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Original Article

Comparative Evaluation of the Anticancer Efficacy of Fluoroquinolone Antibiotics Across Diverse Cancer Cell Lines

Durgesh Soni[1,2], Mohd Hasan Mujahid[1,3], Vijay Jagdish Upadhye[1,*]

[1]Department of Life Science, Parul Institute of Applied Sciences and Research and Development Cell, Parul University, Vadodara, Gujarat, INDIA.
[2]Department of Pharmacology, Sun Pharma Advanced Research Co. Ltd., Savli G.I.D.C. Estate, Savli, Vadodara, Gujarat, INDIA.
[3]Department of Polymer and Process Engineering, IIT Roorkee, Roorkee, Uttarakhand, INDIA.

ABSTRACT

Background: Cancer treatment faces significant challenges due to limited success with conventional therapies. Drug repurposing-identifying new applications for existing drugs—offers a promising strategy to expand therapeutic options. This study aims to evaluate and compare the*in vitro* anticancer potential of three fluoroquinolone antibiotics (ciprofloxacin, moxifloxacin, and levofloxacin) across multiple human cancer cell lines, marking the first comparative analysis of their efficacy. **Materials and Methods:** Cytotoxicity assays were employed to assess the antiproliferative effects of ciprofloxacin, moxifloxacin, and levofloxacin on triple-negative breast cancer (MDA-MB-231), Prostate Cancer (PC3), Glioblastoma (U87), and Ovarian Cancer (SCOV3) cell lines. The experimental design focused on quantifying dose-dependent inhibition of cancer cell proliferation. **Results:** All three fluoroquinolones demonstrated anticancer activity, but moxifloxacin exhibited superior antiproliferative effects compared to ciprofloxacin and levofloxacin across all tested cell lines. Dose-response analyses revealed consistent inhibitory patterns, with moxifloxacin achieving the highest reduction in cancer cell viability. **Conclusion:** This study highlights the potential of fluoroquinolones, particularly moxifloxacin, as adjunctive agents in cancer therapy. The findings underscore the value of drug repurposing for oncology and warrant further investigations into synergistic effects with standard chemotherapeutic regimens. Future studies should prioritize*in vivo* validation and combinatorial efficacy assessments to optimize clinical translation.

Keywords: Antibiotics, Fluoroquinolones, Antiproliferative, Breast cancer, Prostate cancer.

Correspondence:
Prof. Dr. Vijay Jagdish Upadhye
Professor, Department of Life Science, Parul Institute of Applied Sciences (PIAS), Parul University (NAAC A++ Accredited), Limda, Waghodia-391760, Vadodara, Gujarat, INDIA.
Email: dr.vijaysemilo@gmail.com; vijay.upadhye35296@paruluniversity.ac.in

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INTRODUCTION

Cancer is a complex disease characterized by the abnormal and uncontrolled growth of cells, including the potential to invade and/or spread to other body tissues through the process called metastasis. Widespread metastasis is the primary cause of death from cancer. The latest cancer statistics provided by the American Cancer Society estimate that, in 2023, 1.958 million new cancer cases and 6,09,820 cancer deaths are projected to occur in the United States.[1] The number of cancer cases has been steadily increasing over the years due to factors such as aging populations, lifestyle changes, and environmental exposures. Cancer can cause significant morbidity and mortality, leading to physical, emotional, and economic burdens for individuals and society. The high prevalence of cancer underscores the urgent need for effective treatment options for improving patient outcomes, reducing cancer-related deaths, and enhancing the quality of life for cancer patients.[2]

Drug repurposing, drug repositioning, drug reprofiling, therapeutic switching, or indication switching of currently approved drugs, rely on evaluating the effectiveness of known targets and reliable biomarkers against other illnesses.[3] In the last decades, our understanding of the molecular and genetic bases of diseases has increased tremendously, and it is now acknowledged that few diseases share a molecular mechanism of pathogenesis. In this respect, the use of the same drug for more than one disease is also conceivable. The most appropriate approach to end the search for newer drugs is "Repositioning", as it requires less time and cost to explore new indications of existing drugs. In the past, several drugs have been repositioned for different indications, but the full potential remains unharnessed. While delivering innovation (new treatments that resolve unaddressed

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health needs), repurposing also offers several advantages over de novo development, such as lower costs of development, lesser risk of failure, and reduced time frame to registration due to the availability of clinical safety data. With the rise in cancer prevalence and treatment costs, it is imperative to search for newer drugs, and the use of a repositioning approach may help.[4] Fluoroquinolones (FQs) have been used as antibiotics for over four decades now. The research highlighted their use as pharmacological compounds with multifaceted implications.[5-7] Repositioning of fluoroquinolones as anti-cancer molecules seems to be a highly plausible option owing to their profound immunomodulatory, pro-apoptotic, anti-proliferative, and anti-metastatic potential.[8,9] The concept of anticancer FQs gained strong momentum with the emergence of scientific reports describing the cytotoxic properties and apoptotic effects of the clinically approved FQs for their antibiotic action.[10,11]

Fluoroquinolones are broad-spectrum antibiotic agents that inhibit the activity of bacterial gyrase and topoisomerase II leading to a malfunction of DNA synthesis. Fluoroquinolone antibiotics like Enoxacin, Norfloxacin, Ciprofloxacin, Moxifloxacin, and Levofloxacin induced morphological alterations, apoptotic cell death, inhibited cellular proliferation, increased population doubling time, and reduced saturation density in cancer cell lines.[12] Fluoroquinolones exert their anticancer effects through various mechanisms, including the inhibition of eukaryotic topoisomerases, induction of DNA damage, and disruption of critical cell cycle processes. Preclinical studies have demonstrated their efficacy in reducing tumor cell viability, inducing apoptosis, and sensitizing cancer cells to other chemotherapeutic agents.[13] Fluoroquinolones may offer a new strategy for cancer treatment by targeting multiple pathways and mechanisms involved in tumor development and progression.

The current study aims to explore the anticancer potential of fluoroquinolones of different generations against a diverse range of human cancer cell lines. This study is the first to systematically compare the effects of different fluoroquinolones (ciprofloxacin, moxifloxacin, levofloxacin) on multiple cancer cell types. Cell growth inhibition was used as a marker for the anticancer potential of fluroquinolones. The potential of fluoroquinolones as adjuncts to existing cancer treatments, paving the way for further preclinical and clinical evaluations.

MATERIALS AND METHODS

In vitro animal cell culture

Four human cancer cell lines, MDA-MB-231 (Triple Negative Breast Cancer), U87 (Human Glioblastoma), PC3 (Human Prostate Cancer), and SKOV3 (Human Ovarian Cancer) were used for the assessment of antiproliferative activity of fluoroquinolones. MDA-MB 231, U87 and PC3 cell lines were propagated at 37ºC with 95% humidity and 5% CO_2 in DMEM culture media with glutamine (2 mM), HEPES (25 mM), penicillin (1000 IU/mL), streptomycin (1000 µg/mL; Bio Whittaker, Biggs Ford, Md), and 10% fetal calf serum. The culture media used for SCOV3 cells was Mc-Coy media, the remaining contents were similar.

MTT Assay

The cytotoxicity assay was performed as previously by Mujahid *et al.*, with slight modification.[14] Approximately, five thousand ($5x10^3$) cells/well were seeded in a 96-well plate and were incubated for 48 hr, before drug exposures. During drug treatment procedures, it is a general practice that after cell seeding, cells should be kept for a few days to avoid post-trypsinization mechano-physiological stress and gets acclimatized itself for its proliferative property under the new environment. Post-drug treatment, to get drug-mediated effects, it is again a standard protocol to observe for a sufficient time frame where molecular changes can occur because of the drug exposures. Hence, the cells were incubated with compounds for 48 hr post-drug treatment, 5 mg/mL MTT reagent was added for 4 hr at 37ºC. The MTT solution was removed and 100 µL of isopropanol with 4 mM HCl, was added to each well to dissolve the formazan crystals by gentle shaking for 20 min and measured at 570 nm using a Biotek model EL340 plate reader using Kineticalc software. The MTT assay was used to assess the percentage of cells alive relative to the untreated control. This assay is based on the ability of mitochondrial enzymes of living cells to convert a yellow MTT tetrazolium salt into a blue MTT formazan and has a high degree of precision. It is widely used and is reliable in assaying the cytotoxic effects of anticancer drugs. Cell viability (%) was determined by using the following formula:

$$\text{Cell Viability \%} = [\text{OD (Treated)} / \text{OD (Control)}] * 100$$

OD = Optical Density.

Statistical analysis

GraphPad Prism (GraphPad Software, San Diego, CA, USA) was used for statistical analysis and plotting graphs. Values are expressed as Mean±S.E.M. A one-way study of variance (ANOVA), followed by post hoc Dunnett's test, was used to compare the differences between control and at different concentrations of test drugs. A p-value less than 0.05 ($p<0.05$) was considered statistically significant.

RESULTS

Cytotoxicity assessment through MTT assay

The *in vitro* growth inhibition assay was conducted to evaluate the antiproliferative effects of selected fluoroquinolones on various cancer cell lines, including MDA-MB 231 (TNBC), U87 (GBM), PC3 (Prostate Carcinoma) and SCOV3 (Ovarian Carcinoma). Cell viability was assessed using the MTT assay, which measures the metabolic activity of viable cells.

Effect of Ciprofloxacin on cancer cell growth

The antiproliferative effect of ciprofloxacin at concentrations of 62.5 µM to 1000 µM against TNBC (MDA-MB-231), Prostate (PC3), Glioblastoma (U87), and Ovarian (SCOV3) cell lines in MTT assay is presented in Table 1 and Figures 1-4.

Effect of Moxifloxacin on cancer cell growth

The antiproliferative effect of Moxifloxacin at concentrations of 62.5 µM to 1000 µM against TNBC (MDA-MB-231), Prostate (PC3), Glioblastoma (U87), and Ovarian (SCOV3) cell lines in MTT assay is presented in Table 2 and Figures 5-8.

Effect of Levofloxacin on Cancer Cell Growth

The antiproliferative effect of levofloxacin at concentrations of 62.5 µM to 1000 µM against TNBC (MDA-MB-231), Prostate (PC3), Glioblastoma (U87), and Ovarian (SCOV3) cell lines in MTT assay is presented in Table 3 and Figures 9-12.

