Sun Damage and Skin Cancers
Prevention & Management

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OBJECTIVES

At the conclusion of the lecture, participants will be better able to

1. understand the effect of ultraviolet light on skin
2. recognize precursors of skin cancer
3. recognize the most common types of skin cancers
4. identify individuals and populations prone to skin cancers
5. aid in diagnosis and treatment of skin cancers and their precursors

PRE-TEST

What are the two big bucket categories of skin cancer?

Which demographic has the most skin cancer?

Which skin cancer accounts for the highest morbidity & mortality?

What is the most common skin cancer? Second most common?

What is the increased likelihood of individuals with a h/o malignant melanoma (MM) having a 2nd primary lesion as compared to those w/o MM hx?

What is the most effective way to reduce skin cancer risk?

What is the gold standard for treatment of most skin cancers?

What is the most important prognostic indicator of MM?

Which demographic presents with more advanced melanoma at dx?

What is the minimum spf (sun protection factor) recommendation?

Epidemiology – skin cancer

Two “big-bucket” categories – melanoma and nonmelanoma skin cancer (NMSC)

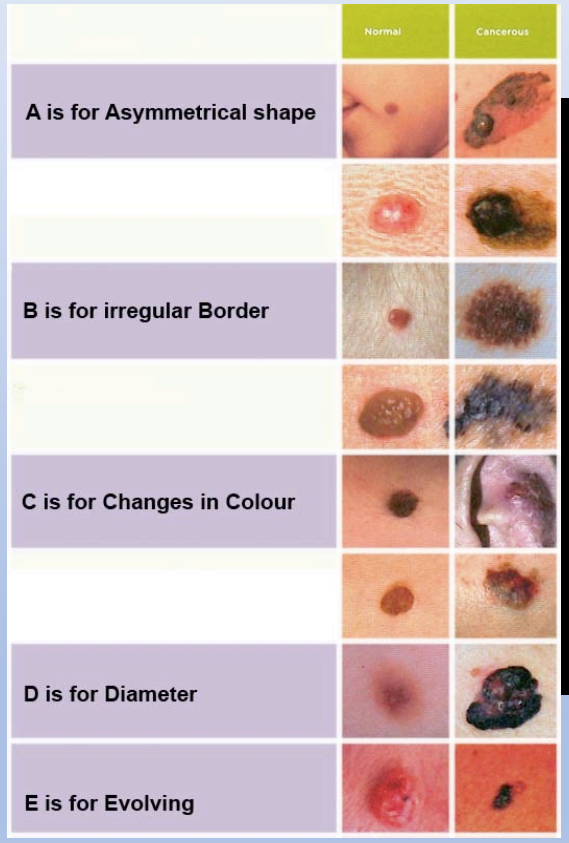
Skin cancer affects an estimated 3.5 million Americans each year

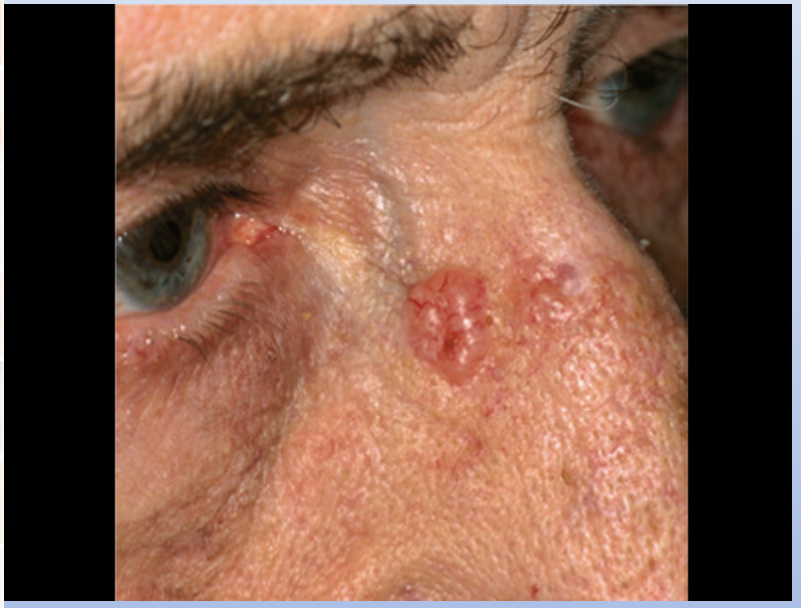
Although predominantly a disease of the elderly white population, skin cancer can affect individuals of all ages and races.

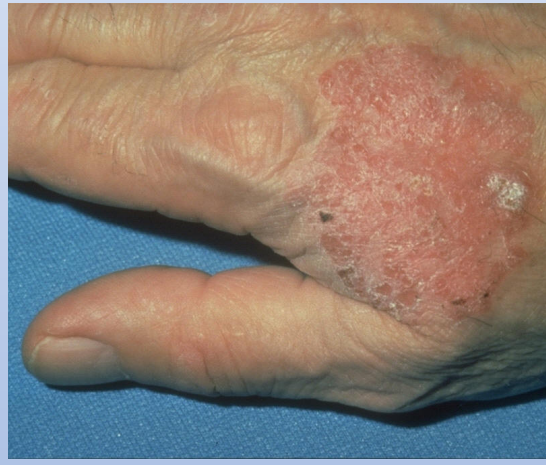
The most common type of skin cancer is basal cell carcinoma, followed in incidence by squamous cell carcinoma.

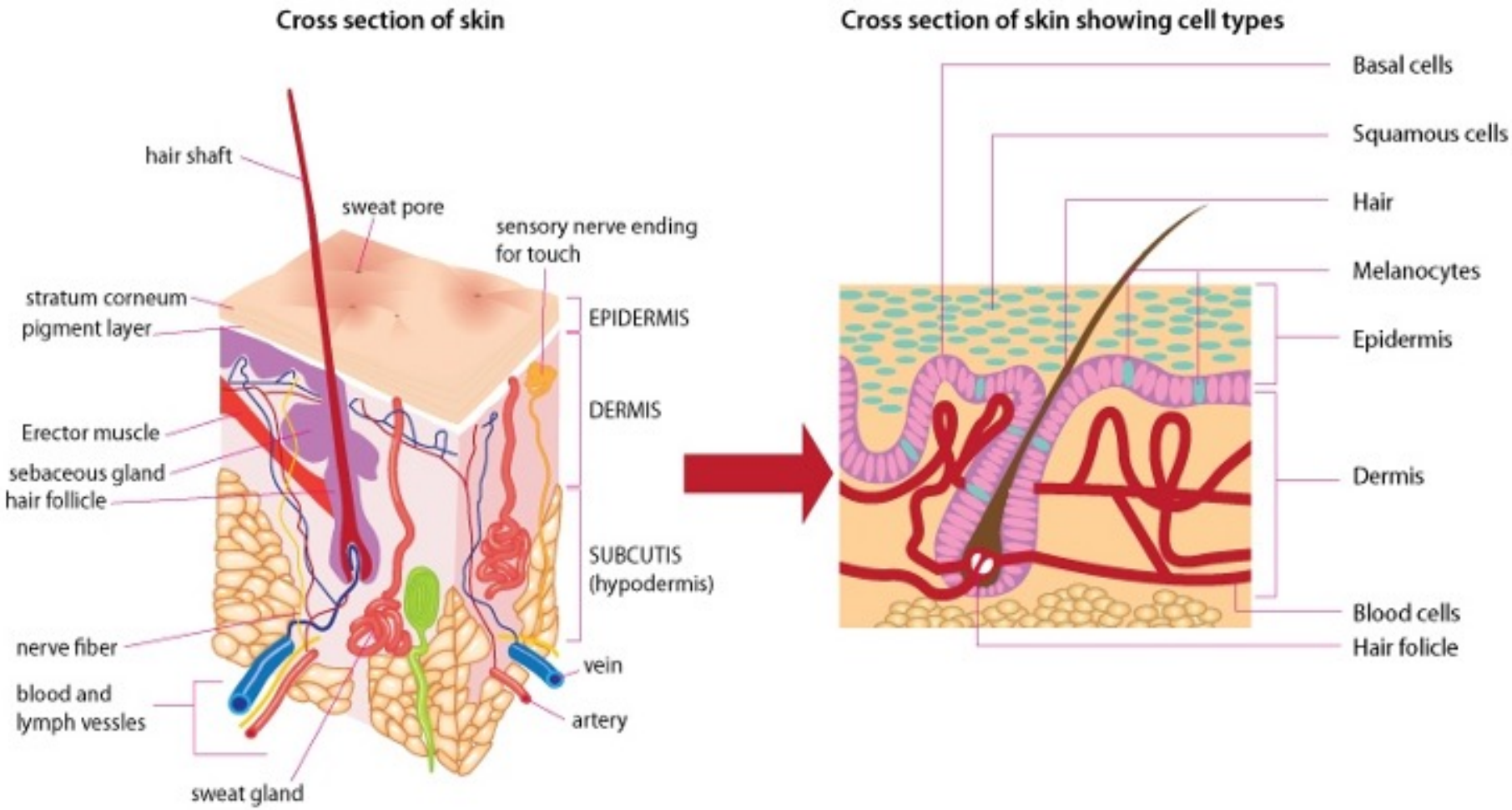
Melanoma, is less common than basal and squamous cell carcinomas but accounts for the greatest skin cancer–related morbidity and mortality.

