thousands)
1975	1.000	213,925
1985	1.072	229,328
2000	1.192	254,999
2025	1.322	282,809
2050	1.373	293,719
2075	1.376	294,361

EXHIBIT B-5

Middle U.S. GNP Scenario

Year	GNP Index	GNP (millions of (1975 U.S.$)
1975	1.000	1,519,890
1985	1.348	2,048,812
2000	2.040	3,100,576
2025	3.242	4,927,483
2050	4.899	7,445,941
2075	7.830	11,900,739

Regional GNP scenarios are specified in a similar manner. Exhibit B-5 shows the middle scenario for U.S. GNP. The base GNP in 1975 is in 1975 U.S. dollars and the indices correspond to projected years. For example, the model would calculate projected GNP for the U.S. in 2050 by multiplying the base year GNP, $1,519,890 million, by the corresponding index for the U.S. in the year 2050, 4.899. This computation results in a projected 2050 GNP for the U.S. of $7,445,941 million (in 1975 dollars). GNP for other regions is also calculated in 1975 U.S. dollars.

USER-MODIFIED SCENARIOS

The user has the option of overriding portions of the scenarios described in the previous sections. This capability expands flexibility by allowing modified scenarios to be analyzed without creating complete scenario data files for each. This option is especially useful for performing sensitivity analysis. These are six options for modifying the scenarios:

- Population -- The user may specify population projections over time for individual regions. The specified production(s) override(s) the appropriate portion(s) of the standard scenario without changing the projections for other regions.

- GNP -- The user may specify GNP projections over time for individual regions, thereby overriding the projections from the standard scenario without changing the projections for other regions.

- Production by End Use -- The user may specify production (by end use) over time for individual regions. Projections for end uses and regions not explicitly specified are drawn from one of the standard production scenarios.

- Production as a Function of Population -- The user may specify the production of each compound as a function of population for individual regions.

- Production as a Function of GNP -- The user may specify the production of each compound in each region as a function of GNP.

- Production as a Function of GNP per Capita -- The user may specify the production of each compound in each region as a function of GNP per capita. GNP per capita is calculated within the model from the GNP and population scenarios for each region.

For each user-modified scenario the user must specify in a table the type of projection (population, GNP, etc.), the applicable region, the compound (if appropriate), and the end use (if appropriate). For example, to specify 2.0

percent annual growth in the production of CFC-11 in the U.S. from 1985 to 2050, and zero growth thereafter, the user would fill out the table shown in Exhibit B-6. The values in this table would override the values for CFC-11 in the U.S. reported in the standard scenarios.

Another example of a user-specified projection type would be to have U.S. CFC-11 production vary with U.S. population. Exhibit B-7 shows an example of how to specify this production versus population information. The model utilizes this information by (1) determining the level of population for that region, and (2) identifying (by interpolation) the associated production for that population. Because the information specifying the user-modified scenarios is entered in the form of tables, the user is free to adopt any arbitrary function or "shape" for his data. This capability enhances the ability to use the model for sensitivity analysis.

LIMITATIONS

The primary limitations of this scenarios module are described below.

- Range of Standard Scenarios. A set of standard (i.e., pre-defined) scenarios are included in the module, and the user may choose among them. To perform an analysis with scenarios outside the range reflected by the standard scenarios, the user must define his own scenario or modify a standard scenario. The limitation of this approach is that the scenarios are comprised of a significant amount of data, thereby making the establishment of an additional scenario somewhat cumbersome. This limitation is alleviated to some extent by allowing the user to make selective modification to standard scenarios to allow sensitivity analysis.

- Sub-regional Scenarios. Scenarios for areas other than the 10 regions in the model cannot be specified.

- Consistency. The user is free to choose or create individual production, population, and GNP scenarios. In fact, the future production of the different compounds was generally projected under various assumptions regarding population and GNP growth. Therefore, the user should be careful to choose scenarios that are consistent with each other. The model does not check for consistency among the scenarios chosen by the user (i.e., the user may choose a high production scenario and a low population scenario simultaneously).

EXHIBIT B-6

User-Modified Scenario Specifying a Growth Rate of 2 Percent Annually for CFC-11 in the U.S.

Year	Millions of kg of CFC-11
1985	78
2000	105
2025	172
2050	283
2075	283

EXHIBIT B-7

A USER-MODIFIED SCENARIO: PRODUCTION AS A FUNCTION OF POPULATION

Population Millions of People	Production Millions of kg of CFC-11
200	50
250	100
300	200

REFERENCES

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APPENDIX C

EVALUATION OF POLICY ALTERNATIVES

This module evalutes control policy alternatives. If a policy specified by the model user requires reductions in the production of potential ozone-depleting substances, the model simulates these reductions by choosing from a list of possible technical control options. Each technical control is defined by its cost and the percentage reduction that it can achieve.

Because this risk assessment is focused on evaluating risks in the absence of regulation, this module is not employed in the analysis at this time. Therefore, this module is not described at this time.

APPENDIX D

EMISSIONS OF POTENTIAL OZONE-DEPLETING COMPOUNDS

INTRODUCTION

Emissions are estimated on a global basis for each compound being analyzed. Two approaches are available for modeling emissions:

- Internal estimates -- Emissions are modeled based on the production scenarios described in Appendix B and modified through the implementation of policies described in Appendix C. A release algorithm is used to describe how fast the compounds are released into the atmosphere after production. This algorithm is based on "release tables" which are specific for each compound and end use. Each release table shows the annual fraction of the chemical that is released over time.

- Exogeneous specification -- Emission scenarios may be defined directly by the user.

INTERNAL ESTIMATES OF EMISSIONS

The model calculates emissions from release tables and estimates of production developed by the Production Scenario Module (Appendix B) and modified by the Policy Alternatives Module (Appendix C). This section first defines a release table, then describes how releases are estimated from one year's production, and, finally, presents how emissions are calculated from production over a series of years.

Release Tables

Compounds are released into the atmosphere at different rates following production, depending on their uses. Some uses (such as aerosol propellant) release compounds into the atmosphere very soon after production. However, some uses (such as refrigeration and rigid foam production) store compounds for many years, releasing them gradually over time. This storage is a called a "bank."

A release table represents the fraction of production emitted over time for a particular compound and end use. Release tables have been defined for each of the end uses in the model based on Quinn (1986), and the user may specify alternative release tables as desired. Exhibit D-1 presents the release tables for CFC-11 in its end uses. A total of 20 years is shown, and the fraction of the production from year 1 released into the atmosphere in the following years is listed. For example, during the year of production of refrigeration uses 19.0 percent of the compound is released. By the end of the second year a total of 27.1 percent of the production from year 1 is released. Releases continue through the fourth year, after which all of the

EXHIBIT D-1

Release Tables for CFC-11

Year	Aerosol Propellant		Flexible Foam		Rigid Polyurethane Foam		Rigid Nonurethane Foam		Refrigeration		Miscellaneous	
	Cumulative Fraction Released	Annual Fraction Released	Cumulative Fraction Released	Annual Fraction Released	Cumulative Fraction Released	Annual Fraction Released	Cumulative Fraction Released	Annual Fraction Released	Cumulative Fraction Released	Annual Fraction Released	Cumulative Fraction Released	Annual Fraction Released
1	1.000	1.000	1.000	1.000	0.145	0.145	0.600	0.600	0.190	0.190	1.000	1.000
2					0.190	0.045	1.000	0.400	0.271	0.081		
3					0.235	0.045			0.344	0.073		
4					0.280	0.045			1.000	0.656		
5					0.325	0.045						
6					0.370	0.045						
7					0.415	0.045						
8					0.460	0.045						
9					0.505	0.045						
10					0.550	0.045						
11					0.595	0.045						
12					0.640	0.045						
13					0.685	0.045						
14					0.730	0.045						
15					0.775	0.045						
16					0.820	0.045						
17					0.865	0.045						
18					0.910	0.045						
19					0.955	0.045						
20					1.000	0.045						

Source: Quinn, T., et al. (1980), "Projected Use, Emissions, and Bank of Potential Ozone Depleting Substances," The RAND Corporation, prepared for the U.S. Environmental Protection Agency, Washington, D.C.

production from year 1 is released. Annual release rates may be calculated by subtracting each year's cumulative fraction from the previous year's cumulative fraction. For example, 8.1 percent (27.1 minus 0.190) is released in year 2, 7.3 percent (0.344 minus 0.271) is released in year 3.

The release tables vary in length (some end uses store their compounds for many years). Also, the release table need not reflect complete emissions. If the cumulative release fraction in the table reaches 1.0, then all of the compound that is produced is eventually released into the atmosphere. If some of the compound is expected to be retained forever within a product, or destroyed, the release table would have as its highest value a number less than 1.0. The difference between the reported value and 1.0 would be the portion of the compound that is never emitted.

Estimating Emissions From One Year's Production

Exhibit D-2 shows the emissions from a hypothetical production of 100 million kg in 1985. The cumulative fraction of production released over time is presented in column 2. The annual fraction of production released is shown in column 3. The model calculates annual emissions by multiplying the annual fraction by the amount of production in the initial year (100 million kg in this example). The projected emissions per year are shown in column 4. In this example the total emissions sum to the initial production amount, indicating that all of the production is eventually emitted into the atmosphere.

Estimating Emissions From Production Over a Series of Years

Exhibit D-3 presents how the model calculates emissions from production over a series of years. Columns 1 and 2 show years and production in that year, respectively. In this example, production grows by 10 million kg per year, and, for simplicity, emissions from production before 1985 are omitted (emissions from past production are not omitted in the model). Columns 3 through 8 show emissions over time due to the production shown in column 2, each row showing how releases of its production are spread over the course of years.

The first row shows the emissions from 1985 production. The second row shows emissions from the 1986 production, and so on. The total emissions for a year are calculated by summing emissions values in the columns to compute a total. For example, emissions in 1985 are 19.0, emissions in 1986 are 29.0, emissions in 1987 are 39.0, and so on. By 1990, a total of 441.1 million kg has been emitted, and a total of 750 million kg has been produced. Therefore, 750 - 441.1 or 308.9 million kg remains to be emitted after 1990.

This method of estimating emissions reflects the delays between the production and release of many potential ozone-depleting substances. The implication of the method are that releases in a given year are caused not only by production in that year, but also by production and use in previous years.

EXHIBIT D-2

Emissions from a Hypothetical 100 Million Kilograms of Production in 1985

1 Year	2 Cumulative Fraction Released	3 Annual Fraction Released	4 Annual Emissions (millions of kg)
1985	0.190	0.190	19.0
1986	0.271	0.081	8.1
1987	0.344	0.073	7.3
1988	1.000	0.656	65.6
TOTAL		1.000	100.0

EXHIBIT D-3

Emissions from Production Over a Series of Years (millions of kilograms)

Year	Production	1985	1986	1987	1988	1989	1990
1985	100	19.0	8.1	7.3	65.6	--	--
1986	110	--	20.9	8.9	8.0	72.2	--
1987	120	--	--	22.8	9.7	8.8	78.7
1988	130	--	--	--	24.7	10.5	9.5
1989	140	--	--	--	--	26.6	11.3
1990	150	--	--	--	--	--	28.5
TOTAL EMISSIONS PER YEAR		19.0	29.0	39.0	108.0	118.1	128.0

EXOGENOUS SPECIFICATION OF EMISSIONS

The user has the option of directly specifying the level of emissions over time. This is accomplished by filling out an emissions table; a sample table is presented in Exhibit D-4. This table shows emissions estimates for eight chemical compounds over a time period of 90 years. The module can interpolate intermediate emissions values by either linear or exponential interpolation. These specified emissions override the estimates of emissions that the model would have generated from production data as discussed in the previous section. This ability to specify emissions directly to the model allows the flexibility to evaluate the ozone depletion and other effects that may be associated with given emission levels, without having to specify a policy that would result in such emissions.

LIMITATIONS

The primary limitation of this module is that the emission tables are not designed to change over time. Therefore, changes in technology that influence emissions rates of existing end use products cannot be modeled.

EXHIBIT D-4

SAMPLE TABLE OF EXOGENOUSLY SPECIFIED EMISSIONS
(millions of kilograms)

Year	CFC-11	CFC-12	CFC-22	CFC-113	CC14	CH3CC13	Halon-1211	Halon-1301
1985	298	438	81	138	71	500	3	3
2000	462	668	151	240	103	721	9	9
2025	897	1,237	279	446	191	1,303	25	22
2050	1,650	2,281	516	828	355	2,356	48	38

REFERENCES

Quinn, T., et al. (1986), "Projected Use, Emissions, and Bank of Potential Ozone Depleting Substances," The RAND Corporation, prepared for the U.S. Environmental Protection Agency, Washington, D.C.

APPENDIX E

ATMOSPHERIC SCIENCE MODULE

INTRODUCTION

This portion of the model computes the level of global average ozone depletion as a function of the emissions of the 10 potential ozone-depleting substances. The emissions are computed in the previous module (presented in Appendix D). The user has three options for estimating ozone depletion:

- Use a relationship between emissions and ozone depletion developed to reflect the results of a one-dimensional (1-D) atmospheric model;
- Specify his own relationship; and
- Specify the level of ozone depletion directly.

Each of these options is described in turn. Then, the method used to reflect uncertainty is presented. Finally, the limitations of the atmospheric science module are presented.

PARAMETERIZED RELATIONSHIP

The module incorporates a series of equations that describe the change in total column ozone as a function of:

- emissions of potential ozone-depleting substances; and
- abundances of three trace gases (CO2 in ppm, N2O in ppb, and CH4 in ppm).

This relationship was developed by Connell (1986), and it computes global total column ozone change (in percent) relative to 1985.

To use this relationship, the user must specify scenarios of trace gas abundances to use in conjunction with the emissions of potential ozone-depleting substances. A range of scenarios has been developed from which the user may choose (see Chapter 4), or the user may specify his own scenarios. The reference case set of abundances used in the development of the relationship is shown in Exhibit E-1. Exhibit E-2 compares the results of the LLNL 1-D model to the relationship for the base case of emissions of ozone-depleting substances used by Connell to develop the relationship.

The following six steps are performed to compute global ozone depletion in year t using the parameterization:

1. Compute the change in the chlorine concentration in year t relative to 1985 as:

EXHIBIT E-1

TRACE GAS ABUNDANCES USED TO DEVELOP THE OZONE-DEPLETION RELATIONSHIP*

Year	Carbon Dioxide (ppm)	Nitrous Oxide (ppb)	Methane (ppm)
1985	344.5	303.1	1.756
1995	362.0	310.8	1.939
2005	382.8	318.6	2.142
2015	407.4	326.7	2.366
2025	436.4	334.9	2.614
2035	470.3	343.4	2.887
2045	509.9	352.1	3.189
2055	555.9	361.0	3.523
2065	609.1	370.1	3.892
2075	670.7	379.5	4.299

* This base case set of abundances was one of several used to develop the relationship between emissions of ozone-depleting substances and ozone depletion.

Source: Connell, Peter S. (1986), "A Parameterized Numerical Fit to Total Column Ozone Changes Calculated by the LLNL 1-D Model of the Troposphere and Stratosphere," Lawrence Livermore National Laboratory, Livermore, California.

EXHIBIT E-2

COMPARISON OF TOTAL COLUMN OZONE DEPLETION RESULTS FROM THE 1-D MODEL AND THE PARAMETERIZED NUMERICAL FIT*

Year	Ozone Depletion (percent) Numerical Fit	1-D Model	Difference
1985	0.0	0.0	0.00
1995	-0.18	-0.18	0.00
2005	-0.74	-0.61	0.13
2015	-1.64	-1.42	0.22
2025	-2.86	-2.65	0.21
2035	-4.48	-4.30	0.18
2045	-6.63	-6.41	0.22
2055	-9.60	-9.10	0.50
2065	-14.0	-12.9	1.10
2075	-21.6	-19.9	1.70

* This base case was one of many cases used to develop the relationship between emissions of ozone-depleting substances and ozone depletion. This result does not necessarily reflect the base case estimates from this model.

Source: Connell, Peter S. (1986), "A Parameterized Numerical Fit to Total Column Ozone Changes Calculated by the LLNL 1-D Model of the Troposphere and Stratosphere," Lawrence Livermore National Laboratory, Livermore, California.

$$dCl(t) = dCl(\bullet) + \Sigma\ [CF(j)\Sigma emissions(i,j) * \exp(-(t-i)/lifetime(j)) * (1-\exp(-(-t-i)/mixing\ time))]$$

where: dCl(t) equals the change in chlorine concentrations in year t;

dCl(•) equals the change in chlorine concentration in year t due to emissions prior to 1985;

CF equals a conversion factor for each compund j;

emissions are the annual emissions for compound j for years 1985 to year t;

lifetime equals the average atmospheric lifetime for each compound j; and

mixing time equals the time it takes for source emissions to become well mixed.

2. Compute the changes in CO2, N2O and CH4 for year t as follows:

$$dCO2 = CO2(t)/CO2(1985);$$
$$dN2O = N2O(t)/N2O(1985);\ \text{and}$$
$$dCH4 = CH4(t)/CH4(1985),$$

where the values for CO2, N2O and CH4 over time are model inputs as discussed above.

3. Compute ozone depletion due to CFCs as:

$$dO3(t) = 14.58[\text{asinh}(.332(21.05(1+(dN2O-1)/(dCH4)^2) - dCl(t)*\exp(-.15(dCH4-1)))) - \text{asinh}(.332*21.05(1+(dN2O-1)/(dCH4)^2))]$$

where dO3(t) equals the ozone depletion in year t (in percent).

4. Compute ozone depletion (in percent) from CO2, N2O, and CH4 as follows:

$$dO3(t) = 3.6\ln(dCO2);$$
$$dO3(t) = 3.75(dCH4-1);\ \text{and}$$
$$dO3(t) = -7.0(dN2O-1).$$

5. Compute ozone depletion (in percent) from Halons as follows:

$$dO3(t) = -0.0302[\text{concentration of Halon-1301}] - 0.0618[\text{concentration of Halon 1211}]$$

6. Compute total ozone depletion (in percent) as the sum of the individual estimates computed in Steps 3, 4, and 5.

These equations are used to represent the potential for ozone depletion as a function of emissions and concentrations of the various substances analyzed. The detailes of this method are described in Connell (1986).

USER-SPECIFIED RELATIONSHIP

The user has the option of specifying his own relationship between emissions and ozone depletion. For each substance, the user specifies a "conversion factor" and a "lifetime." These values are used in the following equation to compute an atmospheric abundance for each compound, over time:

$$\text{Abundance (T)} = \text{Conversion Factor} \sum_{t=t_o}^{T} [\text{emissions (t)} e^{-(T-t)/\text{lifetime}}].$$

The abundances are summed across the compounds to create a total abundance. The amount of ozone depletion associated with each level of abundance is then specified by the user in a table.

This formulation of the user-specified ozone-depletion relationship is similar to the parameterized numerical fit developed by Connell (1986). It can produce abundances of the different compounds that exhibit behavior similar to the results produced by 1-D models. The value of this option is that the user may identify, in tabular form, his best understanding of the manner in which ozone depletion will vary with the abundances of the compounds in the atmosphere.

USER-SPECIFIED OZONE DEPLETION

The user has the option of bypassing the computation of ozone depletion based on emissions of potential ozone-depleting substances. Instead, the user may provide estimates of global ozone depletion directly to the model. This option allows the user to evaluate the impacts of such a level of depletion (possibly computed via other means) without identifying a level of emissions or a control policy that would produce such depletion.

To specify levels of ozone depletion, the user fills out the table shown in Exhibit E-3. For each year, a level of total column ozone depletion (or increase) is identified. The model interpolates between the values supplied in the table.

UNCERTAINTY

The user has the option of recognizing the uncertainty inherent in current models of ozone depletion by specifying a range within which the "true" level of ozone depletion is expected to fall. This range is specified using multiplication scaling factors, as shown in Exhibit E-4. The scaling factors are multiplied by the estimate of ozone depletion produced within the

EXHIBIT E-3

HYPOTHETICAL TABLE OF USER-SPECIFIED OZONE DEPLETION*

Year	Total Column Global Ozone Depletion (percent)
1985	0.00
1990	0.01
2000	0.10
2025	1.00
2050	5.00
2075	10.00
2100	20.00

* Values are illustrative only.

EXHIBIT E-4

EXAMPLE OZONE-DEPLETION SCALING FACTORS*

Ozone Depletion (percent)	Scaling Factors	
	Low	High
≤15.0	0.4	2.0
≥20.0	0.5	1.8

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* These factors represent the 10th and 90th fractile estimates from a lognormal distribution of uncertainty developed from values reported in: Stolarski, R.S., and A.R. Douglass (1986), Sensitivity of an Atmospheric Photochemistry Model to Chlorine Perturbation Including Consideration of Uncertainty Propagation, Draft Report to the U.S. Environmental Protection Agency, Washington, D.C.

model.[1] These multiplications produce a range of ozone depletion values for each year; the end points of the range (as well as the middle value estimated within the model) are passed to the effects module for evaluation.

