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The Health Care Costs of Skin Cancer Caused by Ultraviolet Radiation

Bridger M. Mitchell, James R. Vernon

February 1987
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The Health Care Costs of Skin Cancer Caused by Ultraviolet Radiation

Bridger M. Mitchell, James R. Vernon

February 1987

Prepared for
The U.S. Environmental Protection Agency
This Note assesses the potential health care costs of treating skin cancer that could result from increases in ultraviolet radiation (UV). It reviews the medical and epidemiological findings on the causes and natural history of the three major types of skin cancer, their modes of treatment, and their expected outcomes. These findings are organized in the form of probability trees of alternative outcomes and provide inputs to a prototype computer model of the costs of treatment. The model illustrates the quantitative effects of hypothesized increases in UV levels on the incidence and direct cost of treating skin cancer.

This study is one of a series of publications written at The RAND Corporation on policy issues associated with chemicals that could potentially deplete ozone in the stratosphere ("potential ozone depleters").

The stratospheric ozone layer helps shield the earth from harmful ultraviolet radiation, increases in which may threaten human health, speed deterioration of certain materials, reduce crop yields, and have many other ecological effects. Global emissions of potential ozone depleters may lead to chemical reactions that reduce stratospheric ozone and thereby increase the ultraviolet radiation reaching the earth's surface. However, substantial scientific uncertainty persists about whether human emissions of these chemicals actually threaten the stratospheric ozone layer and, if they do, the extent to which lower ozone levels actually threaten human health.

Policymakers must act in the face of this uncertainty, and RAND's work is designed to help them act with the best information available. To that end, RAND is developing a series of publications addressed to analysts and policymakers responsible for policy decisions on emissions of potential ozone depleters in the United States and elsewhere. These documents include extensive literature reviews, interviews with knowledgeable officials associated with the production and use of potential ozone depleters, and formal chemical, cost, economic, and statistical analyses. The series should also interest the much broader...
audience of analysts and decisionmakers whose organizations would feel the effects of government policies with respect to emissions of such chemicals.

Publications in the series include the following:


This Note was produced under Cooperative Agreement No. CR812066-01-0 with the U.S. Environmental Protection Agency.
SUMMARY

Skin cancers are the most common malignant tumor; about 500,000 new cases occur each year in the United States. Most skin cancers are due to long-term exposure to ultraviolet radiation (UV) and occur on areas of the body most exposed to the sun; they rarely affect nonwhites. The chance of skin cancer increases rapidly with aging.

Skin cancers are classified according to their microscopic cellular characteristics. Basal cell carcinoma (BCC) accounts for about 76 percent of the total, squamous cell carcinoma (SCC) for another 19 percent, and malignant melanoma (MM) perhaps 5 percent. The great majority of BCC and SCC are readily treated and cured with routine outpatient surgical or x-ray procedures. However, a small percentage become invasive and can destroy subcutaneous tissue and body structures. The much rarer MM tumors frequently metastasize, spreading cancer to distant sites in the body, often with fatal results.

The quantitative effect of UV on the rate of the BCC and SCC forms of skin cancer has been measured in a 1977 survey of 10 areas of the United States. Statistical analysis of these data in dose-response models demonstrates an amplified effect of increased UV on cases of BCC and SCC. Each 1 percent increase in UV results in a long-term increase of 1.0 percent to 2.8 percent more skin cancers. Some circumstantial evidence suggests that UV may be one of several causes of malignant melanoma. However, there is as yet no established dose-response model of such an effect.

The natural history of skin cancer and its treatment are quite varied. Probability trees summarize the chance that a particular feature will lead to a cure or develop to the next stage of the disease. It is particularly difficult to obtain data from which to estimate the probabilities of alternatives for diseases that, like skin cancer, are treated in physicians' outpatient offices and clinics. This study reports results gleaned from studies in the medical and epidemiological literature of the proportions of cases having different outcomes. These values are approximate measures that are most nearly applicable to a
cross-section of the U.S. white population; they are not suitable for predicting outcomes for particular age or sex groups.

Basal cell carcinoma is cured some 89 to 98 percent of the time when first treated, and recurring BCC is usually cured on second treatment. Perhaps 1 percent of cases become invasive after 10-20 years and can cause extensive disfigurement and death. BCC can be treated by surgical excision, cryosurgery, electrosurgery, and x-ray radiation; each type of treatment has a high cure rate when undertaken promptly.

Squamous cell carcinoma is frequently preceded by solar keratosis, a benign skin lesion, which occurs commonly at older ages. SCC is treated predominantly with electrosurgery or by excision. About 12 percent of the SCC tumors progress after one to three years, invade the dermal layer of the skin, and are then usually treated with microscopically controlled surgery or surgical excision. These tumors rarely metastasize. In contrast, invasive SCC tumors that appear in the absence of a preceding skin lesion have an 8 to 18 percent chance of metastasizing to the lymph nodes or other areas of the body. Such de novo SCC tumors are treated primarily by excision.

Malignant melanoma originates from the pigment-producing melanocyte cells of the skin. It may spread at the skin's surface or invade the skin more deeply. The probability of metastasis increases directly with the thickness of the tumor; unless treated at an early stage, MM has a high mortality rate. Initial treatment is by excision, but metastatic disease may require extensive surgery or x-ray radiation treatment.

This study estimates the health care costs of treating BCC and SCC types of skin cancer caused by UV radiation using information from dose-response models, the probability trees of disease outcomes and treatment alternatives, and data on costs of treatment. These data are processed in a computer model that can simulate the effects of hypothetical changes in UV levels. Because many components of the model are uncertain and based on incomplete information, the model is illustrative and not suitable for forecasting changes in health care costs. It provides a tool for integrating the available information on incidence, course of disease, and cost of treatment; and it is a promising framework for future research.
Expenditures on health care to treat skin cancer are but one of the health consequences of increased UV. To avoid these risks, people will take steps to reduce their exposure—using sunscreens, changing vacation plans, and perhaps relocating their place of residence. Those who contract skin cancer suffer from anxieties about its uncertain consequences. These consequences are also costly to individual well-being. But because they are less readily measured in monetary terms, the indirect costs of skin cancer are not assessed in this study.
ACKNOWLEDGMENTS

The authors have benefited from discussions with John Hoffman of the U.S. Environmental Protection Agency (EPA) and RAND colleagues Jan Acton, Frank Camm, Mark Chassin, James Hammitt, and Elizabeth Sloss. They extend thanks to Robert Leibowitz, M.D., who reviewed a draft of this Note, and Alfred W. Kopf, M.D., who provided detailed comments. Anonymous reviews by EPA advisers have also contributed to the final form of this Note.
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I. INTRODUCTION

Skin cancer is the most common malignant tumor, with an estimated 500,000 new cases per year in the United States (Scotto, Fears, and Fraumeni, 1983). Sunlight, in the form of ultraviolet radiation (UV-B at wavelengths of 290-320 nanometers) is the most common carcinogenic agent and is the cause of a large proportion of these malignancies (Gordon and Silverstone, 1976).

The importance of UV radiation as a major cause of skin cancer is well-established for the common types of skin cancer—basal cell and squamous cell carcinomas. However, the role of UV radiation in the development of the least common but most serious skin cancer—malignant melanoma—is uncertain. In addition to sunlight, skin cancers are caused by several other factors, including x-ray radiation, burns, arsenic, tars, mineral oils, and chemicals. Indeed, skin cancer has a repertoire of the largest number of identified etiologic factors of any human cancer (Helm, 1979).

TYPES OF SKIN CANCER

The structure of the human skin is diagramed in Fig. 1. A fraction of the solar radiation striking the surface of the skin penetrates into the epidermal and dermal layers. Melanin, which is normally present only in the epidermis, acts as a neutral filter to absorb UV, limiting the radiation reaching the dermis. A theory for the cause of skin cancers by UV radiation is that the radiation damages DNA molecules in the skin, a fraction of which are then misrepaired; the damaged cells become carcinogenic, and invade neighboring tissue (Parrish, White, and Pathak, 1979). Another theory postulates that the UV radiation also changes the body's immune response, greatly reducing its ability to reject tumors (Kripke and Fisher, 1978).

UV-caused types of skin cancers occur almost entirely among lighter-pigmented races and ethnic groups, especially persons of Celtic ancestry. They are essentially unknown among blacks, and rarely occur in other dark-skinned populations. Melanin, produced by the melanocyte
cells, determines the pigmentation of the skin; and the extreme differences in the incidence of skin cancers for these groups is consistent with the theory of UV-caused DNA damage.

The skin is made up of two basic layers—the epidermis and the dermis—separated by the basement membrane or dermoeipidermal junction. Most skin cancers arise in the epidermis and become dangerous when they invade the dermis or structures located beneath the skin, or when they metastasize and spread to other organs.

