

Introduction

Cancer is a disease characterised by the uncontrolled proliferation of cells, their invasion into other parts of the body by exceeding their normal limits and/or their spread to other organs. Cancer is the second leading cause of death worldwide after ischaemic heart disease (8.97 million deaths) and is projected to become the leading cause of death by 2060 (Mattiuzzi & Lippi, 2019). According to the International Agency for Cancer, there are 18.1 million cases of cancer worldwide; this number is expected to rise to 29.5 million by the end of 2040, with around 9.5 million people losing their lives to cancer (Bakan et al., 2021). The ever-increasing number of cancer cases poses an economic threat to all countries worldwide, and it is even expected that expenses could exceed government budgets (Torun & Kutlar, 2018).

Surgery, chemotherapy, radiotherapy and immunotherapy are the most commonly used methods in cancer treatment. Chemotherapy is the first choice for patients whose cancer cells can be induced by apoptosis (Vici et al., 2016). Cisplatin is one of the first-line antitumour drugs in the treatment of many types of cancer. However, although it is administered as first-line chemotherapy, response rates are quite low at 13–36% (Mauricio et al., 2021). Drug resistance, one of the major problems in cancer treatment, poses a particular challenge for patients with advanced-stage cancer and leads to treatment failure (Wang et al., 2023). In addition, many of the drugs used in cancer cause numerous undesirable side effects, particularly hepatotoxicity and nephrotoxicity, and impair patients' quality of life (Pezeshki et al., 2017). Patients may experience short-term and long-term side effects from chemotherapy, including alopecia (hair loss), nausea, vomiting, diarrhoea, infertility, and brain fog (Brianna & Lee, 2023). In recent years, chemotherapy and radiotherapy methods targeting DNA have been frequently used in cancer treatment. However, these treatments increase DNA damage not only in cancer cells but also in the surrounding healthy tissue, causing toxicity and accelerating ageing (van den Boogaard et al., 2022).

People are turning to a natural and healthy diet to prevent cancer or reduce the unwanted side effects of cancer treatment. They want to benefit from the protective and therapeutic effects of natural products by including fruit rich in vitamins, minerals and phenolic substances, as well as fruit jam, fruit juice and marmalade in their daily diet. This study aims to compare the antiproliferative and antioxidant effects of aqueous apricot extract and apricot jam, traditionally made from the same fruit, on healthy and cancer cells. This study aims to answer the question of whether the consumption of fruit or jam should protect against cancer or have a negative effect on the course of the disease.

The apricot (*Prunus armeniaca* L.) is a species of the Rosaceae family known for its fruit, which is consumed as food. Although it is distributed worldwide, Türkiye produces the largest amount of apricots among the Mediterranean countries and exports them worldwide (Ali et al., 2015). Like other fruits in the *Prunus* genus, apricots have a delicious flavour and attractive appearance and are rich in vitamin C, β -carotene, phenols, and carotenoids (Karatas, 2022). These compounds display various bioactivities, including antioxidant, cytotoxic, enzyme inhibitory, and anti-inflammatory properties (Güven et al., 2024; Jaafar, 2021). In our previous study, we reported that *P. armeniaca* and *P. domestica* leaves exhibited tyrosinase enzyme inhibition (Güven & Basaran, 2021).

Jam is a nutrient-rich, energy-rich food that makes it possible to eat fruit all year round without it spoiling. Many of the foods we eat today (fruit, vegetables, nuts, etc.) can be preserved for months by making jam using traditional methods. To make jam, the fruit is cooked whole or in pieces, sweetened with sugar and stored in suitable containers (Tamer, 1999). The high temperatures used in the jam-making process accelerate the degradation of certain antioxidant compounds, particularly phenolic compounds. This loss of antioxidants, which play a significant role in disease prevention and treatment, alters the food's biological effects. Furthermore, increased browning, disruption of tissue integrity, and loss of nutritional value change the fruit's physical, chemical, and sensory properties (Patras et al., 2011; Shinwari & Rao, 2018).

