STUDY

Novel Antibacterial and Emollient Effects of Coconut and Virgin Olive Oils in Adult Atopic Dermatitis

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Background: **Atopic dermatitis (AD) skin is dry and readily colonized by *Staphylococcus aureus* (*SA*). Coconut and olive oils are traditionally used to moisturize and treat skin infections.**

Objective: **To compare virgin coconut oil (VCO) and virgin olive oil (VOO) in moisturizing dryness and removing *SA* from colonized AD skin.**

Methods: **This was a double-blind controlled trial in two outpatient dermatology clinics with adult AD patients who were diagnosed by history, pattern, evolution, and skin lesions and who were randomized to apply VCO or VOO twice daily at two noninfected sites. *SA* cultures, photography, and objective-SCORAD severity index (O-SSI) scoring were done at baseline and after 4 weeks.**

Results: **Twenty-six subjects each received VCO or VOO. Of those on VCO, 20 were positive for *SA* colonies at baseline versus 12 on VOO. Post intervention, only 1 (5%) VCO subject remained positive versus 6 (50%) of those on VOO. Relative risk for VCO was 0.10, significantly superior to that for VOO (10:1, $p = .0028$; 95% CI, 0.01–0.73); thus, the number needed to treat was 2.2. For the O-SSI, the difference was not significant at baseline ($p = .15$) but was significantly different post treatment ($p = .004$); this was reduced for both oils ($p < .005$) but was greater with VCO.**

Conclusion: **VCO and monolaurin's O-SSI reduction and in vitro broad-spectrum activity against *SA* (given clinical validity here), fungi, and viruses may be useful in the proactive treatment of AD colonization.**

ATOPIC DERMATITIS (AD) is characterized by dry skin and the frequent isolation of *Staphylococcus aureus* (*SA*) from infected eczema and chronic lesions and as a colonizer of clinically uninfected atopic skin.[1] The prevalence of colonization in normal skin is about 5%; in lesional and nonlesional atopic skin of adults, children, and infants, it is 64 to 100%.[2] Thus, based on a recent systematic review of AD, it is felt that the use of a topical antibiotic for treating *SA* infection can be effective, but the development of resistance is a concern. Treatment of *SA* colonization is not as clear-cut. The review further states that antibiotics generally have a minimal therapeutic effect on dermatitis without signs of infection.[3]

A recent review on the mechanisms of disease in AD explored (1) the role of *SA* colonization and infection in helping generate the chronic inflammation characteristic of atopic skin and (2) the role of inflammation (from *SA* and from genetic and environmental causes) that leads to barrier dysfunction that culminates in dry skin. Rather than endorse the more common reactive management of AD, the review recommended early and proactive intervention with antiseptic lotions to reduce *SA* colonization.[4]

Few evidence-based data are accepted in modern therapeutics for the widespread traditional practice of using coconut oil (CO) on dry infected skin.[5] In a small trial of patients with xerosis, Agero and Verallo-Rowell found CO comparable to mineral oil in skin moisturization and the absence of irritant effects.[6] No trials have been reported on the topical use of CO specifically for AD, clinically infected or not.

In recent years, the term ''virgin'' has been used to indicate a health-related value in coconut and olive oils. The virgin status of olive oil is achieved by extracting the oil 24 to 48 hours after harvest and through Good Manufacturing Practice (GMP), including the avoidance of heat, light, and air during processing and storage. These precautions protect heat-sensitive phytochemicals and help prevent the hydrolysis of triglycerides into their component free fatty acids (FFAs), which leads to the rancid smell of spoilage and to skin irritation.[7] The amount of FFAs present is used to define the degree of virginity of the oil, as follows: ordinary, a maximum of

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DOI 10.2310/6620.2008.08052

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3.3%; fine virgin, a maximum of 1.5%; and virgin, less than 1%. ''Extra-virgin olive oil'' (EVOO) is a retailing name used to emphasize the fact that the oil is pressed cold immediately after harvest.[8]

