

Produktinformation



Forschungsprodukte & Biochemikalien



Zellkultur & Verbrauchsmaterial



Diagnostik & molekulare Diagnostik



Laborgeräte & Service

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Proteins

Guanfacine-15N13,13C2

Cat. No.: HY-17416AS1

Molecular Weight: 251.06

Molecular Formula:

Target: Adrenergic Receptor; Isotope-Labeled Compounds

GPCR/G Protein; Neuronal Signaling; Others Pathway:

 $C_7^{13}C_2H_9Cl_2^{15}N_3O$

Storage: Please store the product under the recommended conditions in the Certificate of

Analysis.

Product Data Sheet

BIOLOGICAL ACTIVITY

Description	Guanfacine- 15 N $_3$, 13 C $_2$ is 15 N and 13 C labeled Guanfacine (HY-17416A). Guanfacine is an orally active noradrenergic α 2A agonist and has high selective for the α 2A receptor subtype. Guanfacine has effects in producing hypotension and sedation. Guanfacine can be used for the research of a variety of prefrontal cortex (PFC) cognitive disorders, including tourette's syndrome and attention deficit hyperactivity disorder (ADHD)[1][2][3].
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] . Guanfacine increases the delay-related neuronal firing needed for working memory on dIPFC neurons at the cellular level ^[2] [4]. Guanfacine improves PFC cognitive function by inhibiting the production of CAMP, closing HCN channels, and strengthening the PFC networks ^{[2][4]} . MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Guanfacine enhances PFC working memory function in aged monkeys and improves impulse control in monkeys performing a delayed discounting task ^{[2][4]} . Guanfacine improves cognitive performance when infused directly into the rat or monkey PFC ^{[2][4]} . Guanfacine blocks 2A receptors in the monkey dIPFC markedly impairs working memory, behavioral inhibition and greatly reduces persistent neuronal firing ^{[2][4]} . MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

- [1]. Amy FT Arnsten, et al. Guanfacine for the treatment of cognitive disorders: a century of discoveries at Yale. Yale J Biol Med. 2012 Mar;85(1):45-58. Epub 2012 Mar 29.
- [2]. P. A. Van Zwieten, et al. The pharmacology of centrally acting antihypertensive drugs. Br J Clin Pharmacol. 1983; 15(Suppl 4): 455S-462S.
- [3]. Min Wang, et al. Alpha2A-adrenoceptors strengthen working memory networks by inhibiting cAMP-HCN channel signaling in prefrontal cortex. Cell. 2007 Apr 20;129(2):397-410.
- [4]. Russak EM, et al. Impact of Deuterium Substitution on the Pharmacokinetics of Pharmaceuticals. Ann Pharmacother. 2019 Feb;53(2):211-216.

 $\label{lem:caution:Product} \textbf{Caution: Product has not been fully validated for medical applications. For research use only.}$

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