REVIEW
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Combination of apigenin extract in dried parsley and quercetin in onions as a therapeutic modality for atopic dermatitis

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ABSTRACT

Atopic dermatitis is a highly prevalent chronic inflammatory skin condition that accounts for over one fifth of global dermatological cases, affecting children significantly more than adults. The disease is fundamentally driven by genetic mutations in the filaggrin protein and immune system disruptions that weaken the skin barrier, ultimately manifesting as severe, continuous pruritus. While standard medical management relies on topical corticosteroids to rapidly reduce this inflammation, these anti-inflammatory drugs frequently induce adverse effects ranging from localized skin atrophy to systemic suppression of the hypothalamic-pituitary-adrenal axis. Consequently, researchers are investigating botanical flavonoids as safer therapeutic alternatives; for example, apigenin found abundantly in dried parsley increases protective filaggrin expression to 204% ± 7.52% (compared to 100% ± 9.22% in baseline controls), and quercetin derived from onions significantly suppresses allergic markers in murine models by reducing Immunoglobulin E by 50.6% and decreasing Interleukins 4, 5, and 13 by 72.9%, 67.5%, and 34.8%, respectively. Understanding these dosages and effects helps translate preclinical findings into potential clinical applications, which is essential for evaluating their therapeutic relevance.

Keywords: anti-inflammatory, apigenin, atopic dermatitis, dermatology, quercetin.
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INTRODUCTION

Atopic dermatitis represents a growing global health burden, standing as one of the most prevalent chronic skin conditions worldwide. Epidemiological data from the Global Burden of Disease Study indicate that this condition accounts for 21.23% of all dermatological cases globally, with pediatric populations experiencing significantly higher incidence rates (15–20%) than adults (1–3%).[1,2] Furthermore, the International Study of Asthma and Allergies in Childhood (ISAAC) notes a two- to three-fold increase in developing nations, a trend mirrored in Indonesia, where it ranks as the top skin disease with a 23.67% prevalence rate, disproportionately affecting females at a 1.3:1 ratio.[3-5] This widespread and escalating epidemiological footprint highlights a pressing vulnerability in population health, particularly among children in rapidly developing regions. Consequently, addressing the rising incidence of atopic dermatitis requires urgent advancements in scalable, long-term dermatological management strategies.

The clinical complexity of atopic dermatitis arises from a multifactorial etiology driven by both intrinsic genetic predispositions and extrinsic environmental triggers. Central to this pathogenesis is the mutation of the filaggrin (FLG) gene, which depletes essential epidermal proteins and fundamentally compromises the skin barrier.[2,5,6] Concurrently, immunological dysregulation exacerbates the condition, primarily through Th2-mediated hypersensitivity reactions where the excessive release of cytokines (IL-4 and IL-13) triggers immunoglobulin E (IgE) production.[5] The synergistic failure of structural barrier integrity and immunological homeostasis culminates in the classic clinical manifestations of severe nocturnal pruritus, erythema, and desquamation.[2,7] Understanding these interconnected structural and inflammatory pathways is essential for developing targeted interventions that move beyond mere symptom suppression toward true physiological restoration.

While standard medical protocols offer established algorithms for managing atopic dermatitis, long-term adherence is frequently hindered by treatment-associated toxicities. According to the International Consensus Conference on Atopic Dermatitis II, topical corticosteroids remain the definitive first-line therapy, functioning as potent anti-inflammatory agents that inhibit phospholipase A2, induce vasoconstriction, and suppress inflammatory transcription factors.[5,8] Although these pharmacological mechanisms efficiently mitigate acute pruritus and inflammation, their prolonged application is invariably linked to deleterious adverse effects,

ranging from localized skin atrophy to profound systemic complications such as hypothalamic-pituitary-adrenal (HPA) axis suppression and pediatric growth retardation.[5,7] Therefore, the clinical reliance on corticosteroids necessitates the exploration of safer, sustainable therapeutic alternatives that can effectively manage chronic symptoms without compromising patient safety.

