0014-4754/93/090747-07$1.50 + 0.20 © Birkhäuser Verlag Basel, 1993

Biological effectiveness of solar UV radiation in humans

W. Ambach and M. Blumthaler

Institute of Medical Physics, University of Innsbruck, Müllerstraße 44, A-6020 Innsbruck (Austria)

Abstract. Solar UVB radiation is prejudicial to the health of humans in a number of ways. Erythema and photodermatoses are acute reactions of the skin; keratitis and conjunctivitis are acute reactions of the eye. Various types of skin cancer, accelerated aging of the skin, and cataract formation in the crystalline lens are reactions that appear with great latency. UV radiation can also cause damage to the immune system and DNA. For the period 1981–1991, an increase in erythemal effective UVB radiation of $+(7 \pm 4)\%$ per decade was measured in a non-polluted high mountain area (Jungfraujoch, 3576 m a.s.l., Switzerland). This increase is related to a decrease in stratospheric ozone. The effects on human health are discussed. A 10% ozone reduction increases non-melanoma skin cancer by 26% and cataract by 6 to 8%.
Key words. Solar UV radiation; UV-induced biological effects; action spectra; stratospheric ozone.

Introduction

Measurements of solar UV radiation dosages are gaining in importance as stratospheric ozone concentrations are decreasing world-wide. This irradiation has no effect on the weather, but measuring the dosage is important as various biological reactions are induced by UV radiation. The ultraviolet range of the spectrum is divided into three parts: UVA (315–400 nm), UVB (280–315 nm) and UVC (200–280 nm). This division is based upon the widely differing biological effects occurring in these spectral ranges.

While the share of UVB radiation in the spectrum is as high as 1.5% outside the earth's atmosphere, it is less than 0.5% at the earth's surface. Radiation energy with wavelengths smaller than 290 nm reaching the surface of the earth cannot be measured with spectrometers in common use. The temporal and local variability of solar UVB radiation is extremely high as it is influenced by a number of different parameters, such as ozone concentration, solar elevation, altitude, cloud-cover, turbidity and albedo (the reflective power of a surface).

Solar UVB radiation is very important for humans because it has a positive effect on their psychological well-being, and because it promotes the production of vitamin D. These positive effects, however, are offset by a number of deleterious effects. Erythema and photodermatoses occur as acute reactions of the skin, and keratitis and conjunctivitis as acute reactions of the eye. Various types of skin cancer, accelerated aging of the skin and cataract formation in the crystalline lens are reactions that appear with great latency. It has been shown that the immune system is also affected negatively, although more detailed research into this aspect is required.

Action spectra and threshold doses

Each of the UV-induced biological reactions has a definite action spectrum and threshold dose. The action spectrum indicates the biological effectiveness of monochromatic radiation of different wavelengths for a given reaction. Action spectra are normalized to 100% at the maxima. The threshold dose for a specific reaction refers to the smallest dosage of monochromatic irradiance with the wavelength of the maximum in the relevant action spectrum which produces the effect.

Erythema

As a result of historical developments, two different action spectra for erythema are to be found in the literature (fig. 1a, b). The action spectrum established by McKinley and Diffey[28] is recommended by the Commission Internationale de l'Eclairage (CIE) and is used in more recent research. Two characteristics distinguish it from the action spectrum according to DIN 5031[13], which was generally accepted earlier on: a constant 100% effectiveness is given for wavelengths less than 297 nm, and a low but decreasing effectiveness from 1.0%–0.1% is given in the UVA range. This action spectrum thus includes a UVA-induced erythemal reaction, even though the threshold dose is reached only with high UVA irradiance, on account of the low effectiveness in the UVA range.

Immediate and delayed tanning

Tanning of the skin involves two different reactions, immediate and delayed tanning. Immediate tanning is induced by UVA radiation. It occurs as a result of the oxidation and subsequent darkening of melanin precursors that are already present in the skin. The action spectrum of immediate tanning (fig. 1c) has its maxi-

