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Photoaging

Manifestations, Prevention, and Treatment

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Long referred to as "premature aging," the striking skin changes in sun-exposed areas of the face, hands, and arms are increasingly being shown to be quite distinct from those in aged protected skin.[5, 27, 40] In a sense, this "new" knowledge is a rediscovery. The late nineteenth century dermatologists, Unna and Dubreuilh, recognized the devastating effects of sunlight when they compared the skin of farmers and sailors with that of indoor workers. It is true that serious sunworshipers look prematurely aged, even at age 50. Photophobes, however, can reach the ninth decade with smooth, unblemished skin showing only some thinning, looseness, and a deepening of normal expression lines. The multiple wrinkles and the yellowed, nodular, redundant, leathery, telangiectatic skin, with a variety of benign, premalignant, and malignant neoplasms that are characteristic of sunworshipers, constitute the gross cutaneous changes that denote photoaging. Because decades of exposure can be had before photoaging becomes apparent to the naked eye,[16] there has been a lack of urgency concerning prevention. The long latent period has contributed to the belief that photoaged skin is simply an acceleration of the inevitable age-dependent alterations. However, photoaging has unique and distinctive features. The visible manifestations reflect profound structural changes in the dermis. These will be summarized below.

PHOTOAGING VERSUS AGING: HISTOLOGIC COMPARISONS

The most striking histologic feature of seriously photodamaged skin is the presence of massive quantities of thickened, tangled accretions of degraded elastic fibers,[16] which finally degenerate into an amorphous mass (Fig. 1). This is not seen in normal, protected skin of even very old persons. With chronologic aging, elastic fibers may increase slightly in quantity and thickness (Fig. 2) while the vertical candelabra-like pattern of fibers seen

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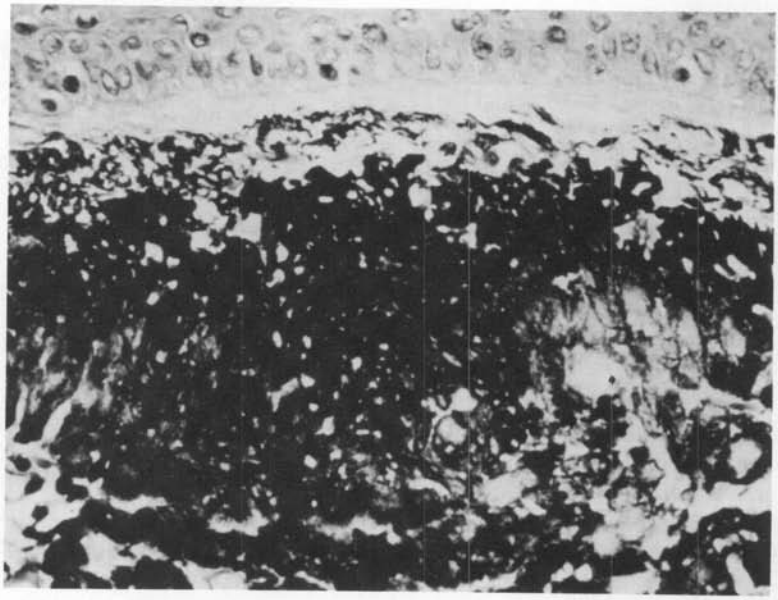


Figure 1. Human elastosis. Severe photodamage resulting in dense amorphous elastic deposits that have lost much of their fibrillary structure. (Luna's stain, × 370.)

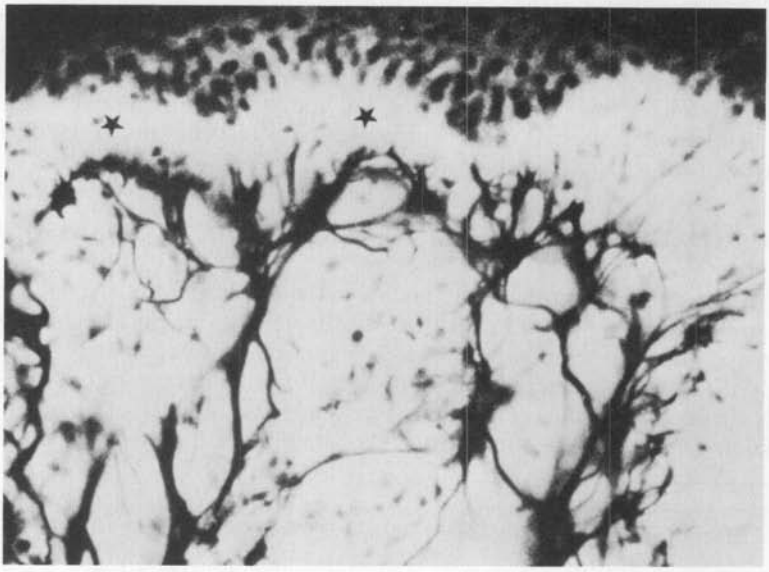


Figure 2. Aged, protected skin. Loss of anchoring fibrils (*stars*). Elastic fibers are thickened, but normal architecture is retained (× 370).

in the uppermost dermis of young skin are effaced. Mild ultrastructural deterioration has also been observed.[5, 26]

In contrast to the hypertrophy of elastic tissue in photoaged skin, the amount of mature collagen decreases.[37] More recent studies with ultraviolet-irradiated human and animal skin suggests an increase in type III collagen with a concomitant decrease in the normally dominant type I collagen.[30, 33] Other experimental studies find that early in the photoaging process there is no increase in the percentage of type III collagen,[39] with both types I and III increasing proportionally. Finally, after months of irradiation, there is a loss of total collagen. Evidence from animal studies suggests that collagen may be enzymatically hydrolyzed by cells of the inflammatory infiltrate provoked by ultraviolet radiation.[24] In normal aging, mature collagen apparently becomes more stable and resistant to enzymatic degradation.[2] The bundles become larger, forming rope-like structures.[36, 40]

