

Atlas of Genetics and Cytogenetics in Oncology and Haematology

[Home](#) [Genes](#) [Leukemias](#) [Solid Tumours](#) [Cancer-Prone](#) [Deep Insight](#) [Case Reports](#)
[Journals](#) [Portal](#) [Teaching](#)

[X](#) [Y](#) [1](#) [2](#) [3](#) [4](#) [5](#) [6](#) [7](#) [8](#) [9](#) [10](#) [11](#) [12](#) [13](#) [14](#) [15](#) [16](#) [17](#) [18](#) [19](#) [20](#) [21](#) [22](#) [NA](#)

PTPRJ (protein tyrosine phosphatase, receptor type, J)

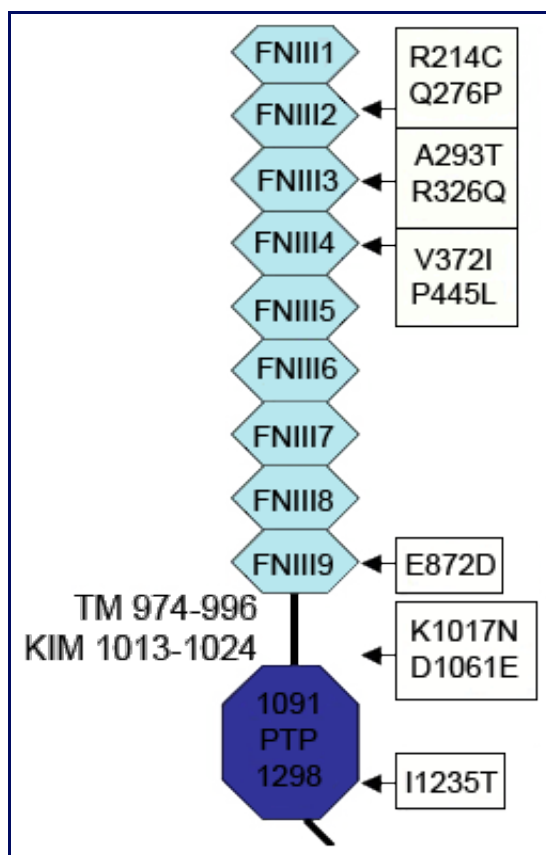
Identity

Other names	CD148 DEP1 HPTPeta R-PTP-ETA SCC1
HGNC (Hugo)	PTPRJ
LocusID (NCBI)	5795
Location	11p11.2
Location_base_pair	Starts at 48002110 and ends at 48154266 bp from pter (according to hg19-Feb_2009) [Mapping]
Note	This gene is a member of the Human CCDS set: CCDS44596, CCDS7945.

DNA/RNA

Description	Gene ENSG00000149177 has 24 alternate exons.
Transcription	<p>Multiple transcript variants encoding different isoforms have been found for this gene. Three transcripts are described in Ensembl (ID: ENSG00000149177). The first (ENST00000418331) represents the longer isoform and encodes a protein that consists of an extracellular fibronectin type 3 domain and a cytosolic catalytic domain. The second isoform (ENST00000440289) has multiple differences in the presence and absence of exons at its 3' end, compared to the first isoform. These differences produce a unique 3' UTR and the encoded protein is shorter and consists of the extracellular fibronectin type 3 domain without the cytosolic catalytic region. The third transcript (ENST00000278456) has the same length as the first, but it encodes a truncated protein, that consists of fibronectin type 3 domains. Only the first two isoforms are members of the human CCDS set and have been described in NCBI (IDs: NM_001098503.1, NM_001098503.1).</p> <p>The cDNA encoding PTPRJ/DEP-1 was isolated from a HeLa cell library (Ostman et al., 1994). The expression level and the activity of DEP-1 increase with cell density. This increase occurs gradually with increasing cell contact and is initiated before saturation cell density is reached. These observations suggest that DEP-1 may contribute to the mechanism of contact inhibition of cell growth.</p>

Protein



DEP-1: location of SNPs and mutations reported in human cancer. DEP-1 sequence is according to UniProtKB/Swiss-Prot: PTPRJ_HUMAN, Q12913. FNIII: Fibronectin domain, TM: Trans-membrane, KIM: Kinase Interaction Motif, PTP: Protein Tyrosine Phosphatase.

