



## Research Article

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### APOPTOSIS-DRIVEN ANTI-LEUKEMIC EFFECTS OF A POLYHERBAL COMBINATION: BIOLOGICAL ACTIVITY AND CHEMICAL CHARACTERIZATION

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#### ABSTRACT

Hematologic malignancies impose a major burden in India; while modern therapies improve survival, toxicity, relapse, and cost limit benefit. We investigated a proprietary Ayurvedic preparation used clinically as a 1:1 mixture of an anti-cancer extract (Can-n-Cer-BC) and a supportive formulation (Detoxigab). Chemical profiling by untargeted LC-MS and in-vitro testing (DPPH scavenging, CellTiter-Glo® viability, and caspase activity) were performed. LC-MS revealed ~35 features across 3.8–17.0 min with a right-tailed distribution. Several high-area library matches resembled siloxanes/organometallics typical of background; these were treated as provisional and reserved for quality control, whereas lower-abundance features were prioritized as putative phytochemicals. Functionally, the combined extract displayed minimal DPPH activity at  $\leq 1,000 \mu\text{g/mL}$ , rising to ~40% at  $2,500 \mu\text{g/mL}$  and ~55–60% at  $10,000 \mu\text{g/mL}$ . In contrast, at  $2,500 \mu\text{g/mL}$  the extract caused a ~90% loss of ATP-based viability (signal ~10% of control) and increased caspase activity to ~135–140% of control. These findings indicate that antioxidant effects emerge only at high concentrations and are unlikely to account for the steep fall in viability; rather, apoptosis appears to be a principal mechanism of action for the mixture, plausibly mediated by multiple low-abundance phytochemicals acting in concert. Quality-control steps (system blanks, targeted MS/MS with internal standards, replicate extractions, and ICP-MS for metals) were implemented to secure chemical attributions. Overall, the Can-n-Cer-BC/Detoxigab combination exhibits potent, caspase-associated anti-leukemic activity in vitro and warrants dose response, selectivity, mechanistic confirmation (e.g., Annexin V/PI) and bioassay-guided fractionation to define active principles and enable standardized, safe development as a potential adjunct to blood-cancer care.

**Keywords:** Blood cancer, Ayurveda, polyherbal extract, DPPH, apoptosis, LC-MS, RPMI 8226, caspase 3/7

#### INTRODUCTION

Hematologic malignancies including leukaemia, lymphoma, and multiple myeloma account for a meaningful share of India's cancer burden<sup>1</sup>. Recent national estimates suggest ~1.46–1.56 million new cancer cases annually, with leukaemia comprising ~7.1% of cancers in men and ~6.4% in women; in children, lymphoid leukaemia is the leading cancer diagnosis. These trends mirror global increases and highlight a sustained public-health need<sup>2</sup>.

Standard care for blood cancers has advanced rapidly but remains heterogeneous and resource-intensive. Chronic myeloid leukaemia outcomes were transformed by tyrosine-kinase inhibitors (TKIs) such as imatinib and its successors; yet long-term therapy, resistance, and adherence remain challenges<sup>3</sup>. Acute myeloid leukaemia care still relies on intensive chemotherapy with risk-adapted use of targeted agents and allogeneic stem-cell transplant. In chronic lymphocytic leukaemia, time-limited regimens built on BTK and BCL-2 inhibition are replacing chemo-immunotherapy. Multiple myeloma therapy increasingly uses antibody-containing quadruplets, followed by autologous transplant when feasible. Cellular immunotherapies have also arrived in India: the indigenous anti-CD19 CAR-T product (actalycabtagene autoleucel; NexCAR19) secured national approval in 2023 for relapsed/refractory B-cell malignancies<sup>4</sup>.

Despite these advances, many patients face toxicity (cytopenias, infections, organ injury), relapse, and substantial financial burden that impede access and adherence - issues that are especially pronounced in low- and middle-income settings. Financial toxicity studies from India document major out-of-pocket

expenditure and treatment discontinuation risks, motivating exploration of adjunctive strategies that may improve tolerability, quality of life, or immune competence during or after conventional care.

Traditional medical systems in India, particularly Ayurveda, offer polyherbal formulations with pleiotropic bioactivity (anti-inflammatory, antioxidant, immunomodulatory, and pro-apoptotic effects) that can be studied rigorously as complementary approaches<sup>5</sup>. A recent peer-reviewed case report from the national AYUSH network described an elderly patient with chronic lymphocytic leukaemia who, during integrated Ayurveda Siddha care, experienced fewer infections and symptomatic improvement alongside stabilization of hematologic parameters an observation that supports formal evaluation of such interventions in hematologic malignancies.

Motivated by this rationale, we investigated an Ayurvedic polybotanical extract whose components include several plants with documented anticancer-relevant pharmacology. Guduchi (*Tinospora cordifolia*) exhibits immunomodulatory and cytotoxic activities and can trigger mitochondrial apoptosis in tumour models; multiple reviews and preclinical studies support these effects<sup>6</sup>. Haritaki (*Terminalia chebula*) yields chebulinic acid, which induces apoptosis in human myeloid leukaemia cells. Yashtimadhu (*Glycyrrhiza glabra*) derivatives (e.g., glycyrrhetic/glycyrrhizic acids) reduce leukaemia-cell viability and activate intrinsic apoptosis<sup>7</sup>. Moringa (*Moringa oleifera*) extracts suppress proliferation and promote apoptosis across cancer lines. Neem (*Azadirachta indica*) modulates the tumour microenvironment and cell survival pathways<sup>8</sup>. Curcuma species (*Curcuma aromatica* and *Cinnamon. caesia*) provide

curcuminoids and essential-oil constituents with well-characterized anticancer actions, including apoptosis induction. *Centella asiatica* supplies asiatic acid, a triterpenoid with emerging antitumor activity and relevance to survival pathways overexpressed in hematologic cancers<sup>9</sup>. Together, this polypharmacology provides a mechanistic basis to test the formulation in blood-cancer settings.

Even though alternative medicine is the need of the hour, at the same time, rigorous safety science is essential. Some botanicals noted on comparable labels of traditional products e.g., *Aristolochia spp.* (aristolochic acids) and *Strychnos nux-vomica* (strychnine) are associated with serious toxicities and, in the case of aristolochic acids, which are considered group 1 human carcinogen; any formulation containing such species requires strict exclusion or validated quality control<sup>10</sup>. Our study accordingly emphasizes analytical characterization and monitoring to ensure patient safety while probing biological activity.

In this work, we therefore evaluate the anti-leukemic potential of the selected Ayurvedic formulation using in-vitro and/or ex-vivo models relevant to hematologic malignancies, with the dual goals of (i) defining cytostatic and pro-apoptotic effects and (ii) establishing a foundation for safe, evidence-based integration of Ayurvedic botanicals as adjuncts to modern blood-cancer care in India.

## MATERIALS AND METHODS

### Phytochemical Formulation

Two proprietary phytochemical formulations were procured from Dr. Gabha's: (i) Can-n-Cer-BC, supplied as an anti-cancer extract, and (ii) Detoxigab, intended to support healthy cells. Unless otherwise specified, all experiments used a 1:1 (v/v) mixture of the two preparations hereafter referred to as the combined extract (S1+S2). Formulations were used as supplied by the manufacturer and mixed immediately before use; product names are provided solely for identification and do not imply endorsement.

### LC-MS Chemical Profiling of the Combined Phytochemical Formulation

Phytochemical analysis was carried out using LC-MS outsourced at Audentes Labs. Chromatographic profiling revealed 18 distinct compounds in the "Blood Cancer" extract and 17 in the "Detox" component. Major bioactive compounds included platinum complexes, adamantane isothiocyanates, and phenylboronic esters.

