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Influence of UVA pre-exposure on UVB-induced erythema

A chromometric study

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We examined the influence of ultraviolet A (UVA) pre-exposure on UVB minimal erythemal dose in 9 Caucasian subjects. Three zones were tested. One zone received only UVB, the second zone received a low UVA dose+UVB, and the third zone received a high UVA dose+UVB. Each zone was divided into 9 circles receiving increasing doses of UVB in order to obtain 3 different UVA-exposed series of 9 circles. Visual and chromometric readings were performed 24 h later. Pre-exposure to UVA caused variations in the slope of the dose-response curve (colorimetric index as a function of the UVB dose). In relation to UVB erythema, these variations indicated a protective effect for 6/9 subjects and an aggressive effect for 3/9 subjects. No predictive criteria were found for inclusion within a group.

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The use of ultraviolet A (UVA) sun-tanning beds has continued to increase despite systematic media information campaigns reporting the dangers of their utilization. For example, in 1992, the number of Americans using tanning beds was estimated at 1 000 000 per day (1). This commercial sector brings in an estimated 1 billion dollars in the U.S. (2) annually. Sun-tanning beds, which originally emited the extremely aggressive UVB and UVC rays, now emit only UVA. UVA radiations have long been considered as less aggressive and to result only in cutaneous aging. However, recently, it has been shown that they are capable of inducing a number of lesions, principally in the dermis. Indeed, while UVB rays penetrate only into the epidermis, UVA rays penetrate to the dermis. Among the lesions induced, we found: erythema: UVA rays are nevertheless 1000 times less erythematogenic than UVB (3–7); cellular damage with lesions at the DNA level: pyrimidine dimer, 6–4 adduct, strain breaks (8, 9); epidermal hyperplasia (10); deficit in Langherhans cells (10); dermal inflammatory infiltration (10); and modification of the immune system (11).

In this context, it was proposed that UVA, as UVB, could be carcinogenic. Several research teams conducted studies to clarify UVA action on animal and human models. It was shown by Strickland (12), Sterenborg & van der Leun (13) and by Roffo (14) as early as 1934 that UVA could induce cutaneous cancers in rats. Nevertheless, Staberg et al. (15, 16) did not observe tumors in hairless mice irradiated with UVA.

In humans, several epidemiological studies have shown a link between the utilization of sun-tanning beds and carcinogenic risk. In 1992, Dinehart et al. (17) showed a correlation between the use of sun-tanning beds and the appearance of baso- and spino-cellular cancer in adults aged 30 years or less. The same correlation was shown with malignant melanoma (18–20). Moreover, two larger studies, including approximately 500 patients and 500 healthy volunteers performed by Walter et al. (21) and Westerdhal et al. (22), showed a link between UVA exposure and the appearance of malignant melanoma.

The mechanisms of this carcinogenesis are not yet known. Certain authors suggest the inter-

vention of oxygen free radicals and/or endogenous photosensitizers (23, 24). The study of UVA's effects on the skin, far from being complete, seems to be an important element in the understanding of cutaneous carcinogenesis.

Solar radiation is composed of UVA and UVB; therefore, it is important to examine the synergic effect of the two types of radiations on lesion formation specific to one or the other. Studies have shown that these effects exist and are non-negligible. For instance, Staberg et al. (15, 16) and Willis et al. (25) showed that solar-simulated exposure followed by only UVA exposure resulted in a photo-augmentation of the number of tumors in mice. It must be noted that in this study, the control group irradiated with only UVA rays did not develop tumors. The observed phenomenon was a photo-augmentation and not a photo-addition.

We have performed a similar study involving UVB erythema in the presence or absence of UVA irradiation in healthy volunteers. This was done with the purpose of investigating solar and/or suntanning bed UVA effects on the development of cutaneous lesions.

Our aim was to examine and quantify the influence of UVA exposure on UVB erythema. Nine subjects received UVB exposure preceded or not by UVA exposure at an infra-erythematogenic dose.

Material and methods

Subjects

The subjects included in this study were 9 fair-skinned healthy volunteers with phototype II or III, aged 18–45 years old, who had not undergone sun exposure for a period of 2 months preceding our study. Table 1 shows the phototype, melanotype (29) and MED of each subject.

