Supplementary Figure Legends

S1 (Related to Figure 1) TCF4 reporter screen identifies BCL9L as essential for β-catenin-driven reporter activity. (A) β-catenin/TCF4 activity following shRNA-mediated suppression of the top 250 genes found to be required in β-catenin dependent cell lines (3 shRNAs/gene) in DLD1 cells. Reporter activity following suppression of *CTNNB1, BCL9L, APC,* and *CSNK1A1* is indicated. (B) Activity of the β-catenin/TCF4 reporter following suppression of BCL9L. The signal generated by LacZ was used for normalization. Data presented as mean ± SD for 3 independent experiments.

S2 (Related to Figure 2) Suppression of *YAP1* does not affect β-catenin/TCF4 signaling. (A) YAP1 expression 4 d after expression of *YAP1*-specific shRNAs as assessed by immunoblotting in the indicated cell lines. (B) Proliferation and (C) anchorage independent growth of HT29 and LSN411N cells following suppression of *YAP1* or β-catenin. (D) TAZ expression 4 d after expression of *TAZ*-specific shRNAs. (E) Proliferation of cells in (D). (F) β-catenin/TCF4 reporter activity was measured 5 d after expression of β-catenin- or *YAP1*-specific shRNAs. Data presented as mean ± SD for 3 independent experiments. (G) RNA extracted from HCT116 cells expressing β-catenin or *YAP1*-specific shRNAs was subjected to qRT-PCR analysis using the indicated primers. Data presented as mean ± SD for 2 independent experiments. (F). (H) β-catenin protein levels 4 d after expression of *YAP1*-specific shRNAs.

S3 (Related to Figure 2) Orthotopic model of colon cancer. (A) H&E staining of subcutaneous or orthotopic colon tumors derived from the HCT116 cell line. Arrows indicate tumor cells infiltrating the intestine. (B) Mice harboring orthotopic colon tumors containing a doxycycline inducible β-catenin-specific shRNA were treated with doxycycline. Tumors were extracted 3 wk post implantation. (C) Immunoblot analysis of tumors in (B).

S4 (Related to Figure 4) Evaluation of shRNAs targeting *BCL2L1* and *BIRC5*. (A) BCL-XL or (B) BIRC5 expression 4 d after expression of the indicated shRNAs.

S5 (Related to Figure 5) Suppression of SRC does not affect transformation phenotypes in β-catenin active cell lines and tissues. (A) YES1 expression 4 d after expression of *YES1*-specific shRNAs as assessed by immunoblotting. (B) Expression of SRC following suppression of *YES1*. (C) SRC expression 4 d after expression of *SRC*-specific shRNAs, as assessed by immunoblotting. (D) Proliferation or (E) anchorage independent growth of the indicated cell lines following SRC suppression. (F) Proliferation or (G) anchorage independent growth of HuTu80 cells co-expressing YAP1 Y357F and the indicated *YAP1*-specific or control (shLacZ) shRNAs. Data presented as mean ± SD for 2 independent experiments. (H) YAP1 expression in the cells analyzed in (F) and (G). (I) Zebrafish embryos were injected with 50 μM or 200 μM of control, *YAP1*- or *YES1*-specific morpholinos (MO). Fish were fixed and analyzed 3 dpf.

S6 (Related to Figure 5) Nuclear localization of YAP1 in colon cancer cell lines independent of Wnt/β-catenin pathway activity. (A) The indicated colon cancer cell lines were plated in sparse or dense cultures, and β-catenin or YAP1 were analyzed by immunofluorescence. (B) β-catenin-specific or control (shLacZ) shRNAs were introduced into the HuTu80 cell line and following sparse or dense plating β-catenin or YAP1 were analyzed by immunofluorescence.

S7 (Related to Figure 5) Expression of YAP1 across CCLE cell lines. YAP1 expression in 807 cancer cell lines found in the CCLE (Barretina et al., 2012).

Extended Experimental Procedures

Cell proliferation assays

Following shRNA infection and puromycin selection, 6000 cells were plated in 24 well plates in quadruplicates. 8 d later cells were fixed in 10% buffered formalin and stained with crystal violet (0.5% w/v). The crystal violet stain was quantified by addition of 10% (v/v) acetic acid.

Anchorage independent growth assays

Cancer cells ($3x10^5$) or HA1EM cells ($7x10^5$) were seeded in 0.3% Noble agar (Sigma, St. Louis) in 6-well plates, three replicates/sample. Bottom agar consisted of Dulbecco's Modified Eagle's Medium (DMEM) with 0.6% Noble agar, and 8% inactivated fetal calf serum. Colony formation was assessed at 3 wk and images of each well were taken at a 6.25X magnification using an Olympus SZX9 microscope equipped with an Olympus Qcolor 3 camera and the QCapture software. Images were analyzed using ImageJ software (NIH, Bethesda).

Small molecule inhibitor experiments

6000 cell/well were plated in 24-well plates and the indicated doses of dasatinib (Selleck, Houston) or vehicle was added 24 h later. Proliferation was measured after 7 d by crystal violet staining.

Two class permutation analysis

The data from Project Achilles (Cheung et al., 2011) was analyzed using a two-class permutation analysis. Specifically we converted the results of massively parallel screening of 54,020 individual shRNAs targeting 11,194 genes to quantitative, gene-level scores using the ATARiS method (A.T., D.D.S, W.C.H and J.P.M. unpublished data), which identifies sets of shRNAs that exhibit consistent proliferation effects across cell lines. We then used a two-class comparison analysis to detect genes that were significantly essential for the survival/proliferation of cell lines classified as β-catenin active (Figure 1D). For each gene, we assigned a mean difference by calculating the mean dependency score for each gene within its designated class and finding the mean difference between classes. To assess the statistical significance of this calculated difference, we randomly permutated the cell lines between the two classes and assigned a new mean difference for each gene. This process was repeated 50,000 times and a p-value was assigned to each gene representing the likelihood that the particular gene distinguishes the two classes (the optimal p-value that can be achieved is $2x10^{-5}$). To correct for multiple hypothesis testing, we generated False Discovery Rate q-values using the Benjamini-Hochberg method (Benjamini and Hochberg, 1995).

