Acne in adolescence: A role for nutrition?

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KEY POINTS

- Acne prevalence varies substantially between populations and is low in non-Westernised societies consuming traditional diets.
- A unifying feature of traditional diets is low glycaemic load.
- High-glycaemic-load diets have recently been implicated in acne aetiology due to their ability to affect insulin demand, androgen bioavailability and insulin-like growth factor-I activity.
- The beneficial results of a low-glycaemic-load diet on acne observed in a recent trial require confirmation in larger studies.

During the past century, much controversy has surrounded the subject of diet in acne management. In the 1930s, major dermatology textbooks advocated dietary restrictions for acne patients based upon ‘clinical experience . . . that [suggests] a diet high in carbohydrates and sweets seems to make some acne cases worse’. However, apart from the individual impressions of physicians, there was little evidence to support dietary practices for the control of acne. Forty years later, dietary advice was removed from standard texts, and the consensus has since been that ‘diet plays no role in acne treatment in most patients’. Nonetheless, many acne patients continue to believe that acne is exacerbated by diet, and a recent survey found that 41% of final-year medical students at Melbourne University regarded diet as an aggravating factor.

Few studies have examined the diet and acne connection, and those that have, show major design faults. Fulton et al. in a crossover single-blind study found no effect of chocolate on acne when compared with a placebo bar. However, a later examination of the ingredients in the placebo bar revealed that the fatty acid composition and sugar contents were virtually identical to that found in the experimental treatment. Anderson examined the effect of the daily consumption of chocolate, milk or nuts and found no effect on acne. However, the present study has been criticised for its small sample size (minimum of 3 subjects per food), its short duration (7 days) and its lack of controls. Although the experiments of Fulton et al. and Anderson are frequently cited as evidence that diet plays no role in acne development, a major limitation of both studies was that they concentrated on single foods with no background dietary analysis.

Variations in acne prevalence worldwide have prompted researchers to question the natural development of the disease. For instance, rates of acne in Kenya, Zambia and Peru are reportedly lower than in the black and Hispanic populations of Western countries. In a recent observational report, Cordain et al. attributed that the absence of acne in two non-Westernised societies (the Kitavan Islanders of Papua New Guinea and the Ache’ hunter-gatherers of Paraguay) to environmental factors, mainly local diets, which are devoid of high-GI (glycaemic-index) carbohydrates. This concept is an extension of an earlier hypothesis put forward by Schaefer, who reported the emergence of acne in the Eskimos of North America following the adoption of a Western lifestyle. Schaefer proposed that the increase in acne prevalence in Eskimo groups was the result of the ‘shift to refined, ... rapidly absorbable carbohydrates’. This is in stark contrast to their earlier diet, which was composed primarily of meat and fish. Although Russian settlers introduced the Eskimos to basic agriculture and carbohydrate foods (e.g. barley, buckwheat, cabbage and potatoes) some 70–100 years ago, these carbohydrates were generally low in GI and did not replace animal protein as the main source of energy. The emergence of acne to varying degrees in Eskimo groups appeared to coincide with the increase in the annual per capita consumption of refined sugar and flour, while the per capita consumption of protein derived from animal sources showed an inverse relationship. Since the relatively recent introduction of refined carbohydrates to the diet, the Eskimos have also demonstrated faster growth (increased final height), earlier puberty and dramatic increases in the incidence of obesity, diabetes and heart disease.

A unifying feature of traditional diets is that they are naturally low in glycaemic load. As the GI can only be used to compare foods of equal carbohydrate content, the glycaemic load was later developed to characterise the glycaemic effect of whole meals or diets (GI × available dietary carbohydrate). The glycaemic load may be interpreted as a measure of the blood glucose- and insulin-raising potential, as it represents the rate of carbohydrate absorption (indicated by the GI) and the quantity of carbohydrate consumed. The glycaemic load may be modulated by altering the absolute amount of carbohydrate consumed or by selecting foods using the GI concept. Accumulating evidence suggests that the glycaemic load of Western diets has increased over recent years: (i) due to an increase in carbohydrate consumption as a consequence of dietary recommendations to decrease dietary fat; and (ii) as dietary trends appear to favour high-GI foods.

High-glycaemic-load diets have recently been implicated in acne aetiology due to their ability to increase insulin resistance and promote the emergence of acne. Pimple formation is triggered by androgen bioavailability, and insulin resistance is commonly associated with elevated androgen levels. The exact mechanism by which high-GI foods exacerbate acne is not well understood; however, one hypothesis is that consumption of high-GI foods may alter insulin sensitivity, thereby increasing insulin levels and androgen bioavailability.

Conclusions and Future Directions

Further research is needed to investigate the role of diet in acne management. Future studies should employ larger sample sizes, longer durations and more rigorous experimental designs. In addition, the relationship between dietary habits and acne severity should be explored, as well as the potential role of specific nutrients and food components in the modulation of acne.

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References

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demand and other factors associated with insulin resistance (e.g. hyperphagia, elevated non-esterified fatty acids, obesity). Clinical and experimental evidence suggests that insulin may increase androgen production and effect, through its influence upon steroidogenic enzymes, and sex hormone-binding globulin (SHBG) production. Furthermore, insulin has been shown to decrease a binding protein for insulin-like growth factor-I (IGF-I), which may facilitate the effect of IGF-I on cell proliferation. Altogether, these events may influence one or more of the four underlying causes of acne: (i) increased proliferation of basal keratinocytes within the pilosebaceous duct; (ii) abnormal desquamation of follicular corneocytes; (iii) androgen-mediated increases in sebum production; and (iv) colonisation and inflammation of the comedo by Propionibacterium acnes.

Support for a role of insulin in acne development can also be found in the high prevalence of acne in women with polycystic ovary syndrome (PCOS), a condition associated with insulin resistance, hyperinsulinaemia and hyperandrogenism. Insulin resistance is believed to be the underlying disturbance in PCOS, as it generally precedes and gives rise to the cluster of endocrine abnormalities that characterise PCOS (increased androgens, increased IGF-I and decreased SHBG). Treatments for PCOS now include oral hypoglycaemic agents which improve insulin sensitivity, restore fertility and alleviate acne.

The extent to which acne is related to the typical Western diet is controversial, but the future holds promise of a shift in paradigm. A recent study by the authors has demonstrated that a low-glycaemic-load diet, which mimics the diets of acne-free populations, significantly reduced acne lesion counts and hormonal aspects of acne (e.g. insulin, androgen bioavailability, free IGF-I). Furthermore, the improvement in clinical and endocrine parameters was significantly greater than the control arm, which followed a high-glycaemic-load Western diet. Although confirmation of the diet–acne hypothesis will require larger-scale experiments, the present study suggests that the institution of a non-Westernised diet, adapted for modern times, may have a therapeutic effect on acne. As a basic principle, this type of diet should contain minimally processed carbohydrate-based foods, including a wide variety of fresh fruits, vegetables, wholegrains, lean meats, fish and seafood.

REFERENCES