The third component of the dermal connective tissue matrix, the ground substance, is composed of proteoglycans (dermatan sulfate, heparan sulfate) and glycosaminoglycans (hyaluronic acid). These are greatly increased in photodamaged skin. Although it is abundant in fetal skin, ground substance decreases rapidly and remains low in adult life. In protected aged skin, this component, if changed at all, is somewhat further decreased.[37]

Vast differences are seen also in the dermal cell population. In photodamaged skin, fibroblasts are numerous and hyperplastic. Inflammatory infiltrates abound. Mast cells are abundant and partially degranulated. In short, photoaged skin is chronically inflamed, a process called *heliodermatitis* (Fig. 3).[28] On the contrary, in chronologic aging, hypocellularity is the rule.[1]

The microcirculation suffers exceedingly from sun exposure.[18] Vessels become dilated and tortuous, producing visible telangiectasias. Finally, many in the horizontal plexuses become completely obliterated. In protected aged skin there is also deletion of small vessels, especially subepidermally, where the capillary loops are affected. However, vessels are not dilated and deranged and the overall horizontal plexuses remain largely undisturbed.[9]

It is generally held that atrophy is a hallmark of photoaged skin. This misconception probably arose from the examination of end-stage photodamage, at which point the epidermis is indeed severely thinned. It should be apparent that photoaging is characterized by "more," whereas with normal aging "less" is usually the rule. Typical of this is the response of the epidermis to chronic stimulation by ultraviolet radiation. It becomes thickened and is accompanied by cellular atypia, loss of polarity, and marked irregularities in cell sizes and staining properties (Fig. 3). Sebaceous glands in the sun-exposed areas of the face also become greatly enlarged.[36] In addition, various benign and malignant changes such as seborrheic and actinic keratoses, solar lentigos, keratoacanthomas, basal-cell epitheliomas, and squamous-cell carcinomas abound. These develop almost exclusively in sun-exposed areas.[44] In contrast, moderate epidermal thinning, with a flattening of the dermal-epidermal junction, is characteristic of protected aged skin (Fig. 4).[34] The predominant age-associated lesion is the senile

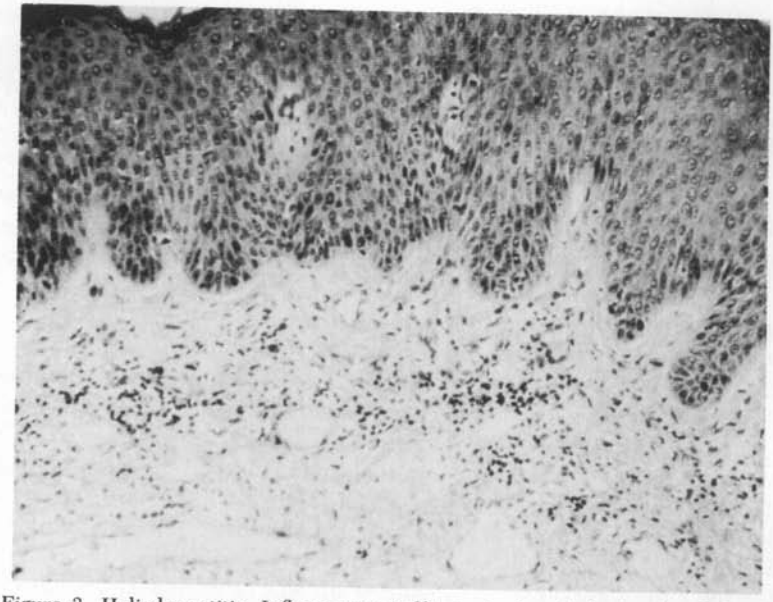


Figure 3. Heliodermatitis. Inflammatory infiltrate in chronically irradiated skin. Note hyperplastic epidermis with cellular disorder and irregularities in cell size and staining. (Hematoxylin and eosin, × 150.)

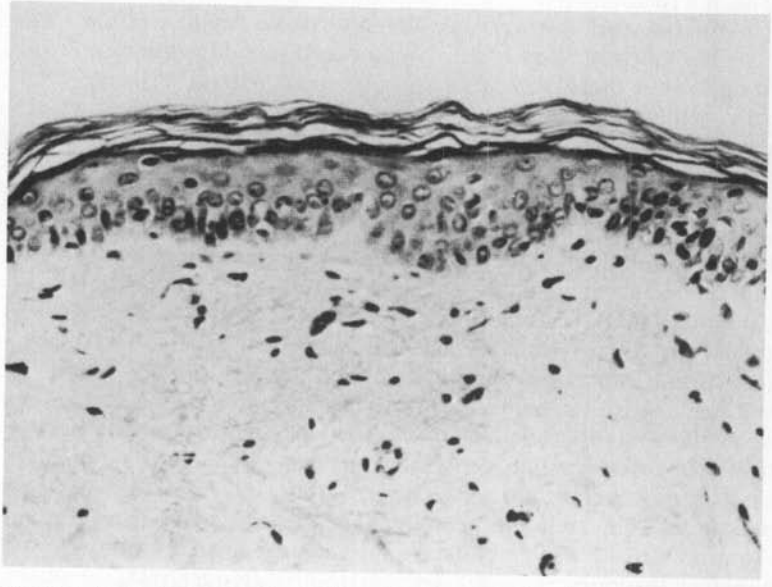


Figure 4. Aged, protected skin. A thinned epidermis with a flattened dermal-epidermal junction. (Hematoxylin and eosin, × 390.)

