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# *Rosa canina* Fruit Extract Exhibits Potent Anticancer and Anti-Inflammatory Activities via Antioxidant Mechanisms

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**ABSTRACT**

**Background & Objective:** Cancer continues to pose a significant global health challenge, and its progression is intricately linked to oxidative stress and persistent inflammation. *Rosa canina* (wild rose) has been traditionally used in Iranian medicine for its therapeutic properties; however, its anticancer and anti-inflammatory properties have not been comprehensively investigated. In this experimental study, the *Rosa canina* fruit extract was evaluated for its antioxidant, anticancer, and anti-inflammatory activities.

**Materials & Methods:** Spectrophotometric techniques were employed to perform phytochemical screening to determine total phenolic content (TPC), total flavonoid content (TFC), and condensed tannins (CTC). Antioxidant activity was assessed using DPPH and ABTS radical scavenging assays. Cytotoxic effects were evaluated on HeLa, Hep3B, and HCT116 cancer cell lines using the MTT assay, while anti-inflammatory effects were evaluated in RAW 264.7 macrophages through nitric oxide (NO), prostaglandin E2 (PGE2), and tumor necrosis factor-alpha (TNF-α) assays.

**Results:** The extract contained high levels of alkaloids, terpenoids, polyphenols, and flavonoids. It demonstrated strong, concentration-dependent antioxidant activity in both DPPH and ABTS assays ($p<0.05$). The viability of cancer cell lines decreased significantly in a dose-dependent manner, while the production of NO, PGE2, and TNF-α in LPS-stimulated macrophages was significantly inhibited ($p<0.05$).

**Conclusion:** The hydroalcoholic extract of *Rosa canina* fruit possesses remarkable antioxidant, anti-inflammatory, and anticancer properties, which may be attributed to its rich polyphenolic and flavonoid composition.

**Keywords:** *Rosa canina, Antioxidant, Anti-inflammatory, Anticancer, Polyphenols, Flavonoids*

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## 1. Introduction

Cancer is a significant contributor to both morbidity and mortality on a global scale, ranking as the second leading cause of death, surpassed only by cardiovascular diseases. Despite substantial advances in diagnostic and therapeutic strategies, the global burden of cancer continues to rise (1). Conventional anticancer therapies such as chemotherapy and radiotherapy frequently entail significant side effects, the development of drug resistance, and limited effectiveness, which has prompted the search for new therapeutic compounds obtained from natural resources (2). In recent years, medicinal plants have gained increasing attention as potential sources of bioactive compounds with anticancer, antioxidant, and anti-inflammatory properties (3). Oxidative stress and persistent inflammation are pivotal in the onset and progression of cancer. Reactive oxygen species (ROS) and reactive nitrogen species (RNS) arise as natural byproducts of cellular metabolic processes, but their excessive accumulation can lead to DNA damage, lipid peroxidation, and activation of pro-inflammatory signaling pathways. Chronic inflammation plays a significant role in cancer progression by facilitating cell proliferation, angiogenesis, and resistance to apoptosis (4).

Therefore, compounds exhibiting potent antioxidant and anti-inflammatory characteristics may represent promising candidates for preventing or suppressing cancer development (5). *Rosa canina L.,* commonly

known as dog rose or wild rose, belongs to the *Rosaceae* family and is widely distributed throughout Europe, the Middle East, and Asia. Its fruits (rose hips) are rich in various phytochemicals, including polyphenols, flavonoids, carotenoids, vitamins (particularly vitamin C), and essential fatty acids. Traditionally, *Rosa canina* has been used in Iranian and European folk medicine for the treatment of respiratory disorders, infections, arthritis, and inflammatory conditions. On study showed that extracts of *Rosa canina* possess significant antioxidant and cytoprotective activities, suggesting possible therapeutic application in diseases associated with oxidative stress, such as cancer (5). Recent studies have suggested that *Rosa canina* and its bioactive constituents may modulate several molecular pathways involved in cancer cell growth, differentiation, and survival (6). Polyphenols, flavonoids, carotenoids, and vitamin C present in *Rosa canina* have been reported to influence key signaling cascades, including PI3K/Akt/mTOR, MAPK, and NF-κB pathways, which regulate cellular proliferation, apoptosis, inflammation, and oxidative stress responses (7). Furthermore, *Rosa canina* extracts have been shown to induce cell-cycle arrest, promote mitochondrial dysfunction, activate caspase-dependent apoptosis, and suppress gene expression associated with tumor progression (8). These findings suggest that *Rosa canina* may exert its anticancer effects through multiple complementary mechanisms targeting cancer cell growth and survival.

