APIGENINA
Benefícios da flavona na pele
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APIGENININA

Benefícios da flavona na pele

DESCRÃO

A Apigenina (4',5,7-triidoxyflavona) é um flavonoide (aglicona), comumente encontrado em muitas frutas e vegetais como salsa e camomila.

MECANISMO DE AÇÃO

Na pele, a Apigenina regula a síntese de colágeno tipo I e tipo III nos fibroblastos aos níveis de mRNA e proteína, através da ativação da via smad2 / 3 e sem alterar a expressão da α-SMA, um marcador fibrótico em tecidos vivos. Além disso, a Apigenina restabelece a viabilidade de fibroblastos dérmicos e regula a expressão da colagenase, MMP-1, promovendo assim aumento da densidade dérmica, hidratação, uniformidade elasticidade, redução do comprimento das rugas e proteção contra a radiação. Apigenina também possui ações defensivas contra o stress oxidativos e a radiação UV/VIS, tais como a eliminação de radicais livres, aumento da estabilidade do DNA e proteção das células contra morte induzida pela fotosensibilização da melanina, contribuindo para a saúde cutânea.

INDICAÇÕES

✓ Ação antioxidante e anti-inflamatória;
✓ Proteção contra danos ao tecido cutâneo;
✓ Redução de rugas e síntese de colágeno.

DOSE USUAL

Recomendação oral de 50 a 100mg de Apigenina ao dia.

SUGESTÕES DE FÓRMULAS

<table>
<thead>
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<th>Fórmula</th>
<th>Doses</th>
<th>Indicação</th>
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<tbody>
<tr>
<td>Apigenina</td>
<td>50mg</td>
<td>Redensificador facial (IN)</td>
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<tr>
<td>Resveratrol</td>
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<td></td>
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<tr>
<td>Vitamina C revestida</td>
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</tr>
<tr>
<td>Manganês quelato</td>
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<tr>
<td>Cobre quelato</td>
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<tr>
<td>Colágeno hidrolisado</td>
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Modo de uso: 1 dose, 2 vezes ao dia.

<table>
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<th>Fórmula</th>
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<tr>
<td>Apigenina</td>
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<td>Hyanify™</td>
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<td>Ecoffea® (Coffea arabica)</td>
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<td>Base Dry touch</td>
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Modo de uso: aplicar 1 vez ao dia, antes de dormir.

PRINCIPAIS REFERÊNCIAS


Apigenin inhibits UVA-induced cytotoxicity in vitro and prevents signs of skin aging in vivo.

Apigenin (4',5,7-trihydroxyflavone) is a flavone that has been reported to have anti-inflammatory, antioxidant and anti-carcinogenic properties. In this study, we investigated the protective effects of apigenin on skin and found that, in experiments using cells, apigenin restored the viability of normal human dermal fibroblasts (nHDFs), which had been decreased by exposure to ultraviolet (UV) radiation in the UVA range. Using a senescence-associated (SA)-β-gal assay, we also demonstrate that apigenin protects against the UVA-induced senescence of nHDFs. Furthermore, we found that apigenin decreased the expression of the collagenase, matrix metalloproteinase (MMP)-1, in UVA-irradiated nHDFs. UVA, which has been previously identified as a photoaging-inducing factor, has been shown to induce MMP-1 expression. The elevated expression of MMP-1 impairs the collagen matrix, leading to the loss of elasticity and skin dryness. Therefore, we examined the clinical efficacy of apigenin on aged skin, using an apigenin-containing cream for clinical application. Specifically, we measured dermal density, skin elasticity and the length of fine wrinkles in subjects treated with apigenin cream or the control cream without apigenin. Additionally, we investigated the effects of the apigenin-containing cream on skin texture, moisture and transepidermal water loss (TEWL). From these experiments, we found that the apigenin-containing cream increased dermal density and elasticity, and reduced fine wrinkle length. It also improved skin evenness, moisture content and TEWL. These results clearly demonstrate the biological effects of apigenin, demonstrating both its cellular and clinical efficacy, and suggest that this compound holds promise as an anti-aging cosmetic ingredient.

Apigenin induces dermal collagen synthesis via smad2/3 signaling pathway.

Decrease in fibroblast-produced collagen has been proven to be the pivotal cause of skin aging, but there is no satisfactory drug which directly increases dermal thickness and collage density. Here we found that a flavonoid natural product, apigenin, could significantly increase collagen synthesis. NIH/3T3 and primary human dermal fibroblasts (HDFs) were incubated with various concentrations of apigenin, with dimethyl sulfoxide (DMSO) serving as the negative control. Real-time reverse-transcription polymerase chain reaction (PCR), Western Blot, and Toluidine blue staining demonstrated that apigenin stimulated type-I and type-III collagen synthesis of fibroblasts on the mRNA and protein levels. Meanwhile, apigenin did not induce expression of alpha smooth muscle actin (α-SMA) in vitro and in vivo, a fibrotic marker in living tissues. Then the production of collagen was confirmed by Masson's trichrome stain, Picrosirius red stain and immunohistochemistry in mouse models. We also clarified that this compound induced collagen synthesis by activating smad2/3 signaling pathway. Taken together, without obvious influence on fibroblasts' apoptosis and viability, apigenin could promote the type-I and type-III collagen synthesis of dermal fibroblasts in vitro and in vivo, thus suggesting that apigenin may serve as a potential agent for esthetic and reconstructive skin rejuvenation.

