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Prestige Antibodies GPCR Markers for Cancer Research

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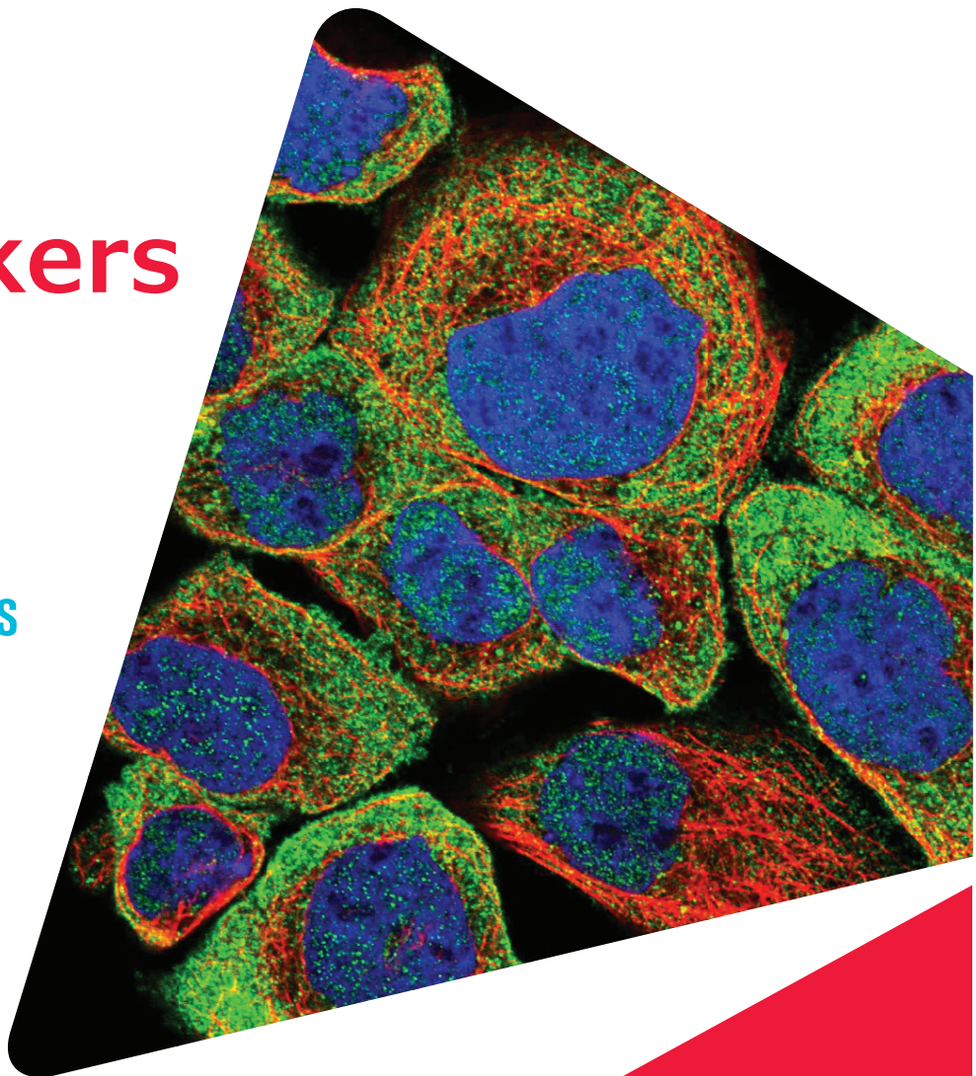

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G protein-coupled receptors (GPCRs) allow the communication between the environment and the cell's interior: they transduce extracellular stimuli into intracellular signals. GPCRs play an essential role in different physiological functions and have been well documented in many forms of cancer, contributing to cancer proliferation, angiogenesis, invasion, and metastasis. GPCRs are suitable biomarkers for the early diagnosis of cancer. The pharmacological inhibition of GPCRs and their downstream targets might provide an opportunity for the development of new, mechanism-based strategies for cancer prevention and treatment.

In this whitepaper, we highlight the role of some selected GPCRs markers in cancer development and treatment.

Introduction

G protein-coupled receptors (GPCRs), also known as seven-transmembrane domain (7TM) receptors, belong to a vast family of signaling proteins that mediate cellular responses to most hormones, metabolites, chemokines, and neurotransmitters.

With more than 800 identified members, the human GPCRs gene family remains the largest in mammals, including humans. Among the plethora of functions assigned to these proteins, one could mention mood and affective behavior, cardiovascular regulation, immunity and inflammation, olfaction, and pain[1].

Among the 800 family members, only four proton-sensing receptors GPR132 (G2A), GPR4, GPR65 (TDAG8), and GPR68 (OGR1), are known to regulate acidity (pH) in the cellular environment[1]. Except for protons, these receptors have no other known ligand and are hence called "orphan".

The GPCRs known so far falls into six classes based on sequence and function:

- Class A: Rhodopsin-like receptors,
- Class B: Secretin family,
- Class C: Metabotropic glutamate receptors,
- Class D: Fungal mating pheromone receptors,
- Class E: cAMP receptors,
- Class F: Frizzled (FZD) and Smoothened (SMO) receptors.

GPCRs constitute the most significant and druggable therapeutic target category[2], in fact, any pharmaceutical drug in development targets a transmembrane receptor[3], mainly a GPCR. Finding new ways to modulate GPCR signaling to increase their efficacy and at the same time blocking the adverse effects remains an important and tremendous challenge in pharmaceutical development.

