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Palm-Kernel-Derived Lauric Acid and Monolaurin Against Antimicrobial-Resistant Pathogens: Membrane Disruption, Biofilm Control, and the Path to Topical Translation—A Critical Review

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ABSTRACT

Antimicrobial resistance (AMR) is eroding the effectiveness of conventional antibiotics and intensifying interest in antimicrobial agents with alternative mechanisms of action. Lauric acid (LA), a major medium-chain fatty acid in palm kernel oil, and its monoacylglycerol derivative glycerol monolaurate (GML; monolaurin) are promising membrane-active compounds with antibacterial, antibiofilm, and antivirulence properties. This critical qualitative literature review synthesizes evidence published during 2020–2026 concerning the biochemical origin, antimicrobial mechanisms, activity against resistant pathogens, formulation challenges, and translational prospects of LA and GML. Particular attention is given to methicillin-resistant *Staphylococcus aureus* (MRSA), antibiotic-resistant *S. aureus* associated with atopic dermatitis, chronic wounds, biofilms, and antibiotic-adjuvant strategies. Contemporary evidence supports membrane perturbation as a central mechanism, complemented by effects on biofilm formation, virulence pathways, and antibiotic susceptibility. Nevertheless, reported antimicrobial potency varies markedly across studies, reflecting differences in microbial strains, compound preparation, solvent systems, physicochemical state, and susceptibility-testing methods. Emerging hydrogels, nanoemulsions, and petrolatum-based formulations demonstrate how delivery systems may overcome poor aqueous solubility and enable high local concentrations. Limited human evidence supports further investigation of local applications, but robust clinical efficacy trials remain absent. The review concludes that palm-kernel-derived LA and GML merit development primarily as topical, decolonisation, wound-care, and antibiotic-adjuvant platforms, while systemic antimicrobial claims remain premature.

Keywords: Palm kernel oil; Lauric acid; Monolaurin; Glycerol monolaurate; Antimicrobial resistance; MRSA; *Staphylococcus aureus*; Membrane disruption; Biofilm; Antimicrobial stewardship

JEL Classification: I10; I15; I18; O32; Q16

INTRODUCTION

Antimicrobial resistance has evolved from a foreseeable microbiological problem into a defining global public-health challenge. The landmark global analysis for 2019 estimated approximately 1.27 million deaths directly attributable to bacterial AMR and almost 5 million deaths associated with resistant bacterial infections, demonstrating that resistance already produces a mortality burden comparable with many major communicable diseases. Subsequent Global Burden of Disease analyses have strengthened rather than weakened this concern. The 1990–2021 analysis estimated that 1.14 million deaths in 2021 were attributable to bacterial AMR and projected that the burden could remain substantial through 2050 without stronger prevention, access, stewardship, and innovation. *Staphylococcus aureus* is especially important because it combines widespread human colonisation, a remarkable capacity to cause invasive and superficial infections, virulence diversity, biofilm formation, and repeated acquisition of antimicrobial resistance[1–3].

The contemporary AMR problem is not simply a shortage of individual antibiotics. It reflects an evolutionary and ecological system in which microorganisms can modify drug targets, produce drug-inactivating enzymes, reduce intracellular drug concentrations, remodel cell envelopes, alter metabolic states, exchange resistance genes, form tolerant biofilms, and persist in host-associated reservoirs. The updated World Health Organization bacterial priority-pathogen framework consequently continues to prioritize methicillin-resistant *S. aureus* (MRSA) among pathogens requiring sustained research and development. The broader ESKAPE framework similarly identifies *S. aureus* as one of the organisms central to the clinical AMR problem[4–6].

The limitations of conventional antibacterial discovery also justify considering mechanisms beyond traditional single-protein targets. Resistance to a protein-targeting antibiotic can sometimes emerge through mutation, target modification, enzyme production, efflux, or pathway bypass, although the probability and consequences vary markedly among drug classes. At the same time, Gram-negative penetration barriers and stringent requirements for target essentiality complicate antibacterial discovery, illustrating why genuinely novel therapeutic concepts are difficult to translate into useful drugs[7,8]. Membrane-active compounds are therefore attracting renewed attention because bacterial membranes are essential, spatially extensive, dynamic targets that cannot simply be deleted without severe fitness consequences. This does not make membrane-active agents resistance-proof, but it provides an alternative design space for antimicrobial development[9,10]. This need has also broadened the antibacterial-development landscape toward non-traditional strategies that complement or bypass conventional antibiotic mechanisms, including approaches aimed at virulence, microbial ecology and alternative cellular vulnerabilities[11].

Naturally occurring fatty acids and monoacylglycerols are particularly interesting in this context. Their amphiphilic structures permit self-assembly and interaction with phospholipid membranes, while chain length, saturation, ionization, headgroup chemistry, concentration, temperature, and surrounding medium influence antimicrobial activity. Among them, lauric acid (dodecanoic acid; C12:0) and glycerol monolaurate, commonly called monolaurin, have long attracted interest because of their activity against multiple microorganisms, particularly Gram-positive bacteria. Recent literature has moved beyond generic descriptions of “natural antibacterial compounds” toward

molecular understanding of lipid self-assembly, membrane perturbation, bacterial adaptation, antibiofilm properties, and delivery technologies[12–14].

Palm kernel oil creates an additional translational opportunity because it is a lauric-rich vegetable lipid. Palm kernel fractions can provide substrate for production of lauric-acid-derived monoacylglycerols, and experimental enzymatic glycerolysis of palm-kernel olein and stearin has demonstrated the feasibility of producing monolaurin-rich fractions with antibacterial activity. This establishes a scientifically plausible bridge between palm-kernel processing and antimicrobial-lipid innovation[15]. Importantly, however, the biochemical identity of monolaurin must be distinguished from feedstock provenance. An antimicrobial study using laboratory-synthesized, coconut-derived, or commercially purchased GML demonstrates the biological activity of GML, but it does not demonstrate that the tested compound was palm-kernel-derived. A rigorous palm-health narrative should therefore frame palm kernel as a potentially scalable renewable feedstock rather than retroactively characterise all GML evidence as palm-specific.

Recent findings make this distinction especially relevant. Monolaurin has shown activity against MRSA and other antibiotic-resistant *S. aureus* isolates, including organisms recovered from wounds and from children with atopic dermatitis. A 2025 study reported inhibition of MRSA, mupirocin-resistant, and fusidic-acid-resistant *S. aureus* at low concentrations, whereas 2024 studies using other clinical isolate collections reported MICs in the hundreds to thousands of micrograms per millilitre. These data provide evidence for antimicrobial activity but simultaneously expose substantial methodological heterogeneity that must be understood before clinically relevant dosing can be inferred[16–18].

The translational evidence has also become more diverse. A human proof-of-concept study reported substantial reduction of nasal *S. aureus* following application of a nonaqueous 5% GML gel, while a 2026 investigation demonstrated antibacterial activity of GML formulated in petrolatum against methicillin-sensitive and methicillin-resistant *S. aureus* and evaluated a high-concentration formulation in a rabbit dermatitis model. Separately, lauric-acid-containing hydrogels have shown antibacterial and wound-healing effects in experimental wound models, including MRSA-infected diabetic wounds[19–21]. These developments shift the research question from whether LA and GML possess antimicrobial activity to whether their biological effects can be converted into safe, reproducible and clinically meaningful formulations.

Accordingly, this review critically examines five linked questions: how LA and GML exert antimicrobial effects; how compelling the contemporary evidence is against resistant pathogens; whether antibiofilm and antibiotic-adjuvant properties expand their therapeutic relevance; why antimicrobial potency varies markedly between studies; and which applications are sufficiently mature to justify translational development. The review particularly examines topical dermatological, nasal-decolonisation and wound-care applications because these settings offer a mechanistic advantage: relatively high local concentrations can be achieved without first proving systemic bioavailability, distribution or pharmacokinetic performance. It therefore intentionally distinguishes biological

plausibility from clinical evidence and avoids inferring systemic therapeutic efficacy from in vitro antimicrobial activity.

