

Acne and milk, the diet myth, and beyond

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Is acne related to the ingestion of dairy products? It is not a new idea. It goes back to the early days of the last century and beyond. The counterclaim, that there is no relationship between diet and acne, has reached mythic proportions. It has never been substantiated, but it is repeated as gospel in all major dermatology textbooks. Epstein,¹ commenting on contributions on the subject by Waisman, Bickers, and Rosenberg, trod the middle road. Inexplicably asserting that “Controlled studies indicate that foods have no effect on acne,” he nonetheless allowed, “the patient should receive any assistance that dietary control may impart.”¹ In a review of 274 clinical trials of acne, the massive 2001 Evidence Report on the Management of Acne² found one solitary paper that mentioned diet, but no trial reported specifically on patients’ diets.³ Cordain’s work in two isolated communities with minimal processed carbohydrate and no dairy intake suggests a mechanism that involves insulin-like growth factor (IGF-1).⁴ Closer to home, Perricone’s acne-free program is almost entirely dairy-free.⁵ The entire subject has recently been beautifully reviewed by Wolf et al.⁶

As a long-term believer in the relationship between diet and acne, I encouraged Dr Walter Willett and his colleagues at the Harvard School of Public Health to explore the possibility. In this issue of the *Journal*, we present evidence of an association between milk consumption and acne. The first paper,⁷ published here, is based upon the Nurses Health Study II cohort. A second and third will be forthcoming, and their data are based on the Growing Up Today Study, consisting of offspring of the Nurses Health Study II subjects.

These papers are not the end of the story; rather, they signal the need for a new beginning. Research into the etiology of acne has been accelerating over

the past few years, and several lines of research may be stimulated by these findings. Thiboutot recently demonstrated that human skin is a steroidogenic tissue.⁸ Can bovine hormones act as substrates for the enzyme systems of the pilosebaceous unit? Likewise, dietary influences upon carbohydrate metabolism and IGF-1 noted by Cordain⁴ will need to be revisited to consider the influences and interactions of bovine dietary source IGF-1. Zouboulis’s highly innovative techniques of sebocyte culture⁹ and the investigation of isolated pilosebaceous unit function by Sanders et al¹⁰ will both be essential tools to further investigate the effect of these bovine hormones on a molecular and cellular level.

The papers from the Harvard School of Public Health establish an *association* between milk consumption and acne. But how could milk *cause* acne? Because drinking milk and consuming dairy products from pregnant cows exposes us to the hormones produced by the cows’ pregnancy, hormones that we were not designed to consume during our teenage and adult years. It is no secret that teenagers’ acne closely parallels hormonal activity,¹¹ and the biochemical links between hormones and pilosebaceous activity and acne are being more closely defined every year.¹² So what happens if exogenous hormones are added to the normal endogenous load? And what exactly is the source of these hormones? Consider that, in nature, milk is consumed from a mother, whether human or bovine, until weaning occurs. Normally, the mother then ceases lactation before the next pregnancy occurs—so that consuming milk from a mother pregnant with her next offspring is not a common occurrence. We’ve all seen nature films of animals chasing their offspring away to encourage weaning at the appropriate time. Further, in nature the offspring consumes only the milk of its own species—but both of these natural rules are broken by humans. Viewed objectively, human consumption of large volumes of another species’ milk, especially when that milk comes mainly from pregnant cows during the human’s normally post-weaned years, is essentially unnatural.

On the producers’ side, economies of scale and market efficiencies have become the drivers of

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the dairy industry, so much so that modern herd management techniques result in 75% to 90% of marketed milk and milk products being derived from pregnant cows. A heifer is bred and produces no milk until she is post-partum with her first calf. She feeds her calf for about a month, and then she is put on the milking line. The calf becomes veal or goes on artificial feed; the cow is bred at her first heat, if possible (about 6 weeks post-partum) and milking continues during that 10-month pregnancy, until the cow is allowed to "dry" a few weeks before delivery. "Freshening," the production of a new fresh milk supply post-partum, results from this cycle. When a cow becomes less efficient than her sisters, usually after five pregnancies or so, she is sold for butchering.

This sequence results in milk that contains not only placenta-derived progesterone but also other dihydrotestosterone (DHT) precursors, including 5α -pregnandione and 5α -androstenedione. These compounds are only a few enzymatic steps away from DHT (the accepted prime acnegen) and the enzymes required to mediate the change are present in the human pilosebaceous unit.¹³ It had been hoped that 5α -reductase inhibitors might block the final common pathway to DHT that these enzymes feed into. Unfortunately, even if 5α -reductase is totally blocked,¹⁴ the blockade is of no value because these compounds have already undergone 5α -reduction—in the bovine mammary gland. These precursors arrive at the pilosebaceous unit with no need for further 5α -reduction, a situation that exposes humans to potent agonists for which we are unprepared by any evolutionary defense mechanism.

It is not, however, just the steroid hormones in milk that are of concern. Milk is a very complex substance. It contains prolactin, somatostatin, growth hormone releasing factor-like activity, gonadotropin-releasing hormone, luteinizing hormone, thyroid-stimulating and thyrotropin-releasing hormones, numerous steroid hormones, insulin, epidermal growth factor (EGF), nerve growth factor (NGF), IGF-1 and -2, transforming growth factors (TGFs), vitamin D, transferrin, lactoferrin, many prostaglandins including $F2\alpha$, erythropoietin, bombesin, neurotensin, vasoactive intestinal peptide, various nucleotides, cyclic adenosine monophosphate and guanosine monophosphate, B-casomorphins, and even relaxin.¹⁵ The concentrations vary among species and with the temporal relationship to parturition, but it should surprise no one that milk contains such a heavy complement of growth-enhancing hormones. *Milk is, after all, specifically designed to make things grow.*

The most likely of all candidates for costimulation (with the steroid hormones) of pilosebaceous func-

tion and dysfunction is IGF-1. The blood level of IGF-1 in prepubertal, pubertal, adolescent, and early adult humans very closely approximates the prevalence curve of acne in this population. IGF-1 is present in ordinary milk and in milk produced in increased volume with the use of bovine somatotropin (BST), also known as recombinant bovine growth hormone (rBGH). BST/rBGH also marginally increases the amount of IGF-1 in milk.¹⁶ Indeed, Adebamawo et al also suggest that ingesting milk may stimulate endogenous IGF-1,⁶ so all these relationships need further investigation.

The next stage in the delineation of possible exogenous hormonal contributions to the pathogenesis of acne should be a qualitative and quantitative determination of all the steroid hormones in all dairy products, using modern methods to update Darling and Laing's work of thirty years ago.¹⁷ That should allow us to formulate meaningful dietary recommendations and allow the design of dietary trials to test the hypothesis that there is an association between dietary dairy intake and diseases arising in hormone-sensitive tissues/organs, such as skin, breast, and prostate gland and, one hopes, sort out which components of milk may be the prime culprit(s). Ultimately, we need to learn what degree of acne prevention can be achieved while still ensuring that the population consumes a nutritious diet. Adding to the importance of this area of research is the growing concern about hormonal stimulation of malignancy in other hormone-responsive tissues. Dairy products have been implicated as a possible factor in the etiology of prostate cancer in several large epidemiological studies,¹⁸ but not in all.¹⁹ The possible influences of dairy hormone in breast cancer are likewise unclear and in need of further definition.^{20,21}

The next few years will be fascinating for those of us interested in hormones and "the blight of youth." One wonders what the impact will be upon our patients, our practices, and the industries that make milk, hormones, and acne products. Time alone will tell.

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