International Journal for Multidisciplinary Research (IJFMR)
E-ISSN: 2582-2160 ● Website: www.ijfmr.com ● Email: editor@ijfmr.com

A Comprehensive Review of the Therapeutic Potential of Boswellic Acid and Curcumin in Lung Cancer: Mechanistic Synergies, Pharmacological Advancements, and Translational Challenges

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Abstract

Curcumin, a polyphenolic compound derived from *Curcuma longa* (turmeric), and Boswellic Acid, obtained from the resin of *Boswellia serrata* (Indian frankincense), are bioactive phytochemicals with a well-documented history in traditional Indian medicine. Both compounds possess established anti-inflammatory and immunomodulatory properties that are directly relevant to cancer biology, given the recognized role of chronic inflammation in tumor development. This review critically examines the preclinical and available clinical evidence on the individual and combined anti-lung-cancer activity of curcumin and Boswellic Acid, addresses existing bioavailability challenges, evaluates innovative formulation strategies, and analyzes the translational gap that continues to limit clinical application. A systematic narrative review was carried out using PubMed, Scopus, Web of Science, and Google Scholar (up to August 2025). Curcumin was found to inhibit proliferation in multiple non-small cell lung cancer (NSCLC) cell lines by inducing apoptosis and cell cycle arrest through suppression of the PI3K/Akt/mTOR, STAT3, and NF-KB pathways. Acetyl-11-keto-beta-Boswellic Acid (AKBA) shows selective cytotoxicity against A549 cancer cells relative to normal lung epithelium and inhibits oncogenic signaling through the PI3K/Akt axis, G0/G1 arrest, and autophagy suppression. Evidence from colorectal cancer and inflammatory models confirms that co-administration produces synergistic cytotoxicity, particularly in p53-mutated tumor backgrounds. Pharmacokinetic data show no adverse drug-drug interactions between the two compounds. Advanced delivery platforms including PLGA nanoparticles, liposomes, and inhalable nano-in-microparticles considerably improve lung-tissue bioavailability. Curcumin and Boswellic Acid together represent a pharmacologically sound, multi-targeted combination with strong preclinical support for lung cancer management. Their clinical translation remains constrained primarily by poor bioavailability and the complete absence of human lung cancer trial data. Development

of inhalable co-delivery systems and initiation of well-designed randomized controlled trials are the most immediate priorities for advancing this therapeutic approach.

Keywords: Boswellia Serrata; Curcuma Longa; Non-Small Cell Lung Cancer; Phytochemistry; Nanoparticle Delivery; PI3K/Akt/mTOR; NF-KB

1. Introduction

1.1 The Unmet Need in Lung Cancer Therapy

Lung cancer remains one of the leading causes of cancer-related death worldwide, a status it has held for several decades [8]. Non-small cell lung cancer (NSCLC) accounts for approximately 80 to 85% of all lung cancer diagnoses, and despite notable advances in surgical techniques, radiation protocols, and systemic chemotherapy, the five-year survival rate for patients diagnosed with advanced-stage disease continues to be disappointingly low [8]. The main drawbacks of conventional treatment are well recognized: considerable procedural morbidity, significant systemic toxicity that affects quality of life, and the gradual emergence of chemoresistance that eventually undermines treatment efficacy.

These are not simply clinical inconveniences. At the root of the problem lies the biological adaptability of tumor cells, which rely on multiple overlapping signaling cascades to resist the targeted pressure of any single therapeutic agent. This understanding has drawn growing scientific interest toward phytochemicals as potential adjunctive or standalone agents: compounds that offer multi-target pharmacological activity, generally favorable toxicity profiles, and in many cases a compelling history of safe use in human populations.

1.2 The Rise of Natural Compounds in Oncology

The pharmacological basis for using phytochemicals in cancer treatment is fairly clear: since cancer is a disease of dysregulated networks rather than a single molecular defect, agents capable of simultaneously modulating multiple oncogenic pathways hold intrinsic therapeutic value. Natural products, owing to their structural complexity and long evolutionary co-existence with biological systems, frequently exhibit this kind of broad-spectrum activity [24].

