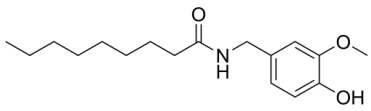
Data Sheet

Product Name:	Nonivamide
Cat. No.:	CS-3003
CAS No.:	2444-46-4
Molecular Formula:	$C_{17}H_{27}NO_3$
Molecular Weight:	293.40
Target:	TRP Channel
Pathway:	Membrane Transporter/Ion Channel; Neuronal Signaling
Solubility:	DMSO : 100 mg/mL (340.83 mM; Need ultrasonic)



BIOLOGICAL ACTIVITY:

Nonivamide is a agonist, which exhibits 4d-**EC_{50}** value of 5.1 mg/L in static toxicity tests. IC50 & Target: TRPV1[1] **In Vitro:** Nonivamide, a synthetic derivate of natural capsaicin, has an effective antifouling activity. Capsaicin exhibits 4d-EC_{50} values of 5.5±0.5 mg/L, 23±2 mg/L, 6.9±0.2 mg/L, and 15.6±0.4 mg/L in static toxicity tests conducted using Pseudomonas putida, Lake Erie bacteria, Vibrio natriegens, and Vibrio parahaemolyticus, respectively. A significant growth inhibitory effect ($p<0.01$) is observed in the group treated with 1 mg/L of Nonivamide for 4 d, and the EC_{50} value (4 d-EC_{50}) is 5.1 mg/L[1]. Nonivamide treatment causes calcium release from the ER and altered the transcription of growth arrest- and DNA damage-inducible transcript 3 (GADD153), GADD45α, GRP78/BiP, ATF3, CCND1, and CCNG2) in a manner comparable with prototypical ER stress-inducing agents. ER calcium flux is evaluated by pretreating cells with 2.5 μM thapsigargin for 5 min followed by addition of 2.5 μM Nonivamide. Treatment of TRPV1-overexpressing cells with 2.5 μM Nonivamide produces marked increases in cytosolic calcium due to release of calcium from ER stores. Treatment of TRPV1-overexpressing cells with 1 μM Nonivamide causes an approximate 50% loss in cell viability after a 24-h period. BEAS-2B cells treated with 100 and 200 μM Nonivamide also exhibits a shift in the relative amount of EIF2α-P and an increase in the expression of GADD153 mRNA and protein[2]. Treatment with Nonivamide reduces lipid accumulation to a similar extent as CAP; the effects are not different from the effects after CAP treatment at any of the tested concentrations. Compared to untreated control cells, treatment with Nonivamide decreases lipid accumulation by 5.34±1.03% ($P<0.05$) at 0.01 μM up to 10.4±2.47% ($P<0.001$) at 1 μM[3].

PROTOCOL (Extracted from published papers and Only for reference)

Cell Assay: Nonivamide is dissolved in ethanol to 1,000× stock solutions freshly each time and final ethanol concentration during the assays never exceeded 0.2% (v/v)[3].[3]In the MTT assay, the reduction of yellow tetrazolium salt MTT to a purple formazan by mitochondrial and ER enzymes is used as a measure for cell viability. Cells are seeded in 96‐well plates and treated with 1 nM-10 μM CAP or Nonivamide with or without addition of 25-100 μM BCH or the corresponding ethanol concentration (0.1-0.2% (v/v), solvent control) for 12 days after initiation of differentiation. Cell culture media is exchanged every second day. On Day 12, 100 μL of the MTT working reagent (0.83 mg/mL MTT diluted in PBS/serum-free media (1:5)), is added to each well, and cells are incubated at 37°C for approximately 15 min. The MTT working solution is removed and the purple formazan formed during incubation is dissolved in 150 μL DMSO per well. Absorbance is measured at 550 nm with 690 nm as reference wavelength using multiwell plate reader. The number of metabolically active cells is calculated relative to untreated control cells or the corresponding solvent control (100%)[3].

References:

[1]. Zhou J, et al. Toxic effects of environment-friendly antifoulant Nonivamide on Phaeodactylum tricornutum. Environ Toxicol Chem. 2013 Apr;32(4):802-9.

[2]. Thomas KC, et al. Transient receptor potential vanilloid 1 agonists cause endoplasmic reticulum stress and cell death in human lung cells. J Pharmacol Exp Ther. 2007 Jun;321(3):830-8.

[3]. Rohm B, et al. Nonivamide enhances miRNA let-7d expression and decreases adipogenesis PPARγ expression in 3T3-L1 cells. J Cell Biochem. 2015 Jun;116(6):1153-63.

CAIndexNames:

Nonanamide, N-[(4-hydroxy-3-methoxyphenyl)methyl]-

SMILES:

CCCCCCCCC(NCC1=CC=C(O)C(OC)=C1)=O

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