Cancers of the skin are broadly classified into three major groups and numerous subgroups, according to their cellular and microscopic structure.
Basal cell carcinoma (also called basal cell epithelioma and rodent ulcer, Jacob's ulcer, and Kronpecher's tumor) accounts for about 76 percent of all skin cancer. Most tumors occur on head and neck, but often on the trunk as well. Six or seven major subtypes of BCC are generally distinguished in various early forms: small, translucent, rounded areas; a pearly plaque; a small ulcer; a pigmented nodule. Unlike normal basal cells, most BCC cells are unable to synthesize keratin, a fibrous protein. Most BCC tumors are typically smooth in appearance and texture; as they enlarge they tend to ulcerate.

Typically, BCC is a slow-growing tumor, requiring ten years or more before becoming invasive. The major risk is that, in a small proportion of cases, untreated BCC invades the subcutaneous layer of the skin and attacks cartilage, bone, and other structures, causing extensive damage, disfigurement, and possible death. Metastasis is almost unknown.

Squamous cell carcinoma (also called epidermoid carcinoma and spinocellular carcinoma) accounts for some 19 percent of skin cancers. Most SCC occurs at sites exposed to the sun. The rate of incidence rises rapidly after age 55.

At the cellular level, SCC are atypical keratinocytes that arise in the epidermis, frequently as precancerous lesions (solar keratoses). The tumor cells vary from large, well-differentiated cells to anaplastic cells with no cytological evidence of their origin (Sanderson and Mackie, 1979).

So long as the tumor remains within the epidermal layer it is termed in situ SCC (intraepidermal SCC). A tumor that has penetrated the basement membrane of the epidermis is termed invasive SCC. In most cases it develops either from an in situ SCC or from a premalignant skin lesion. Its natural history is variable, but SCC generally develops faster than BCC. Adenoid SCC is a histologically distinct type of invasive SCC that appears primarily on the head and neck. SCC of the lip is another type of invasive SCC, which occurs overwhelmingly on the lower lip. It tends to grow rapidly and has a greater frequency of metastasis. The term de novo SCC is used to denote invasive SCC that develops in the absence of a preceding skin lesion.
The major danger from SCC is metastasis of the tumor to distant regions of the body and the consequent threat of uncontrollable malignancy and death.

*Malignant melanoma* is the rarest but most deadly of the three major types of skin cancers; it accounts for about 5 percent of all malignant skin tumors and has been classified as a separate cancer since 1948. The incidence of melanoma has risen rapidly; there has been an equally impressive improvement in overall survival (Sober et al., 1979, p. 630). These tumors originate from melanocytes by a process that is not yet understood. They grow quite rapidly as darkly pigmented macules, patches, plaques, papules, or nodules, which may ulcerate and often show evidence of inflammation.

Three subtypes of malignant melanoma are histologically distinguished by their growth pattern—lentigo-maligna melanoma (14 percent), superficial spreading melanoma (56 percent), and nodular melanoma (30 percent) (Balch, Soong, and Shaw, 1979). Risk of death increases with the depth of invasion of the tumor, which is classified into five levels of thickness.

*Lentigo-maligna melanoma*, the most benign of the three types, is correlated with exposure to sunlight, but the role of UV in causing melanomas is unclear and disputed.

*Superficial spreading melanoma* spreads laterally for one to seven years before developing nodules. The tumors have a haphazard coloration.

*Nodular melanoma* develops de novo as an invasive nodule without lateral spreading.

In addition to BCC, SCC, and MM tumors, several types of premalignant skin tumors are recognized. Clinically, these lesions may resemble malignant tumors, and some fraction of them do progress to a malignant stage.

*Solar keratosis* (also called actinic keratosis, senile keratosis, or Freudenthal's keratosis senilis) is a scaly, round, or irregularly shaped gray to deep brown lesion that is prevalent in older people (average age 62) in skin that is exposed to the sun, principally the backs of the hands and forearms, and the face. Some solar keratoses disappear spontaneously.
Bowen's disease can resemble solar keratosis and psoriasis clinically. Arsenic and sunlight are important causes.

INCIDENCE AND RISKS OF SKIN CANCER

The incidence of nonmelanoma and melanoma skin cancers for the U.S. white population, by age and sex, is show in Table 1.

Skin cancers are a disease of older age. BCC and SCC are rare before age 25, and incidence rates climb steeply after age 50. Men contract BCC and SCC about twice as often as women. Melanoma is only somewhat more frequent at older ages and affects men and women almost equally.

Most skin cancers are curable with prompt diagnosis and outpatient medical procedures. The major types of prompt treatment include surgical excision of the tumor, cauterization with an electrode and removal with a curette, freezing with liquid nitrogen, and x-ray radiation. A small proportion of tumors develop into serious and sometimes life-threatening conditions. Invasive tumors require extensive treatment with surgery or radiation.

If left untreated, a fraction of BCC tumors become so invasive as to aggressively attack cartilage, bone, eye, and other vital structures, causing severe destruction and, rarely, death.

Some SCC tumors invade the dermal layer of the skin. They may then metastasize to the lymph nodes or other locations, causing the initial cancer to spread to distant parts of the body, often with a fatal prognosis.

The rare MM tumor frequently metastasizes and spreads rapidly. Unless treated promptly, its prognosis is grave.

Deaths attributed to skin cancer are primarily due to malignant melanoma (Table 2).
Table 1
ANNUAL INCIDENCE RATES OF SKIN CANCER AMONG SELECTED U.S.
WHITE POPULATIONS BY AGE, SEX, AND TYPE
(Per 100,000 persons)

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aNonmelanoma rates are based on data for Seattle (King County only), Minneapolis-St. Paul SMSA, Detroit SMSA, the state of Utah, San Francisco-Oakland SMSA, Atlanta SMSA, the metro area of New Orleans, and the state of New Mexico.

bMelanoma rates are based on data from the SEER reporting areas of Connecticut, Detroit, Iowa, Atlanta, New Orleans, New Mexico, Utah, Seattle-Puget Sound, San Francisco-Oakland, and Hawaii.

Dashes indicate no cases of melanoma occurred in this age group during 1973-1977.
Table 2

ANNUAL DEATH RATES DUE TO SKIN CANCER AMONG U.S. WHITES DURING 1979 AND 1950-1969, BY AGE AND SEX
(Per 100,000 persons)

| Age (Years) | Nonmelanoma | | | Melanoma | |
|-------------|-------------|-------------|-------------|-------------|
|              | 1979 | 1969 | 1979 | 1969 |
| 0-4         | 0.0  | 0.0  | 0.0  | 0.0  | 0.0  | 0.0  |
| 5-9         | --  | 0.0  | --  | 0.0  | --  | 0.0  |
| 10-14       | --  | 0.0  | --  | 0.0  | --  | 0.0  |
| 15-19       | --  | 0.0  | --  | 0.0  | 0.1 | 0.2  |
| 20-24       | --  | 0.0  | 0.0  | 0.0  | 0.4 | 0.4  |
| 25-29       | 0.0  | 0.1  | 0.1  | 0.0  | 1.0 | 0.8  |
| 30-34       | --  | 0.1  | 0.0  | 0.1  | 1.9 | 1.3  |
| 35-39       | 0.1  | 0.2  | 0.1  | 0.1  | 2.6 | 1.7  |
| 40-44       | 0.2  | 0.4  | 0.1  | 0.2  | 3.4 | 2.0  |
| 45-49       | 0.5  | 0.7  | 0.1  | 0.3  | 4.8 | 2.4  |
| 50-54       | 1.0  | 1.3  | 0.4  | 0.7  | 5.7 | 2.9  |
| 55-59       | 1.5  | 2.3  | 0.3  | 0.9  | 6.5 | 3.2  |
| 60-64       | 2.0  | 3.5  | 0.9  | 1.3  | 7.9 | 3.7  |
| 65-69       | 3.3  | 5.0  | 1.0  | 2.0  | 8.3 | 4.3  |
| 70-74       | 5.3  | 8.2  | 1.5  | 3.4  | 10.6 | 5.1 |
| 75-79       | 7.7  | 17.5 | 2.9  | 8.1  | 12.5 | 6.8 |
| 80-84       | 11.9 | 17.5 | 3.6  | 8.1  | 14.2 | 6.8 |
| 85 and over | 21.7 | 46.8 | 9.4  | 28.4 | 15.7 | 8.9 |


a Rates for nonmelanoma include all deaths coded as Other Malignant Neoplasms of Skin (category number 173) under the Ninth Revision International Classification of Diseases.

b Rates for melanoma include all deaths coded as Melanoma of Skin (category number 172). Dashes indicate that no deaths due to indicated skin cancer occurred in this age group during 1979.
II. DOSE-RESPONSE MODELS OF SKIN CANCER AND ULTRAVIOLET RADIATION

The quantitative effect of solar ultraviolet radiation in causing nonmelanoma skin cancer is analyzed by means of a dose-response function. The customary dose-response model postulates that the relevant dose of UV radiation received by a subject is made up of two factors: the spectral response of the body to the wavelength of radiation, and the quantity of radiation of that wavelength reaching the earth's surface. The product of these factors, integrated over the wavelengths to which the skin is sensitive, determines the dose for a specified period of time.

