CMed®
AESTHETICS

ITALIAN RESEARCH & INNOVATION

EVERYTHING WE DO IS AIMED TO IMPROVE PEOPLE'S WELLNESS AND BEAUTY

CMed® AESTHETICS

LUMINESCENS®

DEPIGMENTING & BRIGHTENING SOLUTION

01. Strenghts

02. Mechanism of Action

03. Meso Patented Formula

04. Home Cream Ingredients

05. Protocol of use

06. Our results

07. How it works

CMed® AESTHETICS

LUMINESCENS®

DEPIGMENTING & BRIGHTENING SOLUTION

The formulation is compatible with **ALL PHOTOTYPES**, for oily and dry skin.

The depigmenting activity is carried out by **SPECIFIC ACTIVE INGREDIENTS**, assisted by a superficial peeling action that promotes epidermal renewal and favors the delivery to deeper derma layers.

It is a protocol with lightening and depigmenting activity specifically designed for the treatment of SKIN PIGMENTATION of the face and the rest of the body.

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The innovative **PROFESSIONAL KIT** is the game changer in depigmentation therapy and **includes**:

PROPRIETARY PATENTED TECHNOLOGY

DERMATOLOGICALLY TESTED

LUMINESCENS® Kit

Face | Body

For clinical use

2 mesotherapy vials x 4 mL each
+ 1 cream bottle 30 mL

For 4 outpatient treatments
(estimated. It may vary depending on severity and body parts to be treated)

LUMINESCENS® Home Cream

Face | Body

For outpatient use

1 cream bottle 30 mL

Innovative formulation for outpatient mesotherapy treatment with a pending patent.

No post treatment downtime and no mask needed to be applied for 6-12 hours, unlike other leading products.

No negative impact on daily life.

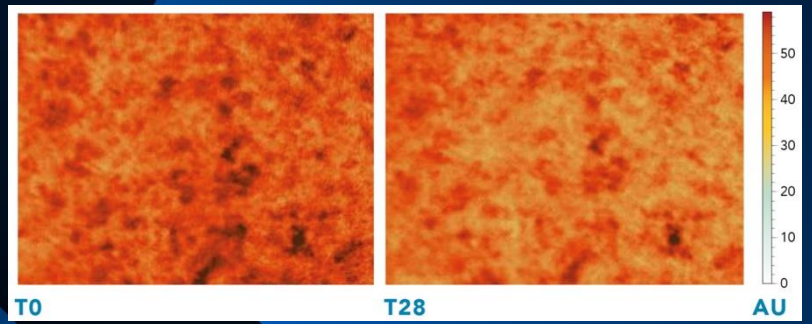
Cutting-edge formulation, rich in active ingredients, adapted for home treatment with noticeable results with maximum safety and comfort.

CLINICALLY TESTED RESULTS

Scientifically proven reduction of stains and dermatologically tested.

Study of the effect and cosmetic properties of a product through evaluations and instrumental analysis performed by professionals under medical supervision and consumer self-assessments.

CMed Aesthetics S.p.A. Report 213E30F-1 Bio Basic Europe s.r.l. in collaboration with University of Pavia Department of Biology and Biotechnology "L. Spallanzani". Studies carried out at CDC Dermo-Clinical Research Insitute.



IMAGES DETECTED AND ANALYZED WITH MIRAVEX ANTERA 3D ®

LUMINESCENS®

95% OF PATIENTS

- said that the **treatment reduces skin spots**.
- gave a **positive opinion to the treatment**.
- said that the **mesotherapeutic treatment is pleasant and** does not create discomfort on the skin.
- said that the **home product is comfortable**.

MICRONEEDLING DEVICES:

DERMA PEN

MULTI-NEEDLE

ELECTROPORATION

Melanogenic enzyme blockers

TYROSINASE INHIBITORS

Oligopeptide-34, Tranexamic acid[1], Aminoethyl phosphinic acid & Glycyrrhetinic acid, Azelaic acid[2], Arbutin[3], Phenylethyl resorcinol[4], Silybum marianum extract[5], Glabridin[6].

Melanin formation and transport blockers

COPPER CHELATORS AND MC1R ENZYME INHIBITORS

Ellagic acid & Phytic acid, Acetyl tetrapeptide-2, Potassium azeloyl diglycinate[8], Biomimetic peptides, Hexapeptide-40 sh-polypeptide-76 inhibitory, Acetyl hexapeptide-1[9].

Increase cellular turnover

REMOVAL OF AND PRE-EXISTING MELANIN

Citric acid, Tartaric acid, Salicylic acid, Retinol[10].

Antioxidants

ANTI FREE RADICAL ACTION

Peptide CG-TGP2, Glutathione, Tocopheryl acetate.

MICRONEEDLING DEVICES:

DERMA PEN

MULTI-NEEDLE

ELECTROPORATION

Melanogenic enzyme blockers

TYROSINASE INHIBITORS

Tranexamic acid is a derivative of an essential amino acid, lysine. It has several mechanism of actions:

- Anti-plasmin activity. Plasmin is responsible for the increase of MSH levels, a melanocyte-stimulating hormone. Moreover, UV light improves the interaction of plasmin with keratinocytes and melanocytes and the release of some mediators, which in turn stimulate the activity of tyrosinase and the formation of melanin. The blocking of plasmin allows for preventing the entire cascade of melanin production.
- Competitive inhibition of tyrosinase with its substrate tyrosin.

Aminoethyl Phosphinic Acid stabilizes DOPAchrome and prevents the further steps of formation of DHI (dihydroxyindole) or its acid form DHICA. It is also a chelating agent: it binds to metal compounds and prevents their fundamental involvement in the melanin cascade regulation. It has an action on the microcirculation by improving epidermal metabolism.

Arbutin is a glycoside that competes with DOPA at its receptor site of tyrosinase enzyme, acting as a substrate. It has good antioxidant capabilities as well, helping to reduce the amount of free radicals generated by UV rays.

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Home Cream Ingredients

Melanogenic enzyme blockers

TYROSINASE INHIBITORS

Tranexamic acid, Aminoethyl phosphinic acid, Arbutin, Oligopeptide-34, Glabridin*, Silybum marianum extract, Phenylethyl resorcinol, Glycyrrhetinic Acid.

*is a natural licorice derivative and an extremely powerful depigmenting agent. It's an example of non-competitive tyrosinase inhibition; this means the binding site does not coincide with the substrate binding site (see arbutin or tranexamic acid), but it involves another portion of the enzyme. Anti-inflammatory effects of glabridin in vitro were also shown by its inhibition of superoxide anion productions and cyclooxygenase activities. These data indicated that glabridin is a unique compound possessing more than one function.

Melanin formation and transport blockers

COPPER CHELATORS AND MC1R ENZYME INHIBITORS

Nicotiana Benthamiana, Hexapeptide-40, sh-polypeptide-76 inhibitory, Acetyl hexapeptide-1, Phytic acid.

Increase cellular turnover

REMOVAL OF AND PRE-EXISTING MELANIN

Citric acid, Tartaric acid, Salicylic acid, Retinol.

Antioxidants

ANTI FREE RADICAL ACTION

Hyaluronic acid, Panthenol, Tocopheryl acetate.

