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Review Article

BOSWELLIC ACIDS AS HERBAL THERAPEUTICS: MECHANISMS, CLINICAL POTENTIAL AND FUTURE DIRECTIONS

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ABSTRACT

Boswellic acids (BAs), the bioactive components extracted from *Boswellia serrata* resins, have garnered considerable interest for their broad range of pharmaceutical applications. However, there have been gaps in clinical studies and its therapeutic effect. This review examines BA's pharmacological processes and therapeutic effects with particular emphasis on mechanistic targets. A comprehensive analysis of preclinical and clinical studies published between 2000 and 2024 was conducted, focusing on key signaling pathways involved in inflammation, oxidative stress, and disease progression. The findings indicate that BAs exert their effects primarily through modulation of inflammatory and oxidative stress-related pathways, notably 5-lipoxygenase, nuclear factor kappa B, and mitogen-activated protein kinases. Among various derivatives, Acetyl-11-keto-β-BA, 11-keto-beta-BA demonstrates enhanced biological activity. Preclinical and clinical results highlight the therapeutic effects of BA in managing diseases, including cancers, metabolic disorders, skin diseases like psoriasis, and neurological conditions. The multitargeted pharmacological profile of BA, along with its safety data, suggests its potential use in the management of disorders. Nonetheless, specific gaps such as dosing standardization, long-term safety, large scale trials, remain essential to confirm efficacy and safety in broader populations.

Keywords: Boswellic acid, *Boswellia serrata*, Anti-inflammatory, Anticancer, Antidiabetic, Acetyl-11-keto-β-boswellic acid.

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INTRODUCTION

The Indian frankincense tree is the prime source of *Boswellia serrata*, native to India, the Middle East, and North Africa. Its resin is extracted from bark incisions to produce oleo-gum-resin, which is used in traditional Ayurvedic medicine for its anti-inflammatory qualities, particularly for inflammatory bowel conditions, asthma, and arthritis. *B. serrata* resin is a rich source of pharmacologically active pentacyclic triterpenoids, collectively known as boswellic acids (BAs), among which 3-O-acetyl-11-keto-β-BA (AKBA) is considered the most potent bioactive constituent as shown in Table 1 [1]. BAs had always been seen as the primary bioactive components of frankincense. *B. serrata* oleo-resin is mostly used in medicine, religion, cosmetics, and scent for commercial purposes. Like other frankincense species, *B. serrata* is a major source of BA, which is employed in the pharmaceutical industry [1,2].

Many parts of the Western Himalayas and the Indian states of Rajasthan, Madhya Pradesh, Bihar, Gujarat, Maharashtra, and Orissa are home to BA cultivation [3]. The genus *Boswellia* consists of nearly 25 species, among which *B. serrata*, *Boswellia carteri*, and *Boswellia neglecta* are considered pharmacologically important [4]. Indian Frankincense, Indian Olibanum, Incense, or Salai Guggal are other common names for oleo-gum resin. The dried gum takes the shape of white-yellow lumps or rips at first [5].

In ayurveda, it is seen that the gum provides various medicinal benefits like anti-inflammatory action and is used to treat various inflammatory ailments affecting the gastrointestinal tract, eyes, skin, and gums [6].

The structural requirements of BA were studies and it was found that all the six acids had inhibition effect against 5-lipoxygenase (5-LOX) [7], the two most powerful being AKBA and 11-keto-beta-BA (KBA). AKBA operates as a non-redox-type, non-competitive inhibitor and binds to 5-LOX in a unique way that is both calcium-dependent and reversible [8].

Many articles suggest the various pharmacological properties of BA in managing inflammatory disorders such as ulcerative colitis, rheumatoid arthritis, Crohn's disease, and bronchial asthma, as well as antidepressive and antianxiety properties and anti-diabetic action. Along with this, BAs have also shown medical effects in the treatment of brain tumors and pancreatic cancer as shown in Figure 1 [9]. BAs are experimentally found to be the most bioactive components of frankincense, and with experimental evidence from *in vitro* research and animal models, it is proven promise of various extracts or constituents from frankincense for the treatment of various ailments. It has been reported that in the U.S., approximately 18% of individuals who take prescription medications also use herbal supplements at the same time. The mechanistic and clinical data on BAs published between 2000 and 2024 are critically synthesized in this review, with an emphasis on AKBA and KBA as important bioactive components. It highlights the translational gap between robust preclinical efficacy and scant clinical confirmation while integrating biological targets, treatment results, and safety data. The study outlines future research goals for successful clinical development of BA-based medicines by highlighting unsolved issues such as bioavailability, dosage optimization, and a lack of large-scale clinical studies [10-12].

