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Diet Alteration in Teenagers with Acne Vulgaris

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

Our knowledge of the relationship between diet and acne is constantly evolving. Acne is both a medical and a social condition, with a multifactorial pathophysiology influenced by hormones, environment, and genetics. Epidemiological data suggests acne vulgaris is the most prevalent skin condition in the world, affecting 85% of people between 12 and 25 (1).

Adolescence is a time of significant social, physical and psychological change. The additional burden of acne at this vulnerable life stage can lead to poor self-esteem, social isolation, and psychological distress including anxiety, depression and suicidal thoughts (2). With easy access to medical information in the digital age, teenagers are increasingly aware of the intersection between lifestyle and skin health. Those suffering from acne are likely to seek out solutions and adapt their lifestyle, including their diet, in alignment with online messaging. But how accurate are those messages? Is there reliable evidence to support a link between diet and acne, and how does this intersect with the vulnerable life-stage of teenagerhood?

This essay prompts a reflection on how to respond to a teenager with acne vulgaris who queries whether diet modification would reduce their symptoms. It aims to outline the pathogenesis of acne vulgaris, uncover how diet influences this, and respond to any social considerations when suggesting diet modification for a teenager.

1. *Acne Pathogenesis and the 'Chain of Factors'*

Before approaching the teenage patient with acne, it is useful to understand the foundations of acne – things the patient cannot change. Characteristic lesions of acne include open and closed comedones, as well as inflammatory papules, pustules and nodules, typically affecting areas with the highest density of sebaceous follicles such as the face, neck and upper trunk (3). The pathogenesis of acne has been long understood as a 'chain of factors': keratinisation at the opening of the follicle, bacterial invasion, sebum alteration and inflammation (4). Dermatologists in the mid-20th century measured sebum excretion rates in acne patients and found a positive association between acne severity and sebum excretion (4-6). Sebum excretion was found to be influenced by systemic biological factors, including hormones – predominantly androgens – and, controversially, diet.

It is important to reinforce to the patient that acne is extremely common in teenagerhood. Androgens such as dihydrotestosterone (DHT) are important metabolic

regulators of the pilosebaceous unit. An increase in circulating androgens during puberty causes hyperkeratosis and abnormal desquamation of keratinocytes, leading to dilation of the follicle and impaired sebum extrusion to the skin surface (3). Hormone production peaks in the mid-teen years, explaining the increased incidence of acne in this age group, and its common resolution in adulthood. Puberty, then, is a non-modifiable cause of acne. But why do some teenagers develop acne, and others do not? Is it fully explained by differences in hormone production and regulation? Importantly, the patient wants to know: how does their diet fit into this picture?

Acne is a multifactorial disease: neither the patient's age nor diet can be considered in isolation. Genetics informs many of the pathogenic processes (the 'chain of factors') leading to acne. Virtually identical rates of sebum excretion were found in monozygotic twins yet displayed significant differences in acne severity like dizygotic twins (7). Similarly, a more recent article reported that 81% of acne variance was attributable to genetic effects; those with a family history of acne were more likely to develop acne themselves (8). The remaining 19% can be attributed to environmental factors. Genetics are thus fundamental to acne development, but do not fully explain the phenotype of acne demonstrated throughout the teenage population. To understand acne pathogenesis, one must address these environmental factors – including diet.

2. *Acne Vulgaris and Diet: Fact or Fiction?*

Interest in the relationship between diet and acne is not a recent development. In 1819, dermatologist Thomas Bateman advocated for 'the copious use of raw vegetables in the diets' to prevent acne development (4). In 1835, Green expanded on this, advising 'the individual affected (to) give up the use of wine, strong beer, spirits, coffee and stimulants of every description and heavy meals of animal foods' (4). Initial beliefs incriminated chocolate, milk and shellfish; however, early studies failed to demonstrate a consistent causal relationship between acne and diet (4). More recently, certain food groups have been associated with the 'chain of factors' leading to acne, most significantly carbohydrates with a high glycaemic index (GI) and dairy derivates (9).

To tackle the patient's question, it would be useful to outline what parts of their diet they believe influences their acne. One study suggested 58.1% of young adult participants with acne perceived diet as an aggravating factor (10). Specifically, greater GI foods (those high in fructose and glucose), added sugar, and greater milk servings were negatively associated with acne development. Studies have shown that a 'non-Western' diet (low GI, higher in fruits, vegetables and fish) have reduced acne incidence (11). The patient may elucidate some dietary behaviours or notice a pattern of acne manifestation with triggering foods. It is important, then, to understand the evidence around these dietary factors and whether or not diet modification is clinically appropriate for this patient.

One thread of research suggests high GI carbohydrates increase the risk of hyperinsulinaemia, enhancing the 'chain of factors' (9). High GI foods, including white rice and white bread, are characterised by rapid absorption and subsequent spike in blood glucose levels, increasing demands for insulin (12). Hyperinsulinaemic states increase the circulating concentration of insulin-like growth factor 1 (IGF-1) and reduce insulin-like growth factor binding protein 3 (IGFBP-3) (9). This imbalance enhances IGF-1 bioavailability to activate IGF-1 receptors (IGFR1) which stimulate lipogenesis of sebaceous glands (11). Insulin and IGF-1 also activate sterol response element binding protein-1 (SREBP-1), a lipogenic transcription factor of the sebaceous unit (12). Importantly, these mechanisms link with androgen-mediated acne development, as IGF-1 can activate 5α-reductase to amplify the conversion of testosterone to DHT (12). A positive correlation between serum IGF-1 concentrations and the number of acne lesions, as well as serum androgen level, has been found in patients with acne (12). It is less clear whether high GI foods cause inflammation or explains a propensity for bacterial colonisation. Nevertheless, based on these findings, a low-GI diet may alleviate some of the patient's acne (13). This could include switching from white bread and rice to whole-grain, or brown rice, avoiding snack foods like potato chips, and reducing sugar consumption in the form of processed sweets.

