RELATA TECHNICA

International journal on dermopharmacological research,
dermopharmaceutical technology and related cosmetic subjects

THE NEW APPROACH TO SUN PROTECTION: FILTERING, DISSIPATING, MODULATING

Published on Relata Technica Web Site
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The new approach to sun protection: filtering, dissipating, modulating

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Reading time: 18'

ABSTRACT

The most recent literature shows that the damage caused by solar radiation continues to be significant even in the hours following sun exposure. This mechanism is mediated by melanin.

Humans are more exposed to solar radiation damage than animals because their melanin is not found in the hair shaft as in other mammals and bird feathers but is dispersed in granules in the epidermis, the melanosomes, at the basal layer and in the overlying layers. In addition, melanin absorbs radiant energy but in the hours following exposure, it transmits the energy to contiguous keratinocytes by direct contact. Researchers at Vevy Europe have therefore thought of synergistically using some of the substances already developed to dissipate this type of oxidising energy in the hours following exposure and reduce the potential damage to DNA and other cellular structures.

This study therefore proposes the use of different ingredients combined synergistically, to be administered appropriately vehiculated, before, during and after sun exposure with the aim of selectively neutralising and modulating the effects of solar radiation during photo exposure and in the following hours.

INTRODUCTION

As is well known, solar radiation, which is indispensable to life, carries harmful effects as well as beneficial effects.

During evolution, living beings have developed very sophisticated defence strategies against solar radiation, at the subcellular and molecular level. However, due to the longer average lifespan of humans and their increased exposure to sunlight, these mechanisms are no longer sufficient to protect the skin.

Recent studies have shown that cell damage occurs both during UV exposure and several hours after exposure due not only to UVA and UVB rays but also UVC.

Until now, it was always thought that melanin was an effective, natural and sufficient defence system for the skin, but recent studies[1,7,8] have shown that there is instead a harmful effect caused by this endogenous pigment.

Melanin, in an excited state after exposure to the sun, produces oxygen molecules with an altered electron in its interior, in a state called 'quantum triplet statum', i.e. a state of excitation that gives up energy to the DNA and surrounding proteins, damaging them.

The underlying mechanism for the formation of this excited oxygen is generated by the action of UV rays via the enzymes Nitric Oxide Synthase (NOS) and Nicotinamide Adenine Dinucleotide Phosphate Oxidase (NADPH Oxidase), which generate $NO^{\cdot}$ nitric oxide radicals and superoxide radical ions $O_2^{\cdot -}$ which combine to generate Peroxynitrite ($ONOO^-$), a powerful oxidant[7].

Solar radiation

Solar radiation consists of the mixture of all wavelengths of visible light in such proportions that it is perceived as white light. This total radiation consists of 50% visible light, 40% infrared and 10% ultraviolet light.

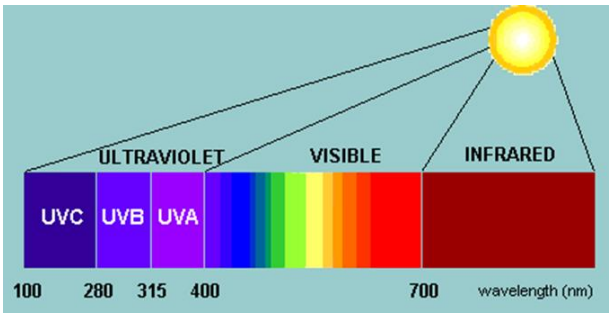


Figure 1: *Light/wavelength spectrum.*

Ultraviolet light is broken down as follows:
UVA 315-400 nm
UVB 280-315 nm
UVC 100-280 nm

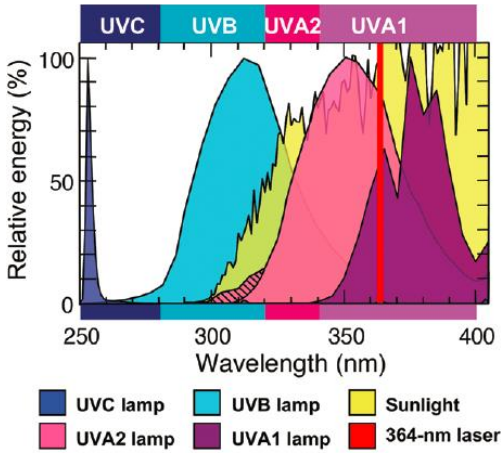


Figure 2: *Ultraviolet spectrum (Ref. Photochem Photobiol Sci. 2018 Dec 5;17(12):1861-1871).*

UVA accounts for about 9.5% of the total solar radiation (Fig.3); it passes through the epidermis, passing the basal layer and melanocytes to reach the dermis. This radiation is responsible for tanning (Fig.4); it is not filtered by glass and 50% of exposure occurs in shaded areas.

UVBs are energetically more charged than UVAs, although they are blocked more on the surface at epidermal level and therefore do not cross the basement membrane. They are responsible for erythema and DNA mutations (Fig.4). These are the ones that have the greatest biological cytotoxic and mutagenic effect and that account for about 0.5% of total solar radiation (Fig.3).

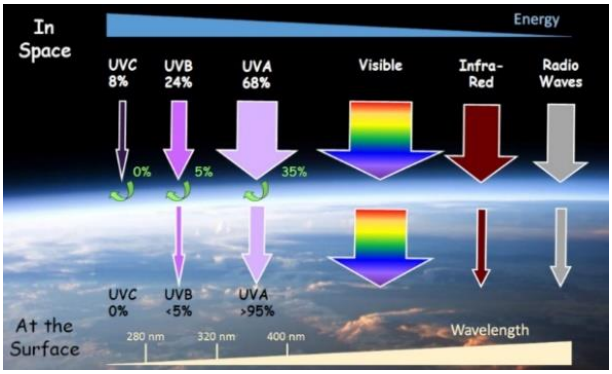


Figure 3: *Composition of solar radiation in space and after passing through the Earth's atmosphere. (Ref. encyclopedie-environnement.org).*

UVC rays are the most energy-intensive rays and are therefore the most dangerous as they transfer more radiant energy to the cells (Fig.4). They are particularly harmful to health as they have a high carcinogenic power. They have the shortest wavelength but the highest energy and are blocked by the stratospheric ozone layer, which is not always present and efficient over all areas of the planet[5].