MECHANISM OF ACTION

BAs, exerts their therapeutic effects through modulation of multiple molecular targets involved in inflammation, metabolic dysfunction, oxidative stress and tumor progression. BAs exhibit a multitarget pharmacological profile, influencing key signaling cascades such as 5-LOX, nuclear factor-κB (NF-κB), mitogen-activated protein kinases (MAPKs), apoptotic regulators, and metabolic enzymes. These pathways together contribute to significant anti-cancer, anti-inflammatory, and immunomodulatory actions, making them potential medicines in the treatment of different chronic disorders. As summarized in Fig. 2, BAs act through multiple interconnected pathways that collectively regulate inflammatory mediators, cell survival, and metabolic homeostasis [15,16].

Inhibition of 5-LOX

Inhibition of the 5-LOX enzyme is the primary way that BAs reduce inflammation as shown in Table 2. AKBA binds to an allosteric site of the 5-LOX, resulting in inhibition of the binding with arachidonic acid and hence inhibiting the biosynthesis of leukotrienes, particularly

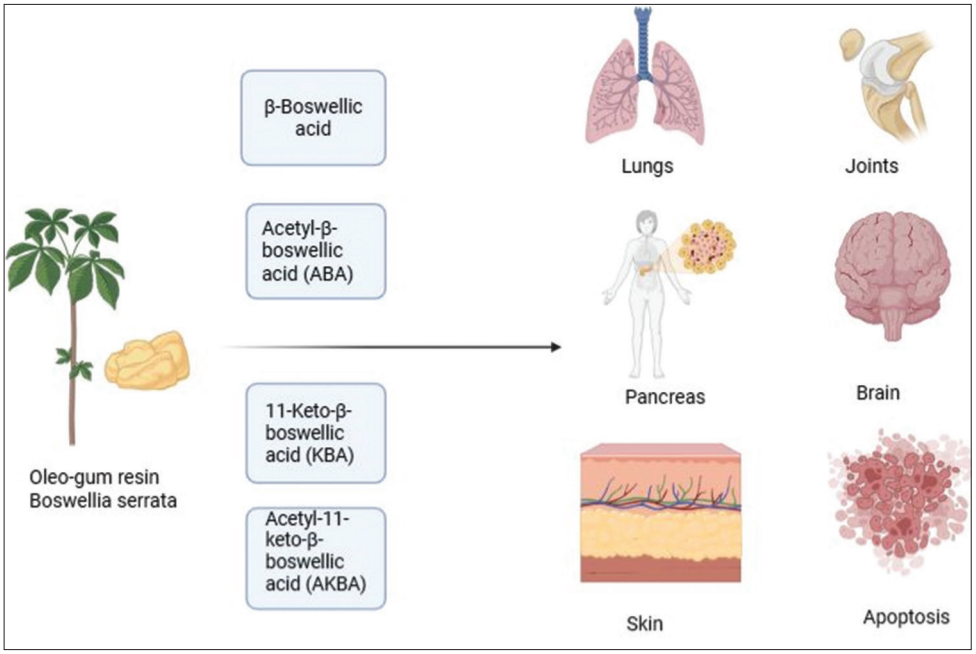

Fig. 1: *Boswellia* gum yields the bioactive compounds β-boswellic acid, Acetyl-11-Keto-β-boswellic acid, 11-Keto-β-boswellic acid, and Acetyl-11-Keto-β-boswellic acid, which exhibit therapeutic potential across multiple organs, including lungs, joints, pancreas, brain, and skin

