Ozone Depletion and Its Consequences for the Fluence of Carcinogenic Sunlight

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ABSTRACT

A slight reduction of the ozone level over the northern hemisphere in the period 1969-1986 has been reported [D. Lindley, Nature (Lond.), 323: 293, 1988]. Ozone measurements performed in Oslo are in agreement with this. However, the ozone level for 1987 and 1988 was above normal, and no negative or positive trend is apparent for the last 10 years. The consequences of an ozone reduction for the fluence rate of carcinogenically effective sunlight was evaluated on the basis of recent action spectra for mutagenesis in cells, carcinogenesis in mice, and erythema induction in humans. Depending on the choice of action spectrum we find amplification factors (defined as percentage increase in yearly fluence of carcinogenically efficient sunlight per percentage reduction of the ozone level) between 1.1 and 1.3 at latitudes between 0 and 20° and between 0.9 and 1.1 for Northern Europe. These estimates are significantly lower than 2.0, which is the value found when the calculations are based on the DNA absorption spectrum (R. B. Setlow, Proc. Natl. Acad. Sci. USA, 71: 3363-3366, 1974).

INTRODUCTION

The ozone layer in the atmosphere reduces the carcinogenic effect of sunlight by absorbing UV light below approximately 320 nm. For some years it has been known that the ozone layer over a large area in Antarctica exhibits a springtime depletion (1, for a review see Ref. 2). Furthermore, the so-called Ozone Trend Panel, an international group of atmospheric scientists set up in 1986 by the National Aeronautics and Space Administration, has announced a negative trend also for the Northern Hemisphere (3). The annual ozone concentration was found to be reduced by 2 to 3% in the period 1969 to 1986. It has been suggested that trace gases such as chlorofluorocarbons may lead to an ozone depletion (1, 3). Since the lifetimes of these relatively inert gases in the atmosphere are long, their effect on the ozone layer may continue for several years after their release. It is consequently of great importance to monitor the ozone layer continuously and furthermore to estimate the biological consequences of an ozone depletion. In the present work we report recent measurements of the ozone layer over the Oslo region and an evaluation of the so-called "radiation amplification factor," A. This factor can be defined by the equation:

\[ A = \frac{\Delta D}{[D_0]} \cdot \frac{D}{[O_3]} \]

where D is the annual exposure to carcinogenically effective sunlight and [O₃] is the total amount of ozone in the atmosphere. Thus, a 1% decrease in the ozone layer will lead to an increase of approximately A % of the annual exposure of a given population at a given geographical location to carcinogenically effective sunlight. A, changes with the latitude and with the initial ozone concentration and can only be used to estimate the effects of moderate (<15%) depletions of the ozone layer.

MATERIALS AND METHODS

Models. The fluence rate of carcinogenically effective solar radiation can be defined by the following expression: \( E_r = \int E_0 \phi_0 d\lambda \), the integration being performed over the wavelength region of the solar spectrum. \( E_0 \) is the solar irradiance at earth's surface, and \( \phi_0 \) is the action spectrum for carcinogenesis. \( \phi_0 \) for human carcinogenesis is not known. In the present work we have used two different approximations for \( \phi_0 \): (a) \( \phi_0 \) is assumed to be identical with the action spectrum for erythema in humans as approximated by the "reference spectrum" proposed by CIE (4); (b) \( \phi_0 \) is approximated by \( T \phi_0 \), where \( T \) is the transmittance through the epidermis and \( \phi_0 \) is the action spectrum for mutation of human cells. Thus, we assume that photocarcinogenesis is induced by mutation of cells in the basal layer. The justification for this is that there seems to be a correlation between mutagenesis and malignant transformation (5).

\( E \) was determined as follows: We used a discrete ordinate algorithm to calculate the propagation of light in vertically inhomogenous, plane parallel media (6). The model atmosphere used was the "US Standard Atmosphere 1976" which was divided in 39 homogeneous layers with a thickness of 2 km. We used the extraterrestrial solar radiation spectra as well as all orders of scattered light (Rayleigh scattering) from the atmosphere. The ground albedo was set to 0.2.

The integrals \( \int E_0 \phi_0 d\lambda \) and \( \int E T \phi_0 d\lambda \) were approximated by sums:

\[ \sum \Delta E_\lambda \phi_0 d\lambda \]  
\[ \sum \Delta T \phi_0 d\lambda \]

with \( \Delta \lambda = 1 \) nm and the percentage ozone depletion was assumed to be the same in all layers of the atmosphere. Almost identical results were found for a depletion concentrated to the region 14 to 24 km in the atmosphere. The absorption spectrum of ozone was taken from reference (7). For \( T \) we used the average transmission spectrum of eight samples of epidermis from the upper leg of Caucasians (8). For \( \phi_0 \) we used an average spectrum based on several publications (9-11, see Fig. 2).

O₃ Measurements. The amount of ozone in the atmosphere over Oslo was measured with a Dobson spectrophotometer at the Institute of Physics, University of Oslo. The spectrophotometer has a double monochromator and measures the radiance at a selected wavelength pair consisting of a short and a long wavelength, with strong and weak radiative absorption by ozone, respectively.

The total amount of ozone in a vertical column is usually expressed as the thickness in millim of an ozone layer converted to normal pressure and temperature. This is the Dobson Unit (DU). Normally, the ozone amount over Oslo varies from about 300 DU in September, October to about 430 DU in March, April. The ozone level has been expressed as percentage deviation from a mean value for a given time period analogously to the procedure in Ref. 12.

The Dobson spectrophotometer in Oslo was calibrated against other instruments in 1977 (at the National Oceanic and Atmospheric Administration's laboratory) and in 1986 and had remained perfectly constant during that time.

