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Original Article

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Non-Invasive ELF Magnetic Fields Suppress Bladder Cancer Cell Viability through Apoptotic Pathways

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Abstract

Background and Aim: This study was undertaken to rigorously investigate the effects of extremely low-frequency magnetic fields (ELF-MF) on bladder cancer cells and apoptosis-related pathways.

Methods: T24 and 5637 cell lines were exposed to a 6 mT, 50 Hz magnetic field for 2 hours per day over three consecutive days. Cell viability was systematically assessed by MTT assay which revealed a significant reduction in survival of cancer cells following ELF-MF exposure.

Results: Quantitative real-time PCR analysis showed downregulation of the anti-apoptotic gene BCL2 and upregulation of the tumor suppressor PTEN. Moreover, the tumor-suppressive microRNA miR-34a was markedly increased, while the oncogenic miR-21 was decreased. These effects were largely specific to cancer cells, with minimal impact on normal urothelial cells, thereby indicating a degree of selective biological responsiveness.

Conclusion: The results suggest that ELF-MF can modulate key apoptotic regulators and selectively impair cancer cell viability. This work highlights the potential of ELF-MF as a non-invasive physical modality to enhance apoptosis and suppress tumor progression, providing a foundation for future studies in combination with conventional chemotherapeutics.

Keywords: bladder cancer, extremely low-frequency magnetic fields (ELF-MF), miR-34a, miR-21, PTEN, BCL2

1. Introduction

Central to its pathogenesis and therapeutic resistance is a dysregulation of the apoptotic machinery: cells escape programmed cell death, enabling survival, proliferation and invasion. In this context, the intrinsic (mitochondrial) and extrinsic (death-receptor) apoptotic pathways are modulated by a complex interplay including proteins that promote or inhibit apoptosis, such as members of the BCL2 family, caspases, IAPs (inhibitor of apoptosis proteins) and upstream regulators such as PTEN and p53 (1). Recent reviews emphasise the BCL2-axis and mitochondrial outer membrane permeabilization (MOMP) as key nodes in cancer cell survival (2). Over the past decade, microRNAs (miRNAs) have emerged as pivotal post-transcriptional regulators of apoptosis in cancer (3-5). In bladder cancer, for instance, the tumour-suppressive miR-34a is frequently down-regulated and has been shown to inhibit cell migration, invasion and promote apoptosis by targeting BCL2, SIRT1 and E2F3 (6).

Conversely, the oncomiR miR-21 is often up-regulated in urothelial carcinoma and promotes proliferation, inhibits apoptosis and confers chemo resistance through suppression of PTEN and up-regulation of BCL2/AKT signaling (7, 8). Drug-resistance and evasion of apoptosis in bladder cancer often correlate with overexpression of BCL-2, decreased PTEN function and altered miRNA signatures (9, 10).

Growing data suggest that ELF-MFs can induce measurable physicochemical alterations in biological structures and membrane-associated molecules, raising the possibility that such non-ionizing stimuli

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may have regulatory functions at the cellular and subcellular levels (11-13) .

Several recent investigations suggest that ELF fields may alter gene expression (14), miRNA profiles, oxidative stress and apoptosis signaling in various cell types including cancer cells. Mansoury et al. demonstrated that ELF electromagnetic fields modulate miRNA expression in cancer cell lines (4). More specifically in bladder cancer, Sandberg et al. reported that pulsed electromagnetic field (PEMF) exposure in HT-1197 bladder cancer cells slowed proliferation and induced broad alterations in cancer-relevant gene-expression pathways (15). Despite existing evidence, the mechanistic link between ELF-MF exposure and miRNA-mediated regulation of apoptosis in bladder cancer remains insufficiently understood. The hypothesis that ELF-MF influences cell survival through modulation of miR-34a and miR-21 and their downstream targets, including BCL2 and PTEN, is supported by the central roles of these miRNAs in apoptotic signaling, their association with clinical outcomes, and similar observations reported in other cancer models. Recent studies indicate that microRNA–gene regulatory networks, particularly miR-34a through targeting BCL2, play a key role in regulating apoptosis in bladder cancer (16-18). Dysregulation of these pathways promotes uncontrolled cell proliferation, therapeutic resistance, and tumor relapse. Accordingly, restoring pro-apoptotic microRNAs or modulating downstream targets such as PTEN represents a promising strategy to inhibit tumor progression(19). Recent studies suggest that exosomal miRNAs regulate apoptotic and survival signaling, providing regulatory principles relevant to bladder cancer biology (3). Understanding the interaction between magnetic fields and miRNA networks is essential for developing non-invasive therapies, as ELF-MF can modulate intracellular signaling and apoptosis without inducing thermal or genotoxic damage (20). Cancer cells may exhibit greater sensitivity to ELF-MF due to altered metabolic and electrophysiological states, enabling selective targeting of malignant pathways (21). Despite accumulating evidence, the molecular effects of ELF-MF on microRNA-mediated regulation of apoptosis in bladder cancer remain poorly defined. To address this knowledge gap, the present study investigated the impact of a well-controlled 6 mT, 50 Hz extremely low-frequency magnetic field on the expression of apoptosis-related microRNAs and their downstream targets, including miR-34a, miR-21, BCL2, PTEN, and associated apoptotic markers, in bladder cancer cells compared with normal urothelial controls. We hypothesized that ELF-MF exposure modulates the expression of miR-34a and miR-21, thereby altering intrinsic apoptotic signaling pathways and promoting apoptosis in bladder cancer cells.

