Product Data Sheet

Product Name: 6H05
Cat. No.: GC15478

Chemical Properties

Cas. No.	1469338-01-9		
Chemical Name	1-(2-((4-chlorophenyl)thio)acetyl)-N-(2-((2-(dimethylamino)ethyl)disulfanyl)ethyl)piperidine-4-carboxamide 2,2,2-trifluoroacetate		
SMILES	ClC1=CC=C(SCC(N2CCC(C(NCCSSCCN(C)C)=O)CC2)=O)C=C1.OC(C(F)(F)F)=O		
Formula	$C_{22}H_{31}ClF_3N_3O_4S_3$	M.Wt	590.14
Solubility	≥ 29.5mg/mL in DMSO	Storage	Store at -20°C
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	6H05		

Background

6H05 is a selective inhibitorof the common oncogenic mutant K-Ras (G12C). 6H05 allosterically modifies and inhibits the oncogenic G12C mutant of highly homologous protein H-Ras, not affecting the wild-type K-Ras [1].

K-Ras plays an important role in human cancer, cause mutations in K-Ras are the most common activating lesions in human cancer[2]. 6H05 modifies the GDP-bound K-Ras (G12C) the most, which can be and be applied as the starting point for drug-discovery efforts targeting K-Ras (G12C) and eventually other alleles of K-Ras. 6H05 can be used as an intermediate for the synthesis of oncogenic K-Ras (G12C) inhibitors [3, 4].

Although it’s necessary to perform continued chemical optimization of 6H05 to be assessed in vivo, preliminary evaluation of 6H05 in lung cancer cell lines suggests that 6H05 shows allele-specific impairment of K-Ras function[1]. There are questions still

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need to be illustrated that the selectivity and efficiency of 6H05 in vivo, as well as its effects on the subcellular localization of other farnesylated GTPases should be assessed further[1, 3].

Questions still need to be answered surrounding the in vivo selectivity and efficacy of 6H05, including its effects on the subcellular localization of other farnesylated GTPases [3].

References:
[1] Ostrem JM, Peters U, Sos ML, et al. K-Ras (G12C) inhibitors allosterically control GTP affinity and effector interactions[J]. Nature, 2013, 503(7477): 548-551.
[2] McCormick F. KRAS as a Therapeutic Target[J]. Clinical Cancer Research, 2015, 21(8): 1797-1801.
[3] Milroy L-G,Ottmann C. The Renaissance of Ras[J]. ACS chemical biology, 2014, 9(11): 2447-2458.
[4] Lu SY, Li S,Zhang J. Harnessing Allostery: A Novel Approach to Drug Discovery[J]. Medicinal Research Reviews, 2014, 34(6): 1242-1285.