Mini Review
Volume 9:7, 2022

Journal of Dermatology and Dermatologic Diseases

ISSN: 2684-4281 Open Access

Immunology of Vitiligo

Khaled Ezzedine*

Department of Dermatology, EpiDermE - Epidemiology in Dermatology and Evaluation of Therapeutics, F-94010 Creteil, France

Abstract

White spots on the skin are the result of melanocytes being destroyed by CD8+ T cells in the autoimmune condition known as vitiligo. Numerous studies indicate that oxidative stress is a significant contributor to the onset of vitiligo from the very beginning. Overproduction of reactive oxygen species (ROS) and ROS accumulation in vulnerable melanocytes are jointly caused by multiple factors. ROS, on the other hand, play a role in the production of autoantigens and melanocyte damage at the level of molecules, organelles and cells through a variety of pathways connected to melanocyte dysregulation. Autoantigen presentation is mediated by innate immunity, which acts as a bridge between adaptive immunity and oxidative stress, according to recent research. The final destruction of epidermal melanocytes is guaranteed by the recruitment of CD8+ T cells induced by cytokines and chemokines. In addition, the reinstatement and relapse of vitiligo can be explained by emerging concerns regarding resident memory T cells and regulatory T cells. In this article, we attempt to uncover additional connections between autoimmunity and oxidative stress, as well as new perspectives on recent developments in our knowledge of the disease's pathogenesis.

Keywords: Autoimmunity • Oxidative stress • Vitiligo

Introduction

Depigmented white spots are the result of CD8+ T cells leading to the focal elimination of melanocytes in vitiligo, a skin autoimmune disease. It has a significant impact on the quality of life of patients and affects less than 1% of the global population. Although they are cumbersome, the current treatments for vitiligo are moderately effective at restoring pigment to the skin and more efficient targeted therapies are required. In spite of the skin's otherwise normal appearance, vitiligo manifests in a variety of patterns with scattered patches of activity. It is unknown how and why immune cells initiate autoimmune responses at one skin site while avoiding others. Previous research suggested that vitiligo patients may lack regulatory T cells (Tregs); But these studies don't agree on whether the defect is caused by fewer systemic Treg, less ability to find a home in the skin, or less function. As a result, it is unclear how Tregs contribute to the onset, development and distribution of vitiligo lesions [1].

Description

Several signaling pathways, such as IFN- and IL-15, have been identified as important drivers of inflammation and disease in recent mechanistic studies on mice. Blocking these interactions has been shown to be effective in vitiligo treatment in the initial drug repurposing studies. Using single cell RNA-sequencing (scRNA-seq), flow cytometry and ELISA, we sought to construct a comprehensive view of the signaling pathways among epidermis cells with the intention of expanding treatment options and posing fundamental questions about the onset and progression of vitiligo. Human vitiligo lesions, unaffected non-lesional skin and healthy control skin were sampled using suction blistering [2,3].

***Address for Correspondence:** Khaled Ezzedine, Department of Dermatology, EpiDermE - Epidemiology in Dermatology and Evaluation of Therapeutics, F-94010 Creteil, France; E-mail: kezzedine@aphp.fr*

Date of submission: 30 June, 2022, Manuscript No JPD-22-80931; **Editor Assigned:** 01 July, 2022, PreQC No P-80931; **Reviewed:** 15 July, 2022, QC No Q-80931; **Revised:** 20 July, 2022, Manuscript No R-80931; **Published:** 27 July, 2022, DOI: 10.37421/2684-4281.2022.9.358

More in-depth research studies were not conducted until the 1940s. In 1947, Haxthausen reported that transplanting nonlesional skin onto a lesional area caused the graft to depigment while grafting vitiligo lesional skin onto nonlesional sites resulted in varying degrees of repigmentation of the lesional skin. He hypothesized that the graft's pigmentation state was influenced by the local environment, which might be controlled by the nervous system. Pedicled and tubed flaps produced comparable outcomes, as Comel demonstrated in 1948. The nonlesional flaps that were attached to the lesional skin depigmented while the lesional flaps repigmented. In contrast, Spencer and Tolmach reported in 1951 that graft transplantation between lesional and nonlesional skin did not result in repigmentation or depigmentation of lesional grafts; rather, they reported an extension of depigmentation around the lesional skin graft. The various approaches that were taken with the skin grafts may have contributed to the divergent outcomes. Spencer and Tolmach utilized full-thickness skin grafts, whereas Haxthausen utilized thin split thickness grafts, also known as a Thiersch graft. Despite the fact that these were brief case studies with varying outcomes, they suggested that the setting in which the graft is transplanted is a significant factor. Behl applied skin grafts as a large-scale treatment option for vitiligo not until 1964. He used the Thiersch grafting technique, which was similar to the one used in the Haxthausen study. He kept the variability in repigmentation and relapse to a minimum by only performing the procedure on patients who had stable disease [4,5].

