European Journal of Therapeutics
pISSN: 2564-7784
eISSN: 2564-7040

Eur J Ther. 2026.
https://doi.org/10.58600/eurjther3044

Original Research

Effects of Boric Acid on *ABL1* Gene Expression in Swiss Albino Mice Exposed to High-Dose Total Body Irradiation

Seda Ekici[1*], Bekir Eren Çetin[2], Yasin Korucu[3], Akın Yiğin[4]

[1] Veterinary Control Central Research Institute, Ankara, Türkiye
[2] Department of Radiation Oncology, Gazi University of Medicine, Ankara, Türkiye
[3] Department of Radiation Oncology, Hitit University Erol Olcok Training and Research Hospital, Çorum, Türkiye
[4] Department of Genetics, Faculty of Veterinary Medicine, Harran University, Şanlıurfa, Türkiye

Received: 2026–02–03 **Accepted:** 2026–08–17 **Published Online:** 2026–09–07

Corresponding Author
Seda Ekici, PhD
Address: Veterinary Control Central Research Institute, Ankara, Turkiye
E-mail: seda.ergen@hotmail.com

© 2026. The copyright of this article is retained by the author(s).

OPEN ACCESS

This work is licensed under a Creative Commons Attribution-NonCommercial 4.0 International License.

This license permits the free sharing and adaptation of the work for non-commercial purposes, provided that appropriate attribution is given to the original author(s) and to its initial publication in this journal.

ABSTRACT

Objective: Radiation-induced DNA damage activates multiple cellular stress response pathways, including those associated with the *ABL1* gene. This study investigated whether boric acid administration alters *ABL1* gene expression following high-dose total body irradiation (TBI) in Swiss albino mice.

Methods: Seventy-two Swiss albino mice were allocated into control, radiotherapy (RT), boric acid (30, 100, or 300 mg/kg), and combined boric acid plus radiotherapy groups. Total body irradiation was delivered as a single 8 Gy dose on day 0. Boric acid was administered orally once daily, beginning two days prior to irradiation and continued until sacrifice on day 2 or day 6 post-irradiation. *ABL1* gene expression levels were quantified by real-time polymerase chain reaction (RT-PCR), normalized to beta-actin (*ACTB*), and calculated using the $2^{-\Delta\Delta Ct}$ method.

Results: *ABL1* gene expression was elevated in irradiated groups compared with controls. A statistically significant increase was observed in the group receiving 100 mg/kg boric acid combined with RT on day 2 ($p < 0.050$). Although other treatment groups also demonstrated increased *ABL1* expression, these differences were not statistically significant, which may be attributable to inter-individual variability.

Conclusion: Boric acid appears to modulate radiation-induced alterations in *ABL1* gene expression in a dose- and time-dependent manner. The observed effects were more pronounced during the early post-irradiation period, suggesting a potential influence of boric acid on early molecular responses to radiation exposure.

Keywords: *ABL1* gene, boric acid, gene expression, total body irradiation

INTRODUCTION

Ionizing radiation exerts its biological effects primarily through the induction of DNA damage and the generation of reactive oxygen species, leading to oxidative stress within the cell. These events trigger a complex network of intracellular signaling pathways responsible for DNA repair, cell cycle regulation, and programmed cell death. The coordinated activation of these mechanisms is critical for maintaining cellular integrity after radiation exposure, whereas disruption of this balance may result in acute toxicity as well as delayed tissue damage [1,2].

Among the proteins involved in the cellular response to genotoxic stress, *ABL1* is a ubiquitously expressed non-receptor tyrosine kinase that plays a role in DNA damage signaling. Previous studies have demonstrated that *ABL1* is responsive to ionizing radiation and participates in pathways that influence cell survival, apoptosis, and repair processes [3,4]. Changes in *ABL1* expression or activity have been linked to altered radiosensitivity, suggesting that this molecule may reflect radiation-induced molecular disturbances at the cellular level [5,6].

High-dose total body irradiation (TBI) is commonly employed in experimental models to investigate systemic radiation effects. Exposure to TBI results in widespread alterations in gene expression, particularly in pathways related to stress responses and DNA repair mechanisms [7,8]. Importantly, radiation-induced molecular responses are not static and may be modified by external factors, including pharmacological agents or nutritional supplements [9].

