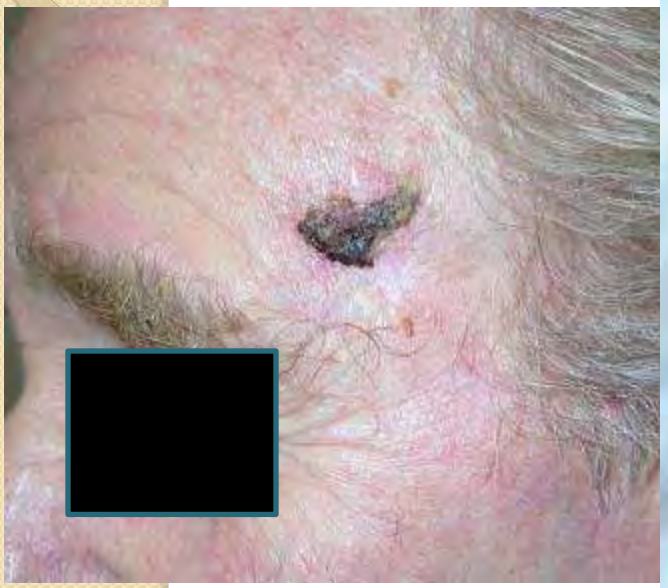
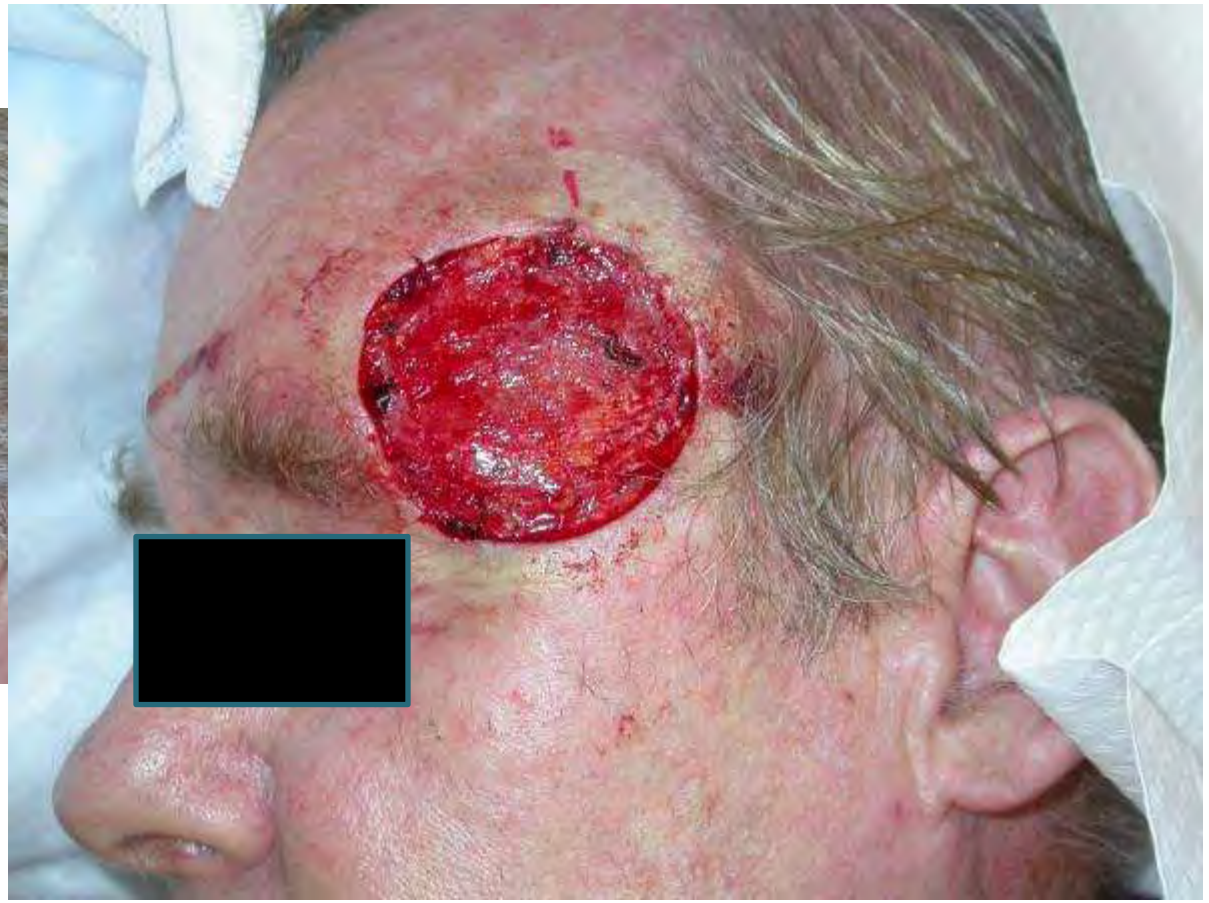
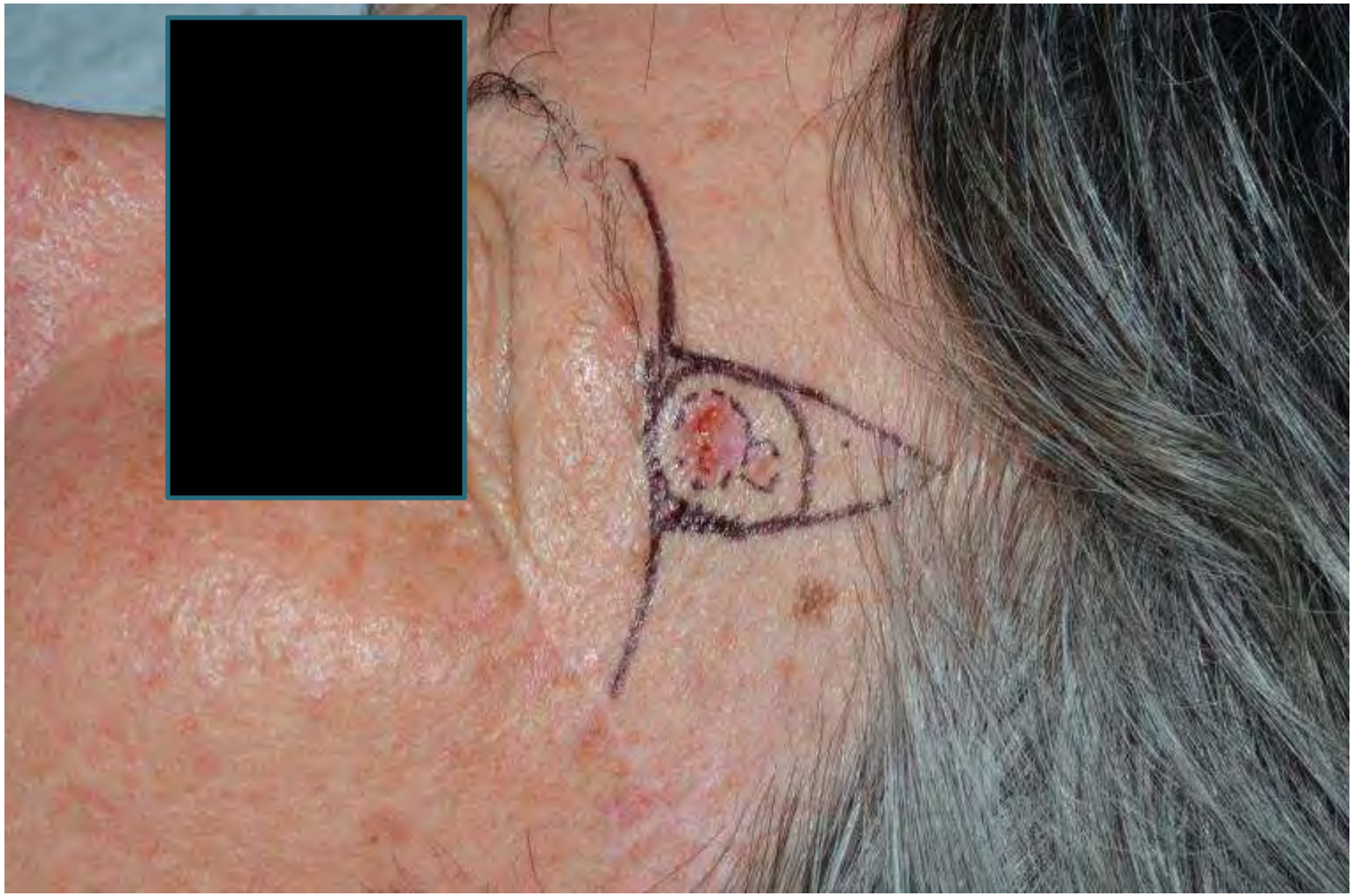
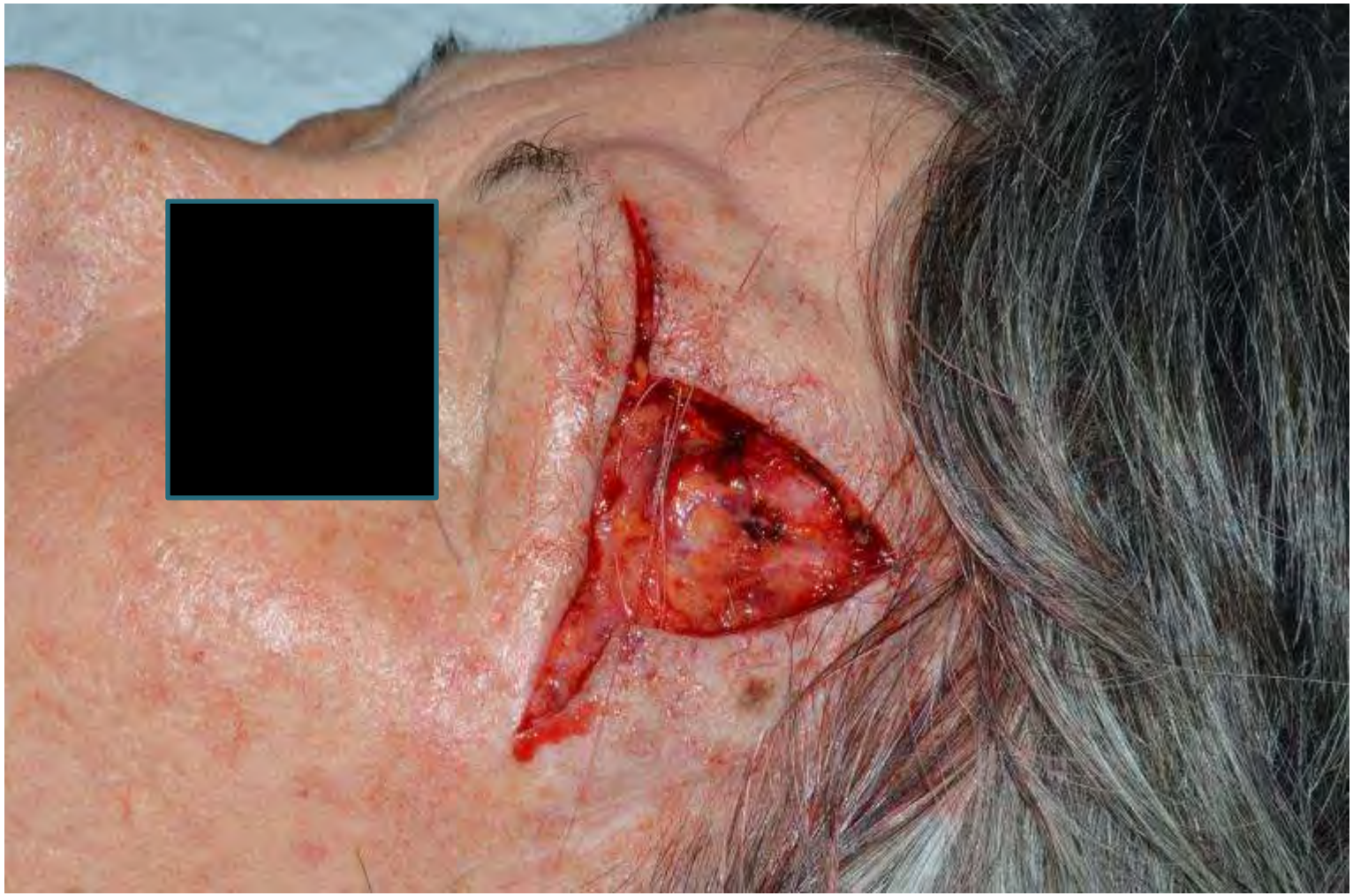
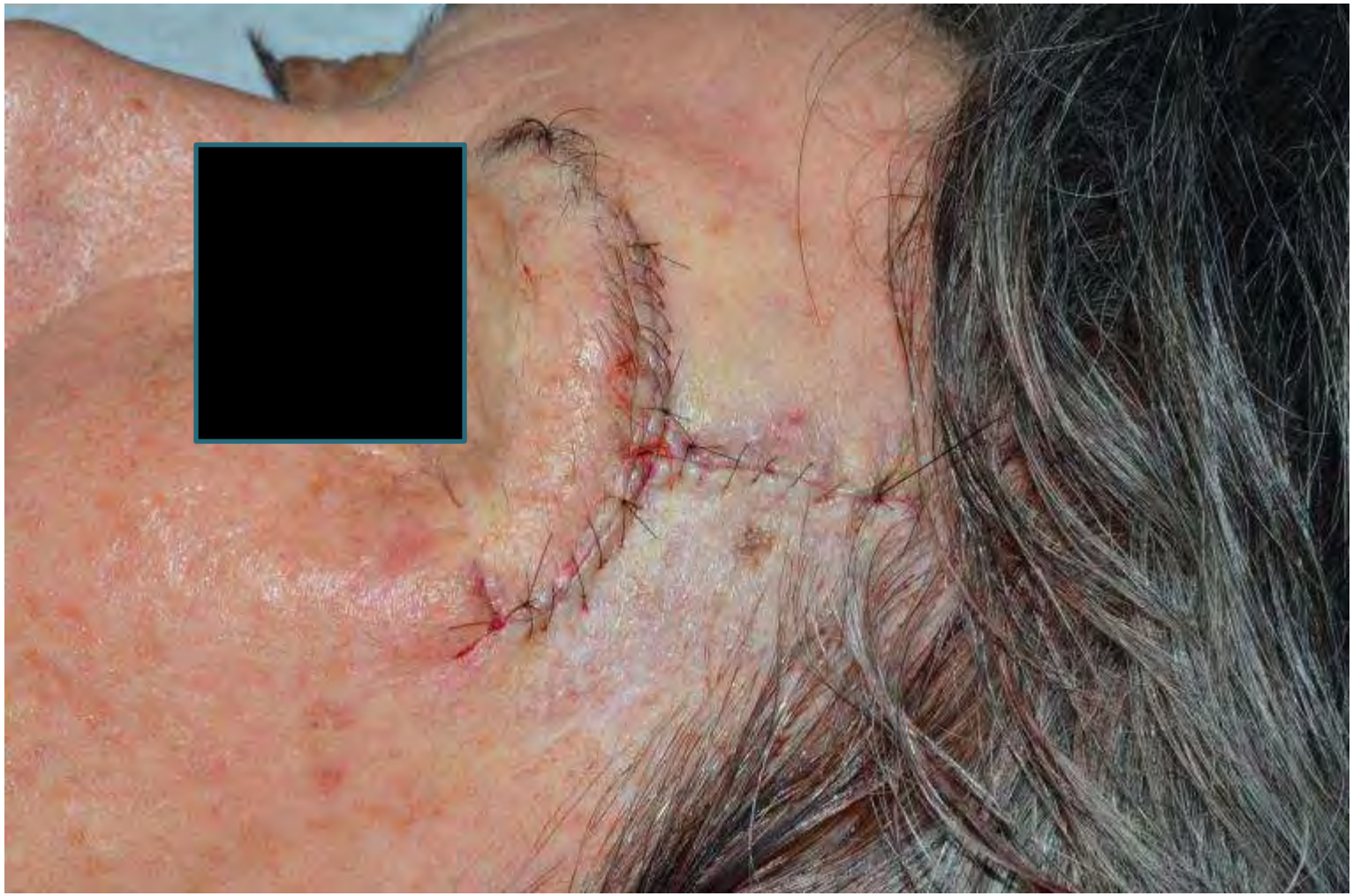