Among the most thoroughly studied phytochemicals in oncology are curcumin and Boswellic Acid. Curcumin is a polyphenolic compound isolated from the rhizome of *Curcuma longa* L. (turmeric), a plant integral to Ayurvedic and South Asian culinary traditions for at least three thousand years. Boswellic Acids are a family of pentacyclic triterpenoids derived from the oleo-gum-resin of *Boswellia serrata* Roxb. ex Colebr. (Indian frankincense), a tree widely used in traditional Indian and North African medicine for the management of inflammatory disorders, including those affecting the joints and the respiratory tract [4, 11]. The anti-inflammatory and anti-tumorigenic properties of both compounds are now well established in laboratory settings. Whether these properties can be translated into meaningful clinical benefit, particularly in lung cancer, remains an open and important question.

1.3 Scope of the Review

This review brings together and critically examines the current evidence on the individual and combined effects of curcumin and Boswellic Acid in lung cancer. It addresses the pharmacological barriers that have historically limited clinical use, with particular focus on bioavailability, and evaluates the formulation strategies designed to overcome these obstacles. The review then examines the mechanistic evidence for anti-cancer activity drawn from in vitro and in vivo studies, considers available data on synergistic interactions, and concludes with a structured analysis of the translational gap that separates laboratory

findings from clinical application. Where contradictory evidence exists, it is presented and discussed openly rather than set aside.

2. Pharmacological Foundations: Extraction, Isolation, and Formulation

2.1 Lab-Scale Extraction and Purification Protocols

2.1.1 Curcumin from Curcuma longa

Curcumin isolation begins with dried rhizomes of *Curcuma longa*, which are ground into a fine powder to increase surface area and improve solvent contact. As a liposoluble compound, curcumin is most efficiently extracted using polar organic solvents; acetone is generally preferred at laboratory scale owing to its low cost, low boiling point, and comparatively high extraction yield [20]. A solid-to-solvent ratio of around 1:8 at 30 °C for three hours has been reported to yield consistent results. After the extraction period, the solvent fraction is separated by distillation, and the resulting oleoresin is further purified using hexane to remove non-polar contaminants, producing a dried curcumin powder of suitable purity for experimental use.

When higher purity or better efficiency is needed, ultrasound-assisted extraction (UAE) offers a clear advantage over conventional solvent extraction. The acoustic cavitation generated by high-frequency sonication disrupts plant cell walls more thoroughly and accelerates mass transfer, producing higher curcumin yields in shorter processing times [19]. UAE is increasingly adopted as the laboratory standard where reproducibility and yield are critical.

2.1.2 Boswellic Acids from Boswellia serrata

The isolation of Boswellic Acids from *Boswellia serrata* gum resin involves a multi-step process that exploits the acidic character of these triterpenoids. In the classical approach, the crushed resin is extracted with a hydro-alcoholic solution and the crude extract is rendered alkaline with potassium hydroxide, which converts the Boswellic Acids into soluble potassium salts. Insoluble gum material is filtered out, and the alkaline filtrate is acidified with a mineral acid, causing the Boswellic Acids to precipitate as a white solid [25].

For laboratory-scale purification of individual Boswellic Acid isomers, particularly the pharmacologically most active acetyl-11-keto-beta-Boswellic Acid (AKBA), a combination of column chromatography on silica gel and preparative high-performance liquid chromatography (HPLC) is standard. Initial extraction is often intensified using UAE or accelerated solvent extraction (ASE), both of which outperform classical Soxhlet extraction in terms of yield and time efficiency [23]. Purity is confirmed by thin-layer chromatography (TLC) and analytical UHPLC/HPLC, which can reliably distinguish AKBA from the other Boswellic Acid isomers.