Description

DEP-1 corresponds to a single-pass, type I membrane receptor-type protein tyrosine phosphatase; it is a 200-250 kDa transmembrane molecule with a 35 residue signal peptide, 940 residue extracellular portion, 21 residues in helical transmembrane region and 341 aminoacids in the intracytoplasmic tail, with a single catalytic domain at the carboxi-terminus.

The protein N-terminus has nine fibronectin type III (FNIII) repeats, contains 34 potential N-linked glycosylation sites and it was found glycosylated in seven asparagine residues by mass spectrometry (Liu et al., 2005; Chen et al., 2009). DEP-1 can dimerize (binding site 972-996) through the transmembrane domain (Chin et al., 2005) but it is unclear whether it can exist as dimers, physiologically. Sedimentation velocity measurements show that DEP1, along with other single-domain receptor phosphatases (e.g., IA2, IA2beta, GLEPP1 and STEP) is monomeric in solution (Barr et al., 2009). The regulation of receptor-like protein tyrosine phosphatases is generally poorly understood. Sorby et al. have identified Matrigel™ (a preparation of extracellular matrix proteins) as a source of DEP-1 ligand(s) and agonist(s) (Sorby et al., 2001).

Expression

DEP-1 is expressed in several cell types, including endothelial, epithelial, and in all haematopoietic lineages. It was found to be present on many epithelial cell types with glandular and endocrine differentiation as well as on fibrocytes, melanocytes and Schwann cells (Autschbach et al., 1999). It is widely expressed on B and T cells, granulocytes, macrophages, certain dendritic cells as well as mature thymocytes and it was shown to negatively regulate T cell receptor and terminating its response to stimulation. DEP-1 is thought to play a role in the down regulation of T cell activation

and signalling, by interfering with the phosphorylation of Phospholipase C Gamma 1 and Linker for Activation of T Cells (LAT) and by reducing TCR-induced transcription factor (nuclear factor of activated T cells) (Lin and Weiss, 2003; Lin et al., 2004; Zhu et al., 2008; Tangye et al., 1998; Baker et al., 2001). DEP-1 expression is regulated during T cell activation and is constitutive in autoimmune murine T cells. These expression data suggest that DEP-1 plays a regulatory role to control an immune response and the proposed model of action describes DEP-1 as excluded from the immunologic synapse (i.e., the site of APC and T cell contact) during early T cell-APC (Antigen Presenting Cell) interactions. After T cell-APC disengagement, DEP-1 is no longer excluded by the synapse and can access and dephosphorylate substrates to down-regulate prolongation of the response (Lin et al., 2004).

It is interesting to note that the expression patterns of DEP-1 are different between humans and mice, where the expression in the T cell lineage is more restricted (Lin et al., 2004). Recently, DEP-1 is indicated as the phosphatase that can compensate for the absence of CD45, the prototypical receptor-like PTP expressed on immune cells (Zhu et al., 2008).

Function DEP-1 is a member of the protein tyrosine phosphatase (PTP) family. Since its expression is increased with increasing cell density, it is strongly suggested that DEP-1 may contribute to the mechanism of contact inhibition of cell growth (Ostman et al., 1994). It seems to be involved in protein tyrosine kinase signaling, cell-cell signaling, vasculogenesis and heart development (Takahashi et al., 2003).

