Journal of Lasers in Medical Sciences

J Lasers Med Sci 2026;17:e51845

Original Article

https://journals.sbmu.ac.ir/jlms

doi:10.34172/jlms.51845

Introducing Cell Cycle as the Main Target of UVA Radiation

Samaneh Azimi Souteh[1], Babak Arjmand[2,3], Zahra Razzaghi[4], Farideh Razi[5], Fatemeh Bandarian[6], Mostafa Rezaei-Tavirani[7*], Nastarn Asri[8]

[1]Department of Radiology, School of Medicine, Iran University of Medical Sciences, Tehran, Iran
[2]Hematology-Oncology and Stem Cell Transplantation Research Center, Research Institute for Oncology, Hematology, and Cell Therapy, Tehran University of Medical Sciences, Tehran, Iran
[3]Iranian Cancer Control Center (MACSA), Tehran, Iran
[4]Laser Application in Medical Sciences Research Center, Shahid Beheshti University of Medical Sciences, Tehran, Iran
[5]Diabetes Research Center, Endocrinology and Metabolism Clinical Sciences Institute, Tehran University of Medical Sciences, Tehran, Iran
[6]Endocrinology and Metabolism Research Center, Endocrinology and Metabolism Clinical Sciences Institute, Tehran University of Medical Sciences, Tehran, Iran
[7]Proteomics Research Center, Faculty of Paramedical Sciences, Shahid Beheshti University of Medical Sciences, Tehran, Iran
[8]Celiac Disease and Gluten Related Disorders Research Center, Research Institute for Gastroenterology and Liver Disease, Shahid Beheshti University of Medical Sciences, Tehran, Iran

***Correspondence to**
Mostafa Rezaei-Tavirani,
Email: tavirany@yahoo.com

Received: February 21, 2026
Accepted: July 13, 2026
ePublished: October 27, 2026

Abstract

Introduction: UVA is characterized with 320-400 nm wavelengths and has considerable biological effects. Assessment of the molecular mechanism of UVA radiation on human normal corneal endothelial cells via gene expression analysis by using protein-protein interaction (PPI) assessment is the aim of this study.

Methods: Gene expression profiles of human normal cornea endothelial cells in the presence and absence of UVA exposure are extracted from the Gene Expression Omnibus (GEO) database and are evaluated via GEO2R program to find the significant differentially expressed genes (DEGs). The identified DEGs were evaluated via directed PPI network analysis and gene ontology assessment to find the critical targeted genes and biological processes.

Results: Results of PPI network analysis revealed that 28 DEGs play roles as actors and 28 individuals are key controlled genes. A total of 49 critical DEGs and eight biological process groups were identified as the targeted genes and biological terms by UVA radiation.

Conclusion: In conclusion, CCND1, CDC20, and PLK1 were highlighted as the top targeted genes by UVA radiation. On the other hand, dysregulation of the cell cycle was pointed out as the main consequence of UVB exposure. Experimental data can be recommended to support the findings of this project.

Keywords: UVA radiation, Network analysis, Gene expression, Human

Introduction

UVA (320-400 nm) is the least destructive part of UV radiation, while UVC (200-280 nm) is a very dangerous portion of the UV spectrum that affects living organisms. UVB (280-320 nm) is the middle part of the UV spectrum, exposure to which is accompanied by considerable damages in creatures.[1] Since UVA is a huge quantity of the solar spectrum which can reach the earth's surface and can also penetrate effectively into human skin, it has attracted researchers' attention in the human health field. The production of reactive nitrogen and oxygen species in the cells exposed to UVA is a risk factor for DNA damage and disturbances to membrane lipids.[2] Investigations have demonstrated that interleukins, intercellular adhesion molecules, transcription factors, and several genes which are involved in inflammation are significantly targeted by UVA.[3]

Gene expression analysis is a suitable method for exploring the molecular mechanism of biological events such as treatments and diseases. The targeted genes demonstrate possible malfunctions which affect the biological and physiological aspects of the studied organisms. Bioinformatics is another method that is applied in biology and medicine to solve the large set of data in high-throughput investigations.[4-6] PPI network analysis is an attractive method for exploring relationships between a set of proteins, genes, or metabolites. In directed PPI networks, the nodes of the studied network are connected to neighbors via directed edges such as activation or inhibition.[7,8]

