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Anticancer Phytochemicals from Selected Indian Ethnomedicinal Plants: Mechanisms and Translational Perspectives

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Abstract

Cancer remains one of the leading causes of morbidity and mortality worldwide, posing a major challenge to healthcare systems despite significant advances in diagnosis and treatment. Therefore, there remains a need to identify more effective and less toxic therapeutic agents. India has one of the world's richest ethnomedicinal tradition which involves Ayurveda, Siddha, Unani and other indigenous system of medicine that have been long using medicinal plants for chronic diseases management and disease control, particularly cancer. Selected ethnomedicinal plants of significance from the traditional and pharmaceutical perspectives like *Curcuma longa, Withania somnifera, Tinospora cordifolia, Rubia cordifolia, Camellia sinensis, Vitis vinifera, Ocimum sanctum* and *Nigella sativa*, along with their key phytochemicals such as curcumin, withaferin A, immunomodulatory diterpenoids, anthraquinones, epigallocatechin-3-gallate (EGCG), resveratrol, ursolic acid, and thymoquinone are reviewed. The present evidence suggests that these phytochemicals may have anticancer activity via multiple molecular targets such as nuclear factor-kappa B (NF-κB), phosphatidylinositol-3-kinase/protein kinase B/mammalian target of rapamycin (PI3K/Akt/mTOR), p53-mediated apoptotic pathway, oxidative stress signalling, angiogenesis, metastasis, and immune-inflammatory response. They are structurally very diverse but remarkably convergent, and some share common oncogenic signalling networks, which may indicate a multi-targeted approach to cancer prevention and therapy. A comparative evidence synthesis provides insight into shared molecular pathways and therapeutic opportunities, while the mechanistic framework and clinical translation perspective highlight their translational relevance. Their potential as anticancer drugs has been demonstrated in the laboratory, but their use in the clinic is hampered by low bioavailability,

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inadequate pharmacokinetic optimization, lack of standardization and lack of large-scale clinical trials. In the future, the development of formulation technologies, network pharmacology, clinical evaluation guided by the use of biomarkers, and more comprehensive clinical evaluation can help to translate ethnomedicinal phytochemicals into evidence-based oncology. Collectively, Indian ethnomedicinal plants represent promising sources of bioactive compounds for future anticancer drug discovery and development.

Keywords: Ethnomedicinal plants, Anticancer phytochemicals, Network pharmacology, Molecular mechanisms, and Translational oncology

1. Introduction

Cancer is still considered one of the most important causes of morbidity and mortality and is a significant socioeconomic burden on health care systems around the world. Recent estimates suggest that there were about 20 million new cases of cancer and 9.7 million cancer deaths reported worldwide during 2022 and that cancer incidence is expected to rise dramatically over the next few decades because of population growth, aging, urbanisation and lifestyle-associated risk factors. Although significant strides have been made in early diagnosis and therapeutic interventions, cancer continues to be a significant public health problem due to the heterogeneity of the disease, the metastatic nature of the disease, drug resistance and the toxicities associated with therapy (Bray et al., 2024).

Current treatment for cancer is mainly through surgery, radiotherapy, chemotherapy, targeted drugs and immunotherapy. These strategies have clearly enhanced survival rates in some cancers, but the side effects, expensive treatments, low adherence with treatment protocols and treatment resistance are limiting their use. However, many chemotherapeutic agents are not very specific to cancer cells and therefore affect normal tissue, causing high incidence of severe systemic toxic effects. Moreover, resistance to treatment due to changes in the apoptotic pathway, activation of alternative survival pathways, increased drug efflux, and genomic instability are common events that limit the long-term effectiveness of treatment. To overcome these limitations, an increased demand for safe, multiple-target, and effective therapeutic agents from natural sources has been observed (Aggarwal et al., 2003; Singh et al., 2015).

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The use of natural products has been a key strategy in the discovery of anticancer drugs in the past. Medicinal plants are the source of several clinically important anticancer agents that still play an important role in modern oncology, such as vincristine and vinblastine from *Catharanthus roseus*, paclitaxel from Taxus species and camptothecin derivatives from *Camptotheca acuminata.* These achievements have reignited both scientific and technological interest in traditional medicinal systems as potential sources of new bioactive compounds that can be used to regulate several molecular pathways that play a role in cancer.

India has one of the richest ethnomedicinal heritages in the world, which includes Ayurveda, Siddha, Unani and various aboriginal folk health systems. All of these systems employ thousands of species of medicinal plants to prevent and treat a variety of diseases. A systematic ethnopharmacological survey of the Himalayan region revealed 42 cancer related species from India which accounted for the highest national contribution among the surveyed regions and 64 species of medicinal plants in total of 59 genera and 37 families (Tariq et al., 2017). A variety of parts of the plant have been used in traditional Indian medicine, and several leaves, roots, bark and whole plant preparations have been found to have measurable anticancer activity in modern pharmacological studies.

The anticancer efficacy of medicinal plants is due to their wide array of phytochemicals such as polyphenols, flavonoids, alkaloids, terpenoids, steroidal lactones, quinones, catechins and triterpenoids. They have anticancer activity by targeting several cellular and molecular processes including nuclear factor-kappa B (NF-κB), phosphatidylinositol-3-kinase/protein kinase B/mammalian target of rapamycin (PI3K/Akt/mTOR), signal transducer and activator of transcription 3 (STAT3), reactive oxygen species (ROS), angiogenic pathways and apoptotic regulators like p53. Many phytochemicals have pleiotropic activity, meaning that they act on multiple molecular targets and regulate interconnected signalling networks, enabling their simultaneous action on multiple processes involved in tumour initiation, proliferation, angiogenesis, invasion, metastasis and resistance to therapy (Aggarwal et al., 2003; Grover et al., 2010; Majdalawieh et al., 2017).

Some ethnomedicinally important medicinal plants of India are highlighted in the present review such as *Curcuma longa, Withania somnifera, Tinospora cordifolia, Rubia cordifolia, Camellia*

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sinensis, Vitis vinifera, Ocimum sanctum and *Nigella sativa.* Their key anticancer phytochemicals, mode of action, experimental findings, converging signalling pathways and translational barriers have been given special consideration. This review discusses ethnomedicinal evidences and mechanistic studies of Indian phytochemical sources as potential source for future anticancer drug discovery and development.