Virgin coconut oil (VCO) is also processed on the day of harvest, under similar GMP guidelines.[9] Unlike virgin olive oil (VOO), which has 82% unsaturated fatty acids (FAs), VCO has only 8% unsaturated FAs. The other 92% of the FAs are saturated and chemically stable such that the standard of 0.5% FFA content in VCO is readily achieved as long as the moisture content is kept at the standard of 0.12% or less. Since the nutmeat is exposed to its water at tropical temperatures for 10 months, extra-virgin coconut oil (EVCO) is considered ''cold pressed'' when the nutmeat is pressed at a temperature that does not exceed 39°C.[10] For the objectives of this study, these differences are minor; hence, the more common terms for these two virgin oils—VCO and VOO—are used in this article.

This 4-week randomized controlled blinded trial compared the effects of VCO and VOO on *SA* colonization of normal AD skin and on the extent and intensity of objective AD parameters, using the objective SCORing Atopic Dermatitis (SCORAD) severity index (O-SSI).

Methods

The patients came from two general dermatology clinics. Because there were no previous topical VCO studies in children, pediatric subjects were not allowed by the institutional review board of the Skin and Cancer Foundation. Adult AD diagnoses were 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 younger age, becoming flexural at adult age; and frequent association with a family history of AD[1] (Table 1).

Eligibility Criteria and Stratification

Those included were newly diagnosed patients aged 18 to 40 years and patients who were previously documented and managed as having AD. Patients with new and old cases of AD had low to high moderate O-SSI scores and had not taken topical steroids or topical or oral antibiotics for at least 2 weeks before enrollment. Excluded were those with (1) grossly infected lesions needing oral or intravenous antibiotics and ancillary therapy; (2) dermatologic diagnoses other than AD; (3) previous hypersensitivity to coconut or olive oil, known diabetes mellitus, or compromised immune states. All patients who met the criteria were oriented on the study's objectives, procedures, and expected outcomes. Informed consent for the study and for photographs was obtained.

Prior to randomization, the participants were stratified on the basis of age and O-SSI score, to control potential confounding variables. The O-SSI score, at baseline and at the end of intervention, was calculated with the formula $A/5 + 7 \times B/2$ (range of 0–83), where ''A'' represents extent (graded 0–100, based on the rule of nines on a front/back drawing of the patient's inflammatory lesions) and ''B'' represents intensity (graded 0–18, based on a 0–3 rating of erythema/darkening, edema/papulation, oozing/crust, excoriation, lichenification/prurigo, and dryness); the cut-off points were mild (score < 15), moderate (15–40), and severe (> 40).[11]

Randomization, Treatment Allocation, and Blinding

The preparation of test bottles, the randomization key, and the codes were carried out by the pharmacist of Skin Sciences Laboratory, Inc., and was disclosed to the investigators only at the end of the study. Subjects previously matched by age and O-SSI score underwent simple concealed random allocation (by drawing rolled pieces of paper labeled ''A'' or ''B'') to control or treatment arms by the two dermatology residents, both of whom were blind to the codes and who also dispensed the packaged bottles according to a random listing.

Preparation and Application of Oils

High-quality pure oils were sourced and repackaged in uniform medicinal opaque plastic bottles with a small opening to mask the color and scent of both oils. Unlike the scent of ordinary CO (which is prepared with heat), that of VCO (prepared without any heat) is like that of VOO (ie, botanical and musty). Upon application of either oil, the scent is notable but disappears within just a few minutes. Hours later, neither the patients nor the investigators who see them can identify either oil on their skin by scent. VCO is clear as water and colorless; VOO is also clear but is light yellow green. As either oil is poured onto the hand and applied to the treatment sites, the skin's color makes the VCO and VOO look similarly brownish and indistinguishable from each other.

For the control arm, a commercial VOO was chosen on the basis of its package literature and perceived market value; it was then subjected to a series of microbiologic tests to ensure that it contained no unwanted pathogens.

