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Unlocking the Anti-Cancer Potential of Gallic Acid - A Scientific Scoping Analysis

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Abstract: Background: Cancer remains one of the top causes of death worldwide and, consequently, sparks the interest in the pursuit of safer and more effective therapeutic approaches. In recent decades, scientific interest has shifted to natural compounds for their potential therapeutic applications. Gallic acid (GA) is a polyphenolic compound derived from various fruits, herbs, and other plant-based sources that has emerged as a promising candidate based on its antioxidant and anticancer properties. This review intended to summarize the state of recent findings from 2010 to 2024 on anticancer effects, focusing on the mode of action of gallic acid, synergism with chemotherapeutic agents, and nanotechnology development in its delivery systems. Methods: A literature search of PubMed, ScienceDirect, and Google Scholar was made for relevant studies. Experimental and preclinical studies dealing with the role of gallic acid in different types of cancers were included. Results: The articles reviewed clearly show that gallic acid exerts its potent anticancer efficacy through a wide variety of mechanisms. It inhibits cancer cell proliferation, induces apoptosis, reduces inflammation, and suppresses metastasis of tumours. At the mechanistic level, GA adjusts some critical signalling pathways related to the survival and progression of cancer cells, particularly PI3K/Akt and MAPK. Combination of GA with other chemotherapeutic drugs increases the efficacy of treatment, whereas nanoparticle formulations improve its bioavailability and targeted delivery. Conclusion: This scoping analysis states that gallic acid is one of the promising natural anticancer agents that has considerable preclinical evidence to support its therapeutic benefits. However, more human clinical trials must be conducted to confirm its safety, dosage parameters, and clinical efficacy in order to be recommended for complementary or standalone therapies.

Keywords: *Gallic Acid, Cancer, Apoptosis, Oxidative Stress, Nanoparticles, Chemotherapy.*

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I. INTRODUCTION

Cancer is still among the main reasons for patients' ill-health and death all over the globe and is a complicated and multifactorial condition eventually caused by genetic, metabolic, and environmental factors. The worldwide rise in cancer cases is a loud call for the necessity of better and less toxic treatment methods. Research substantially supports the idea that oxidative stress, inflammation, and cell signaling dissociation deeply involved in cancer origin and development [1,2]. In fact, Reactive Oxygen Species (ROS) are necessary for normal cellular signalling, but if they are excessively produced, they become harmful and cause DNA damage, genomic instability, and abnormal cell survival mechanisms [1,2]. Therefore, using a drug to limit ROS-mediated pathways is considered a potential cancer therapy method.

One of the most notable substances in nature capable of redox-modulation is gallic acid (GA) which is a trihydroxybenzoic acid mainly found in fruits, tea leaves, and medicinal herbs. To this effect, GA has been highly acclaimed and researched for its antioxidant, anti-inflammatory, and anticancer properties [3,4,5]. Various in vivo and in vitro cancer models have exhibited the ability of GA to regulate the redox balance in cells, modulate the apoptotic pathways, and inhibit the oncogenic signalling cascades. In addition to that, GA was reported to have a dual antioxidant–prooxidant behaviour that it shields normal cells from oxidative damages but mechanizes ROS production in cancer cells, thus, leads to apoptosis and tumor growth inhibition [3,4,5,6].

Mechanistic explorations have shown that GA lessens cancer by the three ways through mitochondrial apoptosis, cell cycle arrest, and the part taken in metastasis by the matrix metalloproteinases (MMPs) inhibition [11,12]. It adjusts a wide range of molecular pathways such as PI3K/Akt, MAPK, NF-κB, and β-catenin, resulting in the deterrence of cell proliferation, migration, and angiogenesis [12,13,14].