GPCRs in cancer research

GPCRs are attractive drug targets due to their relevance in a plethora of disease conditions[4].

Despite a considerable body of information, GPCRs have not presented themselves as a valid "genetic driver" in cancer. Hence GPCRs have remained neglected or at least understudied as molecular targets in oncology.

The past decades have experienced a changed perception towards the role GPCRs in cancers. Today we know that aberrant GPCR expression may contribute to oncogenesis mechanisms, including metastasis, therapy resistance, and immune evasion.

Yet surprisingly, few GPCRs based therapies have found their way to the oncologist at the clinic. Despite recent advancements in the structural and functional knowledge of GPCRs, only eight FDA-approved drugs target GPCRs for cancer therapy[5] (**Table 1**).

Current and future research aim to discover the potential benefits of targeting GPCRs and their signaling circuits for the new era of precision medicine and cancer immunotherapies.

The first study of GPCRs and cancer was published in 1986 when a new human oncogene called MAS was noted for its tumorigenicity in nude mice[6].

This protein was found to be very hydrophobic with seven potential transmembrane domains. In this respect, the structure of the MAS protein was a novelty among cellular oncogene products.

For this reason, MAS constituted an entirely new functional class of oncogenes carrying no oncogenic mutations[6].

The MAS1 proto-oncogene-like GPCR binds the neuroregulatory peptide angiotensin[7] and is expressed in the testis, breast, cervical, lung, and pancreatic cancers.

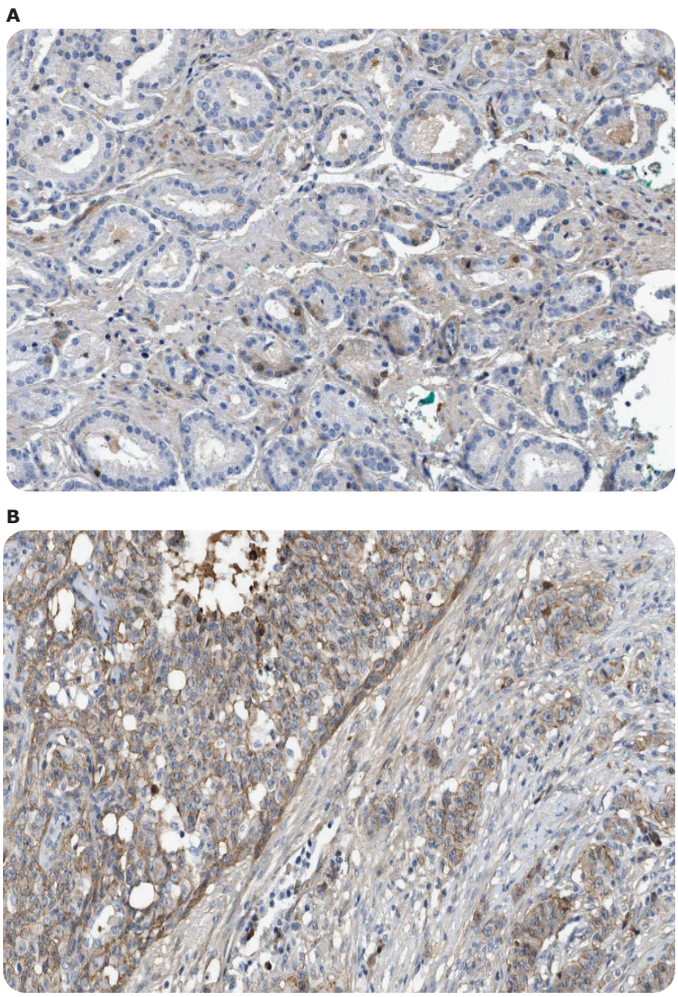

Figure 1. **Anti-MAS1L Antibody (**HPA017983**)**

Immunohistochemical stainings of human prostate cancer (A) and breast cancer (B) with our anti-MAS1L polyclonal antibody show moderate positivity in brown.

The first example of a classical GPCR neurotransmitter receptor with transforming properties was the serotonin 5HT1C-receptor (HTR1C) that, when expressed in mouse fibroblasts, generated sarcomas (tumors)[8].

The identification of activating mutations in the thyrotropin receptor gene (TSHR) in hyperfunctioning thyroid adenomas provided another evidence that mutant GPCRs could initiate a neoplastic disease[9].

The muscarinic acetylcholine (mAChRs) family of the GPCR rhodopsin-like receptors subtypes (CHRM1 HPA014101, CHRM2, and CHRM3) reveals oncogenic potential. Ligand-stimulation of mAChRs linked to phosphatidylinositol hydrolysis can act as conditional oncogenes showing the transforming ability of mouse fibroblast[10].

Like other oncogenes, tumor-generating mutations also occur among GPCRs, such as for the α-adrenergic receptor 1B (ADRA1B, HPA074416) inducing tumor generation in nude mice[11].

These early studies presented a new and unexpected role for GPCRs as capable of oncogenic transformation.

More recently, another interesting example of a mutated GPCR turned tumorigenic is the cysteinyl-leukotriene receptor 2 (CysLTR2, HPA046528), which has a single mutation in transmembrane helix 3 (mutant CysLTR2-L129Q), causing uveal melanomas in humans[12].