Table 1. Demographics and Characteristics of Patients with Atopic Dermatitis (Modified Hanifin Criteria) in the Coconut Oil and Olive Oil Study Groups

Characteristic	*Coconut Oil (N = 26)*	*Olive Oil (N = 26)*	p *Value**
Age in years			
Mean ± SD	32 ± 3	31 ± 4	—
Range	29–35	21–39	.76† (NS)
Sex			
Male	13 (50%)	14 (54%)	—
Female	13 (50%)	12 (46%)	.78† (NS)
Duration in years (mean ± SD)	15 ± 3	18 ± 5	.09† (NS)
Pruritus	25 (96%)	23 (88%)	.515† (NS)
Lesion morphology			(NS)
Childhood: facial/extensor	12 (46%)	13 (54%)	0.58†
Adult: flexures/lichenification	21 (81%)	22 (85%)	1.00†
AD in family			
Yes	12 (46%)	8 (31%)	—
No	14 (54%)	18 (69%)	.26† (NS)

AD = atopic dermatitis; NS = not statistically significant.
*Significant *p*-value if < .05.
†Computed using statistical software Epi-Info V6 for tests of proportions, independent samples *t*-test, and chi-square test.

The VCO used by Agero and Verallo-Rowell was processed without heat but with water-soluble food-derived lipases. The VCO used for the intervention arm of the present study was manufactured without heat under sterile laboratory conditions that followed standard GMP, to avoid the use of chemicals. The temperature was kept at about 33°C and no higher than 39°C, temperatures similar to those experienced under tropical sunlight. The resulting VCO passed a series of microbiologic tests and was found to have no unwanted pathogens.

The instructions for applying both EVCO and EVOO were as follows: ''On the affected areas that include the test sites, apply 5 mL of EVCO two times a day and massage gently but thoroughly into the skin for several seconds. Do not apply other emollients, creams, or oil-based products that can mask the effect of the oil.''

The test bottles were brought in and replaced with new ones at each visit.

All patients were advised to practice good skin hygiene, and all were given a bar of white baby soap to use.

SA Cultures

Collection of Skin Swabs

The two resident-investigators together selected two clinically uninfected AD-affected sites, each with an easy-to-identify anatomic landmark. These sites and their identification landmarks were described on the data collection sheet, and photographs were taken. Starting at the center, cotton swabs soaked with sterile normal saline solution were swept over each site (one swab for each site); the cotton swabs were then submitted to the medical technologist at the microbiology laboratory of the Quirino Memorial Medical Center, Pasig, Philippines. After 4 weeks of treatment, cotton swab samples were again taken from the identical sites of the first cultures and sent to the microbiology laboratory.

Colony Growth and Growth Effectiveness

With standard laboratory technique, *SA* was identified as gram-positive cocci and not resident skin flora or spurious contamination. Colony growth yield of microorganisms considered as significant was also based on standard microbiologic criteria.[12]

At baseline and at the end of intervention, the cultures were examined by a medical technologist who was blind to the treatment arms of the study. The presence of colony counts from one or both sites was reported as ''positive''; the absence of colony counts in both sites was reported as ''negative.'' All colony count reports were given to the investigators after the second set of cultures was done.

SA colony growth effectiveness was based on the absence of *SA* colonies in two separate cultures from two separate sites of clinically noninfected atopic skin after administration of the corresponding intervention.

Statistical Analysis

Descriptive Statistics

Descriptive statistics included (1) means and their standard deviations for categorical variables and (2) percentage frequency distribution for categorical data. Testing of homogeneity of samples was done with the chi-square test for categorical data and an independent *t*-test for continuous numerical data.

The proportion of significant colonies was compared before and after the trial with nonparametric chi-square tests. Precision estimates were pegged at 95% confidence limits. All tests of significance were carried out with NCSS-PASS software (NCSS, East Kaysville, UT).

Dropouts and Noncompliers

Patients were taught to assess their symptoms and their skin's appearance daily. They were advised to contact the investigators for further treatment when their condition was rated as worse. If topical antibiotics were dispensed, the subject was included in analysis as a dropout; patients who failed to comply with the regimen were classed as protocol violators.