(cherry) angioma, occurring on the trunk area. Rarely do other tumors occur in protected skin.

Pigmented skin is only partially resistant to photoaging. Even the most darkly pigmented individuals have only about a decade of grace with regard to elastosis.[17]

THE ACTION SPECTRUM OF PHOTOAGING: EXPERIMENTAL STUDIES

Because the evolution of photoaging is so protracted, it cannot be examined easily in humans. Systematic studies require animal models for practical as well as ethical reasons. Although early seminal work was done with haired mice, especially with regard to photocarcinogenesis,[4] the current animal of choice for photobiologists is the hairless mouse. Convenient and hardy, they are available in two strains, albino and lightly pigmented. Most importantly, the ultraviolet-induced changes, ranging from acute responses[8] to those associated with chronic exposure such as tumorigenesis[20] and connective tissue damage,[22] are comparable to those in human skin. Hence, these animals have high relevance to photoaging.

UVB AND PHOTOAGING

Because it is well established that UVB radiation (280 to 315 nm) is responsible for erythema,[12] DNA damage,[7] and skin cancer,[4] it is assumed that UVB is also the waveband implicated in damaging connective tissue, thereby producing photoaging. Some evidence for this was provided by Sams et al.[37] These authors were able to demonstrate hyperplasia of elastic tissue to a modest degree in haired mice after a severely damaging dose of 30 to 50 MED (minimum erythema dose) delivered with a Hanovia hot quartz lamp. This ultraviolet source emits both UVB and UVC. With the hairless mouse it has been possible to produce extensive connective tissue photodamage with far lower doses. A bank of FS Westinghouse "sunlamps" is a convenient high-energy source of predominantly UVB radiation (290 to 315 nm). The quantity of UVA emitted is inconsequential with regard to connective tissue damage. The small UVC component can be filtered out effectively with a cellulose triacetate film. Normally, the hairless mouse has very little elastic tissue. Nevertheless, it was possible to produce severe elastosis with a total of approximately 5 Joules per cm^2 delivered as 2 MED per exposure thrice weekly for approximately 30 weeks (Fig. 5).[24] In irradiated animals, fibroblasts became more numerous. Ultrastructurally, they appeared metabolically active, producing increased quantities of collagen that resulted in a thickening of the dermis. Reticulin fibers, normally limited to the basement membrane zones, were prominent throughout the upper dermis, an indication of new collagen synthesis.[22] A decreased affinity for van Gieson's stain suggested severe damage to mature collagen. The histologic findings were confirmed by electron microscopy. In addition to a fraying and partial dissolution of large collagen fibers, there were many fibers of small diameters that are likely to be newly synthesized collagen.

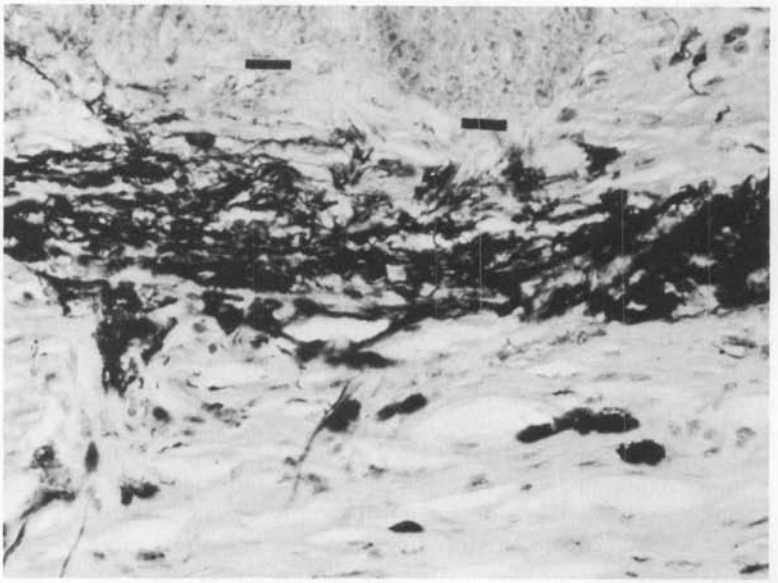


Figure 5. UVB-induced elastosis: hairless mouse (32 weeks). Dense accretions of elastic fibers below the dermal-epidermal junction (*bars*). (Luna's stain, × 370.) (*From* Kligman LH, Akin FJ, Kligman AM: The contributions of UVA and UVB to connective tissue damage in hairless mice. J Invest Dermatol 84:272–276, 1985, with the permission of The Williams & Wilkins Co., Baltimore, Maryland.)

Finally, the glycosaminoglycans (GAGs) of the ground substance were greatly increased compared with those in unirradiated tissue.

As in human photoaging, the general response to chronic ultraviolet radiation is one of hypertrophy. This was especially notable in the keratinizing cysts, typical of this animal, which lie in the lower half of the dermis. Normally present in one or two rows, after chronic UVB irradiation, the cysts increased and occupied four or five rows (Fig. 6).

