expression [7], because seborrheic keratosis is a tumor arising in aged skin, where already well-documented dermal aging processes are responsible for altered dermal environments. The molecular nature of these factors remains to be elucidated.

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REPLY

We thank Nindl et al for their interest and comments on our paper. Like them, we have studied the keratin expression patterns in seborrhoeic keratoses and find them to be very heterogeneous (as is their standard histologic categorization). We look forward to the publication of their study but would like to make the following general observations and responses to their comments.

1) Suprabasal presence of keratin 5 is to be expected, as this keratin polypeptide is present throughout normal epidermis. However, an unexpected finding in our work was the finding of K5-14 epitope negative cells suprabasally.

2) We found K1 and K10 staining to be heterogeneous, with populations of suprabasal cells expressing either K5-14 or K1-10 phenotypes in complementary patterns, that is, K1-10 cells were not always co-expressing K5-14 epitopes, which is unusual.

3) Unlike Nindl et al, we failed to demonstrate in seborrhoeic warts any expression of simple keratins 8–18 using the same multiple battery of strong highly specific monoclonal antibodies that we used in our paper. We await to see the immunologic reagents they used; however, the importance of using multiple strong reactive keratin monospecific antibodies must be emphasized. We have examined more than 40 seborrhoeic warts (British Society of Dermatopathology Meeting, July 1988) and found no evidence of expression of simple epithelial keratins 8 and 18 at all using multiple well-characterized monospecific antibodies. We have also examined a series of dysplastic and malignant warts [18] in renal transplant recipients [Proby CM, Churchill L, Purkis PE, Glover M, Sexton CJ, Leigh IM: Keratin 17 expression as a marker for epithelial transformation in viral warts (submitted)] and similarly found no evidence of keratins 8 and 18 except in poorly differentiated invasive tumours [3]. Thus the finding of keratins 8 and 18 in seborrhoeic warts has to be set in this context.

In seborrhoeic keratoses, therefore, we observed highly heterogeneous alterations in the orderly progression of keratin changes normally seen in the skin but such changes were consistently restricted to keratin phenotypes seen in stratified epithelium, and in particular did not comprise expression of simple epithelial keratins.

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Korean Society for Investigative Dermatology—Happy First Anniversary

To the Editor:

The Korean Society for Investigative Dermatology (KSID) held its First Anniversary meeting in Seoul on March 21, 1992. The meeting was attended by approximately 140 investigators (80 dermatologists, 30 residents, and 30 basic scientists). Considering the fact that South Korea has only about 600 dermatologists, this is a remarkable start for this new society.

The meeting consisted of a main lecture, four teaching sessions, a symposium on PCR, free communications, and a poster exhibition. I was pleased to be invited as the featured guest lecturer. The audience was clearly eager to learn about the latest developments in molecular biology research. All in all, the meeting was well organized and reported considerable enthusiasm about the future development of investigative dermatology in South Korea.

On a personal note, I was extremely pleased to be appointed as an honorary member of the KSID. Incidentally, I am the second honary member; Professor Imamura is the first.

The President of KSID, Professor Young Pio Kim, and Chairman of the Board of Directors, Professor Jung Bock Lee, asked me to convey their warmest regards to the Directors and members of the SID.

I hope that this brief note serves as a catalyst for the SID and ESDR to develop closer ties with our Korean colleagues, and help them to foster dermatologic research in their country under the auspices of the KSID.

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