Web: www.taiclone.com
Tel: +886-2-2735-9682
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Sorafenib

Catalog No: tcsc1590

Available Sizes

Size: 100mg

Size: 500mg

Size: 1g

Size: 2g

Size: 5g

Size: 10g

Specifications

CAS No:
284461-73-0

Formula:
$C_{21}H_{16}ClF_3N_4O_3$

Pathway:
Protein Tyrosine Kinase/RTK;MAPK/ERK Pathway;Protein Tyrosine Kinase/RTK;Protein Tyrosine Kinase/RTK;Autophagy;Protein Tyrosine Kinase/RTK

Target:
VEGFR;Raf;FLT3;PDGFR;Autophagy;c-Kit

Purity / Grade:
>98%

Solubility:
DMSO : ≥ 45 mg/mL (96.81 mM)

Alternative Names:

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Bay 43-9006

Observed Molecular Weight:
464.83

Protocol:
Kinase Assay: To test compound inhibition against various RAF kinase isoforms, Sorafenib is added to a mixture of Raf-1 (80 ng), wt BRAF, or V599E BRAF (80 ng) with MEK-1 (1 μg) in assay buffer [20 mM Tris (pH 8.2), 100 mM NaCl, 5 mM MgCl2, and 0.15% β-mercaptoethanol] at a final concentration of 1% DMSO. The RAF kinase assay (final volume of 50 μL) is initiated by adding 25 μL of 10 μM γ-[33P]ATP (400 Ci/mol) and incubated at 32°C for 25 minutes. Phosphorylated MEK-1 is harvested by filtration onto a phosphocellulose mat, and 1% phosphoric acid is used to wash away unbound radioactivity. After drying by microwave heating, a β-plate counter is used to quantify filter-bound radioactivity. Cell Assay: Sorafenib is dissolved in DMSO and stored, and then diluted with appropriate media before use[1]. [1]The MDA-MB-231 human mammary adenocarcinoma cell lines are plated at 2×105 cells per well in 12-well tissue culture plates in DMEM growth media (10% heat-inactivated FCS) overnight. Cells are washed once with serum-free media and incubated in DMEM supplemented with 0.1% fatty acid-free BSA containing various concentrations of BAY 43-9006 (0.01, 0.03 , 0.1, 0.3, 1, 3 μM) in 0.1% DMSO for 120 minutes to measure changes in basal pMEK 1/2, pERK 1/2, or pPKB. Cells are washed with cold PBS (PBS containing 0.1 mM vanadate) and lysed in a 1% (v/v) Triton X-100 solution containing protease inhibitors. Lysates are clarified by centrifugation, subjected to SDS-PAGE, transferred to nitrocellulose membranes, blocked in TBS-BSA, and probed with anti-pMEK 1/2 (Ser217/Ser221; 1:1000), anti-MEK 1/2, anti-pERK 1/2 (Thr202/Tyr204; 1:1000), anti-ERK 1/2, anti-pPKB (Ser473; 1:1000), or anti-PKB primary antibodies.

Product Description

Sorafenib is a potent multikinase inhibitor with **IC_{50}**s of 6 nM, 20 nM, and 22 nM for **Raf-1**, **B-Raf**, and **VEGFR-3**, respectively.

IC50 & Target: IC50: 6 nM (Raf-1), 20 nM (VEGFR-3), 22 nM (BRAF), 57 nM (PDGFR-β), 58 nM (Flt3), 68 nM (c-KIT), 90 nM (VEGFR-2)[1]

In Vitro: Sorafenib (BAY 43-9006) also inhibits $BRAF^{wt}$ (IC_{50}=22 nM), $BRAF^{V599E}$ (IC_{50}=38 nM), VEGFR-2 (IC_{50}=90 nM), VEGFR-3 (IC_{50}=20 nM), PDGFR-β (IC_{50}=57 nM), c-KIT (IC_{50}=68 nM), and Flt3 (IC_{50}=58 nM) in biochemical assays. In MDA-MB-231 breast cancer cells, Sorafenib completely blocks activation of the MAPK pathway. Cells are preincubated with Sorafenib (0.01 to 3 μM), and dose-dependent inhibition of basal MEK 1/2 and ERK 1/2 phosphorylation (IC_{50}, 40 and 100 nM, respectively)[1].

In Vivo: Sorafenib demonstrates broad oral antitumor efficacy in panel of human tumor xenograft models. Sorafenib is given orally at 7.5 to 60 mg/kg. There is no lethality and no increase in weight loss in any treated group relative to the corresponding control group. Daily oral administration of Sorafenib (30 to 60 mg/kg) produces complete tumor stasis during treatment in five of the six models[1]. The survival rate is 73.3 % in Diethyl nitrosamine (DENA) group and 83.3 % in Sorafenib group compared to 100 % in the normal control group. DENA group shows a significant increase in liver index (1.51-fold increase, p[2].

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