Implicated in

Entity Thyroid cancer

Note Allelic loss of this gene is involved in thyroid carcinogenesis: the loss of heterozygosity (LOH) of PTPRJ is detected in 11/76 (14,5%) thyroid tumors (adenomas and carcinomas). DEP-1 overexpression in a malignant rat thyroid cell line was reported to specifically induce dephosphorylation of the Src-inhibitory Y529 and thus to increase [Src](#) activity. In addition, there are data suggesting that PTPRJ polymorphisms can affect susceptibility to thyroid carcinomas: DEP-1 homozygous for the Gln276Pro, Arg326Gln and the Glu872Asp allele were more frequent in thyroid carcinoma patients than in healthy individuals. Moreover, PTPRJ LOH was more frequent in thyroid carcinomas of heterozygotes for Gln276Pro and Arg326Gln compared with homozygotes, suggesting that the presence of hemizygoty for these polymorphisms in the tumor facilitates tumor progression (Iuliano et al., 2004). It was recently reported that the homozygous genotype PTPRJ/DEP-1 for Glu872Asp is associated with an increased risk to develop [papillary thyroid carcinoma](#) (Iuliano et al., 2010).

DEP-1 can dephosphorylate the single-pass transmembrane tyrosine kinase receptor [RET](#), impairing its signaling. Iervolino et al. show that Tyr1062 is critical for the binding of RET to DEP-1 and that DEP-1 can dephosphorylate the Tyr905 and Tyr1062 of the mutant RET bearing a germ-line Cys634Arg point mutation, responsible for the inheritance of [multiple endocrine neoplasia types 2A](#) (RET-MEN2A) and of the RET/PTC (papillary thyroid carcinoma) chimeric oncoprotein (Iervolino et al., 2006). The authors also show a DEP-1 dose dependent reduction of the transforming activity of RET-MEN2A. Furthermore, Iervolino et al. verified that the conformationally altered RET, bearing the mutation Met918Threo, responsible for MEN2B, does not bind or is dephosphorylated by DEP-1. This resistance to DEP-1 may account for the high oncogenic activity of the MEN2B RET alleles.

The rat protein tyrosine phosphatase eta, homologue of DEP-1, suppresses the neoplastic phenotype of retrovirally transformed thyroid cell, causing G1 growth arrest, by increasing the cyclin-dependent kinase inhibitor [p27Kip1](#) protein level, through reduction of its proteasome-dependent degradation rate (Trapasso et al., 2000).

Entity

[Colon rectal cancer](#)

Note

DEP-1 is a candidate human homologue of the mouse colon cancer susceptibility locus SCC1.

Germline allele-specific epigenetic silencing of the gene PTPRJ, encoding DEP-1, is described in early-onset familial colorectal cancer, due to a 170-kb intragenic duplication affecting the 5' end of the phosphatase gene PTPRJ the tandem duplication in a head-to-tail orientation (Venkatachalam et al., 2010; Ruivenkamp et al., 2002; Ruivenkamp et al., 2003a; Ruivenkamp et al., 2003b). PTPRJ is frequently deleted in human colon, [lung](#) and [breast carcinomas](#) or it shows allelic imbalance in loss of heterozygosity (LOH) and missense mutations. LOH of PTPRJ occurs early in progressed colorectal adenoma. Ruivenkamp et al. (2002) identified 5 somatic missense mutations in the PTPRJ gene, in colon cancer. All are localized in the extracellular fibronectin domains, two, Arg214Cys and Arg326Gln, result in loss of a positive charge. Mita et al. show, in their association study, that the Arg326Gln SNP demonstrates a close association with colon rectal cancer risk (Mita et al., 2010).

Nutrients, which are considered to be chemoprotective with respect to colon cancer development, including butyrate, green tea and apple polyphenols, seem to elevate transcription of endogenous DEP-1 mRNA and expression of DEP-1 protein.

Upregulation of DEP-1 expression with a consequent inhibition of cell growth and migration may present a mechanism of chemoprevention by nutrients (Balavenkatraman et al., 2006).

Entity

[Meningiomas](#)

Note

Suppressor of epithelial tumor.

