A Review of Monolaurin and Lauric Acid

Natural Virucidal and Bactericidal Agents

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Antibiotic resistance has become a worldwide problem for treating infections caused by numerous organisms. Safe, effective antimicrobials that are not easily subject to resistance are desperately needed. The Food and Drug Administration (FDA) states on its website: "Disease-causing microbes that have become resistant to drug therapy are an increasing public health problem. Tuberculosis, gonorrhea, malaria, and childhood ear infections are just a few of the diseases that have become hard to treat with antibiotic drugs."[1] The FDA website also notes:

- Though food-producing animals are given antibiotic drugs for important therapeutic, disease prevention, or production reasons, these drugs can cause microbes to become resistant to drugs used to treat human illness, ultimately making some human sicknesses harder to treat.
- About 70 percent of bacteria that cause infections in hospitals are resistant to at least one of the drugs most commonly used to treat infections.
- Some organisms are resistant to all approved antibiotics and must be treated with experimental and potentially toxic drugs.
- Some research has shown that antibiotics are given to patients more often than guidelines set by federal and other health care organizations recommend. For example, patients sometimes ask their doctors for antibiotics for a cold, cough, or the flu, all of which are viral and do not respond to antibiotics. Also, patients who are prescribed antibiotics but do not take the full dosing regimen can contribute to resistance.
- Unless antibiotic resistance problems are detected as they emerge, and actions are taken to contain them, the world could be faced with previously treatable diseases that have again become untreatable, as in the days before antibiotics were developed.

Not only bacteria develop antibiotic resistance—viruses develop resistance as well. Accordingly, the need for safe, effective antivirals is also becoming paramount. For example, virucidal resistance has occurred in *Herpes simplex* virus type 1 as a result of acyclovir use.[2]

In a site-directed study of mutagenesis, researchers investigated the role of six mutations of *Herpes simplex* virus type 1 thymidine kinase (TK) gene in the acquisition of resistance to acyclovir (ACV).[2] TK activity was not impaired by substitutions located at codons 17, 161, and 374, and these mutations were thus related to TK gene polymorphism. Mutations His105Pro, Leu364Pro, and Asp162Ala lead to the loss of TK activity that could result in ACV-resistance. However, there are very limited antiviral treatments in conventional medicine against the vast majority of viruses that infect humans.

Monolaurin

Monolaurin, a monoester formed from lauric acid (medium-chain fatty acids), has profound antiviral and antibacterial activity.

Recognition of the antimicrobial activity of the monoglyceride of lauric acid (monolaurin) has been reported since 1966. A large body of the research can be credited to Jon J. Kabara, B.S., M.S., Ph.D., a professor emeritus of Michigan State University in East Lansing.[3] His early pioneer work focused on the virucidal effects of monolaurin (Lauricidin,® Med-Chem Laboratories, Inc., Galena, Illinois) on enveloped RNA and DNA viruses. This work continues to be investigated by numerous researchers because of the potential benefits related to food preservation.

A recent study that investigated the sanitizing effects of monolaurin in a laboratory scale system, partially reproduced dairy plant conditions. The study focused on the effectiveness of chlorine and alternative sanitizers for reducing the number of viable bacteria attached to stainless-steel surfaces.[4]

Bacteria-contaminated stainless-steel tubes in a continuous-flow reactor were exposed to a standard clean-in-place regimen (water rinse, 1 percent sodium hydroxide at 70ºC for 10 minutes, a second water rinse, 0.8 percent nitric acid at 70ºC for 10 minutes, and a third water rinse) followed by exposure to either chlorine (200 ppm) or combinations of nisin (500 ppm), Lauricidin (100 ppm), and the lactoperoxidase system (LPS) (200 ppm) for 10 minutes or 2, 4, 8, 18, or 24 hours.[4]

There was significant variation in the effectiveness of the alkaline-acid wash steps in reducing cell numbers (log reduction between 0 and 2). Following a 10-minute treatment, none of the

sanitizers significantly reduced the number of attached cells. Two (2) hours of exposure to chlorine, nisin + the LPS, or Lauricidin + the LPS achieved 2.8, 2.2, and 1.6 log reductions, respectively. Exposure times > 2 hours did not decrease the number of viable bacteria attached to the stainless steel further. The effectiveness of combinations of nisin, Lauricidin, and the LPS was similar to that of chlorine ($P > 0.05$); thus, according to these study results these sanitizers could be used to decontaminate the surfaces of small-volume or critical hard-to-clean milk-processing equipment.[4] (Additional studies of monolaurin's antibacterial effects are discussed in the section entitled Bactericidal Effects.)

