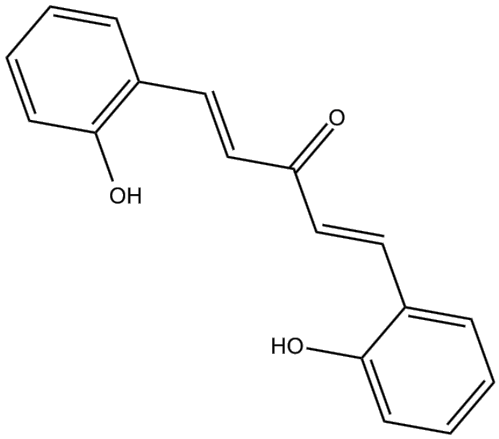
Product Data Sheet

Product Name: 2-HBA
Cat. No.: GC12545

Chemical Properties

Cas. No.	131359-24-5		
Chemical Name	(1E,4E)-1,5-*bis*(2-hydroxyphenyl)-1,4-pentadien-3-one		
SMILES	OC1=C(/C=C/C(/C=C/C2=CC=CC=C2O)=O)C=CC=C1		
Formula	$C_{17}H_{14}O_3$	M.Wt	266.3
Solubility	≤14mg/ml in ethanol;11mg/ml in DMSO;12mg/ml in dimethyl formamide	Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20°C for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		

Structure



Protocol

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Kinase experiment: Cells (20,000 per well) are grown for 24 h in 96-well plates, then exposed to 2-HBA (bis(2-hydroxybenzylidene)acetone) for either 24 h (for glutathione determination) or 48 h (for determination of enzyme activities). At the end of the exposure period, cells are collected by centrifugation (1500×g for 15 min at 4°C), washed with DPBS, and finally lysed in 0.08% digitonin. An aliquot (25 μL) is used for protein analysis. Activity of NQO1 is determined by the Prochaska test[1].

Cell experiment: After exposure to 2-HBA (bis(2-hydroxybenzylidene)acetone) for 24 h, duplicate aliquots of cells (1×106) are collected by centrifugation and washed with cold DPBS. Apoptosis is determined using the Annexin-V-FLUOS assay with simultaneous determination of the necrotic fraction by the uptake of propidium iodide[1].

References:

[1]. Dinkova-Kostova AT, et al. Bis(2-hydroxybenzylidene)acetone, a potent inducer of the phase 2 response, causes apoptosis in mouse leukemia cells through a p53-independent, caspase-mediated pathway. Cancer Lett. 2007 Jan 8;245(1-2):341-9.

Background

2-HBA is an inducer of the Keap1-Nrf2-ARE pathway.

Keap1-Nrf2-ARE directly react with Keap1, the sensor protein for inducers, leads to enhanced transcription of phase 2 genes and protection against oxidant and electrophile toxicities.

In vitro: 2-HBA could markedly increase the activities of NAD(P)H:quinone acceptor oxidoreductase 1 (NQO1) and glutathione reductase, the levels of total glutathione, as well as the phase 2 response markers. In addition, at high concentrations 2-HBA caused G2/M cell cycle arrest and apoptosis. Moreover, the mutant L1210 cell line was found to be more sensitive to the apoptotic effects of 2-HBA [1].

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In vivo: The effect of 2-HBA on the DMBA-induced expression of the Ha-ras gene in isolated RNA tissues of CBA/Ca inbred mice was investigated. According to the previous findings, elevated Ha-ras expression was obserrved even 24 h after DMBA treatment. Administration of 2-HBA with DMBA could lead to a decrease of the DMBA-induced Ha-ras gene expression in all the investigated tissues, suggesting metabolic interaction of 2-HBA and DMBA. In addiiton, administration of 2-HBA 24 h prior to the DMBA treatment was able to reduce the Ha-ras gene expression in all tested tissues except the liver, which could be the result of a possible CYP1A inducer and pro-oxidant effects of 2-HBA [2].

Clinical trial: Up to now, 2-HBA is still in the preclinical development stage.

References:
[1] A. T. Dinkova-Kostova, A. H. Cory, R. E. Bozak, et al. Bis(2-hydroxybenzylidene)acetone, a potent inducer of the phase 2 response, causes apoptosis in mouse leukemia cells through a p53-independent, caspase-mediated pathway. Cancer Letters 245, 341-349 (2007).
[2] Perjési P, Ember I, Bozak RE, Nádasi E, Rozmer Z, Varjas T, Hicks RJ. Effect of the chalcone analog E,E-bis(2-hydroxybenzylidene) acetone on the 7,12-dimethylbenz[a]anthracene-induced Ha-ras gene activity in vivo. In Vivo. 2006 Jan-Feb;20(1):141-6.