MOLECULAR MEDICINE REPORTS 2: 567-572, 2009

Ultraviolet light exposure and skin cancer in the city of Arica, Chile

MIGUEL RIVAS[1], MARÍA C. ARAYA[2], VIVIANA DURÁN[2], ELISA ROJAS[1], JUAN CORTES[3] and GLORIA M. CALAF[4,5]

[1]Departamento de Física, Facultad de Ciencias, Universidad de Tarapacá; [2]Hospital Juan Noé Arica; [3]Departamento de Química, Facultad de Ciencias, [4]Instituto de Alta Investigación, Universidad de Tarapacá, Arica, Chile; [5]Center for Radiological Research, Columbia University Medical Center, New York, NY, USA

Received December 11, 2008; Accepted May 14, 2009

DOI: 10.3892/mmr_00000138

Abstract. An increase in the amount of solar ultraviolet light that reaches the Earth is considered to be responsible for the worldwide increase in skin cancer. Solar ultraviolet B (UVB) light (290-320 nm) has multiple effects that can be harmful to human beings. The city of Arica in Chile receives high UV levels. This can explain the high prevalence of skin cancer in the Arica population. In the present study, pathological reports of skin cancer were collected from an Arica hospital and retrospectively examined to investigate the possible effects of UV radiation. Among the malignant skin tumor types, basocellular and spinocellular carcinomas were more common in men (44.4 and 16.6%, respectively) than in women (24.9 and 10.7%, respectively). Basocellular carcinoma was observed in individuals 40-79 years of age. The incidence of skin cancer significantly increased (P<0.05) between 2000 and 2006 per 100,000 population. The factor of incidence of skin cancer per 100,000 population significantly increased (P<0.05) between 1980 and 2000 in both genders, but was higher in men (0.79-1.99) than in women (0.63-1.56). The results of the study indicate a steady increase in the incidence of skin cancer in Arica, Chile, most probably due to the high levels of ultraviolet light to which individuals are exposed throughout the year, and the cumulative effect of this type of radiation on the skin.

Introduction

An increase in the amount of ultraviolet (UV) light that reaches the Earth is considered to be responsible for the worldwide increase in skin cancer (1). UV light has very high energy, and its interaction with biological material has a detrimental effect (2). For physical and biological reasons, UV light has been divided into 3 regions: ultraviolet A (UVA) (320-400 nm), ultraviolet B (UVB) (290-320 nm) and ultraviolet C (UVC) (100-290 nm) (3). It has been reported that excessive levels of UVA and UVB light have multiple effects, which can be harmful to human beings (1,4-8).

UVB light, a small amount of which reaches the surface of the Earth, is deemed to be the most dangerous of the UV light types (3). The amount of UV light that reaches the Earth is strongly dependent on the ozone layer, with absorption increasing with shorter wavelengths. UVA light has high atmospheric transmission (3), while short-wavelength UVC light is totally absorbed and does not reach the surface of the earth (9).

The progressive depletion of the ozone layer has been observed over the last few decades (3,9-12), and is most prominent in the polar zone at the beginning of spring (13). Atmospheric aerosols and cloudiness also attenuate UVB radiation.

UV solar light has harmful effects on humans and a proven ability to kill unicellular organisms (14). It has been demonstrated that the exposure of DNA to UV light is a key factor in cancer development. The risk of skin cancer varies with altitude, latitude and skin pigmentation, which are all modified by overexposure to UV light (3).

Photons are absorbed by several molecules inside the cell and are either destroyed or recovered. A portion of incident short-wavelength UV light can be absorbed by the stratum corneum of the epidermis, which protects the basal cells of the epidermis. Nonetheless, UV photons can induce skin cancer (15). Lesions originate due to the direct absorption of photons, which induce photochemical reactions in DNA. With the absorption of 320 nm UV light, modifications to DNA bases occur. The more common photoproducts formed are located between two pyrimidines (16).

Research has determined the existence of mechanisms of repair in human skin, such as the excision of nucleotides or the activation of photoreactive enzymes (17). These repair enzymes are capable of correcting the damage induced by UV light. When combined with the dimers of pyrimidines, repair enzymes can monomerize dimers in the presence of UV light

Correspondence to: Dr Gloria M. Calaf, Instituto de Alta Investigación, Universidad de Tarapacá, Calle Antofagasta #1520, Arica, Chile
E-mail: gmc24@columbia.edu

Key words: ultraviolet light index, skin cancer, photons, radiation

Table I. Values for the ultraviolet index in Arica, Chile and assessment of solar risk.