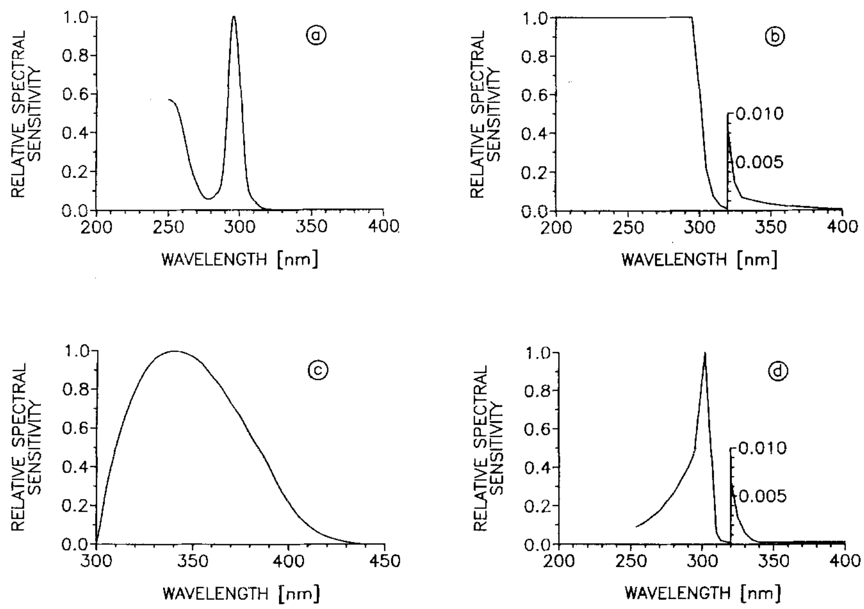

Figure 1. Action spectra of a) erythema according to DIN 5031[13], b) erythema according to McKinlay and Diffey[28], c) immediate tanning according to DIN 5031[13], d) carcinogenicity according to van der Leun et al.[39].

Table. Mean threshold doses for erythema, immediate and delayed tanning, and temporal course of reactions[17,20]

	UVA	UVB
Erythema		
Threshold dose	200 k J m^{-2}	0.25 k J m^{-2}
Onset	4–6 h	2–6 h
Maximum	10–12 h	20–24 h
Duration	36–48 h	72–120 h
Immediate tanning		
Threshold dose	60 k J m^{-2}	-
Onset	5–10 min	-
Delayed tanning		
Threshold dose	180 k J m^{-2}	0.3 k J m^{-2}
Onset	2 days	2 days

mum at 340 nm and extends slightly into the UVB and visible ranges of the spectrum; its half-maximum full width is 70 nm[13]. Delayed tanning is a concomitant symptom of erythema, so the action spectrum for erythema is used as the action spectrum of this process. In delayed tanning, new melanin is produced.

In connection with tanning, it is therefore appropriate to distinguish between UVB- and UVA-induced erythema. UVB radiation only causes delayed tanning in addition to erythema, while UVA radiation induces both immediate and delayed tanning. Immediate tanning cannot be achieved with UVB radiation. The table shows the average threshold doses for erythema and for immediate and delayed tanning. The maximum reaction is reached faster in UVA-induced erythema than in UVB-induced erythema. The UVA-induced erythema subsides faster than the UVB-induced erythema. UVB-induced erythema occurs 2 to 6 h after radiant exposure. It reaches its maximum after 20 to 24 h and heals within 5 days. Delayed tanning starts when the healing process sets in. Immediate tanning, on the other hand, starts immediately after exposure to radiation but fades relatively quickly.

Photodermatoses

UVA and UVB radiation can cause allergic and toxic photodermatoses. A toxic photodermatosis develops when a subject takes certain drugs, e.g. sulphonamides, antibiotics or antidiabetics, and is then exposed to UV radiation. This leads to skin rashes of various types whose action spectra extend from the UVB range to the visible range of the spectrum, with UVA radiation being especially important. Allergic photodermatoses disappear as soon as the radiation source is removed. Examples of photodermatoses are polymorphic light eruption (290–365 nm), systemic lupus erythematosus (290–330 nm), solar urticaria (290–480 nm) and porphyria cutanea tarda (340–600 nm)[21].

Photo-aging

Frequent solar exposure, in particular exposure to UVA radiation, leads to photo-aging of the skin, or heliodermatitis. Wrinkling, thickening of the cornea, dry and rough skin, typical discoloration and increased occurrence of liver spots are some external signs of this condition. Photo-aging occurs with a long latent period, so the reactions involved have not yet been sufficiently investigated. Animal experiments with hairless mice now enable scientists to investigate photo-aging within weeks, so that new results can be expected soon[30].

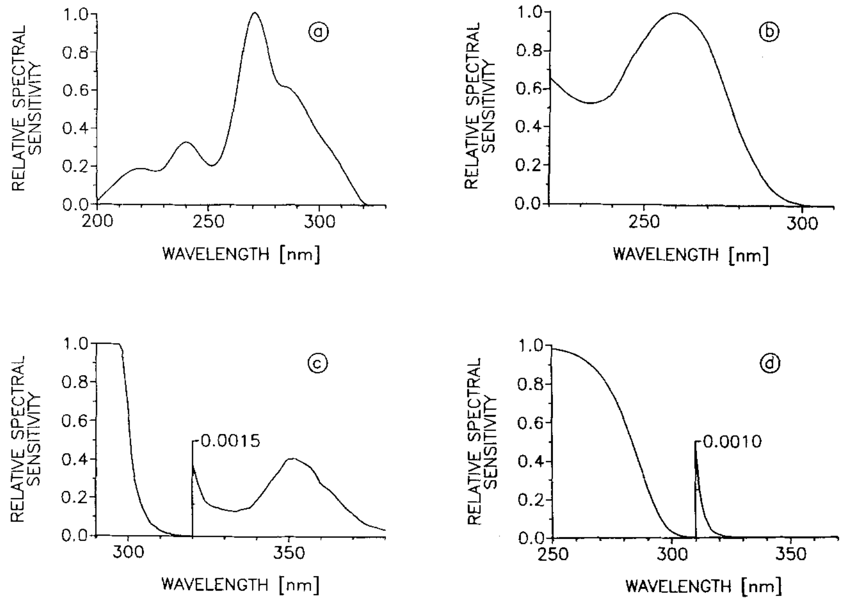

Figure 2. Action spectra of a) keratitis according to DIN 5031[13], b) conjunctivitis according to DIN 5031[13], c) cataract according to Hoover[18], d) DNA damage according to Setlow[35].

Skin cancer

UV-induced types of skin cancer include basal cell carcinoma, squamous cell carcinoma and melanoma. While there is clearly a causal relationship between UV dosage and the incidence of non-melanoma skin cancer (basal cell carcinoma and squamous cell carcinoma)[33], the correlation with UV dosage is highly complex for melanoma skin cancer. UV exposure is an important additional factor, but there are other causes whose role in the genesis of melanoma is little known as yet[12]. It is thought that intermittent severe exposures (severe enough to cause sunburn) are decisive for UV-induced melanoma. It is also pointed out that UV exposures in infancy are more dangerous than exposures later in life. It is also certain that the development of non-melanoma skin cancer by UVB radiation is accompanied by damage to the DNA and its repair system, and by an alteration of the immune system[33]. Consequently, the action spectrum of UV-induced DNA damage is also relevant in this context (fig. 2d).

The varying degree to which UV radiation is transmitted by the epidermis of different skin types is the reason why fair-skinned people are at greater risk than dark-skinned ones given the same amount of radiant exposure. There are also reports about UV-induced lip cancer and salivary gland cancer[36]. Animal experiments have shown that the UVB range of the solar spectrum has the highest carcinogenicity. Epidemiological studies in humans, however, did not permit the same conclusion. For wavelengths greater than 300 nm, the action spectrum of carcinogenicity (fig. 1d) in hairless mice[26] was found to be very similar to the action spectrum of erythema, according to McKinley and Diffey[28].

Keratitis

The action spectrum of keratitis according to DIN 5031[13] is widely accepted (fig. 2a). Its maximum is in the UVC range, i.e. outside the solar spectrum. The slope at the long-wave end extends into the UVB range. UVA radiation does not produce keratitis. As for the threshold dose, it is necessary to distinguish between short-term exposure (as it occurs in arc welding) and long-term exposure (such as in skiing). The threshold dose is 40 J m^{-2} for short-term exposure[31]. For long-term exposure, a considerably higher threshold dose was derived from observations of patients who contracted keratitis when skiing[6]. Keratitis is generally accompanied by conjunctivitis. Figure 2b shows the action spectrum of conjunctivitis.

Cataract

The action spectrum of cataract has not yet been determined for humans because cataract has a long latent period and because the clouding of the lens progresses very slowly. An action spectrum for cataract genesis was determined in animal experiments with rabbits and guinea pigs[3,32] which has its maximum at 297 nm in the UVB range and extends to the UVA range at the long-wave end (fig. 2c). Effectiveness in producing cataract in the UVA range is about 0.1%[18]. While Taylor et al.[38] found a correlation of UVB exposure with cortical cataract in an epidemiological study on watermen, and case-control studies[11,29] have revealed a correlation for all forms of cataract, including nuclear and mixed cataracts. The influence of UVA radiation on cataract genesis has not yet been fully investigated. Although the radiation absorbed by the lens is higher by a factor of 10^4 at 350 nm than at 300 nm[19], UVB radiation seems to be the dominant factor for cataract genesis.

Variability of solar UVB radiation

The variability of solar UVB radiation is due to the following factors: total amount of atmospheric ozone, atmospheric turbidity, cloudiness and albedo. These influences cause daily and seasonal variations in the solar UVB radiation, which show a marked dependence on solar elevation, altitude and geographical situation. Solar UVB radiation is measured either with broad-band detectors or with high-resolution spectrometers. Among the broad-band detectors, the Robertson-Berger Sunburn Meter[4] is a proven instrument that is used world-wide. Its spectral sensitivity is adapted to the erythemal action spectrum, and it is therefore used to measure the erythemal-effective irradiance. As the action spectra seem to be similar, erythemal effective irradiance can be equated with carcinogenic effective radiation. Solar UVA radiation is mostly measured using a broad-band UVA radiometer[14].

Seasonal changes in daily totals

Figure 3 shows daily totals of erythemal effective radiation and of total global radiation (0.3–3 μm) at the high-mountain research station Jungfraujoch (Switzerland, 3576 m a.s.l.) and in Innsbruck (Austria, 577 m a.s.l.). The daily totals were measured during periods of several weeks from 1981 to 1990 to obtain a

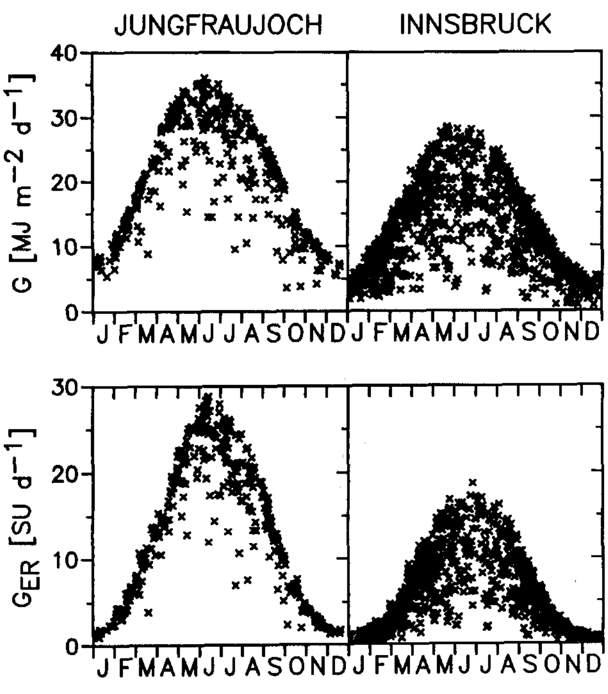

Figure 3. Seasonal course of daily totals of erythema dose (G_{ER}) and total global radiation (G) at the stations Jungfraujoch (3576) m a.s.l.) and Innsbruck (577 m a.s.l.)[9].

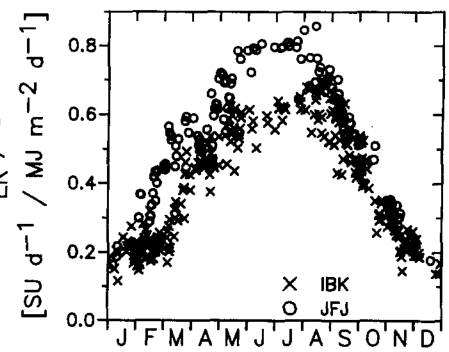

Figure 4. Seasonal courses of the ratio of daily totals of erythema dose and total global radiation (G_{ER}/G) at the stations Jungfraujoch (3576 m a.s.l.) and Innsbruck (577 m a.s.l.) on days with mean cloudiness $<5/10$[9].

detailed picture of the seasonal course. Erythemal effective irradiance is expressed in Sunburn Units (SU), with 1 SU corresponding to an absorbed dose of 250 J m^{-2} at 297 nm and 90° solar elevation[4]. A dose of 250 J m^{-2} is considered as the threshold dose of erythema and is called 'Minimal Erythema Dose' (MED).

