an oversimplification. An indigenous group has a common genetic ancestry, the strongest determinant of familial disorders like acne. In addition, such societies share environmental conditions other than nutrition, including climate, sun exposure, work, stress, physical activity, and local microbes.

In their thoughtful editorial comments, Drs Thiboutot and Strauss[5] point out that the authors failed to test the western diet for acnegenicity in the native groups. In addition, acne efficacy of the primitive diet in westerners was not shown.

Whereas low-glycemic diets are appropriate for patients with polycystic ovary syndrome and insulin resistance, data are nonexistent that diet causes or cures acne.

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Diet and Acne Redux

The science of nutrition has produced dramatic findings over the past few decades. Of particular note is the expansion of knowledge about essential fatty acids during the 1980s. Also, profound changes in the concept of daily nutritional requirements find the United States replacing "recommended daily allowances" with "dietary reference intakes." This reflects the consideration of optimal nutrient levels rather than minimum daily requirements. The vitamin paradigm changed forever when neural tube defects declined radically with folic acid supplementation.[1] No longer do we think only of preventing nutritional deficiency diseases. Now we are learning to provide optimal nutrient intake for optimal function.

Cardiologists have embraced this concept and now prescribe vitamin B_{12}, vitamin B_6, and folate to bring down elevated homocysteine levels in patients at risk for stroke and myocardial infarction.[2] Although causality has not been proved, study findings are highly suggestive, and supplementation carries few risks. The American Heart Association also acknowledges the benefit of daily fish oil supplementation for some patients.[3] However, few physicians in other fields seem to take advantage of recent discoveries in nutrition and apply them clinically.

Dermatology has been particularly tied to older nutrition dogma with regard to acne and diet. The article by Cordain et al[4] in a recent issue of ARCHIVES should serve to awaken us to the relevance of nutrition to skin disease and stir us to review the dogma. The major textbooks of dermatology tend to view diet as irrelevant to the treatment of acne. The primary references to which the texts refer are both more than 30 years old.[5,6]

In 1971, Anderson[5] observed 27 college students on a "typical high-carbohydrate dorm diet." The students believed that specific foods caused inflammatory flares within 3 days of ingestion. They received the culprit foods on a daily basis and returned daily for facial mapping of lesions. None flared. While the uniformity of response was impressive, the study had a few glaring flaws. The sample size was fairly limited. The study was neither controlled nor blinded. The article was not peer reviewed by dermatologists (published in the *American Family Physician*). Most importantly, given the effects of chronically elevated insulin posited by Cordain et al,[4] the baseline diet may have obscured the findings.

In 1969, Fulton et al[6] explored the effect of chocolate on acne by using "pseudo-chocolate" bars made with 28% partially hydrogenated vegetable oil as the control. With our 2002 lens, we can see that the high proportion of *trans* fats in the control bar limits the usefulness of the study. *Trans* fats compete with essential fatty acids in the production of prostaglandins and appear to significantly contribute to inflammation.[7]

In the nutrition literature, evidence supporting dietary effects on health continues to mount, and dermatology is no exception. Many of our patients' skin conditions are affected by what they eat. It may be time for us to open our minds and our nutrition textbooks.

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Omega-3 Fatty Acids and Acne

I read with great interest the recent article by Cordain et al.[1] This group makes a strong argument for the involvement of diet-induced hyperinsulinemia in the pathogenesis of acne vulgaris. In addition to the glycemic load of the typical western refined-food diet, it is also important to note that the western diet typically includes a much lower intake of omega-3 fatty acids, an

excess of proinflammatory omega-6 and *trans* fatty acids, and a reduced intake of dietary antioxidant vitamins. The current ratio of omega-6 to omega-3 fatty acids in the western diet reaches 20:1, while through evolution and in a traditional hunter-gatherer diet it is closer to 1:1.[2] Fish, wild game, and wild plants have much higher levels of omega-3 fatty acids than do refined western foods.