Irradiation equipment

UVB irradiation was performed with Dermollum III Muller equipped with a xenon lamp filtered by a WG 305 (1 mm) Shott filter. The UVA lamp was a medium pressure lamp HPA 2000s (UVA France). The irradiance of the lamp below 340 mm was about 4% of the total irradiance. Dosimetry was performed with an Osram Centra dosimeter with UVA and UVB probes.

Table 1. Phototype, melanotype and MED (minimal erythemal dose) of subjects

Subject	Phototype	Melanotype	MED mJ/cm^2
1-BAC	II	RxBr	19
2-PAI	II III	RxBr	30
4-BLA	II	RxBr	24
5-DEN	II	BrBl	19
6-ROU	II III	RxBr	25
7-DUP	II	RxBr	20
8-HOL	II	RxBr	16
9-CAL	II	BrBr	16–20
10-LER	II	RxBr BrBl	16

Determination of the MED

The MED is the minimal dose required by the subject in order to clearly see a border-defined erythema 24 h after exposure. Increasing doses of UVB (geometric progression of 1.25) were delivered to 9 adjacent zones on the volunteers' upper backs. MED determination was achieved by visual and chromometric assessment 24 h after the initial exposure.

Irradiation procedure

Two 10×10 cm areas on the upper back (close to the areas which were used for MED determination) were irradiated with respectively 22 and 44 J/cm^2 of UVA. After a 10-min pause, the same zone received 9 increasing doses of UVB (geometric progression of 1.25) on 9 circular zones. Subjects received 0.2 to 2 times their MED. Visual and chromometric assessments were performed 24 h later.

Chromometric assessment

Three trials per zone were performed 24 h after irradiation on exposed and unexposed skin. Variations of chromometric parameters L, a, b were measured: "a" variations were representative of the erythema intensity (red component of skin), "b" variations were representative of pigmentation intensity (yellow component of skin color), and L variations were representative of skin luminosity. Colorimetric index, CI, was calculated as follows:

$$CI=(\Delta L^2+\Delta a^2+\Delta b^2)^{1/2}$$

where Δ a is the difference between "a" obtained from unexposed skin and "a" obtained from exposed skin. Δb and ΔL were defined in an identical manner. In the case of UVA- and UVB-exposed zones, chromometric parameter variations were calculated between UVA-exposed skin and UVA+UVB-exposed skin. This was done to take into account color variations originating from immediate pigment darkening.

The colorimetric index was simplified to determine the erythemal rise, as b variations were negligible in regard to a and L variations.

The erythemal rise is defined in the quasilinear part of the curve:skin color variations as a func-

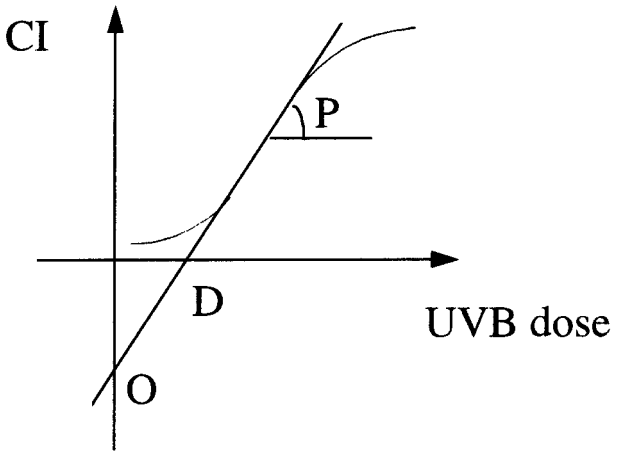

Fig. 1. General aspect of colorimetric index (CI, representing epidermal color variation) as a function of the UVB dose received by the subjects.

tion of the dose received (Fig. 1). We concentrated our interest on the slope of the rise as well as intersection points on the axes.

Results

The 9 subjects received 3 series of 9 circular UVB exposures: UVB alone; UVB preceded by UVA 22 J/cm^2; and UVB preceded by UVA 44 J/cm^2. We compared the erythemal responses obtained from the zones which received only UVB with the zones that received UVA and UVB. This was done both by visual assessment of erythema and by chromometric assessment of skin color by Minolta CR 200 chromometer.