melanoma and non-melanoma skin cancers





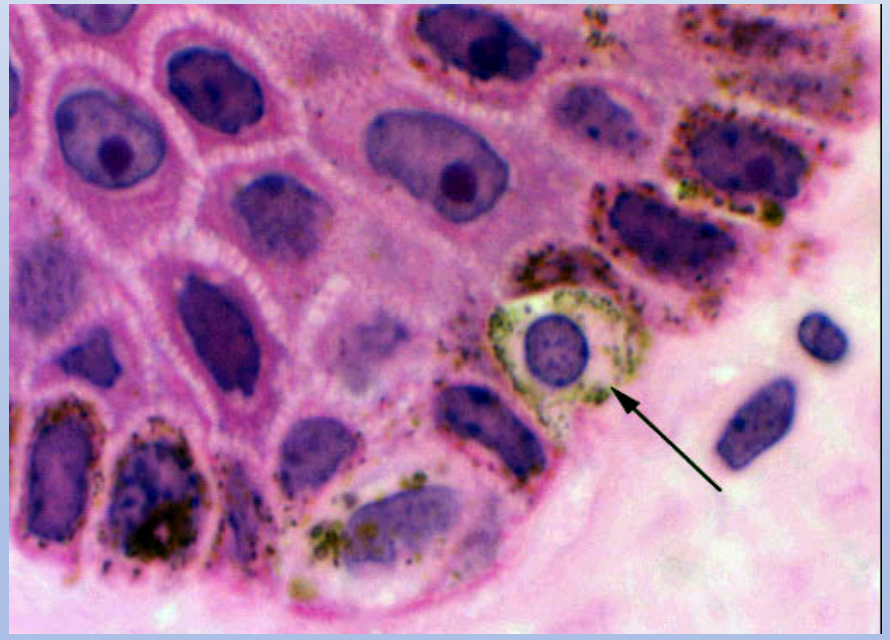


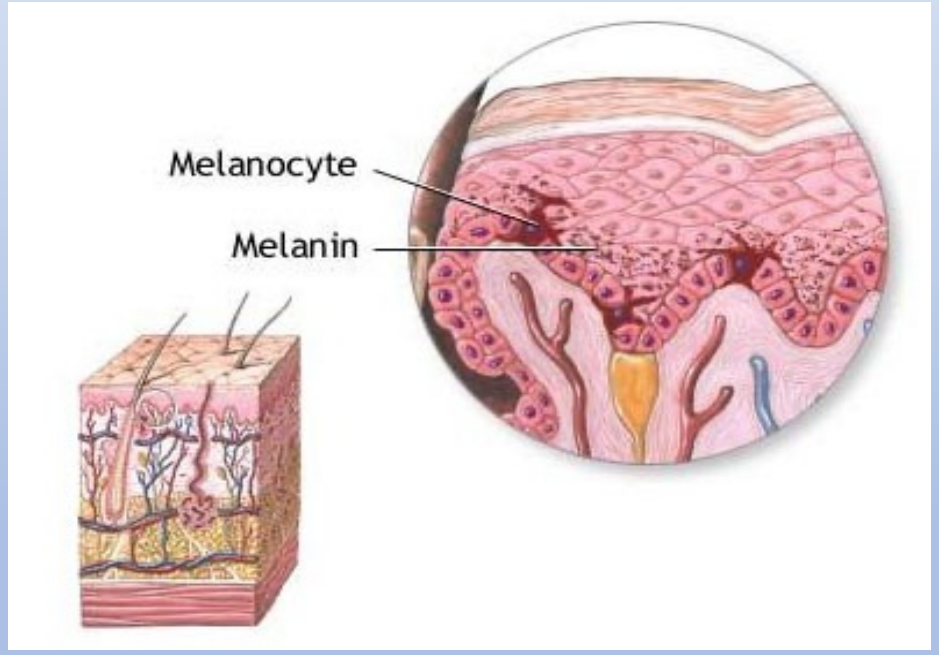

Cross section of skin
hair shaft
sweat pore
sensory nerve ending for touch
stratum corneum
pigment layer
EPIDERMIS
DERMIS
Erector muscle
sebaceous gland
hair follicle
SUBCUTIS (hypodermis)
nerve fiber
vein
blood and lymph vessles
artery
sweat gland
Cross section of skin showing cell types
Basal cells
Squamous cells
Hair
Melanocytes
Epidermis
Dermis
Blood cells
Hair folicle

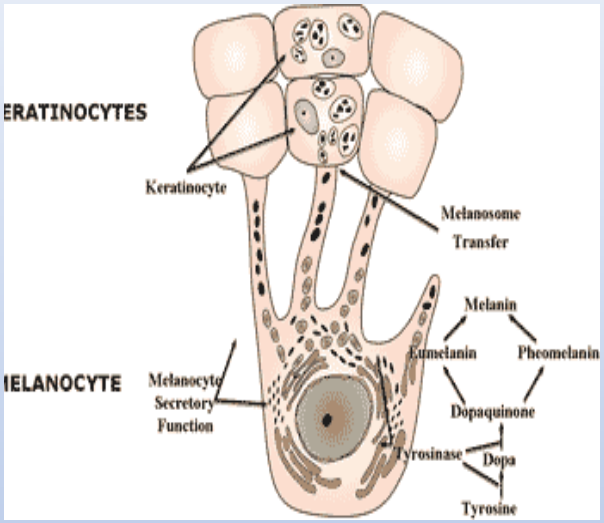
Skin Color

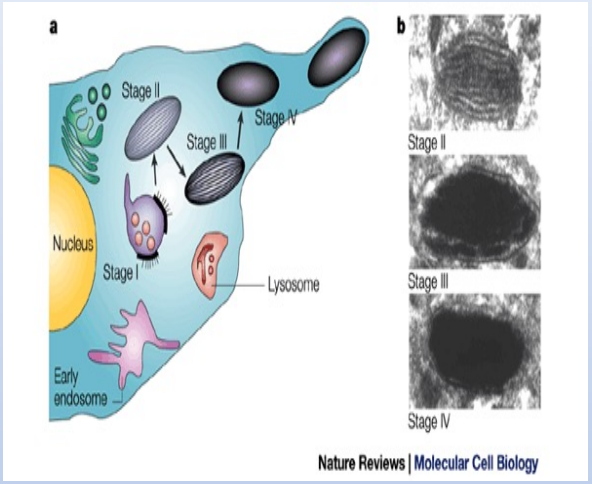
- The skin pigment, **melanin**, is **produced by melanocytes** which are in the basal layer of the epidermis & the matrix portion of the hair bulb
- No significant differences in the *number* of melanocytes in different skin types or among individuals of different race or ethnicity
- Skin color is determined by the epidermal ***density and distribution of melanin*** which is controlled by the enzyme tyrosinase
- There is also variation **in *number, size and groupings of melanosomes*** in melanocytes and keratinocytes and in efficiency of melanosome transfer to a keratinocyte (determines density and distribution of melanin)

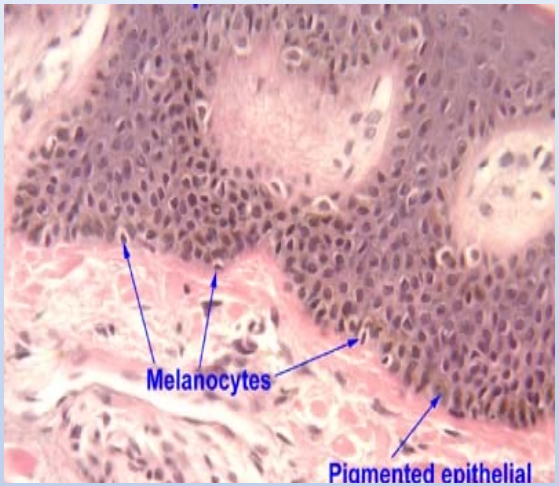
Melanocytes on histology











melanin, melanosomes, & pigmentation

Fitzpatrick Skin Types

TYPE 1: Highly sensitive, always burns, never tans.
Example: Red hair with freckles

TYPE 2: Very sun sensitive, burns easily, tans minimally.
Example: Fair skinned, fair haired Caucasians

TYPE 3: Sun sensitive skin, sometimes burns, slowly tans to light brown.
Example: Darker Caucasians

TYPE 4: Minimally sun sensitive, burns minimally, always tans to moderate brown.
Example: Mediterranean type Caucasians

TYPE 5: Sun insensitive skin, rarely burns, tans well.
Example: Some Hispanics, some Blacks

TYPE 6: Sun insensitive, never burns, deeply pigmented.
Example: Darker Blacks

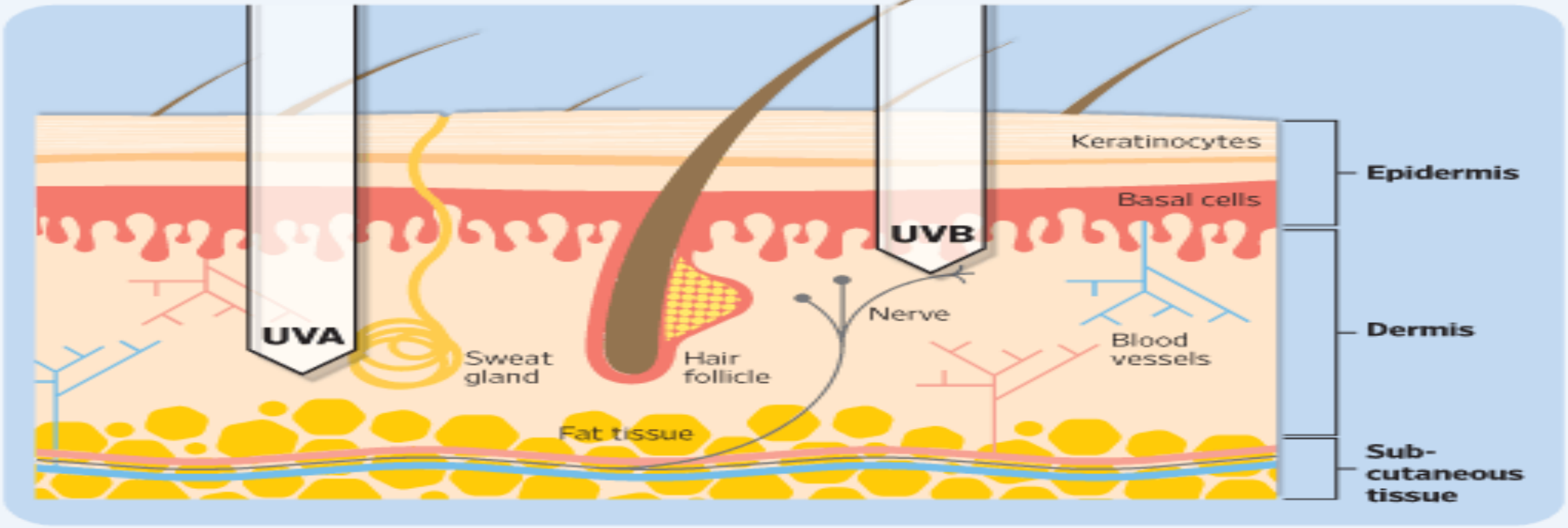
Photo-protective quality of darker skin

	BLACK SKIN	WHITE SKIN
Mean protective factor from UVB light	13.4	3.4
UVB light transmission into the dermis	5.7%	29.4%

- Lower incidence of melanoma & non-melanoma cancers in people of color due to the photo-protective qualities of darker skin
- However, patients of *all* skin types should be educated about skin cancer and screened

What Happens to Your Skin

The sun's ultraviolet rays are classified in three categories, based on their wavelength from the sun to the earth. UVA and UVB rays have harmful effects on the skin, while UVC rays do not reach the earth's surface.



UVA

- These rays penetrate the epidermis and are **dispersed into the dermis.**
- Prolonged exposure shrinks the collagen and elastin, causing the epidermis (top layer) to sag. The result is accelerated **skin aging and wrinkling.**
- Can penetrate **clouds and glass.**
- **Tanning booths** primarily use UVA rays.

UVB

- These rays penetrate the **epidermis.**
- Stimulate the production of melanin, resulting in **freckles and a suntan,** as well as redness and sunburns.
- **Intensity varies** by season, time of day, altitude and environment.
- Help in production of **vitamin D.**

New rules out in October

- The FDA is expected to release **new sunscreen rules** that would include standards for protection against UVA rays. Products would be gauged using **a 1-4 star-rating system,** with one star the lowest and four the highest. Products without a star would be marked, 'No UVA protection.'
- The new rules would also **cap SPF ratings** at 50+.
- The terms **'waterproof' and 'sunblock' would be disallowed** to avoid misleading consumers.

Sources: The Skin Cancer Foundation and American Academy of Dermatology

Ultraviolet (UV) radiation

- UV radiation is part of the natural energy produced by the **sun**. **Tanning beds** also emit UV radiation.
- On the electromagnetic spectrum, UV light has shorter wavelengths than visible light, so your eyes can't see UV, but your skin can feel it.
- Two types of UV light are proven to contribute to the risk for skin cancer:
- **Ultraviolet A** (UVA) has a longer wavelength, and is associated with skin **aging.**
- **Ultraviolet B** (UVB) has a shorter wavelength and is associated with skin **burning**.
- **A majority of nonmelanoma skin cancers** (NMSC) and a large percentage of melanomas are associated with **unprotected exposure to UVA and UVB radiation** which damages the DNA in skin cells, causing **premature aging and skin cancer.**
- **UV rays alter a gene** that suppresses tumors, raising the risk of sun-damaged skin cells developing into skin cancer.

