

## Subclinical speckled perifollicular melanosis of the scalp

European Journal of Dermatology. Volume 12, Number 6, 565-8, November - December 2002, Thérapie

### Summary

**Author(s)** : Ludivine PETIT, Claudine, PIÉRARD-FRANCHIMONT, Didier SAINT LÉGER, Geneviève LOUSSOUARN, Gérald E. PIÉRARD, Department of Dermatopathology, University Medical Center Sart Tilman and Sauvenière, B-4000 Liège, Belgium..

**Summary** : Based on the clinical presentation of some skin pigmentation disorders it is thought that a bicompartamental functional system exists in the epidermal melanocyte population. It corresponds to the perifollicular and interfollicular compartments, respectively. The present study was undertaken looking for the presence of such a system on scalp unaffected by pigmentary disorders. The scalps of 100 men with incipient to severe androgenic alopecia were examined using a videocamera equipped with an internal ultraviolet light-emitting unit. The face, trunk and limbs were similarly examined in 45 of these adults and in 13 children of both sexes. In 92 men, a subclinical hypermelanosis was found as a speckled pattern centered on every single follicle. With increasing baldness severity, another epidermal hyperpigmentation pattern involving the interfollicular area was superimposed to the perifollicular pattern. These stereotyped patterns of subclinical melanoderma were also disclosed on the face of adults, but not in children. In addition, the spotty perifollicular pattern was discrete or not apparent on the other parts of the body. It is concluded that the perifollicular subclinical melanotic pattern is a regional characteristic of cephalic skin, perhaps related to the local production of melanocortins, particularly alpha-MSH by the pilosebaceous unit.

**Keywords** : ultraviolet light, alopecia, photoageing, hair, melanin.

### Pictures

#### ARTICLE

Ageing and chronic exposure to ultraviolet radiations (UVR) provide a series of signals to the epidermal melanocytic unit [1]. Focal melanotic hypermelanosis ensues where individual melanocytes tend to aggregate and are stimulated to produce more melanin. These melanins are a collection of molecules showing different degrees of polymerization, oxidation and sulfur content [2, 3]. Melanosome transfer from melanocytes to surrounding keratinocytes is also enhanced under the same stimulation. In contrast, chronic UVR exposure is also responsible for both replicative and stress-induced premature senescence (SIPS) [4]. Melanocyte apoptosis and decreased epidermal melanization ensues focally. As a result, photoageing is often characterized by a mottled appearance due to an uneven distribution and activity in epidermal melanocytes. The clinical aspect depends upon the individual melanin phenotype, age, cumulative UVR exposure and body site [5-7].

Several clinical and experimental conditions have already highlighted the existence of a bicompartamental system in the epidermal melanocyte population. They indeed correspond to the perifollicular and the interfollicular compartments which are relatively independent although exchanges may occur between them [8]. Guttate hypomelanosis and repigmenting vitiligo are the readily visible examples of the perifollicular compartment. Early subclinical changes in the mosaic pattern of epidermal melanization can be disclosed under ultraviolet light examination [9-16]. This is particularly prominent in light skinned individuals with a pheomelanin-enriched phenotype [14]. The increased contrast between the faint or almost invisible hyperpigmentation and the surrounding skin is the result of the decreased reflection of ultraviolet light by collagen because of its absorption by the epidermal melanin.

The aim of the present study was to document the subclinical to faint melanotic hypermelanosis of the scalp using video recording under ultraviolet light, and to compare the pigmentation pattern on other body sites in adults and children.

## Subjects and method

A total of 100 men aged from 28 to 56 years presenting with androgenic alopecia were examined during winter when they had not been exposed recently to intense sunshine. They had freckles on the dorsal aspect of the forearm, light brown or auburn hair, and they exhibited phototype II or III skin reactivity. A video camera equipped with an internal ultraviolet-emitting unit (Visioscan<sup>®</sup>, C + K Electronics, Cologne, Germany) was used as previously described [14-16] to study the

pattern of pigmentation on the vertex. The target area was shaved or not according to the possibility of viewing the skin surface after combing hair. The camera was closely applied to the skin surface and the aspect of a 1 x 0.8 cm scalp area was recorded. The same areas were also examined by white light epiluminescence using a Heine dermatoscope.

