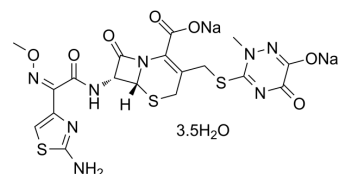


## Ceftriaxone sodium hydrate

<b>Cat. No.:</b>	HY-B0712A
<b>CAS No.:</b>	104376-79-6
<b>Molecular Formula:</b>	C <sub>18</sub> H <sub>23</sub> N <sub>8</sub> Na <sub>2</sub> O <sub>10.5</sub> S <sub>3</sub>
<b>Molecular Weight:</b>	661.6
<b>Target:</b>	Bacterial; Antibiotic; GSK-3; Aurora Kinase
<b>Pathway:</b>	Anti-infection; PI3K/Akt/mTOR; Stem Cell/Wnt; Cell Cycle/DNA Damage; Epigenetics
<b>Storage:</b>	4°C, stored under nitrogen, away from moisture * In solvent : -80°C, 6 months; -20°C, 1 month (stored under nitrogen, away from moisture)



### BIOLOGICAL ACTIVITY

<b>Description</b>	Ceftriaxone sodium hydrate (Ro 13-9904 sodium hydrate) is a broad spectrum $\beta$ -lactam third-generation cephalosporin antibiotic, which has good antibacterial activity against a variety of gram-negative and positive bacteria. Ceftriaxone sodium hydrate is a covalent inhibitor of GSK3 $\beta$ with IC <sub>50</sub> value of 0.78 $\mu$ M. Ceftriaxone sodium hydrate is an inhibitor of Aurora B. Ceftriaxone sodium hydrate has anti-inflammatory, antitumor and antioxidant activities. Ceftriaxone sodium hydrate can be used in the study of bacterial infections and meningitis <sup>[1][2][3][4][5][6][7]</sup> .																
<b>IC<sub>50</sub> &amp; Target</b>	$\beta$ -lactam																
<b>In Vitro</b>	<p>Ceftriaxone sodium hydrate (100 <math>\mu</math>M, 24 h) protects MPP<sup>+</sup> treated astrocytes by inhibiting the NF-<math>\kappa</math>B/JNK/c-Jun signaling pathway [3].</p> <p>Ceftriaxone sodium hydrate (500 <math>\mu</math>M, 24-48 h) effectively inhibits unanchored cell growth in A549, H520 and H1650 lung cancer cells by inhibiting Aurora B<sup>[4]</sup>.</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p> <p>Cell Viability Assay<sup>[3]</sup></p> <table border="1"> <tr> <td>Cell Line:</td> <td>Astrocyte</td> </tr> <tr> <td>Concentration:</td> <td>100 <math>\mu</math>M</td> </tr> <tr> <td>Incubation Time:</td> <td>24 h</td> </tr> <tr> <td>Result:</td> <td>Improved cell viability and increased glutamate uptake after MPP<sup>+</sup> expose.</td> </tr> </table> <p>Western Blot Analysis<sup>[3]</sup></p> <table border="1"> <tr> <td>Cell Line:</td> <td>Astrocyte</td> </tr> <tr> <td>Concentration:</td> <td>100 <math>\mu</math>M</td> </tr> <tr> <td>Incubation Time:</td> <td>24 h</td> </tr> <tr> <td>Result:</td> <td>Enhanced GLT-1 and GFAP expression. Decreased the expression of p-p50<math>\beta</math>p-IKK<math>\alpha</math><math>\beta</math>p-Relb. Decreased the number of TUNEL-positive cells.</td> </tr> </table>	Cell Line:	Astrocyte	Concentration:	100 $\mu$ M	Incubation Time:	24 h	Result:	Improved cell viability and increased glutamate uptake after MPP <sup>+</sup> expose.	Cell Line:	Astrocyte	Concentration:	100 $\mu$ M	Incubation Time:	24 h	Result:	Enhanced GLT-1 and GFAP expression. Decreased the expression of p-p50 $\beta$ p-IKK $\alpha$ $\beta$ p-Relb. Decreased the number of TUNEL-positive cells.
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## In Vivo

Ceftriaxone sodium hydrate (200 mg/kg Intraperitoneal injection for 6 weeks) improves functional markers and oxidative stress and inflammation parameters in a rat model of D-galactose (DGL) -induced liver and kidney injury<sup>[5]</sup>.

Ceftriaxone sodium hydrate (200, 400 mg/kg, Intraperitoneal injection) has a protective effect on convulsion induced by Pentylentetrazol (PTZ) and PTZ-related oxidative damage in rats<sup>[6]</sup>.

Ceftriaxone sodium hydrate (100, 200 mg/kg, Intraperitoneal injection) reduces mechanical dysodynia and hyperalgesia by activating GLT-1 in Streptozocin (HY-13753)-induced diabetic rat models<sup>[7]</sup>.

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

Animal Model:	DGL-induced rat model <sup>[5]</sup>
Dosage:	200 mg/kg
Administration:	i.p.
Result:	Reduced the BUN, Cr, AST and ALT levels. Attenuated the MDA levels and enhanced GPx and CAT activities. Reduced the levels of IL-1 $\beta$ and TNF- $\alpha$ mRNA.
Animal Model:	PTZ-induced rat model <sup>[6]</sup>
Dosage:	200, 400 mg/kg
Administration:	i.p. 60 min before to PTZ (70 mg/kg)
Result:	Both of the two ceftriaxone groups had lower spike percentages than the saline group. Significantly lower MDA levels and higher SOD activity in 200 and 400 mg/kg.

## CUSTOMER VALIDATION

- Nat Commun. 2022 Mar 2;13(1):1116.
- EBioMedicine. 2022 Apr;78:103943.
- Chemosphere. 2023 Oct 3;344:140353.

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## REFERENCES

- [1]. Nahata MC, et al. Ceftriaxone: a third-generation cephalosporin. Drug Intell Clin Pharm. 1985 Dec;19(12):900-6.
- [2]. Nassar H, et al. Molecular docking, molecular dynamics simulations and in vitro screening reveal cefixime and ceftriaxone as GSK3 $\beta$  covalent inhibitors. RSC Adv. 2023 Apr 11;13(17):11278-11290.
- [3]. Zhang Y, et al. Ceftriaxone Protects Astrocytes from MPP(+) via Suppression of NF- $\kappa$ B/JNK/c-Jun Signaling. Mol Neurobiol. 2015 Aug;52(1):78-92.
- [4]. Li X, et al. Ceftriaxone, an FDA-approved cephalosporin antibiotic, suppresses lung cancer growth by targeting Aurora B. Carcinogenesis. 2012 Dec;33(12):2548-57.
- [5]. Hakimzadeh E, et al. Ceftriaxone improves hepatorenal damages in mice subjected to D-galactose-induced aging. Life Sci. 2020 Oct 1;258:118119.
- [6]. Uyanikgil Y, et al. Positive effects of ceftriaxone on pentylentetrazol-induced convulsion model in rats. Int J Neurosci. 2016;126(1):70-5.
- [7]. Gunduz O, et al. Anti-allodynic and anti-hyperalgesic effects of ceftriaxone in streptozocin-induced diabetic rats. Neurosci Lett. 2011 Mar 10;491(1):23-5.

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

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