### DPPH Free Radical Scavenging Assay

The free radical scavenging potential of the extracts were compared with that of the standard gallic acid. Working stock of DPPH (500µM) was prepared in 100% methanol. Different dilutions of the standard gallic acid (1-10µg/mL) were prepared. To a 96 well plate, 10µL of sample was added followed by the addition of 90µL of the working stock of DPPH. After incubation for 30 mins at RT in the dark, absorbance was measured at 517nm using BioTek Cytation 5.

$$\% \text{ Scavenging} = [(OD \text{ Control} - OD \text{ Sample}) / OD \text{ Control}] \times 100$$

### Cell Viability Assay (CellTiter-Glo®)

Assay were performed from the CellTiter-Glo® 2.0 assay kit according to manufacture protocol (Promega, USA). RPMI 8226 cells were seeded in 96 well plate format in 10 x 10<sup>3</sup> density and were treated with test compound and incubated for 48 h. After incubation, 100µl of CellTiter-Glo® 2.0 reagent were added to

100µl of medium containing cells and mixed for 2 min on shaker to induce cell lysis. Further plate was incubated for 10 min to stabilize the luminescent signal. Luminescence was recorded in luminescence channel on Cytation5 multiplate reader. Relative % Luminescence was calculated by normalizing untreated control as 100%.

### Caspase 3/7 Activity Assay

The RPMI 8226 cells were seeded at density of 10,00,000 cells /well in 6 well plate. On the same day, the cells were treated with the combined extract "S1+S2" for 24 hr. The Caspase 3/7 activity was determined as per manufacturer's instructions (Product: E-CK-A383 Caspase3/7 Activity Assay Kit, Elabsciences, USA). The caspase activity was calculated using the following formula:

$$\text{Caspase 3/7 activity} = [\text{OD sample} - \text{OD blank} / \text{Cpr sample}] / [\text{OD negative} - \text{OD blank} / \text{Cpr negative}] * 100$$

## RESULTS

### LC-MS Analysis

The extract exhibited a complex phytochemical profile. The "Blood Cancer" sample showed library matches to adamantane isothiocyanates, platinum-containing species (48.48% relative area), and ferrocene derivatives (Supplementary Material). The "Detox" sample showed library matches to siloxanes (47.9% relative area), tetrazoles, and phenylmercuric species.

However, several of the highest-abundance annotations including siloxanes, organometallic complexes, metal carbonyls, and phenylmercuric derivatives are well-documented LC-MS background contaminants originating from columns, solvents, tubing, seals, or laboratory hardware. These features were assigned with low spectral match factors and lacked diagnostic fragmentation supporting unambiguous structural confirmation. On this basis, these annotations are interpreted as probable analytical background rather than intrinsic constituents of the formulations.

Accordingly, biological interpretation in this study is restricted to non-siloxane, non-metal-associated features with plausible phytochemical origin and consistent chromatographic behavior. Metal-associated and siloxane-related signals are reported for transparency but are excluded from mechanistic or bioactivity attribution.

### DPPH Assay

The combined Ayurvedic extract showed little to no DPPH scavenging at ≤1,000 µg/mL (responses near zero, with small negative values at the lowest doses), followed by a clear dose-dependent rise at higher concentrations: activity was ~40% at 2,500 µg/mL and ~55–60% at 10,000 µg/mL (Figure 1). Thus, antioxidant capacity is detectable only at the high end of the tested range, reaching moderate scavenging at 10 mg/mL, with a threshold between ~1,000 and 2,500 µg/mL.

### CellTiter-Glo® Assay

Exposure to the combined extract produced a pronounced loss of metabolic viability in the tested blood-cancer cells. Normalized ATP-dependent luminescence dropped from 100% in untreated controls to ~10% at 2,500 µg/mL, indicating an ~90% reduction in viable signal (Figure 2). The very small error bars show this effect was consistent across replicates. Taken together, the data demonstrate potent cytotoxic activity at 2,500 µg/mL (i.e., an apparent IC<sub>90</sub> ≤ 2,500 µg/mL for this model). In context with the DPPH assay where activity emerges only above ~1,000 µg/mL the results suggest that at concentrations where redox-scavenging

begins to rise, the formulation simultaneously exerts strong anti-proliferative/cytotoxic effects on blood-cancer cells.

