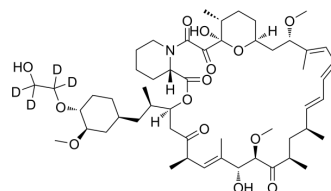


## Everolimus-d<sub>4</sub>

<b>Cat. No.:</b>	HY-10218S
<b>CAS No.:</b>	1338452-54-2
<b>Molecular Formula:</b>	C <sub>53</sub> H <sub>79</sub> D <sub>4</sub> NO <sub>14</sub>
<b>Molecular Weight:</b>	962.25
<b>Target:</b>	Apoptosis; mTOR; Autophagy; FKBP
<b>Pathway:</b>	Apoptosis; PI3K/Akt/mTOR; Autophagy; Immunology/Inflammation
<b>Storage:</b>	Please store the product under the recommended conditions in the Certificate of Analysis.



### BIOLOGICAL ACTIVITY

<b>Description</b>	Everolimus-d <sub>4</sub> is the deuterium labeled Everolimus. Everolimus (RAD001) is a Rapamycin derivative and a potent, selective and orally active mTOR1 inhibitor. Everolimus binds to FKBP-12 to generate an immunosuppressive complex. Everolimus inhibits tumor cells proliferation and induces cell apoptosis and autophagy. Everolimus has potent immunosuppressive and anticancer activities[1][2].
<b>In Vitro</b>	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> . MCE has not independently confirmed the accuracy of these methods. They are for reference only.

### REFERENCES

- [1]. Russak EM, et al. Impact of Deuterium Substitution on the Pharmacokinetics of Pharmaceuticals. *Ann Pharmacother.* 2019;53(2):211-216.
- [2]. Lane HA, et al. mTOR inhibitor RAD001 (everolimus) has antiangiogenic/vascular properties distinct from a VEGFR tyrosine kinase inhibitor. *Clin Cancer Res*, 2009, 15(5), 1612-1622.
- [3]. O'Reilly T, et al. Biomarker Development for the Clinical Activity of the mTOR Inhibitor Everolimus (RAD001): Processes, Limitations, and Further Proposals. *Transl Oncol.* 2010 Apr;3(2):65-79.
- [4]. Zhu Y, et al. Antitumor effect of the mTOR inhibitor Everolimus on human breast cancer stem cells in vitro and in vivo. *Tumour Biol.* 2012 Oct;33(5):1349-62.
- [5]. Kawata T, et al. Dual inhibition of the mTORC1 and mTORC2 signaling pathways is a promising therapeutic target for adult T-cell leukemia. *Cancer Sci.* 2018 Jan;109(1):103-111.

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

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