PRODUCT INFORMATION

(R)-Flurbiprofen
Item No. 70255

CAS Registry No.: 51543-40-9
Formal Name: (R)-(−)-2-fluoro-α-methyl-4-
biphenylacetic acid
Synonyms: E-7869, Flurizan, Tarenflurbil
MF: C₁₅H₁₃FO₂
FW: 244.3
Purity: ≥99%
UV/Vis.: λmax: 247 nm
Supplied as: A crystalline solid
Storage: 22°C
Stability: ≥2 years

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

Laboratory Procedures

(R)-Flurbiprofen is supplied as a crystalline solid. A stock solution may be made by dissolving the (R)-flurbiprofen in the solvent of choice. (R)-Flurbiprofen is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF), which should be purged with an inert gas. The solubility of (R)-flurbiprofen in ethanol and DMF is approximately 25 mg/ml and approximately 10 mg/ml in DMSO. 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 (R)-flurbiprofen can be prepared by directly dissolving the crystalline solid in aqueous buffers. The solubility of (R)-flurbiprofen in PBS, pH 7.2, is approximately 0.9 mg/ml. We do not recommend storing the aqueous solution for more than one day.

Description

(R)-Flurbiprofen is a COX-inactive enantiomer of the racemic non-selective COX inhibitor flurbiprofen (Item No. 70250) that has diverse biological activities. It inhibits γ-secretase activity in vitro and, in vivo, it reduces formation of amyloid-β peptide 1-42 (Aβ42) and improves axonal transport in young Aβ-plaque free mice but not old mice with existing Aβ plaques in the Tg2576 transgenic model of Alzheimer’s disease. (R)-Flurbiprofen inhibits NF-κB activation and DNA binding as well as AP-1 DNA binding in RAW 264.7 macrophages and reduces paw edema in a rat model of zymosan-induced inflammation via COX-independent inhibition of NF-κB and AP-1 activation when administered at doses of 1, 3, and 9 mg/kg. It also suppresses prostate tumor cell growth in vitro by inducing p75NTR protein expression and reduces tumor growth and metastasis in multiple mouse models of intestinal neoplasia.

References