Recombinant Human PTGS2/COX2/PGHS-2 Protein (His Tag)

Catalog Number: PKSH030929

Note: *Centrifuge before opening to ensure complete recovery of vial contents.*

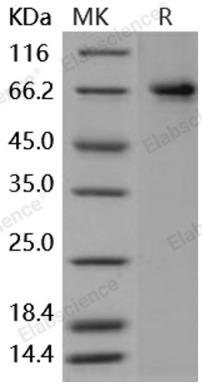
Description

Species	Human
Source	Baculovirus-Insect Cells-derived Human PTGS2/COX2/PGHS-2 protein Met 1-Leu 604, with an C-terminal His
Calculated MW	68.5 kDa
Observed MW	66 kDa
Accession	NP_000954.1
Bio-activity	Not validated for activity

Properties

Purity	> 95 % as determined by reducing SDS-PAGE.
Endotoxin	< 1.0 EU per μg of the protein as determined by the LAL method.
Storage	Store at < -20°C, stable for 6 months. Please minimize freeze-thaw cycles.
Shipping	This product is provided as liquid. It is shipped at frozen temperature with blue ice/ gel packs. Upon receipt, store it immediately at < - 20°C.
Formulation	Supplied as sterile 50 mM Tris, 100 mM NaCl, 10% glycerol, pH8.0.

Data



> 95 % as determined by reducing SDS-PAGE.

Background

PTGS2, also known as COX-2, is s component of Prostaglandin-endoperoxide synthase (PTGS). 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. PTGS2 is over expressed in many cancers. The overexpression of PTGS2 along with increased angiogenesis and GLUT-1 expression is significantly associated with gallbladder carcinomas. Furthermore the product of COX-2, PGH2 is converted by prostaglandin E2 synthase into PGE2, which in turn can stimulate cancer progression. Consequently inhibiting COX-2 may have benefit in the prevention and treatment of these types of cancer. PTGS2 is regulated by specific stimulatory events, suggesting that it is responsible for the prostanoid biosynthesis involved in inflammation and mitogenesis. It mediates the formation of prostaglandins from arachidonate and may have a role as a major mediator of inflammation and/or a role for prostanoid signaling in activity-dependent plasticity.

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