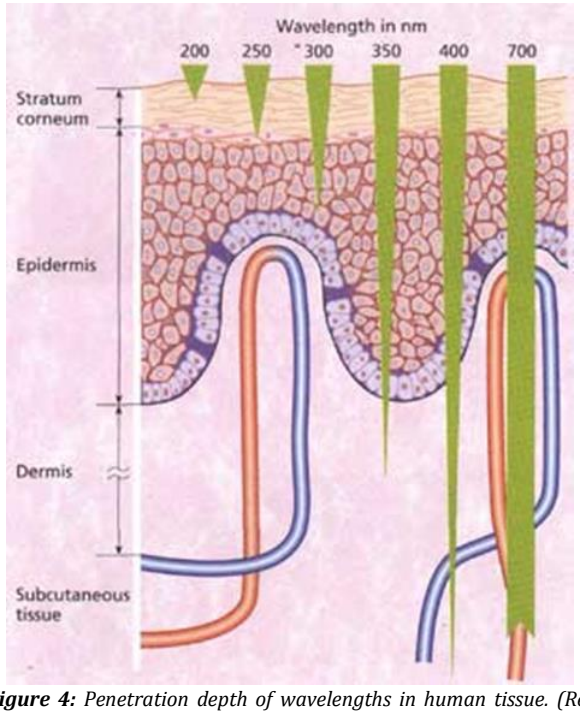


Figure 4: *Penetration depth of wavelengths in human tissue. (Ref. Mauro C. Moreira, Ricardo Prado and Alexandre Campos: August 23rd, 2011; Applied Biomedical Engineering).*

There are also variables such as season, altitude and time of exposure. As early as 1000 metres above sea level there is a 15-20% increase in UVB. In addition, the reflective surfaces of the environment increase the intensity received by the skin: clouds and snow reflect up to 80% of UV radiation, sand 20% and water 9% (Fig.5).

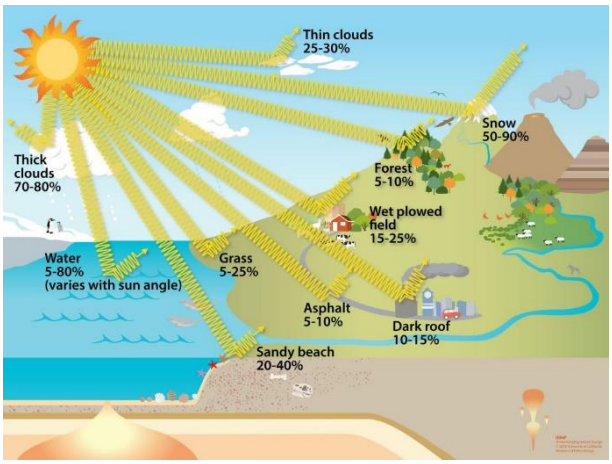


Figure 5: *Different reflection/absorption of sunlight (Ref. ugc.berkeley.edu).*

Effects of sunlight on Man and his skin

Man, unlike almost all other mammals, has almost no fur. This skin annexe formed by a collection of hair formations containing melanosomes (within which melanin is found) effectively protects animals from excess radiation by an 'umbrella mechanism' external to the skin and not endocellular. In our species, however, melanin is dispersed in the epidermis by exocytosis from melanocytes. Its function, however, is not only protective but also induces damage at the atomic level by generating 'excited electrons'.

Solar radiation has both positive functions for humans, such as the transformation of skin cholesterol into active vitamin D (UVB) and circadian and circannual synchronisation (sleep-wake and seasonal rhythms), as well as very negative phenomena such as the formation of Thymine dimers and Cyclobutene Pyrimidine dimers (CPDs) at the level of skin DNA[9], causes of mutations in both melanocytes and epidermal keratinocytes, precursors of tumour neoformation[3,4].

In addition, pheomelanin in fair-skinned individuals increases, when stimulated, the release of histamine that contributes to sunburn.

The effects of chronic exposure to sun radiation also include photoaging, epidermal thickening and damage to the dermal extracellular matrix[3,4]. In fact, skin ageing is mainly due to the breakdown of collagen and elastin molecules by UVA radiation. These, in addition to producing free radicals, induce the formation of metalloproteases that degrade the extracellular collagen matrix and hydrolyse the procollagen, fibrillar collagen and elastin molecules.

To sum up, the main pathological phenomena due to sun exposure are:

- acute erythema, i.e. the production by keratinocytes of inflammatory substances that cause vasodilation of the microcirculation of the papillary dermis[10];
- hyperkeratosis, which consists of thickening of the epidermis especially in the most exposed areas of the body such as the nose and forehead;
- photodamage, actinic elastosis, ageing and wrinkles[10];
- production of free radicals;
- mutagenic-carcinogenic effect.

Vitamin D synthesis

Humans are unable to synthesise sufficient vitamin D without exposure to sunlight as nutritional intake can only provide a small percentage of what is needed. Solar UVB radiation (wavelength between 280 and 315 nm) penetrates the skin and converts 7-dehydrocholesterol (present in the skin) into provitamin D2 (ergocalciferol), which is rapidly converted into vitamin D3 (cholecalciferol), keeping in mind that with advancing age this conversion process would appear to be less and less efficient[11], and therefore any possible supplementation would need to be recalibrated accordingly. Since any excess provitamin D3 or vitamin D3 is destroyed by sunlight itself, excessive exposure to sunlight never causes vitamin D3 intoxication. Vitamin D in its active form is present in the blood and is indispensable to life because it regulates the concentration of calcium and phosphorous in the blood and bones, and modulates the reactivity of the immune system.

It is therefore crucial to find the right balance between sun exposure and sun protection, as total shielding or non-exposure are not completely viable solutions.

Melanins

The melanin synthesis reaction is encoded in the structural genetic makeup.

Melanin synthesis, stimulated by UV exposure, is an example of epigenetic activation that 'switches on' the structural genes that lead to melanin production.