Monolaurin Chemistry in the Human Body

Coconut oil and certain coconut products contain approximately 50 percent lauric acid and approximately 6–7 percent capric acid. The question arises frequently whether ingestion of lauric acid can lead to significant endogenous production of monolaurin. There do not appear to be any clear data concerning how much monolaurin is actually formed from lauric acid in the human body. Nevertheless, there is evidence that some is formed. Lauric acid is the main antiviral and antibacterial substance found in human breast milk.

Comparatively speaking, lauric acid (C12) has a greater antiviral and antibacterial activity than other medium-chain triglycerides such as caprylic acid (C8), capric acid (C10), or myristic acid (C14). Monolaurin is many times more biologically active than lauric acid in killing viruses and bacteria, leading to the interesting question concerning the conversion rate in the human body. Unlike these medium-chain fatty acids, diglycerides and triglycerides are inactive against microorganisms.[5]

Research has suggested that monolaurin exerts virucidal and bactericidal effects by solubilizing the lipids and phospholipids in the envelope of the pathogen causing the disintegration of its envelope. Recent evidence has also indicated that the antimicrobial effect is related to its interference with signal transduction in cell replication.[6]

Bactericidal Effects

Other studies, in addition to the abovementioned one with the milk-processing equipment, indicate that monolaurin has antibacterial activity. This monoester has been shown to be effective against both susceptible and antibiotic-resistant *Staphylococcus aureus*.[7]

Generally, monolaurin is more effective against gram-positive bacteria such as staphylococcus and streptococcus. In a study conducted by Preuss et al., monolaurin was shown to be bactericidal to *S. aureus* and *Mycobacterium terrae* but not *Escherichia coli* and *Klebsiella pneumoniae*, which are both gram-negative, confirming prior work on monolaurin, and was shown to be static to a variant of the virulent anthrax pathogen, *Bacillus anthracis* Sterne.[8]

Monolaurin also inhibits production of staphylococcal toxic shock toxin-1 effectively.[6] The monoester is effective against cytomegalovirus and the expression of virulence factors including protein A, alpha-hemolysin, B-lactamase, and the induction of vancomycin resistance in *Enterococcus faecalis*.[8–10] Monolaurin has also been shown to inactivate *Listeria monocytogenes, Streptococcus agalactiae,* and Groups A, F, and G *streptococci*.[10–12] (See Table 1.)

Table 1. Lipid-Coated Bacteria Inactivated by Monolaurin

- *Listeria monocytogenes*
- *Helicobacter pylori* (gram-negative)
- *Hemophilus influenzae* (gram-negative)
- *Staphylococcus aureus*
- *Streptococcus agalactiae*
- Groups A, B, F, and G streptococci
- Gram-positive organisms
- Gram-negative organisms if pretreated with a chelator

Sources: Refs. 14 and 17.

Unlike conventional antibiotics, monolaurin does not appear to have an adverse effect on gut probacteria.

Antifungal Effects

In addition, a number of fungi, yeasts, and protozoa are reported to be inactivated or killed by monolaurin. These fungi include several species of ringworm. *Candida albicans* and the protozoan parasite *Giardia lamblia* were both reported to be killed by monolaurin.[11–14]

Virucidal Effects

Some of the viruses inactivated to some extent by monolaurin include HIV, measles, *Herpes simplex*-1, vesicular stomatitis, visna virus, and cytomegalovirus.[14] Results of an extremely promising trial with HIV-positive male and female patients compared the effectiveness of monolaurin at two doses (2.4 g versus 7.2 g) with 50 mL of coconut oil in 15 patients.[15] The objective was to document if the patients' viral loads could be lowered by any of these treatments.