As shown in Exhibit E-4, the scaling factors may change at different levels of ozone depletion. The values shown in the exhibit are based on a Monte Carlo uncertainty analysis performed by Stolarski and Douglass (1985). Each Monte Carlo iteration draws from a distribution of estimated reaction rates and cross sections to generate predictions of atmospheric abundances. These predicted abundances can be compared to actual measurements in the atmosphere; those iterations that do not compare favorably with actual measurements can be screened out. The values reported in Exhibit E-4 are based on those iterations that compared favorably with actual measurements and were not screened out.

Because the ozone-depletion models are not considered reliable at high levels of ozone depletion, the user may also specify a "cap" on ozone depletion. If the computed level of ozone depletion (after scaling) exceeds the user specified cap, then the ozone depletion used to evaluate effects (in the next module) is reduced to the level of the cap.

LIMITATIONS

The Atmospheric Science Module is a simplified representation of a complex set of atmospheric reactions. In using the module, the user should become aware of the range of emissions and trace gas values for which the parameterized relationship is considered valid. Outside this range, the equations do not necessarily reflect current estimates from 1-D models of the atmosphere. The range of production scenarios discussed in Appendix B and Chapter 3 are well within the range for which these equations are valid.

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[1] The scaling factors are not used when ozone depletion is specified directly by the user.

REFERENCES

Connell, Peter S. (1986), "A Parameterized Numerical Fit to Total Column Ozone Changes Calculated by the LLNL 1-D Model of the Troposphere and Stratosphere," Lawrence Livermore National Laboratory, Livermore, California.

Stolarski, R.S., and A.R. Douglass (1985), "Sensitivity of an Atmospheric Photochemistry Model to Chlorine Perturbation Including Consideration of Uncertainty Propagation," Draft Report to the U.S. Environmental Protection Agency, Washington, D.C.

APPENDIX F

HEALTH AND ENVIRONMENTAL IMPACTS OF OZONE DEPLETION

INTRODUCTION

This appendix presents the methods used to model the health and environmental effects of ozone depletion in the CFC Policy Model. This module incorporates analyses performed by a variety of researchers, including analyses of:

- the relationship between global ozone depletion and latitudinal-dependent ozone depletion (Isaksen 1986);
- the relationship between ozone depletion and ultraviolet radiation (UV);[1]
- the relationship between UV and the incidence (and mortality) of non-melanoma skin cancers;[2]
- the relationship between UV and the incidence (and mortality) of melanoma skin cancers;[3]
- the relationship between UV and the prevalence (and incidence) of senile cataracts;[4] and
- the relationship between ozone depletion and the costs of modifying polymers to prevent degradation of polymer appearance and/or performance due to increased UV.[5]

These analyses pertain primarily to the U.S., consequently, this module focuses on the risks in the U.S. associated with ozone depletion. The module is designed to allow risk estimates to be performed for other portions of the

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[1] See Serafino and Frederick (1986) for a description of the model used to estimate the relationship between ozone depletion and UV flux.

[2] See Chapter 7 for a discussion of the relationship between non-melanoma skin cancer and UV.

[3] See Chapter 8 for a discussion of the relationship between melanoma skin cancer and UV.

[4] See Chapter 10 for a discussion of the relationship between senile cataracts and UV.

[5] See Chapter 13 for a discussion of the relationship between polymer degradation and UV.

world if: (1) the model user supplies data needed to quantify effects in these other regions; or (2) the model user assumes that the data for the U.S applies to other portions of the world. The descriptions below relate exclusively to the analysis of impacts in the U.S.

This appendix is divided into the following sections:

- Ozone Depletion by Latitude presents the method used to translate the estimates of global ozone depletion obtained from the Atmospheric Science Module (see Appendix E) into estimates of ozone depletion that vary by latitude;
- Changes in UV Flux presents the method used to estimate changes in the flux of UV for given levels of ozone depletion;
- Skin Cancers and Cataracts presents the methods used to model melanoma and non-melanoma skin cancers and cataracts;
- Materials presents the methods used to model the costs associated with the degradation of polymers; and
- Limitations summarizes the major limitations of the methods used, including the risks believed to be associated with increases in ambient levels of UV that are not modeled at this time in the CFC Policy Model.

OZONE DEPLETION BY LATITUDE

The Atmospheric Science Module (Appendix E) uses a parameterized numerical fit to a 1-dimensional model of the stratosphere to estimate average global changes in total column ozone on an annual basis (Connell 1986). Two-dimensional models indicate that ozone changes due to increases in stratospheric concentrations of potential ozone depleters are not likely to be uniform across the globe (Isaksen 1986). A strong latitudinal gradient is expected with larger than average levels of depletion occurring at higher latitudes (e.g., above 40 degrees north latitude).

This portion of the module translates the estimates of average global ozone change from the Atmospheric Science module into latitudinal-dependent estimates based on analysis of data presented by Isaksen (1986). A linear relationship is used to estimate latitudinal ozone depletion as a function of global ozone changes and changes in methane concentrations. Methane concentrations are used in the equation because the effects of methane on the ozone column are latitudinally dependent. The general form of the linear relationship used in this module is:

$$OZ_i = A_{i,GOZ}GOZ + B_{i,GOZ}GM + E_{i,GOZ}$$

where OZ_i = total column ozone depletion at latitude i, GOZ = the average global ozone depletion, GM = the average global methane concentration, and A, B, and E are constants for each latitude i and range of GOZ. The average global methane concentration may be used to estimate latitudinal effects because the latitudinal methane concentrations show a high degree of correlation with their corresponding global average.

Note that in the above equation A, B, and E vary by average global ozone depletion as well as by latitude. These coefficients have been estimated for three ranges of ozone depletion. In each range, the relationship described by the above equation is nearly linear. These ranges are:

- Global ozone depletion greater than three percent. Latitudinal results were found to be highly correlated with the global average.
- Global ozone depletion greater than 1.5 percent but less than three percent. Methane concentrations were found to have an important role in estimating latitudinal ozone changes.
- Global ozone depletion less than 1.5 percent (including ozone abundance). No data existed for this range, linearity was assumed.

Range 1: Global Ozone Depletion Greater Than 3.0 Percent

Within this range, latitudinal ozone depletion was found to be highly correlated with the global average of ozone depletion, across all the latitudes. Methane was found to have a negligible role in this range (i.e., B is zero in the equation). Exhibit F-1 shows the values used for the global ozone coefficient (A) and the intercept term (E) for this range of global ozone values. These coefficients were estimated using ordinary least squares regression analysis with data from Isaksen (1986). Exhibit F-2 compares the results from Isaksen with those generated by the linear relationship, using a scenario where all CFCs grow at 3.0 percent per year. The global ozone depletion values reported by Isaksen (shown at the top of the exhibit) was inserted into the equation using the coefficients listed in Exhibit F-1. The latitudinal values estimated with the equation are very close to those reported by Isaksen.

Range 2: Global Ozone Depletion Greater Than 1.5 Percent and Less Than 3.0 Percent

Within this range, global average methane concentrations (measured in ppm) (in addition to average global ozone depletion) were found to have an important effect on estimates of latitudinal depletion. Coefficients for the linear relationship (A, B and E) were determined using bivariate regressions for each latitude. Exhibit F-3 shows the coefficients obtained from these regressions. Exhibit F-4 compares the results from Isaksen with those generated by the linear relationship, using a scenario where all CFCs grow at 3.0 percent per year.

EXHIBIT F-1

Coefficient used to Estimate Ozone Depletion at Latitudes when Global Average Depletion Exceeds 3.0 Percent

LATITUDE	A	E
60 N	1.32185	1.08857
50 N	1.12836	0.60972
40 N	0.96756	0.15419
30 N	0.91823	-0.02698
20 N	0.92589	-0.02349
10 N	0.90047	-0.05004
0	0.88249	-0.06526
10 S	0.86839	-0.10497
20 S	0.81941	-0.33460
30 S	0.80351	-0.57032
40 S	0.85886	-0.65536
50 S	0.99591	-0.49032
60 S	1.12253	-0.37618

EXHIBIT F-2

Comparison of Estimate of Latitudinal Ozone Depletion when Global Ozone Depletion Exceeds 3.0 Percent: Isaksen versus the Linear Relationship

LATITUDE	YEAR	ISAKSEN	LINEAR MODEL
Global	2010	3.73	--
	2020	5.75	--
	2030	8.73	--
60 N	2010	5.90	6.02
	2020	8.68	8.69
	2030	12.50	12.62
50 N	2010	4.77	4.82
	2020	7.11	7.10
	2030	10.39	10.46
40 N	2010	3.76	3.76
	2020	5.76	5.72
	2030	8.59	8.60
30 N	2010	3.41	3.40
	2020	5.31	5.26
	2030	7.98	7.99
20 N	2010	3.46	3.43
	2020	5.34	5.30
	2030	8.07	8.06
10 N	2010	3.34	3.31
	2020	5.16	5.13
	2030	7.82	7.81
0	2010	3.25	3.23
	2020	5.05	5.01
	2030	7.64	7.64

EXHIBIT F-2
(continued)

Comparison of Estimate of Latitudinal Ozone Depletion when Global Ozone Depletion Exceeds 3.0 Percent: Isaksen versus the Linear Relationship

LATITUDE	YEAR	ISAKSEN	LINEAR MODEL
10 S	2010	3.17	3.13
	2020	4.91	4.89
	2030	7.50	7.47
20 S	2010	2.77	2.72
	2020	4.40	4.38
	2030	6.86	6.82
30 S	2010	2.47	2.43
	2020	4.07	4.05
	2030	6.51	6.44
40 S	2010	2.61	2.55
	2020	4.29	4.28
	2030	6.90	6.84
50 S	2010	3.22	3.22
	2020	5.20	5.24
	2030	8.24	8.20
60 S	2010	3.79	3.81
	2020	6.00	6.08
	2030	9.45	9.42

Source: Isaksen values reported in Isaksen, I. (1986), "Ozone Perturbations Studies in a Two-Dimensional Model with Temperature Feedback in the Stratosphere Included," presented at UNEP Workshop on the Control of Chlorofluorocarbons, Rome, Italy, May 1986.

EXHIBIT F-3

Coefficients used to Estimate Ozone Depletion of Latitudes when Global Depletion Exceeds 1.5 Percent and is Less Than 3.0 Percent

LATITUDE	A	B	E
60 N	1.29733	1.14576	-1.46820
50 N	1.14571	0.47717	-0.56695
40 N	1.01811	-0.10520	0.19535
30 N	0.98513	-0.30283	0.43503
20 N	0.94876	-0.23606	0.39426
10 N	0.89818	-0.17885	0.32953
0	0.88837	-0.13225	0.20192
10 S	0.89806	-0.24961	0.37560
20 S	0.83801	-0.48130	0.66795
30 S	0.82972	-0.67765	0.89665
40 S	0.84437	-0.55604	0.67525
50 S	0.91496	-0.14140	0.12097
60 S	1.00523	0.13850	-0.37618

EXHIBIT F-4

Comparison of Estimates of Latitudinal Ozone Depletion when Global Ozone Depletion Exceeds 1.5 Percent and is Less than 3.0 Percent: Isaksen versus the Linear Relationship

LATITUDE	YEAR	ISAKSEN	LINEAR MODEL
Global	2000	2.33	--
60 N	2000	3.85	3.90
50 N	2000	3.05	3.08
40 N	2000	2.33	2.36
30 N	2000	2.11	2.12
20 N	2000	2.15	2.13
10 N	2000	2.10	2.06
0	2000	2.01	2.00
10 S	2000	1.98	1.96
20 S	2000	1.66	1.64
30 S	2000	1.46	1.45
40 S	2000	1.52	1.51
50 S	2000	1.94	1.97
60 S	2000	2.33	2.36

Range 3: Global Ozone Depletion Less Than 1.5 Percent

Data from Isaksen (1986) did not cover this range of ozone depletion extensively. To estimate the coefficients, it is assumed that a value of zero global ozone depletion implies zero latitudinal ozone depletion (rather than some abundance and some depletion. This assumption implies that in this range of global depletion, both the methane coefficient (B) and the intercept term (E) in the linear relationship must be set to zero, and latitudinal ozone depletion is obtained by multiplying the average global ozone depletion by a constant. Exhibit F-5 shows the coefficients used by the model for this depletion range. The values were estimated as the ratio of latitudinal to global depletion values from Isaksen's data. By averaging over all results in each latitude, a latitudinal-dependent scaling factor was be calculated. Exhibit F-6 compares the results from Isaksen with those generated by the linear relationship, using a scenario where all CFCs grow at 3.0 percent per year.

The module used the relationships described above to estimate ozone depletion by latitude. The global ozone depletion and methane concentrations are estimated in the Atmospheric Science Module (Appendix E). The equation for the global ozone depletion ranges are used as appropriate. Because these equations were developed from a limited data set from a single 2-D model, the latitudinal estimates should be viewed as uncertain. In particular, the estimates of the effects of methane concentrations may be valid for a fairly narrow range of potential future values because the estimates of the coefficients are based on a single methane concentration scenario. The equation and their coefficients do not necessarily correspond to specific physical properties and processes.

CHANGES IN UV FLUX

This portion of the module evaluates the expected changes in UV flux associated with changes in total column ozone. The estimates of changes in UV flux vary by latitude because:

1. the change in UV flux (measured in percent) for a given change in total column ozone (also measured in percent) varies as a function of the initial total abundance of ozone (i.e., the level of ozone abundance in the absence of ozone depletion); and

2. the initial level of total column ozone varies by latitude.

The relationship between ozone change and UV flux will also vary for different measures of UV energy. Three measures of UV, also referred to as action spectra, are currently incorporated into the model: Robertson-Berger Meter (RB-Meter); DNA Action Spectrum; and Erythema Action Spectrum. Each measure has a unique weighting of the wavelengths in the UV spectrum. Because ozone selectively screens out radiation at given wavelengths, the different weighting schemes will produce different estimates of increased radiation levels when total column ozone abundance changes.

EXHIBIT F-5

Coefficients Used to Estimate Ozone Depletion at Latitudes when Global Depletion is Less than 1.5 Percent

LATITUDE	A
60 N	1.87118
50 N	1.40759
40 N	0.99645
30 N	0.85819
20 N	0.87931
10 N	0.86197
0	0.83994
10 S	0.80330
20 S	0.62353
30 S	0.50484
40 S	0.54594
50 S	0.81227
60 S	1.02931

EXHIBIT F-6

Comparison of Latitudinal Ozone Depletion when Global Ozone Depletion is Less Than 1.5 Percent: Isaksen versus the Linear Relationship

LATITUDE	YEAR	ISAKSEN	LINEAR MODEL
Global	1980	0.65	
	1990	1.33	
60 N	1980	1.13	1.22
	1990	3.85	3.90
50 N	1980	0.86	0.92
	1990	1.80	1.88
40 N	1980	0.62	0.65
	1990	1.34	1.33
30 N	1980	0.60	0.56
	1990	1.20	1.15
20 N	1980	0.65	0.57
	1990	1.23	1.17
10 N	1980	0.64	0.56
	1990	1.21	1.15
0	1980	0.62	0.55
	1990	1.19	1.12
10 S	1980	0.62	0.52
	1990	1.15	1.07
20 S	1980	0.44	0.41
	1990	0.91	0.83
30 S	1980	0.36	0.33
	1990	0.76	0.67
40 S	1980	0.36	0.35
	1990	0.79	0.73
50 S	1980	0.50	0.53
	1990	1.10	1.08
60 S	1980	0.64	0.67
	1990	1.33	1.37

The UV Model developed by Serafino and Frederick (1986) was used to evaluate the expected changes in UV flux for given levels of total column ozone change. As described above in Appendix A of Chapter 7, the estimates of UV flux from the UV model reproduce field measurements taken with RB-meters. The correlation between annual RB-meter values from the UV model and field instruments is 0.979. Pitcher (1986) performed a more complete validation of the UV model, comparing monthly model estimates to monthly field instrumentation readings for three cities: El Paso, San Francisco, and Minneapolis. Although the model was found to overestimate UV flux in the months of September through March, and underestimate UV flux in May, June, and July, the annual estimates are generally within 10 percent of the RB-meter readings in the field.

Changes in UV flux (as measured by the three action spectra) as a function of changes in ozone abundance were evaluated using the UV model for 24 cities across the U.S. These cities are divided into three regions of the U.S.: north, middle, and south (see Exhibit F-7). These three regions are used to evaluate potential changes in the incidence and mortality due to skin cancers. The states included in each region are shown in Exhibit F-8. The population weighted average latitudes for the three regions are also shown.

Exhibit F-9 shows the percent change in UV (measured with the DNA action spectrum) in the three regions of the U.S. associated with a range of percent changes in total column ozone (from a 10 percent increase in ozone (listed as a -10 percent ozone depletion) to a 30 percent ozone depletion). As expected, the change in UV for a given level of ozone change is not constant across the three regions of the U.S., although they are very similar. Statistical analysis indicates that the estimates for the three regions are statistically different from each other.

Similar tables have been developed for the RB-Meter and the Erythema action spectra. The values for the Erythema Action Spectrum are approximately 80 to 85 percent of the DNA Action Spectrum values; the RB-Meter values are 35 to 45 percent. The user may define additional action spectra and prepare additional tables that relate changes in UV flux to changes in ozone depletion if he wishes to evaluate effects as a function of different action spectra.

SKIN CANCERS AND CATARACTS

The objective of this portion of the module is to evaluate the potential changes in risks of melanoma and non-melanoma skin cancers and cataracts associated with changes in total column ozone abundance over time. The purpose of the evaluation is to provide information for assessing the implications of alternative emissions rates of potential ozone modifying substances. The inputs to this portion of the module include estimates of changes in UV flux associated with changes in total column ozone abundance. The output of this portion of the module includes estimates of the increased number of cases and deaths associated with skin cancers over time and an estimate of the increased number of cases of cataracts.

EXHIBIT F-7

Cities Used to Evaluate Changes in UV Flux for the Three Regions of the U.S.

REGION 1: NORTH	REGION 2: MIDDLE	REGION 3: SOUTH
New York	Chicago	Los Angeles
Detroit	Philadelphia	San Diego
Milwaukee	Baltimore	Houston
Boston	San Francisco	Dallas/Fort Worth
Seattle	Washington	Phoenix
Minneapolis	Denver	New Orleans
Portland	Salt Lake City	Miami
Buffalo	Kansas City	Atlanta

EXHIBIT F-8

States Includes in the Three Regions of the U.S.

REGION 1: NORTH	REGION 2: MIDDLE	REGION 3: SOUTH
Alaska	California (N) a/	Alabama
Connecticut	Colorado	Arizona
Idaho	Delaware	Arkansas
Maine	District of Columbia	California (S) a/
Massachusetts	Illinois	Florida
Michigan	Indiana	Georgia
Minnesota	Iowa	Hawaii
Montana	Kansas	Louisiana
New Hampshire	Kentucky	Mississippi
New York	Maryland	New Mexico
North Dakota	Missouri	South Carolina
Oregon	Nebraska	Texas
Rhode Island	Nevada	
South Dakota	New Jersey	
Vermont	North Carolina	
Washington	Ohio	
Wisconsin	Oklahoma	
	Pennsylvania	
	Tennessee	
	Utah	
	Virginia	
	West Virginia	
	Wyoming	
Latitude = 43.3 N	Latitude = 39.1 N	Latitude = 31.8 N

a/ California is divided in half, one half being included in the Middle Region, and one half included in the South Region.

Source: Latitude estimates based on population centroids for each state from the 1980 U.S. census, Master Area Reference File #2, Geography Section, U.S. Bureau of Census, Department of Commerce.

EXHIBIT F-9

Percent Change in UV as a Function of Change in Ozone Abundance for Three U.S. Regions
(DNA Action Spectrum)

OZONE DEPLETION (%)	CHANGE IN UV (%) North	Middle	South
-10	-17.3	-17.2	-16.7
-5	-9.3	-9.1	-8.9
-2	-3.8	-3.8	-3.8
0	0.0	0.0	0.0
2	4.2	4.3	4.2
5	10.8	10.6	10.5
10	22.9	22.8	22.2
20	53.8	53.2	51.0
30	96.0	94.8	90.4

Source: Based on analyses using the UV Model developed by Serafino and Frederick (1986).

This analysis is organized into four parts as follows:

1. characterize the population at risk;

2. estimate the baseline incidence of the conditions being evaluated;

3. apply dose-response relationships to estimate the change in the risk of the conditions as a function of changes in ozone abundance over time; and

4. summarize the results.

Data have been developed primarily for the three regions of the U.S. for purposes of evaluating these skin cancer and cataract risks (see above for the definition of the three regions in the U.S.). The risks of these cancers in other portions of the world can be evaluated by the model user if: (1) the user supplies data to characterize the populations at risk in the other regions, the baseline incidence in the other regions, and the dose-response relationships that apply to the other regions; or (2) the user assumes that the information provied for the U.S. applies to the other regions. Each of the four steps is presented for the U.S.

