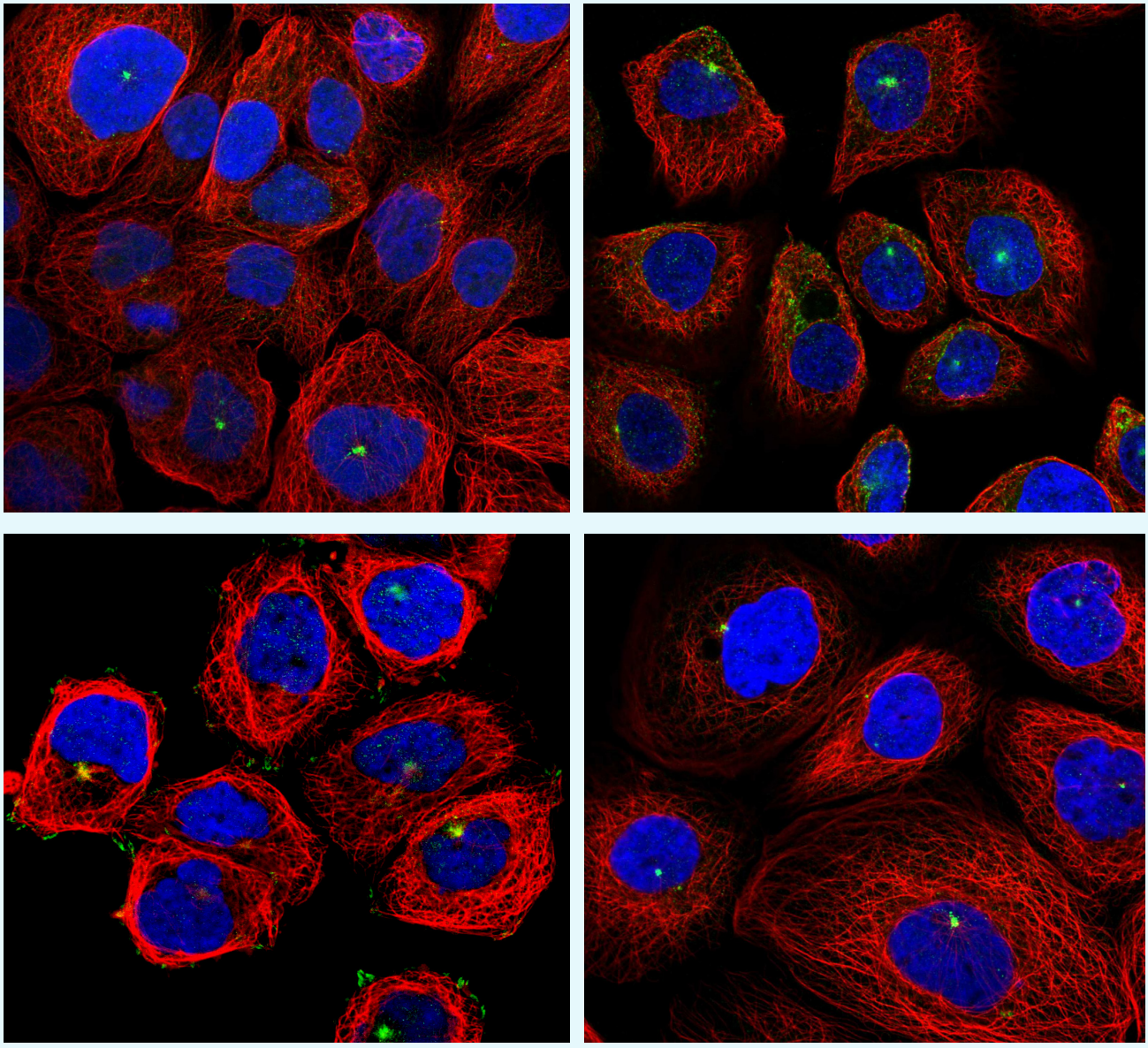
ATLAS ANTIBODIES



CENTRIOLAR SATELLITE MARKERS

The centrosome is the organelle that acts as the primary microtubule-organizing center of the cell. The centrosome includes the centrioles (the structures that enable the formation of spindle fibers) along with a dense mass of protein called the peri-centriolar material, now known as centriolar satellites.

The term “centriolar satellites” was coined in the second half of the 20th century to name the 70–100 nm electron-dense particles in diameter surrounding the centrosome. Centriolar satellites contain several centrosomal proteins and are the core structural elements of centrosomes and cilia.

Proteomics and systematic genome-wide analysis suggest that the human centrosome comprises hundreds of different components. According to the Subcellular section of the Human Protein Atlas (HPA) project, three percent of all protein-coding human genes (564 genes) encode proteins that localize to the centrosome or the centriolar satellites (proteinatlas.org).

Centriolar satellites, ubiquitous in vertebrate cells, are small, cytoplasmic membrane-less spherical granules that localize and move around the centrosomes and cilia. Centriolar satellites can also move along microtubules with the help of motor proteins. Thus, they function as vehicles for protein transport towards and away from the centrosome. However, they are mostly only detectable in interphase cells and undergo dissolution during mitosis (Kubo 2003).

Satellites have been implicated in multiple critical cellular functions, including centriole duplication, centrosome maturation, and ciliogenesis; however, their precise composition and assembly properties still need to be explored (Kubo 1999; Hori 2017).

Because of the substantial overlap of functions and proteomes between the centriolar satellites with centrosomes and cilia, research on centriolar satellites has so far been undertaken with a biased view from a centrosome/cilium-centered perspective.

As a result, centriolar satellites are often referred to as the “third component” of the vertebrate centrosome/cilium complex and as a member of the emerging class of membrane-less organelles.
Only about 50% of the centrosome proteome overlaps with the satellite proteome, challenging the current classification of proteins into “centrosome” or “satellite” categories.

Supporting this view, data show that satellite proteins composition is mainly unaffected by centriole depletion; most proteins of the centriolar satellites are present in cells that lack centrioles, meaning that satellite assembly is centrosome-independent.

Centriolar satellites are detectable in almost all mammalian cell types. Their cellular distribution ranges from clustering at the centrosomes, the nucleus or basal bodies, to scattered throughout the cytoplasm, depending on the cell types and tissues.

However, their size, molecular composition, abundance, and localization can vary considerably so, type and tissue-specific functions remain primarily unexplored.

A complete picture regarding the full set of satellite components and the spatiotemporal constitution of centriolar satellites has not yet been established because the number of proteins identified as satellite components has continued to increase over the last several years. They were reported to be 11 in 2011 (Bärenz 2011) and over 100 according to the most recent studies (Gupta 2015).

The analysis of the satellite interactome, combined with subdiffraction imaging, reveals the existence of multiple unique microscopically resolvable satellite populations that display distinct protein interaction profiles, paving the way for future studies aimed at better understanding the biogenesis and functions of these enigmatic structures (Gheiratmand 2019).

