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Herbal immunomodulators in cancer immunotherapy

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Abstract
Cancer is increasingly recognized as immunological disease shaped by dynamic interactions between tumor cell and the host immune system within the tumor microenvironment. The cancer immunity cycle offers a unifying model in the understanding of how tumors are identified and eliminated in a cascade of steps. With the development of immunotherapy medicinal plants used in traditional systems like Ayurveda have been of interest as sources of bioactive phytochemicals with immunomodulatory properties. This review also combines global and India-specific cancer epidemiology with tumor immunology with specific emphasis on the influence of lifestyle and environmental factors in the immune dysfunction and evaluates clinically relevant medicinal plants including *Curcuma longa, Camellia sinensis, Withania somnifera, Catharanthus roseus, Nigella sativa and Boswellia serrata*, and combines evaluation of phytochemical characterization of the medicinal plants with tumor immunology. Case studies have also been analysed to study efficiency to Traditional Medicinal Plants and their efficient role in cancer management. Ethnobotanical information is discussed to put into perspective the historical uses of medicinal plants, to tie together traditional uses with modern pharmacological data and to identify future directions of personalized and plant-based immunomodulation.

Keywords: Cancer, immunology, medicinal plants, phytochemicals, tumor microenvironment, cancer immunity cycle, immunomodulation

Introduction

Cancer is a disease which cannot be controlled with regard to proliferation, invasion and metastasis. Historically, genetic and environmental factors, including tobacco, radiation, infections, mutations, etc., are traditionally viewed as the factors causing tumor development, but recent knowledge puts the immune system on the centre stage [1]. Traditionally, natural products have played an important role in oncology drug development. An example of a typical case of drug development starting with plants that resulted in drugs is paclitaxel of Taxus brevifolia, vinca alkaloids such as vincristine and vinblastine of *Catharanthus roseus*, camptothecin of *Camptotheca acuminata*, and etoposide based on podophylotoxin of *Podophyllum peltatum*. Cancer is a broad term used to describe diseases that are characterized by uncontrolled cell proliferation, irruption and expansion [2]. The fourth update of reports published by the American Cancer Society and the International Agency on Research on Cancer (IARC) on cancer estimates about 20 million new cancer cases each year in the world, with about 9.7 million of them dying as a result of cancer. The report reveals that about 50 percent of the cancer deaths in the world happen because people in different countries can alter their behavioural patterns, which include the use of tobacco, food choices, alcohol intake, and control of infections [3]. In 2022, it was reported that approximately 9.7 million cases were reported, which proves the growing public health burden of cancer [4]. Immune system is also a key factor in tumor development and treatment, as it is involved in a dynamic process called cancer immunity cycle. This cycle is initiated by tumor antigen release and proceeds on to the activation of antigen presentation, trafficking, infiltration, recognition and ultimately to the destruction of cancerous cells [6]. The immune system is closely related to cancer progression and therapy and may promote or inhibit tumor growth depending on the regulatory factors involved [7]. There has been a growing interest in recent studies in the use of plant-based and herbal agents as complementary methods of cancer prevention and treatment. Phytochemicals are bioactive compounds of medicinal plants that have demonstrated anti-cancer activity by altering the key molecular pathways and upregulating the immune responses [8]. Despite the large volume of literature on phytochemicals and cancer no existing review has systematically mapped the immunomodulatory activity for traditional medicinal plants onto each discrete step of the cancer immunity cycle moreover the integration of India specific cancer epidemiology with ayurvedic and ethnobotanical plant knowledge remains largely unaddressed in the context of modern tumor immunology.This review address the gap directly. The present review is devoted to the existing knowledge of how the immune system regulates cancer and how the activation and suppression of the immune system takes place through the action of definite components of cell organisms and how herbal medicines and phytochemicals can be used to change these processes [10]. Specifically this review uses the cancer immunity cycle as a mechanistic scaffold to evaluate clinically relevant medicinal plants including *Curcuma longa, Camellia sinensis, Withania somnifera, Catharanthus roseus, Nigella sativa and Boswellia serrata* linking their traditional medicinal use with modern pharmacological and immunological evidence. The review further contextualize these findings within both global and India specific cancer epidemiological burdens with particular attention to the high incidence North eastern regions of India, where traditional plant based medical system remains widely practised. The purpose of this review is to combine medicinal plants with

tumor immunology based on the cancer immunology cycle as a conceptual framework to understand the role of medicinal plants to modulate anti-cancer immune responses.

Global Overview of cancer

The Cancer immunity cycle is a multi-step cyclical process through which the immune system recognizes and eliminate transformed cells via seven coordinated and sequential steps [12]. Failure at any one of these steps may lead to immune invasion. Immunity based views are gaining more and more support on the side of plant derived compounds that can regulate tumor immune checkpoints, inflammation, antigen presentation and apoptosis [4]. This cancer-immunity cycle is thus the usual conceptual framework to integrate allopathy, phytotherapy and immunology and is used in this review to integrate all topics [14]. Three key factors that influence the incidence of cancer in various parts of the world are environmental pollution, patterns of human behaviour, and inherited characteristics which influence the production of antigens, inflammatory reactions and activation of immune system [14]. While global data establish the scale of the cancer burden, regional variations particularly within a country as epidemiologically diverse as India reveals the specific environmental, infectious and behavioural drivers that shape immune surveillance failure at the population level.

