

From Palm Kernel to Membrane-Active Anti-Infectives: Lauric Acid and Glycerol Monolaurate Against Antimicrobial-Resistant and Biofilm-Associated Infections— A Review

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ABSTRACT

Antimicrobial resistance (AMR) and biofilm-associated infections increasingly limit the effectiveness of conventional antimicrobial therapy and create demand for adjunctive agents operating through unconventional targets. Lauric acid, a medium-chain saturated fatty acid abundant in palm kernel oil, and its monoacylglycerol derivative glycerol monolaurate (GML; monolaurin) have attracted renewed interest because of membrane-active antimicrobial, antibiofilm, antivirulence, and immunomodulatory properties. This qualitative literature review synthesizes contemporary evidence published principally during 2020–2026 and evaluates the pathway from palm-kernel-derived lauric feedstocks to clinically relevant antimicrobial applications. Evidence indicates that lauric acid and GML can perturb microbial membranes, alter membrane potential and fluidity, suppress virulence-associated signaling, interfere with biofilm development, and enhance the activity of selected antibiotics. Recent studies extend these observations to methicillin-resistant *Staphylococcus aureus*, antibiotic-resistant *S. aureus* recovered from patients with atopic dermatitis, polymicrobial biofilms, and selected multidrug-resistant Gram-negative clinical isolates. However, efficacy is strongly influenced by organism, biofilm state, biological matrix, exposure concentration, and formulation. Liposomes, nanoemulsions, albumin-associated systems, and petrolatum illustrate how delivery technology can bridge—or widen—the gap between biochemical activity and therapeutic feasibility. Major translational deficiencies remain in human pharmacokinetics, standardized formulations, dose optimization, microbiome safety, resistance-evolution studies, and controlled clinical trials. Palm-kernel-derived GML therefore represents a promising but incompletely validated platform whose development requires integration of microbiology, pharmaceuticals, infection biology, and pharmaceutical-grade biorefinery science.

Keywords: Antimicrobial resistance; Glycerol monolaurate; Monolaurin; Lauric acid; Palm kernel oil; Biofilm; MRSA; Membrane disruption; Antimicrobial lipids; Clinical translation

JEL Classification: I15; I18; L65; O32; Q16.

1. INTRODUCTION

Antimicrobial resistance has moved from being primarily a microbiological problem to becoming a major global health, health-system, and development challenge. The most comprehensive global estimates indicate that bacterial AMR is already associated with millions of deaths annually and that its cumulative burden could remain substantial through 2050 without more effective prevention, stewardship, diagnostics, and therapeutic innovation. Particularly concerning are methicillin-resistant *Staphylococcus aureus* (MRSA), vancomycin-resistant enterococci, extended-spectrum β -lactamase-producing Enterobacterales, carbapenem-resistant Gram-negative bacteria, and resistant *Pseudomonas aeruginosa* and *Acinetobacter* species, because therapeutic choices for these organisms may become narrow, toxic, expensive, or clinically unreliable.^{1,2} The challenge therefore requires not only discovery of conventional antibiotics with new molecular targets but also development of adjunctive approaches capable of weakening resistant organisms through mechanisms that differ from established drug classes.

Biofilm formation compounds this problem. Bacteria and fungi growing within biofilms are physiologically and spatially different from freely suspended planktonic organisms. Extracellular polymeric substances, extracellular DNA, proteins, lipids, altered nutrient and oxygen gradients, slow-growing subpopulations, stress responses, persister cells, and horizontal transfer of resistance determinants collectively reduce antimicrobial susceptibility and facilitate chronic infection. Consequently, planktonic minimum inhibitory concentrations cannot be assumed to predict activity within mature biofilms, and standardized biofilm susceptibility testing remains considerably less developed than conventional antimicrobial susceptibility testing.^{3,4,5,6} Biofilms are especially important in chronic wounds, diabetic ulcers, endocarditis, prosthetic and orthopedic infections, intravascular and urinary catheters, respiratory disease, and recurrent mucosal or dermatological infection.^{7,8,9}

These problems have renewed interest in antimicrobial lipids. Free fatty acids and monoacylglycerols occupy an unusual position in antimicrobial biology because they may simultaneously disturb microbial membranes, alter membrane-associated signaling, influence virulence, and interfere with biofilm development. Rather than interacting exclusively with one enzyme or ribosomal target, antimicrobial fatty acids can alter the physical organization of bacterial membranes and modify processes that depend on membrane integrity and lipid organization.^{10,11,12} Such multimodal behavior creates an attractive hypothesis: lipid-based antimicrobials could complement conventional antibiotics and potentially make certain resistant or tolerant populations more vulnerable.

Lauric acid, or dodecanoic acid, is a 12-carbon saturated medium-chain fatty acid of particular relevance because palm kernel oil is exceptionally rich in lauric lipids. Lauric acid can be esterified with glycerol to produce glycerol monolaurate (GML), commonly termed monolaurin. GML has long been used or investigated in food, hygiene, and natural-product applications, but contemporary antimicrobial research has expanded its relevance beyond food preservation. Recent work has examined MRSA isolates from wounds, multidrug-resistant clinical isolates, antibiotic-resistant *S. aureus* associated with atopic dermatitis, antibiotic–GML combinations, and pharmaceutical formulations designed to improve antimicrobial availability.^{13,14,15,16,17}

The palm-kernel connection is important but requires careful interpretation. The biological molecule GML is not intrinsically different because its lauric acid originated from palm kernel rather than another chemically equivalent source, provided pharmaceutical purity and stereochemical specifications are identical. The relevance of palm kernel lies instead in feedstock availability, fractionation, process efficiency, purity, traceability, and opportunities for higher-value biorefinery applications. Enzymatic glycerolysis of palm kernel olein–stearin blends has already demonstrated that palm-kernel fractions can serve as substrates for monolaurin production, supporting a credible feedstock-to-product pathway.¹⁸ Translational medicine therefore provides a potential high-value destination for a compound whose industrial production may be integrated with a lauric-oil biorefinery.

Nevertheless, enthusiasm requires restraint. Demonstrating antibacterial activity in a laboratory strain is fundamentally different from showing efficacy against resistant clinical isolates; demonstrating efficacy against resistant isolates *in vitro* is different from curing infection in an animal; and demonstrating favorable topical activity is different from establishing systemic pharmacokinetic feasibility. Bioavailability, protein binding, formulation, tissue exposure, metabolism, toxicity, microbiome perturbation, and the evolutionary response of microorganisms are all potential determinants of clinical performance. Bacteria can also adapt to antimicrobial fatty acids through membrane remodeling, detoxification, efflux, incorporation of exogenous fatty acids, and other mechanisms, making it inappropriate to describe membrane-active lipids as evolutionarily “resistance-proof”.^{19,20}

This qualitative literature review therefore examines the evidence using a mechanism-to-translation perspective. It asks how lauric acid and GML interact with microbial membranes and signaling systems; how their activity changes in biofilms, resistant organisms, and polymicrobial systems; whether they can enhance established antibiotics; how formulation and biological matrices influence activity; what host and microbiome consequences require attention; and how a palm-kernel-derived pharmaceutical pathway might be developed without overstating the existing clinical evidence. The central argument is that lauric acid and GML constitute scientifically credible membrane-active antimicrobial platforms, but their future clinical value will be determined as much by delivery, pharmacology, and evidence quality as by intrinsic antimicrobial potency.

