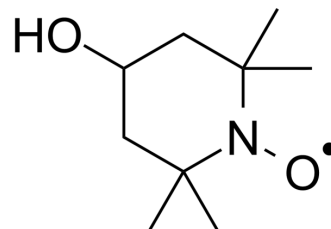


## Tempol

<b>Cat. No.:</b>	HY-100561		
<b>CAS No.:</b>	2226-96-2		
<b>Molecular Formula:</b>	C <sub>9</sub> H <sub>18</sub> NO <sub>2</sub>		
<b>Molecular Weight:</b>	172		
<b>Target:</b>	Autophagy; Reactive Oxygen Species		
<b>Pathway:</b>	Autophagy; Immunology/Inflammation; Metabolic Enzyme/Protease; NF-κB		
<b>Storage:</b>	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	1 year
		-20°C	6 months



### SOLVENT & SOLUBILITY

<b>In Vitro</b>	H <sub>2</sub> O : 5.56 mg/mL (32.33 mM; ultrasonic and warming and heat to 60°C)				
		Solvent Concentration	Mass 1 mg	5 mg	10 mg
	<b>Preparing Stock Solutions</b>	1 mM	5.8140 mL	29.0698 mL	58.1395 mL
		5 mM	1.1628 mL	5.8140 mL	11.6279 mL
10 mM		0.5814 mL	2.9070 mL	5.8140 mL	
Please refer to the solubility information to select the appropriate solvent.					
<b>In Vivo</b>	1. Add each solvent one by one: PBS Solubility: 25 mg/mL (145.35 mM); Clear solution; Need ultrasonic				

### BIOLOGICAL ACTIVITY

<b>Description</b>	Tempol is a general superoxide dismutase (SOD)-mimetic agent that efficiently neutralizes reactive oxygen species (ROS).
<b>IC<sub>50</sub> &amp; Target</b>	ROS <sup>[1]</sup>
<b>In Vitro</b>	Tempol significantly attenuates H <sub>2</sub> O <sub>2</sub> -mediated decrease in mitochondrial respiration and increase in LDH release from rat PT cells, indicating a reduction in cell injury and death. The beneficial actions of Tempol are similar to those obtained using the Fe <sup>2+</sup> chelator DEF. However, coadministration of DEF and Tempol does not produce any additional beneficial actions against renal ischemia/reperfusion injury or against oxidative stress-mediated PT cell injury/death <sup>[2]</sup> . MCE has not independently confirmed the accuracy of these methods. They are for reference only.
<b>In Vivo</b>	SOD-mimetic Tempol is able to mimic resveratrol's effects on heart function. Tempol is administered daily by gavage. Mice treated with Met or Tmp had decreased PR and QTc intervals and increased heart rates compared to peroral vehicle (VEH).

These results are similar to that obtained by treatment with RSV. Pre- and post-treatment profiles of individual mice are illustrated<sup>[1]</sup>. Tempol, a membrane-permeable radical scavenger, reduces oxidant stress-mediated renal dysfunction and injury in the rat. Tempol significantly reduces the increase in urea, creatinine,  $\gamma$ GT, AST, NAG, and  $FE_{Na}$  produced by renal ischemia/reperfusion, suggesting an improvement in both renal function and injury. Tempol also significantly reduces kidney MPO activity and MDA levels, indicating a reduction in PMN infiltration and lipid peroxidation, respectively. Tempol reduces the histologic evidence of renal damage associated with ischemia/reperfusion and caused a substantial reduction in the staining for nitrotyrosine and PARS, suggesting reduced nitrosative and oxidative stress<sup>[2]</sup>. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

## PROTOCOL

### Cell Assay <sup>[2]</sup>

To investigate the effect of Tempol, DEF, and DEF coadministered with Tempol on  $H_2O_2$ -mediated cell injury and death, confluent cultures of PT cells are preincubated (10 min at 37°C) with Tempol (0.03 to 10 mM), DEF (0.03 to 10 mM), or DEF (3 mM) in combination with Tempol (3 mM). The ranges of concentrations of Tempol and DEF are based on those previously shown in this laboratory to reduce on  $H_2O_2$ -mediated cell injury and death in (1) cultured rat cardiac myoblasts (Tempol) and (2) primary cultures of rat PT cells (DEF). PT cell cultures are then incubated with  $H_2O_2$  (1 mM) for four hours, after which cellular injury and death are assessed. Upon completion of incubations, cellular injury and death are assessed using the spectrophotometric assays described later in this article<sup>[2]</sup>.

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

### Animal Administration <sup>[1][2]</sup>

#### Mice<sup>[1]</sup>

Female or male BALB/c mice (5-7 weeks of age) are used. Mice are infected intraperitoneally (i.p.) with  $10^2$  blood trypomastigote forms of the type I Colombian strain of *T. cruzi*. Treatments are performed daily for 30 days from the establishment of CCC (60 dpi) by i.p. injection of 15 mg/kg trans-resveratrol (10% ethanol/PBS), vehicle (10% ethanol/PBS), 5 mg/kg EX527 (0.1% DMSO), or peroral administration of 40 mg/kg Resveratrol (10% ethanol-PBS), 500 mg/kg Metformin (dissolved in water), 100 mg/kg Tempol (dissolved in water), Benznidazole (25 mg/kg, dissolved in water) and vehicle (water or 10% ethanol-PBS).

#### Rats<sup>[2]</sup>

83 male Wistar rats weighing 230 to 320 g are used. Upon completion of surgical procedures, the animals are randomly allocated to the eight groups. At one minute before commencement of reperfusion, animals received a bolus injection of either vehicle (saline, 4 mL/kg, IV), Tempol (30 mg/kg in saline, IV), DEF (40 mg/kg in saline, IV), or DEF (40 mg/kg in saline, IV) in combination with Tempol (30 mg/kg in saline, IV). The corresponding groups then received a continuous infusion of one of the following throughout the reperfusion period: vehicle (saline, 4 mL/kg/h, IV), Tempol (30 mg/kg/h in saline, IV), DEF (40 mg/kg/h in saline, IV), or Tempol and DEF in combination (30 and 40 mg/kg/h, respectively, in saline, IV). To elucidate the effects of Tempol or DEF on cardiovascular hemodynamics and organ function in sham-operated rats, respective groups of animals received the treatments described previously in this article and as outlined. The concentration of Tempol administered in vivo is based on those previously demonstrated by us to provide significant protection against myocardial ischemia/reperfusion injury in an in vivo rat model. Similarly, the concentration of DEF used is identical to that which we have previously used to provide significant protection against hepatic ischemia/reperfusion injury in in vivo rat and rabbit models.

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

## CUSTOMER VALIDATION

- Redox Biol. October 2021, 102115.
- Cells. 2022 Apr 2;11(7):1196.
- J Phys Chem Lett. 2023 Aug 21;7638-7643.
- J Nutr Biochem. 2021 Oct 12;108883.

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- Front Pharmacol. 2020 Feb 21;11:123.

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## REFERENCES

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- [1]. Vilar-Pereira G, et al. Resveratrol Reverses Functional Chagas Heart Disease in Mice. PLoS Pathog. 2016 Oct 27;12(10):e1005947.
- [2]. Chatterjee PK, et al. Tempol, a membrane-permeable radical scavenger, reduces oxidant stress-mediated renal dysfunction and injury in the rat. Kidney Int. 2000 Aug;58(2):658-73.
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Tel: 609-228-6898

Fax: 609-228-5909

E-mail: [tech@MedChemExpress.com](mailto:tech@MedChemExpress.com)

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA