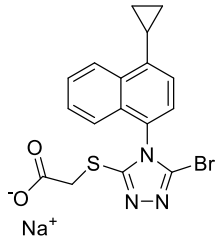
Product data sheet

MedKoo Cat#: 510301 Name: Lesinurad sodium CAS#: 1151516-14-1 (sodium) Chemical Formula: C17H13BrN3NaO2S Exact Mass: 402.99901 Molecular Weight: 426.2638	
Product supplied as:	Powder
Purity (by HPLC):	≥ 98%
Shipping conditions	Ambient temperature
Storage conditions:	Powder: -20°C 3 years; 4°C 2 years.
	In solvent: -80°C 3 months; -20°C 2 weeks.

1. Product description:

Lesinurad, also known as RDEA594, is a selective uric acid re-absorption inhibitor (SURI) and is also a selective inhibitor of URAT1, a transporter in the kidney that regulates uric acid excretion from the body. RDEA594 has been shown to normalize the amount of uric acid excreted by gout patients previously classified as under-excretors. Lesinurad received FDA approval on December 22, 2015.

2. CoA, QC data, SDS, and handling instruction

SDS and handling instruction, CoA with copies of QC data (NMR, HPLC and MS analytical spectra) can be downloaded from the product web page under "QC And Documents" section. Note: copies of analytical spectra may not be available if the product is being supplied by MedKoo partners. Whether the product was made by MedKoo or provided by its partners, the quality is 100% guaranteed.

3. Solubility data

Solvent	Max Conc. mg/mL	Max Conc. mM
DMSO	55.5	137.97
Ethanol	85.0	199.41
Water	8.0	18.77

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	2.35 mL	11.73 mL	23.46 mL
5 mM	0.47 mL	2.35 mL	4.69 mL
10 mM	0.23 mL	1.17 mL	2.35 mL
50 mM	0.05 mL	0.23 mL	0.47 mL

5. Molarity Calculator, Reconstitution Calculator, Dilution Calculator

Please refer the product web page under section of "Calculator"

6. Recommended literature which reported protocols for in vitro and in vivo study

In vitro study

1. Heitel P, Gellrich L, Heering J, Goebel T, Kahnt A, Proschak E, Schubert-Zsilavecz M, Merk D. Urate transporter inhibitor lesinurad is a selective peroxisome proliferator-activated receptor gamma modulator (sPPARγM) in vitro. Sci Rep. 2018 Sep 10;8(1):13554. doi: 10.1038/s41598-018-31833-4. PMID: 30202096; PMCID: PMC6131501.

In vivo study

1. Alghamdi YS, Soliman MM, Nassan MA. Impact of Lesinurad and allopurinol on experimental Hyperuricemia in mice: biochemical, molecular and Immunohistochemical study. BMC Pharmacol Toxicol. 2020 Feb 10;21(1):10. doi: 10.1186/s40360-020-0386-7. PMID: 32041665; PMCID: PMC7011467.

7. Bioactivity

Biological target:

Lesinurad sodium -is a URAT1 and OAT inhibitor, is determined to be a substrate for the kidney transporters OAT1 and OAT3 with Km values of 0.85 and 2 μM, respectively.

Product data sheet

In vitro activity

Cells treated with lesinurad were indistinguishable from DMSO-treated cells and even undifferentiated controls (Fig. 5A). Of note, lesinurad exhibited no cytotoxicity up to 50 μM (data not shown) excluding that the lack of lipid accumulation is a toxic effect. To gain deeper understanding of lesinurad-mediated PPARγ modulation in adipocytes, this study analyzed their gene expression profiles after differentiation (Fig. 5B). Compared to DMSO-treated cells, the full PPARγ agonist rosiglitazone robustly induced the scavenger receptor CD36, adiponectin, fatty acid binding protein 4 (FABP4) and the glucose transporter 4 (GLUT4). In strong contrast, lesinurad at 30 μM caused almost no changes in PPARγ-regulated gene expression with only slight trends for CD36 and adiponectin induction. Thus, the gene expression profiles confirmed the results of the staining experiments and indicated that lesinurad does not activate pro-adipogenic PPARγ target gene transcription in adipocytes to cause lipid accumulation. In contrast, in human hepatoma cells (HepG2 cells), lesinurad caused a more distinguished effect on PPARγ-regulated genes (Fig. 5C). As in adipocytes, lesinurad hardly affected CD36 and adiponectin expression but markedly induced angiopoietin-like 4 (ANGPTL4).

Reference: Sci Rep. 2018; 8: 13554. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6131501/

In vivo activity

Hyperuricemic group showed an increase in serum levels of GPT, GOT, uric acid and BUN. HU group received either ALP or ZUR (Lesinurad) showed a decrease in GPT, GOT, uric acid and BUN levels (Fig. 1a-b). Co-administration of ALP and ZUR revealed an ameliorative and additive synergistic effect ($P < 0.05$) on the normalization of GPT, GOT, uric acid and BUN levels (Fig. 1a). It should be noted that ZUR revealed same effect induced by ALP in hyperuricemic administered mice.

Reference: BMC Pharmacol Toxicol. 2020; 21: 10. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7011467/