This study compared the cytotoxic and antioxidant effects of fruits and traditionally produced jams and determined how added sugar and temperature alter the biological effects of fruits. The study began with healthy L929 fibroblast cells, and a concentration range was determined that did not damage the cells. Subsequently, the antiproliferative effect was determined using the MTT method in PC-3 prostate cancer, MCF-7 breast cancer, CaCo-2 colon cancer, A549 lung cancer and Hep3B liver cancer cells. In addition, the antioxidant effects were compared using the DPPH, CUPRAC and TEAC methods. To our knowledge, this is the first study to compare the antiproliferative and antioxidant effects of traditional apricot jam and an aqueous extract prepared from apricots collected during the same period. This study investigated the cytotoxic and antioxidant effects of high temperatures and added sugar used in traditional jam preparation on cancer and healthy cells, by comparing them with fruit juice extracts. The results are important for increasing consumer awareness of food use and demonstrating how to optimise the health benefits of food for a healthy lifestyle.

Materials and Methods

Plant Material

Plant material, *Prunus armeniaca* L. (apricot) fruits, was collected (1.25 kg) from Üzümlü, Erzincan, Türkiye in June 2025. The voucher specimens (KNYA Herb.No: 30374) were identified by Prof. Dr. Osman TUGAY (Department of Pharmaceutical Botany, Faculty of Pharmacy, Selcuk University, Konya, Türkiye). The studies were conducted using fruits collected from the same tree. The fruits were selected based on criteria including full ripeness, uniform appearance, and absence of textural defects.

Preparation of Extracts

After the fruits (0.25 kg) were brought to the laboratory, their seeds were removed, sliced and placed. Water was added, and the extract was treated in a water bath at 40 °C for 8 hours. After filtration, the water was evaporated under negative pressure in a rotary evaporator to obtain the main aqueous extract.

Preparation of the jam

The jam was prepared according to the traditional methods of the locals, as described below. The apricots (1 kg) were washed and rinsed with 1 L of carbonated water. They were cut in half, and the stones removed. Approximately 1 kg of sugar (1:1) and 0.25 L of water were added to each kilogram of apricots and left to infuse overnight (about 12 hours). The apricots were removed, and the juice was boiled (90 °C) for 3 to 4 hours until it thickened.

The aqueous extract and jam were freeze-dried separately to obtain lyophilised powders for use in biological efficacy studies. This prevented moisture contamination and ensured the extracts could be preserved for an extended period.

Evaluation of Cytotoxic Effect

Antiproliferative effect of the extract was carried out using the MTT method [3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide]. L929 (Mouse fibroblast), A549 (Lung adenocarcinoma), CaCo-2 (colorectal adenocarcinoma), Hep3B (human hepatoma), MCF-7 (Breast adenocarcinoma) and PC-3 (prostate adenocarcinoma) cells obtained from the American Type Culture Collection (ATCC) (Güven et al., 2022; Mosmann, 1983).

Cell suspensions of 1×10^5 cells/mL were prepared from all cells and divided into 96-well plates of 100 µl each. The cells were kept in an incubator with 5% CO₂ at 37°C for 24 hours to proliferate. After the waiting period, the medium in the

wells was removed, and instead, 100 µL solutions with different concentrations of the samples to be tested, which had been prepared in the medium, were added and incubated for 48 hours. After incubation, the sample solutions in the wells were aspirated, and 100 µL of fresh medium was added. 10 µl of an MTT solution with a concentration of 5 mg/mL was added to the wells and incubated for a further 4 hours. At the end of the incubation, the medium in the wells was aspirated, and 100 µl of DMSO (dimethyl sulfoxide) solution was added to ensure dissolution of the formed formazan crystals. The absorbance values of the dissolved crystals were measured at 570 nm.

Evaluation of the Antioxidant Capacity

2,2-Diphenyl-1-picrylhydrazyl (DPPH) radical scavenging effect

The DPPH radical scavenging effect of the aqueous extract and jam was determined using the method from our previous studies. Absorbance values for the resulting colour change were measured at 517 nm, and the percentage inhibition compared to the control group was calculated. Results were expressed as gallic acid equivalents, a natural antioxidant (Güven et al., 2022; Harput et al., 2012).

Trolox equivalent antioxidant capacity (TEAC)

The ABTS [2,2'-azino-bis(3-ethylbenzothiazoline-6-sulfonic acid)] radical scavenging assay was performed according to the procedure used in previous studies. Equal volumes of 0.007 M ABTS and 0.0024 M persulfate were mixed and allowed to stand for 12–16 hours to generate ABTS radicals. The colour change in the experimental medium after treatment with the extract and jam was measured at 734 nm. Antioxidant capacity was calculated as equivalent to the standard compound Trolox and expressed as mg Trolox/g extract (Re et al., 1999).

