Title: Monolaurin

Category: Chemical

SYNONYMS/COMMON NAMES/RELATED SUBSTANCES

Glycerin Monolaurate, Glycerol Monolaurate, Lauricidin, Lauric Acid Monoglyceride

Combination products:

HerbalcidinTM A blend of monolaurin, Glycyrrhizin, and two Phyllanthus species for those with viral, fungal or bacterial concerns.

Monolaurin 300 mg and 600mg (90 capsules) by Ecological Formulas contains monolaurin, calcium, and inosine for those concerned about viral infections, cold and flu.

NISIN CLEAN, an Anti-bacterial and Anti-viral Moisturizing Formula from “New Millennium Foods” is a unique preparation containing nisin and monolaurin.

CLINICAL BOTTOM LINE

The antiviral, antibacterial, and antifungal properties of lauric acid and monolaurin have been recognized for nearly three decades by only a small number of researchers: their work, however, has resulted in 100 or more research papers and numerous U.S. and foreign patents. Prof. Dr. Jon J. Kabara performed the original seminal research in this area of fat research. Kabara in1968 first patented certain fatty acids (FAs) and their derivatives (e.g., monoglycerides (MGs) that can have adverse effects on various microorganisms. While nontoxic and approved as a direct food additive by the FDA, monolaurin (Lauricidin®) adversely affects bacteria, yeast, fungi, protozoa, and envelope viruses.

Kabara1-24 found that the properties that determine the anti-infective action of lipids are related to their structure: e.g., free fatty acids & monoglycerides. While the monoglycerides are active; diglycerides and triglycerides (fats) are inactive. Of the saturated fatty acids, lauric acid has greater antiviral activity than caprylic acid (C-8), capric acid (C-10), or myristic acid (C-14).

Fatty acids and monoglycerides produce their killing/inactivating effects by several mechanisms. An early postulated mechanism was the perturbing of the plasma membrane lipid bilayer. The antiviral action attributed to monolaurin is that of fluidizing the structure in the envelope of the virus, causing the disintegration of the microbial membrane. More recent studies indicate that one antimicrobial effect in bacteria is related to monolaurin's interference with signal transduction/toxin formation (Projan et al 1994)46.

Another antimicrobial effect in viruses is due to lauric acid's interference with virus assembly and viral maturation (Homung et al 1994)31. The third mode of action may be on the immune system itself (Witcher et al, 1993)35.

Antiviral Effects

Hierholzer and Kabara (1982)37 first reported the antiviral activity of the monoglyceride of lauric

acid

(monolaurin) on viruses that affect humans. They showed virucidal effects of monolaurin on enveloped RNA and DNA viruses. This work was done at the Center for Disease Control of the U.S. Public Health Service. This study was carried out using selected virus prototypes or recognized representative strains of enveloped human viruses. All these viruses have a lipid membrane. The presence of a lipid membrane on viruses makes them especially vulnerable to lauric acid and its derivative monolaurin. These initial findings from the Center of Disease Control (CDC) have been confirmed by many other investigators.

Research has shown that enveloped viruses are inactivated by added fatty acids and monoglycerides in both human and bovine milk (Isaacs et al 1991)33. Others (Isaacs et al 198632, 199053, 199133, 199463; Thormar et al

198762) have confirmed Kabara's original statements concerning the effectiveness of monolaurin.

Some of the viruses inactivated by these lipids are the measles virus, herpes simplex virus (HSV-1 and -2), herpes family members (HIV, hepatitis C, vesicular, stomatitis virus (VSV), visna virus, and cytomegalovirus (CMV). Many of the pathogenic organisms reported to be inactivated by these antimicrobial lipids are those known to be responsible for opportunistic infections in HIV -positive individuals. For example, concurrent infection with cytomegalovirus is recognized as a serious complication for HIV positive individuals (Macallan et al 199364).

Thus, it would appear imperative to investigate the practical aspects and the potential benefit of a nutritional supplement such as monolaurin (Lauricidin®) for microbial infected individuals. Until now few nutritionists in mainstream nutrition community seem to have recognized the added benefit of antimicrobial lipids in the support of infected patients. These antimicrobial fatty acids and their derivatives are essentially nontoxic to man. According to the published research, lauric acid is one of the best "inactivating" fatty acids, and its

monoglyceride is even more effective than the fatty acid alone (Kabara 19788, Sands et al 197940, Fletcher et al 198519, Kabara 198521).

It should be emphasized that lauric acid cannot be taken orally because it is severally irritating. Lauricidin® on the other hand, a derivative of lauric acid chemically bonded to glycerin to form monolaurin, can be taken orally without any problem.

Antibacterial Effects

The potentially pathogenic bacteria inactivated by monolaurin include Listeria monocytogenes, Staphylococcus aureus, Streptococcus agalactiae, groups A, F and G streptococci, gram-positive organisms, and some gram- negative organisms if pretreated with a chelator (Boddie and Nickerson, 199236; Kabara, 19788, 198418; Isaacs et al., 199053, 199133, 199463; Isaacs and Schneidman, 199165; Isaacs and Thormar, 198632, 199152; Thormar et al., 198762; Wang and Johnson, 199234).

Decreased growth of Staphylococcus aureus and decreased production of toxic shock syndrome toxin-1 was shown with 150 mg monolaurin per litre (Holland et al., 199430). Monolaurin was shown to be 5,000 times more inhibitory against Listeria monocytogenes than is ethanol (Oh and Marshall, 199369). Helicobacter pylori was rapidly inactivated by medium-chain monoglycerides and lauric acid, and there appeared to be very little development of resistance of the organism to the bactericidal effects of these natural antimicrobials (Petschow et al., 199670).

A number of fungi, yeast, and protozoa are also inactivated or killed by monolaurin. The fungi include several species of ringworm (Isaacs et al 199133). The yeast reported to be affected is Candida albicans (Isaacs et al 199133). The protozoan parasite Giardia lamblia is killed by monoglycerides from hydrolyzed human milk (Hemell et al 198667, Reiner et al 198666, Crouch et al 199168, Isaacs et al 199133).

