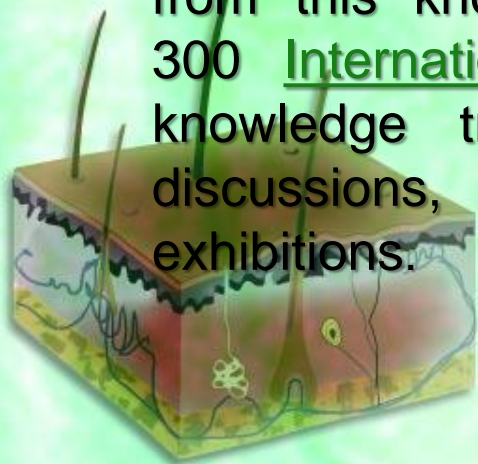


About OMICS Group

OMICS Group International is an amalgamation of Open Access publications and worldwide international science conferences and events. Established in the year 2007 with the sole aim of making the information on Sciences and technology 'Open Access', OMICS Group publishes 400 online open access scholarly journals in all aspects of Science, Engineering, Management and Technology journals. OMICS Group has been instrumental in taking the knowledge on Science & technology to the doorsteps of ordinary men and women. Research Scholars, Students, Libraries, Educational Institutions, Research centers and the industry are main stakeholders that benefitted greatly from this knowledge dissemination. OMICS Group also organizes 300 International conferences annually across the globe, where knowledge transfer takes place through debates, round table discussions, poster presentations, workshops, symposia and exhibitions.



About OMICS Group Conferences

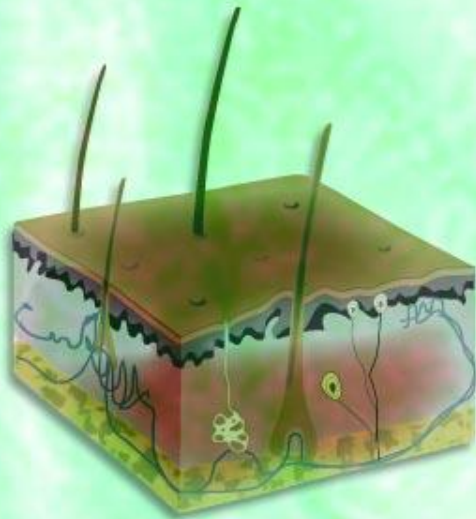
OMICS Group International is a pioneer and leading science event organizer, which publishes around 400 open access journals and conducts over 300 Medical, Clinical, Engineering, Life Sciences, Pharma scientific conferences all over the globe annually with the support of more than 1000 scientific associations and 30,000 editorial board members and 3.5 million followers to its credit.

OMICS Group has organized 500 conferences, workshops and national symposiums across the major cities including San Francisco, Las Vegas, San Antonio, Omaha, Orlando, Raleigh, Santa Clara, Chicago, Philadelphia, Baltimore, United Kingdom, Valencia, Dubai, Beijing, Hyderabad, Bengaluru and Mumbai.

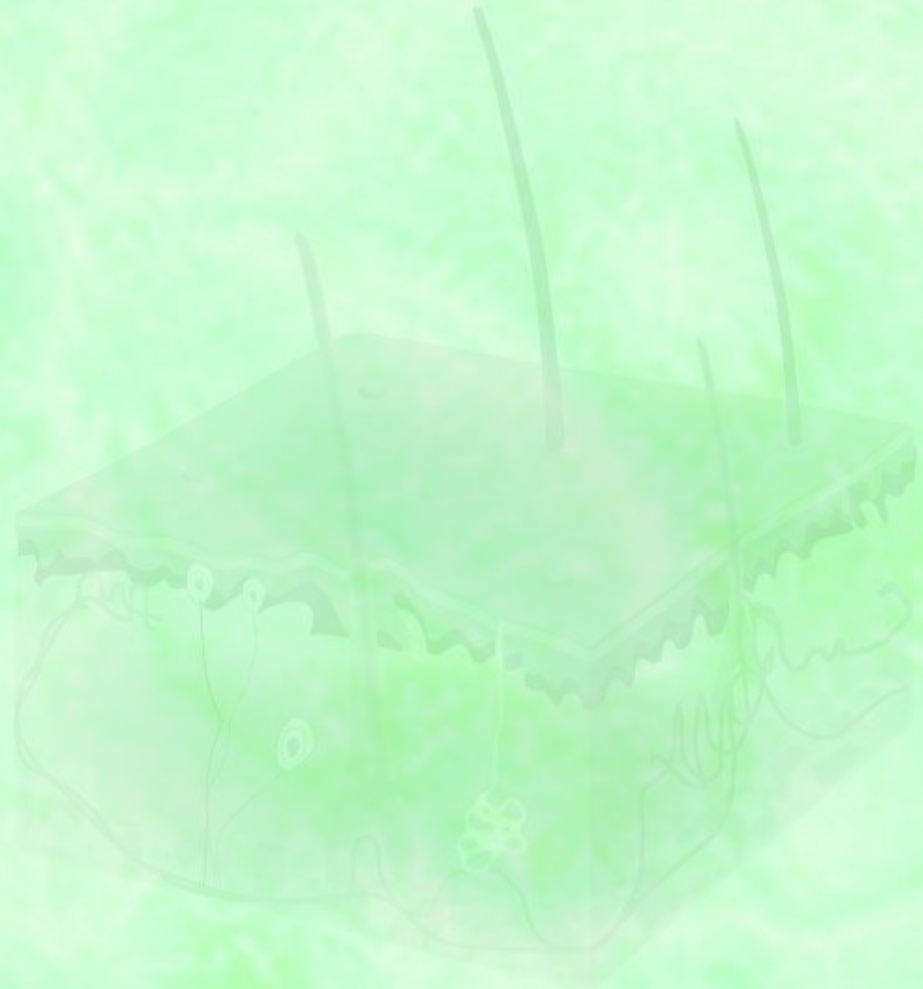
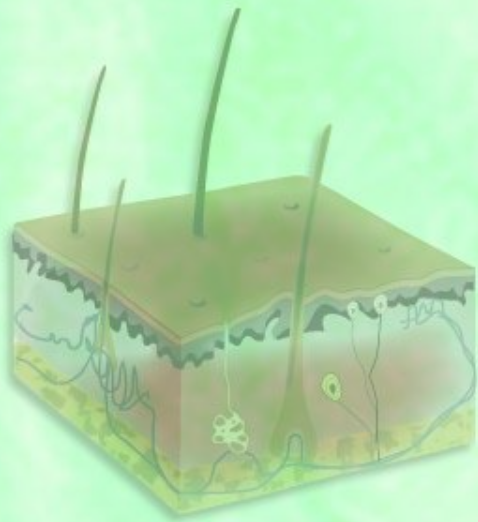


