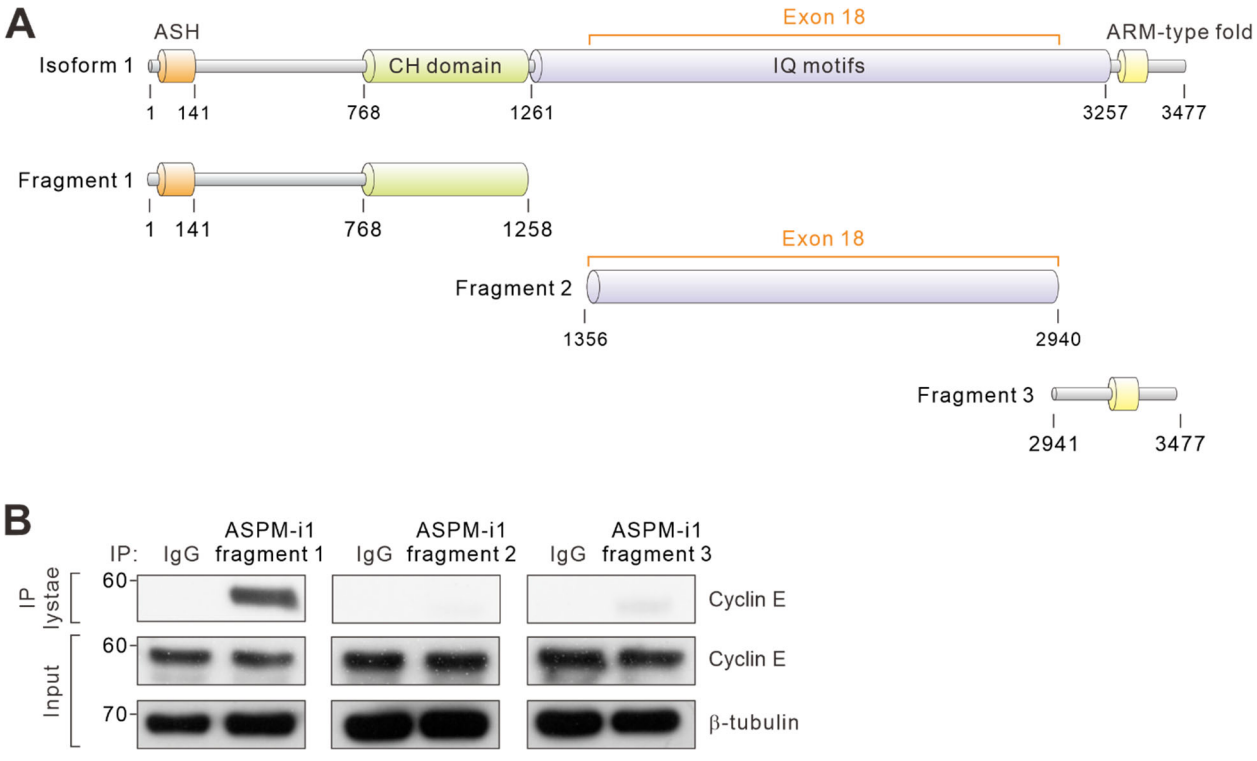
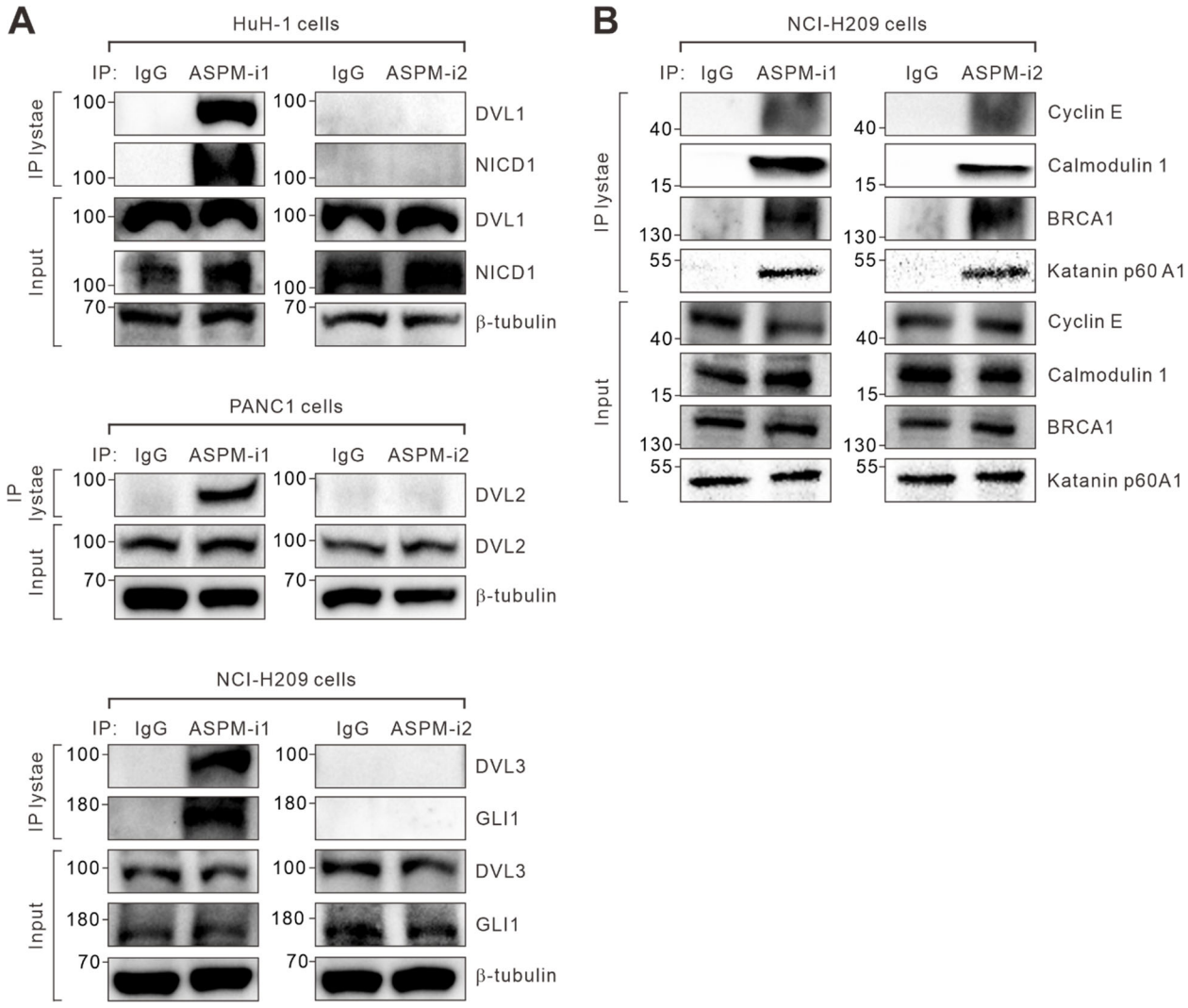
Supplementary Figure S1



