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REVIEW ARTICLE

The Beneficial Properties of Virgin Coconut Oil in Management of Atopic Dermatitis

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ABSTRACT

Atopic dermatitis (AD) is a chronic, inflammatory skin disease that is characterized by intense pruritus and eczematous lesions. It is an increasingly pruritic inflammatory skin disorder which can affect both children and adults. Natural products offer great hope in the identification of bioactive lead compounds and their development into topical cream or ointment in managing skin diseases which are associated with inflammatory response. One of the most popular natural products which have been vastly used in managing AD is virgin coconut oil (VCO). VCO is extracted from the fresh and mature kernel of the coconut (*Cocos nucifera* L.) through wet and dry methods, without altering the valuable phytochemicals and physiochemical properties of the oil. It possesses numerous health benefits from the retained physiochemical properties from its triglycerides and medium chain fatty acids. The use of VCO in the management of AD is one of the topical therapies which have been proven to have good therapeutic effects and it is safe for topical applications. Studies have been proven that VCO exhibits antioxidant, anti-inflammatory, antibacterial, wound healing, and moisturizing properties which were extremely important in the management of AD.

Keywords: Anti-inflammatory, antioxidant, lauric acid, moisturizer, virgin coconut oil, wound healing

INTRODUCTION

Atopic dermatitis (AD) is a chronic, inflammatory skin disease that is characterized by intense pruritus and eczematous lesions. It is an increasingly pruritic inflammatory skin disorder which can affect both children and adults.[1] The prevalence of AD has been detected in 15%–30% of children and 2%–10% of adults.[2] The diagnosis of AD is based on the constellation of clinical findings, such as itchiness, facial and extensor eczema in infant and children, flexural eczema in adults and chronicity of dermatitis.[3]

The Type 2 T-helper (Th2) cytokines interleukin-4 (IL-4) and IL-13 are key drivers involved with the underlying inflammatory process.[4] It is characterized by intensely and incessantly itchy, dry, inflamed skin that begins in infancy, and often persists throughout adulthood. AD often heralds the onset of other allergic immunoglobin E (IgE)-mediated diseases that may affect skin or different epithelial surfaces. Pathogenesis of AD is still unclear. It is believed that epidermal barrier disturbance and immune dysfunction resulting in IgE sensitization are critical factors in the development of cutaneous inflammation.[5] Thymic stromal lymphopoietin (TSLP), an epithelial-derived cytokine that is upregulated in the setting of barrier disruption, has been implicated in the pathogenesis of AD. Levels of TSLP expression in skin correlate with symptoms and severity in AD patients and in animal models. It acts as a potent stimulator of Th2 cytokines, including ILs 4, 5, and 13, that, in turn, trigger IgE production and release from plasma cells, stimulate both the itch and inflammation associated with AD.[5,6]

The flares of AD can be trigger by several factors. The presence of irritants and allergens could trigger the itchiness and scratching which could induce and sustain the inflammatory cascade initiated by the release of proinflammatory cytokines from atopic keratinocytes. AD patients can be triggered by food and environmental aeroallergens.[7] Topical corticosteroids are the first-line treatment when AD flares-up. However, repeated or prolonged use of topical corticosteroids can cause many side effects, include thinning of the skin (atrophy), easy bruising and tearing of the skin, increased in susceptibility to skin infections (i.e., impetigo, Malassezia folliculitis), enlarged blood vessels (telangiectasia), stretch marks in the skin (striae),[3,8,9] hypopigmentation, and corticosteroid acne. Adrenal suppression and growth retardation may happen in children if high-potency agents are used.[9] Furthermore, prolonged use of steroid may result in many AD patients suffer from topical steroid addiction.

Patients who are prescribed with clinically used anti-inflammatory drugs suffer from the disadvantage of side effects and the high cost of treatment. An alternative to these drugs are traditional medicines and natural products, which offer great hope in the identification of bioactive lead compounds and their development into topical cream or ointment in managing skin diseases which are associated with inflammatory response. To lessen the use of topical corticosteroids, approaches using natural-based product can be a better alternative option for AD patients.

