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Effects of Rosmarinic acid on viability and proliferation of breast cancer cell lines

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Abstract

Objective

Breast cancer is one of the most common malignant cancers in women in the world. This cancer has molecular and clinical heterogeneity, so there are still many therapeutic challenges for it. An appropriate strategy must be found for each specific molecular subtype. On the other hand, the side effects of drugs and the resistance they create have caused scientists to seek safer and more effective therapeutic options. One of the leading compounds for this disease is a natural polyphenolic compound called rosmarinic acid (RA). This compound has significant anticancer properties. Because it induces apoptosis and modulates oncogenic signaling pathways with minimal toxicity to normal cells and inhibits tumor proliferation. Therefore, the aim of this study was to investigate the effects of rosmarinic acid on cell survival, proliferation, and morphology in breast cancer cell lines MCF-7 (luminal A subtype) and MDA-MB-231 (triple negative subtype).

Materials and methods

The Alamar Blue and BrdU incorporation assays across a range of RA concentrations (0–200 µM) was used to quantify cell viability and proliferation. The assessment of morphological alterations indicative of cytotoxicity and apoptosis was performed using microscopic

examination. To measure the sensitivity of cell lines to RA, half-maximal inhibitory concentration (IC50) values were measured for both cell lines.

Results

MCF-7 cells were shown to be more sensitive to RA. They also showed a greater decrease in cell viability and proliferation at lower concentrations compared to MDA-MB-231 cells. Morphological observations revealed that MCF-7 cells exhibited features of apoptosis, including cell shrinkage and membrane blebbing. However, a relative resistance was observed, with an IC50 value of 140 μM for MDA-MB-231 cells compared to an IC50 value of 120 μM for MCF-7 cells. Elongation and a nerve-like morphology without significant cell death were observed for MDA-MB-231 cells at intermediate concentrations (140–160 μM). This difference in response can probably be imputed to the aggressive phenotype and cancer stem cell population that are characteristic of triple-negative breast cancer (TNBC).

Conclusions

From the results of this study, it can be concluded that RA has different cytotoxic effects on breast cancer subtypes and is more effective against luminal A cells. Therefore, it can be said that RA has a great potential for further mechanistic and preclinical research to develop and introduce safer and plant-derived therapeutic options for the management of breast cancer.

Keywords: Breast cancer, MDA-MB-231, MCF-7, Phytochemicals, Rosmarinic acid

Paper Type: Research Paper.

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Introduction

Cancer is one of the leading causes of death in women worldwide, and among these cancers, breast cancer (BC) is one of the most prominent (Xiong et al., 2025). The prevalence and impact of BC is increasing in both developed and developing countries (Sirajudeen et al., 2024). Lifestyle changes and environmental factors can be considered as reasons for this increase (Falzone et al.,

2020). Unfortunately, in most cases, BC is detected and diagnosed when the disease is very advanced and has metastasized to distant organs and tissues, making treatment very difficult and in many cases impossible (Ionescu et al., 2024). This highly heterogeneous disease is classified into four major molecular subtypes: luminal A, luminal B, human epidermal growth factor receptor 2-positive (HER2+), and triple-negative breast cancer (TNBC). The lack of expression of estrogen receptors (ER), progesterone receptors (PR), and HER2 in the MDA-MB-231 cell line, which represents TNBC, makes TNBC largely unresponsive to hormone-based therapies (Ionescu et al., 2024). In contrast, the Michigan Cancer Foundation-7 (MCF-7) cell line, which represents the luminal A subtype, does express estrogen receptors (ER), progesterone receptors (PR), and HER2 (Whelan et al., 2023). Therefore, in this study, we used these two cell lines to characterize distinct molecular subtypes of breast cancer. Reportedly, approximately 15% of all reported breast cancer cases are TNBC, which is both highly aggressive and difficult to detect in its early stages (Chai et al., 2022). On the other hand, luminal A breast cancer has a lower metastatic potential, responds better to targeted hormone-based therapies, and has a higher chance of cure (Jain et al., 2020). These treatments for breast cancer include systemic therapies, such as platinum-based chemotherapy or non-specific chemotherapy regimens, which can be used alone or in combination (Jalil et al., 2023). Unfortunately, these methods have very limited efficacy and are associated with many side effects (Burguin et al., 2021). Therefore, it would be a valuable service to society if more effective and targeted therapeutic strategies could be identified and introduced (Jalil et al., 2023). A review of the history of diseases shows that plant-derived compounds can be used to prevent and treat numerous human diseases, including cancer. Today, phytobiotics and medicinal plants have become the focus of researchers as natural alternatives to synthetic agents in nutrition and medicine (Amirteymoori et al., 2021; Mohammadabadi et al., 2022). Many bioactive molecules such as essential oils, alkaloids, flavonoids, and phenolic acids are abundantly found in these products, which have enhanced their antimicrobial, antioxidant, anti-inflammatory, and anticancer properties (Mohammadabadi et al., 2025). Therefore, phytobiotics can be used to help improve immunity, health status, and overall physiological function of the body (Roudbar et al., 2015; Safaei et al., 2025). If these natural growth promoters are used instead of antibiotics, they can increase metabolic efficiency, reduce disease incidence, and improve oxidative stability (Khezri et al., 2025; Mohammadabadi et al., 2023). Modulating the gut microbiota, improving nutrient absorption, optimizing immune responses, improving nutrient utilization, and reducing environmental impacts are other benefits of phytobiotics (Vahabzadeh et al., 2020; 2021; Mohammadabadi et al., 2024). Bioactive compounds naturally produced by plants are also known as phytochemicals. These compounds can be used as promising sources in the discovery of anticancer drugs (Maleki Dana et al., 2022). One of these

