



# ETHNIC DERMATOLOGY

## Principles and Practice

Edited by Ophelia E. Dadzie, Antoine Petit and Andrew F. Alexis





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Principles and Practice



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EDITED BY

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**Nina G. Jablonski**

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# Foreword

*Ethnic Dermatology* is being published during a renaissance in the study of human variation, when studies of the significance of variation in human skin have gained new importance and legitimacy. For most of the history of dermatology, human skin was “White,” northern European skin. White skin was the normal human condition, from which all others deviated. Dermatology rose as an independent discipline during the late 18th and early 19th centuries, at the same time as naturalists and anthropologists were describing human races and philosophers were arguing for hierarchical ranking of those races. People with moderately or darkly pigmented skin were viewed by many at that time as lesser beings and the normal properties of their skin were seen as pathological by definition. The need for books like *Ethnic Dermatology* today arose from the misconceptions about the nature of normal variation in human skin that developed in those benighted times. As institutional and governmentally sanctioned racism declined worldwide in the late 20th century, knowledge and appreciation of the importance of variation in the properties of human skin increased. This promising trend was retarded, ironically, by the power of popular social movements which advocated equality among races and sexes in all matters and which viewed the study of human variation as inherently divisive and socially destructive. Dermatology, more than other medical specialties, is subject to the vicissitudes of social and political movements because it deals with the organ that is humankind’s most visible interface with the physical and social environment.

Dermatologists working to describe and study “ethnic” skin or skin of color and its diseases face many practical problems, one of the most serious being an impoverished vocabulary with which to describe variation. The glossary of descriptive medical terms for skin pigmentation is bereft of accurate and precise words to describe hues, shades, and tints of skin color. “Darkly,” “richly,” and “moderately” pigmented are commonly used in medicine

and are socially acceptable, but are miserably imprecise and are less exact than the rich colloquialisms they seek to replace. The Fitzpatrick scale of skin phototypes, which has dominated dermatology for nearly a half century, is also deficient because it is based on subjective assessment of one phenotypic trait, tanning ability. While this classification method can broadly inform us of an individual’s sun sensitivity and likelihood of developing skin cancer, tanning ability is not determined by a single gene or a single unique set of genes nor is it necessarily informative of other immunological or physiological properties of skin that are relevant to disease susceptibility. Genetic and genomic studies have revealed that pigmentation phenotypes have evolved multiple times as modern humans have dispersed out of and back into the tropics. We now know that lightly pigmented (“White”) skin seen in natives of Berlin and Beijing, for example, was the product of two independent genetic mutation events leading to the evolution of two depigmented human lineages that came to inhabit northwestern Europe and northeastern Asia. The classification of these two individuals as Fitzpatrick type II is of limited usefulness. Similarly, natives of Brasilia, Cape Town, and Naples who are classified as Fitzpatrick type IV are likely to have three different sets of pigmentation gene polymorphisms contributing to their enhanced tanning abilities. The point here is that we are in need of new ways of defining and describing the normal range of variation present in healthy human skin because the current vocabulary and scales for describing variation are inadequate and outdated. The genetic bases for the complex mixtures of melanins and keratins found in skin, and for the interaction of these with various immunoglobulin isotypes, are now beginning to be understood and their significance for health and disease appreciated. As this body of information grows, and our understanding of individual responses to environmental insults develops apace, dermatology will truly come of age.

The synthesis of knowledge on skin and skin diseases presented in *Ethnic Dermatology* is inspiring and provides the foundation for a modern and comprehensive science of dermatology that is based on an inclusive concept of “normal human skin,” including its aging and scarring characteristics and susceptibility to disease. Specialists in ethnic dermatology will find this book to be an excellent guide, but also a call to action. This field requires much

more research and many more avid clinicians and scientists interested in carrying out that research. This book is your starting point.

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# Preface

In the face of life's many challenges we have to ask ourselves why do we do what we do? This simple question is one we have had to reflect upon prior to and during the writing and editing of this textbook. For us the answer to this question is simple: a need to make a difference and/or impact in our community, combined with a genuine interest and passion for the subject matter.

Broadly speaking, mainstream dermatology in most western countries continues to have a eurocentric standard and viewpoint, despite an increasing interest worldwide in the issue of ethnic dermatology. This has primarily been driven by the changing demographics of most western countries, coupled with the emerging economies of many African and Asian countries. While several textbooks now exist on this topic, most originate from the USA, giving an American perspective to this issue.

The purpose of *Ethnic Dermatology: Principles and Practice* is to provide a comprehensive, yet practical

perspective of the subject matter. Both medical and cosmetic dermatology are extensively covered in this textbook. Ample use of good-quality clinical images supplements the text, which are all clinically relevant. Furthermore, there is an excellent foreword written by Professor Nina Jablonski discussing the issue of terminologies pertaining to ethnic dermatology.

This textbook will suit clinical dermatologists, primary care physicians, physicians from other specialties, and specialist nurses. It is our hope that all will find this book of direct relevance to their daily clinical practice. Long-term, we also hope that textbooks such as this will encourage acceptance and incorporation of ethnic dermatology into mainstream dermatology forums in many western countries.