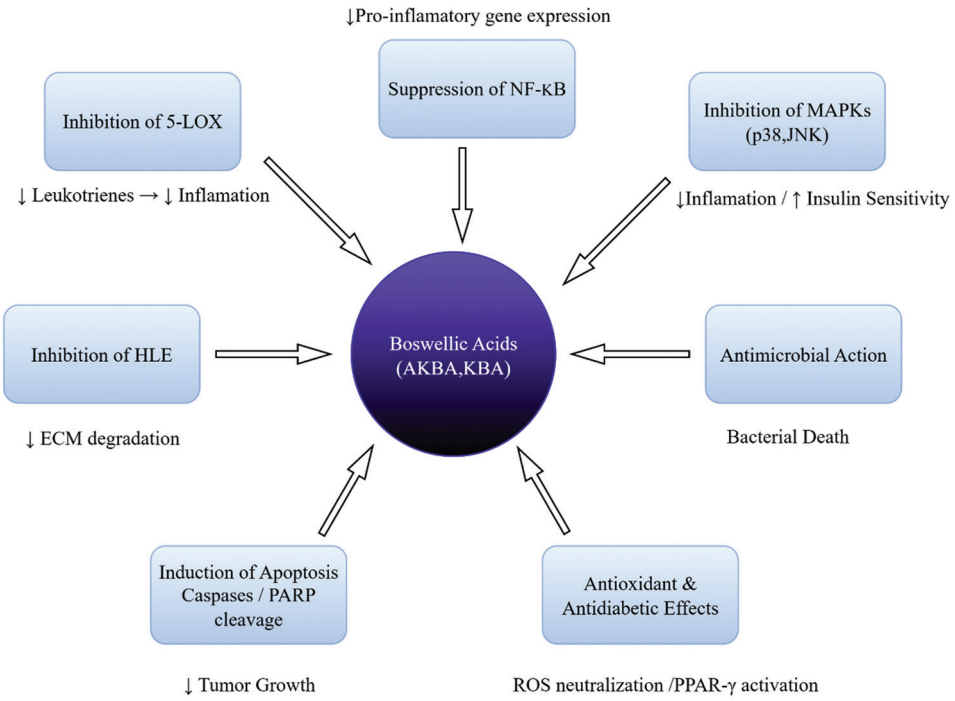

Fig. 2: Boswellic acid: Mechanism of action via different pathways

leukotriene B4. Leukotrienes are potent proinflammatory mediators that play a central role in bronchoconstriction, vascular permeability, and leukocyte recruitment, contributing to the pathogenesis of asthma, inflammatory bowel disease (IBD), and rheumatoid arthritis. Therefore, suppression of leukotriene production by BAs directly attenuates inflammatory responses in these conditions [17,18].

Suppression of NF-kB pathway by BA

BA inhibits the NK-kB signaling, responsible for expression of several genes associated with immunity, inflammation, cell division, and therefore exerts anti-inflammatory effect as shown in Table 2 [15]. Evidence indicates that BAs suppress IκB kinase activity, thereby preventing degradation of IκB and subsequent nuclear translocation of NF-κB. This results in reduced expression of pro-inflammatory cytokines such as tumor necrosis factor (TNF)-α, interleukin (IL)-1β, and IL-6, as well as inflammatory enzymes including cyclooxygenase-2 (COX-2) and inducible nitric oxide synthase. Since persistent NF-κB activation is implicated in chronic inflammatory diseases, autoimmune disorders, and cancer progression, its inhibition contributes to both anti-inflammatory and anticancer effects of BAs [19,20].

Modulation of MAPKs

Studies indicate that BAs suppress activation of stress-responsive MAPK pathways, including p38 MAPK and c-Jun N-terminal kinase (JNK). These pathways regulate inflammatory gene expression and are associated with insulin resistance and tissue damage under chronic inflammatory conditions. Table 2 shows inhibition of MAPK signaling reduces cytokine production and oxidative stress, thereby improving metabolic homeostasis. In addition, activation of AMP-activated protein kinase (AMPK) by BAs enhances insulin sensitivity and promotes glucose uptake, contributing to their antidiabetic effects. Thus, MAPK and AMPK modulation link the anti-inflammatory and metabolic benefits of BAs [21-23].

Initiation of apoptosis in cancer cells

BAs induce apoptosis in cancer cells through activation of caspase cascades, leading to cleavage of poly (ADP-ribose) polymerase (PARP) and programmed cell deathas shown in Table 2. AKBA has been shown to cause G2/M phase cell cycle arrest by downregulating cyclin B1, CDK1, and FOXM1, while upregulating p21 and GADD45A. This disruption of cell cycle progression suppresses tumor cell proliferation. In addition, β-BA inhibits glycolysis by targeting glucose transporter-1, reducing ATP production, and activating AMPK, which in turn suppresses the mammalian target of rapamycin pathway and limits tumor growth. BAs also inhibit topoisomerase I and IIα, impairing DNA replication and transcription, ultimately leading to DNA damage-induced apoptosis. These combined mechanisms contribute to their broad anticancer activity [24,25].

