NATURAL MEDICINE JOURNAL

May 7, 2014

Treatment of Dermal Infections With Topical Coconut Oil

A review of efficacy and safety of Cocos nucifera L. in treating skin infections

By Lindsey K. Elmore, PharmD, BCPS

Abstract

Coconut oil, and many other portions of the plant *Cocos nucifera L*, have been hypothesized to have antimicrobial and antifungal activity. Medium-chain fatty acid constituents of coconut oil including lauric acid, capric acid, and others provide antimicrobial effect by disrupting bacterial, fungal, and viral cell membranes, leading to cell death. This review summarizes in vivo and in vitro studies of topical anti-infective properties of coconut oil and the medium-chain fatty acids contained within, and describes the proposed use of coconut products for dermal infections.

Introduction

Referred to as the “tree of life” because of its many uses, coconut, *Cocos nucifera L.*, is a fruit tree found in warm, humid climates with well-drained soil. Different cultures around the globe have cultivated the coconut tree and utilized the various parts of the coconut fruit [water, meat (from which coconut oil is isolated), and husks] for a plethora of uses from biofuel to food. Coconut oil has traditionally been used as a medicinal agent for cancer, diabetes, diarrhea, dry skin, and psoriasis and is used as an antibacterial, antifungal, and antiviral agent for the treatment of dermal infections.[1–3] Evaluation of *Cocos nucifera L.* as an anti-infective agent is very important due to the increased prevalence of antibiotic-resistant infectious microorganisms, and the dearth of novel antibiotics in the pipeline.[4,5]

Medicinal properties of *C. nucifera* are attributed to 3 medium-chain fatty acids found in coconut fat: lauric acid, the most abundant fatty acid, capric acid, and caprylic acid.[3] Lauric acid is a medium-chain fatty acid that, when esterified with glycerol, results in the monoglyceride monolaurin.[6] Monolaurin has been suggested as the most potent antimicrobial agent among those found in *C. nucifera*.[7]

The anti-infective mechanism of fatty acids such as those found in *C. nucifera* is poorly understood. One hypothesis is that fatty acids interfere with the bacterial cell structure and acids interfere with cellular energy production, causing disruption of the electron transport chain and oxidative phosphorylation.[3,6] Fatty acids may also inhibit enzyme activity, impair nutrient uptake, generate cellular degradation products, or cause direct lysis of infectious cells.[3,6]

This literature review summarizes *in vivo* and *in vitro* studies of virgin coconut oil (VCO), lauric acid, capric acid, monolaurin, and other fatty acids as microbicides against bacteria, fungus, and viruses that cause dermal infections.

Literature Review

A MEDLINE, International Pharmaceutical Abstracts, Natural Standard, and Natural Medicine database search was conducted for clinical trials published in English using the key terms coconut oil, *Cocos nucifera*, *Cocos nucifera L.*, lauric

acid, monolaurin, dermal infection, skin infection, antibiotic, and antimicrobial. *In vitro* and *in vivo* trials published in English that evaluated the anti-infective efficacy and safety of coconut oil and its components were selected and evaluated.

Clinical Evidence

Studies have evaluated the antimicrobial activity of *Cocos nucifera L.* husk fiber, coconut oil, and lauric acid and monolaurin extracts. Table 1 summarizes *in vitro* and *in vivo* studies.

A double-blind, randomized controlled trial compared virgin coconut oil (VCO) to virgin olive oil (VOO) for efficacy in removing colonized *Staphylococcus aureus* in 26 patients aged 18 to 40 years with atopic dermatitis (AD). This study included patients with new and old AD with low to high moderate scores on the SCORing Atopic Dermatitis (SCORAD) severity index (O-SSI, an objective scoring system that accounts for spread and intensity of lesions, as well as subjective symptoms such as pruritus and insomnia; scores range from 0 to 40). Patients were excluded if they had grossly infected lesions requiring antibiotics, any dermatologic diagnosis other than AD, hypersensitivity to VCO or VOO, or an immunocompromised state (including diabetes), or if they were on topical steroids or topical/oral antibiotics within the past 2 weeks. Before initiation and after 4 weeks of treatment, cotton swabs of well-defined lesions were obtained and analyzed for presence of *S. aureus*. Both groups applied 5 mL of either VCO or VOO on the affected area twice daily and were instructed not to put any other emollients, creams, or oil-based products on the lesions.[8]