compounds is rosmarinic acid (RA), which is a phenolic compound that has shown antiviral, antibacterial, anti-inflammatory, antioxidant, and anticancer properties and is abundant in *Rosmarinus officinalis* (rosemary) and related species (Guan et al., 2022; Sirajudeen et al., 2024). Therefore, the aim of this study was to investigate the effects of rosmarinic acid on cell survival, proliferation, and morphology in breast cancer cell lines MCF-7 (luminal A subtype) and MDA-MB-231 (triple negative subtype).

Materials and Methods

Materials: MCF-7 and MDA-MB-231 human breast cancer cell lines were provided to us for this research by the Cancer Research Laboratory, Universiti Teknologi Malaysia (UTM). Rosmarinic acid (RA; CHEMFACES, CFND9103) was bought from CHEMFACES. BigBio Sdn. Bhd kindly provided Dulbecco's Modified Eagle Medium (DMEM) and dimethyl sulfoxide (DMSO) for use in this study. We bought fetal bovine serum (FBS) and trypsin-EDTA from Gibco (Canada), penicillin-streptomycin solution from Sigma (USA), bromodeoxyuridine (BrdU) from Merck-BigBio Sdn. Bhd., and Trypan Blue and Alamar Blue reagents from Thermo Fisher–Interscience.

Cell culture: The DMEM supplemented with 10% FBS and antibiotics (100 U/mL penicillin and 100 µg/mL streptomycin) as previously described (Conner et al., 2024) was used to culture MDA-MB-231 and MCF-7 breast cancer cells. The cells were then centrifuged at 1200 rpm for 6 min and resuspended to be cultured in T25 culture flasks. The cells were maintained at 37°C in a humidified incubator containing 5% CO_2 (Fuster et al., 2021) until they reached a confluence of 80–90%. They were then recultured.

Cell viability assay: The Alamar Blue assay was used to evaluate the effect of RA on breast cancer cell viability in MDA-MB-231 and MCF-7 cells. Cells were cultured in 96-well plates at a density of 3500 cells per well (in triplicate). To allow cell attachment, the cultured cells were incubated overnight at 37°C in a 5% CO_2 atmosphere (Ijaz et al., 2023). Cells were treated with increasing concentrations of RA (0, 20, 40, 60, 80, 100, 120, 140, 160, 180, and 200 µM in 100 µL of culture medium) the next day. After 24 h of incubation, inverted light microscopy was used to examine morphological changes. After that, 20 µL of Alamar Blue reagent was added to each well and the plates were incubated for 4 hours at 37°C with 5% CO_2 (Messeha et al., 2020). A microplate reader was used to measure the absorbance at 570 nm. Cell viability was then calculated relative to the untreated control group.

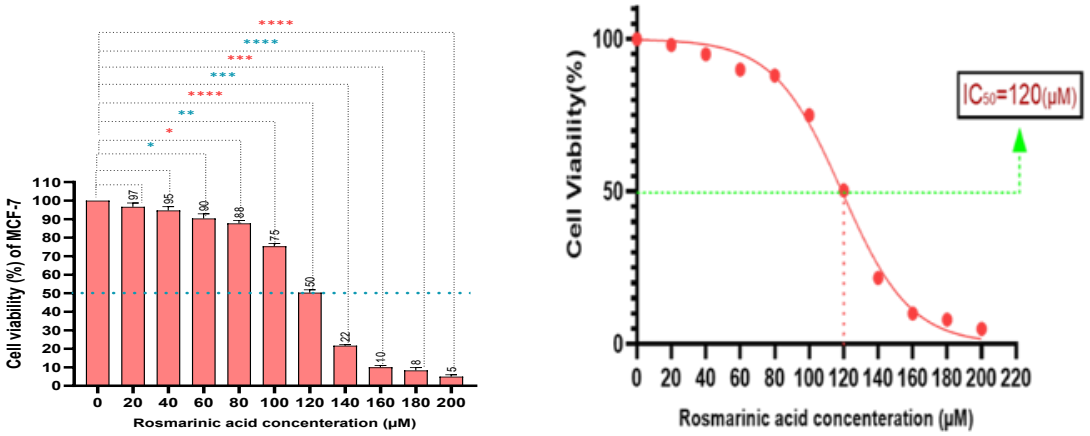
Cell proliferation assay: The BrdU incorporation assay was used to assess the effect of RA on breast cancer cell proliferation. 96-well plates were used to culture MDA-MB-231 and MCF-7 cells at a density of 3500 cells per well (in triplicate) and incubated for 24 h at 37°C. After this