Virgin coconut oil (VCO), a relative newcomer in the fats and oil market, is fast becoming valuable oil. VCO is extracted from the fresh and mature

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kernel of the coconut (*Cocos nucifera* L.) through wet or dry methods, without altering the valuable phytochemicals and physiochemical properties of the oil. It possesses numerous health benefits from the retained physiochemical properties from its medium chain fatty acids. The use of VCO in the management of AD is one of the topical therapies which have been proven to have good therapeutic effects, and it is safe for topical applications.

EXTRACTION OF VIRGIN COCONUT OIL

VCO can be extracted from the coconut meat of a fresh and mature kernel. In general, VCO can be extracted using either dry or wet methods.

In the dry method, the kernel is heated under-specific conditions to remove the moisture while preventing scorching and microbial invasion.[10] Fully-dried coconut copra is separated, and the coconut meat is grated, followed by hydraulic extraction at low temperature (<50°C). On the other hand, the coconut meat/kernel does not undergo the drying process in the wet method.[10] The wet method can be further divided into three alternatives to destabilize the coconut milk emulsion: (1) chilling, freezing, and thawing, (2) fermentation, and (3) enzymatic methods, or any of these in combination.

Chilling, freezing and thawing technique

In chilling, freezing and thawing technique, the emulsion of the coconut milk is centrifuged followed by chilling and freezing at 10°C and −4°C, followed by thawing at 40°C, until the coconut cream reached room temperature. The nonsoluble solids would be eliminated in these steps to yield high-quality VCO.[11] Oil recovery can be achieved by centrifugation. Centrifugation would ensure the coconut oil is packed in globules to crystallize at a lower temperature.

Fermentation

Grated coconut meat is first mixed with water at 30°C, 50°C and 70°C in different ratios (1:1, 1:2 and 1:3), followed by inoculation with *Lactobacillus platarum* 1041 IAM and the mixture is allowed to settle for 2–6 h. *L. platarum* would assist in the rapid breakage of emulsion and the release of the oil, as high as 95%. pH of the extracted VCO is then adjusted to destabilize the coconut milk emulsion. Finally, fermented milk is centrifuged to harvest the VCO.[10,12]

Enzymatic extraction

VCO is extracted by the action of various enzymes, namely, cellules, endoamylase, viscozyme L, neutrase, and alcalase. These enzymes would react on the coconut milk at different concentrations, pH, and temperature parameters to enhance oil extraction. The cell-wall degrading enzymes solubilize the structural cell wall components of the oilseed,[10] and finally, the VCO is recovered by centrifugation.

Chemical composition in VCO

Chemical composition in VCO has been analyzed and studied.[13] Analysis showed that VCO is composed of >99% triglycerides, and it is rich in medium chain fatty acids. Lauric acid (*La*; C12:0) is the fatty acid that present most abundantly in VCO (average 46%–48%). Myristic acid (*M*; C14:0) is the second highest fatty acid present, approximately 17%. Other fatty acids which are present in smaller amount including palmitic acid (*P*; C16:0) (8%–10%), caprylic acid (C8:0) (7%–9%), capric acid (*C*; C10:0) (5%–7%), oleic acid (*O*; C18:1) (5%–7%), stearic acid (C18:0) (2%–4%), linoleic acid (C18:2) (1%–2%), and caproic (*Cp*; C6:0) (0.5%–0.7%). The major triglycerides in VCO samples consisted of 22%–25% of *LaLaLa*, 14%–16% of *CCLa*, 19%–21% of *CLaLa*, 13%–15% of *LaLaM*, and 7%–9% of *LaMM*. Studies have found that VCO from different countries may have slightly different content of medium chain fatty acids and triglycerides. Malaysia VCO has higher contents of *CpCpLa*, *CpCLa*, and *LaOO* while Indonesian VCO has more *LaMP*.[11,13]