Emerging research indicates that specific botanical flavonoids offer promising therapeutic potential by directly targeting the dual pathogenesis of atopic dermatitis. Preclinical investigations highlight two notable compounds: apigenin, abundantly found in dried parsley (45 μg/g), which significantly upregulates protective filaggrin expression by 204% compared to baseline vehicle controls (100%); and quercetin, concentrated in onions (28.4–48.6 mg/100 g), which robustly suppresses allergic inflammatory cascades.[9–12] Specifically, quercetin has been shown to reduce systemic IgE levels by 50.6%, alongside substantial decreases in critical cytokines including IL-4 (72.9%), IL-5 (67.5%), and IL-13 (34.8%).[12] By actively synthesizing epidermal structural repair through filaggrin enhancement while simultaneously dampening hypersensitive immune responses, these phytochemicals comprehensively address the foundational mechanisms of the disease. Harnessing these botanical properties provides a logical pathway toward developing integrated treatments that facilitate complete physiological recovery in dermatological patients.

Given the limitations of current corticosteroid therapies and the complementary mechanisms of specific flavonoids, establishing a multi-targeted approach is critical for advancing atopic dermatitis care. Because the pathogenesis of this condition inherently depends on both structural degradation and inflammatory hyperreactivity, relying on a single therapeutic agent is often insufficient to achieve sustained remission. A synergistic formulation that leverages apigenin's filaggrin-boosting properties alongside quercetin's immunosuppressive effects theoretically provides a comprehensive intervention capable of targeting all major disease pathways. Therefore, the primary objective of this study is to investigate and evaluate the therapeutic efficacy of combining apigenin extract from dried parsley with quercetin from onions as a novel, dual-action modality for the treatment of atopic dermatitis.

RESULTS

Atopic Dermatitis Pathogenesis

The pathogenesis of atopic dermatitis is characterized by skin barrier dysfunction and an immune response that deviates from a Th2/Th22 profile. Genetic mutations in filaggrin (FLG) cause dysfunction of the skin barrier and dehydration, leading to increased permeability to external allergens. The impaired skin epidermal barrier releases substantial amounts of Thymic Stromal Lymphopoietin (TSLP), which triggers a Th2/Th22 immune response. Th2/Th22 deviations are further accelerated during the development of the disease, for example, from acute to chronic atopic dermatitis or childhood to adulthood. Meanwhile, Th1 cells tend to participate in the chronic phase of atopic dermatitis.[13]

Th2 cytokines (IL-4 and IL-13) stimulate B cells to produce IgE antibodies against allergens. Some IgE reacts to self-antigens. IgE autoreactivity also contributes to disease activity. In addition, IL-4, IL-13, and IL-22 suppress FLG expression. The pruritus experienced by the sufferer is caused by TSLP and IL-31 derived from Th2 cells, and subsequent scratching further aggravates dysfunction of the skin barrier. The release of TSLP from keratinocytes depends on the entry of calcium regulated by the ORAI1 channel.[13] The pathogenesis of atopic dermatitis is illustrated in **Figure 1**.

Quercetin Potency in Onion Extract

Onions have been shown to contain many active compounds, including anthocyanins, kaempferol, quercetin, and isorhamnetin.[14] Quercetin is the most abundant in onions when compared to other herbal plants, at 28.4–48.6 mg per 100 g.[15] Quercetin is known to decrease eosinophil count, plasma IgE levels, and the expression of pro-inflammatory cytokines, namely IL-4, IL-5, IL-6, IL-13, IFN-γ, and TNF-α.[12] Based on the research of Hou DD et al., the administration of quercetin for eight days in mice can alleviate symptoms of erythema, edema, dryness, and scaly skin.[16] In addition, quercetin may also increase the expression of antioxidant enzymes, such as SOD1, SOD2, and catalase.[17] Quercetin administration can restore skin tissue damaged by scratching in patients with atopic dermatitis by inducing epithelial-mesenchymal transition.[17] Quercetin has been widely proven to have its potential as an anti-inflammatory, antioxidant-inducing, and wound healing agent,