Please cite this article as follows: Azimi Souteh S, Arjmand B, Razzaghi Z, Razi F, Bandarian F, Rezaei-Tavirani M et al. Introducing cell cycle as the main target of UVA radiation. J Lasers Med Sci 2026;17:e51845. doi:10.34172/jlms.51845

Outdegree value refers to the number of edges that exit from a certain node while indegree refers to the edges that enter into an individual. The nodes with higher values of outdegree play roles as actors. On the other hand, the nodes that receive a higher number of edges are the controlled nodes. There are several nodes that are characterized with high values of outdegree + indegree (the nodes with higher values of count edged). The mentioned nodes function as central individuals in the directed PPI network.[8-10] PPI network analysis demonstrated that SMC3, ARF3, EIF5B, SMARCB1, and LAPTM5 are the critical targeted genes via NB-UVB radiation in the skin of the treated atopic dermatitis patients. Also, cell proliferation, cell cycle, apoptosis, and inflammation were pointed out as the targeted cellular functions in this experiment.[11]

Gene ontology method is an appropriate method for exploring the related molecular function, biological processes, and biochemical pathways for a set of genes.[12] In the present study, gene expression profiles of human corneal endothelial cells in the presence and absence of UVA are extracted from the GEO database and assessed via PPI network analysis and gene ontology assessment to identify the crucial targeted genes and biological processes.

Methods

Data collection

Human corneal endothelial cells and ultraviolet-A exposure were searched in the GEO database. A set of "gene expression profiles", including four untreated cell profiles and four profiles of irradiated individuals, was pointed out as the suitable object to be analyzed (https://www.ncbi.nlm.nih.gov/geo/geo2r/?acc=GSE250224).

Pre-analysis

Comparability of the two candidate groups was assessed via the GEO2R program. The significant DEGs were determined based on an "adjusted *P* value" < 0.05 and a "log fold change" > 1. Data were cleaned via deletion of the uncharacterized individuals and the miRNAs.

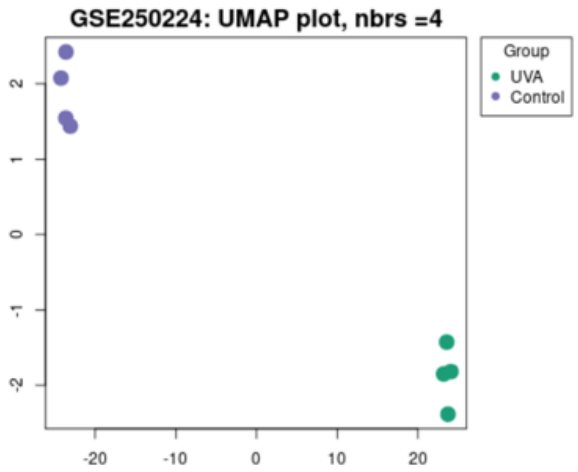


Figure 1. Uniform Manifold Approximation and Projection (UMAP) plot for the gene expression profiles of the irradiated human corneal endothelial cells with UVA in comparison with the controls

PPI network analysis

The cleaned significant DEGs were evaluated via directed PPI network analysis using CluePedia v1.5.10 application within Cytoscape software v3.10.1 to identify activation, inhibition, and expression relationships among the queried DEGs. The PPI network was analyzed using the "Network analyzer" plugin of Cytoscape software in directed edges analysis mode. Outdegree, indegree, and edge count parameters for nodes of the main connected component of the analyzed PPI network were identified.

Gene ontology enrichment

The critical genes were enriched to find the crucial targeted biological processes via ClueGO application v2.5.10. The related biological processes were extracted from the "GO_BiologicalProcess-EBI-UniProt-GOA-ACAP-ARAP_25.05.2022_00h00" source.

Results

Pre-analysis

Four gene expression profiles (GSM7975851-4) as the treated human corneal endothelial cells with UVA radiation were compared with four gene expression profiles of the untreated individuals (GSM7975843-6) as the control group. The Uniform Manifold Approximation and Projection (UMAP) plot of the analysis is presented in Figure 1. Based on the UMAP plot, the gene expression profiles of the irradiated cells with UVA are completely separated from the gene expression profiles of the control cells, which refers to the significant effect of UVA radiation on the gene expression of human corneal endothelial cells. As depicted in the Venn diagram (Figure 2), the expression of 1,245 genes was changed significantly by UVA radiation. After cleaning, a total of 539 significant DEGs were candidates to be analyzed via the directed PPI network.