Recent research suggests that the spectral response function (the "action spectrum") relevant to skin cancer is that for DNA, on the assumption that the biological mechanism causing skin cancers is an alteration of DNA by the impact of UV radiation. However, most dose-response calculations have used either the erythema response function measured by the Robertson-Berger meter, or the Coblentz-Stair response function.

The amount of radiation reaching the earth at a given location is given by the spectral irradiance function. The amount is affected by the concentration of ozone, the air mass, scattering of radiation by atmospheric particles, absorption by clouds, and reflection from surface materials. These factors vary with the wavelength, the sun angle, the thickness of the ozone layer, the aerosol thickness, the ground albedo, the altitude, the cloud cover and thickness, and possibly other atmospheric variables (Green and Hedinger, 1978). Because these factors are multiplicative, small errors in their determination can substantially alter the calculated dose (Scott and Straf, 1977).

For analysis of human skin cancer, this model requires some extensions. The dose actually received by an individual depends on his or her exposure to the UV wavelengths reaching the earth. Behavioral differences will cause significant variation in the actual doses received by different people. Key variables will be occupation, leisure
and vacation activities, protective clothing and use of sunscreen agents, and changes in residence over time.

The response of a population exposed to UV radiation is the rate of incidence of new skin cancers in a given period of time. Response has been found to depend on age, sex, skin type, and other indicators of sensitivity. In general, the response may depend on the lifetime history of the person's exposure.

Figure 2 summarizes the key ingredients of a dose-exposure-response model.

ESTIMATES OF DOSE-RESPONSE MODELS

Evidence suggesting UV as a systematic cause of skin cancers has been compiled in several studies that relate latitude to skin cancer incidence in various areas of the world. In the spectral irradiance function the amount of UV reaching the earth varies inversely (and nonlinearly) with latitude; latitude thus serves as an approximate measure of UV.

Surveys of skin cancer incidence usually measure numbers of skin cancers per 100,000 persons, by age category and sex. Those studies provide qualitative indications of the importance of UV radiation. For

\[
solar\ radiation \quad \downarrow\quad \downarrow\quad \downarrow\quad \downarrow
\]

\[
dose = \int_{290}^{320} \text{spectral response} \times \text{spectral irradiance} \, d\lambda
\]

\[
exposure = f(dose,\ behavior) \quad \downarrow
\]

\[
response = \int_0^{age} g(exposure,\ skin\ type) \, dt
\]

Fig. 2 -- Dose-exposure-response model of skin cancer
example, Auerbach (1961) finds that the incidence of skin cancer for the U.S. white population doubles for each 3° 48' of latitude (265 miles) to the south. Gordon and Silverstone (1976) tabulate an increasing global incidence of melanoma for predominantly white populations located nearer the equator.

Two special U.S. surveys, in 1971-72 and 1977-78, have provided data on both UV-B measurements and skin cancer incidence. In each survey, UV-B was continuously measured by Robertson-Berger meters, providing data from which total UV-B doses were calculated.

The 1971-72 survey included four locations for a six-month period, as part of the Third National Cancer Survey. The 1977-78 survey used the same protocol and covered eight locations for 12 months.

The more recent survey covered areas in Seattle, Minneapolis-St. Paul, Detroit, San Francisco-Oakland, Atlanta, New Orleans, and the states of Utah and New Mexico. These locations recorded annual UV-B meter readings ranging from 101 to 197. From the records of all physicians in these areas who might treat skin cancer, a total of 31,758 patients were identified as cases of nonmelanoma skin cancer newly diagnosed during the one-year survey period.

These key data have provided the basis for several attempts to statistically quantify the dose-response relationship, first for the 1971-72 survey and more recently for the 1977-78 survey. We briefly summarize the findings from the most recent studies.

Fears and Scotto (1983) have estimated two alternative functional forms relating response R (BCC and SCC skin cancer incidence) to dose D (UV-B meter counts):

- exponential form: \( R = a e^{bD} \) or \( \ln R = \ln a + bD \) \hspace{1cm} (2.1)

- power function form: \( R = a D^b \) or \( \ln R = \ln a + b \ln D \) \hspace{1cm} (2.2)

Each of these functions implies that an increase, dD, in the UV-B dose, will have a "biologically amplified" effect on the response, dR. The magnitude of the response is determined by the values of the parameters a and b. This amplification factor, expressed in terms of percentage changes, is:
\[ A = \frac{(dR/R)}{(dD/D)} \] (2.3)

Fears and Scotto estimated several variants of the two statistical dose-response functions, variously accounting for age, sex, and type of tumor.

Rundel (1983), based on earlier work in Rundel and Nachtwey (1978), postulates that the rate of skin cancer incidence is due to promotional effects of UV-B, that a tumor's growth is proportional to its current size, and that the growth rate increases because of exposure to UV-B. The model assumes reciprocity in the dose-time relationship—that a given dose has the same effect independent of the rate at which it has been administered—so that cumulative exposure is proportional to age. From these assumptions Rundel derives a dose-response model having a log-normal distribution of time to first-tumor incidence. This model yields a linear relationship between the mean of the log-normal distribution, \(1/t_m\), and the dose \(D\):

\[ \frac{1}{t_m} = a + bD \] (2.4)

Rundel fits the log-normal model separately for each site, sex, and tumor type and then estimates a one-parameter relationship between the mean and variance of the distribution across sites. The amplification factors implied by these models are summarized in Table 3 for three levels of UV-B. Each model implies that a given percentage increase in UV-B will indeed have amplified effects on the incidence of nonmelanoma skin cancers ranging from 1.0 to 2.8 times the change in UV-B.

In the range of the observations for the United States, the general conclusions are not strongly sensitive to the functional form used, so long as there is only a moderate change in UV level. However, for large changes or for long-range effects, it is important to determine the correct functional form (Scott and Sträf, 1977). Unfortunately, the currently available statistical and experimental data are insufficient to do this.
Table 3

BIOLOGICAL AMPLIFICATION FACTORS FOR
DOSE-RESPONSE MODELS

<table>
<thead>
<tr>
<th>Model</th>
<th>Sex</th>
<th>Skin Cancer</th>
<th>UV Count</th>
<th>Amplification Factor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exponential²</td>
<td>Male</td>
<td>BCC + SCC</td>
<td>1.01</td>
<td>1.3</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1.5</td>
<td>1.9</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>2.0</td>
<td>2.6</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>BCC + SCC</td>
<td>1.01</td>
<td>1.1</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1.5</td>
<td>1.0</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>2.0</td>
<td>2.0</td>
</tr>
<tr>
<td>Power function²</td>
<td>Male</td>
<td>BCC + SCC</td>
<td>1.01</td>
<td>1.8</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1.5</td>
<td>1.8</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>2.0</td>
<td>1.8</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>BCC + SCC</td>
<td>1.01</td>
<td>1.4</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1.5</td>
<td>1.4</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>2.0</td>
<td>1.4</td>
</tr>
<tr>
<td>Log-normal³</td>
<td>Male</td>
<td>BCC</td>
<td>1.0</td>
<td>1.80</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1.5</td>
<td>2.07</td>
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<td></td>
<td></td>
<td></td>
<td>2.0</td>
<td>2.22</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>BCC</td>
<td>1.0</td>
<td>1.07</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1.5</td>
<td>1.33</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>2.0</td>
<td>1.51</td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td>SCC</td>
<td>1.0</td>
<td>2.42</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1.5</td>
<td>2.69</td>
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<td></td>
<td></td>
<td></td>
<td>2.0</td>
<td>2.79</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>SCC</td>
<td>1.0</td>
<td>1.57</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1.5</td>
<td>1.92</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>2.0</td>
<td>2.14</td>
</tr>
</tbody>
</table>

²Fears and Scotto, 1983.
³Rundel, 1983.

DISCUSSION

The two U.S. surveys, and the statistical analyses of the resulting data, have greatly improved the understanding of the dose-response relationship for skin cancer. This work has provided a quantitative basis from which to calculate projected effects of changes in the ozone layer, and these models underlie the preliminary cost model reported in Sec. IV. However, these data have considerable limitations. The dose
measurements, made with Robertson-Berger meters, are at best an approximation of the dose due to the spectral response function for DNA that is presumably the relevant action spectrum for skin cancer. Although a conversion factor has been used in at least one study (Green and Hedinger, 1978), it is considered unsatisfactory by Rundel (1983) because it does not account for absorption of UV-B by clouds. Also, although UV flux changes from year to year, dose measurements at one site for a single year have been assumed to be representative of the lifetime exposure of the population surveyed at that site.

Perhaps most important, the survey data that have been analyzed do not measure actual exposure. Variations in exposure due to occupation, personal activities, and preventive behavior, as well as changes in residence over an individual's lifetime, substantially exceed differences in dose across sites.
III. PROBABILITY TREES FOR COURSES AND TREATMENTS OF SKIN CANCER

The natural history of skin cancer and its treatment are quite varied. It is convenient to represent the courses of skin cancers by diagrams that show the frequency of possible disease and treatment outcomes. These probability trees show the probability that the incidence of a particular feature will progress to the next stage of the disease or type of treatment.