LITERATURE REVIEW: CONCEPTUAL AND THEORETICAL FOUNDATIONS

Lauric acid, monolaurin and the palm-kernel biochemical pathway

Lauric acid is a saturated 12-carbon medium-chain fatty acid possessing a hydrophobic alkyl chain and a carboxyl headgroup. Glycerol monolaurate is the monoester produced by esterification of lauric acid with glycerol. Although the two molecules share a C12 hydrophobic tail, their headgroups differ substantially, influencing charge state, interfacial behaviour, aggregation and membrane interactions. Their antimicrobial function should therefore not be treated as interchangeable. Contemporary analyses of medium-chain fatty acids and monoglycerides emphasize that antimicrobial behaviour emerges from physicochemical interactions among molecular structure, self-assembly, surrounding solution and target membrane composition[13,14].

Palm kernel is relevant because palm kernel oil contains a high proportion of lauric-type fatty acids and can be fractionated into olein- and stearin-rich streams. Ngatirah et al.[15] demonstrated enzymatic glycerolysis of palm-kernel olein–stearin blends to produce monolaurin and assessed the product as both an emulsifier and antibacterial compound. Such work is important for two reasons. First, it demonstrates technical feasibility for transforming a palm-kernel fraction into a higher-value functional lipid. Second, it establishes a potential connection between oleochemical processing and antimicrobial development. However, biomedical-grade production would require much tighter control of purity, positional isomers, residual catalysts or enzymes, contaminants, oxidation products, batch consistency and formulation than may be necessary for food or technical applications.

Amphiphilicity, self-assembly and membrane targeting

The antimicrobial activity of lipid amphiphiles is closely linked to their ability to partition into biological interfaces. Medium-chain fatty acids and monoglycerides can form micelles, vesicles or other supramolecular assemblies depending on concentration and environmental conditions. Their interactions with model and biological membranes may cause lipid packing defects, altered membrane fluidity, curvature stress, leakage and loss of electrochemical homeostasis. These processes differ fundamentally from the lock-and-key inhibition of a single bacterial enzyme and can involve multiple simultaneous physical disruptions[9,14].

Lauric acid activity against *S. aureus* has recently been supported by direct morphological and membrane-integrity observations. Liu et al.[22] reported growth inhibition, altered cell morphology, disruption of membrane integrity and changes consistent with membrane depolarization or fluidity alteration. Laurate-containing derivatives have likewise demonstrated membrane-disruptive antibacterial effects, showing that the C12 moiety can contribute importantly to antimicrobial activity when incorporated into more complex molecules[23]. In 2026, Ma et al. further reported that lauric-acid microemulsions inhibited *S. aureus* through membrane disruption with possible effects related to peptidoglycan biosynthesis. These findings support a multilevel mechanism rather than an exclusively detergent-like explanation.

The membrane concept nevertheless requires qualification. The bacterial envelope is not a passive phospholipid bag. Gram-positive bacteria possess thick peptidoglycan layers, teichoic acids, surface proteins and dynamically regulated lipid compositions, while Gram-negative bacteria possess an additional outer membrane that presents a formidable permeability barrier. These architectural differences help explain why free fatty acids and monoglycerides frequently show stronger activity against Gram-positive organisms. Moreover, bacteria can adapt membrane composition, deploy efflux systems, detoxify antimicrobial lipids, incorporate exogenous fatty acids or modify metabolic pathways in response to lipid stress[10,24]. The correct theoretical proposition is therefore not that membrane-active agents cannot generate resistance, but that they impose a different and potentially multifactorial adaptive challenge.

Biofilm tolerance and the importance of phenotype

AMR measured in planktonic cells represents only part of the therapeutic problem. Biofilms are structured microbial communities embedded within extracellular matrices and characterised by spatially heterogeneous metabolism, diffusion gradients, altered gene expression, persister populations and protection from host immunity. These properties can produce antimicrobial tolerance without conventional resistance mutations, while biofilms may also facilitate the emergence and persistence of resistant populations[25]. MRSA combines genetic resistance with particularly important biofilm-associated protection, making antibiofilm activity a valuable attribute for a potential topical antimicrobial[26]. Recent synthesis of biofilm-associated AMR further shows that matrix effects, altered physiology, stress responses and population heterogeneity interact to reduce antimicrobial susceptibility, reinforcing the need to evaluate candidate agents in sessile as well as planktonic states[27].

Fatty acids can influence biofilm behaviour at concentrations that differ from those needed for planktonic killing. Lauric and related fatty acids have reduced mono- and polymicrobial biofilm formation, altered virulence-associated gene expression, and in some systems increased the effectiveness of conventional antibiotics. Kim et al.[28], for example, demonstrated that lauric and myristic acids inhibited polymicrobial biofilms involving *S. aureus*, *Escherichia coli* and *Candida albicans* and affected virulence-associated pathways. Other fatty-acid studies show that antibiofilm and antivirulence properties are not unique to lauric acid but may represent a broader functional feature of appropriately structured lipids[29,30].

***Staphylococcus aureus* as the principal translational target**

Staphylococcus aureus is an unusually appropriate model for evaluating LA/GML translation because it occupies a continuum from asymptomatic colonisation to severe invasive disease. Its clinical success reflects immune evasion, metabolic versatility, toxin production, adhesins, biofilm formation, genomic plasticity and the ability to adapt to specific host niches[31]. MRSA is consequently not simply a susceptible *S. aureus* strain with one altered antibiotic target; it exists within complex lineages and ecological settings that influence transmission, colonisation, virulence and treatment response[5].

This complexity is especially important in skin disease. Dermatology involves substantial use of systemic and topical antimicrobials, generating concern about selection pressure on both pathogenic organisms and commensal microbiota. Reviews of antimicrobial resistance in dermatology have therefore emphasized stewardship, reduced unnecessary antibiotic exposure and development of treatment strategies that avoid prolonged nonspecific antimicrobial pressure[32–35].

Atopic dermatitis as a high-value application model

Atopic dermatitis (AD) provides a biologically compelling but clinically complex environment for local antimicrobial development. Barrier dysfunction, altered immune signalling, increased skin pH, reduced microbial diversity and *S. aureus* overgrowth interact in ways that can exacerbate inflammation. Importantly, colonisation and overt infection are not synonymous. Treating every culture-positive patient with an antimicrobial could therefore increase selection pressure without necessarily improving disease[36,37]. A 2026 disease primer similarly characterises AD as a heterogeneous inflammatory disorder in which barrier dysfunction, type 2 immune responses and microbial dysbiosis interact, underscoring why antibacterial activity alone cannot be assumed to translate into disease control[38]. Contemporary microbiome research has strengthened the association between *S. aureus* dominance and AD severity while also illustrating the ecological complexity of the disease. Reviews describe interactions among epidermal barrier function, microbial community structure, bacterial virulence factors and host immunity, while longitudinal genomic research has demonstrated within-person evolution of *S. aureus* during AD treatment[39–42]. Experimental work has further connected *S. aureus* colonisation with inflammatory pathways such as NLRP1-associated inflammasome activation[43].

The antimicrobial dimension is equally important. A meta-analysis of pediatric AD found substantially increased *S. aureus* colonisation in eczematous skin and nasal sites and documented MRSA among isolates, while a global systematic review demonstrated clinically relevant antimicrobial resistance patterns among *S. aureus* from AD populations[44,45]. These observations provide a rational context for nontraditional topical agents, especially where repeated fusidic acid, mupirocin or systemic antibiotic exposure is undesirable.

Decolonisation and resistance to established topical agents

Mupirocin has been a cornerstone of *S. aureus* nasal decolonisation, but resistance is well documented. A systematic review and meta-analysis showed that mupirocin resistance is globally distributed, and later surveillance work identified substantial resistance among some contemporary MRSA collections[46,47]. This does not imply that mupirocin should be displaced indiscriminately; rather, it demonstrates why alternative or adjunctive decolonisation approaches warrant continued investigation. Contemporary decolonisation literature likewise emphasises that *S. aureus* carriage is an important reservoir for subsequent infection and that rising resistance to decolonisation agents strengthens the case for carefully evaluated non-antibiotic or adjunctive approaches[48].