Results

A total of 52 subjects met the inclusion criteria and were randomized to receive topical VCO ($n = 26$) or VOO ($n = 26$). There were no dropouts or protocol violators. Tests of baseline homogeneity of the sample revealed no statistically significant differences in age ($p = .76$), sex ($p = .78$), duration of illness ($p = .09$), pruritus ($p = .515$), lesion morphology ($p = .58$), or family history of atopic dermatitis ($p = .26$) (see Table 1).

The clinically noninfected sites that were (1) chosen and recorded for culture swabs at baseline; (2) identified from photographs, drawings, and anatomic landmarks after 4 weeks of intervention with VCO or VOO, and (3) reswabbed, were the following: antecubital (VCO, 16; VOO, 14), popliteal (VCO, 8; VOO, 9), and trunk (VCO, 2; VOO, 3).

SA Culture Growth Effectiveness

Assessment of the growth effectiveness of the *SA* cultures was based on the *SA* culture results (positive or negative colonies) at baseline as compared with postintervention results. Of the 20 patients whose cultures were positive and who were randomized to VCO, 1 subject (5%) remained positive; of the 12 patients whose cultures were positive and who were randomized to the VOO arm, 6 (50%) remained positive. Compared to the VOO arm, the calculated relative risk of nontreatment was 0.10 among those in the VCO arm and was thus superior to that of the control arm. The risk of nontreatment with VOO was 10 times higher (or 1/.10), relative to the VCO arm. The calculated absolute risk reduction was −.45, which means that just two subjects needed to be treated with the VCO (number needed to treat [NNT] = 2) to prevent one treatment failure (or failure to sterilize cultures) (Table 2).

Table 2. Relative Risk for Colonization by *Staphylococcus aureus**

			Post Intervention			
Group	*Baseline*		*SA (+)*	*SA (−)*	*Total*	*Relative Risk*†
VCO	*SA* (+)	20 (77%)	1 (5%)	19 (95%)	20	Rt = 1/20 = 0.05
	SA (−)	6 (23%)	—	—	6	—
Total		26 (100%)			26	
VOO	*SA* (+)	12 (46%)	6 (50%)	6 (50%)	12	Rc = 6/12 = 0.50
	SA (−)	14 (54%)	—	—	14	—
Total		26 (100%)			26	RR = 0.10 (95% CI, .01–0.73) $p = .0028$

Rc = risk of (+) colonies with virgin olive oil treatment; RR = relative risk; Rt = risk of (+) colonies with virgin coconut oil treatment; *SA* = *Staphylococcus aureus*; VCO = virgin coconut oil; VOO = virgin olive oil.

*Based on positive/negative colony readings (number and percentage) at baseline and 4 weeks after topical use of VCO versus VOO.

†Rt (.05)/Rc (.50) = 0.10 (< 1, treatment benefits; > 1, treatment is harmful); absolute risk reduction (ARR) = Rt − Rc; number needed to treat is 1/ARR = 2.2.

Effect on O-SSI Score

At baseline, no significant difference was seen between the O-SSI scores of the two treatment arms ($p = .15$), but postintervention scores differed significantly (-4.1; $p = .004$). Postintervention SCORAD index scores were lower for both oils but were statistically lower in the CO group (Table 3 and Fig 1). The composite interpretation of SCORAD index scores for both groups was from high moderate to low moderate for the VCO group and "worsened" for the VOO group (Fig 2).

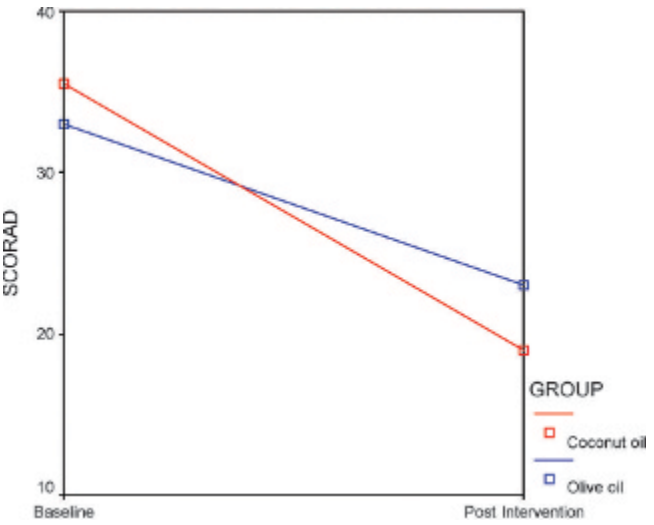

Figure 1. Changes in objective SCORAD index scores from baseline to after intervention.