LUMINESCENS®

treatment is to be carried out in two stages:

1

The mesotherapy solution must be used by professionals only in a clinic environment.
The application method is either microneedling, using devices such as a microneedling pen, dermaroller or similar, or electroporation.
Recommended product amount per application is 1 mL per 10 sq/cm body area including intimate areas; 2 mL for face treatment. The amount of the product can be increased to treat more persistent pigmentation.

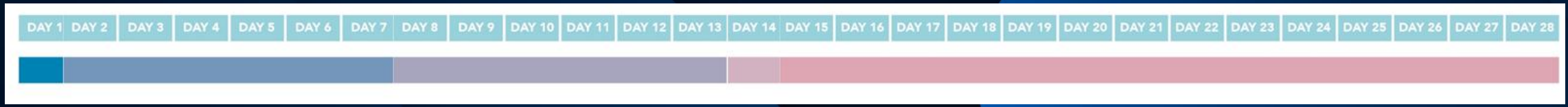
2

After clinic treatment, patient should follow home care protocol with home cream application as described below.

LUMINESCENS®

The treatment protocol is for the exclusive use of a professional who applies it accordingly to the specific needs of the patient and the result to be obtained. It is possible to identify a standard procedure that can be used in cases of skin hyperpigmentation: melasma, chloasma, solar lentigo or as a lightening and illuminating treatment.

