AKBA

Cat. No.: HY-N0892

CAS No.: 67416-61-9

Molecular Formula: $C_{32}H_{48}O_5$

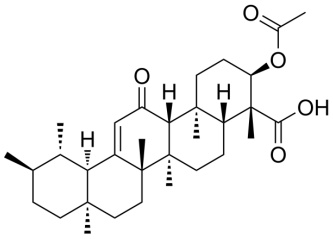
Molecular Weight: 512.72

Target: HIF/HIF Prolyl-Hydroxylase; Reactive Oxygen Species (ROS); Endogenous Metabolite

Pathway: Metabolic Enzyme/Protease; Immunology/Inflammation; NF-κB

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 5.2 mg/mL (10.14 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.9504 mL	9.7519 mL	19.5038 mL
5 mM	0.3901 mL	1.9504 mL	3.9008 mL
10 mM	0.1950 mL	0.9752 mL	1.9504 mL

Please refer to the solubility information to select the appropriate solvent.

BIOLOGICAL ACTIVITY

Description

AKBA (Acetyl-11-keto-β-boswellic acid) is an active triterpenoid compound from the extract of Boswellia serrate and a novel Nrf2 activator.

In Vitro

AKBA (Acetyl-11-keto-β-boswellic acid) significantly reduced infarct volumes and apoptotic cells, and also increased neurologic scores by elevating the Nrf2 and HO-1 expression in brain tissues in middle cerebral artery occlusion (MCAO) rats at 48 hours post reperfusion. In primary cultured neurons, AKBA increased the Nrf2 and HO-1 expression, which provided protection against OGD-induced oxidative insult. Additionally, AKBA treatment increased Nrf2 binding activity to antioxidant-response elements (ARE)[1].

AKBA (Acetyl-11-keto-β-boswellic acid) significantly inhibited human colon adenocarcinoma growth, showing arrest of the cell cycle in G1-phase and induction of apoptosis[3].

AKBA (Acetyl-11-keto-β-boswellic acid) triggered significant lipolysis in 3T3-L1 adipocytes as shown by reduced neutral lipids in cytosol and increased free fatty acids in culture medium. Increased lipolysis by AKBA was accompanied by up-regulation of lipolytic enzymes, adipocyte triglyceride lipase (ATGL) and hormone sensitive lipase (HSL), and a decreased expression of lipid droplet stability regulator perilipin. In addition, AKBA (Acetyl-11-keto-β-boswellic acid) treatment

	reduced phenotypic markers of mature adipocyte aP2, adiponectin and glut-4 in mature adipocytes[5]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	AKBA (Acetyl-11-keto-β-boswellic acid) significantly prevented the formation of intestinal adenomatous polyps without toxicity to mice. AKBA's activity both in the prevention of small intestinal and colonic polyps was more potently than aspirin. Histopathologic examination revealed that AKBA's effect, that is the reduction of polyp size and degree of dysplasia, was more prominent in larger sized polyps, especially those originating in colon[1]. AKBA (Acetyl-11-keto-β-boswellic acid) administration in mice effectively delayed the growth of HT-29 xenografts without signs of toxicity. The activity of AKBA was more potent than that of aspirin[3]. AKBA (Acetyl-11-keto-β-boswellic acid) exhibited anti-cancer activity in vitro and in vivo. With oral application in mice, AKBA significantly inhibited SGC-7901 and MKN-45 xenografts without toxicity[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Int Immunopharmacol. 2024 Nov 6;143(Pt 3):113547.
- Int Immunopharmacol. 2023 Aug:121:110501.
- Sci Rep. 2025 Apr 30;15(1):15207.

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