PPI network analysis

The main connected component of the directed PPI network is presented in Figure 3. To find the critical controller and controlled DEGs, the distribution of nodes based on edge count, indegree, and outdegree parameters was administrated (see Figures 4-6). As depicted in

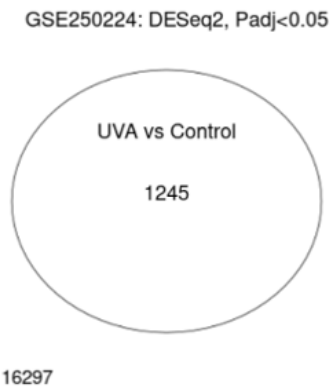


Figure 2. Venn diagram of the gene expression analysis of UVA radiation on human corneal endothelial cells. A total of 1,245 genes are significantly dysregulated

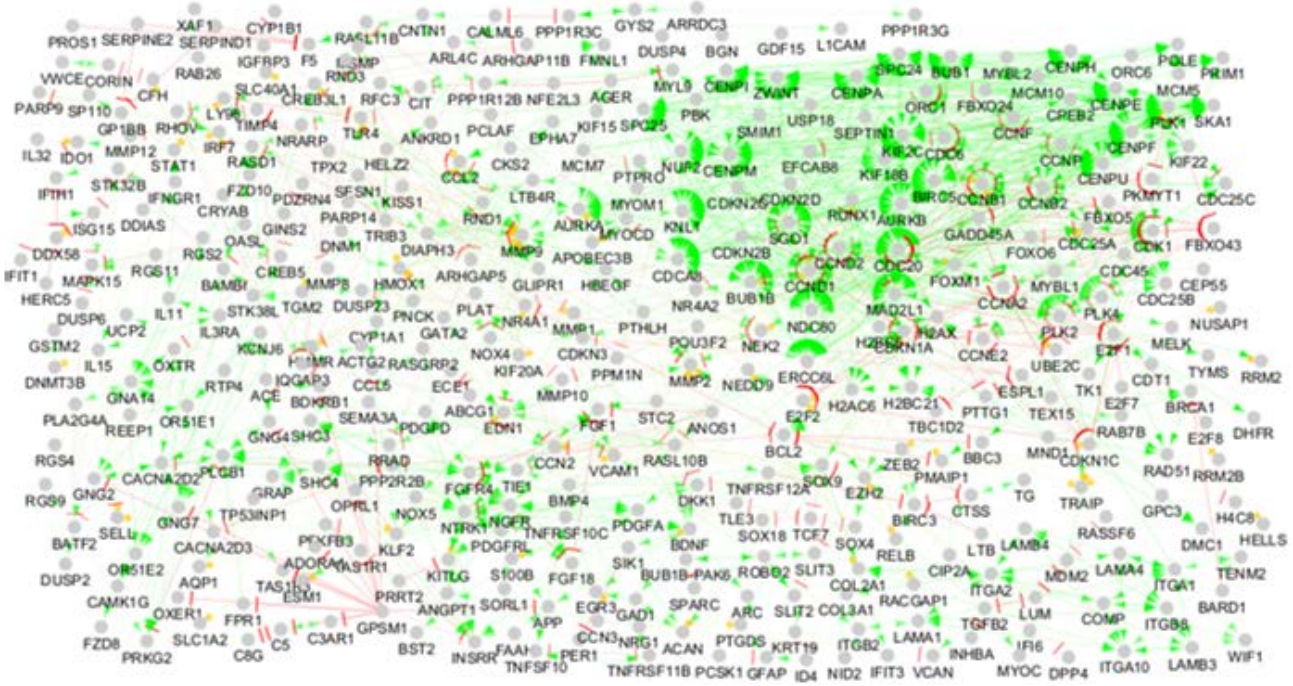


Figure 3. The main connected component of the directed PPI for 539 significant DEGs. The activation, inhibition, and expression actions are presented in green, red, and yellow colors

