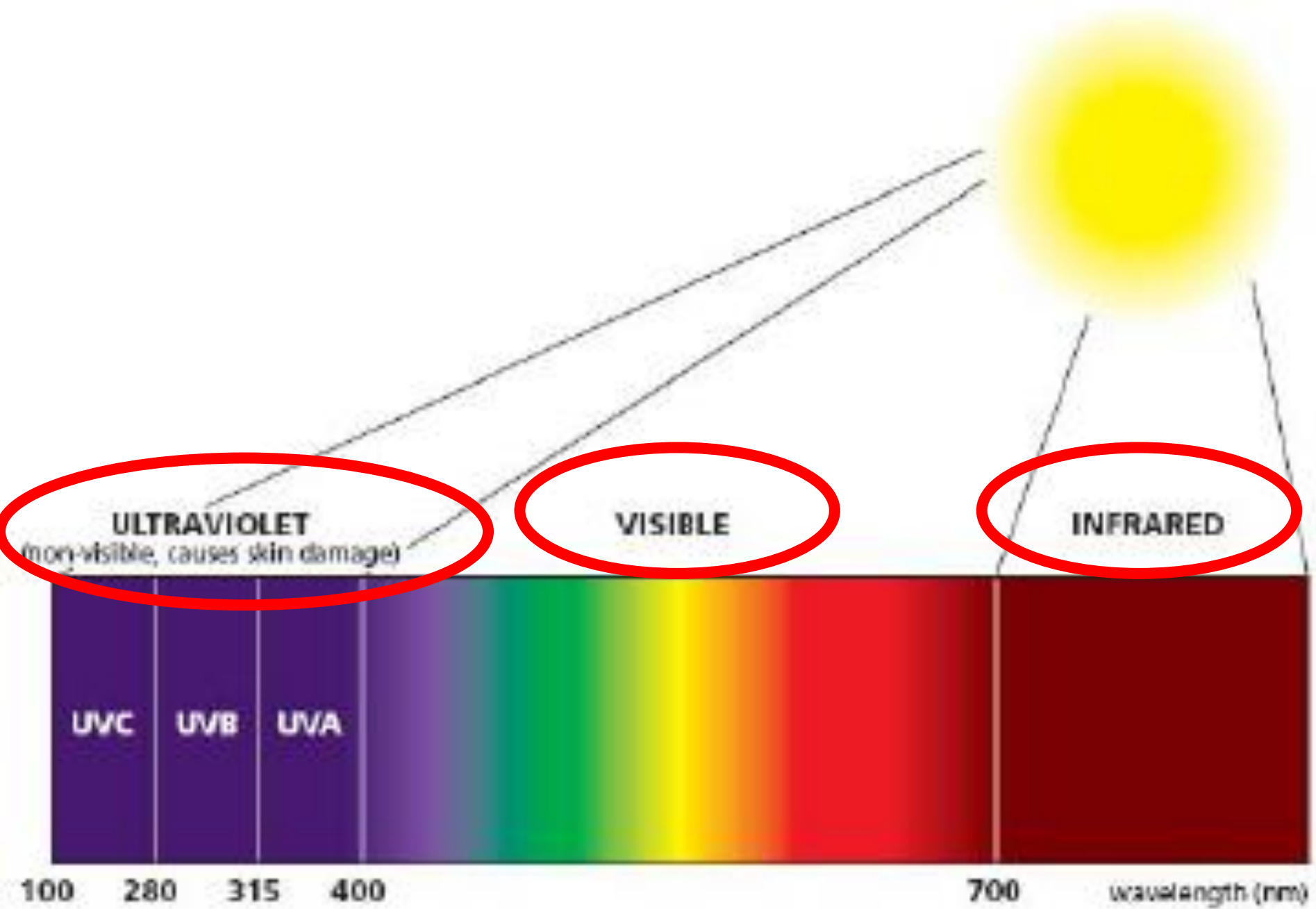


Skin tumours

Neira Puizina-Ivić, Ph.D, Prof.



UV exposure

pros

attractive skin
good mood
production of D vitamine
treatment of psoriasis
and other skin diseases

cons

skin burns
photoaging
skin cancer
photosensibilisation
exacerbation of skin diseases
(lupus, porphyria...)

without UV exposure

1000x lower production D vitamin - **rickets!**
osteoporosis
depression
bad mood
slower aging of the skin



be moderate with
selection of SPF

UVC
200-
280nm

UVB
280-
320nm

UVA
320-
400nm

5%

95%



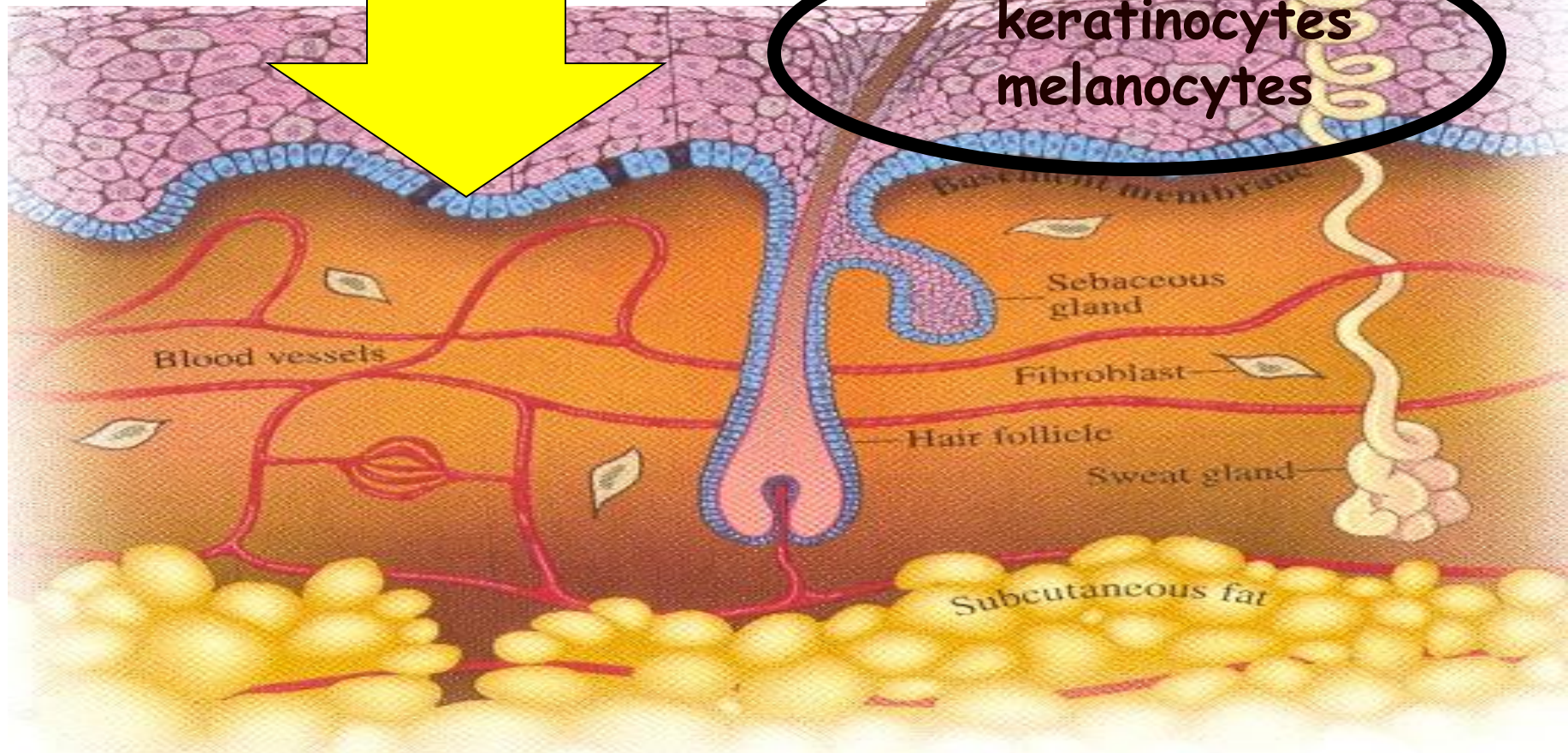
UVA - 95%

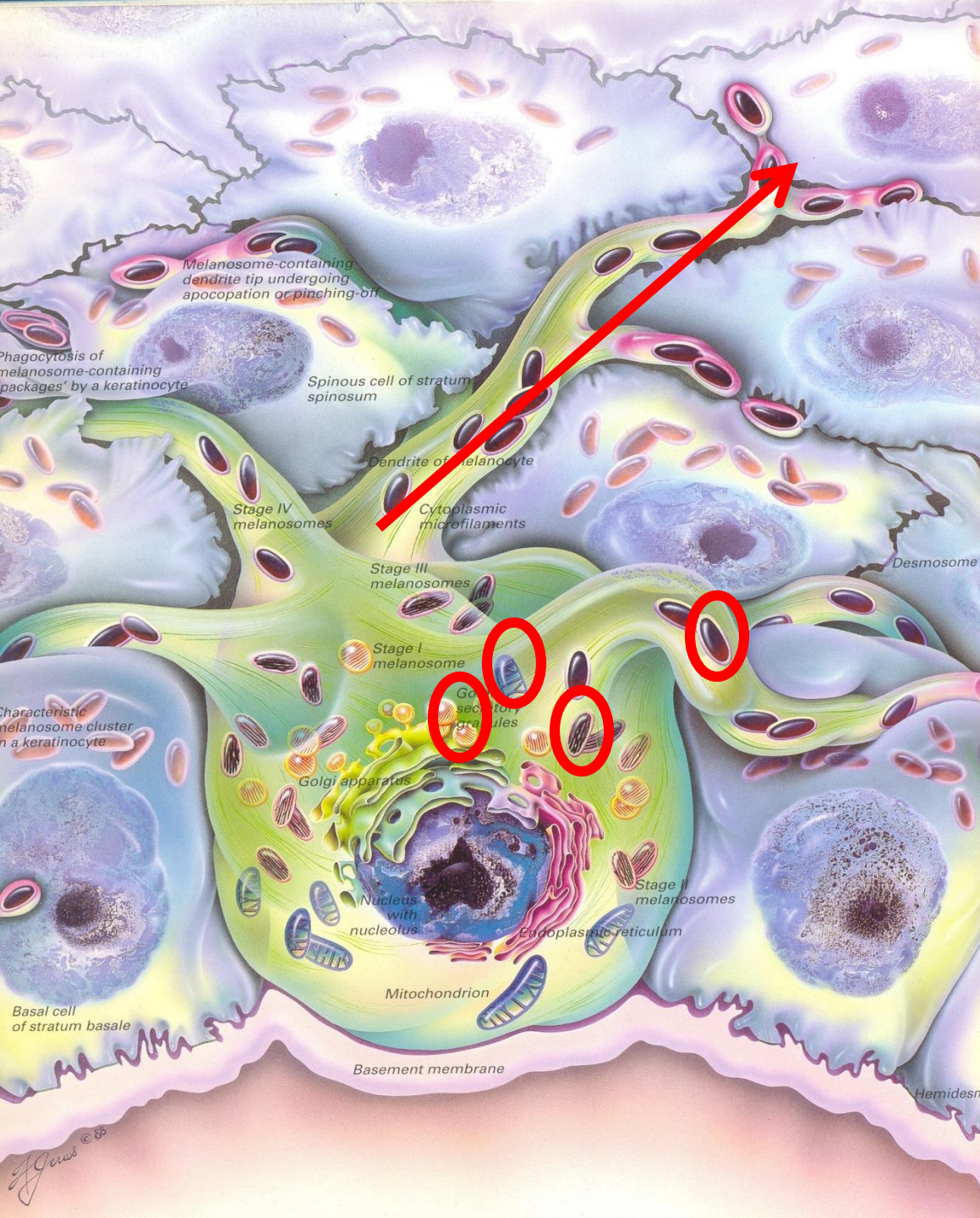
UVB - 5%

1000x stronger biologic
effect than UVA

UVB

keratinocytes
melanocytes





epidermal-melanin unit

face 1/5 keratinocytes

trunk 1/36 keratinocytes

KERATINOCYTES

Keratinocyte

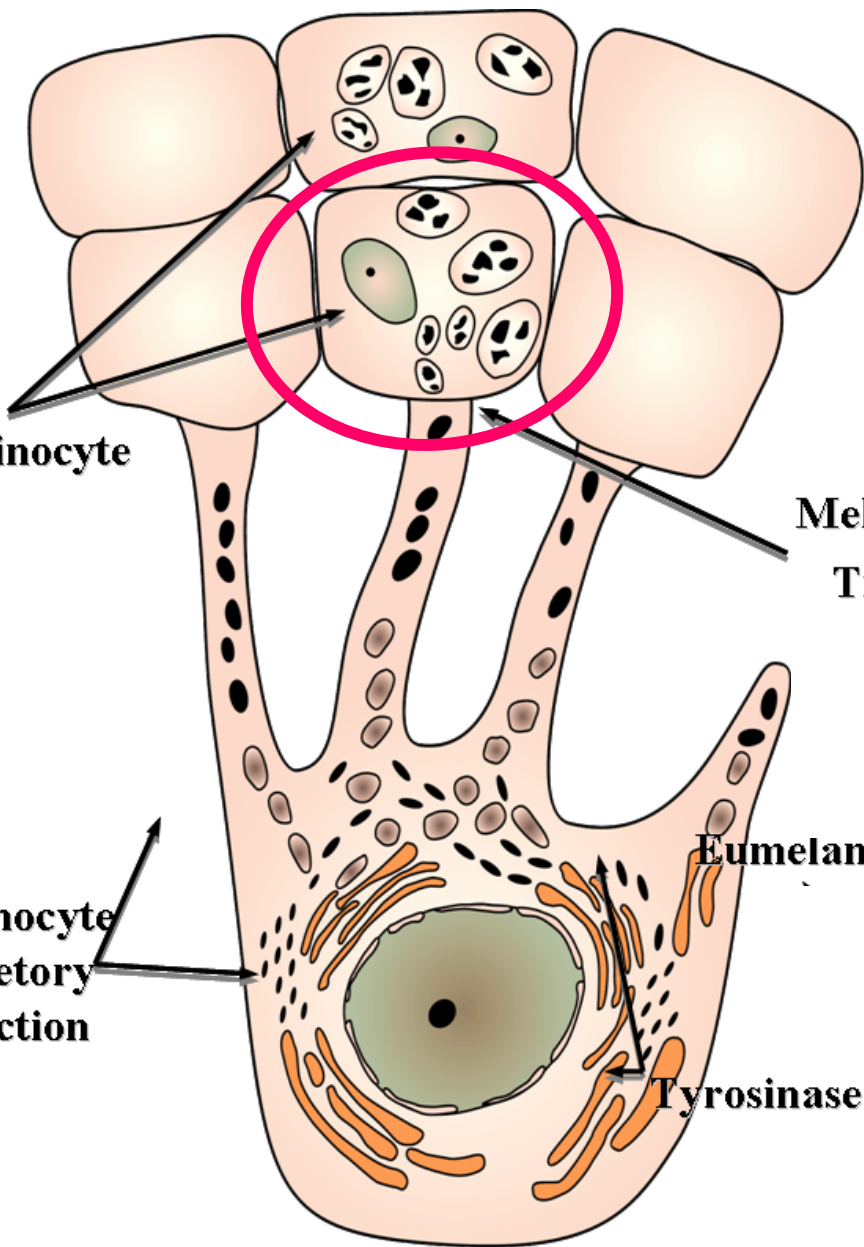
Melanosome
Transfer

MELANOCYTE

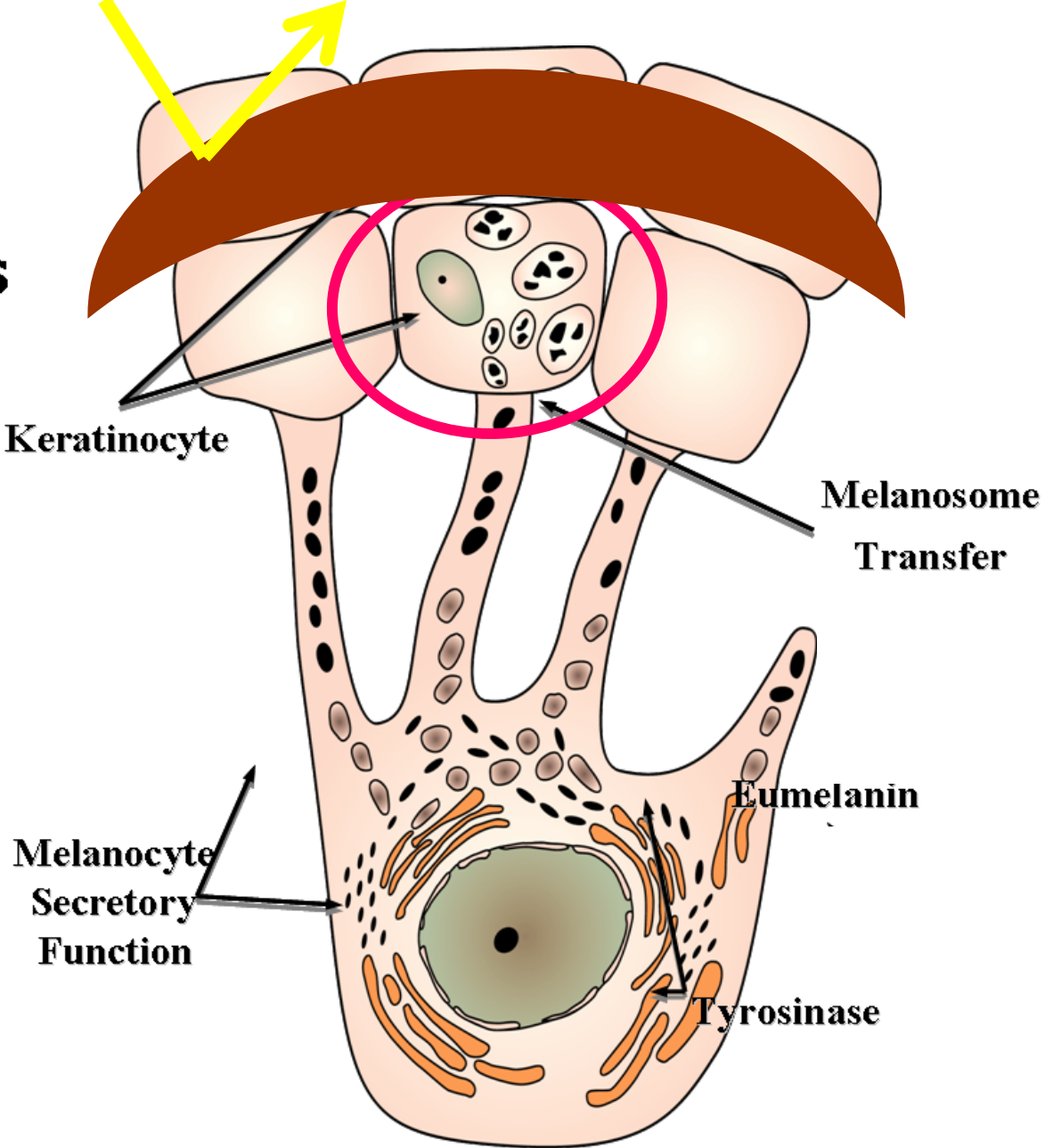
Melanocyte
Secretory
Function

Eumelanin

Tyrosinase



KERATINOCYTES



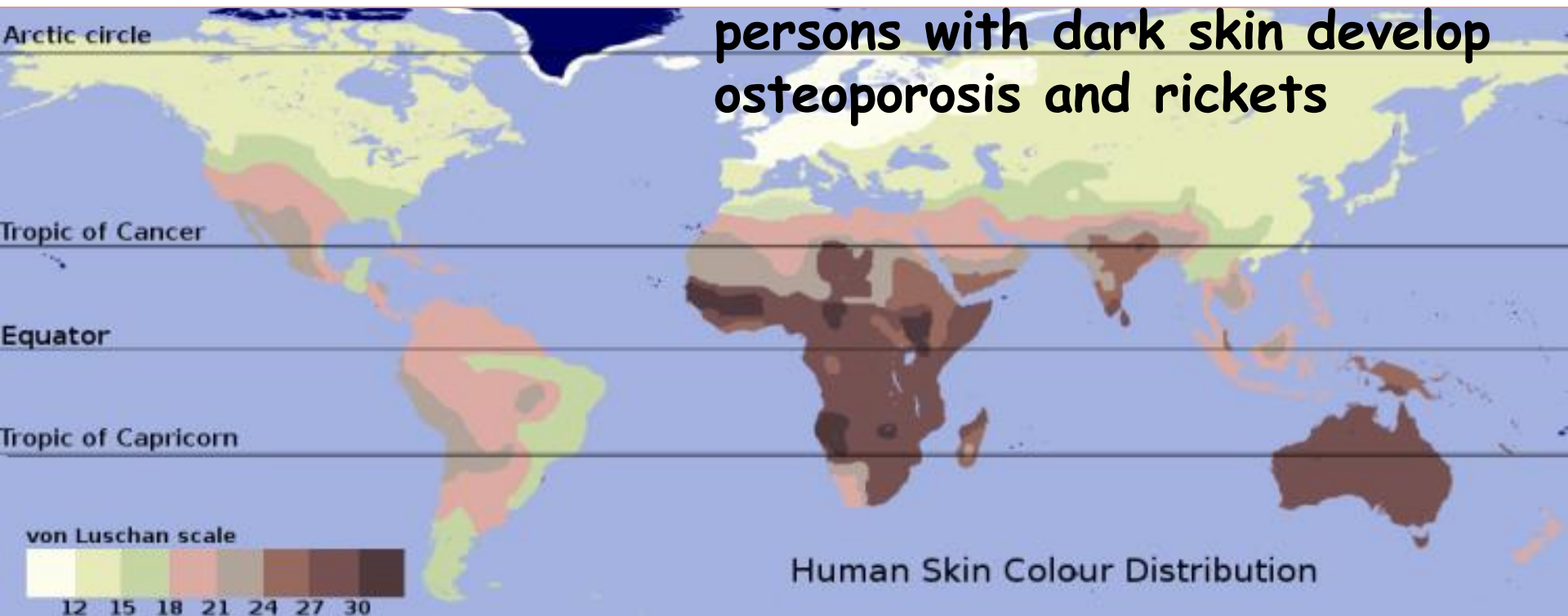
MELANOCYTE

Melanocyte
Secretory
Function

Melanosome
Transfer

Eumelanin

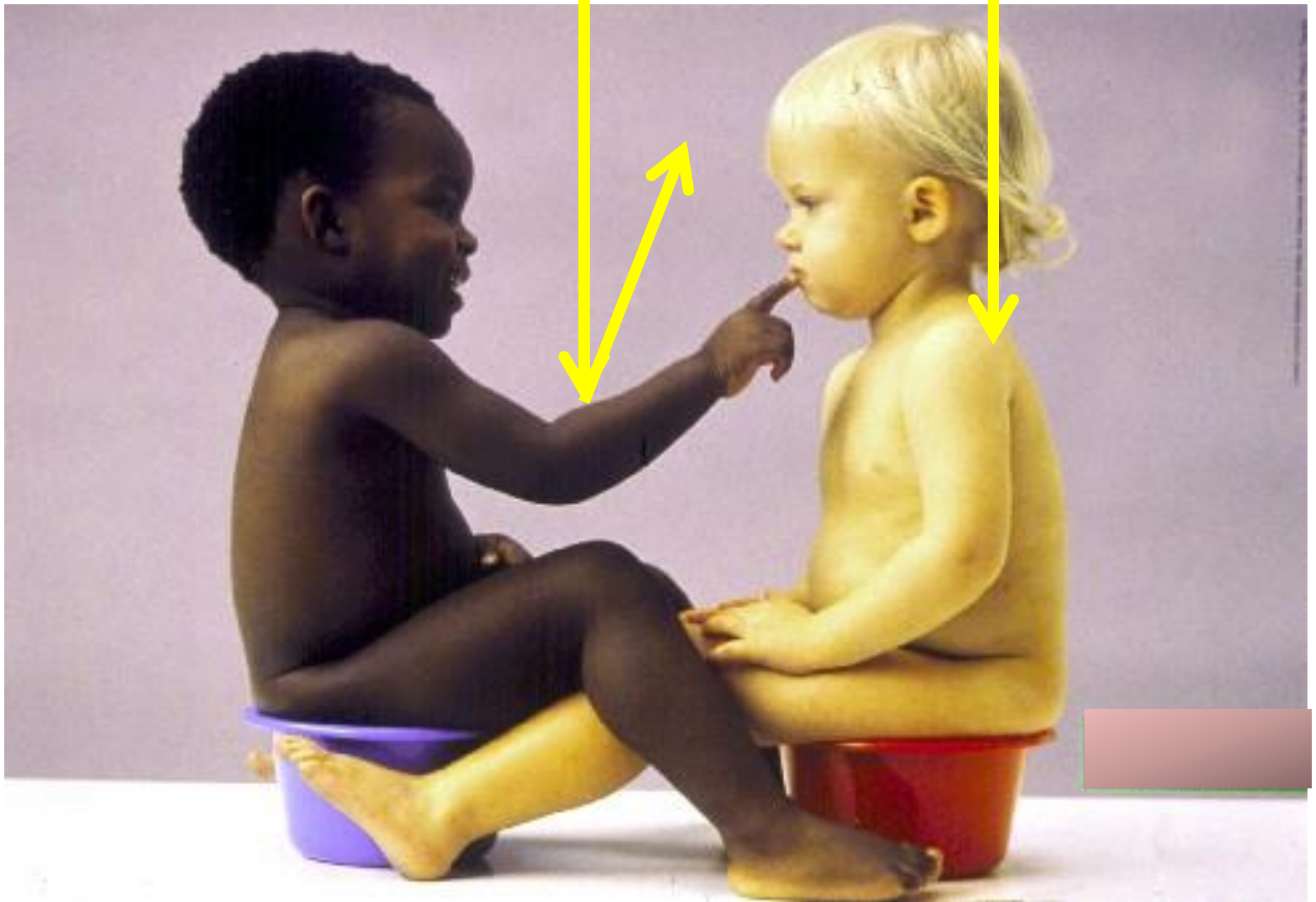
Tyrosinase



persons with dark skin develop osteoporosis and rickets

pigmentation augments heat absorption (30% in dark skin)

Berzelius 1840.g dao naziv melanin

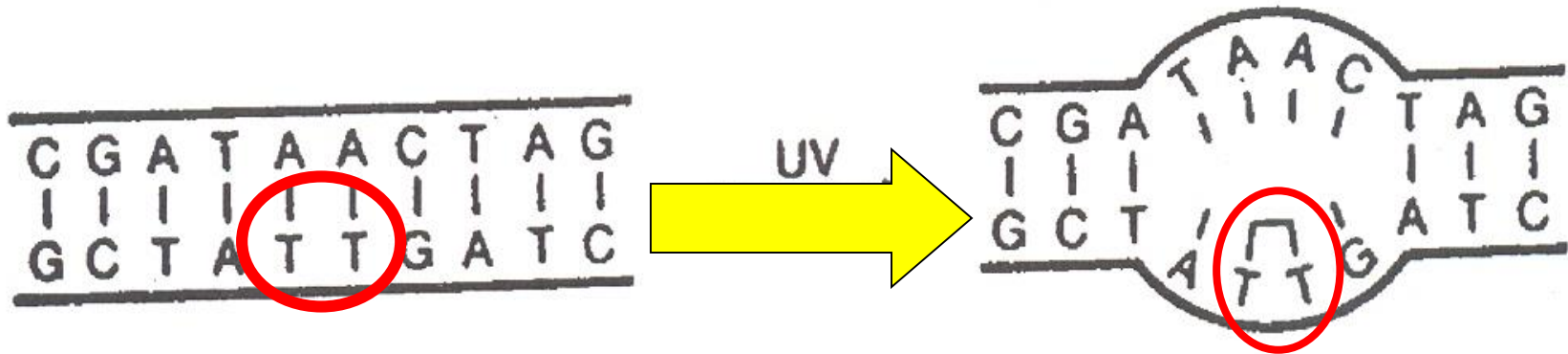


rickets



osteoporosis

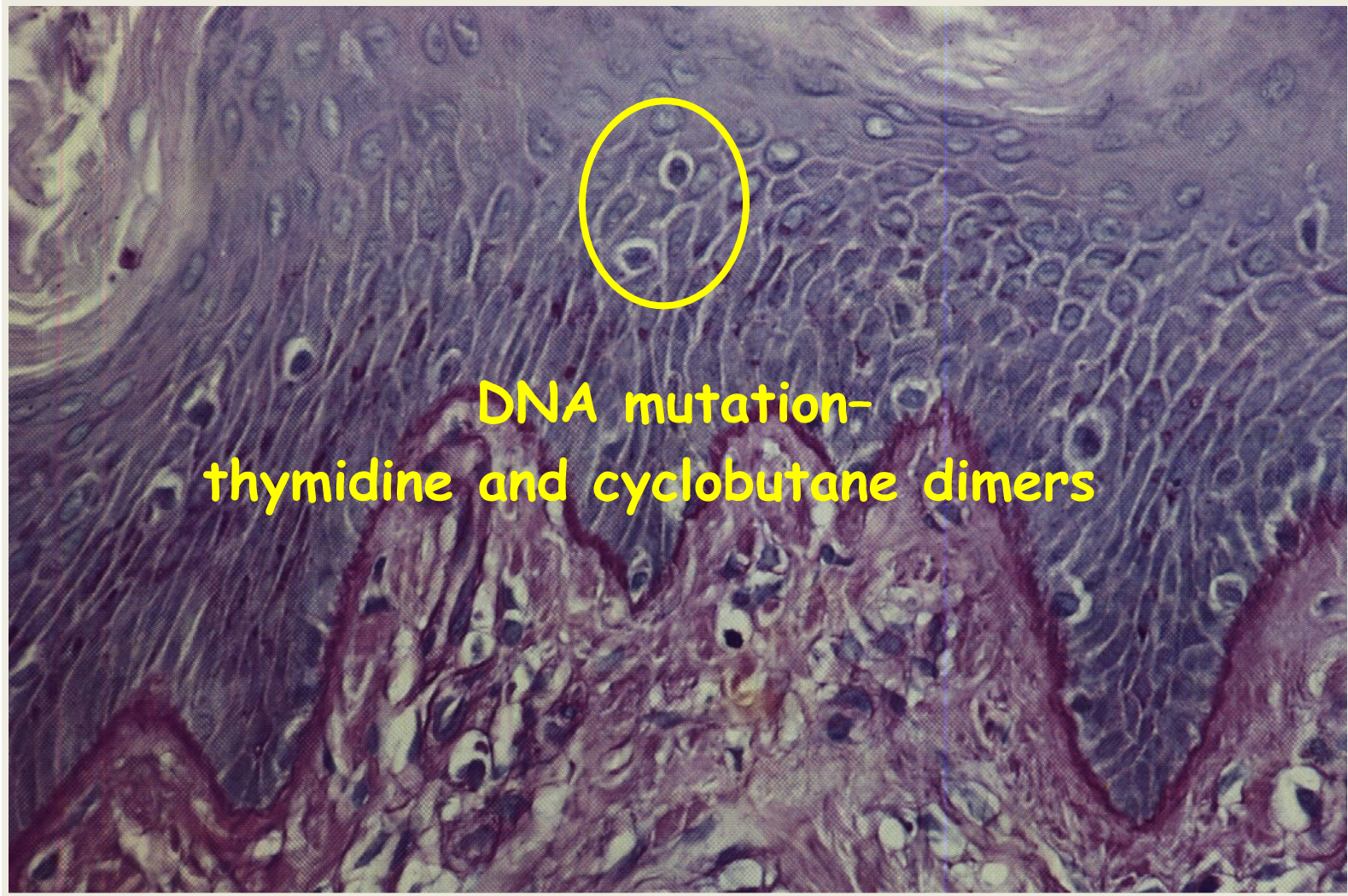
Thymidine's dimers



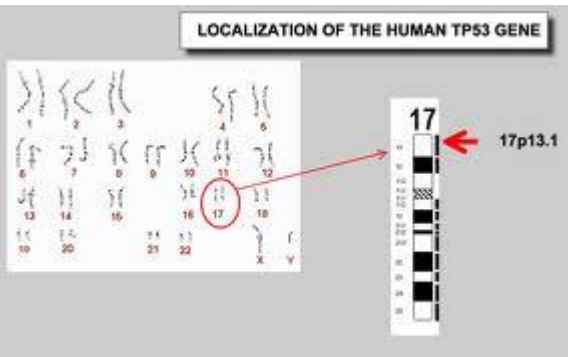
"UV fingerprint"

8-12 hours- sun burn cells

decreased production of DNA next 12 hours



damage of DNA
(UV, osmotic, viruses,
hypoxia stress, ROS)



p53

guardian of the
genome

stops dividing

starting of programmed cell death

cell recovery

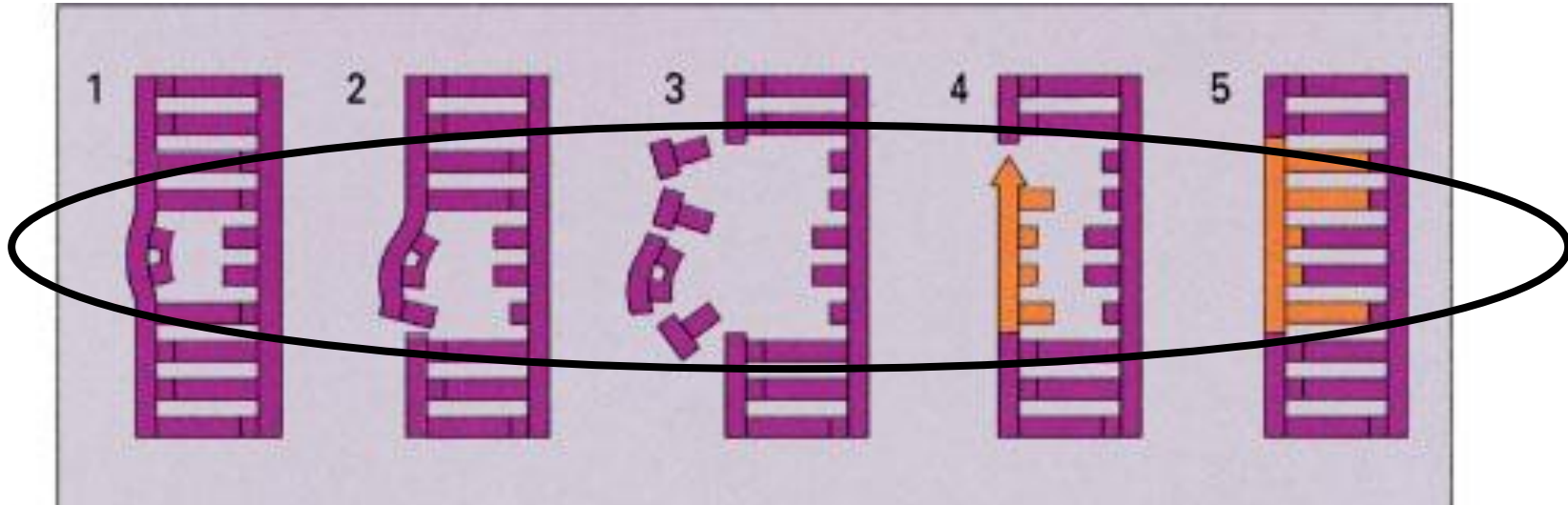
cell death

genetic stability



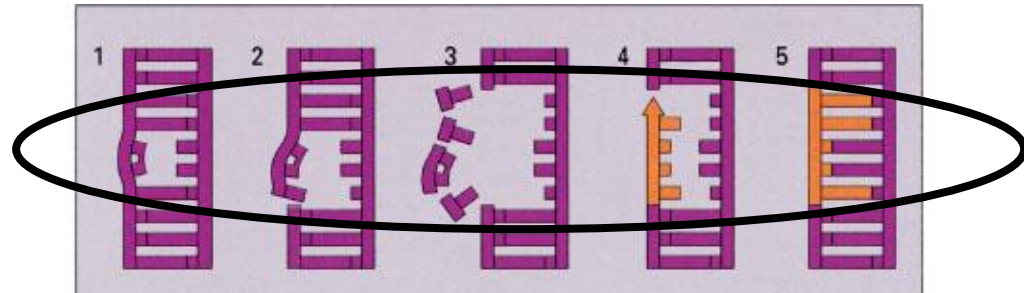
The "Samurai" law of biology:

**"It is better to die,
than to be wrong"**



repair of damaged DNA





repair of damaged DNA





accumulation of errors



20-30 years

skin cancer

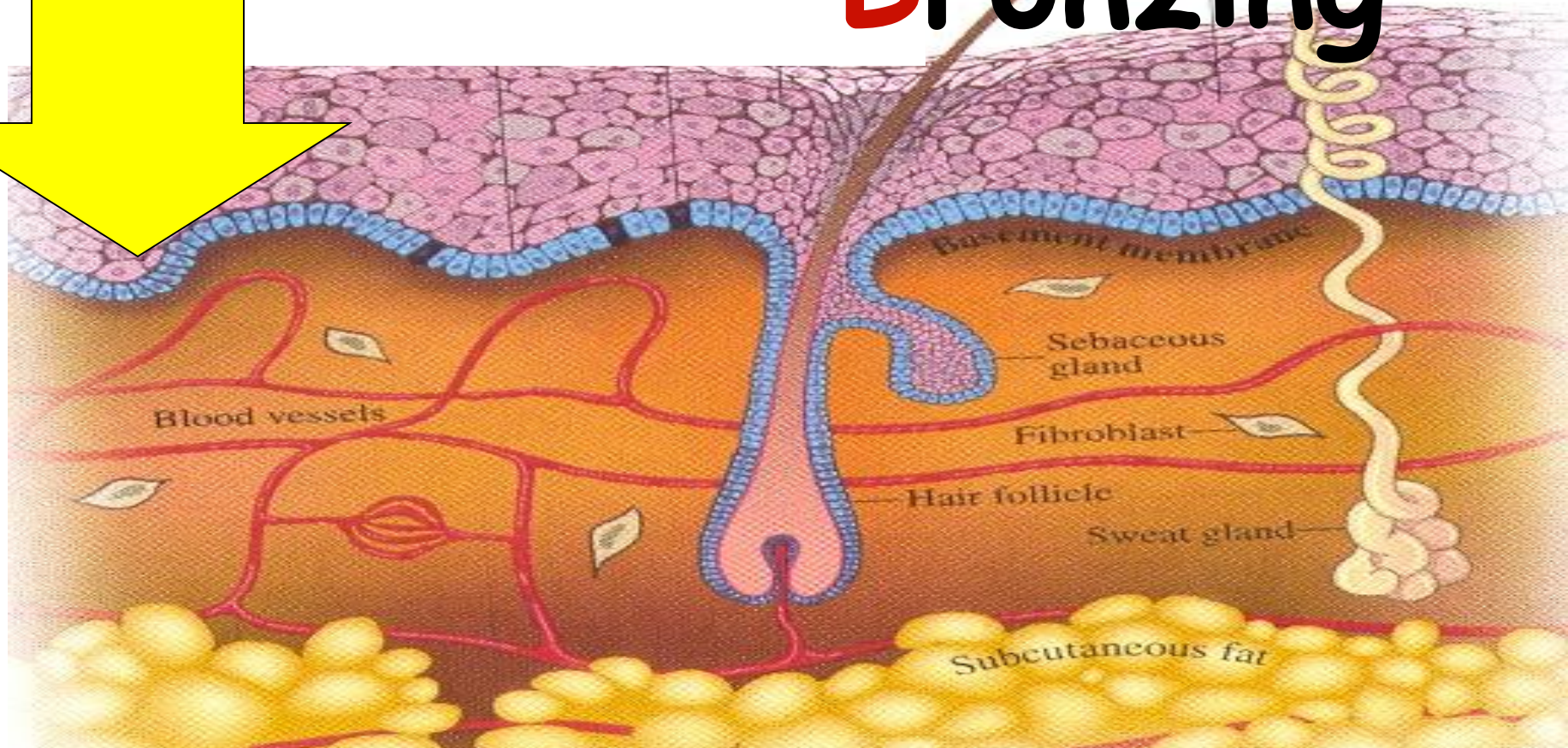




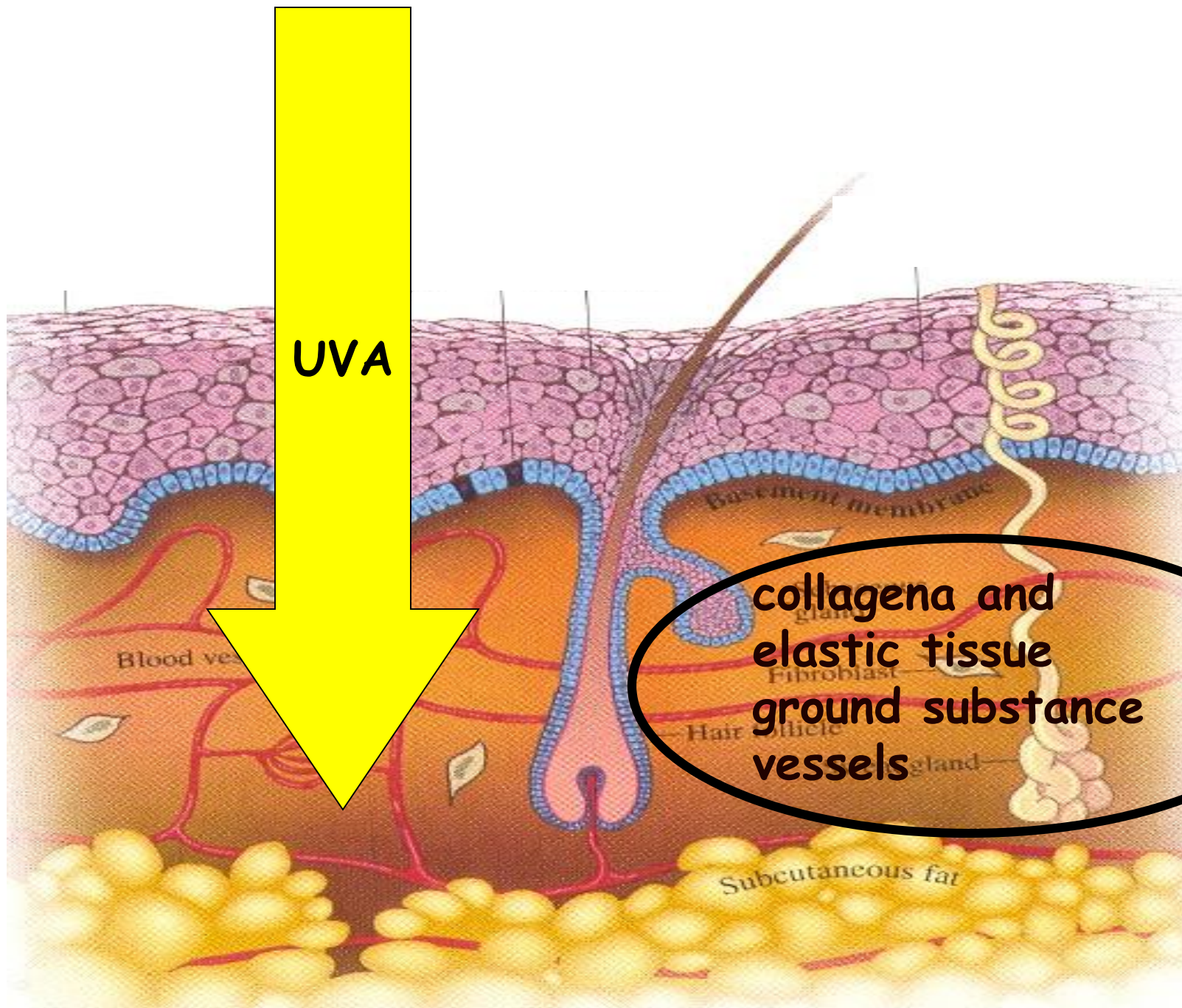
field of cancerisation

UVB Burn / Bad Bronzing

UVB

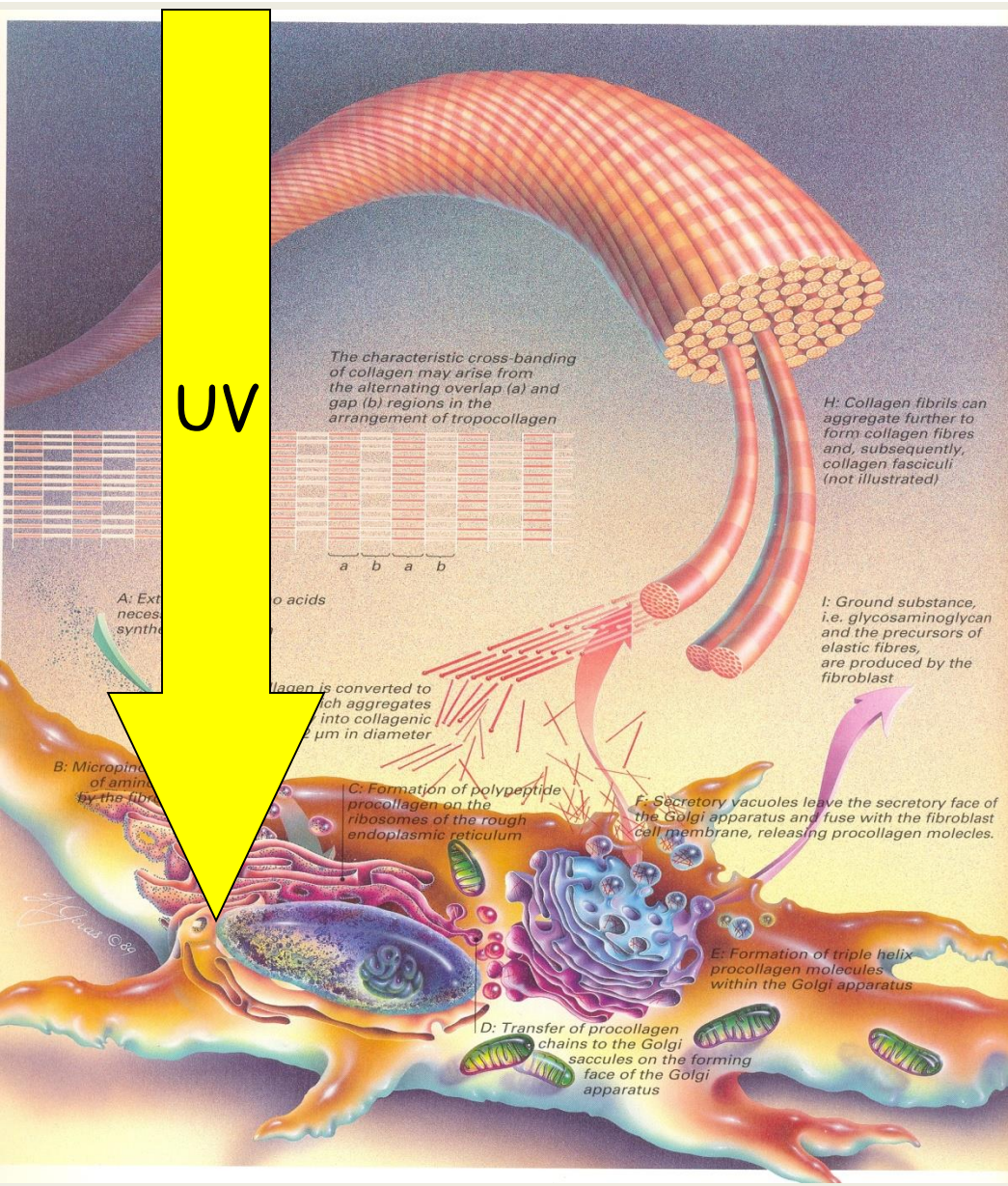


UVB- lentigo, actinic keratosis, carcinoma, melanoma

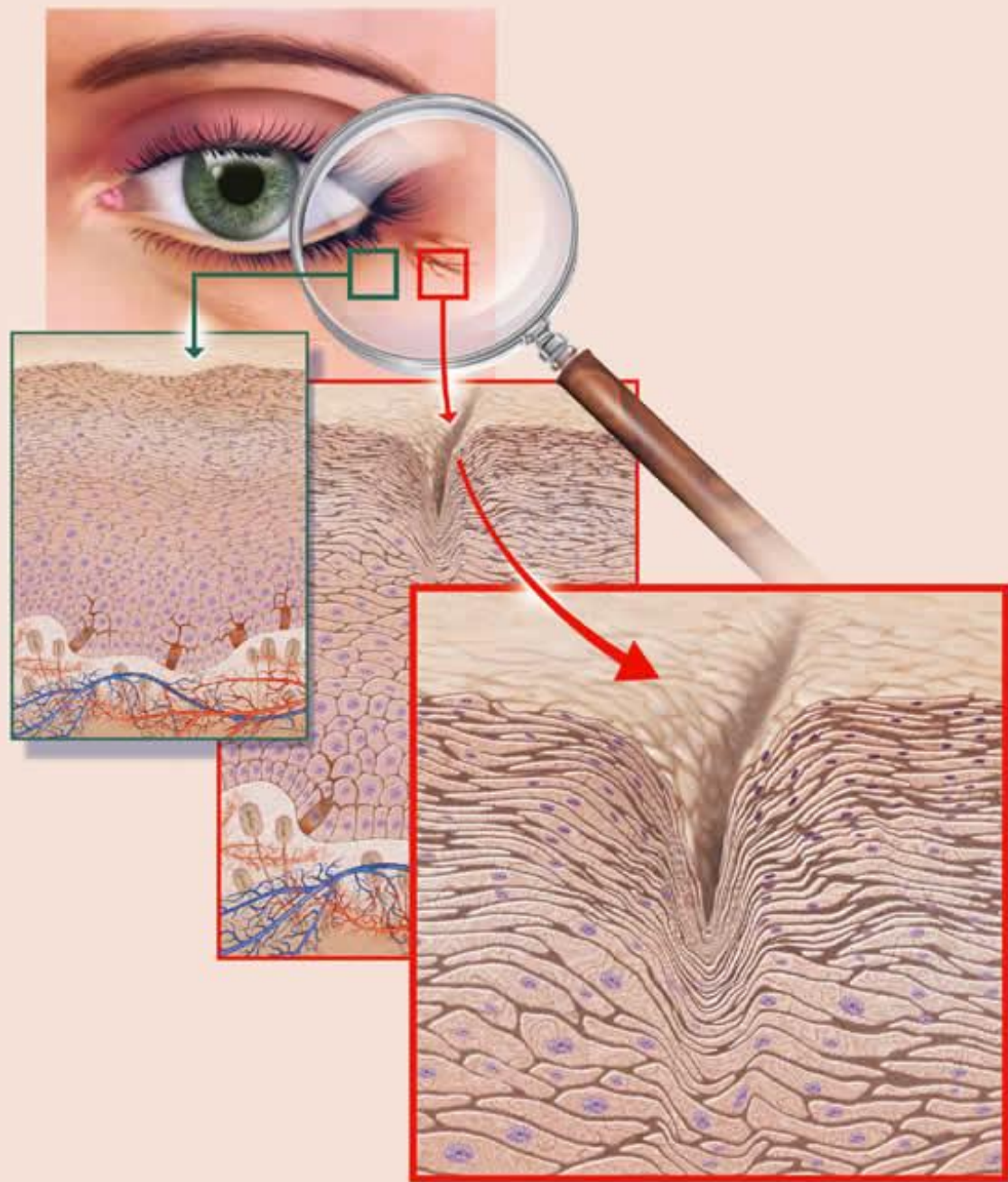


UVA

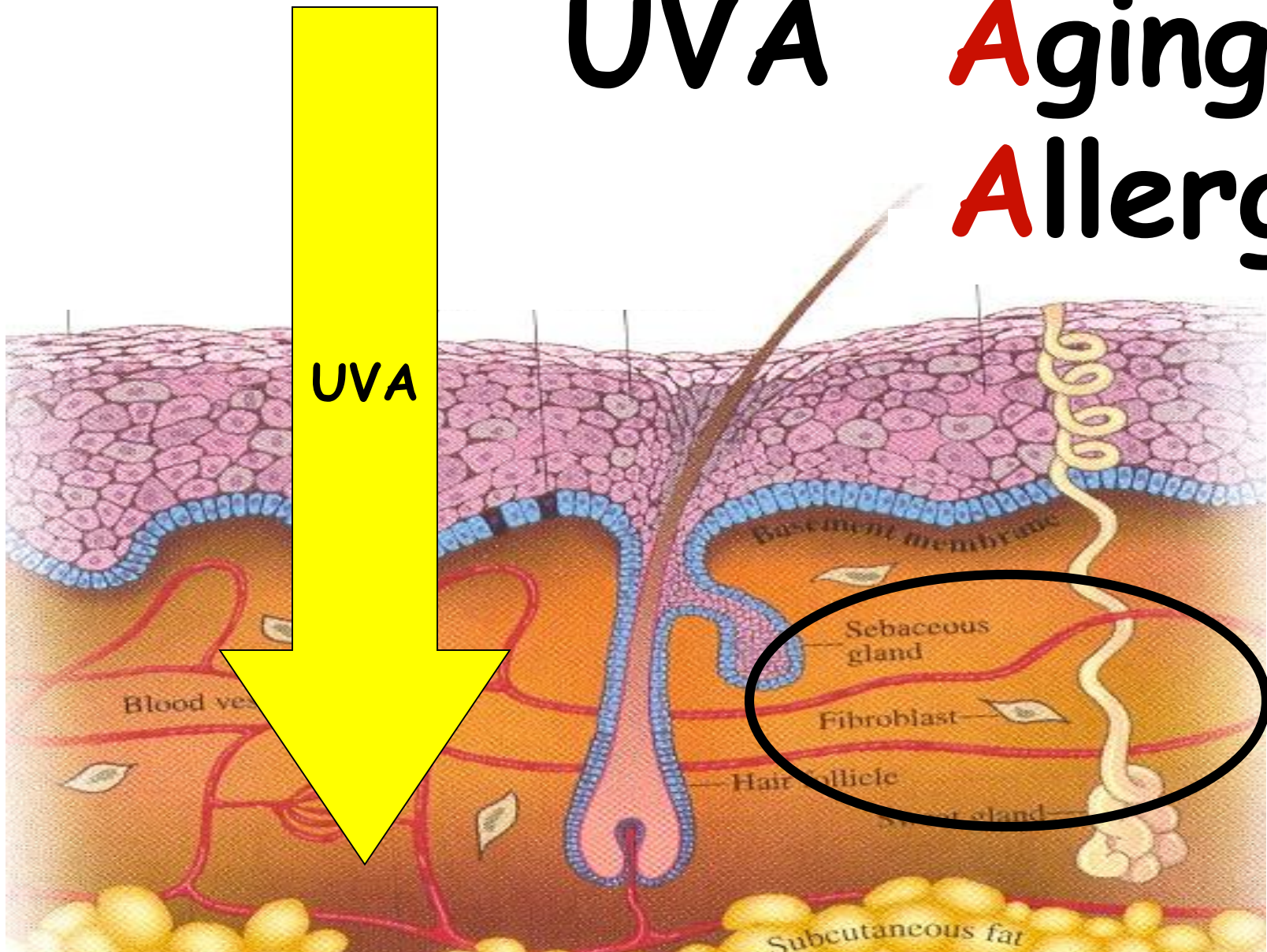
collagena and
elastic tissue
ground substance
vessels



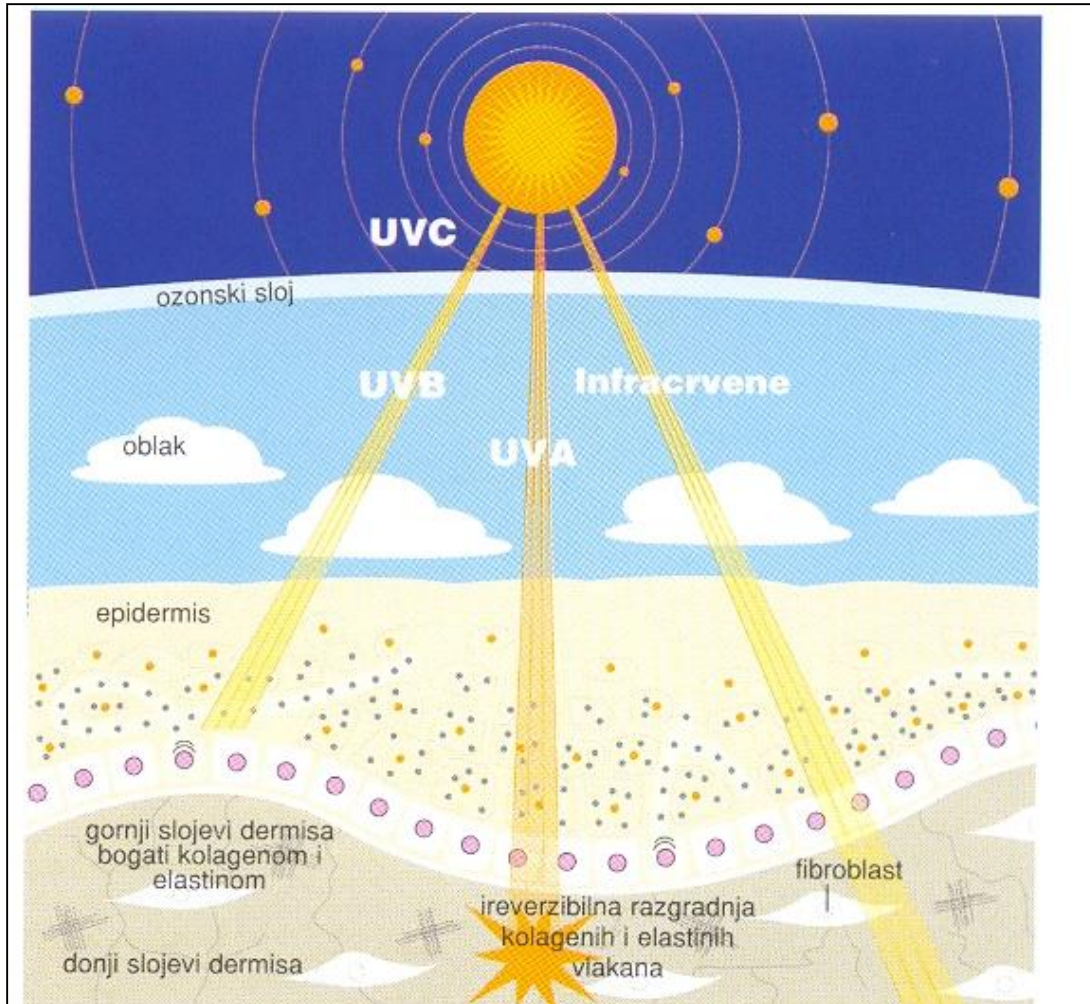
collagen synthesis and
destruction after
UV exposure matrix
metalloproteinases(MMPs)



UVA Aging Allergy



UVA - photoaging, photosensibilisation, skin cancer, melanoma

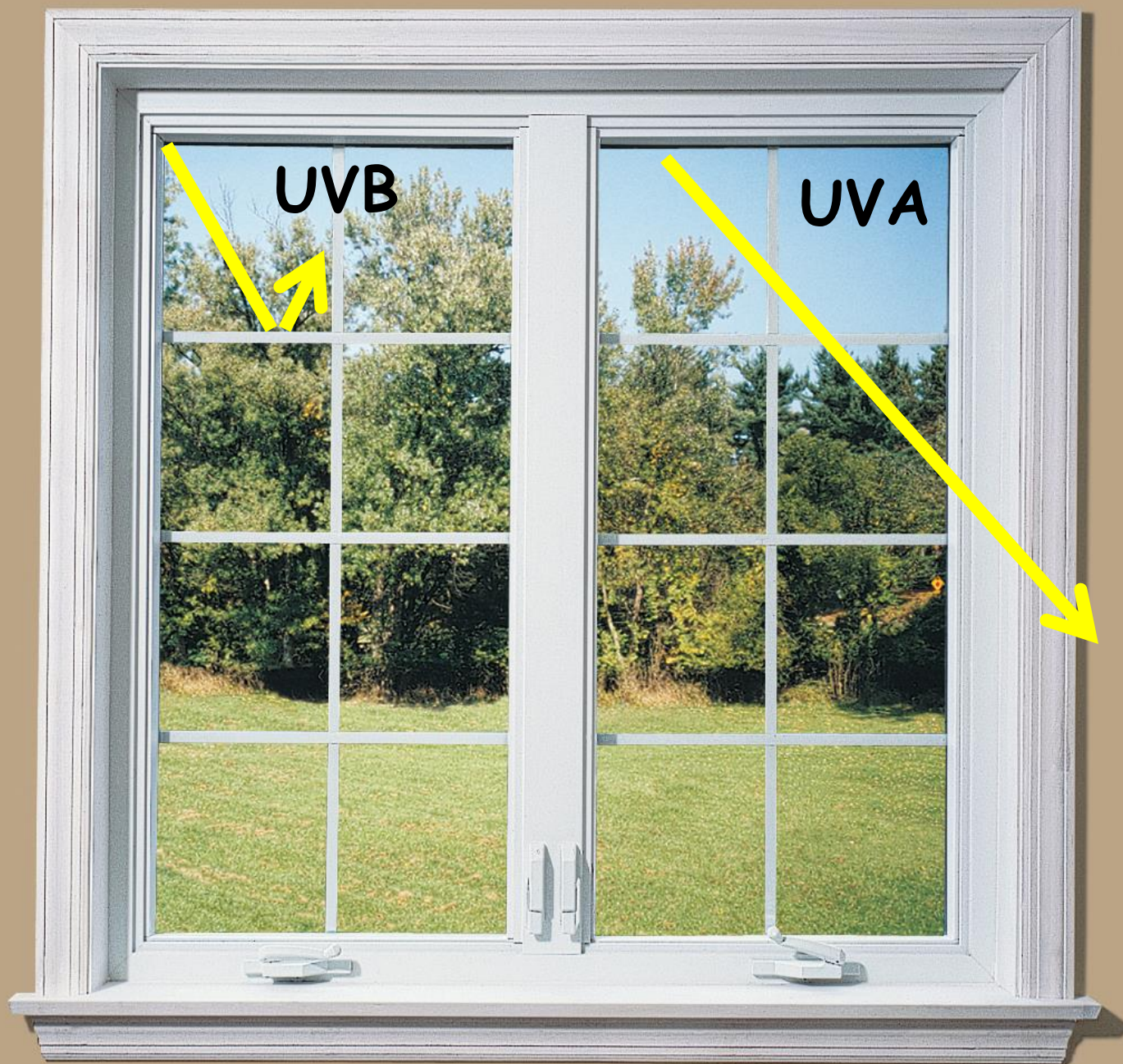


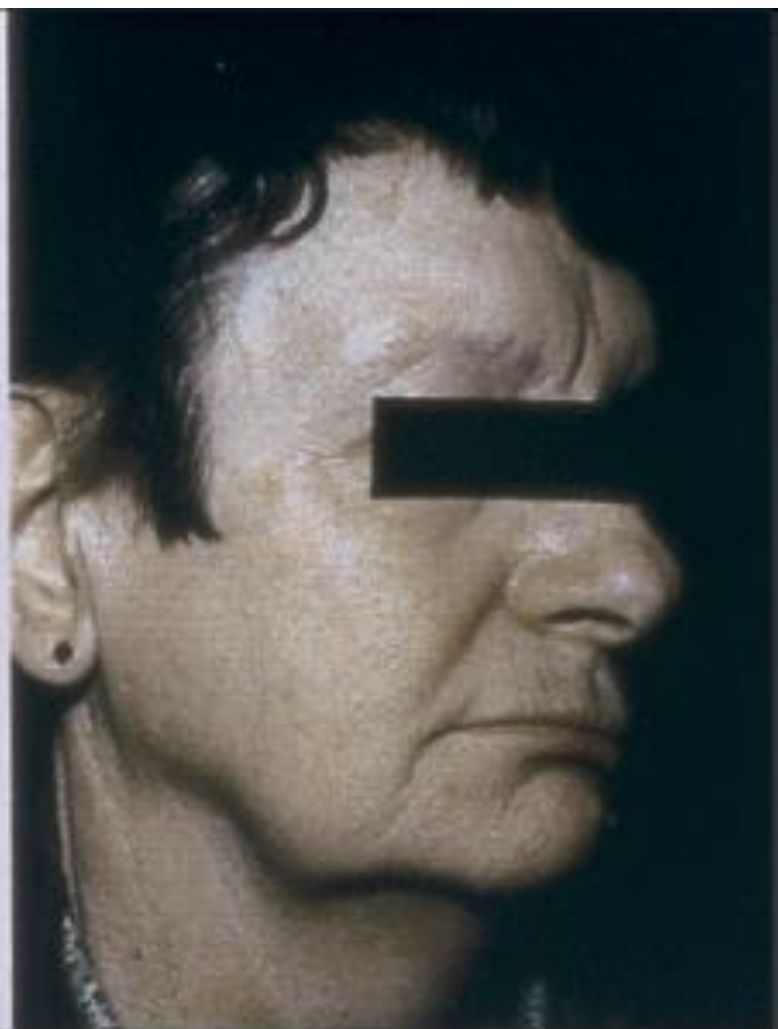
UVA

- passes through the clouds
- passes through the glass
- passes through clothing
- bleaches the colour
- constant throughout the year

UVB

- passes through the clouds
- don't pass through the glass
- the strongest from III-IXm

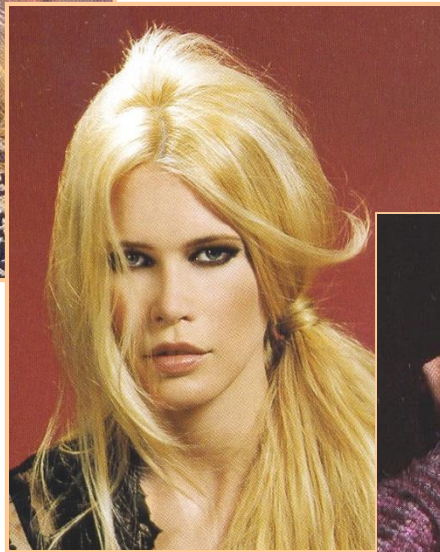




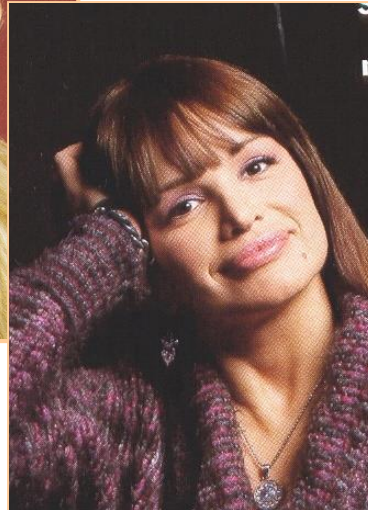
Skin types according to Fitzpatrick:



I type
(Celtic origin)