- **Day 1:** First mesotherapy application in clinic + post treatment home cream.
- **Day 2-7:** Apply the home cream twice a day, preferably morning and evening.
- **Day 8-13:** Apply the home cream massaging until completely absorbed once a day, in the evening.
- **Day 14:** Second mesotherapy application in clinic.
- **Day 15-28:** Apply the home cream massaging until completely absorbed once a day, in the evening.



- For the duration of the treatment it is important to apply an SPF 50+ sunscreen cream and avoid exposure to the sun or UV lamps.
- The minimum recommended period for treatment is 28 days, to be repeated for another 28 if necessary and indicated by the doctor.

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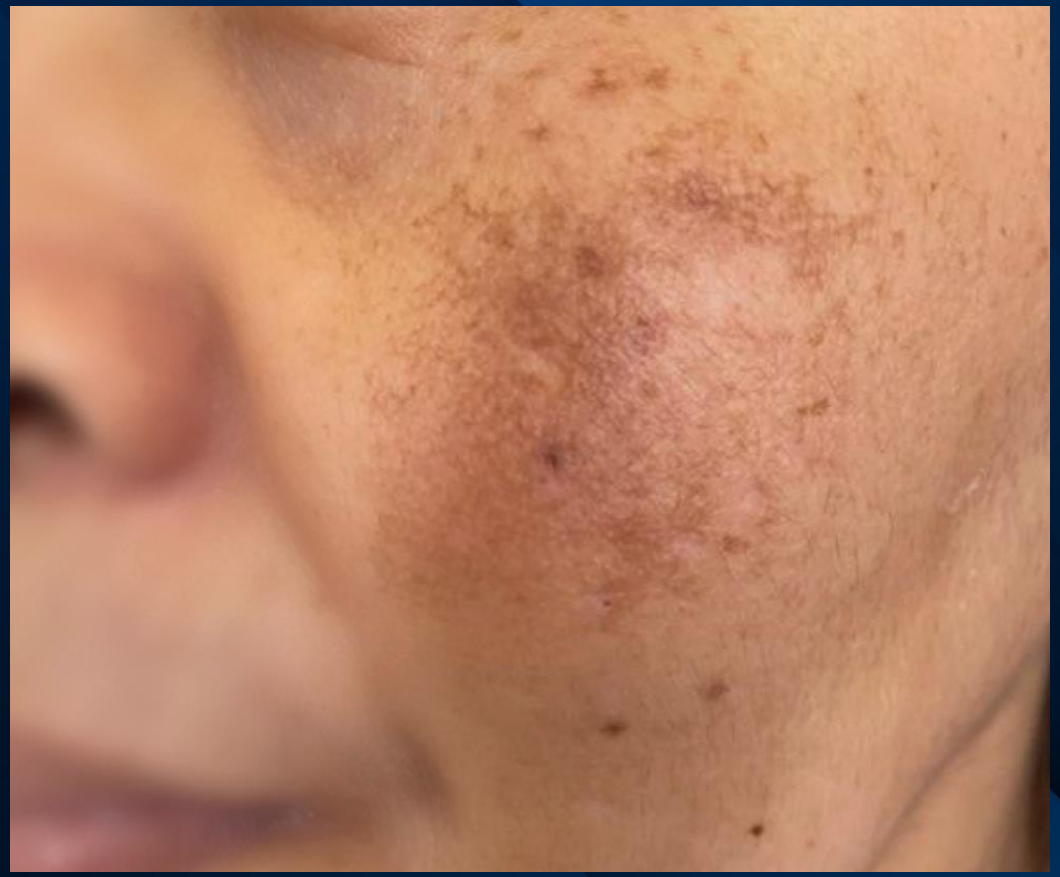
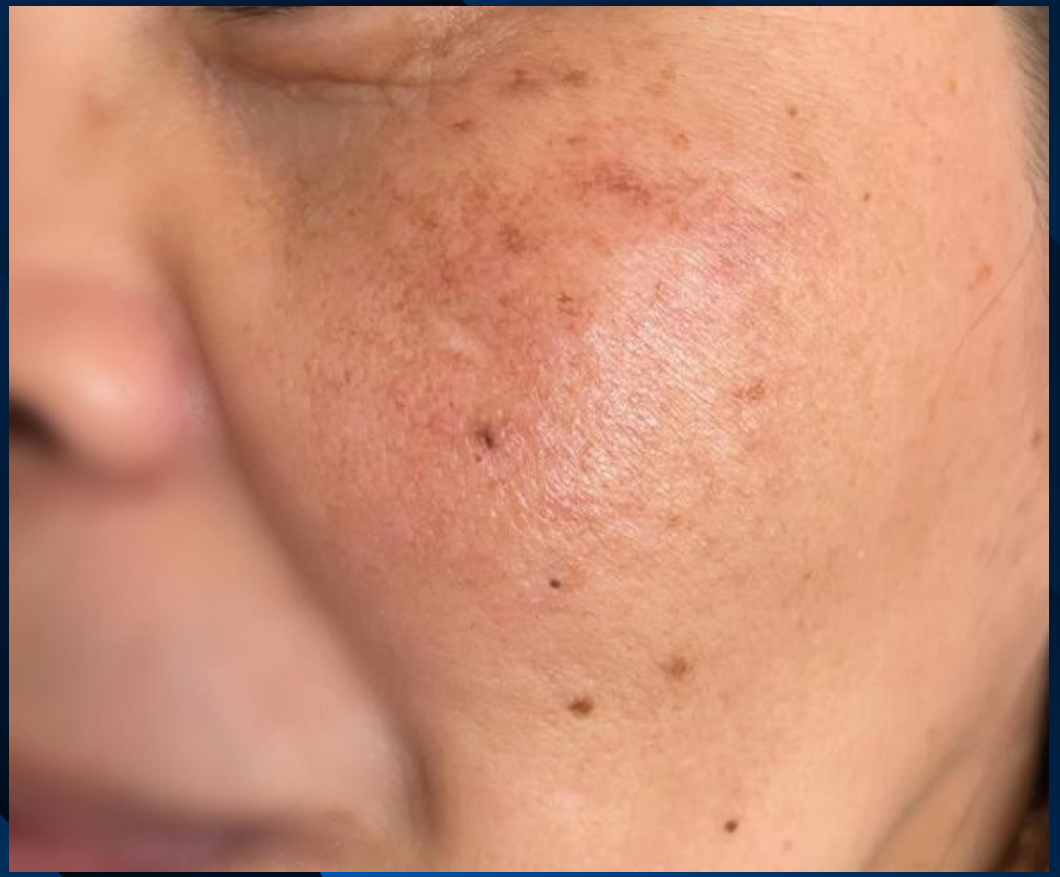
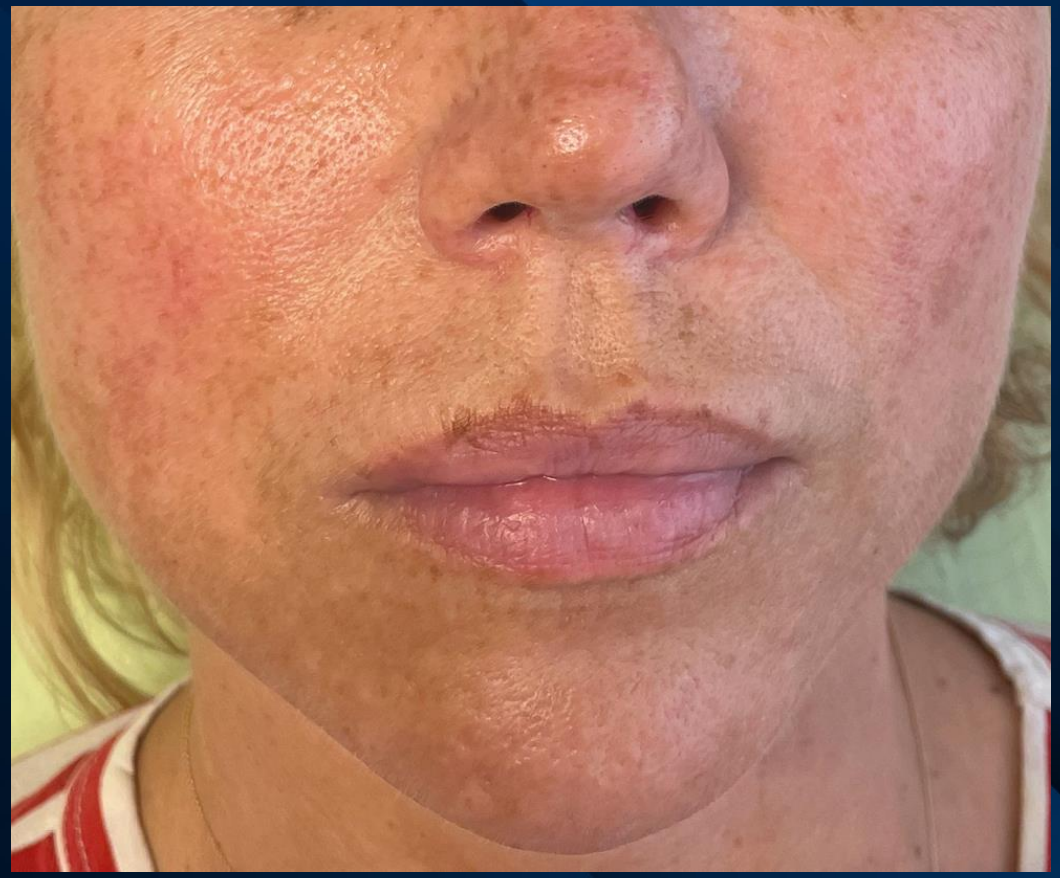
Our results

