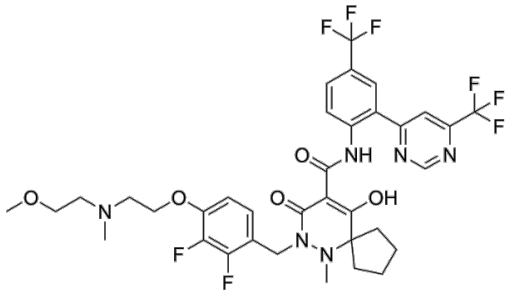
Product Data Sheet

Product Name: Atidafonat
Cat. No.: GC81587

Chemical Properties

Cas. No.	1628848-73-6		
Formula	$C_{35}H_{36}F_8N_6O_5$	M.Wt	772.7
Solubility	DMSO: 100 mg/mL (129.42 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			



Background

Atidafonat is an orally active sodium-dependent phosphate transporter inhibitor, with IC_{50} values of 6.8, 1.5, and 1.7 μM against human NaPi-IIb, PiT-1, PiT-2, respectively; and IC_{50} values of 3.9, 1.9, and 1.7 μM against rat NaPi-IIb, PiT-1, PiT-2, respectively. Atidafonat inhibits intestinal phosphate

absorption, increases fecal phosphate excretion, reduces urinary phosphate excretion, and decreases the levels of serum phosphate, FGF23, and adult parathyroid hormone. Atidafonat ameliorates ectopic thoracic aortic calcification, renal injury and hyperphosphatemia, and inhibits the expression of fibrosis markers. Atidafonat can be used for the research of hyperphosphatemia and chronic kidney disease-mineral and bone disorder (CKD-MBD) [1] [2].

In Vivo, Atidafonat (0.050-0.50%; p.o.; daily; 3 days) dose-dependently increases fecal phosphorus excretion, decreases urinary phosphorus excretion, and reduces serum phosphorus levels in normal male Wistar rats, confirming inhibition of intestinal phosphate absorption[1]. Atidafonat (0.015-1.0%; p.o.; daily; 14 days) dose-dependently reduces serum phosphate, FGF-23, and intact PTH levels, increases fecal phosphorus excretion, and decreases urinary phosphorus excretion in adenine-induced hyperphosphatemic male Wistar rats, with greater potency than a NaPi-IIb-selective inhibitor at equivalent doses[1]. Atidafonat (0.3%; p.o.; ad libitum; day 21 to day 116) sustainably suppresses serum phosphate, FGF-23, and intact PTH levels, ameliorates aortic calcification, reduces serum creatinine, and improves kidney histopathology and fibrotic gene expression in chronic anti-Thy1.1 nephritis male Fisher rats[1]. Atidafonat (0.3%; food admixture; ad libitum; 5 weeks) significantly ameliorates hyperphosphatemia, improves kidney function, and reduces glomerular crescent formation in anti-GBM-induced glomerulonephritis rats, with a significant reduction in serum creatinine to 1.31 mg/dL and glomerular crescent formation to 66.1% of total glomeruli[2].

In Vitro, Atidafonat (various concentrations) inhibits phosphate uptake in normal rat intestinal brush border membrane vesicles with an IC_{50} of 3.1 μmol/l[1]. Atidafonat (0.3-3.0 μM; 24 h) dose-dependently suppresses high phosphate-induced upregulation of Collagen 1A1, Collagen 3A1, and α-smooth muscle actin mRNA in hMes 130hT-9 human mesangial cells[2].

References:
[1]. Tsuboi Y, et al. EOS789, a novel pan-phosphate transporter inhibitor, is effective for the treatment of chronic kidney disease-mineral bone disorder. Kidney Int. 2020;98(2):343-354.
[2]. Tsuboi Y, et al. EOS789, pan-phosphate transporter inhibitor, ameliorates the progression of kidney injury in anti-GBM-induced glomerulonephritis rats. Pharmacol Res Perspect. 2022;10(3):e00973.