



# SZABO SCANDIC

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Diagnostik & molekulare Diagnostik



Laborgeräte & Service

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### SZABO-SCANDIC HandelsgmbH

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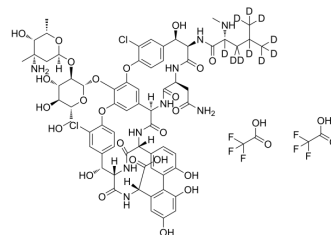
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## Vancomycin-d<sub>10</sub> TFA

<b>Cat. No.:</b>	HY-B0671S3
<b>Molecular Formula:</b>	C <sub>70</sub> H <sub>67</sub> D <sub>10</sub> Cl <sub>2</sub> F <sub>6</sub> N <sub>9</sub> O <sub>28</sub>
<b>Molecular Weight:</b>	1687.36
<b>Target:</b>	Antibiotic; Bacterial; Autophagy; Isotope-Labeled Compounds
<b>Pathway:</b>	Anti-infection; Autophagy; Others
<b>Storage:</b>	Please store the product under the recommended conditions in the Certificate of Analysis.



### BIOLOGICAL ACTIVITY

<b>Description</b>	Vancomycin-d10 (TFA) is a deuterated labeled Vancomycin <sup>[1]</sup> . Vancomycin is an antibiotic for the treatment of bacterial infections.
<b>In Vitro</b>	<p>Stable heavy isotopes of hydrogen, carbon, and other elements have been incorporated into drug molecules, largely as tracers for quantitation during the drug development process. Deuteration has gained attention because of its potential to affect the pharmacokinetic and metabolic profiles of drugs<sup>[1]</sup>.</p> <p>Vancomycin is a large glycopeptide compound with a molecular weight of 1450 Da<sup>[2]</sup>. Vancomycin is a unique glycopeptide structurally unrelated to any currently available antibiotic. It also has a unique mode of action inhibiting the second stage of cell wall synthesis of susceptible bacteria. Vancomycin is active against a large number of species of Gram-positive bacteria, such as <i>Staphylococcus aureus</i>, <i>Staph. epidermidis</i>, <i>Str. agalactiae</i>, <i>Str. bovis</i>, <i>Str. mutans</i>, <i>viridans streptococci</i>, <i>enterococci</i><sup>[3]</sup>.</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>
<b>In Vivo</b>	<p>Vancomycin is a large glycopeptide compound with a molecular weight of 1450 Da<sup>[2]</sup>. Vancomycin is a unique glycopeptide structurally unrelated to any currently available antibiotic. It also has a unique mode of action inhibiting the second stage of cell wall synthesis of susceptible bacteria. Vancomycin is active against a large number of species of Gram-positive bacteria, such as <i>Staphylococcus aureus</i>, <i>Staph. epidermidis</i>, <i>Str. agalactiae</i>, <i>Str. bovis</i>, <i>Str. mutans</i>, <i>viridans streptococci</i>, <i>enterococci</i><sup>[3]</sup>.</p> <p>Vancomycin can be used in animal modeling to construct animal kidney injury models. Vancomycin is administered intravenously, with a standard infusion time of at least 1 h, to minimize infusion-related adverse effects. Subjects with normal creatinine clearance, vancomycin has an <math>\alpha</math>-distribution phase of 30 min to 1 h and a <math>\beta</math>-elimination half-life of 6-12 h. The volume of distribution is 0.4–1 L/kg. The binding of vancomycin to protein ranges from 10% to 50%. Factors that affect the overall activity of vancomycin include its tissue distribution, inoculum size, and protein-binding effects<sup>[2]</sup>. Vancomycin treatment of infected mice is associated with improved clinical, diarrhea, and histopathology scores and survival during treatment<sup>[4]</sup>. Vancomycin is a classic kidney injury modeling agent that induces disease by inducing oxidative stress-related apoptosis in animals. Rats and mice are generally used as animal models<sup>[5][6]</sup>. Dose reference for vancomycin induction<sup>[5][6]</sup>:</p> <p>(1) Model animal: C57BL/6J male mice (6-week)        VIKI: 400 mg/kg/day, i.p, 7 day</p> <p>(2) Model animals: Male Sprague-Dawley (SD) rats (200-250 g)        VIKI: 400 mg/kg/day, i.p, 7 day</p>

## Induction of kidney Injury Model<sup>[5]</sup>

### Background

Vancomycin induces oxidative stress-related apoptosis in animals.

### Specific Modeling Methods

Mice: C57BL/6 • male • 6-week-old

Administration: 400 mg/kg • ip • once daily for 7 weeks

### Note

### Modeling Indicators

Molecular changes: Induced cell apoptosis and kidney Cr, BUN, MDA, IL-1 $\beta$ , IL-6, TNF- $\alpha$ , and NF- $\kappa$ B increase.

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## REFERENCES

- [1]. Rybak MJ, et al. The pharmacokinetic and pharmacodynamic properties of vancomycin. *Clin Infect Dis*. 2006 Jan 1;42 Suppl 1:S35-9.
- [2]. Juan He, et al. Vitamin C reduces vancomycin-related nephrotoxicity through the inhibition of oxidative stress, apoptosis, and inflammation in mice. *Ann Transl Med*. 2021 Aug; 9(16): 1319.
- [3]. Watanakunakorn C, et al. Mode of action and in-vitro activity of vancomycin. *J Antimicrob Chemother*. 1984 Dec;14 Suppl D:7-18.
- [4]. Ping Yu, et al. N-acetylcysteine Ameliorates Vancomycin-induced Nephrotoxicity by Inhibiting Oxidative Stress and Apoptosis in the in vivo and in vitro Models. *Int J Med Sci*. 2022; 19(4): 740–752.
- [5]. Warren CA, et al. Vancomycin treatment's association with delayed intestinal tissue injury, clostridial overgrowth, and recurrence of *Clostridium difficile* infection in mice. *Antimicrob Agents Chemother*. 2013 Feb;57(2):689-96.
- [6]. Russak EM, et al. Impact of Deuterium Substitution on the Pharmacokinetics of Pharmaceuticals. *Ann Pharmacother*. 2019 Feb;53(2):211-216.

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

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