Plasmids

7TFC and 7TFP (Addgene 24308, 24307) were used for characterization of the β-catenin/TCF4 reporter activity (Fuerer and Nusse, 2010). The following plasmids were obtained from Addgene: the FLAG-epitope tagged YAP1 (Addgene 17791) was described (Komuro et al., 2003) and Y357F YAP (Addgene 18882) was from (Levy et al., 2008) the HA-YAP1 (Addgene 27007) was from (Alarcon et al., 2009). For rescue experiments, WT or

mutated YAP1 were cloned into pLX303. For soft agar experiments 5S YAP1 (Addgene 27371) (Zhao et al., 2007) or stabilized β-catenin (Addgene 24313) (Fuerer and Nusse, 2010) were cloned into pLX303 (Yang et al., 2011). WT or dasatinib resistant YES1 were cloned into pLX303. WT SRC, SRC K295R and SRC Y527F were from Dr. Joan Brugge (Addgene 13665, 13659 and 13660). FYN expression vector (Addgene 16032) was from (Mariotti et al., 2001). For TBX5 interaction assays, we cloned a FLAG-epitope tagged version of TBX5 (Addgene 32968) (He et al., 2011) into pLX303.

shRNA and ORF expression

The pLKO.1 vector was used to express shRNAs (sequences of shRNAs are noted in Table S2), and pLX303 was used to express ORFs. Lentiviruses were produced in 293T cells using the three-vector system as described (Moffat et al., 2006; Yang et al., 2011). The virus was diluted (1:15) and added to $2.5x10^5$ cells in a 6 well plate containing 8 μg/ml of polybrene (Sigma). Plates were centrifuged for 15 min, 1178 X *g* at RT. For selection of virally infected cells, 2 μg/ml of Puromycin or 5 μg/ml of Blastocidin was used 24 h post infection.

Immunoblotting

Cells were lysed using RIPA buffer (Cell Signaling, Beverly, MA) with the addition of protease and phosphatase inhibitors (Roche, Indianapolis, IN). Following lysis, cells were sonicated for 10 sec and then centrifuged for 10 min at 4^{o}C. The protein concentration of the lysate was quantified using the BCA protein quantitation kit (Pierce) and 30 μg of lysate was in SDS polyacrylamide gel electrophoresis (Invitrogen). The blots were probed with the indicated antibodies β-catenin (Cell signaling or BD Pharmingen, Sparks, MD) YAP1 (Cell signaling, Beverly, MA), YES1 (BD Transduction, Chicago, IL), actin (Santa Cruz, Santa Cruz, CA), pY (Santa Cruz, Santa Cruz, CA), YAP1 pY357 (Abcam), SRC (Santa Cruz, Santa Cruz, CA), BCLXL (Cell Signaling Technology, Beverly, MA), BIRC5 (Cell Signaling Technology, Beverly, MA).

Reporter assay

For the characterization of β-catenin activity in 85 cancer cell lines, cells were infected with a lentivirally delivered β-catenin reporter that harbors a puromycin selection marker, TFP (Fuerer and Nusse, 2010). Following Puromycin selection, 50,000 cells were plated in 96-well plates, and luciferase luminescence was measured using the Luc-Screen detection kit 24 h later (Applied Biosystems, Carlsbad, CA). For characterization of the β-catenin activity following shRNA expression, the cells were infected with a lentivirally delivered β-catenin reporter harboring a mCherry selectable marker, TFC (Fuerer and Nusse, 2010) and after FACS sorting the same cells were infected with a Blastocidin-selectable LacZ expression vector. Following selection, the cells were infected with lentiviruses containing the indicated shRNAs. Luciferase and LacZ activity were measured 5 d post infection using the Dual-light detection kit (Applied Biosystems, Carlsbad, CA). The Luciferase signal was normalized to that observed upon expression of LacZ.

Co-immunoprecipitation assay

Lysis buffer [50mM Tris-HCl pH=7.4, 150 mM NaCl, 1 mM EDTA, 0.5% Na-Deoxycholate (v/v), 1% Triton X-100 (v/v) containing protease inhibitors (Roche)] was added to cells.

Following scraping, lysates were sonicated lightly and centrifuged for 10 min at 4˚C. 2 mg of lysates was incubated overnight at 4˚C with a β-catenin (BD Pharmingen) or YAP1 (Cell Signaling Technology) antibody. The lysates were then incubated for 2 h at 4˚C with Dynabeads protein G (Invitrogen, Grand Island, NY) and then washed three times with wash buffer (50mM Tris-HCl pH=7.4, 150 mM NaCl, 1% Triton X-100 (v/v)). The beads were then re-suspended with 2X sample buffer boiled for 5 min and analyzed by SDS-PAGE. Following SDS-PAGE separation and transfer to a nitrocellulose membrane, the blot was probed with either a β-catenin specific antibody (Cell Signaling Technology or BD Pharmingen) or with a YAP1 specific antibody (Cell Signaling Technology).