Atlas Antibodies offers numerous markers targeting centriolar satellites proteins, some of which are highlighted in this white paper. **Table 1** summarizes some examples of centriolar satellites proteins and disease involvement. **Table 2** lists a selection of our Triple A Polyclonals™ antibodies targeting centriolar satellite proteins for research use in IHC, ICC-IF and WB. **Figures 1-7** show representative immunofluorescent (ICC-IF) stainings of centriolar satellite proteins in various human cell lines.

Cover image: ICC-IF staining of human A-431 cell line using centriolar satellite antibody markers. Each antibody staining is visible in green. Microtubules are shown in red and nuclei in blue. ***Top Left:*** Anti-PCM1 (HPA023370). ***Top Right:*** Anti-C7orf43 (HPA029463). ***Bottom Left:*** Anti-MYO5A (HPA001356). ***Bottom Right:*** Anti-PCNT (HPA016820).

PCM1: the bona fide centriolar satellite marker

PCM1 (pericentriolar material 1) is a large protein (~230 kDa) and the first identified molecular component of centriolar satellites: it is now considered the bona fide centriolar satellite marker in cells (Balczon 1994; Baron 1988; Kubo 1999). Additional PCM1 location includes the cytosol.

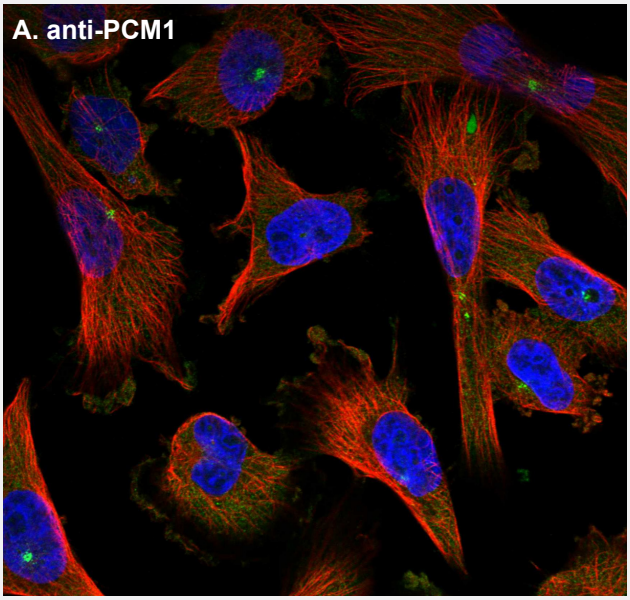
PCM1 serves as a scaffold protein and recruits centrosomal proteins to centriolar satellites (Hames 2005). It is essential to correctly position proteins such as CEP250, CETN3, PCNT, and NEK2 and anchoring microtubules to the centrosome.

PCM1 represents a structural platform for centriolar satellites: when PCM1 becomes dysfunctional, either by depletion, deletion or mutation, satellite particles disassemble (Dammermann 2002).

Therefore, the evaluation of new proteins as components of centriolar satellites is formally made according to two criteria: the first is colocalisation and physical interaction with PCM1, and the second is delocalisation from pericentrosomal locations upon PCM1 depletion (Lopes 2011).

PCM1 accumulates at the nuclear surface of differentiated, non-cycling myocytes. Research studies have demonstrated that antibodies against PCM1 specifically label cardiomyocyte nuclei, and as such, PCM1 has been used by several independent groups to identify cardiomyocytes both in human and rodents (Bergmann 2009, 2012; Hirai 2016; Preissl 2015).

Mutation of genes encoding PCM1 and other centriolar satellite components or regulatory proteins involved in centriolar satellite integrity can cause ciliopathy-related human diseases such as Bardet–Biedl syndrome, Joubert syndrome, Meckel Gruber syndrome, primary microcephaly (MCPH), and oral-facial-digital syndrome (Kodani 2015; Stephen 2015; Sang 2011).



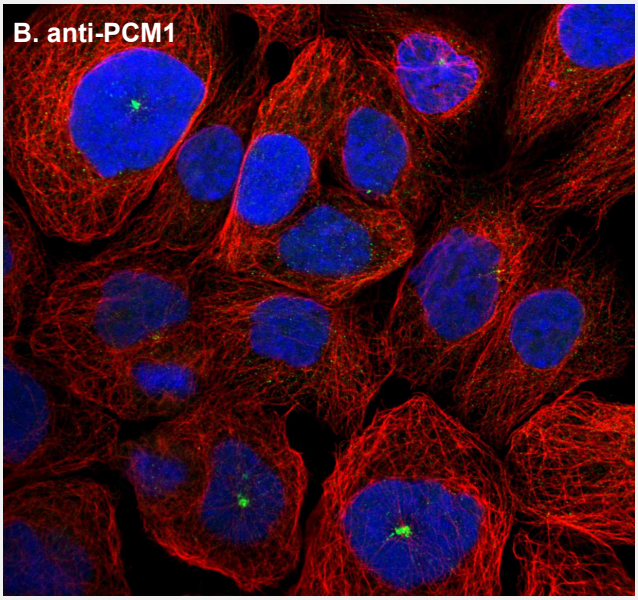


Figure 1.
(A) ICC-IF staining of human U-251 MG cells using the **anti-PCM1 (HPA023374)** antibody shows localization to centriolar satellite, in green. Microtubules, red. Nuclei, blue.

(B) ICC-IF staining of human A-431 cells using the **anti-PCM1 (HPA023370)** antibody shows localization to centriolar satellite, in green. Microtubules, red. Nuclei, blue.