GML is particularly interesting in this context because local administration can produce concentrations far above those that would plausibly be achieved systemically. A 2020 proof-of-concept study using a 5% GML nonaqueous gel in human anterior nares reported substantial reduction of *S. aureus* and no serious tolerability signal in the study setting[19]. This evidence remains preliminary, but it creates a rare bridge between in vitro lipid antimicrobial research and direct human local application.

METHODS

Review design

This article used a critical qualitative literature review rather than a systematic literature review or meta-analysis. The purpose was interpretive synthesis: to integrate mechanistic, microbiological, formulation, dermatological, wound-healing, translational and public-health evidence into a coherent assessment of where palm-kernel-derived LA and GML currently stand as candidate interventions against AMR pathogens. Accordingly, the method prioritised conceptual relevance, scientific quality, recency and explanatory power rather than statistical completeness.

A systematic review typically begins from a tightly bounded question, uses a prespecified reproducible search protocol, applies formal inclusion/exclusion procedures to all retrieved records and frequently includes risk-of-bias assessment or quantitative synthesis. That approach would be poorly matched to the present objective because the evidence spans colloid and interface science, food microbiology, bacterial physiology, clinical microbiology, biomaterials, dermatology, wound models, AMR epidemiology and pharmaceutical formulation. The review therefore used thematic integration and critical comparison while explicitly avoiding claims that the literature corpus was exhaustive.

Literature identification

Priority was given to peer-reviewed journal articles published from 2020 through 29 August 2026. Searches centred on combinations of terms including “lauric acid”, “monolaurin”, “glycerol monolaurate”, “palm kernel”, “antimicrobial resistance”, “MRSA”, “*Staphylococcus aureus*”, “membrane disruption”, “biofilm”, “antibiotic synergy”, “atopic dermatitis”, “wound”, “hydrogel”, “nanoemulsion”, “petrolatum”, “decolonisation” and “mupirocin resistance”. PubMed-indexed literature and publisher journal platforms were prioritised, supplemented by citation chaining from highly relevant articles.

Direct LA/GML studies were given the highest substantive weight. Contextual literature was included when required to interpret mechanisms, resistance biology, biofilms, AD, dermatological stewardship, wound ecology or

global AMR relevance. Recent authoritative reviews were used to establish conceptual frameworks, while claims concerning antimicrobial potency, formulations and therapeutic effects were preferentially grounded in primary experimental studies.

Evidence extraction and critical comparison

For direct antimicrobial studies, the synthesis considered compound identity, microbial species and strain, resistance phenotype, concentration, MIC/MBC or other microbiological endpoint, solvent or carrier, biofilm methodology, antibiotic combinations, host-cell effects and experimental model. Particular attention was paid to studies whose results appeared discordant. For example, differences between low-μg/mL monolaurin MICs reported by Laowansiri et al.[18] and substantially higher MIC ranges reported by Hassan Abd El-Ghany et al.[16,17] were treated as scientifically informative heterogeneity rather than resolved through narrative averaging.

Evidence was interpreted through a translational hierarchy: physicochemical mechanisms; laboratory reference-strain microbiology; resistant clinical isolates; biofilm and tissue-relevant models; animal/local-delivery models; human local-use evidence; and systemic human antimicrobial therapy. This hierarchy enabled the review to distinguish between a molecule that kills bacteria in vitro and a therapeutic intervention that has demonstrated clinically meaningful benefit.

Thematic synthesis

The literature was organised into six interrelated domains: feedstock and biochemical production; membrane-centred mechanism; activity against AMR organisms; antibiofilm and combination effects; delivery and host-interface considerations; and translation to clinical/public-health applications. Cross-cutting attention was given to evidence quality, resistance evolution, formulation dependence, microbiome consequences and claim proportionality. This approach has limitations. Qualitative selection can introduce reviewer judgement, the field is expanding rapidly, and direct comparison is complicated by diverse microbial strains and assay systems. Furthermore, much LA/GML research originates in food, biomaterials or basic microbiology rather than controlled clinical therapeutics. These limitations are not reasons to disregard the evidence; they are reasons to interpret it according to the level of biological and clinical inference each study can support.

RESULTS: THEMATIC FINDINGS

Theme 1: Palm kernel provides a credible feedstock platform—but not proof of clinical efficacy

The literature supports a clear biochemical pathway from palm-kernel-derived lauric lipids to monolaurin production. Enzymatic glycerolysis offers one route for increasing monoacylglycerol yield, and the work of Ngatirah et al.[15] demonstrates that palm-kernel olein–stearin fractions can serve as monolaurin substrates. From an industrial perspective, this suggests an opportunity to move from commodity lipid processing toward higher-value specialty ingredients.

The biomedical implication, however, requires an explicit provenance rule. Chemically equivalent high-purity GML should be expected to possess the same molecular structure irrespective of whether its laurate was obtained from palm kernel, coconut, synthetic chemistry or another source, assuming no meaningful differences in impurities or isomer distribution. Consequently, evidence demonstrating antimicrobial GML activity can support a *molecular* development case, whereas a *palm-kernel-specific* development case additionally requires manufacturing, purification, equivalence, contaminant-control and life-cycle evidence.

Figure 1 illustrates why the antimicrobial-development pathway should be treated as a sequence of evidence gates rather than as a direct leap from palm kernel oil to a clinically effective antimicrobial. The sequence also makes explicit that manufacturing feasibility, antimicrobial efficacy, host compatibility and clinical benefit are separate evidentiary questions that must each be satisfied before translation.

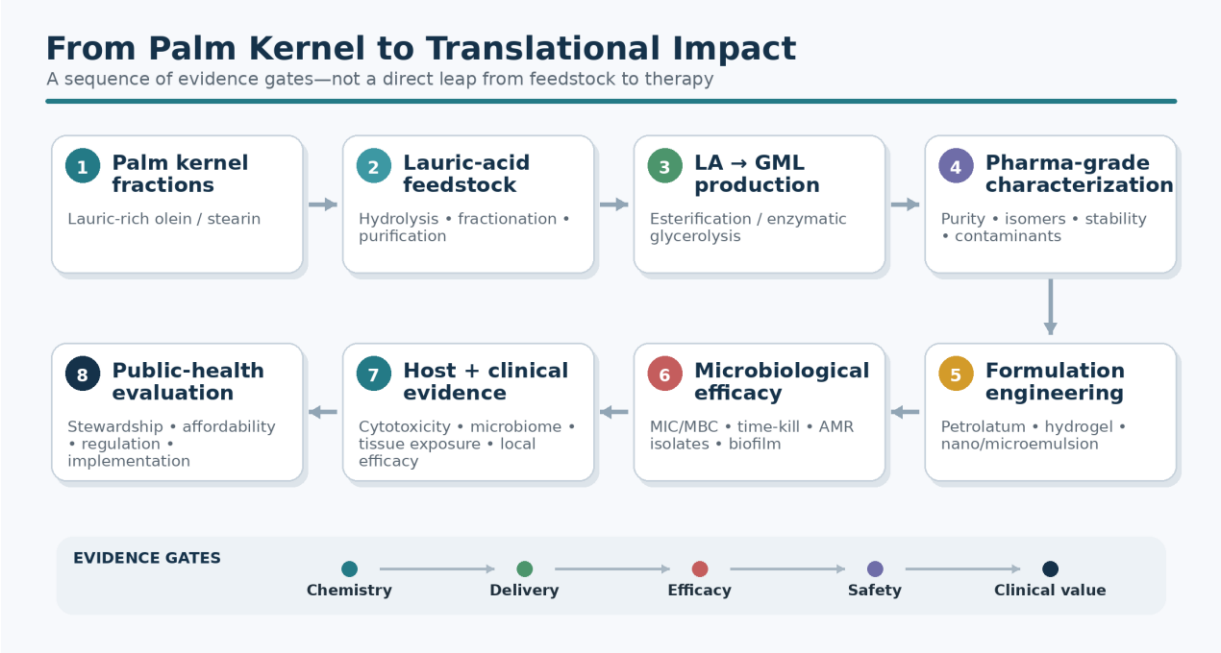

Figure 1: *Proposed palm-kernel-to-clinic translational pathway for lauric acid and monolaurin. Source: conceptual synthesis by the author based on Ngatirah et al.*[15]*, Casillas-Vargas et al.*[13]*, Yoon and Jackman*[14]*, Laowansiri et al.*[18]*, Yang et al.*[20]*, and Schlievert et al.*[21]*.*

Figure 1 emphasizes that feedstock abundance alone does not establish pharmaceutical value. Each transition requires evidence: chemical standardization must precede reproducible formulation; formulation must provide biologically active exposure; antimicrobial activity must occur at concentrations compatible with host tissues; and successful local efficacy must ultimately demonstrate benefit over existing approaches. This framework prevents a common translational error in natural-product research: equating source availability with therapeutic readiness.

