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Product Information

Anti-Phospholipase A2 (iPLA2) (C-terminal region)
produced in rabbit, affinity isolated antibody

Product Number **SAB4200130**

Product Description

Anti-Phospholipase A_2 (iPLA$_2$) (C-terminal region) is produced in rabbit using as the immunogen a synthetic peptide corresponding to a sequence at the C-terminal of human iPLA$_2$ (GeneID 8398), conjugated to KLH. The corresponding sequence is highly conserved in mouse iPLA$_2$ (83% identity) and in rat iPLA$_2$ (72% identity). The antibody is affinity-purified using the immunizing peptide immobilized on agarose.

Anti-Phospholipase A_2 (iPLA$_2$) (C-terminal region), specifically recognizes human and rat iPLA$_2$. The antibody can be used in several immunochemical techniques including immunoblotting (~85 kDa human iPLA2, and ~95 kDa rat iPLA2). Detection of the iPLA$_2$ bands by immunoblotting is specifically inhibited by the iPLA$_2$ immunizing peptide.

Ca^{2+}-independent phospholipase A_2 (iPLA$_2$, also known as PLA2G6, INAD1, PARK14, PNPLA9) is a member of the PLA$_2$ superfamily that catalyzes the cleavage of fatty acids from the *sn-2* position of phospholipids.[1,2] PLA$_2$ isoenzymes vary in their cellular localizations, Ca^{2+} sensitivities, and substrate specificities. They share the ability to catalyze the synthesis of precursors of proinflammatory mediators such as prostaglandins and leukotrienes through the release of arachidonic acid (AA) from membrane phospholipids.

PLA$_2$s play crucial roles in several cellular processes, including intracellular membrane trafficking, differentiation, proliferation, and apoptosis. They are thought to play a role in oxidative and inflammatory responses in cerebral ischemia, Alzheimer's disease (AD), and neuronal injury.[3]

iPLA$_2$ group VIA comprises at least 5 alternatively spliced isoforms. Isoforms LH-iPLA2 (90 kDa), and SH-iPLA2 (85 kDa) iPLA$_2$ have been implicated in phospholipid remodeling, nitric oxide-induced or vasopressin-induced arachidonic acid release, and in leukotriene and prostaglandin production. Mutations in the PLA2G6 gene are the cause of two childhood neurologic disorders, neurodegeneration with brain iron accumulation (NBIA) and infantile neuroaxonal dystrophy 1 (INAD1).[4,5] Recent evidence suggests that both cPLA$_2$ and iPLA$_2$ may play a central role in memory deficits at early stages of AD and in the AD neurodegenerative process.[6]

Reagent

Supplied as a solution in 0.01 M phosphate buffered saline, pH 7.4, containing 15 mM sodium azide.

Antibody concentration: ~1.5 mg/mL

Precautions and Disclaimer

For R&D use only. Not for drug, household, or other uses. Please consult the Safety Data Sheet for information regarding hazards and safe handling practices.

Storage/Stability

Store at −20 °C. For continuous use, the product may be stored at 2−8 °C for up to one month. For extended storage, freeze at −20 °C in working aliquots. Repeated freezing and thawing, or storage in "frost-free" freezers, is not recommended. If slight turbidity occurs upon prolonged storage, clarify the solution by centrifugation. Discard working dilutions if not used within 12 hours.

Product Profile

Immunoblotting: a working antibody concentration of 1-2 μg/mL is recommended using extract of HEK-293T cells overexpressing human iPLA$_2$, and 1-2 μg/mL using rat kidney extract (S1 fraction).

Note: In order to obtain best results in various techniques and preparations, it is recommended to determine optimal working dilutions by titration.

References

1. Balsinde, J., and Balboa, M.A., *Cell. Signal*., **17**, 1052-1062 (2005).
2. Pontus, K.A., et al., *Eur. J. Biochem.,* **262**, 575-585 (1999).
3. Sun, G.Y., et al., *J. Lipid Res.*, **45**, 205-213 (2004).
4. Morgan, N.V., et al., *Nat. Genet.*, **38**, 752-754 (2006).
5. Gregory, A., et al., *Neurol.,* **71**, 1402-1409 (2008).
6. Schaeffer, E.L., and Gattaz, W.F., *Psychopharmacol.,* **198**, 1-27 (2008).

VS,ER,KAA,PHC,MAM 07/19-1