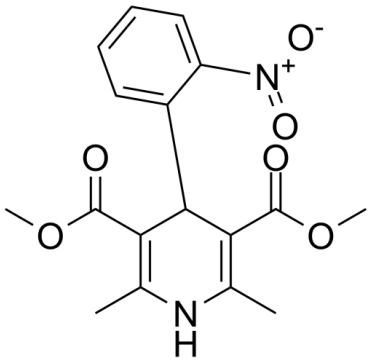
Data Sheet

Product Name:	Nifedipine
Cat. No.:	CS-2296
CAS No.:	21829-25-4
Molecular Formula:	C17H18N2O6
Molecular Weight:	346.33
Target:	Autophagy; Calcium Channel
Pathway:	Autophagy; Membrane Transporter/Ion Channel; Neuronal Signaling
Solubility:	DMSO : ≥ 116.7 mg/mL (336.96 mM)



BIOLOGICAL ACTIVITY:

Nifedipine (BAY-a-1040) is a potent **calcium channel** blocker and drug of choice for cardiac insufficiencies. **In Vitro:** Nifedipine (BAY-a-1040) (100 μM) significantly lowers the viability of the WKPT-0293 Cl.2 Cells, and treatment of nifedipine (10 or 100 μM) plus FAC induces a significant reduction in cell viability, but there are no significant differences in viability between the control cells and the cells treated with 100 μM of FAC or 1 and 10 μM of nifedipine.Nifedipine (BAY-a-1040) (1, 10, or 100 μM) significantly increases iron level in WKPT-0293 Cl.2 cells. Nifedipine treatment also increases expression of TfR1, DMT1+IRE and DMT1-IRE in WKPT-0293 Cl.2 cells. In addition, co-treatment with nifedipine (100 μM) and FAC (100 μM) increases TfR1, DMT1+IRE and DMT1-IRE expression in WKPT-0293 Cl.2 cells[2]. Nifedipine plus ritodrine produces a significantly greater inhibition of contractility than each drug alone in the midrange of concentrations. The combination of nifedipine plus nitroglycerin or nifedipine plus atosiban produces a significantly greater inhibition than nitroglycerin or atosiban alone but not greater than nifedipine. The combination of nifedipine plus NS-1619 (Ca^{2+}-activated K^+ [BKCa] channel opener) reduces the inhibitory effect of each drug[3]. Nifedipine (BAY-a-1040) (2 μM) significantly inhibits P. capsici mycelial growth and sporulation. Nifedipine (BAY-a-1040)-induced inhibition of mycelial growth is calcium-dependent. Nifedipine (0.5 μM) increases P. capsici sensitivity to H_2O_2 in a calcium-dependent manner. Nifedipine inhibition of P. capsici virulence and expression of genes involved in pathogenicity[4]. **In Vivo:** In Nifedipine (BAY-a-1040) (50 mg/kg)- and CsA-treated rats, the BL dimensions (BLi and BLk), MD dimensions (MDk) and vertical dimensions (VHi and VHk) are significantly increased ($P < 0.05$) at the end of the 4th week[1].

PROTOCOL (Extracted from published papers and Only for reference)

Cell Assay: [2]Cell viability is assessed using an MTT assay. Briefly, a total of 25 μL MTT (1 g/L in PBS) is added to each well before incubation is conducted at 37°C for 4 h. The assay is stopped by the addition of a 100 μL lysis buffer (20% SDS in 50% N'Ndimethylformamide, pH 4.7). Optical density (OD) is measured at the 570 nm wavelength by the use of an ELX-800 microplate assay reader and the results are expressed as a percentage of the absorbance measured in the control cells. **Animal Administration:** Nifedipine is prepared in olive oil.[1]All the 30 rats are randomLy distributed into three equal groups of ten animals each. Group 1 (control) receive olive oil for the 8 weeks. Group 2 and Group 3 receive a combination of CsA (30 mg/kg body weight) and Nf (50 mg/kg body weight) in olive oil for 8 weeks. In Group 3 rats, Azi (10 mg/kg body weight) is added to this regimen, in the 5th week. The total study period is 8 weeks.

References:

[1]. Ratre MS, et al. Effect of azithromycin on gingival overgrowth induced by cyclosporine A + nifedipine combination therapy: A morphometric analysis in rats. J Indian Soc Periodontol. 2016 Jul-Aug;20(4):396-401.

[2]. Yu SS, et al. Nifedipine Increases Iron Content in WKPT-0293 Cl.2 Cells via Up-Regulating Iron Influx Proteins. Front Pharmacol. 2017 Feb 13;8:60

[3]. Carvajal JA, et al. The Synergic In Vitro Tocolytic Effect of Nifedipine Plus Ritodrine on Human Myometrial Contractility. Reprod Sci. 2017 Apr;24(4):635-640.

[4]. Liu P, et al. The L-type Ca(2+) Channel Blocker Nifedipine Inhibits Mycelial Growth, Sporulation, and Virulence of Phytophthora capsici. Front Microbiol. 2016 Aug 4;7:1236.

CAIndexNames:

3,5-Pyridinedicarboxylic acid, 1,4-dihydro-2,6-dimethyl-4-(2-nitrophenyl)-, 3,5-dimethyl ester

SMILES:

O=C(C1=C(C)NC(C)=C(C(OC)=O)C1C2=CC=CC=C2[N+]([O-])=O)OC

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