2. LITERATURE REVIEW: CONCEPTUAL AND THEORETICAL FOUNDATIONS

2.1 Lauric Acid, Glycerol Monolaurate, and Antimicrobial Lipid Chemistry

Lauric acid combines a hydrophobic hydrocarbon chain with a polar carboxyl group, enabling interaction with biological lipid interfaces. Antimicrobial behavior among fatty acids is influenced by carbon-chain length, saturation, ionization state, concentration, target-organism membrane composition, temperature, pH, and the surrounding chemical environment. Medium-chain fatty acids occupy a particularly interesting structural space because they are sufficiently hydrophobic to partition into membranes but remain more dispersible than many longer-chain fatty acids. Antimicrobial fatty acids can therefore act both as direct growth inhibitors at sufficiently high concentrations and as modulators of biofilm, adhesion, virulence, and signaling at lower concentrations.^{10,12}

GML is the monoester formed from glycerol and lauric acid. Esterification changes the polar headgroup and physicochemical behavior while preserving the C12 hydrophobic chain. Structure–activity experiments with GML derivatives suggest that acyl-chain length, glycerol headgroup structure, and linkage chemistry strongly influence

biological activity, including membrane-associated immunomodulatory effects.²¹ The relationship between molecular structure and function is therefore more complex than simply attributing all activity to the presence of a lauryl chain.

Natural antimicrobial lipids operate within a broader biological context. Fatty acids and monoacylglycerols occur in host secretions and lipid-rich biological environments and may contribute to innate antimicrobial defense. GML has been identified and quantified in human milk using modern LC–MS/MS methodology, illustrating that monoacylglycerols can exist within complex physiological matrices rather than only in manufactured antimicrobial formulations.²² GML has also shown virucidal activity against enveloped viruses, supporting the broader principle that lipid-envelope or membrane-associated structures can be vulnerable to amphiphilic lipid molecules.²³ These observations do not establish therapeutic efficacy against bacterial infection but support the biological plausibility of membrane-active lipid defense.

2.2 Membrane Disruption as a Core Mechanistic Axis

The bacterial cytoplasmic membrane performs indispensable functions, including maintenance of electrochemical gradients, transport, energy generation, cell-wall precursor organization, signal transduction, and spatial localization of proteins. Disruption therefore produces effects that extend well beyond simple membrane leakage. Lauric acid has been shown to alter *S. aureus* morphology, compromise membrane integrity, and change membrane-associated fluorescence measurements consistent with effects on membrane fluidity and depolarization (Liu et al., 2024). These observations provide contemporary experimental support for membrane disruption as a central component of lauric acid activity.

The concept of membrane targeting also helps explain potential synergy with conventional antibiotics. Antibiotic-induced accumulation of membrane-associated cell-wall intermediates such as lipid II can create abnormal membrane domains that become vulnerable to antimicrobial fatty acids. Although the best mechanistic evidence for this interaction has involved selected unsaturated fatty acids rather than lauric acid specifically, it establishes an important mechanistic principle: perturbation produced by a conventional antibiotic may alter membrane organization in ways that amplify lipid-mediated killing.²⁴ This creates a theoretical basis for combinations in which antimicrobial lipids do not replace antibiotics but function as adjuvants.

GML similarly affects membrane-dependent systems but cannot be reduced to a non-specific detergent. At concentrations below those necessary for rapid killing, monoacylglycerols can influence signal transduction, toxin expression, and other regulatory systems. This is pharmacologically relevant because an intervention that reduces pathogenicity or biofilm formation without immediately sterilizing the environment may still modify infection behavior. Conversely, sublethal exposures could impose selective or adaptive pressures and therefore require careful evaluation rather than automatic interpretation as beneficial antivirulence activity.

Gram-positive and Gram-negative organisms differ markedly in their susceptibility to antimicrobial lipids. Gram-positive bacteria expose a cytoplasmic membrane beneath a thick but porous peptidoglycan layer, whereas Gram-

negative organisms possess an additional outer membrane rich in lipopolysaccharide. This outer membrane can substantially reduce access of hydrophobic compounds to the inner membrane. Reviews of antimicrobial lipids therefore generally report stronger or more reproducible activity against many Gram-positive organisms than against Gram-negative species.^{11,12} Nevertheless, formulation, binding environment, surfactant structure, concentration, and chemical modification can modify this barrier²⁵, for example, demonstrated that polyglycerol monolaurates can show activity against Gram-negative bacteria, emphasizing that headgroup architecture and delivery context matter.

2.3 Biofilm Formation, Tolerance, and Resistance

Biofilm development is commonly conceptualized as progression from reversible attachment to irreversible adhesion, microcolony formation, maturation, and eventual dispersion. During maturation, organisms become embedded in a matrix containing polysaccharides, proteins, extracellular DNA, lipids, and host-derived materials. Biofilms are physiologically heterogeneous: organisms close to nutrient-rich interfaces may remain metabolically active, whereas cells in nutrient-limited regions may grow slowly or enter dormant states. This heterogeneity reduces the effectiveness of antibiotics whose killing depends on active growth.^{4,6}

Biofilm tolerance should be distinguished from inherited antimicrobial resistance. Tolerance can arise from physical protection, slow growth, persister formation, altered metabolism, and diffusion limitations without a conventional resistance gene. At the same time, biofilms can facilitate genuine resistance evolution by providing dense microbial communities in which stress, mutation, horizontal gene transfer, and prolonged subinhibitory exposure occur.²⁶ This distinction is central to evaluating lauric acid and GML. Reduction of planktonic MIC does not establish biofilm eradication, while inhibition of initial biofilm formation is not equivalent to destruction of a mature biofilm.

This distinction also has methodological consequences. Conventional susceptibility testing is predominantly optimized for planktonic organisms, and routine clinical laboratories generally do not perform standardized minimum biofilm eradication concentration testing. Biofilm models vary substantially with surface, medium, incubation duration, inoculum, shear conditions, staining method, viable-cell endpoint, and maturity.⁵ therefore argues that progress in understanding biofilm susceptibility has not yet translated into universally standardized clinical testing. Any review of GML or lauric acid must consequently interpret biofilm data in relation to the assay used rather than presenting different experimental endpoints as directly interchangeable.

2.4 Antibiofilm and Antivirulence Effects of Fatty Acids

Fatty acids can affect biofilm development through several mechanisms. They may alter microbial cell-surface properties and initial attachment, interfere with extracellular matrix formation, modify membrane-dependent signaling, suppress fimbrial or hyphal development, and mimic endogenous signaling molecules. Kumar et al. (2020)¹⁰ highlighted the ability of several fatty acids to influence diffusible signal-factor pathways, autoinducer systems, motility, fimbriae, and virulence. This provides a conceptual framework for interpreting antibiofilm effects at concentrations below the MIC.

Candida biology offers a clear example. Transition from yeast to hyphal growth is central to *Candida albicans* biofilm development and tissue invasion. Lee et al. (2021)²⁷ screened multiple fatty acids and reported that several medium-chain fatty acids, including lauric acid, inhibited hyphal development and biofilm formation at concentrations below their growth-inhibitory concentrations. The transcriptional patterns induced by selected fatty acids partly resembled responses associated with the quorum-sensing molecule farnesol. These findings suggest that antimicrobial lipids may operate through regulatory as well as directly fungicidal mechanisms.

Polymicrobial biofilms are even more clinically relevant because chronic infections frequently contain more than one organism. Kim et al. (2022)²⁸ demonstrated antibiofilm activity of lauric and myristic acids in models involving *S. aureus*, *Escherichia coli*, and *C. albicans*. Bharathi, Lee and Lee (2024)²⁹ subsequently showed that liposomal incorporation of lauric acid markedly enhanced activity against individual and dual *S. aureus*–*C. albicans* biofilms compared with free lauric acid. The latter study is important not merely because liposomes improved numerical activity but because it illustrates how delivery technology can change the biological phenotype of an otherwise poorly dispersed antimicrobial lipid.

