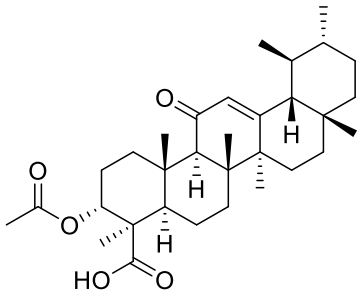
Product data sheet

MedKoo Cat#: 526492 Name: AKBA CAS#: 67416-61-9 Chemical Formula: $C_{32}H_{48}O_5$ Exact Mass: 512.35 Molecular Weight: 512.73	
Product supplied as:	Powder
Purity (by HPLC):	≥ 98%
Shipping conditions	Ambient temperature
Storage conditions:	Powder: -20°C 3 years; 4°C 2 years.
	In solvent: -80°C 3 months; -20°C 2 weeks.

1. Product description:

AKBA is an antiangiogenic and neuroprotective agent, reducing the impact of proliferative retinopathies, protecting neurons against ischemic injury involving the Nrf2/HO-1 pathway. AKBA is a naturally occurring pentacyclic triterpene isolated from the gum resin exudate from the stem of the tree B. serrata (frankincense). It selectively inhibits 5-lipoxygenase (IC50 = 1.5 µM) in an enzyme-directed, nonredox, and noncompetitive manner. 2 3-acetyl-11-keto-β-Boswellic acid and other members of the boswellic acid family have been studied for potential use in the control of inflammatory diseases, including arthritis and cancer.

2. CoA, QC data, SDS, and handling instruction

SDS and handling instruction, CoA with copies of QC data (NMR, HPLC and MS analytical spectra) can be downloaded from the product web page under "QC And Documents" section. Note: copies of analytical spectra may not be available if the product is being supplied by MedKoo partners. Whether the product was made by MedKoo or provided by its partners, the quality is 100% guaranteed.

3. Solubility data

Solvent	Max Conc. mg/mL	Max Conc. mM
DMSO	5.2	10.14

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	**1 mg**	**5 mg**	**10 mg**
1 mM	1.95 mL	9.75 mL	19.50 mL
5 mM	0.39 mL	1.95 mL	3.90 mL
10 mM	0.20 mL	0.98 mL	1.95 mL
50 mM	0.04 mL	0.20 mL	0.39 mL

5. Molarity Calculator, Reconstitution Calculator, Dilution Calculator

Please refer the product web page under section of "Calculator"

6. Recommended literature which reported protocols for in vitro and in vivo study

In vitro study

1. Ahmad S, Khan SA, Kindelin A, Mohseni T, Bhatia K, Hoda MN, Ducruet AF. Acetyl-11-keto-β-boswellic acid (AKBA) Attenuates Oxidative Stress, Inflammation, Complement Activation and Cell Death in Brain Endothelial Cells Following OGD/Reperfusion. Neuromolecular Med. 2019 Dec;21(4):505-516. doi: 10.1007/s12017-019-08569-z. Epub 2019 Sep 12. PMID: 31515728.

In vivo study

1. Han L, Xia Q, Zhang L, Zhang X, Li X, Zhang S, Wang L, Liu C, Liu K. Induction of developmental toxicity and cardiotoxicity in zebrafish embryos/larvae by acetyl-11-keto-β-boswellic acid (AKBA) through oxidative stress. Drug Chem Toxicol. 2019 Oct 28:1-8. doi: 10.1080/01480545.2019.1663865. Epub ahead of print. PMID: 31656113.

7. Bioactivity

Biological target:

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

Product data sheet

In vitro activity

bEND.3 cells were subjected to OGD/reperfusion to investigate the protective role of AKBA in this model. It was found that AKBA treatment attenuated endothelial cell death and oxidative stress assessed by means of TUNEL assay, cleaved-caspase-3, and dihydroethidium (DHE) staining. Furthermore, OGD downregulated tight junction proteins ZO-1 and Occludin levels, and increased the expressions of inflammatory cytokines TNF-α, ICAM-1, and complement C3a receptor (C3aR). It was also noticed that the increased phosphorylation of ERK 1/2 in bEND.3 cells in OGD group. AKBA treatment significantly attenuated expression levels of these inflammatory proteins and prevented the degradation of ZO-1 and Occludin following OGD. In conclusion, AKBA treatment provides protection against endothelial cell dysfunction following OGD by attenuating oxidative stress and inflammation.

Reference: Neuromolecular Med. 2019 Dec;21(4):505-516. https://dx.doi.org/10.1007/s12017-019-08569-z

In vivo activity

A developmental toxicity assay in zebrafish embryos/larvae from 4 to 96 hours post-fertilization (hpf) was performed and a cardiotoxicity assay was designed from 48 to 72 hpf. Markers of oxidative stress and related genes were selected to access the possible mechanisms. According to the results, AKBA induced pericardium edema, yolk-sac edema, abnormal melanin, spinal curvature, hatching inhibition and shortened body length. Further, increased SV-BA distance, reduced heart rate, increased pericardium area and decreased blood flow velocity were detected in AKBA treated groups. The inhibition of cardiac progenitor gene expression, such as Nkx2.5 and Gata4, may be related to cardiotoxicity. The activities of antioxidant enzymes were decreased and the content of MDA was increased. In addition, AKBA treatment decreased the expression levels of Mn-Sod, Cat, and Gpx. These results suggested that AKBA induced developmental toxicity and cardiotoxicity through oxidative stress.

Reference: Drug Chem Toxicol. 2019 Oct 28:1-8. https://www.tandfonline.com/doi/full/10.1080/01480545.2019.1663865