*Ophelia E. Dadzie*  
*Antoine Petit*  
*Andrew F. Alexis*



# List of Abbreviations

<b>AD</b>	atopic dermatitis	<b>EASI</b>	Eczema Area and Severity Index
<b>AJCC</b>	American Joint Committee on Cancer	<b>EBV</b>	Epstein-Barr virus
<b>AKN</b>	acne keloidalis nuchae	<b>ECM</b>	extracellular matrix
<b>ALM</b>	acral lentiginous melanoma	<b>EGFR</b>	epidermal growth factor receptor
<b>AP</b>	actinic prurigo	<b>ENT</b>	ear, nose, and throat
<b>ARV</b>	antiretroviral drugs	<b>EV</b>	epidermodysplasia verruciformis
<b>ART</b>	antiretroviral therapy	<b>EVCH</b>	eruptive vellus hair cysts
<b>ATL</b>	adult T-cell lymphoma	<b>FACE</b>	facial Afro-Caribbean childhood eruption
<b>ATLL</b>	adult T-cell lymphoma/leukemia	<b>FAMMM</b>	familial atypical multiple mole melanoma syndrome
<b>AZT</b>	zidovudine	<b>FBGCR</b>	foreign body giant cell reaction
<b>BCC</b>	basal cell carcinoma	<b>FPHL</b>	female pattern hair loss
<b>BMZ</b>	basement membrane zone	<b>FD</b>	folliculitis decalvans
<b>CAD</b>	chronic actinic dermatitis	<b>FDE</b>	fixed drug eruptions
<b>CBPL</b>	cutaneous B-cell pseudolymphoma	<b>FFA</b>	frontal fibrosing alopecia
<b>CCCA</b>	central centrifugal cicatricial alopecia	<b>FHP</b>	facial hyperpigmentation
<b>CCLE</b>	chronic cutaneous lupus erythematosus	<b>FKN</b>	folliculitis keloidalis nuchae
<b>CGPD</b>	childhood granulomatous periorificial dermatitis	<b>FSP/FST</b>	Fitzpatrick skin phototype/type
<b>CPK</b>	creatine phosphokinase	<b>FUE</b>	follicular unit extraction
<b>CRP</b>	confluent and reticulate papillomatosis	<b>FVC</b>	forced vital capacity
<b>cSLE</b>	childhood-onset systemic lupus erythematosus	<b>G6PD</b>	glucose-6-phosphate dehydrogenase
<b>CTCL</b>	cutaneous T-cell lymphoma	<b>GA</b>	glycolic acid
<b>CTGF</b>	connective tissue growth factor	<b>GRK</b>	G-protein-coupled receptor kinase
<b>CTPL</b>	cutaneous T-cell pseudolymphoma	<b>GVHD</b>	graft-versus-host disease
<b>DCS</b>	dissecting cellulitis of the scalp	<b>GWAS</b>	genome-wide association studies
<b>DEJ</b>	dermo-epidermal junction	<b>HAART</b>	highly active antiretroviral therapy
<b>DFSP</b>	dermatofibrosarcoma protuberans	<b>HHV</b>	human herpes virus
<b>DLCO</b>	diffusing capacity of the lung for carbon monoxide	<b>HIFU</b>	high-intensity focused ultrasound
<b>DMSO</b>	dimethylsulfoxide	<b>HIV</b>	human immunodeficiency virus
<b>DOC</b>	disorders of cornification	<b>HLA</b>	human leukocyte antigen
<b>DPN</b>	dermatosis papulosa nigra	<b>HPV</b>	human papilloma virus
<b>DRESS</b>	drug reactions (or rashes) with eosinophilia and systemic symptoms	<b>HS</b>	hidradenitis suppurativa
<b>DRI</b>	disseminate and recurrent infundibulofolliculitis	<b>HSE</b>	hydrocortisone, silicon and vitamin E lotion
		<b>HSV</b>	herpes simplex virus
		<b>HT</b>	hair transplantation
		<b>HTLV</b>	human T-lymphotropic virus
		<b>HTS</b>	hypertrophic scars

