

Clinical trial

The effect of topical virgin coconut oil on SCORAD index, transepidermal water loss, and skin capacitance in mild to moderate pediatric atopic dermatitis: a randomized, double-blind, clinical trial

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Abstract

Atopic dermatitis (AD) is a chronic skin disease characterized by defects in the epidermal barrier function and cutaneous inflammation, in which transepidermal water loss (TEWL) is increased and the ability of the stratum corneum to hold water is impaired, causing decreased skin capacitance and hydration. This study investigated the effects of topical virgin coconut oil (VCO) and mineral oil, respectively, on SCORAD (SCORing of Atopic Dermatitis) index values, TEWL, and skin capacitance in pediatric patients with mild to moderate AD, using a randomized controlled trial design in which participants and investigators were blinded to the treatments allocated. Patients were evaluated at baseline, and at 2, 4, and 8 weeks. A total of 117 patients were included in the analysis. Mean SCORAD indices decreased from baseline by 68.23% in the VCO group and by 38.13% in the mineral oil group ($P < 0.001$). In the VCO group, 47% (28/59) of patients achieved moderate improvement and 46% (27/59) showed an excellent response. In the mineral oil group, 34% (20/58) of patients showed moderate improvement and 19% (11/58) achieved excellent improvement. The VCO group achieved a post-treatment mean TEWL of 7.09 from a baseline mean of 26.68, whereas the mineral oil group demonstrated baseline and post-treatment TEWL values of 24.12 and 13.55, respectively. In the VCO group, post-treatment skin capacitance rose to 42.3 from a baseline mean of 32.0, whereas that in the mineral oil group increased to 37.49 from a baseline mean of 31.31. Thus, among pediatric patients with mild to moderate AD, topical application of VCO for eight weeks was superior to that of mineral oil based on clinical (SCORAD) and instrumental (TEWL, skin capacitance) assessments.

Introduction

Atopic dermatitis (AD) is a chronic, highly pruritic skin disease characterized by defects in the epidermal barrier function and cutaneous inflammation. The diminished epidermal function seen in patients with AD contributes to their striking susceptibility to colonization and infection with *Staphylococcus aureus* and an increased penetration of allergens.¹

Epidermal barrier function can be measured objectively according to the degree of transepidermal water loss (TEWL).² Transepidermal water loss is increased in AD, both in uninvolved skin and in involved skin in relation to severity.³⁻⁵ In AD patients, the ability of the stratum corneum (SC) to bind and hold water is also impaired, which results in a decreased skin capacitance or hydration.⁶ This impairment reflects a decrease in the content

of osmotically active amino acids in corneocytes (natural moisturizing factors), which impairs their ability to attract and bind water, and abnormal SC lamellae that cannot trap water within the corneocytes.^{7,8}

The management of AD should follow a stepped approach in which treatment steps are tailored to the severity of the disease.⁹ Even when the subject is clear of eczema, moisturizers should always be used and should form the basis of management.⁹ Moisturizers may also have another pivotal role in AD because a recent study showed that they may inhibit the onset of AD in high-risk subjects.¹⁰

Because AD is a global concern, there is a continuous search for the ideal moisturizer. There is little evidence to recommend the use of one moisturizer over another. However, patient preference and compliance remain the most important factors to be considered in selection. In general,

patients should use a dye-free, fragrance-free moisturizer and apply it at least twice per day.¹¹ Because most patients at our institution cannot afford most commercial moisturizers, we are always seeking possible cheaper alternatives.

