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Perspective, Opinion, Commentary

Milk and Skin: A Narrative, Integrative Review of Dairy and Dairy Substitutes Through the Lens of Cutaneous Inflammation

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Nutrition influences pathophysiology in acne and many cutaneous inflammatory disorders. Milk and milk products may worsen inflammation via multiple mechanisms. Recent syntheses on diet and acne, alongside reviews of vegan/plant-forward approaches, justify addressing these questions in dermatology practice. An integrative, patient-centered approach, utilizing short-term trials, objective tracking, and balanced communication is proposed given the heterogeneity of studies and many remaining questions.

INTRODUCTION

Acne vulgaris is a chronic inflammatory disease of the pilosebaceous unit driven by the interaction of androgen signaling, sebum production, follicular hyper-keratinization, microbial dysbiosis, and immune activation.[1,2] Emerging evidence demonstrates that diet, particularly dairy intake, modulates several of these core pathogenic pathways through hormonal, metabolic, and inflammatory signaling mechanisms.[1,2] Among dietary exposures, milk consumption has shown one of the most consistent associations with acne severity across epidemiologic, experimental, and mechanistic studies.[1,3,4]

Patients frequently ask whether milk worsens acne, whether specific dairy products are more problematic, and whether plant-based milk alternatives offer dermatologic benefits. Recent systematic reviews confirm that dairy intake, especially skim milk, is associated with increased acne prevalence and severity, while non-dairy diets show inconsistent protective effects.[2,4] These clinical observations parallel mechanistic data demonstrating that dairy augments insulin and insulin-like growth factor-1 (IGF-1) signaling, activates the mTORC1 pathway, enhances sebocyte lipogenesis, and promotes follicular hyper-keratinization and *Cutibacterium acnes*-driven inflammation.[1,2]

Dairy processing further modifies the biologic activity of milk.[5,6] Skim milk contains a higher proportion of whey proteins relative to fat, amplifying postprandial insulin and IGF-1 responses.[1] Additionally, β-casein variants differ in their inflammatory potential. Digestion of A1 β-casein releases opioid-like peptide β-casomorphin (BCM-7), which has been shown to induce pro-inflammatory immune activation, whereas A2 β-casein does not generate this bioactive fragment.[7] Fermentation, fat fractionation, and ultra-pasteurization further alter milk peptide profiles and lipid mediators, potentially influencing acne risk.[5,6]

This review synthesizes current clinical and mechanistic evidence examining how dairy and selected milk alternatives influence acne pathogenesis, with particular emphasis on insulin-IGF-1-mTORC1 activation, lipid-mediated inflammation, and gut-skin immune signaling. Additionally, wherever relevant, select evidence on human milk bioactives is briefly discussed to contextualize shared inflammatory and immunologic pathways relevant to acne development.[8-13]

METHODS

A comprehensive literature search was conducted using PubMed on dairy and plant-based milks in relation to acne, cutaneous inflammation, and early-life atopy/allergy. We combined keywords/MeSH such as *milk*, *dairy*, *plant-based milk*, *soy*, *oat*, *acne*, *insulin*, *IGF-1*, *mTOR*, *glycemic*, *skin*, *inflammation*, *human milk*, *MFGM*, *HMOs*, *microbiome*, *eczema*, *atopic dermatitis*, *food allergy*. We prioritized human observational studies, randomized trials, and systematic/narrative reviews with dermatology relevance. Animal-only primary studies were not used to support clinical conclusions; when broader reviews included animal data, we cited them only to frame biologic plausibility. Given heterogeneity in exposures and outcomes, we synthesized findings narratively.

DAIRY AND ACNE

Multiple systematic reviews and narrative reviews report an association between dairy consumption and the prevalence or severity of acne with the most consistent signal observed for skim and low-fat milk rather than whole milk.[1,3,4] This preferential association strongly suggests that the acne-promoting effects of dairy are not primarily lipid-driven, but instead mediated through protein-dependent endocrine signaling mechanisms.