Main Points

- Boric acid administration modulated *ABL1* gene expression in irradiated mice in a dose- and time-dependent manner.
- A statistically significant increase in *ABL1* expression was observed only in the 100 mg/kg boric acid + radiotherapy group on day 2 post-irradiation.
- The effect of boric acid on *ABL1* expression was more prominent in the early period after radiation exposure.
- These findings suggest that boric acid may influence early molecular stress responses following high-dose radiation.

Boric acid, a naturally occurring boron-containing compound, has gained increasing interest due to its reported antioxidant and cytoprotective effects. While boron compounds have been evaluated in different experimental and clinical settings, data regarding their potential role in modulating radiation-induced molecular changes remain limited [10–12]. In particular, the effect of boric acid on genes directly involved in DNA damage signaling, such as *ABL1*, has not been clearly elucidated. Moreover, information on dose-related and time-dependent responses to boric acid following high-dose radiation exposure is scarce [13,14].

Therefore, the present study was designed to investigate the effects of varying doses of boric acid on *ABL1* gene expression in Swiss albino mice subjected to 8 Gy total body irradiation. By assessing gene expression at both early and later time points after irradiation, this study aims to contribute to a better understanding of the potential modulatory role of boric acid in radiation-induced molecular responses.

MATERIALS AND METHODS

All experimental procedures were approved by the Local Animal Care and Use Committee of Gazi University Faculty of Medicine (Decision No: G.Ü.ET-26.002, dated January 21, 2026) and were carried out in accordance with national and international guidelines for the care and use of laboratory animals.

Animals and Experimental Design

This study was conducted using 72 Swiss albino mice. Animals were randomly allocated into control, radiotherapy (RT), boric acid-treated, and boric acid plus radiotherapy groups. The day of irradiation was designated as day 0. In the combined treatment groups, animals were further divided into two subgroups, with half sacrificed on day 2 and the remaining half on day 6 following irradiation.

Total Body Irradiation

Total body irradiation was administered to conscious mice placed in well-ventilated acrylic cages, each housing six animals. Irradiation procedures were performed using a GE Saturne 43 linear accelerator (General Electric Medical Systems, Buc, France). A single dose of 8 Gy was delivered as total body irradiation using 6 MV photon X-rays, with a source-to-surface distance of 100 cm.

Boric Acid Treatment

Boric acid of pharmaceutical grade (Merck; EMSURE ACS, ISO; CAS No: 10,043–35–3) was administered by oral gavage at doses of 30 mg/kg, 100 mg/kg, or 300 mg/kg, corresponding to low (b), medium (B), and high-dose (BB) treatment groups, respectively. Treatments were initiated two days prior to irradiation (day −2) and continued once daily until the time of sacrifice. A fixed volume of 0.2 mL was administered per dose. Mice in the control and RT-only groups received an equivalent volume of distilled water.

RNA Isolation and RT-PCR Analysis

Total RNA was extracted from intracardiac blood samples using a column-based extraction method in accordance with the manufacturer's instructions (GeneAll Hybrid-R™ RNA extraction kit). Complementary DNA (cDNA) was synthesized from isolated RNA samples, followed by quantitative RT-PCR analysis using a SYBR Green-based detection system (Roche Diagnostics GmbH, Mannheim, Germany) on a QuantStudio™5 real-time PCR platform (Applied Biosystems, Thermo Fisher Scientific, Waltham, MA, USA).

ABL1 was analyzed as the target gene, while ACTB served as the endogenous reference gene. Primer sequences used in the study are shown in Table 1. PCR reactions were performed using a SYBR Green master mix, and each sample was analyzed in duplicate. Relative expression levels were calculated using the $2^{-\Delta\Delta Ct}$ method, with the control group sacrificed on day 6 used as the reference group [15]. The control group sacrificed on day 6 was used as the calibrator for all comparative analyses.

Statistical Analysis

Statistical analyses were performed using standard statistical software. ΔCt values were used for all statistical comparisons. Data are presented as mean ± standard deviation (SD). Comparisons between each experimental group and the control group were conducted using the independent-samples t-test. A *p*-value of less than 0.05 was considered statistically significant.