Patients were on average 31.5 years old, approximately 50% were female, and duration of AD was 16.5 years; there was no difference in baseline O-SSI scores. At baseline, 20 patients in the VCO group and 12 in the VOO group were colonized with *S. aureus*. Of the patients who were initially colonized, after 4 weeks of treatment, 1 patient (5%) treated with VCO remained colonized with *S. aureus compared* to 6 patients who were treated with VOO (50%) (RR=0.10, 95% CI: 0.01–0.73, *P*=0.028). Post-intervention O-SSI scores in the VCO group were significantly lower than the VOO group (mean difference -4.1, *P*=0.004). No adverse effects to VOO or VCO were reported.[8]

A mixed *in vitro* and *in vivo* study examined the antibacterial activity of lauric acid against *Propionibacterium acnes* and other skin flora. *P. acnes* is the main causative organism of acne vulgaris, a disease that affects between 50% and 95% of adolescents at some point in their lives and 40 million people in the United States.[9] Current treatments, such as benzoyl peroxide (BPO), have undesirable side effects including burning, drying, irritation, and erythema.[10] *S. aureus*, *Staphylococcus epidermidis*, and *P. acnes* were co-cultured with either BPO or lauric acid. Following incubation of agar plates containing *P. acnes* and either BPO or lauric acid, the minimum inhibitory concentration (MIC) against each organism for BPO were 15.6, >100, and 62.5 mcg/mL, respectively, compared to 0.9, 3.9, and 3.9 mcg/mL, respectively, for lauric acid. EC50 were 30, not determined, and 60 mcg/mL for BPO, respectively, versus 6, 4, and 2 mcg/mL for lauric acid, respectively. Lauric acid was bactericidal to *P. acnes* at concentrations over 60 mcg/mL. In the *in vivo* portion, BALB/C mice ears were injected intradermally with 1 X 107 colony forming units (CFU) of *P. acnes*. After 24 hours, significant swelling was observed in the *P. acnes* injected ear. Inflamed ears were then treated with intradermal injections and epicutaneous applications of lauric acid. After 1 day ear inflammation thickness was significantly reduced ($P<0.05$), as were *P. acnes* CFU ($P<0.0005$). TUNEL assays (Terminal deoxynucleotidyl transferase dUTP nick end labeling, a method that identifies DNA fragmentation that results from abnormal apoptosis or cellular DNA damage) reveal that lauric acid was not toxic to keratinocytes.[11]

Eight *in vitro* studies assessed the antimicrobial activity of lauric acid and monolaurin on a wide variety of microorganisms, and all are reviewed in chronological order below.[6,10,12–17] The first *in vitro* study evaluated bactericidal properties of 30 different fatty acids including lauric, capric, and caprylic acids and their derivatives against gram-negative organisms (*Proteus vulgaris*, *P. mirabilis*, *P. rettgeri*, *Escherichia coli*, *Serratia marcescens*, *Pseudomonas aeruginosa*, and *Salmonella typhimurium*), gram-positive organisms (*S. aureus*, *S. epidermidis*, beta-hemolytic streptococci, group D streptococcus, *Bacillus subtilis*, *Sarcina lutea*, *Micrococcus sp.*, *Nocardia asteroids*, *Corynebacterium sp.*, and pneumococcus) and *Candida albicans*. The MIC was determined for each organism after an 18-hour incubation time on an agar plate. Lauric acid and capric acid were active against all gram-positive and gram-negative bacteria and *C. albicans*. Compared to glyceride derivatives, the free acid form of lauric acid had the highest bacteriostatic activity.

