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Exploring the Potential of Asian Medicinal Plants in the Prevention and Treatment of Leukemia: A Comprehensive Ethnopharmacological Review

Running Title: Asian medicinal plants in treatment of leukemia

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ABSTRACT

The effectiveness of Asian herbal medications has been known since ancient times. Their impacts on people's lives are significant, and their efficacy has been demonstrated in various studies. These plants contain phytochemicals, such as antioxidants, in their essential oils or extracts, classifying them as anti-leukemic agents. One of the primary concerns in modern medicine is the search for new treatments and innovative preventive approaches to cancer. Leukemia stands among the 15 most frequent types of cancer. Due to its high mortality rate, further research should focus on treatment and especially prevention. Chemotherapy is the most common treatment for leukemia; however, it causes undesirable side effects. That is why the exploration of alternative strategies should be prioritized. A comprehensive literature search was conducted using the following keywords: anti-leukemic plants, leukemia prevention, and leukemia treatment. The main databases included Google Scholar, Springer Link, PubMed, ScienceDirect, and Elsevier. This review provides a list of 104 medicinal plant species used by Asian folk, with documented anti-leukemic properties. The plants mentioned in this paper contain various phytochemicals, such as flavonoids and alkaloids. Numerous *in vitro* and *in vivo* studies have demonstrated the promising effects of these plant extracts against leukemic cell lines. They help prevent and treat leukemia by inducing apoptosis and cell death, reducing cell viability, suppressing cell invasion, and targeting specific leukemic cell lines through various bioactive compounds like chlorogenic acid, xanthochymol, and berbamine. They also inhibit angiogenesis and disrupt cancer cell proliferation through mechanisms such as vascular endothelial growth factor (VEGF) suppression and STAT3 inhibition. The results of the current review could be applied in the development of a new generation of anti-leukemia drugs with minimal drawbacks.

Keywords: Anti-cancer, Herbal medicine, Leukemia, Plant secondary metabolites

INTRODUCTION

Cancer is one of the main global public health issues and the second leading cause of mortality worldwide [1]. The incidence of cancer is influenced by numerous factors such as age, genetics, smoking, alcohol consumption, obesity, ultraviolet (UV) radiation, and certain viruses [2]. While certain factors, such as genetics, cannot be changed, the occurrence of this disease can be significantly reduced by managing other risk factors. Studies show that in high-income countries, up to 80% of cancer cases can be prevented by addressing environmental factors and lifestyle choices, including nutrition [3].

Phytochemicals are small molecules found in fruits and vegetables and have demonstrated anti-cancer properties in both *in vitro* and *in vivo* studies [4]. These compounds are synthesized by plants to cope with unfavorable environmental conditions, such as biotic and abiotic stresses [5]. Common examples of phytochemicals include polyphenols, carotenoids, phytosterols, flavonoids, and alkaloids [6]. Quercetin, a prominent flavonoid in fruits and vegetables, has been reported to modulate almost all hallmarks of cancer [7]. Other phytochemicals, like alkaloids, have anti-proliferative, antibacterial, and antioxidant potential, and have the potential to be used in drug development [8].

Currently, chemotherapy, radiotherapy, immunotherapy, and even surgical removal are applied to treat cancer; however, these treatments may result in diverse side effects [9], such as skin diseases, cardio-toxicity, headaches, hair loss, and neurological disorders [9, 10]. Additionally, cancer can metastasize from its primary site to different organs [11]. Metastasis is the cause of over 90% of deaths in all cancer types since conventional treatments are often ineffective in such cases [12]. Therefore, prioritizing prevention over treatment is essential.

Among various types of cancer, leukemia stands out with 437,033 cases diagnosed globally and approximately 309,006 deaths recorded in 2018 [13], making it a significant concern in the medical field. Several environmental factors, such as ionizing radiation [14, 15], chemical exposures [16, 17], maternal exposures [17–19], and air pollution [18], have been identified as contributors to leukemia development. However, identifying and quantifying environmental risk factors remains challenging due to difficulties in exposure assessment, the presence of multiple confounding factors, and the need for large study populations to detect effects from low-level exposures [14]. Genetic and inherited factors are also estimated to contribute to approximately 10% of childhood leukemia cases [20, 21]. Genetic and inherited conditions, including inherited syndromes [16, 22], genetic mutations and abnormalities [23], as well as inherited genetic susceptibility, are associated with increased leukemia risk [20, 21]. Lifestyle and personal factors, on the other hand, have been

associated with increased leukemia risk. Smoking, diet and nutrition [24, 25], body composition and physical activity [23], prior medical treatments, such as previous chemotherapy treatment for other cancers, can increase leukemia risk [26] (Figure 1).

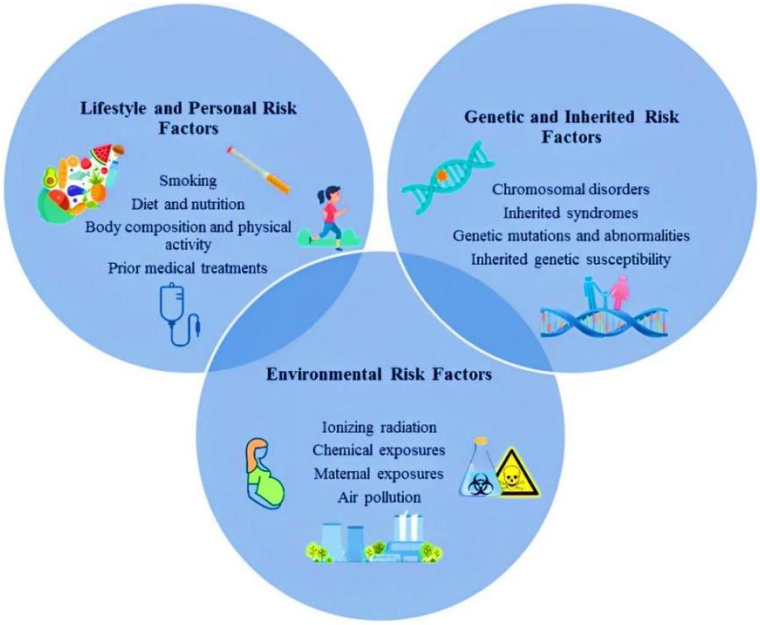

Fig. 1 Factors affecting leukemia risk.

