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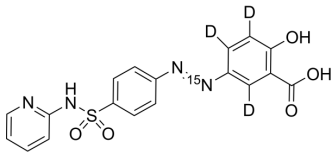
Sulfasalazine-d_3,^{15}N

Cat. No.:	HY-14655S1
Molecular Formula:	$C_{18}H_{11}D_3N_3{}^{15}NO_5S$
Molecular Weight:	402.4
Target:	Antibiotic; Autophagy; NF-κB; Apoptosis; Bacterial; Ferroptosis; Isotope-Labeled Compounds
Pathway:	Anti-infection; Autophagy; NF-κB; Apoptosis; Others
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	Sulfasalazine-d_3,^{15}N is ^{15}N and deuterated labeled Sulfasalazine (HY-14655). Sulfasalazine (NSC 667219) is an anti-rheumatic agent for the research of rheumatoid arthritis and ulcerative colitis. Sulfasalazine can suppress NF-κB activity. Sulfasalazine is a type 1 ferroptosis inducer[1][2][3][4].
In Vitro	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[1]. Treatment of SW620 colon cells with sulfasalazine inhibits TNFα-, LPS-, or phorbol ester-induced NFκB activation. NFκB-dependent transcription is inhibited by sulfasalazine at micro- to millimolar concentrations. TNFα-induced nuclear translocation of NFκB is prevented by sulfasalazine through inhibition of IκBα degradation[2]. Pre-incubation with 5 mM of sulfasalazine alone significantly increases basal mRNA expression of all pro-inflammatory cytokines with levels of IL-6 mRNA increased by 80-fold compared with vehicle control[3]. Once digested, sulfasalazine is cleaved into sulfapyridine and 5-aminosalicylic acid by colonic bacteria, and the latter, too, is reported to suppress NF-kappaB activity[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	At low doses (0.25 mM), SAS is able to suppress glioma growth by over 60% compared to untreated controls[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Wahl C, et al. Sulfasalazine: a potent and specific inhibitor of nuclear factor kappa B. J Clin Invest. 1998 Mar 1;101(5):1163-74.

[2]. Mao C, et al. DHODH-mediated ferroptosis defence is a targetable vulnerability in cancer. Nature. 2021;593(7860):586-590.

[3]. Sykes L, et al. Sulfasalazine augments a pro-inflammatory response in interleukin-1β-stimulated amniocytes and myocytes. Immunology. 2015 Dec;146(4):630-44.

[4]. Chung WJ, et al. Sulfasalazine inhibits the growth of primary brain tumors independent of nuclear factor-kappaB. J Neurochem. 2009 Jul;110(1):182-93.

[5]. 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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