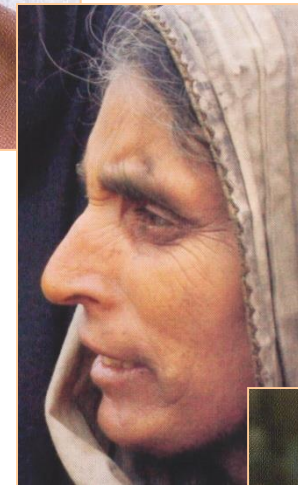
II type



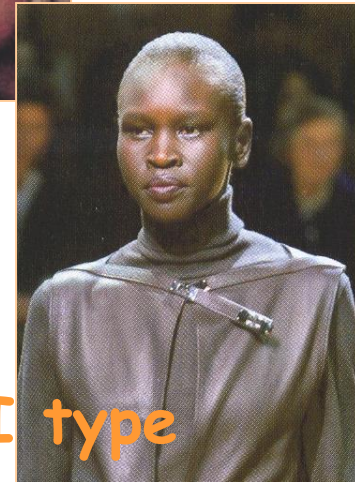
III type



IV type

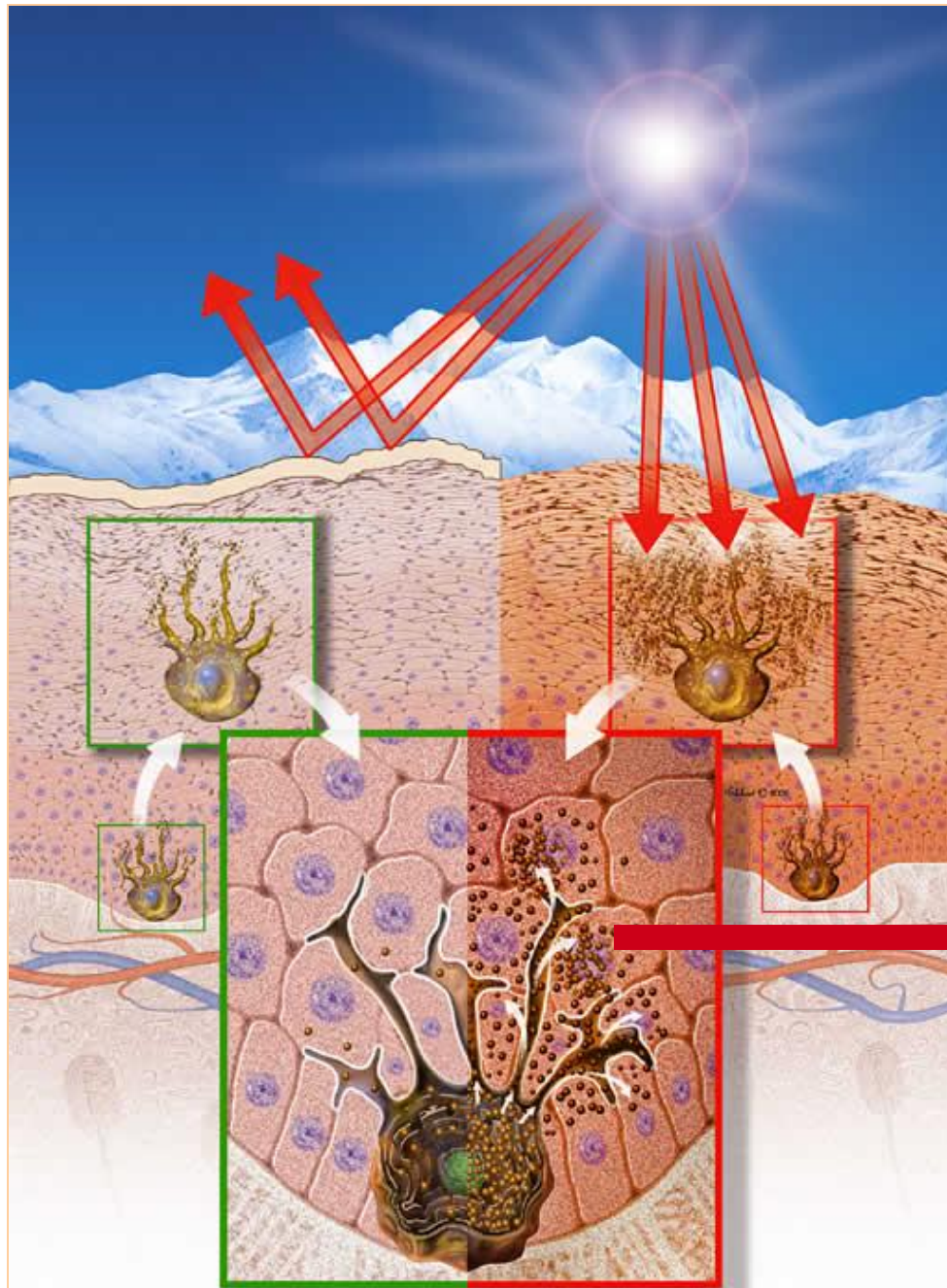


V type

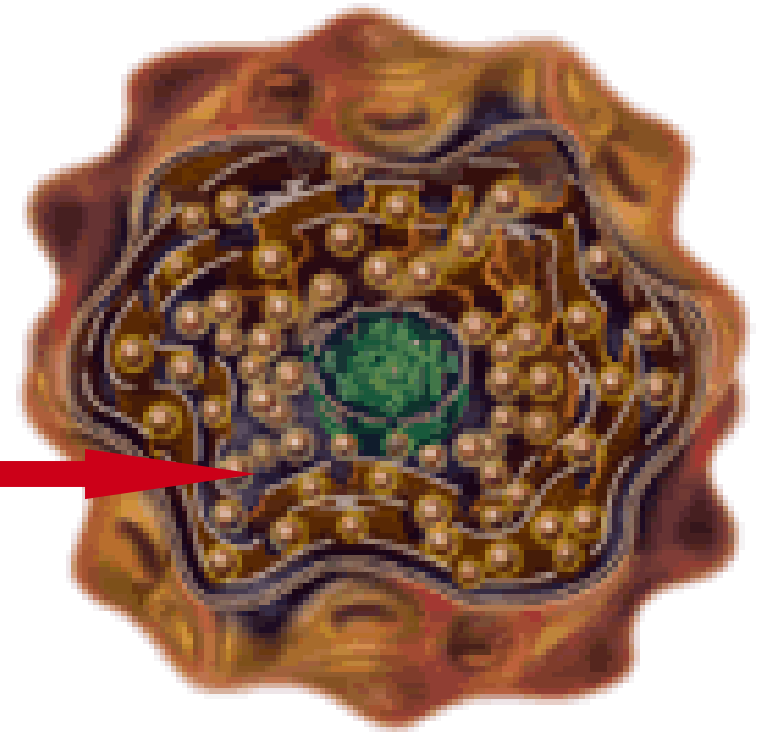


VI type

80%UV up to 18. years



every 10 years 8-20%
less



melanocytes



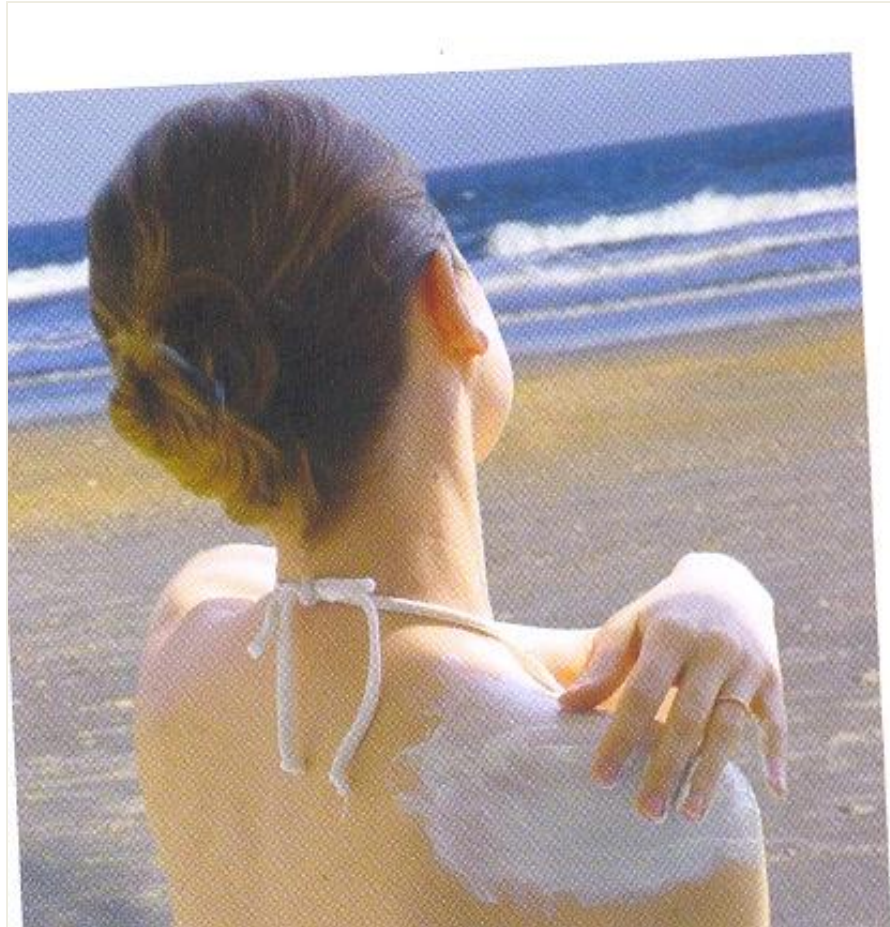
SPF 4

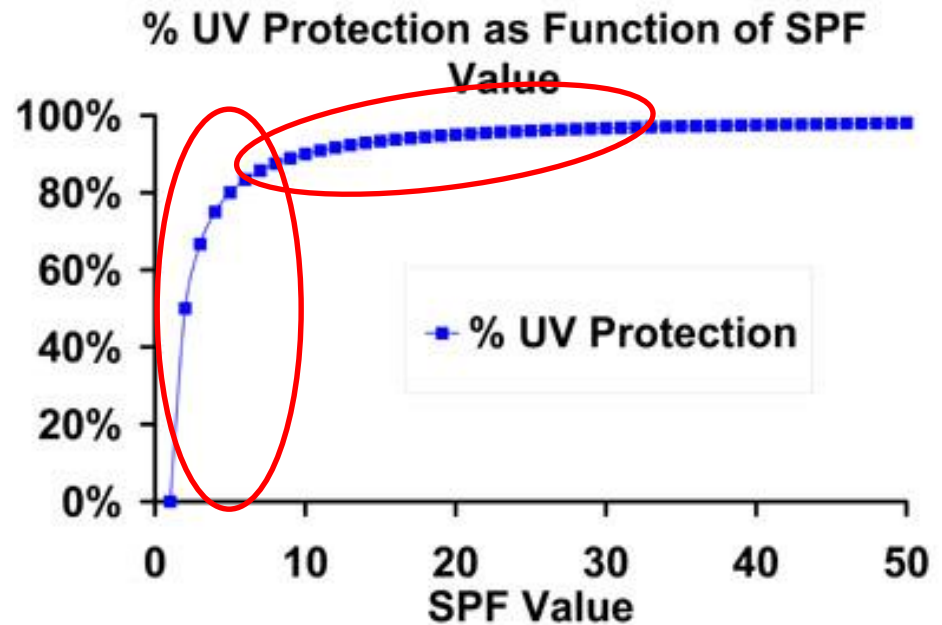
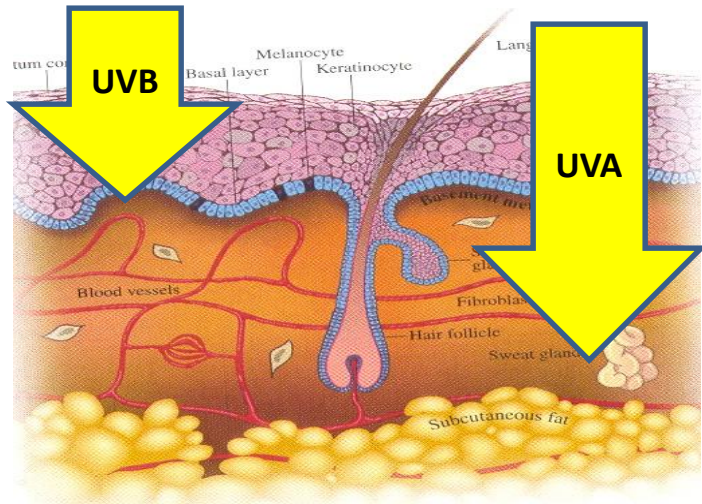
water resistant after 40 min in water

very water resistant /water proof/ after 80 min in water

SPFx20 min

safe period without
burning





SPF

PPD (persistent pigment darkening)

not linear scale

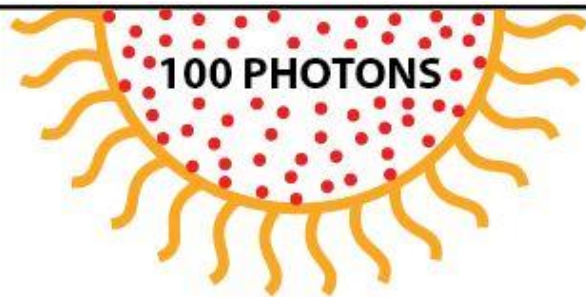
measure for UVA

PPD 10 around 90% protection

PPD 15 and > very good protection

$$\text{PPD} = 1/3 \text{ SPF}$$

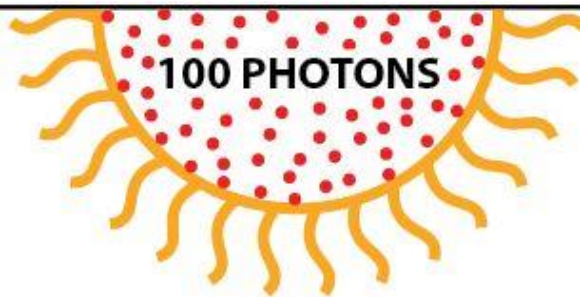
SPF 2	50%
SPF 4	75%
SPF 8	87%
SPF 16	93,6%
SPF 32	96,9%
SPF 64	98,4%



NO SUNSCREEN



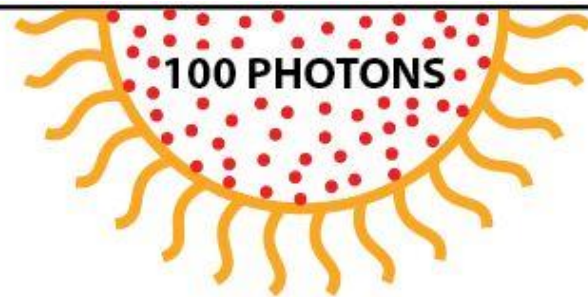
**100 PHOTONS
ENTER THE SKIN**



**SPF15 SUNSCREEN
(93% protection)**



**7 PHOTONS
ENTER THE SKIN**

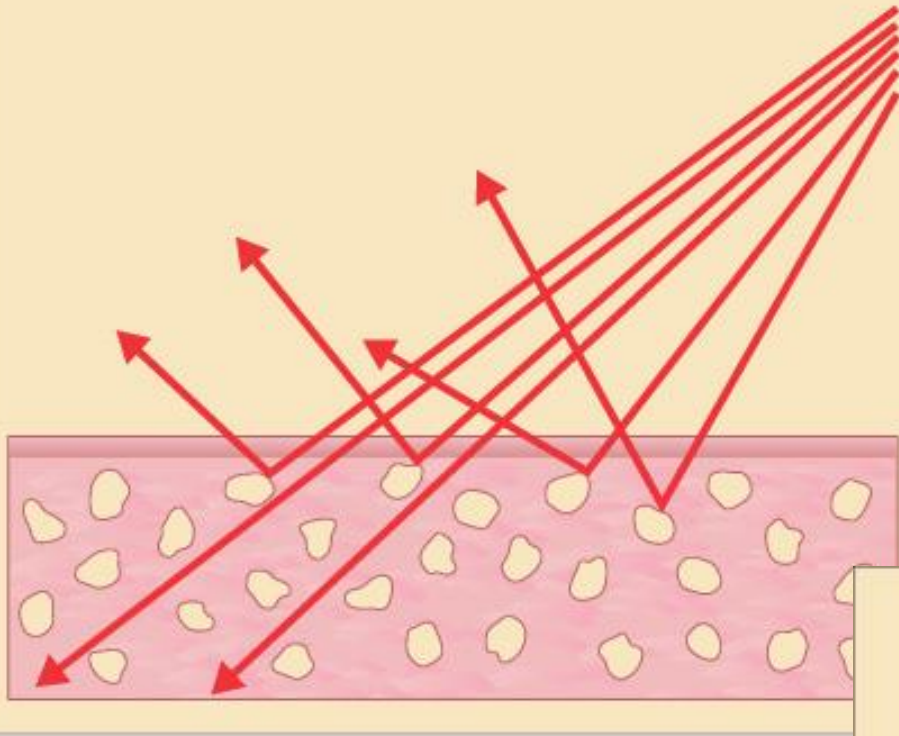


**SPF30 SUNSCREEN
(97% protection)**

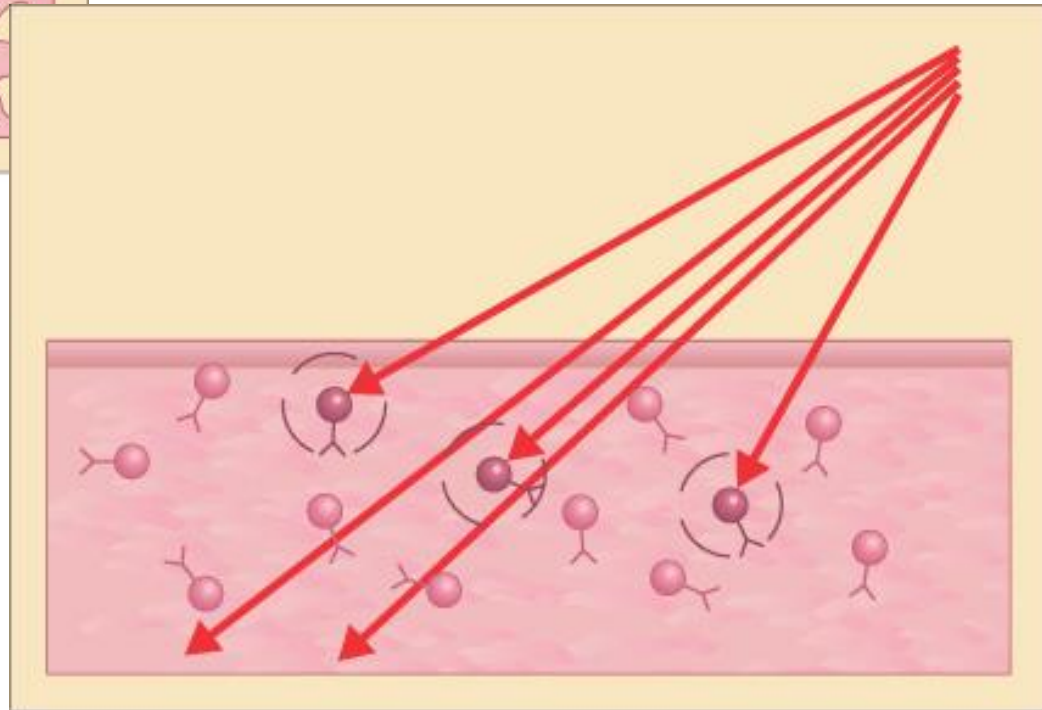


**3 PHOTONS
ENTER THE SKIN**

reflexion



absorption



Be careful of reflexion:

sand

snow

water surface



Tanning booths (entirely UVA)



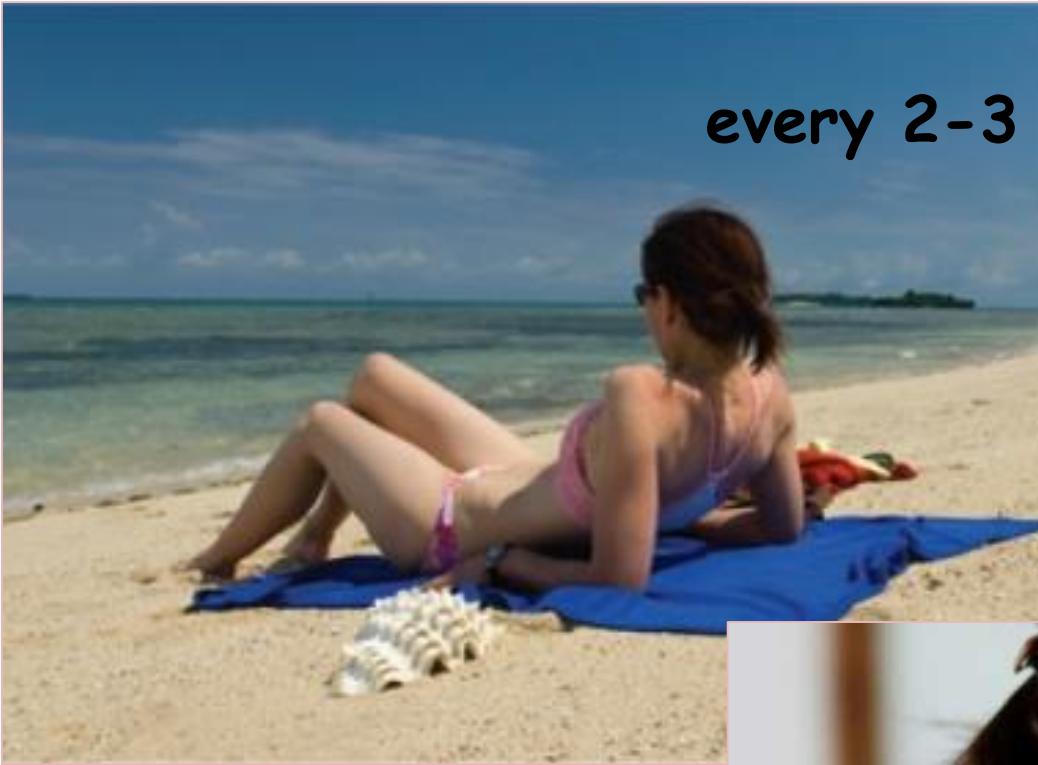
5x more emitted energy than during summer at noon



spreading of creams

20-30' before UV exposure

every 2-3 hours repeat procedure



85% wipe off
with a towel







50% UVA

shadow diminishes
UV for 50 - 95%





**UV protection
clothing**

density weaving
type of material

polyester
wool
silk
nylon

cotton
rayon

**UPF for clothing
(UV protection factor)**



UPF 257

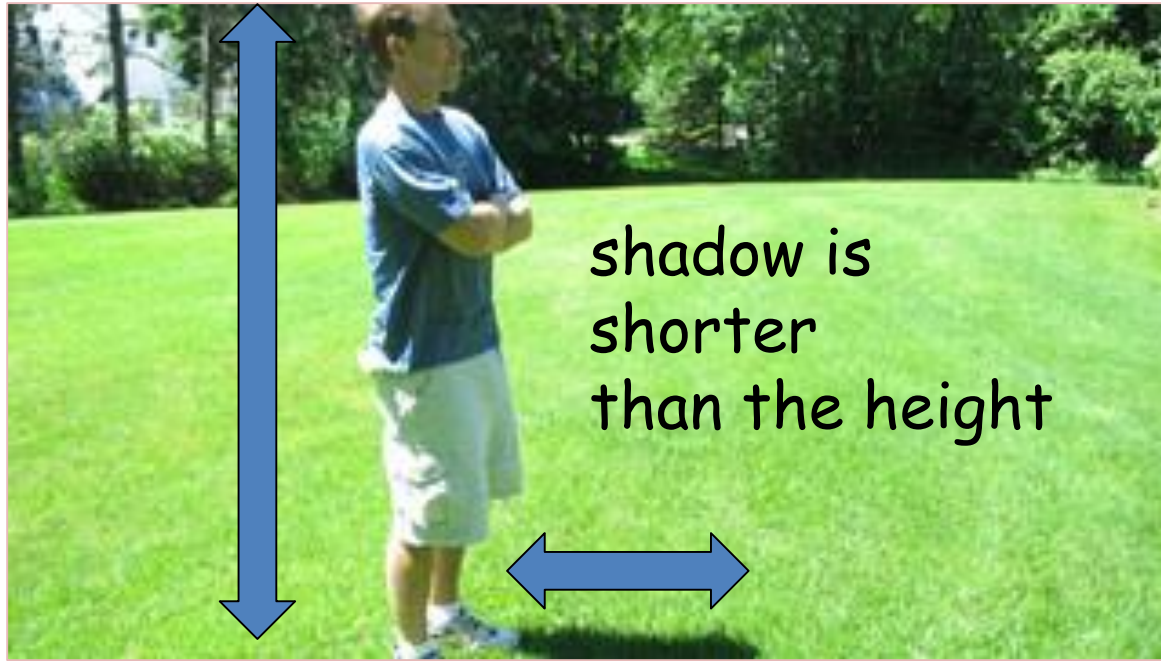


UPF 22



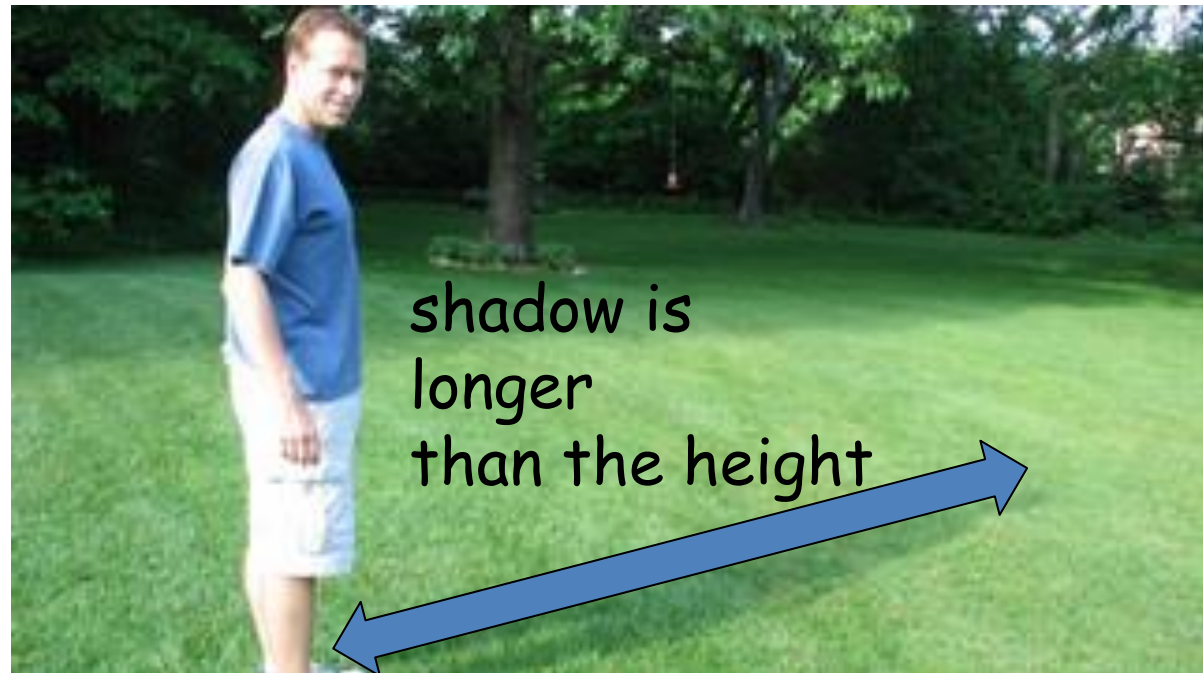
sand colour

UPF <6



shadow is
shorter
than the height

**10 am - 4 pm
the strongest UVB**



shadow is
longer
than the height



wide brimmed ho

UPF 5 - 17



Wide brimmed hat
(7.5 cm or greater)

Sunglasses

Long sleeves
and pants



Factors increasing sun protection of clothing

Increased tightness
of fabric weave

Wearing dry clothing

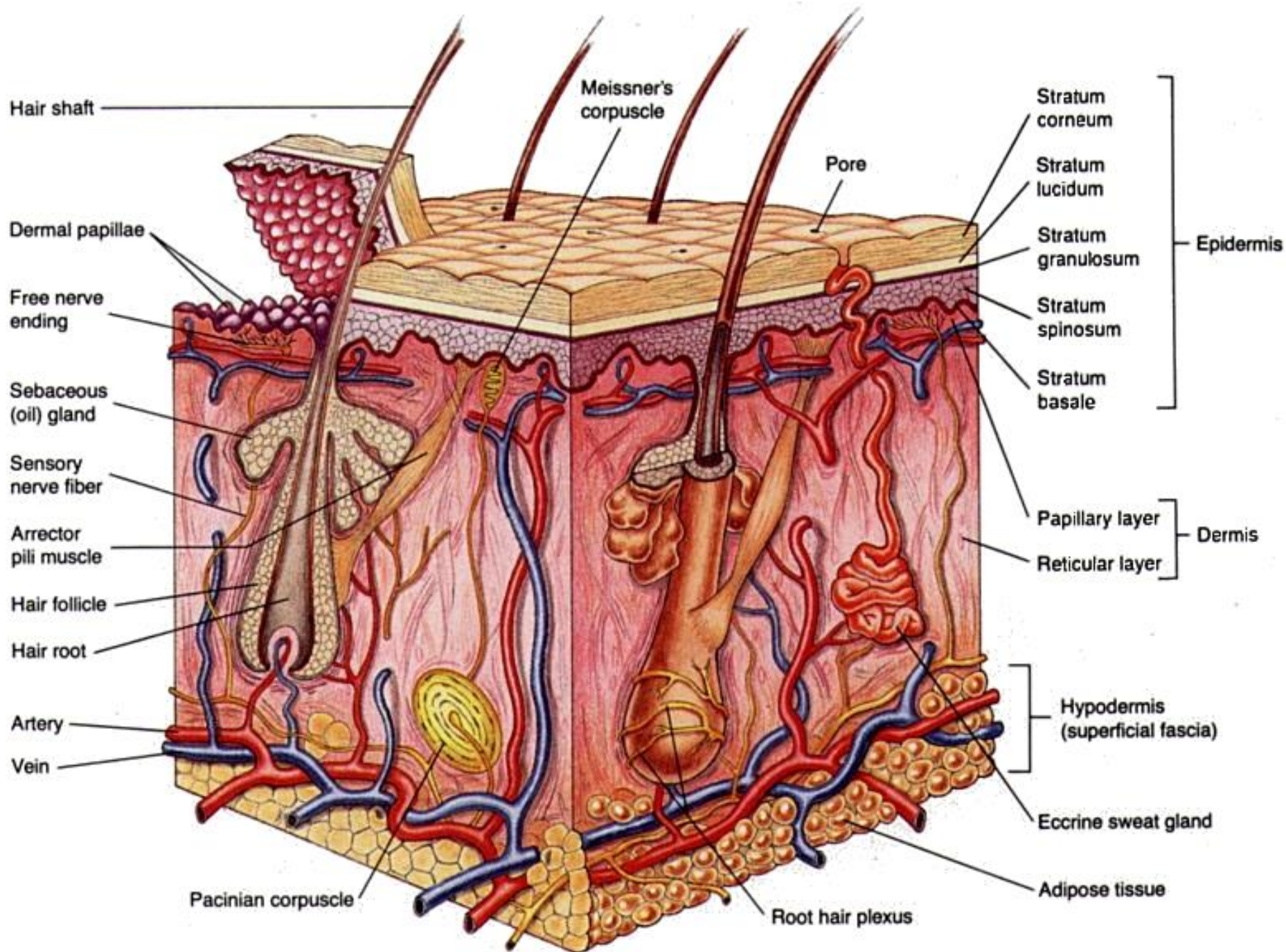
Type of fabric (polyester >
nylon, silk, wool > cotton, rayon)

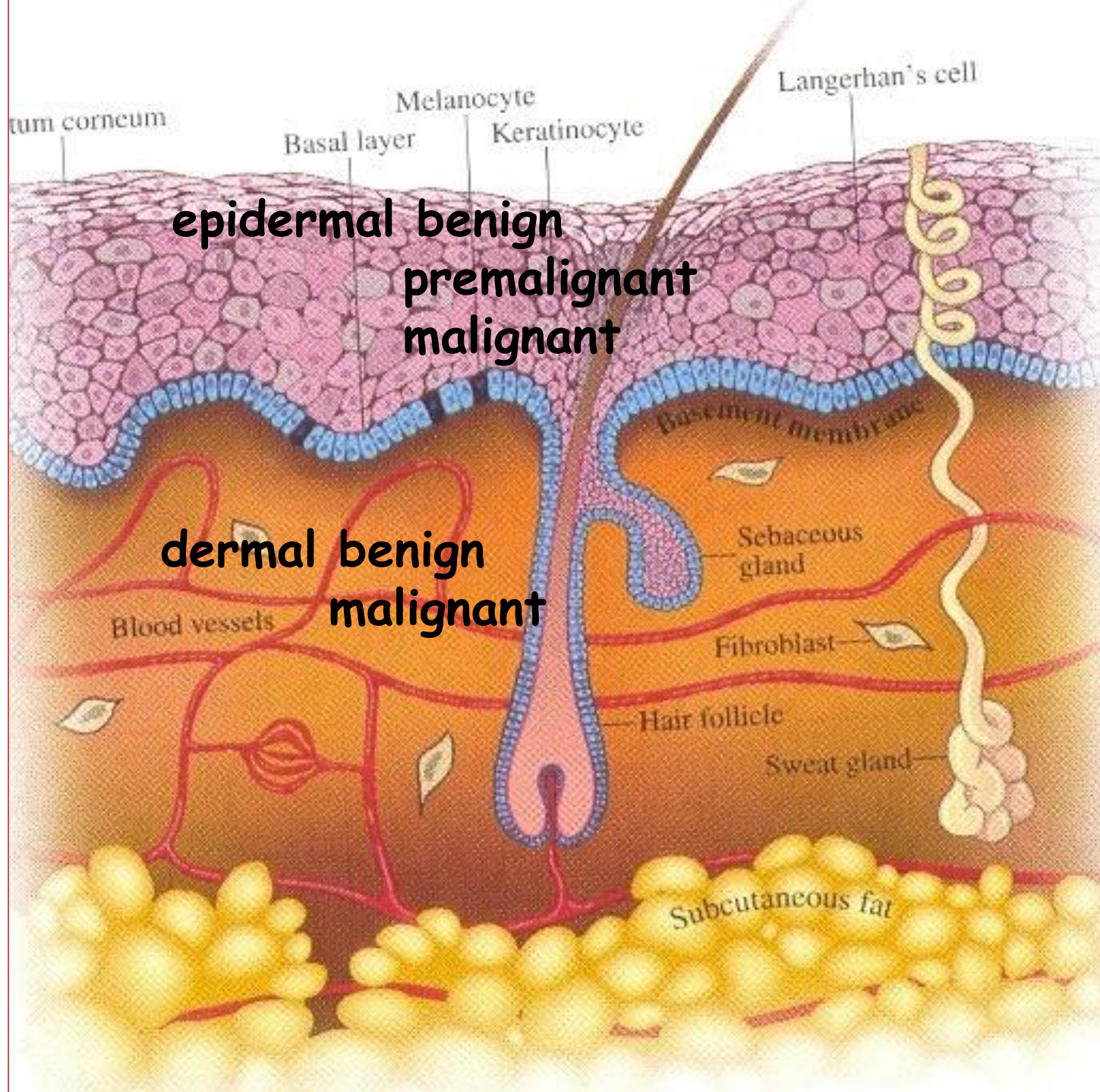
Loose fitting items

Pre-washing

Chemical additives (e.g.
optical whitening agents)







Epidermal benign tumours



- Skin tags

- Seborrhoeic keratosis

- Cutaneous horn

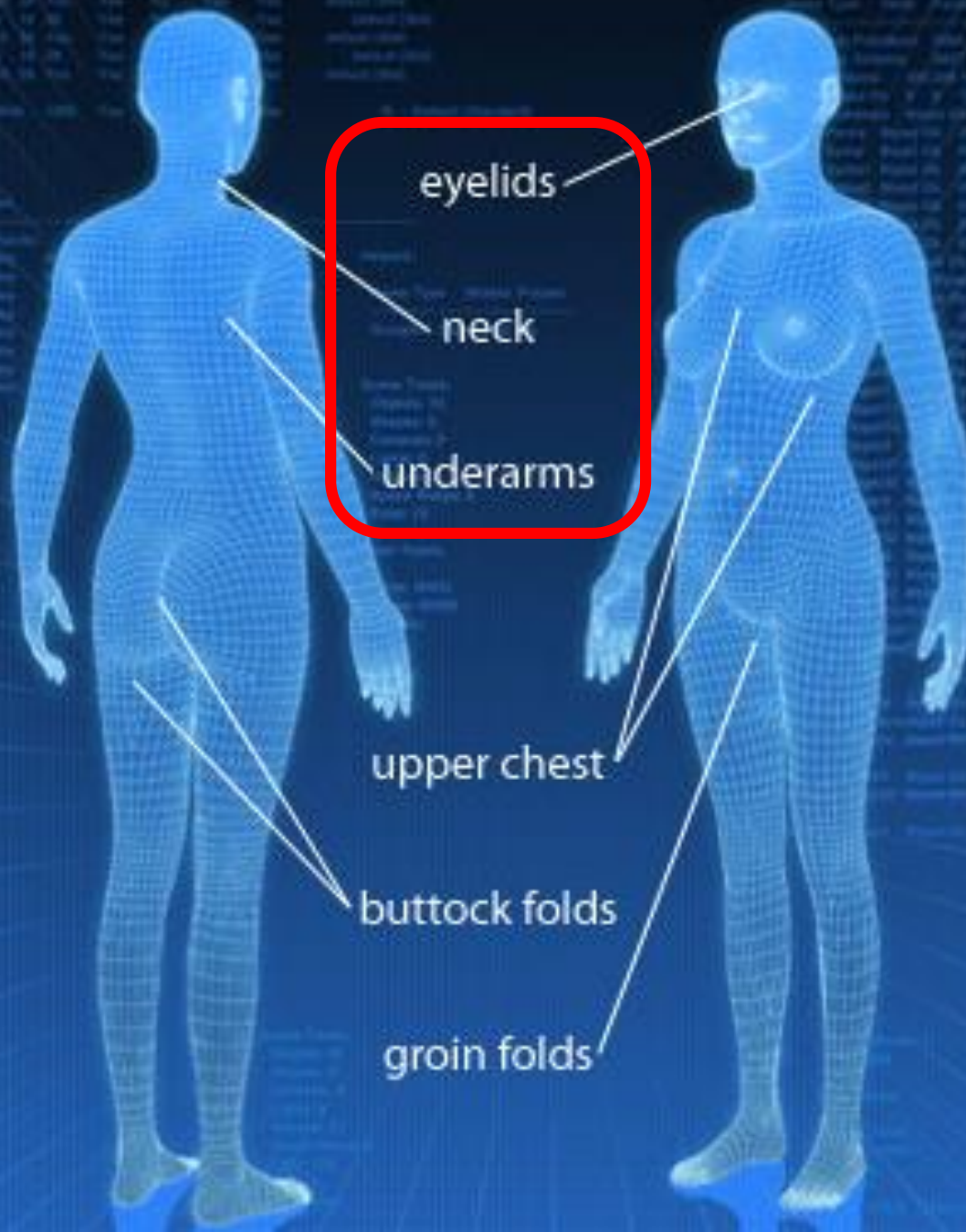
Skin tags:



common benign
outgrowths of skin

most common in middle-aged and elderly

Most Common Areas for Skin Tags



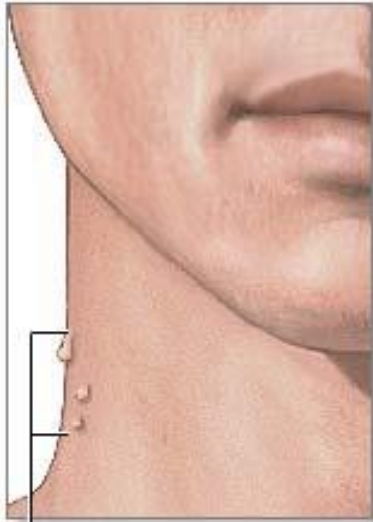


most common in obese women
sometimes familial trait

look unsightly
may catch on clothing and jewellery

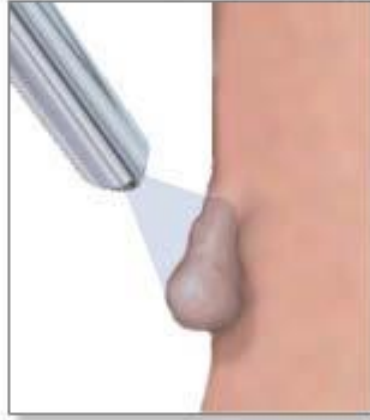


skin tags



Skin tags

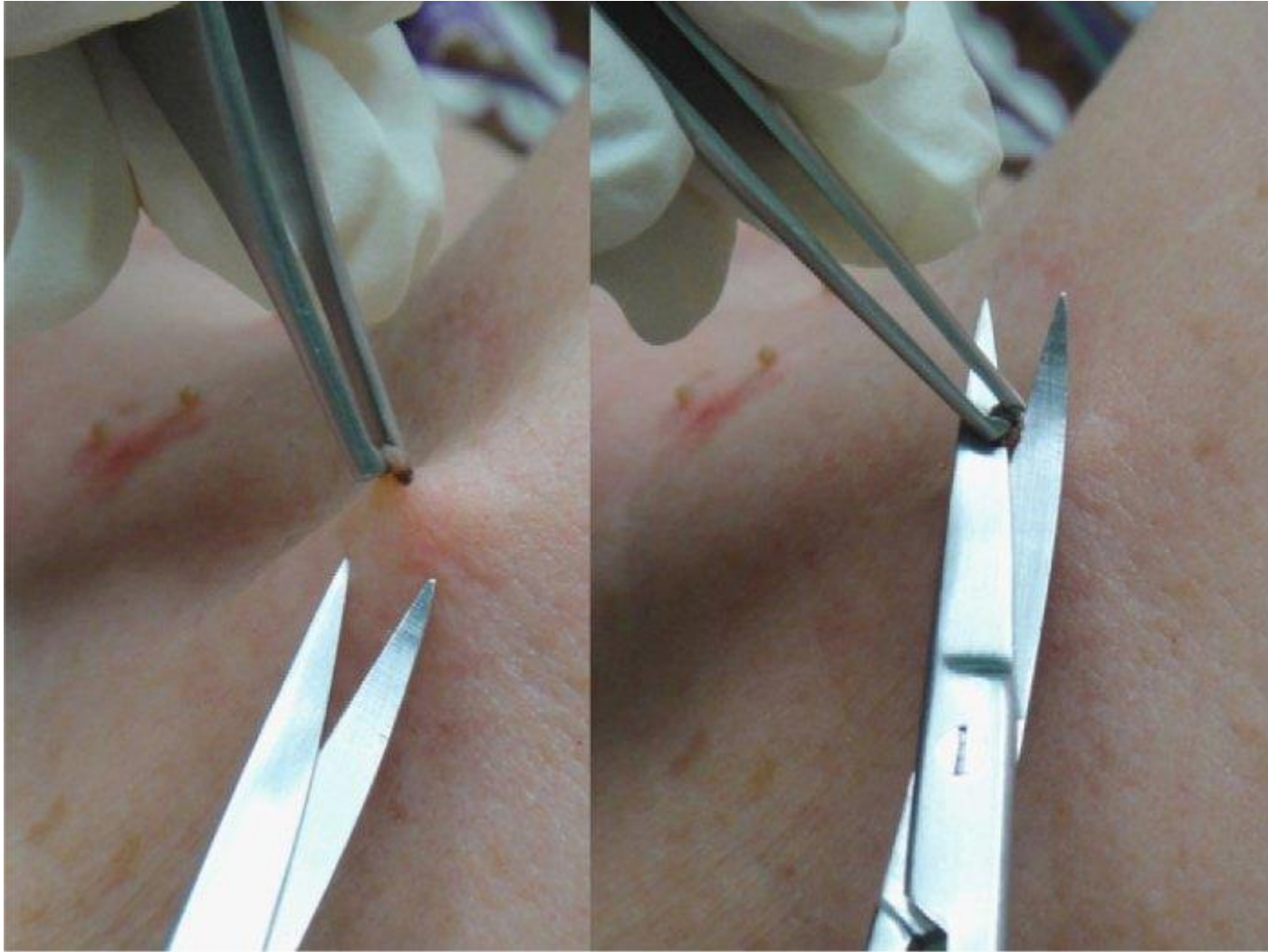
Super-freezing of skin tag tissue (cryotherapy)