Anti-microbial action of BA

BAs exhibit antimicrobial effects primarily due to their lipophilic nature, which enables them to integrate into bacterial cell membranes and disrupt membrane integrity, leading to leakage of intracellular contents and bacterial death. In addition, AKBA inhibits bacterial DNA and RNA synthesis by interfering with essential enzymes such as DNA gyrase and topoisomerases. Since biofilm formation and microbial resistance are major clinical challenges, the ability of BAs to inhibit biofilm development further enhances their therapeutic relevance in infectious diseases [26,27].

Inhibition of human leukocyte elastase (HLE)

HLE, released from activated neutrophils, contributes to tissue destruction in chronic inflammatory diseases such as chronic obstructive pulmonary disease, rheumatoid arthritis, and cystic fibrosis. Excessive HLE activity degrades extracellular matrix proteins, leading to irreversible tissue damage. BAs inhibit HLE by binding to an allosteric site and altering enzyme conformation, thereby preventing matrix degradation. This mechanism supports their protective role in inflammatory tissue injury and airway disorders [28,29].

Antidiabetic activity of BA

BAs improve glycemic control by reducing inflammation-mediated insulin resistance and protecting pancreatic β-cells from oxidative damage. Evidence suggests that suppression of NF-κB and TNF-α signaling in adipose tissue, liver, and pancreas enhances insulin sensitivity. In addition, antioxidant activity of BAs neutralizes reactive oxygen species, preventing β-cell dysfunction and apoptosis. Activation of peroxisome proliferator-activated receptor-γ further enhances glucose uptake in peripheral tissues, while preservation of β-cell function supports sustained insulin secretion. Together, these effects contribute to improved glucose homeostasis and reduced progression of Type 2 diabetes [25-28].

CLINICAL POTENTIAL OF BAS IN CHRONIC DISEASES

Based on multitarget molecular actions (Fig. 2), BAs have been evaluated in multiple disease models. However, the strength of evidence varies considerably across conditions, with robust clinical data available for certain inflammatory disorders, while for others, evidence remains largely preclinical. Therefore, disease-wise evaluation with critical assessment is essential for realistic therapeutic positioning [16].

Inflammatory disorders

Inflammatory illnesses entail sustained immunological activation resulting in chronic inflammation, discomfort, edema, and tissue damage, even in the absence of noxious stimuli [31].

Asthma

Banno *et al*. conducted a double-blind, placebo-controlled clinical study in which 40 patients with bronchial asthma received *B. serrata* extract (300 mg, 3 times daily) for 6 weeks, resulting in clinical improvement in approximately 70% of patients compared to 27% in the placebo group, along with significant improvements in forced expiratory volume in 1 s and peak expiratory flow rate and reductions in eosinophil count and erythrocyte sedimentation rate. These findings support the anti-inflammatory efficacy of BAs in airway inflammation. However, the small sample size and short treatment duration limit the generalizability of these results. Larger, multicenter randomized controlled trials with standardized formulations are necessary to confirm long-term safety and therapeutic benefit in diverse patient populations [32,33].

IBD

Clinical evidence for BAs in ulcerative colitis is comparatively stronger. In a randomized clinical trial, patients receiving *B. serrata* extract (350 mg 3 times daily) for 6 weeks achieved remission rates exceeding 80%, which were comparable or slightly superior to sulfasalazine therapy [32]. Improvement in bowel frequency and rectal bleeding further supports its clinical utility.

Preclinical models corroborate these findings, demonstrating suppression of pro-inflammatory cytokines and oxidative stress markers, along with restoration of colonic architecture. Despite encouraging outcomes, long-term relapse prevention, optimal dosing strategies, and interactions with conventional therapies remain inadequately studied [33,34].

Arthritis

Arthritis is defined by joint inflammation, cartilage deterioration, and increasing disability. The chronic nature of arthritis, as well as the limitations of conventional therapies like NSAIDs and corticosteroids, which are frequently linked with gastrointestinal and cardiovascular side effects, have fuelled the search for safer, plant-based alternatives. BAs, particularly AKBA produced from *B. serrata*, have shown potential anti-inflammatory effects in both experimental and clinical arthritic models [35]. Orally administered *B. serrata* extract (150 mg/kg/day) in a collagen-induced arthritis rat model was effective in minimizing paw edema and arthritic scores over 28 days. Histopathological analysis showed reduced synovial hyperplasia, inflammatory cell infiltration, and pannus formation compared to untreated controls. Similarly, in adjuvant-induced arthritis models, BA treatment markedly suppressed joint swelling and serum levels of C-reactive protein and rheumatoid factor [33]. Umar *et al*. demonstrated in a study that AKBA significantly reduced serum levels of matrix metalloproteinase (MMP)-3, an enzyme implicated in cartilage breakdown, and increased antioxidant enzyme levels, including superoxide dismutase and catalase, in rats with induced arthritis. These findings indicate not only anti-inflammatory but also chondroprotective and antioxidant properties of BA [36].

