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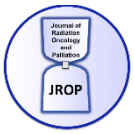

REVIEW ARTICLE **Open Access**

Current Evidence on the Preventive and Therapeutic Potential of Quercetin in Lymphoma

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ABSTRACT

Lymphoma is a general name for malignant diseases of the lymphatic system. Despite substantial advances in chemotherapy, immunotherapy, and targeted therapies, treatment resistance, toxicity, relapse, and refractory disease remain clinically important problems. Studies have shown that plant components may play an important role in the treatment of various diseases. Quercetin is a flavonoid found in various type of plants, and its cytotoxic effects have been demonstrated in many preclinical studies. Despite this, there are very few clinical studies analyzing the effects of quercetin in lymphoid cancers. In this study, a review of studies that have examined the anticancer and therapeutic effects of quercetin in various types of lymphoma has been prepared.

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Introduction

Lymphoma is a general term for malignant diseases of the lymphatic system. Lymphoma represents an important public health problem worldwide because of its substantial incidence, mortality, and contribution to global health loss. According to the Global Burden of Disease Study 2023, non-Hodgkin lymphoma accounted for approximately 632,000 new cases, 282,000 deaths, and 8.29 million disability-adjusted life years globally in 2023.

Hodgkin lymphoma contributed an additional 68,700 new cases, 27,100 deaths, and 1.18 million disability-adjusted life years. These findings highlight lymphoma as a clinically and epidemiologically significant malignancy and underline the need for improved preventive, diagnostic, and therapeutic strategies [1, 2].

The risk factors of lymphoma varies by subtype and may include older age, high BMI, immune dysfunction, autoimmune diseases, immunosuppressive therapy, certain infections, environmental or occupational exposures. Moreover exposure to pesticides, certain chemicals or radiation, and infections including H. pylori, hepatitis C virus, and Epstein–Barr virus, have been associated with increased risk of specific lymphoma subtypes [3].

Chemotherapy is commonly used as an approach in the treatment of non-Hodgkin lymphoma [4]. In recent years, some plant-

derived agents have been shown to have cytotoxic effects on malignant cells [5, 6]. In this context, various studies have shown that quercetin, a flavonoid found in fruits and vegetables, may have antiviral, antiangiogenesis, cell phase changing, antioxidant, and apoptotic effects [7, 8].

Several pathways affected by quercetin have been identified in various malignancies [9]. Although chemotherapy is widely used in the treatment of malignant diseases, including lymphoma, its clinical benefits may be limited by adverse effects and the development of treatment resistance.

Therefore, natural compounds such as quercetin have gained attention as possible supportive agents in cancer prevention and treatment because of their relatively low toxicity and therapeutic potential [10].

Mechanisms of Action of Quercetin

Quercetin can cross cell membranes because of its lipophilic properties and modulate various intracellular signaling pathways. Its biological effects are concentration dependent, with antioxidant and cytoprotective activity at lower concentrations and prooxidant effect at higher concentrations [11].

Quercetin induces G1 phase cell cycle arrest by down regulating cyclin D1/CDK4 and cyclin E/CDK2 and up-regulating p21 expression [12]. It modulates molecular pathways involving cyclins, PI3K/Akt, and mitogen activated protein kinase (MAPK) signaling. Experimental evidence indicates that quercetin can induce p53 phosphorylation and stabilization, a key mechanism involved in its proapoptotic effects [13, 14, 15]

Quercetin may also affect oxidative stress through its electron- or hydrogen-donating properties; however, its oxidative activation to semiquinone and quinone intermediates may contribute to the formation of reactive oxygen species, such as superoxide and H_2O_2, under pro-oxidant conditions [16].

Quercetin can also suppress NF-κB activation, reduce COX-2 expression and TNF-α secretion, decrease cyclin D1 expression, and downregulate Bcl-2 in experimental models [13, 17, 18, 19].

Tyrosine kinases such as JAK and Src kinases are involved in normal cellular signaling pathways. They are essential for cell proliferation and physiological function. In malignant cells, disruption of these pathways can lead to abnormal proliferation and resistance to apoptosis [20].