Chlamydia trachomatis is inactivated by monolaurin (Bergsson et al 199825). Hydrogels containing monocaprin/monolaurin are potent in vitro inactivators of sexually transmitted viruses such as HSV-2 and HIV- 1 and bacteria such as Neisserian gonorrhea (Thormar102-103).

Monolaurin does not appear to have an adverse effect on desirable gut bacteria, but rather on

only potentially pathogenic microorganisms. For example, Isaacs et al (199133) reported no inactivation of the common Esherichiacoli or Salmonella enteritidis by monolaurin, but major inactivation of Hemophilus influenza,

Staphylococcus epidermis and Group B gram positive streptococcus.

The phenomenal rate of prescriptions dispensed for antibiotic use, and to a lesser extent, antiviral has grown exponentially in the past several decades. Antibiotic has limited specificity and generally does not recognize “good” bacteria (often referred to as probiotics or forlife) from “bad” bacteria (meaning those bacteria that may cause disease.) Antibiotics try to destroy all bacteria and are usually unsuccessful.

More antibiotic therapy may start perpetuating a chronic illness. The cycle of antibiotic therapy may go on for months and months, and repetitious indiscriminate use of antibiotics destroys weak bacteria and sets up the stage for the more virulent bacteria to survive (as in survival of the fittest). The new, stronger, pathogenic bacteria are now “resistant” to the established antibiotic and another antibiotic must be found to fight the new pathogen. We are rapidly approaching that point in history of having super bacteria: disease causing bacteria that are unaffected by any antibiotic. In its failure, antibiotic therapy has taken with it the health of those same individuals it strives to help.

The great advantage of Lauricidin® is that it does not produce resistant microorganisms during use. Not only does Lauricidin® not produce resistance but also it is known to help resistant organisms from forming.

A group of researchers from the University of Minnesota led by P.M. Schlievert reported in the March 199294 issue of Antimicrobial Agents and Chemotherapy that monolaurin inhibited strains of strep and staph bacteria that cause toxic shock syndrome. Other researchers also studied effectiveness of monolaurin in this area.71-100 In similar research published in October 2007101, Filipino research wrote in the Journal of Drugs in Dermatology that "sensitivity rates of Gram-positive Staphylococcus aureus, Streptococcus spp., and coagulase negative Staphylococcus, Gram-negative E. vulneris, Enterobacter spp., and Enterococcus spp. to 20 mg/mL monolaurin was 100 percent."

Most recently, Schlievert and his Minnesota colleagues demonstrated that monolaurin prevented acute simian immunodeficiency virus infection in female rhesus monkeys. The monolaurin worked by decreasing the expression of SIV toxins and reducing inflammation of vaginal walls. As the researchers wrote in Nature on April 23, 2009, their findings linking "interfering with innate host responses that recruit the target cells necessary to establish systemic infection, opens a promising new avenue for the development of effective interventions to block HIV-1 mucosal transmission."

Research continues104-124 in measuring the effects of the monoglycerin, monoglyceride derivative of capric acid, monocaprin, as well as the effects of lauric acid. Chlamydia trachomatis is inactivated by lauric acid, capric acid and monocaprin (Bergsson et al., 199825). Hydrogels containing monocaprin are potent in vitro inactivators of sexually transmitted viruses such as HSV-2 and HIV-1 and bacteria such as Neisseria gonorrhoeae (Thormar102- 103).

Research suggests that monolaurin offers some degree of immune support for the **influenza virus** and also for the following viruses, including, cytomegalovirus, according to the article and description of studies on using monolaurin to destroy viruses at: Monolaurin – A Natural Immune Boosting Powerhouse, Friday, October 31, 2008 - Byron J. Richards, CCN:

In studies performed at the Respiratory Virology Branch, Centers for Disease Control, Monolaurin was shown to remove all measurable infectivity against the following RNA and DNA viruses
:

Viruses

HIV or HIV-1, -6 Visna virus

Herpes simplex virus-i (HSV-1 &2) Vesicular stomatitis virus (VSV)

Measles virus Rubella virus

Epstein-Barr virus (EBV) Respiratory syncytial virus Influenza virus Dengue virus (Type 1-4) Leukemia virus Cytomegalovirus (CMV)

Semliki forest virus Lymphocytic choriomeningitis

Human papilloma virus (HPV) Pneumovirus

Monolaurin has also been proven to deactivate the following in laboratory tests:

Bacteria

Gram-positive organisms Gram-negative organisms Bacillas anthracis (Anthrax) Chlamydia pneumonia Listeria monocytogenes Neisseria gonorrhoeae Staphylococcus aureus Helicobacter pylorus

Groups A, B, F & G streptococci Mycoplasma pneumonia Streptococcus agalactiae Vibrio parahaemolyticus Mycobacteria Clostridium perfringens

Yeasts, Fungi, and Molds Aspergillus Niger Malassezia, species Saccharomyces cerevisiae Penicillium citrinum Ringworm or tinea (Trichophyton) Candida utilis A number of protozoa like Giardia lamblia are also inactivated or killed by Lauricidin®

USES & EFFECTIVENESS:

Insufficient Evidence for:

The common cold, the flu (influenza), herpes, shingles, and other conditions

SIDE EFFECTS & SAFETY:

Monolaurin is non-toxic and appears on the U.S. Food and Drug Administration's list of substance generally

recognized as safe--GRAS--for use as a food additive. GRAS status signifies that a substance has not produced serious problems in humans when used in small or moderate amounts. Identified on labels as glycerol monolaurate, monolaurin acts as an emulsifier in ice cream and cosmetics.

FDA has not assessed monolaurin's safety and effectiveness for medical use.

All experts seem to agree that monolaurin is entirely safe. Animals have been fed huge amounts, up to 25 percent of their total diet for ten weeks, without any sign of harm (Kabara, 198416). However, no human or animal scientific tests have studied its effectiveness when used orally as an antiviral.

Lauricidin®, a nontoxic nutritional lipid might increase the die off of microorganisms and result in

a "Herxheimer Reaction," a kind of flu, acne, allergic symptom, as reported on the website of the Lauricidin® product. Present information on Lauricidin® suggests that it does not interfere with drug metabolism or other supplements.