Step 1 -- Characterize the Population at Risk

The incidence of skin cancers and cataracts has been found to vary by race, age, sex, and the ambient level of UV in the area of residence of the individual.[6] The population in the U.S. is characterized by age, sex, and race using data from the U.S. Bureau of the Census.[7] Each of the three regions in the U.S. are characterized separately.

For each region, the current and expected age distribution of the population is defined in a series of tables as shown in Exhibit F-10. These age distributions were obtained by summing the age distributions of all the states in the region (in this case the North region), and dividing by the total population in the region.

The age and regional distributions of the U.S. population are expected to change over time due to migration within the U.S. and due to changing survival and birth rates. These changes in the distribution through 2000 are included in the characterization of the population, as shown in Exhibit F-10. Changes beyond 2000 have not been estimated, and consequently the age and regional

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[6] See Chapter 7 for a discussion of non-melanoma skin cancer, Chapter 8 for a discussion of melanoma skin cancer, and Chapter 10 for a discussion of cataracts.

[7] The Census Data were obtained from NPA Data Services.

EXHIBIT F-10

Age Distribution of the U.S. Population Over Time in the North Region

(fraction of total population in the region)

YEAR	AGE GROUP									FRACTION OF TOTAL U.S. POP.*
	0-14	15-24	25-34	35-44	45-54	55-64	65-74	75-84	85+	
1985	.2074	.1667	.1842	.1288	.0892	.0947	.0716	.0385	.0189	.2486
2000	.2119	.1299	.1314	.1645	.1425	.0892	.0642	.0445	.0219	.2439

* The fraction of the total U.S. population represents the portion of the U.S. population that is estimated to reside in this region (in this case the North region). This fraction changes over time to reflect expected migration patterns.

Source: Based on analyses of data supplied by NPA Data Services, Inc.

distributions are assumed to remain fixed beyond 2000. Because the U.S. population may continue to become older on average into the 21st century, this fixed population distribution under-estimates the population of older individuals, and may consequently under-estimate risks of ozone depletion. The user may wish to relax this assumption by inserting alternative data for years beyond 2000 as appropriate.

The portions of the population in each region that are male and female, black and while are also defined. These portions may change over time within the model to reflect changing demographic patterns.

From these data the fraction of the U.S. population is characterized by age, race, and sex for each of the three regions of the country. The total U.S. population over time is provided as an input to the Scenarios Module (see Appendix B), and is used as the basis of estimating the number of people in each age, race, and sex grouping.

Step 2 -- Baseline Incidence

To evaluate the potential future risk of skin cancers in the U.S. the baseline risk (i.e., the risk in the absence of changes in the level of total column ozone) is required. Incidence of melanoma and non-melanoma skin cancers has been found to vary by latitude. Therefore, baseline incidence values have been develeped for each of the three U.S. regions separately. The age-sex specific baseline rates used for melanoma and non-melanoma are described separately. The baseline data for cataracts follow.

Non-melanoma Skin Cancer

There are two major types of non-melanoma skin cancers: basal cell and squamous cell (Scotto, Fears, and Fraumeni 1981; and see Chapter 7). Because the two types may respond differently to changes in UV, their baseline incidences are estimated separately (Scotto, Fears, and Fraumeni 1981). Data describing the age-sex specific incidence for whites are available for 10 locations from Scotto, Fears, and Fraumeni (1981). The incidence rates by age and sex were estimated for each of the three U.S. regions by taking the population-weighted averages of the rates for the cities reported in Scotto, Fears, and Fraumeni (1981) that are in each of the regions. The locations in each region are:

North: Seattle, Minneapolis-St. Paul, Detroit;
Middle: Utah, San Francisco, Iowa; and
South: Atlanta, New Orleans, New Mexico, Dallas-Forth Worth.

Exhibit F-11 displays an example of the age specific incidence rates for basal and squamous cell skin cancers in the North region of the U.S. These data apply only to whites.

The baseline incidence rates for Blacks and members of other races are not well characterized. The rates are believed to be approximately one order of magnitude or more below the rates for whites, and are consequently not

EXHIBIT F-11

Baseline Incidence for Non-Melanoma Skin Cancers
(North Region of the U.S. -- White population only)
(Rate per 100,000)

	AGE GROUP								
	15	15-24	25-34	35-44	45-54	55-64	65-74	75-84	85+
NONMELANOMA SKIN CANCER									
Basal Cell Skin Cancer									
Male	0.1	2.9	22.1	91.1	259.2	465.8	761.0	1162.8	1311.3
Female	0.5	5.6	22.2	91.0	201.8	287.4	465.9	638.2	754.1
Squamous Cell Skin Cancer									
Male	0.2	0.3	1.6	7.4	32.6	87.4	147.4	349.7	431.8
Female	0.0	0.1	1.4	4.1	10.5	27.5	54.8	112.5	167.7

Source: Derived from Scotto, Fears, and Fraumeni (1981).

expected to play a major role in evaluating risks of skin cancers in the U.S. (they may play a more important role in the risks in other countries). For purposes of this analysis, risks for Blacks and members of other races are not evaluated.

The mortality associated with each of these cancers is believed to be quite low. Approximately 1.0 percent of all cases of nonmelanoma skin cancer result in death (Scotto, Fears, and Fraumeni 1981). The rates for basal and squamous cell cancer differ, and have been estimated from data in Scotto, Fears and Fraumeni to be: basal cell -- 0.3 percent; and squamous cell -- 3.75 percent. These percentages are multiplied by the baseline incidence rates to estimate the baseline rates of death associated with each cancer type over time. The model user may modify these mortality values to explore the implications of alternative assumptions.

The model user may also specify that the baseline incidence and mortality ratio may change over time (increase or decrease). The purpose of this specification is to allow the user to include the implications of potential changes in the baseline incidence of these skin cancer that are not related to changes in UV flux.

Melanoma Skin Cancer

There are four principal classes of melanoma skin cancers: Hutchinson's melanotic freckle, superficial spreading melanoma, nodular melanoma and unclassifiable melanoma (Elder et al. 1980). For purposes of modeling the baseline incidence of these cancers, two groups are defined that are based on the location of the cancer on the body:

- Group 1: Face, Head, Neck, and Upper extremities; and
- Group 2: Trunk and Lower Extremities.

These groups have been defined by Scotto and Fears (in press), and incidence data have been collected for seven U.S. cities that divide the occurrence of melanoma skin cancers into these groups. The division of these cancers into these groups appears to be warranted because Scotto and Fears have identified different correlations with UV for each of the two groups.

The incidence data for these groups of melanoma skin cancer were developed for the three regions of the U.S. by taking population weighted averages of the incidence data reported for the following seven locations in the three regions:[8]

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[8] Although the National Cancer Institute SEER Report (1984) reports data for ten locations, incidence data for only seven locations were used. These seven locations correspond to the locations for which Scotto and Fears (in press) reported body site percentages (face, head, neck, and upper extremities vs. trunk and lower extremities) for melanoma incidence. The three locations that were not included in this analysis are: Connecticut, Hawaii, and Puerto Rico.

- North: Seattle, Detroit;
- Middle: Utah, San Francisco, Iowa; and
- South: Atlanta, New Mexico.

The incidence data were taken from the National Cancer Institute, Cancer Incidence and Mortality in the United States: SEER Report (1984), and the data apply to whites only. Because the SEER data do not divide the incidence by the two groups of melanoma skin cancer defined above, the fractions of age specific incidence associated with each type of cancer were taken from Scotto and Fears (in press).

Exhibit F-12 displays sample baseline incidence data for melanoma skin cancer by sex and age for whites in the North U.S. region. As shown in the exhibit, the incidence rates for the two types of melanoma were estimated separately.

Mortality data were taken from the SEER Report (1984) for whites only. Because the SEER data do not divide the mortality rates by the two groups of melanoma skin cancer defined above, mortality is modeled without dividing the data into the two cancer types. Exhibit F-13 displays the mortality rates currently used in the model.

The incidence and mortality associated with melanoma skin cancers in nonwhite populations in the U.S. are less well defined. The rates for nonwhites are much lower than the rates for whites, and are consequently not modeled.

The model user may also specify that the baseline incidence rates may change over time (increase or decrease). The purpose of this specification is to allow the user to include the implications of potential changes in the baseline incidence of melanoma skin cancer that are not related to changes in UV flux.

Senile Cataracts

The prevalence of senile cataracts has been found to be correlated with UV radiation (Hiller, Sperduto, Ederer 1983). The baseline prevalence, however, is not well characterized by the three regions of the U.S. Therefore, the prevalence (and associated incidence) of senile cataracts is modeled for the entire U.S. as one region.

Several diagnostic criteria have been used to define the occurrence of senile cataracts. The definition used in this module was reported by Leske and Sperduto (1983) and used by Hiller, Sperduto and Ederer (1983) to estimate a multivariate logistic risk function that includes UV-B. The diagnostic criteria include: cataract, defined as "senile lens changes consistent with best corrected visual acuity of 20/30 (6/9) or worse;" or aphakia, defined as "the lens had been surgically removed and there was no history of congenital, traumatic or secondary cataract" (Hiller, Sperduto, and Ederer 1983). Exhibit F-14 displays the baseline prevalence values used in the module. Baseline incidence (as well as increased incidence due to ozone depletion) is estimated by computing the rate of change of prevalence overtime.

EXHIBIT F-12

Baseline Incidence for Melanoma Skin Cancers (North Region of the U.S. -- White Population Only) (Rate per 100,000)

Melanoma Skin Cancer	Age Group 10-14	15-24	25-34	35-44	45-54	55-64	65-74	75-84	85+
Face, Head, Neck and Upper Extremities									
Males	0.0	0.8	2.4	5.3	5.5	8.4	10.4	9.2	9.1
Females	0.1	0.9	3.2	4.2	5.3	5.4	4.5	4.6	4.9
Trunk and Lower Extremities									
Males	0.0	1.2	3.4	7.5	7.9	12.0	14.9	13.2	13.0
Females	0.1	1.4	5.3	6.9	8.6	8.9	7.4	7.4	8.0

Source: Derived from Scotto and Fears (in press) and National Cancer Institute SEE Report (1984).

EXHIBIT F-13

Mortality Rates for Melanoma Skin Cancers (White Population Only) (Rate per 100,000)

	Age Group								
	10-14	15-24	25-34	35-44	45-54	55-64	65-74	75-84	85+
Male	0.0	0.5	1.0	2.9	4.6	5.5	8.0	7.7	10.3
Female	0.0	0.0	0.8	1.6	2.8	2.9	3.3	5.3	5.5

Source: Derived from Scotto and Fears (in press) and National Cancer Institute SEE Report (1984).

EXHIBIT F-14

Baseline Prevalence of Senile Cataracts (Rate per 100,000)

	AGE				
	≤54*	55-64	65-74	75-84	85+
Male	0.0	4,300	16,000	40,900	40,900
Female	0.0	4,700	19,300	48,900	48,900

* Prevalence for individuals under 54 years of age assumed to be zero.

Source: Leske C.L. and R.D. Sperduto (1983), "The Epidemiology of Senile Cataracts: A Review," American Journal of Epidemiology, Vol. 118, No. 2, pp. 152-165.

Step 3 -- Apply Dose-Response Relationships

The purpose of applying the dose-response relationships is to estimate how the risks of melanoma and non-melanoma skin cancers and cataracts may change as the flux of UV reaching the earth's surface changes due to changes in total column ozone abundance. Two factors are required to apply dose response relationshps: (1) a measure of exposure; and (2) a dose-response equation. Each is described in turn.

The measure of exposure used to model skin cancers and cataracts is related to the level of ambient UV flux estimated to reach the earth's surface in the three regions of the U.S. To use the dose-response relationships incorporated into the model, the change in UV flux (in percent) associated with changes in ozone abundance is used -- the change in the absolute level of UV energy reaching the earth's surface (in units such as millijoules per meter squared) is not used. (The method used to estimate changes in UV flux (in percent) along several scales of UV measurement was described above.)

Given these estimates of percent changes in UV over time for given locations in the U.S. (i.e., the three regions), the change in exposure for individuals estimated to live in those areas is estimated on an age specific basis. In year T of the analysis, the change in UV flux for a person who is 40 years old is a function of the change in UV estimated from years T-40 to year T. Changes in UV for years prior to 1985 are assumed to be zero.

The current understanding for non-melanoma skin cancer is that the cumulative exposure received during a person's life is the appropriate measure to use for modeling the dose-response relationship. Insufficient evidence exists to define the appropriate measure of exposure for purposes of modeling cataract prevalence. Similarly, a major question remains regarding the most appropriate measure of exposure for purposes of modeling the incidence and mortality of melanoma skin cancer. Recent analyses of melanoma incidence by Scotto and Fears (in press) have used annual RB-meter values as estimates of exposure. Pitcher (1986) used peak values to model mortality. Although some evidence indicates that peak or intermittent exposures to UV may best explain melanoma incidence patterns (see Chapter 8), significant research remains to be done on this issue. Of note is that the UV Model indicates that changes in UV flux (in percent) as a function of changes in ozone levels are about the same for the annual values and peak values for a given action spectrum. However, the choice of peak UV flux versus annual UV flux (or flux measured over some other time period) influences the estimate of the relationship between mortality risks and UV significantly (by as much as 60 percent). Therefore, the choice of annual or peak UV flux as an exposure estimate for purposes of modeling risk influences the risk estimates substantially.

A related issue regarding modeling exposure for purposes of estimating melanoma risks is the relative weight to put on exposures (annual or peak) experienced throughout a person's life. Migration studies indicate that exposure before the age of 15 may be most important and should be given more weight than subsequent exposures (see Chapter 8). These relative weights have yet to be quantified, however. To allow alternative assumptions regarding the relative importance of exposure during a person's life to be modeled, relative

weights can be assigned to different ages during a person's life for purposes of estimating changes in exposure. Exhibit F-15 presents a simplified version of the tables that the user can use to define these weights.

As shown in the exhibit, for a person who is currently 40 years old, the relative weight to give to the exposure that a person of that age has received during his life is: 2.0 times the exposure received from ages zero to 10, 2.0 times the exposure received from ages 10 to 20; 1.0 times the exposure received from ages 20 to 30; and 1.0 times the exposure received from ages 30 to 40. With these assumptions, the childhood exposures of individuals that are currently 40 years old are weighted twice as heavily as subsequent exposures. The assumptions used in the model for both melanoma skin cancers and cataracts are equal weights across all ages. The user may modify these assumptions as desired to evaluate alternative formulations.

The dose-respose relationships currently included in the model are called "power functions" and are generally of the form:

$$\ln(\text{incidence}) = a + b * \ln(E),$$

where:

incidence = the age specific incidence for a given race and sex;

a = a constant that varies by age, race, and sex;

b = a constant that varies by age, race and sex; and

E = measure of lifetime exposure to ultraviolet radiation.

Using this equation, the fractional change in incidence as a function of the fractional change in UV can be expressed as:

$$\text{fractional change in incidence} = \left(\frac{\text{fraction change in exposure}}{\text{exposure}} + 1\right)^{b} - 1$$

The value of this formulation is that:

- the change in incidence is expressed as a fractional change that can be multiplied by the baseline incidence to compute the increased age specific incidence; and
- the change in exposure can be expressed as a fractional change, thereby avoiding potential difficulties in specifying the absolute levels of changes in exposure to UV (see Serafino and Frederick (1986) for a description of these potential difficulties).

To specify the dose-response relationship, the user must choose a measure of UV (e.g., RB-Meter, DNA, or Erythema action spectrum) and provide estimates of b in the above equations. Scotto, Fears, and Fraumeni (1981) present a

EXHIBIT F-15

Sample Table for Specifying Relative Weights for Exposure During a Person's Lifetime

Current Age (years)	Age During Exposure (years)					
	0-10	10-20	20-30	30-40	40-50	50-60
10	2.0	-	-	-	-	-
20	2.0	2.0	-	-	-	-
30	2.0	2.0	1.0	-	-	-
40	2.0	2.0	1.0	1.0	-	-
50	2.0	2.0	1.0	1.0	1.0	-
60	2.0	2.0	1.0	1.0	1.0	1.0

All values are illustrative.

range of estimates of b for white males and females for use with the RB-Meter action spectrum.[9] Their analysis indicates that these coefficients do not vary by age, indicating that the incidence at each age is correlated to changes in exposure to UV in the same manner.

As described in Chapter 7, an analysis of the data in Scotto, Fears, and Fraumeni (1981) was performed using the DNA action spectrum. The estimated coefficients did not differ significantly from the coefficients reported by Scotto, Fears and Fraumeni for the RB-Meter action spectrum. This result is expected because at current ozone levels the RB-Meter and DNA UV flux values are highly correlated. Exhibit F-16 displays the estimated coefficients for the incidence of basal and squamous cell non-melanoma skin cancer used with the DNA action spectrum. Recent experimental data with mice indicate that an action spectrum that weights the short-wavelength UV-B use more heavily than the DNA action spectrum may be preferred; such as the Erythema Action Spectrum (see Cole, Forbes, and Davies 1986). Although the risk estimates in Chapter 18 are based on the DNA Action Spectrum, work is ongoing to evaluate the implications of using the Erythema Action Spectrum in its place.

The coefficients for the dose-response relationships for each of the types of melanoma were derived from data presented in Scotto and Fears (in press).[10] These coefficients vary by type of melanoma, and sex. As with the coefficients for non-melanoma, the values do not vary by age, and are for whites only. These values are also presented in Exhibit F-16. These coefficients are assumed to be applicable for the DNA action spectrum.

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[9] The coefficients for non-melanoma were derived from the regression coefficients presented in Scotto, Fears, and Fraumeni (1981, p. 10). The values reported were for an exponential formulation of the risk model. To translate the coefficients into the power formulation, the coefficients were multiplied by 135 UV units, the ambient UV exposure level at which the power function and exponential function models predict equal incidence results.

[10] The information reported in Scotto (in press) reported the percent increase in the incidence in each of the different types of melanoma for a 10 percent increase in UV. To translate these data into coefficients in the power formulation of the risk model, the power function was rearranged to solve for the coefficient (b) as follows:

$$b = \frac{\ln\left(1 + \frac{\text{Change in incidence}}{\text{incidence}}\right)}{\ln\left(1 + \frac{\text{Change in UV}}{\text{UV}}\right)}$$

For example, an 8 percent increase in incidence resulting from a 10 percent increase in UV yields the following coefficient: $\ln(1.08)/\ln(1.1) = 0.807$.

EXHIBIT F-16

Coefficients Relating Percent Change in UV to Percent Change in Incidence

(For use with the DNA Action Spectrum -- Whites only)

	a/ Low	Middle	b/ High
NON-MELANOMA SKIN CANCER			
Squamous			
Male	1.42	2.03	2.64
Female	1.47	2.22	2.98
Basal			
Male	0.932	1.29	1.65
Female	0.316	0.739	1.16
MELANOMA SKIN CANCER			
Face, Head and Neck			
Male	0.661	0.846	1.029
Female	0.798	1.019	1.236
Trunk and Lower Extremities			
Male	0.421	0.651	0.875
Female	0.341	0.522	0.700

a/ Middle value minus one standard error.

b/ Middle value plus one standard error.

Sources: Melanoma coefficients derived from Scotto and Fears (in press). Non-melanoma coefficients presented in Chapter 7.

A dose-response relationship for non-melanoma mortality has not been estimated. For purposes of modeling, the relationships for non-melanoma incidence (basal and squamous) are assumed to apply.

A separate analysis of melanoma mortality has been performed by Pitcher (1986). The coefficients estimated as the result of that investigation are used to model the potential increase in mortality due to melanoma skin cancer. The coefficients estimated using peak UV flux values (weighted using the DNA action spectrum) as the measure of exposure are reported in Exhibit F-17. Analysis is continuing on alternative measures of exposure. Preliminary results indicate that using annual UV flux as the exposure measure would result in coefficients that are as much as 60 percent smaller.

Coefficients for dose-response relationships for Blacks and members of other races have not been developed. Skin cancer risks for blacks and members of other races are not modeled.

The correlation between UV-B and cataract prevalence estimated by Hiller, Sperduto and Ederer (1983) is used to model the potential changes in the incidence of cataracts as a function of changes in UV-exposure. The risk due to UV in the multivariate logistic risk equation developed by Hiller, Sperduto and Ederer varies with age (see Chapter 10). For purposes of modeling, a simplified risk equation that is age-invariant was developed. A single coefficient was estimated by weighting the UV risk across the age groups by the current estimate of increased prevalence due to UV. The resulting coefficient (which is used in the "power formulation") provides an approximation of the expected increased risk across all age groups. The estimated coefficients are displayed in Exhibit F-18. As shown in the Exhibit, the same coefficients are used for all races and males and females.

To compute increased incidence of senile cataracts, the changes in prevalance over time are computed. Incidence in a given year is equal to change in prevalence (relative to the previous year), plus the number of cases (counted in the previous time year) that were associated with people who have died since last year. It is assumed that the prevalence of cataracts is the same among people who die or survive from one year to the next.