Moisturizers may be categorized as occlusives, humectants, or emollients.¹² Occlusives coat the SC and reduce TEWL.¹⁰ Humectants are water-soluble materials with high water absorption capabilities, which attract water from the atmosphere to hydrate the skin.¹⁰ Emollients fill the spaces between desquamating corneocytes to create a smooth surface, providing cohesion and flattening the curled edges of corneocytes.¹³ One of the best occlusive ingredients currently available is mineral oil, which is derived from petroleum and consists of complex combinations of hydrocarbons.¹⁴ Mineral oil, as well as being an occlusive, also confers an emollient effect.¹⁴

Virgin coconut oil (VCO) has been demonstrated to be comparable with mineral oil as an emollient (increasing skin capacitance) in patients with xerosis.¹⁵ Another study established the effect of VCO in reducing SCORing Atopic Dermatitis (SCORAD) index values and demonstrated its *in vitro* antibacterial activity against *S. aureus* in adults with atopic disease,¹⁶ an effect that is notable because of the predisposition of AD to microbial colonization.¹ Extra-virgin coconut oil has also been used to reduce TEWL (indicating an improvement in barrier function) in preterm very low birthweight neonates.¹⁷

The beneficial effect of VCO on barrier function, its moisturizing and antibacterial properties, and its availability and safety make it a good candidate for the proactive treatment of AD. However, no trials have been reported on the use of VCO specifically for pediatric AD, although the pediatric population carries most of the burden of this disease. Atopic dermatitis has a prevalence of 10–20% in children in the first decade of life but is estimated to affect only 1–3% of adults.¹⁸ It is also known to be the first manifesting disorder in the atopic triad (AD, asthma, allergic rhinitis), a phenomenon called the atopic march. It is reported that half of AD patients subsequently develop asthma.¹⁹ If AD arises early in life, then perhaps prompt recognition and intervention may improve outcomes with respect to the clinical course of AD and may prevent the development of asthma and allergic rhinitis. In addition to its relevance to the AD-affected population of concern, this study is significant because it uses objective physiologic measurements (TEWL and skin capacitance) to determine efficacy, in addition to measuring clinical improvement. These parameters were not measured in previous studies on the efficacy of VCO in AD.

Objectives

The general objective of this study was to determine the effect of topical VCO versus that of mineral oil on

SCORAD index values, TEWL, and skin capacitance in pediatric patients with mild to moderate AD.

The specific objectives were to determine if outcomes in patients treated with VCO differed significantly from those in patients treated with mineral oil in terms of: (i) the percentage post-treatment change from baseline in mean total SCORAD indices; (ii) the mean post-treatment subjective component of SCORAD; (iii) the mean post-treatment objective component of SCORAD; (iv) the proportion of patients with moderate or excellent improvement based on SCORAD values; (v) the percentage post-treatment change from baseline in mean TEWL values; (vi) the percentage post-treatment change from baseline in mean skin capacitance values; and (vii) the incidence of adverse events.

Materials and methods

Patients and study design

Children aged 1–13 years were recruited between March 2011 and June 2012 to study the effects of topical VCO versus those of mineral oil on SCORAD values, TEWL, and skin capacitance in mild to moderate pediatric AD at the Dermatology Outpatient Department of the Jose R. Reyes Memorial Medical Center. Both newly diagnosed and previously documented AD cases were included. The diagnosis of AD was based on the modified Hanifin major criteria of a history of a chronic and relapsing course, pruritus, a pattern of facial and extensor eczema and xerosis at a young age that becomes flexural at an older age, and frequent association with a family history of AD.¹ The severity of atopic eczema was categorized according to the consensus conference on pediatric atopic dermatitis¹¹ as: mild, indicated by the presence of areas of dry skin with infrequent itching (with or without small areas of redness); moderate, indicated by the presence of areas of dry skin with frequent itching and redness (with or without excoriation and localized skin thickening); or severe, indicated by the presence of widespread areas of dry skin, with incessant itching and redness (with or without excoriation, extensive skin thickening, bleeding, oozing, cracking, and alteration of pigmentation). Only patients with mild to moderate AD were accepted.