Monolaurin is safe for most people when used in amounts commonly found in foods. It is not known if Monolaurin is safe when used in medicinal amounts. Monolaurin, given orally in doses of 600-1800 mg/day was found generally well tolerated. Gastrointestinal symptoms are rare. No significant abnormalities have been noted in liver enzymes, total leukocyte count, red cell count, hematocrit, hemoglobin, or platelet levels.

INTERACTIONS:

No information available for monolaurine interactions

DOSING/TOXICOLOGY:

The appropriate dose of monolaurin depends on several factors such as the user's age, health, and several other conditions. At this time there is not enough scientific information to determine an appropriate range of doses for monolaurin. Be sure to follow relevant directions on product labels and consult your pharmacist or physician or other healthcare professional before using.

Dosing [based on information provided by Kabara JJ]. Lauricidin®: The Natural Way to Better Health Dosing information can be found Online document at: www.lauricidin.com/dosing.htm

MECHANISM OF ACTION:

The way monolaurin kills bacteria is really interesting for those that are curious. All bacteria (and some viruses) have a very flimsy, lipid fat molecule envelopes (skin). This is by design so the bacterium and virus can easily penetrate its targets. As it so happens, monolaurin has the same size lipid fat molecule so it absorbs into the bacteria's skin.

Unfortunately for the bacteria, monolaurin doesn't have very good binding power. The skin envelopes, already loosely held together, begins to break apart. The cells burst open and die. Then the white blood cells takes the debris to the liver where it is eliminated from the body. Your body is much happier and it's a good deal for everyone - but the bacteria and viruses!

Monolaurin produces its killing/inactivating effects by several mechanisms: Disrupts the plasma membrane lipid bilayer of the virus preventing attachment to susceptible host cells. The antiviral action of monolaurin is also attributed to that of fluidizing of the structure in the envelope of the virus, which causes the disintegration of the membrane and prevents uncoating of the virus necessary for replication of the virus. An antimicrobial effect in bacteria of monolaurin is related to monolaurin's interference with signal transduction/toxin formation (Projan et al 199446). Monolaurin has also been shown to interfere with virus assembly and viral maturation (Hornung et al 199431). Another mode of action may be one that is of the immune system itself (Witcher et al, 199635). By disrupting the viruses, yeast and fungus activity so many ways makes it easy for the body's natural defenses to take action against the invaders. The body can easily manage the rest.

Monolaurin removed all measurable infectivity by disintegrating the viral envelope as evidenced by electron micrographs. By disrupting the conformation of the lipid bilayer (or the envelope), Monolaurin prevents viral attachment to susceptible host cells. Viruses that contain an envelope are termed enveloped , whereas those that lack an envelope are referred to as naked ."

The study noted that, "DNA viruses (including herpes viruses) have a loose, outer envelope, which contains lipids. RNA viruses are similar in that they have a double outer envelope or bilayer which contains essential lipids and glycoproteins structurally embedded in this

envelope."

The term virucidal, means to have virus-killing ability. The act of making fat or oil (lipids) soluble in water (or liquids) is called solubilization. That study noted, "Solubilization of the lipids and phospholipids in the envelope are key mechanisms by which Monolaurin's virucidal activity is expressed.

Monolaurin is called a virustatic agent. What virustatic agents do is "directly block the replication of viruses." The good thing is that monolaurin doesn't let you develop any type of drug resistance. So you can use it over and over again to fight flu viruses without a prescription.

What monolaurin actually does is to "potentiate immunological events initiated by other triggering agents (mitogens, antigens, phagocytic stimuli, and lymphokines)." It can be noted that Monolaurin may help one in his/her fight against viruses without harming the good bacteria his/her body uses.

In summary Monolaurin works as follows: Monolaruin destroys lipid-coated "bugs."

Monolaurin inactivates lipid-coated viruses: These viruses can coat themselves with lipids from your cell membranes.

Monolaurin works through cell lysis (breakdown and rupture of the cell):

Monolaurin causes the double layer of fats that envelope the virus (lipid bilayer) to break up. Monolaurin dissolves (solubilizes) the lipids and phospholipids in the lipid envelope of the virus.

Monolaurin interferes with the chemical communication systems of the virus:

Monolaurin interferes with signal transduction of the virus.

Monolaurin interferes with the growth and reproduction of the virus:

Monolaurin interferes with the assembly of new viruses that can spread and infect other of your cells.

Monolaurin interferes with the maturation of the virus.

HISTORY:

Shortly after arriving (1957) at University of Detroit (Dept. of Chemistry), Dr. Kobara began placing undergraduate students into research projects. In the summer of 1960 a young student working for Sister Mary Stanislause (Mercy College, Detroit) was studying the nutritional needs of a protozoa, single-celled organisms, called Tetrahymena Pyriformis. Because of Dr. Kobara's interest in the structure-function of lipids, Sister and he decided to examine the nutritional affects of fatty acids, which were readily available in pure form. The results of the summer experiments indicated that a fatty acid with a 12-carbon chain acid as opposed to the other shorter or longer fatty acids was toxic to this organism. While interesting, Dr. Kobara felt it necessary to repeat the experiments with another student the following year in 1961. The first experiment was confirmed but its significance was not obvious. The reports of the toxicity of hexachlorophene in nursery uses made Dr. Kobara reevaluate the potential uses of lipids as non-toxic antimicrobial agents. This began a retrospective examination of older literature. It was therefore no surprise to learn about lauric acid being reported to be toxic to a variety of microorganisms. Following their initial results and literature survey his students and he began an active program of measuring the modification of lipid structure on antimicrobial activity.

Over the years his students, colleagues, and he examined a wide variety of other lipids hoping to improve on nature. During this period they screened some 300 lipids and other comparable structures for antimicrobial activity. These seminal studies indicated that certain fatty acids and

monoglycerides (a single fatty acid attached to glycerol) obtained from mother's milk or lauric oils had extraordinary antimicrobial properties. They were not able to improve on nature and therefore returned to a certain monoglyceride derivative (monolaurin) obtained from coconut and palm kernel oils and indeed mother's milk. To have a clearer understanding of what a monoglyceride is the following graphic picture may be helpful.

