A teenager comes to see you about their acne vulgaris. You are asked if altering their diet would help. How do you reply?

Lucas (Tze Peng) Tan

University of Auckland

Student Identity Number: 336533427

Email: ttan636@aucklanduni.ac.nz

Word Count: 1997

Postal Address: 26B Hapua Street, Remuera, Auckland, 1050

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Introduction

"Could it be the energy drinks, processed food, or protein powder?" A teenager sits across from me, hoping a dietary fix might clear their skin. My short answer is honest but hopeful. Diet alone isn't a cure for acne; however, targeted dietary changes can serve as a valuable adjunct to evidence-based care for some teenagers. I would explain that care will be anchored in proven first-line therapies (e.g., benzoyl peroxide, topical retinoids, prudent antibiotics, and escalation as needed) and run a time-boxed trial of a lower-glycaemic pattern.[1-3] If intake is high, we'll also consider dialling back on skimmed milk.[4,5] Finally, we'll set goals that protect school, mood, and self-image. Acne is common, burdensome, and closely tied to anxiety and depression, so psychosocial support matters as much as biomedical solutions.[6]

Prevalence and Burden of Acne Vulgaris

Acne vulgaris is a prominent condition among adolescents. In New Zealand's (NZ) Youth2000 survey, two-thirds of secondary school students reported acne, and one in seven described it as "problematic", with substantial barriers to accessing treatment and inequities affecting female, Māori, and Pacific youth.[7] Globally, the burden of acne vulgaris has risen since 1990, peaking between the ages of 15 and 19 and affecting young women more than young men, approximately 25% higher.[8]

The pathogenesis of acne vulgaris is multifactorial. Follicular hyperkeratinisation, androgen-driven sebum, *Cutibacterium acnes*-related dysbiosis, and inflammation are central, with insulin/IGF-1–mTORC1 signalling serving as a biological bridge between puberty, diet, and acne activity.[3,9] Although many cases improve, acne can persist after adolescence in between 26 to 50% of females and 12 to 42% of males, with rates declining with age.[10-14]

In addition, the psychosocial impact of acne vulgaris should not be underestimated. A meta-analysis links acne with depression and anxiety, and a population cohort found a 63% higher risk of major depression in the first year after diagnosis, highlighting the need to screen for mood and support school participation and self-image.[6,15] Physical sequelae also matter. Scarring affects between 40 to 50% of individuals, and post-inflammatory hyperpigmentation is common, particularly in darker skin, with meaningful quality-of-life impacts.[16-18]

The Role of Diet

Low Glycaemic Load (GL)/Index (GI)

Across small but well-designed RCTs, low-GI/GL patterns reduce acne lesion counts and improve hormonal markers.[1,2] In the seminal Australian trial, a 10 to 12-week low-GL plan led to significantly fewer inflammatory lesions than a control diet.[2] A short, controlled feeding study also demonstrated that lowering GI/GL reduces circulating IGF-1, a driver of sebaceous activity and keratinisation, offering a biological bridge between diet and acne.[1] Insulin/IGF-1 signalling activates mTORC1, which promotes sebocyte lipogenesis and follicular hyperkeratinisation; hence, low-GL patterns may attenuate that axis.[1,9]

In contrast, sugary beverages, ultra-processed foods, and dietary patterns with a high GL are associated with the development and exacerbation of acne. Cross-sectional analysis and a prospective cohort study demonstrated that frequent consumption of these foods is linked to higher odds of current acne, with adjusted odds ratios ranging from 1.13 to 1.54 for energy-dense, fatty, and sugary products, and 1.18 for sugary beverages.[19,20]

Dairy

Multiple meta-analyses of observational studies associate dairy—especially milk, and less consistently yoghurt or cheese—with higher odds of acne; effect sizes were modest and heterogeneous, and residual

confounding is likely.[4,5] Still, cohort studies and pooled analyses repeatedly underscore skim or low-fat milk as the most consistent signal, possibly via higher IGF-1 and whey fractions.[21-23] This is not a universal trigger, and randomised controlled trials (RCTs) isolating dairy are scarce.