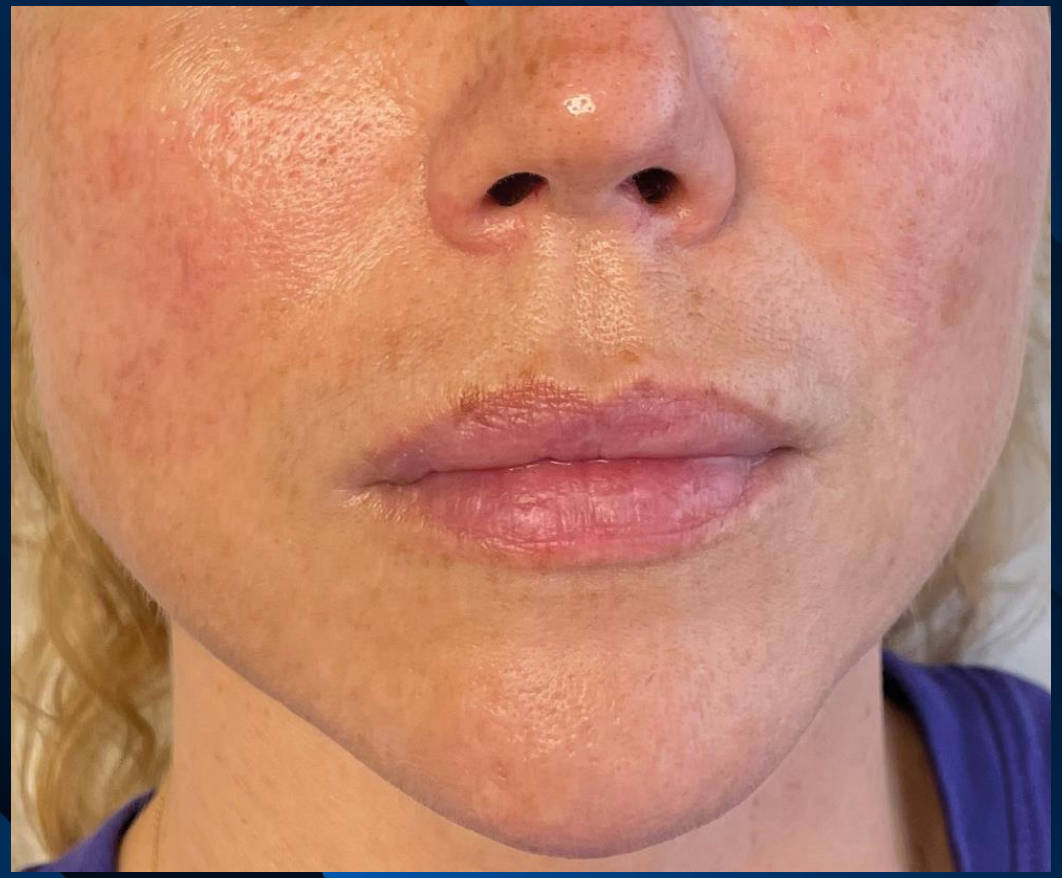




FEMALE | AGE 45

Result after 30 days of treatment



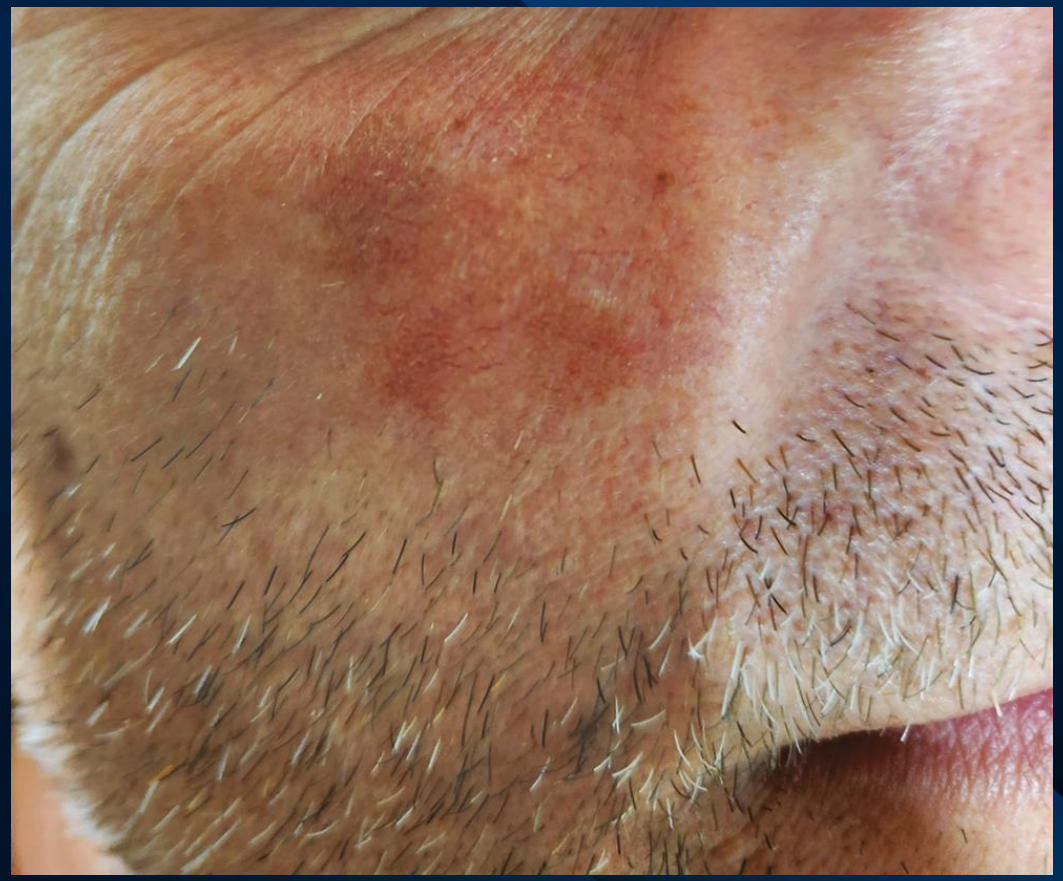


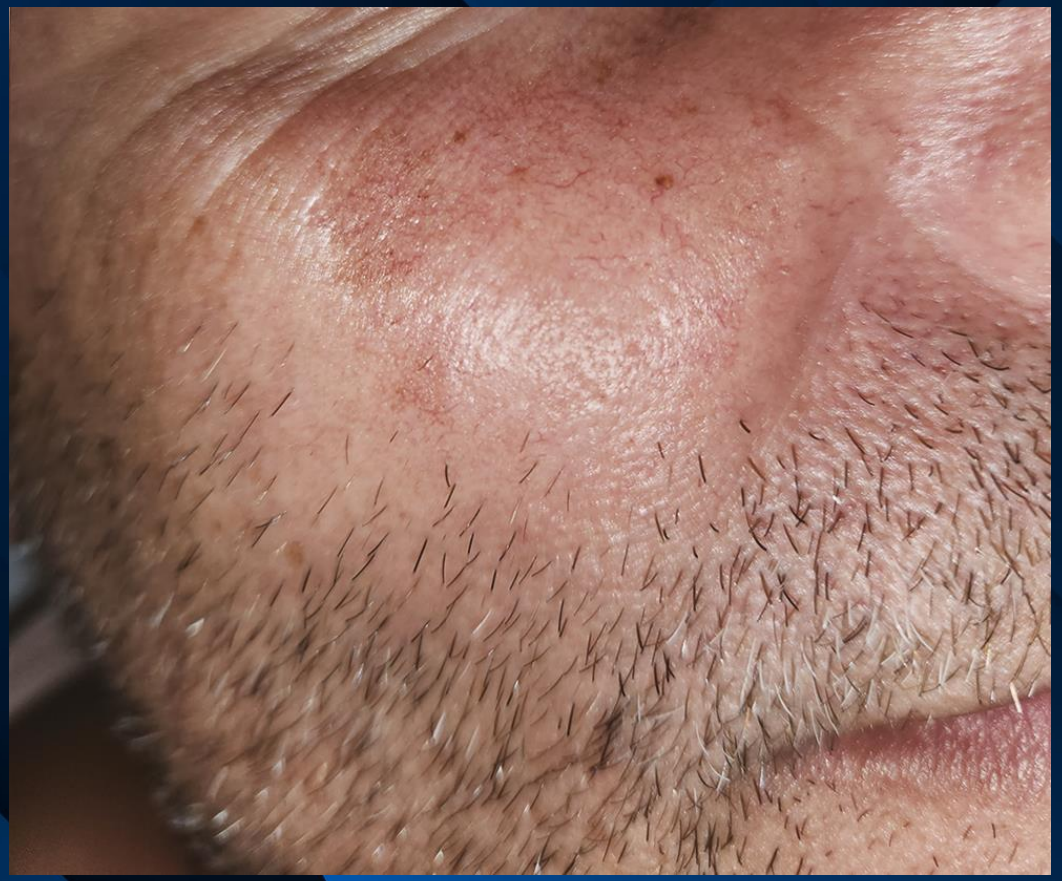
FEMALE | AGE 43

Result after 60 days of treatment

CMed AESTHETICS

Our results

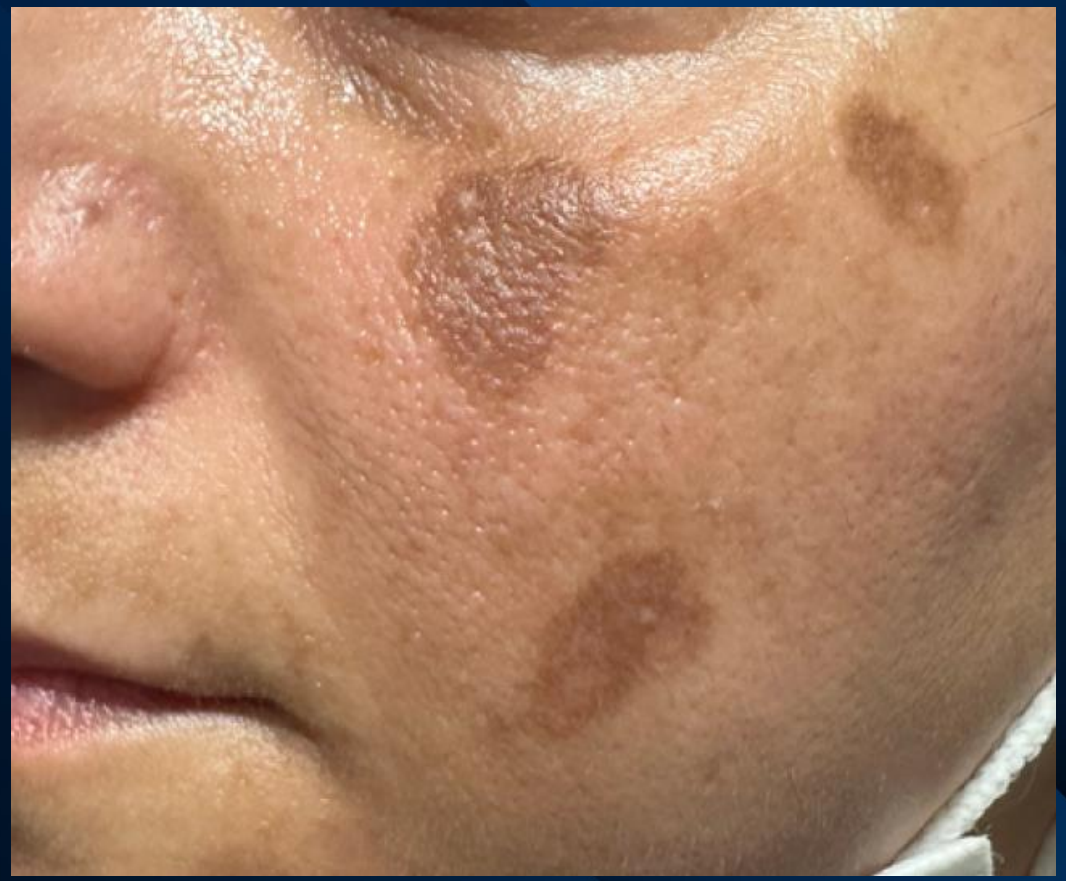


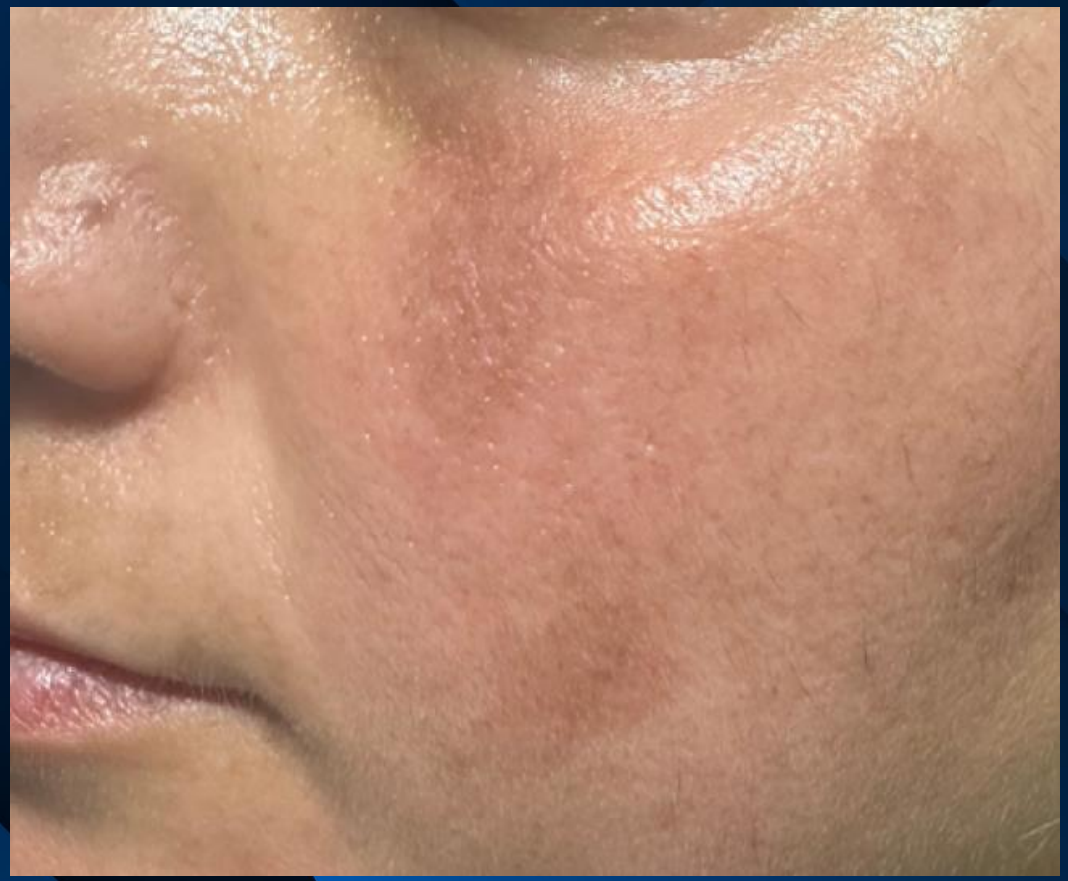


MALE | AGE 51

Result after 30 days of treatment

Our results





FEMALE | AGE 41

Result after 60 days of treatment

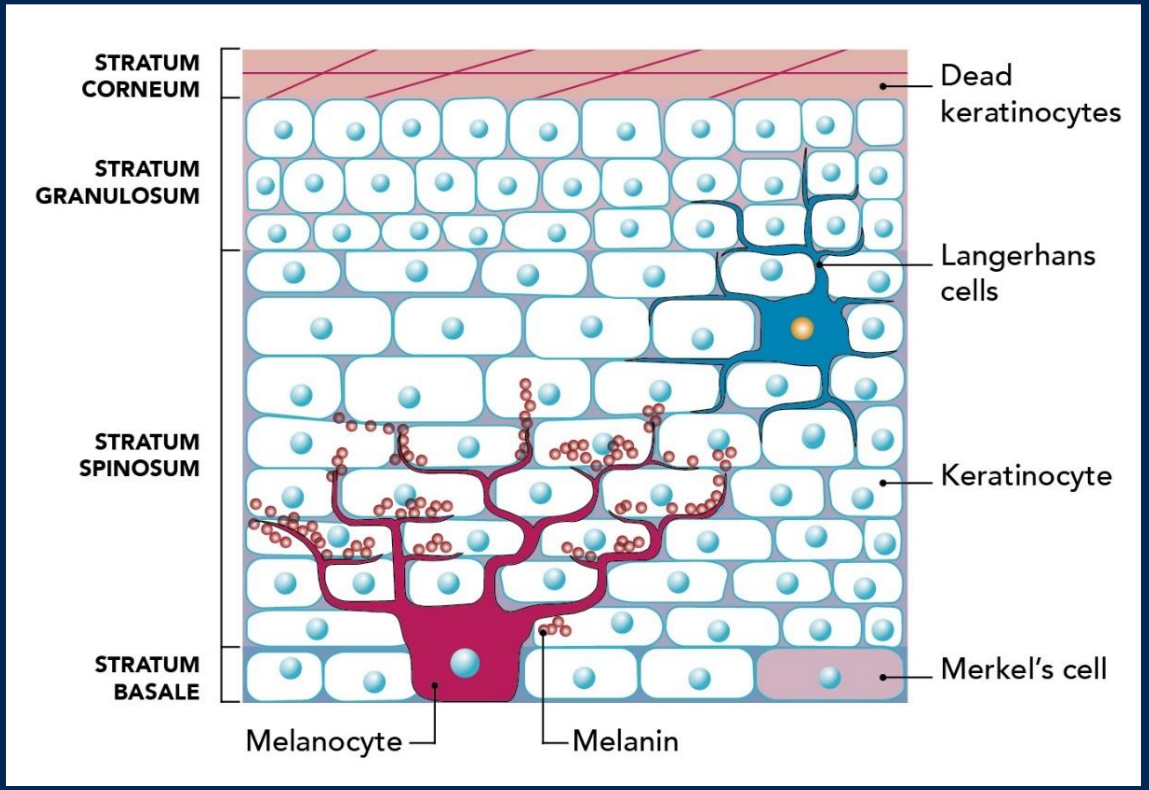
MELANOGENESIS

HYPERPIGMENTATION & SKIN SPOTS

Hyperpigmentation is due to increased melanin production and deposition and is manifested as dark spots on the skin; especially on the face, hands, and other parts of the body often exposed to the sun and difficult to hide.