Visual assessment

Table 2 shows the values of MED (minimal erythemal dose) obtained for the 3 zones tested.

Pre-exposure to UVA resulted in a decrease in MED values for 3 out of 9 volunteers. No UVA influence was noted for the other 6. This suggests that if there was an effect of the pre-exposure to UVA on UVB erythema, it was relatively weak and not readily apparent visually.

Table 2 also shows a visual graduation of immediate pigment darkening (IPD). Weak pigmentation was exhibited in 7 out of 9 subjects. Only 2 subjects (numbers 6 and 7) exhibited strong pigmentation on the zone exposed to 44 J/m^2 UVA. This allows us to suppose a low interaction between IPD and erythema as far as visual assessment is concerned.

Table 2. Data obtained by visual assesment of skin erythema and pigmentation (T: trail)

	UVB	UVB+UVA (22 J/cm^2)		UVB+UVA (44 J/cm^2)	
Subject	MED mJ/cm^2	MED mJ/cm^2	Pigmentation	MED mJ/cm^2	Pigmentation
1-BAC	19	19		19	
2-PAI	30	24	0	30	T
4-BLA	24	24	T	24	+/–
5-DEN	19	15	T	15	+/–
6-ROU	25	25	+/–	25	+/–
7-DUP	20	20–25	+/–	20–25	+/–
8-HOL	16	12–16	T	12	+/–
9-CAL	16–20	20	T	16–20	+/–
10-LER	16	16–20	T	16–20	+/–

Chromometric results

The erythemal rise represents the colorimetric index values as a function of the UVB dose received. They were determined for each subject. The slopes (marked P), the vertical axis intersection points (marked O) and the horizontal axis intersection points (marked D) were compared between the zone exposed to UVB and the zones exposed to UVA and UVB. "D" represents the threshold dose which induced a color variation and will be considered as chromometric MED.

Analysis in function of erythemal rise slope values

Table 3 includes all the subjects for the parameter variations P, O and D on the zones which received both UVA and UVB exposures.

Two groups of subjects were readily apparent: for 6 out of 9 subjects (group A), the UVA pre-exposure resulted in a decrease in the erythemal rise slope, while for 3 out of 9 subjects (group B), an increase was observed.

Predictive criteria for group selection

We identified two types of reactions that we tried to correlate to classically used criteria for this type of study (27–29): phototype, melanotype, MED and immediate pigmentation in response to UVA.

Phototype: the 9 subjects have similar phototypes. Five out of 6 in group A and 2 out of 3 in group B have phototype II. One subject has phototype II–III in each of the groups. Phototype is therefore not a criterion for either group.

Melanotype: Group A includes 4 subjects of RxBr melanotype, one subject BrBr and one subject BrB1. Group B includes 3 RxBr subjects. The populations of these two groups are approximately equivalent. Melanotype is therefore not considered a predictive criterion.

MED: Visual MED values are statistically equivalent in both subject groups. The average

Table 3. Variations in percentage of chromometric parameters O, P, D between UVB-exposed and UVA+B-exposed skin

		6-ROU	9-CAL	10-LER	1-BAC	4-BLA	5-DEN	2-PAI	7-DUP	8-HOL
UVA=22 J/cm^2	% P	−49	−26	−24	−11	−63	−28	+87	+31	+47
	% O	−42	−13	−26	−26	−81	−50	+92	+47	+60
	% D	+31	+31	−4	−34	−68	−46	+4	+31	+25
UVA=44 J/cm^2	% P	−28	−12	−19	−49	−34	−25	+56	−4.6	+31
	% O	−25	+2	−19	−75	−47	−38	+82	+0.1	+32
	% D	+7	+31	+0.2	−71	−60	−31	+35	+4	+9

MED for group A is 20 mJ/cm^2 (SD 3 mJ/cm^2) and 22 mJ/cm^2 for group B (SD 7 mJ/cm^2). The MED value is thus not a predictive criterion either.

UVA pigmentation: Table 4 shows the chromometric measures of the normal non-irradiated skin and the measures of the skin exposed to only UVA. No significant difference in intensity of immediate pigment darkening appeared between the two groups. Therefore, UVA pigmentation does not seem to be a tool that can discriminate between the two groups.