DATA CONSIDERATIONS

The probabilities reported here are approximate measures that are most applicable to a cross-section of the U.S. white population. The values themselves have been gleaned from the medical and epidemiological literature supplemented by judgments of practitioners. Actual probabilities will often vary by age and sex, and the trees reported here should not be considered a prognostic tool. Rather, their purpose is to provide a guide to the overall frequencies of different outcomes.

Several factors make these probability measures themselves subject to considerable uncertainty. Most skin cancers are treated in physicians' outpatient offices and clinics, and histologic diagnoses are not always made. As a result, data on skin cancers are not systematically recorded in registries, hospital charts, and pathology laboratory records. However, data for malignant melanoma, which is separately classified, are more comprehensive.

Skin cancer cases that have not been seen by a physician are not included in these records; all of the tabulated probabilities are conditional on the person having chosen to visit a doctor. This means that the total incidence of skin cancer is somewhat, perhaps substantially, higher than has been measured in surveys. Furthermore, changes in attitudes and behavior with respect to skin cancer could both increase the number of persons seeking care and lead to less exposure through preventive measures.
Clinical diagnosis of skin conditions is often subject to error that is only detected on careful histological examination of specimens. For example, some 20-25 percent of clinically diagnosed BCC is histologically determined to be SCC, and about 20 percent of clinically benign nevi submitted for histological examinations are found to be melanoma (Helm, 1979). Moreover, some skin cancers are not readily classified into a single category, and sometimes there is no sharp boundary between benign and malignant tumors.

For the most part, the BCC and SCC probability trees omit skin cancer outcomes that are not due to UV radiation. Data on detailed classifications of types of tumors, treatments, and treatment outcomes have been assembled from retrospective studies in the medical literature; they represent samples of experience from particular regions, clinical and hospital settings, and several population surveys. The limitations of these sources for accurately assessing skin cancer incidence and history should be kept in mind, particularly the uneven coverage that exists.

A fraction of patients present with more than one primary tumor. Some patients are found on examination to have internal cancers as well as a skin cancer. The frequencies of these other tumors appears to be higher for persons with one or more skin cancers (Graham and Helwig, 1966; Levine et al., 1985); however, these effects are not tabulated in the probability trees.

Finally, the occurrence of skin cancers and the course of the disease could be substantially altered by changes in behavior—by protective measures to avoid intense solar exposure and by early detection of suspicious skin lesions. The extent to which greater awareness of the risks of skin cancer may mitigate the disease consequences traced out in previous clinical records is an important topic, but one that lies beyond the scope of this Note.

The following subsections review the occurrence and treatment of BCC, SCC, and MM skin cancers. Malignant melanoma is included, although it has not been clearly established to be caused by UV, because of its serious consequences. The perspective is that of the frequency of occurrence of the major courses of disease and the alternative modalities of treatment.
BCC--NATURAL HISTORY

Basal cell carcinoma is the most common skin cancer, accounting for 76 percent of all malignant skin tumors, with perhaps 400,000 new cases each year in the United States (Scotto, Fears, and Fraumeni, 1983). Figure 3 shows the major courses of disease in the form of a probability tree.¹

This diagram begins with the diagnosis of BCC and is logically preceded by the probability that a person having BCC will seek medical care. Surveys in Australia have found that there are three unreported skin cancer cases for every four treated (Helm, 1973, p.3).

BCC can be treated successfully in some 89-98 percent of initial cases with good technique. However, some 2-8 percent of treated tumors recur, and perhaps 5 percent of those recur after a second treatment (Albright, 1982). Inadequate treatment markedly alters this pattern; about 50 percent of initial cases recur when inadequately treated (Swanbeck and Hillstrom, 1970) and a similar percentage recur a second time.

\[
\begin{array}{c}
\text{cancerous conditions} \\
\text{BCC} \rightarrow 2-8\% \rightarrow \text{recurrent} \rightarrow 5\% \rightarrow \text{rerecurrent} \rightarrow \text{?\% invasive BCC} \\
\text{?\% cure with disfigurement} \\
\text{?\% death} \\
\text{?\% death} \\
\text{invasive BCC} \\
\end{array}
\]

cure = no recurrence of disease after five years

Fig. 3 -- Probability tree for cutaneous BCC

¹Where no data are available from which to estimate a probability, a question mark is shown in the figures.
The major risk of BCC is the development of an invasive tumor, a process that can occur over 10-20 years in some 1-3 percent of all cases. Once BCC has invaded the subcutaneous tissue, if left untreated, it may spread deeply, attacking cartilage, bone, and nerve structures and cause extensive disfigurement, especially around the eye, nose, or ear. In this advanced stage of the disease, death can occur.

Metastasis of BCC to regional or distant sites is almost unknown (Paver et al., 1973). Most BCC occurs in the absence of detectible precursors.

**BCC--TREATMENT**

BCC can be effectively treated by surgical excision of the tumor, by chemosurgery (Mohs' surgery), electrosurgery, and radiation. The preferred modality varies with the size and location of the tumor, age and health of the patient, and skills of the physician. The principal types of treatment are summarized in Table 4.

Each method of treatment yields similar cure rates, some 92-98 percent, when applied to appropriate cases. Recurrent BCC can be effectively treated by excision, chemosurgery, or, rarely, electrosurgery. Other modalities are less satisfactory because they are unable to accurately determine the margins of the tumor. Cure rates for recurrent BCC are reported to be 95 percent (Albright, 1982).

Microscopically controlled chemosurgery (Mohs' surgery) provides excellent results in most cases but often demands greater time and skill than other methods. Two, three, or four sessions are sometimes required to completely remove the tumor, each separated by two hours to a day or more. Mohs' surgery has a reported recurrence rate of 1-3 percent (Albright, 1982).

Invasive BCC requires far more extensive treatment to repair and, if possible, restore damaged structures, as well as to remove the tumor. The necessary techniques depend very much on the particular case.
Table 4
TREATMENT FOR NONMELANOMA SKIN CANCERS

<table>
<thead>
<tr>
<th>Description</th>
<th>Appropriate for</th>
<th>Not Appropriate for</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Excision</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>The entire tumor and a margin of normal skin is surgically removed. The</td>
<td>Larger BCC/SCC</td>
<td>Certain recurrent BCC/SCC</td>
</tr>
<tr>
<td>surgical site is closed with stitches. The procedure may be followed with</td>
<td></td>
<td>Inoperable tumors</td>
</tr>
<tr>
<td>reconstructive (plastic) surgery to improve the appearance of the excised</td>
<td></td>
<td></td>
</tr>
<tr>
<td>area.</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Chemosurgery (Mohs' surgery)</strong></td>
<td>Indefinite margins</td>
<td>Inoperable tumors</td>
</tr>
<tr>
<td>Mohs' fresh-tissue technique requires special personnel, expertise, and</td>
<td>Recurrent BCC/SCC</td>
<td>Metastases</td>
</tr>
<tr>
<td>equipment. It permits incremental examination and removal of tumor in each</td>
<td></td>
<td></td>
</tr>
<tr>
<td>direction for more precise diagnosis and determination of extent of</td>
<td></td>
<td></td>
</tr>
<tr>
<td>malignancy. Reconstructive surgery is often required following use.</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Electrosurgery (Curettage and electrodesiccation)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heat is applied by an electric needle to coagulate the tissue, causing cell</td>
<td>Thick solar keratoses</td>
<td>BCC/SCC</td>
</tr>
<tr>
<td>death. The coagulated tissue is then removed with a curette.</td>
<td>Many BCC tumors</td>
<td>with indefinite margins</td>
</tr>
<tr>
<td>Follow-up is essential, as an accurate histology diagnosis cannot be</td>
<td>Smaller BCC/SCC</td>
<td>Certain recurrent BCC/SCC</td>
</tr>
<tr>
<td>obtained at the margins of the surgery.</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Radiation</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>X-ray beams are directed at the tumor. The total dose is administered in</td>
<td>BCC/SCC</td>
<td>Unclear margins</td>
</tr>
<tr>
<td>several fractions delivered over several days or weeks.</td>
<td>- on face</td>
<td>Invasion of bone, cartilage</td>
</tr>
<tr>
<td>Procedure provides no diagnostic data.</td>
<td>- in poor health</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- beyond age 40</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- when surgery is contraindicated</td>
<td></td>
</tr>
<tr>
<td><strong>Cryosurgery</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Liquid nitrogen is applied to the tumor, freezing it and causing cell</td>
<td>Solar keratosis, unless thick</td>
<td>Certain BCC/SCC</td>
</tr>
<tr>
<td>death.</td>
<td>Bowen's disease</td>
<td></td>
</tr>
<tr>
<td><strong>5-Fluorouracil (5-FU)</strong></td>
<td>Solar keratosis</td>
<td>BCC/SCC</td>
</tr>
<tr>
<td>A cytotoxic agent applied in an ointment or lotion that produces an</td>
<td>Some Bowen's disease</td>
<td></td>
</tr>
<tr>
<td>inflammatory reaction. Daily application by the patient for three to four</td>
<td></td>
<td></td>
</tr>
<tr>
<td>weeks is required. Frequently reveals and destroys solar keratoses not</td>
<td></td>
<td></td>
</tr>
<tr>
<td>clinically visible. Supervision is essential.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
SCC--NATURAL HISTORY

Squamous cell carcinoma of the skin accounts for about 19 percent of all skin cancers. There are perhaps 100,000 new cases each year in the United States (Scotto, Fears, and Fraumeni, 1983). The average incidence increases rapidly with age (see Table 1).