**Caspase 3/7 Activity**

At the apoptosis-inducing dose identified above (2,500 µg/mL), the combined extract increased caspase signal to ~135-140% of the untreated control (Figure 3). This elevation indicates activation of the caspase cascade consistent with engagement of

executioner caspases (e.g., 3/7) and an apoptotic mode of death. When read together with the CellTiter-Glo® data (~90% loss of ATP-based viability at the same dose), the results suggest that the extract's cytotoxicity in blood-cancer cells is accompanied by or driven by apoptosis rather than primary necrosis.

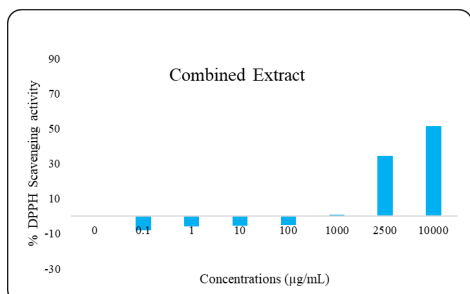


Figure 1: Radical scavenging activity of the combined extract

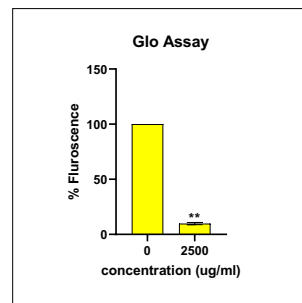


Figure 2: CellTiter-Glo® viability after exposure to the combined extract

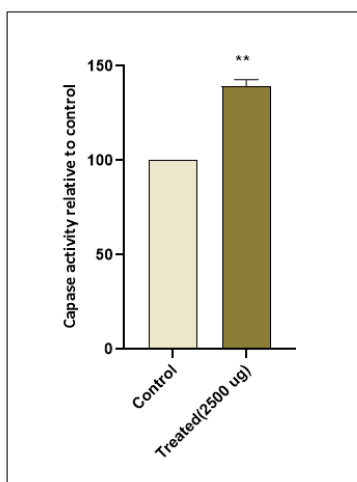
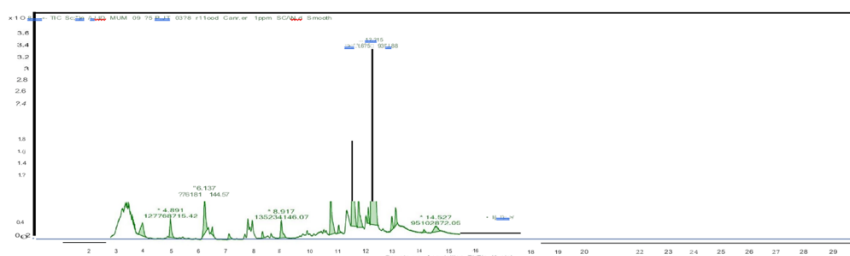
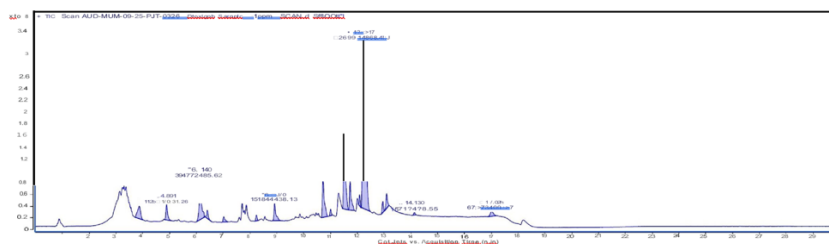