However, the anticancer and anti-inflammatory effects of *Rosa canina* fruit extract have not been thoroughly elucidated, particularly in relation to specific cancer cell lines. Therefore, the present study aimed to examine the phytochemical composition and assess the antioxidant, anti-inflammatory, and anticancer activities of the hydroalcoholic extract of *Rosa canina* fruit in vitro.

## 2. Materials and Methods

### 2.1 Plant Material and Extract Preparation

Fresh fruits of *Rosa canina* were collected from the suburbs of Kermanshah, Iran, and authenticated by a botanist at Kermanshah University of Medical Sciences. The fruits were washed, air-dried, and ground into fine powder. Fifteen grams of the powdered material were extracted with 150 mL of 70% ethanol for 48 hours at room temperature in the dark. The extract was filtered through Whatman No. 42 filter paper and then concentrated by evaporation of the solvent at room temperature (9).

### 2.2 Phytochemical Screening

Phytochemical analysis was performed using standard colorimetric and precipitation tests to detect the presence of alkaloids, terpenoids, flavonoids, tannins, saponins, and polyphenols. Alkaloids were identified using Mayer's, Dragendorff's, and Wagner's reagents; tannins were detected with ferric chloride; terpenoids with the Liebermann–Burchard test; and flavonoids using the cyanidin reaction (10).

### 2.3 Determination of Total Phenolic, Flavonoid, and Tannin Content

The total phenolic content (TPC) was assessed using the Folin–Ciocalteu method and is reported as µg of gallic acid equivalents (GAE) per mg of dry extract. The total flavonoid content (TFC) was quantified through the aluminum chloride colorimetric assay and is expressed as µg of quercetin equivalents (QE) per mg of extract. The condensed tannin content (CTC) was determined by the vanillin–HCl method and is expressed as µg of catechin equivalents (CE) per mg of extract (11).

### 2.4 Antioxidant Activity Assays

The antioxidant potential of the extract was determined using DPPH and ABTS radical scavenging assays. For the DPPH assay, 2 mL of the extract at various concentrations was mixed with 2 mL of 0.1 mM DPPH in methanol. After a 30-minute incubation in the dark, the absorbance was measured at 517 nm. In the ABTS assay, the ABTS radical cation was generated by combining 7 mM ABTS with 2.45 mM potassium persulfate, incubating for 16 hours in the dark, and adjusting the absorbance to 0.70 ± 0.02 at 734 nm. Two hundred microliters of the extract were then added to 1 mL of the ABTS solution, and the absorbance was recorded after 3 minutes. The percentage of radical scavenging activity was calculated relative to the control (10).

### 2.5 Cell Lines and Culture Conditions

Human cancer cell lines, including cervical carcinoma (HeLa), hepatocellular carcinoma (Hep3B), and colorectal carcinoma (HCT116), along with the murine macrophage cell line (RAW 264.7), were sourced from the National Cell Bank of Iran (NCBI). The cells were maintained in RPMI-1640 medium enriched with 10% fetal bovine serum (FBS) and 1% penicillin-streptomycin, incubated at 37°C in a humidified environment containing 5% $CO_2$.

### 2.6 Cell Viability Assay (MTT Assay)

The cytotoxic properties of *Rosa canina* extract were investigated using the MTT assay. Briefly, $1.5 \times 10^4$ cells per well were seeded in 96-well plates and allowed to adhere overnight. The cells were then treated with different concentrations of *Rosa canina* extract (100, 200, 400, 800, and 1600 µg/mL) for 24 hours. After treatment, 30 µL of MTT solution (5 mg/mL) was added to each well and incubated for 3 hours at 37°C. The resulting formazan crystals were dissolved in 100 µL of DMSO, and the absorbance was measured at 570 nm using a microplate reader. Cell viability was expressed as a percentage relative to the untreated control cells (12). $IC_{50}$ values were calculated using GraphPad Prism version 9.0.