Ultraviolet B radiation

- **UVB** penetrates and damages the outermost layers of your skin. Overexposure causes suntan, sunburn and, in severe cases, blistering.
- **UVB** is connected to the Sun Protection Factor (SPF) on labels of products. The SPF number tells you how long the sun's radiation (including some of the UVA) would take to redden your skin when using that product compared to the time without sunscreen. **Recommendation: >/=spf 30**
- **UVB** intensity fluctuates, strongest and posing the highest risk late-morning to mid-afternoon from spring to fall in temperate climates ; greater timespans in tropical climates. UVB rays can damage your skin year-round, especially at high altitudes or on reflective surfaces like snow or ice.
- **UVB** rays can be filtered and do not penetrate glass.

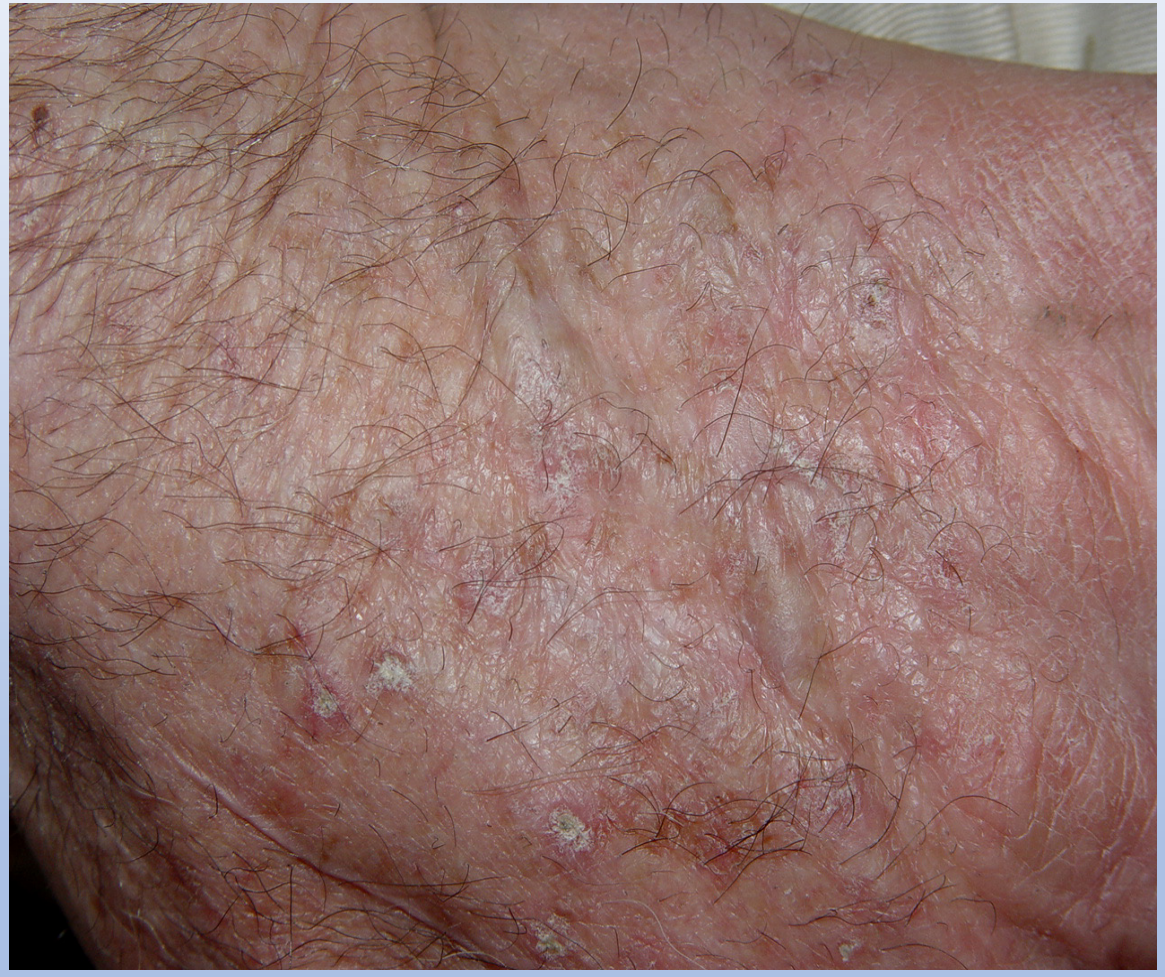
Ultraviolet A radiation

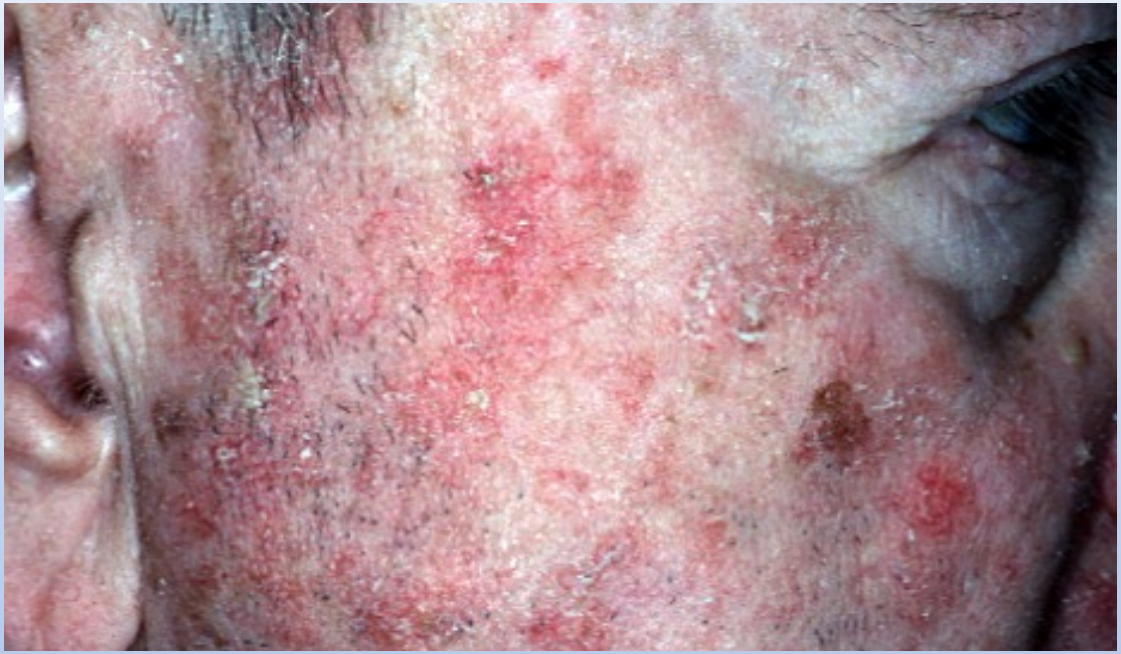
- **UVA** rays cause tanning and the shorter wavelengths of UVA also cause sunburn. There is no such thing as a safe or healthy tan.
- **UVA** is connected to the **“broad-spectrum protection”** you see on the labels of sunscreen products. Early sunscreens only protected against UVB rays, but once understood how dangerous UVA rays were, sunscreen manufacturers added ingredients to protect against UVB and UVA.
- **UVA** rays, while slightly less intense than UVB, **penetrate skin more deeply** Exposure causes genetic damage to cells on the **basal layer** of skin, where most skin cancers occur. The skin tries to prevent further damage by darkening, resulting in a tan.
- **UVA** radiation is the main type of light used in most tanning beds.

Ultraviolet light damage

- **Damage from UV exposure is cumulative** and increases skin aging and skin cancer risk over time. While the body can repair some of the DNA damage in skin cells, it can not repair all of it. The **unrepaired damage** builds up over time and triggers mutations that cause skin cells to multiply rapidly, potentially leading to skin cancers.
- **The degree of damage** depends on the **intensity** of UV rays and the length of time the skin has been exposed without protection. **Location** is also a factor. If you live where the sun is strong year-round, your exposure level and risk increases.

Actinic keratoses



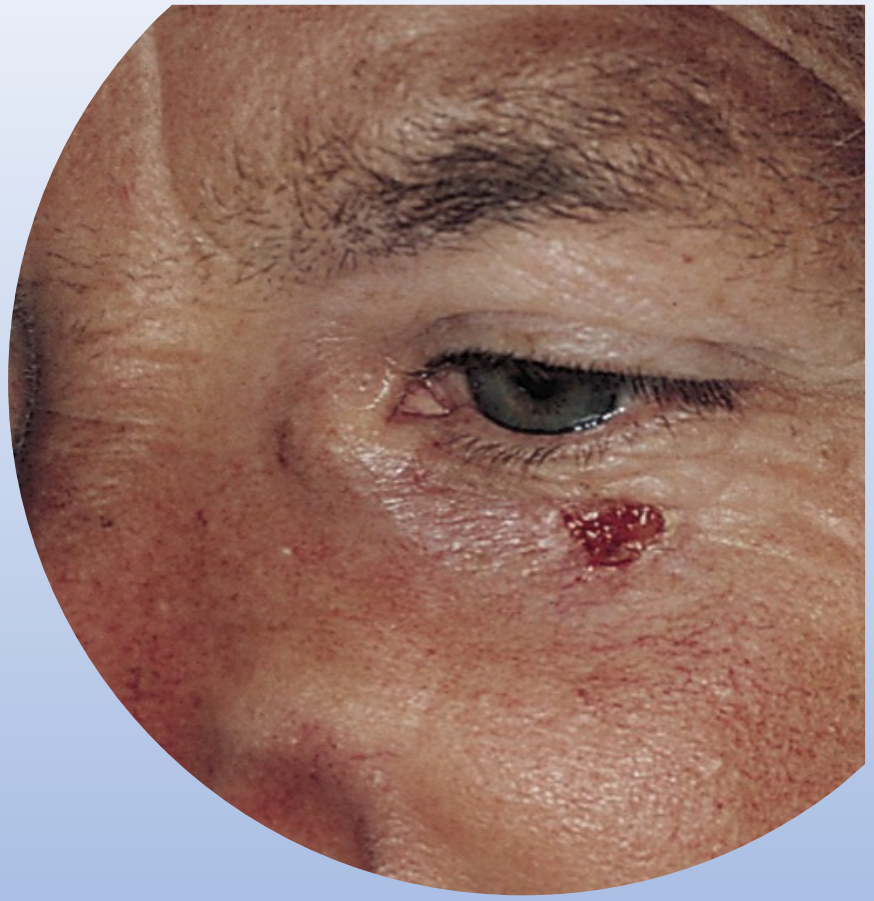


Actinic keratoses

- Actinic keratoses (AKs) are important markers of UV-induced photodamage
- Their presence generally signals an increased risk of NMSC
- Untreated lesions have up to a 20% risk of progression to squamous cell carcinoma.
- Treatments include LN2, topical agents, photodynamic therapy (PDT) and surgery