Cancer

Cancer remains a major global health burden, with 14 million new cases and over 6 million deaths reported annually. BAs exhibit anticancer activity by modulating multiple signaling pathways and are known to inhibit NF-kB [37].

Breast cancer

In vitro studies using human breast cancer cell lines (MCF-7, MDA-MB-231) have demonstrated that AKBA induces dose-dependent growth inhibition and apoptosis. Saraswati and Agrawal showed that AKBA significantly inhibited MCF-7 cell viability with an IC_{50} value of approximately 20 μM, along with reduced expression of vascular endothelial growth factor and MMP-2, implicating its antiangiogenic and antimetastatic potential [38]. Another study found that BA

Table 1: The main boswellic acids derived and their pharmacological relevance

Boswellic acid	Abbreviation	Structural notes	Pharmacological activity	References
β-Boswellic acid	β-BA	Tri terpenoid compound	Anti-inflammatory activity	[10]
Acetyl-β-boswellic acid	ABA	Acetyl group at C-3	Mild anti-inflammatory and cytotoxic effect	[13]
11-Keto-β-boswellic acid	KBA	Keto group at C-11	Anti-cancer, anti-inflammatory, 5 -LOX inhibitor	[10,14]
Acetyl-11-keto-β-boswellic acid	AKBA	Acetyl at C-3+keto at C-11	Anti-microbial, Anti-cancer, anti-inflammatory, 5 -LOX inhibitor	[13,10]

5 -LOX: 5-lipoxygenase

Table 2: Molecular targets of boswellic acids and their therapeutic effects

Molecular targets	Mechanism of action	Therapeutic effect	References
5-LOX	Allosteric inhibition of 5-LOX, blocking leukotriene synthesis	Anti-inflammatory	[14]
NF-κB	Inhibition of IκB kinase (IKK) degradation, preventing NF-κB nuclear translocation	Anti-inflammatory, immunomodulatory	[16,17]
p38 MAPK and JNK	Inhibition of MAPK pathway activation	Anti-inflammatory, antidiabetic	[18]
Caspases/PARP	Caspase activation leading to PARP cleavage and apoptosis	Anticancer	[30]
Cyclin B1, CDK1, FOXM1	Downregulation causing G2/M phase cell cycle arrest	Anticancer	[20]
GLUT1/mTOR/AMPK	GLUT1 inhibition reducing glycolysis and ATP; mTOR suppression; AMPK activation	Anticancer	[21]
Topoisomerase I and IIα	Inhibition disrupting transcription and DNA replication	Anticancer	[21]
Bacterial cell membrane	Membrane integration causing leakage of intracellular contents	Antimicrobial	[22]
Bacterial topoisomerases and DNA gyrase	Inhibition of bacterial DNA and RNA synthesis	Antimicrobial	[23]
HLE	Allosteric inhibition preventing extracellular matrix degradation	Anti-inflammatory	[24]
ROS	Neutralization of ROS protecting β-cells	Antidiabetic	[26]
Peroxisome proliferator-activated receptor-γ	Activation enhancing glucose uptake	Antidiabetic	[27]

5-LOX: 5-lipoxygenase, NF-κB: Nuclear factor-κB, MAPKs: Mitogen-activated protein kinases, JNK: c-Jun N-terminal kinase, PARP: Poly (ADP-ribose) polymerase, GLUT1: Glucose transporter-1, mTOR: mammalian target of rapamycin, AMPK: AMP-activated protein kinase, ROS: Reactive oxygen species

treatment led to mitochondrial membrane depolarization, caspase-3 activation, and PARP cleavage, suggesting the intrinsic pathway of apoptosis in MDA-MB-231 cells [39,40].

