The Relationship of Skin Color, UVB-Induced Erythema, and Melanogenesis

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The relationship between skin color, delayed erythema, and delayed tanning (DT) elicited by a single exposure of UVB was investigated. Both constitutive and facultative pigmentation were determined by skin reflectance using a melanometer. Skin reflectance using visible light was well correlated to the minimal immediate pigment darkening dose elicited by UVA irradiation, which may relate to epidermal melanin content, a determinant of skin color. Minimal erythemal dose (MED) was well correlated to skin color, but there was less correlation between minimal melanogenic dose and skin color or the MED, since melanogenesis is controlled by genetic factors. DT also correlated to the dose of UVB in terms of MED. A coefficient of the regression line of DT may suggest the tanning capacity of skin. The possibility of detecting mild photosensitivity in individuals from a regression line of the MED on skin color is suggested.

The erythema response of skin to ultraviolet radiation (UVR), i.e., the sunburn reaction, has long been known to correlate with skin color. Dark-skinned individuals are able to tolerate a longer sun exposure than light-skinned individuals.

Skin color in humans is mainly determined by the quantity, quality, and distribution of the complex biopolymer, melanin, and its organized form, the melanosome [1,2]; melanin is thought to protect skin from the deleterious effects of UVR. Olson, Gaylor, and Everett reported that the minimal erythemal dose (MED) in various skin colors correlated with melanosome size, quantity, density, and distribution [3]. Facultative pigmentation elicited by PUVA (psoralen plus UVA) or UVB has been reported to increase the MED by a factor of 3–8 [4].

From a clinical point of view, a more quantitative relationship between the skin color and the sensitivity to UVR of an individual, as measured by MED, may be useful, since by this quantitation mild photosensitivity or phototoxicity can be recognized. In this report we have investigated the erythemal and melanogenic skin responses to UVB in relation to skin color as determined by skin reflectance of visible light. We found a linear correlation between them, and a regression line of the MED on skin color might permit us to estimate mild photosensitivity.

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Abbreviations:
DT: delayed tanning
MED: minimal erythema dose
MIPDD: minimal immediate pigment darkening dose
MMD: minimal melanogenic dose
PUVA: psoralen plus UVA
SLE: systemic lupus erythematosus
UVR: ultraviolet radiation

MATERIALS AND METHODS

MEDs, minimal melanogenic doses (MMDs), and minimal immediate pigment darkening doses (MIPDDs) were determined on the unexposed back skin of 8 healthy Japanese volunteers (4 males, 4 females) without apparent skin diseases, during spring to early summer 1983. Two females suspected of having systemic lupus erythematosus (SLE) were also tested. The MIPDD was defined as the lowest exposure dose of UVA that elicited an immediate pigment darkening with 4 sharp borders.

The UVB source was an array of 4 FL20s-E fluorescent tubes (Toshiba) set in parallel within a Dermalay (Eisai) lamphouse; the UVA source was an arrangement of 20 Sylvania fluorescent tubes usually used for PUVA therapy. The spectral irradiance of the UVB source was 280–370 nm, with the peak irradiance at 305 nm; the spectral irradiance of the UVA source has been published elsewhere [5]. The intensity of the radiation at the skin surface was 2 mW/cm^2 for UVB, and 5.5 mW/cm^2 for UVA, as measured by a Torex UV305/365 spectroradiometer.

The MIPDD was determined immediately after UVA exposure; the MED and MMD were determined 24 h and 7 days after a single UVB exposure, respectively. For the determination of the MIPDD, 2 × 2 cm^2 skin sites were irradiated with UVA using 2 J/cm^2 increments. For the determination of the MED, 1 × 1 cm^2 skin sites were irradiated with UVB using 10 mJ/cm^2 increments. The MMD was determined, in most cases, by the administration of 1.0, 1.2, 1.4, 1.6, and 2.0 MEDs of UVB.

Skin color was measured using a DS-506 melanometer (Keihin Densokkuki) which measured skin reflectance of visible light at 457 nm. The melanometer could detect the lightness of 1 × 1 cm^2 skin sites. The melanometer was calibrated by a white ceramic board before each measurement, and at least 4 measurements were done at each site. Data were expressed as percentages of skin reflectance, i.e., 100% meant complete reflection and 0% meant complete absorption.