A, Ultraviolet index and solar risk as determined by the World Health Organization and World Meteorological Organization.

UVI	Solar risk
0-3	Low
3-5	Moderate
5-7	High
7-10	Very High
≥11	Extreme

B, Ultraviolet index values measured at solar midday in Arica, Chile.

Months in 2002	Average UVI (± SD) per month
January	10.7±0.6
February	12.6± 1.2
March	11.8±1.7
April	8.8±1.8
May	7.0±0.8
June	5.4±0.9
July	5.2±1.2
August	7.1±1.4
September	10.2±1.0

C, Ultraviolet index determined according to average dose of erythema and maximum time of sun exposure in minutes.

UVI	UV energy per time unit P(μW/cm^2)	Skin type I MED=200 min	Skin type II MED=250 min	Skin type III MED=300 min	Skin type IV MED=450 min
1	2.78	120	150	210	270
2	5.56	58	72	101	130
3	8.34	40	50	70	90
4	11.1	30	37	53	67
5	13.9	24	30	42	54
6	16.7	20	25	35	45
7	19.5	17	21	30	39
8	22.2	15	19	26	34
9	25.0	13	17	23	30
10	27.8	12	15	21	27
11	30.6	11	14	16	25
12	33.4	10	13	15	23
13	36.1	9	12	14	21
14	38.9	9	11	13	19
15	41.7	8	10	12	18

UVI, ultraviolet index; MED, minimal erythema dose. Skin type I, white skin that burns very easily; type II, skin that burns slightly; type III, skin that rarely burns; type IV, dark skin that never burns.

in the 300- to 600-nm region (17), removing the dimers from the human skin within minutes.

Melanocytes are a type of cell which protect the eyes from the photoallergenic and phototoxic reactions of UVA. The cumulative effects of UVA can be as harmful as UVB (9). UVA radiation contributes to as much as 15% of erythematosis at midday (18), initially inducing a tan with subsequent melanogenesis, such as burns or erythemogenesis (18). The effect of UVA on this alteration is limited in comparison to that of UVB (600-1000 less effective) (9). Experimental studies have found that individuals with light complexions require between 20 and 100 J/cm^2 of UVA to induce redness, an erythemogenic and melanogenic reaction (19). The UVA and UVB average intensities that reach the surface of the Earth are 5-6 and 0.3-0.5 mW/cm^2, respectively (9). The ultraviolet index (UVI) is obtained by multiplying the value of erythemic radiance in W/m^2 by a factor of 40 by agreement (9), which provides an important value of reference.

Arica is a city located in the subtropical zone of northern Chile (25 meters above sea level; latitude 18˚49'S, longitude 70˚19'W). It has a microclimate characterized by stable meteorological conditions throughout the year, including low precipitation (<5 mm per decade), predicable winds, a high percentage of clear sky days and high ground reflectivity due to the presence of light sand. Due to its location near sea level, the population performs a great number of activities outdoors. A national UV network exists in Chile that informs the population regarding the daily value of UVI from the city of Arica to Antarctica.

The aim of the present study was to determine the relationship between the UVI and the risk of skin cancer.

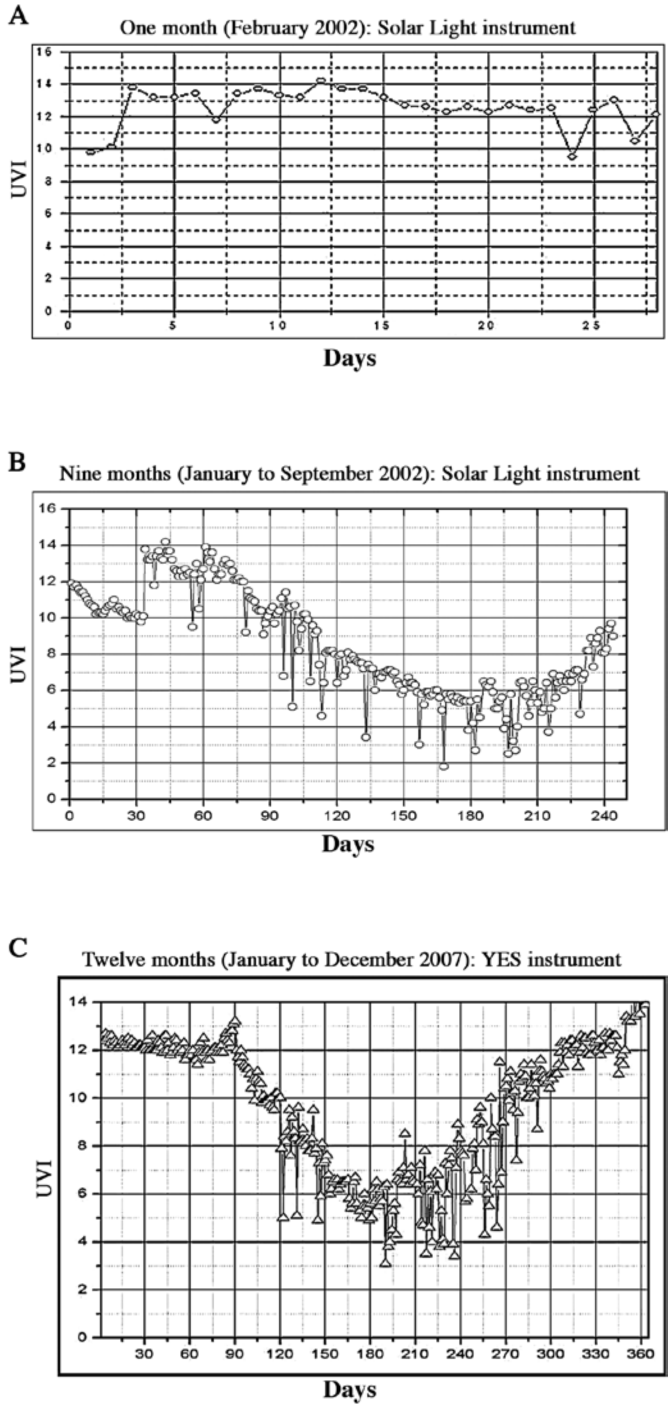

Figure 1. Experimental values for the ultraviolet index obtained from measurements of UV light taken over (A) 1 month, (B) 6 months and (C) 12 months in Arica, Chile using a wide band solar light biometer (2002) and a UVB-1 ultraviolet pyranometer 501 (YES) instrument (2005-2007).

Materials and methods

UVI measurements were classified according to the World Health Organization (WHO) parameters, and were taken in accordance with the regulations of the World Meteorological Organization (WMO) in the city of Arica.

Experimental measurements were obtained using a wide band solar light biometer during 2002 and a UVB-1 ultraviolet pyranometer 501 (YES) between 2005 to 2007, calibrated according to WMO criteria. This latter instrument measures global solar UVB irradiance or the power per unit area of UVB radiation on a horizontal surface received from the sky.

The study was carried out at the Laboratory of UV Solar Radiation of the University of Tarapacá (Arica, Chile). To explain the possible effect of UV radiation on skin cancer, revised pathological reports recorded between 1989 and 2006 were collected from Juan Noé Crevani Hospital (Arica, Chile).

Results

The present study investigated the characteristics of UV light in relation to skin cancer in a population from Arica, Chile. Table IA shows the relationship between UVI value and skin cancer risk due to sun exposure, indicating that the UVI varied from 0 to ≥11, corresponding to a variation in energy from 100 to 1100 J/m^2/h measured at ground level. Table IB shows the average maximum daily values of UVI from January to September, 2002. Fig. 1A shows experimental data regarding the UVI over one month (February, 2002), considered to have the maximum daily level of solar UVI. Fig. 1B shows the maximum daily value of UVI over 9 months (January-September 2002). Notably, even with clear skies, UVI values in winter (June and July) were classified as high. The rest of the year fluctuated between very high or extreme values in the summer (beginning of January to end of March). Fig. 1C shows the maximum daily value of UVI over 12 months (January-December, 2007). According to these measurements, the UVI fluctuated between 10.7 and 12.6 from January to March (summer) and between 5.2 and 10.2 during the winter and part of spring. Table IC lists UVI values in terms of the maximum time of exposure required to induce erythema according to skin type. The average dose of erythema was assessed in terms of the amount of radiation required for skin redness to be induced, skin type, intensity of radiation and time of exposure. Based on these values, the maximum time of sun exposure that does not cause redness was calculated.

Table II provides information regarding skin cancer reported in the population of Arica. Table IIA documents the benign and malignant skin tumors of patients according to pathological type and gender, collected between 1978 and 1988. Basocellular carcinoma was more common in men (44.4%) than in women (24.9%); spinocellular carcinoma was more common in men (16.6%) than in women (10.7%), while melanoma was more common in women (2.4%) than in men (1.2%). Table IIB lists the total number of skin tumors of patients according to pathological type and age, collected between 1978 and 1988. A high incidence of basocellular carcinoma was found in individuals between the ages of 40 and 79 years; for spinocellular carcinoma, the highest incidence occurred between the ages of 50 and 79 years, and for malignant melanoma, between 50 and 69 years. Table IIC lists the skin tumors of patients according to pathological type, collected between 1989 and 1993. Of the malignant types, basocellular carcinoma (62.2%) was the most common in comparison to spinocellular carcinoma (31.6%) and malignant melanoma (6.2%). The mortality rate induced by skin cancer per 100,000 population significantly increased ($P<0.05$) between 1980 and 2000 in both genders, though it was higher in men (factor 0.79-1.99) than in women (factor 0.63-1.56) (Table IID). Table IIE documents

Table II. Data regarding skin tumors from pathological reports on patients treated at Juan Noé Crevani Hospital, Arica, Chile.

A, Percentage of skin tumors collected between 1978 and 1988 according to pathological type and gender.

	No. male patients (%)	No. female patients (%)
Basocellular carcinoma	75 (44.4)	42 (24.9)
Spinocellular carcinoma	28 (16.6)	18 (10.7)
Malignant melanoma	2 (1.2)	4 (2.4)

B, Number of skin tumors collected between 1978 and 1988 according to pathological type and age.

	Age (years)						
Tumor type	No. of cases (%)	<40	40-49	50-59	60-69	70-79	>79
Basocellular carcinoma	117 (69.2)	6	22	25	24	26	14
Spinocellular carcinoma	46 (27.2)	6	3	9	11	8	9
Malignant melanoma	6 (3.6)	0	1	2	2	1	0

C, Number of skin tumors collected between 1989 and 1993 according to pathological type and year.

	Year					
Tumor type	1989	1990	1991	1992	1993	Total (%)
Basocellular carcinoma (% cases)	6	15	16	16	8	61 (62.2)
Spinocellular carcinoma	3	6	8	7	7	31 (31.6)
Malignant melanoma	0	0	2	1	3	6 (6.2)

D, Incidence factor per 100,000 population of skin tumors collected in 1980 and 2000.

	Incidence factor per 100,000 population.	
Gender	Year 1980	Year 2000
Male	0.79	1.99
Female	0.63	1.56

E, Incidence of skin cancer per 100,000 population from 2000 to 2006.

Year	No. of cases	Incidence per 100,000 population
2000	35	17.6
2001	32	15.9
2002	31	15.3
2003	47	23.0
2004	80	38.7
2005	58	29.4
2006	64	32.4

benign and malignant skin tumors from patients according to pathological type, collected between 2000 and 2006. The incidence of skin cancer significantly increased ($P<0.05$) from 2004 to 2006 per 100,000 population, reaching 38.7, 29.4 and 32.4 each year, respectively. Representative images of the carcinomas are shown in Fig. 2.

Figure 2. Representative images of skin cancer registered in the city of Arica. A-N,S,V, basocellular carcinoma; O-Q,T, spinocellular carcinoma; R,U,W,X, actinic keratosis; Y, melanoma.

Discussion

The present study examined skin cancer in relation to average maximum daily ultraviolet index (UVI) values measured between 2002 and 2007 in Arica, Chile. The average maximum daily value of UVI was determined based on measurements taken on clear sky days, obtained daily and monthly. Very high levels of UVI were noted throughout the year due to the subtropical location of Arica, which is in a microclimate with a high number of clear sky days and the presence of a desert in the area, causing ground reflectivity.

The experimental data indicate that UVI values fluctuated from 5 to 7 (high) in the winter (June and July) and from 7 to 10 (very high) in January, April, May, August and September, and reached 11 or more (extreme) in February and March. Values for the remainder of the year fluctuated between high, very high and extreme, at the beginning of November and the end of February. These results were corroborated using a UVB-1 ultraviolet pyranometer during 2005. According to the WMO, values were higher than 11 (extreme) on 24 of 28 days. Data from cloudy days in February of 2002 were not taken into account. It was found that the month of February had an average UVI of 14, whereas from January to September 2002 the UVI value was 11 (extreme). Even in June (winter) the average value was 6 (high). Throughout the rest of the year, the UVI fluctuated between very high and extreme, between January and the end of March.