The exact mechanism by which any SCC arises, whether caused by UV or another factor, has not been established; most investigators propose a two- or multiple-stage process (Stoll, 1979, p. 364). For clinical purposes, the simplest assumption is that one epidermal cell has undergone a heritable change, causing it to propagate differently than normal cells (Pinkus, 1979, p. 356).

Figure 4 summarizes the major courses of the disease. Most SCC is preceded by some type of nonmalignant skin lesion. However, frequencies of precursors and the incidence of specific types of SCC have not been reported.

Solar keratosis (also called actinic keratosis) is a very widespread skin lesion, and one discussion suggests that in Australia almost the entire population of northern European extraction has one or more solar keratoses at some time in life (Gordon and Silverstone, 1976). These lesions are considered an approximate indicator of an individual's cumulative solar exposure (Green and O'Rourke, 1985).

For an estimated 12-25 percent of patients with one or more solar keratoses, one of the lesions will develop into an in situ SCC (a tumor located entirely within the epidermal layer of the skin) (Graham and Helwig, 1966; Montgomery, 1967) over a 10 or more year period (Sanderson and Mackie, 1979). However, SCC that develops from solar keratosis is the most benign type of squamous cell carcinoma; metastasis at this stage is almost unknown (Graham and Helwig, 1966; Lund, 1965). The major risk is that the in situ SCC will invade the dermal layer, a process that typically takes 1 to 3 years and occurs in some 12 percent of in situ SCC developed from solar keratosis (Bendl and Graham, 1968).

At least 98-99 percent of invasive SCC due to solar keratosis is cured by prompt treatment (Helm, 1979, p. 122). Rarely, a tumor may metastasize and spread cancer to other areas of the body, most commonly to the regional lymph nodes, after some 1 to 3 years (Levine, Ratz, and
Fig. 4 -- Probability tree for cutaneous SCC
Bailin, 1985). Once metastasis has occurred, prognosis and treatment depend on the locations and extent of spreading of the original tumor.

In situ SCC that is not associated with solar keratosis is more aggressive and likely to metastasize some 8 percent of the time (Levine, Ratz, and Bailin, 1985).

Bowen's disease or intraepidermal SCC is sometimes classified as a precursor to SCC. It is known to be caused by exposure to arsenic, and it is possible that all cases are due to arsenic (Pinkus, 1979, p. 357). Prompt treatment of Bowen's disease yields a cure rate of 95 percent or better, but the 2-5 percent of cases that become invasive proceed to metastasize an estimated 37 percent of the time (Graham and Helwig, 1966). The rate of metastasis is likely to be considerably lower for tumors arising at sun-damaged sites.

Adenoid SCC, a distinctive form of invasive SCC, metastasizes in about 2 percent of cases. It may be preceded by precursor lesions in a few cases (Johnson and Helwig, 1966).

De novo SCC is a rare, rapidly invasive SCC that presents with no apparent precursors (Stoll, 1979, p. 367). Some 8 percent (Levine, Ratz, and Bailin, 1985) to 18 percent (Graham and Helwig, 1966) of de novo tumors metastasize.

Lip cancer, a form of invasive SCC, may grow rapidly (Stoll, 1979). The frequency of metastasis is variously reported to be 5 percent (Traenkle, 1962), 10 percent (Ward and Hendrick, 1950, p. 2191), and as high as 37 percent (Martin, 1941).

**SCC--TREATMENT**

The types of treatment appropriate for SCC depend on the particular form of skin cancer and stage of development, tumor location on the body, age and health of patient, and other factors. The principal methods of treatment are excision or chemosurgery, electrosurgery, and radiation. Each of the techniques cures the cancer, provided the treatment encompasses the entire continuum of the tumor (Stoll, 1979, p. 372).
The alternative courses of treatment for cutaneous SCC are traced in the probability trees in Figs. 5-7. Recurrence rates have been reported for only a few types of SCC and treatment modalities; question marks in the figure indicate that published data for the indicated frequency are not available.

SCC that occurs with solar keratosis (Fig. 5) is treated by excision or chemosurgery (37 percent), electrosurgery (50 percent), or radiation (9 percent) (Bendl and Graham, 1970). Clinical series for surgical treatments indicate an 88 percent cure rate, with 30 percent of the cases undergoing reconstructive (plastic) surgery after one year of observation (Riefkohl, Pollack, and Georgiade, 1985). Recurrent cases are usually treated by chemosurgery.

---

**Fig. 5** -- Probability tree for treatment of SCC with solar keratosis
Local excision of Bowen's disease (Fig. 6) has achieved cure rates of 80 percent (Graham and Helwig, 1961) and topical application of 5-FU to small, well-defined lesions has achieved cure rates of 91-94 percent (Sturm, 1979). However, treatment by radiation or electrosurgery causes recurrence more than 75 percent of the time (Graham and Helwig, 1966). Some 50 percent of recurrences occur within 10 months of excision (Turk and Winder, 1980).
De novo SCC (Fig. 7) is treated surgically in 61 percent of cases, with electrosurgery (20 percent) and radiation (18 percent) used in other cases (Bendl and Graham, 1970). Cure rates for treatment of de novo SCC have not been separately reported. Rieffkohl, Pollack, and Georgiade (1985) have reported recurrence rates of some 12 percent for treatment by Mohs' surgery of SCC patients, 73 percent of whom have previously been treated by other methods.

Followup visits are important to detect possible recurrence of the treated tumor. Some 85 percent of recurrences are estimated to occur within two years, and followups are recommended for a three to five year period following treatment. Visits should be at two to three-month intervals for the first two years, and twice a year thereafter (Boysen et al., 1985).

---

**Fig. 7 -- Probability tree for treatment of de novo SCC**

- Frequency of treatment: excision or chemosurgery (Mohs' surgery) - 61%
- 20% electrosurgery (ED&C)
- 18% radiation (x-ray)
- Recurrence rates: 12% for those previously treated by other methods.
MELANOMA--NATURAL HISTORY

The incidence of malignant melanoma has been rising rapidly. For 1986, 23,000 new cases were predicted to occur, a rate of nine cases per 100,000 whites. Malignant melanoma today accounts for about 5 percent of all malignant skin tumors (Cancer Statistics, 1986).

Association with UV

The precise role of ultraviolet radiation in cutaneous malignant melanoma has not been determined (Schreiber, Moon, and Bozzo, 1984) but many factors indicate that UV does play some causative role (Kopf, Kripke, and Stern, 1984). The incidence of MM increases in areas closer to the equator (Green and Siskind, 1983), following periods of maximum sunspot activity and in summer months (Scotto and Nam, 1980). MM patients usually have a history of painful sunburning (Green et al., 1985) and poor tanning ability. Red and blond hair and tendency to freckle are important risk factors (Milton, Balch, and Shaw, 1985). A case-control study of MM finds that the presence of actinic tumors significantly increased the risk of melanoma (Green and O'Rourke, 1985). However, MM frequently occurs on areas of the body not exposed to the sun; it appears on the head and neck less than 25 percent of the time, whereas BCC and SCC occur predominantly in those areas. MM occurs frequently on the back, and on women's lower legs.

A majority of MM patients observe a pre-existing "mole" at the site of the melanoma. Melanomas have a variety of clinical appearances, but their common denominator is their changing nature. (Milton, Balch, and Shaw, 1985.)

There is a considerable risk of erroneous clinical diagnosis with melanoma, as with other skin cancers. Some 15 percent of benign tumors have been incorrectly diagnosed as MM (Watson, 1963). However, perhaps 20 percent of lesions that have been clinically diagnosed as benign nevi but are nevertheless submitted for histological examination are found to be malignant (Lee, in Helm, 1974, p. 151).
TYPES OF MALIGNANT MELANOMA

Cutaneous malignant melanoma is classified into four types, according to its noninvasive component. The probability tree of Fig. 8 summarizes the courses of this disease.

1. Superficial spreading melanoma (SSM) arises in melanoma in situ in 61 percent of the cases of one series (MacGovern and Murad, 1985), or in a pre-existing nevus over a one to five year period (Milton, Balch, and Shaw, 1985).

Fig. 8 -- Probability tree for cutaneous MM
2. About 5 percent of Hutchinson's melanotic freckle (lentigo maligna) develops into lentigo maligna melanoma (LMM) over a five to 40-year period (Milton, Balch, and Shaw, 1985). LMM begins as a tan, flat, freckle-like lesion that over time changes color, pattern, and size. It is more common in women; almost all tumors are located on the face or neck.