Among three fluoroquinolones, moxifloxacin showed the best effect with 81% - 90% cell growth inhibition at 1000 µM in all four cell lines evaluated. At 500 µM concentration of moxifloxacin, 63%-72% cell growth inhibition was observed in MDA-MB-231, SCOV3 and PC3 cell lines. Data is presented in Table 2.

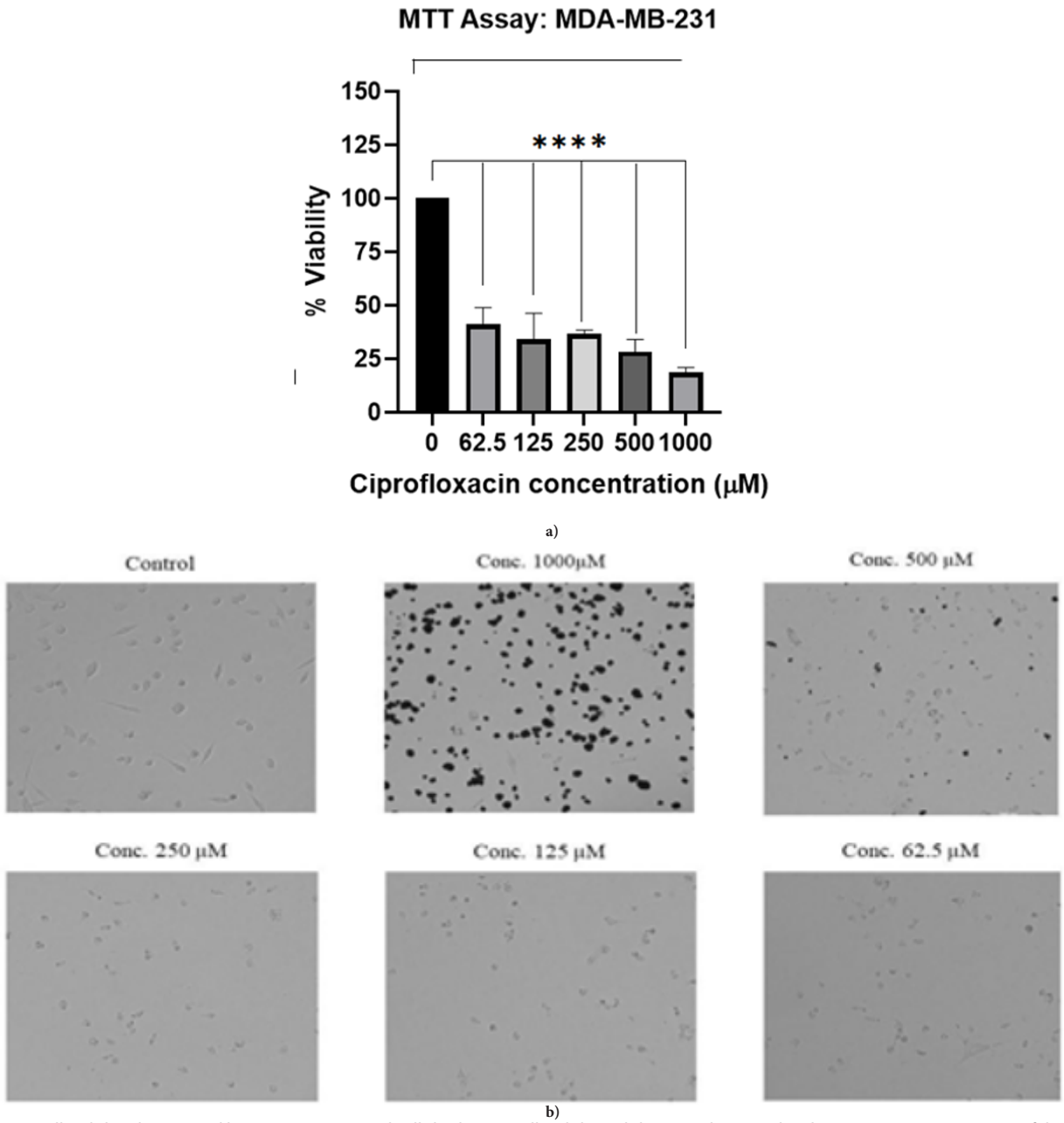
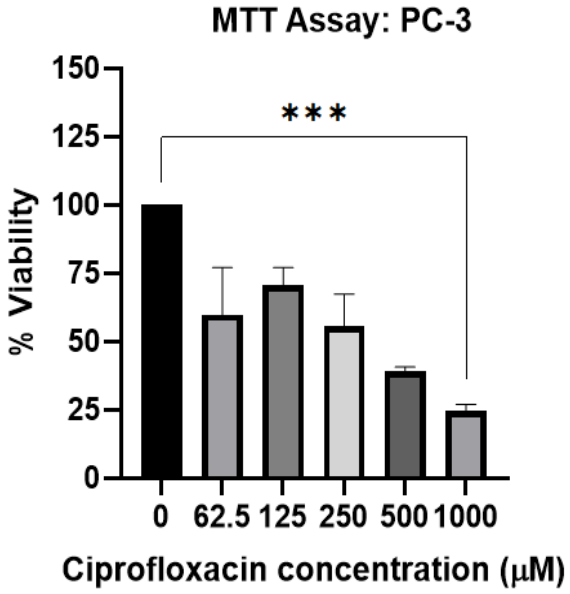


Figure 1: (a) Cell viability determined by MTT assay. Control cells had 100% cell viability, while it was decreased with increasing concentration of the ciprofloxacin against MDA-MB-231, a human TNBC (Triple Negative Breast Cancer) cell line. (b) Photomicrographs of MDA-MB-231 cells post ciprofloxacin treatment. Utilizing the statistical software program GraphPad Prism 8.0.2, a one-way ANOVA analysis was carried out. A significant p value <0.0001, p*** was found and data are presented as Mean±SEM with error bars represented.



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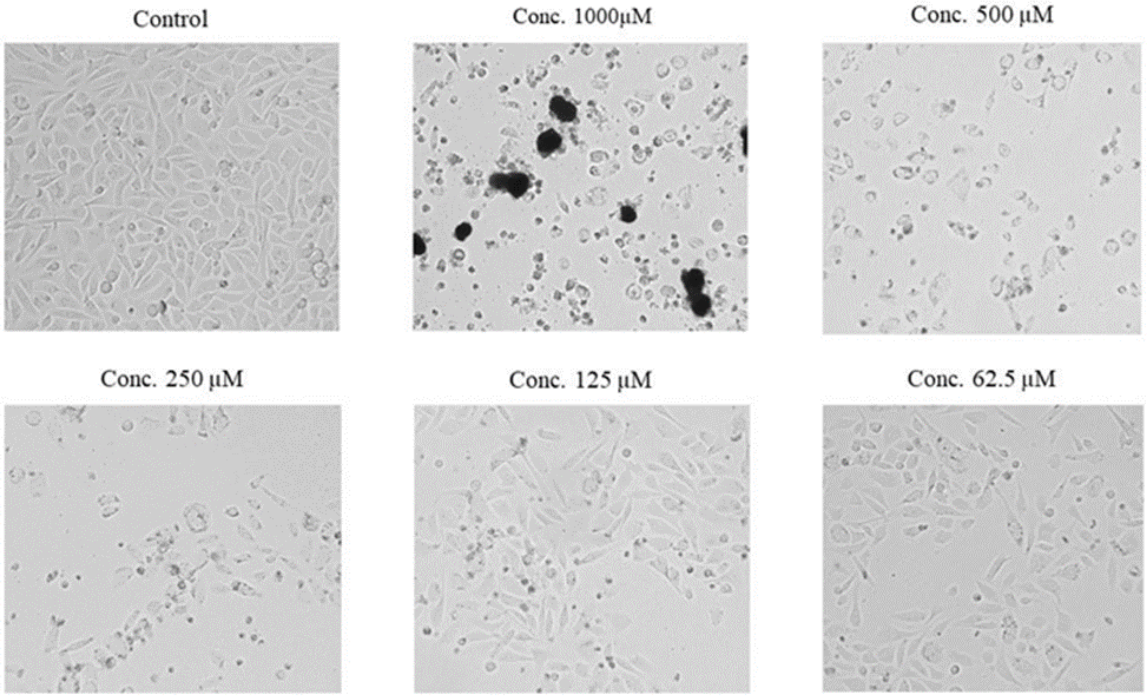
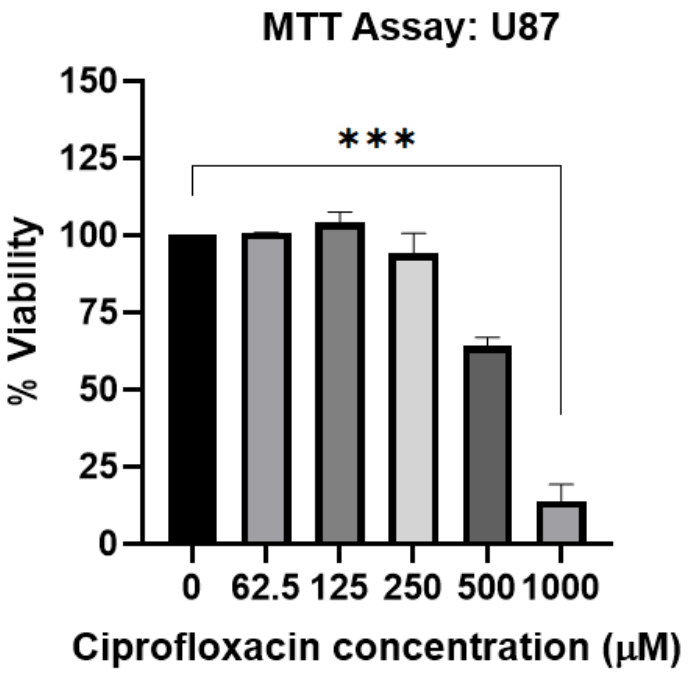


