Product Data Sheet

Product Name: Dexmedetomidine
Cat. No.: GC17494

Chemical Properties

Cas. No.	113775-47-6		
Chemical Name	5-[(1S)-1-(2,3-dimethylphenyl)ethyl]-1H-imidazole		
SMILES	CC1=C(C(=CC=C1)C(C)C2=CN=CN2)C		
Formula	$C_{13}H_{16}N_2$	M.Wt	200.28
Solubility	≥ 9.95mg/mL in DMSO	Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 ℃ and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20℃ 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	Dexmedetomidine		

Protocol

Cell experiment [1]:

Cell lines	Cortical neurons
Preparation Method	Sprague-Dawley rat frontal cortices were isolated and cultured. Some cultures were treated with various concentrations of Dexmedetomidine (0.05μM, 0.1μM, 1μM, 2.5μM, 5μM, or 10μM) dissolved in culture media, whereas controls only had culture media. The cells were cultured for 3 or 7 days.

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Reaction Conditions	0.05μM, 0.1μM, 1μM, 2.5μM, 5μM, or 10μM; 3 or 7 days
Applications	On days 3 and 7 after Dexmedetomidine exposure, no significant effect on cell viability was observed at concentrations below 10μM. However, at 10μM, Dexmedetomidine markedly reduced cell viability, resulting in increased cell death: the percentage of viable cells on day 3 was significantly lower than that of controls. By day 7, none of the tested concentrations exerted any additional effect on viability.

Animal experiment [2]:

Animal models	C57BL/6 mice
Preparation Method	C57BL/6 mice (eight-week-old, male, 22-25g) were housed at an ambient temperature of 22±2°C under a fixed 12h light/dark cycle. Prior to experimentation, animals were randomly assigned into three groups (n=6 per group): Control (Con), acute liver injury (ALI) model, and ALI+Dexmedetomidine: 200mg/kg. The ALI model was induced by intraperitoneally injecting lipopolysaccharide (LPS)/D-galactose (D-Gal) (200μL; 30μg/kg; 600mg/kg) dissolved in PBS. Dexmedetomidine was administered intraperitoneally 1h prior to the LPS/D-Gal challenge. At 6h after LPS/D-Gal treatment, the mice were then anesthetized with isoflurane and subjected to enucleation for exsanguination. Subsequently, the liver tissues were harvested, fixed with formalin, and embedded in paraffin.
Dosage form	200mg/kg; administered intraperitoneally

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Applications	Dexmedetomidine treatment significantly suppressed the increases in ALT and AST levels, as well as the elevations of serum IL-6, IL-8, IL-1β, and TNF-α in the ALI model group.

References:
[1] Jimenez-Tellez N, Iqbal F, Pehar M, et al. Dexmedetomidine does not compromise neuronal viability, synaptic connectivity, learning and memory in a rodent model. *Sci Rep*. 2021;11(1):16153.
[2] Zhang C, Fan Y, Qin Z, et al. Network pharmacology and experimental validation reveal dexmedetomidine's protective mechanisms against acute liver injury in mice. *Sci Rep*. 2025;15(1):9044.

Background

Product Data Sheet

Dexmedetomidine is a highly selective α2-adrenergic receptor agonist that exerts sympatholytic effects in specific brain regions, providing analgesic, sedative, and anxiolytic actions[1-2]. In addition, Dexmedetomidine preconditioning effectively shields the heart against ischemia-reperfusion injury[3].

In vitro, after 3 days of treatment with graded Dexmedetomidine concentrations (0.05μM, 0.1μM, 1μM, 2.5μM, 5μM, or 10μM), cortical neurons showed unchanged viability below 10μM but a significant drop in viability and increased cell death at 10μM[4]. Exposure of PC12 cells to Dexmedetomidine at 50ng/ml, 200ng/ml, or 800ng/ml for 12 or 24h enhanced cell viability in both a time- and dose-dependent manner[5].

In vivo, in acute liver injury (ALI) C57BL/6 mice, a single intraperitoneal dose of Dexmedetomidine (200mg/kg) markedly blunted the rises in ALT and AST and suppressed the ALI-induced elevations of serum IL-6, IL-8, IL-1β, and TNF-α[6]. In male C57BL/6 mice, a single intraperitoneal dose of Dexmedetomidine (40μg/kg) suppresses lipopolysaccharide (LPS)-induced inflammatory factor expression and protects renal cells from apoptosis[7]. Administering Dexmedetomidine (10 or 20μg/kg; i.p.) to Sprague-Dawley rats dose-dependently reduces mortality and suppresses pulmonary inflammation by inhibiting the Toll-like receptor 4 (TLR4)/myeloid differentiation factor 88 (MyD88)/NF-κB pathway[8].

References:
[1] Hou M, Chen F, He Y, et al. Dexmedetomidine against intestinal ischemia/reperfusion injury: A systematic review and meta-analysis of preclinical studies. *Eur J Pharmacol*. 2023;959:176090.
[2] Dardalas I, Stamoula E, Rigopoulos P, et al. Dexmedetomidine effects in different experimental sepsis in vivo models. *Eur J Pharmacol*. 2019;856:172401.
[3] Takahashi K, Yoshikawa Y, Kanda M, et al. Dexmedetomidine as a cardioprotective drug: a narrative review. *J Anesth*. 2023;37(6):961-970.
[4] Jimenez-Tellez N, Iqbal F, Pehar M, et al. Dexmedetomidine does not compromise neuronal viability, synaptic connectivity, learning and memory in a rodent model. *Sci Rep*. 2021;11(1):16153.
[5] Guo Q, Ma M, Yu H, et al. Dexmedetomidine enables copper homeostasis in cerebral ischemia/reperfusion via ferredoxin 1. *Ann Med*. 2023;55(1):2209735.

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[6] Zhang C, Fan Y, Qin Z, et al. Network pharmacology and experimental validation reveal dexmedetomidine's protective mechanisms against acute liver injury in mice. *Sci Rep*. 2025;15(1):9044.
[7] Kang K, Gao Y, Wang SC, et al. Dexmedetomidine protects against lipopolysaccharide-induced sepsis-associated acute kidney injury via an α7 nAChR-dependent pathway. *Biomed Pharmacother*. 2018;106:210-216.
[8] Wu Y, Liu Y, Huang H, et al. Dexmedetomidine inhibits inflammatory reaction in lung tissues of septic rats by suppressing TLR4/NF-κB pathway. *Mediators Inflamm*. 2013;2013:562154.

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