
Product Data Sheet

Product Name: ALLO-1
Cat. No.: GC14749

Chemical Properties

Cas. No. 37468-32-9

Chemical Name 3-(4-chlorophenyl)-5-methyl-1-(phenylmethyl)-2,4-imidazolidinedione

SMILES O=C1N(C2=CC=C(Cl)C=C2)C(N(CC3=CC=CC=C3)C1C)=O

Formula $C_{17}H_{15}ClN_2O_2$ M.Wt 314.8

Solubility $\leq 20\text{mg/ml}$ in DMSO; 30mg/ml in dimethyl formamide 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

IC50: 50 nM for wile type Smo

ALLO-1 is a SMO antagonist.

Hedgehog (Hh) proteins are important development regulators that bind the cell-surface protein, which allows the activation of a GPCR-like receptor, Smoothened (SMO). In vertebrates, the SMO activation finally results in the activation of the zinc-finger transcription factors of the Gli family. In addition, the overactivation of SMO may lead to certain cancers.

In vitro: Previous study found that ALLO-1 and its close analog ALLO-2 could inhibit Smo agonist Hh-Ag 1.5-induced luciferase expression in TM3-Gli-Luc cells. The potency of ALLO-1 did not change when either low dose or high dose of Hh-Ag 1.5 was used, in contrast to other known Smo antagonists that are strong SAG or Hh-Ag 1.5 competitors. Moreover, it was found that in contrast to GDC-0449, both ALLO-1 and ALLO-2 inhibited

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

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wild-type and the D477G mutant with only around 2-fold shift in IC₅₀, indicating that the D477G mutation did not significantly interfere with the binding of ALLO-1 and ALLO-2 to Smo. In addition, ALLO-1 as well as ALLO-2 were able to inhibit both wild-type and D473H mutant human SMO with similar potencies [1].

In vivo: Up to now, there is no animal in vivo data reported.

Clinical trial: So far, no clinical study has been conducted.

Reference:

[1] Tao, H., Jin, Q., Koo, D.I., et al. Small molecule antagonists in distinct binding modes inhibit drug-resistant mutant of smooth muscle. *Chemistry & Biology* 18, 432-437 (2011).

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