Discussion

Antimicrobial Action of FAs from Edible Oils

VCO has a long tradition of use in treating infections. Like all edible oils, VCO is made up of triglycerides, each one with a simple glycerol core of three carbons, to each of which an FA is attached. In the stomach and the upper part of the small intestine, ingested VCO is digested by lipase enzymes into di- and monoglycerides, glycerol, and FFAs.[13]

Lipases are also present in the aerobic organisms of the normal skin flora. Holt reported that from the normal skin of adults and children, almost all 42 strains of isolated Micrococcaceae *Sarcina*, 40% of 50 aerobic skin diphtheroids, and 20% of 58 aerobic nasal diphtheroids produced strong or active lipases. The author postulated that action of these lipases on skin lipids accounts for the production of FAs that acidify the skin (average hydrogen ion concentration [pH], 5.5) and provide the surface of the skin its "acid mantle."[14]

Similarly, we postulated that these lipases (and those known to be produced by *SA*)[15] may hydrolyze the triglycerides of topical VCO to levels higher than the 1 to 7% monoglycerides and the 0.5% FFAs that are normally present in unhydrolyzed VCO.[16]

Studies on lipids in the 1960s by Kabara and colleagues showed medium-chain (C-8 to C-14) FAs and their monoglycerides to have antimicrobial effects against several laboratory organisms.[17] In the 1990s, more laboratory studies confirmed the antimicrobial activity of these lipids against gram-positive and some gram-negative organisms (including *Neisseria gonorrhoeae*,[18] *Helicobacter pylori*,[19] and *Chlamydia trachomatis*[20]) as well as *Candida albicans* yeast[21] and enveloped viruses.[22]

Since 1998, some clinical studies have confirmed these laboratory data, specifically data on monolaurin, the monoglyceride of lauric acid from VCO. A 2% gel preparation of Lauricidin (Skin Sciences Laboratory, Inc, Pasig City, Philippines), which contains 90% pure monolaurin, significantly degermed *SA* cultured from health workers' hands after hospital duty.[23]

Another study cultured the skin lesions of 100 pediatric patients. The top isolates were *SA*, coagulase-negative *SA*,

Table 3. Objective SCORAD Index Scores at Baseline and Post Intervention

Objective SCORAD Index	*VCO Group (N = 26) (%)*	*VOO Group (N = 26) (%)*	*Mean Difference (%)*	p *Value*
Baseline	39.2 ± 6.4	36.6 ± 6.3	2.6	.15 (NS)
Post intervention	22.6 ± 3.6	26.7 ± 5.7	−4.1	.004*†
Mean difference	16.6	9.9	—	—
Percent reduction	46.8	30.1	—	—

NS = not statistically significant; SCORAD = SCORing Atopic Dermatitis; VCO = virgin coconut oil; VOO = virgin olive oil.
*Significant difference at $p < .05$.
†Mann-Whitney *U* test.
The drop in objective SCORAD index score from baseline was statistically significant within both the VOO group (36.6 to 26.7 points) and the VCO group (39.2 to 22.6 points), both Wilcoxon signed ranks ($p < .005$), although percent reduction with VCO is more than that with VOO.