Exclusion criteria precluded the participation of: patients who had applied or ingested antibiotic or steroid treatments in the two weeks prior to enrollment; patients with AD unresponsive to standard treatments (including moderate-potency topical corticosteroids); patients with persistent disease and/or frequent flares; patients who had been hospitalized as a direct consequence of AD; patients who required systemic therapies for flares and/or maintenance; patients with grossly infected lesions that required oral or IV antibiotics and ancillary therapy; patients with

dermatologic diagnoses other than AD; patients with hypersensitivity to VCO or mineral oil; patients with any genetic skin disorder or compromised immune state; and patients with any other major medical problem that the investigator deemed likely to increase the risk for adverse events associated with the intervention.

A certificate of approval from the ethics board committee of the Jose R. Reyes Memorial Medical Center was obtained before the clinical trial was initiated. Informed consent from one parent or caregiver and the assent of children aged ≥ 7 years were likewise secured prior to treatment.

Materials

Both VCO and mineral oil were obtained from local companies. They were repackaged into uniform opaque plastic bottles with a small opening to mask the color and scent of both oils. There are no other apparent differences between the oils as both are clear, colorless, and of similar viscosity. The bottles were coded (A or B) by the pharmacist.

For the treatment arm, a commercial VCO (Natures Blessings, Inc., Pasig, Philippines) was chosen on the basis of the literature on its packaging and its perceived market value. The oil was manufactured without heat but with water-soluble lipases, under sterile laboratory conditions that followed standard good manufacturing practice. Certifications of the oil's organic status from the US Food and Drug Administration (FDA) and the Food and Drug Administration of the Philippines (formerly the Philippines Bureau of Food and Drugs) were provided by the company. Subjects in the control group were treated with mineral oil (Marife C. Biscocho, Manila, Philippines).

Randomization, treatment allocation, and blinding

The study statistician generated a list of random numbers using the table of random numbers. An assigned resident, who was blinded to the codes, allocated the treatments randomly using the list and dispensed the packaged bottles accordingly. The codes were not disclosed to the investigators until the end of the study.

Study intervention

Each patient was given one bottle (250 ml) of VCO or mineral oil at each visit. The patient's parents were instructed to apply 5 ml of the assigned oil twice daily for several seconds to all body surfaces excluding the diaper area/inguinal area and the scalp. They were advised to neither give (excluding multivitamins) nor apply any other medication or emollient during the study period. All parents were asked to give the patients a bath once daily with warm water for 5–10 minutes and to apply the assigned oil immediately after bathing and at night. The

same mild soap (Dove™; Unilever PLC, London, UK) was given to all patients.

Clinical assessment

Patients were evaluated at baseline, and at 2, 4, and 8 weeks by the primary investigator. The primary endpoints of the study were the proportion of patients who demonstrated improvement in AD (excellent and moderate) in both groups. Secondary endpoints included the percentage changes from baseline in SCORAD values, TEWL, and skin capacitance, and the incidence of adverse events during the study. The study endpoints and digital photographs were obtained at every visit.

The clinical severity of eczema was determined using a standardized scoring system (SCORAD) developed by the European Task Force for Atopic Dermatitis.²⁰ This severity grading takes into account the intensity and extent of the eczema (objective score) and itch and sleep loss (subjective score) and is validated for the pediatric population.²⁰ After determining the objective and subjective components and total SCORAD index, patients were further stratified based on the percentage decrease of SCORAD values: moderate improvement was considered if the decrease from the baseline SCORAD index was $\geq 30\%$ but $< 75\%$,^{21,22} (National Institute of Allergy and Infectious Diseases, National Institutes of Health, Bethesda, MD, USA; pilot study using anakinra/kineret for the treatment of patients with severe atopic dermatitis; ongoing study), and excellent improvement was considered if the decrease was $\geq 75\%$.^{23,24}

Transepidermal water loss and skin capacitance were measured using the TEWAmeter® TM210 and the Corneometer® CM825 (Courage & Khazaka Electronic GmbH, Cologne, Germany), respectively. Measurements were taken on the volar surface of the right forearm of the patient after 15 minutes of inactivity in the room and according to the manufacturer's guidelines for the correct ranges of humidity (40–60%) and temperature (20–25 °C).

