

Product Data Sheet

Product Name: CDN1163

Cat. No.: GC31332

Chemical Properties

Cas. No. 892711-75-0

SMILES O=C(NC1=C2N=C(C)C=CC2=CC=C1)C3=CC=C(OC(C)C)C=C3

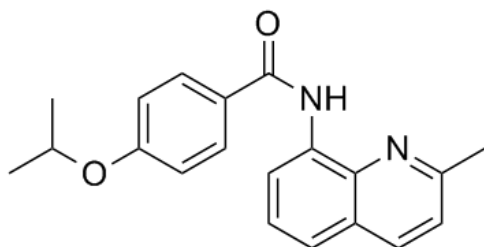
Formula C20H20N2O2 M.Wt 320.39

Solubility DMSO : 50 mg/mL (156.06 mM); Water : < 0.1 mg/mL (insoluble) Storage Store at -20°C

General tips For obtaining a higher solubility, please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution : ship with blue ice All other available size: ship with RT, or blue ice upon request.

Structure



Protocol

Cell experiment [1]:

Cell lines

MIN6 cells (mouse pancreatic beta cell line)

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Preparation Method	MIN6 cells were maintained in Dulbecco's minimal essential medium (DMEM) supplemented with 10% fetal bovine serum (FBS) at 37°C, 5% CO ₂ . MIN6 cells were treated with CDN1163 at 5μM for 24h or 48h; glucose-stimulated insulin secretion was measured by ELISA, cytosolic Ca ²⁺ oscillation was recorded by Fura-2, ER and mitochondrial Ca ²⁺ levels were measured by G-CEPIA1er/D1ER and CEPIA-2mt/mtD3cpv, mitochondrial membrane potential was measured by JC1 and TMRM, oxygen consumption rate was measured by Seahorse XF-96, ATP content was measured by bioluminescence, protein and mRNA expression were analyzed by Western blot and PCR, and growth hormone secretion was measured by ELISA.
Reaction Conditions	5μM; 24h or 48h
Applications	CDN1163 augmented glucose-stimulated insulin secretion and high K ⁺ induced insulin secretion, potentiated cytosolic Ca ²⁺ oscillation in response to glucose and high K ⁺ , elevated basal ER and mitochondrial Ca ²⁺ content, increased mitochondrial membrane potential, accelerated mitochondrial respiration and ATP synthesis, up-regulated mRNA and protein expression of IP3 receptors, phosphorylated IP3 receptors, PGC1α, TFAM, SOD2 and antioxidant genes. CDN1163 increased total and docked insulin granules and insulin mRNA, prevented palmitate-induced ER Ca ²⁺ depletion, mitochondrial dysfunction, reduction of insulin-positive cells and apoptotic cell death.
Animal experiment [2]:	
Animal models	10-12 week old male ob/ob mice (leptin-deficient obese mice)

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Preparation Method	ob/ob mice were injected intraperitoneally with CDN1163 (50mg/kg/day) or vehicle (10% DMSO+10% Tween 80 in 0.9% NaCl) for 5 consecutive days. After treatment, mice were fasted overnight and sacrificed 7 weeks after CDN1163 administration. Echocardiography was conducted at baseline and 7 weeks post-treatment. Blood glucose, plasma insulin, triglycerides, cholesterol, free fatty acids, and malondialdehyde were measured. Heart tissues were collected for real-time quantitative PCR, Western blotting, mitochondrial DNA copy number, and Ca ²⁺ -ATPase activity assays.
Dosage form	50mg/kg/day; i.p.; 5 consecutive days
Applications	CDN1163 attenuated ventricular dysfunction, improved fractional shortening and ejection fraction, decreased LV end systolic and diastolic dimensions, and restored E wave and deceleration time. CDN1163 reduced fasting blood glucose. CDN1163 decreased cardiac expression of lipid oxidation genes and lipogenesis genes. CDN1163 increased SERCA2a ATPase activity, upregulated protein levels of ATP5A, MTCO1, UQCRC2, SDHB, NDUFB8, PGC1 α , ERR α , NRF1, and TFAM, and elevated mitochondrial DNA content. CDN1163 upregulated antioxidant enzymes GPX1, PRDX3, SOD2, downregulated NADPH oxidase subunits p22phox, p47phox, gp91phox and nitrotyrosine, and reduced malondialdehyde formation.
References:[1] CDN1163, an activator of sarco/endoplasmic reticulum Ca ²⁺ ATPase, up-regulates mitochondrial functions and protects against lipotoxicity in pancreatic β -cells. Br J Pharmacol. 2023;180(21):2762-2776.[2] Kang S, Majid A, Hammoudi N, et al. Gene and Drug-Mediated SERCA2a Activation Restores Cardiac Function and Metabolic Balance in Diabetic Mice. Biomed Pharmacother. 2025 Nov;192:118617.	

