The Editor's Choice

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Transcriptome profiling in psoriasis: NB-UVB treatment associated transcriptional changes and modulation of autoinflammation in perilesional skin in early-phase disease

Psoriasis is a chronic inflammatory skin condition. It is widely treated with phototherapy using narrowband ultraviolet B (NB-UVB). The therapeutic mechanisms of NB-UVB, however, remain unclear, particularly in the early phases of the disease. Vacharanukrauh P et al investigated the mechanisms underlying the effects of NB-UVB on psoriasis in a model of perilesional psoriasis. NB-UVB treatment may exert its effects by suppressing nuclear factor kappa B, which leads to upregulation of the sirtuin signaling pathway, as well as by decreasing the function of major upstream regulators associated with proinflammatory and inflammatory cytokines, which blocks the expression of downstream toll-like receptors. Psoriasis improvement after NB-UVB treatment was associated with decreased expression of NFKBIZ, SERPINB4, ATG13, and CTSS and increased expression of SKP1 gene. These results also highlighted the expression of proposed genes associated with the modulation of autoinflammation. providing new insights into the disease pathogenesis and novel genetic information for the development of new therapeutic modalities and potential treatment targets.

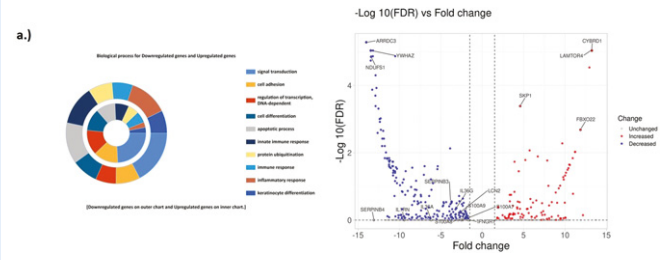

Fig. 1. Differentially expressed genes (DEGs) and functional annotation. (a.) Gene expression analysis. Number of DEGs before and after treatment is shown. Gene ontology analysis was used to compare the biologic processes of upregulated and downregulated genes. A volcano plot of DEGs after treatments with NB-UVB is shown.

Understanding the Systemic Burden of Disease in Hidradenitis Suppurativa from Plasma Lipidomic Analysis

Hidradenitis suppurativa (HS) is an inflammatory skin condition that is often considered a systemic disease due to its association with metabolic comorbidity. Choi E et al aimed to identify differences in plasma lipidomic profiles between HS patients and control subjects. 60 HS patients and 73 control subjects were recruited. Differential level($p < 0.05$) of 32 lipid species in HS patients compared to controls were observed, including a decrease in the long chain base d19:1 containing ceramides, and elevation of hydroxyeicosatetraenoic acid (HETE) and dihydroxyeicosatrienoic acid (DHET) oxylipins. These lipids along with several other molecules showed associations with Hurley, HS-PGA and disease activity scores. This study found mild changes in plasma lipidomic profiles, consistent with previous studies showing attenuated metabolomic changes in plasma as opposed to lesional skin. However, a number of lipid species were associated with increasing activity and severity of the disease. Further, the significant lipid species within the same class showed consistent trends of increase or decrease in HS as compared to controls.

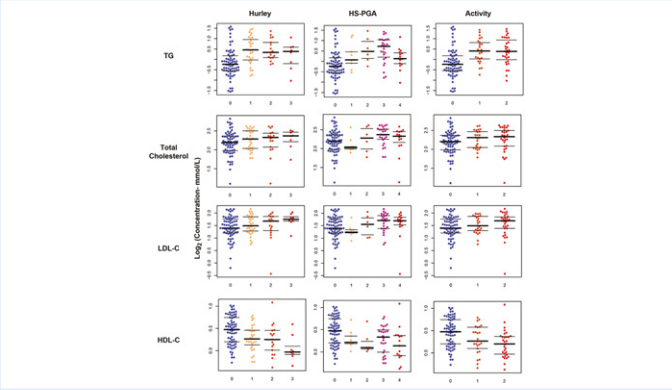

Fig. 4. Relationship of clinical lipid measures with Hurley, HS-PGA and activity. There was a statistically significant ($p < 0.05$) decrease in HDL-C and increase of TG with increasing Hurley, HS-PGA and disease activity scores. However after adjusting for confounders, only the association between HDL-C and Hurley stage remained significant.".

Herpes simplex virus-induced murine dry skin model through sweating disturbance

Given that ocular glands become infected secondarily to HSV-1 keratitis, resulting in the loss of tear production, sweat glands may also be susceptible to HSV-1 infection, resulting in sweating disturbance, which is observed frequently in atopic dermatitis. However, the role of sweating in the maintenance of skin hydration has not been elucidated. Asanuma Y et al determine the relationship between HSV-1 infection of sweat glands and sweating disturbance-induced dry skin. The sweating response and skin surface hydration were significantly decreased at 7–14 days post-infection. Sweating disturbance and dry skin was markedly enhanced when HSV-1 inoculation was followed by hyperthermic stress. HSV-1 DNA was detected in sweat glands and dorsal root ganglia. The sweating response remained decreased after subcutaneous injection with pilocarpine, correlating histologically with marked dilatation of sweat gland lumens. Sweating disturbance is unlikely to be the outcome of nerve damage by HSV-1 infection. Sweating disturbance could be due to HSV-induced dysfunction of sweat glands.

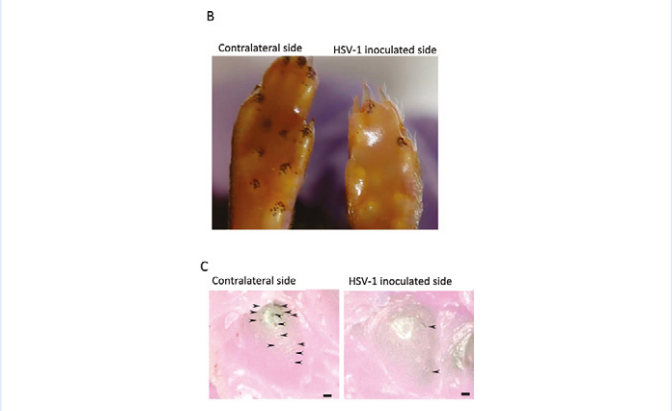

Fig. 1. HSV-1 induced zoster-like inflammation and decreased sweating responses in mouse footpad. B. Representative sweating responses on the plantar skin of the hind paw on the HSV-1-inoculated side and contralateral side determined using Minor's iodine-starch test. C. Representative sweating responses in the footpad on the HSV-1-inoculated side and contralateral side determined using the impression mold technique. Sweating droplets are indicated by arrow heads. Bar,100 μm. Differences between four groups were examined by the Tukey–Kramer test.