Immunofluorescence

The indicated cells were plated on a microscope slide at either a dense (100,000 cells/well) or sparse (10,000 cells/well) concentration. After 24 h at 37˚C, cells were fixed by adding 10% buffered formalin. Cells were permeabilized by adding 0.5% Triton in PBS (v/v) for 15 min at room temperature. The slides were then washed with PBS and blocked for 2 h with serum free protein block (DakoCytomation). Each well was incubated with a cocktail containing 1:50 anti-β-catenin (BD Pharmingen) and 1:50 of anti-YAP1 (Cell signaling) for 2 h at RT. Following three washes secondary mouse and rabbit fluorescent antibodies were added (1:400) and incubated for an additional h at RT. The slides were then washed and visualized using an Olympus BX50 microscope equipped with a Qimaging Retiga EXi camera.

Chromatin Immunoprecipitation

Cells growing on monolayer were fixed for 15 min at RT with 1% formaldehyde. The crosslinking reaction was stopped by adding 2.5 M glycine and 10 min incubation at room temperature. Cells were washed twice with cold PBS and then were incubated with RIPA buffer (Cell Signaling Technology) containing protease inhibitors (Roche) for 20 min on ice. Cells were then scraped and sonicated, and the lysates obtained were centrifuged at 9300 X *g*, 4˚C for 10 min, and 1 mg of the supernatant was incubated with 10 µl of either YAP1 (Santa Cruz) or β-catenin (BD Pharmingen) antibody overnight at 4˚C. The next day 50 µl of Dynabead protein G (Invitrogen) were added for 2 h at 4˚C. The beads were then washed twice with cold RIPA following by 4 washes with wash buffer (100 mM Tris-HCl pH=8.5, 500 mM LiCl, 1% NP-40 (v/v), 1% deoxycholic acid (v/v). Beads were then washed again twice with RIPA buffer and beads were incubated with 50 µl of TE buffer. The DNA was then reverse cross-linked by adding 200 µl of Talianidis buffer [70 mM Tris-HCl pH=8, 1 mM EDTA, 1.5% SDS (w/v)] and incubating for 10 min at 65˚C. The beads were then centrifuged and the supernatant containing DNA was collected. DNA was amplified with primers specific for BIRC5 or BCL2L1 (Table S3) and using the KOD polymerase kit (EMD). The primers for BIRC5 and BCL2L1 were chosen after analyzing these promoters for β-catenin binding sites identified by β-catenin ChIP-sequencing. Specifically, we isolated β-catenin-associated chromatin from four β-catenin dependent colon cancer cell lines (DLD1, LoVo, HT29 and HCT116) using an anti-β-catenin antibody (Santa Cruz Biotechnology, sc-7199). Twenty ng of ChIP DNA or whole-cell extract from three biological replicates were used to generate an Illumina sequencing library. Briefly, DNA fragments were end-repaired using the End-It DNA End-Repair Kit (Epicentre) and then a single “A” base was added

using Klenow (NEB). The fragments were ligated with Illumina Indexed adaptors (TruSeq DNA Sample Prep Kits) using DNA ligase (NEB). The ligated product was selected for 300-400 bp on 2% agarose gel to remove the non-ligated adaptors and was subjected to 18 PCR cycles with Illumina PCR primer cocktail (TruSeq DNA Sample Prep Kits). PCR product was purified on 2% agarose gel to retain fragment between 300-400 bp. Library concentrations were quantified by Qubit fluorometer (Invitrogen) and by quantitative PCR (Kapa Biosystem). Two barcoded libraries were pooled and sequenced to 50 bp in a single lane on Illumina HiSeq2000 using standard procedures for cluster amplification and sequencing by synthesis using the latest versions of Illumina software (instrument: HCS 1.5.15.1 - RTA 1.13.48, Pipeline: Illumina CASAVA 1.8.2). The MACs analysis method (Zhang et al., 2008) was used to call β-catenin occupied peaks.

Quantitative Reverse Transcriptase PCR

RNA was harvested from cells using QiaShredder and RNeasy (Qiagen). Complementary DNA was prepared using Advantage RT-for-PCR according to manufacturer's instructions (Clontech). Quantitative PCR was carried out using SYBR green (Applied Biosystems). The primers used are listed in Supplementary Table S3.

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Supplementary Table 1 - (Related to Figure 1): β-catenin/TCF4 reporter signal in 85 cancer cell lines.

