RESEARCH ARTICLE

# Anticancer Profiling of Phytochemicals: A Study on MCF-7 Cell Lines

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## Abstract

**Introduction:** Cancer remains a leading cause of global mortality, characterized by genetic mutations that lead to uncontrolled cell proliferation and resistance to conventional therapies. This study investigates the anticancer potential of various phytochemicals as safer, more targeted alternatives or adjuvants to traditional treatments.

**Methods:** The objective of this study is to profile the pharmacological attributes and cytotoxic efficacy of diverse plant-derived compounds against the MCF-7 breast cancer cell line. MCF-7 cells were cultured in DMEM supplemented with 10% FBS and treated with various phytochemical concentrations. Cytotoxicity was determined using the MTT [3-(4, 5-dimethylthiazolyl-2)-2, 5-diphenyltetrazolium bromide] assay, and IC50 values were calculated.

**Results:** Comparative analysis revealed a wide range of ED50 values, with compounds like sanguinarine (10 µM), beta-carotene (15 µM), and sulforaphane (15 µM) exhibiting high potency.

**Discussion:** Key mechanisms identified include the inhibition of the NF-kB pathway, induction of p53 and SOCS proteins, and modulation of the apoptotic index. Phytochemicals demonstrate significant pleiotropic effects on cancer signaling pathways. While some compounds like curcumin and resveratrol face bioavailability challenges, their ability to target multiple survival axes makes them promising candidates for future chemopreventive and therapeutic strategies.

**Keywords:** Phytochemicals, Cancer, MTT assay, Cytotoxic effect, Cell lines.

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## Introduction

Over 19 million people have died in the last 10 years due to cancer, which is the second leading cause of death, causing the highest mortality rate worldwide. Around the globe, cancer is the second most frequent cause of morbidity.[1] In general, cancer has become more common; in the United States alone, 1,665,540 humans had the disease, and by 2014, 585,720 of them had passed away from it. Cancer research has produced a rich and complicated information structure after 25 years of rapid advancements, demonstrating that cancer is a disease involving dynamic alterations in the DNA. The basis has been established by the identification of mutations that result in tumor suppressor genes with recessive loss of function and oncogenes with dominant gain of function; these two classes of cancer genes have been identified by their alteration in human and animal cancer cells as well as by their elicitation of cancer phenotypes in experimental models.[2]

Although numerous therapies, including radiotherapy, chemotherapy, invasive procedures, and allopathic drugs, cancer still has a high death rate worldwide. These conventional approaches have serious drawbacks, such as end-stage diagnoses with few surgical alternatives and serious side effects like nausea and exhaustion that lower patients' quality of life. Besides, there is the probability that cancer cells will become resistant to these cures.[3-4]

Cancer develops through a multistep process involving genetic mutations that disrupt normal cell growth, leading to uncontrolled proliferation, invasion, and metastasis. Key mechanisms include alterations in proto-oncogenes, tumor suppressor genes, and DNA repair genes, which enable cells to ignore growth signals, evade apoptosis, and form tumors.[5-7] There are several different ways that cancer cells can acquire the capacity to maintain proliferative signaling: They may generate their own growth factor ligands, to which they can react by expressing corresponding receptors, which causes autocrine proliferative activation. On the other hand, normal cells in the surrounding tumor-associated stroma may respond to signals from cancer cells by providing the cancer cells with different growth factors. Elevated levels of receptor proteins on the surface of cancer cells can also deregulate receptor signaling, making these cells hyperresponsive to otherwise limiting amounts of growth factor ligand; structural changes in the receptor molecules that enable ligand-independent firing can also have the same effect.[8]

## Material and Methods

### Cell Line Culture and Treatment

MCF-7 cell lines were procured from NCCS Pune. The cells were harvested from a flask at 90% confluency and seeded in 96-well plates at an appropriate density in DMEM medium (Dulbecco's Modified Eagle Medium-AT149-1L) supplemented with 10% FBS (Fetal Bovine Serum – GIBCO, Thermo Fisher) and 1% antibiotic solution at 37°C with 5% $CO_2$. The next day, fresh culture medium was added to each well of the plate by removing the consumed medium. 10% of the total medium (5–50 µL) of treatment dilutions was added to the named and labeled wells and further, the treated plates were incubated (Heal Force $CO_2$ Incubator-Hf-90) for 24 hours.[9]