NF-κB is an important transcription factor for the immune system. It is particularly associated with inflammatory, neurological, and cancerous diseases [21].

Quercetin suppresses proliferation by reducing PI3K/AKT activity. It can inhibit AKT activation and increase PTEN levels. In this way, quercetin can inhibit T-cell lymphoma cell proliferation through PI3K/AKT-related survival pathways [22].

Immun effects of quercetin shon in Figur 1.

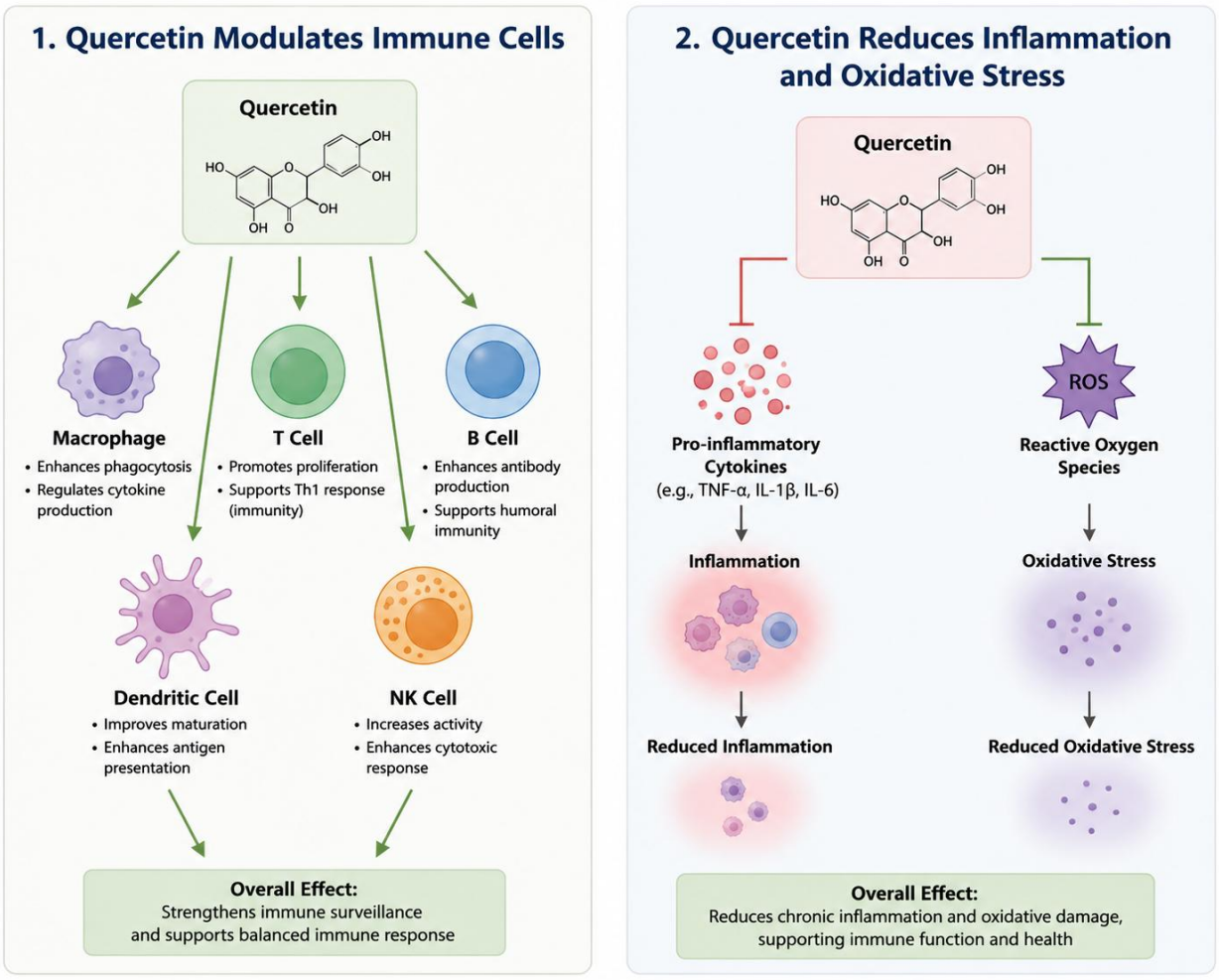

Figure 1. Immun effects of quercetin

Quercetin in Lymphoma Models

In their study on the B-cell lymphoma cell line Namalwa, Fil'chenkov et al. showed that resveratrol combined with quercetin inhibited cell growth and caused time-dependent arrest in the G2/M phase [23]. Quercetin induces cell death through caspase-3 activation by modulating Bcl-2 and Bcl-xL.

Frankenfeld et al. showed that higher intake of flavonoid-containing foods, including quercetin, epicatechins, anthocyanidins, and proanthocyanidins, reduced the risk of lymphoma by 47% [24].

Kawahara et al. found that cyclopamine and quercetin inhibited proliferation and stimulated apoptosis [25].

Jacquemin et al. found that combining quercetin with tumor necrosis factor-related apoptosis-inducing ligand (TRAIL) therapy may be beneficial in NHL.

They found that pretreatment of lymphoma cell lines with 20 µM quercetin for 24 hours reduced resistance to TRAIL-induced cell death by activating caspase-3, caspase-8, and caspase-9 [26].

Rituximab is a chimeric monoclonal antibody developed against the CD20 protein. Li et al. found that the combination of 5 µg/mL rituximab with 20 µM quercetin induced apoptosis more strongly in DLBCL cell lines than rituximab alone. They found that the quercetin–rituximab combination inhibited the p-STAT3 pathway [27].

PI3K/AKT/mTOR and Wnt/β-catenin pathways are involved in aggressive B-cell lymphomas such as primary effusion lymphoma (PEL). These signaling pathways can also be inhibited by quercetin. Granato et al. showed that quercetin was effective in PEL cell lines by increasing the G1-phase cell population and inhibiting PI3K/AKT/mTOR and STAT3 signaling. They also showed that quercetin reduced the release of IL-6 and IL-10 cytokines by inhibiting these pathways. They stated that the cytotoxic effect of bortezomib, a proteasome inhibitor, increased when it was combined with quercetin. Quercetin also increased the surface expression of HLA-DR and calreticulin in malignant cells [28].