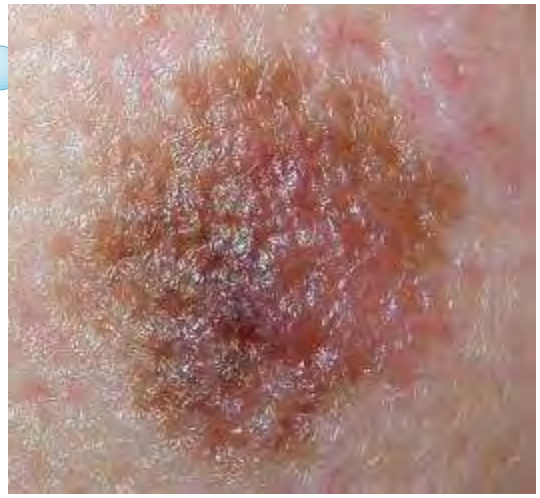


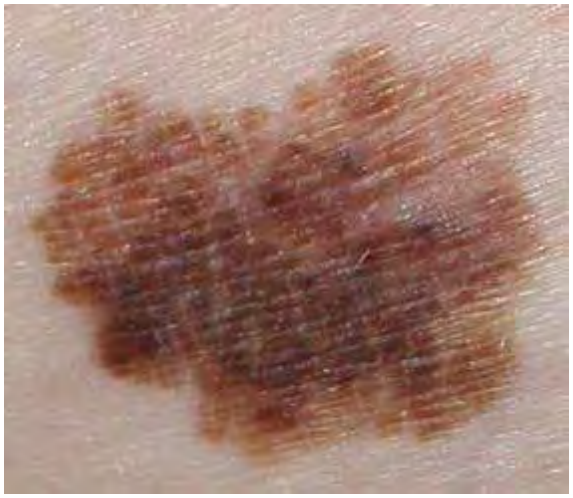


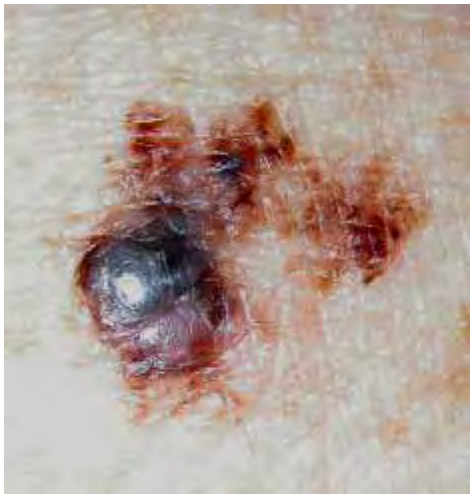


Melanoma

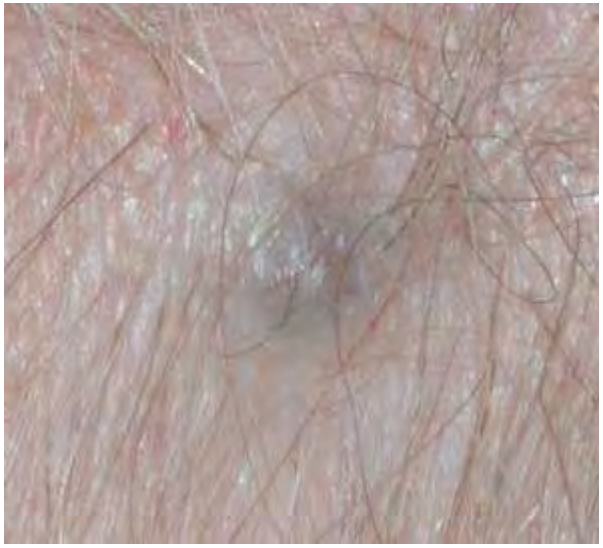
Common Skin Cancers

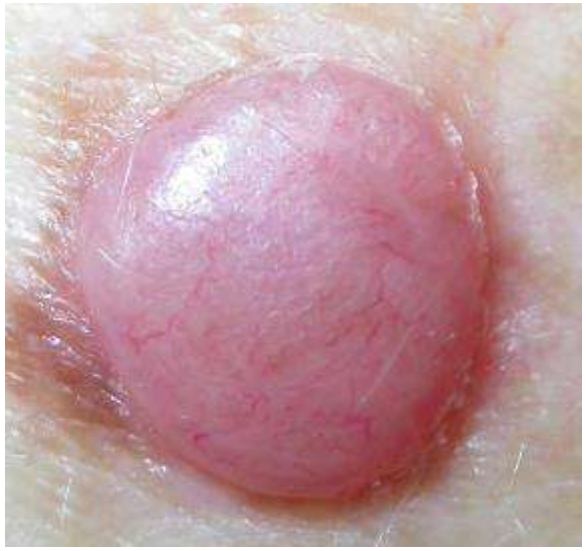






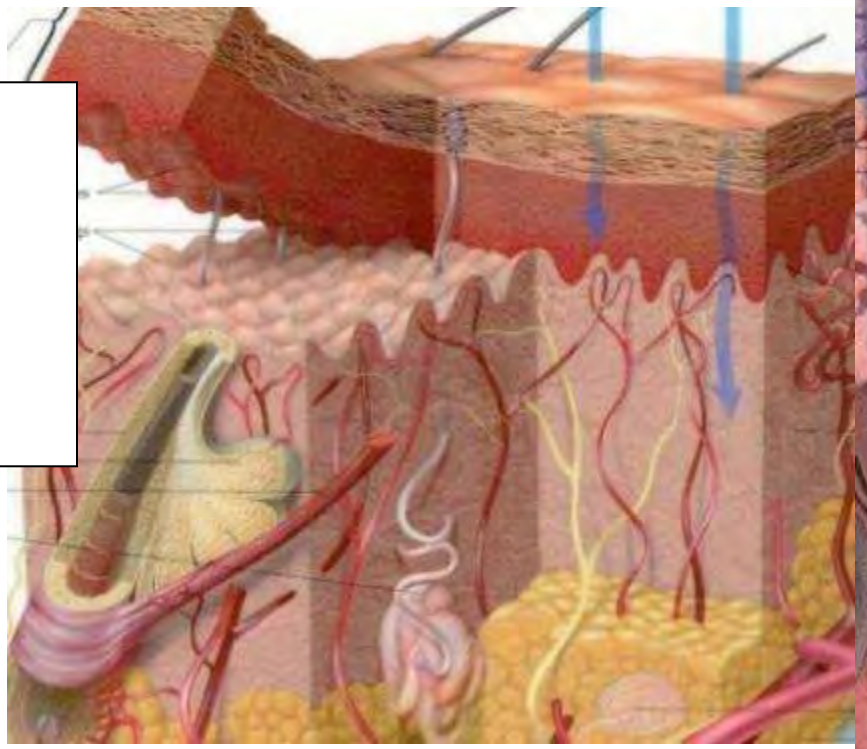
Melanoma
- Insitu
- Invasive
- Metastatic