Neurohormonal GPCRs

The two members of the bombesin-family of regulatory peptides, gastrin-releasing peptide (GRP) and neuromedin B (NMB), are strongly implicated in cancer development[13,14]. The GRP receptor (GRPR) has higher selectivity for GRP over NMB, whereas the NMB receptor (NMBR) has the reverse pattern.

These GPCR-ligands are highly expressed/ produced in different tumors where they act as autocrine or paracrine growth factors. GRPR and NMBR are frequently overexpressed in colorectal tumors, gliomas and lung carcinomas, ovarian epithelial cancers, and neuroendocrine tumors of the gut and lung[15,16].

Many of the growth stimulatory effects of these GPCRs are mediated through the transactivation of the tyrosine kinase receptors like the EGF and HER2 receptors, belonging to an entirely different class of transmembrane proteins with kinase activity[17].

Another category of neurohormonal GPCRs involved in cancer is the category of somatostatin receptors SSTR1 and SSTR2, expressed in both small cell (SCLC) and non-small cell lung cancer (NSCLC)[18].

Radiolabeled SSTR2 analogs are utilized for radiographic imaging of tumors, which, in combination with PET-CT, provides improved lung cancer detection. SSTR2 analogs are also an interesting candidate for targeted chemotherapy and radiotherapy[19,20].

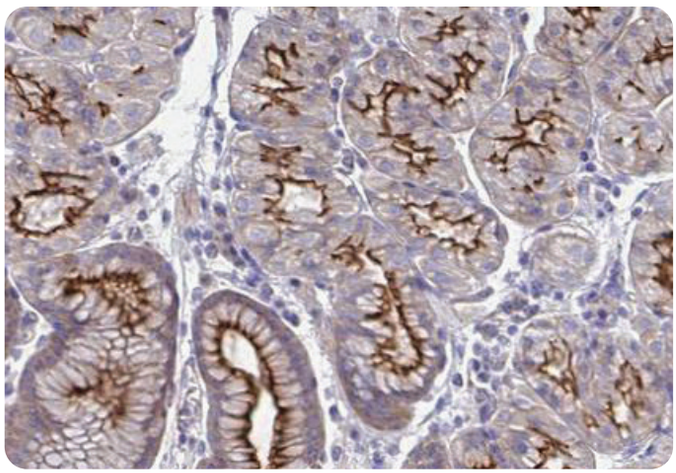

Figure 2. Anti-SSTR1 Antibody (HPA031506)

Immunohistochemical staining of human stomach with our anti-SSTR1 polyclonal antibody shows moderate to strong positivity in luminal membrane in glandular cells, in brown.

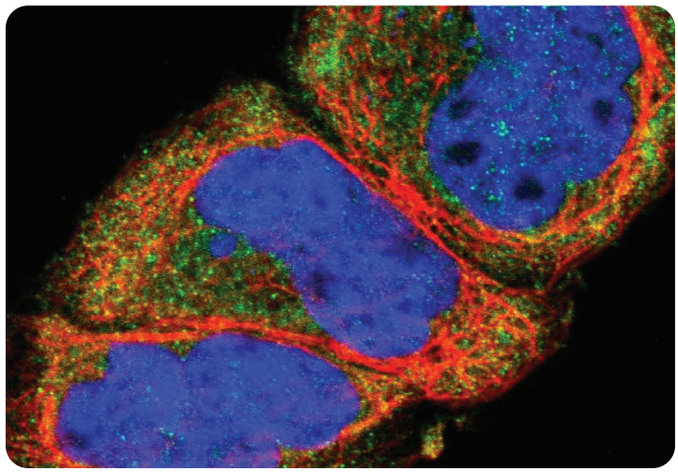

Figure 3. Anti-SSTR2 Antibody (HPA007264)

Immunofluorescent staining of human cell line A-431 with our anti-SSTR2 polyclonal antibody shows localization to cytosol, in green. Microtubules are shown in red and nuclei in blue.

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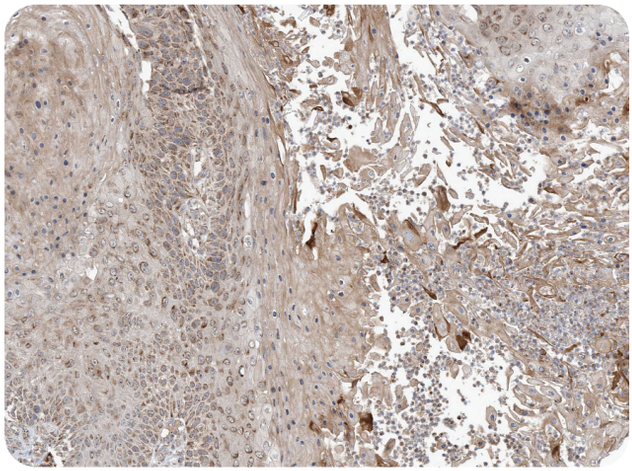