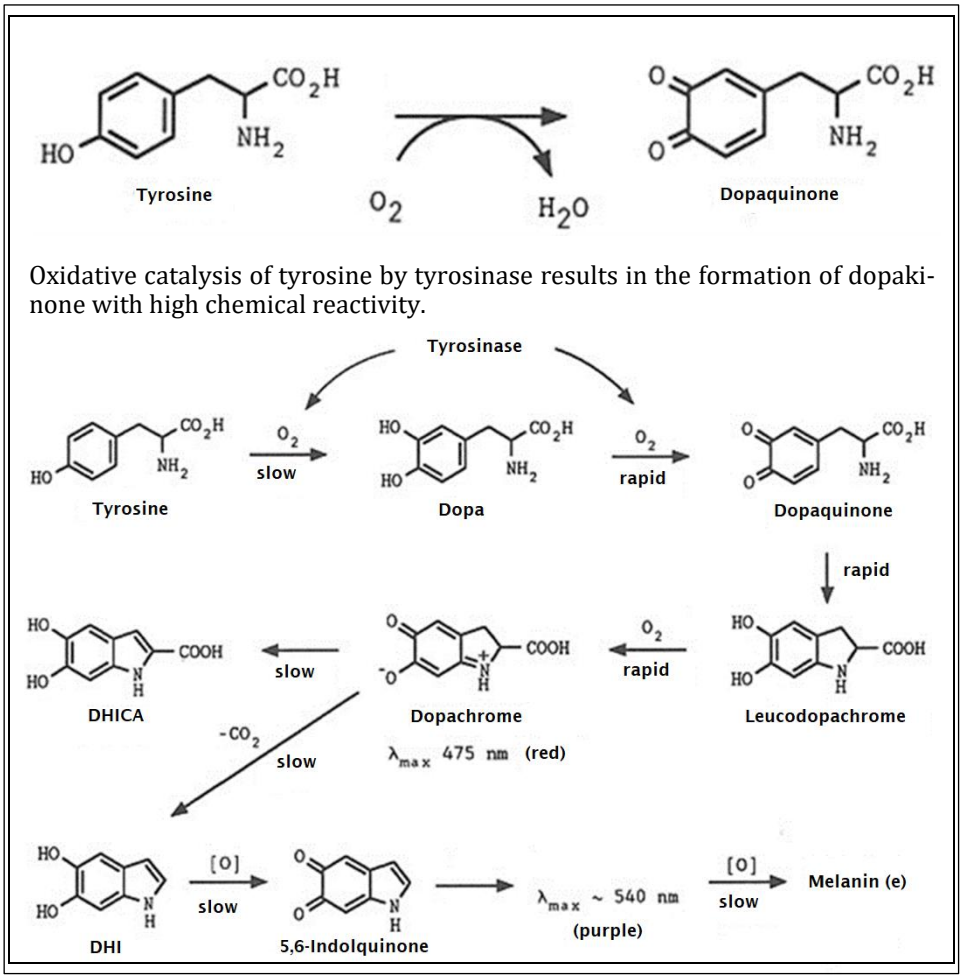


Figure 6: *Scheme of melanogenesis according to Raper-Mason.*

Melanocytes

Melanocytes are neuroectodermal-derived dendritic cells from the neural crests of the embryo and therefore not epithelial elements (Fig.7,8). During embryonic life, they migrate into the skin and establish themselves in higher numbers in the basal and spinous layer of the epidermis. They produce melanins that are packaged in cytoplasmic organelles called melanosomes (melanogenesis). During UV irradiation, the melanosomes transferred to the keratinocytes assemble above the nucleus (capping phenomenon) thus protecting the DNA of the keratinocyte nucleus.

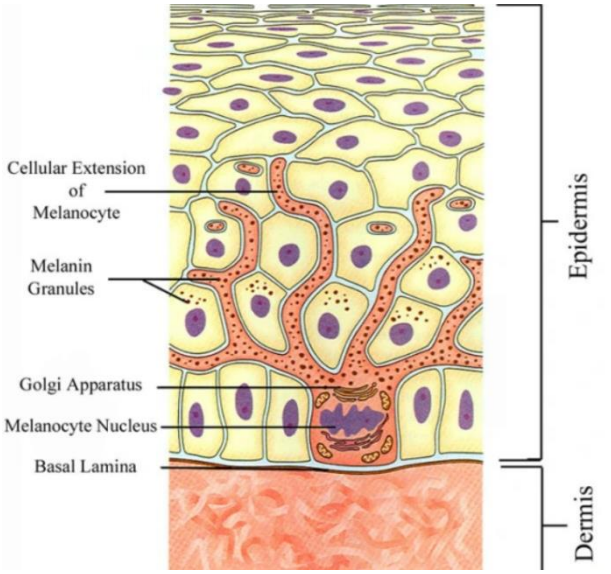


Figure 7: *Melanocyte at the level of the basal lamina. (Ref: odobio-chem.wordpress.com)*

The melanosomes are transported (cytokinesis) by exocytosis to the epidermal keratinocytes of the basal layer (Fig.8,9) and subsequently, with the growth of other basal cells, pass to the more superficial layers resulting in tanning. Melanin is then lost through desquamation of the stratum corneum.

Melanins can be of two types: eumelanins (dark brown or black) and pheomelanins (yellow to orange). Under the influence of UV light, melanin synthesis increases and its transfer to the keratinocytes is accelerated.

Melanin production is an adaptive response of the body to prolonged exposure to the sun. In this way, melanocytes produce inducible pigmentation (epigenetics), which highlights each individual's ability to develop a tan, a natural adaptive skin-protection mechanism.