3. METHODS

This study employed a qualitative literature review rather than a systematic literature review or meta-analysis. The objective was interpretive integration of mechanistic, microbiological, formulation, host-response, and translational evidence rather than exhaustive enumeration of every study satisfying a predetermined protocol. Accordingly, the review does not claim PRISMA compliance, pooled-effect estimation, or systematic risk-of-bias scoring.

The literature base emphasized peer-reviewed journal articles published from 2020 through September 2026. PubMed/MEDLINE, Scopus, Web of Science, and journal-publisher databases were considered the principal sources for relevant literature, supplemented by citation chaining and targeted searches for recent studies. Search concepts combined exposure terms such as “lauric acid,” “dodecanoic acid,” “glycerol monolaurate,” “monolaurin,” “monoacylglycerol,” and “palm kernel” with terms including “antimicrobial resistance,” “MRSA,” “multidrug resistant,” “carbapenem resistant,” “biofilm,” “*Candida*,” “wound,” “membrane,” “virulence,” “quorum sensing,” “synergy,” “nanoemulsion,” “liposome,” “albumin,” “petrolatum,” “microbiome,” and “clinical translation.”

Evidence directly evaluating lauric acid or GML was prioritized. However, contextual literature on AMR burden, biofilm biology, resistance adaptation, implant infection, chronic wounds, *Candida* biofilms, atopic dermatitis, and pharmaceutical delivery was included when necessary to interpret translational relevance. Studies involving laboratory strains, clinical isolates, cell cultures, animals, biological matrices, and formulation platforms were included because the principal aim was to map the translation continuum. Food microbiology studies were retained only where they provided mechanistic or formulation evidence applicable to antimicrobial biology.

Qualitative synthesis was organized across seven themes: molecular mechanism; biofilm and virulence modulation; pathogen and resistance phenotype; antibiotic combinations; formulation and biological matrices; host and microbiome effects; and pharmaceutical and industrial translation. Greater interpretive weight was assigned to

evidence that used resistant clinical isolates rather than only laboratory strains, mature biofilms rather than attachment alone, physiologically relevant matrices rather than solvent-only systems, and host or clinical contexts rather than food-preservation endpoints. Importantly, use of a clinical isolate was not treated as clinical evidence. Similarly, evidence from animal or cellular studies was not extrapolated as proof of human therapeutic benefit. The major methodological limitation is the qualitative design itself. Narrative selection may introduce emphasis bias, while large differences among antimicrobial assays limit quantitative comparison. MIC, minimum bactericidal concentration, time-kill curves, biofilm inhibitory concentration, biofilm eradication endpoints, and microscopy are not interchangeable. Concentrations are also reported in different units, and apparent activity can be altered by solvent, carrier, protein binding, inoculum, pH, and growth medium. These limitations were treated as findings relevant to translation rather than merely as weaknesses of individual studies.

4. RESULTS: THEMATIC FINDINGS

4.1 Contemporary Evidence Landscape

The contemporary evidence base has evolved from broad descriptions of antimicrobial lipids toward increasingly specific models of resistant pathogens, biofilms, combinations, and drug delivery. Reviews published during 2020–2022 established the general antimicrobial, antibiofilm, and antivirulence properties of fatty acids and highlighted membrane effects, signaling interference, and opportunities for combination therapy.^{10,11,12} Subsequent experimental studies increasingly focused on resistant *S. aureus*, polymicrobial biofilms, and formulation-dependent enhancement. A recent review of antimicrobial and antibiofilm microbial lipids similarly emphasizes membrane perturbation, permeability changes, and delivery-enabled antibiofilm strategies while identifying *in vivo* translation as a persistent evidence gap.³⁰

MRSA is currently the strongest organism-specific evidence cluster. Abd El-Ghany et al. (2024a)¹³ evaluated 103 MRSA isolates from wound infections and reported monolaurin activity against both biofilm formation and preformed biofilms. Monolaurin also reduced expression of *icaD*, a gene associated with polysaccharide intercellular adhesin-related staphylococcal biofilm development. Importantly, the concentrations necessary for biofilm inhibition and eradication differed, reinforcing the principle that prevention of biofilm establishment and treatment of mature biofilms are distinct therapeutic problems.

A companion study examined monolaurin in combination with β -lactam antibiotics and reported strong *in vitro* synergistic interactions against *S. aureus*.¹⁴ Such synergy is particularly interesting because β -lactam resistance in MRSA ordinarily limits the utility of these drugs. However, checkerboard or fractional inhibitory concentration results should be interpreted as pharmacodynamic evidence warranting further study rather than proof that monolaurin restores clinical β -lactam susceptibility in patients.

Evidence expanded further in 2025. Laowansiri et al. (2025)¹⁶ isolated *S. aureus* from children and adults with atopic dermatitis and identified methicillin-, mupirocin-, and fusidic-acid-resistant isolates. Monolaurin inhibited resistant isolates at low concentrations in the reported assay and did not demonstrate cytotoxicity in the tested cultured epidermal and dermal cells. This study strengthens clinical relevance because the organisms originated from patients and were linked to a disease in which *S. aureus* colonization is highly relevant. Nevertheless, the

intervention was applied to isolates rather than directly to patient disease; the study therefore supports therapeutic plausibility but not clinical efficacy.

Alrabiah et al. (2025)¹⁵ provided another important extension by evaluating GML against multidrug-resistant clinical isolates, including carbapenem-resistant Gram-negative organisms and vancomycin-resistant bacteria. The investigators reported measurable activity against carbapenem-resistant *E. coli*, *Klebsiella pneumoniae*, and *Enterobacter cloacae* and observed enhanced killing when GML was associated with human serum albumin. This result challenges the simplistic assumption that GML has no meaningful activity against Gram-negative bacteria. At the same time, it should not be generalized to all Gram-negative species, formulations, or physiological conditions.

4.2 Membrane-Centric Antimicrobial Activity

Direct contemporary mechanistic evidence supports membrane disruption as a major effect of lauric acid. Liu et al. (2024)³¹ showed concentration-dependent inhibition of *S. aureus*, morphological abnormalities by electron microscopy, increased membrane permeability, and changes consistent with membrane depolarization and altered fluidity. These effects provide mechanistic continuity between the physicochemical properties of medium-chain fatty acids and observed bacterial killing.

Membrane disruption is likely to produce secondary consequences. Loss of membrane integrity alters ionic homeostasis, proton motive force, nutrient transport, protein localization, and cellular energetics. Antibacterial lipids may therefore generate a cascade rather than a single molecular lesion. Casillas-Vargas et al. (2021)¹² emphasized that fatty-acid antimicrobial activity can include membrane insertion, leakage, interference with energy production, oxidative processes, and disruption of macromolecular synthesis. This complexity may make single-target descriptions inadequate.

However, the bacterial envelope simultaneously provides opportunities for adaptation. Kengmo Tchoupa, Eijkelkamp and Peschel (2022)¹⁹ described several bacterial responses to antimicrobial host fatty acids, including extracellular shielding, modification or detoxification of fatty acids, efflux, metabolic utilization, and incorporation of exogenous lipids into bacterial membranes. MacDermott-Opeskin, Gupta and O'Mara (2022) similarly emphasized the importance of lipid remodeling in antimicrobial susceptibility and resistance. These observations are highly relevant to GML development: a broad membrane-associated mechanism may reduce dependence on a single mutable drug target but does not eliminate evolutionary adaptation.

