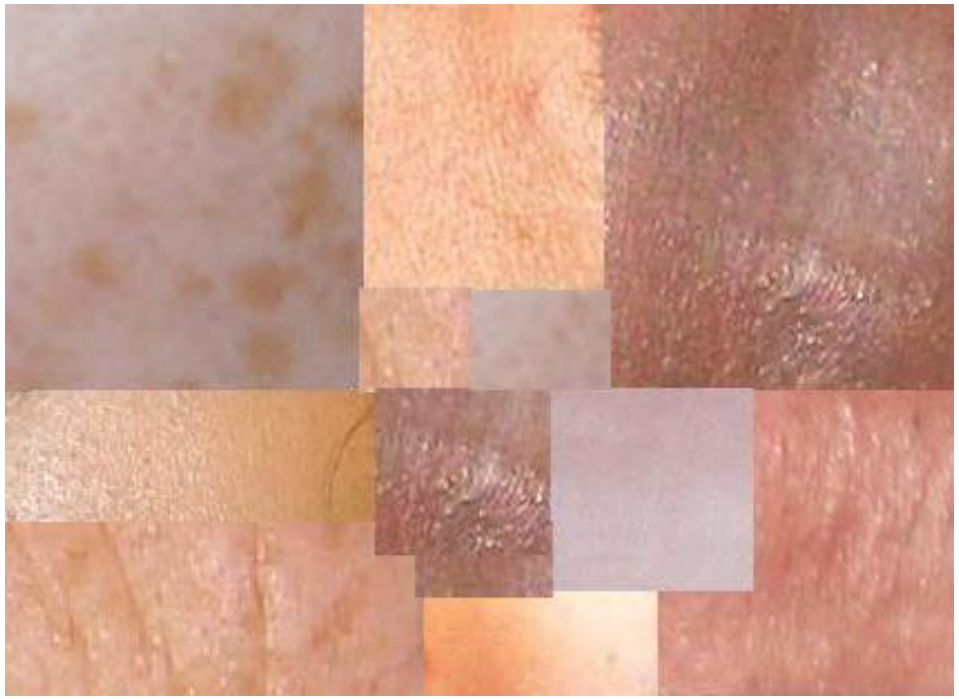
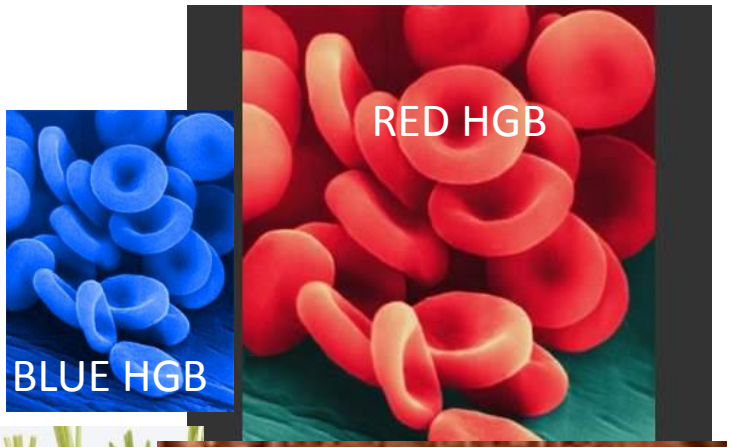
Disorders of pigmentation

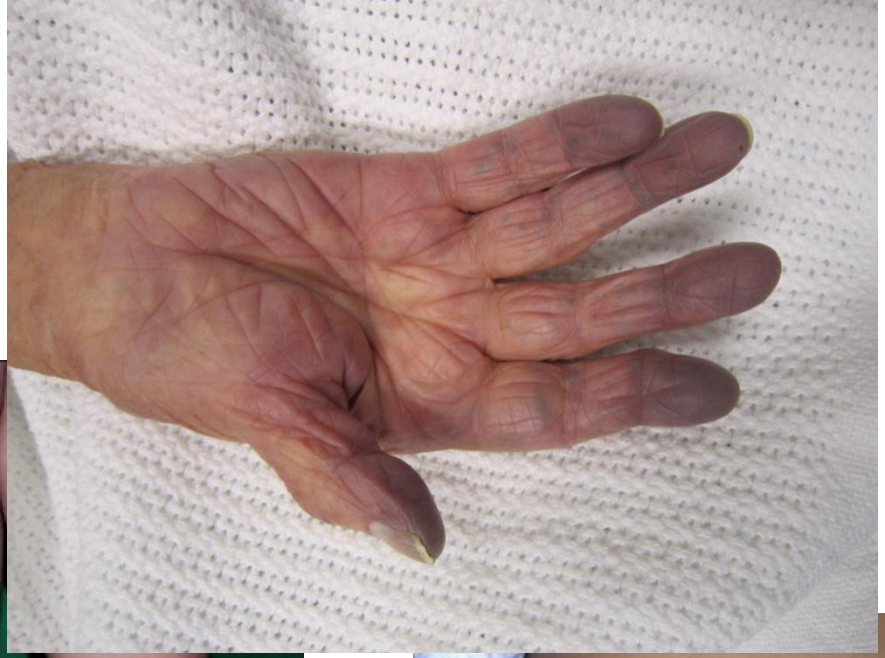
Antoanela Čarija, dr. med

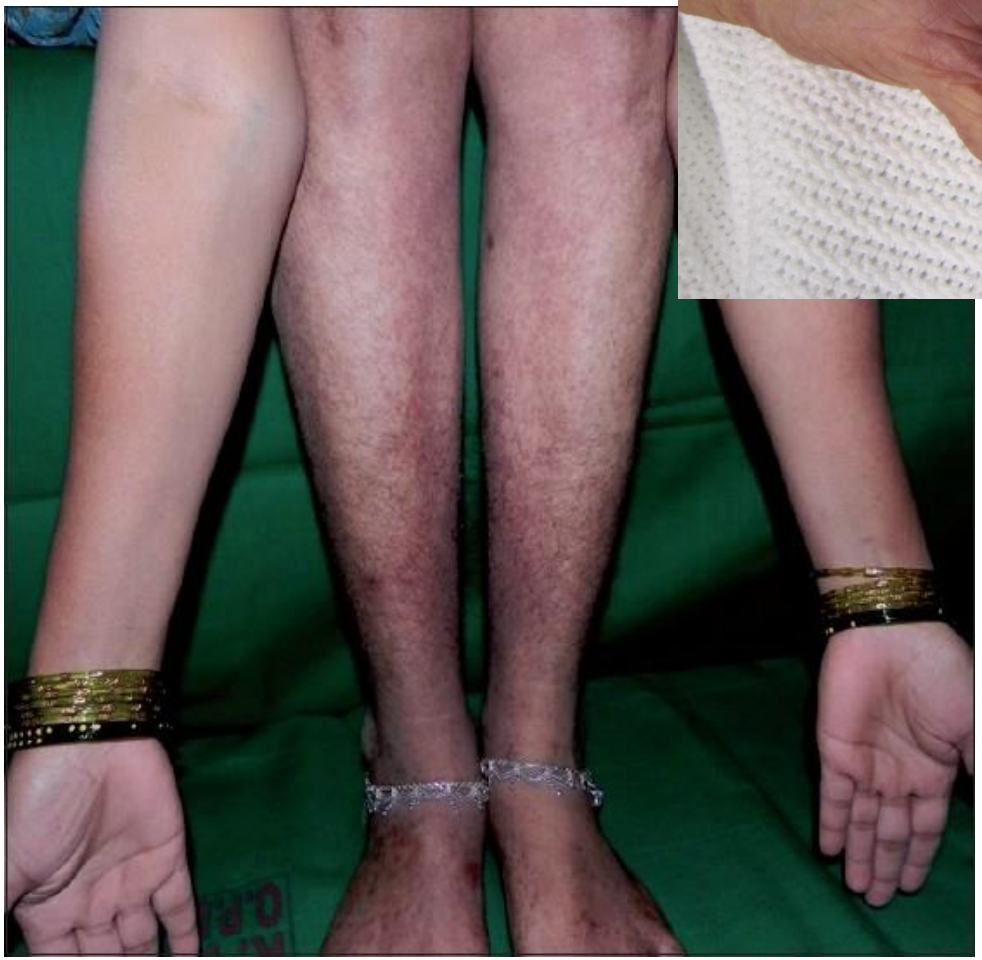
Skin colour

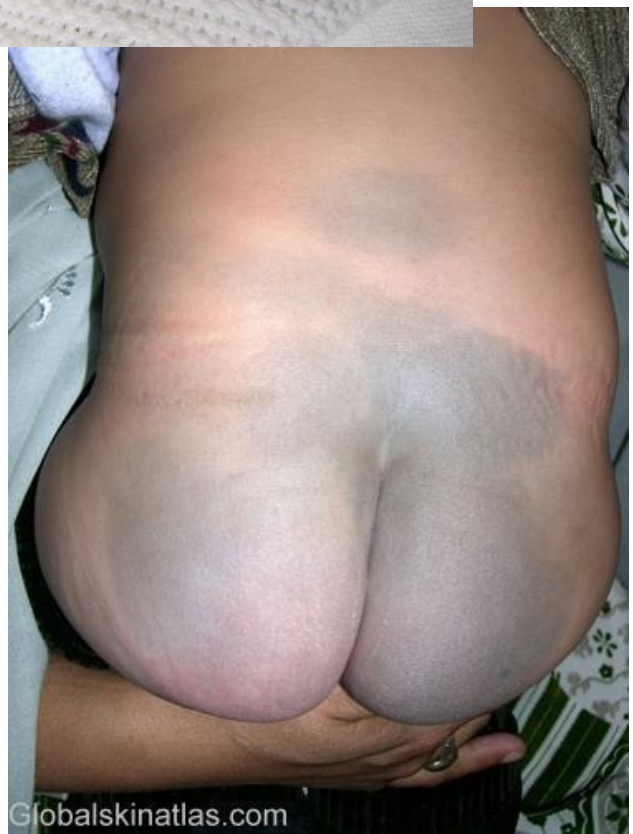




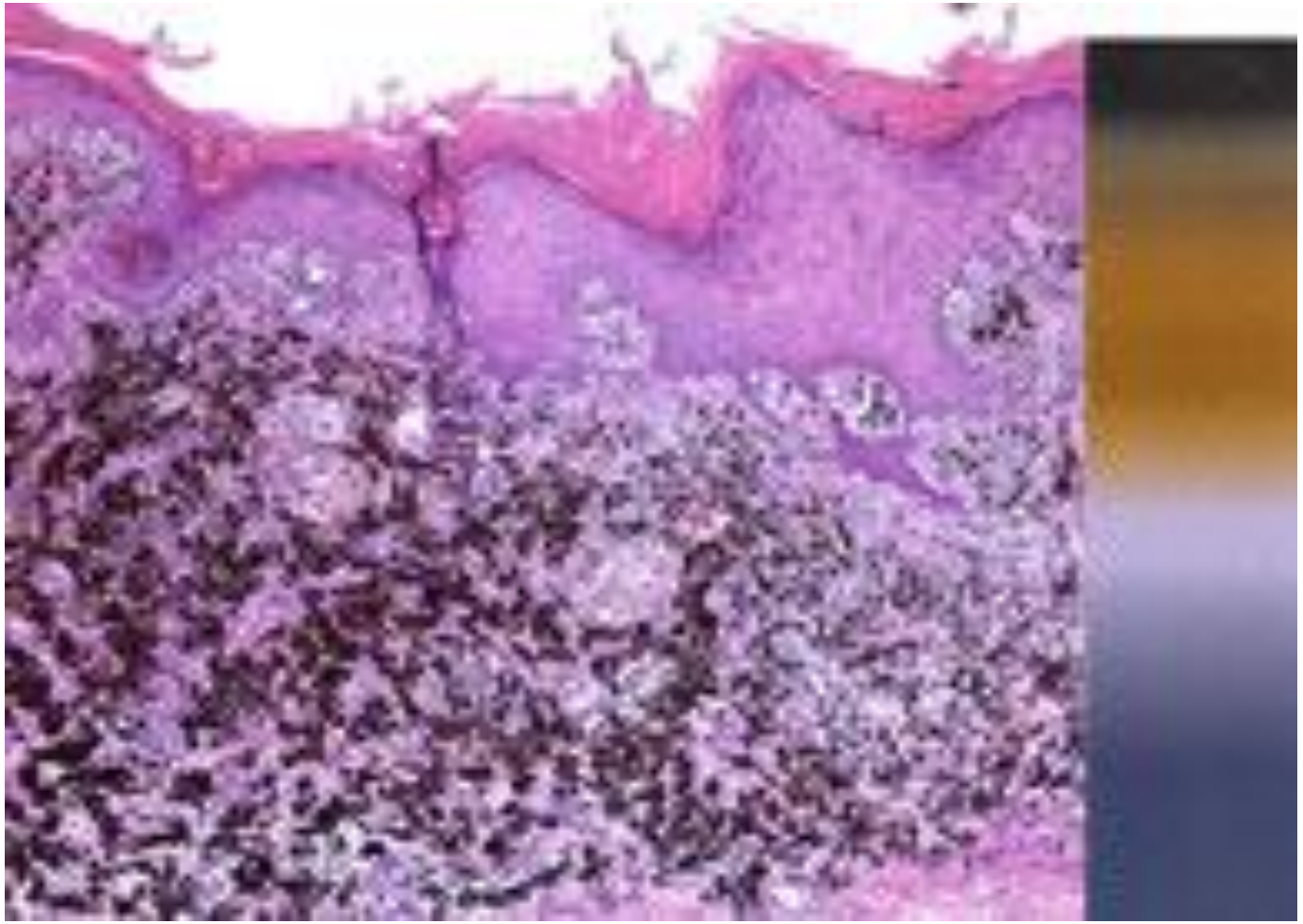
Where is melanin?







Melanin in the skin

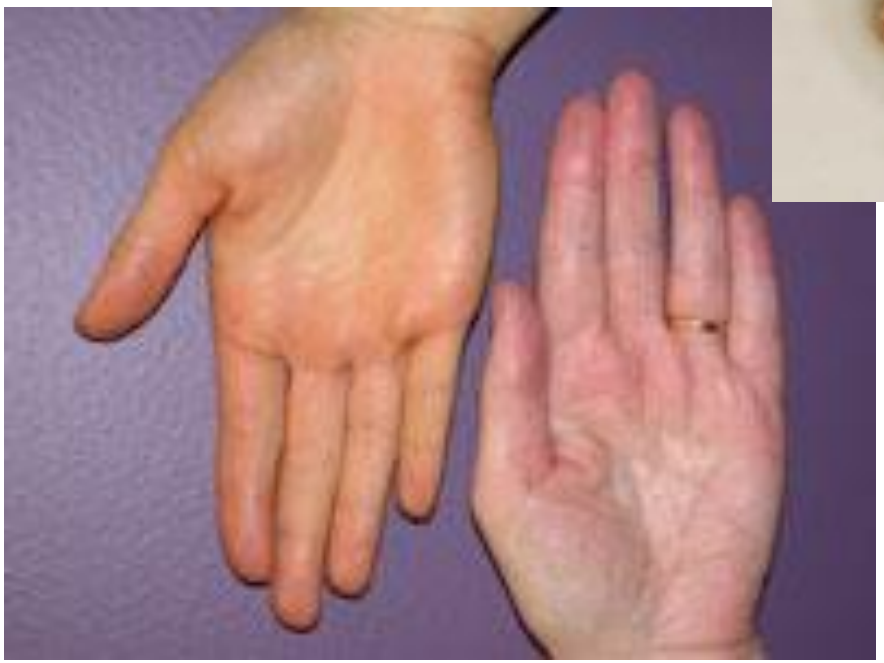


CAROTINOSIS

1. PRIMARY

2. SECONDARY

(hypothyroidism , liver and kidneys, DM)



in cooked ,
pureed food

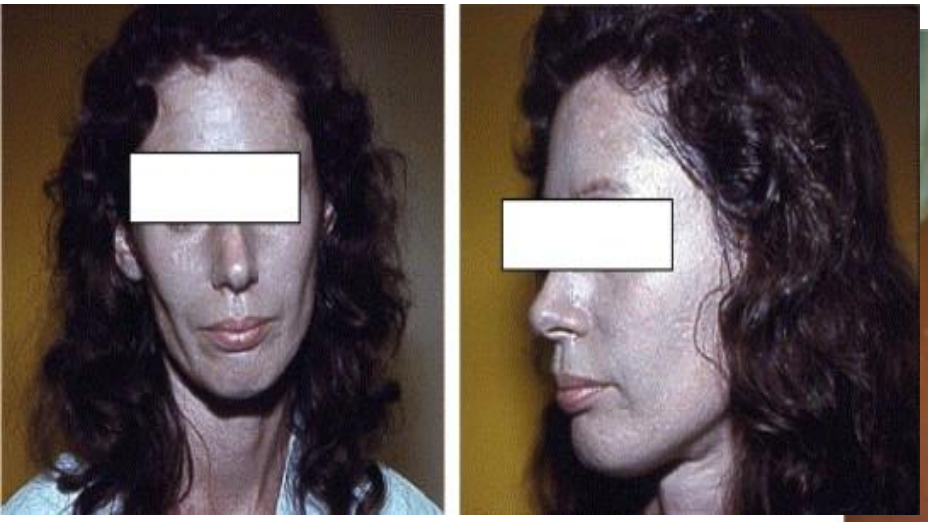
Breast milk
(colostrum)

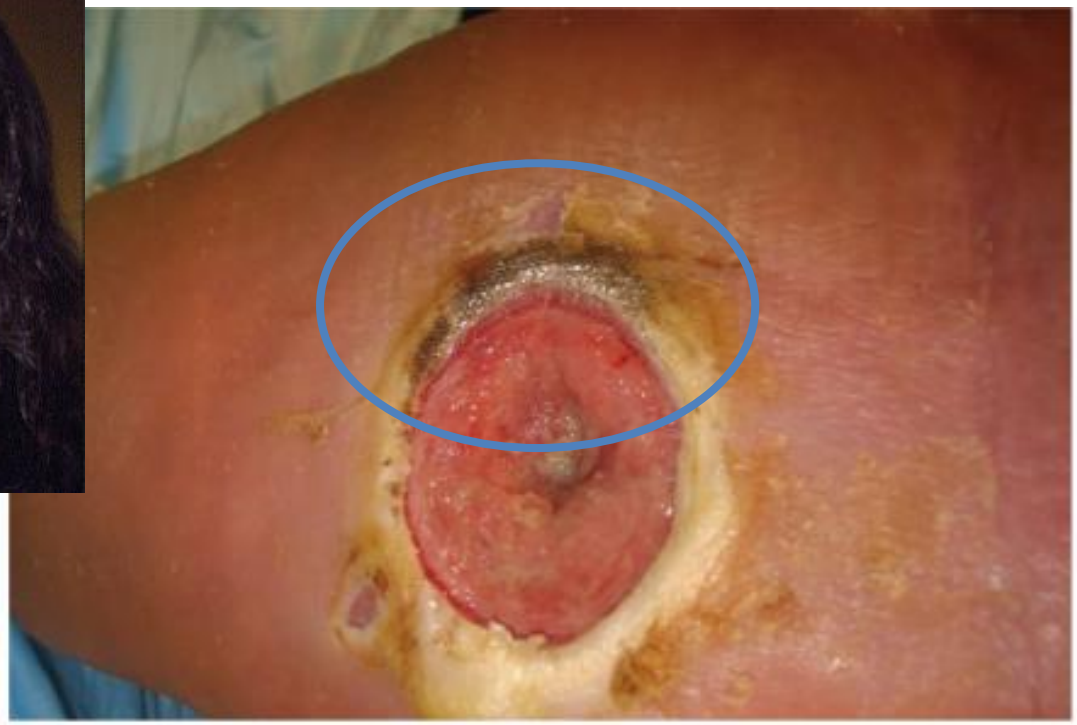
It accumulates in
epidermis
and
in the sweat glands

Drug induced pigmentations

10-20% acquired pigmentations

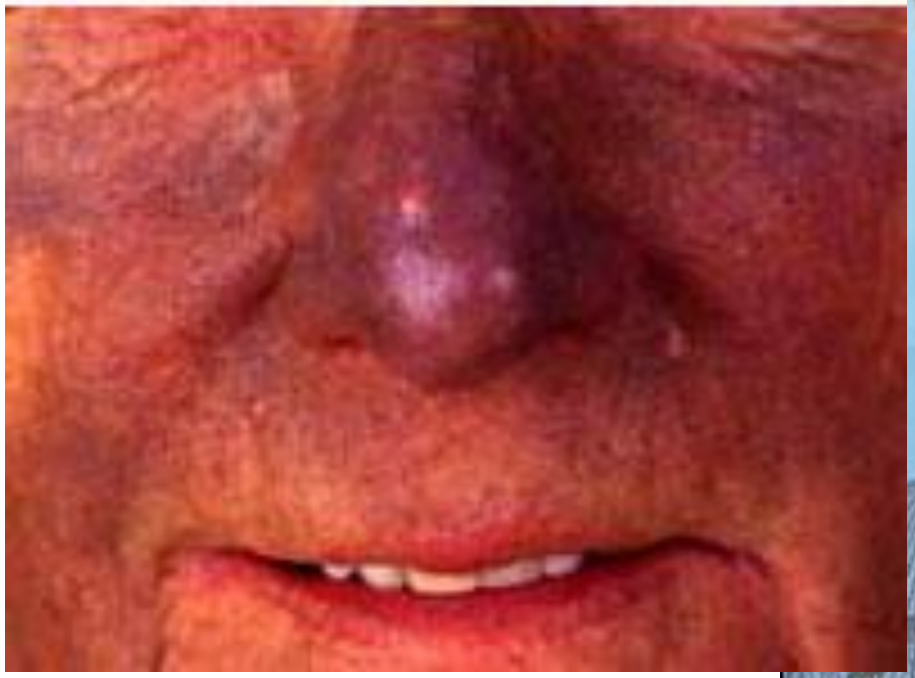
Argyria - silver

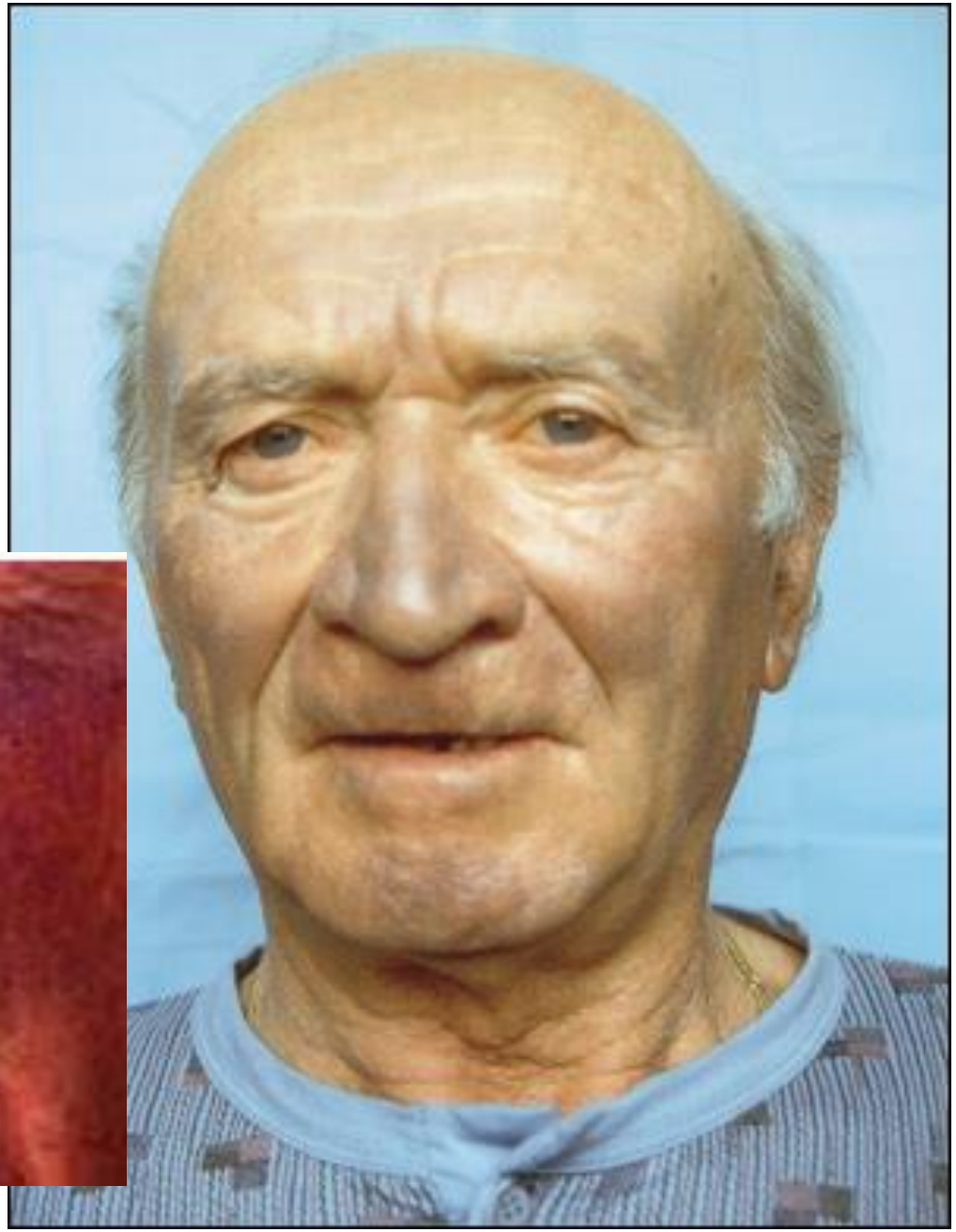




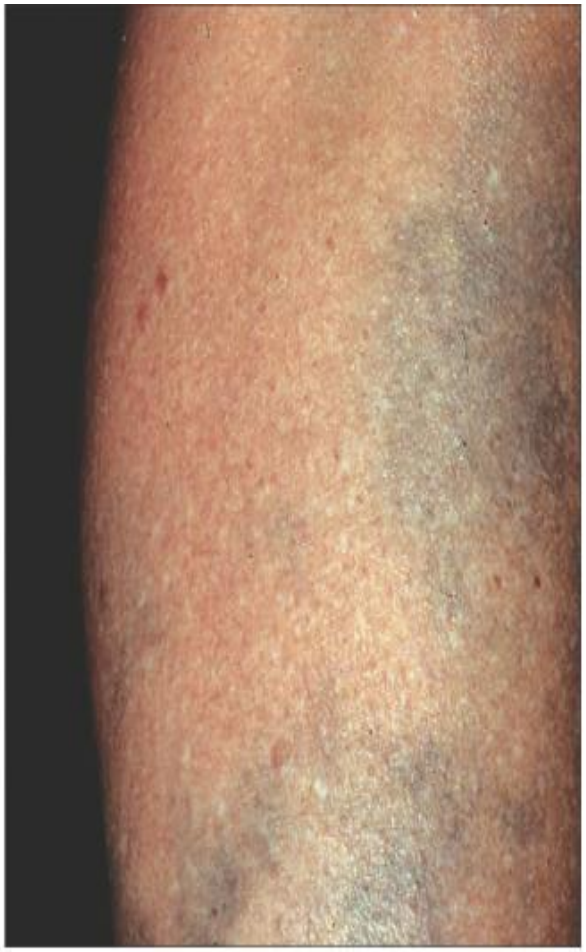
Amiodaronom

– induced pigmentation

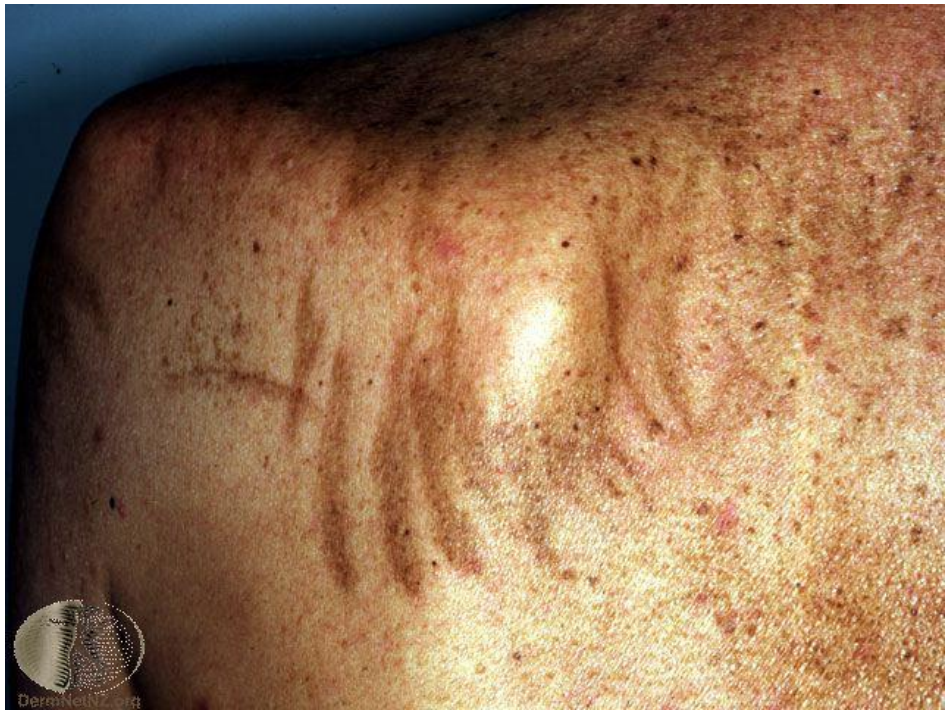


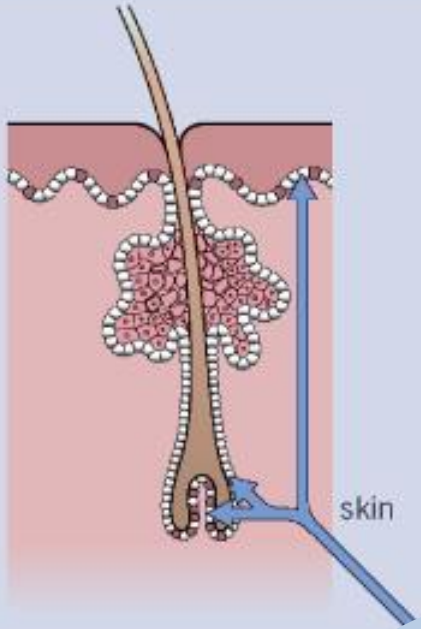


Minociklin



Bleomicin -flagellate erythema



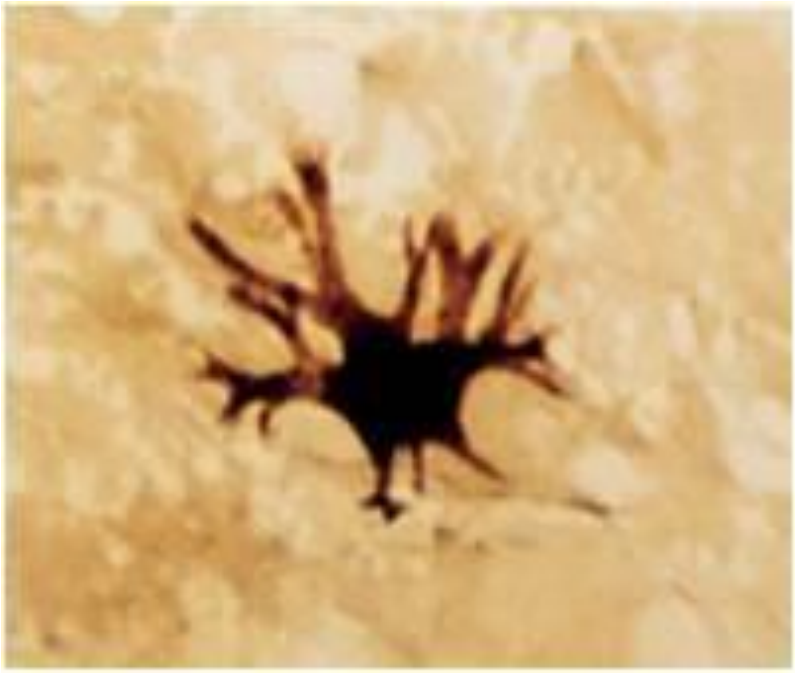


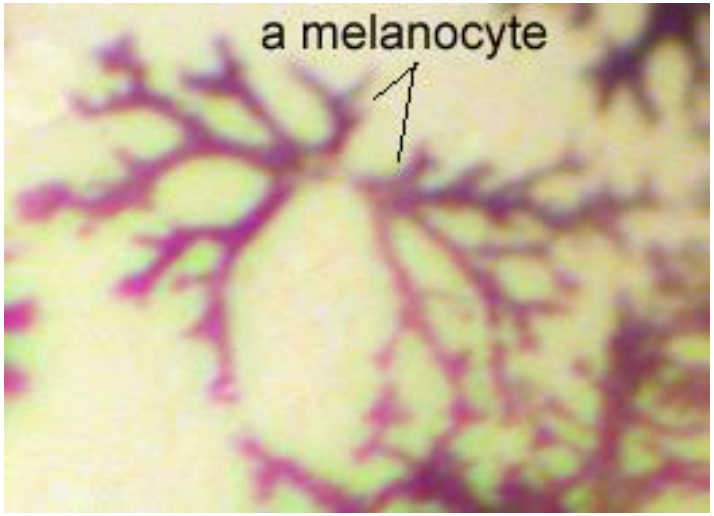
ral crest

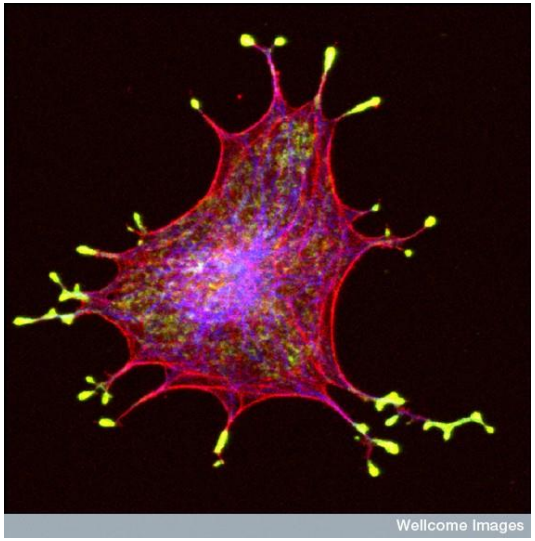
Embriogenesis

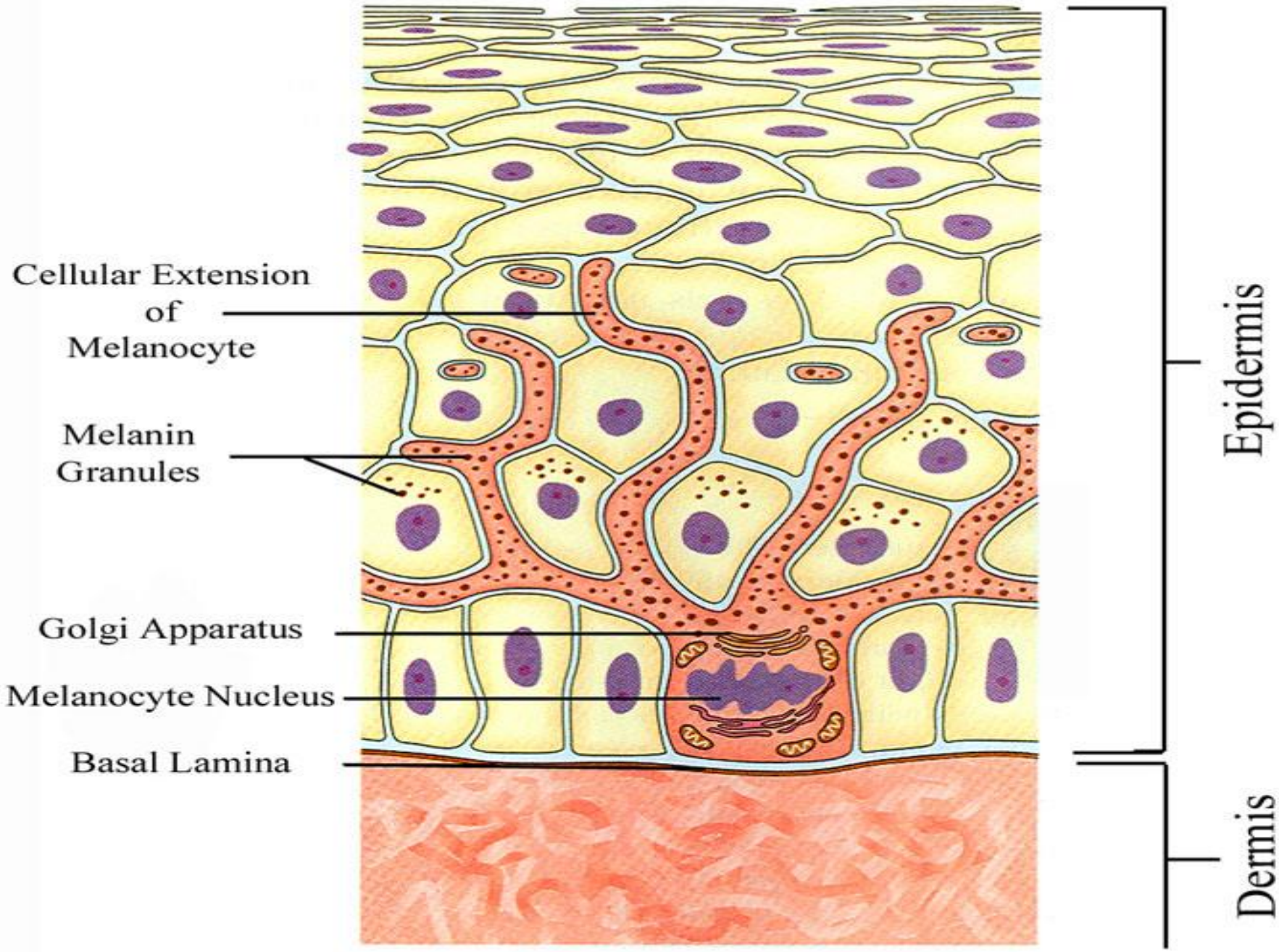
Where the human body
Is
melanin found?