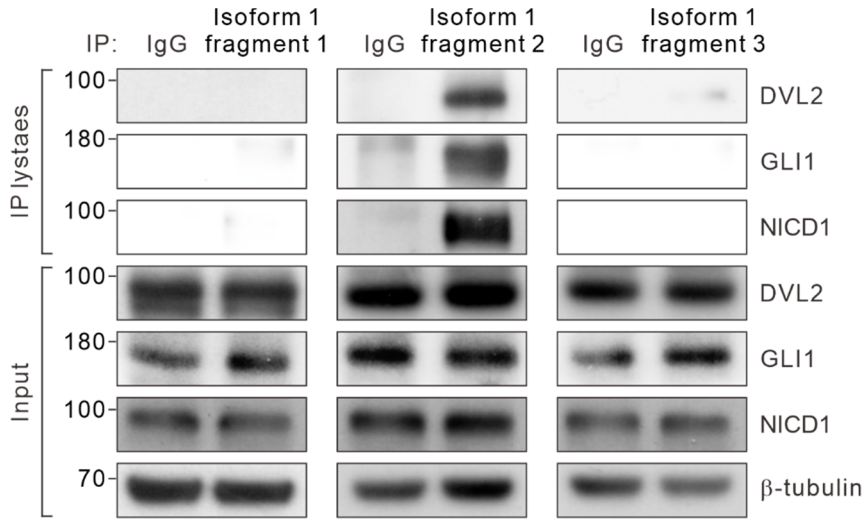
Supplementary Figure 1. ASPM isoform 1 interacts with cyclin E through the amino-terminal region. A, Schematics representing the different ASPM isoform 1 (ASPM-i1) protein segments, including the amino-terminal region (fragment 1; amino acids 1-1258), the exon-18-encoded segment (fragment 2; amino acids 1356-2940), and the carboxy-terminal region (fragment 3; amino acids 2941-3477). **B,** Representative co-immunoprecipitation (IP) analyses demonstrating that ASPM-i1 interacts with cyclin E through the amino-terminal region (*i.e.*, fragment 1 in (**A**)) in MDA-MB-436 cancer cells. β-tubulin was included as a loading control. Note that the exon-18-encoded segment (*i.e.*, fragment 2) or the carboxy-terminal region of ASPM-i1 (*i.e.*, fragment 3) does not interact with cyclin E.