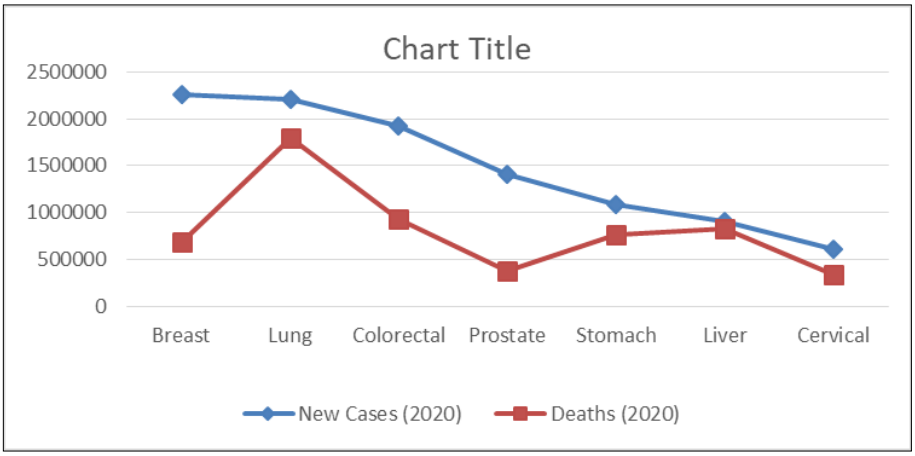

Fig 1: Comparative bar graph showing global cancer incidence and mortality for major cancer types (2022) [15]

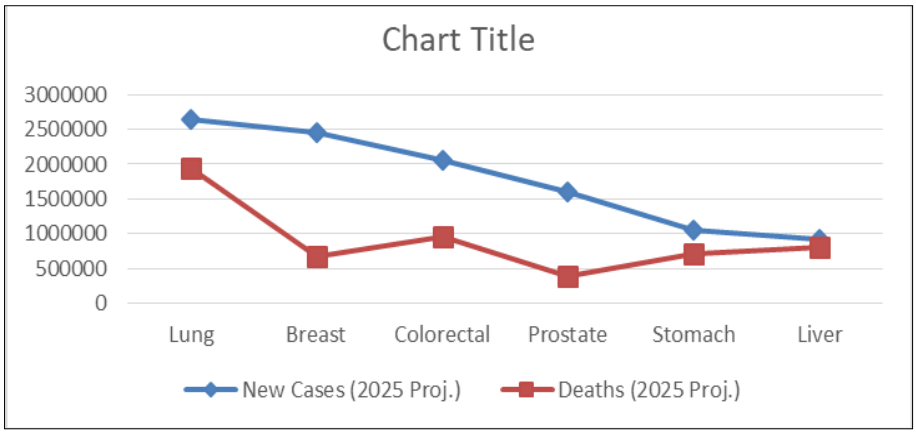

Fig 2: Projected Global Incidence and Mortality of Major Cancer Types (2025) [16]

India-specific Epidemiology

Analysis of cancer distribution across Indian regions suggests a disproportionately high incidence in the North east attributable to the persistence of chronic inflammatory conditions, infectious disease burden, environmental carcinogens and lifestyle related factors that collectively compromise early immune defence mechanism. The recent estimate shows increased cancer burden in India. The lifetime risk of cancer in India is 1 in 9 with an estimated 1.46 million new cancer cases in 2022 with a crude incidence of 100 per 100,000 [17, 18]. These trends concur with the global estimates that have been put together by the Global Cancer Observatory on India. The cancer burden in India differs dramatically according to the region. The population-based analyses of cancer registries have indicated that the North-East is always the highest Incidence. The Papumpare district of Arunachal Pradesh has the highest age-adjusted incidence rate (AAR) of men at about 269 per 100,000 and Mizoram Aizawl district has the highest AAR of women at around 220 per 100,000 which is significantly higher than major metros outside the NE [19]. These epidemiological differences are directly related to chronic inflammation, infections, and environmental exposures that have a direct impact on immune surveillance and tumorigenicity [19]. These regional epidemiological disparities driven by chronic inflammation, infectious exposure and environmental carcinogens directly compromise immune surveillance and create conditions favourable for tumorogenesis [19]. This creates a direct and underexplored intersection between local cancer burden and regionally available immunomodulatory plant resources as an intersection that is logically explained in this review

The Cancer Immunity Cycle: The Major Conceptual Framework.

The cancer immunity cell Cycle is made up of seven fundamental steps as shown in the diagram that includes: Release of tumor antigens, dendritic cell presentation of antigens, T cells priming and activation, Trafficking of T cells to tumor, invasion to tumor microenvironment, Cellular recognition, Death of cancer cells

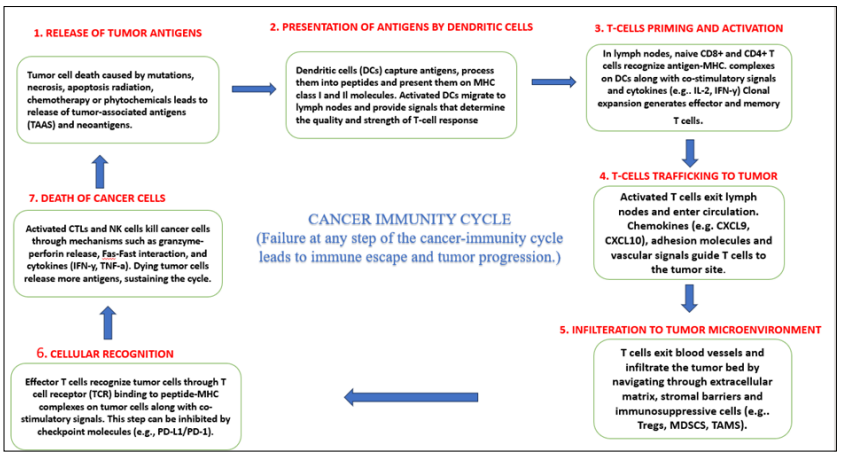

Fig 3: The Cancer Immunity Cycle and its Steps

Step 1: Release of tumor antigens: Cancer cells undergo cell death as a result of genomic instability, oxidative stress, hypoxia or therapy (caused by mutations, necrosis, apoptosis, radiation, chemotherapy, and phytochemicals). This causes tumor associated antigens (TAAs) and neoantigens to be released into the microenvironment [20]. It triggers the whole immunity cycle, determines the immunogenicity of the tumor, and affects the response to immunotherapy. Phytochemicals like curcumin, resveratrol and dandelion extracts have the capacity to induce apoptosis and increase release of antigens. Dysregulation of the cell cycle results in a high mutation load that ultimately results in more neoantigens being produced Due to epidemiology (e.g. smoking in lung cancer), that results in high mutation burden which causes a high antigen release. It is essential that the tumor antigens are well presented so that the cancer-immunity cycle is initiated. When such a process is effectively accomplished, the immune system is in a position to identify tumor cells and mount immune responses which consequently leads to an improved treatment effectiveness [21]. Conversely, low levels of antigen release makes tumor cells invisible to the immune system hence promoting tumor growth [21].

