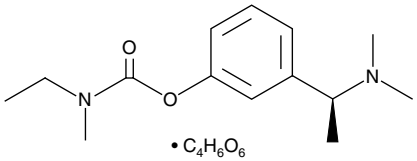
# Product Information

## Rivastigmine (tartrate)

*Item No. 14270*

**CAS Registry No.:** 129101-54-8
**Formal Name:** 3-[(1S)-1-(dimethylamino)ethyl]phenyl ester N-ethyl-N-methyl-carbamic acid, 2R,3R-dihydroxybutanedioate
**Synonyms:** Exelon, SDZ-ENA 713
**MF:** $C_{14}H_{22}N_2O_2 \bullet C_4H_6O_6$
**FW:** 400.4
**Purity:** ≥95%
**Stability:** ≥2 year at -20°C
**Supplied as:** A crystalline solid




### Laboratory Procedures

For long term storage, we suggest that rivastigmine (tartrate) be stored as supplied at -20°C. It should be stable for at least two years.

Rivastigmine (tartrate) is supplied as a crystalline solid. A stock solution may be made by dissolving the rivastigmine (tartrate) in the solvent of choice. Rivastigmine (tartrate) is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF), which should be purged with an inert gas. The solubility of rivastigmine (tartrate) in ethanol and DMSO is approximately 16 mg/ml and in DMF it is approximately 25 mg/ml.

Further dilutions of the stock solution into aqueous buffers or isotonic saline should be made prior to performing biological experiments. Ensure that the residual amount of organic solvent is insignificant, since organic solvents may have physiological effects at low concentrations. Organic solvent-free aqueous solutions of rivastigmine (tartrate) can be prepared by directly dissolving the crystalline solid in aqueous buffers. The solubility of rivastigmine (tartrate) in PBS, pH 7.2, is approximately 10 mg/ml. We do not recommend storing the aqueous solution for more than one day.

Rivastigmine is an irreversible inhibitor of acetylcholinesterase ($IC_{50}$ = 4.15 µM) and butyrylcholinesterase ($IC_{50}$ = 37 nM).[1-2] It acts by covalently modifying a serine residue in the active site by carbamoylation.[3] Rivastigmine has found use in modifying the course of neurodegenerative diseases, including Parkinson's disease and Alzheimer's disease, as it stabilizes or reduces the rate of decline in certain cognitive functions.[4-6]

### References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2. Luo, W., Yu, Q.-S., Kulkarni, S.S., *et al.* Inhibition of human acetyl- and butyrylcholinesterase by novel carbamates of (-)- and (+)-tetrahydrofurobenzofuran and methanobenzodioxepine. *J. Med. Chem.* **49(7)**, 2174-2185 (2006).
3. Muñoz-Torrero, D. Acetylcholinesterase inhibitors as disease-modifying therapies for Alzheimer's disease. *Curr. Med. Chem.* **15**, 2433-2455 (2008).
4. Baskys, A. and Hou, A.C. Vascular dementia: Pharmacological treatment approaches and perspectives. *Clin. Interv. Aging* **2(3)**, 327-335 (2007).
5. Grossberg, G., Cummings, J., Frölich, L., *et al.* Efficacy of higher dose 13.3 mg/24 h rivastigmine patch on instrumental activities of daily living in patients with mild-to-moderate Alzheimer's disease. *Am. J. Alzheimers Dis. Other Demen.* **28(6)**, 583-591 (2013).
6. Possin, K.L., Kang, G.A., Guo, C., *et al.* Rivastigmine is associated with restoration of left frontal brain activity in Parkinson's disease. *Mov. Disord.* **28(10)**, 1384-1390 (2013).


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