Leukemia can be broadly classified into two main categories: acute leukemia (AL) and chronic leukemia (CL). AL progresses rapidly and is characterized by severe clinical signs, whereas CL develops more slowly and tends to have a milder initial presentation. The four primary types of leukemia fall under these two categories: acute lymphocytic leukemia (ALL), acute myeloid leukemia (AML), chronic lymphocytic leukemia (CLL), and chronic myeloid leukemia (CML) [27]. Notably, CLL and CML are less common among Asians compared to European populations, where their incidence is higher [28, 29]. This disparity may be influenced not only by genetic factors but also by dietary habits and the use of traditional herbal remedies in Asian cultures, which may offer a protective effect against cancer [30, 31]. Asian medicinal plants, in particular, have long been recognized for their potent therapeutic properties.

Given the potential of many Asian medicinal herbs to prevent or possibly be effective in treating leukemia patients [32], this review compiles a list of medicinal plants used in traditional Asian medicine with anti-leukemic effects. These plants can be used as traditional medicines or dietary supplements. Moreover, future studies can explore their potential for developing a generation of anti-leukemia drugs.

MATERIALS AND METHODS

A comprehensive literature search was conducted, and the information from 270 articles (reviews and research articles) has been gathered in this review. The main relevant keywords used in finding results were: anti-leukemic plants, leukemia prevention, leukemia treatment, and Asian anti-leukemic herbal medicine. The results and the names of the 104 plant species are combined in a single table, which describes their usage in Asian traditional medicine and their mechanisms of action. The challenges in gathering this information were that not all plants have been fully investigated, and further research is needed to understand their exact mechanisms. The main databases used for this comprehensive search were: Google Scholar, Springer Link, PubMed, ScienceDirect, and Elsevier.

Types of Leukemia

Acute Lymphocytic Leukemia (ALL)

This type of leukemia is characterized by a disruption in the differentiation of early lymphoid progenitors, leading to the accumulation of immature lymphoblasts [33]. The most common symptoms include fever, fatigue, or purpuric rash [34]. Aberrant DNA methylation of multiple CpG islands (CGI) in ALL has been identified as a prevalent factor contributing to a poor prognosis [33]. ALL cells can originate from either B or T cells [35]. Notably, in T-cell ALL, only a few molecular abnormalities have been identified, whereas B-lineage ALL often involves additional genetic abnormalities that can influence the clinical course of the disease [36].

Folic acid deficiency is considered a risk factor for adult ALL [37], potentially related to chromosome breaks that increase the risk of folate-deficiency-associated cancers [37]. ALL in adults often involves the central nervous system (CNS) [38]. CNS involvement occurs in 3-5% of patients at diagnosis, rising to 30-40% at relapse, with CNS relapse being a major cause of treatment failure [39–41]. Leukemic cells infiltrate the CNS through adhesion molecules (VLA-4, ICAM-1) and integrin-mediated migration [42, 43]. While some patients present with neurological symptoms such as headaches, seizures, or cranial nerve deficits, many remain asymptomatic, necessitating lumbar puncture (LP) for detection [44]. It is recommended that all newly diagnosed patients with ALL undergo routine LP testing [38]. LP classifies CNS involvement as CNS1 (<5 white blood cells (WBCs)/μL, no blasts), CNS2 (<5 WBCs/μL with blasts), or CNS3 (≥5 WBCs/μL with blasts or cranial nerve involvement) [45]. Flow cytometry enhances sensitivity over conventional cytology, improving detection rates from 30% to 65% [41, 46]. CNS3 status is associated with poor outcomes (50% 5-year survival vs. 80% for CNS1), underscoring the importance of early CNS-directed therapy [39, 47].

Acute myeloid leukemia (AML)

AML ranks among the common leukemias in adults [48]. AML affects myeloid, erythroid, megakaryocytic, and monocytic cells [49], and typically presents with symptoms such as fatigue, dizziness, bone pain, and extreme weakness [50].

It is characterized by poorly differentiated or abnormal cells and clonal transformation within the hematopoietic system [49, 51]. In this hematopoietic disorder, mutant cells undergo changes that result in increased turnover and proliferative capacity, as well as altered hematopoietic differentiation [52]. Mutations can occur in various genes such as *NPM1*, *CEBPA*, *RUNX1*, *KIT*, *NRAS*, *FLT3-ITD*, *TP53*, *TET2*, *KMT2A-PTD, ASXL1*, *DNMT3A*, *IDH1*, and *IDH2* [51]. The most frequent mutation occurs in the *NPM1* gene in about 25 to 35 percent of patients, and it is often associated with other mutations in genes like *TET2*, *FLT3-ITD*, *DNMT3A*, *IDH1*, and *IDH2* [51]. Unlike ALL, AML is not substantially influenced by folic acid deficiency, as AML is not affected by the polymorphism [37].

Effective chemotherapy for AML, particularly in older patients, remains a significant challenge, with a median survival of 5 to 10 months in this demographic [51]. However, in patients aged 60 or younger, AML can be cured in 35%-40% of cases with intense induction therapy and consolidation, often followed by allogeneic stem cell transplantation [51, 53]. Notably, the treatment of AML can lead to long-term side effects, including chronic fatigue and cognitive impairment, which may impact the quality of life for survivors [53].

Chronic Myelocytic Leukemia (CML)

CML accounts for approximately 20% of all leukemia cases [54, 55]. It is characterized by high WBC counts and extramedullary hematopoiesis (EMH), which causes spleen enlargement. These abnormalities result from a (9; 22) chromosomal translocation that creates the BCR-ABL fusion oncoprotein [54]. This genetic swap between chromosomes 9 and 22 produces the shorter Philadelphia chromosome [56]. The resulting fusion proteins (p210 or p185) lead to abnormal kinase activity [57–59], causing excessive proliferation of WBC [60]. Targeting BCR-ABL is therefore a key treatment strategy [56].

Accurate molecular and cytogenetic analyses are crucial for selecting the most effective treatment [61]. CML is often diagnosed during the chronic phase when it can be effectively managed with relatively low-toxicity drugs or bone marrow transplant [54].