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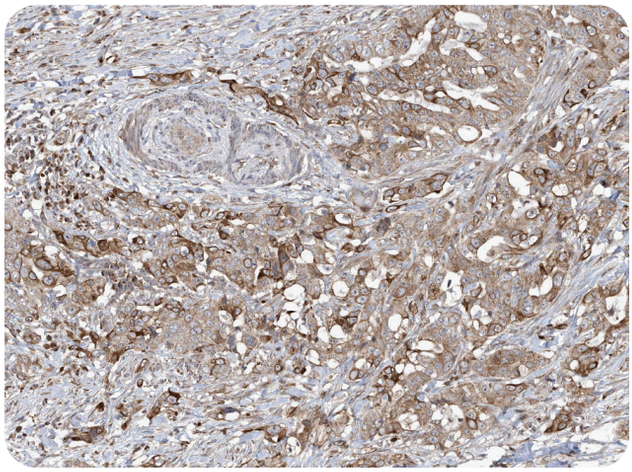


Figure 4. Anti-C2ORF74 antibody (HPA026826**).**

Immunohistochemical staining with our anti-C2ORF74 polyclonal antibody in human skin with squamous cell carcinoma (A) and lung adenocarcinoma (B) shows moderate cytoplasmic and membranous positivity, in brown. (C) Anti-C2ORF74 immunofluorescent staining of human cell line U-251 MG shows positivity in centrosome & nucleus but not nucleoli.

Box 1. GPCR and oncoviruses

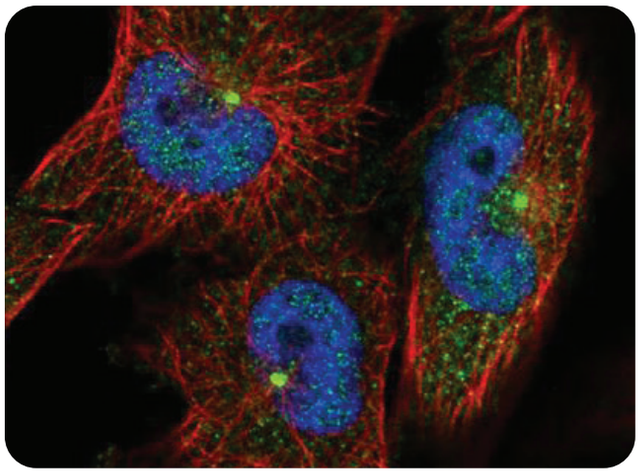
Oncoviruses are cancer-causing viruses. The human hepatitis B (HBV) and C (HCV), the human papillomaviruses (HPV), the Epstein–Barr virus (EBV), and the Kaposi sarcoma herpesvirus (KSHV) are the most important human oncoviruses currently known. Interestingly, some virally encoded and constitutively activated GPCRs promote and sustain tumorigenesis.

An example of GPCR and oncovirus is the “hijacked” GPCR known as KSHV-vGPCR (or ORF74). ORF74 is a highly constitutively active GPCR encoded and expressed by the HHV herpesvirus 8 (HHV8).

ORF74 is involved in the pathogenesis of Kaposi’s sarcoma (a tumorous skin lesion in immunocompromised patients, including those with progressed HIV/AIDS)[21].

As for the “classical” oncogenes belonging to the kinase category, most were originally identified in retroviruses. The dawn of these studies opened a new door to establish the link between GPCRs and cancers.

C



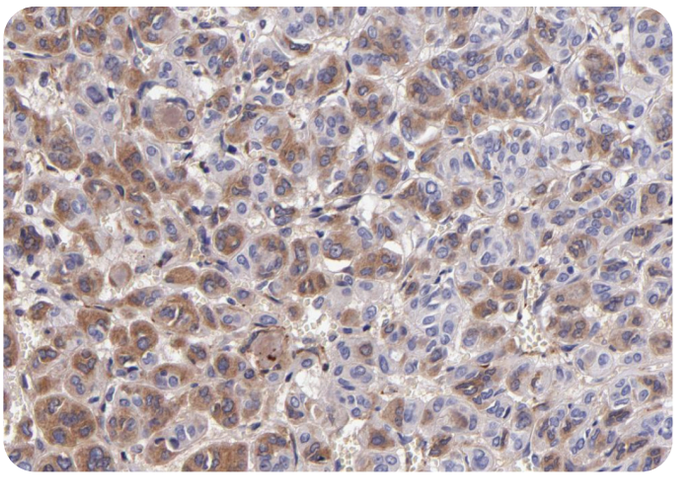


Figure 5. Anti-KNG1 antibody (HPA001616**)**

Immunohistochemical staining of a human thyroid gland affected by follicular adenoma carcinoma with our anti-KNG1 polyclonal antibody shows moderate cytoplasmic and membranous positivity, in brown.

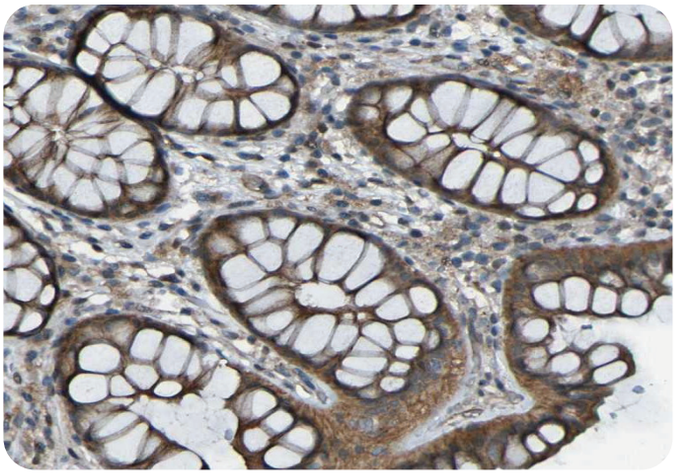


Figure 6. Anti-LGR5/GPR49 Antibody (HPA012530**)**

Immunohistochemical staining of human colon with our anti-LGR5/GPCR49 polyclonal antibody shows cytoplasmic positivity in glandular cells, in brown.

