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TESTING & beyond

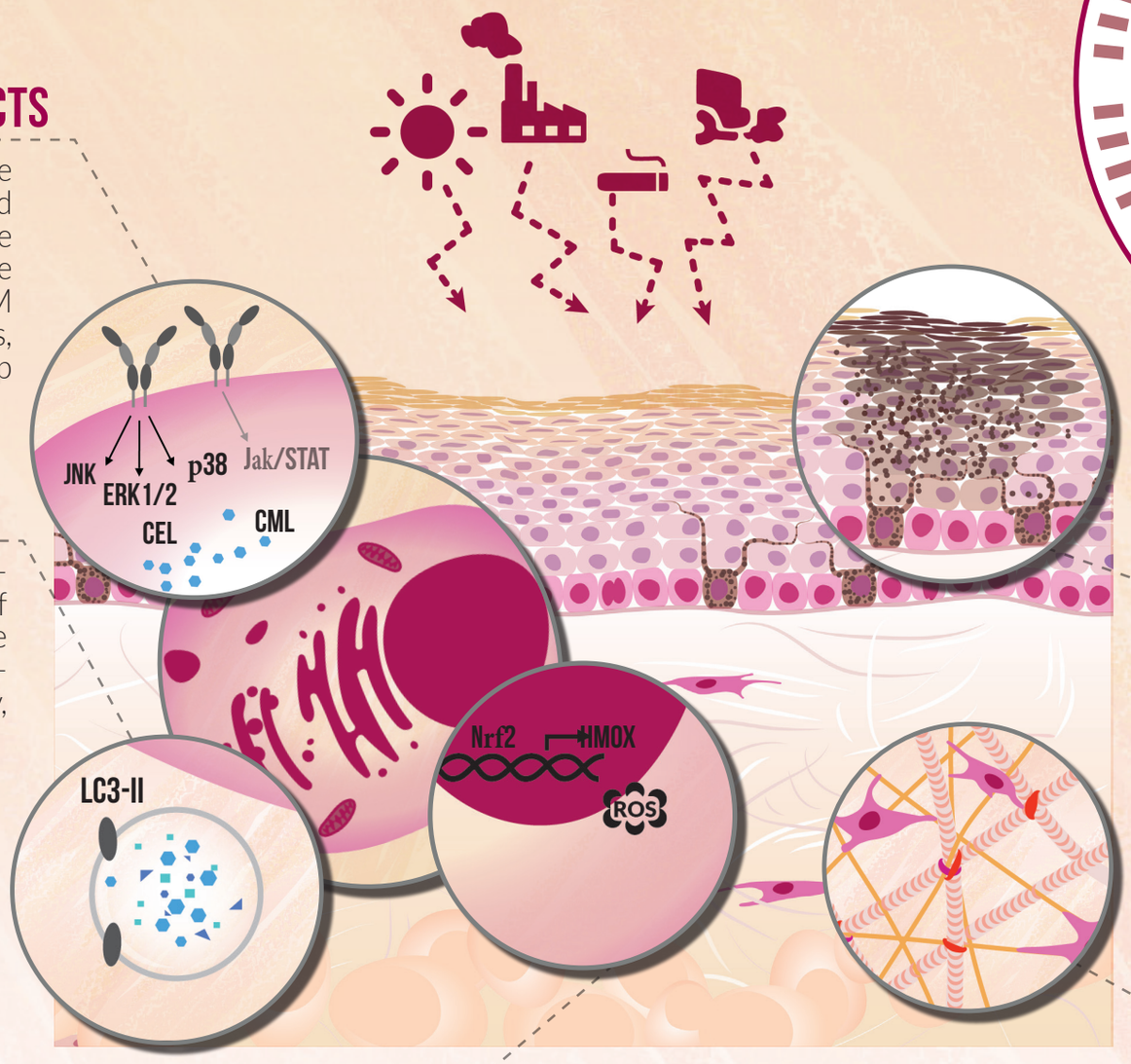
ANTI - AGEING
EFFICIENCY
DISCOVER OUR **COMPLETE** IN-VITRO
EVALUATION METHODS

ADVANCED GLYCATION END PRODUCTS

A main consequence of oxidative stress is the appearance of protein glycation and advanced glycation end products (AGEs) during life time, damaging skin cell membranes. AGEs are directly involved in damage and loss of ECM components, the appearance of roughness, uneven tone, brown patches, thin skin and deep wrinkles.