Step 4 -- Summarize the Results

The dose-response relationships are applied to each of the age groups of the population over time using estimated changes in UV exposure as outlined above. The results of the method include:

- changes in the total population incidence rates (e.g., per 100,000 people) for a standard population age, sex, and race distribution;

- the total number of cases of each type of cancer over time, and the change in the number of cases from the number that would exist in the absence of changes in UV flux associated with changes on total column ozone; and

EXHIBIT F-17

Coefficients Relating Percent Change in UV to Percent Change in Melanoma Mortality (For Use with DNA Action Spectrum -- Whites Only)

	Low [a/]	Middle	High [b/]
Males	0.78	0.85	0.92
Females	0.50	0.58	0.66

a/ Middle estimate minus one standard error.

b/ Middle estimate plus one standard error.

Source: Pitcher (1986).

EXHIBIT F-18

Coefficients Relating Percent Change in Senile Cataract Prevalence for a One Percent Change in UV (all races)

	Low [a/]	Middle	High [b/]
Males and Females	0.171	0.311	0.419

a/ Middle estimate minus one standard error.

b/ Middle estimate plus one standard error.

Source: Derived from Hiller, R., R. Sperduto, and F. Ederer (1983), "Epidemiologic Associations with Cataract in the 1971-1972 National Health and Nutrition Examination Survey," American Journal of Epidemiology, Vol. 118, No. 2, pp. 239-248.

- the total number of deaths due to each type of cancer over time, and the change in the number of deaths from the number that would exist in the absence of changes in UV flux associated with changes on total column ozone.

These results are reported for the U.S. as a whole (the calculations are performed separately for each of the three U.S. regions). The increased number of cases and deaths are reported separately for those individuals that are alive today and for those individuals who are expected to be born in the future.

DEGRADATION OF POLYMERS

The purpose of this portion of the module is to evaluate the potential damages and costs associated with polymer degradation that may be caused by increases in the flux of ultraviolet radiation (UV) reaching the earth's surface. As described in Chapter 13, UV can degrade the physical and appearance characteristics of polymers. This module evaluates the economic impacts that increased UV flux may have on polymer performance based on studies by Andrady (1986) and Horst (1986).

There are several types of polymers that may be adversely affected by increases in UV, including: polyvinylchloride (PVC); acrylics; polycarbonate; polypropylene; and polyester (Andrady 1986, p. 22). To date, data for assessing the potential future markets and potential damages due to UV have only been developed for the portion of the PVC market used in construction (siding, window profiles, rainwater systems, and pipe and conduit). This PVC market accounts for about 26 percent of all polymers subject to exposure to UV (measured by production volume), and consequently the estimates from this module are underestimates of the potential risks of polymer degradation due to ozone depletion.

Recognizing that the current data only account for a portion of the polymer market, the module is designed to evaluate up to five different types of polymers using the method outlined for PVC. As data for other polymers become available, it will be integrated into the module as appropriate.

The effects of UV on polymers are evaluated in three steps:

1. Project the size of the market for polymers that may be subject to degradation due to UV.

2. Assess the damage that increases in UV (due to ozone depletion) may have on the polymers.

3. Assess the costs of the damage to the polymers.

Each step is described in turn for evaluating PVC in construction applications.

Step 1 -- Market for Polymers

The future market for PVC polymers in construction will be influenced by construction activity and the costs and performance of substitute materials. Horst (1986) developed several approaches for projecting the expected production and use of PVC through 2075. The approach used here reflects expected market saturation and is of the following form:

$$Q = A \left(1 - \exp \left(-k(T - T_o)\right)\right)$$

where:

Q = quantity per person per year in pounds;
A = a constant;
k = a constant;
T = the current year of the projection; and
T_o = a base year identified for the polymer type.

The constants and base year computed for PVC by Horst (1986, p. 4A-3) using statistical analyses of historical data are as follows: A = 63.0308; k = 0.01265; and T_o = 1966. These values are used along with the U.S. population scenario (see Appendix B) to compute a middle estimate of the size of the future market for PVC in pounds. By using the population scenario from the scenarios module, the estimates of the PVC market will be consistent with the values used to develop the scenarios of production of CFCs and the other trace gases, and the evaluation of human health effects.

Because there is uncertainty in the expected future market for PVC, the module also produces low and high estimates of future demand. The low and high estimates are based on the statistical uncertainty in the estimates of A and k. By approximating the future demand, Q, with the first three terms of its expansion series, Horst (1986) showed that he variance of Q could be estimated from the covariance matrix of A and k. Specifically,

$$\begin{aligned} \mathrm{Var}\,(Q) \approx\ & \mathrm{Var}(Ak)\ (T-T_o)^2 . \\ & - \mathrm{Covar}(Ak, Ak^2)\ (T-T_o)^3 + \\ & \mathrm{Var}(Ak^2/4)\ (T-T_o)^4 . \end{aligned}$$

The model computes the variance of demand in each year. The low estimate is set at one standard deviation below the middle estimate; the high estimate is set at one standard deviation above. These low and high values (along with the middle value described above) are used in step 3 to evaluate the costs associated with the degradation of these polymers.

Step 2 -- Polymer Damage

A damage index that indicates the level of degradation of polymer characteristics was developed by Andrady (1986). When there is no degradation, the index value equals 1.0. Degradation is indicated by increasing values of the index, such as 1.1. This index is used here to

describe the potential damage to polymers due to increased UV flux associated with ozone depletion.

Andrady (1986) also developed an estimate of how the damage index may vary as a function of ozone depletion. Due to uncertainties in the relationship between ozone depletion and UV, and UV and polymer characteristics, the relationship between ozone depletion and the damage index is represented as a range. These ranges are displayed in Exhibit F-19.

To evaluate the damage index in each year, the ozone depletion estimate for that year is used get a range of values in the damage index table. The ozone depletion estimate for the U.S. is taken as the estimate for the middle U.S. region. The result of this method is a range of damage index values for each year (low, middle, and high).

Step 3 -- Assess Damage Costs

The costs of polymer degradation depend on the manner in which the characteristics of the polymers degrade, and the steps that are taken in response to the degradation. Horst (1986) developed an approach for evaluating the costs associated with the production of new PVC each year. These costs represent the implications of changing the formulation of PVC during manufacture in order to maintain its characteristics in light of increased UV exposure. These costs do not include the potential damages to PVC already in place. Additionally, these estimates do not reflect the losses that may be associated with polymer degradation in the absence of changes in the polymer formulation. Therefore, these estimates are an underestimate.

Horst (1986) identified the costs of changing the formulation of PVC as a function of the increased amount of stabilizer that needs to be added to the polymer to maintain its characteristics. A 25 percent increase in stabilizer was estimated to lead to a 1.86 percent increase in the price of PVC, from its current cost of $0.604 per pound (Horst 1986, p. 5-16 and 6-8). The increased amount of stabilizer required as a function of ozone depletion was identified by Andrady (1986) and is presented in Exhibit F-19.

To estimate the costs of the increasing the stabilizer in response to ozone depletion, Horst recommends using a ten year lag between the increase in stabilizer required and the increase in the price of PVC. This lag reflects the time needed for the industry to respond to changing environmental conditions and the fact that the current stabilizer concentrations include a margin of safety. This ten year lag is used in the module, although the model user may override it with an alternative assumption.

To compute the costs in year T, the following computations are performed:

- compute the increased amount of stabilizer required, based on the ozone depletion in year T - 10;
- compute the increase in price by interpolating between no increase in price, and a 1.86 percent increase in price for a 25 percent increase in stabilizer;

EXHIBIT F-19

Damage Index and Increase in Stabilizer For Ranges of Ozone Depletion

Ozone Depletion (percent)	Damage Index			Stabilizer Increase (%)		
	Low	Middle	High	Low	Middle	High
0-5	1.01	1.015	1.02	1.0	3.0	5.0
5-10	1.01	1.025	1.04	1.0	5.0	9.0
10-15	1.02	1.055	1.09	3.0	11.5	20.0
15-20	1.03	1.105	1.18	3.0	20.5	38.0

Source: Derived from Horst (1986), p, 6-10.

- given the increase in price, compute the cost as follows:

$$C(T) = (D(T)/P_o^{\,b})/(1+b) * [P_o^{\,(1+b)} - P_1^{\,(1+b)}]$$

where:

C(T) = cost in year T;

D(T) = demand in year T;

P_o = price of PVC in the absence of changes in the formulation of the polymer;

b = price elasticity of demand for PVC;

P_1 = the new price for PVC given the change in the formulation of the polymer.

This equation represents the annual loss in consumer surplus associated with the the estimated changes in price. The price elasticity of demand estimated by Horst (1986) is -1.956. This value represents (in part) the estimated availability of appropriate substitutes for PVC.

This equation can be evaluated three times each year to generate low, medium, and high estimates of the costs. The low estimate uses the low demand value (from step 1) and the estimate of the change in price associated with the low estimate of the increase in stabilizer associated with ozone depletion. The middle and high estimates use the middle and high values for these components respectively. If there is an increase in ozone abundance, the damage index is set to 1.0 and there are no estimated costs. The maximum price increase (1.86 percent) estimated by Horst is associated with a 25 percent increase in stabilizer. Price increases are capped at this value (1.86 percent) even when an increase in stabilizer exceeding 25 percent may be indicated as being required. The present value of the costs over time can be evaluated by discounting these annual costs at user-supplied discount rates.

F.6 LIMITATIONS

The Effects Module draws together a diverse set of analyses and applies these analyses to estimates of global ozone depletion (from the Atmospheric Science Module) to evaluate potential risks to human health. The analyses require that a chain of events from ozone depletion to health outcomes be modeled. There are limitations in each link of this chain, creating uncertainties in the estimates of the outcomes.

First global ozone depletion is translated into latitudinal values. The current algorithm used for these latitudinal estimates is based on a limited set of data from a single two-dimensional (2-D) model. Additional 2-D analyses should be performed.

The estimates of changes in UV as a function of changes in the ozone level are based on a model. The model is in its early stages of field validation, and additional analyses remain to be performed.

The dose-response relationships for skin cancers and cataracts all depend on epidemiologic studies of humans in the U.S. Because the studies are based on humans in the U.S., there are no extrapolation problems (e.g., from animals to humans, or from one population of humans to the U.S. population). However, the epidemiologic studies are generally limited by their measures of environmental exposure to UV, as opposed to individual lifetime exposures. In addition to the limitations of the estimates of the dose-response relationship themselves, the characterization of baseline evidence (in the absence of ozone depletion) and baseline population characteristics are subject to uncertainty.

In addition to uncertainties and limitations in the estimates produced by the module, the module is limited because there are a variety of important effects that are not evaluated. These omitted effects include:

- polymers other than PVC;
- serious (non-fatal) cases of non-melanoma skin cancers;
- impacts on plants;
- impacts on aquatic organics;
- impacts on urban smog formation;
- potential immune suppression in humans; and
- possible other diseases.

The omission of these effects biases the estimates downward. In Chapter 18, qualitative estimates of some of these effects are presented based on interpolations and extrapolations from case studies and research in early stages. Data used to prepare these estimates include:

- Imacts on soybeans. For depletion of 25 percent, a 19 percent reduction in yield is used (average of results reported in Chapter 11 for Essex cultivar). For depletion between zero and 25 percent, a value is interpolated from between zero and 19 percent. Values are not extrapolated to outside this range.

- Impacts on Ground-Based Ozone (Smog). Estimates of increased smog levels are interpolated from case study estimates (Chapter 14) as follows:

  -- Los Angeles: 16.6% depletion yields a 4.5 percent increase;
  33.3% depletion yields a 9.4 percent increase;

-- Philadelphia: 16.6% depletion yields a 13.4 percent increase;
33.3% depletion yields a 33.0 percent increase;

-- Nashville: 16.6% depletion yields a 23.8 percent increase; and
33.3% depletion yields a 50.0 percent increase.

- Impacts on Aquatic Organisms. Estimates for reduced anchovy populations are interpolated from laboratory tank experiments (described in Chapter 12) as follows:

Percent Change in UV-B	Percent Annual Population Death
0%	0%
10%	0%
20%	0% to 4.8%
30%	0% to 11.5%
40%	2.5% to 18.0%
50%	6.0% to 23.0%
60%	11.0% to 25.0%

Because these data are based on case studies and research in early stages, all the estimates of these effects are very preliminary, and should be viewed with caution.

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CHAPTER 18

HUMAN HEALTH AND ENVIRONMENTAL EFFECTS

SUMMARY

If no actions are taken to regulate future emissions of ozone-modifying substances and greenhouse gases, damage to human health and the environment is likely to occur. The purpose of this chapter is to present a range of quantitative estimates of the potential risks to human health and the environment arising from emissions of ozone-modifying substances.

The risk estimates draw on the data and methods described in earlier chapters. These chapters describe: (1) potential future emissions and concentrations of ozone-modifying substances; (2) the effects that these substances may have on stratospheric ozone and global climate; (3) the potential damage to human health and the environment arising from ozone modification and climate change; and (4) a comprehensive modeling framework for estimating these risks.

The major types of effects for which quantitative estimates are provided include ozone depletion; additional cases and deaths related to skin cancers; additional cases of senile cataract; damage to polyvinyl chloride materials; rise in global equilibrium temperature; sea level rise; cost of sea level rise; reduction in soybean yield; increases in ground-based ozone; and effects on the survivability of a selected aquatic organism. Certain important risks discussed in earlier chapters are not evaluated because of data limitations and the absence of relevant scientific information. In most instances, the estimates cover the U.S. only.

Estimates of the potential risks due to emissions of ozone-modifying substances are necessarily uncertain for two reasons. First, estimating future risks requires projections of key factors, such as population and economic growth. Second, the many relationships that define how emissions result in risks are themselves uncertain.

To investigate the significance of key assumptions and data used to model risks, the chapter presents a range of risk estimates varying the most important assumptions, one at a time. The range of results obtained by varying each assumption is used to determine which factors used to model risks are most important.

FINDINGS

1. FOR THE MOST LIKELY ESTIMATES OF CFC GROWTH, ATMOSPHERIC MODIFICATION AND DOSE-RESPONSE, AN INCREASE IN SKIN CANCER CASES AND DEATHS, CATARACT CASES, MATERIALS DAMAGE, GLOBAL TEMPERATURE AND SEA LEVEL IS EXPECTED.

   1a. An additional 40 million skin cancer cases and 800 thousand deaths from skin cancer are projected for people alive today and born in the next 88 years, along with 12 million cases of cataracts (U.S. only).

   1b. The earth would be committed to global warming from 3.0°C to 9.5°C by 2075, sea level rise would be from 80 cm to 94 cm.

2. FOR GASES THAT COUNTER DEPLETION, SOME ASSUMPTION IS NEEDED ABOUT FUTURE LIMITS TO GREENHOUSE WARMING. THE STANDARD ASSUMPTION HAS BEEN THAT NO LIMIT WILL EVER BE PLACED ON GLOBAL TEMPERATURE INCREASES. ESTIMATES OF THE RISK OF OZONE DEPLETION FROM CFC AND HALON GROWTH ARE HIGHLY SENSITIVE TO ASSUMPTIONS ABOUT GREENHOUSE WARMING. IF GLOBAL WARMING IS EVENTUALLY LIMITED, THE RISK OF COLUMN OZONE DEPLETION FROM CFCS AND HALONS WILL RISE.

   2a. A limit of global warming of 3°C would more than double the risks of CFC and Halon growth.

   2b. A more stringent global warming limit would have even greater effect.

   2c. Model results are sensitive to the rate of methane growth, which is highly uncertain even without regard to greenhouse warming considerations.

3. ALTERNATIVE ESTIMATES OF CFC GROWTH PRODUCE DIFFERENT LEVELS OF RISKS.

   3a. The low case of 1.2 percent CFC growth (about half of the 2.5 percent central case with greenouse gases that counter depletion growing) would lower the estimates of increased risks of skin cancer cases and deaths and cataracts cases by more than 90 percent.

   3b. The high case of 3.8 percent CFC growth (with greenhouse gases that counter depletion growing) would increase the estimates of increased risks of skin cancer cases and deaths and cataracts cases by over 400 percent.

   3c. Lowering growth rates to zero (with greenhouse gases that counter depletion growing) would have the effect of reducing skin cancer cases and deaths by more than an additional 10 percent, and cataracts by a similar amount.

   3d. The highest growth rate, which is unrealistically high for the whole period, would add a tremendous number of cases and deaths.

4. RISK ESTIMATES ARE SENSITIVE TO UNCERTAINTIES ABOUT ATMOSPHERIC RESPONSE AND DOSE-RESPONSE.

   4a. Uncertainty about atmospheric response introduces a greater uncertainty than uncertainty about dose-response parameters for skin cancers or cataracts. Both uncertainties affect the risk estimates by more than 35 percent.

   4b. Preliminary results of ongoing analyses of melanoma mortality risks indicate that significant uncertainties remain in the specification of the dose-response relationship. The magnitude of this specification uncertainty is still being identified, and as a result the full implications of this uncertainty cannot be quantified at this time.

5. WHILE NATIONAL QUANTITATIVE ESTIMATES OF AQUATIC, CROP, GROUND-BASED OZONE, AND SEA LEVEL RISE DAMAGE CANNOT BE MADE AT THIS TIME, CASE STUDY RESULTS INDICATE THAT SIGNIFICANT INCREASES IN GROUND-BASED OZONE, LOSS OF AQUATIC LIFE, SEA LEVEL RISE DAMAGE AND LOSS OF CROP YIELD ARE POSSIBLE.

INTRODUCTION

The previous chapters of this document have described: (1) potential future emissions and concentrations of ozone-modifying substances; (2) the effects that these emissions and concentrations may have on stratospheric ozone and global climate; (3) the potential risks to human health and the environment arising from ozone-modification and global climate change; and (4) a comprehensive modeling framework for estimating these risks. This chapter integrates the data and methods presented in earlier chapters to develop quantitative estimates of risks for human health and the environment due to ozone modification and climate change.

The risk estimates are based on the assumption that no action is taken to regulate CFCs or other ozone-depleting substances. However, the data and analyses used here could be applied to alternative regulatory strategies. Additionally, all of the effects, with the exceptions of global sea level rise, global ozone depletion, and change in global equilibrium temperature, are estimated for the U.S. only.[1]

The following ozone-depleting substances are considered in estimating risks:[2]

- CFC-11, 12, 22, and 113;
- methyl chloroform (CH3CCl3);
- carbon tetrachloride (CCL4); and
- Halon-1211 and 1301.

The following trace gases that add ozone or counter ozone depletion (when chlorine concentrations rise) are considered:[3]

- carbon dioxide (CO2);
- methane (CH4); and
- nitrous oxide (N2O).

Exhibit 18-1 displays the types of effects that are estimated in this chapter along with the chapter that describes the data and methods used to derive the effects. The effects are divided into two sets: (1) effects that can be estimated quantitatively for the U.S.; and (2) effects estimated on the basis of case studies and research in early stages. The first set is

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[1] The estimates presented in this chapter are for the entire U.S. As described in Chapter 17, the U.S. estimates combine analyses of three geographic regions (by latitude): north, middle, and south.

[2] See Chapter 3 for a description of the scenarios of future emissions used.

[3] See Chapter 4 for a discussion of the future concentrations of these greenhouse gases. These greenhouse gases also counter ozone depletion, at least in high chlorine cases.

EXHIBIT 18-1

Types of Human Health and Environmental Effects Estimated

TYPE OF EFFECT (Reference Chapter)	UNITS
Effects Estimated Quantitatively for the U.S.	
Ozone Depletion (Chapter 5)	Percentage Depletion from 1985 to 2075
Non-melanoma Skin Cancer (Chapter 7)	Additional Cases and Deaths for People Alive Today; People Born 1985-2029; and People Born 2030-2074.
Melanoma Skin Cancer (Chapter 8)	Additional Cases and Deaths for People Alive Today; People Born 1985-2029; and People Born 2030-2074.
Senile Cataracts (Chapter 10)	Additional Cases for People Alive Today; People Born 1985-2029; and People Born 2030-2074.
Damage to Polyvinyl Chloride Materials (Chapter 13)	Present Value Costs in 1985 U.S. Dollars
Rise in Equilibrium Temperature from 1980 to 2075 (Chapter 6)	Degrees Centigrade
Sea Level Rise from 1980 to 2075 (Chapter 15)	Centimeters

EXHIBIT 18-1
(Continued)

Types of Human Health and Environmental Effects Estimated

TYPE OF EFFECT (Reference Chapter)	UNITS
Effects Based on Case Studies and Research in Early Stages:	
Reduction in Soybean Yield (Chapter 11)	Percent in 2075
Increase in Fatality Rate of Northern Anchovy (Chapter 12)	Percent in 2075
Increase in Ground-Based Ozone (Chapter 14)	Percent in 2075
Cost of Sea Level Rise in Charleston, SC and Galveston, TX (Chapter 15)	Present Value Costs in 1985 U.S. Dollars

estimated using detailed epidemiologic, economic, demographic, and other scientific information within the modeling framework described in Chapter 17. Effects estimated in this first set are representative of risks to the entire U.S., and include:

- global average ozone depletion and associated ozone depletion by latitude;[4]
- non-melanoma skin cancer (cases and deaths);
- melanoma skin cancer (cases and deaths);
- senile cataracts (cases);
- damage to polyvinyl chloride (PVC) materials;
- increased global average temperature; and
- global sea level rise.