Several phenolic acids have been identified, namely, protocatechuic acid, vanillic acid, syringic acid, p-coumaric acid, caffeic acid, and ferulic acid.[14] Ferulic acid is the major phenolic acids found in VCO (5 mg/kg VCO). Polyphenols present in VCO include gallic acid, quercetin, methyl catechin, dihydrokaempferol, and myricetin glycoside.[15] These phenolic acids and polyphenols could exhibit therapeutic effects, such as antioxidative, anti-inflammatory, antibacterial, and promote wound healing.[15-18]

Therapeutic effects of virgin coconut oil

VCO has been reported to possess many therapeutic effects. VCO exhibited several key properties which are extremely crucial in the management of AD, namely, antioxidant, anti-inflammatory, antibacterial, wound healing, and moisturizing.

Antioxidant

VCO is rich in medium chain fatty acids, phenolic, and polyphenols which made it beneficial as an antioxidant source. Nevin and Rajamohan[19] reported that polyphenols in VCO could inhibit the microsomal lipid peroxidation. They reported that VCO fed animals were found to have lower lipid peroxide levels in the heart, liver, and kidney, but higher total glutathione content in the blood. Glutathione could efficiently scavenge reactive species and toxic intermediates of incomplete oxidation. Thus, the ability to maintain total glutathione concentration and glutathione in the reduced state is crucial in antioxidant defense mechanism.

Nevin and Rajamohan[20] researched on the effect of lipid peroxidation inhibition activity of VCO and refined coconut oil. They discovered that VCO had the better capability in maintaining lipid metabolism. Their well-preserved fatty acids and polyphenols were capable of exhibiting antioxidant activities, which also contributed to the anti-inflammatory activity. VCO exhibited promising edema inhibition. It protected the adjuvant-induced arthritic rats from free radicals and joint inflammation by increasing the level of antioxidant enzymes such as glutathione peroxidase and catalase.

Anti-inflammatory

VCO has been proven to exhibit anti-inflammatory activity. It has been proposed that VCO could demonstrate anti-inflammatory activity in acute and chronic inflammation in AD. Evangelista *et al.*[21] proposed that the medium chain fatty acids in VCO could be broken down by the lipases of skin flora to free fatty acids, which could then aid in the reduction of cellular inflammation.

Intahphuak *et al.*[22] have reported that VCO could exhibit moderate anti-inflammatory effects on the acute inflammatory model, including ethyl phenylpropiolate-induced ear edema in rats, carrageenin- and arachidonic acid-induced paw edema are used. The reduction was observed at 15 and 30 min after the application of 1, 2, and 4 mg/20 µL VCO/ear, and at 60 and 120 min after the application of 2 and 4 mg/20 µL VCO/ear. The inhibitory effect displayed in a dose-dependent manner. It also exhibited an inhibitory effect on chronic inflammation by reducing the transudative weight, granuloma formation, and serum alkaline phosphatase activity.[22,23] Vysakh *et al.*[16] have reported that VCO is also effective against treating chronic inflammation in arthritis, which could be due to its anti-inflammatory properties and ability to overcome nitric oxide-induced oxidative damage.

Antibacterial

Staphylococcus aureus frequently colonize infected eczema skin. *S. aureus* colonization on atopic skin could lead to chronic inflammation, skin barrier dysfunction and result in dry and flaky skin. Antibiotic treatment and antiseptic lotion are commonly prescribed in managing the infection resulted from *S. aureus*. However, Verallo-Rowell *et al*.[18] discovered that lipases which were produced by *S. aureus* on the skin would hydrolyze triglycerides in VCO to monoglycerides. These monoglycerides and medium chain fatty acids present in VCO could exhibit antibacterial, antifungal, and antiviral activities.[18,24] This finding was firstly noticed in monolaurin, a monoglyceride produced from hydrolysis of lauric acid which is also present in VCO. Monolaurin was reported could significantly inhibit the *S. aureus*.[18,25] This finding is also supported by Loung *et al*.[26] where the authors also noticed better antibacterial activity exhibited by the enzymatic hydrolyzed VCO, compared to nonhydrolyzed VCO. It was explained that the small molecular size of monoglycerides could penetrate the membrane barrier more readily, disintegrate the bacteria cell membrane, inhibit the action core enzymes, and finally lead to bacteria cell death.[24] Shilling *et al*.[24] have reported that examination of VCO fatty acids and hydrolyzed VCO treated *Clostridium difficile* under transmission electron microscope showed the changes in the bacterial cell membrane and disruption of the cell cytoplasm. This showed that hydrolyzed VCO could potentially contribute to the antimicrobial activity on infected AD skin.

