

AMYGDALIN AS A POTENTIAL THERAPY FOR BREAST CANCER VIA BAX/BCL2 GENE-MEDIATED APOPTOSIS

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ABSTRACT

Breast cancer (BC) remains one of the most common and difficult-to-treat malignant diseases in global public health, requiring the development of more selective and less toxic therapeutic approaches. In this context, natural compounds have gained increasing attention due to their biological potential in regulating tumor cell processes. Amygdalin (C₂₀H₂₇NO₁₁), a natural cyanogenic glycoside found in the seeds of plants from the Rosaceae family and often referred to as “vitamin B17”, has been studied for its possible anticancer effects. This study aimed to evaluate the effect of amygdalin on the expression of apoptosis-related genes and on cell viability in two human BC cell lines with different molecular profiles: MDA-MB-231 and MCF-7. The cells were treated with amygdalin at a concentration of 50 mg/mL for 24 hours, while NHDF (Normal Human Dermal Fibroblast) cells (Sigma-Aldrich) were used as the control group. The expression levels of the pro-apoptotic gene BAX and the anti-apoptotic gene BCL-2 were assessed using RT-qPCR, while cell viability was analyzed using standard cytotoxicity methods. The results showed a cell-specific gene expression modulation. In MDA-MB-231 cells, amygdalin treatment led to an increase in the expression of both BAX and BCL-2 genes. In MCF-7 cells, a decrease in the expression of both genes was observed, together with a significant reduction in cell viability. These findings suggest that amygdalin has different biological effects depending on the cellular context and tumor molecular profile, thus supporting its potential role in modulating apoptotic pathways in BC cells and pointing towards the amygdalin therapeutic applicability.

KEYWORDS: Amygdalin, breast cancer, apoptosis, BAX, BCL-2, MCF-7, MDA-MB-231.

1. INTRODUCTION

Breast cancer remains one of the most frequently diagnosed malignancies worldwide and continues to represent a leading cause of cancer-related mortality, despite significant advancements in diagnosis and treatment (Pasi et al. 2026). However, problems such as therapy resistance, disease recurrence, and the systemic toxicity of conventional chemotherapy persist. These limitations have driven research toward alternative compounds with safer profiles and more specific activity.

In this context, molecules of natural origin have attracted growing scientific interest. Many plant-based compounds have demonstrated the ability to interfere with the proliferation, survival, and signalling pathways of cancer cells. Amygdalin is one such compound, extensively studied for its potential anticarcinogenic properties. It is a natural cyanogenic glycoside found primarily in apricot kernels and bitter almonds (EFSA Panel on Contaminants in the Food Chain (CONTAM) 2016; Jaszczak-Wilke et al. 2021).

Historically, amygdalin has been associated with alternative cancer treatments; however, its clinical use remains controversial due to inconsistent scientific evidence and concerns regarding cyanide release (Barakat et al. 2022; Chang et al. 2006). Nevertheless, recent in vitro experimental studies conducted on prostate, lung, breast, colorectal, cervical, and gastrointestinal cancers suggest that it may exert effects on tumor cell behavior by modulating apoptosis, inhibiting proliferation, and affecting cellular metabolic pathways. Apoptosis plays a fundamental role in maintaining tissue homeostasis and eliminating damaged or transformed cells. Its regulation is controlled by a network of proteins, including the BCL-2 family, which is overexpressed in malignant cells (Markouli, Pagoni, and Diamantopoulos 2025). BAX functions as a pro-apoptotic factor, whereas BCL-2 acts as an anti-apoptotic protein that promotes cell survival. It is demonstrated that the interplay between these molecules is critical to the fate of cancer cells (Qian et al. 2022; Vogler et al. 2025).

Previous studies have suggested that amygdalin may alter the expression of regulatory genes in cancer cells; however, its effects appear to depend on the cancer type and experimental conditions (Chang). Therefore, understanding how different cell types respond to this compound is essential.