Stopping guidelines

The study was stopped in patients who experienced a flare of AD, defined as an episode requiring an escalation of treatment (necessitating other oral/topical medications).^{25,26} Appropriate rescue treatment and monitoring were delivered. These patients were considered as withdrawals from the study. Those who did not comply with the protocol of a twice-per-day application of test oils and those who used other moisturizers were also withdrawn from the study.

Dropouts were defined as patients who did not attend for follow-up within two weeks and whose outcomes were unknown by the end of the study period.

Sample size

The sample size was calculated accepting a power of 80%, with a two-sided alpha of 0.025, using the formula for computing the difference between two proportions. The projected success rate (those who will show at least moderate improvement) for VCO was set at 60% and the assumed success rate for mineral oil at 30%, based on a success rate of the placebo arm in another study.²¹ Calculations indicated that 49 patients in each study arm were needed (80% power, 5% level of significance). We aimed to recruit 108 patients to allow for a 10% drop-out rate.

Data processing and analysis

For demographic characteristics, Students *t*-test was used for continuous variables and Pearson’s chi-squared test for categorical data. Pearson’s chi-squared test was used to estimate differences in the proportions of patients achieving at least moderate improvement in SCORAD values, as previously defined. To measure the association between treatment and the occurrence of moderate or excellent improvement, treatment effects such as relative and absolute risk reductions and number needed to treat (NNT) were also computed. Statistical analyses were performed using STATA Version 10 (StataCorp LP, College Station, TX, USA). An intention-to-treat analysis was used for all patients included who received at least one dose of treatment. Test results that produced *P*-values of <0.05 were regarded as statistically significant.

Results

Study population

Of the 132 individuals who were screened, 117 met the entry criteria and were randomized to the treatment (VCO, *n* = 59) and control (mineral oil, *n* = 58) groups (Fig. 1). Of these patients, 12 were withdrawn because of poor compliance, adverse effects, and application of other emollients, and four were considered as dropouts as a result of their non-attendance at scheduled visits. There was no statistical difference in the number of withdrawals and dropouts between the two groups (*P* = 0.099). Because an intention-to-treat analysis was performed, all 117 patients were included in the full analysis. The baseline characteristics of the study population are summarized in Table 1. No statistically significant differences were noted between the two groups based on age, sex, duration of AD, lesion morphology, family or personal history of atopy, SCORAD indices, TEWL, or skin capacitance.

Clinical effects

Mean SCORAD indices decreased from baseline in both the VCO and mineral oil groups (Fig. 2). However, the percentage reductions from baseline in the VCO group were significantly higher at all measurement time-points. The post-treatment reduction in the mean SCORAD value in the VCO group was 68.23%, which was significantly higher (*P* < 0.001) than that in the mineral oil group (38.13%).

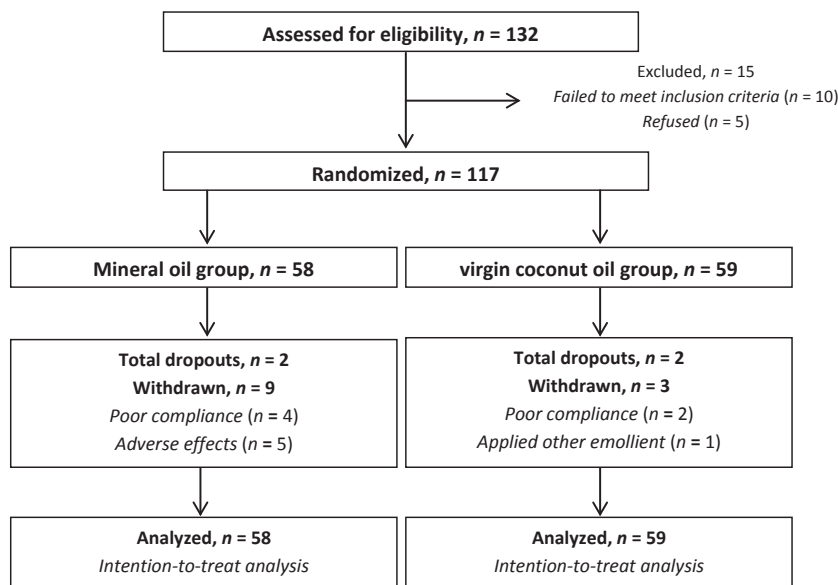


Figure 1 Trial profile showing patient populations

Table 1 Demographic characteristics of the study population (n = 117)

