

# The New ABnCs of Skin Care: Aging, Bariatric, Non-Caucasian Skin Considerations for the Healthcare Provider

Heather Hettrick PT, PhD, CWS, CLT, CLWT  
Associate Professor  
Department of Physical Therapy  
Nova Southeastern University

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## Objectives & Overview

- Appreciate age related skin changes and the impact on integumentary function
- Describe common skin conditions associated with bariatrics
- Recognize normal dermatological variants of non-caucasian skin



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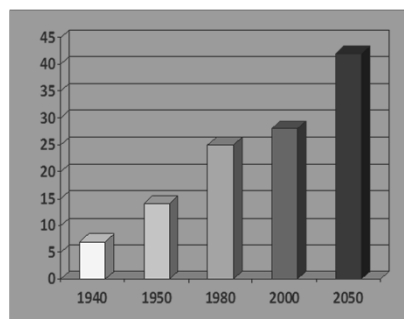
" I DON'T THINK OF MY SKIN AS SAGGY... I THINK OF IT AS RELAXED-FIT! "

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## Demographics

- The next generation of retirees will be the healthiest, longest-lived, best-educated and most affluent in history
  - Those reaching 65 have an additional 17.9 years of life expectancy
  - Likelihood of surviving to 90 has doubled
  - By 2050, 40% of 65 year olds are likely to reach 90

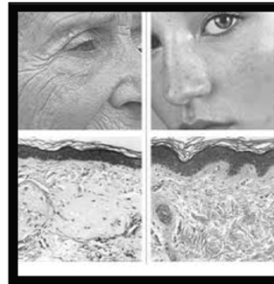


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## Aging Skin: Gerontodermatological Changes

Over the lifespan, skin becomes drier, less elastic, less perfused → vulnerable to damage from pressure, friction, shear, moisture, malnutrition, etc.

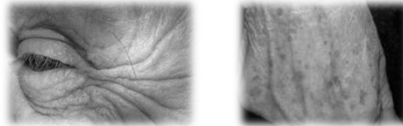


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## Aging Skin: Gerontodermatological Changes

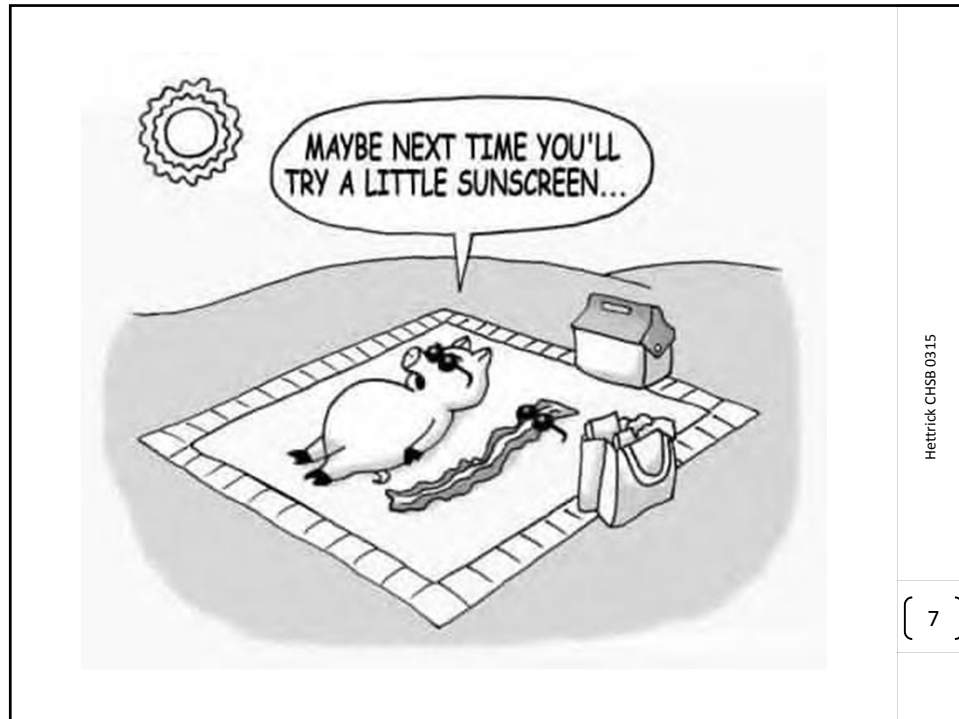
- Skin aging is a complex process
  - Most major changes occur in the dermis
- Two independent aging processes
  - Normal aging → slow, irreversible degeneration of tissue; caused primarily by buildup of reactive oxygen species as a byproduct of cellular metabolism



- Extrinsic aging → AKA photoaging/dermatoheliosis due to exposure of the elements (primarily UV irradiation) as well as pollution and smoking

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## Aging Skin: Gerontodermatological Changes

- Changes begin in 3<sup>rd</sup> decade, accelerate in 6<sup>th</sup> decade
    - Keratinocytes become short and fat while corneocytes become bigger due to a decrease in epidermal turnover
    - Melanocytes decrease 8-20% per decade resulting in uneven pigmentation in elderly skin
    - Sebum production decreases ~60%
    - Global lipid content is reduced ~65%
    - Flattening of dermo-epidermal junction by more than 1/3 due to loss of dermal papillae\*
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## Aging Skin: Gerontodermatological Changes

