

Product Data Sheet

Product Name: Gln-AMS

Cat. No.: GC30683

Chemical Properties

Cas. No. 209543-57-7

SMILES O[C@@H]([C@H]([C@H](N1C=NC2=C1N=CN=C2N)O3O)[C@H]3COS(=O)(NC([C@@H](N)CCC(N)=O)=O)=O

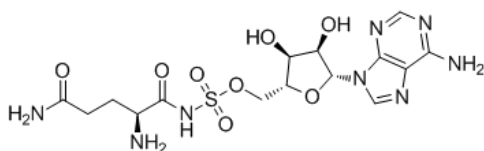
Formula $C_{15}H_{22}N_8O_8S$ M.Wt 474.45

Solubility DMSO : 120 mg/mL (252.92 mM; Need ultrasonic) Storage Store at -20°C

General tips For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C 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



Protocol

Cell experiment [1]:

Cell lines Primary hippocampal neuron cells from C57BL/6 neonatal mice

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

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Preparation Method	Primary hippocampal neurons neuron cells were grown in DMEM medium with 10% (v/v) fetal bovine serum (FBS), 100µg/ml streptomycin, and 100U/ml penicillin at 37°C in 5% CO ₂ /atmosphere. Cells were treated with 2mM lactate for 12h in the presence or absence of Gln-AMS (10µM). Cell viability was evaluated. Use Western blot to analyze the protein expression levels in the cytoplasmic lysate.
Reaction Conditions	10µM; 12h
Applications	Gln-AMS treatment significantly suppressed lactate-induced K _{la} accumulation in primary hippocampal neurons neuron cells.

References:

[1] Luo S, Lyu Z, Huang H, et al. Targeting AARS1-dependent lactylation improves neuronal process plasticity and mitigates cognitive deficits in sepsis-associated encephalopathy[J]. Brain, Behavior, and Immunity, 2026: 106493.

Background

Gln-AMS is an aminoacyl-tRNA synthetase (AARS) inhibitor, with the K_i value of 1.32µM [1]. Gln-AMS inhibits ferroptosis in hyperglycemic cells by simultaneously suppressing the expression of AARS1 and H3K18_{la} [2]. Gln-AMS is widely used as a potent and synergistic immunosuppressant to regulate the allogeneic mixed lymphocyte reaction [3].

In vitro, Gln-AMS (10µM) treatment for 12 hours significantly inhibited the accumulation of K_{la} in lactate-induced mouse primary hippocampal neurons neuron cells and the lactylation of ATR-interacting protein (ATRIP), as well as the ATR/CHK1 signaling pathway [4].

References:

- [1] Rath V L, Silvan L F, Beijer B, et al. How glutaminyI-tRNA synthetase selects glutamine[J]. Structure, 1998, 6(4): 439-449.
- [2] Hong J, Xu H, Yu L, et al. AARS1-mediated lactylation of H3K18 and STAT1 promotes ferroptosis

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in diabetic nephropathy[J]. Cell Death & Differentiation, 2026, 33(3): 589-604.

[3] Van de Vijver P, Ostrowski T, Sproat B, et al. Aminoacyl-tRNA synthetase inhibitors as potent and synergistic immunosuppressants[J]. Journal of medicinal chemistry, 2008, 51(10): 3020-3029.

[4] Luo S, Lyu Z, Huang H, et al. Targeting AARS1-dependent lactylation improves neuronal process plasticity and mitigates cognitive deficits in sepsis-associated encephalopathy[J]. Brain, Behavior, and Immunity, 2026: 106493.

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