Treatment:

snipped off with scissors
liquid nitrogen
electrocoagulation





snipped of skin tags with scissors

Epidermal benign tumours

- Skin tags

- Seborrhoeic keratosis

- Cutaneous horn

Seborrhoeic keratosis



common benign
epidermal tumour
unrelated to
sebaceous gland

usually arise after the age
of 50 years



unknown causative agent
multiple - may be inherited



"stuck-on"
appearance

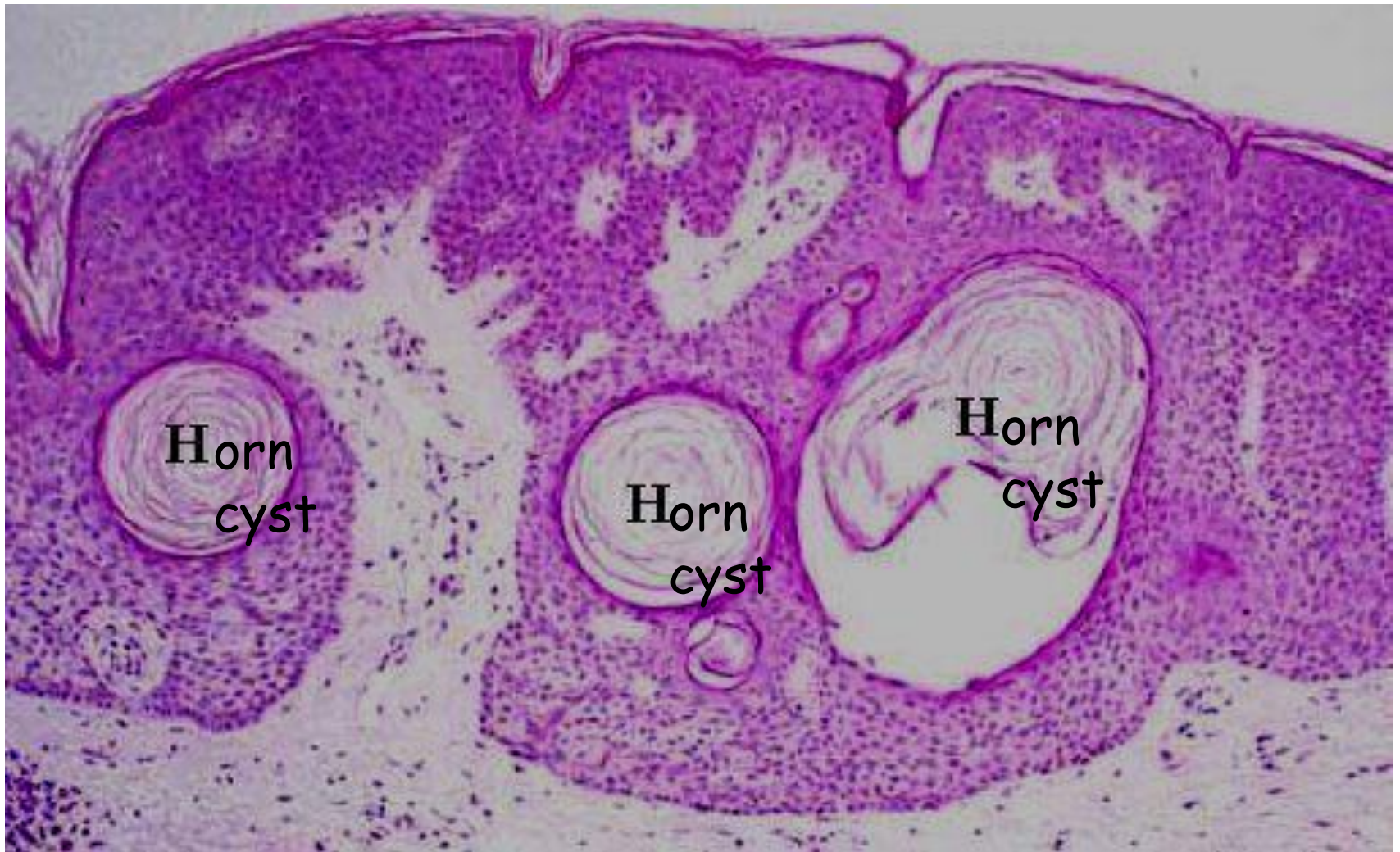
surface:
smooth or verrucous
greasy scaling
scattered keratin plugs

seborrheic keratosis (seborrheic wart)

numerous small dry white/grey stuck-on lesions



stucco-keratosis around the ankles after age of 50 years



histology of a seborrheic keratosis

Leser-Trelat sign



the sudden eruption of hundreds of itchy lesions associated with internal malignancy

Treatment:

liquid nitrogen

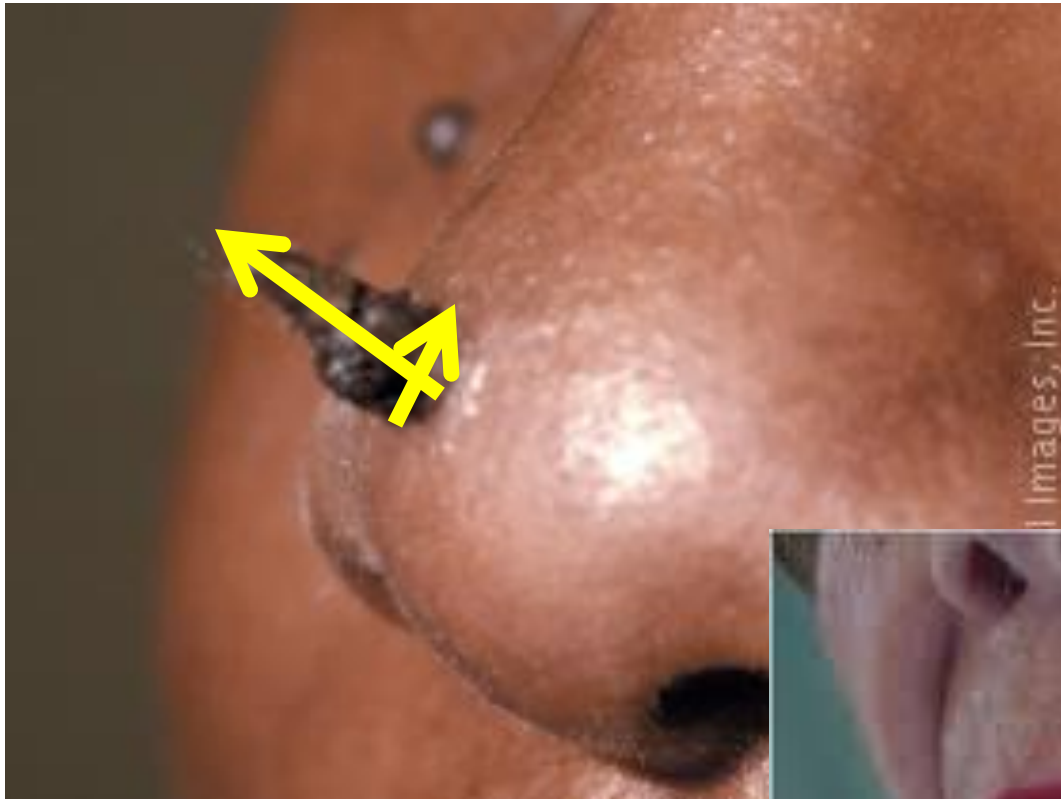
removing with curette

electrocoagulation

snipping of

Epidermal benign tumours

- Skin tags
- Seborrhoeic keratosis
- Cutaneous horn



may resemble a viral wart
keratin projection that is
taller than it is broad

cutaneous horn

**The histology should be
checked**



Naevi

refers to a skin lesion that has a localized excess of one or more types of cell in a normal cell site -
cutaneous hamartoma



linear epidermal naevus of temporal area



example of cutaneous mosaicism
follow Blaschko's lines

linear epidermal naevus



The current hypothesis is that a patch of tissue that follows a Blaschko line represents a clone of cells derived from a single cell in the early embryo



Treatment:

liquid nitrogen
laser
excision

linear epidermal naevus - keratinocytes are genetically different from their normal appearing neighbours

Melanocytic naevi



localized **benign tumours** of melanocytes
the cause is unknown /genetic factors
sun exposure during childhood/



most naevi appear in early childhood
sharp increase during adolescence and after sunburns

Further crops may appear during:

pregnancy

oestrogen therapy

flare-ups of LE

after cytotoxic chemotherapy or immunosuppression

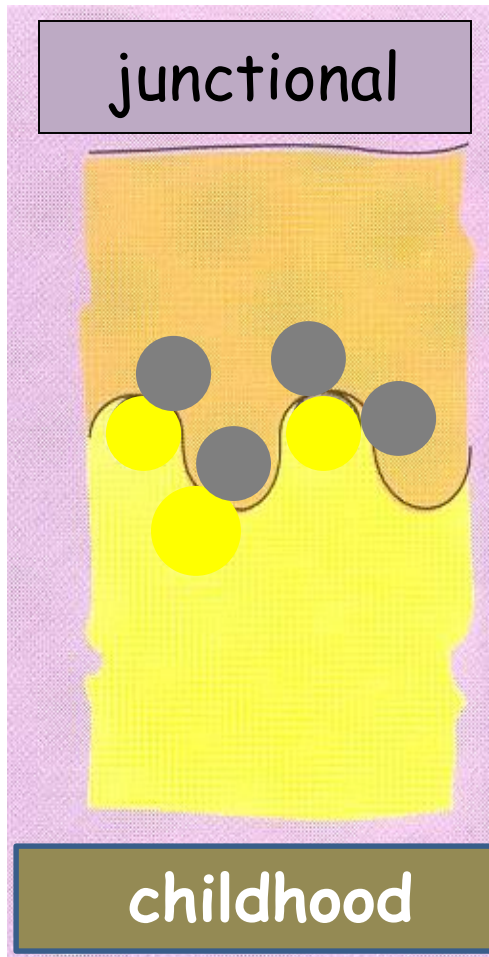
**New melanocytic naevi appear less often after the age
of 20 years**



melanocytes are sorted
in form of nests

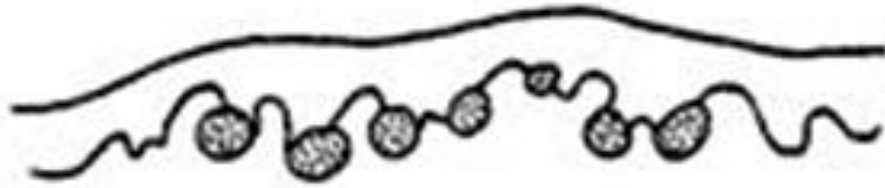


Maturation of naevi

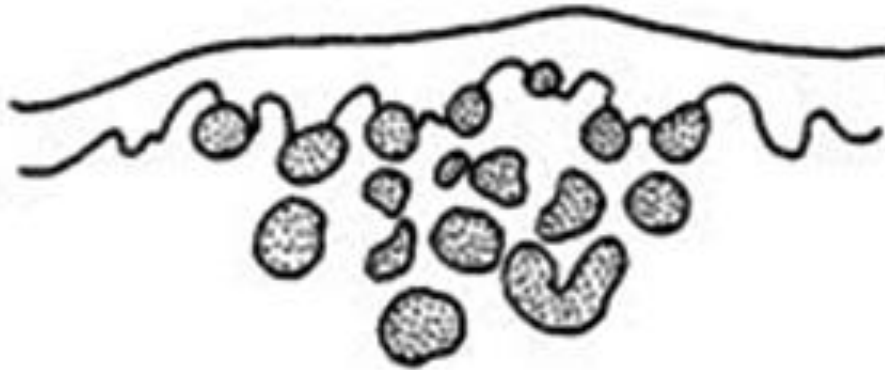


mole must follow or be accompanied by chronological age

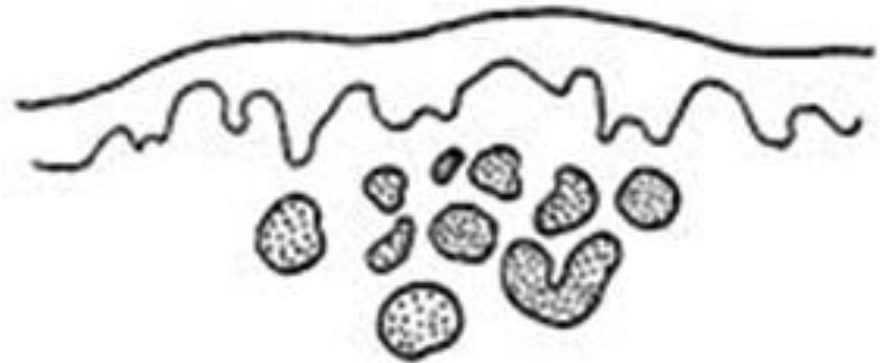
Junctional



Compound



Intradermal

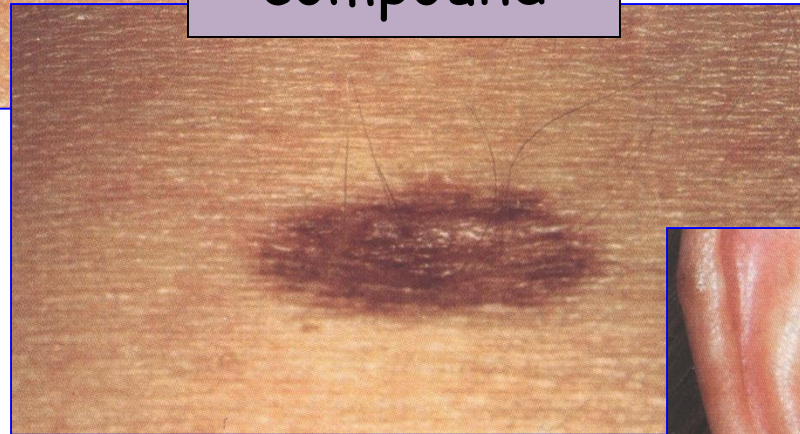


Maturation of naevi

junctional

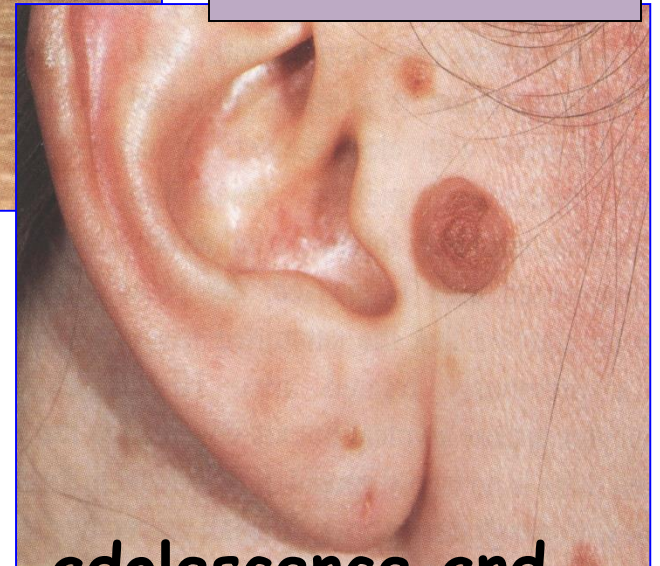


compound



childhood

intradermal



puberty

adolescence and
mature age

Melanocytic Nevus

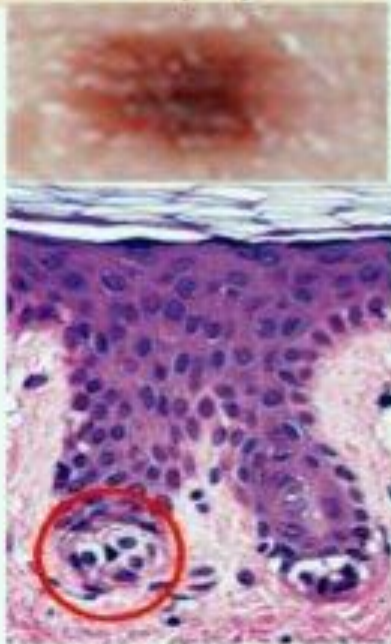
Junctional → Compound → Intradermal

Range in size 2-10 mm; most 3-7 mm in diameter.

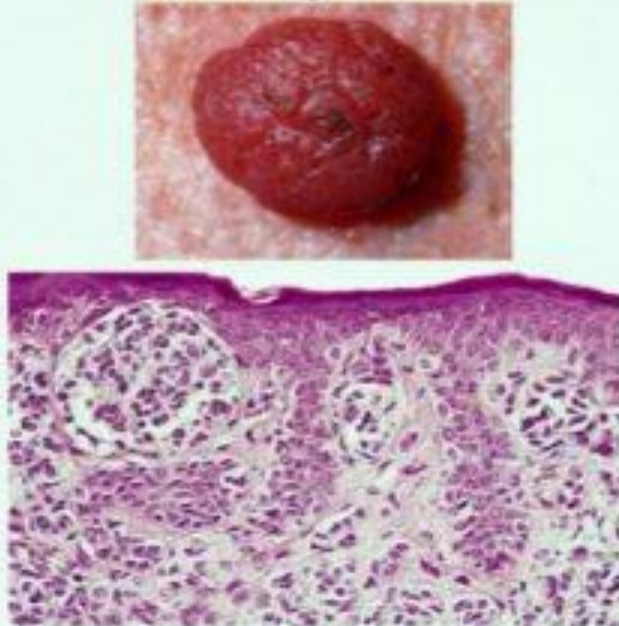
Remarkably round, evenly pigmented, soft on palpation.

Occur on sun-exposed areas - **vast majority on face & neck**

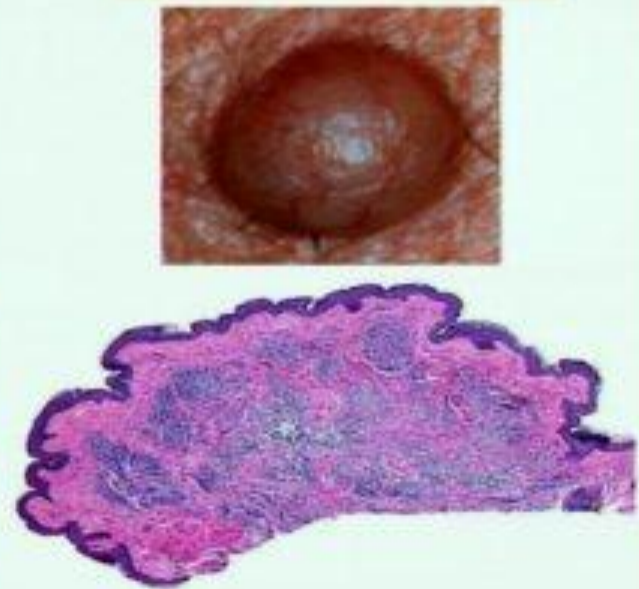
Junctional
Maculopapular



Compound
Papule

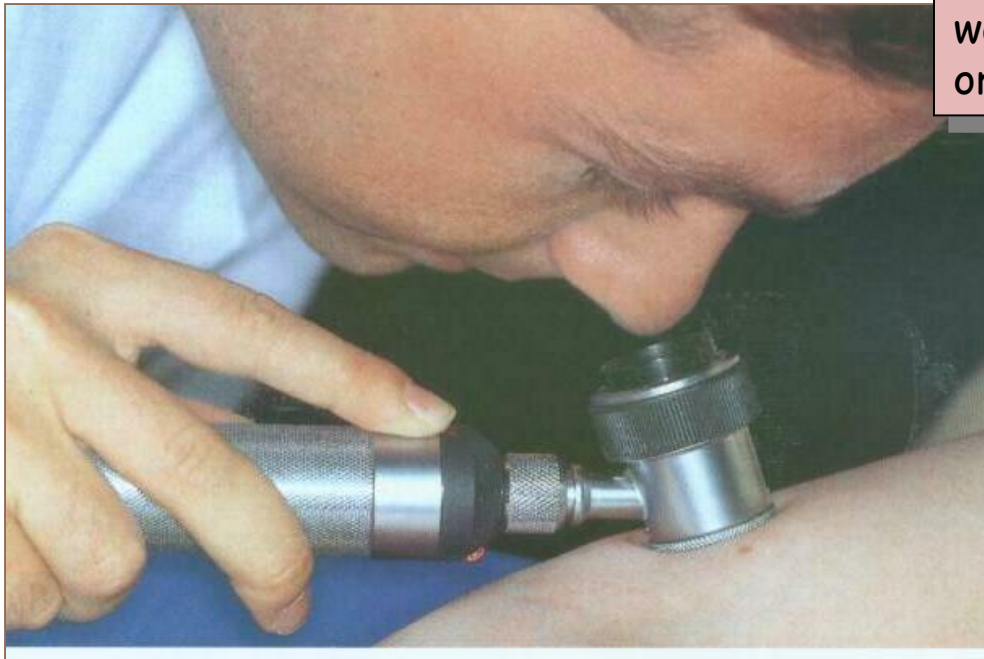


Intradermal
Nodule



Dermatoscopy:

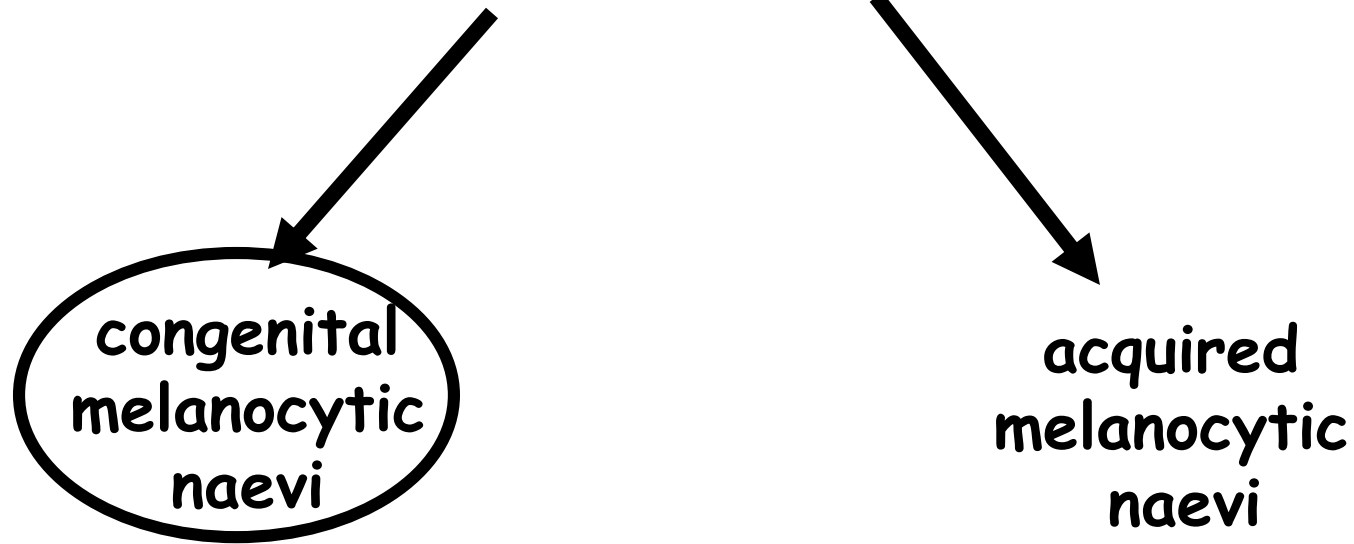
Serves for examination of pigmented
and vascular lesions



immersion with
water, paraffin oil
or alcohol



Naevi



Congenital naevi

controls 1-2x/year



small < 2 cm



medium-sized
/Zitelli's naevus/
2-20 cm



large >20 cm



congenital naevi

the risk of developing melanoma appears to be maximum in the childhood and adolescence



Naevi



```
graph TD; Naevi --> congenital[congenital melanocytic naevi]; Naevi --> acquired([acquired melanocytic naevi]); acquired --> junctional[junctional naevus]; acquired --> compound[compound naevus]; acquired --> intradermal[intradermal naevus]; acquired --> Spitz[Spitz naevus]; acquired --> blue[blue naevus]; acquired --> atypical[atypical naevus];
```

congenital
melanocytic
naevi

acquired
melanocytic
naevi

junctional naevus
compound naevus
intradermal naevus
Spitz naevus
blue naevus
atypical naevus

Junctional naevus



roughly circular macules
mid brown to black

palms, soles, genital region, mucous membranes

Compound naevus



domed pigmented nodules up to 1 cm
arise from junctional naevi as melanocytes "drop off"
light brown, smooth, hyperkeratotic papillomatous

Intradermal naevus



like compound but less pigmented and skin-coloured

Spitz naevus (juvenile melanoma)



most often in children
develop over months or two as pink or red nodule

Naevus Spitz:

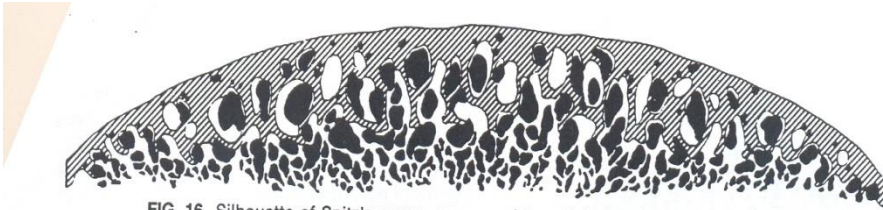
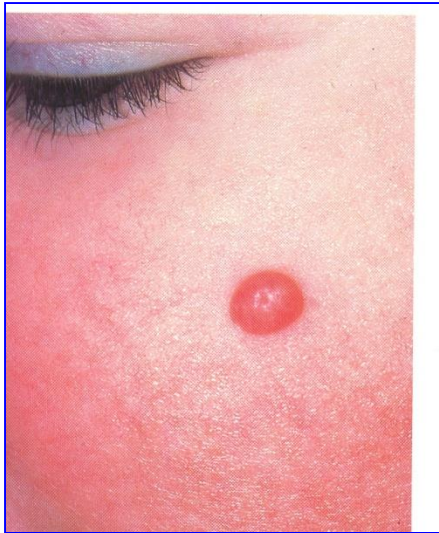
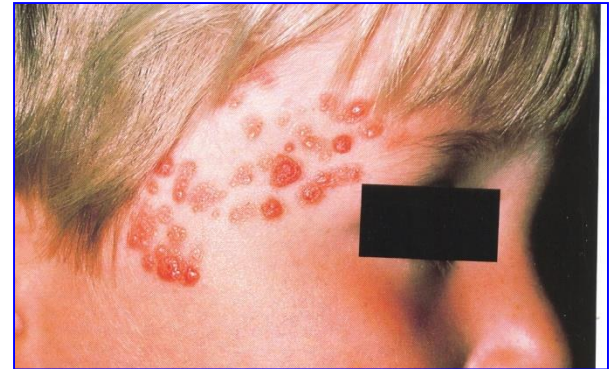


FIG. 16 Silhouette of Spitz's naevus, compared to that of a melanoma.



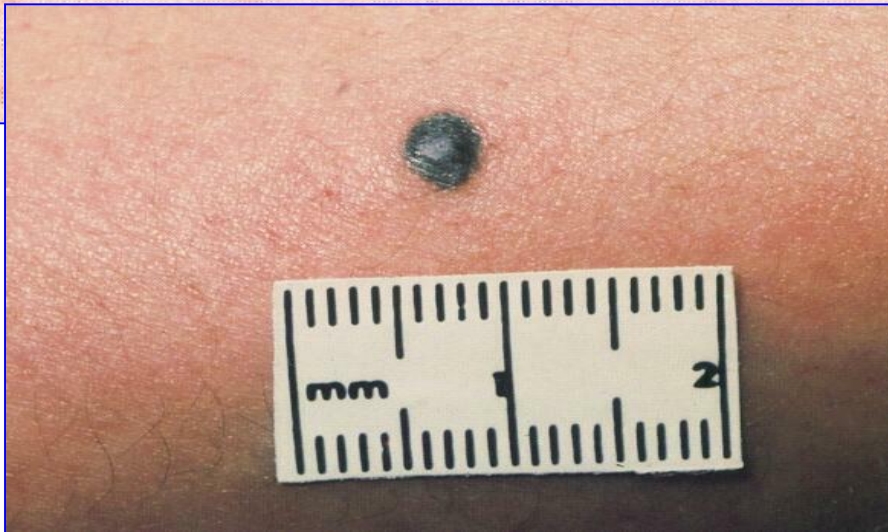
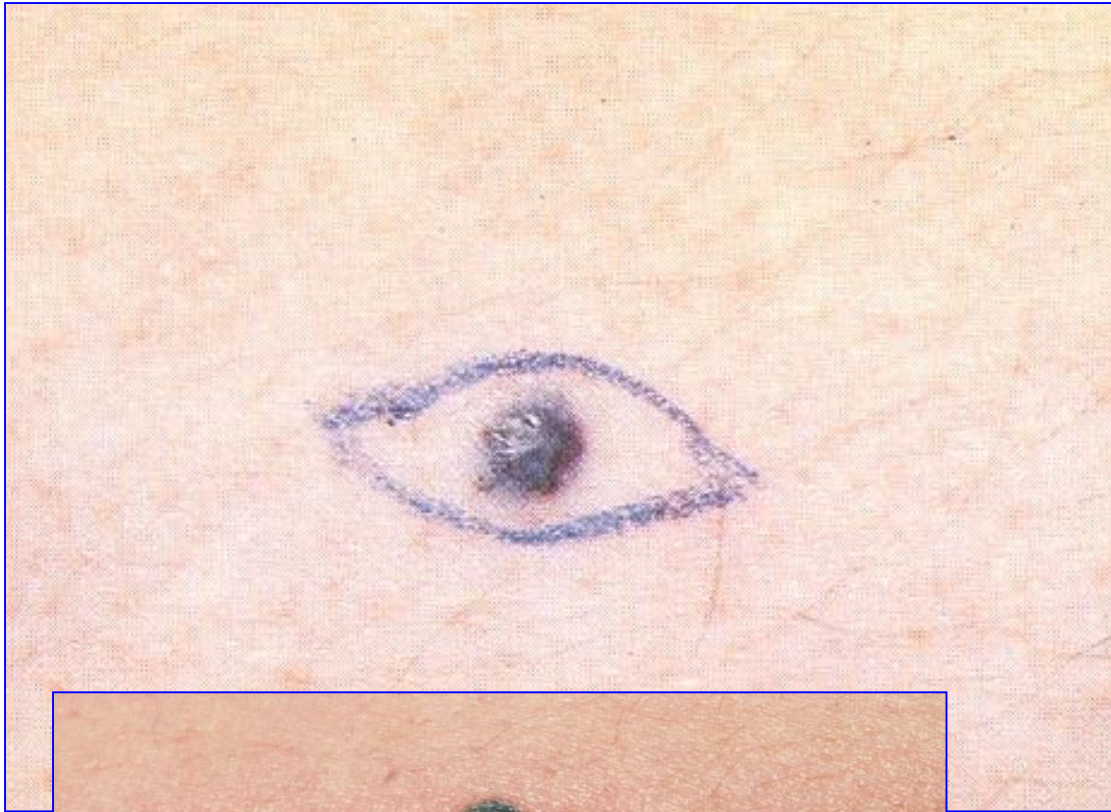
face and legs

excised due to rapid growth



no alteration toward melanoma

Blue naevi



slate grey-blue colour
limbs, lower back

excision should be recommended

Mongolian spots

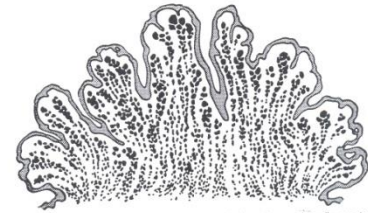


bruise-like greyish areas
lumbosacral region

fade during childhood

Ackerman's division according to silhouette:

1. Unna's naevus



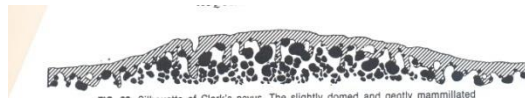
2. Miescher's naevus



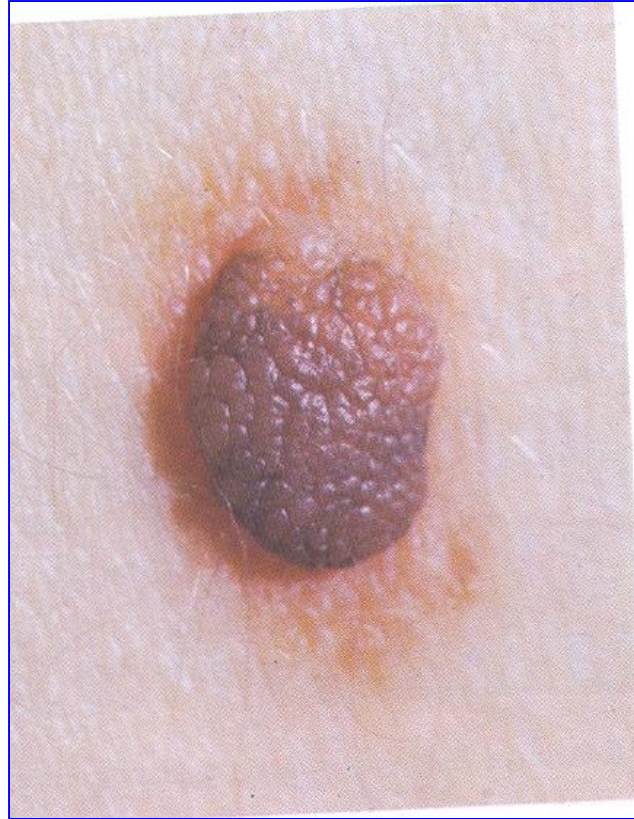
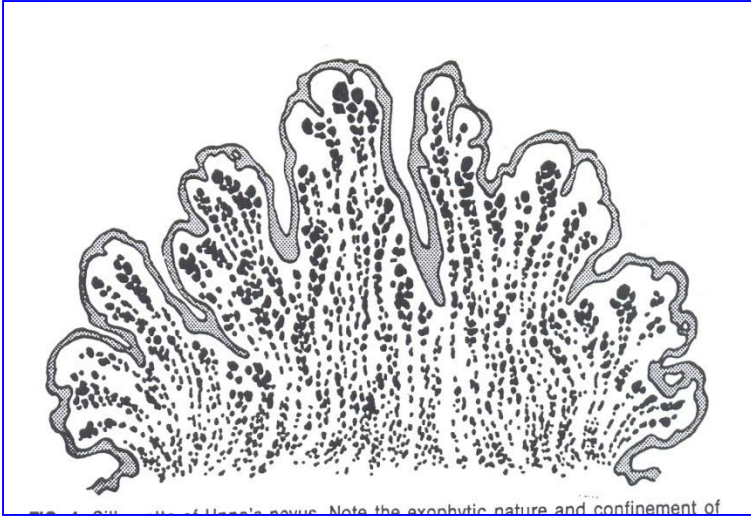
3. Spitz's naevus