Figure 3: Combined phytochemical formulation increases caspase-3/7 activity



Peak	Component RT	Compound Name	Component Area	Area%	Formula	CAS#	Match Factor	Lib.
1	3.875	Adamantane, 1-isothiocyanato-3,5-	167477883	3.44	C13H19NS	136860-49-6	27.56	NIST20.L
2	4.891	2-(4-Methoxyphenyl)-1,3,2-	127768715	2.62	C9H11BO3	69519-11-5	29.23	NIST20.L
3	6.137	(1-Acetyl-2,3,4,5-	276181145	5.67	C18H23MnO4	12267-19-5	62.09	NIST20.L
4	6.411	Ferrocene, benzoyl-	38388653	0.79	C17H14FeO	1272-44-2	43.35	NIST20.L
5	7.019	Propane-1,3-diol 3-amino-4-	40614675	0.83	C10H12BN04	85107-40-0	24.1	NIST20.L
6	8.236	Mesoporphyrin ix dimethyl ester	36608071	0.75	C36H42N4O4	1263-63-4	12.06	NIST20.L
7	8.917	Pyrrrol[3-(bistrifluoromethylamino)-	135234146	2.78	C8F18N2O	1010322-36-0	18.84	NIST20.L
8	10.717	Diboroxane, bis-b-isopropyl-bis-B-	315267942	6.47	C16H32B2O5	1000151-23-5	15.35	NIST20.L
9	11.001	7-Bromo-2-chlorophenazine 5-	44278867	0.91	C12H6BrClN2O	29458-72-8	12.07	NIST20.L
10	11.501	Hexane-2,6-di(isonitrile), 1-	666050780	13.67	C9H12N2O2	1000159-54-3	1.42	NIST20.L
11	11.724	Thioether, bis[2-(9-	247475518	5.08	C28H48B2O2S	1000156-62-9	2.83	NIST20.L
12	12.076	2-a]benzimidazolium, 6-hydroxy-1,3-dimet	61890771	1.27	C16H16N4O3	27356-02-1	23.6	NIST20.L
13	12.215	Platinum, [bis[1,2-	2362595940	48.48	C28H64P4Pt	1000164-08-4	19.33	NIST20.L
14	12.933	Cobaltocene, 1,1'-diphenyl-	73583850	1.51	C22H18Co	93083-44-4	43.82	NIST20.L
15	13.073	1-(tert-Butyldimethylsilyl)-5-	98929460	2.03	C21H34ClNO2Si2	1000373-34-8	5.06	NIST20.L
16	14.1	1,3,2-Dioxaborinane, 2-[[(3.beta.)-	18677281	0.38	C32H55BO3	55162-73-7	8.29	NIST20.L
17	14.527	1H-Tetrazol-5-amine	95102872	1.95	CH3N5	4418-61-5	21.33	NIST20.L
18	16.927	4-i-Propylcarbamoyl-1-hydroxy-	67105523	1.38	C11H21N3O3	57006-62-9	5.63	NIST20.L

