A New Apparatus with High Radiation Energy Between 320–460 nm: Physical Description and Dermatological Applications

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A new apparatus (UVASUN 5000) is presented with high radiation energy between 320–460 nm. The measurable energy below 320 nm was shown to be many orders of magnitude too low to produce erythema. The radiator is a specially developed source for high UV-A intensity, housing a quartz bulb with a mixture of argon, mercury and metal-halides. At a skin-target distance of 0.2 m the size of the irradiated area is 0.35 × 0.35 m, and the measured mean UV-A intensity is about 1400 $W \cdot m^{-2}$ (140 $mW \cdot cm^{-2}$). The UV-A energy in the range of 320–400 nm is about 84% of the total radiation energy.

Effects of very high doses of UV-A on human skin were studied. Following single UV-A applications the minimal tanning dose UV-A (MTD) and the immediate pigment darkening (IPD) dose of UV-A were established. The calculated IPD threshold time was 1.8 min at 0.2 m. Repeated exposure to this UV-A delivering system yields long lasting dark brown skin pigmentation without any clinical or histological signs of "sunburn" (UV-B) damage, epidermal hyperplasia or thickening of the stratum corneum.

The instrument was also successfully used for photo-patch testing and reproduction of skin lesions of polymorphous light eruption. Minimal therapeutic results were seen in the phototherapy of vitiligo and inflammatory acne.

For the investigation of photobiological effects of high UV-A intensities on human skin, UV-A irradiating instruments are required which deliver particularly high UV-A intensity over a sufficiently large area within a suitable time. The conventional gas discharge sources and incandescent sources [1] have not so far been used in an apparatus to meet these requirements. No UV-A emitting source without the unwanted UV-B was available, and the use of additional cut-off filters lowered the UV-A intensity considerably.

Since pulsed nitrogen (N_2) lasers were introduced, high UV-A intensity, for instance 20–30 $J \cdot cm^{-2}$ within 200 sec [2] could be delivered. The disadvantage of this N_2 laser is the monochromatic wave length of 337.1 and the much too small skin area irradiated.

Abbreviations:
IPD: immediate pigment darkening UV-A
MED-UV-A: minimal erythema dose UV-A
MED-UV-B: minimal erythema dose UV-B
MTD: minimal tanning dose UV-A
TBA: tribromosalicylanilide
TCSA: tetrachlorosalicylanilide
UV-A: long wave length ultraviolet rays 315–400 nm
UV-B: medium wave length ultraviolet rays 280–315 nm
UVC: short wave length ultraviolet rays >280 nm

A newly developed apparatus*, emitting long wave ultraviolet radiation free of measurable erythemogenic radiation below 320 nm, was designed. The physical properties of this apparatus and some dermatological applications will be presented herein.

MATERIALS AND METHODS

UVASUN 5000

The goal of the UVASUN 5000 project is to produce the highest possible radiation energy limited to the UV-A range. Wave lengths shorter than 320 nm are UV-B-erythema effective [1,3–6]. For that reason the measurable energy below 320 nm should be too low to be erythemogenic for the irradiation times to be used.

Design

The design in principle is shown in Fig 1. The radiator is a specially developed source for high UV-A intensity. In the interior of the quartz bulb is a mixture of argon, mercury and metal-halides. The bulb, made of special quartz, has an operation temperature of approximately 900°C and works as a UV-C filter. Therefore no ozone is produced. Around the radiator is a blue-violet filter tube with a wall thickness of 1 mm, which absorbs the visible radiation above 460 nm and part of the UV-B. The temperature of this filter tube should not exceed 350°C. Therefore a cooling-system is necessary. A ventilator blows air into the space between radiator and filter tube and cools the filter tube. In order to keep the temperature of the radiator in the ideal range (900°C), the quartz bulb has to be protected against the cooling air jet. This protection is obtained by a quartz tube which is partly closed on the air-supply side. Because the filter tube transmits infrared, a special infrared absorbing filter is required. This filter has a thickness of 6 mm, is shatterproof, absorbs the infrared rays and further reduces the UV-B. The anodized aluminum reflector, paraboloid in two planes, has a high reflectance in the UV-A range.

The whole optical part of the apparatus is enclosed in a housing which is swivel-mounted on a horizontal and a vertical axis. With a swiveling range of more than 300° for each axis, practically each position for irradiation can be adjusted. The height can be adjusted by a crank, so that a person standing, sitting, or lying down can be irradiated comfortably.

Technical Data

Table I shows the relative spectral energy distribution of the total relative energy from 320–460 nm, UV-A energy in the range of 320–400 nm is about 84% of the total radiation energy, 16% is in the blue-violet section of the visible light. There are varying definitions of UV-A, however, the differences are minimal. Most authors define UV-A as wavebands between 320–400 nm [1,5].

