UVA ERYTHEMA IN SKIN: IS IT A SUNBURN?

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Specimens obtained from human skin following the induction of erythema responses with long ultraviolet (UVA) and with middle ultraviolet (UVB) radiation alone were compared histologically to determine whether the effects of these two portions of the electromagnetic spectrum were the same. It was found that, except for similar dermal inflammatory changes, the effects of UVA were not the same as those of UVB; characteristic epidermal sunburn damage was induced by the latter, but not by the former.

It has been said that the effects of UVB radiation (290–320 nm) on the skin may result in acute sunburn, premature aging, keratoses, and malignancies. On the other hand, it has been felt that UVA radiation (320–400 nm) is relatively harmless or may induce protection in skin against the effects of UVB [1–8]. Willis, Kligman, and Epstein [9] and van der Leun and Stoop [10] in separate studies reported that, contrary to accepted beliefs, the effects of UVA radiation on skin are neither harmless nor helpful but can markedly enhance the sunburning effects of UVB radiation. This enhancement was defined by Willis et al as a photoaugmentation phenomenon. Since the publication of these findings, Ying, Parrish, and Pathak [11] have reported that the photoaugmentation phenomenon may be, in reality, a photoadditive effect, and implied that the effects of UVA and UVB on skin may be the same, with UVA being less potent in its ability to induce a sunburn. An effort was made to reexamine the photoaugmentation hypothesis and to determine whether the effects of UVA and UVB radiation, when given in combination, may be additive rather than augmentative. An erythema response was induced using UVA radiation alone and the skin was examined histologically to determine whether the erythema was indeed a sunburn reaction.

MATERIALS AND METHODS

Subjects. These were 20 normal adult volunteers, 8 females and 12 males. The lower backs, buttocks, and thighs served as the test sites.

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Abbreviations:
MED: minimal erythema dose
UVA: long ultraviolet (320–400 nm)
UVB: middle ultraviolet (290–320 nm)

Light sources. Radiation of 290 to 400 nm was obtained from a 1600-watt xenon solar-simulating lamp as previously described [10]. Radiation of 320 to 400 nm (UVA) was achieved by adding a Schott WG-345 filter to the above system; this filter gives a transmission of 50% of its peak at 345 nm and cuts off practically all rays below 320 nm (less than 0.01% transmission at 315–320 nm). Narrow spectral bands (20 nm) of radiation peaking at 310 nm (i.e., 300–320 nm) and 360 nm (i.e., 350–370 nm) were obtained from the same lamp source filtered via a Bausch & Lomb high-intensity grating monochromator.

Energy measurements were obtained using an Eppley photocell and Keithley 149 millimicrovoltmeter. Spectral characteristics were obtained using an International Light Spectroradiometric System. Energy in the 320 to 400 nm range delivered to the surface of a test site was 252 mw/cm². Energy in the 290 to 400 nm range was 343 mw/cm². Monochromatic light energies at 310 nm and 360 nm were 7 mw/cm² and 11 mw/cm², respectively.

EXPERIMENTAL PROCEDURES AND RESULTS

Separate series of 1-cm² test sites on each subject were exposed to 1, 2, and 3 times the previously established minimal erythema doses (MEDs) for each type of radiation. Forty-eight hours later all sites were evaluated and biopsy specimens obtained. The biopsy samples were fixed in formalin, sectioned, and stained with hematoxylin and eosin. The average dose requirement for minimal erythemas to the various types of light were as follows: 290–400 nm = 14 joules, 320–400 nm = 91 joules, 310 nm = 0.07 joules, and 360 nm = 26 joules.

Histologically, there was a striking difference between sites receiving UVB as compared to UVA radiation. This difference was manifested mainly in the epidermis. Specimens that received 2 to 4 MEDs of UVB radiation showed the characteristic epidermal changes of a severe sunburn reaction. These consisted of numerous sunburn cells (recognized by their eosinophilic cytoplasm and pyknotic nuclei), vesication, and liquification degeneration of the basal layer in each of the sections examined (Fig. 1). In the upper dermis, there was
FIG. 1. Three-MED exposure to UVB. Microvesication and many sunburn cells are present in this section (x 230).

FIG. 2. Three-MED exposure to UVA. No epidermal changes (x 230).

A perivascular infiltrate of lymphocytes, a few scattered histiocytes, and occasional polymorphonuclear leukocytes.

In contrast, epidermis of specimens receiving 2 to 4 MEDs of UVA radiation showed none of the changes characteristic of a sunburn, and appeared unaffected by the radiation (Fig. 2). There was a dermal infiltrate indistinguishable in cell type and location from that described for the UVB-exposed specimens. In some instances, this infiltrate was more dense than that in the corresponding specimens that received UVB.

DISCUSSION

We believe that this study illustrates that there is a basic difference in the damage done by UVA as opposed to UVB irradiation. Miescher [12] reached a similar conclusion nearly two decades ago when he reported that "whereas epidermal cellular cytotoxic disturbances are noted in the classical reaction provoked by short ultraviolet rays, the long ultraviolet rays bring about principally an injury of the capillaries, with necrosis of the endothelial cells."

Despite the fact that both UVA and UVB can induce clinically similar erythema responses, histologically the UVA response does not resemble the UVB sunburn reaction. This finding further correlates with our previously published data which showed that the effects of UVA on DNA synthesis and repair and on protein synthesis inhibition were entirely different from those due to UVB radiation exposure [10]. Based on these findings, it becomes apparent that the enhancement of UVB sunburn damage by either preexposure or postexposure to UVA is not additive since UVA erythema is not a sunburn.

REFERENCES