Analysis of group A behavior

Group A includes the 6 subjects in whom UVA pre-exposition led to a decrease in the slope of the dose-response curve. This decrease is on average 33% for a UVA exposition of 22 J/cm^2 and on average 27% for a UVA exposition of 44 J/cm^2. This suggests an UVA protective effect in relation to UVB erythema for high UVB doses. Two behaviors were observed with regards to the axis intersection points (O):

Group A1: This group includes 4 out of 6 subjects. We observe a decrease in "O" (an average of 45% for 22 J/cm^2 and 44% for 44 J/cm^2) and "D" (an average of 38% for 22 J/cm^2 and 40% for 44 J/cm^2). The UVA pre-expositions therefore seem to have the double effect of being protective (by the decrease of "P") and aggressive (by the decrease of the chromometric MED).

The subjects' response in this group is illustrated in Fig. 2a.

Group A2: This group includes 2 out of 6 subjects. We observe a decrease in "O" (an average of 27% for 22 J/cm^2 and 11% for 44 J/cm^2) and an increase of the chromometric MED (intersection point at horizontal axis: an average of 31% for 22 J/cm^2 and 19% for 44 J/cm^2). This suggests a protective effect as a result of the UVA pre-exposure. This can be seen by an erythemal sensitivity (in terms of chromometric MED) which is increased by an average of 25% and a decrease of erythemal slope by an average of 30%. This group's behavior is illustrated in Fig. 2b, and the data summarized in Table 5.

Table 4. Average skin color variations (colorimetric index) induced by UVA for the two groups of subjects

	UVA (22 J/cm^2)	UVA (44 J/cm^2)
Group A	4.0±1.3	3.8±1.0
Group B	3.6±1.8	3.5±1.5

Analysis of group B reactions

This group includes 3 subjects who show, in the presence of UVA pre-exposition, an increase of the slope for their dose-response curve (+55% for 22 J/cm^2 and +43% for 44 J/cm^2). This is accompanied by an increase in the horizontal and vertical axis intersection points (Table 5). The increase of P translates into an agressive effect in response to UVA pre-exposition, whereas the increase of D translates into a protective effect by the decrease of erythemal sensitivity for the weak doses of UVB received. We find here a dual UVA aggression-protection effect on UVB-induced erythema. The subjects' reactions are illustrated in Fig. 2c.

Analysis of subject reaction in function of chromometric MED variations

If we consider the chromometric MED, ("D"), as the principal parameter for data analysis, we ob-

Table 5. Mean variations of O, P, D for the two groups

	Group A		Goup B
	1	2	
P (slope)			
UVA=22 J/cm^2	−33%		+55%
UVA=44 J/cm^2	−27%		+43%
O (y axis intersection point)			
UVA=22 J/cm^2	−45%	−27%	+66%
UVA=44 J/cm^2	−44%	−11%	+38%
D (chromometric MED)			
UVA=22 J/cm^2	−38%	+31%	+20%
UVA=44 J/cm^2	−40%	+19%	+16%