The Deutsche Institut für Normung e.V. (DIN) defines UV-A as wavebands between 315–380 nm [7]. The "Commission Internationale De L'Eclairage" (CIE) defines UV-A from 315–400 nm. This last range is used in Fig 2 and 5. In Fig 2 the relative spectral irradiance is shown together with the curves of the relative spectral effectiveness (action spectrum) of the UV-B-erythema and the immediate pigment darkening (IPD) [3]. Fig 3 shows the dependency of the UV-A irradiance (E_{UV-A}) on the distance from the UV-A source. Distance zero is defined as the surface of the infrared filter.

In Fig 4 the length of side of the irradiation area is plotted against the distance. Standards in illuminating engineering and also in the draft of the DIN 5050 [8] define the uniformity of an irradiation area as the proportion between maximal irradiance (E_{max}) and minimal irradiance

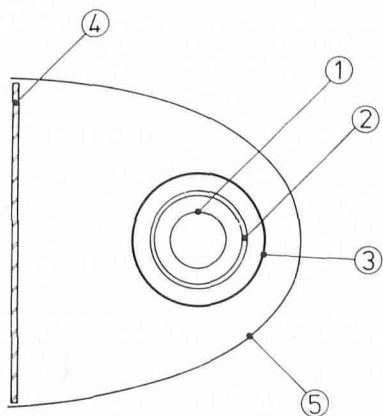* UVASUN 5000, manufactured by Mutzhas Co., Munich, Germany.

FIG 1. Cross-section of the optical part of the light source. *1* = radiator; *2* = protecting quartz tube; *3* = blue-violet filter tube; *4* = infrared absorbing filter; *5* = anodized aluminum reflector.

TABLE I. *Relative spectral energy distribution of UVASUN 5000*

λ nm	$E_{e, rel}$
310–320	0.00
320–330	0.05
330–340	0.39
340–350	2.58
350–360	7.50
360–370	20.51
370–380	20.91
380–390	20.31
390–400	11.64
400–410	7.26
410–420	3.42
420–430	2.73
430–440	1.83
440–450	0.78
450–460	0.09
Sum	100.00

(E_{min}) being smaller than the value of 2.

The uniformity was measured between the distance of 0.2–3.0 m. At a distance of 0.2 m the size of the area is 0.35×0.35 m, and the measured mean UV-A intensity is about 1400 $W \cdot m^{-2}$ (140 $mW \cdot cm^{-2}$). The "Commission Internationale De L'Eclairage" (CIE) defines the UV-A (315–400 nm) of the global radiation (altitude of the sun 90°, cloudless day and air mass = 1) as 63 $W \cdot m^{-2}$ (6.3 $mW \cdot cm^{-2}$) [9]. If we compare these values, the UVASUN 5000 produces 22 times the UV-A intensity of the sun.

Spectrophotometry and Exclusion of UV-B

To determine the spectral energy distribution a spectrophotometer† with 2 monochromators† was used. The detector was a photo-multiplier† which was cooled ($-35°C$) to get a lower noise level. The spectral radiant energy was measured automatically in steps of 0.1 nm and stored in a computer. The relative error was less than ±1%, the absolute error less than 5%.

In the UV-B and shortwave UV-A range the sensitivity of the spectrophotometric unit is 10^{-4} $W \cdot sr^{-1} \cdot nm^{-1}$. If the sum of the radiant energy in a bandwidth of 1 nm is less than $10^{-4} W \cdot sr^{-1}$ the computer reads zero, because the signal and the noise level cannot be separated. In our measurements the values were printed in 5 nm intervals so that in the most adverse case the following error could happen: From 315–320 nm all the measured radiant energies are so close to the indication limit that the value zero is printed. This means that the maximal indication error is $5 \cdot 10^{-4} W \cdot sr^{-1} \cdot nm^{-1}$. At the distance of 0.2 m from the UVASUN 5000 an irradiance of $18.75 \cdot 10^{-4} W \cdot m^{-2}$ is equivalent to the radiant energy of $5 \cdot 10^{-4} W \cdot sr^{-1} \cdot nm^{-1}$.

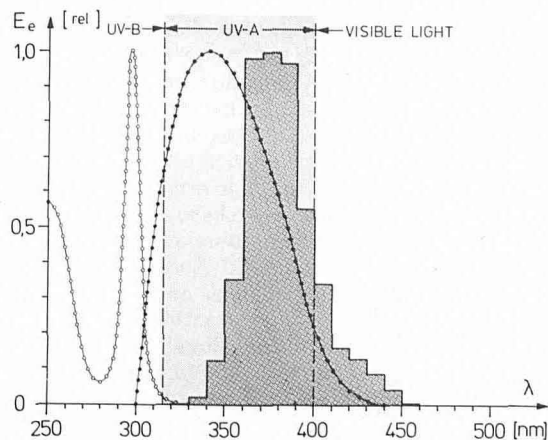† Laboratories of the Osram Company, Munich, not available commercially.

