

Product Data Sheet

Product Name: STA-013

Cat. No.: GC79464

Chemical Properties

Cas. No.

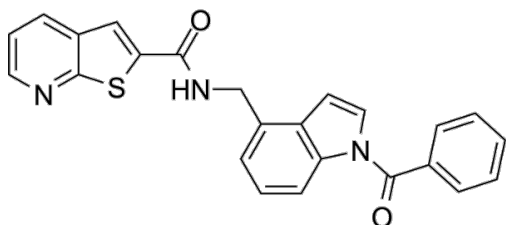
Formula $C_{24}H_{17}N_3O_2S$ M.Wt 411.48

Solubility DMSO: 25 mg/mL (60.76 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

STA-013 is a EphB tyrosine kinase inhibitor. STA-013 shows promising potency against EphB1 ($IC_{50} = 0.69 \mu M$), EphB2 ($IC_{50} = 1.73 \mu M$), and EphB4 ($IC_{50} = 1.02 \mu M$) tyrosine kinases. STA-013 results in inhibition of the EphB phosphorylated signal, coupled with increased p-AKT / AKT signaling, to suggest insulin signaling activation. STA-013 inhibits EphB tyrosine kinase by enhancing insulin receptor beta (IR β) signaling, and decreasing TGF- β levels in the heart. STA-013 can be used for the study of type 2 diabetes and cardiac complications (diabetic cardiomyopathy) [1].

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

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In Vivo, STA-013 (12.5, 25 and 50 mg/kg, i.p., daily for 6 weeks) promotes weight loss, improves glucose clearance and insulin sensitivity, and enhances insulin signaling in isolated brown adipose tissue and liver in high-fat diet-induced C57BL/6J mice. In these obese mice^[1]. STA-013 (12.5, 25 and 50 mg/kg, i.p., daily for 6 weeks) induces weight loss not through reduced food intake or elevated metabolic rates in DIO mice^[1].

In Vitro, STA-013 (5 μ M) inhibits EphB1 (54%), EphB2 (42%), EphB4 (52%), SGK1 (47%), and PKB β (43%) without affecting the rest of 140 kinases via kinome profiling assay^[1]. STA-013 (0.032-20 μ M, 96 h) significantly inhibits lipid accumulation in a dose-dependent manner in the embryonic mouse 3T3-L1 cells^[1]. STA-013 (1 μ M, 30 min) results a significant reduction in basal respiration, proton leak, ATP production, and non-mitochondrial oxygen consumption, increases maximal respiration and spare respiratory capacity, exhibits a significant increase in glycolysis and glycolytic capacity and reduces palmitate oxidation in 3T3-L1 cells^[1].

References:

[1]. Tareq S, et al. Discovery of pan-EphB tyrosine kinase inhibitor for metabolic syndrome sparing EphB3 signaling in mice. Pharmacol Res. 2025 Sep;219:107900.

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