MIC ranges for lauric acid were 0.062–2.249 micromoles/mL, for capric acid were 1.45 and 5.8 micromoles/mL, and for caprylic acid were not inhibitory at concentrations tested.[12]

Next, an *in vitro* study evaluated fungicidal activity of *C. nucifera* fatty acids capric acid, lauric acid, and a variety of monoglycerides against *Candida albicans*. *C. albicans* was incubated with a suspension of each fatty acid. After a short inactivation time of 10 and 30 minutes and 2 and 5 hours, CFU of yeast were measured. The study determined that capric acid had the fastest killing of *C. albicans* with no colonies present after 10 minutes versus 3.5 $\log_{10}$ CFUs in the lauric acid sample. Higher concentrations of both lauric and capric acids were more effective at killing than lower concentrations. Following longer incubation times of 30 minutes and above, lauric acid had the most reliable killing compared to other agents with no detectable growth at 30 minutes, 2 hours or 5 hours.[13]

Another study evaluated the activity of 15 antimicrobial medications (including, but not limited to, ampicillin-sulbactam, vancomycin, oxacillin, levofloxacin) and 6 saturated fatty acids (lauric acid, stearic acid, octanoic acid, myristic acid, palmitic acid) against methicillin sensitive and 4 strains of methicillin resistant *S. aureus* (MSSA and MRSA, respectively). MICs for antibacterial drugs were determined following microbroth dilution. Time-kill curves were also determined, as were MICs in the presence of human plasma. Lauric acid inhibited growth of all strains of S. aureus, and had a lower MIC compared to other saturated fatty acids (400 mcg/mL for all strains versus 800–1,600 mcg/mL for other fatty acids). MICs were increased to 800 mcg/mL in the presence of 10% human plasma. All antimicrobial agents displayed much lower MICs than the fatty acids (on the order of ≤0.5-2 mcg/mL for most agents against MSSA, and ≤0.5–>16 mcg/mL for MRSA with arbekacin as the agent with the lowest MIC). Lauric acid showed a bacteriostatic effect at concentrations at and above the MIC, and was bactericidal at 6 hrs at concentrations 2 and 4 times the MIC.[14]

Ogbolu et al (2007) performed an *in vitro* study that focused on the antifungal properties of coconut oil compared to fluconazole, a first-line option for a variety of *Candida* yeasts. Fifty-two isolates obtained from vaginal, endocervical, urine, ear swab/discharge, and wounds were studied for their susceptibilities to VCO and fluconazole using an agar-well diffusion technique. Progressively dilute solutions of VCO or fluconazole were placed on an agar medium with yeasts. After 24-hours,

the sensitivity patterns were measured, and zone of inhibition diameter ≤27 mm were considered resistant. All *Candida* species were sensitive to 100% coconut oil vs 92% in fluconazole. It is important to note that some species such as *C. krusei* and *C. tropicalis* are known to be less susceptible to fluconazole, and this medicine is not recommended for these infections. *C. albicans* had the highest susceptibility to coconut oil, and the percent of *Candida* species sensitive to VCO was greater than the percent of species sensitive to fluconazole at most concentrations.[15]

An *in vitro* study of skin samples from infected atopic dermatitis and impetigo lesions from 100 newborn to 18-year-old patients evaluated sensitivity to monolaurin. Dermatoses were infected with gram-positive organisms and gram-negative organisms including *S. aureus*, coagulase negative *Staphylococcus*, *Streptococcus pyogenes*, *E. coli*, *Serratia marcescens*, *Klebsiella rhinosclermatis,* and others. Skin scrapings were incubated for 24 hours then added to a blood agar plate with monolaurin, penicillin, oxacillin, erythromycin, mupirocin, fusidic acid, or vancomycin. All *S. aureus*, coagulase-negative *Staphylococcus*, *Streptococcus spp*, *Enterobacter*, *Enterococcus*, and *E. vulneris* species were 100% sensitive to monolaurin; 100% sensitivity was not observed in any of the antibiotics. *K. rhinosclermatis* were less sensitive to monolaurin, but still showed 92.31% sensitivity to monolaurin compared to 0–7.69% sensitivity to other antibiotics ($P<0.05$ for each antibiotic).[6]