2. Methods

2.1Cell Culture

Bladder cancer cell lines T24 and 5637 were obtained from the Pasteur Institute of Iran. Cells were grown in RPMI-1640 medium enriched with 10% fetal bovine serum (FBS) and 1% penicillin-streptomycin, under standard conditions of 37 °C and 5% CO_2 in a humidified incubator. Cultures were monitored daily and passaged every 2–3 days to maintain healthy exponential growth. Only actively dividing cells were utilized for subsequent experimental procedures.

2.2 ELF-MF Set up

Extremely low-frequency magnetic field (ELF-MF) exposure was performed using a solenoid-based exposure system. The apparatus consisted of a cylindrical solenoid coil wound with 1200 turns of insulated copper wire (1 mm diameter), arranged in four uniform layers, with a total length of 30 cm. The solenoid was powered by a AC current source (model: PS-A305D, DAZHENG, Shanghai, China), operating at 50 Hz, generating a uniform magnetic field with a flux density of 6 mT.

To ensure magnetic field homogeneity within the cell culture volume, culture plates were positioned at the central region of the solenoid along its longitudinal axis, where the field distribution is maximally uniform. Magnetic flux density and field uniformity were verified prior to experimentation using a high-precision Gaussmeter. The entire solenoid system was placed inside a standard humidified CO_2 incubator (37 °C, 5% CO_2) to maintain physiological culture conditions during exposure. The system was equipped with an active cooling unit, and temperature was continuously monitored throughout exposure; no measurable temperature fluctuations were detected, confirming the absence of thermal effects. Cells were exposed to ELF-MF for 2 hours per day over three consecutive days. Sham control cultures were maintained under identical environmental and incubator conditions and placed within an identical solenoid apparatus that remained unenergized, thereby controlling for potential effects of residual electrical currents, heat generation, mechanical vibrations, or handling.

2.3 RNA and miRNA Extraction

After exposure to ELF-MF, cells were rinsed twice with ice-cold phosphate-buffered saline (PBS) and subsequently lysed. Total RNA was isolated using TRIzol reagent (Invitrogen) in accordance with the manufacturer's instructions. In brief, following

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Archives of Advances in Biosciences

chloroform treatment, the aqueous phase containing RNA was collected, precipitated with isopropanol, washed with 75% ethanol, and finally resuspended in RNase-free water. RNA yield and purity were assessed using a NanoDrop spectrophotometer, while RNA integrity was verified through agarose gel electrophoresis.

For small RNA, including microRNAs, extraction was carried out using the miRNeasy Mini Kit (Qiagen). This procedure involved cell lysis, binding of RNA to a silica membrane, sequential washing, and elution. Both total RNA and isolated miRNAs were stored at −80 °C until further analysis.

2.4 Quantitative Real-Time PCR (qRT-PCR)

Complementary DNA (cDNA) was generated from total RNA using a reverse transcription kit (Thermo Fisher Scientific). For microRNA profiling, stem-loop primers specific to miR-34a and miR-21 were utilized during the reverse transcription step. Quantitative PCR was carried out with SYBR Green Master Mix on a Bio-Rad CFX96 Real-Time PCR system, employing a cycling protocol of 40 cycles consisting of denaturation at 95 °C for 15 seconds and annealing/extension at 60 °C for 60 seconds. Gene expression was normalized to GAPDH for messenger RNAs and U6 small nuclear RNA for microRNAs. The relative abundance of transcripts was determined using the comparative ΔΔCt method.

2.5 Cell Viability Assay (MTT)

The impact of ELF-MF on cell viability was evaluated using the MTT colorimetric assay. Cells were plated at a density of 5×10^3 per well in 96-well plates and exposed to the magnetic field treatment. Following exposure, 20 µL of MTT solution (5 mg/mL) was added to each well and incubated for 4 hours to allow for formazan crystal formation. The crystals were subsequently solubilized in 150 µL of DMSO, and absorbance was recorded at 570 nm using a microplate reader. Cell viability was calculated as a percentage relative to untreated control wells.