2. Major Molecular Targets of Anticancer Ethnomedicinal Phytochemicals

Phytochemicals found in ethnomedicinal plants exert anticancer activity by modulating various cellular signalling pathways involved in tumour initiation, proliferation, survival, angiogenesis, invasion, metastasis and chemoresistance. Conventional chemotherapeutic drugs usually target only one specific molecular network, whereas plants contain bioactive compounds that typically activate a plethora of different molecular networks, also interconnected. The targets studied most include nuclear factor-kappa B (NF-κB), phosphatidylinositol-3-kinase/protein kinase B/mammalian target of rapamycin (PI3K/Akt/mTOR), p53-mediated apoptotic pathways, oxidative stress signalling, angiogenesis and metastasis-associated pathways, and immune-inflammatory regulators.

2.1 NF-κB Signalling Pathway

NF-κB is a transcription factor which is also involved in regulating inflammation, cell proliferation, apoptosis, angiogenesis and immune responses in an important way. It has been found that NF-κB is constitutively activated in a wide variety of malignancies, such as breast, colorectal, pancreatic, prostate and haematological cancer. Aberrant NF-κB signalling has been shown to stimulate the expression of genes involved in cell survival (Bcl-2, Bcl-xL), cell proliferation (cyclin D1), angiogenesis (VEGF), invasion (MMP-9) and inflammatory mediators (TNF-α, IL-6) that promote tumour progression (Aggarwal et al., 2004). Some of the phytochemicals mentioned in this review such as curcumin, withaferin A and resveratrol have anticancer activities as they have been shown to inhibit NF-κB activation, making the tumour cells less viable and less metastatic.

2.2 PI3K/Akt/mTOR Signalling Pathway

One of the most common aberrantly activated pathways in human cancers is the PI3K/Akt/mTOR signalling pathway, which helps regulate cell growth, metabolism, proliferation and survival. Once activated, PI3K phosphorylates Akt, resulting in the activation of mTOR and increased

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protein synthesis, cell growth and resistance to apoptosis. Continued activation of this pathway is implicated in tumourigenesis, metastasis and therapeutic resistance (Porta et al., 2014). The phytochemicals known as ursolic acid, thymoquinone, and curcumin have been shown to inhibit the PI3K/Akt/mTOR pathway, thereby suppressing tumour growth and promoting apoptotic cell death.

2.3 p53-Mediated Apoptotic Pathway

One reason p53 is so important for preserving genomic stability, it has been dubbed the "guardian of the genome". In response to DNA damage or cellular stress, p53 induces cell-cycle arrest, DNA repair, senescence, or apoptosis depending on the cellular context. In about 50% of all human cancers, p53 is mutated or rendered inactive, and is closely linked to poor prognosis and resistance to treatment (Levine, 2020). Many plant-derived compounds such as resveratrol, epigallocatechin-3-gallate (EGCG) and thymoquinone have been demonstrated to activate p53-dependent signalling pathways, leading to activation of caspases, mitochondrial dysfunction and apoptotic cell death in cancer cells.

2.4 Oxidative Stress and Reactive Oxygen Species (ROS)

Reactive oxygen species have opposing roles in cancer. Moderate ROS levels may have a role in tumour initiation and progression by causing DNA damage, genomic instability and activation of oncogenic pathways. Conversely, however, is that if ROS builds up too much, it can lead to oxidative stress-induced apoptosis and the growth of tumours (Moloney & Cotter, 2018). There are several phytochemicals that have a redox modulating activity, both re-establishing the balance of oxidative cellular environment and promoting ROS production to a cytotoxic concentration within malignant cells. Curcumin, EGCG, withaferin A and thymoquinone have all been reported to modulate ROS-dependent pathways involved in their anticancer properties.

2.5 Angiogenesis and Metastasis

Tumours larger than a few millimetres require angiogenesis to sustain growth and facilitate metastatic dissemination. This process is controlled by a process called vascular endothelial growth factor (VEGF) and is often over-expressed in aggressive cancers. Worldwide, the spread of malignant cells to other parts of the body (metastasis) is the main cause of cancer-related deaths. VEGF mediating signalling, matrix metalloproteinases (MMPs), epithelial-mesenchymal transition (EMT), and other

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pathways that promote tumour invasion are important therapeutic targets (Hanahan, 2022). Experimental studies show anti-angiogenic and anti-metastatic activities for anthraquinone-containing compounds isolated from *Rubia cordifolia*.

2.6 Immune Modulation and Inflammation

Chronic inflammation is now known to be a major component of cancer and to play a role in the onset, development, new blood vessel growth and immune system evasion of the tumour. The tumour microenvironment is a complex interplay involving inflammatory mediators, immune cells, stromal cells, and tumour cells. Modulating immune response and inflammatory signalling pathways has become a key approach to cancer prevention and treatment (Hanahan, 2022). The ethno-medicinal plants, e.g., *Tinospora cordifolia,* possess immunomodulatory diterpenoids, polysaccharides which boost the activity of macrophages, regulate cytokine production and aid antitumour immune responses. Altogether, these molecular targets are the major signalling pathways by which anticancer phytochemicals are able to exert these therapeutic effects. Many phytochemicals share some common pathways of action while many share common chemical structures, and they are all derived from a variety of plant sources, indicating a shared mechanism of action. The convergence yields a new integrated mechanistic framework (Figure 1), which shows that ethnomedicinal plants could be a source of multi-targeted anticancer agents.

3. Convergent Mechanisms of Anticancer Phytochemicals from Indian Ethnomedicinal Plants

The phytochemicals mentioned in this review vary greatly in their chemical structure, natural source, and traditional uses but have converged on a relatively limited number of key molecular pathways of carcinogenesis. This convergence implies that various ethnomedicinal plants have anticancer effects that could be mediated by similar mechanistic pathways or networks, instead of through specific molecular targets. Multi-target activity is one of the best key features of phytochemicals over many conventional anticancer drugs and offers strong arguments for further exploration as therapeutic and chemopreventive agents.