Figure 3 illustrates the following characteristics: The fictitious envelope of the measuring data indicates the maximal daily totals that are possible, as a function of the season. The maxima are 28.7 SU at Jungfraujoch and 18.7 SU in Innsbruck. The effect of altitude on the maximal daily totals, expressed as the ratio of daily totals of mountain station to valley station, is 1.53 for the erythemal effective radiation and 1.24 for the total global radiation for a difference in altitude of 2999 m. The seasonal change in the daily totals shows a steeper increase towards the seasonal maximum for erythemal effective radiation than for total global radiation. This is due to the stronger decrease in erythemal effective radiation caused by ozone, which is especially effective at low solar elevations. Total global radiation, by contrast, is not influenced by ozone to any measurable extent. Daily totals below the fictitious envelope in figure 3 are due to heavier cloudcover, higher ozone concentrations or stronger atmospheric turbidity. Owing to the influence of ozone on UVB radiation and the varying optical paths in the course of the year, the ratio of UVB radiation to total global radiation shows a marked seasonal course with maxima in summer and minima in winter (fig. 4).

Influence of cloudiness and altitude

To illustrate the influence of cloudiness on UVB radiation, daily totals in the seasonal records were normalized by the ratio G_{ER}/G^*_{ER}. G^*_{ER} denotes the respective maximal daily totals of erythemal effective radiation that are possible on a given day, which are indicated by the fictitious envelope in figure 3. G_{ER} denotes the measured daily totals of erythemal effective radiation. The mean cloudiness was calculated on the basis of

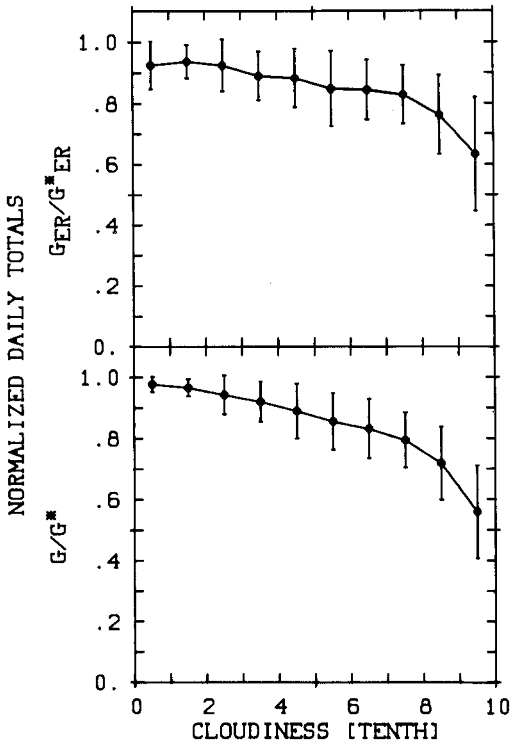

Figure 5. Influence of cloudiness on normalized erythema dose (G_{ER}/G^*_{ER}) and normalized total global radiation (G/G*) at Jungfraujoch (3576 m a.s.l.) G^*_{ER} and G* are maximal values under cloudless sky. Bars indicate S.D.[7].

half-hourly observations. Figure 5 shows the decrease of the daily totals to 0.6 with increasing cloudiness (10/10). The deviation is naturally very high. The fact that values less than 1 can even occur at a cloudiness of 0/10 can be ascribed to the variability of the ozone content.

The increase in biologically effective UVB radiation with altitude depends strongly on the albedo (reflective power) of the terrain. According to Green's radiation transfer model, the calculated altitude effect for erythemal effective radiation at horizontal surfaces is 1.25 to 1.57 (irradiance at 3000 m/irradiance at sea level) at 60° solar elevation[1]. The lowest value is measured when both stations are snow-free, and the highest value occurs when the terrain around the valley station is snow-free and the terrain around the mountain station is covered with snow. Cloudless sky and representative atmospheric turbidity were assumed for the calculation.

Trend of solar UVB radiation with atmospheric ozone reduction

The increase in solar UVB radiation resulting from the world-wide decrease in stratospheric ozone was calculated by means of radiation transfer models. Measurements revealed no uniform trend. For an experimental trend analysis of erythemal effective radiation, it is best to use a series of measurements made with the Robertson-Berger Sunburn Meter, although the long-term

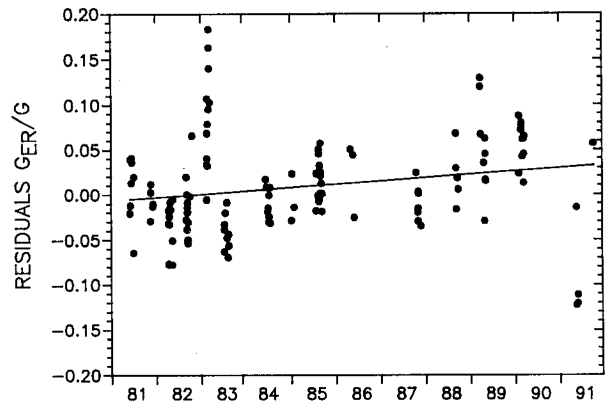

Figure 6. Trend of erythemal UVB radiation at Jungfraujoch (3576 m a.s.l.). Residuals are deviations from the averaged seasonal course[5].