In the chronic phase, lower quantities of BCR-ABL-positive stem cells gradually multiply while still producing normal blood cells (except T-cells)[62]. Several studies suggest that CML has a lower incidence in Asian and Middle Eastern countries compared to Western countries [28, 63], indicating that genetics or environmental factors, particularly diet, should be explored for a viable solution.

Chronic Lymphocytic Leukemia (CLL)

CLL is one of the most common forms of leukemia, representing 1.1% of all cancers in 2021. It predominantly affects elderly patients, with only 9.1% of cases occurring in individuals younger than 45 years old [64]. In its early stages, CLL symptoms may be vague, but signs such as an enlarged spleen, bone marrow failure, or swollen lymph nodes indicate the progression of the disease [65]. Genetic abnormalities play a key role in CLL development. Approximately 80% of patients carry at least one of four chromosomal changes: trisomy 12, deletions in 13q14.3, 11q22.3, or 17p13.1 [64, 66]. The 17p13.1 deletion, which disrupts the *p53* tumor suppressor gene, is rare at diagnosis but becomes more frequent as the disease advances. This deletion is particularly concerning because it is linked to chemotherapy resistance and poorer treatment outcomes [64, 67, 68]. CLL arises from abnormal $CD5^+/CD19^+$ B lymphocytes that weaken immune function [68]. Diagnosis involves blood tests, microscopic examination of blood smears, and immunophenotyping to detect these atypical B cells [64]. Interestingly, CLL is the most common form of leukemia in European countries, while Asia has one of the lowest age-adjusted incidences [69].

Traditional use of Plants in Cancer Treatment

Current cancer therapies like chemotherapy, immunotherapy, surgery, or radiation, while effective, often cause serious side effects such as heart toxicity, alopecia, skin disease, oral and stomach ulcers, epithelial layer destruction, neural problems, or bone marrow suppression [10]. While medicinal plants have demonstrated significant potential in cancer prevention and treatment, they cannot replace modern therapies. Instead, they may serve as complementary adjuncts to conventional treatments, helping to mitigate side effects or enhance efficacy.

Ethnomedicine, which refers to the use of medication in different cultures and examines innovative local ideas and behaviors related to disease treatment and health improvement [70], can be used as a source of inspiration for next-generation drugs, as a huge number of today's useful therapies are derived from folk medicine [71]. The use of plants for disease treatment and combating infections has a long-term history [72], and has been addressed in numerous studies [73, 74]. Some studies have discovered that Chinese folk medicine plays an important role in cancer treatment, such as in skin cancer [75]. In Korea, traditional medicine is still used by licensed traditional doctors under the supervision of the Ministry of Health and Welfare of Korea [76].

Different species of plants are used traditionally in the treatment of various diseases, including cancer. For example, species of *Acacia* Mill. have been used for treating different types of cancer, such as mouth, bone, and skin cancers [77]. However, while these natural compounds show promise, they should be integrated thoughtfully into cancer care, ensuring they support, rather than replace, evidence-based medical treatments.

The Role of Plants and Plant Metabolites in the Prevention and Treatment of Leukemia

The rising number of cancer cases and the adverse effects of conventional treatments have prompted researchers to explore medicinal plants as an emerging and promising source of anti-cancer compounds [10]. Plant-derived compounds serve as a valuable source of pharmaceutical agents for various human diseases [78]. Moreover, the undeniable role of a diet rich in fruits and vegetables in reducing cancer risk underscores the potential of plant-based therapies [79].

Numerous plant species have exhibited anti-leukemic properties in various ways, including inducing apoptosis and cell death, reducing viability in leukemic cells, and suppressing cell invasion or cytotoxicity [68, 78, 80, 81]. *Piper betle* L., as a traditional medicinal plant in Southeast Asia, has been extensively studied for its anti-microbial, antioxidant, and chemopreventive effects [82]. The leaves of *P. betel*, often chewed fresh or dried [83], contain compounds such as tannins, hydroxychavicol, and eugenol [84]. Extracts from the leaves have

demonstrated an inhibitory effect against the growth of human CML in xenograft models [82]. Chlorogenic acid (Chl) extracted from *P. betel* leaves effectively targets the BCR-ABL-positive K562 cells (*in vitro*); however, it has no effects on the viability and growth of BCR-ABL-negative lymphocytic and myeloid cell lines or primary CML cells [57]. Moreover, sodium chlorogenate (NaChl) extracted from leaves exhibited a destructive effect on BCR-ABL positive KU812 and KCL22 leukemic cell lines (*in vitro*) [57]. NaChl showed a higher efficacy in killing K562 cells compared to Chl [57].

Garcinia indica (Thouras) Choisy (Figure 2), a medicinal plant native to India, has shown remarkable inhibitory activity and apoptosis induction in both CML and AML cells. In CML, overexpression of vascular endothelial growth factor (VEGF) and enhanced angiogenesis are common [85]. While xanthochymol (from *G. indica*) decreases cell invasion, VEGF secretion, and metalloprotease production in both CML and AML [80], xanthohumol (from hops) exerts similar effects but through distinct pathways. Xanthohumol inhibits the angiogenic process by suppressing VEGF secretion via AKT/NF-κB blockade [85]. Furthermore, it disrupts the reciprocal stimulatory loop between endothelial and leukemic cells by impairing endothelial cell activation [85]. In contrast, xanthochymol's anti-angiogenic effects are linked to its antioxidant properties and modulation of STAT3/VEGF signaling [80].

Fig. 2 *Garcinia indica* in different stages of growth (www.inaturalist.org)

Berbamine (Figure 3), a bioactive alkaloid derived from *Berberis amurensis* Rupr. (Figure 4), has shown significant potential in targeting both imatinib-sensitive and -resistant Ph+ CML cells through multiple pro-apoptotic pathways [86]. This compound induces mitochondrial-mediated apoptosis by disrupting the Bcl-2/Bax balance, leading to cytochrome c release and subsequent activation of the caspase cascade. Additionally, berbamine triggers endoplasmic reticulum stress through CHOP activation while simultaneously inhibiting key survival kinases, including BCR-ABL1 and Aurora kinases, making it particularly effective against therapy-resistant CML populations[86].

