

Wrinkles from UVA Exposure

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Research on mechanisms of wrinkle formation and methods of prevention

Recently, the increase in UV irradiation due to progressive depletion of the Earth's ozone layer has become a serious human health concern. In Japan, the frequency of skin cancer of persons living in sunny regions has showed a clear increasing trend from the 1970s to the 1990s. Solar keratosis, a precancerous condition, is also increasing rapidly.

Many UV protective products are now available on the market. However, regardless of the amount of the product used, completely protecting against UV radiation under the intensity of the midsummer sun is difficult. In autumn, winter and spring, UV protective cosmetics are rarely used, making adequate protection impossible.

UV radiation reaching the Earth's surface may increase substantially in the future. What is needed for protection may not only be sunscreen products with high SPF, but also anti-photoaging products, which can be used irrespective of region, season, lifestyle, gender and age.

Of the effects of UV irradiation on the Earth's surface, skin damage caused by UVB irradiation has been extensively investigated. However, the mechanisms of skin damage and photoaging caused by long-term exposure to UVA irradiation have not yet been sufficiently elucidated.

This study investigated the mechanism of skin sagging caused by long-term exposure to UVA radiation, in hope that better understanding of the mechanism will assist the development of new concepts in skin care and new products for the prevention of photoaging.

Chronic UVA and Sagging

We used hairless mice SKh:HR-1^a as our animal model for chronic UVA damage to skin, modifying the method of Bissett et al.¹ At the start of experiments, the mice were nine to 10 weeks old.

We produced UVA radiation by passing output from eight lamps^b through a 5 mm thick glass filter to cut out wavelengths below 320 nm. Animals housed individually in glass boxes with walls 2 mm thick were irradiated five times weekly for six months with

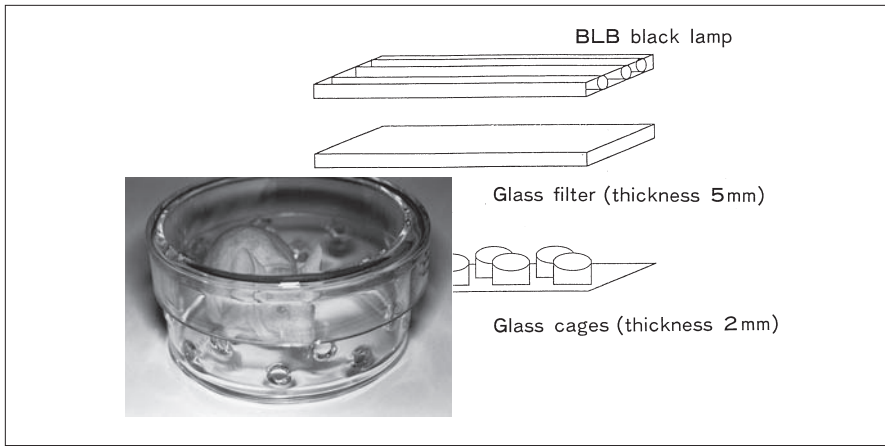


Figure 1. Experimental apparatus

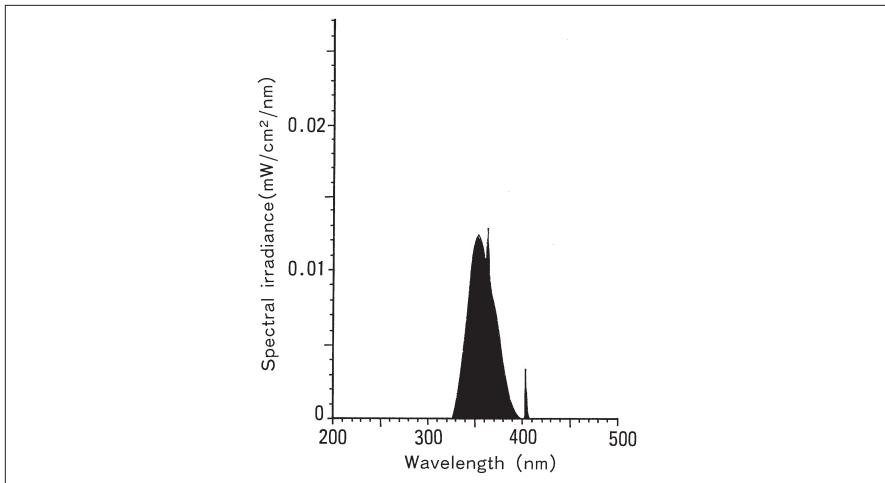


Figure 2. Spectrum of UVA light from the Toshiba BLB lamp passed through a 5 mm thick glass filter

27 J/cm² UVA radiation per daily exposure. The irradiation energy was determined using a radiometer.^c The radiation spectrum through the glass boxes was determined using a spectroradiometer^d (Figures 1 and 2).

Visual examination: The degree of sagging was graded on a scale of 0-3 (Table 1), according to the method of Bissett et al.¹

After long-term exposure (24 weeks) to UVA radiation, hairless mice exhibited large, loose folds (sagging)¹ on the dorsal and lateral skin. At three months after the first exposure, the dorsal skin became blanched and the skin texture became rough. After 6 months, there was complete blanching of the skin, nodular texture

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^bToshiba-BLB, Toshiba, Tokyo, Japan

Table 1. Scale for grading sagging, 0 to 3

- 0 = Pale pink color, fine striation (head to tail), no loose folds
- 1 = Slight blanching, slight fine striation, no loose folds
- 2 = Complete blanching, no fine striation, slight loose folds
- 3 = Complete blanching, no fine striation, large loose folds



Figure 3. Visible changes at 24 weeks
 A = UVA irradiated hairless mouse
 B = nonirradiated hairless mouse

and loose folds (Figures 3, 4). Figure 5 shows the time-course for development of sagging.

Histological examination: On the last day of the experimental period, biopsies of the dorsal skin were performed on five animals per group. We fixed tissue specimens in formalin, embedded them in paraffin and sectioned to 5 μm in thickness. The sections were stained with H-E, van Gieson's and Luna stains, then examined for several parameters. One of the tissue specimens was reserved for transmission electron microscopy (TEM).

Histological and TEM findings after exposure to UVA for 24 weeks were difficult to analyze. Marked thickening of the epidermis and dermis was observed. In the dermis, an increase in mast cells and definite hypertrophic cysts were observed. In addition, partial infiltration into the dermis of inflammatory cells, including polymorphonuclear leukocytes (PMNs), was observed. In the upper layer of the dermis, there was a marked decrease in collagen fibers. As for elastin fibers, aggregations were intermingled with areas of partial absence (Figures 6 through 14).

Determination of elastase activity: We removed the subcutaneous tissue from circular, dorsal skin tissue specimens (2.1 cm in diameter) not used in the histological

^cTopcon-UV radiometer 305/365 DII, Topcon, Tokyo, Japan

^dUshio spectroradiometer USR-20B, Ushio Denki, Yokohama, Japan