LOH of the PTPRJ gene are observed in average 38% of human meningiomas of all grades (Petermann et al., 2010). Also, RNAi-mediated suppression of DEP-1, in DEP-1 positive meningioma cell lines (i.e., KT21, SF3061), causes enhanced motility and colony formation in semi-solid media (Petermann et al., 2010). It is therefore suggested that DEP-1 acts as a negative regulator of PDGF signaling and as a positive regulator of adhesion signalling, cooperating in inhibition of cell motility and tumor invasiveness and behaving as a positive regulator of paxillin tyrosine phosphorylation in epithelial cells. DEP-1 negatively affects PDGF stimulated activation of inositol trisphosphate formation, [Erk1/Erk2](#), [p21Ras](#), and Src. Activation of receptor-associated PI3K activity and of [Akt/PKB](#) are weakly attenuated at early time points of PDGF stimulation (Jandt et al., 2003). [PDGFR](#) and DEP-1 physically interact and there is a site-specific reduction in tyrosine phosphorylation of the PDGF b-receptor after in vivo and in vitro exposure to DEP-1. In fact, DEP-1 dephosphorylates the PDGFb-receptor P-Tyr751 and P-Tyr1009/1021, which are required for activation of PI3-K and PLCgamma, respectively, while the PDGFR regulatory P-Tyr857 is less affected (Kovalenko et al., 2000). Moreover, DEP-1 interacts with and dephosphorylates the p85 subunit of PI3K, bound to PDGFR (Tsuboi et al., 2008).

Entity

Various cancers

Note

EGFR and DEP-1

EGFR and DEP-1 physically associate, and EGFR phosphorylates a substrate-trapping mutant of DEP-1. Sacco et al. suggest that DEP-1 could act on EGFR phospho-Tyr998 (Sacco et al., 2009). Tarcic et al. suggest a mechanism by which DEP-1 affects EGFR trafficking and signaling. They show that DEP-1 dephosphorylates EGFR at the cell surface and affects the major phosphorylation site on [CBL](#) (at Tyr731), thus hampering EGFR ability to associate with the dedicated ubiquitin ligase complex (i.e., [CBL-GRB2](#)). This blocks EGFR ubiquitinylation, translocation to endosomes and, consequently, downstream signalling. DEP-1 silencing, on the contrary, enhances tyrosine phosphorylation of endosomal EGFRs, accelerates degradation of EGFR and increases cell proliferation (Tarcic et al., 2009). Consistent with this observation and with the induction of DEP-1 expression in contact-inhibited cells, the gene encoding DEP-1 is often deleted or mutated in carcinomas (Ruivenkamp et al., 2003a).

A genomic analysis of four different DNA samples (peripheral blood, primary tumor, brain metastasis and an early passage xenograft) from a patient with basal-like breast cancer (i.e., [ERBB2](#) and ER negative), identify a missense mutation (K1017N) in DEP1/PTPRJ that is highly enriched during disease progression, since it is found enriched in metastasis and in the human-in-mouse xenograft, compared to primary tumor (Ding et al., 2010). The mutation site is in the juxtamembrane domain (a basic residue motif) and is in close proximity to the tyrosine-protein phosphatase domain (aa 1041-1298). Sacco et al. reported that the DEP-1 charged peptide 1013-IKPKKSKLIRVE-1024 is responsible for interaction with its substrates, such as ERK1/2 (Sacco et al., 2009). The K1017N mutation found in the basal tumor and the K1016A mutation described in Sacco's report, both describe a change of a basic residue into a neutral residue. The DEP-1 positively charged peptide has a loose resemblance to the KIM domains that is essential to bind the common docking domain of ERK1/2 proteins. Therefore, a mutation in the DEP-1 KIM domain affects ERK1/2 recruitment, thus impacting specifically on phospho-Tyr204 ERK1/2 dephosphorylation efficiency. DEP-1 can therefore suppress proliferation by sequestering ERK1/2, irrespective of ERK1/2 phosphorylation state and by dephosphorylating the ERK1/2 activation loop. Loss of heterozygosity, and missense mutations in the DEP-1 gene have been identified in several human cancers including colon, lung, and breast, which have also been associated with aberrant Hepatocyte Growth Factor Receptor Tyrosine Kinase ([Met](#)) signaling. Palka et al. identify the hepatocyte growth factor/scatter factor receptor Met, the adapter protein GAB1 and the junctional component [p120 catenin](#) as potential DEP-1 substrates. Moreover, they show that DEP-1 preferentially dephosphorylates Met at sites involved in Met signaling: a GAB1 binding site (Tyr1349) and a COOH-terminal tyrosine implicated in morphogenesis (Tyr1365), while leaving unaltered the phosphorylation at the Met activation loop (Palka et al., 2003; Wang et al., 2010). A phosphatome RNAi (RNA interference) screen in A549 cells (lung adenocarcinoma) designed to identify phosphatases that behave as key negative regulators of oncogenic Ras signalling, characterized DEP-1 as a specific inhibitor of oncogenic [K-Ras](#) activation of Akt. The basis for this may be due to the role of DEP-1 in dephosphorylating residues within the p85 subunit of PI3K, resulting in attenuated PI3K/Akt activation (Omerovic et al., 2010).

