Product Data Sheet

Product Name: BAY-3827
Cat. No.: GC67772

Chemical Properties

Cas. No.	2377576-35-5		
Formula	$C_{27}H_{25}FN_6O$	M.Wt	468.53
Solubility	DMSO : 25 mg/mL (53.36 mM; ultrasonic and warming and heat to 60°C)	Storage	4°C, protect from light
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	BAY-3827		

Protocol

Cell experiment [1]:

Cell lines	LNCaP□VCaP cells
Preparation Method	LNCaP and VCaP cells were treated with 1nM R1881, enzalutamide or BAY-3827, as indicated for 24 or 48h. RNA was extracted using the RNeasy Plus Mini kit. Synthesis of cDNA was performed with the SuperScript® III First Strand Synthesis SuperMix for qRT-PCR. Analysis was performed using the RT^2 Proler™ PCR Array Human AMPK Signaling 330231.
Reaction Conditions	1nM; 24, 48h

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Applications	BAY-3827 regulates the expression of several genes involved in lipid metabolism such as LIPE, PRKAR2B, AKT3 and CPT1 family members.

Animal experiment [2]:

Animal models	C57BL/6J mice
Preparation Method	24 mice were divided randomly into the control group, the sh-NC group, the sh-NTRK1 group, and the sh-NTRK1+BAY-3827 group (n=6 in each group). The mice in the control group, the sh-NC group, and the shNTRK1 group were treated as described in the “Mouse transfection through brain stereotactic injection” section. BAY-3827, a selective inhibitor of AMPK, dispersed in PBS to prepare a concentration of 100μM. Next, the mice in the sh-NTRK1+BAY-3827 group were administered a brain stereotactic injection of BAY-3827 (5μL per mouse). After 1h, the mice were injected with the lentivirus carrying shRNA-NTRK1. All mice were then maintained for two weeks.
Dosage form	5μL, 100μM; brain stereotactic injection
Applications	The treatment with BAY-3827 exacerbated the mouse depressive-like behavior induced by NTRK1 knockdown.

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References:
[1] Lemos C, Schulze V K, Baumgart S J, et al. The Potent and Selective AMPK Inhibitor BAY-3827 Shows Strong Efficacy in Androgen-Dependent Prostate Cancer Models[J]. 2020.
[2]Yang K, Wu J, Li S, et al. NTRK1 knockdown induces mouse cognitive impairment and hippocampal neuronal damage through mitophagy suppression via inactivating the AMPK/ULK1/FUNDC1 pathway[J]. Cell Death Discovery, 2023, 9(1): 404.

Background

BAY-3827 is a potent and selective AMP-activated protein kinase (AMPK) inhibitor with IC_{50} values of 1.4nM at low ATP concentration (10μM) and 15nM at high ATP concentration (2mM)[1]. AMPK is a global sensor of cellular energy levels and a key regulator of nutrient metabolism[2]. BAY-3827 inhibits the phosphorylation of acetyl-CoA carboxylase (ACC)[3].

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In vitro, treatment of human prostate cancer cell lines (LNCaP, VCaP cells) with BAY-3827 (1nM) for 24h or 48h downregulated the expression of multiple genes involved in lipid metabolism, such as LIPE, PRKAR2B, AKT3, and CPT1 family members[4]. Pretreatment of cardiac fibroblasts (HCFs) with BAY-3827 (500nM) for 1h effectively eliminated the phosphorylation of intracellular acetyl-CoA carboxylase (ACC) induced by SGLT2i[5]. Treatment of corneal epithelial cells infected with Pseudomonas aeruginosa (PA) with BAY-3827 (10μM) significantly reduced the levels of inflammatory markers IL-6 and IL-8 in the cells[6].

In vivo, stereotactic injection of BAY-3827 (5μL, 100μM) into neurotrophic tyrosine kinase receptor 1 (NTRK1) knockdown mice exacerbated the impairment of working memory and reference memory, and exacerbated depressive-like behavior[7].

References:
[1] Lemos C, Schulze V K, Baumgart S J, et al. The potent AMPK inhibitor BAY-3827 shows strong efficacy in androgen-dependent prostate cancer models[J]. Cellular Oncology, 2021, 44(3): 581-594.
[2] He L, Zhou X, Huang N, et al. AMPK regulation of glucose, lipid and protein metabolism: mechanisms and nutritional significance[J]. Current Protein and Peptide Science, 2017, 18(6): 562-570.
[3] Strang J E, Astridge D D, Nguyen V T, et al. Small molecule modulators of AMP-activated protein kinase (AMPK) activity and their potential in cancer therapy[J]. Journal of Medicinal Chemistry, 2025, 68(3): 2238-2254.
[4] Lemos C, Schulze V K, Baumgart S J, et al. The Potent and Selective AMPK Inhibitor BAY-3827 Shows Strong Efficacy in Androgen-Dependent Prostate Cancer Models[J]. 2020.
[5] Baufays C, Cumps J, Dufeys C, et al. Comparison of the Effects of Sodium-Glucose Cotransporter 2 Inhibitors on Cardiac Fibroblast Properties[J]. International Journal of Molecular Sciences, 2025, 26(20): 10098.
[6] Cao D W, Ramachandran R A, Robertson D M. AMPK modulates mitochondrial homeostasis during Pseudomonas aeruginosa infection in corneal epithelial cells[J]. Investigative Ophthalmology & Visual Science, 2024, 65(7): 1971-1971.
[7] Yang K, Wu J, Li S, et al. NTRK1 knockdown induces mouse cognitive impairment and hippocampal neuronal damage through mitophagy suppression via inactivating the AMPK/ULK1/FUNDC1 pathway[J]. Cell Death Discovery, 2023, 9(1): 404.

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