Apigenin, a bioactive flavonoid from Lycopodium clavatum, stimulates nucleotide excision repair genes to protect skin keratinocytes from ultraviolet B-induced reactive oxygen species and DNA damage.

In this study, we examined the antioxidative and the DNA protective potentials of apigenin, a flavonoid polyphenol isolated from Lycopodium clavatum, in both in-vitro (HaCaT skin keratinocytes) and in-vivo (mice) models against UV-B radiation. We used DAPI staining in UV-B-irradiated HaCaT skin keratinocytes pre-treated with and without apigenin to assess DNA damage. We also used a flow-cytometric analysis in mice exposed to UV-B radiation with or without topical application of apigenin to assess, through a comet assay, chromosomal aberrations and quanta from reactive oxygen species (ROS) generation. Data from the stability curves for the Gibb's free energy determined from a melting-temperature profile study indicated that apigenin increased the stability of calf thymus DNA. Immunofluorescence studies revealed that apigenin caused a reduction in the number of cyclobutane pyrimidine dimers (CPDs) after 24 h, the time at which the nucleotide excision repair (NER) genes were activated. Thus, apigenin accelerated reversal of UV-B-induced CPDs through up-regulation of NER genes, removal of cyclobutane rings, inhibition of ROS generation, and down-regulation of NF-κB and MAPK, thereby revealing the precise mechanism of DNA repair.
Topical apigenin improves epidermal permeability barrier homoeostasis in normal murine skin by divergent mechanisms.

The beneficial effects of certain herbal medicines on cutaneous function have been appreciated for centuries. Among these agents, chrysanthemum extract, apigenin, has been used for skin care, particularly in China, for millennia. However, the underlying mechanisms by which apigenin benefits the skin are not known. In this study, we first determined whether topical apigenin positively influences permeability barrier homoeostasis, and then the basis thereof. Hairless mice were treated topically with either 0.1% apigenin or vehicle alone twice daily for 9 days. At the end of the treatments, permeability barrier function was assessed with either an electrolytic water analyzer or a Tewameter. Our results show that topical apigenin significantly enhanced permeability barrier homoeostasis after tape stripping, although basal permeability barrier function remained unchanged. Improved barrier function correlated with enhanced filaggrin expression and lamellar body production, which was paralleled by elevated mRNA levels for the epidermal ABCA12. The mRNA levels for key lipid synthetic enzymes also were upregulated by apigenin. Finally, both cathelicidin-related peptide and mouse beta-defensin 3 immunostaining were increased by apigenin. We conclude that topical apigenin improves epidermal permeability barrier function by stimulating epidermal differentiation, lipid synthesis and secretion, as well as cutaneous antimicrobial peptide production. Apigenin could be useful for the prevention and treatment of skin disorders characterized by permeability barrier dysfunction, associated with reduced filaggrin levels and impaired antimicrobial defenses, such as atopic dermatitis.

Preparation of novel apigenin-enriched, liposomal and non-liposomal, antiinflammatory topical formulations as substitutes for corticosteroid therapy.

Two oil-in-water formulations, containing equal amounts of apigenin-enriched chamomile flower extracts, for potential use as topical antiinflammatory agents, were prepared and their physicochemical properties evaluated. A pilot clinical study was then carried out to assess patient acceptability and efficacy. The creams were either non-liposomal or liposomal. The liposomal formulations were more viscous, thus producing superior release characteristics in vitro. The clinical study also showed that the liposomal creams were, as antiinflammatory agents, slightly more effective in vivo than the non-liposomal formulations. These results suggest that there is scope for the further development of even more effective and safer alternatives to corticosteroids.

Enhancement of UVB-induced apoptosis by apigenin in human keratinocytes and organotypic keratinocyte cultures.

Topical application of the bioflavonoid 4',5,7-trihydroxyflavone (apigenin) to mouse skin effectively reduces the incidence and size of skin tumors caused by UVB exposure. The ability to act as a chemopreventive compound indicates that apigenin treatment alters the molecular events initiated by UVB exposure; however, the effects of apigenin treatment on UVB-irradiated keratinocytes are not fully understood. In the present study, we have used three models of human keratinocytes to study the effect of apigenin treatment on UVB-induced apoptosis: HaCaT human keratinocyte cells, primary keratinocyte cultures isolated from human neonatal foreskin, and human organotypic keratinocyte cultures. Each keratinocyte model was exposed to a moderate dose of UVB (300-1,000 J/m(2)), then treated with apigenin (0-50 micromol/L), and harvested to assess apoptosis by Western blot analysis for poly(ADP)ribose polymerase cleavage, annexin-V staining by flow cytometry, and/or the presence of sunburn cells. Apigenin treatment enhanced UVB-induced apoptosis >2-fold in each of the models tested. When keratinocytes were exposed to UVB, apigenin treatment stimulated changes in Bax localization and increased the release of cytochrome c from the mitochondria compared with UVB exposure alone. Overexpression of the antiapoptotic protein Bcl-2 and expression of a dominant-negative form of Fas-associated death domain led to a reduction in the ability of apigenin to enhance UVB-induced apoptosis. These results suggest that enhancement of UVB-induced apoptosis by apigenin treatment involves both the intrinsic and extrinsic apoptotic pathways. The ability of apigenin to enhance UVB-induced apoptosis may explain, in part, the photochemopreventive effects of apigenin.
REFERÊNCIAS


