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Introducing Aurora Kinase A as a Gene Target Against Photoaging: A Network Analysis

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Abstract

Introduction: The solar radiation spectrum, which reaches the Earth's surface, spans from infrared to ultraviolet. In the present study, human skin response to solar-stimulation radiation was assessed via directed protein-protein interaction (PPI) network analysis.

Methods: Data were extracted from the Gene Expression Omnibus (GEO) database. The gene expression profiles of human skin after exposure to 100J/M^2 versus the control were selected from GSE22083. The data were evaluated via box plot analysis and Uniform Manifold Approximation and Projection (UMAP) plot assessment. The differentially expressed genes (DEGs) were visualized via a Venn diagram. After cleaning the data, the queried DEGs were assessed via the directed PPI network by the application of the CluePedia plugin of Cytoscape software. The critical DEGs were pointed out based on out-degree value. Key actor genes were searched in GeneCards to find a suitable description of them.

Results: Among 1482 significant DEGs, AURKA, CENPA, PP2R1B, UBE2O, RPA3, YKT6, SMARCA5, and SMAD3 were identified and highlighted as the critical actor genes in response to solar-stimulated radiation in human skin.

Conclusion: In conclusion, AURKA appeared as the top-ranked DEGs in response to solar radiation in the human skin. Based on the findings, AURKA was pointed out as a suitable target against photoaging. The relationship between solar-stimulated radiation and photoaging, cancer promotion, innate immune system, DNA replication, repair of UV damage-induced, vesicular trafficking between the Golgi apparatus and the endoplasmic reticulum, exocytosis of neurotransmitter, exosome production, autophagy, maintenance of nucleosome spacing, and process of progressive fibrosis were established.

Keywords: Solar radiation; Human; Skin; Gene; Network analysis.

Introduction

The solar radiation spectrum, which reaches the earth's surface, spans from infrared to ultraviolet.[1] Discussion about the effects of excessive sun exposure on human health is accompanied by the negative effects of solar UV radiation on human health. The prominent role of UV and visible light in vitamin D synthesis and the decrease of blood pressure, together with other valuable effects of sunlight, implies complementary investigations into its harmful and beneficial effects on human health. Light

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photons interact with the skin physically, chemically and biologically. These manors describe how light photons penetrate, absorb, and evoke biological responses. Explorations have shown that the electromagnetic solar spectrum includes 2% UV radiation, 47% visible radiation, and 51% infrared radiation.[2,3] Investigations have also demonstrated that opsins play a role as a skin light-detecting system. This process is comparable to the eye photosensitive system.[4] It has been reported that melanopsin has a protective role against UVA-induced damages in the irradiated skin.[5] The complete set of opsins in skin cells and their relative phototransduction cascades are involved in the entire solar spectrum.[6]

The molecular mechanism of many diseases and disorders are studied via gene expression change assessment. It has been confirmed that each physiological or pathological conditions are associated with the expression of a certain gene function.[7,8] Due to the complex nature of genomics, integrated genomics-bioinformatics has been established to analyze genomics findings.[9] Protein-protein interaction (PPI) network analysis as a bioinformatics approach has commonly been used to evaluate the large number of differentially expressed genes (DEGs). In such investigations, the queried DEGs are connected together to form an interactome. Each node of the interactome plays a role in the constructed network. The number of connections between a certain node and its first neighbors is known as degree. In a directed network, the connection between a certain node and its first neighbors is called out-degree parameter. The nodes with a higher value of out-degree are branded as actor nodes. The actor nodes are the critical elements of a directed PPI network.[10-12] In the present study, the gene expression profiles of the human skin exposed to 100 J/m^2 of solar-simulated radiation versus the control were extracted from the Gene Expression Omnibus (GEO) database and analyzed via the directed PPI network to explore the critical targeted genes by light radiation. Function, associated diseases, pathways, and related gene ontology annotations of the critical DEGs were investigated and discussed. Findings have opened a new window about the molecular event in the skin after exposure to solar-simulated radiation.