Pancreatic cancer

Due to late diagnosis, a very low survival rate, and aggressive nature, pancreatic cancer is one of the deadliest diseases currently. In a recent study, it is found that the combination of metformin and BA nanoparticles effectively showed a decrease in cancer cells in the pancreas [41]. *In vitro* studies have demonstrated that AKBA suppresses proliferation and induces apoptosis in pancreatic cancer cell lines. A paper by Liu *et al*. reported that AKBA inhibited the growth of PANC-1 and BxPC-3 cells in a dose-dependent manner with IC_{50} values around 20–25 μM. Treated cells showed G1 phase arrest, increased cleaved caspase-3, and decreased Bcl-2 expression, confirming apoptosis induction as shown in Table 3 [42,43].

In an orthotopic mouse model of pancreatic cancer, oral administration of AKBA for a month resulted in a 65% decrease in tumor volume compared to controls. Immunohistochemical analysis showed decreased Ki-67 (proliferation marker) and CD31 (angiogenesis marker), indicating anti-proliferative and anti-angiogenic effects [44].

Colon cancer

Colon Cancer is developed in the larger part of the intestine. It has a very high mortality rate. An experiment conducted on cell lines of colon cancer by Hostanska *et al* observed the antiproliferative activity of AKBA due to the inhibition of cell growth and apoptosis in the colon through caspase activation and p21-dependent pathway as shown in Table 3 [45]. AKBA and KBA have also proven to work against the non-cancerous growth in the colon and rectum by inhibiting NF-B/Cox-2 signaling pathways in mice. When combined with curcumin, AKBA demonstrated antitumorigenic effects *in vitro* and *in vivo* by controlling particular cancer-related miRNAs in colorectal cancer cells, including miR-34a and miR-27a [46]. Further research on cells from human colon cancer *in vitro* revealed that the strong anticancer effects of BA may be mediated through PI3K/Akt signaling pathway inhibition, apoptosis activation, and cell cycle arrest [47].

Prostate cancer

Prostate cancer is a rapid growth of cells in the prostate gland, which has a very high mortality rate, and in a report by GLOBOCAN, there are nearly 2 million new cases of prostate cancer around the world. Therapy of extracts of BA has been proved to inhibit cell growth in PC3 prostate cancer cell lines by inhibiting the NF-kB signaling pathway as shown in Table 3 [48]. In a different investigation, it was discovered that AKBA suppresses vascular endothelial growth factor receptor 2-mediated process of formation of more cells in prostate cancer [49,50].

Lung cancer

In lung cancer, an *in vitro* investigation of the anticancer potential of 11-carbonyl BBA was conducted on H446 cells was studied. It was discovered to have inhibitory effects on lung cancer cells by activating the JNK signaling pathway, cleaving PARP, and downregulating the production of the surviving protein as shown in Table 3 [51,52].

Liver cancer

In Liver cancer studies, when KBA and AKBA's effects were assessed, it was discovered that they caused apoptosis and inhibited growth in liver cancer cells through the caspase-8-dependent pathway [53].

Metabolic disorders

Diabetes

It is a condition where the pancreas releases little or no insulin, which leads to a higher amount of glucose in the blood. Diabetes is a rising concern as the number of cases is rapidly increasing every year due to obesity and a shift in living standards. Type 1 diabetes is a condition

Table 3: Clinical and preclinical evidence on the therapeutic potential of boswellic acids in various diseases