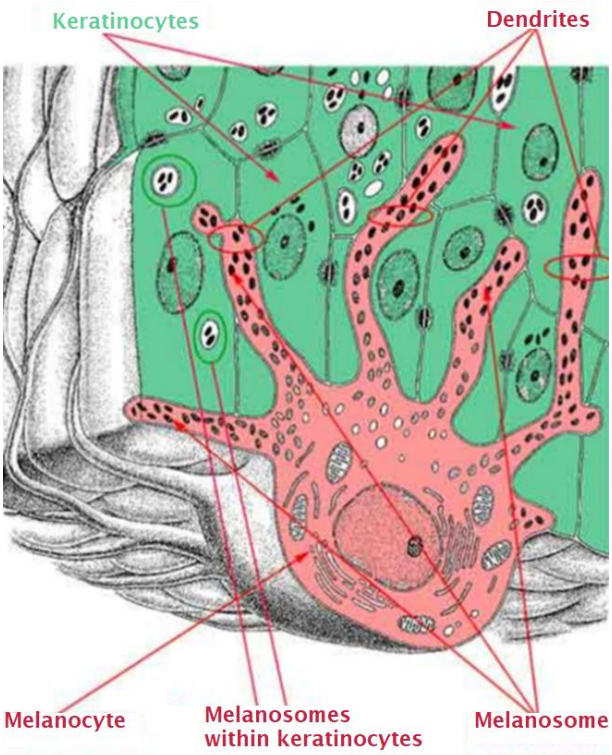


Figure 8: *Melanocyte and melanogenesis (Rif. Junqueira-Carneiro, 04).*

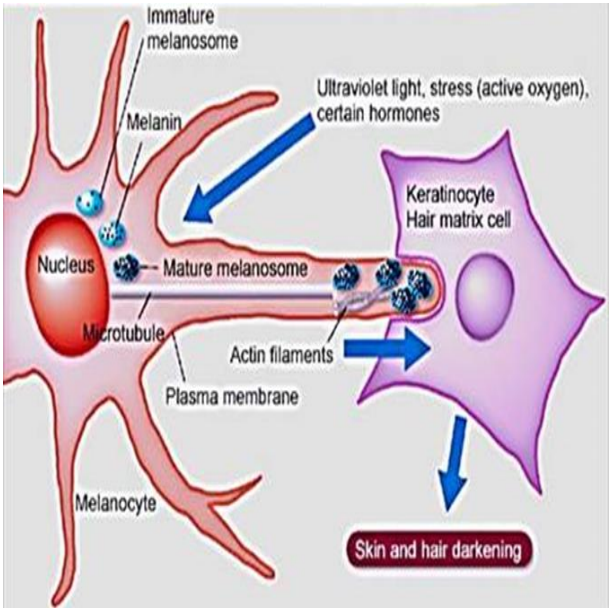


Figure 9: *Melanocyte and melanogenesis.*

Melanin: not only protection

The main function of this pigment, which is present in various concentrations in different human phototypes, is therefore protective.

Research has recently been published (Brash D.E.)[1] that shows that following exposure to UV radiation within the melanin itself, a potentially very dangerous electro-chemical excitation phenomenon occurs, i.e. the production of an oxygen molecule with a 'quantum triplet statum' electron, i.e. an excited state of a supernumerary electron (generated with the energy of a UV photon) (Fig.10). This energy is transferred to the DNA in a manner independent of previous sun exposure: it is a highly reactive excited electron that immediately produces Cyclobutane Pyrimidine dimers (CPDs) in the DNA of the keratinocyte nucleus.

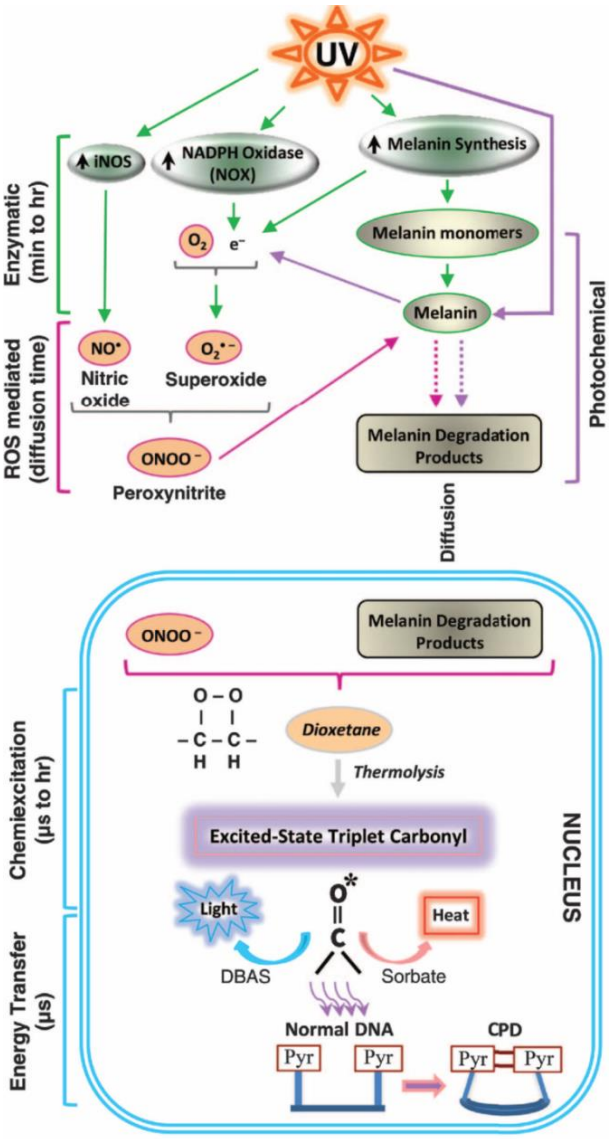


Figure 10: *Mechanism of dark CPD generation in melanocytes, by chemo-excitation, with melanin as an active participant (Ref. Science, February 2015, 347(6224):842-847).*

The study also shows that solar radiation affecting melanin produces superoxide radical ions ($O_2^{\bullet-}$) even hours after exposure to sunlight and that this production is highest after three hours and remains high for the next forty-eight hours.

Prolonged photo-excitation is very dangerous as it is potentially mutagenic (first step towards the possible development of tumours) for keratinocytes as well as melanocytes. This electromagnetic activation of oxygen is caused with the same intensity by both UVA and UVB radiation.

Therefore, the two types of radiation prove to be equally dangerous. It must also be taken into account that UVA rays are not filtered out by the windows and continue their action even when you are inside the room.