UVA AND PHOTOAGING

Recently, it has been shown that acute exposure to UVA (315 to 400 nm) can, like UVB, produce erythema[15] and damage to blood vessels.[10] Because such effects require doses that may be 1000 times greater than UVB, the role of UVA in photoaging was thought to be negligible. However, UVA is present in sunlight in amounts that can be 500 to 1000 times that of UVB. Furthermore, its longer wavelength allows more of it to reach the dermis than does UVB. Because of these considerations, we were interested in examining the effects of UVA alone, in comparison with UVB.[24] A 5000W compact arc xenon solar simulator equipped with a Schott WG345 filter provided UVA with a spectral power distribution similar to solar UVA (315 to 400 nm). Animals irradiated with this source for 34 weeks, with a total

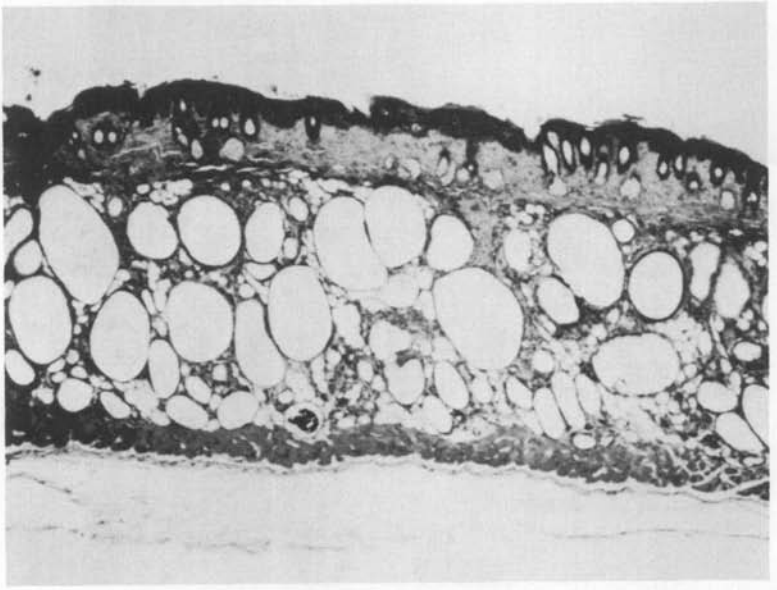


Figure 6. UVB-induced cyst proliferation: hairless mouse (32 weeks). Rows of dermal cysts increased from the normal 1 to 2 of unirradiated mice to 5 to 6. (Hematoxylin and eosin, × 37.) (*From* Kligman LH, Akin, FJ, Kligman AM: The contributions of UVA and UVB to connective tissue damage in hairless mice. J Invest Dermatol 84:272–276, 1985, with the permission of The Williams & Wilkins Co., Baltimore, Maryland.)

UVA dose of 3000 Joules per cm^2, developed a significant degree of elastosis (Fig. 7). Although the deposition of elastic fibers was less dense than that produced in animals irradiated with a total of 5 Joules per cm^2 UVB, it extended more deeply into the dermis. UVA from a black-light source (340 to 400 nm), with peak emission at approximately 365 nm, produced only mild elastic fiber hyperplasia despite a total UVA dose of 13,000 Joules per cm^2. This strongly suggests that the UVA wavelengths of 315 to 340 nm, abundant in solar UVA but lacking in the black light spectrum, are those most responsible for UVA-induced photodamage. However, even long wavelength UVA (340 to 400 nm) with a spectral distribution similar to that of sunlight (that is, high levels of radiation throughout the waveband as opposed to the parabolic distribution of black light emission) is capable of producing severe elastic fiber hyperplasia with cumulative doses totalling less than 8000 J per cm^2.[26]

In contrast to UVB, UVA had little or no effect on collagen. There is no substantive proof that ultraviolet radiation can damage collagen directly. However, it is well known that collagen can be degraded by proteolytic enzymes secreted by macrophages,[42] neutrophils,[33] and tumor cells.[3] Both macrophages and neutrophils are abundant in the severe inflammatory infiltrate evoked by UVB. Inflammation was strikingly absent in UVA hairless mouse skin. There were also vast differences in the tumor burden produced by the two wavebands, with multiple squamous-cell carcinomas

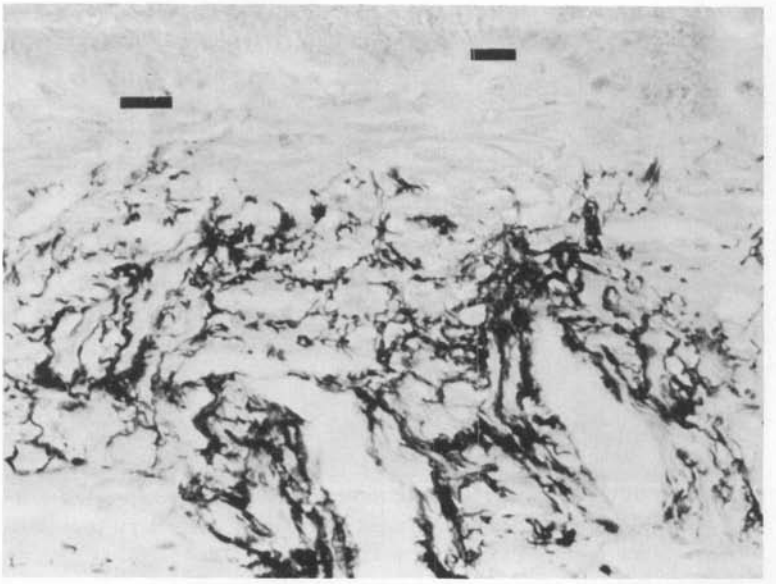


Figure 7. UVA-induced elastosis: hairless mouse (34 weeks). Elastotic fibers extend deeply into the dermis. Dermal-epidermal junction (*bars*). (Luna's stain, × 370.) (*From* Kligman LH, Akin FJ, Kligman AM: The contributions of UVA and UVB to connective tissue damage in hairless mice. J Invest Dermatol 84:272–276, 1985, with the permission of The Williams & Wilkins Co., Baltimore, Maryland.)

in UVB-irradiated animals compared with very few papillomas in the UVA group.