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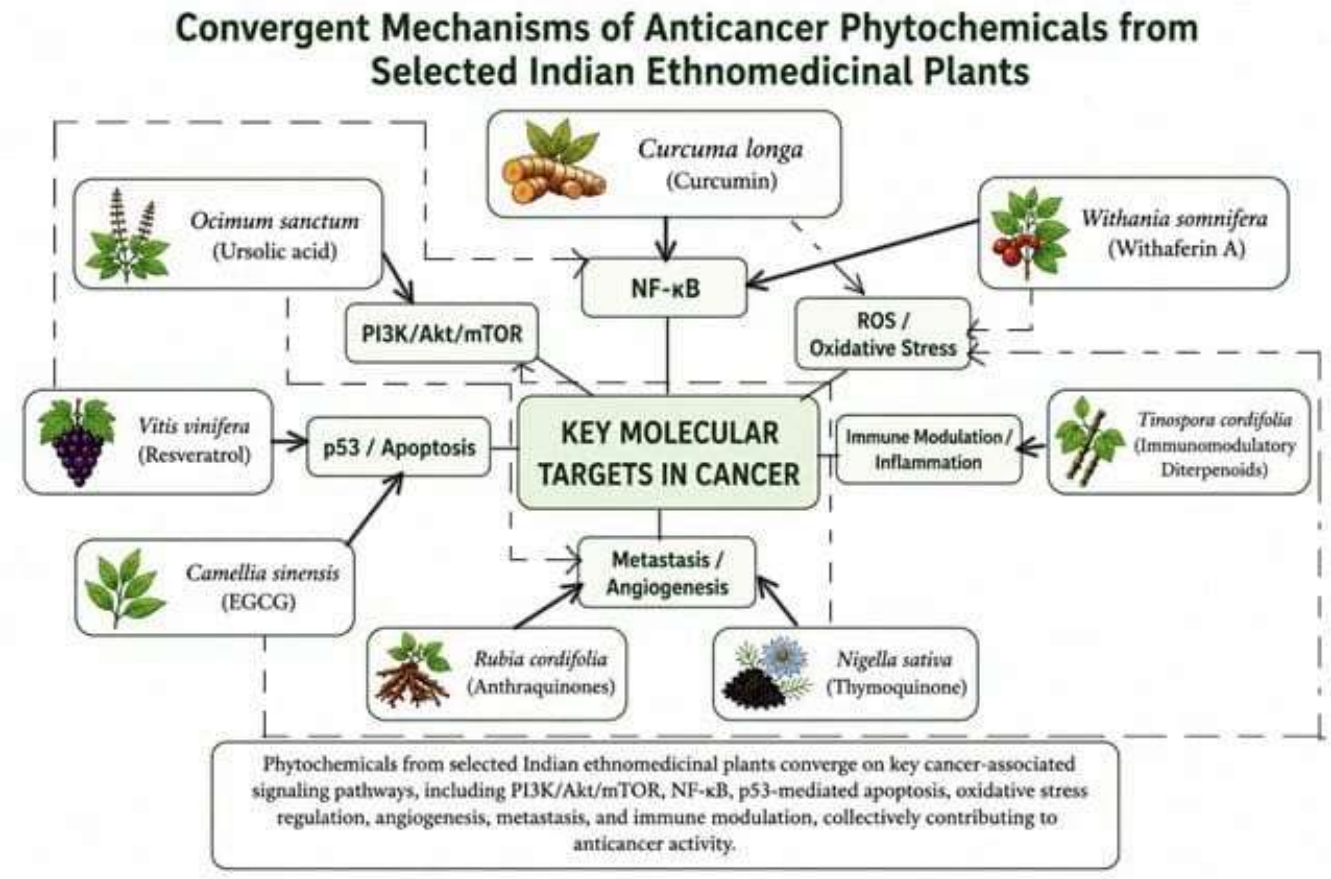
Figure 1. Convergent Molecular Mechanisms of Anticancer Phytochemicals from Selected Indian Ethnomedicinal Plants



Solid arrows indicate primary molecular targets, whereas dashed arrows indicate secondary or indirect molecular interactions

Figure 1 shows the main signalling pathways that are typically targeted by some of these phytochemicals from Indian ethnomedicinal plants. Regardless of the structures of these compounds, they can consistently affect several pathways related to cell proliferation, apoptosis, oxidative stress, angiogenesis, metastasis, and immune regulation.

The anticancer activity of phytochemicals of selected Indian ethnomedicinal plants is exhibited by targeting various cancer associated signalling pathways such as PI3K/Akt/mTOR, NF-κB, p53-mediated apoptosis, regulation of oxidative stress, angiogenesis, metastasis and immune modulation.

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Curcumin from *Curcuma longa* (Turmeric)

The main polyphenolic pigment of turmeric rhizome, curcumin, is one of the most widely studied phytochemicals in oncology. Aggarwal and co-workers showed that curcumin inhibited proliferation of various tumour cell lines by downregulating the transcription factor NF-κB, AP-1 and Egr-1, decreasing expression of COX-2, MMP-9, uPA and cyclin D1, and inhibiting growth factor receptors like EGFR and HER2 (Aggarwal et al., 2003). Curcumin has been found to induce apoptosis in breast cancer cells by generating high levels of ROS, and to inhibit invasion in hepatocellular carcinoma by suppression of heat-shock protein 70 (HSP70) (Tan & Norhaizan, 2019). The main translational challenge for curcumin is its limited water solubility and rapid metabolism in the body, which is being actively studied to make nanoparticles.

Withaferin A from *Withania somnifera* (Ashwagandha)

Ashwagandha or "Indian winter cherry" is the herb for *Withania somnifera,* which produces the steroidal lactone Withaferin A, one of its key active withanolides. In vitro cytotoxic activity of root, stem and leaf extracts of *W. somnifera* has been reported by Yadav et al., (2010) against the various human cancer cell lines, which supports its ethnomedicinal use as an anticancer agent. On the mechanistic level, withaferin A has been demonstrated to inhibit the NEMO/IKKβ complex leading to suppression of NF-κB activation (Grover et al., 2010) and to be an electrophilic modifier of cysteine residues in the activation domain of kinases, thereby providing a mechanism for simultaneous inhibition of NF-κB, PI3K/Akt/mTOR and MAPK/ERK signalling (Bourgeois et al., 2011). Other effects reported are inhibition of estrogen receptor-α in breast cancer cells (Hahm et al., 2011) and interference with the assembly of vimentin filaments, which is believed to play a role in anti-metastatic activity (Kambir et al., 2012).

Tinospora cordifolia (Guduchi)

Tinospora cordifolia, a significant rasayana (rejuvenative herb) in Ayurveda has long been appreciated for its immunomodulatory and health promoting properties. Most of its anticancer properties have been attributed to bioactive clerodane diterpenoids, diterpene glycosides and polysaccharides, which modulate immune responses and inflammatory pathways. Experimental investigations have shown that *T. cordifolia* improves the function of macrophages and lymphocytes,

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which consequently enhances host anti-tumour immune responses (Bala et al., 2015). This immunoregulatory effect is different from the direct cytotoxic effects of some compounds such as curcumin and withaferin A, indicating a complementary role for these compounds in cancer management.