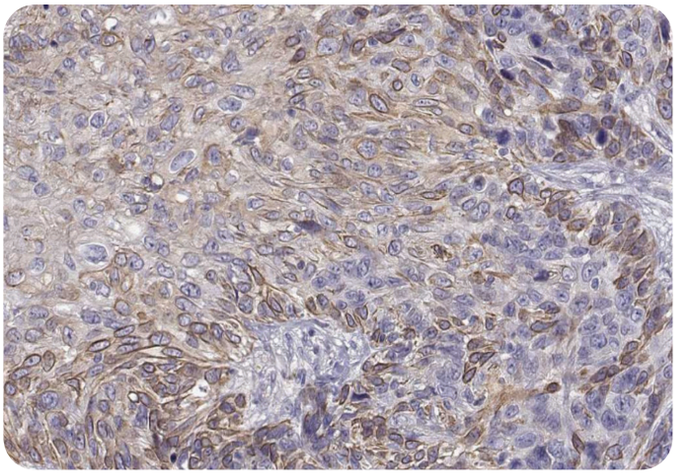


Figure 7. Anti-TACR1 Antibody (HPA074573**)**

Immunohistochemical staining with our anti-TACR1 polyclonal antibody of human skin affected by squamos cell carcinoma shows high positivity, in brown.

Rhodopsin-like GPCRs

The neuropeptide Y (NPY) is a neuropeptide that can also regulate the growth of various tumors. The NPY receptors belong to the class A or rhodopsin-like GPCR[22].

Neuroblastoma and Ewing's sarcoma (two pediatric tumor types) produce high levels of NPY. The hypoxic condition of the tumor microenvironment of these solid cancers upregulates and promotes the Y2R/Y5R NPY signaling axis[23]. By stimulating the cognate receptors, Y2R and Y5R, NPY promotes the survival, migration, and angiogenesis of these neoplasias.

Another category of rhodopsin-like GPCRs involved in cancer is the tachykinin receptors, including the substance P receptor/NK1R/TACR1. Substance P induces tumor cell proliferation, angiogenesis, and cancer cell migration. Moreover, tumor cells frequently overexpress TACR1 (HPA074573).

It is of interest in this context to note that the TACR1 antagonist, Aprepitant, shows promising anti-tumorigenic activity[24].

Frizzled GPCRs and Wnt signaling

The frizzled group of GPCRs is evolutionarily conserved and serves to transduce signals from the Wnt-type lipoprotein growth factors. Several cancers display dysregulated Wnt signaling pathways.

Originally defined as a Wnt target gene, potentiating the canonical Wnt/β-Catenin signaling, the Leucine-rich repeat-containing G-protein-coupled receptor 5 (LGR5 or GPR49, HPA012530) represents an exquisitely specific and almost "generic" marker of normal stem cells in several tissues, including the intestine[25].

LGR5 has been proposed as an independent prognostic marker and as therapeutic target for colorectal cancer, although, in this context, its role appears very complex[26].

Adhesion GPCRs

The adhesion G protein-coupled receptors (aGPCRs) constitute a large yet poorly understood family of seven-transmembrane proteins.

There is ample evidence for the implication of tumor-associated adhesion-GPCRs in tumorigenesis. These receptors affect the growth of tumor cells, angiogenesis, tumor cell migration, invasion, and metastasis, either positively or negatively[27].

Some adhesion-GPCRs are in addition considered potential biomarkers for specific types of cancers.

ADGRG1/GPR56 is upregulated in some colorectal cancer tissues but shows anti-metastatic properties in melanoma via inhibition of cell-extracellular matrix signaling[28,29].

Another member of the adhesion GPCRs family involved in cancer is the glioma marker ADGRL4 (AMAb91268), which promotes glioblastoma through hypoxia signaling and is considered a target for anti-cancer therapy[30].

GPCRs and chemokines

Chemokines are small soluble cytokines that guide the migration of other cell types, particularly immune cells. The chemokines have a plethora of important roles in cancer, not least in association with metastasis and tumor immunity[31].

Most chemokines act through GPCRs and play critical roles in tumor growth, angiogenesis, invasion, and leukocyte infiltration into the tumor. One example includes CCL2 (HPA019163), a chemokine secreted from tumors acting through its receptor CCR2 to promote prostate tumor growth[32] and stimulates tumor angiogenesis[33].

Most solid tumor undergoing metastasis does so via lymph nodes. One example is the chemokine (C-C motif) ligand 1 CCL1 (HPA049861) that attracts CCR8-positive distal tumor cells towards the lymph node[34]. Strategies to block the ligand interactions with these GPCR would be of great therapeutic value.

Chemokine/chemokine receptor signaling is also known to regulate tumor immunity. The ovarian cancer tumor hypoxia causes the induction of CCL28 (HPA077434). The upregulated CCL28 accelerated tumor growth by binding to its cognate GPCR, CCR10 (HPA013819) and activation of the immunosuppressive regulatory T-cells (Tregs)[35].