Like UVB, UVA radiation produced an increase in GAGs as visualized with Mowry's stain. The distribution, however, was different. Whereas UVB produced granular blue staining material localized in the upper dermis, UVA-induced GAGs were deposited throughout, imparting a bluish hue to the entire dermis. Additionally, densely staining material was deposited at the dermal-epidermal junction. One of the most striking results was proliferation of the keratinizing dermal cysts in response to solar-simulating UVA. Attaining six to seven rows (Fig. 8), this exceeded the proliferation produced by UVB.

These findings provide compelling evidence that UVA radiation, especially solar-simulating UVA, is capable of inducing profound photodamage to dermal connective tissue. Furthermore, the dose used in these experiments was realistic in terms of human exposure. For example, a 1-hour exposure at mid-day in June at 40 degrees north latitude will provide approximately 10 Joules per cm^2 UVA. Within 4 years, summertime exposure alone could result in the accumulation of 3000 Joules per cm^2.

Solar-Simulating Radiation and Photoaging

Although the effects of UVA alone are interesting from a scientific point of view, it is the cumulative effect of the entire ultraviolet spectrum

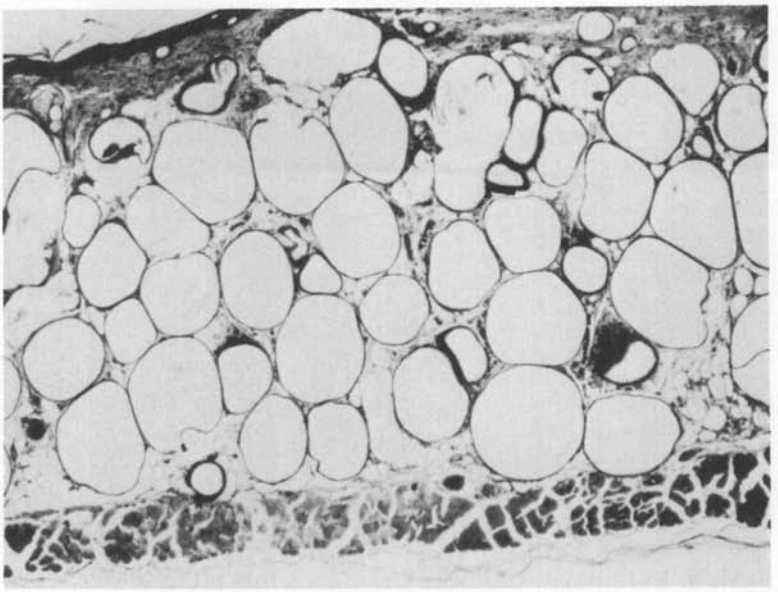


Figure 8. UVA-induced cyst proliferation: hairless mouse (34 weeks). Skin thickness is vastly increased, and cysts occupy 6 to 7 rows. (Hematoxylin and eosin, × 37.) (*From* Kligman LH, Akin FJ, Kligman AM: The contributions of UVA and UVB to connective tissue damage in hairless mice. J Invest Dermatol 84:272–276, 1985, with the permission of The Williams & Wilkins Co., Baltimore, Maryland.)

that is of prime concern. In the same study described above, a group of animals was exposed to solar-simulating radiation emitted by the xenon source fitted with a Schott WG320 filter.[24] The total UVB dose, delivered during the 34-week experiment, was approximately 5 Joules per cm^2 with a 100-fold more solar-simulating UVA. In some aspects, the photodamage reflected the influence of both wavebands, whereas in others, it was like UVB alone. Elastosis was severe but not confined to the upper dermis as with UVB. More dense than UVA elastosis, it extended deeply into the dermis. Collagen was damaged to the extent comparable to UVB alone. GAGs were equal to or slightly increased compared with UVB-irradiated tissue, with darkly stained material at the dermal-epidermal junction, providing evidence for the additional effect of UVA. Cyst proliferation, too, was greater than with UVB alone.

INFRARED RADIATION AND PHOTOAGING

It is necessary to note that terrestrial sunlight is polychromatic, extending beyond the ultraviolet region into the visible (400 to 700 nm), infrared (700 to 1×10^6 nm), and ultimately radiowaves. The latter can probably be ignored with regard to skin. Visible light, however, is known to produce deleterious effects in skin that range from phototoxic reactions in humans[14] to tumor enhancement in experimental animals.[11] Its role in photoaging has not been investigated. On the other hand, the harmful