- Reduction in the following:
  - dermal thickness
  - Volume of subq fat
  - vascularity
  - cellularity
  - mast cells
  - fibroblasts
  - GAG, HA, ground substance
  - Collagen turnover
  - Elastin (becomes more calcified)
- Leads to:
  - Increased rigidity
  - Decreased torsion extensibility
  - Diminished elasticity
  - Increased vulnerability to tear-type injuries
  - Significantly increased time to recover from mechanical depression
    - Minutes in young skin; > 24h in aged skin



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## Aging Skin: Gerontodermatological Changes

- Combination of normal aging and photoaging results in altered wound healing processes
  - Progressive loss of skin function
  - Increased vulnerability to the environment
  - Decreased homeostatic ability

*Healing is delayed but is as effective as that of younger adults*



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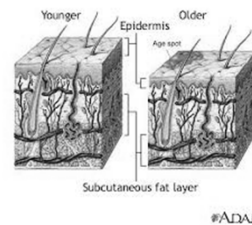
"Your doctor can only do so much.  
The rest is up to you. Stop getting older."

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## Aging Skin: Gerontodermatological Changes

- These changes are called *replicative senescence* → normal aging process where...
  - Epithelial and fatty layers thinner
  - Collagen and elastic fibers shrink 1% per year
  - Sweat glands decrease in number and size
  - Skin vascularity decreases
  - Vessel walls thin
  - Atherosclerotic changes occur in and large vessels



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## Aging Skin: Gerontodermatological Changes

- With these changes...
  - Oxygen-carbon dioxide exchange decreases
  - Tissue turnover slows
  - Increase occurrence of ecchymosis
  - Inflammatory response decreases
  - Tissue regeneration is slower which can delay healing and make tissue more susceptible to infection

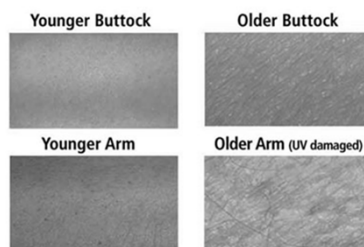
*All these factors can ultimately lead to skin breakdown and impact wound healing*

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## Aging *Changes* Skin

- Normal aging *changes* skin
- 80% of adults older than 65 suffer one or more chronic conditions
- Drug therapies contribute to fragility of the skin
- Advanced age, co-morbidities, medications, environmental factors and lifestyle contribute to the overall condition of the skin



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## Comorbidities Contributing to Skin Alterations and Delayed Wound Healing

- Diabetes-- anhydrosis
- Vascular disease-- skin infarction
- Cardiac disease-- diminished perfusion
- Dementia-- can impair care and outcomes
- Immobility-- pressure, friction, shear

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## Medications Contributing to Skin Alterations

- Corticosteroids
- Antibacterials
- Antihypertensives
- Analgesics
- Tricyclic antidepressants
- Antihistamines
- Antineoplastic agents
- Antipsychotic drugs
- Diuretics
- Hypoglycemic agents



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## Xerosis



*Chief complaint of nearly 80% of all long-term care residents*

What can be done?

- Humidifier
- Room temperature as low as comfortable
- Bathe in warm (not hot) water
- Avoid harsh soaps and drying agents (such as powders)
- Use skin emollients (after bath while skin damp and before bed)
- Reduce bathing frequency to 1-2 times/week
- Offer fluids to residents/patients

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Reddy M. Skin and Wound Care: Important Considerations in the Older Adult: Advances in Skin & Wound Care, Vol 21, No 9:424-438, 2008

## So what does this mean for the skin?

Dr. Vincent Falanga...  
 “We are outliving our skin”

Our skin does not  
 have an official expiration  
 date, but it can fail too!



When potato salad goes bad

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## Skin Barrier Failure

Skin failure is a multifactorial process that is directly related to the functions, or dysfunctions of the other organ systems.

For instance...pressure ulcers are an example of *failure of skin barrier function*.

Similar to other organ failure (heart , liver, kidney failure) skin barrier failure can be classified as acute, chronic or end-stage.

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## Pressure Ulcers & Skin Failure

- **Pressure Ulcers**

“...localized injury to the skin and/or underlying tissue usually over a bony prominence, as a result of pressure, or pressure in combination with shear and/or friction.”

-- NPUAP

- **Skin Failure**

“...an event in which the skin and underlying tissue die due to hypoperfusion that occurs concurrent with severe dysfunction or failure of the organ systems.”

-- Langemo/Brown

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Skin failure and pressure ulcer development in older adults and the terminally ill is not always preventable

*‘Permissible PrU’*

*‘skin failure’*

*‘unavoidable PrU’*

In the failing individual, skin deterioration is often the most outward manifestation of overall faltering physiology

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## Skin Failure

### Acute

- Skin & underlying tissue die due to hypoperfusion concurrent with a critical illness
- More often seen in ICU or acute care settings

### Chronic

- Skin & underlying tissue die due to hypoperfusion concurrent with ongoing, chronic disease states
- Occurs in a more steady fashion
- Individuals usually older and have multiple comorbidities
- Internal organ systems increasingly and irreversibly lose their ability to function as the end of life nears

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## End-Stage Skin Failure

- An event in which skin and underlying tissue die due to hypoperfusion concurrent with the end of life
- Chronically critically ill patients/residents present many challenges to maintain skin integrity
- The transition from acute to chronic to end-stage skin failure may not follow an easily observable continuum

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## End-Stage Organ Decompensation & Failure

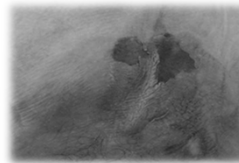
- Large and unusual presentations of skin failure
- Body shunts blood to vital organs
- Widespread and deep tissue destruction over stressed areas can appear in a matter of hours or less
  - Sacrum
  - Heels
  - Posterior calf muscles
  - Arms
  - Elbows