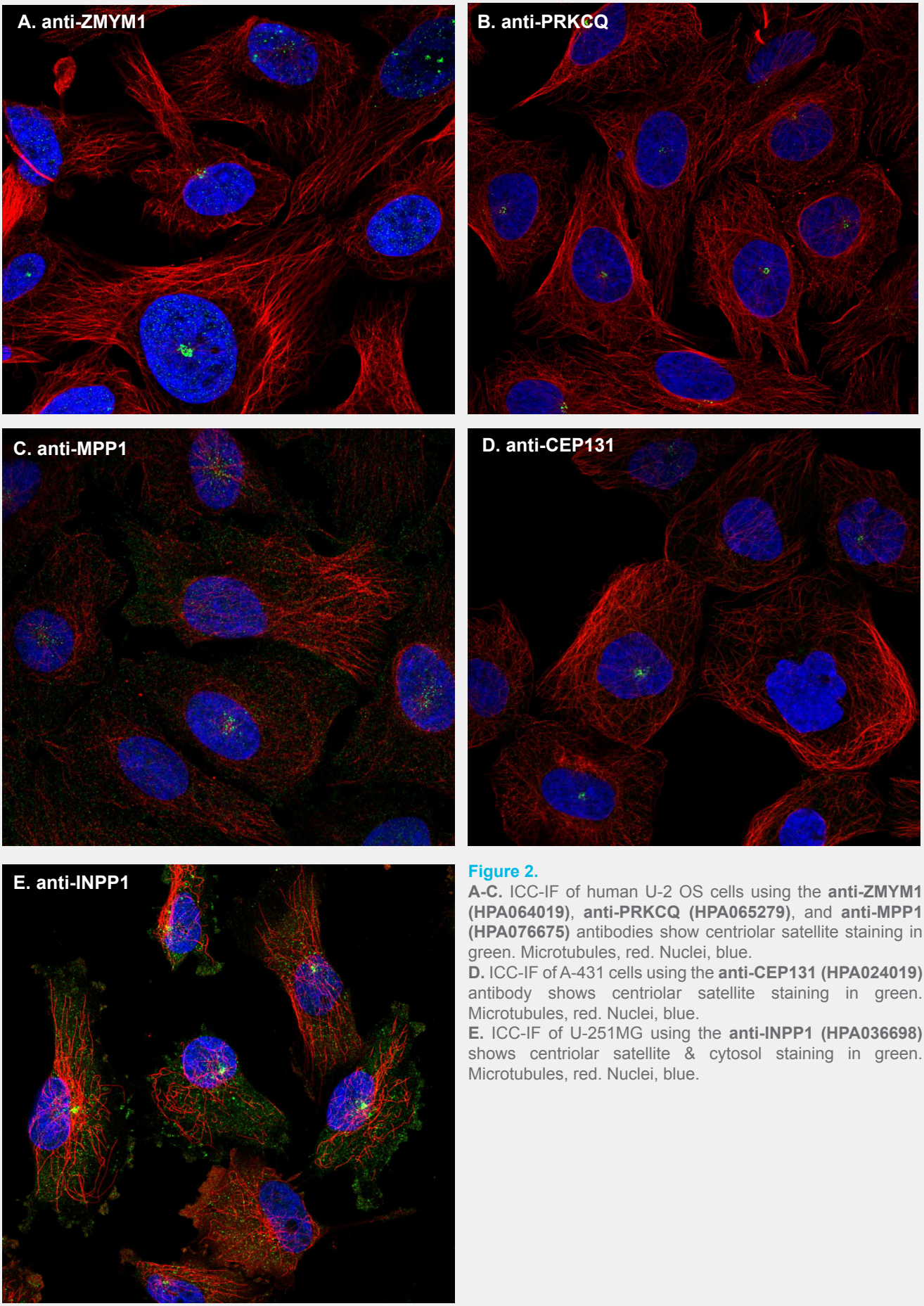


Figure 2.
A-C. ICC-IF of human U-2 OS cells using the **anti-ZMYM1 (HPA064019)**, **anti-PRKCQ (HPA065279)**, and **anti-MPP1 (HPA076675)** antibodies show centriolar satellite staining in green. Microtubules, red. Nuclei, blue.
D. ICC-IF of A-431 cells using the **anti-CEP131 (HPA024019)** antibody shows centriolar satellite staining in green. Microtubules, red. Nuclei, blue.
E. ICC-IF of U-251MG using the **anti-INPP1 (HPA036698)** shows centriolar satellite & cytosol staining in green. Microtubules, red. Nuclei, blue.

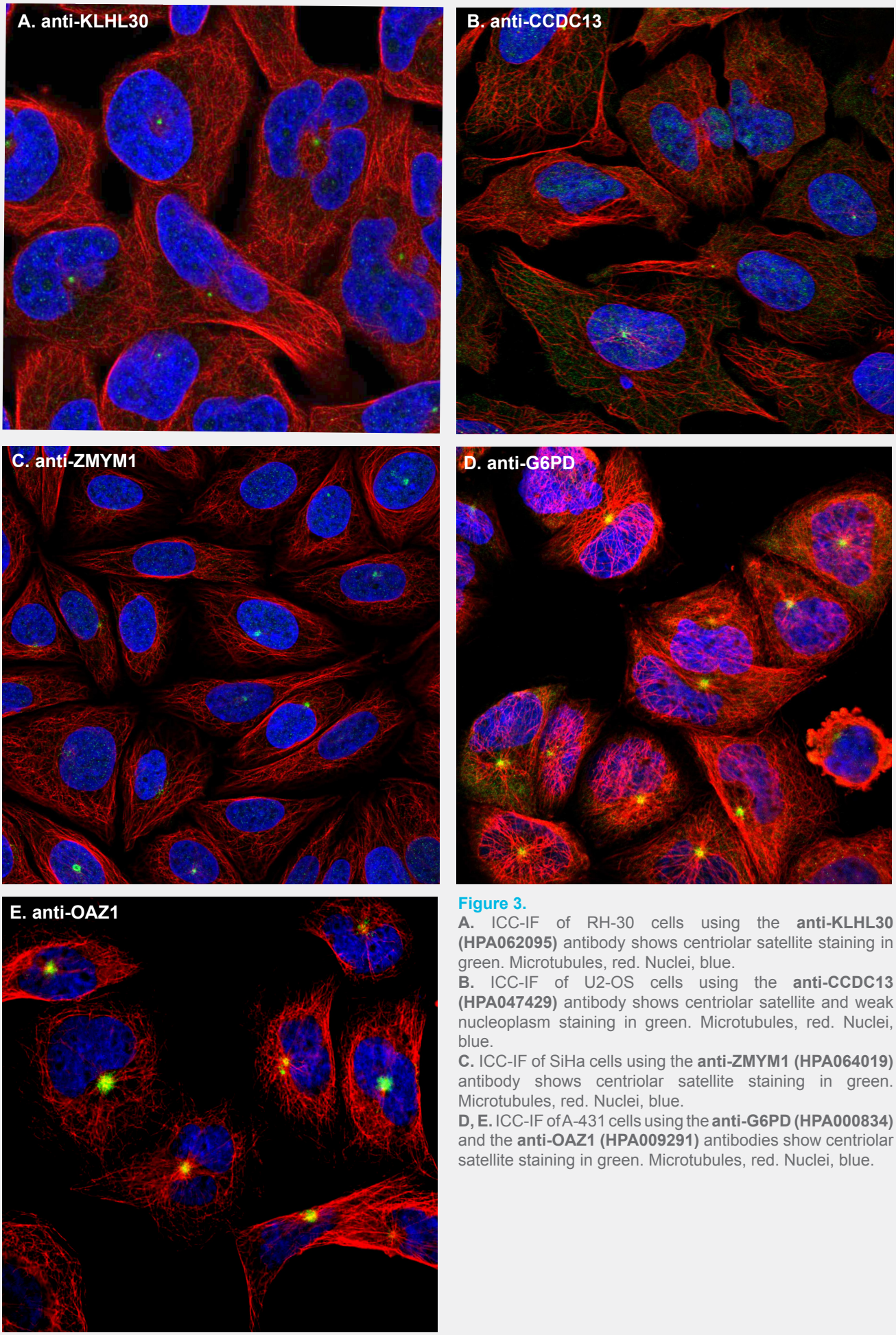


Figure 3.
A. ICC-IF of RH-30 cells using the **anti-KLHL30 (HPA062095)** antibody shows centriolar satellite staining in green. Microtubules, red. Nuclei, blue.
B. ICC-IF of U2-OS cells using the **anti-CCDC13 (HPA047429)** antibody shows centriolar satellite and weak nucleoplasm staining in green. Microtubules, red. Nuclei, blue.
C. ICC-IF of SiHa cells using the **anti-ZMYM1 (HPA064019)** antibody shows centriolar satellite staining in green. Microtubules, red. Nuclei, blue.
D, E. ICC-IF of A-431 cells using the **anti-G6PD (HPA000834)** and the **anti-OAZ1 (HPA009291)** antibodies show centriolar satellite staining in green. Microtubules, red. Nuclei, blue.