2.6 Statistical Analysis

All experiments were carried out in triplicate. Results are expressed as mean ± standard deviation (SD). Differences between two groups were evaluated using an unpaired Student's t-test, while comparisons among multiple groups were conducted with one-way ANOVA followed by Tukey's post-hoc analysis. A p-value less than 0.05 was considered indicative of statistical significance. All statistical analyses were performed using GraphPad Prism version 9.

3. Results

3.1 MTT Analysis of Cell Viability Following ELF-MF Exposure

MTT assay results demonstrated that exposure to extremely low-frequency magnetic fields (ELF-MF) significantly reduced the viability of bladder cancer cells. In the T24 cell line, cell viability was markedly decreased compared with the control group, reaching approximately 58% of control levels, corresponding to a 42% reduction ($p < 0.001$). Similarly, in 5637 cells, viability was reduced to approximately 62% of control levels, indicating a 38% decrease relative to controls ($p < 0.01$).

In contrast, ELF-MF exposure did not significantly affect the viability of normal urothelial cells, with survival rates remaining comparable to those of the control group ($p > 0.05$). These findings indicate that the inhibitory effect of ELF-MF on cell viability is predominantly restricted to malignant bladder cancer cells, suggesting a selective cytotoxic effect on can

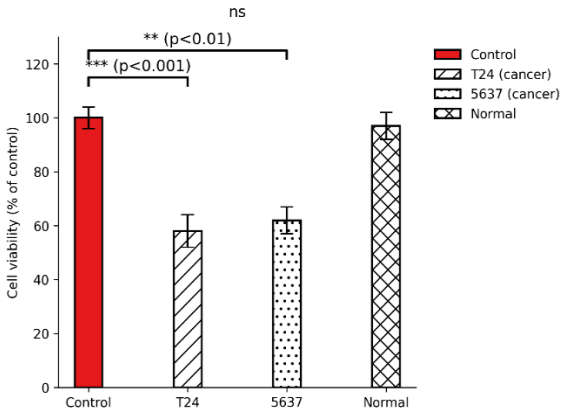


Figure 1. Effect of ELF-MF exposure on cell viability assessed by MTT assay; data are expressed as mean ± SD relative to the control group (set to 100%), with statistical significance indicated (** *p < 0.01*, *** *p < 0.001; ns, not significant vs. control*).

3.2 Gene and microRNA Expression Analysis After ELF-MF Exposure

Table 1 and figure 2 summarizes the fold changes in gene and microRNA expression in T24 bladder cancer cells following ELF-MF exposure. As shown in this table, expression of BCL2 was significantly reduced, showing an approximately 0.42±0.06 fold decrease compared with control cells ($p < 0.01$). In contrast, PTEN expression was significantly increased, exhibiting a 1.28±0.1 fold upregulation ($p < 0.001$). In addition, miR-34a expression was markedly upregulated by approximately 1.35±0.14 fold ($p < 0.001$), whereas miR-21 expression was significantly downregulated by approximately 0.38 ±0.05 fold relative to controls ($p < 0.01$).

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Table 1. expression change of BCL2, PTEN, miR-34a, and miR-21 in T24 urothelial cells following exposure to ELF-MF. Expression levels were determined using qPCR and normalized using the 2^-ΔCt method. Values are presented as mean ± standard deviation (SD) from three independent experiments. Statistical significance was calculated using Student's t-test, with $p < 0.05$ considered significant.

Gene/miRNA	Expression change (Mean ± SD)	pvalue
BCL2	0.42 ± 0.06	p < 0.01
PTEN	1.28± 0.1	p < 0.001
miR34-a	1.35 ± 0.14	p < 0.001
miR-21	0.38± 0.05	p < 0.01

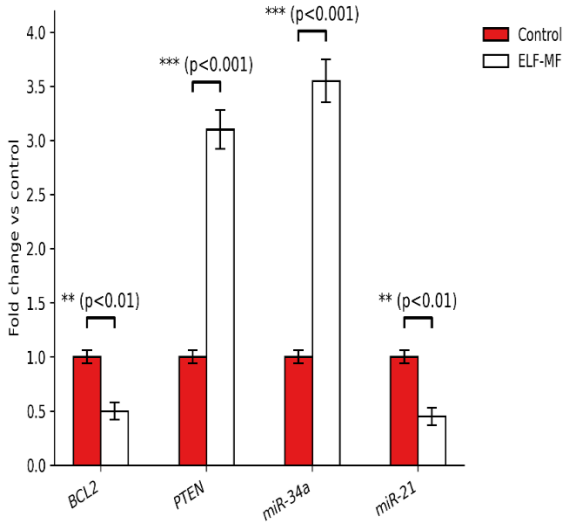


Figure 2. qRT-PCR analysis of BCL2, PTEN, miR-34a, and miR-21 expression in T24 cells after ELF-MF exposure. Data are shown as fold change versus control (mean ± SD, n = 3). Statistical analysis was performed using one-way ANOVA with Tukey's post hoc test (*p < 0.05, **p < 0.01, ***p < 0.001). ELF-MF, extremely low-frequency magnetic field.