Anthraquinone from *Rubia cordifolia* (Manjishta) and Other Documented Species

Rubia cordifolia L. (Indian madder, Manjishta) is a medicinal plant well recognized in Ayurveda where it is used for blood cleansing, inflammatory disorders and chronic diseases. An anticancer activity of *R. cordifolia* has been mostly attributed to anthraquinones like purpurin, munjistin, alizarin and derivatives of the quinones. Anthraquinone containing extracts also have demonstrated cytotoxic against multiple cancer cell lines such as HeLa, MCF-7, and PA-1 cells (Tariq et al., 2017; Itokawa et al., 1993). Experimental studies have demonstrated that the *R. cordifolia* extracts cause apoptosis via reactive oxygen species-mediated pathway and activation of caspase-dependent pathway (Shilpa et al., 2012). The anti-proliferative activity of phytochemically characterized *R. cordifolia* extracts against human cancer cell lines has also been reported, indicating their therapeutic potential recently (Jadhav et al., 2022). Besides, anthraquinones extracted from *R. cordifolia* have been reported to modulate various cancer-related signalling pathways such as NF-κB, Wnt, Notch, and Myc, and are also anti-metastatic and anti-angiogenic by regulating matrix metalloproteinases (MMPs) and tumour associated signalling networks (Balachandran et al., 2021). Overall, the results indicated that *R. cordifolia* could be a promising plant source for phytochemicals with multi-targeted anticancer activity, which is worthy to be explored in preclinical and clinical studies.

Epigallocatechin Gallate from *Camellia sinensis* (Green Tea)

Camellia sinensis, consumed across India as a daily beverage and employed in traditional health practices for general wellbeing, is the principal dietary source of epigallocatechin gallate (EGCG), its most abundant and pharmacologically active catechin. Early mechanistic evidence reported that EGCG potently inhibited growth of the prostate cancer cell lines LNCaP, PC-3, and DU145, with cytotoxicity proceeding through apoptotic cell death evidenced by nuclear morphological changes and DNA fragmentation (Paschka et al., as cited in Da Silva Pinto, 2013). EGCG additionally interferes with urokinase-type plasminogen activator activity, a protease implicated in tumour invasion. In breast

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cancer models, EGCG and related catechins (EGC, ECG) reduce proliferation in vitro and decrease tumour growth in rodent models, and synergize with tamoxifen against estrogen receptor-negative breast cancer cells (Da Silva Pinto, 2013). More recent work has also implicated EGCG-mediated inhibition of CYP17A1, a key enzyme in androgen biosynthesis, as a contributing mechanism in prostate cancer.

Resveratrol from *Vitis vinifera* (Grape)

Resveratrol is a naturally occurring stilbene phytoalexin concentrated in grape (*V. vinifera*) skins, and is traditionally believed to have benefits for cardiovascular health. Singh et al. summarized in a detailed review the chemopreventive and therapeutic potential of resveratrol in various types of cancer such as breast, colorectal, liver, prostate and lung cancers. Mechanistically, resveratrol has anticancer properties by affecting oxidative stress, inducing cell-cycle arrest and apoptosis, inhibiting NF-κB signalling and regulating genes associated with proliferation and survival (Varoni et al., 2016). Furthermore, extracts from *V. vinifera* and resveratrol have been shown to have anti-migratory, anti-invasive, and anti-metastatic effects in tumour cells, mechanisms that involve modulation of inflammatory signalling pathways, inhibition of matrix metalloproteinases (MMPs), and regulation of the tumour microenvironment (Avtanski & Poretsky, 2018; Varoni et al., 2016).

Ursolic Acid from *Ocimum sanctum* (Tulsi)

Ocimum sanctum (Tulsi, Holy Basil) is an important rasayana herb that is regarded as sacred in Ayurveda to be used for its immunomodulatory, adaptogenic, and rejuvenating properties. Phytochemical studies have revealed that ursolic acid, in addition to eugenol and rosmarinic acid, are among the principal bioactive constituents in the leaves (Joseph & Nair, 2013). Ursolic acid, a pentacyclic triterpenoid, has broad-spectrum anticancer activity, which is mediated by modulation of multiple oncogenic pathways such as NF-κB, PI3K/Akt/mTOR, p53-mediated apoptosis, angiogenesis and metastasis (Sandhu et al., 2023). Experimental studies have shown that ursolic acid promotes apoptosis through p53 and caspase-3 signalling and inhibits the NF-kB mediated survival pathways (Manu & Kuttan, 2008). Moreover, it regulates the expression of genes involved in tumour angiogenesis by downregulating the expression of VEGF and matrix metalloproteinases, which limits the growth and metastasis of tumour cells (Manu & Kuttan, 2010). Ursolic acid has also been shown

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to inhibit various molecular pathways involved in the pathogenesis of cancer in comprehensive reviews, making it a potential candidate for future cancer prevention and treatment strategies (Pratheeshkumar et al., 2012; Sandhu et al., 2023).

Thymoquinone from *Nigella sativa* (Kalonji)

Nigella sativa commonly known as black seed or kalonji is a traditional medicine that has been used in Ayurvedic, Unani and larger south Asian medicine for vast range of ailments. The main bioactive component of it, thymoquinone, has a well-established and large body of mechanistic literature. Majdalawieh et al. (2017) comprehensively reviewed the anticancer mechanisms of thymoquinone across multiple cancer types. Thymoquinone has been shown to induce p53-dependent apoptotic cell death in colorectal cancer cells (Gali-Muhtasib et al., 2004) and inhibit both JAK/STAT and PI3K/Akt/mTOR signalling in myeloid leukemia cell lines (Al-Rawashde et al., 2022). Thymoquinone has also been reported to induce p73-dependent apoptosis in ALL by epigenetic regulation of UHRF1 and to inhibit DNA methyltransferase 1 (DNMT1) with nanomolar potency. Thymoquinone has been shown to exert radiosensitizing effect by causing both cell cycle arrest and apoptosis (Velho-Pereira et al., 2011) and targets Akt to induce G1 arrest via translational inhibition of cyclin D1 (Rajput et al., 2013).