Centriolar satellites disease involvement

Centriolar satellites are crucial regulators of a wide range of cellular processes. Research on centriolar satellites focuses on how important they are in centrosome activities such as centrosome maintenance and centriole duplication.

Scientists examine mice deficient in centriolar satellite formation and the relationship with the organelle. In addition, they are looking at cell lines from patients with protein aggregation disorders to see if there are effects on the composition and function of the centriolar satellite.

Centriolar satellites contain numerous proteins involved in a variety of mechanisms that can go wrong, thus causing ciliopathic diseases, carcinogenesis, neurogenesis, and other diseases such as dwarfisms and microcephaly (Firat-Karalar 2014; Ge 2010; Gupta 2015; Mahmood 2011, Rauch 2008, Silva 2016; Stowe 2012; Ye 2014).

There are dozens of publications reporting the involvement of centriolar satellite proteins in diverse kinds of diseases. Table 1. summarizes some examples.

Centriolar satellites are required for efficient ciliogenesis and ciliary content regulation.

Centriolar satellites play pivotal roles in centrosome assembly and primary cilium formation through the delivery of centriolar/centrosomal components from the cytoplasm to the centrosome.

Odabasi et al. (2019) generated a kidney epithelial cell line (IMCD3) lacking satellites using CRISPR/Cas9-mediated PCM1 deletion and investigated the cellular and molecular consequences of satellite loss. They found that cells lacking satellites could still form full-length cilia, although at significantly lower numbers, with changes in the centrosomal and cellular levels of key ciliogenesis factors. Surprisingly, other functions of satellites, namely proliferation, cell cycle progression, and centriole duplication, were unaffected in these cells.

Recent studies implicate primary cilium proteins in the etiologies of various polycystic kidney diseases. The mammalian serine/threonine kinases, Nek1 are involved in primary cilium formation. Mice with NEK1 mutation suffer from polycystic kidney diseases, suggesting that NEK1 may be engaged in cilium control. Moreover, NEK1 overexpression inhibits ciliogenesis (Shalom 2008).

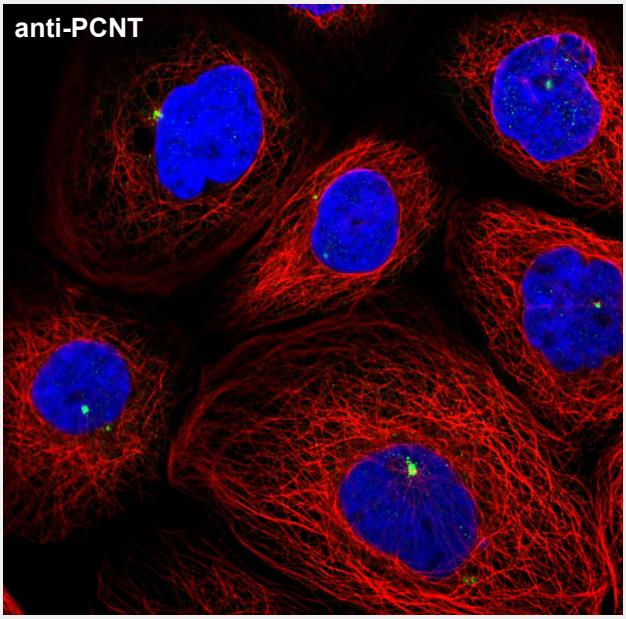


Figure 4.
ICC-IF staining of human A-431 cell line using the **anti-PCNT (HPA016820)** antibody shows positivity to the centrosome, in green. Microtubules, red. Nuclei, blue.

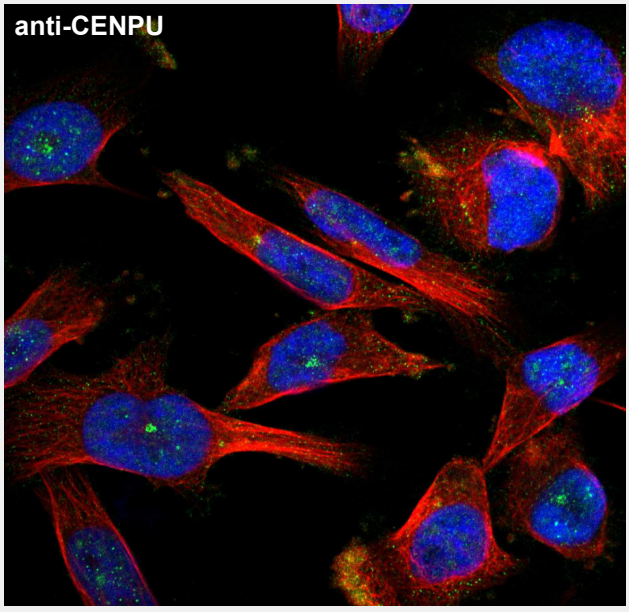


Figure 5.
ICC-IF staining of human U-251M cell line using the **anti-CENPU (HPA022048)** antibody shows positivity to the centrosome and nucleoplasm, in green. Microtubules, red. Nuclei, blue.

Centriolar proteins as tumor suppressor

DMBT1 (the gene deleted in malignant brain tumors 1) located in the centriolar satellite is an intracellular, secreted protein considered a candidate tumor suppressor gene for brain, lung, esophageal, gastric, and colorectal cancers.

For example, in gastric cancer, DMBT1 may mediate mucosal protection, reducing the risk of developing gastric precancerous lesions. However, the increased expression in human gastric precancerous lesions points to a more complex role of DMBT1 in gastric carcinogenesis (Garay 2017).

Dysregulation of LATS2 (large tumor suppressor 2) gene functions has been found in different tumors. As a core kinase in the Hippo pathway, LATS2 plays a pivotal role in organ size control and tumor suppression by restricting proliferation and promoting apoptosis. LATS2 is a putative tumor suppressor gene with potential roles in regulating cell proliferation and apoptosis in lung cancer (Luo, 2014).

Moreover, LATS2 is downregulated in gliomas and exhibits a negative correlation with the prognosis of glioma: over-expression of LATS2 inhibited glioma cell proliferation and migration/invasion, while LATS2 downregulation promoted them (Shi 2019). Furthermore, data from a genome-wide array-based comparative genomic hybridization analysis show that LATS2 can act as a tumor suppressor gene for malignant mesothelioma (an aggressive neoplasm associated with asbestos exposure) (Murakami 2011).