Methods

Data Collection

To find the effect of sunlight on the human skin, we explored the skin gene expression profiles of 8 healthy volunteers with Fitzpatrick type II skin from the GEO database (https://www.ncbi.nlm.nih.gov/geo/geo2r/?acc=GSE22083). GSE22083 is expression profiling by array. Data are linked to a published document by Tock et al.[13] As described in this document, the skin gene expression profiles of the samples after exposure to a fixed dose of 100 J/m^2 solar-simulated radiation (ssR) were compared with the controls. Light was produced by an Oriel solar simulator (300-Watt full spectrum model #81160, Oriel, Stratford, CT). The produced ssR included UVB, UVA, visible light, and infrared light. A 2x2 cm area of sun-protected buttock skin was radiated by the applied ssR. The extracted total RNA of the radiated skins was compared with that of the non-irradiated samples.

Pre-evaluation Analysis

To investigate the gene expression profiles of the samples, after using the force normalization option, we compared GSM48984-91 as the radiated samples with GSM49016-23 (as controls) via the GEO2R program. Box plot visualization, Uniform Manifold Approximation and Projection (UMAP) plot, and Venn diagram were provided to explore differences between radiated and control samples. Data were cleaned considering "fold change" ≥ 1.5, deletion of the repeated individuals and the uncharacterized genes. The cleaned data were prepared to be evaluated via PPI network analysis.

PPI Network Analysis

A directed PPI network was selected as a tool to screed the queried genes. The cleaned data were included in CluePedia application[14] of Cytoscape software[15] to form a directed PPI network. Nodes were interacted via expression, activation, inhibition, binding, catalysis, post-translation modification, and reaction actions. To find the central nodes, the PPI network was analyzed using the "Network Analyzer" application of Cytoscape software via directed mode. The out-degree centrality parameter[16] was considered to explore the actor genes in the PPI network. The nodes of the main connected component of the analyzed PPI network were laid out based on out-degree value.

Results

Comparability of the samples is shown in Figure 1. As demonstrated in the box plot, the distribution of dysregulated genes in the samples is median centric. UMAP analysis showed that the dysregulated genes separate the radiated samples from the individual controls (see Figure 2). A total of 1482 DEGs were identified as the significant dysregulated genes (see Figure 3). Assessment showed that 20,801 genes were dysregulated with "adjusted *P* value" ≥ 0.05. After cleaning the data, 223 significant DEGs were identified for PPI network analysis. The main component of the analyzed PPI network is presented in Figure 4. The central nodes, including AURKA, CENPA, PP2R1B, UBE2O, RPA3, YKT6, SMARCA5, and SMAD3, were identified and highlighted based on out-degree value. As depicted in Figure 4, AURKA with an out-degree value of 25 appeared as the potent actor gene. On the other hand, SMAD3 was the weakest central gene with out-degree value of 9. The summarized information about the central nodes, including associated diseases,

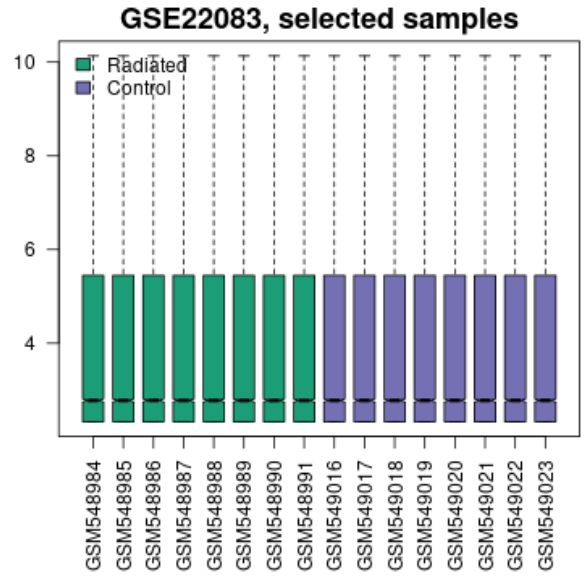

Figure 1. Box Plot of the Gene Expression Profiles of the Radiated and Control Samples