Another *in vitro* study by Yang et al compared the effectiveness of lauric acid, palmitic acid, and oleic acid against *P. acnes* on Brucella broth agar plates after 3 days incubation. Lauric acid began to kill *P. acnes* at concentrations above 50 mcg/mL and completely killed *P. acnes* at 80 mcg/mL. Growth was still noted at concentrations as high as 100 mcg/mL for both palmitic and oleic acids. To improve water solubility and potentially improve delivery of medicinal agents, lauric acid was loaded into liposomes. It was found that lauric acid–loaded liposomes could fuse with *P. acnes* bacterial membranes and were effective at delivering lauric acid. Complete killing of *P. acnes* by liposomal spheres was noted at concentrations above 51 mcg/mL.[10]

Coconut oil in water cream emulsions varying in concentrations from 5–40% were tested for *in vitro* antimicrobial activity against *C. albicans*, *Aspergillus niger*, *S. aureus*, and *Ps. aeruginosa*. Each cream was inoculated with a standardized culture of bacteria or yeast, and survival was measured at 6, 24, and 48 hrs as well as at 7,

14, and 28 days. Creams were compounded as preservative-free or with preservatives of lemon grass oil, parabens, or cetrimide. No growth of *S. aureus* was observed after 6 hrs for any of the creams, no growth of *Ps. aeruginosa* after 48 hrs, and none after 7 days for *Candida* or *A. niger*. The results of this study indicate that coconut oil could be formulated into a cream and maintain its antimicrobial activity on both fungus and bacteria.[16]

Lastly, Fischer et al (2012) examined the efficacy of different sphingoid bases and fatty acids against 4 gram-positive and 7 gram-negative bacteria typically found in the oral and epithelial microbiome, including *Fuscobacterium nucleatum*, *S. aureus*, *Streptococcus sanguinis*, *S. marcescens*, *Streptococcus mitis*, *E. coli*, *Ps. aeruginosa*, *Corynebacterium bovis*, *C. striatum*, and *C. jeikeium*. Bacterial cultures were added to dilute lipid suspensions in microtiter plates, and after incubation, the MIC and MBC were measured and evaluated. While all sphingoid bases were antimicrobial for gram-positive organisms (MIC range 0.3–13 mcg/mL), lauric acid was the only fatty acid that displayed antibacterial activity against *C. bovis*, *C. striatum*, and *C. jeikeium*. Lauric acid did not have any activity against *E. coli*, *Ps. aeruginosa*, or *S. marcescens* (MBC >500 mcg/mL) in this study.[17] This study had different results toward gram-negative bacteria than previous studies, and this may be due to difference in study methodology (direct inoculation of bacteria followed by incubation with fatty acids on an agar plate versus addition of bacteria to a dilute fatty acid suspension followed by incubation on microtiter plates).

Adverse Effects

When applied topically, coconut oil has a very low risk of allergic reaction or adverse effects. However IgE binding proteins are present, and allergic reactions have been described in a small number of patients, as has localized pruritus.[1,18–20] Lightening of skin tone may occur.[21] Avoid topical use of coconut oil if a known allergy or hypersensitivity exists to coconut, coconut oil, or any member of the *Arecaceae* family. Systemic absorption is low for coconut oil; however, when administered orally, hypotension and hyperlipidemia have been noted, as have reductions in serum lipids.[22,23] Lauric acid is known to be excreted in breast milk and may induce allergies in infants.[20] There are no known drug or food interactions with coconut oil when applied topically; however, antihypertensive

and antihyperlipidemic medications may be affected if administered orally.[1,8,24] Topical coconut oil has been studied in children, the elderly, and pregnant and lactating women; adverse effects are similarly rare in all groups.[1]

Conclusions

Cultures across the globe have used the *Cocos nucifera L.* plant for many generations. Constituents of coconut oil, predominantly lauric acid, have *in vitro* and *in vivo* evidence for killing a wide variety of gram-positive and gram-negative bacteria and *Candida* species. Though lauric acid has a lower MIC compared to other fatty acids, it does not achieve the same bacteriostatic or bactericidal potential as commercially available antibiotics. Coconut oil can be prepared in emulsions and liposomes and retain anti-infective properties. Given the low side effect burden, it may be a reasonable option for patients with mild to moderate dermal infections, especially acne vulgaris caused by *P. acnes*, polymicrobial atopic dermatis, impetigo, or wound infections. Additional randomized controlled trials are needed to solidify the place in therapy of *C. nucifera* as a treatment of dermal infections.