step, the cells were treated with RA at the same concentrations used in the viability assay (0–200 µM in 100 µL total volume per well) for an additional 24 h (Youness et al., 2021). Control wells contained untreated cells. After treatment, 20 µL of BrdU solution was added to each well and the plate was incubated for another 24 h at 37 °C. 200 µL of denaturing solution was used to treat the wells for 30 min at room temperature (Wu et al., 2021). This was followed by washing with 100 µL of buffer diluted 1:50, anti-BrdU monoclonal antibody was added, and incubation was performed for 1 h. The wells were washed three times to remove residual reagents. Preparation of peroxidase-conjugated goat anti-mouse IgG secondary antibody was performed using a 0.2 µm syringe filter. At this stage, 100 µL of this conjugate solution was added to each well and incubation was performed for 30 min at room temperature. Then, washing was done with distilled water, 100 µL of substrate solution was added, and the plate was incubated for 30 minutes in the dark. After this step, 100 µL of stopping solution was added. A microplate reader was used to measure the optical absorbance at a wavelength of 570 nm.

Statistical analysis: GraphPad Prism version 10 was used to perform statistical analyses. The mean ± standard deviation (SD) of triplicate experiments was used to report the results of cell viability and proliferation assays. Two-tailed Student's t-test was used to compare between treatment and control groups.

Results

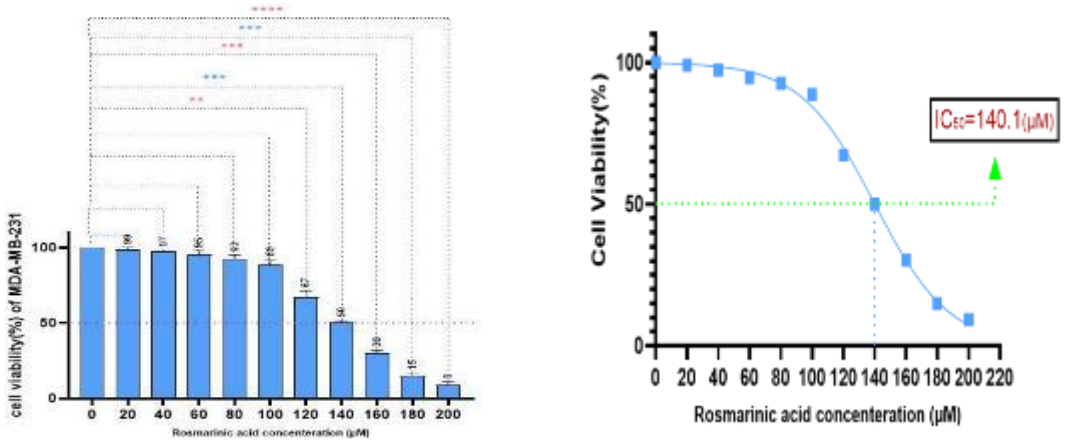
The Alamar Blue assay was used to evaluate the potential anticancer effects of rosmarinic acid (RA) on breast cancer cells, cell viability in MCF-7 and MDA-MB-231 cell lines at RA concentrations ranging from 0 to 200 µM. A concentration-dependent decrease in cell viability was observed for both cell lines, but the magnitude of response was different between the two cell lines. A progressive and statistically significant decrease in the viability of MCF-7 cells treated with RA was observed, with the percentage of viable cells decreasing from 98% at 20 µM to approximately 5% at 200 µM (Figure 1A). Cell growth was initially significantly inhibited at 100 µM, with viability decreasing to approximately 75%. At 120 µM, this inhibition was further increased, with viability decreasing to approximately 50% (IC50 value; Figure 1B). The decrease was greater with increasing concentrations, with viability decreasing to 22%, 10% and 5% at 140 µM, 160 µM, 180 µM and 200 µM RA concentrations, respectively (Figure 1A).