2.2 Overcoming Bioavailability Challenges

2.2.1 The Bioavailability Problem

The pharmacological promise of both curcumin and Boswellic Acid is substantially undermined by their poor oral bioavailability, a problem that has long hindered their clinical application. Curcumin is poorly water-soluble, undergoes rapid degradation at alkaline intestinal pH, and is extensively metabolized in the gut wall and liver via glucuronidation and sulfation to yield less pharmacologically active conjugates that are quickly eliminated from systemic circulation [5]. Boswellic Acids face similar absorption challenges, and reaching pharmacologically relevant tissue concentrations typically requires substantial doses, in the range of 500 to 1000 mg of curcumin and 500 mg of Boswellia extract twice daily, raising real compliance

concerns [14].

2.2.2 Modern Formulation Strategies

Several strategies have been developed to address these limitations, with varying degrees of clinical validation. The simplest and best-documented is co-administration of curcumin with piperine, an alkaloid from black pepper that inhibits cytochrome P450 enzymes and UDP-glucuronosyltransferase, thereby slowing first-pass metabolism. In a landmark pharmacokinetic study, piperine co-administration increased curcumin serum concentration by approximately 2000% in healthy human volunteers compared with curcumin given alone, with no measurable adverse effects [22].

Encapsulation within lipid-based nanocarriers represents a more sophisticated and increasingly preferred approach. Liposomal formulations encase the hydrophobic active compounds within phospholipid bilayers that are compatible with biological membranes, thereby improving both solubility and cellular uptake. A notably effective system is FenuMat® technology, which employs a fenugreek-derived galactomannan hydrogel matrix to create a self-emulsifying delivery vehicle for curcumin. This formulation showed a 45.5-fold increase in the bioavailability of free, unconjugated curcuminoids relative to unformulated curcumin in a randomized, double-blind, crossover clinical study, along with a sustained plasma concentration profile that suggests meaningful protection against rapid hepatic clearance [7].

Nanotechnology, particularly the use of poly(lactic-co-glycolic acid) (PLGA) nanoparticles, offers an especially relevant pathway for lung cancer because these carriers can be engineered for inhalable pulmonary delivery. The pulmonary route provides direct access to lung tissue, bypasses hepatic first-pass metabolism entirely, and can achieve high local drug concentrations with minimal systemic exposure. Nano-in-Microparticle (NiMP) formulations have been developed specifically to provide the aerodynamic properties required for efficient deposition in the bronchopulmonary tree [10]. Co-encapsulation of Boswellia extract and curcumin within a single delivery vehicle has been shown to yield superior anti-inflammatory effects compared with either compound alone, pointing toward an integrated delivery and pharmacological strategy rather than a simple additive one [14].

3. Individual Anticancer Activities in Lung Cancer: Preclinical Evidence

3.1 Curcumin

3.1.1 Proliferation Inhibition and Apoptosis

A substantial body of preclinical literature documents the anti-proliferative effects of curcumin across multiple human lung cancer cell lines, including A549 (NSCLC, adenocarcinoma), H1299, and H460 [1]. These effects are dose- and time-dependent and have been reproduced across numerous independent laboratories, adding considerable credibility to the findings. Mechanistically, curcumin induces cell cycle arrest at both the G0/G1 and G2/M checkpoints and promotes apoptosis through the mitochondria-dependent (intrinsic) pathway. The hallmarks of this apoptotic response include upregulation of the pro-apoptotic protein Bax, downregulation of the anti-apoptotic protein Bcl-2, and activation of caspase-3, the principal executioner protease [26].

3.1.2 Modulation of Key Signaling Pathways

The PI3K/Akt/mTOR axis is constitutively activated in a large proportion of NSCLC tumors and drives cell survival, proliferation, and resistance to chemotherapy. Curcumin inhibits this pathway in A549 cells, leading to the coordinated induction of both apoptosis and autophagy; its effect is strengthened by co-treatment with specific PI3K inhibitors, confirming that pathway inhibition plays a central mechanistic role in its anti-cancer effects [27]. Similarly, curcumin suppresses constitutive STAT3 and NF-KB

activation in both SCLC and NSCLC models, reducing the transcription of downstream targets including cyclin D1, cyclin B1, and survivin, all of which promote cell cycle progression and apoptosis resistance [6, 9]. At the epigenetic level, curcumin modulates microRNA expression, notably upregulating the tumor-suppressive miR-206, which in turn further suppresses the PI3K/Akt/mTOR pathway and inhibits cell migration and invasion [12].