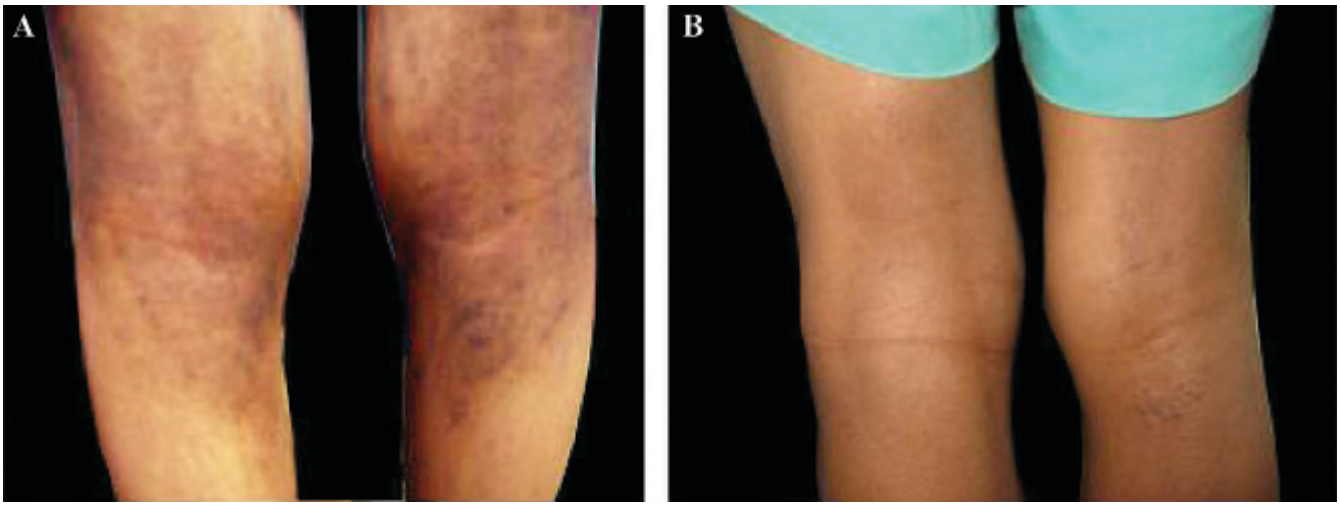

Figure 2. *A*, Non-infected atopic dermatitis site before treatment with virgin coconut oil, SCORAD 35. *B*, The same site 4 weeks after treatment with virgin coconut oil, SCORAD 20.

Streptococcus spp, *Enterobacter* spp, and *Escherichia vulneris*. The sensitivity of these organisms to penicillin, oxacillin, erythromycin, fusidic acid, mupirocin, and vancomycin varied significantly, demonstrating low to high susceptibility, across the different isolates (Fisher exact test = 0.000; $p < .05$). In marked contrast, sensitivity to monolaurin did not significantly differ across the different bacterial isolates (Fisher exact test = 0.110; $p > .05$), reflecting high antibacterial activity. There also was a statistically significant and marked difference in resistance rates. *SA*, coagulase-negative *SA*, and *Streptococcus* spp did not exhibit any resistance to monolaurin as opposed to the varying resistance observed with the other antibiotics in this study.[24]

Still another study showed significant activity against *SA* by 13 lauric monoester formulations in vitro and in vivo in mice.[25]

Mechanism of Action of Monolaurin

The mechanism of action (MOA) of monolaurin as an antimicrobial is ''novel'' in that it differs from that of most conventional antibiotics. A recent review of the many lipid studies conducted during the last 50 years (mostly in the laboratory), showed similar study results and similar proposed MOAs for the antimicrobial effects observed.[26] A common hypothesis explains the antimicrobial effects of monolaurin and the other medium-chain monoglycerides as being based on their capacity to alter the bacterial cell envelope. It is postulated that by virtue of size, these lipids are small enough to be readily dissolved in the lipid phase, to penetrate and physically disrupt cell membranes, and to inhibit enzymes involved in energy production and nutrient transfer, leading to reversible and irreversible changes that may lead to the death of the cell. A sophisticated electron microscopic and two-color fluorescent assay showed that on contact with these monoglycerides, bacteria show visible changes by 5 minutes and shrinkage and disintegration of cell membranes after 10 minutes, leading to the death of the bacteria.[27]

Conversely, conventional antibiotics are ionized molecules that do not readily bridge the membrane barrier because of charge or size and that act more on bacterial enzymes (although more antibiotics with similar action on the bacterial cell wall, called ''novel,'' have been described recently[28]).