RESULTS

Fig 1 shows the percent of skin reflectance measured by the melanometer was well correlated to MIPDD, which may be related to the epidermal melanin content ($r = 0.96$, $p \leqq 0.01$). Thus, in our experiment, the results obtained by the melanometer seemed to relate to the epidermal melanin content.

When the MEDs were plotted against the percent of skin reflectance (Fig 2), there was a good correlation ($r = -0.96$, $p \leqq 0.01$). An interesting observation was that the value obtained from the patients suspected of having SLE deviated considerably from the regression line in Fig 2 (skin reflectance: 61.2% and 60.5%; MEDs: 90 mJ/cm^2 and 80 mJ/cm^2, respectively).

The relationship between delayed tanning (DT), as measured by the melanometer, and MED is shown in Fig 3. DT correlated linearly with UVB dose in the 1–2 MED range. The dotted band in Fig 3 represents skin color as measured by skin reflectance (mean ± SE × $t_{0.05}$). The point of intersection between the regression line of DT on the MED and skin color line may represent the MMD. In fact, the value obtained from the figure coincided with the MMD, shown in Fig 3 by an arrow.

The subject, whose melanogenic response is shown in Fig 3*A*, had lighter skin than do most Japanese, and experienced sunburn after the first exposure to sunlight every summer; however she never experienced severe sunburn because of her high tanning capacity (melanogenic ability), as shown in the figure. On the other hand, another subject, whose melanogenic response to UVB is shown in Fig 3*B*, had well-tanned skin of a

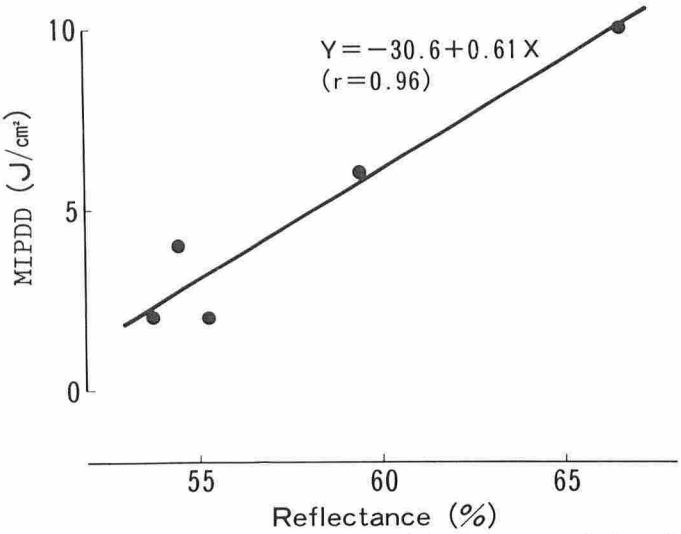

FIG 1. Relationship between skin color (% reflectance) and minimal immediate pigment darkening dose (*MIPDD*) which was defined as the lowest exposure dose of UVA that elicited immediate pigment darkening. The experiments were done on unexposed back skin of healthy Japanese volunteers as described in *Materials and Methods*. Skin color was determined by the melanometer prior to UVA irradiations. MIPDD was determined immediately after the UVA irradiation. Skin color was well correlated to the MIPDD ($p \leqq 0.01$).

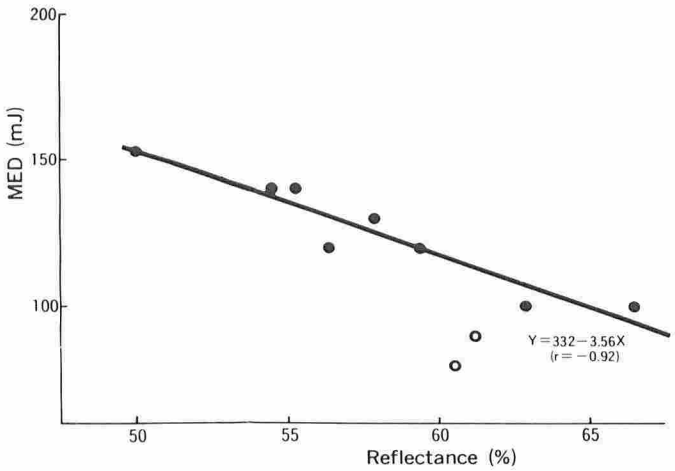

FIG 2. Relationship between skin color (% reflectance) and MED. The experimental conditions were as in Fig 1. MEDs were determined at 24 h after the UVB irradiations. Skin color was well correlated to the MED ($p \leqq 0.01$). MEDs of 2 patients suspected of having SLE were also determined (*open circles*); these deviated considerably from the regression line.