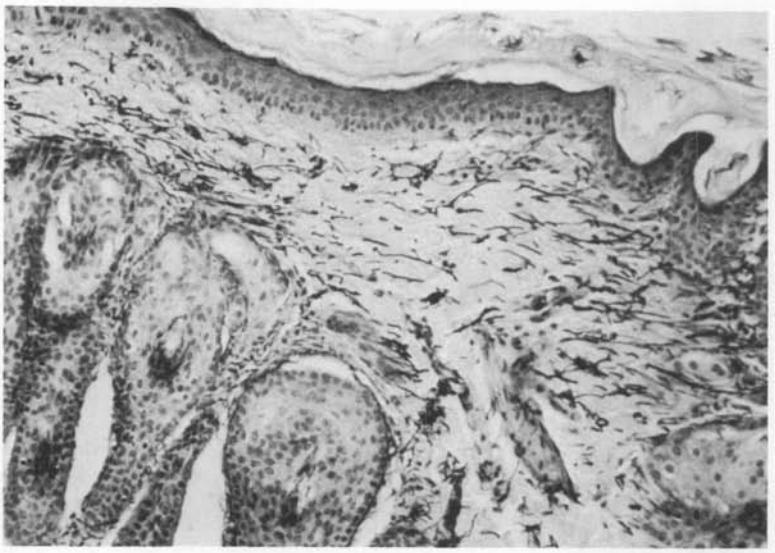


Figure 9. UVB-induced elastic fiber hyperplasia: guinea pig (20 weeks). A moderate thickening and increase in fiber content compared with unirradiated controls. (Luna's stain, × 110.)

effect of heat is ancient knowledge.[21] Human skin chronically exposed to infrared radiation often develops the condition erythema ab igne, with its characteristic mottled pigmentation resulting from damaged, leaking blood vessels. Severe elastic fiber hyperplasia, extending deeply into the dermis, is often present, along with thermal keratoses that are almost identical to those produced by ultraviolet radiation.

Despite vast clinical experience with the effects of infrared, little experimental work has been done. We were interested in determining if heat, in the physiologic range, could contribute significantly to ultraviolet-induced photoaging. Because it was expected that long periods of irradiation might be necessary, the guinea pig, a longer-lived animal than the mouse, was chosen.[19] Groups of albino animals, epilated weekly, were irradiated either with UVB or infrared radiation, or the two wavebands combined for a total of 45 weeks. Animals irradiated with UVB alone showed a modest increase in the quantity and thickness of elastic fibers (Fig. 9). Infrared alone produced moderately dense skeins of fine, delicate fibers (Fig. 10). The combined irradiations produced substantially increased amounts of thickened fibers, interspersed with many fine, parallel fibers, resulting in dense mats of elastosis (Fig. 11).

In contrast to the hairless mouse, guinea-pig collagen proved very resistant to damage, even after prolonged, combined irradiations. It should be noted that this animal did not mount a significant inflammatory response to these wavebands, nor did it develop tumors during the course of the study. Increased GAGs were produced by both ultraviolet and infrared radiation. Taken together, the epidemiologic, clinical, and experimental data strongly implicate infrared radiation as a contributing factor in pho-

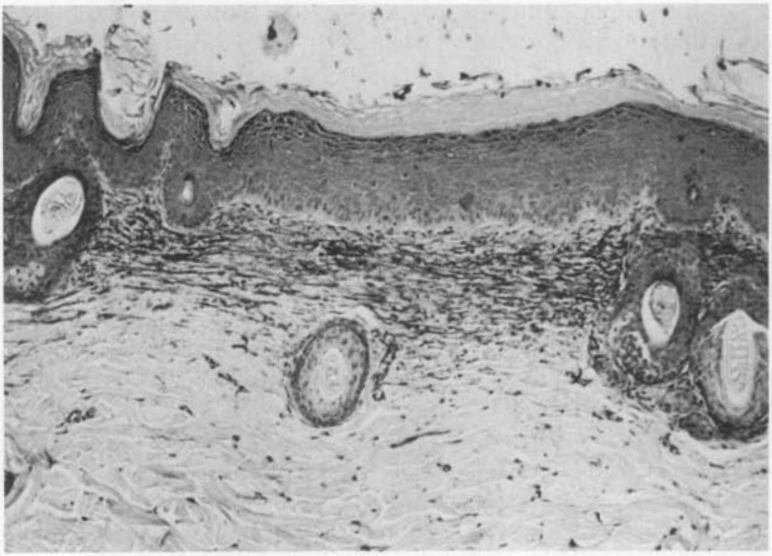


Figure 10. Infrared-induced elastosis: guinea pig (20 weeks). Deposits of fine parallel plastic fibers. (Luna's stain, × 70.)

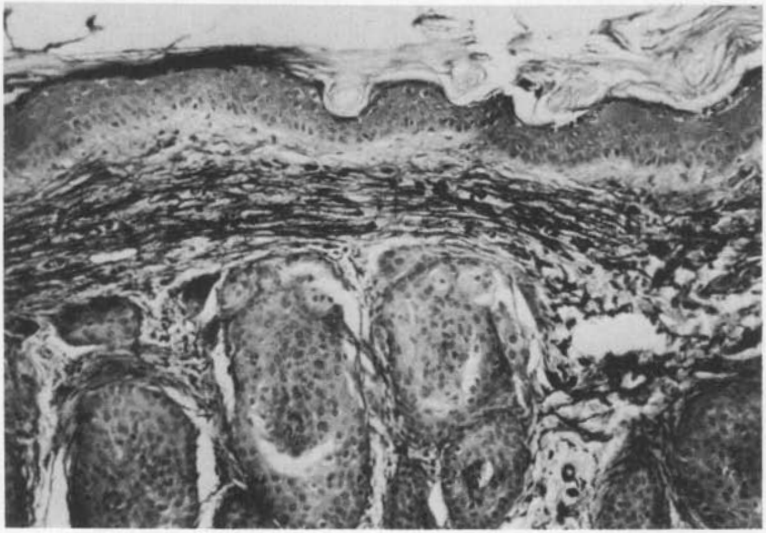


Figure 11. Elastosis produced by ultraviolet and infrared: guinea pig (20 weeks). A summation effect; dense mats of elastosis composed of thickened elastic fibers and fine parallel fibers. (Luna's stain, × 110.)

toaging.[21] Like UVA, infrared is inseparable from sunlight, so the cumulative effects of all the exposures are of concern.