Skim milk retains its full complement of whey proteins while removing milk fat. The absence of fat eliminates fat-mediated delays in gastric emptying and satiety signaling, leading to more rapid amino acid absorption and greater post-prandial insulin release.[1,2] Whey proteins are partic-

ularly enriched in branched-chain amino acids such as leucine, which directly stimulate pancreatic insulin secretion and amplify hepatic insulin-like growth factor-1 (IGF-1) production.[1,2] In contrast, whole-fat milk attenuates glycemic and insulin responses by slowing nutrient absorption and modifying incretin signaling. This mechanistic distinction provides a biologically plausible explanation for why skim milk consistently demonstrates a stronger epidemiologic association with acne than full-fat dairy.[1,3,4]

At the intracellular level, insulin and IGF-1 converge on mammalian target of rapamycin complex-1 (mTORC1), a central nutrient-sensing pathway that drives sebocyte lipogenesis, keratinocyte hyperproliferation, and follicular hyper-keratinization, core processes in comedogenesis.[1,2] These signaling pathways also intersect with androgen receptor activation, creating synergistic amplification of sebum production and inflammatory lesion formation.[1,2] Collectively, this endocrine–metabolic model explains how dairy, particularly skim milk, can biologically potentiate acne beyond simple caloric load.

THE ROLE OF DAIRY FORM, PROCESSING, AND BIOACTIVE PROTEINS IN ACNE

Milk is often treated as a single dietary exposure, but its biological effects vary substantially depending on its form and method of processing. These differences matter in acne because processing changes the balance of fat, protein, and bioactive peptides that influence insulin signaling, inflammation, and downstream mTORC1 activation.[1,2] As discussed above, the preferential association of skim and low-fat milk with acne severity highlights the importance of protein-driven endocrine signaling rather than milk fat.[1,3,4]

Beyond macronutrient balance, additional industrial processing steps further modify milk's bioactive structure. Milk fat is naturally packaged within the milk fat globule membrane (MFGM), a complex trilayer composed of phospholipids, sphingolipids, cholesterol, and embedded bioactive proteins involved in epithelial barrier integrity and immune modulation.[5,6] Homogenization mechanically disrupts MFGM structure, increasing fat surface area and altering lipid–protein interactions, while pasteurization and ultra-high-temperature processing denature heat-sensitive proteins and modify peptide fragmentation.[5,6] Although dermatology-specific outcome trials isolating these effects are limited, such changes potentially influence lipid mediator delivery, inflammatory signaling, and metabolic responses relevant to acne.

In addition to structural lipids, dairy contains multiple bioactive proteins with endocrine and immunologic activity. Whey proteins, including α-lactalbumin and β-lactoglobulin, are enriched in branched-chain amino acids such as leucine and are strongly insulinotropic, amplifying insulin and IGF-1 signaling central to acne biology.[1,2] Casein-derived peptides represent another biologically active class that may influence immune pathways relevant to inflammatory skin disease.

Fermentation further modifies dairy composition through bacterial metabolism of lactose and milk proteins, generating short-chain fatty acids and bioactive peptides that can alter immune signaling and metabolic responses. This may partially explain why fermented dairy products such as yogurt often demonstrate weaker or inconsistent epidemiologic associations with acne compared to liquid milk,[3,4] although controlled acne-specific trials comparing fermented and non-fermented dairy remain limited.

Public and scientific interest has also focused on β-casein genetic variants, particularly A1 versus A2 β-casein. Digestion of A1 β-casein releases the opioid-like peptide β-casomorphin-7 (BCM-7), which has been shown in vitro to induce pro-inflammatory immune activation and increased proliferation of human peripheral blood mononuclear cells, whereas A2 β-casein does not generate this fragment.[7] Despite this mechanistic possibility, controlled clinical trials directly comparing A1-dominant and A2-predominant milk in acne populations are lacking, and major acne reviews do not yet distinguish outcomes based on β-casein subtype.[3,4] Any dermatologic benefit of A2 milk should therefore be framed as theoretically plausible but clinically unproven.

From a practical standpoint, these distinctions support prioritizing reduction of skim and low-fat milk in acne-prone patients while recognizing that whole-fat, fermented, or structurally less disrupted dairy products may exert different biologic effects, though outcome data remain limited.[1-4] A short, structured trial of dairy modification with objective acne tracking represents a pragmatic, patient-centered approach consistent with the current evidence.

NON-DAIRY MILKS: GLYCEMIC LOAD, ENDOCRINE SIGNALING, AND ACNE-RELEVANT CONSIDERATIONS

Unlike dairy, which directly activates acne-relevant endocrine and inflammatory pathways such as insulin, IGF-1, and mTORC1, plant-based milk alternatives do not have established direct inflammatory or acne-promoting mechanisms based on clinical outcome data[1,3,4,14] Therefore, the acne relevance of non-dairy milks is best understood indirectly through their effects on glycemic load, insulin resistance, and overall nutrient balance, rather than through intrinsic inflammatory mediators.