Structurally a fat or triglyceride has a glycerol backbone bearing three fatty acid (R) "arms". These fatty acids occupy three positions of the glycerol molecule labeled sn1, sn2, or sn3. Diglycerides have two fatty acids attached while the fatty acid of a synthesized monoglyceride preferably occupies the only the sn1 position. The sn 3 position being similar to the sn 1 position

H2-C---OH sn1

H2-C----- OH sn2

H2-C---OH sn1 (3)

Glycerol + Lauric Acid ---> Monolaurin (Glycerol monolaurate, GML)

Kabara was the first to patent that certain fatty acids (MCFAs) (e.g., medium-chain- fatty acids) and their derivatives (e.g., monoglycerides (MGs)) can have adverse effects on various microorganisms.

REVIEW OF THE EVIDENCE:

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."41

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 treatinfections.

• 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.42

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.43 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.44

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.45

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.46

Bactericidal Effects

Other studies indicate that monolaurin has antibacterial activity. This monoester has been shown to be effective against both susceptible and antibiotic-resistant *Staphylococcus aureus*.47

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.48

Monolaurin also inhibits production of staphylococcal toxic shock toxin-1 effectively.46 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*.48–50
Monolaurin has also been shown to inactivate
Listeria monocytogenes, Streptococcus agalactiae,
and Groups A, F, and G
streptococci.
50–52 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.51–54

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.55 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 x 103 to 1190.0 x 103 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.55

It is significant that several of the viruses inactivated 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.

REVIEW OF THE EVIDENCE: (DISCUSSION)

Given Monolaurin's broadspectrum 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.

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 same microorganisms, at least to some extent. 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.

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.56-57 Ingesting monolaurin on a daily basis may be an inexpensive way to both treat and prevent infection from microorganisms.58 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

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.

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.59-60

Chronic Fatigue Syndrome (CFS) is a newly classified syndrome whose main characteristic is extreme lethargy and lassitude in combination with a variety of nonspecific symptoms such as swollen lymph nodes, recurrent sore throats, low grade fevers, joint and muscle pains, intestinal discomforts, depression, and other neuropsychiatric complaints. These symptoms can last for months or years. Chronic infection with the Epstein- Barr virus (EBV) is thought by many to be the etiology of CFS, but evidence suggests that viral infections by other viruses may also induce a post-viral fatigue state.

EBV is a member of the genetically-related herpes class of viruses, which includes Herpes simplex virus 1 and 2,cytomegalovirus and Varicella zoster virus. These viruses share the ability to establish latent infections by inserting their viral genome into human DNA. Although the latency is usually maintained in a symptom-free state, any disturbance of that balance or depression of the immune system will result in the activation of these viruses with a flaring of CFS symptoms. The inclusion of a natural non-toxic antiviral agent is of vital importance to the success of any treatment plan to overcome chronic low-grade viral infections such as in CFS. A large number of individuals have benefited from the use of the natural fatty acid complex, monolaurin.

FORMULARY: BRANDS USED IN CLINICAL TRIALS/THIRD PARTY TESTING:

Approved by the FDA, Lauricidin® is the only monolaurin supplement that has undergone clinical trials and laboratory testing. Its antiviral effect is due to monolaurin’s ability to disintegrate the lipid membrane of the virus and interfere with viral maturation. Some of the viruses inactivated by Lauricidin® are oral and genital herpes, Epstein Barr (chronic fatigue and mononucleosis), shingles, HIV, hepatitis C, cytomegalovirus, visna virus, measles, influenza, leukemia, viral pneumonia, vesicular-stomatitis, rubella, respiratory synctial virus, dengue virus, and lymphocytic choriomeningitis. Current research in Kenya with villagers suffering from HIV and other severe illnesses has shown dramatic improvements in the health of those given Lauricidin®.

Lauricidin® is a highly concentrated extremely pure extract of monolaurin which is a natural monoglyceride lipid (fat) that is a potent nontoxic antibacterial, antiviral and antifungal.

Monolaurin is also present in a healthy mother's breast milk, saw palmetto and bitter melon. Dr. Jon Kabara formulated this extraordinary supplement that is far stronger an infection fighter than coconut oil or any other substance containing monolaurin, yet does not harm healthy cells or beneficial microflora. Lauricidin® also helps to regulate bowel function, biochemically balances the body, and can be taken with other medications when necessary.

Lauricidin® inactivates a large number of harmful bacteria, including dangerous antibiotic-resistant staph infections such as MRSA (methicillin resistant staphylococcus aureus), enterococcus, and others. These superbugs continually mutate and become immune to even the most powerful drugs. MRSA infections are most common among those with weak immune systems and who are living in hospitals and nursing homes.

Infections usually appear around surgical wounds or invasive devices like catheters and feeding tubes. It usually presents on the skin as a rash or sore that won't heal but can spread throughout the body. Rates of MRSA infection are rapidly rising around the world, causing more than 60% of staph infections. Recently MRSA has been showing up in healthy people in the community with cases growing at an alarming rate. One study of children in south Texas found that there had been a 14 fold increase of MRSA cases between 1999 and 2001. In

2007 the CDC reported that 14% of all MRSA cases were acquired outside of health care facilities. Unlike antibiotics, harmful microbes do not become resistant to Lauricidin®.

Other strains of bacteria that are inactivated by Lauricidin® are the gram-positive bacteria including bacillus anthracis (Anthrax), Streptococcus agalactiae, clostridium perfringens, listeria monocytogenes, groups A, B, F

& G streptococci, and mycobacteria. Lauricidin® inactivates some gram-negative bacteria including Chlamydia trachomatis, helicobacter pylorus (stomach ulcers), vibrio parahaemolyticus, mycoplasma pneumonia, neisseria gonorrhea, and salmonella typhimurium (responsible for infecting those who ate contaminated peanuts).

Lauricidin® is also effective against a wide variety of harmful yeasts, fungi and molds including candida utilis and candida albicans, toenail fungus, aspergillus niger (black mold), penicillum citrinum, and several species of ringworm. Lauricidin will also kill a number of protozoa like

giardia, and has been highly effective with

Lyme's Disease as well. Further studies are underway at the Jon and Betty Kabara Cancer Institute at

Gundersen Hospital in La Crosse, Wisconsin and the research is promising.