The evidence around dairy and acnegenesis is less conclusive. An association between milk consumption and acne in teenagers has been found in multiple studies (14-16). Theories of this relationship outline the same insulinotropic mechanisms: bovine IGF-1, which can bind to human IGF1R, can survive the pasteurization and homogenization process, leading to its increased bioavailability for milk consumers (12). Some studies show the strongest association between skim milk intake and acne, perhaps owing to an altered balance of hormonal constituents (androgens and nonsteroidal growth factors) introduced during processing (15). Alternatively, other studies show no significant association skimmed dairy products and acne, instead demonstrating a relationship between high intakes of full-fat dairy products and acne (14). A systematic review of 78,529 youth connected the consumption of dairy to a tepid increase in acne but warn of the heterogeneity and bias across studies that provide this correlation (17). If the patient believes there to be a worsening of acne with dairy consumption, trialling an elimination period would be worthwhile, but it is important to acknowledge that evidence is conflicting. They may notice specific dairy products have worse effects than others.

After sharing the evidence, the patient may want to eliminate these acne-aggravating foods from their diet. Before encouraging diet modification, however, the implications must be considered.

3. *Acne as a 'Social' Disease*

Although reducing high-GI carbs and dairy products is generally health-promoting, it is a form of "dieting" which increases the risk of developing eating disorders. If the patient is female, the risk is greater (18). In one study of a group of adolescents, females who dieted at a severe level (including calorie counting, reducing food quantities, and skipping meals) were 18 times more likely to develop an eating disorder compared to those who did not diet; those who dieted at a moderate level were 5 times more likely (18). Although the purpose of this diet is not to achieve thinness, dieting has psychosocial consequences including food preoccupation, irritability, fatigue, and in severe cases, delayed development (19). Importantly, teenagers with low self-esteem are more likely to diet in attempt to improve their perception of self. Teenagers with acne are already at increased risk of reduced self-esteem, anxiety and depression, which may compound when adding avenues of potential distress (2). Taking a full medical and social history and, screening for current or previous eating disorder where appropriate, would be in the patient's best interest when suggesting diet modification.

Diet and nutrition are not socially neutral concepts. Firstly, nutrition is largely determined by socioeconomic status, creating barriers for accessing healthier foods (20). If following the advice of eliminating high-GI carbs, the patient will pay an additional cost when switching from white bread to brown bread, for example. The retail price of wholegrain bread in New Zealand in 2025 is $4.30, whilst white bread is $1.81 (21-22). Patients in low-socioeconomic households (especially adolescents who have limited financial autonomy) may be unable to make these changes. It would be inappropriate to push major dietary changes to the patient without being mindful for the cost of living. Secondly, food is a marker of cultural identity. For example, *kai* in Māori culture is essential for upholding *mana, manaakitanga* (reciprocity of kindness, respect and humanity), and reinforcing *whakapapa* (23). Modifying a patient's diet requires consideration of what food means to them as it may put them in a position where they reject certain foods that serve a sociocultural purpose. If the patient would like to trial dietary changes, it is important to encourage healthy engagement with social and cultural eating to avoid isolation from other important components of their wellbeing.

If the patient does not want to pursue diet modification, what else can be offered? The patient may express already having tried skin products, and acquiring this information can help guide suggestions. Skin care products have a high safety and generally good tolerance, with evidence of effectiveness in reducing signs of inflammatory lesions, uneven skin, dilated pores and hyperseborrhea when used consistently (24). Suggesting products with 'active ingredients', antiseptics, oil-controlling substances and softening keratin components, (such as salicylic acid) may prove sufficient to improve the patient's skin over time (25). In some moderate to severe cases oral prescription medication may be necessary, particularly if the patient has already trialled topical antibiotics or retinoids. Isotretinoin, for example, is an oral retinoid prescribed for management of severe acne. If this is clinically indicated, it is important to advise the

patient of side effects (including an initial flare up) and care around concomitant use of active skin ingredients.

4. Conclusion

Acne is a biopsychosocial condition. Reassuring that acne is extremely common at this age should be the launch pad to address factors affecting skin health. It is important to not dismiss the patient's acne as something they will 'grow out of'; rather, adopting changes may help their symptoms and improve their self-esteem. When it comes to diet, caution must be taken around the conflicting evidence. It is better for the patient to be informed, particularly where the data is murky, to provide them with the autonomy to make decisions that are right for them (including alternative options to diet modification, like skincare and medications). Importantly, one must consider the psychosocial aspects of diet modification in a teenager, including socioeconomic burdens, culture and relationship with food, and create a care plan tailored to the individual. The relationship between diet and acne will be revealed with more research, but for now, considering the acne as a multifactorial process is the best approach.

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