	Cell line	Origin	APC/β-catenin mut	TCF4 Reporter Signal	SD	Status		Cell line	Origin	APC/β-catenin mut	TCF4 Reporter Signal	SD	Status
1	SW480	Colon	Yes	13034.6	1124.8	active	44	Caov-4	Ovarian	No	7.4	1.3	non-active
2	SNU-C1	Colon	No	2123.2	3.2	active	45	Hey-A8	Ovarian	No	7.4	2.1	non-active
3	C2BBe1	Colon	Yes	1969.0	210.4	active	46	NCI-H1975	Lung	No	7.1	5.3	non-active
4	DLD1	Colon	Yes	1360.7	39.8	active	47	F5	Meningioma	No	6.7	12.1	non-active
5	TOV-112D	Ovarian	No	972.6	66.4	active	48	OVCAR-4	Ovarian	No	6.4	4.3	non-active
6	Colo-205	Colon	Yes	816.8	82.5	active	49	T.T	Ovarian	No	6.3	5.5	non-active
7	LS513	Colon	Yes	712.9	100.4	active	50	SU.86.86	Pancreas	No	6.0	6.1	non-active
8	HuTu80	Colon	Yes	685.5	22.1	active	51	HT29	Colon	Yes	5.6	1.2	non-active
9	HT55	Colon	Yes	345.4	162.3	active	52	KURMOCHI	Ovarian	No	5.3	5.6	non-active
10	AGS	Gastric	Yes	235.7	10.3	active	53	RMG-I	Ovarian	No	5.0	2.5	non-active
11	SK-CO-1	Colon	Yes	192.5	34.5	active	54	NCI-H82	Lung	No	5.0	2.5	non-active
12	NCI-H508	Colon	Yes	158.0	6.8	active	55	IGR39	Ovarian	No	4.6	16.9	non-active
13	GP2d	Colon	Yes	124.0	14.0	active	56	HCC-364	Lung	No	4.3	5.6	non-active
14	SW48	Colon	Yes	86.2	23.3	active	57	OVCAR-8	Ovarian	No	4.3	2.1	non-active
15	OE33	Esophageal	No	73.9	5.6	active	58	KMS12BM	Multiple Myeloma	No	4.2	3.7	non-active
16	Hu-G1N	Gastric	No	68.6	9.6	active	59	IGROVI	Ovarian	No	3.5	3.2	non-active
17	LoVo	Colon	Yes	61.5	32.1	active	60	NCI-H1650	Lung	No	3.5	4.4	non-active
18	A549	Lung	No	46.0	11.7	active	61	A2780	Ovarian	No	3.2	8.4	non-active
19	IOMM-LEE	Meningioma	No	41.3	8.5	active	62	KYSE-450	Esophageal	No	3.1	2.1	non-active
20	COV-504	Ovarian	No	36.8	7.5	non-active	63	MDA-MB-453	Brest	No	2.4	3.2	non-active
21	OVISE	Ovarian	No	32.5	5.6	non-active	64	HL-60	Leukemia	No	2.4	3.2	non-active
22	NCI-H2122	Lung	No	28.4	3.6	non-active	65	KYSE-510	Esophageal	No	2.4	4.4	non-active
23	OVMANA	Ovarian	No	26.4	2.3	non-active	66	HCC-70	Brest	No	1.7	3.2	non-active
24	A2058	Melanoma	No	24.5	2.1	non-active	67	EFO-27	Ovarian	No	1.2	0.3	non-active
25	CFPAC-1	Pancreas	No	23.4	5.6	non-active	68	Panc.03.27	Pancreas	No	1.1	2.1	non-active
26	RKO	Colon	No	21.3	15.1	non-active	69	BxPC-3	Pancreas	No	1.0	8.4	non-active
27	HPAC	Pancreas	No	21.3	4.2	non-active	70	LN215	GBM	No	0.1	4.2	non-active
28	TOV-21G	Ovarian	No	20.0	7.6	non-active	71	MIA-Paca-2	Esophageal	No	-0.4	4.4	non-active
29	Panc.10.05	Pancreas	No	18.3	3.7	non-active	72	EFO-21	Ovarian	No	-0.4	2.4	non-active
30	JHESOAD1	Esophageal	No	17.7	16.1	non-active	73	HCC-827	Lung	No	-0.7	4.4	non-active
31	COLO-704	Ovarian	No	17.3	3.3	non-active	74	NCI-H661	Lung	No	-0.7	3.2	non-active
32	LN319	GBM	No	17.0	4.2	non-active	75	U251	GBM	No	-1.3	3.2	non-active
33	Panc.08.13	Pancreas	No	15.9	2.5	non-active	76	SNU-C2A	Colon	No	-1.8	4.4	non-active
34	HLF	Liver	No	14.5	2.1	non-active	77	LN229	GBM	No	-2.0	2.1	non-active
35	LS411N	Colon	Yes	12.8	2.1	non-active	78	SF767	GBM	No	-2.7	5.3	non-active
36	TE-9	Esophageal	No	11.4	9.5	non-active	79	Colo-741	Colon	No	-2.7	14.8	non-active
37	OV-90	Ovarian	No	10.6	5.6	non-active	80	TE-15	Esophageal	No	-3.9	8.0	non-active
38	HEC-1A	Endometrial	No	10.5	2.3	non-active	81	AsPC-1	Pancreas	No	-4.6	2.4	non-active
39	KP-4	Pancreas	No	9.4	2.1	non-active	82	CH157-MN	Meningioma	No	-4.6	4.9	non-active
40	Caov-3	Ovarian	No	8.8	8.8	non-active	83	KM12	Colon	No	-5.7	4.4	non-active
41	SJSA-1	Osteosarcoma	No	8.7	7.4	non-active	84	KYSE-30	Esophageal	No	8.5	8.5	non-active
42	KYSE-150	Esophageal	No	8.7	5.2	non-active	85	NIH:OVCAR3	Ovarian	No	7.8	7.5	non-active
43	LN464	GBM	No	8.5	8.4	non-active							

Supplementary Table 2 (Related to experimental procedures): target sequences of shRNAs used in this study.

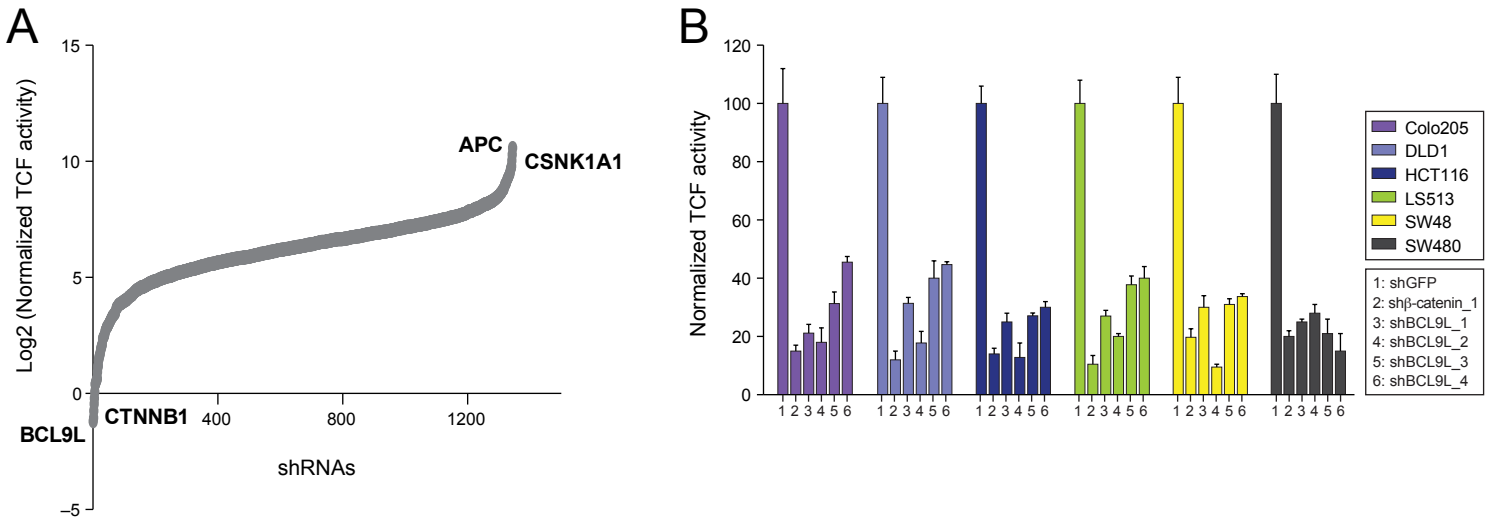
shRNA name	Target sequence
shLacZ	CGCTAAATACTGGCAGGCGTT
shGFP	ACAACAGCCACAACGTCTATA
shβ-catenin 1	GGTGTCTGCTATTGTACGTAC
shβ-catenin 2	GCTTGGAATGAGACTGCTGAT
shTCF4 1	AGAGAAGAGCAAGCGAAATAC
shTCF4 2	TATCGAGTTCATTGGTCAATA
shBCL9L 1	GCCACCCACAATTGTAATGTA
shBCL9L 2	CATGGGCAATACCCAAGACAT
shBCL9L 3	GCATCTCATGAACCTGCAGAA
shBCL9L 4	CCCAGCAGAATTTCATGCTGA
shYAP 1	GCCACCAAGCTAGATAAAGAA
shYAP 2	CCCAGTTAAATGTTCACCAAT
shYAP 3	GACCAATAGCTCAGATCCTTT
shYAP 4	CGACCAATAGCTCAGATCCTT
shTAZ 1	CAGCCAAATCTCGTGATGAAT
shTAZ 2	GCCCTTTCTAACCTGGCTGTA
shYES1 1	GCAGATTCCATTCAGGCAGAA
shYES1 2	ACCACGAAAGTAGCAATCAAA
shBCL2L1 1	GTGGAACTCTATGGGAACAAT
shBCL2L1 2	GCTCACTCTTCAGTCGGAAAT
shBIRC5 1	CCTTTCTGTCAAGAAGCAGTT
shBIRC5 2	CCGCATCTCTACATTCAAGAA
shTBX5 1	GCTGCACAGAATGTCAAGAAT
shTBX5 2	CCTATGCGATTATGTCTCTTT

Supplementary Table 3 (Related to experimental procedures): primers used in this study.

Primer name	Sequence (5'→3')	Used for
FBIRC5	AGGACCACCGCATCTCTACAT	qRT-PCR
RBIRC5	CACTGAGAACGAGCCAGACTT	qRT-PCR
FcMYC	GCCACGTCTCCACACATCAG	qRT-PCR
RcMYC	AAGGACTATCCTGCTGCCAAGA	qRT-PCR
FSOX4	AAACCAACAATGCCGAGAACA	qRT-PCR
RSOX4	GGCACATCAAGCGACCCAT	qRT-PCR
FAXIN2	AGCCAAAGCGATCTACAAAAGG	qRT-PCR
RAXIN2	GTGATGGAGGAAAATGCCTACC	qRT-PCR
FBCL2L1	CTTCCGGGATGGGGTAAACTGG	qRT-PCR
RBCL2L1	AGCCTTGGATCCAGGAGAACGG	qRT-PCR
FACTIN	CAAGAGATGGCCACGGCTGCT	qRT-PCR
RACTIN	TCCTTCTGCATCCTGTCGGCA	qRT-PCR
FTBX5	CCAGCCTAGATTACACATCGTG	qRT-PCR
RTBX5	AAATACAGCGTTCTGCACTCA	qRT-PCR
FBIRC5	TTCTCTTGTCATATTAAGTCCACACCGC	CHIP
RBIRC5	GCTGTCCTTGTCCATGCATTTGAT	CHIP
FBCL2L1	GGAGGAGGAAGCAAGCGAGGGG	CHIP
RBCL2L1	TAAACTGGGGTCGCATTGTGGCC	CHIP