Table 1: Summary of in vitro and in vivo studies of the antimicrobial properties of coconut oil

Reference	Study Design	Study Overview	Study Results

Verallo-Rowell, 2008	DB, RCT	VCO was compared to VOO in removing colonized S. aureus from 26 patients aged 18 to 40 with mild to moderate high scores on SCORAD O-SSI atopic dermatitis. Patients were administered 5 mL of either VCO or VOO twice daily.	Of patients who were originally colonized with S. aureus, only 1 patient treated with VCO remained colonized after 4 weeks vs 6 patients treated with VOO (RR=0.10, 95% CI: 0.01–0.73, P=0.028). Post-intervention O-SSI scores in the VCO group were significantly lower than the VOO (mean difference -4.1, P=0.004). No ADEs to VOO or VCO were reported.

Nakatsuii, 2009	Mixed: In vivo and in vitro	In vitro portion: S. aureus, S. epidermidis, and P. acnes were co-cultured with either BPO or lauric acid on agar plates. In vivo portion: BALB/C mouse ear were injected intradermally with to P. acnes. Inflamed ears were then treated intradermally and epicutaneous with lauric acid.	MIC for S. aureus, S. epidermidis, and P. acnes against each organism for BPO were 15.6, >100, and 62.5 mcg/mL, respectively, versus 0.9, 3.9, and 3.9 mcg/mL, respectively, for LA. After 1 day ear inflammation thickness was significantly reduced ($P<0.05$), as were P. acnes CFU ($P<0.0005$), and TUNEL assays reveal that lauric acid was not toxic to keratinocytes.

Kabara, 1972	In vitro	The bactericidal properties of 30 FAs including LA and CA and their derivatives were studied against gram-negative and gram-positive bacteria and C. albicans. MICs were determined after an 18-hour incubation on an agar plate.	LA and CA were active against all gram-positive and gram-negative organisms and Candida. MIC ranges for lauric acid were 0.062–2.249 micromoles/mL, and were 1.45 and 5.8 micromoles/mL for capric acid.
Bergusson, 2001	In vitro	The susceptibility of C. albicans to CA and LA were evaluated particularly following 10 min, 30 min, 2 hr, and 5 hr incubation periods.	CA was found to have to fastest killing of C. albicans, and LA had the most reliable killing at times greater than 30 min.

Kitahara, 2004	In vitro	The MIC of saturated FAs, including LA, was determined against 6 strains of MRSA and MSSA using a microbroth dilution and compared to a variety of antibiotics.	Of the saturated FAs examined, LA was the most effective against strains of S. aureus with a MIC of 400 μg/mL vs 800–1,600 μg/mL for other FAs. LA was not as effective compared to antibiotic agents that had MICs as low as 0.5 μg/mL.

Ogbolu, 2007	In vitro	52 isolates of Candida species obtained from vaginal, oral, wound, and ears were studied for their susceptibilities to VCO and fluconazole by using the agar-well diffusion technique.	100% of Candida species were susceptible to VCO, including Candida species known to have inherent resistance to fluconazole. C. albicans had the highest susceptibility to VCO and VCO consistently killed more species than fluconazole.

Carpo, 2007	In vitro	Skins samples for 100 pediatric patients, newborn to 18 years old, with infected impetigo or actopic dermatitis dermatoses were tested for sensitivity against monolaurin, and a variety of antibiotics. Infections were polymicrobial.	All the present gram-positive organisms were 100% susceptible to monolaurin, as were Enterobacter spp., E. vulneris, and Enterococcus spp. K. rhinosclermatis was less sensitive to monolaurin, at 92.31%, but antibiotic sensitivities ranged from 0–7.69% (P<0.05 for each antibiotic)