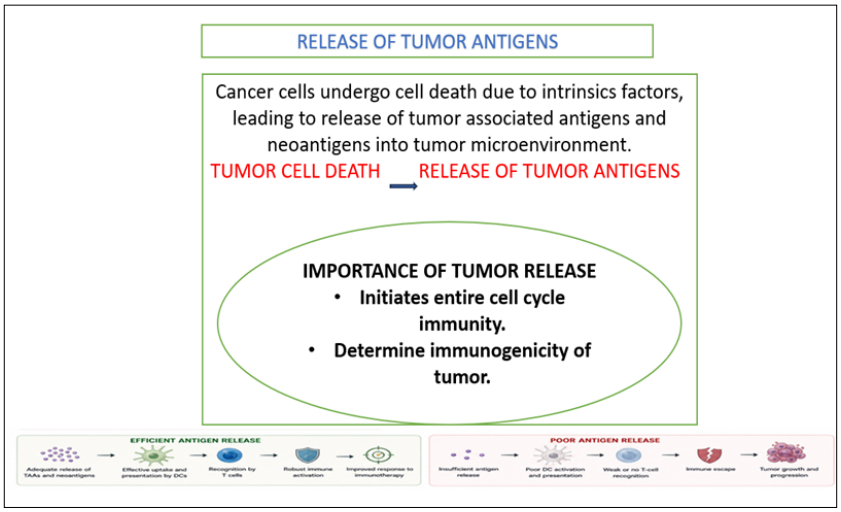

Fig 4: Release of tumor-associated antigens and danger signals from dying cancer cells, initiating the cancer immunity cycle [21]

Step 2: Uptake of antigens by Dendritic Cells (DCs): The antigens released are taken up by the dendritic cells and processed into peptides which bind to the MHC class 1 and 2 molecules. These cells migrate to lymph nodes where they are provoked to act as an interface between the innate and adaptive immune systems and also to regulate the quality and magnitude of the T-cell response. Therefore, the immune ignorance or tolerance is a consequence of poor antigen presentation [22]. TME suppressors including regulatory T cells, myeloid derived suppressor cells and tumor associated macrophages inhibit DC maturation thereby blocking effective antigen presentation. They also release pro-inflammatory cytokines like IL-12, IFN- g that are essential for T cell priming. Exogenous phytochemicals such as Withaferin A, curcumin and EGCG have been shown to enhance DC maturation and augment T cell primimg activity [22]. Priming of naïve T-cells to enable adaptive immunity requires successful presentation of antigen by DCs [22]. Such an antigen presentation is required for the development of a strong antitumor immune response. Immune tolerance can be induced by blocking antigen presentation, leading to poor T-cell activation and immunotherapy being ineffective in eliminating tumors [22].

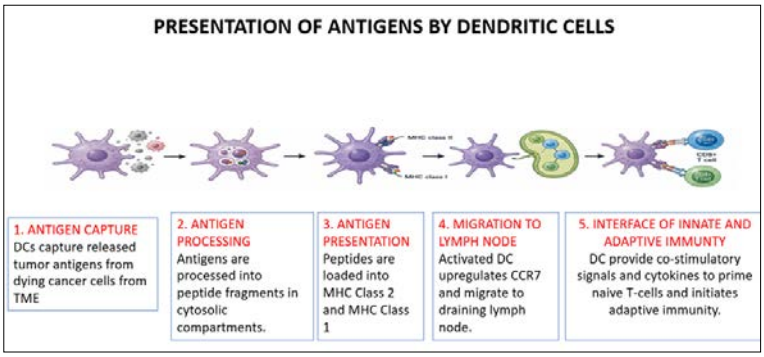

Fig 5: Dendritic cell-mediated processing and presentation of tumor antigens to T cells, leading to their activation in lymphoid tissues [22].

Step 3: T Cells priming and activation: Naive CD8 and CD4 T cells undergo clonal expansion, Activation, Differentiation into effector and memory cells [23]. This step requires a balance of co-stimulatory (CD28, CD40, OX40) and cytokine signals (IL-2, IFN-γ) [24]. Inhibitory checkpoint molecules such as CTLA-4 and PD-1 begin exerting suppressive influence during this step limiting T cell activation. Checkpoint inhibitor drug (anti CTLA-4 and anti-PD-1antibodies) work by blocking these molecules thereby restoring T cells [25]. Incomplete priming determines the immunoediting equilibrium stage. Tumor-secreted immunosuppressive cytokines (IL-10 and TGF- B) inhibit T-cell priming. Phytochemicals, including curcumin, decrease Tregs and MDSCs, and reestablish priming [25]. Activation and priming of T-cells result in cytotoxic T-lymphocytes that attack tumor cells and form immunological memory. Failure to activate T-cells might lead to ineffective immunity where tumor cells are able to survive and even evade immune responses [25]. Such circumstances also restrict the effectiveness of immunotherapy with checkpoint inhibitors [25].

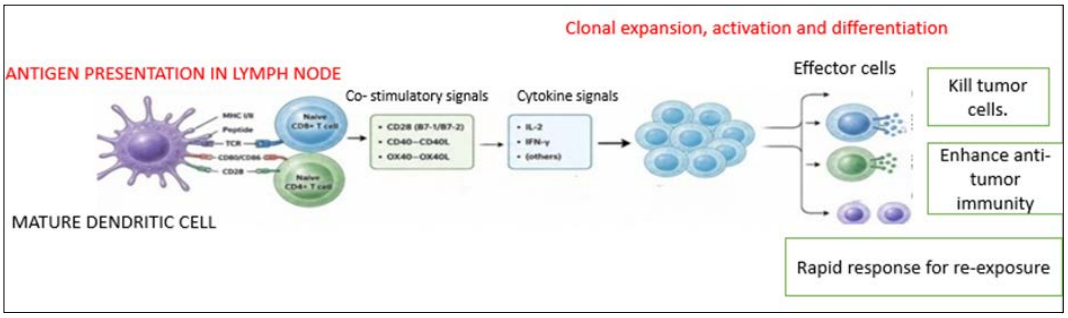

Fig 6: Priming and activation of naïve T cells by dendritic cells through antigen presentation, co-stimulation, and cytokine signalling [25].