Supplementary Figure S2



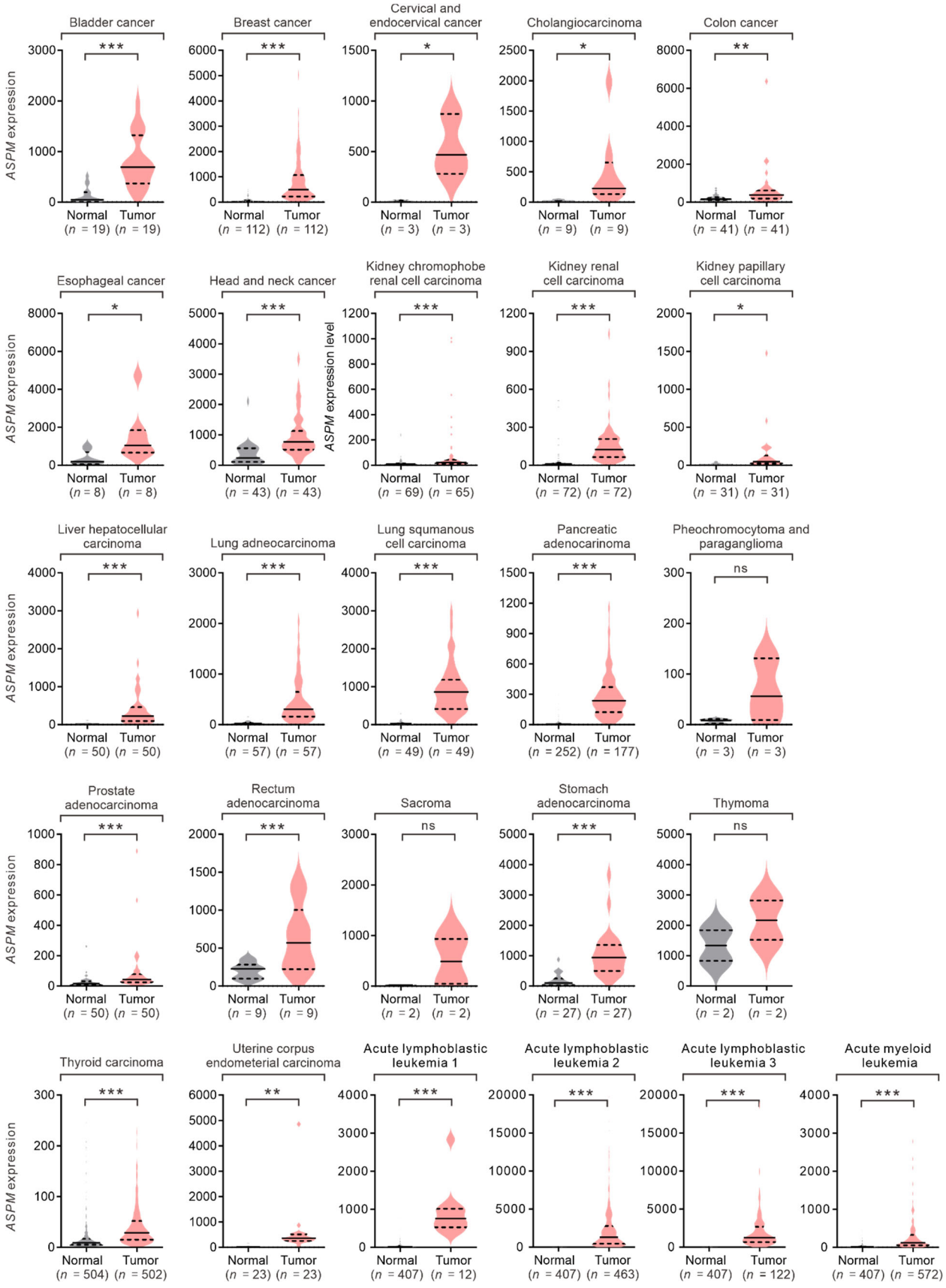
Supplementary Figure 2. The binding partners of ASPM isoforms 1 and 2 in cancer cells. A, Representative co-immunoprecipitation (IP) analyses demonstrating that endogenous ASPM isoform 1 (ASPM-i1), but not ASPM isoform 2 (ASPM-i2), associates with DVL proteins, including DVL1, DVL2, and DVL3, and GLI1 and NOTCH1 Intracellular Domain (NICD1) in HuH-1 (hepatocellular carcinoma), PANC1 (pancreatic ductal adenocarcinoma), or NCI-H209 (small cell lung cancer) cells. β-tubulin was included as loading controls. **B,** Representative co-IP analyses demonstrating that both endogenous ASPM-i1 and ASPM-i2 associate with cyclin E (involved in the cell cycle), calmodulin 1 (involved in calcium signal transduction), BRCA1 (involved in DNA damage repair), and katanin (involved in mitosis) in NCI-H209 cancer cells.