FIG 2. Relative spectral irradiance of UVASUN 5000 between 320–460 nm (out of which 84% are in the UV-A range). Curves of the relative spectral effectiveness (action spectrum) of UV-B-erythema (-O-O-) and the immediate pigment darkening (IPD) in the UV-A (-●-●-).
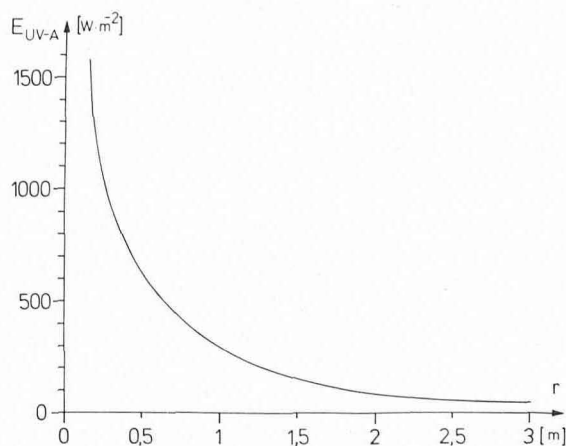

FIG 3. Dependency of UV-A irradiance (E_{UV-A}) on the skin-target distance from the light source.
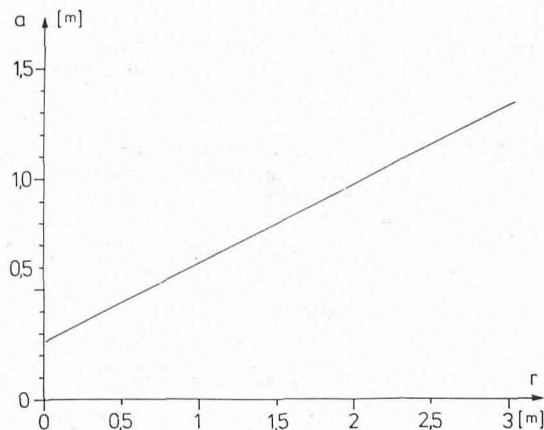
FIG 4. Side-length of irradiation area (a) versus skin-target distance (r).

With equation

$$E_{er} = \int E(\lambda)_e \cdot s(\lambda)_{er, rel} \cdot d\lambda$$

the erythema-effective irradiance (E_{er}) can be calculated [10]. $E(\lambda)_e$ is the spectral physical irradiance and $s(\lambda)_{er, rel}$ is the spectral sensitivity of the UV-B erythema.

The time to reach the UV-B-erythema threshold ($t_{s, er}$) is the quotient
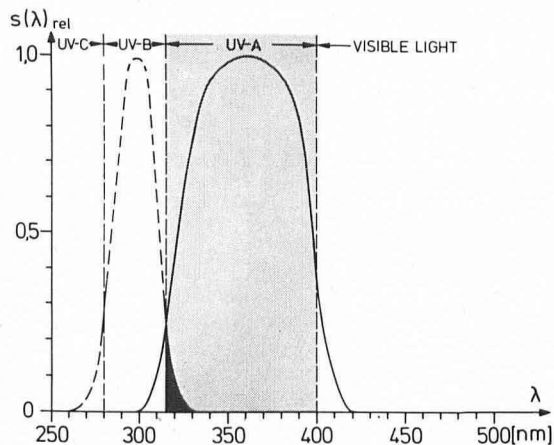
FIG 5. Indication-error of the integrating UV-instrument in the short wave UV-A range (315–330 nm). The end of the longwave UV-B curve (---) reaches into the UV-A range (*stippled bar*). A small portion of UV-A is measured with the UV-B detector and is indicated as UV-B (black area). $S(\lambda)_{rel}$ = relative spectral sensitivity of the integrating UV Centra detectors.