The second set of estimates is based either on research in early stages or on a small number of case studies the results of which cannot be generalized to the entire U.S. Effects estimates included in the second set should be considered preliminary and are included here only to indicate the potential order of magnitude of these effects. The second set of estimates includes the following types of effects:

- costs of sea level rise;
- reductions in soybean yields (for one cultivar; 2 out of 3 cultivars appear sensitive);
- increases in ground-based ozone (smog); and
- effects on the survivability of a selected aquatic organisms (the Northern Anchovy).

Because of data limitations and the absence of relevant scientific information, certain important effects discussed in earlier chapters were not evaluated. The major effects not analyzed in this chapter are:

- overall impacts on agricultural and natural plants (Chapter 11);

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[4] Estimates of ozone depletion are generated using a one-dimensional model of the atmosphere that is modified to emulate the results of a two-dimensional model. As discussed below, the ozone depletion estimates would be higher if estimated using a two-dimensional model.

- impacts of climate change on human health, water resources, forestry, and wetlands (Chapter 16);
- increased incidence of cutaneous diseases from immune suppression (Chapter 9);
- impacts of sea level rise on wetlands (Chapter 15); and
- impacts on materials other than polyvinyl chloride (Chapter 13).

Estimates of human health and environmental effects are necessarily uncertain for two reasons. First, effects are estimated into the future, requiring projections of such factors as population growth; economic growth; and production, use, and emissions of ozone-modifying substances and greenhouse gases. Second, the linkages from emissions to risks are themselves uncertain. For example, the models and analyses used to evaluate ozone modification and global warming continue to be developed.

Critical to understanding the risks of ozone depletion and climate change is a consideration of the significance of uncertainties in the numerous factors and assumptions used to model the chain of events that starts with emissions and results in risks. Therefore, this chapter starts with "central case" estimates of risk that are based on the most likely estimate of each factor. Following the presentation of the central case, the sensitivity of the risk estimates to the factors linking emissions to risks are explored systematically across the most important factors.

The remainder of this chapter is organized as follows:

- Methods for Estimating Health and Environmental Risks summarizes the major steps used to estimate risks.
- Health and Environmental Risks: Central Case presents estimates of risks associated with the most likely set of assumptions and parameters, the "central case."
- Comparison of Central Case with Results Using Alternative Assumptions explores the sensitivity of the risk estimates to assumptions about: (1) emissions of ozone-modifying gases; (2) the ozone-depletion model used; (3) sensitivity of global warming to concentrations of greenhouse gases; and (4) future concentrations of greenhouse gases.
- Sensitivity of Effects to Parameter Uncertainty identifies how the risk estimates are influenced by statistical uncertainty in estimates of: (1) dose-response relationships; and (2) ozone level response to emissions.

- Relative Importance of Key Uncertainties summarizes the results of previous sections and compares the significance of the different uncertainties analyzed in this chapter.

METHODS FOR ESTIMATING HEALTH AND ENVIRONMENTAL RISKS

Emissions of ozone-modifying substances and greenhouse gases lead to effects on human health and the environment by means of the following principal mechanisms:

1. Emissions and ozone depletion. Emissions of ozone-modifying substances deplete total column ozone.

2. Ozone depletion and ultraviolet radiation. Depletion of total column ozone increases the amount of ultraviolet radiation (UV) reaching the earth's surface.

3. Ultraviolet radiation and health effects. Melanoma skin cancer, non-melanoma skin cancer, and cataracts are related to increases in UV. Changes in the expected number of cases and/or deaths associated with increases in UV are described by dose-response relationships.

4. Emissions and global warming. Emissions of greenhouse gases cause the earth's equilibrium temperature to rise.

5. Global warming and human health, environmental, and other effects. Changes in the global equilibrium temperature affect the sea level, human health, aquatic organisms, agricultural yields, energy demand and other factors.

To quantify risks for human health and the environment, each of these mechanisms was evaluated using mathematical relationships. For example, estimates of additional cases of melanoma skin cancers rely on mathematical relationships between: (1) emissions and ozone depletion, (2) ozone depletion and UV, and (3) UV and cancer incidence. The sources and derivation of the mathematical relationships are described in earlier chapters.

The mathematical relationships were integrated in the modeling framework described in Chapter 17. The framework provides a method of estimating expected risks as well as analyzing the joint implications of key assumptions and uncertainties in each mathematical relationship. The primary period of analysis used in the models is 1985 to 2075. Emissions of ozone-modifying substances and concentrations of greenhouse gases are analyzed during this period. Risks to human health are evaluated for individuals born before 2075, in three discrete cohorts (i.e., groups):

- those individuals alive today;

- those individuals born between now and 2029; and

- those individuals born between 2030 and 2074 (the end of the analysis period).

Risk estimates for these individuals are based on simulated lifetime exposures to increased UV. For example, a 50 year old person who is alive today is assumed to experience no increase in UV prior to 1986. During his remaining life, however, this 50 year old may experience increased exposure to UV as total column ozone depletes. Exposure histories for each population cohort are analyzed separately as a basis for estimating risks.

The key exposure variable for non-melanoma skin cancer can be approximated using cumulative lifetime exposure (see Chapter 7). For melanoma skin cancers, a major question remains regarding the appropriate measure of exposure for purposes of modeling risks (see Chapter 8 and Chapter 17). Evidence suggests that peak or intermittent exposures may be preferred indicators of risk. Additionally, exposures to a person prior to the age of 15 may be more important than subsequent exposures. Insufficient evidence is available to define conclusively the preferred exposure measure, and work is continuing in this area. Similar uncertainty exists regarding the preferred exposure measure for modeling potential cataract risks (see Chapter 10). Also, the quantification of the relationship between ultraviolet radiation and cataracts is in the early stages. The risk estimates presented below for non-melanoma (incidence and mortality) and melanoma (incidence) and cataracts are based on cumulative lifetime exposure. Risk estimates for melanoma mortality are based on an equation that relates peak UV flux (as a measure of exposure) to mortality rates. Although work is continuing in this area, preliminary results indicate that this relationship (based on peak UV flux) may result in larger risk estimates than would equations based on exposures averaged over longer periods (e.g., annual UV flux). All the risk estimates presented below assume constant baseline incidence and mortality rates; recent secular trends in these rates are not extrapolated into the future.

Of note is that some of the individuals in the second and third cohorts (born after 1985) will live beyond the end of the primary period of analysis (2075). To estimate the lifetime risk for these individuals, the time horizon was extended to 2165 holding emissions and concentrations constant after 2075. Risk estimates for the second two population cohorts are affected by the assumption that emissions and concentrations are constant after 2075. In most of the scenarios investigated, depletion, UV radiation, and temperature are still increasing in 2075.

Non-health risks (e.g., global warming, sea level rise, and PVC damages) are reported for 2075. Risk estimates based on preliminary research and case studies are also reported for 2075.

It is important to note that the models used to estimate risks do not include feedbacks among factors such as an emissions growth rate (2.5 percent for CFC emissions, for example) and the consequences of that growth rate (6.0°C warming, for example). To the extent that there are feedbacks from effects to subsequent economic growth, that is, to the extent that effects may reduce economic growth, the system will in fact be self-limiting in the growth of CFCs. As now conceived, the risk models implicitly assume that the damage from effects is not large enough to alter the growth of society.

HEALTH AND ENVIRONMENTAL RISKS: CENTRAL CASE

This section presents central case estimates of human health and environmental risks due to ozone modification and climate change.[5] The central case assumes that no actions are taken to limit: (1) emissions of ozone modifyers; or (2) future global warming due to the greenhouse effect. The significance of these and other assumptions is investigated in later sections.

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[5] The central case estimates reflect the most likely values of key assumptions and inputs used to model risks:

- Annual production of CFC 11 and 12 grow at an annual average rate of 2.5 percent from 1985 to 2050, and remains constant following 2050; others as described in Chapter 3. The compounds analyzed include: CFC-11, CFC-12, CFC-22, CFC-113, methyl chloroform, carbon tetrachloride, Halon-1211, and Halon-1301. Emissions estimates reflect the storage of some substances in their end-use products for many years.

- Consensus estimates of the annual rates of increases in atmospheric concentrations of other trace gases are used: carbon dioxide (CO2) at 0.6 percent; methane (CH4) at 1.0 percent; and nitrous oxide (N2O) at 0.25 percent. Trace gas assumptions are discussed in Chapter 4.

- A parameterized relationship between emissions of ozone modifyers, trace gas concentrations and global ozone depletion is used that reflects the results of a one-dimensional model of the atmosphere using the most recent estimates of reaction rates. The parameterized atmospheric model (described in Chapter 17) represents the results of the Wuebbles Model (reported in Chapter 5).

- The latitudinal distribution of ozone depletion is evaluated using the results of a time-dependent two-dimensional model of the atmosphere. The latitudinal analysis of ozone depletion is presented in Chapter 17.

- The relationship between changes in ozone abundance and changes in UV flux reaching the earth's surface is based on best estimates of a radiation model of the atmosphere. The estimates of UV flux are described in Chapter 17.

- The risks to human health due to increases in UV are evaluated using middle estimates of dose-response coefficients developed in epidemiologic analyses in the U.S. The quantifiable risks to human health are described in Chapters 7, 8, and 10.

- The middle estimate by the National Academy of Sciences (NAS) for the sensitivity of the global climate to greenhouse gas forcings is used -- 3.0°C equilibrium warming for a doubling of the concentration of CO2. The National Academy of Sciences estimates are presented in Chapter 6. Recent analyses of climate sensitivity indicate that a 4°C sensitivity may be a preferred central case assumption (Manabe and Wetherald 1986; Washington and Meehl 1984; Hansen et al. 1984).

Exhibit 18-2 shows projections of average global ozone depletion for 1985 through 2100 for central case assumptions (see footnote 5). As shown in the exhibit, average global ozone levels are projected to have declined by about 2 percent by 2020, about 19 percent by 2075, and about 30 percent by 2100. The actual depletion will vary by latitude. In the northern hemisphere, depletion will be greater than average at latitudes near 40°N and above and lesser at latitudes below 40°N.

Of note is that the estimates show continued ozone depletion beyond 2050 when production is assumed to level off and remain constant. Because ozone-modifying substances are stored in products, emissions can lag production by many years. In the central case, emissions do not become constant until approximately 2070. Even after emissions level off in 2070, ozone depletion will continue as atmospheric concentrations of chlorine continue to increase.[6] For estimating health effects beyond 2100, the modeling assumption is that ozone levels are constant after 2100.

Estimates of human health risks in the U.S. are presented in Exhibit 18-3. Health risks were estimated as the additional number of cases and/or deaths related to skin cancer and senile cataracts for three population cohorts: (1) people alive today, (2) people expected to be born between today and 2029, and (3) people expected to be born from 2030 through 2074. People alive today can expect to experience the following additional estimated risks due to ozone depletion over their lifetimes: (1) approximately 630 thousand cases of basal cell skin cancer; (2) 390 thousand cases of squamous cell skin cancer; (3) 16 thousand deaths from both basal and squamous cell skin cancer; (4) 12 thousand cases of melanoma skin cancer; (5) 4 thousand deaths due to melanoma skin cancer; and (6) 600 thousand cases of senile cataracts. These estimates are of additional cases and deaths over and above what would have occurred in the absence of ozone depletion.

The number of additional cases and deaths due to ozone depletion is greater for the latter two population cohorts. Two factors account for increased risks in future generations. First, people alive today were assumed to experience increased exposure to UV only during the remainder of their lives. For example, the increase in cumulative lifetime exposure for a person who is 40 years old today results only from increases in UV flux during the remaining years of his life. People who are born after today (i.e., the latter two cohorts) experience increased exposure to UV during a larger fraction of their lives. Therefore, the impact of increases in UV flux over time on the incidence and mortality of these conditions is greater for people exposed for longer periods.

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[6] Concentrations rise even though emissions are constant because the atmosphere is out of equilibrium. The removal of CFCs and chlorine from the atmosphere is slow, meaning that it takes a long time for equilibrium (i.e., steady state) to be reached even though emissions are constant.

EXHIBIT 18-2

Global Average Ozone Depletion: Central Case

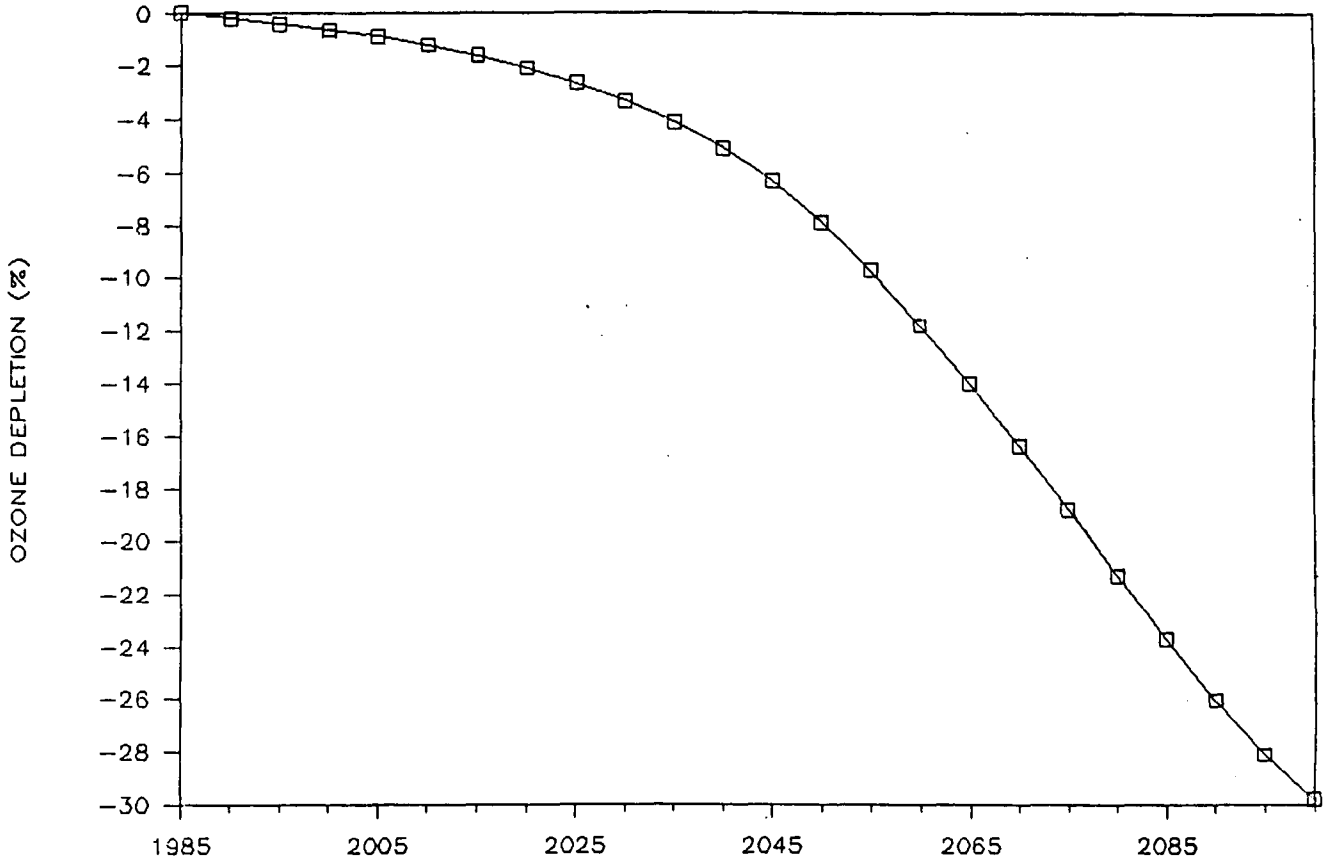

EXHIBIT 18-3

Human Health Effects: Central Case
(Additional Cumulative Cases and Deaths by Population Cohort)

HEALTH EFFECT	ALIVE TODAY[a]	BORN 1985-2029[b]	BORN 2030-2074[c]
Non-Melanoma Skin Tumors	2.5% growth (halving of current level)		
Additional Basal Cases	630,600	5,012,900	17,630,500
Additional Squmaous Cases	386,900	3,185,800	12,122,400
Additional Deaths	16,500	135,000	509,300
Melanoma Skin Tumors			
Additional Cases	12,300	109,800	430,500
Additional Deaths	3,900	32,200	115,100
Senile Cataract			
Additional Cases	593,600	3,463,400	8,295,800

[a] Analysis period for health effects: 1985-2074.

[b] Analysis period for health effects: 1985-2118.

[c] Analysis period for health effects: 2030-2164.

Second, ozone depletion, and hence exposure to UV radiation, is simulated to increase over time (except after 2100 when ozone depletion -- and consequently UV flux -- is held constant). As a result, persons in the later two cohorts are exposed to higher levels of UV flux.

Exhibit 18-4 presents estimates of two groups of non-health effects (1) estimated quantitatively for the U.S. -- including effects on PVC materials, global equilibrium temperature, and sea level; and (2) based on case studies and research in early stages -- including impacts on agricultural output, aquatic life, and ground-based ozone (smog). Each effect is as follows:

- PVC materials damage represents the present value of costs of producing PVC materials in a manner that will prevent damages due to increased UV flux. Damages are estimated at $550 million (1985 U.S. dollars) using a real discount rate of 3 percent. Estimates of PVC damage were derived from case studies described in Chapter 13. The damage estimate represents the additional cost of producing PVC polymers in a manner that prevents deterioration in their physical properties and appearence that would otherwise result from increased exposure to UV flux.

- Rise in equilibrium temperature represents the increase in global average annual temperature that would be expected under equilibrium conditions with central case estimates of the concentrations of the greenhouse gases in 2075. The middle NAS estimate of climate sensitivity to a doubling of CO2 is used (3°C), resulting in an estimate of a global equilibrium temperature increase of about 6.2°C between 1980 and 2075. Of note is that the actual warming that may be realized will lag by several decades or more. Also of interest is that recent analyses indicate that a climate sensitivity of 4°C may be a preferred central case assumption (see Chapter 6). Assuming a doubled CO2 climate sensitivity of 4°C implies an equilibrium warming of over 8°C by 2075. (These estimates do not include the effect of additional stratospheric water vapor due to methane. The effect of vertical ozone depletion is taken from Ramanathan et al. (1985), and is not based on the ozone depletion estimates presented here.)

- Sea level rise occurs as global warming leads to thermal expansion of ocean waters and to increased discharge of ice and meltwater from Antarctica and from glaciers (see Chapter 15). Using an estimate of the transient change in temperature by 2075, a sea level rise of 101 cm is estimated.

EXHIBIT 18-4

Materials, Climate and Other Effects: Central Case

TYPE OF EFFECT	EFFECT	UNITS
Effects Estimated Quantitatively for the U.S.		
Materials Damage a/	550	Present Value (millions of 1985 dollars)
Rise in Equilibrium Temperature by 2075 b/	6.2	Degrees Centigrade
Sea Level Rise by 2075	101	Centimeters
Effects Based on Case Studies and Research in Early Stages		
Cost of Sea Level Rise in Charleston and Galveston c/	1,145-2,807	Present Value (millions of 1985 dollars)
Reduction in Soybean Seed Yield d/	14.3	Percent in Year 2075
Increase in Ground-Based Ozone e/	5.1-27.2	Percent in Year 2075
Loss of Northern Anchovy Population f/	3.8-19.8	Percent in Year 2075

a/ Discounted over 1985-2075 using a real discount rate of 3 percent.

b/ Estimated using an assumed climate sensitivity of 3°C (middle NAS estimate). Recent analysis indicates that 4°C may be a preferred central case assumption. Using a 4°C sensitivity, the estimated equilibrium warming in 2075 is about 8.4°C.

c/ Lowest estimate with anticipation of sea level rise; highest estimate without anticipation.

d/ Essex cultivar only in normal years.

e/ Lowest estimate is for Los Angeles, California; highest estimate is for Nashville, Tennessee.

f/ Lowest estimate assumes 15-meter vertical mixing of the top ocean layer; highest estimate assumes 10-meter vertical mixing.

- Costs of sea level rise impacts were estimated from two case studies (see Chapter 15). At a global rise of 101 cm, costs for Charleston, S.C. and Galveston TX range from about $1,145 to $2,800 million (1985 dollars, present value at a real 3 percent discount rate). This range reflects different assumptions about the rate and type of adjustments made to prepare for or respond to the impacts of sea level rise. The major impacts include increased storm damage and land lost to increased shoreline retreat (due to inundation and erosion).

- Reductions in soybean yield are based on preliminary experiments described in Chapter 11. At the central case global ozone depletion in 2075 of about 19 percent, the reduction in yield is estimated at of 14.3 percent in the year 2075. This estimate is very uncertain and is based on a series of experiments with a sensitive cultivar. Two out of three cultivars tested in the greenhouse appear sensitive to UV-B. The cultivar tested is one that is becoming preferred in many areas. It is included only to indicate the potential magnitude of effects on plants and cannot be extrapolated to other cultivars or species.