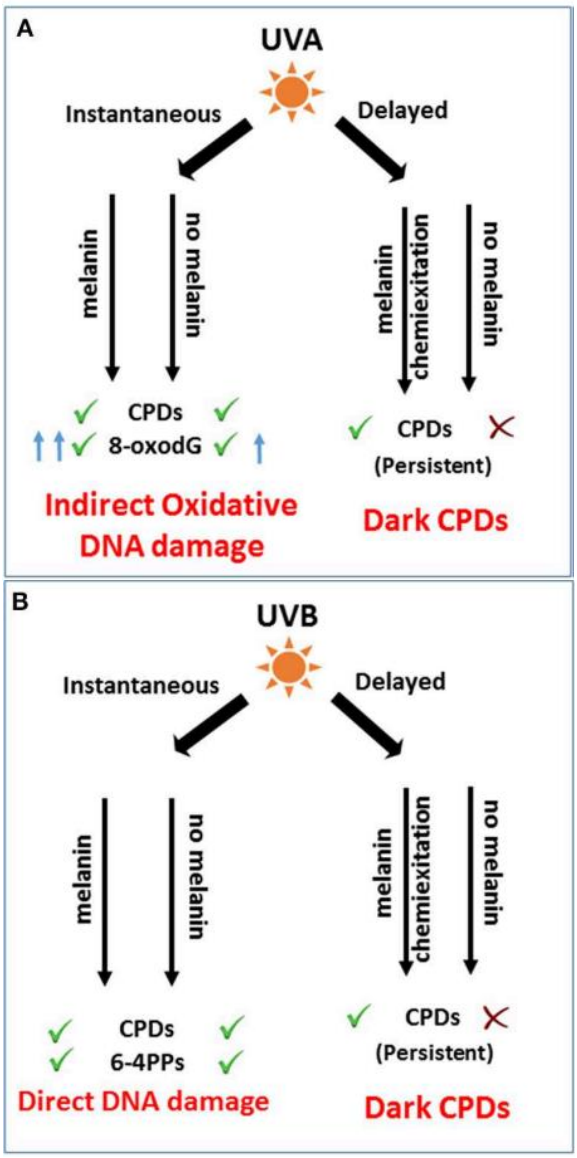


Figure 11: *Role of melanin in DNA damage and melanomagenesis induced by the immediate and delayed effects of UV (Ref. Frontiers in Physio-logy, October 2015 | Volume 6 | Article 276).*

Experiments on K14-Kitl mice[1] exposed to UVA show that the levels of Thymine dimers (the cause of mutation) two hours after exposure to radiation are three times higher (Graph 1) than those present immediately after exposure.

For example, during a day in the open air, there are several moments of exposure to the sun's rays at different times and with different radiation intensities (sum of radiation). The electromagnetic excitation mechanisms that continue for hours after exposure thus result in an ever-increasing sum of effects (Graph 2).

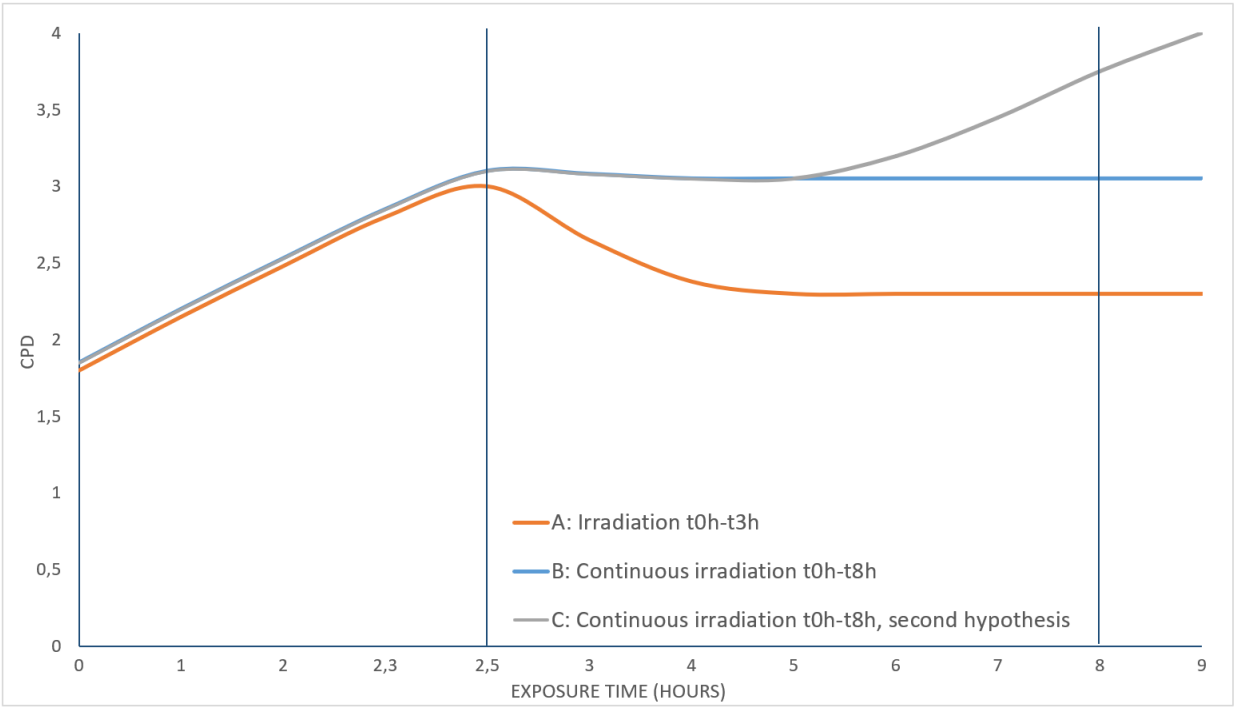
Excessive pigmentation and the possible accumulation of melanin in the form of skin spots lead to an increased amount of melanin, which is more likely to have an inflammatory and potentially mutagenic effect for a long time, even after exposure to the sun.

In light of new findings on the subject, it is therefore recommended to develop a non-excessive tan, free of spots and melanin pigment accumulation[1,2].

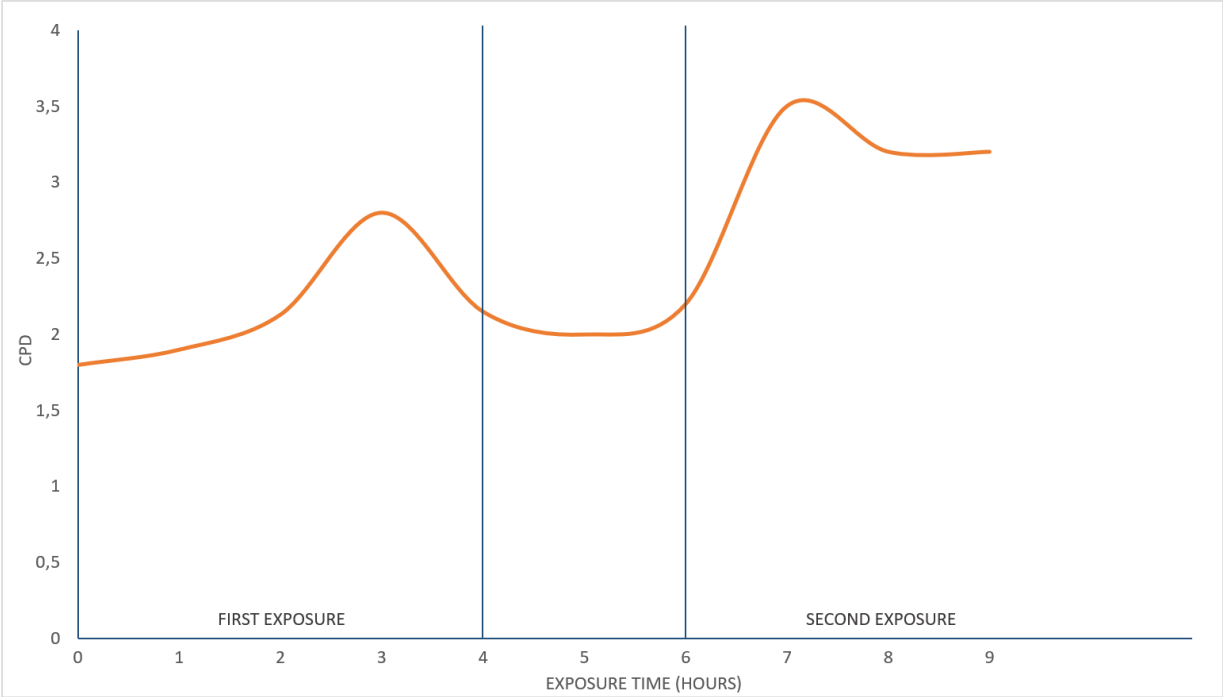
Studies have also shown an increased development of superoxide radical ions ($O_2^{\cdot-}$) by molecules of pheomelanin, the pigment present in blond and fair-skinned individuals (phototypes 1 and 2), compared to those with a darker complexion (phototypes 3-4-5), hence the need for extra care in these individuals.