Concurrent with this MOA in explaining the significant difference in the antimicrobial action of VCO versus VOO is the difference in the sizes of their monoglyceride FAs.[29] After lipase hydrolysis, all FAs produced by VOO are long-chain FAs, mostly C-18 (C-16 to C-24, except for 0.1% C-14). VCO produces 82% medium-chain FAs, mostly C-12 (C-6 to C-14) FA.[12] This may also explain the initial observations and pilot studies that prompted this study. At the authors' clinics, consistent improvement or clearing of inflamed or mildly to moderately infected psoriasis and AD lesions was noted after VCO application.[30]

VCO Natural FAs and AD Dry Skin

Emollients are a standard of care for prevention of dryness, steroid-sparing effect, and maintenance therapy in AD. Fixed vegetable oils coat the skin, occluding and protecting it by slowing down transepidermal water loss and increasing

hydration within the stratum corneum and top layers of the dermis. They also ''glue down'' dry and desquamating skin cells, making the skin look less rough and scaly.[31] The AD patients who were treated with VCO in this study had significantly lower objective SCORAD scores for dryness and dryness-related conditions, such as excoriation and lichenification, and for erythema, edema, and papulation.

Adverse Reactions to Olive Oil, VCO, and Monolaurin

Olive oil is a very weak irritant, and adverse side effects from topical use are rare. Of 21 patients with reported cases of contact allergy to olive oil, 4 patients had occupation-related hand eczema; 1 of these 4 had positive patch-test and use-test results after 2 days.[32] One possible cause for these reactions may have been the gallates—antioxidants that may be used to stabilize the mostly monounsaturated (and some polyunsaturated) FAs of olive oil—that have been reported to produce contact dermatitis.[33] Antioxidants are not needed for (nor added to) stable and saturated VCO.

VCO has caused no reported contact dermatitis and should not be mistaken for the cocamides, which are VCO FAs treated with amidoamines. These popular surfactants and foam boosters in shampoos and cleansers have increasingly been reported to produce allergic reactions. However, a double-blind controlled pilot retest study of 12 patients previously allergic to cocamidopropyl betaine (CAPB) found that only 3 patients (25%) had doubtful reactions. The authors concluded that the results substantiated previous experience that doubtful and mild reactions to CAPB may represent irritant rather than true allergic reactions.[34] In this patch-testing study and in a toxicology report that implicated the nitrosylation of the FAs as a cause of reactions, the test results for CO and lauric acid were negative.[35]

The inadvertent intake of topical VCO and monolaurin is safe: CO has a long dietary history among tropical people, and monolaurin is a component of breast milk. Since 1964, monolaurin has been ''generally recognized as safe'' (GRAS) by the US Food and Drug Administration. A similar safety record has been shown in animals for which monolaurin constitutes up to 25% of the total diet.[29] The extensive topical use of VCO and the topical and oral use of monolaurin in our clinics have caused no adverse reactions.[30]

Conclusion

A history of safe topical use and no known or reported cases of contact dermatitis, along with its dual effects as moisturizer and antiseptic, opens up more research and clinical possibilities for virgin coconut oil (VCO) and monolaurin. In the laboratory, VCO and monolaurin have also shown antimicrobial effects on fungi[36] and enveloped viruses[37] that (like *Staphylococcus aureus)* may infect or colonize sites of atopic dermatitis.

Acknowledgments

Patients were seen and examined at the two charity clinics of the Skin and Cancer Foundation, Inc., in Pasig, Rizal, and Makati, Rizal, Philippines. The microbiologic testing was done at the microbiology laboratory of the Quirino Memorial Medical Center, Pasig, Rizal, Philippines.

Skin Sciences Laboratory, Inc., of Pasig, Rizal, Philippines, provided material support by supplying and packaging virgin coconut oil and virgin olive oil in uniform bottles.

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