Theme 2: Membrane perturbation is the strongest unifying mechanism

The most consistent mechanistic interpretation of LA/GML activity involves interactions with bacterial membranes. Lauric acid can integrate into lipid interfaces and disturb membrane organisation; above appropriate concentration thresholds, this can produce increased permeability, altered fluidity, depolarization, leakage and impaired energetic homeostasis. Liu et al.[22] directly observed morphological and membrane-integrity changes in *S. aureus* following LA treatment. Likewise, recent formulation research suggests that effective dispersion of lauric acid can facilitate membrane interaction and potentially compound the effect through interference with cell-envelope processes[49].

A membrane-centred mechanism also provides a plausible explanation for concentration-dependent antimicrobial activity. Below a threshold, lipid molecules may alter signalling or virulence without producing lethal membrane failure; above it, accumulation can destabilize membrane architecture sufficiently to impair viability. Broader fatty-acid research shows that chain length, saturation and headgroup chemistry influence antibacterial activity, while the surrounding colloidal environment affects the concentration of monomeric or aggregated lipid available to interact with membranes[13,14].

This framework does not exclude secondary mechanisms. Membrane disruption can affect membrane-associated enzymes, transporters, electron transport, cell-wall precursor handling and signalling systems. Altered membrane state may therefore produce downstream changes in metabolism, gene expression and antibiotic penetration. Lauric-acid derivatives have likewise produced membrane-mediated antibacterial effects, reinforcing the structural importance of the lauryl moiety[23].

As summarized in Figure 2, LA/GML antimicrobial action is best considered a cascading physicochemical process in which membrane interaction initiates several downstream effects rather than a single isolated molecular lesion. This representation distinguishes primary membrane effects from downstream biological consequences that may vary with concentration, formulation and bacterial context.

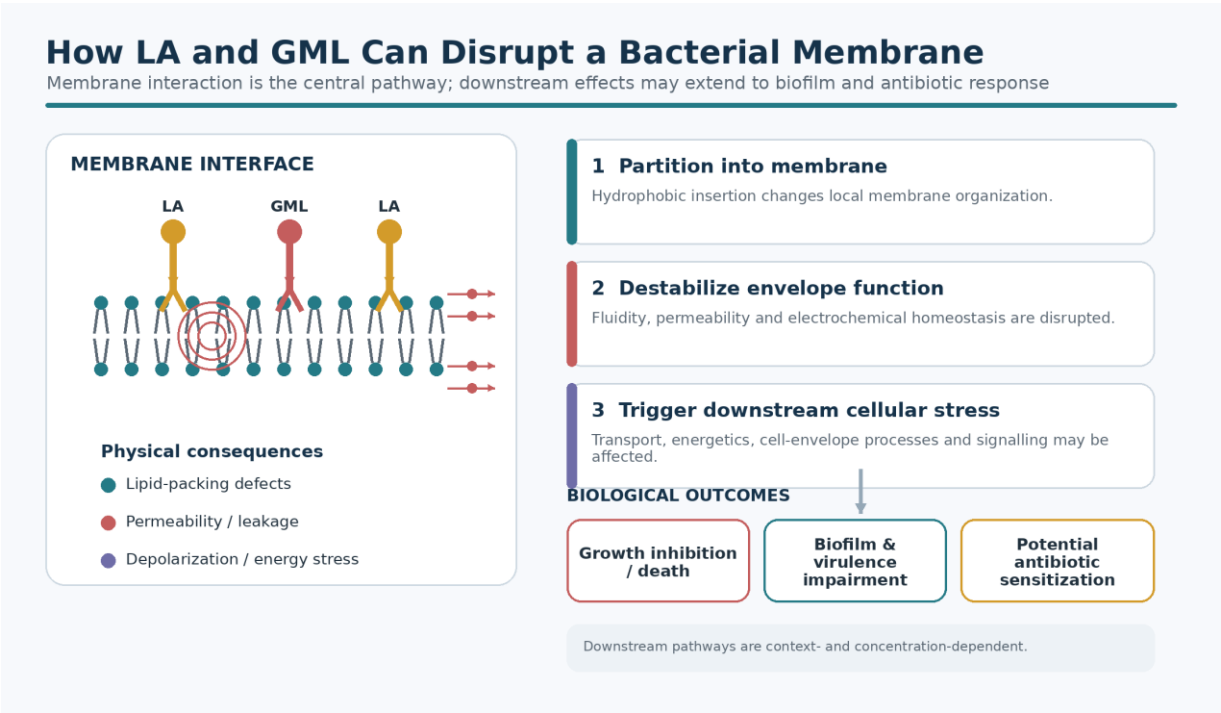

Figure 2: *Mechanistic model linking physicochemical membrane interaction with antimicrobial, antibiofilm, and potential antibiotic-adjuvant outcomes. Source: conceptual synthesis by the author based on Mehta et al.*[9]*, Casillas-Vargas et al.*[13]*, MacDermott-Opeskin et al.*[10]*, Liu et al.*[22]*, Yoon and Jackman*[14]*, and Ma et al.*[49]*.*

The figure also highlights why membrane activity should be evaluated using orthogonal methods. MIC alone cannot establish mechanism. Electron microscopy, membrane-potential assays, leakage measurements, lipidomics, transcriptomics and direct membrane-biophysics experiments should be integrated to distinguish lethal membrane injury from secondary cellular stress.

Theme 3: Activity against resistant *S. aureus* is convincing, but potency estimates are highly heterogeneous

The strongest contemporary anti-AMR evidence for monolaurin concerns *S. aureus*. Hassan Abd El-Ghany et al.[17] examined clinical *S. aureus* isolates and reported monolaurin MICs from approximately 250 to 2,000 µg/mL. The study also reported morphological changes and reduced *blaZ* expression, together with synergistic effects when monolaurin was combined with β-lactam antibiotics. In a related investigation of MRSA wound isolates, monolaurin MICs ranged from approximately 500 to 2,000 µg/mL, and the compound inhibited biofilm formation and reduced expression of the biofilm-associated *icaD* gene[16].

By contrast, Laowansiri et al.[18] reported MICs of approximately 1–2 µg/mL against reference *S. aureus*, MRSA, mupirocin-resistant *S. aureus* and fusidic-acid-resistant isolates, with bactericidal concentrations near 2 µg/mL for the resistant strains evaluated. These concentrations differ by two to three orders of magnitude from some earlier

reports. The authors themselves recognized this discrepancy and proposed possible roles for monolaurin source, synthesis and purification, bacterial susceptibility and assay conditions.

This variation is too large to be dismissed as routine biological noise. It is arguably one of the most important findings of the contemporary literature. Possible explanations include differences in GML purity and isomer composition; solvents; aggregation or precipitation; concentration of bioavailable monomer; agar versus broth methodology; surfactants or emulsifiers; inoculum; medium composition; strain genotype; growth phase; incubation time; and endpoint interpretation. The food-formulation literature provides direct support for the importance of physicochemical state: Xu et al.[50] showed that nanoemulsion incorporation altered the effective antimicrobial performance of GML, whose poor aqueous solubility otherwise constrained activity.

The implication is that the field needs standardised susceptibility-testing procedures specifically adapted to hydrophobic antimicrobial lipids. Conventional MIC methodologies were developed largely for water-soluble or readily dispersible antimicrobials. Without transparent reporting of solvent controls, dispersion state, actual dissolved concentration, vehicle composition and compound characterisation, nominal concentrations may not represent comparable exposures.

