# In This Issue

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## Lights at the End of the Tunnel

After years of taking care of patients with psoriasis, it has become quite obvious to me that a family of factors (genetic, environmental, and infectious) regulates its expression. The implication of this is that we cannot expect a single animal model to reproduce the disease or a single target to serve all novel therapies. In fact, reports from the last several years about various biologic response modifiers demonstrate clearly that some patients respond well, while others (often my patients) seemingly do not, thus supporting the concept of multiple genetic factors. Into this confusion of pathogenic models, we now have In this issue, a brief paper from Joanna Shepherd and her associates from the Division of Genomic Medicine at the University of Sheffield (p 605). They report that mice on a BALB/c background with a genetic absence of the IL-1$\alpha$ receptor antagonist develop a scaling eruption on the pinnas of each ear. This eruption, which is acquired as the mice develop adulthood, has a clinical appearance that resembles psoriasis. Moreover, histological changes include acanthosis, hyperkeratosis, and infiltrates of both neutrophils and lymphocytes, all in the right place. This eruption is limited in its distribution (think elbows and knees or palms and soles), and it does not occur in all strains of mice. At the same time, some strains have an associated arthritis and others have an associated arterial inflammation. We predict that ultimately there will be many models of psoriasis because its pathogenesis may not be singular. As we reach the end of the tunnel, there is more than one light.

## *Deja Vu* all Over Again (Attributed to Yogi Berra, New York Yankees)

Thirty-five years ago, most dermatologists and prospective dermatologists in the USA invested 2 years of their lives in the service of their country, some as trainees at the National Institutes of Health and many others as practising physicians in the military. One of the realities of military service was the need to deal with the many African-American servicemen who suffered from the hair follicle disorder pseudofolliculitis barbae. We knew at the time that a closely cropped beard would lead (after several weeks in transition) to complete resolution, but this was largely unacceptable to our military superiors. You can imagine the tension, especially for military personnel who had already survived the horrors of one year in Viet Nam and were "marking time" in their terminal year of service. Not only did we know that once hair shafts were liberated from the follicle, the disorder would remit, but also that the presence of tightly coiled hair shafts was not the only factor, since some men had the disease and others did not. Thirty-five years later, an impressively multinational collection of investigators and clinicians have come together to provide at least one answer. In this issue, Winter and Schweizer and their colleagues from around the world demonstrate that a single nucleotide polymorphism for a protein expressed in the inner root sheath of hair follicles plays a major role in the expression of pseudofolliculitis (p 652). The idea is that this "defect" disrupts the "guiding" nature of the follicle itself. It is particularly noteworthy that the patients who served as the core of the well-defined clinical populations examined in this study are the descendants of the patients we struggled to help in 1970. This paper also illustrates graphically the complex fashion in which several genetic and environmental factors may conspire to produce disease. We salute the service personnel who work under extraordinarily difficult conditions to serve, the military physicians who help to care for them, and of course the scientists who conduct experiments that address important questions.

## Out of Left Field

It is appropriate to continue the baseball allusion, since the unanticipated seems to come "out of left field" and since Japan and Cuba are the only major countries in which baseball remains the national pastime. In this issue, a surprising paper from Masaaki Yoshikawa and his associates at two prominent agricultural institutions in Japan provides surprising data to indicate that an orally administered soy-derived peptide inhibits the acute alopecia caused by etoposide to a substantial degree (p 848). First, those who take care of patients with hair disorders can attest to the incredible importance of hair in our cultures. I suspect that money spent on hair in Japan, the USA, or in Europe would exceed by far the entire economy of Cuba, and it is certainly larger than the economy of baseball worldwide. Likewise, the capacity of anticancer drugs to cause alopecia is both recognized and feared. But a derivative of soy beans? Orally administered? And from experts in Food Science? Who would have thought? This process of discovery is exactly what members of Congress in the United States need to know, that the path of discovery is unpredictable and that only under unusual circumstances is it useful to "plan" for it. We tip our hat to the inquisitive and obviously well-informed scientists from Japan who have put something new together, to leaders in our own institutions who emphasize the importance of basic research, to our colleagues in the USA who are attempting to measure "the burden" of skin disease (including alopecia), and to an imaginative Editor, who has the insight to accept a letter from "left field."

DOI: 10.1111/j.0022-202X.2004.22348.x