

COMPARISON OF ANTIOXIDANTS LEVEL IN TWO AREAS WITH HIGH AND LOW LEVELS OF ULTRAVIOLET B IN ISFAHAN PROVINCE

Mohammad-Bagher Behyar⁽¹⁾, Shahram Moradi⁽²⁾, Mohammad-Ali Nilforoushzadeh⁽³⁾,
Leila Shirani-Bidabadi⁽⁴⁾

Abstract

BACKGROUND: Ultraviolet radiation can contribute to various damages to body organs especially to skin. However, the effect of Ultraviolet B on the function of viruses, immunologic changes and antioxidants levels which protect human skin health are still unknown. We aimed to find the harmful level of UVB that can be helpful in taking preventive action against the increasing depletion of Ozone layer.

METHODS: In this cross-sectional study conducted in Isfahan population, two areas with high and low levels of UVB radiation were investigated based on the information obtained from Meteorological Office. Samples in each area were selected by a cluster-sampling method. 250 persons were assigned to each group. The antioxidant capacity and glutathione peroxide level of samples were measured. Data were analyzed with Student t test by using SPSS software.

RESULTS: The mean (\pm standard deviation) of Malondialdehyde in high and low UVB radiation areas were 2.8 ± 0.32 and 1.65 ± 0.38 nmoles/ml, respectively ($P < 0.001$). Mean (\pm standard deviation) of antioxidants capacity levels were 81.6 ± 2.36 $\mu\text{mol/l}$ in patients living in low UVB radiation area and 76.5 ± 2.6 $\mu\text{mol/l}$ in patients living in high UVB radiation area ($P < 0.0001$). Glutathione Peroxide levels were 38.2 ± 1.7 and 35.3 ± 1.9 $\mu\text{mol/l}$ in areas with low and high UVB radiation respectively ($P < 0.0001$).

CONCLUSION: Findings of this study indicates that the amount of free radicals and antioxidant capacity in high UVB radiation areas are less than areas with low levels of UVB radiation. Therefore, it is suggested that necessary considerations should be taken into account for the residents of such areas in order to reduce its health damages.

Keywords: *Ultraviolet B, antioxidant, malondialdehyde, glutathione peroxide.*

ARYA Atherosclerosis Journal 2009, 5(1): 7-10

Date of submission: 12 Jun 2009, *Date of acceptance:* 22 Feb 2009

Introduction

UVB radiation is recognized as one of the major factors of developing melanoma and non-melanoma skin cancers. It is shown that 1 percent depletion of Ozone layer is associated with 2.7 percent increase in non-melanoma skin cancers.¹ However, the mechanisms of viruses and immunologic changes due to UVB radiation have not been known yet.²⁻⁶ Some studies have investigated the effect of antioxidants on plasma and blood samples of patients with actinic keratosis and basal cell carcinoma rather than their skin tissues. In the same vain, these studies indicated that plasma antioxidants (ascorbic acid, alpha tocopherol, glutathione) have significantly decreased in

these patients.⁷ However, the relation between UVB dosage, the duration of radiation and the kinds of exposure to UVB as well as the emergence and development of tumor have not been known yet. Considering the role of antioxidants in skin health and the increasing trend of industrialization in mass cities which can contribute to the more depletion of Ozone layer, it would be expected that UVB radiation is increasing as well. The aim of this study was finding the harmful level of UVB that can direct the preventive actions against more depletion of Ozone layer on the part of authorities.

1) Assistant Professor, Meteorological Research Center, Meteorological Office, Tehran, Iran.

2) Specialist in Infectious Diseases, Skin Diseases and Leishmaniosis Research Center, Isfahan University of Medical Sciences, Isfahan, Iran.

3) Assistant Professor of Dermatology, Tehran University of Medical Sciences, Tehran Skin and Leprosy Research Center, and Isfahan Skin Diseases and Leishmaniosis Research Center, Isfahan University of Medical Sciences, Isfahan, Iran.

4) Medical Entomology, Skin Diseases and Leishmaniosis Research Center, Isfahan University of Medical Sciences, Isfahan, Iran.