PREMATURE AGING INDUCED BY SUNLIGHT

Dhelya Widasmara



INTRODUCTION



AGING

- A “biological reality” → its own dynamic beyond human control
- Complex process → progressive functional decline due to the accumulation of molecular damage over time
- Human skin like all other organs → chronological aging and aging as a consequence of environmental damage (photoaging)
- Aging skin has a marked susceptibility to dermatologic disorders → structural and physiologic changes → consequence of intrinsic and extrinsic aging



PHOTOAGING

damage to the skin

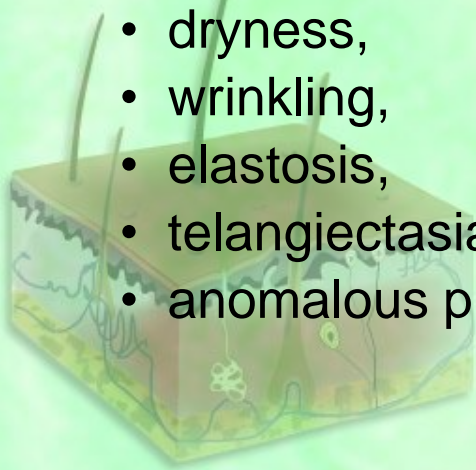
- caused by intense and chronic exposure to sunlight

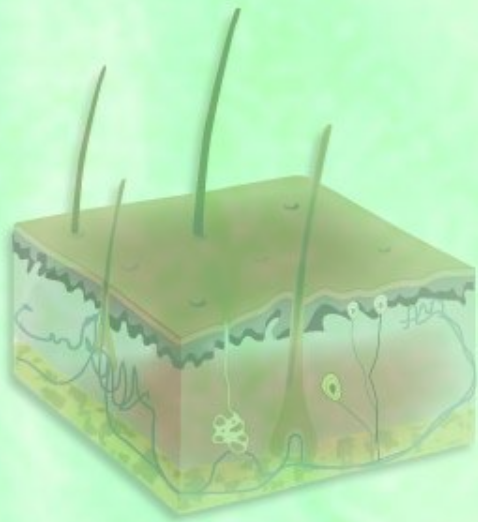
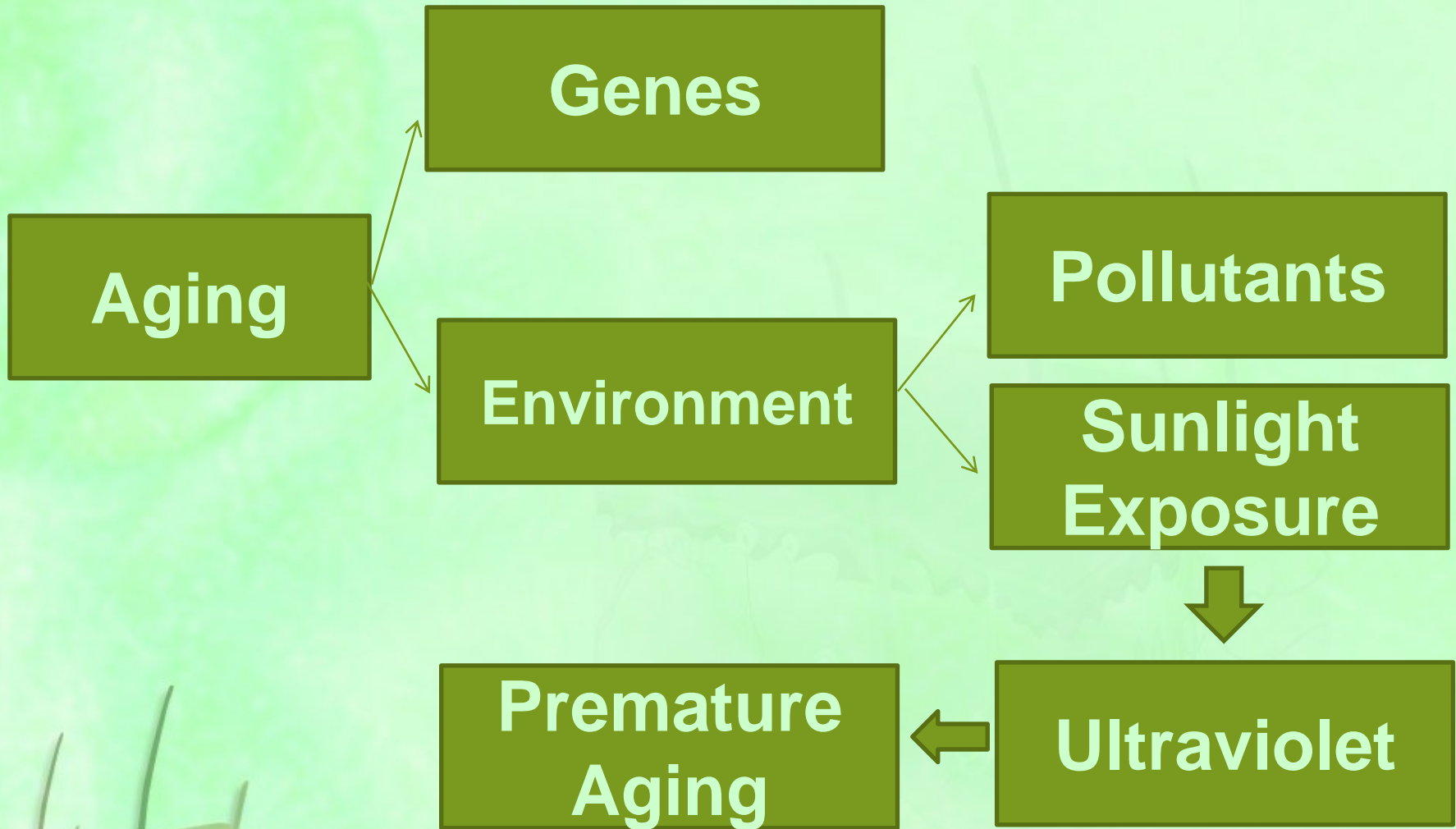
types of aging:

- intrinsic aging
- extrinsic aging

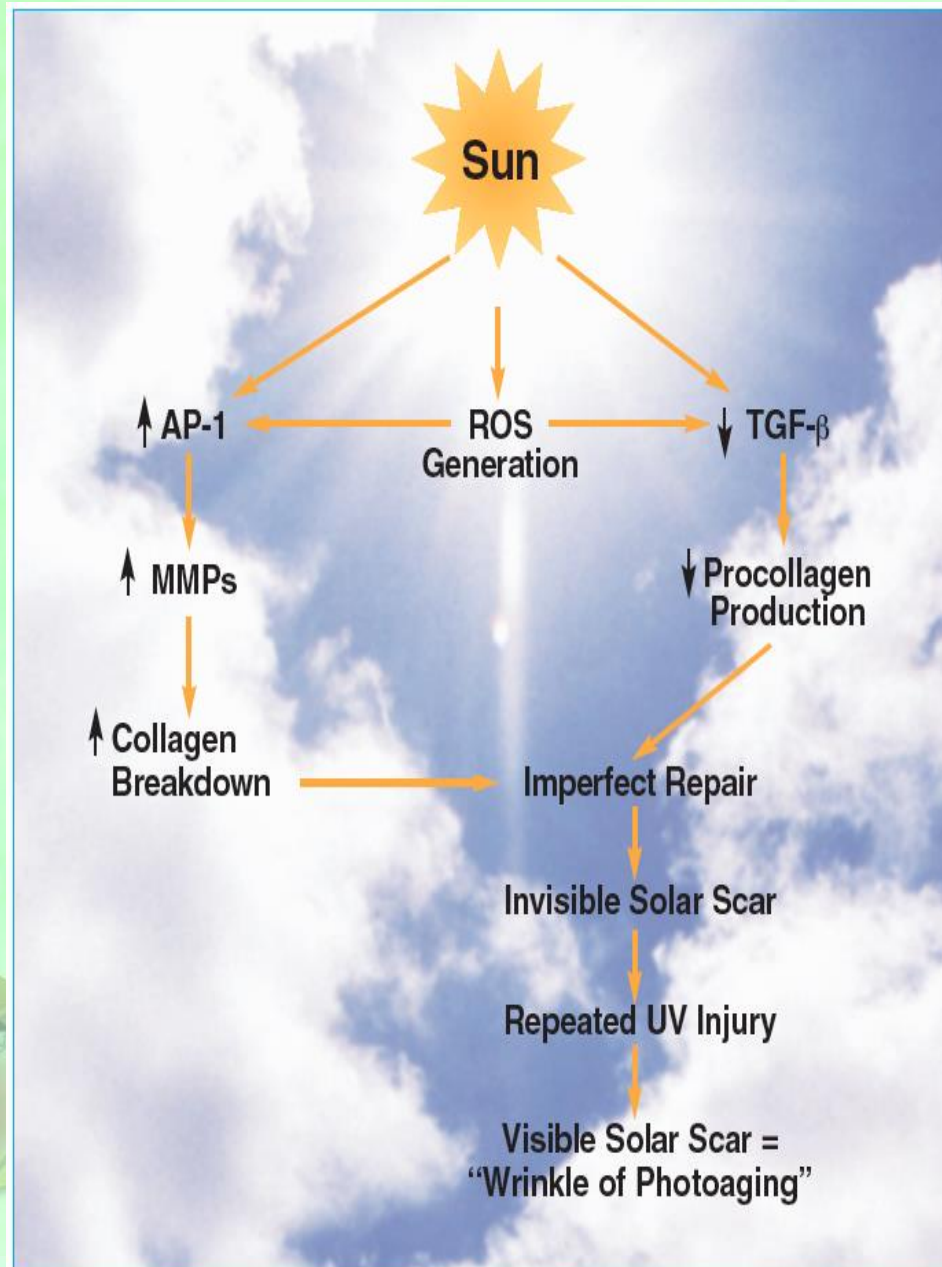
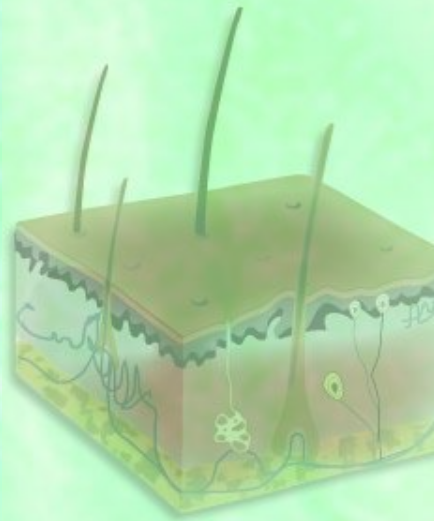
characterized by

- dryness,
- wrinkling,
- elastosis,
- telangiectasia,
- anomalous pigmentation





Mechanism of extrinsic skin photoageing



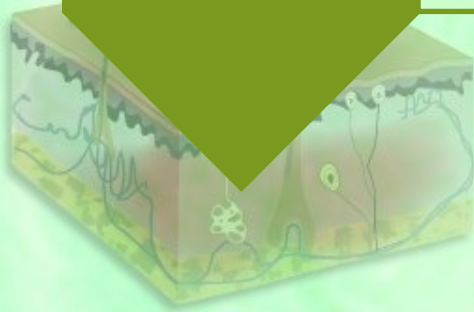
Sunlight and photoaging

Sunlight

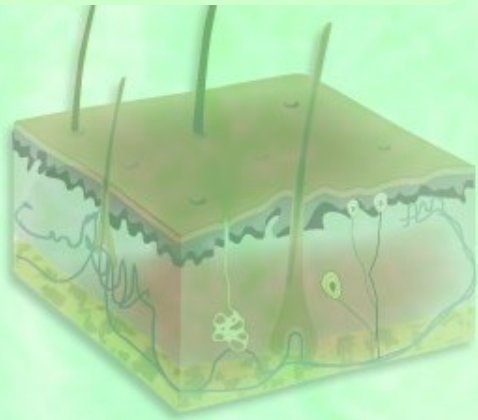
- Ultraviolet B (UVB, 280-315 nm) → cause DNA damage
- Ultraviolet A (UVA, 315-400 nm) → most effective in penetrating skin
- Visible light (400-760 nm)
- Infrared (760-106 nm)

Ultraviolet

- ROS Production → DNA damage, MMP, MAPK, PKC
- Induction various cytokines
- Inhibit skin cell renewal processes



UV radiation



Beneficial effect

Stimulate psychophysical energies, biochemical process, hypophysis & supra adrenal gland

Favor vitamin D synthesis

Production of melanin

Regulation of sebaceous secretion

Negative effect

Premature photo aging & cutaneous cancer

- Duration
- Time
- Gradual character of sun exposure
- Human prototype
- Type of protection used

**SUNLIGHT
EXPOSURES**

UV RADIATION

**SKIN PHOTO
AGING**

- Loss of skin level
- Wrinkles
- Uneven hyperpigmentation
- Skin thickenings
- Skin becomes rough

UV RADIATION

Inflammatory process occurs at skin level

Involvement of dermis layer (monocytes & netrophils)

Free radicals leading to tissues destruction



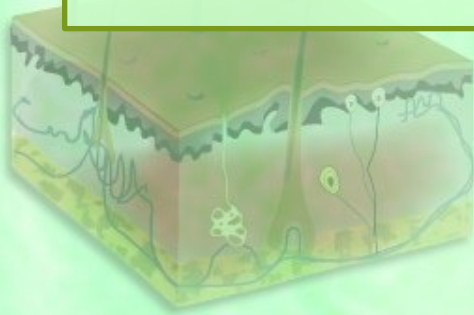
FREE RADICALS

Accumulation → DNA Damage

Modified protein & molecules → destruction skin collagens network (dermis)