3. Nodular melanoma tumors usually begin de novo rather than from a pre-existing nevus (Milton, Balch, and Shaw, 1985) and have a shorter clinical onset. They are more aggressive than SSM and occur more frequently in men. Most nodular melanomas are raised or dome-shaped; 95 percent are pigmented.

4. Acro- and lentiginous or palmar-plantar-subungual melanoma is a fourth type of MM that involves the soles of the feet, the palms of the hands, and the subungual regions. It is unlikely to be associated with UV radiation and is not considered here.

Melanoma is classified according to the extent to which the tumor has spread. A three-level staging system has frequently been used to distinguish localized melanoma (stage I), nodal metastasis (stage II) or distant metastasis (stage III). This excludes in situ melanoma—tumors that have not become invasive. This staging system is the basis of the probability tree in Fig. 9. It summarizes the patterns of metastases in 850 cases of cutaneous melanoma treated at the University of Alabama in Birmingham over a 25-year period (Balch et al., 1983).

The majority of patients (82 percent) presented with localized (stage I) disease, and 70 percent of these survived free of disease after five years. After treatment, 15 percent of the patients developed nodal metastases some 16 months later; of these cases 25 percent survived and 75 percent progressed to distant metastases 12 months later. In all cases, stage III prognoses were very poor; 97 percent of those patients died within six months. As shown in the figure, some 15 percent of stage I cases developed directly into distant metastases after 34 months, with a similarly grave outcome.

However, a revised staging system has been adopted by the American Joint Committee on Cancer (see Balch and Milton, 1985).
survived = no recurrence of disease after five years.

SOURCE: C. M. Balch et al., 1983.
NOTE: Data from 850 cutaneous melanoma patients treated at the University of Alabama at Birmingham during a 25-year period. The presenting stage of the melanoma could not be determined from the data for 3 percent of the cases.

Fig. 9 -- Probability tree for treatment of MM

Twelve percent of the melanoma cases presented with nodal metastases, and 3 percent presented with distant metastases. The course of disease for these cases is shown in the lower sections of the figure. For 3 percent of the cases, the presenting stage could not be determined in the data series.
The prognosis for a melanoma patient depends on the site of the melanoma, thickness of the tumor, whether the tumor is ulcerated, and surgical treatment. These factors have been included in a multifactorial statistical model of proportional hazard for scoring melanoma cases and predicting outcome in patients with localized melanoma (Soong, 1985).

**MELANOMA--TREATMENT**

Primary melanoma is diagnosed by biopsy. Small tumors are removed by excision, larger tumors may require an incisional biopsy. Definitive surgery requires complete removal with adequate surgical margins of normal skin. A skin graft may be required.

If malignant melanoma metastasizes, it usually does so first to the regional lymph nodes that drain the site of the primary tumor. If the lymph nodes are palpable and no other cause is present, the nodes are removed by a lymphadenectomy. Some surgeons will dissect the lymph nodes as a prophylactic operation in the absence of evidence of metastasis, but opinion is divided as to the desirability of this procedure when the tumor is thin. Removal of the lymph nodes is major surgery that requires hospitalization.

Immunotherapy and chemotherapy have been used to treat advanced cases of MM. These methods are considered experimental. Surgery, drug therapy, and radiation may be used as palliative methods to retard the progress of metastatic disease.

The overall risk of a local recurrence of melanoma is about 3 percent. Thin tumors (less than 0.76 mm) rarely recur, whereas tumors thicker than 4 mm recur locally in some 13 percent of cases (Urist, Balch, and Milton, 1985). Followups at two-month intervals are recommended for two-three years for some tumors (Roses, 1985).
IV. OUTCOME PROBABILITIES AND EXPECTED COSTS

A probability tree consists of nodes, branches, and leaves. At each node there are two or more branches, indicating different possible outcomes, given the condition that characterizes the node. A probability is associated with each branch indicating the proportion of cases at that node that have the indicated outcome. At the final nodes the alternative outcomes are termed leaves; they are the end result of one possible course of disease or treatment.

The likelihood of any particular ultimate outcome—one leaf—can be calculated from the conditional probabilities of the branches that lead to it. Similarly, the expected cost of treatment for a particular outcome can be calculated from the conditional probabilities and the costs of treatment at each node.

The form of these calculations can be illustrated using the first part of the probability tree for BCC in Fig. 3. Assume that at the first BCC node the probabilities of the three branches are 97 percent for a cure, 2 percent for a recurrence, and 1 percent for progression to invasive BCC.

The probability of the top leaf in the figure—cure after initial treatment—is 0.97. The probability of the bottom leaf in this tree—death from invasive BCC after treatment without recurrence—is the product of 0.01 times an (unknown) conditional probability of death from invasive BCC, given that the original BCC was treated without recurrence.

The likelihood of the intermediate leaves can be calculated in a similar fashion. For example, the probability of the second-to-top leaf in the figure—cure after treatment for one recurrence—is $0.02 \times 0.95 = 0.019$.

The expected cost at a leaf is obtained by calculating the expected treatment cost at each node from a similar probability tree that records the probabilities of alternative treatments. First, the cost of each type of treatment, including followup procedures, is weighted by the frequency of that treatment. This expected cost is then discounted to
account for the occurrence of these expenditures at a future date. The
discounted expected costs of treatment at each node leading to the leaf
are summed and multiplied by the probability of the leaf.

MODEL OF HEALTH CARE COSTS

The health care costs of skin cancer caused by UV radiation can be
estimated by combining information from dose-response models in Sec. II,
the probability trees of disease and treatment outcomes in Sec. III, and
data on costs of treatment. In this section we develop a prototype
model for simulating changes in these health care costs due to changes
in levels of UV radiation.

There are major uncertainties in several components of the model.
Data on probabilities and costs are incomplete, and the reliability of
the available dose-response functions has not been established for
higher dose levels. The calculations reported here are not forecasts.
Rather, they illustrate a methodology that can be used to analyze the
effect on costs of changes in UV levels, changes in treatment outcomes
and costs, and, potentially, changes in exposure to UV radiation.
Future studies may lead to greater understanding of these factors and an
improved ability to analyze health risks and costs.

CONCEPTUAL OVERVIEW OF THE MODEL

To model the health care costs of skin cancer caused by ultraviolet
radiation, we begin with the dose-response models discussed in Sec. II.
Substituting values for UV dose into the equations in that section
yields annual incidence rates, which are then multiplied by population
values to obtain the annual numbers of new skin cancer cases for the
population. To calculate the costs due to this rate of incidence we
will multiply by the cost of treating a single new case. To analyze the
costs of skin cancer for several different future years, we will account
for possible changes in population, dose, and cost. Our analysis
assumes that the underlying dose-response relationship will not change,
even if we do not characterize it completely in the dose-response
equations.
Figure 10 depicts the relationship among the various components of the model.

MODEL IMPLEMENTATION

Because the model of health costs is deterministic for given levels of UV, population, and treatment cost per case, an electronic spreadsheet can be used to combine the components of the model as a series of formulas. The spreadsheet allows a user to substitute different values for the various parameters and, by sensitivity analyses, easily examine the effects of these changes on both numbers of new cases and total costs of skin cancer due to ultraviolet radiation.

The model is also readily extended to include additional structural features and other types of data, when available, by straightforward programming in the spreadsheet language.

MODEL COMPONENTS AND DATA SOURCES

This analysis employed Version 2 of the 1-2-3 spreadsheet program by Lotus Development Corporation. The Lotus worksheet consists of four primary components:

1. Estimates of UV radiation levels by state.
2. Estimates of white population, by age and sex, by state.
4. Estimates of costs of treatment by region.

These four components are linked in the worksheet to obtain estimates of skin cancer incidence (both BCC and SCC) and total cost of treatment. A more detailed description of the worksheet is included in the appendix.
Baseline UV Radiation as a Function of Location

Baseline levels of UV radiation were calculated for U.S. locations from the following equation, which uses coefficient estimates and mean values (to compute the intercept) taken from Scotto, Fears, and Gori (1975).

\[ D = 362.022523 + 0.0067(ALT) - 3.38357(LAT) - 17.4299(SKY) \]

where

- \( D \) is the annual integral of UVB counts \((10^{-4})\) measured by a Robertson-Berger meter. This is the dose variable for the dose-response model.
- \( ALT \) is the altitude above sea-level in feet.
- \( LAT \) is the latitude in degrees north of the equator.
- \( SKY \) is the mean daily sky cover.

ALT, LAT and SKY values were taken from Local Climatological Data, Annual Summaries for 1983. These data come from the National Oceanic and Atmospheric Administration; they are long-run (usually 10-year) averages, not data limited to 1983.
UV estimates were calculated for as many Metropolitan Statistical Areas (MSA) as possible for each state. In each state these estimates were combined to estimate a single UV count by computing weighted averages of UV counts for MSAs, where the weights used were population estimates taken from Current Population Reports.\(^1\) Where MSAs crossed over state boundaries, the population weight used was the MSA population for only those counties within the given state.

To allow for the possibility of changing UV levels over time (as well as errors in our baseline estimates), these baseline values for the UV level of each state can be multiplied by a change factor (initially set to 1). Varying the change factor allows for a sensitivity analysis of the effects of UV on both number and cost of skin cancers.