Changes in metabolism and immune function, as well as environmental triggers and genetic predispositions, all contribute to the loss of melanocytes. Epigenetic changes may likewise be engaged with vitiligo pathogenesis, as recommended in the Pu et al. study in vitiligo melanocyte cell lines that identifies altered methylation levels of key genes involved in oxidation-reduction, inflammatory, or pigmentation processes. The immune response's role in non-segmental vitiligo (or vitiligo), which accounts for approximately 90% of clinical forms, was the focus of the majority of published studies. segmental vitiligo has received less research. in their analysis. emphasize the role that autoimmunity plays in segmental vitiligo, probably through a specialized immune response against melanocytes with a somatic mutation. Demonstrate, Dysregulated immune pathways were found to be similar in both segmental and non-segmental vitiligo lesions of the skin.

We focused on differentially expressed genes within CD8+ T cells because of their significance in vitiligo and autoimmunity. A series of transcriptional transitions between healthy, non-lesional and lesional skin were observed when the aggregated gene expression profile was clustered within CD8+ T cells. Gene Ontology (GO) was used to determine whether each bin of the final heatmap contained significantly enriched gene sets. There is a significant response to IFN (FDR = 1. 9E3) and T cell activation (FDR = 3. 3E6) within

CD8+ T cells in non-lesional skin of vitiligo patients, including transcripts for costimulatory molecules CD28 and ICOS as well as chemokines CCL3/4/5, despite normal melanocyte numbers. This contrasts with the expression of transcripts for cytotoxic molecules like GZMB, GNLY and PRF1, which do not get any higher until the skin turns from non-lesional to lesional at the end. The presence of transcripts for GZMB, GNLY, PRF1 and other cytotoxic molecules in NK cells of lesional skin raises the possibility that these cells are involved in melanocyte clearance.

Conclusion

Pfizer, Genzyme/Sanofi, Aclaris Therapeutics Inc. , Incyte, Rheos Medicines, Sun Pharmaceuticals, LEO Pharma, Villaris Therapeutics Inc. , Dermavant, Temprian, AbbVie Inc. , Janssen, TeVido BioDevices, EMD Serono, Almirall, Boston Pharma, Sonoma Biotherapeutics Inc. , Methuselah Health, Twi Biotech, PPfizer, Genzyme/Sanofi, Incyte, Rheos Medicines, Sun Pharmaceuticals, LEO Pharma, Villaris Therapeutics Inc. , Dermavant, AbbVie, TeVido BioDevices, EMD Serono and Pandion employ JEH as an investigator. Villaris Therapeutics, Inc., which is developing therapeutic treatments for vitiligo and NIRA Biosciences are scientifically founded by JEH. JEH is an inventor on patents #62489191 (Diagnosis and Treatment of Vitiligo), #067988 (Anti-Human CXCR3 Antibodies for Treatment of Vitiligo) and #029531 (Compositions and Methods for Treating Vitiligo), all of which include methods for treating vitiligo by targeting IL-15 and Trm.

Acknowledgement

None.

Conflict of Interest

There are no conflicts of interest by author.

References

1. Bergsagel, P. Leif, María-Victoria Mateos, Norma C. Gutierrez and S. Vincent Rajkumar, et al. "Improving overall survival and overcoming adverse prognosis in the treatment of cytogenetically high-risk multiple myeloma." *Am J Hematol* 121 (2013): 884-892.
2. Frosch, Michael, Thomas Vogl, Rüdiger Waldherr and Clemens Sorg, et al. "Expression of MRP8 and MRP14 by macrophages is a marker for severe forms of glomerulonephritis." *J Leuko Biol* 75 (2004): 198-206.
3. Stockfleth, Eggert, Ralph von Kiedrowski, Rolf Dominicus and John Ryan, et al. "Efficacy and safety of 5-fluorouracil 0. 5%/salicylic acid 10% in the field-directed treatment of actinic keratosis." *Dermatol Ther* 7 (2017): 81-96.
4. Fargnoli, M.C., A. Piccioni, L. Neri and S. Tambone, et al. "Conventional vs. daylight methyl aminolevulinate photodynamic therapy for actinic keratosis of the face and scalp." *J Eur Acad Dermatol Venereol* 29 (2015): 1926-1932.
5. Rosmarin, David, Amit G. Pandya, Mark Lebwohl and Pearl Grimes, et al. "Ruxolitinib cream for treatment of vitiligo: A randomised, controlled, phase 2 trial. " *Lancet* 396 (2020): 110-120.

How to cite this article: Ezzedine, Khaled. “Immunology of Vitiligo." J Dermatol Dis 9 (2022): 358.