Lauricidin® is safe for all ages and is highly recommended as a daily supplement to help enhance the immune system, prevent disease, and achieve optimal health. Dosage should be increased gradually to ease detox symptoms from toxins released by the dead pathogens that are eliminated.

NISIN CLEAN, an Anti-bacterial and Anti-viral Moisturizing Formula from "New Millennium Foods" is a unique preparation containing nisin and monolaurin.

Nisin is claimed to be effective against the following pathogenic bacteria as well as many others:

- Staphyloccus aureus (S. aureus)
- Streptococcus agalactiae(S.agalatiae)
- Escherichia coli (E. coli)
- Streptococcus uberis (S. uberis)

- Klebsiella pneumoniae (K. pneumoniae)

Cardiovascular Research Ltd. (Arteria, Inc.) in Concord, California, a well-regarded health food company, distributes products containing monolaurin. Most of their business is through physicians, but they also distribute 300 mg capsules of monolaurin in health-food stores, under the "Arteria" or "Ecological Formulas" label.

BIBLIOGRAPHY:

1] Kabara. J.J, Swieczkowski, D M. Conley. A J and Truant, J P Fatty Acids and Derivatives as Antimicrobial Agents Antimicrobial Agents and Chemotherapy 2(l):23-28 (1972)

2] Kabara. J.J.. Conley. A J.- Swieczkowski. D M. Ismail, I.A . Lie Ken Jie and Gunstone, F D Antimicrobial Action of Isomeric Fatty Acids on Group A Streptococcus J. Med Chem 16:1060-1063 (1973)

3] Conley. A J and Kabara. J.J. Antimicrobial Action of Esters of Polyhydric Alcohols. Antimicrob. Ag and Chemother 4:501-506 (1973)

4] Kabara, J J and Conley. A J A Non-Caloric Role for MCT and Other Lipids In: Mittelkettige Trigiyceride (MCT) in der Diat. Zur Zeitschrih Fur Ernahrungswissenschah Supplenta No 17. H. Kaunitz K Lang and W Fekl, eds pp 17-26 (1974)

5] Kabara. J.J. Lipids as Safe and Effective Antimicrobial Agents for Cosmetics and Pharmaceuticals. Cosmetics and Perfumery 90:21-25 (1975)

6] Kabara, J.J. Monolaurin as an Antimicrobial Agent. U.S. Patent Number 4,002,775. Med-Chem Laboratories, January 1977

7] Kabara. J.J., Vrable, R. and Lie Ken Jie, M.S.F Antimicrobial Lipids: Natural and Synthetic Fatty Acids and Monoglycerides. Lipids 12:753759 (1977)

8] Kabara, J.J. Synergistic Microbiocidal Composition and Method. U.S. Patent Number 4,067,997. January 10, 1978

9] Kabara, JJ Fatty Acids and Derivatives as Antimicrobial Agents-A Review. In: The Pharmacological Effect of Lipids. Jon J. Kabara, ed Champaign, Illinois: The American Oil Chemists' Society (1979),pp. 1-14

10] Li, C.Y. and Kabara. J.J. Effects of Lauricidin-on Fornes Annosus and Phellinus Weirii. In The Pharmacological Effect of Lipids. Jon J. Kabara, ed. Chamaign, Illionis: The American Oil Chemists' Society (1979). pp. 45-50.

11] Kabara, J.J. Toxicological, Bactericidal and Fungicidal Properties of Fatty Acids and Some Derivatives JAOCS 56:760-767

12] Kabara, J.J., editor The Pharmacological Effect of Lipids I . American Oil Chemists' Society: Champaign, Illinois (1979)

13] Kabara. J.J. Lipids as Host Resistance Factors of Human Milk Nutrition Reviews 38:65-73 (1980)

14] Kabara, J.J. Antimicrobial Compositions. U.S. Patent Number 4,189,481 February 19 1980

15] Chipley. J R . Story. L.D.. Todd, P,T. and Kabara, J.J. Inhibition of Aspergillus Growth and Extracellular Aflatoxin Accumulation by Sorbic Acid and Derivatives of Fatty acids. J. Food Safety 3:109-119 (1981)

16] Kabara, J.J. Medium-Chain Fatty Acids and Esters as Antimicrobial Agents. In: Cosmetic and Drug Presentation: Principles and Practice, Jon J. Kabara, ed., New York: Marcel Dekker, Inc., pp. 275-304 (1984)

17] Hierholzer, J.C. and Kabara, J.J. In Vitro Effects of Monolaurin Compounds on Enveloped RNA and DNA Viruses. J. Of Food Safety 4:1-12 (1982)

18] Kabara. J.J. Antimicrobial Agents Derived From Fatty Acids. J. American Oil Chemists' Society 61:397-403 (1984)

19] Fletcher, R.D., Albers, A.C., Albertson, J.N. Jr. and Kabara, J.J. Effect of Monoglycerides on Mycoplasma pneumoniae Growth. In: The Pharmacological Effect of Lipids 11, Jon J. Kabara, ed. American Oil Chemists' Society: Champaign, Illinois, pp. 59-63 (1985

20] Chipley, J.R., Todd, P.T., Atchley, F. and Kabara, J.J. Effects of Fatty Acid Derivatives on the Release of Extracelluiar Enzymes from Bacteria. In: The Pharmacological Effect of Lipids 11, Jon J. Kabara, ed. American Oil Chemists' Society: Champaign, Illinois, pp. 97-102 (1985)

21] Kabara, J.J. Ohkawa, M., Ikekawa, T., Katori, T. and Nishikawa, Y. Examinations on Antitumor Immunological, and Plant-Growth Inhibitory Effects of Monoglycerides of Caprylic, Capric, and Lauric Acids and Related Compounds. In: The Pharmacological Effect of Lipids 11, Jon J. Kabara, ed. American Oil Chemists' Society: Champaign, Illinois, pp. 263-272 (1985). Kabara, J.J., Editor. The Pharmacological Effects of Lipids II, American Oil Chemists' Society: Champaign, Illinois (1985)

22] Flournoy, D.J. and Kabara, J.J. The Role of Lauricidin® as an Antimicrobial Agent. In: Drugs of Today 21(8):373-377 (1985)

23] Kabara, J.J., Editor. The Pharmacological Effects of Lipids III, American Oil Chemists' Society: Champaign, Illinois (1989)

24] Kabara, Jon J, Pharmacological effects of coconut oil vs. monoglycerides Inform June 2005 pp386

25] Bergsson G, Amfinnsson J, Karlsson SN'rSteingrinisson 0, Thormar H. In vitro inactivation of Chlamydia trachomatis by fatty acids and monoglycerides. Antimicrobial Agents and Chemotherapy 1998;42:2290-2294

26] Crouch AA, Seow WK, Whitman LM, Thong YH. Effect of human milk and infant milk formulae on adherence of Giardia intestinalis.; Transactions of the Royal Society of Tropical Medicine and Hygiene 1991;85:617-619.