4.3 GML, Biofilm Formation, and Mature Biofilms

Among recent studies, the MRSA wound-isolate investigation by Abd El-Ghany et al. (2024a)¹³ provides particularly direct evidence linking GML with biofilm phenotypes. Biofilm formation was widespread among the tested isolates, and monolaurin reduced both new biofilm formation and established biofilms in a concentration-dependent manner. Scanning electron microscopy demonstrated diminished cellular attachment and biofilm structure, while *icaD* expression decreased after monolaurin exposure. The convergence of phenotypic, imaging, and transcriptional evidence is more compelling than reliance on a single crystal-violet endpoint.

Nevertheless, substantial methodological uncertainty remains. Biofilm assays differ in the time allowed for maturation, the surface used, medium composition, organism density, and the distinction between reduced biomass and actual loss of viable organisms. Coenye (2023)⁵ noted that clinically standardized biofilm susceptibility testing is still lacking, while Ciofu et al. (2022)⁴ and Liu, Prentice and Webber (2024)⁶ emphasized that biofilms can simultaneously display matrix protection, metabolic dormancy, persister formation, and heritable resistance mechanisms. Consequently, claims that GML “eradicates biofilms” should specify the organism, assay, concentration, exposure duration, and definition of eradication.

The importance of polymicrobial biofilms further complicates interpretation. Kim et al. (2022)²⁸ showed that lauric acid interfered with mixed bacterial–fungal biofilms, while Bharathi, Lee and Lee (2024) reported markedly improved activity when lauric acid was incorporated into liposomes. *Candida* biofilms are particularly important because *Candida* species display reduced antifungal susceptibility in the biofilm state and can participate in cross-kingdom interactions with bacteria.^{32,33} Medium-chain fatty acids may add an antivirulence dimension because they can suppress *C. albicans* hyphal development at sub-growth-inhibitory concentrations.²⁷

The evidence therefore supports a multilevel mechanism rather than a single bactericidal pathway. **Figure 1** integrates the major mechanistic routes through which lauric acid and GML may act, beginning with membrane partitioning and extending to effects on signaling, virulence, biofilms, and antibiotic susceptibility.

Figure 1: Proposed multilevel antimicrobial mechanisms of lauric acid and glycerol monolaurate

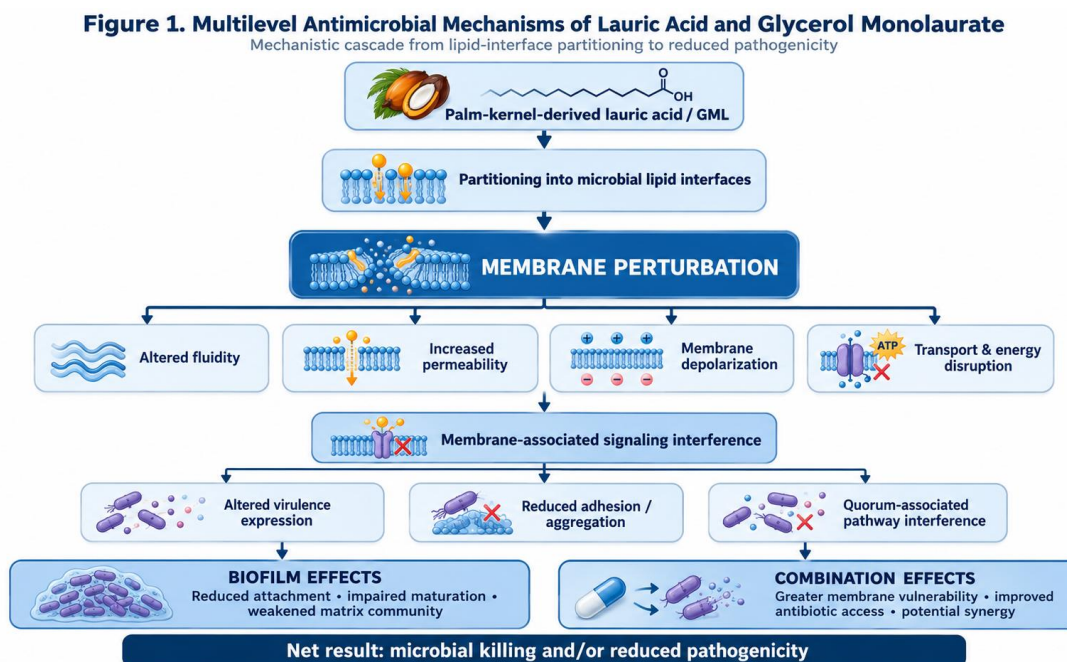


Figure 1 emphasizes that membrane interaction should be treated as an initiating event rather than the sole biological endpoint. Lauric acid-mediated membrane damage can propagate into altered metabolism and cell-wall organization, while GML at sublethal concentrations may interfere with membrane-linked signaling and virulence. The resulting phenotype can include reduced biofilm formation or enhanced susceptibility to conventional

antimicrobials. At the same time, the relative contribution of each pathway is organism- and formulation-dependent and should not be assumed to be identical across Gram-positive bacteria, Gram-negative bacteria, and fungi.^{10,12,31}

4.4 Antibiotic Combination Effects

Combination therapy is potentially one of the most clinically relevant directions for antimicrobial lipids. An adjunctive agent does not necessarily need to replace an antibiotic if it can lower the concentration needed, restore activity against a resistant population, accelerate killing, or disrupt a biofilm sufficiently to make established drugs more effective.

The monolaurin- β -lactam findings reported by [Abd El Ghany et al. \(2024b\)](#) and [Abd El-Ghany et al. \(2024a\)](#)^{13,14} are therefore noteworthy. Strong fractional inhibitory concentration interactions were observed against *S. aureus* and MRSA isolates. Mechanistically, an antimicrobial lipid could facilitate antibiotic activity through membrane perturbation, altered transport, changes in cell-wall synthesis dynamics, or disruption of envelope-associated resistance processes. [Sidders et al. \(2023\)](#)²⁴ demonstrated a related mechanism in which antibiotic-induced lipid II accumulation sensitized Gram-positive bacteria to antimicrobial fatty acids by creating abnormal membrane domains and loss of membrane integrity.

These results support a conceptual shift from “natural antimicrobial versus antibiotic” to “lipid-antibiotic pharmacodynamic partnership.” Such a shift is potentially important for stewardship because an effective adjunct might extend the useful life of established agents. Yet several steps remain necessary. Synergy should be reproduced across genetically diverse isolates, confirmed with time-kill methods, evaluated within mature biofilms, tested in physiological matrices, and studied in animal infection models before clinical conclusions are justified.

4.5 Gram-Negative Activity and the Biological-Matrix Paradox

Gram-negative activity represents both an opportunity and a source of uncertainty. The outer membrane usually limits penetration of hydrophobic antimicrobial compounds, explaining why many fatty acids and monoacylglycerols have historically shown stronger activity against Gram-positive organisms. [Chen et al. \(2021\)](#)³⁴, studying GML-containing disinfectant systems against *Salmonella*, and [Zhao et al. \(2021\)](#), examining polyglycerol monolaurates against Gram-negative bacteria, demonstrated that activity may be modified substantially by formulation and surfactant chemistry.

The 2025 albumin-bound GML study is especially provocative. Albumin binding might ordinarily be expected to reduce the free concentration of a lipophilic antimicrobial. Instead, [Alrabiah et al. \(2025\)](#)¹⁵ reported enhanced killing against selected resistant organisms when GML was combined with human serum albumin. Several explanations are plausible, including improved dispersion, delivery of GML to the microbial surface, altered equilibrium between bound and free fractions, or stabilization of otherwise poorly soluble material. Definitive mechanism requires further study.