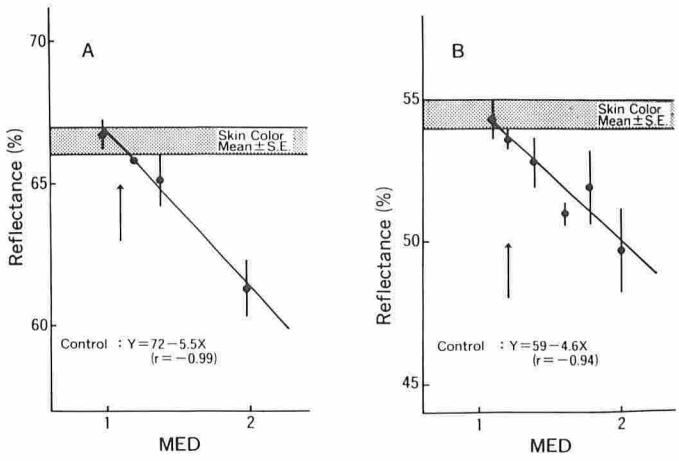

FIG 3. Relationship between UVB dose in terms of MED and DT as measured by the melanometer 7 days after UVB irradiation. The *dotted bands* represent skin color measured by melanometer before UVB irradiation. MMDs are indicated by the *arrows*. Data represent mean ± SE × $t_{0.05}$. *A*, DT regression line of 36-year-old female. *B*, DT regression line of 42-year-old male. The correlations were statistically significant ($p \leqq 0.01$).

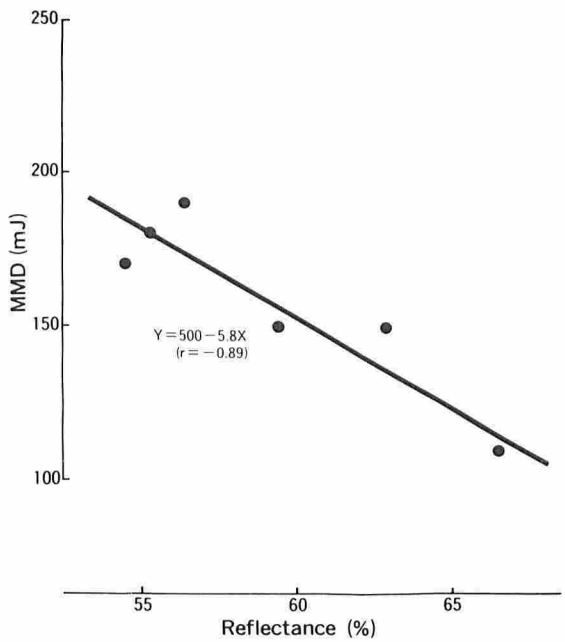

FIG 4. Relationship between skin color (% reflectance) and MMD. MMD was estimated visually at 7 days after the UVB exposure. The correlation was statistically significant only at $p \leqq 0.05$.

deep brown color, but he still had a high tanning capacity as shown by the coefficient of the regression line ($r[\delta y/\delta x] = -4.6$).

The correlations between the percent of skin reflectance (skin color) and MMD ($r = -0.89$), and between the MED and the MMD ($r = 0.82$) were relatively poor, as shown in Fig 4 and Table I. They had statistically significant correlation only at $p \leqq 0.05$.

TABLE I. *The relationship between minimal erythema close (MED) and minimal melangenic dose (MMD)*

	Age	Sex	MED (mJ/cm²)	MMD (mJ/cm²)
H. I.	39	F	90	110
K. U.	35	F	100	110
S. O.	32	M	100	130
M. O.	34	F	100	150
M. O.	31	F	120	190
M. I.	35	M	140	170
F. K.	40	M	140	180
Mean ± SE × $t_{0.05}$			112.9 ± 54.5	148.6 ± 87.0
Regression line:[a] MMD = 0.84 + 1.31 MED				($r = 0.82$)

[a] The regression line was statistically significant only at $p \leqq 0.05$.