Background

CDN1163 is an orally active allosteric activator of sarco/endoplasmic reticulum Ca²⁺-ATPase 2

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(SERCA2). CDN1163 enhances SERCA2-mediated endoplasmic reticulum Ca^{2+} uptake to restore endoplasmic reticulum calcium homeostasis, alleviate endoplasmic reticulum stress, and improve mitochondrial function. CDN1163 regulates glucose and lipid metabolism and oxidative stress through the SERCA2b-AMPK axis. CDN1163 is used for type 2 diabetes, obesity-related metabolic abnormalities, Parkinson's disease, Alzheimer's disease, and oxidative stress-induced muscle atrophy research^[1-4].

In vitro, MIN6 cells were treated with 5 μM CDN1163 for 24-48h. CDN1163 increased endoplasmic reticulum and mitochondrial Ca^{2+} content, enhanced mitochondrial membrane potential, respiration, and ATP synthesis, and upregulated IP3R, PGC1 α , TFAM, and SOD2 expression^[5]. H2K-mdx myotubes were incubated with 100 μM CDN1163 for 30min. CDN1163 reduced cytosolic Ca^{2+} levels^[6]. High glucose-induced H9c2 rat cardiomyocytes were treated with 10 μM CDN1163 for 24h. CDN1163 blocked NFATc nuclear translocation, decreased resistin and nuclear NFATc expression, increased AMPK α phosphorylation, and reduced NFATc-mediated resistin promoter luciferase activity^[7].

In vivo, ob/ob diabetic mice received intraperitoneal injection of 50mg/kg/day CDN1163 once daily for 5 days. CDN1163 improved left ventricular systolic and diastolic function, reduced fasting blood glucose, restored cardiac lipid oxidation and lipogenesis-related gene expression, increased SERCA2a ATPase activity, and decreased malondialdehyde levels^[8]. C57BL/6J mice received intraperitoneal injection of 20mg/kg CDN1163 once daily for 17 days. CDN1163 impaired spatial reversal learning and memory in the Morris water maze^[9]. Acetylcholine-induced db/db mice received intraperitoneal injection of 100mg/kg CDN1163 once daily for 5 days. CDN1163 improved aortic endothelium-dependent relaxation, reduced hepatic steatosis, decreased ALT and TC, increased early exercise oxygen consumption, and increased modified ventilatory threshold oxygen consumption^[10].

References:

- [1] Huang YP, Huang WW, Tsai KF, et al. CDN1163, a SERCA activator, causes intracellular Ca^{2+} leak, mitochondrial hyperpolarization and cell cycle arrest in mouse neuronal N2A cells. *Neurotoxicology*. 2023 Sep;98:9-15.
- [2] Hunter KD, Crozier RWE, Braun JL, et al. Acute activation of SERCA with CDN1163 attenuates

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IgE-mediated mast cell activation through selective impairment of ROS and p38 signaling. FASEB J. 2023 Feb;37(2):e22748.

[3] Zaripova KA, Belova SP, Sharlo KA, et al. SERCA activation prevents Ca^{2+} and ATP upregulation during 3-day soleus muscle unloading in rats. Am J Physiol Regul Integr Comp Physiol. 2025 Jul 1;329(1):R108-R122.

[4] Rahi V, Kaundal RK. A Small-Molecule Activator of Sarco/Endoplasmic Reticulum Ca^{2+} -ATPase Attenuates Cerebral Ischemia-Reperfusion Injury by Suppressing Endoplasmic Reticulum Stress and Apoptosis. ACS Pharmacol Transl Sci. 2025 Aug 19;8(9):2953-2963.

[5] Nguyen H, Noriega Polo C, Wiederkehr A, et al. CDN1163, an activator of sarco/endoplasmic reticulum Ca^{2+} ATPase, up-regulates mitochondrial functions and protects against lipotoxicity in pancreatic β -cells. Br J Pharmacol. 2023;180(21):2762-2776.

[6] Nogami K, Maruyama Y, Sakai-Takemura F, et al. Pharmacological activation of SERCA ameliorates dystrophic phenotypes in dystrophin-deficient mdx mice. Hum Mol Genet. 2021;30(11):1006-1019.

[7] Singh R, Moreno P, Hajjar R, et al. A role for calcium in resistin transcriptional activation in diabetic hearts. Sci Rep. 2018 Oct 23;8:15633.

[8] Kang S, Majid A, Hammoudi N, et al. Gene and Drug-Mediated SERCA2a Activation Restores Cardiac Function and Metabolic Balance in Diabetic Mice. Biomed Pharmacother. 2025 Nov;192:118617.

[9] Klocke B, Moore C, Ott H, et al. Chronic pharmacological activation of SERCA with CDN1163 affects spatial cognitive flexibility but not attention and impulsivity in mice. Behav Pharmacol. 2023 Dec;34(8):477-487.

[10] Kimura T, Kagami K, Sato A, et al. Sarco/Endoplasmic Reticulum Ca^{2+} ATPase 2 Activator Ameliorates Endothelial Dysfunction; Insulin Resistance in Diabetic Mice. Cells. 2022 Apr;11(9):1488.

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