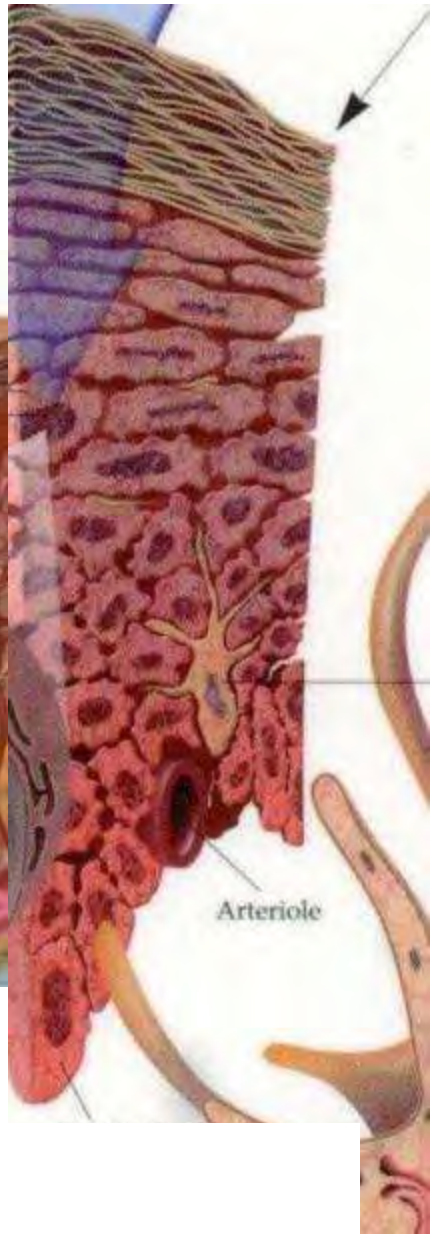


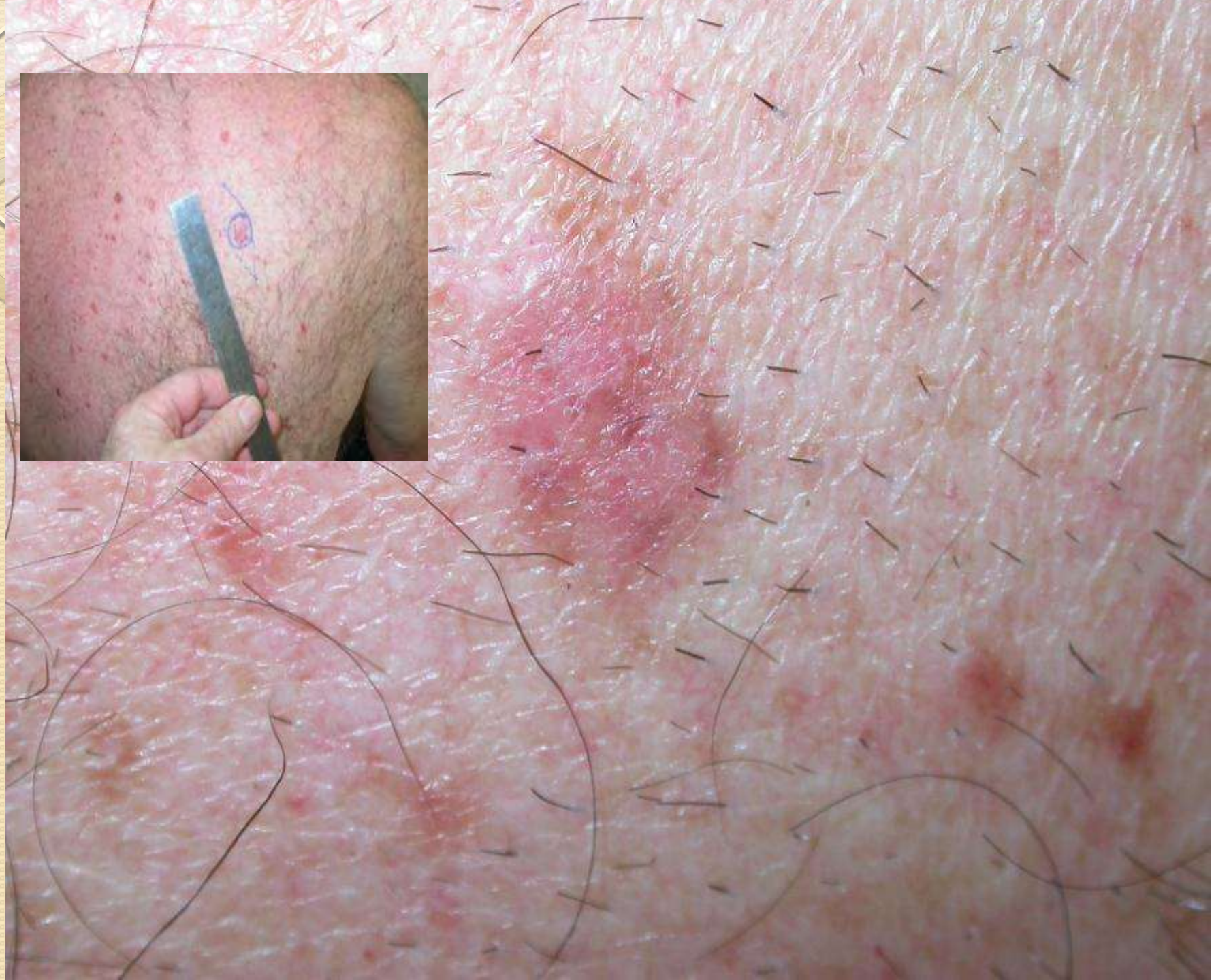


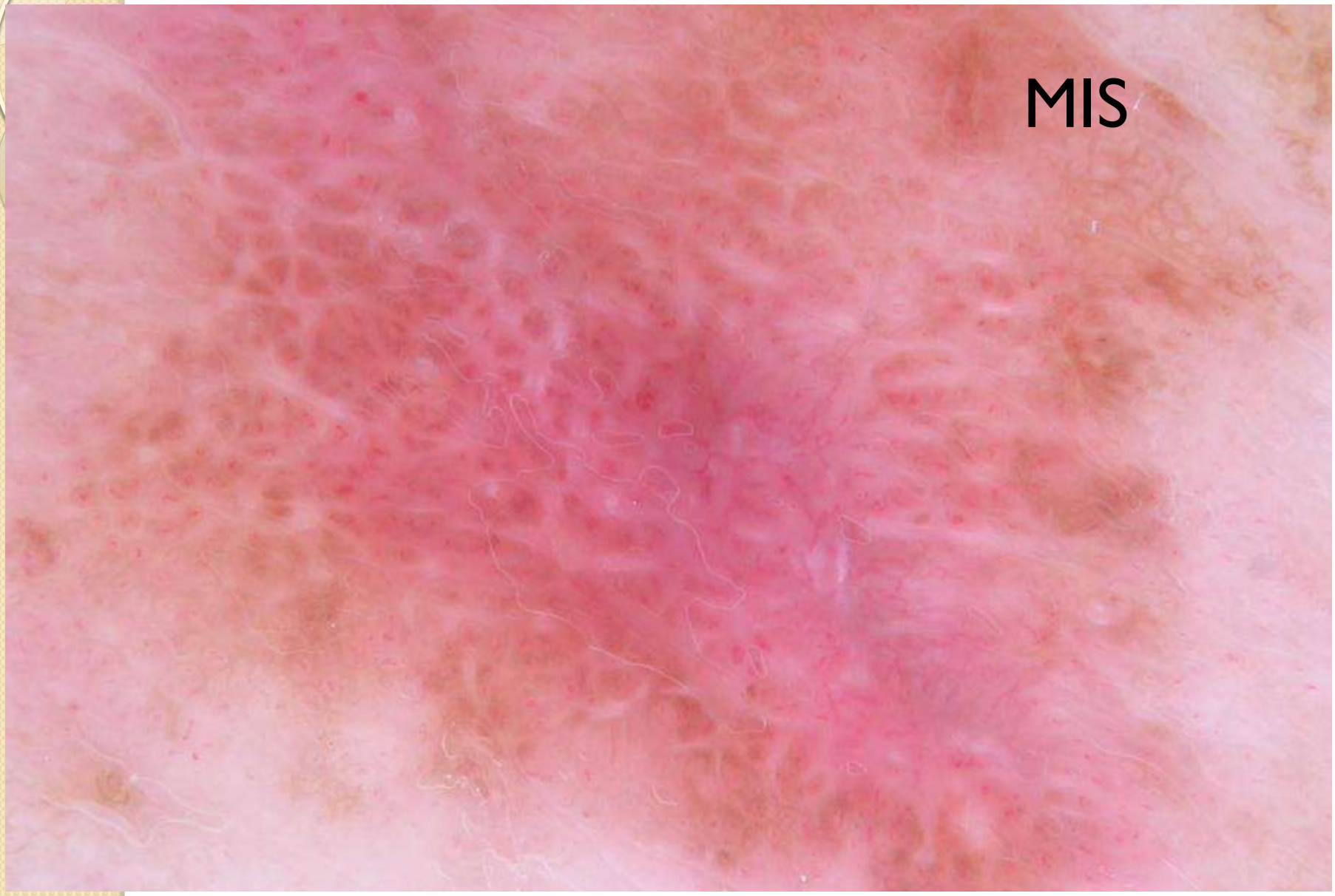
Epidermal – insitu

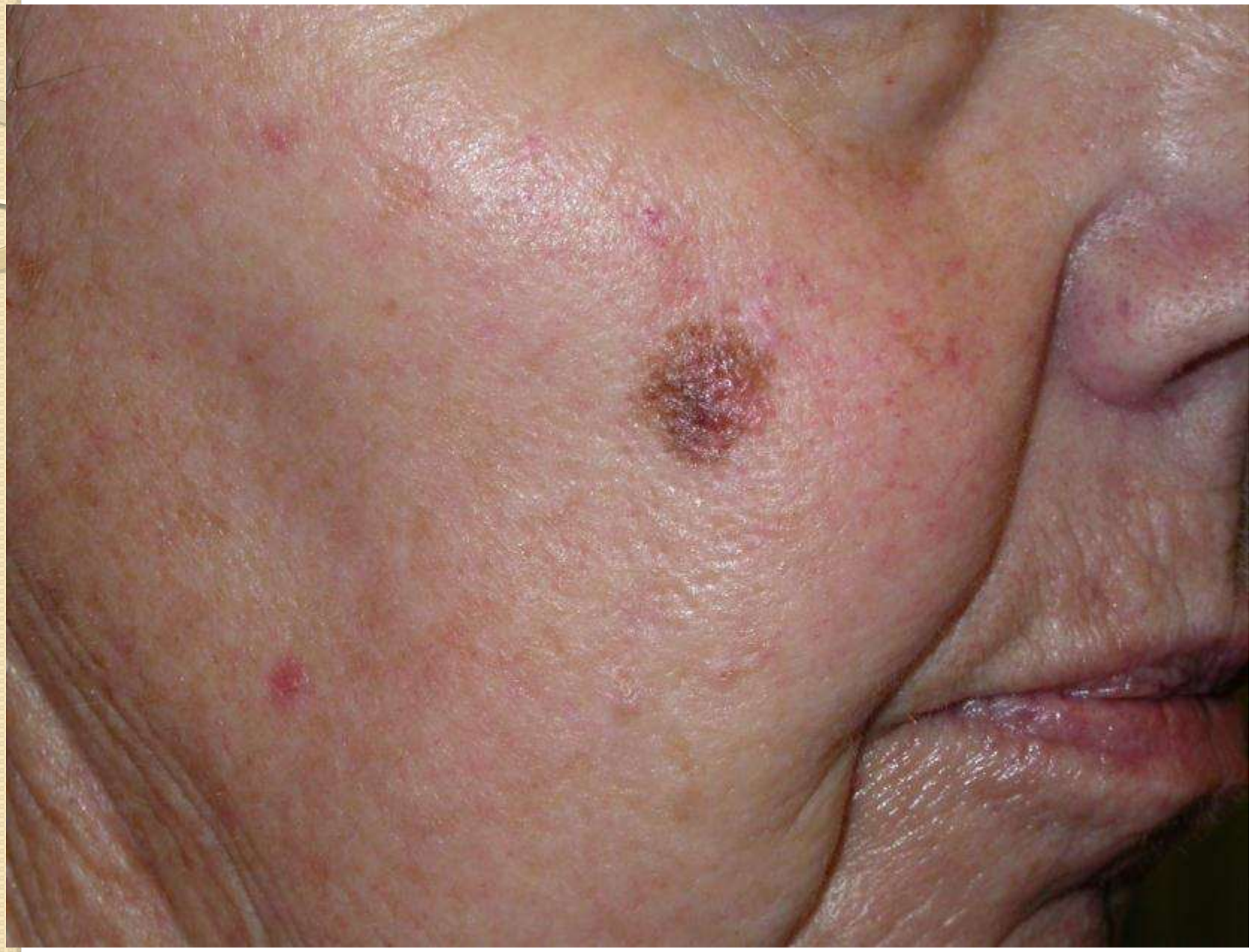
Dermis - invasive





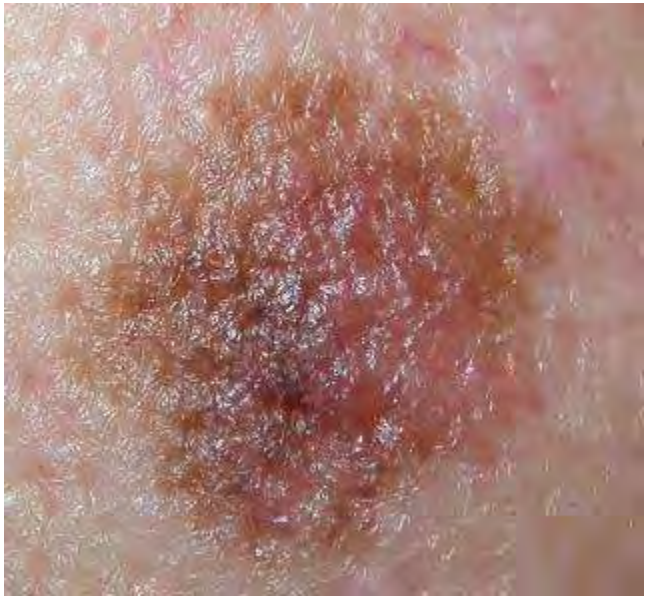


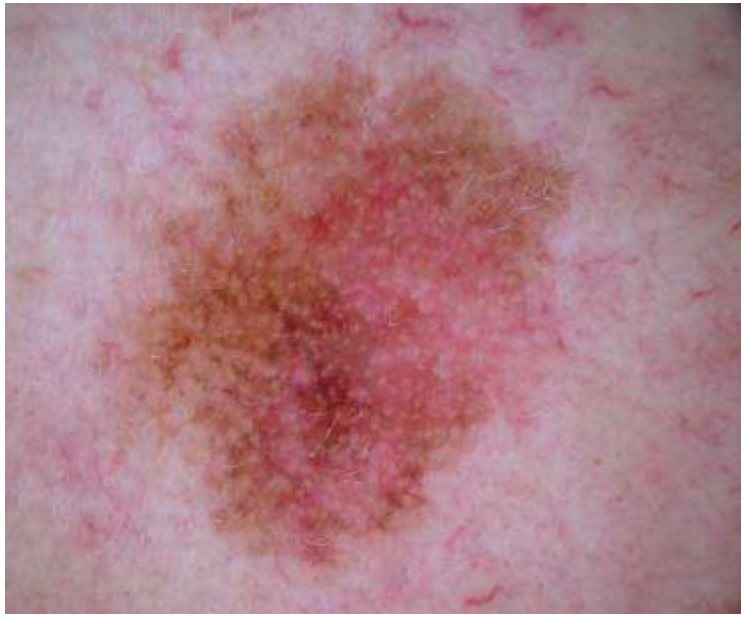

MIS

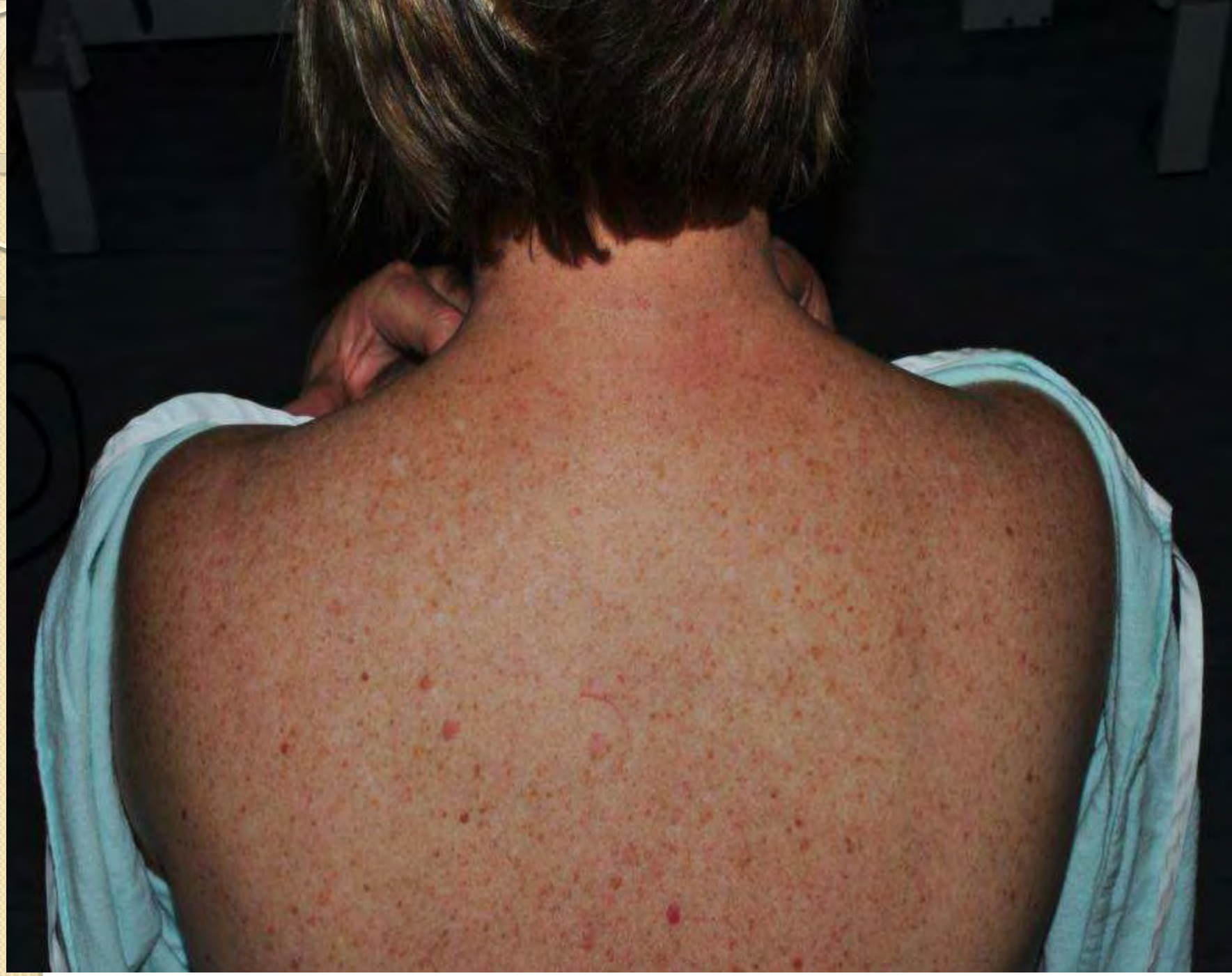


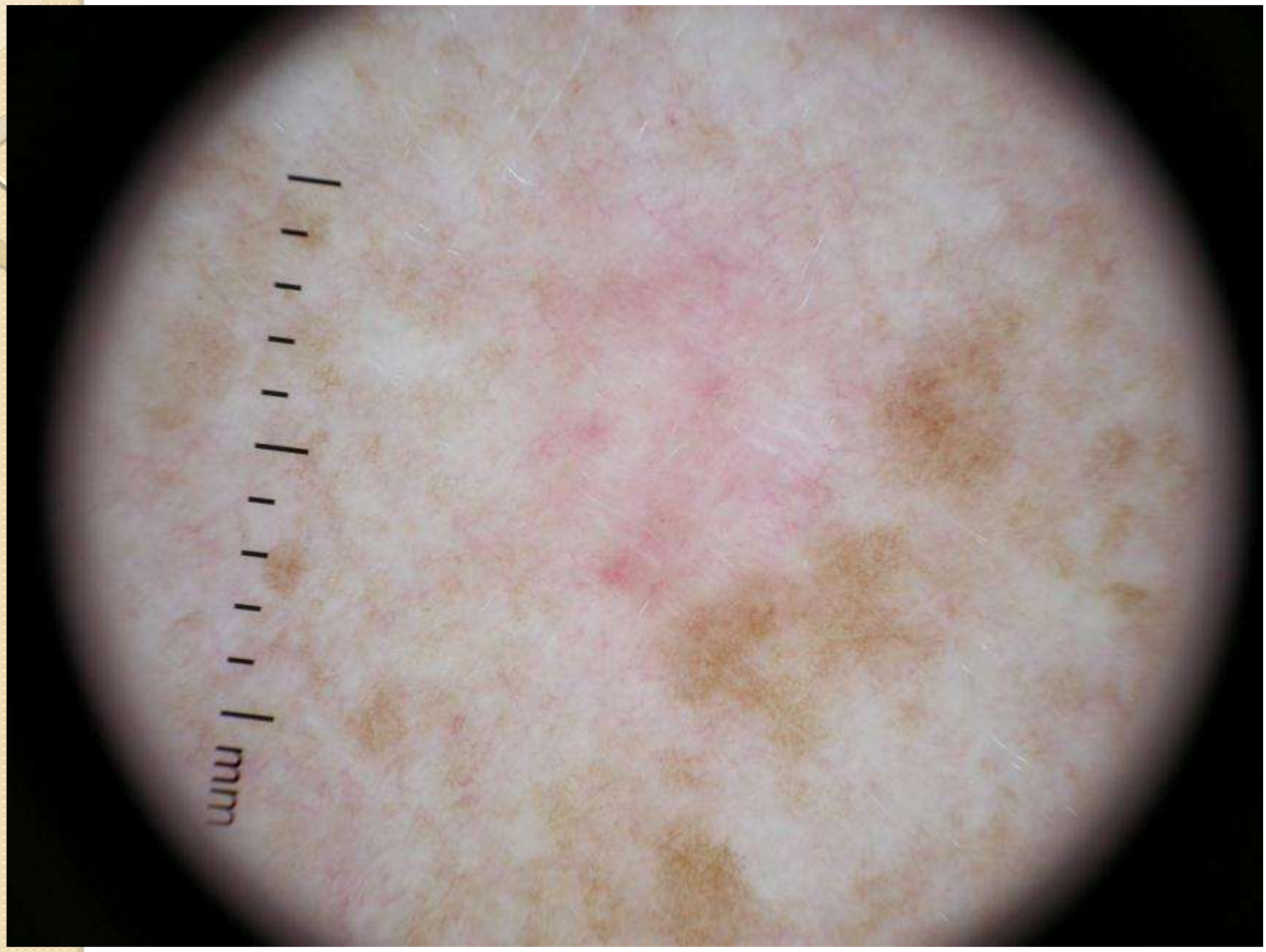
The Skin Clinic Marlborough

Melanoma Insitu

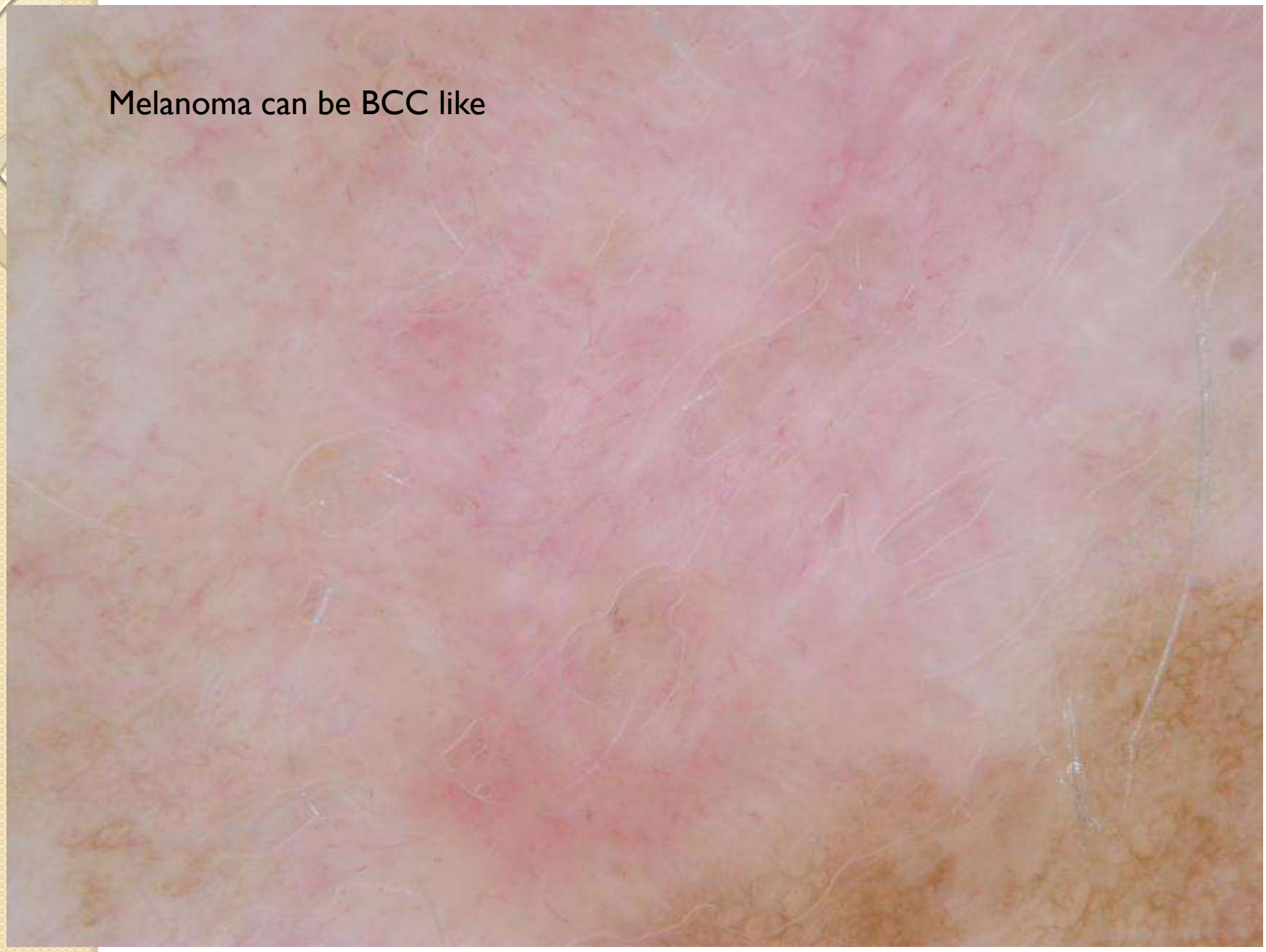


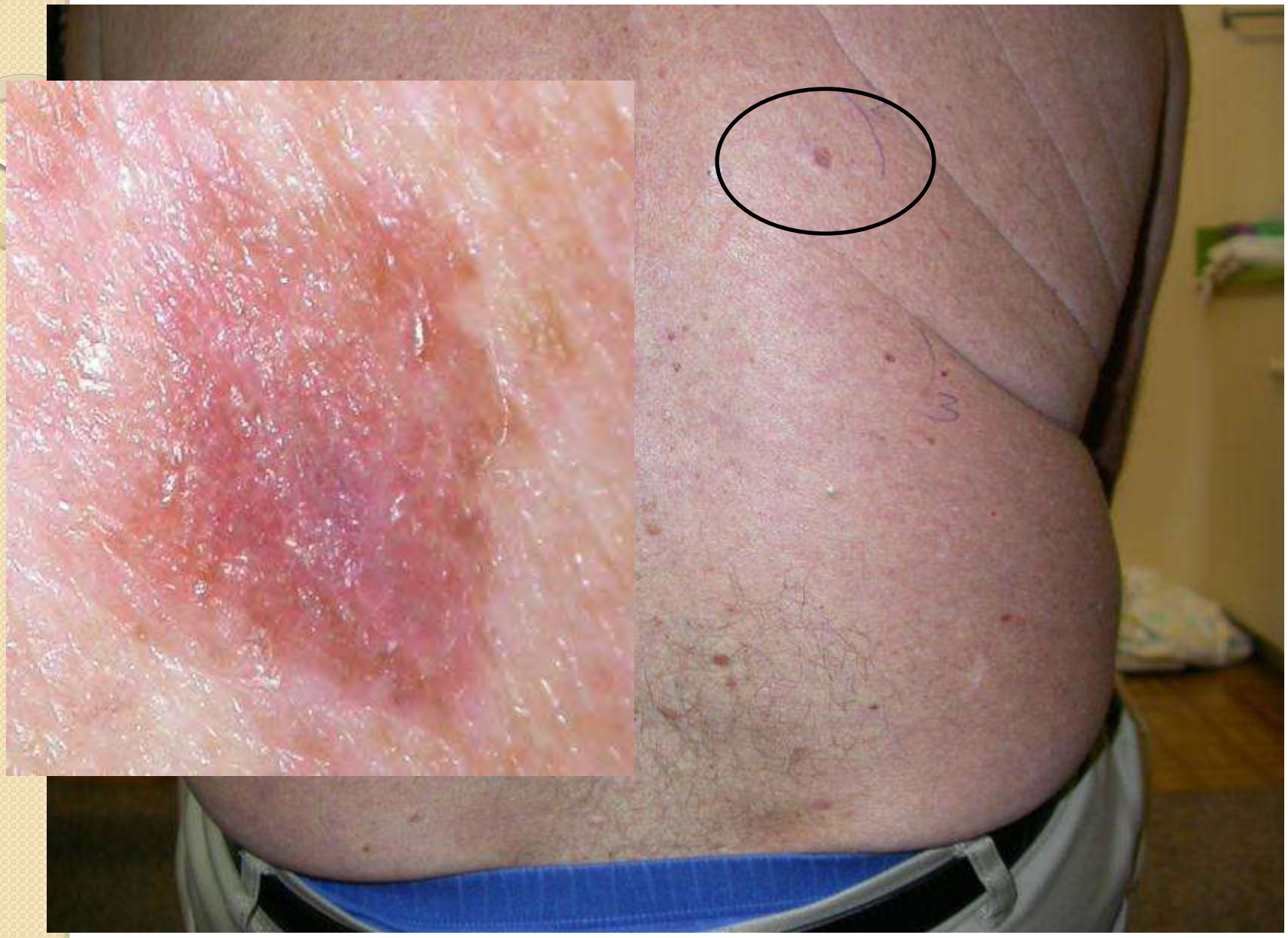


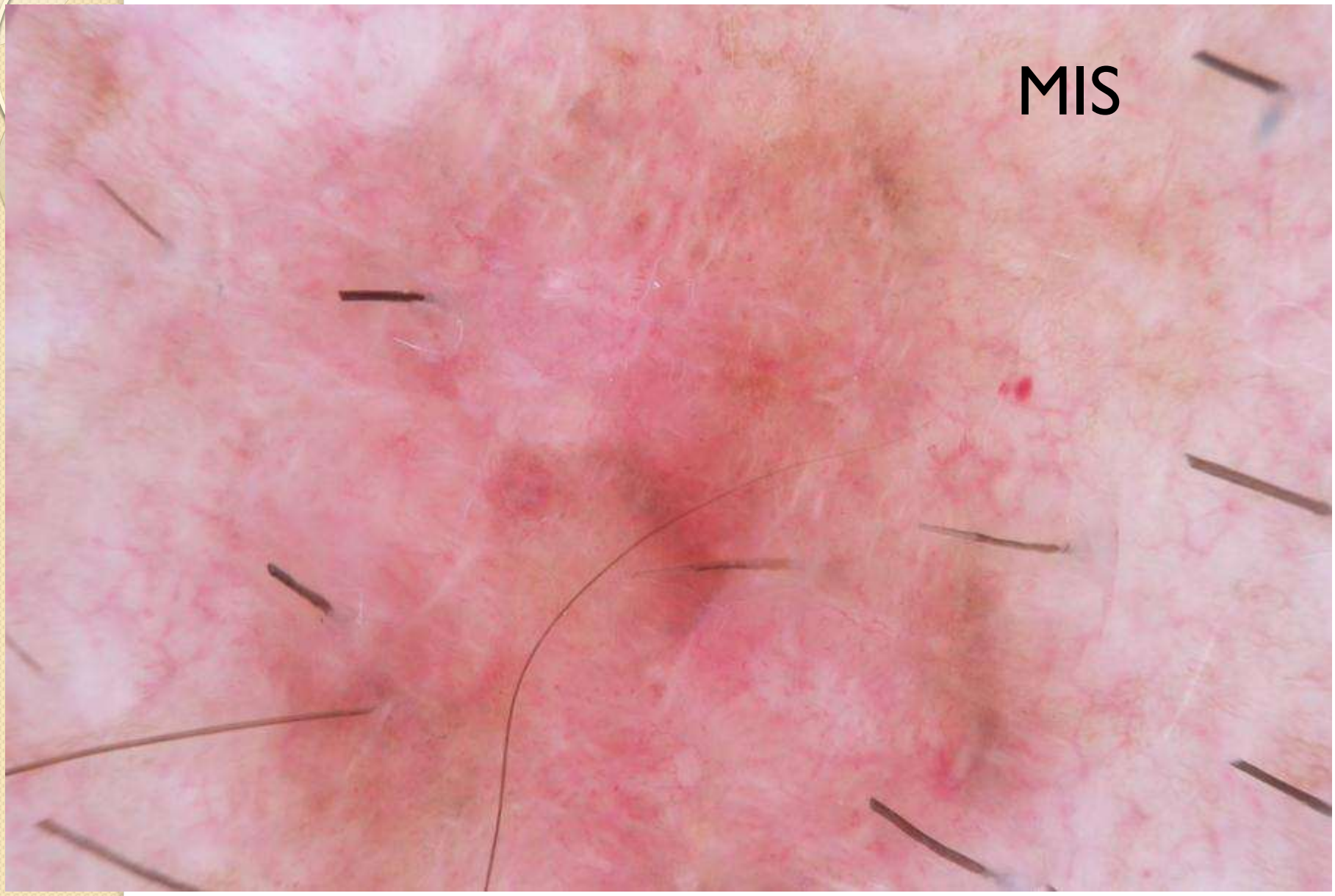


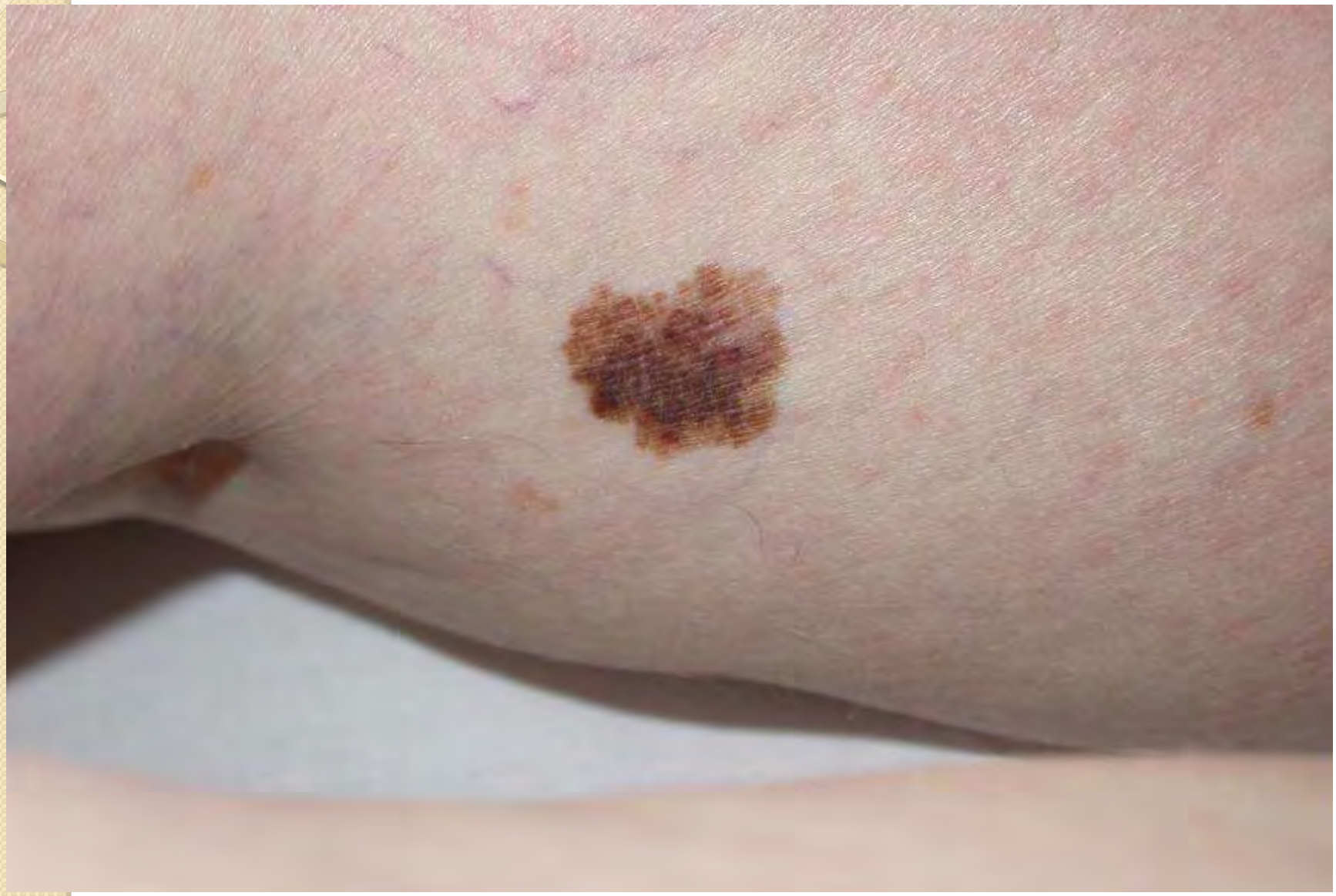

mm

Melanoma can be BCC like

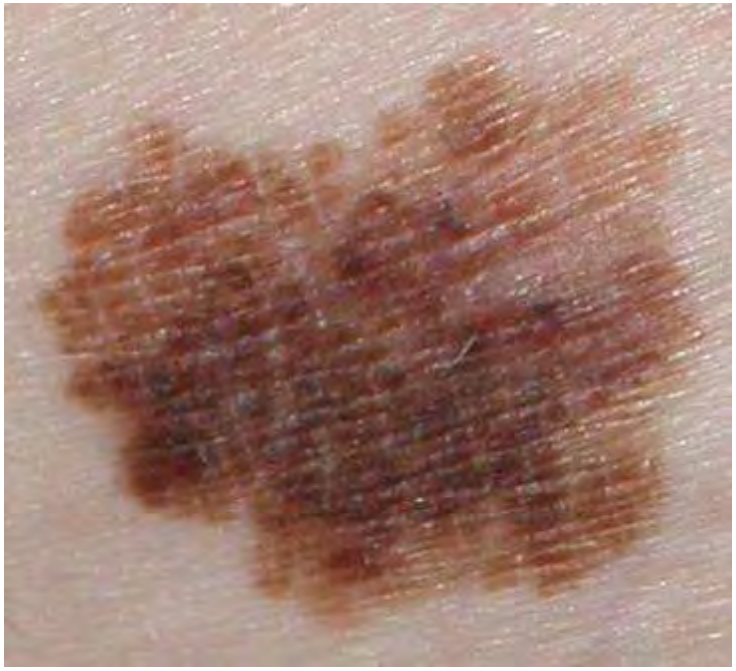


