MULTISCREEN™ STABLE CELL LINE
HUMAN RECOMBINANT GPR4 RECEPTOR

PRODUCT INFORMATION

Catalog Number: C1100

Lot Number: C1100-030717

Quantity: 1 vial (2 x 10^6) frozen cells

Freeze Medium: Sigma Freezing Medium (C-6164)

Host cell: HEK293T

Transfection: Expression vector containing full-length human GPR4 cDNA (GenBank Accession Number NM_005282.1) with FLAG tag sequence at N-terminus

Recommended Storage: Liquid nitrogen upon receiving

Propagation Medium: DMEM, 10% FBS, 1 µg/mL puromycin

Stability: Stable for a minimum of 2 months in continuous culture

Data Sheet

Background: The GPR4 is family of proton-sensing G protein-coupled receptors (GPCRs) and has recently been identified as novel pH sensors. GPR4 sense extracellular protons through histidine residues of the receptors and are coupled to G-proteins to stimulate intracellular signaling pathways. This receptor is expressed in vascular endothelial and smooth muscle cells, as well in a wide range of tissues such as the lung, kidney, heart, and liver. GPR4, upon activation by acidic pH stimulates the G_s/cyclic adenosine monophosphate (cAMP) signaling pathway in endothelial cells and regulates micro vessel growth.

Application: Functional assays

Figure 1

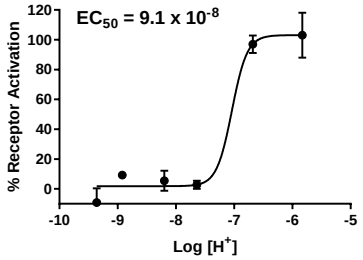

Figure 2

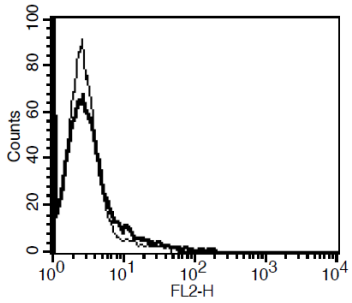

Figure 1. Dose-dependent increase of intracellular cAMP level upon treatment with ligand, measured with MultiScreen™ TR-FRET cAMP 1.0 No Wash Assay Kit (Multispan MSCM01) **Figure 2.** Receptor expression on cell surface measured by flow cytometry (FACS) using an anti-FLAG antibody. Thin line: parental cells; thick line: receptor-expressing cells.

References:

Chen A, Dong L, Leffler NR, Asch AS, Witte ON, et al. (2011) Activation of GPR4 by Acidosis Increases Endothelial Cell Adhesion through the cAMP/Epac Pathway. PLOS ONE 6(11): e27586. doi: 10.1371/journal.pone.0027586

Yang LV, Radu CG, Roy M, Lee McLaughlin J, et al. (2007) Vascular abnormalities in mice deficient for the G protein- coupled receptor GPR4 that functions as a pH sensor. Mol Cell Biol 27: 1334–1347.

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