In addition to being higher in omega-3 fatty acids, the diets consumed by the Kitavans and Aché may also include increased quantities of plant-derived antioxidant vitamins, minerals, and phytochemicals that support antioxidant pathways. Research shows that omega-3 fatty acids can increase insulinlike growth factor binding protein 3 in animals[3] and decrease insulinlike growth factor 1 in healthy humans.[4] Therefore, in support of the ideas of Cordain et al,[1] a diet high in omega-3 fatty acids may also be involved in the prevention of the hyperkeratinization of sebaceous follicles. In addition, the involvement of proinflammatory leukotriene B_4 (LTB_4) in the pathogenesis of acne has recently been described; administration of a novel LTB_4 blocker led to a 70% reduction in inflammatory acne lesions, improvements that correlated with a reduction in proinflammatory lipid levels.[5] The anti-inflammatory properties of omega-3 fatty acids, including LTB_4 inhibition, are well known.[1] Arachidonic acid, the major dietary omega-6 fatty acid, is a precursor to the manufacture of LTB_4,[5] indicating that dietary choices may play a role in inflammatory acne lesions. It is possible that dietary omega-3 fatty acids could have a synergistic effect on any potential benefit of adhering to a diet with a low glycemic load.

Clearly, further research is required. In particular, a more detailed dietary analysis of the Kitavans and Aché may help determine if additional relevant differences occur between the western diet and that consumed by nonwesternized populations.

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In reply

In his letter, Dr Logan proposes that a lower dietary omega-6–omega-3 ratio in the Kitavan and Aché people may play a synergistic role along with a lower glycemic load in preventing the development of acne via reductions of proinflammatory eicosanoids. This hypothesis is certainly reasonable given recent evidence showing that an LTB_4 blocker led to a 70% reduction in inflammatory acne lesions after 3 months.[1] We have previously reported that the Kitavans indeed maintain a significantly lower dietary omega-6–omega-3 ratio than do western populations[2] and that a lower dietary omega-6–omega-3 ratio is characteristic of virtually all hunter-gatherer diets.[3] Another feature that distinguishes Kitavan and Aché diets from western diets is the absence of milk, which exhibits a low glycemic index but paradoxically is highly insulinotropic.[4] As with high-glycemic-load carbohydrates, dietary interventions will be required to assess the effectiveness of any nutritional treatment on the development of acne.

In her letter, Dr Treloar implies that "the emperor wears no clothes" by pointing out to the dermatology community that the nearly universal assumption that diet and acne are unrelated is based largely on 2 marginal and poorly designed studies that are now over 30 years old.[5,6] Her conclusion is not unique; a previous report summarized, "There are few, if any, well-controlled studies on the effects of various dietary factors in acne."[7] Although the single article by Fulton et al[5] has been often cited as the definitive work dissociating diet and acne, serious design flaws in the study were identified more than 25 years ago showing that the fat and sugar content of the placebo bar did not differ significantly from chocolate.[8] If high-glycemic-load carbohydrates represent the environmental trigger for the development of acne in genetically susceptible individuals, then the double-blind study by Fulton et al[5] would not have been able to detect a treatment effect because the glycemic load of the placebo and treatment were nearly identical.

Many early 20th-century anecdotal observations by dermatologists and physicians have linked sugars and refined cereals to acne.[9,10] Unfortunately, at the time, these observations lacked objectivity because the mechanistic understanding of the endocrine and cytokine basis underlying the development of acne was in its infancy and because well-controlled dietary interventions were rarely or never performed. Regrettably, we still cannot confirm or deny these early observations because well-controlled dietary interventions have yet to be conducted. In the 21st century, we now have the tools and knowledge to adequately test the diet-acne hypothesis—be it high-glycemic-load carbohydrates, insulinotropic dairy products, trans *fatty acids, a high dietary omega-6–omega-3 ratio, or all of the above.*

In science, when observable facts are inconsistent with prevailing theory, the facts are not necessarily thrown out or ignored. Frequently, new facts make prevailing theory untenable. Our report[11] demonstrates that an inconsistency may exist between the observable facts (the total absence of acne in nonwesternized populations) and the prevailing theory (that diet and acne are unrelated). This information should not be viewed as an "unwelcome return of the acne diet," but rather should provide a theoretical construct for critically reexamining the diet-acne hypothesis. As Dr Treloar has pointed out, the current foundation for rejecting the diet-acne hypothesis is virtually nonexistent and relies almost entirely on two 30-year-old, poorly controlled studies. No amount of discussion in "letters to the editor" will ultimately resolve this issue. The currency of science is good data generated from well-controlled experi-