The selected Indian ethnomedicinal plants, their main bioactive phytochemicals and major anticancer mechanisms reported in the literature are summarised in Table 1. These plants are part of a variety of chemical classes, which target multiple oncogenic signalling pathways implicated in the initiation and progression of cancer (Aggarwal et al., 2003; Grover et al., 2010; Joseph & Nair, 2013; Majdalawieh et al., 2017; Sandhu et al., 2023).

Table 1. Selected Indian Ethnomedicinal plants, principal phytochemicals, and major anticancer mechanisms

Plant (Common name)	Key Phytochemical(s)	Molecular Target(s)	Reported Anticancer Evidence
Curcuma longa (Turmeric)	Curcumin	NF-κB, AP-1, STAT3, COX-2, MMP-9	Apoptosis induction; suppresses proliferation, invasion and angiogenesis (Aggarwal et al., 2003)
Withania somnifera (Ashwagandha)	Withaferin A	NF-κB/IKK, PI3K/Akt/mTOR, proteasome, vimentin	Mitochondrial ROS-mediated apoptosis; inhibits invasion/metastasis (Yadav et al., 2010; Grover et al., 2010)

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Tinospora cordifolia (Guduchi)	clerodane diterpenoids, diterpene glycosides and polysaccharides	Immunomodulation, macrophage activation, cytokine regulation	Validated immunomodulatory and cytotoxic activity; HPTLC-quantified bioactives (Bala et al., 2015)
Rubia cordifolia (Manjishta)	Anthraquinones (purpurin, munjistin)	Apoptosis induction, anti-metastatic and anti-angiogenic activities in multiple cancer models.	Cytotoxic to HeLa, MCF-7, and PA-1 cell lines (cited in Himalayan anticancer plants review, Tariq et al., 2017)
Camellia sinensis (Green tea)	Epigallocatechin gallate (EGCG)	Apoptosis (DNA fragmentation), urokinase inhibition, AR modulation	Growth inhibition in LNCaP, PC-3, DU145 prostate lines and breast cancer cells (Paschka et al., cited in Da Silva Pinto, 2013)
Vitis vinifera (Grape)	Resveratrol	p53/p21 axis, NF-κB, topoisomerase II, telomerase (hTERT)	Antiproliferative/apoptotic activity across breast, colon, liver, and prostate lines (Singh et al., 2015)
Ocimum sanctum (Tulsi / Holy basil)	Ursolic acid (with eugenol, rosmarinic acid)	p53 pathway, oxidative stress modulation, angiogenesis inhibition	Cytotoxic to oral and breast cancer cell lines; component profile per Joseph & Nair (2013); ursolic acid mechanisms reviewed in Sandhu et al. (2023)
Nigella sativa (Black seed / Kalonji)	Thymoquinone	p53-dependent apoptosis, PI3K/Akt/mTOR, JAK/STAT, DNMT1	Apoptosis in colorectal, breast, leukemia and multiple solid-tumour lines (Majdalawieh, Fayyad & Nasrallah, 2017)

The ethnomedicinal anticancer plants documented in the Himalayas are summarized in Table 2 which includes the contribution of Indian medicinal flora, extent of experimental validation, and most commonly used plant parts in traditional medicine. These results highlight the diverse ethnopharmacological values of the region, and a scientific basis for further studies of Indian medicinal plants as potential sources of anticancer drugs (Tariq et al., 2017).

Table 2. Scale of Ethnomedicinal Anticancer Plant Documentation (Himalayan Region, with Indian Contribution)

Parameter	**Reported Value**
Total medicinal plants documented with anticancer ethnomedicinal use (Himalayan region)	64 species, 59 genera, 37 families
Plants reported specifically from India	42 of 64 (largest national share)
Plants experimentally tested in vitro	27 of 30 evaluated species
Plants tested in vivo	16 of 30 evaluated species
Most common plant part used in traditional recipes	Leaves (32%) and roots (30%), typically as decoctions

(Source: Tariq et al., 2017)

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4. Comparative Analysis of Anticancer Mechanisms

The phytochemicals mentioned belong to plants that show a wide range of taxonomic diversity, and are structurally very different; but these compounds share a limited number of molecular pathways that have been shown to be important in the initiation, progression and metastasis of cancer. This convergence confirms that there is a common pharmacological theme between ethnomedicinal anticancer agents and also reflects network pharmacology, in which multiple molecular targets are modulated simultaneously for therapeutic efficacy.

Among all the identified pathways, NF-κB signalling is one of the most commonly hijacked oncogenic pathways. Curcumin from *Curcuma longa* and withaferin A from *Withania somnifera* have been widely reported to inhibit activation of NF-κB, thus ultimately downregulating the expression of genes involved in inflammation, proliferation, angiogenesis and cell survival (Aggarwal et al., 2003; Grover et al., 2010). In the same manner, resveratrol and thymoquinone have indirect effects on NF-κB signalling via their effects on oxidative stress and upstream kinase pathways; thus, preventing inflammatory signalling is a common anticancer mechanism shared by several phytochemicals.

Regulation of oxidative stress represents another important shared mechanism. Curcumin, EGCG, withaferin A and thymoquinone have context-dependent antioxidant and pro-oxidant activities, allowing them to selectively disrupt the redox homeostasis of cancer cells and protect normal tissues against excess oxidative damage (Moloney & Cotter, 2018).

The induction of apoptosis both through p53-dependent and p53-independent pathways is another area of commonality. Resveratrol, EGCG and thymoquinone have been shown to activate p53-dependent signalling pathways leading to the activation of caspases, mitochondrial dysfunction, cell-cycle arrest and apoptotic cell death, in diverse cancer models (Yang et al., 2009; Majdalawieh et al., 2017). The above observations indicate that enhancement of apoptotic susceptibility is a common mechanism by which phytochemicals inhibit the survival of malignant cells and drug resistance.

Several compounds reviewed here impact the PI3K/Akt/mTOR pathway, one of the key pathways that regulates cell growth and metabolism. Ursolic acid and thymoquinone have been shown to inhibit Akt-dependent survival-signalling, which leads to inhibition of proliferation and induction

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of apoptosis. The multiple targeting of this pathway by structurally different phytochemicals further emphasizes the significance of this pathway in the anticancer activity mediated by phytochemicals.