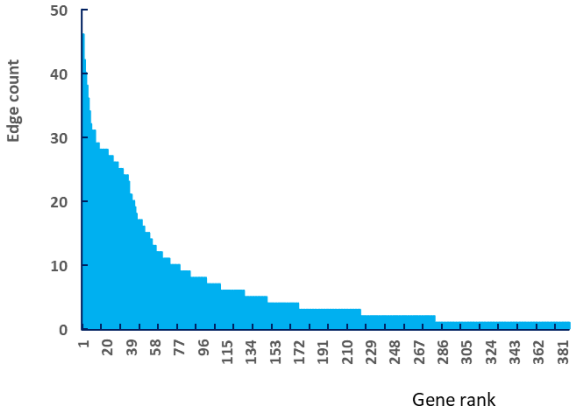


Figure 4. Distribution of nodes based on the edge count for the genes in the main connected component of the PPI network. The horizontal axis refers to the rank of nodes when they were arranged from the largest to the smallest based on edge counts for the nodes. A total of 386 nodes with the edge count > 0 are included in the diagram

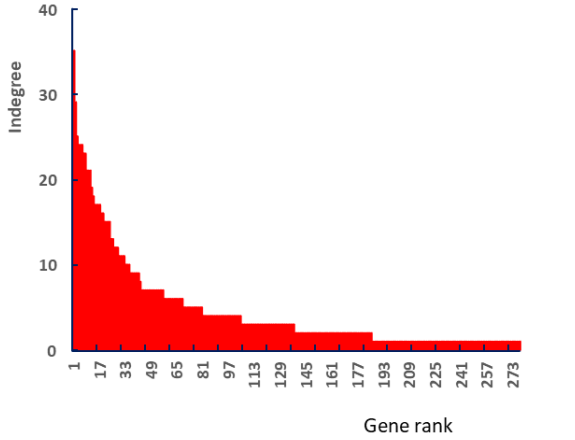


Figure 5. Distribution of nodes based on indegree value for the genes in the main connected component of the PPI network. The horizontal axis refers to the rank of nodes when arranged from the largest to the smallest based on the indegree value for the nodes. A total of 277 nodes with an indegree value > 0 are included in the diagram

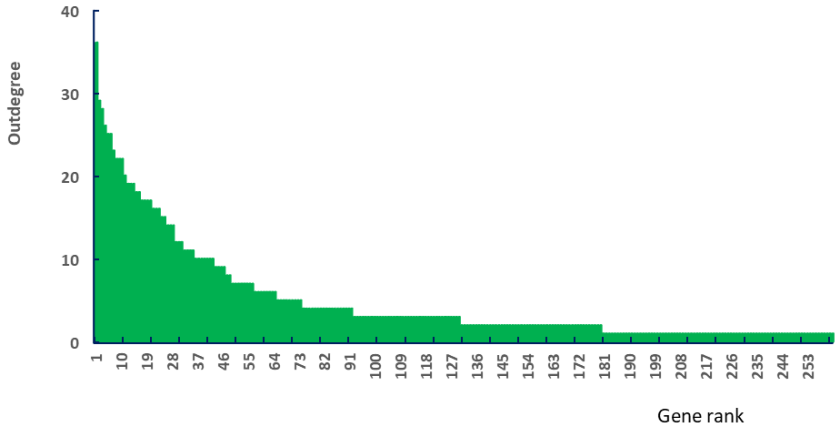


Figure 6. Distribution of nodes based on outdegree value for the genes in the main connected component of the PPI network. The horizontal axis refers to the rank of nodes when arranged from the largest to the smallest based on outdegree value for the nodes. A total of 261 nodes with outdegree value > 0 are included in the diagram

Figure 4, the diagram is biphasic and 40 nodes (about 10% of all nodes) are presented in the first phase, and the others are arranged in the second phase. To find a logical relationship between nodes based on edge count, outdegree, and indegree values, the top 10% of nodes based on these parameters were selected for more investigations (see Tables 1-3). CCL2 is the last node in Table 2 with an edge count of 21. Therefore, CDKN1A with an edge count of 21 was added to Table 1. The last node in Table 2 was BIRC5 which was characterized with an outdegree value of 14. Therefore, MMP9 and EZF2 that have a similar situation to BIRC5 were added to Table 2. After deletion of the repeated nodes between the three presented tables, a total of 49 genes remained for more analysis. The relationship between the 49 critical genes is demonstrated in Figure 7.