Hyperpigmentation is caused by an increase of melanin in the basal and suprabasal layers of the epidermis, and it is associated with a normal or high number of melanocytes. It can be caused by various mechanisms, such as melanin transfer from the epidermis to the dermis, and its accumulation within melanophages (relating to pigmentary incontinence). It's commonly seen in inflammatory skin diseases affecting the basal layer and/or junction dermo-epidermal layer.

The main causes of these disorders are ultraviolet light, chronic inflammation, mechanical irritation of the skin, and an anomalous release of the hormone α-MSH, which stimulates melanocytes.



Intrinsic and extrinsic regulation of human skin melanogenesis and Pigmentation C. Serre, V. Busuttil and J.-M. Botto Global Skin Research Center, Ashland, 655, route du Pin Montard, Sophia Antipolis 06904, France

MELANOGENESIS

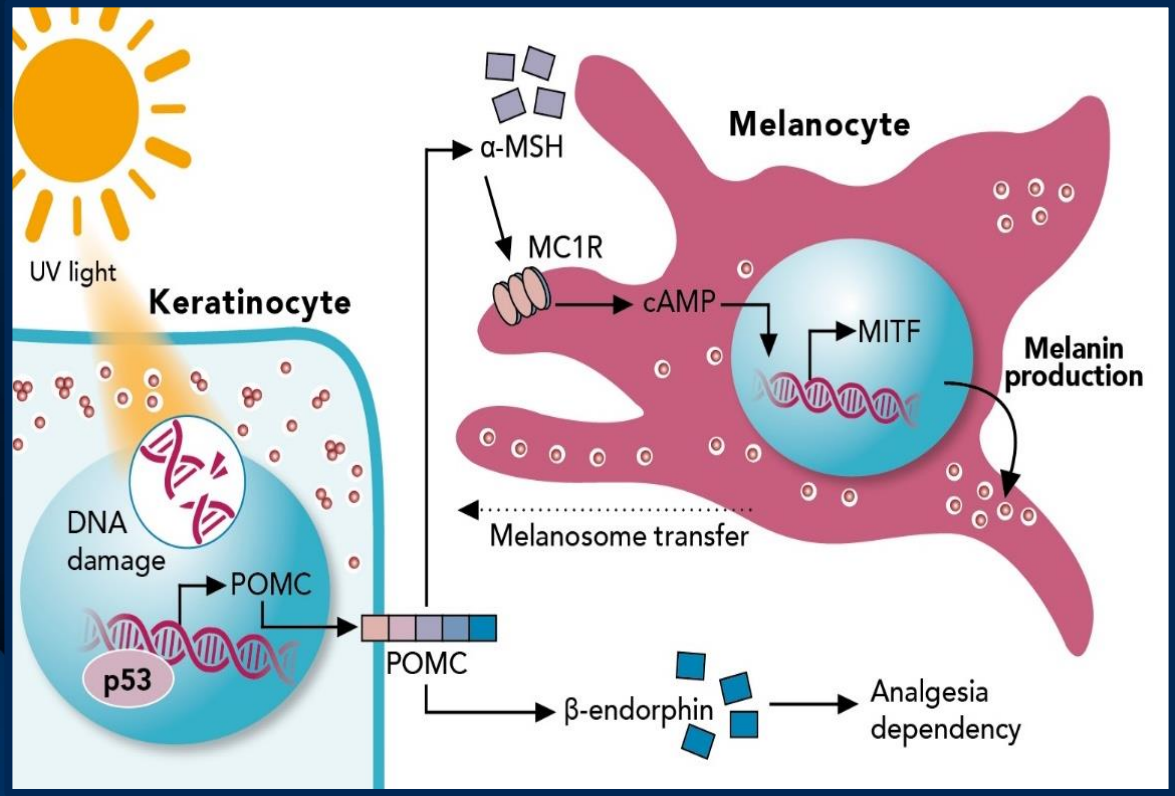
MELANIN BIOSYNTHESIS

Melanin synthesis in the skin is carried out by specific cells present among the basal cells of the epidermis called melanocytes.

These cells, through chemical signals, communicate with the neighboring keratinocytes, creating an epidermal-melanin unit that is responsible for melanogenesis.

Melanocytes produce melanin in particular structures, called melanosomes.

Thanks to an active movement system mediated by the transport proteins Rab (monomeric GTPases), the melanosome manages to reach the terminal portion of melanocyte dendrites and anchor itself to the cell membrane ready to be transferred to the corneocytes.