The face, trunk and limbs were similarly examined in 45 of these adults and in 13 children of both sexes.

## Results

At the conventional clinical examination the skin looked uniformly pigmented at the evaluation sites. White light epiluminescence revealed in 58 men light fawn spots that remained discrete on the scalp and dorsal forearms.

Ultraviolet light examination revealed aspects undisclosed under white light. The skin of the vertex appeared uniformly and fairly pigmented in 8 men. All were younger than 32 years and had light brown hair. An infraclinical mottled melanoderma was disclosed in the 92 other men. Two main patterns were predominant and clearly related to the hair density. An evenly distributed perifollicular speckled pattern corresponded to sharply demarcated round and dark tiny spots ([Fig. 1](#)). Every single follicle was marked by these pigmented dots.

With decreasing hair density related to androgenic alopecia, an accretive globular melanotic pattern was evidenced. Large irregularly shaped macules with ill defined borders merged and covered the perifollicular dots and part of the interfollicular area ([Fig. 2](#)). Most of the infundibula without hair were studded with a dark horny impaction rimmed by a lighter well demarcated exophytic ring ([Fig. 3](#)).

Facial skin of adults showed aspects similar to those found on the scalp ([Fig. 4](#)). By contrast, the perifollicular spotty pattern was much less prominent or even not disclosed on the trunk and limbs of the same subjects ([Fig. 5](#)). Nor was it found on the scalp, face and other body parts in children.

## Discussion

Objective analyses of colours and chromophore densities in the skin are widely used to measure physiological variations and to monitor treatment modalities of various pigmentary disorders [17]. Appropriate ultraviolet light illumination of the skin excites dermal fluorophores, particularly collagen [18, 19]. It is acknowledged that fluorescence intensity is reduced by intraepidermal melanin [11-16]. In the present study, a previously described device [14-16] was used to assess melanotic hypermelanosis which remained infraclinical or faint when examined under visible light and by dermoscopy. In the present cases, phaeomelanin might be present because freckles were seen on the forearms.

Multiple diverse and redundant molecular changes characterize senescence and photodamage. Only a fraction of the very complex pathways of human pigmentation is presently understood, and much is left to be discovered. After exposure to solar radiation, keratinocytes and melanocytes synthesize and release some melanocortins including alpha-MSH [20-22]. This compound is also a component specifically produced and released by the pilosebaceous unit in rodents [23-26]. In man, alpha-MSH is particularly present in the outer root sheath of anagen hair follicles and at the pilosebaceous orifice [27, 28]. Melanocytes from different skin types show dissimilar rates of proliferation and levels and types of melanin accumulation in response to alpha-MSH [29-31]. The present panelists did not have the red hair of individuals whose alpha-MSH receptor MC1R may induce a less potent signaling cascade or even a complete unresponsiveness to alpha-MSH [29]. Beyond increased melanogenesis, alpha-MSH formed and released in the pilosebaceous unit may stimulate sebum secretion [23, 24, 26] and may assist in coping with the local oxidative stress following UVR exposure [32].

We had previously described three main patterns of phaeomelanin-enriched melanotic hypermelanosis on the face, namely the spotty perifollicular type, the accretive globular type and the elongated type on the sunny side of wrinkles [14]. The first two types are now similarly described on the scalp. The spotty perifollicular pattern seemed to represent the primary intrinsic melanocytic activation when hair density is still almost normal. The globular pattern was only seen when alopecia was present. It corresponded to an accretive process by extension and merging of the peri-follicular spots to the interfollicular area.