Characteristic	VCO group (n = 59)	Mineral oil group (n = 58)	P-value
Age, years, mean (95% CI)	4.69 (3.84–5.55)	4.14 (3.43–4.85)	0.3171
Sex, male/female, n	27/22	20/28	0.213
Duration, months, mean (95% CI)	18.53 (14.35–22.70)	18.60 (14.28–22.93)	0.9793
Lesion morphology, n (%)			
Infantile	10 (17)	11 (19)	0.776
Childhood	49 (83)	47 (81)	
Family history of atopy, n (%)	51 (86)	49 (84)	0.764
Asthma or allergic rhinitis, n (%)	9 (15)	9 (16)	0.834
SCORAD index, mean (95% CI)			
Total	13.28 (11.57–14.99)	12.29 (10.57–14.02)	0.4184
Extent and intensity (objective)	6.80 (5.25–8.35)	6.62 (5.23–8.01)	0.8622
Pruritus and sleep loss (subjective)	6.51 (5.72–7.29)	5.59 (4.78–6.39)	0.1036
TEWL, mean (95% CI)	26.68 (22.96–30.40)	24.12 (20.71–27.53)	0.3120
Skin capacitance, mean (95% CI)	32.00 (28.35–35.65)	31.31 (27.90–34.72)	0.7814

VCO, coconut oil; 95% CI, 95% confidence interval; SCORAD, SCORing Atopic Dermatitis; TEWL, transepidermal water loss.

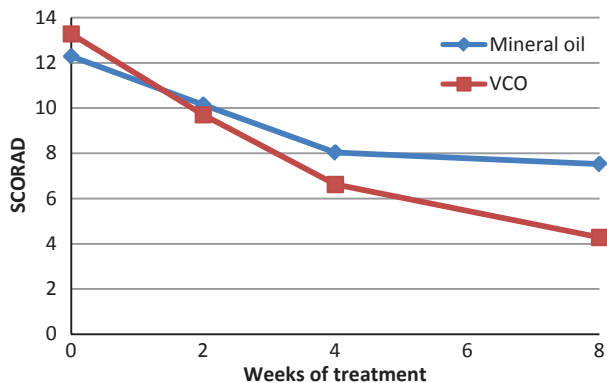


Figure 2 Mean SCORAD (SCORing Atopic Dermatitis) indices throughout treatment. VCO, virgin coconut oil

In addition, when the efficacy of each oil was assessed by comparing the proportions of patients who achieved reductions in the SCORAD index of $\geq 30\%$ and $\geq 75\%$, outcomes in the VCO group were likewise superior to those in the mineral oil group (Fig. 3). In the VCO group, 93% (55/59) of patients improved; 47% (28/59) achieved moderate improvement and 46% (27/59) showed an

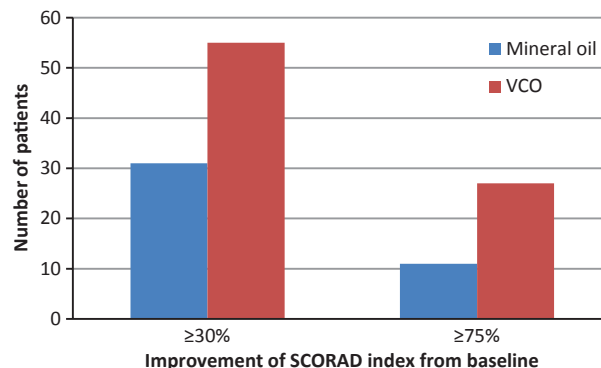


Figure 3 Patients achieving reductions of $\geq 30\%$ and $\geq 75\%$ in SCORAD (SCORing Atopic Dermatitis) indices after 8 weeks of treatment. VCO, virgin coconut oil