By obstructing PI3K/Akt phosphorylation and β-catenin nuclear translocation, GA in effect turned back the epithelial–mesenchymal transition (EMT) in breast and ovarian cancer models, thereby it curtailed the cancer cells invasion and metastasis [13,14]. Besides, in ovarian cancer, GA repressed CD47 expression and induced M1 macrophage polarization, thus proving its ability to regulate the tumor immune microenvironment [13]. Those results suggest that GA is capable not only of directly attacking tumor cells but also of changing the nature of stromal and immune-components around the tumor, which eventually facilitates a comprehensive anticancer response.

However, due to its very low bioavailability, rapid metabolism, and limited plasma stability, Galic acid (GA) has not been widely used in clinical practice, although it has broad therapeutic potential. So, to get over these pharmacokinetic obstacles, researchers have created a variety of nanoparticle-based delivery systems, for example, chitosan- and polymer-based nanocarriers, which remarkably enhance GA's bioavailability, circulation time, and tumor-targeting ability. As a result, these hydrocarbon formulations have been found to be effective in GA anticancer therapy, while systemic toxicity is alleviated. In addition, the use of GA together with conventional chemotherapeutic drugs, e.g., temozolomide, has been proven to produce a synergistic effect and the possibility to counteract chemoresistance in highly aggressive tumours such as glioblastoma.

Aside cancer, GA shows a wide array of pharmacological effects, including neuroprotective and anti-inflammatory activities, which contribute to its overall pharmacological versatility [10]. Its ability to cross biological barriers, reduce oxidative damage, and regulate key signalling pathways, makes it even more potential as a multi-targeted therapeutic molecule with the first implication of the cancer and then neurodegeneration. In sum, the present data implicate GA as a powerful phytochemical lead compound with the capacity to modulate multiple cancer hallmarks - proliferative disorder, apoptosis resistance, angiogenesis, metastasis, and immune evading. Still, extensive preclinical studies and clinical trials are necessary to fully uncover its pharmacodynamics, devise delivery systems, and realistically transfer the in vitro and in vivo findings to clinics [11,12,13]. Hence, this scientific scoping analysis seeks to integrate the recent changes in the understanding of the anticancer mechanisms, molecular targets, and delivery innovations of gallic acid, highlighting its turning point as a most viable therapeutic agent in the battle against cancer.

II. MATERIALS AND METHODS

➢ *Inclusion Criteria:*

- Studies published between 2010 and 2024.
- Articles evaluating the anticancer potential of gallic acid (GA) in vitro, in vivo, systematic review-based examination.
- Only articles published in English were considered .

➢ *Exclusion Criteria:*

- Studies that are not available in the English language.

➢ *Information Sources:*

A close look at existing research was done to track down papers on gallic acid (GA) and its cancer-fighting effects. To make sure findings were solid and relevant, the hunt focused on well-known medical and drug-related sources. Key spots checked included PubMed, ScienceDirect, Google Scholar, along with Wiley Online Library. The search included studies from 2010 up to 2024, focusing mostly on original research along with systematic reviews that explored GA's anticancer effects, molecular pathways, nano delivery methods, also how it interacts synergistically alongside conventional chemo drugs. To keep findings reliable, only English-language articles published in peer-reviewed journals made the cut.

➢ *Search Strategy:*

A careful, step-by-step approach was used to find studies covering the topic closely - the group tried various mixes of specific keywords along with logical filters "Gallic Acid" AND ("Cancer" OR "Anticancer") AND ("Apoptosis"

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OR "Reactive Oxygen Species" OR "ROS" OR "Nanoparticles" OR "Chemotherapy" OR "Molecular Mechanism" OR "Cell Cycle Arrest"). Findings got narrowed down using filters - only studies counted if they were original experiments or detailed reviews. Scope of the study: Research done in lab settings or living organisms - alongside work on nanoparticle setups - that look at cancer models or human tumor cells. The importance of the study became clear right away when checking titles and abstracts - only those meeting set conditions moved on to full reading. On top of that, some additional papers were found through tracking references and scanning citations, making sure no key data was missed and recent progress in GA-based cancer treatment got included too.