- Increase in Ground-based Ozone (smog) is based on the range reported for three urban case studies (see Chapter 14). Using the central case estimate of global ozone depletion in 2075 (19 percent), ground-based ozone will rise an estimated 5 to 27 percent in 2075. This estimate is preliminary and cannot be extrapolated to the entire U.S.

- Loss of Anchovy Population of about 4 to 20 percent is based on experiments in laboratory tanks. The range represents alternative assumptions about the depth of the mixed layer of the oceans and incorporates the 2075 global ozone depletion value (19 percent). The results of the laboratory tank studies cannot be extrapolated to natural ecosystems (see Chapter 12).

COMPARISON OF CENTRAL CASE WITH RESULTS USING ALTERNATIVE ASSUMPTIONS

This section identifies the sensitivity of central case risk estimates to assumptions regarding: (1) the future production, use and emissions of ozone-modifying substances; (2) the type of model used to characterize ozone depletion; (3) the sensitivity of the global climate to greenhouse gas forcings; and (4) the concentration of greenhouse gas emissions.

Alternative Scenarios of Production, Use, and Emissions

Four alternative scenarios of the production, use, and emissions of ozone-modifying substances were explored. The scenarios (discussed in Chapter 3) reflect a wide range of uncertainties and estimates made by authors from around the world. Key factors varied in the development of the scenarios include:

- projected future population;
- economic growth;
- the relationship between economic growth and population; and
- technological innovation that would affect the intensity of use of ozone-modifying substances.

To investigate the significance of alternative assumptions about future production and emissions, all other assumptions were held fixed.

The scenarios of future production and emissions range from 0.0 percent growth to 5.0 percent growth per year through 2050. This wide range represents the results of discussions at the United Nations Environment Programme (UNEP) Workshop on the Control of Chlorofluorocarbons held in Rome, Italy in May 1986. Discussion at the workshop underscored the uncertainty about potential future production, use, and emissions of ozone-modifying substances. The Workshop discussion indicated that it is unlikely that future production and emissions growth would fall outside this range.

A narrower range of uncertainty was also presented at the Workshop. This range, from 1.2 to 3.8 percent annual growth through 2050, reflects the likely range of future emissions. These rates are referred to as the low and high scenarios respectively. The extreme rates of 0.0 and 5.0 percent annual growth are referred to as the lowest[7] and highest scenarios respectively.

Exhibits 18-5 through 18-9 illustrate the sensitivity of risk estimates to this wide range of assumptions about emissions. Exhibit 18-5 displays estimates of ozone depletion for the central case and for the emissions scenarios. The lowest emissions scenario (zero percent annual growth) shows a slight decline in ozone levels until 2030, after which ozone levels increase.

The high and highest emissions scenarios result in greatly accelerated ozone depletion. In contrast to the central case, ozone depletion reaches 30 percent well before 2100 in the high and highest scenarios. The sensitivity of ozone depletion to emissions estimates carries over into health risks. The

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[7] The zero percent emissions growth scenario describes what would happen if production was totally frozen at today's levels.

EXHIBIT 18-5

Global Average Ozone Depletion: Emission Scenarios

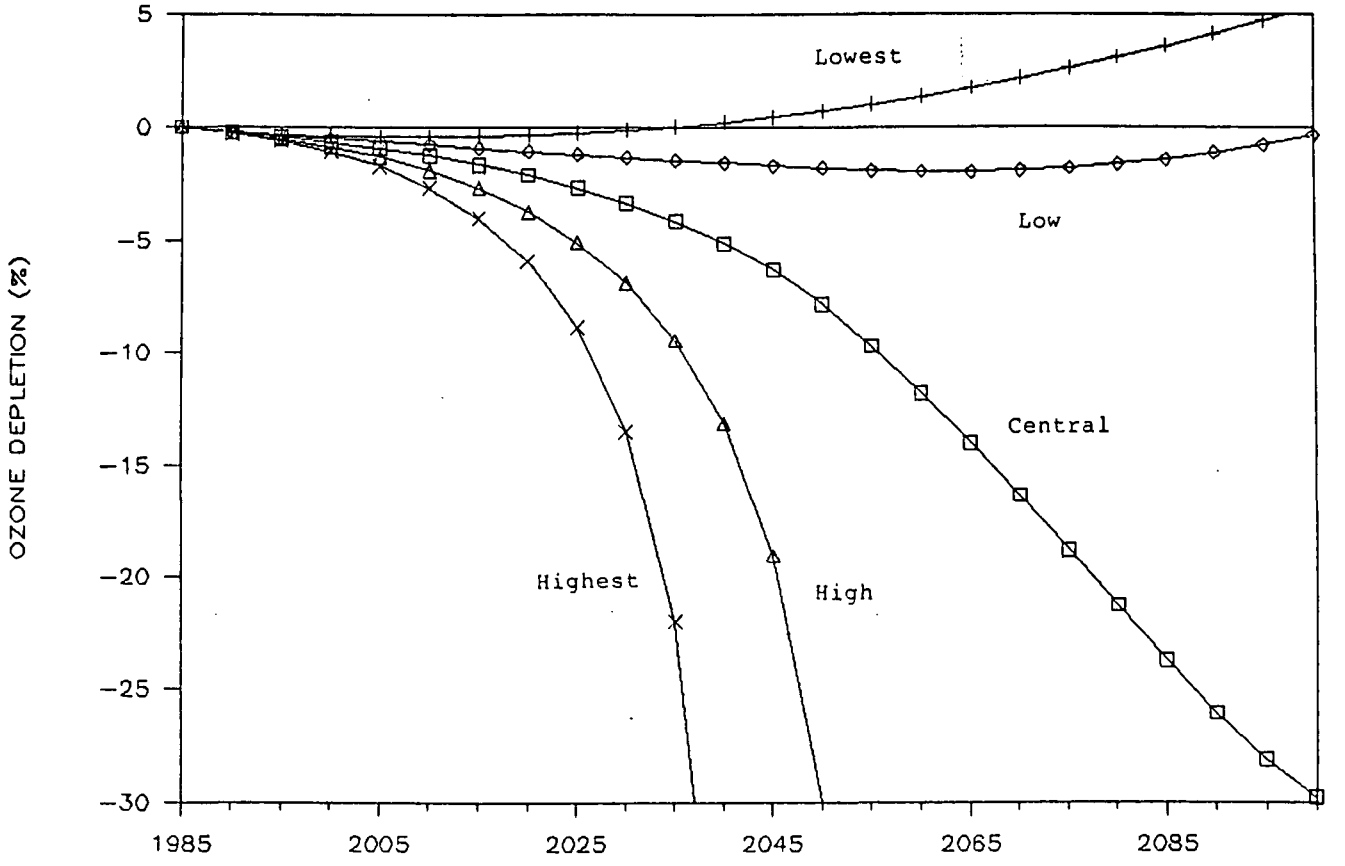


EXHIBIT 18-6

Human Health Effects: Emissions Scenarios
Additional Cumulative Cases and Deaths Over Lifetimes of People Alive Today
(Figures in Parentheses are Percent Changes from Central Case)

HEALTH EFFECT	EMISSIONS SCENARIOS			EXTREME CASES	
	Low	Central	High	Lowest	Highest
Non-Melanoma Skin Tumors					
Additional Basal Cases	265,000 (-58)	630,600	1,947,700 (209)	78,700 (-88)	5,167,500 (719)
Additional Squamous Cases	152,600 (-61)	386,900	1,355,800 (250)	40,900 (-89)	3,868,800 (900)
Additional Deaths	6,500 (-61)	16,500	56,800 (244)	1,700 (-90)	161,100 (876)
Melanoma Skin Tumors					
Additional Cases	5,700 (-54)	12,300	33,900 (176)	2,100 (-83)	84,200 (584)
Additional Deaths	1,700 (-56)	3,900	11,400 (192)	600 (-85)	29,000 (644)
Senile Cataract					
Additional Cases	210,500 (-65)	593,600	2,124,800 (258)	38,400 (-94)	5,133,100 (765)

EXHIBIT 18-7

Human Health Effects: Emissions Scenarios
Additional Cumulative Cases and Deaths Over Liftimes of People Born 1985-2029
(Figures in Parentheses are Percent Changes from Central Case)

HEALTH EFFECT	EMISSIONS SCNEARIOS			EXTREME CASES	
	Low	Central	High	Lowest*	Highest
Non-Melanoma Skin Tumors					
Additional Basal Cases	713,800 (-86)	5,012,900	23,325,300 (365)	-265,300 (-105)	45,210,400 (802)
Additional Squamous Cases	369,100 (-88)	3,185,800	18,418,500 (478)	-161,100 (-105)	37,279,900 (1,070)
Additional Deaths	16,000 (-88)	135,000	763,000 (465)	-6,600 (-105)	1,538,200 (1,039)
Melanoma Skin Tumors					
Additional Cases	20,500 (-81)	109,800	466,500 (325)	-4,200 (-104)	948,400 (764)
Additional Deaths	5,300 (-84)	32,200	137,200 (326)	-1,500 (-105)	266,800 (729)
Senile Cataract					
Additional Cases	389,600 (-89)	3,463,400	12,243,400 (254)	-262,800 (-108)	20,285,300 (486)

* Negative numbers indicate that fewer cases and/or deaths are estimated to occur compared to the expected number of cases and/or deaths that would arise if total column ozone levels are unchanged.

EXHIBIT 18-8

Human Health Effects: Emissions Scenarios
Additional Cumulative Cases and Deaths Over Lifetimes of People Born 2030-2074
(Figures in Parentheses are Percent Changes from Central Case)

HEALTH EFFECT	EMISSIONS SCENARIOS			EXTREME CASES	
	Low	Central	High	Lowest*	Highest
Non-Melanoma Skin Tumors					
Additional Basal Cases	620,700 (-96)	17,630,500	58,482,700 (232)	-1,429,500 (-108)	84,939,900 (382)
Additional Squamous Cases	301,700 (-98)	12,122,400	52,033,800 (329)	-736,400 (-106)	76,661,100 (532)
Additional Deaths	13,200 (-97)	509,300	2,132,600 (319)	-31,800 (-106)	3,138,100 (516)
Melanoma Skin Tumors					
Additional Cases	21,100 (-95)	430,500	1,397,000 (225)	-38,400 (-109)	2,046,900 (375)
Additional Deaths	5,100 (-96)	115,100	354,200 (208)	-10,300 (-109)	513,900 (346)
Senile Cataract					
Additional Cases	284,900 (-97)	8,295,800	19,858,700 (139)	-887,700 (-111)	28,011,400 (238)

* Negative numbers indicate that fewer cases and/or deaths are estimated to occur compared to the expected number of cases and/or deaths that would arise if total column ozone levels are unchanged.

highest emissions scenario, for example, leads to a larger increase in UV radiation reaching the earth's surface and to correspondingly larger health risks. In contrast, the lowest scenario results in an ozone abundance after 2035 and thus a reduction in UV radiation.

For purposes of evaluating potential health effects, the maximum ozone depletion was (arbitrarily) set to 50 percent. Depletion exceeding 50 percent was considered to be outside the range for which the atmospheric models are valid. This arbitrary cutoff affected ozone depletion estimates in the high and highest emissions scenarios only. Once 50 percent depletion is reached, depletion is held constant for the remainder of the analysis period.

Exhibits 18-6, 18-7, and 18-8 present estimates of the health risks across the emissions scenarios for the three population cohorts. The exhibits provide a range of risk estimates reflecting only the differences in the emission scenarios. The exhibits illustrate how risk estimates respond to assumptions about emissions.

The low and lowest emissions scenarios result in fewer additional cumulative cases and deaths related to non-melanoma skin cancer, melanoma skin cancer, and senile cataract. The reduction in risk results from lower ozone depletion (and UV radiation) in these scenarios. For example, comparing the lowest and central emissions scenarios in Exhibit 18-6, people alive today would experience an estimated 88 percent fewer additional cases of basal cell skin cancer if the lowest scenario is used.

The high and highest emissions scenarios produce parallel increases in cases and deaths. People alive today would experience an estimated 7-fold increase in additional cases of basal non-melanoma skin cancers if emissions grow at 5 percent (highest emissions scenario) instead of 2.5 percent (central case).

Exhibits 18-7 and 18-8 show the results for the latter two cohorts respectively. Compared to the results for the cohort of people alive today and the cohort of people born from 2030 to 2075 (last cohort), the results for the people born between now and 2029 (middle cohort) indicate a larger sensitivity. For example, using the highest emissions scenario would increase cases of basal cell skin cancers by 800 percent for the middle cohort but by only 720 percent for people alive today and 380 percent for the last cohort.

The increased sensitivity of the risk estimates to emissions for the middle cohort stems from the exposure of this cohort to larger changes in UV radiation and the longer exposure of individuals in the cohort to increased UV. The reduced sensitivity for the last cohort is an artifact of the method used to model risks. The risk estimates for this cohort are affected by the arbitrary 50 percent cap placed on ozone depletion. This assumption dampens the significance of emissions assumptions particularly for individuals born near 2075.

The risk estimates for the high and highest emissions scenarios should be treated with caution because the dose-response coefficients used to derive risk estimates were estimated over a limited range of UV radiation. The dose-response parameters may not be valid for the extreme changes in UV radiation expected in these scenarios.

Exhibit 18-9a displays estimates of non-health risks for the emissions scenarios. As with the health risks, the range of effects is very large. Of note is that the global warming estimates for 2075 range from 4.9°C in the lowest scenario to over 12°C in the highest. Exhibit 18-9b shows the greenhouse equilibrium temperature increase over time for the five scenarios.

Comparison with Results from a 2-Dimensional Atmospheric Model

A variety of models have been developed to assess potential changes in the stratosphere due to emissions of CFCs and other ozone-modifying substances (see Chapter 5). The central case risk estimates rely on a parameterized representation of a one-dimensional model of the atmosphere with latitudinal gradients generated to conform to Isaksen's latitudinal distribution (see Chapter 17). For comparison purposes, the results of a time-dependent two-dimensional model developed by Isaksen (1986) are presented here. Two-dimensional models are believed to be preferable for risk assessment (see Chapter 5).

The results of Isaksen's analysis are currently only available through 2030. Therefore, a complete comparison of risks is not possible at this time (as additional data become available, a full comparison of risks will be performed).

Exhibit 18-10 displays estimates of ozone depletion from the one- and two-dimensional atmospheric models for two scenarios of emissions. The first scenario is a 3.0 percent growth rate of CFCs-11, 12, and 22, methyl chloroform, and carbon tetrachloride. This scenario is not directly comparable to the central case estimates described above (which includes more compounds, and grows at 2.5 percent per year). The exhibit shows that the two-dimensional model results in <u>larger</u> estimates of ozone depletion than does the parameterized representation of the one-dimensional model for this scenario.

The second scenario shown in the exhibit is a 0.0 percent per year growth scenario. Comparing the one-and two-dimensional model estimates, the results from Isaksen's two-dimensional model show more depletion.

In general, two-dimensional models appear to produce larger estimates of ozone depletion than do one-dimensional models (see Chapter 5). Therefore, the use of a one-dimensional model in the central case will bias risk estimates downward, perhaps by as much as one-half.

EXHIBIT 18-9a

Materials, Climate, and Other Effects: Emissions Scenarios
(Figures in Parentheses are Percentage Changes from Central Case)

TYPE OF EFFECT	EMISSIONS SCENARIOS			EXTREME CASES		UNITS
	Low	Central	High	Lowest	Highest	
Effects Estimated Quantitatively for the U.S.						
Materials Damage a/	268 (-51)	550	907 (65)	188 (-66)	1336 (143)	Present Value (millions of 1985 dollars)
Rise in Equilibrium Temperature by 2075	5.3 (-14)	6.2	8.2 (32)	4.9 (-42)	12.2 (97)	Degrees Centigrade
Sea Level Rise by 2075	94 (-7)	101	115 (14)	91 (-10)	140 (39)	Centimeters
Effects Based on Case Studies and Research in Early Stages						
Cost of Sea Level Rise in Charleston and Galveston b/	1094-2673	1145-2807	1243-3076	1073-2616	1420-3556	Present Value (millions of 1985 dollars)
Reduction in Soybean Seed Yield c/	1.4	14.3	>19	f/	>19	Percent in Year 2075
Increase in Ground-Based Ozone d/	0.5-2.6	5.1-27.2	>9.4->50.0	f/	>9.4->50.0	Percent in Year 2075
Loss of Northern Anchovy Population e/	0.0	3.8-19.8	>11.0->25.0	f/	>11.0->25.0	Percent in Year 2075

a/ Discounted over 1985-2075 using a real discount rate of 3 percent.

b/ Lowest estimate with anticipation of sea level rise; highest estimate without anticipation.

c/ Essex cultivar only in normal years.

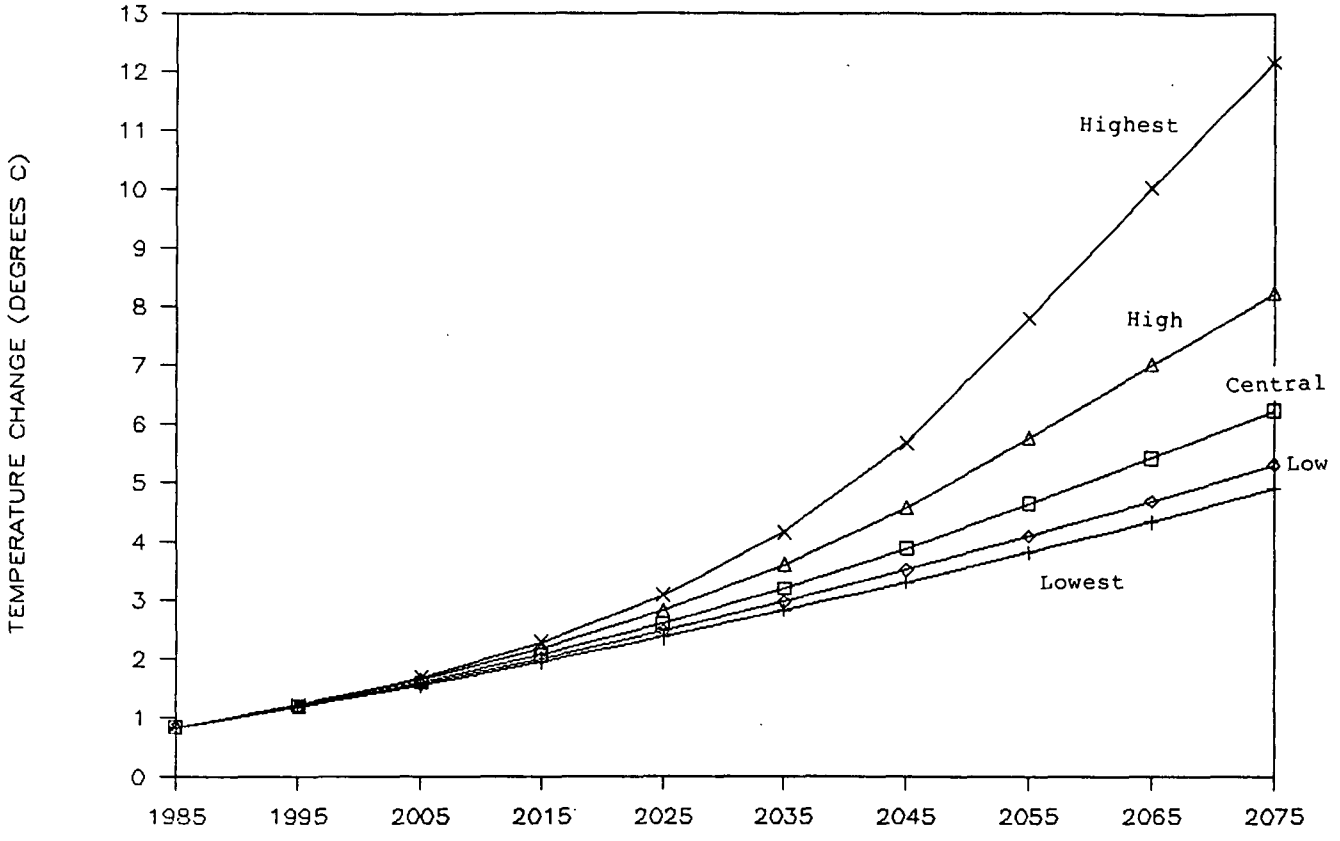
d/ Lowest estimate is for Los Angeles, California; highest estimate is for Nashville, Tennessee.

e/ Lowest estimate assumes 15-meter vertical mixing of the top ocean layer; highest estimate assumes 10-meter vertical mixing.

f/ Impact not evaluated for increased ozone or decreased UV-B radiation projections.