Step 4: Trafficking of T Cells to Tumor Microenvironment: The stimulated T Cells, which are cytotoxic, move into the tumor guided by chemokines (CXCL9, CXCL10), adhesive molecules and vascular signals [26]. Even robust T-cell priming is pointless unless T cells can access the tumor, as tumors generate vascular barriers which impede immune trafficking. VEGF prevents the adhesion molecules in endothelial adhesion, as well as, inhibiting T-cell entry. Tumor derived factors that disrupt chemokine gradients and vascular adhesion represent a key mechanism of immune exclusion at this stage [26]. It is critical that activated T-cells are transported into the tumor microenvironment to accomplish antitumor activity [27]. In case the migration process is disrupted, the T-cells will not be able to localize to the tumor site and perform their functions. Although they can be turned on correctly, they are unable to combat tumors [27].

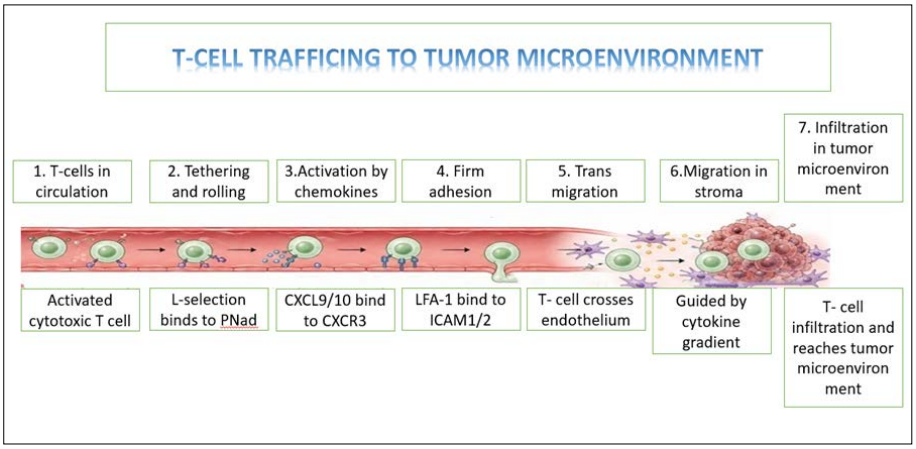

Fig 7: Migration of activated T cells to the tumor site mediated by chemokines, adhesion molecules, and endothelial interactions [27]

Step 5: Infiltration to the tumor microenvironment: Once in the vicinity of the tumor, the T cells must penetrate the tumor bed through: Dense extracellular matrix, Hypoxic zones, and Immunosuppressive immune cells. High infiltration (hot tumors) and poor infiltration (cold tumors) tumors respond in different ways to immunotherapy and Tumors with poor infiltration (cold tumors) are likewise resistant. Cancer associated fibroblasts (CAFs) and M2 polarised macrophages create a physical and chemical barrier that limits T cell penetration into the tumor core. Hypoxia, further limits T-cell movement caused by TME acidity. Phytochemicals have the potential to decrease ECM stiffness and inhibit suppressor cells enhances infiltration. Radiotherapy enhances the release of chemokines and the attraction of CTLs. The high natural infiltrations of colorectal MSI-high tumors explain the success of their immunotherapy. It results in tumor microenvironment becoming hot when there is effective T-cell infiltration of the tumor microenvironment, which makes them suitable in the implementation of immunotherapy. When, on the other hand, the process fails, the tumors will become cold, i.e. the tumors will exclude the immune cells in the centre of the tumours. The problem renders the immunotherapy of patients a challenge.

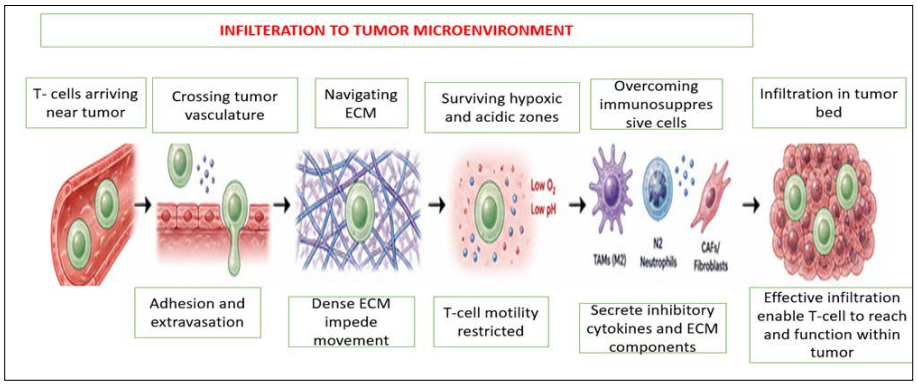

Fig 8: Infiltration of activated T cells into tumor tissue influenced by adhesion molecules, chemokines, and tumor micro environmental barriers.

Step 6: Cellular recognition: Cellular recognition is via T cell receptor (TCR) binding to peptide MHC, Co-receptors (CD8, CD4), Co-stimulatory and co-inhibitory signals [27]. Tumors can often down regulate MHC-I to become invisible, PD-L1 expression blocks TCR recognition, Mutation burden affects antigen visibility. Recognition is restored with checkpoint inhibitors (anti-PD1 and anti-PDL1). Transcriptional dysregulation induces changes in the antigen-presentation genes. Cancer stem cells avoid detection that aids in relapse. Cancers associated with viruses (HPV, EBV) produce immunogenic epitopes that result in improved recognition. Here the cancer–immunity cycle, endogenous cytotoxic T lymphocytes recognize tumor cells via TCR–MHC interactions [29]. This step is often avoided by tumors either by down regulating the MHC-I molecules or by modulating the antigen presentation pathways. Failure in recognition caused commonly due to the down-regulation of MHC proteins facilitates the escape of tumors from immune attack and increases chances of recurrence [29].