Chromosomal aberrations involving the PCM1 gene are associated with papillary thyroid carcinomas and a variety of hematological malignancies, including atypical chronic myeloid leukemia and T-cell lymphoma. For example, PCM1 had a significantly lower expression in primary ovarian carcinoma than controls promoting PCM1 as a potential tumor suppressor (Pils 2005).

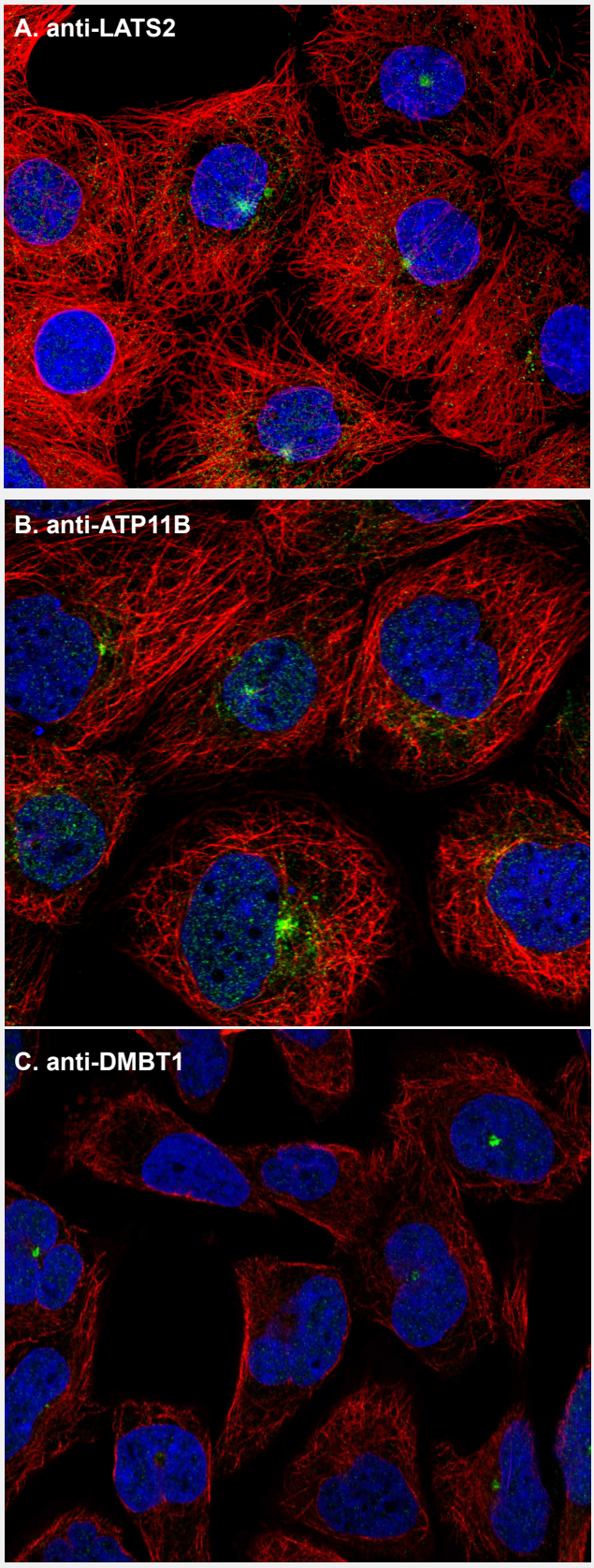


Figure 6.
A,B. ICC-IF of human A-431 cells using the **anti-LATS2 (HPA049037)** and the **anti-ATP11B (HPA036237)** antibodies show centriolar satellite stainings in green. Microtubules, red. Nuclei, blue.
C. ICC-IF of human RH-30 cells using the **anti-DMBT1 (HPA040778)** antibody shows centriolar satellite staining in green. Microtubules, red. Nuclei, blue.

Table 1. Centriolar satellites disease involvement

Gene	Protein Name	Protein Location	Associated Disease
CCDC65	Coiled-coil domain containing 65	Centriolar satellite Vescicles	Ciliopathy, Kartagener syndrome, Primary ciliary dyskinesia
CD2AP	CD2 associated protein	Centriolar satellite Plasma membrane	Neurodegeneration, Alzheimer disease
DDHD2	DDHD domain containing 2	Centriolar satellite Cytosol	Hereditary spastic paraplegia, Neurodegeneration
DMBT1	Deleted in malignant brain tumors 1	Centriolar satellite	Tumor suppressor
DNAL1	Dynein axonemal light chain 1	Centriolar satellite Nucleoplasm	Ciliopathy, Disease variant, Kartagener syndrome, Primary ciliary dyskinesia
DYSF	Dysferlin	Centriolar satellite Plasma membrane	Disease variant, Limb-girdle muscular dystrophy
G6PD	Glucose-6-phosphate dehydrogenase	Centriolar satellite Cytosol	Cancer-related genes, Hereditary hemolytic anemia
KIF1A	Kinesin family member 1A	Centriolar satellite Cytosol	Hereditary spastic paraplegia, Mental retardation, Neurodegeneration, Neuropathy
KIF5B	Kinesin family member 5B	Centriolar satellite Cytosol	Cancer-related genes
IFT43	Intraflagellar transport 43	Centriolar satellite Microtubules	Ciliopathy, Disease variant, Ectodermal dysplasia, Retinitis pigmentosa
LAS1L	LAS1 like ribosome biogenesis factor	Centriolar satellite Nucleoplasm Cytosol	Disease variant, Mental retardation, Obesity
LATS2	Large tumor suppressor kinase 2	Centriolar satellite Cytosol	Tumor suppressor
MID1	Midline 1	Centriolar satellite Cytosol	Disease mutation
NEK1	NIMA related kinase 1	Centriolar satellite Nucleoplasm Cytosol	Amyotrophic lateral sclerosis, Ciliopathy, Neurodegeneration
PCM1	Pericentriolar material 1	Centriolar satellite Cytosol	Schizophrenia. Glioblastoma.
PCNT	Pericentrin	Centriolar satellite Centrosome Cytosol	Dwarfism.
PIBF1	Progesterone immunomod binding factor 1	Centriolar satellite	Ciliopathy. Joubert syndrome
PIK3R5	Phosphoinositide-3-kinase, reg sub 5	Centriolar satellite Cytosol	Neurodegeneration
PLA2G6	Phospholipase A2 group VI	Centriolar satellite Cytosol	Dystonia, Neurodegeneration, Parkinson disease
PRKCQ	Protein kinase C theta	Centriolar satellite	Cancer-related genes
SASS6	SAS-6 centriolar assembly protein	Centriolar satellite	Primary microcephaly
TMEM63A	Transmembrane protein 63A	Centriolar satellite Vescicles	Leukodystrophy
TUB	TUB bipartite transcription factor	Centriolar satellite Nucleoli	Obesity
WDR60	WD repeat domain 60	Centriolar satellite Centrosome	Ciliopathy
WDR62	WD repeat domain 62	Centriolar satellite Cytosol	Mental retardation, Primary microcephaly
ZNF408	Zinc finger protein 408	Centriolar satellite Cytosol	Retinitis pigmentosa

Genome-wide association studies have identified several risk factors for neurodegenerative diseases and dementia involving centriolar satellite proteins.