### 2.7 Anti-Inflammatory Activity

RAW 264.7 macrophages were treated with different concentrations of the extract (1–10 µg/mL) for 1 hour, followed by stimulation with lipopolysaccharide (LPS, 1 µg/mL) for 24 hours. Nitric oxide (NO) production was measured using Griess reagent at 540 nm (13). The levels of prostaglandin $E_2$ ($PGE_2$) and tumor necrosis factor-alpha (TNF-α) in the culture supernatants were quantified using commercial ELISA kits according to the manufacturer's protocols.

### 2.8 Statistical Analysis

All experiments were performed independently three times (biological replicates), and each measurement within a single experiment was carried out in triplicate (technical replicates). Data are expressed as mean ± standard deviation (SD). Statistical analyses were performed using GraphPad Prism version 9.0 (GraphPad Software, San Diego, CA, USA). Comparisons among multiple groups were made using one-way analysis of variance (ANOVA), followed by Tukey's multiple comparison post hoc test. A p-value of less than 0.05 was considered statistically significant.

## 3. Result

### 3.1 Phytochemical Composition

Preliminary phytochemical screening revealed that the hydroalcoholic extract of *Rosa canina* fruit contained high levels of alkaloids, terpenoids, polyphenols, and flavonoids, moderate amounts of gallic and catechin tannins, saponins, and no detectable mucilage, resin, or coumarins (Table 1).

The extract therefore contains a broad range of bioactive secondary metabolites that may contribute to its biological activities.

**Table 1.** Phytochemical screening results of hydroalcoholic extract of *Rosa canina* fruit.

| Chemical components | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| Alkaloids | Gallic tannins | Catechic tannins | Terpenoids | Mucilage | Coumarine | Polyphenols | Resin | Saponins | Flavonoides |
| ++ | + | + | ++ | - | - | +++ | - | + | ++ |

**Note: (-): absence of compound; (+): low compound content (++): medium compound content; (+++): High compound content.**

### 3.2 Total Phenolic, Flavonoid, and Tannin Contents

The total phenolic content (TPC) of the extract was 68.19 ± 3.24 µg GAE/mg, total flavonoid content (TFC) was 35.41 ± 1.88 µg QE/mg, and condensed tannin content (CTC) was 72.46 ± 3.69 µg CE/mg of dry extract. The high polyphenolic and flavonoid levels suggest strong antioxidant capacity.

### 3.3 Antioxidant Activity

The *Rosa canina* extract demonstrated potent, concentration-dependent radical scavenging activity in both DPPH and ABTS assays (Figure 1 A and B). Increasing extract concentration significantly enhanced DPPH and ABTS radical inhibition ($p \leq 0.05$). These results confirm the strong ability to neutralize free radicals, reflecting its high content of phenolic and flavonoid compounds.

### 3.4 Cytotoxic Effects on Cancer Cell Lines

The hydroalcoholic extract of *Rosa canina* significantly reduced the viability of HeLa (cervical), Hep3B (liver), and HCT116 (colon) cancer cell lines in a dose-dependent manner ($p < 0.05$) (Figure 2). At higher concentrations, cell viability decreased markedly compared with the untreated control group. These findings indicate that *Rosa canina* exerts cytotoxic effects against multiple human cancer cell types.

### 3.5 Anti-Inflammatory Effects in Macrophages

In RAW 264.7 macrophages, the extract significantly suppressed lipopolysaccharide (LPS)-induced nitric oxide (NO) production in a dose-dependent manner ($p < 0.05$) (Figure 3 A). Moreover, *Rosa canina* treatment markedly reduced the secretion of pro-inflammatory mediators, prostaglandin $E_2$ ($PGE_2$) and tumor necrosis factor-alpha (TNF-α), compared with LPS-stimulated controls ($p < 0.001$) (Figure 3 B and 3 C).

These findings demonstrate that the hydroalcoholic extract of *Rosa canina* exerts strong anti-inflammatory activity by inhibiting the release of key inflammatory factors in macrophages.