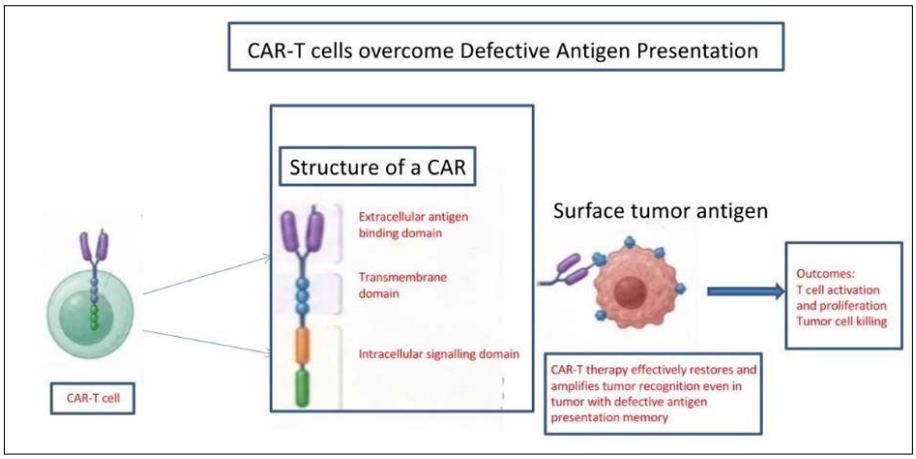

Fig 9: Antigen-specific recognition of cancer cells by T cells via TCR–MHC interactions within the tumor microenvironment [29].

Step 7: Death of Cancer Cells: Activated CTLs and NK cells induce apoptosis by: Granzymeperforin pathway, Fas-FasL interactions, IFN-g mediated cytotoxicity and TNF-a mediated apoptosis. The process of killing must be successful since it will be the culmination of the process. The unsuccessful invasion enables the tumor to expand and enter the body. When it is interacting with a target antigen, CAR-T cells generate potent cytotoxic effects, which is the completion of Step 7 of the immunity cycle. When CAR-T cells bind the antigen, they induce: Perforin-granzyme-induced cell death, Fas-FasL induced cell death. Killing of cancer cells essentially causes them to produce pro-

inflammatory cytokines such as IFN-γ, TNF-α) [30]. Its release results in cellular death that trigger step one of the cancer immunity cycle basically a positive feedback loop of immune activation [30]. A clinical effect of this mechanism is high rates of remission in hematological malignant neoplasm like B cell acute lymphoblastic leukaemia, diffuse large B cell lymphoma, and multiple myeloma [31]. It is the stage of the successful elimination of the cancer cells through the cytotoxic activity of the immune system. Not only does effective removal help reduce the size of the tumor, but also leads to the release of other antigens, therefore making the process even stronger as a result. The tumor cells will get more aggressive due to the inability to effectively remove the tumor [31].

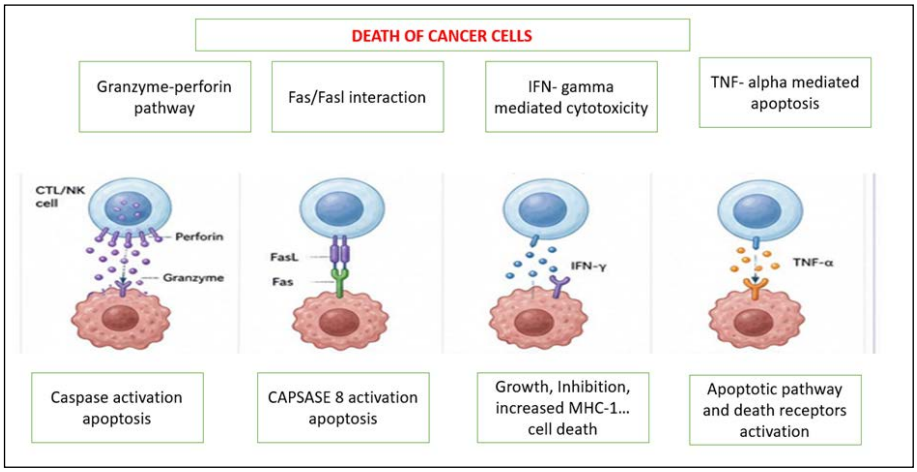

Fig 10: Cytotoxic T cell-mediated killing of cancer cells via perforin granzyme release and death receptor pathways [31]

Immunoediting within the Immunity Cycle

Immunoediting describes the long term evolution of tumors under immune pressure and fits directly onto the cancer immunity cycle.

Table 1: Overview of the elimination, equilibrium and escape phases of cancer immunoediting and their impact on cancer immunity cycle

Phase	Immunity Cycle Status
Elimination	All steps active leads to complete tumor control
Equilibrium	T cells Priming and activation and Death of cancer cells partially active and ultimately leads to dormant tumor
Escape	Immune suppression blocks all steps and leads to progression

The seven step cancer immunity cycle reveals multiple distinct points at which tumor progression can be intercepted. Crucially each of this step is also a point of potential failure that contributes to immune evasion and treatment resistance. This mechanistic understanding creates a rational framework for evaluating plant based immunomodulatory agents. Traditional medicinal system particularly. The following section examine whether and how specific medicinal plants and their bioactive phytochemicals can intervene at these defined steps of cancer immunity cycle- moving the discussion from historical use to mechanistic evidence.

Significance of Medicinal Plants in Cancer immunotherapy

The study of interrelationships between humans and plants within the cultural and traditional contexts, known as ethnobotany, provides a framework on which one can study the historical use of medicinal plants in disease prevention and treatment. Traditional medical systems like Ayurveda, Traditional Chinese Medicine and other indigenous healing traditions extensively used plant-based formulations to treat conditions that resembled cancer, such as chronic inflammation, abnormal growths and tissue degeneration [32]. These systems focused on a holistic approach; disease was seen as an imbalance of physiological and immunological homeostasis and treatment was aimed to bring about systemic balance as opposed to addressing individual symptoms [32]. In Ayurvedic medicine, various medicinal plants that are classified under Rasayana therapy are traditionally used to boost vitality, immunity and life span. Plant-based immunomodulatory and anti-inflammatory effects have been widely used that include *Curcuma longa*, *Withania somnifera* and *Tinospora cordifolia*. These classical uses are being augmented by recent findings of their ability to control cytokine production, to regulate the activity of immune cells and to influence key signalling pathways implicated in tumor progression [32]. In a similar vein, *Nigella sativa* has been utilized in Middle Eastern and South Asian cultures due to its broad therapeutic effects, which are often referred to as a “panacea” in traditional literature and nowadays it is known to have anticancer and immune-stimulating effects. The indigenous peoples of various geographical locations have also played a great role in ethnobotanical knowledge through empirical observations and long-term use of local plants. As an example, plant species like *Taxus brevifolia* and *Catharanthus roseus* have been used by tribal populations in India in the North-Eastern and Himalayan regions to treat various illnesses such as tumors and inflammatory diseases. Notably, such plants subsequently emerged as sources of clinically useful anticancer drugs including paclitaxel and vincristine, the translational importance of ethnobotanical knowledge in contemporary pharmacology. The shift in the traditional use to the drug discovery highlights the need to conserve the indigenous knowledge system as pools of possible therapeutic agents. The cultural practices related to the use of medicinal plants also contribute significantly to the influence that therapeutic outcomes have. The bioavailability and efficacy of phytochemicals can be greatly affected by such methods of preparation as decoctions, infusions, and fermentation [33]. Also, the