RESULTS

***ABL1* Gene Expression**

Changes in *ABL1* gene expression following high-dose total body irradiation and boric acid administration were evaluated by quantitative real-time PCR analysis. *ABL1* gene expression levels are presented in Table 2. The relative *ABL1* gene expression levels in the experimental groups are presented in Figure 1.

Table 1. Primer sequences used for quantitative real-time PCR

Gene	Primer	Sequence (5′–3′)	Reference
ABL1	Forward	AGCCACTTCGGATTTAGTTGAA	[15]
	Reverse	GCAACCGGCTTGCATGT	
ACTB	Forward	CCTCCCTGGAGAAGAGCTATG	[16]
	Reverse	TTACGGATGTCAACGTCACAC	

Table 2. Relative ABL1 Gene Expression Levels

Group	Fold Change ($2^{-}\Delta\Delta Ct$, Mean ± SD)	p
RT (2 days)	20.58 ± 27.95	0.070
RT (6 days)	6.10 ± 4.66	0.154
bRT (2 days)	1.17 ± 1.11	0.668
bRT (6 days)	11.35 ± 16.87	0.090
BRT (2 days)	5.83 ± 4.62	0.038*
BRT (6 days)	4.22 ± 3.37	0.760
BBRT (2 days)	8.98 ± 10.17	0.946
BBRT (6 days)	21.31 ± 34.73	0.191

* $p < 0.050$ compared with control group.

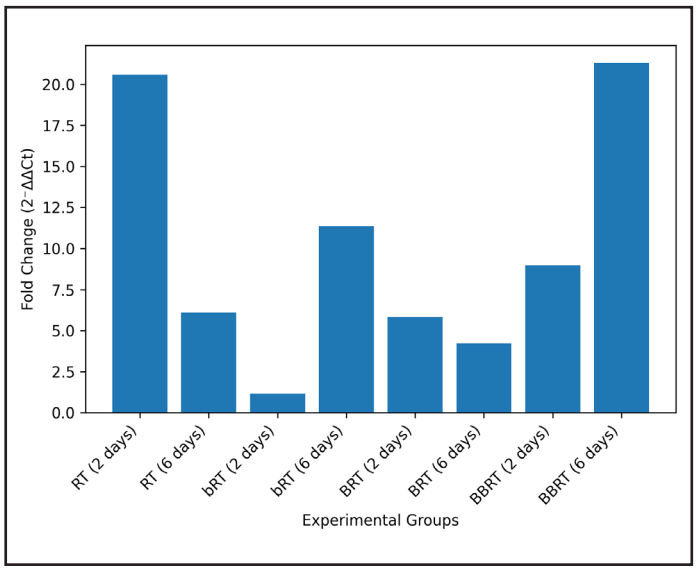

Figure 1. Relative *ABL1* gene expression levels in the radiotherapy (RT), low-dose boric acid plus radiotherapy (bRT), medium-dose boric acid plus radiotherapy (BRT), and high-dose boric acid plus radiotherapy (BBRT) groups at 2 and 6 days post-irradiation. Radiotherapy was defined as day 0.

In the radiotherapy (RT) group, *ABL1* expression was increased on day 2 compared with the control group, although this increase did not reach statistical significance ($p = 0.070$). By day 6, *ABL1* expression in the RT group decreased relative to day 2.

In the low-dose boric acid plus radiotherapy group (bRT), *ABL1* expression on day 2 was comparable to control levels. On day 6, a slight increase in expression was observed; however, this change was not statistically significant ($p = 0.090$).

In the medium-dose boric acid plus radiotherapy group (BRT), *ABL1* expression was significantly increased on day 2 compared with the control group ($p < 0.050$). By day 6, expression levels decreased and were not significantly different from control values.

In the high-dose boric acid plus radiotherapy group (BBRT), *ABL1* expression showed high inter-individual variability at both time points. Although increased fold changes were observed in some samples, these differences were not statistically significant.

