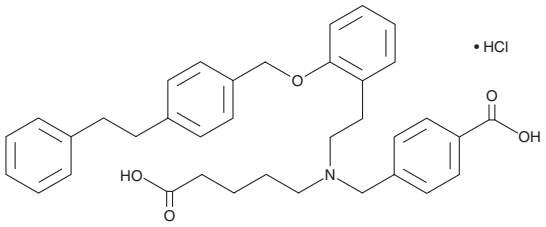
PRODUCT INFORMATION

Cinaciguat (hydrochloride)

Item No. 17468

CAS Registry No.: 646995-35-9
Formal Name: 4-[[(4-carboxybutyl)[2-[2-[[4-(2-phenylethyl)phenyl]methoxy]phenyl]ethyl]amino]methyl]-benzoic acid, monohydrochloride
Synonym: BAY 58-2667
MF: $C_{36}H_{39}NO_5 \bullet HCl$
FW: 602.20
Purity: ≥98%
Stability: ≥2 years at -20°C
Supplied as: A crystalline solid
UV/Vis.: λ_{max}: 223 nm



Laboratory Procedures

For long term storage, we suggest that cinaciguat (hydrochloride) be stored as supplied at -20°C. It should be stable for at least two years.

Cinaciguat (hydrochloride) is supplied as a crystalline solid. A stock solution may be made by dissolving the cinaciguat (hydrochloride) in the solvent of choice. Cinaciguat (hydrochloride) is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF), which should be purged with an inert gas. The solubility of cinaciguat (hydrochloride) in ethanol is approximately 5 mg/ml and approximately 30 mg/ml in DMSO and DMF.

Cinaciguat (hydrochloride) is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, cinaciguat (hydrochloride) should first be dissolved in DMSO and then diluted with the aqueous buffer of choice. Cinaciguat (hydrochloride) has a solubility of approximately 0.5 mg/ml in a 1:1 solution of DMSO:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

Soluble guanylate cyclase (sGC) is the primary cellular receptor for NO. NO binds and activates a heme group in sGC, initiating the conversion of GTP to the second messenger cGMP. cGMP subsequently mediates a number of signaling cascades leading to vasorelaxation and inhibiting smooth muscle proliferation, leukocyte recruitment, and platelet aggregation. Oxidation of the heme results in its dissociation from sGC and an impairment of NO signaling, which has been linked to hypertension, hyperlidemia, cardiovascular disease, and diabetes.[1] Cinaciguat is an activator of sGC that binds to a regulatory site, resulting in activation in an NO-independent manner (K_d = 3.2 nM).[2] It activates the enzyme even if it has been oxidized or rendered heme deficient.[2] In animals, cinaciguat has been shown to reduce hypertension, limit cardiomyocyte hypertrophy, protect against ischemia/reperfusion injury, and reduce morbidity and mortality in response to endotoxic shock.[2-5]

References

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2. Stasch, J.-P., Schmidt, P., Alonso-Alija, C., *et al. Brit. J. Pharmacol.* **136**, 773-783 (2002).
3. Irvine, J.C., Ganthavee, V., Love, J.E., *et al. PLoS One* **7(11)**, 1-11 (2012).
4. Salloum, F.N., Das, A., Samidurai, A., *et al. Am. J. Physiol. Heart Circ. Physiol.* **302**, H1347-H1354 (2012).
5. Vandendriessche, B., Rogge, E., Goossens, V., *et al. PLoS One* **8(9)**, 1-10 (2013).

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