Table 1. List of top 10% nodes of the main connected component of the PPI network based on the edge count

No.	Term	Edge count	Indegree	Outdegree
1	CDC20	46	35	11
2	PLK1	42	17	25
3	CCND1	40	4	36
4	CDK1	38	29	9
5	E2F1	36	19	17
6	FOXM1	34	23	11
7	BUB1	32	16	16
8	MAD2L1	31	15	16
9	BUB1B	31	12	19
10	NDC80	31	3	28
11	BIRC5	29	15	14
12	SGO1	29	3	26
13	AURKA	29	0	29
14	CENPI	28	24	4
15	CDCA8	28	24	4
16	CENPF	28	21	7
17	KIF2C	28	21	7
18	SPC25	28	9	19
19	NUF2	28	6	22
20	CCNB1	28	6	22
21	KNL1	27	17	10
22	AURKB	27	15	12
23	CENPH	27	9	18
24	CENPE	27	4	23
25	SPC24	26	25	1
26	MMP9	26	12	14
27	SKA1	26	9	17
28	CENPM	26	1	25
29	ZWINT	25	23	2
30	ERCC6L	25	15	10
31	PLK2	25	7	18
32	CENPU	25	6	19
33	KIF18B	24	24	0
34	EDN1	24	21	3
35	CENPA	24	17	7
36	SOX9	24	2	22
37	EZH2	23	9	14
38	CDKN1A	21	18	3
39	CCL2	21	4	17
40	CDKN1A	21	18	3

Gene ontology enrichment

The 49 critical genes were enriched to find the related targeted biological processes by UVA radiation (see Figure 8). Eight groups of biological processes were identified that were related mostly to "mitotic cell cycle" processes. As depicted in Figure 8, 39 DEGs were related to biological process groups, of which 29 individuals (about 74%) were connected to the largest group (negative regulation of the mitotic cell cycle phase transition).

Discussion

It is well established that UVA radiation in various cell cycle stages induces the formation of multinucleated Cells.[13] In the present study, UVA radiation has targeted 49 critical

Table2. List of the top 10% of the main connected components of the PPI network nodes based on outdegree value

N0.	Term	Edge count	Indegree	Outdegree
1	CCND1	40	4	36
2	AURKA	29	0	29
3	NDC80	31	3	28
4	SGO1	29	3	26
5	PLK1	42	17	25
6	CENPM	26	1	25
7	CENPE	27	4	23
8	NUF2	28	6	22
9	CCNB1	28	6	22
10	SOX9	24	2	22
11	CDC6	20	0	20
12	BUB1B	31	12	19
13	SPC25	28	9	19
14	CENPU	25	6	19
15	CENPH	27	9	18
16	PLK2	25	7	18
17	E2F1	36	19	17
18	SKA1	26	9	17
19	CCL2	21	4	17
20	NGFR	17	0	17
21	BUB1	32	16	16
22	MAD2L1	31	15	16
23	PLK4	20	4	16
24	CCND2	17	2	15
25	MMP2	15	0	15
26	BIRC5	29	15	14
27	MMP9	26	12	14
28	EZH2	23	9	14

Table3. List of the top 10% of the main connected components of the PPI network nodes based on the indegree value

No.	Term	EdgeCount	Indegree	Outdegree
1	CDC20	46	35	11
2	CDK1	38	29	9
3	SPC24	26	25	1
4	CENPI	28	24	4
5	CDCA8	28	24	4
6	KIF18B	24	24	0
7	FOXM1	34	23	11
8	ZWINT	25	23	2
9	CENPF	28	21	7
10	KIF2C	28	21	7
11	EDN1	24	21	3
12	E2F1	36	19	17
13	CDKN1A	21	18	3
14	PLK1	42	17	25
15	KNL1	27	17	10
16	CENPA	24	17	7
17	DIAPH3	17	17	0
18	BUB1	32	16	16
19	BDNF	17	16	1
20	MAD2L1	31	15	16
21	BIRC5	29	15	14
22	AURKB	27	15	12
23	ERCC6L	25	15	10
24	TLR4	16	13	3
25	CCNP	13	13	0
26	BUB1B	31	12	19
27	MMP9	26	12	14
28	GPSM1	14	12	2

