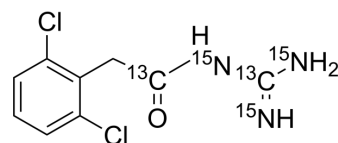


Guanfacine-¹⁵N¹³, ¹³C₂

Cat. No.:	HY-17416AS1
Molecular Formula:	C ₇ ¹³ C ₂ H ₉ Cl ₂ ¹⁵ N ₃ O
Molecular Weight:	251.06
Target:	Adrenergic Receptor; Isotope-Labeled Compounds
Pathway:	GPCR/G Protein; Neuronal Signaling; Others
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



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 ₂ A agonist and has high selective for the α ₂ A 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	<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^[1].</p> <p>Guanfacine increases the delay-related neuronal firing needed for working memory on dIPFC neurons at the cellular level^[2] [4].</p> <p>Guanfacine improves PFC cognitive function by inhibiting the production of cAMP, closing HCN channels, and strengthening the PFC networks^{[2][4]}.</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>
In Vivo	<p>Guanfacine enhances PFC working memory function in aged monkeys and improves impulse control in monkeys performing a delayed discounting task^{[2][4]}.</p> <p>Guanfacine improves cognitive performance when infused directly into the rat or monkey PFC^{[2][4]}.</p> <p>Guanfacine blocks 2A receptors in the monkey dIPFC markedly impairs working memory, behavioral inhibition and greatly reduces persistent neuronal firing^{[2][4]}.</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>

REFERENCES

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- [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.

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

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