<b>IGA</b>	Investigator Global Assessment	<b>PDGFR</b>	platelet-derived growth factor receptor
<b>IGH</b>	idiopathic guttate hypomelanosis	<b>PDIR</b>	premature desquamation of the inner root sheath
<b>IH</b>	infantile hemangioma	<b>PDL</b>	pulsed dye laser
<b>IK</b>	inverse keratoderma	<b>PET</b>	positron emission tomography
<b>IP</b>	inflammatory pigmentations	<b>PFB</b>	pseudofolliculitis barbae
<b>IPL</b>	intense pulsed light	<b>PHACES</b>	Posterior fossa abnormalities, Hemangioma-large, segmental, Arterial lesions, Cardiac/coarctation findings, Eye abnormalities, and Sternal abnormalities
<b>IRS</b>	immune reconstitution syndrome	<b>PIH</b>	postinflammatory hyperpigmentation
<b>ISD</b>	infantile seborrheic dermatitis	<b>PMLE</b>	polymorphous light eruption
<b>IUS</b>	intense ultrasound	<b>PPAR<math>\gamma</math></b>	peroxisome proliferator-activated receptor gamma
<b>IVIG</b>	intravenous immunoglobulin	<b>PPD</b>	paraphenylenediamine
<b>KP</b>	keratosis pilaris	<b>PPE</b>	papular pruritic eruption
<b>KPC</b>	keratosis punctata of the palmar creases	<b>PPK</b>	palmoplantar keratoderma
<b>KS</b>	Kaposi's sarcoma; keloid scars	<b>PR</b>	pityriasis rosea
<b>LE</b>	lupus erythematosus	<b>PUVA</b>	psoralen plus ultraviolet light-A
<b>LED</b>	light-emitting diode	<b>PUVAsol</b>	psoralen plus sunlight
<b>LN</b>	lichen nitidus	<b>PV</b>	pityriasis versicolor
<b>LP</b>	lichen planus	<b>RegisCAR</b>	Registry of severe cutaneous adverse reactions to drugs and collection of biological samples
<b>LPP</b>	lichen planopilaris	<b>RF</b>	radiofrequency
<b>MAI</b>	<i>Mycobacterium avium-intracellulare</i>	<b>RLX</b>	relaxin
<b>MAP</b>	magnesium-L-ascorbyl-2 phosphate	<b>RSTL</b>	relaxed skin-tension line
<b>MASI</b>	Melasma Area and Severity Index	<b>SA</b>	<i>Staphylococcus aureus</i>
<b>MB</b>	multibacillary	<b>SCC</b>	squamous cell carcinoma
<b>MED</b>	minimal erythema dose	<b>SCLE</b>	subacute cutaneous lupus erythematosus
<b>MF</b>	mycosis fungoides	<b>SCORAD</b>	Scoring Atopic Dermatitis Scale
<b>MFU</b>	multifollicular unit	<b>SD</b>	seborrheic dermatitis
<b>MK</b>	marginal keratoderma	<b>SJS</b>	Stevens-Johnson's syndrome
<b>MKTP</b>	melanocytes-keratinocytes transplantation	<b>SLE</b>	systemic lupus erythematosus
<b>MPHL</b>	male pattern hair loss	<b>SLNB</b>	sentinel lymph node biopsy
<b>MSH</b>	melanocyte stimulating hormone	<b>SM</b>	subungual melanoma
<b>MTB</b>	<i>Mycobacterium tuberculosis</i>	<b>SMAS</b>	superficial musculoaponeurotic system
<b>MTZ</b>	microthermal zone	<b>SNP</b>	single-nucleotide polymorphism
<b>NB-UVB</b>	narrowband-UVB	<b>SPF</b>	sun protection factor
<b>NLE</b>	neonatal lupus erythematosus	<b>SS</b>	Sézary's syndrome
<b>NNRTI</b>	non-nucleoside reverse transcriptase inhibitor	<b>SU</b>	solar urticaria
<b>NRTI</b>	nucleoside reverse transcriptase inhibitor	<b>SV</b>	segmental vitiligo
<b>NSV</b>	nonsegmental vitiligo	<b>TA</b>	traction alopecia
<b>OTC</b>	over-the-counter	<b>TAC</b>	triamcinolone acetate
<b>PA</b>	pityriasis alba	<b>TC</b>	tinea capitis
<b>PAR-2</b>	protease-activated receptor 2	<b>TCA</b>	trichloroacetic acid
<b>PASI</b>	psoriasis area and severity index	<b>TEN</b>	toxic epidermal necrolysis
<b>PB</b>	paucibacillary		
<b>PCA</b>	primary cutaneous amyloidosis; principal component analysis		
<b>PCBCL</b>	primary cutaneous B-cell lymphoma		
<b>PCFCL</b>	primary cutaneous follicle centre lymphoma		
<b>PCMZL</b>	primary cutaneous marginal zone lymphoma		
<b>PDGF</b>	platelet-derived growth factor		



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<b>TEWL</b>	transepidermal water loss	<b>UVA</b>	ultraviolet light-A
<b>TIS</b>	Three-Item Severity Scale	<b>UVB</b>	ultraviolet light-B
<b>TGF</b>	transforming growth factor	<b>UVR</b>	ultraviolet radiation
<b>TLR</b>	toll-like receptors	<b>VDRL</b>	Venereal Disease Reference Laboratory
<b>TNM</b>	tumor-node-metastasis	<b>VETF</b>	Vitiligo European Task Force
<b>TNPM</b>	transient neonatal pustular melanosis	<b>VZV</b>	varicella zoster virus
<b>TPMT</b>	thiopurine S-methyltransferase		



# Defining Ethnic Dermatology: Challenges, Limitations, and Merits

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Ethnic dermatology is a term used to describe an aspect of dermatology pertaining to individuals of diverse racial and ethnic backgrounds, who have richly pigmented skin and who share broadly similar cutaneous characteristics, notably the risk of scarring and dyspigmentation in response to cutaneous trauma. The term is analogous to skin of color, which is commonly used in North America. Defining the ethnic dermatology/skin of color cohort is challenging. However, broadly speaking and in this textbook, this cohort equates to individuals with Fitzpatrick skin phototypes (FSP) IV–VI and/or those of African, Asian, Middle Eastern, and/or Hispanic ancestry [1–2].