TABLE II. *Calculated UV-B-erythema effective irradiance (E_{er}) for λ 315–319 nm*

λ nm	$s(\lambda)_{er, rel}$	E_{er} $W \cdot m^{-2}$	$E_{er, rel}$ $W \cdot m^{-2}$
315	0.015	$3.75 \cdot 10^{-4}$	$5.625 \cdot 10^{-6}$
316	0.013	$3.75 \cdot 10^{-4}$	$4.875 \cdot 10^{-6}$
317	0.010	$3.75 \cdot 10^{-4}$	$3.75 \cdot 10^{-6}$
318	0.008	$3.75 \cdot 10^{-4}$	$3.00 \cdot 10^{-6}$
319	0.006	$3.75 \cdot 10^{-4}$	$2.25 \cdot 10^{-6}$
Sum		$18.75 \cdot 10^{-4}$	$19.5 \cdot 10^{-6}$

of the UV-B-erythema threshold dose ($H_{s, er}$) and the UV-B erythema effective irradiance (E_{er}). The mean UV-B-erythema threshold dose (for average untanned caucasians) can be represented with the value $250 \ W \cdot s \cdot m^{-2}$ (25 mJ $\cdot$ cm^{-2}) [1,4,10].

$$t_{s, er} = \frac{250}{19.5 \cdot 10^{-6}} = 12.82 \cdot 10^6 \qquad s = 3561 \ hr$$

The time of 3561 hr indicates that an uninterrupted irradiation of 5 mo would, theoretically, lead to the slight UV-B-erythema (MED-UV-B).

Dosimetry

It is not practical to use the spectrophotometer for daily measurements. For this purpose an integrating instrument (Centra UV-Meßgerät, Osram, Munich), with different detectors for UV-B and UV-A was used in our laboratory.

The instrument consists of a detector for UV-B, and one for UV-A, an electronic amplifier and a digital display. The detector contains silicon-planar-photocells. The edge steepness of the spectral sensitivity curve of the detector is satisfying.

When the UVASUN 5000 was measured with the UV-B detector, it still indicated low values, although no UV-B was present. The reason for this indication error is explained by the *dashed curve* in Fig 5. The long wave end of the UV-B curve reaches into the UV-A range; a small portion of UV-A is measured with the UV-B detector and indicated as UV-B.

In the UV-A range the comparison of the values measured with the spectrophotometer and the integrating UV-instrument showed an acceptable accordance. The difference was ±2%.

With this integrating UV-instrument doses can be measured as well. Any doses can be pre-set. A light indicates the end of the exposure time. The newest model of this UV-instrument (Mutzhas UV-A Meßgerät) has a measuring range for UV-A irradiance of 9999 W $\cdot$ m^{-2} (999 mW $\cdot$ cm^{-2}). The maximal pre-settable dose of $999 \cdot 10^4 W \cdot s \cdot m^{-2}$ (999 J $\cdot$ cm^{-2}).

Eye Protection

Some publications document UV-A causes cateracts in experimental animals [11]. It is conceivable that this may also occur in the human,

although no damage to the human eye due to the UV-A has been reported. Specially designed goggles were used in these experiments (UVASUN special goggles). They absorb radiation below 400 nm and have an average transmittance of about 30% between 400–780 nm.

DERMATOLOGICAL APPLICATIONS

Throughout our investigations the UV-A apparatus was set at a skin target distance between 0.4 and 0.7 m. At these distances, the areas, which were uniformly irradiated, measured about 0.5 × 0.5 m, and the UV-A irradiance on the skin surface varied from 40 to 70 mW $\cdot$ cm^{-2}. Under these conditions, for instance, 10 J $\cdot$ cm^{-2} were irradiated within 2.4 to 4.0 min.

I. Biological Effects of High Doses of UV-A on Normal Human Skin

Response to single exposures: Pigment darkening and delayed tanning. Effects of single exposures were investigated in 69 patients (skin type I–IV). They had no known altered photosensitivity or disorders of the pigmentary system. Six sites on the volar aspect of the forearm measuring 1.0 × 2.0 cm were irradiated once with increasing doses of UV-A between 5.0–30.0 J $\cdot$ cm^{-2}. The subjects were seen immediately and after 1, 7 and 14 days, when they were examined for immediate pigment darkening or delayed tanning respectively.

Response to multiple exposures: Tanning. To study tanning, 16 patients were repeatedly exposed to high doses of UV-A between 30.0–60.0 J $\cdot$ cm^{-2}, mostly 50.0 J $\cdot$ cm^{-2}; the mean cumulative dose was 855.3 J $\cdot$ cm^{-2} (SD = ±488.8), the mean number of treatments 21.3 (±11.2). Treatments were given on an average 3 times a week over a period up to 8 weeks. Forearm, chest, abdomen, back or face were irradiated, in fields 10.0 × 15.0 cm.

Histology: Biopsies were obtained from the lower back 72 hr after application of 60.0 J $\cdot$ cm^{-2} UV-A. Specimens were formalin fixed, cut into 6μ thin sections, stained with hematoxylin and eosin and examined particularly for UV-induced epidermal damage.