DISCUSSION

Skin color is produced by visible light remitted from skin. Chromophores which absorb visible light, such as carotenoids, melanin, oxyhemoglobin, and reduced hemoglobin, determine the amount and wavelengths of remitted light, and thus an individual's skin color [6]. Melanin is a major determinant of skin color, as it is distributed in keratinocytes in the form of melanosomes. Since the absorption spectrum of melanin ex-

tends into the UV region, it is also an important protectant against UV damage to skin. Anderson and Parrish showed that epidermal thickness and melanization were the major protective factors against the effects of UV wavelengths less than 300 nm, while the attenuation of UVA and visible radiation was primarily via melanin [7].

We estimated skin color by measuring the visible light remitted by skin. The data obtained by the melanometer did not reflect the absolute melanin content of skin, since the contribution of other factors, such as the surface structure of skin, and the absorption or reflection of light by chromophores or molecules other than melanin, must be recognized. In addition, the distribution pattern—not quantity—of melanin, e.g., melanosome size, density, and distribution, may have contributed to the data obtained. Because we used Japanese subjects only, the percent of skin reflectance was well correlated to MIPDD ($p \leqq 0.01$), which may be related to epidermal content of the melanin (Fig 1); thus, in this experiment the difference in the quality of melanin (mainly the distribution pattern) could be neglected.

The MED was correlated to skin color ($p \leqq 0.01$, Fig 2), but there was relatively poor correlations ($p \leqq 0.05$) between skin color and MMD (Fig 4) and between MED and MMD (Table I). The correlation between MED and skin color is not surprising, since the quantity of melanin influences both the protection of skin from UVB and the skin color. Previous reports also confirm these observations [3,4,8–10].

In our experiment, erythema always preceded melanogenesis regardless of their skin color (Table I). Pathak and Fanselow reported that the MMD was the same as the MED (skin type II) or the MMD was distinctly less than MED (skin type III or IV) [10]. The discrepancy between our results and theirs might be caused by the difference in the light source. We used a fluorescent lamp that elicited UVB only (with only little UVA contamination), but they used the full spectrum of sunlight. The difference might also be caused by the subjects used in these experiments; the melanogenic responses of Caucasians and those of Mongolian are quite different.

MMD is one indicator of melanogenic ability, i.e. facultative pigmentation. Facultative pigmentation elicited by UVR or visible light is under the genetic control of the epidermal melanin unit [11]. This genetic control by the epidermal melanin unit may account for the relatively poor correlation between skin color and the MMD (Fig 4) or between the MED and the MMD (Table I). Since constitutive pigmentation is under the same genetic control as facultative pigmentation, the relatively poor correlation ($p \leqq 0.05$) between skin color or the MED and the MMD seemed to be unreasonable, as compared to the good correlation ($p \leqq 0.01$) between skin color and MED. However, the process of pigmentation within the epidermal melanin unit is a complex process which includes production, organization, transference, redistribution, and degradation of melanin. Each process of the epidermal pigmentation may not be uniformly controlled by a single gene. Thus, especially in the acute phase of facultative pigmentation, the new pigment production does not precisely reflect an individual's constitutive skin color. We designate acute melanogenic response as an individual's tanning capacity.

DT, as measured by skin reflectance using the melanometer, increased (decreased in terms of percent reflectance) linearly with UVB exposure dose (Fig 3). Since erythema, a measure of the skin's reaction to UVB, preceded melanogenesis in our experimental condition, MED may be an indicator of DT, as shown in Fig 3. Thus, the coefficient of the regression line in Fig 3 may represent tanning capacity. Tanning capacity may be defined as follows: (tanning by newly formed melanin) − (fading by degradation of melanin)/(UVB dose in terms of MED); in this study degradation of melanin was neglected. MMD and tanning capacity may be useful measures in establishing the most effective photochemotherapy schedules.

The possibility that subclinical photosensitivity can be recognized from the regression line of MED on skin color was suggested in this investigation (Fig 2). Two patients suspected of having SLE showed decreased MEDs as compared to their skin color. These results are still preliminary, and the melanometer may not be able to quantitate skin color beyond the interracial difference. Finally, the relationship between MED and skin color in the Japanese population has been reported, and further research may give us a useful tool in diagnosing mild photosensitivity.

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