Melanocit



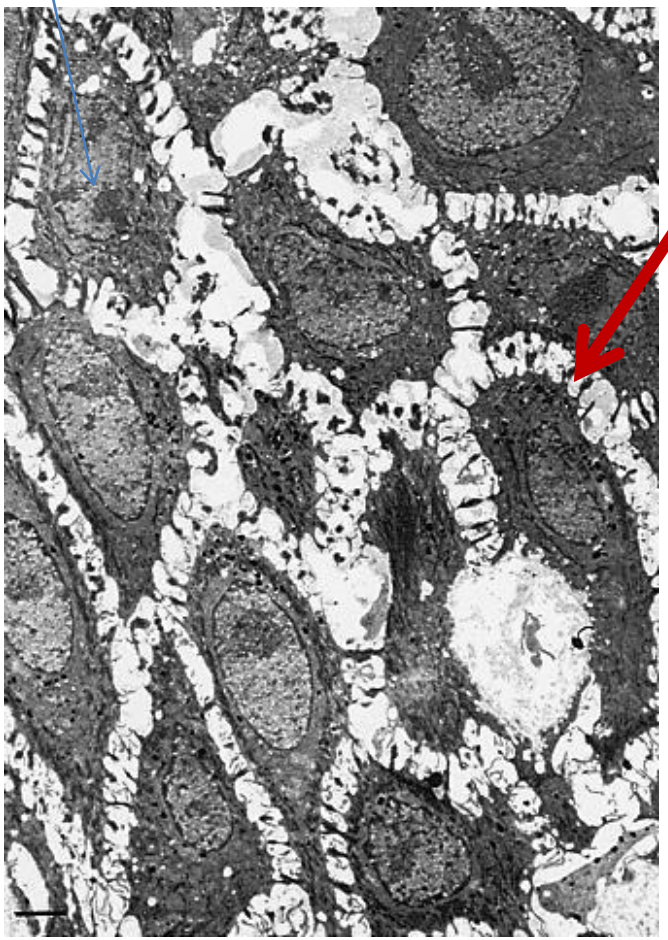


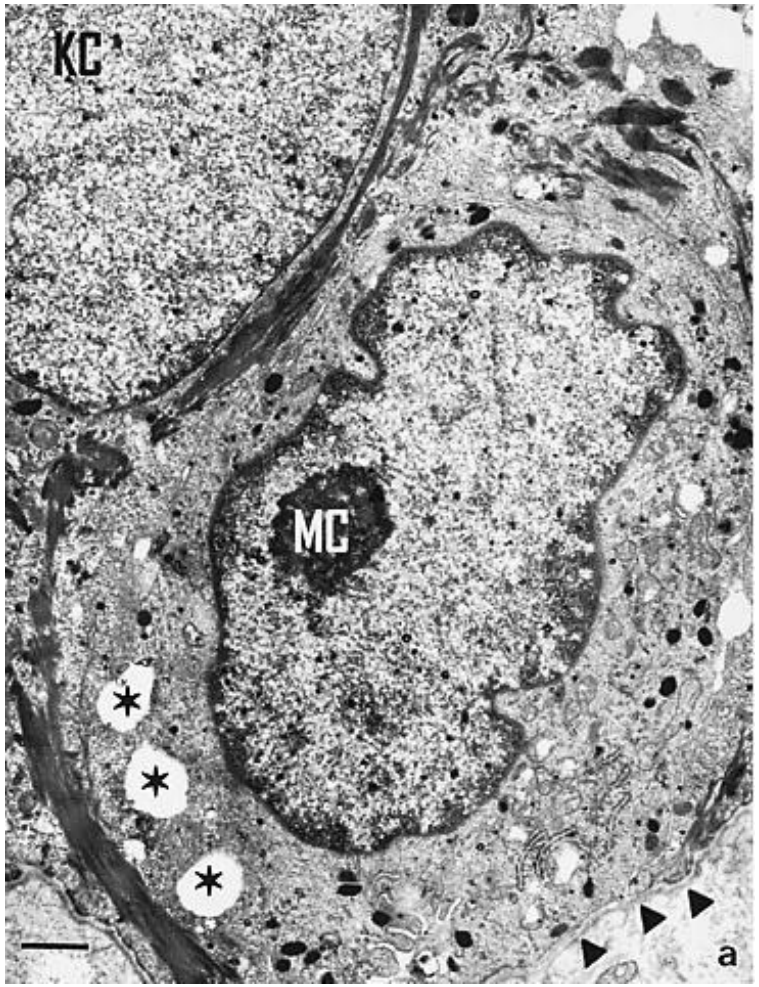



Cellular Extension of Melanocyte
Melanin Granules
Golgi Apparatus
Melanocyte Nucleus
Basal Lamina
Epidermis
Dermis

Keratinociti KC

desmosoms



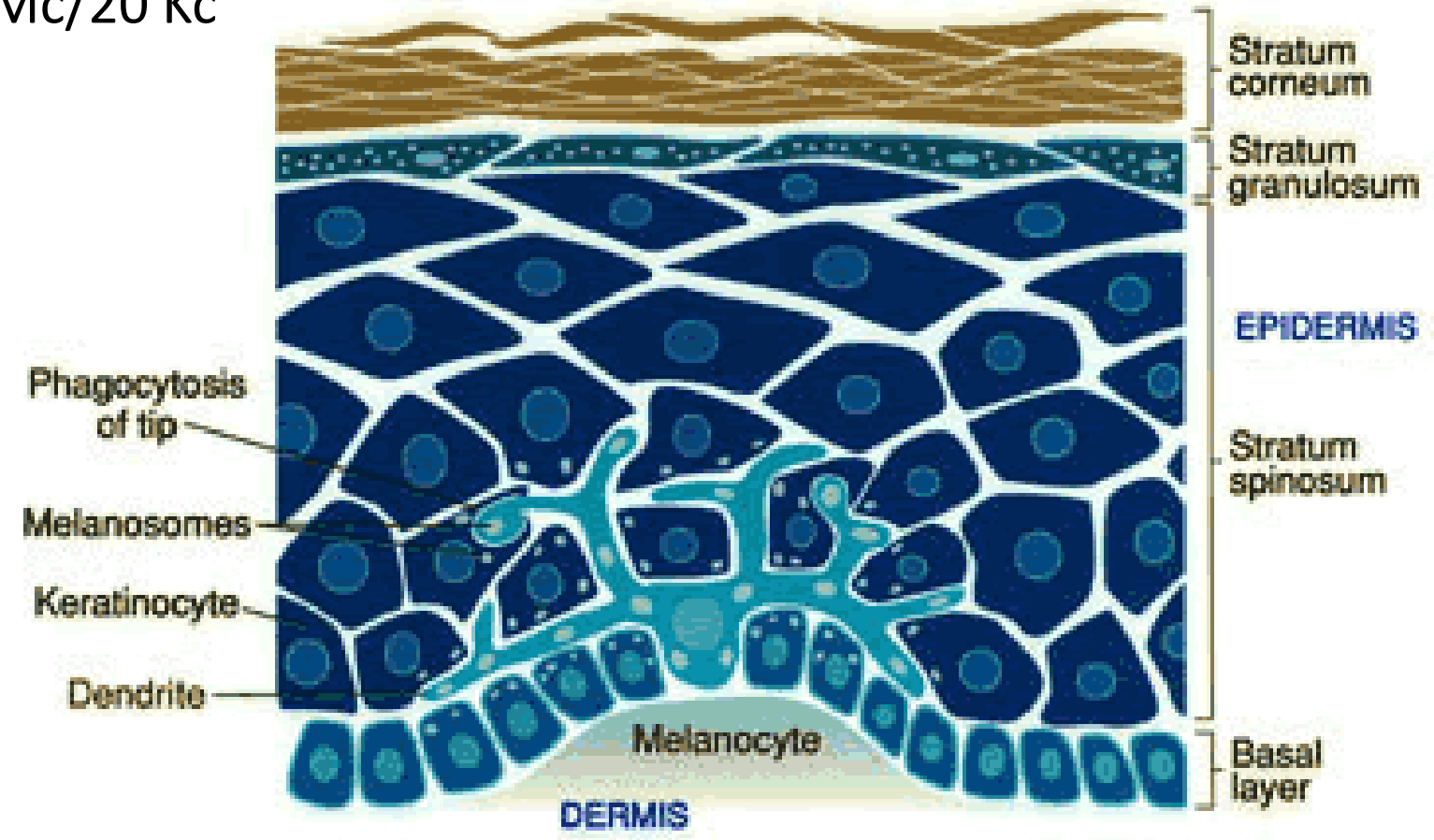


Who has more melanocytes ?

EPIDERMAL MELANOCYT UNIT

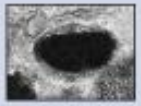
FACE:1Mc/5 Kc

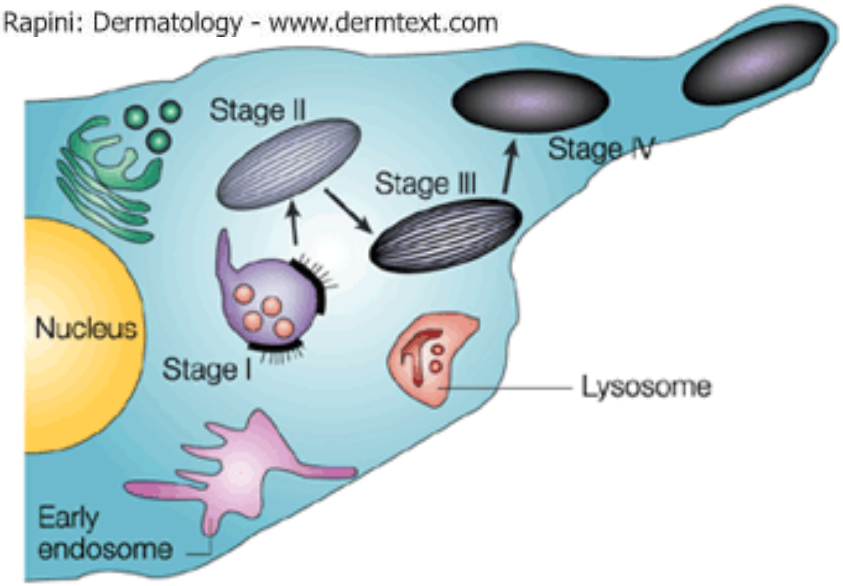
BACK: 1Mc/20 Kc

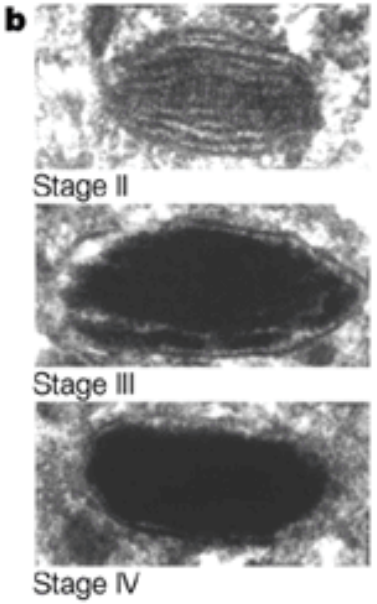


Melanosomes

DESCRIPTIONS AND ELECTRON PHOTOMICROGRAPHS OF THE FOUR MAJOR STAGES OF EUMELANIN MELANOSOMES

Stage	Description	Electron micrographs
I	Spherical; no melanin deposition	
II	Oval; obvious matrix in the form of parallel longitudinal filaments; minimal deposition of melanin; high tyrosinase activity	
III	Oval; moderate deposition of melanin; high tyrosinase activity	
IV	Oval; heavy deposition of melanin; electron-opaque; minimal tyrosinase activity	





Nature Reviews | Molecular Cell Biology

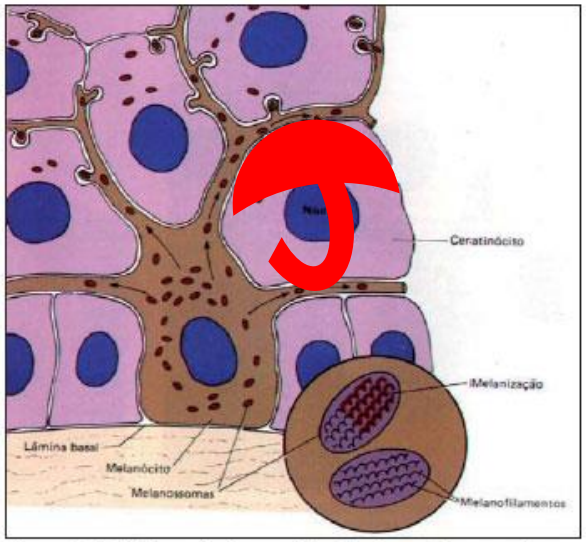


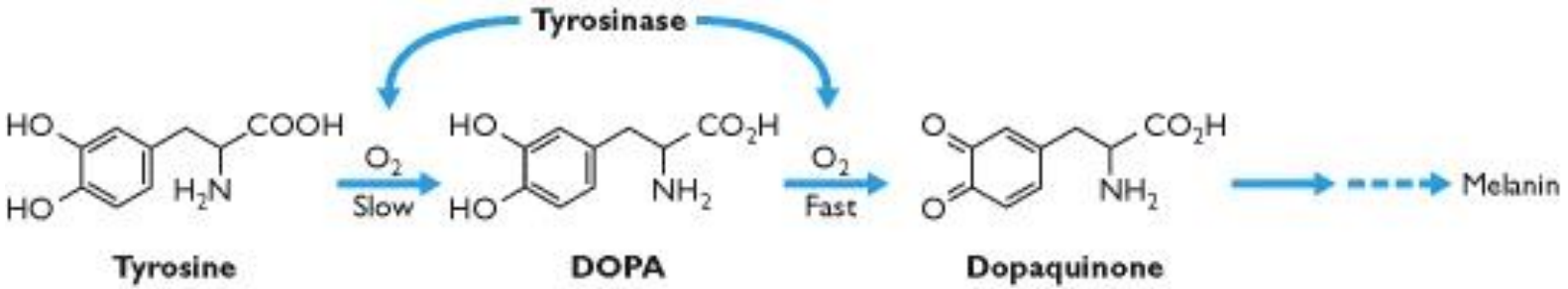
Figure 3: Melanin production and distribution pathway on the epidermis, through melanosomes

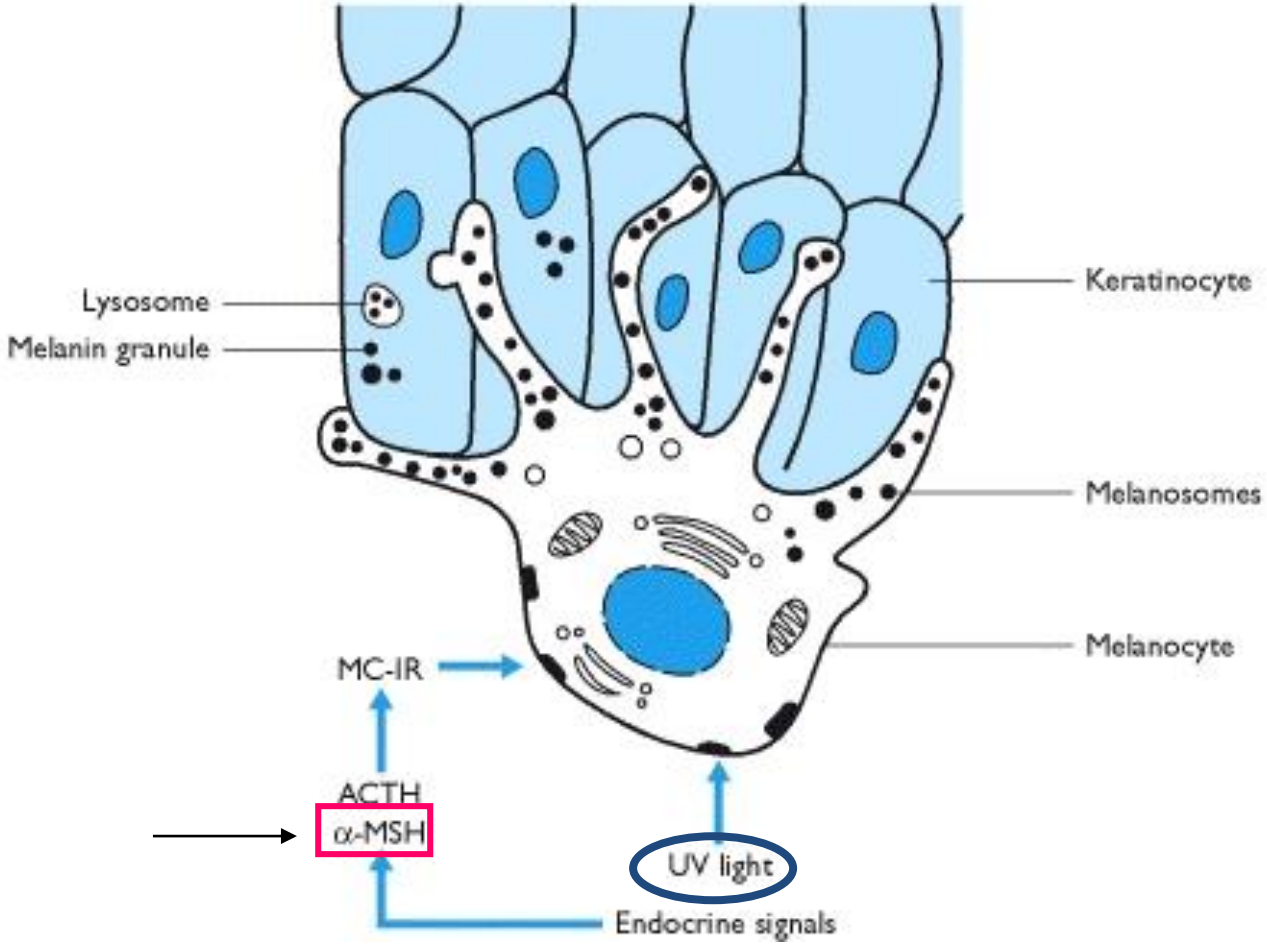
Table 65.2 Variation of types of melanosome within melanocytes and keratinocytes with level of cutaneous pigmentation.

VARIATION OF TYPES OF MELANOSOMES WITHIN MELANOCYTES AND KERATINOCYTES WITH LEVEL OF CUTANEOUS PIGMENTATION		
	Predominant melanosomal stages	
Pigmentation of skin	Melanocytes	Keratinocytes
Fair	II, III	Occasional III
Medium	II, III, IV	III, IV
Dark	IV > III	IV

VARIATION OF TYPES OF MELANOSOMES WITHIN MELANOCYTES AND KERATINOCYTES WITH LEVEL OF CUTANEOUS PIGMENTATION

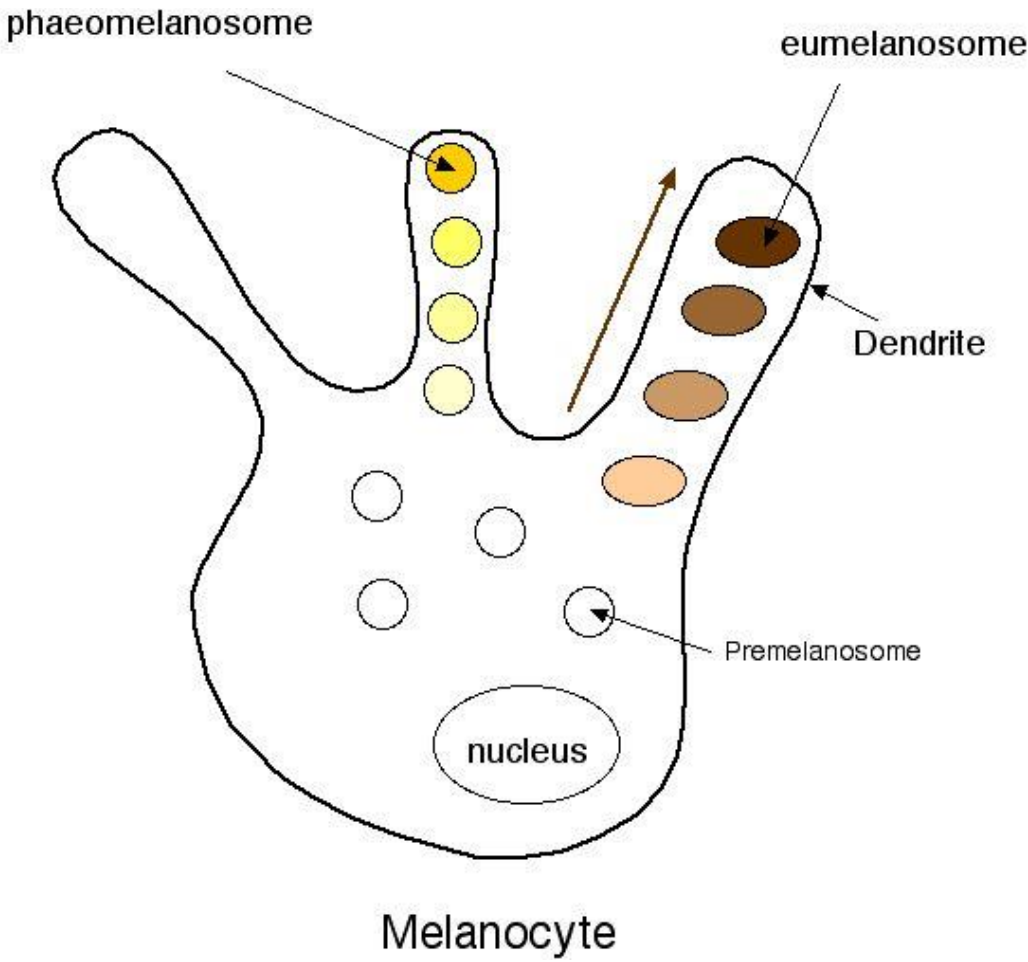
	Lightly pigmented skin	Darkly pigmented skin
Melanization	Stages II, III	Stage IV
Size (diameter)	0.3–0.5 mm	0.5–0.8 mm
Number per cell	<20	>200
Distribution of keratinocytes within the lysosomes	Groups of 2–10	Single
Degradation	Fast	Slow


Tyrosinase
HO
COOH
HO
H_2N
O_2
Slow
HO
CO_2H
HO
NH_2
O_2
Fast
O
CO_2H
O
NH_2
Melanin
Tyrosine
DOPA
Dopaquinone


Lysosome
Melanin granule
Keratinocyte
Melanosomes
Melanocyte
MC-IR
ACTH
α-MSH
UV light
Endocrine signals

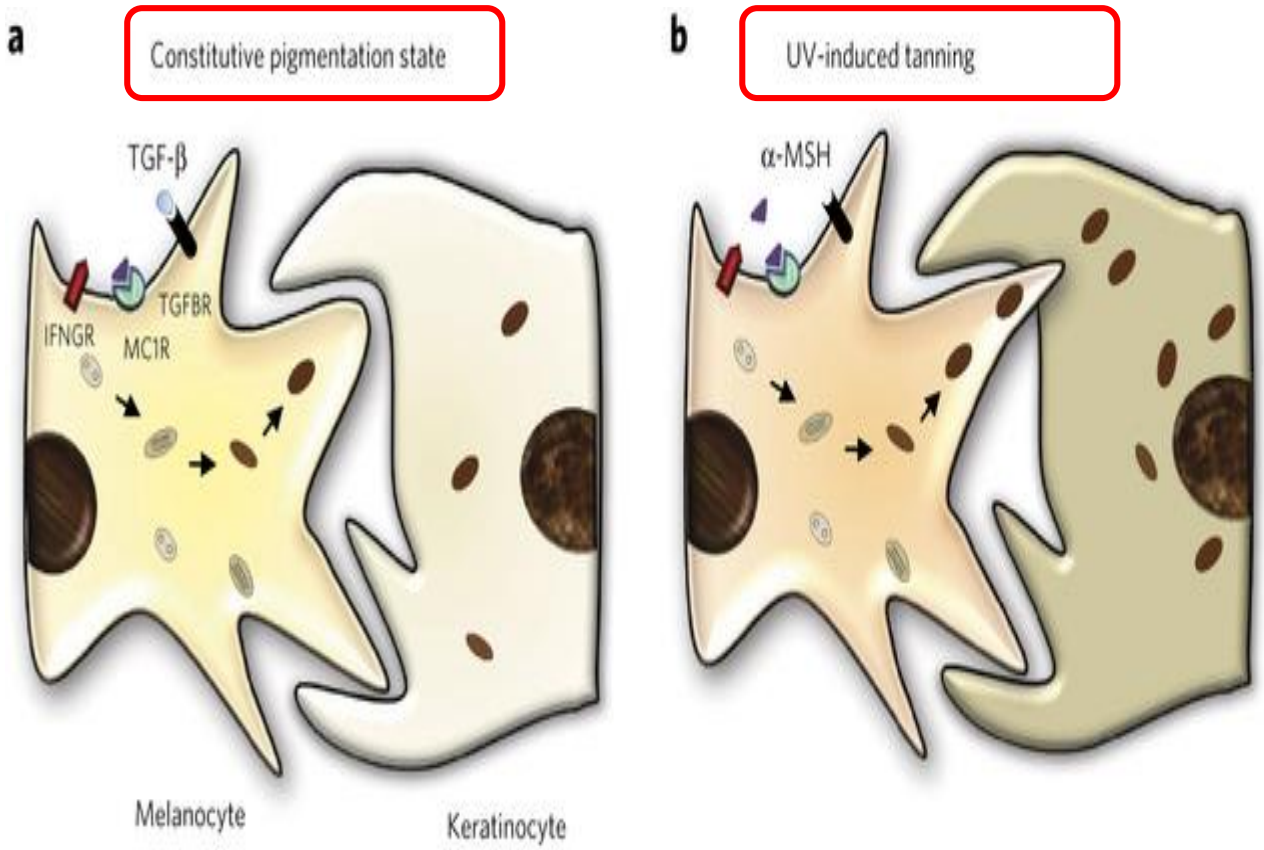
	African	Oriental	Caucasian
Type of Melanin Mixture	Pheomelanine Eumelanine	Pheomelanine Eumelanine	Pheomelanine Eumelanine
Proportion of Free Melanin Grains in the Epidermis	Complexed Free	Complexed Free	Complexed Free
Melanine Grain Morphology			
Melanine Grain Size (nm)	1 x 0.5	0.6 x 0.3	0.5 x 0.3

Table 1.



Melanocyte

Constitutive and facultative pigmentation

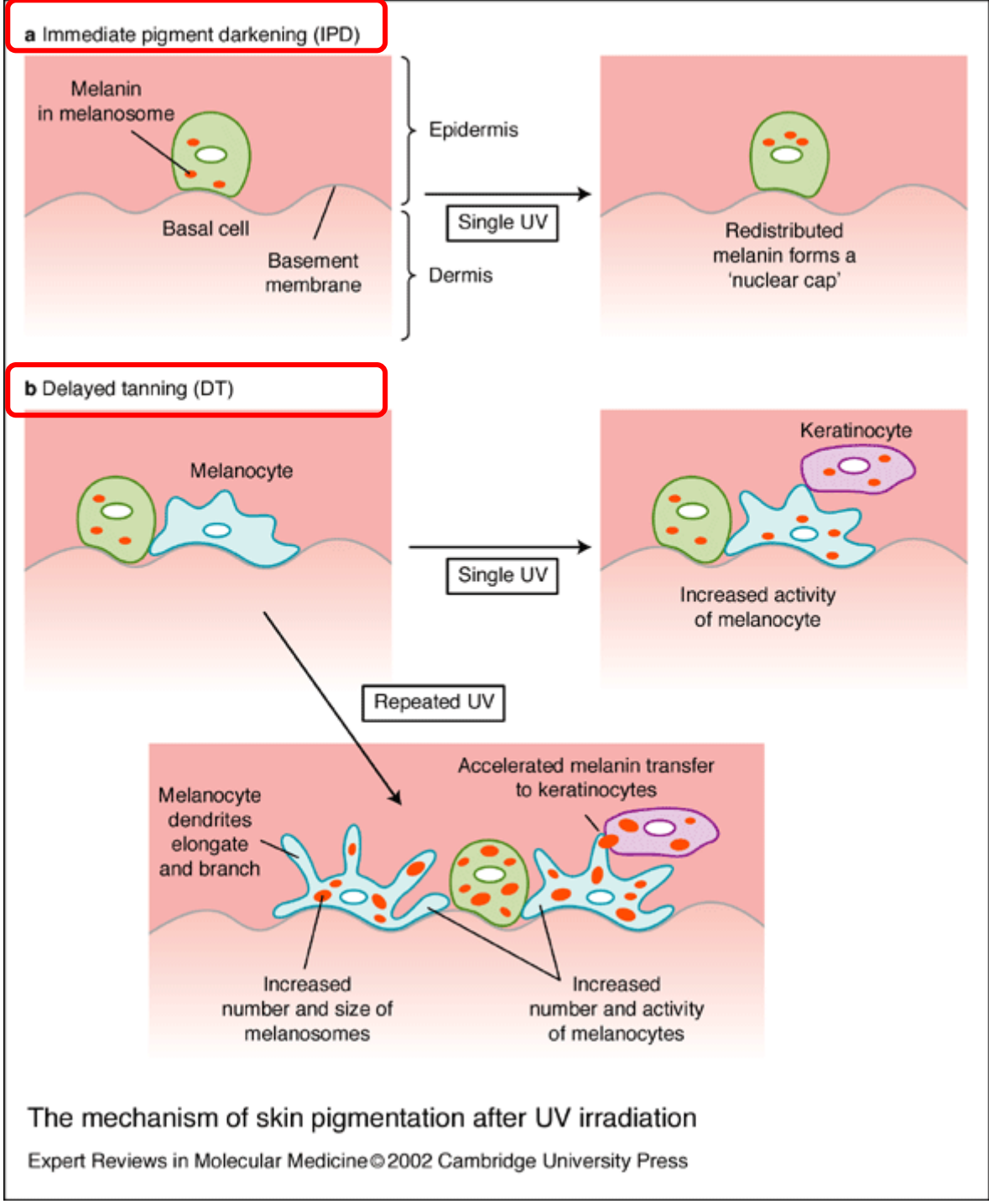


FITZPATRICK TIP KOŽE

Fitzpatrick Classification of Sun Reactive Skin Types

Skin Type	Color	Reaction to the Sun
I	White	Always burns, never tans
II	White	Usually burns, tans with difficulty
III	White	Sometimes burns, but tans easily
IV	Moderate Brown	Rarely burns, tans very easily
V	Dark Brown	Very rarely burns, most often tans
VI	Black	Never burns, always tans

I II III IV V VI

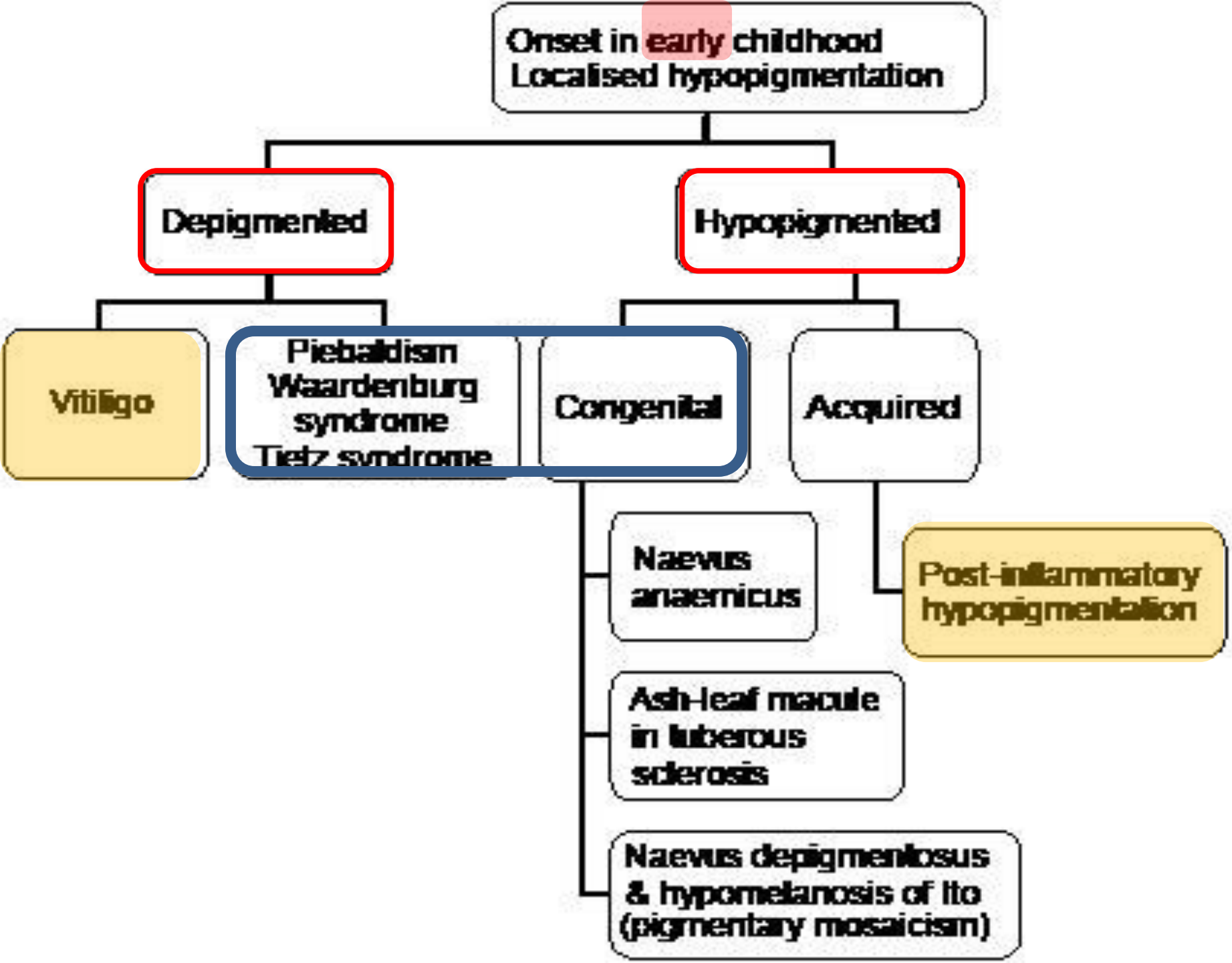


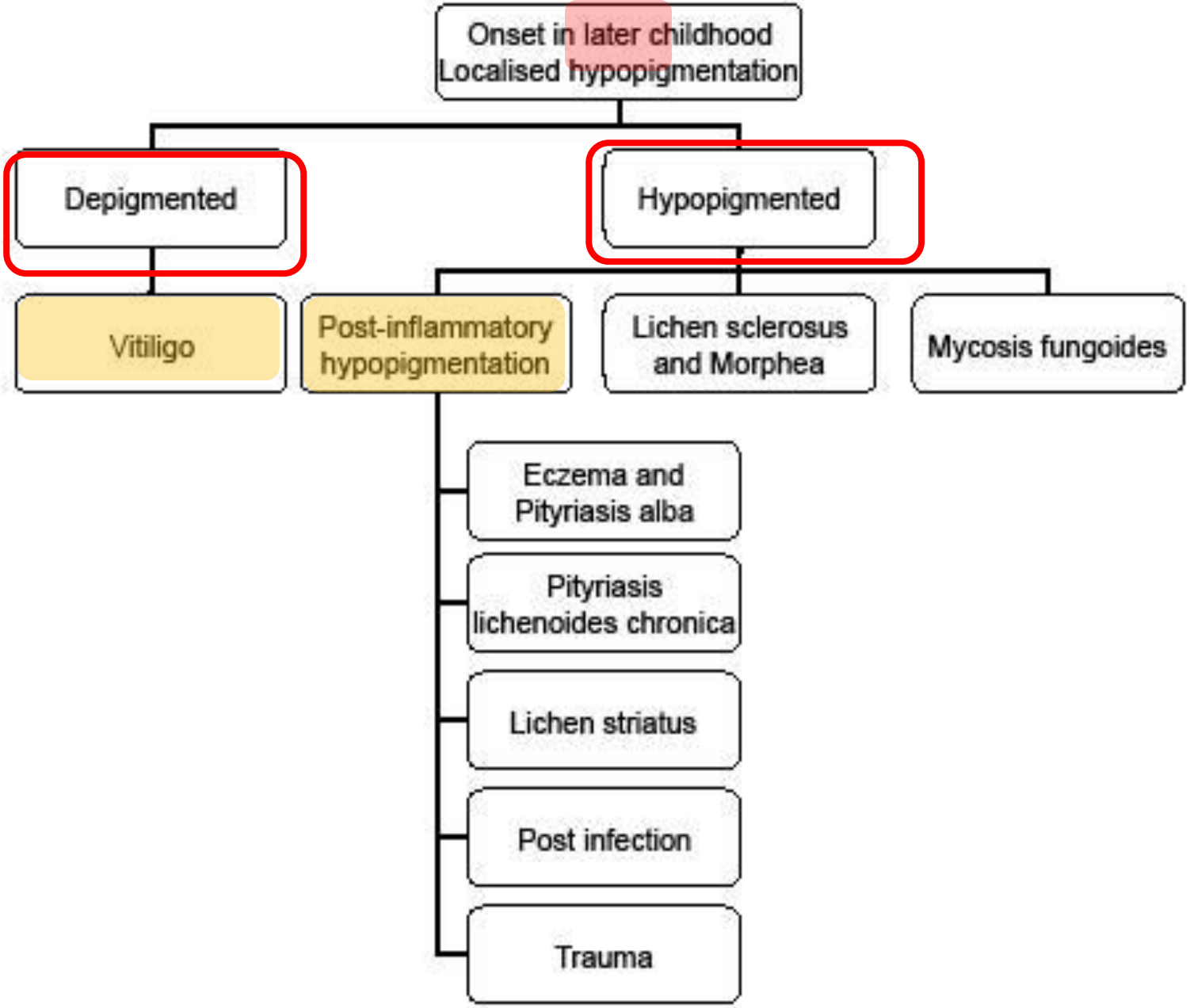
The mechanism of skin pigmentation after UV irradiation