Table 2 and figure 3 presents the corresponding results for the 5637 bladder cancer cell line, demonstrating a gene and microRNA expression pattern comparable to that observed in T24 cells. Specifically, BCL2 expression decreased by approximately 0.45±0.06 fold ($p < 0.01$), while PTEN expression increased by approximately 1. 25±0.1 fold ($p < 0.001$). Similarly, miR-34a was upregulated by approximately 1.32±0.14 fold ($p < 0.001$), whereas miR-21 expression was reduced by approximately 0.4±0.05 fold ($p < 0.01$).

Table 2. expression change of BCL2, PTEN, miR-34a, and miR-21 in 5637 urothelial cells following exposure to ELF-MF. Expression levels were determined using qPCR and normalized using the 2^-ΔCt method. Values are presented as mean ± standard deviation (SD) from three independent experiments. Statistical significance was calculated using Student's t-test, with $p < 0.05$ considered significant.

Gene/miRNA	Expression change (Mean ± SD)	pvalue
BCL2	0.45 ± 0.06	p < 0.01
PTEN	1.25± 0.1	p < 0.001
miR34-a	1.32 ± 0.14	p < 0.001
miR-21	0.4 ± 0.05	p < 0.01

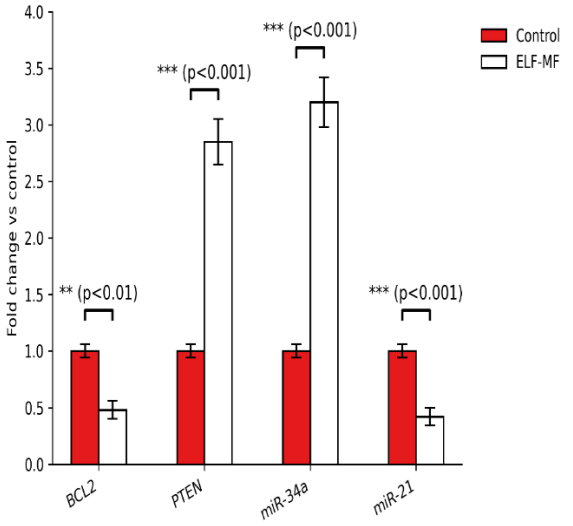


Figure 3. qRT-PCR analysis of BCL2, PTEN, miR-34a, and miR-21 expression in 5637 cells after ELF-MF exposure. Data are shown as fold change versus control (mean ± SD, n = 3). Statistical analysis was performed using one-way ANOVA with Tukey's post hoc test (*p < 0.05, **p < 0.01, ***p < 0.001). ELF-MF, extremely low-frequency magnetic field.

Table 3 and figure 4 shows the effects of ELF-MF exposure on normal urothelial cells. In contrast to cancer cell lines, no statistically significant changes were detected in the expression of BCL2, PTEN, miR-34a, or miR-21 in normal cells ($p > 0.05$), indicating that ELF-MF exposure selectively alters gene and microRNA expression profiles in malignant bladder cells while sparing non-malignant urothelial cells.

Table 3. expression change of BCL2, PTEN, miR-34a, and miR-21 inand normal cells following exposure to ELF-MF. Expression levels were determined using qPCR and normalized using the 2^-ΔCt method. Values are presented as mean ± standard deviation (SD) from three independent experiments. Statistical significance was calculated using Student's t-test, with $p < 0.05$ considered significant.

