Genomic Profiling of Inflammatory Biomarkers in Human Skin Explores Transcriptomic Indicators of Acute Photodamage Following UVB Exposure

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BACKGROUND

UVB exposure is known to induce inflammatory responses and alter biomarkers in the skin, however specificity of genes affected, and their expression trends are not fully characterized. The development of skin cancer driven by ultraviolet radiation involves intricate cellular pathways such as programmed cell death, cell proliferation, cellular self-recycling, DNA repair, checkpoint signaling, metabolic regulation, and inflammatory responses.[1] Additional studies provided insights into the UV response of the skin, emphasizing the involvement of the MAPK, NFκB, and TNFα signaling pathways in the skin's reaction to UV radiation.[2] These studies provide a foundation for the current research, which aims to further elucidate the genomic responses to UVB exposure by examining the expression of specific inflammatory genes in human skin.

OBJECTIVE

The aim of this pre-post interventional study is to quantify expression changes in a panel of six inflammatory genes in response to UVB exposure, to elucidate genomic indicators of cutaneous photodamage.

METHODS

Stratum-corneum cells were collected with adhesive patches from non-sun exposed skin of 10 caucasian adults (age 34-69) before and 48 hours after receiving one15 minute treatment Xtrac Velocity 400 excimer laser to induce UVB radiation. Expression of inflammatory genes S100A7, TXNDC5, Vimentin, Filaggrin, Kallikrein-6, and Heme-oxygenase-1 was measured using RT-qPCR.

REFERENCES

1.Kim Y, He YY. Ultraviolet radiation-induced non-melanoma skin cancer: Regulation of DNA damage repair and inflammation. Genes Dis. 2014;1(2):188-198.
2.Muthusamy V, Piva TJ. The UV response of the skin: a review of the MAPK, NFkappaB and TNFalpha signal transduction pathways. Arch Dermatol Res. 2010;302(1):5-17.

RESULTS

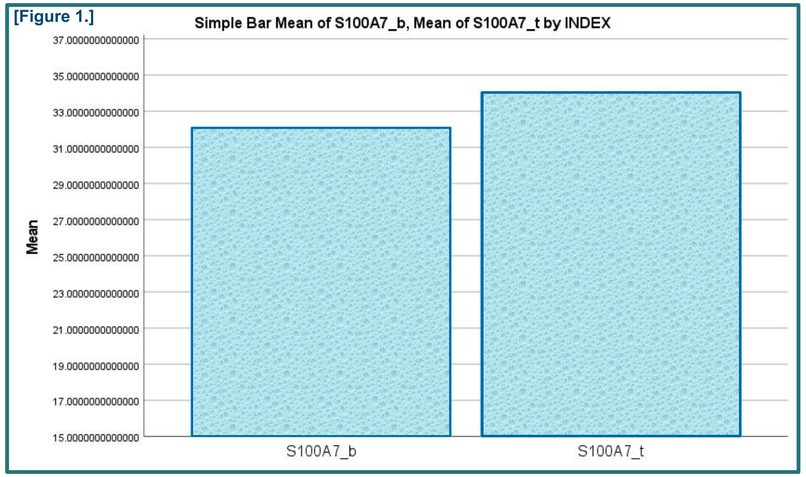
Paired-sample T-tests comparing means and variances showed no significant differential expression of the 6 genes from baseline to treatment. However, S100A7 exhibited the strongest positive correlation between baseline and treatment with a mean increase of 1.94 (correlation = 0.54, p = 0.108). Additionally, a significant positive correlation was found between age and TXNDC5 expression (r = 0.652, p = 0.041).

[Table 1.]

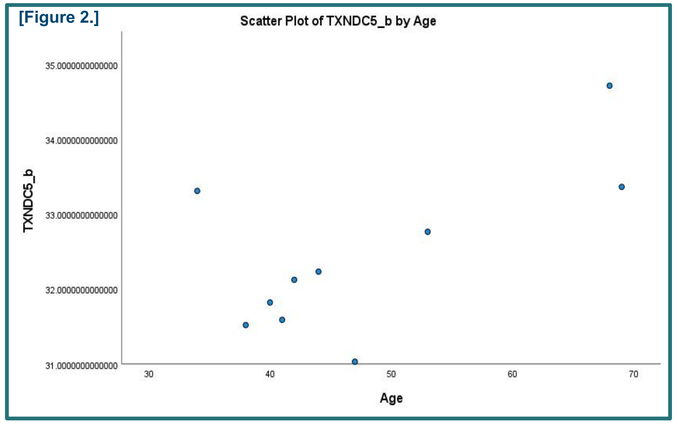
Paired Samples Correlations

		N	Correlation	Significance One-Sided p	Two-Sided p
Pair 1	FLG_b & FLG_t	10	.395	.129	.259
Pair 2	KLK6_b & KLK6_t	5	-.134	.415	.830
Pair 3	HMOX_b & HMOX_t	9	.428	.125	.251
Pair 4	S100A7_b & S100A7_t	9	.540	.067	.133
Pair 5	VIM_b & VIM_t	10	.197	.293	.586
Pair 6	TXNDC5_b & TXNDC5_t	9	.048	.451	.902

[Figure 1.]



[Figure 2.]



[Table 2.]

Correlations

		Age
FLG_b	Pearson Correlation	-.114
	Sig. (2-tailed)	.754
	N	10
FLG_t	Pearson Correlation	-.116
	Sig. (2-tailed)	.749
	N	10
KLK6_b	Pearson Correlation	.119
	Sig. (2-tailed)	.744
	N	10
KLK6_t	Pearson Correlation	.413
	Sig. (2-tailed)	.490
	N	5
HMOX_b	Pearson Correlation	-.321
	Sig. (2-tailed)	.365
	N	10
HMOX_t	Pearson Correlation	-.367
	Sig. (2-tailed)	.332
	N	9
S100A7_b	Pearson Correlation	.395
	Sig. (2-tailed)	.293
	N	9
S100A7_t	Pearson Correlation	.385
	Sig. (2-tailed)	.272
	N	10
VIM_b	Pearson Correlation	-.487
	Sig. (2-tailed)	.154
	N	10
VIM_t	Pearson Correlation	-.456
	Sig. (2-tailed)	.185
	N	10
TXNDC5_b	Pearson Correlation	.652*
	Sig. (2-tailed)	.041
	N	10
TXNDC5_t	Pearson Correlation	.142
	Sig. (2-tailed)	.716
	N	9

Table 1. Details paired sample T-Tests showing correlation values of baseline gene expression levels to post-treatment levels for all 6 genes.

Figure 1. Highlights correlation of S100A7 at baseline to post-treatment, with a mean increase after UVB exposure.

Table 2. Details correlation of gene expression of all 6 genes at baseline and post-treatment to age.

Figure 2. Graphs the significant correlation (r= 0.652, p= 0.041) between baseline expression of TXNDC5 with age.

Authors have no financial interests to disclose.

CONCLUSION

Early evidence suggests S100A7 and TXNDC5 may indicate a unique inflammatory response; S100A7, a chemotactic agent for CD4+ T-cells identified as a potential therapeutic target for acute inflammation, while significant correlation with age proposes TXNDC5, an apoptosis regulator as a novel indicator of skin-aging. Larger studies are needed to validate these findings and fully characterize expression patterns of inflammatory genes following UVB damage. These results, demonstrating an inflammatory genomic profile response to UVB could predict response to immunotherapy.