The objective of this study was to investigate the effect of amygdalin on the expression of apoptosis-related genes and on cell viability in two breast cancer cell lines: an estrogen-dependent line (MCF-7, derived from a pleural effusion of breast adenocarcinoma) and an estrogen-independent line (MDA-MB-231, also derived from a pleural effusion of breast adenocarcinoma).

2. MATERIALS AND METHODS

2.1. Cell line cultivation and amygdaline treatment

The MCF-7, MDA-MB-231, and NHDF cell lines (Sigma-Aldrich, Germany) were cultured in the Dulbecco's Modified Eagle Medium (DMEM) supplemented with 10% fetal bovine serum. Cell cultures were maintained in an incubator at 37°C in an atmosphere containing 5% CO₂. These conditions ensure the preservation of metabolic activity and normal cell proliferation.

The use of two cell lines with distinct biological characteristics allows for a comparison of their responses to amygdalin treatment. The MCF-7 line represents a less aggressive form of breast cancer, whereas MDA-MB-231 is considered a model of more invasive and treatment-resistant tumours. Analysing the effect of amygdalin on both cell lines provides more comprehensive information regarding its therapeutic potential.

After reaching the appropriate cell density of 70% of the confluence, the cells were treated with Amy-CDs at a concentration of 50 mg/mL for a period of 24 hours, while untreated MDA-MB-231, MCF-7, and NHDF cells served as control groups for comparison. This exposure time was selected to enable the assessment of changes occurring at the cellular and molecular levels following contact with the substance. We evaluate its effect on cell survival and the activation of apoptotic mechanisms, cell proliferation, cytotoxicity, and the expression of genes involved in the apoptosis process.

2.2 Cytotoxicity assessment and gene expression analysis

The indirect lactate dehydrogenase (LDH) assay was used to evaluate the cytotoxic effect of amygdalin. LDH is a cytoplasmic enzyme present in virtually all cells of the organism. When the cell membrane is damaged or the cell undergoes death, this enzyme is released into the surrounding medium (Kaja et al. 2017). Measuring LDH activity in the culture medium serves as an indicator of cellular damage and treatment-induced cytotoxicity, according to the following equation:

$$\text{Cytotoxicity(\%)} = \frac{(X-Z)}{(Y-Z)} \times 100$$

where X represents the absorbance of the treated sample, Y the absorbance of the maximum lysis control, and Z the absorbance of the negative control.

Cytotoxicity was assessed using the CyQuant LDH Cytotoxicity Assay kit according to the manufacturer's instructions, after the cell treatment with 50 mg/mL Amy@CDs and incubated for 24 hours. Lactate dehydrogenase (LDH) release was measured at a wavelength of 490 nm, with a reference wavelength of 690 nm, using a microplate reader. Cytotoxicity was calculated according to the manufacturer's instructions and expressed as a percentage of total cell lysis or as a percentage of cytotoxicity relative to untreated control cells.

Following treatment with amygdalin, total RNA was isolated from cultured cells using the Quick-RNA MiniPrep Kit according to the manufacturer's protocol.

The isolated RNA underwent quality and quantity analysis using a NanoDrop spectrophotometer. To evaluate the effect of amygdalin on the apoptosis process, the expression of BAX and BCL-2 genes was analyzed. Specific primers for BAX and BCL-2 were used to determine the expression levels of these genes.

Table 1 : Primers used for BAX and BCL-2 genes under standard reaction conditions.

Primer name	Nucleotide sequence	Detected transcript	GenBank database accession number
BAX-F	(5' sense) 5'-GTT TCA TCC AGG ATC GAG CAG-3'	BAX mARN	L22474
BAX-R	(3' antisense) 5'-CAT CTT CTT CCA GAT GGT GA-3'		
BCL-2F	5'-CCT GTG GAT GAC TGA GTA CC-3'	BCL2 mARN	M14745
BCL-2R	5'-GAG ACA GCC AGG AGA AAT CA-3'		

Amplification of the sequences of interest was performed via RT-PCR using a ProFlex thermal cycler. The ratio between BAX and BCL-2 expression is considered an important indicator of the cell's apoptotic status. The RT-qPCR method was used to measure mRNA levels of the BAX and BCL-2 genes. This technique enables real-time monitoring of gene sequence amplification and provides accurate, sensitive results for gene expression analysis. The reaction was carried out using the SYBR Green RT-PCR Kit and specific primers complementary to each gene.