Unfortunately the use of terminologies such as ethnic dermatology and/or skin of color is not without its critics [3–4]. This is because of the problems and limitations of defining individuals by race, ethnicity, and/or skin pigmentation (an inherent problem in any scientific endeavor, which Richard Dawkins refers to as “the tyranny of the discontinuous mind”) [5]. Essentially humans do not fit into neat racial or ethnic categories, but represent a continuum. Thus, at what point does someone become “black” or “white”? Since evidence indicates that modern humans originate from Africa [6], are we not all of African ancestry? Furthermore, in advocating separating and defining specific groups based on racial, ethnic and/or skin pigmentation, are we contributing to a divisive society? After all, at a genetic level, humans share more similarities than differences [6]. In addition, the use of FSP has specific limitations when applied to pigmented skin (see Box 1.1 for discussion on this issue).

There is also a risk that terms such as ethnic dermatology will justify studies that use skin color and/or ethnicity to validate a biological construction of race that is actually rooted in socio-historical processes [7], e.g., “scientific studies” that supported the notion that people of African race are less prone to contact sensitization and hence better able to handle certain noxious substances [8].

All the above represent challenging questions and difficulties that we have had to navigate before embarking on this ethnic dermatology/skin of color “journey.” In response to these challenges we first have to consider the problems faced by practicing dermatologists.

First, epidemiological studies and data obtained from hospital and/or private practices indicate that there are differences in the observed dermatoses in different ethnic/racial groups [9–10]. For instance, hair and scalp disorders are one of the major concerns in individuals with Afro-textured hair. Cultural factors also impact the range of dermatoses observed (e.g., the misuse of skin lightening agents in certain racial and/or ethnic groups and the occurrence of prayer nodules in Muslims [Fig. 1.1]). Thus, as practicing dermatologists, we need to be aware of these observed differences and the implications for managing our patients. Second, studies have highlighted deficiencies in dermatological educational resources and the training of dermatologists with regard to the field of skin of color/ethnic dermatology [11–12]. Finally, the demographics of most western countries is changing. This means that

### Box 1.1 Fitzpatrick skin phototype

The Fitzpatrick skin phototype (FSP) classification system (see also Box 1.2) [15] is used routinely by dermatologists to categorize and classify different skin types. It was initially developed by Thomas Fitzpatrick in 1975 to classify persons with “white skin” in order to select the correct initial dose of UVA for an upcoming large-scale oral PUVA photo-chemotherapy trial in the US in the mid-1970s. It was based primarily on a brief personal interview to evaluate individuals’ history of sunburn and tanning and not on phenotype (hair and eye color) [15]. The initial classification system placed all non-white/pigmented skin in one category, skin type V. Over time this classification system evolved and skin type V was divided into three sub-groups (IV, V, and VI) to encompass the diversity observed in those with pigmented skin. Furthermore, over time phenotype has had a greater impact on this classification system. It is the author’s opinion that often phenotype is the prime method used to categorize skin types, instead of proper evaluation of ultraviolet radiation response. This is one of the main limitations of FSP as a method of classifying individuals with pigmented skin. Furthermore, studies have shown a lack of a direct correlation between constitutive skin color and response to ultraviolet radiation. For instance, individuals originating from various Asian countries encompass a diverse group and skin color does not always predict their skin phototypes [16,17]. Another limitation of FSP is that it is based on self-reported erythema sensitivity and tanning ability, and hence it is not quantitative or reliable. Furthermore, it cannot be applied for *in vitro* conditions. For this reason, new classification systems have been developed, such as the colorimetric classification of constitutive pigmentation by individual typology angle [18,19] and the Roberts skin classification system [20] (Box 1.2). The former is of relevance in the research setting, while the latter is of practical relevance in predicting response to trauma, prior to procedural dermatology. There are four elements to the Roberts skin classification system, which should be evaluated based on a thorough history, examination, and evaluation of test site reaction.

most practicing dermatologists need to be competent in the diagnosis and management of cutaneous disorders in people of diverse racial and ethnic backgrounds. For example, in 1990 the United States census revealed that 76% of the population was white; 12% black; 9% Hispanic; 2.8% Asian/Pacific Islander; and 0.7% American Indian, Eskimo, and Aleut [6]. Projections for the US population in 2050 forecast a substantial decline in the white population to approximately 53%, with an increase in other racial groups (black 14%; Hispanic 25%; Asian 8%; American Indian, Eskimo, and Aleut

### Box 1.2 Roberts skin type classification system

#### Fitzpatrick (FZ) scale: measures skin phototype

- FZ<sub>1</sub> White skin. Always burns, never tans
- FZ<sub>2</sub> White skin. Always burns, minimal tan
- FZ<sub>3</sub> White skin. Burns minimally, tans moderately and gradually
- FZ<sub>4</sub> Light brown skin. Burns minimally, tans well
- FZ<sub>5</sub> Brown skin. Rarely burns, tans deeply
- FZ<sub>6</sub> Dark brown/black skin. Never burns, tans deeply

