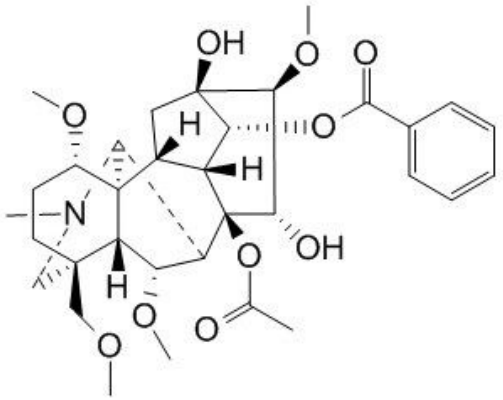
Natural Products

ChemFaces

Hypaconitine Datasheet

4th Edition (Revised in July, 2016)

[Product Information]



Name: Hypaconitine

Catalog No.: CFN99200

Cas No.: 6900-87-4

Purity: > 98%

M.F: $C_{33}H_{45}NO_{10}$

M.W: 615.71

Physical Description: White powder.

Synonyms:(1-alpha,6-alpha,14-alpha,15-alpha,16-beta)-acetatebenzoat;15-tetraol,16,16-trimethoxy-4-(methoxymethyl)-20-methyl-18-aconitane-14;(1α,6α,14α,15α,16β)1,6,16-Tri Methoxy-4-(MethoxyMethyl)-20-Methylaconitane-8,13,14,15-tetrol-8-Acetate14-Benzoate; Aconitane-8,13,14,15-tetrol,1,6,16-trimethoxy-4-(methoxymethyl)-20-methyl-,8-acetate 14-benzoate, (1α,6α,14α,15α,16β)-.

[Intended Use]

1. Reference standards;
2. Pharmacological research;
3. Synthetic precursor compounds;
4. Intermediates & Fine Chemicals;
5. Others.

[Source]

The root of *Aconitum carmichaeli Debx.*

[Biological Activity or Inhibitors]

Hypaconitine (HA), an active and highly toxic constituent derived from Aconitum species, is widely used to treat rheumatism, the hepatic cytochrome P450-catalyzed metabolism of HA. [1]

Hypaconitine, aconitine (AC) and mesaconitine(MA) are aconitum alkaloids, have highly toxic, however, their hydrolysates are considerably less toxic; the intracellular amounts in the presence of inhibitors, P-gp and BCRP were involved in the transport of AC, MA and HA; and MRP2 might transport AC, MA, and HA.[2]

Hypaconitine and aconitine produce neuromuscular blockade by reducing the evoked quantal release, the mechanism of this effect was attributed mainly to blocking of the nerve compound action potential.[3]

Hypaconitine induces QT prolongation, mediated through inhibition of KCNH2 (hERG) potassium channels in conscious dogs.[4]

Hypaconitine can inhibit CaM expression and Cx43 (Ser368) phosphorylation, and liquiritin can interfere with this kind of effect by synergistically inhibiting CaM expression and by antagonizing Cx43 (Ser368) dephosphorylation induced by hypaconitine.[5]

[Solvent]

Chloroform, Dichloromethane, DMSO, Acetone.

[HPLC Method][6]

Mobile phase: Methanol-Water-Chloroform-Triethylamine=70: 30:2:0.1;

Flow rate: 1.0 ml/min;

Column temperature: Room Temperature;

The wave length of determination: 230 nm.

[Storage]

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

[References]

[1] Ye L, Wang T, Yang C, *et al. Toxicol. Lett., 2011, 204(1):81-91.*

[2] Ling Y, Yang X, Zhen Y, *et al. Toxicol. Lett., 2013, 216(2-3):86-99*.

[3] Muroi M, Kimura I, Kimura M. *Neuropharmacology, 1990, 29(6):567-72.*

[4] Xie S, Ying J, Liu A, *et al. J. Ethnopharmacol., 2015, 166:375-9.*

[5] Yi M, Peng W, Chen X, *et al. J. Pharm. Pharmacol., 2012, 64(11):1654-8.*

[6] Zheng H S, Feng N P. *Chinese Journal of Pharmaceutical Analysis, 2005, 25(1):34-6.*

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