Figure 2: (a) Cell viability determined by MTT assay. Control cells showed 100% cell viability, while it was decreased with an increasing concentration of the ciprofloxacin against the PC3 cell line, a human prostate cancer cell line. (b) Photomicrographs of PC3 cells post ciprofloxacin treatment. Utilizing the statistical software program GraphPad Prism 8.0.2, a one-way ANOVA analysis was carried out. A significant *p* value <0.0001, *p**** was found and data are presented as Mean±SEM with error bars represented. a)



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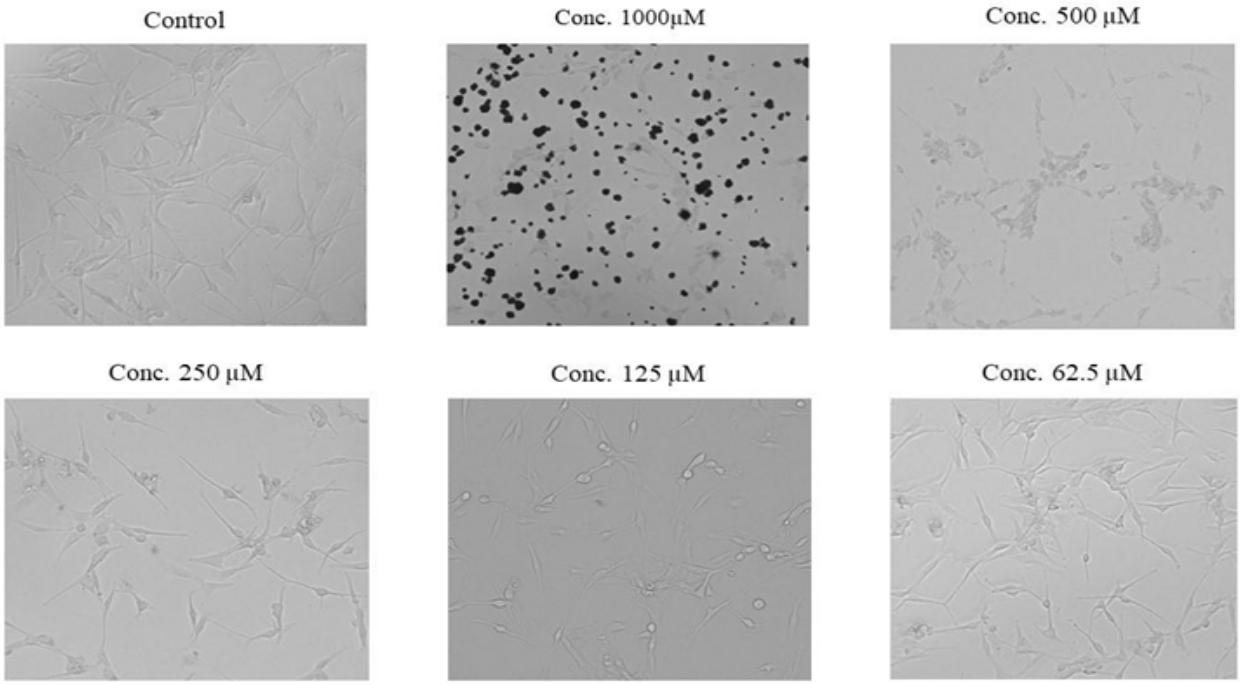

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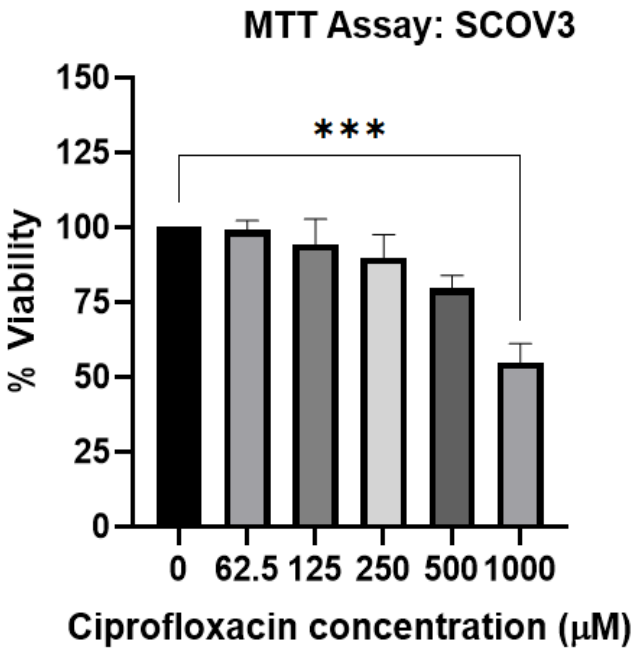
Figure 3: (a) Cell viability determined by MTT assay. Control cells showed 100% cell viability, while it was decreased with an increasing concentration of the ciprofloxacin against U87, a human glioblastoma cell line. (b) Photomicrographs of U87 cells post ciprofloxacin treatment. Utilizing the statistical software program GraphPad Prism 8.0.2, a one-way ANOVA analysis was carried out. A significant *p* value <0.0001, *p**** was found and data are presented as Mean±SEM with error bars represented. a)



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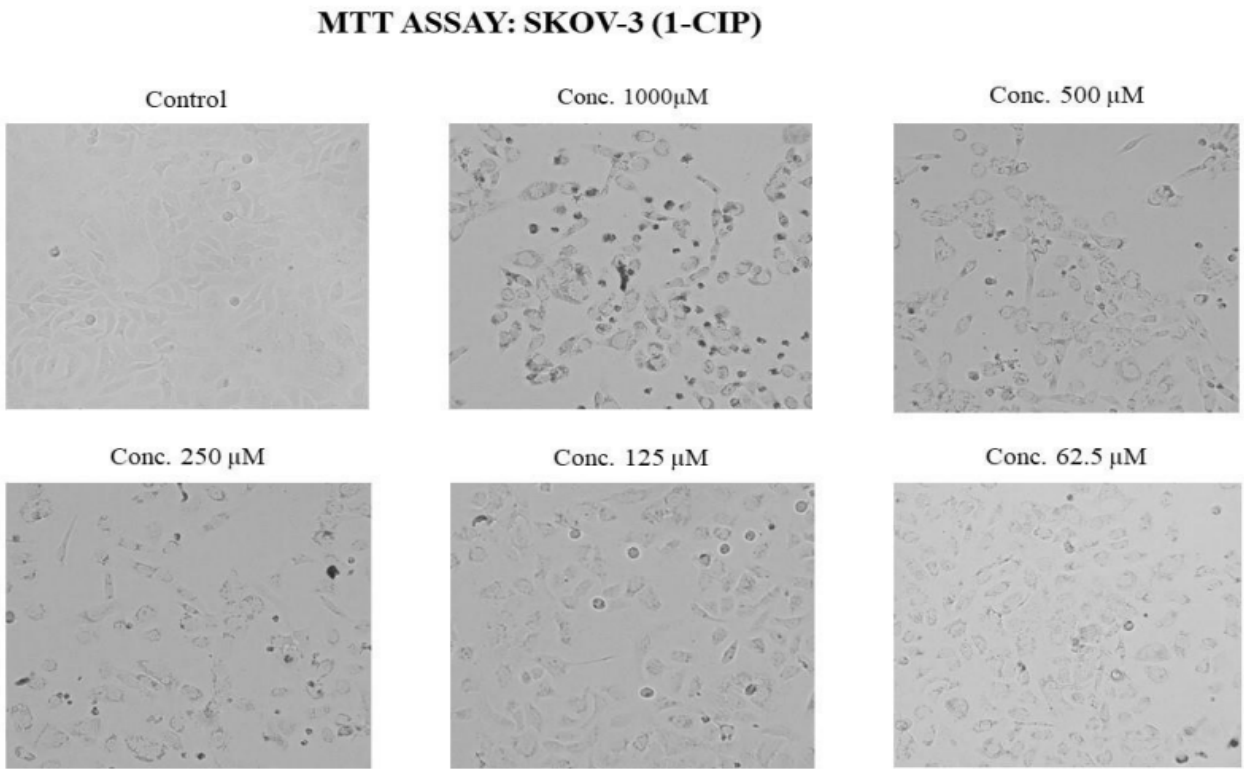

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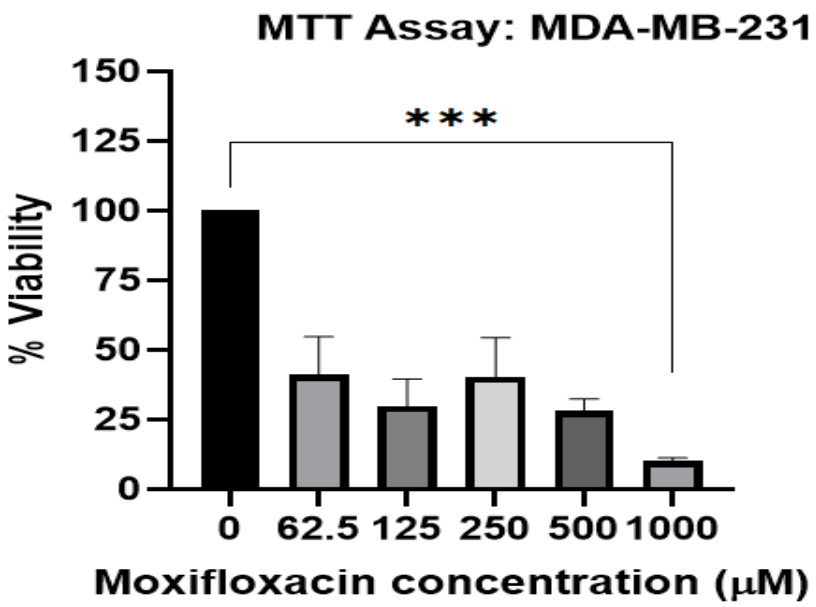
Figure 4: (a) Cell viability determined by MTT assay. Control cells showed 100% cell viability, while it was decreased with an increasing concentration of the ciprofloxacin against SCOV3, a human ovarian cancer cell line. (b) Photomicrographs of SCOV3 cells post ciprofloxacin treatment. Utilizing the statistical software program GraphPad Prism 8.0.2, a one-way ANOVA analysis was carried out. A significant p value <0.0001, p^{***} was found and data are presented as Mean±SEM with error bars represented. a)