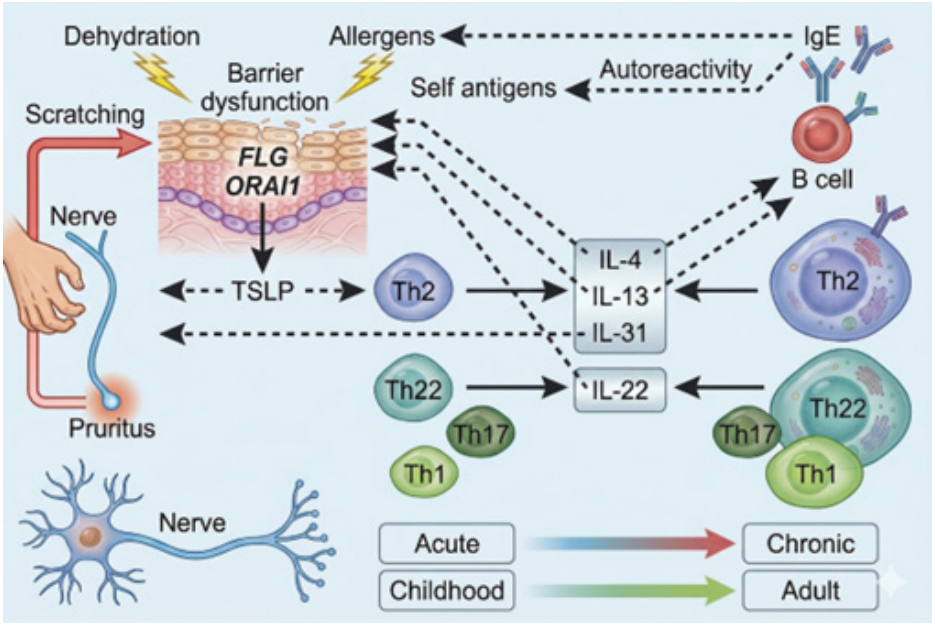

Figure 1. Pathogenesis of Atopic Dermatitis

making it a promising modality in the treatment of atopic dermatitis.

Apigenin Potential in Dried *Parsley Extract*

Dried parsley has been shown to contain several active compounds, including isorhamnetin, apigenin, and luteolin.[18] Apigenin is the most abundant in dried parsley than in other herbal plants, at 45.035 µg per 1 gram of dried parsley.[19] Apigenin is known to increase filaggrin protein expression, which is decreased by FLG gene mutations in people with atopic dermatitis.[9] Based on the research of Hou et al., topical apigenin administered to mice may increase the expression of epidermal filaggrin characterized by the presence of a thickening of the filaggrin epidermis upon immunostaining.[9] With increased filaggrin production, the skin's barrier function returns to normal. In addition, apigenin can act as an anti-inflammatory, as shown in a study by Yano et al., which reported significant decreases in IgE, IL-4, and IFN-γ.[20,21] Apigenin has been widely proven to have potential as an anti-inflammatory and filaggrin-inducing agent, making it a promising modality in the treatment of atopic dermatitis.

Working Mechanism

Mechanism of Action of Quercetin

The mechanism of action of quercetin is to inhibit the production of lipopolysaccharide-induced tumor necrosis factor (TNF-α) in macrophages and LPS-induced IL-8 production in lung A549 cells. In addition, within glial cells, quercetin has been shown to inhibit LPS-induced TNF-α and interleukin (IL)-1α mRNA expression. Quercetin significantly lowers the regulation of Th-2-derived interleukin 4 (IL-4) by normal peripheral blood mononuclear cells (PBMCs). Furthermore, the administration of quercetin may increase the phenotypic expression of IFN-γ cells and decrease IL-4 cells. These results suggest that the beneficial immunostimulatory effects of quercetin can be mediated through the induction of Th-1, IFN-γ-derived cytokines, and inhibition of Th-2, IL-4-derived cytokines.[22]