A **B**

Figure 1. Viability of MCF-7 cells exposed to different doses of rosmarinic acid (RA). A: Effect of RA on cell viability. B: Half-maximal inhibitory concentration (IC_{50}) of RA in MCF-7 cells (120 µM). Data are expressed as mean ± SEM. GraphPad Prism 10 was used to determine statistical significance using one-way ANOVA ($p < 0.05$; *$p < 0.001$; **$p < 0.0001$; ***$p < 0.00001$). Bars indicate significant differences between the treated groups and the untreated control group in each independent experiment

MDA-MB-231 cells responded less well to RA treatment at lower concentrations than MCF-7 cells. As shown in Figure 2A, exposure to 0 to 100 µM RA resulted in only a slight decrease in cell viability, ranging from 1% to 11%. Observations showed that a clear dose-dependent inhibition of cell viability was observed at higher RA concentrations (120 to 200 µM). When the concentration was increased from 20 µM to 200 µM, the percentage of viable cells gradually decreased from 99% to 9%. Cell viability first showed a significant and significant decrease at 120 µM RA concentration, at which the viability decreased to approximately 67% (33% inhibition). This effect was exacerbated at a concentration of 140 µM RA, which corresponds to half the maximal inhibitory concentration (IC50; Figure 2B), and then decreased to 70%, 85%, and 91% inhibition at concentrations of 160 µM, 180 µM, and 200 µM, respectively (Figure 2A).



A **B**

Figure 2. Viability of MDA-MB-231cells exposed to different doses of rosmarinic acid (RA). A: Effect of RA on cell viability. B: Half-maximal inhibitory concentration (IC_{50}) of RA in MDA-MB-231 cells (140.1 µM). Data are expressed as mean ± SEM. GraphPad Prism 10 was used to determine statistical significance using one-way ANOVA ($p < 0.05$; *$p < 0.001$; **$p < 0.0001$; ***$p < 0.00001$). Bars indicate significant differences between the treated groups and the untreated control group in each independent experiment

In addition to reducing cell viability, RA treatment also induced significant morphological changes in MCF-7 and MDA-MB-231 cells. The correlation between RA concentration, cell viability, and morphological changes can be seen in Figures 3 and 4. Concentrations of 20 to 80 (low) µM RA did not affect cell morphology, and their normal shape was maintained without significant change. Concentrations of 100 to 120 µM (medium) caused subtle changes in the size and density of MCF-7 cells, but their characteristic epithelial phenotype was maintained. A concentration of 140 µM RA induced significant and prominent morphological changes, causing MCF-7 cells to become smaller, irregular, and rounded (Figure 3). The use of concentrations of 160–200 µM RA (high) resulted in complete loss of epithelial features and the cells became small and detached (Figure 3), showing all the characteristics of apoptotic cells. As shown in Figure 4, MDA-MB-231 cells at concentrations of 20–120 µM RA (bottom) showed minimal morphological changes, unlike MCF-7 cells. However, at concentrations of 120–200 µM RA (top), morphological changes in the cells were significant. At 140 µM RA, MDA-MB-231 cells became more elongated and thinner, and their appearance resembled that of neurons, while maintaining their apparent viability compared to untreated controls (Franchi et al., 2020). The use of concentrations of 160–200 µM RA resulted in significant morphological changes. These changes included shrinkage, rounding, and detachment of the cells, which are indicative of cytotoxic effects (Figure 4). These observations showed that MDA-MB-231 cells exhibit

morphological changes when exposed to RA depending on the RA concentration, and when the concentration increases, structural disorder in these cells increases.