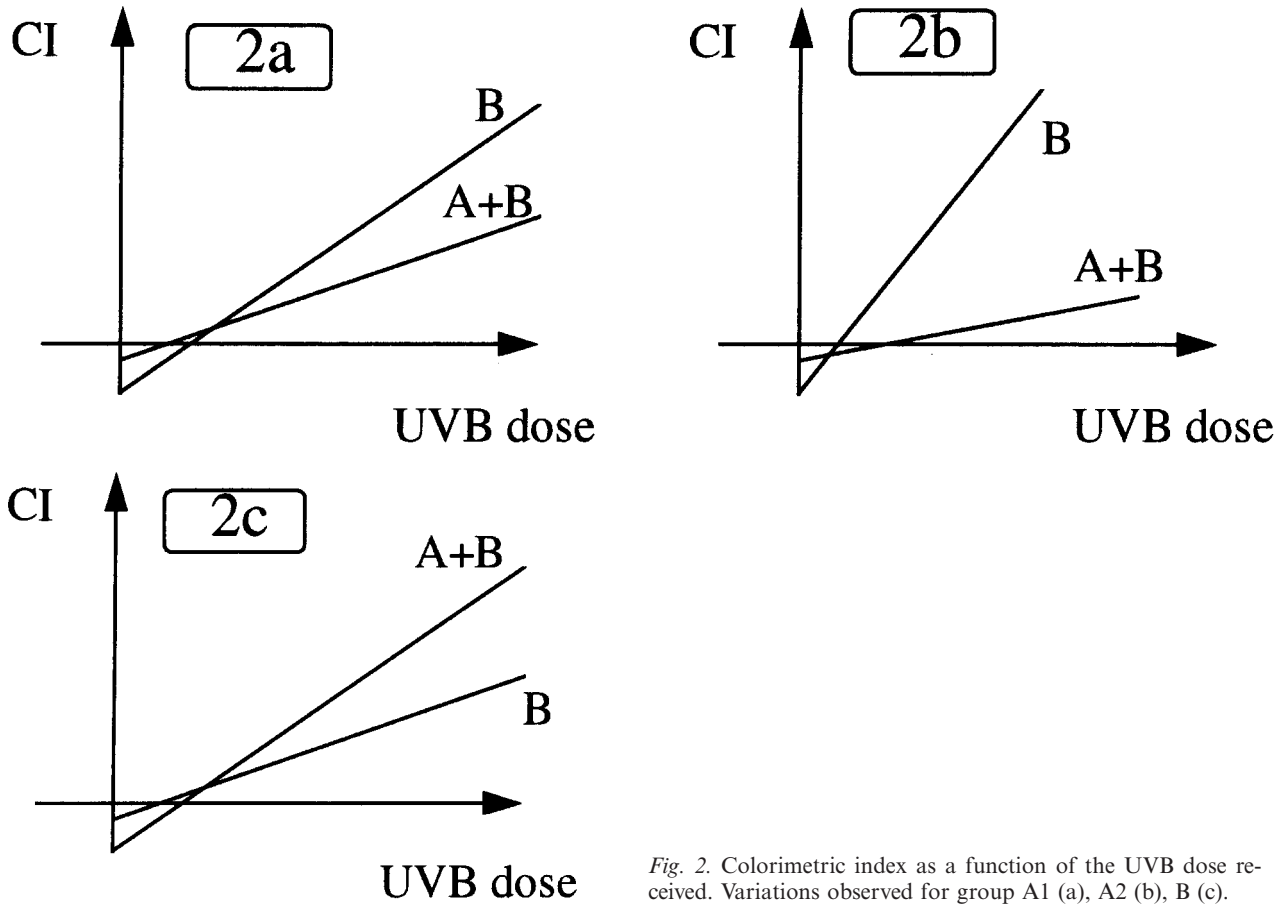

Fig. 2. Colorimetric index as a function of the UVB dose received. Variations observed for group A1 (a), A2 (b), B (c).

serve after UVA pre-exposure, a decrease of 38% (average) in 4 out of 9 subjects and an increase of 24% (average) in 5 out of 9 subjets. The variations observed (Table 5) are non-negligible (from −68 to 31%); the fact that the subjects are divided into two numerically equivalent groups can not be attributed to a weak effect. As regards function of chromometric MED, UVA pre-exposure has a protective effect against UVB erythema in 5 out of 9 subjects and an aggressive effect in 4 out of 9 subjects.

Influence of the UVA dose received

Table 3 illustrates the parameter variations for the two UVA doses received.

In 8 out of 9 subjects the evolution of the three parameters is in the same direction (as one would anticipate) whether the UVA dose is 22 or 44 J/cm^2. However, in 6 to 8 cases out of 9 (depending on the concern parameter), the amplitude of the variations is weaker for the highter UVA dose.

This suggests two hypotheses:

1. The increase of the UVA dose applied raises the interference of redness with the immediate pigment darkening in the colorimetric readings and our method is therefore inadequate. We stress that we used the zones which only received UVA for the control skin readings in order to eliminate the most interference possible with immediate pigment darkening.

2. The observed phenomenon can be obtained by a relatively weak UVA dose. We observed a saturation for stronger doses. This suggests also the possible presence of several superimposed effects of different natures explaining, perhaps, the aggression-protection duality observed in 7 out of 9 subjects in the analysis of the function of P.

Conclusion

We studied the influence of UVA pre-exposure on the intensity of erythema induced by UVB in 9 Caucasian subjects. Using chromometric readings of the skin, we plotted the dose-response curves and we compared their characteristics in the presence and absence of UVA irradiation.