Whey Protein Supplements

A recent case-control study by Muhaidat et al. suggests an association between whey supplementation and acne flares in adolescents or young adults; however, an RCT conducted by researchers in Thailand found no increase in total lesions or facial/truncal severity between whey and non-whey groups.[24,25]

Chocolate/Cocoa

Evidence on chocolate is limited but suggestive. A small RCT in acne-prone men reported more inflammatory lesions after 100% cocoa, and another found that dark chocolate worsened comedones and papules over 2–4 weeks.[26,27] A mechanistic study also demonstrated that dark chocolate increased corneocyte desquamation and Gram-positive bacterial colonisation on facial skin, potentially aggravating acne.[28] Still, samples are small and short-term, so generalisability is low.

Omega-3 Fatty Acids and Gamma-Linolenic Acid (GLA)

Small RCTs suggest a modest adjuvant benefit of long-chain omega-3s (EPA/DHA) and GLA in acne. In a 10-week double-blind RCT, omega-3 (2 g/day) and GLA groups (400 mg/day from borage oil) demonstrated significant reductions in inflammatory and non-inflammatory lesions, with histologic evidence of reduced IL-8 staining.[29] There is emerging evidence that omega-3 supplementation can modulate the cutaneous microbiome, likely through effects on local immune responses and barrier function, although direct human data in acne vulgaris are limited.[30,31]

Probiotics

Probiotics may modestly aid acne vulgaris when used alongside standard care. In a 12-week RCT of 12–30-year-olds, a capsule containing the probiotic *Lacticaseibacillus rhamnosus* (CECT 30031) and the cyanobacterium *Arthrospira platensis* (BEA_IDA_0074B) improved clinical response on the Acne Global Severity Scale and produced a greater reduction in non-inflammatory lesions, with a near-significant drop in total lesions ($p = 0.06$).[32] Separately, Kim et al.'s double-blind RCT reported that *Lactobacillus plantarum* CJLP55 reduced lesion counts and sebum measures, and a pilot trial of *Lactobacillus rhamnosus* SP1 showed improvement with shifts in IGF-1/FOXO1 expression.[33,34]

Zinc

Zinc demonstrates a modest, adjunctive signal in acne vulgaris. Patients often possess lower serum zinc, and small RCTs of oral zinc gluconate reduced inflammatory lesions, although effects were generally smaller than with minocycline.[35-37] Gastrointestinal upset is common with the use of zinc, and benefits appear to be context- and dose-dependent.[38]

Culture, Diet, and Equity in Aotearoa NZ

NZ is proudly multicultural, with extensive communities bringing diverse foodways. Population data demonstrate high consumption of sugar-sweetened beverages among NZ school-age children—with higher rates in socio-economically disadvantaged groups—and NZ children derive a large share of energy from ultra-processed foods.[39,40] Although these studies focused on younger individuals, patterns appear to persist into adolescence. NZ studies report high added-sugar intakes in 15- to 18-year-olds (~11.8% of total energy) and frequent sugary-drink consumption, dietary behaviours typically driven by UPF availability.[41,42] These patterns plausibly increase dietary glycaemic load, aligning with the abovementioned mechanisms and associations.

Counselling should be culturally safe and practical: reduce sugary drinks, choose low-GL staples within each cuisine (e.g., more legumes/vegetables and swap ultra-processed snacks for minimally processed options), and if trialling dairy reduction, protect calcium through culturally familiar alternatives. Changes should be framed as adjuncts, not moral judgements, and goals should be co-designed with the teenager and/or family.

Other Influencing Factors

Beyond diet, several other variables influence the development or severity of acne vulgaris in teenagers.

Nicotine exposure is one example. The relationship between combustible cigarette smoking and acne vulgaris is mixed, yet biologically plausible through mechanisms like increased oxidative stress, sebum peroxidation, and activation of the aryl hydrocarbon receptor pathway, all promoting comedogenesis and inflammation.[43-45] In parallel, electronic cigarettes are increasingly recognised as potential aggravators. A recent systematic review concluded that electronic cigarettes can initiate or worsen a range of dermatological conditions.[46] This is particularly relevant in Aotearoa, where youth vaping has risen steeply since 2015, with the highest rates in younger age groups and in more socio-economically deprived communities.[47,48]

Psychophysiologic stress is another well-established exacerbator. Prospective and cross-sectional studies demonstrate that acne severity increases during periods of heightened stress, like academic examinations, and correlates with validated stress scores in students and broader populations of young women.[49-52] Sleep quality is also closely linked to acne outcomes, with case-control and review data demonstrating that poorer sleep is associated with increased acne severity.[53,54] This relationship is likely bidirectional since stress, sleep, and mood interact to influence behaviour and skin outcomes.