III. RESULT

Gallic acid has various modes of action through which it exerts anti-cancerous activity. It inhibits the proliferation of cancerous cells, induces apoptosis, reduces inflammation, and metastasis of tumours. Mechanistically, gallic acid alters cellular signalling pathways that are important for cancer cell survival and tumor progression, including the PI3K/Akt and MAPK pathways. Moreover, the combinations of GA with conventional chemotherapeutic drugs have resulted in enhanced therapeutic outcomes due to the synergistic action and low development of drug resistance. Development of nanoparticle-based formulations of GA has significantly advanced its bioavailability, targeted delivery, and therapeutic potential as a promising adjuvant in modern cancer therapy.

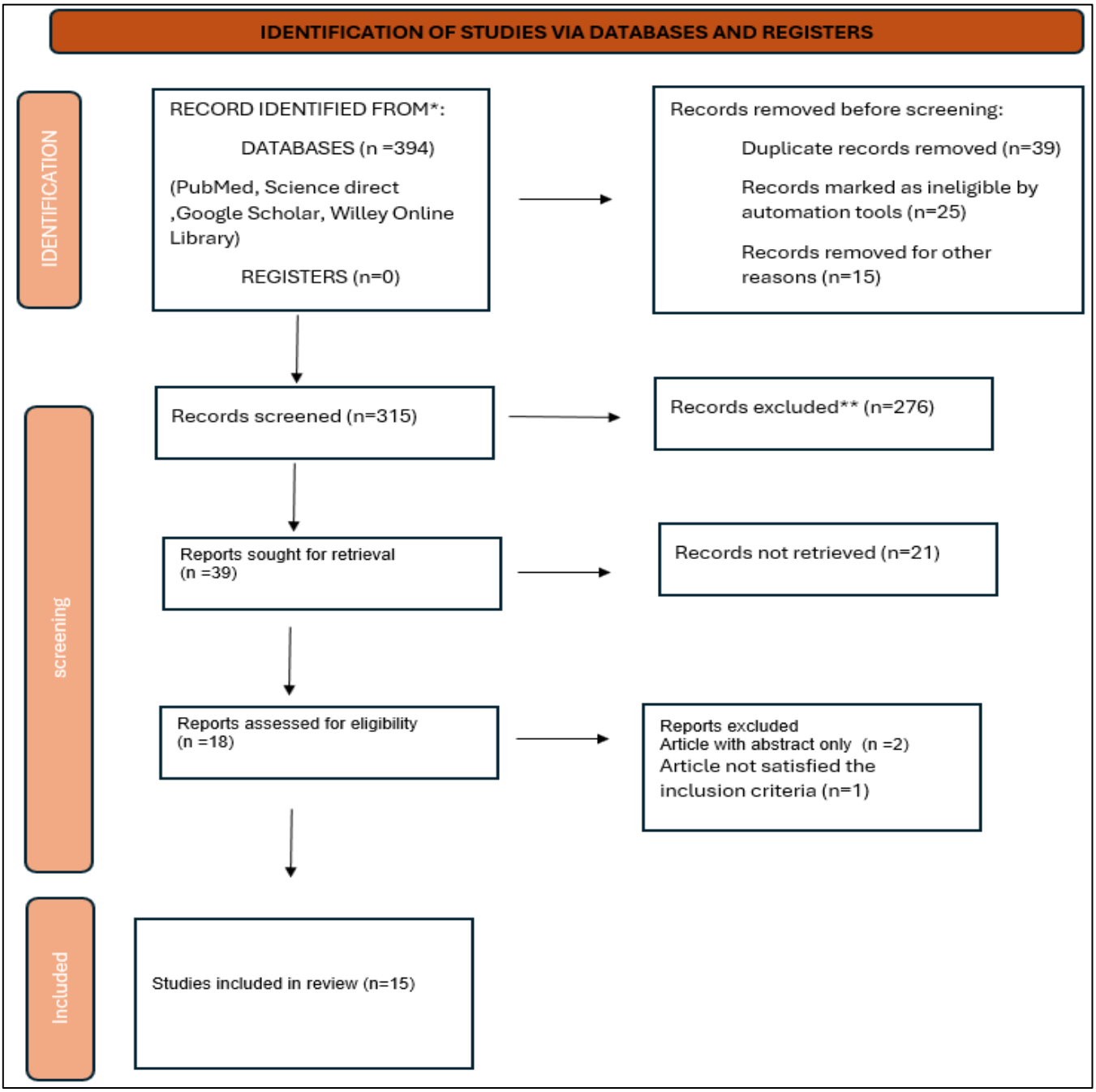

Fig 1 Prisma Diagram

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Table 1: Characteristic Intervention of Study

