

DEPIGMENTING & BRIGHTENING SOLUTION

Innovation, safety, efficiency and comfort





MADE IN ITALY



"EVERYTHING WE DO IS FINALIZED TO CONTRIBUTE TO PEOPLE'S WELLNESS AND BEAUTY"



INNOVATION IN THE FIELD OF AESTHETIC MEDICINE, DERMATOLOGY AND PLASTIC SURGERY







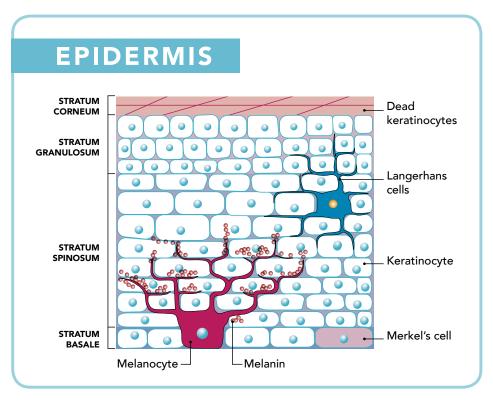
MELANOGENESIS

HYPERPIGMENTATION AND SKIN SPOTS

Hyperpigmentation is due to increased melanin production and deposition, and is manifested as dark spots on the skin; especially on face, hands and other parts of the body more 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 is associated with a normal or high number of melanocytes. It can be caused due to 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

MELANIN BIOSYNTHESIS

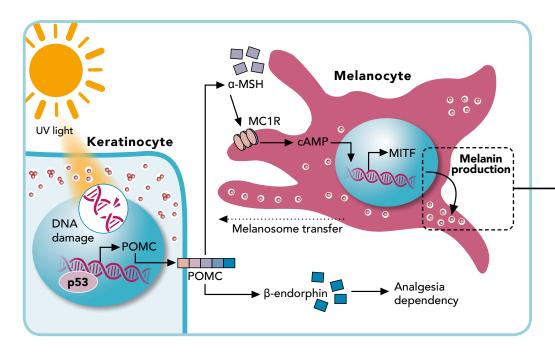
Melanin synthesis in the skin is carried out by specific cells present among the basal cells of the epidermis called melanocytes.

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 themselves to the cell membrane ready to be transferred to the corneocytes.

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.



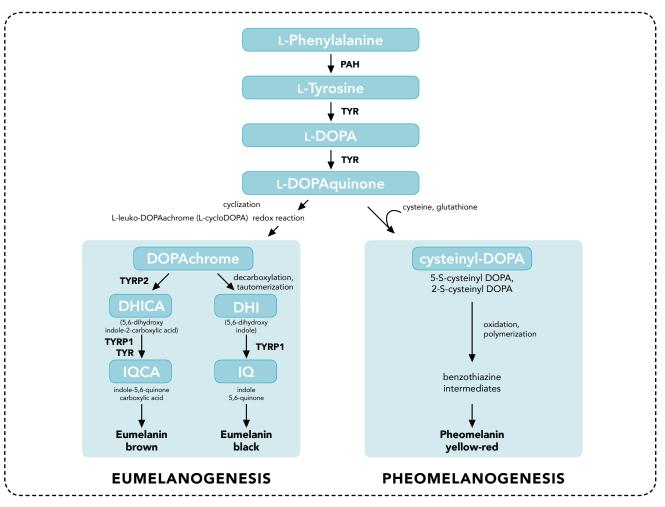
The damage causes the release of a particular hormone called MSH (melanocyte stimulating hormone) which acts on the melanocyte by activating the synthesis of melanosomes storing melanin and their release to epidermal cells. Stimulation of MC1R induces the melanocyte to synthesize melanin as the stimulation 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, throught 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.

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 quality and quantity of formed melanin.



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LUMINESCENS[•] is a protocol with lightening and **depigmenting activity** specifically designed for the treatment of **skin pigmentations** of the face and the rest of the body.

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.

STRENGTHS



Innovative formulation for outpatient mesotherapy treatment with a patent pending.

No waiting time after treatment and no mask to be kept in application for 6-12 hours, unlike other leading products.



No negative impact on daily life.

Cutting-edge formulation, full of active ingredients, made for home treatment with evident results with maximum safety and comfort.

MECHANISM OF ACTION



Strategies for brighter skin.



Blockade of melanogenic enzymes.



Blockade of excess melanin formation.



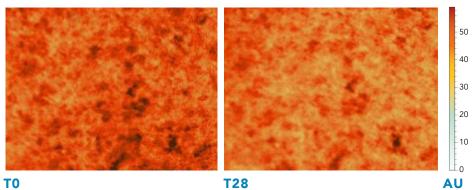
Antioxidant and anti-free radical action.

Increased turnover and hyperpigmentation removal.

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.



IMAGES DETECTED AND ANALYZED WITH MIRAVEX ANTERA 3D®



say that the treatment reduces skin spots.

95% OF PATIENTS

say that the mesotherapeutic treatment is pleasant and does not create discomfort skin.

say that the home product is comfortable.

CMed Aesthetics s.r.l. 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-Clinica Research Insitute.

