Galanin inhibits hair follicle growth

Holub et al. present the first evidence that human hair follicles (HFs) are both a source and a functionally relevant target of galanin. Galanin-like immunoreactivity was detected in the outer root sheath (ORS) and inner root sheath. Additionally, galanin mRNA was detected in ORS keratinocytes and all HF samples tested. Galanin receptor transcripts (GalR2, GalR3) were also detected in selected samples. Galanin reduced proliferation of hair matrix keratinocytes in situ compared with vehicle-treated controls, shortened the hair growth phase (anagen) in vitro and reduced hair shaft elongation. This was accompanied by the premature development of a catagen-like morphology of galanin-treated HFs. The current data suggest that galanin can serve as a new lead compound in the search for novel agents that reduce or prevent unwanted hair growth. Br J Dermatol 2012; 167: 10–16

Narrowband UVB, oral cholecalciferol and vitamin D balance

Ala-Houhala et al. compared the effects of a short course of narrowband ultraviolet B (NB-UVB) and oral vitamin D substitution in healthy subjects in winter. Healthy participants with 25(OH)D below 75 nmol L⁻¹ were randomly given either 12 NB-UVB exposures or 20 mg of oral cholecalciferol daily for 4 weeks. The baseline serum 25(OH)D concentrations were 52.9 ± 10.4 (mean ± SD) in the 33 NB-UVB-treated and 53.5 ± 12.7 nmol L⁻¹ in the 30 oral vitamin D-treated subjects. The mean increase in serum 25(OH)D was 41.0 nmol L⁻¹ in the NB-UVB group and 20.2 nmol L⁻¹ in the cholecalciferol group. The difference between the two treatments was significant at 2 weeks (P = 0.033) and at 4 weeks (P < 0.001). It is concluded that a short NB-UVB course is an effective way to improve vitamin D balance in winter and the response is still evident 2 months after the course. Br J Dermatol 2012; 167: 160–64

Efficacy and safety of certolizumab pegol in plaque psoriasis

Reich et al. report results of a randomized, placebo-controlled trial of 176 patients with moderate to severe psoriasis who received placebo or certolizumab pegol (CZP) 400 mg at week 0 followed by placebo or CZP (200 or 400 mg) every other week until week 10. A re-treatment extension study was conducted in 71 CZP PASI 75 responders who relapsed during a 12- to 24-week observation period without treatment. PASI 75 was achieved by 75%, 83% and 7% of patients in the CZP 200 mg, CZP 400 mg and placebo groups, respectively (P < 0.001 for both treatment arms vs. placebo). The efficacy observed during the re-treatment period was similar to that observed during the first treatment period. These phase II results support the continued clinical programme for development of CZP in psoriasis. Br J Dermatol 2012; 167: 180–90

Etanercept acutely suppresses the IL-20 cytokine subfamily

Wang et al. investigated early biochemical and cellular effects of etanercept on psoriasis lesions prior to substantial clinical improvement. By 1 week, etanercept suppressed gene expression of the interleukin (IL)-20 subfamily of cytokines (IL-19, IL-20, IL-24), and suppression of other keratinocyte-derived products. Suppression of epidermal regenerative hyperplasia occurred within 1–3 weeks. Th17 elements (IL-23p19, IL-12p40, IL-17A, IL-22) were suppressed by 3–4 weeks. These findings would support that epidermal activation is a very early target of etanercept. As many of these keratinocyte markers are stimulated by tumour necrosis factor (TNF)-α, their rapid down-regulation is likely to reflect etanercept’s antagonism of TNF-α. Additionally, decreased epidermal hyperplasia might result specifically from acute suppression of the IL-20 subfamily, which is also a likely consequence of etanercept’s antagonism of TNF-α. Thus, the IL-20 subfamily has potential importance in the pathogenesis of psoriasis and therapeutic response to etanercept. Br J Dermatol 2012; 167: 92–102