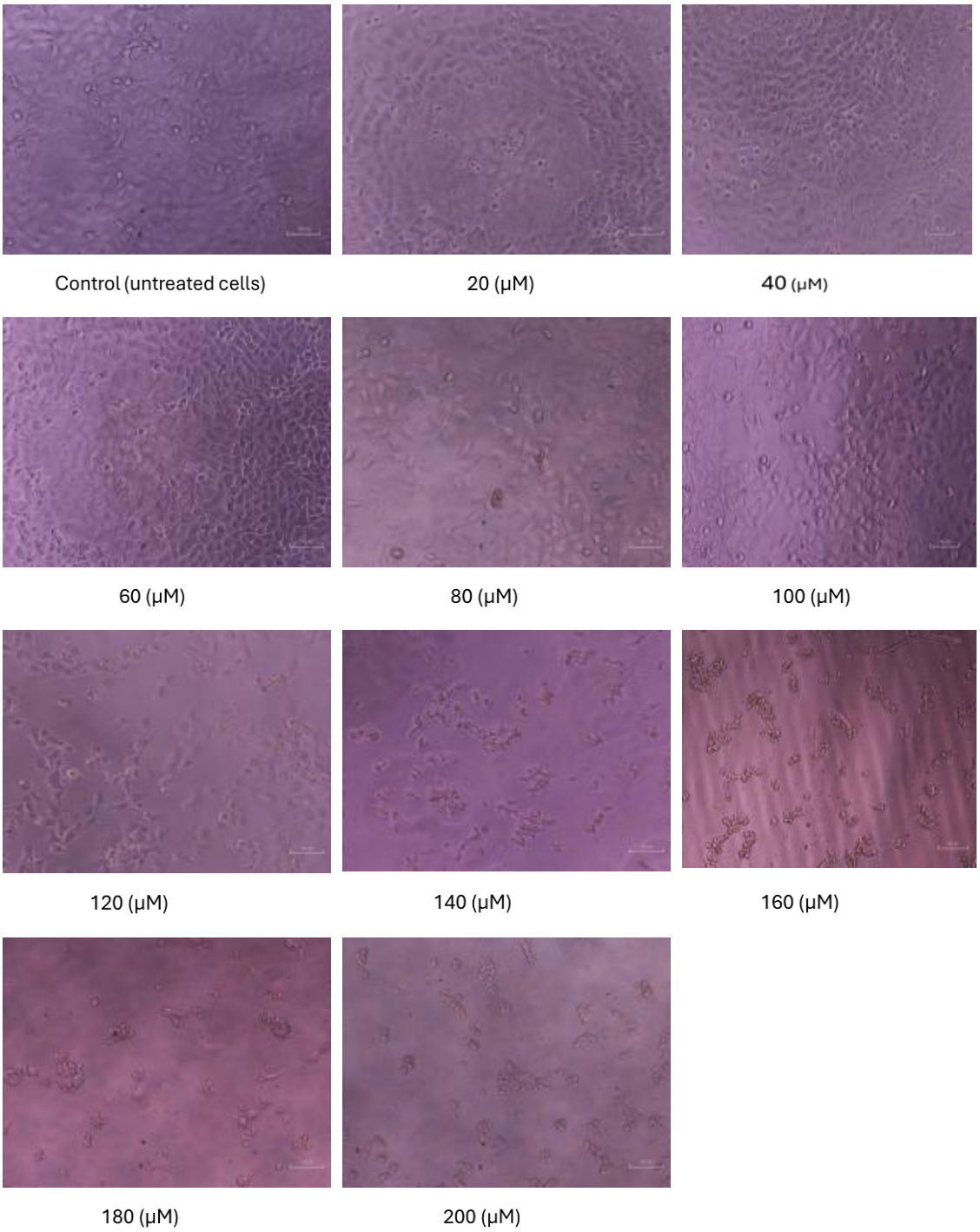


Figure 3. Observed morphological changes in MCF-7 cells after treatment with RA. Microscopic images of untreated and RA-treated MCF-7 cells at different concentrations (0, 20, 40, 60, 80, 100, 120, 140, 160, 180, and 200 μM). All images were taken at 10x magnification using a light microscope

