PIGMENTATION

MAPPING ARCHITECTURAL AND TRANSCRIPTIONAL ALTERATIONS IN NON-LESIONAL AND LESIONAL EPIDERMIS IN VITILIGO

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Background: In vitiligo, chronic loss of melanocytes and consequent absence of melanin from the epidermis presents a challenge for long-term tissue maintenance. The stable vitiligo patches are known to attain an irreversible depigmented state. However, the molecular and cellular processes resulting in this remodelled tissue homeostasis is unclear.

Objective: To investigate the complex interplay of inductive signals and cell intrinsic factors that support the new acquired state, we compared the matched lesional and non-lesional epidermis obtained from stable non-segmental vitiligo subjects.

Material and Methods: Skin punch biopsies (3–4 mm) from lesional and non-lesional (gluteal) sites from vitiligo patients were obtained after taking informed consent. Punch biopsies were immediately fixed in buffered formalin solution and four-micron thick cryo-sections were stained with hematoxylin and eosin. For detecting melanocytes, sections were stained with S100 antibody.
Total RNA was isolated using Trizol method from the whole epidermal cells from 15 pairs of non-lesional and lesional skin samples. Whole genome microarray was carried out using Illumina WG-6 array using manufacturer's guidelines.

Results: Hierarchical clustering of genome-wide expression of transcripts surprisingly segregated lesional and non-lesional samples in two distinct clades, despite the apparent heterogeneity in the lesions of different vitiligo subjects. Pathway enrichment showed the expected downregulation of melanogenic pathway and a significant downregulation of cornification and keratinocyte differentiation processes. These perturbations could indeed be recapitulated in the lesional epidermal tissue, including blunting of rete-ridges, thickening of stratum corneum and increase in the size of corneocytes. In addition, we identify marked increase in the putrescine levels due to the elevated expression of spermine/spermidine acetyl transferase.

Conclusion: Our study provides insights into the intrinsic self-renewing ability of damaged lesional tissue to restore epidermal functionality in vitiligo.