#### Roberts hyperpigmentation (H) scale: propensity for pigmentation

- H<sub>0</sub> Hypopigmentation
- H<sub>1</sub> Minimal and transient (<1 year) hyperpigmentation
- H<sub>2</sub> Minimal and permanent (>1 year) hyperpigmentation
- H<sub>3</sub> Moderate and transient (<1 year) hyperpigmentation
- H<sub>4</sub> Moderate and permanent (>1 year) hyperpigmentation
- H<sub>5</sub> Severe and transient (<1 year) hyperpigmentation
- H<sub>6</sub> Severe and permanent (>1 year) hyperpigmentation

#### Glogau (G) scale: describes photoaging

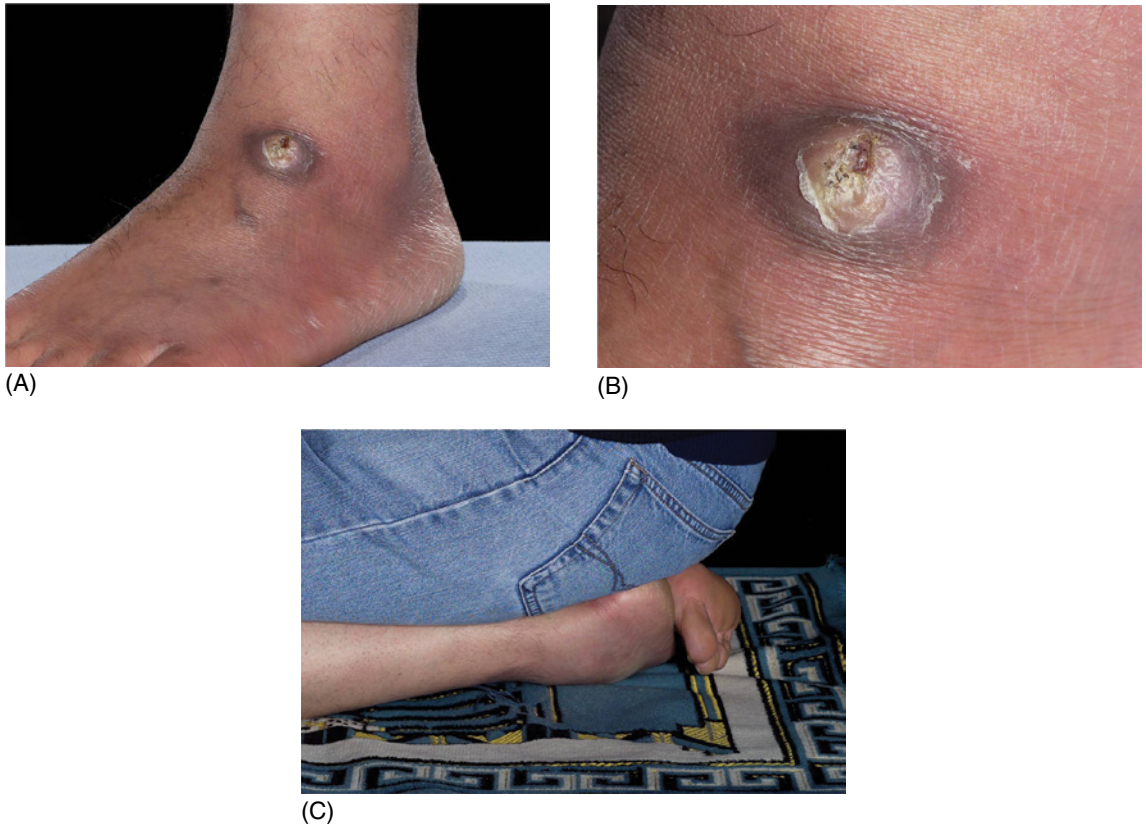
- G<sub>1</sub> No wrinkles, early photoaging
- G<sub>2</sub> Wrinkles in motion, early to moderate photoaging
- G<sub>3</sub> Wrinkles at rest, advanced photoaging
- G<sub>4</sub> Only wrinkles, severe photoaging

#### Roberts scarring (S) scale: describes scar morphology

- S<sub>0</sub> Atrophy
- S<sub>1</sub> None
- S<sub>2</sub> Macule
- S<sub>3</sub> Plaque within scar boundaries
- S<sub>4</sub> Keloid
- S<sub>5</sub> Keloidal nodule

approaching 1%) [6]. In the United Kingdom, the 2001 census demonstrated that ethnic minorities made up 7.9% of the population, an increase of 53% compared to the previous 1991 census [13].

Based on the above and despite the valid limitations and difficulties in defining ethnic dermatology, the use of this term is helpful, given that it enables interested parties (dermatologists, other physicians, nurses, scientists, and patients) to come together to help advance this aspect of dermatology [2]. In time it is likely that advances in genomics will increase our understanding of the role of genetic variation among human populations, thereby influencing our use of terminologies such as ethnic dermatology and skin of color [14].