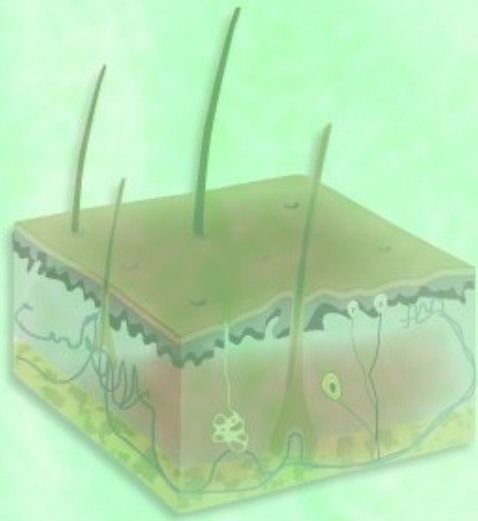
Collagen destruction → Skin becomes fragile

Fine/deep wrinkles, irregular pigmentation, rough, inflamed, telangiectases



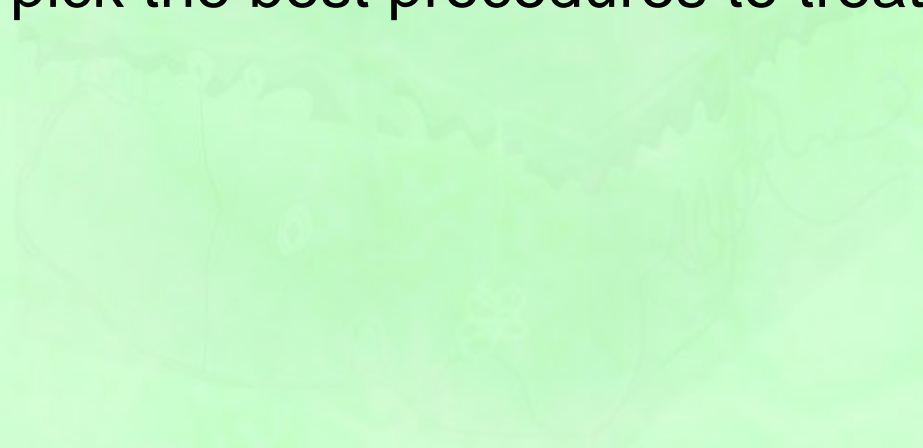
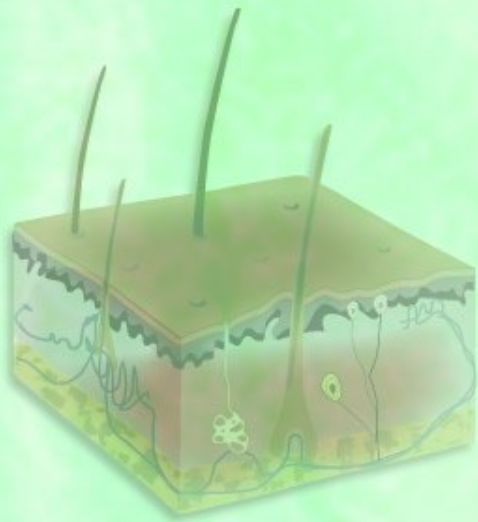
METHODS OF AGING ANALYSIS

- Glogau Classification of Photoaging
- FSIA (Face Skin Image Analysis)
- Fitzpatrick-Goldman Classification Wrinkle Scale
- Daniell's skin Wrinkle Grading Methods
- The Wrinkle Severity Rating Scale (WSRS)



GLOGAU CLASSIFICATION

- The Glogau classification system was developed to objectively measure the severity of photoaging and especially wrinkles
- It helps practitioners pick the best procedures to treat photoaging.



GLOGAU CLASSIFICATION

Type	Characteristics
1: No wrinkles	Typical age 20s to 30s Early photoaging Mild pigmentary changes No keratosis No or minimal wrinkles
2: Wrinkles in motion	Typical ages late 30s to 40s Early to moderate photoaging Early senile lentigines Palpable but not visible keratoses Parallel smile lines beginning to appear laterally to mouth
3: Wrinkles at rest	Typical age 50 or older Advanced photoaging Obvious dyschromias, telangiectasias Visible keratoses
4: Only wrinkles	Typical age 60 or older Severe photoaging Yellow-gray skin Precancerous lesions No normal skin

FSIA (FACE SKIN IMAGE ANALYSIS)

Gajah Mada University, Indonesia, has developed **DCT** (Discrete Cosinus Transformation) digital image analysis

- This program is for measuring surface roughness of skin including **wrinkles** that can be **captured by digital camera** and named **FSIA** (Face Skin Image Analysis)



FSIA (FACE SKIN IMAGE ANALYSIS)...

Classification of variable **roughness wrinkles** assessed by hue on the color spectrum **LUX** (Logarithmic hUe eXtension)

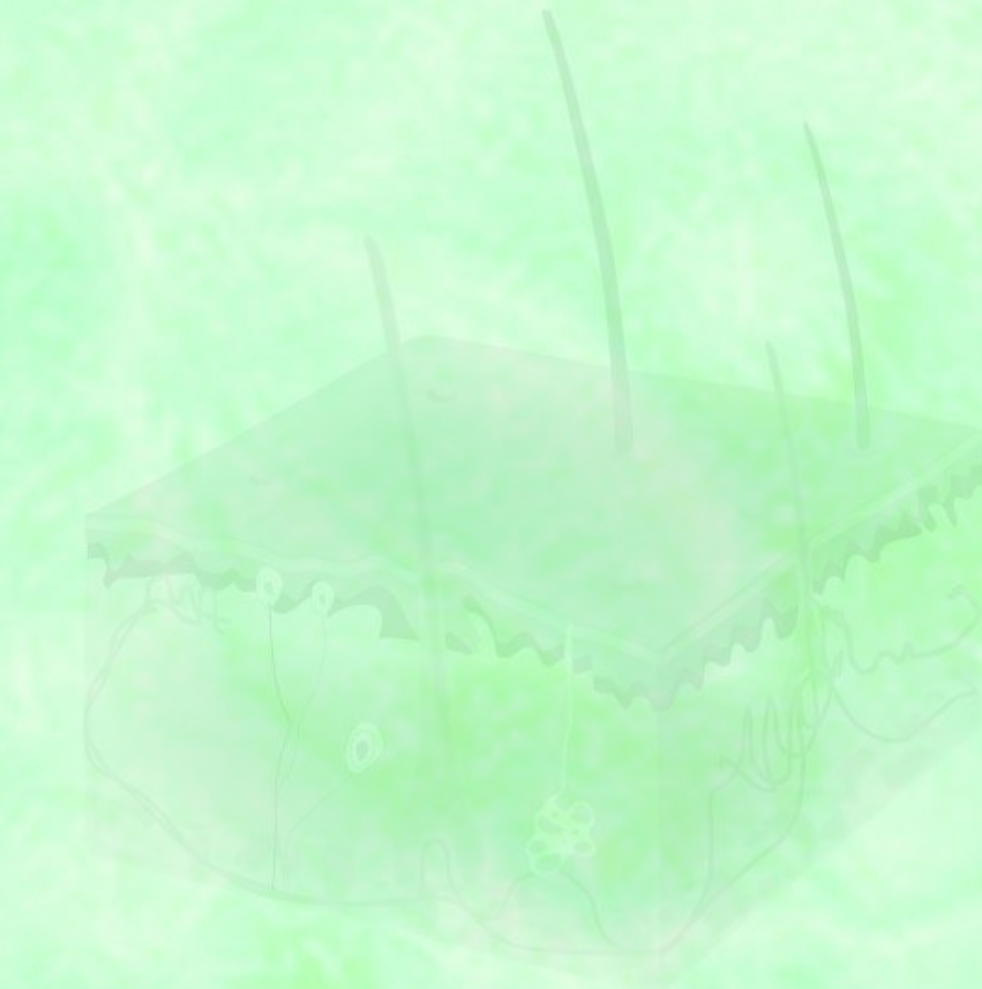
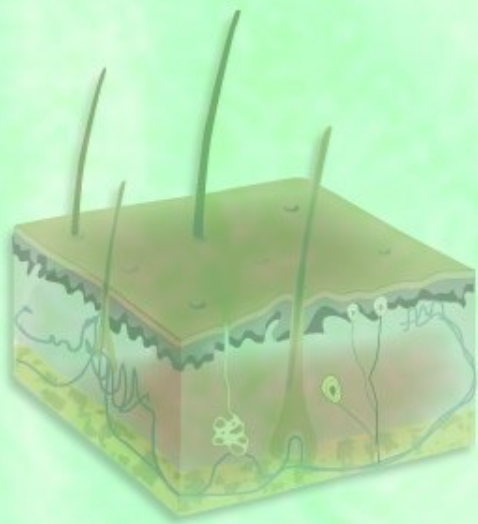
- The result images are taken from the consideration that the skin element consist of **red spectrum**