The patients were divided into 3 groups of 5 patients. They were seen daily with laboratory values determined at the beginning of the study and after 3 months and 6 months. At the onset of the trial the viral load of the patients measured by PCR, ranged from 1.96×10^3 to 1190.0×10^3 copies.

One (1) male had a viral count too low to measure and was not included in the final statistics. Seven (7) of the remaining 14 patients had reduced loads at 3 months (2 males and 5 females), and 8 patients of the 14 patients had reduced viral loads at 6 months.

However, the reduction was significant in only 3 patients (2 males and 1 female). Two (2) of these 3 patients were in the coconut-oil group and 1 was in the lower dose (2.4 g) monolaurin group.[15]

Significant funding is being sought in order to carry out a larger investigation. It is significant that several of the viruses inacti-

Table 2. Lipid-Coated Viruses Inactivated by Monolaurin

- Human immunodeficiency virus HIV-1, HIV+
- Measles virus
- *Herpes simplex* virus-1
- *Herpes simplex* virus-2
- *Herpes viridae* (all)
- Human lymphotropic viruses (type 1)
- Vesicular stomatitis virus
- Visna virus
- Cytomegalovirus
- Epstein-Barr virus
- Influenza virus
- Pneumonovirus
- Sarcoma virus
- Syncytial virus
- Rubeola virus

Sources: Refs. 14 and 17.

vated by monolaurin are responsible for opportunistic infections in HIV-positive individuals and may also be implicated in other illnesses such as chronic fatigue syndrome and immune dysfunction syndrome. (See Table 2.)

Loss of Lauric Acid from the American Diet

Over the past 40 years, the American diet has undergone drastic changes, few of which appear to be improving human health. Many of these changes have involved fats and oils. Humans have increased intake of partially hydrogenated vegetable oils and trans fatty acids as well as dramatically increasing intake of linoleic acid. These types of changes and many others have set the stage for a proinflammatory state in the human body. In contrast, there is very little intake of healthy fats, such as omega-3 fatty acids from certain fish and plants, as well as almost a total loss of antimicrobial fatty acids such as lauric acid from coconut oil that could produce monolaurin with all its described benefits.

Lauric acid has also been shown to be virucidal and bactericidal although monolaurin has even greater activity. Enteral feedings have been reformulated without coconut oil and formulas such as Ensure and Nutren are made with hydrogenated oils. Both humans and animals can metabolize some monolaurin from lauric acid.[14] However, it is worth repeating that the level of this metabolizing has not been quantified. Dr. Kabara has postulated that, if monolaurin is formed from lauric acid in coconut fat, the level is no greater than 3 percent.[16]

Table 3. Lauric Acid Content of Selected Foods

Food	Grams/cup
Coconut cream, raw	37.0
Coconut cream, canned	23.3
Fresh grated coconut, packed	19.4
Fresh grated coconut, loose	11.9
Granola cereal (with coconut oil and coconut)	6.05
Coconut cream pudding	1.29
Whole milk	0.23

Source: U.S. Department of Agriculture Handbook Nos. 8-1, (1976), 8-8 (1982), 8-12 (1984), and 8-19 (1991).

Mothers' milk, a rich source of lauric acid, may also provide a lipase that converts the triglycerides to monoglycerides by the infant. Even small amounts of monolaurin converted from lauric acid in coconut fat or mothers' milk and lauric acid are still virucidal and bactericidal. Coconut fat also provides caprylic acid, capric acid, and myristic acid, which are virucidal and bactericidal as well.[15]

In the past, infant formulas were good sources of lauric acid, because a greater amount of coconut oil was used. However, they have been reformulated with little or no coconut oil, thus losing their similarity to mothers' milk, which provides antimicrobial and antiviral fats. Human milk provides approximately 3.5 percent of calories as lauric acid for the human infant. Mature human milk has been noted to have up to 12 percent of the total fat as lauric acid (6.6 percent of calories).

Discussion

Unlike monolaurin, which has been patented as a product, lauric acid is not, and cannot be, patented. This clearly limits the funding available for research. It is hoped that the companies that manufacture coconut-based products will see the need to extend the research and start funding studies.