4.4 Theme 4: Biofilm activity substantially strengthens the development case

MRSA biofilms are important because they combine genetic drug resistance with physiological tolerance. Within biofilms, slower-growing cells, nutrient gradients, extracellular matrix and persister states can reduce antimicrobial effectiveness and facilitate recurrence[25,26]. Therefore, an intervention capable only of inhibiting planktonic cells would address a limited portion of clinically difficult *S. aureus* infections.

Monolaurin appears to have activity against both biofilm development and established MRSA biofilms. Hassan Abd El-Ghany et al.[16] reported concentration-dependent effects on biofilm-forming clinical MRSA isolates, including reduced *icaD* expression and structural disruption. These findings are consistent with broader lipid research demonstrating that fatty acids can influence adhesion, biofilm-associated signalling and virulence at concentrations below those required for complete planktonic killing.

Lauric acid likewise has antibiofilm properties. Kim et al.[28] found that lauric acid inhibited polymicrobial biofilm formation involving *S. aureus, E. coli* and *C. albicans*, with transcriptomic changes in biofilm- and virulence-associated genes. Importantly, the activity of antimicrobial lipids cannot be reduced to bactericidal effects alone. Fatty acids may modify community behaviour and virulence expression at subinhibitory concentrations, as illustrated by research on other structurally related fatty acids against *S. aureus*[29].

This creates an attractive conceptual distinction between "killing" and "disarming". Complete bacterial eradication may impose strong selective pressure, whereas antibiofilm or antivirulence effects could potentially reduce pathogenicity or improve antibiotic accessibility. Whether LA/GML can exploit this distinction clinically remains unknown, but it should be incorporated into future dose-response research.

Theme 5: Antibiotic potentiation may be as important as stand-alone killing

One of the most strategically important findings is the potential of antimicrobial lipids to function as antibiotic adjuvants. In the 2024 *BMC Infectious Diseases* study, monolaurin combined with β-lactams reduced antibiotic MIC values across numerous *S. aureus* isolates and produced synergistic effects in fractional inhibitory concentration and time-kill analyses[17]. Similar combination benefits were observed in MRSA wound isolates[16].

The concept is biologically plausible. Membrane perturbation may increase drug entry, change localization of membrane-associated resistance processes, alter energetic requirements for efflux, influence expression of resistance genes or generate physiological states in which conventional antibiotics recover activity. Broader fatty-acid research supports the principle that lipid compounds can potentiate antibiotics. Park et al.[30], for example, showed that selected fatty acids enhanced aminoglycoside activity against *S. aureus*, including antibiofilm effects.

Combination therapy could offer several advantages over attempting to develop monolaurin as a replacement antibiotic. It may lower the concentration required for either component, expand activity against biofilm-associated cells, restore susceptibility in selected phenotypes, and enable use of existing drugs with well-characterised pharmacology. Nevertheless, synergy demonstrated in checkerboard or time-kill assays does not guarantee improved clinical efficacy. Protein binding, tissue composition, local pH, wound exudate and bacterial density may profoundly alter interactions in vivo.

Theme 6: Atopic-dermatitis isolates create an important clinical bridge

The 2025 investigation by Laowansiri et al. represents a significant advance because the study linked clinically obtained *S. aureus* from children with AD, antibiotic-resistance phenotyping and monolaurin susceptibility. Resistant phenotypes included MRSA, mupirocin-resistant and fusidic-acid-resistant *S. aureus*. At inhibitory concentrations, monolaurin did not produce detectable toxicity in the primary human keratinocyte and dermal fibroblast assays reported, although toxicity increased at higher concentrations and the calculated selectivity margin remained modest[18].

The correct interpretation is important. The study demonstrated activity **against isolates from patients**; it did not demonstrate **therapeutic efficacy in patients**. No conclusion should therefore be drawn that monolaurin improves eczema severity, eradicates infection in vivo, prevents AD flares or replaces clinically established treatments.

Nevertheless, this evidence is translationally valuable because it tests organisms with real clinical resistance phenotypes rather than relying solely on laboratory reference strains.

The significance is reinforced by the AD microbiome literature. *S. aureus* overrepresentation is strongly associated with AD lesions and severity, while microbial dysbiosis interacts with barrier and inflammatory processes[36,39,41]. A pediatric meta-analysis found markedly increased odds of *S. aureus* colonisation on eczematous skin relative to controls and identified MRSA in both nasal and skin sites[44]. Elizalde-Jiménez et al.[45] further documented the global relevance of antimicrobial susceptibility patterns in AD-associated *S. aureus*.

At the same time, *S. aureus* should not be viewed as a static target. Key et al.[40] demonstrated within-person adaptive evolution during AD treatment, emphasizing that host-associated *S. aureus* populations change over time. Vaher et al.[43] linked *S. aureus* colonisation with enhanced inflammasome-related activity, further illustrating the interaction among bacterial presence, strain properties and inflammatory biology. Future monolaurin trials therefore need endpoints beyond culture negativity, including microbiome diversity, inflammatory markers, barrier restoration and clinical disease severity.

Theme 7: Wound applications provide another strong route for local translation

Chronic and diabetic wounds are attractive targets for membrane-active lipid formulations because high concentrations can be maintained locally and because biofilms and resistant organisms are common. MRSA is especially relevant in chronic wounds, where bacterial burden interacts with inflammation, impaired vascularization and defective tissue repair. Surveillance and meta-analytic literature confirms that resistant *S. aureus* remains an important problem in diabetic-foot and chronic-wound settings[51]. This rationale is consistent with contemporary wound-microbiome evidence showing that *S. aureus*, *Pseudomonas aeruginosa*, polymicrobial interactions and biofilm formation can impair repair and complicate antimicrobial therapy[52].

Lauric-acid biomaterials provide proof that delivery design can integrate antimicrobial effects with wound-healing functions. Yang et al.[20] developed an LA-containing gelatin/hyaluronic-acid composite hydrogel and reported antibacterial, anti-inflammatory and reparative effects in an MRSA-infected diabetic wound model. The work is notable because the delivery matrix was designed not merely to release an antimicrobial but also to interact with the wound environment and immune-regenerative processes.

Balavigneswaran et al.[53] similarly developed a pH-responsive self-healing hydrogel for controlled LA release and reported accelerated wound healing in rats together with immunomodulatory effects. These studies illustrate a broader shift in wound therapeutics: the relevant product is not simply “lauric acid”, but a biomaterial–drug system whose physical structure, adhesion, release profile, moisture management and biological interactions determine performance.

This distinction also helps avoid overgeneralization. Chitosan-based and other multifunctional wound hydrogels can themselves exhibit antibacterial or regenerative properties, showing that carrier materials are active components rather than inert packaging[54]. Therefore, efficacy observed with an LA-containing hydrogel must be decomposed experimentally to distinguish the contribution of LA from that of the matrix.

Theme 8: Formulation is a determinant of efficacy rather than a downstream technical detail

Hydrophobicity is one of the central obstacles facing LA/GML development. A compound that is highly active when directly exposed to a membrane may perform poorly if it aggregates, precipitates or partitions into a carrier that prevents sufficient free concentration from reaching bacteria. This creates an apparent paradox in which higher nominal concentration does not necessarily translate into higher biologically active exposure.

Nano- and microemulsion approaches attempt to resolve this problem through stable dispersion. Xu et al.[50] showed that incorporation of GML into a nanosystem could overcome constraints associated with poor aqueous solubility and restore effective antibacterial performance. Ma et al.[49] likewise used microemulsion strategies to improve lauric-acid delivery against *S. aureus*. Although these studies originated largely in food or material contexts, their formulation principles are directly relevant to biomedical design.

Petrolatum offers a different approach. Rather than forcing GML into water, it uses a nonaqueous hydrophobic carrier already familiar in topical dermatology. Schlievert et al.[21] reported that GML formulated in petrolatum killed both methicillin-sensitive and methicillin-resistant *S. aureus* in vitro, with activity also observed against coagulase-negative staphylococci and viridans streptococci. A high-concentration formulation was bactericidal in a rabbit dermatitis model. The study therefore strengthens the argument that topical GML development should focus on vehicle–compound systems rather than GML in isolation.