3. RESULTS

3.1 Microscopic evaluation of the effect on cell cultures.

Microscopic examination of cell cultures revealed visible morphological changes distinguishing treated cells from control cells. In MDA-MB-231 and MCF-7 tumor cells, a reduction in cell density, a loss of typical colony organization, and the presence of cells with altered morphology were observed compared to controls.

Treated cells exhibited cell shrinkage, detachment from the culture surface, and reduced intercellular contacts, which are changes characteristic of cells undergoing cellular damage and programmed cell death. Figure 1 and 2 show a comparison of the confluence level between cell cultures treated with amygdalin, and control cultures (untreated with amygdalin). The microscopic image [100x] was obtained after 24 hours of cell culture incubation. These alterations were more pronounced in the MCF-7 cell line, where a greater reduction in cell confluence was observed following treatment with Amy@CDs. NHDF cells displayed more limited morphological changes, suggesting that normal cells are less sensitive to the treatment compared to tumour cells.

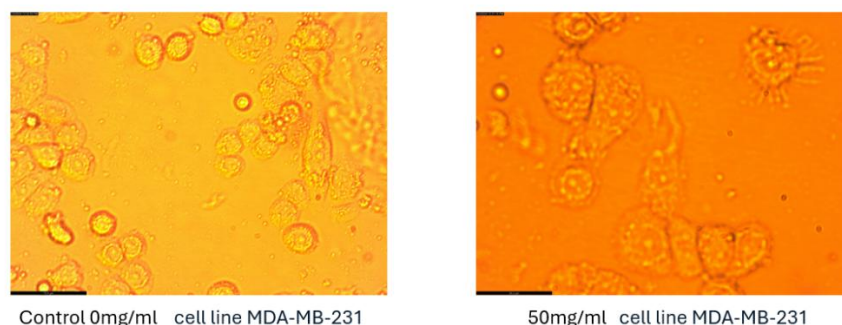


Figure 1. MDA-MB-231 control (left) and treated with amygdaline (50 mg/mL, right image)

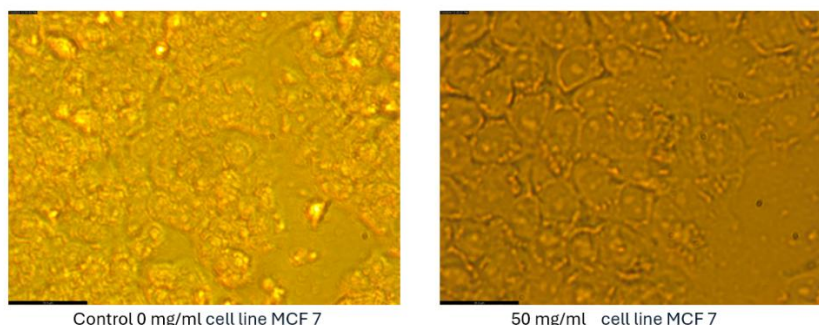


Figure 2. MCF-7 control (left) and treated with amygdaline (50 mg/mL, right image)

3.2 Effect of amygdaline on cell viability

The results of cytotoxic effect of amygdalin are given in Table 2. Treatment of cells with 50 mg/mL Amygdalin for 24 hours caused a significant increase in LDH release in both cell lines, suggesting cell membrane damage and high cytotoxicity. The effect was more pronounced in MCF-7 cells compared to MDA-MB-231 cells, a result that reinforces the previously reported higher sensitivity of MCF-7 cells to Amygdalin (Mosayyebi et al. 2021). The increased LDH activity in the culture medium supports the microscopic observations and indicates that Amy@CDs cause cellular damage and loss of membrane integrity in tumour cells.