Copper reducing antioxidant capacity (CUPRAC)

The antioxidant activity of apricot aqueous extract and jam was determined using the CUPRAC method developed by Apak et al., and the copper-reducing capacity was measured spectrophotometrically at 450 nm. The antioxidant capacity of trolox, used as the standard compound, was calculated under the same experimental conditions, and the results were expressed as mg trolox/ g extract equivalent to Trolox (Apak et al., 2007).

Statistical Analysis

All experiments were repeated three times, and because the data were normally distributed, the results are presented as mean ± SD. Comparative statistical analysis of the means was

performed using one-way ANOVA (analysis of variance) and Tukey's post hoc test for normally distributed parameters. $P < 0.05$ was considered significant.

Results and Discussion

Cytotoxic Effect Evaluation of the Extract and Jam

The antiproliferative effects of apricot fruit aqueous extract and jam, both prepared from the same fruits using traditional methods, were evaluated on healthy L929 (mouse fibroblast) cells and cancer cell lines A549 (lung adenocarcinoma), CaCo-2 (colorectal adenocarcinoma), Hep3B (human hepatoma), MCF-7 (breast adenocarcinoma), and PC-3 (prostate adenocarcinoma) using the MTT assay. The studies began by determining a concentration range that would not harm the healthy cell line. Both the jam and the aqueous extract were applied to cells at concentrations ranging from 50 to 1600 $\mu\text{g/mL}$. Cell viability in the aqueous extract remained virtually unchanged at all concentrations. This may be due to the cell selectivity of the compounds in the aqueous extract, the low concentration of cytotoxic compounds within the tested range of 50–1600 $\mu\text{g/mL}$, or the resistance of healthy cells to the compounds in the extract. In the jam, cell viability decreased to 90.5% and 80.8% at the highest concentrations of 800 and 1600 $\mu\text{g/mL}$, respectively (Figure 1).

Antiproliferative effects were also investigated in cancer cell lines at the same concentration range. The aqueous extract showed increased cytotoxicity with increasing concentration (Fig. 2). The highest cytotoxic effect was observed against Hep3B cells at a concentration of 1600 $\mu\text{g/mL}$, with cell viability decreasing to 58.8%. At concentrations above 400 $\mu\text{g/mL}$, the jam reduced the viability of healthy cells in a concentration-dependent manner (Fig.1). In cancer cells, however, concentrations above 400 $\mu\text{g/mL}$ led to increased cell proliferation compared to the control group (Fig.3). Notably, at 1600 $\mu\text{g/mL}$, cell proliferation increased by 20.4% in the MCF-7 cell line, 16.4% in the CaCo-2 cell line, 14.8% in the A549 cell line, 14.2% in the Hep3B cell line, and 12.8% in the PC-3 cell line. The fact that jam, unlike fruit, may induce proliferation in cancer cells could be linked to the added sugar in the traditional jam-making process, or it may be due to the loss of antioxidant and cytotoxic compounds resulting from exposure to high temperatures.

Numerous studies confirm the relationship between sugar consumption and cancer. Experimental data indicate that sugar can play a role in the development of cancer via obesity, inflammatory and oxidative mechanisms and insulin resistance. A cohort study of 101,279 individuals investigating the association between total or added sugar intake and cancer risk

(breast and prostate cancer) found that higher total sugar intake was associated with higher cancer risk, particularly for breast cancer (Debras et al., 2020). Another meta-analysis evaluating the potential causal relationship between sugary beverage consumption and cancer risk and mortality showed that sugary beverage consumption may increase cancer risk and mortality, particularly the risk of breast cancer, hepatocellular carcinoma, colorectal cancer and prostate cancer, as well as mortality from breast cancer (Li et al., 2021). Cancer cells must increase their glucose uptake to grow and survive. Increased glucose supplies the energy required and enables the production of essential intermediates for macromolecule synthesis in cancer cells. Furthermore, the glycolytic dependence observed in cancer cells increases resistance to chemotherapeutic treatments (Chelakkot et al., 2023). Epidemiological data suggest that long-term consumption of high glycaemic index diets, which rapidly raise blood sugar, may increase cancer risk, at least in part through the insulin/insulin-like growth factor 1 (IGF-1) signalling pathway (Kasprzak, 2021). Furthermore, studies show a direct relationship between bone marrow and tumour glucose uptake and systemic inflammation, with increased glucose uptake worsening the course of cancer (Dolan et al., 2019). Similarly, increased glucose uptake is associated with epigenetic modifications such as Breast Cancer 1 (BRCA1) and Mutl Homolog 1 (MLH1) hypermethylation, and promotes tumour progression (Saggese et al., 2020).