On the basis of the available scientific data (Diagram 1), our subsequent deductions show the development over time of successive and prolonged exposure to the sun within the same day (Graph 2)[1,2].

The two comparison graphs show in the ordinates the increase in CPD (Cyclobutane Pyrimidine dimers) under the two indicated conditions (A and B).



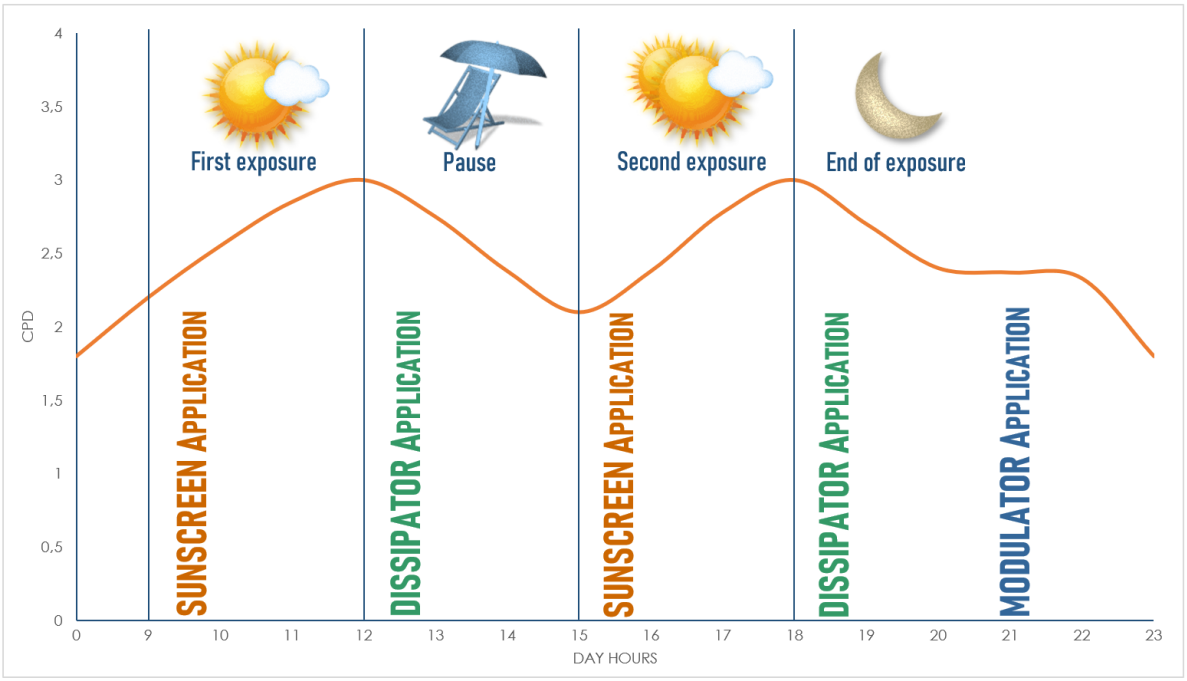
Graph 1
A: amount of CPD after irradiation from T_{0h} to T_{3h}
B: amount of CPD after irradiation from T_{0h} to T_{8h}
C: amount of CPD after irradiation from T_{0h} to T_{8h}, second hypothesis
Graph 1 is based on studies (1,2) and is not supported by our additional experimental data.



Graph 2
Amount of CPD after sum of irradiations from (T_{0h} to T_{3h}) plus (T_{6h} to T_{8h}), with interval from (T_{3h} to T_{6h}). Graph 2 is based on studies[1,2] and is not supported by our additional experimental data.

In light of this important data available in the scientific literature, the phenomenon of tanning needs to be re-considered from this new angle.

Some strategies for the formulation of sun products with an emphasis on energy dissipation are proposed below.



Graph 3
Amount of CPD assumed after administration of dissipator in the exposure interval (3-hour break).

COSMETIC TECHNIQUE

Megasol® Sun Protection System

Synergetic and time-controlled protection according to the three new fundamentals of sun protection: filtering, dissipating, modulating

When formulating a sunscreen product, the synergistic effect of the various ingredients must be evaluated, but also it must be ensured that the sunscreen is not chemically modified due to the interaction between the various constituents and thus loses its effectiveness[6]. Indeed, there is a risk of protection decay due to incorrect or inappropriate combinations of the substances used.

Active ingredients optimisation and formulation stability

Behind every successful active there is a valid vehicle and delivery system. The actives must be enabled to reach their target and fully perform their intended functions. Therefore, formulations that include actives should be stable under the conditions of use, which in the case of sunscreen products are often complicated, and capable of releasing the actives where and as intended; they should also be eudermic, i.e. not alter the complex biochemistry of the skin.

Finally, they should offer a good skin feel and give the skin a pleasant appearance.

Among the many ingredients from Vevy Europe that meet these requirements, here are some that are of specific use in the formulation of high-performance sunscreen products.