Salazar et al. found that HIGD2A gene expression, which is associated with cell survival in hypoxic conditions, was increased in DLBCL and decreased in nodal marginal zone lymphoma (NMZL) [29].

Evaluating the Antitumor Effects of Quercetin in Lymphoma shown in Table 1.

Table 1. Major Studies Evaluating the Antitumor Effects of Quercetin in Lymphoma

Author (Ref)	Model/Disease	Quercetin Intervention	Main Findings
Fil'chenkov et al. [19]	Namalawa B-cell lymphoma cell line	Quercetin + resveratrol	Induced time-dependent G2/M cell cycle arrest and inhibited cell proliferation.
Frankenfeld et al. [20]	Epidemiological study	Dietary intake of flavonols including quercetin	Higher flavonol consumption was associated with a 47% reduction in lymphoma risk.
Kawahara et al. [21]	Lymphoma cell models	Quercetin + cyclopamine	Inhibited cell proliferation and promoted apoptosis.
Jacquemin et al. [22]	Non-Hodgkin lymphoma (NHL) cell lines	20 µM quercetin + TRAIL	Reduced resistance to TRAIL-induced cell death through activation of caspase-3, caspase-8, and caspase-9.
Li et al. [23]	Diffuse large B-cell lymphoma (DLBCL) cell lines	Rituximab (5 µg/mL) + 20 µM quercetin	Induced stronger apoptosis than rituximab alone and inhibited the p-STAT3 signaling pathway.
Granato et al. [24]	Primary effusion lymphoma (PEL) cell lines	Quercetin alone or combined with bortezomib	Increased G1 phase arrest, inhibited PI3K/mTOR signaling and PARP activity, reduced IL-6 and IL-10 secretion, and enhanced proteasome inhibition when combined with bortezomib.
Salazar et al. [25]	DLBCL and nodal marginal zone	Molecular expression analysis	Demonstrated increased HIGD2A expression in DLBCL and decreased expression in NMZL,

Author (Ref)	Model/Disease	Quercetin Intervention	Main Findings
	lymphoma (NMZL)		suggesting a role for hypoxia-related pathways in lymphoma biology.

