

3-n-Butylphthalide Datasheet

4th Edition (Revised in July, 2016)

[Product Information]

Name: 3-n-Butylphthalide

Catalog No.: CFN90235

Cas No.: 6066-49-5

Purity: > 98%

M.F: C₁₂H₁₄O₂

M.W: 190.24

Physical Description: Oil

Synonyms:Butylphthalide;3-Butylphthalide;3-butyl-phthalid;n-Butylphthalide;3-butyl-1(3H)

-Isobenzofuranone.

[Intended Use]

- 1. Reference standards:
- 2. Pharmacological research;
- 3. Food research;
- 4. Cosmetic research;
- 5. Synthetic precursor compounds;
- 6. Spice flavor;
- 7. Intermediates & Fine Chemicals;
- 8. Ingredient in supplements;
- 9. Aromatics:
- 10. Others.

[Source]

The root of Angelica acutilobac (Sieb. et Zucc.) Kitag.

[Biological Activity or Inhibitors]

DI-3-n-Butylphthalide(NBP), an established natural antioxidant for clinical stroke treatment in China, can reportedly reduce beta-amyloid-induced neuronal toxicity in cultured neuronal cells, and attenuate neurodegenerative changes in aged rats; it can upregulate the vesicular monoamine transporter 2 gene expression in vitro and in vivo; it protects dopaminergic (DA) neurons likely by reducing oxidative stress, offering an alternative neuroprotective medication for Parkinson's disease.^[1]

3-n-Butylphthalide may have a protective effect for diabetic brain damage through enhancing VEGF expression to inhibit caspase-3 mediated apoptosis.^[2]

3-n-Butylphthalide is a potentially beneficial drug for the treatment of ischemic stroke with multiple actions on different pathophysiological processes, NBP exerts oral anti-platelet and anti-thrombotic efficacy without perturbing systemic hemostasis in rats, and I-NBP is more potent than d- and dI-NBP as antiplatelet agent.^[3]

L-Butylphthalide may protect neurons against Abeta-induced neurotoxicity via inhibiting tau protein hyperphosphorylation.^[4]

3-n-Butylphthalide, especially its s-(-)-enantiomer, can potently reduce the release of cytochrome c, decrease the activation of caspase-3, and inhibit DNA fragmentation after transient focal cerebral ischemia; the beneficial effects of NBP on cerebral ischemia-induced apoptosis might have important implications for the study and treatment of ischemic cerebrovascular diseases.^[5]

[Solvent]

Chloroform, Dichloromethane, DMSO, Acetone.

[HPLC Method]^[6]

Mobile phase: 0.2 M Sodium Dihydrogen Phosphate (pH 4.5)- Acetonitrile=50:50;

Flow rate: 1.0 ml/min;

Column temperature: 30 °C;

The wave length of determination: 228 nm.

[Storage]

2-8°C, Protected from air and light, refrigerate or freeze.

[References]

[1] Xiong N, Huang J, Chen C, et al. Neurobiol. Aging, 2012, 33(8):1777-91.

[2] Zhang T, Jia W, Sun X. Neurol. Res., 2013, 32(4):390-6.

[3] Peng Y, Zeng X, Feng Y, et al. J. Cardiovasc Pharm., 2004, 43(6):876-81.

[4] Peng Y, Xing C, Lemere C A, et al. Neurosci. Lett., 2008, 434(2):224-9.

[5] Chang Q. Acta Pharmacol. Sin., 2003, 24(8):796-804.

[6] Wang Q, Li S, Liu P, et al. Journal of Applied Pharmaceutical Science, 2012,2(10):

16-20.

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