I appreciate the opportunity to provide a commentary on this article and am pleased and gratified that it has been so often cited. I am doubly fortunate because of your request for the above abstract, since the publishing journal in 1963 somehow omitted printing the abstract provided with the paper at the time of its submission—the only time it has ever happened to me.

"The work reported, performed while I was at the National Cancer Institute in the Dermatology Branch, was undertaken because of the need for more information on determinants of hyperplasia in benign and cancerous processes. More particularly, it was started to understand better the events causing the immense degree of epidermal cell exfoliation in psoriasis, a disease that still plagues over a million Americans today.

"The techniques available then were tedious and time consuming, requiring resolute counting of mitoses, planimetric measurements, and arithmetic computing. These were doggedly carried out by Thomas Ekel, who, because he was also an accomplished semiprofessional bridge player, must have been by nature exceptionally exacting and deliberate.

"The reasons for the article having been cited so much, in my judgment, are: (1) It represented one of the early works relevant to later work that aimed to define cell cycles, and cycling and noncycling cell compartments of various epithelia; (2) The conclusions drawn by us from data obtained by those now-antiquated methods have retained their validity over time; and (3) Our findings in regard to the epidermal-dermal relationships were pertinent to the general phenomena of epithelial-mesenchymal interrelationships, in that they suggested that an epithelial event (hyperplasia) was secondary to connective tissue proliferation.

"A consequence of our findings, and those of others that called attention to excessive epidermal cell replication in psoriasis, has been a wide search for and trial of antimitotic agents, other than methotrexate, for treating the disease. Unfortunately the search has not yet proven fruitful, and many of us are prepared to consider that further search may be futile. As our paper in 1963 suggested, epidermal hyperplasia probably is consequent to events and changes in the underlying connective tissue. Therefore, to treat psoriasis with drugs that restrain epidermal replication without diminishing the dermal determinants may be somewhat like bailing out a leaking boat without attempting to seal the leak. While retrospectively this may seem elementary, to assume such apparent logic at an earlier time would have required a kind of clairvoyance that few would dare to admit to or attempt to defend."