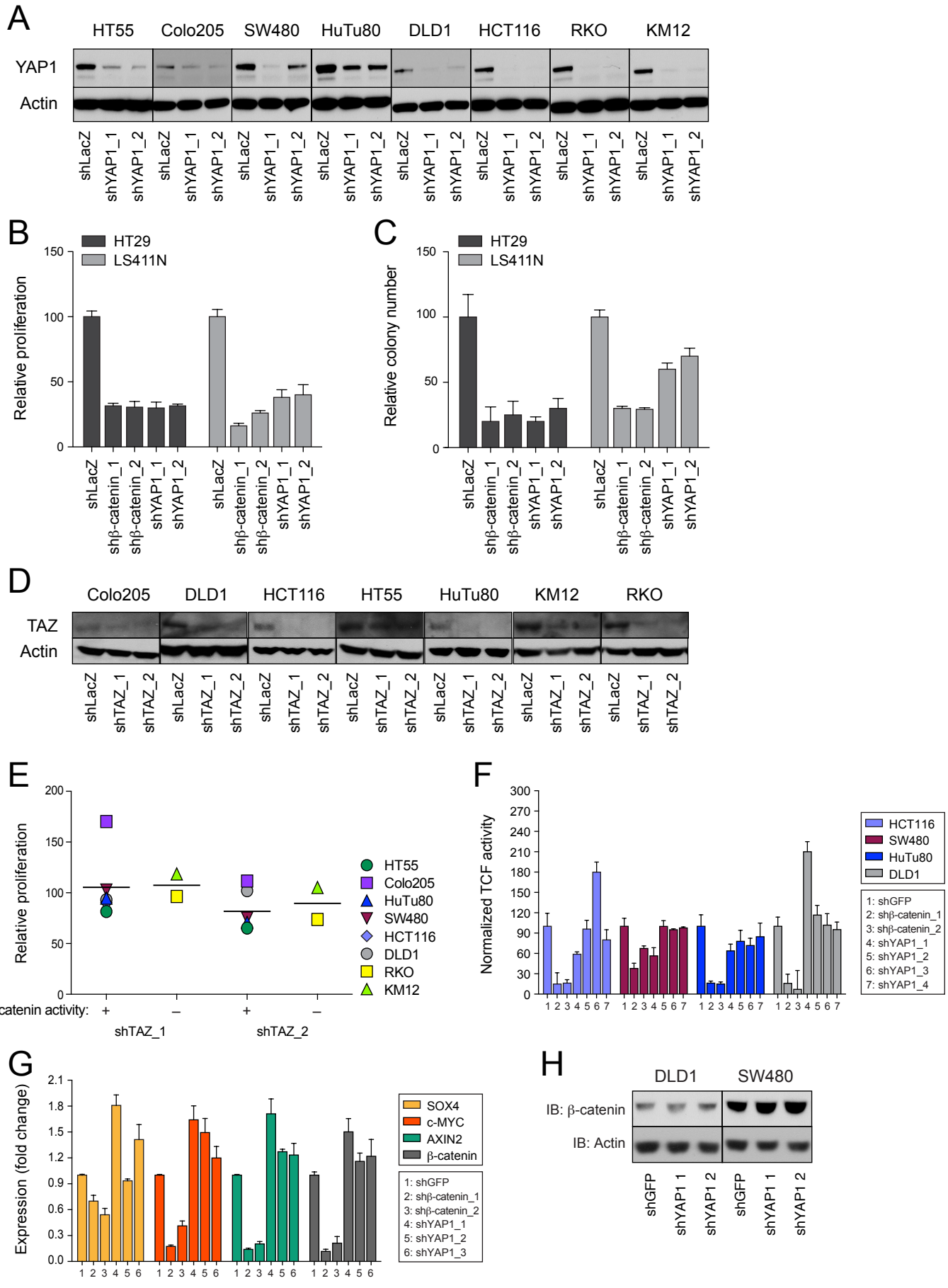
Supplemental Figure 1

Supp. Figure 1



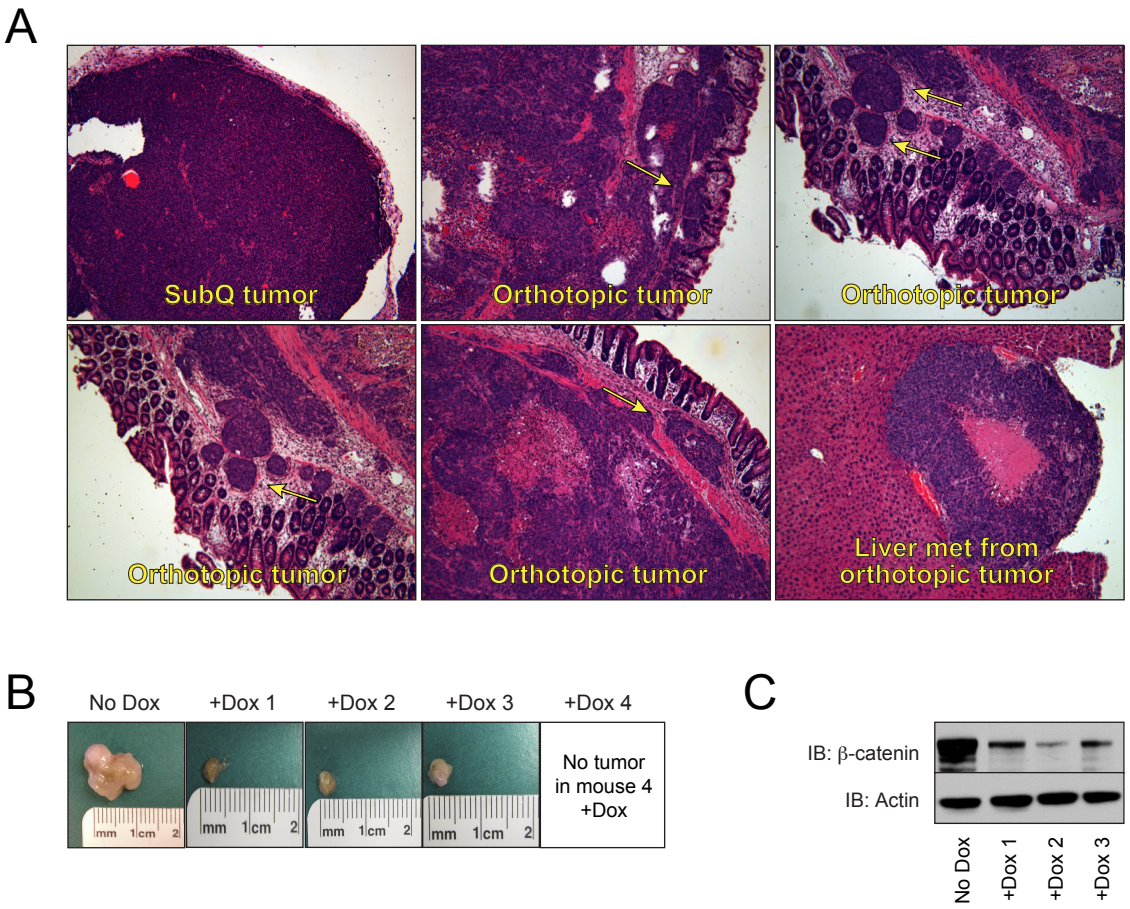
Supplemental Figure 2

Supp. Figure 2



