Product Datasheet

Technical Data

Formula	$C_{15}H_{12}O_5$					
Molecular Weight	272.25	**CAS No.**	487-52-5			
Solubility (25°C)*	In vitro	DMSO	55 mg/mL (202.02 mM)			
		Ethanol	55 mg/mL (202.02 mM)			
		Water	Insoluble			
	In vivo (Add solvents to the product individually and in order)	Oral Administration	Homogeneous suspension	CMC-NA	≥5mg/ml	Taking the 1 mL working solution as an example, add 5 mg of this product to 1 ml of CMC-Na solution, mix evenly to obtain a homogeneous suspension with a final concentration of 5 mg/ml.
		Injection	Clear solution	5%DMSO [1] → 40%PEG300 [2] → 5%Tween80 [3] → 50%ddH2O Validated by Capsid Bio labs. Should you need adjustments to this formulation, contact our sales team for custom testing.	2.75mg/ml (10.10mM)	Taking the 1 mL working solution as an example, add 50 μL of 55 mg/ml clarified DMSO stock solution to 400 μL of PEG300, mix evenly to clarify it; add 50 μL of Tween80 to the above system, mix evenly to clarify; then continue to add 500 μL of ddH2O to adjust the volume to 1 mL. The mixed solution should be used immediately for optimal results.
			Clear solution	5% DMSO [1] → 95% Corn oil Validated by Capsid Bio labs. Should you need adjustments to this formulation, contact our sales team for custom testing.	0.39mg/ml (1.43mM)	Taking the 1 mL working solution as an example, add 50 μL of 7.8 mg/ml clear DMSO stock solution to 950 μL of corn oil and mix evenly. The mixed solution should be used immediately for optimal results.

* <1 mg/ml means slightly soluble or insoluble.
* Please note that Capsid Bio tests the solubility of all compounds in-house, and the actual solubility may differ slightly from published values. This is normal and is due to slight batch-to-batch variations.
* Room temperature shipping (Stability testing shows this product can be shipped at 2-8°C for storage purposes.)

Preparing Stock Solutions

Concentration \ Volume / Mass	1 mg	5 mg	10 mg
1 mM	3.6731 mL	18.3655 mL	36.7309 mL
5 mM	0.7346 mL	3.6731 mL	7.3462 mL
10 mM	0.3673 mL	1.8365 mL	3.6731 mL
50 mM	0.0735 mL	0.3673 mL	0.7346 mL