DISCUSSION

The present study investigated the effects of radiotherapy alone and radiotherapy combined with varying doses of boric acid on *ABL1* gene expression at early and late time points following irradiation. The results demonstrate that *ABL1* expression is influenced by both post-irradiation time and boric acid dose, indicating a potential modulatory effect of boric acid on radiation-induced cellular signaling pathways.

Ionizing radiation induces DNA double-strand breaks and activates DNA damage sensors, including ataxia telangiectasia mutated (*ATM*) kinase [17,18]. *ATM* activation triggers downstream signaling cascades involved in cell-cycle arrest, DNA repair, and apoptosis, predominantly through *p53*-dependent pathways. Targeting key components of the DNA damage response, such as *ATM* or poly (ADP-ribose) polymerase (*PARP*), has been shown to enhance radiosensitivity by impairing DNA repair mechanisms and promoting cell death [19,20].

ABL1 is recognized as an important mediator in genotoxic stress responses and apoptotic regulation [21]. Although it is not a core DNA damage response kinase such as *ATM* or poly (ADP-ribose) polymerase *(PARP)*, *ABL1* can be activated following DNA damage and interact with p53 family members, particularly *p73,* thereby contributing to apoptosis under conditions of severe genomic stress [21]. Previous studies have shown that *ABL1* activation may enhance *p53*-dependent apoptotic signaling and increase apoptosis-related molecular responses following DNA damage [22].

In the radiotherapy-only groups, *ABL1* expression exhibited a transient increase at day 2 post-irradiation, followed by a decrease at day 6. This pattern likely reflects an acute response to radiation-induced DNA damage, followed by partial recovery through DNA repair processes and/or elimination of highly damaged cells via apoptosis. Similar temporal expression patterns have been reported for other DNA damage response-related genes, supporting an early activation phase followed by a decline during recovery [23,24].

When radiotherapy was combined with boric acid, distinct dose-dependent expression profiles were observed. In the low-dose boric acid plus radiotherapy group, *ABL1* expression at the early time point remained close to control levels. This suggests that low-dose boric acid does not substantially modify radiation-induced activation of DNA damage signaling pathways. This observation is consistent with reports indicating that low concentrations of boron compounds may exert antioxidant or stabilizing effects, potentially attenuating stress-responsive gene activation [25].

In the medium-dose boric acid plus radiotherapy group, *ABL1* expression was significantly increased at day 2 post-irradiation. This finding suggests that medium-dose boric acid may enhance early radiation-induced DNA damage signaling. One possible explanation is that boric acid at this concentration may increase oxidative or genotoxic stress, thereby amplifying activation of DNA damage response pathways, including *ABL1* associated signaling [26,27]. Increased *ABL1* activity may facilitate apoptotic signaling through interactions with *p73* and other *p53* family members, particularly when DNA damage exceeds cellular repair capacity. Such a shift toward apoptosis over repair is a well-established mechanism of radiosensitization [28,29].

By day 6, *ABL1* expression in the medium-dose group decreased toward baseline levels, suggesting that the modulatory effect of boric acid is transient and predominantly confined to the early post-irradiation phase. This indicates a time-dependent regulatory influence rather than sustained gene activation. In contrast, high-dose boric acid combined with radiotherapy resulted in marked inter-individual variability in *ABL1* expression at both time points. Although increased fold changes were observed in some samples, the variability precluded statistical significance. This heterogeneity may reflect excessive cellular stress or dose-related toxicity, leading to divergent activation of compensatory survival and repair mechanisms.

Consistent with these findings, studies targeting key DNA damage response kinases such as *ATM* have demonstrated that inhibition of these pathways can enhance radiation-induced apoptosis and impair DNA repair in experimental models [30,31,32]. In particular, pharmacological inhibition of *ATM* has been reported to increase radiation-induced cell death and to promote immunomodulatory effects that may improve tumor control [32,33]. These observations support the concept that modulation of DNA damage response signaling pathways can significantly influence cellular radiosensitivity. Taken together, the results suggest that moderate modulation of DNA damage signaling, including pathways involving *ABL1*, may contribute to radiosensitization by favoring apoptotic responses over DNA repair. Nevertheless, current evidence remains insufficient to fully explain the mechanistic relationship between boric acid exposure, *ABL1* activation, and radiation-induced cellular responses [11].