Soy-based beverages provide the most complete protein profile among plant-based milks and are frequently included in plant-forward dietary patterns discussed in dermatology nutrition reviews.[14] However, clinical data directly linking soy beverages to improved or worsened acne outcomes are inconsistent.[1,3,4] From a practical standpoint, unsweetened soy formulations are preferred to minimize postprandial glycemic spikes that could secondarily amplify insulin and IGF-1 signaling.

Other plant-based milks illustrate why dairy substitution is not one-size-fits-all. Almond milk is typically low in protein and calories and provides little direct endocrine stimulation, however, when used as a sole dairy replacement it may fail to meaningfully blunt postprandial glycemic load if

consumed alongside carbohydrate-heavy foods.[1,4] Oat milk varies widely in free sugar and processing-derived maltose content, which can produce higher postprandial glucose and insulin responses than other plant-based options, theoretically enhancing acne-relevant endocrine signaling.[1,4] Hemp milk offers a more favorable omega-3 to omega-6 fatty acid ratio, which is biologically relevant given the role of lipid mediators in cutaneous inflammation.[15] Coconut milk primarily provides medium-chain triglycerides with little protein, while rice milk is typically low in protein and relatively high in glycemic index. None of these beverages contain intrinsic bioactive mediators comparable to dairy-derived whey proteins or IGF-1, but their net effect on acne depends on how they influence glycemic load and insulin secretion within the overall diet.[1,4,15]

Functional ingredients such as prebiotic oligosaccharides and probiotic (synbiotic) components may further modify skin-relevant immune pathways. Prebiotic oligosaccharides have been shown to support epithelial barrier function and antioxidant defenses, while probiotic effects on inflammation are strain-dependent and context-specific.[16-21] Currently, these agents should be viewed as adjunctive modifiers of barrier and immune signaling rather than primary acne therapies.

From a clinical perspective, when patients replace dairy with non-dairy milks, counseling should focus on minimizing added sugars, avoiding glycemic spikes, and ensuring adequate micronutrient fortification (calcium, vitamin D, vitamin B12, iodine). Rather than framing plant-based milks as anti-inflammatory by default, substitutions should be individualized based on the patient's acne phenotype, glycemic sensitivity, and overall dietary composition.

EARLY-LIFE FEEDING, MICROBIOME, AND ATOPY

Although acne is classically regarded as a disease of adolescence and young adulthood, early life nutritional exposures provide important mechanistic context for understanding how milk-derived bioactives influence immune programming and inflammatory skin trajectories later in life. Human milk is a complex biological system that delivers transforming growth factor-β (TGF-β), microRNAs, human milk oligosaccharides (HMOs), and a diverse milk-associated microbiome.[8,9,11,12,22,23] These bioactive components regulate epithelial tolerance, microbial colonization, and Th2/Th17 immune balance during sensitive early-life periods when the immune system is learning what to tolerate and what to react to.[9,11,12]

Despite strong mechanistic data, clinical trials attempting to reduce atopic disease through isolated early-life interventions have produced mixed results. Large systematic reviews and randomized trials evaluating maternal diet, infant formula modification, and emollient-based skin barrier strategies do not demonstrate consistent prevention of eczema or food allergy.[13,24-26] These findings highlight the multifactorial nature of immune-mediated skin disease and demonstrate the limitations of targeting single nutritional or topical variables in isolation.

Within the context of this review, early-life milk bioactives are discussed not as direct determinants of acne risk, but as foundational modifiers of immune and epithelial signaling pathways that remain relevant to later cutaneous inflammatory conditions. These shared pathways, including IGF-1 signaling, lipid mediator balance, and microbiome-immune overlap, help demonstrate why milk remains biologically active across the lifespan.[8,9,11]

SKIN AS AN IMMUNE ORGAN

The skin functions as an active immune organ, integrating barrier integrity, innate immune signaling, antigen presentation, and adaptive immune programming.[27] Food allergy and inflammatory skin disease models emphasize that epithelial dysfunction, microbial dysbiosis, and immune skewing act together rather than in isolation.[28] From a dermatologic perspective, this reinforces that nutrition-related antigen exposure, including dairy-derived bioactive proteins, interacts dynamically with the cutaneous immune environment rather than acting through a single pathway.

