# (S)-Coriolic acid

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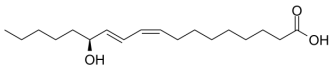
| **Cat. No.:** | HY-113884B |
| **CAS No.:** | 29623-28-7 |
| **Molecular Formula:** | $C_{18}H_{32}O_3$ |
| **Molecular Weight:** | 296.44 |
| **Target:** | PPAR; Mitochondrial Metabolism |
| **Pathway:** | Cell Cycle/DNA Damage; Vitamin D Related/Nuclear Receptor; Metabolic Enzyme/Protease |
| **Storage:** | Solution, -20°C, 2 years |




## BIOLOGICAL ACTIVITY

**Description**

(S)-Coriolic acid (13(S)-HODE), the product of 15-lipoxygenase (15-LOX) metabolism of linoleic acid, functions as the endogenous ligand to activate PPARγ. (S)-Coriolic acid is an important intracellular signal agent and is involved in cell proliferation and differentiation in various biological systems. (S)-Coriolic acid induces mitochondrial dysfunction and airway epithelial injury[1][2][3].

**In Vitro**

(S)-Coriolic acid (25μM) causes mitochondrial structural alterations and injury in bronchial epithelium[2].
(S)-Coriolic acid (30 nM; 6 hours; E-FABP$^{-/-}$ keratinocytes) induces K1 expression through NF-κB activation. (S)-Coriolic acid increases the phosphorylation of IκBαat serine 32, which induces IκB degradation and thereby activates NF-κB. (S)-Coriolic acid also increases the phosphorylation of Ikkinase-bat tyrosine 199, which promotes IκBα phosphorylation and subsequent NF-kB activation[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

**In Vivo**

(S)-Coriolic acid (0-0.6 mg per mouse; Intranasally once a day for 3 consecutive days) causes severe airway dysfunction, airway neutrophilia, mitochondrial dysfunction and epithelial injury[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

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| Animal Model: | BALB/c mice (6-8 weeks)[2] |
| Dosage: | 0-0.6 mg per mouse |
| Administration: | Intranasally once a day for 3 consecutive days |
| Result: | BALB/c mice developed features of mitochondrial dysfunction such as reduction in mitochondrial membrane potential, reduction in complex IV activity in lung mitochondria, and increase in the levels of cytochrome c in lung cytosol. |

## REFERENCES

[1]. Yuan H, et al. 15-Lipoxygenases and its metabolites 15(S)-HETE and 13(S)-HODE in the development of non-small cell lung cancer. Thorax. 2010;65(4):321-326.

[2]. Mabalirajan U, et al. Linoleic acid metabolite drives severe asthma by causing airway epithelial injury. Sci Rep. 2013;3:1349.

[3]. Ogawa E, et al. Epidermal FABP (FABP5) regulates keratinocyte differentiation by 13(S)-HODE-mediated activation of the NF-κB signaling pathway. J Invest Dermatol. 2011;131(3):604-612.



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