For Caucasian subjects with equivalent phototype, melanotype and MED, we found that pre-exposure to UVA had no significant effect on UVB-

induced erythema as far as visual readings are concerned. Nevertheless, the doses used were progressively increased by a geometric progression of 1.25. This means that there was a difference of 25% between the two successive doses. The error (potentially) made in the visual estimation of MED is therefore 12.5% of the indicated dose. This error could be made for the two visual readings (MED B, MED AB). In this case, the global error could reach 25% of visual MED; however, if the researched effect is weak it can not be seen visually.

Our chromometric analysis allowed us to observe, for 7 out of 9 subjects, both a protective and aggressive effect due to UVA, and for 2 out of 9 subjects, a protective effect. The protective effect is linked in 6 out of 9 subjects to the decrease in the slope of the dose-response curve in the presence of pre-exposure. The aggressive effect, in 4 out of 9 subjects, is due to a decrease in the chromometric MED, and in 3 out of 9 subjects to an increase in the slope of the dose-response curve in the presence of pre-exposure.

It must be noted that the variations of the chromometric MED observed are determined by extrapolating the erythemal rise on the horizontal axis in the nonlinear part of the curve. These values do not correspond to measured values, so the most relevant criteria from a clinical view seemed to be the slope of the dose-response curve.

If we concentrate on the slope variations, a protective effect is indicated by a slope decrease in 6 out of 9 subjects. An aggressive effect in 3 out of 9 subjects is observed by a slope increase. No criteria (phototype, melanotype and MED) permitting the prediction of the subject's reaction type could be defined.

Few data are available in recent literature concerning this problem. In the 1970's, van der Leun & Stoop (30) observed a protection of 20 to 30% due to UVA in relation to UVB erythema. The UVB irradiation source was monochromatic, emitting at 300 nm. UVA exposure was given before UVB. However, photo-addition of UVA and UVB erythema was observed by van Weelden in 1980 (31) regardless of the irradiation order used. Contrary to these results, Willis in 1973 (32) observed an aggressive effect due to pre-exposition to UVA, which resulted in a decrease in UVB MED. Spiegel et al. (33) exposed 17 subjects to 7.5 J/cm^2 UVA followed by an exposure to UVB 0.5 times their MED. He observed 24 h after exposure an erythema in 14 out of 17 subjects. This suggests an aggressive effect of UVA. Boer et al. (34) made the same observation and showed a decrease of 20% of the MED after UVA exposure of 10 J/cm^2.

The lack of homogeneity in results is probably due to several factors: 1. difference in spectral delivering of different UV sources; 2. use of visual assessment; 3. the recruitment of subjects is done principally on criteria linked to erythema, (e.g. phototype) whereas other phenomena play a role in this type of experiment (e.g. immediate pigment darkening).

The phototype, melanotype, and MED do not take into account the subject's capacity to develop immediate pigment darkening. The most pertinent classification seems to come from Chardon et al. (26). This classification is based on the subject's skin color. According to this classifcation, 3/9 subjects had "intermediate skin", and among these three, 2 were in Group B; 6/9 subjects had "light skin" and one of these 6 was in Group B. In our study, this classification did not permit us to distinguish Group A from Group B. The number of subjects in Group B was not sufficient.

A further factor is that it is important to situate the dose of UVA exposure in relation to the UVA MED. In our study, no UVA erythema was observed. Furthermore, we were careful to eliminate the color variations originating from UVA pigmentation in the calculation of the colorometric index for the zones irradiated by UVA and UVB.

It is also necessary to situate the dose of UVA exposure in relation to the minimal immediate pigment darkening dose. In our study, all UVA exposures were done with doses higher than the minimal immediate pigment darkening dose.

Certain researchers did not measure precisely the UVA doses received by the subject. They did not indicate whether the doses induced immediate pigment darkening. Therefore, comparison with our results is difficult.

In conclusion, we observed that UVA pre-exposition induced a variation in the erythemal slope. This variation resulted in a protective effect in relation to erythema in 6/9 subjects. Predictive criteria have to be defined and compared with those used to assess non-melanoma skin cancer sensitivity.

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