Finally, recreational and appearance-enhancing drug use plays a role. Anabolic-androgenic steroids (AAS) are strongly linked to acne, with contemporary data suggesting that acne develops in approximately 40 to 50% of users. In some, this progresses to severe variants such as acne fulminans, driven by potent androgen receptor stimulation in sebaceous glands.[55-57] Alongside these, comedogenic cosmetics and hair pomades are recognised exogenous factors that can trigger acne and should be considered in management.[58-60] These behaviours are often shaped by rapidly evolving beauty standards, which increasingly glamourise muscularity or flawless skin, and can contribute to body dysmorphic concerns in teenagers. In this context, acne vulgaris is not only a dermatological condition but also deeply entangled with identity, self-esteem, and cultural ideals of appearance.

Practical Next Steps and Plan

Considering the literature above, my plan would be measured and blame-free while relying on proven care. One would begin by normalising and validating the teenager's concerns, sharing that acne vulgaris is common, treatable, and not the teenager's fault. It would be essential to align expectations for the visit, including what currently matters most (pain, scarring, confidence, visibility at school), what they're willing to try, and what is realistic over the next school term. One should also briefly ask about mood, sleep, and stress, because these are as important as any prescription. The evidence for diet is an adjunct, not a cure. Nonetheless, this doesn't preclude dietary management. It simply means testing changes in a collaborative, time-limited, and reversible approach.

Management would be anchored in guideline-based therapy and gentle skin care.[3] First-line topical therapy includes initiating or optimising a topical retinoid together with benzoyl peroxide. Topical antibiotics may be used, but only in combination with these agents and with careful attention to antibiotic stewardship to reduce the risk of resistance.[3,14] Twice daily cleansing with avoidance of scrubs and gentle cleansers for sensitive skin is advised, along with regular use of a non-comedogenic moisturiser and broad-spectrum sunscreen, especially for patients with post-inflammatory hyperpigmentation.[14,61] Alongside this backbone, one could co-design an eight- to 12-week dietary trial focused on the most plausible levers with the lowest downsides, including (1) shifting towards a lower-

glycaemic pattern (fewer sugar-sweetened drinks and refined starches, more minimally processed carbohydrates, legumes, vegetables, and whole grains), (2) trialling a reduction of milk, particularly skimmed, and (3) attempting to pause the use of whey protein. Avoiding restrictive dieting, protecting overall nutrition, and tailoring a plan to the teenager's cuisine, budget, and food preferences is crucial.

Beyond these levers, research is ongoing into other dietary factors like omega-3 fatty acids, probiotics, and zinc. Nonetheless, evidence is not strong enough to recommend these as routine practice, though they may be discussed as optional adjuncts if a motivated patient wishes to explore them.

To track progress, one could use baseline photographs and a simple global severity or lesion count. Key follow-up questions are whether the skin improved, whether the changes felt manageable, and what to continue or stop. If the trial shows no benefit, dietary measures can be discontinued, and adjustments to medical therapy can be considered rather than intensifying dietary rules. Where disease is severe, scarring, or distressing, escalation of therapy is appropriate. In menstruating teenagers with irregular cycles or hirsutism, polycystic ovarian syndrome should be considered.

Alongside treatment planning, safeguarding is essential. One should screen for eating-disorder risk before and during dietary changes and involve caregivers where appropriate. It is also important to advise against the use of AAS and highlight other modifiable aggravators like vaping. Throughout the patient's journey, communication should remain collaborative and shame-free, with shared decision-making involving the teenager, and if helpful, their whānau.

Conclusion

In responding to a teenager who asks whether diet can help their acne, it is vital to acknowledge the promise and limits demonstrated by evidence. Acne vulgaris is a multifactorial disease with significant psychological and social consequences. Diet is not a cure, but well-designed modifications, particularly adopting a lower-glycaemic pattern and, in some cases, reducing skim milk or whey protein, can serve as safe adjuncts to guideline-based therapy. Other factors like sleep, stress, vaping, and evolving beauty pressures deserve attention, underscoring the need for holistic care. Above all, communication with teenagers must be blame-free and collaborative. Apart from acknowledging acne as common and treatable, it is crucial to protect nutrition and self-esteem. A plan grounded in evidence, cultural safety, and shared decision-making offers the best chance of clearer skin while restoring confidence, well-being, and everyday participation.

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