excellent response. In the mineral oil group, 53% (31/58) of patients improved; 34% (20/58) showed moderate improvement and 19% (11/58) achieved excellent improvement (Table 2). Computation of the relative risk reduction (RRR) for failed outcomes (those who did not achieve any improvement after the study period) revealed that VCO will improve SCORAD values 85.44% more (RRR = 0.8544, 95% confidence interval [CI] 0.5232–1.1401) than mineral oil. The absolute risk reduction (ARR) was 39.77%, favoring VCO (ARR = 0.3977, 95% CI 0.2436–0.5307). The NNT revealed that three and four patients were required to be treated with VCO for eight weeks to demonstrate moderate and excellent improvement, respectively.

SCORAD signs (intensity and extent of eczema) and symptoms (itch and sleep loss) were analyzed separately because the extent of eczema was reported to correlate well with intensity but weakly with itch and sleep loss.²⁷ Both the objective and subjective components of the SCORAD index were statistically lower in the VCO group than in the mineral oil group after treatment ($P = 0.0069$ and $P = 0.0021$, respectively) (Table 3).

To establish the protective effects of both oils on the skin barrier, percentage reductions in TEWL were compared between the groups. As with the SCORAD indices, a decreasing trend was observed in both groups throughout treatment: the VCO group achieved a post-treatment mean TEWL of 7.09 from a baseline mean of 26.68, whereas the mineral oil group demonstrated baseline and post-treatment TEWL values of 24.12 and 13.55, respectively (Fig. 4). Percentage reductions were also significantly higher in the VCO group at all points of measurement ($P < 0.001$ at all time-points); the VCO group demonstrated a post-treatment decrease in TEWL of 70.07%, whereas that in the mineral oil group was only 35.36%.

Table 2 Treatment success and failure in both groups

	Treatment success (reduction in SCORAD value of $\geq 30\%$ from baseline)			Treatment failure (reduction in SCORAD value of $< 30\%$ from baseline)	Total
	Moderate improvement	Excellent improvement	Total improvement		
VCO group, <i>n</i>	28	27	55	4	59
Mineral oil group, <i>n</i>	20	11	31	27	58
Total, <i>n</i>	48	38	86	31	117

SCORAD, SCORing Atopic Dermatitis; VCO, virgin coconut oil.

Table 3 Comparison of subjective and objective baseline and post-treatment mean SCORAD values between groups

	SCORAD	Baseline (95% CI)	Post-treatment (95% CI)
VCO group	Objective	6.80 (5.25–8.35)	2.39 (1.51–3.28)
	Subjective	6.51 (5.72–7.29)	2.12 (1.52–2.71)
Mineral oil group	Objective	6.62 (5.23–8.01)	4.49 (3.24–5.74)
	Subjective	5.59 (4.78–6.39)	3.48 (2.85–4.12)

SCORAD, SCORing Atopic Dermatitis; 95% CI, 95% confidence interval; VCO, virgin coconut oil.

To evaluate the emollient effects of the oils, the percentage increase in skin capacitance was compared between the groups. Both groups demonstrated an increasing trend in skin hydration (Fig. 5). In the VCO group, post-treatment skin capacitance rose to 42.30 from a baseline mean of 32.0, whereas that in the mineral oil group increased to 37.49 from a baseline mean of 31.31. However, although percentage changes were consistently higher in the VCO group than the mineral oil group, the difference was only statistically significant after eight weeks of treatment ($P = 0.0309$).

Five patients in the mineral oil group experienced adverse effects (increase in erythema, pruritus, or body surface area involved) necessitating rescue therapy with topical corticosteroids. However, the difference between the two groups in the occurrence of adverse effects was not statistically significant ($P = 0.089$).