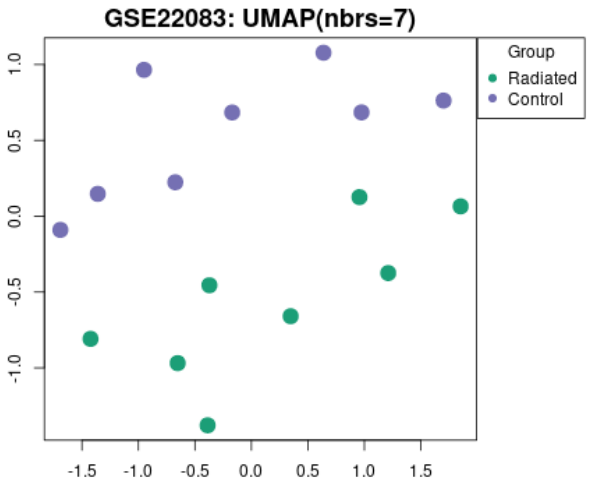

Figure 2. UMAP Plot of the Radiated and Control Samples. Each gene expression profile is compared with 7 neighbors (nbrc = 7). The dimensionless values of the axes show the position of samples relative to their neighbors

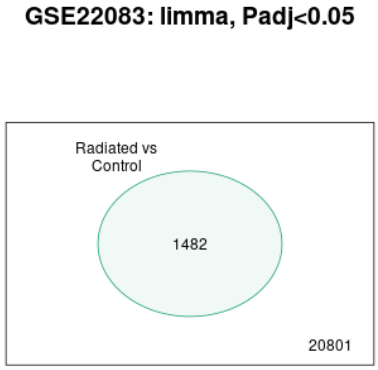

Figure 3. Venn Diagram of the Radiated-Control Gene Expression Analysis. A total of 1,482 significant DEGs are determined

related pathways, and related gene ontology annotations, was extracted from GeneCards, a human gene database