Wound healing

The wound healing process involves five important stages, namely inflammation, neovascularization, granulation tissue formation, reepithelization, and new extracellular matrix formation and tissue remodeling.[17] Plant products with antioxidant, anti-inflammatory and antimicrobial properties are believed to be effective in promoting wound healing. VCO is an effective adjuvant for a faster healing wound. Srivastava and Durgaprasad[27] have reported that VCO could promote the wound healing effect 2 times faster than the control group. It was noticed that the wound contraction of VCO treated animal group was 84.7%, whereas untreated group could only manage to achieve 34.9% on day 16 of the treatment. The mean period of epithelialization was also shorter in VCO treated group.[27] Besides, the wound healing properties of VCO had also been supported by Nevin and Rajamohan,[17] which their study also reported that VCO-treated wounds healed faster and the time for complete epithelization was shorter. The levels of various skin components observed in the wound was higher, as indicated by the higher amount of pepsin soluble collagen, collagen cross-linking and total collagen content.

Moisturizing

AD is characterized by dry skin and defects in the epidermal barrier and cutaneous inflammation. Moisturizer is commonly used as a basis of management in AD, to ensure that that the transepidermal water loss (TEWL) is minimized. Evangelista *et al*.[21] had studied the effect of topical VCO on SCORing of Atopic Dermatitis (SCORAD) index, TEWL and skin capacitance in mild-to-moderate pediatric AD. Results showed that VCO could significantly improve SCORAD, TEWL, and skin capacitance value in AD patients. They had reported that the mean SCORAD indices for VCO-treated group reduced significantly, up to 68.23%. The reduction in SCORAD index was also supported by the earlier study, reported by Verallo-Rowell *et al*.[18] Evangelista *et al*.[21] reported that 47% of patients achieved moderate improvement and 46% showed an excellent response. Patients who were under VCO treatment also showed a significant reduction in TEWL (26.68-7.09) and increment in skin capacitance (32.0–42.3). Therefore, it was proposed that VCO is an occlusive agent that form a film on the AD skin to inhibit TEWL, strengthen the skin barrier from foreign substances and improve skin capacitance and hydration.[21]

CONCLUSION

Many studies have shown that VCO is a good alternative in the management of AD. VCO consists of triglycerides, medium chain fatty acids, phenolic acid, and polyphenols which exhibit several important therapeutic effects on AD skin. It is safe for topical application to both adult and children, nontoxic, and exhibits various biological properties such as antioxidant, anti-inflammatory, antibacterial, promotes wound healing and capable of moisturize AD skin. Studies have found that the phytochemicals present in VCO could function as a potential source of antioxidant in scavenging free radicals and toxic intermediates and hence decrease inflammation. Triglycerides of VCO could be hydrolyzed by skin flora to monoglycerides which are effective in inhibiting the growth of bacteria and reducing skin infection. Application of VCO could promote faster wound healing and encourage complete epithelialization. The occlusive and emollient properties of VCO could also inhibit TEWL, strengthen the skin barrier from foreign substances and improve skin capacitance and hydration. With the combination of these therapeutic effects and positive outcomes of the AD management, VCO is an effective, inexpensive treatment which could result in significant improvement to the skin condition.

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Conflicts of interest

There are no conflicts of interest.

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