27] Dodge JA and Sagher FA. Antiviral and antibacterial lipids in human milk and infant formula. Archives of Disease in Childhood 1991;66:272-273.

28] Enig, MG. Lauric oils as antimicrobial agents: theory of effect, scientific rationale, and dietary applications as adjunct nutritional support for HIV-infected individuals. in Nutrients and Foods in AIDS (RR Watson, ed) CRC Press, Boca Raton, 1998, pp. 81-97.

29] Epstein SE, Speir E, Zhou YF, Guetta E, Leon M, Finkel T. The role of infection in restenosis and atherosclerosis: focus on cytomegalovirus. Lancet 19%;348 Supplement 1:513-17.

30] Holland KT, Taylor D, Farrell AM. The effect of glycerol monolaurate on growth of, and production of toxic shock syndrome toxin-1 and lipase by, Staphylococcus aureus. Journal of Anti-microbial Chemotherapy 1994;33:41-55.

31] Homung B, Arntmann E, Sauer G. Lauric acid inhibits the maturation of vesicular storriatitis virus. Journal of General Virology 1994;75:353-361.

32] Isaacs CE, Thormar H. Membrane-disruptive effect of human milk: inactivation of enveloped viruses. Journal of Infectious Diseases 1986; 154:966-971.

33] Isaacs CE, Schneidman K. Enveloped Viruses in Human and Bovine Milk are Inactivated by Added Fatty Acids (FAs) and Monoglycerides (MGs). FASEB Journal 1991;5: Abstract 5325, p. A1288.

34] Wang LL and Johnson EA. Inhibition of Listeria monocytogenes by fatty acids and monoglycerides. Applied and Environmental Microbiology 1992;58:624-629.

35] Witcher KJ, Novick RP, Schlievert PM. Modulation of immune cell proliferation by glycerol monolaurate. Clinical and Diagnostic Laboratory Immunology 1996;3:10-13.

36] Boddie, RL, Nickerson, SE, Evaluation of postmilking teat germicides containing Lauricidin, saturated fatty acids, and lactic acid **.** Journal of Dairy Science, 75: 6: 1725-30, 1992.

37] Hierholzer, JC, Kabara, JJ, In vitro effects of monolaurin compounds on enveloped RNA and DNA viruses, Journal of Food Safety, 4:1, 1982.

38] Ismail-Cassim, N. et al, Inhibition of the uncoating of bovine enterovirus by short chain fatty acids, Journal of General Virology, 71: 10: 22 83-9, 1990.

39] Karbara, Lipids as host-resistance factors in human milk**,** Nutrition Reviews 38:65, 1980.

40] Sands, J, et al. Extreme sensitivity of enveloped viruses, including Herpes Simplex, to long

chain unsaturated monoglycerides and alcohols **,** Antimicrobial Agents and Chemotherapy. 15; 1:67-73, 1979.

41] 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.

42] 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.

43] 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.

44] 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.

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

46] 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.

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

48] Preuss HG, Echard B, Enig M, et al. Minimum inhibitory concentrations of herbal essential oils and monolaurin for gram-positive and gramnegative bacteria. Mol Cell Biochem

2005;272:29–34.

49] 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.

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

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

52] 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.

53] 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.

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

55] Tayag E, Dayrit CS. Results analysis of clinical HIV trial with monoglycerides [presentation]. The 37th Cocotech Meeting, Chennai, India, July 25, 2000.

56] Kabara JJ. Pharmacological effects of coconut oil vs. monoglycerides. Inform June 2005;286.

57] Enig, M. Coconut: In support of good health in the 21st century [presentation]. Presented to the Federated States of Micronesia, Pohnpei, 1999.7

58] Preuss HG, Echard B, Zonosi RR. The potential for developing natural antibiotics: Examining oregano and monolaurin. Original Internist 2005;12:119–124.

59] 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

60] Anonymous. Medium chain triglyceride—monograph. Alt Med Rev2002;7:418–420.

61] Erna Rafaela L. Abraham, M.D., Vermen M. Verallo-Rowell, M.D. and Benilda Q. Baello, R.M.T. Testing of Lauricidin Versus Isopropyl Alcohol for Antisepsis of Cutaneous Hand Microbes to Prevent Infection. *Phil J Microbiol Infect Dis 2000; 29(3):128-135*

62] Thormar H et al. (1987) Inactivation of enveloped viruses and killing of cells by fatty acids and monoglycerides. Antimicrobial Agents Chemother 31:27-31.

63] Isaacs CE et al. (1994) Inactivation of enveloped viruses in human bodily fluids by purified lipids. Ann NY Acad Sci 724:457-464.

64] Macallan D. C., Noble C., Baldwin C., Foskett M., McManus T., Griffin G. E. Prospective analysis of patterns of weight change in stage IV human immunodeficiency virus infection. Am. J. Clin. Nutr. 1993; 58:417-424

65] Isaacs CE and Schneidman K. (1991) Enveloped viruses in human and bovine milk are inactivated by added fatty acids(FAs) and monoglycerides(MGs). *J FASEB* 5: Abstract 5325, p.A1288.

66] Reiner DS et al. (1986) Human milk kills Giardia lamblia by generating toxic lipolytic products. *J Infect Dis* 154:825-832.

67] Hernell O et al. (1986) Killing of Giardia lamblia by human milk lipases: an effect mediated by lipolysis of milk lipids. *J Infect Dis* 153:715-720.