Disease/ Condition	Study type	Intervention (Dose, Duration)	Main findings	Level of evidence	References
Asthma	Clinical trial	*Boswellia serrata* extract standardized to boswellic acids, 300 mg TID, 6 weeks	70% clinical improvement versus 27% placebo; ↓ asthma attacks, dyspnea; ↑ FEV_1, PEFR; pariha↓eosinophils, ESR	Clinical	[67]
	Pre-clinical trial	Boswellic acids	↓ BAL eosinophils, ↓ IL-4, TNF-α; reduced airway inflammation	*In vivo*	[68-70]
Ulcerative colitis (IBD)	Clinical trial	*B. serrata* extract 350 mg TID, 6 weeks	>80% remission versus 74% with sulfasalazine; ↓ rectal bleeding; improved bowel habits	Clinical	[71,72]
	Preclinical	Boswellic acids	↓ TNF-α, IL-1β, IL-6; improved colonic histology; ↑ SOD, ↓ MDA	*In vivo*	[73,74]
Arthritis	Preclinical (CIA rat model)	*B. serrata* extract 150 mg/kg/day, 28 days	↓ paw edema, ↓ arthritis scores; reduced synovial hyperplasia, inflammatory infiltration	*In vivo*	[75,76]
	Preclinical (AIA model)	Boswellic acids	↓ CRP, RF; ↓ MMP-3; ↑ SOD, catalase	*In vivo*	[77,78]
Breast cancer	*In vitro* (MCF-7, MDA-MB-231)	AKBA	IC_{50}=20 μM; ↓ VEGF, MMP-2; mitochondrial depolarization; caspase-3 activation; PARP cleavage	*In vitro*	[18,79,80]
Pancreatic cancer	*In vitro* (PANC-1, BxPC-3)	AKBA	IC_{50} 20–25 μM; G1 arrest; ↑ caspase-3; ↓ Bcl-2	*In vitro*	[81,39]
	In vivo (mouse model)	AKBA 100 mg/kg/ day, 28 days	↓ tumor volume by 65%; ↓ Ki-67, CD31	*In vivo*	[82, 83]
Colon cancer	*In vitro*	AKBA, KBA	Caspase activation, p21 upregulation; ↓ Wnt/ β-catenin, NF-κB/COX-2; PI3K/Akt inhibition	*In vitro*	[84,85]
Prostate cancer	*In vitro* (PC3 cells)	AKBA	NF-κB inhibition; VEGFR2 suppression	*In vitro*	[52,86]
Lung cancer	*In vitro* (H446 cells)	11-carbonyl BBA	JNK activation; PARP cleavage; ↓ survivin	*In vitro*	[54,87]
Liver cancer	*In vitro*	KBA, AKBA	Caspase-8 dependent apoptosis; ↓ proliferation	*In vitro*	[56,18]
Diabetes	Preclinical (alloxan-induced rats)	*Bougainvillea glabra* extract	↓ glucose, cholesterol, TG, urea, creatinine; ↑ insulin sensitivity	*In vivo*	[88-90]
	Preclinical (MLD-STZ model)	KBA, AKBA	↓ hyperglycemia, ↓ insulitis	*In vivo*	[91-93]
Atherosclerosis	Preclinical (ApoE-deficient mice)	AKBA	NF-κB inhibition; ↓ plaque formation	*In vivo*	[94,95]
Thyroid disorders	Preclinical (PTU-induced hyperthyroidism in rats)	Boswellic acids	Mild effects on T3/T4; ↓ inflammation	*In vivo*	[96,97]
Psoriasis	Preclinical (mouse model)	AKBA	Modulated TLR7/8, IRF signaling; ↓ psoriatic inflammation	*In vivo*	[98]
Parkinson's disease	Preclinical (rat model)	Boswellic acids	↓ inflammation; ↑ dopamine; improved motor function	*In vivo*	[99,100]

IBD: Inflammatory bowel disease, CIA: Collagen-induced arthritis, AIA: Adjuvant-induced arthritis, MLD-STZ: Multiple low dose streptozotocin, ApoE: Apolipoprotein E-deficient, AKBA: Acetyl-11-keto-β-boswellic acid, BAA: (23-O-(1,4′-bipiperidine-1-carbonyl) betulinic acid), KBA: 11-keto-beta-boswellic acid, FEV_1: Forced expiratory volume in 1 second, PEFR: Peak expiratory flow rate, ESR: Erythrocyte sedimentation rate, IL: Interleukin, TNF-α: Tumor necrosis factor-alpha, SOD: Superoxide dismutase, CRP: C-reactive protein, RF: Rheumatoid factor, MMP: Matrix metalloproteinase, VEGF: Vascular endothelial growth factor, VEGFR2: Vascular endothelial growth factor receptor 2, JNK: c-Jun N-terminal kinase, PARP: Poly (ADP-ribose) polymerase, TG: Triglycerides, NF-κB: Nuclear factor kappa B, IRF: Interferon regulatory factor

where the body produces no insulin, and in Type 2 diabetes, the body does not respond to the insulin produced. Type 2 diabetes is a metabolic disorder, and Type 1 diabetes is an autoimmune disorder [54]. Extracts of BA tend to improve insulin sensitivity and inhibits alpha glucosidase and hence help in maintain blood glucose levels. In a study where the rats were given alloxan and diabetes was induced; it was observed that extracts of *Boswellia glabria* (leaves and roots) produces hypoglycemic effects, decrease in enzyme activity, urea and creatinine levels, cholesterol, triglycerides, and serum glucose levels. Similarly, table 3 depicts, in diabetes caused by multiple low-dose streptozotocin, the plant's isolated components, such as KBA and AKBA, decreased hyperglycemia and avoided inflammatory responses and insulitis [55,56].