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Archives of Advances in Biosciences

Gene/miRNA	Expression change (Mean ± SD)	pvalue
BCL2	1 ± 0.08	p > 0.05
PTEN	1.02± 0.9	p > 0.05
miR34-a	1.05 ± 0.1	p > 0.05
miR-21	0.98± 0.09	p > 0.05

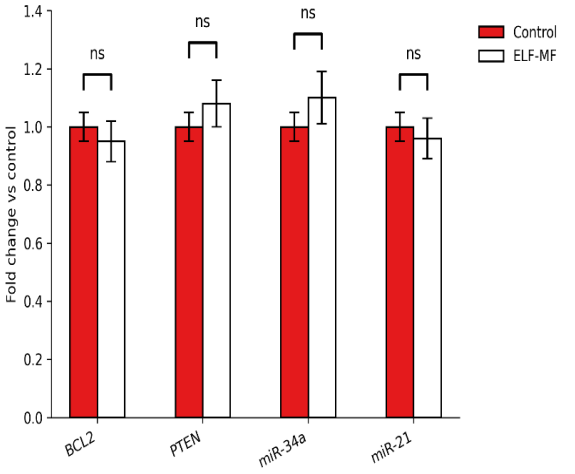


Figure 4. qRT-PCR analysis of BCL2, PTEN, miR-34a, and miR-21 expression in normal cells after ELF-MF exposure. Data are shown as fold change versus control (mean ± SD, n = 3). Statistical analysis was performed using one-way ANOVA with Tukey's post hoc test (*p < 0.05, **p < 0.01, ***p < 0.001).

4. Discussion

Exposure of bladder cancer cells to extremely low-frequency magnetic fields (ELF-MF) at 6 mT and 50 Hz produced a consistent anti-tumor response in our experiments. This response was characterized by a significant reduction in cell viability, downregulation of the anti-apoptotic gene BCL2, and a pronounced upregulation of the tumor-suppressive microRNA miR-34a, along with increased expression of its downstream target PTEN. These alterations were observed in comparison with the cancer-cell control group, which consisted of malignant cells not exposed to the magnetic field.

These findings strengthen the hypothesis that malignant cells inherently exhibit heightened sensitivity to physical perturbations such as ELF-MF, a concept increasingly supported by emerging studies in cancer biology. Specifically, multiple investigations have reported that transformed cells display unstable redox homeostasis, mitochondrial fragility, and disrupted microRNA networks, features that render them more vulnerable to additional stressors when compared with their own untreated cancer-cell controls, leading to a more pronounced response to magnetic-field exposure (22).

One of the most striking results in our study is the coordinated upregulation of miR-34a, a well-established tumor suppressor regulated by TP53, and the concomitant suppression of BCL2, a canonical cell-survival regulatory protein. The miR-34a/BCL2 axis is a recognized cell survival–regulating pathway in bladder cancer, and decreased miR-34a is strongly linked with progression, recurrence, and chemoresistance in urothelial tumors (23). Restoration of miR-34a has been shown to enhance TP53-dependent stress responses and sensitize cancer cells to stressors, including oxidative signals and DNA damage (24). Thus, the magnetic fields–induced elevation of miR-34a observed here provides a plausible mechanistic explanation for the reduced cellular viability in bladder cancer cells. Moreover, the significant increase in PTEN expression following ELF-MF exposure suggests another mechanistic route through which magnetic fields may impair malignant cell survival. PTEN is a master inhibitor of PI3K/AKT survival signaling and one of the most frequently dysregulated tumor suppressors across cancers. Even modest increases in PTEN expression can shift cells toward suppression of pro-survival signaling, especially under metabolic or oxidative stress conditions (19). It has been demonstrated that exposure to extremely low-frequency electromagnetic fields (ELF-EMF) reduces BCL2 expression and modulates its regulatory miRNAs, miR-15b and miR-16, thereby influencing cell survival–related signaling pathways(25). Magnetic fields can influence biological systems through multiple mechanisms, ranging from modulation of enzyme activity to changes in gene expression. Physically, this may occur via interactions with mobile charges inside cells, which can alter their biological function. Evidence suggests that enzymes such as Na,K-ATPase and cytochrome oxidase are sensitive to magnetic fields. Additionally, magnetic fields may directly affect DNA by accelerating electrons, generating repulsive forces that could locally unwind the double helix. Certain promoter sequences, like nCTCTn, may act as magnetic sensors, amplifying these effects on DNA structure (26). Another proposed mechanism involves the impact of oscillating magnetic fields on free ions in the cell membrane. Forced vibrations of these ions can perturb membrane channels, such as calcium channels, disrupting electrochemical gradients. This disturbance may trigger intracellular signaling cascades that enhance enzymatic reactions and modify the transcription of specific genes (27).

