

# CHRNA7 Antibody

Purified Rabbit Polyclonal Antibody (Pab)

Catalog # AP51984

## Product Information

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Application	WB
Primary Accession	<a href="#">P36544</a>
Reactivity	Human, Mouse, Rat
Host	Rabbit
Clonality	Polyclonal
Calculated MW	56449

## Additional Information

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Gene ID	1139;89832
Other Names	Neuronal acetylcholine receptor subunit alpha-7, CHRNA7, NACHRA7
Target/Specificity	KLH-conjugated synthetic peptide encompassing a sequence within the center region of human CHRNA7. The exact sequence is proprietary.
Dilution	WB~~1:1000
Format	0.01M PBS, pH 7.2, 0.09% (W/V) Sodium azide, Glycerol 50%
Storage	Store at -20 °C.Stable for 12 months from date of receipt

## Protein Information

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Name	CHRNA7 ( <a href="#">HGNC:1960</a> )
Synonyms	NACHRA7
Function	<p>Component of neuronal acetylcholine receptors (nAChRs) that function as pentameric, ligand-gated cation channels with high calcium permeability among other activities. nAChRs are excitatory neurotransmitter receptors formed by a collection of nAChR subunits known to mediate synaptic transmission in the nervous system and the neuromuscular junction. Each nAChR subunit confers differential attributes to channel properties, including activation, deactivation and desensitization kinetics, pH sensitivity, cation permeability, and binding to allosteric modulators (PubMed:<a href="#">15609996</a>, PubMed:<a href="#">33735609</a>, PubMed:<a href="#">8145738</a>). CHRNA7 forms homopentameric neuronal acetylcholine receptors abundantly expressed in the central nervous system, characterized by fast desensitization and high calcium permeability (PubMed:<a href="#">31560909</a>, PubMed:<a href="#">33735609</a>, PubMed:<a href="#">38382524</a>, PubMed:<a href="#">8145738</a>). Also forms heteropentamers with CHRNB2, mainly expressed in basal forebrain cholinergic neurons. Involved in the modulation</p>

of calcium- dependent signaling pathways and influences the release of neurotransmitters, including dopamine, glutamate and GABA (PubMed:[33239400](#)). Also expressed in non-neuronal cells such as immune cells like lymphocytes, monocytes and macrophages (PubMed:[12508119](#), PubMed:[16968406](#), PubMed:[25259522](#)). In T cells, activation induces metabotropic signaling that results in an increase of intracellular Ca<sup>2+</sup> concentrations, independent of ionotropic receptor functions (PubMed:[17709503](#)). In macrophages, required for acetylcholine-mediated inhibition of TNF and other inflammatory cytokine release (PubMed:[12508119](#)). Once activated by acetylcholine, nicotine or other agonists, selectively inhibits production of pro-inflammatory cytokines while leaving anti-inflammatory cytokines undisturbed (PubMed:[12508119](#), PubMed:[25259522](#)). Stimulates the cholinergic anti-inflammatory pathway, controlling inflammation by inhibiting NFκB nuclear translocation and activating the JAK2-STAT3 pathway, independently of ion channel activity (PubMed:[16968406](#), PubMed:[25259522](#)). Also expressed in the urothelium where it modulates reflex bladder activity by increasing intracellular calcium through internal stores and decreasing basal ATP release (By similarity).

#### Cellular Location

Postsynaptic cell membrane {ECO:0000250|UniProtKB:Q05941}; Multi-pass membrane protein. Cell membrane; Multi-pass membrane protein. Note=TMEM35A/NACHO promotes its trafficking to the cell membrane (PubMed:27789755). RIC3 promotes its trafficking to the cell membrane (By similarity) {ECO:0000250|UniProtKB:Q05941, ECO:0000269|PubMed:27789755}

#### Tissue Location

Expressed in neuronal cells (PubMed:8145738). Expressed in macrophages (at protein level) (PubMed:12508119)

## Background

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After binding acetylcholine, the AChR responds by an extensive change in conformation that affects all subunits and leads to opening of an ion-conducting channel across the plasma membrane. The channel is blocked by alpha-bungarotoxin.

## References

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- Peng X.,et al.Mol. Pharmacol. 45:546-554(1994).  
 Logel J.,et al.Submitted (DEC-1995) to the EMBL/GenBank/DDBJ databases.  
 Elliott K.J.,et al.J. Mol. Neurosci. 7:217-228(1996).  
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