## SB203580

## (4-(4-Fluorophenyl)-2-(4-methylsulfinylphenyl)-5-(4-pyridyl)-1H-imidazole) (p38 MAPK inhibitor)

| Cat.-No. | Amount |
| :---: | :---: |
| INH-007 | 2 mg |




Selected references:
Kalmes et al. (1999) Raf-1 is activated by the p38 mitogen-activated protein kinase inhibitor SB203580. FEBS Lett. 444:71.
Lali et al. (2000) The pyridinyl imidazole inhibitor SB203580 blocks phosphoinositide-dependent protein kinase activity, protein kinase B phosphorylation, and retinoblastoma hyperphosphorylation in interleukin-2-stimulated T cells independently of p38 mitogenactivated protein kinase. J. Biol. Chem. 275:7395.
Clerk A. and Sugden P. H. (1998) The p38-MAPK inhibitor, SB203580, inhibits cardiac stress-activated protein kinases/c-Jun Nterminal kinases (SAPKs/JNKs). FEBS Lett. 426:93.

Lyophilized.

SB203580 is a recognized inhibitor of p38-MAPKs. The IC50 for inhibition of p38-MAPK stimulation of MAPKAPK2 was approximately $0.07 \mu \mathrm{M}$, whereas that for total SAPK/JNK activity was 3-10 $\mu \mathrm{M}$. SB203580 did not inhibit immunoprecipitated JNK1 isoforms.
The pyridinyl imidazole compounds were found to act as specific inhibitors of $\mathrm{p} 38 \alpha$ and $\mathrm{p} 38 \beta$ but not $\mathrm{p} 38 \gamma$ and p38\% through competition with ATP for the same binding site on the p38 kinase.
SB203580 can inhibit the key cell cycle event of retinoblastoma protein phosphorylation in interleukin-2stimulated $T$ cells. Studies on the proximal regulator of this event, the phosphatidylinositol 3-kinase/protein kinase B (PKB)(Akt/Rac) kinase pathway, showed that SB203580 blocked the phosphorylation and activation of PKB by inhibiting the PKB kinase, phosphoinositidedependent protein kinase 1.

## For in vitro use only!

Purity: $\geq 98 \%$
Molecular formula: $\mathrm{C}_{21} \mathrm{H}_{16} \mathrm{FN}_{3} \mathrm{OS}$
Molecular weight: $377.43 \mathrm{~g} / \mathrm{mol}$
Solubility: $50 \mathrm{mg} / \mathrm{ml}$ in DMSO.
Store: - $20^{\circ} \mathrm{C}$