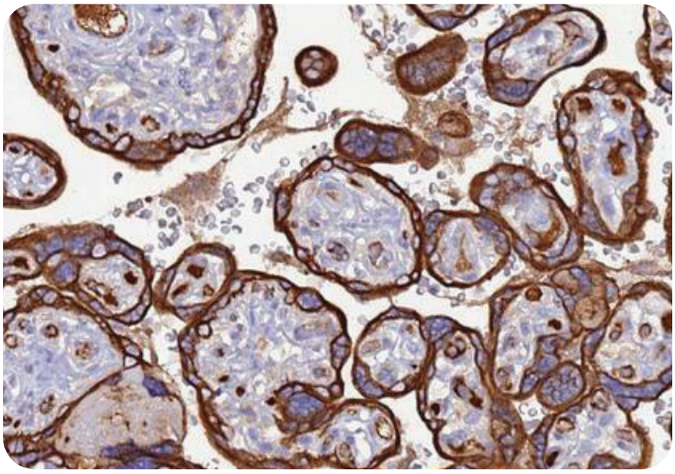


Figure 8. Anti-CYSLTR2 Antibody (HPA046528)

Immunohistochemical staining of human placenta with our anti-CYSLTR2 polyclonal antibody shows cytoplasmic and membranous positivity in trophoblastic cells, in brown.

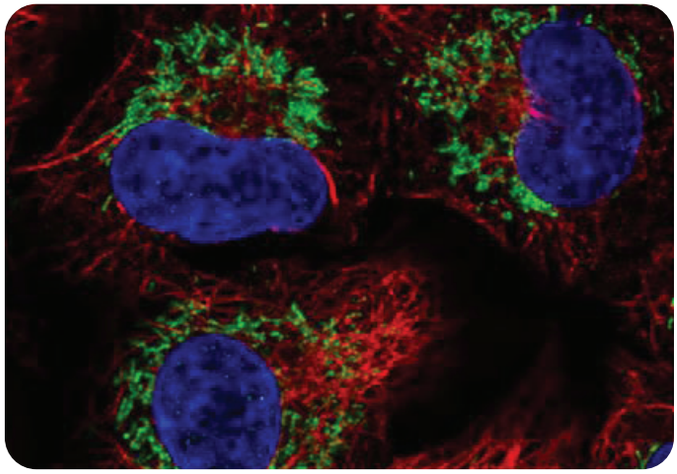


Figure 9. Anti-CCR7 Antibody (HPA074467)

Immunofluorescent staining of human cell line A549 with our anti-CCR7 polyclonal antibody shows localization to mitochondria in green. Microtubules are shown in red and nuclei in blue.

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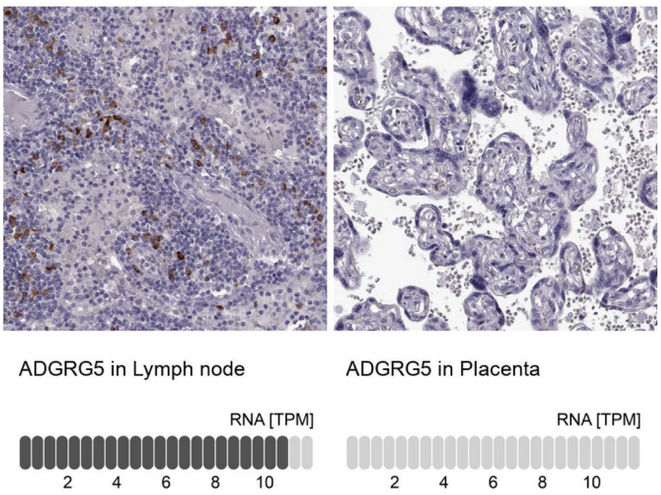

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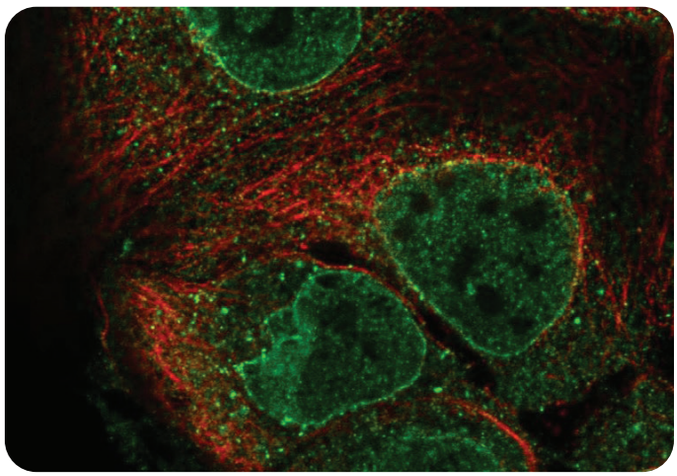

Figure 10. Anti-ADGRG5 antibody (HPA007133**)**

A. Immunohistochemistry analysis in human lymph node and placenta tissues using our anti-ADGRG5 polyclonal antibody.Corresponding RNA-seq data (TPM) are presented for the same tissues (orthogonal enhanced validation).

B. Immunofluorescent staining of human cell line CACO-2 shows localization to nucleoplasm, nuclear membrane & vesicles, in green. Microtubules are shown in red and nuclei in blue.

Table 1. FDA approved GPCRs drugs against different cancers.