The present observations suggest that hair, even in normal density, does not fully protect skin against UVR. The reduction in hair fullness is accompanied by a dramatic failure in the hair shielding effect on the interfollicular epidermis. This is probably the first preliminary step of scalp actinodermatosis before increasing the sun-induced neoplastic risk [13, 15]. Whether or not hair shedding is further increased by UVR effects is not yet elucidated and requires other methods of investigation [33]. It could be hypothesized that, similarly to the epidermis [34], the speckled perifollicular melanodermal may act as a photoprotection for the follicular stem cells. It remains a major challenge to decipher which epidermal melanization pattern results from alopecia and which one may cause alopecia through the SIPS mechanism.

Article accepted on 5/8/02

## REFERENCES

1. Haddad MM, Xu W, Medrano EE. Aging in epidermal melanocytes: cell cycle genes and melanins. *J Invest Dermatol Symp Proc* 1998; 3: 36-40.
2. Thody AJ, Higgins EM, Burchill SA. Epidermal eumelanin and phaeomelanin concentrations in different skin types and in response to PUVA. *Br J Dermatol* 1990; 122: 288-9.

3. Prota G. Melanins, melanogenesis and skin photoprotection. *Eur J Cancer* 1994; 30A: 553-4.
4. Toussaint O, Medrano EE, Von Zglinicki T. Cellular and molecular mechanisms of stress-induced premature senescence (SIPS) of human diploid fibroblasts and melanocytes. *Exp Gerontol* 2000; 35: 927-45.
5. Ortonne JP. Pigmentary changes of the ageing skin. *Br J Dermatol* 1990; 122 (suppl. 35): 21-8.
6. Piérard GE, Piérard-Franchimont C, Laso Dosal F, Ben Mosbah T, Arrese Estrada J, Rurangirwa A, Dowlati A, Vardar M. Pigmentary changes in skin senescence. *J Appl Cosmetol* 1991; 9: 67-73.
7. Nikkels A, Ben Mosbah T, Piérard-Franchimont C, de la Brassinne M, Piérard GE. Comparative morphometry study of eruptive PUVA-induced and chronic sun-induced lentigines of the skin. *Anal Quant Cytol Histol* 1991; 13: 23-6.
8. Ortonne JP, Prota G. Hair melanins and hair color: ultrastructural and biochemical aspects. *J Invest Dermatol* 1993; 101: S2-289.
9. Kikuchi I, Inoue S, Idermori M, Uchimura H. Reflection ultraviolet photography as surface photography of the skin. *J Dermatol* 1983; 10: 551-5.
10. Arai S. Analysis of pigmentation of human skin (UV-light images). In: Wilhelm KP, Elsner P, Berardesca E, Maibach HI, eds. *Bioengineering of the skin: skin surface imaging and analysis*. Boca Raton, CRC Press, 1997: 85-94.
11. Fulton JE. Utilizing the ultraviolet (UV detect) camera to enhance the appearance of photodamage and other skin conditions. *Dermatol Surg* 1997; 23: 163-9.
12. Kollias N, Gillies R, Cohen-Goihman C, Phillips SB, Muccini JA, Stiller MJ, Drake LA. Fluorescence photography in the evaluation of hyperpigmentation in photodamaged skin. *J Am Acad Dermatol* 1997; 36: 226-30.
13. Piérard-Franchimont C, Piérard, GE. Héliodermie hétérochrome et risque de cancers cutanés. *Rev Med Liege* 1993; 53: 355-6.
14. Hermanns JF, Petit L, Piérard-Franchimont C, Cauwenbergh G, Piérard GE. Unraveling the patterns of subclinical pheomelanin-enriched facial hyperpigmentation: effect of depigmenting agents. *Dermatology* 2000; 202: 118-22.
15. Hermanns JF, Henry F, Piérard-Franchimont C, Piérard GE. Quantification analytique du vieillissement du système mélanocytaire. Implication dans la détermination objective du risque de cancers cutanés. *Année Gerontol* 2001; 15: 233-9.
16. Petit L, Smitz S, Uhoda I, Piérard-Franchimont C, Piérard GE. Regional variability in mottled photo-induced melanoderma in the elderly. *Exp Gerontol* (in press).
17. Piérard GE. EEMCO guidance for the assessment of skin colour. *J Eur Acad Dermatol Venereol* 1998; 10: 1-11.
18. Leffell DJ, Stetzel ML, Milstone LM, Deckelbaum LI. *In vivo* fluorescence of human skin: a potential marker of photoaging. *Arch Dermatol* 1999; 124: 1514-8.
19. Takema Y, Yorimoto Y, Ohsu H, Osanai O, Kawai M. Age-related discontinuous changes in the *in vivo* fluorescence of human facial skin. *J Dermatol Sci* 1997; 15: 55-8.
20. Chakraborty AK, Funasaka Y, Slominski A, Ermka G, Hwang J, Pawelek JM, Ichibashi M. Production and release of proopiomelanocortin (POMC) derived peptides by human melanocytes and keratinocytes in culture: regulation by ultraviolet B. *Biochim Biophys Acta* 1996; 1313: 130-8.
21. Böhm M, Luger TA. The role of melanocortin in skin homeostasis. *Horm Res* 2000; 54: 287-93.
22. Suzuki I, Kato T, Motokawa T, Tomita Y, Nakamura E, Katagiri T. Increase of pro-opiomelanocortin mRNA prior to tyrosinase, tyrosinase-related protein 1, dopachrome tautomerase, Pmel-17/gp100, and P-protein mRNA in human skin after ultraviolet B irradiation. *J Invest Dermatol* 2002; 118: 73-8.
23. Thody AJ, Shuster S. Control and function of sebaceous glands. *Physiol Rev* 1989; 69: 383-416.
24. Böhm M, Luger TA. The liposebaceous unit is part of the skin immune system. *Dermatology* 1998; 196: 75-9.
25. Paus R, Botchkarev VA, Botchkareva NV, Mecklenburg L, Luger T, Slominski A. The skin POMC system (SPS). Leads and lessons from the hair follicle. *Ann NY Acad Sci* 1999; 885: 350-63.
26. Slominski A, Wortsman J, Luger T, Paus R, Solomon S. Corticotropin releasing hormone and proopiomelanocortin involvement in the cutaneous response to stress. *Physiol Rev* 2000; 80: 979-1020.
27. Slominski A, Wortsman J, Mazurkiewicz JE, Matsuoka L, Dietrich J, Lawrence K, Gorbani A, Paus R. Detection of proopiomelanocortin-derived antigens in normal and pathologic human skin. *J Lab Clin Med* 1993; 122: 658-66.
28. Liu PY, Lontz W, Bondesson L, Johansson O. The possible role of alpha, beta and gamma3 melanocyte stimulating