Future studies incorporating protein-level analyses of DNA damage response and apoptotic markers such as *ATM*, *p53*, *p73*, and caspases, as well as functional assays evaluating radiation sensitivity (e.g., clonogenic survival or tumor control models) would provide further insight into these interactions.

In conclusion, this study demonstrates that boric acid modulates radiation-induced *ABL1* gene expression in a dose- and time-dependent manner, with the most pronounced effect observed at the medium dose during the early post-irradiation period. These findings contribute to the understanding of boron–radiation interactions and provide a molecular basis for future investigations into the potential role of boric acid as a radiomodulatory agent.

Limitations

ABL1 expression was assessed only at the mRNA level; protein expression and functional activity were not evaluated. In addition, gene expression was analyzed at limited post-irradiation time points, which may not fully capture the long-term molecular effects of boric acid following radiation exposure.

CONCLUSION

The findings of this study demonstrate that boric acid influences radiation-induced *ABL1* gene expression in a dose- and time-dependent manner. Radiotherapy alone induced a transient increase in *ABL1* expression, whereas combined treatment with boric acid resulted in different expression patterns depending on dose level. The most pronounced effect was observed with the medium dose of boric acid, which was associated with increased *ABL1* expression during the early post-irradiation period. In contrast, low-dose boric acid did not significantly alter *ABL1* expression, while high-dose administration resulted in inter-individual variability in expression levels. Overall, the results summarize dose- and time-dependent changes in radiation-induced *ABL1* gene expression following boric acid administration. The study provides molecular-level evidence of an association between boric acid exposure and radiation-induced gene expression changes. Further mechanistic studies are required to clarify the biological relevance of these findings.

Funding: None.

Conflict of Interest: The authors declare no conflict of interest.

Informed Consent: Not applicable, as this study was conducted on experimental animals.

Data Availability Statement: The data supporting the findings of this study are available from the corresponding author upon reasonable request.

Ethical Approval: All experimental procedures were approved by the Local Animal Care and Use Committee of Gazi University Faculty of Medicine (Decision No: G.Ü.ET-26.002, dated January 21, 2026) and were carried out in accordance with national and international guidelines for the care and use of laboratory animals.

Author Contributions: Conception: S, E; B, E, Ç - Design:B, E, Ç; S, E; Y, K - Supervision: A, Y - -Materials: S, E; B, E, Ç ; Y, K - Data Collection and/or Processing: S, E; B, E, Ç ; Y, K Analysis and/or Interpretation S, E; B, E, Ç ; Y, K - Literature: A,Y, Review: S, E; B, E, Ç ; Y, K Writing: S, E; B, E, Ç ; Y, K Critical Review: A,Y

Author Statement Regarding the Use of Artificial Intelligence-Enhanced Tools: The authors declare that artificial intelligence-enhanced tools were used during the preparation of this manuscript solely for language editing, grammar correction, and improvement of clarity.

REFERENCES

[1] Saini S, Gurung P (2025) A comprehensive review of sensors of radiation-induced damage, radiation-induced proximal events, and cell death. Immunol Rev. 329(1):e13409. https://doi.org/10.1111/imr.13409

[2] Hu Z, Chen Y, Yang S, Diao S, Ma L, Shi C (2025) Radiation-induced biological effects: molecular and cellular mechanisms and applications to radiation/nuclear emergency and cancer therapy. MedComm. 6(12):e70478. https://doi.org/10.1002/mco2.70478

[3] Long F, Wang H, Sun Z, Yuan J (2025) BCR::ABL1-positive secondary B-acute lymphoblastic leukemia mimicking acute megakaryoblastic leukemia following multiple myeloma. Turk J Haematol. https://doi.org/10.4274/tjh.galenos.2025.2025.0277

[4] Kosik P, Skorvaga M, Belyaev I (2023) Preleukemic fusion genes induced via ionizing radiation. Int J Mol Sci. 24(7):6580. https://doi.org/10.3390/ijms24076580

[5] Vilar JB, Christmann M, Tomicic MT (2022) Alterations in molecular profiles affecting glioblastoma resistance to radiochemotherapy. Cancers (Basel). 14(10):2416. https://doi.org/10.3390/cancers14102416