In the meantime, there is an excellent body of research demonstrating that monolaurin is a safe and effective virucidal and bactericidal agent. One of the authors of this review (SL) has used it

Table 4. Three Foods with Lauric Acid and Capric Acid (for Your Patients)

Brand	Origin	Lauric acid/capric acid
Jennies Macaroons[a]	United States	7.0 g per 2 oz
Grace Coconut Milk[b]	Sri Lanka	3.5 g per 2 oz or 0.4 g per 2 oz
Taste of Thai[c]	Thailand	3.5 g per 2 oz or 0.4 g per 2 oz

Source: Refs 14 and 17; [a]Red Mill Farms Inc., Brooklyn, NY.; [b]Grace, Kingston, Jamaica; [c]Andre Prost, Inc., Old Saybrook, CT.

for more than a decade with excellent results. Given its broad-spectrum activity, it has been used along with glycyrrhizin successfully to treat numerous cases of chronic fatigue syndrome, CMV infection, Epstein-Barr virus, *Herpes simplex*-1 and -2, fungal infections, and *C. albicans*—all common conditions that are seen in an integrative practice. SL has not personally used it for *G. lamblia*, which has become resistant to metronidazole and other antiparasite drugs.

Despite the unanswered question regarding whether substantial amounts of lauric acid are metabolized to monolaurin, it is important to note that lauric acid is also effective against, at least to some extent, the microorganisms listed in Tables 1 and 2. Lauric acid produces greater activity against microorganisms than caprylic acid, capric acid, or myristic acid, all of which are present in coconut oil. Given that coconut oil provides approximately 50 percent lauric acid, a substantial amount of this bactericidal and virucidal fatty acid can be obtained from consuming coconut fat in pure coconut oil, and in many foods and products. (See Tables 3 and 4; Table 4 suggests three coconut-based products.)

In addition, it is important to note that lauric acid appears to have immune-boosting properties as evidenced by feeding coconut oil to laboratory animals in whom the expected immune-factor responses (inhibition of interleukin-1) to endotoxin were induced via corn-oil feeding.[17] Ingesting monolaurin on a daily basis may be an inexpensive way to both treat and prevent infection from microorganisms.[18] This would be especially valuable, given that we are now living under a constant threat of bioterrorism.

The results of the small study examining the effects of monolaurin versus coconut oil were compelling.[15] It is a shame that a larger study has not been funded thus far. Despite the small number of patients in the study, both coconut oil and the lower dose of monolaurin (2.4 g) were both effective in significantly lowering the viral load for several patients. Perhaps a large amount of coconut oil may have a similar effect to a smaller dose of monolaurin. Because monolaurin is more bioactive than lauric acid, a smaller amount is needed for its antimicrobial effects.

Conclusions

Monolaurin has Generally Recognized As Safe (GRAS) status and is considered to be nontoxic. It is effective against many microorganisms and can be taken on a daily basis, given that evidence suggests it does not create antiviral or antibacterial resistance.

The general recommended adult dose of monolaurin is 1–3 g. Higher amounts can be used to achieve desired results if necessary. It is safe for children (ages 3–10) with the recommended dose being smaller—30 mg, one to three times per day. Monolaurin is available as minipellets that should be swallowed and not chewed.[19]

Coconut oil, coconut cream, grated coconut, and other products listed in Tables 3 and 4 are excellent sources of lauric acid and the other medium-chain fatty acids as well. Coconut oil provides mostly medium-chain triglycerides that are rapidly absorbed and transported to the mitochondria where they are utilized for fuel and may be less likely to be stored as body fat.[20]

Coconut oil does not have a deleterious effect on cholesterol or other blood lipids. In fact, it may raise high-density lipoprotein cholesterol. Coconut oil is rich in "good" saturated fatty acids that conserve the elongated omega-3 fatty acid. Animal studies have shown that some omega-3 fatty acids can be formed as a result of ingestion of coconut oil. Coconut oil does not contain trans fatty acids that have deleterious effects on blood lipids and insulin binding.[17]

Both monolaurin and coconut oil are excellent choices for combating a host of microorganisms—both therapeutically and preventively. More human studies are needed to elucidate the best therapeutic dose of monolaurin and coconut for addressing specific microorganisms and conditions.

It is also important to quantify how much monolaurin is metabolized from a specific quantity of coconut oil. Finally, it is equally important to compare large doses of coconut oil to specific doses of monolaurin for their antimicrobial action as was done in the HIV study population. Dietary supplements such as monolaurin may be unavailable to some populations while coconut oil may be a less-costly alternative. □

References

1. U.S. Food and Drug Administration Facts About Antibiotic Resistance. Online document at: www.fda.gov/oc/opacom/hottopics/antiresist_facts.html Accessed November 8, 2006.

2. Frobert E, Ooka T, Cortay JC, et al. Resistance of *Herpes simplex virus type* 1 to acyclovir: Thymidine kinase gene mutagenesis study. Antiviral Res 2006 Aug 30;epub ahead of print.

3. Kabara JJ. Fatty acids and derivatives as antimicrobial agents—a review. In: Kabara JJ, ed. The Pharmacological Effects of Lipids. Champaign IL: American Oil Chemists Society,1978:75.

4. Dufour M, Simmonds RS, Bremer PJ. Development of a laboratory scale clean-in-place system to test the effectiveness of "natural" antimicrobials against dairy biofilms. Food Prot 2004;67:1438–1443.

5. Kabara JJ, Vrable R. Antimicrobial lipids: Natural and synthetic acids and monoglycerides. Lipids 1977;12:753–759.

6. Projan SJ, Brown-Skrobot S, Schlievert PM. Glycerol monolaurate inhibits the production of B-lactamase, toxic shock syndrome toxin-1 and other staphylococcal exoproteins by interfering with signal transduction. J Bacteriol 1994;176:4204–4209.

7. Preuss HG, Echard B, Dadgar A, et al. Effects of essential oils and monolaurin on *Staphylococcus aureus*: In vitro and in vivo studies. Toxicol Mech Method 2005;15:279–285.

8. Preuss HG, Echard B, Enig M, et al. Minimum inhibitory concentrations of herbal essential oils and monolaurin for gram-positive and gram-negative bacteria. Mol Cell Biochem 2005;272:29–34.

9. Ruzin A, Novick RP. Equivalence of lauric acid and glycerol monolaurate as inhibitors of signal transduction in *Staphylococcus aureus*. J Bacteriol 2000;182:2668–2671.

10. Ruzin A, Novick RP. Glycerol monolaurate inhibits induction of vancomycin resistance in *Enterococcus faecalis*. J Bacteriol 1998;180:182–185.

11. Boddie RL, Nickerson SC. Evaluation of postmilking teat germicides containing Lauricidin® saturated fatty acids and lactic acid. J Dairy Sci 1992;75:1725–1730.

12. Isaacs CE, Thormar H. The role of milk-derived antimicrobial lipids as antiviral and antibacterial agents. In: Mestecky J, et al, eds. Immunology of Milk and the Neonate. New York: Plenum Press, 1991.

13. Isaacs CE, Kashyap S, Heird WC, et al. Antiviral and antibacterial lipids in human milk and infant formula feeds. Arch Dis Child 1990;65:861–864.

14. Enig M. Lauric oils as antimicrobial agents: Theory of effect, scientific rationale, and dietary application as adjunct nutritional support for HIV-infected individuals. In: Watson R, ed. Nutrients and Foods in AIDS. Boca Raton, FL: CRC Press, 1998.

15. Tayag E, Dayrit CS. Results analysis of clinical HIV trial with monoglycerides [presentation]. The 37th Cocotech Meeting, Chennai, India, July 25, 2000.
16. Kabara JJ. Pharmacological effects of coconut oil vs. monoglycerides. Inform June 2005;286.
17. Enig, M. Coconut: In support of good health in the 21st century [presentation]. Presented to the Federated States of Micronesia, Pohnpei, 1999.
18. Preuss HG, Echard B, Zonosi RR. The potential for developing natural antibiotics: Examining oregano and monolaurin. Original Internist 2005;12:119–124.
19. Dosing [based on information provided by Kabara JJ]. Lauricidin®: The Natural Way to Better Health Dosing information. Online document at: www.lauricidin.com/dosing.htm
20. Anonymous. Medium chain triglyceride—monograph. Alt Med Rev 2002;7:418–420.

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