Table 2: LDH cytotoxicity with a "high control" absorbance of approximately 1.05.

Cell line	Control (0 mg/ml)	Amygdalin 50 mg/ml
MCF-7	0.182 ±0.014	0.694±0.031
MDA-MB-231	0.201 ±0.018	0.473 ± 0.027

These results suggest that Amy@CDs exert a cytotoxic effect on breast cancer cells and affect the integrity and organization of cell cultures.

3.3 Analysis of gene expression

To assess the activation of apoptotic pathways, the expression of the pro-apoptotic gene BAX analysed using the RT-qPCR method are shown in figure 3.

In MDA-MB-231 cells, treatment with Amy@CDs caused a statistically significant increase in BAX mRNA copy numbers compared to the control group. This increase suggests the activation of cellular mechanisms that promote apoptosis.

In MCF-7 cells, a statistically significant decrease in BAX expression was observed ($p = 0.0130$), although this decrease was accompanied by a marked reduction in cell viability. Despite the observed decrease in BAX

expression, the treatment was associated with a significant reduction in cell viability, suggesting that the cytotoxic effect of amygdalin may also be mediated by other molecular mechanisms.

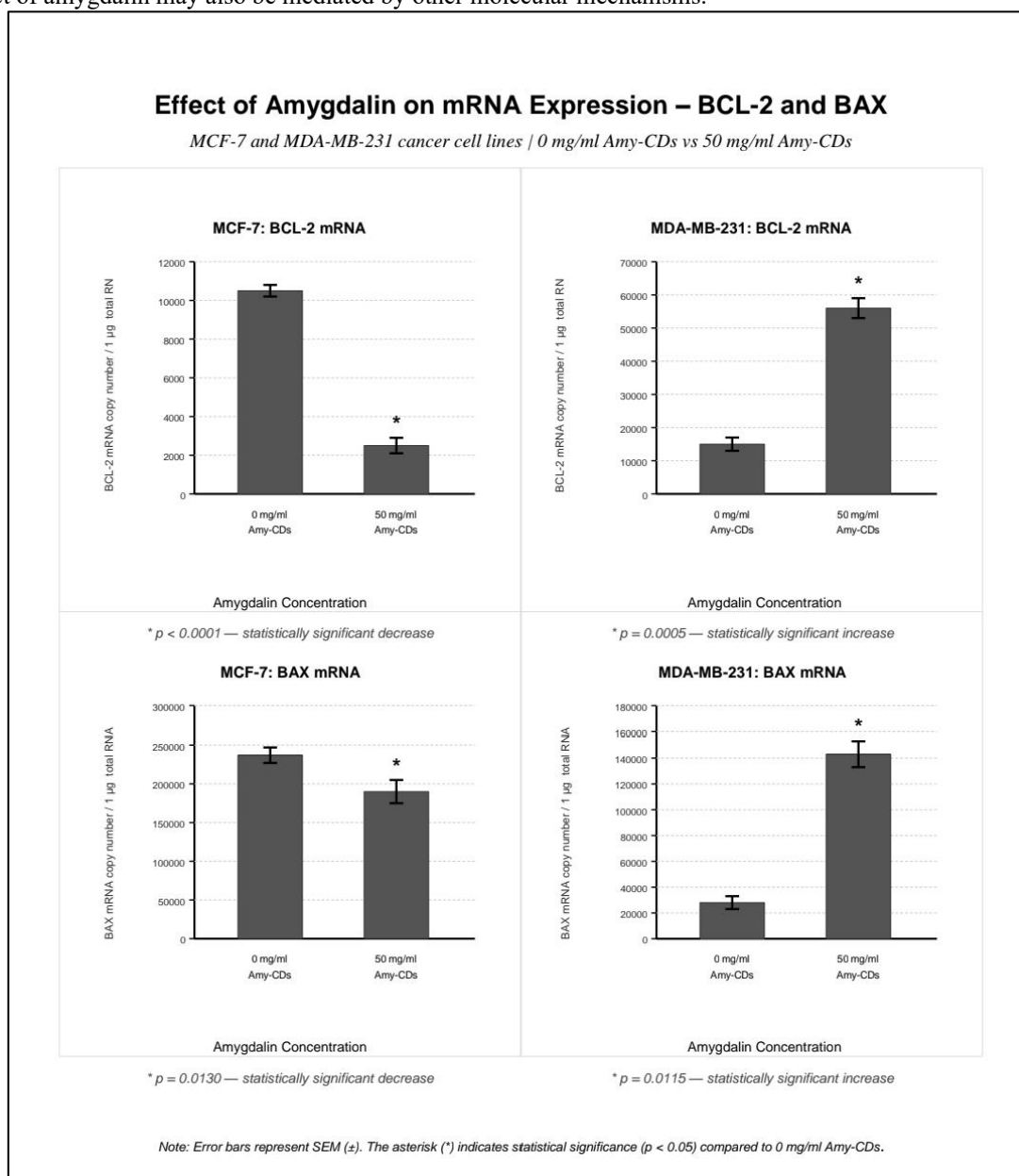


Figure 3. Relative expression of the BAX gene in MCF-7 and MDA-MB-231 cells and relative expression of the BCL-2 gene in MCF-7 and MDA-MB-231 cells after treatment with Amy-CDs (50 mg/mL for 24 hours) compared to control groups

