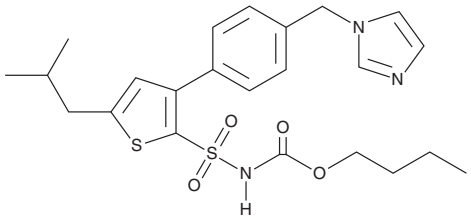
PRODUCT INFORMATION

AT_2 Agonist C21

Item No. 33758

CAS Registry No.: 477775-14-7
Formal Name: N-[[3-[4-(1H-imidazol-1-ylmethyl) phenyl]-5-(2-methylpropyl)-2-thienyl] sulfonyl]-carbamic acid, butyl ester
Synonym: Compound 21
MF: $C_{23}H_{29}N_3O_4S_2$
FW: 475.6
Purity: ≥98%
Supplied as: A solid
Storage: -20°C
Stability: ≥4 years



Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Laboratory Procedures

AT_2 agonist C21 is supplied as a solid. A stock solution may be made by dissolving the AT_2 agonist C21 in the solvent of choice, which should be purged with an inert gas. AT_2 agonist C21 is soluble in DMSO.

Description

AT_2 agonist C21 is an agonist of the angiotensin II type 2 (AT_2) receptor.[1] It selectively binds to the AT_2 receptor over the AT_1 receptor (K_is = 0.4 and >10 µM, respectively). AT_2 agonist C21 (0.1 µM) induces neurite outgrowth in NG 108-15 cells. It reduces mean arterial blood pressure in anaesthetized spontaneously hypertensive rats (SHRs) when administered at a dose of 0.05 mg/kg. AT_2 agonist C21 (0.03 mg/kg) reduces right ventricle hypertrophy and fibrosis, as well as lung interstitial and perivascular fibrosis, in a rat model of pulmonary hypertension induced by monocrotaline (MCT; Item No. 16666).[2] It increases survival and reduces neurological deficits in a mouse model of cerebral ischemia induced by middle cerebral artery occlusion (MCAO) when administered at the same dose.[3]

References

1. Wan, Y., Wallinder, C., Plouffe, B., *et al.* Design, synthesis, and biological evaluation of the first selective nonpeptide AT_2 receptor agonist. *J. Med. Chem.* **47(24)**, 5995-6008 (2004).
2. Bruce, E., Shenoy, V., Rathinasabapathy, A., *et al.* Selective activation of angiotensin AT_2 receptors attenuates progression of pulmonary hypertension and inhibits cardiopulmonary fibrosis. *Br. J. Pharmacol.* **172(9)**, 2219-2231 (2004).
3. Schwengel, K., Namsolleck, P., Lucht, K., *et al.* Angiotensin AT_2-receptor stimulation improves survival and neurological outcome after experimental stroke in mice. *J. Mol. Med. (Berl.)* **94(8)**, 957-966 (2016).

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