DEP-1 and VEGFR

Whether or not DEP-1 plays a role in hematologic malignancies is not clear. DEP1 targets VEGFR2 phospho-tyrosines: In contact-inhibited endothelial cells, the phosphatase DEP-1, by binding [beta-catenin](#) and p120, may associate with the cadherin-receptor complex and dephosphorylate VEGFR-2 that is in complex with VEC (vascular endothelial cadherin). Retention of VEGFR-2 at the membrane by VEC and its dephosphorylation by DEP-1 limits its internalization and signaling (Lampugnani et al.,

2003; Holsinger et al., 2002; Lampugnani et al., 2006; Lampugnani and Dejana, 2007a; Lampugnani and Dejana, 2007b; Dejana et al., 2008).

Despite its negative role on global VEGFR2 phosphorylation, DEP-1 is a positive regulator of VEGF mediated Src and Akt activation and endothelial cell survival. In fact, when DEP-1 is depleted, inhibitory Src Y529 phosphorylation is increased and consequently, Src activation is impaired. Reduced Src activity correlates with decreased phosphorylation of GAB1 that cannot associate with PI3K (phosphatidylinositol 3-kinase), thus impairing AKT activation (Chabot et al., 2009).

Entity Cogan's syndrome

Note DEP-1 has been implicated in an autoimmune disease, the Cogan's syndrome, which is a chronic inflammatory disease characterized by sensorineural hearing loss, keratitis, and vasculitis. A screen for autoantigens in this disease revealed antibodies to DEP-1, which is expressed on the sensory epithelia of the inner ear and on endothelial cells (Lunardi et al., 2002). These autoantibodies inhibit proliferation of DEP-1 expressing cells and can replicate the hearing loss feature of Cogan's disease, when infused into mice. It is not determined whether these antibodies are causative for this autoimmune disease and/or others (e.g., pseudo-Pendred syndrome) (Davis et al., 2006).

External links

	Nomenclature
HGNC (Hugo)	PTPRJ 9673
Entrez Gene (NCBI)	PTPRJ 5795 protein tyrosine phosphatase, receptor type, J
	Cards
Atlas	PTPR,JID41932ch11p11
GeneCards (Weizmann)	PTPRJ
Ensembl (Hinxton)	ENSG00000149177 [Gene_View] chr11:48002110-48154266 [Contig_View] PTPRJ [Vega]
AceView (NCBI)	PTPRJ
Genatlas (Paris)	PTPRJ
euGene (Indiana)	5795
SOURCE (Stanford)	NM_001098503 NM_002843
	Genomic and cartography
GoldenPath (UCSC)	PTPRJ - 11p11.2 chr11:48002110-48154266 + 11p11.2 [Description] (hg19-Feb_2009)
Ensembl	PTPRJ - 11p11.2 [CytoView]
Mapping of homologs : NCBI	PTPRJ [Mapview]
OMIM	114500 600925
	Gene and transcription
Genbank (Entrez)	AI218139 AL359057 BC019824 BC063417 BC098447
RefSeq transcript	NM_001098503 NM_002843