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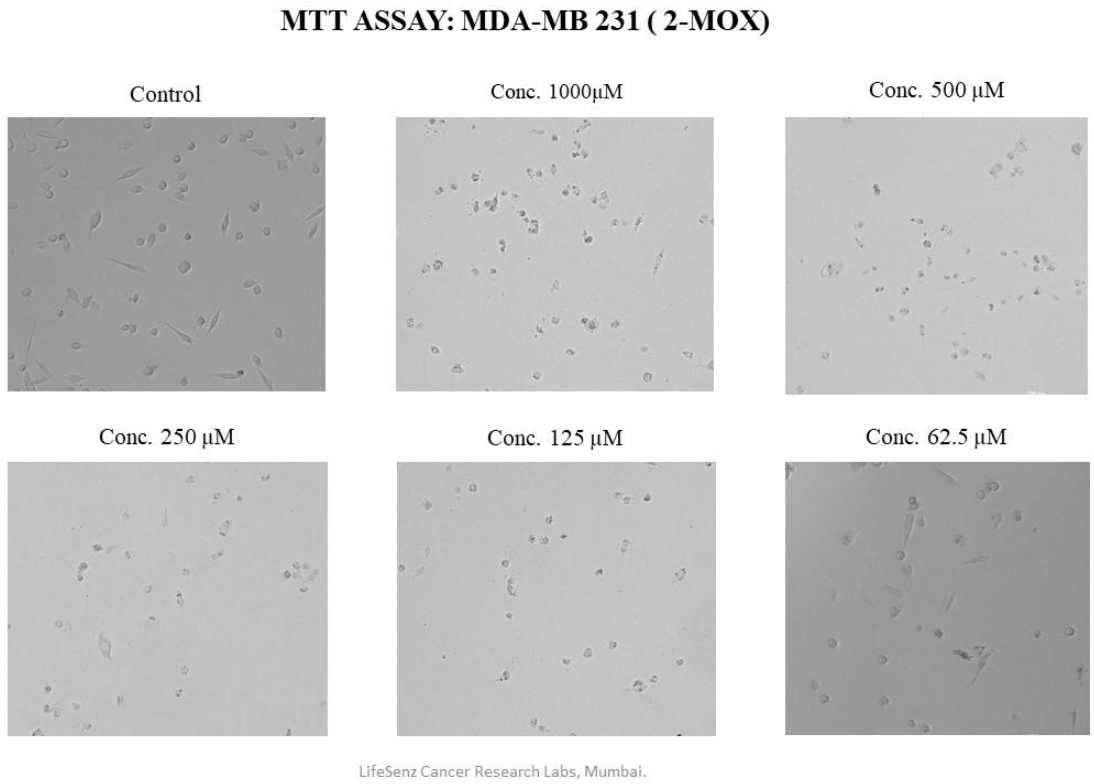

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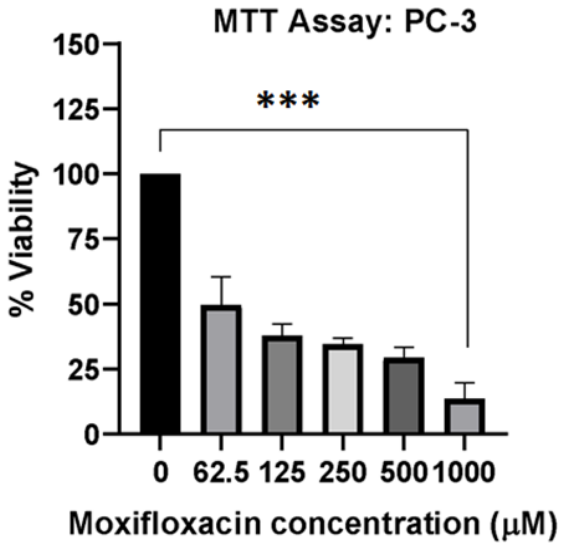
Figure 5: (a) Cell viability determined by MTT assay. Control cells had 100% cell viability, while it was decreased with an increasing concentration of moxifloxacin against MDA-MB-231, a human Triple-Negative Breast Cancer (TNBC) cell line. (b) Photomicrographs of MDA-MB-231 cells post moxifloxacin treatment. Utilizing the statistical software program GraphPad Prism 8.0.2, a one-way ANOVA analysis was carried out. A significant p value <0.0001, p^{***} was found and data are presented as Mean±SEM with error bars represented. a)



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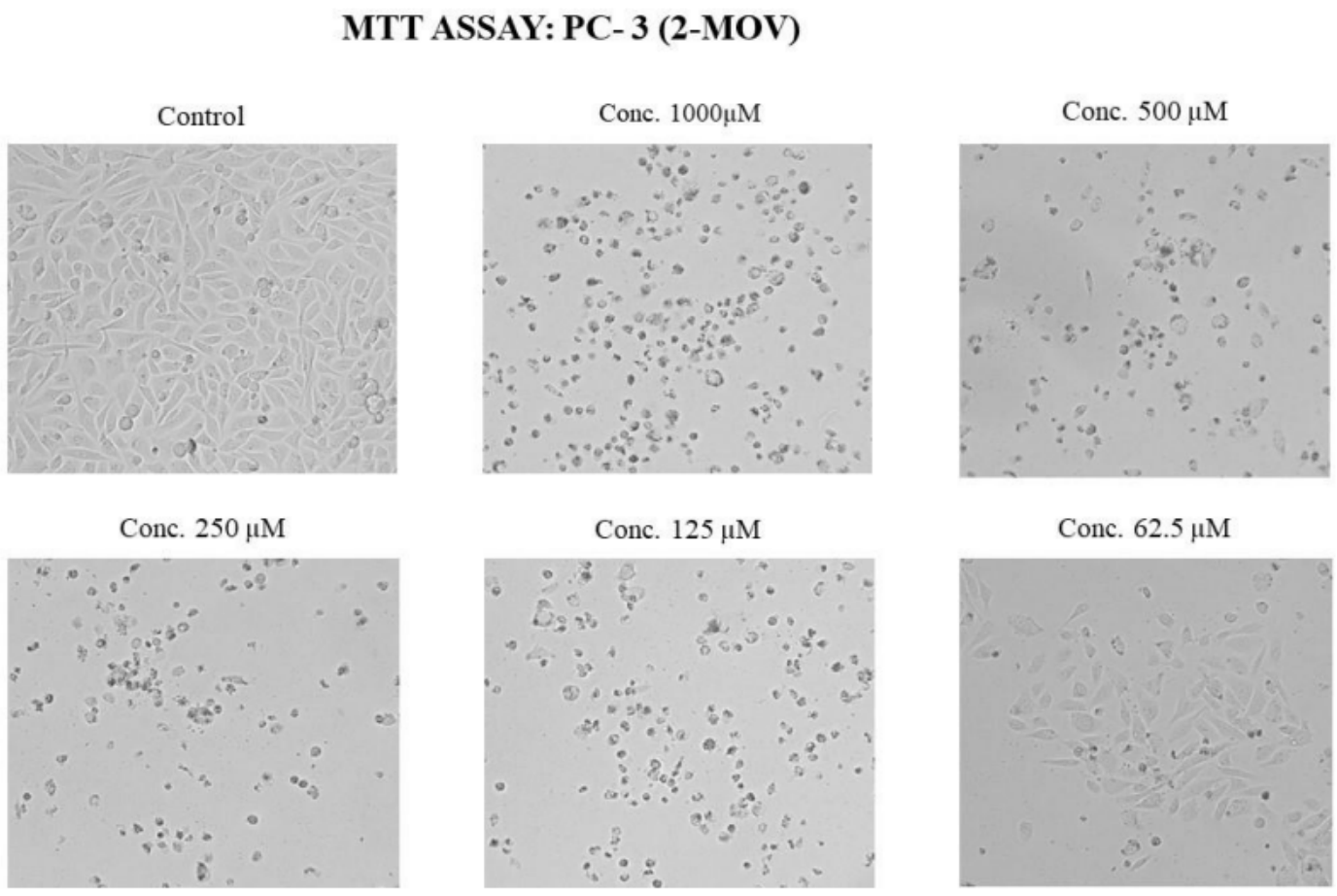

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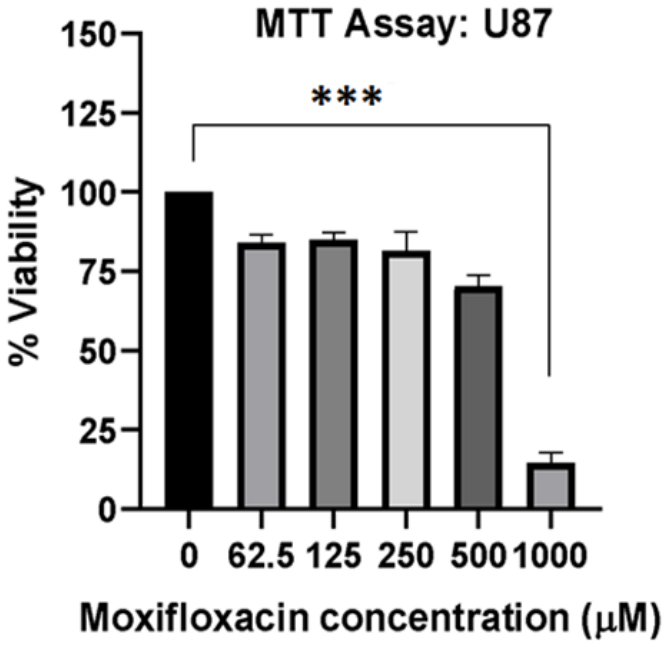
Figure 6: (a) Cell viability determined by MTT assay. Control cells had 100% cell viability, while it was decreased with an increasing concentration of moxifloxacin against PC3 cell line, a human prostate cancer cell line. (b) Photomicrographs of PC3 cells post moxifloxacin treatment. Utilizing the statistical software program GraphPad Prism 8.0.2, a one-way ANOVA analysis was carried out. A significant *p* value <0.0001, *p**** was found and data are presented as Mean±SEM with error bars represented. a)