hormone containing keratinocytes in the initiation of vitiligo vulgaris. *Eur J Dermatol* 1995; 5: 625-30.

- 29.** Hunt G, Todd C, Thody AJ. Unresponsiveness of human epidermal melanocytes to melanocyte-stimulating hormone and its association with red hair. *Moll Cell Endocrinol* 1996; 116: 131-6.
- 30.** Ha T, Rees JL. Melanocortin 1 receptor: what's red got to do with it? *J Am Acad Dermatol* 2001; 45: 961-4.
- 31.** Tsatmali M, Ancans J, Thody AJ. Melanocyte function and its control by melanocortin peptides. *J Histochem Cytochem* 2002; 50: 125-34.
- 32.** Haycock JW, Rowe SJ, Cartledge S, Wyatt A, Ghenem G, Morandini R, Rennie IG, Mac Neil S. Alpha-melanocyte-stimulating hormone reduces impact of proinflammatory cytokine and peroxide-generated oxidative stress on keratinocyte and melanoma cell lines. *J Biol Chem* 2000; 275: 15629-36.
- 33.** Piérard-Franchimont C, Uhoda I, Saint Léger D, Piérard GE. Androgenic alopecia and stress-induced premature senescence by cumulative ultraviolet light exposure. *Exog Dermatol* (in press).
- 34.** Bykov VJ, Marcusson JA, Hemminki K. Protective effects of tanning on cutaneous DNA damage *in situ*. *Dermatology* 2001; 202: 22-6.

[Copyright © 2007 John Libbey Eurotext - Tous droits réservés](#)