Studies have demonstrated that magnetic fields can modulate transcriptional and epigenetic regulators of PTEN and other cell fate–associated genes in a range of normal and cancerous tissues (20). In accordance

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with our study, these data suggest that ELF-MF exposure may influence tumor biology through both microRNA-mediated and transcription-mediated control of cell survival regulatory networks. The selectivity of ELF-MF toward cancer cells, with only marginal effects on normal urothelial controls in our study, is consistent with several reports showing cancer-specific susceptibility. Metabolic studies indicate that malignant cells operate closer to the threshold of redox imbalance, making them more sensitive to mitochondrial perturbation induced by magnetic exposure, including changes in ATP synthase activity and proton flux along the respiratory chain (21). Furthermore, ELF-MF has been shown to reshape miRNA networks involved in proliferation and cell survival regulation, as reported in gastric carcinoma models exposed to 50 Hz fields, where shifts in miRNA expression correlated with reduced proliferation and increased cellular stress markers (28, 29). As a future perspective, comparative analysis of intracellular ROS levels and mitochondrial membrane potential between bladder cancer and normal urothelial cells following ELF-MF exposure could provide mechanistic insight into the observed selective sensitivity. Such studies would clarify whether redox imbalance and mitochondrial fragility underlie the preferential anti-tumor response to magnetic field stimulation. Despite the growing evidence supporting anti-cancer effects of ELF-MF, the field remains controversial. Some studies report negligible or inconsistent effects across cell types, emphasizing that biological responses to magnetic fields may depend on field strength, frequency, exposure duration, cell lineage, and metabolic state (30). Furthermore, while most high-quality animal studies do not demonstrate a carcinogenic effect of ELF-MF, they highlight the complexity of dose-response relationships and raise the need for rigorous standardization of exposure systems (31). It is therefore essential to interpret our findings within the broader context of highly variable biological sensitivity to ELF-MF.

Nevertheless, the pattern observed in our study suppressed BCL2, enhanced PTEN, elevated miR-34a, and reduced viability strongly suggests that magnetic fields can modulate essential cell survival and stress-response circuits in bladder cancer cells. A plausible explanation is that ELF-MF perturbs cellular electrical or redox homeostasis, triggering compensatory stress-response pathways that ultimately converge on gene regulatory mechanisms controlling cell survival and adaptation. This interpretation aligns with the established role of electric and magnetic cues in modulating ion channel activity, redox signalling, and DNA repair responses in mammalian cells(32, 33).

In summary, the present study demonstrates that exposure to extremely low-frequency magnetic fields can selectively modulate cell viability as well as gene and microRNA expression profiles in bladder cancer cells, while exerting minimal effects on normal urothelial cells. The consistent downregulation of BCL2, upregulation of PTEN, and coordinated changes in key microRNAs following ELF-MF exposure suggest that magnetic fields are capable of influencing critical regulatory networks involved in cancer cell survival and stress responsiveness. Despite these limitations, the overall pattern observed—suppressed BCL2, enhanced PTEN, elevated miR-34a, and reduced viability provides strong evidence that ELF-MF modulates essential cell survival and stress-response circuits in bladder cancer cells. A plausible mechanism involves perturbation of cellular electrical and redox homeostasis, triggering compensatory stress-response pathways that converge on gene regulatory networks controlling cell survival and adaptation. These findings highlight the potential of ELF-MF as a selective anti-cancer physical intervention and underscore its relevance for further mechanistic exploration. Looking forward, future studies should focus on in vivo validation of these effects, direct functional assays to confirm downstream cellular outcomes, and comparative analyses of ROS levels and mitochondrial membrane potential in cancer versus normal cells. Additionally, exploring variable field strengths, exposure durations, and frequencies will be essential to fully characterize the biological and translational significance of ELF-MF exposure in bladder cancer research.

5. Conclusion

In conclusion, this study shows that extremely low-frequency magnetic field exposure selectively reduces cell viability and modulates gene and microRNA expression in bladder cancer cells, while having minimal effects on normal urothelial cells. These findings suggest that malignant cells exhibit increased sensitivity to ELF-MF and highlight the potential of magnetic fields as a non-invasive approach for influencing cancer-associated molecular profiles.

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Ethical Considerations and Compliance with Ethical Guidelines

All cell lines used in this study were obtained from authenticated and reputable cell repositories and were handled in accordance with institutional bio safety and ethical guidelines. Standard laboratory practices were followed to prevent contamination and

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misidentification of cell lines, including routine authentication and mycoplasma testing. All experiments were conducted following established bio safety protocols to ensure responsible and ethical research practices.

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Conflict of interest

The Authors declare that there is no conflict of interest.

AI Using Declaration

Chat GPT was used for language and grammar corrections. The final output was read and modified by all authors.

Author's contributions

All authors equally contributed to preparing this article.