The expression of the anti-apoptotic gene BCL-2 was analysed to evaluate the impact of the treatment on cell survival mechanisms (Figure 3). The results showed that treatment with Amy@CDs induced significant changes in the expression level of this gene in both cell lines. In MDA-MB-231 cells, a statistically significant increase in BCL-2 expression was observed ($p = 0.0002$; $p < 0.0005$); however, the increase in BAX was more pronounced, suggesting a relative shift in the balance toward pro-apoptotic signals. In MCF-7 cells, a significant decrease in BCL-2 expression was observed ($p < 0.0001$). Given that BCL-2 acts as a protective factor against apoptosis, its downregulation suggests a weakening of cell survival mechanisms and increased susceptibility to programmed cell death, which is a desirable outcome for the anti-cancer therapies.

Overall, the results indicate that Amy@CDs affect molecular pathways involved in the regulation of apoptosis and promote the activation of pro-apoptotic signals in MDA-MB-231 cells.

4. DISCUSSIONS

The results of this study demonstrate that treatment with amygdalin formulated with carbon dots (Amy@CDs) exerts a significant biological effect on MCF-7 and MDA-MB-231 breast cancer cell lines. Microscopic analysis, the LDH cytotoxicity assay, and RT-qPCR gene expression analysis revealed that Amy@CDs affect both cell viability and the molecular mechanisms involved in apoptosis regulation. Morphological changes observed post-