Xalifin®-15

Bioemulgoid O/W, non-enzymotoxic, with HLB=12, compatible with physiological skin metabolism. Characterised by excellent vehiculating and release capacity of the active ingredients. It confers high stability to the final product, even in the presence of salts, electrolytes, acids, essential oils and silicones, bright white appearance and high Skin Feel Index.

Cetacene®

Non-conventional multifunctional, non-occluding, plasticising, homogenising and dispersing wax for insoluble matters. Helps to maintain better hydration of the upper epidermal layers, proving very useful after exposure to air and sun. Helps to increase the stability of the finished product, without changing its viscosity. Gives the product a silky, velvety touch and is an excellent emollient. Optimises the transport of actives.

Megasol® Sun Protection System: filtering

MEGASOL® 3D

A broad-spectrum sun filter, consisting of a calibrated, high-performance mixture of four sun filters. This new compound has been specially designed to cover 44.5% of UVA, 44.5% of UVB and 11% of UVC radiation (in silico test).

In addition, it is a worldwide ingredient.

It can be combined with Titanium Dioxide and/or Zinc Oxide in both micro and nano forms to synergistically achieve SPF protection of even 50+.

Table 1 shows the filtering capacities of some possible combinations of Megasol 3D.

Product type	% MEGASOL® 3D	% Titanium Dioxide	in vivo SPF	Labelled Category
O/W Solid Emulsion	16	11	60	Very High Protection
O/W Solid Emulsion	10	6	30	High Protection
O/W Fluid Emulsion	10	-	15	Medium Protection
Oily Gel	10	-	15	Medium Protection

Table 1

Megasol® Sun Protection System: dissipating

MEGASOL® E-SINK

It aims to disperse the excess thermal and photo-electric energy radiated by the sun, which, captured by the melanocytes in the first instance, is transmitted in the second instance (post-exposure) to the neighbouring cells.

The keratinocytes, but also Langerhans' and Merkel cells of the epidermis and dermis cells, fibroblasts and immunocompetent cells, suffer this excess energy by oxidising and causing DNA damage. In addition, the extracellular matrix of the dermis, consisting of an amorphous component (mucopolysaccharides and glycosaminoglycans) and an extracellular fibrillar component (collagenous, reticular and elastic), also suffers CPD (Cyclobutane Pyrimidine dimers) damage, as protein molecules easily break down under the effect of this excess energy. This contributes considerably to skin ageing.

Megasol E-sink, a calibrated mix of specialised active ingredients for dissipating heat and excess photoelectric energy accumulated during sun exposure, is especially suitable for after-sun products: it helps prevent skin ageing and its active ingredients can be grouped into three functional groups one with marked anti-lipoxidant activity, one with the function of modulating the skin's inflammatory response, which also improves the keratinisation process, and one with the function of activating microcirculation and lymphatic drainage.

Specifically, respectively:

Filagrinol®

Specific epidermal moisturiser obtained from active fractions of botanical unsaponifiables that modulate Filaggrin production.

It has a pronounced action against lipoperoxidation of the cell membranes induced by solar UV radiation (Malondialdehyde test) and more precisely by the formation of free radicals by the 'overheated' melanin of the contiguous melanocytes.

Filagrinol neutralises free radicals and oxidised substances thanks to plant-derived substances such as tyrosol (20-25 ppm) and hydroxytyrosol (4-5 ppm), metabolites of oleuropein, which boast strong antioxidant properties measured at around 40,000 µmolTE/g ORAC (Oxygen Radical Absorbance Capacity)(*).

Bearing in mind that the main electron and free radical acceptor in a biological system is water, the more water-rich a tissue is, the easier it is to neutralise the oxidising electrical charges generated in free radicals. Therefore, Filagrinol is essential for the integrity of the epidermal barrier and, due to its demonstrated ability to increase Filaggrin production, ensures adequate hydration of the epidermis and helps prevent skin ageing.

Since Filaggrin (as well as pro-Filaggrin, from which it is derived by ATPase dephosphorylation and other subsequent steps) is a protein rich in bound histidine, there is also the production of urocanic acid in its stable (trans) form, a catabolite obtained through the action of the cytosolic enzyme 'histidine ammonia-lyase' also known as histidase; it is an endogenous shielding agent protecting against UVB radiation but is not sufficient on its own and must also be 'protected' with specific shielding substances to prevent its transformation into its unstable and non-performing isomer (cis): all the more reason to implement adequate and effective protection. Finally, the increase in the Natural Moisturizing Factor (NMF) as a consequence of the final degradation of Filaggrin completes the activity of Filagrinol, keeping epidermal homeostasis adequate and constant.

Salycuminol®

Consisting of two long-chain esters of salicylic acid, it is capable of modulating the skin's inflammatory response (locally and not systemically), and thus the activity of Ornithine Decarboxylase (ODC) and Cyclooxygenase (COX). Also characterised by anti-lipoperoxidant, hence anti-ageing, and keratoplastic (non-keratolytic) activity, it optimises physiological epidermal turnover, improving the skin's appearance and reinforcing its protective (barrier) effect.

Skin reddening is evident in sun-exposed and post-exposed conditions, which is a consequence of the keratocyte release of chemokines and other pro-inflammatory factors that cascade into an intense inflammatory and vasodilatory reaction.

Salycuminol is suggested to modulate this process, and its immediate effect makes it possible to reduce the occurrence of any post-exposure manifestations, in addition to the aforementioned electronic excitation damage already triggered, and allows a physiological return to pre-existing conditions by regulating capillary dilatation to normal by decreasing the adhesion molecules that would slow down the circulation. Its keratoplastic properties, on the other hand, support epidermal physiological turnover in conditions of desquamation such as excessive dehydration and alteration of the keratinisation process (paracheratosis).

(*) ORAC: Unit of measurement of the antioxidant capacity of a product - Trolox equivalent micromoles per 100 g of product.

Zedomine®

Calibrated compound of essential oils, not hyperemizing and not rubefacient, which activates the microcirculation and lymphatic drainage by sympathetic activation of the contraction of capillaries and venules, and of the larger lymphatic vessels, resulting in an anti-oedemigenous and vasoconstrictor effect, with local thermoregulation of the tissue that would find relief from the stress caused by the irradiation and the electrochemical effect of the ($O_2^{\cdot-}$).

It also gives an initial cool sensation (about 15 minutes) followed by a rebound sensation of heat, in an alternation that can last from a few tens of minutes up to two hours.