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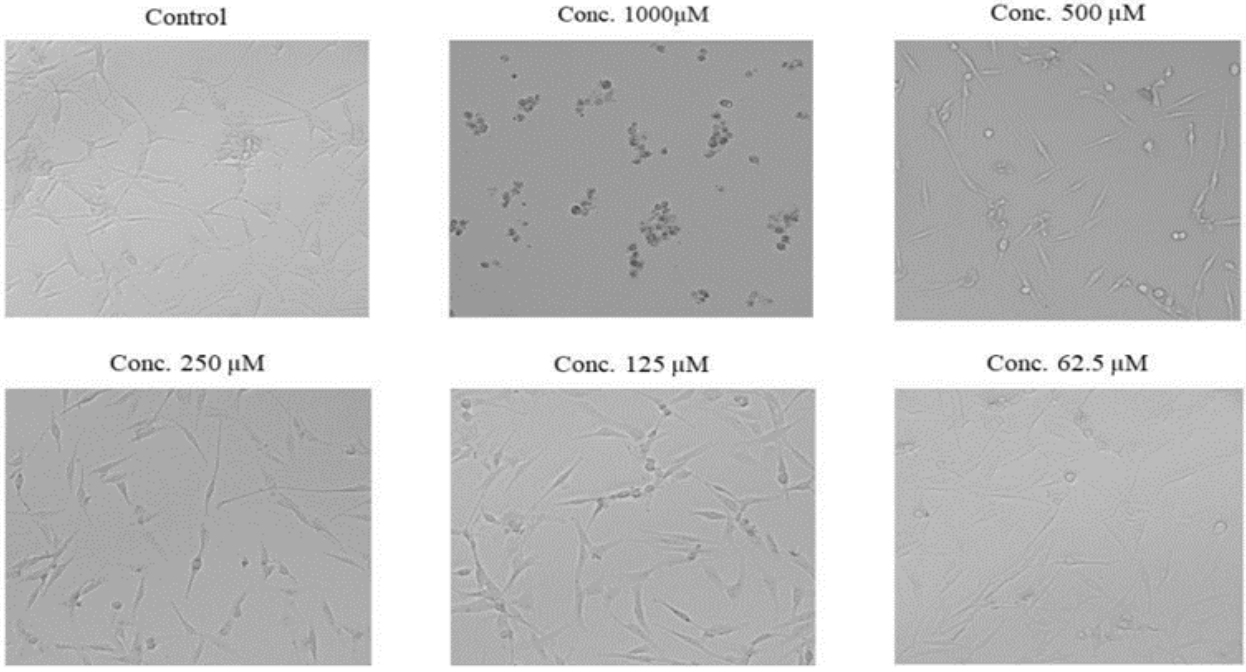

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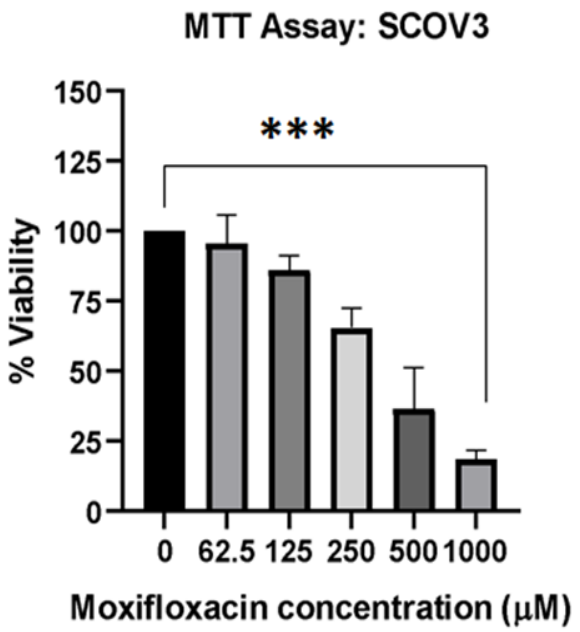
Figure 7: (a) Cell viability determined by MTT assay. Control cells showed 100% cell viability, while it was decreased with an increasing concentration of the moxifloxacin against U87, a human glioblastoma cell line. (b) Photomicrographs of U87 cells post moxifloxacin treatment. Utilizing the statistical software program GraphPad Prism 8.0.2, a one-way ANOVA analysis was carried out. A significant *p* value <0.0001, *p**** was found and data are presented as Mean±SEM with error bars represented. a)



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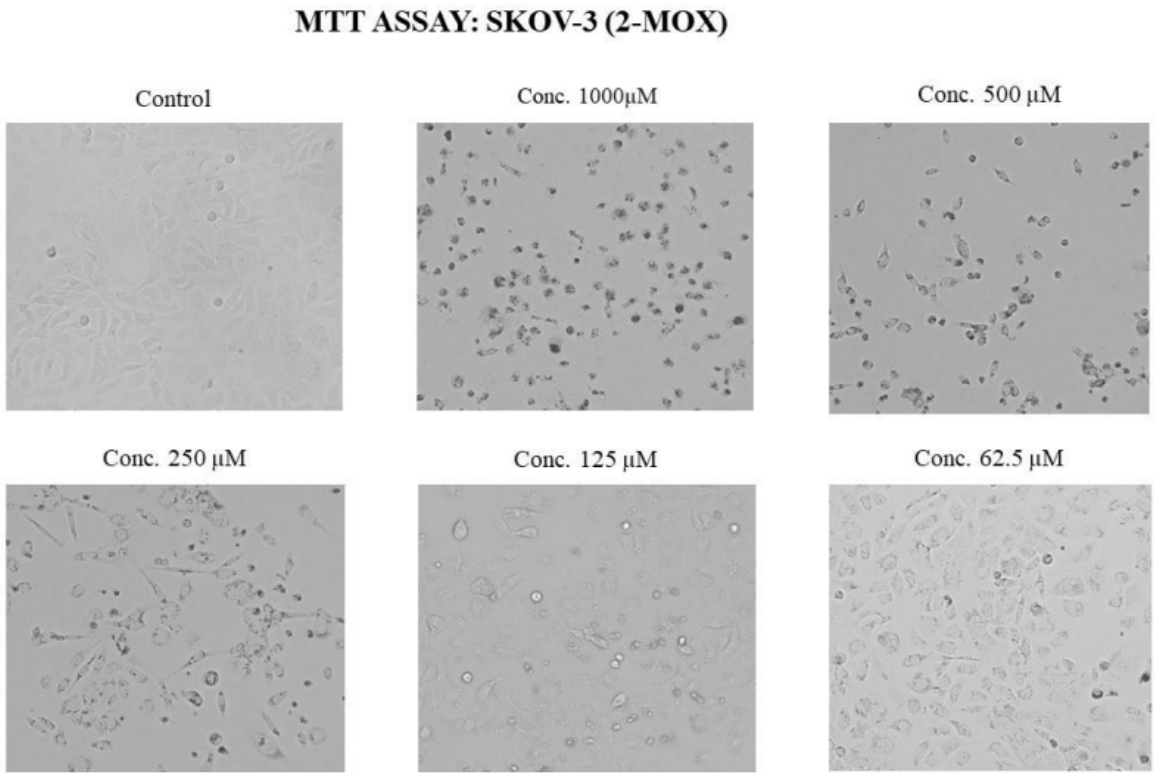

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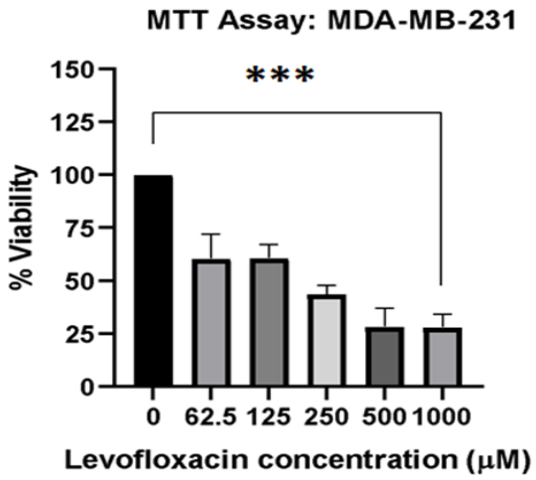
Figure 8: (a) Cell viability determined by MTT assay. Control cells showed 100% cell viability, while it was decreased with an increasing concentration of the moxifloxacin against SCOV3, a human ovarian cancer cell line. (b) Photomicrographs of SCOV3 cells post moxifloxacin treatment. Utilizing the statistical software program GraphPad Prism 8.0.2, a one-way ANOVA analysis was carried out. A significant *p* value <0.0001, *p**** was found and data are presented as Mean±SEM with error bars represented. a)



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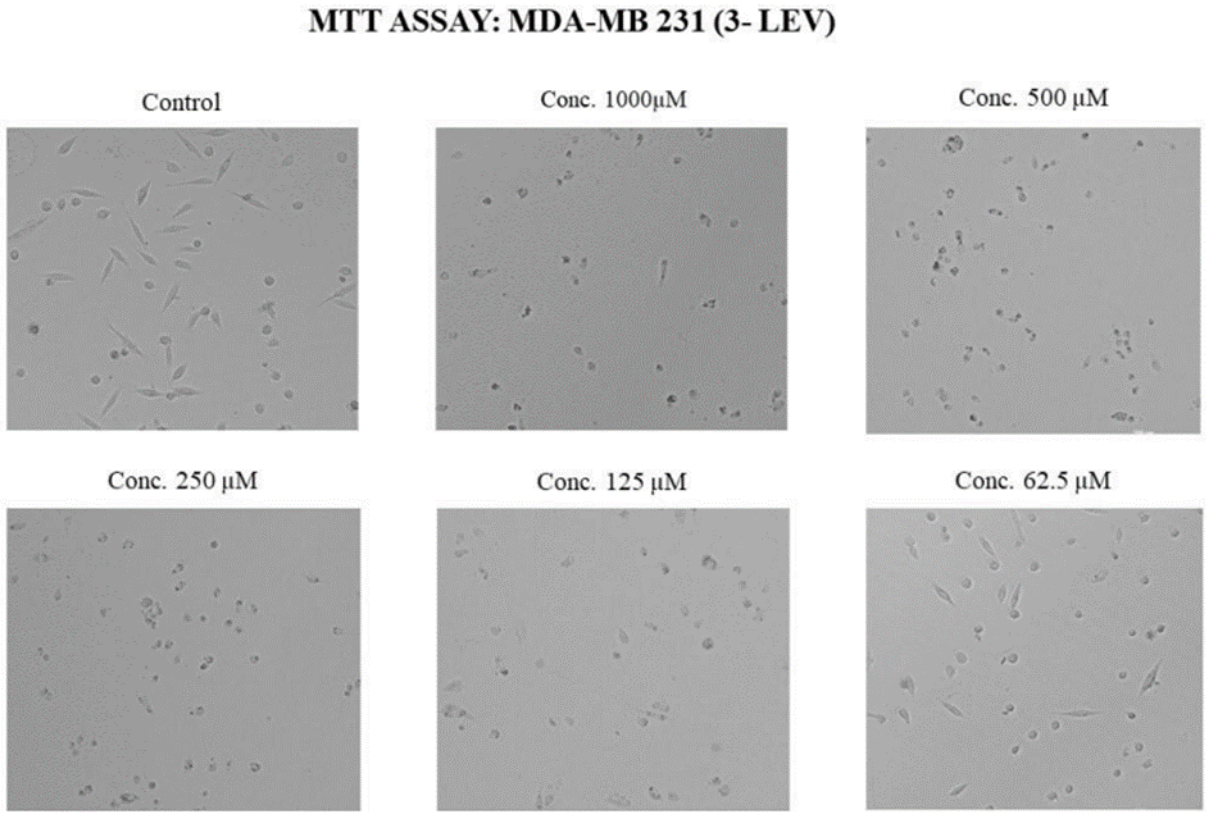

