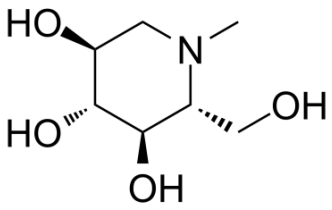
N-Methylmoranoline

Cat. No.:	HY-U00090
CAS No.:	69567-10-8
Molecular Formula:	$C_7H_{15}NO_4$
Molecular Weight:	177.2
Target:	Glucosidase
Pathway:	Metabolic Enzyme/Protease
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	N-Methylmoranoline (MOR 14) is an α-glucosidase inhibitor.
In Vitro	N-Methylmoranoline dose-dependently decreases the α-1,6-glucosidase activity in rabbit heart extract. The myocardial uptake of a considerable amount of N-Methylmoranoline is sufficient to fully inhibit alpha-1,6-glucosidase. Preischemic treatment with 25, 50, and 100 mg/kg of N-Methylmoranoline dose-dependently reduces the infarct size without altering the blood pressure or heart rate[1]. MOR-14 significantly increases levels of PKC-ε in the particulate fraction at 20 and 30 min of ischaemia and in the cytosolic fraction at 30 min of ischaemia[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	N-Methylmoranoline decreases the alpha-1,6-glucosidase activity to approximately 20%, reduces the glycogen breakdown, and attenuates the lactate accumulation at both 10 and 30 minutes of ischemia[1]. MOR-14 is protective against postischemic left ventricular dysfunction through the inhibition of glycogenolysis in the isolated rat heart[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Kinase Assay [1]	The inhibitory action of N-Methylmoranoline against myocardial α-1,6-glucosidase is first examined in rabbit heart extracts. The substrate mixture contained 44 mM glycylglycine (pH 6.5), 12.5% rabbit liver glycogen, 2.5 mM [^{14}C]glucose (20 μCi/μM), 2.1 mM EDTA, 4.1 mM mercaptoethanol, 0.02% gelatin, and N-Methylmoranoline (0, 0.01, 0.03, 0.1, 0.3, or 1.0 μM). This solution (16 μL) is warmed at 30°C for 2 minutes, and the reaction is then initiated by the addition of 4 μL of the rabbit heart homogenate. The reaction is stopped 60 minutes later by the addition of 20 μL of 0.2N HCl. An aliquot (30 μL) is spotted onto a Whatman GF/A glass fiber disk. The disk is immediately washed in 66% ethanol for 20 minutes three times each and dipped in 15 mL of acetone for 10 minutes. Then the disk is dried, and the [^{14}C] activity incorporated into glycogen is measured with a liquid scintillation counter[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [1]	Rabbits: To investigate the infarct size-reducing effect of N-Methylmoranoline, 54 rabbits are assigned randomly into drug treatment or saline control groups. There are four drug treatment groups, ie, three preischemic treatment groups given 100 mg/kg, 50 mg/kg, or 25 mg/kg of N-Methylmoranoline 10 minutes before ischemia, and one prereperfusion treatment group given 100 mg/kg of the drug 5 minutes before reperfusion. In all treatments, the injected volume is <1 mL/kg body wt. After the treatment, the coronary artery is occluded for 30 minutes and reperfused. The blood pressure and heart rate are

	monitored throughout the experiment until 20 minutes after reperfusion and are recorded at baseline, at 0, 1, 3, 5, 10, 20, and 30 minutes of ischemia, and at 5, 10, and 20 minutes of reperfusion[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Arai M, et al. N-methyl-1-deoxynojirimycin (MOR-14), an alpha-glucosidase inhibitor, markedly reduced infarct size in rabbit hearts. Circulation. 1998 Apr 7;97(13):1290-7.

[2]. Arai M, et al. Role of protein kinase C in the reduction of infarct size by N-methyl-1-deoxynojirimycin, an alpha-1,6-glucosidase inhibitor. Br J Pharmacol. 2001 Jul;133(5):635-42.

[3]. Nishida Y, et al. N-methyl-1-deoxynojirimycin (MOR-14), an alpha-glucosidase inhibitor, markedly improves postischemic left ventricular dysfunction. Heart Vessels. 2000;15(6):268-73.

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