Discussion

The present results indicate that although both oils showed beneficial effects on SCORAD, TEWL, and skin capacitance values, VCO was significantly better in improving all outcomes. Both mineral oil and VCO act as occlusives, coating the SC to retard TEWL.^{14,28} The weakened skin barrier is then strengthened, making the skin less suscepti-

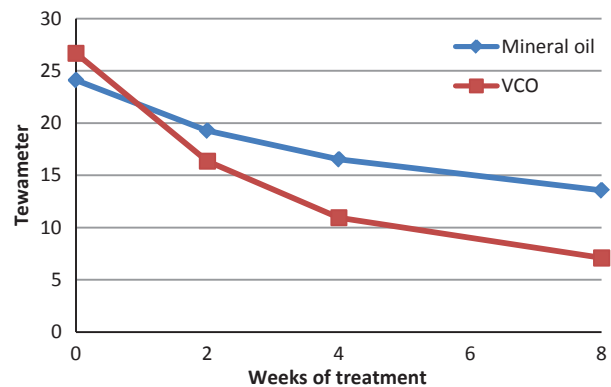


Figure 4 Mean transepidermal water loss (TEWL) throughout treatment. VCO, extra-virgin coconut oil

ble to attacks from noxious substances and preventing the development of eczema.²⁹ Both oils also exhibit emollient properties as evidenced by an increase in skin capacitance.

The superior effect of VCO over mineral oil may be explained by the fact that, as demonstrated in randomized controlled trials (RCTs), topically applied lipids not only coat but also penetrate the skin^{30,31} and are postulated to have anti-inflammatory activity,¹² making them invaluable in AD.

The permeation and penetration of the skin by vegetable oils are limited by their molecular size and other physiochemical properties.³² Oils high in short-chain and polyunsaturated components are superior. Although VCO has primarily saturated fats and contains 62% medium-chain fatty acids (MCFAs), it may provide greater permeation in AD patients because of their impaired skin barrier.³³

As well as improving barrier function, VCO may also address the chronic inflammation characteristic of AD. Virgin coconut oil has demonstrated anti-inflammatory activity in animal models of both acute and chronic inflammation.³⁴ It is postulated that the active compo-

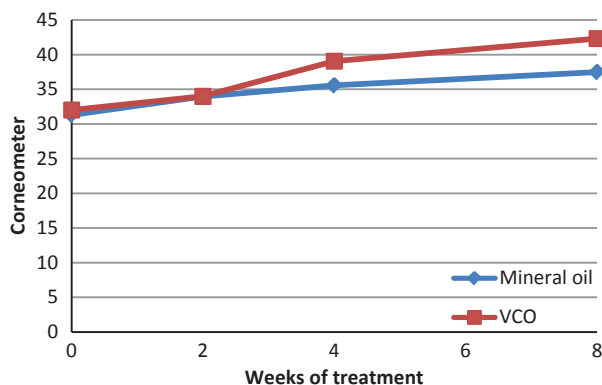


Figure 5 Mean skin capacitance throughout treatment. VCO, virgin coconut oil

nents against inflammation are MCFAs. When applied to the skin, MCFAs react with the lipases of the resident flora of the skin and are turned into free fatty acids, which penetrate the dermis where they reduce cellular inflammation. The exact mechanism for this has yet to be elucidated.¹⁶ In addition, VCO has been shown to destroy free radicals, compounds that may encourage skin inflammation. The antioxidant capacity of VCO may be attributable to phenolic compounds such as ferulic acid and p-coumaric acid.³⁵

Our results show that the difference between the groups in skin capacitance became statistically significant only after eight weeks of treatment. A possible explanation for this is that the anti-inflammatory effects of VCO require some time to become apparent, as evidenced by reductions in transudative weight and serum alkaline phosphatase activity in animal models of chronic inflammation after several weeks of treatment.^{34,35} It may be that the occlusive and emollient effects of the oil predominate during the first few weeks of treatment but that the inflammatory effect sets in as an adjunct to its occlusive and emollient effects as time goes by, further improving the barrier. A more improved skin barrier with the absence of inflammation will prevent further TEWL and better alleviate dryness, thereby increasing skin capacitance. These results indicate better outcomes with longer use. Studies on VCO have been short-term and have investigated its immediate effects (at 2 or 4 weeks of treatment) on the skin. This is the first study to investigate its long-term effect (defined as seven weeks in a previous study³⁶), which is preferred in a chronic disease such as AD.