In 4 subjects initially 30.0 J $\cdot$ cm^{-2}, later on 50.0 J $\cdot$ cm^{-2} UV-A were administered 3 times a week to the back. Cumulative doses were 500, 620, 750 and 760 J $\cdot$ cm^{-2} UV-A respectively. Biopsies were obtained after 4–8 weeks of treatment. Specimens were handled as described above. In addition half of the tissue was snap-frozen in liquid nitrogen and cryostat sections were prepared. With DOPA stain melanocytes were counted over a given length of skin surface. The number of stratum corneum cell layers was investigated with the fluorescein isothiocyanate technique [12].

II. Diagnostic Techniques

Photopatch tests: Eighteen patients, males and females (age 19–67 yr), with suspected photoallergic dermatitis were investigated. Five compounds (0.1% tetrachlorosalicylanilide (TCSA), 1% tribromosalicylanilide (TBA), 1% bithionol, 1% hexachlorophene, 1% chlorpromazine, all in hydrophilic ointment or vaseline) were applied semi-occlusively to the upper back and secured with Leukosilk (Beiersdorf AG, Hamburg) using the standards of the International Contact Dermatitis Group. A duplicate set of patches was applied to the contralateral site of the back and served as unirradiated control. The patches were removed at 24 hr and exposed to 15.0 J $\cdot$ cm^{-2} UV-A. In special cases, where increased UV-A sensitivity was suspected, the threshold sensitivity for UV-A without the drug was determined first. Irradiated sites were light-protected for the next 24 hr. Readings were done immediately after irradiation, and 24 and 48 hr later, and also as late as 7 days.

Reproduction of skin lesions in polymorphous light eruption: As a pretest, to exclude increased photosensitivity, in 72 patients with a history of polymorphous light eruption, the volar aspect of the forearm was irradiated with UV-A doses of 5.0–30.0 J $\cdot$ cm^{-2} with increments of 5.0 J $\cdot$ cm^{-2}. Following the pretest, in 60 patients single large doses of 60.0 (skin type I and II) or 100.0 J $\cdot$ cm^{-2} UV-A (skin type III and IV) were administered to areas which were afflicted during past episodes of polymorphous light eruption. These sites were usually the sun-exposed side of the lower or upper arm, the back of the hands the neck and upper chest. In 12 patients, in whom no skin lesions of polymorphous light eruption could be produced with this technique, additional 60.0 or 100.0 J $\cdot$ cm^{-2} UV-A were administered for a second and if necessary for a third time on consecutive days. After each irradiation readings were done immediately and after 1 and 24 hr. In 8 patients biopsies were taken from experimentally induced skin lesions 24 hr after the last application of the UV-A.

III. Therapeutic Trials

Acne: Eight patients with inflammatory acne with lesions on the face and upper back or chest were treated 3 to 4 times weekly with single

doses between 30.0–50.0 J·cm^{-2} UV-A. The number of treatments varied from 4 to 33, cumulative doses from 120 to 1480 J·cm^{-2} (m = 821 ± 464).

Vitiligo: Eight patients with widespread vitiligo of many years' duration and no spontaneous repigmentation over the past 9 mo were treated 3 to 4 times weekly with single doses of 20.0–50.0 J·cm^{-2} UV-A. The number of treatments ranged from 4 to 35, cumulative doses from 110 to 1880 J·cm^{-2} (m = 890 ± 542).

RESULTS

I. Biological Effects of High Doses of UV-A on Normal Human Skin

Response to single exposures: Immediate pigment darkening and delayed tanning. Threshold IPD was seen in 62 out of 69 patients. Nineteen patients had threshold doses of below 5.0 J·cm^{-2}, 24 patients of 5.0 J·cm^{-2}, 15 patients of 10.0 J·cm^{-2} and 4 of more than 10.0 J·cm^{-2} UV-A. IPD was dose-dependent in all patients and reached in some subjects an intense brown with a greyish hue. Pigment darkening appeared immediately after the irradiation, e.g., when the threshold dose was 5.0 J·cm^{-2}, within 120 seconds. The maximum occurred immediately and the intensity decreased over the next few hours. IPD blended into delayed tanning within 24 hr. This was seen in 43 out of 69 patients. IPD correlated with the skin type. Patients with a darker complexion (skin type III–IV) reacted more intensely than types I and II. Seven patients did not pigment immediately in response to doses up to 30.0 J·cm^{-2} UV-A (Fig 6). These were all very fair skinned people.

At 24 hr tanning was present in 43 patients. Threshold doses (MTD-UV-A) are given in Fig 6. The pigment was now brownish and resembled the classical sun-induced pigmentation. This pigmentation was visible over the next days, sometimes up to several weeks. The intensity of the tan was greater in the IPD reaction than it was in the delayed tanning. There was no delayed tanning, without an immediate pigment darkening response. The doses required for delayed tanning were much higher than those for an IPD reaction. For instance immediate pigment darkening was achieved in most of the subjects with less than 15.0 J·cm^{-2} compared to the minimal tanning dose of 15.0–30.0 J·cm^{-2} or more in the majority of the subjects. Patients who needed 20.0–30.0 J·cm^{-2} UV-A for IPD were those who did not show delayed tanning.