This finding also illustrates why pharmacological translation cannot rely on nominal concentration alone. A concentration reported in broth does not indicate how much compound will remain unbound, biologically

accessible, or stable in plasma, wound exudate, sebum, milk, tissue fluid, or an intracellular environment. Protein binding may decrease activity for one compound and facilitate delivery for another. Biological matrices should therefore be incorporated early in future development rather than treated as a late-stage pharmacokinetic complication.

4.6 Formulation as a Determinant of Biological Potency

Poor aqueous dispersibility is an intrinsic development challenge for lipid antimicrobials. Several contemporary studies demonstrate that delivery systems can fundamentally alter apparent activity. Liposomal lauric acid showed substantially greater antibiofilm activity than unformulated lauric acid in *S. aureus*, *C. albicans*, and mixed biofilms.²⁹ GML-containing microemulsions and nanoemulsion systems have likewise been developed to improve dispersion, stability, or antimicrobial contact.^{35,36} Recent reviews of liposomal and other lipid nanocarriers likewise indicate that encapsulation can improve antimicrobial stability, biofilm penetration, controlled release, and local exposure, although safety, manufacturing scalability, and formulation reproducibility remain important translational constraints.^{37,38}

Electrospun systems provide another approach. Clitherow et al. (2020)³⁹ incorporated medium-chain fatty acids into polymeric electrospun patches and demonstrated inhibition of *C. albicans* and reduced biofilm viability. Zhang et al. (2021)⁴⁰ developed GML microemulsion-loaded gelatin nanofibers with antimicrobial properties. These systems are particularly relevant for topical wounds or biomedical surfaces because local delivery may provide sustained exposure while avoiding the systemic distribution problem encountered with orally or intravenously administered lipids.

The 2026 study of GML formulated in petrolatum advances the topical-translation argument. Schlievert et al. (2026)¹⁷ compared several glycerol fatty-acid monoesters and found GML to be particularly active against *S. aureus*. Incorporation into petrolatum retained or supported antibacterial activity against methicillin-susceptible and methicillin-resistant strains. Because petrolatum is already widely used as a topical semisolid vehicle, this finding creates a plausible development path for conditions involving superficial *S. aureus* colonization. It does not, however, establish efficacy in atopic dermatitis, diabetic ulcers, or invasive infection until controlled therapeutic studies demonstrate clinical benefit.

Formulation should therefore be positioned between molecular potency and clinical performance rather than placed at the end of drug development. Figure 2 illustrates this translation bottleneck.

Figure 2: Formulation as the bridge between intrinsic antimicrobial activity and therapeutic exposure

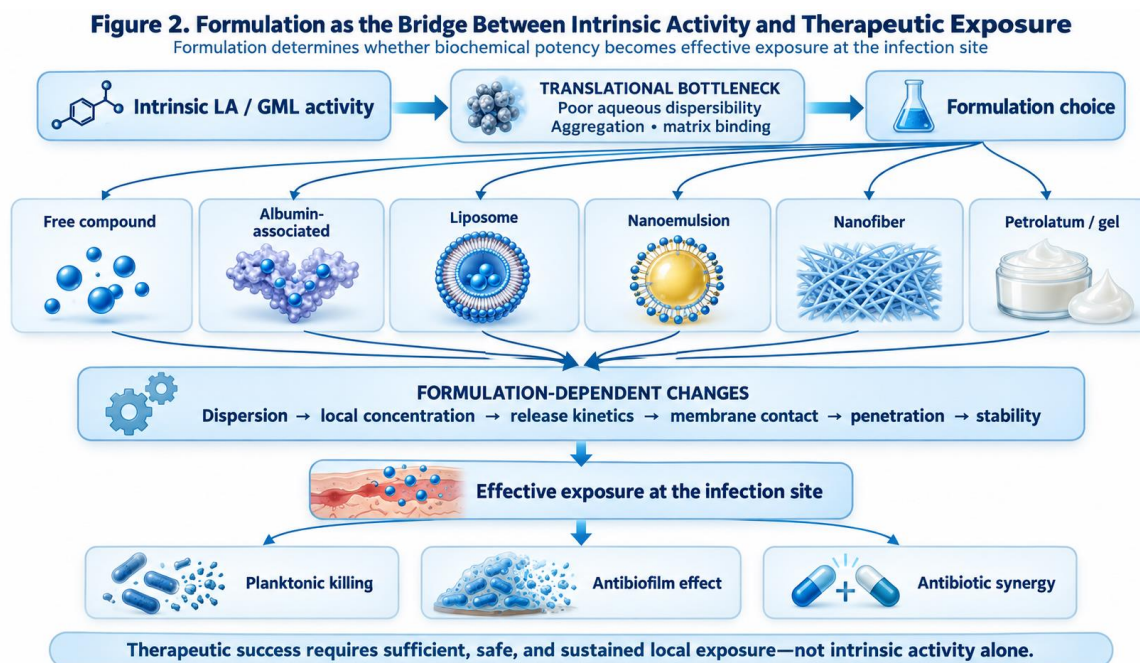


Figure 2 conceptualizes formulation as a pharmacological variable rather than an inert carrier. Liposomes can increase interaction with microbial or biofilm surfaces; nanoemulsions can improve dispersion; proteins can unexpectedly modify delivery; nanofibers can provide sustained release; and semisolid vehicles can maintain high topical concentrations. Consequently, contradictory results between studies may reflect delivery conditions as much as intrinsic microbiological susceptibility.^{17,29,39,40}

4.7 Host, Inflammation, and Microbiome Effects

GML is unusual among candidate antimicrobials because its biological activity extends into host signaling. Fosdick et al. (2021)²¹ demonstrated suppression of human T-cell activation by GML and related derivatives, with structure–activity differences depending on lipid architecture. These findings could be advantageous in diseases in which excessive inflammation contributes to pathology, but they also raise a safety question: immunomodulation beneficial in one tissue or dose may be undesirable in another.

Animal intestinal studies similarly indicate that GML can influence both immune responses and microbial community composition. Mo et al. (2021)⁴¹ reported altered gut-microbiota patterns and anti-inflammatory responses in a mouse model of dextran-sodium-sulfate-induced colitis. He et al. (2022)⁴² observed changes in inflammatory-cell infiltration, inflammatory signaling, epithelial barrier markers, and gut microbiota. Related work has reported effects on intestinal mucosal integrity and immune pathways in animal models.⁴³

These findings support biological activity but complicate the translational picture. A compound intended to suppress a pathogen can also affect commensals and host immune signaling. For topical applications, this matters because healthy skin microbiota contribute to colonization resistance and immune homeostasis. Atopic dermatitis is

characterized by reduced microbial diversity and frequent expansion of *S. aureus*, particularly during flares.^{44,45,46} further demonstrated genomic diversity and convergent biofilm traits among *S. aureus* populations associated with severe atopic dermatitis. An ideal topical GML formulation would therefore reduce pathological *S. aureus* burden without indiscriminately damaging protective commensal communities.

5. DISCUSSION AND ANALYSIS

5.1 What the Evidence Now Supports

The most defensible interpretation of the contemporary evidence is that lauric acid and GML are biologically credible membrane-active antimicrobial compounds with meaningful activity against selected resistant and biofilm-forming organisms. The evidence is strongest for *S. aureus*, including MRSA, where multiple recent studies converge on direct antimicrobial activity, antibiofilm effects, gene-expression changes, antibiotic synergy, and formulation-dependent enhancement.^{13,14,16}

The evidence also supports a broader antivirulence concept. Fatty acids can affect quorum-associated signaling and structural transitions important to pathogenicity, as demonstrated particularly clearly for *Candida* hyphal development and biofilm formation.²⁷ The value of an antivirulence strategy lies in weakening the pathogenic phenotype even when immediate sterilization is not achieved. Nevertheless, whether antivirulence approaches exert lower evolutionary pressure than bactericidal drugs remains context dependent and cannot be assumed.