combinatorial approach of using multiple plant species in traditional formulations is indicative of an early awareness of synergistic interactions, which are congruent with current approaches in combination therapy of cancer treatment [33]. These practices tend to incorporate dietary, lifestyle, and spiritual factors and further support the notion of the multidimensional approach to health and disease management. Notably, the ethnobotanical knowledge may be regarded as valuable source of information about the choice of the plants with immunomodulatory potential which is especially relevant in the context of cancer as an immunological disease. A variety of traditionally used plants have now been demonstrated to modulate important steps in the cancer immunity cycle such as antigen presentation, T-cell activation and tumor cell killing Nevertheless even with its potential, integration of ethnobotanical knowledge in clinical oncology is fraught with challenges that include a lack of standardization, variability in plant composition and limited large-scale clinical validation [14]. To overcome these shortcomings, evidence-based medical interventions based on traditional plant-based therapies will require rigorous scientific research and interdisciplinary efforts. In short, ethnobotany is an essential linkage between the traditional healing system and contemporary cancer immunotherapy [14]. It helps both to discover new immunomodulatory agents to include in integrative cancer treatment strategies, and to enrich our comprehension of the use of medicinal plants within the cultural and historical frameworks [33]. Natural compounds from medicinal plants such as alkaloids, polyphenols, and flavonoids have been found to be effective in anticancer treatment in different mechanisms that include apoptosis induction, antiangiogenics, cell cycle arrest and immune modulation [34]. Most of these agents have preclinical support and some clinical history (e.g., vinca alkaloids, taxanes), but challenges exist in standardisation, bioavailability, dosing and rigorous clinical validation [14].

Table 2: Traditional Medicinal Plants and their Immunomodulatory mechanism

Plant (Common / Scientific)	Traditional Use in System	Cancer Immunology Mechanism	Primary Immune Target / Pathway	References
Turmeric *Curcuma longa L.*	Used for inflammatory conditions as describes in Charaka Samhita. Used as Haridra to purify blood and treat abdominal tumors (Gulma).	Inhibits NF- **κ**B and STAT3 which are two master regulators of cancer-based inflammation. Suppresses IL-6 and TNF- **α**, key cytokines that drive tumor immune evasion and T-cell exhaustion.(step 5)	NF- **κ**B · STAT3 IL-6 / TNF- **α** axis Tumor Microenvironment (TME)	35-36
Green Tea *Camellia sinensis (L.) Kuntze*	Used in TCM for over 4,000 years to clear heat, detoxify, and sharpen the mind. Classified as a cooling herb prescribed for inflammatory and febrile disorders.	Inhibits VEGFR-2 to cut off tumor blood supply; Downregulates HER2 reducing tumor immune escape signals.(step 4)	VEGFR-2 67LR receptor HER2 expression Anti-tumor angiogenesis	37-38
Ashwagandha *Withania somnifera (L.) Dunal*	A Rasayana (rejuvenating) herb used since 3000 BCE. Used to counter wasting diseases resembling cancer cachexia.	Directly activates NK cells via NKG2D and NKp44 receptors; enhances dendritic cell cross-presentation of tumor antigens; disrupts Hsp90 chaperone activity, impairing tumor cell survival signaling (step 2-3)	NK cell activation Dendritic cell function Hsp90 chaperone Innate immune system	39-40
Black Seed *Nigella sativa L.*	Called Habbatus Sauda and referenced as a cure for everything except death. Used in Unani for Mizaj correction (humoral balance), specifically for Balgham (phlegm) disorders linked to tumor-like growths.	Depletes myeloid-derived suppressor cells (MDSCs). One of the most potent immunosuppressive cell types in the tumor microenvironment thereby restoring T-cell function and enhancing checkpoint immunotherapy response. (step 6)	MDSC depletion T-cell restoration Anti-PD-1 synergy TME remodeling	41-42
Sweet Wormwood *Artemisia annua L.*	Listed in the Bencao Gangmu for 'clearing summer heat' and alleviating blood stagnation a TCM concept overlapping with tumor pathology.	Promotes Immunogenic Cell Death (ICD), a form of tumor death that activates the immune system via HMGB1 release and danger signals. Converts immunologically cold tumors (invisible to immunity) into hot tumors (immune-infiltrated).(step 1)	Immunogenic Cell Death HMGB1 / DAMPs Anti-PD-L1 synergy TME immune conversion	43-44
Indian Frankincense *Boswellia serrata Roxb.*	Extensively described in Ayurvedic texts (Sushruta Samhita) for Shotha (inflammatory swelling) and Gulma (abdominal masses).	Inhibits topoisomerase I/II, causing DNA double-strand breaks that activate ATM/CHK2 immune-related checkpoint signaling. Reduces GBM stem cell self-renewal, a key factor in immune surveillance evasion by brain tumors.(step 6)	ATM/CHK2 pathway GBM stem cell suppression DNA damage signaling Immune checkpoint priming	10-11
Berberine Plants *Berberis spp. / Coptis spp. Hydrastis canadensis*	Berberis used in TCM for 'damp-heat' syndromes, a pattern classically associated with gastrointestinal inflammation and infection-driven carcinogenesis.	Remodels the gut microbiome by selectively promoting Akkermansia muciniphila and suppressing pro-tumorigenic Fusobacterium nucleatum resulting in increased intratumoral CD8+ T-cell infiltration and restoration of immune surveillance.(step 4)	Gut microbiome-immune axis CD8+ TIL infiltration Anti-PD-1 sensitization CXCL9 chemokine upregulation	48-49
Garlic	In Ayurveda, classified as a Rasayana	DATS functions as an HDAC inhibitor re-	HDAC inhibition Tumor	

