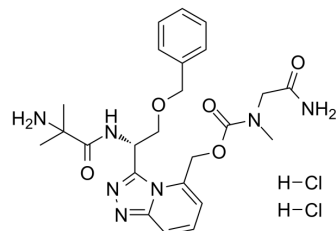


BMS-604992 dihydrochloride

Cat. No.:	HY-14495B
CAS No.:	1469750-46-6
Molecular Formula:	C ₂₄ H ₃₃ Cl ₂ N ₇ O ₅
Molecular Weight:	570.47
Target:	GHSR
Pathway:	GPCR/G Protein
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	BMS-604992 (EX-1314) dihydrochloride is a selective, orally active small-molecule growth hormone secretagogue receptor (GHSR) agonist. BMS-604992 dihydrochloride demonstrates high-affinity binding ($K_i=2.3$ nM) and potent functional activity ($EC_{50}=0.4$ nM). BMS-604992 dihydrochloride can stimulate food intake in rodents ^[1] .																
IC₅₀ & Target	EC ₅₀ : 0.4 nM (GHSR), K _i : 2.3 nM (GHSR) ^[1]																
In Vitro	BMS-604992 exhibits high-affinity binding ($K_i=2.3$ nM) and potent functional activity ($EC_{50}=0.4$ nM) for ghrelin receptor ^[1] . MCE has not independently confirmed the accuracy of these methods. They are for reference only.																
In Vivo	<p>BMS-604992 (500 µg/kg; i.p.; 5 minutes) results in a significant increase in gastric emptying compared with vehicle-treated mice^[1].</p> <p>BMS-604992 (1~1000 mg/kg; p.o.; 1 hour) Shows a dose-linear increase in plasma concentrations at the 1 hour time point and elicits a dose-responsive increase in food intake relative to vehicle-treated controls, with a minimum effective dose of approximately 10 mg/kg^[1].</p> <p>BMS-604992 (300 mg/kg; p.o.; 5~20 minutes) produces a significant difference at the 5 minutes time point^[1].</p> <p>BMS-604992 (500 µg/kg; i.p.; 4 hours) increases food intake approximately 2-fold compared with vehicle-treated controls^[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p> <table border="1" style="width: 100%; border-collapse: collapse;"> <tr> <td style="width: 30%;">Animal Model:</td> <td>C57BL/6 mice</td> </tr> <tr> <td>Dosage:</td> <td>500 µg/kg</td> </tr> <tr> <td>Administration:</td> <td>i.p.; 5 minutes</td> </tr> <tr> <td>Result:</td> <td>Resulted in a significant increase in gastric emptying compared with vehicle-treated mice.</td> </tr> </table> <table border="1" style="width: 100%; border-collapse: collapse;"> <tr> <td style="width: 30%;">Animal Model:</td> <td>C57BL/6 mice</td> </tr> <tr> <td>Dosage:</td> <td>1~1000 mg/kg</td> </tr> <tr> <td>Administration:</td> <td>P.o.; 1 hour</td> </tr> <tr> <td>Result:</td> <td>Showed a dose-linear increase in plasma concentrations at the 1 hour time point and</td> </tr> </table>	Animal Model:	C57BL/6 mice	Dosage:	500 µg/kg	Administration:	i.p.; 5 minutes	Result:	Resulted in a significant increase in gastric emptying compared with vehicle-treated mice.	Animal Model:	C57BL/6 mice	Dosage:	1~1000 mg/kg	Administration:	P.o.; 1 hour	Result:	Showed a dose-linear increase in plasma concentrations at the 1 hour time point and
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elicited a dose-responsive increase in food intake relative to vehicle-treated controls, with a minimum effective dose of approximately 10 mg/kg.

Animal Model: SD rat

Dosage: 300 mg/kg

Administration: P.o.; 5~20 minutes

Result: Observed a significant difference at the 5 minutes time point.

Animal Model: Male GhrR KO and WT mice

Dosage: 500 µg/kg

Administration: I.p.; 4 hours

Result: Increased food intake approximately 2-fold compared with vehicle-treated controls.

REFERENCES

[1]. Charoenthongtrakul S, et al. Enhanced gastrointestinal motility with orally active ghrelin receptor agonists. J Pharmacol Exp Ther. 2009;329(3):1178-1186.

Caution: Product has not been fully validated for medical applications. For research use only.

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