68] Crouch AA et al. (1991) Effect of human milk and infant milk formulae on adherence of Giardia intestinalis. *Trans Royal Soc Trop Med Hygiene* 85:617-619.

69] Oh DH and Marshall DL. (1993) Antimicrobial activity of ethanol, glycerol monolaurate or lactic acid against Listeria monocytogenes. *Int J Food Microbiol* 20:239-246.

70] Petschow BW et al. (1996) Susceptibility of Helicobacter pylori to bactericidal properties of medium-chain monoglycerides and free fatty acids.

71] Antonio, M. A., S. E. Hawes, and S. L. Hillier. 1999. The identification of vaginal *Lactobacill us species*
and the demographic and microbiologic characteristics of women colonized by these species. J. Infect. Dis.180:1950–1956.

72] Blomster-Hautamaa, D. A., and P. M. Schlievert. 1988. Preparation of toxicshock syndrome toxin-1. Methods Enzymol. 165:37–43.

73] Boris, S., and C. Barbes. 2000. Role played by lactobacilli in controlling the population of vaginal pathogens. Microbes Infect. 2:543–546.

74] Eckert, L. O. 2006. Clinical practice. Acute vulvovaginitis. N. Engl. J. Med. 355:1244–1252.

75] Eschenbach, D. A., S. Hillier, C. Critchlow, C. Stevens, T. DeRouen, and K. K. Holmes. 1988. Diagnosis and clinical manifestations of bacterial vaginosis.Am. J. Obstet. Gynecol. 158:819–828.

76] Fichorova, R. N., P. J. Desai, F. C. Gibson III, and C. A. Genco. 2001. Distinct proinflammatory host responses to *Neisseria gonorrhoeae* infection in immortalized human cervical and vaginal epithelial cells. Infect. Immun.69:5840–5848.

77] Fichorova, R. N., L. D. Tucker, and D. J. Anderson. 2001. The molecular basis of nonoxynol-9-induced vaginal inflammation and its possible relevance to human immunodeficiency virus type 1 transmission. J. Infect. Dis. 184:418–428.

78] Fidel, P. L., Jr. 2004. History and new insights into host defense against vaginal candidiasis. Trends Microbiol. 12:220–227.

79] Galvin, S. R., and M. S. Cohen. 2004. The role of sexually transmitted diseases in HIV transmission. Nat. Rev. Microbiol. 2:33–42.

80] Geisler, W. M., S. Yu, M. Venglarik, and J. R. Schwebke. 2004. Vaginal leucocyte counts in women with bacterial vaginosis: relation to vaginal and cervical infections. Sex. Transm. Infect. 80:401–405.

81] Hawes, S. E., S. L. Hillier, J. Benedetti, C. E. Stevens, L. A. Koutsky, P. Wolner-Hanssen, and K. K. Holmes. 1996. Hydrogen peroxide-producing lactobacilli and acquisition of vaginal infections. J. Infect. Dis. 174:1058–1063.

82] Laga, M., A. Manoka, M. Kivuvu, B. Malele, M. Tuliza, N. Nzila, J. Goeman, F. Behets, V. Batter, M. Alary, et al. 1993. Non-ulcerative sexually transmitted diseases as risk factors for HIV-1 transmission in women: results from a cohort study. AIDS 7:95–102.

83] Onderdonk, A. B., M. L. Delaney, G. R. Zamarchi, M. L. Hirsch, A. Munoz, and E. H. Kass. 1989. Normal vaginal microflora during use of various forms of catamenial protection. Rev. Infect. Dis. 11(Suppl. 1) **:**S61– S67.

84] Onderdonk, A. B., G. R. Zamarchi, M. L. Rodriguez, M. L. Hirsch, A. Munoz, and E. H. Kass. 1987. Quantitative assessment of vaginal microflora during use of tampons of various compositions. Appl. Environ. Microbiol. 53:2774–2778.

85] Patton, D. L., G. G. Kidder, Y. C. Sweeney, L. K. Rabe, and S. L. Hillier.1999. Effects of multiple applications of benzalkonium chloride and nonoxynol 9 on the vaginal epithelium in the pigtailed macaque (*Macaca nemestrina*).Am. J. Obstet. Gynecol. 180:1080–1087.

86] Patton, D. L., Y. C. Sweeney, L. K. Rabe, and S. L. Hillier. 1996. The vaginal microflora of pig-tailed macaques and the effects of chlorhexidine and benzalkonium on this ecosystem. Sex. Transm. Dis. 23:489–493.

87] Peterson, M., K. Ault, M. J. Kremer, A. J. Klingelhutz, C. C. Davis, C. A. Squier, and P. M. Schlievert. 2005. Innate immune system is activated by stimulation of vaginal epithelial cells with *Staphylococcus aureus* and toxic shock syndrome toxin-1. Infect. Immun. 73:2164–2174.

88] Peterson, M. L., P. M. Schlievert, K. L. Strandberg, L. C. Case, Y. C. Lin, M. C. Pack, and D. J. Chase. 2007. *Staphylococcus aureus* exotoxin production in vivo is reduced by glycerol monolaurate (GML) tampons, abstr. E450B. Abstr. 47th Intersci. Conf. Antimicrob. Agents Chemother. American Society for Microbiology, Washington, DC.

89] Peterson, M. L., and P. M. Schlievert. 2006. Glycerol monolaurate inhibits the effects of gram-positive select agents on eukaryotic cells. Biochemistry 45:2387–2397.

90] Plummer, F. A. 1998. Heterosexual transmission of human immunodeficiency virus type 1 (HIV): interactions of conventional sexually transmitted diseases, hormonal contraception and HIV-1. AIDS Res. Hum. Retrovir.14(Suppl. 1) **:**S5–S10.

91] Pollack, M., W. G. Weinberg, W. J. Hoskins, W. F. O'Brien, P. B. Iannini, S. E. Anderson, and P. M. Schlievert. 1983. Toxinogenic vaginal infections due to *Staphylococcus aureus* in menstruating rhesus monkeys without toxicshock syndrome. J. Infect. Dis. 147:963964.

92] Reinhardt, V. 2008. Taking better care of monkeys and apes: refinement of housing and handling practices for caged nonhuman primates. Animal Welfare Institute, Washington, DC.