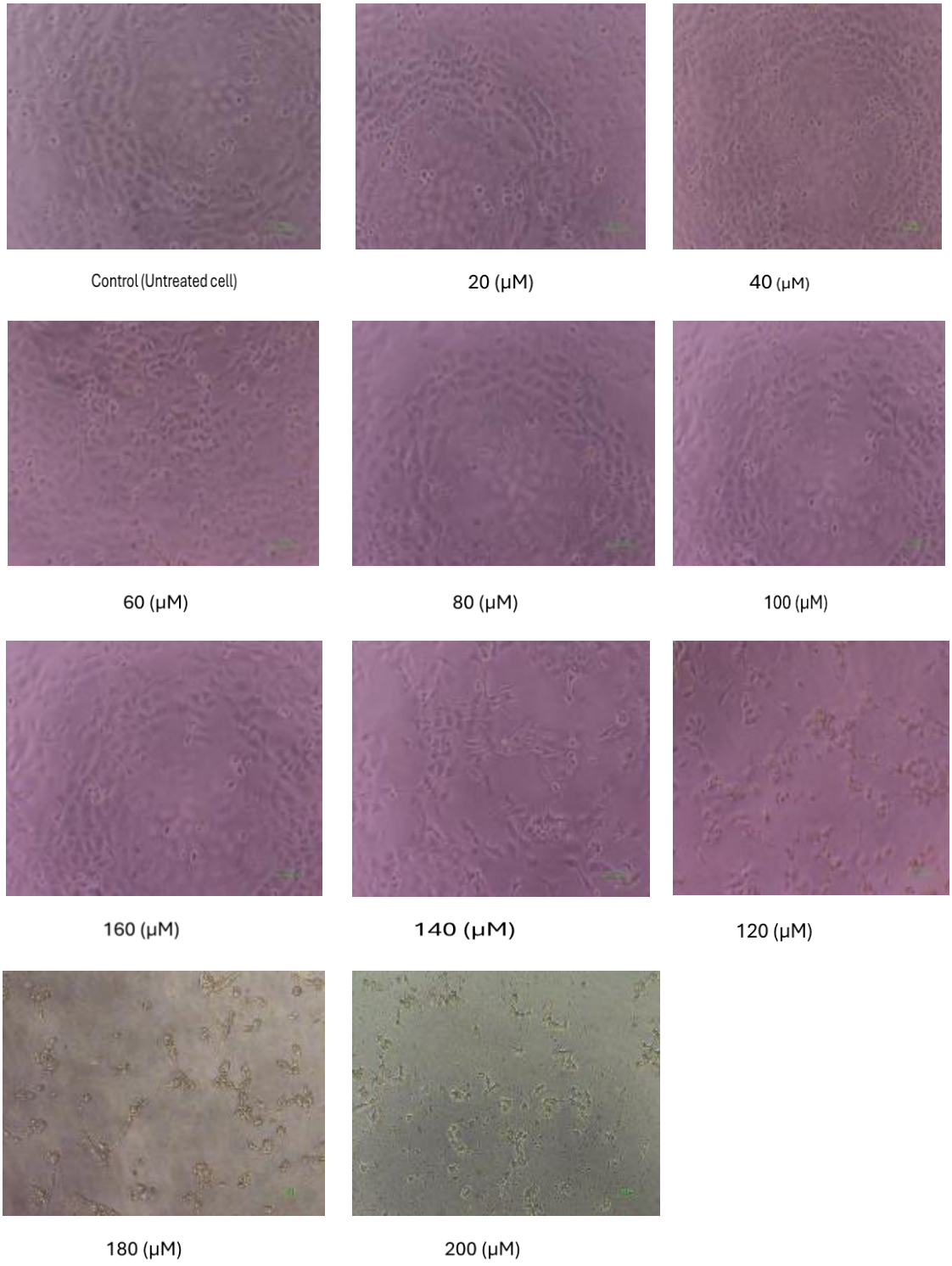
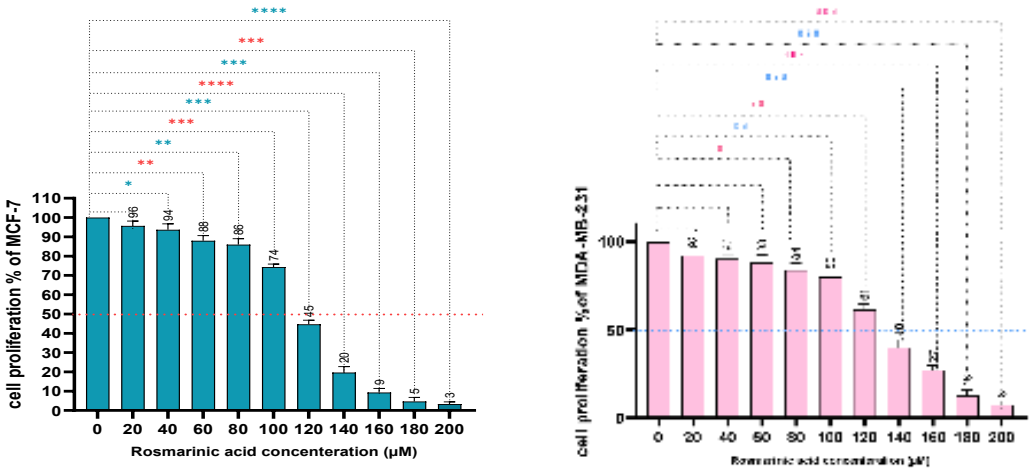


Figure 4. Observed morphological changes in MDA-MB-231 cells after treatment with RA. Microscopic images of untreated and RA-treated MDA-MB-231 cells at different concentrations (0, 20, 40, 60, 80, 100, 120, 140, 160, 180, and 200 µM). All images were taken at 10x magnification using a light microscope

BrdU incorporation assays were used at different concentrations from 0 to 200 µM to assess the effect of RA on the proliferation of MCF-7 and MDA-MB-231 cells, in addition to its effects on cell viability. This colorimetric assay is used to detect DNA synthesis, to detect cell division and proliferation, and to assess cell proliferation activity (Youness et al., 2021). As shown in Figure 5A, RA treatment resulted in a concentration-dependent inhibition of proliferation in both MCF-7 and MDA-MB-231 cells. The results showed that in MCF-7 cells, the proliferation capacity was reduced by 4% at a concentration of 20 µM, and then decreased by 7%, 12%, 15%, 27%, 53%, 80%, 91%, 95%, and 97% with increasing RA concentrations from 40 to 200 µM, respectively. Similarly, a decrease in proliferation was observed in MDA-MB-231 cells starting at 8% inhibition at 20 µM RA, and further reductions of 10%, 12%, 16%, 20%, 29%, 54%, 73%, 87%, and 92% were observed after treatment with 40–200 µM RA (Figure 5B). The results showed that the proliferation capacity of both breast cancer cell lines was suppressed by RA in a dose-dependent manner. MCF-7 cells also showed a slightly higher sensitivity to RA at intermediate concentrations compared to MDA-MB-231 cells.