BEFORE & AFTER TREATMENT



GENDER: FEMALE / AGE 41



RESULT AFTER 30 DAYS OF TREATMENT



GENDER: FEMALE / AGE 43



RESULT AFTER 60 DAYS OF TREATMENT



GENDER: MALE / AGE 51



RESULT AFTER 30 DAYS OF TREATMENT

ACTIVE COMPLEXES

MELANOGENIC ENZYME BLOCKERS

Tyrosinase inhibitors

- OLIGOPEPTIDE-34: Regulation of TYRP-1, TYRP-2, MITF and tyrosinase.
- TRANEXAMIC ACID Involved in anti-plasmin activity and inhibits tyrosinase transcription. (1)
- AMINOETHYL PHOSPHINIC ACID & GLYCYRRHETINIC ACID Inhibit tyrosinase.
- AZELAIC ACID Inhibits tyrosinase and regulates the activity of thioredoxin reductase. (2)
- ARBUTIN Competitive inhibitor of tyrosinase, reducing catalysis of DOPA. (3)
- PHENYLETHYL RESORCINOL Reduces both melanin production and tyrosinase activity. (4)
- SILYBUM MARIANUM EXTRACT Inhibits tyrosinase activity, with strong antioxidant properties (5)
- GLABRIDIN Potent inhibitory effect on not only tyrosinase activity, but also tyrosinase-related proteins. (6)

MELANIN FORMATION AND TRANSPORT BLOCKERS

Copper chelators and MC1R enzyme inhibitors

- ELLAGIC ACID & PHYTIC ACID Chelating effect on copper.
- ACETYL TETRAPEPTIDE-2 Mimics thymopoietin, the youth enzyme.
- POTASSIUM AZELOYL DIGLYCINATE Regulates the synthesis of hyperactive melanocytes. (8)
- BIOMIMETIC PEPTIDES, HEXAPEPTIDE-40 SH-POLYPEPTIDE-76 INHIBITORY Inhibits both tyrosinase and melanin formation.
- \bullet ACETYL HEXAPEPTIDE-1 Regulates pigmentation and melanin production through interactions with skin receptor MC1R. (9)

INCREASE IN CELL TURNOVER

Removal of pre-existing melanin

- CITRIC ACID, TARTARIC ACID, SALICYLIC ACID Exfoliates the stratum corneum, composed of dead cells.
- **RETINOL** Promotes rapid loss of pigment through epidermopoiesis and an increased epidermal turnover. (10)

ANTIOXIDANTS AND DERMO-NORMALIZING AGENTS

Action against pigment-increasing free radicals

- **PEPTIDE CG-TGP2** Has an anti-inflammatory effect.
- GLUTATHIONE Powerful antioxidant capabilities.
- TOCOPHERYL ACETATE Increases antioxidant activity.

INGREDIENTS

MESOTHERAPY | 2x4 mL

PATENTED FORMULA:

Tranexamic Acid Salicylic Acid Aminoethyl Phosphinic Acid Sodium Phytate Rutin Ellagic Acid Oxothiazolidinecarboxylic Acid Vitis Vinifera Leaf Extract Rosmarinus Officinalis Extract Melissa Officinalis Leaf Extract Salvia Officinalis Leaf Extract Camellia Sinensis Leaf Extract Betula Pubescens Twig Extract Acetyl Tetrapeptide-2 Arbutin Sodium DNA Potassium Azeloyl Diglycinate Oligopeptide-34 Glutathion Hyaluronic Acid



MULTI-NEEDLE

DERMA ROLLER

HOME CREAM 30 mL

Tranexamic Acid Aminoethyl Phosphinic Acid Azelaic Acid Phytic Acid Arbutin Oligopeptide-34 Citric Acid Tartaric Acid Nicotiana Benthamiana Hexapeptide-40 Sh-Polypeptide-76 Acetyl Hexapeptide-1 Salicylic Acid Panthenol Silybum Marianum Extract Phenylethyl Resorcinol Glycyrrhetinic Acid Retinol Hyaluronic Acid Glabridin Tocopheryl acetate



MESOTHERAPY

ENDIN





DEPIGMENTING & BRIGHTENING SOLUTION



HOME CREAM* 30 mL DEPIGMENTING CREAM

Home format to continue with professional treatment.

1)Tranexamic acid inhibits melanogenesis partially via stimulation of TGF-121 expression in human epidermal keratinocytes Xiaoxue Xing 1, Zhongyi Xu 1, Li Chen 1, Shanglin Jin 1, Chengfeng Zhang 1, Leihong Xiang 1

2) Melanin hyperpigmentation of skin: melasma, topical treatment with azelaic acid, and other therapies A S Breathnach 1 Affiliations expand • PMID: 8654129

3) Arbutin as a Skin Depigmenting Agent with Antimelanogenic and Antioxidant Properties. Yong Chool Boo

4) Characterization and topical delivery of phenylethyl resorcinol Y. Zhang, B. C. Sil, C.-P. Kung, J. Hadgraft, M. Heinrich, B. Sinko, M. E. Lane

5) The treatment of melasma by silymarin cream Tagreed Altaei BMC Dermatology volume 12, Article number: 18 (2012) Cite this article

6) The inhibitory effect of glabridin from licorice extracts on melanogenesis and inflammation T Yokota 1, H Nishio, Y Kubota, M Mizoguchi

7) Efficiency of ellagic acid and arbutin in melasma: a randomized, prospective, open-label study. Ilgen Ertam 1, Basak Mutlu, Idil Unal, Sibel Alper, Bijen Kivçak, Ozgen Ozer

8) Maramaldi G, Esposito M. Potassium Azeloyl Diglycinate. Cosm & Toil. 2002;117:3. [Google Scholar] [Ref list]

9) Up- or Downregulation of Melanin Synthesis Using Amino Acids, Peptides, and Their Analogs Yong Chool Boo1,2,3
10) Gupta AK, Gover MD, Nouri K, Taylor S. The treatment of melasma: A review of clinical trials. J Am Acad Dermatol. 2006;55:1048–65.

ESTHETICS

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INFORMATION MATERIAL FOR THE MEDICAL PROFESSION

*Professional product for home use. Product to be used under professional opinion, not intended for the retail channel