

L-Nicotine Datasheet

5th Edition (Revised in January, 2017)

[Product Information]

Name: L-Nicotine

Catalog No.: CFN99512

Cas No.: 54-11-5

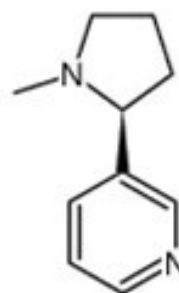
Purity: >=98%

M.F: C₁₀H₁₄N₂

M.W: 162.23

Physical Description: Oil

Synonyms: 3-[(2S)-1-methyl-2-pyrrolidinyl]pyridine.



[Intended Use]

1. Reference standards;
2. Pharmacological research;
3. Food research;
4. Cosmetic research;
5. Synthetic precursor compounds;
6. Intermediates & fine chemicals;
7. Others.

[Source]

The herbs of *Nicotiana tabacum*.

[Biological Activity or Inhibitors]

Nicotine is a potent inhibitor of cardiac A-type K⁺ channels, with blockade probably due to block of closed and open channels, this action may contribute to the ability of nicotine to affect cardiac electrophysiology and induce arrhythmias.^[1]

Nicotine has a two-phase effect on osteoblasts, showing as low level of nicotine could promote the proliferation and differentiation of the rabbit osteoblasts while the high level gets the opposite effect, vitamin C can antagonize the inhibitory effect of higher concentration of nicotine on proliferation and differentiation of osteoblasts in part.^[2]

Chronic nicotine exposure augments atherosclerosis by enhancing the production of proinflammatory cytokines by macrophages, which, in turn, activate atherogenic NF-kappaB target genes in the aortic lesions.^[3]

Nicotine is able to activate mitogenic signalling pathways, which promote cell growth or survival as well as increase chemoresistance of cancer cells, nicotine activates its downstream signalling to interfere with the ubiquitination process and prevent Bcl-2 from being degraded in lung cancer cells, resulting in the increase of chemoresistance.^[4]

Intramuscular administration of nicotine for 3 weeks can not increase arteriogenesis in ischemic hindlimb of rabbits, but is capable of significantly promoting intramyocardial angiogenesis, it can also accelerate intimal proliferation and thickening of balloon catheter denuding injury iliac artery, so it may contribute to the development of restenosis.^[5]

[Solvent]

Chloroform, Dichloromethane, Ethyl Acetate, DMSO, Acetone, etc.

[HPLC Method]^[6]

Mobile phase: H₂O-Methanol-0.1 M Buffer acetate (pH = 4.5)- Acetonitrile - Acetic acid =74 : 3 : 20 : 2 : 1 , adjusted to pH = 4.2 with diethylamine;

Flow rate: 0.7 ml/min;

Column temperature: Room Temperature;

The wave length of determination: 254 nm.

[Storage]

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

[References]

- [1] Wang H, Shi H, Zhang L, *et al. Circulation*, 2000, 102(10):1165-71.
- [2] Shen Y, Cheng S, Nie P, *et al. Chinese Journal of Cell Biology*, 2012, 40(10):627-38.
- [3] Lau P P, Li L, Merched A J, *et al. Arterioscler Thromb. Vasc. Biol.*, 2006, 26(1):143-9.
- [4] Nishioka T, Luo L Y, Shen L, *et al. Br.J. Cancer*, 2014, 110(7):1785-92.
- [5] Ruixing Y, Qi B, Tangwei L, *et al. Cardiology*, 2007, 107(2):122-31.
- [6] G. R. Pereira, J. M. Marchetti, M. V. L. B. Bentley. *Anal. Lett.*, 2001, 34(10):1669-76.

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