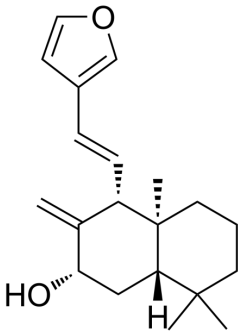
Coronarin A

Cat. No.:	HY-N3628
CAS No.:	119188-33-9
Molecular Formula:	$C_{20}H_{28}O_2$
Molecular Weight:	300.44
Target:	mTOR; Ribosomal S6 Kinase (RSK)
Pathway:	PI3K/Akt/mTOR; MAPK/ERK Pathway
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

Coronarin A is an orally active natural compound that inhibits mTORC1 and S6K1 to increase IRS1 activity. Coronarin A shows anti-inflammatory activity and can also be used for type 2 diabetes mellitus research[1].

IC_{50} & Target

mTORC1	S6K1

In Vitro

Coronarin A (3-30 μM; 4 or 12 h) stimulates glycogen synthesis through activating PI3K/Akt/GSK3β signaling and inhibits gluconeogenesis by activating ERK-dependent Wnt/β-catenin/TCF7L2 pathway in rat primary hepatocytes[1].
Coronarin A (1-30 μM; 4 h) increases tyrosine phosphorylation of IRS1 through inhibiting mTOR/S6K1 signaling[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[1]

Cell Line:	Primary rat hepatocytes
Concentration:	1, 3, 10 and 30 μM
Incubation Time:	4 h
Result:	Increased the Akt and GSK3β phosphorylation dose-dependently. Dose-dependently stimulated the phosphorylation of both ERK1 and ERK2. Increased the phosphorylation of β-catenin and mitogen-activated protein kinase kinase (MEK). Dose-dependently enhanced the tyrosine phosphorylation of IRS1 at Tyr1222, whereas the serine phosphorylation of IRS1 was dose-dependently inhibited. Reduced the phosphorylation of mTOR, S6K1 and S6.

Cell Viability Assay[1]

Cell Line:	Primary rat hepatocytes
Concentration:	1, 3, 10, 30, 100 and 300 μM
Incubation Time:	5.5 h or 12 h
Result:	Showed no toxicity at 1-30 μM, decreased cell viability after 12 h incubation at 100 μM.

In Vivo

Coronarin A (30 or 100 mg/kg; i.p. or p.o.; once daily for 22 days) ameliorates hyperglycemia in mice[1].

Coronarin A (100 mg/kg; p.o.; once daily for 22 days) inhibits the mTOR/S6K1 pathway to activate PI3K/Akt and ERK/β-catenin signaling in livers of ob/ob mice[1].

Pharmacokinetic properties of Coronarin A after single administration[a] in ***ob/ob*** mice[1].

Coronarin A	$t_{1/2}$ (h)	t_{max} (h)	C_{max} (ng/mL)	AUC_{0-t} (ng⋅h/mL)	$AUC_{0-\infty}$ (ng⋅h/mL)	MRT (h)
i.p.	14.8	1.0	1073	4571	11045	21.7
p.o.	3.01	1.0	388	1694	1856	4.88

Data are presented as the mean of three mice.

[a]Coronarin A was intraperitoneally or orally administered at 30 mg/kg to ob/ob mice.

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Animal Model:	Male ob/ob mice[1]
Dosage:	30 mg/kg (IP) or 100 mg/kg (PO)
Administration:	Oral or intraperitoneal administration, once daily for 22 days
Result:	Significantly decreased the non-fasting and fasting blood glucose. Significantly reduced the serum insulin concentration at 15 min after glucose loading, reduced the average daily food intake while the body weight was unaffected. Increased hepatic glycogen content and the expression levels of gluconeogenic gene Pck1 and G6pc were significantly decreased.

Animal Model:	Female ob/ob mice[1]
Dosage:	30 mg/kg
Administration:	Intraperitoneal or oral administration (Pharmacokinetic Analysis)
Result:	Intraperitoneal injection exhibited higher plasma exposure than oral gavage at the same dose of 30 mg/kg, with C_{max} value of 1073 and 388 ng/mL, respectively.

REFERENCES

[1]. Huang SL, et al. Coronarin A modulated hepatic glycogen synthesis and gluconeogenesis via inhibiting mTORC1/S6K1 signaling and ameliorated glucose homeostasis of diabetic mice. Acta Pharmacol Sin. 2022 Sep 9.

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

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