Quercetin can inhibit the metalloproteinase matrix, which is typically inhibited by plasminogen activator inhibitor 1 (PAI-1) in human dermal fibroblasts. Different biochemical pathways of IgE-induced degranulation regulate IL-6 production from human mast cells in response to IL-1 stimulation, and quercetin can block IL-6 secretion and the two key signal transduction steps involved.[22] Quercetin directly regulates the basic functional properties of immune cells, an effect mediated by extracellularly regulated mitogen-activated protein kinase 2 (Erk2) signaling pathways in human mitogen-activated PBMCs and purified T lymphocytes.[22] In addition, quercetin is able to block IL-12-induced tyrosine phosphorylation of JAK2, TYK2, STAT3, and STAT4, resulting in decreased IL-12-induced T cell proliferation and Th1 differentiation.[22]

Mechanism of Action of Apigenin

The mechanism by which apigenin acts in atopic dermatitis is its potential to induce filaggrin protein expression. According to Hou et al., the increased expression of the filaggrin protein is due to apigenin-induced upregulation of filaggrin mRNA. This was demonstrated by mRNA expression analysis in human keratinocyte cultures, which showed increased filaggrin mRNA at 24 and 48 hours.[9] In the first 24 hours, the expression of filaggrin mRNA increased by 204 + 7.52 compared to the control vehicle of 100 + 9.22, while after 48 hours, there was an increase in mRNA expression by 160 + 16 compared to the control of 100 + 10.20.[9]

The mechanism of upregulation of the filaggrin gene begins with the antioxidant ligand apigenin, which activates nuclear factor-erythroid 2-related factor-2 (NRF2) through the aryl hydrocarbon receptor (AHR) and inhibits ROS (reactive oxygen species) production.[23] Inhibition of ROS production increases the expression of filaggrin genes along with other skin barrier proteins. The mechanism of filaggrin upregulation is illustrated in Figure 2.

In addition, apigenin, an anti-inflammatory, can inhibit the inflammatory response by lowering IgE levels and reducing inflammatory mediators that contribute to the pathogenesis of atopic dermatitis. The anti-inflammatory