The most important new development may be the expanding recognition that formulation changes biological performance. Free lauric acid, liposomal lauric acid, free GML, albumin-associated GML, nanoemulsions, fibers, and petrolatum-based GML should not be treated as pharmacologically interchangeable. A future medicinal product will be a formulation, not an abstract molecule.

5.2 Why Topical Translation Currently Appears More Plausible than Systemic Translation

A recurring translational barrier for GML is achieving sufficient exposure where infection occurs. Topical delivery has several advantages. High local concentrations can be generated with relatively modest total dose; systemic absorption may be reduced; the formulation can maintain residence time; and the infection site can sometimes be directly accessed. Skin, superficial wounds, nasal mucosa, and device surfaces therefore provide rational early targets. This rationale is consistent with recent analyses of lipid-based nanocarriers for skin infections, which highlight improved solubility, sustained release, and tissue delivery alongside stability, regulatory, and scale-up challenges.⁴⁷

Atopic dermatitis represents one such application. *S. aureus* frequently expands during disease exacerbation and contributes to dysbiosis and inflammatory signaling.⁴⁴ Laowansiri et al. (2025)¹⁶ demonstrated susceptibility of resistant *S. aureus* isolates obtained from atopic dermatitis patients, while Schlievert et al. (2026)¹⁷ demonstrated the feasibility of delivering GML from a petrolatum base. Together these studies justify formulation and human proof-of-concept research. They do not yet demonstrate that GML improves eczema severity, prevents flares, or replaces established anti-inflammatory and antimicrobial management. Recent pediatric cohort evidence also

reinforces the clinical importance of accurately characterizing *S. aureus* skin colonization in atopic dermatitis rather than assuming that colonization and overt infection are interchangeable clinical states.⁴⁸

Chronic and diabetic wounds represent another potentially important target. Chronic wounds frequently contain polymicrobial biofilms, and their treatment is complicated by impaired perfusion, necrotic material, inflammation, and repeated antimicrobial exposure. Biofilm-directed strategies are increasingly regarded as an important complement to debridement, wound-bed preparation, and targeted antimicrobial therapy.⁴⁹ A local GML formulation might therefore function as an adjunct rather than as a stand-alone replacement for wound care.

Device-associated infection is conceptually attractive but more demanding. Biofilm formation on catheters, orthopedic implants, prostheses, and other biomedical surfaces is a major therapeutic challenge, often requiring prolonged antimicrobials or removal of infected hardware.^{8,9} Antimicrobial coatings or GML-releasing materials could theoretically prevent early colonization, but preclinical testing would need to establish release kinetics, mechanical stability, compatibility, cytotoxicity, persistence, and realistic biofilm performance. Wang et al. (2023)⁵⁰ have emphasized that antimicrobial medical-device technologies require rigorous preclinical performance testing rather than reliance on simple zone-of-inhibition assays.

5.3 Biofilm Prevention Should Not Be Conflated with Biofilm Treatment

Many candidate antibiofilm compounds are highly effective when administered during the initial adhesion phase but much less active against mature biofilms. Clinically, these are different indications. A catheter coating designed to prevent initial colonization may be useful even if it cannot eradicate a seven-day mature biofilm. Conversely, a wound treatment requires evidence against established communities.

This distinction should influence future GML study design. Biofilm-prevention studies should examine early adhesion, surface conditioning, and microcolony development. Treatment studies should use mature, physiologically relevant biofilms and measure viable-cell reduction, not biomass alone. Polymicrobial models should be incorporated because *S. aureus*, Gram-negative organisms, and *Candida* frequently coexist in chronic wound and device environments. The literature on *S. aureus* biofilm therapy makes clear that conventional antibiotic susceptibility results often fail to predict performance against organized biofilm communities.^{51,52}

The distinction between prevention and treatment is summarized in **Figure 3**, which maps the different points at which lauric acid or GML could theoretically intervene during biofilm development. The figure also separates early-stage prevention from treatment of established biofilms so that these biologically distinct endpoints are not interpreted as equivalent evidence.

Figure 3: Potential intervention points of lauric acid and GML across the biofilm lifecycle

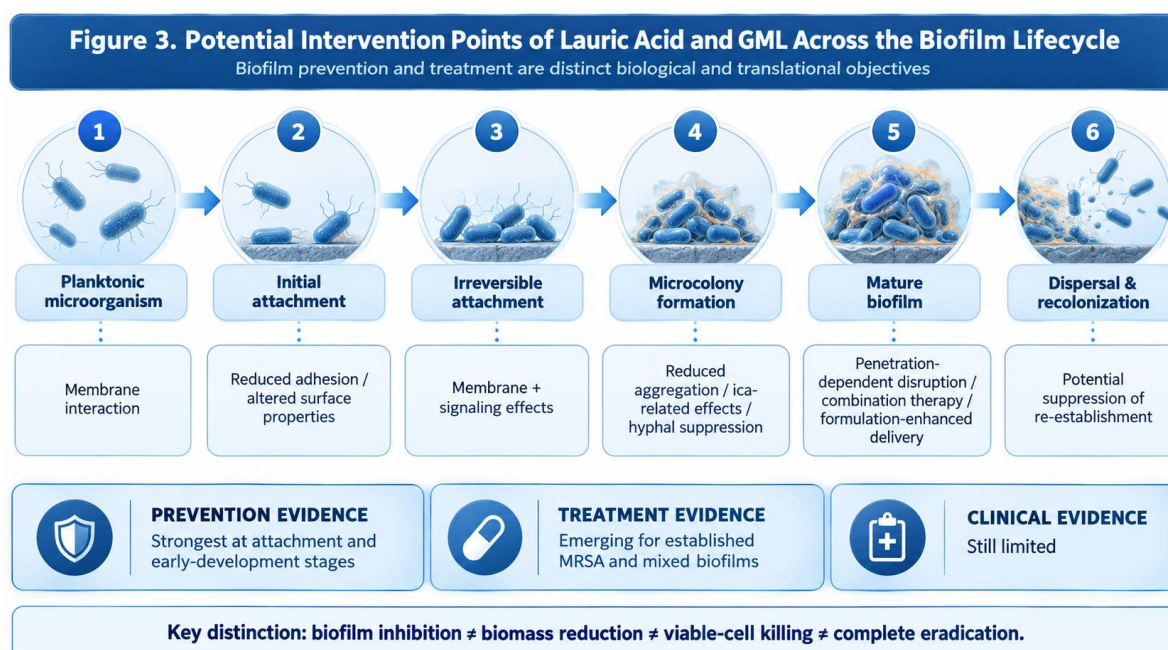


Figure 3 illustrates that a biofilm-active intervention may work at one or several stages. Lauric acid and GML have evidence for inhibiting initial development, reducing virulence-associated processes, and affecting established biofilms in selected models. However, the matrix, metabolic heterogeneity, and altered physiology of mature biofilms create a much higher therapeutic barrier. Future papers should therefore clearly separate biofilm inhibition, biomass reduction, viable-cell killing, and complete eradication.^{4,5,13}

5.4 GML as an Antibiotic Adjuvant

The antibiotic-adjuvant strategy may ultimately prove more feasible than treating GML as a universal stand-alone antibiotic. Combination treatment has three conceptual advantages. First, membrane perturbation can increase bacterial vulnerability to an antibiotic. Second, two mechanistically different stresses may reduce the concentration required for either agent. Third, a biofilm-directed lipid could potentially expose metabolically active or previously protected subpopulations to conventional drugs.