Due to its cerebrovascular role, the centriolar satellite CD2-associated protein (CD2AP) is a leading genetic risk factor for Alzheimer's disease (Liao 2015; Furusawa 2019). Furthermore, the role of CD2AP in maintaining the blood-brain barrier integrity was reported by Cochran et al. (2015), using the anti-CD2AP polyclonal antibody (HPA003326).

Lan et al. (2016) used CRISPR/Cas9 genome editing to generate primary glioblastoma (GBM) cell lines depleted from PCM1. The results suggest that PCM1 plays multiple roles in GBM pathogenesis and that PCM1-associated pathways could be targeted to augment current or future anti-GBM therapies.

A more recent study emphasizes the role of PCM1 in the postnatal brain. Using the anti-PCM1 polyclonal antibody HPA023370, Monroe et al. (2020) support a contributory role for PCM1 in some individuals diagnosed with schizophrenia.

PLA2G6 is involved in parkinsonism-induced dystonia and neurodegeneration (Karkheiran 2015). In addition, PLA2G6 has also been certified as a causative gene in patients with autosomal recessive early-onset Parkinson's disease (Paisán-Ruiz 2010, Shen 2019).

A significantly reduced head circumference characterizes primary microcephaly. Mutations in the centromere proteins genes, such as WDR62 and CENPU, cause primary microcephaly (Mahmood 2011).

Truncating mutation in intracellular phospholipase A1 gene (DDHD2) is involved in hereditary spastic paraplegia with intellectual disabilities (Alrayes 2015).

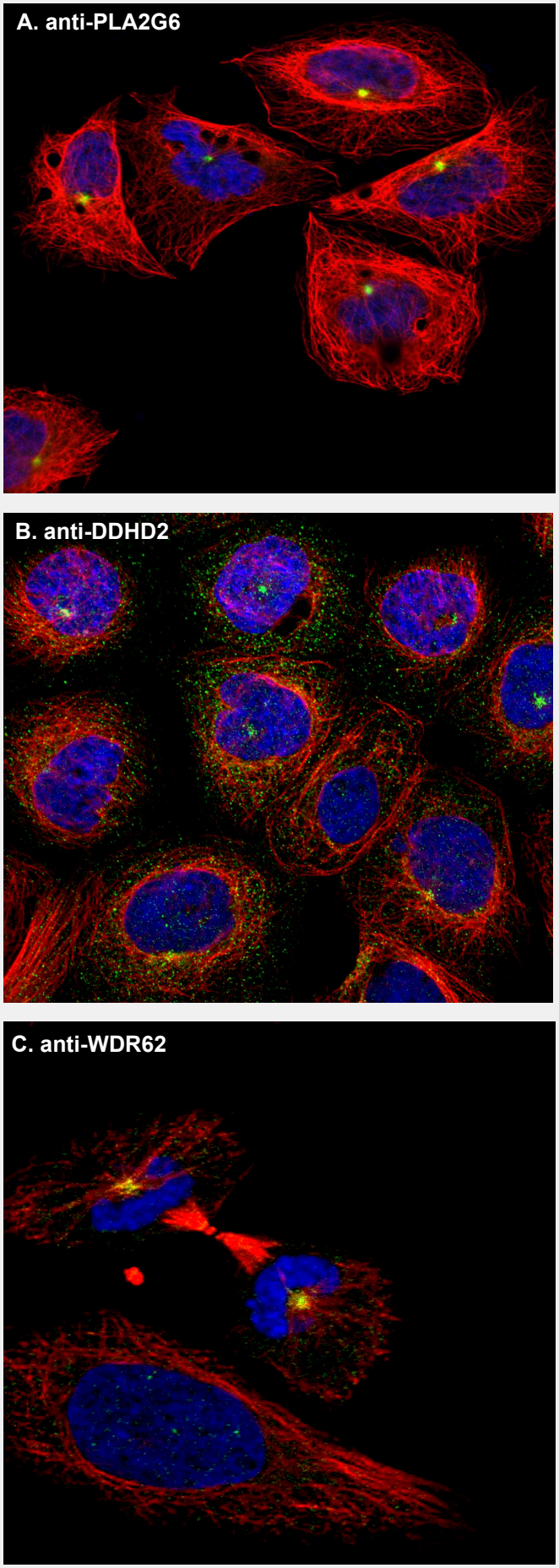


Figure 7.
A. ICC-IF of human A-431 cells using the **anti-PLA2G6 (HPA001171)** antibody show centriolar satellite staining in green. Microtubules in red, nuclei in blue.
B. ICC-IF of human A-431 cells using the **anti-DDHD2 (HPA023147)** antibody show centriolar satellite and cytosol stainings in green. Microtubules in red, nuclei in blue.
C. ICC-IF of human U-2 OS cells using the **anti-WDR62 (HPA043255)** antibody shows centriolar satellite staining in green. Microtubules in red, nuclei in blue.