Hypopigmentations

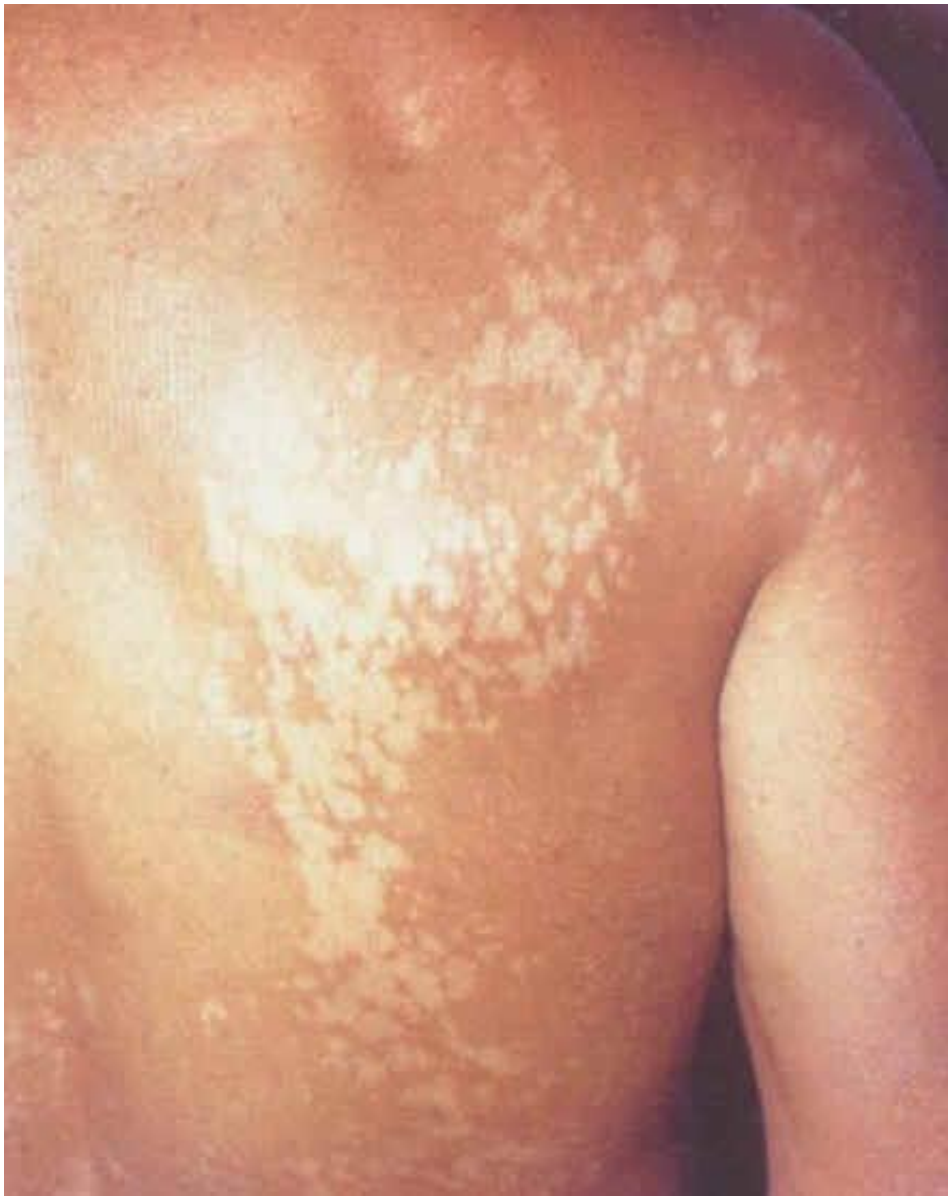
localized

difuse


Onset in early childhood
Localised hypopigmentation
Depigmented
Hypopigmented
Vitiligo
Piebaldism
Waardenburg syndrome
Tietz syndrome
Congenital
Acquired
Naevus anaemicus
Post-inflammatory hypopigmentation
Ash-leaf macule in tuberous sclerosis
Naevus depigmentosus & hypomelanosis of Ito (pigmentary mosaicism)


Onset in later childhood
Localised hypopigmentation
Depigmented
Hypopigmented
Vitiligo
Post-inflammatory hypopigmentation
Lichen sclerosus and Morphea
Mycosis fungoides
Eczema and Pityriasis alba
Pityriasis lichenoides chronica
Lichen striatus
Post infection
Trauma

CASE 1.



Man, 32 y old.
On the beach, he noticed a white macules on the back and they are spreading
He is healthy. Mother has hypothyreosis.

His grandfather
had white patches on the body.

Hypo –pigmentation
or
De-pigmentation?

TABLE Differential diagnosis for hypopigmentation

Hypopigmented skin lesions	Age at onset	Diagnosis
Diffuse pigmentary dilution	Birth or infancy	Angelman syndrome, Hermansky-Pudlak syndrome, oculocutaneous albinism, Chédiak-Higashi syndrome, Griscelli syndrome
Circumscribed areas of hypopigmentation	Birth or infancy	Vitiligo, piebaldism, Waardenburg syndrome, tuberous sclerosis, Blaschkoid hypopigmentation, nevus depigmentosus
Circumscribed hypopigmentation	Childhood	Vitiligo, pityriasis alba, atopic dermatitis, lichen striatus, pityriasis lichenoides chronica, lichen sclerosus, postinfectious hypopigmentation (ie, following varicella infection), posttraumatic (ie, cryotherapy or thermal burn)

From: Orlow SJ[1]; Bolognia JL[2]; Spritz RA[3]; Józwiak S, et al[4]; Loomis CA[5]; Ehrenreich M, et al.[6]

Pityriasis versicolor

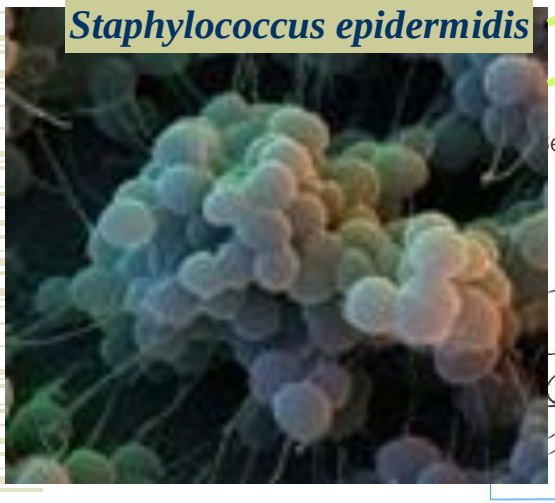
Malassezia furfur

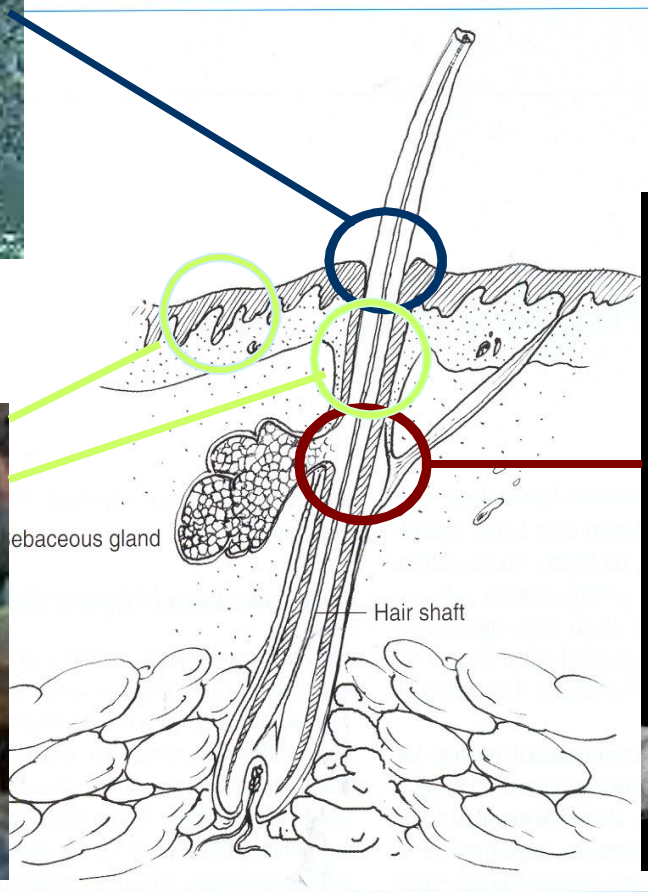
Wood lampa
Yellow florescense

Normalna fiziološka flora kože

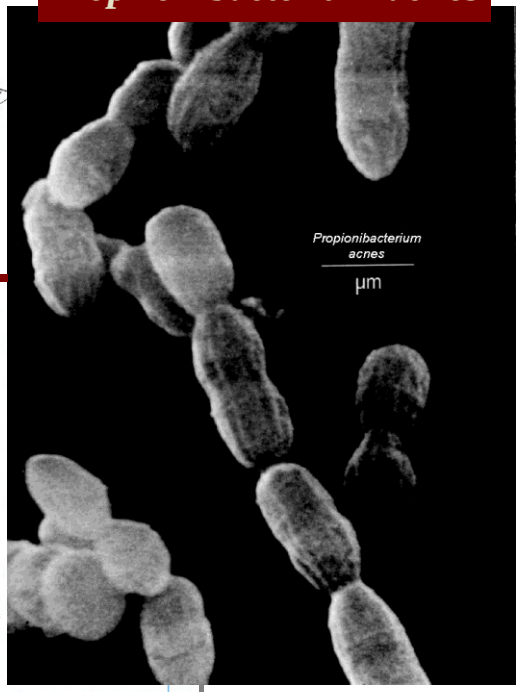
Malassezia furfur

Staphylococcus epidermidis

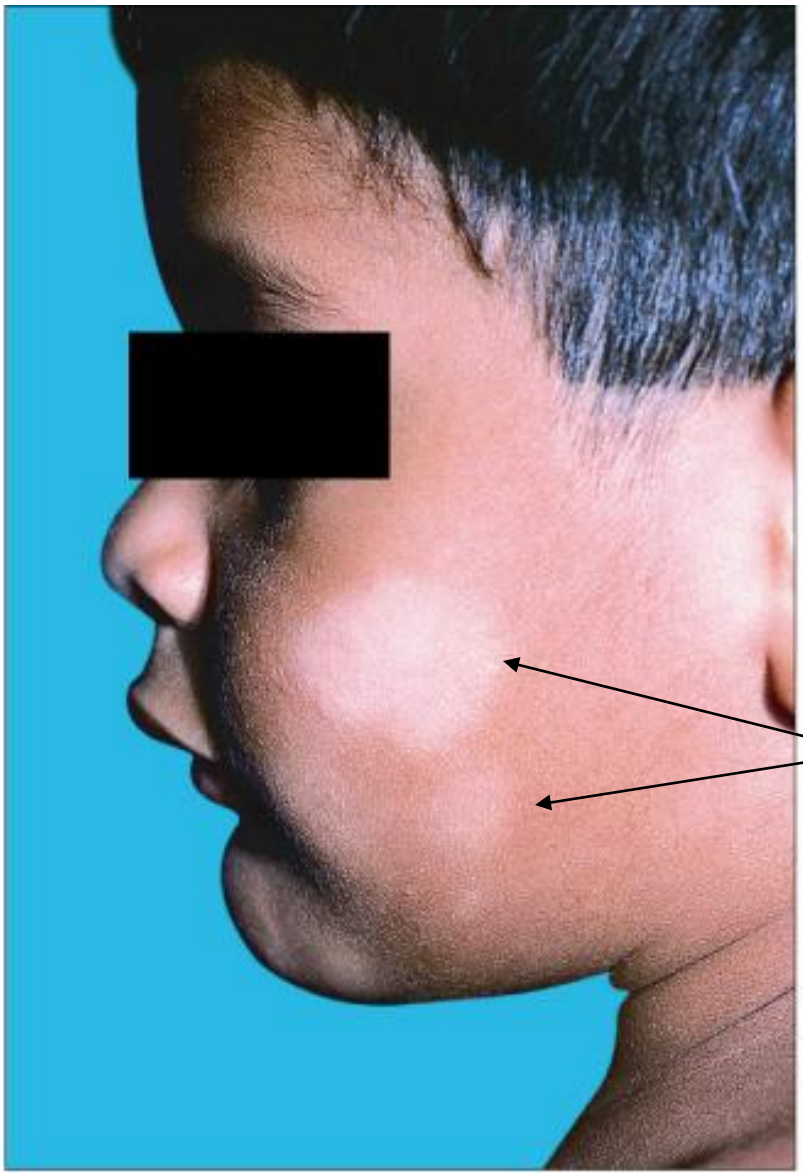




Proprionibacterium acnes



Pityriasis alba



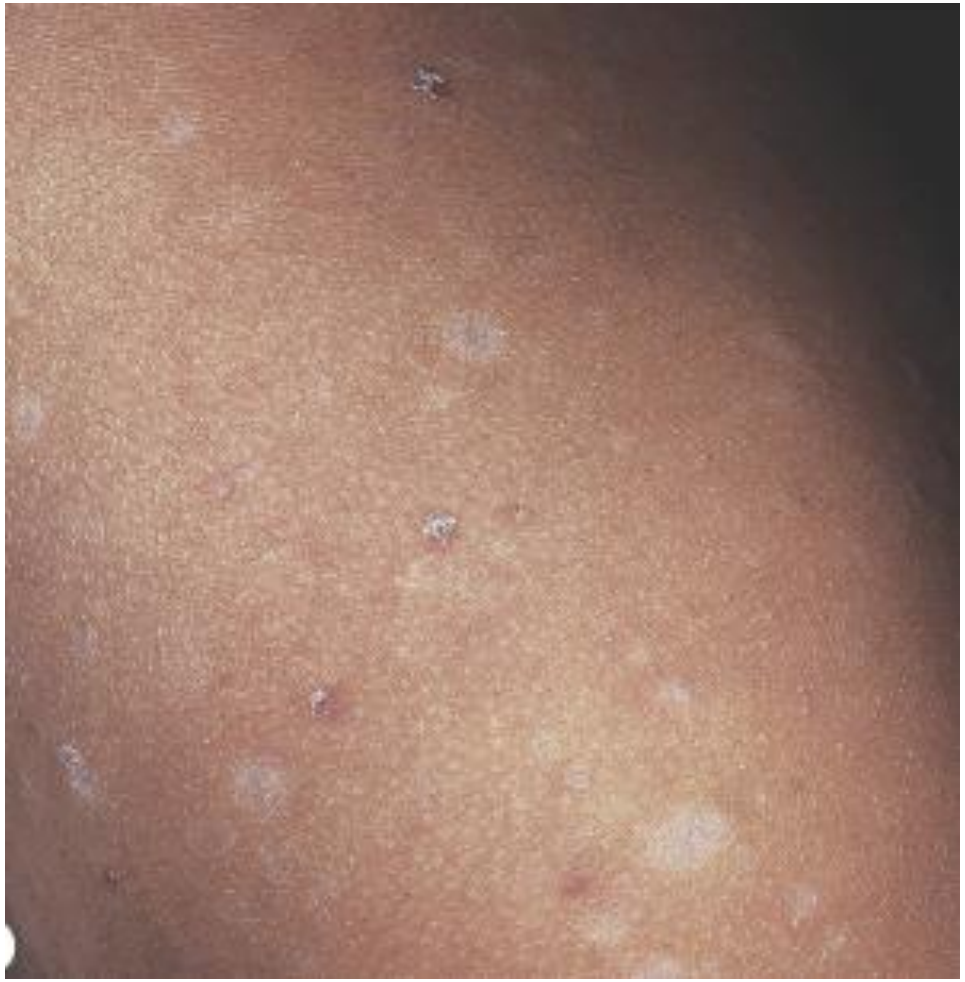
Post-inflammatory hypopigmentation

Children

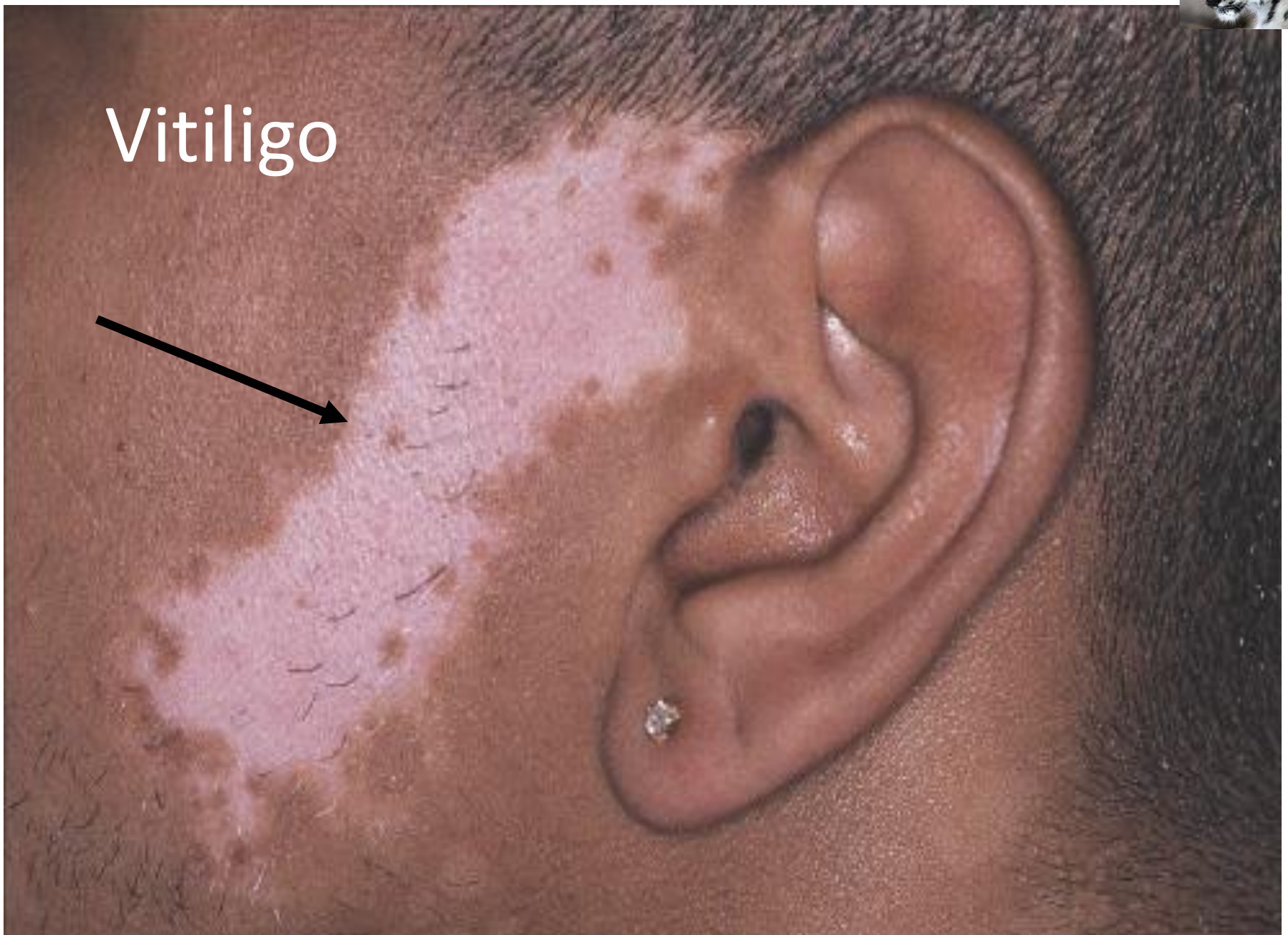
Often on the face, upper arms

Atopic dermatitis (32%)

Postinflammatory hypopigmentation



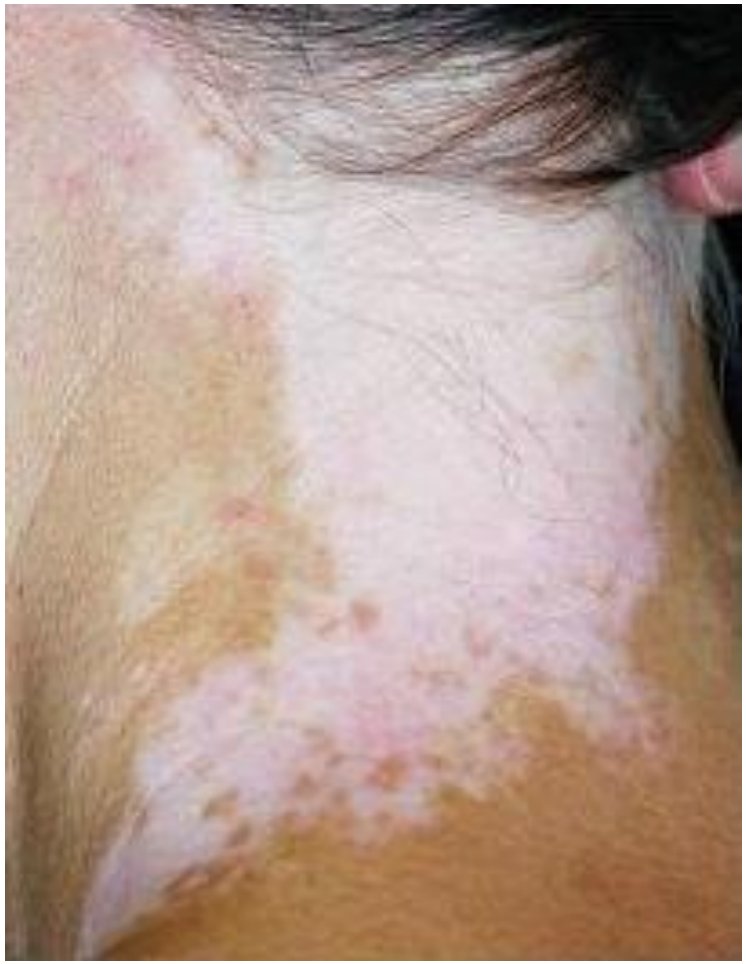
Vitiligo

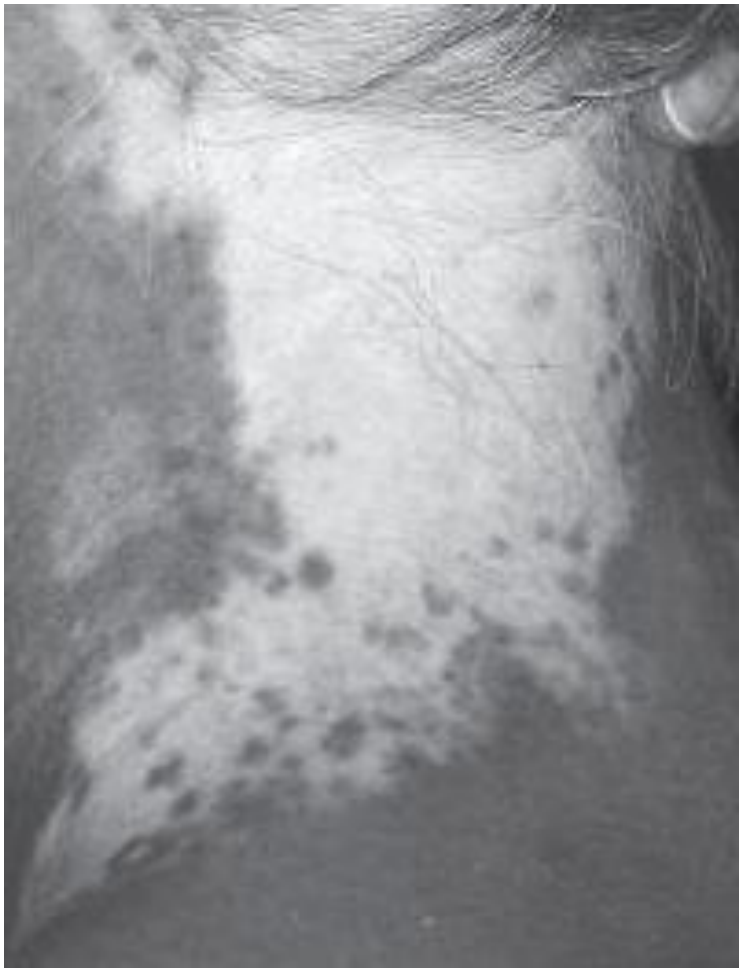


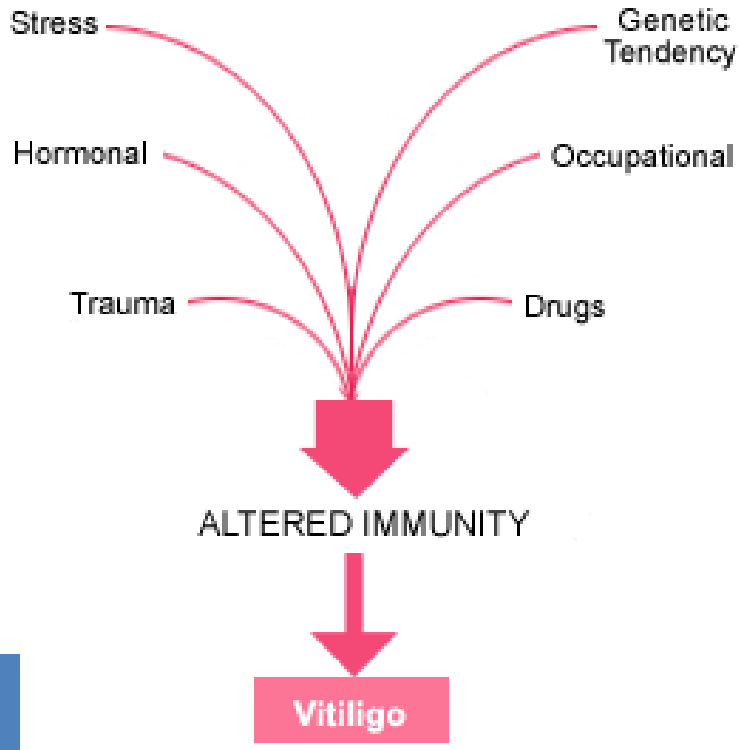
Leukotrichia (10-60%)

Wood lampa

Wood lampa







Major Theories

- Auto-immnne
- Neural

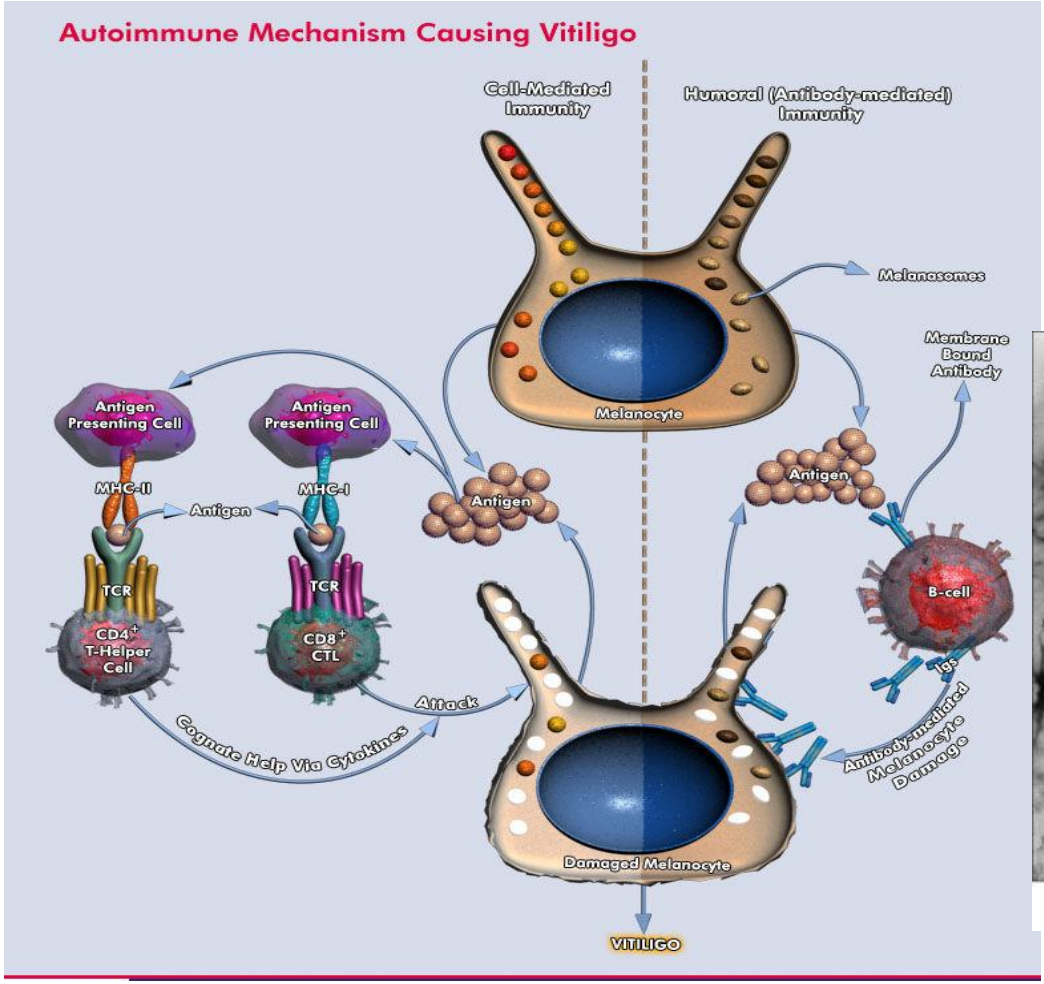
peripheral nerve endings may secrete a substance that is cytotoxic to melanocytes and causes their destruction

- Autocytotoxic

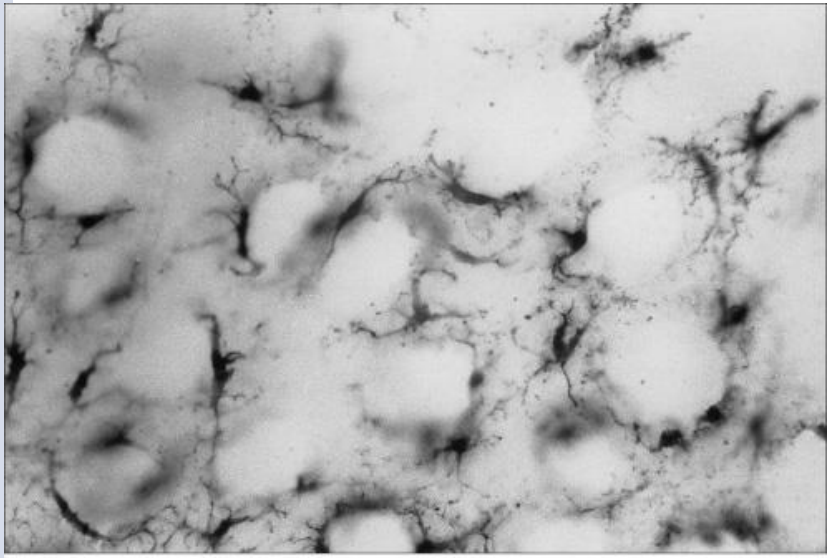
intracellular pre-melanogenesis metabolites are toxic to melanocytes, such as dopa and dopachrome.

- Growth factor defect

cells exhibited "defective growth and passage capacities." The researchers then noted that the growth defects of the melanocytes were partially corrected by adding a growth factor



loss of functional melanocytes



Epidermal melanocytes
"coloured " with **L-DOPA**

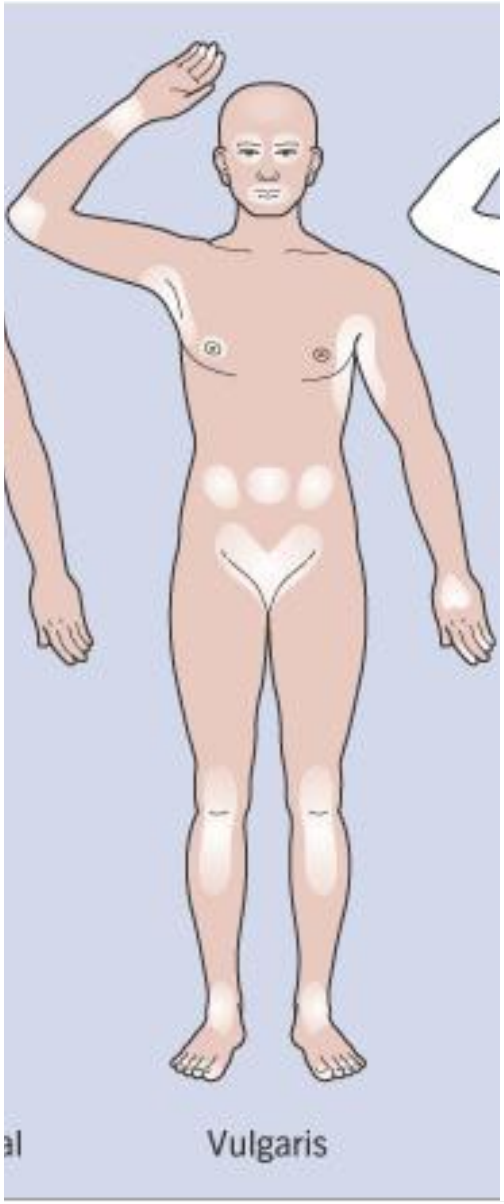
1.vitiligo appears in conjunction with several other autoimmune disorders,
(juvenile DM,Addison's disease,

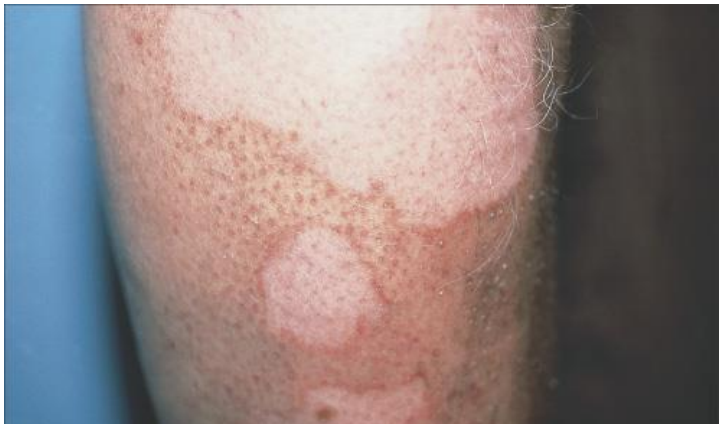
pernicious anemia),

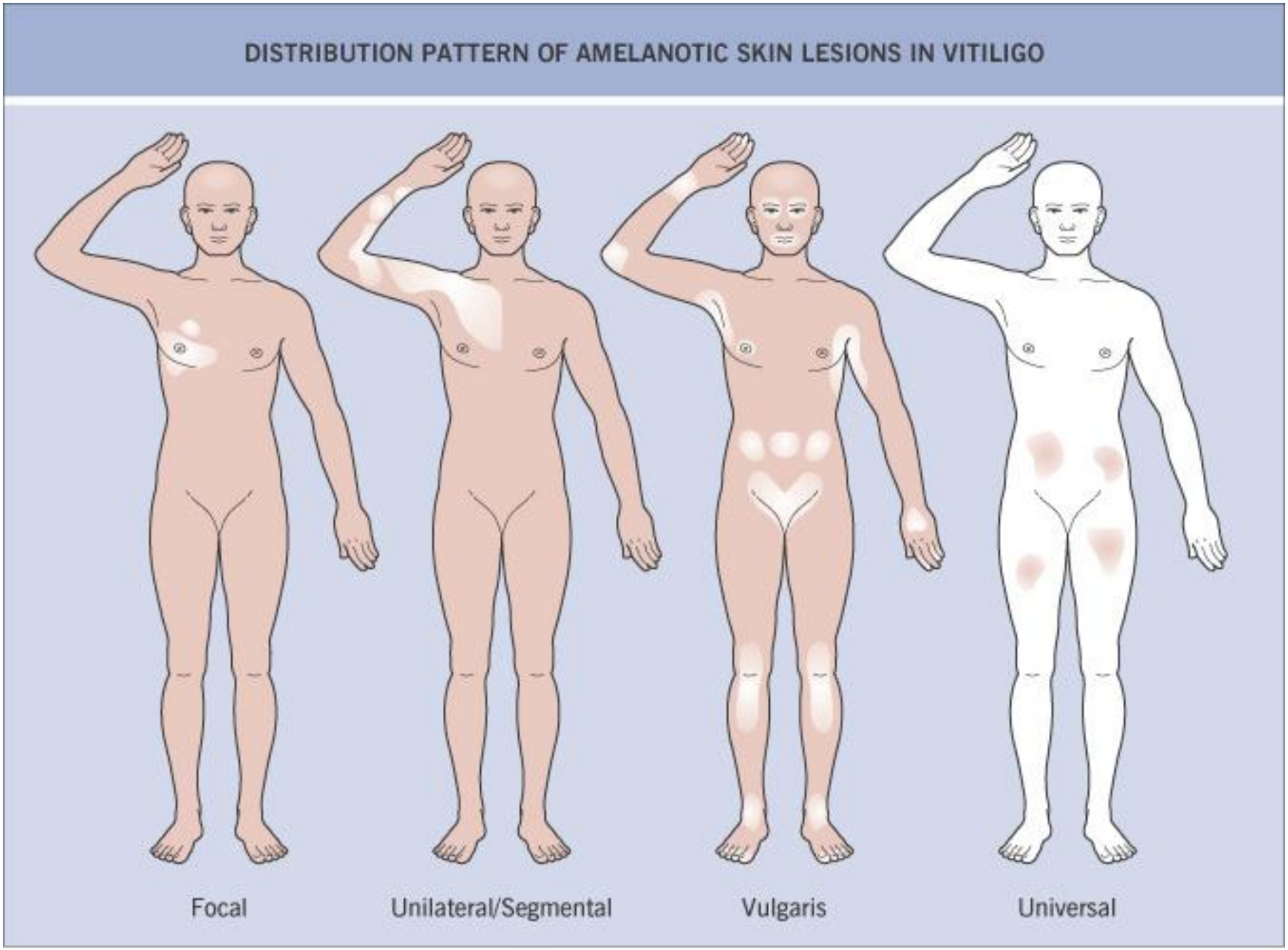
2. organ-specific antibodies (tirozinazu i TYRP-1 i 2)
can often be seen in patients with vitiligo.