Corresponding author: Shahram Moradi, E-mail: shahram50047@yahoo.com

Materials and Methods

In an analytic cross-sectional study in Isfahan urban population, Isfahan province was divided into two areas based on the information obtained from Meteorological Office; low level UVB radiation and high level UVB radiation areas. Considering at least 20% difference in the level of antioxidant in the susceptible population in these two areas and the confidence interval of 95% as well as the test reliability of 90%, in each area samples were selected by a cluster-sampling method. 250 samples were assigned to each group. Two groups were matched considering sex and age (> 20 years), did you match your subjects considering their job? It is important. 5cc blood sample was taken after getting participants' permission. Blood samples were sent to the laboratory of Sedigheh Tahere Research Center Complex. Antioxidant capacity and glutathione peroxide level of samples were estimated. Stress oxidants test was done by spectrophotometer with amplitudes of 532 nm, 234 nm, and 540 nm.^{8,9} UVB radiation pattern of these areas in the year 2004 was estimated based on the information of Isfahan Meteorological Office. Data was entered into SPSS version 13 and was analyzed by student t-test. In addition, the effect of sex and types of occupation on these two groups was investigated by the regression test. P value under 0.05 was considered as significant.

Results

This study did not show any significant difference between the malondealdehyde level of men proportionate to women (P = 0.11). The mean level of malondealdehyde was 2.78 ± 0.36 nmoles/ml for residents of the area with high UVB radiation level (Figure 1).

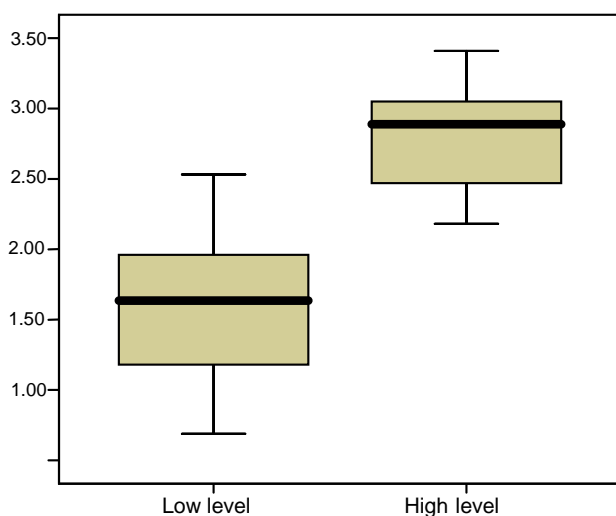


Figure 1. Mean (\pm SD) of malondealdehyde in different areas of Isfahan province with different amounts of UVB in 2004

Also, their mean antioxidant capacity was 76.5 ± 2.6 μ mol/l (Figure 2) and their mean glutathione peroxide was 35.3 ± 1.9 μ mol/l (Table 1 and Figure 2).

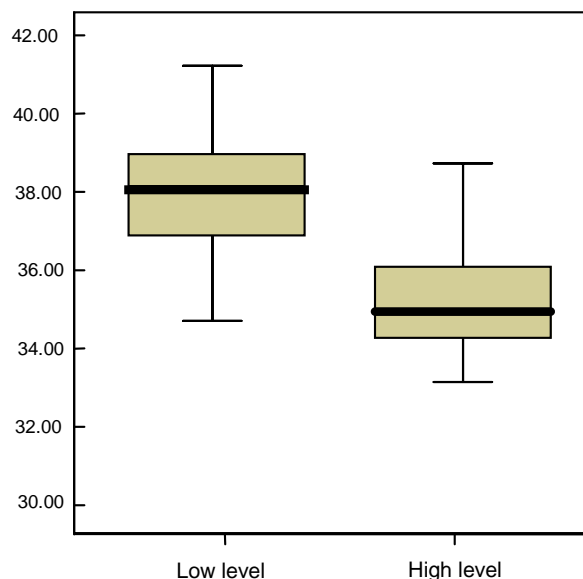


Figure 2. Mean (\pm SD) of glutathione level in residents of high and low UVB

Table 2 and Figure 4 indicate the UVB variation in the two areas with high and low level of UVB radiation.