In clinical practice, this framework supports pairing nutrition modification for acne with barrier-supportive strategies such as: gentle cleansing, regular moisturization in barrier-compromised patients, and avoiding known irritants. Positioning dietary intervention as a component of a broader immune-barrier model may help prevent overinterpretation of nutrition as a stand-alone therapy while still acknowledging its mechanistic relevance (See Table).

CLINICAL PEARLS AND PITFALLS

For acne-prone patients, a structured 6–8-week reduction of skim/low-fat milk while maintaining nutrient adequacy through whole foods or supplementation (protein, calcium, iodine, B12) could be considered.[1-4,14,29] If dairy is replaced, preferences should be given to unsweetened, higher-protein plant-based options (eg, soy or pea) or deliberately pairing low-protein beverages with adequate protein elsewhere, while minimizing added sugars and emulsifiers.[1,2,4,14,15]

Patients should be counseled that plant-based milks are not inherently "anti-inflammatory," and their relevance depends on how they influence postprandial glycemic load and insulin signaling within the total dietary pattern. Short, objective dietary trials with standardized lesion counts or photographic documentation allow individualized assessment while limiting unnecessary long-term restrictions.

In infants and high-risk families, clinicians should support breast feeding when feasible, emphasizing skin barrier protection, and carefully communicate the mixed evidence surrounding nutritional and topical allergy-prevention strategies to avoid overpromising benefits.[8-10,13,24-26,30]

LIMITATIONS

This narrative review reflects the heterogeneity of dietary exposures, study designs, and outcome measures. Most associations between dairy intake and acne remain observational, and causality cannot be definitively established. Dermatology-specific trials directly comparing individual plant beverages, fermented dairy products, or A1 versus A2 β-casein dominant milk are limited. Although mechanistic models strongly support endocrine and immune mediation, many processing-related and microbiome dependent pathways remain missing in acne populations.

Animal-only primary studies were not used for clinical recommendations, and a quantitative meta-analysis was not performed due to methodological heterogeneity.

CONCLUSION

A growing body of observational and mechanistic evidence links skim and low-fat dairy consumption to acne, with the insulin, IGF-1, and mTORC1 signaling pathways providing a coherent biological framework for this association.[1-4] Dairy form, degree of processing, whey protein content, and β-casein variant composition further modify endocrine and immune responses in ways that potentially influence acne pathogenesis.

In contrast, non-dairy milk alternatives are highly heterogeneous, and with limited dermatology-specific outcome data, substitutions should be individualized and integrated within the broader context of glycemic control and overall diet quality.[1,14,15] An integrative, patient-centered approach, utilizing short-term dietary trials, objective acne tracking, and caution against overgeneralization, respects scientific uncertainty while empowering patients to take an active role in their care.

Future research should include randomized trials comparing specific dairy beverages with specific plant-based alternatives on validated acne outcomes, controlled A1 versus A2 milk in acne populations, and mechanistic studies of MFGM components, prebiotic oligosaccharides, and microbiome dependent immune signaling in cutaneous inflammation.

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Table 1. Evidence Map of Dairy, Plant Milks, and Skin Outcomes