genes that are mostly connected to the mitosis process. A total number of 39 genes from the critical 49 DEGs were connected to "negative regulation of the mitotic cell cycle phase transition", "regulation of the G2/M transition of the mitotic cell cycle", "establishment of mitotic spindle localization", "establishment of spindle orientation", "centromeric sister chromatid cohesion", "mitotic DNA integrity checkpoint signaling", "establishment of mitotic spindle orientation", and "kinetochore assembly" biological processes. As we know, continuous and excessive cell division leads to a group of diseases which are named cancer. Therefore, cell division is strongly controlled by critical and evolutionarily conserved cell cycle mechanisms. These controller systems operate to ensure the normal production of two identical cells.[14] The role of many genes that control the cell division process (e.g., 1,871 unique cell cycle–regulated genes) is reported in the literature.[15]

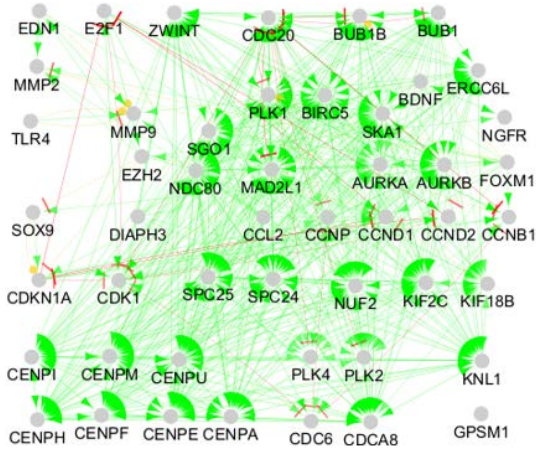


Figure 7. Relationships (activation: green, inhibition: red, and expression: yellow) between the 49 critical genes resulting from edge count, outdegree, and indegree analyses

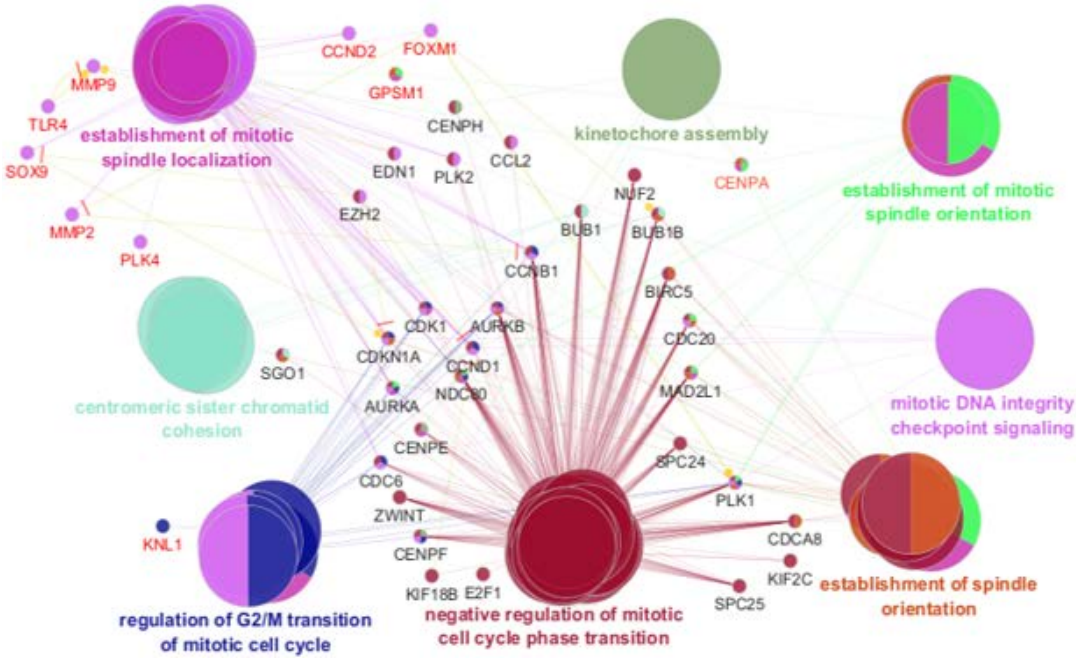


Figure 8. Gene ontology results for the 49 critical genes. Eight groups of biological processes and the associated genes are presented. The black labeled genes are linked to the "negative regulation of cell cycle phase transition" biological process group