**Figure 1.1** (A,B) A prayer nodule (talar callosity) located on the dorsal aspects of the left foot associated with the specific prayer stance undertaken by this devout Muslim (C).

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# Skin Semiology and Grading Scales

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## Introduction

Despite technological advances in diagnostics, the art of clinical medicine still lies in the recognition and interpretation of clinical signs and symptoms. In no field is this more apparent than dermatology. In particular, the dermatologist has acquired skills for the detection of the most representative lesions of any skin disease – the so-called “elementary lesions” – and a precise evaluation of their color, size, border, thickness, number, and topography, as well as the pruritus, pain or tenderness that may be associated with them. This analytic approach to clinical diagnosis is a complex cognitive process complementary to a global, more intuitive process; the latter probably represents the ground of daily dermatological practice and allows the non-specialist to recognize most skin lesions and diseases, provided they have already seen them before. However, the “global” approach may reach its limit in unusual diagnostic situations. Such a situation may be encountered, for example, in countries where a massive campaign for the detection of leprosy has been conducted by general practitioners, nurses or other field agents who had received basic minimal instruction for the detection of leprosy lesions. As the prevalence of this disease progressively decreased due to the efficacy of these campaigns, so did the teams’ diagnostic capabilities, due to a lack of clinical experience and awareness of the differential diagnosis

when confronted with a larger variety of skin lesions [1]. This example also reminds us that, whatever the diagnostic approach (global or analytic), the negative and positive predictive values of any clinical sign or group of signs vary with the prevalence of the disease being sought.

The conception of the analytic approach in clinical dermatology mainly occurred during the 19th and 20th centuries in Europe. Thus almost all the classic texts describe skin diseases and their elementary lesions as they would have presented in fair-skinned patients. This has led to a significant lack of precise description of physical signs in ethnic skin and therefore a poor understanding of the clinical presentation of common skin diseases in ethnic populations. In a similar way, the grading scales for the severity of skin disease have been built almost exclusively on fair-skinned individuals, making it difficult to accurately diagnose and assess the severity of cutaneous disease in people with heavily pigmented skin [2]. However, most of these practical problems can be overcome through additional knowledge and clinical training; in this way some diseases may even be easier to recognize in patients with richly pigmented skin.

Whatever the patient’s skin color, there are few systematic studies of dermatological semiology, and most published data stems from the clinical knowledge acquired by individual physicians. Regardless of their individual clinical experience, this data is still largely subjective.

This chapter will first focus on the particularities in semiology that are linked to pigmentation and color. Other differences in the clinical presentation of skin lesions among patients of various ethnic backgrounds will be discussed in the second part, except for hair semiology, which will be discussed in subsequent chapters. We will finally consider the problem of grading scales and scores that are commonly used in dermatology and the challenges that arise when these are applied to ethnic skin.

## Pigmentation and color

This is the most crucial and undeniable source of differences in skin semiology among ethnic populations. However, it is important to first consider the words we use for skin pigmentation and color. If assessed in a scientific manner, using a three-axis scale such as “LAB,” it is very likely that the way we characterize the various skin phototypes is mostly determined by differences in the “L” axis, which describes lightness or darkness, whereas the colored hues, which may vary between people of the same phototype, and which have mainly been used to describe classical skin semiology in fair-skinned patients, are better described by the “A” and “B” axis (green to magenta and blue to yellow, respectively). In addition, the lightness or darkness of the skin is predominantly, but not always, linked to its melanin content (a darker tone could result, for example, from keratin oxidation in the horny layer or erythrocyte extravasation in the dermis). Thus, since a change in darkness is not always related to a variation in melanin content, some have advocated the use of the terms “hyperchromia” and “hypochromia” and reserve their counterparts “hyperpigmentation” and “hypopigmentation” until it can be confirmed that melanin is specifically involved [3]. Finally, the word “color” in the expression “skin of color”, as well as the Greek root “khroma” (meaning “color”) in the words hyper- and hypochromia, refers to lightness/darkness rather than color per se.

In patients with heavily pigmented skin, most dermatoses induce changes in lightness/darkness which may overwhelm other clinical manifestations, not only as a major source of patients’ concerns or even distress, but also because the intensity of the natural pigmentation may affect our perception of the color of the lesion. In particular, one of the key challenges encountered by dermatologists in the interpretation of skin semiology in the ethnic patient is the clinical presentation of erythema (and of jaundice,

especially in newborns and children). In some circumstances, it seems that richly pigmented skin simply does not allow for the observation of “redness,” resulting in an almost unchanged skin hue (Figs 2.1 and 2.2). In these situations, it is crucial to ask the patient for their own opinion about the color of the putative pathological areas, since they may be able to detect very subtle changes in



**Figure 2.1** Urticaria. The redness usually associated with the wheal in fair skin is absent, as well as the central pallor, which may be a manifestation of dermal edema. However, the widening of the follicular openings and the increased distance between them clearly demonstrate dermal edema.