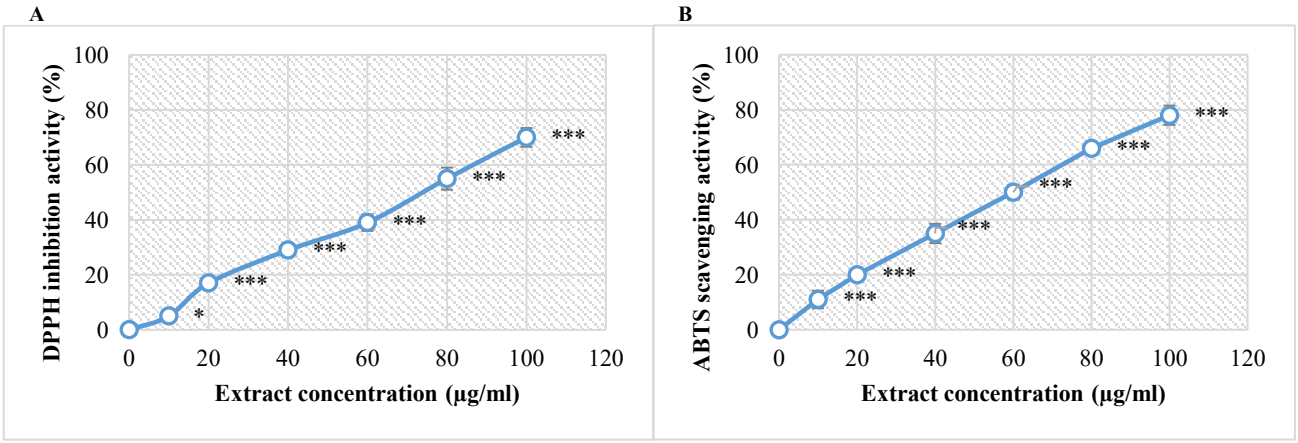



**Figure 1.** Figure 1 (A) DPPH and (B) ABTS radical scavenging activities of hydroalcoholic extract of *Rosa canina* fruit at different concentrations. Data are presented as mean ± SEM from three independent experiments. * $p < 0.05$, ** $p < 0.01$, and ***$p < 0.001$ compared with the control group. **(Prepared by Authors, 2026)**.

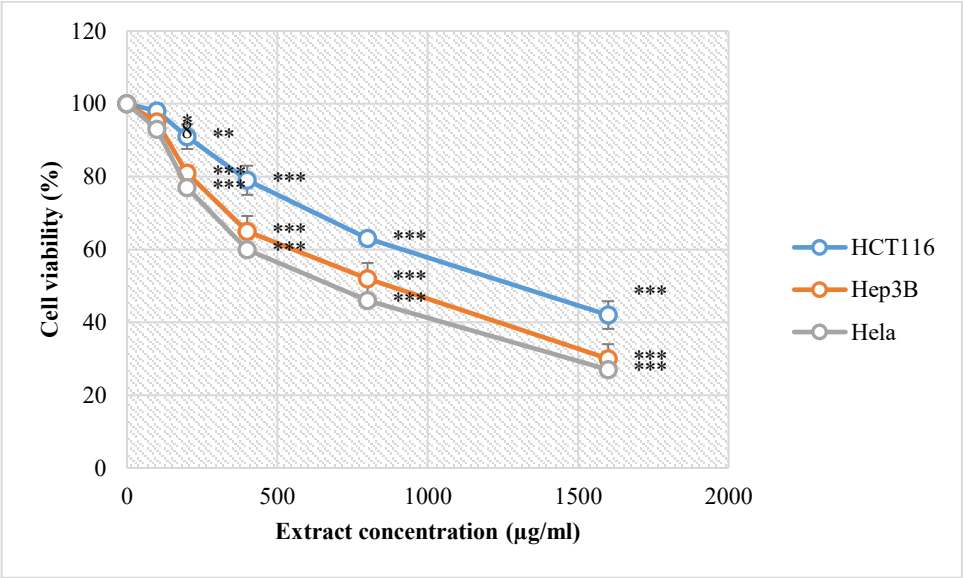



**Figure 2.** Cytotoxic effects of *Rosa canina* fruit extract on human cancer cell lines (HeLa, Hep3B, and HCT116). Cell viability was measured after 24 h treatment using the MTT assay. Data are presented as mean ± SEM from three independent experiments. * $p < 0.05$, ** $p < 0.01$, and ***$p < 0.001$ compared with the control group. **(Prepared by Authors, 2026)**.

