

Product Data Sheet

Product Name: CORM 2
 Cat. No.: GC50347

Chemical Properties

Cas. No. 22594-69-0

SMILES [Cl-][Ru++]1([Cl-][Ru++]([Cl-])([Cl-]1)(C#[O])(C#[O])C#[O])(C#[O])(C#[O])C#[O])

Formula C₆Cl₄O₆Ru₂ M.Wt 512.01

Solubility DMSO : 30 mg/mL (58.59 mM; Need ultrasonic); H₂O : < 0.1 mg/mL (insoluble) Storage 4°C, away from moisture and light

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
$$\text{Ru}_2\text{Cl}_4(\text{CO})_6$$

Protocol

Cell experiment [1]:

Cell lines RAW264.7 murine macrophage cell line

Cells were incubated with the increasing dosages of CORM 2 (12.5-100μM) for Preparation Method 24h (measure NO) or 48h (measure IL-1β) in the presence of P. intermedia lipopolysaccharide (LPS) (10μg/ml).

Reaction Conditions 12.5, 25, 50, 100μM; 24h (measure NO) or 48h (measure IL-1β)

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

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Applications The secretion levels of NO and IL-1 β caused by P. intermedia LPS alone were obviously upregulated over those of the untreated control cells, and the administration of CORM 2 potently reduced the release of these proinflammatory mediators, and the suppressive influence was elevated with the increase of doses. CORM 2 reduced the synthesis of NO and IL-1 β by approximately 83% and 84%, respectively, at the concentration of 100 μ M.

Animal experiment [2]:

Animal models Male Sprague-Dawley rats

Preparation Method Cardiac arrest was induced by asphyxia. After 6min of untreated arrest, cardiopulmonary resuscitation (CPR) was initiated. Return of spontaneous circulation (ROSC) was defined as the restoration of a supraventricular rhythm with a mean arterial pressure (MAP) $\geq$ 60mmHg sustained for at least 5min. After successful resuscitation, animals were randomly assigned to three groups: (1) CPR group—cardiac arrest was induced and CPR performed; (2) CPR+CO group—cardiac arrest was induced and CPR performed, followed by treatment with CORM 2; (3) Control group—identical surgical procedures were carried out, but cardiac arrest was not induced and CPR was omitted. Thirty minutes after ROSC, rats in the CPR+CO group received a continuous intravenous infusion of CORM 2 at 10mg/kg/h for 30min (total dose 5mg/kg). Concurrently, rats in the control and CPR groups received an equal volume of placebo (0.9% NaCl solution containing DMSO) at an infusion rate of 4mL/kg/h.

Dosage form 5mg/kg; intravenous infusion

Applications The CORM 2 treatment results showed that CO increased Neurologic deficit scores (NDS) and decreased S-100B levels significantly at 24h after ROSC. CO treatment significantly increased the 3-day survival rates of the rats after ROSC.

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References:

- [1] Choi EY, Keum BR, Choe SH, et al. Tricarbonyldichlororuthenium(II) dimer, the lipid-soluble carbon monoxide-releasing molecule, attenuates *Prevotella intermedia* lipopolysaccharide-induced production of nitric oxide and interleukin-1 β in murine macrophages. *Int Immunopharmacol.* 2021;90:107190.
- [2] Wang P, Yao L, Zhou LL, et al. Carbon Monoxide Improves Neurologic Outcomes by Mitochondrial Biogenesis after Global Cerebral Ischemia Induced by Cardiac Arrest in Rats. *Int J Biol Sci.* 2016;12(8):1000-1009.

Background

CORM 2 is a carbon monoxide-releasing molecule that can liberate a controlled amount of CO in biological systems and has been developed as a carbon monoxide donor^[1-2]. When CORM 2 is in contact with reduced deoxymyoglobin or deoxyhaemoglobin rapidly transfers ca. one CO equivalent to the protein haem, forming carboxymyoglobin (COMb) or carboxyhaemoglobin (COHb), respectively^[3]. In addition, CORM 2 also exerts a protective effect against gastric mucosal injury induced by 75% ethanol^[4].

In vitro, after treating human umbilical vein endothelial cells (HUVECs) with 100 μ M CORM 2 for 0-5min, a bidirectional current can be recorded. This current is insensitive to the small-, intermediate-, and large-conductance Ca²⁺-activated K⁺ channel blockers apamin (the small-conductance Ca²⁺-activated K⁺ channel blocker), TRAM-34 (the intermediate-conductance Ca²⁺-activated K⁺ channel blocker), and iberiotoxin (the big-conductance Ca²⁺-activated K⁺ channel blocker), and it is not affected by EGTA in the pipette solution^[2]. RAW264.7 murine macrophage cell line was exposed to *P. intermedia* lipopolysaccharide (LPS) (10 μ g/mL) together with escalating concentrations of CORM 2 (12.5, 25, 50, 100 μ M) for 24h (NO) or 48h (IL-1 β), and the release of both NO and IL-1 β was markedly and dose-dependently suppressed^[5].

In vivo, intravenous infusion of CORM 2 at 10mg/kg/h for 30min (total 5mg/kg) after cardiac arrest significantly increased neurological deficit scores (NDS), reduced S-100B levels, and improved 3-day survival in resuscitated Sprague-Dawley rats^[6]. Administering CORM 2 (30mg/kg; i.p.) before a

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lethal dose of LPS significantly attenuated the severe hypothermia induced by LPS in C57BL/6 mice^[7].

References:

- [1] Khir NAM, Noh ASM, Long I, et al. Recent progress on anti-nociceptive effects of carbon monoxide releasing molecule-2 (CORM-2). *Mol Cell Biochem.* 2024;479(3):539-552.
- [2] Dong DL, Chen C, Huang W, et al. Tricarbonyldichlororuthenium (II) dimer (CORM2) activates non-selective cation current in human endothelial cells independently of carbon monoxide releasing. *Eur J Pharmacol.* 2008;590(1-3):99-104.
- [3] Fagone P, Mangano K, Coco M, et al. Therapeutic potential of carbon monoxide in multiple sclerosis. *Clin Exp Immunol.* 2012;167(2):179-187.
- [4] Magierowska K, Magierowski M, Hubalewska-Mazgaj M, et al. Carbon Monoxide (CO) Released from Tricarbonyldichlororuthenium (II) Dimer (CORM-2) in Gastroprotection against Experimental Ethanol-Induced Gastric Damage. *PLoS One.* 2015;10(10):e0140493.
- [5] Choi EY, Keum BR, Choe SH, et al. Tricarbonyldichlororuthenium(II) dimer, the lipid-soluble carbon monoxide-releasing molecule, attenuates Prevotella intermedia lipopolysaccharide-induced production of nitric oxide and interleukin-1 β in murine macrophages. *Int Immunopharmacol.* 2021;90:107190.
- [6] Wang P, Yao L, Zhou LL, et al. Carbon Monoxide Improves Neurologic Outcomes by Mitochondrial Biogenesis after Global Cerebral Ischemia Induced by Cardiac Arrest in Rats. *Int J Biol Sci.* 2016;12(8):1000-1009.
- [7] Riquelme SA, Bueno SM, Kalergis AM. Carbon monoxide down-modulates Toll-like receptor 4/MD2 expression on innate immune cells and reduces endotoxic shock susceptibility. *Immunology.* 2015;144(2):321-332

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