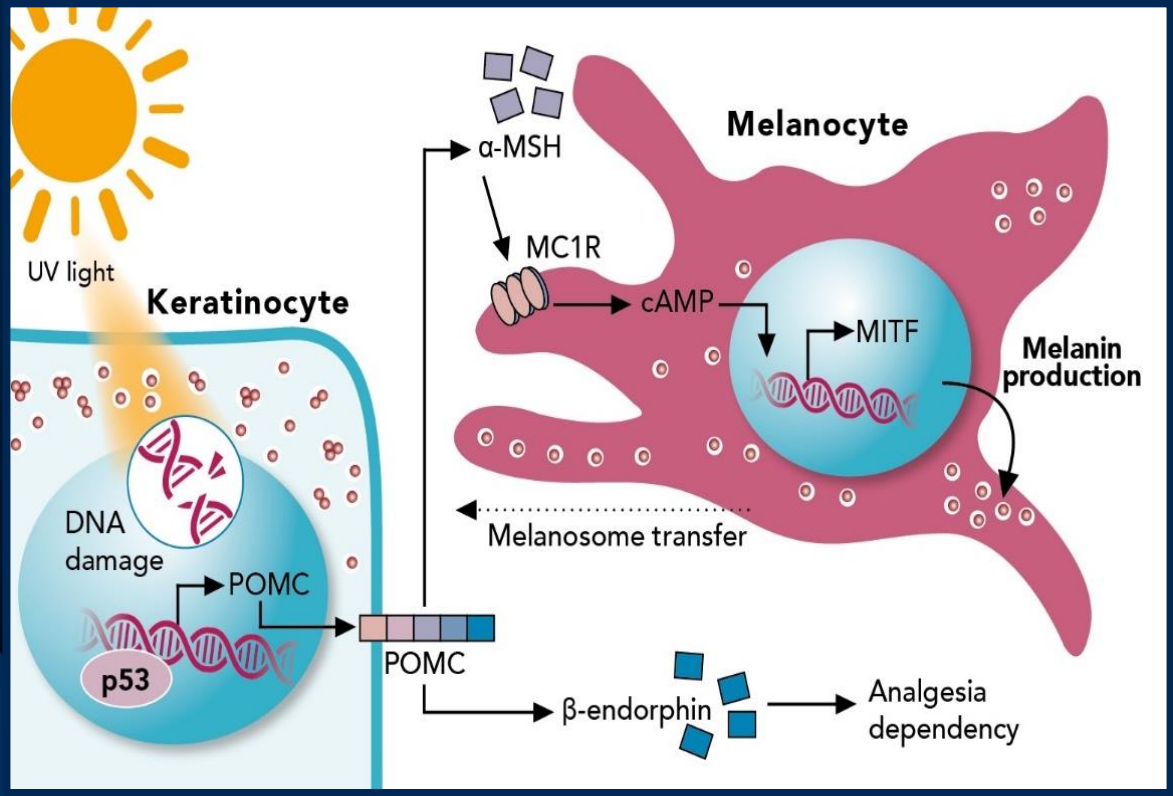
MELANOGENESIS

MELANIN BIOSYNTHESIS

The passage of melanosomes from melanocyte to corneocyte is another active step regulated by PAR2 receptors, activated by the action of a protease, trypsin.

The production of melanin has protective functions from solar damage, therefore its synthesis is activated by biological damage processes.

The damage causes the release of a particular hormone called MSH (melanocyte stimulating hormone) which acts on the melanocyte by activating the synthesis of melanosomes storing melanin and their release to epidermal cells, through the stimulation of specific membrane receptor MC1R.

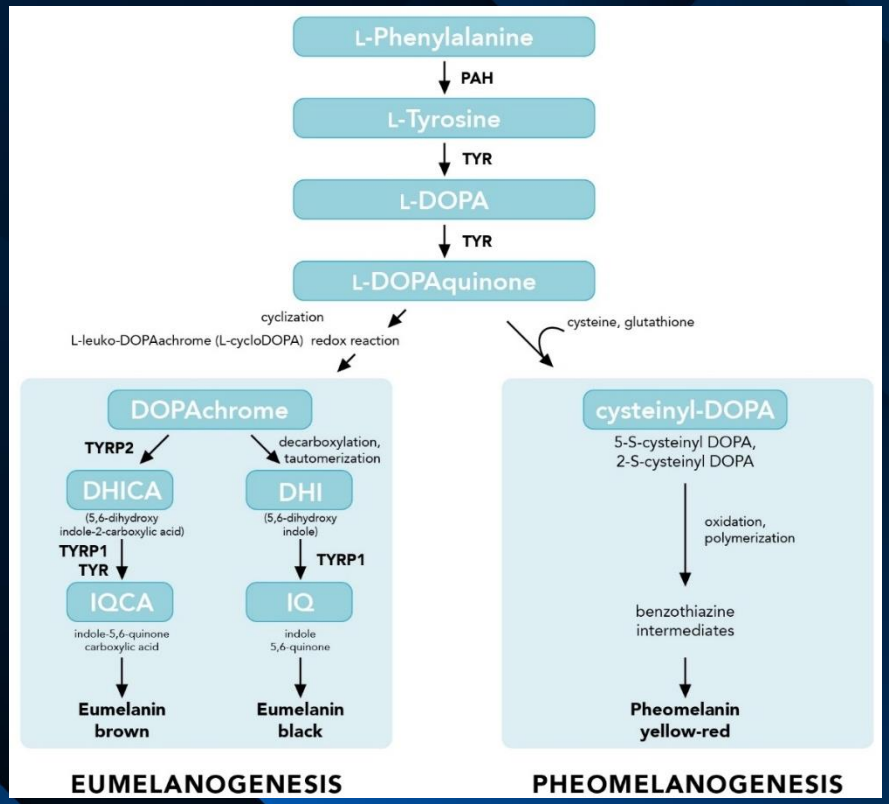


MELANOGENESIS

MELANIN BIOSYNTHESIS

Stimulation of MC1R induces the melanocyte to synthesize melanin as the stimulation of MC1R induces the expression of a transcription factor called MIFT (Microphthalmia-Associated Transcription Factor). This allows the expression of the genes responsible for the synthesis of enzymes necessary for the transformation of tyrosine into melanin.

Starting from aminoacid tyrosine, through the activity of tyrosinase enzyme, DOPA is formed. Then, several further conversions follow to form DOPAquinone, Leuko-DOPAchrome, DOPAchrome, 5,6-dihydroxy indole (decarboxylated and/or carboxylated), indole-5,6-quinone(decarboxylated and/or carboxylated). Eventually polymerization of this last product forms eumelanin.



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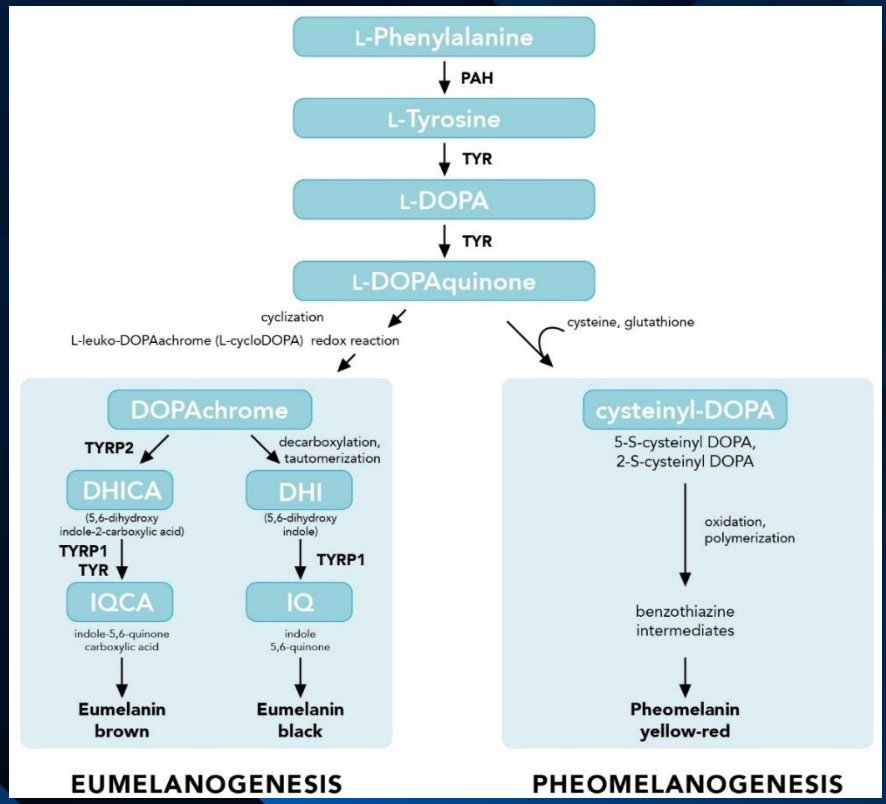
MELANOGENESIS

MELANIN BIOSYNTHESIS

DOPAquinone reacts with cysteine to form cysteinyl DOPA; after oxidation, cysteinyl DOPA undergoes ring closure to yield benzothiazine intermediates that may couple through a peroxidase/H2O2-promoted reaction or tyrosinase-catalyzed oxidation; the multistep process ends with the formation of pheomelanin.