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## Kennedy Terminal Ulcer

- An unavoidable ulcer
- Present at end of life
- Onset typically 2 to 6 weeks before death
- Common at sacrum in the shape of a pear, butterfly or horseshoe with irregular borders
- Side effect of the dying process
  - May starts out superficially as a blister or a Stage 2
  - Looks much like an abrasion with small black vasculitic-like spots
  - Rapidly progresses to a Stage III or a Stage IV



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## Multiple Organ Death

- Reveals itself on the external skin
- Few interventions lessen the external skin damage - the body begins to shut down
- Clear, honest communication of medical assessments and prognosis among:
  - Health care providers
  - Patient/resident
  - Significant others
- Establish realistic goals for treatment of unavoidable complications, pain and suffering at the end stages of life

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## SCALE

- Skin Changes At Life's End
- Expert panel published paper in 2009
- Current understanding of complex skin changes at life's end limited

*Not all pressure ulcers are avoidable*

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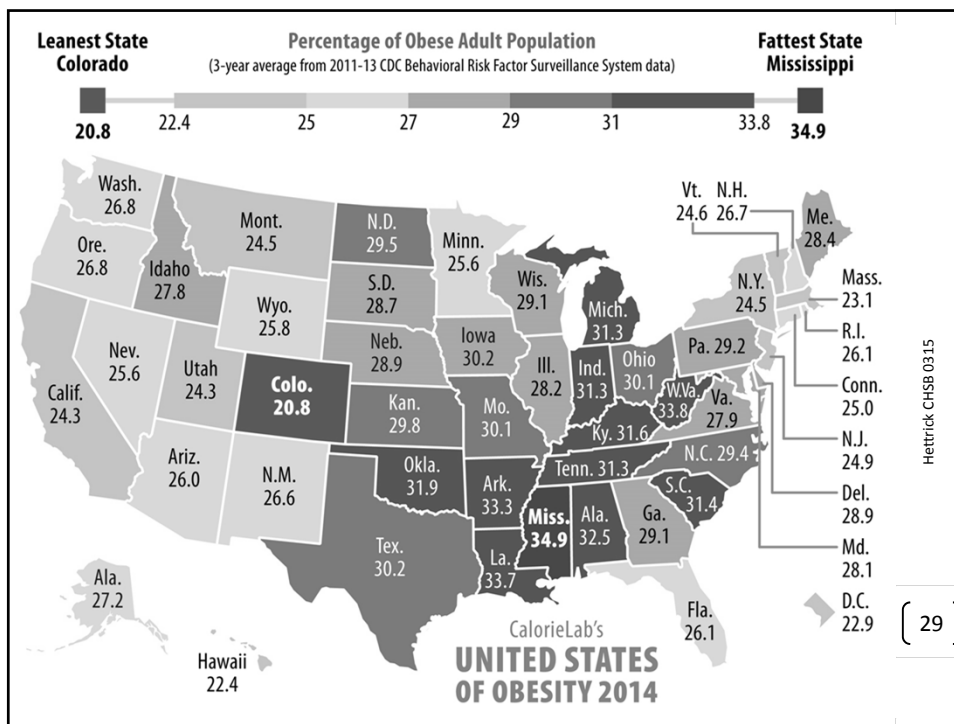
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## Obesity Complications and the Skin

- Bariatric is derived from the Greek word *baros* meaning weight; Bariatric patient usually has a BMI > 30

cancer  
 joint problems  
 low self-esteem  
 lower life expectancy  
 heart attacks  
 depression  
 fewer employment opportunities  
 breathing problems  
 high blood pressure  
 hernia  
 type 2 diabetes  
 bone problems  
 increased sweating  
 limited mobility  
 deep vein thrombosis  
 high cholesterol  
 arthritis  
 social discrimination

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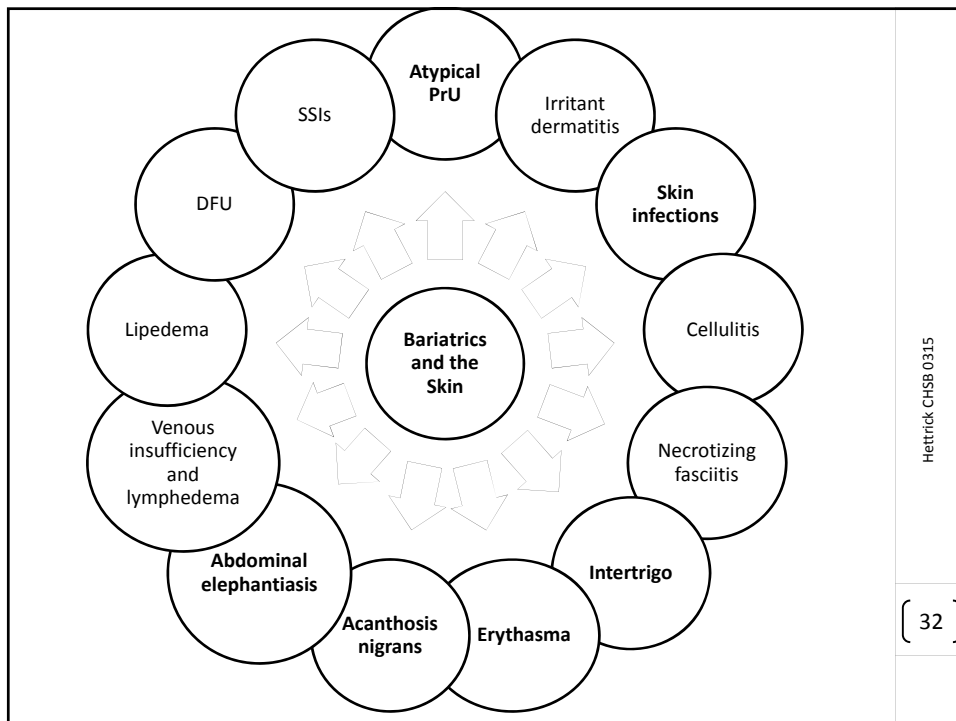
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# Obesity and the Skin