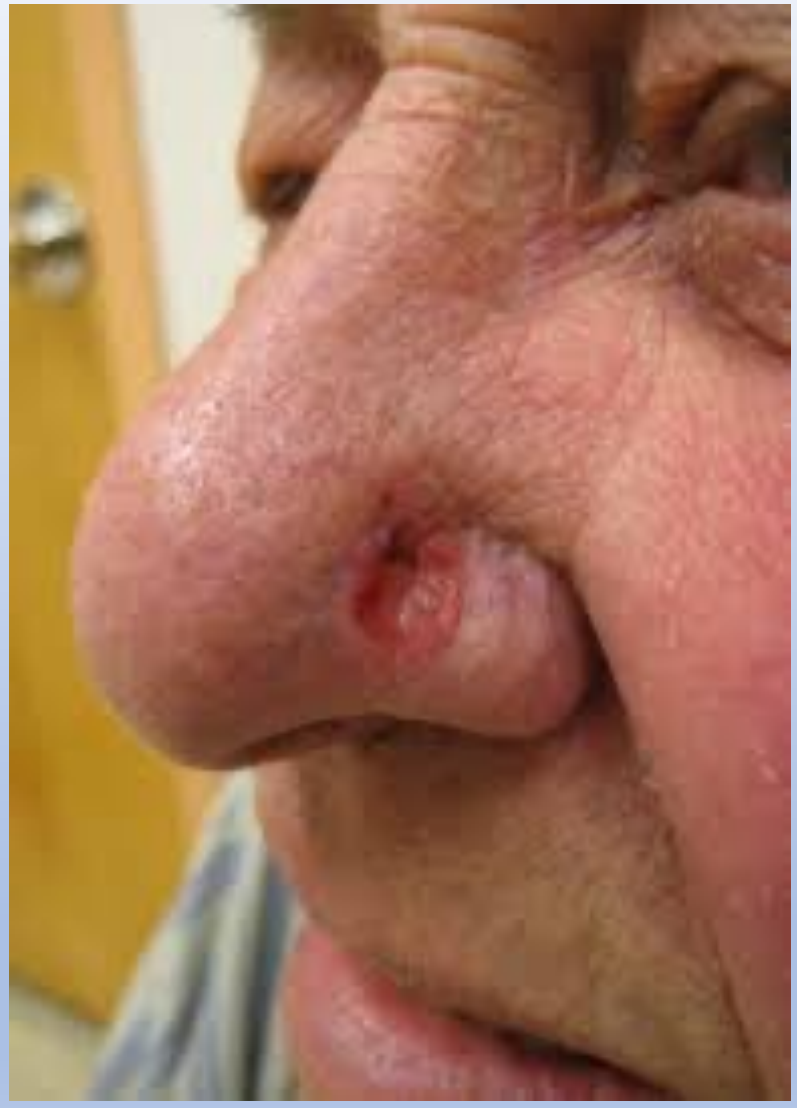
Epidemiology – non-melanoma skin cancer

- Like its more serious counterpart, non-melanoma skin cancer is related to sun exposure.
- Unlike melanoma, however, ***chronic sun exposure*** is believed to play a greater role in the formation of non-melanoma skin cancers than intermittent sun exposure
- **Photo-protective measures**, such as avoiding the sun during the peak midday hours, wearing sunscreen and sun-protective clothing, and avoiding tanning bed use, are believed to decrease the incidence of non-melanoma skin cancers.



Incidence – non-melanoma skin cancer

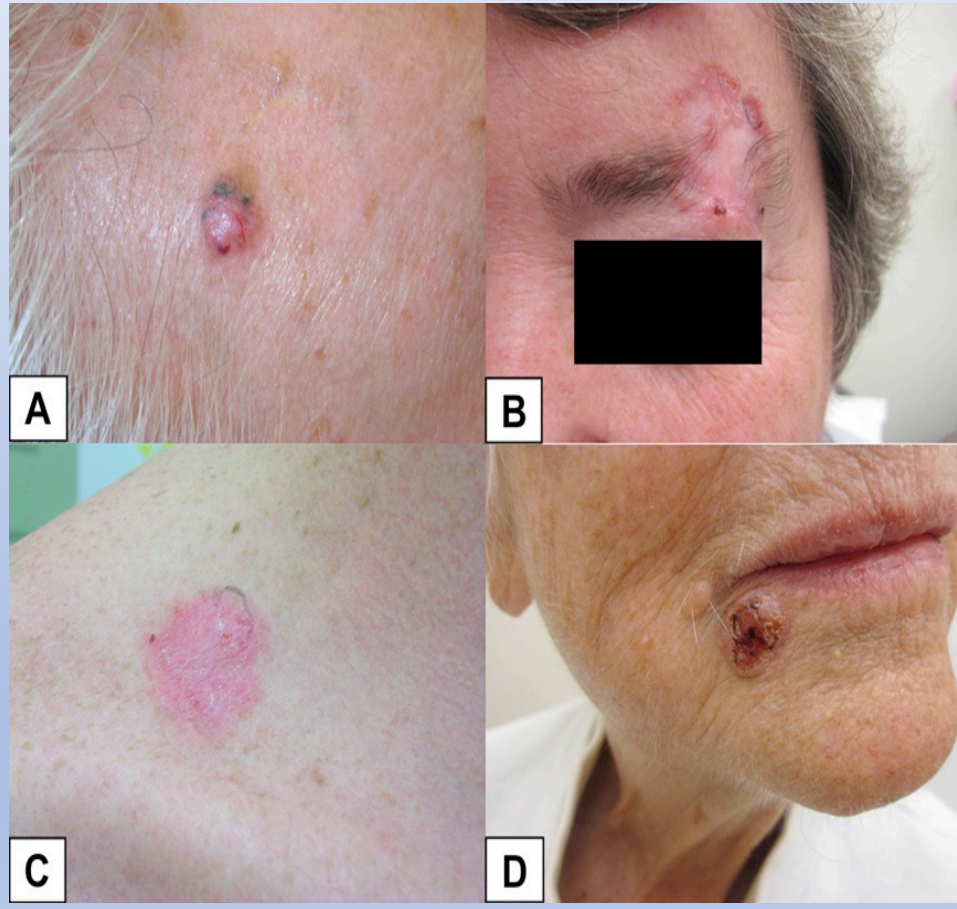
- More than 2 million new cases of non-melanoma skin cancer (NMSC) occur annually in the United States
- ~80% basal cell carcinomas (BCCs), 20% squamous cell carcinomas (SCCs), and a few rarer types
- Incidence is increasing 2% to 3% per year
- Thirteen to 26% of solid organ transplant recipients will develop NMSC within 10 years



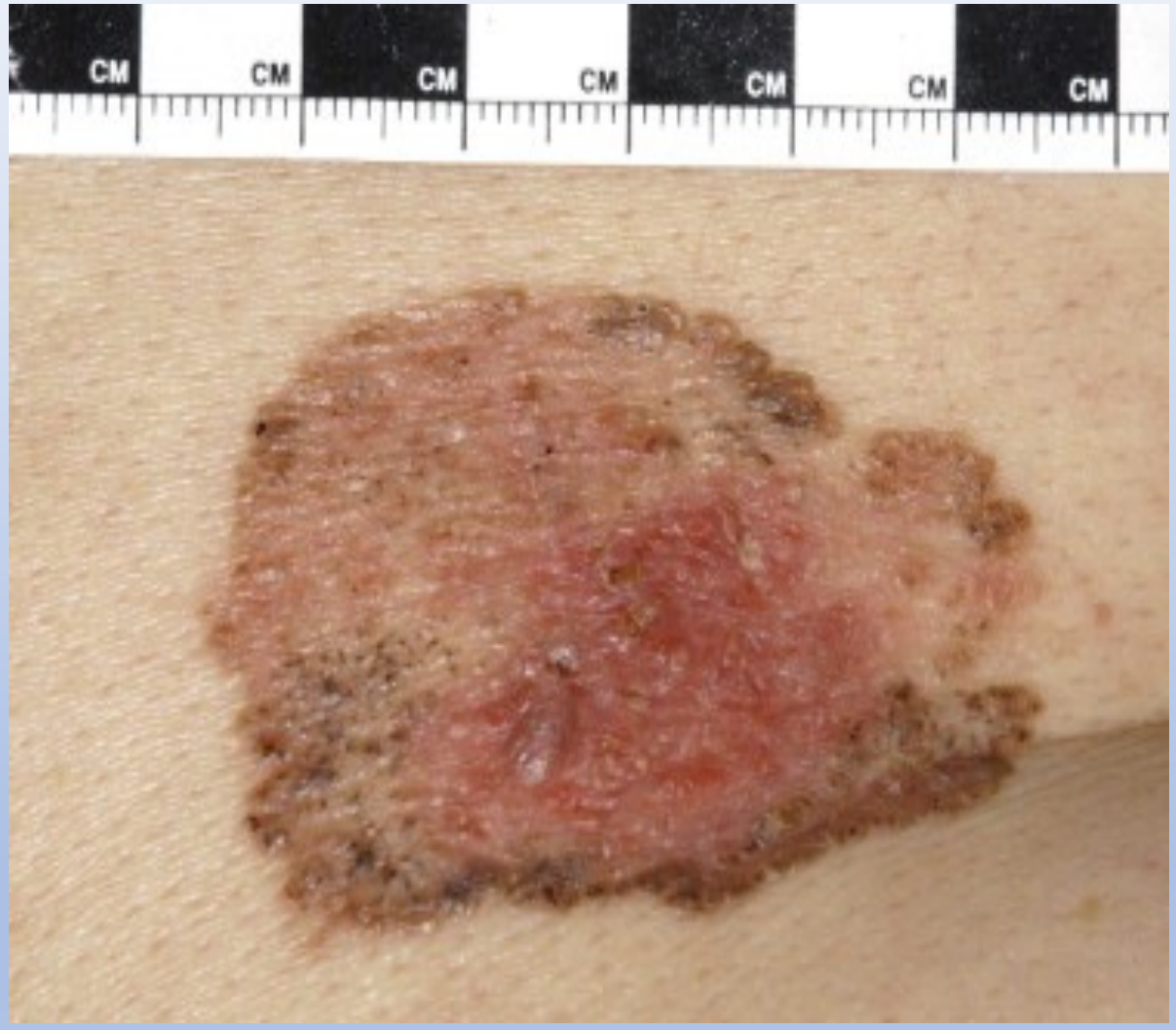
NMSC – basal cell cancer

Clinical:

- Pigmented
- Morpheiform:
  - indurated, resembling scars, may have wide margins histologically, more aggressive
- Superficial Spreading:
  - red, scaly plaque
- Nodular:
  - shiny, pearly, translucent papule with overlying telangiectasias, rolled borders, frequently centrally depressed ulcer