Supplementary Figure S3



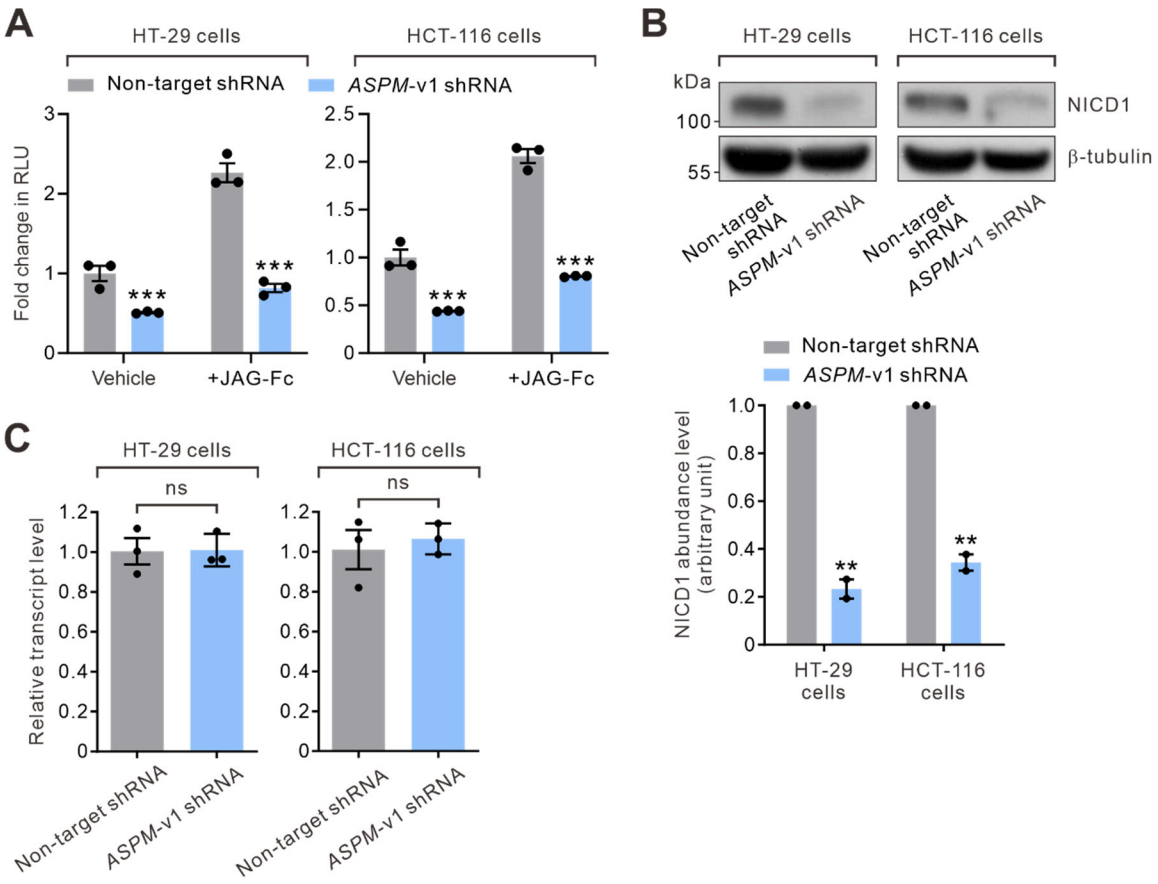
Supplementary Figure 3. ASPM isoform 1 interacts with DVL2, GLI1, and NOTCH1 Intracellular Domain (NICD1) through its exon-18-encoded segment. Shown are representative co-immunoprecipitation (IP) analyses demonstrating that the exon-18-encoded region (*i.e.*, fragment 2 in Supplementary Fig. S1A) of ASPM-i1 specifically interacts with DVL2, GLI1, and NICD1 in MDA-MB-436 cancer cells. β-tubulin was included as a loading control.