- Adipose tissue poor perfusion, inadequate oxygenation and increased diffusion distance
- Excessive sweating increased risk for bacterial/fungal infections
- Immobility, friction and shear due to weight stress skin's barrier function
- Malnutrition
- Striae (stretch marks) reflect tension on skin
- Hirsutism (women) may result due to production of testosterone associated with visceral obesity
- Acrochordons (skin tags) are common

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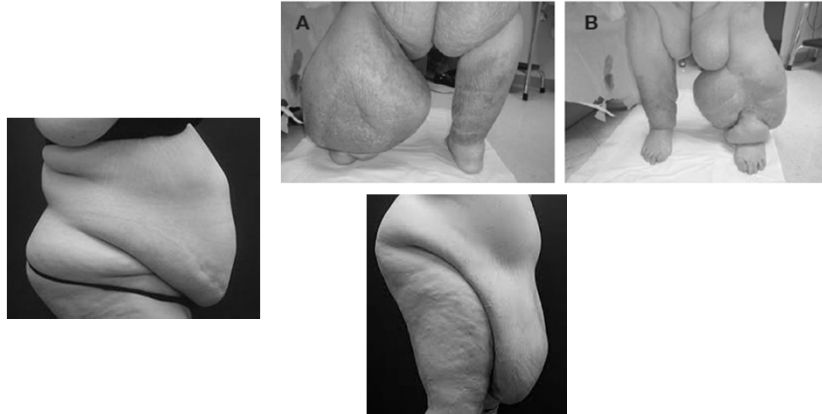


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## Atypical Pressure Ulcers

- Located deep within skin folds
- Requires frequent skin fold assessment



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## Skin Infections

- Spectrum of benign conditions to life-threatening necrotizing infections
- Skin folds: trap moisture, induce maceration, promote microbial overgrowth
- Obesity:
  - Hinders lymphatic flow
  - Impairs perfusion (greater diffusion distance)
  - Skin pH higher – increase risk of candida

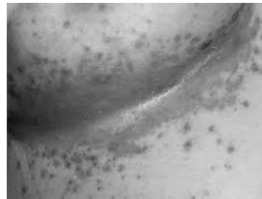


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## Intertrigo

- Infectious or non-infectious inflammatory condition of two opposed skin surfaces
- Presents as erythematous, macerated plaques and erosion with possible scaling



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## Erythrasma

- Common in skin folds and perineum
- Common in tropical, hot or humid climates
- Superficial infection of skin caused by *Corynebacterium minutissimum*
  - Bacterial infection
- Presents as macerated reddened scaly plaques common in intertriginous areas- groin, axilla and foot



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## Acanthosis Nigricans

- Bariatric patients at high risk
- Benign condition characterized by velvety, hyperpigmented plaques on skin and intertriginous areas
- More common in persons of African, Hispanic and Native American origin
- Disorders related to insulin resistance also seen with AN

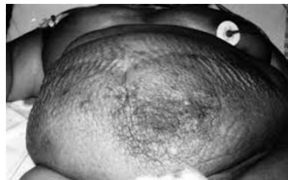


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## Abdominal Elephantiasis

- Unusual condition
- Can occur in large abdominal pannus due to prolonged lymphedema and associated fibrous tissue proliferation



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# Management Considerations

- Skin and wound care
  - Hygiene
  - Skin fold management
  - Perigenital care
  - Toileting
  - Odor management
  - Bariatric equipment
  - Psychosocial

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**“Kevin’s skin is red, yellow, brown, blue, purple, green, pink, orange and black. His tattoos really add a lot to our diversity!”**

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## What does the term “skin of color” encompass?