Figure 3 places contemporary LA/GML evidence on a translational ladder and illustrates why the strongest current conclusions concern local therapy rather than systemic treatment. Its intentionally conservative progression prevents mechanistic or preclinical evidence from being treated as a substitute for demonstration of clinical benefit.

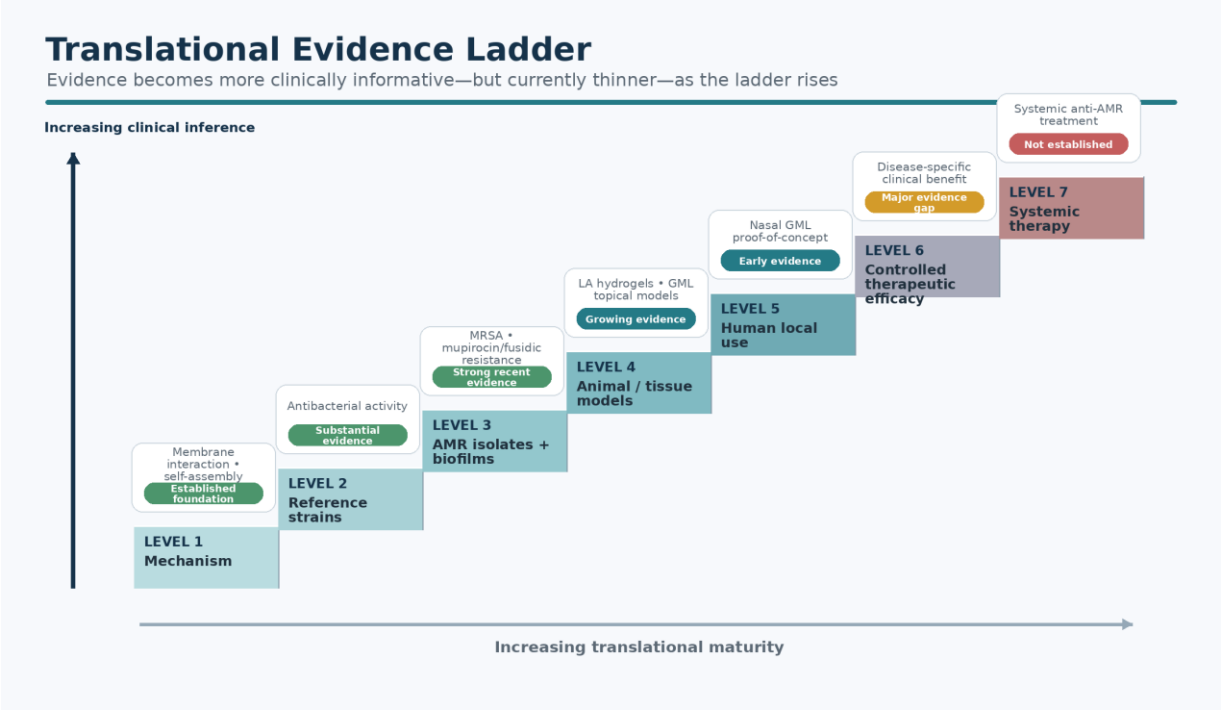

Figure 3: *Evidence ladder distinguishing mechanism and preclinical activity from therapeutic clinical evidence. Source: conceptual synthesis by the author based on Schlievert and Peterson*[19]*, Hassan Abd El-Ghany et al.*[16,17]*, Laowansiri et al.*[18]*, Yang et al.*[20]*, Yoon and Jackman*[14]*, and Schlievert et al.*[21]

The ladder is intentionally asymmetric. Strong mechanistic and microbiological evidence cannot compensate mathematically for missing clinical trials because different forms of evidence answer different questions. MIC studies establish susceptibility; animal models establish biological plausibility in complex tissues; human trials establish whether benefits and harms occur in the intended population.

Theme 9: Local human evidence exists, but it remains preliminary

The 2020 nasal study by Schlievert and Peterson is important because it demonstrated that topical GML research has crossed the human-exposure threshold. Forty healthy volunteers participated in the study, and the 5% GML nonaqueous gel reduced nasal *S. aureus* by roughly three log units in the experimental setting without reported serious adverse outcomes. This result justifies additional trials but should not be converted into a claim of established decolonisation therapy.

Several methodological questions remain. Future studies should compare GML directly with mupirocin or povidone-iodine approaches, evaluate recurrence after treatment, assess selection of GML-tolerant phenotypes, examine effects on commensal organisms, and measure acceptability and local irritation. The comparator issue is particularly relevant because mupirocin resistance exists and may compromise decolonisation in some settings[46,47].

The microbiome should also become a formal safety endpoint. Selective depletion of *S. aureus* can be advantageous if beneficial skin organisms are preserved. Wilkinson et al.[55], using an unrelated *S. aureus*-targeted antimicrobial strategy, demonstrated that selective pathogen reduction can restore microbial diversity and improve tissue repair. This provides an important conceptual benchmark: future GML development should aspire not merely to lower bacterial counts but to achieve ecologically appropriate control.

Theme 10: "Resistance-proof" claims are scientifically unjustified

A recurrent argument for membrane-active antimicrobials is that bacteria will find it difficult to evolve resistance because the membrane is essential and broadly distributed. There is logic to this proposition, but strong versions of the claim are not supported. Bacterial membranes are evolutionarily plastic. Organisms can alter fatty-acid composition, membrane charge, phospholipid distribution, cell-wall structures and efflux activity, while some pathogens can incorporate exogenous fatty acids into their own lipids[10,24].

More broadly, bacterial defensive systems are evolutionary products that often predate modern clinical antibiotics. Efflux pumps, stress responses, biofilm formation and envelope remodeling can protect bacteria from multiple environmental threats and subsequently provide cross-protection against antimicrobials[56]. Evolution-informed therapeutic development therefore requires deliberate testing of resistance trajectories rather than assuming that a natural compound is inherently evolution-proof[57].

For LA/GML, this means future studies should perform serial-passage experiments at sublethal concentrations, genome sequencing of adapted isolates, membrane lipidomics, cross-resistance testing, fitness analysis and reversal experiments after removal of selection pressure. Combination studies should determine whether LA/GML slows or accelerates evolution of resistance to companion antibiotics. Without such data, claims of a "low propensity for resistance" remain hypotheses.

DISCUSSION AND ANALYSIS

What the evidence now supports

The central conclusion of this synthesis is that LA and GML have progressed beyond the status of vaguely promising natural antimicrobials. Their antibacterial activity is supported by mechanistic work, reference-strain experiments, resistant clinical isolates, biofilm studies, combination assays and increasingly sophisticated delivery systems. The evidence is particularly convincing for Gram-positive bacteria and *S. aureus*, including MRSA[13,16,18].

Three properties make these compounds particularly interesting. First, their membrane-centred activity differs from many conventional single-target antibiotics. Second, they possess functions beyond planktonic bactericidal activity, including biofilm and virulence modulation. Third, they can potentially be incorporated into local carriers capable of achieving concentrations that would be difficult to obtain systemically. Together, these properties create a plausible

niche in topical anti-AMR development rather than requiring LA/GML to compete directly with intravenous antibiotics for invasive sepsis.

The MIC discrepancy should become a research agenda

The striking variation in reported monolaurin MIC values deserves particular emphasis. Laowansiri et al.[18] reported low-µg/mL activity, whereas Hassan Abd El-Ghany et al.[16,17] reported MIC values up to 1,000-fold higher in some isolates. A simplistic review could select whichever estimate best supports its thesis. A critical review should instead ask why the field lacks reproducible potency benchmarks.

Hydrophobic compounds are vulnerable to susceptibility-testing artifacts. If GML precipitates or adsorbs to plastic, nominal concentration can substantially exceed freely available concentration. If a solvent, surfactant or nanoemulsion increases molecular dispersion, apparent potency can change dramatically even when the chemical structure remains identical. Differences in agar dilution, broth systems, vehicle composition and incubation may therefore become determinants of observed activity[14,50].

