

PTGS1 (S585) polyclonal antibody

Catalog: BS1075

Host: Rabbit

Reactivity: Human, Mouse, Rat

BackGround:

Cyclooxygenase1 (Cox1) and cyclooxygenase2 (Cox2), family members with 60% homology in humans, catalyze prostaglandin production from arachidonic acid. While Cox1 expression is constitutive in most tissues, Cox2 expression is induced by lipopolysaccharide (LPS) and peptidoglycan (PGN). PGN activates Ras, leading to phosphorylation of Raf at Ser338 and Erk1/2 at Tyr204. The activation of MAP kinase signaling results in subsequent activation of IKK α/β , phosphorylation of I κ B α at Ser32/36, and NF- κ B activation. Finally, activation of the transcription factor NF- κ B is responsible for the induction of Cox2 expression. Investigators have shown that LPS and PGN induce the clinical manifestations of arthritis and bacterial infections, such as inflammation, fever, and septic shock. Research studies have indicated that Cox1 and Cox2 may also play a role in the neuropathology of Alzheimer's disease by potentiating γ -secretase activity and β -amyloid generation.

Product:

Rabbit IgG, 1mg/ml in PBS with 0.02% sodium azide, 50% glycerol, pH7.2

Molecular Weight:

~ 70 kDa

Swiss-Prot:

P23219

Purification&Purity:

The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen and the purity is > 95% (by SDS-PAGE).

Applications:

WB: 1:500~1:1000

IHC: 1:50~1:200

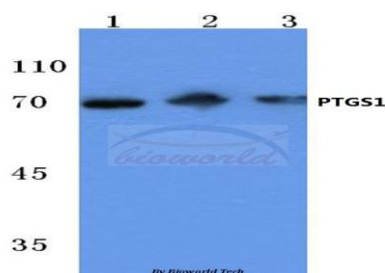
Storage&Stability:

Store at 4 °C short term. Aliquot and store at -20 °C long term. Avoid freeze-thaw cycles.

Specificity:

PTGS1 (S585) polyclonal antibody detects endogenous levels of PTGS1 protein.

DATA:

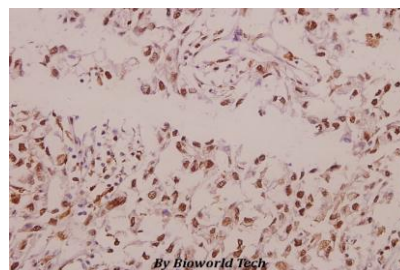


Western blot (WB) analysis of PTGS1 (S585) polyclonal antibody at 1:500 dilution

Lane1:Hela cell lysate

Lane2:Raw264.7 cell lysate

Lane3:Rat kidney tissue lysate



Immunohistochemistry (IHC) analyzes of PTGS1 (S585) pAb in paraffin-embedded human colorectal carcinoma tissue at 1:50.

Note:

For research use only, not for use in diagnostic procedure.

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