stability of the calibration factor may pose problems. Evaluations at 25 stations in the USA showed a decrease in UVB radiation between −5% and −11% per decade from 1974 to 1985[34]. This unexpected result is ascribed to local air pollution as most stations are located in urban areas. A decrease in UVB radiation of −8% per decade was also measured in Russia (Moscow) from 1968 to 1983, while turbidity increased by +10% per decade and cloudiness rose by +9% per decade[16]. When performing a trend analysis in connection with atmospheric ozone reduction, it is necessary to separate the influences of cloudiness and turbidity from the influence of ozone on solar UVB radiation. Such an analysis was made in a non-polluted high mountain area (Jungfraujoch, Switzerland), where measurements carried out with a Robertson-Berger Sunburn Meter[8] showed an increase in erythemal effective solar UVB radiation of +(7 ± 4)% per decade from 1981 to 1991 (fig. 6). The 1992 data were probably affected by the abnormal turbidity caused by the eruption of Pinatubo, so that extrapolation seems to be impossible for the time being.

Conclusion

The key issue is to what extent the increase in solar UV radiation resulting from the decrease in atmospheric ozone can impair human health. In addition to the ozone hole over Antarctica, which has appeared in the months of October and November since the early 1970s[15,25] and which causes a temporary reduction of stratospheric ozone of about 50%, a moderate decrease in ozone is also observed in the northern hemisphere[37]. However, this moderate decrease can not be described as an ozone hole. The longest series of ozone measurements started in Arosa, Switzerland, in 1926 and shows a trend of −5.9% per decade in winter and of −2.8% per decade in summer for the period from November 1978 to March 1991. The annual mean for Europe for the period 1970–1991 shows a decrease of −1.8% per decade[37]. However, this trend must be seen in relation with the marked seasonal changes in ozone. The variability in mean northern latitudes is about 50% over the seasons[10].

The eruption of El Chichón in Mexico (March/April 1982) brought about an ozone reduction accompanied by a marked increase in solar UVB radiation in northern latitudes[8]. Hypotheses that the eruption of Pinatubo in the Philippines (June 1991) would further reduce the ozone in the Antarctic ozone hole are being discussed[22]. Speculations that the eruption of Pinatubo would cause an increased chlorine monoxide concentration in the Northern Hemisphere, and would thus produce an ozone hole in the Arctic comparable to that over Antarctica in the spring of 1992[23], did not come true[24]. However, the long-term effects of the Pinatubo eruptions on ozone in the Northern Hemisphere must be considered.

A correlation between the ozone hole and the greenhouse effect was established by model calculations. These showed that a cooling of the lower stratosphere is accompanied by global warming. This cooling can promote the formation of polar stratospheric clouds that are involved in ozone depletion. Recent model calculations have given rise to speculations[2] that a doubling of CO_2 will lead to an Arctic ozone hole in the Northern Hemisphere winter stratosphere that is comparable to the Antarctic ozone hole.

While there is scientific proof of the trend of atmospheric ozone depletion, the corresponding increase in solar UVB radiation remains to be fully investigated. Up to now, the multifactorial influence of ozone, cloudiness and turbidity on solar UVB radiation has limited the interpretation of measurements, therefore the effects of ozone reduction on solar UVB radiation are generally studied by means of radiation transfer models[27]. However, these models are often based on idealized assumptions like a cloudless sky and an aerosol-free atmosphere, limiting their relevance to actual atmospheric conditions.

Given the general ozone fluctuations in the course of the seasons, it is possible to establish a correlation between ozone concentration and spectral UVB radiation by means of experiment. The percent change of spectral UVB radiation, given by a 1% change in atmospheric ozone, is called the Radiation Amplification Factor (RAF)[26]. It depends strongly on the wavelength and changes during the season because of changing solar elevations. Wavelengths that cause skin damage have an RAF between 1.1 and 1.7, depending on the type of disease. For wavelengths that damage the eyes, the RAF value lies between 0.8 and 1.2. An RAF of 1.7, for example, means that a 10% decrease in ozone results in a 17% dose increase. The correspondingly enhanced health risks depend in addition on the dose-response relationship. A 10% ozone reduction, for example, results in a 26% increase in non-melanoma skin cancer and increases cataracts by 6 to 8%[26], depending slightly on the solar spectrum. However, these figures apply only if in future the increase in solar UVB radiation is not offset by other factors like greater turbidity, increased cloudiness or higher tropospheric ozone concentrations.