A **B**

Figure 5. Investigation of breast cancer cell proliferation under RA treatment. (a) MCF-7 cells and (b) MDA-MB-231 cells. Cells were treated with different concentrations of RA for 24 h. Inhibition of cell proliferation by RA was observed in a dose-dependent manner. This assay was performed in three independent experiments and data are expressed as mean ± SEM. GraphPad Prism 10 was used to determine statistical significance using one-way ANOVA ($p < 0.05$; *$p < 0.001$; **$p < 0.0001$; ***$p < 0.00001$)

Discussion

This study demonstrated that rosmarinic acid (RA) produced different sensitivities and effects in two breast cancer subtypes, luminal A (MCF-7) and TNBC (MDA-MB-231). Alamar

Blue and BrdU assays were used to measure and evaluate viability (metabolic activity) and cell proliferation (DNA synthesis) (Youness et al., 2021). The results suggested different mechanisms of action of breast cancer subtypes to RA. MCF-7 cell viability was significantly reduced at 100 μM RA with an IC50 of 120 μM (Figure 1). This decrease may be due to disruption of cellular homeostasis and the initial production of reactive oxygen species (ROS), which in turn causes mild cellular stress (Noor et al., 2022). Some morphological changes, including cell shrinkage and rounding, which are signs of apoptosis, were observed at concentrations of 120–200 μM (Figure 3). Previous studies (Kowalczyk et al., 2024; Bouamali et al., 2024) have shown that RA leads to the activation of cell death pathways through mechanisms such as ROS-induced damage to DNA, proteins, and lipids, as well as interference with mitochondrial function, which is consistent with and confirms the results of the present study. The effect of RA in disrupting metabolic activity and DNA synthesis in MCF-7 cells is confirmed by the correlation between reduced cell viability and increased proliferation. MDA-MB-231 cells were more resistant to RA, requiring higher concentrations to achieve cytotoxic effects (IC_{50} = 140 μM; Figure 2). Concentrations of 140–160 μM induced distinct morphological changes in the surviving cells. These cells survived and became thinner and more elongated, resembling neuronal cells (Figure 4). These findings suggest that RA may be able to limit the proliferative and metastatic properties of TNBC cells and potentially induce partial differentiation (Ali et al., 2021). The majority of MDA-MB-231 cells underwent apoptosis at concentrations of 180–200 μM (high concentration), although a small population of resistant cells survived, likely representing a population of cancer stem cells (CSCs) (Carneiro et al., 2023; Lahiri et al., 2023). Comparative analysis showed that MCF-7 cells were more sensitive to RA and their viability and proliferation were similarly reduced, but for MDA-MB-231 cells, the decrease in proliferation was less than the decrease in viability. Therefore, it can be concluded that concentrations of 20–80 μM (low concentration) could not cause sufficient stress to completely inhibit proliferation. However, for the latter concentrations, significant cytotoxic effects were induced (Kowalczyk et al., 2024). The morphological and functional differences between the two cell lines may be explained by the TNBC characteristics of MDA-MB-231 cells and the presence of CSCs, which are involved in drug resistance and tumor relapse (Nguyen et al., 2020). It is believed that the anticancer effects of RA may be exerted through the interaction between the phenolic and carbonyl groups of RA with cellular proteins, forming complexes that disrupt essential cellular structures (Nadeem et al., 2019). It has been reported that the expression of apoptotic genes such as Bax, Bcl-2, and caspase-3 is modulated by RA, thereby activating intrinsic mitochondrial apoptotic pathways (Mahmoud et al., 2021). Therefore, it can be said that different breast cancer subtypes are selectively affected

by RA, and apoptosis is induced in sensitive luminal A cells. While RA causes proliferation restriction and also promotes differentiation in resistant TNBC cells.

Conclusion: This study reported findings on the sensitivity of two breast cancer subtypes, MCF-7 (luminal A) and MDA-MB-231 (TNBC), to the anticancer effects of RA. Cell viability and proliferation were reduced in both cell lines treated with RA, although the sensitivity varied between the subtypes. MCF-7 cells showed higher sensitivity even at low concentrations of RA (20–80 μM), and effective induction of apoptosis and growth inhibition were observed in them. However, higher concentrations of RA were required to produce similar cytotoxic effects in MDA-MB-231 cells. Morphological changes indicative of differentiation were observed at intermediate doses, followed by apoptosis at higher concentrations (160–200 μM). The results of this study indicate that the anticancer effects of RA and possibly other phytochemicals have specific anticancer effects in each subgroup. Therefore, they can be used as therapeutic agents for different breast cancer subgroups according to the biological characteristics of each subgroup. Therefore, it can be said that RA has a great potential for further mechanistic and preclinical research to develop and introduce safer and plant-derived therapeutic options for the management of breast cancer.