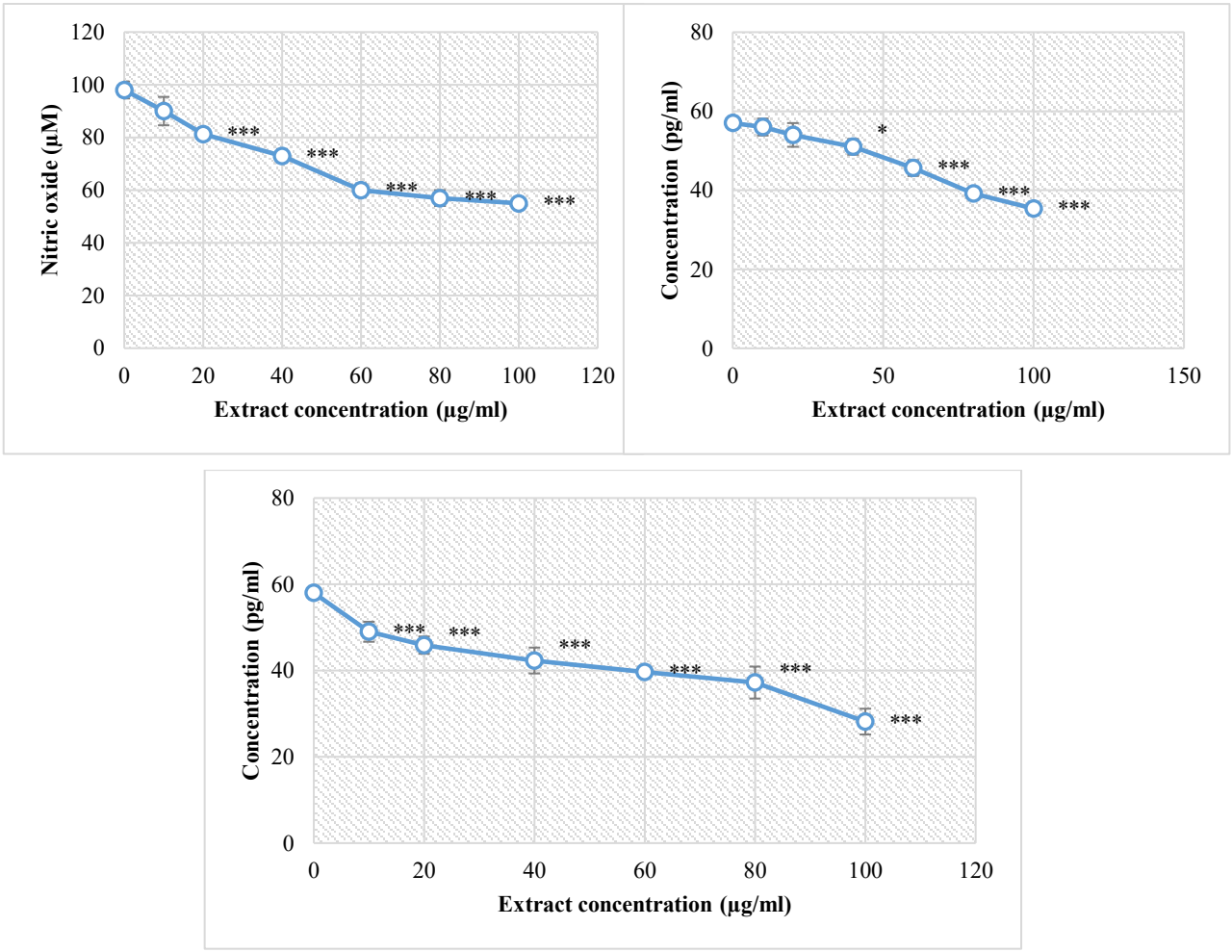



**Figure 3.** Effects of *Rosa canina* extract on (A) nitric oxide, Prostaglandin $E_2$ (B), and (C) tumor necrosis factor-alpha levels in RAW 264.7 cells. Data are shown as mean ± SEM from three independent experiments. * $p < 0.05$, and *** $p < 0.001$ compared with the control group. **(Prepared by Authors, 2026)**.