IMMUNOHISTOCHEMISTRY ANALYSIS

- Immunohistochemistry has emerged as a powerful investigative tool → provide supplemental information to the routine morphological assessment of tissue
- Study cellular markers → define specific phenotypes → provided important diagnostic, prognostic and predictive information relative to disease status and biology
- The application of antibodies to the molecular study of tissue pathology → adaptation and refinement of immunohistochemical techniques (use in fixed tissues)

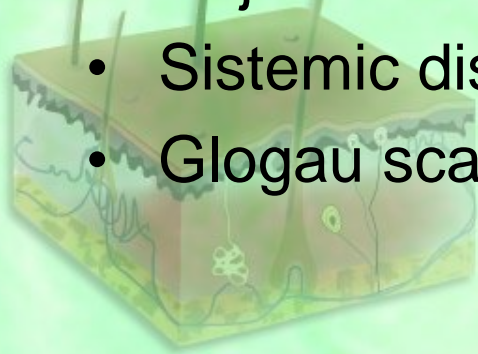


CASE

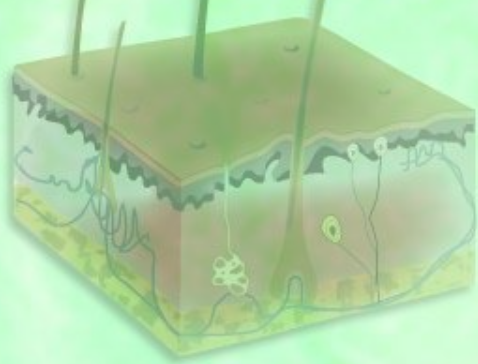


PATIENT A

- Name : Mr. A
- Age : 19 y.o
- Working status : Hospital employer
- Smoking history : yes (sometimes)
- Alcohol history : no
- Using cosmetical : no
- Using hormonal contraception : no
- Rejuvenation therapy : no
- Systemic diseases : no
- Glogau scale : type I

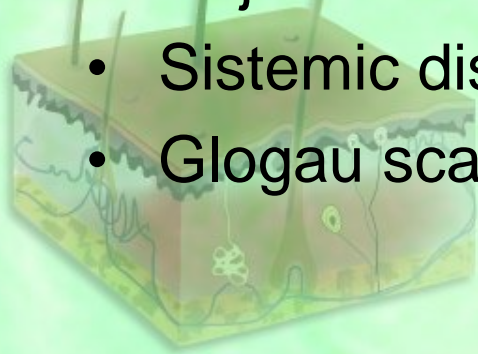


PATIENT A

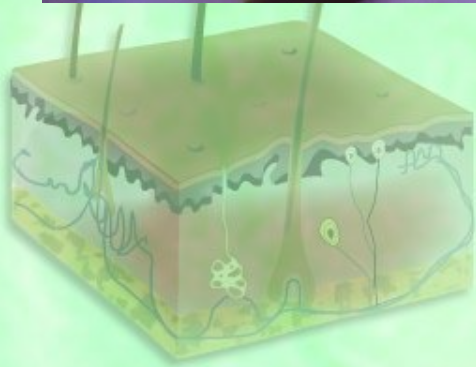


PATIENT B

- Name : Ms. B
- Age : 19 y.o
- Working status : Student
- Smoking history : no
- Alcohol history : no
- Using cosmetical : yes
- Using hormonal contraception : no
- Rejuvenation therapy : no
- Sistic diseases : no
- Glogau scale : type I

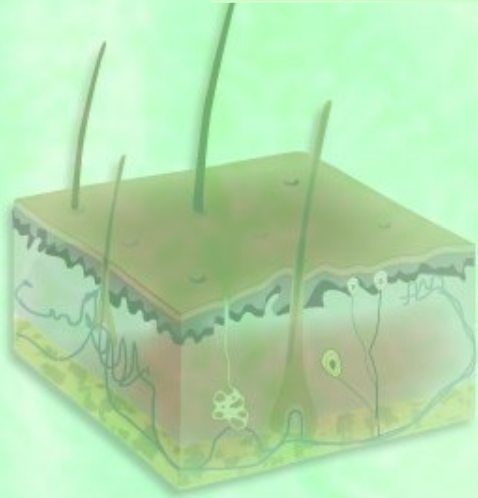


PATIENT B



FSIA (FACE SKIN IMAGE ANALYSIS)

FSIA result	Patient A	Patient B
Forehead	8,882	2,438
Left Cheek	6,835	1,535
Right Cheek	8,594	7,931



FSIA (FACE SKIN IMAGE ANALYSIS)

FSIA result	Patient A	Patient B
Forehead	8,882	2,438
Left Cheek	6,835	1,535
Right Cheek	8,594	7,931

From the FSIA result, compare patient A with patient B, we could see that there are significant different results from both patients. The premature aging can be caused by many factors such as sunlight exposure (patient A).