Table 2. Centriolar satellite antibody markers

Product Name	Protein Name	Other Subcellular Location	Product Number	Validated Applications
Anti-AK5	Adenylate kinase 5	Cytosol	HPA019128	IHC*, WB*, ICC-IF
Anti-ATP11B	ATPase, class VI, type 11B	Cytosol	HPA036237	IHC
Anti-ATP11B	ATPase, class VI, type 11B	Cytosol	HPA036238	IHC
Anti-CCDC13	Coiled-coil domain containing 13	-	HPA047429	IHC, ICC-IF
Anti-CCDC66	Coiled-coil domain containing 66	-	HPA044185	IHC, ICC-IF
Anti-CD2AP	CD2-associated protein	Plasma membrane	HPA003326	IHC*, WB*, ICC-IF
Anti-CENPU	Centromere protein U	Nucleoplasm	HPA022048	IHC, ICC-IF
Anti-CEP97	Centrosomal protein 97kDa	-	HPA002980	IHC*, WB, ICC-IF
Anti-CEP128	Centrosomal protein 128kDa	-	HPA001116	IHC
Anti-CEP131	Centrosomal protein 131kDa	Cytokinetic bridge	HPA024019	IHC, WB, ICC-IF
Anti-CEP290	Centrosomal protein 290kDa	-	HPA064397	ICC-IF
Anti-CNTRL	Centriolin	Cytosol	HPA020468	IHC, WB, ICC-IF
Anti-C7orf43	Chromosome 7 open read frame 43	Plasma membrane & Vescicle	HPA019359	IHC, ICC-IF
Anti-DDHD2	DDHD domain containing 2	Cytosol	HPA023147	IHC, WB, ICC-IF
Anti-DLGAP5	Discs, large homolog-associated prot 5	Cytosol	HPA005546	IHC*, WB*, ICC-IF
Anti-DMBT1	Deleted in malignant brain tumors 1	-	HPA040778	IHC*, ICC-IF
Anti-FNIP2	Folliculin interacting protein 2	Cytosol	HPA052758	ICC-IF
Anti-G6PD	Glucose-6-phosphate dehydrogenase	Cytosol	HPA000247	IHC*, WB*, ICC-IF
Anti-GAS6	Growth arrest-specific 6	Cytosol	HPA008275	IHC
Anti-GKAP1	G kinase anchoring protein 1	Cytosol	HPA035118	IHC*, WB*, ICC-IF
Anti-GPR162	G protein-coupled receptor 162	-	HPA055135	IHC*, WB, ICC-IF
Anti-KCTD4	K+ channel tetramerization domain 4	-	HPA040734	IHC, WB*
Anti-KIF5B	Kinesin family member 5B	Cytosol	HPA037589	IHC, WB*, ICC-IF
Anti-KIRREL2	Kirre-like nephrin family adhesion mol 2	-	HPA071587	ICC-IF
Anti-KLHL30	Kelch-like family member 30	-	HPA062095	ICC-IF
Anti-INPP1	Inositol polyphosphate-1-phosphatase	Cytosol	HPA036699	IHC, ICC-IF
Anti-LATS2	Large tumor suppressor kinase 2	Cytosol	HPA049037	ICC-IF
Anti-LGR4	Leucine-rich repeat GPCR4	-	HPA030267	IHC
Anti-MAPRE1	MAP, RP/EB family, member 1	Cytosol	HPA003600	IHC*, WB*, ICC-IF
Anti-MPP1	Membrane palmitoylated protein 1	Plasma membrane	HPA076675	IHC*, ICC-IF
Anti-MYO5A	Myosin VA	Focal adhesion sites	HPA001356	IHC*, ICC-IF
Anti-NME9	NME/NM23 family member 9	Cytosol	HPA043881	IHC*
Anti-OAZ1	Ornithine decarboxylase antizyme 1	Vesicles	HPA009291	IHC, ICC-IF
Anti-PCM1	Pericentriolar material 1	Cytosol	HPA023370	IHC*, ICC-IF
Anti-PCNT	Pericentrin	Centrosome	HPA016820	IHC*, ICC-IF
Anti-PIBF1	Progest immunomod binding factor 1	-	HPA052269	WB, ICC-IF
Anti-PIK3R5	PIK3R5, p101, P101-PI3K	Cytosol	HPA044505	ICC-IF
Anti-PLA2G6	Phospholipase A2 group VI	-	HPA001171	WB*, ICC-IF
Anti-PRKCQ	Protein kinase C, theta	-	HPA065279	ICC-IF
Anti-PTAR1	Protein prenyltransferase alpha sub repeat containing 1	Rods & Rings	HPA021248	IHC, ICC-IF

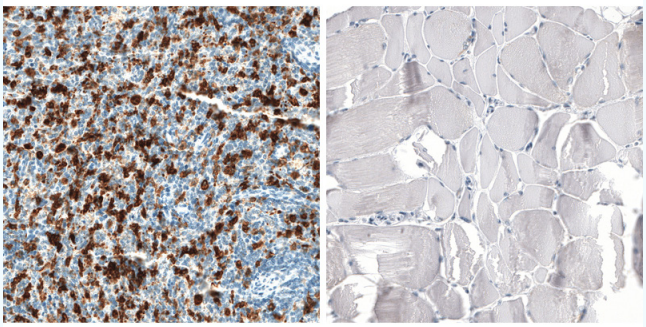
Table 2. (*cont.*)

Product Name	Protein Name	Other Subcellular Location	Product Number	Validated Applications
Anti-PTPN20	Protein tyr-phosp non-receptor type 20	-	HPA069148	IHC*
Anti-RAB3IL1	RAB3A interacting prot (rabin3)-like 1	Cytosol	HPA039723	IHC, ICC-IF
Anti-RAB11FIP3	RAB11 family interacting prot 3 (class II)	Cytokinetic bridge	HPA028631	IHC
Anti-RASSF7	Ras association domain fam member 7	-	HPA078015	ICC-IF
Anti-SLC35D3	Solute carrier family 35 member D3	-	HPA067497	ICC-IF
Anti-SLC9B1	Solute carrier family 9 member B1	Mitochondria	HPA065520	ICC-IF
Anti-SPAG9	Sperm associated antigen 9	Cytosol	HPA040446	IHC*, ICC-IF
Anti-TMEM63A	Transmembrane protein 63A	Vesicles	HPA066504	ICC-IF
Anti-TRAT1	T-cell rec associated transmemb adap 1	Plasma memb & Mitotic spindle	HPA002356	IHC, WB
Anti-TUB	TUB bipartite transcription factor	Nucleoli	HPA017997	IHC, ICC-IF
Anti-WDR60	WD repeat domain 60	-	HPA020607	IHC, ICC-IF
Anti-WDR62	WD repeat domain 62	-	HPA043255	IHC*, ICC-IF
Anti-ZMYM1	Zinc finger MYM-type containing 1	-	HPA064019	ICC-IF
Anti-ZNF408	Zinc finger protein 408	Cytosol	HPA017892	IHC
Anti-ZNF721	Zinc finger protein 721	Cytosol	HPA065313	ICC-IF

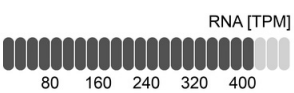
* Products with enhanced validation for indicated application.

ENHANCED VALIDATION: AN ADDITIONAL LAYER OF SECURITY IN ANTIBODY VALIDATION

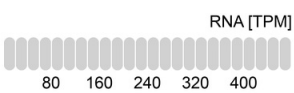
Enhanced Validation



HMOX1 in Spleen



HMOX1 in Skeletal muscle



Enhanced validation offers increased security of antibody specificity in a defined context. By using 5 different enhanced validation methods we validate our antibodies for each combination of protein, sample, and application.

The 5 methods are: genetic validation, orthogonal validation, validation by independent antibodies, recombinant expression validation, and migration capture MS validation.