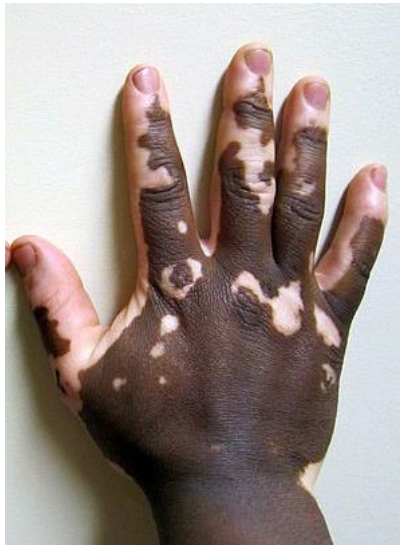
1. Places that are normally
   hyperpigmented
   (face, breast, axillary, sacrum, inguinal, anogenital)
2. Places exposed to trauma
   (Koebner phenomenon)
   (elbows, knees, dorsum of the hand)

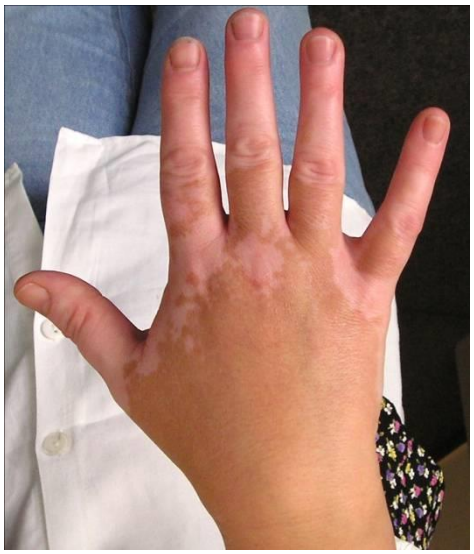
Provoking factors:
trauma,
sunburns,
association with autoimmune disease






DISTRIBUTION PATTERN OF AMELANOTIC SKIN LESIONS IN VITILIGO
Focal
Unilateral/Segmental
Vulgaris
Universal





No therapy at all !

Therapy

1. Medical

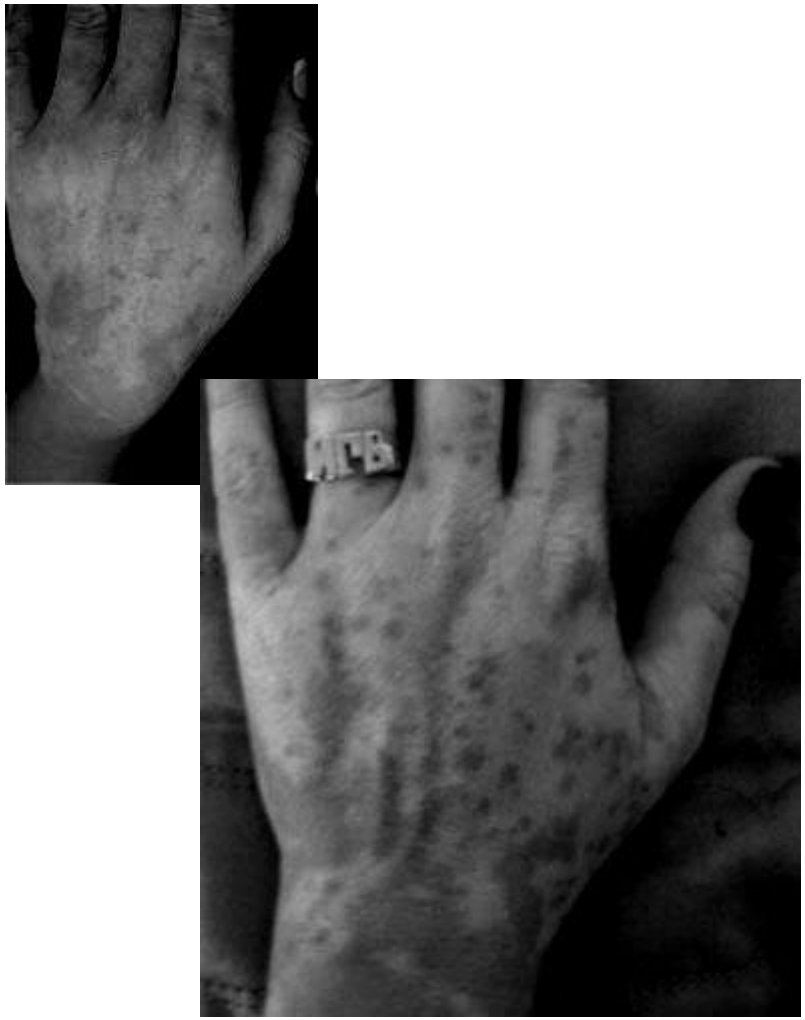
Early , and potent KS

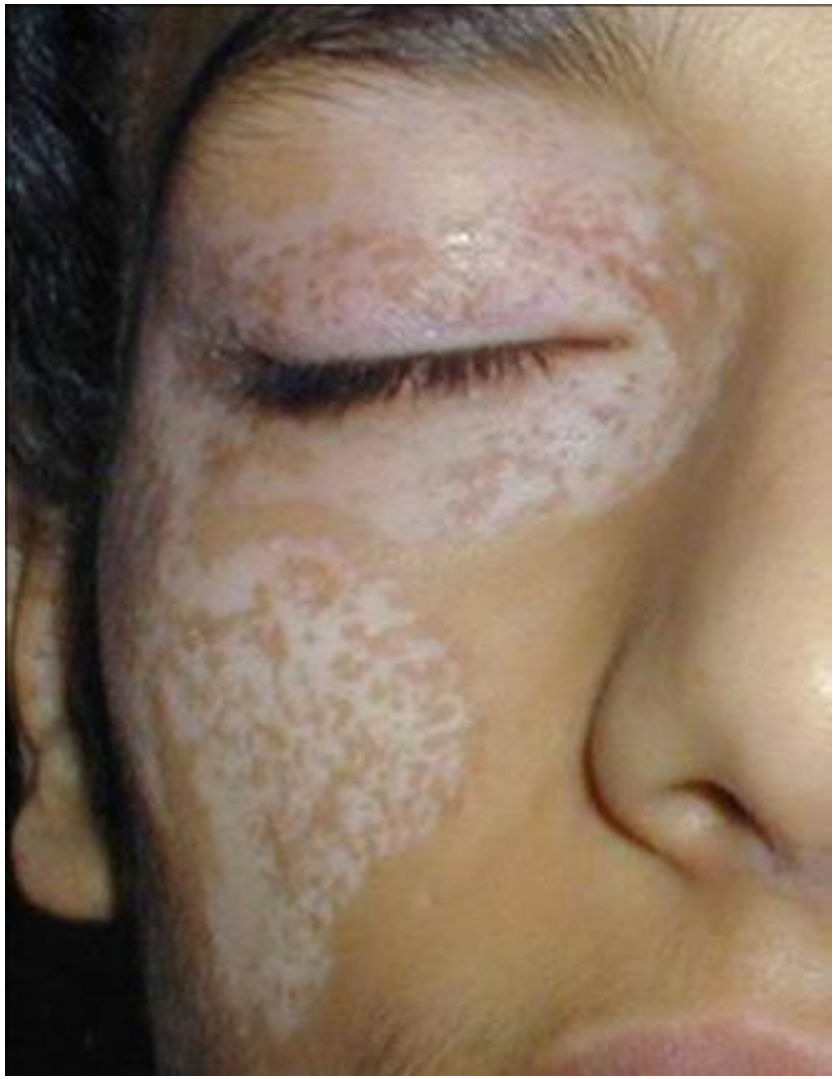
   a. Topical
      - Corticosteroids
      - Tacrolimus/pimecrolimus
      - Calcipotriol
      - Pseudocatalase
      - Combination

   b. Systemic
      - Corticosteroids (OMP with betamethasone/ methylprednisolone)

2. Phototherapy

- Topical PUVA
- NB-UVB
- Systemic PUVA (>12 years)
- Phenylalanine + PUVA
- Excimer laser (308 nm)/targeted NB-UVB phototherapy





NB-UVB

Surgical therapy – an option for vitiligo?

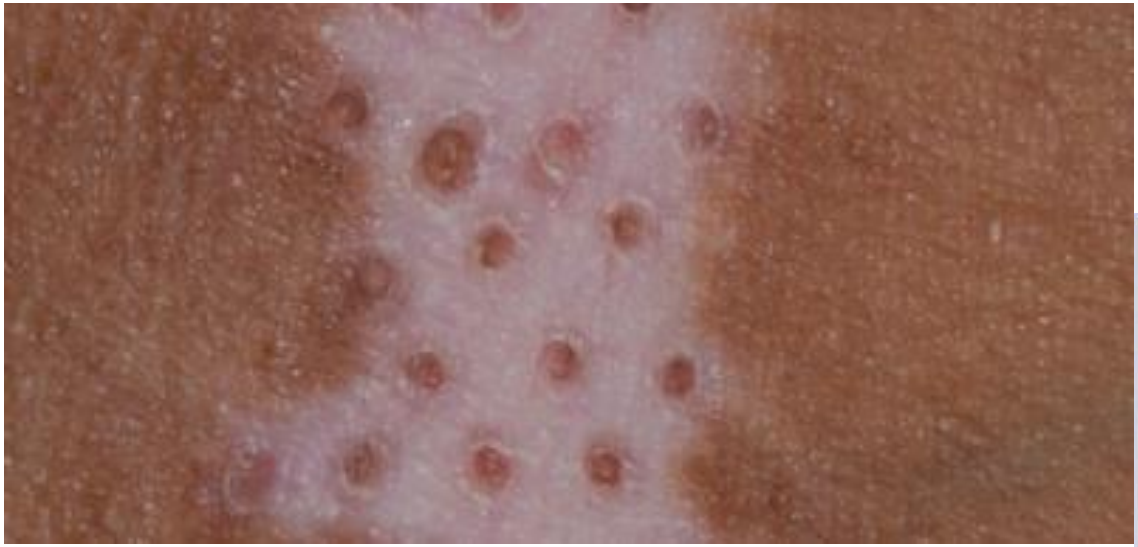
3. Surgical therapy

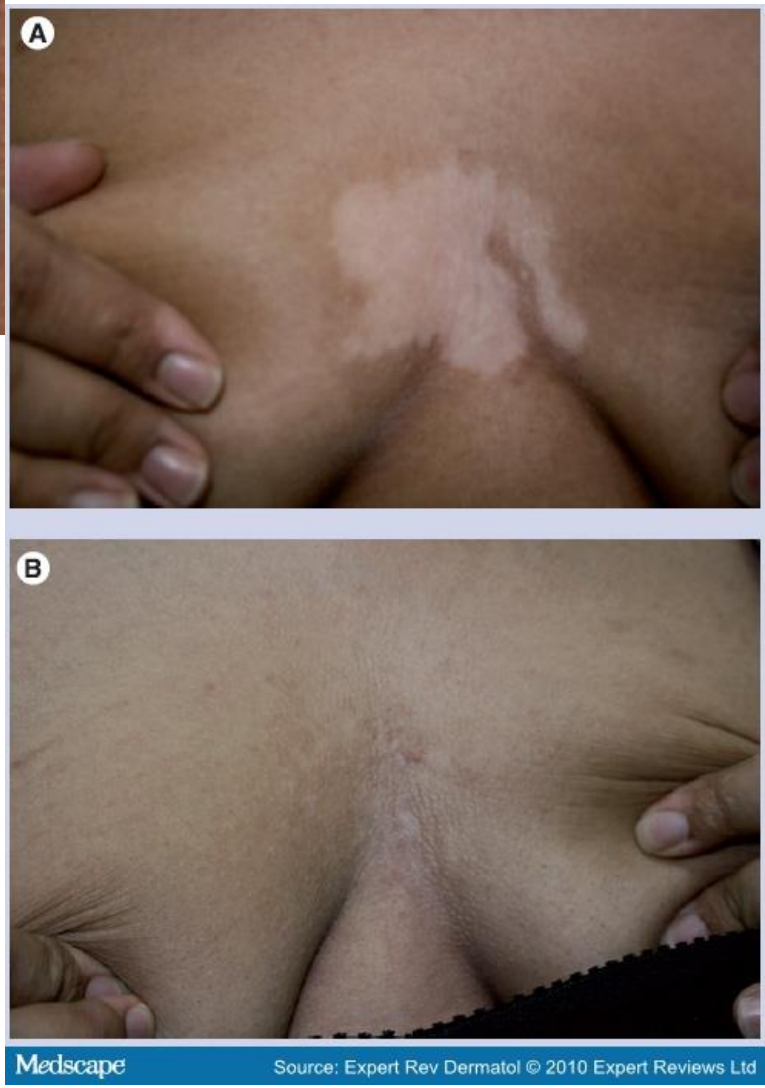
Conventional

- Mini-punch graft
- Suction blister epidermal graft
- Thin Thiersch graft

Newer cellular transplantation techniques

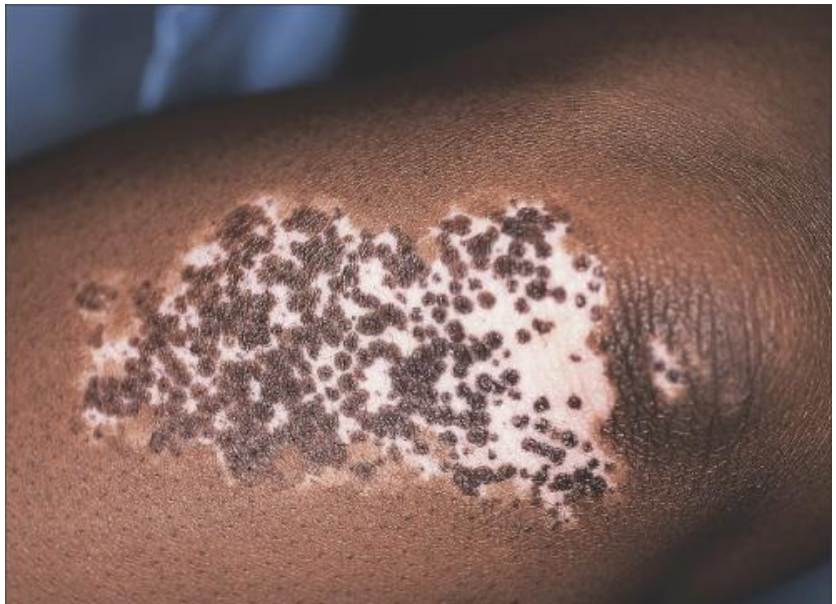
- Epidermal cell suspension
- Cultured melanocyte suspension
- Cultured epidermis





Split-skin graft, punch graft, blister grafting

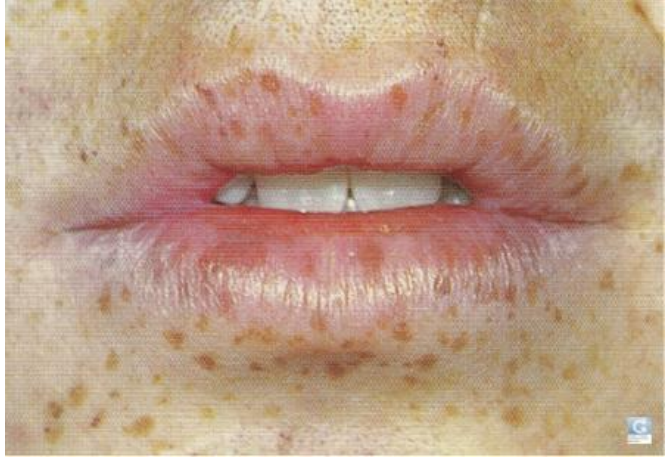
Melanocyte transplantation

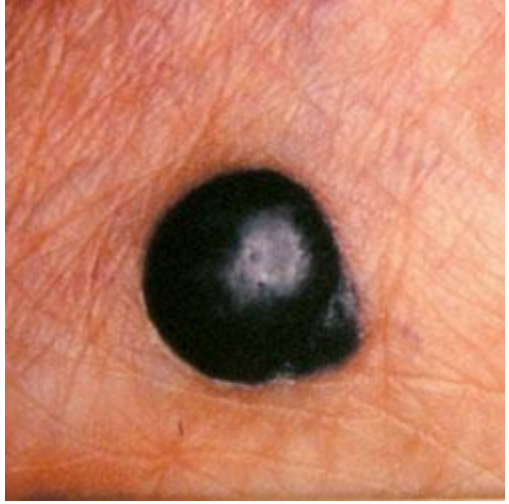


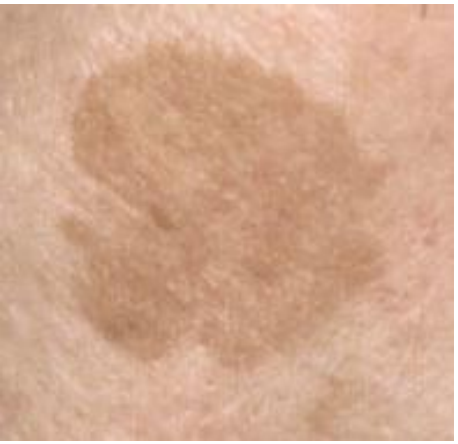
4. Cosmetic camouflage

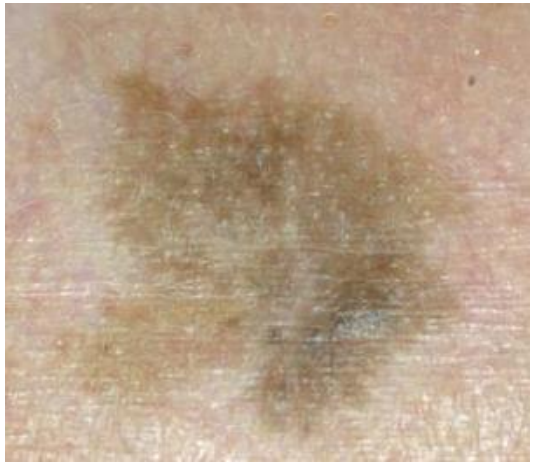
5. Total depigmentation using MBEH

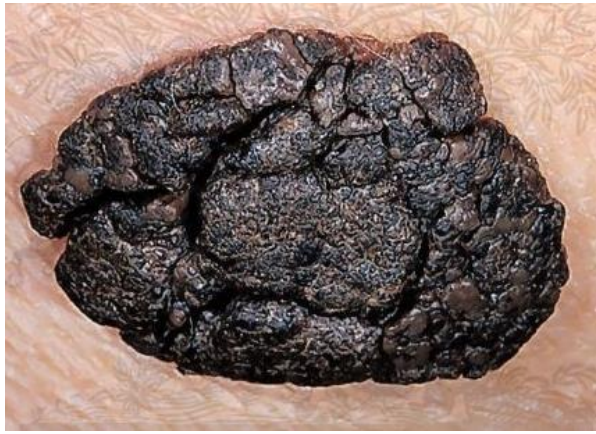
Hiperpigmentacije

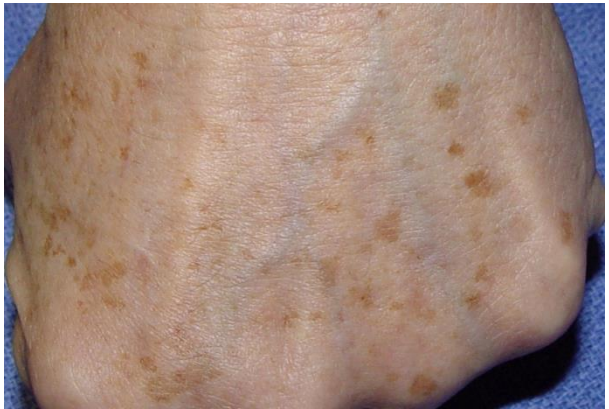


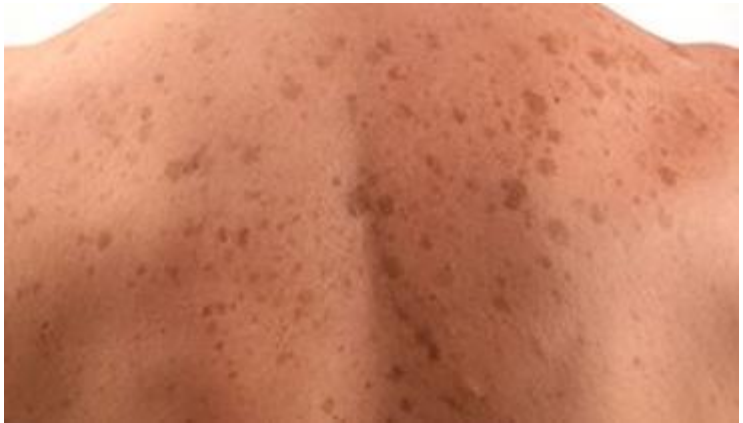






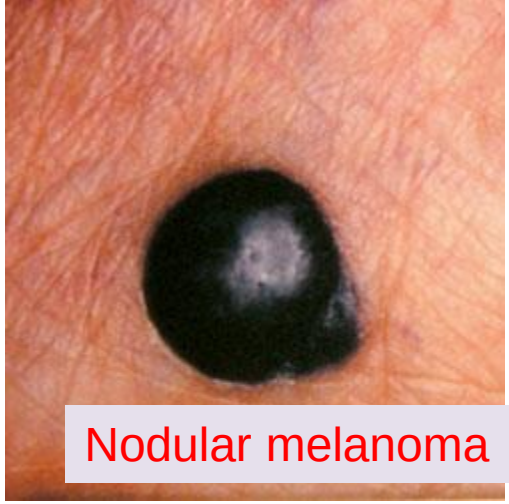






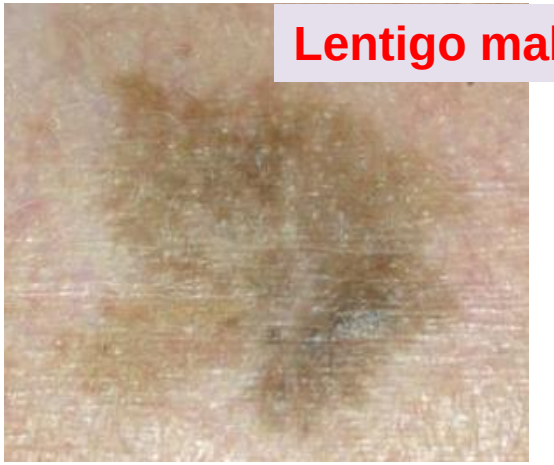
Freckles

Peutz-Jeghers sy



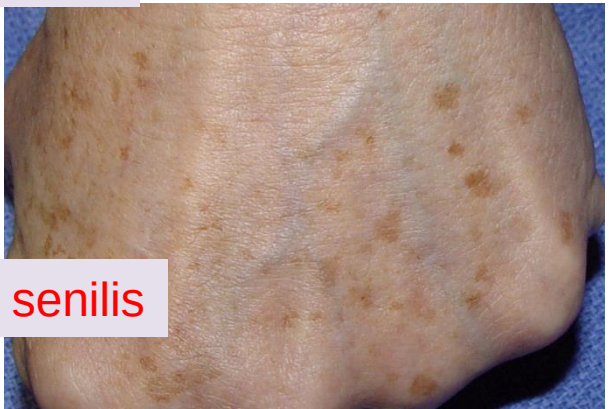
Nodular melanoma

Lentigo senilis

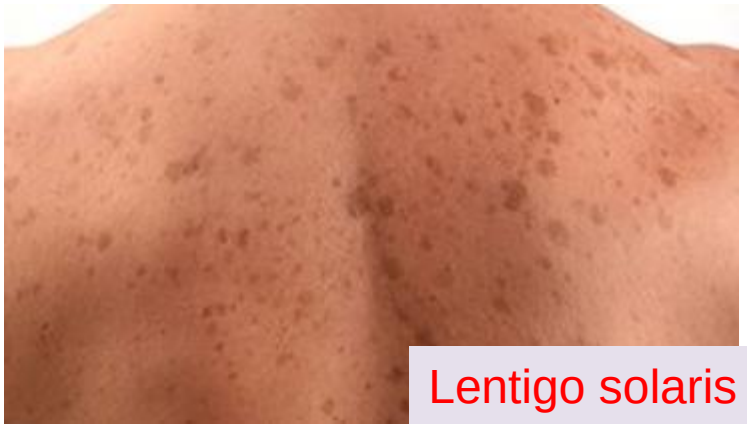


Lentigo maligna

Keratosis seborrhoica

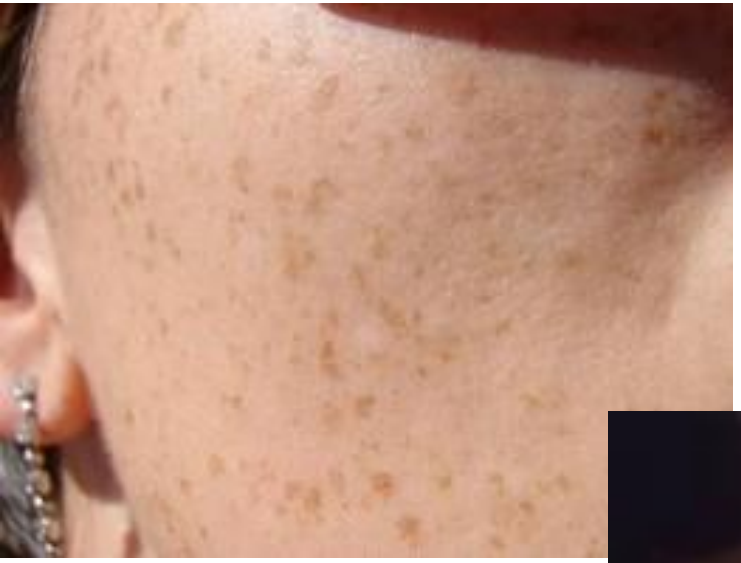


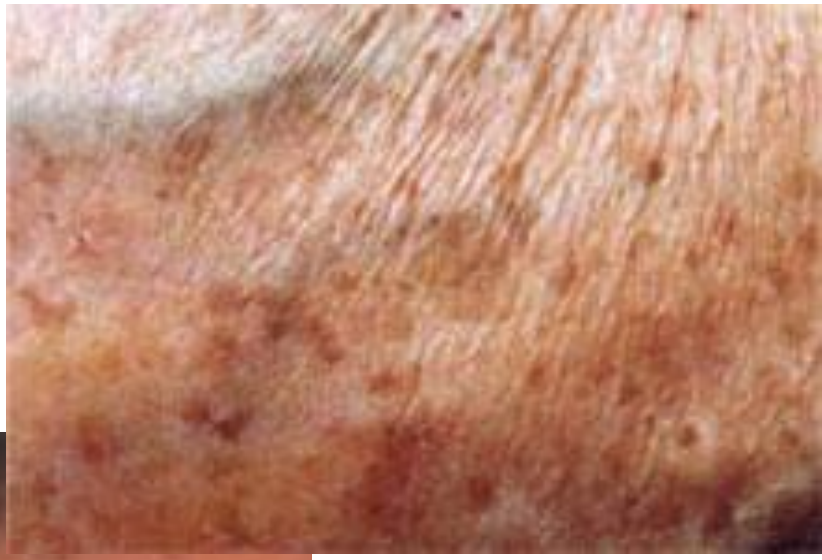
Lentigo senilis

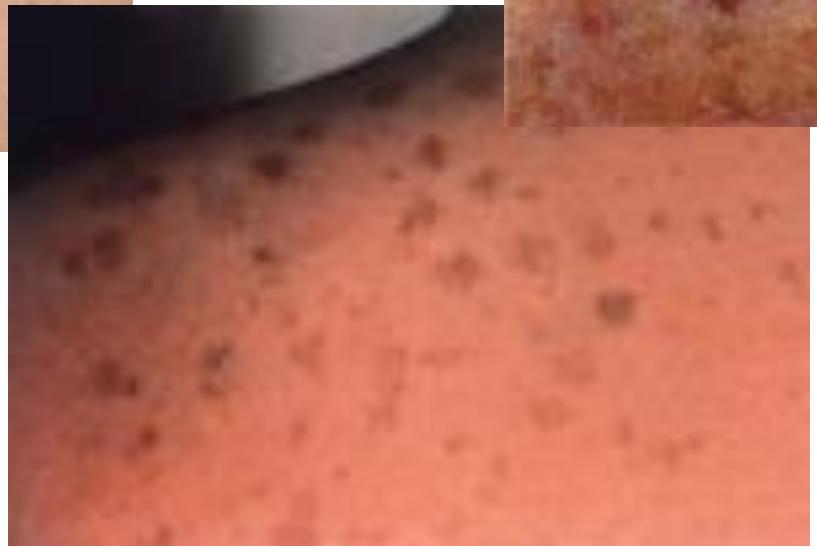


Lentigo solaris

What is lentigo and what ephelide?

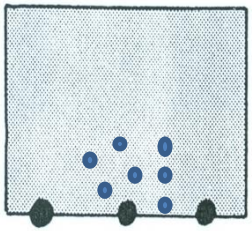






Ephelides

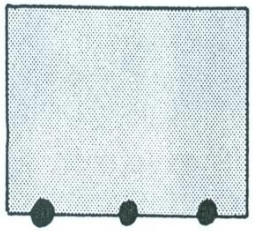
- Pale in the winter, in the summer darker.
- The increased number of **melanosomes** in the basal layer of the epidermis

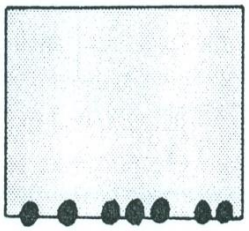


Lentigo

Always the same regardless of the UV

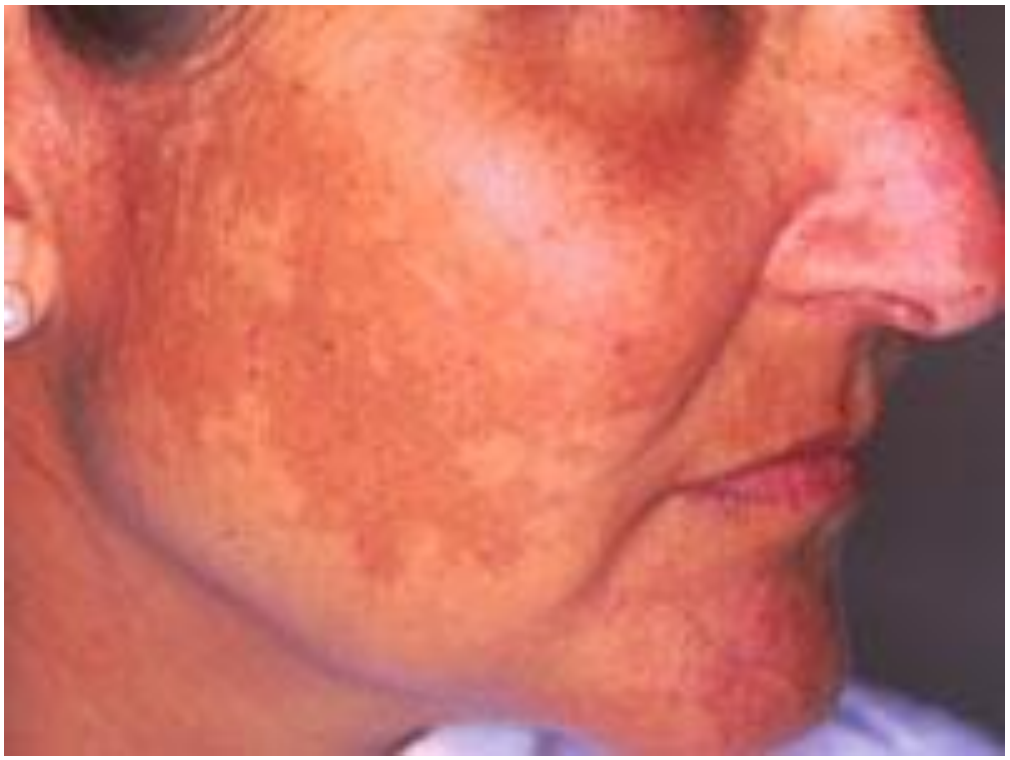
The increased number of melanosomes and melanocytes





normal

Case 2



Female, 41.y.

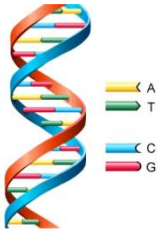
Dark macules on the face for the last 10 years (after the second pregnancy.)