Domain	Citation(s)	Ref(s)	Type	Population/Model	Outcome/Focus	Acne-relevant takeaway
Acne–Dairy (clinical)	Meixiong 2022	3	Systematic review	Humans (observational)	Diet and acne associations	Dairy, especially skim/low-fat, associates with acne; heterogeneity noted.
Acne–Dairy (clinical)	Conforti 2021	4	Narrative review	Humans (observational)	Diet and acne	Links diet patterns (skim milk/high glycemic) with acne; evidence not causal.
Acne mechanisms	Melnik 2015; Melnik 2023	1,2	Mechanistic reviews	Translational	IGF-1/mTORC1, comedogenesis	Insulin/IGF-1 and mTORC1 drive sebocyte lipogenesis/keratinocyte proliferation → acne pathophysiology.
Milk matrix/MFGM	Fontecha 2020; Gallier 2020	5,6	Narrative/ Translational reviews	Infant nutrition context	MFGM composition/ functions; species/ processing effects	Processing/ species alter MFGM; immune/ gut-barrier relevance suggested; direct acne outcomes lacking.
Casein variants (A1/ A2)	Gard 2024	7	In-vitro study	Human PBMCs	A1 vs A2 and β-casomorphin-7	Differential immune proliferation; dermatology clinical outcomes absent.
Plant-forward diet (derm)	Lee 2023	14	Review	Humans	Vegan diet in dermatology	Potential benefits/caveats; highlights nutrient adequacy considerations.
Immune lipids	Miles 2021	15	Narrative review	Humans (development)	LCPUFAs and the immune system	Fatty-acid balance may modulate immune tone; derm applications indirect.
Human milk bioactives	Khaleva 2019	8	Systematic review	Humans	TGF-β in human milk & allergic outcomes	Links milk TGF-β to allergy outcomes; context for atopy risk.
Breast milk miRNA	Simpson 2015	22	Study/ Review	Humans	Maternal probiotics, breast milk miRNA, AD	Explores milk miRNA and probiotic exposure in relation to AD.
Milk microbiome	Ruiz 2019; Power 2024	11,12	Reviews	Humans/Mammals	Human/ mammalian milk microbiomes	Characterizes milk microbiomes; potential to influence infant immunity.
Maternal microbiota → infant immunity	Kalbermatter 2021	9	Review	Humans/Translational	Maternal microbiota, colonization, breast milk	Maternal/early colonization and milk drive neonatal immune development.
HMOs & AD	Rahman 2023	23	Review	Humans	HMO-metabolizing bacteria & eczema	Mechanistic/ clinical links between HMOs, bacteria, and AD.
Diet in pregnancy/ infancy	Garcia-Larsen 2018	13	Systematic review/ meta-analysis	Humans	Diet and allergy/autoimmunity risk	Mixed/limited preventive effects across interventions.
Formula modification RCT	Boyle 2016	24	Randomized controlled trial	High-risk infants	Prebiotic-supplemented partially hydrolyzed formula	No consistent eczema prevention benefit across outcomes.

Domain	Citation(s)	Ref(s)	Type	Population/Model	Outcome/Focus	Acne-relevant takeaway
Emollients from birth (evidence)	Kelleher 2021; Kelleher 2022; Bradshaw 2024	25,26, 30	Cochrane reviews + RCT	Infants	Skin care to prevent eczema/ food allergy	Routine emollients from birth do not prevent eczema/food allergy.
Prebiotic oligosaccharides	Zeng 2025	17	Review	Translational	Prebiotic oligosaccharide in skin health	Prebiotic oligosaccharides may support skin barrier integrity, oxidative defense, and cutaneous immune modulation.
Probiotics—food allergyPrebiotic oligosaccharides & skin	Huang 2023; Zeng 2025	16,17	Review	HumansTranslational/ Human use	Probiotics for immunomodulation in food allergyPrebiotic oligosaccharide in skin health	Strain/timing/ host dependent; supports cautious adjunctive use.Potential barrier/ oxidative benefits; clinical data evolving.
Bifidobacterium bifidum BGN4Probiotics—food allergy	Ku 2016; Huang 2023	16,18	Review	Humans/ TranslationalHumans	BGN4 functionalityProbiotics for immunomodulation in food allergy	Describes probiotic properties; dermatology outcomes not primary.Strain/timing/ host dependent; supports cautious adjunctive use.
Lactobacillus reuteriBifidobacterium bifidum BGN4	Mu 2018; Ku 2016	18,19	Review	Humans/Translational	L. reuteri in health/ diseaseBGN4 functionality	Immunomodulatory potential; derm endpoints context-dependent.Describes probiotic properties; dermatology outcomes not primary.
Lactobacillus (updates)Lactobacillus reuteri	Un-Nisa 2022; Mu 2018	19,20	Review	Humans/Translational	Probiotics across conditionsL. reuteri in health/disease	Overview across strains; dermatology applications indirect.Immunomodulatory potential; derm endpoints context-dependent.
Probiotics (women/ children)Lactobacillus (updates)	Kwok 2022; Un-Nisa 2022	20,21	Review	Women & childrenHumans/ Translational	Inflammation/health markersProbiotics across conditions	Variable effects; underscores heterogeneity.Overview across strains; dermatology applications indirect.
Breast milk–associated microbiotaProbiotics (women/children)	Kwok 2022	21	Mini-review	Women & children	Breast milk microbiota & newborn immunityInflammation/health markers	Supports role of breast milk microbes in immune education.Variable effects; underscores heterogeneity.
Breastfeeding & allergy	Nuzzi 2021	10	Review	Humans	Breastfeeding and allergic diseases	Summarizes benefits/limits; prevention evidence mixed.

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