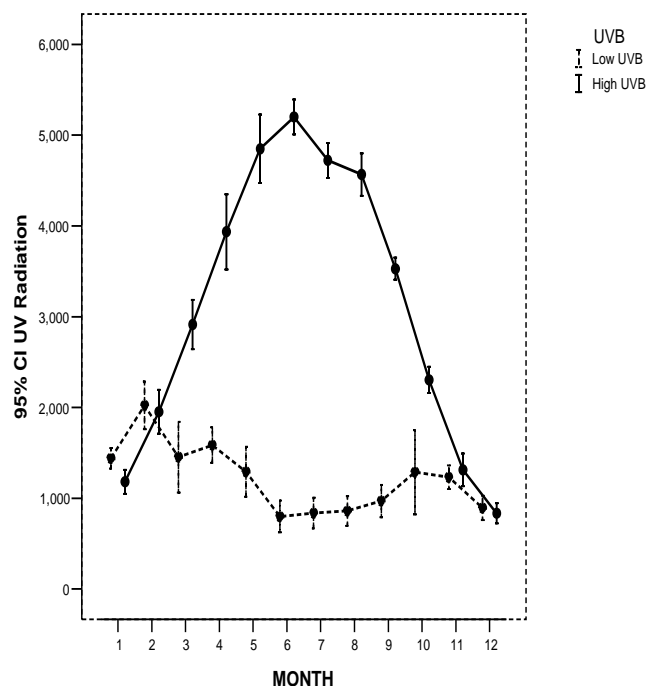


Figure 4. UVB variation in different areas of Isfahan province in different months in 2004

Table 1. Comparison of mean (\pm SD) of demographic features and stress oxidant in areas with high and low levels of UVB radiation

UVB	High UVB area	Low UVB area	P.value
Age (years)	31.3 \pm 9.03	32 \pm 597.56	0.60
Malondealdehyde (μ mol/l)	2.8 \pm 0.32	1.65 \pm 0.38	< 0.0001
Antioxidant capacity (μ mol/l)	% 76.5 \pm %2.6	% 81.6 \pm %2.36	< 0.0001
Glutathione peroxide (μ mol/l)	35.1 \pm 1.9	38.2 \pm 1.7	< 0.0001

Table 2. UVB levels (nm) in 2004

Year	Mean	SD	Minimum	Maximum
High UVB	2392	1410	211	4820
Low UVB	3390	1484	311	5475

Discussion

The results of our study showed that the mean and standard deviation of antioxidants capacity of residents of the low UVB radiation was significantly higher than the residents of high UVB areas (81% vs 76%). The mean and standard deviation of glutathione peroxide level in persons exposed to low level of UVB was 38.2 and in persons exposed to high level of UVB were 35.3 μ mol/l which show a significant statistical difference. In the same vain, Malondealdehyde level of persons exposed to high level of UVB was significantly higher than those exposed to low level of UVB. In addition, the study reveals that the indicators of stress oxidants in persons exposed to high level of UVB was significantly lower than others.

Some studies investigate the effect of antioxidants level in plasma and blood samples of people with actinic keratosis and basal cell carcinoma rather than their skin tissues. These studies indicated that plasma antioxidants levels (ascorbic acid, alphas-tocopherol, glutathione) decreased significantly.⁷ Kobayashi and his colleagues in a study, which was done on basal cell carcinoma, showed that MnSOD and CuZnSOD decreased in these patients which can be contributed to the disruptive effect of UVB on antioxidants.¹⁰ In addition, in a study by Borello et al, they concluded that the amount of CuZnSOD in squamous cell carcinoma decreased; however, such a decrease is not seen in basal cell carcinoma. The function of glutathione did not show any significant difference.¹¹

Different studies on the effect of UVB radiation indicated the carcinogenic effect of UVB on tissues so that 1% depletion in Ozone layer is associated with 2.3% increase in UVB radiated to body¹² and as a result, 4% increase in skin cancers.¹³ In addition to mechanical defense of skin layers which act against this risk factor, antioxidants defense system acts as a strong network in order to nullify free oxygen due to

UVB radiation. Antioxidants control the lipid peroxidation which is activated by UVB and as a result, prevent from chronic damages.¹⁵ Despite the antioxidant activity level of skin cells, the presence of this defense system can control the harmful effects of UVB radiation. Studies indicate the that increase in UVB dose and duration of radiation would increase the lipid peroxidation level which is one of the cell's damaging factors.^{16,17} In general, the increase in UVB dose is associated with the inability of antioxidant defense system to act against the UVB. Table 1 indicates the antioxidants variation against high UVB radiation.¹⁸⁻²⁰ Considering the harmful effects of UVB, in addition to preventing from the increasing depletion of Ozone layer, defense system of body must be strengthened as well which includes reduction of exposure to UVB and promoting the antioxidants network.