The data indicate that the incidence of skin cancer in Arica increased from 17.6 to 32.4 between 2000 and 2006 per 100,000 population. This increase is related to the high UV levels that reach the Earth's surface at this location. It has been reported that approximately 3% of the ozone layer is depleted every 10 years at the latitude of Arica (20), which may contribute to an increase in UV levels. Since erythermal doses are accumulative, individuals living in the northern part of Chile have a greater risk of skin cancer than those living in the southern areas of the country. According to the WHO, over the last 10 decades the incidence of skin cancer has increased by 8.3%. This is likely also the result of changes in the habits of the population.

In conclusion, there has been a steady increase in the incidence of skin cancer in the Arica, Chile population, most likely caused by the high levels of UVI to which individuals are exposed throughout the year, as well as by the cumulative effect of solar UV radiation on the skin and changes in the habits of the Arica population in recent years.

Acknowledgements

The authors are grateful to Danissa Barahona for secretarial support. This study was supported by grant MECESUP/UTA 2.

References

1. Van der Leun JC, De Gruijl FR, Tevini M and Worrest RC: Ultraviolet carcinogenesis: environmental effects of ozone depletion. Recent Results Cancer Res 128: 263-267, 1993.
2. Parrish JA, Anderson F, Urbach F and Pitts D: UVA, Biological Effects of Ultraviolet Radiation With Emphasis On Human Response To Longwave Ultraviolet. Plenum Press, New York, p151, 1978.
3. Madronich S, Mckenzie RI, Bjorn LO and Caldwell MM: Changes in biologically active ultraviolet radiation reaching the Earth's surface. J Photochem Photobiol 46: 5-19, 1998.
4. Jagger J: Solar UV Actions On Living Cells. Praeger, New York, 1985.
5. Godlee F: Dangers of ozone depletion. BJM 303: 1326-1328, 1992.
6. Jones RR:. Ozone depletion and cancer risk. Lancet 2: 443-446, 1987.
7. Jones M: Dermatological effects from years in the sun: compouding opportunities. Int J Pharm Comp 10: 336-342, 2006.
8. Sánchez F: Consideraciones sobre la capa de ozono y su relación con el cáncer de piel. Rev Méd Chile 134: 1185-1190, 2006.
9. WMO (1994): Report of the WMO meeting of experts on UV-B measurements, data quality and standardization of UV indices. Report no. 36, 1994.
10. WMO (1995): Scientific assessments of ozone depletion: 1994. World Meteorological Organization, Global Ozone Research and Monitoring Project. Report no. 37, 1994.
11. WMO (1999): Scientific assessment of ozone depletion: 1998. World Meteorological Organization. Global Ozone Research and Monitoring Project. Report no. 44, 1998.
12. Gleason JF, Bhartia PK, Herman JR, et al: Record low global ozone in 1992. Science 260: 523-526, 1993.
13. Herman J, Bhartía P, Zemke J, Ahmad Z and Larko D: UVB increases (1979-1992) from decreases on total ozone. Geophys Rev Lett 23: 2117-2120, 1996.
14. Hargreaves A, Taiwo FA, Duggan O, Kirk SH and Ahmad SI: Near-ultraviolet photolysis of beta-phenylpyruvic acid generates free radicals and results in DNA damage. J Photochem Photobiol B 89: 110-116, 2007.
15. Cooper SJ and Bowden GT: Ultraviolet B regulation of transcription factor families: roles of nuclear factor-kappa B (NF-kappaB) and activator protein-1 (AP-1) in UVB-induced skin carcinogenesis. Curr Cancer Drug Targets 7: 325-334, 2007.
16. Rünger TM and Kappes UP: Mechanisms of mutation formation with long-wave ultraviolet light (UVA). Photodermatol Photoimmunol Photomed 24: 2-10, 2008.
17. Pathak MA, Riley FC and Fitzpatrick TB: Melanogenesis in human skin following exposure to long-wave ultraviolet and visible light. J Invest Dermatol 39: 435-443, 1962.
18. Parrish JA, Jaenicke KF and Anderson RR: Erythema and melanogenesis action spectra of normal human skin. Photochem Photobiol 36: 187-191, 1982.
19. Szotz MJ: Estudio realizado en los certificados médicos de defunción. Rev Chilena Dermatología 19: 173-177, 2003.
20. Isaksen ISA: Stratospheric ozone depletion and UV-B changes. In: Solar Ultraviolet Radiation: Modelling Measurements, and Effects. Zerefos CS and Bais AF (eds). Springer, Berlin, Heidelberg, pp13-21, 1997.