PREVENTION OF PHOTOAGING: SUNSCREENS

In the hairless mouse model, broad-spectrum sunscreens, with a sun protection factor (SPF) of 15, provided effective protection against ultraviolet-induced connective tissue damage. This was especially true for animals irradiated with UVB at approximately 6 MED per exposure.[22] Histologic specimens were virtually indistinguishable from normal age-matched controls. Animals irradiated with solar-simulating radiation (UVB approximately 2 MED + UVA) and protected by the same sunscreens showed some mild dermal damage.[24] Elastosis was prevented, but elastic fibers were mildly hyperplastic. Collagen appeared undamaged, and although dermal cellularity was increased, massive inflammation did not occur. GAGs were slightly but not remarkably increased compared with unirradiated controls.

In both experiments, the sunscreen applications began at the time of the first exposure. This does not conform to human practice, in which sunscreens are generally not used until some degree of photodamage (such as the first wrinkle or actinic keratosis) is visible clinically. By that time, the connective tissue has already sustained severe photodamage. Previously, it was believed that this damage was irreversible. In our early studies with hairless mice, it was discovered that considerable repair could occur within a few months, even after extensive photodamage, if the UV exposures were stopped.[22] Broad regimens of new collagen were deposited in the subepidermal dermis, pushing downward the old elastotic material (Fig. 12). The new collagen was normal, histochemically and ultrastructurally; new elastic



Figure 12. Repair zone: hairless mouse (15 weeks after irradiation). A region of new subepidermal collagen deposition. Compressed elastosis, formerly in the uppermost dermis, delineates the lower boundary (*arrow*). Mast cells (*stars*) and fine new elastic fibers are present in the new dermis. (Luna's stain, × 280.) (*From* Kligman LH, Akin FJ, Kligman AM: Prevention of ultraviolet damage to the dermis of hairless mice by sunscreens. J Invest Dermatol 78:181–189, 1982, with the permission of The Williams & Wilkins Co., Baltimore, Maryland.)

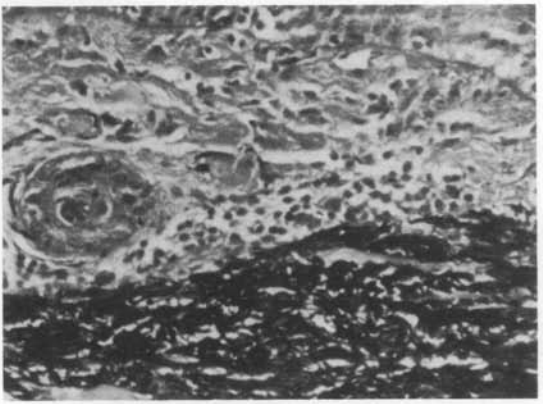


Figure 13. Human repair. Region of new collagen pushing down the old elastotic material. Biopsy from bald scalp of retired outdoor worker. (Luna's stain, × 280.)

fibers in this region were sparse and delicate as in unirradiated tissue. Interestingly, evidence for similar repair is seen in human skin when former sunworshipers eschew further solar exposure (Fig. 13).

Because of these findings, it was of interest to determine if the skin could repair itself in a like manner under conditions that simulated human experience.[23] Groups of animals were irradiated for a total of 30 weeks with UVB (approximately 3 MED per exposure). Sunscreens of SPF 6 and 15 were applied to separate groups after 10 or 20 weeks of irradiation. Control animals irradiated without sunscreen protection for the full 30 weeks sustained severe connective tissue damage that included extensive elastosis, loss of mature collagen, and greatly increased GAGs. Protected animals had only the degree of damage that reflected the weeks of irradiation before sunscreens were applied. As expected, animals irradiated for 20 weeks before sunscreens were applied had considerable photodamage. Nevertheless, during the final 10 weeks of irradiation, with sunscreen protection, a repair zone of new subepidermal collagen was deposited.

These experiments demonstrated that further damage could be prevented and repair could occur, even in the face of continuing irradiation, once sunscreens were applied. In general, earlier application of higher SPF provided the greatest degrees of protection against photoaging. It should be noted that at present, broad-spectrum sunscreens absorb effectively up to approximately 330 nm, providing little protection against a substantial portion of potentially dangerous UVA and none against infrared radiation.

THERAPY FOR PHOTOAGING

When it became apparent that repair processes could operate in skin if it was protected from further photoinsult, we wondered if it was possible to augment the repair. A number of considerations led us to try retinoic acid in this context. For example, retinoids have been reported to stimulate wound repair, possibly by enhancing collagen deposition in granulation tissue.[30] They also appear capable of reversing the inhibitory effect of

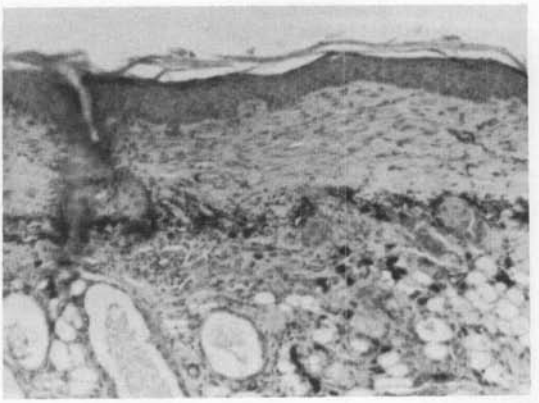


Figure 14. Retinoic acid enhanced repair: hairless mouse. Application of 0.05 per cent retinoic acid for 10 weeks in the post-irradiation period produced a region of new collagen deposition that was significantly wider than that of controls. Note extreme cellularity of dermis. (Luna's stain, × 115.) (*From* Kligman LH, Chen HD, Kligman AM: Topical retinoic acid enhances the repair of ultraviolet damaged dermal connective tissue. Connect Tissue Res 12:139–150, 1984, with permission.)