Complementing these effects, saponins isolated from *Rubus parvifolius* L., a traditional Chinese medicinal herb, demonstrate potent anti-leukemic activity through a distinct but equally compelling mechanism of action [87]. These bioactive compounds activate AMPK-mediated metabolic stress pathways, resulting in mTORC1 inhibition and subsequent suppression of protein synthesis. Concurrent STAT3 pathway inhibition leads to downregulation of critical anti-apoptotic proteins such as Mcl-1 and survivin. The saponins further impair leukemic proliferation by inducing G1/S cell cycle arrest through modulation of cyclin D1/CDK4/6 complexes and upregulation of p21/p27 cyclin-dependent kinase inhibitors [87].

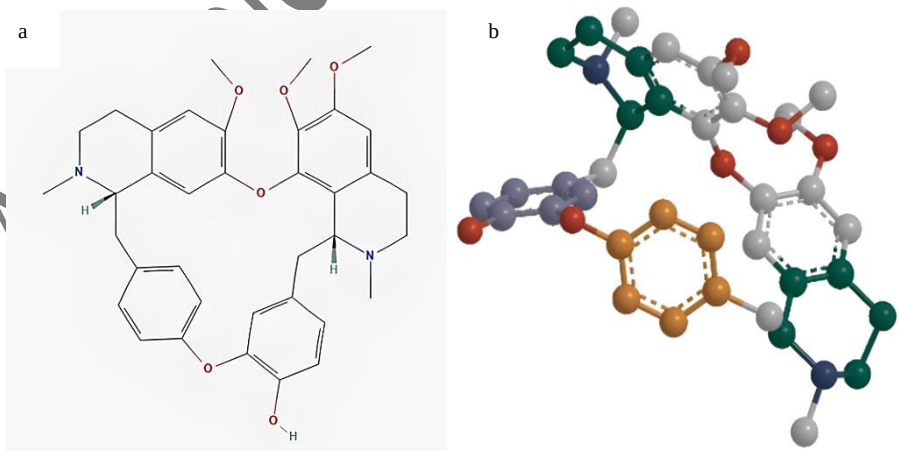

Fig. 3 2D structure (a) (https://pubchem.ncbi.nlm.nih.gov/) and 3D structure (b) of berbamine [88]

Fig. 4 *Berberis amurensis* in different stages of growth (https://www.inaturalist.org/) (https://www.biolib.cz/)

Ulmus davidiana Planch., a tree widely distributed in China, Korea, and Japan, has shown anti-tumor effects against human leukemia cells such as HL-60, K562, and THP-1 cell lines [89]. Another promising function of certain herbs is their inhibitory activity against Jurkat tumor cell lines. *Thymus vulgaris* L., which exhibits such activity, further underscores the potential of medicinal herbs in leukemia treatment [11, 71, 90].

Several medicinal plant species, including *Aglaia leptantha* Miq. and *Combretum caffrum* Kuntze, have demonstrated their efficacy, particularly in the treatment of CLL. *A. leptantha*, found in Southeast Asia and tropical regions of East Asia, contains silvestrol, that induces the production of total growth inhibition (TGI) and ultimately leads to cell death [68, 91]. *C. caffrum*, native to Asia, contains CA4P (combretastatin-A4 prodrug) and has a specific effect on CLL cell lines [92]. CA4P is considered an anti-tubulin/anti-angiogenic agent which induces cell death mainly by mitotic catastrophe [93]. *Moringa oleifera* Lam., also known as drumstick tree, contains quercetin (a flavonoid with antioxidant properties), which inhibits the viability of both AML and ALL cell lines [94, 95]. Additionally, extracts from aerial parts of *Arctium lappa* L. have demonstrated anti-cancer effects on the K562 cell line, further expanding the list of medicinal plants with potential anti-leukemic properties [71]. For a comprehensive list of Asian medicinal plants with potential anti-leukemic properties and insights into their mechanisms of action, refer to Table 1.

Table 1 Asian medicinal plants, their phytochemicals, and their mechanisms of action in leukemia treatment or prevention.

