Neutrophil and Eosinophil Chemotactic Factors Released from Human Keratinocytic (Hair Follicular) Tumor Cells

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Key Words
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Introduction
We often encounter funny but very interesting phenomena in our skin clinic. One example is kerion, i.e. fungal infection of the scalp. We observed heavy eosinophil, but not neutrophil, infiltration in diseased hair follicles in the ocean of neutrophils (abscess) (2/5 cases in my recent experience). Why do eosinophils infiltrate the hair follicles? Do hair follicles release an eosinophil chemotactic factor (ECF)? How about a neutrophil chemotactic factor (NCF)? In order to solve these questions, I investigated these factors using in vitro established human hair follicular tumor (trichilemmoma) cell lines (K-TL-1 and TK-TL-3) and several trichilemmomas.

Material, Methods and Results
One cell line, K-TL-1, was established from a trichilemmoma which had heavy neutrophil infiltration in tumor lobules producing neutrophil microabscesses. When trichilemmomas were made in nude mice by subcutaneous injection of tissue-cultured cells, tumors were found to have neutrophil infiltration and abscesses, suggesting that K-TL-1 cells produced and released an NCF. Then cell-cultured medium (serum-free) was examined for NCF using the Boyden chamber method, the skin window technique and injection into nude mice. These studies confirmed the presence of NCF in K-TL-1 cell-cultured medium and the NCF were found to be heat-labile and nondialysable [1,2].

Another cell line, TK-TL-3, was established from a trichilemmoma which had dense eosinophil infiltration among tumor cells producing eosinophil microabscesses in tumor lobules. When tumors were made in nude mice by injecting tissue-cultured cells, they were revealed to contain eosinophils but no neutrophils, suggesting that TK-TL-3 cells released ECF. And the factor was confirmed by similar methods as mentioned previously. This factor was also heat-labile and nondialysable; it had no activity of NCF [3].

Nine human trichilemmomas were examined histo-pathologically and found to have neutrophil infiltrations in 5 cases, eosinophil in 2 cases and both in 1 case (8/9 cases showed neutrophil and/or eosinophil migrations among tumor cells) [4].

Discussion
These results clearly demonstrate that diseased hair follicular keratinocytes produce and release NCF or ECF, which are different from IL-1 or LTB-4, resulting in leukocytes infiltration among these keratinocytes. Although the biological significance of these phenomena is uncertain at the
present time, these factors appear to play a role in the leukocyte infiltration in the epidermis or epidermal appendages in various skin diseases.

References