Yet synergy must be assessed rigorously. Fractional inhibitory concentration indices can identify candidate combinations but may vary with assay design. Time-kill experiments provide stronger information about the kinetics of interaction. Mature-biofilm models add another layer. In vivo models are required to determine whether pharmacokinetic exposures overlap sufficiently for synergy to occur in tissue. Finally, clinical benefit must be demonstrated directly rather than inferred from laboratory synergy.

The concept is consistent with broader antimicrobial-fatty-acid research. Sidders et al. (2023)²⁴ showed that membrane changes associated with cell-wall antibiotic activity can create vulnerability to antimicrobial fatty acids. This suggests that rational combinations should be selected mechanistically rather than by screening all possible

antibiotic pairs. Future GML research should investigate whether specific cell-wall, membrane, or metabolic antibiotics create predictable GML susceptibility states.

5.5 Resistance Evolution Must Be Tested Rather than Assumed Away

Membrane-active agents are sometimes described as having low resistance potential because disrupting the bacterial membrane does not depend on a single protein target. This is plausible but incomplete. Bacteria possess extensive capacity to alter membrane fatty-acid composition, surface charge, envelope architecture, efflux, metabolic pathways, and lipid uptake. Exposure to exogenous fatty acids can itself alter susceptibility to antibiotics.¹⁹

Future GML development should therefore include serial-passage experiments, whole-genome sequencing of adapted populations, lipidomic analysis of membrane remodeling, assessment of cross-resistance, and evaluation of fitness costs. Resistance surveillance should also distinguish decreased susceptibility to GML from altered susceptibility to conventional antibiotics. A particularly important question is whether GML adaptation could inadvertently select membrane phenotypes with altered susceptibility to host innate immune lipids.

Thus, the most scientifically defensible statement is not that resistance to GML cannot occur, but that its membrane-associated and multimodal activity may generate a different resistance landscape from conventional antibiotics and deserves systematic evolutionary investigation. Accordingly, resistance evolution should be measured empirically in future GML development rather than inferred solely from its membrane-associated mechanism.^{19,20}

5.6 Pharmacokinetic and Pharmacodynamic Knowledge Gaps

Systemic therapeutic development faces major unanswered questions. For a drug to treat bloodstream or deep-tissue infection, effective concentrations must be achieved at the infection site for an adequate duration. GML may be hydrolyzed by lipases, incorporated into lipid metabolism, bound to circulating proteins, distributed into lipoprotein fractions, or otherwise transformed. These processes could reduce free exposure or, as suggested by the albumin study, potentially modify delivery in less intuitive ways.

Human milk measurements show that GML can be detected in a complex physiological lipid matrix, but presence in human milk cannot be treated as evidence that therapeutic plasma concentrations are achievable.²² Pharmacokinetic studies must determine absorption, bioavailability, plasma protein binding, tissue partitioning, metabolism, and elimination. Pharmacodynamic work should then relate these exposures to MIC, MBC, time-kill, MBIC, and MBEC endpoints.

A topical product has a different PK/PD problem. Rather than plasma concentration, relevant parameters may include concentration in the stratum corneum, wound exudate, follicular units, mucosal secretions, or device surface films; release from petrolatum or hydrogel; penetration into biofilm; and persistence between applications. Translational studies therefore require indication-specific PK/PD models.

5.7 Host Immunology and Microbiome Preservation

GML's immunological effects could become either an advantage or liability. Suppression of excessive T-cell activation might theoretically help inflammatory skin disorders, while inhibition of inflammatory signaling has been observed in experimental intestinal models (Fosdick et al., 2021; Mo et al., 2021; He et al., 2022). However, antimicrobial therapy should not impair necessary host defense, particularly in immunocompromised patients or infected wounds.

Microbiome safety is similarly important. Traditional broad-spectrum antibiotics may disrupt commensal ecosystems, and any proposed GML advantage should be demonstrated experimentally rather than assumed from its natural origin. Studies should compare susceptibility of pathogenic *S. aureus* with common skin commensals, evaluate recovery of microbial diversity after treatment, and examine whether repeated GML exposure alters colonization resistance.

Animal nutrition and intestinal studies already demonstrate that GML can modify microbial community composition.⁴³ That property may be beneficial in some models but confirms that GML is not microbiologically neutral. Clinical development should therefore incorporate microbiome endpoints, especially for repeated topical, mucosal, or oral exposure.

5.8 From Palm Kernel to Pharmaceutical-Grade GML

Palm kernel oil provides an industrially important lauric feedstock, and monolaurin synthesis from palm kernel fractions has been demonstrated through enzymatic glycerolysis (Ngatirah et al., 2022). A biomedical value chain could therefore begin with fractionation of palm kernel oil into lauric-rich fractions, followed by purification, controlled esterification with glycerol, separation of mono-, di-, and triglyceride products, and pharmaceutical purification.

The key translational issue is not simply producing monolaurin but producing a reproducible active pharmaceutical ingredient. Pharmaceutical-grade development requires defined chemical identity, isomer composition, residual glycerol and free fatty acid specifications, solvent and catalyst residues, oxidative stability, microbial limits, contaminant control, and validated analytical procedures. Batch-to-batch reproducibility is essential because antimicrobial activity may be influenced by minor compositional changes.

Feedstock traceability also matters. If palm-kernel-derived GML is promoted as a high-value health product, responsible sourcing, land-use impacts, traceability, and sustainable processing become part of product governance. The biomedical value proposition should therefore be based on verified pharmaceutical quality and sustainability rather than an assumption that natural or plant-derived origin alone establishes superiority.

The complete palm-kernel-to-health pathway and the major translational “gates” are summarized in **Figure 4**. It links feedstock processing, pharmaceutical quality, formulation, preclinical evidence gates, and human development in a single translational sequence.