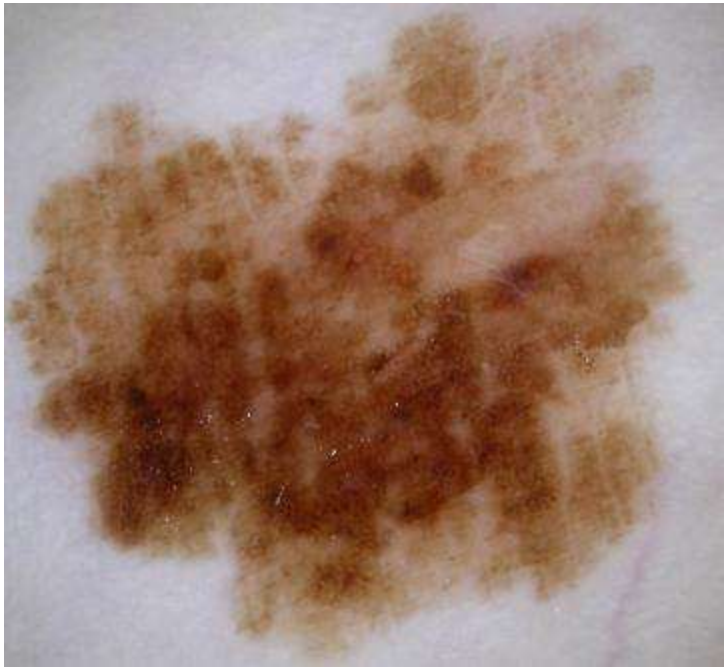


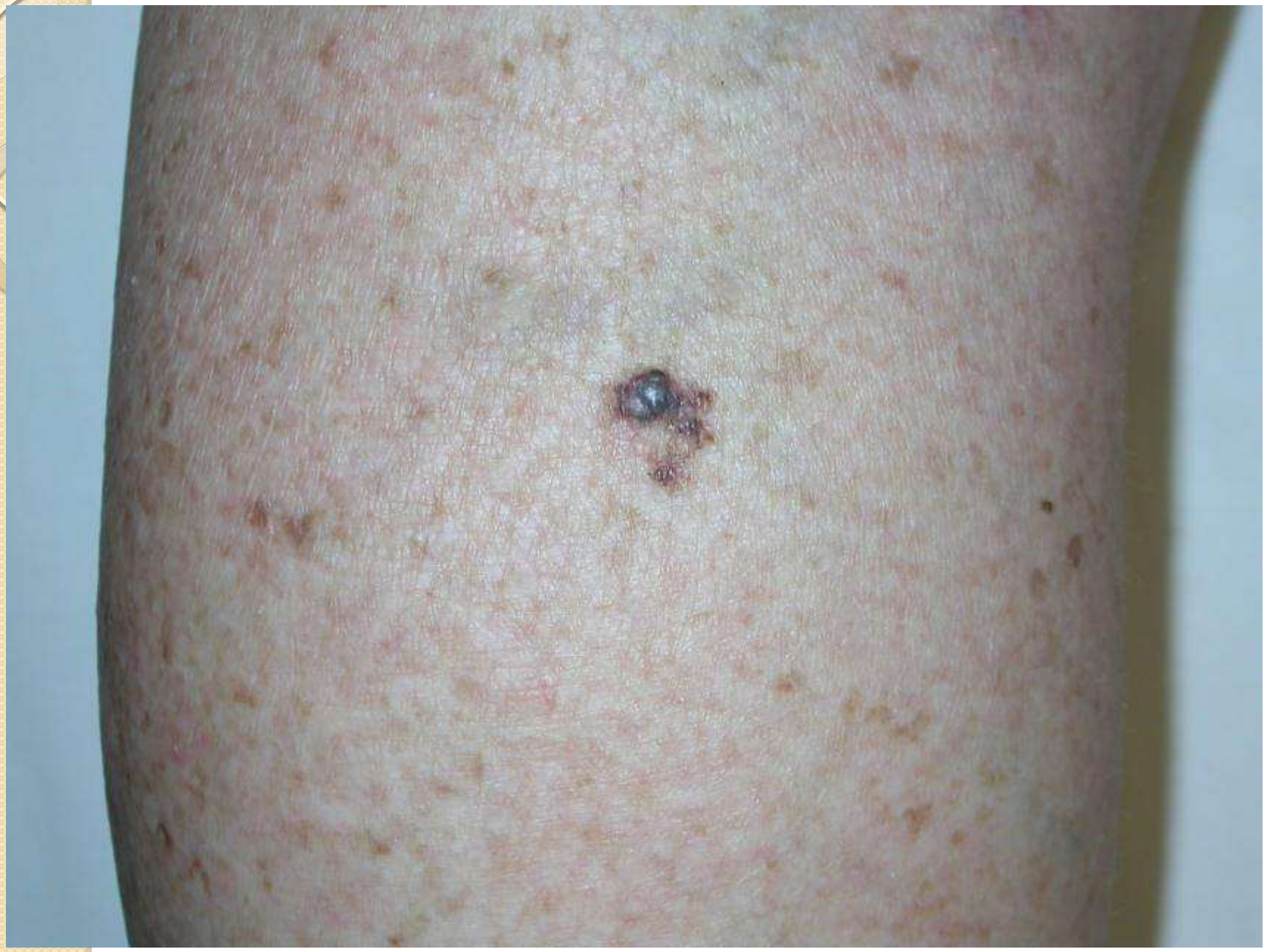

MIS



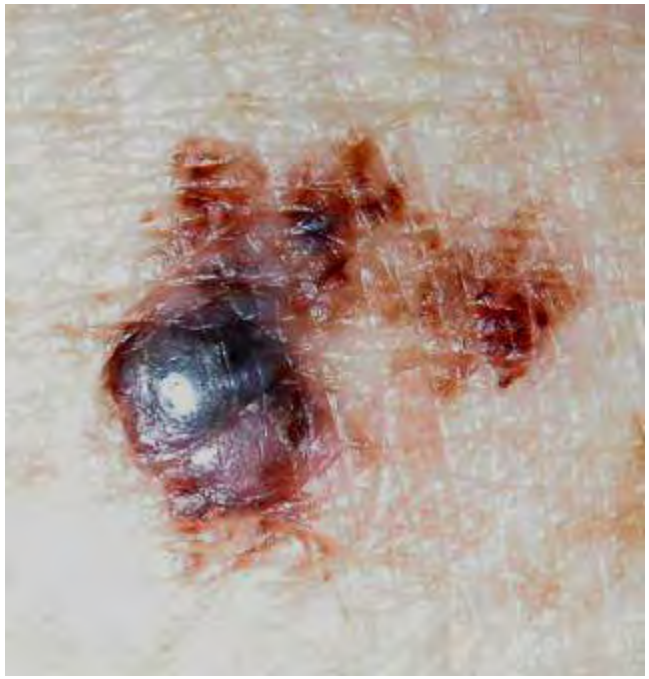
Breslow 0.55mm

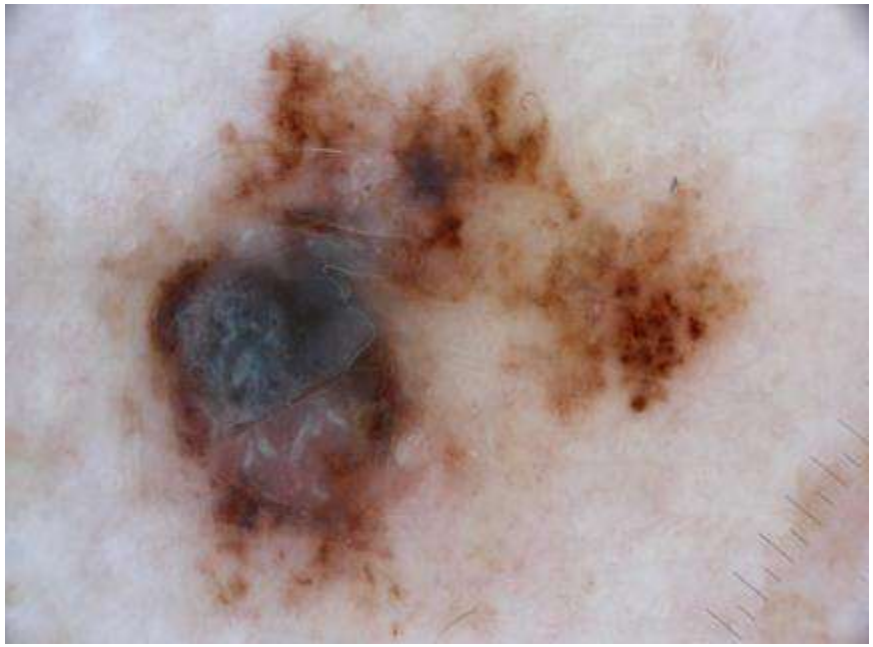


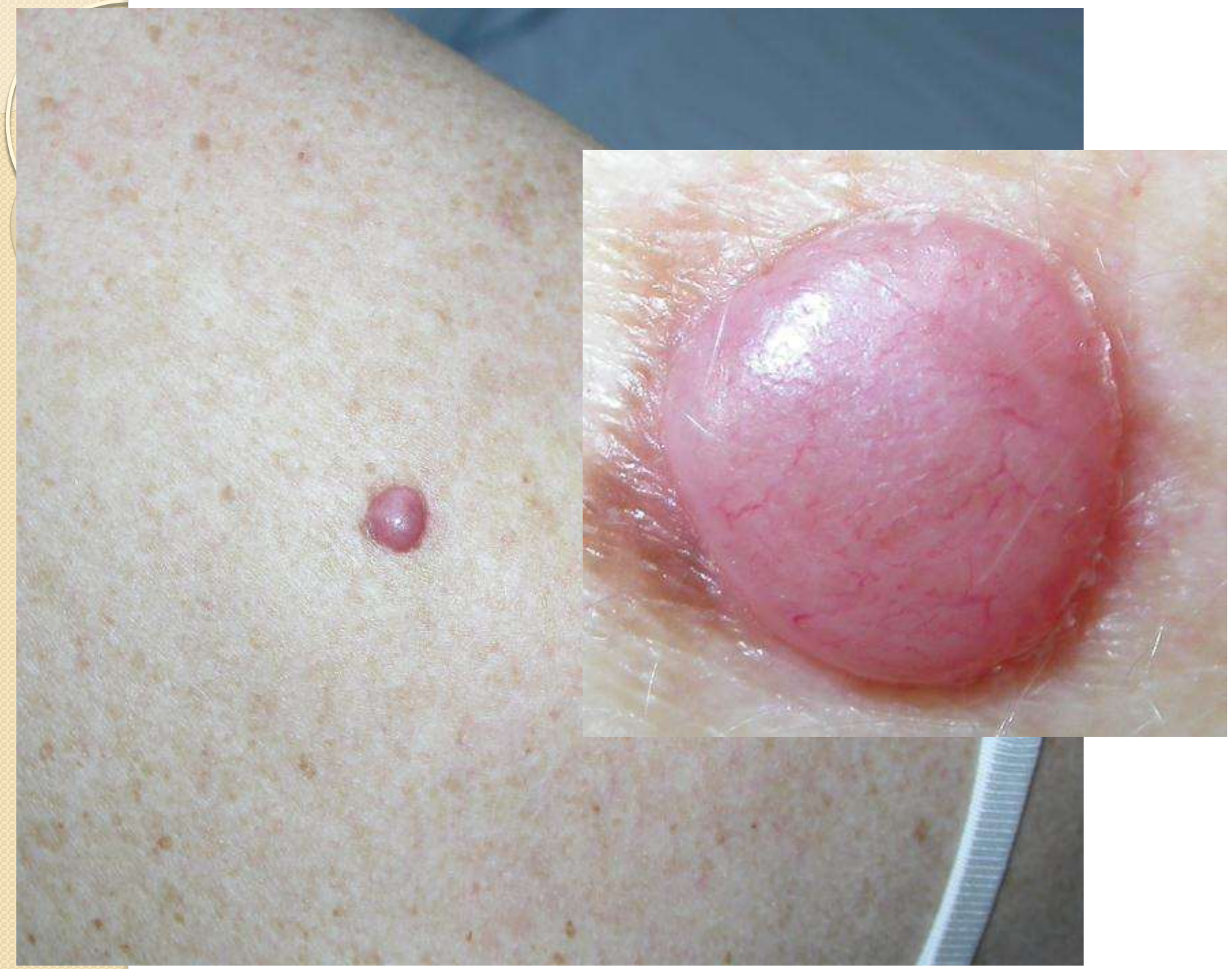


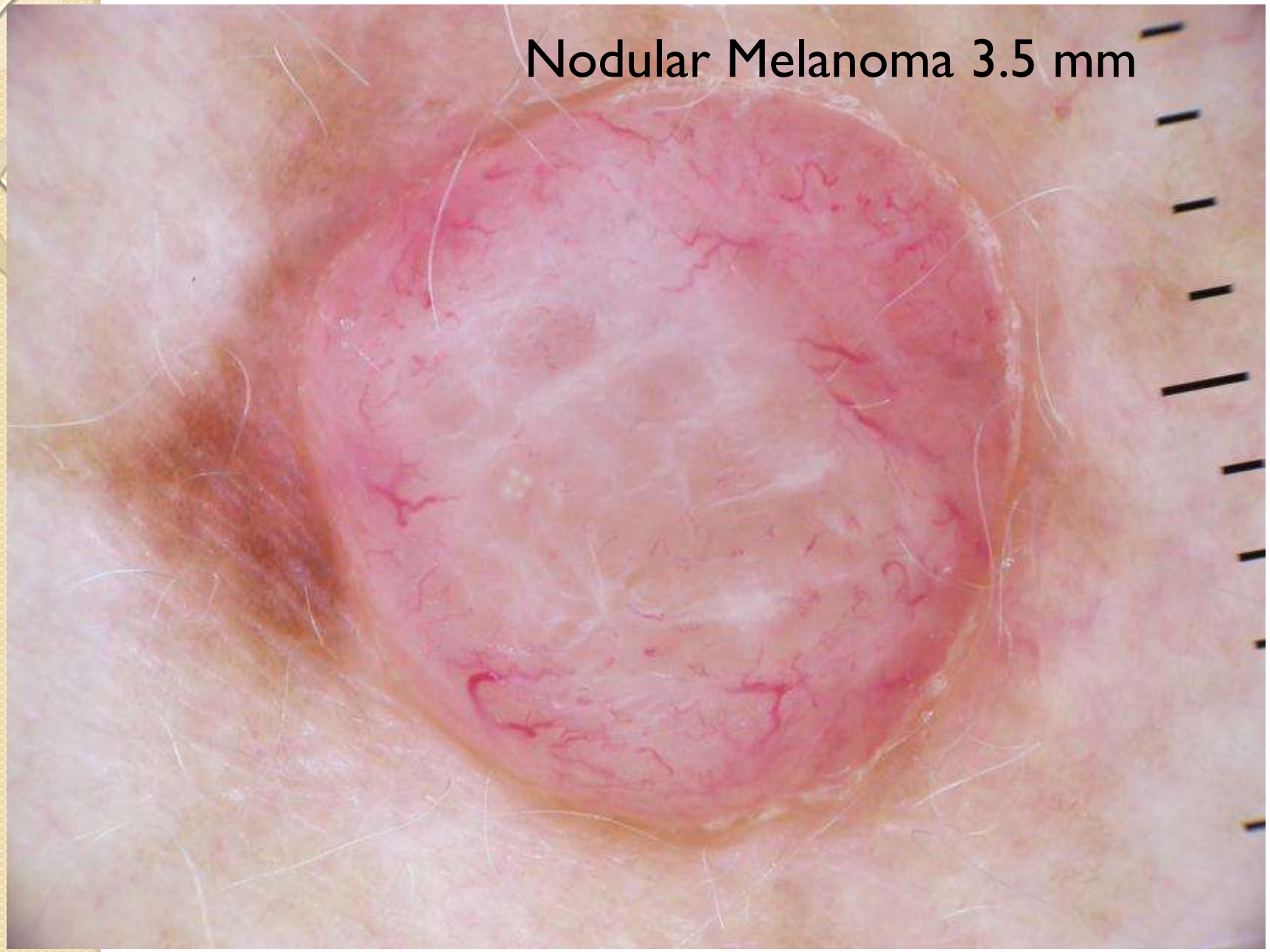


Melanoma Breslow 2.5 mm







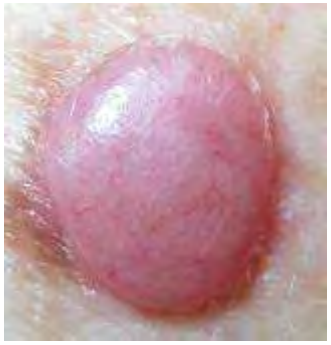
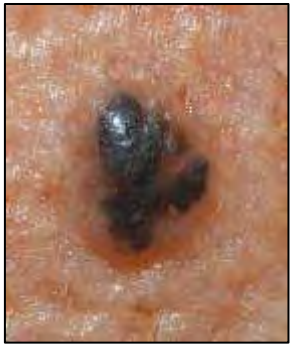

Nodular Melanoma 3.5 mm

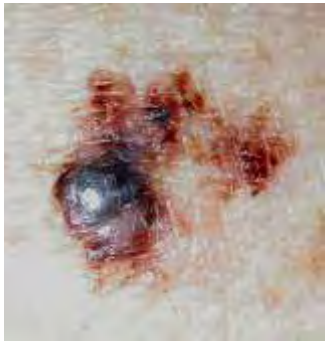
THANK YOU

November 2013 – Dr Mark Foley

cphr CENTRE FOR PUBLIC HEALTH RESEARCH

Cancer Society
Te Kāhui Matepukupuku o Aotearoa



- Why the delay in UV exposure and development of skin cancer