Yang, 2009	In vitro	LA, palmitic acid, and oleic acid were incubated with P. acnes then diluted and grown on agar plates for 3 days. After 3 days, CFUs of P. acnes were quantified. LA was also formulated into liposomes in an effort to increase delivery of LA, and then evaluated for antimicrobial activity.	LA completely killed P. acnes at 80 mcg/mL, and this was not mimicked by palmitic and oleic acids. LA loaded liposomes fused with P. acnes, were effective at delivering lauric acid, and completely killed at concentrations above 51 mcg/mL
Oyi, 2010	In vitro	VCO in water emulsion creams with either no preservative or preservative were evaluated for antimicrobial activity against C. albicans, A. niger, S. aureus, and Ps. aeruginosa.	Regardless of preservative status VCO in water emulsions killed S. aureus was by 6 hrs, Ps. aeruginosa by 48 hrs, Candida or A. niger by 7 days.

Fischer, 2012	In vitro	The MIC and the MBC of sphingoid bases, including LA, were evaluated from antimicrobial assays measured the susceptibility of 4 gram-negative and 7 gram-positive bacteria.	All FAs were antimicrobial for gram-positive organisms (MIC range 0.3–13 mcg/mL), and LA was the only FAs that was antibacterial to C. bovis, C. striatum, and C. jeikeium. Lauric acid did not have any activity against E. coli, Ps. aeruginosa, or S. marcescens (MBC >500 mcg/mL) in this study

Abbreviations: CA: capric acid, CFU: colony forming units, DB: double blind, FA: fatty acid, LA: lauric acid, MBC: minimum bactericidal concentration, MIC: minimum inhibitory concentration, MRSA: methicillin resistant Staphylococcus aureus, MSSA: methicillin sensitive Staphylococcus aureus, RCT: Randomized controlled trial, spp: species, VCO: virgin coconut oil, VOO: virgin olive oil

Categorized Under

PEER-REVIEWED ARTICLES (/PEER-REVIEWED)

ABOUT THE AUTHOR

Lindsey K. Elmore

(/author/lindsey-k-elmore)

PharmD, BCPS

(/author/samantha-singleton)

ABOUT THE AUTHOR

Samantha Singleton (/author/samantha-singleton)

(/author/luke-lorenz)

ABOUT THE AUTHOR

Luke Lorenz (/author/luke-lorenz)

(/author/gwen-nance)

ABOUT THE AUTHOR

Gwen Nance (/author/gwen-nance)

References

1. Coconut (Cocus nucifera). In: Natural Standard. Somerville, (MA): Natural Standard. http://www.naturalstandard.com.ezproxy.samford.edu/databases/herbssupplements/coconutoil.asp? (http://www.naturalstandard.com.ezproxy.samford.edu/databases/herbssupplements/coconutoil.asp?). Accessed 12/08/13.
2. DebMandal M, Mandal S. Coconut (cocos nucifera l.: arecaceae): in health promotion and disease prevention. *Asian Pac J Trop Med*. 2011;4(3):241-247.
3. Parfene G, Hornicar V, Tyagi AK, Malik A, Bahrim G. Production of medium chain saturated fatty acids with enhanced antimicrobial activity from crude coconut fat by solid state cultivation of yarrowia lipolytica. *Food Chem*. 2013;136(3-4):1345-1349.
4. Antibiotic Resistance Questions and Answers. http://www.cdc.gov/getsmart/antibiotic-use/antibiotic-resistance-faqs.html#e (http://www.cdc.gov/getsmart/antibiotic-use/antibiotic-resistance-faqs.html#e). Accessed 11/25/13.
5. Mullin E. CDC director warns antibiotic pipeline 'nearly empty'. http://www.fiercebiotech.com/story/cdc-director-warns-antibiotics-pipeline-nearly-empty/2013-09-16 (http://www.fiercebiotech.com/story/cdc-director-warns-antibiotics-pipeline-nearly-empty/2013-09-16). Accessed 11/25/13.
6. Carpo BG, Verallo-Rowell VM, Kabara J. Novel antibacterial activity of monolaurin compared with conventional antibiotics against organisms from skin infections: an in vitro study. *J Drugs Dermatol*. 2007;6(10):991-998.
7. Preuss HG, Echard B, Dadgar A, et al. Effects of essential oils and monolaurin on staphylococcus aureus: in vitro and in vivo studies. *Toxicology Mechanisms and Methods*. 2005;15:279-285.
8. Verallo-Rowell VM, Dillague KM, Syah-Tjundawan BS. Novel antibacterial and emollient effects of coconut and virgin olive oils in adult atopic dermatitis. *Dermatitis*. 2008;19(6):308-315.
9. Simonart T. Newer approaches to the treatment of acne vulgaris. *Am J Clin Dermatol*. 2012;13(6):357-364.
10. Yang D, Pornpattananangkul D, Nakatsuji T, et al. The antimicrobial activity of liposomal lauric acids against propionibacterium acnes. *Biomaterials*. 2009;30(30):6035-6040 (tel:6035-6040).
11. Nakatsuji T, Kao MC, Fang JY, et al. Antimicrobial property of lauric acid against propionibacterium acnes: its therapeutic potential for inflammatory acne vulgaris. *J Invest Dermatol*. 2009;129(10):2480-2488 (tel:2480-2488).
12. Kabara JJ, Swieczkowski DM, Conley AJ, Truant JP. Fatty acids and derivatives as antimicrobial agents. *Antimicrob Agents Chemother*. 1972;2(1):23-28.
13. Bergsson G, Arnfinnsson J, Steingrimsson O, Thormar H. In vitro killing of candida albicans by fatty acids and monoglycerides. *Antimicrob Agents Chemother*. 2001;45(11):3209-

