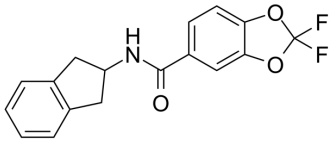
# AVE3085

| | |
|---|---|
| **Cat. No.:** | HY-19504 |
| **CAS No.:** | 450348-85-3 |
| **Molecular Formula:** | $C_{17}H_{13}F_2NO_3$ |
| **Molecular Weight:** | 317.29 |
| **Target:** | NO Synthase |
| **Pathway:** | Immunology/Inflammation |
| **Storage:** | Powder -20°C 3 years<br>4°C 2 years<br>In solvent -80°C 2 years<br>-20°C 1 year |



## SOLVENT & SOLUBILITY

**In Vitro**

DMSO : 250 mg/mL (787.92 mM; Need ultrasonic)

| Preparing Stock Solutions | Solvent / Mass / Concentration | 1 mg | 5 mg | 10 mg |
|---|---|---|---|---|
| | 1 mM | 3.1517 mL | 15.7585 mL | 31.5169 mL |
| | 5 mM | 0.6303 mL | 3.1517 mL | 6.3034 mL |
| | 10 mM | 0.3152 mL | 1.5758 mL | 3.1517 mL |

Please refer to the solubility information to select the appropriate solvent.

**In Vivo**

1. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (6.56 mM); Clear solution

## BIOLOGICAL ACTIVITY

**Description**

AVE3085 is a potent endothelial nitric oxide synthase enhancer, used for cardiovascular disease treatment.

**In Vitro**

Pre-incubation with AVE3085 restores the bradykinin-induced relaxation, reverses the decrease of p-eNOS$^{Ser1177}$, and loares the level of p-eNOS$^{Thr495}$ and nitrotyrosine. NO release in response to bradykinin is significantly reduced by ADMA and such reduction is restored by AVE3085. AVE3085 also prevents the elevation of $O_2^{\cdot -}$ and $ONOO^-$ levels in coronary arteries exposed to ADMA[2]. AVE3085 (10 μM) markedly increases ACh-induced relaxations in SHR aortae without affecting those in WKY aortae, and also increases the eNOS expression in WKY aortae[3].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

**In Vivo**

AVE3085 (10 mg/kg/day, p.o.) treatment prevents the increases in the left ventricular weight, left ventricular weight/body weight ratio, mean myocyte diameter, and the expression of the hypertrophic markers ANP and β-MHC compared to the vehicle-treated mice. AVE 3085 treatment also attenuates the collagen volume fraction levels compared to the vehicle-

treated mice. In the AVE3085-treated mice, the EFs, FSs, mitral E velocity, E/A ratio and LVDds are significantly improved compared to the vehicle-treated mice. AVE 3085 treatment attenuates the increase in the expression of Smad2 mRNA. Furthermore, the levels of the eNOS protein expression are significantly up-regulated in the AVE3085 group than in the vehicle-treated AB group[1]. AVE3085 (10 mg/kg, p.o.) significantly improves ACh-induced endothelium-dependent relaxations in the aortae of SHRs, and reduces systolic blood pressure in SHRs. AVE3085 treatment for 4 weeks increases levels of p-eNOS and eNOS in SHR aortae without affecting levels of eNOS and p-eNOS in WKY aortae[3].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

## PROTOCOL

**Animal Administration [1]**

Male C57BL/6J mice (8-10 weeks old; 24-26 g) are housed in individual cages on a 12 h light-dark cycle in a temperature- (24 ± 2°C) and humidity-controlled room with ad libitum access to tap water and standard rodent chow. The mice are anesthetized via an intraperitoneal injection of 1.5% pentobarbital (W*0.06), and cardiac hypertrophy is induced by pressure overload, which is achieved via descending aortic banding (AB). Similar surgeries that do not include aortic banding are performed on the sham-operated. Twenty-four hours after ligation, the surviving mice are randomly divided into the following three groups (n=8 per group): (1) a sham-operated group (Sham group), (2) a vehicle-treated AB group (vehicle-treated group), and (3) an AB group treated with AVE 3085 (AVE 3085 group). AVE 3085 is administered orally once daily at a dose of 10 mg kg day−1 for 4 weeks, and isovolumic sodium chloride is administered in the same manner to the sham-operated and vehicle-treated groups.

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

## REFERENCES


[1]. Chen Y, et al. AVE 3085, a novel endothelial nitric oxide synthase enhancer, attenuates cardiac remodeling in mice through the Smad signaling pathway. Arch Biochem Biophys. 2015 Mar 15;570:8-13.

[2]. Xue HM, et al. AVE3085 protects coronary endothelium from the impairment of asymmetric dimethylarginine by activation and recoupling of eNOS. Cardiovasc Drugs Ther. 2012 Oct;26(5):383-92.

[3]. Yang Q, et al. AVE3085, an enhancer of endothelial nitric oxide synthase, restores endothelial function and reduces blood pressure in spontaneously hypertensive rats. Br J Pharmacol. 2011 Jul;163(5):1078-85





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