Immuno-histochemistry analysis

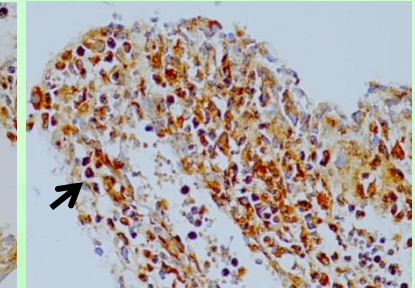
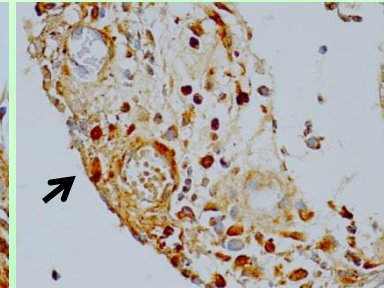
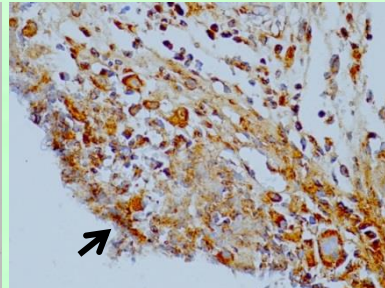
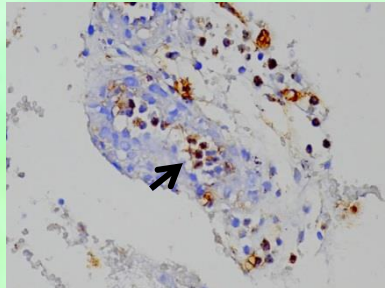
Apoptotic cells

MMP expression

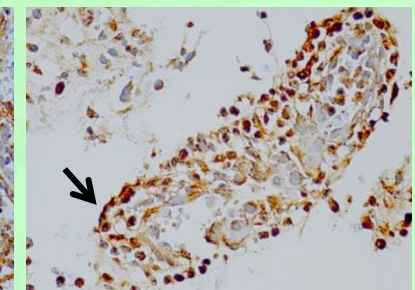
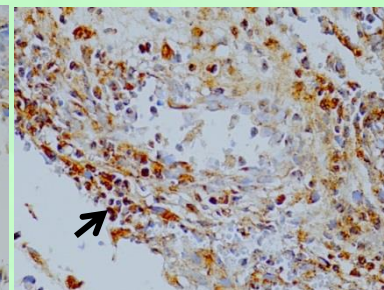
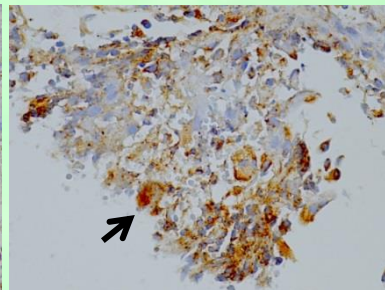
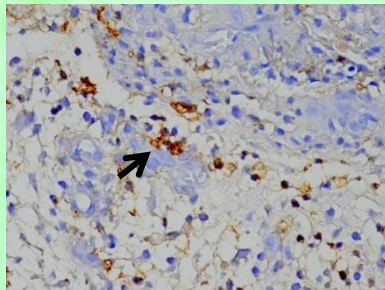
PKC- α expression

MAPK p38 activation

PATIENT A



PATIENT B



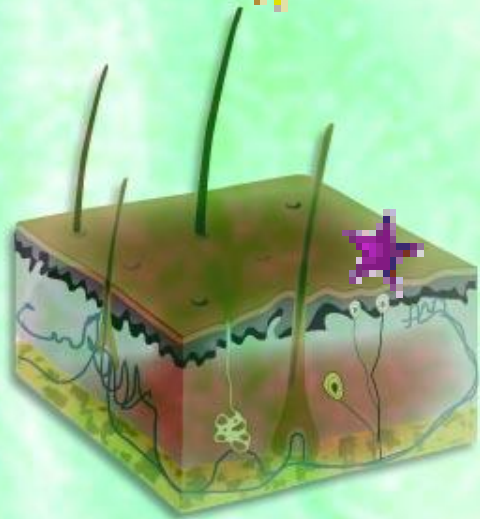
Representation of immunohistochemistry analysis of MMP1, PKC- α and MAPK-p38 cutaneous biopsy. Relative expression of MMP1, PKC- α and MAPK-p38 patient A increases against patient B. However, apoptotic cells - analysis with Terminal deoxynucleotidyl transferase dUTP nick end labeling assay (TUNEL assay) - it appears that the number of apoptotic cells showed a tendency to increase patient A to compared with patient B.

Summary

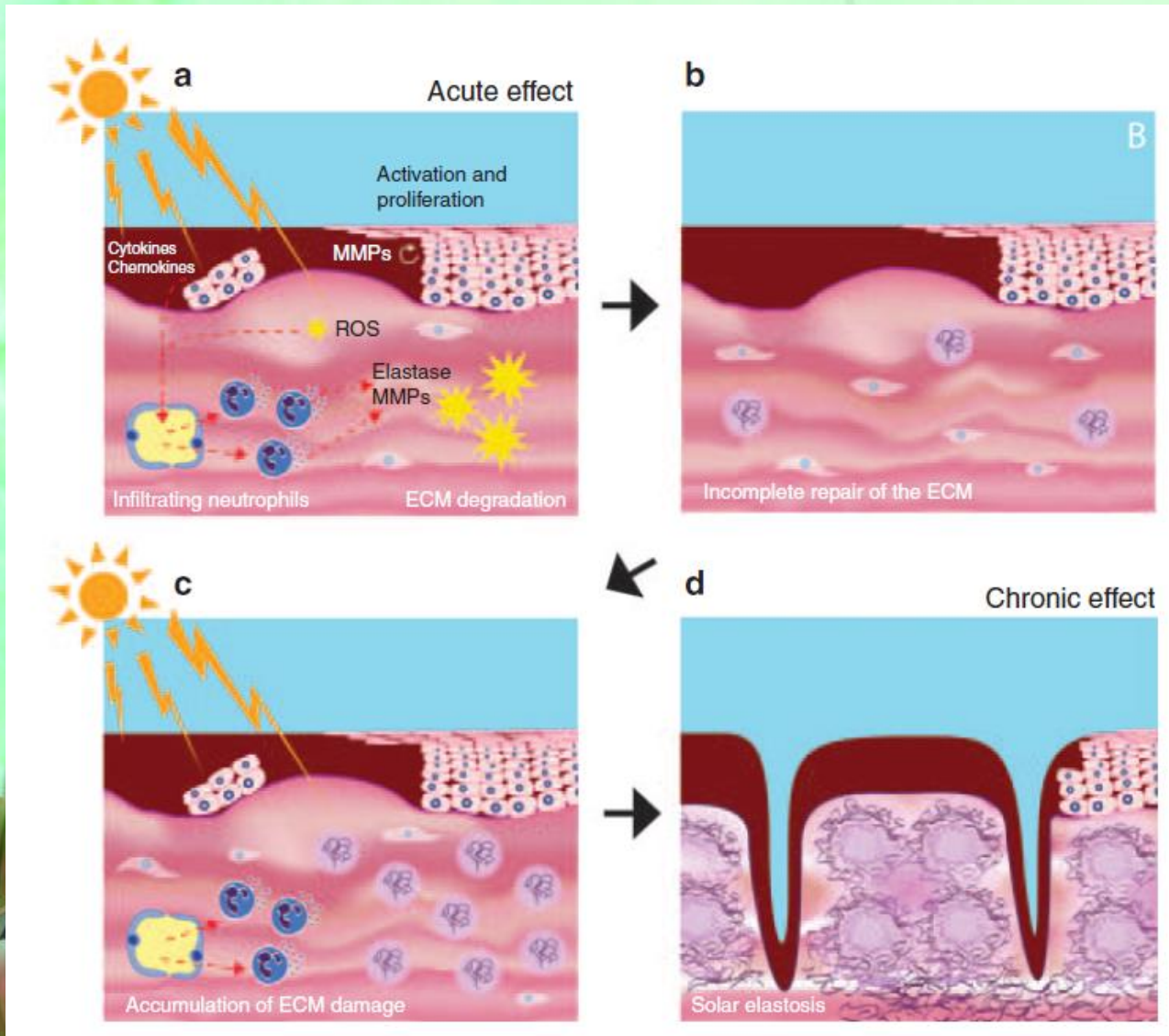
- It is important to distinguish between natural skin aging and photoaging.
- Knowledge about the mechanisms underlying these processes can be utilized for developing future therapies
- Educating patients is essential, as sunscreens and sun protection can prevent many of the changes associated with photoaging
- Many treatments are available to treat photoaged skin; however, the best treatment is prevention.



