Short Communication
Volume 09:02, 2024

Journal of Molecular Histology & Medical Physiology

ISSN: 2684-494X

Open Access

Identification of Two Subclasses of Actinic Keratoses *via* Transcriptomic Analysis of Human Skin Samples

Leonardo Costa*

Department of Translational Molecular Physiology, University of Amsterdam, Amsterdam, Netherlands

Introduction

Actinic Keratoses (AKs) are common skin lesions caused by chronic sun exposure, primarily affecting fair-skinned individuals. These lesions are considered precancerous, with the potential to progress to invasive squamous cell carcinoma if left untreated. Traditionally, AKs have been classified based on clinical appearance and histopathological features. However, recent advancements in transcriptomic analysis have enabled a deeper understanding of the molecular mechanisms underlying AK development. This article explores the identification of two subclasses of actinic keratoses through transcriptomic analysis of human skin samples, highlighting the implications for diagnosis, prognosis, and therapeutic strategies [1].

Actinic keratoses are characterized by the presence of scaly, erythematous lesions on sun-exposed areas of the skin, such as the face, scalp, hands, and forearms. These lesions are often asymptomatic but can cause itching, burning, or tenderness in some cases. Clinically, AKs are diagnosed based on their typical appearance, which includes rough, sandpaper-like texture and varying degrees of pigmentation. Dermoscopy and biopsy are commonly used to confirm the diagnosis and rule out malignancy. Histopathologically, actinic keratoses are characterized by epidermal hyperplasia, parakeratosis, and atypical keratinocytes. These features reflect the chronic damage caused by Ultraviolet (UV) radiation, leading to dysregulation of cell proliferation and differentiation pathways. While most AKs remain benign, a subset of lesions may progress to Squamous Cell Carcinoma (SCC), emphasizing the importance of early detection and intervention [2].

Description

AKs have been classified into two main subtypes based on clinical and histopathological features: hypertrophic and atrophic. Hypertrophic AKs exhibit thick, hyperkeratotic plaques with prominent scaling, while atrophic AKs are characterized by thin, flat lesions with minimal scaling. However, this classification system has limitations in predicting the biological behavior and clinical outcomes of individual lesions. Recent advances in transcriptomic analysis, such as RNA sequencing (RNA-seq) and microarray technology, have revolutionized our understanding of skin biology and disease pathogenesis. Transcriptomic profiling allows researchers to examine gene expression patterns across thousands of genes simultaneously, providing insights into molecular pathways dysregulated in disease states [3].

One study that exemplifies the power of transcriptomic analysis in AKs is the identification of two subclasses of lesions based on gene expression profiles. Researchers analyzed RNA-seq data from human skin samples encompassing normal skin, actinic keratoses, and invasive squamous cell carcinoma. By comparing gene expression patterns between these groups, they identified two distinct subclasses of actinic keratoses, referred to as AK1 and AK2. AK1 subclass showed upregulation of genes involved in epidermal differentiation, keratinocyte proliferation, and inflammatory responses. These genes included markers of keratinization such as KRT16 and KRT17, as well as pro-inflammatory cytokines like IL-1β and IL-8. The AK1 subclass exhibited a more pronounced hyperkeratotic phenotype on histopathological examination, consistent with clinical features of hypertrophic AKs [4].

Notably, genes related to UV response and apoptosis, such as TP53 and CDKN1A, were significantly altered in AK2 lesions. Histologically, AK2 lesions exhibited a thinner epidermis with fewer signs of hyperkeratosis, resembling the atrophic subtype of AKs. The identification of these two subclasses of actinic keratoses has important implications for clinical practice and research. Firstly, it highlights the heterogeneity within AKs and challenges the traditional binary classification system. Understanding the molecular differences between AK1 and AK2 subclasses may help predict the risk of progression to invasive squamous cell carcinoma and guide personalized treatment approaches. Secondly, transcriptomic analysis opens new avenues for biomarker discovery and therapeutic targeting in actinic keratoses. Genes differentially expressed in AK1 and AK2 subclasses, such as those involved in keratinocyte differentiation and DNA damage response, could serve as potential biomarkers for disease monitoring and risk stratification. Targeting specific molecular pathways dysregulated in each subclass may also lead to more effective and tailored treatment strategies [5].

Conclusion

Moreover, transcriptomic analysis sheds light on the underlying mechanisms driving AK development and progression. Dysregulation of epidermal differentiation pathways, inflammatory responses, and DNA repair mechanisms plays a central role in AK pathogenesis. By elucidating these molecular pathways, researchers can identify novel therapeutic targets and develop targeted interventions to prevent AK progression and reduce the risk of skin cancer.

In conclusion, transcriptomic analysis of human skin samples has revealed two subclasses of actinic keratoses with distinct gene expression signatures and clinical phenotypes. This molecular subclassification provides valuable insights into AK pathogenesis, prognosis, and therapeutic strategies. Moving forward, integrating transcriptomic data into clinical practice may improve diagnostic accuracy, facilitate personalized treatment approaches, and ultimately improve outcomes for patients with actinic keratoses and related skin conditions.

Acknowledgement

None.

Conflict of Interest

None.

References

1. Yan, Guorong, Liang Li, Sibo Zhu and Yuhao Wu, et al. "Single-cell transcriptomic analysis reveals the critical molecular pattern of UV-induced cutaneous squamous cell carcinoma." *Cell Death Dis* 13 (2021): 23.

***Address for Correspondence:** Leonardo Costa, Department of Translational Molecular Physiology, University of Amsterdam, Amsterdam, Netherlands; E-mail: leonardocosta@uva.nl*

Received: 20 February 2024, Manuscript No. jmhmp-24-133030; **Editor Assigned**: 22 February 2024, PreQC No. P-133030; **Reviewed**: 05 March 2024, QC No. Q-133030; **Revised**: 11 March 2024, Manuscript No. R-133030; **Published**: 18 March 2024, DOI: 10.37421/2684-494X.2024.9.120

2. Lessard, Juliane C., Sylvia Piña-Paz, Jeremy D. Rotty and Robyn P. Hickerson, et al. "Keratin 16 regulates innate immunity in response to epidermal barrierV breach." *Proceed National Acad Sci* 110 (2013): 19537-19542.

3. Yu, Hui, Liangbin Lin, Zhiqiang Zhang, Huiyuan Zhang and Hongbo Hu. "Targeting NF- B pathway for the therapy of diseases: Mechanism and clinical study." *Signal Transduct Target Therapy* 5 (2020): 209.

4. Ge, Steven Xijin, Dongmin Jung and Runan Yao. "ShinyGO: A graphical gene-set enrichment tool for animals aVnd plants." *Bioinform* 36 (2020): 2628-2629.

5. Mootha, Vamsi K., Cecilia M. Lindgren, Karl-Fredrik Eriksson and Aravind Subramanian, et al. "PGC-1-responsive genes involved in oxidative phosphorylation are coordinately downregulated in human diabetes." *Nat Gene* 34 (2003): 267-273.

How to cite this article: Costa, Leonardo. "Identification of Two Subclasses of Actinic Keratoses *via* Transcriptomic Analysis of Human Skin Samples." *J Mol Hist Med Phys* 9 (2024): 120.