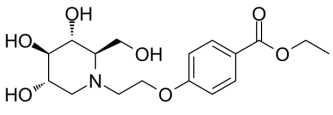
Emiglitate

Cat. No.:	HY-16194
CAS No.:	80879-63-6
Molecular Formula:	$C_{17}H_{25}NO_7$
Molecular Weight:	355.38
Target:	Others
Pathway:	Others

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



BIOLOGICAL ACTIVITY

Description	Emiglitate (BAY o 1248) is a potent, selective and competitive inhibitor of α-glucoside hydrolase.
In Vitro	Emiglitate greatly suppresses the glucose-stimulated insulin release in parallel with an inhibitory effect on the activities of acid glucan-1,4-α-glucosidase and acid α-glucosidase. In contrast, the activities of acid phosphatase and N-acetyl-β-D-glucosaminidase tend to increase in the presence of the α-glucoside hydrolase inhibitor. The CO-induced amplification of the glucose-stimulated insulin release as well as of the increased activities of the acid α-glucoside hydrolases are abrogated by emiglitate and displayed the same levels as in the absence of CO. The CO-induced rise in the activities of acid phosphatase and acid N-acetyl-β-D-glucosaminidase is not appreciably affected by emiglitate[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	In fasted rats, emiglitate inducec a significant, dose-dependent increase of hepatic glycogen concentrations. The increase in hepatic glycogen is due to lysosomal storage of glycogen only. Emiglitate in the amount of 5 mg/kg b.wt. does not induce significant changes either of glycogen concentrations or at the EM-level[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Kinase Assay [1]	Effect of the selective α-glucoside hydrolase inhibitor emiglitate (100 μM) on glucose-stimulated insulin secretion and islet lysosomal enzyme activities at 12 mM glucose in the absence and presence of CO gas is studied. Islets are incubated in the absence (open columns) or presence (solid columns) of emiglitate. Experiments are performed both in the presence (the two columns to the right) and in the absence (the two columns to the left) of exogenous CO[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Mosén H, et al. Nitric oxide inhibits, and carbon monoxide activates, islet acid alpha-glucoside hydrolase activitiesin parallel with glucose-stimulated insulin secretion. J Endocrinol. 2006 Sep;190(3):681-93.

[2]. Lembcke B, et al. Lysosomal storage of glycogen as a sequel of alpha-glucosidase inhibition by the absorbed deoxynojirimycin derivative emiglitate (BAYo1248). A drug-induced pattern of hepatic glycogen storage mimicking Pompe's disease (glycogenosis type II). Res Exp Med (Berl). 1991;191(6):389-404.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com
Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA