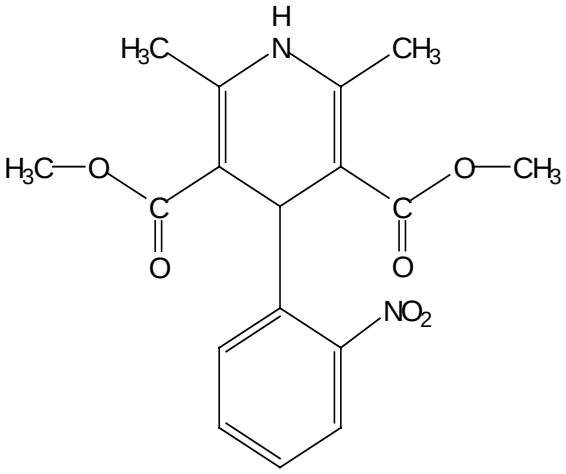
# NIFEDIPINE

## Sigma Prod. No. N7634

**CAS NUMBER:** 21829-25-4
**SYNONYMS:** BAY 1040; BAY-a 1040; Corinfar; Procardia, Adalat, Nifedin[1]



**PHYSICAL DESCRIPTION:**

Appearance: Yellow powder[2]
Melting point: 172-174°C[3]
Molecular formula: $C_{17}H_{18}N_2O_6$
Molecular weight: 346.3.
Purity: Not less than 98% by Thin-Layer Chromatography.[2]
$E^M$(340nm) = 5010 (Methanol)
$E^M$(235nm) = 21,590 (Methanol)
The distribution coefficient in an octanol-water system is about 10,000:1. The UV, IR, $^{13}$CNMR, mass spectra and chromatographic methods of analysis have been reported.[4]

**METHOD OF PREPARATION:**

Synthetic methods of preparation have been reported.[4,5,6]

**STABILITY / STORAGE AS SUPPLIED:**

Nifedipine is expected to be stable for at least two years when stored at 2-8°C.[2]

**SOLUBILITY / SOLUTION STABILITY:**

Nifedipine can be dissolved in DMSO at 50 mg/ml[4]. It is sparingly soluble in absolute ethanol.[7] Herembert, T. et al., dissolved nifedipine in absolute ethanol (no concentration reported); the maximum ethanol concentration in cultures was 0.2% without any effect of solvent on the cells.[8] Nifedipine is soluble (g/L, at 20°C) in the following solvents: acetone, 250; methylene chloride, 160; chloroform, 140; ethyl acetate, 50; methanol, 26; ethanol, 17.[4] It is practically insoluble in water. The solubilities at 37°C in buffer solutions of different pH values are: pH 4, 0.0058 g/L; pH 7, 0.0056 g/L; pH 9.0, 0.0078 g/L; pH 13, 0.006 g/L.[4]

**SOLUBILITY / SOLUTION STABILITY:** (continued)

Nifedipine solutions are unstable and extremely photosensitive. Decomposition parameters of photodegradation have been reported.[4] The compound is converted to a nitrosophenylpyridine derivative when exposed to daylight or certain wavelengths of artificial light; exposure to UV light may lead to the formation of a nitrophenylpyridine derivative. Solutions should be prepared immediately before use in the dark or under light of wavelength greater than 420 nm. Protect solutions from light and air.[7] Studies in an electrolyte solution indicated that nifedipine degraded more rapidly at 25°C than at 4°C even when solutions were protected from light. The concentration declined to about 90% of the orginal value within 6 hours of preparation.[9]

**USAGE/APPLICATIONS:**

Nifedipine is reported to inhibit $Ca^{2+}$-sensitive $K^+$ channels at 100 µM.[10] Doses for different animals have been reported.[11] In randomly growing cultures of aortic cells of rats, nifedipine at 10 µM inhibited cell proliferation. Nifedipine also inhibited serum-induced DNA synthesis at the same concentration possibly by acting on the early $G_1$ phase. The results of this study suggest that nifedipine may alter the cell cycle of cultured aortic cells and the existence in aortic fibroblasts of interactions between calcium channel blockers and the mitogenic signalling pathways of growth factors contained in serum.[8] Animal studies on the pharmacokinetics and biotransformation of radioactive labelled nifedipine were reported.[12] A growth hormone releasing peptide (GHRP-1)-induced increases in calcium ion concentration and growth hormone release were shown to be suppressed by nifedipine at 10 µM in rat anterior pituitary cells.[13] Calcium ion influx in unstimulated vascular smooth muscle cell was inhibited 44% by 10 µM nifedipine.[14]

**GENERAL NOTES:**

Nifedipine is a dihydropyridine L-type voltage sensitive calcium-channel blocker with peripheral and coronary vasodilator properties.[7,15] Its mode of action is at the slow channel where it inhibits calcium ion influx into the cells. In the heart, nifedipine as well as verapamil and diltiazem depress cardiac contractions and heart rate. The pharmacodynamic and pharmacokinetic effects of nifedipine have been published.[6,15,16,17] Swanson, T. et al. report that nifedipine exerts part of its physiological actions through potentiation of adenosine, an endogenous calcium channel blocker, and suggest that nifedipine may act through a mechanism much more complex than simple calcium channel blockade.[18] Additional mechanisms of action have been proposed.[6]

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**CITED REFERENCES:**


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**ADDITIONAL REFERENCES:**


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