6. References

1. Sadrkhanloo M, Paskeh MDA, Hashemi M, Raesi R, Bahonar A, Nakhaee Z, et al. New emerging targets in osteosarcoma therapy: PTEN and PI3K/Akt crosstalk in carcinogenesis. Pathology-Research and Practice. 2023;251:154902. (DOI: 10.1016/j.prp.2023.154902)
2. Campbell KJ, Tait SW. Targeting BCL-2 regulated apoptosis in cancer. Open biology. 2018;8(5):180002. (DOI: 10.1098/rsob.180002)
3. Hashemi M, Khosroshahi EM, Chegini MK, Abedi M, Matinahmadi A, Hosnarody YSD, et al. miRNAs and exosomal miRNAs in lung cancer: New emerging players in tumor progression and therapy response. Pathology-Research and Practice. 2023;251:154906. (DOI: 10.1016/j.prp.2023.154906)
4. Mansoury F, Babaei N, Abdi S, Entezari M, Doosti A. Extremely low frequency magnetic fields induce mTOR and Hsa_Circ_100338 expression changes in gastric cancer and normal fibroblast cell lines. Cell Journal (Yakhteh). 2022;24(7):364. (DOI: 10.22074/cellj.2022.7922)
5. Sadrkhanloo M, Entezari M, Rashidi M, Hashemi M, Raesi R, Saghari S, et al. Non-coding RNAs in EMT regulation: association with tumor progression and therapy response. European Journal of Pharmacology. 2022;932:175212. (DOI: 10.1016/j.ejphar.2022.175212)
6. Ding Z-S, He Y-H, Deng Y-S, Peng P-X, Wang J-F, Chen X, et al. MicroRNA-34a inhibits bladder cancer cell migration and invasion, and upregulates PTEN expression. Oncology letters. 2019;18(5):5549-54. (DOI: 10.3892/ol.2019.10877)
7. Chen D, Guo Y, Chen Y, Guo Q, Chen J, Li Y, et al. LncRNA growth arrest-specific transcript 5 targets miR-21 gene and regulates bladder cancer cell proliferation and apoptosis through PTEN. Cancer medicine. 2020;9(8):2846-58. (DOI: 10.1002/cam4.2664)
8. Gan L, Zheng L, Zou J, Luo P, Chen T, Zou J, et al. MicroRNA-21 in urologic cancers: from molecular mechanisms to clinical implications. Frontiers in Cell and Developmental Biology. 2024;12:1437951. (DOI: 10.3389/fcell.2024.1437951)
9. Ghafouri-Fard S, Abak A, Shoorei H, Mohaqiq M, Majidpoor J, Sayad A, et al. Regulatory role of microRNAs on PTEN signaling. Biomedicine & Pharmacotherapy. 2021;133:110986.(DOI: 10.1016/j.biopha.2020.110986)
10.Wang H, Luo W, Wang X, Xue D, Ren L, Xu L, et al. Testicular nuclear receptor 4 regulates proliferation and apoptosis of bladder cancer via Bcl-2. Frontiers in Molecular Biosciences. 2021;8:670409. (DOI: 10.3389/fmolb.2021.670409)
11.Abdi S, Dorranian D, Naderi GA, Razavi AE. Changes in physicochemical charachteristics of human low density lipoprotein nanoparticles by electromagnetic field exposure. Stud U Babes-Bol Che. 2016;61(1):185-97.
12.Abdi S, Dorranian D, Razavi AE, Naderi GA, Boshtam M, Ghorannevis M. Evaluation of the effects of weak and moderate static magnetic fields on the characteristics of human low density lipoprotein in vitro. Bioelectromagnetics. 2013;34(5):397-404. (DOI: 10.1002/bem.21779)
13.Bahar M, Majd A, Abdi S. Effects of (ELF) extremely low frequency (50 Hz) AC and DC magnetic fields on lentil germination and seedlings growth. 2009.
14.Raesi R, Haji Ghasem Kashani M. In Vitro Study of the Osterix and Osteopontin Genes Expression in Conditioned Medium-and Pulsed Electromagnetic Field-induced Bone Marrow Mesenchymal Stem Cells. The Open Public Health Journal. 2024;17(1). (DOI: 10.2174/0118749445351568241211161854)
15.Sandberg M, Whitman W, Bissette R, Ross C, Tsivian M, Walker SJ. Pulsed Electromagnetic Field Therapy Alters the Genomic Profile of Bladder Cancer Cell Line HT-1197. Journal of Personalized Medicine. 2025;15(4):143. DOI: 10.3390/jpm15040143
16.Bahari A, Baharara J, Nejad Shahrokhabadi K, Lotfi M. Investigating the expression changes of several key genes in prostate cancer cells under exposure to the ELF pulsed electromagnetic fields. Scientific Reports. 2025;15(1):37394. (DOI: 10.1038/s41598-025-20910-0)
17.Consales C, Cirotti C, Filomeni G, Panatta M, Butera A, Merla C, et al. Fifty-hertz magnetic field affects the epigenetic modulation of the miR-34b/c in neuronal cells. Molecular neurobiology. 2018;55(7):5698-714. (DOI: 10.1007/s12035-017-0791-0)
18.Siasi E, Moniri E. The effect of extremely low frequency electromagnetic fields following on upregulation of miR-21 and miR-29 in gastric cancer cell line. Gastroenterology and Hepatology From Bed to Bench. 2021;14(1):67. (PMID)
19.Knowles TP, Vendruscolo M, Dobson CM. The amyloid state and its association with protein misfolding diseases. Nature reviews Molecular cell biology. 2014;15(6):384-96. (DOI: 10.1038/nrm3810)
20.Wydorski PJ, Zmijewska A, Franczak A. The Extremely-Low-Frequency Electromagnetic Field Affects Apoptosis and Oxidative-Stress-Related Genes and Proteins in the Porcine Endometrium—An In Vitro Study. International Journal of Molecular Sciences. 2024;25(13):6931. (DOI:10.3390/ijms25136931)