## 4. Discussion

In the present study, the hydroalcoholic extract of *Rosa canina* fruit exhibited significant antioxidant, cytotoxic (anticancer), and anti-inflammatory activities in vitro. These findings are consistent with accumulating evidence that plant-derived polyphenolic and flavonoid-rich extracts can exert multifaceted effects against cancer and inflammation, mediated through redox regulation, apoptosis induction, and suppression of pro-inflammatory mediators (14). A key finding of this study was the potent DPPH and ABTS radical scavenging activity exhibited by the extract, consistent with its high content of phenolics and flavonoids. *Rosa canina* is well known for its rich phytochemical profile, including vitamin C, carotenoids, polyphenols, and flavonoids, which afford potent antioxidant activity (5).

The link between oxidative stress and carcinogenesis is well documented: an excess of reactive oxygen species (ROS) can lead to DNA damage, lipid peroxidation, and the activation of oncogenic signaling pathways, whereas moderate ROS levels can function as signaling molecules that promote proliferation in cancer cells (15). By neutralizing ROS, antioxidant compounds may prevent some of the early events in tumorigenesis or reduce the oxidative stress burden in cancer cells. Moreover, some anticancer agents exert their cytotoxic effects by further increasing ROS levels beyond a tolerable threshold, driving cancer cells into oxidative stress–mediated cell death (16). Thus, the potent antioxidant activity of *R. canina* extract may contribute to its cytotoxic properties, both by directly protecting normal cells and by modulating ROS homeostasis in cancer cells.

In our experiments, *R. canina* extract significantly reduced the viability of HeLa, Hep3B, and HCT116 cell lines. This is concordant with prior studies: for example, Jiménez et al. observed significant antiproliferative activity of rose hip extracts in Caco-2 colorectal cancer cells, where the antioxidant fractions were the most effective in decreasing viability (17).

Also, in human lung (A549) and prostate (PC-3) cancer cells, *R. canina* extract induced cell cycle arrest, mitochondrial membrane potential loss (MMP), and activation of caspases — key signatures of apoptosis (8). Furthermore, another study has demonstrated the cytotoxicity of *R. canina* extract against WiDr colon cancer cells, where it induced S-phase arrest, mitochondrial depolarization, and downregulation of telomerase expression (hTERT) (18).

Collectively, these findings suggest that *R. canina* may exert anticancer effects through apoptosis induction, the disruption of mitochondrial function, and regulation of cell-cycle progression. Given that cancer is a highly heterogeneous disease, the efficacy of the extract across multiple cell lines (cervical, hepatic, colorectal) underscores a potentially broad mechanism of action. This activity is likely mediated by general stress induction (e.g., oxidative stress, mitochondrial dysfunction) rather than targeting a single oncogene.

Chronic inflammation is a well-established contributor to cancer initiation, promotion, and metastasis. Inflammatory mediators like nitric oxide (NO), prostaglandins (e.g., $PGE_2$), and tumor necrosis factor-alpha (TNF-α) can promote DNA damage, stimulate angiogenesis, and create a microenvironment favorable to tumor progression (19).

In our study, *R. canina* extract inhibited LPS-induced NO production in macrophages and reduced secretion of $PGE_2$ and TNF-α. Many plant polyphenols are shown to modulate NF-κB signaling, inhibit COX-2, or block inducible nitric oxide synthase (iNOS) expression (20).

In the context of cancer, suppressing inflammation can reduce pro-tumorigenic signals, diminish oxidative DNA damage, and hinder tumor-promoting microenvironment changes (21). Thus, the anti-inflammatory activity of *R. canina* strengthens its potential as an adjunct in chemoprevention or therapy.

The observed bioactivities can be rationalized by the synergistic action of its multiple bioactive constituents. Polyphenolic compounds, flavonoids such as quercetin, phenolic acids, and vitamin C, all present in rose hip, may act in concert to modulate multiple signaling axes (22).