Biological Activity

Description	Butein, a plant polyphenol isolated from **Rhus verniciflua**, is able to inhibit the activation of protein tyrosine kinase, **NF-κB** and **STAT3**, and also inhibits **EGFR**.
Targets	EGFR [1]
In vitro	Butein inhibits the epidermal growth factor (EGF)-stimulated auto-phosphotyrosine level of EGF receptor in HepG2 cells, and also inhibits tyrosine-specific protein kinase activities of EGF receptor and $p60^{c\text{-}src}$ with IC50 of 65 μM in vitro. The inhibition is competitive to ATP and non-competitive to the phosphate acceptor, poly (Glu, Ala, Tyr) 6:3:1 for EGF receptor tyrosine kinase. In contrast, this compound non-significantly inhibits the activities of serine- and threonine-specific protein kinases such as PKC or PKA. [1] This chemical inhibits Nuclear Factor(NF)-κB and NF-κB-regulated gene expression through direct inhibition of IκBα Kinase β on Cysteine 179 Residue. [2] It (10 μM) inhibits over 90% iNOS and COX-2 expression, as well as nitrite and TNF-α production in LPS-stimulated RAW 264.7 cells. This compound (10 μM) inhibits LPS-induced DNA binding activity of NF-κB, which is mediated through inhibition of the degradation of inhibitory factor-κB and phosphorylation of Erk1/2 MAP kinase, as well as increases binding of the osteopontin a vb3 integrin receptor. [3] It (20 μM) treatment induces morphologic changes of bladder cancer cells BLS(M) from elongated morphology to rounded epithelial-like cells, accompanied by downregulation of vimentin, and gaining of E-cadherin compared to untreated control cells, indicating the reversal of mesenchymal-like phenotype. This chemical (20 μM) suppresses motility and invasion capacity of BLS(M) cells, and reverts EMT-like phenotype induced by TNF-α, through the ERK1/2 and NF-κB signaling pathways. [4] It inhibits the constitutive activation of STAT3 in HepG2 cells in a dose-dependent manner, with maximum inhibition occurring at 50 μM, mediated through the inhibition of activation of upstream kinases c-Src and Janus-activated kinase2. This compound (50 μM) also could completely inhibit IL-6-induced STAT3 phosphorylation in SNU-387 cells. It downregulates the expression of cyclin D1, Bcl-2, Bcl-xL, survivin, and VEGF, markers of STAT3 activation. This chemical (50 μM) significantly enhance the apoptotic effects of doxorubicin from 18% to 55% and of paclitaxel from 15% to 42%. [5] It is as a powerful antioxidant against lipid and LDL peroxidation. This compound inhibits iron-induced lipid peroxidation in rat brain homogenate with an IC50 of 3.3 μM. It is as potent α-tocopherol in reducing the stable free radical diphenyl-2-picrylhydrazyl (DPPH) with IC0.2 of 9.2 μM. This chemical also inhibits the activity of xanthine oxidase with an IC50 of 5.9 μM. It scavenges the peroxyl radical derived from 2,2-azobis(2-amidinopropane) dihydrochloride (AAPH) in aqueous phase. Furthermore, this compound inhibits copper-catalyzed oxidation of human low-density lipoprotein (LDL) in a concentration-dependent manner. It is a chelator of ferrous and copper ions. [6]
In vivo	Butein at 2 mg/kg induces significant inhibition of hepatocellular tumor growth compared with the corn oil-treated controls. At necropsy on day 22 after initial treatment, there is more than 2-fold decrease in tumor growth in this compound-treated group (mean relativetumor burden, 3.90) compared with the control group (8.46), associated with reduced constitutive p-STAT3 (9% vs 81% of vehicle group), Bcl-2 levels (26% vs 96% of vehicle group), and increased caspase-3 level (98% vs 21% of vehicle group) in HCC tumor tissues. [5] This compound shows antifibrogenic activity. It (25 mg/kg/day) reduces serum AST and ALT activation to 35% and 69%, respectively, of control CCl_4-induced rat levels. This chemical (25 mg/kg/day) reduces liver hydroxyproline contents and TBAR4 concentration to 54% and 54%, respectively. α1(I) collagen and TIMP-1 expression in this compound-treated rats is 28% and 20.3% compared with the values for the respective CCl_4-treated control. [7]

Protocol (from reference)

Cell Assay:[5]

- **Cell lines**
  Human hepatoma cells HepG2
- **Concentrations**
  ~50 μM
- **Incubation Time**

1 days

- **Method**

  The cells (5× 10^3/mL) are incubated in triplicate in a 96-well plate in the presence or absence of indicated concentration of Butein in a final volume of 0.2 mL for different time intervals at 37 ℃. Thereafter, 20 μL MTT solution (5 mg/mL in PBS) is added to each well. After a 2-hour incubation at 37 ℃, 0.1 mL lysis buffer (20% SDS, 50% dimethylformamide) is added, incubation is continued overnight at 37 ℃, and then the optical density at 570 nm is measured by plate reader.

Animal Study:[5]

- **Animal Models**

  Human hepatocellular carcinoma xenografts HepG2

- **Dosages**

  2 mg/kg

- **Administration**

  intraperitoneal injection, 5 doses per week for 3 consecutive weeks

References

https://pubmed.ncbi.nlm.nih.gov/9571170/ https://pubmed.ncbi.nlm.nih.gov/17439942/ https://pubmed.ncbi.nlm.nih.gov/15351711/

+ Expand

RETURN POLICY

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SHIPPING & STORAGE

Capsid Bio products are transported at room temperature. If you receive the product at room temperature, please rest assured, the Capsid Bio Quality Inspection Department has conducted experiments to verify that the normal temperature placement of one month will not affect the biological activity of powder products. After collecting, please store the product according to the requirements described in the datasheet. Most Capsid Bio products are stable under the recommended conditions.

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EU Headquarters
Gajeva ulica 28, HR-10000 Zagreb, Croatia

US Headquarters
12222 Merit Dr STE 130 Dallas, Texas, 75251

UK Headquarters
121 ABILITY HOUSE, BROOKER ROAD, EN9 1JH, ESSEX

Orders: Sales@capsidbio.com
Enquiries: info@capsidbio.com
www.capsidbio.com