EXHIBIT 18-9b

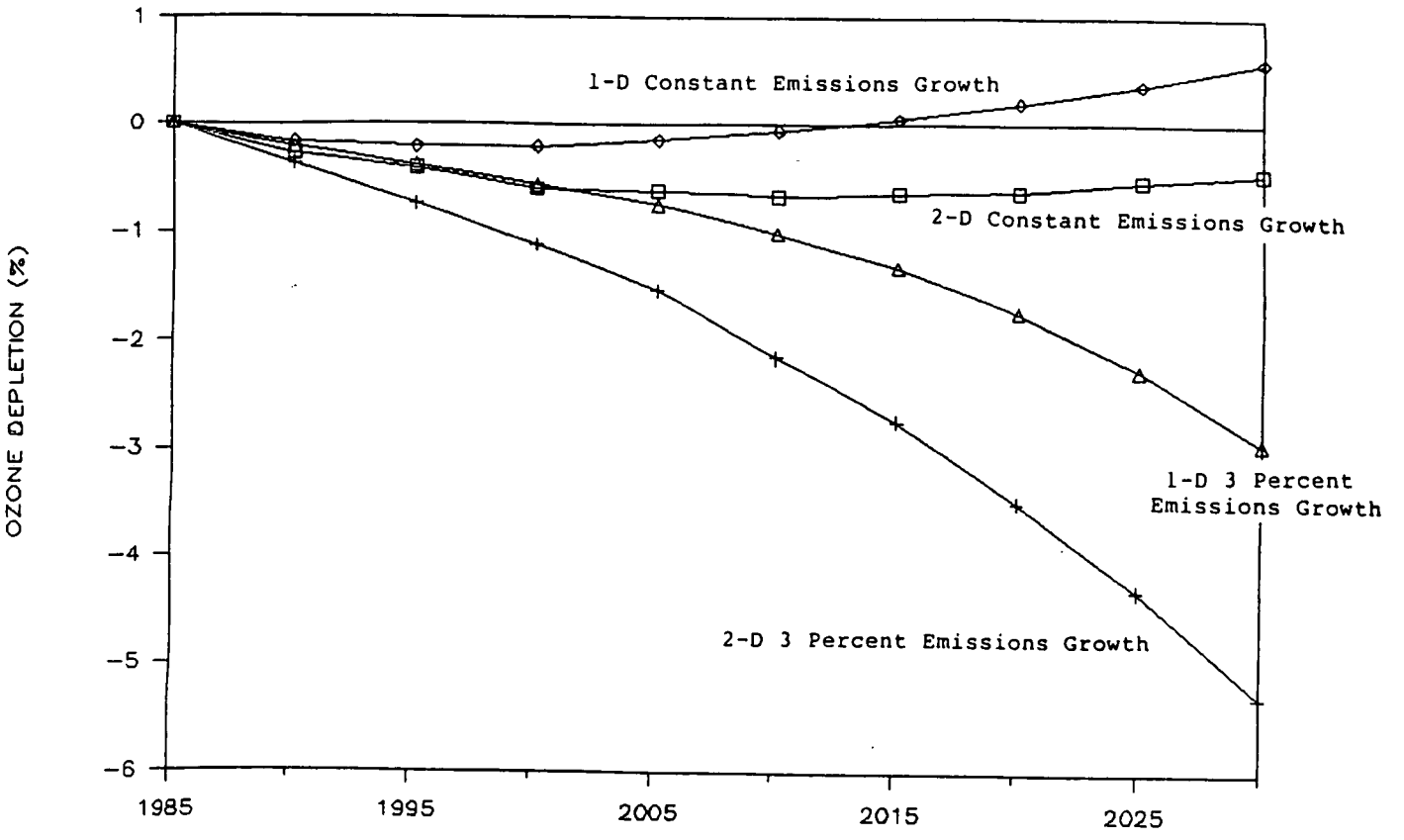
Equilibrium Temperature Change: Emissions Scenarios*



* Computed assuming that the climate sensitivity to a doubling of carbon dioxide is 3°C. This assumption is in the middle of the NAS range of 1.5°C to 4.5°C (see Chapter 6). Note that the actual warming that may be realized will lag by several decades or more.

EXHIBIT 18-10

Global Average Ozone Depletion: Comparison to Results with a 2-Dimensional Atmospheric Model



Alternative Assumptions about Sensitivity of Atmosphere to Greenhouse Gases

The central case assumes that the sensitivity of the global climate system is such that a doubling of atmospheric CO2 would result in a 3°C increase in the average global temperature. This climate sensitivity is the middle of a range published by the NAS in 1983 of 1.5°C to 4.5°C. Exhibit 18-11 displays estimates of temperature-sensitive effects for the limits of this NAS range. As expected, the warming and sea level rise estimates are lower for the 1.5°C sensitivity and higher for the 4.5°C sensitivity.

Also shown in the exhibit are estimates for a 4°C sensitivity. Recent analyses indicate that 4°C may be a preferred central case assumption for climate sensitivity (see Chapter 6).

Alternative Assumptions about Emissions of Trace Gases

Uncertainty exists about potential future changes in atmospheric concentrations of the greenhouse gases: carbon dioxide (CO2); methane (CH4); and nitrous oxide (N2O). This uncertainty arises from at least two sources. First, to limit the potental for future global warming, governments may agree to take steps to limit growth in the concentrations of these gases. However, the timing and stringencies of such potential steps are not known. Second, future concentrations of methane are influenced by numerous natural as well as anthropogenic factors that have not been well characterized to date (see Chapter 4). Therefore, the rate of future increases in methane concentrations is particularly uncertain even in the absence of potential government regulations to limit global warming.

Through their roles in chemical reactions in the stratosphere, trace gases affect both total column ozone and the potential for future global warming. Exhibit 18-12 summarizes the implications of changes in concentrations of the gases for ozone depletion and global warming. In general, each greenhouse gas <u>increases</u> global temperature but <u>counters</u> ozone depletion.

Any risk assessment requires an assumption about future global warming and whether governments take actions to limit warming. The standard assumption generally has been that warming is never limited. To examine the sensitivity of the risk estimates to this issue, the implications of reducing the growth in concentrations of the trace gases to limit warming to 3°C by 2075 was examined. In this case the growth rate of each of the three gases was reduced to approximately 20 percent of its central case growth rate after 2000. Because the trace gases counter depletion, lowering rates of growth in concentrations to limit warming to 3°C by 2075 results in estimates of more ozone depletion than in the central case.

Exhibits 18-13 to 18-17 compare the central case results to the results if global warming is limited to 3°C. In Exhibit 18-13 the increase in ozone depletion over the central case is substantial; by 2050 the difference exceeds 5 percent depletion. Exhibits 18-14 to 18-16 show the increased health risks

EXHIBIT 18-11

Materials, Climate, and Other Effects:
Sensitivity to Relationship Between Climate Change and CO2 Emissions
(Figures in Parentheses are Percentage Changes from Central Case)

Type of Effect	DOUBLED CO2				UNITS
	1.5 C	Central (3 C)	(4 C)	4.5 C	
Effects Estimated Quantitatively for the U.S.					
Rise in Equilibrium Temperature by 2075	3.0 (-52)	6.2	8.4 (35)	9.5 (53)	Degrees Centigrade
Sea Level Rise by 2075	80 (-21)	101	110 (9)	114 (13)	Centimeters
Effects Based on Case Studies and Research In Early Stages					
Cost of Sea Level Rise in Charleston and Galveston a/	985-2405	1145-2807	1208-2980	1236-3057	Present Value (millions of 1985 dollars)

a/ Lowest estimate with anticipation of sea level rise; highest estimate without anticipation.

EXHIBIT 18-12

Summary of Effects of Greenhouse Gases on Ozone Depletion and Global Equilibrum Temperature

GREENHOUSE GAS	STRATOSPHERIC OZONE	GLOBAL TEMPERATURE
Methane (CH4)	Counters Depletion	Increases
Nitrous Oxide (N20)	Counters Depletion in High Chlorine Cases	Increases
Carbon Dioxide (CO2)	Adds Ozone	Increases

EXHIBIT 18-13

Global Average Ozone Depletion: Scenario of Limits to Future Global Warming

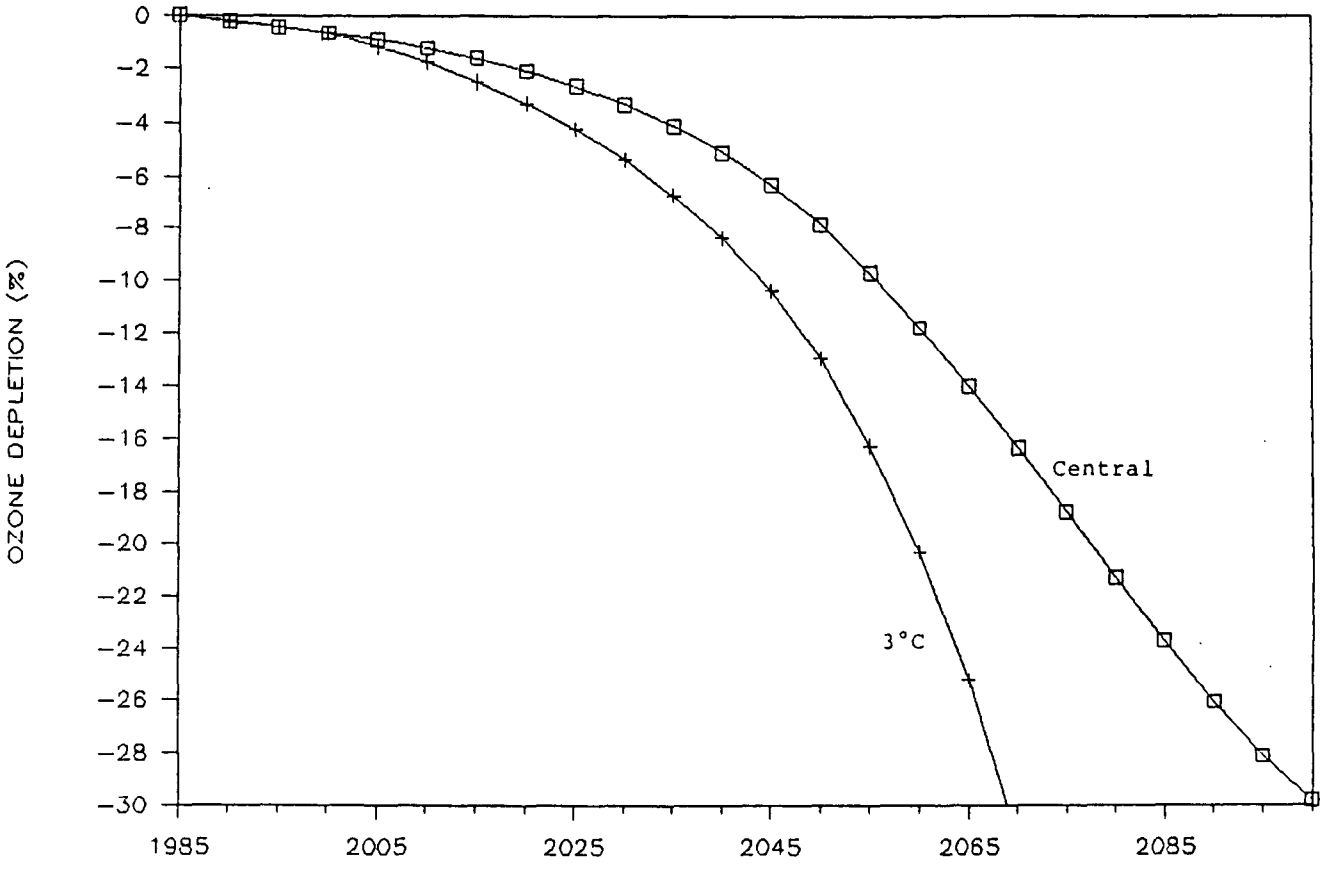


EXHIBIT 18-14

Human Health Effects: Scenarios of Limits to Future Global Warming Additional Cumulative Cases and Deaths Over Lifetimes of People Alive Today (Figures in Parentheses are Percentage Changes from Central Case)

HEALTH EFFECT	LIMITS TO FUTURE GLOBAL WARMING 3°C	Central Case (no limit)
Non-Melanoma Skin Tumors		
Additional Basal Cases	1,001,800 (59)	630,600
Additional Basal Cases	632,700 (64)	386,900
Additional Deaths	26,800 (62)	16,500
Melanoma Skin Tumors		
Additional Cases	18,900 (54)	12,300
Additional Deaths	6,200 (59)	3,900
Senile Cataract		
Additional Cases	970,900 (64)	593,600

EXHIBIT 18-15

**Human Health Effects: Scenarios of Limits to Future Global Warming
Additional Cumulative Cases and Deaths Over Lifetimes of People Born 1985-2029
(Figures in Parentheses are Percentage Changes from Central Case)**

HEALTH EFFECT	LIMITS TO FUTURE GLOBAL WARMING 3°C	Central Case (no limit)
Non-Melanoma Skin Tumors		
Additional Basal Cases	10,679,700 (113)	5,012,900
Additional Squamous Cases	7,781,500 (144)	3,185,800
Additional Deaths	324,900 (141)	135,000
Melanoma Skin Tumors		
Additional Cases	215,000 (96)	109,800
Additional Deaths	64,500 (100)	32,200
Senile Cataract		
Additional Cases	6,899,400 (99)	3,463,400

EXHIBIT 18-16

**Human Health Effects: Scenarios of Limits to Future Global Warming
Additional Cumulative Cases and Deaths Over Lifetimes of People Born 2030-2074
(Figures in Parentheses are Percentage Changes from Central Case)**

HEALTH EFFECT	LIMITS TO FUTURE GLOBAL WARMING 3°C	Central Case (no limit)
Non-Melanoma Skin Tumors		
Additional Basal Cases	39,303,000 (123)	17,630,500
Additional Squamous Cases	33,400,200 (176)	12,122,400
Additional Deaths	1,374,300 (170)	509,300
Melanoma Skin Tumors		
Additional Cases	903,700 (110)	430,500
Additional Deaths	237,800 (107)	115,100
Senile Cataract		
Additional Cases	14,947,300 (80)	8,295,800

EXHIBIT 18-17

Materials, Climate, and Other Effects: Scenarios of Limits to Future Global Warming
(Figures in Parentheses are Percentage Changes from Central Case)

TYPE OF EFFECT	LIMITS TO FUTURE GLOBAL WARMING 3 C	Central Case (no limit)	UNITS
Effects Estimated Quantitativety for the U.S.			
Materials Damage a/	726	550	Present Value (millions of 1985 dollars)
Rise in Equilibrium Temperature by 2075	3.0 (-52)	6.2	Degrees Centigrade
Sea Level Rise by 2075	76 (-25)	101	Centimeters
Effects Based on Case Studies and Research in Early Stages			
Cost of Sea Level Rise in Charleston and Galveston b/	967-2328	1145-2807	Present Value (millions of 1985 dollars)
Reduction in Soybean Seed Yield c/	>19.0	14.3	Percent in Year 2075
Increase in Ground-Based Ozone d/	>9.4->50.0	5.1-27.2	Percent in Year 2075
Loss of Northern Anchovy Population e/	>11.0->25.0	3.8-19.8	Percent in Year 2075

a/ Discounted over 1985-2075 using a real discount rate of 3 percent.

b/ Lowest estimate with anticipation of sea level rise; highest estimate without anticipation.

c/ Essex cultivar only in normal years.

d/ Lowest estimate is for Los Angeles, California; highest estimate is for Nashville, Tennessee.

e/ Lowest estimate 15-meter vertical mixing of the top ocean layer; highest estimate 10-meter vertical mixing.

expected for the three population cohorts. The people alive today may experience an increase in health risks on the order of 60 percent relative to the central case values. The latter cohorts are more sensitive.

Exhibit 18-17 displays non-health risks. As shown in the exhibit, warming is reduced from 6.2°C in the central case to 3°C. As a result, sea level rise impacts are also reduced. The ozone-sensitive risks are increased, however. If global warming were held to less than 3°C by 2075 the risks from ozone depletion would be even greater.

A second sensitivity case is examined in which several alternative scenarios of methane concentrations are examined. To evaluate the sensitivity of the results to the methane growth rates, three methane scenarios were examined in addition to the central case. Other trace gases were kept at constant values consistent with the central case. In the lowest scenario, the methane concentration grows through 2010 at 1 percent per year, and then is held constant. In the low scenario, the methane concentration grows at 1 percent through 2000, then grows at about 0.6 percent through 2050, after which it remains constant. In the highest scenario, the methane concentration grows at 1 percent per year through 2020, after which it grows more rapidly than 1 percent.[8]

Exhibit 18-18 shows that higher concentrations of atmospheric methane would reduce ozone depletion, while lower concentrations result in more depletion. Contrasting the highest methane scenario to the central case, ozone depletion is roughly equal until 2025, when the contributions to ozone from increased methane concentrations begin to dampen depletion. The influence of increased methane concentration becomes more pronounced after 2025, and depletion reaches an inflection point in 2085 after which ozone levels rise.

The lower methane scenarios cause the ozone depletion estimates to increase. The implications of the resulting differences in ozone depletion for the risk estimates is shown in Exhibits 18-18 through 18-22. Of note in Exhibit 18-22 is that the lower methane scenarios reduce the estimate of global warming, while the high scenarios increase it.

SENSITIVITY OF EFFECTS TO PARAMETER UNCERTAINTY

This section analyzes the sensitivity of risk estimates to statistical uncertainties in two areas: (1) estimates of dose-response between UV radiation and health effects, and (2) the relationship between ozone depletion and atmospheric concentrations of chlorine (i.e., the sensitivity of the atmosphere to total column ozone depletion from chlorine sources). These uncertainties are investigated together because the parameters describing the dose-response and the depletion-emissions relationships were estimated using statistical methods the results of which included statistical estimates of lower- and upper-bound parameters.

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[8] See Chapter 4 for a discussion of the sources of these scenarios.

EXHIBIT 18-18

Global Average Ozone Depletion: Methane Emissions Cases

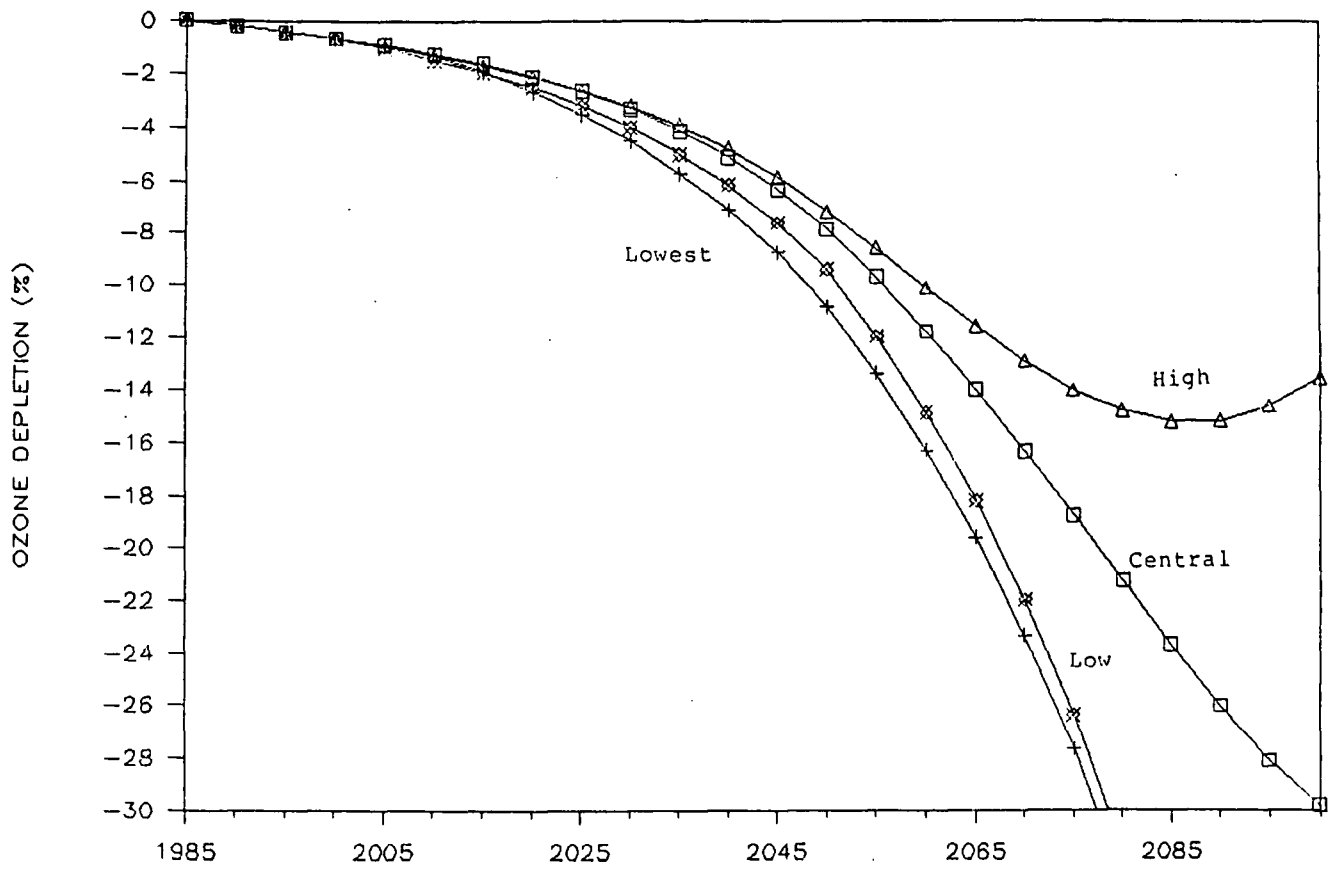

EXHIBIT 18-19

Human Health Effects: Methane Emissions Cases
Additional Cumulative Cases and Deaths Over Lifetimes of People Alive Today
(Figures in Parentheses are Percentage Changes from Central Case)

