Skin cancer and UV radiation

SIR — Ultraviolet-B radiation (280–320 nm) has been implicated in induction of skin cancers in fair-skinned people. Reductions in stratospheric ozone (O_3) are expected to allow more solar ultraviolet-B to reach the Earth's surface. Calculated increases corresponding to O_3 reductions over 1979–89 have already been given[1,2]; we have now updated those estimates using O_3 measured until the end of December 1992 by the Total Ozone Mapping Spectrometer (TOMS) aboard the Nimbus 7 satellite[3]. We also combine the ultraviolet-B increases with new data on skin cancer induction by ultraviolet radiation to estimate the corresponding increase in skin-cancer incidence.

We used a radiative transfer model to calculate the increase in ultraviolet-B, assuming that the overhead ozone column[3] is the only atmospheric parameter to change[1]. Biologically effective ultraviolet doses are computed as

$$D = \iint F(\lambda,t)\, B(\lambda)\, d\lambda\, dt$$

where D is the dose received over time t, $F(\lambda,t)$ is the solar spectral irradiance at the surface and $B(\lambda)$ is the relative spectral sensitivity (action spectrum) of a specific biological effect. We computed doses using monthly average TOMS data (Jan 1979 to Dec 1992) from pole to pole in 10° latitude bands, then integrated over each year. We considered three action spectra relevant to the induction of skin cancer: for ultraviolet-induced skin cancer in mice[4], for erythema induction in fair-skinned people[5] and for DNA damage[6]. Obviously, no direct action spectrum is available for carcinogenesis in human skin.

Increases in annually integrated ultraviolet doses, shown in the table, are within one standard deviation (1σ) of zero only within about 10° latitude of the Equator, and become significant at the 2σ level polewards of 35° in both hemispheres. Relative to the 1979–89 changes[1], mid- and high-latitude trends are generally persisting and now carry substantially smaller uncertainties. A noteworthy difference is the appearance of trends which differ from zero by 1σ or more at latitudes within 15° of the Equator.

The relation between skin cancer incidence and ultraviolet dose is generally nonlinear, and is commonly represented by an exponential model[7]

$$\text{incidence} - \text{constant} \times (D)^{\text{BAF}}$$

where BAF is the so-called biological amplification factor, and is a function of, among other things, the cancer type and the action spectrum used to compute the ultraviolet dose. In a recent review[7] of epidemiological determinations of the BAF for fair-skinned people, values of 1.4±0.4 were given for basal cell carcinomas and 2.5±0.7 for squamous cell carcinomas (for cutaneous malignant melanoma, the scatter in the BAF determinations is still too large to allow for a reliable estimate of the increase of incidence). Application of these factors to the O_3-induced-skin-cancer dose increases leads to estimation of increases in the incidence of the non-melanoma skin cancers at middle and high latitudes of both hemispheres, as shown in the last two columns of the table. Note that the largest per cent increases are at high latitudes where baseline incidence of skin cancer is usually small[7].

Our calculation assumes that behavioural and demographic risk factors remain constant; that the 1979–92 increases in ultraviolet are sustained without further increase over the several decades required for skin-cancer development; and that the BAF model is independent of latitude and extent of ozone depletion. Some of these assumptions may be quite conservative, particularly the assumption that no additional ultraviolet increases will occur over the next few decades, while the assumption of no behavioural change may be too pessimistic. With stratospheric chlorine expected to increase until the turn of the century and not return to 1990 levels for several decades, it seems likely that ultraviolet-B will remain above natural levels, and perhaps increase further, well into the next century.

An important additional note is that the various ultraviolet-B monitoring networks now being planned will begin data collection in an environment which is already significantly perturbed from its natural baseline level at middle and high latitudes.

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EXPECTED INCREASES (PER CENT) IN ANNUAL ULTRAVIOLET DOSES AND SKIN CANCER INCIDENCE DUE TO STRATOSPHERIC OZONE DEPLETION FROM 1979–92

Latitude	Total ozone	Erythema induction dose	DNA damage dose	Skin cancer dose	Basal cell carcinoma incidence	Squamous cell carcinoma incidence
85 N	−8.8±3.2	7.1±1.7	14.8±3.6	10.6±2.5	15.1±5.6	28.5±11.2
75 N	−9.0±2.9	7.6±1.7	14.9±3.3	10.8±2.4	15.4±5.8	29.1±11.5
65 N	−7.4±1.7	7.6±1.7	14.1±3.2	10.3±2.3	14.7±5.6	27.7±11.0
55 N	−7.4±1.3	7.2±1.7	12.9±3.2	9.5±2.3	13.5±5.3	25.4±10.3
45 N	−6.6±1.2	6.5±1.7	10.9±3.0	8.1±2.2	11.6±4.7	21.6±9.0
35 N	−4.8±1.4	5.0±1.8	8.2±3.0	6.1±2.2	8.6±4.0	16.0±7.6
25 N	−2.7±1.5	3.0±1.9	4.8±3.1	3.5±2.2	5.0±3.5	9.0±6.4
15 N	−1.5±1.1	1.7±1.4	2.6±2.3	1.9±1.6	2.7±2.4	4.8±4.4
5 N	−0.6±1.6	0.7±1.9	1.2±3.0	0.8±2.2	1.2±3.1	2.1±5.5
5 S	−1.1±1.4	1.2±1.7	2.0±3.0	1.4±2.0	2.0±2.8	3.6±5.2
15 S	−1.9±1.3	2.2±1.5	3.5±2.4	2.5±1.7	3.6±2.6	6.5±4.8
25 S	−2.6±1.6	3.1±1.7	5.0±2.4	3.6±1.9	5.1±3.1	9.2±5.8
35 S	−4.0±1.6	4.8±1.6	7.9±2.6	5.7±1.9	8.1±3.6	14.9±6.8
45 S	−5.6±1.4	7.2±1.6	12.5±2.7	8.9±1.9	12.7±4.8	23.9±9.2
55 S	−9.0±1.5	10.9±2.0	19.7±3.6	14.2±2.6	20.4±7.4	39.3±15.1
65 S	−15.0±2.0	16.3±3.0	30.5±5.8	21.9±4.1	31.9±12.2	64.0±26.6
75 S	−19.5±2.6	24.1±5.4	49.8±12.0	34.0±7.7	50.6±21.4	107.7±52.0
85 S	−21.1±3.0	31.0±6.8	72.0±17.6	46.5±10.5	70.6±31.2	159.6±83.6

Increases in doses are evaluated over the 14-year data record, expressed as per cent relative to the 1979 intercept. Uncertainties are 1σ. See text for description of action spectra used in the dose calculations. The figures for carcinoma incidence are based on skin-cancer dose increase and biological amplification factors given in text.

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Fitness-related dispersal

SIR — Brown[1] offered an alternative explanation to my conclusion[2] that lifetime reproductive success payoffs, related to territory quality, predicted observed dispersal behaviour in the group-territorial bird the Seychelles warbler *Acrocephalus sechellensis*. He suggested that breeding vacancies on low-, medium- and high-quality territories were mainly filled by nonbreeders from territories of the same quality because of the easier access to these vacancies (for example high-quality territories almost never border on low-quality habitat and because there are fewer medium-quality territories — the 'area-restricted' hypothesis). Another suggestion, which in my view my data support, is that the dispersal to high-quality territories could be due to higher competition because there are more nonbreeders present in these territories (the 'queue' hypothesis).

During the removal experiments, as I