Although these are common mechanisms there are differences among the plants reviewed. Curcumin and ursolic acid in addition to anthraquinones obtained from *Rubia cordifolia* also demonstrate significant anti-angiogenic and anti-metastatic activities by regulating the VEGF signalling pathway, MMPs, and ECM remodelling, limiting tumour invasion and dissemination. Conversely, *Tinospora cordifolia* shows more significant anticancer effects through immunomodulatory actions mediated by the clerodane diterpenoids, diterpene glycosides and polysaccharides which boost macrophages and lymphocytes activities. *T. cordifolia* acts by a host-directed action, unlike many phytochemicals which act through direct cytotoxicity.

All these points together suggest that ethnomedicinal phytochemicals work in a similar and complementary way to suppress the growth of tumours. Combinations of phytochemicals and multi-target approaches may prove to be interesting for the cancer prevention and treatment due to their potential to simultaneously affecting several pathways including the inflammatory, apoptotic, oxidative, angiogenic, metastatic and immune pathways. Collectively, these observations support the concept that ethnomedicinal phytochemicals act through network pharmacology rather than single-target inhibition.

5. Clinical Evidence and Translational Challenges

Numerous phytochemicals isolated from ethnomedicinal plants have been thoroughly studied and it has been found to have potential against cancer but some issues have to be resolved before clinical oncology can translate these findings. Most of the data come from in vitro studies and animal studies, and there are only a few well-designed human clinical studies. Most of the phytochemicals have shown anticancer activity in preclinical studies, but not very much in the clinic because of poor bioavailability, low metabolic stability, limited tissue exposure etc. and hence requires the development of new delivery systems and clinical testing (Singh et al., 2015). While there are many phytochemicals that have strong antiproliferative, pro-apoptotic, anti-angiogenic, and immunomodulatory effects, the therapeutic potential of these phytochemicals is limited due to poor

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bioavailability, rapid metabolism, the lack of aqueous solubility, inadequate tissue distribution, and inter-individual differences in pharmacokinetics (Newman & Cragg, 2020).

Curcumin has been one of the most extensively studied phytochemicals with regard to cancer. In multiple Phase I and Phase II clinical trials, it has been shown to be safe and tolerable in patients with colorectal, pancreatic, breast, and prostate cancers. However, it is limited in its clinical use due to its poor aqueous solubility, poor gastrointestinal absorption and extensive metabolism/elimination in the systemic circulation (Heger et al., 2014). Similar limitations in systemic bioavailability have also been reported for resveratrol, which has been found to have potentially beneficial anticancer effects in preclinical studies but has very low systemic bioavailability because of its high first-pass metabolism (Singh et al., 2015).

Epigallocatechin-3-gallate (EGCG) is the major catechin in green tea that has been shown to have positive activity in chemoprevention studies and preliminary clinical trials for prostate and colorectal cancers. However, further studies on the chemical instability and optimization of dosage and long-term safety are needed before there can be wide clinical use (Yang et al., 2009). Similarly, thymoquinone, ursolic acid and withaferin A have strong anticancer effects in experimental models, but are still in relatively early stages of clinical development due to pharmacokinetic issues and a lack of clinical validation.

In order to overcome these drawbacks, considerable research has been directed towards the development of smart drug delivery systems, including polymeric based drug carriers, liposomes, micelles, phospholipid complexes and nanoparticles. This can lead to higher solubility, bioavailability and reduced systemic toxicity, along with higher tumour-specific accumulation. For example, liposomal ursolic acid formulations have exhibited improved pharmacokinetic parameters with good safety profiles in Phase I clinical trial (Qian et al., 2015). Likewise, the therapeutic effects of nanoformulated curcumin and resveratrol have demonstrated greater therapeutic efficacy in the preclinical models, thereby enabling their clinical translation.

One of the major challenges to be solved is standardization of the herbal preparations. Biological activity may vary due to geographical variation, growing conditions, harvest conditions, extraction methods and phytochemical composition. Therefore, to make natural medicines that are

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based on phytochemicals a success in evidence-based oncology, there is a need for strict quality control, phytochemical profiling for the assessment, and regulatory guidelines.

Although clinical evidence remains limited, the multi-targeted effects of phytochemicals are important to recognize as compared to many conventional therapies. Since they can regulate a wide variety of pathways such as NF-κB, PI3K/Akt/mTOR, p53, oxidative stress, angiogenic signalling and immune responses simultaneously, they have also great potential to be used as adjunctive therapy, chemopreventive agents, or lead molecules for developing anticancer drugs. Large-scale randomized clinical trials, delivery system optimization, patient selection based on biomarkers, and combination therapies with current drugs should be emphasized in future studies to achieve the therapeutic potential of ethnomedicinal phytochemicals.

A summary of the selected phytochemicals' current clinical development status and their major translation challenges are given in Table 3. Several compounds have shown promising preclinical efficacy, with a few moving to Early-Phase clinical evaluation due to pharmacokinetic properties and lack of clinical validation (Heger et al., 2014; Singh et al., 2015; Zhu et al., 2013; Sandhu et al., 2023).

Table 3. Clinical Status and Translational Challenges of Selected Anticancer Phytochemicals

Phytochemical (Plant Source)	Clinical Development Status	Major Anticancer Activities	Key Translational Limitation	Representative References
Curcumin ***(Curcuma longa)***	Phase I/II clinical trials	NF-κB inhibition, apoptosis induction, anti-inflammatory activity	Poor bioavailability, rapid metabolism	Aggarwal et al., 2003; Heger et al., 2014
Resveratrol ***(Vitis vinifera)***	Early clinical investigation	p53 activation, NF-κB inhibition, antioxidant activity	Extensive first-pass metabolism	Singh et al., 2015; Varoni et al., 2016
EGCG ***(Camellia sinensis)***	Chemoprevention studies	Apoptosis induction, antioxidant activity	Chemical instability, dose optimization	Yang et al., 2009
Ursolic acid ***(Ocimum sanctum)***	Early clinical evaluation (liposomal formulation)	PI3K/Akt/mTOR inhibition, apoptosis, anti-angiogenic activity	Poor aqueous solubility, low bioavailability	Zhu et al., 2013; Sandhu et al., 2023
Anthraquinones ***(Rubia cordifolia)***	Predominantly preclinical evidence	Anti-metastatic, anti-angiogenic, pro-apoptotic activity	Limited clinical validation	Itokawa et al., 1993; Balachandran et al., 2021; Jadhav et al., 2022
Withaferin A ***(Withania somnifera)***	Advanced preclinical development	NF-κB inhibition, ROS-mediated apoptosis	Lack of clinical trials	Grover et al., 2010
Thymoquinone ***(Nigella sativa)***	Advanced preclinical development	p53 activation, PI3K/Akt/mTOR inhibition, JAK/STAT inhibition	Limited pharmacokinetic data	Majdalawieh et al., 2017; Al-Rawashdeh et al., 2022
Immunomodulatory diterpenoids ***(Tinospora cordifolia)***	Preclinical evidence	Immunomodulation, macrophage activation, cytokine regulation	Insufficient clinical evidence	Bala et al., 2015

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Table 4 shows selected phytochemicals that have been clinically investigated. The results of these studies suggest that compounds such as curcumin, EGCG, resveratrol and ursolic acid have encouraging safety profiles and preliminary therapeutic potential, and highlight the need for larger, well-designed randomized clinical trials (Yang et al., 2009; Heger et al., 2014; Zhu et al., 2013; Qian et al., 2015).