(SRS)	NM_001098503 NM_002843
RefSeq transcript (Entrez)	NM_001098503 NM_002843
RefSeq genomic (SRS)	AC_000054 AC_000143 NC_000011 NG_012209 NT_009237 NW_001838022 NW_925006
RefSeq genomic (Entrez)	AC_000054 AC_000143 NC_000011 NG_012209 NT_009237 NW_001838022 NW_925006
Consensus coding sequences : CCDS (NCBI)	PTPRJ
Cluster EST : Unigene	Hs.318547 [SRS] Hs.318547 [NCBI]
Alternative Splicing : Fast-db (Paris)	6346
Alternative Splicing Gallery	ENSG00000149177
Gene Expression	PTPRJ [NCBI-GEO] PTPRJ [EBI - ARRAY_EXPRESS]
	Protein : pattern, domain, 3D structure
UniProt/SwissProt	Q12913 (SRS) Q12913 (Expasy) Q12913 (Uniprot)
With graphics : InterPro	Q12913
Splice isoforms : VarSplice FASTA	Q12913 (VarSplice FASTA)
Domaine pattern : Prosite (SRS)	FN3 (PS50853) TYR PHOSPHATASE 1 (PS00383) TYR PHOSPHATASE 2 (PS50056) TYR PHOSPHATASE PTP (PS50055)
Domaine pattern : Prosite (Expasy)	FN3 (PS50853) TYR PHOSPHATASE 1 (PS00383) TYR PHOSPHATASE 2 (PS50056) TYR PHOSPHATASE PTP (PS50055)
Domains : Interpro (SRS)	Dual-sp/Tyr phosphatase Fibronectin III dom FN III Ig-like fold Tyr Pase AS Tyr Pase rcpt/non-rcpt
Domains : Interpro (EBI)	Dual-sp/Tyr phosphatase Fibronectin III dom FN III Ig-like fold Tyr Pase AS Tyr Pase rcpt/non-rcpt
Related proteins : CluSTr	Q12913
Domain families : Pfam (SRS)	fn3 (PF00041) Y_phosphatase (PF00102)
Domain families : Pfam (Sanger)	fn3 (PF00041) Y_phosphatase (PF00102)
Domain families : Pfam (NCBI)	pfam00041 pfam00102
Domain families : Smart (EMBL)	FN3 (SM00060) PTPc (SM00194)
Blocks (Seattle)	Q12913
PDB (SRS)	2CFV 2DLE 2NZ6
PDB (PDBSum)	2CFV 2DLE 2NZ6
PDB (IMB)	2CFV 2DLE 2NZ6
PDB (RSDB)	2CFV 2DLE 2NZ6