Melanogenic flow is regulated by melanogenic enzymes (tyrosinase, TYRP1, TYRP2), divalent metal cations, activators and inhibitors and other regulatory factors.

Tyrosinase is the rate-limiting enzyme of hydroxylation of tyrosine to L-DOPA while other actors in melanogenesis control the quality and quantity of formed melanin.



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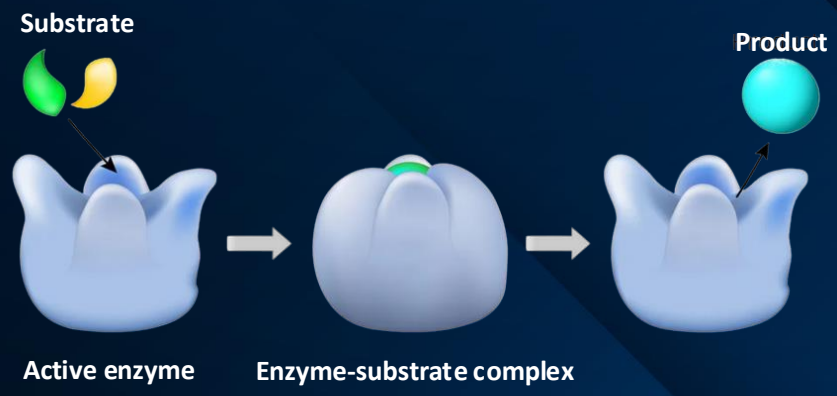
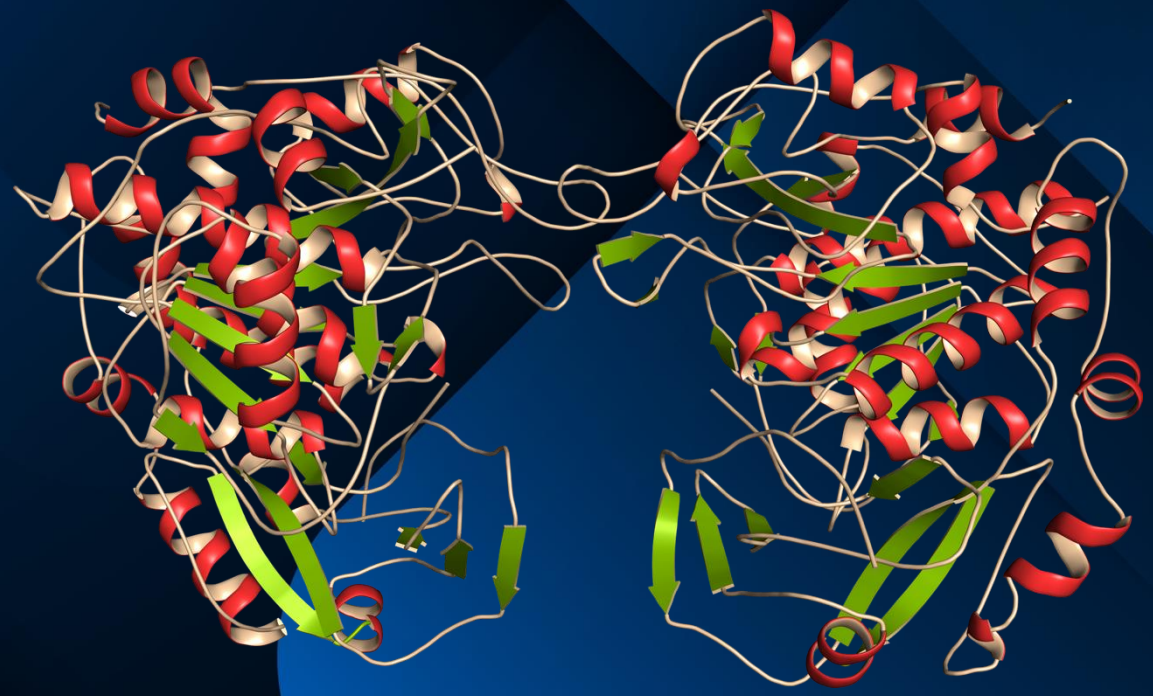
What an enzyme?

Enzymes are proteins that act as biological catalysts by accelerating chemical reactions.

The molecules upon which enzymes may act are called substrates and the enzyme converts the substrates into different molecules known as products.

How do an enzyme works?

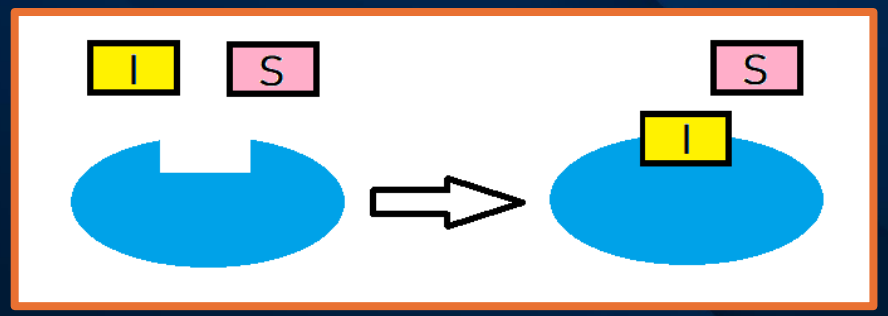




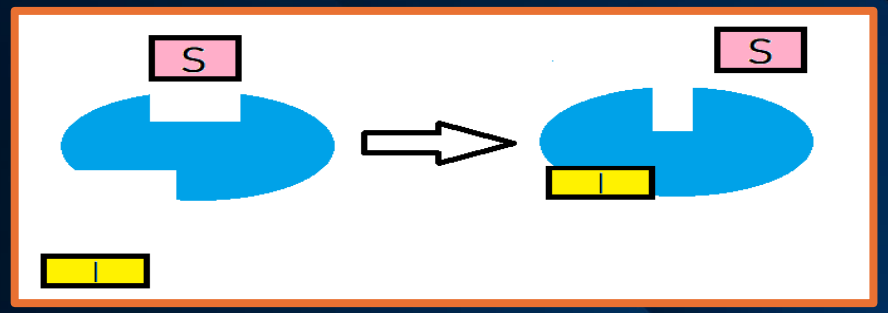
ENZYME

ENZYMATIC INHIBITION

Competitive



Non-competitive



MELANOGENIC ENZYME

Tyrosinase inhibitors

- **TRANEXAMIC ACID:** competitive inhibition of tyrosinase with the substrate Tyr at the binding site.

- **ARBUTIN:** competitive inhibition of tyrosinase with the substrate L-DOPA at the binding site.

- **GLABRIDIN:** example of non-competitive tyrosinase inhibition.

- **AMINOETHYL PHOSPHINIC ACID:** chelates the DOPAchrome preventing further steps of transformation.

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