Melasma (grč. “black macule”)

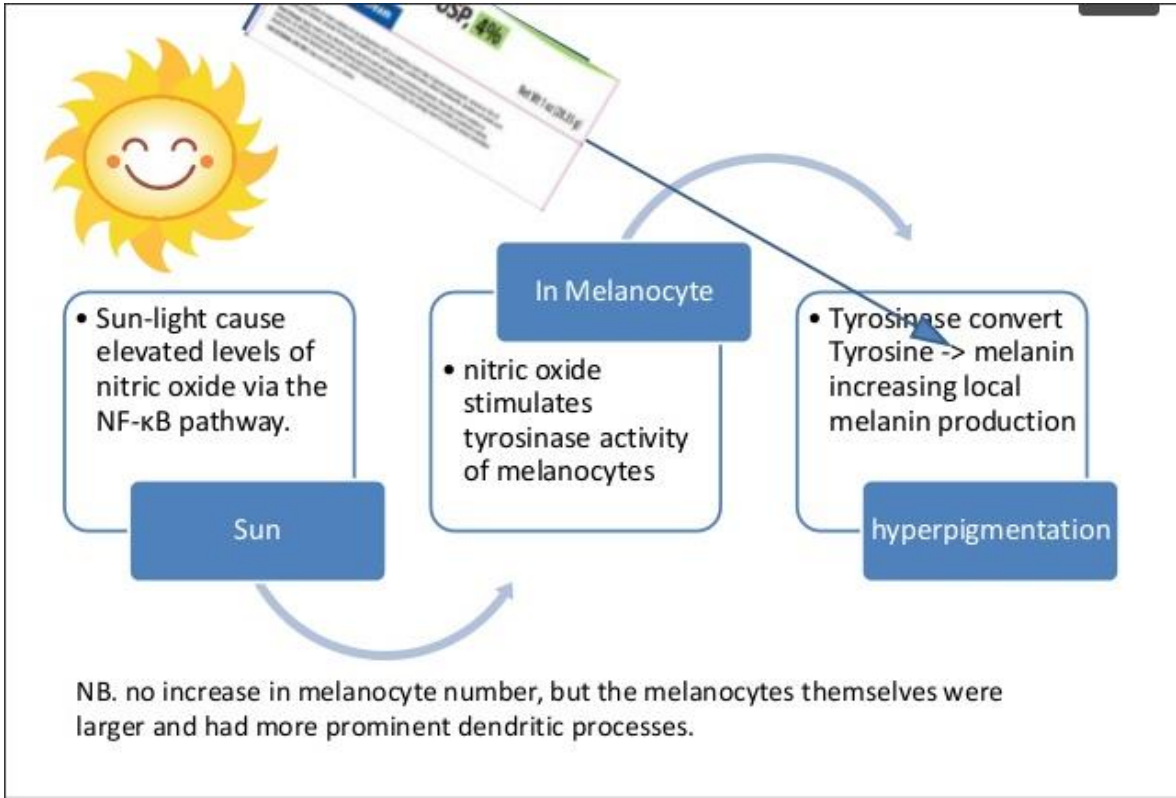
UV

Hormons

(pregnancy, hormonal th, menopause, cirrhosis)



Genetics



Melasma

Depth of the pigment:
Epidermal
Dermal
mixted

Woodova lampa

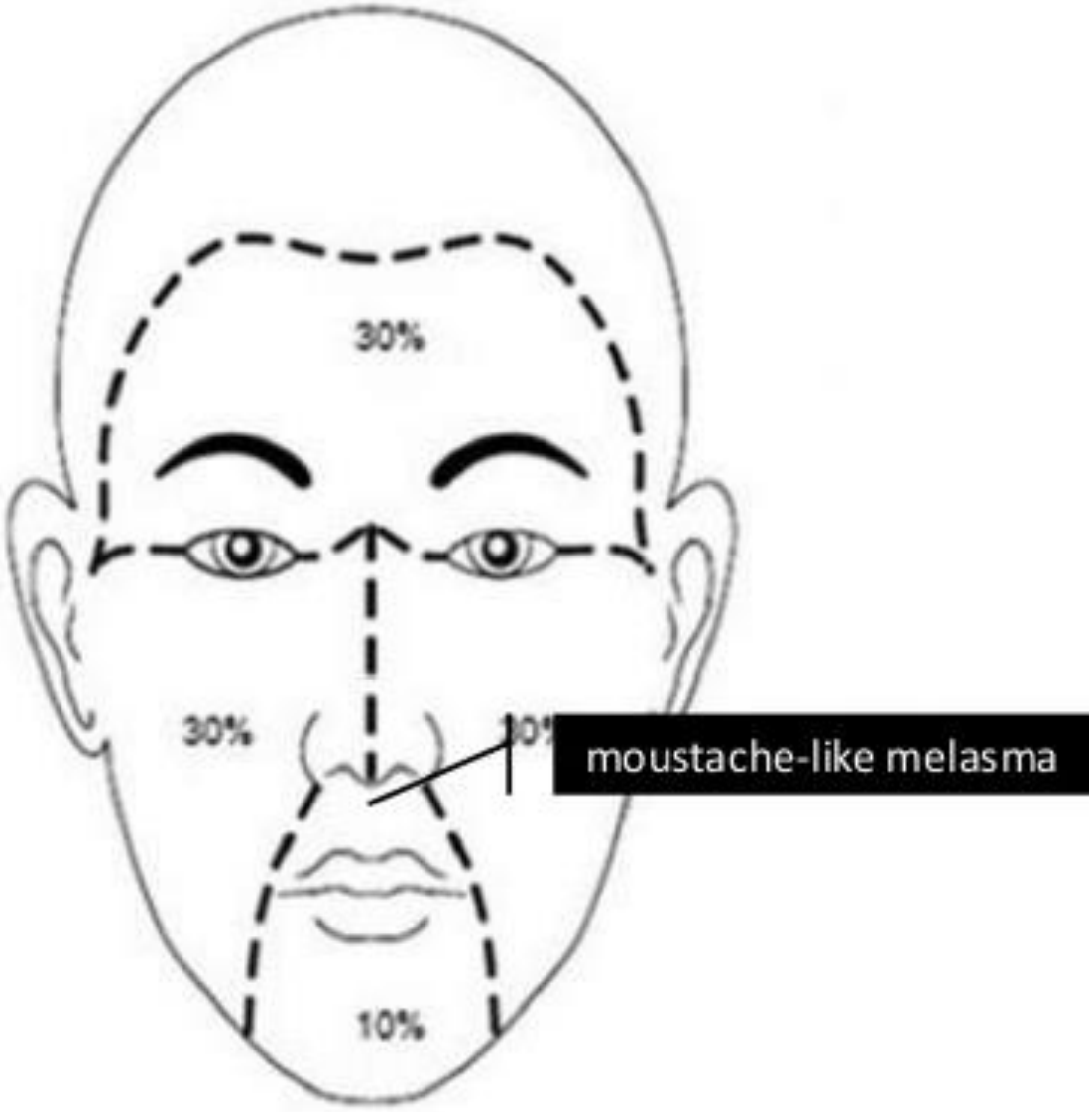
- by Wood's light examination !!!/ physical examination:

Type of melasma	Clinical features
Epidermal	▪ Well-defined border ▪ Dark brown colour ▪ Appears more obvious under black light ▪ Responds well to treatment
Dermal	▪ The most common type ▪ Ill-defined border ▪ Light brown or bluish in colour ▪ Unchanged under black light ▪ Responds poorly to treatment
Mixed	▪ Combination of bluish, light and dark brown patches ▪ Mixed pattern seen under black light ▪ Partial improvement with treatment

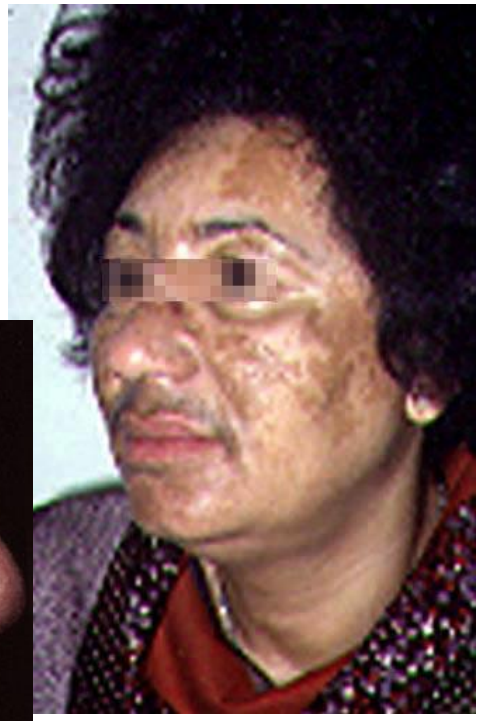
Where?

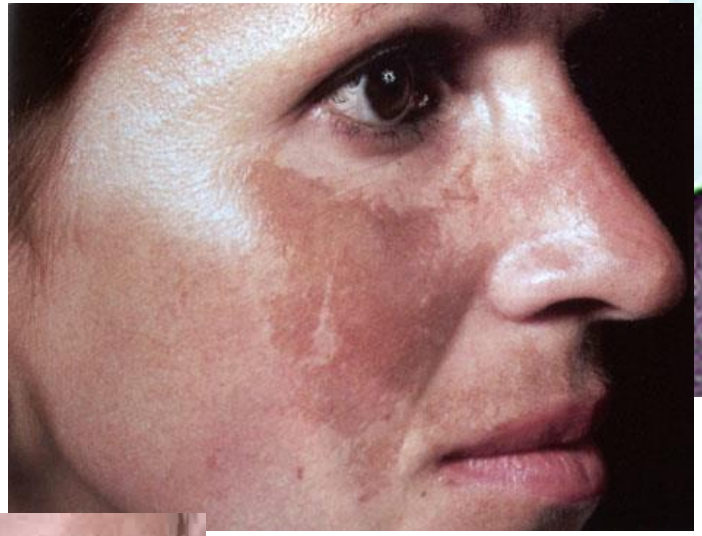
Sun exposed areas:

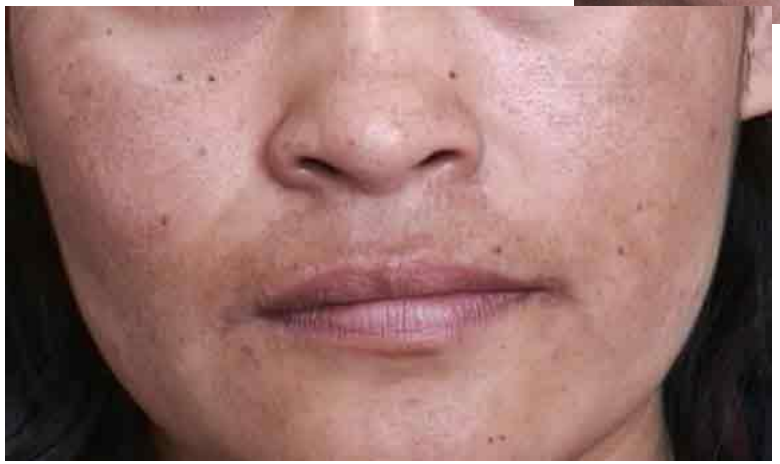
- Forehead, cheeks , nose bridge, upper lip (moustache-like melasma), chin, V -neck.



Distribution:
Centrofacial
Maxilar
Mandibular

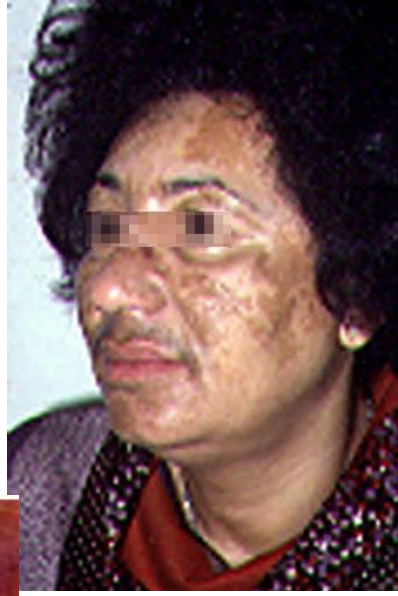


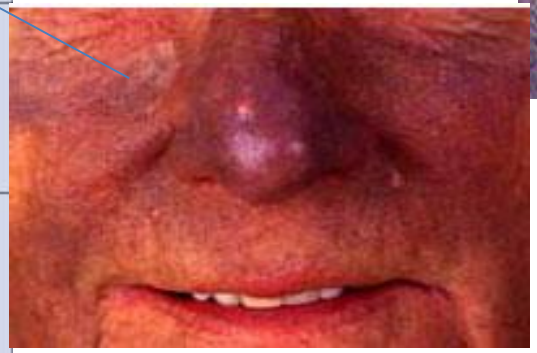




Diferencijalna dijagnoza melazme

MELASMA DIFFERENTIAL DIAGNOSIS	
Disorder	**Key differences from melasma**
Drug-induced hyperpigmentation	• History of medication use • Hyperpigmentation less patterned and less irregular in outline
Postinflammatory hyper-pigmentation due to cutaneous lupus erythematosus, skin infections, photosensitivity reactions, atopic dermatitis or contact dermatitis	• Presence (or history) of inflammatory phase, erythema, scale and possible pruritus • Primary lesions – admixed or elsewhere on body; often absent in pigmentory contact dermatitis
Exogenous ochronosis	• History of hydroquinone application • Small pigmented papules may also been seen • Banana-shaped, yellow-brown deposits in the epidermis
Actinic lichen planus	• Fine scale overlying violaceous lesions • Biopsy findings include hydropic degeneration of basal layer, with a band of lymphocytes at the dermal–epidermal junction
Erythema dyschromicum perstans	• Inflammatory phase with rim of erythema occasionally seen • Lesions slate-gray to blue-brown • Distribution includes non-sun-exposed areas
Poikiloderma of Civatte	• Presence of atrophy and telangiectasias • Favors lateral and inferior anterior neck
Erythromelanosis	• Red–brown patches of lateral cheeks and neck
Follicularis faciei et colli	• Superimposed tiny pale follicular papules
Cutaneous mercury deposits	• History of use of mercury-containing soaps or creams • Dermatitis often present





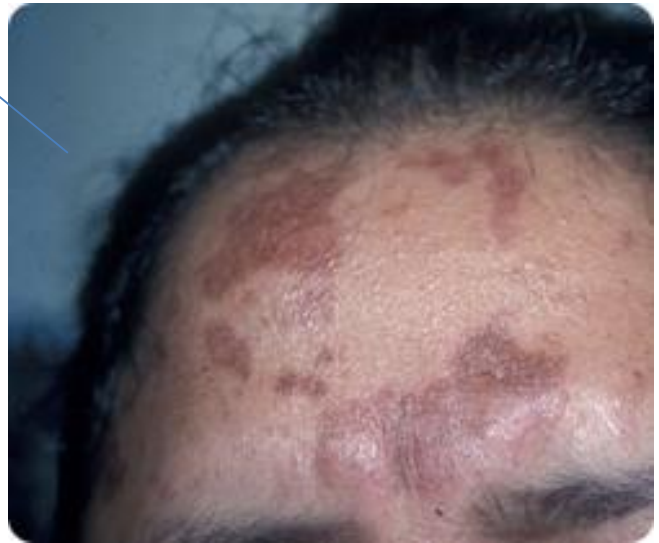


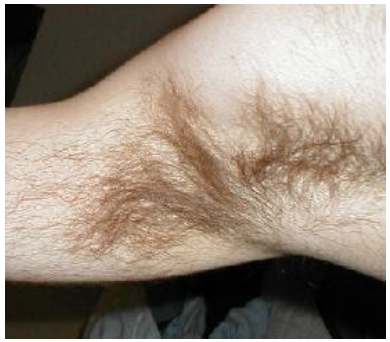
Table II. Managing melasma

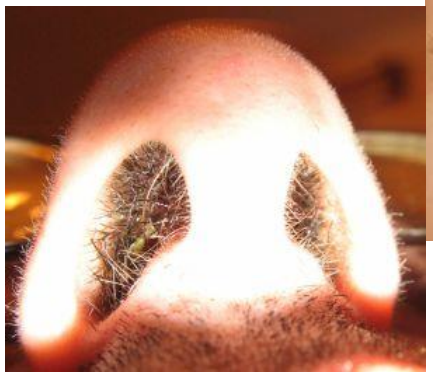
	Primary agent (strength of recommendation)	Alternative agents (strength of recommendation)
First-line	Triple combination products containing hydroquinone, a retinoid, and a fluorinated steroid once daily (A), OR hydroquinone 4% twice daily for up to 6-month periods (A)	Azelaic acid (A)
Adjunctive treatment	Ascorbic acid (C)	Kojic acid (B)
Second-line	Glycolic acid peels every 4-6 wks starting at 30% and increasing in concentration as tolerated (B)	
Third-line	Fractional laser therapy (C)	Intense pulsed light (B)

It is recommended that all patients use regular broad-spectrum photoprotection and practice sun avoidance.

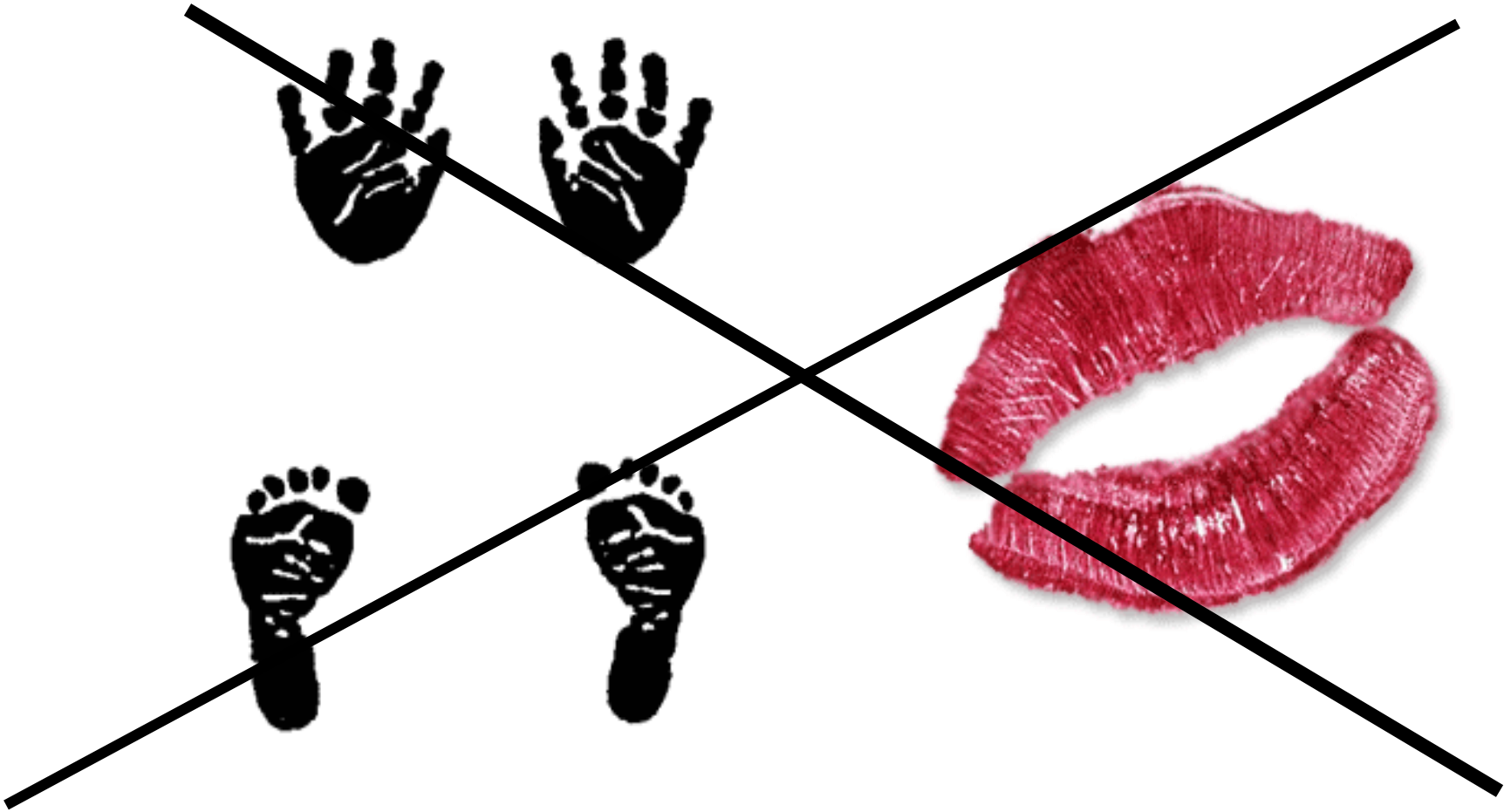
HAIR DISORDERS

What is the purpose of hair?

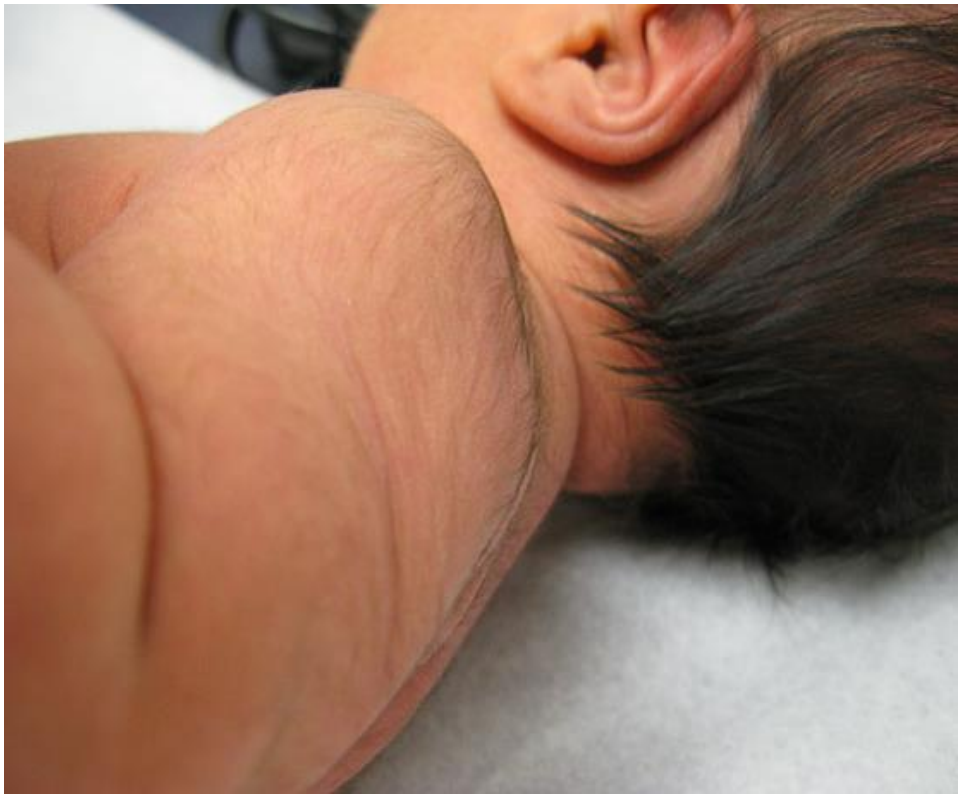


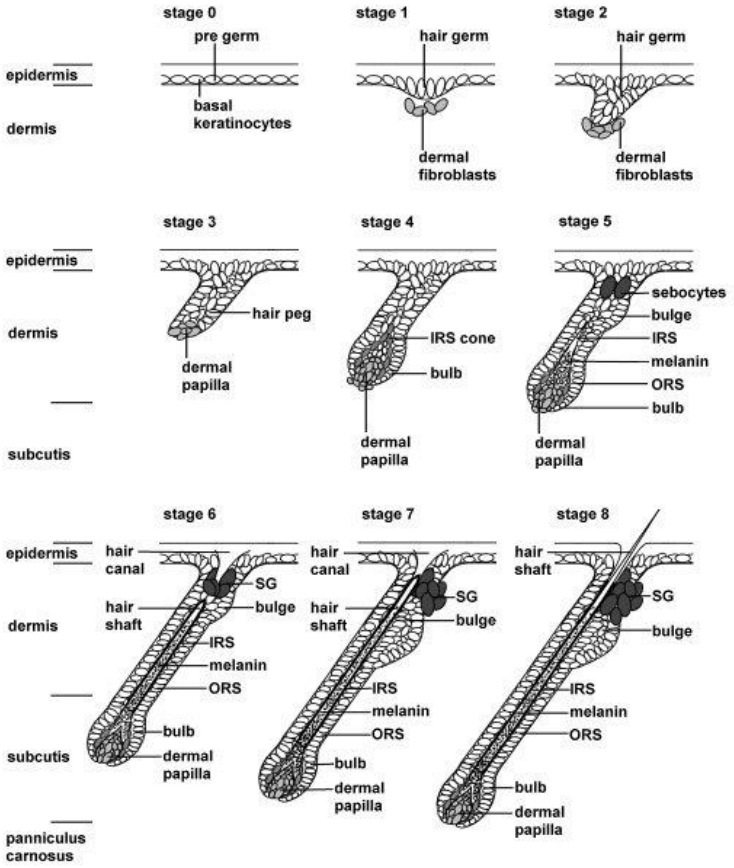


Is our whole body covered with hair?



The initial lanugo hair of the first anagen hair growth phase starts from 24-28 weeks of gestation.





Hair follicle appendage formation is the result of a complex sequence of signals between the dermal mesenchyme and the overlying epithelium.

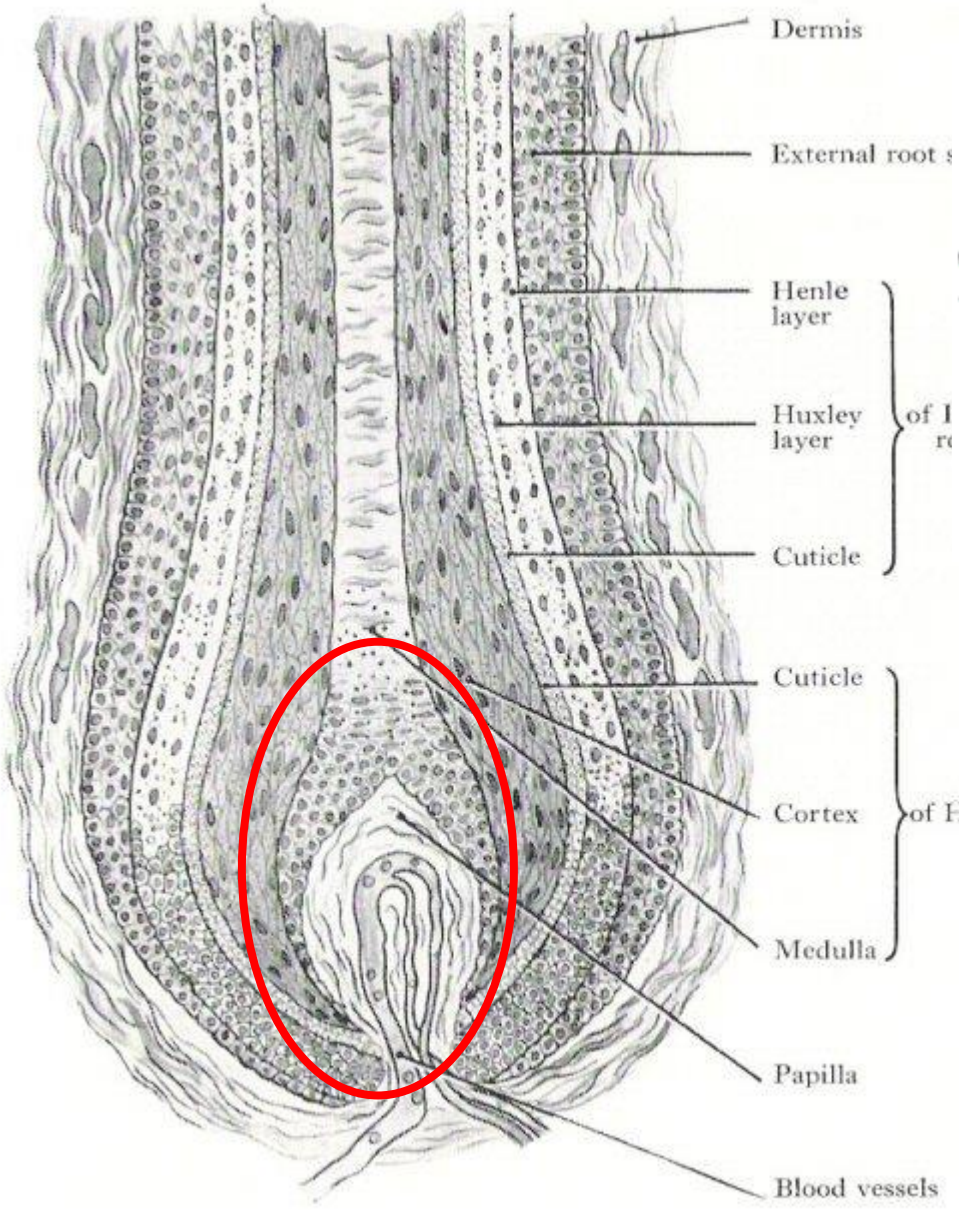
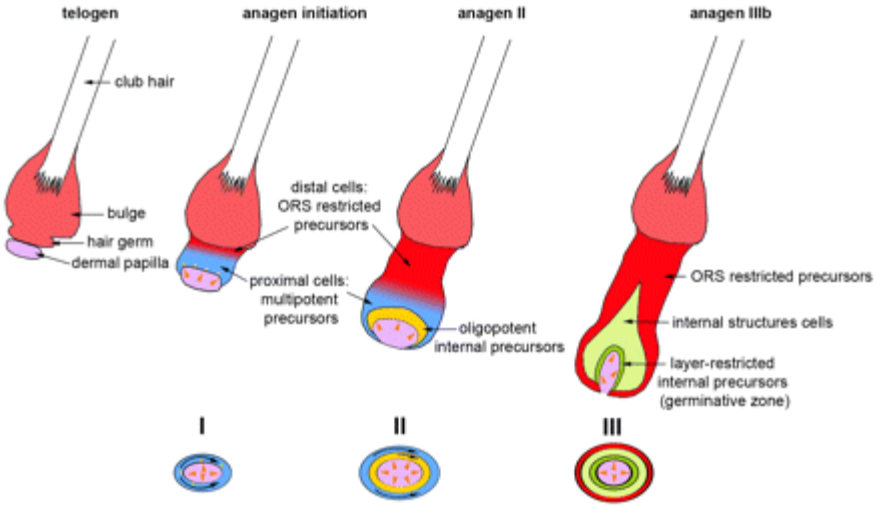
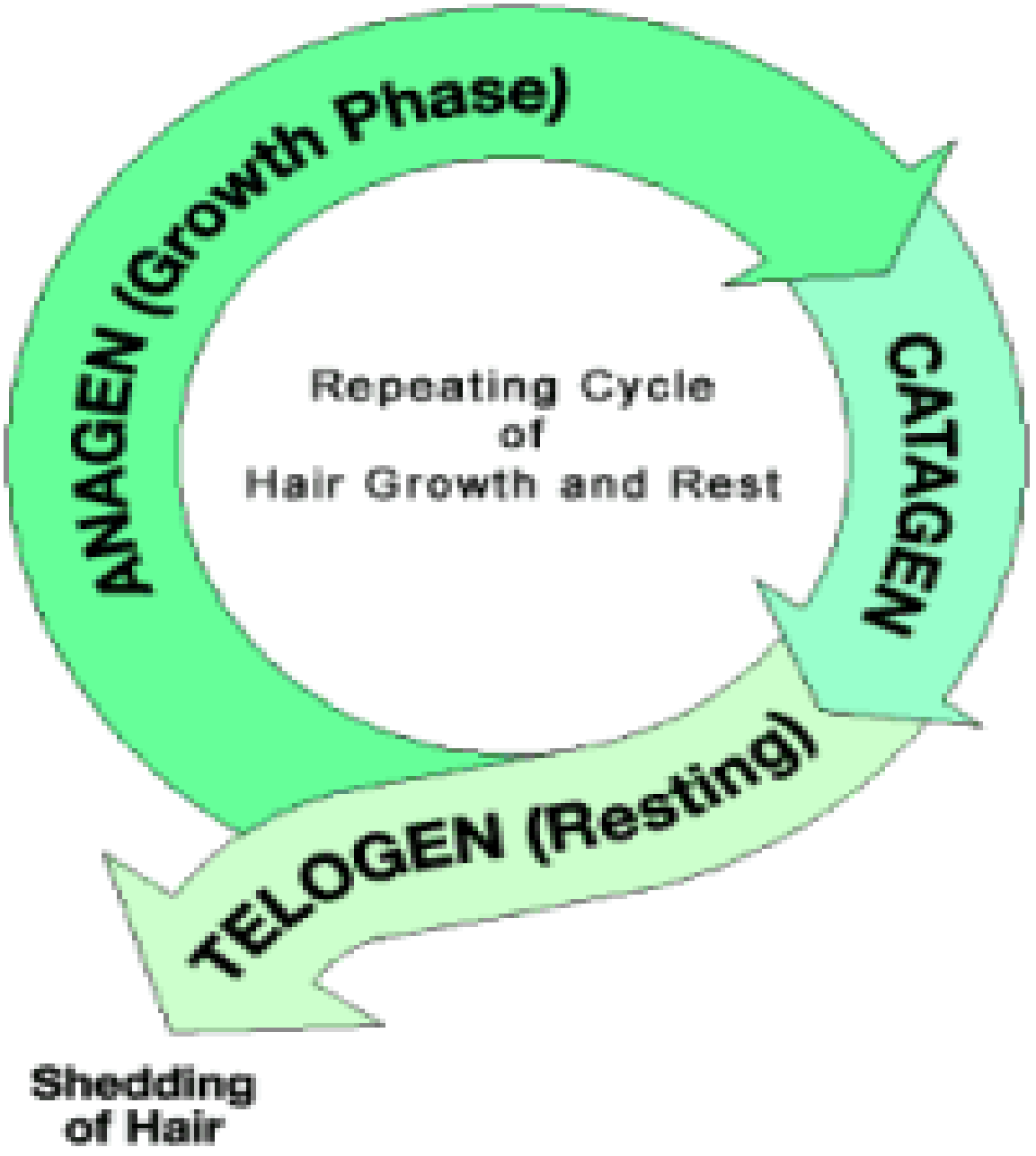
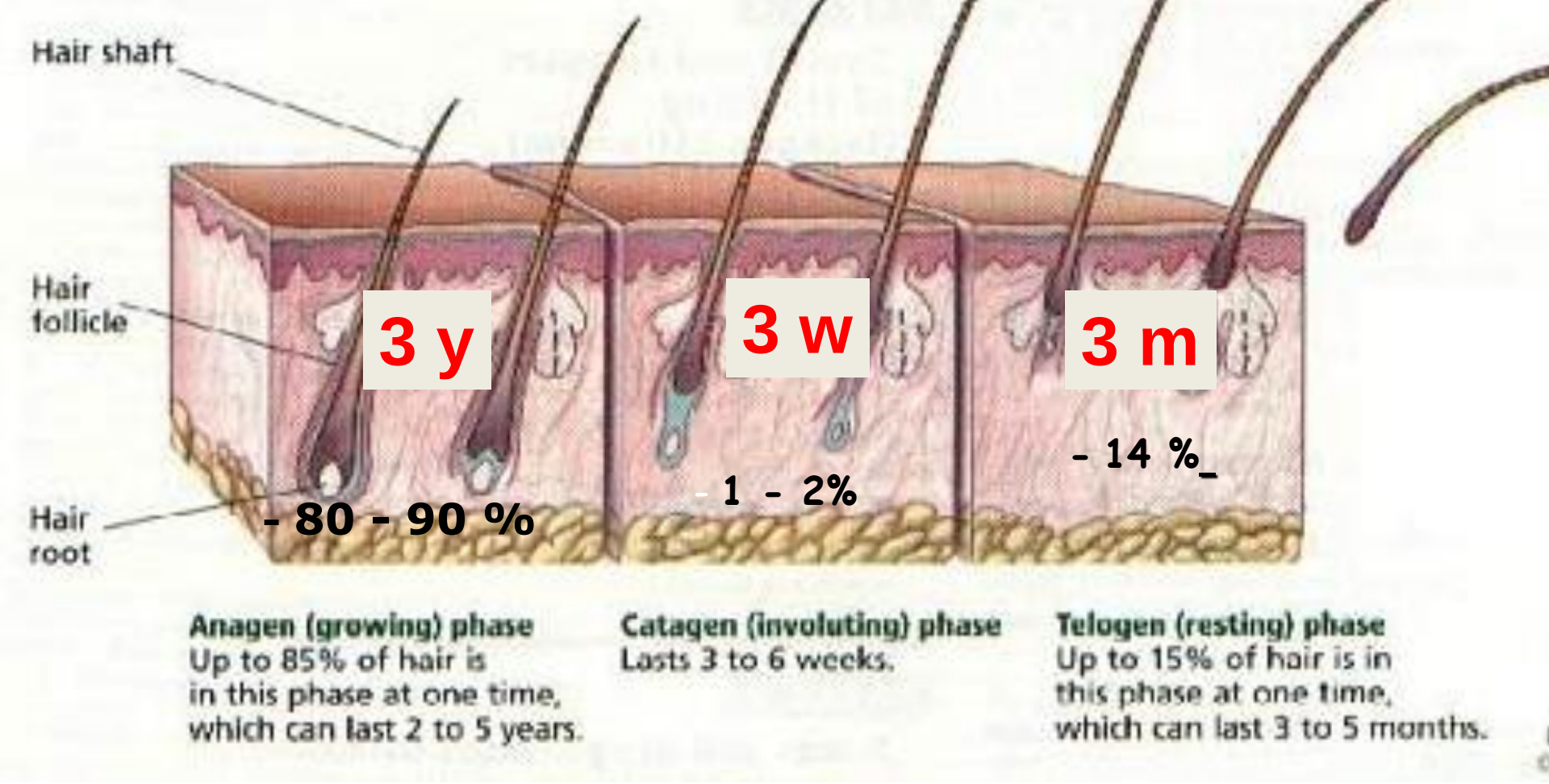


Diagram of a longitudinal section of a hair in a follicle.