3.1.3 Contradictory In Vivo Evidence: A Critical Note

The largely positive preclinical picture for curcumin must be tempered by at least one significant contradictory observation. In a transgenic mouse model carrying the oncogenic Ki-rasG12C mutation, dietary curcumin unexpectedly accelerated lung tumor development and increased tumor multiplicity, an effect of similar magnitude to that of the known lung tumor promoter butylated hydroxytoluene (BHT) [18]. The proposed mechanism involves a context-dependent pro-oxidant effect of curcumin in damaged lung epithelium, a role that runs directly counter to its well-established antioxidant activity in normal tissue. This finding does not disqualify curcumin as a therapeutic candidate, but it clearly illustrates that its effects are not universally anti-tumoral and are strongly shaped by the genetic background of the tumor and the state of the surrounding tissue. The implications for clinical trial design are significant: stratifying patients by oncogenic mutation status may be important for both the safety and the efficacy of curcumin-containing regimens.

Table 1: Summary of Selected Preclinical Anti-Lung Cancer Effects of Curcumin and Boswellic Acid

Compound	Model	Primary Mechanism(s)	Outcome	Reference
Curcumin	A549 cells (in vitro)	PI3K/Akt/mTOR inhibition; G2/M arrest; caspase-3 activation	Apoptosis induction; autophagy	Zhou et al., 2018 [27]
Curcumin	H1299/SCLC cells (in vitro)	STAT3 and NF-KB suppression; cyclin D1 downregulation	Cell cycle arrest; inhibited invasion	Chakravarti et al., 2006 [9]
AKBA	A549 vs. BEAS-2B (in vitro)	PI3K/Akt inhibition; G0/G1 arrest; Beclin-1/LC3 suppression	Selective cancer cell death; autophagy suppression	Liu et al., 2020 [13]
AKBA	Orthotopic colorectal xenograft (in vivo; lung metastasis endpoint)	NF-kB suppression; anti-angiogenic; anti-invasive	Significantly reduced lung metastasis	Ahn et al., 2012 [3]

AKBA = acetyl-11-keto-beta-Boswellic Acid; SCLC = small cell lung cancer; NSCLC = non-small cell lung cancer.

3.2 Boswellic Acid

3.2.1 Cytotoxicity and Selective Activity Against Lung Cancer Cells

Among the Boswellic Acid isomers, AKBA has attracted the greatest pharmacological interest owing to

its potency and reported selectivity. Studies on NSCLC cell lines (A549, H460, H1299) have confirmed dose- and time-dependent inhibition of cell viability and proliferation. A notable finding from a well-controlled study is that AKBA showed considerably greater cytotoxicity in A549 cancer cells than in BEAS-2B cells, a normal bronchial epithelial line included as a healthy tissue control [13]. This difference in toxicity, representing a therapeutic index that favors malignant over normal lung cells, is a clinically meaningful property not commonly seen with many standard chemotherapeutic agents.

3.2.2 Mechanisms of Anti-Cancer Action

The mechanistic profile of AKBA in lung cancer cells involves several converging pathways. Cell cycle analysis shows primary arrest at the G0/G1 phase, associated with downregulation of cyclin A2, cyclin E1, and phosphorylation of cdc2, effectively blocking entry into S phase and halting tumor cell progression. The apoptotic component is characterized by an increase in the Bax/Bcl-xl ratio, favoring mitochondrial outer membrane permeabilization and caspase activation [13]. Upstream of these events, AKBA suppresses PI3K/Akt signaling, which appears to be a primary mechanism coordinating both cell cycle arrest and apoptosis induction.

