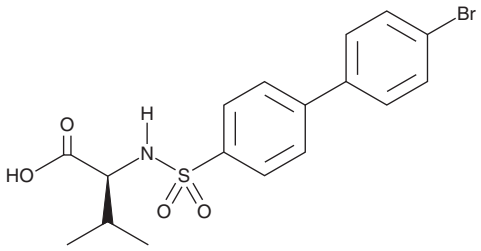
PRODUCT INFORMATION

PD 166793

Item No. 23762

CAS Registry No.:	199850-67-4
Formal Name:	N-[(4'-bromo[1,1'-biphenyl]-4-yl) sulfonyl]-L-valine
MF:	$C_{17}H_{18}BrNO_4S$
FW:	412.3
Purity:	≥98%
UV/Vis.:	λ_{max}: 204, 272 nm
Supplied as:	A crystalline solid
Storage:	-20°C
Stability:	≥4 years



Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Laboratory Procedures

PD 166793 is supplied as a crystalline solid. A stock solution may be made by dissolving the PD 166793 in the solvent of choice. PD 166793 is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF), which should be purged with an inert gas. The solubility of PD 166793 in ethanol is approximately 10 mg/ml and approximately 50 mg/ml in DMSO and DMF.

PD 166793 is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, PD 166793 should first be dissolved in DMSO and then diluted with the aqueous buffer of choice. PD 166793 has a solubility of approximately 0.5 mg/ml in a 1:1 solution of DMSO:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

PD 166793 is a broad-spectrum matrix metalloproteinase (MMP) inhibitor (IC_{50}s = 6.1, 0.047, 0.012, 7.2, 7.9, 0.008, and 0.24 µM for MMP-1, MMP-2, MMP-3, MMP-7, MMP-9, MMP-13CD, and MMP-14CD, respectively).[1] It reverses peroxynitrite-induced decreases in contraction cease time, a measure of cardiac contractility, in isolated rat ventricular myocytes when used at a concentration of 2 µM.[2] *In vivo*, PD 166793 (2 mg/kg per day, p.o.) reduces left ventricular (LV) peak wall stress in a porcine model of congestive heart failure.[3] It reduces LV dilation and preserves systolic function in a rat model of progressive heart failure.[1] PD 166793 also inhibits age-associated increases in aortic gelatinase and interstitial collagenase activity and mean arterial pressure in rats.[4]

References

1. Peterson, J.T., Hallak, H., Johnson, L., *et al.* Matrix metalloproteinase inhibition attenuates left ventricular remodeling and dysfunction in a rat model of progressive heart failure. *Circulation* **103(18)**, 2303-2309 (2001).
2. León, H., Baczkó, I., Sawicki, G., *et al.* Inhibition of matrix metalloproteinases prevents peroxynitrite-induced contractile dysfunction in the isolated cardiac myocyte. *Br. J. Pharmacol.* **153(4)**, 676-683 (2008).
3. McElmurray, J.H., III, Mukherjee, R., New, R.B., *et al.* Angiotensin-converting enzyme and matrix metalloproteinase inhibition with developing heart failure: Comparative effects on left ventricular function and geometry. *J. Pharmacol. Exp. Ther.* **291(2)**, 799-811 (1999).
4. Wang, M., Zhang, J., Telljohann, R., *et al.* Chronic matrix metalloproteinase inhibition retards age-associated arterial proinflammation and increase in blood pressure. *Hypertension* **60(2)**, 459-466 (2012).

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