Supplementary Figure S4



Supplementary Figure 4. *ASPM* is overexpressed in most human solid tumors and leukemia. Shown are violin plots demonstrating the expression level of *ASPM* in normal and cancer tissues based on the RNA sequencing data downloaded from TNMplot (https://tnmplot.com/analysis/). Data represent paired normal and tumor tissues except acute lymphoblastic leukemia, acute myeloid leukemia, kidney chromophobe renal cell carcinoma, pancreatic adenocarcinoma, and thyroid carcinoma, in which the expression level of *ASPM* in normal tissues was included. Solid bar, mean. Dashed bar, first and third quartile cutoff values. ns, not significant, $*P < 0.05$; $**P < 0.01$; $***P < 0.001$, two-tailed unpaired *t*-test

Supplementary Figure S5



Supplementary Figure 5. ASPM isoform 1 regulates the Notch pathway activity and the protein abundance level of NOTCH1 Intracellular Domain (NICD1) in cancer cells. A, Relative Notch-specific luciferase activity (relative light unit; RLU) in HT-29 and HTC-116 cancer cells without (non-target) or with small hairpin RNA (shRNA)-mediated knockdown of *ASPM* variant 1 (*ASPM*-v1; encoding ASPM isoform 1) expression and stimulated with the activating Notch ligand JAG1-Fc (5 μg/ml for 24 hours). *n* = 3 independent experiments. **B**, Immunoblotting analysis showing the protein abundance level of NICD1 in HT-29 or HCT-116 cells without (non-target) or with shRNA-mediated knockdown of *ASPM*-v1 expression (left). The arbitrary densitometry unit of the immunoblots is shown at the bottom. β-tubulin was included as a loading control. *n* = 2 independent experiments. **C**, The transcript level of *NOTCH1* in HCT-116 and HT-29 cells with shRNA-mediated knockdown of *ASPM*-v1 expression as measured by qRT-PCR. *n* = 3 independent experiments. The lentivirus shRNA construct #4 used to knock down *ASPM*-v1 expression was described previously (1). The knockdown of *ASPM*-v1 expression did not significantly affect the transcript level of *NOTCH1*. Data represent

mean ± SEM. ns, not significant, $^{**}P < 0.01$; $^{***}P < 0.001$ compared with non-target shRNA; two-tailed unpaired *t*-test.