It is also notable that AKBA suppresses autophagy in lung cancer cells, as evidenced by reduced expression of the autophagy markers Beclin-1 and LC3A/B. This is pharmacologically meaningful because autophagy often serves as a pro-survival mechanism in tumor cells under stress, and its suppression by AKBA may prevent cancer cells from using this pathway to escape cytotoxic injury. Boswellic Acids also suppress NF-KB transcriptional activity and modulate microRNA expression in ways consistent with an anti-tumor profile, including upregulation of the tumor-suppressive let-7 and miR-200 microRNA families in colorectal cancer models [21].

Important evidence for AKBA's anti-lung cancer potential also comes from in vivo studies on colorectal and pancreatic cancer models that specifically examined lung metastasis as an endpoint. In orthotopic xenograft models, AKBA substantially reduced the number and burden of pulmonary metastatic deposits, demonstrating anti-metastatic activity in lung tissue even without a primary lung tumor model [3]. These findings constitute direct evidence that AKBA can suppress tumor cell colonization and survival in lung tissue.

4. Synergistic Effects of Curcumin and Boswellic Acid in Combination

4.1 Rationale and Available Evidence

While a dedicated literature on curcumin-Boswellic Acid combination therapy specifically for lung cancer does not yet exist, the case for synergy is supported by evidence from two sources: studies on other cancer types and data from inflammatory disease models where both compounds have been used together. The scientific and pharmacokinetic rationale for combining these agents is sound, and the available experimental evidence supports it.

In a well-designed clinical study on patients with spondylitis, a bioavailability-enhanced formulation combining a full-spectrum Boswellia extract with curcumin (C-BSE) produced superior reductions in pain and inflammatory markers compared with Boswellia extract alone [14]. This advantage was attributed to synergistic suppression of the NLRP3 inflammasome and reduced interleukin-1beta (IL-1beta) secretion, an inflammatory axis with direct relevance to lung cancer progression, given that chronic NLRP3 activation has been implicated in the promotion of the NSCLC tumor microenvironment. From the oncological domain, the most direct evidence comes from a study in colorectal cancer (CRC) models in which the curcumin-AKBA combination produced significantly enhanced cytotoxicity, more pronounced

apoptosis, and stronger cell cycle arrest than either compound alone, with these results reproduced in both in vitro cell line experiments and in vivo mouse xenograft models [17].

4.2 Molecular Basis of Synergy

The molecular basis for the synergistic interaction is that curcumin and AKBA target different but functionally connected nodes within the cancer signaling network. While both compounds independently suppress the NF-KB and PI3K/Akt pathways, they differ in their secondary targets. Curcumin has broad epigenetic activity, modulating a range of microRNAs and exerting inhibitory effects on STAT3 and the Wnt/beta-catenin pathway. AKBA, by contrast, is a specific and potent inhibitor of 5-lipoxygenase (5-LOX), an enzyme that generates pro-inflammatory leukotrienes and whose activity has been linked to tumor immune evasion and angiogenesis [4]. Simultaneously blocking these distinct pathways limits the capacity of cancer cells to activate compensatory signaling, which is essentially the same mechanism that drives acquired resistance to single-agent therapy.

An additional mechanistic detail comes from the observation that the synergistic interaction between curcumin and AKBA in CRC models was dependent on the tumor cell's p53 status. The combination produced a synergistic outcome (Combination Index < 1) in p53-mutated cell lines but only an additive effect in the p53 wild-type setting, suggesting that AKBA specifically complements curcumin-induced cytotoxicity in cells that have lost functional p53 [17]. This finding is directly relevant to lung cancer, in which p53 mutation is among the most common oncogenic events, occurring in approximately 50% of NSCLC and over 70% of SCLC cases.

4.3 Pharmacokinetic Compatibility

A combination therapy is only clinically viable if the constituent agents do not adversely affect each other's absorption, distribution, metabolism, or elimination. A pharmacokinetic study examining the co-administration of Boswellia extract, curcumin, pine bark extract, and methylsulfonylmethane in healthy human volunteers found no significant differences in the area under the plasma concentration-time curve or peak plasma concentrations for any of the individual constituents when given in combination versus alone [15]. This absence of pharmacokinetic interference is an important finding that reduces the risk of unexpected toxicity from drug interactions and supports the clinical investigability of this combination.