The field would benefit from a minimum reporting standard for antimicrobial lipids. Researchers should report compound purity, synthesis/source, positional isomer composition when relevant, analytical verification, solvent, carrier, concentration units, dispersion stability, inoculum, medium, pH, incubation conditions and vehicle controls. Where possible, actual dissolved concentrations should be measured. Reference strains and a small panel of shared resistant isolates could enable cross-laboratory benchmarking.

Why topical translation is the most rational development pathway

Systemic antimicrobial development imposes demanding pharmacological requirements. A molecule must be absorbed or administered parenterally, survive metabolism, achieve effective concentrations in target tissues, remain sufficiently unbound to interact with bacteria, avoid rapid clearance, and maintain a therapeutic index compatible with repeated dosing. For amphiphilic molecules that readily interact with lipid membranes and proteins, these requirements may be particularly challenging.

Topical administration changes the problem. Concentrations in the milligram-per-millilitre range can potentially be achieved at a local surface without requiring equivalent plasma exposure. A carrier can maintain contact with skin or wound tissue, reduce washout, and modulate release. This is why the 2020 nasal GML study, LA wound hydrogels and 2026 petrolatum formulation are disproportionately important: they identify situations in which the physical properties of GML become assets rather than liabilities[19–21,53].

Potential indications should nevertheless be prioritized according to clinical need and evidence. Nasal *S. aureus* decolonisation is attractive because the target is accessible, mupirocin resistance exists and microbiological endpoints can be measured readily. AD-associated resistant *S. aureus* is another candidate, but trials must distinguish bacterial colonisation from clinically infected lesions. Chronic or diabetic wounds provide a third opportunity because biofilm, inflammation and impaired healing can be addressed simultaneously through multifunctional biomaterials.

Atopic dermatitis requires microbiome-aware rather than indiscriminate antimicrobial therapy

Atopic dermatitis demonstrates why pathogen eradication is not necessarily equivalent to disease treatment. *S. aureus* abundance correlates with disease activity, but AD is fundamentally a multifactorial inflammatory-barrier disorder. Barrier dysfunction and immune dysregulation can create an ecological environment favourable to *S. aureus*, which in turn can exacerbate inflammation and tissue damage. Thus, antimicrobial intervention can be useful in selected contexts without being sufficient to treat the underlying disease[37,41,42].

Broad antimicrobial suppression could also damage commensal networks that inhibit *S. aureus* or participate in immune homeostasis. Dermatological stewardship literature therefore warns against unnecessary or prolonged antimicrobial exposure[32,34]. GML will not automatically solve this problem merely because it is lipid-derived. Its spectrum against commensal staphylococci, streptococci and other skin microorganisms must be mapped at clinically relevant concentrations.

The ideal product might therefore be one that preferentially reduces pathogenic *S. aureus* burden while supporting ecological recovery. Whether GML can achieve such selectivity remains uncertain. The 2026 petrolatum study reported activity against several Gram-positive organisms, which may be beneficial for polymicrobial disease but also underscores the need for microbiome-level evaluation[21].

Wound applications reveal the importance of multifunctionality

The wound literature suggests that the most successful LA product may not look like a conventional antibiotic formulation. Chronic wounds involve bacterial burden, biofilm, oxidative stress, inflammation, impaired angiogenesis, excess exudate and disrupted extracellular matrix. A hydrogel that releases LA while controlling moisture, adhering to tissue and supporting regenerative processes can therefore address multiple constraints simultaneously[20,53]. More broadly, next-generation wound technologies increasingly integrate antimicrobial delivery with sensing, moisture control and regenerative functions, indicating that translation should be benchmarked against multifunctional wound-management platforms rather than against simple topical antibiotics alone[58].

This is consistent with broader advances in multifunctional wound materials. Xu et al.[54], for example, demonstrated that chitosan-based systems can combine antibacterial and regenerative characteristics. Consequently, future LA biomaterials should be compared not only with antibiotics but also with next-generation wound dressings. Evaluation should include bacterial load, biofilm architecture, inflammatory response, epithelial closure, collagen deposition, angiogenesis, pain, dressing changes and safety.

Antibiotic-adjuvant use deserves dedicated development

Combination therapy may ultimately offer a more realistic path than monotherapy. If GML can sensitise resistant organisms or disrupt biofilms sufficiently to improve β-lactam penetration and activity, it could preserve existing antibiotics rather than replace them. This aligns conceptually with antimicrobial stewardship because the value would lie in increasing effectiveness while potentially reducing required antibiotic exposure[16,17].

However, combination development should be rigorous. Checkerboard synergy should be confirmed with time-kill assays and relevant resistant lineages. Antagonism should be actively investigated. Combination performance should then be evaluated in tissue models, since membrane-active lipids may interact with host proteins or wound components. Evolution experiments should determine whether combination exposure alters resistance trajectories. The principles of eco-evolutionary therapy are directly relevant here: the immediate reduction in bacterial burden is not the only endpoint; the evolutionary consequences of the treatment environment also matter[57].

The Gram-negative boundary should be acknowledged

The strongest current evidence base does not justify presenting LA/GML as universal solutions for AMR. The outer membrane of Gram-negative bacteria restricts entry of many hydrophobic and amphiphilic compounds, and the medicinal-chemistry challenges of reaching Gram-negative intracellular targets are well documented[8]. Although antimicrobial lipids can affect selected Gram-negative species under particular conditions, the present clinical development case is considerably stronger for Gram-positive targets such as *S. aureus*.

This limitation should be considered strategic focus rather than failure. AMR innovation does not require one compound to cover every priority pathogen. A topical agent that safely controls MRSA colonisation, resistant AD-associated organisms or chronic wound biofilms could have clinical value even with a relatively narrow spectrum.

Public-health potential must be demonstrated rather than assumed

Palm-kernel-based production invites attractive claims concerning low cost, renewable feedstocks and accessibility, particularly for palm-producing countries. These possibilities merit investigation but should not be presented as established facts. Pharmaceutical-grade purification, formulation materials, sterile or controlled manufacturing,

analytical testing, stability programmes, clinical trials, regulatory compliance and distribution can dominate product cost regardless of feedstock price.

Similarly, "natural" and "renewable" do not automatically mean environmentally preferable. A credible sustainability assessment would need to evaluate land-use implications, energy and solvent consumption, process yield, waste streams, purification intensity and comparison with synthetic or alternative lauric-acid sources. Palm-kernel sourcing would also need robust traceability and sustainability criteria.

The appropriate economic proposition is therefore conditional: palm-kernel-derived LA/GML **could** create a higher-value antimicrobial platform if pharmaceutical quality can be achieved competitively and if clinical effectiveness is demonstrated. Techno-economic analysis and life-cycle assessment should be incorporated relatively early in development rather than added after a product has been optimised.

Antimicrobial stewardship implications

Dermatology presents a particularly relevant stewardship opportunity because topical and systemic antibiotics may be used for skin infections and inflammatory disorders in which microbial involvement varies among patients[33,35]. An effective nonconventional antimicrobial could theoretically reduce reliance on repeated topical antibiotic courses. However, replacing one indiscriminately used antimicrobial with another would not constitute stewardship.

A GML or LA product should therefore be embedded within diagnostic and indication-specific protocols. Use could be targeted to confirmed colonisation before selected procedures, resistant *S. aureus* in clinically infected AD lesions, or infected chronic wounds in which microbiological evidence supports its role. Surveillance for reduced susceptibility should accompany deployment.

Community-associated MRSA further reinforces the need for geographically responsive surveillance. The epidemiology of skin and soft-tissue MRSA varies by region and over time, and recent review evidence indicates declining frequency in some high-income settings while epidemic clones remain important globally[59]. A local antimicrobial platform should therefore not be developed on the assumption of a uniform global MRSA epidemiology.

A translational roadmap

The evidence synthesized in this review supports a staged rather than speculative development programme. Figure 4 proposes a translational decision matrix in which topical indications advance only when formulation, microbiological, safety, and clinical requirements are progressively satisfied.

