Revision 1

e at -20C	Sorafenib	J.	Cell Signaling
Store		Orders:	877-616-CELL (2355) orders@cellsignal.com
10	10 mg	Support	:: 877-678-TECH (8324)
705		Web:	info@cellsignal.com cellsignal.com
#8		3 Trask Lane Danvers	Massachusetts 01923 USA

For Research Use Only. Not for Use in Diagnostic Procedures.

Background	Sorafenib, also known as Bay 43-9006, is a novel multikinase inhibitor that targets the RAF family of serine/threonine kinases and tyrosine kinase receptors involved in tumor progression and tumor angiogenesis, including: VEGFR-2 (IC ₅₀ = 90 nM), VEGFR-3 (IC ₅₀ = 20 nM), PDGFR- (IC ₅₀ = 57 nM), c-KIT (IC ₅₀ = 68 nM), and Flt3 (IC ₅₀ = 58 nM) (1). Research studies have demonstrated that sorafenib induces apoptosis in several tumor cell lines through the down-regulation of the antiapoptotic protein myeloid cell leukemia-1 (Mcl-1). Down-regulation of Mcl-1 by sorafenib is associated with the release of cytochrome c from mitochondria into the cytosol and caspase activation, leading to apoptotic cell death (2). STAT3 inhibition by sorafenib has been observed in multiple cell types (3-5).
Molecular Formula	C ₂₁ H ₁₆ CIF ₃ N ₄ O ₃ • C ₇ H ₈ O ₃ S
Molecular Weight	637.03 g/mol
Purity	>99%
CAS	475207-59-1
Solubility	Soluble in DMSO at 200mg/ml.
Storage	Store lyophilized or in solution at -20°C. In lyophlized form, the chemical is stable for 24 months. Once in solution, use within 3 months to prevent loss of potency.
Directions for Use	Sorafenib is supplied as a lyophilized powder. For a 10 mM stock, reconstitute the 10 mg in 1.57 ml DMSO. Working concentrations and length of treatment can vary depending on the desired effect, but it is typically used as a pretreatment at 0.1-10 μ M for 0.5-2 hr prior to treating with a stimulator. It can also be used alone, with varying treatment times lasting up to 24 hr. Soluble in DMSO at 200 mg/ml; very poorly soluble in ethanol and water with maximum solubility in water ~10-20 μ M.
Background References	1. Wilhelm, S.M. et al. (2004) <i>Cancer Res</i> 64, 7099-109. 2. Yu, C. et al. (2005) <i>Oncogene</i> 24, 6861-9. 3. Zhao, W. et al. (2011) <i>Anticancer Drugs</i> 22, 79-88. 4. Huang, S. and Sinicrope, F.A. (2010) <i>Mol Cancer Ther</i> 9, 742-50. 5. Yang, F. et al. (2008) <i>Mol Cancer Ther</i> 7, 3519-26.
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