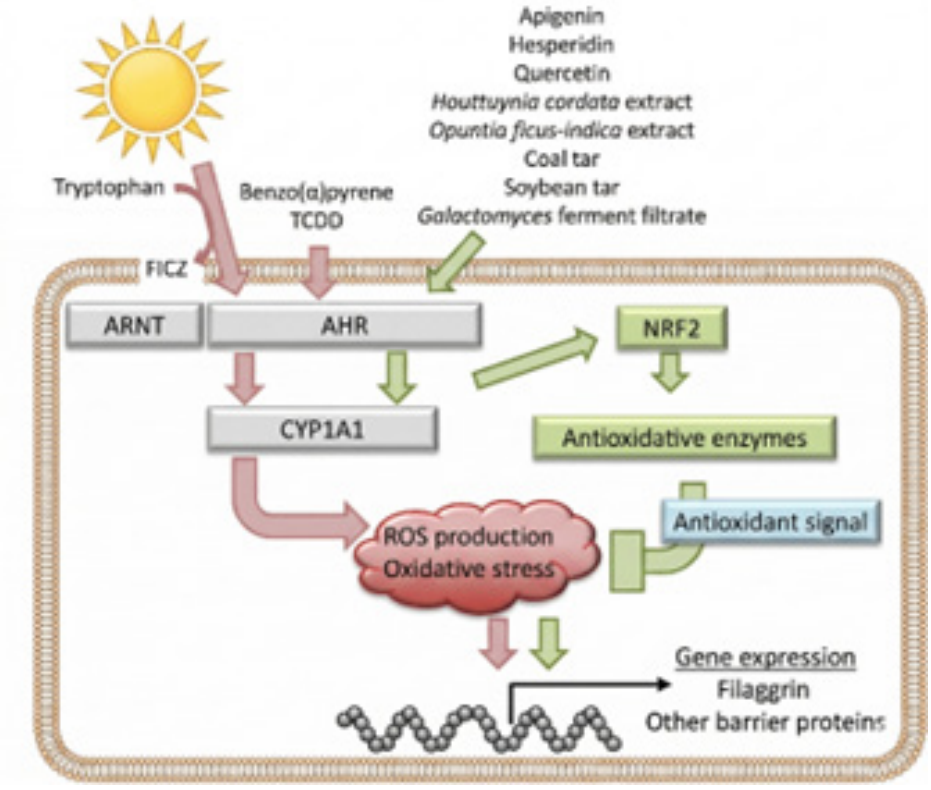

Figure 2. Mechanism of Enhancing Filaggrin Gene Expression

mechanisms of apigenin include (1) apigenin regulates Th cell differentiation, by decreasing the increase in Th2-induced cytokines to restore the balance between Th1 and Th2; (2) affect OVA-specific antibodies produced by B cells; (3) inhibits mast cell degranulation and release of inflammatory cytokines through inhibition of MAPK signaling pathways by competitively binding ER with E2.[24] The mechanism of action of apigenin (API) as an anti-inflammatory can be seen in **Figure 3**.

In Vivo and In Vitro Effects

Clinical Effects of Quercetin

Based on in vivo experiments in mice from the MC903 group, the ears of mice that were initially erythematous, edematous, and dry showed symptom improvement after 8 days of quercetin administration.[16] In addition, administration of quercetin in experimental mice also decreased the number of eosinophils (79.48%), serum IgE (50.6%), IL-4 (72.9%), IL-5 (67.5%), and IL-13 (34.8%). Based on research by Beken et al., administration of 1.5 µM quercetin can increase the expression of antioxidant enzymes, such as superoxide dismutase-1 (SOD1), SOD2, catalase (CAT), and glutathione peroxidase (GPx).[17] Quercetin can also heal wounds by inducing an epithelial-mesenchymal transition characterized by a 50% upregulation of mRNA expression of twist and a 70% decrease in snail regulation.[17] More detailed clinical effects of topical quercetin can be seen in **Table 1**.

Clinical Effects of Apigenin

In vitro, apigenin treatment of HaCaT cells at 20 µM resulted in a significant increase in filaggrin protein levels (>100 pg/ml) compared to the control (100 pg/ml).[10] Based on the research of Hou et al., in mice administered topical apigenin, filaggrin thickened after immunohistochemical staining compared to the control vehicle.[9] In addition, apigenin may also act as an anti-inflammatory by significantly lowering serum IgE levels (<2000 ng/mL) when compared to controls. IL-4 and IFN-γ were also reported to decrease after apigenin administration to mouse spleen cells, to ±0.8 and <0.8, respectively.[20]