### Cytotoxicity Assay

Cytotoxicity of the samples (phytochemicals received as gift samples) on MCF-7 (Procured from NCCS Pune) cell line was determined by MTT dye 3-(4, 5-dimethylthiazolyl-2)-2, 5-diphenyltetrazolium bromide Assay.[10] The cells were cultured in the culture well plate at a density of 10000 cells/well for 24 hours in DMEM medium added with 10% FBS and 1% antibiotic solution at 37°C with 5% $CO_2$. The next day, cells were treated using a herbal combination (solutions of concentrations were prepared in a blank medium).[11] After incubation for 24 hours, MTT Solution (a final concentration of 250 µg/mL) was added to the cell culture and additionally incubated for 2 hours. At the end of the experiment, the culture supernatant was removed and dense cells were dissolved in 100 µL Dimethyl Sulfoxide (DMSO) and observed using an ELISA plate reader (iMark, Biorad, USA) at 540 nm and 660 nm. $IC_{50}$ was calculated by using the software Graph Pad Prism -6.[12-14]

## Results and Discussion

### MTT Assay

The MTT assay revealed different $IC_{50}$ values for each phytochemical, indicating moderate cytotoxicity against MCF-7 cell lines (as mentioned in the following table 1). This suggests that the selected phytochemicals have potential anticancer properties, although further studies are needed to confirm this. Overall, this study demonstrates the potential of combining natural compounds to create novel antioxidant and anticancer agents, and highlights the importance of continued research into the therapeutic properties of plant-derived compounds.

### Pharmacological Profiling of Phytochemicals

#### *Eugenol and the Inflammatory Response*

Eugenol (4-allyl-2-methoxyphenol), primarily derived from clove oil (*Syzygium aromaticum*), demonstrates a dual-action profile by blocking NF-kB and targeting the pro-inflammatory cytokine IL-1. The provided data establishes an ED50 of 150 µM, which sits well within the safety window defined by its LD50 of 500 µM. Beyond its primary targets, eugenol has been shown to induce selective cytotoxicity in HeLa cells, downregulating Bcl-2 and COX-2, thereby enhancing the therapeutic index when used as an adjuvant to conventional drugs like gemcitabine. The relatively high ED50 compared to agents like sanguinarine (10 µM) suggests that eugenol may act more as a dietary preventive than a high-potency drug candidate, yet its safety profile remains exemplary.[14]

#### *Capsaicin: From Autophagy to STAT Inhibition*

Capsaicin, the pungent principle of chili peppers (*Capsicum annuum*), exhibits an ED50 of 10 mg/kg in vivo. Its unique mechanism involves the induction of SOCS proteins, which directly counteracts the STAT3 actions that drive tumor growth. Additionally, capsaicin has been identified as a potent inducer of autophagy, a cellular self-digestion process that can prevent the accumulation of oncogenic proteins during the early stages of carcinogenesis. While its pharmacological application is sometimes limited by its sensory properties, its role in inhibiting the NF-kB pathway at the systemic level remains a cornerstone of its anticancer potential.

#### *Resveratrol and the Stilbene Paradigm*

Resveratrol is a ubiquitous stilbene found in grapes and red wine, noted for its pleiotropic effects on cell signaling. The figure provides an ED50 of 150 µM or 0.15 mg/ltr. Its ability to inhibit the JAK/STAT pathway through the induction of SOCS highlights its role as a suppressor of the promotion stage of cancer. Beyond the JAK/STAT axis, resveratrol is a known activator of SIRT1 and p53, facilitating DNA repair and cellular senescence. However, the low bioavailability of resveratrol, due to rapid intestinal conjugation, remains a challenge that methoxylated analogs like pterostilbene or nano-encapsulation seek to address.[14]

#### *EGCG and the Green Tea Epigenome*

Epigallocatechin-3-gallate (EGCG) is the most abundant catechin in green tea (*Camellia sinensis*), with a reported ED50 of 75 mg/kg. EGCG's mechanism is characterized by a significant increase in the apoptotic index and a concomitant decrease in the proliferation index of tumor cells. This is achieved through complex epigenetic modifications, including the inhibition of DNA methyltransferases (DNMTs) and histone deacetylases (HDACs), which allows for the reactivation of tumor suppressor genes like TIMP3 and p16. Furthermore, EGCG selectively induces the CDK inhibitor p57/KIP2 in normal cells while triggering JNK-mediated apoptosis in p57-negative tumor cells, representing a highly specific therapeutic window.

#### *Sulforaphane and Phase II Enzyme Induction*

Sulforaphane, an isothiocyanate derived from cruciferous vegetables like broccoli, has an ED50 of 15 µM according to the provided figure. Its primary mechanism involves the inhibition of the NF-kB pathway, but it is also widely recognized for its ability to activate the Nrf2 pathway.