**Figure 2.2** Varicose veins. The blue color of these dilated veins is masked by melanin.



**Figure 2.3** Scarler fever. These two girls are sisters; the girl on the right is experiencing postinflammatory desquamation predominant on the flexural area and shows hyperchromic lesions corresponding to the residual rash, while the girl on the left has been infected 2 weeks later; her whole skin is hyperchromic due to the rash but there is no apparent erythema. Courtesy of Dr Edouard Raynaud, Hôpital Victor Dupouy, Argenteuil, France.



**Figure 2.4** Lichen planus. Papules of lichen planus usually exhibit a purple hue on white skin and lead to hyperpigmentation. Here they present mainly with an atypical whitish or light-gray color, probably due to scales.



**Figure 2.5** Hyperchromia in lichenified atopic dermatitis.

their own skin color. Palpation may also give some additional diagnostic help, with elevated temperature over the affected skin a common feature with erythema (e.g., in a drug or viral rash or bacterial cellulitis); and some indirect help when showing infiltration, edema, oozing or other pathological signs (Fig. 2.3).

Furthermore, the nuances of redness, which usually are of some importance for the clinical recognition of diseases such as lichen planus (dark purple), pityriasis rosea (pink), psoriasis (bright red), or sarcoidosis (“apple jelly”) in fair-skinned patients, may look completely different in patients with richly pigmented skin. Such differences do not follow absolute rules; lesions of the same nature may present with various hues according to their stage and the

patient’s skin shade, resulting in confusing visual patterns of many skin diseases in the ethnic populations (Fig. 2.4). For convenience, we will here consider hyperchromic and hypochromic lesions separately, and then pigmentary patterns that appear physiological (and which therefore can hardly be designated “hyper-” or “hypochromic”).

### Hyperchromia

More commonly, cutaneous inflammation, which would have been characterized by redness in a fair-skinned patient, appears as a darker, brown to black area in the dark-skinned patient (Figs 2.5 and 2.6). This is often believed to be a consequence of either the nonspecific



**Figure 2.6** Extensive *Trichophyton rubrum* infection. The affected area appears mainly hyperchromic.



**Figure 2.7** Hyperpigmented patches of unknown origin showing histological changes of postlichenoid dermatitis with pigment incontinence.

melanocytic hyperactivity that occurs with most inflammatory states, or the pigmentary incontinence that occurs with some specific types of skin inflammation, when injury of the epidermal basal layer leads to the leak of melanosomes into the dermis and their phagocytosis by dermal macrophages (melanophages) (Fig. 2.7). However, one may also hypothesize that the erythema itself could appear as a darkening of the skin without any melanocyte hyperactivity or pigmentary incontinence. For this reason, the way a dermatologist looks at the skin of patients with richly pigmented skin has been compared to the way we consider skin semiology in black and white photographs (Figs 2.8, 2.9, 2.10, and 2.11). Thus, some hyperpigmented states that would have been diagnosed

as “postinflammatory hyperpigmentation” actually represent “per-inflammatory hyperpigmentation.” This is not without therapeutic consequences: for example, in our experience, many hyperchromic lesions on the face that would have been designated as “postinflammatory hyperpigmentation” actually respond quickly to acne therapeutics such as doxycycline (Fig. 2.12). Moreover, inflammatory or purpuric lesions of important medical significance may be considered wrongly as “pigmentary skin disorders,” with potentially serious consequences (Figs 2.13, 2.14, and 2.15).

### Hypochromia

In patients with heavily pigmented skin, hypochromia (paler skin) and achromia (white skin showing a complete loss of melanin) also indicate various skin conditions, many of which pigmentary changes would have been regarded as absent (or at least discreet and of little significance) in fair-skinned patients. However, in the ethnic patient, hypo- or achromic lesions seem far less frequent and are far more informative than hyperchromic lesions, often being associated with a much smaller list of differential diagnoses (Figs 2.16 and 2.17; Table 2.1). More detailed information on skin disease leading to hypochromia or achromia can be found in Chapters 12 and 13.

### Physiological patterns

An uneven distribution of melanin throughout the skin is not always of pathological significance. Indeed, there are many physiological variations of skin tone that seem more frequently noticed in dark-skinned people, even if they can also be encountered in fair skins. Occasionally, some of these changes may induce a real cosmetic concern for the patient, prompting them to seek dermatological advice. It is therefore important for physicians to be aware of these physiological patterns.

Remarkable patterns are well known under the name of Futcher’s or Voigt’s lines. Type A Futcher’s lines are the most frequent, being visible in up to 25% of patients with phototype V or lighter phototype VI. They appear as sharply delimited straight lines extending symmetrically on a vertical or slanting axis (depending on the position of the arm) at the anterior-external part of each upper arm, separating a lighter inner-anterior part from a darker posterior-external one (Fig. 2.18). Type B Futcher’s lines are less frequent; they can be seen on the



**Figure 2.8** Spontaneous keloids of the breast, showing annular pattern and centrifugal extension, are predominantly found in black women where they usually present as hyperpigmented lesions.



**Figure 2.9** Spontaneous keloids of the breast occasionally involve white women, where they present as erythematous lesions.



