Bavachin

Cat. No.: HY-N0233

CAS No.: 19879-32-4

Molecular Formula: $C_{20}H_{20}O_4$

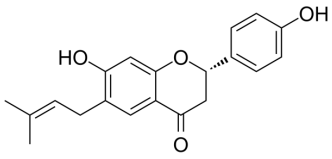
Molecular Weight: 324.37

Target: Estrogen Receptor/ERR

Pathway: Vitamin D Related/Nuclear Receptor

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 : 100 mg/mL (308.29 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.0829 mL	15.4145 mL	30.8290 mL
5 mM	0.6166 mL	3.0829 mL	6.1658 mL
10 mM	0.3083 mL	1.5414 mL	3.0829 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.08 mg/mL (6.41 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (6.41 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (6.41 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Bavachin, a flavonoid first isolated from seeds of P. corylifolia, acts as a phytoestrogen that activates the estrogen receptors ERα and ERβ with EC_{50}s of 320 and 680 nM, respectively.

IC_{50} & Target

IC50: 320 nM (ERα), 680 nM (ERβ)

In Vitro

Bavachin significantly inhibits melanin synthesis and TYR activity. Bavachin (10 μM) inhibits the expression of TYR and JNK proteins, and the expression of TYR, TRP-1, TRP-2, ERK1, ERK2 and JNK2 mRNA in A375 cells. ICI182780 and U0126 cAN

significantly reverse the bavachin treatment on the protein expression levels and the mRNA expression of TYR, TRP-1, TRP-2, ERK1, ERK2 and JNK2[1]. Bavachin accumulates lipid in a dose dependent manner in ORO staining experiments. Bavachin significantly increases the growth of preadipoctye at 10 μM compared with the control cells in MTT assay. Bavachin also increases BrdU incorporation into newly synthesized DNA during pre-adipocyte proliferation. BrdU incorporation is enhanced by insulin and further enhanced by co-treatment with insulin and bavachin at 2 and 10 μM. Bavachin activates adipogenic factors and increases PPARγ transcriptional activity in differentiated adipocytes. Bavachin enhances insulin-stimulated glucose uptake through GLUT4 translocation via Akt and AMPK pathway[2]. BVN significantly increases both hMAO-A and hMAO-B activities[3]. Bavachin shows ER ligand binding activity in competitive displacement of [^{3}H] E2 from recombinant ER. The estrogenic activity of bavachin is characterized in a transient transfection system using ERα or ERβ and estrogen-responsive luciferase plasmids in CV-1 cells with an EC_{50} of 320 nM and 680 nM, respectively. Bavachin increases the mRNA levels of estrogen-responsive genes such as pS2 and PR, and decreases the protein level of ERα by proteasomal pathway[4].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Kinase Assay [3]

The chemiluminescent assay is used to confirm PCSEE MAO-A and MAO-B inhibitory effects and to test BNN and BVN hMAO-A and hMAO-B inhibition using MAO-Glo kit. Each enzyme's Arbitrary Light Unit (ALU) is measured in the presence of PCSEE, BNN, BVN, and standard DEP as an MAO-BI positive control. Briefly, hMAO-A and hMAO-B isozymes are diluted to 2× with reaction buffer (pH 7.4) and preincubated with 4× PCSEE, BNN, BVN, or DEP working solutions at RT for 30 min in white opaque 96-well plates. For determining activity inhibition, final 8.5 μg/mL concentrations of PCSEE, BNN, BVN, and DEP are used. For IC_{50} determination, 8× PCSEE and BNN working solutions are serially diluted using reaction buffers (pH 7.4) to make a 4× concentration. Ten points' range of PCSEE (1.0 to 250.0 μg/mL) and BNN (up to 400 μM (135.4 μg/mL)) final concentrations is used. Controls used are with and without ethanol. Ethanol solvent in controls is kept to a maximum final (volume) of ≤2%. Each isozyme is substituted with the reaction buffer for the blank. Based on our preliminary optimizations and Valley's method, the reaction is initiated by adding 4× luciferin derivative substrate (LDS) for a final (concentration) of 40 and 4 μM for hMAO-A and hMAO-B reactions, respectively. The final volume per well of each reaction is 50 μL. The reaction is optimized for the amount of A and B enzyme used to be incubated for less than 3.5 h at RT. To stop the reaction and produce the luminescence signal RLDR is added to all wells, 50 μL to each well, and incubated for a further 30 min.

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Assay [1]

MTT solution (20 μL) is added to each well of the 96-well plates, the cells are cultured for 4 h, the solution is discarded, and the purple crystal is dissolved in the wells with 150 μL DMSO solution, agitated in a 37°C incubator shaker for 10 min, and the optical density (OD) is measured at 490 nm by the microplate reader.

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Acta Pharm Sin B. 2021 Jan;11(1):143-155.
- Front Med. 2021 Apr 28.
- J Ethnopharmacol. 2022 Aug 13;115593.
- Genomics. 2021 Jun 7;S0888-7543(21)00220-2.
- Oxid Med Cell Longev. 18 Oct 2021.

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REFERENCES

[1]. Wang JH, et al. Effects of bavachin and its regulation of melanin synthesis in A375 cells. Biomed Rep. 2016 Jul;5(1):87-92. Epub 2016 May 20.

[2]. Lee H, et al. Bavachin from Psoralea corylifolia Improves Insulin-Dependent Glucose Uptake through Insulin Signaling and AMPK Activation in 3T3-L1 Adipocytes. Int J Mol Sci. 2016 Apr 8;17(4):527

[3]. Zarmouh NO, et al. Evaluation of the Inhibitory Effects of Bavachinin and Bavachin on Human Monoamine Oxidases A and B. Evid Based Complement Alternat Med. 2015;2015:852194

[4]. Park J, et al. Activation of Estrogen Receptor by Bavachin from Psoralea corylifolia. Biomol Ther (Seoul). 2012 Mar;20(2):183-8

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