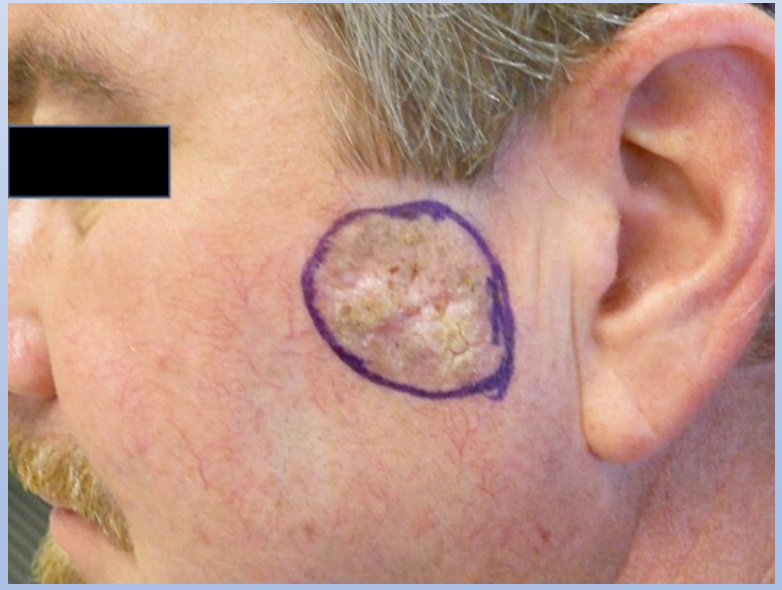


NMSC – pigmented basal cell cancer

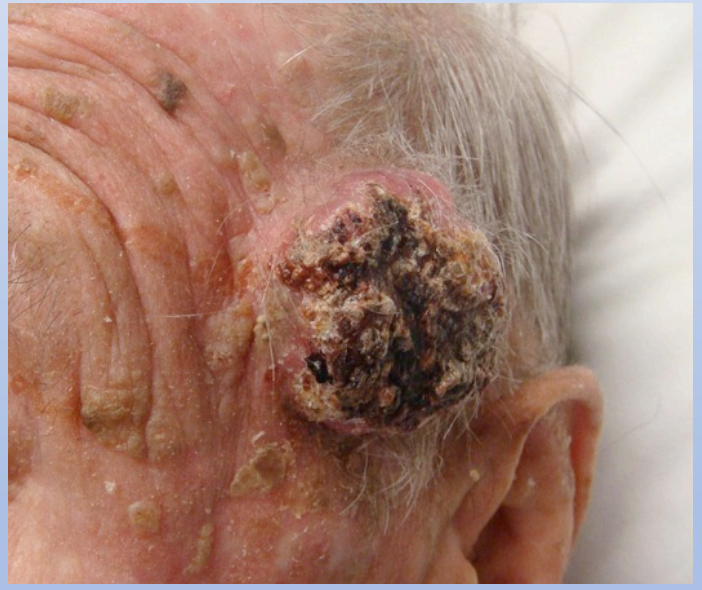


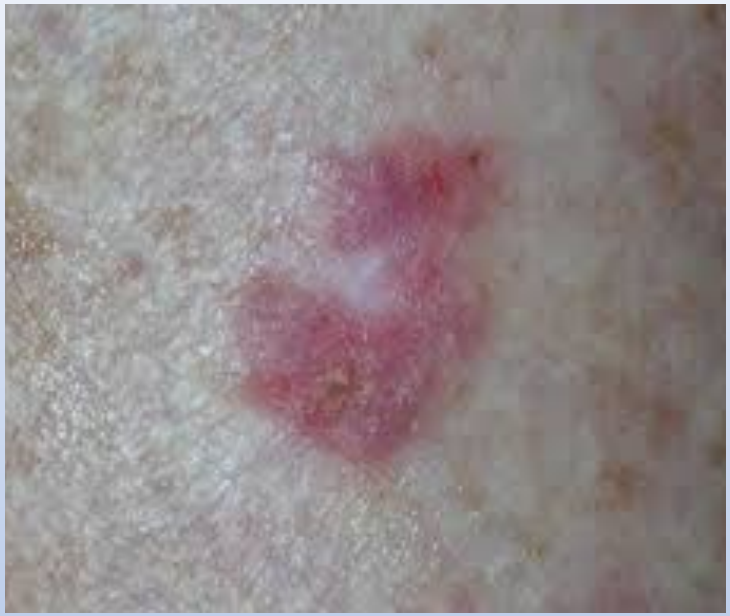
NMSC – squamous cell cancer

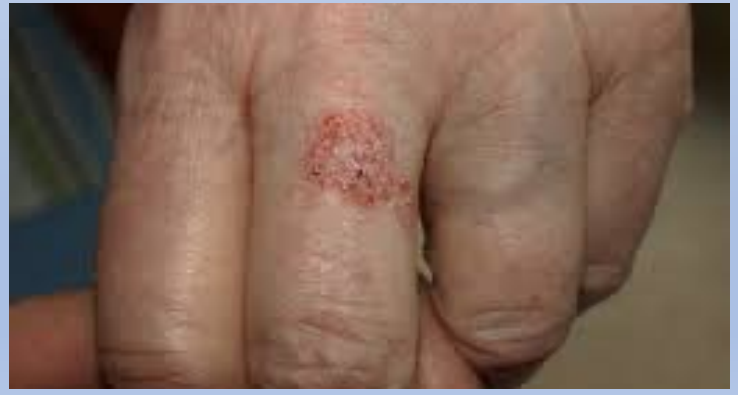
SCC in situ



SCC - invasive







Bowen's disease (SCC in situ)

- Bowen's disease is a common superficial cancer of the skin.
- It appears most commonly as a slow-growing, persistent red scaly patch on areas of skin exposed to the sun.

Treating Bowen's disease

Factors to consider:

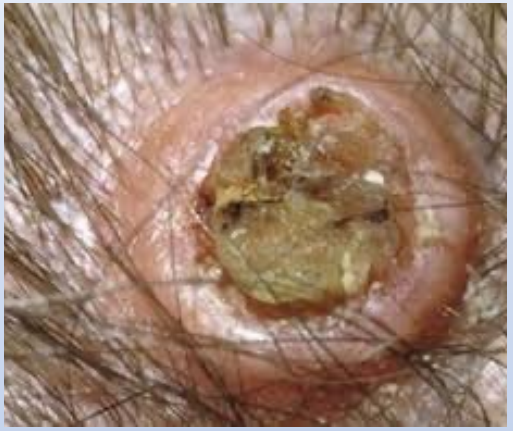
- Size and thickness
- Location
- Age and health of the person
- their preference and ability to undertake the treatment
- Cosmetic concerns
- Previous treatments
- Cost and availability of treatment.

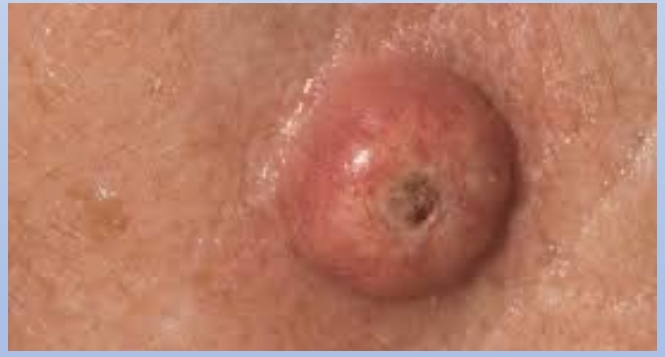
Treatments that may be used:

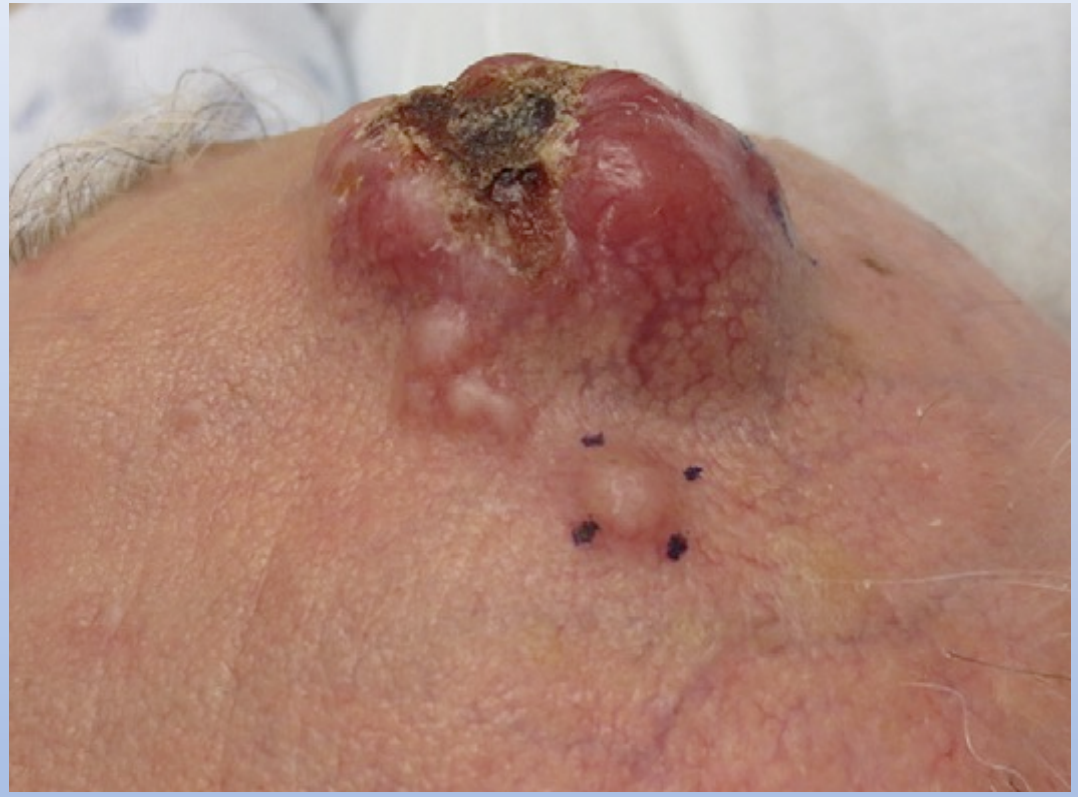
- Cryotherapy or liquid nitrogen
- Topical chemotherapy
  - 5-Fluorouracil; Imiquimod; Ingenol mebutate
- Electrocautery & Curettage
- Photodynamic therapy (PDT)
- Surgery including Mohs micrographic surgery
- Radiotherapy

Keratoacanthoma

- **Keratoacanthoma** is a skin lesion that erupts in sun-damaged skin, like a little volcano.
- It grows for a few months; then it may shrink and resolve by itself.
- **C**onsidered to be a well-differentiated squamous cell carcinoma (SCC).







Staging Evaluation: non-melanoma skin cancer

Diagnosis requires **biopsy**

BCCs that are large, deep, or infiltrative may be locally aggressive and recurrent, but metastasize only rarely (<0.05%)

SCCs have a greater metastatic rate, especially those that are **large, deep,** have **perineural invasion**, or are located on the **dorsal hands, lips, ears, penis**, or **sites of chronic infection**, ulceration, or radiation

TNM staging classifications exist for most types of NMSC and depend on clinical characteristics, pathological features and radiologic evaluation of the primary tumor, adjacent structures, lymph nodes, and viscera

Treatment of non-melanoma cancers

Primary treatment for both BCCs and SCCs is surgical.

Mohs surgery preferred for: face, scalp and ear lesions; ill-defined (infiltrative) & aggressive lesions

Aggressive lesions: large, deep, perineural invasion; lesions located on the dorsal hands, lips, ears, penis, or sites of chronic infection, ulceration, or radiation

Mohs : microscopic tumor margins clearance; tissue-sparing

The 5-year recurrence rate for BCC is 1% for Mohs compared to 5% for other types of surgical excision.

Alternative primary therapies include various forms of physical destruction (EDC x 3; cryotherapy), photodynamic therapy (PDT) and radiation therapy.