Despite the absence of antihistamine medications, pruritus scores in our patients also decreased post-treatment, as shown by the subjective component of the SCORAD index. The resolution of pruritus may be explained by the concomitant improvement in skin barrier function.

Skin barrier disruption alters epidermal innervation and increases nerve density in the skin. Furthermore, emollients have shown anti-nerve growth effects in an animal model of acute dry skin.³⁷

Although this factor was not measured in the present study, VCO may also act as an antibacterial, which is beneficial in AD because the skin barrier defect predisposes to bacterial colonization. It is postulated that MCFAs have the capacity to alter bacterial cell envelopes, penetrate and physically disrupt cell membranes, and inhibit enzymes involved in energy production and nutrient transfer, leading to reversible and irreversible changes that may result in the death of the microbe.³⁸

One limitation of the present study is that it did not include atopic patients aged < 1 year. This was suggested by the institutional review board at the study institution because the prevalence of atopic eczema peaks in children at one year of age¹⁸ and in order to minimize the occurrence of possible adverse effects in infants. In addition, this study investigated the effects of VCO and mineral oil as monotherapy in mild to moderate AD and not as adjunctive therapy in patients with more severe disease. Another limitation of this study concerns the possibility that patient and parent blinding may have been compromised by the scent of VCO. However, as the oils were placed in similar opaque bottles and were of similar appearance and viscosity, and because the objective measures (TEWL and skin capacitance) were obtained by a blinded assessor, any possible bias caused by the scent is considered to have been sufficiently addressed.

None of the patients in the VCO group developed adverse reactions. This is concurrent with the findings of our literature search, which showed no published reports of contact dermatitis caused by VCO. Virgin coconut oil was not included in a recent listing of oils causing contact reactions.³⁹ To further establish the safety of VCO in pediatric patients, the authors conducted a quasi-experimental pilot study prior to this RCT, in which all patients underwent open testing to VCO (Evangelista MT, Villafuerte LL, Ismael DK; The effect of topical virgin coconut oil on skin barrier function and hydration of mild to moderate pediatric atopic dermatitis: a quasi-experimental, open-label trial; unpublished data 2011). There were no documented reactions.

Virgin coconut oil is different from coconut oil, which is produced by drying the coconut meat (copra). Because the impurities of copra are released into the coconut oil, this oil must be purified or refined. Once the coconut oil has been refined, it is bleached to remove any remaining impurities and then deodorized under high heat to remove the coconut fragrance. Sodium hydroxide is used to break down the fatty acids so that the coconut oil will have a longer shelf life. By contrast, VCO is obtained by a wet-

milled, cold-press process which does not involve the addition of chemicals or application of heat.⁴⁰

A study comparing the use of, respectively, oral VCO and coconut oil as an antioxidant and agent to maintain lipid metabolism clearly showed the former to be superior.⁴⁰ Although there are no trials comparing the use of coconut oil and VCO for dermatologic conditions, as the antioxidant content is postulated to contribute to the anti-inflammatory activity,^{34,35} and because antioxidant capacity and fatty acids (the active components of VCO) in coconut are adversely affected by refining, heat, and solvent extraction, cold-pressed, unrefined oils such as VCO may be more favorable in AD.

Conclusions

Among pediatric patients diagnosed with mild to moderate AD, topical application of VCO for eight weeks was superior to that of mineral oil based on clinical (SCORAD) and instrumental (TEWL, skin capacitance) assessments.

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