93] Schlievert, P. M. 1982. Enhancement of host susceptibility to lethal endotoxin shock by staphylococcal pyrogenic exotoxin type C. Infect. Immun. 36:123–128.

94] Schlievert, P. M., J. R. Deringer, M. H. Kim, S. J. Projan, and R. P. Novick. 1992. Effect of glycerol monolaurate on bacterial growth and toxin production. Antimicrob. Agents Chemother. 36:626–631.

95] Schlievert, P. M., K. N. Shands, B. B. Dan, G. P. Schmid, and R. D. Nishimura. 1981. Identification and characterization of an exotoxin from *Staphylococcus aureus* associated with toxic-shock syndrome. J. Infect. Dis.143:509–516.

96] Sobel, J. D., S. Faro, R. W. Force, B. Foxman, W. J. Ledger, P. R. Nyirjesy, B. D. Reed, and P. R. Summers. 1998. Vulvovaginal candidiasis: epidemiologic, diagnostic, and therapeutic considerations. Am. J. Obstet. Gynecol. 178:203–211.

97] Spence, D. 2004. Candidiasis (vulvovaginal). Clin. Evid. 2:493–511.

98] Stone, R. L., and P. M. Schlievert. 1987. Evidence for the involvement of endotoxin in toxic shock syndrome. J. Infect. Dis. 155:682–689.

99] Thormar, H., C. E. Isaacs, K. S. Kim, and H. R. Brown. 1994. Inactivation of visna virus and

other enveloped viruses by free fatty acids and monoglycerides. Ann. N. Y. Acad. Sci. 724:465–471.

100] Vetter, S., T. J. Tripp, and P. M. Schlievert. 2005. Glycerol monolaurate inhibits virulence factor production in *Bacillus anthracis*. Antimicrob. Agents Chemother. 49:1302–1305.

101] Beatriz G. Carpo, Vermen M. Verallo-Rowell, Jon Kabara. (2007) Novel antibacterial activity of monolaurin compared with conventional antibiotics against organisms from skin infections: an in vitro study. Journal of Drugs in Dermatology.

102] Thormar, H., Bergsson, G., Gunnarsson, E., Georgsson, G., Witvrouw, M., Steingrímsson, Ó., De Clercq, E., and Kristmundsdóttir, Y. Hydrogels containing monocaprin have potent microbicidal activities against sexually transmitted viruses and bacteria in vitro. Sexually Transmitted Infections
75, 181-185, 1998.

103] Kristmundsdóttir, T., Árnadóttir, S.G., Bergsson, G., and Thormar, H. Development and evaluation of microbicidal hydrogels containing monoglycerides as the active ingredient. J ournal of Pharmaceutical Science
88, 1011-1015, 1999.

104] Glycerol monolaurate prevents mucosal SIV (simian immunodeficiency virus) transmission. *Nature. 2009 April; 458: 1034-1038*

105] Equivalence of Lauric Acid and Glycerol Monolaurate as Inhibitors of Signal Transduction in Staphylococcus aureus *. Journal of Bacteriology. 2000 May; 182(9): 2668-2671*

106] New Vaginal Gel Stops AIDS Virus. Cosmetic Ingredient glycerol monolaurate (GML) protects monkeys from AIDs Virus. *WebMD Health News Reviewed by Louise Chang, MD. 2009 March*

107] Glycerol Monolaurate (GML) inhibits the production of beta-lactamase, toxic shock toxin-1, and other staphylococcal exoproteins by interfering with signal transduction. *Journal of Bacteriology. 1994 July; 176(14): 4204-4209*

108] Modulation of immune cell proliferation by glycerol monolaurate. *Clinical and Diagnostic Laboratory Immunology. 1996 January; 3(1): 10-13*

109] Formulation of food-grade microemulsions with glycerol monolaurate: effects of short-chain alcohols, polyols, salts and nonionic surfactants. *Eur Food Res Technol. 2008; 226: 613-619*

110] Glycerol Monolaurate Inhibits the Effects of Gram Positive Select Agents on Eukaryotic Cells. *Biochemistry. 2006 February; 45(7): 2387-2397*

111] Detection of human papillomaviruses and other agents causing sexually transmitted diseases (STD) with molecular diagnosis methods. *Acta Med Croatica. 2003; 57(4): 295-301*

112] Booster Concentrate: A patented highly purified distilled monoglyceride (Microorganisms inactivated by Booster concentrate in Laboratory Studies)

113] In the battle with humans, HIV stays one step ahead. *Ethiopian News. 2009 March*

114] Glycerol Monolaurate inhibits Virulence Factor Production in Bacillus anthracis. *Antimicrobial Agents and Chemotherapy. 2005 April; 49(4): 1302-1305*

115] Glycerol Monolaurate does not alter rhesus macaque (Macaca mulatta) vaginal lactobacilli and is safe for chronic use. *Antimicrobial Agents and Chemotherapy. 2008 December;*

52(12): 4448-4454

116] University of Minnesota discovery offers potential breakthrough in preventing HIV transmission. *Minnesota Public Radio, 2009 March.*

117] Glycerol Monolaurate (GML) in Virgin Coconut Oil destroys tuberculosis bacteria. *Living with Nature by*
Dr. Abercio V. Rotor, 2009 June.

118] Radical Approach to Block HIV Gets Some Results. *Dream Big Dreams, 2009 March*

119] In vitro effects of monolaurin compounds on enveloped RNA and DNA viruses. *Journal of Food Safety.*
1982; 4: 1-12

120] Inactivation of Enveloped Viruses and killing of cells by Fatty Acids and Monoglycerides. *A ntimicrobial*
Agents and Chemotherapy. 1987 January; 31(1): 27-31

121] Hydrogels containing monocaprin have potent microbicidal activities against sexually transmitted viruses and bacteria in vitro. *Sex Transm Infect. 1999 June; 75(3): 181-185.*

122] Coconut: In Support of Good Health in the 21st Century

123] HIV Can Be Killed, During Sex or In an Infected Person

124] Reduction in Staphylococcus aureus Growth and Exotoxin Production and Levels Due to Glycerol Monolaurate in Tampons. Clin Infect Dis. 2009 Oct 28; 19863450