topical



The pathogenesis of photoaging



Mechanism of skin photoageing

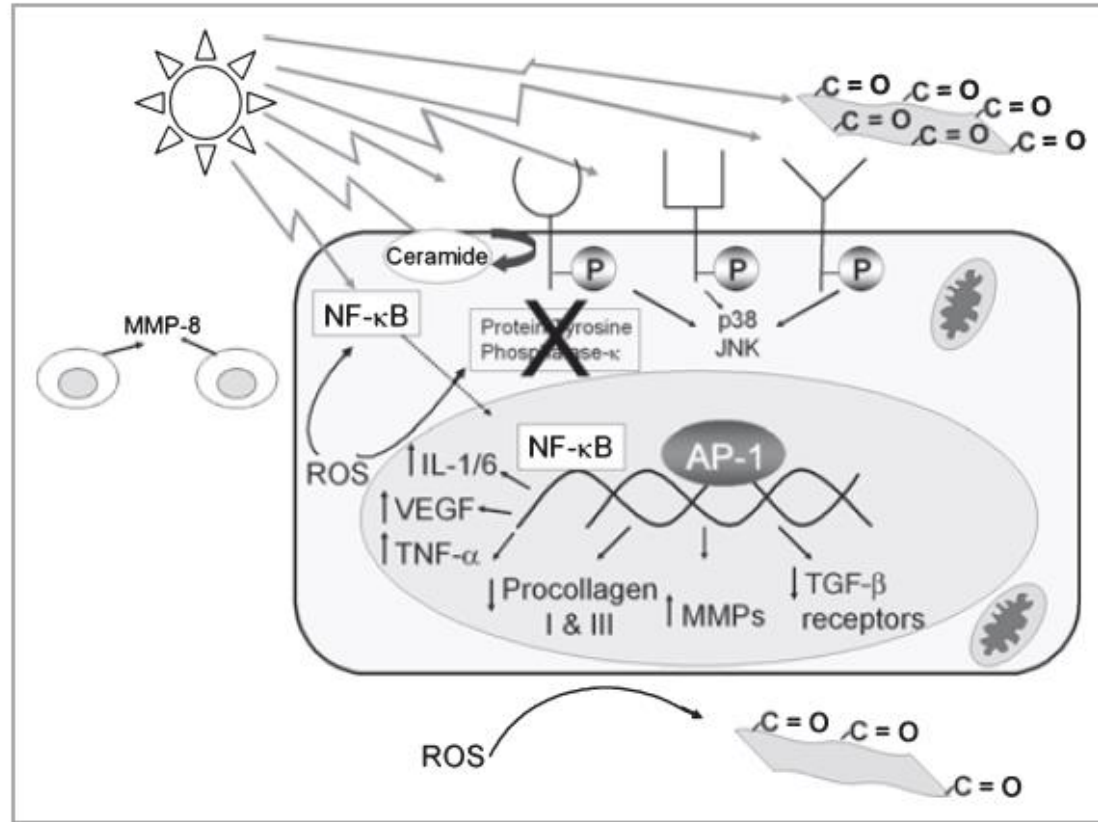
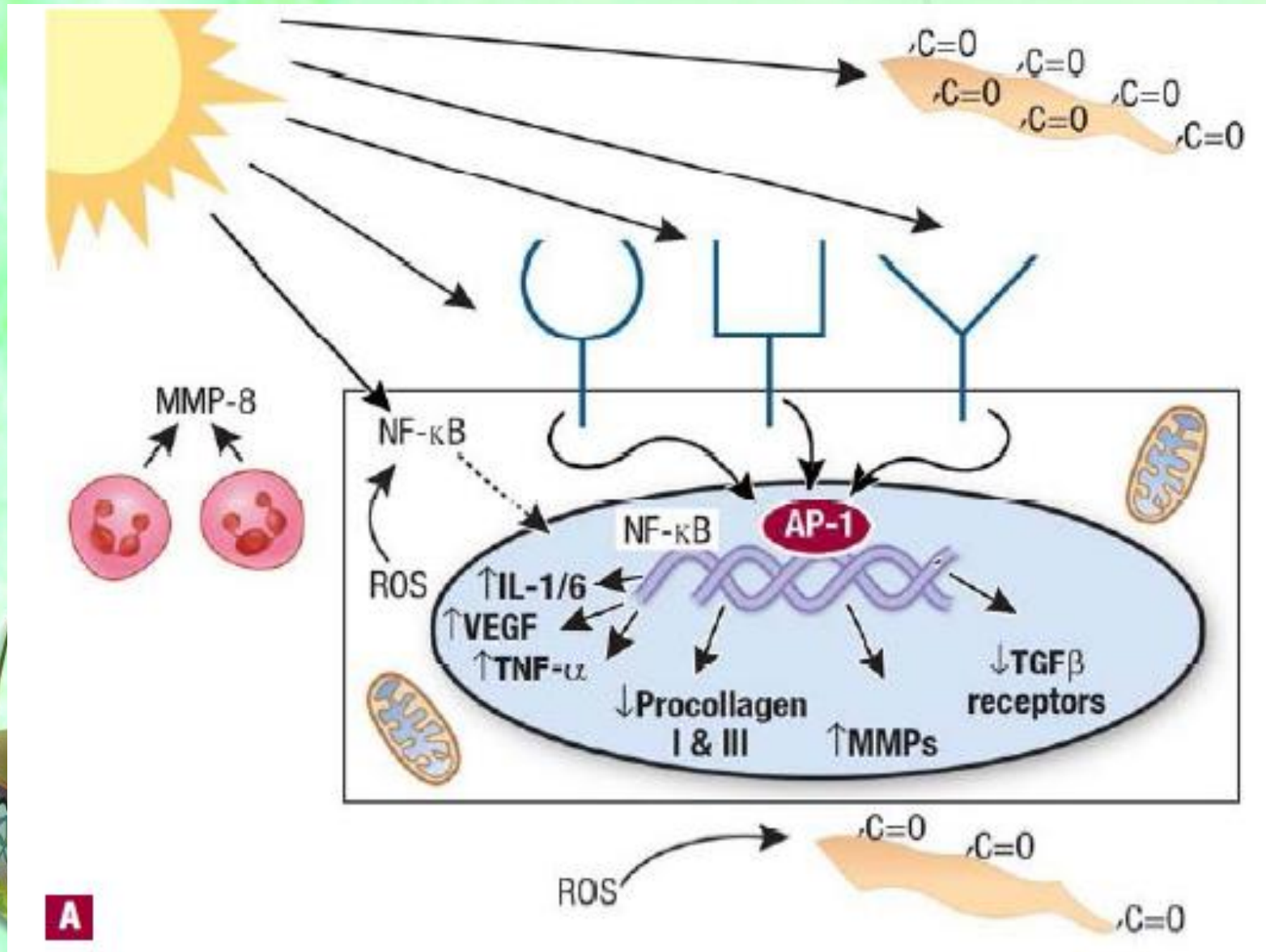
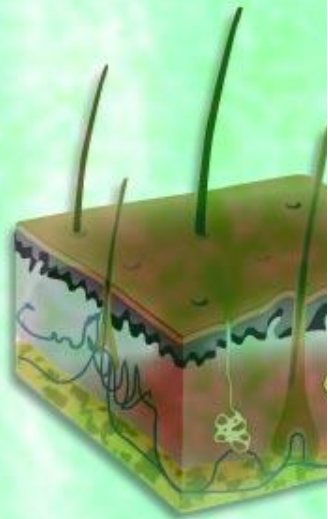
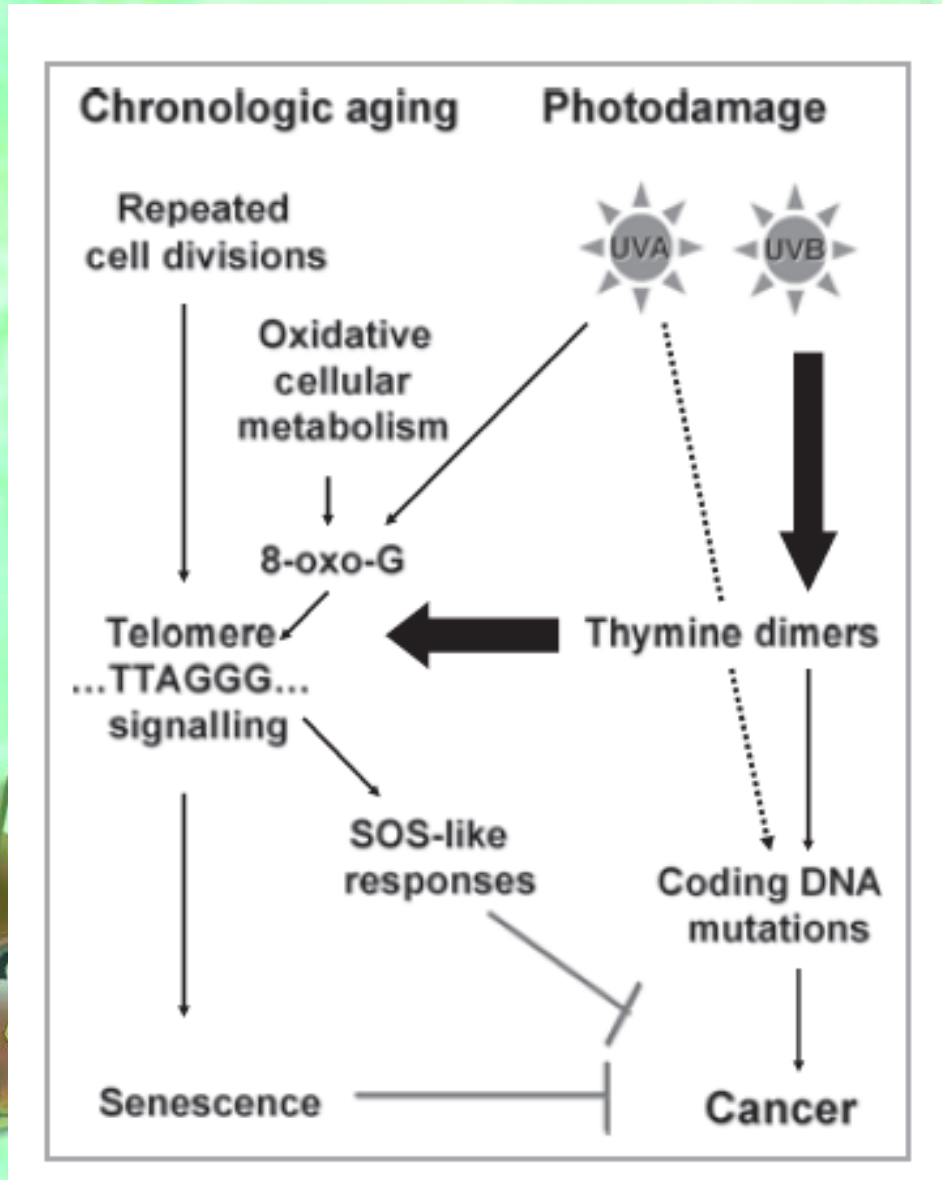


Fig 1. Mechanisms of skin photoageing. Reactive oxygen species (ROS) generated during aerobic metabolism and after ultraviolet (UV) irradiation activate the transcription factor NF-κB, which induces the expression of pro-inflammatory cytokines. ROS also inhibit the enzyme protein-tyrosine phosphatase-κ leading to receptor activation. Receptor activation leads to intracellular signalling through the MAP kinases, p38 and JNK. These enzymes as well as ceramide released from the cell membrane activate the nuclear transcription complex AP-1. AP-1 increases transcription of MMPs and decreases expression of the procollagen I and III genes and TGF-β receptors, with a final consequence of reduced dermal matrix formation. UV also activates the NF-κB transcription factor and through neutrophil recruitment and MMP-8 release further exacerbates matrix degradation. ROS also lead to the formation of carbonyl groups (C=O) and accumulation of oxidized damaged dermal proteins. Damaged proteins containing carbonyl groups accumulate in the upper portions of the dermis. In addition, mitochondria display large DNA deletions and compromised function. Modified from Fisher et al.³; from Halachmi S, Yaar M, Gilchrist BA. [Advances in skin aging/photoaging: theoretical and practical implications]. *Ann Dermatol Venereol* 2005; 132:362–7. [Article in French], with permission.

Mechanism of intrinsic skin photoageing



Hypothetical common mechanism for intrinsic ageing & photoageing



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