Receptor	Drug	Cancer Therapy	Approval
Dopamine receptor D1 (DRD1)	Cabergoline	Neuroendocrine tumors, pituitary tumors	1996
Somatostatin receptor (SSTR)	Lanreotide	Pancreatic cancer	2007
Gonadotropin releasing factor hormone receptor (GnRH)	Degarelix	Prostate cancer	2008
C-X-C chemokine receptor 4 (CXCR4)	Plerixafor	Multiple myeloma	2008
Smoothened receptor (SMO)	Vismodegib (Erivedge)	Locally advanced, and metastatic basal cell carcinoma	2012
Estrogen receptor (ER)	Raloxifene	Breast cancer	2014
Smoothened receptor (SMO)	Sonidegib	Locally advanced, and metastatic basal cell carcinoma	2015
C–C Chemokine receptor 4 (CCR4)	Mogamulizumab	T cell lymphoma	2018

Box 2. GPR132, a potential tumor suppressor

The early GPCR-derived oncogene G2A, also known as GPR132 (HPA029694, HPA029695), is a member of the proton-sensing GPCRs subfamily. This GPCR induces a full range of phenotypes characteristic of oncogenic cellular transformation of mouse fibroblasts and tumorigenicity[36,37].

Inhibition of GPR132 is a potentially novel treatment for reducing cancer metastasis. Loss of GPR132 in mice inhibits breast cancer metastasis, and lower GPR132 expression in patients with breast cancer correlates with better metastasis-free survival[38].

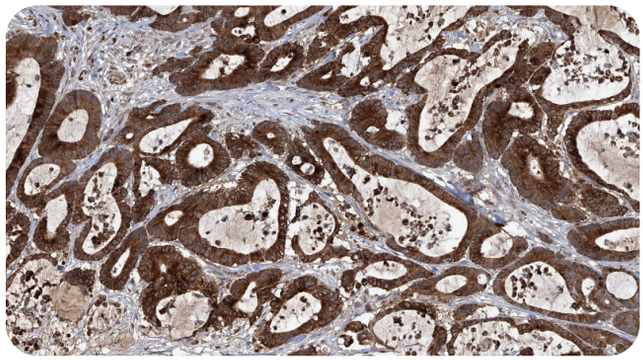

Figure 11. Anti-GPR132 antibody (HPA029695**).**

Immunohistochemical staining of a human colon with adenocarcinoma. The anti-GPR132 antibody binding is visible in brown.

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Table 2. Selection of polyclonal and monoclonal markers targeting GPCRs.

Product Name	Alternative Gene Names	Cat. No.	Clonality	Validated Applications	Seq Identity Mouse/Rat
Anti-ACKR2	CCBP2, CCR10, CCR9	**HPA013819**	Polyclonal	IHC*	56% / 56%
Anti-ADGRG2	EDDM6, GPR64,HE6	**HPA001478**	Polyclonal	IHC*	78% / 78%
Anti-ADGRG2	EDDM6, GPR64,HE6	**HPA050029**	Polyclonal	IHC*, WB*	76% / 75%
Anti-ADGRG3	GPR97, Pb99, PGR26	**AMAB91712**	Monoclonal	WB	-
Anti-ADGRG3	GPR97, Pb99, PGR26	**AMAB91713**	Monoclonal	IHC*	-
Anti-ADGRG4	GPR112, PGR17	**HPA035324**	Polyclonal	IHC	79% / 27%
Anti-ADGRL4	ELTD1, ETL	**AMAB91268**	Monoclonal	IHC, WB	53% / 55%
Anti-ADGRE5	CD97, TM7LN1	**HPA013707**	Polyclonal	IHC*	49% / 51%
Anti-ADGRG5	GPR114, PGR27	**HPA007133**	Polyclonal	IHC*, ICC-IF	61% / 61%
Anti-ADGRG6	FLJ14937, GPR126	**HPA017346**	Polyclonal	IHC	55% / 53%
Anti-ADRA1B		**HPA074416**	Polyclonal	ICC-IF	96% / 94%
Anti-C2orf74	LOC339804	**HPA026826**	Polyclonal	IHC, ICC-IF	50% / 47%
Anti-CCL1	I-309, P500, SCYA1	**HPA049861**	Polyclonal	IHC	36% / 37%
Anti-CCL2	GDCF-2, HC11, MCAF	**HPA019163**	Polyclonal	IHC, ICC-IF	65% / 60%
Anti-CCL28	CCK1, MEC, SCYA28	**HPA077434**	Polyclonal	IHC	87% / 94%
Anti-CCR7	CD197, CDw197, CMKBR7	**HPA031383**	Polyclonal	ICC-IF	83% / 85%
Anti-CCR7	CD197, CDw197, CMKBR7	**HPA074467**	Polyclonal	ICC-IF	88% / 88%
Anti-CCR8	CMKBRL2, CY6, GPR-CY6	**HPA042383**	Polyclonal	IHC	50% / 58%
Anti-CHRM1		**HPA014101**	Polyclonal	IHC*	98% / 97%
Anti-CXCR3	CD183, CMKAR3, GPR9	**HPA045942**	Polyclonal	IHC*	67% / 67%
Anti-CXCR5	BLR1, CD185, MDR15	**HPA042432**	Polyclonal	IHC*	47% / 42%
Anti-CYSLTR2	CysLT(2), CYSLT2R	**HPA046528**	Polyclonal	IHC	35% / 37%
Anti-FPR2	FMLP-R-II, FMLPX, FPR2A	**HPA029154**	Polyclonal	IHC	63% / 63%
Anti-GPBAR1	GPCR, GPCR19, GPR131	**HPA062890**	Polyclonal	IHC*	82% / 75%
Anti-GPR4		**HPA014278**	Polyclonal	IHC	96% / 96%
Anti-GPR27	SREB1	**HPA029395**	Polyclonal	WB, ICC-IF	91% / 91%
Anti-GPR39		**HPA022111**	Polyclonal	IHC	55% / 54%
Anti-GPR132	G2A	**HPA029694**	Polyclonal	IHC	54% / 54%
Anti-GPR132	G2A	**HPA029695**	Polyclonal	IHC	36% / 42%
Anti-GPR143	OA1	**HPA003648**	Polyclonal	IHC*	55% / 55%
Anti-GPR162	A-2, GRCA	**HPA055135**	Polyclonal	IHC*, WB, ICC-IF	97% / 94%
Anti-GPR17		**AMAB91624**	Monoclonal	IHC*, WB	92% / 92%
Anti-GPR17		**HPA029766**	Polyclonal	IHC*	92% / 92%
Anti-GPR37L1	ETBR-LP-2	**HPA064454**	Polyclonal	IHC*	77% / 81%
Anti-GPR65	hTDAG8, TDAG8	**HPA054454**	Polyclonal	ICC-IF	61% / 63%
Anti-KNG1	BDK, BK, KNG	**HPA001616**	Polyclonal	IHC*, WB	62% / 61%
Anti-KNG1	BDK, BK, KNG	**HPA001645**	Polyclonal	IHC*, WB*	59% / 58%
Anti-LGR5	FEX, GPR49, GPR67, HG38	**HPA012530**	Polyclonal	IHC	88% / 87%
Anti-LRP6	ADCAD2	**HPA029925**	Polyclonal	IHC*	96% / 97%
Anti-MAS1L	dJ994E9.2, MAS-L, MRG	**HPA017983**	Polyclonal	IHC, ICC-IF	25% / 23%
Anti-NMUR2	NMU2R	**HPA045836**	Polyclonal	IHC	56% / 53%
Anti-SSTR1		**HPA031506**	Polyclonal	IHC	98% / 98%
Anti-SSTR2		**HPA007264**	Polyclonal	IHC, WB*, ICC-IF	98% / 96%
Anti-TACR1	NK1R, SPR, TAC1R	**HPA074573**	Polyclonal	IHC	100% / 100%
Anti-TACR2	NK2R, NKNAR, SKR, TAC2R	**HPA076573**	Polyclonal	ICC-IF	81% / 78%
Anti-TACR3	NK3R	**HPA009418**	Polyclonal	IHC	60% / 55%
Anti-TBXA2R		**HPA077366**	Polyclonal	ICC-IF	82% / 75%