In our previous study, we compared the antiproliferative effects and tyrosinase enzyme inhibition of white mulberry water extract and molasses, both obtained from the same fruit, against L929, A549, Hep3B, and PC-3 cells. Molasses, produced by prolonged boiling, showed a much lower cytotoxic effect on cancer cells than the water extract. Similarly, the tyrosinase enzyme inhibition of molasses was also lower. Unlike this study, molasses prepared using traditional methods was made without added sugar but was boiled at approximately 90 °C for 18–20 hours. These temperatures and durations are considerably higher than those used in jam preparation. The difference in results between the fruit and molasses may be due to the degradation of compounds in the mulberry fruit when exposed to high temperatures for an extended period, leading to the formation of new compounds that may be harmful to health (Güven, 2025). In a study, the antioxidant and anti-tumor effects of strawberry jams prepared using two different methods were compared with those of unprocessed fruit, and changes in phytochemical content were reported. Significant decreases in ellagic acid and total flavanol levels were observed in the jam compared to the fruit. Furthermore, due to heat treatment, the amount of carcinogenic volatile compounds in the jams increased. These compounds are Maillard reaction products formed by the reaction of glutamic acid and

glucose, such as 5-hydroxymethyl-2-furfural, mesifuran, and γ -butyrolactone (Flores and Ruiz del Castillo, 2016). Acrylamide and furans are toxic substances that produce genotoxic and carcinogenic effects in foods when exposed to high tem-

peratures. Experiments on rats have shown that chronic exposure to furans leads to hepatocellular adenomas and cancer. The threshold value for cancer is approximately 0.064 mg/kg body weight per day (El Hosry et al., 2025; Von Tungeln et al., 2017).

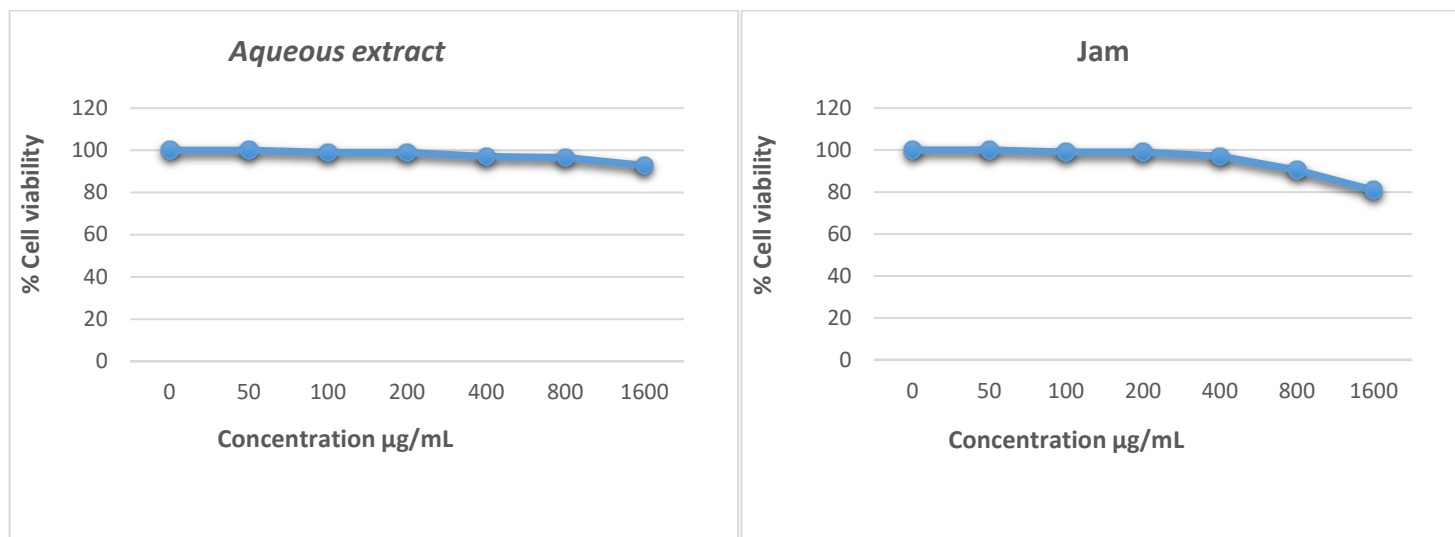


Fig. 1. The antiproliferative effect of different concentrations of aqueous extracts and jam on L929 cells

