Ferulic acid sodium

**Cat. No.:** HY-N0060A  
**CAS No.:** 24276-84-4  
**Molecular Formula:** $C_{10}H_9NaO_4$  
**Molecular Weight:** 216.17  
**Target:** 5-HT Receptor; Reactive Oxygen Species  
**Pathway:** GPCR/G Protein; Neuronal Signaling; Immunology/Inflammation; Metabolic Enzyme/Protease; NF-κB  
**Storage:**  
- Powder: -20°C 3 years, 4°C 2 years  
- In solvent: -80°C 6 months, -20°C 1 month

### SOLVENT & SOLUBILITY

**In Vitro**  
DMSO: 16.6 mg/mL (76.79 mM; Need ultrasonic and warming)

<table>
<thead>
<tr>
<th>Solvent Concentration</th>
<th>Mass (1 mg)</th>
<th>Mass (5 mg)</th>
<th>Mass (10 mg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 mM</td>
<td>4.6260 mL</td>
<td>23.1299 mL</td>
<td>46.2599 mL</td>
</tr>
<tr>
<td>5 mM</td>
<td>0.9252 mL</td>
<td>4.6260 mL</td>
<td>9.2520 mL</td>
</tr>
<tr>
<td>10 mM</td>
<td>0.4626 mL</td>
<td>2.3130 mL</td>
<td>4.6260 mL</td>
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</tbody>
</table>

Please refer to the solubility information to select the appropriate solvent.

### BIOLOGICAL ACTIVITY

**Description**
Ferulic acid (4-hydroxy-3-methoxycinnamic acid) is a phenolic compound present in several plants with claimed beneficial effects in prevention and treatment of disorders linked to oxidative stress and inflammation.

**IC50 value:**  
**Target:** 5-HT Receptor

In vitro: In the present study we have showed that pre-treatment with Ferulic Acid (FA) reduces NO accumulation in the culture medium of LPS-induced macrophage cells. Moreover, real-time experiments have revealed that FA has an inhibitory effect at the transcriptional level on the expression of some inflammatory mediators such as IL-6, TNF-α and iNOS and an activation effect on the expression of some antioxidant molecules such as Metallothioneins (MT-1, MT-2). Importantly, we have found that FA reduced the translocation of NF-E2-related factor 2 (Nrf2) and nuclear transcription factor-κB (NF-κB) into the nuclei through a reduction of the expression of phosphorylated IKK and consequently inhibited IL-6 and NF-κB promoter activity in a luciferase assay [1]. FA treatment significantly, although not completely, protected the cells against lead acetate-induced neurite outgrowth inhibition. The effects of FA could be blocked by PD98059, zinc protoporphyrin (Zn-PP), and Nrf2 shRNA. In addition, FA induced heme oxygenase 1 (HO-1) gene expression, enhanced antioxidant response element (ARE) promoter activity, promoted ERK1/2 phosphorylation, and Nrf2 translocation in PC12 cells exposed to lead acetate.
ERK1/2 locate upstream of Nrf2 and regulate Nrf2-dependent HO-1 expression in antioxidative effects of FA [2]. In vivo: We aimed to verify the possible antidepressant-like effect of acute oral administration of Ferulic acid produced an antidepressant-like effect in the FST and TST (0.01–10 mg/kg, p.o.), without accompanying changes in ambulation. The pretreatment of mice with WAY100635 (0.1 mg/kg, s.c., a selective 5-HT1A receptor antagonist) or ketanserin (5 mg/kg, i.p., a 5-HT2A receptor antagonist) was able to reverse the anti-immobility effect of ferulic acid (0.01 mg/kg, p.o.) in the TST. The combination of fluoxetine (5 mg/kg, p.o.), paroxetine (0.1 mg/kg, p.o.) or sertraline (1 mg/kg, p.o.) with a sub-effective dose of ferulic acid (0.001 mg/kg, p.o.) produced a synergistic antidepressant-like effect in the TST, without causing hyperlocomotion in the open-field test. ferulic acid in the forced swimming test (FST) and tail suspension test (TST) in mice [3].

REFERENCES