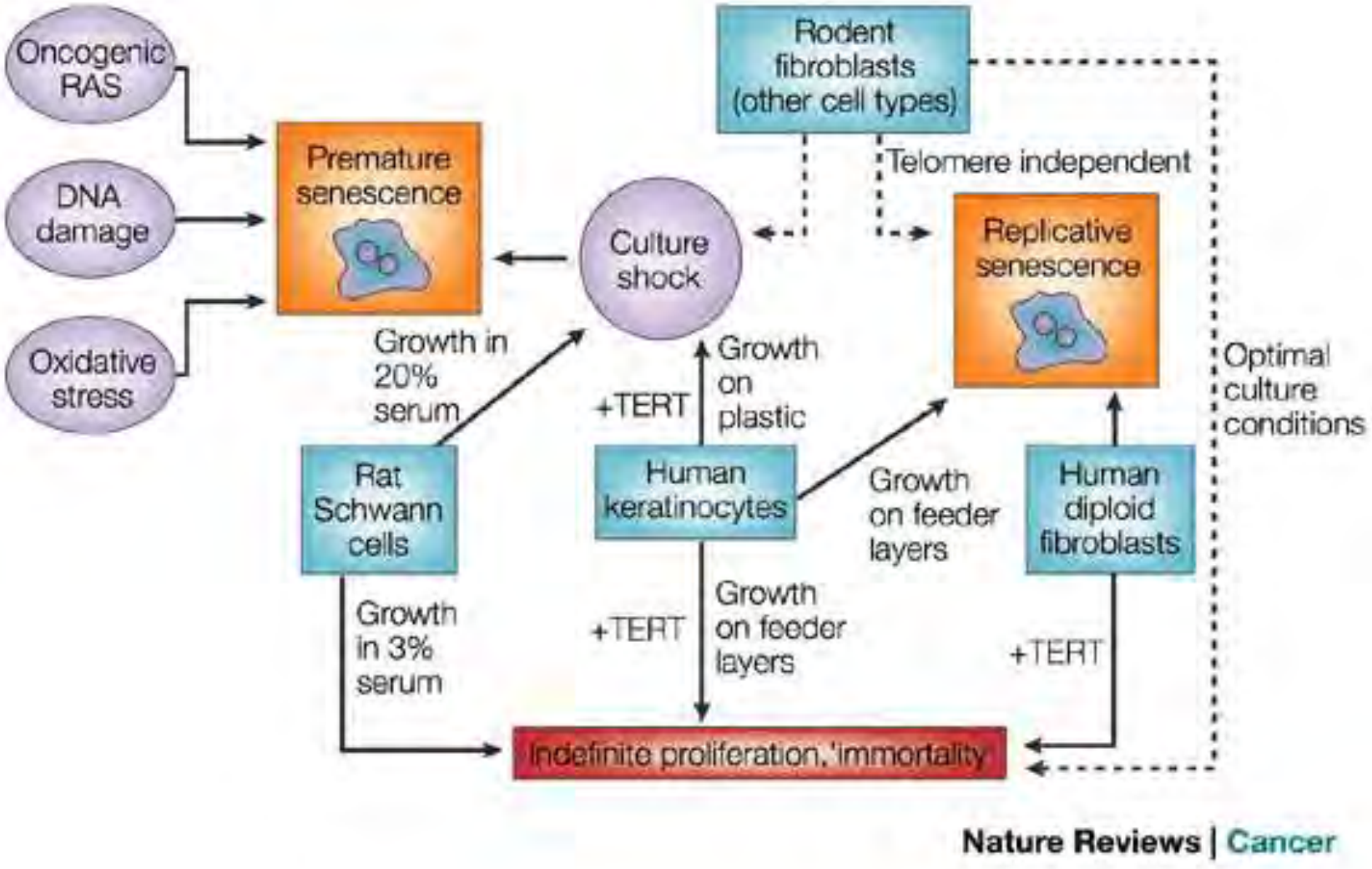
- UV light causes DNA damage
- Initial understanding
  - **DNA repair** or
  - If not able to be repaired -> **apoptosis** (programmed cell death).
  - If both fail DNA damage should be reproduced in the next cell cycle – but if this was true skin cancers would start in early childhood…..
  - In reality DNA damage can take decades to be expressed.

Response to damaged DNA

- DNA repair
- Apoptosis
- Senescence
  - Replicative
  - Premature

Replicative senescence - result of the countdown of an intrinsic mitotic counter.
The only known mitotic counter mechanism is telomere shortening. This mechanism seems to be responsible for the induction of replicative senescence in human diploid fibroblasts.

Premature senescence - induced by extrinsic factors that can act at any point in a cell's replicative history


Oncogenic RAS
DNA damage
Oxidative stress
Premature senescence
Rodent fibroblasts (other cell types)
Telomere independent
Culture shock
Replicative senescence
Growth in 20% serum
+TERT
Growth on plastic
Optimal culture conditions
Rat Schwann cells
Human keratinocytes
Growth on feeder layers
Human diploid fibroblasts
Growth in 3% serum
+TERT
Growth on feeder layers
+TERT
Indefinite proliferation, 'immortality'
Nature Reviews | Cancer