**Figure 2.10** Fig. 2.8 converted into black and white presents simply as a darker area.



**Figure 2.11** Fig. 2.9 converted into black and white presents simply as a darker area.



**Figure 2.12** The so-called “postinflammatory hyperpigmentation” (PIH), e.g., in acne, actually frequently consists of per-inflammatory hyperpigmentation.



**Figure 2.14** These apparently trivial hyperchromic macules actually showed histological features of Kaposi's sarcoma.



**Figure 2.13** External ear hyperchromic lesions in a young Algerian woman, phototype V. This corresponds to active lupus erythematosus, not to post-inflammatory sequellae.



**Figure 2.15** “Pigmented livedo” is the usual expression by which French dermatologists describe erythema ab igne. However, this livedo (which clinically showed a discrete infiltrate) was histologically proven to be periarteritis nodosa. On lighter skin, it would have appeared a violaceous color, not as “hyperpigmentation”.

inner part of the thighs, sometimes extending below the knee. In our experience, type B Futcher's lines sometimes appear as hypopigmented lines rather than a demarcation between surfaces of different tones (Fig. 2.19). A thin vertical line of hypopigmentation is frequently observed over the midline of the chest along

the sternum; these are referred to as “type C” Futcher's lines. They can be associated with other lines of the same kind, such as a curved line extending between the nipples, reminiscent of the Greek letter “psi.” Small hypopigmented circles may also be seen around the nipples. In our experience, the position and direction of the lines



**Figure 2.16** Progressive macular and confluent hypomelanosis in a 28-year-old man from Algeria, phototype V.



**Figure 2.17** Sarcoidosis on the neck. This picture illustrates the diagnostic value of hypopigmentation. Numerous discrete flesh-colored or hyperchromic papules correspond to beard folliculitis; only the two hypochromic lesions (above, right) represent sarcoidosis.

**Table 2.1** Main causes of hypochromia and achromia in heavily pigmented skin.

Disease	Achromia	Hypochromia	Achromia or hypochromia + hyperchromia	Notes
Vitiligo	x	x	x	
Futcher's/Voigt's lines		x		See "Physiological patterns"
Lenticular "idiopathic" achromia	x	x		
Scars (prurigo, lichenification, burns, radiodermatitis, bullous diseases, deep injuries)	x	x	x	
Chemically/drug induced	x	x	x	
Systemic sclerosis	x	x	x	
Morphea (localized scleroderma)	x	x	x	
Lichen sclerosis	x	x	x	
Lupus erythematosus	x	x	x	
Dermatitis	x	x	x	
Eczematids		x	x	
Pityriasis versicolor		x	x	
Seborrheic dermatitis		x	x	
Psoriasis		x	x	
Progressive macular and confluent hypomelanosis		x		Fig. 2.16
Lichen striatus		x	x	
Viral warts	x	x		
Pityriasis lichenoides chronica		x	x	
Sarcoidosis		x	x	
Hansen's disease		x	x	
Mycosis fungoides		x	x	
Pian, other endemic <i>Treponema</i> infections	x	x	x	



**Figure 2.18** Futcher's line, type A.



**Figure 2.19** Futcher's line, type B, on the thighs of an 8-year-old child.

may vary slightly, leading sometimes to striking patterns (Fig. 2.20). All these pigment lines are frequent in persons of phototypes V to VI but can occasionally be seen in persons of phototype IV or even lighter, whereas they may be less frequent in the darkest phototype VI. They are usually present from childhood, sometimes with a familial inheritance. Their precise mechanisms and significance remain unknown.

Another common pigmentary variant that we see in skin of color, and particularly in black skin, is palmar and



**Figure 2.20** Rare extensive demarcation mark showing a continuous line on both shoulders and arms, together with white chest midline.

plantar hyperpigmentation. This typically presents in the form of hyperpigmented macules; lesions vary in size and morphology, with postulated etiology including trauma leading to postinflammatory hyperpigmentation. The differential diagnosis includes melanocytic nevi and malignant melanoma. The mouth is another common location for physiological pigmentation in ethnic populations, with the gums being the commonest site. Other intraoral surfaces affected include the buccal mucosa, hard palate, and tongue. Lesions vary in morphology, with gingival pigmentation presenting as a well-demarcated brown band that typically spares the gingival margin. As with palmar and plantar pigmentation, it is thought that trauma plays a possible etiological role as well as chemical stimulation. Nonphysiological causes of intraoral pigmentation include postinflammatory hyperpigmentation, smoking-related changes, metallic tattoos from older dental prostheses, and of course melanoma, with the latter appearing rapidly within adulthood. Leukedema is also an example of physiological pigmentation within the mouth. It presents as a light-grayish lesion on the buccal mucosa, and although strongly associated with those of Afro-Caribbean descent, it has been reported to occur commonly in whites too [4]. Differential diagnoses include frictional keratosis and white sponge nevus.

Apart from the skin, physiological pigmentary variants are also observed in the nails. Benign melanonychia, characterized by longitudinal nail pigmentation, is reported to exist in 50–90% of black individuals over the