Quercetin and Burkitt Lymphoma

Another malignant disease of the lymphatic system is Burkitt lymphoma, an aggressive B-cell lymphoma. The endemic or African variant is commonly associated with EBV infection and is mainly seen in children. The sporadic variant is seen outside Africa, whereas the immunodeficiency associated variant is usually seen in individuals with HIV infection or immunosuppression [30, 31]. These variants are generally associated with MYC gene alterations [32]. Treatments such as intensive chemotherapy and rituximab-based immunotherapy are used in Burkitt lymphoma, while stem cell transplantation and radiotherapy may be considered in selected cases [33]. Okamoto et al. found that quercetin inhibited EBV early antigen activation in Raji Burkitt lymphoma cells. Quercetin at doses of 25, 10, and 2.5 μg/mL inhibited early viral antigen activation by 82%, 74%, and 32%, respectively [34]. It has been suggested that quercetin can exert its effect by inhibiting c-Myc expression, the PI3K/AKT/mTOR pathway, and PARP-related apoptotic signaling, and by reducing heat shock protein 70 (HSP70) [35].

Preclinical Studies with Quercetin

Previous studies have shown that quercetin can exhibit beneficial biological effects in many diseases. In recent years, its potential role in the treatment of various malignancies has attracted increasing attention. Experimental studies show that quercetin can exhibit anticancer activity through different molecular mechanisms and can target various steps of tumor development. It has been reported that it can reduce tumor cell proliferation and contribute to tumor volume reduction through effects such as cell-cycle arrest, induction of apoptosis, suppression of angiogenesis, and inhibition of metastatic spread. For example, in leukemia and breast cancer xenograft models, quercetin administered at different doses has been shown to induce cell-cycle arrest, inhibit the AKT/mTOR signaling pathway, and increase programmed cell death [36].

In a study on prostate cancer, quercetin was found to significantly inhibit the in vivo growth of prostate tumors by increasing the expression of thrombospondin-1 (TSP-1), a potent anti-angiogenic factor [37]. Furthermore, a study in HepG2 tumor-bearing BALB/c/nu nude mice reported that quercetin administered at a dose of 10 mg/kg delayed tumor growth, prolonged survival, increased tumor necrosis, and reduced cyclin D1 expression [38]. These findings suggest that quercetin may exhibit multifaceted antitumor effects in different cancer types and could be considered as a potential supportive agent to conventional treatments in the future. However, further preclinical and clinical studies are needed to clarify the relevance of these effects in clinical practice.

Clinical studies with quercetin

In a Phase I clinical trial conducted by Ferry et al., intravenous quercetin was reported to be tolerable in humans, to produce in vivo tyrosine kinase inhibition, and to show biochemical responses suggestive of antitumor activity in some patients. A

significant decrease in CA-125 levels was detected in a patient with ovarian cancer, and a significant decrease in AFP levels was detected in a patient with hepatoma [39].

The prodrug QC12, developed to overcome the bioavailability limitations of quercetin, was evaluated in cancer patients. Therapeutic plasma levels of quercetin were achieved after intravenous administration, whereas oral bioavailability remained insufficient. The researchers recommended further Phase I studies [40].