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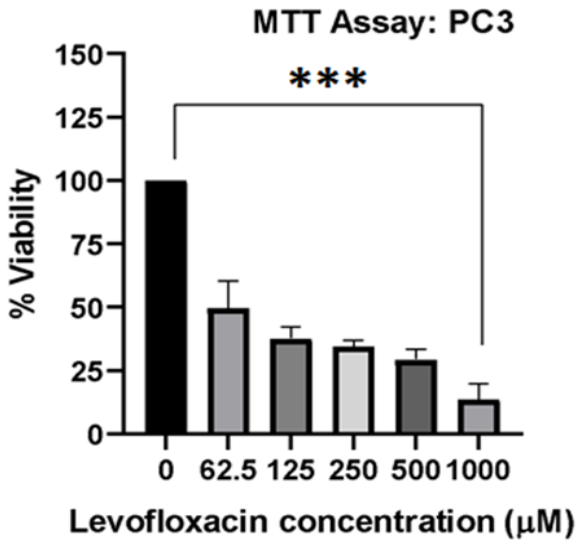
Figure 9: (a) Cell viability determined by MTT assay. Control cells had 100% cell viability, while it was decreased with an increasing concentration of levofloxacin against MDA-MB-231, a human Triple-Negative Breast Cancer (TNBC) cell line. (b) Photomicrographs of MDA-MB-231 cells post levofloxacin treatment. Utilizing the statistical software program GraphPad Prism 8.0.2, a one-way ANOVA analysis was carried out. A significant p value <0.0001, p*** was found and data are presented as Mean±SEM with error bars represented.



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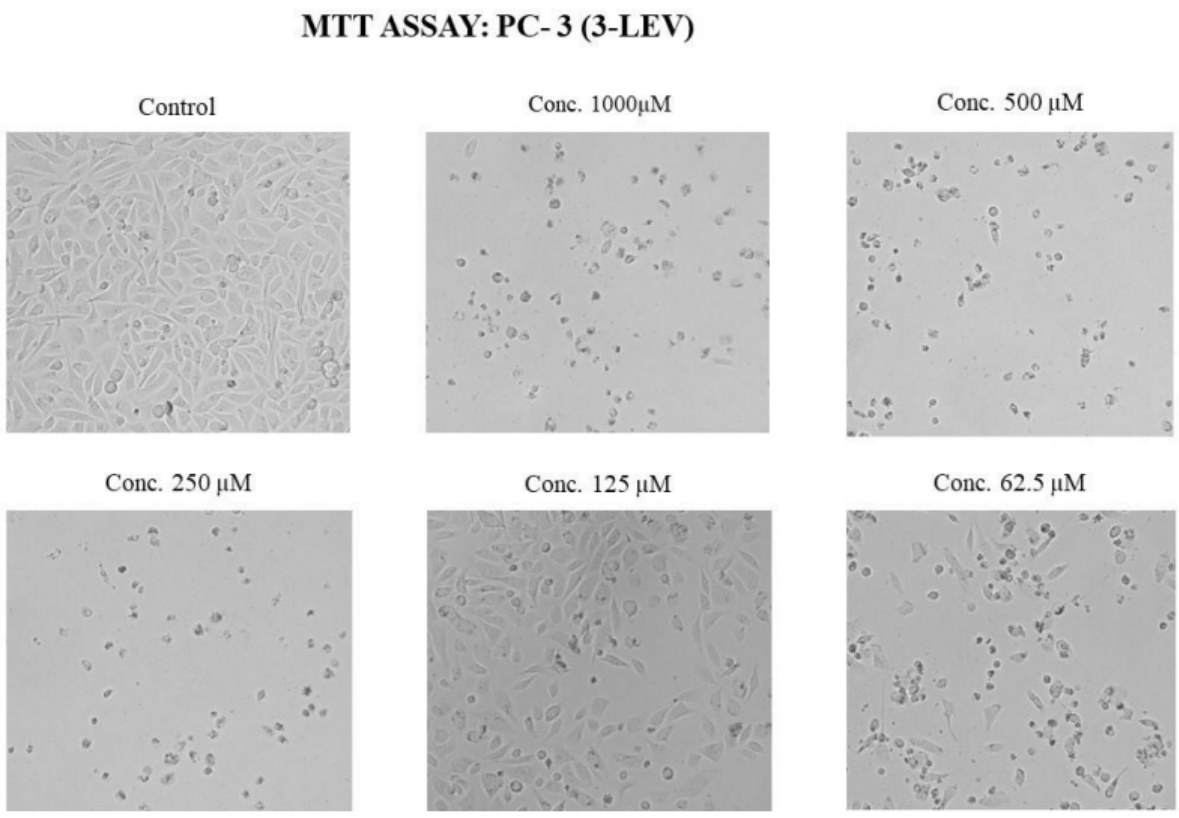

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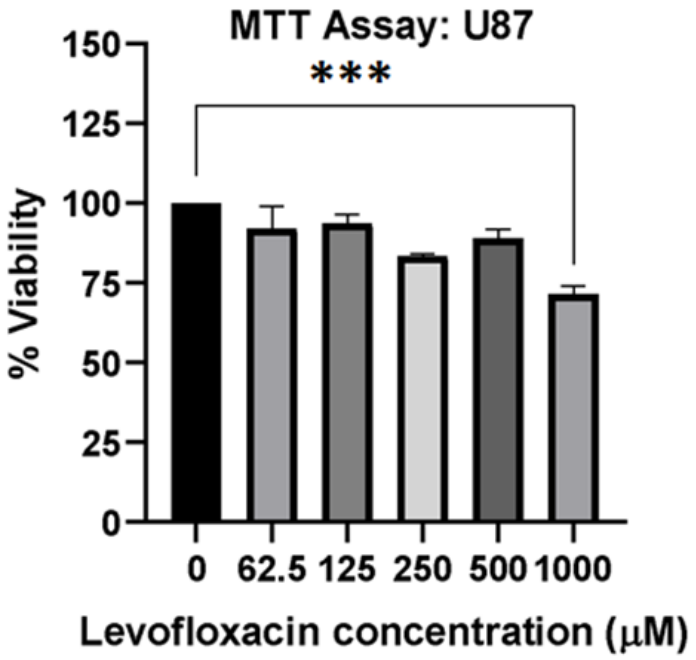
Figure 10: (a) Cell viability determined by MTT assay. Control cells showed 100% cell viability, while it was decreased with an increasing concentration of levofloxacin against the PC3 cell line, a human prostate cancer cell line. (b) Photomicrographs of PC3 cells post levofloxacin treatment. Utilizing the statistical software program GraphPad Prism 8.0.2, a one-way ANOVA analysis was carried out. A significant *p* value <0.0001, *p**** was found and data are presented as Mean±SEM with error bars represented. a)



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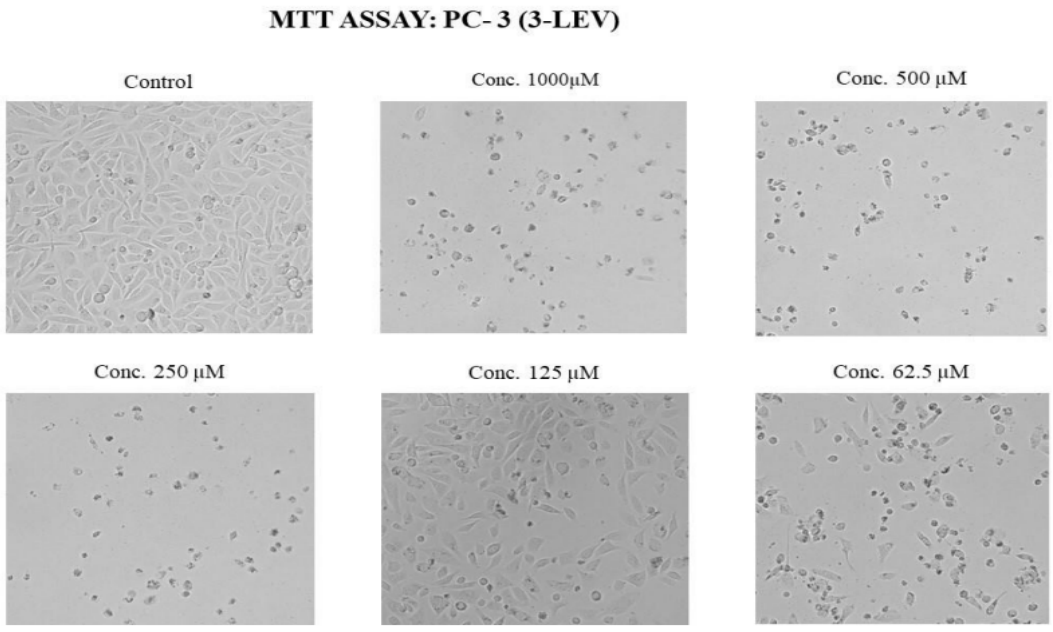