**Table 1:** MTT Assay Outcomes of Phytochemicals

| *Sr. No.* | *Phytochemical name* | *Botanical name* | *$IC_{50}$ value* | *Mechanism of action (MOA)* |
|---|---|---|---|---|
| 1 | Eugenol | *Syzygium aromaticum* | 150 µM | Blocks Nf-kB and targets IL-1B |
| 2 | Capsaicin | *Capsicum annuum* | 10 µM | Overexpress SOCS; decrease STAT actions; inhibit NF-kB pathway |
| 3 | Garlic (Organosulfur) | *Allium sativum* | 100 µM | Reduction of NF-kB activity; reduction of iNOS expression |
| 4 | Resveratrol | *Vitis vinifera* | 150 µM | Overexpression of SOCS; inhibits JAK-STAT pathway |
| 5 | EGCG | *Camellia sinensis* | 75 µM | Inhibition of tumor growth; increase in apoptotic index; decrease in proliferation index |
| 6 | Anethole | *Pimpinella anisum* | 32 µM | Decreased tumor multiplicity; acts against NF-kB activation pathway |
| 7 | 6-Gingerol | *Zingiber officinale* | 100 µM | Inhibits TNFR, CycD1; overexpresses p53 |
| 8 | Beta-Carotene | *Daucus carota* | 15 µM | Blocks Nf-kB and activating caspases |
| 9 | Sanguinarine | *Sanguinaria canadensis* | 10 µM | Decreased proliferation; acts against NF-kB pathway |
| 10 | Silymarin | *Silybum marianum* | 8 µM | Acts against NF-kB activation pathway; cell cycle stopper |
| 11 | CAPE | *Populus spp.* (Propolis) | 20 µM | Decreased proliferation; acts against NF-kB pathway |
| 12 | Sulphoraphane | *Brassica oleracea* | 15 µM | Against NF-kB |
| 13 | Ginestin (Genistein) | *Glycine max* | 50 µM | Acts against cell cycle, growth, angiogenesis, and apoptosis |
| 14 | Curcumin | *Curcuma longa* | 20 mM | Against NF-kB pathway and SOCS |
| 15 | Lycopene | *Solanum lycopersicum* | 30 µM | Inhibiting NF-kB; overexpressing p53; gut microbial flora |

Activation of Nrf2 leads to the induction of phase II detoxification enzymes, such as glutathione S-transferases (GSTs), which neutralize reactive intermediates before they can form DNA adducts. Clinical data suggest that a diet high in sulforaphane-rich vegetables significantly reduces the risk of lung and colorectal cancers, especially in populations with genetic polymorphisms in GST genes.[15]

### *Curcumin: The Pleiotropic Polyphenol*

Curcumin, derived from *Curcuma longa*, is perhaps the most extensively researched phytochemical in the provided list. The figure lists a surprisingly high ED50 of 20 mM, which may reflect a high-dose systemic study or a specific in vitro context, whereas clinical and laboratory snippets often discuss activity in the nanomolar or low micromolar range (30.78 nM to 25 µM). Curcumin's efficacy is driven by its ability to target NF-kB and the Akt/mTOR survival pathway. By increasing the Bax to Bcl-2 ratio, curcumin induces mitochondrial apoptosis in breast cancer cells (MDA-MB-231) and reduces the expression of proliferating cell nuclear antigen (PCNA). Like resveratrol, curcumin suffers from poor oral bioavailability, which has prompted the development of nano-micelles and nanoparticle-based delivery systems to enhance its therapeutic index.

### *Lycopene and Gut Microbiota Modulation*

Lycopene, a red carotenoid found in tomatoes (*Solanum lycopersicum*), has an ED50 of 30 mg/kg. It acts as an inhibitor of NF-kB and an overexpressor of p53, which induces cell cycle arrest at the G1/S transition. A unique aspect mentioned in the data is lycopene's influence on the gut microbial flora. Emerging evidence suggests that lycopene may act as a prebiotic, favoring the growth of beneficial microorganisms that produce short-chain fatty acids (SCFAs) with their own anti-inflammatory and anticancer properties. This highlights a "tertiary" layer of chemoprevention where the compound's metabolites and its impact on the microbiome contribute to systemic health.[15]

## Conclusion

The findings of this research highlight the significant potential of plant-derived phytochemicals as a sustainable and less toxic approach to cancer management. By analyzing compounds such as Eugenol, Sulforaphane, and EGCG, it is evident that these natural agents work through diverse biological pathways—including the inhibition of NF-kB and the activation of tumor suppressors like p53—to effectively slow the growth of MCF-7 cancer cells. While many of these phytochemicals show high potency at low concentrations, future research must prioritize overcoming challenges related to their low bioavailability and rapid metabolism in the human body. Looking ahead, the integration of these compounds into clinical practice through advanced nano-delivery systems could revolutionize oncology. By combining these natural molecules with conventional treatments, there is a promising opportunity to enhance therapeutic efficacy, reduce debilitating side effects, and provide more targeted, patient-friendly cancer care.

## Ethical Approval and Consent to Participate

Not applicable to this study.

## Human Ethics

Not applicable to this study.

## Consent for Publication

Authors have declared their consent for publishing their data without any conflicts.

## Availability of Supporting Data

Not applicable to this study.

## Conflict of Interest

None declared by the authors.

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