The US National Cancer Institute lists several active clinical trials involving quercetin. These include studies evaluating the combination of dasatinib and quercetin with CAR-T therapy in patients with relapsed or refractory multiple myeloma, studies investigating combinations of dasatinib, quercetin, fisetin, and temozolomide in previously treated gliomas, and studies on isoquercetin or Kinisoquin for the prevention of thromboembolic events in various cancers. These studies focus on the potential effects of quercetin on the elimination of senescent cells and the regulation of the tumor microenvironment, rather than its direct cytotoxic effects [41, 42].

Preclinical and Clinical Studies Investigating the Anticancer Effects of Quercetin shown in Table 2.

Table 2. Preclinical and Clinical Studies Investigating the Anticancer Effects of Quercetin

Author/Study (Ref)	Study Type	Cancer Model/Population	Quercetin Intervention	Main Findings
Experimental studies [32]	Preclinical (xenograft)	Leukemia and breast cancer xenograft models	Quercetin at various doses	Inhibited the AKT/mTOR signaling pathway, induced cell cycle arrest, increased apoptosis, and reduced tumor growth.
Prostate cancer study [33]	Preclinical (in vivo)	Prostate cancer model	Quercetin administration	Increased thrombospondin-1 (TSP-1) expression, inhibited angiogenesis, and significantly suppressed tumor growth.
BALB/c mouse study [34]	Preclinical (in vivo)	Liver tumor model	Quercetin (150 mg/kg)	Enhanced apoptosis and suppressed tumor progression.
Ferry et al. [35]	Phase I clinical trial	Patients with advanced malignancies	Intravenous quercetin	Demonstrated acceptable safety, tyrosine kinase inhibition, and signs of antitumor activity; reductions in CA-125 and AFP levels were observed in individual patients.

Author/Study (Ref)	Study Type	Cancer Model/Population	Quercetin Intervention	Main Findings
QC12 study [36]	Early-phase clinical study	Cancer patients	Intravenous and oral QC12 (quercetin prodrug)	Achieved therapeutic plasma quercetin concentrations after intravenous administration; oral bioavailability remained inadequate. Further Phase I studies were recommended.
NCI-sponsored trials [37]	Ongoing clinical trials	Multiple myeloma, glioma, and various cancers	Quercetin-based combinations (dasatinib + quercetin ± fisetin; isoquercetin)	

Conclusion

Quercetin shows significant antitumor activity against malignant cells. Quercetin is a naturally occurring flavonoid that exerts its anticancer effects by modulating numerous cellular signaling pathways involved in cancer development, progression, and treatment resistance. Experimental studies have shown that quercetin increases caspase-3 activation while suppressing AKT/mTOR and ERK/MAPK signaling pathways, thereby inducing apoptosis and autophagy in tumor cells. Furthermore, quercetin inhibits tumor cell proliferation and limits tumor growth by causing arrest in different phases of the cell cycle. It may also affect metastatic processes by suppressing tumor vascularization and molecules related to angiogenesis and invasion, such as vascular endothelial growth factor (VEGF) and matrix metalloproteinases (MMPs). In addition, it may target mitochondrial functions, leading to disruption of cellular bioenergetic balance and supporting the activation of intrinsic apoptotic pathways. Current data suggest that quercetin may be evaluated as a chemopreventive agent in cancer prevention and as a supportive therapeutic candidate with a low toxicity profile in cancer treatment. Furthermore, various preclinical studies have shown that quercetin, when used in combination with chemotherapeutic agents and targeted therapies, can enhance antitumor efficacy, reduce treatment resistance, and produce potential synergistic effects. However, comprehensive preclinical studies, as well as well-designed clinical trials, are needed to fully elucidate its mechanisms of action, optimal dosing strategies, and clinical efficacy, particularly in lymphoid malignancies. In conclusion, quercetin may be considered a promising adjuvant or supportive therapy candidate in the treatment of lymphoid malignancies due to its mechanism of action targeting multiple cellular pathways, its relatively low toxicity profile, and its potential for combination with other anticancer agents. Considering the global burden of lymphoma, further research on low-toxicity supportive agents such as quercetin may also contribute to public health by supporting prevention strategies, reducing treatment-related complications, and improving long-term disease control.

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