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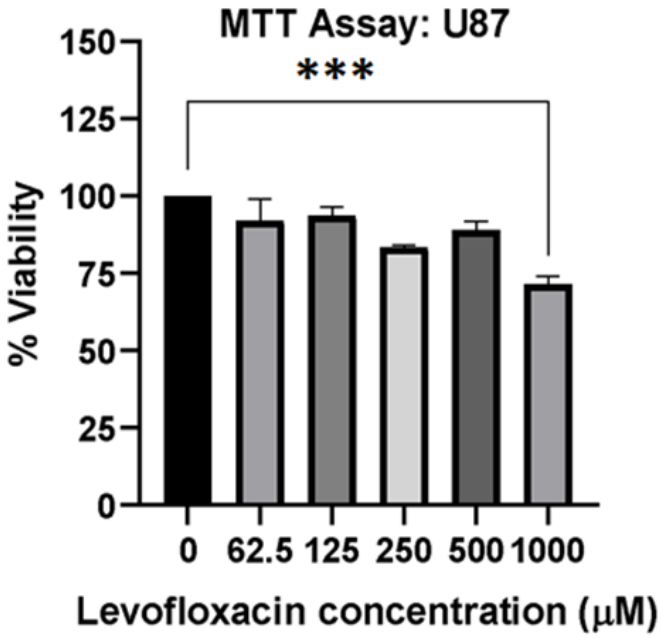
Figure 11: (a) Cell viability determined by MTT assay. Control cells showed 100% cell viability, while it was decreased with an increasing concentration of the levofloxacin against U87, a human glioblastoma cell line. (b) Photomicrographs of U87 cells post levofloxacin treatment. Utilizing the statistical software program GraphPad Prism 8.0.2, a one-way ANOVA analysis was carried out. A significant p value <0.0001, p*** was found and data are presented as Mean±SEM with error bars represented.



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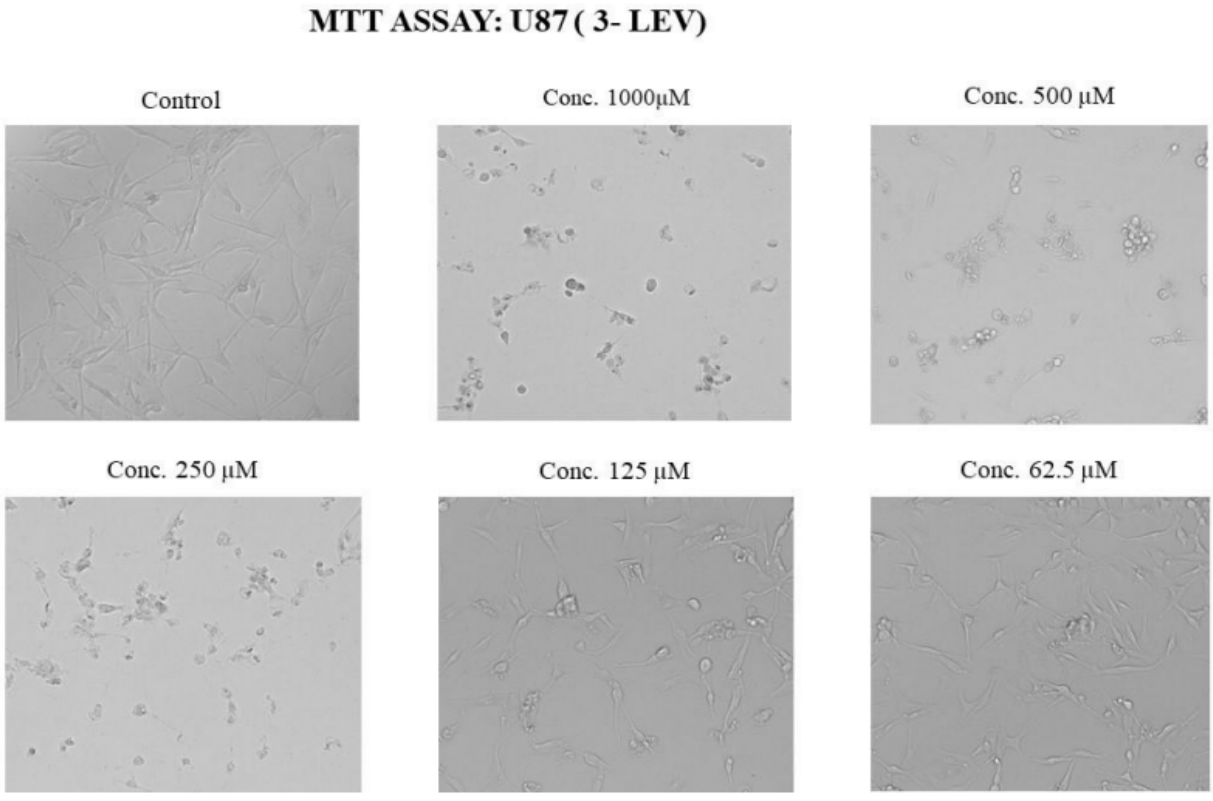

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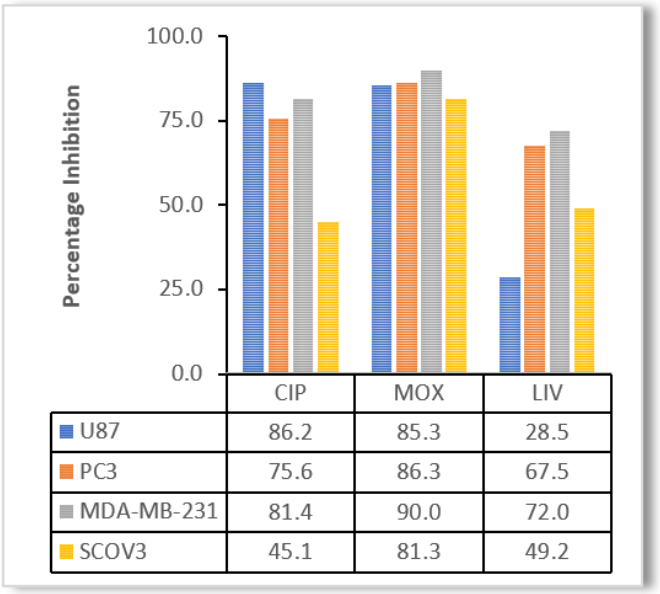
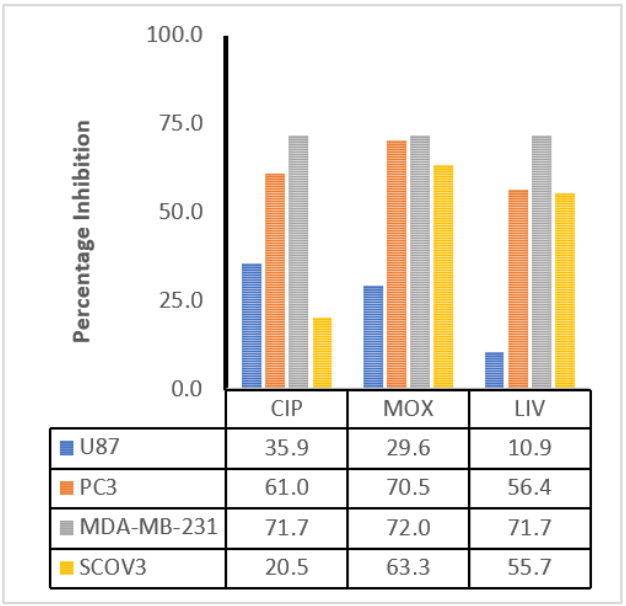
Figure 12: (a) Cell viability determined by MTT assay. Control cells showed 100% cell viability, while it was decreased with an increasing concentration of the levofloxacin against SCOV3, a human ovarian cancer cell line. (b) Photomicrographs of SCOV3 cells post levofloxacin treatment. A significant p value <0.0001, p*** was found and data are presented as Mean±SEM with error bars represented.



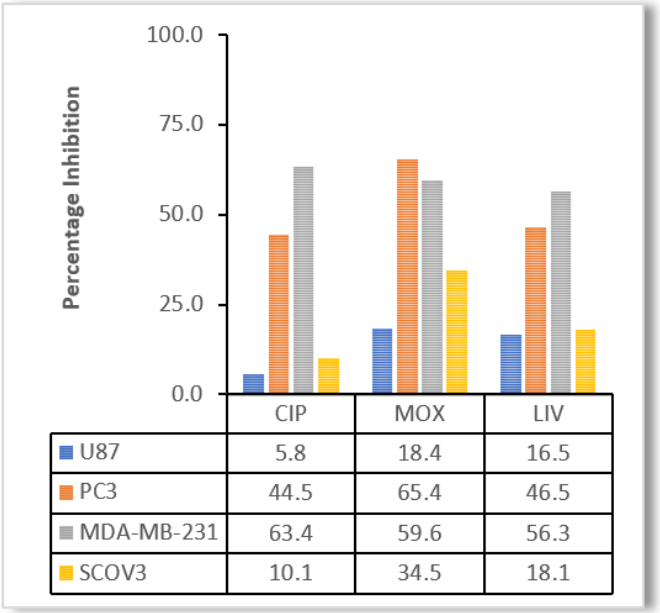
	CIP	MOX	LIV
U87	86.2	85.3	28.5
PC3	75.6	86.3	67.5
MDA-MB-231	81.4	90.0	72.0
SCOV3	45.1	81.3	49.2

