



## Montelukast (sodium)

**Catalog No: tcsc0548** 

<u></u>	Available Sizes
Size	s 50mg
Size	100mg
Size	s 500mg
	Specifications
<b>CAS</b> 1517	<b>No:</b> 67-02-1
Forn C <sub>35</sub> H	nula: <sub>35</sub> CINNaO <sub>3</sub> S
	<b>way:</b> k/G Protein;Autophagy
<b>Targ</b> Leuk	<b>et:</b> otriene Receptor;Autophagy
<b>Purit</b> >98%	gy / Grade:
	<b>bility:</b> : ≥ 70 mg/mL (115.10 mM)
<b>Alte</b> i MK04	rnative Names:

## **Product Description**

608.17

**Observed Molecular Weight:** 



Montelukast (sodium) is a potent, selective  $\mathbf{CysLT_1}$  receptor antagonist.

**In Vitro:** Montelukast may contribute to the reduction of eosinophilic inflammation in upper-airway inflammatory diseases such as rhinitis and nasal polyposis. Montelukast has a significant inhibitory effect on FBS-induced GM-CSF, IL-6, and IL-8 secretion, but not sICAM-1, in nasal mucosa and polyp epithelial cells. Montelukast also shows an inhibitory effect (p[1].

In Vivo: Montelukast significantly reduces mild, moderate, and part of severe exacerbations in chronic mild to moderate asthma, but it has inferior efficacy to ICS or ICS plus LABA<sup>[2]</sup>. Rats with induced asthma have up-regulated NK1R expression in the airway, and montelukast can down regulate NK1R expression during airway remodeling<sup>[3]</sup>. Blockade of CysLT<sub>1</sub>R by repeated treatment with montelukast (1 or 2 mg/kg, ig, 4 weeks) reduces  $A\beta_{1-42}$ -induced CysLT<sub>1</sub>R expression and also suppresses  $A\beta_{1-42}$ -induced increments of NF- $\kappa$ B p65, TNF- $\alpha$ , IL-1 $\beta$  and caspase-3 activation, and Bcl-2 downregulation in the hippocampus and cortex. Correspondingly, montelukast treatment significantly improves  $A\beta_{1-42}$ -induced memory impairment in mice, but has little effect on normal mice<sup>[4]</sup>.

All products are for RESEARCH USE ONLY. Not for diagnostic & therapeutic purposes!