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ACNE AS A METABOLIC DISEASE: THE ROLE OF DIET, DYSBIOSIS, AND THE GUT–SKIN INTERFACE

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Acne vulgaris is no longer viewed as a superficial disorder of the pilosebaceous unit alone. Current evidence positions it as a systemic metabolic condition driven by microbial dysbiosis. The traditional emphasis on Cutibacterium acnes overgrowth has given way to a more nuanced understanding: specific virulent strains – particularly phylotype IA_1 ribotypes RT4 and RT5–are the primary instigators. These strains activate Toll-like receptor 2 (TLR-2) on keratinocytes and sebocytes, triggering a cascade of pro-inflammatory cytokines such as interleukin-1β and tumor necrosis factor-α. This pathogenic shift underscores that acne arises not merely from bacterial presence but from a disrupted host-microbe interaction.

Dietary factors play a decisive role in shaping this dysbiosis. High-glycemic foods and dairy products elevate circulating insulin-like growth factor-1 (IGF-1). Elevated IGF-1 suppresses the transcription factor FOXO1–a key metabolic regulator–while activating the mTORC1 signaling pathway. The result is follicular hyperkeratinization, increased sebum production, and a lipid-rich microenvironment that favors the proliferation of pathogenic C. Acnes RT4/RT5 over commensal strains like RT6. This metabolic reprogramming transforms a balanced ecosystem into one that perpetuates inflammation and tissue injury.

The gut-skin axis provides the mechanistic link between intestinal health and cutaneous inflammation. Intestinal dysbiosis compromises gut epithelial integrity, leading to increased permeability–often termed “leaky gut.” This allows bacterial lipopolysaccharides (LPS) to enter the circulation, amplifying systemic inflammation and indirectly exacerbating acne. Conversely, restoring gut microbial balance offers a therapeutic opportunity. Supplementation with Lactobacillus rhamnosus SP1 has been shown to downregulate IGF-1 expression by approximately 32% and upregulate FOXO1 expression by 65%, effectively suppressing mTORC1-driven lipid synthesis and reducing inflammatory signaling.

Beyond probiotics, prebiotics and postbiotics are emerging as viable alternatives to broad-spectrum antibiotics. Prebiotics such as inulin and fructo-oligosaccharides selectively promote beneficial gut bacteria that produce short-chain fatty acids (SCFAs). These SCFAs reach the skin via the circulation, reinforce the epidermal acid mantle, and lower follicular pH–creating an environment less conducive to colonization by pathogenic strains. Postbiotics, including bacterial lysates, offer a stable, non-antibiotic approach that modulates local immunity without disrupting the wider microbial ecosystem.

Clinically, these insights support a shift away from antibiotic-centric protocols toward personalized microbiome-based strategies. Targeting the gut-skin axis through L. Rhamnosus supplementation, prebiotic-rich nutrition, and low-glycemic dietary patterns can regulate the mTORC1/FOXO1 pathway and attenuate TLR-2-mediated inflammation. Such interventions address the root drivers of acne while minimizing the risk of antimicrobial resistance–a growing concern in dermatology.

In summary, modern acne management demands a holistic view that integrates diet, microbial ecology, and systemic metabolic signaling. By recognizing acne as a condition shaped by the gut-skin axis, clinicians can move beyond conventional topical and antibiotic therapies toward durable, pathogenesis-based solutions. This paradigm not only improves clinical outcomes but also aligns with the broader movement toward precision medicine in dermatology.