RESULTS AND DISCUSSION

As shown in Fig. 1 the ozone measurements performed in Oslo in the period 1969-1986 agree with those given by Bojkov (12). However, high values for the ozone layer have been measured in 1987 and 1988, indicating that the decreasing trend reported for the period 1969-1986, notably for the winter season (Fig. 1), may represent fluctuations rather than report a
general decrease of the \( O_3 \) level. In view of the variations shown in this figure we have considered the carcinogenic effects of ozone variations of the order of a small fraction (<10%).

Fig. 2 shows data from recent publications for mutation frequency induced by light at different wavelengths. For comparison we have included data for formation of (6-4) photoproducts and for endonuclease sensitive sites in DNA (254–334 nm) (13) as well as for inactivation of human skin cells (14). With a couple of exceptions all these action spectra are similar. Exceptionally low values for mutation were found at 334 nm (10) and at 365 nm (11). This may be related to the fact that light in this wavelength region can act on the highly mutagenic (6-4) photoproducts (15). Thus, the approximate average spec-

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**Fig. 1.** The fluctuations in the \( O_3 \) level given by Bojkov (12) (---) and those measured in Oslo. Winter values are the average of the months December to March and summer values include the months May to August.

![Image](image-url)

**Fig. 2.** Action spectra for mutagenesis and inactivation of cells in culture. All spectra are normalized to 1 at 254 nm. Data for mutagenesis are from (9) (\( V \), human fibroblasts); (10) (\( C \), human epithelial P3 cells); (11) (\( E \), Chinese hamster cells). For comparison, data for formation of endonuclease sensitive sites and (6-4) photoproducts in DNA are included (13) (\( \delta \), as well as data for cell killing (14) (\( \ast \), mean values for several human cell lines). The latter action spectrum agrees closely with similar action spectra for cell inactivation given in the references cited above.

![Image](image-url)

**Fig. 3.** Action spectra for carcinogenesis in mice (---) [Steenenborg, 1987 (17)]; CIE risk spectrum, similar to the spectrum for erythema in humans (---) (4); and \( T_{255} \) calculated as described in the text (---). All spectra are normalized to the same value at 295–300 nm.

![Image](image-url)

**Fig. 4.** Effectiveness spectra \( (ET\phi_m) \) for mutation of cells in the basal layer.

The product \( ET\phi_m \) gives an effectiveness spectrum for mutation of cells in the basal layer (Fig. 4). Using the Commission Internationale de l'Éclairé reference spectrum (Fig. 3) yields qualitatively similar results (data not shown). These spectra are similar to those for cytotoxic effectiveness of sunlight at the basal layer (14). As the zenith angle increases the maximum of this spectrum is shifted towards longer wavelengths, and accordingly the role of UVA increases. Since UVA is only weakly absorbed by ozone, we can already conclude from this observation that the relative effect of a given ozone depletion will...
decrease with increasing latitude. This is more explicitly shown below.

Since sunlight-induced carcinogenesis can be assumed to take several years, one needs to calculate the yearly exposure $D$ to carcinogenic sunlight in order to estimate the amplification factor $A_s$. Fig. 5 shows the result of the calculation of the integral $\int E_c dt = D$ over 1 year using the two before-mentioned approximations for the action spectrum for carcinogenesis. In these calculations we have taken into account the average ozone level as well as the yearly variation of this level at all latitudes. The variation of the annual exposure $D$ with the latitude is almost independent of whether the CIE- or the $T_{\Phi_m}$ action spectrum (Fig. 3) is used in the calculations.

Finally, using the absorption spectrum of ozone (7) we have approximated $A_s$ by calculating $\frac{dD}{dD} \frac{d[O_3]}{[O_3]}$ for 1% ozone depletion (Fig. 6). From these data we can draw several conclusions: (a) Using the CIE risk spectrum for carcinogenesis, $A_s$ values which are similar to within 20% are obtained for latitudes below $50^\circ$. At higher latitudes the difference becomes greater, reflecting the difference between the action spectra in the UVA region (Fig. 3). (b) Regardless of which of the action spectra for carcinogenesis that is used, $A_s$ decreases with increasing latitude. Thus, the relative consequences of a given ozone depletion for skin carcinogenesis decreases with increasing latitude. (c) At any latitude the amplification factor calculated here is significantly smaller than 2 which is the amplification factor at $30^\circ$ obtained by approximating the action spectrum for carcinogenesis at the basal cell layer by the absorption spectrum of DNA (19). The reason for this is that UVA is more carcinogenic than indicated by the absorption spectrum of DNA.

It should be noted that these calculations are relevant only for nonmelanoma skin cancer since the fluence rate and episodes of sunburn may play a larger role than the total exposure for induction of melanomas.

How large then will the increase in incidence ($R$) of nonmelanoma skin cancer be for a given ozone reduction? To answer this question one needs to know also the biological amplification factor: $A_s = \frac{dR}{R} \frac{dD}{D}$. The overall amplification factor is:

$$A = A_s A_h = \frac{dR}{d[O_3]} \frac{d[O_3]}{R}$$

Using recent data from the Norwegian Cancer Registry for $R$ at different latitudes in Norway, where $D$ can be determined (see Fig. 6) preliminary calculations yield $A_s$ values of about 1.6 to 1.8 for squamous cell carcinomas and 2.1 to 2.3 for basal cell carcinomas (details will be published separately). This is in agreement with other estimations (see review in Ref. 20) and indicates that the overall amplification factor is between 1.3 and 2.1 in Northern Europe.

REFERENCES

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