4. Clark's naevus



Naevus Unna:

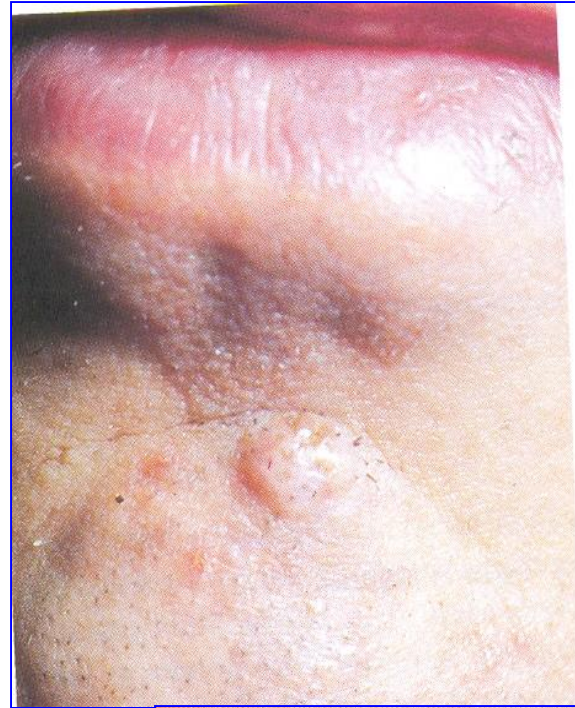


soft naevus

women neck and trunk

20% melanomas

Naevus Miescher:



women on the face

/nose, chin, forehead, around mouth/

no alteration toward melanoma

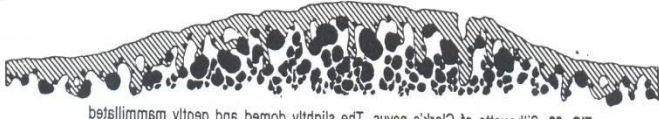


Naevus Miescher

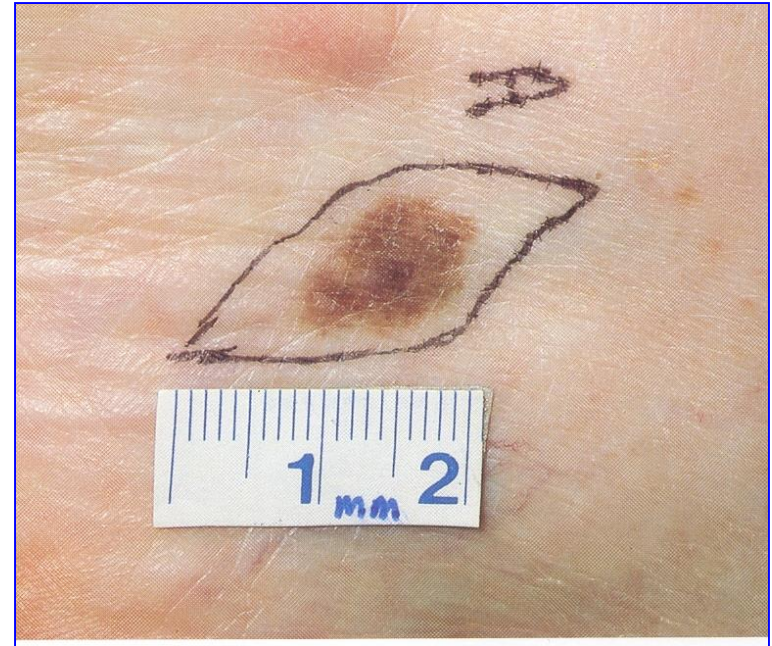
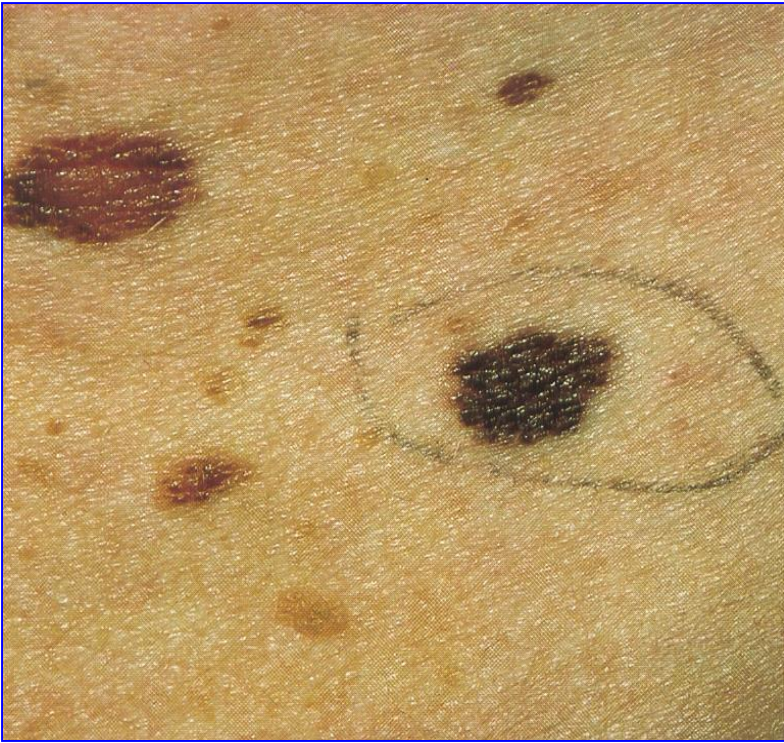


Naevus Clark:

dysplastic or atypical naevus



50 % melanoma



dysplastic or atypical naevi

50% melanomas

Dysplastic naevi are more often found in person with numerous naevi



Atypical naevus/mole syndrome



most on the trunk
irregular edges
>1 cm

sporadically
run in families as an AD

melanoma prone
CDKN2A mutation (9p21)

follow up 6-monthly
for life

Naevus Sutton /hallo nevus/



very often with vitiligo



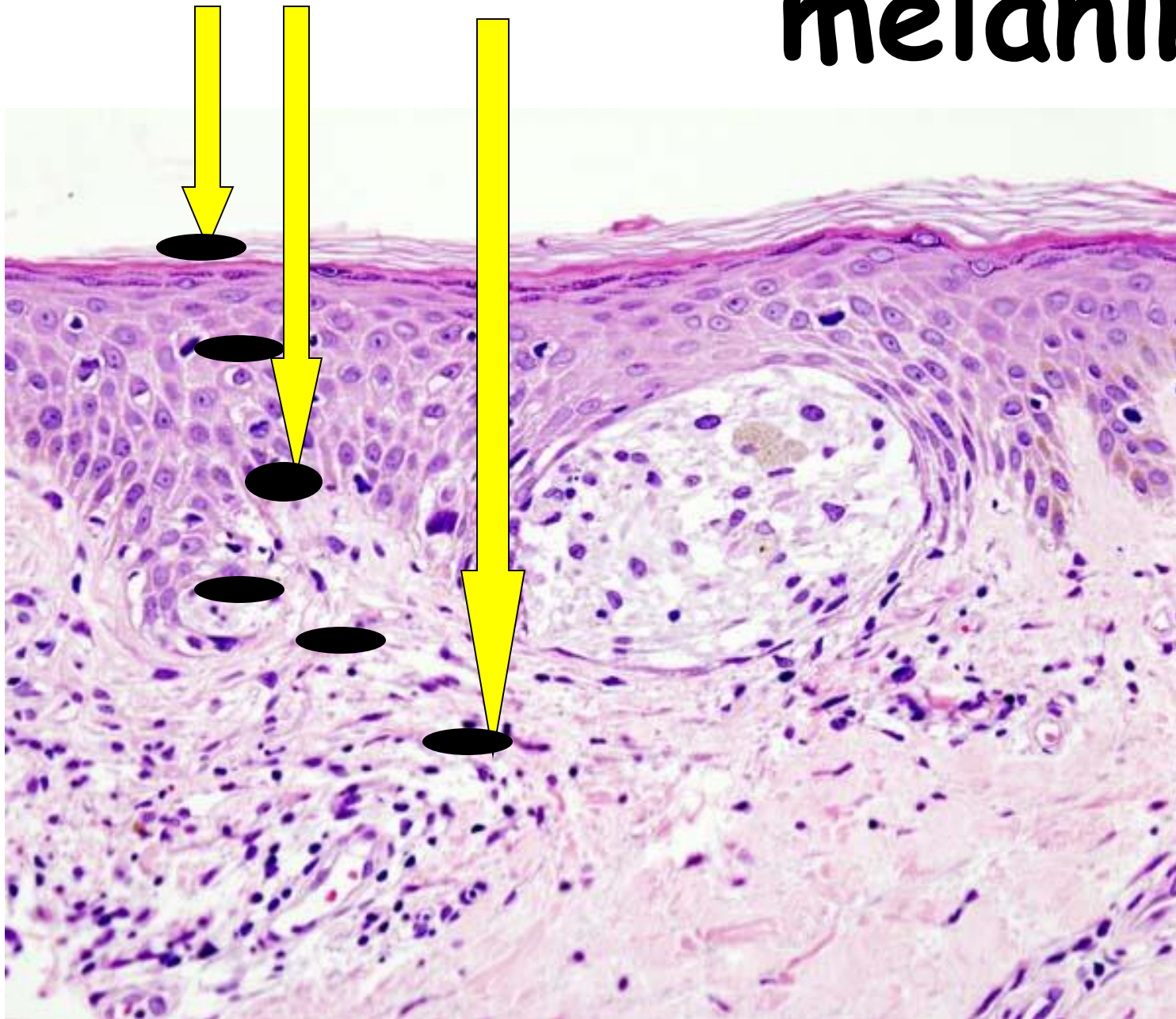
observation

UV protection

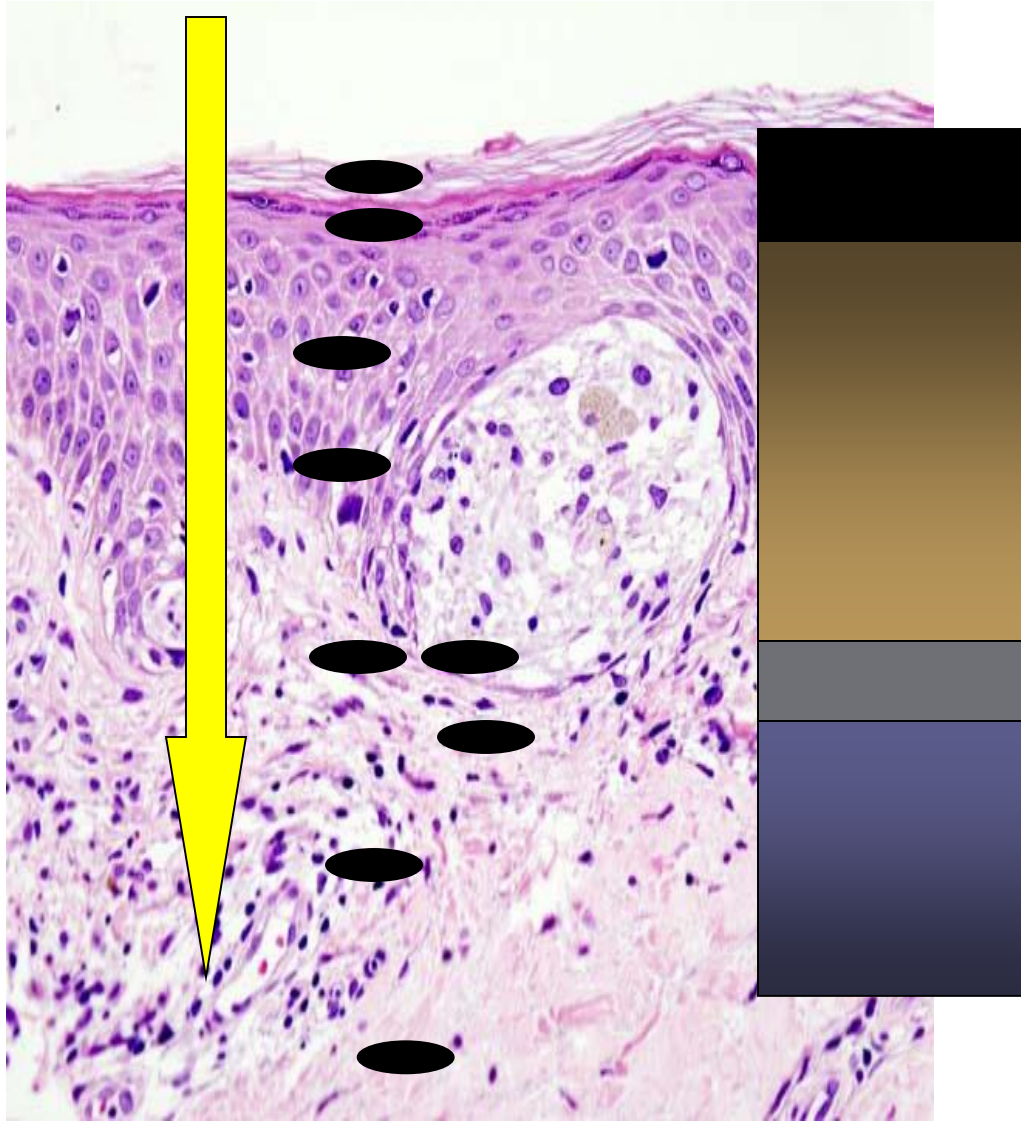
in older persons-be careful

expectation

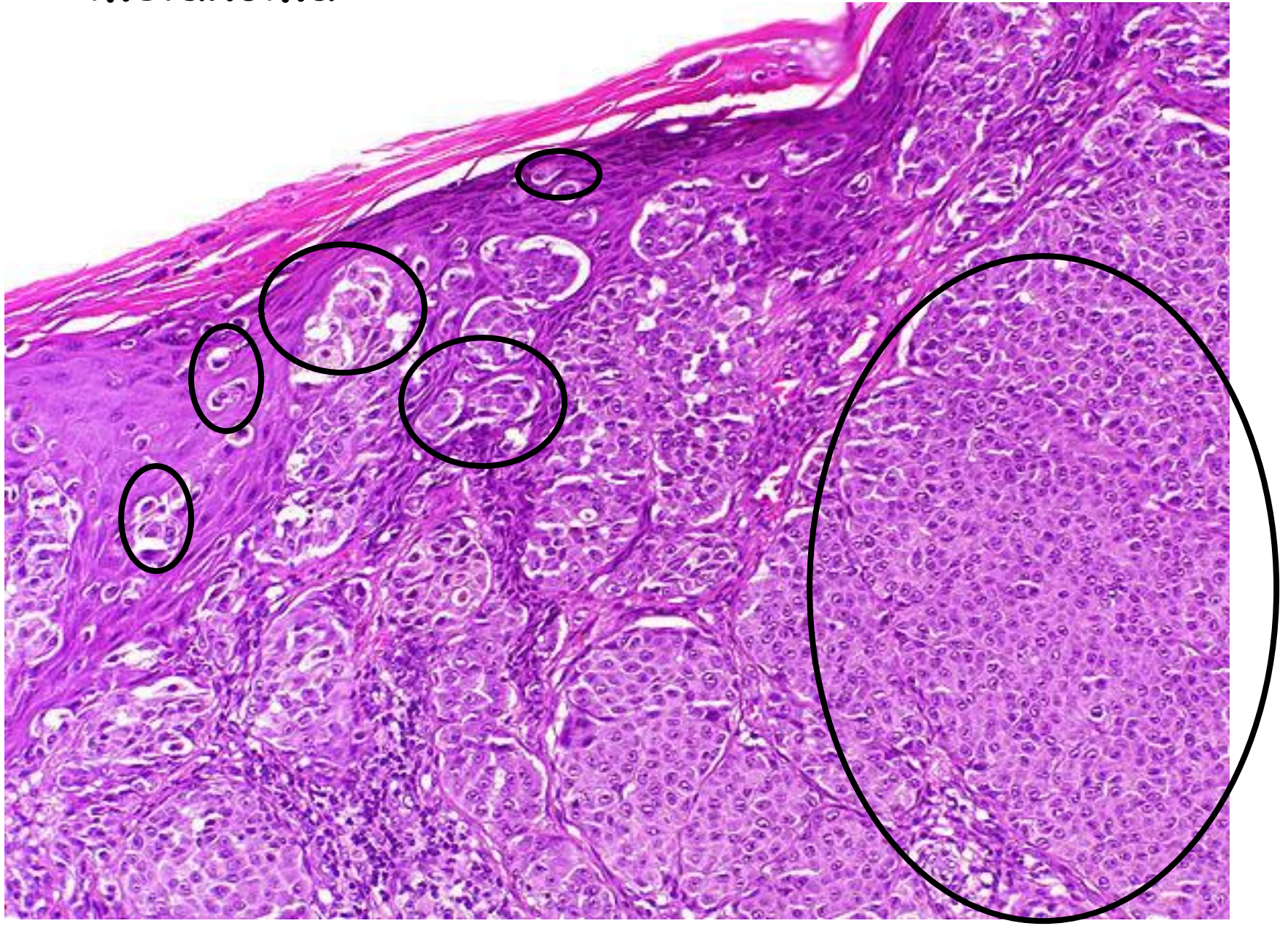
melanin

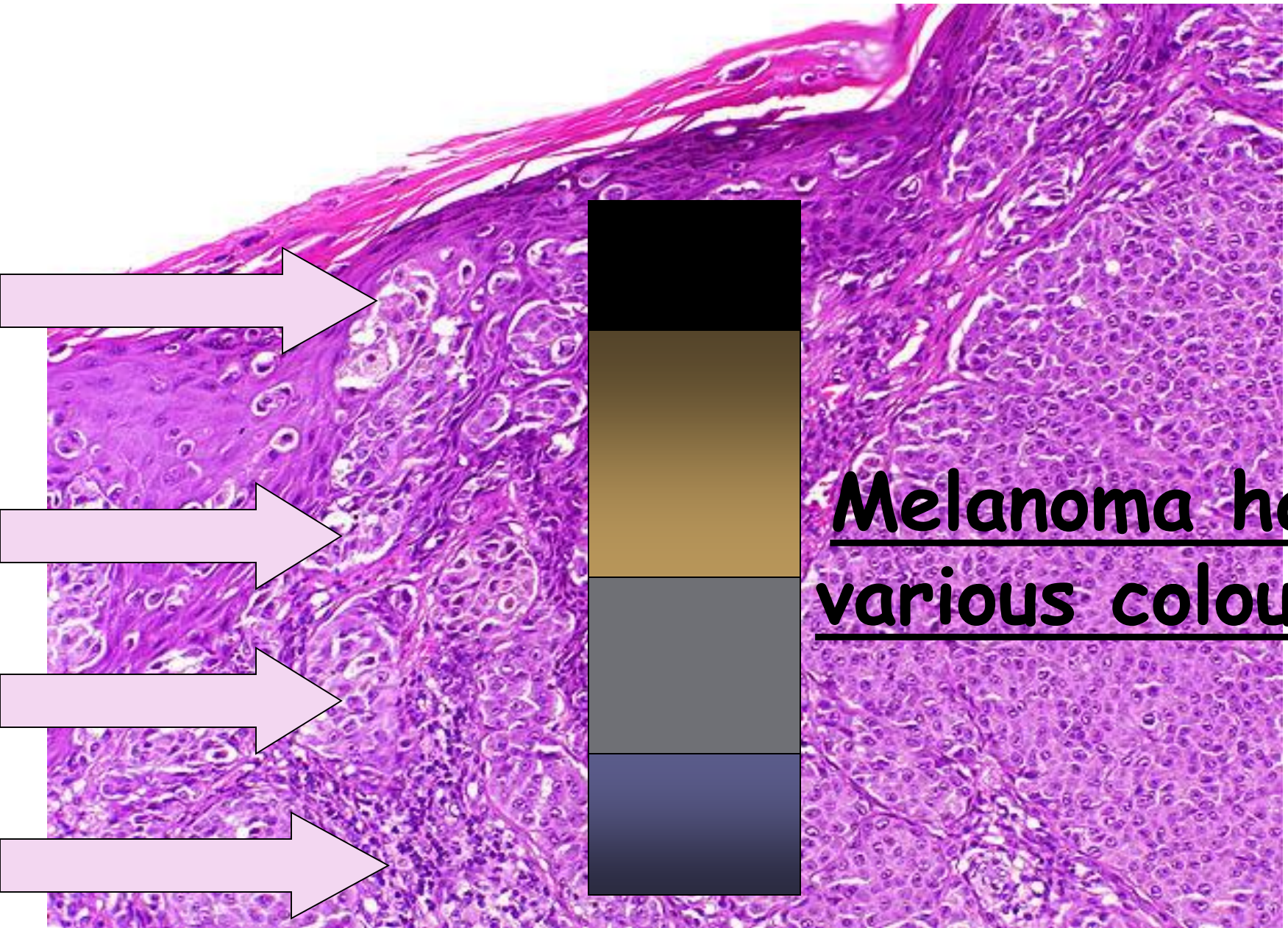


melanin



melanoma





Melanoma has
various colours

Complications:

Inflammation – pain and swelling (trauma, folliculitis)

Malignant changes – 0,5-10% (for large congenital naevi and atypical naevi of melanoma-prone families). It should be considered if the following changes occur:

- enlargement
- increased and decreased pigmentation
- altered shape
- altered contour
- inflammation and ulceration
- itch or bleeding

ABCDE

A - asymmetry

B - border

C - colour

D - diameter greater than 0,5 cm

E - evolution (change)

Treatment:

Excision is needed when:

- a naevus is ugly
- malignancy is suspected or is known risk
- naevus is repeatedly inflamed or traumatized

Sebaceous naevus



flat hairless area at birth usually in the scalp

more raised and yellow during puberty



Epidermoid cysts



often on scalp, face,
behind the ears and trunk

after rupture or squeezing
cheesy material comes out



Milia



small subepidermal
keratin cysts

face

seed-like papules

Chondrodermatitis nodularis helices (ear corn)



chronic inflammation
painful nodule
often in men

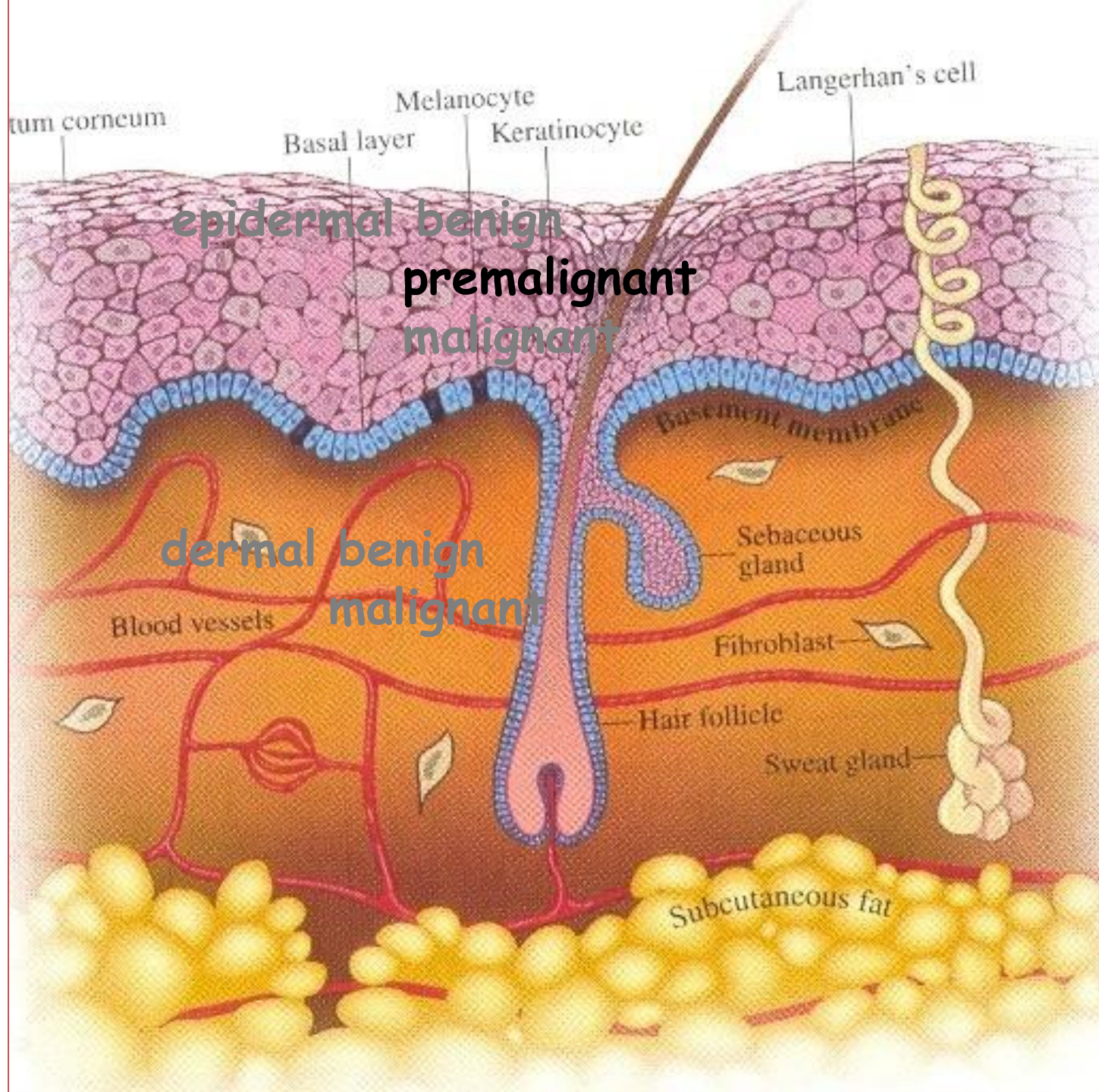
tender
prevents sleep

Treatment:
wedge resection

Chondrodermatitis nodularis helices (ear corn)







Premalignant lesions (carcinoma in situ)

long-lasting UV exposure

HPV (16 and 18)

exposure to chemicals- arsenic

- tar

chronic infections - osteomyelitis

- fistula

scars due to burns, TBC, DLE, LSA

scars after irradiation

Premalignant lesions

Keratosis actinica

Cheilitis actinica

Morbus Bowen

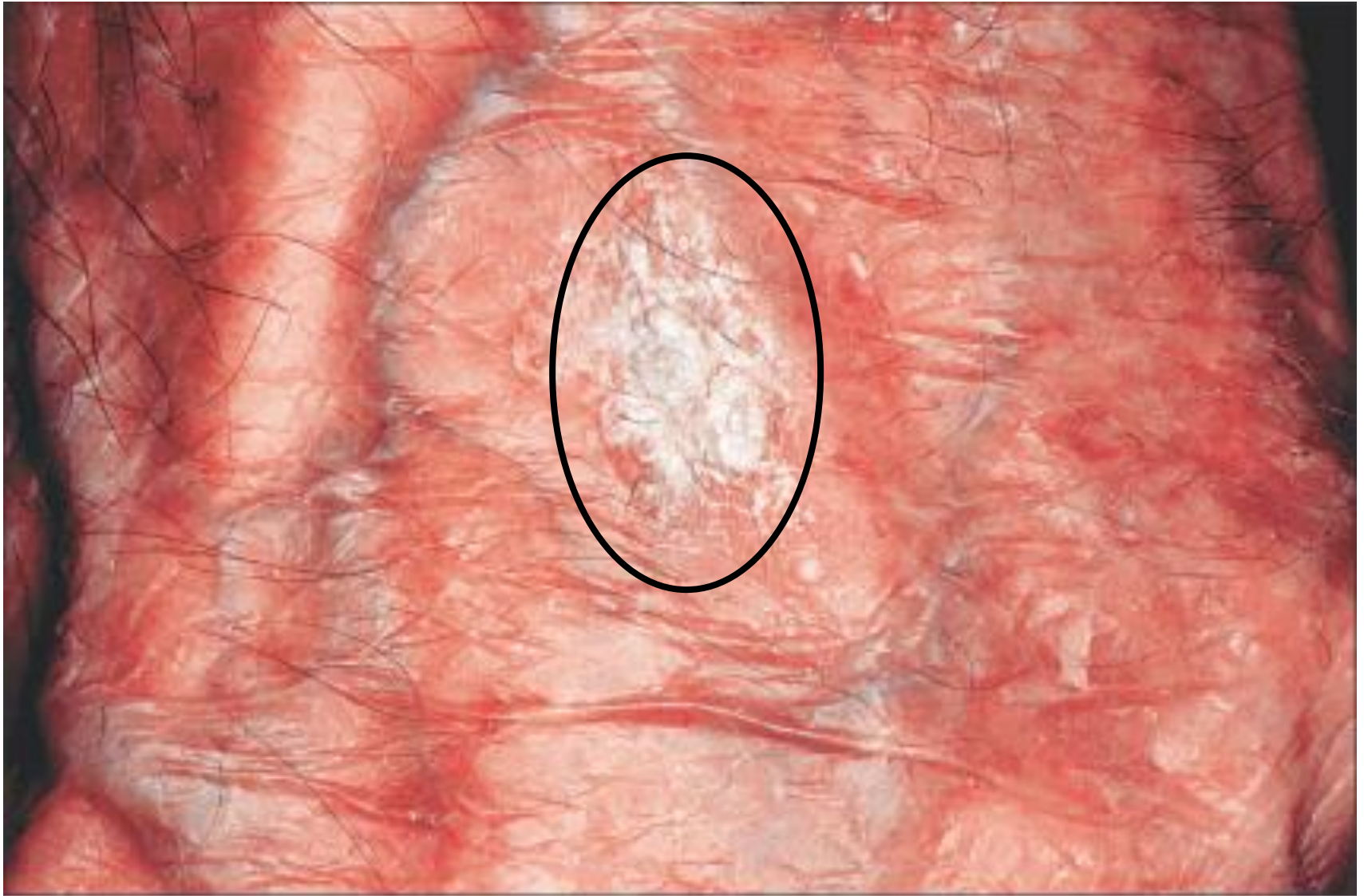
carcinoma planocellulare
in situ

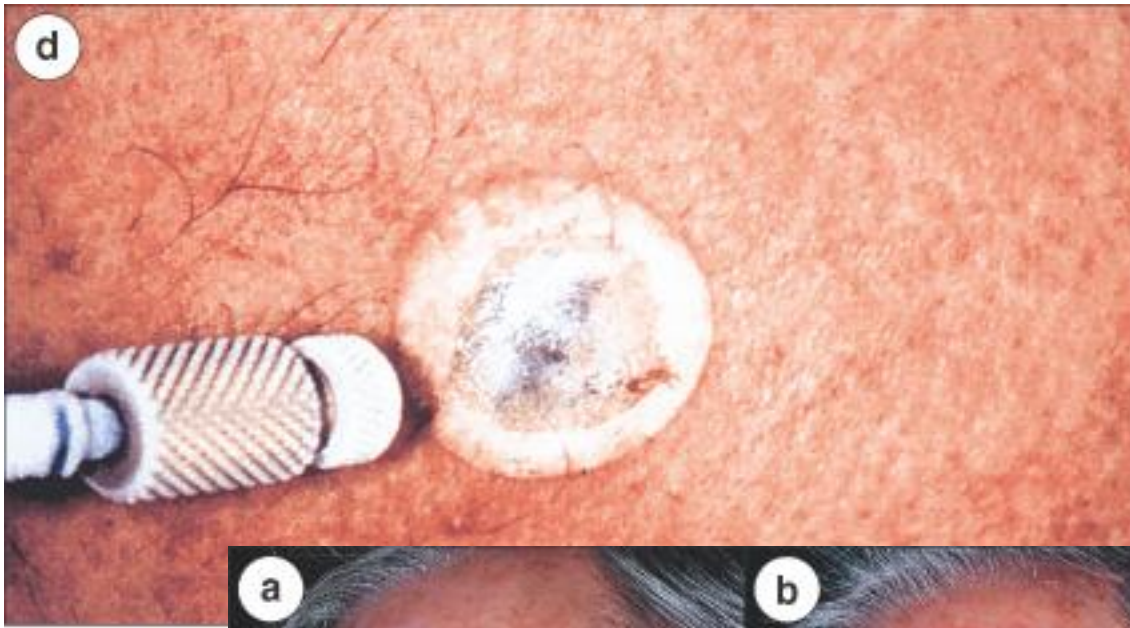
Keratosis actinica:



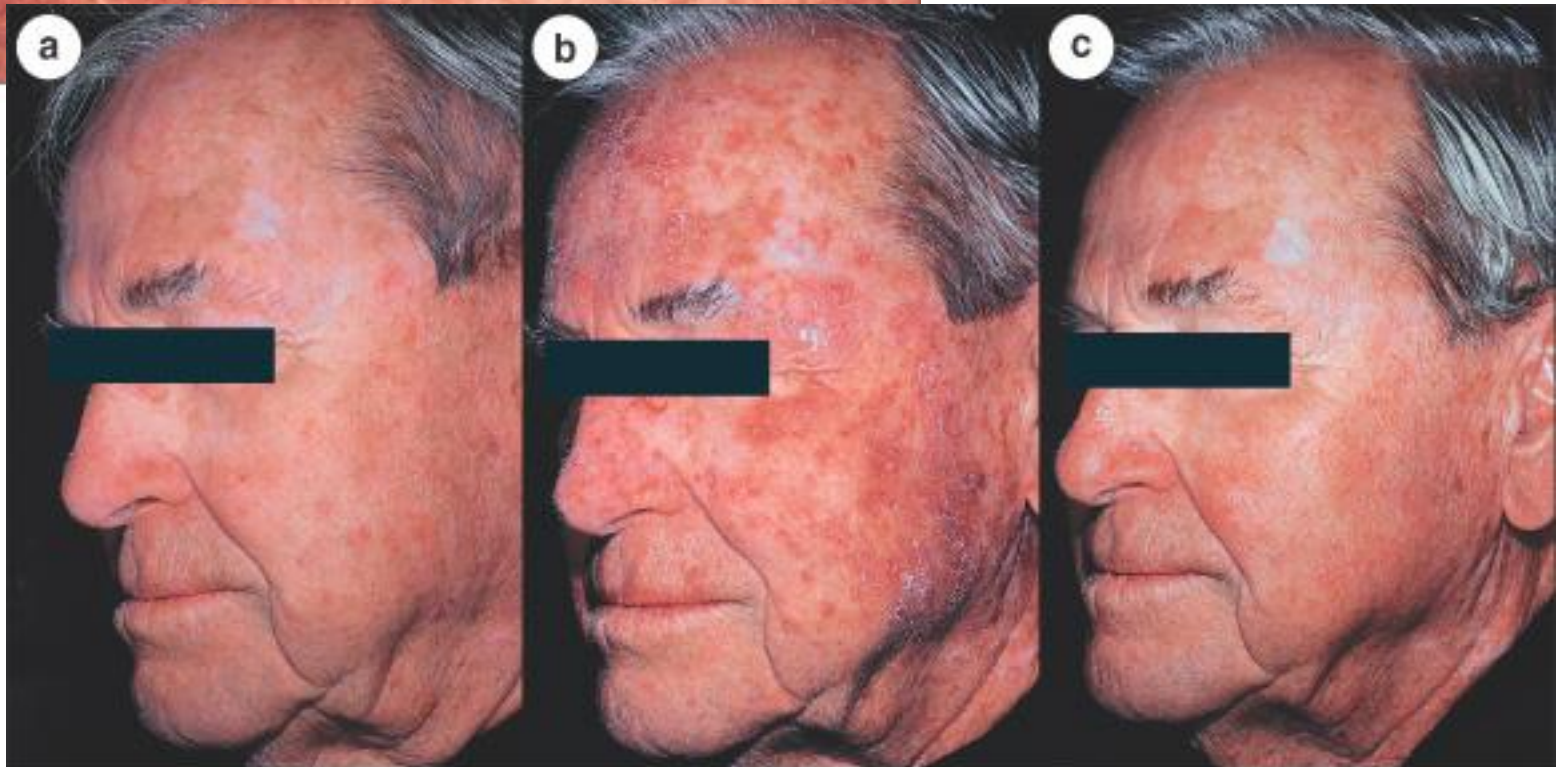
60% squamous cell carcinomas
1/3 of men over 70 years had AK

rough-surfaced lesions
sun-damaged skin
not seen in black skin





liquid nitrogen



5-FU
1-2xd
4-6 w



2-3x /week/16 weeks



lower cure rate



photodynamic therapy

Premalignant lesions

Keratosis actinica

Cheilitis actinica

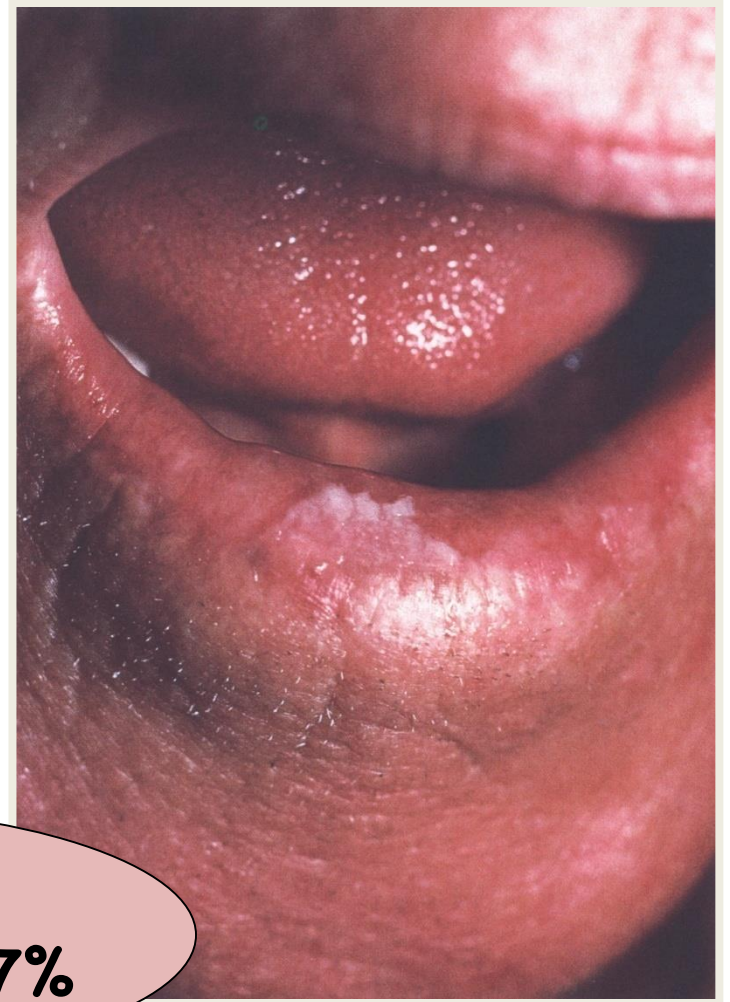
Morbus Bowen

Cheilitis actinica:

Therapy:

cryotherapy

5-FU cream



squamous cell
carcinoma in 17%

Premalignant lesions

Keratosis actinica

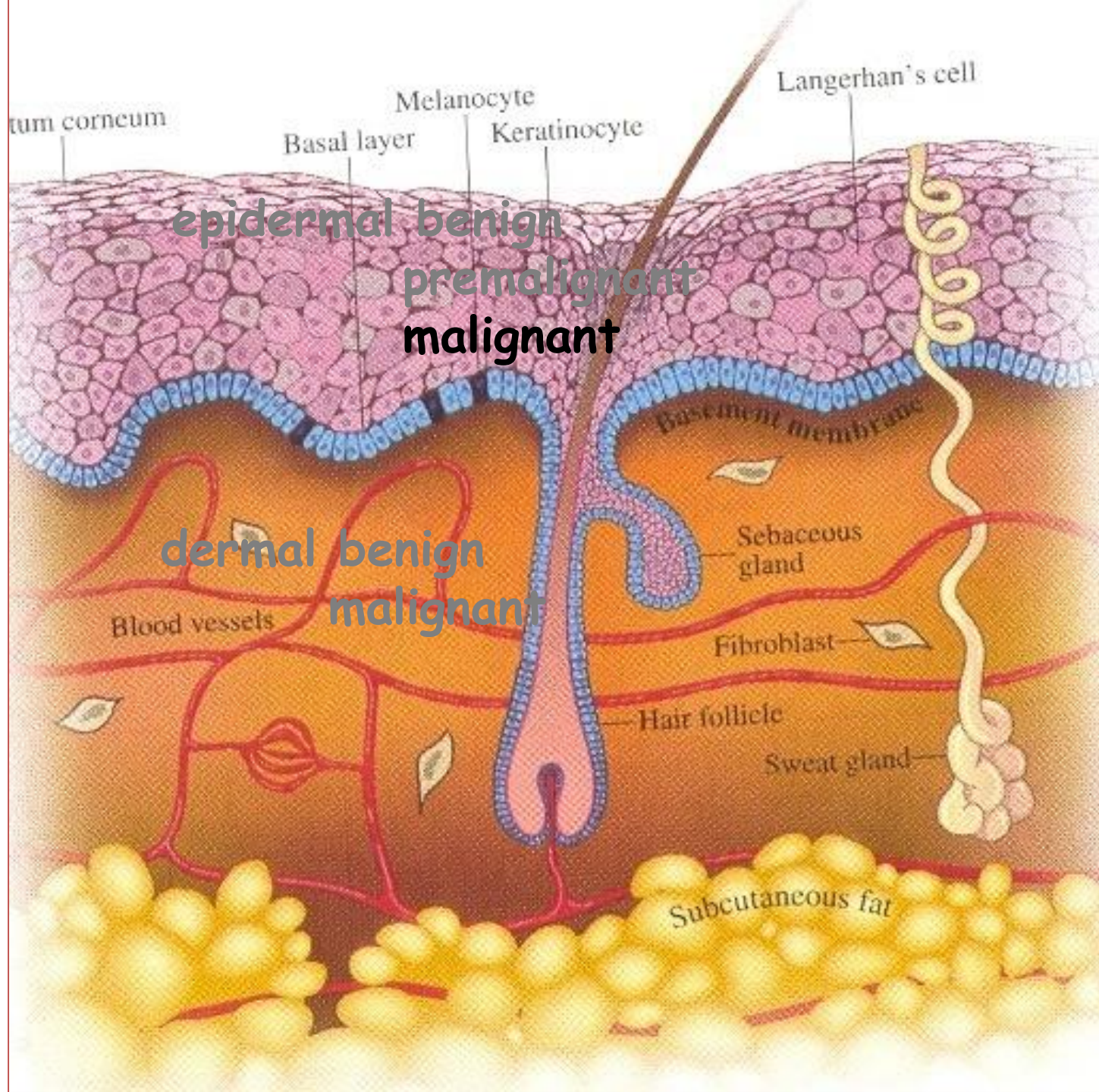
Cheilitis actinica

Morbus Bowen

carcinoma planocellulare
in situ

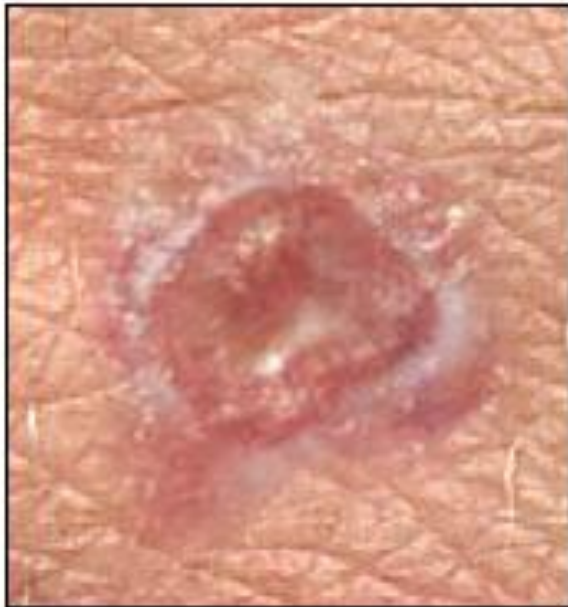
Bowen's disease



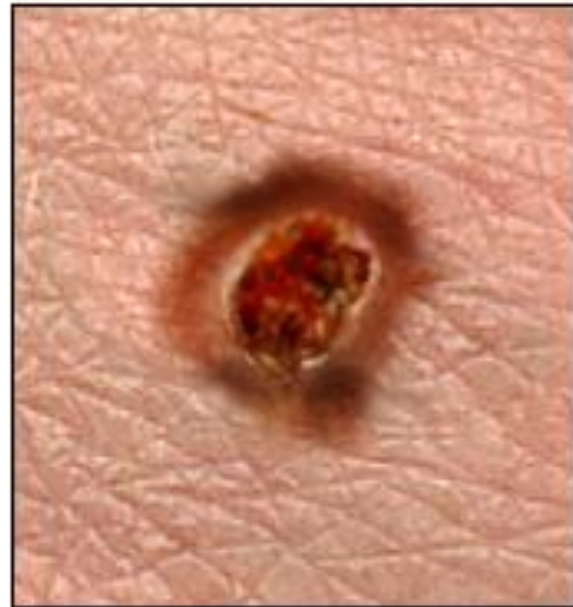


Malignant epidermal tumours

basal cell carcinoma

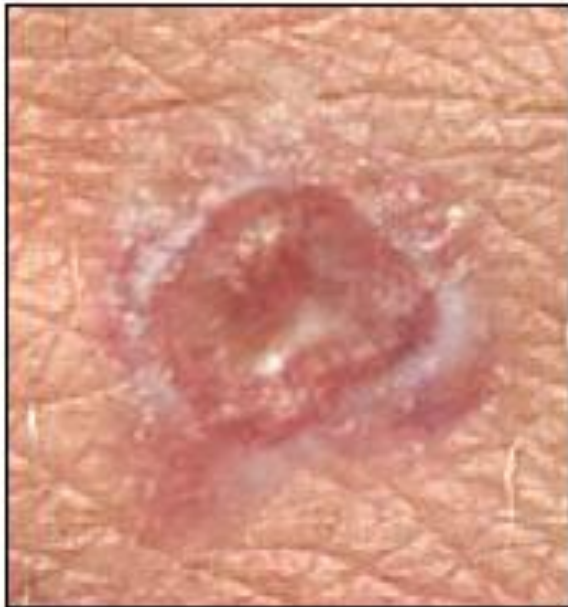


squamous cell carcinoma



Malignant epidermal tumours

basal cell carcinoma



squamous cell carcinoma



Basal cell carcinoma



white race

sunny areas

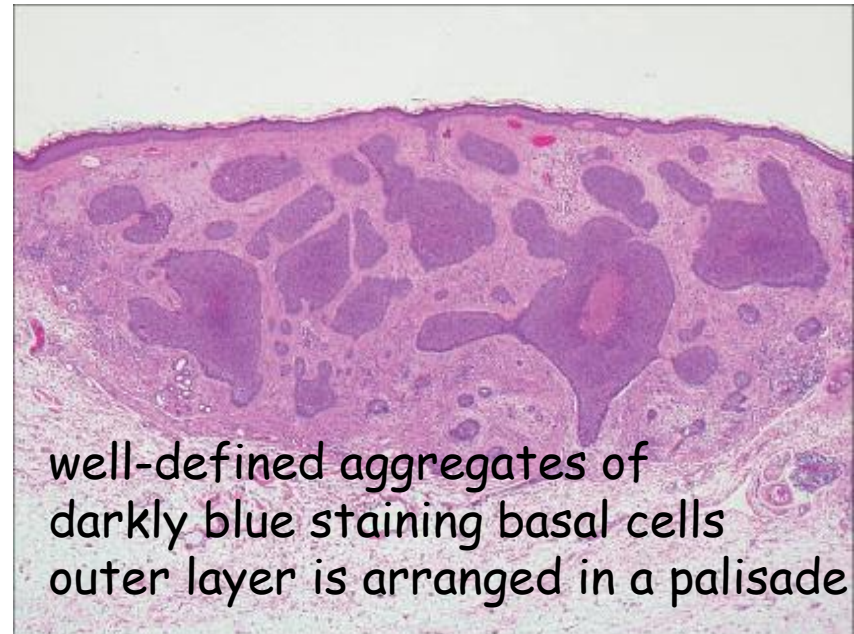
high latitude

near equator

**prolonged sun
exposure**

Basal cell carcinoma (rodent ulcer)

85% appears on the face, the most common
invade locally, very rarely metastasize



**nodulo-ulcerative
basal cell carcinoma**



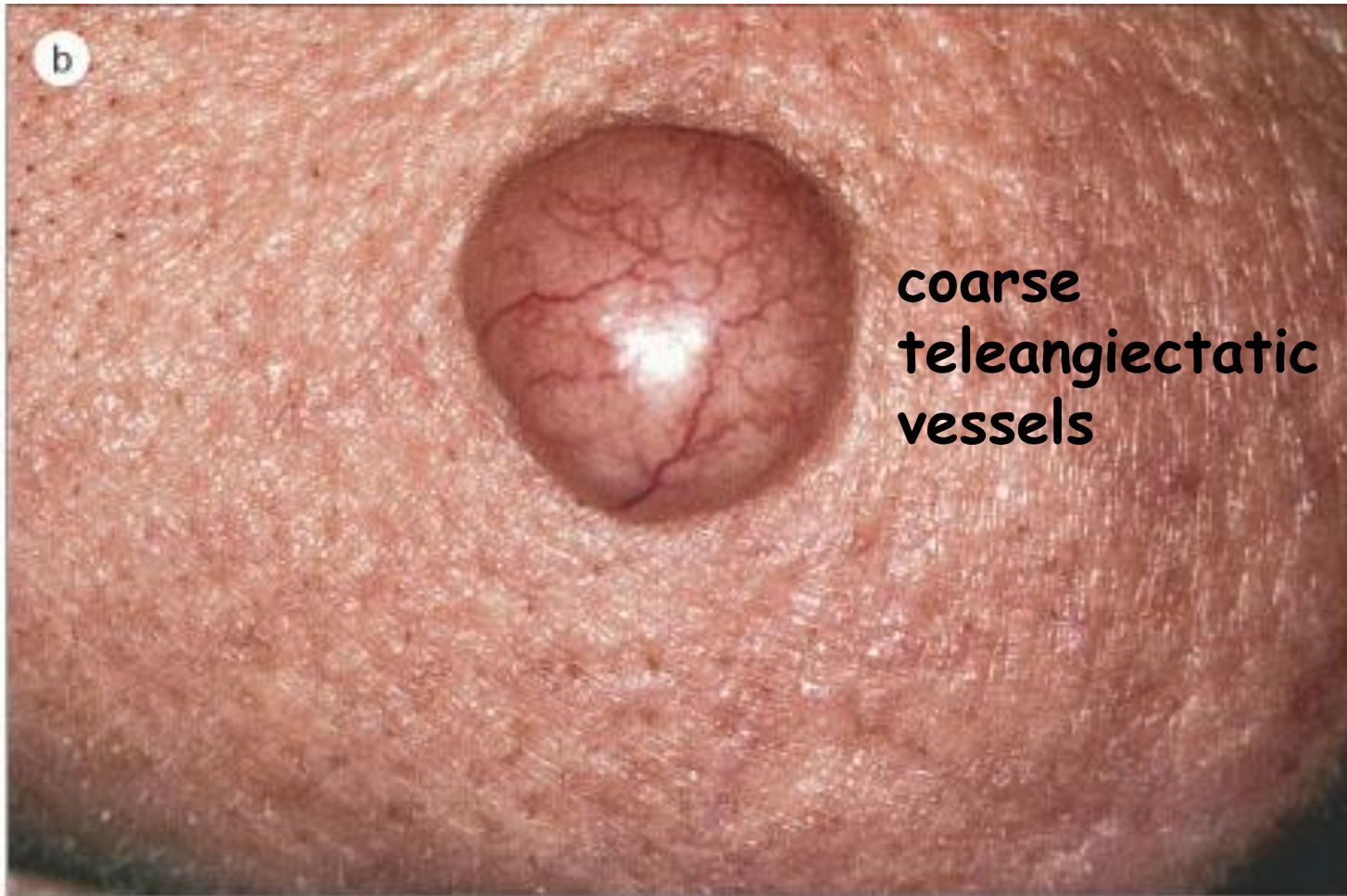
palisade - a high fence



1-2 cm/5-10 years

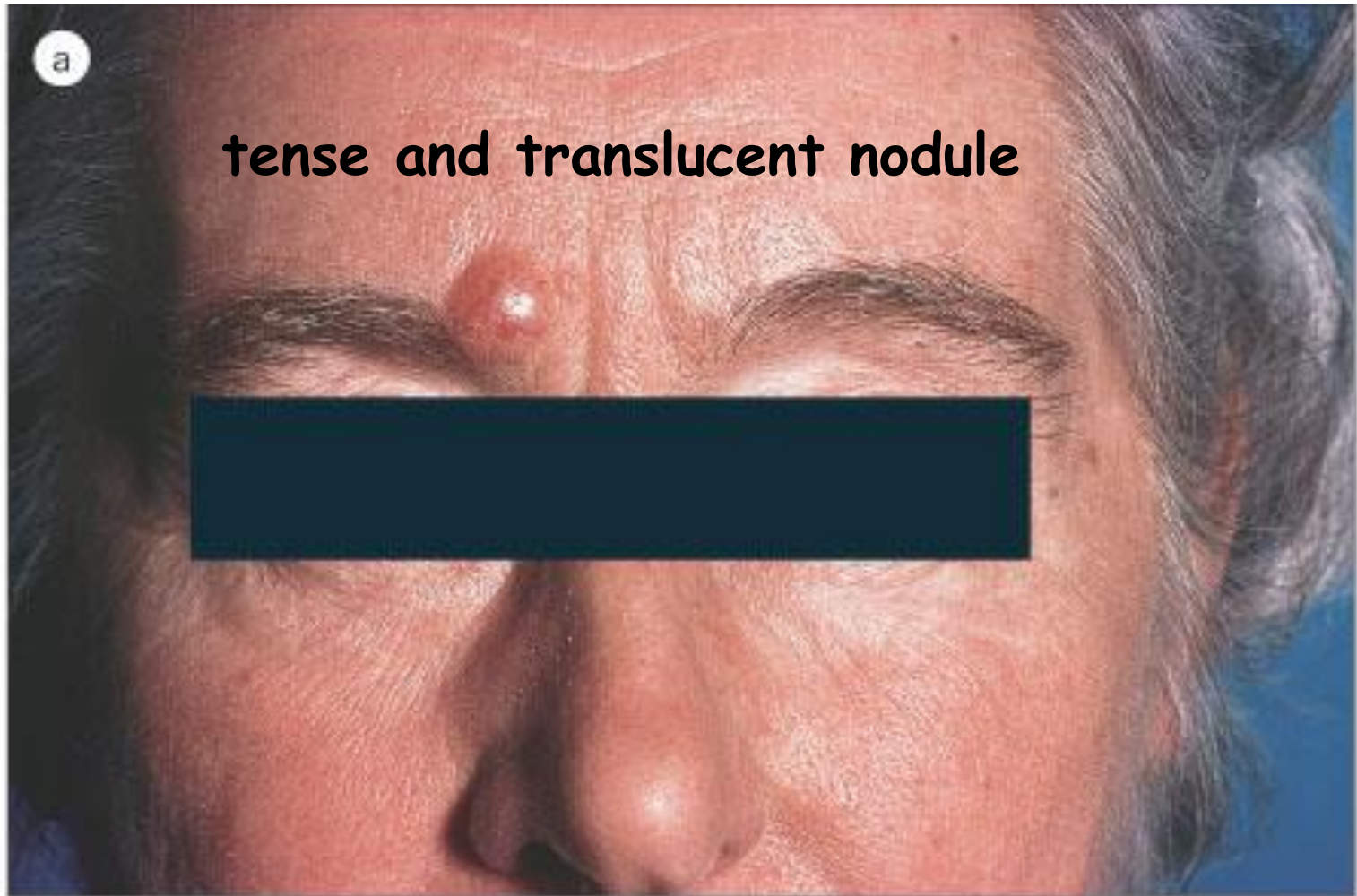


coarse telangiectatic vessels



**coarse
teleangiectatic
vessels**

nodular basal cell carcinoma



cystic type of basal cell carcinoma



cicatricial (morphoeic) type of BCC



**slowly expanding
enlarging scar**



**superficial (multicentric)
type of BCC**

the most often on the trunk
can grows to more than 10 cm



pigment may be present
in all types of BCC

pigmented BCC

Treatment:

surgical excision (0,5 cm of surrounding normal skin)

Mohs's surgery (larger than 1 cm) , nose, inner canthus
nasolabial fold

radiotherapy - if is surgery contraindicated

PDT, cryosurgery - for superficial types of BCC

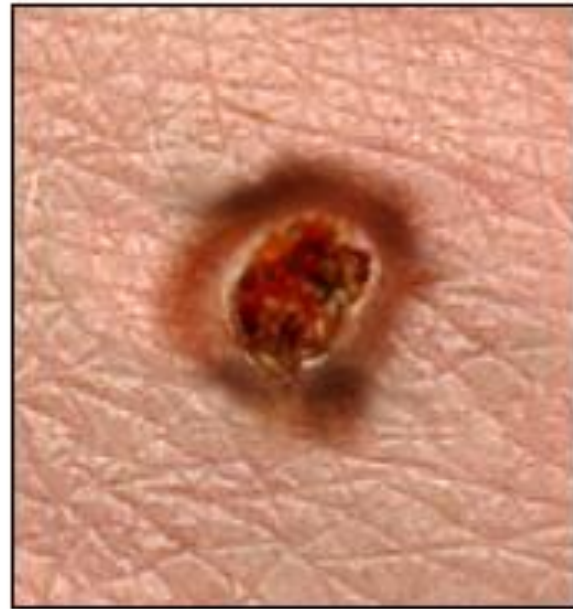
follow-up for up to 5 years

Malignant epidermal tumours

basal cell carcinoma



squamous cell carcinoma





**squamous cell
carcinoma**

SCC on photo-damaged skin



less aggressive

most SCC carry typical UV-induced p53 mutation - significant part UV radiation

SCC in scar after X-rays



more aggressive

SCC in scar after

chronic draining sinuses

chronic ulcers

previous thermal injury

chronic inflammation

**most likely
to metastasize**

SCC of the lower lip



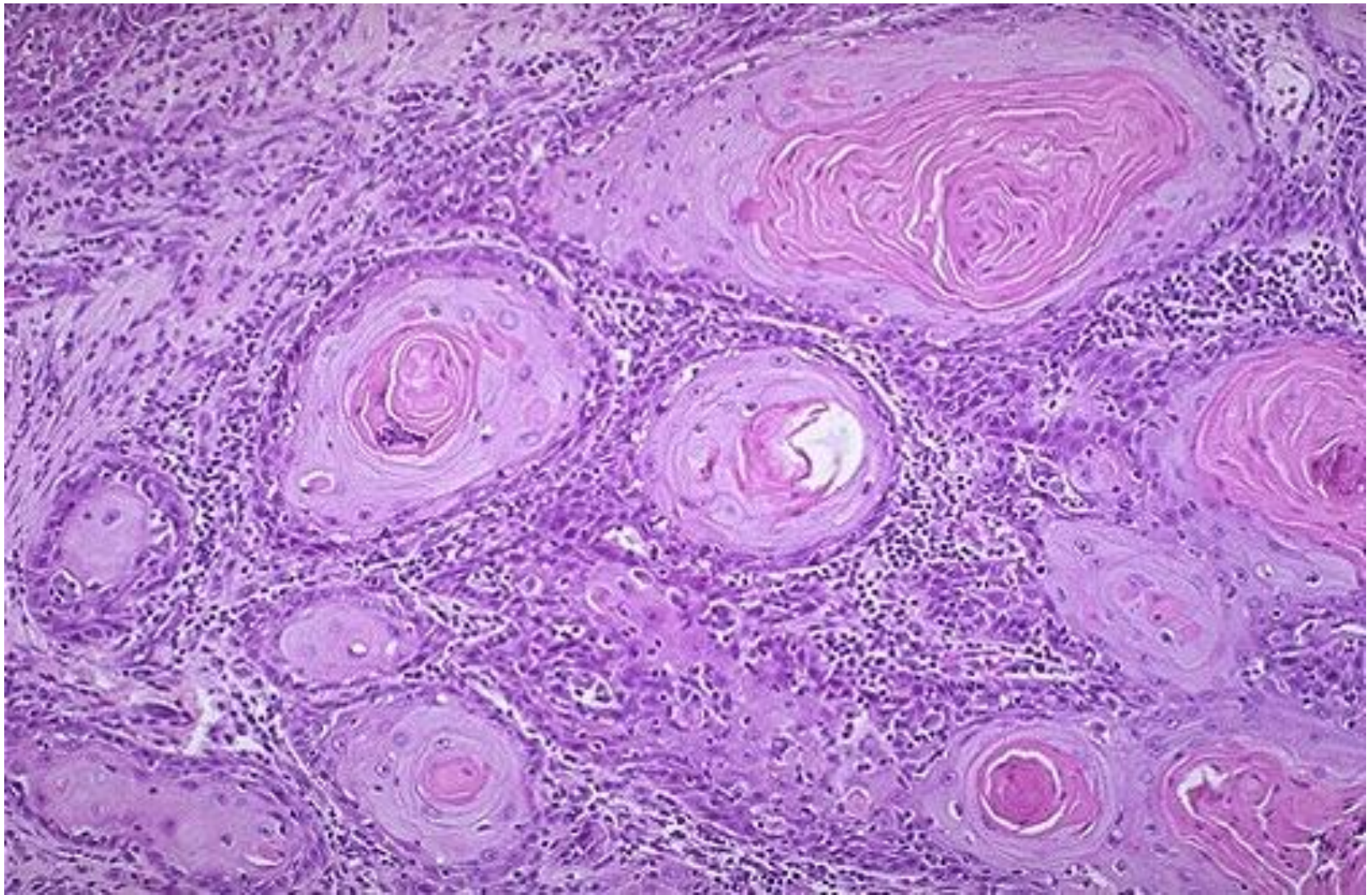
always have to palpate
lower lip



**surgical excision
and irradiation**

**SCC of lower lip
with metastasis**





SCC with multiple keratin pearls

Treatment:

surgical excision (0,5 cm of surrounding normal skin)

Mohs's surgery (larger than 1 cm) , for high risk tumours

radiotherapy - if is surgery contraindicated

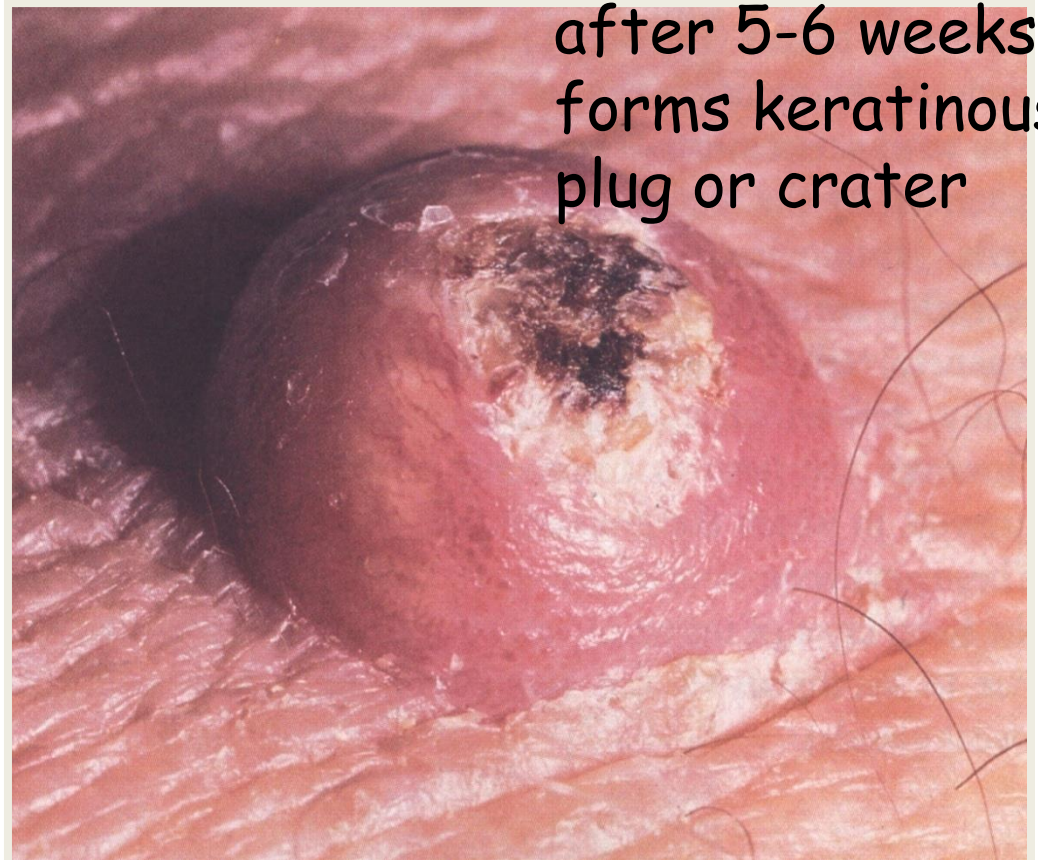
follow-up for up to 5 years

Other types of SCC

Keratoacanthoma:

Rapidly growing squamous cell tumours do not
invade and resolve spontaneously

very short
history:
1 cm/1month



after 5-6 weeks
forms keratinous
plug or crater



tar
mineral oils
UV radiation
immunosuppression



keratoacanthoma

2/3 on the face



the lesion may resolve spontaneously over 6-12 months

Treatment:

surgical excision

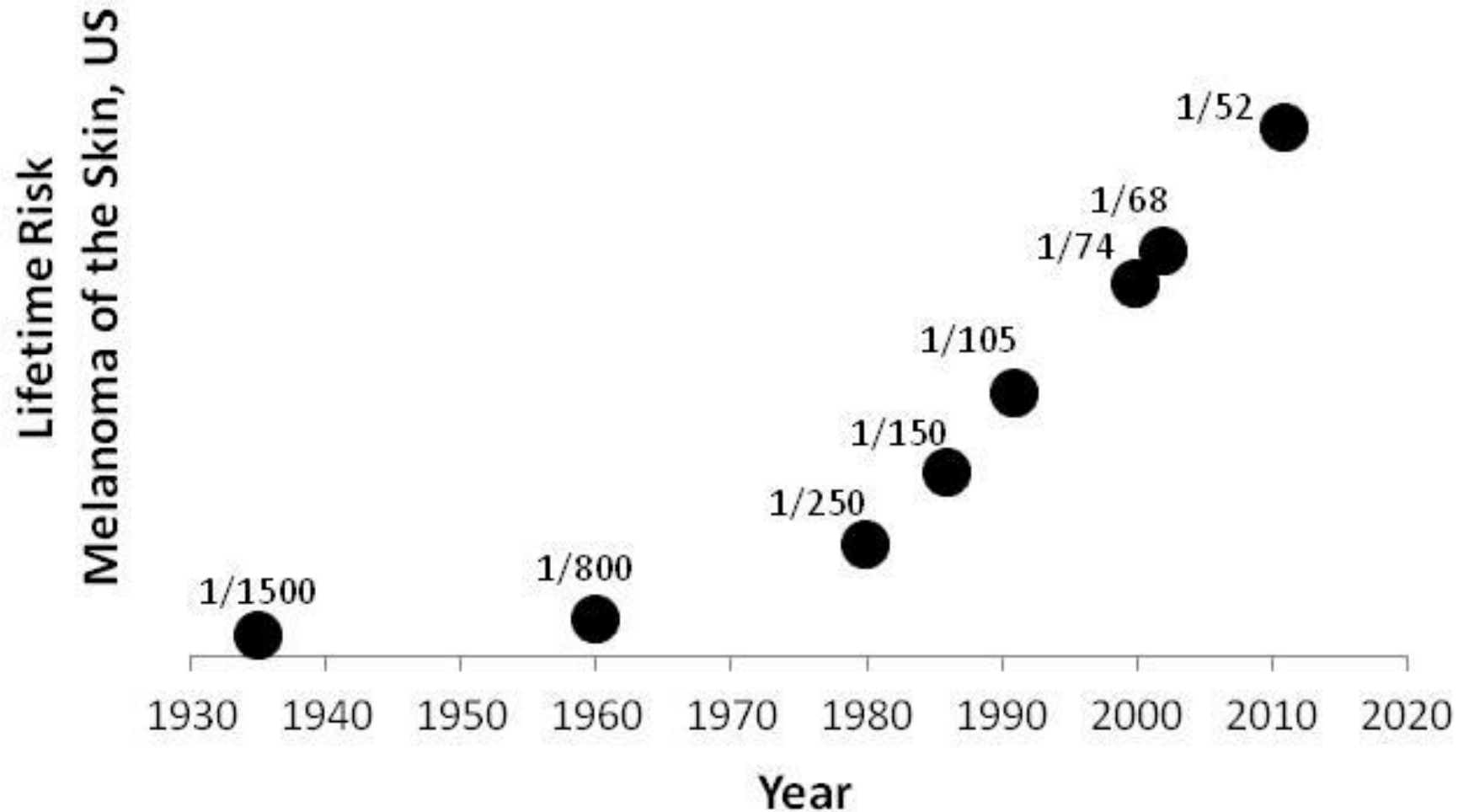


Melanoma

attracts a huge amount of publicity - often lethal

the incidence is **doubling every 10 years**

higher incidence in white race



**Increasing incidence of
melanoma of the skin, US.**

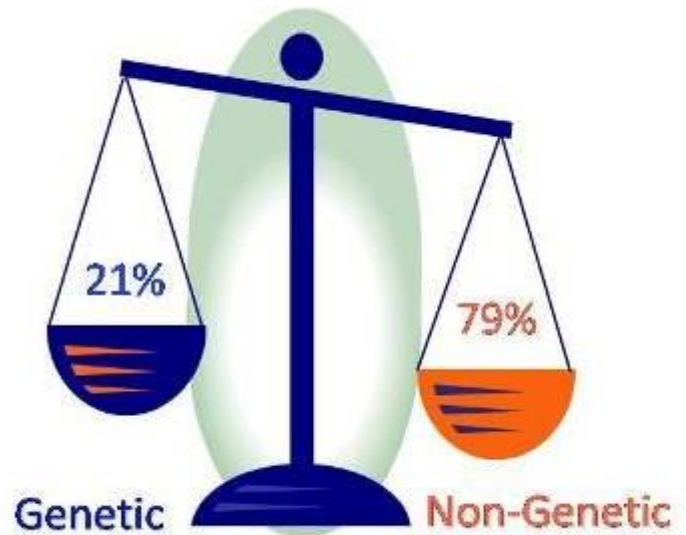
Causative agents

Susceptibility genes

Susceptible phenotypes

Sunlight

Pre-existing melanocytic naevi



Causative agents

Susceptibility genes

Susceptible phenotypes

Sunlight

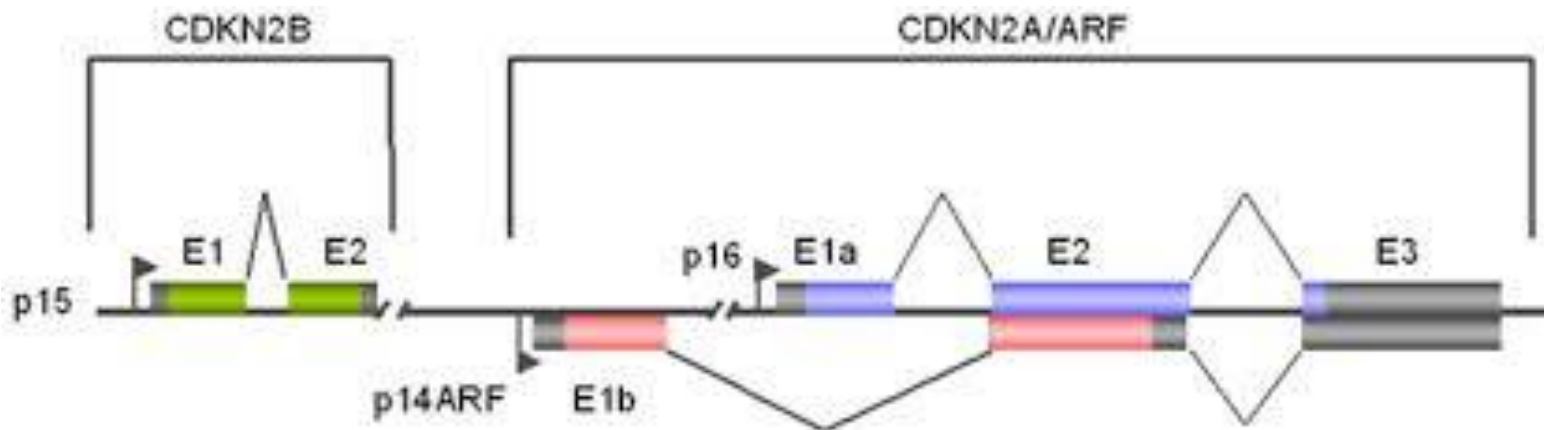
Pre-existing melanocytic naevi



Susceptibility genes

rarely (around 6%) melanomas are familial
occurring in families where two or more first-degree relatives have a melanoma