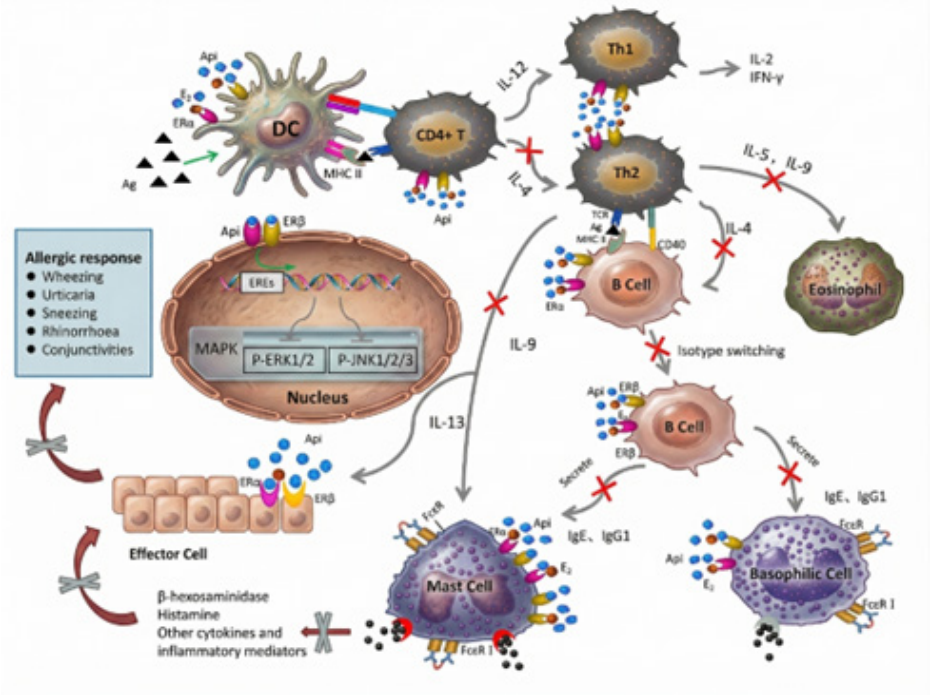

Figure 3. Mechanism of Action of Apigenin as an Anti-Inflammatory

Table 1. Clinical Effects of Topical Quercetin

No.	Author (Year)	Method	Results
1.	Karuppagounder et al. (2015).[11]	Administration of quercetin in NC/Nga rats.	There was a significant decrease in the score of atopic dermatitis at the fourth week, a decrease in the number of mast cells per field of view, as well as a decrease in TNF-α, IL-1β, IFN-γ, IL-4, and other cytokines.
2.	Beken et al. (2019).[17]	Administration of quercetin on HaCaT cells in vitro.	There was a significant decrease in the expression of mRNA from IL-1β, IL-6, and IL-8 as well as a decrease in TSLP concentration.
3.	Park et al. (2015).[12]	Administration of topical quercetin in NC/Nga rats.	There was a significant decrease in eosinophil type count and IgE concentration as well as a significant decrease in IL-4, IL-5, IL-13.
4.	Hou et al. (2019).[16]	Topical administration of quercetin in MC903 induced atopic dermatitis.	There was a significant improvement in the symptoms of atopic dermatitis in the skin of the rats' ears as well as a significant decrease in the expression of IFN-γ, TNF-α, IL-4, and IL-6.

Construction Mechanism

Mechanism of Construction of Apigenin

Parsley extract is obtained using three extraction techniques: macerated solvent extraction, ultrasonic extraction, and microwave-assisted extraction. 0.5 g of dried parsley leaves and 20 mL of solvent mixture are used for the extraction. After filtration and washing, the extract is diluted to 25 mL with a suitable solvent mixture.[25]

The maceration time is 14 days at 35°C. Ultrasonic-assisted solvent extraction was performed by immersing

the T310 Transsonic at 35 kHz and an installed power of 95 W. Samples were first immersed in the extraction solvent mixture for 10 minutes and then sonicated for 30 minutes at 35 °C. Microwave-assisted solvent extraction is performed using homemade equipment. Considering the specificity of the plant material, the following parameters are selected: a maximum temperature of 35°C, a working time of 1 minute and a coefficient of duty of 40% at an installed power of 900 W.[25]

Mechanism of *Construction of Quercetin*

3 grams of onion are thinly sliced to a uniform size. Then, these onion slices are dried in the oven at 40 °C for 5 days, yielding a dry material weighing 580 g. The next stage is to determine the moisture content and perform the extraction using the maceration method. The previously obtained dry material is then crushed to reduce the sample size. Furthermore, 200 g of the sample was macerated with 500 mL of ethyl acetate in three repeats to obtain the maceration results. The macerated material is then concentrated using a rotary evaporator, yielding a crude extract of 4.2519 g. To ensure the extract contains flavonoids, a phytochemical test is performed using the Wilstatter test. This test is carried out by dissolving a coarse extract into ethyl acetate, which is then added to magnesium powder granules, concentrated hydrochloric acid, and amyl alcohol. If the reaction is reddish, the extract contains flavonols (quercetin), whereas a yellowish reaction indicates flavones.[26]