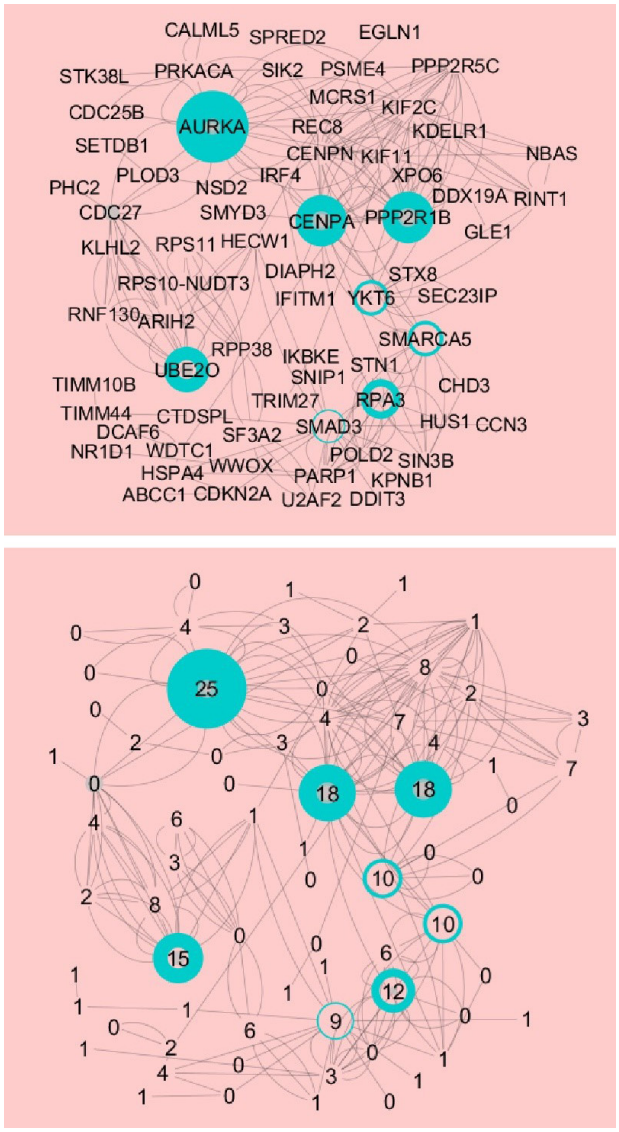

Figure 4. The Analyzed Main Connected Component of the Directed PPI Network of Radiated-Control Gene Expression Analysis. Nodes are connected via expression, activation, inhibition, binding, catalysis, post-translation modification, and reaction actions. Nodes are laid out based on out-degree value

(https://www.genecards.org). The organized data are presented in Table 1. Information about PP2R1B was not found in the GeneCards database.

Discussion

Investigations into the biological effects of various parts of solar radiation are frequently available in the literature. Svobodová A published a review about induced skin damages by solar radiation. UVC (200-280nm), UVB (280-320nm), UVA2 (320-340), UVA1 (340-400nm), Visible light (400-700nm), and Infrared light (700nm-1mm) are introduced as various parts of the solar radiation spectrum. As definite, atmosphere blocks 200-295 nm, 295-320 cause sunburn and DNA damage, 320-340nm is related with immune suppression, 400-1000 nm is involved in oxidative damage DNA (and other

Table 1. The summarized information about the central nodes (except PP2R1B, which was not found in the GeneCards database), including associated diseases, related pathways, and related gene ontology annotations from GeneCards, a human gene database (https://www.genecards.org)

Gene	Associated Diseases	Related Pathways	Related Gene Ontology Annotations
Aurora kinase A (AURKA)	Colorectal cancer and thrombocythemia 1	Loss of proteins required for interphase microtubule organization from the centrosome and CDK-mediated phosphorylation and removal of Cdc6.	Transferase activity, transferring phosphorus-containing groups and protein tyrosine kinase activity.
Centromere protein A (CENPA)	Crest syndrome and luminal breast carcinoma A	EML4 and NUDC in mitotic spindle formation and separation of sister chromatids.	Protein heterodimerization activity and chromatin binding.
Ubiquitin conjugating enzyme E2 O (UBE2O)	Pontocerebellar hypoplasia, type 2A and pontocerebellar hypoplasia, type 16	Class I MHC mediated antigen processing and presentation and innate immune system.	RNA binding and ubiquitin-protein transferase activity.
Replication protein A3 (RPA3)	Rheumatoid arthritis interstitial lung disease and bloom syndrome	Homologous DNA pairing and strand exchange and HDR through homologous recombination (HRR) or single strand annealing (SSA).	Single-stranded DNA binding and damaged DNA binding.
YKT6 V-SNARE homolog (YKT6)	Foodborne botulism and infant botulism	Transport to the Golgi and subsequent modification and signaling by Rho GTPases.	SNARE binding and protein-cysteine S-palmitoyltransferase activity.
SNF2 related chromatin remodeling ATPase 5 (SMARCA5)	Ewing sarcoma and microcephaly	RNA polymerase I promoter opening and HDR through HRR or SSA.	Nucleic acid binding and hydrolase activity.
SMAD family member 3 (SMAD3)	Loeys-Dietz syndrome 3 and aortic aneurysm	Autodegradation of the E3 ubiquitin ligase COP1 and TGF-beta receptor signaling activates SMADs.	DNA-binding transcription factor activity and sequence-specific DNA binding.