The top actor in this project is cyclin D1 (CCND1), which is targeted by UVA radiation in human corneal endothelial cells. As depicted in Table 2, this gene regulates 36 genes which are involved in controlling the cell cycle process. There is evidence that CCND1 is a critical intermediary of cell cycle progression and the foremost cyclin which controls the cell transition from G1 to S phase. The crucial role of CCND1 in the pathogenesis of cancer is highlighted in the literature. Clinical application of CCND1 is pointed out by researchers.[16,17] Cyclin D1 is ranked as the third gene in Table 1 based on the edge count value, which refers to the regulation of CCND1 by other genes. Induced accumulation of CCND1 following UVA radiation in human HaCaT keratinocytes is reported in the literature.[18]

On the other hand, cell division cycle 20 (CDC20) appears as the top controlled gene with an indegree value of 35 (see Table 3). Similar to CCND1, CDC20 is linked to the most biological processes groups (see Figure 8). The significant role of CDC20 in cell cycle regulation has been investigated and confirmed. It is pointed out that the regulation of CDC20 can be supportive in cancer therapy.[19] CDC20 appears as the top gene in Table 1 because it is characterized with an edge count value of 46. This means that CDC20 is not only an important controlled gene but also a major actor in cell cycle regulation.

The second-ranked gene in Table 1 is polo-like kinase 1 (PLK1). PLK1 is characterized with outdegree and indegree values of 25 and 17, respectively. PLK1 is connected to the six groups of biological processes (see Figure 8). Higher edge cut value and connection to 75% of the biological process groups refer to the crucial role of PLK1 in the regulation of the cell cycle. This position of PLK1 leads to introducing PLK1-inhibition base combination as a cancer therapy protocol.[20]

As demonstrated in Tables 2 and 3, E2F1, PLK1, BUB1, MAD2L1, BIRC5, and BUB1B are highlighted as key controlled and actor genes which are targeted by UVA radiation. Involvement of E2F transcription factor 1 (E2F1), BUB1 mitotic checkpoint serine/threonine kinase (BUB1), mitotic arrest deficient 2 like 1 (MAD2L1), and baculoviral IAP repeat-containing 5 (BIRC5) in cancer progression is pointed out by researchers.[21-24]

Conclusion

In conclusion, the cell cycle and the related genes such as CCND1, CDC20, and PLK1 were highlighted as the target of UVA radiation. The findings can be considered as the key points of the molecular mechanism of the effect of UVB radiation on human cells. Experimental investigations are recommended to evaluate the results of this project.

Acknowledgments

This project is supported by Shahid Beheshti University of Medical Sciences.

Authors' Contribution

Conceptualization: Nikoo Hossein-Khannazer, Farideh Razi Fatemeh Bandarian Mostafa Rezaei-Tavirani
Data Curation: Samaneh Azimi Souteh
Formal Analysis: Nastarn Asri
Investigation: Nastarn Asri
Methodology: Samaneh Azimi Souteh, Fatemeh Bandarian
Project Administration: Zahra Razzaghi, Babak Arjmand, Fatemeh Bandarian
Resources: Nastarn Asri
Software: Samaneh Azimi Souteh
Supervision: Mostafa Rezaei-Tavirani
Validation: Zahra Razzaghi, Farideh Razi
Visualization: Zahra Razzaghi
Writing–Original Draft: Samaneh Azimi Souteh, Zahra Razzaghi
Writing–Review & Editing: Mostafa Rezaei-Tavirani, Babak Arjmand, Farideh Razi

Competing Interests

There is no Competing interests.

Ethical Approval

This project is confirmed with IR.SBMU.RETECH.REC.1404.1113 ethical code.

Funding

This project is supported by Shahid Beheshti University of Medical Sciences.

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