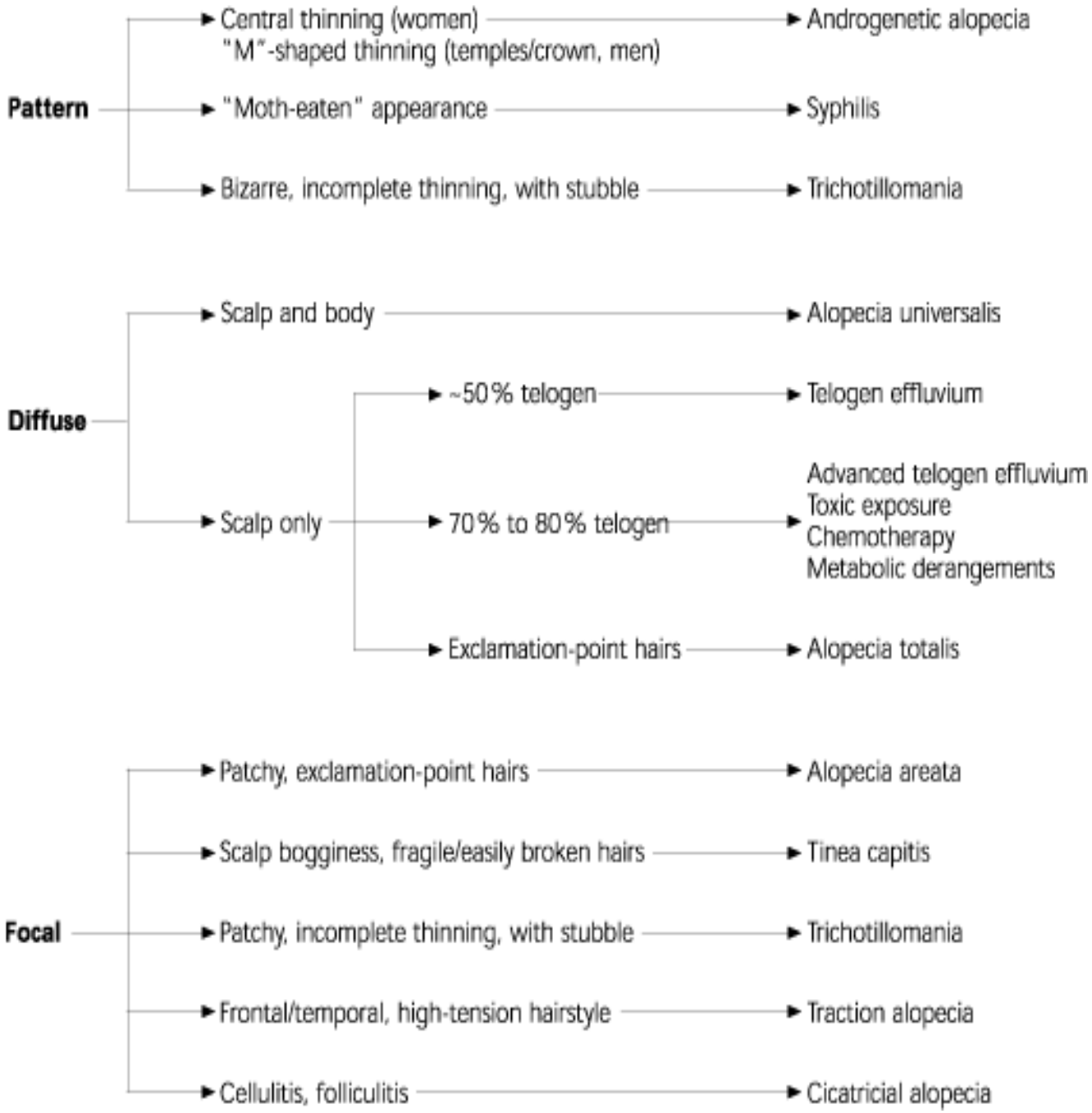
Hair follicle renewal:
authentic morphogenesis that depends
on a complex progression of stem cell lineages


ANAGEN (Growth Phase)
CATAGEN
TELOGEN (Resting)
Repeating Cycle
of
Hair Growth and Rest
Shedding
of Hair



in eyelashes and hair on the trunk and extremities anagen phase is very short and lasts up to 6 months – because the hair in these locations remain less

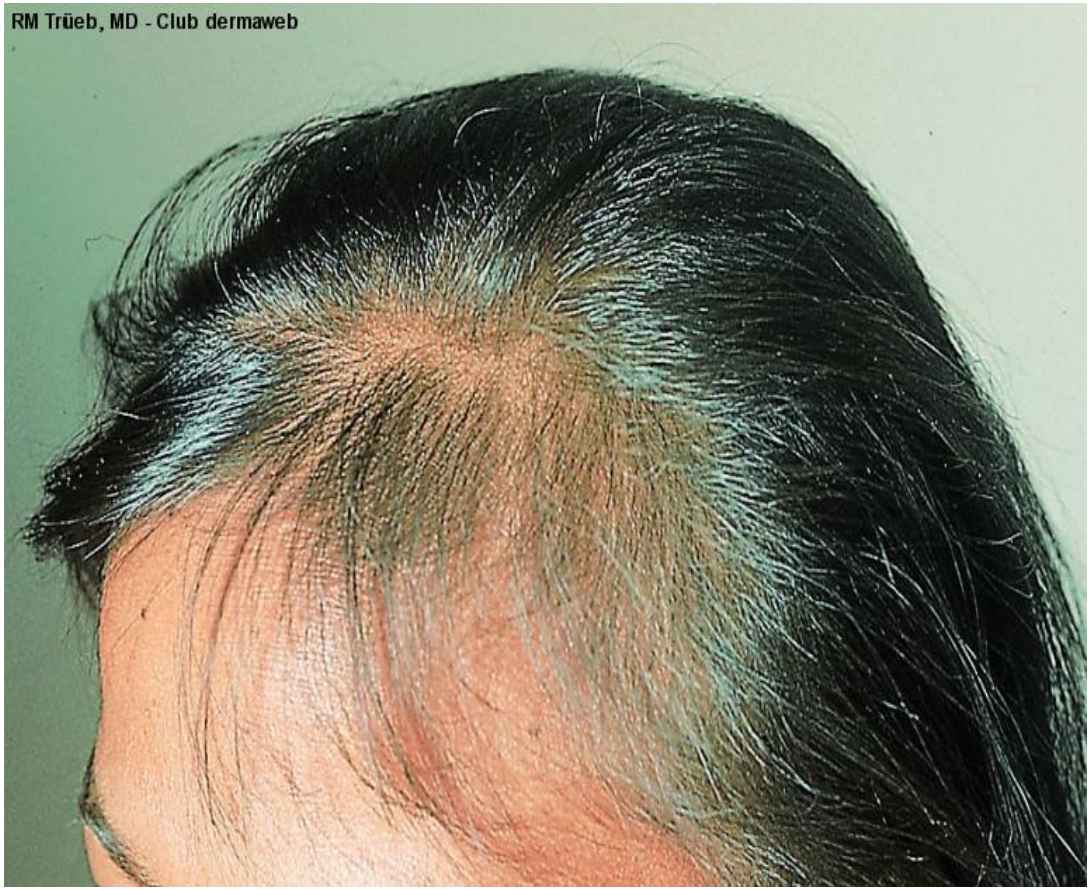
ALOPECIA


Pattern
Central thinning (women)
"M"-shaped thinning (temples/crown, men)
Androgenetic alopecia
"Moth-eaten" appearance
Syphilis
Bizarre, incomplete thinning, with stubble
Trichotillomania
Diffuse
Scalp and body
Alopecia universalis
Scalp only
~50% telogen
Telogen effluvium
70% to 80% telogen
Advanced telogen effluvium
Toxic exposure
Chemotherapy
Metabolic derangements
Exclamation-point hairs
Alopecia totalis
Focal
Patchy, exclamation-point hairs
Alopecia areata
Scalp bogginess, fragile/easily broken hairs
Tinea capitis
Patchy, incomplete thinning, with stubble
Trichotillomania
Frontal/temporal, high-tension hairstyle
Traction alopecia
Cellulitis, folliculitis
Cicatricial alopecia

Diagnostic methods in trihology

- Light pull test
- Hard pull test (trihogram)
- **Trichoscopy**
- Micrbiologic and micologic tests (nativno,kultura,Wood)
- PHD
- DIF
- Other tests (endokrinology autoimmune,...)

Case 1



26.y. old female patient

three weeks ago
notes significant hair loss (especially after washing hair).

1 month ago she took (over 10 days) Bromergon (bromocriptine) in order stop lactation.

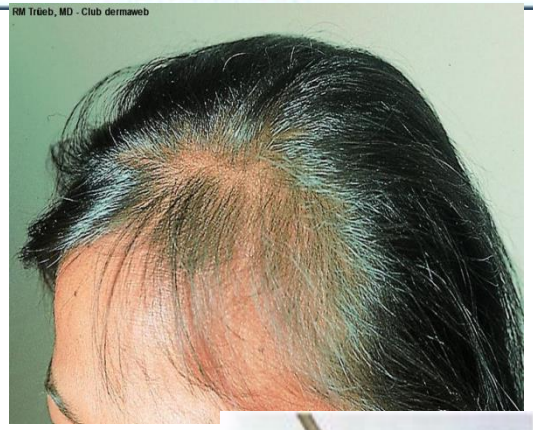
5000 000 body hair

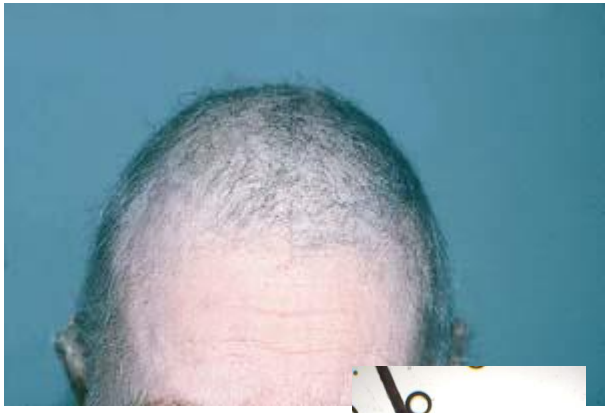
1000 000 hairs on the head

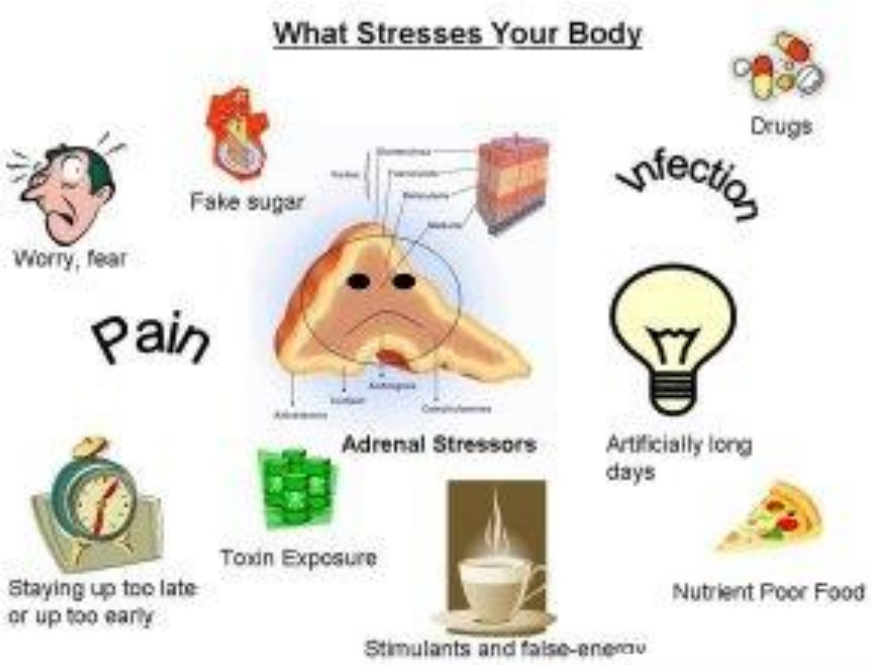
100,000 scalp

In a day
lose 75-100 hairs

Clinical presentation	Telogen	Anagen
Onset of shedding after insult	2-4 months	1-4 weeks
Percent of hair loss	20-50	80-90
Type of hair loss	Normal club (white bulb)	Anagen hair (pigmented bulb)
Hair shaft	Normal	Narrowed or fractured





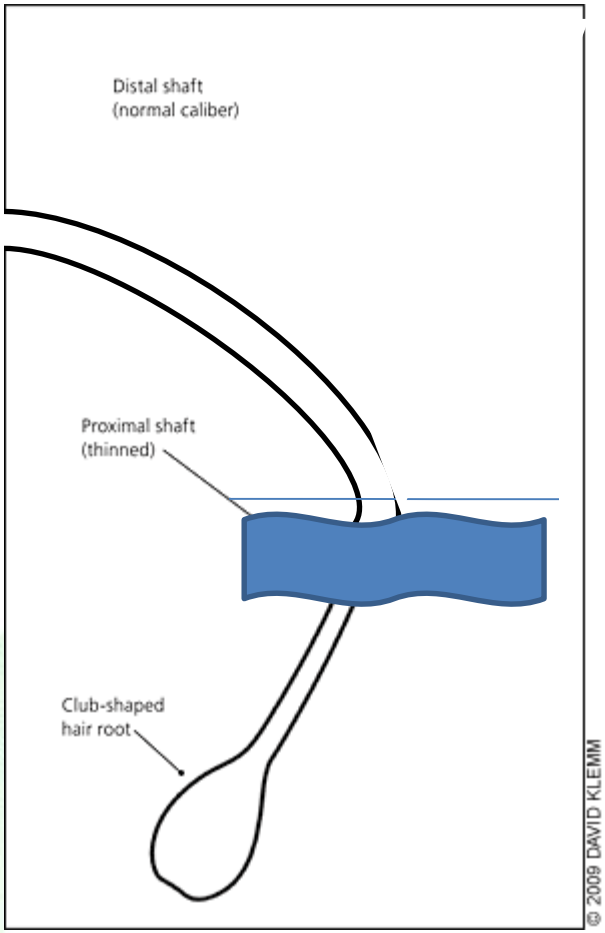


Stress event

critical is the
intensity and duration
of the stress
not the type and quality
of the stress event

Anagen arrest

Commonly or frequently associated	Uncommon/Infrequently associated
Doxorubicin	Vincristine
Daunorubicin	Vinblastine
Paclitaxel	5-Fluorouracil
Docetaxel	Hydroxyurea
Cyclophosphamide	Thiotepa
Ifosfamide	
Etoposide	
Mechlorethamine	
Methotrexate	
Bleomycin	



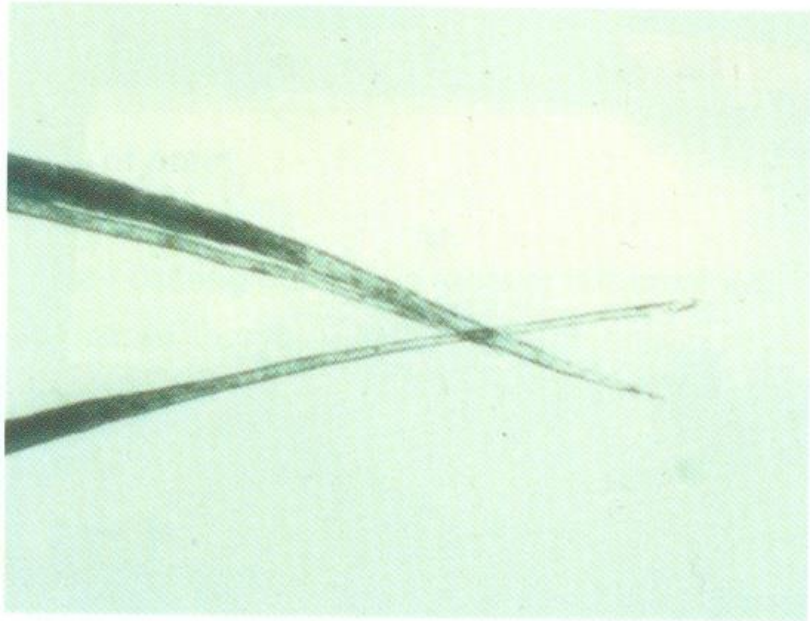


Fig. 4. Anagen arrest with chemotherapy. Mitotic arrest narrows the shafts and hairs break when they reach the scalp surface.

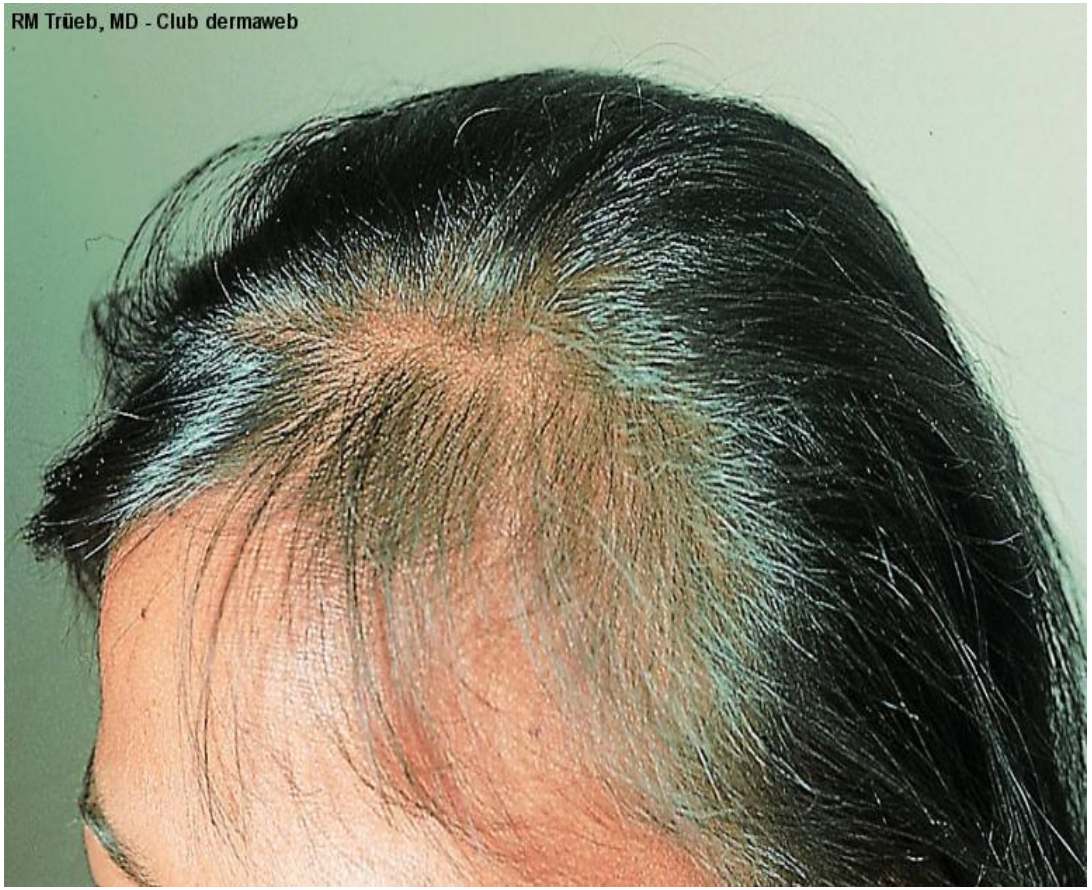
causes of telogen effluvium

Nutritional Deficiency
Stress
Fever
Drugs
Postpartum
Hypothyroidism/hyperthyroidism
Hepatic failure
Chronic renal failure
Idiopathic

- **Telogen effluvim and drugs**

- aminosalicilna kiselina
- amfetamini
- bromokriptin
- kaptopril
- karbamazepin
- cimetidin
- kumadin
- danazol
- enalapril
- etretinat
- levodopa
- litij
- metoprolol
- propranolol
- piridostigmin
- trimetadion

Case 1



26.y. old female patient

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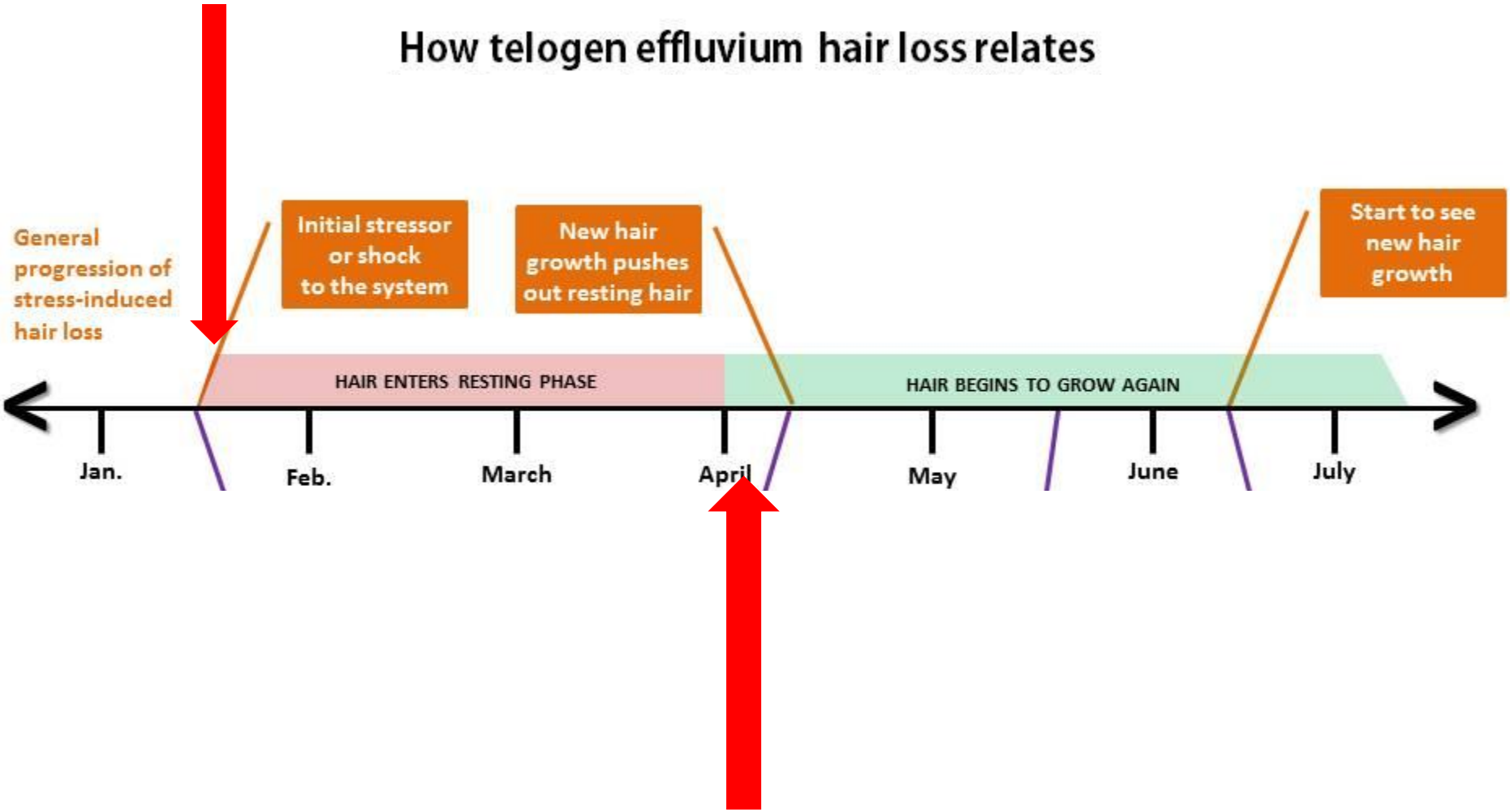
stres



effluvium

Diferential diagnosis - a. androgenetica

- trichotillomania

- psihogeni pseudoefluvij

	FPHL	Chronic TE
Distribution	Central portion of scalp and preserved frontal hairline	Generalized
Onset	Gradual	Abrupt with a trigger
Appearance	Hair thinning with wide midline part	Diffuse thinning
Hair shedding	Minimal	Prominent
Hair pull test	Usually negative	Positive
Other history	Family history +	H/O previous major illness or stress
Scalp biopsy	T:V≤4	T:V≥7

FPHL: Female pattern hair loss, TE: Telogen effluvium; T:V: Terminal:Vellus ratio

♀

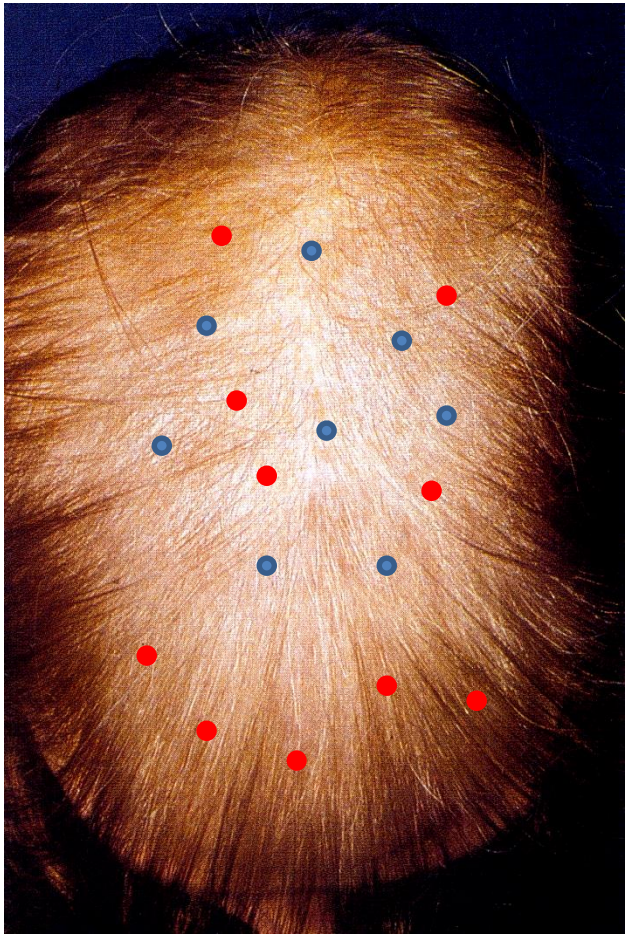
5a reduktaza
aromataza

CLINICAL PICTURE

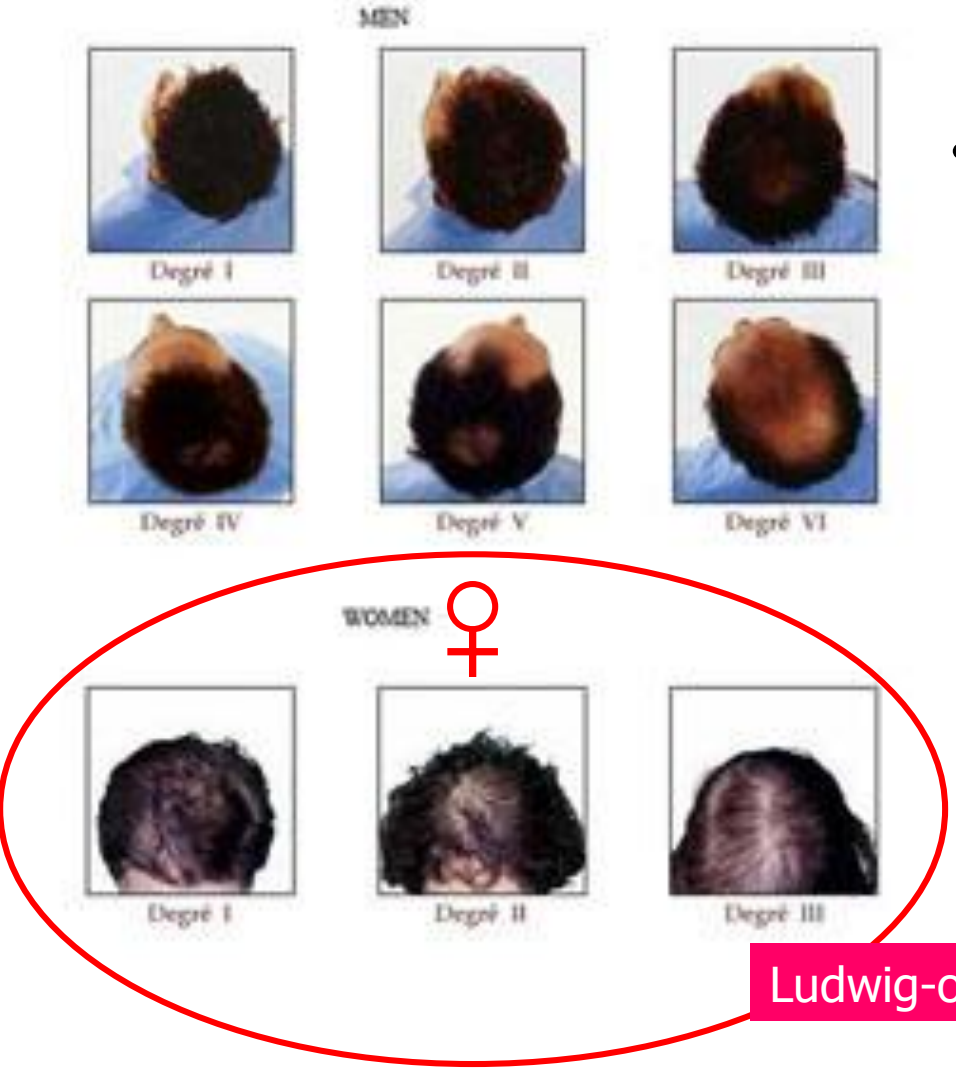
preserved frontal scalp line

Without complete baldness

Type "Christmas trees”



Alopecia androgenetica – u žena



Ludwig-ova scale

- incidence increases with age (> 65th year -75% of women)

  - reflection of increased secretion of androgens (PCO)
  - **increased sensitivity of the receptor follicles**
  - **increased production of androgens in target tissues**

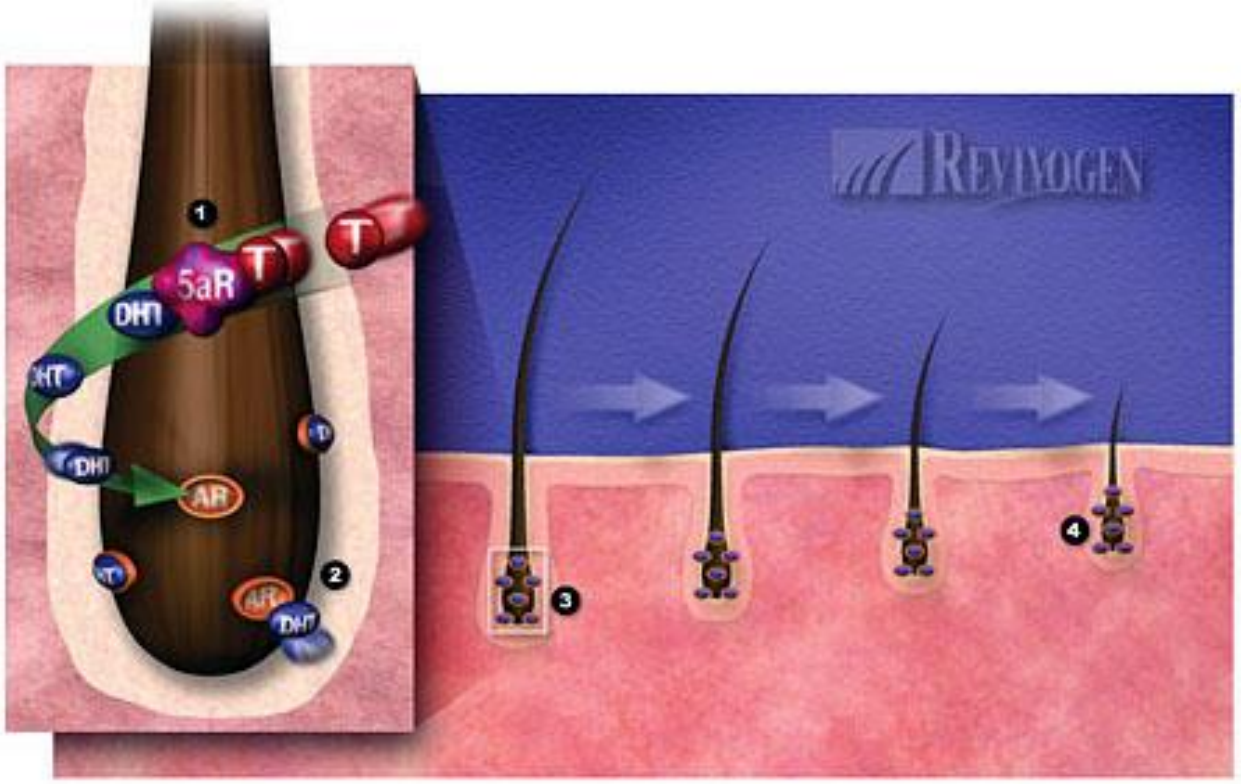
Alopecia androgenetica

- **the most common type of alopecia (95% of all forms)**
- **1/3** ♂

The influence of
1. androgens,
2. age factor
3. Genetics
(AD or polygenetic)

The influence of androgens

dihydrotestosterone (DHT)
transported
into the follicle
Stimulates
inhibition of growth factors
and
regressive metamorphosis



- **shortening of the anagen phase,**
- **greater number of telogen hairs**
- **reducing the number of follicles**

Alopecia androgenetica

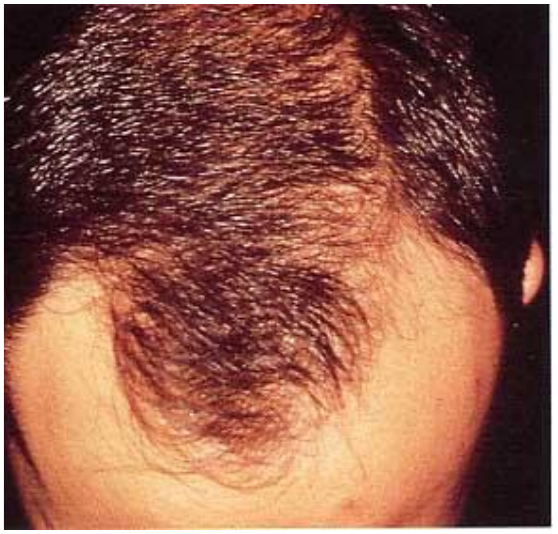
miniaturization of the hair

appearance
at
young age

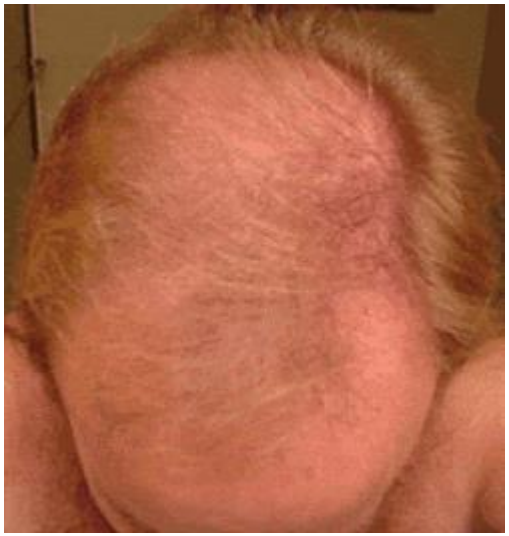
baldness

appearance
later
in life

sparse scalp

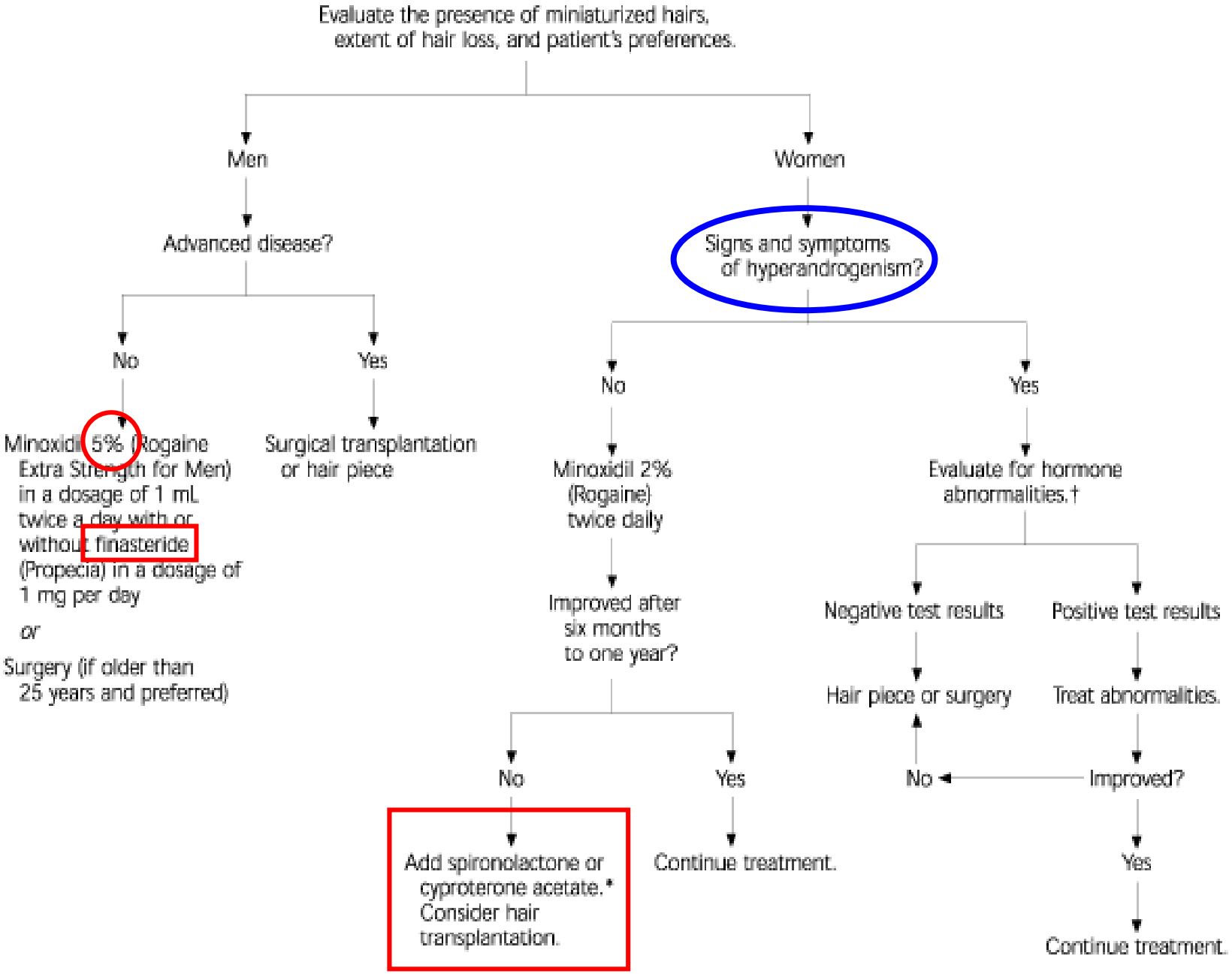


Terapy AgA



- Androgenetic Alopecia in Women

- Hair pieces
- Camouflage
- Hair enhancers
  - shampoos
  - mousse products
  - electromagnetic fiber particles (to hide scalp color in light-skinned people)
- Hair transplantation
- Topical minoxidil 2%


Evaluate the presence of miniaturized hairs, extent of hair loss, and patient's preferences.
Men
Women
Advanced disease?
Signs and symptoms of hyperandrogenism?
No
Yes
No
Yes
Minoxidil 5% (Rogaine Extra Strength for Men) in a dosage of 1 mL twice a day with or without finasteride (Propecia) in a dosage of 1 mg per day
or
Surgery (if older than 25 years and preferred)
Surgical transplantation or hair piece
Minoxidil 2% (Rogaine) twice daily
Improved after six months to one year?
Evaluate for hormone abnormalities.†
Negative test results
Positive test results
Hair piece or surgery
Treat abnormalities.
No
Improved?
No
Yes
Add spironolactone or cyproterone acetate.* Consider hair transplantation.
Continue treatment.
Yes
Continue treatment.

