Clinical Snippets
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GRANDMOTHER—WHAT LONG HAIR YOU HAVE

Hair length is related to the duration of the anagen phase of the hair cycle. Mice with the Angora mutation have long hair because of the absence of the Fibroblast Growth Factor-5 (FGF-5). FGF-5 has a role in the anagen to catagen transition. Foley and colleagues have determined that parathyroid hormone related protein (PTHrP) and its receptor have a role in regulating the transition of anagen to catagen independent of FGF-5 (p. 715). Overexpression of this protein caused hairs to be 30–40% shorter. Inhibitors of PTHrP will be available since this protein is associated with the humoral hypercalcemia of malignancy, and may make human hair grow longer.

MAST CELLS AND ITCHY SENIORS

Humans with bullous pemphigoid (BP) have eosinophils in the skin, while mice with induced BP have a neutrophilic infiltrate. Dimson and colleagues found increased total IgE levels in human BP and the IgE antibodies reacted with the pathogenic NC16A domain of BP-180 (collagen XVII). Mast cells had both IgE and surface bound BP-180 (p. 784). Having identified a role for mast cells (arrows), the urticarial lesions in BP may be more understandable and treatable.

PREVENTING PHOTOAGING

The complex process of sun-induced acute and chronic skin damage involves multiple molecular cascades. Kang and the Michigan team that has been studying the process for years found two small molecules that block the UVB-induced production of collagenase, which leads to the dermal damage characteristic of photoaging. Topical application of Genistein, a tyrosine kinase inhibitor, or N-acetylcysteine, a reducing agent, blocked collagenase induction but had no effect in UV-induced erythema. The agents were not sunscreens. This advances studies defining pathways and inhibitors of the various consequences of UV exposure in the skin (p. 835).

OIL AND WATER DO MIX

The devil is always in the details. Fluhr and colleagues report that Asebia, a natural mouse mutation from the days before custom-made knockout mice, has sebaceous gland hypoplasia and decreases in its attendant lipids, as well as normal barrier homeostasis, but markedly decreased stratum corneum hydration (page 728). The triglycerides of sebaceous glands are esters of glycerol and fatty acids. The glycerol content of the stratum corneum in asebic mice was markedly (85%) reduced. Topical glycerol normalized SC hydration. Therefore, glycerol’s utility (under its alias glycerin or glycerine—a mainstay of many skin moisturizers for centuries) may be based in its ability to retain water. Propylene glycol may also be effective through a similar mechanism. The experiment of nature in Asebia may be the long-awaited proof of many a magazine advertisement’s claims.

STUDYING PSORIASIS STUDIES

A group of European researchers analyzed 249 randomized clinical trials of psoriasis treatments published between 1977 and 2000. Their long-term aim is to determine a rational, academically-driven agenda for long-term comparative clinical trials in patients with psoriasis. Many trials had no satisfactory description of blinding criteria or entry criteria, and failed to consider dropouts in the analysis. Clinical trials that mimic the actual uses of drugs in clinical practice are thought to be necessary. Furthermore, Naldi and colleagues emphasize the importance of a variety of outcome measures and measures of the duration of remission (p. 738). The paper and its backup data, safe in cyberspace at http://www.blackwellpublishing.com/products/journals/support/ CID/jid_12145/jid_12145sm.htm, are worth perusal by those involved in clinical research or those who treat patients with psoriasis.