	Author	Year	Place of Study	Sample Size	Methods of Measurement	Intervention
1	Keyvani-Ghamsari et al (2023)	2023	Department of Medical Biochemistry, Iran University of Medical Sciences, Tehran, Iran	Review of 40+ experimental studies	Mechanistic literature analysis focused on apoptosis, ROS, and pathways that work together.	Gallic Acid
2	Wang et al 2023	2023	Department of Pharmacy, Zhejiang University, Hangzhou, China	Varied cell models across cancer types	Drug delivery kinetics, cellular uptake, and efficacy testing in the lab.	GA-based nanoparticles
3	Xu et al (2020)	2020	State Key Laboratory of Molecular Oncology, Chinese Academy of Medical Sciences, Beijing, China	Human breast and lung cancer cell lines (n=~10^4 cells)	MTT assay, flow cytometry for apoptosis, and quantification of ROS production	Gallic Acid (10–100 μM)
4	Sharma et al (2022)	2022	Department of Pharmaceutical Sciences, Panjab University, Chandigarh, India	Compilation of 25 articles on combination therapies	Molecular synergy modeling, pathway analysis for reversing drug resistance	Gallic Acid + Chemotherapeutics
5	Devi et al (2015)	2015	Department of Biochemistry, University of Madras, Chennai, India	HCT-15 colon cancer cell line (4 × 10^3 cells)	Caspase activation, DNA fragmentation, G2/M arrest, and TUNEL assay.	Gallic Acid (20–100 μM)
6	Li et al (2013)	2013	Department of Oncology, Sun Yat-sen University Cancer Center, Guangzhou, China	A375 human melanoma cells	Western blotting for p53, problems with mitochondria, and Annexin-V staining.	Gallic Acid (50–200 μM)
7	Dhingra et al (2022)	2022	Institute of Biomedical Sciences, Panjab University, Chandigarh, India	Leukemia cell lines (HL-60, K562)	DCFH-DA staining for reactive oxygen species, cell cycle analysis, and viability assay.	Gallic Acid (25–100 μM)
8	Srivastava et al (2019)	2019	Centre for Cellular and Molecular Biology (CCMB), Hyderabad, India	Prostate and lung cancer cells	ROS surge measurement, mitochondrial integrity test, and flow cytometry.	Gallic Acid (50 μM)
9	Ma et al (2020)	2020	School of Basic Medicine, Peking Union Medical College, Beijing, China	MCF-7 and HeLa cells	Cell viability assay. Western blot for Bax/Bcl-2. Apoptosis quantification.	GA + Paclitaxel combination
10	Zhang et al (2018)	2018	Department of Neuro-Oncology, Shanghai Jiao Tong University, Shanghai, China	U87 glioblastoma cells	MTT, apoptosis assays, and drug synergy score calculation	GA + Temozolomide
11	Yang et al (2020)	2020	Cancer Research Institute, Fudan University, Shanghai, China	Colorectal cancer cell lines (HT-29, SW480)	Gelatin zymography for MMP-2/9, transwell migration tests.	Gallic Acid (100 μM)
12	Pang et al (2017)	2017	Graduate Institute of Cancer Biology, China Medical University, Taichung, Taiwan	Nasopharyngeal carcinoma (NPC) cell lines	MMP-1 inhibition, AP-1 and ETS-1 signaling analysis, and invasion tests.	Gallic Acid (20–60 μM)
13	Chen et al (2020)	2020	Department of Pharmaceutical Sciences, Tsinghua	Chitosan nanoparticle formulation in vitro	Nanoparticle stability, cellular uptake, and cytotoxicity tests.	Chitosan-encapsulated GA