HEALTH EFFECT	METHANE EMISSIONS SCENARIOS			
	Lowest	Low	Central Case	High
Non-Melanoma Skin Tumors				
Additional Basal Cases	818,200 (30)	757,000 (20)	630,600	611,600 (-3.0)
Additional Squamous Cases	512,100 (32)	469,200 (21)	386,900	372,500 (-3.7)
Additional Deaths	21,700 (32)	19,900 (21)	16,500	15,900 (-3.6)
Melanoma Skin Tumors				
Additional Cases	15,500 (26)	14,600 (19)	12,300	12,100 (-1.6)
Additional Deaths	5,000 (28)	4,700 (21)	3,900	3,800 (-2.6)
Senile Cataract				
Additional Cases	791,700 (33)	720,400 (21)	593,600	562,700 (-5.2)

EXHIBIT 18-20

Human Health Effects: Methane Emissions Cases
Additional Cumulative Cases and Deaths Over Lifetimes of People Born 1985-2029
(Figures in Parentheses are Percentage Changes from Central Case)

HEALTH EFFECT	METHANE EMISSIONS SCENARIOS			
	Lowest	Low	Central Case	High
Non-Melanoma Skin Tumors				
Additional Basal Cases	8,055,800 (61)	7,421,300 (48)	5,012,900	3,825,400 (-24)
Additional Squamous Cases	5,600,900 (76)	4,140,200 (61)	3,185,800	2,284,800 (-28)
Additional Deaths	235,000 (74)	215,800 (60)	135,000	97,600 (-28)
Melanoma Skin Tumors				
Additional Cases	166,500 (52)	152,500 (39)	109,800	89,600 (-18)
Additional Deaths	49,700 (54)	45,700 (42)	32,200	25,500 (-21)
Senile Cataract				
Additional Cases	5,492,900 (59)	5,192,200 (50)	3,463,400	2,450,700 (-29)

EXHIBIT 18-21

Human Health Effects: Methane Emissions Cases
Additional Cumulative Cases and Deaths Over Lifetimes of People Born 2030-2074
(Figures in Parentheses are Percentage Changes from Central Case)

HEALTH EFFECT	METHANE EMISSIONS SCENARIOS			
	Lowest	Low	Central Case	High
Non-Melanoma Skin Tumors				
Additional Basal Cases	33,293,900 (89)	32,360,400 (84)	17,630,500	8,848,100 (-50)
Additional Squamous Cases	27,448,800 (126)	26,581,900 (119)	12,122,400	5,231,900 (-59)
Additional Deaths	1,132,500 (122)	1,097,100 (115)	509,300	223,600 (-56)
Melanoma Skin Tumors				
Additional Cases	751,500 (75)	726,400 (69)	430,500	243,300 (-43)
Additional Deaths	201,800 (75)	196,000 (70)	115,100	62,600 (-46)
Senile Cataract				
Additional Cases	13,686,000 (65)	13,479,800 (62)	8,295,800	4,247,100 (-49)

EXHIBIT 18-22

Materials, Climate, and Other Effects: Methane Emissions Cases
(Figures in Parentheses are Percentage Changes from Central Case)

TYPE OF EFFECT	METHANE EMISSION SCENARIOS				UNITS
	Lowest	Low	Central Case	High	
Effects Estimated Quantitatively for the U.S.					
Materials Damage a/	661 (20)	617 (12)	550	529 (-4)	Present Value (millions of 1985 dollars)
Rise in Equilibrium Temperature by 2075 b/	5.9 (-5)	6.0 (-3)	6.2	6.4 (3)	Degrees Centigrade
Sea Level Rise by 2075	98 (-3)	99 (-2)	101	102 (1)	Centimeters
Effects Based on Case Studies and Research in Early Stages					
Cost of Sea Level Rise in Charleston and Galveston c/	1123-2750	1123-2770	1145-2807	1151-2827	Present Value (millions of 1985 dollars)
Reduction in Soybean Seed Yield d/	>19	>19	14.3	10.6	Percent in Year 2075
Increase in Ground-Based Ozone e/	7.7-41.1	7.4-39.1	5.1-27.2	3.8-20.0	Percent in Year 2075
Loss of Northern Anchovy Population f/	>11.0->25.0	>11.0->25.0	3.8-19.8	0.2-12.1	Percent in Year 2075

a/ Discounted over 1985-2075 using a real discount rate of 3 percent.

b/ Estimates of the rise in equilibrium temperature neglect increases and decreases in tropospheric that would occur with deviations from the central case of methane growth (1%) and changes in stratospheric water vaor.

c/ Lowest estimate with anticipation of sea level rise; highest estimate without anticipation.

d/ Essex cultivar only in normal years.

e/ Lowest estimate is for Los Angeles, California; highest estimate is for Nashville, Tennessee.

f/ Lowest estimate 15-meter vertical mixing of top ocean layer; highest estimate 10-meter vertical mixing.

The statistical uncertainty reported here does not reflect uncertainties in the underlying specifications of the dose-response and ozone-depletion relationships. For example, as described in Chapter 8 there remains a major question regarding the appropriate measure of exposure to use for modeling melanoma skin cancer risks. The uncertainty estimates reported below reflect the implications of the statistical variation reported using one particular measure of exposure. Using alternative exposure measures would increase the range of risk estimates; preliminary results from ongoing work indicate that melanoma mortality risks may be as much as 60 percent lower than the values reported here if an alternative specification of exposure is used (annual UV flux in the place of peak UV flux). Consequently, the full range of uncertainty in these relationships (including specification uncertainty) is larger than the values reported here, which reflect only parameter uncertainty.

Effects at High and Low Estimates of UV Radiation Impact on Human Health

The dose-response relationships between increases in UV radiation and associated health risks were obtained using a variety of statistical techniques (see Chapters 7, 8, 10 and 17). In these studies, the responsiveness of health effects to changes in UV radiation were characterized by dose-response parameters estimated using mathematical functions fitted statistically to detailed epidemiologic data. The results of the statistical analysis included estimates of the standard errors of dose-response parameters.

The standard errors of the dose-response parameters were used to develop high and low estimates of health risks. Low risk estimates use the central case dose-response coefficients minus one standard error; high risk estimates use central case coefficients plus one standard error. All other assumptions are held fixed.

Health risks estimated using the low, central, and high coefficients are presented in Exhibits 18-23 through 18-25. For people alive today, the low dose-response coefficients produced approximately 30 to 45 percent fewer cases of non-melanoma skin tumors, melanoma skin tumors, and senile cataract (Exhibit 18-23). Deaths due to non-melanoma skin tumors, however, decrease by 81 percent in the low case. The larger reduction in deaths results from a combination of two factors: (1) a lower dose-response coefficient for non-melanoma tumors, and (2) an assumption in the low case that a smaller fraction of individuals contracting tumors would die (0.28 percent in place of the 1.0 percent used in the central case -- see Chapters 7 and 17). About 33 percent more cases among people alive today would occur if the high estimates of dose-response coefficients were used.

Effects at High and Low Sensitivities of Ozone Depletion to Emissions

A previous section analyzed the sensitivity of risk estimates to assumptions about the levels of emissions of ozone-modifying substances. This section explores another source of uncertainty in the risk estimates: the relationship between emissions (and their resulting chlorine concentrations) and ozone depletion. This uncertainty was quantified using Monte Carlo modeling techniques to reflect the implications of the convolution

EXHIBIT 18-23

Human Health Effects: Sensitivity to Dose-Response Relationship Additional Cumulative Cases and Deaths Over Lifetimes of People Alive Today (Figures in Parentheses are Percentage Changes from Central Case)

HEALTH EFFECT	SENSITIVITY OF EFFECT TO UV DOSE		
	Low	Central	High
Non-Melanoma Skin Tumors			
Additional Basal Cases	407,500 (-35)	630,600	857,400 (36)
Additional Squamous Cases	262,500 (-32)	386,900	515,400 (33)
Additional Deaths	3,100 (-81)	16,500	22,000 (33)
Melanoma Skin Tumors			
Additional Cases	8,900 (-28)	12,300	15,700 (28)
Additional Deaths	3,500 (-10)	3,900	4,300 (10)
Senile Cataract			
Additional Cases	325,600 (-45)	593,600	801,900 (35)

EXHIBIT 18-24

Human Health Effects: Sensitivity to Dose-Response Relationship
Additional Cumulative Cases and Deaths Over Lifetimes of People Born 1985-2029
(Figures in Parentheses are Percentage Changes from Central Case)

HEALTH EFFECT	SENSITIVITY OF EFFECT TO UV DOSE		
	Low	Central	High
Non-Melanoma Skin Tumors			
Additional Basal Cases	3,142,500 (-37)	5,012,900	7,044,100 (41)
Additional Squamous Cases	2,028,100 (-36)	3,185,800	4,540,000 (43)
Additional Deaths	24,000 (-82)	135,000	192,100 (42)
Melanoma Skin Tumors			
Additional Cases	78,000 (-30)	109,800	142,200 (30)
Additional Deaths	28,800 (-11)	32,200	35,600 (11)
Senile Cataract			
Additional Cases	1,872,500 (-46)	3,463,400	4,732,800 (37)

EXHIBIT 18-25

Human Health Effects: Sensitivity to Dose-Response Relationship
Additional Cumulative Cases and Deaths Over Lifetimes of People Born 2030-2074
(Figures in Parentheses are Percentage Changes from Central Case)

HEALTH EFFECT	SENSITIVITY OF EFFECT TO UV DOSE		
	Low	Central	High
Non-Melanoma Skin Tumors			
Additional Basal Cases	10,496,400 (-40)	17,630,500	26,229,000 (49)
Additional Squamous Cases	6,951,400 (-43)	12,122,400	19,329,900 (59)
Additional Deaths	82,100 (-84)	509,300	806,200 (58)
Melanoma Skin Tumors			
Additional Cases	297,400 (-31)	430,500	574,200 (33)
Additional Deaths	102,000 (-11)	115,100	128,600 (12)
Senile Cataract			
Additional Cases	4,402,100 (-47)	8,295,800	11,503,600 (39)

of the individual ranges of uncertainty that surround estimates of rate constants for atmospheric reactions. The range analyzed here is the tenth and ninetieth percentile estimates.

The effect of varying the sensitivity of ozone depletion to emissions is illustrated in Exhibit 18-26. The exhibit shows that the tenth percentile estimate of ozone depletion is about one half of the central case value. The ninetieth percentile estimate is about twice the central case value and reaches 30 percent depletion in approximately 2067.

The implications for health risks are displayed in Exhibits 18-27 to 18-29 for each of the three population cohorts. In general, increased health risks would be 60 percent lower than central case in the tenth percentile case, and roughly double the increased risks in the ninetieth percentile case.

RELATIVE IMPORTANCE OF KEY UNCERTAINTIES

The analysis presented in this chapter suggests that the estimates of risks to human health and the environment are sensitive to the assumptions, methods, and data used to model risks. Changes in the assumptions, methods, and data were found to alter the risk estimates significantly.

It appears that all risks are underestimated by using the 1-D parameterization to model ozone depletion instead of a 2-D model; the risks may be underestimated by as much as one-half.

The largest range of risk estimates was produced by varying assumptions about future production, use, and emissions of ozone-modifying substances. Estimates of ozone depletion were particularly sensitive to these assumptions. The lowest case emissions scenario leads to a projected increase in ozone levels; the highest case scenario results in extremely rapid depletion. In addition, the high and highest emissions scenarios caused ozone depletion estimates to reach an amount believed to be outside the valid range of the atmospheric models used to analyze changes in total column ozone.

Because estimates of human health effects are driven by projections of ozone depletion, health risks were similarly affected by this range of emissions assumptions. Increased risks to people alive today were found to vary by as much as 500 percent. The sensitivity of health risks to emissions is greater in future generations; individuals yet to be borne were simulated to experience greater changes in exposure to UV radiation over a larger fraction of their lives. Non-health effects were also shown to be most sensitive to emissions assumptions; however, compared to health effects the range of results was narrower.

After assumptions about emissions, assumptions about potential actions taken to limit global warming were found to be most important for estimating health risks. Actions aimed at limiting future warming to 3°C would reduce future potential concentrations of greenhouse gases. Because these gases also counter ozone depletion, an increase in ozone depletion and associated UV

EXHIBIT 18-26

Global Average Ozone Depletion: Sensitivity to Relationship Between Ozone Depletion and Emissions

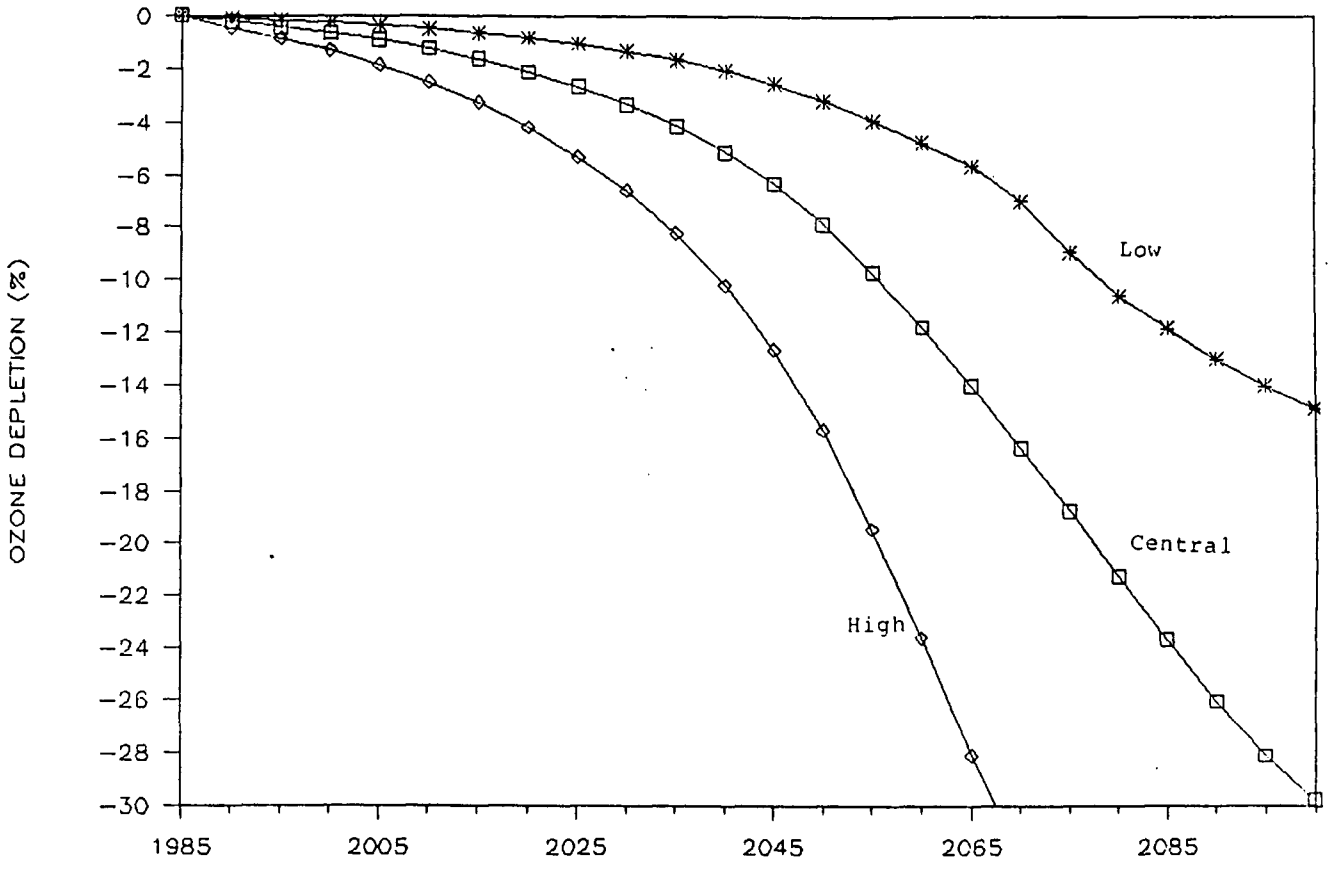

EXHIBIT 18-27

Human Health Effects:
Sensitivity to Relationship Between Ozone Depletion and Emissions
Additional Cumulative Cases and Deaths Over Lifetimes of People Alive Today
(Figures in Parentheses are Percentage Changes from Central Case)

HEALTH EFFECT	SENSITIVITY OF OZONE DEPLETION TO EMISSIONS		
	Low	Central	High
Non-Melanoma Skin Tumors			
Additional Basal Cases	245,000 (-62)	630,600	1,327,600 (111)
Additional Squamous Cases	147,700 (-62)	386,900	838,600 (117)
Additional Deaths	6,300 (-62)	16,500	35,500 (115)
Melanoma Skin Tumors			
Additional Cases	4,800 (-61)	12,300	25,500 (107)
Additional Deaths	1,500 (-62)	3,900	8,200 (110)
Senile Cataract			
Additional Cases	232,400 (-61)	593,600	1,242,100 (109)

EXHIBIT 18-28

Human Health Effects:
Sensitivity to Relationship Between Ozone Depletion and Emissions
Additional Cumulative Cases and Deaths Over Lifetimes of People Born 1985-2029
(Figures in Parentheses are Percentage Changes from Central Case)

HEALTH EFFECT	SENSITIVITY OF OZONE DEPLETION TO EMISSIONS		
	Low	Central	High
Non-Melanoma Skin Tumors			
Additional Basal Cases	1,938,500 (-61)	5,012,900	11,343,900 (126)
Additional Squamous Cases	1,157,300 (-64)	3,185,800	8,099,600 (154)
Additional Deaths	49,400 (-63)	135,000	338,900 (151)
Melanoma Skin Tumors			
Additional Cases	43,200 (-61)	109,800	237,700 (116)
Additional Deaths	12,700 (-61)	32,200	69,700 (116)
Senile Cataract			
Additional Cases	1,449,600 (-58)	3,463,400	6,984,800 (102)

EXHIBIT 18-29

Human Health Effects:
Sensitivity to Relationship Between Ozone Depletion and Emissions
Additional Cumulative Cases and Deaths Over Lifetimes of People Born 2030-2074
(Figures in Parentheses are Percentage Changes from Central Case)

HEALTH EFFECT	SENSITIVITY OF OZONE DEPLETION TO EMISSIONS		
	Low	Central	High
Non-Melanoma Skin Tumors			
Additional Basal Cases	6,983,100 (-60)	17,630,500	37,901,400 (115)
Additional Squamous Cases	4,164,000 (-66)	12,122,400	31,848,500 (163)
Additional Deaths	177,800 (-65)	509,300	1,311,800 (158)
Melanoma Skin Tumors			
Additional Cases	178,900 (-58)	430,500	875,800 (103)
Additional Deaths	48,000 (-58)	115,100	230,500 (100)
Senile Cataract			
Additional Cases	3,726,100 (-55)	8,295,800	14,654,400 (77)

exposure would accompany such actions. People alive today would experience about 60 percent more cases of skin cancer and senile cataract compared to the number of cases estimated to occur in the absence of actions to limit warming. The sensitivity of health risks to assumptions about potential limits to global warming is greater for future generations; up to 175 percent more cases of skin cancer are estimated for people born during 2030 through 2074.

A comparable sensitivity in the health risk estimates was obtained by varying the relationship between ozone depletion and emissions. Changing the responsiveness of ozone depletion to emissions resulted in up to 60 percent fewer additional cases of skin cancers and senile cataract among people alive today (low sensitivity) and up to 120 percent more additional cases for these individuals (high sensitivity). For reasons discussed above, the range of health risk estimates widens for the two future population cohorts investigated in this chapter.

Health risk estimates also were sensitive to assumptions about future concentrations of methane, but to a lesser extent. The analysis suggests that people alive today will contract about 30 percent more cases of skin cancer and senile cataract compared to central case methane concentrations if lowest case assumptions are used. Note that lowering methane concentrations increases ozone depletion and hence increases UV radiation damaging to human health.

Health risks were also sensitive to the uncertainty of the parameters used to characterize dose-response. However, low and high estimates of cases and deaths associated with low and high dose-response coefficients (plus and minus one standard error) fell well within the ranges of risk estimates produced by varying assumptions about emissions and other factors. Nonetheless, health risks were found to vary by as much is 40 percent in either direction if statistical uncertainty in dose-response coefficients is taken into account. The uncertainty in the specification of the risk models is not reflected in these estimates. Work is ongoing to evaluate the implications of using alternative specifications, including alternative exposure measures.

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