Table 4. Representative Clinical Studies of Selected Anticancer Phytochemicals

Compound	Clinical Development Stage	Cancer Type	Principal Clinical Findings	Representative References
Curcumin	Multiple Phase I/II clinical trials	Pancreatic, colorectal, breast, prostate	Safe and well tolerated; modest clinical efficacy; biomarker modulation	Aggarwal et al., 2003; Heger et al., 2014
EGCG	Chemoprevention studies; early clinical evaluation	Prostate	Reduction in prostate-specific antigen (PSA) levels; favorable safety profile	Yang et al., 2009
Resveratrol	Phase I clinical studies	Colorectal	Biomarker modulation and acceptable safety profile despite limited bioavailability	Singh et al., 2015; Varoni et al., 2016
Ursolic acid (nanoliposomal formulation)	Phase I clinical trial	Advanced solid tumours	Favorable pharmacokinetic profile and acceptable safety in Phase I evaluation	Zhu et al., 2013; Qian et al., 2015

In contrast to previous reviews that concentrated on the individual anticancer properties of ethnomedicinal plants or isolated phytochemicals, the current review highlights that structurally diverse phytochemicals target a limited number of key oncogenic signalling pathways, such as NF-κB, PI3K/Akt/mTOR, p53-mediated apoptosis, oxidative stress, angiogenesis, metastasis, and immune-inflammatory regulation. In this review, the ethnomedicinal knowledge, contemporary molecular oncology, comparative mechanistic analysis and clinical translation evidence are considered to form a network pharmacology framework, highlighting common therapeutic mechanisms and possible clinical applications. This holistic view offers a better basis for designing multi-target phytochemical combinations rationally, for developing future evidence-based anticancer agents and for biomarker-guided therapeutics.

6. Future Perspectives

The evidence of the anticancer activity of ethnomedicinal phytochemicals suggests that they could be useful tools in future cancer research and treatment. Their widespread adoption into evidence-based clinical practice has not yet been realised. These limitations will need to be overcome by inter-

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disciplinary approaches to ethnopharmacology, molecular oncology, medicinal chemistry, nanotechnology, systems biology, and clinical medicine.

Accessibility of the phytochemicals is one of the major priorities. The systemic exposure of many promising compounds, such as curcumin, resveratrol, EGCG, ursolic acid and thymoquinone, is low due to poor aqueous solubility, limited gastrointestinal absorption, rapid metabolism and/or low systemic exposure. In order to address these drawbacks, a number of formulation-based strategies have been explored. A Phase I nanoliposome pharmacokinetic study showed a linear pharmacokinetic profile, good tolerability, and no significant drug accumulation after multiple doses of ursolic acid in healthy volunteers and patients with advanced solid tumours, which supports the potential for translation of the nanoliposome formulation of ursolic acid into the clinic (Zhu et al., 2013). These barriers to the pharmacokinetic route frequently lead to a disconnect between promising preclinical results and relatively poor clinical results. The use of nanotechnology-based drug delivery systems, such as liposomes, polymeric nanoparticles, micelles, dendrimers, and phytosomes, are promising approaches to improve the bioavailability of the drug, increase the targeting of tumours and minimize the systemic toxicity (Heger et al., 2014; Qian et al., 2015). Further research on formulation stability and the establishment of a standard pharmacokinetic profile and therapeutic dosing are needed in the future.

The use of network pharmacology and systems biology is another potential area of research. In contrast to traditional anticancer agents that typically act on one molecule, phytochemicals tend to interact with several signalling pathways at once. Combined effects of different phytochemicals on NF-κB, PI3K/Akt/mTOR pathway, p53, ROS, angiogenic, and immune regulating pathways show that these compounds are specifically suitable for multi-target therapeutic strategies. The coupling of transcriptomics with proteomics and metabolomics and the use of artificial intelligence to define interactions of phytochemicals with targets can help identify complex phytochemical-target interactions and reveal novel mechanisms of action (Hopkins, 2008).

The combination of phytochemicals and synergistic formulations is also an interesting area of research. Traditional medicinal systems make use of multi-component formulations and not just single active components; thus providing potential for synergistic interactions between components with

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therapeutic activities. There are emerging indications that certain combinations of phytochemicals can act on multiple oncogenic pathways, increase therapeutic efficacy and decrease resistance. A combination of targeting signalling pathways such as NF-κB and PI3K/Akt/mTOR using multiple phytochemicals, for instance, may yield more comprehensive anticancer effects. The synergistic interactions should therefore be a major research priority in the future, using rigorous experimental validation.

There is also a growing interest in the importance of tumour microenvironment in cancer progression and therapeutic response. Many ethnomedicinal phytochemicals are known to have anti-inflammatory, immunomodulatory, anti-angiogenic and microbiome-modulating effects in addition to direct cytotoxicity. For example, a plant like *Tinospora cordifolia* has potent immunoregulatory functions that can be used in conjunction with pro-apoptotic phytochemicals such as curcumin, withaferin A and thymoquinone. Further research is warranted to explore the role of phytochemicals on immune checkpoints, tumour associated macrophages, cancer associated fibroblasts and gut microbiota, which are all known to be critical factors in cancer progression and treatment.

Precision oncology also opens up new possibilities for phytochemical interventions. By leveraging the genetic characteristics of the tumour, the pattern of activation of signalling pathways and metabolic profiles, a biomarker-guided approach can help identify patient groups that are most likely to benefit from a particular phytochemical. Future clinical trials could benefit from the use of molecular biomarkers, which could help refine patient stratification and aid in the development of personalized approaches to the use of phytochemicals for therapeutic purposes.