Allium sativum L.	for its anti-toxic and blood-purifying properties. Hippocrates prescribed it for uterine tumors.	expressing epigenetically silenced tumor suppressor genes (p21, p53, E-cadherin) that are critical for immune recognition of tumor cells and prevention of immune evasion via EMT.(step 6)	suppressor re-expression EMT reversal Immune recognition restoration	45
Asian Ginseng *Panax ginseng C.A. Mey.*	Considered the 'king of herbs' in TCM. Used for over 5,000 years as a supreme Qi tonic. Bencao Gangmu classifies it for 'tonifying the five viscera, calming the mind, and stopping palpitations' conditions overlapping with immune deficiency and cancer-related fatigue.	Rg3 reprograms tumor-associated macrophages (TAMs) from immunosuppressive M2 phenotype to anti-tumor M1 phenotype via TLR4/NF- κB signaling directly restoring innate immune anti-tumor activity within the TME(step5)	TAM repolarization (M2-M1) TLR4/NF-□B signaling Anti-tumor innate immunity VEGFR-2 angiogenesis block	46-47
Milk Thistle *Silybum marianum (L.) Gaertn.*	In Ayurveda, used as Dugdhapheni for Yakrit (liver) conditions. European medieval herbalists prescribed it for 'melancholic' diseases which is an ancient concept encompassing cancer-related conditions.	Suppresses IL-6-mediated JAK2/STAT3 signaling a pathway that drives myeloid cell immunosuppression and tumor-promoting inflammation while simultaneously inhibiting androgen receptor activity that contributes to immune evasion in prostate cancer(step3)	JAK2/STAT3 suppression IL-6 immune axis Anti-tumor inflammation Immune-mediated tumor suppression	50-51

The plants summarized in table 2 exert their immunomodulatory effects through specific bioactive phytochemicals. Understanding which chemical classes are responsible for these immune directed activities and which molecular targets they engage is essential for moving from traditional use to evidence based therapeutic application. The following section characterises the major phytochemical classes found in these medicinal plants and maps their documented anticancer mechanisms.

Phytochemicals and their role in Cancer Immunity Cycle

The various bioactive phytochemicals derived from medicinal plants are responsible for extensive anticancer and immunomodulatory properties. They interrupt various phases of cell cycle of tumor cells by controlling proliferation, promoting apoptosis, inhibiting angiogenesis and modulating the immunity at tumor milieu. Some of the key anticancer phytochemicals belong to major groups such as polyphenol, flavonoids, alkaloids, terpenoids, saponins and organosulfur compounds; most of which affect multiple signaling pathways of tumor cells. Curcumin, resveratrol, epigallocatechin gallate (EGCG), genistein and quercetin etc. Are some examples which have been extensively investigated and have potential in preventing and curing the tumor cell proliferation? Their ability to simultaneously engage multiple molecular targets make these phytochemicals compelling candidates for both chemoprevention and adjuvant cancer therapy particularly in combination with conventional treatments

Table 3: Selected phytochemicals from medicinal plants and their primary Anti-cancer Mechanisms

Class	Key Compound(s)	Plant Source	Primary Anticancer Mechanism(s)	References
Alkaloids	Vincristine, Vinblastine	*Catharanthus roseus*	Microtubule destabilisation; mitotic arrest; apoptosis induction	5
Alkaloids	Camptothecin (CPT)	*Camptotheca acuminata*	Topoisomerase I inhibition; DNA strand break induction	21
Alkaloids	Berberine	*Berberis vulgaris, Coptis chinensis*	NF-κB inhibition; STAT3 suppression; apoptosis via intrinsic pathway	15
Terpenoids	Paclitaxel (Taxol)	*Taxus brevifolia*	Microtubule hyperstabilisation; G2/M arrest; apoptosis	28
Terpenoids	Artesunate	*Artemisia annua*	ROS generation; ferroptosis; anti-angiogenic activity	52
Flavonoids	Quercetin	*Allium cepa, Camellia sinensis*	PI3K/Akt/mTOR inhibition; cell cycle arrest; anti-angiogenesis	53
Flavonoids	EGCG	*Camellia sinensis*	HER2/EGFR inhibition; VEGF suppression; apoptosis induction	54
Flavonoids	Apigenin	*Petroselinum crispum, Matricaria chamomilla*	CDK inhibition; G2/M arrest; autophagy induction	55
Phenolic Acids	Curcumin	*Curcuma longa*	NF-κB/STAT3 dual inhibition; PD-L1 downregulation; ICD induction	56
Phenolic Acids	Resveratrol	*Vitis vinifera, Polygonum cuspidatum*	Sirtuin activation; p53 stabilisation; anti-angiogenic	57
Saponins	Ginsenoside Rg3	*Panax ginseng*	VEGF suppression; Treg inhibition; MHC-I upregulation	58
Lignans	Podophyllotoxin	*Podophyllum peltatum*	Topoisomerase II inhibition; microtubule depolymerisation	59
Lignans	Silymarin	*Silybum marianum*	Wnt/β-catenin inhibition; STAT3 suppression	60

The phytochemical mechanisms outlined in the table 3 are supported by a body of preclinical and experimental evidences. The following case study analysis examine three representative compounds from *Taraxacum officinale*, *Curcuma longa and Vitis vinifera* to illustrate how these mechanisms translate into staged multi-level anticancer activity across the initiation, progression and advanced phases of carcinogenesis.

Justification of Efficient Role of Medicinal Plants in Cancer Management through Case Study Based Analyses

A growing body of preclinical and experimental evidence supports the role of medicinal plants in cancer treatment and immune modulation. This is supported by experimental and clinical investigations in which plant bioactive compounds have shown ability to inhibit tumor proliferation, induce apoptosis, regulate the immune responses and increase the efficacy of existing cancer drugs. Furthermore evidence indicates that medicinal plants target numerous signal transduction pathways involved in cancer and have considerably less toxic effects.