References

1. Kelfkens G, de Gruijl FR, van der Leun JC. Ozone depletion and increase in annual carcinogenic ultraviolet dose. *Photochem Photobiol* 1990; 52(4): 819-23.
2. Bentham G. Depletion of the ozone layer: consequences for non-infectious human diseases. *Parasitology* 1993; 106(Suppl): S39-S46.
3. Garssen J, Goettsch W, de Gruijl F, Slob W, van Loveren H. Risk assessment of UVB effects on resistance to infectious diseases. *Photochem Photobiol* 1996; 64(2): 269-74.
4. Norval M, Garssen J, van Loveren H, el Ghorr AA. UV-induced changes in the immune response to microbial infections in human subjects and animal models. *J Epidemiol* 1999; 9(Suppl 6): S84-S92.
5. Young AR. The biological effects of ozone depletion. *Br J Clin Pract Suppl* 1997; 89: 10-5.
6. Thiele JJ, Dreher F, Maibach HI, Packer L. Impact of ultraviolet radiation and ozone on the transepidermal water loss as a function of skin temperature in hairless mice. *Skin Pharmacol Appl Skin Physiol* 2003; 16(5): 283-90.

7. Vural P, Canbaz M, Selcuki D. Plasma antioxidant defense in actinic keratosis and basal cell carcinoma. *J Eur Acad Dermatol Venereol* 1999; 13(2): 96-101.
8. Esterbanrc H, Chessman KH. Determination of ajahidic lipid peroxidation product: malondialdehyde and 4 hydroxnoneal. *Meth enzimal* 1990; 12: 186-407.
9. Chajes V, Sattler W, Stultschnig M, Kostner GM. Photometric evaluation of lipid peroxidation products in human plasma and copper oxidized low density lipoproteins: correlation of different oxidation parameters. *Atherosclerosis* 1996; 121(2): 193-203.
10. Kobayashi T, Matsumoto M, Iizuka H, Suzuki K, Taniguchi N. Superoxide dismutase in psoriasis, squamous cell carcinoma and basal cell epithelioma: an immunohistochemical study. *Br J Dermatol* 1991; 124(6): 555-9.
11. Borrello S, Seccia A, Galeotti T, Bartoli GM, Farallo E, Serri F. Protective enzymes in human epidermal carcinomas and psoriasis. *Arch Dermatol Res* 1984; 276(5): 338-40.
12. Zurer PS. Ozone depletion's recurring surprises challenge atmospheric scientists. *Chem Eng News* 1993; 71(8): 18.
13. Moan J. UV-A radiation, melanoma induction, sunscreens, solaria and ozone reduction. *J Photochem Photobiol B* 1994; 24(3): 201-3.
14. Black HS, Chan JT. Suppression of ultraviolet light-induced tumor formation by dietary antioxidants. *J Invest Dermatol* 1975; 65(4): 412-4.
15. Shindo Y, Witt E, Han D, Epstein W, Packer L. Enzymic and non-enzymic antioxidants in epidermis and dermis of human skin. *J Invest Dermatol* 1994; 102(1): 122-4.
16. Shindo Y, Witt E, Han D, Packer L. Dose-response effects of acute ultraviolet irradiation on antioxidants and molecular markers of oxidation in murine epidermis and dermis. *J Invest Dermatol* 1994; 102(4): 470-5.
17. Schallreuter KU, Wood JM. Free radical reduction in the human epidermis. *Free Radic Biol Med* 1989; 6(5): 519-32.
18. Maisuradze VN, Platonov AG, Gudz' TI, Goncharenko EN, Kudriashov I. Effect of ultraviolet rays on lipid peroxidation and various factors of its regulation in the rat skin. *Nauchnye Doki Vyss Shkoly Biol Nauki* 1987; (5): 31-5. [In Russian].
19. Pence BC, Naylor MF. Effects of single-dose ultraviolet radiation on skin superoxide dismutase, catalase, and xanthine oxidase in hairless mice. *J Invest Dermatol* 1990; 95(2): 213-6.
20. Picardo M, Zompetta C, De Luca C, Amantea A, Faggioni A, Nazzaro-Porro M, et al. Squalene peroxides may contribute to ultraviolet light-induced immunological effects. *Photodermatol Photoimmunol Photomed* 1991; 8(3): 105-10.