[6] Cyran M, Stawarz K, Chambily L, Kusza K, Siemionow M (2024) Assessment of hematopoietic response to total body irradiation in a rat experimental model. Ann Plast Surg. 93(1):100–106. https://doi.org/10.1097/SAP.0000000000003962

[7] Liu R, Bian Y, Liu L, Liu X, Ma S (2022) Molecular pathways associated with oxidative stress and their potential applications in radiotherapy. Int J Mol Med. 49(5):65. https://doi.org/10.3892/ijmm.2022.5121

[8] Obrador E, Salvador-Palmer R, Villaescusa JI, Gallego E, Pellicer B, Estrela JM, et al. (2022) Nuclear and radiological emergencies: biological effects, countermeasures and biodosimetry. Antioxidants (Basel). 11(6):1098. https://doi.org/10.3390/antiox11061098

[9] Kumar R, Thakur A, Kumar S, Hajam YA (2024) Royal jelly as a promising therapeutic intervention and functional food supplement: a systematic review. Heliyon. 10(17):e37138. https://doi.org/10.1016/j.heliyon.2024.e37138

[10] Koldemir-Gündüz M, Aydin HE, Berikten D, Kaymak G, Köse DA, Arslantaş A (2021) Synthesis of new boron-derived compounds: anticancer, antioxidant and antimicrobial effects in vitro glioblastoma tumor model. J Korean Neurosurg Soc. 64(6):864–872. https://doi.org/10.3340/jkns.2021.0032

[11] Maliszewska-Olejniczak K, Kaniowski D, Araszkiewicz M, Tymińska K, Korgul A (2021) Molecular mechanisms of DNA damage response induced by mixed radiation fields during boron neutron capture therapy. Front Oncol. 11:676575. https://doi.org/10.3389/fonc.2021.676575

[12] Punshon LD, Fabbrizi MR, Phoenix B, Green S, Parsons JL (2024) Current insights into the radiobiology of boron neutron capture therapy and the potential for further improving biological effectiveness. Cells. 13(24):2065. https://doi.org/10.3390/cells13242065

[13] Skorski T (2002) BCR/ABL regulates response to DNA damage: role in resistance to genotoxic treatment and genomic instability. Oncogene. 21(56):8591–8604. https://doi.org/10.1038/sj.onc.1206087

[14] Matheson CJ, Venkataraman S, Amani V, Harris PS,

Backos DS, Donson AM, et al. (2016) A WEE1 inhibitor analog of AZD1775 maintains synergy with cisplatin. ACS Chem Biol. 11(4):921–930. https://doi.org/10.1021/acschembio.5b00938

[15] Livak KJ, Schmittgen TD (2001) Analysis of relative gene expression data using real-time quantitative PCR and the 2−ΔΔCT method. Methods. 25(4):402–408. https://doi.org/10.1006/meth.2001.1262

[16] Malnassy G, Keating CR, Gad S, Bridgeman B, Perera A, Hou W, Cotler SJ, Ding X, Choudhry M, Sun Z, Koleske AJ, Qiu, W (2023) Inhibition of ABL2 suppresses alcohol-associated liver disease. Cell Mol Gastroenterol Hepatol. 16(5):685–709. https://doi.org/10.1016/j.jcmgh.2023.07.006

[17] Kosir R, Acimovic J, Golicnik M, Perse M, Majdic G, Fink M, Rozman D (2010) Determination of reference genes for circadian studies. BMC Mol Biol. 11:60. https://doi.org/10.1186/1471-2199-11-60

[18] Huang RX, Zhou PK (2020) DNA damage response signaling pathways and targets for radiotherapy sensitization. Signal Transduct Target Ther. 5(1):60. https://doi.org/10.1038/s41392-020-0150-X

[19] Olive PL (1998) The role of DNA single- and double-strand breaks in cell killing by ionizing radiation. Radiat Res. 150(Suppl 5):S42–S51. https://doi.org/10.2307/3579807

[20] Han W, Yu KN (2010) Ionizing radiation, DNA double strand breaks and mutation. In: Advances in Genetic Research, Vol. 4. Nova Science Publishers, New York, pp. 197–210.