DETOXIFICATION

Healthy skin relies on many endogenous systems to insure repair, disposal or recycling of disrupted biological components. These include machineries of the proteasome, lysosome, autophagy, DNA damage and repair, NRF2 pathway, unfolded protein response, etc.



AGE SPOTS

Ageing process leads to pigmentation disorders (skin macular lesions) of variable melanin content which generally increase (senile lentigo). Rencent data also suggest a link between environmental pollution, ageing signs, and increased age spots.

EXTRACELLULAR MATRIX COMPONENTS

Structural and functional components of the ECM are profoundly disrupted during skin ageing processes resulting in weakened contraction of collagen fibers, decrease of elastin and proteoglycans abundance, increased metalloproteases activity, etc.

OXIDATIVE STRESS

Exhaustion of the endogenous antioxidative machinery is incriminated in skin ageing. Overall oxidative stress can be measured in presence or absence of external factors, as well as mitochondrial complex II abundance which is inversely correlated with skin ageing.

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CHALLENGING SKIN PIGMENTATION

DISCOVER OUR **EXCLUSIVE** *IN VITRO* DEVICE

DERMOSCOPY

MELANOSOME TRANSFER

Melanosomes are transferred to surrounding keratinocytes in which they localize to the perinuclear area.

MELANOGENIC PATHWAYS

The complex & dynamic process of melanogenesis involves an extensive intercellular cross-talk that activates signalling responses such as MC1-R pathway activation.

ENZYMATIC CONVERSION

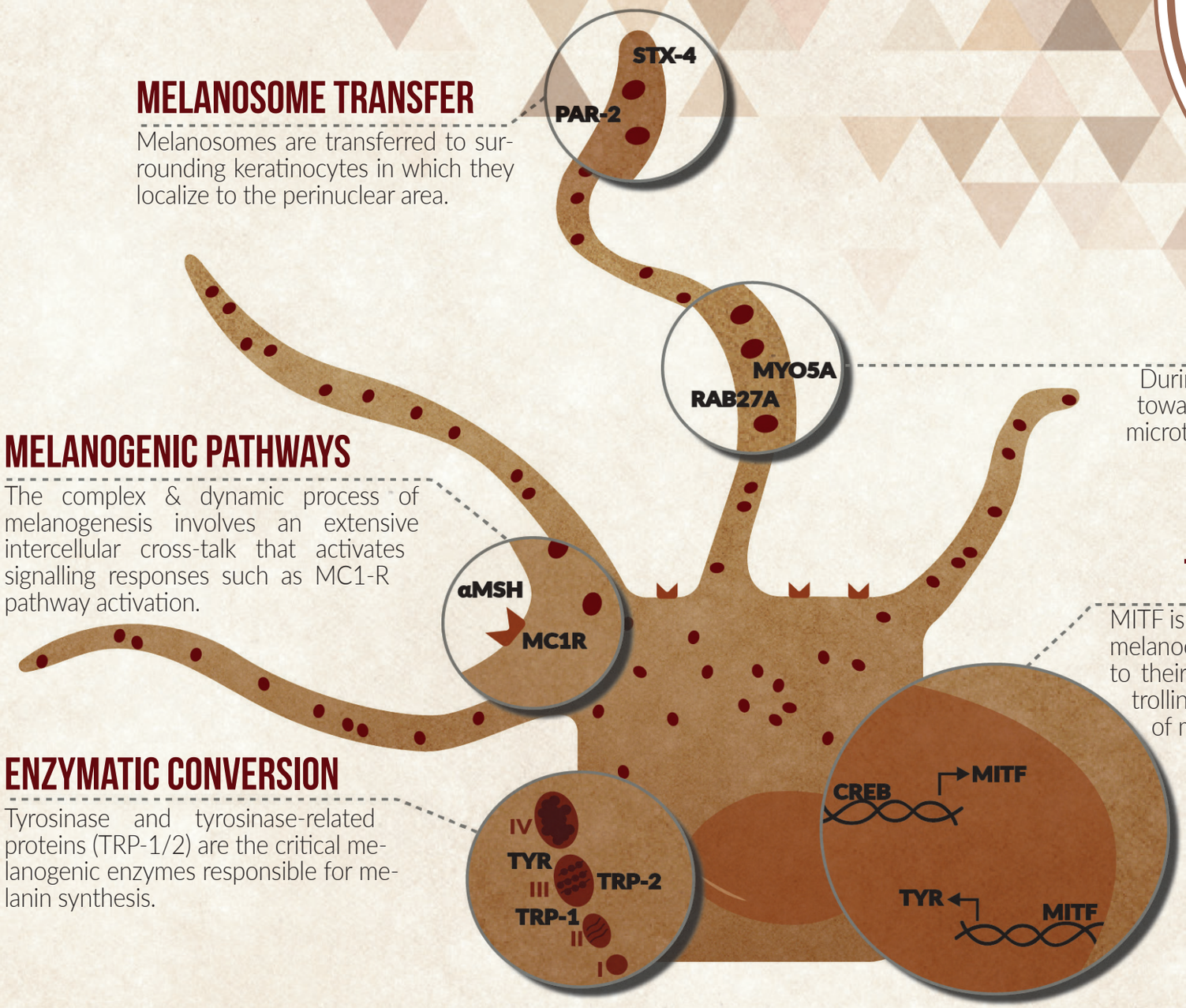
Tyrosinase and tyrosinase-related proteins (TRP-1/2) are the critical melanogenic enzymes responsible for melanin synthesis.