5. Translational Gaps and Future Research Directions

5.1 The Absence of Lung-Specific Clinical Trial Data

The most significant gap in the current evidence base is the total absence of randomized controlled trial data examining either curcumin or Boswellic Acid, alone or in combination, as a treatment for lung cancer in human patients. While some clinical studies have looked at curcumin in the context of pancreatic and breast cancer and in inflammatory pulmonary conditions such as chronic obstructive pulmonary disease (COPD), no comparable data exist specifically for lung cancer [16]. This is a fundamental translational barrier: moving from strong in vitro results to meaningful clinical benefit requires exactly this kind of evidence, which is currently lacking.

The reasons for this gap are not hard to identify. Curcumin's poor oral bioavailability has historically made it difficult to design trials with a credible pharmacokinetic rationale, and the absence of patent protection for natural compounds has reduced commercial incentives for the costly clinical trial infrastructure required in oncology. The situation is further complicated by the heterogeneity of commercial curcumin and Boswellia formulations, which vary considerably in purity, standardization, and bioactive content, making cross-trial comparisons problematic.

5.2 Recommendations for Future Research

Bridging this translational gap will require a focused and coordinated research effort. Four priority areas deserve attention.

First, more diverse and mechanistically rigorous in vivo models are needed. Current animal data are promising but limited, and the contradictory results from the Ki-rasG12C transgenic model highlight the need for studies across a range of lung cancer genetic subtypes before clinical advancement can be responsibly pursued.

Second, the field urgently requires well-powered, randomized, placebo-controlled clinical trials. These should begin with safety and pharmacokinetic objectives, establishing whether therapeutic concentrations can be achieved in lung tissue and that the compounds are tolerated at effective doses. Given the bioavailability limitations discussed above, such trials should employ enhanced-formulation delivery systems rather than unmodified botanical extracts.

Third, inhalable co-delivery platforms, particularly PLGA-based NiMPs formulated for pulmonary deposition, represent the most pharmacologically rational route for lung-targeted delivery and deserve dedicated preclinical and Phase I investigation. The ability to bypass systemic metabolism and deliver therapeutic drug concentrations directly to pulmonary tissue is a strategic advantage that no oral formulation can fully replicate.

Fourth, the p53-dependent synergy data from CRC models provides a specific and actionable hypothesis for patient stratification. Clinical trials of the curcumin-AKBA combination in lung cancer should consider enriching for patients with p53-mutated tumors, as this subpopulation may represent the group most likely to benefit from the specific mechanistic advantages of the combination.

6. Conclusions

Curcumin (from *Curcuma longa*) and Boswellic Acid (from *Boswellia serrata*) are multi-targeted phytochemicals with well-documented anti-inflammatory and anti-tumorigenic properties that are mechanistically relevant to lung cancer biology. The preclinical evidence for their individual anti-cancer activity, drawn from a substantial body of in vitro and in vivo studies, is compelling: both compounds inhibit oncogenic signaling through the PI3K/Akt/mTOR and NF-KB pathways, induce apoptosis, and reduce cancer cell viability in NSCLC models. AKBA additionally demonstrates preferential cytotoxicity toward malignant versus normal lung cells, a pharmacologically desirable characteristic. The limited but consistent evidence for synergistic interactions, including p53-dependent combination effects, and the absence of adverse pharmacokinetic interactions, provides a rational scientific basis for co-administration.

Significant obstacles remain. Poor bioavailability continues to limit clinical translation, although modern nanoformulation strategies and piperine co-administration offer increasingly validated solutions. More critically, no human clinical trial data exist for the combination in lung cancer, and this absence represents the single largest impediment to clinical adoption.

The direction for future work is fairly clear, even if the practical challenges are substantial: advanced inhalable co-delivery systems must be developed and clinically validated, and randomized controlled trials must be initiated. The coming together of traditional pharmacological knowledge, modern molecular science, and nanotechnology-based drug delivery offers a real and promising, though not yet fully realized, avenue for improving outcomes in one of the world's most deadly cancers.

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