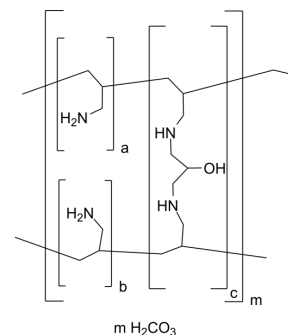


Sevelamer carbonate

Cat. No.:	HY-13995B
CAS No.:	845273-93-0
Molecular Formula:	$(C_3H_7N.C_3H_5ClO)_x.xH_2CO_3$
Target:	Others
Pathway:	Others
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	Sevelamer carbonate is an orally active and non-calcium-based phosphate binding agent and used for the hyperphosphatemia of chronic kidney disease (CKD) research. Sevelamer carbonate effectively lowers serum phosphorus levels while having minimal effect on serum calcium or serum chloride levels in vivo. Sevelamer carbonate is considered as an improved, buffered form of sevelamer (HY-13995) ^{[1][2]} .								
In Vitro	Sevelamer carbonate (15 mg/mL; pH=6 or 8) decreases serum levels of gut-derived uremic toxins (such as IAA) or limits the elevation of gut-derived uremic toxins (initial concentration=1 µg/mL or 10 µg/mL) ^[5] . MCE has not independently confirmed the accuracy of these methods. They are for reference only.								
In Vivo	<p>Sevelamer carbonate is an anion-exchange resin with the same polymeric structure as that of sevelamer hydrochloride, but with carbonate replacing chloride as the anion^[4].</p> <p>Sevelamer carbonate lowers serum phosphorus levels and calcium-phosphorus product to a similar extent as sevelamer hydrochloride. Additionally, Sevelamer carbonate is associated with significant effects on decreasing low-density lipoprotein cholesterol levels and may cause less metabolic acidosis than sevelamer hydrochloride in vivo^[1].</p> <p>Sevelamer carbonate (oral administration; 1% mixed in diet; 2-3 weeks) significantly reduces serum phosphate level in Npt2b-deficient mice. Npt2b attenuates the hyperphosphatemia in control animals and that sevelamer carbonate treatment has an additional benefit in maintaining serum phosphate in the normal range^[3].</p> <p>Sevelamer carbonate (oral administration; 1% mixed in diet; 2-3 weeks) does not alter serum phosphate levels in uremic WT mice (10.04 mg/dl, untreated versus 9.67mg/dl, binder-treated mg/dl) but further decreased serum phosphate levels in uremic Npt2b^{-/-} mice in uremic mouse model^[3].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p> <table border="1"> <tr> <td>Animal Model:</td> <td>WT and Npt2b^{-/-} CKD mice model^[3]</td> </tr> <tr> <td>Dosage:</td> <td>1% mixed in diet</td> </tr> <tr> <td>Administration:</td> <td>Oral administration; 1% mixed in diet; 2-3 weeks</td> </tr> <tr> <td>Result:</td> <td>Attenuated chronic hyperphosphatemia in mice.</td> </tr> </table>	Animal Model:	WT and Npt2b ^{-/-} CKD mice model ^[3]	Dosage:	1% mixed in diet	Administration:	Oral administration; 1% mixed in diet; 2-3 weeks	Result:	Attenuated chronic hyperphosphatemia in mice.
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REFERENCES

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- [1]. Bennis Y, et al. The Effect of Sevelamer on Serum Levels of Gut-Derived Uremic Toxins: Results from In Vitro Experiments and A Multicenter, Double-Blind, Placebo-Controlled, Randomized Clinical Trial. *Toxins (Basel)*. 2019 May 17;11(5):279.
- [2]. Mary M Barna, et al. Sevelamer carbonate. *Ann Pharmacother*. 2010 Jan;44(1):127-34.
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- [4]. Susan C Schiavi, et al. Npt2b deletion attenuates hyperphosphatemia associated with CKD. *J Am Soc Nephrol*. 2012 Oct;23(10):1691-700.
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Caution: Product has not been fully validated for medical applications. For research use only.

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