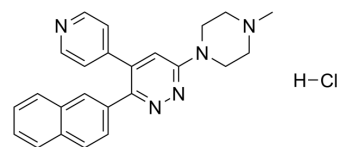


## MW-150 hydrochloride

<b>Cat. No.:</b>	HY-120111A
<b>CAS No.:</b>	1923773-01-6
<b>Molecular Formula:</b>	C <sub>24</sub> H <sub>24</sub> ClN <sub>5</sub>
<b>Molecular Weight:</b>	417.93
<b>Target:</b>	p38 MAPK; Autophagy
<b>Pathway:</b>	MAPK/ERK Pathway; Autophagy
<b>Storage:</b>	Please store the product under the recommended conditions in the Certificate of Analysis.



### BIOLOGICAL ACTIVITY

<b>Description</b>	MW-150 hydrochloride (MW01-18-150SRM hydrochloride) is a selective, CNS penetrant, and orally active inhibitor of p38 $\alpha$ MAPK with a K <sub>i</sub> of 101 nM. MW-150 hydrochloride (MW01-18-150SRM hydrochloride) inhibits the ability of the endogenous p38 $\alpha$ MAPK to phosphorylate an endogenous substrate MK2 in activated glia <sup>[1]</sup> .								
<b>IC<sub>50</sub> &amp; Target</b>	p38 $\alpha$ 101 nM (K <sub>i</sub> )								
<b>In Vitro</b>	MW-150 hydrochloride (MW01-18-150SRM hydrochloride) inhibits in a concentration-dependent manner the ability of the endogenous p38 $\alpha$ MAPK to phosphorylate an endogenous substrate MK2 in activated glia <sup>[1]</sup> . MW-150 hydrochloride (MW01-18-150SRM hydrochloride) blocks in a concentration-dependent manner the increased IL-1 $\beta$ production by activated glia. The IC <sub>50</sub> values are 332 nM and 936 nM for MK2 and IL-1 $\beta$ , respectively <sup>[1]</sup> . MCE has not independently confirmed the accuracy of these methods. They are for reference only.								
<b>In Vivo</b>	MW-150 hydrochloride (MW01-18-150SRM hydrochloride) (2.5 mg/kg; oral daily for 3–4 months) improves the APP/PS1 transgenic (Tg) mice performance in radial arm water maze (RAWM) and contextual fear conditioning tests <sup>[1]</sup> . MW-150 hydrochloride (MW01-18-150SRM hydrochloride) (2.5 mg/kg; given i.p.; daily for 14 days) treatment in APP <sup>NLh/NLh</sup> × PS <sup>P264L/P264L</sup> knock-in mouse (with no overexpression of the amyloid precursor protein) exhibits RAWM behavior indistinguishable from WT mice <sup>[1]</sup> . MCE has not independently confirmed the accuracy of these methods. They are for reference only.								
	<table border="1"> <tr> <td>Animal Model:</td> <td>APP/PS1 transgenic (Tg) mouse (overexpresses amyloid-beta) <sup>[1]</sup></td> </tr> <tr> <td>Dosage:</td> <td>2.5 mg/kg</td> </tr> <tr> <td>Administration:</td> <td>Oral daily; 3-4 months (until cognitive impairment is present)</td> </tr> <tr> <td>Result:</td> <td>Improved the Tg mice performance in both cognitive tests.</td> </tr> </table>	Animal Model:	APP/PS1 transgenic (Tg) mouse (overexpresses amyloid-beta) <sup>[1]</sup>	Dosage:	2.5 mg/kg	Administration:	Oral daily; 3-4 months (until cognitive impairment is present)	Result:	Improved the Tg mice performance in both cognitive tests.
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Result:	Improved the Tg mice performance in both cognitive tests.								

### REFERENCES

[1]. Roy SM, et al. Targeting human central nervous system protein kinases: An isoform selective p38 $\alpha$ MAPK inhibitor that attenuates disease progression in Alzheimer's

**Caution: Product has not been fully validated for medical applications. For research use only.**

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