

Product Information

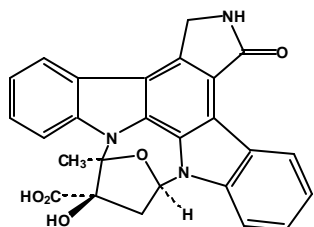
K-252b Ready-Made solution from *Nonomuraea longicatena*

Catalog Number **K2139**
Storage Temperature $-20\text{ }^{\circ}\text{C}$

CAS RN: 99570-78-2

Product Description

Molecular Weight: 453.45
Molecular formula: $\text{C}_{26}\text{H}_{19}\text{N}_3\text{O}_5$



K-252b belongs to the K-252 family of compounds, which are general and potent inhibitors of various protein kinases, possibly acting by interfering at or near the ATP binding site.¹ K-252b is an inhibitor of protein kinase A, G, and C. The highest inhibition potency is of protein kinases C with a K_i value of 20 nM, comparable to K-252a (with a K_i value of 18-25 nM).² Compared to K-252a, K-252b exhibits reduced inhibitory potency for cyclic nucleotide-dependent protein kinases.² K-252b can potentiate the neurotrophin-3-induced tyrosine phosphorylation of Trk on PC12 cells.^{1,3} K-252b is also a selective and non-toxic inhibitor of nerve growth factor (NGF) action in the brain.⁴ K-252b was found to be as potent as K-252a in inhibiting the nerve growth factor-induced neurite outgrowth on PC12, although it does not permeate the cell membrane as does K-252a.⁵ In cultured cortical neurons, K-252b not only blocks the synapse formation but also inhibits the phosphorylation of some cell surface proteins.^{6,7} K-252b was used, as a blocker, in studies of the mechanism of ATP-induced long-term potentiation (LTP).⁸ It was found to markedly reduce the relaxation of the K^+ -contracted rat vas deferens caused by ATP.⁹

Reagent

K-252b is supplied as a 1 mM solution in DMSO.

Precautions and Disclaimer

This product is for R&D use only, not for drug, household, or other uses. Please consult the Material Safety Data Sheet for information regarding hazards and safe handling practices.

Storage/Stability

Store protected from light at $-20\text{ }^{\circ}\text{C}$. Under these conditions the product is stable for 3 years.

References

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3. Knusel, B., et al., *J. Neurochem.*, **59**, 715-722 (1992).
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5. Nagashima, K., et al., *FEBS Lett.*, **293**, 119-123 (1991).
6. Kuroda, Y., et al., *Neurosci. Lett.*, **135**, 255-258 (1992).
7. Muramoto, K., et al., *Biochem. Biophys. Res. Commun.*, **205**, 1467-1473 (1994).
8. Fujii, S., et al., *Neuroscience*, **113**, 617-628 (2002).
9. Bultmann, R., et al., *Eur. J. Pharmacol.*, **436**, 135-143 (2002).

ES,SP,AH,PHC 10/05-1

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