Analysis of Itch Relief in Psoriasis
Prior studies have demonstrated that pruritus occurs in 60-90% of psoriasis patients, negatively affecting their health-related quality of life. A systematic literature review and subsequent meta-analysis by Théréné and colleagues identified 35 studies that assessed resolution of pruritus following treatment of psoriasis with UVB phototherapy, calcineurin inhibitors, biological therapies, and small molecules. The evaluated treatments, including anti interleukin-17 antibodies, Janus kinase inhibitors, adalimumab, and apremilast, reduced pruritus in the meta-analysis. Interventional studies typically emphasize treatment effects on extent of disease. Since disease severity and pruritus do not necessarily correlate, these results suggest that itch improvement should be included as a separate endpoint in future trials of systemic psoriasis treatments. See page 38.

Blue light-induced pigmentation
Visible light, which comprises nearly half of the solar spectrum, induces a more potent and more long-lasting pigmentation than the oft-studied UVA and UVB radiation. Regazzetti and colleagues reported that shorter wavelengths of visible light (blue light, 415 nm) activate melanocytes and cause hyperpigmentation. Opsin 3, a light-sensitive G protein-coupled receptor found in eyes and skin, senses blue light in melanocytes and stimulates melanogenesis via increases in calcium flux and activation of downstream pathways. This results in sustained tyrosinase activation in dark skin type melanocytes and persistent hyperpigmentation. These findings highlight potential targets that might modulate melanogenesis in dark-skinned individuals in response to blue light under physiological and pathologic conditions. See page 171.

Evolution of Cancer Gene Mutations in PIPs
Epidermal p53-immunopositive patches (PIPs) that may represent cancer precursors frequently contain mutated TP53 genes. Whether PIPs harbor additional somatic mutations in cancer-driver genes has not been determined. Albibas and colleagues identified mutations in several cutaneous squamous cell carcinoma driver genes in PIPs that were not present in normal-appearing chronically-exposed skin from the same skin samples. While many of these genetic alterations, including those in TP53, were determined to be clonal, mutations within PIPs were also subclonal, indicating that subclones of cells containing multiple cancer gene mutations develop within PIPs as they evolve in sun-exposed skin. See page 189.

Mental Health Burden in Hidradenitis Suppurativa Patients
Hidradenitis suppurativa (HS) is a chronic inflammatory disease characterized by inflamed suppurative and scarring lesions in the axillary, inguinal, and anogenital regions that diminish patients’ quality of life. Several dermatological conditions, including psoriasis, are associated with a higher prevalence of psychiatric disorders. Recent studies have suggested that psychiatric comorbidities occur in HS patients, as well. Hulaja and colleagues conducted a retrospective study using Finnish medical registry data and compared the prevalence of psychiatric conditions in more than 4,000 HS patients with that in psoriasis patients. In this study, patients with HS exhibited a high psychiatric burden. Indeed, at least one psychiatric diagnosis was found in 24.1% of the patients with HS as compared to 19.1% of patients with psoriasis. Anxiety, depression, schizophrenia, disorders of adult personality and behavior, and bipolar disorder were all more prevalent in patients with HS than in patients with psoriasis. In an independent contemporaneous study, Thorlacius and colleagues reported a significantly increased risk of completed suicide in a cohort of more than 7500 patients with HS even after adjustment for confounding factors. Interestingly, previous studies failed to identify similar suicide trends in patients with psoriasis. Taken together, these results illustrate the profound impact of HS on patient quality of life and support consideration of the psychiatric needs of patients with HS undergoing clinical treatment. See pages 46 and 52.