Its mild peripheral sympathetic stimulation activity causes a slight contraction of the sweat glands which increase their aqueous eccrine secretion thus increasing the phenomenon of perspiratio insensibilis and consequently lowering the surface temperature of the epidermis.

There will also be a natural stimulation of the sebaceous glands, which will increase the cutaneous lipid film with the consequent known benefits.

Fitoestesina

Specialised compound of natural essential oils with strong antiseptic, reparative, soothing and antioxidant properties; the latter ability enables it under optimal conditions to bind the excited supernumerary electron and neutralise it. It offers the skin a pleasant sensation of relief.

Megasol® Sun Protection System: modulating

MEGASOL® M-MOD

Skin stress and pigmentation modulator.

It intervenes in tissue stressed by radiation and the electrochemical effect of ($O_2^{\cdot-}$) and is therefore intended for after-sun products at the end of the day (Graph 3).

Thanks to the activity of its constituents, it helps to reduce melanin accumulation by modulating its production, and to disperse the excess energy returned, even several hours after exposure.

ACS-AntiCytoStressor®

Fractionated organic phytoderivative from rathany root, a physiological bioregulator capable of modulating the release of stress-hormones by keratinocytes. Skin soothing agent against stress-inducing stimuli such as UV radiation, pollutants, environmental (cold, wind, air conditioning) or genetic factors.

Suitable for protective-preventive and restorative-reparative treatments.

Particularly indicated in this context for its properties as a modulator of the stress hormones produced autonomously by keratinocytes, with a clear reduction in the production of dopamine, adrenalin and noradrenalin.

The result is a soothing activity, useful in reducing the release of histamine, responsible for the unpleasant burning and itching sensation caused by sunburn, and the lipoperoxidation of cell membranes.

Thanks to rathany's intrinsic properties (astringent, anti-inflammatory, vasculotropic and bacteriostatic), Megasol M-Mod contributes to normalising the excited state of keratinocytes.

In addition, its natural bacteriostatic activity protects skin that is stressed by UV exposure and thus more sensitive to aggression by microorganisms.

Azamide®

A safe amide of azelaic acid, a modulator of pigmentation, thanks to its activity as an inhibitor of tyrosinase, a key enzyme in melanin synthesis, which acts selectively on melanin accumulation skin spots, i.e. those most dangerous for the formation of triplet oxygen superoxide, even in the hours following exposure.

This safe and soluble form of azelaic acid, in addition to modulating melanin synthesis, also possesses bacteriostatic properties that synergistically contribute to active skin protection.

CONCLUSION

Recent literature shows that solar radiation can also cause damage in a significant way in the hours following sun exposure.

In fact, the reaction products (CPD) in a melanocyte arise mainly after the end of UV exposure. It could therefore be assumed that this is the time when the keratinocytes, receiving melanosomes donated by the melanocytes, need to be protected the most. The slowness of the photochemical process, however, comes to the rescue and allows early intervention, reducing the possibility of damage.

The prospect is to be able to develop solar creams and after-sun creams to be applied in the evening or after sun exposure that could potentially prevent mutagenic processes from occurring in the skin hours after exposure to sunlight has ended.

Vevy Europe has therefore come up with the idea of synergistically using some of its already developed substances to dissipate this type of oxidising energy, and proposes several ingredients, to be administered in a suitable vehicle, before, during and after sun exposure, with the aim of selectively modulating the effects of solar radiation during sun exposure and in the hours following exposure.

The new approach to sun protection consists of three steps: filtering, dissipating, modulating.

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WHAT IS RELATA TECHNICA?

Starting from the beginning of the human story numberless substances have been applied on the skin to favour wound healing, for the management of skin diseases, or simply and perhaps more often for cosmetic aims. In sharp contrast, only in recent years, and with a great delay as compared with otherfields of pharmacology, the study of the effects of chemicals on the skin moved from art to science; now it is soundly based on a rational approach. Regulatory Authorities classify substances and formulations to be applied on the skin in two distinct categories: drugs and cosmetics. This in order to prevent that harmful or extremely active chemicals, contained in cosmetic preparations, are used without medical control.

Nevertheless, all pharmacologists know that in its widest meaning drug is every substance capable of modifying cell function, and it is difficult to admit that chemicals used in cosmetic preparations are devoid of any influence on biochemical mechanisms of epidermal cells, in particular in the case of long-term treatments. Thus dermopharmacology and cosmetology are at least overlapping disciplines, and there is no doubt that the same methodology should be employed in both fields.

Over the years Relata Technica has achieved a wide readership; at present its aim is to broaden the journal to make it a truly comprehensive dermopharmacology research journal in which articles in all of the most interesting and exciting areas of modern skin care have their forum. As a consequence, Relata Technica should attract manuscripts concerning the pharmacokinetic behaviour and the pharmacodynamic activity of old and new chemicals used to control skin diseases or to prevent skin aging, as well as studies providing insights on which to base rational development of new compounds for medicinal or cosmetic use.

Investigations on the various aspects of the interaction of chemicals with the skin can be analysed by the use of several experimental models: the intact animal, fragments of surviving skin, keratinocytes cultures or the more sophisticated in vitro reconstructed human skin, subcellular fractions and pure enzyme systems. The end point examined in the study may be the macroscopic appearance of the skin, its histological, histochemical or ultrastructural features, and a biochemical or molecular marker.

An important aspect of dermopharmacology, and even more of cosmetology, is safety assessment. Therefore the journal will be also very interested in publishing the results of research dealing with the local and systemic tolerability of new compounds. In this respect, one of the major goals of Relata Technica is to promote studies on the use and validation of the so called alternative assays which should have the final aim of substituting, at least for cosmetics, the use of laboratory animals in the assessment of systemic toxicity, local irritant activity and, in a broader sense, of any possible adverse effect.

Finally, Relata Tecnica should be the natural publication outlet for manuscripts concerning the formulation of dermopharmaceutical and cosmetic preparations, and in particular for those which analyse the influence of the vehicle and other ingredients on the efficacy and tolerability of the active substance.

It is essential that the quality of papers published in Relata Technica be good and, on the other hand, it is important for the journal to process and publish papers promptly. We will make every possible effort to improve and shorten the review process, and I believe that Relata Technica will become a preeminent journal in the field of dermopharmacology.

Stampato da Vevy Europe S.p.A.
Autorizzazione del Tribunale di Genova 31/84, del 18/6/84.
Iscrizione al Registro Operatori di Comunicazione (ROC) al n. 6411 del 10.12.01.