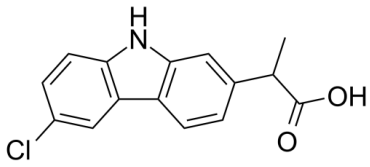
Data Sheet

Product Name:	Carprofen
Cat. No.:	CS-4875
CAS No.:	53716-49-7
Molecular Formula:	C15H12ClNO2
Molecular Weight:	273.71
Target:	Autophagy; COX; FAAH
Pathway:	Autophagy; Immunology/Inflammation; Metabolic Enzyme/Protease; Neuronal Signaling
Solubility:	DMSO : ≥ 33 mg/mL (120.57 mM)



BIOLOGICAL ACTIVITY:

Carprofen is a nonsteroid anti-inflammatory agent, acts as a multi-target **FAAH/COX** inhibitor, with **IC_{50}**s of 3.9 μM, 22.3 μM and 78.6 μM for COX-2, COX-1 and FAAH, respectively. IC50 & Target: IC50: 3.9 μM (COX-2), 22.3 μM (COX-1), 78.6 μM (FAAH)[1] **In Vitro:** Carprofen (Compound 1) is a nonsteroid anti-inflammatory agent, acts as a multi-target FAAH/COX inhibitor, with IC_{50}s of 3.9 μM, 22.3 μM and 78.6 μM for COX-2, COX-1 and FAAH, respectively[1].

Carprofen (10 μg/mL) shows cytoprotective effects in CCL and CaCL cells and decreases apoptosis of both cells. Carprofen (10 μg/mL) exhibits nonsignificant increase in PGE2 concentration, compared with that of the respective CCL or CaCL controls[2]. **In Vivo:** Carprofen (2.2 mg/kg, p.o.) significantly decreases PGE2 concentration in blood of dogs on days 3 and 10. Carprofen also decreases amounts of gastric PGE2 synthesis on day 3, but the inhibition is not obvious on day 10. In addition, Carprofen shows no activity against gastric PGE1 synthesis in dogs on day 3 and 10[3].

PROTOCOL (Extracted from published papers and Only for reference)

Cell Assay: [2]**Cruciate ligament cells** are used and incubated with DMEM supplemented with 10% FCS for 24 hours to synchronize cell cycles. The cell cultures are then preincubated without (control) or with a nonselective COX inhibitor (acetylsalicylic acid) or a preferential COX-2 inhibitor (**Carprofen**, meloxicam, or robenacoxib) to ssess whether NSAIDs prevented apoptosis when the cells are subsequently incubated with SNP. For all cell cultures except those designated as controls, 1 of 3 concentrations of 1 of the 4 NSAIDs (10, 100, or 200 μg of acetylsalicylic acid/mL; **0.1, 1, or 10 μg of Carprofend/mL**; 0.1, 1, or 10 μg of meloxicame/mL; or 0.1, 1, or 10 μg of robenacoxibf/mL) is added to the culture media of each cell culture, and the cells are incubated for 2 hours[2].

Animal Administration: [3]Dogs[3]

Each **dog** receives **Carprofen (2.2 mg/kg, PO, q 12 h)**, deracoxib (2 mg/kg, PO, q 24 h), or etodolac (10 to 15 mg/kg, PO, q 24 h) for 10 days in a crossover design with a 30- to 60-day washout period between treatments. On days 0, 3, and 10 of each treatment period, blood is collected for evaluation of **TXB2** and **PGE2** concentrations. In addition, anesthesia is induced with propofol (4 mg/kg) and maintained with isoflurane. Synovial fluid is collected from both stifle joints by use of a standard arthrocentesis technique for evaluation of PGE2 concentrations. Gastroscopy is performed during each anesthetic episode, and 3 to 6 endoscopic biopsy specimens are collected from the gastric antrum for evaluation of PGE1 and PGE2 synthesis. On day 0 for each dog, a gastric biopsy specimen is placed into a Campylobacter-like organism test kit and evaluated for up to 24 hours for Helicobacter spp. Stained slides (H&E) of gastric biopsy specimens are also evaluated for the presence of underlying inflammation[3].

References:

[1]. Favia AD, et al. Identification and characterization of carprofen as a multitarget fatty acid amide hydrolase/cyclooxygenase inhibitor. J Med Chem. 2012

Oct 25;55(20):8807-26.

[2]. Waldherr K, et al. In vitro cytoprotective effects of acetylsalicylic acid, carprofen, meloxicam, or robenacoxib against apoptosis induced by sodium nitroprusside in canine cruciate ligament cells. Am J Vet Res. 2012 Nov;73(11):1752-8.

[3]. Sessions JK, et al. In vivo effects of carprofen, deracoxib, and etodolac on prostanoid production in blood, gastric mucosa, and synovial fluid in dogs with chronic osteoarthritis. Am J Vet Res. 2005 May;66(5):812-7.

CAIndexNames:

9H-Carbazole-2-acetic acid, 6-chloro-α-methyl-

SMILES:

O=C(O)C(C)C1=CC(NC2=C3C=C(Cl)C=C2)=C3C=C1

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