* Products with enhanced validation for indicated application.

In addition to the extensive validation and characterization always performed for our antibodies, we conduct application-specific enhanced validation to secure the antibody specificity in a defined context.

Very Reliable Antibodies

Prestige Antibodies® provides over 21,000 primary antibodies targeting the majority of human proteins. Building on our heritage with the Human Protein Atlas project, we provide highly validated reagents that enable leading research in biology, diagnostics, and medicine. All our products are rigorously evaluated for specificity, reproducibility and performance and characterized in multiple applications. Our team of researchers develops the next generation of innovative and reliable tools, fundamental to advancing research in neuroscience, oncology, cell biology, stem cells and development.

Created by the Human Protein Atlas

With our roots in the Human Protein Atlas project, an integration of antibody-based imaging, proteomics, and transcriptomics, our antibodies are affinity-purified, reproducible, selective, and specific for their target proteins through our enhanced validation process. Our Triple A PolyclonalsTM are developed within the Human Protein Atlas project.

Validated by Enhanced Validation

We take great care to validate our antibodies in IHC, WB, and ICC-IF. Our antibodies are validated in all major human tissues and organs and 20 cancer tissues. Each antibody is supported by over 500 staining images. As an additional layer of security, we perform Enhanced Validation. By using 5 different enhanced validation methods we validate our antibodies for each combination of protein, sample, and application. Discover our Triple A Polyclonals™ and PrecisA Monoclonals™ antibodies targeting the majority of human proteins in cells, tissues, and organs.

Evidenced by Science

Made by researchers for researchers our products are used all over the world and referenced in 1000s of scientific peer-reviewed papers.

We Support your Research

Our scientific content and newsletter provide you with timely information about new product releases, research highlights, and much more. In addition, from our website you can download informative white papers, protocols, guides, posters, infographics, roundups of recent research papers, read blog posts and interviews.

How to Buy Our Products

Our products are available worldwide. We deliver to all destinations in across Asia Pacific. We expand our offering through trusted partners worldwide.

You can shop our full catalog online or find your local supplier.

Prestige® Antibodies Advanced Polyclonals

Prestige® Antibodies Polyclonals are rabbit polyclonal primary antibodies developed within the Human Protein Atlas project. IHC characterization data from 44 normal and 20 cancer tissues is available on the Human Protein Atlas portal. Available as 25 µL and 100 µL unit size.

Precise. Accurate. Targeted.

Prestige® Antibodies Monoclonals are mouse monoclonal primary antibodies developed against a number of carefully selected targets. Clones are selected to recognize only unique non-overlapping epitopes and isotypes. Available as 25 µL and 100 µL unit size.

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