Left: Example of orthogonal validation of protein expression using IHC by comparison of the staining signal to the RNA-seq data (TPM) of corresponding target in high and low expression tissues. The image shows the immunohistochemistry analysis in human spleen and skeletal muscle tissues using the Anti-HMOX1 (AMAb91719) antibody. Corresponding HMOX1 RNA-seq data (TPM) are presented for the same tissues

References

Alrayes N, et al. Truncating mutation in intracellular phospholipase A1 gene (DDHD2) in hereditary spastic paraplegia with intellectual disability (SPG54). BMC Res Notes 2015:8, 271

Balczon R, et al. PCM1, A 228-kD centrosome autoantigen with a distinct cell cycle distribution. J Cell Biol. 1994 Mar;124(5):783-93

Baron AT, et al. Identification and localization of a novel, cytoskeletal, centrosome-associated protein in PtK2 cells. J Cell Biol. 1988 Dec;107(6 Pt 2):2669-78

Bergmann O, et al. Evidence for cardiomyocyte renewal in humans. Science. 2009 Apr 3;324(5923):98-102

Bergmann O, et al. Isolation of cardiomyocyte nuclei from post-mortem tissue. J Vis Exp. 2012 Jul 10;(65):4205

Bärenz F, et al. Review: Centriolar satellites: busy orbits around the centrosome. Eur J Cell Biol. 2011 Dec; 90(12):983-9

Cochran et al. The Alzheimer's disease risk factor CD2AP maintains blood–brain barrier integrity. Hum Mol Genet, 2015 Sep 10; 24(23):6667-6674

Dammermann A, et al. Assembly of centrosomal proteins and microtubule organization depends on PCM-1. J Cell Biol. 2002;159:255–266

Firat-Karalar EN, et al. Proximity interactions among centrosome components identify regulators of centriole duplication. Curr Biol 2014; 24, 664–670

Furusawa K, et al. CD2-associated protein (CD2AP) overexpression accelerates amyloid precursor protein (APP) transfer from early endosomes to the lysosomal degradation pathway**.** J Biol Chem, 2019 May 28; 294(28):10886-10899

Garay J, et al. Increased expression of deleted in malignant brain tumors (DMBT1) gene in precancerous gastric lesions: findings from human and animal studies. Oncotarget vol. 8,29 (2017): 47076-47089

Ge X, et al. Hook3 interacts with PCM1 to regulate pericentriolar material assembly and the timing of neurogenesis. Neuron. 2010 Jan 28;65(2):191-203

Gheiratmand L, et al. Spatial and proteomic profiling reveals centrosome-independent features of centriolar satellites. EMBO J. 2019 Jul 15;38(14):e101109

Gupta GD, et al. A dynamic protein interaction landscape of the human centrosome-cilium interface. Cell. 2015 Dec 3;163(6):1484-99

Hames RS, et al. Dynamic recruitment of Nek2 kinase to the centrosome involves microtubules, PCM1, and localized proteasomal degradation. *Mol Biol Cell*. 2005;16(4):1711-1724

Karkheiran S, et al. PLA2G6-associated dystonia-parkinsonism: case report and literature review. Tremor & Other Hyperkinet Mov (NY); 2015;5:317

Kodani A, et al. Centriolar satellites assemble centrosomal microcephaly proteins to recruit CDK2 and promote centriole duplication. E-life. 2015 Aug 22; 4

Kubo A, et al. Centriolar satellites: molecular characterization, ATP-dependent movement toward centrioles and possible involvement in ciliogenesis. J Cell Biol. 1999, Nov 29;147(5):969-80. Erratum in: J Cell Biol 1999 Dec 27;147(7):1585

Kubo A, et al. Non-membranous granular organelle consisting of PCM-1: subcellular distribution and cell-cycle-dependent assembly/disassembly. J Cell Sci. 2003 Mar 1;116(Pt 5):919-28

Hirai M, et al. Revisiting preadolescent cardiomyocyte proliferation in mice. Circ Res. 2016;118(6):916-919

Hori A, et al. Regulation of centriolar satellite integrity and its physiology. Cell Mol Life Sci. 2017;74(2):213-229

Lan B Hoang-Minh, et al. PCM1 depletion inhibits glioblastoma cell ciliogenesis and increases cell death and sensitivity to temozolomide. *Translational oncology* vol. 9,5 (2016): 392-402

Liao F, et al. Effects of CD2-associated protein deficiency on amyloid-β in neuroblastoma cells and in an APP transgenic mouse model. Mol. Neurodegener, 10 (2015),12

Lopes CA, et al. Centriolar satellites are assembly points for proteins implicated in human ciliopathies, including oral-facial-digital syndrome 1. J Cell Sci. 2011 Feb 15; 124(4):600-12

Luo SY, et al. Aberrant large tumor suppressor 2 (LATS2) gene expression correlates with EGFR mutation and survival in lung adenocarcinomas. Lung Cancer. 2014;85(2):282-292

Mahmood S, et al. Autosomal recessive primary microcephaly (MCPH): clinical manifestations, genetic heterogeneity and mutation continuum. Orphanet J Rare Dis. 2011;6:39

Monroe TO, et al. PCM1 is necessary for focal ciliary integrity and is a candidate for severe schizophrenia. Nat Commun, 2020 Nov 19; 11:5903

Murakami H, et al. LATS2 is a tumor suppressor gene of malignant mesothelioma. Cancer Res 2011; 71(3) 873-883

Odabasi E, et al. Centriolar satellites are required for efficient ciliogenesis and ciliary content regulation. EMBO Rep. 2019;20(6):e47723

Paisán-Ruiz C, et al. Early-onset L-dopa-responsive parkinsonism with pyramidal signs due to ATP13A2, PLA2G6, FBXO7 and spatacsin mutations. Mov Disord. 2010 Sep 15; 25(12):1791-800

Pils D, et al. Five genes from chromosomal band 8p22 are significantly down-regulated in ovarian carcinoma: N33 and EFA6R have a potential impact on overall survival. Cancer. 2005 Dec 1;104(11):2417-29

Preissl S, et al. Deciphering the epigenetic code of cardiac myocyte Transcription. Circ Res. 2015 Aug 14;117(5):413-23.

Rauch A, et al. Mutations in the pericentrin (PCNT) gene cause primordial dwarfism. Science. 2008 Feb 8;319(5864):816-9

Sang L, et al. Mapping the NPHP-JBTS-MKS protein network reveals ciliopathy disease genes and pathways. Cell. 2011 May 13; 145(4):513-28

Silva E, et al. Ccdc11 is a novel centriolar satellite protein essential for ciliogenesis and establishment of left-right asymmetry. Mol Biol Cell. 2016 Jan 1;27(1):48-63

Stephen LA. et al. TALPID3 controls centrosome and cell polarity and the human ortholog KIAA0586 is mutated in Joubert syndrome (JBTS23). Elife. 2015 Sep 19;4

Stowe TR, et al. The centriolar satellite proteins Cep72 and Cep290 interact and are required for recruitment of BBS proteins to the cilium. Mol Biol Cell. 2012 Sep;23(17):3322-35

Shalom O, et al. The mammalian Nek1 kinase is involved in primary cilium formation. FEBS Lett. 2008 Apr 30;582(10):1465-70

Shen T, et al. Early-onset Parkinson's disease caused by PLA2G6 compound heterozygous mutation, a case report and literature review. Front Neurol. 2019;10:915

Shi Y, et al. LATS2 inhibits malignant behaviors of glioma cells via inactivating YAP. J Mol Neurosci. 2019 May;68(1):38-48

Ye X, et al. C2cd3 is critical for centriolar distal appendage assembly and ciliary vesicle docking in mammals. PNAS USA. 2014 Feb 11;111(6):2164-9

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