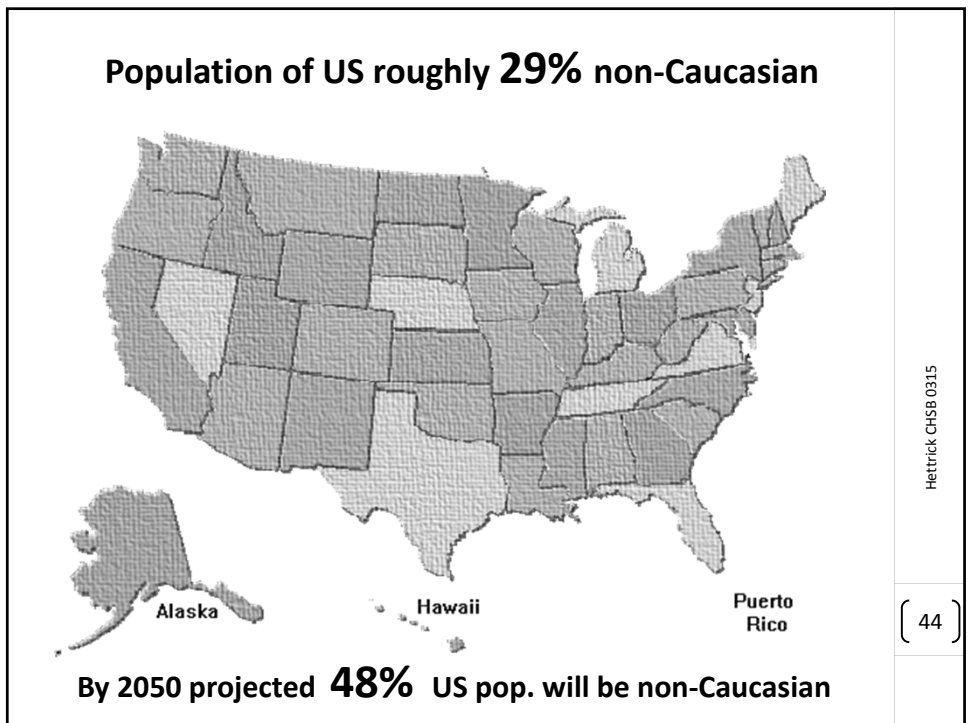
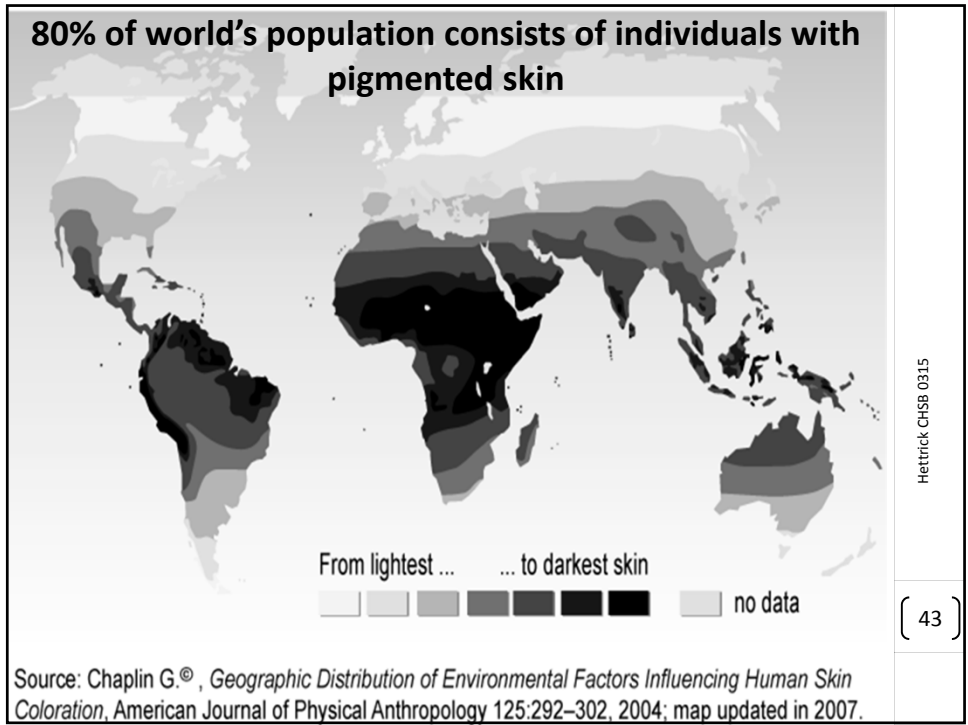
Accepted dermatologic term to describe people with all shades of pigmented skin

Level	Associated Ethnicities
I	Northern European, British
II	European, Scandinavian
III	Southern European, Central European
IV	Mediterranean, Asian, Latino
V	East Indian, African, Native American
VI	African, Aboriginal

Pigmentary phototype (Fitzpatrick scale)

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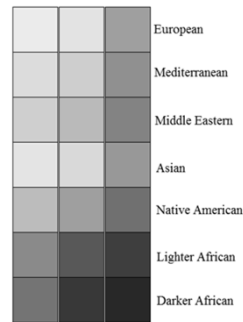
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# Pigmentation

Normal skin color / tone composed of biochromes:

- Melanin - brown
- Carotenoids - yellow
- Oxyhemoglobin - red
- Reduced hemoglobin – blue



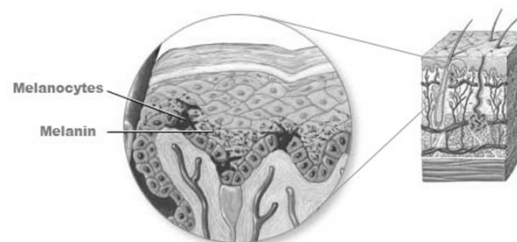
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The total amount of **melanin** is the principle determinant of skin color

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# Pigmentation

- Cutaneous pigment *melanin* produced by melanocytes
- *No significant differences* in the actual number of melanocytes (Szabo 1969)
- Differences in skin color are attributed to **differences in the rate at which melanosomes are produced and melanized**

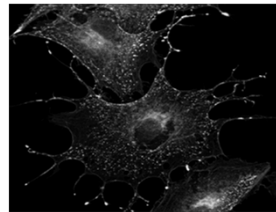


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## Pigmentation

- *Distribution* of melanosomes within melanocytes & keratinocytes different between skin pigments
- Tyrosinase (, a copper-containing enzyme involved in melanin synthesis) levels are 10 x higher in people of African-decent
  - Produce 10 x more melanin than melanocytes in Caucasian skin (Lozumi et al, 1993)
  - People with albinism have an absence or defect of tyrosinase
    - They also do not keloid...