Acknowledgements. Gratitude is expressed to the Österreichische Akademie der Wissenschaften for financial support of the research project 'Spectral measurements of solar UVA- and UVB radiation in high mountain areas' and to the Fonds zur Förderung der wissenschaftlichen Forschung for financing the spectrometer.

1 Ambach, W., Blumthaler, M., and Schöpf, T., Increase of biologically effective UV-radiation with altitude. Journal of Wilderness Medicine, *4* (1993) 189–197.

2 Austin, J., Butchart, N., and Shine, K. P., Possibility of an Arctic ozone hole in a doubled-CO_2 climate. Nature *360* (1992) 221–225.

3 Bachem, A., Opthalmic ultraviolet action spectra. Am. J. Ophthal. *41* (1956) 969–975.

4 Berger, D. S., The sunburning ultraviolet meter: Design and performance. Photochem. Photobiol. *24* (1976) 587–593.

5 Blumthaler, M., High altitude UVB-measurements and trends in Switzerland; in: UV-B Monitoring Workshop: A review of the science and status of measuring and monitoring programs, 10–12 March 1992, Washington D.C., p. C-145. Science and Policy Associates, Inc., Washington D.C. 1992.

6 Blumthaler, M., Ambach, W., and Daxecker, F., On the threshold radiant exposure for Keratitis solaris. Invest. Ophthalmol. Vis. Sci. *28* (1987) 1713–1716.

7 Blumthaler, M., and Ambach, W., Human solar ultraviolet radiant exposure in high mountains. Atmos. Environ. *22* (1988) 749–753.

8 Blumthaler, M., and Ambach, W., Indication of increasing solar ultraviolet-B radiation flux in alpine regions. Science, Vol. 248 (1990) 206–208.

9 Blumthaler, M., Ambach, W. and Rehwald, W., Solar UV-A and UV-B radiation fluxes at two Alpine stations at different altitudes. Theor. appl. Climatol. *46* (1992) 39–44.

10 Caldwell, M. M., Madronich, S., Björn, L. O., and Ilyas, M., Ozone reduction and increased solar ultraviolet radiation, in: Environmental Effects Panel Report, pp. 1–10. United Nations Environment Programme, Nairobi 1989.

11 Collman, G. W., Shore, D. L., Shy, C. M., Checkoway, H., and Luria, A. S., Sunlight and other risk factors for cataracts: An epidemiologic study, AJPH *78* (1988) 1459–1462.

12 De Gruijl, F. R., Ozone change and melanoma, in: Atmospheric ozone research and its policy implications. Proceedings of the 3rd US-Dutch International Symposium, 1988, Nijmegen, pp. 813–821. Eds T. Schneider, S. D. Lee, G. J. R. Wolters and L. D. Grant. Elsevier Science Publishers, B. V., Amsterdam 1989.

13 DIN 5031, Deutsche Normen, Strahlungsphysik im optischen Bereich und Lichttechnik, Teil 10, Beuth-Verlag Berlin 1979.

14 Drummond, A. J., and Wade, H. A., Instrumentation for the measurement of solar ultraviolet radiation, in: The Biological Effects of Ultraviolet Radiation, pp. 391–407. Ed. F. Urbach. Pergamon Press, Oxford, New York 1969.

15 Farman, J. C., Gardiner, B. G., and Shanklin, J. D., Largest losses of total ozone in Antarctica reveal seasonal ClO_x/NO_x interaction. Nature *315* (1985) 207–210.

16 Garadazha, M. P., and Nezval, Y. I., Ultraviolet radiation in large cities and possible ecological consequences of its changing flux due to anthropogenic impact, in: Proc. WMO, WHO, UNEP Symp. on Climate and Human Health, World Climate Programme Applications, pp. 64–68, Leningrad, WCAP Report No. 2 (1987).

17 Hawk, J. L. M., and Parrish, J. A., Responses of normal skin to ultraviolet radiation, in: The Science of Photomedicine, pp. 219–260. Eds J. D. Reagan and J. A. Parrish. Plenum Press, New York, London 1982.