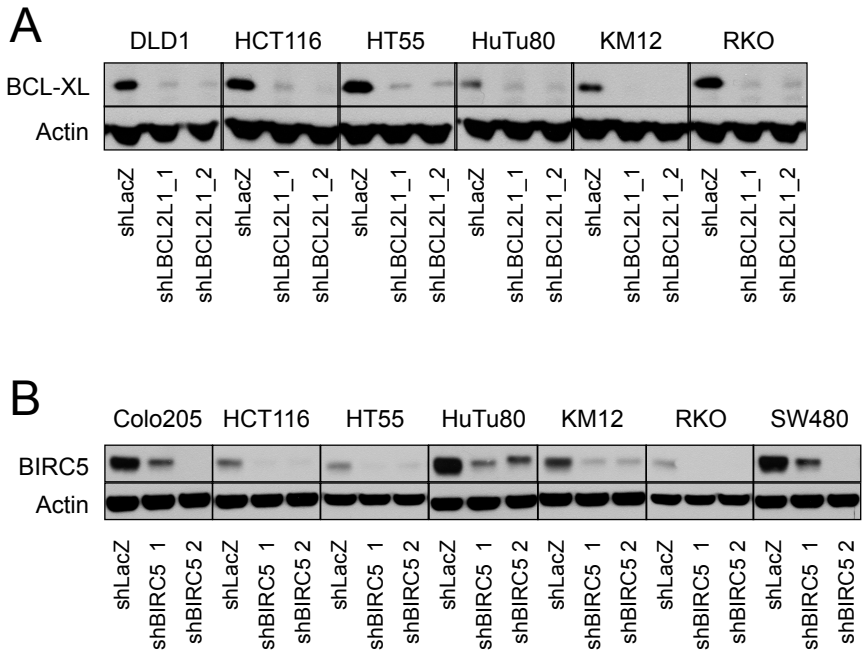
Supplemental Figure 3

Supp. Figure 3

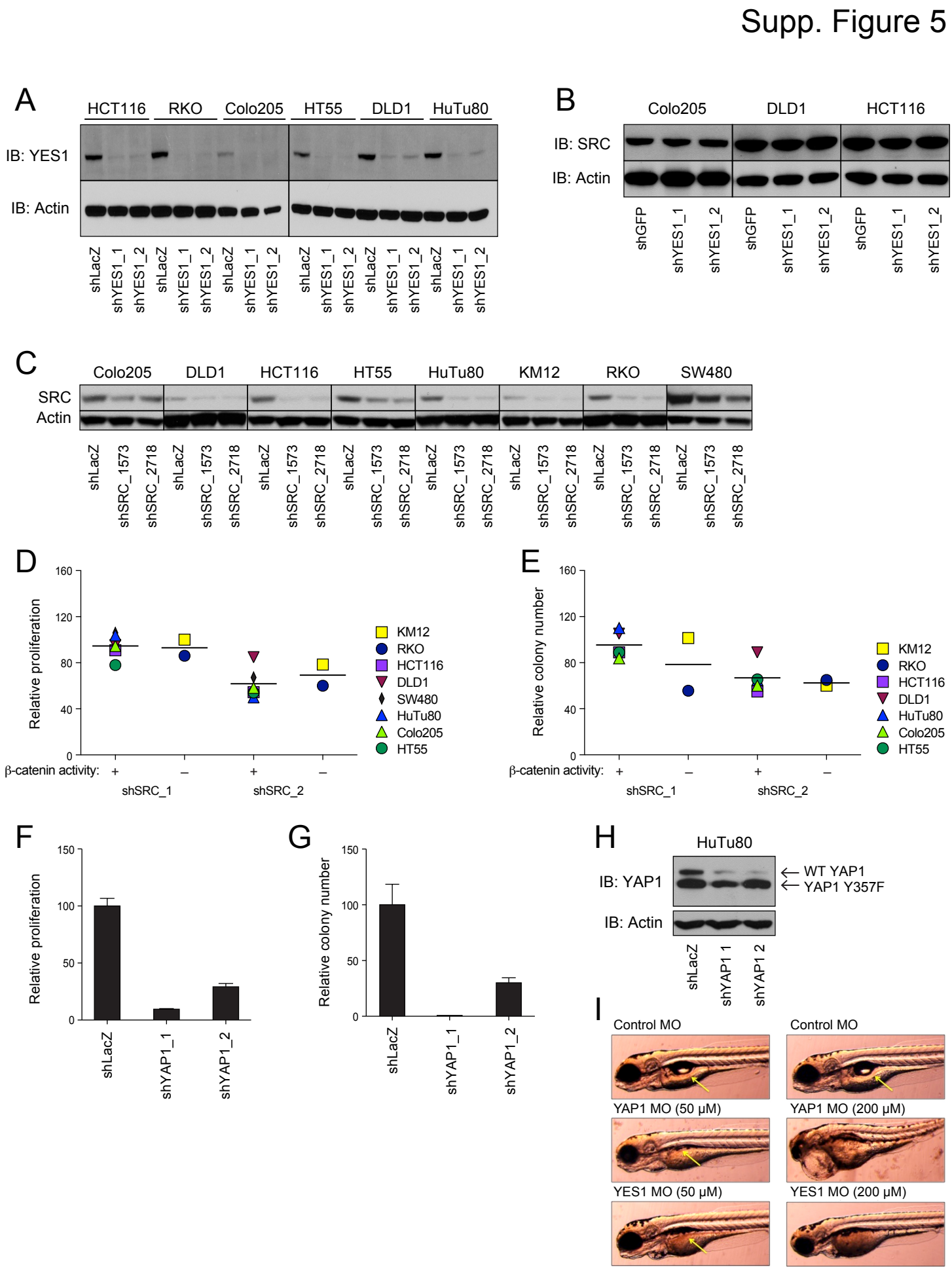


Supplemental Figure 4

Supp. Figure 4

