

PTGS2 Antibody (Center H193) Blocking peptide
Synthetic peptide
Catalog # BP14091c**Specification**

PTGS2 Antibody (Center H193) Blocking peptide - Product InformationPrimary Accession [P35354](#)**PTGS2 Antibody (Center H193) Blocking peptide - Additional Information**

Gene ID 5743

Other Names

Prostaglandin G/H synthase 2, Cyclooxygenase-2, COX-2, PHS II, Prostaglandin H2 synthase 2, PGH synthase 2, PGHS-2, Prostaglandin-endoperoxide synthase 2, PTGS2, COX2

Target/Specificity

The synthetic peptide sequence used to generate the antibody AP14091c was selected from the Center H193 region of PTGS2. A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

Format

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

PTGS2 Antibody (Center H193) Blocking peptide - Protein InformationName PTGS2 ([HGNC:9605](#))**Function**

Dual cyclooxygenase and peroxidase in the biosynthesis pathway of prostanoids, a class of C20 oxylipins mainly derived from arachidonate ((5Z,8Z,11Z,14Z)-eicosatetraenoate, AA, C20:4(n-6)), with a particular role in the inflammatory response (PubMed:7947975, PubMed:7592599, PubMed:9261177, PubMed:16373578, PubMed:22942274, PubMed:26859324, PubMed:27226593, PubMed:11939906, PubMed:19540099). The

cyclooxygenase activity oxygenates AA to the hydroperoxy endoperoxide prostaglandin G₂ (PGG₂), and the peroxidase activity reduces PGG₂ to the hydroxy endoperoxide prostaglandin H₂ (PGH₂), the precursor of all 2-series prostaglandins and thromboxanes (PubMed:7947975, PubMed:7592599, PubMed:9261177, PubMed:16373578, PubMed:22942274, PubMed:26859324, PubMed:27226593). This complex transformation is initiated by abstraction of hydrogen at carbon 13 (with S-stereochemistry), followed by insertion of molecular O₂ to form the endoperoxide bridge between carbon 9 and 11 that defines prostaglandins. The insertion of a second molecule of O₂ (bis-oxygenase activity) yields a hydroperoxy group in PGG₂ that is then reduced to PGH₂ by two electrons (PubMed:7947975, PubMed:7592599, PubMed:9261177, PubMed:16373578, PubMed:22942274, PubMed:26859324, PubMed:27226593). Similarly catalyzes successive cyclooxygenation and peroxidation of dihomo-gamma-linoleate (DGLA, C20:3(n-6)) and eicosapentaenoate (EPA, C20:5(n-3)) to corresponding PGH₁ and PGH₃, the precursors of 1- and 3- series prostaglandins (PubMed:11939906, PubMed:19540099). In an alternative pathway of prostanoid biosynthesis, converts 2-arachidonoyl lysophospholipids to prostanoid lysophospholipids, which are then hydrolyzed by intracellular phospholipases to release free prostanoids (PubMed:27642067). Metabolizes 2-arachidonoyl glycerol yielding the glyceryl ester of PGH₂, a process that can contribute to pain response (PubMed:22942274). Generates lipid mediators from n-3 and n-6 polyunsaturated fatty acids (PUFAs) via a lipoxygenase-type mechanism. Oxygenates PUFAs to hydroperoxy compounds and then reduces them to corresponding alcohols (PubMed:11034610, PubMed:11192938, PubMed:9048568, PubMed:9261177). Plays a role in the generation of resolution phase interaction products (resolvins) during both sterile and infectious inflammation (PubMed:12391014). Metabolizes docosahexaenoate (DHA, C22:6(n-3)) to 17R-HDHA, a precursor of the D- series resolvins (RvDs) (PubMed:12391014). As a component of the biosynthetic pathway of E-series resolvins (RvEs), converts eicosapentaenoate (EPA, C20:5(n-3)) primarily to 18S-HEPE that is further metabolized by ALOX5 and LTA4H to generate 18S-RvE1 and 18S- RvE2 (PubMed:21206090). In vascular endothelial cells, converts docosapentaenoate (DPA, C22:5(n-3)) to 13R-HDPA, a precursor for 13-series resolvins (RvTs) shown to activate macrophage phagocytosis during bacterial infection (PubMed:26236990). In activated leukocytes, contributes to oxygenation of hydroxyeicosatetraenoates (HETE) to diHETES (5,15-diHETE and 5,11-diHETE) (PubMed:22068350, PubMed:26282205). Can also use linoleate (LA, (9Z,12Z)- octadecadienoate, C18:2(n-6)) as substrate and produce hydroxyoctadecadienoates (HODEs) in a regio- and stereospecific manner, being (9R)-HODE ((9R)-hydroxy-(10E,12Z)-octadecadienoate) and (13S)- HODE

((13S)-hydroxy-(9Z,11E)-octadecadienoate) its major products (By similarity). During neuroinflammation, plays a role in neuronal secretion of specialized preresolving mediators (SPMs) 15R-lipoxin A4 that regulates phagocytic microglia (By similarity).

Cellular Location

Microsome membrane; Peripheral membrane protein. Endoplasmic reticulum membrane; Peripheral membrane protein. Nucleus inner membrane; Peripheral membrane protein. Nucleus outer membrane; Peripheral membrane protein. Note=Detected on the luminal side of the endoplasmic reticulum and nuclear envelope

PTGS2 Antibody (Center H193) Blocking peptide - Protocols

Provided below are standard protocols that you may find useful for product applications.

- [Blocking Peptides](#)

PTGS2 Antibody (Center H193) Blocking peptide - Images**PTGS2 Antibody (Center H193) Blocking peptide - Background**

Prostaglandin-endoperoxide synthase (PTGS), also known as cyclooxygenase, is the key enzyme in prostaglandin biosynthesis, and acts both as a dioxygenase and as a peroxidase. There are two isozymes of PTGS: a constitutive PTGS1 and an inducible PTGS2, which differ in their regulation of expression and tissue distribution. This gene encodes the inducible isozyme. It is regulated by specific stimulatory events, suggesting that it is responsible for the prostanoid biosynthesis involved in inflammation and mitogenesis.

PTGS2 Antibody (Center H193) Blocking peptide - References

Duggan, K.C., et al. J. Biol. Chem. 285(45):34950-34959(2010) Feher, A., et al. Am J Geriatr Psychiatry 18(11):983-987(2010) Wang, C.H., et al. Anticancer Res. 30(9):3649-3653(2010) Han, E.H., et al. J. Toxicol. Environ. Health Part A 73 (21-22), 1451-1464 (2010) :Cao, H., et al. Tohoku J. Exp. Med. 222(1):15-21(2010)