Topical agents

Systemic agents

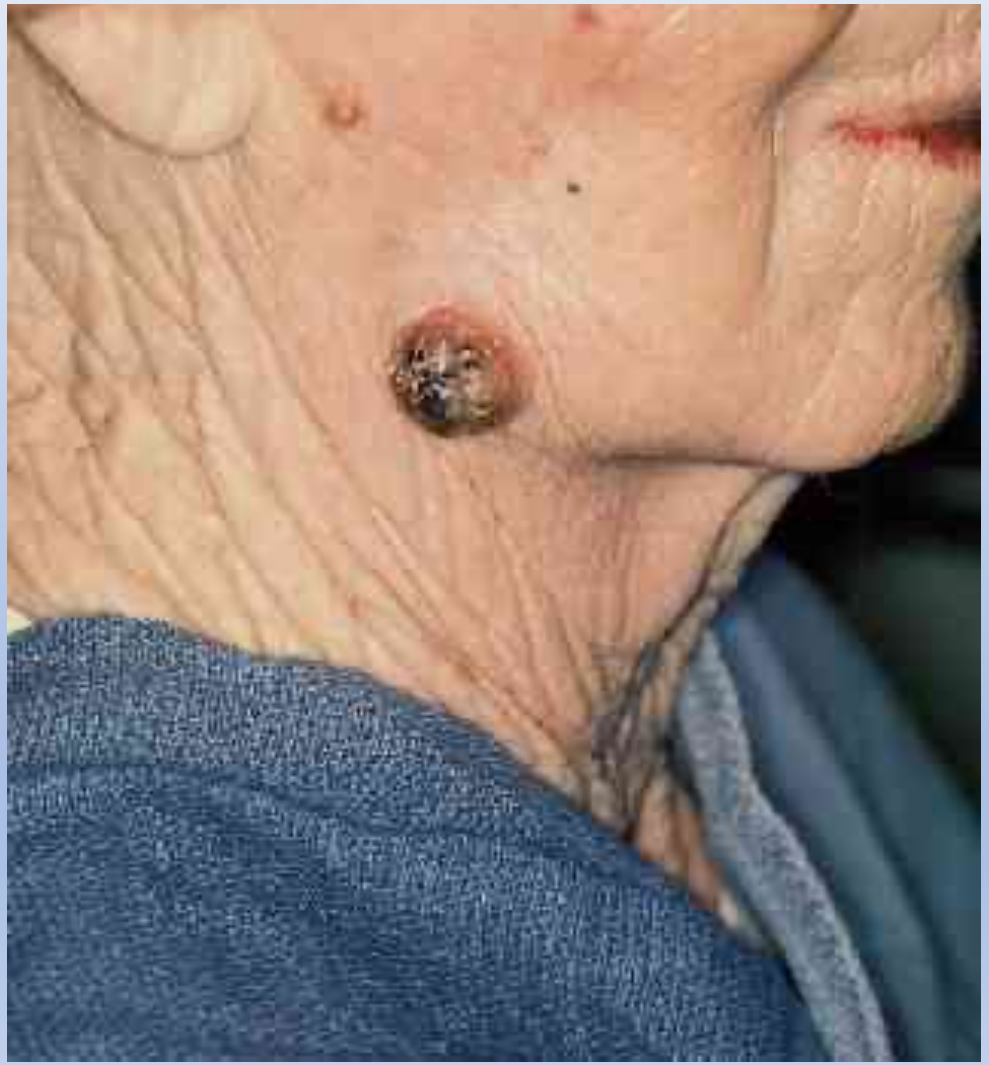
Non-melanoma skin cancers – Treatment of locally advanced and metastatic disease

- Local recurrence is a problem for large, deep, or histologically infiltrative variants. SCCs with these features may also metastasize. The 5-year survival for patients with metastatic SCC is <50%.

- Combinations of surgery, radiation therapy, and chemotherapy can be used for metastatic disease.

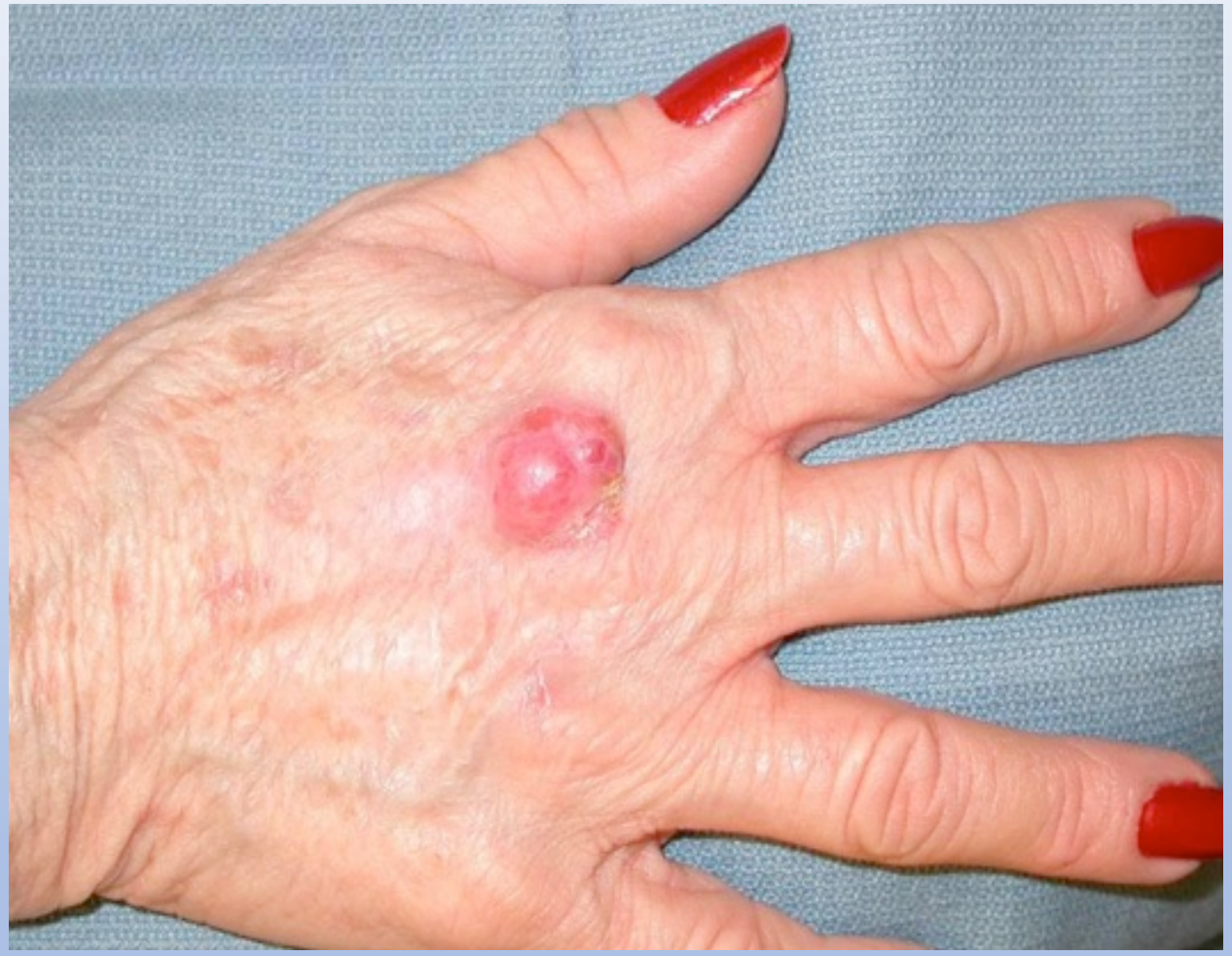
- The rarer forms of non-melanoma skin cancer have a significantly more aggressive clinical course as compared with BCC and SCC. These include, Merkel cell carcinoma, sebaceous carcinoma and atypical fiboxanthoma.

Merkel Cell Carcinoma



- Usually 60-80 y/o age group
- most common in fair skin
- Equal male : female
- Extensive sun exposure history
  - Chronic immune suppression
  - Feng, 2008 - Merkel cell polyoma virus
- Papules or nodules, pink to red to violet in color, often with overlying telangiectasia; typically, < 2 cm
- Most common sites: head and neck (49%), extremities (38%) – lower > upper; trunk (13%), mainly lower back and buttocks

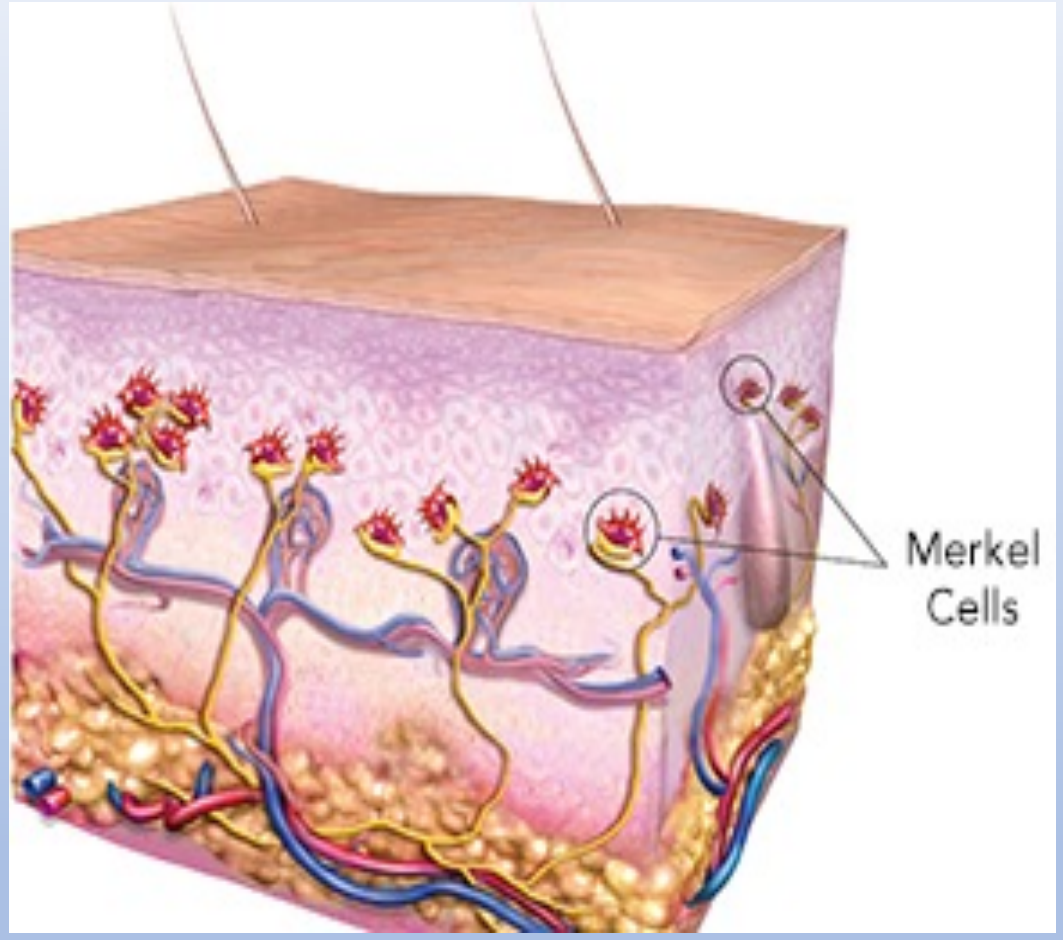
Merkel Cell Carcinoma



The Merkel Cell

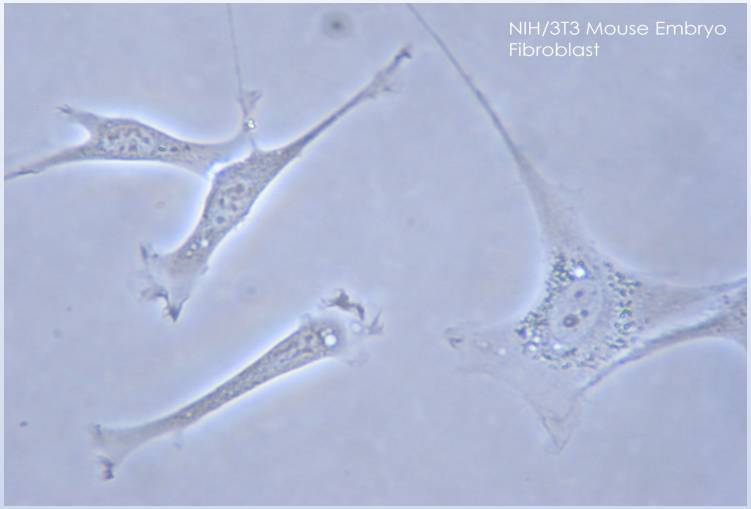
Normal Merkel cells (red)
connecting to nerves (yellow).