Figure 4: Supplementary



Peak	Component RT	Compound Name	Component Area	Area%	Formula	CAS#	Match Factor	Lib.
1	3.871	1-((1-Butyl-1H-tetrazol-5-	171482550.4	3.62	C21H27N7	1010483-69-1	22.81	NIST20.L
2	4.891	Phthalic acid, cyclobutyl ethyl ester	112537031.3	2.37	C14H16O4	1000315-41-1	53.28	NIST20.L
3	6.14	3-Chlorophenol.	394772485.6	8.33	C9H4ClFO2	1000467-45-9	20.13	NIST20.L
4	6.412	2,8-Dihydroxyquinoline.	56080089.69	1.18	C15H23NO2S2	1000463-06-3	18.02	NIST20.L
5	7.022	Germane, tetrapropyl-	44882816.45	0.95	C12H28Ge	994-65-0	34.46	NIST20.L
6	8.241	Boron, diethyl[1-	32820182.4	0.69	C14H33BO5In	130219-32-8	16.06	NIST20.L
7	8.92	Phenylmercuric propionate	151844438.1	3.20	C9H10HgO2	103-27-5	11.02	NIST20.L
8	10.725	2H-Naphtho[1,8-cd]isothiazole, 2-	330601269.5	6.98	C18H15N3S	1000317-16-6	24.9	NIST20.L
9	11.008	Sydnone, 3-(dimethylamino)-4-	35416073.3	0.75	C5H8N4O3	69978-11-6	15.4	NIST20.L
10	11.511	Nickel(II), [O-methyl-N(1),N(4)-	626430279.4	13.22	C16H13N3NiO3	1000164-81-5	29.31	NIST20.L
11	11.735	Chromium, tricarbonyl[eta.6-1,4-	215336765.5	4.54	C9H4Cl2CrO3	86409-62-3	7.4	NIST20.L
12	12.09	4-Phenoxy-2-phenyl-1-	53615219.32	1.13	C22H16O2	253801-45-5	19.86	NIST20.L
13	12.217	Permethyldecasilsesquioxane	2269914868	47.90	C10H30O15Si10	18106-15-5	21	NIST20.L
14	12.956	Methyl 2-	62107684.9	1.31	C23H48O3Si	1000424-20-9	7.86	NIST20.L
15	13.097	Benzenamine, 4-methyl-N-	98357311.26	2.08	C25H22NP	2327-67-5	32.22	NIST20.L
16	14.13	Tetradodecylammonium bromide	15712428.55	0.33	C48H100BrN	14866-34-3	25.61	NIST20.L
17	17.025	1,3,5-Trifluoro-2,4,6-	67273489.27	1.42	C24F18O3	141216-00-4	10.02	NIST20.L

Figure 5: Supplementary

## DISCUSSION

This study evaluated a combined phytochemical formulation prepared as a 1:1 mixture of the anti-cancer extract (Can-n-Cer-BC) and the supportive formulation (Detoxigab) for antioxidant capacity and anti-leukemic activity, and profiled its chemistry by LC-MS. Across assays, the mixture displayed modest direct radical scavenging but marked cytotoxicity with apoptotic features in blood-cancer cells. Specifically, DPPH activity was negligible below 1,000  $\mu\text{g}/\text{mL}$  and rose only at higher doses (~40% at 2,500  $\mu\text{g}/\text{mL}$ ; ~55–60% at 10,000  $\mu\text{g}/\text{mL}$ ), indicating that classical antioxidant effects emerge late and are unlikely to be the principal driver of the biological response at low- mid doses. By contrast, at 2,500  $\mu\text{g}/\text{mL}$  the formulation reduced CellTiter-Glo signal to ~10% of control (~90% loss of viable signal) and increased caspase activity to ~135-140% of control, supporting apoptosis-linked cytotoxicity at concentrations where DPPH begins to rise.

Taken together, these data suggest that the extract's anti-leukemic effect is not explained by bulk free-radical scavenging; instead, pro-apoptotic signalling is more consistent with the observations. Polyherbal components reported on the labels such as *Tinospora cordifolia*, *Terminalia chebula*, *Glycyrrhiza glabra*, *Moringa oleifera*, *Azadirachta indica*, and *Centella asiatica* are each associated in prior literature with immunomodulatory, redox-active, and mitochondrial apoptosis pathways (e.g., caspase-3/7 and PARP cleavage, BCL-2 family modulation). Although we did not test single-herb fractions here, the caspase increase alongside the steep drop in ATP is compatible with activation of the intrinsic apoptotic pathway. Follow-up experiments should map the time course of caspase-3/7,  $\Delta\Psi\text{m}$  loss, and Annexin V/PI staining; evaluate BAX/BCL-2 ratios and cleaved PARP by immunoblot; and assess whether the pan-caspase inhibitor z-VAD-fmk rescues viability, thereby establishing causality.