Figure 4: Palm-kernel biorefinery-to-clinical-translation pathway

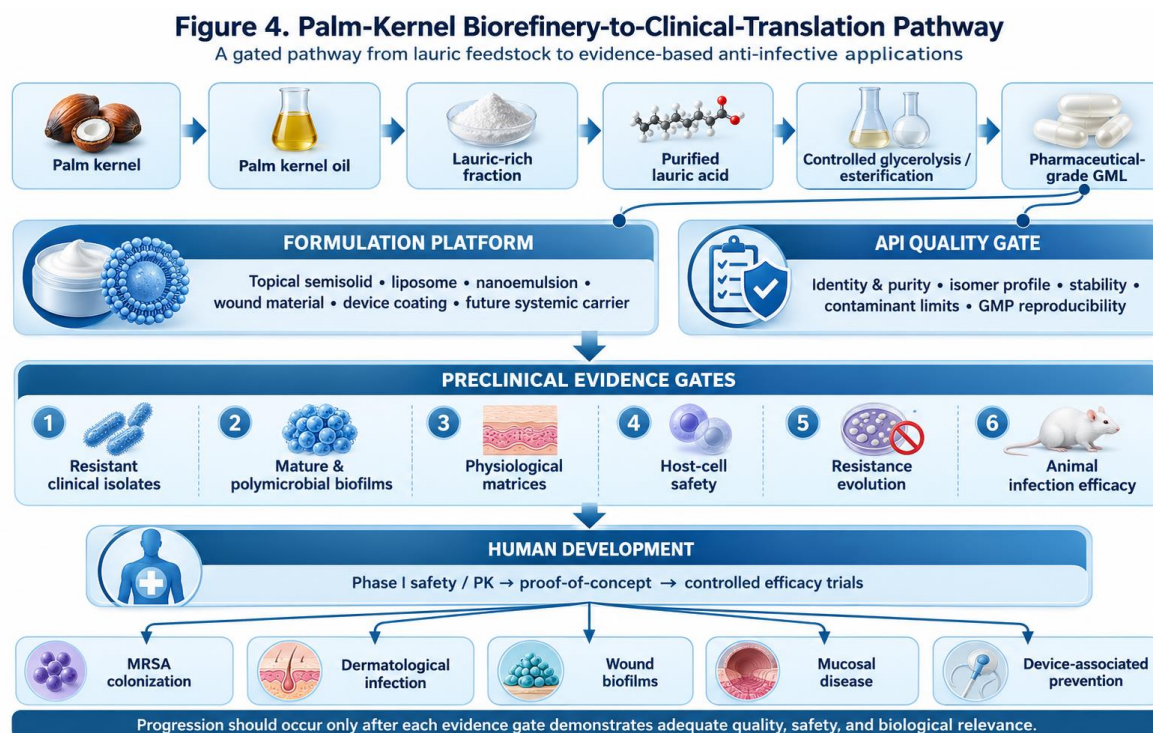


Figure 4 illustrates why demonstration of antimicrobial activity is only an early development step. Feedstock processing must produce a chemically defined pharmaceutical ingredient; formulation must generate effective exposure; preclinical development must demonstrate activity in clinically realistic systems; and human studies must establish safety, dose, and benefit. The pathway also prevents conflation of food-grade monolaurin, experimental GML, and a future pharmaceutical product.

5.9 Priorities for Preclinical and Clinical Development

A rational research agenda can be organized as a sequence of increasingly demanding evidence gates. First, susceptibility testing should use diverse contemporary clinical isolates, particularly MRSA, vancomycin-resistant enterococci, carbapenem-resistant Enterobacterales, and difficult-to-treat *P. aeruginosa*. Because the [Alrabiah et al. \(2025\)](#) findings challenge previous assumptions about Gram-negative inactivity, independent replication is particularly important.

Second, biofilm experiments should progress from prevention models to mature single-species and polymicrobial biofilms. Models incorporating wound exudate, serum proteins, host extracellular matrix, low oxygen, and clinically relevant surfaces would provide more translational information than static polystyrene assays. Reviews of implant-associated infection and antimicrobial surfaces emphasize precisely this need for realistic preclinical conditions ([Kadirvelu et al., 2024](#); [Wang et al., 2024](#)). This emphasis on carrier-mediated penetration and combination delivery is consistent with recent reviews of liposomal and lipid-nanocarrier strategies for recalcitrant biofilms ([Panthi, Fairfull-Smith and Islam, 2024](#); [Ahsan et al., 2024](#)).

Third, formulation screening should occur concurrently with microbiology. Xu et al. (2024) and Ge et al. (2024) illustrate the continued development of stable nanosystems and nanoemulsions containing GML-related components. A pharmaceutical program should compare formulations on release kinetics, stability, cytotoxicity, penetration, antimicrobial activity, scalability, and sterilization compatibility.

Fourth, safety must include more than acute cytotoxicity. Repeated exposure, epithelial-barrier effects, inflammatory signaling, sensitization, wound healing, microbiome disruption, and systemic absorption require evaluation. The immune-modulatory observations in T cells and experimental colitis reinforce the need to understand host effects (Fosdick et al., 2021; Mo et al., 2021).

Fifth, resistance-evolution experiments should run in parallel with efficacy testing. Serial passage at subinhibitory concentration, adaptation in biofilms, genomic sequencing, and lipidomics could identify emerging tolerance mechanisms before human use.

Finally, indication-specific human studies are necessary. For topical applications, a first-in-human program could initially evaluate irritation, sensitization, local microbiome effects, systemic absorption, and reduction of *S. aureus* colonization. Proof-of-concept studies could then evaluate clinical endpoints in carefully selected conditions. Deep systemic infection should represent a later and substantially more demanding development pathway because human PK/PD information is currently inadequate.

5.10 Broader Clinical and Public-Health Interpretation

The attraction of palm-kernel-derived antimicrobial lipids extends beyond discovery of another bactericidal molecule. AMR requires diversification of therapeutic strategies, including antibiotic adjuvants, antibiofilm agents, antivirulence approaches, bacteriophages, immune-based interventions, antimicrobial materials, and improved delivery technologies. Natural and semisynthetic lipids could occupy more than one of these categories simultaneously.

Their value should nevertheless be judged according to the same standards applied to conventional anti-infectives. Natural origin does not remove the need for reproducible chemistry, toxicology, pharmacokinetics, dose optimization, controlled trials, or resistance surveillance. Similarly, the historical use of monolaurin in food or consumer products cannot be used as a substitute for indication-specific clinical safety.

The economic and health implications of biofilms also justify investment in non-conventional approaches. Biofilms create costs across healthcare, industrial systems, and infrastructure, while medical biofilms contribute to device failure, recurrent hospitalization, and prolonged antimicrobial use.⁷ Effective local antimicrobial lipids could therefore have value not only by directly killing organisms but by preventing establishment of difficult-to-eradicate communities.

At present, the evidence supports prioritizing local and adjunctive applications. This conclusion reflects the maturity of the available evidence rather than an assumption that systemic use is impossible. A successful topical GML product could itself provide critical information on human tolerability, microbial response, microbiome effects, resistance evolution, and formulation performance that informs more ambitious applications.

6. CONCLUSION

Lauric acid and glycerol monolaurate occupy a scientifically important intersection between antimicrobial lipid chemistry, membrane biology, biofilm control, antimicrobial resistance, and pharmaceutical formulation. Contemporary evidence supports several substantive conclusions. First, lauric acid and GML can disrupt or modify microbial membranes and thereby influence permeability, membrane potential, signaling, metabolism, virulence, and viability. Second, their biological activity extends beyond planktonic organisms to selected biofilm-forming and antimicrobial-resistant pathogens, with the strongest contemporary evidence involving *Staphylococcus aureus* and MRSA. Third, antibiofilm effects include both inhibition of biofilm development and effects on established biofilms, although these outcomes are highly assay- and concentration-dependent. Fourth, formulation is a decisive determinant of performance: liposomes, nanoemulsions, biological protein carriers, nanofibers, and petrolatum can substantially modify the exposure and activity of antimicrobial lipids.

The evidence does not yet justify treating GML or lauric acid as clinically established alternatives to conventional antibiotics. Controlled therapeutic trials, human PK/PD data, standardized medicinal formulations, microbiome-safety studies, and systematic resistance-evolution experiments remain insufficient. Clinical-isolate studies should therefore be interpreted as an important translational bridge rather than as clinical efficacy evidence.

A rational development strategy should prioritize standardized testing against resistant clinical isolates, mature and polymicrobial biofilms, physiologically realistic matrices, combination therapy, topical formulations, host-cell safety, and longitudinal microbial adaptation. Early clinical translation appears particularly plausible for localized *S. aureus*-associated conditions, wound biofilms, decolonization strategies, and selected antimicrobial surfaces, where high local concentrations can potentially be achieved without requiring systemic exposure.

Palm kernel oil provides a credible lauric-rich industrial feedstock for such development. The opportunity, however, lies not merely in converting palm-kernel lipids into monolaurin, but in establishing a traceable, reproducible, pharmaceutical-grade pathway from feedstock and molecular synthesis through formulation, preclinical validation, and controlled clinical evaluation. If these gaps are addressed rigorously, palm-kernel-derived GML could evolve from an antimicrobial lipid of food and natural-product interest into a scientifically validated adjunctive platform for selected antimicrobial-resistant and biofilm-associated infections.

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