Dermoscopy / Dermatoscopy


dermlite


dermlite

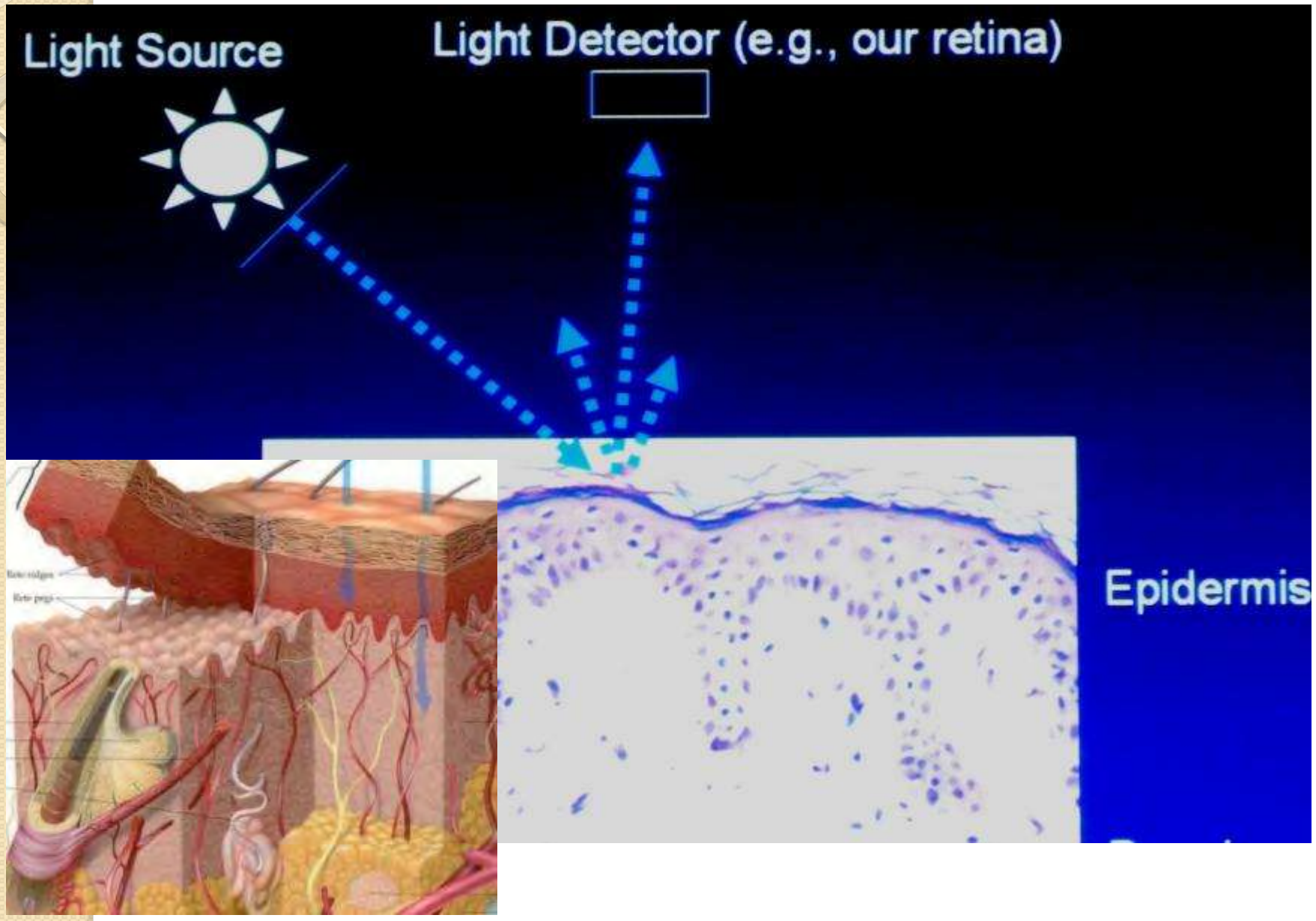
Dermoscopy

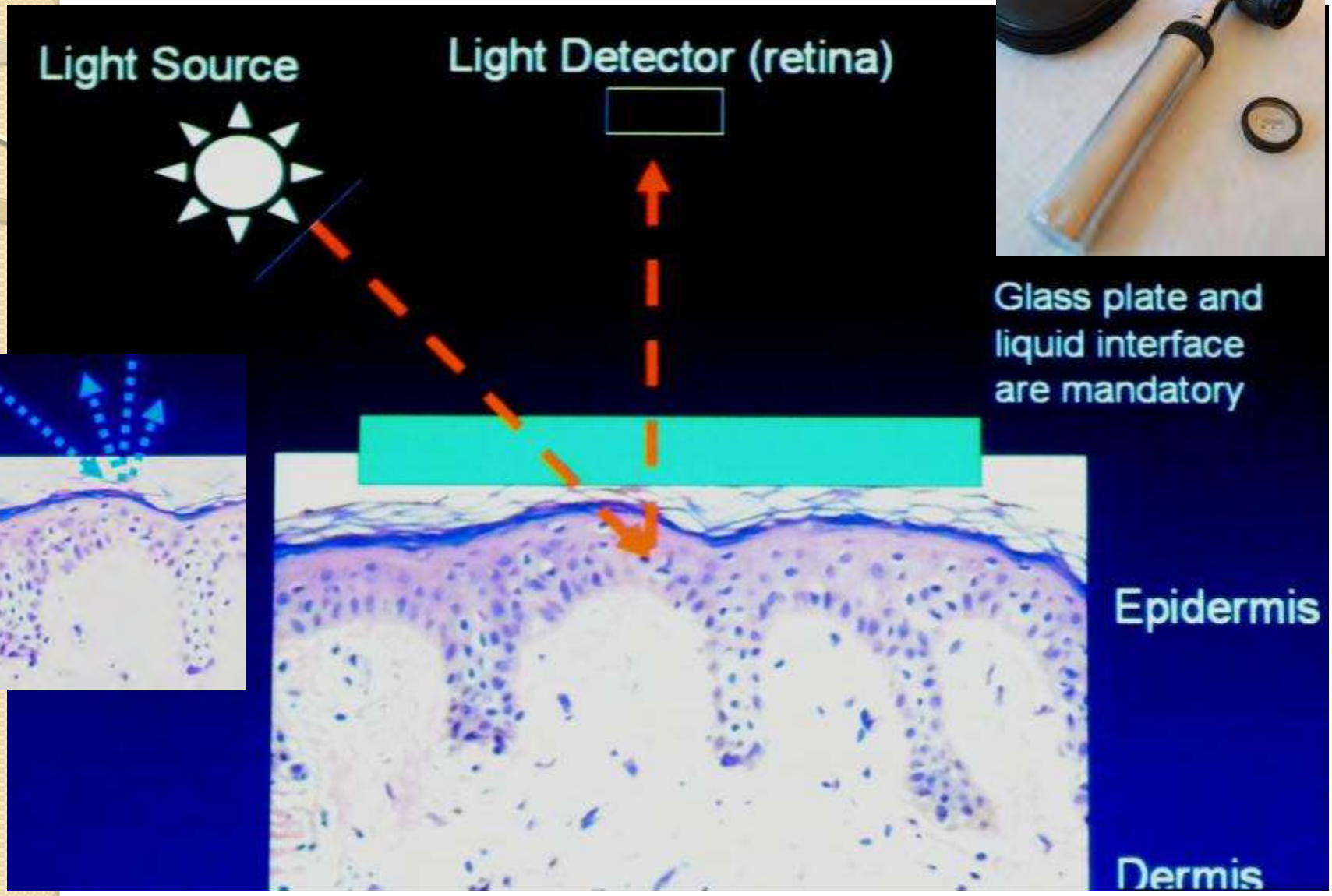
Allows visualisation of skin architecture not able to be seen with the naked eye.

Dramatically improves ability to diagnose early skin cancer and benign lesions

Contact vs Polarising

Dermoscopy vs TBP


Light Source
Light Detector (e.g., our retina)
Epidermis


Light Source
Light Detector (retina)
Glass plate and liquid interface are mandatory
Epidermis
Dermis

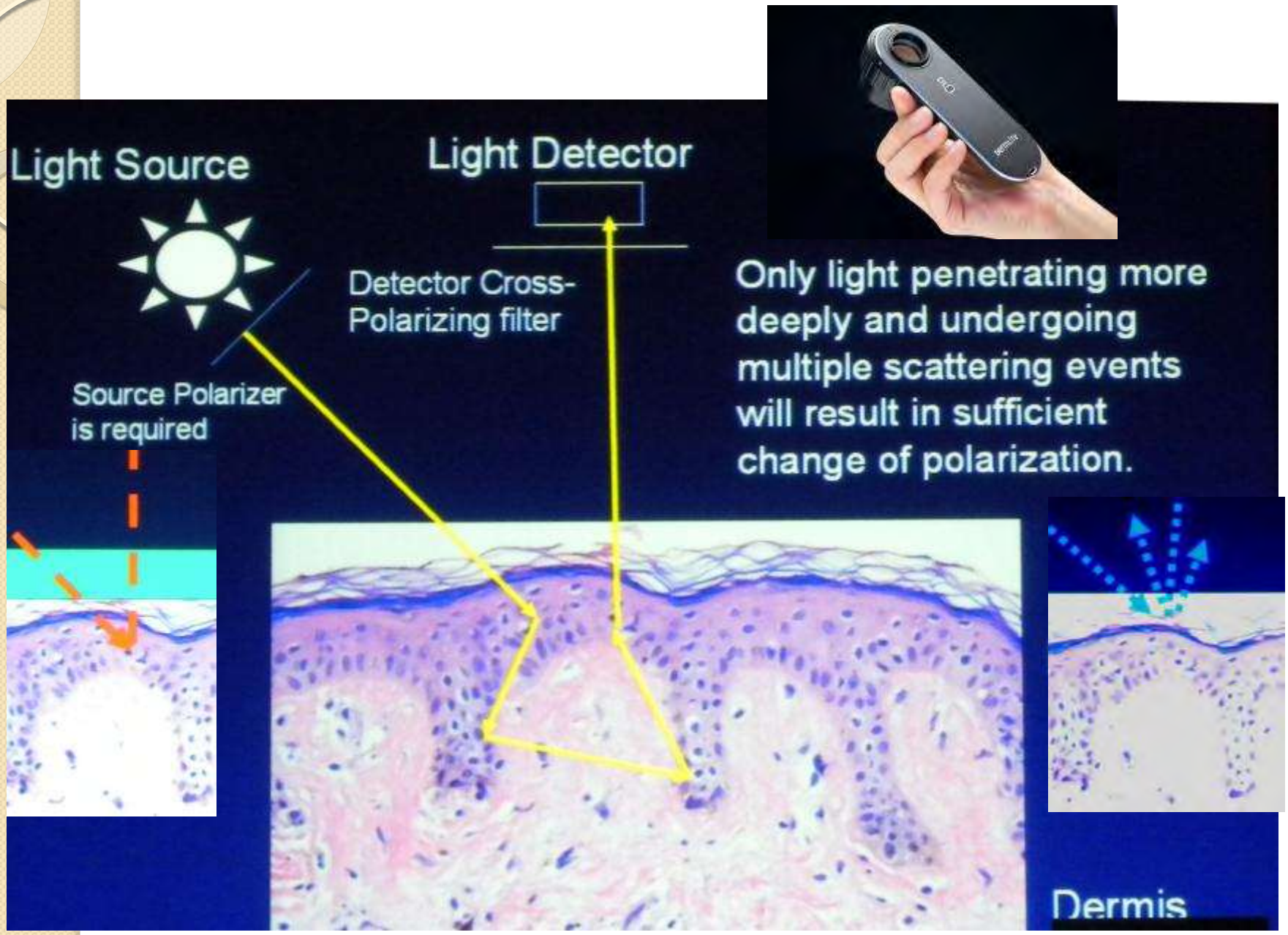
Light Source

Light Detector

Light reflected from superficial epidermis – blocked by crossed polarized filter - insufficient change in the direction of light

Epidermis

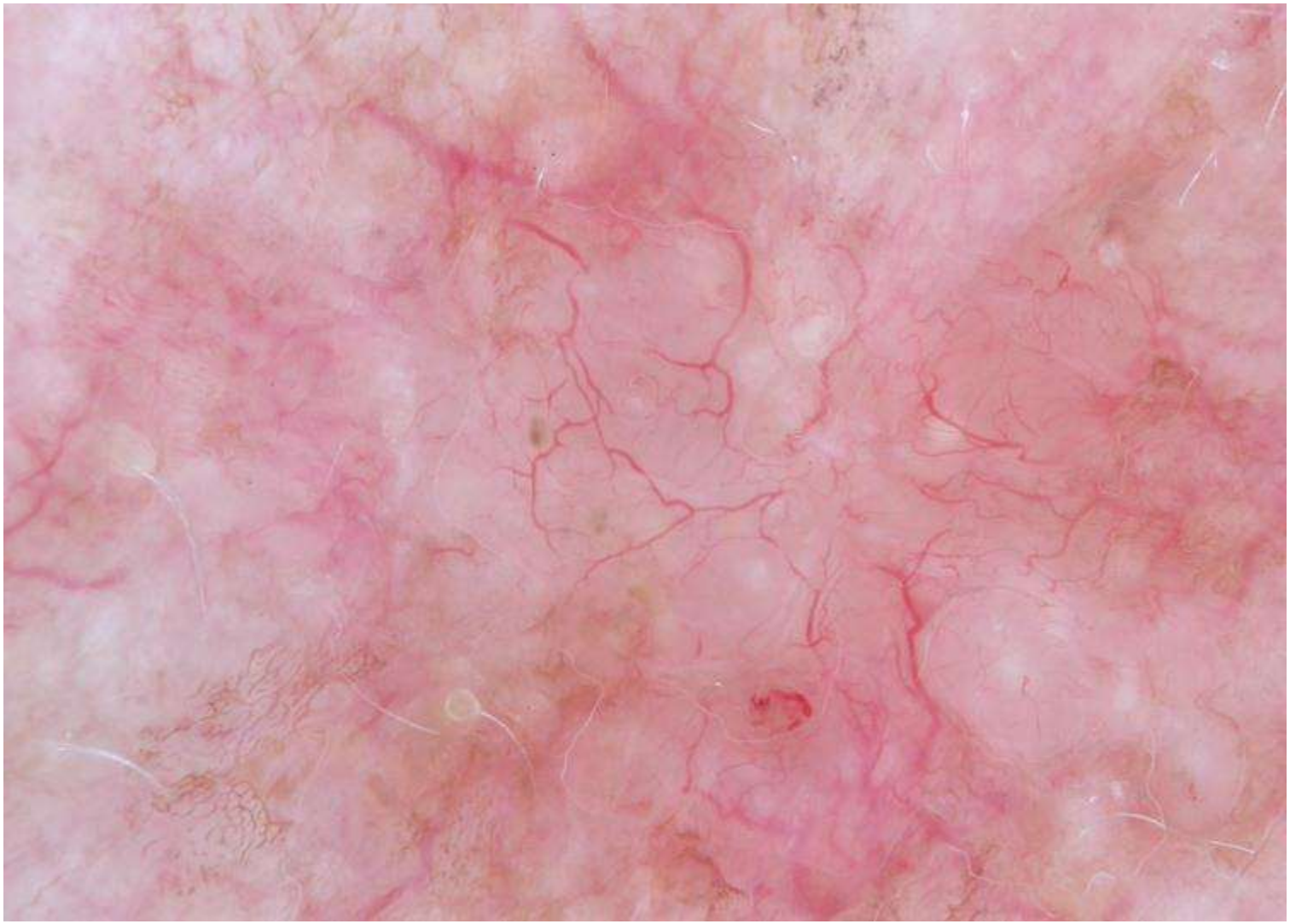
Dermis


Light Source
Light Detector
Detector Cross-Polarizing filter
Source Polarizer is required
Only light penetrating more deeply and undergoing multiple scattering events will result in sufficient change of polarization.
Dermis