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[www.ccs.k12.in.us/chsBS/kons/kons/melanocyte.png](http://www.ccs.k12.in.us/chsBS/kons/kons/melanocyte.png)

## Why Is This Important?

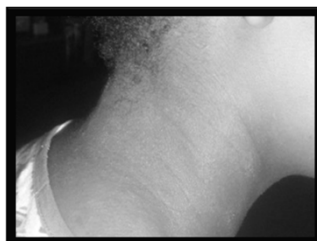
- Problem for clinicians when assessing patients with pigmented skin is lack of guidance and/or evidence
- Understanding racial differences in skin function and appearance is essential for:
  - Skin care
  - Prevention
  - Recognition & intervention
- Outside of color spectrum, there are very few differences within the integumentary system across ethnicities

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## Black Skin Care Considerations

- Although its thickness does not vary according to skin color, the stratum corneum of black people contains more layers of corneal cells.
  - For this reason, the protective mantel is more compact and robust, despite the fact that it contains fewer ceramides (essential lipids).
  - Because of this, black skin can sometimes appear ashy when it becomes dry.



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## Black Skin Care Considerations

- The pores, sweat glands and sebaceous glands in black skin are larger. Dark skinned people produce more sebum around hair follicles, have more microbial flora and a lower pH (more acidic) skin.
  - Because of this, black skin is more prone to scarring from acne and to spontaneous peeling. However, it is also less sensitive to certain chemicals that irritate the skin of white and Asian people.
- Regardless of skin color, it is recommended to use products that contain squalane. (Montagna et al, 1993)



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## Main Structural Differences in Stratum Corneum Barrier Function (Color vs. Caucasian) (Berardesca & Maibach, 2003)

- Equal thickness
- ↑ number of cell layers (cohesion) & increased resistance to stripping
- ↑ recovery after stripping
- ↑ lipid content (yet decreased ceramides)
- ↑ electrical resistance
- ↑ number of fibroblasts, larger & more physiologically active (Montagna, 1991)



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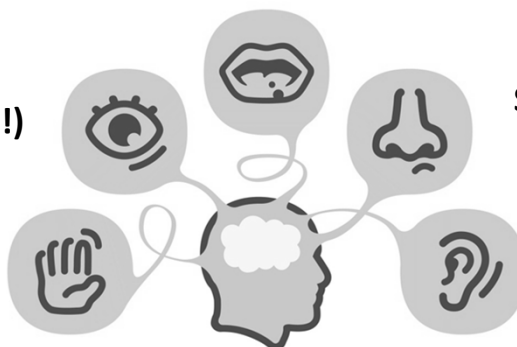
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## Skin Assessment Basics

Visual inspection alone NOT sufficient !!!

Talk/document

Look  
(lighting!!!)



Smell

Touch

Listen

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## Post Inflammatory Hyper/Hypopigmentation

- Black skin may respond to trauma or inflammation by either an increase or decrease in pigmentation (**dyschromia**)
  - Melanocytes respond in exaggerated way
  - Marked change in pigment
- Dyschromia following an inflammatory event is known as post-inflammatory hyperpigmentation
  - Increase in melanin production or uneven distribution of melanin
  - Excess pigment either in epidermis only or epidermis and dermis

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## Post Inflammatory Hyper/Hypopigmentation

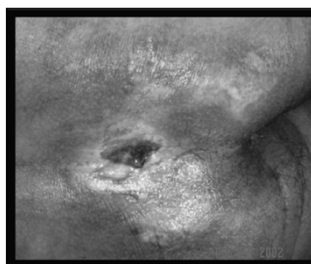
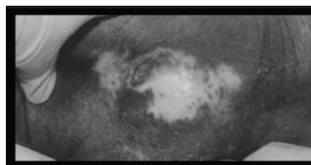


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## Post Inflammatory Hyper/Hypopigmentation

- Hypopigmentation represents as either as localized or widespread loss of melanin in the skin
  - May be due to loss of functional melanocytes
  - Presents as depigmented macules and patches with feathered edges

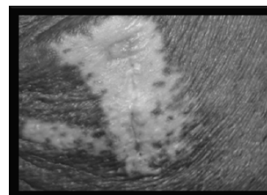
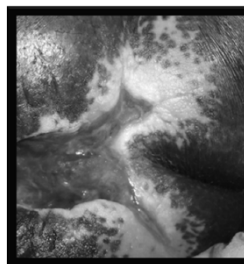


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## Post Inflammatory Hyper/Hypopigmentation

- Many of these pigment alterations normalize over time
- Cause is unknown...possible influence of inflammatory mediators and reactive oxygen species
- Pigmentation is transient...may take months or years to normalize



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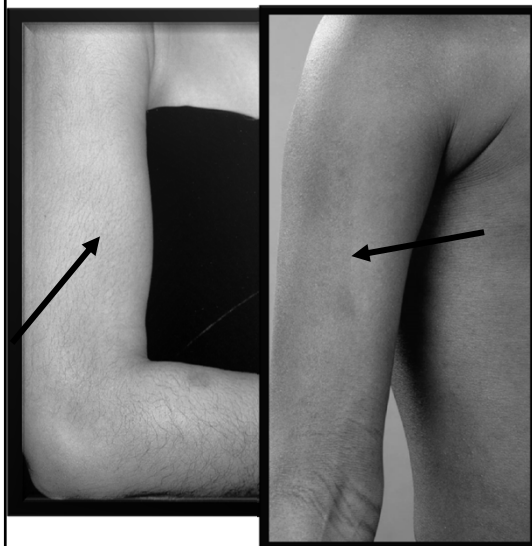
## Normal Variations in Black Skin