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21.Li Y, Héroux P. Extra-low-frequency magnetic fields alter cancer cells through metabolic restriction. Electromagnetic biology and medicine. 2014;33(4):264-75. (DOI: 10.3109/15368378.2013.817334)
22.Seton-Rogers S. Blocking the gatekeeper. Nature Reviews Cancer. 2014;14(12):766-. (DOI:10.1016/j.bbabio.2017.02.001)
23.Aziz MH, Shen H, Maki CG. Acquisition of p53 mutations in response to the non-genotoxic p53 activator Nutlin-3. Oncogene. 2011;30(46):4678-86. (DOI: 10.1038/onc.2011.185)
24.Matthews SB, Fettig LM, Ward AV, Jackman MR, MacLean PS, Sartorius CA. Progestins target mitochondrial respiration to shift metabolic phenotype in luminal breast cancer. Cancer Research. 2019;79(13_Supplement):796-. (DOI:10.1158/1538-7445.AM2019-796)
25.Heidari S, Abdi S, Karizi SZ. Evaluation of BCL2 and its regulatory miRs, miR-15-b and miR-16 expression changes under the exposure of extremely low-frequency electromagnetic fields on human gastric cancer cell line. Radiation protection dosimetry. 2021;197(2):93-100.(DOI: 10.1093/rpd/ncab163)
26.Blank M, Goodman R. Electromagnetic initiation of transcription at specific DNA sites. Journal of Cellular Biochemistry. 2001;81(4):689-92. (DOI: 10.1002/jcb.1102)
27.Pall ML. Electromagnetic fields act via activation of voltage-gated calcium channels to produce beneficial or adverse effects. Journal of cellular and molecular medicine. 2013;17(8):958-65. (DOI: 10.1111/jcmm.12088)
28.Aalami Zavareh F, Abdi S, Entezari M. Up-regulation of miR-144 and miR-375 in the human gastric cancer cell line following the exposure to extremely low-frequency electromagnetic fields. International Journal of Radiation Biology. 2021;97(9):1324-32. (DOI: 10.1080/09553002.2021.1941376)
29.Mansoury F, Babaei N, Abdi S, Entezari M, Doosti A. Changes in NOTCH1 gene and its regulatory circRNA, hsa_circ_0005986 expression pattern in human gastric adenocarcinoma and human normal fibroblast cell line following the exposure to extremely low frequency magnetic field. Electromagnetic Biology and Medicine. 2021;40(3):375-83. (DOI: 10.1080/15368378.2021.1891092)
30.Liu Y, Liu W-b, Liu K-j, Ao L, Cao J, Zhong JL, et al. Extremely low-frequency electromagnetic fields affect the miRNA-mediated regulation of signaling pathways in the GC-2 cell line. PloS one. 2015;10(10):e0139949. (DOI: 10.1371/journal.pone.0139949)
31.Brabant C, Honvo G, Demonceau C, Tirelli E, Leonard F, Bruyere O. Effects of extremely low frequency magnetic fields on animal cancer and DNA damage: a systematic review and meta-analysis. Progress in biophysics and molecular biology. 2025;195:137-56. (DOI: 10.1016/j.pbiomolbio.2024.12.005)
32.Lai H, Levitt BB. Cellular and molecular effects of non-ionizing electromagnetic fields. Reviews on Environmental Health. 2024;39(3):519-29. (DOI: 10.1515/reveh-2023-0023)
33.Tota M, Jonderko L, Witek J, Novickij V, Kulbacka J. Cellular and molecular effects of magnetic fields. International Journal of Molecular Sciences. 2024;25(16):8973. (DOI: 10.3390/ijms25168973)

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