MELANOSOME MATURATION

During their maturation, melanosomes move towards the extremity of dendrites through a microtubule-dependant intracellular transport.

TRANSCRIPTIONAL REGULATION

MITF is a major transcription factor that regulates melanocyte function from their development to their response to environmental stimuli controlling melanosome formation and expression of melanogenic enzymes.



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INFLAMMATION

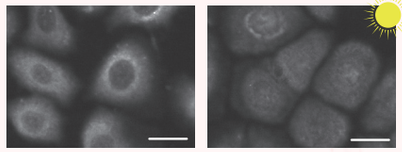
ATOPIC DERMATITIS

PSORIASIS

Sensitive Skin

NF-κB TRANSLOCATION

Increased nuclear translocation of NF-κB is an important pathogenic factor involved in many acute & chronic inflammatory disorders.



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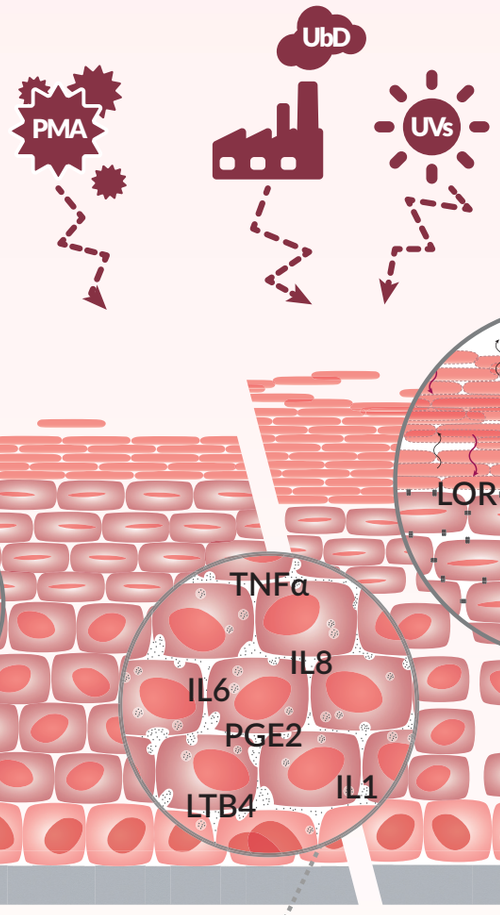
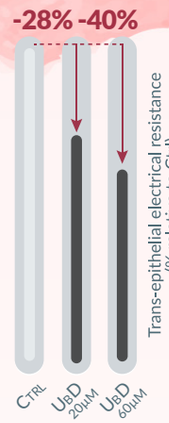
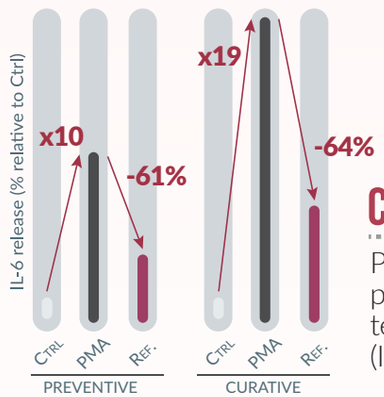