- **Futcher's (Voigt's) line**
  - Sharp demarcation between darkly pigmented and lightly pigmented skin in the upper extremity
  - Follows spinal nerve distribution
- **Midline hypopigmentation**
  - Line of hypopigmentation over the sternum
  - Lessens with age
- **Nail pigmentation**
  - Diffuse nail pigmentation or linear dark bands on the nail
  - May appear brown, blue, or blue-black
- **Palmar changes**
  - Creases may be hyperpigmented
  - May contain hyperkeratotic papules or pits in the creases
- **Plantar changes**
  - Hyperpigmented macules may vary in color and distribution
  - May present with irregular borders
- **Dermatosis Papulosa Nigra**
  - Brown to black papules
  - Family history, more common in females

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## Normal Variations in Black Skin



### Futcher's or Voigt's Line:

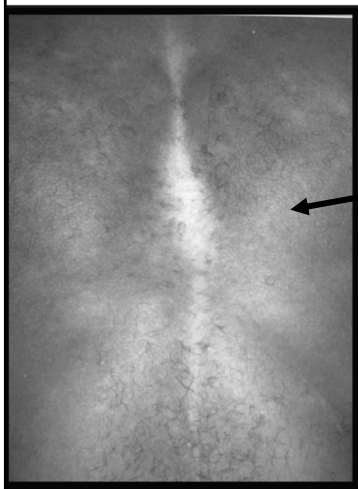
- Sharp, bilateral, pigmentary demarcation lines usually on lateral side of biceps
- Incidence of 25% reported in heavily pigmented black persons
- James found 79% of black females had at least one type of line
- Benign condition

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## Normal Variations in Black Skin



### Midline Hypopigmentation:

- Linear band overlying the sternum
- Unknown etiology; may be inherited in an autosomal dominant pattern
- Incidence approximately 30-40% in black persons
- Males primarily affected; becomes less noticeable with age

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## Normal Variations in Black Skin



### Longitudinal melanonychia:

- Linear hyperpigmented nail streaks
- Represents normal variant in over 50% of black people
- Melanin is deposited in nail plate/matrix possibly due to trauma or UV light
- Positive correlation with advancing age
- Thumb & index nails most commonly involved
- Often bilaterally
- Drugs such as antimalarials, bleomycin, doxorubicin, and zidovudine may cause nail pigmentation
- Associated with systemic diseases such as Addison's and Peutz Jegher's
- *An irregular nail pigment or history of changing lesion warrants biopsy as 20% of melanomas in black people are found in the nails*

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## Normal Variations in Black Skin



### **Palmoplantar Hyperpigmentation:**

- Due to localized hypermelanosis
- Polymorphous brown macules with sharp or indistinct borders
- Creases on the palms often present with hyperpigmentation & may contain hyperkeratotic papules or pits

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## Normal Variations in Black Skin



### **Idiopathic Guttate Hypomelanosis:**

- AKA Disseminate Lenticular Leucoderma
- Small, white, irregularly shaped macules primarily on anterior legs
- Unknown etiology; benign
- Macules range in size from 2-6 mm
- More common in women over age of 40
- May be due to sun exposure
- Histologically, decrease in number of melanocytes

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## Normal Variations in Black Skin



### Dermatosis Papulosa Nigra:

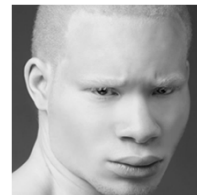
- 35-77% of black individuals may be affected
- 50% have family history; more common in females and peaks in the 6<sup>th</sup> decade of life
- Benign, brown to black papules most common at the neck, face, trunk
- 'Flesh moles' do not require treatment although some seek cosmetic excision

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## Abnormal Variations

- Pigmentary skin *disorders* can cause emotional distress & social stigma
- Most of these can be seen in various ethnicities
  - Disorders result of altered melanin production
  - *Most common pigmentary disorders for all races:*
    - Albinism
    - Vitiligo
    - Melasma – often seen in pregnancy
    - Ephelis (freckles)
    - Lentigo (liver spots)

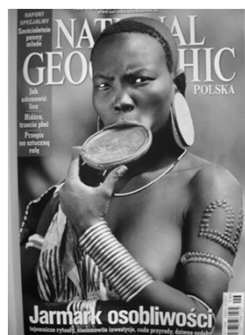
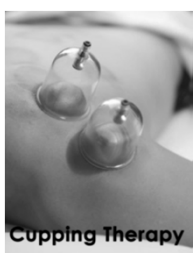


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## Impact of Culture, Cosmetic Customs, Tribal/Social Markings

- Cupping, coining, spooning, moxibustion, salting, herbal rubs, acupuncture, body modification

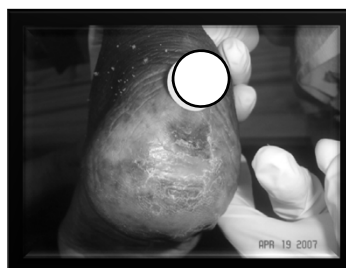


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## How Does This Impact Wound Management?

- Thorough history & physical exam should reveal normal/abnormal dermatological conditions
- Early detection of skin lesions is top priority
- This can be problematic in darker pigmented individuals
  - *Erythema and/or blanching are not reliable indicators on their own*



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## How Does This Impact Wound Management?

- Must use all your senses...