Supplementary Table 1. The expression pattern of ASPM and its prognostic significance in cancers

Cancer type	Gene/ protein	Assay	Expression level compared with normal tissue	Correlation with clinical variables	Prognostic correlation	References
Bladder cancer	*ASPM*	GEP, RNA-seq	Elevated	Tumor size, grade, node status, TNM stage	OS, PFS	(2)
	ASPM	qRT-PCR	Elevated	Muscle invasion, tumor grade, size, clinical stage, metastasis	OS, PFS	(3)
	ASPM	GEP, qRT-PCR	Elevated		OS	(4)
Breast cancer	*ASPM*/ ASPM	GEP, IHC	Elevated	Tumor size, grade, clinical stage	OS, RFS	(5)
Cholangio-carcinoma	*ASPM*	RNA-seq	Elevated			(6)
CRC	ASPM	IHC	Elevated	Clinical stage, lymph node metastasis		(7)
DLBCL	ASPM	IHC	Elevated		OS	(8)
Esophageal cancer	*ASPM*	RNA-seq, GEP	Elevated			(9)
Gastric cancer	*ASPM*/ ASPM	GEP, IHC	Elevated			(10)
	ASPM-i1	IHC	Elevated		OS	(11)

Glioblastoma	*ASPM*	qRT-PCR	Elevated	Tumor grade, recurrence		(12)
HCC	*ASPM*	RT-PCR	Elevated	AFP, tumor grade, stage, early tumor recurrence	OS	(13)
	ASPM	GEP	Elevated	Disease progression from cirrhosis to HCC		(14)
	ASPM	RNA-seq	Elevated		OS, RFS	(15)
	ASPM	IHC			OS	(16)
Lung squamous cell carcinoma	ASPM	IHC	Elevated	Tumor size, clinical stage	OS, PFS	(17)
Lung adenocarcinoma	*ASPM*/ ASPM	RNA-seq, GEP, IHC, IB	Elevated	Pathological tumor status, lymph node status, differentiation status, clinical stage	OS, PFS	(18)
Osteosarcoma	ASPM	IHC	Elevated	Tumor size, clinical stage		(19)
Ovarian cancer	*ASPM*	qRT-PCR	Elevated			(20)
	ASPM	IHC	Elevated	Tumor grade, clinical stage	OS, PFS	(21)
PDAC	*ASPM*/ ASPM	GEP, IHC	Elevated		OS	(22)
	ASPM	GEP	Elevated	Tumor grade		(23)

	ASPM-i1	IHC	Elevated		OS	(24)
Prostate cancer	*ASPM*/ ASPM	GEP, IHC	Elevated	Gleason score, PSA, clinical stage, tumor grade	OS, Biochemical (PSA) recurrence-free survival	(25)
	ASPM	IHC	Elevated	Seminal vesicle invasion, Gleason score, cribriform and intraductal carcinoma patterns	RFS	(26)
SCLC	*ASPM*-v1/ ASPM-i1	GEP, IHC	Elevated		OS	(1)
Uterine cancers	*ASPM*	qRT-PCR	Elevated			(20)

ASPM-i1, ASPM isoform 1; *ASPM*-v1, *ASPM* variant 1; CRC, colorectal cancer; DLBCL, diffuse large B-cell lymphoma; GEP, gene-expression profiling by microarray; HCC, hepatocellular carcinoma; IB, immunoblotting; IHC, immunohistochemistry; OS, overall survival; PDAC, pancreatic ductal-adenocarcinoma; PFS, progression-free survival; PSA, prostate specific antigen; qRT-PCR, quantitative reverse transcription polymerase chain reaction; RFS, relapse-free survival; RNA-seq, RNA sequencing; SCLC, small cell lung cancer.

Supplementary Table 2. Functional studies implicating the oncogenic role of ASPM

Cancer type	Gene	Approach	Type of study	Findings	References
CRC	*ASPM*	shRNA-mediated KD	*In vitro*; *in vivo*	Reduced migration and invasion of cancer cells; reduced pulmonary metastasis	(27)
Gastric cancer	*ASPM*-v1	shRNA-mediated KD	*In vitro*	Reduced tumorsphere formation	(11)
Glioblastoma	*ASPM*	siRNA- and shRNA-mediated KD	*In vitro*; *in vivo*	G0/G1 cell cycle arrest and reduced proliferation of cancer cells; reduced growth of xenograft tumors	(28)
	ASPM	shRNA-mediated KD	*In vitro*	Increased cell death; reduced cell proliferation and secondary sphere formation	(12)
	ASPM	siRNA-mediated KD	*In vitro*	Reduced proliferation and secondary sphere formation	(29)
	ASPM	shRNA-mediated KD	*In vitro*; *in vivo*	Reduced proliferation of cancer cells; reduced growth of xenograft tumors	(30)
HCC	*ASPM*	shRNA-mediated KD	*In vivo*	Reduced frequency of tumor-initiating cells; reduced growth of xenograft tumors	(16)
	ASPM	siRNA-mediated KD	*In vitro*	Reduced proliferation, migration and invasion of cancer cells	(15)