BARRIER DISRUPTION

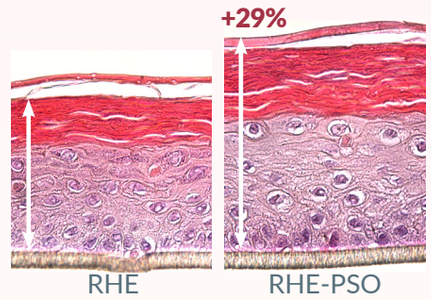
Defects in the protective physical barrier lead to a decrease in expression of differentiation markers such as filaggrin (FLG), involucrin (IVL), loricrin (LOR) & trans-epithelial electrical resistance (TEER) while facilitating allergens/pathogens invasion.





CYTOKINES & CHEMOKINES RELEASE

Production of primary proinflammatory cytokines prostaglandin E2 (PGE2), leukotriene B4 (LTB4), interleukin-1 (IL-1), interleukin-6 (IL-6), interleukin-8 (IL-8), & tumour necrosis factor-α (TNF-α).



MORPHOLOGICAL CHANGES

Inflammatory dermatoses are characterized by the presence of abnormal keratinocyte proliferation (acanthosis) or accumulation of intraepidermal edema (spongiosis) observed in psoriasis & AD, respectively.

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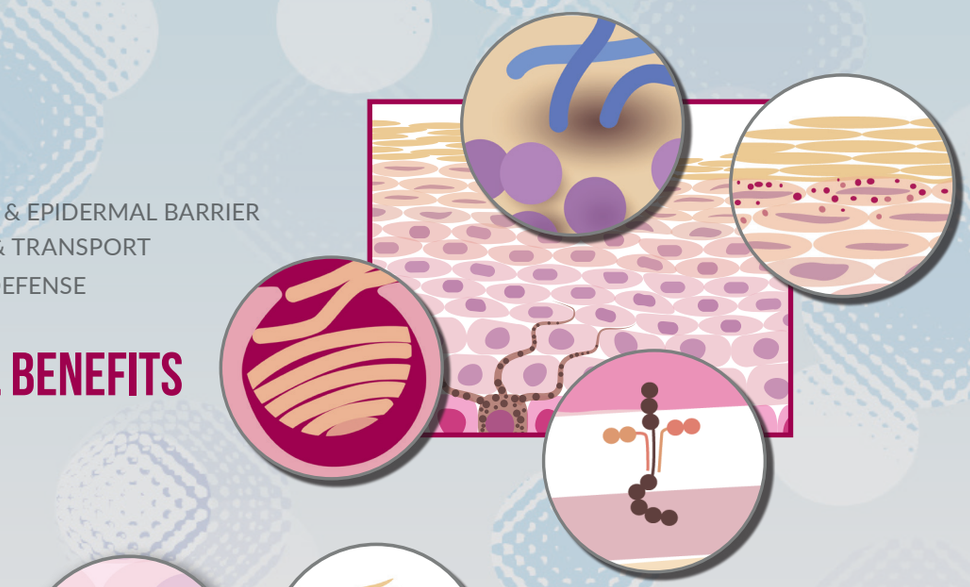
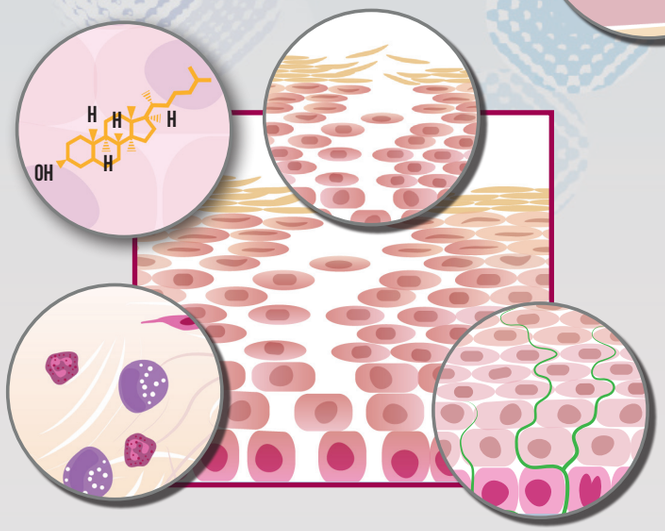
DISCOVER our TRANSCRIPTOMICS TOOLS for TARGET IDENTIFICATION & MECHANISM OF ACTION