[Human Protein Atlas](#) [ENSG00000149177](#)[HPRD](#) [02955](#)**Protein Interaction databases**[DIP \(DOE-UCLA\)](#) [Q12913](#)[IntAct \(EBI\)](#) [Q12913](#)[FunCoup](#) [ENSG00000149177](#)[REACTOME](#) [PTPRJ](#)[BioGRID](#) [PTPRJ](#)[InParanoid](#) [Q12913](#)[Interologous
Interaction database](#) [Q12913](#)**Polymorphism : SNP, mutations, diseases**[SNP Single](#)[Nucleotide](#) [PTPRJ](#)[Polymorphism \(NCBI\)](#)[SNP \(GeneSNP Utah\)](#) [PTPRJ](#)[SNP : HGBase](#) [PTPRJ](#)[Genetic variants :
HAPMAP](#) [PTPRJ](#)[Somatic Mutations in
Cancer : COSMIC](#) [PTPRJ](#)[CONAN: Copy
Number Analysis](#) [PTPRJ](#)[Mutations and
Diseases : HGMD](#) [PTPRJ](#)[OMIM](#) [114500](#) [600925](#)[GENETests](#) [114500](#) [600925](#)[Disease Genetic
Association](#) [PTPRJ](#)[Genomic Variants](#) [PTPRJ](#)[snp3D : Map Gene to
Disease](#) [5795](#)**General knowledge**[Homologs :
HomoloGene](#) [PTPRJ](#)[Homology/Alignments
: Family Browser
\(UCSC\)](#) [PTPRJ](#)[Phylogenetic
Trees/Animal Genes :
TreeFam](#) [PTPRJ](#)[Catalytic activity :](#) [3.1.3.48](#) [Enzyme-Expasy] [3.1.3.48](#) [Enzyme-SRS] [3.1.3.48](#) [IntEnz-EBI] [3.1.3.48](#) [BRENDA] [3.1.3.48](#) [KEGG][Chemical/Protein](#)

Chemical/Protein Interactions : CTD5795

[vasculogenesis](#) [immunological synapse](#) [protein tyrosine phosphatase activity](#) [protein tyrosine phosphatase activity](#) [platelet-derived growth factor receptor binding](#) [protein binding](#) [plasma membrane](#) [integral to plasma membrane](#) [cell-cell junction](#) [heart development](#) [beta-catenin binding](#) [negative regulation of cell proliferation](#) [cell surface](#) [negative regulation of platelet-derived growth factor receptor signaling pathway](#) [hydrolase activity](#) [phosphatase activity](#) [phosphatase activity](#) [protein kinase binding](#) [regulation of cell adhesion](#) [negative regulation of cell growth](#) [negative regulation of cell migration](#) [ruffle membrane](#) [peptidyl-tyrosine dephosphorylation](#) [peptidyl-tyrosine dephosphorylation](#) [negative regulation of epidermal growth factor receptor signaling pathway](#) [cell projection](#) [negative regulation of vascular permeability](#) [negative regulation of MAP kinase activity](#) [gamma-catenin binding](#) [positive regulation of cell adhesion](#) [positive regulation of survival gene product expression](#) [platelet-derived gr](#)

Ontology : AmiGO

[vasculogenesis](#) [immunological synapse](#) [protein tyrosine phosphatase activity](#) [protein tyrosine phosphatase activity](#) [platelet-derived growth factor receptor binding](#) [protein binding](#) [plasma membrane](#) [integral to plasma membrane](#) [cell-cell junction](#) [heart development](#) [beta-catenin binding](#) [negative regulation of cell proliferation](#) [cell surface](#) [negative regulation of platelet-derived growth factor receptor signaling pathway](#) [hydrolase activity](#) [phosphatase activity](#) [phosphatase activity](#) [protein kinase binding](#) [regulation of cell adhesion](#) [negative regulation of cell growth](#) [negative regulation of cell migration](#) [ruffle membrane](#) [peptidyl-tyrosine dephosphorylation](#) [peptidyl-tyrosine dephosphorylation](#) [negative regulation of epidermal growth factor receptor signaling pathway](#) [cell projection](#) [negative regulation of vascular permeability](#) [negative regulation of MAP kinase activity](#) [gamma-catenin binding](#) [positive regulation of cell adhesion](#) [positive regulation of survival gene product expression](#) [platelet-derived gr](#)

Ontology : EGO-EBIPathways :
BIOCARTAPathways : KEGGAdherens junction**Other databases****Probes**Probes : ImagenesPTPRJ Related clones (RZPD - Berlin)**Litterature**PubMed46 Pubmed reference(s) in EntrezPubGenePTPRJ**Bibliography****Molecular cloning, characterization, and chromosomal localization of a novel protein-tyrosine phosphatase, HPTP eta.**

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