Repeated equally effective suberythemogenic exposures to ultraviolet (UV)A1 or narrowband UVB induce similar changes of the dermoscopic pattern of acquired melanocytic nevi that can be prevented by high-protection UVA-UVB sunscreens

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Background: Sunlight modifies the size and the dermoscopic pattern of acquired melanocytic nevi (AMN).

Objective: We investigated whether repeated exposures to equally effective suberythemogenic doses of ultraviolet (UV)A or UVB can induce changes in the dermoscopic features of AMN.

Methods: Twenty volunteers received equally effective doses of narrowband UVB or UVA1. During exposures, an AMN was covered with an opaque tape, another was shielded with the sunscreen, and another was left unprotected.

Results: Nevi exposed to either narrowband UVB and UVA1 showed statistically significant changes in their dermoscopic features: increased size, increase of pigment network, overall color darkening, formation of focal branched streaks, and increased number and size of brown dots and globules.

Limitations: The study is a clinical cohort study on a small number of selected patients.

Conclusion: AMN show similar changes in size and dermoscopic pattern after narrowband UVB and UVA1 exposures. (J Am Acad Dermatol 2008;58:763-8.)

Sunlight has been shown to modify the size and the dermoscopic pattern of acquired melanocytic nevi (AMN).[1-3] However, it is unclear whether these changes are caused by the ultraviolet (UV)B (280-320 nm) or UVA (320-400 nm) wavebands. Previous investigations exposing AMN to artificial UV radiation applied sources with a variety of UVB/UVA ratios.[4-7] Therefore, these earlier studies do not adequately clarify this question.

Abbreviations used:

AMN:	acquired melanocytic nevi
BB:	broadband
COLIPA:	European Cosmetic, Toiletry, and Perfumery Association
NB:	narrowband
SED:	standard erythema dose
UV:	ultraviolet

To address the waveband dependence of UV-induced changes, AMN were exposed to repeated equally erythemogenic doses of UVA1 or narrowband (NB) UVB radiation. In addition, we evaluated the efficacy of a commercially available sunscreen in preventing these changes.

METHODS

Patients

Twenty healthy volunteers with at least 3 AMN on the trunk with similar clinical and dermoscopic features

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were enrolled in this study. Exclusion criteria included: age under 18 years; pregnancy or lactation; any active infectious, inflammatory, or neoplastic disease; personal or family history of melanoma and other skin tumors; atypical mole syndrome; history of photosensitivity; use of immunosuppressive or photosensitizing drugs; participation in other investigational studies in the past 30 days; and likelihood of poor compliance.

The study was approved by the local ethics committee and all participants gave written informed consent before entry.

UV equipment and dosimetry

Our NB-UVB radiation source (Waldmann 7001 cabinet, Waldmann Lichttechnik, Villingen-Schwenningen, Germany) was equipped with 40 100-W lamps (TL-01, Philips, Eindhoven, The Netherlands) with approximately 80% of the emission concentrated in a narrow UVB peak at 312 ± 2 nm, and 20% in the UVA range. For UVA1 radiation we used an irradiation unit (Dermalight UltrA1-24KW, Hönle GmbH, Martinsried, Germany) with UV emission strictly confined in the range from 340 to 400 nm. Both radiation sources emit small amounts of visible light.

Irradiance and emission spectra were measured with a spectroradiometer (SR 9910, Macam Photometrics Ltd, Livingston, Scotland).

Irradiation protocol

As our objective was to compare the effects of two UV sources with different erythemogenic activities, we needed to adjust the doses to compensate. This was done according to the following method. The spectral output of the two radiation sources was quantified with the spectroradiometer. The spectral irradiance of the radiation at any given wavelength λ_{nm} was then multiplied by the effectiveness of this wavelength to cause erythema.[8] For this calculation we used the erythema action spectrum that was proposed in 1987 by the International Committee of Illumination as reference.[9] The effective or weighted irradiance of the two UV sources was the integral of these products in the erythemogenic UV range and was given in Watt effective*m^{-2}. Finally, the erythemal effective dose was obtained by calculating the time integral of the erythemal effective irradiance and was given in Joule effective*m^{-2} units. An erythemal effective dose of 100 Joule effective m^{-2} is called a standard erythemal dose (SED). This unit of measurement is widely used to compare the erythemal effectiveness of different UV sources.[8]

The minimal erythemal dose in participants with skin types II and III would be expected to be between erythemal effective radiant exposures of 200 to 400 Jm^{-2}, equivalent to 2 to 4 SED.[8]

To deliver suberythemogenic doses and to compensate for progressive tanning during sequential UV exposures, the starting dose was set at 1 SED and increased by 1 SED once a week. Three exposures per week were delivered for a total of 4 weeks.

Irradiations were delivered to the whole body surface.

Ten volunteers were randomly selected to receive NB UVB, and 10 to receive UVA1 exposures.

Three AMN with similar clinical and dermoscopic features were selected in each volunteer. During exposures, one AMN was left unprotected, a second one was shielded with an opaque adhesive tape to serve as unirradiated control, and the third nevus was covered with a commercial sunscreen (Dermasol Globalcare Ultra, Biochimici PSN, Bologna, Italy) that provides high protection against both UVB (sun protection factor > 50) and UVA (persistent pigment darkening protection factor = 30), as determined according to the European Cosmetic, Toiletry, and Perfumery Association (COLIPA) guidelines.[10] Its active ingredients are ethylhexyl methoxycinnamate, 4-methylbenzyliden camphor, methylene bisbenzotriazonyl tetramethylbutylphenol, and butyl methoxydibenzoylmethane, which are all commonly used in commercial sunscreen formulations worldwide. The sunscreen was applied according to the COLIPA recommendations.[10] Briefly, at least 15 minutes before irradiation, 2 mg of the product was weighed and applied uniformly to the surface of the nevus and a small area surrounding it (1 cm^2 of skin in all). The sunscreen was applied by an experienced phototherapy technician to guarantee complete coverage.

This investigation was carried out during the winter months, to minimize the effects of concomitant uncontrolled exposure to natural sunlight.

Dermoscopy

Dermoscopic images of all nevi were taken under standardized conditions. Baseline images were taken within 1 week before the first UV exposure. Follow-up images were taken 1 week after the last exposure. A final examination was done 3 months later. We used a digital dermoscopy (DD04, Dermotrichos, Brescia, Italy) equipped with software for digital image analysis.

Images were stored without compression in bitmap format and were independently evaluated by two blinded (neither one had information about the treatment or whether the nevi had been irradiated or protected during exposures) investigators (A. M. M. and P. G. C-P.) when all patients had completed the experimental protocol. The investigators assessed the maximal diameter and the main dermoscopic