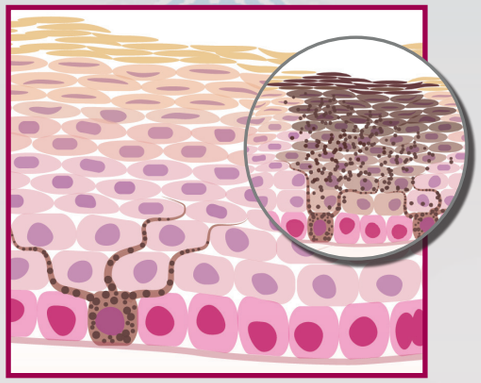
DIFFERENTIATION & EPIDERMAL BARRIER
LIPID SYTNHESIS & TRANSPORT
ANTIMICROBIAL DEFENSE
CELL JUNCTIONS

EPIDERMAL BENEFITS



INFLAMMATION & CHEMOTAXIS
LIPID HOMEOSTASIS
BARRER RECOVERY
PRURITUS

SKIN BARRIER & TISSUE COHESION

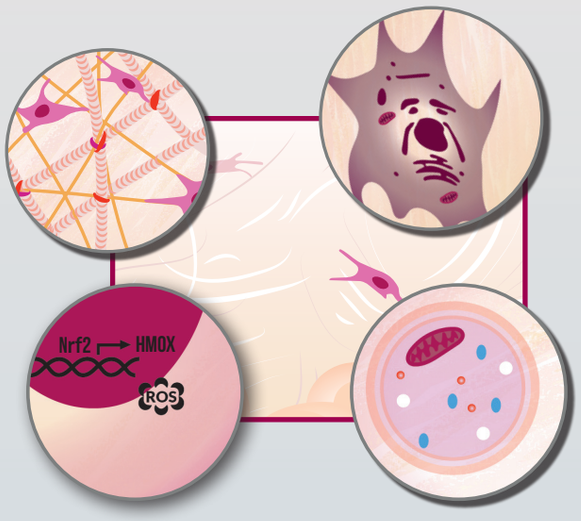


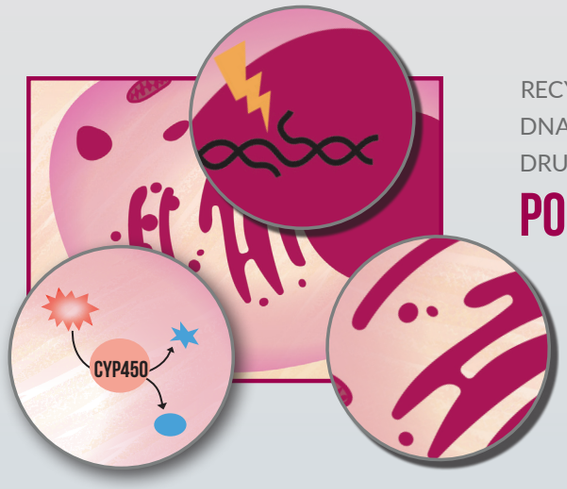
DEPIGMENTATION
MELANOGENESIS
AGE SPOTS

PIGMENTATION

EXTRA CELLULAR MATRIX MODELING
OXYDATIVE STRESS RESPONSE
AUTOPHAGY & RECYCLING
CELLULAR SENESCENCE

DERMAL BENEFITS





RECYCLING & DETOXIFICATION
DNA DAMAGE REPAIR
DRUG METABOLISM

POLLUTION & DETOXIFICATION

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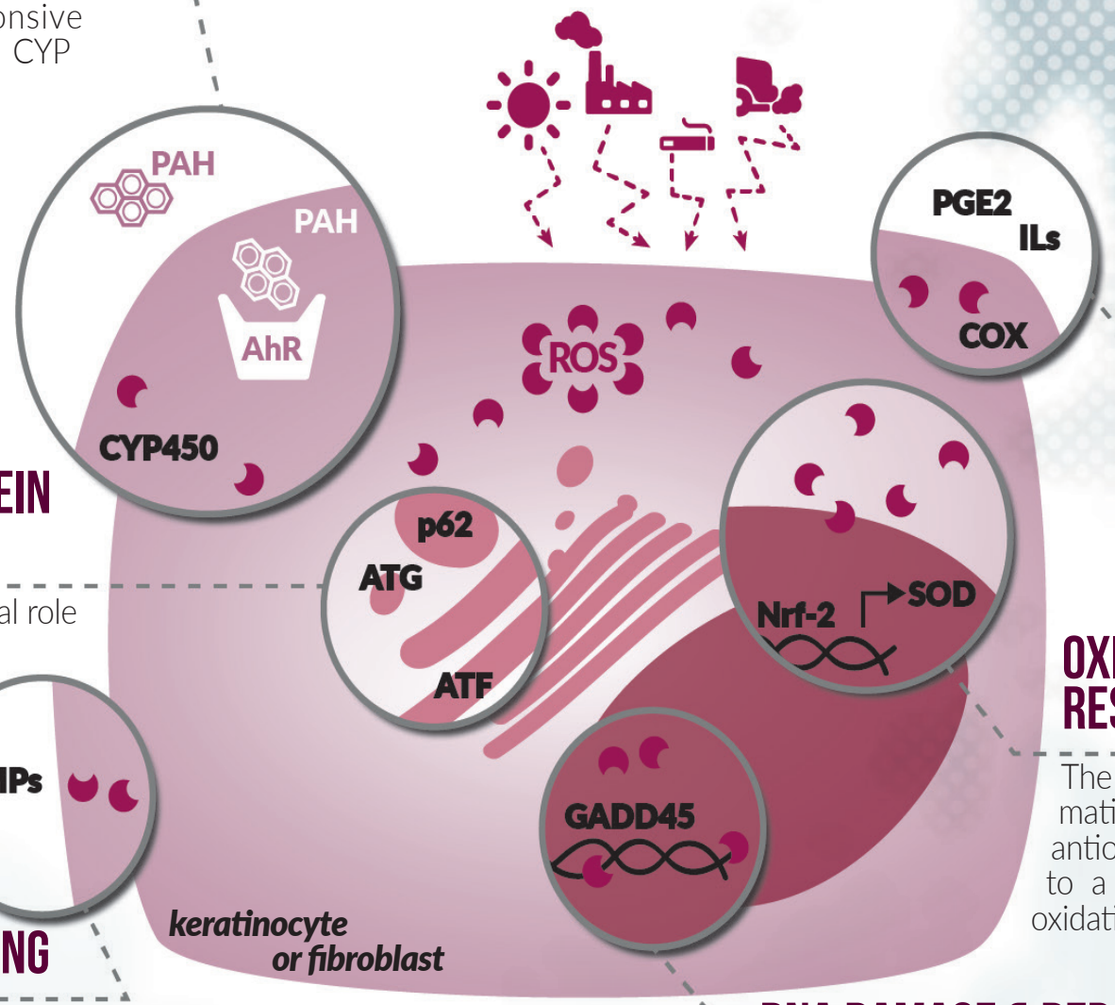
POLLUTION
AND ENVIRONMENTAL STRESSES

DRUG METABOLISM

The cytoplasmic xeno-sensor AhR leads to induction of xenobiotic-responsive elements such as CYP proteins family.



INFLAMMATORY RESPONSE

Pollutants stimulate the release of pro-inflammatory mediators such as PGE2 & IL cytokines.

UNFOLDED PROTEIN RESPONSE

Autophagy has a pivotal role in recycling damaged non-functional organelles & unfolded proteins.

OXIDATIVE STRESS RESPONSE

The depletion in enzymatic and nonenzymatic antioxidant capacity leads to a chronic and vicious oxidative state.

MATRIX MODELLING

Among others, Matrix MetalloProteinases contribute to matrix modulation that affects cell-cell/matrix interactions critical for skin integrity.

DNA DAMAGE & REPAIR

If not controlled, DNA alterations could affect cell cycle and induce apoptosis.