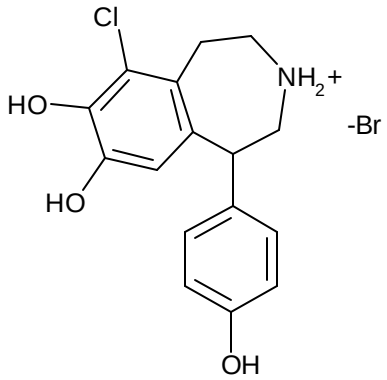
3050 Spruce Street
Saint Louis, Missouri 63103 USA
Telephone 800-325-5832 • (314) 771-5765
Fax (314) 286-7828
email: techserv@sial.com
sigma-aldrich.com

ProductInformation

FENOLDOPAM MONOHYDROBROMIDE
Product Number **F6800**
Storage Temperature: 2-8°C



CAS#: 67287-54-1
Synonyms: SKF-82526

Product Description

Molecular Formula: $C_{16}H_{16}ClNO_3 \bullet HBr$
Molecular Weight: 386.68 (anhydrous)

Dopamine (DA) receptors were initially divided into two general categories on the basis of differences in receptor pharmacology and biochemical mechanisms of signal transduction. With the application of molecular biology techniques, several groups cloned the two predominant DA receptors, D_1 and D_2. Additional dopamine receptors were identified based on their homology to $D_1$and D_2. Two families of DA receptors are currently recognized. The D_1-like family is composed of the D_1 and D_5 receptors. The D_2-like family includes D_2, D_3, and D_4.[1] The availability of agonists and antagonists selective for the subtypes of dopamine receptors allows physiological roles to be attributed to these receptors.

D_1 receptors on vascular smooth muscle cells mediate vasodilation, while on renal tubule cells they increase sodium excretion by inhibiting the Na^+/K^+ ATPase responsible for sodium reuptake. Fenoldopam is a selective agonist at peripheral D_1 dopamine receptors.[2] It activates vascular D_1 receptors and induces vasodilation of the vascular beds of the kidney, heart, brain, and mesentery. It also activates renal tubule D_1 receptors and induces diuresis and natriuresis that may be mediated in part through increased nitric oxide production.[3] Both mechanisms are involved in the reduction of blood pressure induced by fenoldopam in normotensive animals.[4] In contrast, fenoldopam reduced the blood pressure of spontaneously hypertensive rats, but has little effect on renal blood flow, indicating a defect in renal D_1 receptors in these animals.[5,6] Fenoldopam does not cross the blood-brain barrier.[2]

Preparation Instructions

Soluble in water (4.75 mg/ml) and DMSO (>12 mg/ml).

Storage/Stability

Store at 2-8°C sealed tightly under argon. Protect from exposure to light and air.

References

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4. Quevedo, M., et al., Gen. Pharmacol. **32**, 123-125 (1999).
5. deVries, P.A., et al., J. Cardiovasc. Pharmacol., **34**, 191-198 (1999).
6. Hussain, T., and Lokhandwala, M.F., Am. J. Physiol. **272**, F339-F346 (1997).

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