Number	Plant scientific name	Origin/grows in	Plant material/ compound(s)	Function	Reference(s)
1	*Aglaia leptantha* Miq.	South East Asia and tropical regions of East Asia	Silvestrol	ProducesTGI and induces cell death in B-leukemia cells. Affects CLL, ALL, and P388.	[68, 91]
2	*Alhagi pseudalhagi* (M.Bieb) Desv. ex Shap.	Iran	Extracts from aerial parts	Acts against K562 (AML) and Jurkat tumor cell lines (T cell leukemia).	[11, 71]
3	*Allamanda cathartica* L.	South East Asia	Allamandin derivatives	Acts against the P388 leukemia in mice (*in vivo*).	[94, 96]
4	*Alstonia scholaris* (L.) R.Br.	Southern China, tropical Asia, India	Extract of stem bark	Has an anti-cancer effect on Dalton's lymphoma ascites (DLA) cells in culture.	[95, 97]
5	*Alstonia venenata* R.Br.	Tropical regions of Asia	Leaf extract	Has cytotoxic and antioxidant effects.	[95, 97, 98]
6	*Amoora rohituka* (Roxb.) Wight & Arn.	South Asia	Amooranin (triterpenic acid) from bark	Has a cytotoxic effect against multidrug-resistant leukemia.	[99]
7	*Annona glabra* L.	Asia	Seed, leaf, and pulp extracts	Has cytotoxic activity against a leukemia cell line and its multidrug-resistant-derived cell line. Seed extracts induce apoptosis and necrosis in CEM and CEM/VLB cell lines and upregulate the expression of cyclin kinase inhibitors, leading to cell arrest at the G0/G1 phase. The most potent extract is derived from seeds.	[78]
8	*Arctium lappa* L.	Asia	Shoot extract	Acts against K562 (AML) and Jurkat tumor cell lines (T cell leukemia).	[71, 100]
9	*Arctium lappa* L.	Asia	Root extract	Induces mitochondrial-mediated caspase-dependent apoptosis in Jurkat human leukemic T cells and cell death.	[100, 101]
10	*Artemisia annua* L.	China	Leaf extract, artemisinin	Artesunate has anti-leukemic activity in AML. Kills human leukemia cells.	[102–105]
11	*Artemisia argyi* H.Lév. & Vaniot	South East Asia	Plant extract (scopoletin and isocopoletin)	Acts against P388 murine leukemia cell line. Has repressive effects against CCRF-CEM human leukemia cells.	[106, 107]
12	*Astragalus trimestris* L. (syn. *Astragalus membranaceus* Moench)	China, Asia	Lectin from root	Induces apoptosis in CML cells (K562).	[108–110]
13	*Barleria prionitis* L.	South Asia	Leaf extract	Has cytotoxicity against THP-1 (AML cell line).	[111–114]
14	*Berberis amurensis* Rupr.	Asia	Berbamine	Causes apoptosis in Gleevec-sensitive and -resistant Ph+ CML cells.	[86]
15	*Bidens pilosa* L. (syn. *Bidens Pilosa* var. *minor* (Blume) Sherff)	Asia	Whole plant extract	Acts against L1210, P3HR1, Raji, U937, and K562 leukemic cell lines.	[115, 116]
16	*Blumea balsamifera* (L.) DC.	Asia	Flavonoid quercetin	Enhances TRAIL activity in human leukemia cells, has anti-proliferative activity, and induces apoptosis.	[117, 118]
17	*Blumea lacera* (Burm.f.) DC.	Bangladesh and India	Dried plant	Acts against K562, U937, L1210, and P3HR1 leukemic cell lines.	[99, 119]
18	*Boswellia sacra* Flück.	Asia	AKBA (acetyl-11-keto-β-boswellic acid) and KBA (11-keto-β-boswellic acid)	Has antimicrobial activity. Inhibits the cell growth and proliferation by inhibiting topoisomerase Ⅰ and Ⅱa.	[120–122]
19	*Boswellia serrata* Roxb.	Asia especially India	Triterpenediol (TPD)	Induces apoptosis in human HL-60 leukemic cells by producing oxidative stress in cancer cells.	[111, 113, 123, 124]
20	*Calluna vulgaris* (L.) Hill	Asia	Ursolic acid from flowers	Has anti-proliferative activity against HL-60 leukemic cells *in vitro.*	[125–127]
21	*Calotropis procera* (Aiton) Dryand.	South Asia	Extract of stem	Has cytotoxicity against human leukemia HL-60 cell line.	[99]
22	*Calotropis procera* (Aiton) Dryand.	India	Bufalin, Digoxin	Induces apoptosis in HL-60, THP-1, K562, U937 and ML1 cell lines (*in vitro*).	[128]
23	*Camellia sinensis* (L.) Kuntze	East and South East Asia	Leaf aqueous extract	Decreases the viability of AML cells in humans (HL-60) and mice (C1498).	[31, 52, 111]
24	*Cannabis sativa* L.	Asia	Metallic nanoparticles, especially gold nanoparticles from the leaves	Acts against lymphoblastic leukemia cells and acute T cell leukemia.	[129, 130]

25	*Carapichea ipecacuanha* (Brot.) L.Andersson (syn. *Cephaelis acuminate* H.Karst.)	Asia	Alkaloids	Acts against P388 and L1210 leukemia cell lines.	[111, 131]
26	*Catharanthus roseus* (L.) G.Don	China and Malaysia	Vinblastine, vincristine, vinflunine, catharanthine	Acts as an anti-proliferative and anti-cancer agent and induces apoptosis in Jurkat cells and K562.	[10, 86, 132]
27	*Cephalotaxus harringtonii* (Knight ex. J.Forbes) K.Koch (syn. *Cephalotaxus harringtonii* var. *drupacea* (Siebold & Zucc.) Koidz.)	East Asia	Alkaloids harringtonine, homoharringtonine, and isoharringtonine	Acts against myeloid leukemias.	[111, 131, 133]
28	*Cichorium intybus* L.	Iran	Shoot extract	Acts against K562 (AML) and Jurkat tumor cell lines (T cell leukemia).	[11, 71]
29	*Citrus hystrix* DC.	Asia	Crude extracts and essential oils from the leaves	Has cytotoxic and anti-proliferative activity against the murine leukemia (P388) cell lines.	[134, 135]
30	*Clausena lansium* (Lour.) Skeels	South East Asia	Stem	Has cytotoxic activity against K562 cell line.	[136]
31	*Clinacanthus nutans* (Burm.f.) Lindau	Asia	Fresh leaves extract	Acts against K562 cell line.	[136]
32	*Combretum caffrum* Kuntze	Asia	CA4P	Acts against ALL and CLL. Induces cell death by mitotic catastrophe in human B-lymphoid tumors.	[8, 92]
33	*Coptis chinensis* Franch.	China	Rhizome (rhizoma coptidis)	The alkaloid berberine downregulates telomerase activity and nucleophosmin/B23, resulting in apoptosis in HL-60 human leukemia cell lines.	[102]
34	*Coscinium fenestratum* (Gaertn.) Colebr.	Asia	Berberine	Has cytotoxic activity against HL-60 leukemia cells.	[137, 138]
35	*Cosmos caudatus* Kunth	South East Asia	Ethanol extract	Acts against P388 murine leukemia cell line.	[106]
36	*Curcuma longa* L.	Asia	Curcumin	Acts against K562 cell line in human CML.	[86, 139]
37	*Daucus carota* L.	West Asia	Extract of umbels and fruits	Has a high cytotoxic effect on 11 AML cell lines (HL-60, U937, ML1, ML2, Mono-Mac-1, Mono-Mac-6, KG-1, MV-4-11, TF1-vRaf, TF1vSrc, and TF1HaRas).	[48, 140]
38	*Dracocephalum kotschyi* Boiss.	South West Asia especially Iran	Flavonoid aglycones, naringenin, cirsimaritin, penduletin apigenin, luteolin, isokaempferide, xanthomicrol, calycopterin	Has anti-proliferative activities against several cancer cell lines including human HL-60 leukemia.	[141–143]
39	*Dracocephalum kotschyi* Boiss. in combination with *Peganum harmala* L.	*Peganum harmala* grows in Central Asia and Middle East	Flavonoids luteolin, naringenin, apigenin, isokaempferoide, cirsimaritin, penduletin, xanthomicrol, and calycoperin	Acts against HL-60 cell lines.	[141, 143, 144]
40	*Eleutherococcus* spp. Maxim.	Eastern Asia	Roots and leaves	Exhibits cytotoxic effect towards leukemic HL-60 cells. The root extract of *E. divaricatus* shows the highest proapoptotic and cytotoxic effect on the HL-60 cell line.	[145, 146]
41	*Etlingera elatior* (Jack) R.M.Sm.	South East Asia	Flowershoots	Inhibits cell proliferation in MV4-11 and K562 leukemic cells, mainly through apoptosis in a dose-dependent manner.	[147, 148]
42	*Euphorbia hirta* L.	Asia	Powder and liquid extract	Acts against HL-60 leukemic cells *in vitro* (AML).	[111, 113, 149]
43	*Euphorbia kansui* Liou ex S.B.Ho	China	Diterpenes kansuinines and ingerols from root	Has anti-tumor activity in different types of cancer such as leukemia.	[150, 151]
44	*Eurycoma longifolia* Jack	Malaysia, Vietnam, Thailand, Myanmar, Cambodia, and Indonesia	Bark, stem, and root	Eurycomanone and eurycomanol have cytotoxic effects against murine lymphocytic leukemia (Jurkat and K562 cell lines).	[55]
45	*Garcinia hanburyi* Hook.f.	Central and South East Asia	Gambogic acid	Has cytotoxic activity against K562 cell line. Downregulates mRNA levels in HL-60 and Jurkat cells, including apoptosis.	[92, 152]