corticosteroids[13] and salicylates[29] on wound healing. In addition, retinoids have been shown to suppress collagenase activity in synovial tissue.[6]

For a model in which to test our hypothesis, we preirradiated hairless mice with UVB for 10 weeks to produce mild connective tissue damage. We found that topical all-trans retinoic acid greatly accelerated the formation of a zone of new connective tissue during the post-irradiation period. Repair zones were significantly wider than those in animals treated with vehicle or nothing (Figs. 14 and 15). In addition to being time-dependent, with wider repair zones after 10 weeks compared to 5 weeks (Table 1), stimulation of repair was retinoic acid dose-dependent within the range

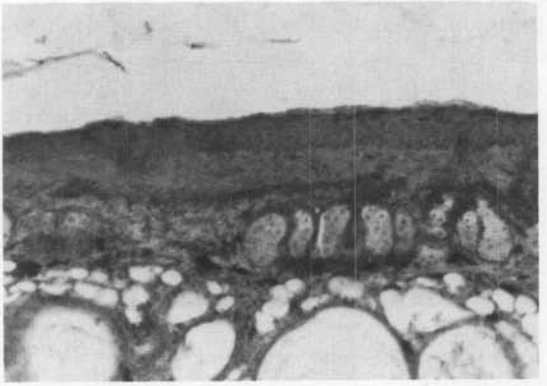


Figure 15. Vehicle control: hairless mouse. Application of the cream vehicle for 10 weeks in the post-irradiation period. The narrow repair zone was similar to that of untreated controls post-irradiation. (Luna's stain, × 115.) (*From* Kligman LH, Chen HD, Kligman AM: Topical retinoic acid enhances the repair of ultraviolet damaged dermal connective tissue. Connect Tissue Res 12:139–150, 1964, with permission.)

Table 1. *Reconstruction Zone Measurements as a Function of Time*

TREATMENT	MEAN THICKNESS (μ)	S.D.
Retinoic acid (5 weeks)	88.7*	17.3
Vehicle (5 weeks)	48.3	11.9
Retinoic acid (10 weeks)	117.0*	27.1
Vehicle (10 weeks)	67.2	17.3

*Significantly greater than controls ($p < .01$). Statistical analysis with Duncan's Multiple Range Test.

From Kligman LH, Chen HD, Kligman AM: Topical retinoic acid enhances the repair of ultraviolet damaged dermal connective tissue. Connect Tissue Res 12:139–150, 1984; with permission.

tested (0.005 to 0.05 per cent).[25] The new collagen in the repair zone resembled that of normal, unirradiated tissue. Ultrastructurally, bundles were parallel to the surface in a normal "plywood-like" arrangement. Fibroblasts were very numerous and had morphologic features of high metabolic activity. The greatly expanded cytoplasm, far in excess of what is seen in ultraviolet-irradiated fibroblasts, was filled with an abundance of widely dilated endoplasmic reticulum.

Because retinoic acid can be irritating to hairless mouse skin, we wondered if other irritants could produce the same effect. A number of agents (salicylic acid, croton oil, propylene glycol, hyamine, and sodium lauryl sulfate) were tested at concentrations that were clinically and microscopically irritating. In no case was repair greater than would be expected from just stopping the irradiation. Thus, the enhanced repair of photodamage appears to be retinoid-specific.

SUMMARY

In recent years there has been an increasing awareness that many of the so-called attributes of aging skin are, instead, a reflection of environmental assault upon exposed areas of the body. Of special import are the deleterious effects of solar radiation on dermal connective tissue, leading to the visible manifestations of photoaging. Often termed "premature aging," the salient features of the process are distinctly different from those found in normal intrinsic aging. In general, chronically irradiated skin is metabolically hyperactive with epidermal hyperplasia and neoplasia, increased production of elastic fibers, GAGs, accelerated breakdown and synthesis of collagen, and enhanced inflammatory processes. In contrast, protected aged skin is usually characterized by a slow decline in many of these components.

Experimental studies with animal models have confirmed the notion that the shorter, more energetic portion of the ultraviolet spectrum (UVB) is responsible for the dermal connective tissue destruction observed in photoaged skin. More recently, it has been shown that UVA and infrared radiation contribute significantly to photoaging, producing, among other changes, severe elastosis. Because the three broad wavebands are inseparably linked in terrestrial sunlight, all are of concern in the photoaging of human skin.

Photoaged skin has been thought to be irreversibly damaged. However, our findings indicate that destruction and repair go on simultaneously under continued assault by actinic radiation. The balance is shifted toward repair when the radiation stress is relieved. Both epidermis and dermis are capable of moderate self-restoration when exogenous injury ceases, either by avoidance of sunlight or by the use of broad-spectrum, high-SPF sunscreens. Repair of the dermis, characterized by broad regions of new collagen deposited subepidermally, can be pharmacologically enhanced by topical application of retinoic acid.

Although early protection from sunlight, before severe photodamage occurs, is most desirable, it is deemed advisable to counsel even older persons with photoaged skin to adopt protective measures, thereby allowing repair processes to occur.

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