The LC-MS overview revealed ~35 chromatographic features across 3.8-17 min with a right-tailed distribution (few dominant peaks and many low-abundance signals), consistent with a polypharmacologic matrix. Several top library matches resembled siloxanes and organometallic species entities commonly arising from column bleed, tubing, or laboratory background. While these are unlikely to represent true actives, their prominence underlines the importance of analytical stringency before attributing bioactivity. The practical

implication is that the observed cytotoxicity more likely stems from the lower-abundance phytochemicals present in the tail of the distribution, which often include phenolics, triterpenoids, and alkaloids known to trigger apoptosis in leukaemia models.

From a translational perspective, the potent in-vitro effect at 2.5 mg/mL argues that the crude mixture contains active principles but also that potency enrichment is desirable. Bioassay-guided fractionation (solid-phase extraction followed by semi-prep HPLC) could identify fractions with stronger activity at lower mass doses; targeted MS/MS and authentic standards can then secure identifications. Once active sub-fractions are defined, combination testing (Bliss or Loewe models) can determine whether the two formulations act additively or synergistically, and whether the "detox" component modulates the therapeutic index (e.g., preserves normal PBMCs while sensitizing leukaemia cells). Clonogenic assays, cell-cycle profiling, and tests in additional hematologic lines (myeloid and lymphoid) will strengthen generalizability.

## CONCLUSION

The 1:1 combination of Can-n-Cer-BC and Detoxigab produces strong, caspase-associated cytotoxicity in blood-cancer cells, while exhibiting only moderate DPPH scavenging at high concentrations. The chemical profile indicates a complex mixture with a few dominant non-biogenic signals and many low-abundance constituents likely responsible for activity. Overall, the findings support the anti-leukemic potential of the combined Ayurvedic formulation and justify advancing to dose-response, mechanism-confirmation, selectivity, and fractionation studies under strengthened analytical QC to define active principles and a safe, standardized path toward adjunctive use.

## REFERENCES

- Bhutani M, Vora A, Kumar L & Kochupillai V. Lympho-hemopoietic malignancies in India. *Medical oncology* (Northwood, London, England) 2002;19: 141-150, doi: 10.1385/mo:19:3:141
- Bora K. Distribution of multiple myeloma in India: Heterogeneity in incidence across age, sex and geography. *Cancer epidemiology*. 2019; 59: 215-220, doi:10.1016/j.canep.2019.02.010

3. Guilhot F & Hehlmann R. Long-term outcomes of tyrosine kinase inhibitors in chronic myeloid leukemia. *Blood* 2025;145: 910-920, doi:10.1182/blood.2024026311
4. Rehman MA & Arshad H. A Discount on the Cost of Cancer: India's Homegrown CAR-T Cell Therapy. *Blood cell therapy*. 2024; 7:121-123, doi:10.31547/bct-2024-008
5. Pandey MM, Rastogi S & Rawat AK. Indian traditional Ayurvedic system of medicine and nutritional supplementation. Evidence-based complementary and alternative medicine: eCAM 2013;2013: 376327, doi: 10.1155/2013/376327
6. Verma R & Khan A. Antioxidant, Immunomodulatory and Anticancer potential of *Tinospora cardifolia* – A Review. *International Journal of Pharmacy and Biological Sciences*. 2018;8: 54-69
7. Chhabra S. *et al.* Chebulinic Acid Isolated From the Fruits of *Terminalia chebula* Specifically Induces Apoptosis in Acute Myeloid Leukemia Cells. *Phytotherapy research: PTR* 2017;31: 1849-1857, doi:10.1002/ptr.5927
8. Hao F, Kumar S, Yadav N & Chandra D. Neem components as potential agents for cancer prevention and treatment. *Biochimica et biophysica acta*. 2014;1846: 247-257, doi:10.1016/j.bbcan.2014.07.002
9. Dosoky NS & Setzer WN. Chemical Composition and Biological Activities of Essential Oils of Curcuma Species. *Nutrients*. 2018;10: 1196-1238 doi:10.3390/nu10091196
10. Arlt VM, Stiborova M & Schmeiser HH. Aristolochic acid as a probable human cancer hazard in herbal remedies: a review. *Mutagenesis*. 2002; 17:265-277, doi:10.1093/mutage/17.4.265

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