- LOOK

- What is normal for the individual?
    - Compare area to surrounding skin or contralateral side if applicable
    - Is the area in question a site of previous injury/scar?



- LISTEN

- Is the individual complaining of pain, itching or other sensory changes?

- TOUCH

- Is the area warmer/cooler?
    - Is the area firm/boggy?



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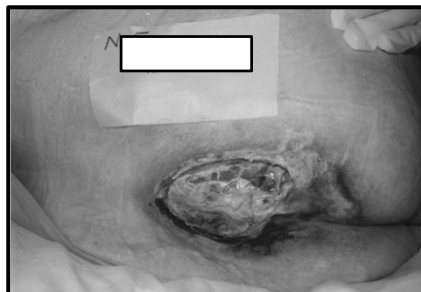
[ 67 ]

## Differential Diagnosis and Photo Gallery

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## Clinical Presentation Comparison

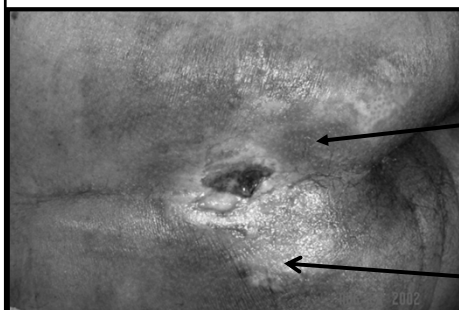


Unstageable sacral pressure ulcer  
in dark skin (left) & light skin (right)

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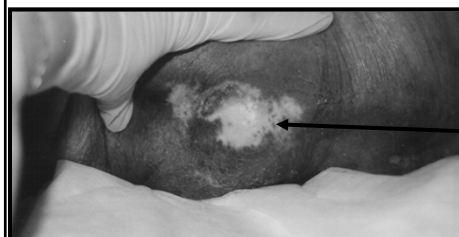
( 69 )

## Clinical Presentation Comparison



Hyperpigmentation

Hypopigmentation

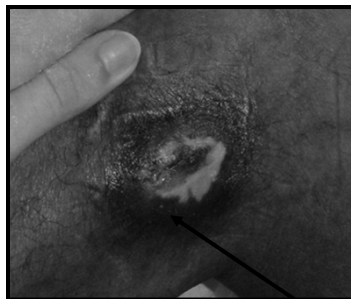


Macules of re-pigmentation

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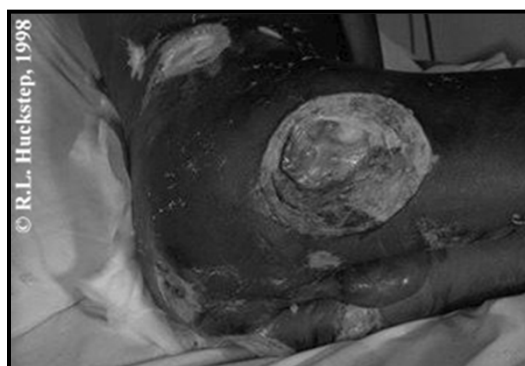
## Clinical Presentation Comparison



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- Note characteristics of wound margin & periwound area
- Hyperpigmentation present due to inflammatory response
- Difficult to determine if tissue is bruised, infected or suspected deep tissue injury ( 71 )

## Clinical Presentation Comparison



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- From photo, very difficult to determine viable vs nonviable
- Clinicians cannot rely on visual cues alone in darkly pigmented individuals ( 72 )
- Thorough skin & wound assessment involves all senses!



## Clinical Presentation Comparison



Stevens-Johnson Syndrome in dark and light skin. Lesions appear hyperpigmented and somewhat flush in dark skin and red and elevated in light skin.

( 73 )

## Clinical Presentation Comparison



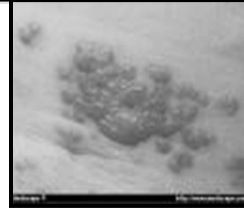
- Kaposi's sarcoma presents as confluent macules on dark skin and purple/red elevated nodules on light skin.
- Same disease with significantly different clinical presentations.

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## Clinical Presentation Comparison



- Presentation of shingles
- Pattern still follows dermatome
- Yet different pigmentary response to active & resolved lesions



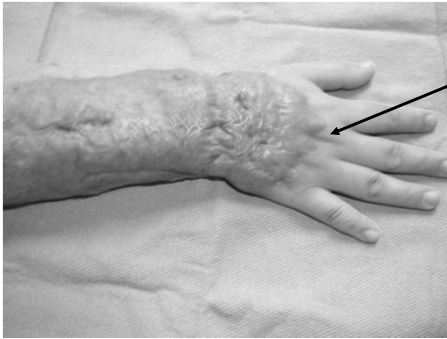
[ 75 ]

## Clinical Presentation Comparison




- Maturing scar tissue on dark and light skin
- Black individuals are 2-19 times more likely to develop Keloids than their Caucasian counterparts. (Connolly, Bikowski 2006)

[ 76 ]



**Hypertrophic scar-** scar tissue is raised & rigid yet confined to the boundaries of initial injury.

Photo courtesy Dayna Gary



**Keloid scar-** scar tissue that extends well beyond the boundaries of initial injury

Photo shows a keloid after an ear piercing.




Photo: dermatnet.com used with permission

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## Summary- A, B, nCs of Skin

- Appreciate age related skin changes and the impact on integumentary function
- Describe common skin conditions associated with bariatrics
- Recognize normal dermatological variants of non-caucasian skin



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Thank  
You



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