molecular defects in tumor suppressor genes and oncogenes have been linked (cycline dependant kinase inh -CDKN2A)



the most attracting and intersting area lies on chromosome 9p, known as CDKN2A

Causative agents

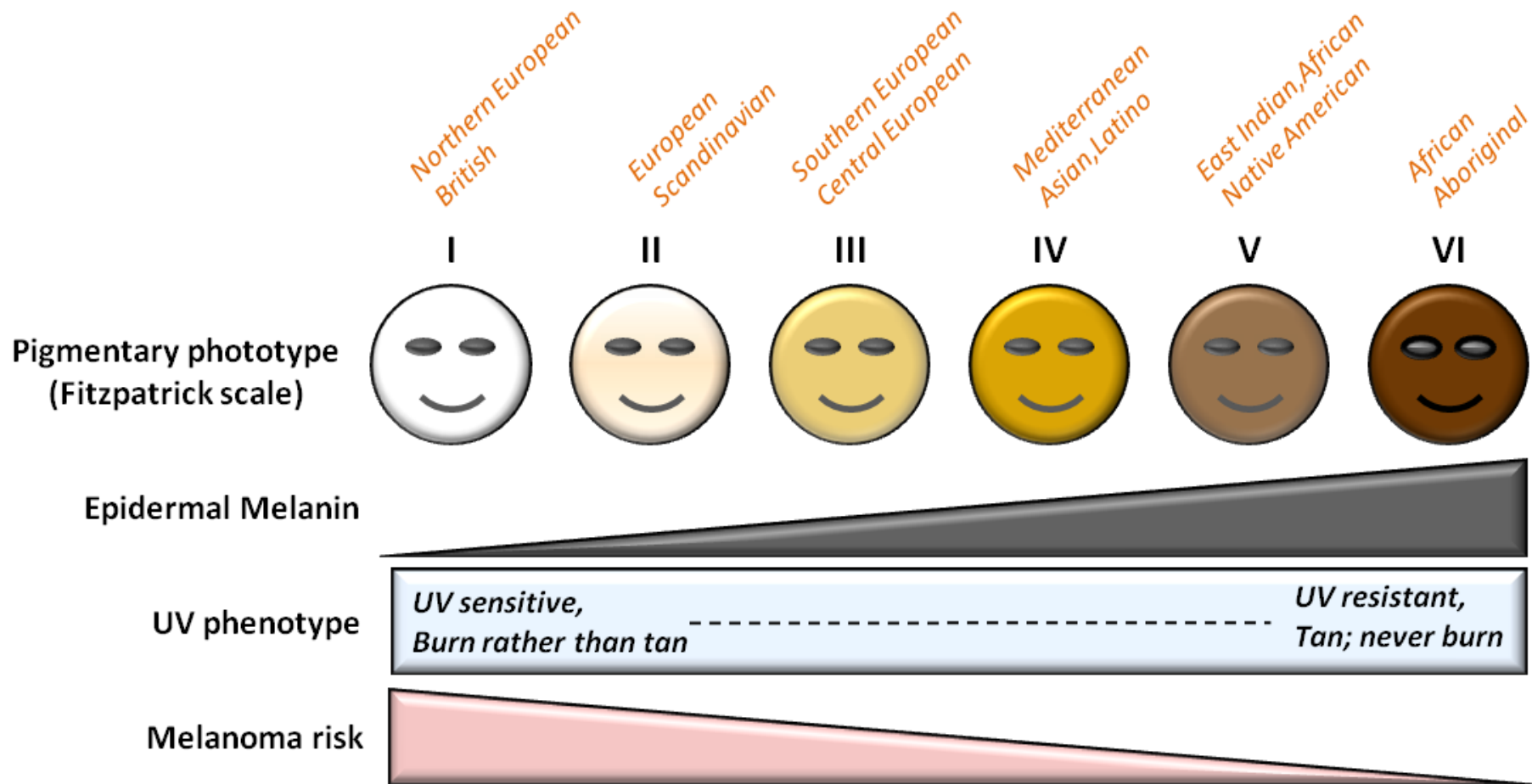
Susceptibility genes

Susceptible phenotypes

Sunlight

Pre-existing melanocytic naevi





Melanoma risk varies according to skin complexion

Causative agents

Susceptibility genes

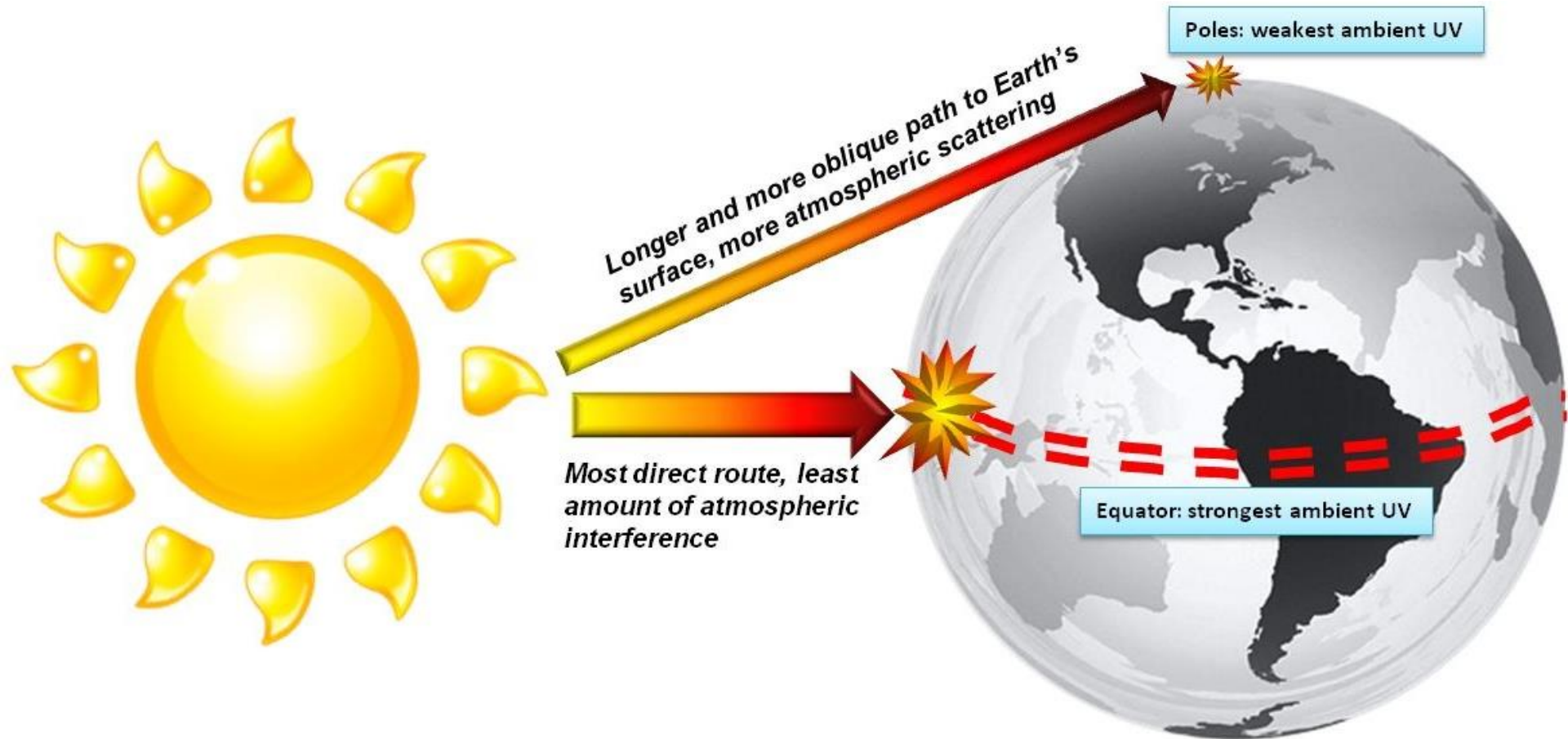
Susceptible phenotypes

Sunlight

Pre-existing melanocytic naevi



the incidence and mortality increase with decreasing latitude
number of sunburns more relevant than cumulative UV dose



**Strength of ambient UV varies with
geographic location**

Causative agents

Susceptibility genes

Susceptible phenotypes

Sunlight

Pre-existing melanocytic naevi





in 30% of melanomas
is pre-existing naevus

the risk is highest in those with
**atypical naevi, congenital or many
banal melanocytic navi**

Prevention

avoidance of excessive sun exposure and tanning booths

sunscreen during outdoor activities

the sunscreen reapplied every 2 hours

early diagnosis - publicity campaign, self examination

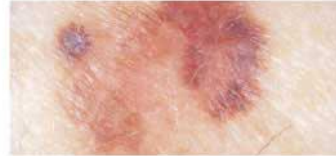
MOLE

MELANOMA

Asymmetry



Round and symmetrical



Asymmetrical

Borders



Regular and geometric

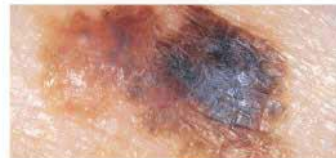


Irregular and jagged

Color



One Color

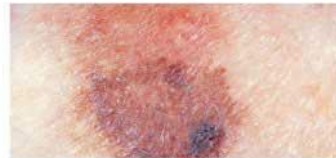


Several: light to dark

Diameter

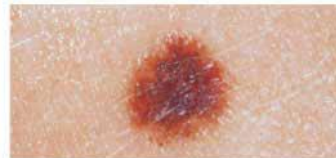


Small: less than 6 mm



Big: greater than 6 mm

Evolution



Evolutionary in its size, color or thickness

Step 1



Examine body front and back in mirror, then right and left sides, arms raised.

Step 2



Bend your elbows. Look carefully at forearms, back of upper arms, and palms.

Step 3



Next, look at the back of your legs and feet and spaces between your toes and soles.

Step 4



Examine the back of your neck and scalp with a hand mirror.

Step 5



Finally, check your back, buttocks and genital area with a

1

Examine your face, especially the nose, lips, mouth, and ears - front and back. Use one or both mirrors to get a clear view.



2

Thoroughly inspect your scalp, using a blow dryer and mirror to expose each section to view. Get a friend or family member to help, if you can.



3

Check your hands carefully: palms and backs, between the fingers and under the fingernails. Continue up the wrists to examine both front and back of your forearms.



4

Standing in front of the full-length mirror, begin at the elbows and scan all sides of your upper arms. Don't forget the underarms



5

Next focus on the neck, chest, and torso. Women should lift breasts to view the underside.





properly applied sun protective cream 20' before sunexposure



**around 30 gr
for whole
body**

1 tube/ monthly

Wide brimmed hat
(7.5 cm or greater)

Sunglasses

Long sleeves
and pants



Factors increasing sun protection of clothing

Increased tightness
of fabric weave

Wearing dry clothing

Type of fabric (polyester >
nylon, silk, wool > cotton, rayon)

Loose fitting items

Pre-washing

Chemical additives (e.g.
optical whitening agents)

may provide a
false sense of
security

Melanoma...

Clinical feature

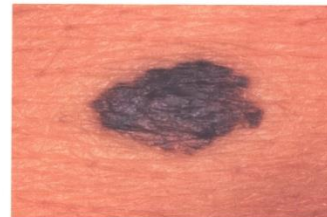
Lentigo maligna melanoma
10-20%

6. decade
long-standing superficial
phase



Melanoma...

Superficial spreading
melanoma 55%



4. and 5. decade

superficial phase with regression
lasts for few months to 2 years

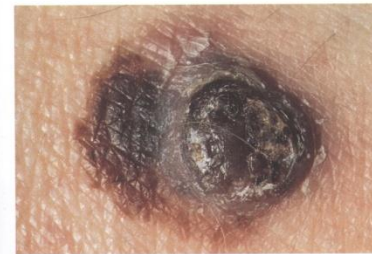
various colours

Melanoma...

Nodular melanoma
15%

5. and 6. decade
often in males
without preceeding in situ
phase (agressive)

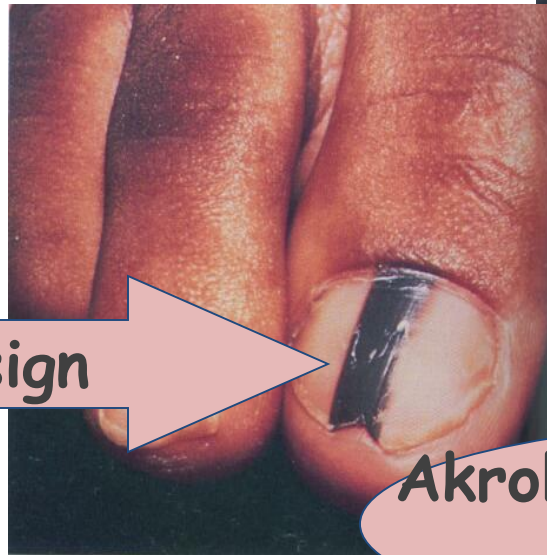
could be amelanotic



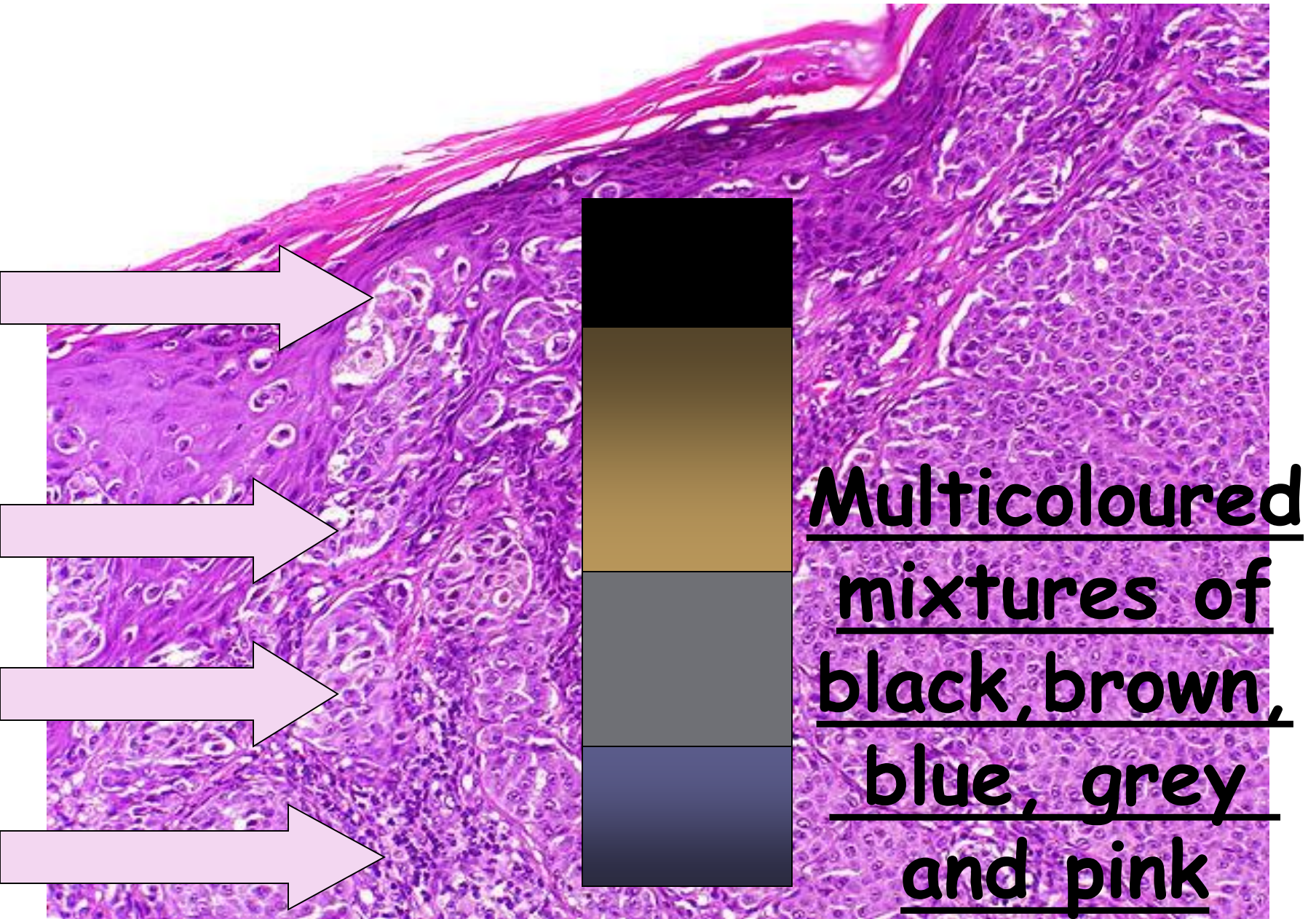
Melanoma...

palms and soles
mucosa

Hutchin's sign



Acrolentiginous melanoma
10%



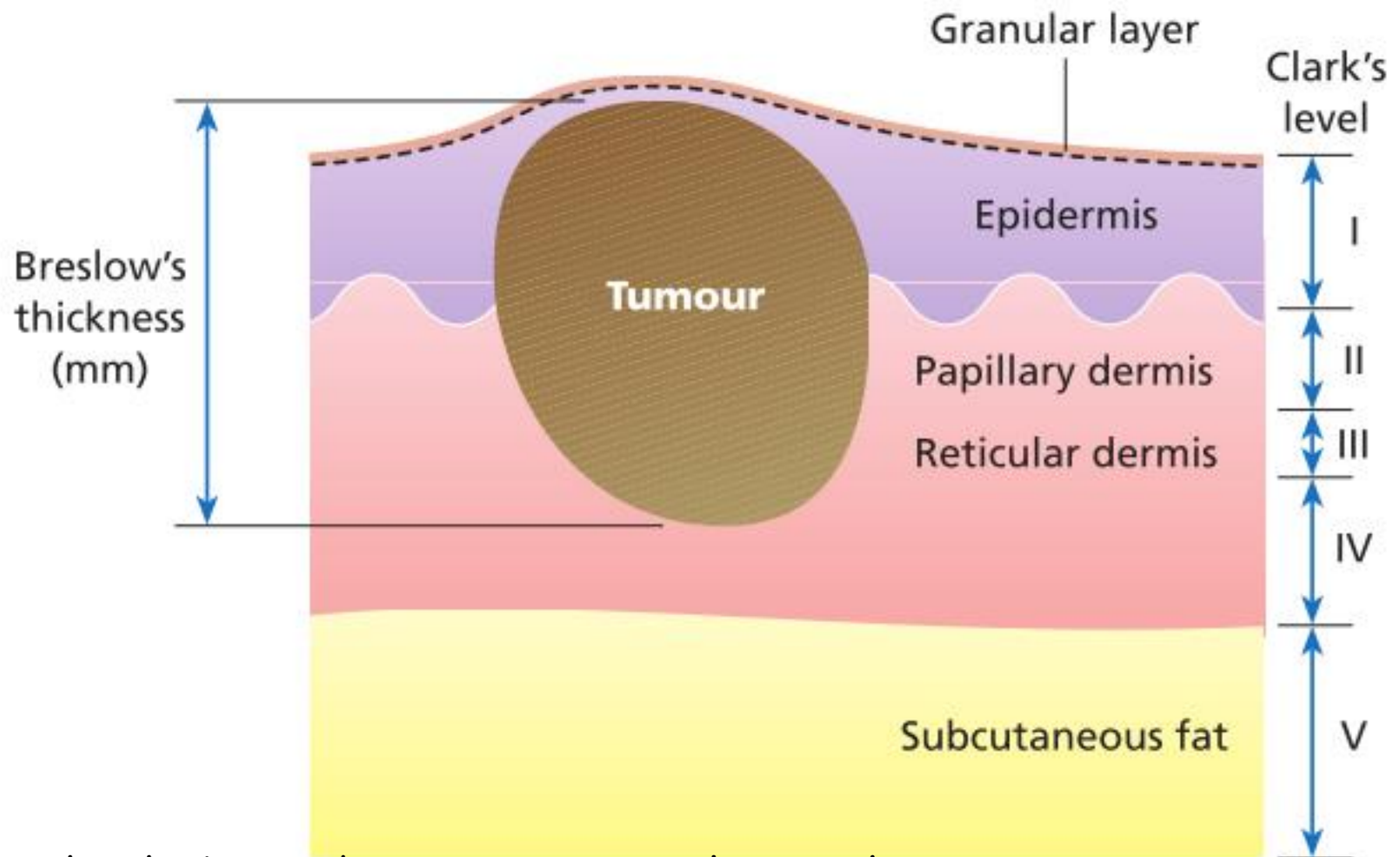
Staging EU and USA:

TNM stage	AJCC stage	Breslow thickness (mm)	5-year survival (%)
I	Ia	Up to 0.75	95
	Ib	0.76–1.5	85
II	IIa	1.51–4.0	65
III	IIb	>4.0	45
	III	Nodal disease	40
IV	IV	Metastatic disease	<10

AJCC, American Joint Committee on Cancer; TNM, tumour, node, metastasis.

the system provide a useful guide to prognosis

Breslow's and Clark's methods of microstaging



the thicker and more penetrating lesion- the worse prognosis

Prognostic indicators in melanoma

Indicator	Significance
Depth of primary tumour	Breslow <0.75 mm, 5-year survival 95% 0.76–1.5 mm, 5-year survival 85% 1.51–4.0 mm, 5-year survival 65% >4.0 mm, 5-year survival 45%
Sex	Females do better than males
Age	Prognosis worsens after 50 years of age, especially in males
Site	The prognosis is poor for tumours on trunk, upper arms, neck and scalp
Ulceration	Signifies a poor prognosis
Sentinel node	Prognosis worsens with tumour-positive sentinel node
Clinical stage	Prognosis worsens with advancing stage

Treatment

surgery - 2-5 mm margin of clearance laterally and down
biopsy do not provoke metastasis

if the histology confirms the diagnosis of melanoma



wider excision including wound of excision biopsy as soon as possible
0,5 cm clearance for melanoma in situ
1 cm clearance for all invasive melanoma
(or **1 cm normal skin** around for **every mm** of tumour thickness
up to 3 cm -the maximum)

Treatment

Sentinel lymph node biopsy (SLNB)

the first and nearest lymph node in the lymphatic drainage of the tumour

it is detected with radiolabelled colloid injected around tumour

often for tumours thicker than 1 mm, ulcerated with numerous mitoses, signs of regression

may provide a
false sense of
security

Treatment

Adjunctive therapies

melanoma specific antigen vaccines

INF-alpha controversial

vermurafenib (for BRAF positive melanoma)

Treatment

Chemotherapy

may be palliative in 25% of patients

decarbazine is often drug of choice

Follow up (screening for recurrence and metastases)

first two years 3-4x a year

after 1-2x a year for whole life

regional nodes must be palpated

chest X-rays 1x a year

ultrasound of liver and lymph nodes

complete blood examination with

AST, ALT, GGT, LDH, S100

PET/CT and CT in invasive tumours

Follow up (screening for recurrence and metastases)

80% of recurrent and/or metastatic melanomas occur within 3 years of definitive surgery

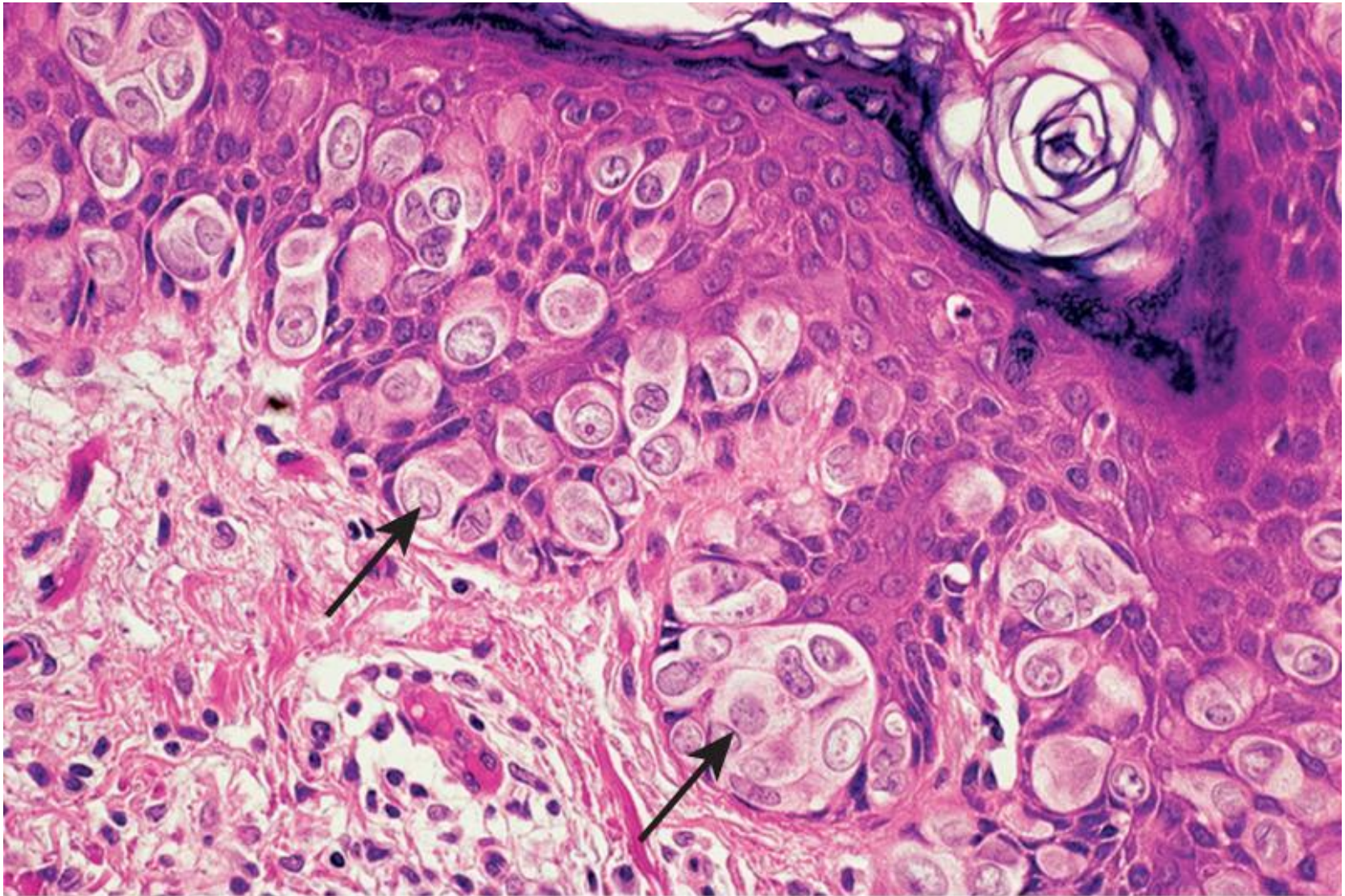
5-8% metastatic melanomas are discovered after 5 years



Paget's disease



a well-defined red scaly plaque around nipple

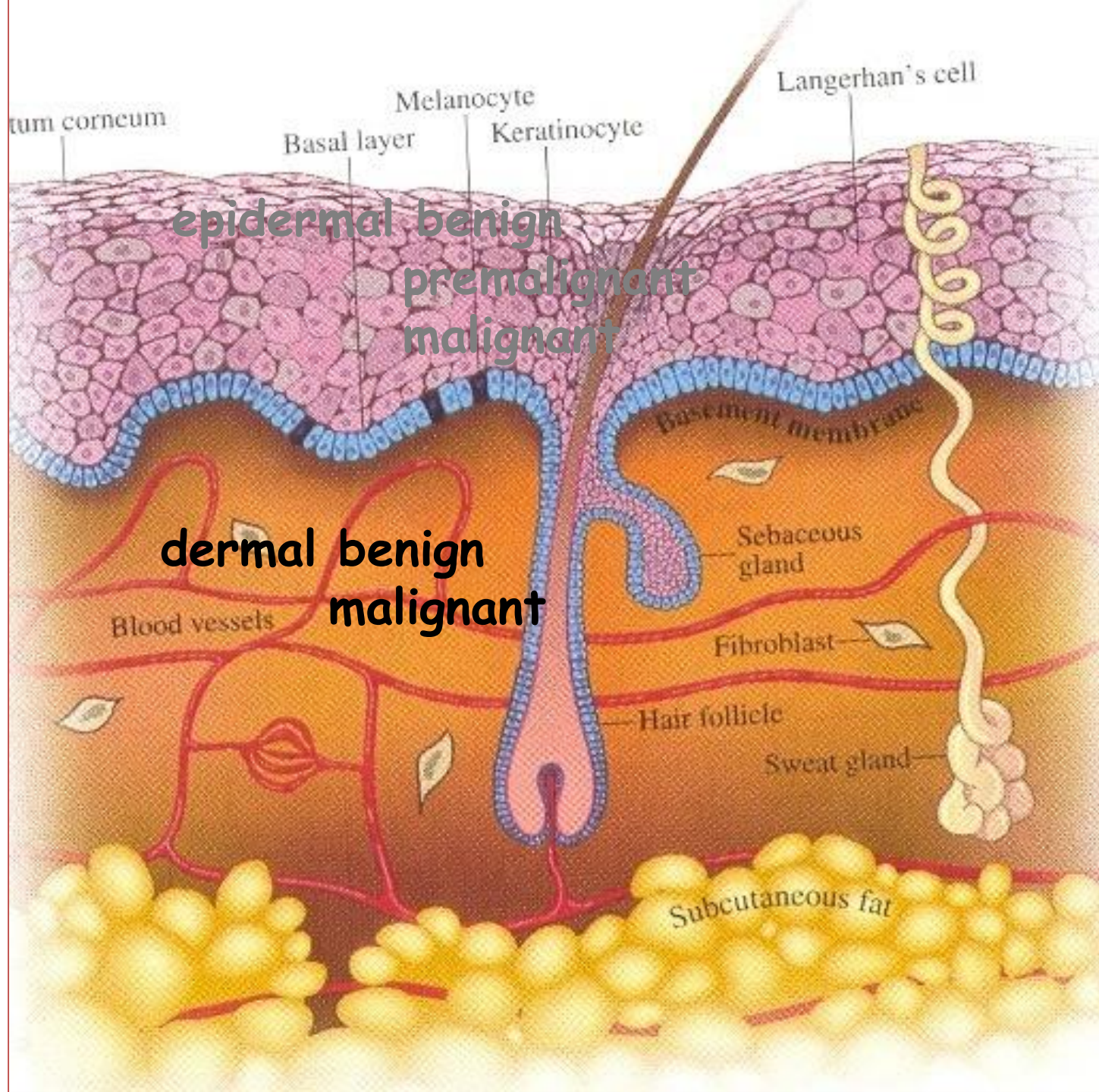


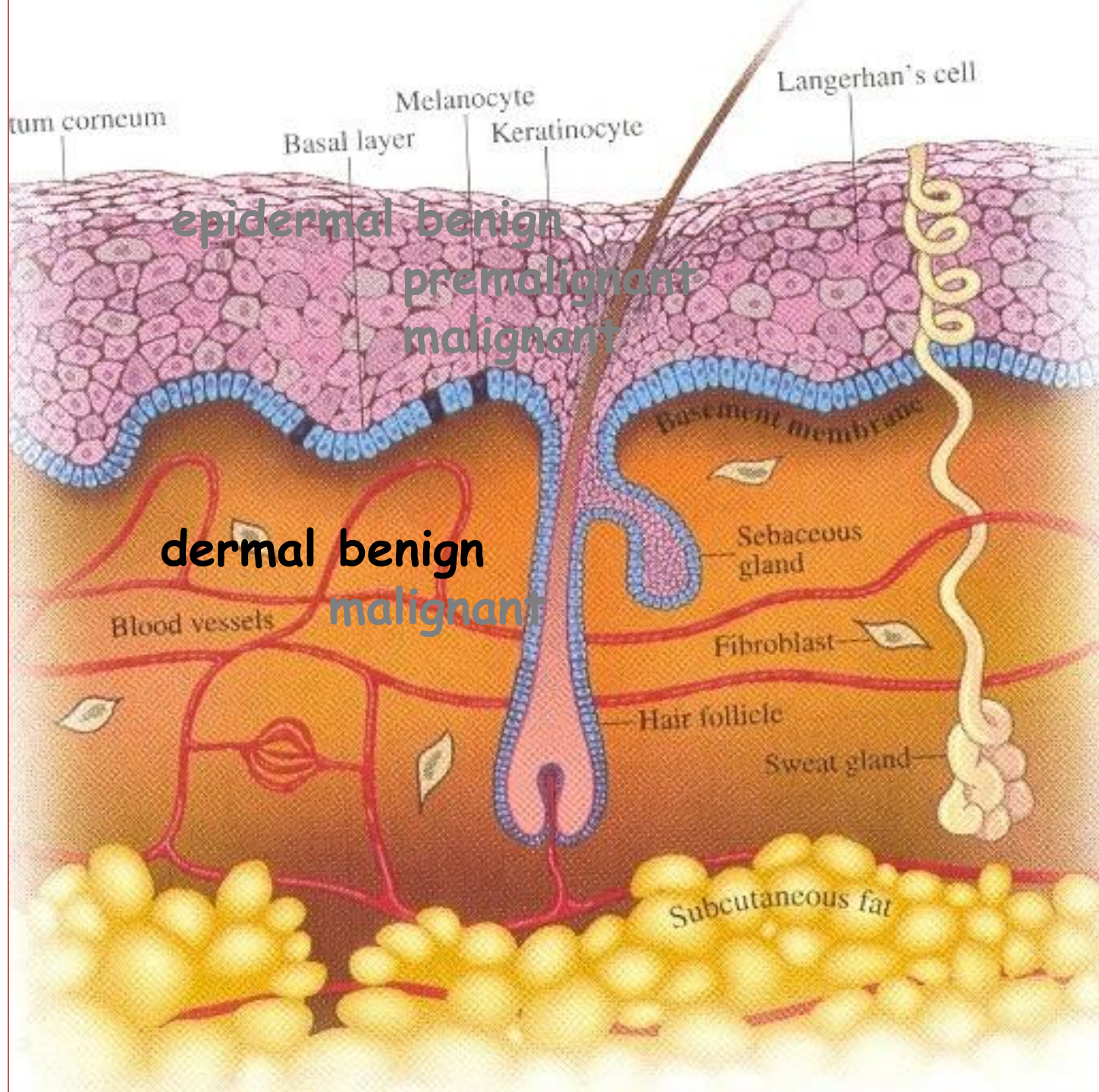
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invasion of the epidermis by cells from the underlying intraductal breast carcinoma (Paget cells)

Treatment

Surgery





vascular lesion

**other benign dermal tumours
(dermatofibroma, keloid, lipomas)**

vascular lesion

other benign dermal tumours
(dermatofibroma, keloid, lipomas)

Developmental abnormalities of blood vessels

present at the birth or appear soon after

Malformations

Present at birth. Do not involute ('salmon' patch is exception)

- 1 Capillary ('salmon' patch and 'port-wine' stain)
- 2 Arterial
- 3 Venous
- 4 Combined

Haemangiomas sometimes called angiomatous naevi)

Usually appear after birth. More common in females, 50–60% on head and neck. Involute by 5–9 years after initial proliferation

- 1 Superficial (capillary)
- 2 Deep (cavernous)
- 3 Mixed

Developmental abnormalities of blood vessels

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"Salmon" patches or "stork bites"



composed of
capillary network
in superficial
dermis

in 50% of babies

nuchal region may remain unchanged



"salmon" patch



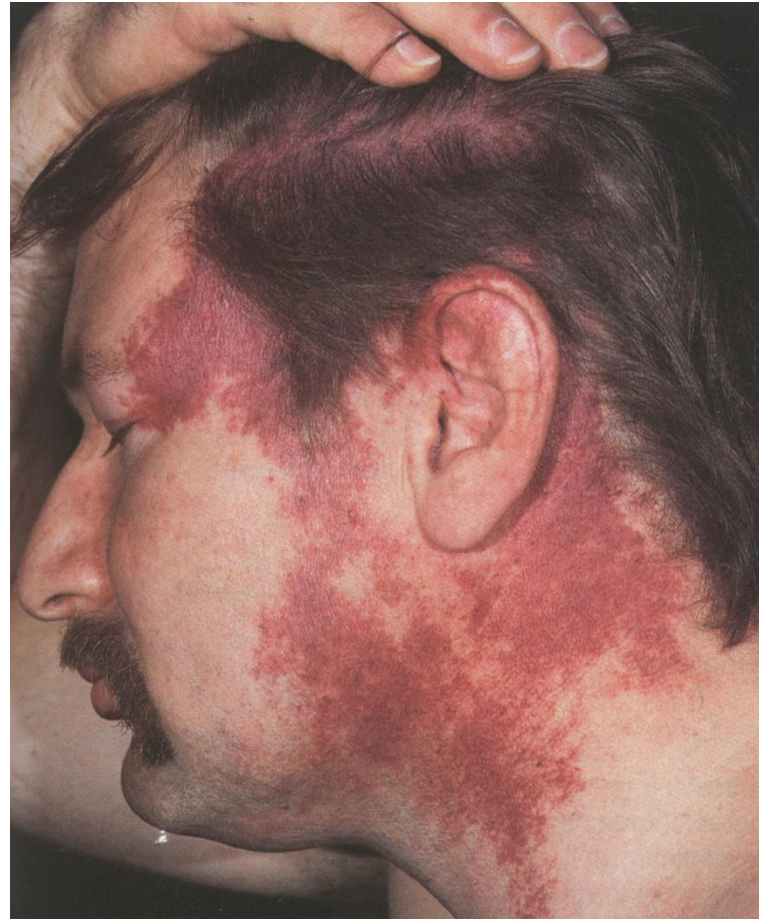
patches from other areas usually disappear within a year

"Port-wine" stains (naevus flammeus)

present at the birth

dilated dermal capillaries
persist
in the middle age - angi-
omatous nodules

0,1-0,3%
of infants





be careful of possible
vascular malformations
of leptomeninges of the
trigeminal area

(epilepsy, hemiparesis,
glucoma)

Treatment:

laser (pulsed dye laser) 40-50 pulses per session/monthly
cosmetic camouflage

Combined vascular malformations of the limb



large port-wine stain
overgrowth of all soft
tissues with or without
bony hypertrophy of that limb
(Klippel-Trenaunay sy.)