46	*Garcinia indica* (Thouars) Choisy	India	Fruit extract	Garcinol induces apoptosis and growth inhibition in both AML and CML, especially in the human HL-60 leukemia cell line.	[80, 102]
47	*Garcinia mangostana* L.	Southeast Asia, South West India	Xanthones	Inhibits the cell growth in human leukemia HL-60, K562, U937, and NB-4 cell lines.	[152, 153]
48	*Getonia floribunda* Roxb. (syn. *Calycopteris floribunda* (Roxb.) Lam. ex Poir.)	Asia	5,30-dihydroxy-3,6,7,8,40 - pentamethoxy-flavone and 30 - amino-5-hydroxy-3,6,7,8,40 – penta-methoxyflavone	Has anti-proliferative activity against HL-60, HL-60R, and K-562 leukemia cell lines.	[154]
49	*Glycine max* (L.) Merr.	South East Asia	Genistein	Causes apoptosis in Jurkat cells. Affects the proliferation and differentiation of human U937 leukemic cell line.	[31, 155–158]
50	*Glycyrrhiza glabra* L.	Iran	Shoot extract Root extract	Acts against K562 (AML) and Jurkat tumor cell lines (T cell leukemia).	[11, 71]
51	*Gymnanthemum amygdalinum* (Delile) Sch.Bip. (syn.*Vernoria amygdalina* Delile)	Asia		Has a cytotoxic effect against primary cells harvested from AML and ALL patients.	[136, 159]
52	*Hibiscus cannabinus* L.	Asia	Seed oil	Induces cell death in human leukemia HL-60 and K562 cell lines and murine myelomonocytic WEHI-3H cells through induction of apoptosis.	[32, 160]
53	*Houttuynia cordata* Thumb.	China	Crude extract from stem and leaf	Acts against U937, HL-60 and Molt4 leukemic cell lines.	[116, 161]
54	*Hyptis suaveolens* (L.) Poit.	Asia	Leaf extract	Inhibits the viability of Jurkat cells and human T-leukemia cells.	[81]
55	*Ixeris chinensis* (Thunb. ex Thunb.) Nakai	China	Extract	Inhibits the growth of leukemia cells.	[119, 162]
56	*Jatropha curcas* L.	Asia and Middle East	α-amyrin and taraxerol (triterpenes) and the diterpenes: jatrphol, jatropholone A, B, and phorbols	The seed powder of the plant, when combined with honey, has been traditionally used to treat tumors. The plant extract shows strong inhibitory activity against P388 lymphocytic leukemia (*in vitro* and *in vivo*).	[163, 164]
57	*Jatropha gossypiifolia* L.	South Asia	Lignans, falodone, and jatrophone	Has cytotoxic and anti-proliferative effect against P338 lymphocytic leukemia cell line.	[99]
58	*Kaempferia galanga* L.	South East Asia	Rhizome	Has anti-proliferative activity against CCRF-CEM leukemia cells.	[165, 166]
59	*Kaempferia parviflora* Wall. ex Baker	South East Asia	Ethanolic extract	Exhibits cytotoxicity against U937 cells (in a dose- and time-dependent manner). Has an anti-apoptotic role at low concentrations but induces apoptosis at high doses (activating caspase-3 at high doses).	[167, 168]
60	*Lavandula angustifolia* ssp. *angustifolia* (syn. *Lavandula officinalis* Chaix)	South West Asia to India	Essential oil	Induces apoptosis in HL-60 leukemic cells (in a dose-dependent manner).	[111, 169]
61	*Medicago sativa* L.	China	Flavonoids medicarpin and millepurpan	Medicarpin and millepurpan induce apoptosis and overcome multidrug resistance in P388 leukemic cells. Has anti-proliferative activity against the DU-145 cell line.	[71, 90, 170–174]
62	*Mentha longifolia* (L.) L.	Iran	Shoot extract	Acts against K562 (AML) and Jurkat tumor cell lines (T-cell leukemia).	[11, 71, 121]
63	*Momordica charantia* L.	Asia	Ripe fruit Leaf extract Plumericin (lactone)	Has *In vitro* anti-leukemic activity. Inhibits the proliferation of human HL-60 leukemia cell line. Inhibits the proliferation of K562 and NB4 cell lines.	[175, 176]
64	*Moringa oleifera* Lam.	Asia especially North Western India	Quercetin	Shows anti-tumor activity and is active against leukemia cells *in vitro*, inhibiting the viability of AML and ALL cells.	[94, 95, 177]
65	*Morus alba* L.	Eastern and Central China	Roots	Albanol A acts against HL-60 human leukemia cell line.	[102, 178]