a) Concentration 1000 µM



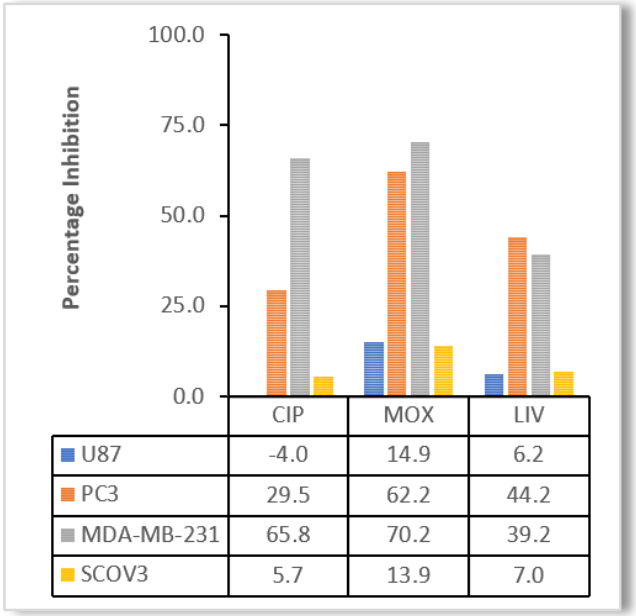
	CIP	MOX	LIV
U87	35.9	29.6	10.9
PC3	61.0	70.5	56.4
MDA-MB-231	71.7	72.0	71.7
SCOV3	20.5	63.3	55.7

b) Concentration 500 µM



	CIP	MOX	LIV
U87	5.8	18.4	16.5
PC3	44.5	65.4	46.5
MDA-MB-231	63.4	59.6	56.3
SCOV3	10.1	34.5	18.1

c) Concentration 250 µM



	CIP	MOX	LIV
U87	-4.0	14.9	6.2
PC3	29.5	62.2	44.2
MDA-MB-231	65.8	70.2	39.2
SCOV3	5.7	13.9	7.0

d) Concentration 125 µM

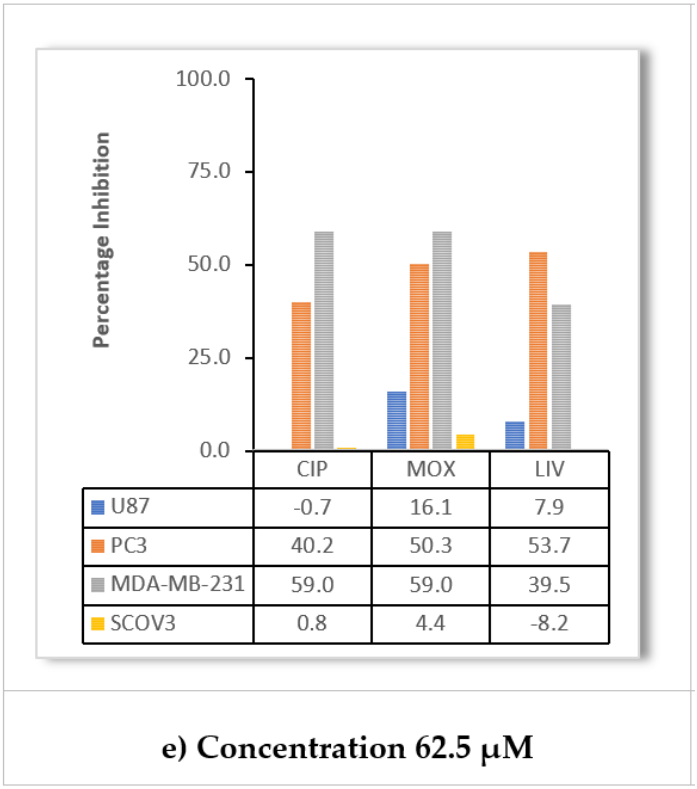


Figure 13: Comparative efficacy of ciprofloxacin, moxifloxacin, and levofloxacin against different cell lines assessed using MTT assay.

Ciprofloxacin showed a good antiproliferative effect with 81%-86% cell growth inhibition against U87, MDA-MB-231 and PC3 cell lines at 1000 µM concentration. Against U87 cell line, up to 500 µM concentration, the maximum effect was 39% inhibition. With the SKOV3 cell line, a 45% growth inhibition was observed. Data are presented in Table 1.

With levofloxacin, the maximum 72% and 68% cell growth inhibition was observed with MDA-MB-231 and PC3 cell lines, respectively at 1000 µM, however, with U87 and SCOV3 cell lines the maximum effect was 36% and 56% only as illustrated in Table 3.

Comparative antiproliferative activities of fluoroquinolones showed different profiles across the cell lines tested. Second-generation ciprofloxacin and third-generation levofloxacin and moxifloxacin were used for the comparative antiproliferative activity against human cancer cells as illustrated in Figure 13. Overall, results indicate that moxifloxacin showed a robust and comparatively strong growth inhibition across the cell lines tested in this study.

DISCUSSION

Fluoroquinolones are indeed used to prevent bacterial infections in cancer patients, especially those undergoing chemotherapy. Chemotherapy-induced neutropenia (a significant drop in white blood cells) increases the risk of infections, and fluoroquinolones can help reduce this risk along with chemotherapy or other cancer treatment. These antibiotics are typically administered prophylactically to prevent infections during periods of neutropenia or any other complications.[15] The choice of the best antibiotic will provide prevention from infection as well and the anticancer property of the same will provide a synergistic or additive effect in cancer patients. Drug repurposing, alternatively termed 'new uses for old drugs' or ' drug repurposing offers a promising pathway for therapeutic innovation but requires careful consideration of its complexities and ethical implications to maximize benefits and minimize risks.[1,8] The majority of noncancer drugs approved for anticancer treatment have common features, including well-defined pharmacokinetic and pharmacodynamic properties, and well-characterized cancer targets.[16,17] The FDA supports drug repurposing primarily through the 505(b)(2) pathway, requiring demonstration of safety and efficacy for the new indication, and provides related guidance and regulatory resources.[18] Fluoroquinolones have known safety profiles and well-defined pharmacokinetic properties that have been established through a long clinical history.

In this study, the two generations of fluoroquinolone drugs-ciprofloxacin, levofloxacin and moxifloxacin were subjected to anticancer activity evaluation against breast cancer (MDA-MB-231), prostate cancer (PC3), Glioblastoma (U87), and ovarian cancer (SCOV3) cell lines. The fluoroquinolones were evaluated for antiproliferative activity using 5 dose

Table 1: Antiproliferative effect of Ciprofloxacin (62.5 µM to 1000 µM) concentration on different cancerous cell lines.

Cell lines	Concentrations vs % cell growth inhibition				
	1000 µM	500 µM	250 µM	125 µM	62.5 µM
MDA-MB-231	81±1.4	72±3.4	63±1.1	66±7.0	59±4.6
PC3	76±1.5	61±1.0	45±6.8	29±3.8	40±9.9
U87	86±3.2	36±1.6	6±3.7	0	0
SKOV3	45±3.7	20±2.6	10±4.5	6±4.9	1±1.8

Table 2: Antiproliferative effect of Moxifloxacin (62.5 µM to 1000 µM) concentration on different cancerous cell lines.

Cell lines	Concentrations vs % cell growth inhibition				
	1000 µM	500 µM	250 µM	125 µM	62.5 µM
MDA-MB-231	90±1.4	72±3.4	60±1.1	70±7.0	59±4.6
PC3	86±1.5	71±1.0	65±6.8	62±3.8	50±9.3
U87	85±3.2	30±1.6	18±3.7	15±1.2	16±1.5
SKOV3	81±3.7	63±2.6	35±4.5	14±4.9	4±1.8

Table 3: Antiproliferative effect of levofloxacin (62.5 µM to 1000 µM) concentration on different cancerous cell lines.

Cell lines	Concentrations vs % cell growth inhibition				
	1000 µM	500 µM	250 µM	125 µM	62.5 µM
MDA-MB-231	72±3.6	72±5.1	56±2.5	39±3.7	40±6.8
PC3	68±4.8	56±5.0	47±0.5	44±5.3	54±4.5
U87	28±3.2	11±2.7	17±3.7	8±7.0	6±2.7
SKOV3	49±1.0	56±3.0	18±6.8	7±10.3	0

determinations (62.5 µM, 125 µM, 250 µM, 500 µM and 1000 µM). The growth inhibition of cancer cell lines was assessed using MTT assay. The comparative assessment of all data showed that out of the three fluroquinolones, moxifloxacin demonstrated the maximum growth inhibition activity across the cancer cell lines evaluated in this study.

The comparative assessment of anticancer activity *in vitro* system, as alone fluoroquinolones, showed a promising result against different cancer cell lines. These *in vitro* observations need to be further explored in combination with approved drugs in an *in vitro* system and later on these findings can be further strengthened with *in vivo* studies.

CONCLUSION

In this study, we investigated the effects of different fluoroquinolones on various cancer cell lines. Our findings demonstrated that all fluoroquinolones possess significant anticancer activity, characterized by their ability to inhibit cell proliferation, however, there is a comparative difference. Among second and third-generation fluoroquinolones, moxifloxacin showed the best effect against U87, MDA-MB-231, PC3, and SCOV3 cell lines. The results support the potential repurpose of fluoroquinolones as viable anticancer agents. However, further research is needed to explore their efficacy *in vivo* and to determine the optimal combination with existing chemotherapeutic agents. Despite the limitations of this study, our findings provide a foundation for future investigations into the therapeutic potential of fluoroquinolones in oncology. In the current study, we for the first time evaluated the comparative *in vitro* activity of different generations of fluoroquinolone drugs against various cancer cell lines. This work showed the good anticancer potential of all fluoroquinolones in different cancer cell lines. These observations need to be further explored in combination with the approved anticancer drugs to get a synergism against the cancer cells, which can be used as a potential therapeutic option for cancer patients.

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None.

ABBREVIATIONS

MTT: 3-(4,5-dimethyl thiazol-2-yl)-2,5-diphenyl tetrazolium bromide; **DMEM:** Dulbecco's Modified Eagle Medium; **HEPES:** 4-(2-hydroxyethyl)-1-piperazine ethane sulfonic acid.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

SUMMARY

The study explored the comparative therapeutic potential of fluoroquinolone antibiotics (ciprofloxacin, moxifloxacin, and levofloxacin) against the different cancer cell lines (MDA-MB-231, PC3, U87, and SCOV3). All three antibiotics proved to have anticancer effectiveness, but moxifloxacin was distinctly superior, inhibiting 81-90% of growth at the highest concentration (1000 µM) across all cell lines. Ciprofloxacin also produced powerful responses (81-86% inhibition in most cell lines at 1000 µM), while levofloxacin was less effective overall. The research focuses on drug repurposing in cancer and recommends further exploration of the effectiveness of these antibiotics along with routine chemotherapy drugs.

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