**Population**

Estimates of the white population of each state, by age and sex, were taken from the State Projections Tape of the Bureau of the Census. The age cohorts used were:

25-34, 35-44, 45-54, 55-64, 65-74, 75-84, 85 and older.

This data source groups persons over 65 years old into one cohort. Therefore, national projections of population by age, sex, and race were used to determine the proportion of the population over age 65 in each of the last three cohorts used in this analysis.

National projections were taken from Current Population Reports.\(^2\) For each of the projection years (1980, 1990, 2000) the national proportion of persons over 65 (by sex) in each of the last three age cohorts (65-74, 75-84, and 85+) was multiplied by the projected state population over 65 to estimate these three cohorts by state.

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\(^1\) Series P-25, Number 976 of the Bureau of the Census.
\(^2\) Series P-25, Number 952 of the Bureau of the Census.
Cancer Incidence Rates

Regression coefficients to predict cancer incidence rates were reestimated from survey data reported by Fears and Scotto (1983) to obtain separate dose-response equations for BCC and SCC. For each sex, two types of equations can be estimated—an exponential model and a power model. The exponential model has the following form:

\[ \ln(R) = C_i + b(D). \]

The power model has the form:

\[ \ln(R) = C_i + \ln(D), \]

where

- \( R \) is the number of new cases of skin cancer each year,
- \( C_i \) is the intercept for a particular age-cohort,
- \( D \) is defined as above.

These equations were estimated separately for males and females and for basal cell and squamous cell cases. In each regression, the variables were weighted by the actual number of cases observed to correct for heteroscedasticity. These estimates are essentially those obtained by Fears and Scotto, except that they are disaggregated to account for different parameters by type of skin cancer. The data came from the 1977-78 survey covering Seattle, Minneapolis-St. Paul, Detroit, San Francisco-Oakland, Atlanta, New Orleans, and the states of Utah and New Mexico (see Sec. II).

COSTS OF TREATMENT

Medical Costs

The costs of treating skin cancer were developed from two major primary sources. Medicare claims records that had been previously processed for another RAND project (Chassin et al., 1986) provided cost data for some skin cancer procedures. The fact that the incidence of
skin cancer rises rapidly with age and that the Medicare program covers nearly the entire U.S. population over age 65 makes these data especially useful for this study.

The dataset covers all physician claims for the Medicare part B insurance carriers in 13 regions of eight states (Arkansas, Colorado, Iowa, Massachusetts, Montana, Pennsylvania, South Carolina, and northern California) in 1981, a total of 75 million claims. These claims had already been edited and aggregated into major procedure groups for the earlier project. For this study, average 1985 billed costs in the 13 regions were calculated for four of the procedure groups:

- excision of benign skin lesion $ 95
- excision of malignant skin lesion $ 217
- destruction of benign skin lesion $ 48
- skin biopsy $ 54

These costs per procedure were derived from cost and frequency of procedure data for the 13 regions. To compute the cost of treatment, the average costs measured for each of the 13 regions were first deflated to comparable base year (1977) costs by Indices of Professional Medical Services for All Urban Consumers\(^3\) for four regions--North East, North Central, South, and West. These 13 average costs were then combined into a single (unweighted) estimate of the U.S. average cost for each of the four procedure groups for the base year. To obtain cost figures in 1985 dollars, the base year costs were then adjusted by the Index of Physicians Services for All Urban Consumers.\(^4\)

The second source of data is the RAND Health Insurance Study, a large research study that enrolled 7700 persons in three- to five-year experimental health insurance plans during 1975-82 in six sites (Newhouse et al., 1981). The available outpatient data from this study are for physician claims coded by diagnosis rather than procedure; therefore they include all procedures performed for the case. Because of the low incidence of skin cancer in the general population, even this

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\(^3\) Bureau of Labor Statistics, Dialog File 175.

very large research dataset includes only 232 skin cancer cases. Average billed charges for entire period of enrollment (including multiple visits for the same diagnosis) by diagnostic category were:

- benign skin lesion (ICDA 216-217) $ 96
- precancerous lesion (ICDA 702-703) $ 134
- nonmelanoma skin malignancy (ICDA 173) $ 208
- excluding 173.1, 173.2, 173.5 and 173.9
- other skin neoplasm (ICDA 232.2) $ 95

Estimated Cost of BCC

The average cost per incidence of BCC or SCC can, in principle, be developed from the probability trees for the disease and its treatment outcomes, as described in Sec. II. Because we lack data for some branches of the trees and some procedures, we constructed approximate costs per type of cancer using just the major branches of the trees.

Using the first part of the probability tree for BCC in Fig. 3 we assumed that at the first BCC node, the probabilities of the three branches are 95 percent for a cure, 4 percent for a recurrence, and 1 percent for progression to invasive BCC after 15 years. Second treatment was assumed effective in 95 percent of cases, with 5 percent progressing to invasive BCC after 10 years.

We assumed initial treatment costs consisted of procedures for excision ($217), biopsy ($54), and four followup visits (4 x $25), for a total cost associated with initial treatment of $371. Treatment costs for recurrence were assumed to be the same. Invasive BCC can involve hospitalization and extensive, repeated surgery. We arbitrarily assumed a total treatment cost of $2000, discounting future costs at a rate of 5 percent per year.

These values combine to give an estimated average cost for the incidence of BCC of $397, which can be compared with the per-diagnosis costs found in the RAND Health Insurance study. The estimated cost is obtained as follows:
.95 \times \$371 \quad \text{cure after first treatment}
+ .04 \times .95 \times \$742 \quad \text{cure after second treatment}
+ .04 \times .05 \times (\$742 + D10 \times \$2000) \quad \text{invasive BCC after second treatment}
+ .01 \times (\$371 + D15 \times \$2000) \quad \text{invasive BCC after first treatment}

\text{where:} \quad D15 = .461
D10 = .614

\text{15-year discount factor at 5 percent}
\text{10-year discount factor at 5 percent}

\textbf{Estimated Cost of SCC}

The procedure for SCC was similar. Using the first part of the probability tree for SCC in Fig. 4 we assumed that at the first SCC node the probabilities of the three branches are 88 percent for a cure and 12 percent for progression to invasive SCC in two years. Treatment for invasive SCC was assumed effective in 99 percent of cases, with 1 percent metastasizing after an additional two years.

We assumed initial treatment costs consisted of excision ($217), biopsy ($54), and four followup visits (4 \times $25) for a total cost associated with initial treatment of $371. Treatment costs for invasive SCC were assumed to be twice as large, reflecting more complex procedures and more extensive followup. Metastatic cancer requires hospitalization and considerable surgery or radiation treatment. We arbitrarily assumed a total treatment cost of $2000, discounting future costs at a rate of 5 percent per year.

These values combine to give an average cost for the incidence of SCC of $454, which is obtained as follows:

.88 \times \$371 \quad \text{cure after treatment for in situ SCC}
+ .12 \times .99 \times (\$371 + (D2 \times \$742)) \quad \text{cure after treatment for invasive SCC}
+ .12 \times .01 \times (\$371 + (D2 \times \$742) + (D4 \times \$2000)) \quad \text{metastatic SCC}

\text{where:} \quad D2 = .907 \quad \text{two-year discount factor at 5 percent}
D4 = .823 \quad \text{four-year discount factor at 5 percent}.
RESULTS

The spreadsheet model provides a report of estimates of the number of new cases of BCC and SCC, by state, for the base population in 1980 and for the projected 1990 and 2000 populations. The baseline case assumes UV levels predicted by the UV radiation equation, and simulation of increased doses is accomplished by increasing those values by specified percentages. Treatment costs are measured in constant 1985 dollars, using the per-incidence costs calculated above. The estimates for future years do not include possible increases in skin cancer due to factors other than changes in UV-B radiation.

Tables 5, 6, and 7 report the number of new cases of skin cancer expected in the 1980 white population, the rate of incidence per 100,000 persons, and health care costs in 1985 dollars. About 440,000 cases of BCC and 103,000 cases of SCC are projected by the dose-response functions, implying total costs of $221 million.

The dose-response equations used in the model are implicitly projections of the long-term change in skin cancer incidence due to changed UV levels. The spreadsheet calculations should therefore be interpreted as measures of the eventual effect of higher UV levels; they are not satisfactory indicators of the year-to-year incidence of skin cancer that would occur because of greater UV levels.

Although the increase in skin cancers would only develop over the lifetimes of the exposed population, summary measures of the eventual effect can be obtained from comparisons to the baseline case. Tables 8 and 9 provide estimates of the number of cases and total costs for a 10 percent increase in UV levels. Total cases of BCC increase 14 percent and cases of SCC increase 26 percent; total costs rise by $36 million.