The need for standardization and quality control of ethnomedicinal products is also crucial. The genetic diversity of plant materials, the geographic origin, cultivation conditions, harvesting time, extraction procedures, storage conditions, etc., can significantly affect the phytochemical content and biological activity of plant materials. To ensure the reproducibility and regulatory acceptance of phytochemical preparations, it is necessary to use advanced analytical techniques like high performance liquid chromatography (HPLC), high performance thin layer chromatography (HPTLC), liquid chromatography-mass spectrometry (LC-MS) and metabolomic profiling (Bala et al., 2015).

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Large, well-designed clinical trials in the future are one of the most important needs. While there is a wealth of mechanistic and preclinical data for curcumin, EGCG, resveratrol, ursolic acid, withaferin A and thymoquinone, there are limited high-quality clinical data. Randomized controlled trials with pharmacokinetic and biomarker measurements, safety monitoring, and long-term efficacy evaluations are needed in the future. These trials will contribute to the incorporation of traditional ethnomedicinal knowledge into the modern practice of oncology.

Considering recent progress in formulation, systems pharmacology, molecular characterization and clinical trial design, the potential for ethnomedicinal phytochemicals to become clinically useful anticancer drugs is favourable. The integration of traditional knowledge of medicinal plants and biomedical research can lead to the development of new multi-targeted drugs that could offer some solutions to some of the most difficult scenarios in the current management of cancer.

7. Conclusion

Indian ethnomedicines such as Ayurvedic, Siddha, Unani and other indigenous healing traditions are a rich source of biologically active compounds that have a high anticancer potential. The medicinal plants reviewed in this article contain diverse classes of bioactive phytochemicals, including polyphenols, catechins, stilbenes, steroidal lactones, triterpenoids, anthraquinones, and quinones, which collectively contribute to their anticancer potential. Although structurally different, and ethnomedicinally distinct, these compounds show a remarkable convergence on a limited number of molecular pathways that play an important role in the initiation, progression and metastasis of cancer.

The evidence presented showed the anticancer activity of phytochemicals, including curcumin, withaferin A, EGCG, resveratrol, ursolic acid and thymoquinone, being mediated in concert via NF-κB, PI3K/Akt/mTOR, p53-dependent apoptosis, oxidative stress signalling, angiogenesis, metastasis, and immune-inflammatory pathways. Phytochemicals have a multi-targeted mode of action, distinguishing them from many of the traditional anticancer drugs, which can provide advantages in the treatment of pathway redundancy, tumour heterogeneity and therapeutic resistance. Multiple compounds with similar target regulation on the same oncogenic pathway further substantiate the network pharmacology concept, and indicate the potential of phytochemical combinations and polyherbal formulation.

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Among the examined phytochemicals, curcumin, EGCG, and resveratrol have progressed furthest toward clinical translation and have demonstrated promising safety profiles in early clinical trials. However, the majority of the ethnomedicinal phytochemicals are still at the preclinical stage, and the mechanistic evidence is extensive, and supportive of the therapeutic potential of these phytochemicals. Poor aqueous solubility, limited bioavailability, rapid metabolism, lack of standardization of herbal preparations and lack of large-scale randomized clinical trials are the common obstacles to clinical translation.

The future of the translation of ethnomedicinal phytochemicals into evidence-based oncology is expected to be driven by continued advances in drug delivery technologies based on nanotechnology, systems pharmacology, molecular profiling, and clinical research based on biomarkers. Additionally, the combination of traditional medicinal knowledge and modern pharmacological and clinical approaches provides a promising approach for the identification of more effective and safer multi-targeted anticancer drugs.

Ethnopharmacological plants of India continue to be a valuable and growing source of anticancer drug leads. Although they present many obstacles, there is strong support in the rapidly accumulating mechanistic, experimental and emerging clinical evidence for further study as potential sources for new therapeutic agents and complementary approaches to cancer prevention and treatment. The potential therapeutic use of these phytochemical resources could be fully achieved by future multidisciplinary studies, which combine ethnopharmacological, medicinal, molecular oncological and clinical science.

Glossary of Abbreviations

Abbreviation	Full Form
AI	Artificial Intelligence
ALL	Acute Lymphoblastic Leukemia
AP-1	Activator Protein-1
AR	Androgen Receptor
Bcl-2	B-cell Lymphoma 2
Bcl-xL	B-cell Lymphoma-extra Large
COX-2	Cyclooxygenase-2
CYP17A1	Cytochrome P450 Family 17 Subfamily A Member 1
DNA	Deoxyribonucleic Acid
DNMT1	DNA Methyltransferase 1
ECG	Epicatechin Gallate

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EGC	Epigallocatechin
EGCG	Epigallocatechin-3-gallate
EGFR	Epidermal Growth Factor Receptor
EMT	Epithelial–Mesenchymal Transition
ER	Estrogen Receptor
ERK	Extracellular Signal-Regulated Kinase
G1	Gap 1 Phase of the Cell Cycle
H2AX	H2A Histone Family Member X
HER2	Human Epidermal Growth Factor Receptor 2
HPLC	High-Performance Liquid Chromatography
HPTLC	High-Performance Thin-Layer Chromatography
HSP70	Heat Shock Protein 70
hTERT	Human Telomerase Reverse Transcriptase
IκB	Inhibitor of Nuclear Factor Kappa B
IKK	IκB Kinase
IL	Interleukin
LC–MS	Liquid Chromatography–Mass Spectrometry
MAPK	Mitogen-Activated Protein Kinase
MMP	Matrix Metalloproteinase
mTOR	Mammalian Target of Rapamycin
NEMO	NF-κB Essential Modulator
NF-κB	Nuclear Factor-kappa B
PA-1	Ovarian Teratocarcinoma Cell Line
PC-3	Human Prostate Cancer Cell Line
PI3K	Phosphatidylinositol-3-Kinase
PSA	Prostate-Specific Antigen
PTEN	Phosphatase and Tensin Homolog
ROS	Reactive Oxygen Species
STAT3	Signal Transducer and Activator of Transcription 3
TAMs	Tumour-Associated Macrophages
TNF-α	Tumour Necrosis Factor-alpha
uPA	Urokinase-Type Plasminogen Activator
UHRF1	Ubiquitin-like with PHD and RING Finger Domains 1
VEGF	Vascular Endothelial Growth Factor
Wnt	Wingless/Integrated Signalling Pathway

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