[21] Hintelmann K, Kriegs M, Rothkamm K, Rieckmann T (2020) Improving tumor radiosensitization through combined molecular targeting. Front Oncol. 10:1260. https://doi.org/10.3389/fonc.2020.01260

[22] Gonfloni S (2010) DNA damage stress response in germ cells: role of c-Abl and clinical implications. Oncogene. 29(47):6193–6202. https://doi.org/10.1038/onc.2010.362

[23] Borlak J, Ciribilli Y, Bisio A, Selvaraj S, Inga A, Oh JH, Spanel R (2024) The Abl1 tyrosine kinase is a key player in doxorubicin-induced cardiomyopathy and its p53/p73 cell death mediated signaling differs in atrial and ventricular cardiomyocytes. J Transl Med. 22(1):845. https://doi.org/10.1186/s12967-024-05623-8

[24] Cruz-Garcia L, Nasser F, O'Brien G, Grepl J, Vinnikov V, Starenkiy V, Artiukh S, Gramatiuk S, Badie C (2022) Transcriptional dynamics of DNA damage-responsive genes during radiotherapy. Cancers (Basel). 14(11):2649. https://doi.org/10.3390/cancers14112649

[25] Kar F, Hacioglu C, Senturk H, Donmez DB, Kanbak G (2020) The Role of Oxidative Stress, Renal Inflammation, and Apoptosis in Post Ischemic Reperfusion Injury of Kidney Tissue: the Protective Effect of Dose-Dependent Boric Acid Administration. Biol Trace Elem Res. 195:150–158. https://doi.org/10.1007/s12011-019-01824-1

[26] Paganetti H (2023) Lymphocyte radiosensitivity and its impact on radiotherapy. Front Oncol. 13:1201500. https://doi.org/10.3389/fonc.2023.1201500

[27] Kim W, Lee S, Seo D, Kim D, Kim K, Kim E, Kang J, Seong KM, Youn H, Youn B (2019) Cellular stress responses in radiotherapy. Cells. 8(9):1105. https://doi.org/10.3390/cells8091105

[28] Bhattacharya S, Asaithamby A (2017) Repurposing DNA repair factors to eradicate tumor cells upon radiotherapy. Transl Cancer Res. 6(Suppl 5):S822–S830. https://doi.org/10.21037/tcr.2017.05.22

[29] Chen KH, Lai ZY, Li DY, Lin YC, Chou FI, Chuang YJ (2019) Analysis of DNA damage responses after boric acid-mediated boron neutron capture therapy in hepatocellular carcinoma. Anticancer Res. 39(12):6661–6671. https://doi.org/10.21873/anticanres.13881

[30] Bohio AA, Wang R, Zeng X, Ba X (2020) c-Abl-mediated tyrosine phosphorylation of DNA damage response proteins and implications in important cellular functions. Mol Med Rep. 22(2):612–619. https://doi.org/10.3892/mmr.2020.11156

[31] Garcia MEG, Kirsch DG, Reitman ZJ (2022) Targeting the ATM kinase to enhance the efficacy of radiotherapy and outcomes for cancer patients. Semin Radiat Oncol. 32(1):3–14. https://doi.org/10.1016/j.semradonc.2021.09.008

[32] Hak CMCW, Rullan A, Patin EC, Pedersen M, Melcher AA, Harrington KJ (2022) Enhancing anti-tumour innate immunity by targeting the DNA damage response and pattern recognition receptors in combination with radiotherapy. Front Oncol. 12:971959. https://doi.org/10.3389/fonc.2022.971959

[33] Chiu LY, Sun Q, Zenke FT, Blaukat A, Vassilev LT (2023)

Selective ATM inhibition augments radiation-induced inflammatory signaling and cancer cell death. Aging (Albany NY). 15(2):492–505. https://doi.org/10.18632/aging.204487

How to Cite;

Ekici S, Cetin BE, Korucu Y, Yigin A (2026) Effects of Boric Acid on Abl1 Gene Expression in Swiss Albino Mice Exposed to High-Dose Total Body Irradiation. Eur J Ther. https://doi.org/10.58600/eurjther3044