The parameters of the spreadsheet model can be readily changed to calculate the effects of different assumptions about UV levels and costs of treatment, for different projected populations. Other factors can be accounted for by extending the structure of the basic model. For example, an increase in preventive activities could be assumed to change the coefficients of the dose-response equations and to add an annual cost for sunscreen preparations for an assumed fraction of the population. Greater awareness of the risks of skin cancer could
increase the frequency of office visits and alter the probabilities used to calculate the average cost per BCC or SCC case. The appendix contains a guide to the model and its major components.
Table 5  
TOTAL NUMBER OF NEW CASES

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Table 6
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<td>241</td>
<td>1556</td>
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<td>2279</td>
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<td>9548</td>
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<td>141</td>
<td>689</td>
<td>560</td>
<td>144</td>
<td>704</td>
</tr>
<tr>
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<td>1231</td>
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<tr>
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<td>382</td>
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<td>1135</td>
<td>380</td>
<td>1513</td>
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<tr>
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<td>99</td>
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<td>53</td>
<td>316</td>
<td>251</td>
<td>46</td>
<td>298</td>
</tr>
<tr>
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<td>2323</td>
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<td>321</td>
<td>2146</td>
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<tr>
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<td>362</td>
<td>1813</td>
<td>1482</td>
<td>368</td>
<td>1850</td>
</tr>
<tr>
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<td>121</td>
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<td>765</td>
<td>4001</td>
<td>3274</td>
<td>757</td>
<td>4030</td>
</tr>
<tr>
<td>WY</td>
<td>165</td>
<td>379</td>
<td>117</td>
<td>497</td>
<td>382</td>
<td>120</td>
<td>502</td>
</tr>
</tbody>
</table>

Total U.S. 198779 59096 257875 197665 58398 256063
V. CONCLUSIONS

This Note has calculated illustrative estimates of the direct health care costs of treating skin cancer caused by UV radiation by combining information from dose-response models, the probability trees of disease outcomes and treatment alternatives, and data on costs of treatment. The prototype computer model that was developed can simulate the effects of hypothetical changes in UV levels. Because many components of the model are uncertain and based on incomplete data, the model is illustrative only; it should not be used for forecasting changes in health care costs.

The computer model does provide a tool for integrating the available information on incidence, course of disease, and cost of treatment; it can serve as a basis for developing a more complete analysis. This study suggests several directions for additional research.

The probability trees reported here contain numerous gaps where no literature-based frequencies of outcomes or treatments have been reported. Very large clinical series are necessary to estimate probabilities of low-frequency outcomes, and these gaps are unlikely to be filled in rapidly. One approach to extending the data used here would be to survey practicing dermatologists and develop subjective estimates of the missing frequencies based on their professional experience.

The dose-response models used in this study provide an approximate measure of the long-term effect of a change in UV, but they do not account for the dynamic effects of increased UV over lifetimes. An increase in UV today would increase the rate of skin cancer only gradually over the next 80-100 years. Development of a dynamic dose-response model would require taking account of the time-path of UV exposure.

Expenditures on health care to treat skin cancer are but one of the health consequences of increased UV. Alerted to the risks, some people will take steps to reduce their exposure by using sunscreens, changing
vacation plans, and even their permanent place of residence. Those who contract a skin cancer suffer the anxiety of its uncertain consequences. These losses are less readily measured in monetary terms, and data on willingness to pay to avoid them are not currently available. A special survey of the general population and of skin-cancer patients could be developed to quantify these nonmonetary costs.
Appendix
SPREADSHEET USER'S GUIDE

REQUIREMENTS
The files for the health cost model were created using Version 2 of the 1-2-3 spreadsheet program by Lotus Development Corporation. The user should be comfortable enough with this program to be able to retrieve worksheet files and move the cursor around the spreadsheet. The Lotus program can be run on any IBM-compatible machine (using the Disk Operating System, or DOS). However, the size and complexity of the model of health costs require a minimum of 512 kilobytes of Random Access Memory (RAM). The operating system, the Lotus program, and the worksheet files use almost all of this when the entire spreadsheet is loaded into the workspace. Peripheral programs that reside in RAM must leave at least 464 kilobytes of RAM available to Lotus.

GETTING STARTED
The files needed for analyzing the model of health costs are contained on three floppy diskettes, labeled "PART1", "PART2", and "PART3". Making sure that sufficient RAM is available, the following steps will bring up the spreadsheet displaying the entire model:

1. At the DOS prompt (usually A>), place the 123 System Disk in the default drive and then type

```
123
```

followed by a carriage return to invoke the 1-2-3 program.

2. Place the diskette labeled "PART1" in the b: drive.¹

¹If the PC has only one disk drive, continue to use the "a:" drive as the default drive. Remove the 123 System Disk and continue following these instructions, substituting "a:" where "b:" is used.
3. When Lotus is ready (indicated by the READY mode indicator),
   change the default drive to b: by issuing the keystrokes

   /fdb:

   followed by a carriage return.

4. Retrieve the file "PART1.WK1" by issuing the keystrokes

   /fr

   followed by a carriage return when the filename is highlighted.

5. Place the diskette labeled "PART2" in the b: drive.

6. Combine the file "PART2.WK1" by issuing the keystrokes

   /fcce

   followed by a carriage return when the filename is highlighted.

7. Move the cursor to cell BW1.

8. Place the diskette labeled "PART3" in the b: drive.

9. Import a range from the file "WHITES.WK1" by issuing the keystrokes

   /fccn

   followed by the name of the population forecast, one of the
   following:

   Name      Description

   P1970      (Baseline Year)
P1980A (Forecast based on migration rates from 1965-1975)
P1990A
P2000A
P1980B (Forecast based on migration rates from 1965-1970)
P1990B
P2000B

After typing the name of the forecast, type two carriage returns. (The second accepts the filename "WHITES.WK1" when it is highlighted.)

10. Press the F9 key to recalculate all the formulas in the spreadsheet. Move the cursor around the worksheet to see the effects of the various parameters.

USING THE MODEL

After following the above instructions, you should have the entire model loaded into the spreadsheet. Figure A.1 provides a map of the spreadsheet, indicating the relative positions of the various components of the model. By moving the cursor around the spreadsheet, you can examine each of these components in detail. The macros provided in the

---

<table>
<thead>
<tr>
<th>UV levels</th>
<th>Dose-response model</th>
<th>Predicted incidence rates</th>
<th>Population</th>
<th>Predicted number of cases</th>
<th>Totals</th>
<th>Macros</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Number</td>
<td>Rate</td>
</tr>
</tbody>
</table>

In file PART2.WK1  In file WHITES.WK1  In file PART1.WK1

Fig. A.1 -- A map of the spreadsheet model of health care
spreadsheet will make it easier to view the results, once the population has been selected. In addition to changing population forecasts, you can change the projected level of UV radiation by changing the values of the variable UV DEL. These are initially set to 1.0, so that the values used for the UV level are the predicted UV levels for each state. A 10 percent increase in UV can be used by setting UV DEL to 1.1 for each state. (Users familiar with the copy command in Lotus will find this feature quite helpful for such an exercise.) After changing any values, be sure to press the F9 key to recalculate all the formulas in the spreadsheet.

**USER OPTIONS**

The user can change any of the values in the worksheet to see the effects of a particular change on the health costs of skin cancer due to ultraviolet radiation. The parameters of interest here, however, are:

- UV Levels
- Population
- Cost Per Case

As described above, UV Levels can be changed in the spreadsheet by changing the values of UV DEL (Column D, Rows 6 through 56) for each state. UV DEL is multiplied by UV to obtain UV USED, the parameter used in the dose-response model to obtain incidence rates.

The file "WHITES.WK1" (on the diskette labeled "WHITES.WK1") contains the seven population series mentioned above. Each series is in a named range that Lotus can import into the current spreadsheet. The user can examine the file "WHITES.WK1" in a separate Lotus session to understand the format of each population series. Other series can be created, either in "WHITES.WK1" or another spreadsheet file. These series can be imported into the spreadsheet model just as the existing series are, provided they have identical formats and the cursor is positioned in cell BW1.
The costs per case for both basal cell and squamous cell carcinomas are entered in the worksheet as simple numbers (not formulas), with a single national cost per case for each cell type. These can be changed in the manner that UV LEVEL can be changed, by changing the values for COST DEL (Column J, Rows 22 and 23), for each cell type.

To summarize the user options, the values in the following cells may be changed to analyze the sensitivity of health costs to changes in three parameters:

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Cell Ranges to Change</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>UV Level</td>
<td>D6 to D56</td>
<td>UV DEL (change factor)</td>
</tr>
<tr>
<td>Population</td>
<td>BW6 to CJ56</td>
<td>population by state, age, sex</td>
</tr>
<tr>
<td>Cost Per Case</td>
<td>J22 to J23</td>
<td>COST DEL (change factor)</td>
</tr>
</tbody>
</table>

MACROS

To make the worksheet easier to view (and to facilitate printing of results) several macros are provided. These macros are predefined keystroke sequences that change the display but not any values or formulas. The following macros are available. For each, hold down the ALT key and the designated letter key at the same time.

KEY RESULT

h hides most of the unnecessary columns
d redisplays the columns hidden by h
r displays rates next to state labels
c after r, displays costs next to state labels
n redisplays numbers of cases next to state labels
REFERENCES