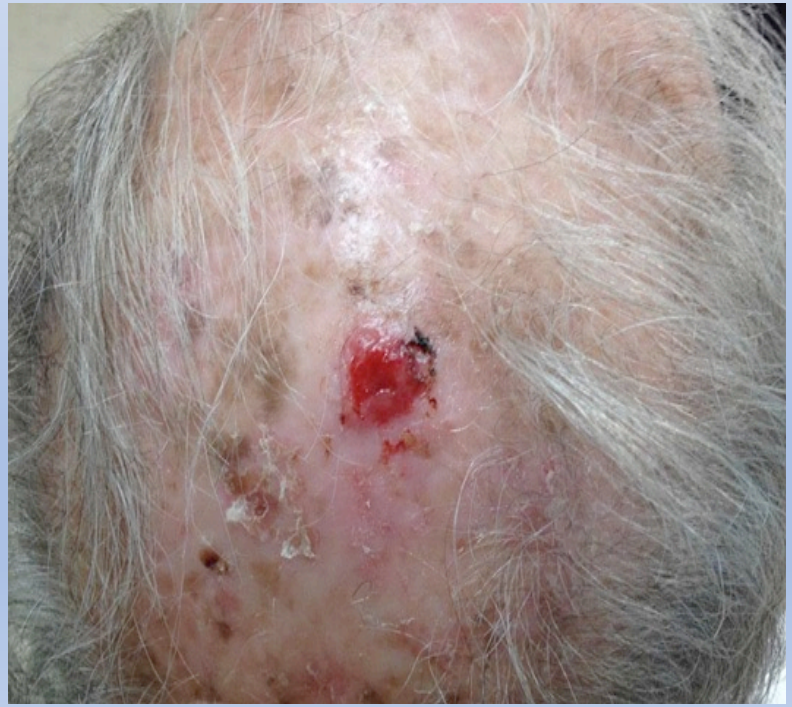
The structures drawn include the epidermis (upper third), dermis (middle), and deeper adipose layer containing the fatty tissue. Arteries are depicted as red and veins are blue.



Atypical fibroxanthoma

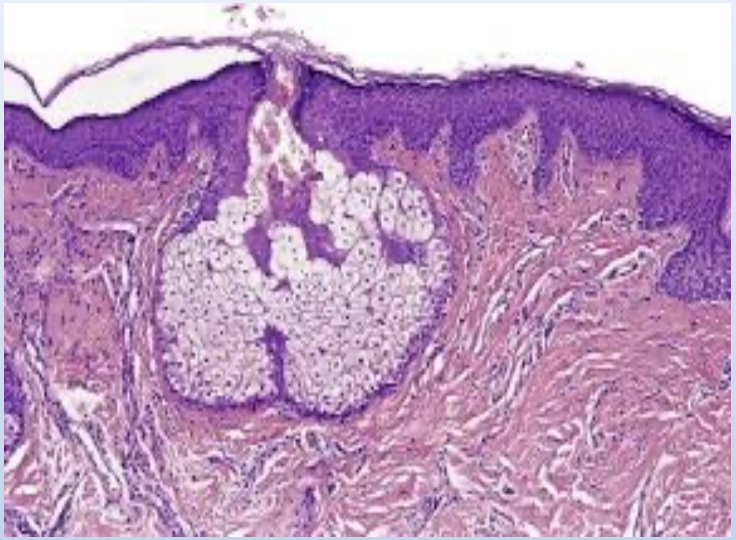
- Atypical fibroxanthoma (AFX) - is a malignancy of dermal **fibroblasts**
- rare spindle cell tumor with intermediate malignant potential
- most commonly affects **elderly white men**
- current case reports estimate an overall **recurrence rate** of approximately 6%.
- microsatellite and in-transit **metastases** reported, resulting in 5 patient deaths.

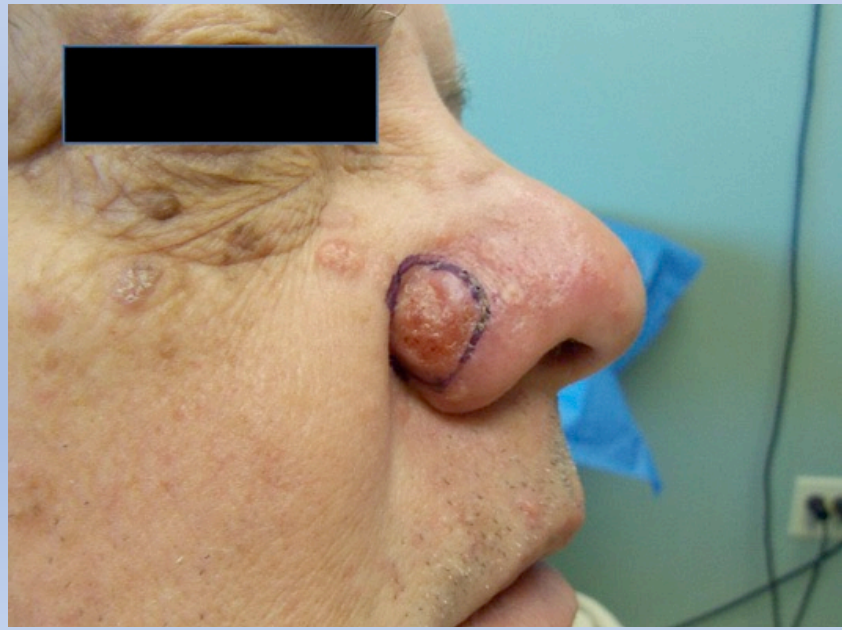




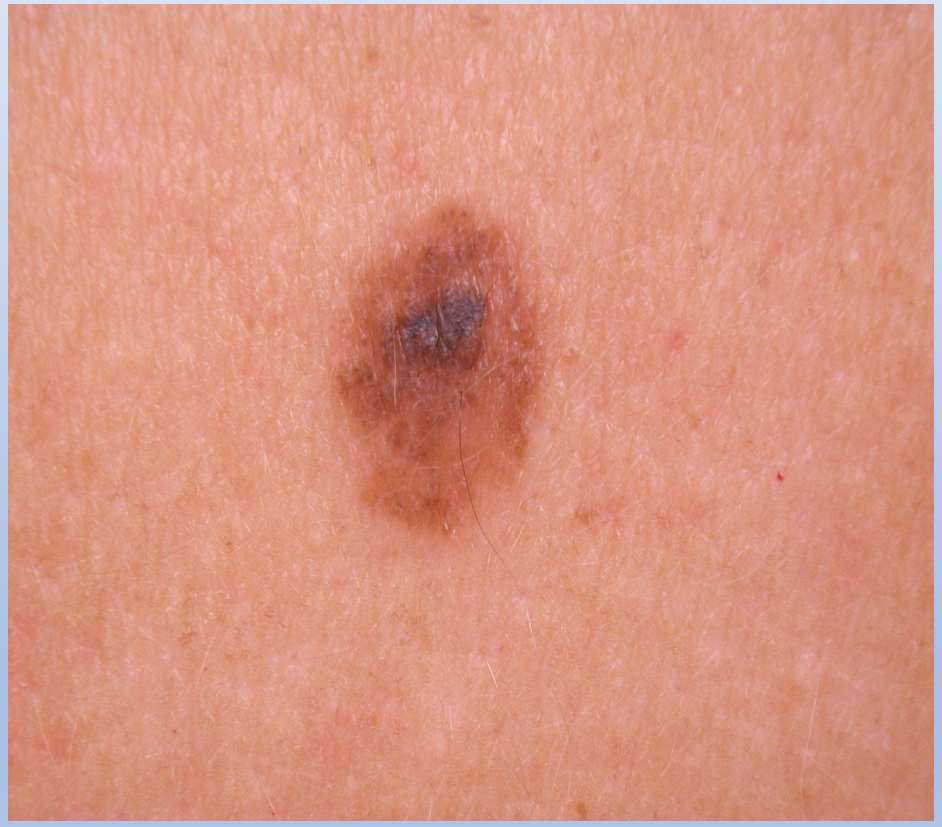
Sebaceous carcinoma

- rare (0.5 cases per million)
- neoplasm of sebaceous cells
- high rate of recurrence and metastasis to regional lymph nodes and distant organs.
- mortality - 9% to 50%, depending on disease progression at time of diagnosis.
- risk factors: older age - at dx median 72; female sex (73%)
- most arise de novo; some in preexisting sebaceous lesions

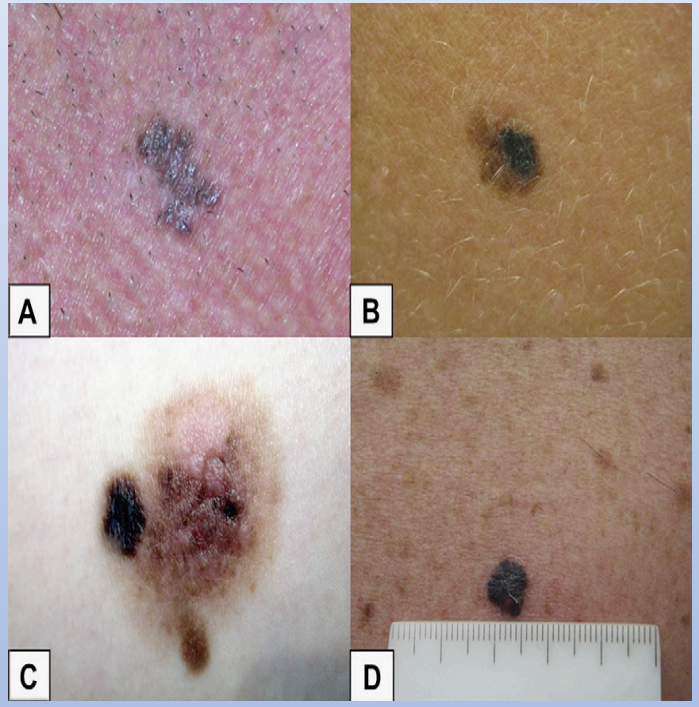


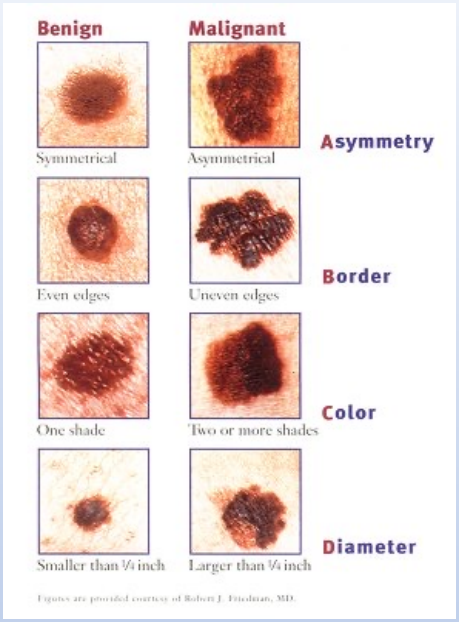


Atypical (dysplastic) nevus



- Atypical Nevus
  - irregular outline, pigmentation, borders, maybe >6mm, some have a “fried-egg” appearance: dome-shaped central brown papular component w/ zone of lighter surrounding pigmentation
  - dysplastic nevus syndrome, correlate with FH





Melanoma skin cancer: A B C D plus E (evolution)

Dermoscopy

- A technique that can aid in the evaluation of a new or changing pigmented lesion
- Dermoscopy is the use of magnification and polarized light to examine a patient's lesion for findings characteristic of a benign or malignant process.
- Can be helpful in assessing whether or not further evaluation is warranted, but is not a substitute for histopathologic examination
- Skin biopsy remains the gold standard for the diagnosis of melanoma.

