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REPLY

We thank Dr. Didierjean and colleagues for their comments, and for pointing out their findings concerning the relative amounts of IL-1α and β in psoriasis.

We absolutely agree that different extraction and assay systems may give variable results, and wish to emphasize the point that the naming of an analyte as being identical to a given substance should only be done if evidence for identity is beyond doubt. Although it seems likely that the biologically active compound we have reported in normal skin is IL-1α, we have been careful to term it “IL-1α-like” in the paper as its structure is not absolutely beyond doubt. Such caution is further justified by the fact that interleukin 1α mRNA has not yet been identified on analysis of uncultured samples from human skin, as Dr. Didierjean and colleagues have outlined.

We have also found a discrepancy between the levels of immunoreactive and biologically active IL-1β in extracts of human heel stratum corneum. Little or no biologically active IL-1β has been detected by us in such samples, as determined by bioassay in combination with HPLC or neutralizing antibodies and reported in the paper under discussion [1]. However, substantial IL-1β immunoreactivity was detectable in PBS extracts of normal heel stratum corneum, by ELISA [2]. Along similar lines, immunoreactive tumour necrosis factors (TNF) α and β, and interferons (IFN) α and δ, were detected in aqueous extracts of psoriatic lesional stratum corneum, but no TNF or IFN activity was detectable in relevant bioassays [2]. Furthermore, Sticherling, Schröder, and Christophers have reported the presence of IL-8 immunoreactivity in normal skin, but were not able to demonstrate IL-8 biologic activity in these samples, sequential HPLC eventually yielding immunoreactive material that was biologically inactive and chromatographically distinct from IL-8 (reported to the European Society for Dermatological Research, Turin, 1990). These few examples therefore support the note of caution of Didierjean et al concerning interpretation of the results of IL-1 analysis in skin, and extend the need for caution to the analysis of other cytokines as well.

Finally, we agree that the IL-1α-like material obtained from heel stratum corneum might originate in part from sweat. However, sweat seems a less likely source of the biologically active material recovered by sampling of thigh skin by the skin chamber method.

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Minoxidil Stimulates Mouse Vibrissae follicles in Organ Culture

To the Editor:

We read with great interest the article by Buhl et al [1] in the March 1989 issue. The authors describe a new system for studying the biology of mouse vibrissae follicles. They demonstrated in vitro that minoxidil-treated follicles grew significantly longer than controls and showed increased proliferative activity and differentiation of vibrissae follicle cells by means of 3H-Thymidine, 35S-cysteine and 3H-glycine uptake. The results of their experiments indicate direct effects of minoxidil on hair follicle cells in vitro.

Minoxidil, a drug for treatment of cardiovascular hypertension, has shown positive effects on hair regrowth in male pattern baldness [2]. Therefore, we investigated the influence of topically applied minoxidil (3% alcohol solution, 3 ml daily) on the proliferative activity of plucked anagen hairs by means of DNA-flow cytometry (DNA-FCM).

Forty-two healthy men with male pattern baldness underwent this therapy and before months 3, 6, and 12 hairs were plucked from the frontal scalp region. Hair shafts were cut off and anagen hairs with intact outer root sheath (ORS) were carefully selected and only those parts surrounded by ORS were prepared for DNA-FCM analysis.

DNA-FCM is a valuable tool measuring cell cycle kinetics of hair segments [3] and data gained from plucked anagen hairs reflect the proliferative activity of keratinocytes of ORS (ORSK) [4]. The calculation of the proliferation index (S + G2 + M-phase cells) re-
vealed a significant (p < 0.01, Student t test, paired samples) continuous increase (month 3, 34%; month 6, 82%; and month 12, 105% on average) during minoxidil treatment (Fig 1), whereas controls (alcoholic solution without minoxidil) remained unchanged. The proliferation index increased in 78% of patients after 3, in 88% after 6, and in 100% after 12 months, compared to data before therapy. It must be mentioned, however, that after 3 months, 100% of patients, after 6 months, 78% of patients, but after one year, only 43% of patients participated in the study. Additionally, despite the increasing proliferation index, no regrowth of terminal hair was clinically noted so far.

Our results yield evidence that topically applied minoxidil influences the proliferative activity of ORSK in vivo suggesting, in agreement with Buhl et al [1], a possible direct effect of minoxidil on hair follicle cells. Any changes of the blood flow, however, can not be excluded.

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