3212 (tel:3209-3212).
14. Kitahara T, Koyama N, Matsuda J, et al. Antimicrobial activity of saturated fatty acids and fatty amines against methicillin-resistant staphylococcus aureus. *Biol Pharm Bull*. 2004;27(9):1321-1326.
15. Ogbolu DO, Oni AA, Daini OA, Oloko AP. In vitro antimicrobial properties of coconut oil on candida species in Ibadan, Nigeria. *J Med Food*. 2007;10(2):384-387.
16. Oyi AR, Onaolapo JA, Obi RC. Formulation and antimicrobial studies of coconut (cocos nucifera linne) oil. *Res J App Sci, Eng, Technol*. 2010;2(2):133-137.
17. Fischer CL, Drake DR, Dawson DV, Blanchette DR, Brogden KA, Wertz PW. Antibacterial activity of sphingoid bases and fatty acids against Gram-positive and Gram-negative bacteria. *Antimicrob Agents Chemother*. 2012;56(3):1157-1161.
18. Teuber SS, Peterson WR. Systemic allergic reaction to coconut (Cocos nucifera) in 2 subjects with hypersensitivity to tree nut and demonstration of cross-reactivity to legumin-like seed storage proteins: new coconut and walnut food allergens. *J Allergy Clin Immunol*. 1999;103(6):1180-1185.
19. Rosado A, Fernandez-Rivas M, Gonzalez-Mancebo E, Leon F, Campos C, and Tejedor MA. Anaphylaxis to coconut. *Allergy*. 2002;57(2):182-183.
20. Couturier P, Basset-Stheme D, Navette N, and Sainte-Laudy J. A case of coconut oil allergy in an infant: responsibility of "maternalized" infant formulas. *Allerg Immunol* (Paris). 1994;26(10):386-387.
21. Agero AL, Verallo-Rowell VM. A randomized double-blind controlled trial comparing extra virgin coconut oil with mineral oil as a moisturizer for mild to moderate xerosis. *Dermatitis*. 2004;15(3):109-116.
22. Prior IA, Davidson F, Salmond CE, and Czochanska Z. Cholesterol, coconuts, and diet on Polynesian atolls: a natural experiment: the Pukapuka and Tokelau island studies. *Am J Clin Nutr*. 1981;34(8):1552-1561.
23. Mendis S. and Kumarasunderam R. The effect of daily consumption of coconut fat and soya-bean fat on plasma lipids and lipoproteins of young normolipidaemic men. *Br J Nutr*. 1990;63(3):547-552.
24. Mumcuoglu KY, Miller J, Zamir C, Zentner G, Helbin V, Ingber A. The in vivo pediculicidal efficacy of a natural remedy. *Isr Med Assoc J*. 2002;4(10):790-793.