Melanoma - epidemiology

~ 68,000 new cases of cutaneous melanoma in the US each year

- Lifetime risk: 1 in 75 and rising
- 70% de novo, 30% from nevus/dysplastic nevus
- 30% from nevus/dysplastic nevus

Risk factors:

- Personal history of melanoma – 12-fold higher risk
- family history of melanoma
- Prior blistering sunburns
- Fair skin
- >50 common nevi
- Atypical nevi
- Parkinson's disease – 4-fold higher risk

Melanoma subtypes

SUPERFICIAL SPREADING (most common); **NODULAR** (more invasive)

- Associated with lighter skin, upper back in men, thighs/lower legs in women
- Risk factors: family hx locations: volar skin of palms/soles, nailbeds, sun-exposure, dysplastic nevus syndrome

LENTIGO MALIGNA

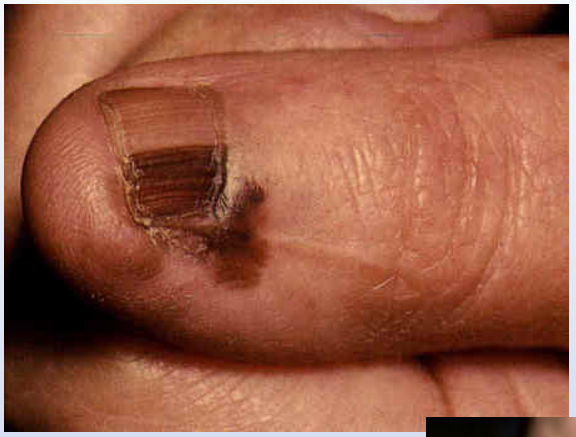
- 5% of all melanomas locations: face, scalp, extensor forearms/upper trunk, sun-exposed areas

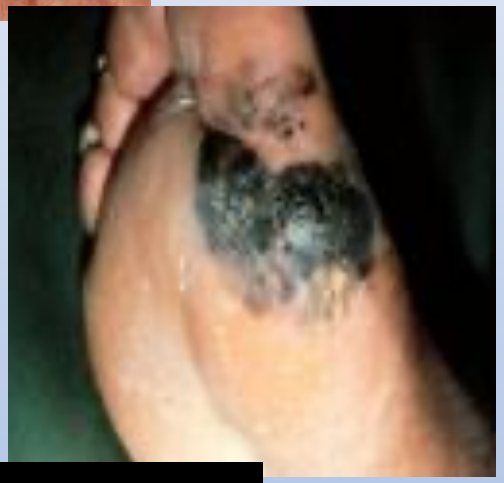
ACRAL LENTIGINOUS

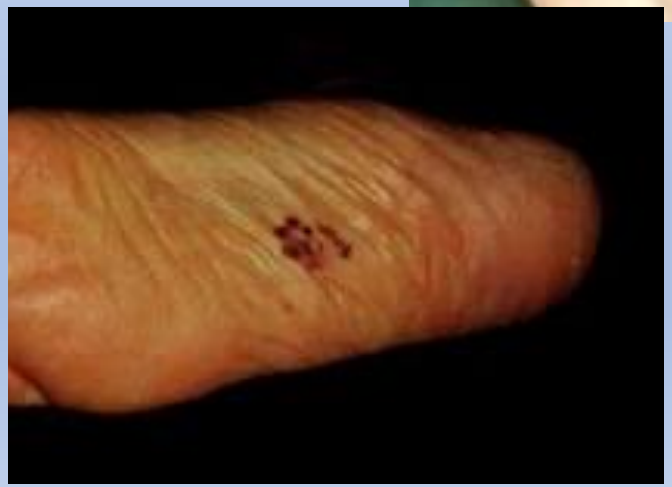
5% of total melanomas, but 30-70% of melanomas in Blacks, Asians, & Hispanics ;

1-9% of Whites; locations: volar skin of palms/soles, nailbeds;

not associated with sun



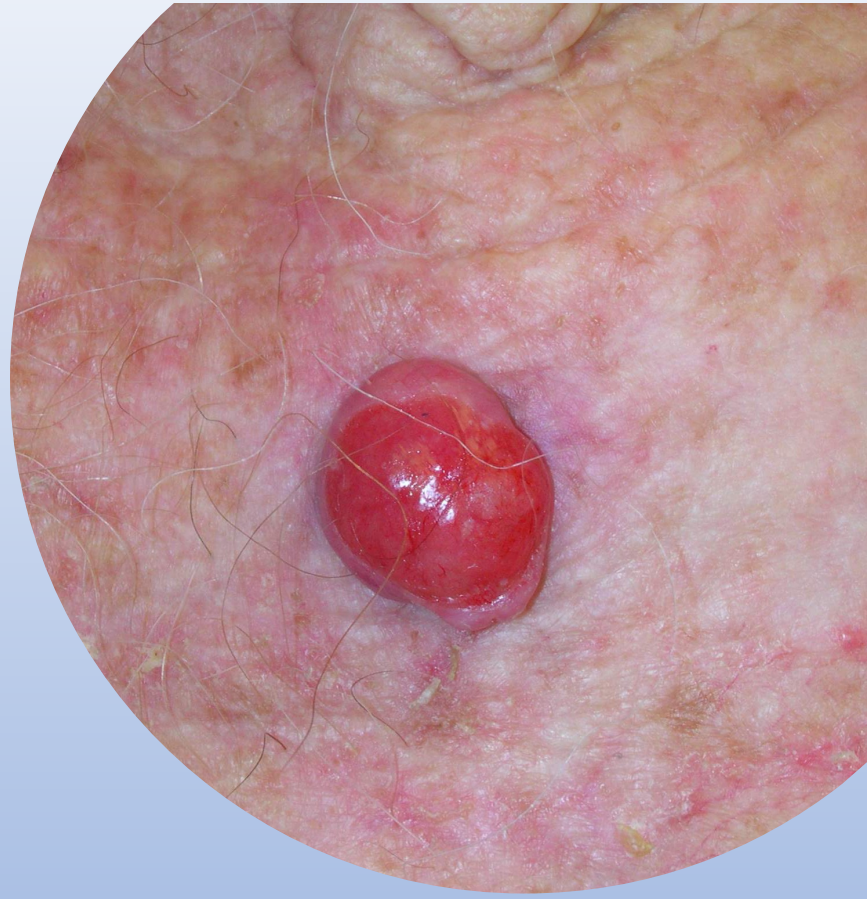




Acral melanomas

Amelanotic melanoma

- Amelanotic melanomas represent 8% of all melanomas
- They can lack classical features of other melanomas and tend to be red or skin colored and more symmetrical
- They might be confused with basal cell or squamous cell carcinomas, pyogenic granulomas, or benign acral nail lesions, depending on their appearance



Treatment

Breslow thickness most important prognosticator	Margin width at excision
in situ – 0 mm	5 mm margin
Invasive: Thin – </=1 mm	1 cm margin
Invasive: Intermediate – 1.01-4mm	2 cm margin plus SLN biopsy; if positive, lymphadenectomy. Maybe systemic and or radiation.
Invasive -Thick - >4mm	Same as intermediate

Melanoma 5-year survival rate

For early-stage melanoma that is only located near where it started, the 5-year survival rate is **98%**.

The survival rate for melanoma that has spread to the nearby lymph nodes is **63%**.

If it has spread to other parts of the body, the survival rate is **17%.**

However, survival rates vary depending on a number of factors

(Cancer.net)

Prevention

Continued monitoring by dermatology

- Individuals with a history of melanoma have 12 times the likelihood of developing a second primary lesion compared with patients without melanoma
- LDH – non-specific – but can be used to monitor for recurrence or upstaging
- CXR or CT for patient-specific symptoms; PET for accurate staging

Patients should

- Do self exams
- wear sun-protective clothing
- Apply a daily broad-spectrum sunscreen of sun protection factor of 30 or greater to exposed areas
- Avoid tanning (outdoors and at tanning facilities

Screen immediate family members

- They may have similar phenotype, genetic risk (although most melanomas are sporadic vs genetic mutation), or exposure practice

Skin Cancer - Public Health Considerations

Skin cancer risk perceptions: A comparison across ethnicity, age, education, gender, and income

Buster et al

J ACAD DERMATOL
VOLUME 66, NUMBER 5
May, 2012

- Minorities and the socioeconomically disadvantaged have increased morbidity and mortality from melanoma and nonmelanoma skin cancer (SC).
- SC risk misperceptions are more prevalent in groups with poorer SC outcomes. This indicates that awareness and knowledge may affect the presenting stage of SC.
- There is a need for public education directed toward not only those at greatest risk for SC, but also toward those at risk for high morbidity and mortality.

Skin cancer surveillance behaviors among US Hispanic adults

J AM ACAD DERMATOL
Vol 68, No 4 *Coups et al*

Efforts are needed to promote skin cancer surveillance behaviors among at risk Hispanics.

Few Hispanic adults have ever conducted a skin self-examination or received a total cutaneous examination from a health professional

Engagement in these behaviors is particularly low in several subgroups, including those who are acculturated to the Spanish language and individuals lacking a physician recommendation.

Clinical Characteristics and Awareness of Skin Cancer in Hispanic Patients

Dermatology Online Journal U C Davis, 19(9) Javed et al, 2013

Findings:

- a lower incidence of AKs in Hispanic skin cancer patients as compared to their age matched non-Hispanic Caucasians.
- Skin malignancies in Hispanics present at a more advanced state and there is usually a lack of awareness in such cases.
- Patient knowledge and education is crucial for early detection and prevention of skin cancer in the Hispanic population.

Social Determinants of Racial and Ethnic Disparities in Cutaneous Melanoma Outcomes

Harvey et al, *Cancer Control*. 2014 October ; 21(4): 343–349.

African Americans and Hispanics diagnosed with cutaneous melanoma are more likely to present with more advanced stages of disease at diagnosis and have higher rates of mortality than their nonminority counterparts.

These disparities may be a consequence of economic, social, and cultural barriers such as low income, public forms of health insurance, lower levels of education, lower levels of melanoma awareness and knowledge, and lower rates of participation in melanoma screening.

It takes a village …

- The patient
- Hairdressers
- Massage therapists
- Physical therapists
- Primary care, dermatology, surgery
- Skin cancer incidence rising and patients living longer - epidemic

A Pragmatic Approach to Melanoma Screening in Collaboration With Primary Care Providers

Editorial - *Cutis.* 2016 June;97(6):382-383

There is abundant anecdotal evidence of the value of skin cancer screening; however, population-based screening performed exclusively by dermatologists is not practical.

As health care payment and delivery models evolve, there is greater emphasis on outcomes and team-based care.

The authors believe that skin cancer screening by primary care physicians (PCPs) will allow for the development of effective teams of PCPs, dermatologists, and other experts in melanoma, public health, and informatics to reduce melanoma mortality in a cost-effective manner.

POST-TEST

What are the two big bucket categories of skin cancer?

Which demographic has the most skin cancer?

Which skin cancer accounts for the highest morbidity and mortality?

What is the most common skin cancer? Second most common?

What is the increased likelihood of individuals with a h/o malignant melanoma (MM) having a 2nd primary lesion vs those w/o MM hx?

What is the most effective way to reduce skin cancer risk?

What is the gold standard for treatment of most skin cancers?

What is the most important prognostic indicator of MM?

Which demographic presents with more advanced melanoma at dx?

What is the minimum spf (sun protection factor) recommendation?