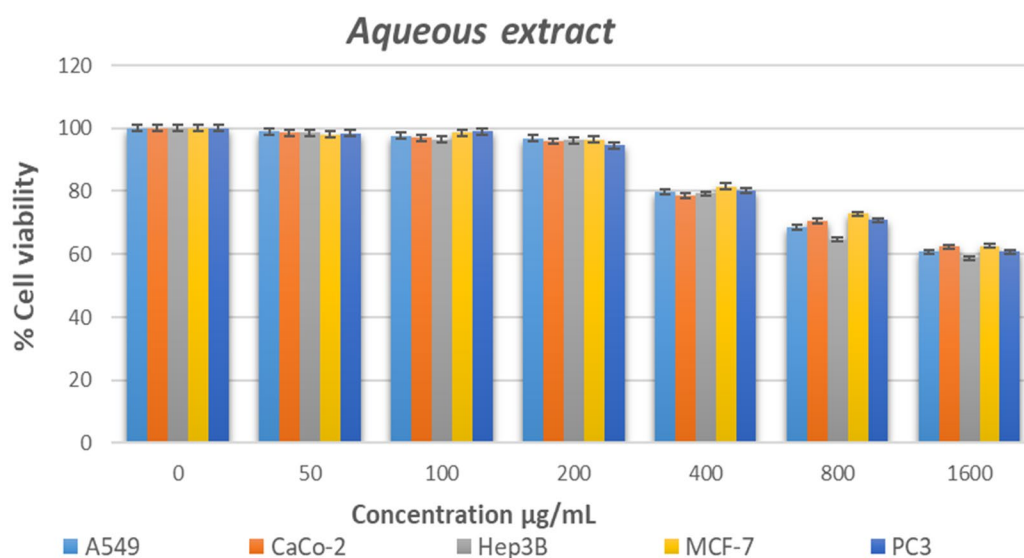


Fig. 2. The antiproliferative effect of aqueous extracts at different concentrations against cancer cells

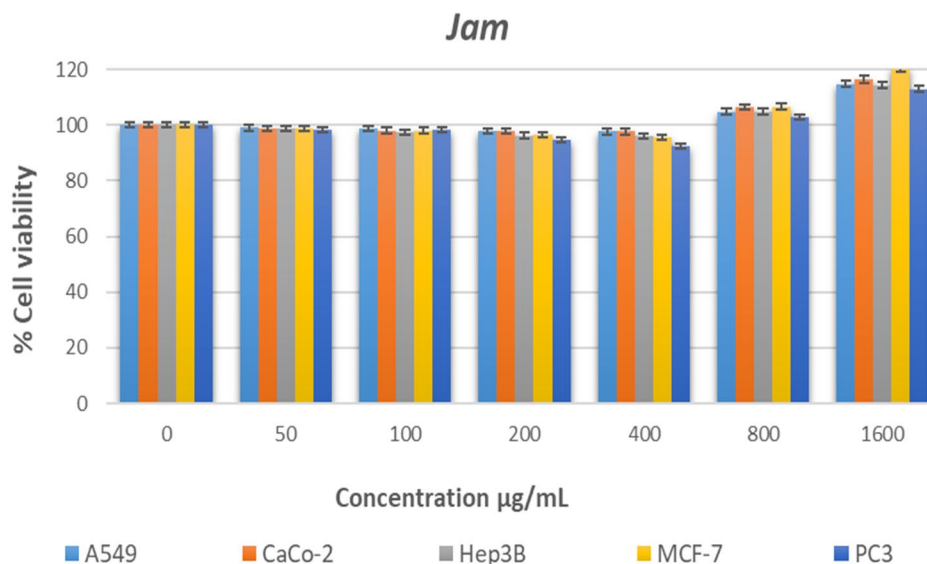


Fig. 3. The antiproliferative effect of jam at different concentrations against cancer cells

Table 1. Antioxidant capacity of aqueous extract and jam

	DPPH	CUPRAC	TEAC
Aqueous extract	124.12 ± 1.62	148.12 ± 2.02	118.22 ± 1.84
Jam	68.02 ± 1.82	102.14 ± 1.42	90.16 ± 1.68