18 Hoover, H. J., Sun glasses, pupil dilation, and solar ultraviolet irradiation of the human lens and retina. Appl. Optics *26* (1987) 689–695.

19 Huber, M., Ambach, W., Blumthaler, M., Schöpf, T., Ambach, E., Tributsch, W., Daxecker, F., and Daxer, A., Spektrale Absorptionsmessungen an optischen Elementen des menschlichen Auges, pp. 328–329. Ed. J. Roth. Medizinische Physik 1992, Jahrestagung, Basel 7–10 Oktober 1992.

20 Kaidbey, K. H., and Kligman, A. M., The acute effects of long-wave ultraviolet radiation on human skin. J. invest. Derm. *72* (1978) 253–256.

21 Kaplan, L. A., Suntan, sunburn, and sun protection. J. Wilderness Med. *3* (1992) 173–196.

22 Kerr, R. A., Pinatubo fails to deepen the ozone hole. Science *258* (1992) 395.

23 Kerr, R. A., New assaults seen on earth's ozone shield. Science *255* (1992) 797–798.

24 Kerr, R. A., Not over the Arctic – for now. Science Vol. *256* (1992) 734.

25 Krueger, A., Schoeberl, M., Newman, P., and Stolarski, R., The 1991 Antarctic ozone hole; TOMS observations. Geophys. Res. Lett. *19* (1992) 1215–1218.

26 Longstreth, J. D., De Gruijl, F. R., Takizawa, Y., and van der Leun, J. C., Human health, in: Environmental effects of ozone depletion: 1991 update, pp. 15–24. United Nations Environmental Programme, Nairobi 1991.

27 Madronich, S., Implications of recent total atmospheric ozone measurements for biologically active ultraviolet radiation reaching the earth's surface. Geophys. Res. Lett. *19* (1992) 37–40.

28 McKinlay, A. F., and Diffey, B. L., A reference action spectrum for ultraviolet induced erythema in human skin. Commission Internationale de l'Eclairage (CIE) – Journal *6* (1987) 17–22.

29 Mohan, M., Sperduto, R. D., Angra, S. K., Milton, R. C., Mathur, R. L., Underwood, B. A., Jaffrey, N., Pandya, C. B., Chhabra, V. K., Vajpayee, R. B., Kalra, V. K., and Sharma, Y. R., The India-U.S. case-control study group. India-U.S. case-control study of age related cataracts. Archs Opthal., N.Y. *107* (1991) 670–676.

30 Moloney, S. J., Edmonds, S. H., Giddens, L. D., and Learn, D. B., The hairless mouse model of photoaging: Evaluation of the relationship between dermal elastin, collagen, skin thickness and wrinkles. Photochem. Photobiol. *56* (1992) 505–511.

31 Pitts, D. G., The ocular effects of ultraviolet radiation. Am. J. Optom. physiol. Optics *55* (1978) 19–35.

32 Pitts, D. G., and Cullen, A. P., Ocular ultraviolet effect from 300 nm to 400 nm, a preleminary report. US Department of Health, Education and Welfare 1976.

33 Preston, D. S., and Stern, R. S., Non-melanoma cancers of the skin. New Engl. J. Med. *327* (1992) 1649–1662.

34 Scotto, J., Cotton, G., Urbach, F., Berger, D., and Fears T., Biologically effective ultraviolet radiation: Surface measurements in the United States, 1974 to 1985. Science *239* (1988) 762–764.

35 Setlow, R. B., The wavelengths in sunlight effective in producing skin cancer: A theoretical analysis, Proc. natl Acad. Sci. *71* (1974) 3363–3366.

36 Spitz, M. R., Sider, J. G., Newell, G. R., and Batsakis, J. G., Incidence of salivary gland cancer in the United States relative to ultraviolet radiation exposure. Head Neck Surg. *10* (1988) 305–308.

37 Stolarski, R., Bujkov, R., Bishop, L., Zerefos, C., Staehelin, J., and Zawodny, J., Measured trends in stratospheric ozone. Science. *256* (1992) 342–349.

38 Taylor, H. R., West, S. K., Resenthel, F. S., Beatrix, M., Newland, H. S., Abbey, H., and Emmett, E. A., Effect of ultraviolet radiation on cataract formation. New Engl. J. Med. *319* (1988) 1429–1433.

39 Van der Leun, J. C., Takizawa, Y., and Longstreth, J. D., Human Health, in: Environmental Effects Panel Report, pp. 11–24. United Nations Environment Programm, Nairobi 1989.