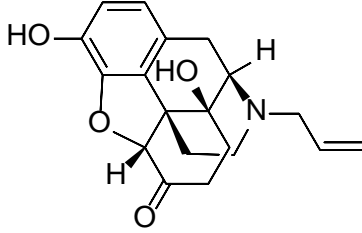


Product data sheet



MedKoo Cat#: 318301 Name: Naloxone CAS: 465-65-6 (free base) Chemical Formula: C ₁₉ H ₂₁ NO ₄ Exact Mass: 327.1471 Molecular Weight: 327.3743		
Product supplied as:		Powder
Purity (by HPLC):		≥ 98%
Shipping conditions		Ambient temperature
Storage conditions:		Powder: -20°C 3 years; 4°C 2 years. In solvent: -80°C 3 months; -20°C 2 weeks.

1. Product description:

Naloxone is a μ -opioid receptor (MOR) inverse agonist. Naloxone has an extremely high affinity for μ -opioid receptors in the central nervous system (CNS). Its rapid blockade of those receptors often produces rapid onset of withdrawal symptoms. Naloxone also has an antagonist action, though with a lower affinity, at κ - (KOR) and δ -opioid receptors (DOR). The mechanism of action is not completely understood, but studies suggest it functions to produce withdrawal symptoms by competing for opiate receptor sites within the CNS (a competitive antagonist, not a direct agonist), thereby preventing the action of both endogenous and xenobiotic opiates on these receptors without directly producing any effects itself. Naloxone was approved for opioid overdose by the Food and Drug Administration in 1971.

2. CoA, QC data, SDS, and handling instruction

SDS and handling instruction, CoA with copies of QC data (NMR, HPLC and MS analytical spectra) can be downloaded from the product web page under "QC And Documents" section. Note: copies of analytical spectra may not be available if the product is being supplied by MedKoo partners. Whether the product was made by MedKoo or provided by its partners, the quality is 100% guaranteed.

3. Solubility data

Solvent	Max Conc. mg/mL	Max Conc. mM
TBD	TBD	TBD

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	3.05 mL	15.27 mL	30.55 mL
5 mM	0.61 mL	3.05 mL	6.11 mL
10 mM	0.31 mL	1.53 mL	3.05 mL
50 mM	0.06 mL	0.31 mL	0.61 mL

5. Molarity Calculator, Reconstitution Calculator, Dilution Calculator

Please refer the product web page under section of "Calculator"

6. Recommended literature which reported protocols for in vitro and in vivo study

In vitro study

1. Bimonte S, Barbieri A, Cascella M, Rea D, Palma G, Del Vecchio V, Forte CA, Del Prato F, Arra C, Cuomo A. The effects of naloxone on human breast cancer progression: in vitro and in vivo studies on MDA.MB231 cells. *Onco Targets Ther.* 2018 Jan 3;11:185-191. doi: 10.2147/OTT.S145780. PMID: 29379300; PMCID: PMC5757202.
2. Tsai RY, Cheng YC, Wong CS. (+)-Naloxone inhibits morphine-induced chemotaxis via prevention of heat shock protein 90 cleavage in microglia. *J Formos Med Assoc.* 2015 May;114(5):446-55. doi: 10.1016/j.jfma.2014.12.004. Epub 2015 Jan 31. PMID: 25649471.

In vivo study

1. Bimonte S, Barbieri A, Cascella M, Rea D, Palma G, Luciano A, Forte CA, Cuomo A, Arra C. Naloxone Counteracts the Promoting Tumor Growth Effects Induced by Morphine in an Animal Model of Triple-negative Breast Cancer. *In Vivo.* 2019 May-Jun;33(3):821-825. doi: 10.21873/invivo.11545. PMID: 31028203; PMCID: PMC6559888.

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2. Xu YF, Fu LL, Jiang CH, Qin YW, Ni YQ, Fan JW. Naloxone inhibition of lipopolysaccharide-induced activation of retinal microglia is partly mediated via the p38 mitogen activated protein kinase signalling pathway. *J Int Med Res.* 2012;40(4):1438-48. doi: 10.1177/147323001204000422. PMID: 22971495.

7. Bioactivity

Biological target:

Naloxone is a μ -opioid receptor (MOR) inverse agonist.

In vitro activity

The results showed that morphine directly enhanced microglia chemotaxis and membrane ruffling and that these actions were accompanied by an increase in Iba1 protein expression in the microglia, especially in the ruffling membrane. Pretreatment with 1nM (+)-naloxone significantly inhibited microglia migration, activation, and reduced Iba1 expression in microglia. On the basis of these results, Iba1 is involved in microglia migration and that ultralow dose (+)-naloxone may regulate actin cytoskeleton dynamics.

Reference: *J Formos Med Assoc.* 2015 May;114(5):446-55. <https://pubmed.ncbi.nlm.nih.gov/25649471/>

In vivo activity

The addition of naloxone at 1.0 or 2.0 μ M significantly inhibited the LPS-induced production of TNF α and IL-1 β ($P < 0.01$ for all comparisons). The lowest concentration of naloxone (0.5 μ M) significantly inhibited IL-1 β ($P < 0.05$), but not TNF- α production. Western blotting showed that p38 MAPK, JNK and ERK were constitutively present in microglia (Fig. 2A). LPS treatment induced a visible increase in the amount of P-p38 MAPK, but p38 MAPK, JNK, P-JNK, ERK and P-ERK appeared unchanged. Pretreatment with naloxone dose-dependently reversed the LPS-induced increase in P-p38 MAPK ($P < 0.05$, Fig. 2B). Naloxone had no significant effect on the levels of P-JNK or P-ERK (Fig. 2B). Immunofluorescence microscopy showed weak P-p38 MAPK immunostaining in OX42-positive rat microglial cells in untreated cultures (Fig. 3A). LPS-activation was accompanied by an increase in P-p38 MAPK immunostaining (Fig. 3B). Pretreatment with naloxone (Fig. 3C) resulted in P-p38 MAPK staining similar to that seen in untreated control cultures.

Reference: *J Int Med Res.* 2012;40(4):1438-48. <https://pubmed.ncbi.nlm.nih.gov/22971495/>

Note: The information listed here was extracted from literature. MedKoo has not independently retested and confirmed the accuracy of these methods. Customer should use it just for a reference only.