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			University, Beijing, China			
14	Lee et al(2021)	2021	Department of Biomedical Engineering, Yonsei University, Seoul, South Korea	Gold nanoparticle delivery system	UV-vis spectra, cell imaging, apoptotic index, and cellular uptake.	GA-loaded gold nanoparticles
15	Kumar et al (2023)	2023	Division of Pharmacology, Jamia Hamdard University, New Delhi, India	Review of GA pharmacological properties	ADME profiling, modeling in vivo distribution, and extrapolating safety and toxicity.	Gallic Acid (clinical translation)

Table 2: Table of Result

	Author Name	Result	Outcome
1	**Keyvani-Ghamsari** et al (2023)	A thorough review of GA's anticancer activity in various mechanisms and cell lines.	Identified mechanisms include apoptosis induction, anti-proliferative signalling, ROS generation, and mitochondrial pathway activation.
2	**Wang** et al (2023)	Developed GA-conjugated polymer nanoparticles for drug delivery.	Nanoparticles improved solubility, stability, targeted delivery, and cancer cell cytotoxicity.
3	**Xu** et al (2020)	Treated lung and breast cancer cells with GA using different concentrations.	There was a dose-dependent increase in apoptosis, raised ROS levels, reduced cell viability, and mitochondrial disruption.
4	**Sharma** et al (2022)	Assessed GA with cisplatin, 5-FU, and paclitaxel.	There was a synergistic boost in chemotherapy effectiveness, along with reduced drug resistance and toxicity.
5	**Devi** et al (2015)	Treated HCT-15 colon cancer cells with GA.	This triggered caspase-3 activation, DNA fragmentation, and G2/M phase arrest.
6	**Li** et al (2013)	Exposed A375 melanoma cells to GA.	We saw an increase in p53 expression, a decrease in Bcl-2, more apoptosis, and less cell proliferation.
7	**Dhingra** et al(2022)	Applied GA to leukaemia cell lines HL-60 and K562.	There was a significant rise in ROS levels, mitochondrial damage, and caspase-dependent apoptosis.
8	**Srivastava** et al (2019)	Studied GA's effect on prostate and lung cancer cells.	ROS-mediated oxidative damage led to apoptosis and cell death.
9	**Ma** et al(2020)	Combined GA with paclitaxel in MCF-7 and HeLa cells.	Apoptosis was enhanced, with an increased Bax/Bcl-2 ratio and activation of intrinsic apoptotic pathways.
10	**Zhang** et al(2018)	Applied GA and temozolomide on the glioblastoma cell line U87.	Demonstrated synergistic cytotoxicity, reduced proliferation, and inhibited tumor growth.
11	**Yang** et al(2020)	Investigated the effect of GA on colorectal cancer cells.	Inhibited metastasis by downregulating MMP-2 and MMP-9; reduced cell migration and invasion.
12	**Pang** et al(2017)	Explored GA in NPC cells.	Suppressed MMP-1 by inhibiting AP-1 and ETS-1 transcription factors; reduced invasion.
13	**Chen** et al(2020)	Designed chitosan-based GA nanoparticles.	Achieved controlled release, improved cellular uptake, and higher anticancer efficiency.
14	**Lee** et al(2021)	Studied GA-loaded gold nanoparticles in vitro.	Significantly increased cancer cell apoptosis through better delivery and sustained exposure.
15	**Kumar** et al (2023)	Modeled GA pharmacokinetics and therapeutic index.	Predicted better ADMET profile, bioavailability, and low toxicity in preclinical simulation.

