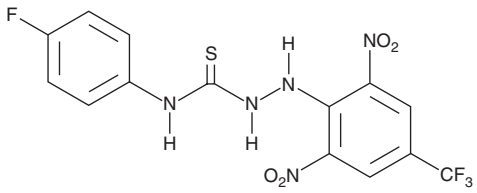
PRODUCT INFORMATION

Kobe 2602

Item No. 16262

CAS Registry No.: 454453-49-7
Formal Name: 2-[2,6-dinitro-4-(trifluoromethyl)phenyl]-N-(4-fluorophenyl)-hydrazinecarbothioamide
MF: $C_{14}H_9F_4N_5O_4S$
FW: 419.3
Purity: ≥98%
UV/Vis.: λ_{max}: 226, 232, 366 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

Kobe 2602 is supplied as a crystalline solid. A stock solution may be made by dissolving the kobe 2602 in the solvent of choice, which should be purged with an inert gas. Kobe 2602 is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide. The solubility of kobe 2602 in these solvents is approximately 5, 20, and 10 mg/ml, respectively.

Kobe 2602 is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, kobe 2602 should first be dissolved in DMSO and then diluted with the aqueous buffer of choice. Kobe 2602 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

The Ras family of small GTPases (H-Ras, K-Ras, and N-Ras) function as molecular switches, cycling between a GTP-bound active state and a GDP-bound inactive state, to turn on downstream Raf protein kinases. This initiates complex signaling pathways involved in cell growth, differentiation, and apoptosis. Mutations leading to aberrant Ras activation are frequently associated with various human cancers. Kobe 2602 is a selective Ras inhibitor that blocks H-Ras GTP binding to c-Raf-1 (K_i = 149 µM).[1] Kobe 2602 has been shown to inhibit both anchorage-dependent and -independent growth and to induce apoptosis of H-Ras^{G12V}-transformed NIH 3T3 cells (IC_{50} = 1.4-2 µM).[1] At an oral dose of 80 mg/kg, it also exhibits antitumor activity in mice bearing a xenograft of human colon cancer SW480 cells expressing K-Ras^{G12V}.[1]

Reference

1. Shima, F., Yoshikawak, Y., Ye, M., *et al.* In silico discovery of small-molecule Ras inhibitors that display antitumor activity by blocking the Ras-effector interaction. *Proc. Natl. Acad. Sci. USA* **110(20)**, 8182-8187 (2013).

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