	ASPM	shRNA-mediated KD	*In vitro*; *in vivo*	Reduced proliferation, migration and invasion of cancer cells; reduced growth of xenograft tumors	(31)
NSCLC	*ASPM*	siRNA- and shRNA-mediated KD	*In vitro*; *in vivo*	Reduced proliferation, migration and invasion of cancer cells; reduced growth of xenograft tumors	(18)
	ASPM	siRNA-mediated KD; lentivirus-mediated OE	*In vitro*	Reduced invasion of cancer cells (KD); enhanced invasion of cancer cells (OE)	(32)
Osteosarcoma	*ASPM*	shRNA-mediated KD	*In vitro*; *in vivo*	Reduced proliferation of cancer cells; reduced growth of xenograft tumors	(19)
Ovarian cancer	*ASPM*	siRNA-mediated KD	*In vitro*	Reduced proliferation and migration of cancer cells	(21)
PDAC	*ASPM*	shRNA-mediated KD	*In vitro*; *in vivo*	Reduced proliferation and migration of cancer cells; reduced growth of xenograft tumors	(22)
	ASPM-v1	shRNA-mediated KD	*In vitro*	Reduced tumorsphere formation	(24)
Prostate cancer	*ASPM*	shRNA-mediated KD	*In vitro*; *in vivo*	Reduced proliferation and invasion of cancer cells; reduced tumorsphere formation; reduced growth of xenograft tumors	(26)
SCLC	*ASPM*-v1	shRNA-mediated KD	*In vitro*; *in vivo*	Reduced proliferation and invasion of cancer cells; reduced tumorsphere	(1)

				formation; reduced growth of xenograft tumors	
Thyroid carcinoma	*ASPM*	CRISPR/Cas9-mediated KO	*In vitro*; *in vivo*	Reduced migration and invasion of cancer cells; reduced growth of xenograft tumors	(33)

ASPM-v1, *ASPM* variant 1; CRC, colorectal cancer; HCC, hepatocellular carcinoma; KD, knockdown; KO, knockout; NSCLC, non-small cell lung cancer; OE, overexpression; PDAC, pancreatic ductal adenocarcinoma; SCLC, small cell lung cancer; shRNA, short hairpin RNA; siRNA, small interfering RNA.

SUPPLEMENTARY MATERIALS AND METHODS

Cell culture

PANC-1, MDA-MB-436 (American Type Culture Collection; RRID:CVCL_0480 and RRID:CVCL_0623), and HuH-1 cells (Japanese Collection of Research Bioresources; RRID:CVCL_2956) were maintained in DMEM (Invitrogen) supplemented with 10% fetal bovine serum and antibiotics. NCI-H209 cells (American Type Culture Collection; RRID:CVCL_1525) were maintained in HITES medium consisting of DMEM:F12 medium (Invitrogen), 5% fetal bovine serum, 0.005 mg/ml insulin, 0.01 mg/ml transferrin, 30 nM sodium selenite, 10 nM hydrocortisone, 10 nM β-estrodiol, and 2 mM L-glutamine. HT-29 and HCT-116 (American Type Culture Collection; RRID:CVCL_0320 and RRID:CVCL_AS10) were maintained in McCoy's 5a (Invitrogen) supplemented with 10% fetal bovine serum and antibiotics.

Immunoblot (IB) and co-immunoprecipitation (IP)