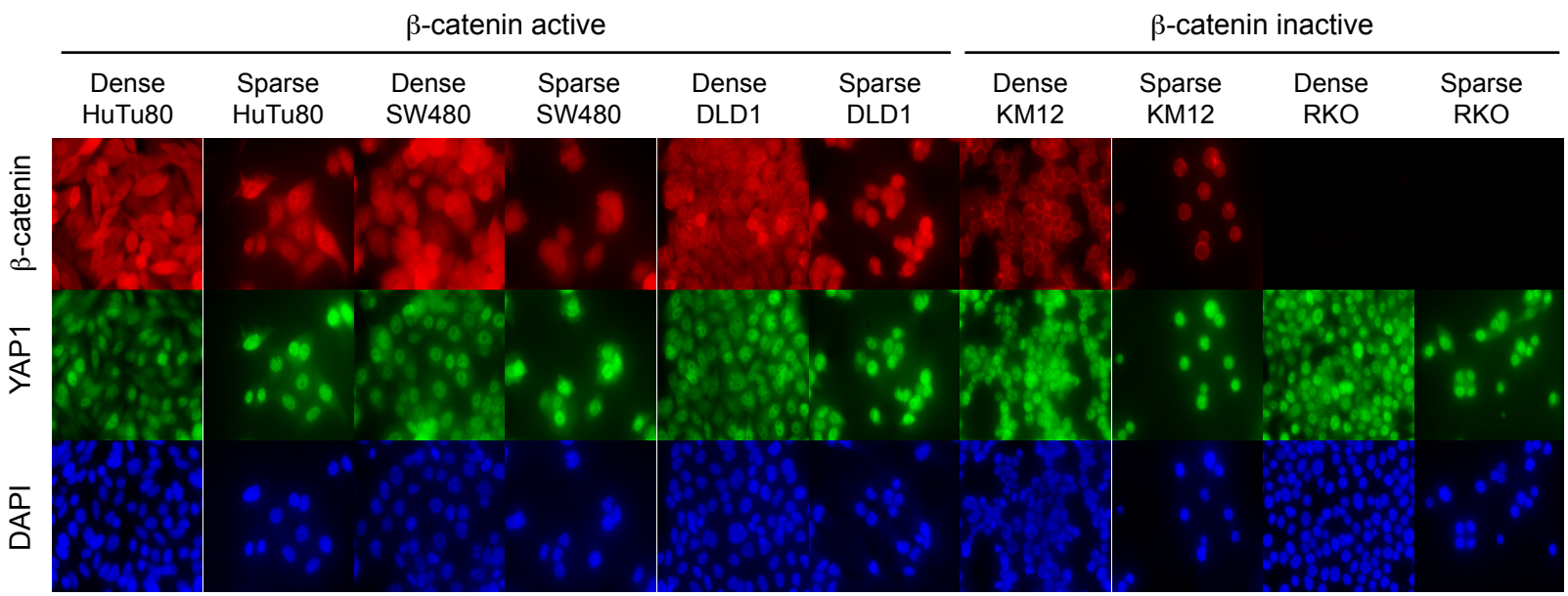


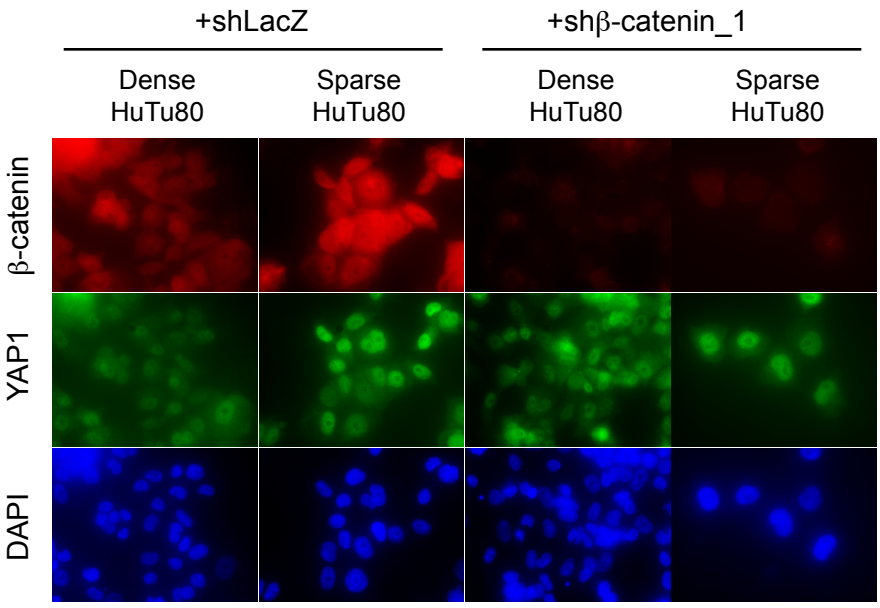
Supplemental Figure 5



Supplemental Figure 6

Supp. Figure 6





Supplemental Figure 7

Supp. Figure 7