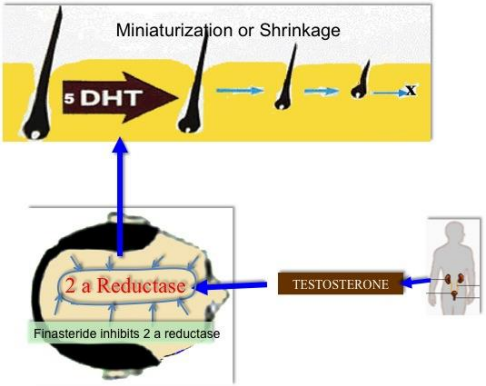
MINOXIDIL

(derivat piperidinopirimidina)

strong vasodilator
1970 - hypertension;
1986 - local preparation for alopecia

shortens telogen,
extends the anagen

The mechanism of action is unclear



FINASTERIDE

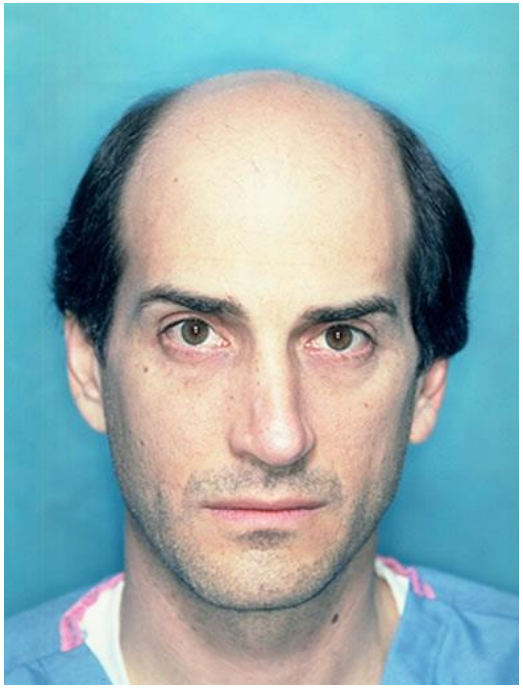
(inhibitor of the type II 5-Alpha reductase isoenzyme, which is predominant in the scalp hair root sheath)

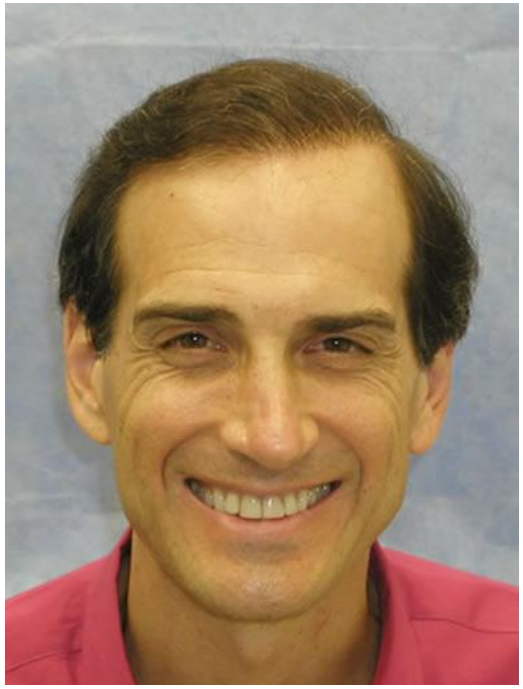
Table 1

Dosing, Adverse Effects, and Price of Minoxidil and Finasteride

Product	Dosing	Adverse Effects
Minoxidil solution: 2% and 5% (available as generic or Rogaine) Minoxidil foam: 5% also available	2% and 5% solution: Apply 1 mL (25 drops) to scalp bid Foam: Apply one-half capful to scalp bid	Scalp irritation (7% with 2% solution and may be slightly higher with 5% solution) Allergic contact dermatitis (0.6%-3.7%)
Finasteride (available as Propecia only)	1 mg po daily	Decreased libido (1.8%) Erectile dysfunction (1.3%) Decreased ejaculate volume (0.8%)

Source: References 7, 13-16.

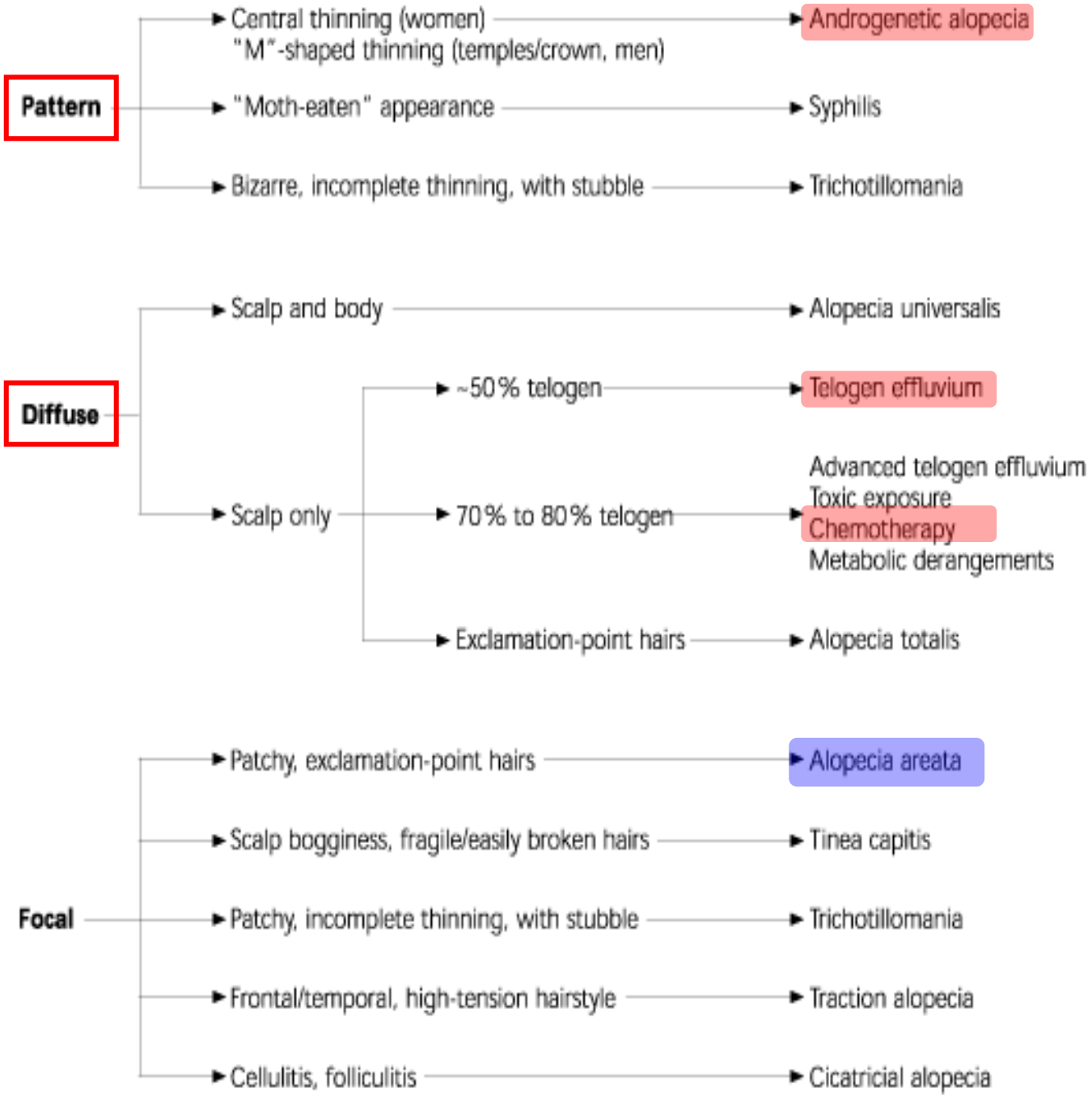




Transplantation - with punch grafts
from the occipital region to the frontal region

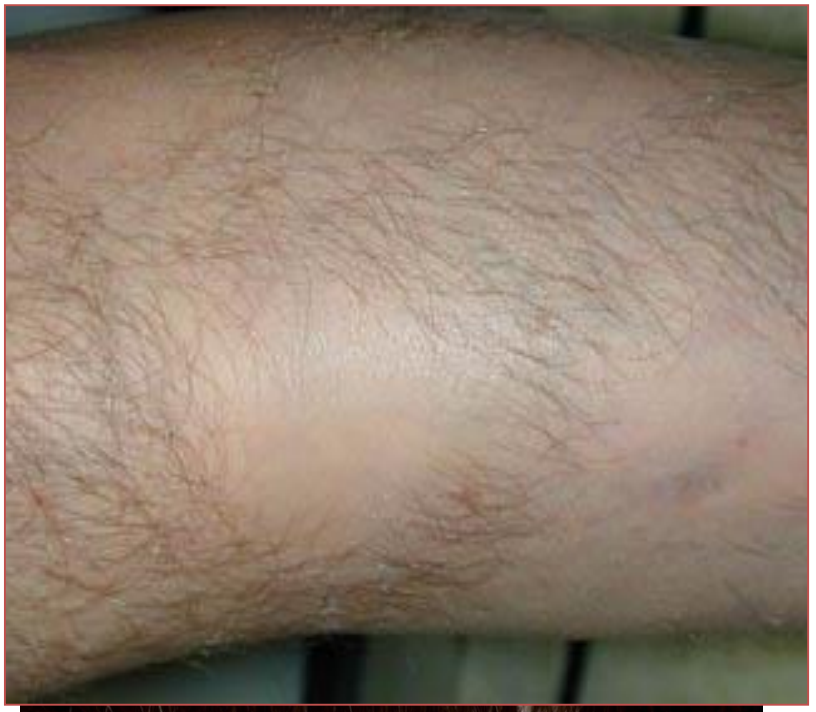
hair from the nape of hormonally independent
and such grafts are preserved for life

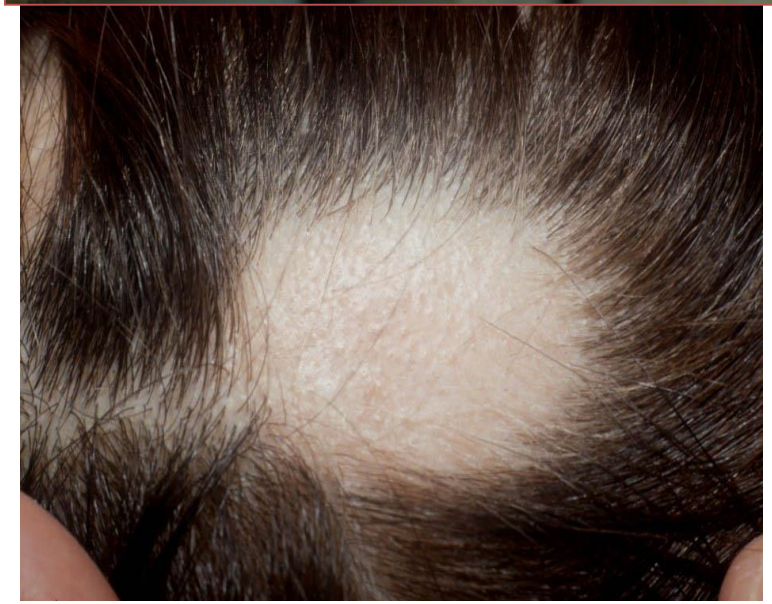
ALOPECIA


Pattern
Central thinning (women)
"M"-shaped thinning (temples/crown, men)
Androgenetic alopecia
"Moth-eaten" appearance
Syphilis
Bizarre, incomplete thinning, with stubble
Trichotillomania
Diffuse
Scalp and body
Alopecia universalis
Scalp only
~50% telogen
Telogen effluvium
70% to 80% telogen
Advanced telogen effluvium
Toxic exposure
Chemotherapy
Metabolic derangements
Exclamation-point hairs
Alopecia totalis
Focal
Patchy, exclamation-point hairs
Alopecia areata
Scalp bogginess, fragile/easily broken hairs
Tinea capitis
Patchy, incomplete thinning, with stubble
Trichotillomania
Frontal/temporal, high-tension hairstyle
Traction alopecia
Cellulitis, folliculitis
Cicatricial alopecia

Alopecia areata

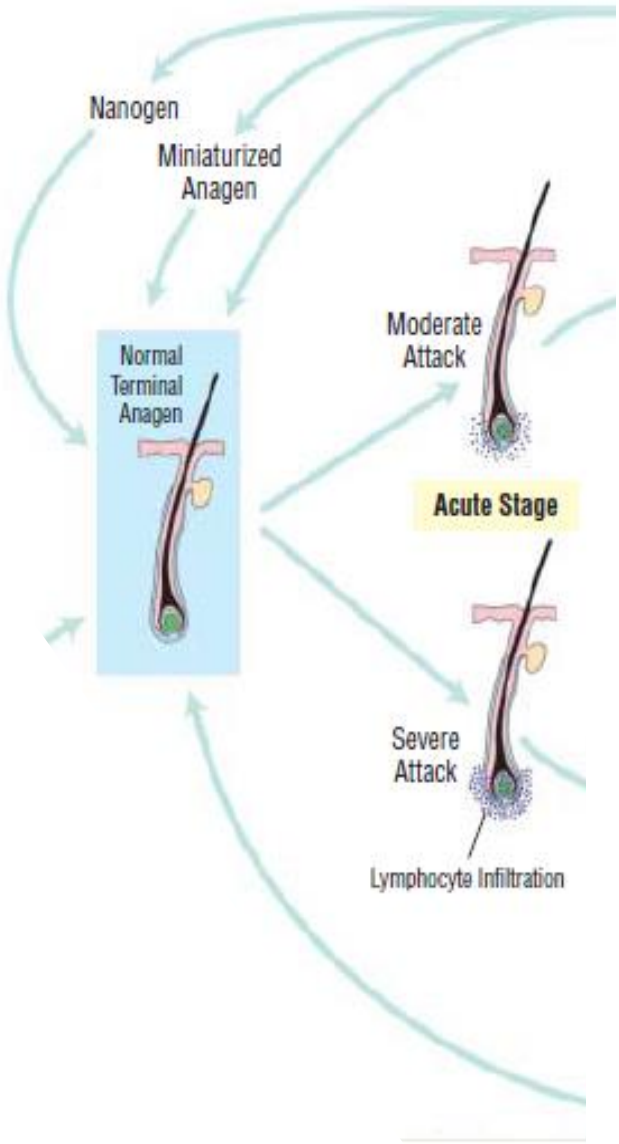
- is a common autoimmune skin disease
- resulting in the loss of hair on the scalp and elsewhere on the body
- Affects 2% of the population
- highly unpredictable and cyclical.
- Hair can grow back in or fall out again at any time, and the disease course is different for each person.

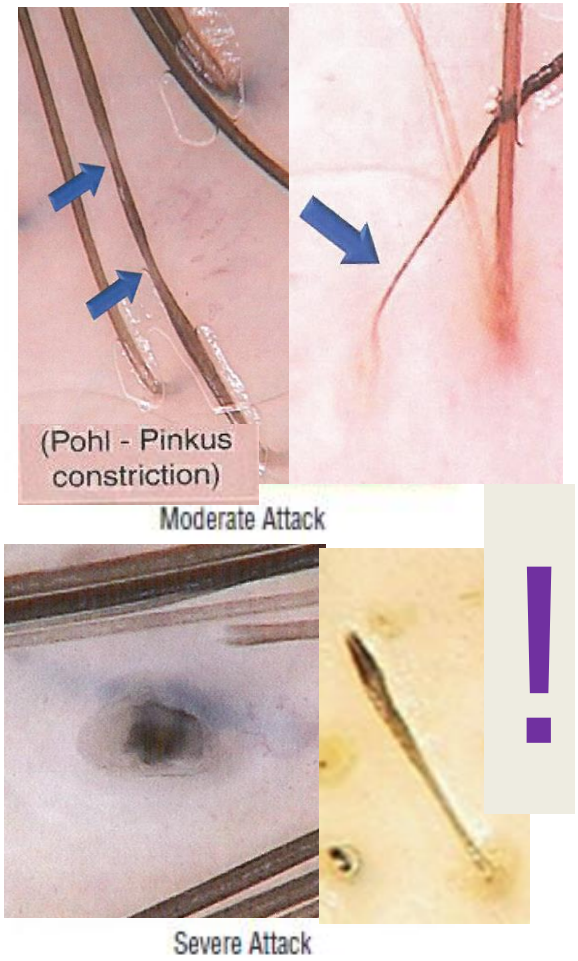




pathologic process - autoimunne inflamation

tapering hairs correspond to rapid catagen transition from anagen, broken hairs and black dots to the resultant hair fragility





Arch Dermatol. 2003;139:1555-1559

-Types of Alopecia Areata

three types of

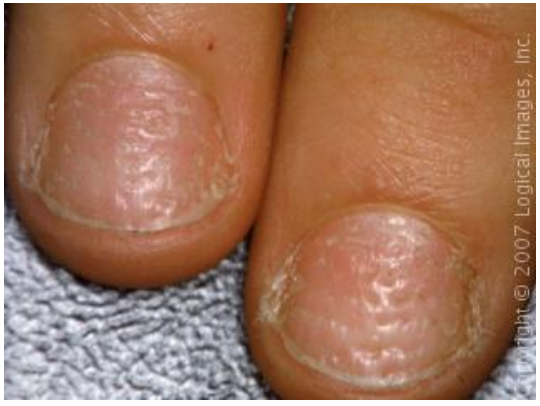
alopecia areata;

- a.unilocularis
- a. multilocularis

- alopecia areata totalis
- alopecia areata universalis.

Subtypes

- ophiasis
- difuzan oblik



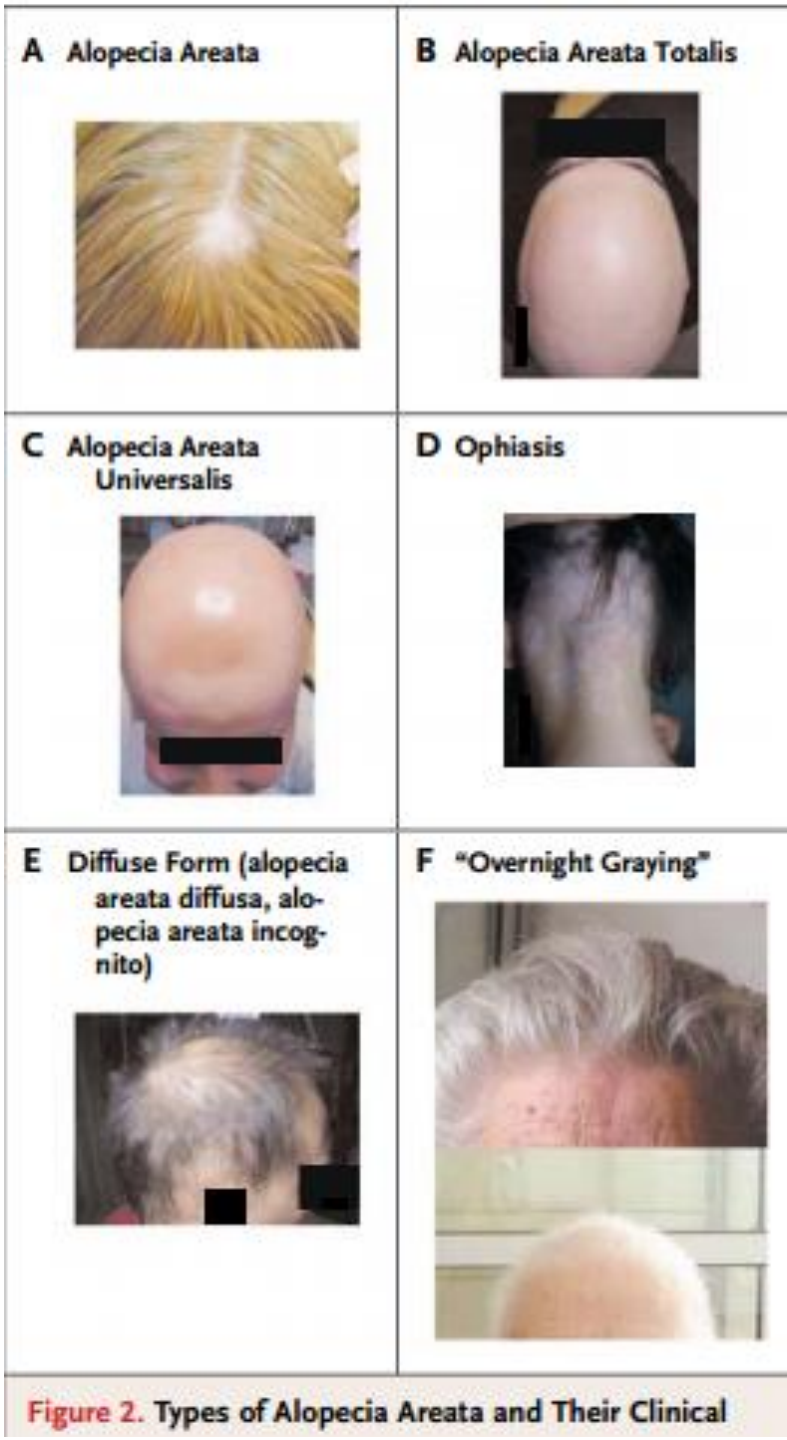


Figure 2. Types of Alopecia Areata and Their Clinical

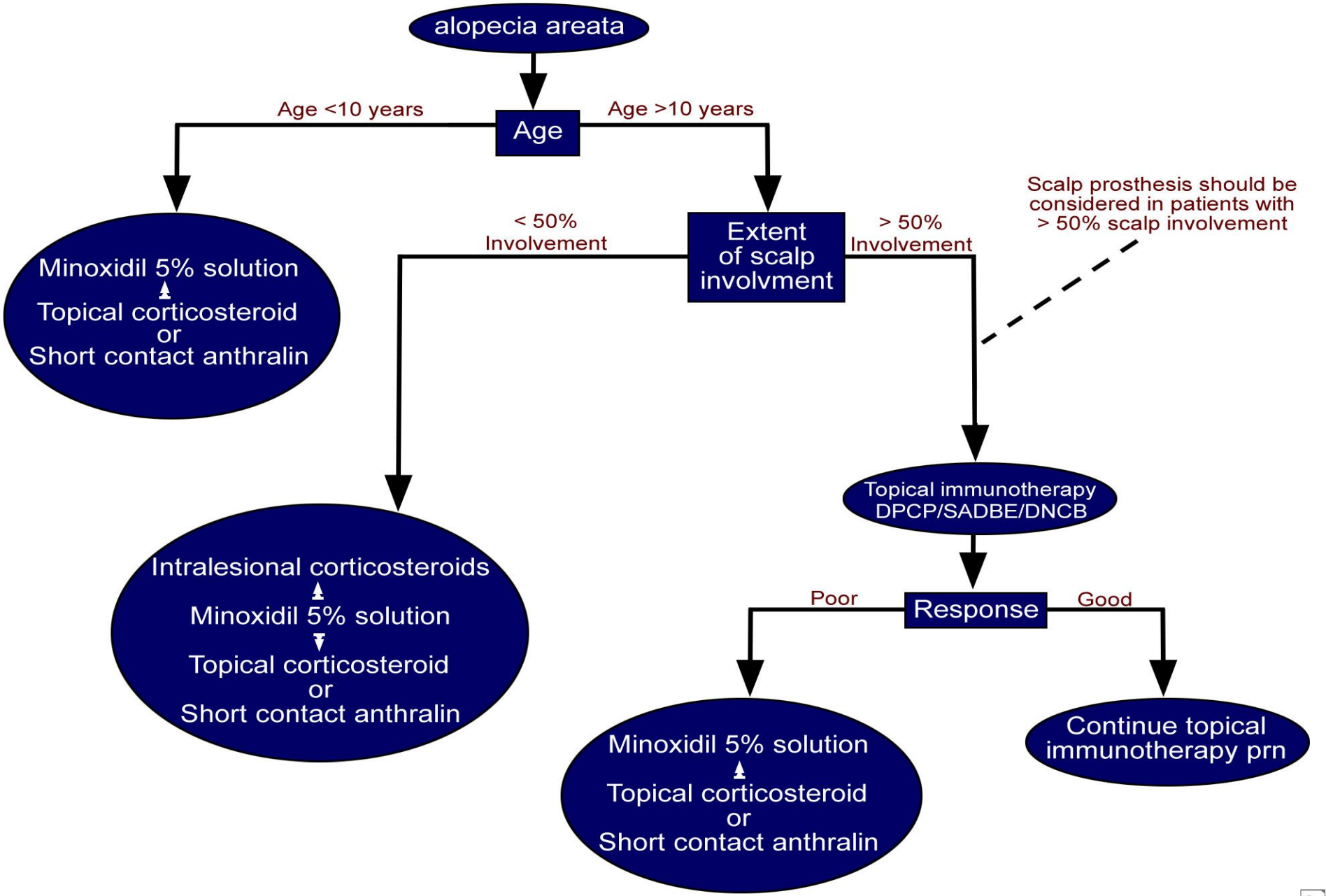
Table 1. Diagnostic Criteria for Alopecia Areata.

Diagnostic Tool	Diagnostic Findings
Family history	Atopy, thyroid disease, or other autoimmune disorders may be associated with alopecia areata; a family history of any of these disorders may therefore be diagnostic
Physical examination	
Hair and skin	Most characteristic diagnostic finding is the presence of circumscribed, hairless patches or large alopecic areas in otherwise normal-appearing skin areas; pigmented hair is preferentially attacked and lost in active disease, whereas regrowth is frequently characterized by tufts of white hair; sudden pseudowhitening of hair is observed in a rare, rapidly progressing, diffuse variant form of alopecia areata
Nails	Nail changes, if present, are usually characterized by pitting; onychodystrophy is less common
Eyes	Ocular abnormalities include lens opacities and abnormalities of retinal pigment epithelium[43]
Dermoscopy	Yellow dots (i.e., keratotic plugs in follicular ostia) are often seen in alopecia areata[32] but are not specific for the diagnosis
Cadaver hairs	Comedo-like cadaver hairs (black dots) may also be present
Exclamation-mark hair	Distal segment of the hair shaft is broader than its proximal end, resembling an exclamation mark
Follicular ostia	Openings in the hair follicles through which the hair fiber emerges from the skin; these ostia are well preserved in alopecia areata, in contrast to the findings in scarring alopecia
Pull test*	A positive pull test at the margins of alopecic lesions that produces telogen ("club") or dystrophic anagen hairs supports a clinical working diagnosis
Laboratory tests	None of the available tests will confirm the diagnosis, but thyroid-function tests and tests for thyroid antibodies may be advisable because of the increased association between alopecia areata and thyroid autoimmunity[29]; abnormal results of thyroid-function tests, the presence of thyroid autoantibodies, or both further support a clinical or histologic working diagnosis of alopecia areata
Histologic examination†	Biopsy specimens should be obtained only if the clinical diagnosis is in doubt; on histologic examination, a dense, peribulbar lymphocytic infiltrate is seen in acute alopecia areata

20%

10 – 66%

Treatment protocol for alopecia areata



Hirsutismus

increased hair growth in women – male pattern

very common

nearly always genetic in origin

Some women have increased amounts of androgens

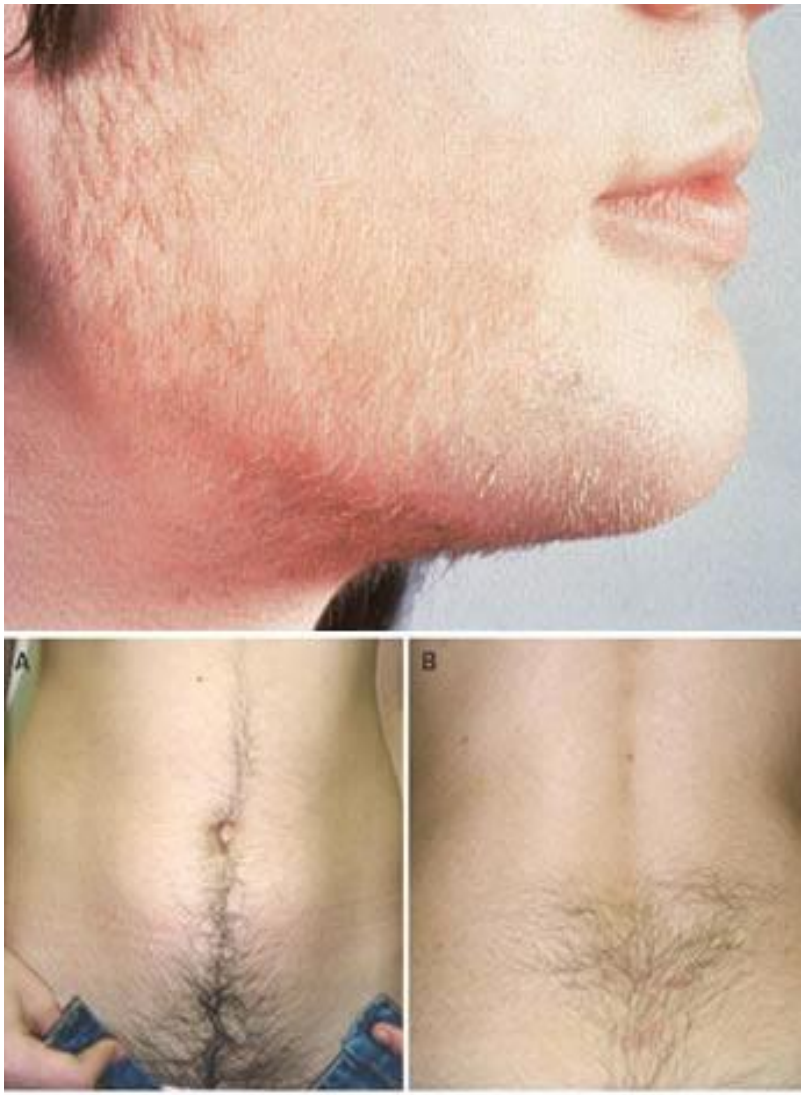


Figure 1

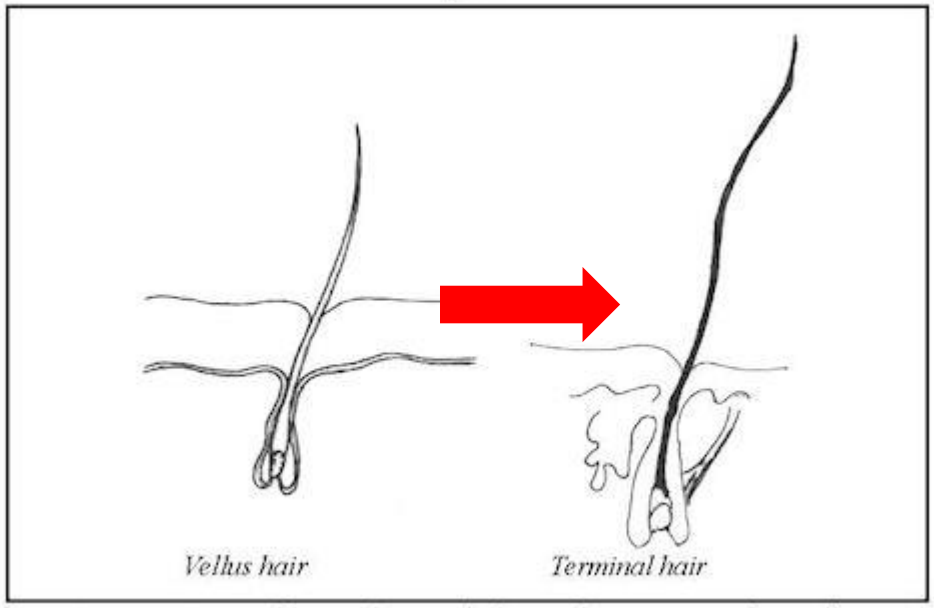
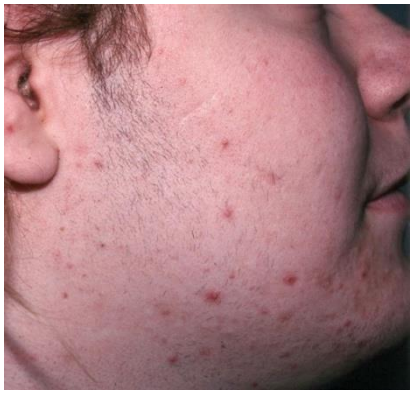


Figure 1. Terminal hair is longer, darker, and more coarse than vellus.

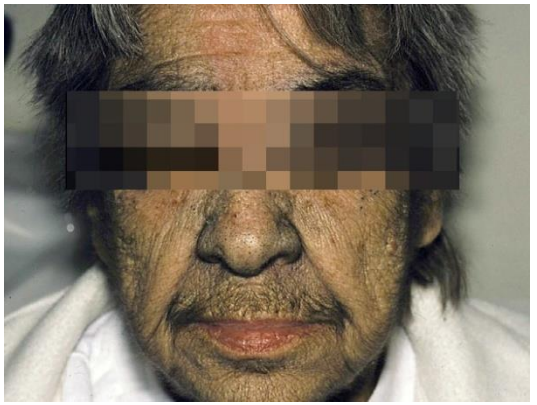
Table 2. Causes of hirsutism

- Excessive production of androgens by the ovaries (polycystic ovary syndrome, tumor)
- Excessive sensitivity of hair follicles to androgens (genetic)
- Excessive production of androgens by the adrenal glands (non-classical adrenal hyperplasia [NCAH])
- Insulin resistance
- Hyperandrogenism, insulin resistance, acanthosis nigricans (HAIR-AN syndrome)
- Excessive production of cortisol by the adrenal glands (Cushing syndrome)
- Menopause
- Medications



SIMPTOMATIC

IDIOPATIC



Ferriman–Gallwey scor

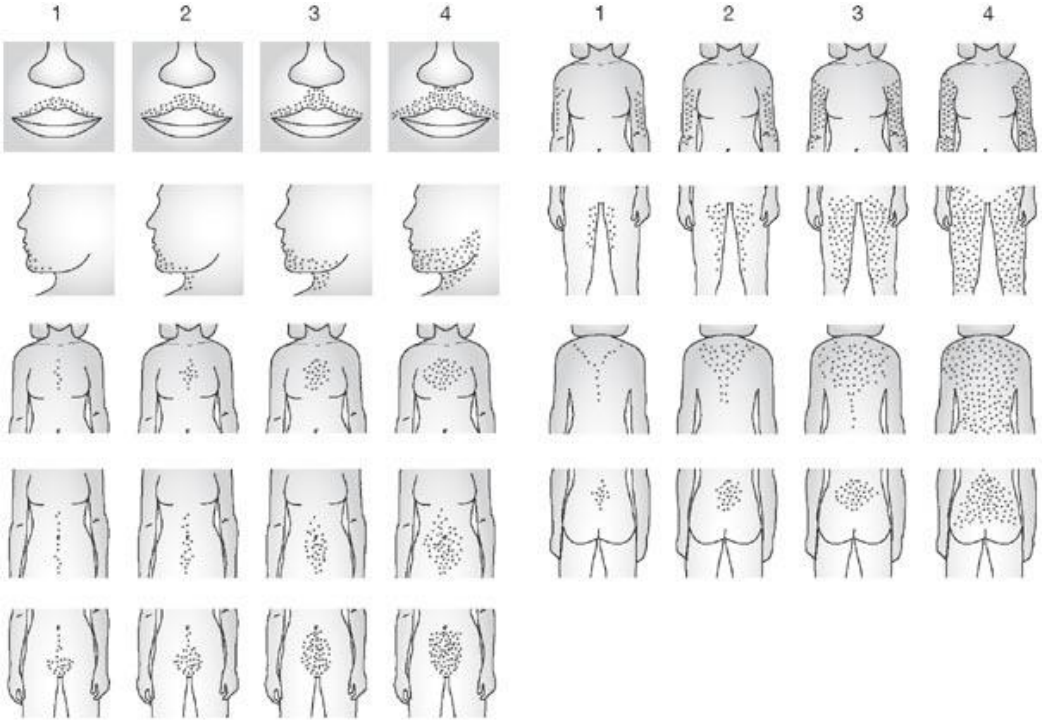
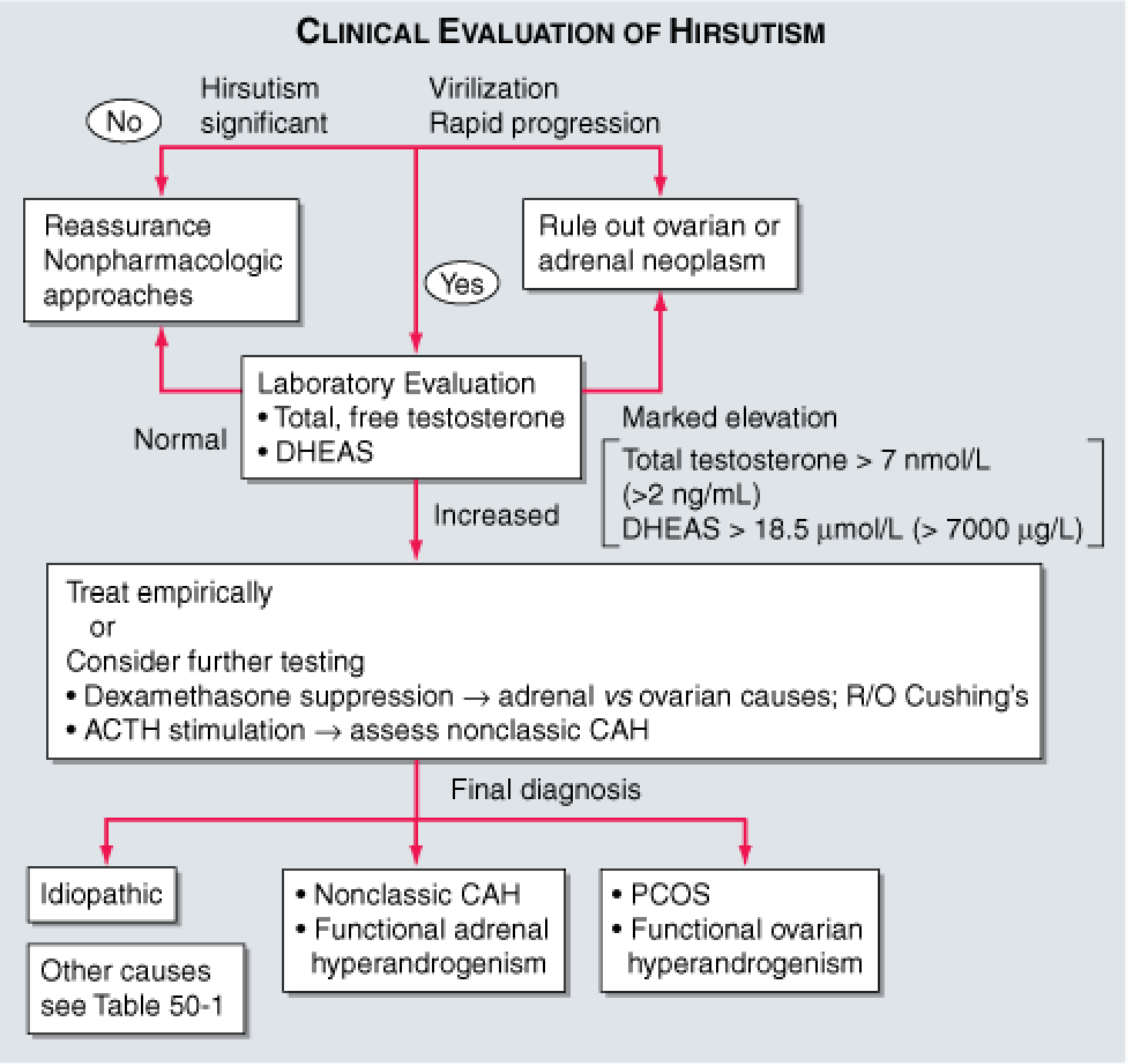


Table 1. Androgen-sensitive sites of hair growth

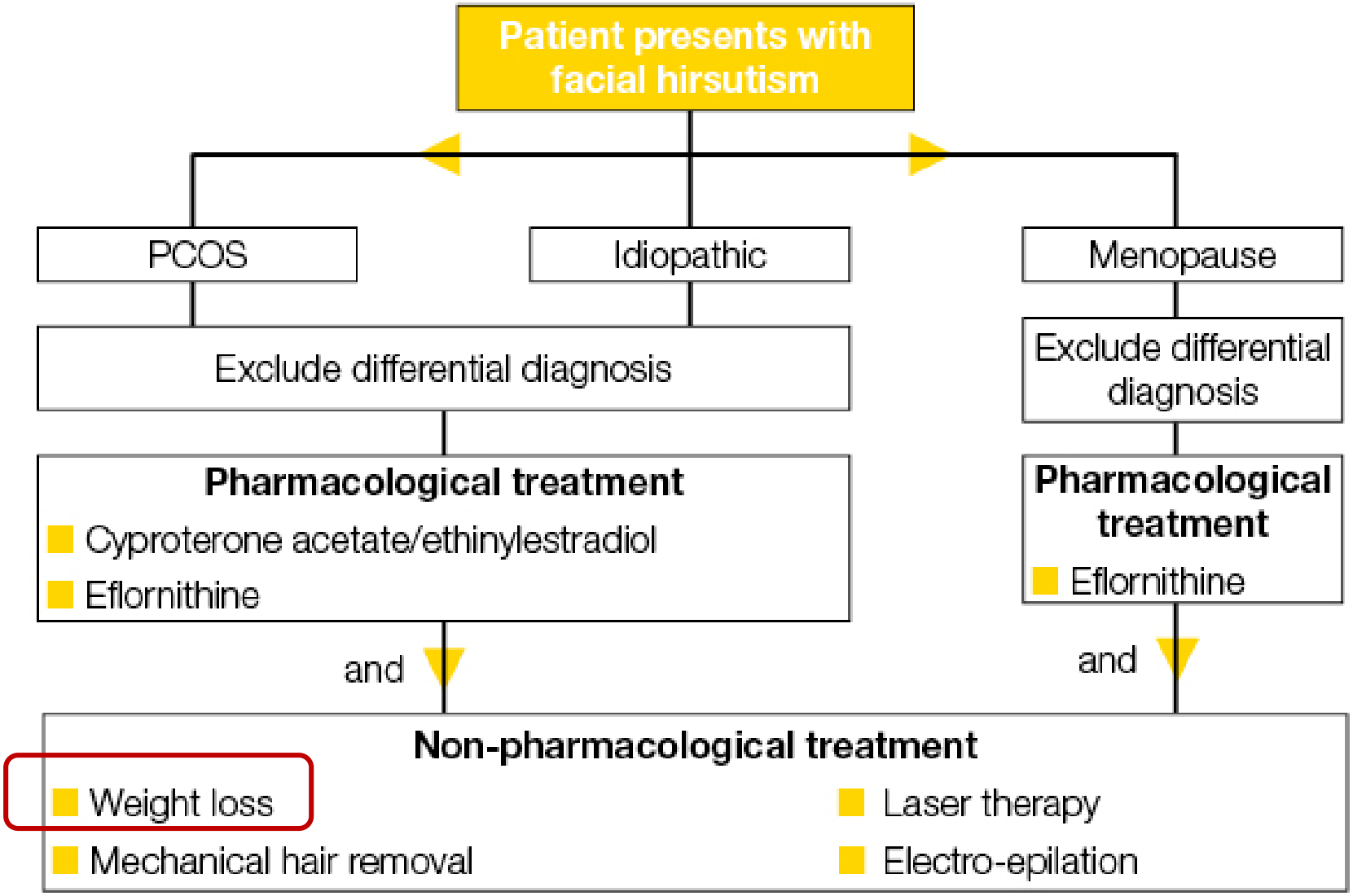
More common	Less common
Upper lip	Chest and sternum
Beard area	Upper abdomen
Breasts	Upper back
Lower abdomen	
Inner thighs	
Lower back	



Source: Fauci AS, Kasper DL, Braunwald E, Hauser SL, Longo DL, Jameson JL, Loscalzo J:

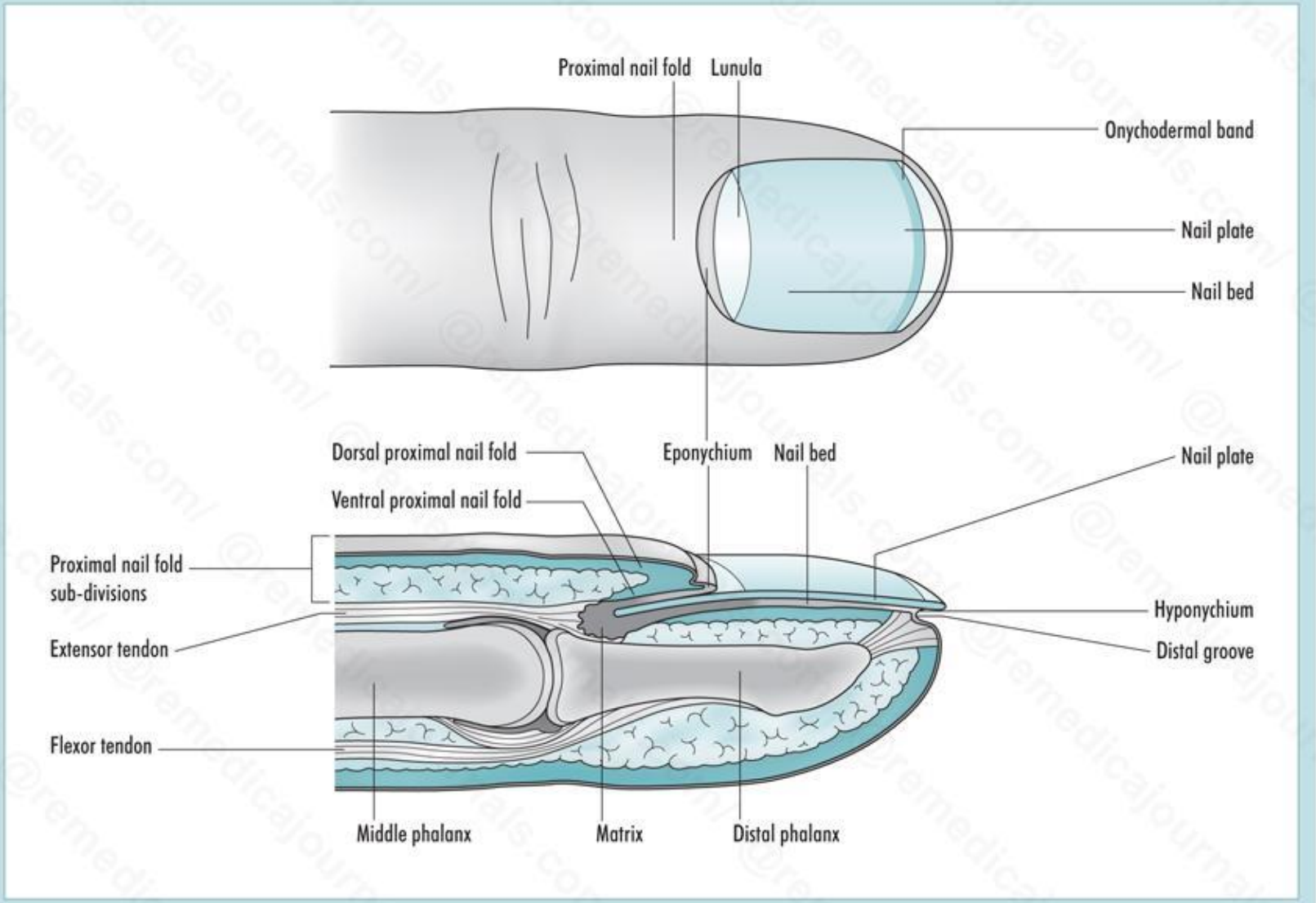
Table 3. Treatment of hirsutism

Medications	Cosmetic treatments
Birth control pills	Shaving
Androgen receptor blockers	Eflornithine cream
Spironolactone	Waxing
Flutamide	Bleaching
Glucocorticosteroids	Plucking
Dexamethasone	Depilatory agents
Prednisone	Electrolysis
Methylprednisolone	Laser
Enzyme inhibitors	
Finasteride	
GnRH analogs	

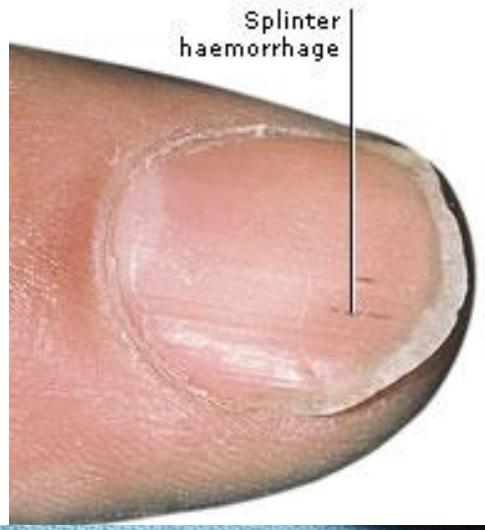

Patient presents with facial hirsutism
PCOS
Idiopathic
Menopause
Exclude differential diagnosis
Exclude differential diagnosis
Pharmacological treatment
Cyproterone acetate/ethinylestradiol
Eflornithine
Pharmacological treatment
Eflornithine
and
and
Non-pharmacological treatment
Weight loss
Mechanical hair removal
Laser therapy
Electro-epilation

NAIL

Figure 1. Longditudinal cross-section displaying the dorsal and ventral proximal nail folds of the nail unit.

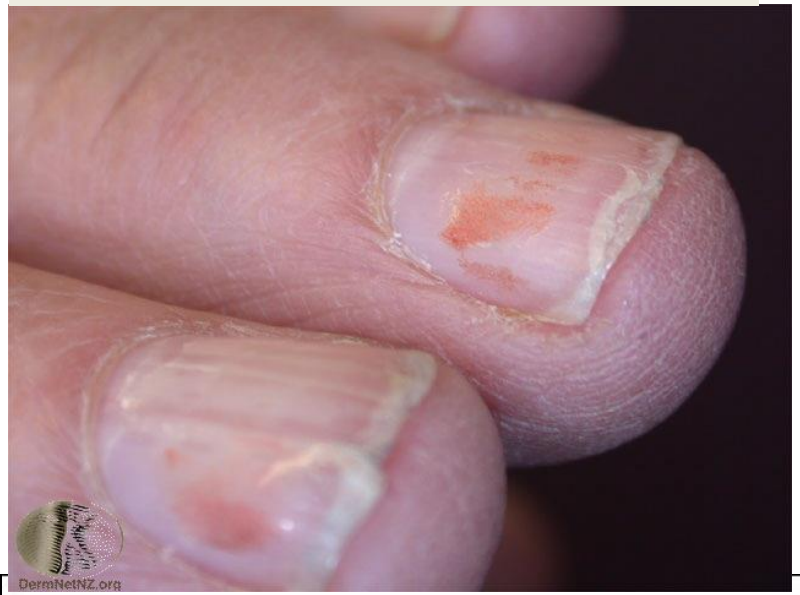


Nail **trauma**



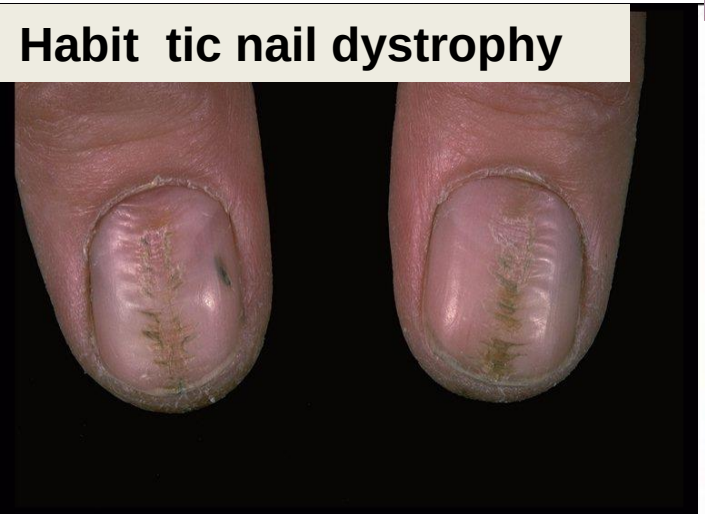
Onycholysis

Distal lamellar splitting; brittle nails

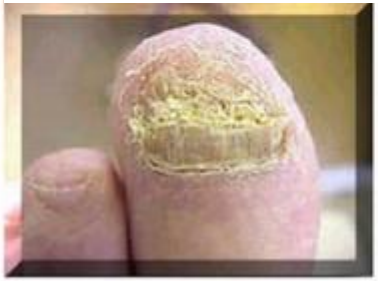


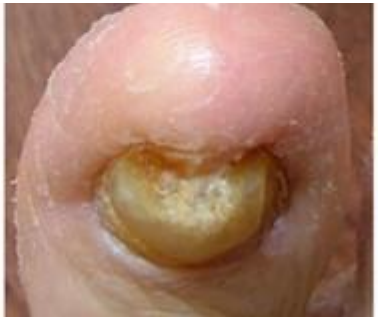
Subungual haemorrhagia

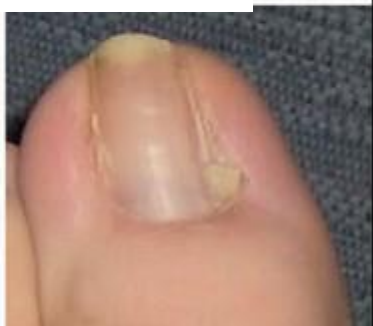
Habit tic nail dystrophy



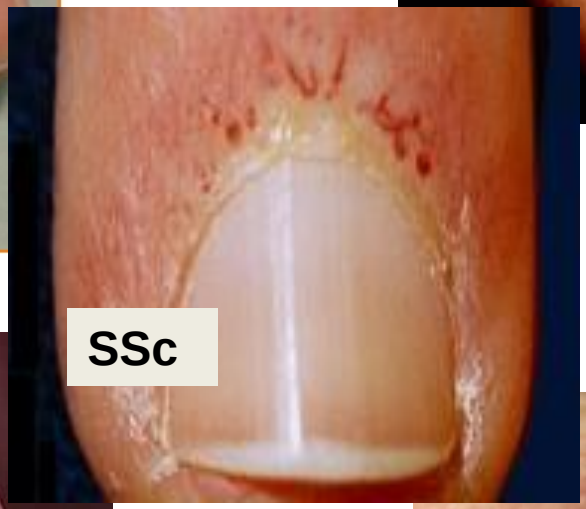
Onychogriphosis

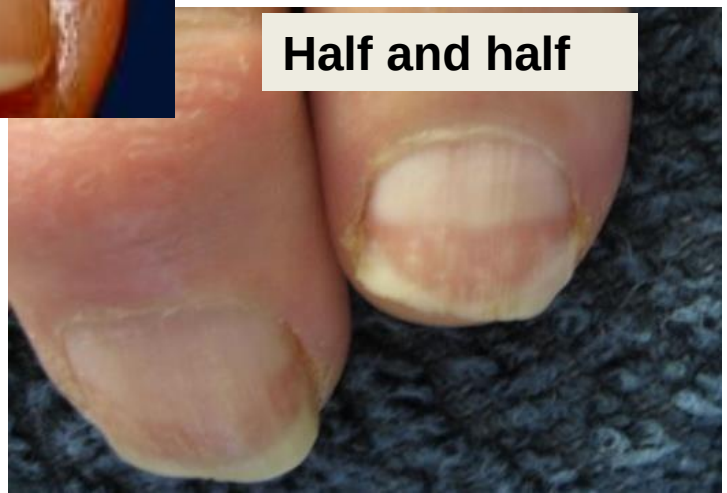






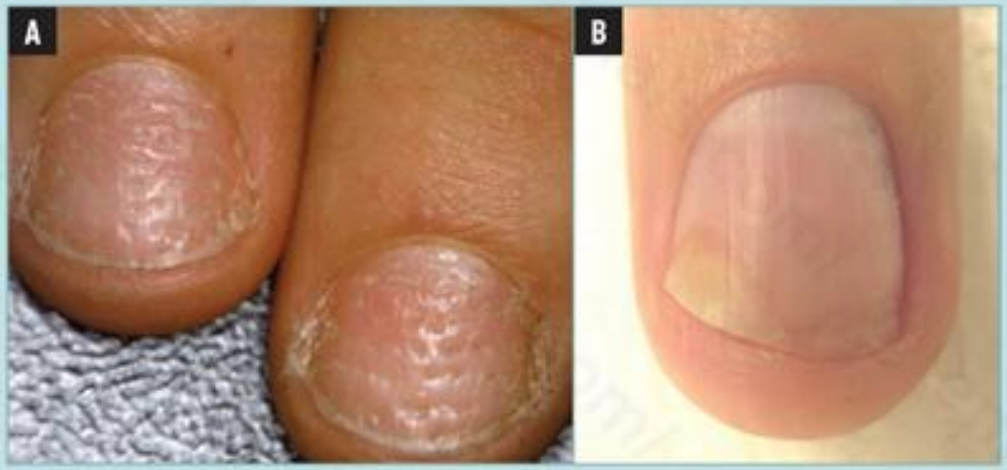
Nail in systemic disease

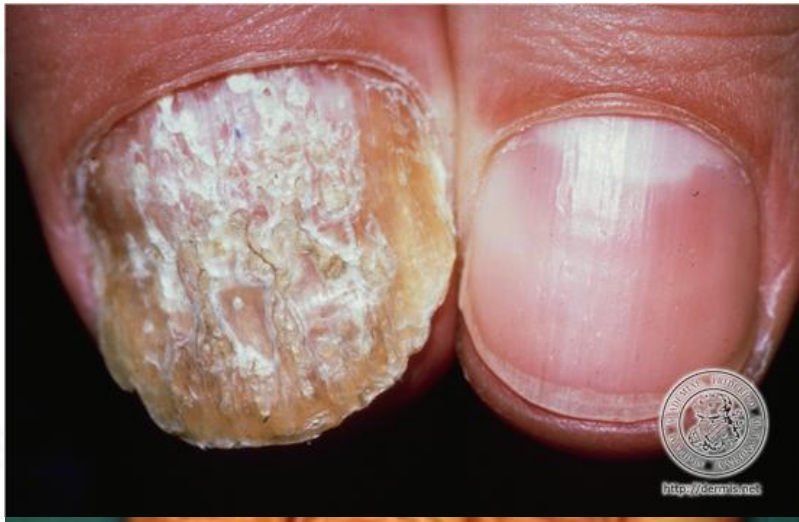


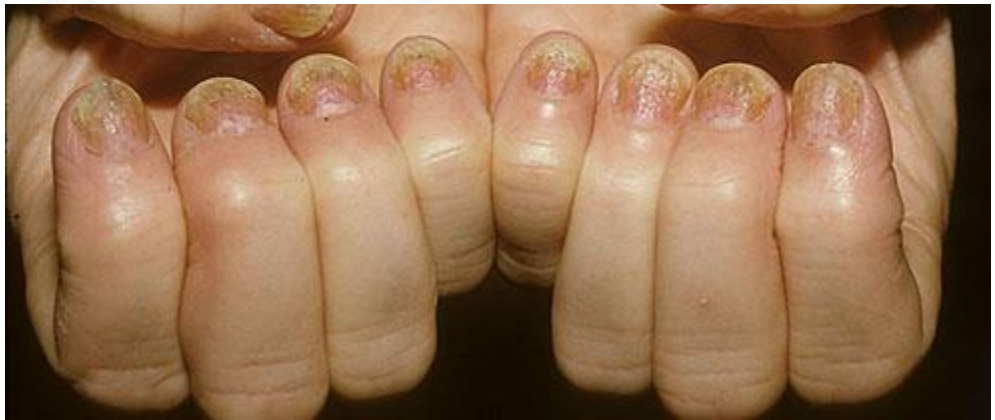


Nail psoriasis

Figure 2. A: Pitting of the fingernail. **B:** Onycholysis with an erythematous border.

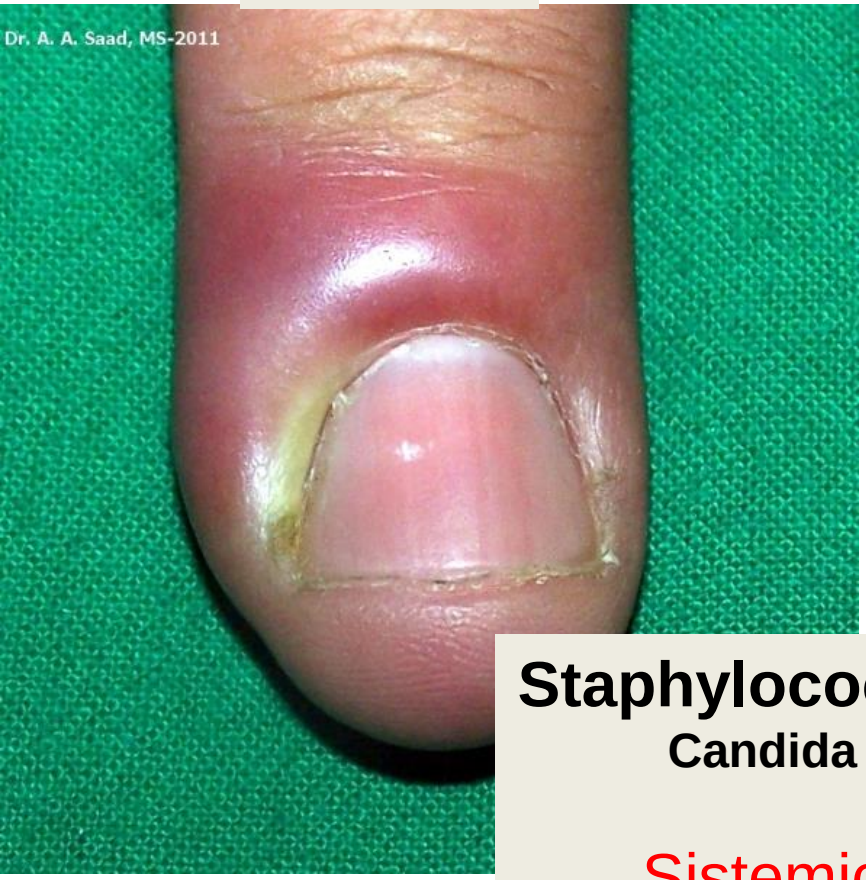






Paronychia

ACUTE



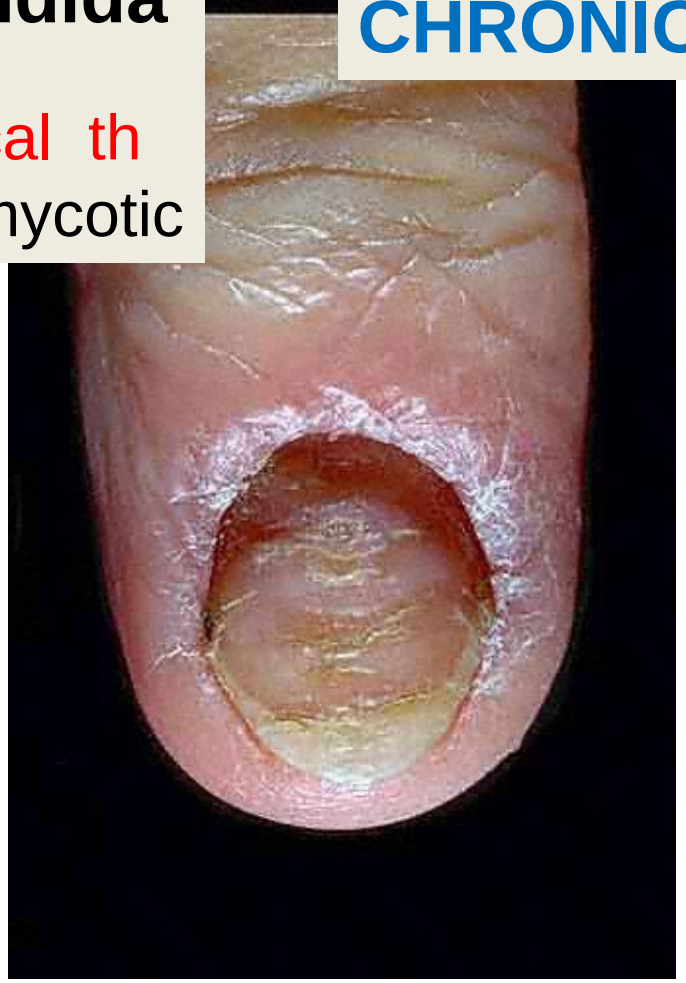
Staphylococcus
Candida

Sistemic
Antibiotic therapy

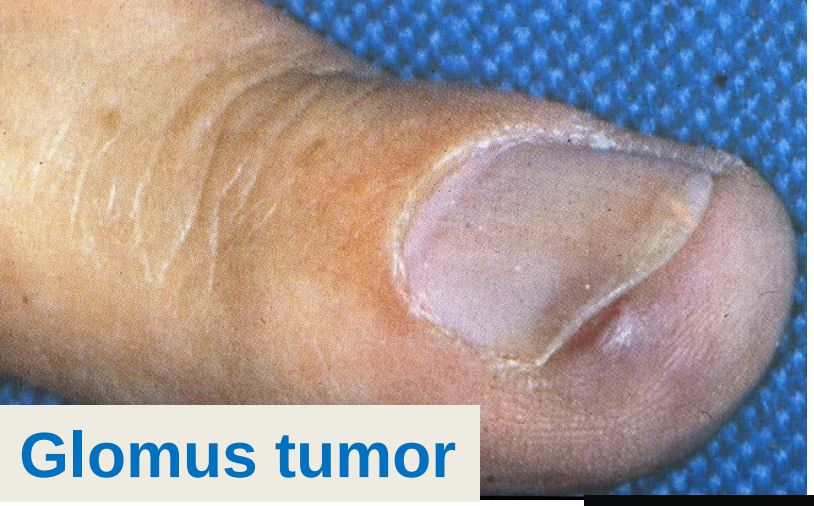
CHRONIC

Candida

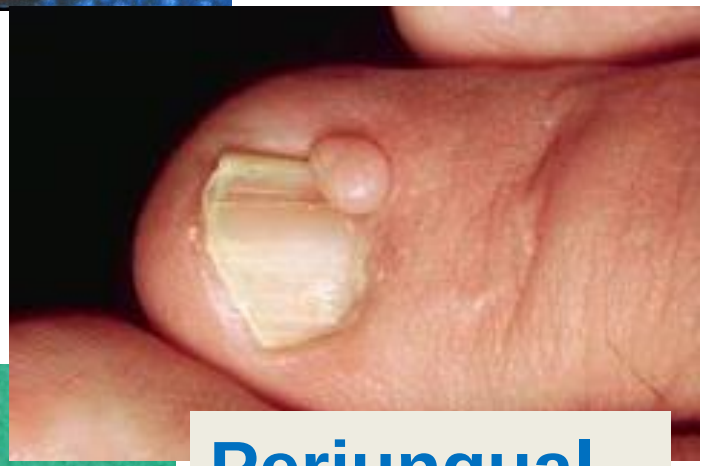
Local th
Antimycotic



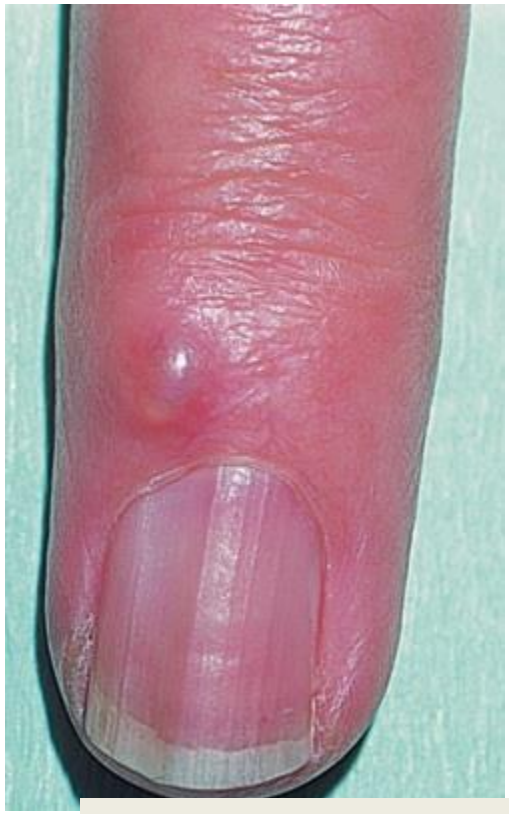
Nail tumors



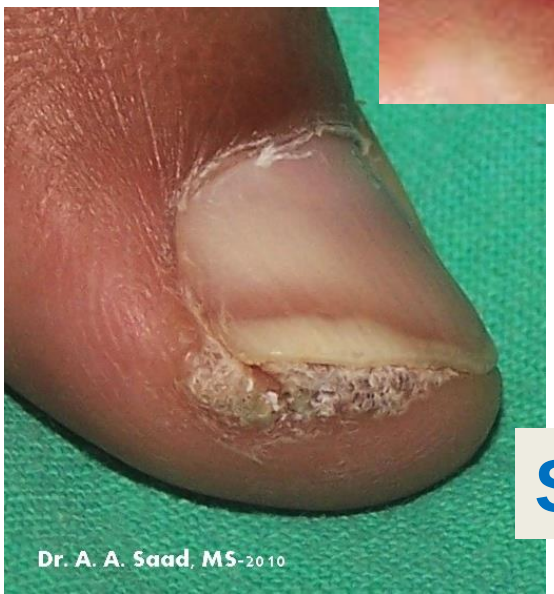
Glomus tumor



Periungual fibroma



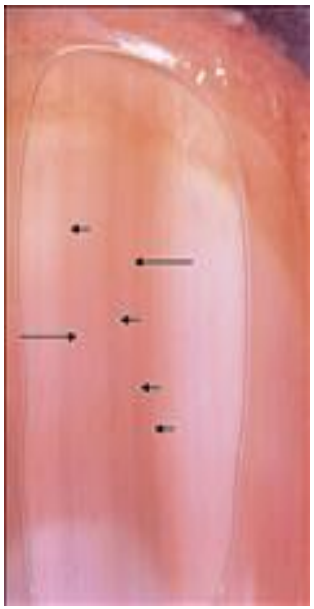
Myxoid cyst



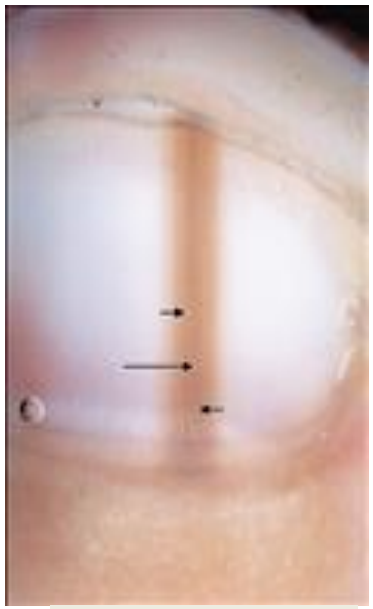
Subungual wart

Nail pigment

Subungual haemorrhage



Melanonychia striata



Naevus

MM in situ

Acr.MM