DPPH: 2,2-Diphenyl-1-picrylhydrazyl; CUPRAC: Copper Reducing Antioxidant Capacity; TEAC: Trolox equivalent antioxidant capacity. The results were expressed as mg gallic acid/g extract for DPPH and CUPRAC, while TEAC was expressed as mg trolox/g extract; Data are presented as mean ± SD, n=3 experiments, ($p < 0.05$)

Antioxidant Capacity of the Extracts

The antioxidant capacity of aqueous extracts and jams from apricot fruits was determined using the DPPH, TEAC, and CUPRAC methods. Comparisons were made to assess the effects of sugar addition and temperature on antioxidant activity. DPPH radical scavenging activity was expressed as gallic acid equivalents, while CUPRAC and ABTS radical scavenging activities were expressed as Trolox equivalents. In all methods, the antioxidant activity of the aqueous extract was significantly higher than that of the jam. According to the CUPRAC method, the copper ion reducing power of the aqueous extract was 148.12 mg Trolox/g extract, while ABTS radical scavenging activity was 118.22 mg Trolox/g extract (Table 1). The difference in antioxidant capacity between jam and aqueous extract may be because heat treatment and added sugar negatively affect certain classes of antioxidants (such as flavonoids, carotenoids, and vitamin C) through various degradation mechanisms, including thermal instability, oxidation, and

interaction with sugars (Ahmed et al., 2025; Kopjar et al., 2016; Tang et al., 2012).

Since fruits are exposed to high temperatures for a long time during jam making, antioxidant capacity changes depending on the phenolic compounds present. In a study, the antioxidant capacity of cherry, plum, and raspberry fruits was found to be 354.8–692.3 mg/100 g vitamin C, while the antioxidant capacity of jams was reported as 205.6–373.5 mg/100 g. The same study showed that heat reduced not only the antioxidant effect but also the total phenol and total anthocyanin levels (Kim et al., 2004). In another study, the antioxidant capacity of strawberry and blueberry fruits and commercially available jams was determined using the DPPH method. Blueberry fruits had a value of 2,143.8 mg TE/kg FW, while jams showed DPPH radical scavenging activity ranging from 1,155.8 to 1,965.4 mg TE/kg FW. Strawberry fruits had a lower value than blueberry fruits (1,193.9 mg TE/kg FW), and their jams ranged from 1,076.1 to 1,178.9 mg TE/kg FW (Mustafa et al., 2022).

In a study by Prvulović et al., contrary to other literature, blueberry jam showed higher antioxidant capacity compared to the fruit. Total phenolic, total tannin, and total flavonoid contents were also found to be higher than in the fruit (Prvulović et al., 2021). This difference may be attributed to specific phytochemical compositions, various processing methods, or different analytical techniques used in the studies.

Conclusion

Since ancient times, people have included natural foods rich in phytochemicals in their daily diets to protect themselves from disease and maintain good health. However, because fruits are available fresh only in certain seasons, they are often processed into products such as jam, molasses, and marmalade for year-round consumption and long-term storage without spoilage. This study, for the first time, determined and compared the cytotoxic and antioxidant effects of apricot fruits and jam prepared traditionally. Prolonged exposure to heat and the addition of sugar made the jam more toxic to healthy cell lines at high concentrations compared to the aqueous extract. It was also found that jam at high concentrations increased cancer cell proliferation. The fact that traditionally prepared jam, at high concentrations *in vitro*, can damage healthy cells and increase the proliferation of cancer cells indicates that caution should be exercised when consuming jam. Antioxidant capacity was determined using the DPPH, CUPRAC, and TEAC methods, and in all methods, the aqueous extract showed a higher antioxidant effect than the jam. These *in vitro* results may influence cancer patients' preference for consuming fresh fruit rather than jam to improve their quality of life, especially during treatment. However, it should not be overlooked that these findings must also be confirmed *in vivo* and in clinical studies.

Compliance with Ethical Standards

Conflict of interest: The author(s) declare that they have no actual, potential, or perceived conflicts of interest related to this article.

Ethics committee approval: The authors declare that this study includes no experiments with human or animal subjects. Ethics committee approval is not required for this study.

Data availability: The data will be made available upon request by the author(s).

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Disclosure: -

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