biomolecules), 295-1000 nm region is complicated in ROS (reactive oxygen species)/RNS (reactive nitrogen species) generation. It is emphasized that 320 nm-1000 nm is associated with photoaging.[17] H_2O_2, as a major ROS member, is involved in the regulation of AURKA. Evidence indicates that additional H_2O_2 causes mitotic delay and an abnormal mitotic spindle. On the other hand, AURKA plays a prominent role in spindle formation.[18] As depicted in Table 1, "loss of proteins required for interphase microtubule organization from the centrosome" is associated with AURKA activity. AURKA is identified as the top actor gene in our analysis. AURKA is upregulated in response to solar-simulated radiation in the skin of radiated people. Investigations indicate that the treated mammalian cells with H_2O_2 have administrated mitotic delay and abnormal spindle formation via the elevated phosphorylation level of AURKA.[19] This finding is consistent with the upregulation of AURKA in response to solar-stimulated radiation in the human skin. Based on the literature, high-intensity focused ultrasound (HIFU) application downregulates AURKA and NF-κB and upregulates HSP70 in the aged face. This process leads to increased adipogenesis in subcutaneous fat via modifying cilia on adipose-derived stem cells[20]. It can be concluded that AURKA is a suitable target to treat photoaging.

CENPA is the second gene actor which is upregulated in response to solar-stimulated radiation in the human skin. As depicted in Table 1, EML4 and NUDC in mitotic spindle formation and separation of sister chromatids are highlighted as the related pathways of CENPA. Investigations demonstrated that the expression of centromeric histone H3 variant CENPA is suppressed in aged cells[21]. This finding corresponds with the role of CENPA in photoaging. It seems that CENPA is the second appropriate drug target to treat photoaging in the human skin. PP2R1B is the third upregulated actor gene in the treated human skin. There was not any description about this gene in the GeneCards database. Type 2A protein phosphatase (PP2A) includes a varied family of phosphothreonine- and phosphoserine-specific enzymes. The involvement of the PP2A family member in several human cancers has been reported by researchers.[22,23] Ubiquitin Conjugating Enzyme E2 O (UBE2O) is another actor gene which is associated with the Innate Immune System pathway (see Table 1). As mentioned above, the 320-340nm part of the solar radiation spectrum is related to immune suppression.[17]

As demonstrated in Table 1, single-stranded DNA binding and damaged DNA binding are Replication Protein A3 (RPA3) related gene ontology annotations. It is verified that RPA plays a significant role in DNA replication. The role of RPA3 in the repair of UV-induced damage is confirmed. A low dose of UVB radiation upregulates the RPA gene.[24] This gene is upregulated after solar-stimulated radiation in the original data. YKT6 V-SNARE Homolog (YKT6) is another upregulated actor gene. The prominent role of YKT6 in vesicular trafficking between the Golgi apparatus and the endoplasmic reticulum, exocytosis of neurotransmitters, exosome production, and autophagy are attributed to YKT6 function.[25] Bomber ML et al. introduced human SNF2 Related Chromatin Remodeling ATPase 5 (SMARCA5) as a player in maintaining nucleosome spacing.[26] Gauldie et al highlighted the involvement of SMAD Family Member 3 (SMAD3), the last actor gene, in the process of progressive fibrosis.[27] As discussed above, the crucial targets in the radiated skin opened a new perspective on the molecular events. However, the prominent role of the AURKA gene in photoaging was pointed out as a key mechanism of skin damage.

Conclusion

In conclusion, AURKA which is involved in mitotic delay and abnormal spindle formation, appeared as the top dysregulated gene in response to solar radiation in the human skin. Findings demonstrate that AURKA is a suitable target to treat photoaging. The second-ranked gene was CENPA, the gene that was associated with photoaging in the human skin. The effect on cancer promotion, innate immune system, DNA replication, repair of UV damage-induced, vesicular trafficking between the Golgi apparatus and the endoplasmic reticulum, exocytosis of neurotransmitter, exosome production, autophagy, maintenance of nucleosome spacing, and process of progressive fibrosis were highlighted as the consequences of solar-stimulated radiation in human skin.

Authors' Contribution

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Competing Interests

The authors declare that there is no conflict of interest regarding the publication of this paper.

Ethical Approval

This project is approved by Shahid Beheshti University of Medical Sciences (ethical code: IR.SBMU.LASER.REC.1403.028).

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