Atherosclerosis

Atherosclerosis occurs due to the plaque formation on the inside of the arteries, constricting them and giving a resistance to the flow of blood. Apolipoprotein E deficiency was induced in mice, and the effect of AKBA was studies which reported that due inhibition of Nf kb, AKBA shows promise as a potential therapeutic agent for atherosclerosisas depicted in Table 3. This research implies that plant resins belonging to the Boswellia family may be able to replace traditional therapeutic approaches for long-term inflammatory conditions like atherosclerosis [57,58].

Thyroid

Evidence supporting the role of BAs in thyroid dysfunction is limited and largely restricted to animal studies. Experimental models of chemically induced hyperthyroidism have reported mild modulation of T3 and T4 levels, possibly secondary to anti-inflammatory and antioxidant effects [59]. However, no well-controlled clinical trials have evaluated BAs in thyroid disease, and current data are insufficient to support therapeutic application. Future studies are required to clarify whether observed hormonal changes are direct or secondary to systemic anti-inflammatory effects [60,61].

Psoriasis

B. serrata gum-resin has potential in treating a variety of skin conditions, including psoriasis. A psoriasis-like mouse model and murine bone marrow-derived dendritic cells (BMDCs) were used in a study to assess the impact of AKBA. The findings supported AKBA's anti-inflammatory effects in psoriasis through TLR7/8 and interferon regulatory factor signaling pathway regulation [62,63].

Central nervous disorders

Central nervous system disorders, including Parkinson's, Alzheimer's, and cognitive impairment, may also benefit greatly from the use of BAs. In Parkinsonian rats, BA treatment has been associated with decreased

inflammatory markers, enhanced nigral tyrosine hydroxylase immunostaining, better general motor function, and elevated striatal dopamine levels. By modifying miRNA-155, AKBA demonstrated antiapoptotic and anti-amyloidogenic effects in a neuroinflammatory mouse model [64-66].

CONCLUSION

BAs, particularly AKBA and KBA, exhibit a broad spectrum of pharmacological activities through modulation of key inflammatory, apoptotic, metabolic, and immune-regulatory pathways, including inhibition of 5-LOX, suppression of NF-κB signaling, regulation of MAPK cascades, induction of apoptosis, and antioxidant effects. Consistent preclinical evidence supports their therapeutic potential in inflammatory disorders, metabolic diseases, and multiple cancer models. Among the clinical indications, asthma and ulcerative colitis demonstrate the most encouraging human trial data, whereas for conditions such as gastric ulcers, thyroid disorders, and neurological diseases, the evidence remains largely preclinical. Despite strong mechanistic justification and experimental efficacy, the translation of BAs into routine clinical practice remains limited. This disparity highlights the need for improved formulation strategies, standardized preparations, and well-designed clinical studies to validate their therapeutic applicability and optimize patient outcomes.

Future directions

Future research on BAs should prioritize the development of standardized botanical extracts with well-defined concentrations of bioactive constituents, particularly AKBA and KBA, to ensure reproducibility and dose consistency across studies. Given the inherently poor oral bioavailability of BAs due to low aqueous solubility and extensive first-pass metabolism, advanced drug delivery strategies such as lipid-based formulations, phospholipid complexes, polymeric nanoparticles, solid lipid nanoparticles, and bilayer or sustained-release systems should be explored to enhance systemic exposure and therapeutic efficacy.

Furthermore, large-scale, randomized, double-blind clinical trials are urgently required for indications with strong preclinical and preliminary clinical support, particularly asthma, IBD, arthritis, and metabolic disorders, to establish optimal dosing regimens, treatment duration, and long-term outcomes. Future studies should also investigate synergistic combinations of BAs with conventional anti-inflammatory, antidiabetic, and anticancer therapies to evaluate whether adjunctive use can improve efficacy while reducing adverse effects of standard drugs.

Although existing studies indicate a favorable safety profile, systematic long-term toxicological evaluations are necessary to assess potential cumulative effects, herb–drug interactions, and organ-specific toxicity associated with chronic administration. In addition, pharmacokinetic–pharmacodynamic correlation studies should be conducted to better understand dose–response relationships and to guide rational clinical dosing. Collectively, addressing these challenges will be critical for the successful translation of BAs from promising phytochemicals into clinically reliable phytopharmaceutical agents.

AUTHOR CONTRIBUTIONS

The authors were responsible for the literature survey, data analysis, manuscript writing, and final approval of the submitted version.

CONFLICTS OF INTEREST

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