The next stage is the separation of flavonoid fractions using a flash chromatography tool with an ethyl acetate:petroleum benzene eluent at a 1:1 ratio. Before the sample is inserted, the tool is run at a flow rate of 25 ml/min for 4 minutes without a sample. This action is performed to wash and saturate the silica column in the tool and to prevent any remaining compounds. Next, a sample of crude extract is injected into the injector valve at a flow rate of 10 ml/min to obtain the separated fractions. The fractions will then be placed in a test tube containing 3 mL of each. To determine which test tubes contain a fraction of quercetin, each tube is tested using a Thin Layer Chromatography (TLC) plate to assess its separation pattern. If the separation pattern or number of spots in a fraction is the same as the standard quercetin, then it can be ascertained that the fraction contains quercetin.[26]

To obtain pure quercetin, the fraction will be qualitatively analyzed using flash chromatography. Furthermore, the fraction will be analyzed using UV-VIS (Ultraviolet–Visible) and FTIR (Fourier-Transform Infrared) spectrophotometers.[26]

Administration of Apigenin and Quercetin Extracts

The administration of apigenin and quercetin extracts will be topical as a cream, as several studies have, on average, administered these two flavonoids topically and found that topical apigenin and quercetin in experimental mice can provide significant clinical effects, such as increased filaggrin expression and anti-inflammatory effects. In addition, cream preparations can last longer on the surface of the skin than gel preparations, providing a protective layer for a longer period.[9,16]

Pharmacokinetics

Pharmacokinetics Apigenin topical

Apigenin and quercetin belong to the class II class of drugs with poor solubility and high permeability properties of the membranes of the digestive tract.[27] These chemicals are highly hydrophilic or have very low solubility rates in nonpolar solvents. In general, apigenin is absorbed throughout the intestine. These chemicals are transported through both active and passive mechanisms in the duodenum and jejunum. However, the main role of passive transport occurs in the ileum and colon. The absorption of apigenin and quercetin is mostly conjugated and partially transported in its whole state.[28] Topical apigenin and quercetin systems will deliver apigenin and quercetin to the local parts of the skin membrane rather than to the bloodstream.[27] Apigenin and quercetin are then distributed into the liver for metabolism. The apigenin metabolic pathway involves conjugation and deglycosylation reactions. After undergoing the metabolic stage, apigenin and quercetin are excreted in urine.[27]

Topical Quercetin Pharmacokinetics

A topical administration mechanism is recommended because it can act directly on affected areas. In addition, topical drugs are minimally absorbed into the systemic circulation, so systemic side effects are smaller than with oral drugs or other routes of administration. According to the research by Saija et al., topical quercetin showed minimal skin absorption.[29] However, the distribution of topical quercetin is quite high. This is proven based on research from Belo et. al. which found that after 24 hours of administration of the extract, quercetin accumulated in the stratum corneum layer (0.17 ± 0.02 μg/cm^2 or 23.6% of the administered dose) and epidermis (0.23 ± 0.04 μg/cm^2 or 33.0% of the overall dose administered), but was not detected in the dermal layer.[29]

CONCLUSION

The combination of apigenin in dried parsley extract and quercetin in onion extract has therapeutic potential for atopic dermatitis. Topical administration results in minimal side effects, works directly on the inflamed areas of the skin, and can remain active in the skin layer longer. Regarding clinical effects, quercetin acts as an anti-inflammatory by degrading inflammatory cells and cytokines, while apigenin can significantly increase filaggrin expression by upregulating filaggrin mRNA. The selection of the two therapeutic targets cannot be separated from the dominant influence of the two factors on the occurrence of atopic dermatitis. Thus, physiological changes, including skin barrier damage, can be recovered following the administration of apigenin and quercetin extracts. Further translational research on effective dosing and safety is needed to confirm this study's findings.

CONFLICT OF INTEREST

All authors declared that there is no conflict of interest regarding this article.

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ETHICS APPROVAL

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AUTHOR'S CONTRIBUTION

All authors contributed equally in the writing process of this article.

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