Response to multiple exposures: Tanning. Long lasting pigmentation was always induced with repeated irradiations. It increased with the number of exposures and was dose-related. A chocolate brown skin color appeared in virtually all of the subjects (Fig 7). It is noteworthy that this pigmentation was not generally preceded by a sunburn-like erythema. Also no scaling was induced even in long-term studies.

Histology: The living epidermal cells looked undamaged in specimens taken 72 hr after the UV-A irradiation. No increased numbers of so-called sunburn cells (dyskeratotic or vacuolated cells) appeared in the Malpighian layer compared to controls (Fig 8). There was also no dilatation of capillaries or edema. The clinically visible delayed tanning had no detectable histological correlate.

After cumulative doses of up to 760 J·cm^{-2} UV-A the epithelial band looked normal without acanthosis and without signs of sunburn cells. The dermal papillae including the vascular architecture did not differ from controls. There was, however, marked increase of pigment in the basal cell layer. Dopa-stain revealed heavily pigmented melanocytes compared to untreated skin. However, the number of melanocytes per unit skin surface length was not increased. In repeatedly irradiated skin 32.4 ± 7.1 melanocytes per mm skin surface length were counted. This did not significantly differ from untreated controls (33.8 ± 3.9). The number of cell layers in the stratum corneum was unchanged as revealed by the fluorescein isothiocyanate technique.

II. Diagnostic Techniques

Photopatch tests: Phototoxic reactions to chlorpromazine occurred in over 80%. This was judged from an immediate stinging sensation (smarting), erythema confined to the site of the patch and histologic examination. The smarting reaction may be similar to the one seen in irradiated tar patch-tests. Positive photopatch tests to chlorpromazine, indicating photoallergy, were seen in 3 patients. In addition, 3 patients reacted to TCSA, 1 patient to TBA and 1 to hexachlorophene.

Reproduction of Skin Lesions in Polymorphous Light Eruption: Single exposures with UV-A doses between 5.0–30.0 J·cm^{-2} never resulted in abnormal responses; merely immediate pigment darkening blending into delayed tanning was noticed. However, single high doses of 60.0 or 100.0 J·cm^{-2} elicited in 19 out of 60 patients erythema and papules, indistinguishable from naturally occurring lesions of polymorphous light eruption. With repeated exposures of UV-A doses of 60.0 or 100.0 J·cm^{-2} we were able to provoke the typical lesions of polymorphous light eruption in 8 out of 12 patients. The onset of erythema and lesions was preceded by itching. Biopsies revealed the essential features of polymorphous light eruption (Fig 9) with subepidermal edema and perivascular round cell infiltrate. In contrast, epidermal changes were absent [13].

III. Therapeutic Trials

Acne: Six patients remained unchanged, one got better and one worsened. Tanned skin camouflaged the inflammatory lesions. Comedones were not affected.

Vitiligo: A good result was seen in only 1 patient, who had received 35 treatments and a cumulative UV-A dose of 1420 J·cm^{-2}. Spotty follicular-bound repigmentation occurred. Vitiliginous skin reacted with a persistent erythema and never showed signs of scaling (Fig 10). The repigmenting capacity of the high UV-A doses is not better than that from conventional trimethylpsoralen-(TMP)-UV-A-photochemotherapy.

DISCUSSION

Phototherapy and photochemotherapy has gained much interest in the past 5 yr, especially with the introduction of high intensity UV-A units for PUVA-treatment. Due to physical problems of the irradiation equipment, effects of UV-A are very difficult to study. Lasers with emission in the UV-A range cannot overcome this dilemma. Many questions concerning the effects of single high doses of UV-A (60–100 J·cm^{-2}) or repeated high doses of UV-A were left unanswered.

The new apparatus, with high irradiance between 320–460 nm, presented in this paper is worth mentioning for several reasons:

1. Its effects can be considered to be those of a UV-A delivering device, as there is no UV-C and no detectable UV-B. The visible radiation beyond 400 nm does not interfere with the biological problems under study.