treatment, accompanied by increased cytotoxicity, indicate that Amy@CDs negatively impact tumor cell survival. The cytotoxic effect was more pronounced in MCF-7 cells compared to MDA-MB-231 cells, suggesting that the response to treatment depends on the molecular and biological characteristics of each cell line. This supports the concept that breast cancer heterogeneity plays a significant role in determining the efficacy of antitumor treatments. Analysis of BAX and BCL-2 gene expression confirmed that Amy@CDs interfere with apoptotic regulatory pathways. A statistically significant increase in the expression of both genes was observed in MDA-MB-231 cells, suggesting the simultaneous activation of pro-apoptotic mechanisms and protective cell-survival mechanisms. This phenomenon may reflect the ability of these more aggressive cells to activate adaptive responses to treatment-induced stress. In contrast, MCF-7 cells exhibited a significant decrease in BAX and BCL-2 expression, accompanied by a marked reduction in cell viability, indicating higher sensitivity to the cytotoxic effect of Amy@CDs. A particularly important aspect of this study is the use of the Amy@CDs nanoformulation. Carbon dots are known for their biocompatibility, high stability, and ability to enhance the intracellular transport of bioactive molecules. In this context, the formulation of amygdalin with carbon dots (Amy@CDs) may have contributed to increasing the compound's biological efficacy, enabling more effective interaction with tumour cells and a more pronounced modulation of apoptosis-related molecular pathways. The results obtained are consistent with previous *in vitro* studies reporting the anti-proliferative, and pro-apoptotic activity of amygdalin in various cancer cell types (Arshi et al. 2019; Sireesha et al. 2019). At the study of Chang (Chang et al. 2006) reported that treating DU145 and LNCaP prostate cancer cells with amygdalin caused an increase in the expression of the pro-apoptotic gene BAX, a decrease in the expression of the anti-apoptotic gene BCL-2, and the activation of caspase-3, thereby inducing programmed cell death. These authors suggested that the antitumor effect of amygdalin is mediated primarily through an increase in the BAX/BCL-2 ratio and the activation of the mitochondrial apoptotic pathway. Meanwhile, (Zielińska et al. 2022) demonstrated that amygdalin reduced the proliferation and metabolic activity of MCF-7, MDA-MB-231, and T-47D breast cancer cells in a dose-dependent manner. The authors also reported changes in the transcription levels of the BAX, CASP, and BCL-2 genes, supporting the involvement of apoptotic mechanisms in the anticancer action of amygdalin. MCF-7 cells were found to be more sensitive to the treatment compared to other cell lines, indicating that the effect of amygdalin may depend on the molecular characteristics of each tumour type. Our results are consistent with the findings of the present study and suggest that Amy@CDs may exert their effects through a similar modulation of apoptotic pathways. Furthermore, the results of this study are supported by the findings of Saleem (Saleem et al. 2018), who reported that amygdalin exerts anticancer activity in various tumour cell types through the induction of apoptosis, cell cycle arrest, and the modulation of signalling pathways associated with cell survival. The authors highlighted that the biological effect of amygdalin is associated with alterations in the balance between pro-apoptotic and anti-apoptotic factors, thereby promoting programmed cell death. However, the study's findings suggest that the response to Amy@CDs is not uniform and varies depending on the molecular profile of the tumour cells. This indicates that the effect of the nanoformulation may be closely linked to the biological characteristics of each breast cancer subtype and genetics (Hasa et al. 2026; Xhetani et al. 2026). This study presents some limitations that should be taken into account. First, the analysis was conducted solely *in vitro* and over a limited treatment duration (24 hours). Second, only two key apoptotic genes were analysed, whereas cell death pathways involve a much more complex molecular network. Future studies should include the analysis of relevant proteins (e.g., BAX, BCL-2, and caspase-3), the assessment of oxidative stress, and *in vivo* studies to confirm the therapeutic potential and safety of Amy@CDs.

CONCLUSIONS

The results support the study's objectives and demonstrate that Amy@CDs exert antitumor activity in breast cancer cells by affecting cell viability and apoptosis-related molecular pathways. The differences observed between the MCF-7 and MDA-MB-231 cell lines suggest that the response to treatment is influenced by the biological characteristics of each tumour subtype. Although the results are promising, further studies are required to fully elucidate the underlying molecular mechanisms, analyse other apoptosis-related proteins, and evaluate the efficacy of Amy@CDs in more complex biological models, including *in vivo* studies.

Ethics Approval. Not applicable. This study does not involve human participants, animals, or sensitive personal data.

Data Sharing Statement. The datasets generated and analysed during the current study are available from the corresponding author upon reasonable request.

Authors' Contributions. MXh.: Conceptualization, Methodology, Software, Supervision, Data curation, Writing – original draft, AH. and AB.: Validation, Investigation, Visualization, Writing – review & editing, KSh.: Methodology, Data curation, Writing – original draft, LM. and AV: Software, Formal analysis, Writing – review & editing, MP.: Data curation, Visualisation, Validation, Writing – review & editing.

All authors have read and agreed to the published version of the manuscript.

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Declaration of Interest. The authors declare that they have no competing financial interests.

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