For instance, combining *R. canina* extract with chemotherapeutic agents such as cisplatin has shown enhanced anticancer effects, involving suppression of PI3K/Akt/mTOR and NF-κB signaling while simultaneously inducing in autophagy/apoptosis lung cancer models (6).

Despite this promising preclinical result, a recent review emphasizing the potential of *R. canina* noted that mechanistic investigations remain insufficient and inconsistent across studies (5).

A key strength of this study is the integrated assessment across antioxidant, anti-inflammatory, and cytotoxic endpoints, providing a holistic view of the extract's bioactivity. The use of multiple cancer cell lines also adds to generalizability.

The results of the current research align with earlier studies that demonstrate the biological activities of *Rosa canina* extracts against various cancer models (8,14).

In the current study, the hydroalcoholic extract significantly reduced the viability of HeLa, Hep3B, and HCT116 cells in a concentration-dependent manner, suggesting a broad-spectrum antiproliferative effect. Similar observations have been reported in colorectal (14), lung (8), and prostate cancer cell lines (8), where *Rosa canina* extracts inhibited cell growth through induction of apoptosis, cell-cycle arrest, and mitochondrial dysfunction. Furthermore, the marked inhibition of NO, $PGE_2$, and TNF-α production observed in our study is consistent with previous evidence indicating that *Rosa canina*-derived polyphenols suppress inflammation (23).

The strong antioxidant activity further supports the hypothesis that the anticancer and anti-inflammatory properties of *Rosa canina* are closely associated with its high content of phenolic and flavonoid compounds. Our findings are consistent with recent evidence indicating that *Rosa canina* is a rich source of phenolic compounds, flavonoids, carotenoids, and vitamins, all of which possess antioxidant, anti-inflammatory, and anticarcinogenic properties. Notably, Negrean et al.

(2024) highlighted that these bioactive constituents may inhibit tumor growth and modulate inflammatory responses through the regulation of oxidative stress-related pathways (24).

Despite the promising findings of the present study, several limitations must be acknowledged. First, the biological activities were evaluated using a crude hydroalcoholic extract; thus, the specific bioactive compounds responsible for the observed effects were not individually identified. Second, the study was limited to in vitro experiments, and the efficacy and safety of the extract under in vivo conditions remain to be established. Finally, the cellular uptake and intracellular localization of the extract constituents were not investigated, limiting our understanding of their bioavailability and cellular interactions.

Future studies should focus on detailed phytochemical characterization, mechanistic investigations, cellular uptake analyses, and in vivo validation to fully elucidate the therapeutic potential of *Rosa canina* extract.

## 5. Conclusion

Overall, the findings presented here, supported by corroborating literature, indicate that *Rosa canina* fruit extract exhibits potent antioxidant, anti-inflammatory, and anticancer activities under the experimental conditions of this in vitro study. By modulating oxidative stress, inducing apoptosis, and suppressing pro-inflammatory mediators, the extract may exert concerted effects that inhibit cancer cell survival and proliferation. Consequently, further in vivo and mechanistic studies are warranted to fully elucidate and harness its therapeutic potential.

## 6. Declarations

### 6.1 Acknowledgments

We would like to express our sincere gratitude to Kermanshah University of Medical Sciences for its financial support.

### 6.2 Ethical Considerations

The study was approved by the Ethics Committee of Kermanshah University of Medical Sciences (Ethical Code: IR.KUMS.MED.REC.1402.394).

### 6.3 Authors' Contributions

“Conceptualization, resources, supervision, and funding acquisition, D. R.; methodology, investigation, M. P.; writing, J. N; editing the manuscript, H. J; validation, C. J.; formal analysis and data curation, H. N.

### 6.4 Conflict of Interest

The authors declare no conflict of interest.

### 6.5 Fund or Financial Support

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### 6.6 Using Artificial Intelligence Tools (AI Tools)

Artificial intelligence (AI) was not used in the generation, processing, analysis, or manipulation of any experimental data. AI-assisted tools were used solely for language editing and improving the grammar, spelling, and readability of the manuscript. All authors have carefully reviewed and approved the final version of the manuscript and take full responsibility for its content.

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