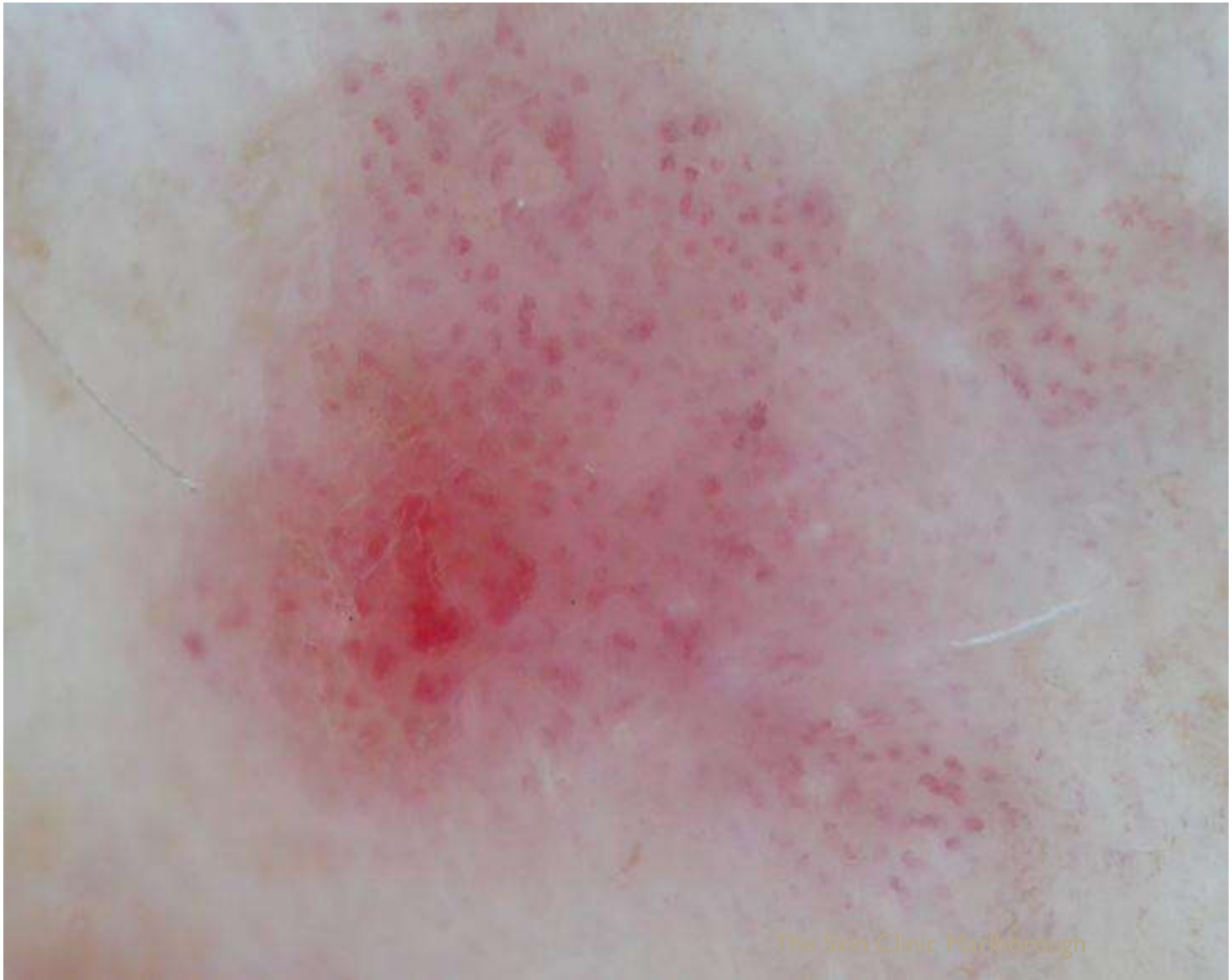
Two important dermoscopic signs for red scaly lesions

- These will improve your pick up rate dramatically

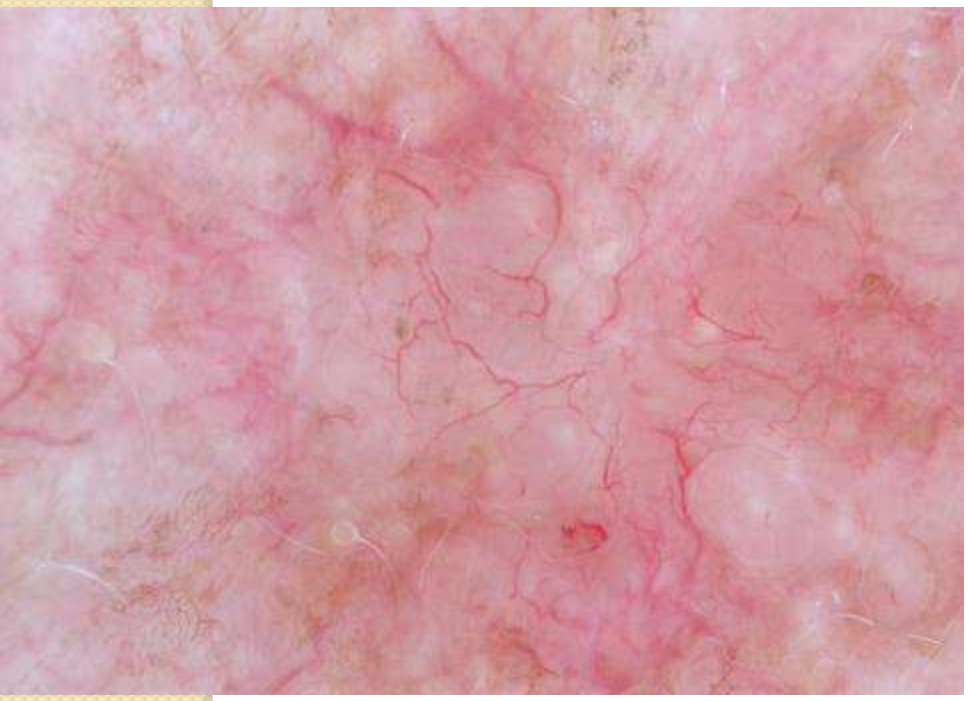
Arborising vessels



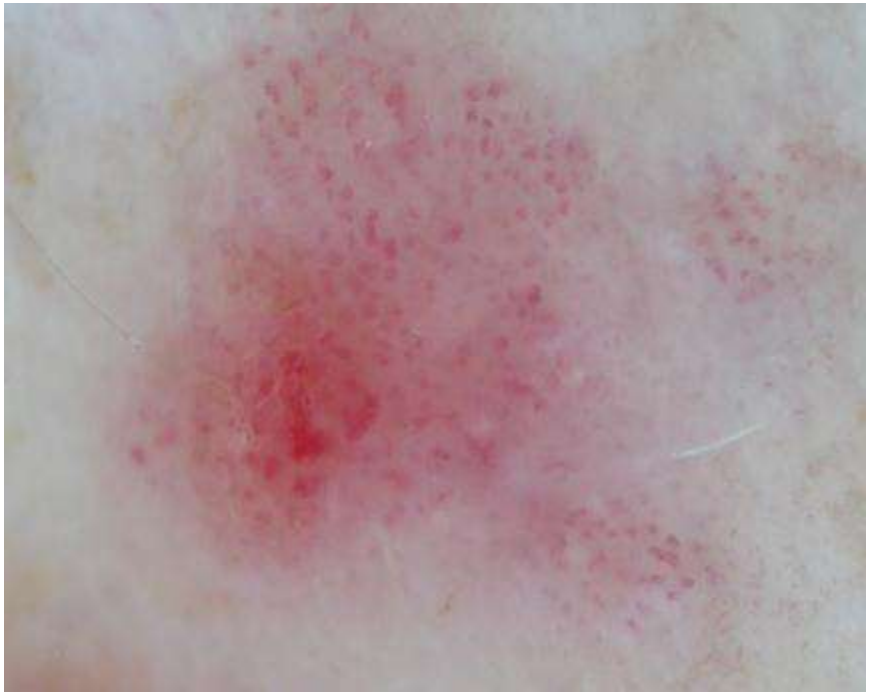
Coiled vessels



BCC



SCC insitu



SCC background

Cutaneous Squamous Cell Carcinoma and Human Papillomavirus: Is There an Association?

Bishr Aldabagh, MD,* Jorge Gil C. Angeles, PhD,† Adela R. Cardones, MD,* and Sarah T. Arron, MD, PhD†

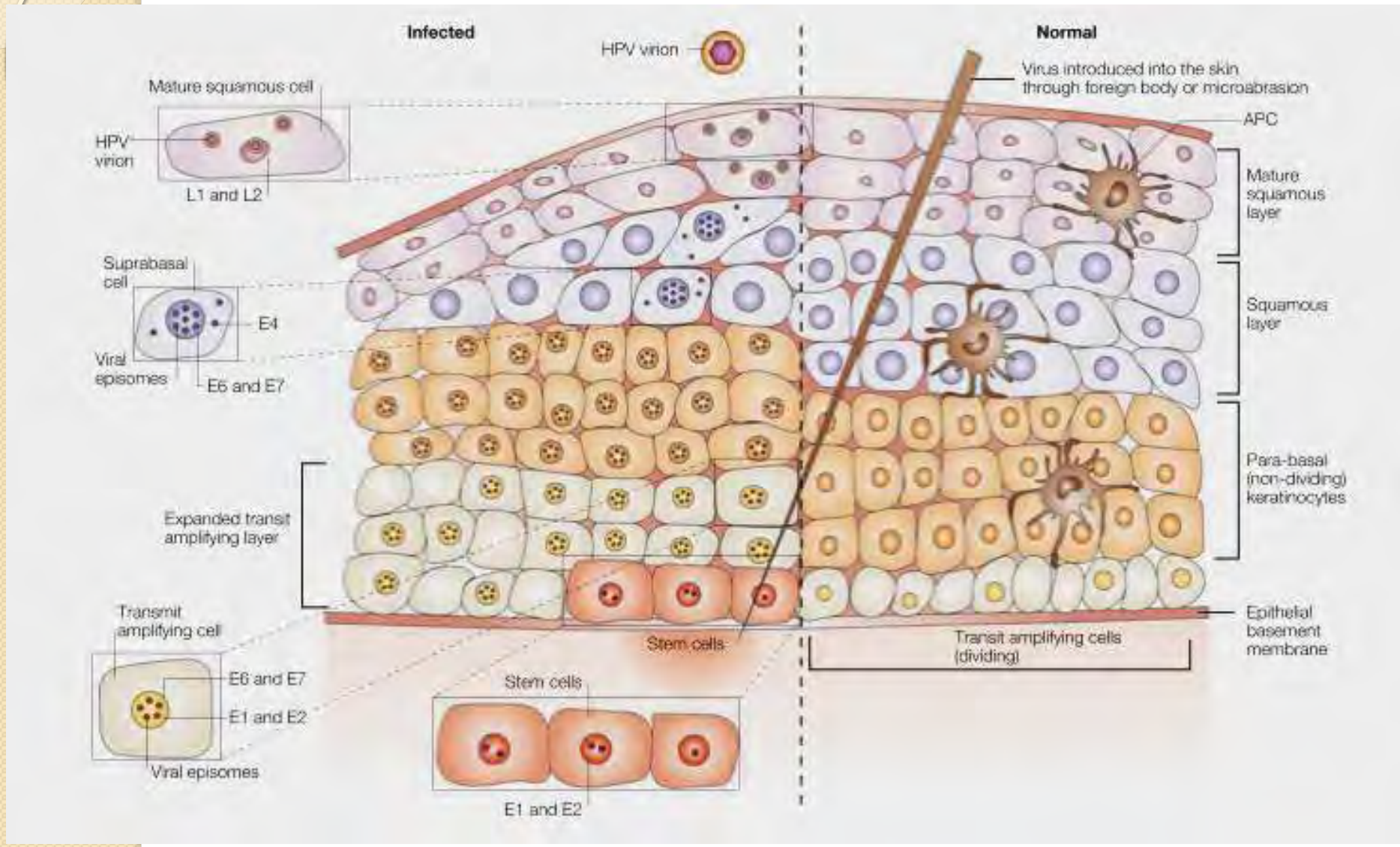
BACKGROUND The role of human papillomavirus (HPV) in the induction and maintenance of cervical, anogenital, and some oropharyngeal carcinomas is well recognized, but its role in cutaneous squamous cell carcinoma (SCC) remains to be elucidated. HPV is thought to act as a possible cocarcinogen in the development of SCC.

OBJECTIVE To review the literature assessing the correlation between and possible causation of HPV and cutaneous SCC in immunocompetent and immunocompromised populations.

METHODS We reviewed HPV sampling and detection methods, epidemiologic studies examining HPV carriage in immunocompetent and immunosuppressed individuals, and evidence asserting an association between HPV and cutaneous SCC.

RESULTS Although an abundant body of evidence points toward a link between HPV and cutaneous SCC, many studies indicate otherwise. Recent studies have focused on viral activity in addition to DNA presence.

CONCLUSION The possibility exists that HPV may play a role in the induction but not maintenance of cutaneous SCC.


Infected
HPV virion
Normal
Virus introduced into the skin through foreign body or microabrasion
Mature squamous cell
HPV virion
L1 and L2
APC
Mature squamous layer
Suprabasal cell
E4
Viral episomes
E6 and E7
Squamous layer
Para-basal (non-dividing) keratinocytes
Expanded transit amplifying layer
Transmit amplifying cell
E6 and E7
E1 and E2
Viral episomes
Stem cells
Epithelial basement membrane
Transit amplifying cells (dividing)
Stem cells
E1 and E2

Fibroblast Senescence and Squamous Cell Carcinoma: How Wounding Therapies Could Be Protective

Jeffrey B. Travers, MD, PhD,*†§¶ Dan F Spandau, PhD,*‡ Davina A. Lewis, MS,* Christiane Machado, MD,* Melanie Kingsley, MD,* Nico Mousdicas, MD,*¶ and Ally-Khan Somani, MD, PhD*

BACKGROUND Squamous cell carcinoma (SCC), which has one of the highest incidences of all cancers in the United States, is an age-dependent disease, with the majority of these cancers diagnosed in people age 70 and older. Recent findings have led to a new hypothesis on the pathogenesis of SCC.

OBJECTIVES To evaluate the potential of preventive therapies to reduce the incidence of SCC in at-risk geriatric patients.

MATERIALS AND METHODS Survey of current literature on wounding therapies to prevent SCCs.

RESULTS This new hypothesis of SCC photocarcinogenesis states that senescent fibroblasts accumulate in the dermis, resulting in a reduction in dermal insulin-like growth factor-1 (IGF-1) expression. This lack of IGF-1 expression sensitizes epidermal keratinocytes to fail to suppress ultraviolet light B (UVB)-induced mutations, leading to increased proclivity to photocarcinogenesis. Recent evidence suggests that dermal wounding therapies, specifically dermabrasion and fractionated laser resurfacing, can decrease the proportion of senescent dermal fibroblasts, increase dermal IGF-1 expression, and correct the inappropriate UVB response found in geriatric skin, protecting geriatric keratinocytes from UVB-induced SCC initiation.

CONCLUSIONS In this review, we will discuss the translation of pioneering basic science results implicating commonly used dermal fibroblast rejuvenation procedures as preventative treatments for SCC.

The authors have indicated no significant interest with commercial supporters.