66	*Ocimum basilicum* L.	Tropical and subtropical regions of Asia	Extract from leaves Shoot extract Essential oil	Has anti-proliferative and anti-cancer activitiy in the murine P388 leukemia cell line.	[121, 179, 180]
67	*Olea europaea* L.	Asia	Leaf extract	Inhibits cell growth in HL-60 human promyelocytic leukemia and proliferation in Jurkat.	[121, 181]
68	*Pereskia sacharosa* Griseb.	Asia	Triterpines, flavonoid, saponin, and tannins from leaves	Inhibits proliferation in MV4-11 and K562 leukemic cells, mainly through apoptosis in a dose-dependent manner.	[136, 147, 182]
69	*Phaleria macrocarpa* Boerl.	South Asia	Fruit extract	Gallic acid in the fruit extract induces apoptosis in leukemic cell lines.	[102, 183]
70	*Phaleria macrocarpa* Boerl.	South Asia	Benzophenone glucoside compound from bark	Has an inhibitory effect on L1210 leukemic cell line (mouse).	[184]
71	*Phyllanthus niruri* L.	South and South East Asia	Lignans Extract of whole plant	Has cytotoxic effect on K562 cells.	[111, 113, 185, 186]
72	*Piper betle* L. (syn. *Piper betel* Blanco)	South East Asia especially India	Leaf extract (tannins, Chl, NaChl)	Inhibits the growth of human chronic myelogenous leukemia in xenograft models. Chl, acts against K562 leukemic cell line. NaChl, acts against KU812 and KCL22 leukemic cell lines.	[57, 82, 84]
73	*Plantago asiatica* L.	Central Asia	Whole plant extract	Inhibits the proliferation of K562, U937, P3HR1, HL-60, and CCRF-CEM leukemic cells.	[187]
74	*Plantago major* L.	Central Asia	Allantoin, aucubin, ursolic acid, flavonoids, asperuloside	Has anti-proliferation, cytotoxicity, and growth inhibition effects on MCF-7, MDAMB-231, HelaS3, A549, KB, THP-1, and AML cells.	[10, 187, 188]
75	*Platycodon grandiflorus* A.DC.	China	Roots	Platycodin D (a triterpene saponin) induces cell death and apoptosis in the human U937 cell line.	[102]
76	*Prunella vulgaris* L.	North Eastern Asian countries such as Korea, China, and Japan	Ursolic acid DHURS (2α,3α-dihydroxyurs-12-en-28-oic acid)	Inhibits lipoxygenase and cyclooxygenase in HL-60 leukemic cell line. Induces apoptotic DNA fragmentation in Jurkat T cells in human acute leukemia.	[126, 189]
77	*Psychotria serpens* L.	Asia	Ursolic acid	Has a cytotoxic effect against HL-60 leukemic cells.	[126, 190, 191]
78	*Punica granatum* L.	Asia	Peels Juice and peels Seeds/punicic acid	The peels of the plant inhibit cell proliferation in MV4-11 and K562 leukemic cells, mainly through apoptosis in a dose-dependent manner. The juice and peels of the plant promote apoptosis in human HL-60 leukemia cells. Punicic acid from seeds has cytotoxic effects in leukemia cells via lipid peroxidation.	[147, 192, 193]
79	*Rheum ribes* L.	Iran	Shoot extract	Acts against K562 (AML) and Jurkat tumor cell lines (T-cell leukemia).	[11, 71]
80	*Rubus parvifolius* L.	Traditional Chinese herb	Saponins	Causes apoptosis and changes in cell cycle checkpoints in human CML cells.	[87]
81	*Salvia miltiorrhiza* Bunge	Asia	Cryptotanshinone (CPT) from roots 15,16-Dihydrotanshinone (DHTS)	Induces apoptosis in tumor cells *in vitro* or inhibits tumor cell growth. Causes cell cycle arrest in the G1 phase through downregulation of cyclin D1 expression by CPT. Inhibits proliferation of human glioma cells by suppressing STAT3 signaling. Causes apoptosis in human AML type 3 (HL-60 cells). Inhibits the proliferation of K562 human CML.	[158, 194]
82	*Satureja bachtiarica* Bunge	Iran	Shoot extract	Induces apoptosis and has strong inhibitory activity against Jurkat tumor cells (200 µg/ml, >80% inhabitation) and the K562 cell line (200µg/ml, >50% inhabitation).	[11, 71, 195]
83	*Satureja hortensis* L.	Iran	Shoot extract	Strongly inhibits cell growth in: - Jurkat tumor cells (200 µg/ml, >80% inhabitation) - K562 cell line (200µg/ml, >50% inhabitation)	[10, 11, 71, 196]
84	*Scurrula parasitica* L.	Asia	Extract of leaves	Has anti-cancer and inhibitory activity against AML (HL-60 cell line). Flavonoids cease the cell cycle of mutant cells at the G0-G1 phase.	[102, 197]