IB analysis and co-IP experiments were performed according to standard protocols. Antibodies used for IB experiments include anti-dishevelled (DVL)-1 (Santa Cruz; 3F12, RRID:AB_627430), anti-DVL2 (RRID: AB_2093330), anti-DVL3 (Proteintech, RRID:AB_2093451), anti-cyclin E (Merck, HE12, RRID: AB_2071085), anti-GLI1 (GeneTex; HL247, RRID: AB_2888543), anti-BRCA1 (GeneTex, RRID: AB_2888543), anti-katanin p60 A1 (Proteintech, RRID: AB_10694670), and anti-cleaved Notch1 (NICD1) (Cell signaling, D3B8, RRID: AB_2153348). The rabbit polyclonal antibodies specifically detecting ASPM isoform 1 (ASPM-i1; NCBI RefSeq: NP_060606.3) or isoform 2 (ASPM-i2; NCBI RefSeq: NP_001193775.1) were described previously (24). A goat anti-rabbit IgG (Jackson ImmunoResearch, RRID:AB_2337913) was used in conjunction with the polyclonal antibodies raised for the immune detection of the ASPM isoforms as described above. Proteins were revealed after SDS/PAGE and immunoblotting with the indicated antibodies. IB protein analysis was performed according to standard protocols. For co-IP, cells were lysed by non-denaturing lysis buffer (1 mM PMSF, 1mM Na3VO4, 1 μg/ml Pepstatin, 20 mM NaF, phosphatase inhibitor cocktail, 0.5% NP-40 and 10% Glycerol in PBS) and the 1mg lysates were cleared by incubation with 50% protein A-Sepharose bead slury, after which 1 mL of the cleared lysates were incubated with antibody-conjugated 50% protein A-Sepharose beads and 10 μL of 10% BSA overnight at 4°C. The beads were washed three times with washing buffer (0.5% NP-40, 0.1% Triton X-

100, 1 mM PMSF, and 1 mM Na3VO4 in PBS). Proteins were revealed after SDS/PAGE and immunoblotting with the indicated antibodies.

Gene expression manipulations

The DNA constructs encoding the amino-terminal region of ASPM-i1 (a.a. 1-1258; fragment 1 in Supplementary Fig. 1A), the protein segment encoded by exon 18 of *ASPM* (a.a. 1356-2940; fragment 2 in Supplementary Fig. 1A), and the carboxy-terminal region (a.a. 2941-3477; fragment 3 in Supplementary Fig. 1A) were PCR-amplified from the full-length human *ASPM* variant 1 (*ASPM*-v1) expression construct pCMV6-Entry-ASPM-Myc-DDK (Origene, RC214770). The amplicons were then subcloned into a V5 epitope- and polyhistidine-tagged expression vector pcDNA 3.1/V5-His A (Invitrogen). The vectors were transduced into cancer cells using the Lipofectamine LTX transfection reagent (Invitrogen). The specific knockdown of *ASPM*-v1 expression, which encodes ASPM-i1, was carried out by lentivirus-mediated transduction of shRNA target sequences specific for the exon 18 of the *ASPM* gene (unique to *ASPM*-v1), as described previously (24). The lentiviral vector encoding a non-target shRNA (MISSION® pLKO.1-puro Non-Mammalian shRNA; SHC002V; Sigma-Aldrich) was used as a control.

Luciferase reporter assay

HT-29 and HCT-116 cells were lentivirally transduced with the lentiviral triple Wnt/Hh/Notch pathway reporter pMuLE_EXPR_CMV-eGFP_TOP-NLuc1.1_12GLI-FLuc_CBF-GLuc (Addgene plasmid #113862, RRID:Addgene_113862) and then stimulated with recombinant human JAG1-Fc (5 μg/ml for 24 hours; Sigma-Aldrich) or vehicle (34). The Notch reporter activity of the cells was measured using the ONE-Glo® Luciferase Assay System (Promega).

Quantitative reverse transcription-polymerase chain reaction (qRT-PCR) analysis

qRT-PCR analysis was performed on the amplified RNA using the LightCycler FastStart DNA MASTERPLUS SYBR Green I Kit and the LightCycler System (Roche Diagnostics GmbH, Mannheim, Germany) according to the manufacturer's instructions. Transcript expression was quantified by normalizing the gene of interest of an endogenous, stably expressed reference gene, ribosomal protein L13a (*RPL13A*). Oligonucleotide primers were designed using Primer Bank (http://pga.mgh.harvard.edu/

primerbank/index.html). The Primers for *NOTCH1* are forward: “5’-GAGGCGTGGCAGACTATGC”, and reverse: “5’-CTTGTACTCCGTCAGCGTGA”. The primers for *RPL13A* are forward: “5’-GCCATCGTGGCTAAACAGGTA”, and reverse: “5’-GTTGGTGTTCATCCGCTTGC”.

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