Table 4: Case study justifying Anticancer Mechanisms of *Taraxacum officinale* (Dandelion)

Cancer Stage	Primary Biological Effects	Key Molecular Targets & Mechanisms	Immune related effect	References
Initiation Stage (Early/Preventive)	Chemoprevention, antioxidant, and anti-inflammatory activity.	Upregulation of antioxidant enzymes (superoxide dismutase, catalase). Suppression of NF-κB and COX-2 signalling.	Dandelion polysaccharides activate macrophages and increase secretion of TNF- and IL-6 enhancing innate immune surveillance at the tumor initiation stage.(step 1)	68
Intermediate Stage (Progression)	Inhibition of uncontrolled growth and induction of apoptosis.	G2/M cell cycle arrest. Activation of intrinsic apoptosis (increased Bax/Bcl-2 ratio, caspase-3/9 activation, and mitochondrial depolarization).	Aqueous dandelion root extract selectively induces apoptosis in leukaemia cells while sparing normal lymphocytes, preserving the immune cell pool. Also shown to increase IFN-γ secretion by NK cells enhancing cytotoxic immune surveillance (step 7).	67
Advanced Stage (Metastasis & Resistance)	Anti-metastatic activity and chemo-sensitization.	Reversal of Epithelial-Mesenchymal Transition (EMT): Increased E-cadherin; reduced N-cadherin and vimentin. Inhibition of MMP-2/9 expression.	EMT reversal by dandelion extract restore surface expression of immune recognition molecules enabling cytotoxic T lymphocyte recognition of previously immune evading mesenchymal phenotype tumor cells (step 6)	65-66

Table 5: Anticancer Mechanisms of Curcumin (*Curcuma longa*)

Cancer Stage	Primary Role	Biological Mechanisms & Molecular Targets	Immune related effect	References
Initiation Stage	Protective Agent	Acts as a strong antioxidant to neutralize free radicals. Prevents DNA damage from carcinogenic compounds. Decreases oxidative stress to prevent genetic mutations.	Curcumin promotes DC maturation by upregulating MHC-2 and molecules (CD80, CD86) enhancing step 2 of cancer immunity cycle. Also supress IL-10 production by reducing early immunosuppressive conditioning of the TME.	56
Intermediate Stage	Anti-proliferative & Tumor-suppressing Agent	Blocks essential oncogenic pathways: NF-κB, COX-2, and STAT3. Triggers selective apoptosis in tumor cells while sparing healthy tissue.	Curcumin significantly reduces circulating MDSCs and regulatory T cells restoring CD8+cytotoxic T cell priming and activation (step 3) also supress IL-6 mediated JAK2/STAT3 signalling that drives immune evasion.	56
Advanced Stage	Adjuvant Therapy	Anti-angiogenic: Inhibits new blood vessel growth by downregulating VEGF. Anti-metastatic: Interferes with the epithelial-mesenchymal transition (EMT) to prevent cell mobility.	It downregulates PDL-1 expression on tumor cells, restoring TCR mediated recognition by exhausted T cells (step 6) .	61

Table 6: Anticancer Mechanisms of Resveratrol (*Vitis vinifera*)

Cancer Stage	Primary Role	Biological Mechanisms & Molecular Targets	Immune related effect	References
Initiation Stage	Protective Agent	Eliminates Reactive Oxygen Species (ROS) to safeguard DNA. Blocks carcinogen-activating enzymes (e.g., CYP1A1). Strengthens DNA damage repair processes.	Activates SIRT1 in DCs enhancing their antigen presenting capacity and promoting Th-1polarised immune response essential for anti tumor immunity at step 2 . it also supress Treg differentiation by inhibiting TGF-BETA mediated FOXP3 induction preserving effector T cell pools.	62
Intermediate Stage	Tumor Suppressor	Stops PI3K/Akt and NF-κB signalling pathways to prevent cell growth. Activates caspases and the p53 pathway to	Enhances NK cell cytotoxic activity by upregulating NKG2D activating receptor expression on NK cells and increasing expression of NKG2D ligand on	63

		trigger apoptosis. Decreases associated inflammatory responses.	tumor cells facilitating direct NK mediated killing (step6,7).	
Advanced Stage	Adjuvant Treatment	Anti-angiogenic: Stops production of VEGF and HIF-1α to starve tumors of resources. Anti-metastatic: Blocks the epithelial-mesenchymal transition (EMT) to prevent spreading.	Repolarises tumor associated macrophages from immunosuppressive M2 phenotype toward anti tumor M1 phenotype via SIRT1/NF- **κ**B axis directly remodelling the TME. It also synergises with anti PD-1 immunotherapy by reducing PD-L1 expression on tumor cells (step 6,7).	64

The case study analysis of *Taraxacum officinale, Curcuma longa and Vitis vinifera* collectively demonstrate the plant-derived phytochemicals can intervene at multiple stages of cancer progression from early chemoprevention to reversal of drug resistance at advanced stages. These compounds engage both direct cytotoxic pathways and immune modulatory mechanism that correspond to defined steps of the cancer immunity cycle. Taken together with medicinal plants and phytochemical characterisation presented in preceding sections these findings support the rational integration of plant based agents into immunity directed cancer treatment strategies.

Conclusion

Cancer is not only a genetic disease but is fundamentally an immunological aspect that determines the fate of tumors. The interplay between the immune system and cancerous cells is dynamic and is captured by the cancer-immunity cycle and the immunoediting paradigm. Plant derived compounds that have long featured in traditional medicine continue to yield clinically validated anticancer agents including vincristine, paclitaxel and irinotecan while a new generation of phytochemicals such as curcumin, thymoquinone and artesunate is being investigated for their specific capacity to modulate the cancer immunity cycle. Based on the comparative analysis of *Taraxacum officinale* (Dandelion), *Curcuma longa* (curcumin), Resveratrol (*Vitis vinifera*) and others it is evident that these phytochemicals offer a comprehensive, multi-stage defence against carcinogenesis. Future directions should prioritise Nano formulation strategies to improve bioavailability standardised extraction and dosing protocols by evaluating phytochemicals as adjuncts to checkpoint immunotherapy. India's exceptional medicinal plant biodiversity and its deep rooted ayurvedic tradition represent an underutilised strategic resource for developing the next generation of plant based immunomodulatory cancer therapies

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