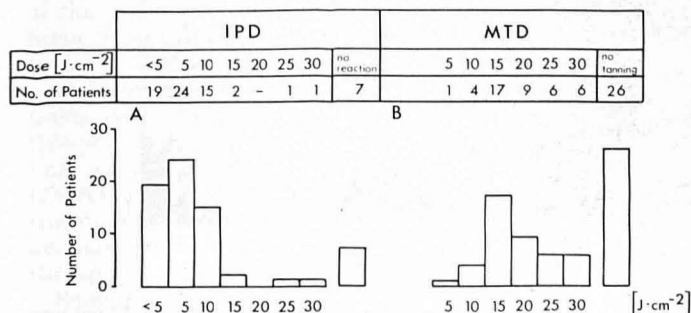2. In addition, the size of the irradiated area on the human

Dose [J·cm^{-2}]		IPD							MTD					
Dose [J·cm^{-2}]	<5	5	10	15	20	25	30	no reaction	5	10	15	20	25	30	no tanning
No. of Patients	19	24	15	2	–	1	1	7	1	4	17	9	6	6	26

Fig 6. Threshold doses for immediate pigment darkening (*IPD*) and minimal tanning dose (*MTD*). Single exposures to the volar aspect of the forearm with 5–30 J·cm^{-2} UV-A. Most of the patients had an IPD-threshold-dose of 10 J·cm^{-2} UV-A or less. The MTD was read at 24 hr.
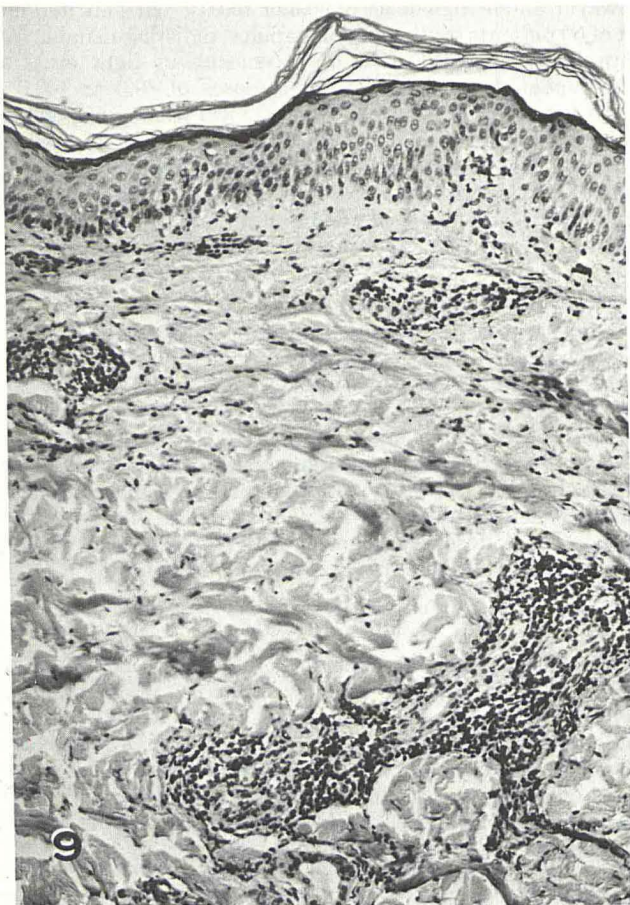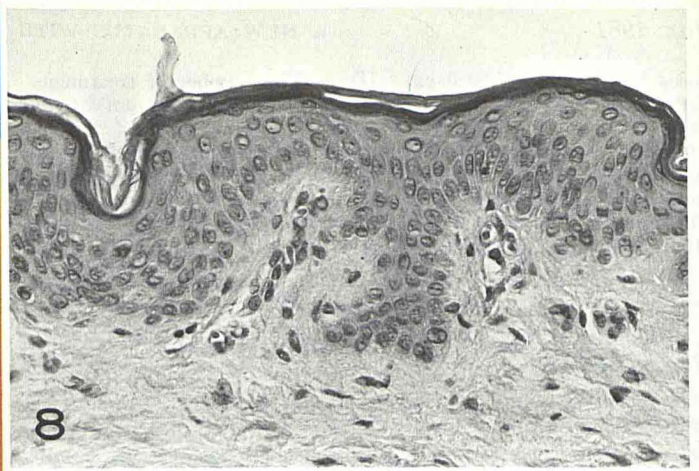
Fig 7. Uniform dark brown hyperpigmentation after 19 treatments (780 J·cm⁻² UV-A) of entire irradiated back except for 8 × 12 cm lead-stencil protected area on upper left back.

Fig 8. Histological appearance of normal skin 72 hr after application of 60 J·cm⁻² UV-A. There are no UV-damaged cells and the tissue looks quite normal.

Fig 9. Histology of polymorphous light eruption experimentally induced by 60 J·cm⁻² UV-A. Biopsied 24 hr after irradiation. Perivascular round cell infiltrate, minimal spongiosis, slight subepidermal edema (hematoxylin-eosin stain).