				Causes apoptosis of cancer cells *in vivo* by downregulating cyclinD1, Bcl-2, and Ki-67 protein expression and upregulating Bax protein expression.	
85	*Scutellaria baicalensis* Georgi	China	Baicalin Wogonoside	Baicalin is a selective and potent inhibitor of HTLV-1 replication *in vitro*, inhibits gag expression in chronically infected T and B cells, induces apoptosis in K562 cells, causes cell cycle cease, and inhibits cell growth by increasing GATA-1 expression and binding in CML. Wogonoside increases PLSCR1 (phospholipid scramblase 1) transcription, a cell cycle regulator, exerting anti-cancer effects in AML.	[92, 198]
86	*Scutellaria orientalis* ssp. *Carica* J.R.Edm.	Western Turkey	Baicalein and wogonin	Baicalein has anti-proliferative effects, and wogonin exhibits genotoxic properties.	[199]
87	*Simarouba glauca* DC.	Asia (Malaysia)	Leaf extracts/petroleum ether	Inhibits the proliferation of K562 cells.	[200]
88	*Sophora flavescens* Aiton	Asia	Root (Kushen) Sophoranone	Matrine (an alkaloid isolated from Kushen) has anti-tumor activity (reduces leukemia cell survival and cytotoxic effects (induces apoptosis in K562 and U937 human leukemic cell lines).	[201, 202]
89	*Sophora tonkinensis* Gagnep.	Asia	Roots	Inhibits cell growth and induces apoptosis in the U937 leukemic cell line.	[201]
90	*Sophora tonkinensis* var. *tonkinensis* (syn. *Sophora subprostrata* Chun & H.Y.Chen)	Asia	Roots	Has antioxidant and cytotoxic activity.	[111, 201]
91	*Spermacoce articularis* L.f.	Tropical Asia	Ursolic acid	Has a cytotoxic effect against HL-60 leukemic cells.	[191]
92	*Spermacoce exilis* (L.O.Williams) C.D.Adams	Malaysia	Ursolic acid	Has a cytotoxic effect against HL-60 leukemic cells.	[191]
93	*Spermacoce latifolia* Aubl.	Malaysia	Ursolic acid	Has a cytotoxic effect against HL-60 leukemic cells.	[191]
94	*Tanacetum parthenium* (L.) Sch.Bip.	Central Asia	Parthenolide	Induces apoptosis in AML cells (THP-1 leukemia cells).	[92, 203]
95	*Thymus daenensis* Čelak.	Iran	Shoot extract	Has strong inhibitory activity against Jurkat tumor cells (200 µg/ml, >80% inhabitation) and the K562 cell line (200 µg/ml, >50% inhabitation).	[11, 71, 204]
96	*Thymus vulgaris* L.	Asia	Shoot extract	Has strong inhibitory activity against Jurkat tumor cells (200 µg/ml, >80% inhabitation) and the K562 cell line (200 µg/ml, >50% inhabitation).	[11, 71, 90, 100]
97	*Tinospora cordifolia* (Willd.) Miers ex Hook.f. & Thomson	India	Diterpenoid lactones	Induces apoptosis in leukemia cells by activating caspase-3 and BAX and inhibiting Bcl-2.	[111, 113, 205]
98	*Tithonia diversifolia* (Hemsl.) A.Gray	Asia	Sesquiterpenoids	Inhibits the growth of leukemia cells and induces cell differentiation in the HL-60 human promyelocytic leukemia cell line.	[119, 206]
				Has anti-proliferative activity.	
99	*Trigonella foenum-graecum* L.	Asia	Extract of the seeds	Acts against K562, U937, and L1210 leukemia cell lines.	[121, 207, 208]
				Has cytotoxic effects against CML.	
100	*Trigonella foenum-graecum* L.	Asia	Extract of dry seeds	Causes membrane disintegration and large vacuole formation in Jurkat cell line.	[208]
101	*Ulmus davidiana* Planch.	Korea, China, and Japan	Mansonone E	Has an anti-tumor effect on leukemia cell lines HL-60, K562, THP-1, and U937 in a hollow fiber assay.	[89]
102	*Vachellia nilotica* (L.) P.J.H.Hurter & Mabb. (syn. *Acacia nilotica* (L.) Willd. ex Delile)	Asia	Apigenin	Induces leukemia cell cycle blockage at the G2/M phase in K562 cells (CML).	[77, 209, 210]
103	*Withania somnifera* (L.) Dunal	Asia	Withaferin A from roots and leaves	JNK and Akt pathway inhibition and NF-κB suppression result in apoptosis in human leukemic cells.	[211–213]
				Increases Bax levels via MAPK signaling, inducing a mitochondrial death cascade.	
104	*Withania somnifera* (L.) Dunal	Asia	Crude from the leaves	Has anti-proliferative and proapoptotic effects in different leukemia cell lines.	[214]

CONCLUSION and Future Perspectives

While chemotherapy remains the standard treatment for leukemia, its severe side effects and the growing issue of drug resistance, particularly in cases like CLL, where chromosomal deletions or mutations lead to treatment failure, highlight the urgent need for safer and more effective alternatives. Medicinal plants, with their diverse bioactive compounds, offer a promising solution by targeting leukemia through multiple mechanisms, including apoptosis induction, angiogenesis suppression, selective cytotoxicity, and immune modulation. Promising species such as *Piper betle* (hydroxychavicol, eugenol), *Garcinia indica* (xanthochymol), *Berberis amurensis* (berbamine), and *Moringa oleifera* (quercetin) demonstrate potent anti-leukemic effects, even against drug-resistant cell lines, while minimizing toxicity to healthy cells. Additionally, emerging research on *Artemisia annua* (artemisinin), *Curcuma longa* (curcumin), and *Withania somnifera* (withaferin A) suggests further potential due to their ability to target cancer stem cells and overcome chemoresistance.

However, several critical research gaps and unmet clinical needs must be addressed to translate these findings into viable therapies. A major challenge lies in optimizing the bioavailability of plant-derived compounds, as many exhibit poor solubility and absorption that limit their therapeutic potential. Advanced delivery systems such as nanoformulations and lipid-based carriers could help overcome these pharmacokinetic limitations. Another crucial area is the development of effective combination therapies that pair plant bioactives with conventional drugs, such as curcumin with imatinib or artemisinin with venetoclax, to enhance efficacy while reducing the need for chemotherapy doses. Furthermore, the field requires a deeper investigation into subtype-specific mechanisms, particularly for high-risk molecular variants such as TP53-mutated CLL versus BCR-ABL1-positive CML. The identification of predictive biomarkers for treatment responsiveness could enable more personalized approaches to plant-based therapies. Alongside these needs, the requirement for standardized extraction methods, comprehensive pharmacokinetic studies, and rigorous clinical trials remains essential to validate efficacy and safety in human patients.

To fully realize this potential, interdisciplinary collaboration among pharmacologists, oncologists, and traditional medicine experts is essential. The integration of advanced technologies, from AI-driven drug discovery to innovative delivery platforms, could accelerate the development of plant-based anti-leukemic therapies. The shift from conventional chemotherapy to plant-based treatments, supported by rigorous scientific validation, could revolutionize leukemia care, offering more effective, affordable, and patient-friendly solutions. By addressing these unmet needs and bridging current knowledge gaps, medicinal plants may indeed transition from complementary options to first-line adjuvants or alternatives, marking a transformative leap in oncology and emerging as the cornerstone of next-generation leukemia therapies.

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Conflict of Interests

The authors have no conflicts of interest.

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