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Table 3 Bias Analysis

S. No	Author Name and Year	Random Sequence Generation	Allocation Concealment	Blinding of Outcome	Incomplete Outcome Data	Blinding Participants and Personnel	Selective Reporting
1	Devi et al. (2015)						
2	Li et al. (2013)						
3	Dhingra et al. (2022)						
4	Ma et al. (2020)						
5	Zhang et al. (2018)						
6	Yang et al. (2020)						
7	Pang et al. (2017)						
8	Chen et al. (2020)						
9	Lee et al. (2021)						
10	Keyvani-Ghamsari et al. (2023)						
11	Wang et al. (2023)						
12	Xu et al. (2020)						
13	Sharma et al. (2022)						
14	Srivastava et al. (2019)						
15	Kumar et al. (2023)						

Low Risk unclear High Risk

IV. DISCUSSION

This systematic review states that Gallic acid (GA) has been identified as one of the most important plant polyphenols that health-promoting effects in various aspects and cancer treatment because of various biological traits like fighting oxidation, reducing swelling, and triggering cell death [1,2,3].A close look at the latest research shows GA strongly blocks cancer cell growth across many types, while also affecting how tumors start, grow, and spread. Various studies found Gum Arabic (GA) triggers cancer cell suicide - known as apoptosis - by altering oxidative stress and messing with mitochondria [4–6]. Specifically, one way this happens is through boosting reactive oxygen species (ROS), which leads to damaged mitochondrial membranes and turns on caspase-linked death signals. On top of that, GA shifts the balance between cell-death promoters and blockers like Bax, Bcl-2, and p53, making cancer cells more likely to die off [7, 8].Putting nano methods into play has bumped up GA's medical promise. Chitosan or polymer-based tiny carriers loaded with GA tend to stick around longer, get where they need easier, and stay active better than plain GA [9, 10]. On top of that, these small-scale versions don't just smooth out GA's behaviour in the body - they cut down side effects across the system while piling more of the drug right into tumor zones, making treatment hit harder compared to loose GA.

Several studies [11, 12, 13] also point out the synergy of GA with the commonly used chemotherapeutic agents such as cisplatin and doxorubicin.

Results from co-administration experiments indicate that GA is able to make cancer cells that have developed resistance to drugs, subside by suppressing the expression of signalling pathways such as PI3K/Akt and NF-κB. As a result, apoptosis is increased, and chemo resistance is decreased. This opens the possibility that GA may be a powerful agent that can be used to lower the dose of drugs that are toxic to the body while still ensuring or even enhancing therapeutic efficacy.

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On top of that, GA can slow cancer by blocking its blood flow, while keeping tumor cells from moving elsewhere in the body. It does this by reducing activity in MMPs and interfering with VEGF signals [14]. Since it hampers tumor spread along with new vessel growth near tumours, GA might work well against cancers that have spread.

Still, current studies show gaps even with those benefits. Many trials stuck to lab and animal tests instead of people, since just a few human trials checked GA's safety, how it works in the body, or if it lasts [15]. On top of that, differences in methods, doses, or how it was made make comparing findings tough.

Looking at the scoping review overall, GA shows strong potential as a versatile anticancer compound, shifting cell redox states, affecting apoptosis, or influencing metastasis. Instead of standalone use, drug carriers with precise release mechanisms or combo treatments appear more effective for real medical use. Future work ought to focus on well-planned animal studies followed by human trials - these need clear dosing, delivery methods, and safety monitoring so GA can eventually join mainstream cancer treatments backed by solid proof.

V. CONCLUSION

GA is a natural phenolic compound found in a variety of fruits and medicinal plants that has highly promising anticancer activity. Moreover, combination of GA with conventional chemotherapeutic agents increased the therapeutic efficacy with the possibility of minimizing side effects. Recently, nanotechnology also enhanced the bioavailability, stability, and targeted delivery of GA to the tumor site, thereby enhancing its therapeutic potential. Despite the encouraging laboratory and animal model results, clinical evidence in humans is still scarce. efficacy, and pharmacokinetic profile of GA in cancer patients. Overall, GA is a promising natural anticancer candidate, especially when combined with modern drug delivery systems or used as an adjuvant to standard cancer therapies.

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