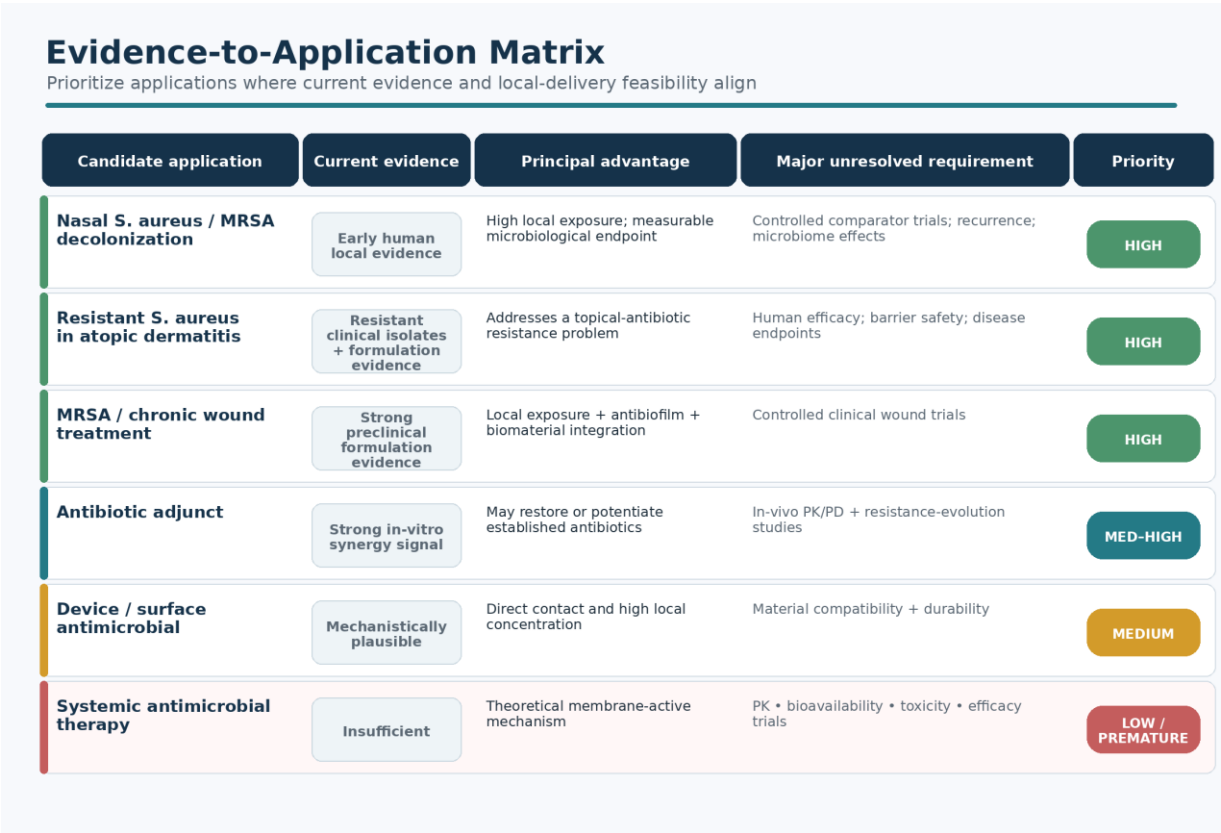

Figure 4: *Translational prioritization of possible LA/GML applications based on the present evidence base. Source: synthesis by the author.*

The first developmental stage should establish a pharmaceutical reference material with reproducible purity and physicochemical behaviour. Palm-kernel-derived material should be analytically compared with high-purity reference GML to demonstrate biochemical and functional equivalence. The second stage should standardise susceptibility, time-kill, biofilm and combination methodologies across representative clinical isolates. This should include MRSA lineages, mupirocin-resistant isolates, fusidic-acid-resistant isolates and strains with clinically relevant biofilm phenotypes.

The third stage should optimise formulation according to indication. Nasal decolonisation may favour nonaqueous gels or ointments; AD may require cosmetically acceptable barrier-compatible formulations; and chronic wounds may favour adhesive or stimuli-responsive hydrogels. Formulations should undergo stability, release, tissue-penetration, cytotoxicity and microbiome studies.

The fourth stage should deliberately address resistance. Serial passage, whole-genome sequencing, transcriptomics or lipidomics, phenotypic stability and cross-resistance studies should be performed before large clinical

programmes. This recommendation follows from the broader recognition that bacterial defences and membrane adaptation can undermine apparently robust antimicrobial strategies[24,56].

The fifth stage should focus on carefully selected human trials. Nasal decolonisation is arguably the most straightforward because bacterial load, recurrence and comparator efficacy can be measured clearly. AD studies should enroll patients with defined *S. aureus* colonisation or infection phenotypes and assess both microbiological and dermatological endpoints. Wound trials should incorporate bacterial burden, biofilm, healing rate and patient-centred outcomes. Only after these steps would larger comparative-effectiveness, economic and implementation studies be justified.

An evidence-proportionate interpretation of "palm-derived antimicrobial"

The palm-specific contribution of this research domain should ultimately be framed as a manufacturing and innovation proposition rather than as a distinct pharmacological mechanism. A GML molecule derived from palm kernel is not expected to interact with bacterial membranes differently merely because of its botanical origin. The scientifically meaningful questions concern whether palm-kernel processing can produce high-purity material consistently, economically and sustainably; whether that material is equivalent to established reference GML; and whether regional palm-based supply chains can support biomedical manufacturing.

This interpretation is important for publication credibility. Exaggerating palm specificity would weaken the article, whereas explicitly separating **molecular antimicrobial evidence** from **feedstock-development evidence** creates a novel interdisciplinary contribution. The former establishes why LA/GML deserve investigation; the latter establishes why palm-kernel-rich economies may have an industrial opportunity to participate in their development.

CONCLUSION

Lauric acid and glycerol monolaurate occupy a scientifically interesting intersection between lipid chemistry, microbiology, antimicrobial resistance, formulation science, dermatology and biomaterials. Contemporary research supports genuine antimicrobial activity against *S. aureus*, including resistant phenotypes, and indicates that membrane perturbation is a central mechanism accompanied by biofilm, virulence and potentially antibiotic-sensitizing effects. The emergence of studies involving MRSA wound isolates, antibiotic-resistant *S. aureus* from patients with atopic dermatitis, advanced lauric-acid wound hydrogels, local human nasal exposure and GML-petrolatum formulations indicates meaningful translational progress.

Nevertheless, the evidence does not support describing LA or GML as established clinical alternatives to systemic antibiotics. Reported antimicrobial concentrations vary dramatically between studies, susceptibility methodology is insufficiently standardized, formulation strongly influences biological availability, and clinically relevant information concerning resistance evolution, microbiome effects, tissue pharmacology and comparative efficacy

remains limited. These uncertainties should be treated as a research agenda rather than obscured by enthusiasm for natural antimicrobials.

The most rational near-term strategy is therefore to prioritize **local and topical applications** where the physicochemical properties of LA/GML are advantageous. Nasal decolonisation, resistant *S. aureus* associated with atopic dermatitis, chronic or diabetic wounds, and antibiotic-adjuvant formulations represent the strongest candidates. Systemic antimicrobial development should remain a lower-priority exploratory objective until convincing pharmacokinetic, toxicological and efficacy evidence exists.

Palm kernel contributes an additional industrial dimension because it offers a renewable lauric-acid-rich feedstock from which monolaurin can be manufactured. However, the scientific case for palm-kernel-derived antimicrobials will depend on pharmaceutical-grade standardization, functional equivalence, sustainable sourcing, techno-economic viability and clinical evidence—not simply on feedstock abundance. If these requirements can be satisfied, palm-kernel-derived LA and GML could evolve from commodity-derived lipids into precision local antimicrobial technologies that complement existing antibiotics and contribute to antimicrobial stewardship. The opportunity is therefore credible but conditional: the next phase must move from demonstrating antimicrobial activity toward demonstrating reproducible formulation, selective biological performance, resistance resilience, patient benefit and public-health value.

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