Author contributions

Rafif Aman Mahammed designed and conducted all experiments, collected and analyzed the data, prepared the figures, and wrote the manuscript. Khudhur Raheem Obayes have helped in revising this manuscript. Praseetha Prabhakaran supervised the research, provided guidance on experimental design and data interpretation, and contributed to manuscript review and editing. All authors read and approved the final version of the manuscript.

Data availability statement

The datasets generated and analyzed during the current study are available from 1st author RAFIF Aman Mohammed.

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Ethical considerations

No ethical approval was required as this study did not involve human participants or laboratory animals.

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Conflict of interest

The authors declare that they have no conflict of interest.

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تأثیر اسید روزمارینیک بر زنده‌مانی و تکثیر رده‌های سلولی سرطان پستان

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چکیده

هدف: سرطان پستان یکی از شایع‌ترین سرطان‌های بدخیم در میان زنان جهان است. این سرطان از نظر مولکولی و بالینی ناهمگن است، از این‌رو هنوز چالش‌های درمانی متعددی برای آن وجود دارد. برای هر زیرگونه مولکولی خاص باید راهبرد درمانی مناسب یافت شود. از سوی دیگر، عوارض جانبی داروها و مقاومت دارویی ناشی از آنها، پژوهشگران را به سوی یافتن گزینه‌های درمانی ایمن‌تر و مؤثرتر سوق داده است. یکی از ترکیبات برجسته در این زمینه، ترکیب طبیعی پلی‌فنولی موسوم به اسید روزمارینیک (RA) است. این ترکیب دارای خواص ضدسرطانی قابل‌توجهی است؛ زیرا با سمیت حداقل بر سلول‌های طبیعی، مرگ برنامه‌ریزی‌شده سلول (آپوپتوز) را القا کرده و مسیرهای سیگنال‌دهی سرطان‌زا را تعدیل می‌کند و در عین حال، از تکثیر تومور جلوگیری می‌نماید. بر این اساس، هدف این پژوهش بررسی اثرات اسید روزمارینیک بر زنده‌مانی، تکثیر و مورفولوژی رده‌های سلولی سرطان پستان MCF-7 (زیرگروه لومینال A) و MDA-MB-231 (زیرگروه سه‌گانه منفی) بود.

مواد و روش‌ها: آزمون‌های Alamar Blue و BrdU incorporation در بازه غلظتی ۰ تا ۲۰۰ میکرومولار از RA به‌منظور سنجش زنده‌مانی و تکثیر سلولی به‌کار رفت. ارزیابی تغییرات مورفولوژیک نشانگر سمیت سلولی و آپوپتوز از طریق مشاهده

میکروسکوپی انجام شد. برای تعیین حساسیت رده‌های سلولی به RA، مقادیر غلظت بازدارنده نیمه‌حداکثری (IC50) برای هر دو رده اندازه‌گیری شد.

نتایج: نتایج نشان داد که سلول‌های MCF-7 حساسیت بیشتری نسبت به RA دارند و کاهش قابل‌توجه‌تری در زنده‌مانی و تکثیر سلولی در غلظت‌های پایین‌تر نسبت به سلول‌های MDA-MB-231 نشان دادند. مشاهدات مورفولوژیک حاکی از بروز ویژگی‌های آپوپتوتیک، از جمله کوچک شدن سلول و برجستگی غشایی (membrane blebbing) در سلول‌های MCF-7 بود. در مقابل، مقاومت نسبی در سلول‌های MDA-MB-231 مشاهده شد؛ به‌طوری‌که مقدار IC50 برای این سلول‌ها ۱۴۰ میکرومولار و برای سلول‌های MCF-7 حدود ۱۲۰ میکرومولار بود. در غلظت‌های میانی (۱۶۰-۱۴۰ میکرومولار)، سلول‌های MDA-MB-231 کشیده و دارای مورفولوژی شبیه سلول‌های عصبی شدند، بدون آنکه مرگ سلولی قابل‌توجهی مشاهده شود. این تفاوت در پاسخ احتمالاً ناشی از فنوتیپ تهاجمی و جمعیت سلول‌های بنیادی سرطانی است که از ویژگی‌های ذاتی سرطان پستان سه‌گانه منفی (TNBC)به شمار می‌رود.

نتیجه‌گیری: بر اساس نتایج این پژوهش می‌توان نتیجه گرفت که اسید روزمارینیک اثرات سیتوتوکسیک متفاوتی بر زیرگونه‌های سرطان پستان دارد و بر سلول‌های نوع لومینال A مؤثرتر است. بنابراین، می‌توان گفت که RA پتانسیل بالایی برای انجام پژوهش‌های مکانسیتی و پیش‌بالینی بیشتر دارد تا به‌عنوان گزینه‌ای ایمن‌تر و گیاه‌مبنا برای درمان سرطان پستان معرفی شود.

کلمات کلیدی: اسید روزمارینیک، سرطان پستان، فیتوشیمیایی‌ها، MCF-7، MDA-MB-231

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