Fig 10. Treatment of vitiligo to right arm only after 33 treatments. Dark brown hyperpigmentation of normal skin and spotty follicular bound repigmentation. Persistent non-scaling erythema in vitiliginous skin.

skin is sufficiently large to treat, for instance, the upper chest or back, one half of an extremity or the face at one time. Full body irradiation is also possible by a combination of five units UVASUN 5000.

3. Likewise important is the fact that the time required for diagnostic or therapeutic procedures is remarkably short. For instance, at a skin-target distance of 0.3 m a dose of 30.0 J·cm^{-2} UV-A is delivered within 5.7 min.

One of the interesting observations of the study was the induction of delayed (permanent for days to weeks) brownish tan in almost every subject. Tanning is achievable without a UV-B-burn (or "sunburn"). Neither clinically nor histologically were signs of phototoxic damage to the epidermis detected. Epidermal hyperplasia, the so-called "Lichtschwiele" [14] was not induced, and also the number of corneocyte layers was not affected. Subjects receiving multiple treatments carry a brown tan, indistinguishable from sun-induced tanning or photochemotherapy (8-MOP-UV-A = PUVA) induced tanning.

We observed immediate pigment darkening following doses as low as 5.0–10.0 J·cm^{-2} UV-A. We feel unable to decide at what time delayed melanogenesis follows immediate pigment darkening. The adverse long-term effects of high doses of UV-A upon various components of the human skin are unknown. Epidermal as well as dermal UV-A induced tissue alterations are discussed. It seems likely from preliminary animal experiments (symposium on the biological effects of UV-light, German Department of Health, February 27–28, 1980) that UV-A does not induce actinic keratosis or tumors. The long term effects on the dermis have yet to be evaluated.

In this context an interesting question arises. How good a shield is a UV-A tan against the deleterious effects of UV-B on skin, in particular for the prevention of solar elastosis, actinic keratosis or UV-B induced malignant skin tumors? Previous photoaugmentation studies [15, 16] considered only immediate effects from UV-B, such as erythema and sun-burn cells, although the sunburn protective effect of UV-A-induced tan has been noticed [17] in a short-term experiment. It is conceivable that a good UV-A tan provides photoprotection.

One of the most useful applications of this instrument was seen in photopatch testing. It is convenient to irradiate large sites, e.g., one half of the back of a patient at one time.

The reproduction of skin lesions of polymorphous light eruption has puzzled many investigators. Repeated, tissue-damaging doses of UV-B most often used and prescribed for the reproduction en miniature, do not mimic clinically or histologically what is so persistently produced by natural sun [18]. With high intensity grating monochromators (Bausch & Lomb, U.S.A.) we could never elicit skin lesions in our patients, neither with UV-A or with UV-B. With this new UV-A apparatus we were now able to reproduce papulo-vesicular skin lesions, with 1 to 3 applications of UV-A. Histologically, the edema and cellular infiltrate were like its natural variant. So far polymorphous light eruption has been diagnosed clinically and by excluding other differential diagnoses. We suggest that the reproduction of skin lesions of polymorphous light eruption with the technique described in this paper provides an additional diagnostic tool.

Light or sunburn-radiation is often prescribed for acne patients as it is common belief that it ameliorates the disease. In this preliminary experiment, even high doses of UV-A did not improve non-inflammatory nor inflammatory acne lesions. The UV-A tan camouflaged the papulo-pustules. Our results support the notion of other investigators [19] who were unable to demonstrate beneficial effects on acne vulgaris by UV-phototherapy.

Some repigmentation was seen in 1 out of 8 patients with vitiligo. Repigmentation arose around follicular openings. The vitiliginous skin always showed a persistent erythema lasting many days, up to 10, after the last UV-A application. This was never followed by scaling. The nature of this erythema is obscure. Immediately after the irradiation with high doses of

UV-A, the skin of most of the normal subjects showed a definite erythema. Normally it faded within 1 hr. Twenty-four hours after UV-A application, an erythematous response was seen only in a few subjects. At that time pigment blended already into the erythema. It is well known that similar to the erythemogenic effects of UV-B, UV-A also induces redness to the skin [5]. It is appropriate to use the term erythema dose (or minimal erythema dose MED) for the particular wavelength under study, e.g., MED-UV-B, MED-UV-A [20]. It is also necessary to differentiate between the wavelength specific erythema and heat- or radiant-energy induced erythema. Heat erythema may be a problem when working with close skin-target distance, e.g., below 0.5 m with UVASUN 5000. Heat erythema fades soon, usually within 1 hr and flares beyond the irradiated site. At present we cannot differentiate it exactly from UV-A induced immediate erythema. Often the erythema is obscured by the simultaneous appearance of immediate pigment darkening from UV-A. For this reason the MED-UV-A was not established in the present investigation.

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