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ProductInformation

Tyrphostin 51

Product Number **T 7665**
Storage Temperature 2-8 °C

Product Description
Molecular Formula: $C_{13}H_8N_4O_3$
Molecular Weight: 268.2
CAS Number: 126433-07-6
Melting Point: 275 °C (with decomposition)[1]
IC_{50}: 0.8 µM[1]
Synonym: 2-amino-1,1,3-tricyano-4(3',4',5'-trihydroxyphenyl)butadiene

Tyrphostin 51 is one of a series of small molecular weight inhibitors of epidermal growth factor (EGF) receptor kinase activity which were designed to bind to the substrate subsite of the protein tyrosine kinase (PTK) domain.[1,2] The synthesis and characterization of tyrphostin 51 and the related family of compounds has been described.[1]

Tyrphostin 51 has been shown to inhibit phosphorylation of rat hepatic lectin 1 (0-1 mM tyrphostin 51) and to prevent ATP-dependent asialoglycoprotein receptor inactivation (0-0.1 mM tyrphostin 51) in rat hepatocytes.[3] Tyrphostin 51 has been utilized to probe the role of EGF in the regulation of glycolipid sulfotransferase activity in human renal cancer cells.[4] A study in rat pulmonary arterties has demonstrated that tyrphostin 51 can diminish both the initial, transient contraction and subsequent sustained contractions that are initially induced by H_2O_2.[5]

Tyrphostin 51 has been used at 10 µM to investigate rabbit jejunal brush-border and basolateral membrane transport.[6] An investigation in cultured rat retinal pigment epithelium cells with 10 µM tyrphostin has a investigated the alteration of Ca^{2+} transport by the Ca^{2+}/InsP3 second messenger system.[7]

Precautions and Disclaimer
For Laboratory Use Only. Not for drug, household or other uses.

Preparation Instructions
This product is soluble in DMSO (50 mg/ml), yielding a clear, orange-red solution.

Storage/Stability
Tyrphostins should be stable for months in DMSO stored frozen. The presence of water in the solution may accelerate hydrolysis.

References
1. Gazit, A., et al., Tyrphostins I: synthesis and biological activity of protein tyrosine kinase inhibitors. J. Med. Chem., **32(10)**, 2344-2352 (1989).
2. Yaish, P., et al., Blocking of EGF-dependent cell proliferation by EGF receptor kinase inhibitors. Science, **242(4880)**, 933-935 (1988).
3. Haynes, P. A., et al., Inhibition of tyrosine phosphorylation in the rat hepatic lectin 1 subunit of the rat asialoglycoprotein receptor prevents ATP-dependent receptor inactivation in permeabilized hepatocytes. J. Biol. Chem., **269(52)**, 33152-33158 (1994).
4. Balbaa, M., et al., Regulation of glycolipid sulfotransferase by tyrosine kinases in human renal cancer cells. Biochim. Biophys. Acta, **1299(1)**, 141-145 (1996).
5. Jin, N., and Rhoades, R. A., Activation of tyrosine kinases in H_2O_2-induced contraction in pulmonary artery. Am. J. Physiol., **272(6 Pt 2)**, H2686-2692 (1997).
6. Hardin, J. A., et al., Effect of luminal epidermal growth factor on enterocyte glucose and proline transport. Am. J. Physiol., **271(3 Pt 1)**, G509-515 (1996).
7. Mergler, S., and Strauss, O., Stimulation of L-type Ca^{2+} channels by increase of intracellular InsP3 in rat retinal pigment epithelial cells. Exp. Eye Res., **74(1)**, 29-40 (2002).

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