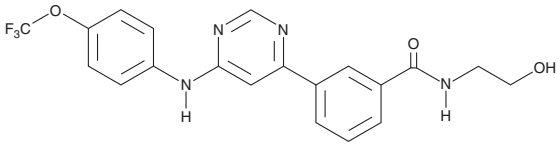
PRODUCT INFORMATION

GNF-5

Item No. 16254

CAS Registry No.:	778277-15-9
Formal Name:	N-(2-hydroxyethyl)-3-[6-[[4-(trifluoromethoxy)phenyl]amino]-4-pyrimidinyl]-benzamide
MF:	$C_{20}H_{17}F_3N_4O_3$
FW:	418.4
Purity:	≥95%
UV/Vis.:	λ_{max}: 268 nm
Supplied as:	A crystalline 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

GNF-5 is supplied as a crystalline solid. A stock solution may be made by dissolving the GNF-5 in the solvent of choice. GNF-5 is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF), which should be purged with an inert gas. The solubility of GNF-5 in these solvents is approximately 0.25, 10, and 25 mg/ml, respectively.

GNF-5 is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, GNF-5 should first be dissolved in DMF and then diluted with the aqueous buffer of choice. GNF-5 has a solubility of approximately 0.25 mg/ml in a 1:2 solution of DMF:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

GNF-5 is an allosteric inhibitor of Bcr-Abl (IC_{50} = 0.121 µM) and a derivative of GNF-2 (Item No. 16253).[1] It also inhibits the Bcr-Abl mutants Bcr-Abl^{G250E}, Bcr-Abl^{E255V}, and Bcr-Abl^{M351T} (IC_{50}s = 4.52, 0.38, and 0.93 µM, respectively).[2] GNF-5 inhibits the growth of Ba/F3 cells (IC_{50} = 0.145 µM).[1] It reduces viral titers in Vero cells infected with infectious bronchitis virus (IBV), a coronavirus, when used at a concentration of 10 µM *via* inhibition of IBV surface glycoprotein-induced syncytia formation and virus-cell fusion.[3] GNF-5 (75 mg/kg) increases survival in a recalcitrant mutant Bcr-Abl^{T315I} mouse bone marrow transplantation model when administered alone or in combination with nilotinib.[2]

References

1. Fabbro, D., Manley, P.W., Jahnke, W., *et al.* Inhibitors of the Abl kinase directed at either the ATP- or myristate-binding site. *Biochim. Biophys. Acta* **1804(3)**, 454-462 (2010).
2. Zhang, J., Adrián, F.J., Jahnke, W., *et al.* Targeting Bcr-Abl by combining allosteric with ATP-binding-site inhibitors. *Nature* **463(7280)**, 501-506 (2010).
3. Sisk, J.M., Frieman, M.B., and Machamer, C.E. Coronavirus S protein-induced fusion is blocked prior to hemifusion by Able kinase inhibitors. *J. Gen. Virol.* **99(5)**, 619-630 (2018).

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