Developmental abnormalities of blood vessels

present at the birth or appear soon after

Malformations

Present at birth. Do not involute ('salmon' patch is exception)

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- 3 Mixed

Haemangiomas

superficial (capillary)



1-3% of infants

Haemangiomas

deep (cavernous) (strawberry naevus)



within few weeks of birth



spontaneous regression
with whitens centrally and
complete regression by the age of 5-9 years

Giant lesions consume lot of platelets - **Kasabach-Merritt sy**



Treatment:
systemic corticosteroids in high doses (2-4 mg/kg/day)
tapered to zero after 1 month

Cherry angiomas

middle-aged and elderly



Cherry angioma

Pyogenic granulomas

benign acquired haemangiomas

often in children and young adults

develop on sites of trauma

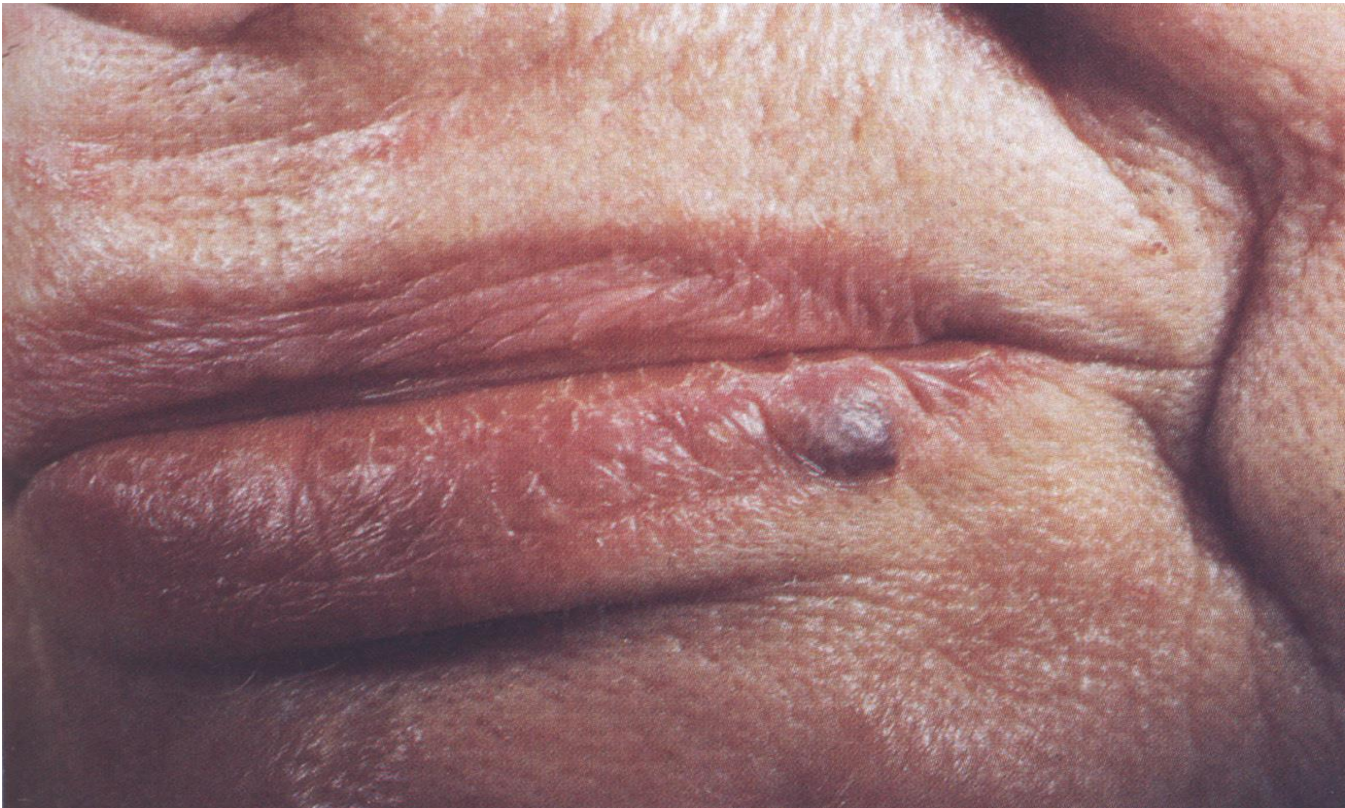
course of
few weeks



differential diagnosis: amelanotic melanoma

Venous lake

venous haemangioma of lower lip in elderly



vascular lesion

**other benign dermal tumours
(dermatofibroma, keloid, lipomas)**

Dermatofibroma



firm, solitary nodule
often on the extremities
they feel larger than they look

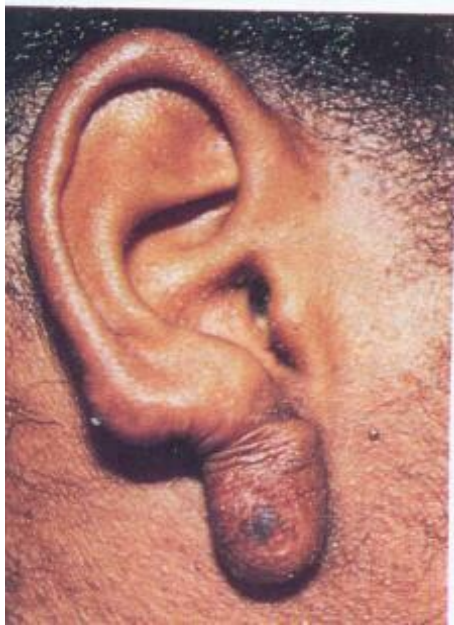
on squeezing dimple appear



Dermatofibroma



Keloid



Treatment:

- silicone sheeting
- CS intralesional
- cryotherapy 20' before CS

overgrowth of dense fibrous tissue

arising in response to trauma, infection, foreign material

inherited tendency for development

Lipomas



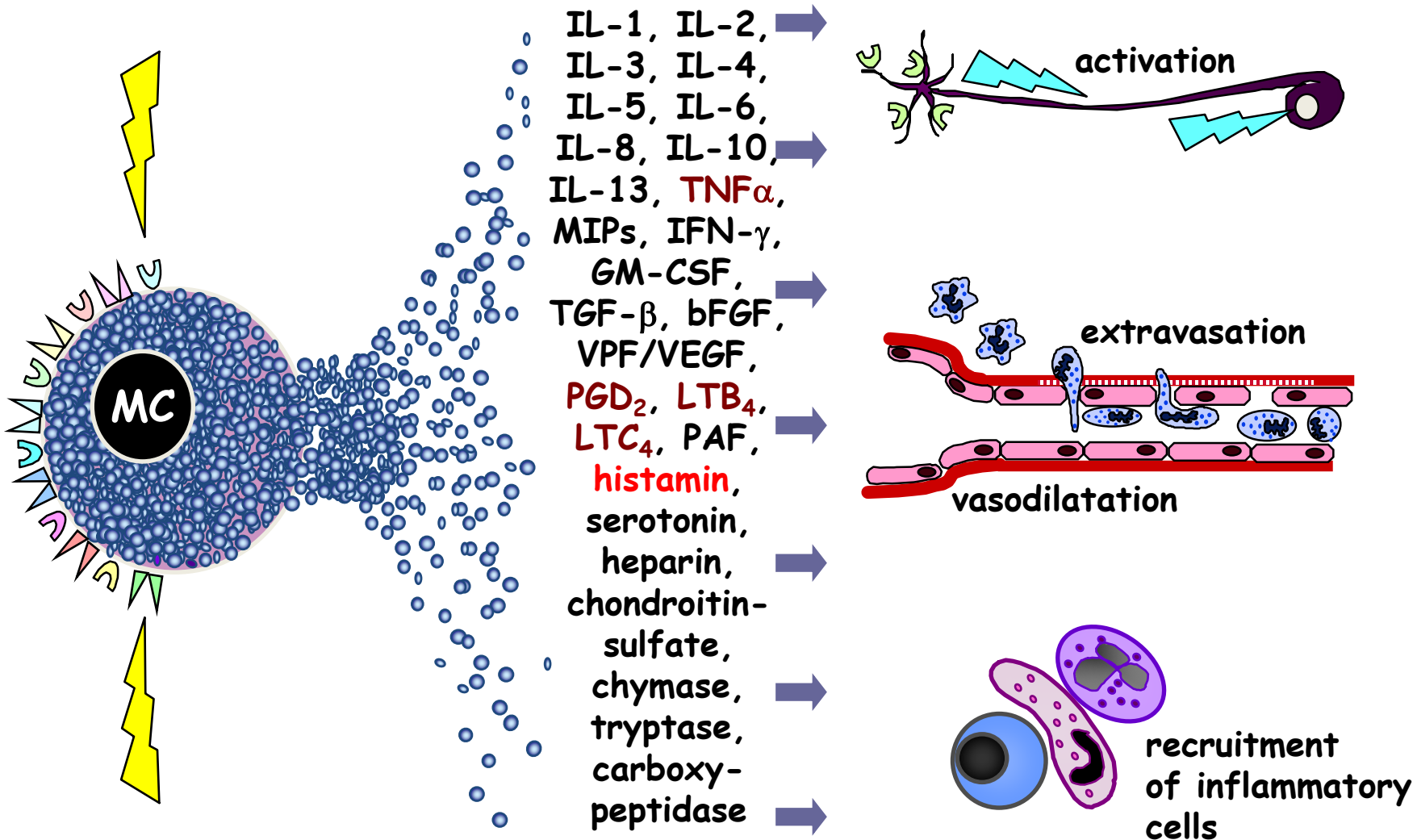
common benign tumours of mature fat cells
soft rubbery consistency
may be one or many
proximal part of limbs

Mastocytosis (urticaria pigmentosa)

various conditions in which the skin and other tissues contains an excess of mast cells



Mastocyte



Positive Darier's sign





Darier's sign in mastocytosis

Main types:

Mastocytoma - solitary pink or brown itchy papule which wheals on rubbing



no systemic involvement

Juvenile mastocytosis - the most common type



no systemic involvement

often mistaken
for multiple naevi

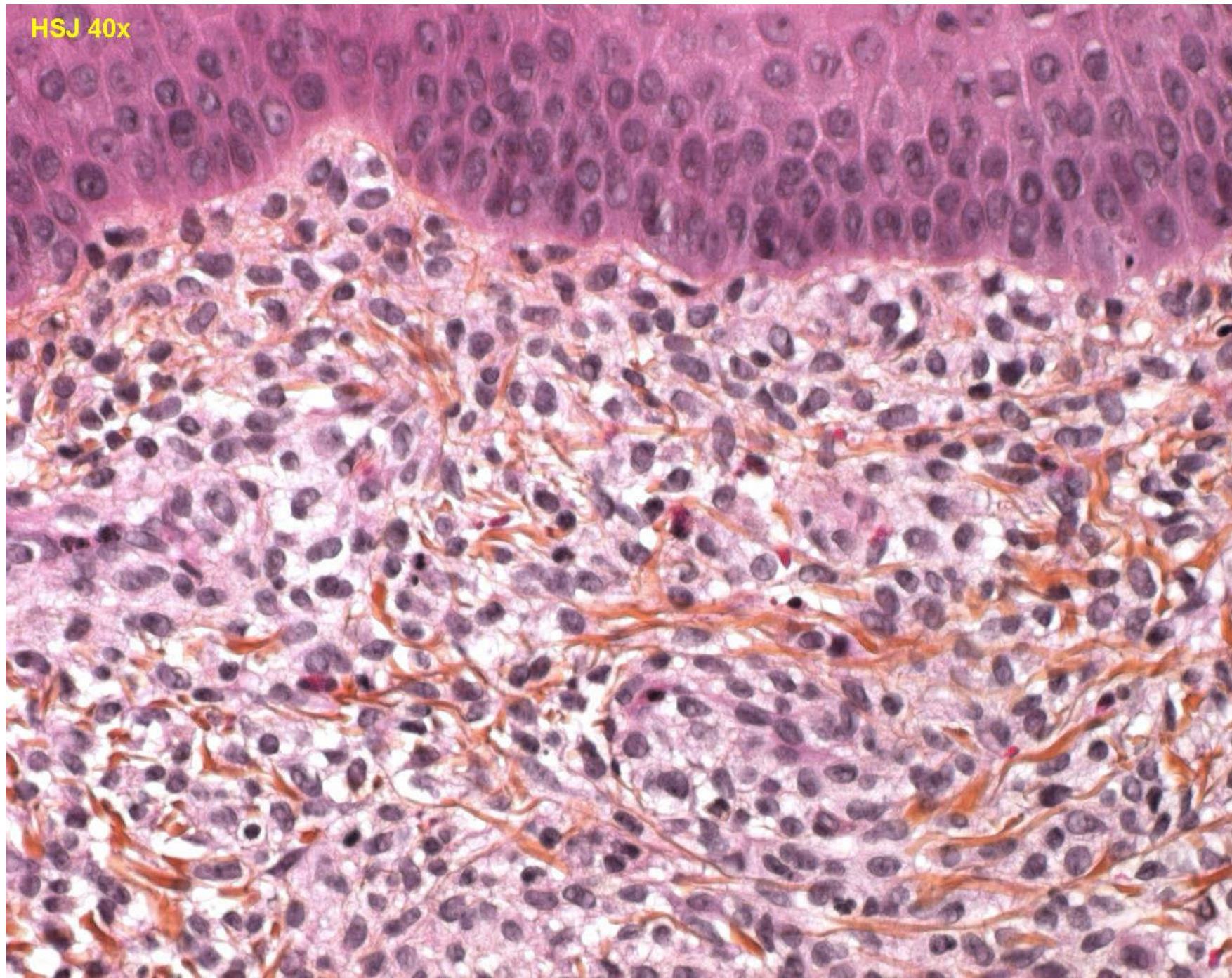
Diffuse cutaneous mastocytosis - rare



persistent wheals that appear after minor friction

the bone marrow, liver and spleen may be involved

HSJ 40x



Diffuse cutaneous mastocytosis - rare



thickened appearance like pigskin

death from massive histamine release is a real risk

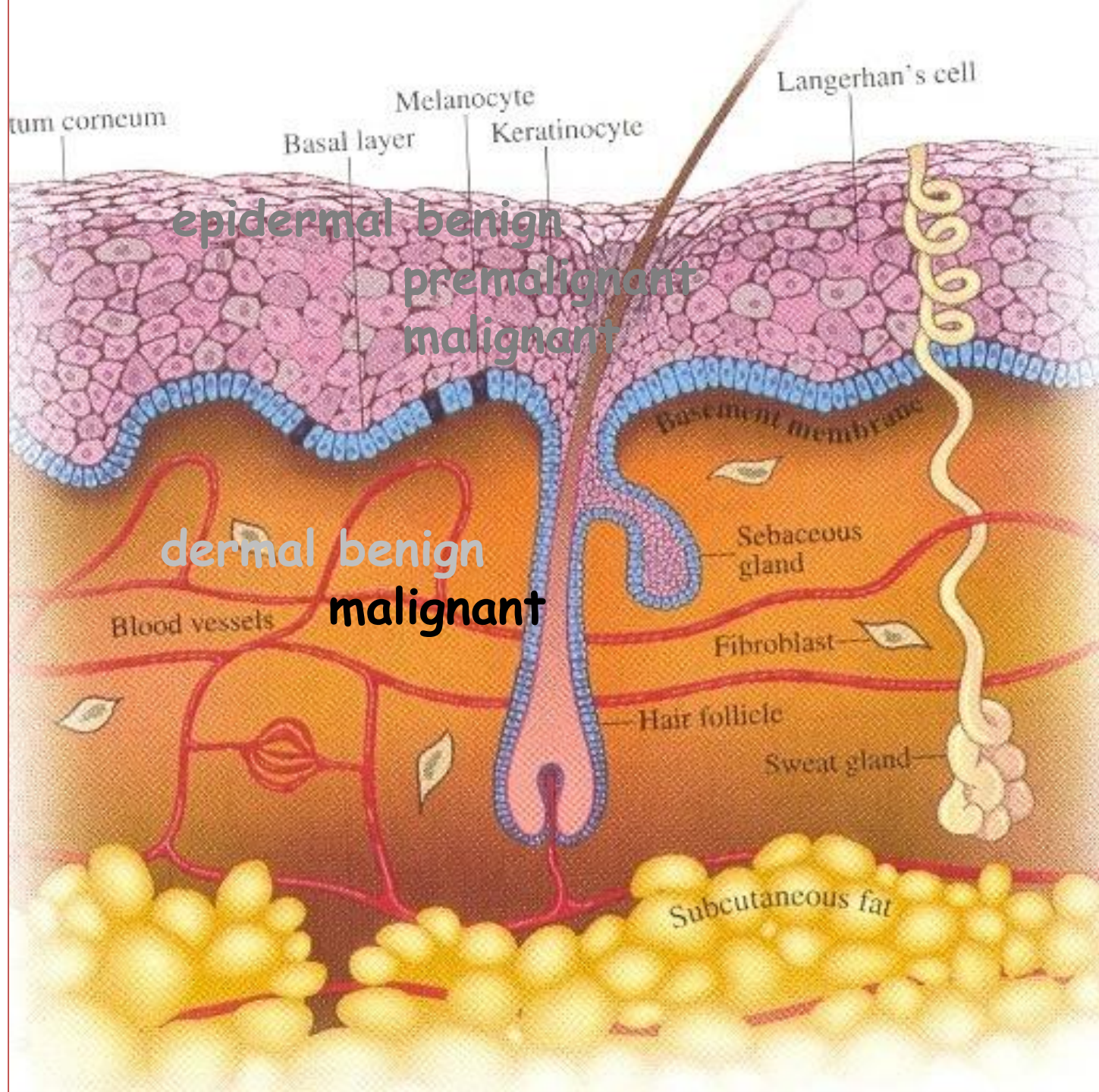
Adult type of mastocytosis - rare



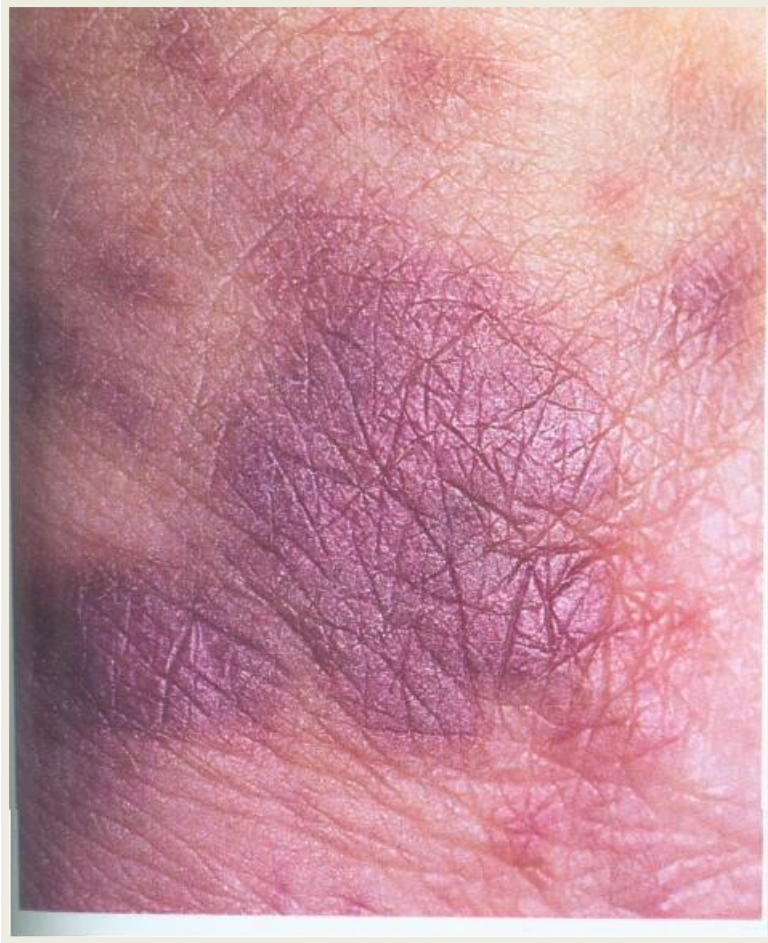
pink-brown teleangiectatic
macules in early adult life

flushing
headaches
palpitations

the bone marrow, liver and spleen may be involved in 20%



Kaposi's sarcoma



malignant tumour of capillaries
(HHV8 has been isolated)

classic type

associated with immunosuppression

Classical Kaposi's sarcoma



- in Africans

- in elderly Yews of European origin

feet, hands, legs (cold parts)

dark blue and purple macules progressing to tumours

slowly progressing

may metastasize to lymph nodes
life expectancy 5-9 years

Treatment:

sensitive to radiotherapy
chemotherapy

Kaposi's sarcoma and immunosuppression



smaller and bruise-like lesions
in tension lines evolve into nodules

associated with AIDS (HIV-1)

appear anywhere
the most often upper trunk,
head and neck

poor prognosis in HIV+ patients

life expectancy around 1 year



Treatment:

treatment of HIV infection
with antiretroviral therapy

Lymphomas and leukaemias

Skin lymphomas

Kiels's classification

Mb Hodgkin


Non-Hodgkin's lymphoma

- low malignancy

lymphocytic, immunocytic, centrocytic, centroblastic-centrocytic

- high malignancy

centroblastic, lymphoblastic lymphoma

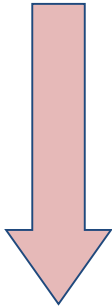


**Mycosis fungoides,
sy. Sezary**

Mycosis fungoides - Cutaneous T-cell lymphoma

T-helper lymphocytes (CD4+)
evolves slowly

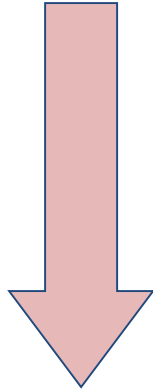
starts in skin



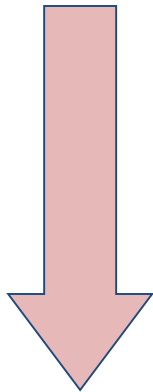
affects lymph nodes and other tissues

the patch stage

months...
years...

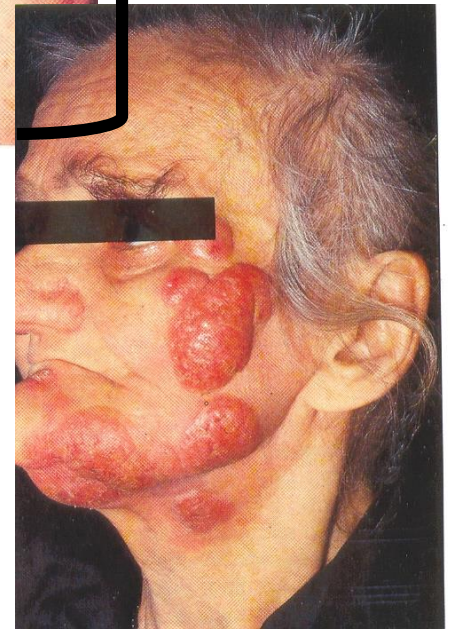
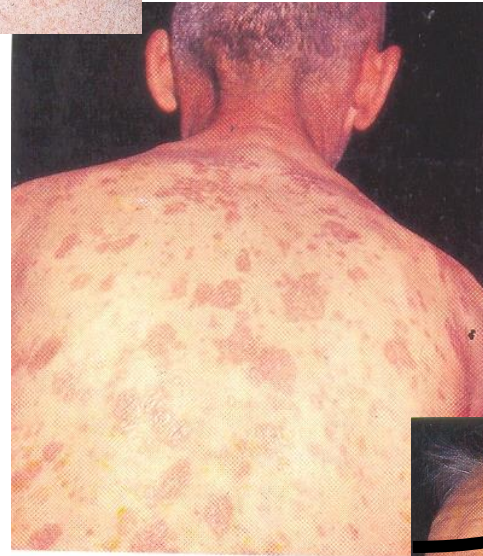


plaque stage



tumor stage

short phase, death usually within 3 years



up to 20
years

Variants:

subcutaneous panniculitis-like T-cell lymphoma
(similar to ulcerated LE panniculitis profundus)

anaplastic large cell CD30+ lymphomas

granulomatous slack skin
(young patients, indurated plaques become atrophic)

pagetoid reticulosis
(on acral parts of young patients, slow growing plaque)

folliculotropic mycosis fungoides
(itchy plaque with follicular prominence followed by alopecia)

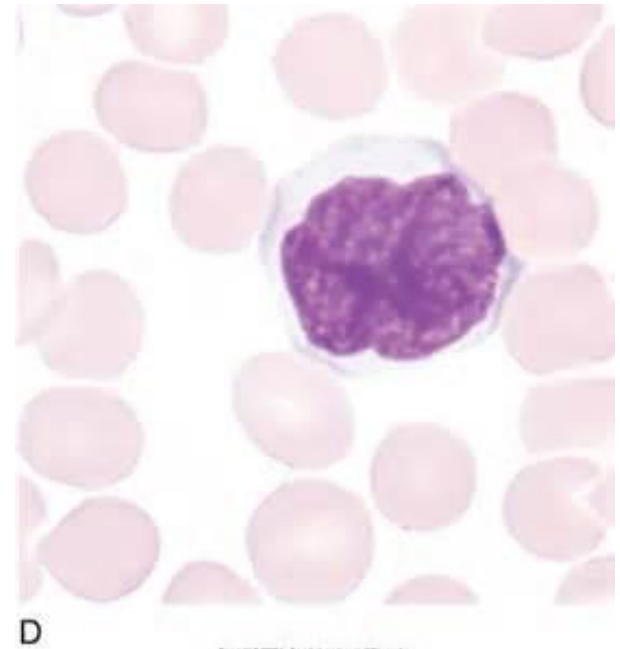
Variants:

Sezary syndrome:

generalized erythroderma

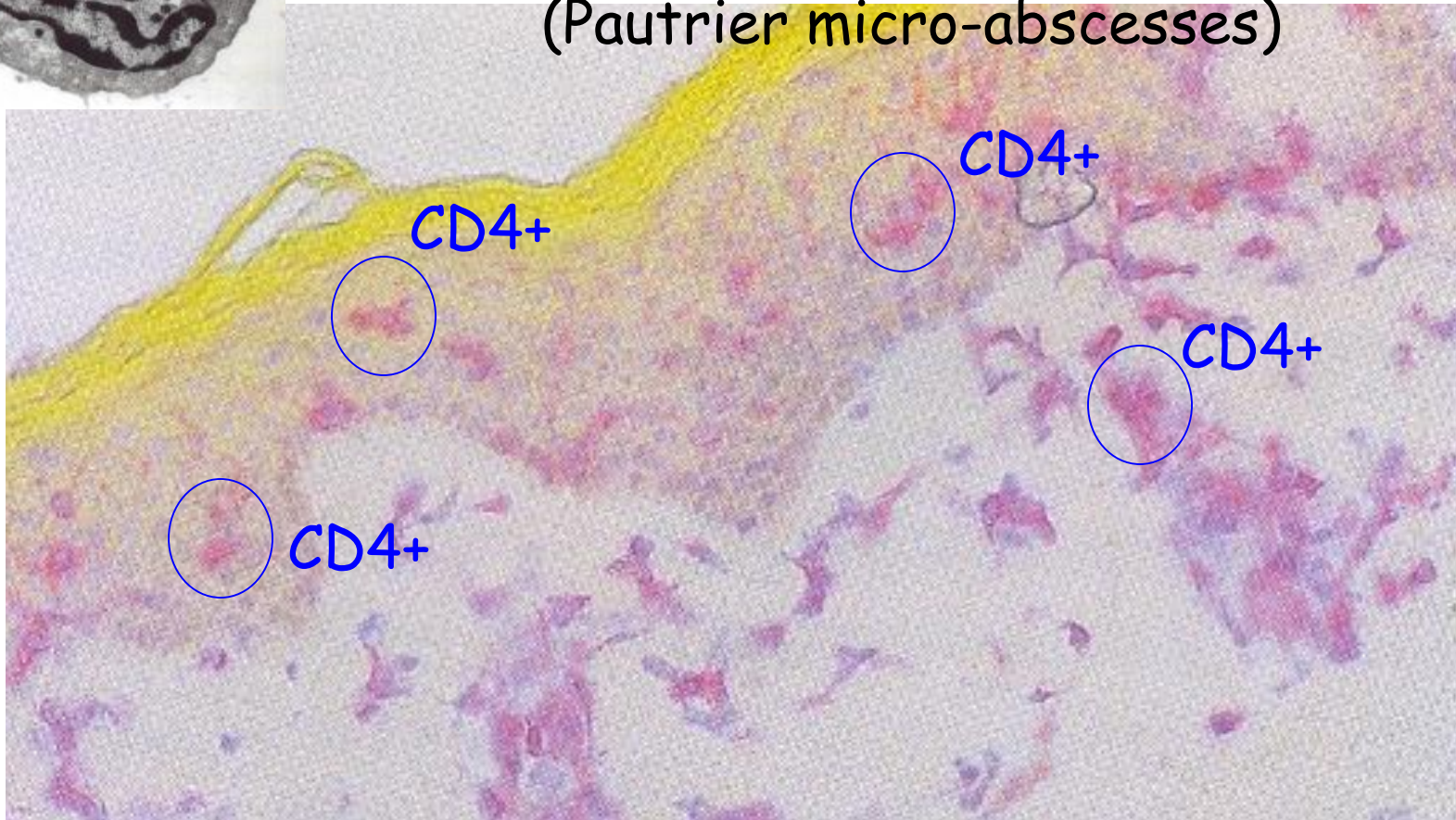
lymphadenopathy

abnormal T lymphocytes with convoluted nuclei in the blood





intraepidermal lymphocytic microabscesses
(Pautrier micro-abscesses)

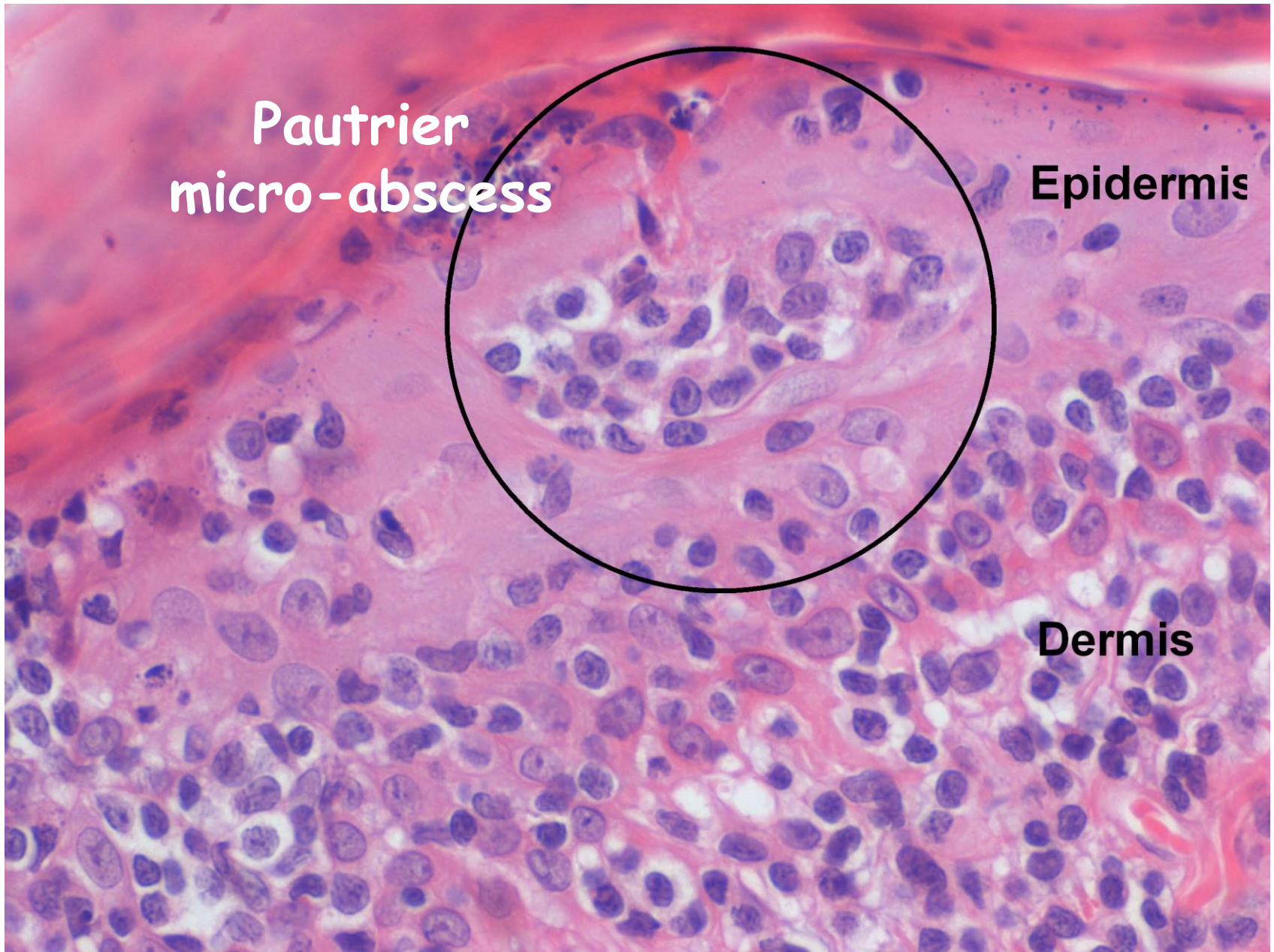


Mycosis fungoides- immunohistochemical staining

**Pautrier
micro-abscess**

Epidermis

Dermis



The End

